International Journal of Clinical and Experimental Medical Sciences
2015; 1(4): 70-77
Published online December 4, 2015 (http://www.sciencepublishinggroup.com/j/ijcems)
doi: 10.11648/j.ijcems.20150104.11

Review Article

Hemodynamic Assessment Using Pressure-Volume (PV) During Mechanical Circulatory Support

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To cite this article:
Filip Konecny. Hemodynamic Assessment Using Pressure-Volume (PV) During Mechanical Circulatory Support. *International Journal of Clinical and Experimental Medical Sciences*. Vol. 1, No. 4, 2015, pp. 70-77. doi: 10.11648/j.ijcems.20150104.11

Abstract: Mechanical circulatory support devices (MCS), namely percutaneous ventricular assist devices (pVAD) are temporarily introduced to support circulation in hemodynamically compromised patients and also during mid to high risk coronary artery procedures. Their multiple responsibilities include maintaining an adequate systemic blood pressure and cardiac output to provide satisfactory end-organ perfusion in unloading of the failing ventricle, and to temporary lower myocardial contractility while reducing myocardial oxygen demand supporting favorable ventricular remodeling. To timely and quantitatively assess hemodynamics during pVAD circulatory support post-cardiogenic shock or acute myocardial infarction (MI), pressure-volume (PV) measurements are becoming progressively more appreciated as they can longitudinally evaluate the status of the support. Hemodynamically, importance of constant circulatory interrogations by PV during pVAD support lies in its capacity to "fine-tune" the device for a specific patient to work in synergy with the ailing organ. In this review basic characteristics of a diagnostic value of pressure-volume during pVAD hemodynamic support will be discussed fostering conversation about the necessity of e.g. combining pump flow with load-independent indices creating indexes that can be used to further characterize pump unloading in relation to innate cardiac contractility during axial or centrifugal flow support. Additionally, discussion about central hemodynamics during different flow support will be provided evaluating pVADs to assess its ability to work in synergy and to anticipate potential difficulties that might occur during the procedure. Brief description of recent efforts to combine PV exam with pump flow during circulatory support using pVAD and the concept of pressure-volume area (PVA) and myocardial oxygen consumption (mVO_2) during unloading will be also discussed.

Keywords: Mechanical Circulatory Support (MCS), Percutaneous Ventricular Assist Device (pVAD), Pressure-Volume (PV), Myocardial Oxygen Consumption (mVO_2), Load-Dependent, Load-Independent, Contractility

1. Basic Characteristics of pVAD Devices and Its Hemodynamic Support Using Diagnostic Pressure-Volume or Central Pressure Catheter

Mechanical circulatory support devices like pVADs can be divided into categories as described in (Table 1) based on e.g. placement of inflow and outflow cannulas, the design of its rotating components, possible interference with PV and or centrally placed pressure catheter and complication of its insertion, and also the effect they might have on myocardial contractility.

As each device is implanted and inflow and outflow cannulas are deployed, it is important to identify in advance the arrangement of e.g. pressure-volume (PV), pressure or pressure area (PA) (Swan-Ganz) catheter not to interfere with each other during recordings. For placement of cannulas and catheters it is important to use fluoroscopy guidance. Placement of PV catheter is done centrally into the left ventricular (LV) or right ventricular (RV) chamber, while secondary pressure-only catheter is restricted to the areas close to e.g. the aortic valve. In some instances pVAD device might need to be placed in advance to limit intravascular interactions, in case of e.g. intra-aortic balloon pump (IABP), percutaneous placement of PV catheter might not be possible.

Table 1. *Selected percutaneous VADs and its description.*

Device (rotating components)	Inflow/out flow cannula	Possible Interference with PV or central P catheter	Unloading CO augmentation	CPO (Watt) compared to post cardiac insult	Effect on native PVA post cardiac insult
IABCP (NA)	Prox. descend Aorta thru FA	Yes	0.4-0.5 l/min with need of native CO	Moderate decrease	Certain degree of reduction
Impella 2.5; CP or 5.0 (Axial)	LV/Aorta	Yes	2.5l/min (model 2.5) up to 4 l/min (CP- model) 5l/min (model 5.0)	Moderate decrease	Reduction
TandemHeart (Centrifugal)	LA/ Artery (Fem)	No	3.5 up to 4.5 l/min	Decrease	Reduction
ECMO (Centrifugal)	RA/ artery	No	4.5 up to 8l/min	Increase	Increase

VADs: Ventricular assist devices, IABCP: Intra-aortic balloon counter pulsation, ECMO: Extra-corporeal membrane oxygenation, PVA: Pressure-volume area, CO: Cardiac output, CPO: Cardiac power output, LA: Left atrium, LV: Left ventricle, RA: Right atrium, PV: Pressure-volume, P: Pressure, FA: Femoral artery.

