Přehledový článek | Review article

How to invasively assess left ventricular function and its efficiency of work

Lukáš Povišer, Karol Čurila

Cardiocenter, Third Faculty of Medicine, Charles University and University Hospital Královské Vinohrady, Prague, the Czech Republic

ARTICLE INFO

Article history:
Submitted: 20. 9. 2023
Revised: 22. 10. 2023
Accepted: 24. 10. 2023
Available online: 29. 1. 2024

Klíčová slova:
dP/dt_{max}
Hemodynamika
Srdeční funkce
Tepová práce
Výkonnost srdce

SOUHRN

Efektivita práce levé komory vyžaduje udržení srdečního výdeje, který je nárokován systémovým oběhem, a to bez vysokých metabolických nároků nebo nároků na kyslík ze strany myokardu levé komory. V tomto článku je levá komora uvažována jako pumpa a výkonnost je založena na hodnocení měření jejích tlaků, objemů a průtoku. Analýza funkce komory z hlediska vztahů mezi tlakem a objemem umožňuje plně analyzovat globální a regionální dynamiku komory, kterou lze poměrně snadno a přesně získat pomocí vodivostního katétru. Maximální změna tlaku za jednotku času v levé komoře je považována za ukazatel kontraktility levé komory, za jistých situací může být alternativou arteriální dP/dt_{max} jakožto méně invazivní metoda. Při hodnocení nových kardiostimulačních technik a srdeční resynchronizační terapie se jako nejpraktičtější metoda jeví invazivní systolický krevní tlak s průměrováním více tepů a doplněním o několikrát opakované střídání stimulací.

Keywords:
Cardiac function
dP/dt_{max}
Hemodynamics
Performance of heart
Stroke work

ABSTRACT

Effective performance of the left ventricle requires the maintenance of a cardiac output as demanded by the systemic circulation without a high hemodynamic cost or pressure and without a high metabolic cost or oxygen demand by the left ventricular myocardium. In this article the left ventricle is considered as a pump and performance is based on evaluation of measurements of its pressure, volumes, and flow. Analysis of ventricular function in terms of pressure-volume relationships allows global and regional ventricular dynamics to be fully analyzed and relatively easily and precisely obtained with conductance catheter. The maximum rate of left ventricular pressure is classically considered as a marker of left ventricular contractility and in specific situation arterial dP/dt_{max}, as minimally invasive method, can be an alternative. When assessing new pacing techniques and cardiac resynchronization therapy, invasive systolic blood pressure appears to be the most practical measure with multi-beat averaging and the addition of multiple spaced repeated alternations.

Introduction

The main function of the left ventricle (LV) is to pump oxygenated blood from the lungs into the aorta for distribution to the rest of the body through systemic circulation. The LV function is believed to be optimized through thousands of years of evolution because the survival of the biological system depends on the efficient transport of blood.[1,2]

Usually, in cardiology, the term ventricular function means parameters such as ejection fraction, long-axis shortening, myocardial systolic strain or strain rate, and mitral annular systolic velocity. Left ventricular ejection fraction (LVEF) is still the most widely used parameter to evaluate overall left ventricular systolic function, but it has fundamental limitations for assessment of subclinical dysfunction and regional function. Hemodynamic parameters, like blood pressure, cardiac output (CO), left or right ventricular stroke volume (SV), ventricular stroke work (VSW) are used by physicians and scientists in order to evaluate cardiac function more accurately and at a given time.[1,3]

Effective performance of the left ventricle requires the maintenance of a cardiac output as demanded by

Address: Ing. Lukáš Povišer, Cardiocenter, Third Faculty of Medicine, Charles University and University Hospital Královské Vinohrady, Šrobárova 50, 100 34 Prague 10, the Czech Republic, e-mail: lukas.poviser@lf3.cuni.cz
DOI: 10.33678/cor.2023.080

Please cite this article as: Povišer L, Čurila K. How to invasively assess left ventricular function and its efficiency of work. Cor Vasa 2024;66:44–51.

the systemic circulation without a high hemodynamic cost or pressure and without a high metabolic cost or oxygen demand by the left ventricular myocardium.[4]

In the recent years, new pacing modalities as His bundle (HBP), left bundle (LBBP), and left septal myocardial pacing entered clinical practise to treat patients with bradycardia and heart failure. They appear to be associated with a more physiological ventricular activation, and possibly a better clinical outcome of patients.[5] Until the data from clinical studies will be available, we need other tools to assess their effect on the myocardial work effectivity. Hemodynamic measurements may appear as useful tools in assessing these methods, because it is a final common pathway through which cardiac function expresses itself. But the question remains, which hemodynamic measurements can accomplish this task best? The ideal method should combine precision, be easy to provide during the procedure and should allow determining acute changes in cardiac function.

There are two approaches to evaluation of the mechanical performance of the left ventricle. One of these is to consider the left ventricle as a muscle, the other is to consider the left ventricle as a pump and to base its evaluation on measurements of its pressure, volumes, and flow.[4,6] For the sake of brevity and applicability in clinical practice, preferably view on the left ventricle as a pump will be discussed in this review.

Even though our knowledge in measuring efficiency of the work of the left ventricle and hemodynamics is still far from complete, many advances have been made and these are presented.

How can performance of the ventricle be assessed?

