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Cardiac ultrasound: a true haemodynamic monitor?

Jan Poelaert[1], Manu L.N.G. Malbrain[2, 3]

[1]*Department of Anaesthesiology and Perioperative Medicine, UZ Brussel, and Faculty of Medicine and Pharmacology, VUB, Brussels, Belgium*
[2]*Department of Intensive Care and High Care Burn Unit, Ziekenhuis Netwerk Antwerp, ZNA Stuivenberg, Antwerp, Belgium*
[3]*Intensive Care Unit, University Hospital Brussels (UZB), Jette, Belgium and Faculty of Medicine and Pharmacology, Free University Brussels (VUB), Brussels, Belgium*

Abstract

Cardiac ultrasound has been used in the critically ill for more than thirty years. The technology has made enormous progression with respect to image quality and quantity, various Doppler techniques, as well as connectivity, the transfer of data and offline calculations. Some consider cardiac ultrasound as the stethoscope of the Twenty-first century. The potential of eye-balling moving cardiac structures gives undeniable power to this diagnostic and monitoring tool. The main shortcoming is the discontinuous mode of monitoring and the fact that optimal information acquisition can only be obtained when one is well-trained and experienced. Cardiac ultrasound has become an indispensable tool, especially in haemodynamically unstable patients. This review summarizes some important aspects of cardiac ultrasound with use of Doppler monitoring for assessment of the three most important pillars of haemodynamics, namely cardiac preload, afterload and contractile function.

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Key words: echocardiography; cardiac ultrasound; preload; afterload; contractility; vascular resistance; cardiac output

Cardiac ultrasound has been used in the critically ill for more than thirty years. The technology has made enormous advances with respect to image quality and quantity, various Doppler techniques, as well as connectivity, the transfer of data and offline calculations. Because this technological progression, the technique has developed as an all-round and versatile tool, offering haemodynamic information at the bedside regarding major cardiac and vascular issues. Furthermore, in the critical-care setting, this tool can be utilized as a haemodynamic monitor. Whereas transthoracic echocardiography (TTE) is mostly used in ICU and postoperative patients, transoesophageal echocardiography (TEE) and Doppler is used more often intra-operatively. The latter is even so utilized to answer specific questions and as a monitoring tool in ventilated ICU patients.

No single monitoring tool can offer more information, permitting the assessment of ventricular and valvular function, flow and flow velocities and regional wall motion abnormalities, pressure gradients, and even information on intra-cardiac pressures [1]. The potential of moving cardiac structures gives undeniable power to this diagnostic and monitoring tool.

The main shortcoming is the discontinuous mode of monitoring. It goes without saying that most optimal information acquisition can only be obtained when one is well-trained and experienced not only at the level of cardiac ultrasound imaging and technology but also in the understanding (patho-)physiology of different disease states. A major difficulty of cardiac ultrasound is obtaining and recognizing the different images and structures to allow confident Doppler/2-D imaging, which permits correct estimation of pressure gradients, flows across valves or regional wall motion abnormalities. All of these requires a learning curve in order to attain a level of proficiency.

Left and right ventricular systolic function and cardiac output are determined by contractility, preload, afterload and heart rate. The present report reviews consecutively systolic function and contractility, preload and afterload as

pivotal haemodynamic variables from cardiac ultrasound in the critically ill and how these variables can be accomplished in a straightforward manner with the help of echo-Doppler techniques.

VENTRICULAR SYSTOLIC FUNCTION

Systolic function of both the left ventricle (LV) and right ventricle (RV) can be quickly assessed by eye-balling at different levels of the heart using short or long axis views. Systolic failure is the first issue to be assessed in shock patients and whenever hypotension occurs and persists, notwithstanding a rapid filling manoeuvre such as passive leg raising or the Trendelenburg position [2]. Managing unexplained hypotension is always a great challenge. In this respect, echo-Doppler is a great help: the short axis of the LV provides an interesting view in order to obtain an idea of global ventricular function, preload, regional wall motion abnormalities, left ventricular hypertrophy or pericardial fluid. Furthermore, a first impression of RV function can also be acquired.

Although the most frequently used technique, eye-balling requires a lot of experience, which can be attained only throughout lots of training. Different methods are available to assess LV systolic function easily. As most of these methods are load dependent, preload conditions should be taken into account to interpret LV systolic function correctly. The echo analogue of ejection fraction (EF) is the *fractional area contraction* (FAC), which can be estimated when end-diastolic and end-systolic area, assessed at the endocardial border at a short axis level, are taken into the following formula:

$$FAC = (LVEDA - LVESA) / LVEDA$$

Increase of preload (LVEDA) will augment fractional area contraction. A LVEDAI (LVEDA indexed for body surface area) < 5.5% cm^2 m^{-2} suggests low preloading conditions [3]. In the presence of regional wall motion abnormalities, the value of LVEDA to circumscribe preloading conditions decreases. Therefore, increased susceptibility on regional wall motion abnormalities in the apical regions against the basis of the heart is important in this respect [4].