2. Methodology of Hemodynamic Measurement

Briefly, the aortic blood pressure along the left ventricular pressure and volume were measured using commercially available control unit from (Transonic, Inc.) allowing real-time assessment of left ventricular volume in addition to the aortic pressure. Pigtail 7F PV catheter was inserted through the right carotid artery and positioned in the LV under fluoroscopic guidance. Pressure catheter 7F pigtail was placed in close proximity of the aortic valve into the ascending aorta using left carotid artery. Data were collected along with simultaneous two-lead electrocardiography (ECG) recordings, providing accurate timing throughout the cardiac cycle. Male swine were randomly selected to receive percutaneous circulatory support in the closed-chest setting to record baseline PV loops and preload reduction hemodynamics using inferior vena caval (IVC) occlusion during baseline (no pump support) and thru pump support. Percutaneous VADs were inserted under fluoroscopic guidance according to manufacturer's instructions. Collection and analysis of all hemodynamic parameters were performed using commercially available data acquisition system (iWorx Systems, Inc.).

3. Hemodynamic Assessment of Intra-aortic Balloon Counter Pulsation (IABCP)

IABCP is a method of using percutaneously inserted a balloon catheter into the proximal descending aorta where it is deployed. Function of deployed balloon at that location is to create similar counter pulsation as it is made by native physiological impedance to blood flow of vascular branching points ensuring partial blood return towards the aortic valve. This pVAD uses the mechanism of balloon inflation during diastole to create a temporary increase of aortic blood pressure. During systole balloon is deflated allowing efficient afterload. Interestingly, simulated by Kolyva et al. blood volume displaced towards the coronary circulation during balloon inflation is no more than 10% of balloon volume, nonetheless it is enough to improve coronary artery perfusion.[1] One of the hemodynamic limitation of this mechanism is device timing. As balloon inflation follows cardiac systole during aorta's diastole when its elastic wall is in recoil, the inflating balloon is at this time is trying to expand it.[1] As a result of these divergent actions, the fluid volume that can be stored in the aortic wall is smaller than the volume that can be stored when the aorta can freely expand. Just how much of this volume storage in the aorta plays role in overall ventriculo-arterial coupling or improvement of cardiac power output (CPO) or its influence on mVO_2 or preload recruitable stroke work (PRSW) is yet to be thoroughly accounted for using e.g. direct apical insertion of the pressure-volume catheter. Other hemodynamic related limitations of IABCP include its dependence on inner mechanical and electrical cardiac timing at balloon inflation/deflation triggered from electrocardiogram or from aortic pressure wave tracings.[2] When the aortic pressure wave intensity analysis was performed, during switched on balloon autoregulation, systolic unloading measured by tension time index (TTI) was decreased. Balloon deflation significantly altered diastolic time index (DTI) helping to generate a backward decompression wave with "suction-like" effect on the aortic blood flow helping LV unloading.[3] During diastolic augmentation, inflation of balloon created a backward expansion-like wave with final effect on pushing blood flow towards the aortic root/coronary sinus, increasing diastolic aortic blood pressure and coronary perfusion pressure.[3] When autoregulation was switched off and when premature balloon deflation occurred, backward wave was generated with less intensity (lesser increase in final diastolic aortic blood pressure). Others also showed that properly triggered IABCP (1:1) had a beneficial effect on heart failure (HF) patients by decreasing aortic impedance hence improving ejection by decreasing afterload while lessening end-systolic pressure (ESP) and end-systolic volume (ESV) as early as during first 4 cardiac cycles.[4] Premature IABP balloon inflation just before the aortic valve opening in late systole created undesirable level of afterload impairing not only immediate systole but next cycle relaxation phase e.g. isovolumetric relaxation time (Tau) and end-diastolic pressure (EDP). Clinically, the IABCP

is used during HF or during cardiogenic shock. Modulation using animals has to be performed at this stage to closely recapitulate these clinical conditions. Moreover, as shock settles in derangements of electrical stimuli occurs, decreasing ability to effectively use ECG-R wave triggering. Animal models might be used to set up a variety of IAB counter pulsations to e.g., determine the role of counter pulsations in regulation of oxygen supply vs. oxygen consumption. Recent development also uses subendocardial viability ratio (SEVR),[5] as a useful parameter coming from the ratio between diastolic pressure-time index (DPTI), an estimate of myocardial oxygen supply using coronary driving pressure in diastole and diastolic time and systolic pressure-time index (SPTI), an estimate of myocardial oxygen consumption-mVO_2.[6] Overall, to improve the ventricular unloading while supporting contractility of failing myocardium using counter pulsation, empirical hemodynamic study using PV and central/peripheral pressures combined with pump flow interrogation is necessary to determine whether the deployment of several smaller counter pulsating balloon(s) of a different size(s) at branching point location(s) might be more desirable.

