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Can the Esophageal Doppler Monitor Be Used to Clinically Evaluate Peak Left Ventricle dP/dt?

GLEN M. ATLAS*

The esophageal Doppler monitor (EDM) is minimally invasive and allows for rapid and continuous cardiovascular measurements which are based upon aortic blood flow characteristics. Using a model derived from the modified Bernoulli equation, a measure of peak left ventricle (PLV) dP/dt has been developed which utilizes EDM-based parameters: PLV $\frac{dP}{dt} \propto \frac{V_P^2 \cdot \sqrt{\text{HR}}}{T_{\text{FC}}}$; where V_p represents peak velocity of aortic blood flow, T_{FC} is corrected flow time, and HR is heart rate. Additional clinical research is necessary to provide a correlation of this formula with invasive measurements. A wave transmission model of PLV dP/dt has also been examined. This model requires simultaneous measurement of aortic pulse wave velocity, or aortic flow wave velocity, in order to calculate PLV dP/dt. Current echocardiographic analyses of PLV dP/dt show that the wave transmission model provides better correlation with in vivo catheterization results when compared with the modified Bernoulli equation. The EDM remains a useful tool for rapid and continuous evaluation of cardiovascular indices. Further research and development, of this monitor for PLV dP/dt assessment, is warranted.

Key words: esophageal Doppler monitor; dP/dt; aortic pulse wave velocity; aortic flow wave velocity.

INTRODUCTION

It is frequently useful to know peak left ventricle (PLV) dP/dt for patients with aortic dissections as well as those undergoing aortic reconstructive surgery. Propagation of aortic dissections has been shown to be a function of dP/dt (Carney *et al*., 1975; Prokop *et al*., 1970). In addition, dP/dt may be a useful cardiac index as well (Schertel, 1998).

Using only continuous wave Doppler ultrasound, PLV dP/dt has been shown to correlate with peak aortic blood flow velocity and acceleration (Bargiggia *et al*., 1989; Hunt *et al*., 1991; Saeian *et al*., 1990). PLV dP/dt has also been successfully derived using a combination of aortic pulse wave velocity and Doppler ultrasound measurements (Senda *et al*., 1992; Sugawara *et al*., 1994). These physical models are based either upon the modified Bernoulli equation or a wave transmission model and are reviewed in Appendixes A and B respectively.

Table 1 summarizes the results of several human studies in which PLV dP/dt was determined noninvasively from aortic blood flow characteristics and then simultaneously compared to left ventricle cardiac catheterization data.

An esophageal Doppler monitor (EDM) has several advantages over traditional ultrasound devices in that it is less expensive, easier to use, and offers continuous real-time measurement of both peak velocity and mean acceleration of descending aortic blood flow (Singer, 1993). Furthermore, the EDM probe can easily be placed orally and used without difficulty with intubated patients who are anesthetized or sedated. Nasal placement, in awake patients, has also been described (Atlas and Mort, 2001).

This device, with its ability to readily measure aortic blood flow indices, would then seem to be potentially useful in minimally-invasive clinical PLV dP/dt evaluation.

DERIVATION

Using only continuous wave transthoracic Doppler ultrasound, PLV dP/dt has been shown to correlate with

*Department of Anesthesiology, University of Medicine and Dentistry of New Jersey, New Jersey Medical School, 185 S. Orange Avenue, MSB E-538, Newark, New Jersey 07103. E-mail: atlasgm@umdnj.edu

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Table 1. Various Methods Have Been Used to Noninvasively Determine Peak Left Ventricle dP/dt from Aortic Blood Flow Characteristics

Correlation with PLV dP/dt	Physical model	Reference	Correlation coefficient[a]	Notes[b]
Peak aortic blood flow velocity (V_P)	$V_P \propto \text{PLV}\frac{dP}{dt}$	Hunt *et al.*, 1991	0.70	(1)
Mean aortic blood flow acceleration	$\text{AVG}(\frac{dV}{dt}) \propto \text{PLV}\frac{dP}{dt}$	Hunt *et al.*, 1991	0.75	(2, 3)
$\frac{V_p^2}{T}$	Modified Bernoulli	Hunt *et al.*, 1991	0.77	(1)
$\frac{dP}{dt} = \rho \cdot V_{pw} \cdot \frac{dV}{dt}$	Wave transmission	Senda *et al.*, 1992; Sugawara *et al.*, 1994	0.83; 0.84	(3)

