

Cardiovascular pressure-volume loops

P Motshabi 

Department of Anaesthesiology, School of Clinical Medicine, Faculty of Health Sciences, Charlotte Maxeke Johannesburg Academic Hospital, University of the Witwatersrand, South Africa
Corresponding author, email: palesa.motshabi@wits.ac.za

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Introduction

Pressure-volume (PV) loops are a modality used to depict the relationship between pressure and volume in the ventricle.¹ They are used to represent ventricular performance during a cardiac cycle. A loop is drawn for a single cardiac cycle that is divided into four primary phases. The four phases are ventricular filling (diastole), isovolumetric contraction (systole), ejection (systole), and isovolumetric relaxation (diastole).

Acquiring PV measurements

PV loops are mostly performed in animal studies.² A PV catheter is inserted into the ventricle being studied. This is achieved either through the carotid/aorta in a closed chest or the ventricle in an

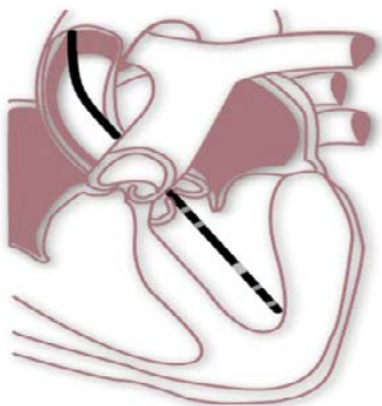


Figure 1: Aortic approach of the PV catheter¹

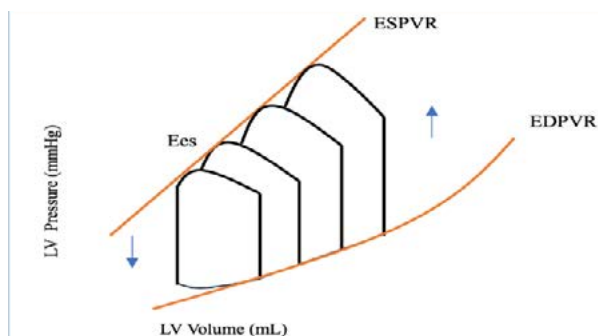


Figure 2: Illustration of simulated preload conditions concerning pressure and SV

*LV - left ventricle, Ees - end-systolic elastance, ESPVR - end-systolic pressure volume relationship, EDPVR - end-diastolic pressure volume relationship

open chest (Figure 1). Changes in volume, pressure, and inotropy are simulated as measurements are taken.

An example of volume simulation is achieved with the manipulation of the inferior vena cava to change the loading conditions to simulate different preload scenarios (Figure 2). The haemodynamic and physiological events that can be derived from these loops include stroke volume (SV), cardiac output, ejection fraction, and myocardial contractility.

Characteristics of a PV loop (Figure 3)

The PV loop depicts four phases:

1. Phase a – ventricular filling (diastole)
2. Phase b – isovolumetric contraction (systole)
3. Phase c – ejection (systole)
4. Phase d – isovolumetric relaxation (diastole)

Phase a

Phase a is characterised by the mitral valve opening and the filling of the ventricle. This phase is preceded by a phase where the left ventricular (LV) pressure drops significantly below the volume of the full left atrium. The volume in the LV at the beginning of the phase is termed end-systolic volume (ESV). The volume in the LV increases, accompanied by a gradual increase in pressure as the ventricle fills up. This phase ends with a full ventricle with a higher pressure than in the atrium. The mitral valve, therefore closes. The volume in the LV at this point is termed end-diastolic volume (EDV). The difference between the EDV and the ESV determines the SV. This phase is termed diastole.^{3,4}

Phase b

Phase b begins with a full LV and closed mitral and aortic valves. The LV then generates pressure against both closed valves while the volume remains the same (isovolumetric contraction), aimed at exceeding the pressure in the aorta to generate flow. The end of this phase occurs when the aortic valve opens.^{3,4}

Phase c

Phase c begins when the aortic valve opens, and flow through the aortic valve starts while the ventricle continues to generate pressure to a peak ventricular pressure before it begins to relax. This phase is termed systole.^{3,4}