4. Centrifugal and Axial Continuous Pumps During Hemodynamics

Design of the rotating components is one of the major distinctions between pVADs. In case of centrifugal (CF) pump, rotating component acts as a spinning disk with blades, while in case of axial pump rotating elements operate like a propeller enclosed in a cylinder.[7] TandemHeart, the CF pump works in parallel with native myocardium while Impella, the axial pump works in series. Difference in pump performance plays role in determining the relationship between pump flow and pressure, difference across the pump ports known as (Δ P or pump head pressure) is relevant to operating pump speed. For each pump characteristic relationship between Δ P and volume flow exists that is relevant to speed of pump rotations per minute (RPMs). Major difference in pump head curves is between axial flow pumps that has downward curve-linear shape (where small changes of pressure lead to higher volume flow, therefore overall less pulsatility) as compared to CF (TandemHeart) designed pump that have flat-head curve-linear relationship (where across large volume flows only small pressure changes occurs leading to higher inner pump-pulsatility).[7] Head pump pressure relationships can be observed while using e.g. axial pump during situations of sudden decrement of flow (bleeding) as axial pump might encounter rapid increase of suction on inflow cannula as pump pull the hardest at low flow situations. During these situations axial pumps will still try to empty the ventricle, which can lead to arrhythmias, haemolysis or pump shutting down.[7] Implication of hemodynamic control and value of PV exam combined with pump flow during circulatory support using MCS (pVAD) are discussed in next chapters.

5. Importance of PV Exam During Circulatory Support Using MCS (pVAD)

Functional assessment of each pVAD benefits from using hemodynamic diagnostic tools to objectively determine quality and quantity of unloading to directly fine tune each device for a patient. For the duration of chamber unloading measurements of PV load-dependent indices including (preload and afterload) in the left or right ventricle(s) needs to be continuously monitored and recorded as changes occurring abruptly while "on pump or off" pump. To simplify, cardiac preload is characterized by the end diastolic volume EDV (or sometimes by EDP), and the ventricle's afterload is characterized by the effective arterial elastance (Ea=ESP/SV). New parameters specific to pVADs were introduced by SHOCK investigators in 1999 using aortic contra pulsation.[8] These parameters include cardiac power output (CPO) and cardiac power index (CPI). Both are PV load-dependent parameters (CPO= SW*HR; in Watts (W), while the CPI is the CPO per body surface area (W/m^2). Another more recent report using this calculation during pVAD was published by Møller-Helgestad,[9] where CPO was calculated as follows (CPO=CO*MAP/451). This formula is using measurement of (CO) by using PA catheter dilution technique along with measurement of mean arterial pressure (MAP) during unloading thus taking into consideration the end-organ perfusion. Clinical values of CPO associated with negative outcomes (higher in-hospital mortality) e.g. in cardiogenic shock patients are reported as being less than 0.53 W.[10] One caveat of using the PA dilution technique to report the CO during pump unloading is that pump in parallel might influence saline bolus travel time, influencing final CO and as a result CPO read out. Attention has to be paid when reporting other load-dependent parameters while using different pVADs (in series vs. in parallel) while measuring saline travel time using dilution technologies.

It is speculated that post-acute myocardial infarct (AMI), improvements using pVAD is through lowering the EDP as most of coronary blood flow occurs during diastole while immediate improving of oxygen balance greatly benefits the ischemic myocardium. As myocardial tissue reacts on AMI by further diminishing blood perfusion, it creates foundation for activating mechanism of temporary release of catecholamines, increasing myocardial contractility and peripheral blood flow. As catecholamines are released they increase mVO_2 and chances of arrhythmias and calcium cycling derangements. As MAP in AMI and also in cardiogenic shock decreases, the RA pressure increases. Important balancing act, adding diagnostic hemodynamic value to any pVAD, would be to support the stabilization of circulation by increasing the driving MAP while lowering the RA pressure supporting regional contractility by conserving calcium cycling as it was recently observed by Wei et al.[11] Diagnostic value of PV would be then to "fine-tune" the pVAD, taking in consideration both central

and peripheral innate hemodynamics providing continuous assessment of e.g. arterial elastance and ventriculo-arterial coupling (Ea/Ees) helping to interrupt this vicious circle.