The performance of the ventricle as a pump can be assessed by measuring the pressure developed by the ventricle, the stroke volume ejected by the ventricle, or preferably, the stroke work generated by the ventricle. Stroke work gives credit to the ventricle for both pressure and shortening work in a single integrated index.[7,8]

Left ventricular stroke work index

Left ventricular stroke work index (LVSWI) is often calculated in order to describe left ventricular function. LVSWI incorporates both LV systolic and diastolic function and dysfunction, respectively. This provides a broader assessment of overall LV function, particularly in the critical care patient in which preload and afterload are dynamic.[7,8] The definition of work in classical physics, force multiplied by distance, is in case of heart function replaced by pressure multiplied by volume. When one takes into consideration that the mechanical work is performed during systole, it seems appropriate to use left ventricular mean systolic pressure (LVSP) minus left ventricular end-diastolic pressure (LVEDP) multiplied by stroke index (SI). This formula LVSWI = SI × (LVSP – LVEDP) is being used by cardiologists who can often measure left ventricular pressure during catheterization.[8,9] LVSWI also reflects the increased workload of the left ventricle in aortic stenosis, when there is a large pressure difference between the left ventricle and the aorta during systole.[9] Another formula, LVSWI = SI × mean arterial pressure (MAP) minus pulmonary artery capillary wedge pressure (PCWP), is commonly used when access to left heart pressure is not possible. A third possible way to calculate LVSWI is to use peak systolic arterial pressure (SAP) instead of mean systolic.[8,11] The fact that at least three different formulas to calculate LVSWI are in use simultaneously is not without problems and could cause misleading results across different studies.

Settergren showed that it is possible to calculate LVSP from pressure measurements in a peripheral artery with reasonable accuracy. Values obtained during normo- or hypertension and from patients with normal or depressed left ventricular function did not differ.[8,12]

However, the different ways of calculating LVSWI mentioned above using SAP, LVSP or MAP are only approximations. As may be seen, the best approximation of the pressure-volume area is to use LVSP.[8,12] But the correct estimation is the area of the pressure volume loop of the left ventricle in a pressure volume diagram.

Evaluating LV function through pressure-volume relationship

The gold standard for evaluating LV function and effect of afterload is through the relation of ventricular pressure and volume.[13] The relationship between ventricular pressure and ventricular volume could be visualized as a closed left ventricular pressure-volume loop (LV-PVL), which is linear correlated to myocardial oxygen consumption and could quantify whole heartbeat's stroke work and contractility.[14,15]

A classic ventricular function curve can be constructed by plotting coordinates of performance against preload. When contractility is increased, the stroke work versus preload relationship is shifted upward. When contractility is decreased, the stroke work versus preload relationship is shifted downward.[16] Such a family of ventricular function curves credits the ventricle for pressure development and ejection, and it incorporates load and contractility. Thus, stroke work and end-diastolic volume (EDV) data allow construction of a Frank–Starling ventricular function curve or a preload recruitable stroke work relationship.[17,18]

Stroke work

Stroke work refers to the work done by the left ventricle to eject the volume of blood during one cardiac cycle, called stroke volume (SV).[14] The stroke work is best depicted using ventricular pressure-volume diagrams, in which stroke work is the area within the pressure-volume loop as depicted in Figure 1.

Therefore, the area within the pressure-volume loop is an example that mathematicians call a normal domain, from which the equation for the stroke work follows:

$$VSW = \int_{ESV}^{EDV} p_{systolic}(V)dV - \int_{ESV}^{EDV} p_{diastolic}(V)dV \qquad (1)$$

In equation (1), $p_{systolic}(V)$ denotes the intra-ventricular pressure during the ejection phase, is the intra-ventricular pressure during the filling phase. The integral is the area below the integrand or here the pressure function *p(V).* Therefore, the difference is the area within the loop, which equals the VSW.[14]

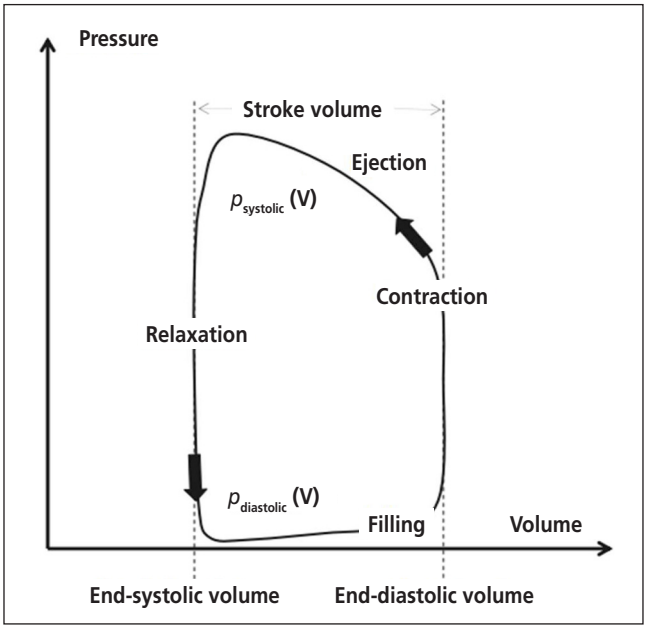

Fig. 1 – The pressure-volume loop for the ventricle is generated by plotting intraventricular pressure against ventricular volume during one cardiac cycle.

