opinions/hypotheses

Hemodynamic Monitoring Utilizing Transesophageal Echocardiography*

The Relationships Among Pressure, Flow, and Function

Jan I. Poelaert, MD, PhD, FCCP; and Guido Schüpfer, MD


Key words: echocardiography; hemodynamic; left ventricular function; monitoring

Abbreviations: A wave = late-filling wave; CW = continuous-wave; +dP/dtmax = positive maximum first derivative of pressure, corrected for time; Ea = arterial elastance; E/A = ratio of the early and rapid-filling wave to the late-filling wave; E wave = early and rapid-filling wave; LA = left atrium atrial; LAP = left atrial pressure; LV = left ventricle/ventricular; LVEDA = left ventricular end-diastolic area; LVEDP = left ventricular end-diastolic pressure; LVEDV = left ventricular end-diastolic volume; PCWP = pulmonary capillary wedge pressure; PW = pulsed-wave; PWR = cardiac power; RV = right ventricle/ventricular; RVOT = right ventricular outflow tract; SV = stroke volume; TDI = tissue Doppler imaging; TEE = transesophageal echocardiography; VTI = velocity-time integral

Cardiac imaging is an important cornerstone in decision making and management in cardiovascular medicine. Echocardiography and Doppler ultrasonography developed as the main and most commonly used bedside imaging techniques. Transthoracic echocardiography is an invaluable imaging technique with respect to the diagnosis of cardiovascular disease. The impact of echocardiography on the change in the therapy and management of hospitalized patients was found to be as high as 57%, although changes occurred more frequently in ICU patients compared to those admitted to the hospital (54% vs 37%, respectively).1,2 Nevertheless, the transthoracic approach for echocardiography has certain specific limitations regarding the visualization of different cardiac structures and great vessels. Table 1 demonstrates in detail the potential advantages and shortcomings of both techniques. Transesophageal echocardiography (TEE) with Doppler imaging has opened a completely different window, and has become a fascinating and appealing tool, not only for diagnosis in routine cardiology practice3 but also for perioperative hemodynamic monitoring, having a decisive impact on surgical management. Moreover, owing to its bedside availability, TEE facilitates the routine diagnosis and management of cardiovascular failure both intraoperatively and in ICU patients.4

Circulatory failure of cardiac origin in adults often is due to ventricular failure. More than 50% of all patients who are scheduled for surgery or are admitted to the ICU have experienced cardiac problems. Impaired cardiac function, which is often seen in critically ill patients, is mostly evidenced as pump failure. Several etiologic factors of pump dysfunction (eg, ischemia, septic cardiomyopathy, inflammatory responses, right ventricular (RV) failure in conjunction with pulmonary hypertension, or a combination of several factors) have been well-studied during the last decades. Left ventricular (LV) function is a determinative parameter in various diseases, although the role of the RV in critically ill patients should not be underestimated.5 Therefore, the clinical evaluation of cardiac function must pertain to the assessment of the LV and the RV with respect to increased morbidity and mortality. Crucial in the discussion is the early detection of LV systolic and diastolic dysfunction by sensitive monitors, in partic-

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This research was supported in part with a grant from the International Research Centre, Gent, Belgium. Jan Poelaert was supported by a grant from the Research Fund “Bijzonder Onderzoeksfonds” (No. B/03719) of Ghent University, Gent, Belgium (2002–2003).

Manuscript received January 9, 2004; revision accepted August 24, 2004.