Phase d

Phase d begins with the closure of the aorta whilst the mitral valve remains open with a constant volume (isovolumetric relaxation). The LV continues to relax until the LV pressure is below the left atrial pressure for phase a to begin again.^{3,4}

The pressure and volume measurements are performed directly at the catheter as load-independent parameters. Pressures are plotted on the y-axis, and volumes are on the x-axis. The following parameters can be determined from the measurements:^{2,3}

- SV – the volume of blood pumped from the left ventricle per beat ($SV = EDV - ESV$).
- Stroke work (SW) – work performed to eject a volume of blood [$SW = SV \times \text{mean arterial pressure (MAP)}$], the yellow area on the graph.
- **End-diastolic pressure-volume relationship (EDPVR) slope** – ventricular compliance.
- **End-systolic pressure-volume relationship (ESPVR) slope** – ventricular inotrope.
- End-systolic elastance (Ees) – compliance/cardiac contractility.

Valve pathology and PV loops

Mitral stenosis (Figure 4)

Stenosis of the mitral valve presents a mechanical obstruction to the filling of the LV. The effect is reduced preload (red loop), represented by a decrease in EDV. The lower EDV leads to a lower

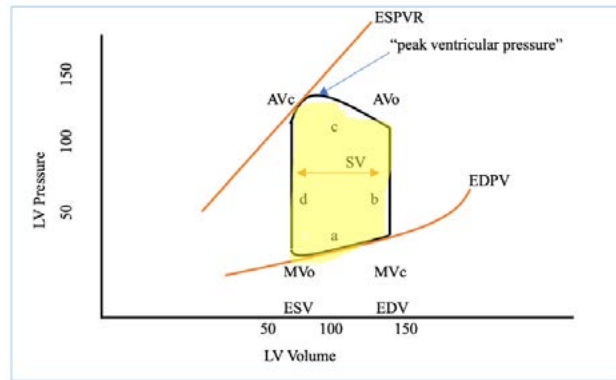


Figure 3: Characteristics of the PV loop
 *LV - left ventricle, Ees - end-systolic elastance, ESPVR – end-systolic pressure-volume relationship, EDPVR – end-diastolic pressure-volume relationship, AVc – aortic valve closure, AVo - aortic valve opening, MVo – mitral valve opening, MVc – mitral valve closure, ESV – end-systolic volume, EDV – end-diastolic volume, SV – stroke volume, phases a,b,c,d – ventricular filling /diastole, isovolumetric contraction/systole, ejection/systole, and isovolumetric relaxation/diastole.

ventricular filling pressure left ventricular end-diastolic pressure (LVEDP).²

The heart contractility is, therefore reduced through the intrinsic Frank-Starling mechanism leading to lower peak ventricular pressure. Therefore, there is a lower ESV, however not enough to offset the reduction in EDV and consequent lower SV. Ventricular SW is also reduced.⁵

Mitral regurgitation (Figure 5)

Mitral regurgitation is characterised by losses in both true isovolumetric contraction and relaxation. This leads to the loss of phases b and d in the PV loop. The ESV is reduced to flow back into the atrium during isovolumetric relaxation, and the EDV is increased due to unregulated excessive flow from the atrium during diastolic filling.⁶

This leads to increased LVEDP, SV, and SW. The ventricle cannot generate an adequate peak pressure and, therefore, ejection into the aorta is reduced.