LV pressure-volume areas are representative of the total mechanical energy available to the LV to perform external work. These areas have the components of external work and potential energy.[18] The latter can perform external work under the appropriate set of LV loading conditions.[18–20]

How can be pressure-volume loops measured?

Regional ventricular wall motion could be measured by sonomicrometry and later by the development of sophisticated data acquisition and analysis based on contrast or radioisotopic angiography including first-pass angiography and biplane cineangiography.[11,21–23] These methods have made it possible to obtain instantaneous pressure--volume relationships in patients and not only in experimental animals.[14]

But only with the development of conductance catheters, the interest and the scope for experimental and clinical studies have increased.[24] It has been possible, with very small conductance catheters, to obtain pressure--volume loops. These catheters provide a unique method for acquiring hemodynamic and contractile data directly from the heart. By inserting a pressure-volume catheter into the left or right ventricle, it is possible to measure all load-dependent data for every cardiac cycle from the moment you complete your instrument until the end of the experiment.[25,26]

Moreover, pressure-volume (dimension) loops provide additional information regarding synchrony or dyssynchrony of contraction, segmental work, contractility, and diastolic function.[27]

Pressure-volume loop measurements with conductance catheters

A multielectrode catheter that continuously measures instantaneous left ventricular conductance, from which left ventricular volume can be estimated.[24–26] Studies in which this volume signal was compared with absolute volume in an isolated beating heart or with cardiac output measured independently have indicated a strong linear correlation between them.[24,28] The catheter requires careful calibration since the total signal combines left ventricular cavity conductance with conductance of the ventricular wall and other structures outside the ventricle. The unique advantage of providing continuous instantaneous volume information, when combined with left ventricular pressure micromanometry, enables an easy and rapid determination of end-systolic pressure-volume relation (ESPVR).[24–29]

PV catheters allow us to measure end-diastolic volumes and end-systolic volumes, and from these, calculate the stroke volume, ejection fraction, and cardiac output. It is also possible to calculate parameters such as stroke work, Tau, dP/dt_{max}, contraction time and relaxation time. All of which are associated with the contractility.[30]

This type of catheter provides two important advantages over non-invasive techniques. First, it generates an instantaneous signal providing immediate information on ventricular performance, allowing rapid loading changes to be used, and enabling examination of quick-response phenomena. Second, it is essentially free from pure shape change influences, as have previously demonstrated.[28]

The ESPVR measured by conductance catheter has showed a good degree of short-term reproducibility within a given subject, which has not been a uniform finding in previous studies.[24,29] It is believed this relates to the rapid and reversible load alteration and improved accuracy of the volume signal.[30]

Interaction of the left ventricle with the systemic arterial system

To fully comprehend the performance of the left ventricle it is necessary to understand the interaction of the LV with the systemic arterial system.[31] End-systolic elastance (Ees) and arterial elastance (Ea) are parameters that best characterize LV pump function under varying loading and inotropic conditions. End-systolic elastance represents the slope of the end-systolic pressure volume relationship and arterial elastance is a measure of arterial load and is calculated as the simple ratio of ventricular end-systolic pressure to stroke volume.[31,32] Arterial elastance is also possible to obtain by analysis of the peripheral arterial curve as the ratio of the pressure at the dicrotic point to the pulse volume.[33]

How to determine maximal stroke work and maximal efficiency?

Maximal LV stroke work occurs when the LV elastance and arterial elastance values are matched.[34–36] Maximal efficiency is defined as the ratio of LV stroke work to myocardial oxygen consumption (MVO_2) and occurs when end--systolic elastance is approximately 2 times greater than arterial elastance.[36,37] For normal physiologic LV pressures, end-diastolic volumes, stroke volumes, and ejection fractions to exist, the normal LV would probably operate

closer to maximal efficiency rather than to maximal stroke work.[34–36] Despite linear relationship between oxygen consumption and pressure volume area, there is a basal energy requirement and therefore mechanical efficiency (SW/PVA) cannot be equated to myocardial efficiency (SW/MVO_2).[38,39] But there are very strong correlations between the PVA and myocardial oxygen consumption for isovolumic (r = 0.973) and for ejecting contractions (r = 0.989) so that myocardial oxygen consumption may be calculated from the pressure-volume area.[25]

Measure of left ventricular contractile state

Ventricular contractility refers to the contractile or inotropic state of the whole ventricle. Indices of ventricular contractility conventionally have been divided into isovolumic phase indices (peak positive dP/dt), ejection phase indices (systolic wall stress versus endocardial shortening), and those determined at the end of ejection (end-systolic elastance).[16,40] The concept of ventricular contractility is similar to myocardial contractility, but ventricular contractility is not independent of loading conditions or ventricular remodeling. Peak positive dP/dt may be altered by an acute change in preload, whereas end-systolic elastance may be affected by chronic changes in LV volume and mass.[41,42] Myocardial contractility refers to a basic property of heart muscle that reflects the intensity of cross-bridge activity and as a result, the extent and velocity of force development and fiber shortening. The contractile state of the myocardium represents a basic characteristic that is independent of loading conditions and remodeling. Such measurements can be made only in vitro in isolated cardiac muscle cells, muscle strips, or Langendorff-perfused hearts, but attempts to assess myocardial contractility in humans in vivo present a continuing challenge. This is the reason why ventricular contractility is superior to myocardial contractility in evaluation in cardiac function.[42–45]