6. Historical Perspectives on Values of Load-Independent PV Exam During Circulatory Support Using MCS (pVAD)

In the past cardiac ionotropy (contractile properties of myocardium) unaffected by preload or afterload, also known as myocardial contractility; the *load-independent* indices; were derived from measuring pressure only as in case of the left ventricle pressure (LVP) or combined with LVP's first derivate dpdt max or linking LV dpdt max with HR[12] or further arrangements with ECG i.e. using ECG Q wave in combination with LVP were made as in e.g. two index comparison called (QA interval) at Figure 1.[13,14]

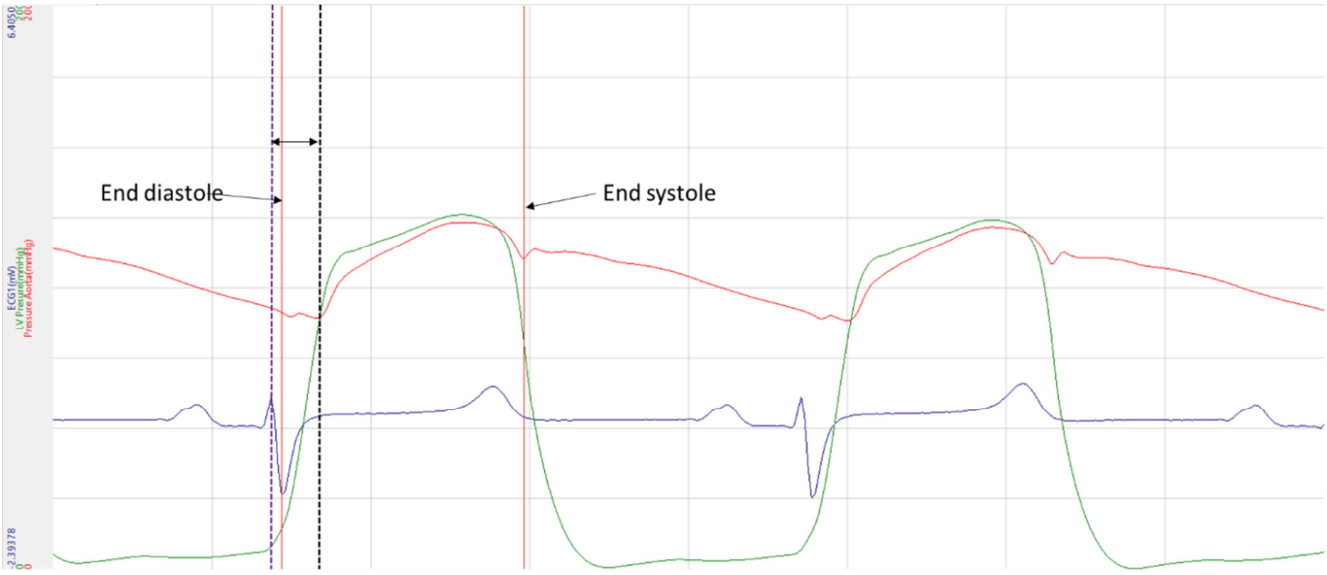

Figure 1. *QA interval is selected by the purple dashed line marking the Q point (Q wave is a negative deflection preceding R wave) on the ECG while the black dashed line marks the beginning of the aortic valve opening.*

Some investigators used formulas to limit the influence of preload by relating dpdt max to peak isovolumic pressure (dpdt max/PIP LVP), but later all these relationships were also deemed to be preload dependent. As all pressure-only parameters that can be derived or directly observed depend on some levels on the preload, investigators shifted attention to combination of pressure-volume derived myocardial contractility. Using PV during different loading conditions, researchers are able to quantify e.g. the curve linearity of the end systolic elastance along with volume-axis intercept (V_0 notch). Assessment of myocardial contractility can be provided while on pump as compared instances during the off-pump cycles or under different pump flow speed. The relationship of end systolic pressure with end systolic volume (ESPVR) is utilized for such assessment. In this assessment it is important that the slope of the ESPVR, the end systolic elastance, and proper V_0 is determined during unloading while (on pump) with matching temporary preload reduction while using the same pump speed.[15] In case of incremental pump flow, increase of peripheral resistance (afterload) might occur activating innate baroreflexes causing decrease of cardiac cycle (increase of heart rate) and decrease of stroke volume (SV) leading to increase of arterial elastance with effect on end systolic elastance. For this assessment the relationship of ventriculo-arterial coupling (Ea/Ees) can be utilized.