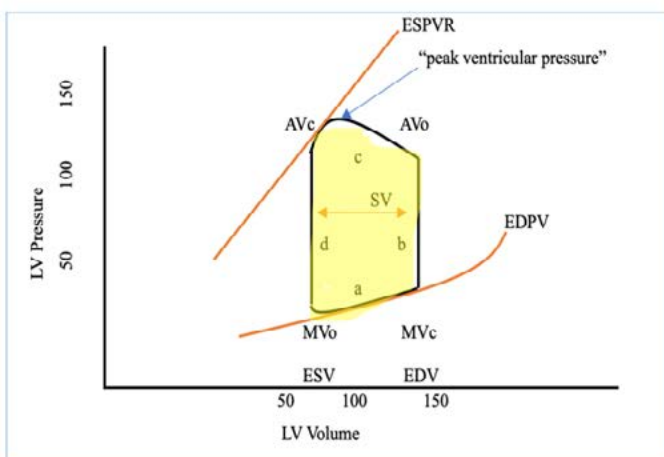
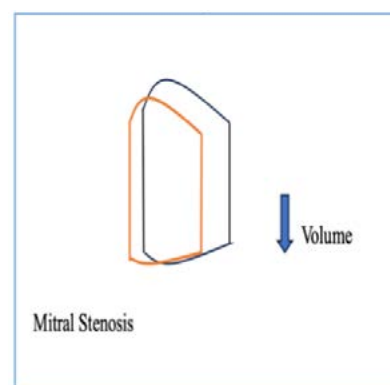
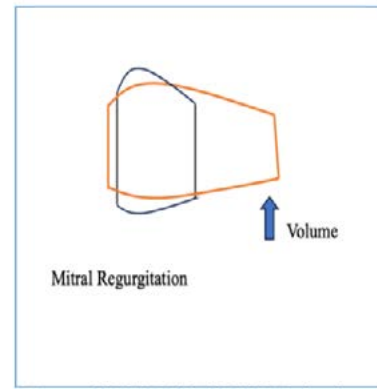
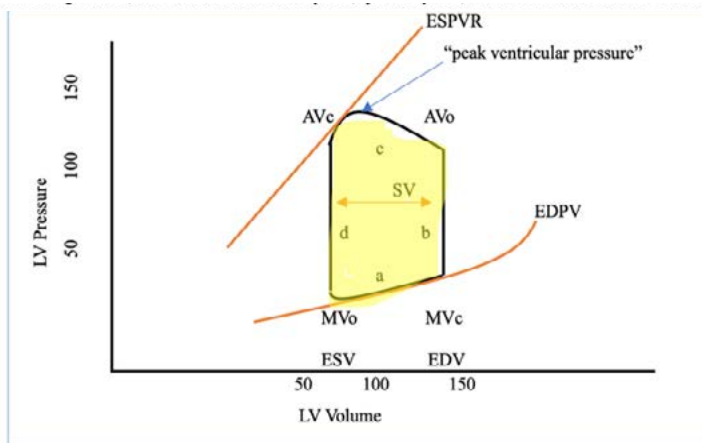


Figure 4: Mitral stenosis
 *LV - left ventricle, Ees - end-systolic elastance, ESPVR – end-systolic pressure-volume relationship, EDPVR – end-diastolic pressure-volume relationship, AVc – aortic valve closure, AVo - aortic valve opening, MVo – mitral valve opening, MVc – mitral valve closure, ESV – end-systolic volume, EDV – end-diastolic volume, SV – stroke volume, phases a,b,c,d – ventricular filling /diastole, isovolumetric contraction/systole, ejection/systole, and isovolumetric relaxation/diastole.



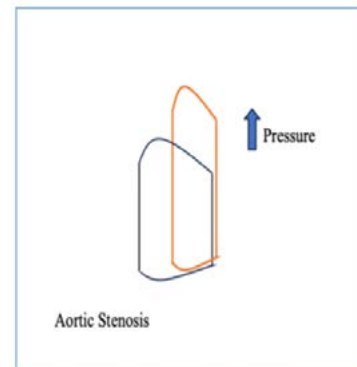
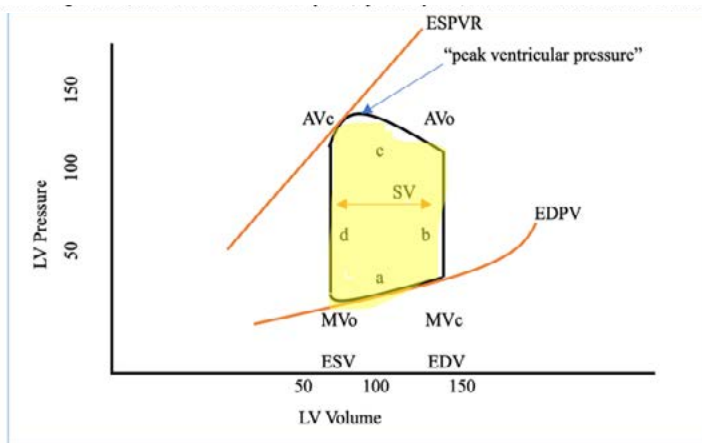
Decreased ESV, EDV, SV, SW



Increased LVEDP, SW, SV, EDV. Reduced ESV.