Stroke volume (SV) is another measure to assess systolic function of the LV indirectly. It may be calculated from the following formula:

$$SV = TVI \times AVA$$

TVI, the time-velocity integral, resembles the area under the Doppler curve as a distance one erythrocyte is projected forward with one heart beat if the sample volume is set at the aortic valve cusps; while AVA, is the effective time-averaged aortic valve opening area (cm^2). Whereas blood pressure remains remarkably constant during the early phases of hypovolaemic shock, stroke volume declines are the earliest warning of compromised circulation. TVI monitoring is a handsome monitoring tool in this respect. From SV, cardiac output could be calculated. A good correlation was found between Doppler-based estimations and non-invasive uncalibrated pulse-contour assessments of cardiac output [5].

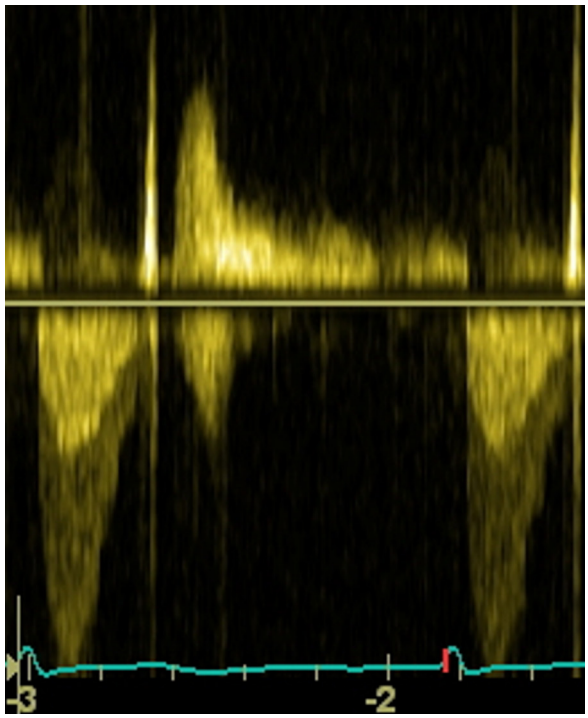

Figure 1. Double envelope of aortic flow (5 chamber view)

$$CO = SV \times HR = HR\ (bpm) \times AVA\ (cm^2) \times TVI\ (cm)$$

Doppler-based SV methodology can also be utilized when aortic valve stenosis is present. A continuous wave Doppler across the aortic valve will demonstrate a double envelope image as depicted in Figure 1. The dense Doppler signal demonstrates the stroke volume of the left ventricle, whereas the external contour of the peak Doppler signal depicts the pressure gradient, calculated from the modified Bernoulli equation:

$$\Delta p = 4 \times v^2$$

Global LV systolic function can also be circumscribed by a physiologic variable, derived from the regurgitation flow across the mitral valve, assuming no gradient across the mitral valve. The DP/dt max is assessed during catheterization and provides a flow-derived, load-dependent descriptor of global systolic LV function. The continuous wave (CW) Doppler signal of a flow wave depicting the regurgitation flow into the left atrium could be analyzed as a pressure change in time (dP/dt mean), utilizing the

modified Bernoulli equation [6]. Figure 2 shows how to assess the dP/dt mean from the ascending limb of the mitral regurgitation CW Doppler flow signal. An alternative option is the presence of a significant aortic regurgitation CW Doppler flow signal, which allows assessment of the dP/dt mean from the descending limb of the continuous wave Doppler signal. The advantage of this variable is that it may be assessed across the mitral valve, even with small high-velocity jets. It has to be remembered this variable is preload dependent and relatively afterload independent [7]. A normal value lies between 800 and 1200 mm Hg s^{-1}. Often the Doppler technique will underestimate the true value of the dP/dt mean.

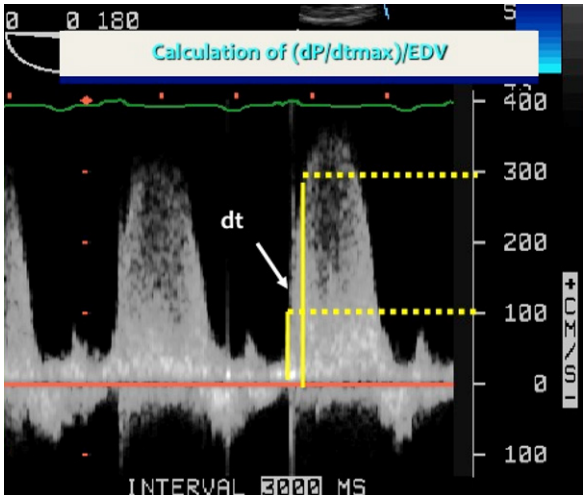

Figure 2. Calculation of dP/dt mean by echo-Doppler. At two different Doppler velocities, the marker is set, from which the time difference (dt) could be estimated. The modified Bernoulli equation is utilized calculating the dP. In this example, we choose 3 and 1 m s^{-1}, respectively: $3^2–1^2$/dt

The myocardial performance index has been introduced by Tei *et al.* [8, 9], providing an index circumscribing both systolic and diastolic performance of the left or right ventricle. The index is calculated from time intervals as shown in the following formula:

$$MPI = (a - b) / b = (ICT + IRT)/ET$$

in which; a, time interval between end of atrial contraction wave and start of the early diastolic filling wave; b, ejection time (cfr. ET); ET, ejection time (measured in Doppler mode of the flow across the aortic valve); ICT, isovolumetric contraction time; and MPI, myocardial performance index. Figure 3 depicts the practical aspects of the calculation of this index.

