# DETERMINANTS AND CONTROL OF CARDIAC OUTPUT

Assessing myocardial performance The overall function of the cardiovascular system is to deliver oxygen and metabolic substrates to the to the tissues and to remove the products of metabolism. It performs these functions by pumping blood, therefore most assessments of myocardial performance are based on how well the heart can pump blood. Measuring pump effectiveness is usually by the volume of blood pumped per unit time. This is done by measuring the cardiac output (CO) measured in litres per minute. To enable comparison it is sometimes divided by body surface area, this is the cardiac index (CI).

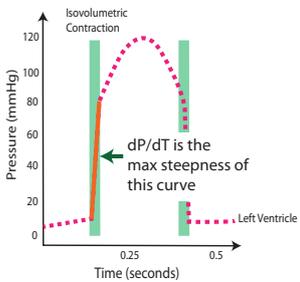**Cardiac Output** is the product of Stroke Volume (SV) and Heart Rate (HR).
Stroke volume is determined by three factors: preload, afterload and contractility.

Preload is the initial fibre length. Preload is the load on myocardial fibres just prior to contraction. It can be used to refine our understanding of myocardial performance beyond just CO or CI. If a heart can pump 5 litres from a low preload then it is usually considered to be performing better than a heart requiring more preload. As stated above preload is the inital fibre length of a sarcomere, this however is not possible to measure in an intact heart. Therefore volume and pressure are used as surrogate markers of preload. The most commonly used is the volume of blood in the left ventricle at the end of diastole (LVEDV) which is represented on the pressure volume loop at the bottom right. Volumes are often difficult to estimate minute to minute without monitoring such as an ECHO. Pressure is also used but it is problematic because of compliance as a confounder. Left atrial pressure will equal ventricular pressure at the end of diastole (LVEDP) (when sarcomeres are stretched prior to contraction). Left atrial pressure can be estimated using a swan ganz catheter and measuring pulmonary artery occulsion pressure (PAOP) on the right side of the circulation because it is a low resistance circuit, the artery is occluded and they are at the same horizontal level. If there is mitral stenosis however then this will be inaccurate. Finally the CVP and right atrial pressure may be used as indices of the right ventricle preload. This may sometimes also correlate to left sided filing pressures although this is not always the case.

RESTING

CONTRACTION

PRELOAD   PRELOAD
                  AFTERLOAD
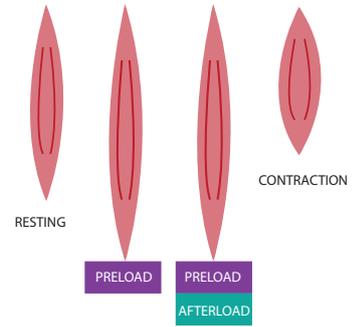
1. RESTING is at the end of isovolaemic relaxation before any filling. 2.PRELOAD is at the end of ventricular filling note the muscle is now stretched. 3. AFTERLOAD is the extra tension which does not stretch the heart but must be overcome to inorder to eject. 4. CONTRACTION is at the conclusion of systole. Note that contractility can be measured in two muscles if preload, afterload and rate are kept the same.

Afterload is the tension which needs to be generated in cardiac muscle before shortening will occur. Again this is unable to be measured in the intact heart and the nearest estimation is instantaeous wall tension. There are several surrogate markers. In its simplest terms afterload is thought of as the impedence to flow from the ventricle during systole. As such mean arterial pressure may be used as an estimate. More accurate still it to consider the relationship between mean pressure and mean flow represented by systemic vascular resistance (or PVR on the right). Again however this is inaccurate because the flow generated by the heart is not continuous but intermittant and pulsitile. Using a pressure-volume loop afterload is represented by the end systolic pressure connected to the LVEDV point.

Isovolumetric Contraction

dP/dT is the max steepness of this curve

Left Ventricle

Pressure (mmHg) — 120, 100, 80, 60, 40, 20, 0
Time (seconds) — 0.25, 0.5

Myocardial Contractility is defined as the intrinsic ability of the myocardial fibre to shorten independent of preload and afterload. Brandis defines this as the factor that is responsible for changes in myocardial performance that is not due to changes in heart rate, preload and afterload. The intracellular mechanism that is responsible for all factors which increase contractility is increased intracellular calcium. Measurement of contractility is difficult. dp/dt_{max} refers the the maximum rate of change in pressure in the left ventricle during isovolumetric contraction. A more forceful contraction would be associated with a greater rise in pressure and for this reason this is often used as a marker of contractility.