Figure 5: Mitral regurgitation

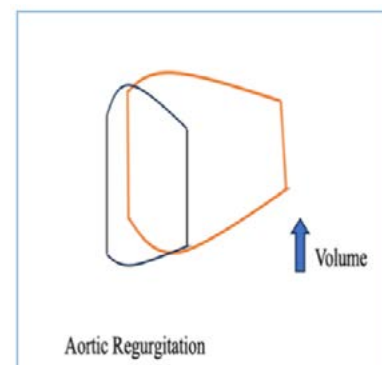
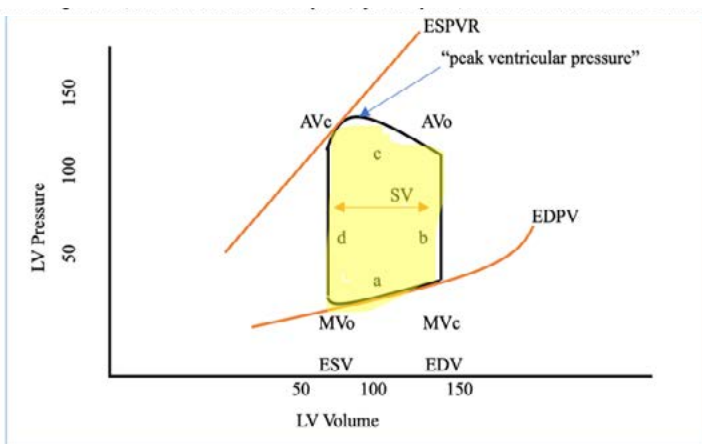
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Increase in EDV, peak pressure, decreased SV.

Figure 6: Aortic stenosis

*LV - left ventricle, Ees - end-systolic elastance, ESPVR – end-systolic pressure volume relationship, EDPVR – end-diastolic pressure volume relationship, AVc – aortic valve closure, AVo - aortic valve opening, MVo – mitral valve opening, MVc – mitral valve closure, ESV – end-systolic volume, EDV – end-diastolic volume, SV – stroke volume, phases a,b,c,d – ventricular filling /diastole, isovolumetric contraction/systole, ejection/systole, and isovolumetric relaxation/diastole.



Increased ESV, EDV, LVEDP.

Figure 7: Aortic Regurgitation

*LV - left ventricle, Ees - end-systolic elastance, ESPVR – end-systolic pressure volume relationship, EDPVR – end-diastolic pressure volume relationship, AVc – aortic valve closure, AVo - aortic valve opening, MVo – mitral valve opening, MVc – mitral valve closure, ESV – end-systolic volume, EDV – end-diastolic volume, SV – stroke volume, phases a,b,c,d – ventricular filling /diastole, isovolumetric contraction/systole, ejection/systole, and isovolumetric relaxation/diastole.

Aortic stenosis (Figure 6)

Aortic stenosis presents a mechanical obstruction to ventricular outflow. The ventricle has to generate a very high peak pressure to overcome this pressure for ejection (phase c). An inadequate volume is ejected with residual volume at the end of systole, represented as a high ESV.

During diastole, there is an elevation in preload due to volume incoming from the atrium added to the increased ESV. This leads to an increase in EDV. An increased EDV requires the ventricle to generate very high pressures during isovolumetric contraction following the Frank-Starling mechanism to eject SV. There is, however, still a reduction in SV in the face of an increase in SW.⁷