The index is preload dependent, as demonstrated by several authors [10–12] and, in fact, there is a close relationship with the dP/dt max [13]. MPI is independent of ventricular geometry and therefore has been utilized in grading ventricular function in congenital heart disease [14–17], in particular in univentricular surgery [14, 16, 18], ischaemic heart disease [19] and dilated cardiomyopathy [20].

Tissue Doppler imaging provides another variable, which appears extremely useful in clinical practice to assess global ventricular function, both on the left and right side [21, 22]. Tissue Doppler Imaging utilizes high filter low-velocity signals to depict velocities within the myocardial wall. If the sample volume is set at the mitral annular ring at the lateral or median border, a tissue Doppler image shows the velocities during systole and diastole (Fig. 4). The systolic velocity of the myocardial tissue is a measure of systolic function. A normal value for the LV is > 12 cm s^{-1}, whereas decreased LV systolic function offers values < 8 cm s^{-1}.

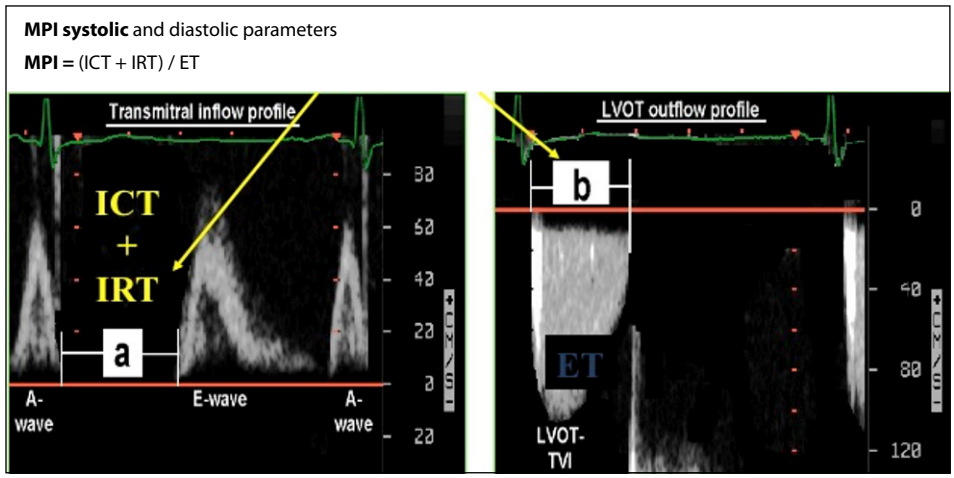

Figure 3. Calculation of myocardial performance index (MPI). MPI is calculated utilizing the formula a-b/b, with *a* being the difference in time between the end of the atrial contraction wave and the start of the early diastolic inflow; *b* being the ejection time (estimated from the flow wave across the aortic valve). MPI close to 1 shows a severely decreased function

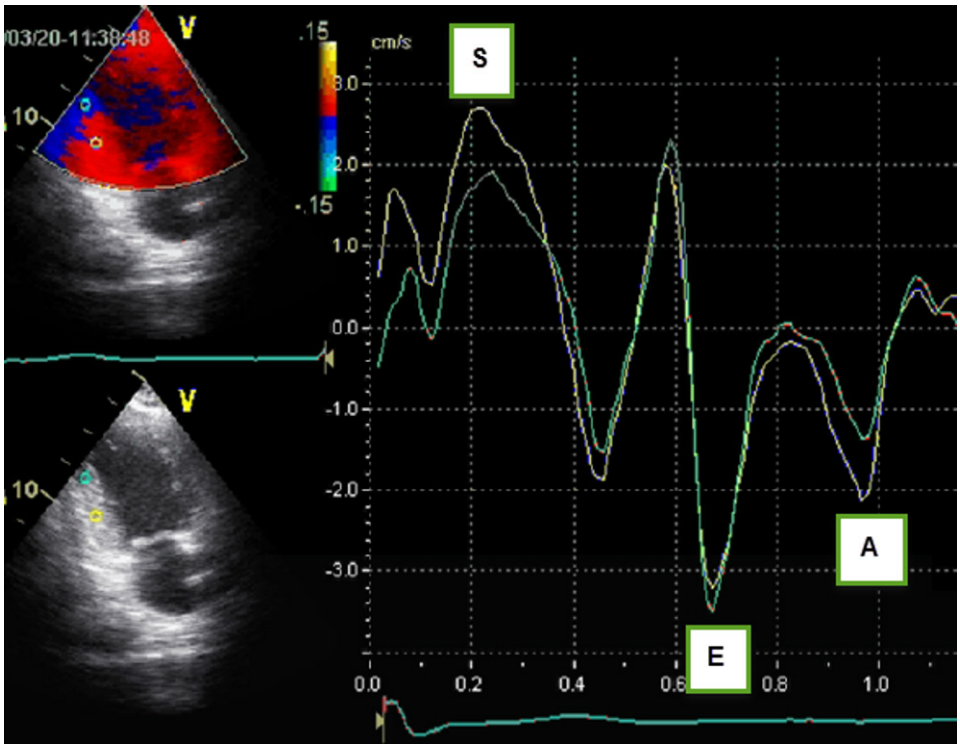

Figure 4. Tissue Doppler image at the level of the mitral annulus. A — atrial contraction induced velocity wave of the mitral annulus; E — velocity wave following early filling; S — systolic velocity wave, following contraction of the left ventricle

Again, this measure is load dependent, as demonstrated by our group [23] and others [24]. With decreasing systolic LV function, load dependency is lower. This variable can be utilized as a continuous monitoring tool intra-operatively with transoesophageal echocardiography [25], or in the ICU.