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Several reviews\textsuperscript{6–8} already have stressed the function, namely, contractility, preload, and afterload, as well as determinants of ventricular filling and volumes. Nevertheless, pressures and flow rates are physiologically relevant determinants of ventricular function. To describe the hemodynamic status of a patient, most clinicians assess the independence of loading conditions. To describe the hemodynamic status of a patient, most clinicians routinely employ an analysis of pressures rather than volumes and flow rates. To obtain relevant measurements of change, Doppler echocardiography has to be used. When an ultrasound beam strikes a moving object (e.g., a moving RBC), the frequency of the reflected sound is altered. This phenomenon is known as the Doppler shift (Christian Doppler, 1842). Doppler echocardiography shows the velocity data, which are derived from the measured frequency shift, and illustrates them as a spectral display. Movements of RBCs, which are moving toward the transducer, are displayed above the zero line, and negative signals display the movements of the RBCs away from the transducer. The following general principles have to be kept in mind when obtaining and interpreting Doppler signals and waveforms:\textsuperscript{9}:

1. The Doppler beam must be aligned with the estimated direction of blood flow. The intercept angle should be $<20^\circ$. Larger angles induce an unacceptable error. To correct this error, the cosine of the intercept angle must be taken into account in calculating the true Doppler velocity. At an angle of $60^\circ$, the transmitted velocity will be half that the true velocity, which is important data in clinical practice.

2. Continuous-wave (CW) Doppler ultrasound must be used whenever high velocities are present. Pulsed-wave (PW) Doppler ultrasound can induce an underestimation of flow velocities. This is due to the physical characteristics of both techniques. CW Doppler (with one transmitter and one receiver crystal) records all velocities in the beam axis without a limitation in analyzing high flow velocities. Nevertheless, CW Doppler lacks the spatial resolution needed to estimate the correct depth at which the measurement is obtained. PW Doppler ultrasound is characterized by a crystal, which sends and receives consecutively, considerably retarding the process of the measurement of Doppler velocities. This allows a precise determination of the location of the source of the frequency shift but immediately limits also the range of velocity, which can be measured. The latter can be explained as follows:

- The depth of the sampling gate. The further away the sampling place (interrogated target), the longer the waiting time because of the consecutiveness of action of both the transmitter crystals and receiver crystals.

### Table 1—Indications of Transthoracic Echocardiography and TEE

<table>
<thead>
<tr>
<th>Transthoracic Echocardiography</th>
<th>TEE</th>
</tr>
</thead>
<tbody>
<tr>
<td>General screening—overall assessment</td>
<td>Hemodynamic instability</td>
</tr>
<tr>
<td>Hemodynamic evaluation</td>
<td>Echocardiography when ventilated patient (also when prone ventilation)</td>
</tr>
<tr>
<td>Exclusion of tamponade</td>
<td>Tamponade (local)</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>Cardiac surgery: intraoperative and postoperative hemodynamic monitoring</td>
</tr>
<tr>
<td>Acute RV overload (pulmonary embolism, pulmonary hypertension)</td>
<td>Noncardiac surgery: intraoperative monitoring</td>
</tr>
<tr>
<td>Thoracic trauma (patient not ventilated)</td>
<td>Thoracic trauma (in a ventilated patient)</td>
</tr>
<tr>
<td>Contraindications for TEE</td>
<td>Diagnosis/exclusion of thoracic aortic dissection; diagnosis/exclusion of endocarditis; exclusion of left atrial appendage thrombi</td>
</tr>
</tbody>
</table>

LV systolic performance is governed by the following three major determinants: (1) the Frank-Starling mechanism, necessitating the measurement of pressures and volumes; (2) contractility, independent of loading conditions; and (3) heart rate. An assessment of ventricular contractility must include the independence of loading conditions. To describe the hemodynamic status of a patient, most clinicians routinely employ an analysis of pressures rather than a determination of volumes. Nevertheless, pressures only provide a rudimentary approximation of the physiologically relevant determinants of ventricular function, namely, contractility, preload, and afterload. Several reviews\textsuperscript{6–8} already have stressed the elegance of how TEE provides data in this respect. Clinically, pressure and flow appear to be the most important features. From these parameters, numerous variables have been derived describing more precisely hemodynamics of the (left) heart and the peripheral circulation. The interplay between pressure and flow is an important characteristic of cardiac function and the dynamics of the circulation that is more precise than one parameter alone. The relationship between flow estimated with echocardiography and related pressure measurements and the energy generated by the LV pumping blood into the circulation is the subject of this review.
• The PW Doppler ultrasound signal is obtained at the rate of the pulse repetition frequency, which is approaching the frequency shift of the Doppler effect.

