2. Heart as a Pump

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Cardiac Cycle

- With av HR of 72/min:
  - Total cycle = 0.8 seconds
  - Systole = 0.3 s
  - Diastole = 0.5s
    - vent filling ~ 2/3 cycle

Mid Diastole
- Atrial & vent pressure both low
- Rate filling ventricles as V pressure rises above A pressure due to wall stretch
- Vents now 80% full
- Cusps of mitral/tricuspid valves drift towards closed
- 80% vent filling occurs passively

Atrial Systole (late Diastole)
- SA node fires ⇒ P wave on ECG ⇒ atrial contraction (atrial a wave)
- Contraction of atria narrows IVC & SVC orifices to ↓backflow
  - is some regurg
- See small rise in vent pressure
- Atrial kick contributes ~20% vent EDV
  - (vent EDV in supine ~160ml; stand ~130ml)
- Impt in fast AF with loss of kick

Vent Systole
- @ start AV valves close ⇒ isovolumetric contraction:
  - 1st heart sound
  - Sharp rise in intravent pressure
  - Lasts 0.05s
  - Mitral/tricuspid valves bulge into atrium
    - C wave of atrial pressure wave (↑pressure LA:10mmHg. RA:5mmHg)
- Once vent pressure higher than aorta & pulmon ⇒ ejection
- Rapid vent ejection phase after valve open followed by prolonged reduced phase
- Pressure changes:
  - Aorta: 80⇒120
  - Pulmonary a: 5⇒25
- Late systole: pressure aorta > L vent but momentum keeps blood flowing
- Elasticity of aortic walls & periph resistance to flow maintain aortic pressure
- SV ~ 70-90ml
- End diastolic vent volume ~ 120ml
- End systolic vent volume ~50ml
  - Ejection fraction ~65% in norm heart
- Atria:
  - Rapid ejection phase: mitral/tricuspid valves pulled down by vent systole ⇒ ↓atrial pressure⇒
    - Aiding filling
    - = x descent
  - Rest vent systole: rise atrial pressure as blood fills atria = v wave
    - (↑in tricuspid regurg c & v merged into one large v wave)

Early diastole
- Protodiastole =
  - Before aortic/pulmon valve closed
  - Rapid drop vent pressure
  - Lasts 0.04s
  - Ends with valve closure
- Isovolumetric vent relaxation:
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- Begins with closure of aorta & pulmon valves (2nd heart sound – may be split if aortic closes 1st)
- Incursura in aortic pressure waveform produced by closure of valve causing brief backflow of blood
- Atrial pressures: LA ~5mmHg; RA ~2mmHg
- Ends when vent pressure falls below atrial pressure ⇒ mitral/tricuspid valve opening

- Atrial pressure ↑ after systole until mitral/tricuspid valves open
- Rapid filling of ventricle occurs after mitral/tricuspid opening – most imp part of vent filling as time to fill is shortened with tachycardia
- Y descent of atrial pressure as it empties

**CVS Pressures**

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**Heart Failure**
- Systolic failure =
  - weakened systolic contraction
  - ↓ ejection fraction
  - responses:
    - activation of genes ⇒ myocardial hypertrophy
    - ↑ sympathetic n.s.
    - ↑ renin & aldosterone secretion ⇒ Na & water retention
      - initially compensatory but then failure worsens with ventricular dilation
- diastolic failure:
  - ↓ elasticity of myocardium ⇒ ↓ filling of vent in diastole ⇒ ↓ SV ⇒ same responses in systolic failure
- high output failure:
  - relative low CO (not absolute)
  - seen in:
    - large AV fistula
    - thyrotoxicosis
    - thiamine deficiency

- Rx:
  - ACEI - ↓ VC & aldosterone ⇒ volume ⇒ ↓ bp ↓ afterload
  - Nitrates – venous VD ⇒ ↓ preload
  - Diuretics ↓ fluid overload ⇒ ↓ preload & afterload
  - B Blockers - ↓ chance of arrhythmia
  - Digoxin - ↑ Ca [in] ⇒ ↑ force of contraction

**Pericardium**
- Myocardium – epicardium – pericardium
• Between epicardium & pericardium = 5-30ml fluid

**Timing**

• R atrial systole then L atrial systole
• Left ventricular contraction then R vent
• R vent ejection just before L vent
  ➔ as pressure in pulmon circuit < aortic pressures
• End of systole (S2 heart sound)
  o During inspiration – pulmon valve closure delayed
  o During expiration – aortic & pulmon valves together