More complicated function assessment is also available when integrating cardiac ultrasound technology and arterial pressure tracing. Examples can be found relating time-based altering LV areas, a surrogate of ventricular volumes, with their relative arterial pressure time point to determine Emax, as an offline measure of ventricular contractility (per definition load independent) [26, 27]. Furthermore, preload-adjusted maximal power is a single-beat index of ventricular contractility, relating arterial pressure and peak transaortic flow velocity [28], which can be clinically replaced by preload-adjusted peak power [29]. The pumping heart is seen as an energy source generating hydraulic energy, exerting a certain amount of ventricular work (power). Both approaches have been abandoned because of the complexity in assessing contractility [30]. In addition, preload-adjusted maximal power has some important physiologic shortcomings, related to the correction factor and correct preload estimation, both at the level of the left [30] and right ventricle [31]. Both methods, however, clearly demonstrate the potential within cardiac ultrasound in conjunction with an arterial pressure trace analysis.

PRELOAD AND FILLING PRESSURES

The filling status of the patient is a static variable, which does not imply filling necessity per se. Each two-dimensional or three-dimensional measure of the LV, such as left ventricle end-diastolic diameter (LVEDD), LVEDA or left ventricle end-diastolic volume (LVEDV), serves as a static variable of preload. Fluid responsiveness is the predictability of a beneficial consequence of filling, without an association with filling necessity. The introduction of a (reversible) fluid challenge allows for testing a static variable in a dynamic manner. Whereas echo-Doppler is mostly directed towards flow assessment, this technology is able to estimate pressures. Echo-Doppler often indirectly offers information of right atrial pressures and the pulmonary circulation using right atrial or/and ventricular (RV) dilatation, as well as the presence of tricuspid or/and pulmonary valve regurgitation. RV dilatation is defined as RV diameter > 0.6 of the LV diameter and significant RV dilatation as RV diameter > LV diameter. Though RV dilatation is sometimes related with severe RV dysfunction after acute myocardial infarction, most often RV dilatation is related with increased RV afterload. Right ventricular end-systolic pressure (RVESP) is a good ultrasound measure of the pulmonary artery systolic pressure (Fig. 5).

Quantification of pulmonary valve regurgitation is often more difficult and could be most easily be assessed in a deep transgastric view (120°).

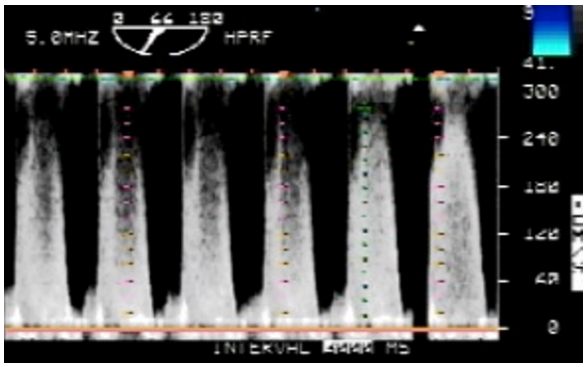

Figure 5. Tricuspid flow velocity. Right ventricular end-diastolic pressure can be estimated from tricuspid flow velocity, applying the modified Bernoulli equation ($P = 4 \times v^2$)

For many years, dynamic variables have been introduced and discussed during mechanical ventilation with altering intra-thoracic pressures, such as pulse pressure (PPV) or stroke volume variation (SVV). The echo-Doppler analogue is a TVI variation, as a measure of SVV, with the sample volume across the aortic valve. In a rabbit model of hypovolaemic shock with controlled bleeding during mechanical ventilation, Slama *et al.* [32] demonstrated a decreasing TVI. Intra-thoracic pressure variations induced increased TVI variations, which coincided with augmented systolic pressure variations (SPV). Moreover, the 2-D and 3-D measures of static preload could be assessed in a dynamic way, namely: after basic estimation, a passive leg raising manoeuvre can be performed examining the evolution of the particular variable with a filling volume of ± 300 mL. An overview of different approaches at the bedside to assess preload responsiveness in an elegant approach with echo-Doppler was published recently [34].

Mean right atrial pressure is the consequence of venous return, right ventricular systolic function and pulmonary artery pressure (PAP). It is seldom estimated with echo-Doppler [35]. In particular, right atrial dilation is a measure of overload, as well as a permanent shift of the inter-atrial septum towards the left atrium.

In contrast, pulmonary artery pressure is often assessed. Pulmonary artery systolic pressure can be derived from RVESP, which is estimated from the regurgitation flow across the tricuspid valve (Fig. 5). For three decades, a clear relationship has been demonstrated between these two pressures [36]. If significant pulmonary valve regurgitation is present, right ventricular volume will increase and result in severe tricuspid insufficiency, from which pulmonary artery systolic pressure could be estimated. If pulmonary stenosis is present (very rare in our regions), RVESP will underestimate true pulmonary hypertension.