Using other derived parameters to compare myocardial contractility, the relationship of pre-load recruitable stroke work (PRSW) describes a linear relationship of ventricle stroke work (SW) with the end diastolic volume. Single beat estimation of PRSW by Karunanithi & Feneley[16] might be able to eliminate need of caval occlusions in future, helping to capture this load independent contractility index during course of different pump unloading speeds. Advantage of PRSW in the assessment of pVADs might stem from the fact that SW diminishes at much faster rate with preload reduction as compared to ESP or dP/dtmax. Consequently, a smaller amount of extrapolation of the measured data is necessary to determine the volume-axis intercept, making this relationship more suitable as compared to dpdt max vs. EDV or ESPVR.[16] Using preload reduction during "pump on" and "pump off" cycles can help to characterize diastolic properties of myocardial contractility, namely the ventricular stiffness. Using the end diastolic elastance curve-linear relationship, both the (active/passive) diastole contractile properties can be scrutinized. Physical models are instrumental in modeling pVAD unloading. During pump unloading preload/afterload changes occur, influencing myocardial contractility. Currently, myocardial contractility is probed using different unloading speeds of pVAD in combination with additional preload alteration using caval occlusions. Additional preload reductions, however have to be limited to the minimum as e.g. 10 sec as described by Karunanithi & Feneley.[16] Moreover,

blocking autonomic system, enables to prevent excessive autonomic reflex during these preload alterations. The overall value of empirical testing of myocardial contractility is to assess differences between the pVADs (in-parallel vs. in-series), its unloading capacities and to enable constant read outs of myocardial ionotropic and coupling properties at different stages of experimentation. Detailed research methods might be used in future to discover, e.g. rate of depletion of calcium-handling reserves, the degree of reduction of contractile reserves or impairment of myocyte relaxation capacities and its relationships to cardiac muscle oxygen levels.

7. Efforts to Combine PV Exam with Pump Flow During Circulatory Support

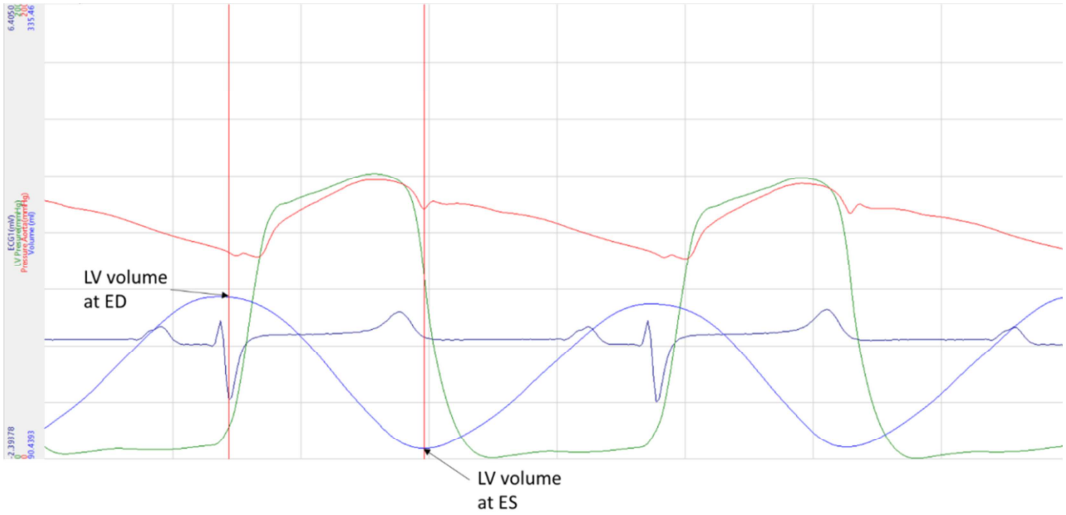

Figure 2. *Baseline example of LV volume at end diastole (ED) and end systole (ES). A pressure - volume catheter was inserted past the aortic valve and positioned in midline of the LV. Pressure catheter was left in place just before the aortic valve.*