[a]The correlation coefficients are based upon comparisons with in vivo catheterization results.
[b](1) V_P is peak aortic blood flow velocity and T is the time from the start of systole until peak velocity. See Figs. 1 and 2; (2) Mean aortic blood flow acceleration is determined from the start of systole until peak velocity. See Figs. 1 and 2; (3) ρ represents blood density and V_{pw} is aortic pulse wave velocity. dV/dt is acceleration of aortic blood flow.

peak aortic blood flow velocity (V_P) by the following formula (Hunt *et al.*, 1991):

$$\text{PLV}\frac{dP}{dt} \propto \frac{V_p^2}{T} \tag{1}$$

where T represents the time from the onset of aortic blood flow until it reaches peak velocity V_p. It should be noted that Eq. (1) represents an approximation of the first derivative, with respect to time, of the modified Bernoulli equation. This is described in Appendix A.

Figure 1 illustrates the relatioship between V_p and T for a representative EDM-derived aortic Doppler signal. Hunt *et al.* (1991) showed good correlation of $\frac{V_p^2}{T}$ to PLV dP/dt and only slightly less correlation of V_p and mean acceleration to PLV dP/dt. These results are summarized in Table 1 (see Figs. 1 and 2).

Therefore, the ease of measuring V_p and mean acceleration, in real-time with current EDM technology, would make either of these clinically appealing "markers" for quickly assessing PLV dP/dt at the bedside.

Corrected flow time, T_{FC}, ordinarily correlates with preload and is readily determined with the EDM. The relationship between cycle time and flow time, T_F, is shown in Fig. 1. Equation (2) defines T_{FC}, utilizing T_F, and is based upon a modification of Bazett's equation:

$$T_{FC} = \frac{T_F}{\sqrt{\text{Cycle time}}} \tag{2}$$

Cycle time is equivalent to RR interval and is related to heart rate, HR, as shown below:

$$\text{Cycle time} = \text{RR interval} = \frac{60}{\text{HR}} \tag{3}$$

where HR is expressed in beats per minute and cycle time, as well as RR interval, are expressed in seconds. In addition, the time to peak velocity, T, and flow time, T_F, are related:

$$T \approx \frac{T_F}{2} \tag{4}$$

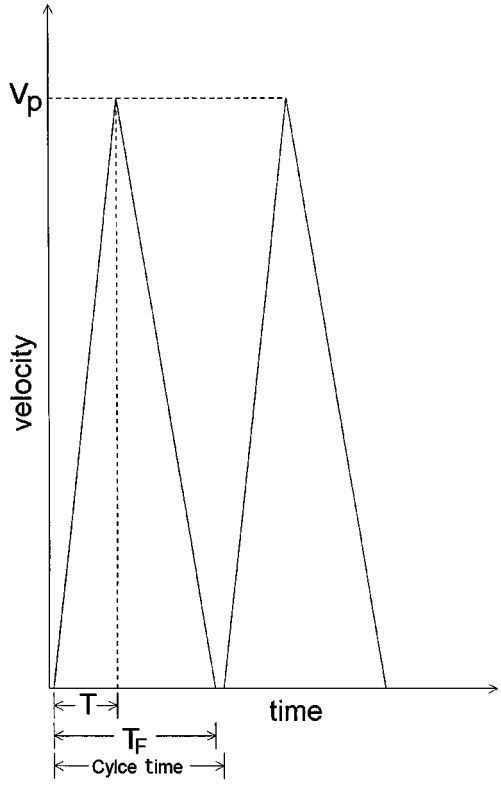

Figure 1. This diagram illustrates the aortic blood flow velocity vs. time relationship. The EDM is a minimally-invasive device which is able to measure this accurately and in real time. T represents time until peak velocity and T_F is flow time or left ventricle ejection time.