• With respect to Doppler physics, the transducer must sample twice as fast with a 5-MHz probe as with a 2.5-MHz probe to measure the same Doppler shift. Hence, at the same pulse repetition frequency the maximum recordable Doppler velocity with a 2.5-MHz probe is twice the recordable velocity with a 5-MHz probe. This phenomenon implies that a 5-MHz probe will scan more superficial layers of tissue than a 2.5-MHz probe.

3. The sample volume has to be placed in the middle of the vessel in order to diminish potential turbulence.

4. PW Doppler echocardiography is not useful whenever a pressure gradient across a valve is present or a subvalvular stenosis has to be assessed. In both circumstances, CW Doppler echocardiography must be utilized.

Echocardiography is not only an established tool for the assessment of global ventricular function, but appears a well-recognized technique in the evaluation of regional ventricular function in patients with potential cardiac disease. For many years, routine echocardiography allowed only the visual analysis of a regional dysfunction. The interpretation of myocardial thickening and endocardial motion requires an experienced observer, and the method is subjective and not useful in research. Color-coded tissue Doppler imaging (TDI) was introduced a few years ago, permitting the assessment of low-velocity Doppler high-amplitude shifts and the real-time display of color-coded tissue velocities. This technique enables the noninvasive delineation of myocardial velocities in a noninvasive manner in the human heart at the level of a segment of the myocardial wall or the mitral annulus. In addition, both diastolic function and regional systolic function parameters can be derived from the myocardial wall motion Doppler pattern.

Left-Sided Filling Pressures

Echocardiography permits the indirect estimation of LV filling pressures in various ways, utilizing both Doppler echocardiography and two-dimensional echocardiography. In patients with normal cardiac function, LV end-diastolic pressure (LVEDP) is directly related to LV end-diastolic volume (LVEDV). Clinically, in patients with decreased ventricular compliance, however, the relationship between pressure and volume is difficult to determine, in particular when myocardial ischemia or tamponade occur, although, in theory, pressure-volume loops will allow the determination of LV compliance. The direct assessment of ventricular volumes to estimate preload is superior to measurements of pressures because of the important interference by ventricular compliance. Numerous factors interfere with this relationship, such as altering compliance during myocardial ischemia, inotropic stimulation, and altered intrathoracic pressures.

Utilizing two-dimensional echocardiography, the position of the interatrial septum and the curvature reflects the relationship between right and left atrial pressures (LAPs). Hence, relative pressure differences between the right and left atrium can be detected. In addition, the filling status can be roughly estimated. In particular, the movement of the interatrial septum during the cardiac cycle suggests pressure differences between the right and left atrium. In patients with hypovolemia, this movement is increased. The estimation of RV and LV preload by means of the position of the interatrial septum cannot be used in the following situations:

• The presence of tricuspid regurgitation with a consequent shift of the septum toward the left atrium;
• Acute mitral regurgitation, in which an increased septal movement is present; and
• Mitral stenosis or chronic mitral regurgitation, as in severe LV failure, in which the septal amplitude is decreased. This is important information as it provides insight into the duration of a diagnosed cardiac disease.