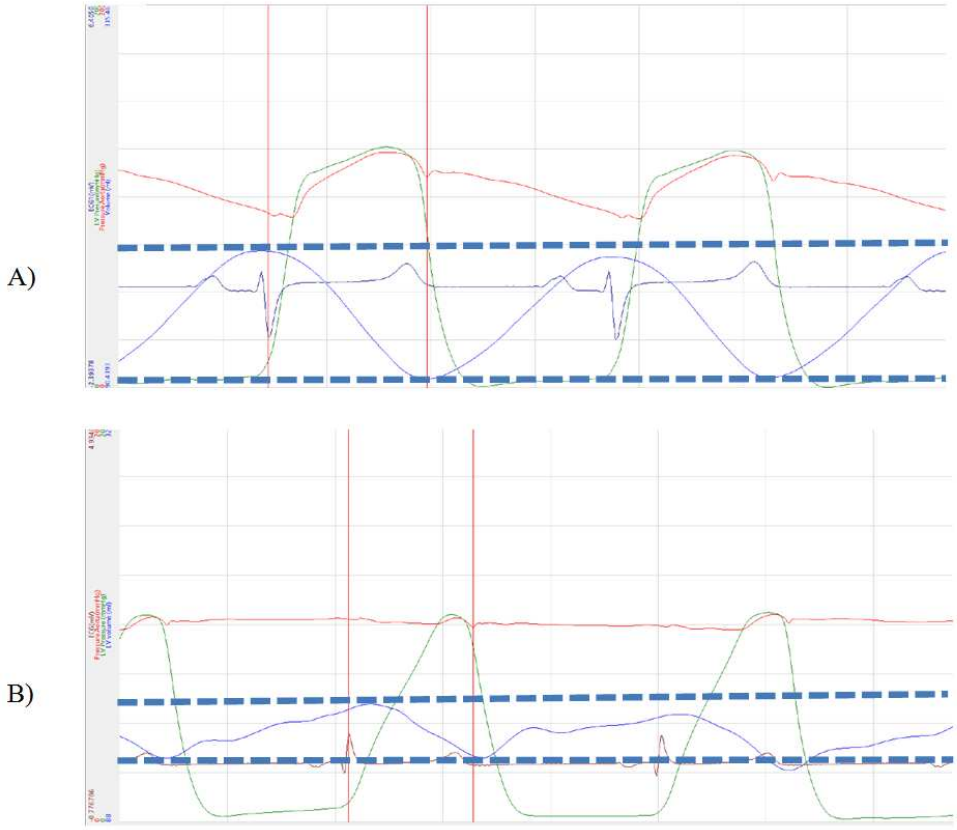

Figure 3. *Direct visualization of LV unloading using B) MCS (pVAD) device as compared to A) baseline. Using blue dashed lines the level of volume registered in LV can be plotted during each cardiac cycle.*

During the period of 2005-2010 investigators concentrated on both the parameters important to native circulation while trying to combine it with simulated pump flow and pressure. Here the triple product-TP idea came from, where TP is the product of LV systolic pressure, heart rate and the maximal time-derivative of LV pressure. TP = LVSP* dpdt max*heart rate (HR).[17] The slope of the linear regression between the LV-TP and the LV EDP was again evaluated while trying to alleviate preload dependency by dividing pressure values by another pressure index.[17] Some have used the index derived from the pump flow (IQ). IQ was defined as the slope of the linear regression between the maximal time derived pump flow (dQ/ dtmax) and its peak to peak amplitude variation called (QP2P).[18] In 2011 Ferreira et al., developed method to assess myocardial contractility using pump flow based on a curvilinear relationship between pump EF (pEF) and pump diastolic volume called (Vd).[19] Some drawbacks in these calculations stemmed from not properly capturing the Vd and Vs i.e. (Ts and Td) effectively matching the occurrences of diastole and systole using the LVP, AoP and Qt tracings and again its dependence on preload.[19] PV load-dependent original and (or) derived/combined parameters such as pEF are important to collect and (or) calculate to reflect on different ability of each device to unload, nevertheless both original ejection fraction (EF) or fractional shortening (FS) are also deemed to be highly dependent on afterload. At the present time, PV catheter measurements allows assessment of instantaneous load-dependent relationship at different pump derived flows throughout individual cardiac cycle, moreover PV enables tracking long-term changes caused by the unloading (Figure 2 and 3).