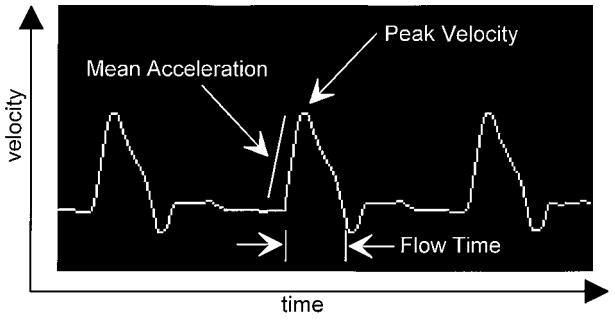

Figure 2. Typically EDM waveform. Mean acceleration is the average slope of aortic blood flow velocity from onset of systole until peak velocity. Flow time represents left ventricle ejection time.

Eqs. (2)–(4) can be substituted into Eq. 1 to yield the following:

$$\mathrm{PLV}\frac{dP}{dt} \propto \frac{V_{\mathrm{P}}^2 \cdot \sqrt{\mathrm{HR}}}{T_{\mathrm{FC}}} \tag{5}$$

Thus, based on Eq. (5), it would seem that the EDM could provide a useful real-time assessment of PLV dP/dt using a model similar to that of Hunt *et al.* (1991).

By combining simultaneous pulse wave velocity measurement with aortic Doppler ultrasound, excellent correlations, of derived-to-measured PLV dP/dt, have also been obtained (Senda *et al.*, 1992; Sugawara *et al.*, 1994). This has been accomplished with the use of a wave transmission model in which PLV dP/dt is determined with the following equation of continuity (see Appendix B):

$$\frac{dP}{dt} = \rho \cdot V_{\mathrm{pw}} \cdot \frac{dV}{dt} \tag{6}$$

where ρ represents blood density, V_{pw} is aortic pulse wave velocity, and dV/dt is acceleration of aortic blood flow.

DISCUSSION

Given the clinical benefit of the EDM for safe and rapid assessment of cardiovascular parameters, it would seem reasonable to evaluate this device for its usefulness as a minimally-invasive tool for bedside evaluation of PLV dP/dt. Pharmacological management of aortic dissections, as well as other aortic injuries, could than potentially be improved.

Currently, labetalol, a combination nonspecific β blocker and α blocker, remains the treatment of choice for hemodynamic control of patients with aortic dissections (Grubb *et al.*, 1987). Esmolol, an ultra-short acting β blocker, has also been reported as useful (O'Conner and Luntley, 1995). Therefore, the utility of these agents could possibly be evaluated, in real-time, with EDM-based measurements of PLV dP/dt.

Using existing EDM technology, clinical results, based on Eq. (5), would seem easily comparable with simultaneously obtained measurements from in vivo catheterization.

It is important to realize that dP/dt can be modeled, with Eq. (6), as a function that is based upon aortic pulse wave velocity. This has been substantiated using the wave transmission model by Senda *et al.*, 1992 and Sugawara *et al.*, 1994. With this model, they have demonstrated excellent correlations of PLV dP/dt directly to catheterization results.

The clinical utility of aortic pulse wave velocity has been investigated. As shown in Appendix B, aortic V_{pw} is inversely associated with aortic compliance (Lehmann, 1999). In subjects over 70 years old, aortic pulse wave velocity was found to be a marker of cardiovascular disease (Meaume *et al.*, 2001). In hemodialysis patients, an increased V_{pw} has been shown to be an independent predictor of both all-cause and cardiovascular mortality (Blacher *et al.*, 1999a). V_{pw} has also been shown to increase with atherosclerotic disease (Blacher *et al.*, 1999b; Hopkins *et al.*, 1994; Lehmann, 1999). Furthermore, in Marfan syndrome patients, β blocker therapy has been shown to decrease aortic V_{pw} (Groenink *et al.*, 1998).

It should be noted that typical values for aortic V_{pw}, in humans, range from 3 to 10 $\mathrm{M} \cdot s^{-1}$. In addition, V_{pw} normally increases with age (Bulpitt *et al.*, 1999; Rogers *et al.*, 2001).

Therefore, V_{pw} appears to be a clinically useful marker of aortic compliance. The wave transmission model of dP/dt takes this parameter into account in contrast to models based upon the modified Bernoulli equation.

If the effects of reflected waves are assumed to be negligible, then aortic flow wave velocity and pulse wave velocity would be identical. It should be noted that aortic flow wave velocity, V_{fw}, has also been investigated using magnetic resonance imaging. Age-related values for V_{fw}, that are similar to those for V_{pw}, were found. In addition, values of V_{fw} were shown to inversely correlate with those of aortic compliance (Kraft *et al.*, 2001; Mohiaddin *et al.*, 1993).