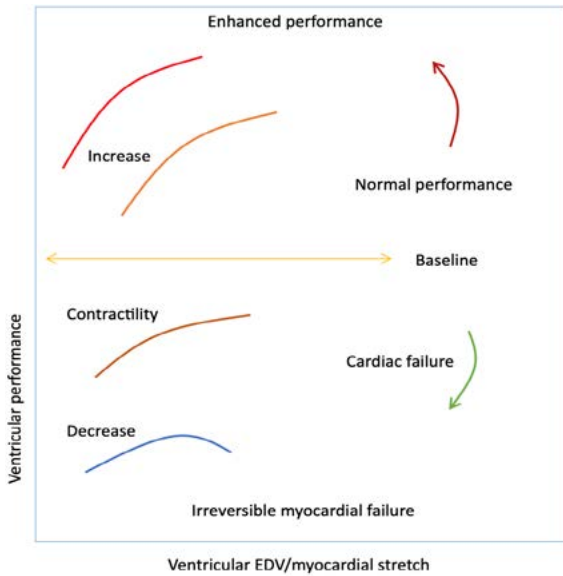


Figure 8: Ventricular performance

Aortic regurgitation (Figure 7)

Aortic regurgitation is characterised by the failure to close during both isovolumetric contraction and relaxation. The ventricle starts filling up during isovolumetric relaxation before the mitral valve opens.

This leads to an increase in ESV. During true diastolic filling, the ventricle continues to fill up from two sources, the mitral and aortic valves, leading to an increase in EDV. The increase in EDV is accompanied by an increased LVEDP. Isovolumetric contraction is shortened with the ventricle reaching a peak pressure above aortic pressure through activation of the Frank-Starling mechanism to increase the force of contraction.⁴

Frank-Starling relationships

The Frank-Starling mechanism is an intrinsic cardiac response to changes in heart rate or SV (Figure 8).^{8,9}

The mechanism was defined by Otto Frank in the 19th century using isolated frog hearts. He found that the strength of the ventricular contraction was increased by the level of the stretch of the ventricle before contraction. Ernest Starling and his group added to this knowledge by discovering that increased venous return increased the LVEDP, increasing the SV. On the contrary, they described the decrease in SV as related to reductions in venous return.^{9,10}

Systolic dysfunction (Figure 9)

Ventricular systolic dysfunction is characterised by reduced contractility. In prolonged failure of contractility, the ventricle dilates and retains more fluid as ventricular emptying is incomplete. There is a compensatory increase in preload as SV decreases. There is a downward shift in the Frank-Starling