**Length of Systole & Diastole**

• Duration of systole is more fixed than diastole
  ➔ although speed of systole does decr with ↑HR
• Marked decr in diastole with high HRs – results in:
  o ↓ vent filling
    ➔ up to 180/min filling adequate if enough venous return
  o ↓ perfusion of coronary circulation
• cardiac mm cannot tetanise like skeletal mm
• max theoretical rate of vents =400
• only see rate >230 in vents in VT
  ➔ max AV node d/c rate

**Arterial Pulse**

• pulse felt is a pressure wave NOT blood moving forward
• pressure wave moves faster than blood flow:
  o aorta 4m/s
  o large arteries 8m/s
  o small arteries 16m/s
  ➔ older rigid arteries wave moves faster
• pulse pressure ∝ strength of felt pulse eg
  ➔ no relation to mean pressure
  o shock – narrow pulse pressure 2nd to ↑ diastolic pressure ⇒ thready pulse
  o aortic insufficiency – high pulse pressure 2nd to regurg & ↓ diastolic ⇒ collapsing pulse
• dicrotic notch =
  o notch on falling phase of pulse
  o unable to feel; only measure
  o vibrations from aortic/pulmon valve shutting

**JVP**

• atrial pressure:
  o ↑ in atrial systole
  o ↑ in isovolumetric contraction – bulge of AV valve into atria
  o rapid↓ in early systole – AV valves pulled into ventricle
  o slow ↑ through systole – atrial filling
  o ↓ as AV valves open in diastole
• JVP waves:
  o A – atrial systole
  o C – isovolumetric contraction - bulging of AV-valves into atrial chambers
  o V – rise in atrial pressure until tricuspid valve opens (due to atrial filling during ventricular systole (*lasts until end of isovolumetric relaxation*))

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Heart as a pump

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- **x-descent**: *(follows c wave)* initial pressure drop in atria during initial rapid ventricular ejection = due to atrial relaxation as well as ballistic effect of contracting ventricles on atria.

- **y-descent**: *(follows v wave)* drop in atrial pressure as AV valves open at end of isovolumetric relaxation and onset of early diastole.

- Respiration effects volume of JVP waves:
  - Inspiration & \( \uparrow \text{-ve} \) intra-thoracic pressure \( \Rightarrow \downarrow \text{venous pressure} \)
  -Expiration \( \Rightarrow \uparrow \text{venous pressure} \)

**Cardiac Graph**

= Wiggers diagram:

**Heart Sounds**

- **S1** = closure of AV valves = start of systole
- **S2** =
  - closure of aortic & pulmon valves = end of systole

Phases:
1. atrial systole
2. isovolumetric contraction
3. vent ejection
4. isovolumetric relaxation
5. vent filling
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Heart as a pump

- inspiration ⇒ physiological splitting of S2 by late closure of pulmon valve due to incr preload
- S3 (always follows S2) =
  - rapid vent filling 1/3 through diastole
  - can be normal
- S4 =
  - Filling of stiff ventricle following atrial contraction
  - Just before S1
  - Always pathological

Murmurs
- Blood flow speeds up through narrowing eg:
  - stenosis
  - regurgitation
- turbulent flow ⇒ murmur
- aortic or pulmon valve:
  - stenosis ⇒ systolic
  - regurg ⇒ diastolic
- AV Valve:
  - Stenosis ⇒ diastolic
  - Regurg ⇒ systolic

Cardiac Output

Measurement
- Fick principle = amount of substance taken up by organ/time = arterial level of the substance minus the venous level (A-V difference) x blood flow
- Norm CO = ~70ml/kg/min or 5 litre/min
- Cardiac index =
  - CO/body surface area
  - output/min/m²
  - norm ~ 3.2
  - CI adjusts for differing body sizes

Factors effecting CO
- CO = HR x SV
- As circ is a close system CO usually = VR
- SV determined by:
  - Loading factors –
    - preload (PL)
    - afterload (AL)
  - Myocardial contractility (Cx)
- 4 determinants of CO:
  - HR
  - Cx
  - AL
  - PL
  - All these factors also determine Myocardial O2 demand