Thus, using the wave transmission model, it may be reasonable to correlate aortic flow wave velocity with aortic pulse wave velocity in noninvasive, or minimally-invasive, determinations of PLV dP/dt.

SUMMARY

Therefore, the EDM may provide a useful means of assessing PLV dP/dt. This could easily be accomplished with existing EDM indices. Currently, there is no readily-available instrument that allows for simple and rapid bedside determination of PLV dP/dt.

Clearly, this information could potentially provide the clinician with an improved ability to care for patients with aortic dissections and related injuries. The use of PLV dP/dt, as a cardiac index, would also be valuable.

It should also be remembered that a clinically useful guide to "gauging" PLV dP/dt may be obtained from simply observing the peak velocity, or mean acceleration, of aortic blood flow.

Further studies are warranted to correlated EDM-derived aortic dP/dt data with in vivo catheterization results. In addition, future means of assessing PLV dP/dt could possibly include simultaneous measurement of aortic pulse wave velocity.

Determination of aortic flow wave velocity, and its correlation with aortic pulse wave velocity, may also facilitate real-time PLV dP/dt evaluation.

APPENDIX A: MODIFIED BERNOULLI EQUATION

The Bernoulli equation states that hydraulic pressure, P, is the sum of ambient pressure, kinetic energy per unit volume, and hydrostatic pressure (Milnor, 1982):

$$P = P_a + \frac{1}{2}\rho v^2 + \rho g h \tag{1A}$$

where P_a is ambient pressure, ρ is blood density, v is the velocity of aortic blood flow, and g and h are acceleration due to gravity and height respectively. These effects are neglected in the modified Bernoulli equation:

$$P = \frac{1}{2}\rho v^2 \tag{2A}$$

P_a and $\rho g h$ are both time-invariant and therefore:

$$\frac{d}{dt}(P_a) = 0 \quad \text{and} \quad \frac{d}{dt}(\rho g h) = 0 \tag{3A}$$

Thus

$$\frac{dP}{dt} = \frac{d}{dt}\left(\frac{1}{2}\rho v^2\right) \tag{4A}$$

With T being the time until peak velocity, V_p:

$$\text{PLV}\frac{dP}{dt} \approx \frac{\frac{1}{2}\rho V_p^2}{T} \tag{5A}$$

Thus

$$\text{PLV}\frac{dP}{dt} \propto \frac{V_p^2}{T} \tag{6A}$$

APPENDIX B: WAVE TRANSMISSION MODEL

As described in Appendix C, the wave transmission model is based upon the following equation of continuity:

$$\frac{dP}{dt} = \rho \cdot V_{pw}^2 \cdot \frac{dV}{dx} \tag{1B}$$

It should be noted that:

$$V_{pw}^2 = \left(\frac{dx}{dt}\right)^2 \tag{2B}$$

The propagation of dx/dt, which is pulse wave velocity, V_{pw}, is illustrated in Fig. 3. Substituting (2B) into (1B) yields:

$$\frac{dP}{dt} = \rho \cdot \left(\frac{dx}{dt}\right)^2 \cdot \frac{dV}{dx} = \rho \cdot \left(\frac{dx}{dt}\right) \cdot \frac{dV}{dt} \tag{3B}$$

Thus

$$\frac{dP}{dt} = \rho \cdot V_{pw} \cdot \frac{dV}{dt} \tag{4B}$$

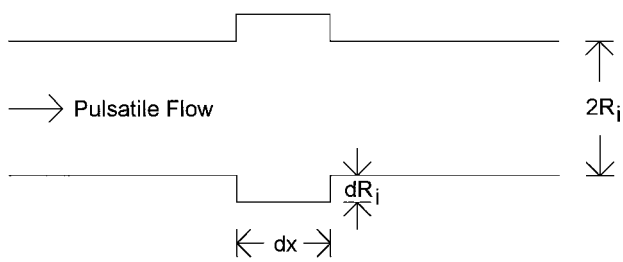

Figure 3. Pulsatile flow, in a distensible vessel, generates a pulse wave dx. The velocity of the pulse wave, V_{pw}, is equal to dx/dt and is a function of the stiffness of the vessel and its geometry (Milnor, 1982).