Left ventricular contractility influences global hemodynamic status directly. Therefore, it is an important element of the hemodynamic evaluation of the critically ill patients.[46] Impaired LV contractility is frequently seen in patients with acute coronary syndrome and sepsis.[47] Although LV end-systolic elastance is the reference method for assessing LV contractility, its bedside use is limited by its invasiveness and the technical difficulties associated with its estimation.[21,48] The maximum rate of LV pressure during isovolumetric contraction (LV dP/dt_{max}) has been classically considered as a marker of LV inotropic state.[49]

However, as LV dP/dt_{max} requires a direct measure of LV pressure, other approaches have been proposed using the arterial pressure waveform. Peripheral dP/dt_{max}, as measured from catheters inserted into the femoral or radial arteries, have been suggested as feasible surrogates for LV dP/dt_{max}. However, as the arterial pressure results from the combined interaction of the LV ejection and the arterial system properties, other potential factors could also contribute to the peripheral dP/dt_{max}, possibly degrading its accuracy as a measure of LV contractile state.[50,51]

Assessing left ventricle contractility through arterial blood pressure

Arterial dP/dt_{max} can be calculated from the arterial pressure waveform, obtained invasively from a peripheral arterial line.[51–53] It is available bedside and in patients with an arterial line already used for pressure monitoring and blood gas analyses and it does not require any additional invasive access. Moreover, arterial dP/dt_{max} can be measured on a beat-by-beat basis and continually monitored. On the other hand, arterial dP/dt_{max} is not only determined by LV contraction but is also influenced by various peripheral arterial factors and load conditions.[50–53]

Recently, several experimental studies demonstrating a significant relationship between arterial dP/dt_{max} and LV contractility have been published.[51–53] The values from continual arterial dP/dt_{max} monitoring were significantly correlated with LV dP/dt_{max} assessed. Linear regression revealed that LV dP/dt_{max} = 1.25 × arterial dP/dt_{max}. Also, arterial dP/dt_{max} was significantly correlated with CO. Monitoring LV contractility is most desirable in patients with heart failure with critical hemodynamic collapse, such as in cardiogenic shock, characterized by increased SVR and decreased CO and SV.[54] Several authors reported that the arterial dP/dt_{max} is significantly influenced by vascular filling conditions and is a relatively accurate method for obtaining left ventricular contractility only when adequate vascular filling is achieved.[52–56] In contrast, in the subgroup of patients with lower SVR, higher CO, and higher SV, arterial dP/dt_{max} doesn't give accurate and reliable information about LV dP/dt_{max}.[54]

Efficiency of the heart performance during different pacing settings: the precise measurement of the acute hemodynamic response

In recent years the delivery of pacing therapy has become more sophisticated, and the indications have expanded. But the fundamental aim has remained the same, to improve cardiac function by normalizing cardiac electrical activation in patients with conduction system disease.[57]

Throughout the history of developing pacing therapy, hemodynamic assessment has provided a useful tool in guiding the development of new pacing approaches. Hemodynamic measurements allow determining acute changes in cardiac function and their quantification. Hemodynamic measurements can also guide optimal programming of pacemakers because it is a final common pathway through which cardiac function expresses itself.[57,58]

Importance of precise hemodynamic measurement is that typical augmentation of systolic blood pressure with biventricular pacing in left bundle branch block is approximately 6 mmHg and is associated with approximately 20% mortality reduction.[59] The measurement protocol for a potential advance on CRT should be able to detect an increment about half this size (3 mmHg), since it might provide a corresponding approximately 10% mortality reduction. This means the 95% confidence interval must be <±3 mmHg.[60]

Signal versus noise and reproducibility

Hemodynamic parameters fluctuate in response to numerous biological phenomena, including respiration and other autonomic phenomena. There are many processes

that are continuous and variable. All together they make the identification of the hemodynamic consequences of a change in pacing configuration more challenging. It is almost impossible to measure and subtract the contributions of these background biological phenomena on hemodynamics that is why they are treated as noise.[61]

The signal is the element that is consistent in every instance and the noise varies between repeated measurements. When an average of multiple measurements of effect is taken, the impact of noise shrinks with the square root of the number of repeat measurements consistently. Signal-to-noise ratio is an important feature of any measure. It is a simple way to characterize the efficiency of measurement. One simple definition of the signal-to--noise ratio is the range of values obtained for different pacing settings, divided by the standard error of the measurements at each pacing setting. By prolonging the optimization session, signal-to-noise ratio can be improved.[62]

The second question that needs to be assessed is the degree of similarity of the optimal pacing configuration determined by the different hemodynamic measures. If the measures produce dramatically different optimum, then the use of hemodynamics for optimization is cast into doubt.[60,62]

The third important characteristic of a measure put forward for use in optimization is reproducibility over time. If there is a large change in measured optimum, then either the physiological optimum is changing or the measuring method does not have sufficient reproducibility.[60,62]

Which hemodynamic parameter to use in pacing optimization: Invasive blood pressure, non-invasive beat-to-beat blood pressure or LV dP/dt$_{max}$?