Doppler echocardiography provides a good estimate of left-sided filling pressures using the transmural flow pattern, the peak flow regurgitation flow velocity, the pulmonary venous flow pattern, and myocardial Doppler imaging. The transmural flow, obtained in a four-chamber view, is directly related to the filling of the LV and is governed by the transmural pressure gradient. The normal filling pattern is displayed as a biphasic tracing with an early and rapid-filling wave (E wave), followed by a diasstasis period with minimal or no flow (Fig 1). Finally, a late-filling wave (A wave) follows, caused by the atrial contraction. Both velocities of the respective flow waves and corresponding time velocity integrals are as important characteristics of LV filling. Another easily calculated parameter is the ratio of the peak flow velocities (ie, the ratio of E wave to A wave [E/A]). A normal flow velocity ratio is in the range of 0.75 to 1.40. The E/A ratio is strongly age-dependent. With increasing age, the E-wave velocity diminishes in favor of the late-filling A wave. It is important to remark that the various
Doppler velocities are the resulting effect of several physiologic characteristics, such as LV compliance and relaxation, left atrial (LA) compliance, mitral valve area, and LA pressure (LAP). In addition, mechanical ventilation as well as external constraints (thrombi in the pericardial sac, stiffness of the pericardium, or high positive end-expiratory pressure ventilation with potential cardiac compression by the lungs) affects LV filling. Owing to these various interfering factors, it is conceivable that there is no correlation between the early filling velocity and the left-sided filling pressure (ie, LAP). As long as LAP is low in patients with dilated cardiomyopathy, the E-wave velocity, as well as the E/A ratio, will be low. With the progression of heart failure in these patients, the E-wave velocity will increase, and the E/A ratio will be > 1. The higher E-wave velocity will have a shorter deceleration time, reflecting decreased ventricular compliance, making an evolution toward a restrictive pattern (E/A) a very stiff LV with a rapid and important rise of ventricular pressures with small amounts of fluid.

When present, mitral regurgitation can also be used to estimate LV filling. Doppler echocardiography permits the estimation of the LAP using the modified Bernoulli equation, as described in the following formula:

\[
\text{LAP} = \text{RRao} - (4 \times V^2_{\text{TMF}})
\]

in which RRao is the systolic aortic pressure, and \( V^2_{\text{TMF}} \) is the transmitral peak regurgitant flow velocity. This method is based on the assumption there is no pressure gradient between the LV and the ascending aorta.

The pulmonary venous flow pattern (Fig 2) is a third possibility for estimating left-sided filling pressures. The systolic flow wave is frequently biphasic whenever a low filling status is present. The change in systolic flow velocity from the pulmonary vein is directly related to changes in cardiac output. The ratio of the systolic time-velocity integral and the sum of both systolic and diastolic time-velocity integrals correlates most strongly \((r = -0.88)\) whenever pulmonary venous flow wave duration (the a wave) exceeds the transmitral flow wave. This is usually thought to be a sign of adequate filling pressures. A systolic/diastolic flow velocity ratio of < 0.4 reflects the markedly increased ventricular filling pressures. This is mainly due to reduced LA compliance. This study also shows that LVEDP is the main determinant of the systolic velocity of the pulmonary vein Doppler pattern (regression analysis). A minor interfering factor is the systolic ventricular function. To estimate the LVEDP, a pulmonary venous Doppler pattern obtained at one of the inlet orifices of the pulmonary veins should help. A ratio of the systolic and diastolic flow wave velocities of < 0.4 is suggestive of an increased LVEDP. Another important feature is the ratio of the duration of the atrial reverse flow wave at the level of the pulmonary veins and the atrial contraction wave at the level of the mitral valve. The difference between the duration of the atrial reversal flow and the duration of the atrial inflow wave is independent of age and thus may be used as a reliable index of LVEDP, even in elderly patients. A reverse a wave duration that exceeds the duration of the atrial inflow wave predicts an LVEDP of > 15 mm Hg. No correlation was found between this index and the LAP, suggesting that this index is a measure of late diastolic LV compliance. A significant relationship was seen between the systolic pulmonary vein Doppler velocity-time integral (VTI, ie, the area under the curve of a Doppler signal) and the atrial wave VTI.
suggesting a relation between atrial filling and emptying. LVEDP is the main determinant of the systolic velocity of the pulmonary vein Doppler pattern.\textsuperscript{18,26} In clinical practice, all three Doppler methods will be used consecutively.