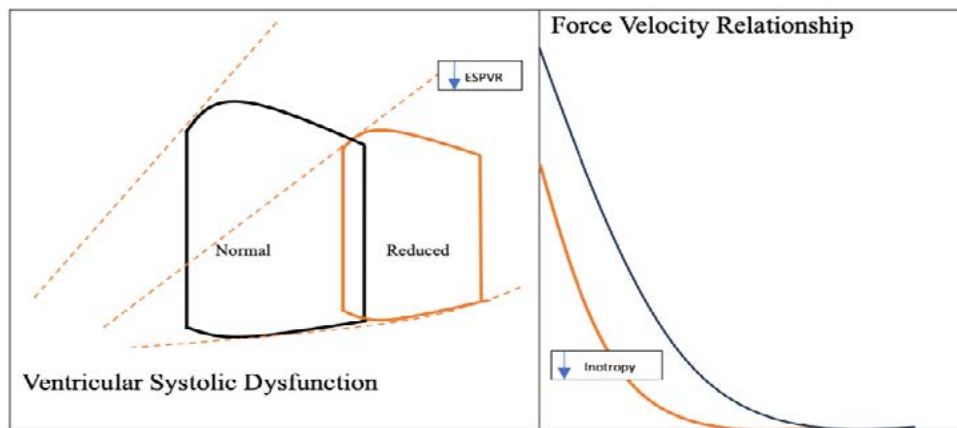


Figure 9: Ventricular systolic dysfunction

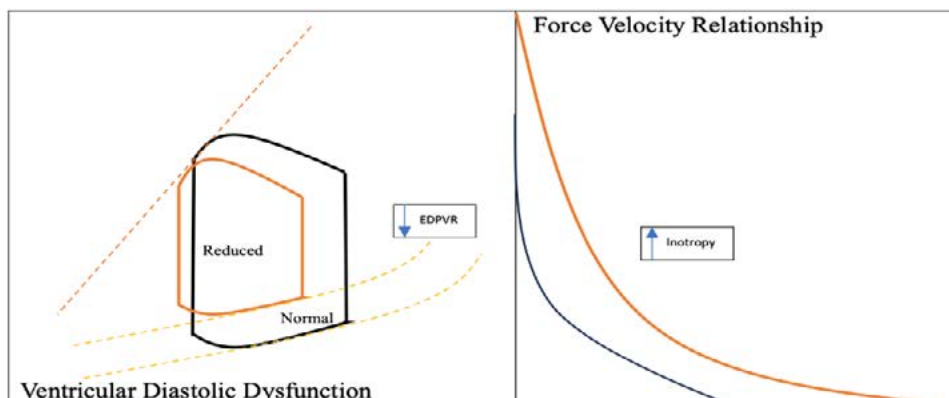


Figure 10: Ventricular diastolic dysfunction

relationship/curve.^{3,4} The PV loop of a systolic dysfunctional ventricle shows an increase in ESV, a compensatory increase in EDV, reduced SV, a normal EDPVR, and a depressed ESPVR slope.⁶

Diastolic dysfunction (Figure 10)

Diastolic dysfunction occurs in ventricles that are either thickened or stiff. These ventricles have a lusitropic challenge.^{3,4} The ventricle has an reduced EDV and increased EDP, fails to generate a supra-aortic peak pressure, and therefore eject a reduced stroke volume. The ESPVR is preserved whereas the EDPVR is reduce. Contractility may be increased.^{6,11,12}

ORCID

P Motshabi  <https://orcid.org/0000-0001-9990-6336>

References

1. Knottenbelt M. Pressure-volume catheter placement in the left ventricle - carotid vs. apical approach. [Internet]. ADInstruments; 2020. Available from: <https://www.adinstruments.com/blog/Left-ventricular-catheterization-carotid-vs-apical-approach>. Accessed 12 October 2023.
2. Miranda-Silva D, Sequeira V, Lourenço AP, Falcão-Pires I. Assessing rodent cardiac function in vivo using hemodynamic pressure-volume loops. *Front Physiol.* 2022;12:751326. <https://doi.org/10.3389/fphys.2021.751326>.
3. Klabunde RE. Ventricular pressure-volume relationship. [Internet]. Cardiovascular physiology concepts; c1998-2023. Available from: <https://cvphysiology.com/cardiacfunction/cf024>. Accessed 12 October 2023.
4. Klabunde R. Cardiovascular physiology concepts. 3rd Edition. Philadelphia: Wolters Kluwer; 2021.
5. Kaplan JA. Kaplan's Cardiac Anesthesia. 8th Edition. Philadelphia: Elsevier; 2023.
6. Klabunde R. Ventricular diastolic dysfunction. [Internet]. Cardiovascular physiology Concepts; c1998-2023. Available from: <https://cvphysiology.com/heart-failure/hf006>. Accessed 12 October 2023.
7. Dekker AL, Barenbrug PJ, van der Veen FH, et al. Pressure-volume loops in patients with aortic stenosis. *J Heart Valve Dis.* 2003;12(3):325-32.
8. Bloria SD. Lessons from the frank-starling curve. *Ann Card Anaesth.* 2021;24(1):118-9. https://doi.org/10.4103/aca.ACA_29_20.
9. Medert R, Bacmeister L, Segin S, Freichel M, Camacho Londoño JE. Cardiac response to β -adrenergic stimulation determined by pressure-volume loop analysis. *J Vis Exp.* 2021;(171). <https://doi.org/10.3791/62057>.
10. Han JC, Taberner AJ, Loiseau DS, Tran K. Cardiac efficiency and Starling's law of the heart. *J Physiol.* 2022;600(19):4265-85. <https://doi.org/10.1113/JP283632>.
11. Leite-Moreira AF. Current perspectives in diastolic dysfunction and diastolic heart failure. *Heart.* 2006;92(5):712-8. <https://doi.org/10.1136/hrt.2005.062950>.
12. Leite S, Rodrigues S, Tavares-Silva M, et al. Afterload-induced diastolic dysfunction contributes to high filling pressures in experimental heart failure with preserved ejection fraction. *Am J Physiol Heart Circ Physiol.* 2015;309(10):H1648-54. <https://doi.org/10.1152/ajpheart.00397.2015>.