Shun-Shin et al. have compared invasive systolic blood pressure, non-invasive systolic blood pressure and invasive LV dP/dt$_{max}$. They have found out that there was a strong correlation between assessment by invasive and non-invasive systolic blood pressure and a weaker correlation between LV dP/dt$_{max}$ and invasive systolic blood pressure. For all three hemodynamic measures, the noise, quantified as percentage error in the quantifications of response, declined progressively as more alternations were analyzed. Invasive blood pressure showed a smaller such noise than non-invasive blood pressure and LV dP/dt$_{max}$. Their results showed that invasive blood pressure is the best parameter which follows during implantation or optimalization procedure. LV dP/dt$_{max}$ is not sufficient to guarantee precise optimization because of the worst reproducibility, and even with the replicate measurements the level of uncertainty is wide, but the accuracy improves with each repetition.[59,60,63]

What should the precise hemodynamic protocol look like for measuring acute hemodynamic response during various pacing types?

Clinicians and researchers designing protocols for optimization of pacing should start with their desired level of precision and then use standard methods to ensure that their protocols deliver it.[62] Many widely used optimization protocols, even if conducted carefully, cannot deliver reproducible optimum. The driver of reproducibility of the optimum is the signal-to-noise ratio, namely the size of the changes caused by alterations in tested setting in relation to the size of the changes occurring spontaneously. Even performing invasive, intraventricular measurements does not grant immunity to biological noise, which is important to recognize in the design of optimization protocols.[62,63]

Measure values relative to a reference setting

There are multiple biological intercurrent processes happening during pause between 2 hemodynamic measurements that will be displacing the blood pressure up and down. With longer time interval, the greater the variance between the first and second measurement appears. The solution of this problem is to only measure changes over a few seconds or minutes. To allow a wide range of settings to be tested, one can test each setting against a reference setting that is common to all.[62]

Detecting the hemodynamic changes occurring at the time of transition between one tested setting and another is a useful approach if a rapid and reproducible process is sought, depicted on Figure 2. The reason for this is that these beats have the smallest opportunity for drift noise to develop between them.[60,62]

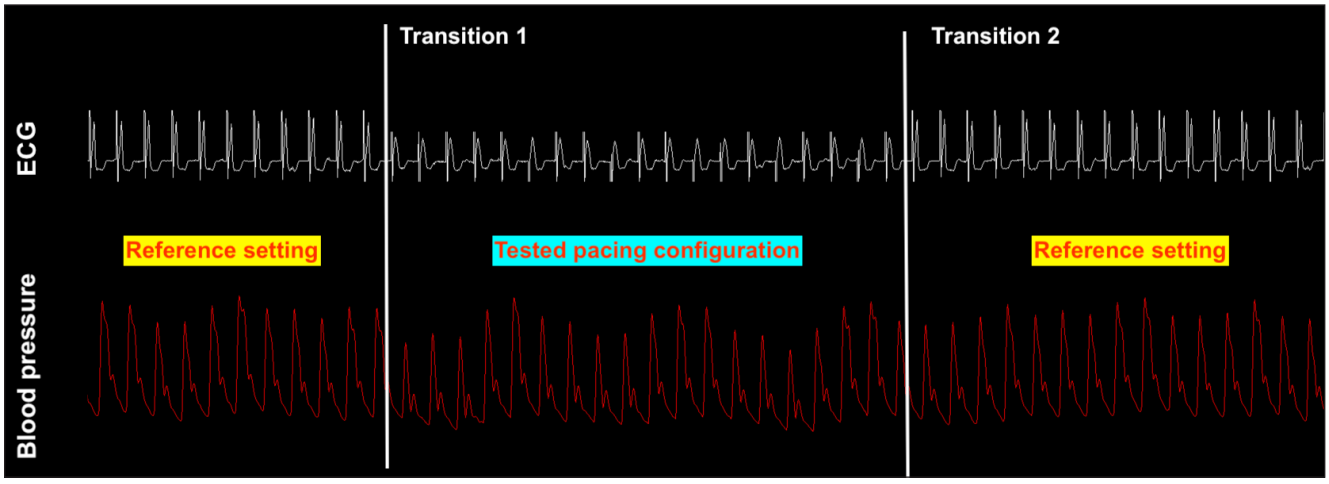

Fig. 2 – Hemodynamic protocol with reference and tested pacing configuration.

Miyazawa et al. have found that to maximize the efficiency of the optimization procedure, it is best to address the beats immediately before and immediately after the change in tested setting, not to skip any beats. They believe this is because the hemodynamic effect of (in their case) changing AV delay occurs immediately, whereas there is a more gradual homeostatic process of adaptation of peripheral tone that occurs when the initial change in hemodynamics is detected by cardiovascular reflexes. More efficient cardiac function would be expected to result in reflex reductions in vasoconstrictor tone, which would tend to reduce the measured increase in pressure. The fact that the blood pressure does not remain elevated should therefore not be considered to indicate a short-lasting physiological improvement.[62,63]

What is the most efficient number of beats to average?

Fixed averaging window of six beats provides the best optimization efficiency, it provides a robust averaging window duration that can be used for a wide range of different patients and is easy to use. For greatest efficiency, the ideal window width appears to be directly related to the number of beats occurring within a one respiratory cycle (beats-per-breath). The reason for six beats being the most efficient fixed window width is that this value is close to the average number of beats-per-breath for the whole group of patients they tested.[60,62] It is possible to further improve the optimization efficiency by tailoring the width of the averaging window to match the duration of one whole respiratory cycle. They believe the reason for this is that changes in blood pressure induced by respiration are the single largest source of noise within the signal. Alternatively, the effect of respiratory noise could be reduced by asking the patient to hold their breath or breathe at a fixed respiratory rate, at the time of the transition. However, both requests are difficult for the patient to comply with, particularly patients who have heart failure.[62,63]

Perform repeated measurements and multi-beat averaging

A key step to avoid false positive detection of differences is the addition of multiple spaced repeated alternations to the hemodynamic protocol, even when the protocol includes multi-beat averaging.