Pulmonary capillary wedge pressure (PCWP) can be estimated from the ratio of the transmitral early filling wave velocity (E) versus the tissue Doppler analogue (e') [37–39]. Although there is no direct correlation between E/e' and PCWP, it can be derived from the following formula [40]:

$$PCWP\ (mm\ Hg) = 1.24 \times (E/e') + 1.9\ mm\ Hg$$

It is important to remark that E/e' is easily and rapidly obtained at the bedside with a transthoracic echo-Doppler, without any invasiveness. This variable has been shown to be a very practical monitoring tool in various situations: predicting successful weaning off the ventilator [41], filling status in hypertrophic cardiomyopathy [42], and the predictability and stratification of survival in sepsis and septic shock [43, 44].

AFTERLOAD

The determinants of arterial afterload are arterial compliance and systemic vascular resistance; both are derived from arterial pressure and flow. They reflect the primary and steady pulsatile component of arterial load, respectively. Both in cardiac failure and septic shock, large and small artery elastic dysfunction occur and, as they both contribute to an increased cardiovascular risk, monitoring is warranted. More than 60% of total arterial compliance resides in the ascending and thoracic aorta, focusing monitoring of this variable to these parts of the aorta [45]. Traditional haemodynamic monitoring offers only limited access to afterload indices. An echo-Doppler, in conjunction with arterial tracing characteristics, could result in a more appropriate approach of afterload.

End-systolic meridional wall stress $\sigma_{m(es)}$ is calculated from the following formula:

$$\sigma_{m(es)} = 1{,}33 \times RRs \times (A_m/A_c)\ (dyne\ .\ cm^{-5})$$

in which A_c, left ventricular short axis end-systolic area within the endocardial borders; A_m, left ventricular short axis end-systolic area of the myocardial wall; RRs, systolic blood pressure. It exemplifies the end-systolic wall stress and increases with hypertrophy of the myocardial wall and with systolic blood pressure [46]. Table 1 offers more insight into the contrasting differences of information obtained from the systemic vascular resistance versus end-systolic meridional wall stress.

Another measure which could be used in clinical practice to circumscribe ventricular afterload can be derived from the end-systolic pressure-area product:

$$SVR \approx RRs \times LVESA$$

with LVESA, left ventricular end-systolic area and RRs, systolic blood pressure.

It is obvious that afterload estimation by means of echo-Doppler techniques is not at all easy and simple, as many factors have to be taken into account: not only 2-D image and

Doppler signal quality but, in particular, the alignment of area changes and pressure changes, suggesting the most complex assessments and calculations cannot be conveyed properly. Two-D imaging is in particular hampered at the level of the ascending aorta, especially in postoperative cardiac surgical patients [45]. Therefore, the most useful afterload descriptors in clinical practice can be reduced to SVR as a measure of the steady components of arterial load, and E_a, being a measure of the main pulsatile components of arterial load [45, 47]. Both incorporate flow components (cardiac output and stroke volume, respectively) and arterial pressure.

On the right side, a pulsed wave Doppler of the pulmonary artery permits the calculation of the pulmonary vascular resistance (PVR) in a simple formula:

$$PVR = (PEP/AcT)/ET$$

with AcT, acceleration time, the time from baseline to peak pulmonary artery pulsed wave Doppler velocity; ET, ejection time, measured during the complete systole of the pulmonary artery pulsed wave Doppler signal; PEP, pre-ejection period, time interval from QRS on the ECG until the start of ejection on the pulmonary artery pulsed wave Doppler signal. Figure 6 depicts the different time intervals used in this formula. Finally, pulmonary arterial elastance (E_{pa}) may be estimated from:

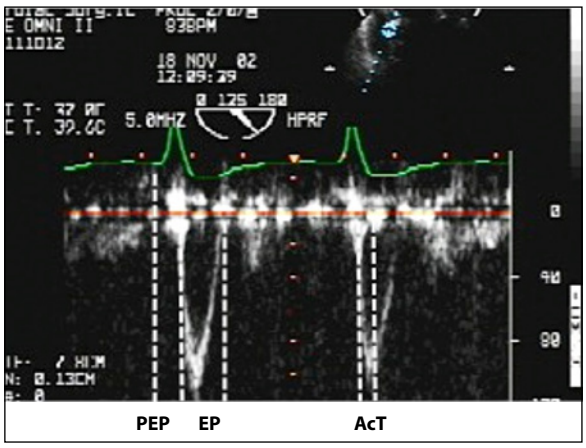

Figure 6. Pulmonary artery flow velocity. AcT, acceleration time (time from start of ejection till peak velocity is reached); EP, ejection period, from start of ejection across the pulmonary valve until end of ejection; PEP, pre-ejection period, from start of QRS (ECG) until start of ejection phase

Table 1. Comparison of information provided by systemic vascular resistance and end-systolic meridional wall stress in a normal left ventricle versus a dilated left ventricle

HD variable	Normal LV	Dilated LV
Wall thickness (cm)	1	0.5
Area diameter (cm)	2	4
RRs (mm Hg)	100	100
MAP (mm Hg)	75	75
CO (L min^{-1})	5	5
SVR (mm Hg.s.cm^{-5})	1200	1200
σm(es) (dynes.cm^{-2})	45	270

$$E_{pa} = RVESP/SV = RVESP/(TVI_{ao} \times AVA)$$

with AVA, mean aortic valve area; RVESP, right ventricular end-systolic pressure; SV, stroke volume; TVI_{ao}, time velocity integral of aortic flow [48] (Table 2).