8. Limitations and Experimental Improvements in PV Diagnostic of pVAD Function

Taking additional hemodynamic records combined simultaneously with pump derived flow are important to describe the chain of events during the unloading as very high unloading speed, e.g. might invoke sudden decrease of cardiac output, terminating study in cardiogenic shock or at severe ventricular arrhythmia(s). Besides, having knowledge about central and peripheral hemodynamics as e.g. post-valvular pressures are important to ensure detection of any regurgitations.[20] Pressures collected using PA catheter i.e. central venous pressure (CVP), right ventricle pressure (RVP), pulmonary artery pressure (PAP), wedge pressure (PCWP) are also vital, offering additional hemodynamic information specific to pVAD.[21] Other practical considerations of experimental set up include e.g. using suitable animal model (amount of unloading vs. circulating blood amount), (the size of in/outflow cannula vs. animal vessel diameter), (percutaneous insertion site vs. length of outflow cannula), (similarities of human coronary artery anatomy to model e.g. AMI), (ability to perform aortogram prior to device insertion) and others. Recording these parametric data further characterizes pVAD unloading in relation to e.g. end diastolic elastance, hydrodynamic performances of pump in relation to cardiac energetics, unloading speed in relations to ability to assess cardiac contractile recovery, afterload vs. preload sensitivity, control of suction or creation of vacuum. Lastly, creation of new parameters to be captured as it is in recent simultaneous recordings of pVAD pump flow[19] would be warranted. Future experimental recordings should combine pVAD pump flow with single beat-elastance (SBE), or single-beat PRSW enabling to collect information about innate cardiac ionotropic state while "on or off the pump" eliminating need of temporary preload reductions using caval occlusions. Optimal pump flow speed would then be set based on instantaneous ventriculo-arterial coupling and matched to myocardial contractile performance.

9. The Concept of Pressure-Volume Area (PVA) and Myocardial Oxygen Consumption (mVO2) During Unloading

To maintain myocardial contraction, conversion of chemical energy of metabolic substrates is taking place into mechanical energy. As for high energy requirements of the myocardium, with relatively low content of high energy compounds adenosine triphosphate (ATP) and creatine phosphate, both have to be constantly generated at a high rate. Majority of ATP is generated from substrates of different caloric value processed directly by the myocardium via the oxidative phosphorylation. As the myocardium is the main organ to liberate the energy, amount of oxygen in (ml) has to be matched per gram of the substrate. Consequently, the unit of oxygen consumption by myocardium mVO_2 can be used as a surrogate of the total myocardial energy utilization. Native energy released and used has different amount of efficiency. This efficiency in PV hemodynamics can be measured by e.g. PVA, which represents the ratio between ventricular stroke work (SW) and its potential energy (PE). PE is the area defined by relationships of ESPVR and end-diastolic pressure volume relationship (EDPVR) for cardiac cycles during temporary preload reduction. Moreover, the myocardial efficiency can be calculated as SW/PVA (Figure 4).

As PE concept goes back to innate ATP being produced by myocardium (>95%), thus mVO_2 can be correlated to PE as it is mechanical-elastic energy stored in the myocardial syntitium ready to be released along with the heat generated during uncoupling. To expand the concept, during e.g. the acute MI, as conditions of inequality between energy accessibility on demand rise it leads to reduction of PE since the oxidative phosphorylation becomes reduced as the oxygen availability is shortened.

Currently, temporary preload reduction has to be performed empirically to address PE during different types of unloading

(in parallel vs. in series). PE can also be associated with an amount of unloading during different pump speed. At this time it is very complex to use an estimation of single beat-elastance to characterize PE as would be e.g. SBE_{PE} during unloading (in parallel vs. in series). I envision that multiple research teams would highly benefit using SBE_{PE} eliminating the caval occlusions. Presently PE allows comparisons between pVADs (in series vs. in parallel) using contemporary methods of temporary unloading. During pVAD unloading the energy consumption along with mVO_2 measured in native myocardium should be reasonably alleviated. Table 2 shows expected outcomes during unloading after temporary introduced ischemia (AMI), while still using contemporary methods of temporary unloading that is using the caval occlusions and not SBE_{PE}.