Averaging of independent repetitions of the same measurement is the single most effective method of increasing precision. The number of repetitions required for any desired degree of precision can be calculated.[63] Experiments showed the dramatic decline in measurement imprecision with increasing numbers of replicates.[60] For example, using 8 replicates with 8 beats per replicate eliminates 90 % of the variance seen with a single beat.[62]

Pacing heart rate importance

Pacing should be at 100 bpm rather than just above resting rate. The authors state three reasons for it. Firstly, to standardize the results between patients, so a rate higher than typical intrinsic rates should be used. Secondly, using a rate of 100 bpm as compared to 60 bpm, reduces the time taken by 40%. At higher heart rates, the magnitude of changes is larger, increasing the signal-to-noise ratio. Nevertheless, it must be recognized that the magnitudes of differences observed at this elevated heart rate are likely to be larger than those in day-to-day life. The reason to choose this artificially magnified response is solely to make it easier to discern differences between configurations during a realistic protocol duration. As well, selecting a heart rate significantly above the intrinsic rate ensures that changes in the intrinsic heart rate would not cause breakthrough and require that the protocol is repeated.[60]

Conclusion

In conclusion, there is no single invasive method that is suitable in all situations and procedures with respect to the patient's health status to assess the efficiency of the left ventricular function. Analysis of ventricular function in terms of pressure-volume relationships allows global and regional ventricular dynamics to be fully analyzed. In addition, this approach allows the relationships between muscle function (contractility, stiffness, potential energy) and pump function (stroke volume, stroke work) to be determined and predicted. Acquisition of pressure volume loops is precise and relatively easily obtained with conductance catheter inserted in the left ventricle.

Left ventricular end-systolic elastance is the reference method for assessing left ventricular contractility, its bedside use is limited by its invasiveness and the technical difficulties associated with its estimation. Instead of this method the maximum rate of left ventricular pressure is classically considered as a marker of left ventricular inotropic state – contractility. In recent years a new approach in assessing of contractility was examined. Arterial dP/dt_{max} is a minimally invasive method assessed from femoral or radial artery with fluid-filled pressure catheter and is a relatively accurate method for obtaining left ventricular contractility in patients with hemodynamic collapse and when adequate vascular filling is achieved.

Acute hemodynamic measurements have the potential to be a useful and precise tool for assessing whether a new pacing intervention improves acute cardiac function and what the magnitude of the improvement is. Testing of different pacing configurations requires precise measurements because of natural biological variability. Systolic blood pressure appears to be the most suitable measure and also shows advantages over the gold standard of left ventricular dP/dt_{max} in reproducibility and in signal-to-noise ratio. Multi-beat averaging and the addition of multiple spaced repeated alternations to the hemodynamic protocol is a key step to avoid false positive detection of differences. The most efficient way is to use an averaging window of one respiratory cycle, and not to skip any beats between the pretransition and posttransition averaging windows.

There is no direct comparison in the available literature of invasively assessed arterial systolic blood pressure and intra-ventricular systolic pressure using the precise hemodynamic protocol, so it needs to be still discovered, whether both provide the same accuracy and are both suitable for assessment of acute hemodynamic response.

Conflict of interest
The authors declare that they have no conflict of interest.

Funding
This paper was supported by the Charles University Research Program Cooperatio – Cardiovascular Science (KC), Ministry of Health of the Czech Republic, grant number NU21-02-00584 (KC), National Institute for Metabolic and Cardiovascular Research "CarDia" (project nr LX22NPO5104), Next Generation EU (KC).

Ethical statement
The authors declare that their work was carried out according to ethical standards.