The systolic forward flow wave in the pulmonary vein Doppler pattern is preceded by a small reverse atrial contraction wave. The systolic forward flow wave sometimes shows two peaks, which are related to the atrial relaxation and the mitral annular descent, respectively.\textsuperscript{29} The systolic flow wave is followed by a diastolic flow wave. The pulmonary capillary wedge pressure (PCWP) correlates best with the atrial reverse flow wave velocity \( (r = 0.81) \).\textsuperscript{25} In a study of patients who had experienced acute myocardial infarction, it was shown that PCWP had a strong negative relationship with the deceleration time of the pulmonary venous Doppler pattern in diastole \( (r = -0.89) \), with a sensitivity and specificity of this measure of \( < 160 \text{ ms} \) in predicting a PCWP of \( > 18 \text{ mm Hg} \) of 97% and 96%, respectively.\textsuperscript{29}

In 1998, Nagueh et al\textsuperscript{30} demonstrated that left-sided filling pressures can also be estimated fairly accurately by utilizing Doppler myocardial imaging \( (\text{ie, TDI}) \). TDI assesses velocities of the moving myocardium and, hence, detects the phase shift of the ultrasound signals reflected by myocardial tissues. Velocities are much lower \( (\text{ie,} < 10 \text{ to } 15 \text{ cm/s}) \) than the blood flow velocities. The amplitude of the reflected ultrasound wave is higher \( (40 \text{ decibels}) \). These two major changes make important adjustments necessary in interpreting the results.

Therefore, TDI works with a high-pass filter, allowing the low velocities to be measured. The Doppler settings are adjusted for a Nyquist limit of 20 cm/s with the lowest wall filter and a minimum gain setting. Furthermore, TDI has some beneficial attributes, improving its power to be used in critically ill patients as follows: TDI is based on frequency shift rather than signal amplitude; TDI uses lower transmitting frequencies, permitting better tissue penetration; and TDI has favorable temporal resolution.

The early diastolic velocity measured with TDI \( (\text{ie, arterial elastance [Ea]}) \) behaves as a relative load-independent index of LV relaxation. The ratio of the E wave to Ea showed the strongest relation to PCWP \( (r = 0.86) \), irrespective of the pattern and the ejection fraction.\textsuperscript{31} Whereas the E wave of the transmitral flow pattern is load-sensitive, the Ea, obtained by TDI, behaves as a relatively load-independent index of ventricular relaxation. In 100 patients, a relation was described between the E wave/Ea ratio and the PCWP \( (r = 0.86) \), irrespective of the pattern and ejection fraction,\textsuperscript{30} although this has not been confirmed.\textsuperscript{32}

### Estimation of Preload

TEE permits good qualitative and quantitative parameters to estimate preloading conditions in patients with either normal ventricular function or dysfunction. The short-axis view is in this respect of the utmost importance. In an adequately sedated ICU patient, the presence of a hyperdynamic ventricle in the absence of inotropic drugs could be a sign of hypovolemia.\textsuperscript{33} In addition, the presence of kissing walls, with a potential, nearly complete obliteration of the outflow tract, is very suggestive of a low filling status.\textsuperscript{33}

LV volume assessment is done by tracing the end-diastolic endocardial border in a mid-esophageal long-axis view in a longitudinal plane.\textsuperscript{34} The software used to calculate the volume in the echocardiograph uses the methods of discs (the Simpson rule), which is an extremely well-validated technique, providing very accurate data both in adults and children.\textsuperscript{35} A quantitative estimation can be performed in different ways using two-dimensional echocardiography. The LV end-diastolic area (LVEDA), measured per definition at the midpapillary level of the LV, correlates well with volumetric analogues.\textsuperscript{36} In particular, the changes in LVEDA closely resemble the changes in LVEDV. Very suggestive for low filling status of the LV is the LVEDA index, referring to a body surface area of \( < 5.5 \text{ cm}^2/\text{m}^2 \).\textsuperscript{37} Other authors described in patients with normal LV function during graded hypovolemia a linear decline of LVEDA of 0.3 cm\(^2\) per percentage blood loss.\textsuperscript{38,39} A good correlation \( (r = 0.87) \) was found between changes in the LVEDA index and intrathoracic blood volume, which was measured with the dye dilution technique in postoperative cardiac surgical patients, although neither parameter (evidently) correlated with PCWP.\textsuperscript{40}

Several shortcomings and limitations of LVEDA have to be recognized. When regional wall motion abnormalities are present, the correct use of LVEDA as a preload parameter is limited. Furthermore, the apical region of the LV is more susceptible to regional wall motion abnormalities than the base of the heart.\textsuperscript{41} In this respect, the above-mentioned Doppler echocardiographic methods and the respiration-induced variation in maximal Doppler velocities must be appreciated.\textsuperscript{42} Echocardiographic data from a single plane seldom provide information about filling status.