Frank-Starling mechanism states that the strength of cardiac contraction is dependent on the initial fibre length. Increased fibre length alters cardiac performance mainly by changing the Ca^{2+} sensitivity of the myofilaments and, in part by changing the number of myofilament crossbridges that can interact. Beyond an optimal fibre length, contraction is actually impaired. From a practical perspective this is important because increases in end-diastolic volume cause an increase in ventricular fibre length, which produces an increase in developed tension. This is very important because it matches cardiac output to venous return this ensures a precise matching of the outputs of the right and left ventricles. Positive iontropy increases Ca^{2+} sensitivity and therefore results in an increase in CO for any level of right atrial pressure or LVEDV. Negative iontropy does the opposite. The Frank-Starling mechanism is best represented by a family of so called ventricular function curves which map the CO (or ignore the HR and just use the SV) on the ordinate (y-axis) and the end diastolic atrial pressure or LVEDV. From left shift implies improved inotropy, right shift negative inotropy.

Positive Iontropic Effect
Control
Negative Inotropic Effect
Stroke Volume or Cardiac Output
Right Atrial Pressure or LVEDV
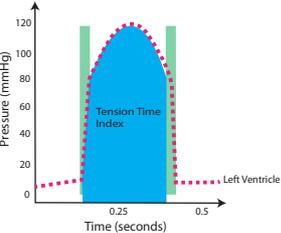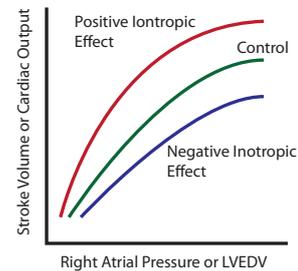
Myocardial oxygen consumption The heart is an aerobic organ and cannot tolerate (except for brief periods) oxygen debt. At rest MVO_2 is about 7-9 mls/100g/min (which equates to about 21 to 27mls O_2 /min for a 300g heart). An understanding of myocardial oxygen consumption (MVO_2) has both physiological and clinical relevance (especially in the setting of ischaemia). Clinical studies have shown that the three major determinants of MVO_2 are Myocardial Wall Tension, Contractility and Heart Rate. Minor determinants include electrical depolarisation, direct metabolic effect of catecholamines, basal metabolic activity and external work. This explains why patients with mitral regurg rarely develop ischaemia (decreased wall tension) why beta blockade is very effective in angina (decreased heart rate) and why angina is rare in dilated cardiomyopathy (decreased contractility). Further studies have shown a close relationship between the area under the systolic portion of the LV Pressure-Time curve with MVO_2 and this is known as the 'tension time index'.

Tension Time Index

Left Ventricle

Pressure (mmHg) — 120, 100, 80, 60, 40, 20, 0
Time (seconds) — 0.25, 0.5
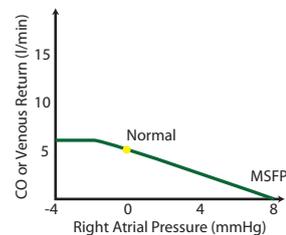
Vascular Function Curves If the entire circulation was brought to a standstill (eg asystole) then there would be a residual pressure in the vascular system due to a degree of overfilling. This is the mean systemic pressure (which is measured in the right atria) when cardiac output is zero and is said to be 8 mmHg this is also known as the mean systemic filling pressure MSFP. The mean systemic pressure may be altered by either increased volume or resistance. Peter Kam identifies the normal point to by RAP of 0mmHg and CO of 5l/min. The vascular function curve intersects the y-axis at the point where decreasing the RAP no longer increases vascular return due to starling resistor forces in the extracardiac vessels. Increasing resistance alters the steepness of the curve but not the MSFP. Increasing the load shifts the curve but not the steepness.

CO or Venous Return (l/min) — 15, 10, 5
Normal
MSFP
Right Atrial Pressure (mmHg) — -4, 0, 4, 8

Cardiac and Vascular Function curves integrated it is possible to overlay the normal Frank-Starling curve shown above and the vascular function curve using RAP as a surrogate for preload. This is important because it gives the equilibrium point which matches venous return to cardiac output. It will also allow you top predict the changes that occur with increased contractility, increased fluid volume and changes in peripheral resistance.

Cardiac Function
Equilibrium
Vascular Function
CO or Venous Return (l/min) — 15, 10, 5
Right Atrial Pressure (mmHg) — -4, 0, 4, 8

Cardiac Function
Hypervolaemia
Hypovolaemia
CO or Venous Return (l/min) — 15, 10, 5
Right Atrial Pressure (mmHg) — -4, 0, 4, 8

Cardiac Function
Increased Resistance
Decreased Resistance
CO or Venous Return (l/min) — 15, 10, 5
Right Atrial Pressure (mmHg) — -4, 0, 4, 8

Increased Inotropy
Cardiac Function
Decreased Inotropy
Vascular Function
CO or Venous Return (l/min) — 15, 10, 5
Right Atrial Pressure (mmHg) — -4, 0, 4, 8

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