Literature

1. Geyer H, Caracciolo G, Abe H, et al. Assessment of myocardial mechanics using speckle tracking echocardiography: fundamentals and clinical applications. J Am Soc Echocardiogr 2010;23:351–455. Erratum in: J Am Soc Echocardiogr 2010;23:734.
2. Monahan-Earley R, Dvorak AM, Aird WC. Evolutionary origins of the blood vascular system and endothelium. J Thromb Haemost 2013;11(Suppl 1):46–66.
3. Chengode S. Left ventricular global systolic function assessment by echocardiography. Ann Card Anaesth 2016;19(Suppl):S26–S34.
4. Mitchell JH, Hefner LL, Monroe RG. Performance of the left ventricle. Am J Med 1972;53:481–494.
5. Lewis AJM, Foley P, Whinnett Z, et al. His Bundle Pacing: A New Strategy for Physiological Ventricular Activation. J Am Heart Assoc 2019;8:e010972. Erratum in: J Am Heart Assoc 2019;8:e002310.
6. Baicu CF, Zile MR, Aurigemma GP, Gaasch WH. Left ventricular systolic performance, function, and contractility in patients with diastolic heart failure. Circulation 2005;111:2306–2312.
7. Jentzer JC, Wiley BM, Anavekar NS. Echocardiographic left ventricular stroke work index: An integrated noninvasive measure of shock severity. PLoS One 2022;17:e0262053.
8. Settergren G. The calculation of left ventricular stroke work index. Acta Anaesthesiol Scand 1986;30:450–452.
9. Boldt J. Clinical review: hemodynamic monitoring in the intensive care unit. Crit Care 2002;6:52–59.
10. Maeder MT, Rickli H, Weber L, et al. Systemic blood pressure in severe aortic stenosis: Haemodynamic correlates and long-term prognostic impact. ESC Heart Fail 2023;10:274–283.
11. Stephens JD, Dymond DS, Spurrell RA. Radionuclide and hemodynamic assessment of left ventricular functional reserve in patients with left ventricular aneurysm and congestive cardiac failure. Response to exercise stress and isosorbide dinitrate. Circulation 1980;61:536–542.
12. Ross J Jr, Braunwald E. The study of left ventricular function in man by increasing resistance to ventricular ejection with angiotensin. Circulation 1964;29:739–749.
13. Boudoulas KD, Triposkiadis F, Boudoulas H. Evaluation of Left Ventricular Performance: Is There a Gold Standard?. Cardiology 2018;140:257–261.
14. Bastos MB, Burkhoff D, Maly J, et al. Invasive left ventricle pressure-volume analysis: overview and practical clinical implications. Eur Heart J 2020;41:1286–1297.
15. Boldt J. Clinical review: hemodynamic monitoring in the intensive care unit. Crit Care 2002;6:52–59.
16. Aurigemma GP, Zile MR, Gaasch WH. Contractile behavior of the left ventricle in diastolic heart failure: with emphasis on regional systolic function. Circulation 2006;113:296–304.
17. Moss RL, Fitzsimons DP. Frank-Starling relationship: long on importance, short on mechanism. Circ Res 2002;90:11–13.
18. Starling EH, Visscher MB. The regulation of the energy output of the heart. J Physiol 1927;62:243–261.
19. Suga H. Ventricular energetics. Physiol Rev 1990;70:247–277.
20. Chen CH, Fetics B, Nevo E, et al. Noninvasive single-beat determination of left ventricular end-systolic elastance in humans. J Am Coll Cardiol 2001;38:2028–2034.
21. Sagawa K. The end-systolic pressure-volume relation of the ventricle: definition, modifications and clinical use. Circulation 1981;63:1223–1227.
22. Starling MR, Gross MD, Walsh RA, et al. Radionuclide determination of the relationship between left ventricular contractile state and ejection fraction. Am Heart J 1988;116:790–798.
23. Redington AN, Gray HH, Hodson ME, et al. Characterisation of the normal right ventricular pressure-volume relation by biplane angiography and simultaneous micromanometer pressure measurements. Br Heart J 1988;59:23–30.
24. Baan J, van der Velde ET, de Bruin HG, et al. Continuous measurement of left ventricular volume in animals and humans by conductance catheter. Circulation 1984;70:812–823.
25. Foëx P, Leone BJ. Pressure-volume loops: a dynamic approach to the assessment of ventricular function. J Cardiothorac Vasc Anesth 1994;8:84–96.
26. Kass DA, Midei M, Graves W, et al. Use of a conductance (volume) catheter and transient inferior vena caval occlusion for rapid determination of pressure-volume relationships in man. Cathet Cardiovasc Diagn 1988;15:192–202.
27. Monge García MI, Jian Z, Hatib F, et al. Relationship between intraventricular mechanical dyssynchrony and left ventricular systolic and diastolic performance: An in vivo experimental study. Physiol Rep 2023;11:e15607.
28. Burkhoff D, van der Velde E, Kass D, Baan J, et al. Accuracy of volume measurement by conductance catheter in isolated, ejecting canine hearts. Circulation 1985;72:440–447.
29. McKay RG, Spears JR, Aroesty JM, et al. Instantaneous measurement of left and right ventricular stroke volume and pressure-volume relationships with an impedance catheter. Circulation 1984;69:703–710.
30. Kass DA, Yamazaki T, Burkhoff D, et al. Determination of left ventricular end-systolic pressure-volume relationships by the conductance (volume) catheter technique. Circulation 1986;73:586–595.
31. Sunagawa K, Maughan WL, Burkhoff D, Sagawa K. Left ventricular interaction with arterial load studied in isolated canine ventricle. Am J Physiol 1983;245(5 Pt 1):H773–H780.
32. Sunagawa K, Sagawa K, Maughan WL. Ventricular interaction with the loading system. Ann Biomed Eng 1984;12:163–189.
33. Monge Garcia MI, Jian Z, Settels JJ, et al. Reliability of effective arterial elastance using peripheral arterial pressure as surrogate for left ventricular end-systolic pressure. J Clin Monit Comput 2019;33:803–813.
34. Sunagawa K, Maughan WL, Sagawa K. Optimal arterial resistance for the maximal stroke work studied in isolated canine left ventricle. Circ Res 1985;56:586–595.
35. Wilcken DE, Charlier AA, Hoffman JI, Guz A. Effects of alteration in aortic impedance on the performance of the ventricles. Circ Res 1964;14:283–293.
36. Little WC, Cheng CP. Left ventricular-arterial coupling in conscious dogs. Am J Physiol 1991;261(1 Pt 2):H70–H76.
37. Burkhoff D, Sagawa K. Ventricular efficiency predicted by an analytical model. Am J Physiol 1986;250(6 Pt 2):R1021–R1027.
38. Suga H, Hayashi T, Shirahata M, et al. Regression of cardiac oxygen consumption on ventricular pressure-volume area in dog. Am J Physiol 1981;240:H320–H325.
39. Starling MR, Mancini GB, Montgomery DG, Gross MD. Relation between maximum time-varying elastance pressure-volume areas and myocardial oxygen consumption in dogs. Circulation 1991;83:304–314.
40. Baicu CF, Zile MR, Aurigemma GP, Gaasch WH. Left ventricular systolic performance, function, and contractility in patients with diastolic heart failure. Circulation 2005;111:2306–2312.
41. Carabello BA. Evolution of the study of left ventricular function: everything old is new again. Circulation 2002;105:2701–2703.
42. Quinones MA, Gaasch WH, Alexander JK. Influence of acute changes in preload, afterload, contractile state and heart rate