CONCLUSIONS

Echo-Doppler provides important bedside monitoring facilities. Traditional invasive haemodynamic pressure monitoring offers haemodynamic information only in an incomplete manner, without any knowledge of ventricular performance, pressure gradients or any valve regurgitation. This review summarizes some important aspects of echo-Doppler monitoring in view of monitoring the three most important pillars of haemodynamics.

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Table 2. Various afterload variables and the respective formulas

Variable	Abbreviation	Formula	Normal value — units
Systemic vascular resistance	SVR	MAP-CVP/CO	800–1200 dynes.s.cm^{-5}
Total arterial compliance [49]	C	SV/PP	mL mm Hg^{-1}
Effective arterial elastance	E_a	P_{es}/SV	1.5–2.5 mm Hg mL^{-1}
Pulmonary vascular resistance	PVR	(PEP/AcT)/ET	40–250 dynes.s.cm^{-5}
Pulmonary arterial elastance	E_{pa}	RVESP/SV	mm Hg mL^{-1}

integrated within the not-for-profit charitable organization iMERiT, International Medical Education and Research Initiative, under Belgian law. The IFA website (http://www.fluidacademy.org) is now an official SMACC affiliated site (Social Media and Critical Care) and its content is based on the philosophy of FOAM (Free Open Access Medical education — #FOAMed). The site recently received the HONcode quality label for medical education (https://www.healthonnet.org/HONcode/Conduct.html?HONConduct519739).

References:

1. Poelaert JI, Schüpfer G. Hemodynamic monitoring utilizing transesophageal echocardiography: the relationships among pressure, flow, and function. Chest. 2005; 127(1): 379–390, doi: 10.1378/chest.127.1.379, indexed in Pubmed: 15654003.
2. Subramaniam B, Talmor D. Echocardiography for management of hypotension in the intensive care unit. Crit Care Med. 2007; 35(8 Suppl): S401–S407, doi: 10.1097/01.CCM.0000270277.29661.3B, indexed in Pubmed: 17667465.
3. Skarvan K, Lambert A, Filipovic M, et al. Reference values for left ventricular function in subjects under general anaesthesia and controlled ventilation assessed by two-dimensional transoesophageal echocardiography. Eur J Anaesthesiol. 2001; 18(11): 713–722, indexed in Pubmed: 11580777.
4. Reuter DA, Kirchner A, Felbinger TW, et al. Usefulness of left ventricular stroke volume variation to assess fluid responsiveness in patients with reduced cardiac function. Crit Care Med. 2003; 31(5): 1399–1404, doi: 10.1097/01.CCM.0000059442.37548.E1, indexed in Pubmed: 12771609.
5. Scolletta S, Franchi F, Romagnoli S, et al. Pulse wave analysis Cardiac Output validation (PulseCOval) Group. Comparison Between Doppler-Echocardiography and Uncalibrated Pulse Contour Method for Cardiac Output Measurement: A Multicenter Observational Study. Crit Care Med. 2016; 44(7): 1370–1379, doi: 10.1097/CCM.0000000000001663, indexed in Pubmed: 27097293.
6. Nakatani S, Garcia MJ, Firstenberg MS, et al. Noninvasive assessment of left atrial maximum dP/dt by a combination of transmitral and pulmonary venous flow. J Am Coll Cardiol. 1999; 34(3): 795–801, indexed in Pubmed: 10483962.
7. Lee JD, Tajimi T, Widmann TF, et al. Application of end-systolic pressure-volume and pressure-wall thickness relations in conscious dogs. J Am Coll Cardiol. 1987; 9(1): 136–146, indexed in Pubmed: 3794091.
8. Tei C, Dujardin KS, Hodge DO, et al. Doppler echocardiographic index for assessment of global right ventricular function. J Am Soc Echocardiogr. 1996; 9(6): 838–847, indexed in Pubmed: 8943444.
9. Tei C, Nishimura RA, Seward JB, et al. Noninvasive Doppler-derived myocardial performance index: correlation with simultaneous measurements of cardiac catheterization measurements. J Am Soc Echocardiogr. 1997; 10(2): 169–178, indexed in Pubmed: 9083973.
10. Cheung MMH, Smallhorn JF, Redington AN, et al. The effects of changes in loading conditions and modulation of inotropic state on the myocardial performance index: comparison with conductance catheter measurements. Eur Heart J. 2004; 25(24): 2238–2242, doi: 10.1016/j.ehj.2004.07.034, indexed in Pubmed: 15589642.
11. Poelaert J, Heerman J, Schüpfer G, et al. Estimation of myocardial performance in CABG patients. Acta Anaesthesiol Scand. 2004; 48(8): 973–979, doi: 10.1111/j.0001-5172.2004.00466.x, indexed in Pubmed: 15315614.
12. Haney MF, A'Roch R, Johansson G, et al. Beat-to-beat change in "myocardial performance index" related to load. Acta Anaesthesiol Scand. 2007; 51(5): 545–552, doi: 10.1111/j.1399-6576.2007.01287.x, indexed in Pubmed: 17430314.
13. Broberg CS, Pantely GA, Barber BJ, et al. Validation of the myocardial performance index by echocardiography in mice: a noninvasive measure of left ventricular function. J Am Soc Echocardiogr. 2003; 16(8): 814–823, doi: 10.1067/S0894-7317(03)00399-7, indexed in Pubmed: 12878990.
14. Eidem BW, O'Leary PW, Tei C, et al. Usefulness of the myocardial performance index for assessing right ventricular function in congenital heart disease. Am J Cardiol. 2000; 86(6): 654–658, indexed in Pubmed: 10980218.
15. Ishii M, Eto G, Tei C, et al. Quantitation of the global right ventricular function in children with normal heart and congenital heart disease: a right ventricular myocardial performance index. Pediatr Cardiol. 2000; 21(5): 416–421, doi: 10.1007/s002460010100, indexed in Pubmed: 10982698.
16. Salehian O, Schwerzmann M, Merchant N, et al. Assessment of systemic right ventricular function in patients with transposition of the great arteries using the myocardial performance index: comparison with cardiac magnetic resonance imaging. Circulation. 2004; 110(20): 3229–3233, doi: 10.1161/01.CIR.0000147284.54140.73, indexed in Pubmed: 15533860.
17. Abd El Rahman MY, Abdul-Khaliq H, Vogel M, et al. Value of the new Doppler-derived myocardial performance index for the evaluation of right and left ventricular function following repair of tetralogy of fallot. Pediatr Cardiol. 2002; 23(5): 502–507, doi: 10.1007/s00246-002-1469-5, indexed in Pubmed: 12189405.
18. Morhy SS, Andrade JL, Soares AM, et al. Non-invasive assessment of right ventricular function in the late follow-up of the Senning procedure. Cardiol Young. 2005; 15(2): 154–159, doi: 10.1017/S1047951105000338, indexed in Pubmed: 15845158.
19. Chockalingam A, Gnanavelu G, Alagesan R, et al. Myocardial performance index in evaluation of acute right ventricular myocardial infarction. Echocardiography. 2004; 21(6): 487–494, doi: 10.1111/j.0742-2822.2004.03139.x, indexed in Pubmed: 15298683.
20. Elbl L, Hrstkova H, Tomaskova I, et al. Late anthracycline cardiotoxicity protection by dexrazoxane (ICRF-187) in pediatric patients: echocardiographic follow-up. Support Care Cancer. 2006; 14(2): 128–136, doi: 10.1007/s00520-005-0858-8, indexed in Pubmed: 16034614.
21. Vitarelli A, Gheorghiade M. Transthoracic and transesophageal echocardiography in the hemodynamic assessment of patients with congestive heart failure. Am J Cardiol. 2000; 86(4A): 36G–40G, indexed in Pubmed: 10997352.
22. Galderisi M, Severino S, Cicala S, et al. The usefulness of pulsed tissue Doppler for the clinical assessment of right ventricular function. Ital Heart J. 2002; 3(4): 241–247, indexed in Pubmed: 12025373.
23. Amà R, Segers P, Roosens C, et al. The effects of load on systolic mitral annular velocity by tissue Doppler imaging. Anesth Analg. 2004; 99(2): 332–338, doi: 10.1213/01.ANE.0000131972.99804.28, indexed in Pubmed: 15271700.
24. Cannesson M, Jacques D, Pinsky MR, et al. Effects of modulation of left ventricular contractile state and loading conditions on tissue Doppler myocardial performance index. Am J Physiol Heart Circ Physiol. 2006; 290(5): H1952–H1959, doi: 10.1152/ajpheart.01090.2005, indexed in Pubmed: 16361366.
25. Skarvan K, Filipovic M, Wang J, et al. Use of myocardial tissue Doppler imaging for intraoperative monitoring of left ventricular function. Br J Anaesth. 2003; 91(4): 473–480, indexed in Pubmed: 14504145.
26. Royse CF, Connelly KA, MacLaren G, et al. Evaluation of echocardiography indices of systolic function: a comparative study using pressure-volume loops in patients undergoing coronary artery bypass surgery. Anaesthesia. 2007; 62(2): 109–116, doi: 10.1111/j.1365-2044.2006.04911.x, indexed in Pubmed: 17223800.
27. Gorcsan J, Gasior TA, Mandarino WA, et al. Assessment of the immediate effects of cardiopulmonary bypass on left ventricular performance by on-line pressure-area relations. Circulation. 1994; 89(1): 180–190, indexed in Pubmed: 8281645.
28. Schmidt C, Roosens C, Struys M, et al. Contractility in humans after coronary artery surgery. Anesthesiology. 1999; 91(1): 58–70, indexed in Pubmed: 10422929.
29. Amà R, Claessens T, Roosens C, et al. A comparative study of preload-adjusted maximal and peak power: assessment of ventricular performance in clinical practice. Anaesthesia. 2005; 60(1): 35–40, doi: 10.1111/j.1365-2044.2004.03974.x, indexed in Pubmed: 15601270.
30. Segers P, Tchana-Sato V, Leather HA, et al. Determinants of left ventricular preload-adjusted maximal power. Am J Physiol Heart Circ Physiol. 2003; 284(6): H2295–H2301, doi: 10.1152/ajpheart.01110.2002, indexed in Pubmed: 12637350.
31. Leather HA, Segers P, Sun YY, et al. The limitations of preload-adjusted maximal power as an index of right ventricular contractility. Anesth Analg. 2002; 95(4): 798–804, table of contents, indexed in Pubmed: 12351248.
32. Slama M, Masson H, Teboul JL, et al. Respiratory variations of aortic VTI: a new index of hypovolemia and fluid responsiveness. Am J Physiol Heart Circ Physiol. 2002; 283(4): H1729–H1733, doi: 10.1152/ajpheart.00308.2002, indexed in Pubmed: 12234829.
33. Poelaert J. Assessment of loading conditions with cardiac ultrasound. A comprehensive review. Anaesthesiol Intensive Ther. 2015; 47(5): 464–470, doi: 10.5603/AIT.a2015.0068, indexed in Pubmed: 26505579.