V_{pw} has been described, based on physical parameters, with the Moens–Korteweg equation:

$$V_{pw} = \sqrt{\frac{E \cdot h}{2 \cdot \rho \cdot R_i}} \tag{5B}$$

where E represents Young's modulus which increases as vessel wall stiffness increases. The width of the vessel wall is denoted as h and its internal radius is R_i.

The Moens–Korteweg equation (5B) can also be expressed based on compliance $C = \frac{d(\text{Vol})}{dP}$ and total vessel volume (Vol):

$$V_{pw} = \sqrt{\frac{dP \cdot (\text{Vol})}{\rho \cdot d(\text{Vol})}} = \sqrt{\frac{(\text{Vol})}{\rho \cdot C}} \tag{6B}$$

Thus, pulse wave velocity is inversely proportional to the square root of compliance (Milnor, 1982).

APPENDIX C: DERIVATION OF THE EQUATION OF CONTINUITY

Utilizing Fig. 3, the equation of continuity, Eq. (1B), can be derived (Milnor, 1982) based upon the assumption that:

$$\frac{dQ}{dx}|_{\text{radial}} = \frac{dQ}{dx}|_{\text{axial}} \tag{1C}$$

$$\frac{dQ}{dx}|_{\text{radial}} = \frac{\left(\frac{d(\text{vol})}{dt}\right)}{dx} = \frac{d}{dt}\left(\pi R_i^2\right) \tag{2C}$$

Thus

$$\frac{dQ}{dx}|_{\text{radial}} = 2\pi R_i \frac{dR_i}{dt} \tag{3C}$$

For flow in the axial direction, $Q = (\text{area}) \cdot (\text{velocity})$ and therefore:

$$\frac{dQ}{dx}|_{\text{axial}} = \left(\pi R_i^2\right) \frac{dV}{dx} \tag{4C}$$

Equations (3C) and (4C) can be substituted into (1C) to yield:

$$2\pi R_i \frac{dR_i}{dt} = \left(\pi R_i^2\right) \frac{dV}{dx} \tag{5C}$$

Rearranging

$$\frac{dR_i}{dt} = \frac{R_i}{2} \cdot \frac{dV}{dx} \tag{6C}$$

Young's modulus is defined as $E = \frac{\text{stress}}{\text{strain}}$, where strain $= \frac{\frac{dR_i}{R_i}}{}$. Stress can then be defined as:

$$\text{stress} = \frac{R_i \cdot dP}{h} \tag{7C}$$

where h represents vessel wall thickness.

By substitution, Young's modulus is then:

$$E = \frac{R_i \cdot dP}{h} \cdot \frac{R_i}{dR_i} \tag{8C}$$

Rearranging, dR_i can then be expressed as:

$$dR_i = \frac{R_i \cdot dP}{h} \cdot \frac{R_i}{E} \tag{9C}$$

Substituting Eq. (9C) into Eq. (6C) yields:

$$\frac{\left(\frac{R_i \cdot dP}{h} \cdot \frac{R_i}{E}\right)}{dt} = \frac{R_i}{2} \cdot \frac{dV}{dx} \tag{10C}$$

Rearranging Eq. (10C) yields:

$$\frac{dP}{dt} = \frac{E \cdot h}{2 \cdot R_i} \cdot \frac{dV}{dx} \tag{11C}$$

The following is the Moens–Korteweg equation for pulse wave velocity:

$$\left(\frac{dx}{dt}\right)^2 = V_{pw}^2 = \frac{E \cdot h}{2 \cdot \rho \cdot R_i} \tag{12C}$$

The equation of continuity can be expressed by substituting Eq. (12C) into (11C):

$$\frac{dP}{dt} = \rho \cdot V_{pw}^2 \cdot \frac{dV}{dx} \tag{13C}$$

REFERENCES

Atlas G and Mort T. Placement of the esophageal Doppler ultrasound monitor probe in awake patients. *Chest* 119: 319, 2001.

Bargiggia GS, Bertucci C, Recusani F, Raisaro A, de Servi S, Valdes-Cruz LM, Sahn DJ, and Tronconi L. A new method for estimating left ventricular dP/dt by continuous wave Doppler-echocardiography. Validation studies at cardiac catheterization. *Circulation* 80: 1287–1292, 1989.