on ejection and isovolumic indices of myocardial contractility in man. Circulation 1976;53:293–302.
43. Anand IS, Liu D, Chugh SS, et al. Isolated myocyte contractile function is normal in postinfarct remodeled rat heart with systolic dysfunction. Circulation 1997;96:3974–3984.
44. Anand IS. Ventricular remodeling without cellular contractile dysfunction. J Card Fail 2002;8(6 Suppl):S401–S408.
45. Norton GR, Woodiwiss AJ, Gaasch WH, et al. Heart failure in pressure overload hypertrophy. The relative roles of ventricular remodeling and myocardial dysfunction. J Am Coll Cardiol 2002;39:664–671.
46. Cecconi M, De Backer D, Antonelli M, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med 2014;40:1795–1815.
47. Vieillard-Baron A. Septic cardiomyopathy. Ann Intensive Care 2011;1:6.
48. Suga H, Sagawa K. Instantaneous pressure-volume relationships and their ratio in the excised, supported canine left ventricle. Circ Res 1974;35:117–126.
49. Wallace AG, Skinner NS Jr, Mitchell JH. Hemodynamic determinants of the maximal rate of rise of left ventricular pressure. Am J Physiol 1963;205:30–36.
50. Tartiere JM, Logeart D, Beauvais F, et al. Non-invasive radial pulse wave assessment for the evaluation of left ventricular systolic performance in heart failure. Eur J Heart Fail 2007;9:477–483.
51. De Hert SG, Robert D, Cromheecke S, et al. Evaluation of left ventricular function in anesthetized patients using femoral artery dP/dt(max). J Cardiothorac Vasc Anesth 2006;20:325–330.
52. Morimont P, Lambermont B, Desaive T, et al. Arterial dP/dtmax accurately reflects left ventricular contractility during shock when adequate vascular filling is achieved. BMC Cardiovasc Disord 2012;12:13.
53. Monge Garcia MI, Jian Z, Settels JJ, et al. Performance comparison of ventricular and arterial dP/dt_{max} for assessing left ventricular systolic function during different experimental loading and contractile conditions. Crit Care 2018;22:325.
54. Ostadal P, Vondrakova D, Krüger A, et al. Continual measurement of arterial dP/dt_{max} enables minimally invasive monitoring of left ventricular contractility in patients with acute heart failure. Crit Care 2019;23:364.
55. Scolletta S, Bodson L, Donadello K, et al. Assessment of left ventricular function by pulse wave analysis in critically ill patients. Intensive Care Med 2013;39:1025–1033.
56. Vaquer S, Chemla D, Teboul JL, et al. Influence of changes in ventricular systolic function and loading conditions on pulse contour analysis-derived femoral dP/dtmax. Ann Intensive Care 2019;9:61.
57. Ali N, Shin MS, Whinnett Z. The Emerging Role of Cardiac Conduction System Pacing as a Treatment for Heart Failure. Curr Heart Fail Rep 2020;17:288–298.
58. Padeletti L, Pieragnoli P, Ricciardi G, et al. Simultaneous His Bundle and Left Ventricular Pacing for Optimal Cardiac Resynchronization Therapy Delivery: Acute Hemodynamic Assessment by Pressure-Volume Loops. Circ Arrhythm Electrophysiol 2016;9:e003793.
59. Arnold AD, Shun-Shin MJ, Keene D, et al. His Resynchronization Versus Biventricular Pacing in Patients With Heart Failure and Left Bundle Branch Block. J Am Coll Cardiol 2018;72:3112–3122.
60. Shun-Shin MJ, Miyazawa AA, Keene D, et al. How to deliver personalized cardiac resynchronization therapy through the precise measurement of the acute hemodynamic response: Insights from the iSpot trial. J Cardiovasc Electrophysiol 2019;30:1610–1619.
61. Buchner T. A quantitative model of relation between respiratory-related blood pressure fluctuations and the respiratory sinus arrhythmia. Med Biol Eng Comput 2019;57:1069–1078.
62. Miyazawa AA, Francis DP, Whinnett ZI. Basic Principles of Hemodynamics in Pacing. Card Electrophysiol Clin 2022;14:133–140.
63. Whinnett ZI, Davies JE, Nott G, et al. Efficiency, reproducibility and agreement of five different hemodynamic measures for optimization of cardiac resynchronization therapy. Int J Cardiol 2008;129:216–226.