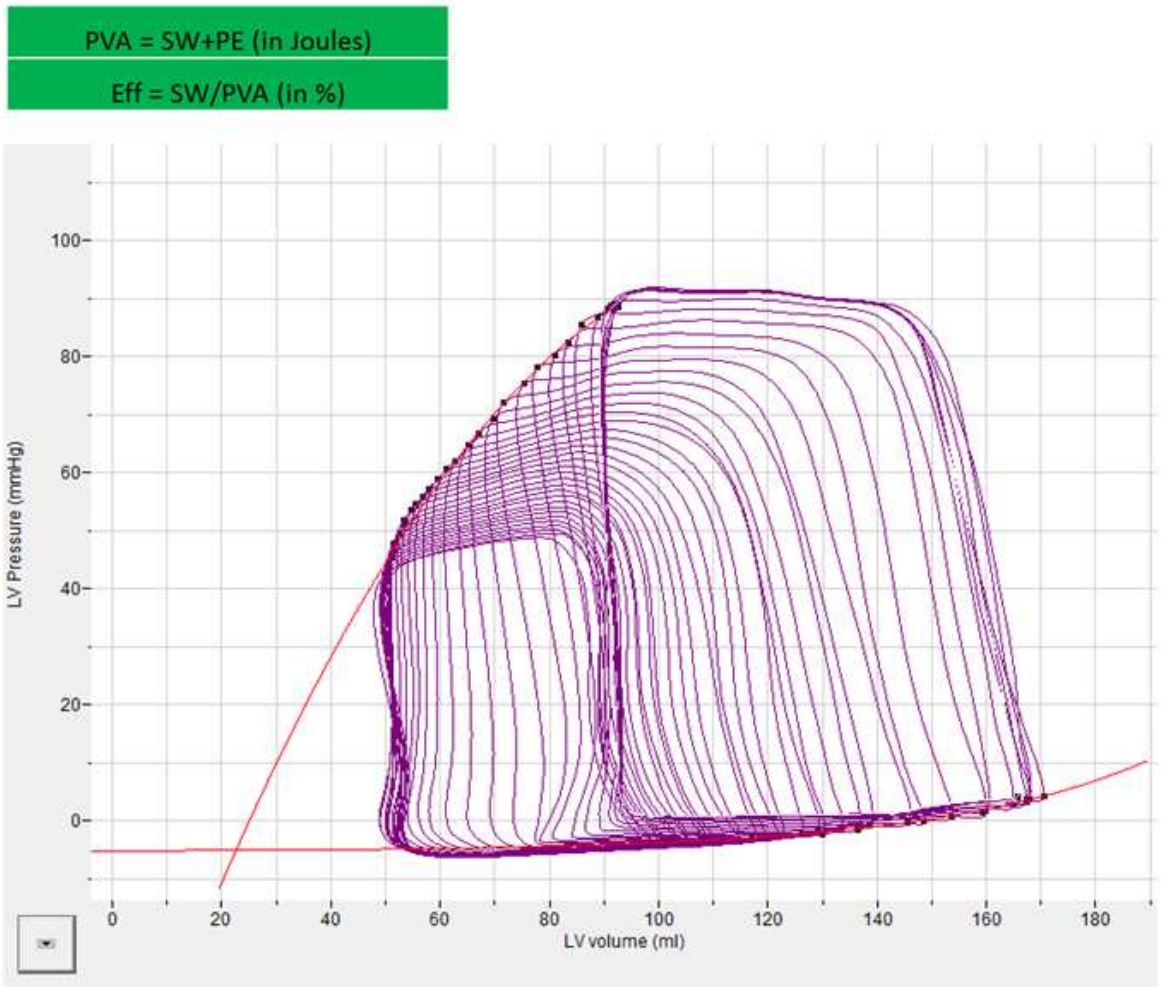

Figure 4. *PVA and myocardial Efficiency formulas along with a baseline example of preload reduction by occlusion of inferior vena cava (IVC) balloon occlusion.*

Table 2. *Changes taking place during unloading using MCS (pVAD). Both, PVA and overall efficiency of myocardium has to be minimized using pVAD during unloading to decrease necessities of excess consumption of oxygen and ATP as compared to post injured and failing heart.*

Post preload reduction	HR (bpm)	SW (Joule)	PE (Joule)	PVA (Joule)	Eff (%)
Values during pVAD support compared to injured native myocardium	Higher	Lower	Reduced	Reduced	Lower

MCS: Mechanical circulatory support, pVAD: Percutaneous ventricular assist devices, ATP: Adenosine triphosphate, HR: heart rate, SW: Stroke work, PE: Potential energy, PVA: Pressure volume area.

As heart is trying to cope with ischemic injury, temporary increase of PE along with SW is observed (not shown in table). Cardiac oxygen consumption is also increased immediately post ischemia as the oxygen cost of the contractile function is increased.[22]

To conclude, Saku et al. has recently observed that as soon as the post-ischemic muscle is relieved from its load (entirely not partially) to minimize its temporary energy and oxygen costs, this enables contractile cardiac machinery to partially recover and it leads to a remarkable decrease of the infarct size.[23] Given that the post-AMI preload is still creating energy and oxygen demand, placed on the ailing organ, this research brings additional evidence that volume data are important to be correlated with pressure while interrogating pump

unloading. The role of PV during the longitudinal assessment of the amount of unloading is necessary during HF and cardiogenic shock to evaluate status of the support. Moreover, the importance of constant circulatory interrogations (possibly hundreds of cardiac cycles recorded) by PV during pump support benefit "fine-tuning" pVAD to work in synergy with the ailing organ. Conversation about necessity of combining pump flow with load-independent values while creating indexes like SBE_{PE} that can be used to further characterize pump unloading in direct relation to innate cardiac contractility during axial or centrifugal flow support might need further empirical evidence to comprehend the benefits.

Conflict of Interest

None declared

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