Besides using the short-axis view to obtain the LVEDA, a midesophageal view allows the scanning of the long axis of the LV. Volume determination of the LV can be performed utilizing the Simpson rule. The LV is divided into 20 ellipsoid disks of equal heights but different diameters. The sum of the
respective volumes of the slices allows the calculation of the total ventricular volume. This technique has been validated against angiography.

Over the last years, the term fluid responsiveness has been proposed as an important estimate of the optimization of preload (Fig 3). Using methods that are analogous to systolic respiratory pressure variation and stroke volume variation, Slama et al demonstrated the value of flow measurements across the aortic valve in the assessment of flow velocity variation with cyclic altering of intrathoracic pressures. The VTI at that level decreased progressively, in close relationship with a graded and controlled blood loss performed in an animal experimental setting. Respiratory variations of VTI are a sensitive index of fluid responsiveness, concurring completely with the findings of systolic pressure variation, stroke volume (SV) variation, and pulse pressure variation.

FLOW AND FUNCTION

Multiple indexes have been proposed to describe global ventricular function. Both load-dependent and load-independent parameters have been discerned. Ejection fraction is a classic example of a load-dependent description of global ventricular function. Nevertheless, in view of the discussion on flow and function, ejection fraction or, in echocardiographic terms, fractional area contraction will not be disputed.

With TEE and Doppler echocardiography, it is relatively easy to estimate swiftly the load-dependent characteristics as SV, cardiac output, and positive maximum first derivative of pressure, corrected for time (+dP/dtmax). The assessment of SV includes both the measurement of the flow and the determination of the area through which this flow moves forward. The area through which this flow is propagated is one aspect of the determination of the stroke volume. Either the diameter of a certain location or, preferably, the effective time-averaged surface area must be measured. Several localizations have been used, as follows: RV outflow tract (RVOT) and pulmonary artery; mitral valve; and LV outflow tract at the level of the aortic valve. It is clear from earlier studies that the diameter of the pulmonary artery is sometimes difficult to measure, explaining the lower correlation coefficient. Nevertheless, placing the sample volume in the position at the level of the midpulmonary artery is an easy and elegant method for assessing SV. Some training

![Figure 3. Clinical approach of hemodynamic monitoring. Art.syst. = arterial systolic; CO = cardiac output; PHT = pulmonary hypertension; LCO = low cardiac output; SVO₂ = mixed venous oxygen saturation. Pulm. = pulmonary.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/22020/ on 05/23/2017)
permits even the assessment of SV in the RVOT from a deep transgastric view.\textsuperscript{54} The diameter of the mitral valve is difficult to measure as the annulus is not circular and changes throughout the cardiac cycle, not permitting the use of the mitral valve for measuring SV.\textsuperscript{55} The measurement of the flow across the aortic valve is a third possibility.\textsuperscript{56} To permit the measurement of the diameter of the aortic valve, a longitudinal midesophageal image must be obtained (Fig 4). It has been demonstrated that the direct measurement of the effective time-averaged aortic area allows for superb accuracy.\textsuperscript{50,51} This method can be performed with the transducer at the midesophageal position, rotating the multiplane probe toward 25° to 40° (Fig 5). In routine clinical practice, however, the method including the measurement of the diameter is more easily performed with a lower, albeit acceptable, precision.\textsuperscript{52,57–59}

The flow is obtained by the measurement of the area under the curve of a Doppler wave. This gives information on the VTI (in centimeters) [Fig 6]. VTI provides the distance that an RBC is projected forward during one cardiac cycle and is therefore directly related to the systolic function of the LV or RV, depending on the place of the sample volume.