Blacher J, Asmar R, Djane S, London GM, and Safar ME. Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients. *Hypertension* 35: 1111–1117, 1999a.

Blacher J, Guerin AP, Pannier B, Marchais SJ, Safar ME, and London GM. Impact of aortic stiffness on survival in end-stage renal disease. *Circulation* 99: 2434–2439, 1999b.

Bulpitt CJ, Rajkumar C, and Cameron JD. Vascular compliance as a measure of biological age. *J Am Geriatr Soc* 47: 657–663, 1999.

Carney WI, Rheinlander HF, and Cleveland RJ. Control of acute aortic dissection. *Surgery* 78: 114–120, 1975.

Groenink M, de Roos A, Mulder BJM, Spaan JAE, and van der Wall EE. Changes in aortic distensibility and pulse wave velocity assessed with magnetic resonance imaging following beta-blocker therapy in the Marfan syndrome. *Am J Cardiol* 82: 203–208, 1998.

Grubb BP, Sirio C, and Zelis R. Intravenous labetalol in acute aortic dissection. *JAMA* 258: 78–79, 1987.

Hopkins KD, Lehmann ED, and Gosling RG. Aortic compliance measurements: A non-invasive indicator of atherosclerosis? *Lancet* 343: 1447, 1994.

Hunt AC, Chow SL, Escaned J, Perry RA, Seth A, and Shiu MF. Evaluation of a theoretical Doppler index to noninvasively estimate peak dP/dt using continuous wave Doppler ultrasound of ascending aortic flow in man. *Cathet Cardiovasc Diagn* 23: 219–222, 1991.

Kraft KA, Itskovich VV, and Fei DY. Rapid measurement of aortic wave velocity: In vivo evaluation. *Magan Reson Med* 46: 95–102, 2001.

Lehmann ED. Clinical value of aortic pulse-wave velocity measurement. *Lancet* 354: 528–529, 1999.

Lehmann ED, Hopkins KD, Rawesh A, Joseph RC, Kongola K, Coppack SW, and Gosling RG. Relation between number of cardiovascular risk factors/events and noninvasive Doppler ultrasound assessments of aortic compliance. *Hypertension* 32: 565–569, 1998.

Meaume S, Rudnichi A, Lynch A, Bussy C, Sebban C, Benetos A, and Safar ME. Aortic pulse wave velocity as a marker of cardiovascular disease in subjects over 70 years old. *J Hypertens* 19: 871–877, 2001.

Milnor WR. *Hemodynamics*. Baltimore, MD: Williams & Wilkens, 1982.

Mohiaddin RH, Firmin DN, and Longmore DB. Age-related changes of human aortic flow wave velocity measured noninvasively by magnetic resonance imaging. *J Appl Physiol* 74: 492–497, 1993.

O'Conner B and Luntley JB. Acute dissection of the thoracic aorta. Esmolol is safer than and as effecive as labetalol. *Br Med J* 310: 875, 1995.

Prokop EK, Palmer RF, and Wheat MW. Hydrodynamic forces in dissecting aneurysms. *Circ Res* 27: 121–127, 1970.

Rogers WJ, Hu YL, Coast D, Vido DA, Kramer CM, Pyretiz RE, and Reichek N. Age-associated changes in regional aortic pulse wave velocity. *J Am Coll Cardiol* 38: 1123–1129, 2001.

Saeian K, Wann LS, and Sagar KB. Doppler echocardiographic evaluation of left ventricular function. *Echocardiography* 7: 21–25, 1990.

Schertel ER. Assessment of left-ventricular function. *Thorac Cardiovasc Surg* 46(Suppl 2): 248–254, 1998.

Senda S, Sugawara M, Matsumoto Y, Kan T, and Matsuo H. A noninvasive method of measuring Max(dP/dt) of the left ventricle by Doppler echocardiography. *J Biomech Eng* 114: 15–19, 1992.

Singer M. Esophageal Doppler monitoring of aortic blood flow: Beat by beat cardiac output monitoring. *Int Anesthesiol Clin* 31: 99–125, 1993.

Sugawara M, Senda S, Katayama H, Masugata H, Nishiya T, and Matsuo H. Noninvasive estimation of left ventricular Max(dP/dt) from aortic flow acceleration and pulse wave velocity. *Echocardiography* 11: 377–384, 1994.