34. Poelaert J. Assessment of loading conditions with cardiac ultrasound. A comprehensive review. Anaesthesiol Intensive Ther. 2015; 47(5): 464–470, doi: 10.5603/AIT.a2015.0068, indexed in Pubmed: 26505579.
35. Scapellato F, Temporelli PL, Eleuteri E, et al. Accurate noninvasive estimation of pulmonary vascular resistance by Doppler echocardiography in patients with chronic failure heart failure. J Am Coll Cardiol. 2001; 37(7): 1813–1819, indexed in Pubmed: 11401116.
36. Berger M, Haimowitz A, Van Tosh A, et al. Quantitative assessment of pulmonary hypertension in patients with tricuspid regurgitation using continuous wave Doppler ultrasound. J Am Coll Cardiol. 1985; 6(2): 359–365, indexed in Pubmed: 4019921.
37. Kim YJ, Sohn DW. Mitral annulus velocity in the estimation of left ventricular filling pressure: prospective study in 200 patients. J Am Soc Echocardiogr. 2000; 13(11): 980–985, indexed in Pubmed: 11093099.
38. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study. Circulation. 2000; 102(15): 1788–1794, indexed in Pubmed: 11023933.
39. Combes A, Arnoult F, Trouillet JL. Tissue Doppler imaging estimation of pulmonary artery occlusion pressure in ICU patients. Intensive Care Med. 2004; 30(1): 75–81, doi: 10.1007/s00134-003-2039-x, indexed in Pubmed: 14634723.
40. Nagueh SF, Middleton KJ, Kopelen HA, et al. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol. 1997; 30(6): 1527–1533, indexed in Pubmed: 9362412.
41. Caille V, Amiel JB, Charron C, et al. Echocardiography: a help in the weaning process. Crit Care. 2010; 14(3): R120, doi: 10.1186/cc9076, indexed in Pubmed: 20569504.
42. Nagueh SF, Lakkis NM, Middleton KJ, et al. Doppler estimation of left ventricular filling pressures in patients with hypertrophic cardiomyopathy. Circulation. 1999; 99(2): 254–261, indexed in Pubmed: 9892592.
43. Hillis GS, Møller JE, Pellikka PA, et al. Noninvasive estimation of left ventricular filling pressure by E/e' is a powerful predictor of survival after acute myocardial infarction. J Am Coll Cardiol. 2004; 43(3): 360–367, doi: 10.1016/j.jacc.2003.07.044, indexed in Pubmed: 15013115.
44. Ishizu T, Seo Y, Kawano S, et al. Stratification of impaired relaxation filling patterns by passive leg lifting in patients with preserved left ventricular ejection fraction. Eur J Heart Fail. 2008; 10(11): 1094–1101, doi: 10.1016/j.ejheart.2008.07.011, indexed in Pubmed: 18755627.
45. Heerman JR, Segers P, Roosens CD, et al. Echocardiographic assessment of aortic elastic properties with automated border detection in an ICU: in vivo application of the arctangent Langewouters model. Am J Physiol Heart Circ Physiol. 2005; 288(5): H2504–H2511, doi: 10.1152/ajpheart.00368.2004, indexed in Pubmed: 15604135.
46. Municino A, Simone Gde, Roman M, et al. Assessment of left ventricular function by meridoneal and circumferential systolic mass/minor-axis shortening relations in dilated cardiomyopathy. Am J Cardiol. 1996; 78(544): 549.
47. Segers P, Stergiopulos N, Westerhof N. Relation of effective arterial elastance to arterial system properties. Am J Physiol Heart Circ Physiol. 2002; 282(3): H1041–H1046, doi: 10.1152/ajpheart.00764.2001, indexed in Pubmed: 11834502.
48. Taghavi S, Esmaeilzadeh M, Amin A, et al. Pulmonary arterial elastance for estimating right ventricular afterload in systolic heart failure. Congest Heart Fail. 2011; 17(6): 288–293, doi: 10.1111/j.1751-7133.2011.00222.x, indexed in Pubmed: 22103920.
49. Segers P, Verdonck P, Deryck Y, et al. Pulse pressure method and the area method for the estimation of total arterial compliance in dogs: sensitivity to wave reflection intensity. Ann Biomed Eng. 1999; 27(4): 480–485, indexed in Pubmed: 10468232.

Corresponding author:
Prof. Jan Poelaert, M.D., Ph.D.
Department of Anaesthesia
University Hospital Brussels
Laarbeeklaan 101
1090 Jette, Belgium
e-mail: jan.poelaert@uzbrussel.be

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