Hence, the SV can be calculated from the following different formulas:

\[
SV = \frac{VTI}{AVA}
\]

when the method of the effective time-averaged aortic valve area (AVA) is chosen. This method provides higher accuracy.

\[
SV = \frac{VTI \times 0.78 \times D^2}{AVA}
\]

when the method of measurement of the diameter (D) is preferred. In clinical practice, however, the method of measurement of the diameter at the level of the pulmonary artery or the aortic valve provides an adequate estimation of the SV.

When aortic stenosis is present, the CW Doppler signal shows a characteristic image with two densities of flow.\textsuperscript{60} The VTI of the most intense part represents the SV, whereas the external contour shows the peak velocity, which permits the calculation of the aortic valve gradient utilizing the modified Bernoulli
equation. This technique cannot be used when significant aortic regurgitation is present.

The technique of flow calculation is also utilized when intracardiac shunts are present (eg, atrial septal defect). The VTI calculated from in the RVOT (including the measurement of the diameter of the pulmonary annulus) can be compared with VTI obtained from flow analysis in the LV outflow tract, in order to calculate the flow through the shunt.61

\[ +\frac{dP}{dt_{\text{max}}} \]

is another flow-derived, load dependent parameter to circumscribe global left ventricular function (Fig 6).62 The estimation of \( +\frac{dP}{dt_{\text{max}}} \), as the mean rate of pressure rise, with Doppler echocardiography obliges a leakage of the mitral or aortic valve. At the mitral valve, \( +\frac{dP}{dt_{\text{max}}} \) is calculated using the modified Bernoulli equation.63 If the change pressure gradient is measured from the ascending limb of the mitral regurgitant flow wave between the velocity levels of eg 1 m/s and 3 m/s (Fig 7), change in ventriculo-atrial pressure gradient is calculated as 4(1)^2 + 4(3)^2 = 32. A change corresponding to the left ventriculo-atrial pressure gradient of 4 and 36 mm Hg, is measured with Doppler echocardiography. With this method, it is assumed that any gradient across the mitral valve is absent. A normal value of \( +\frac{dP}{dt_{\text{max}}} \) is between 800 to 1,200 mm Hg/s. Analogous measurements can be performed from the descending limb of the aortic regurgitation flow wave in a deep transgastric transverse view utilizing CW Doppler providing Doppler-derived \( \frac{dP}{dt_{\text{max}}} \).64 Although easy applicable, the Doppler technique may underestimate the true maximum \( \frac{dP}{dt} \).

PRESSURE, FLOW, AND FUNCTION

In clinical practice, the arterial pressure and morphology of the tracing are the mainstays of hemodynamic monitoring. Although this tracing offers considerable information about SV, contractility, and loading conditions of the left heart, all information is mainly based on eyeballing. Many investigators searched for other more revealing variables, which could be, however, be more difficult to obtain. Many years ago, LV maximal elastance, the line connecting all end-systolic points in a pressure-volume diagram, was considered for use.65 Although significant improvements have been proposed to upgrade this variable to a more clinically useful parameter,66 several drawbacks have to be recognized, diminishing considerably the usefulness of this technique in clinical practice.

Another, more clinical, parameter is derived from hydraulic energy. Hydraulic power is based on the fact that the heart is a pump that is used to circulate the blood into both the pulmonary and systemic circulation. The cardiac muscle provides the energy necessary for this circulation, dispersing more energy as the blood proceeds deeper into the smaller vessels. The effort performed by the ventricle to pump the blood against gravity and to overcome the inertia of the blood is nothing else than ventricular work. Cardiac hydraulic power output, the work per time unit, is the product of cardiac flow output and its pressure delivered in the arterial system.67–70 It increases proportionally to the exercise workload performed and represents the rate at which the ventricle performs external work.71 Cardiac power (PWR) provides the best representation of the performance obtained in a single cardiac cycle to counterbalance the demand imposed by the metabolizing tissues on the cardiac pump. In patients with cardiac failure, the measurement of PWR at rest and after the administration of positive inotropic stimulation provides insight into the cardiac energetic reserve.72

In the absence of mitral regurgitation, the ventricular volume change during systole equals aortic volumetric flow. Hence, PWR can be described as follows:

\[ \text{PWR} = \text{PLV} \times \text{FAO} \]

where PLV is instantaneous LV pressure and FAO is instantaneous aortic flow. The product reaches its maximum after the attainment of the peak flow and before peak aortic pressure is obtained.8 At this time, the aortic and ventricular pressures are similar. Hence, the determination of the different factors, building up PWR, is in this way markedly facilitated. As the various parameters of volumetric flow are easily obtained, the previously mentioned formula can be rewritten:

\[ \text{PWR}_{\text{max}} = \text{PAO} \times \text{VAO}_{\text{max}} \times \text{AVA} \times 1.333 \times 10^{-4} \]

where PAO is instantaneous aortic pressure, VAO-
max is instantaneous maximum aortic blood flow velocity, AVA is time-averaged aortic valve area, and PWRmax is the maximum PWR (in watts). In clinical conditions, instantaneous aortic pressure is arterial BP at the time point at which the product of pressure and flow becomes is at the maximum.

The clinical attractiveness of the parameter PWR is high, taking into consideration various criteria, such as the integrity of the heart in relation to the circulation, accounting for both the pressure-generating and flow-generating capacity of the cardiac muscle in healthy and diseased hearts. In addition, PWR can be measured with a single-beat technique, without cumbersome manipulations of loading conditions, which is even useful in patients with atrial fibrillation. A close relationship was shown to exist between preload-adjusted maximal power and ventricular maximal elastance.

PWR shows great stability concerning the changes of afterload but is also highly sensitive to preload alterations. Therefore, several authors have proposed the correction of PWR with the square of the LVEDV, the LV end-diastolic diameter, or the LVEDA. A close relationship between LVEDV, as a marker of preload, and maximum PWR/LVEDA ratio, as a measure of myocardial contractility, is shown in Figure 8. This graph demonstrates clearly the higher dependence of ventricles with poor performance on preload in maintaining their output possibilities.

The ability of PWR to characterize global LV contractility is less accurate beyond physiologic pressures and volumes. This has mainly to do with the reliance on the intercept of the preload-adjusted maximal power diagram with respect to LVEDV. This implies certain limitations of the power measurement in, for instance, severely hypertensive or hypotensive patients. This method cannot be used in patients with severe mitral regurgitation or aortic valve disease.

**Consequences for Daily Practice**

Echocardiography permits a rational approach to the problem of hypotension. As stated earlier, the basic and starting view of each type of investigation (i.e., either transthoracic or transesophageal) must be a short-axis view of the LV, as this image provides information on the following three fundamental issues: (1) global contractility; (2) the presence of regional wall motion abnormalities; and (3) the first indication of volemia. If global contractility is normal, any other cause of the hypotension than the heart should be investigated (e.g., sepsis, vasoplegia, and technical problems). If hypotension is combined with a decreased global LV function, a complete echocardiogram should reveal the causes of this hemodynamic instability. In addition, some relatively easy hemodynamic features can be measured and estimated to obtain a global picture of the hemodynamics. Contractility, preload, and afterload can be readily estimated in a fairly reliable manner. In Figure 3, we have proposed a practical scheme that can be used whenever the problem of hypotension is present, and that can be worked out rapidly to allow quick and adequate management. In another study, the importance of the quick and decisive
management of hemodynamic instability and hypotension has been shown again. After the interpretation of these data, therapeutic handling can already start. For the purposes of continuous monitoring of cardiac output and mixed venous oxygen saturation, there is still time enough to insert a pulmonary artery catheter afterward.

**Conclusions**

The end points of hemodynamic management have traditionally focused on improving cardiac output. Over the last years, an analysis of the literature has shown some fascinating advances when combining Doppler flow measurements and arterial pressure waveforms. It might be speculated that modern hemodynamic management will be directed toward the enhancement of the cardiac pumping reserve, and the optimization of perfusion at the tissue and cellular levels.

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