Definitions
- Preload = amount of stretch of ventricular mm fibres at end of ventricular filling (aka initial fibre length)
- Afterload = impedance to the ejection of blood into the arterial circulation
  - Impedance rather than resistance as it is a changing resistance
  - Afterload because ventricle only performs work after the aortic valve opens
• Contractility = factor responsible for changes in myocardial performance which are not due to HR, preload or afterload
  ↩ not to confuse with Starling's law
• All myocytes display 5 chars:
  o Bathmotropy = excitability
  o Dromotropy = conductivity
  o Chronotropy = rhythmicity
  o Inotropy = contractility
  o Lusitropy = relaxation

Preload
• Starling's Law of heart = diastolic length of vent mm fibres determine the force of their contraction
  ↩ ie intrinsic quality of cardiac myocytes NOT contractility
• ∴ Amount of vent mm fibre stretch at end of diastole forms basis of Starling Law

Summary preload factors:
  o filling pressure  ↩ CVP:
    ▪ blood volume
    ▪ gravity
    ▪ Tx pump
    ▪ mm pump
    ▪ CO
    ▪ periph tone
  o ventricular compliance

Right Side of Heart
• cannot measure stretch but EDV is next best indicator of preload
• EDV can be measured with ECHO
• Another indicator of preload = end-diastolic pressure (EDP) aka filling pressure
• EDV is proportional to EDP but EDV achieved will depend on ventricular compliance
  compliance = ΔV / ΔP
  ∴ EDV = ventricular compliance x filling P

Left Side of Heart
• LVEDP cannot be measured directly
• ∴ next best way to determine LV preload is surrogate pressure measurement = PCWP
• PCWP theory:
  o End diastole: continuous communicating column of blood from LV ⇒ LA ⇒ pulm veins ⇒ pulmon caps
  o Inflate pulmon art catheter balloon, float pressure sensor distally until wedged in pulmon capillary
  o Pressure measured is PCWP
  o Ideally catheter should be in West zone 3 (Pa>Pv>PA)
• Next surrogate of LV preload = CVP:
  o CVP ≈ RAP ≈ RVP ≈ PAP ≈ PCWP ≈ LAP ≈ LVP ≈ LV volume ≈ LV preload
• Downsides of R side pressure surrogates of LV preload are many!!:
  o Valvular disease
  o Pulmon disease
  o Changes in LV/RV compliance
Factors Effecting EDV

- EDV depends on:
  - Compliance – of ventricle
  - Transmural pressure distending it:
    - internal pressure – external pressure
    \[ \text{external p} = \text{intrathoracic pressure} \]
- compliance:
  - ↓ compliance ⇒ ↓ EDV for a given distending pressure
- external pressure:
  - ITP (intrathoracic pressure)
    - end expiration = -5 cmH20
    - end inspiration = -10 cmH20
  - inspiration creates a suction effect around heart & central veins ⇒ ↑ vent filling
- internal pressure:
  - EDP in RV ~ CVP
  - Therefore CVP plays key role in determining preload…

Factors Influencing CVP

- Blood volume:
  - 2/3 of total blood volume is in venous system
  - any ↑ in volume ⇒ sig ↑ in CVP
- gravity:
  - influence distribution of venous blood between periph & thoracic veins
  - eg ~500 ml blood pools in LLs in erect position
- Perih venous tone:
  - Esp skin, kidney, splachnic veins
  - Innervated by symp ns ⇒ venoconstriction ⇒ ↑ CVP
- MM pump: esp imp in exercise ⇒ ↑ VR ⇒ ↑ CVP
- Throacic pump:
  - With ↑ ing insp ⇒ ↑ negative ITP & abdo pressure ↑ ve ⇒ ↑ venous gradient from abdo to thorax
  - venous valves prevent back flow during expiration
- Cardiac output:
  - Heart transfers blood from venous to arterial system ⇒ ↑ MAP & ↓ CVP
  - ∴ Everything else being the same ↑ MAP & ↓ CVP acts as a brake

Functional Examples

- decreasing EDV:
  - ↑ intrapericardial pressure
    - eg pericardial effusion,
    - pressure from tumour
  - ↓ ventricular compliance:
    - MI
    - Inflam disease
- increasing EDV:
  - atrial contraction
  - hypervolaemia ⇒ ↑ venous return
  - ↑ pressure gradient to heart along venous flow:
    - inspiration
    - muscular activity
    - moving to lying
Relation of Tension to Length in Myocardium (Starlings Law)

- Nb force of contraction or initial fibre length difficult to measure .
  - Y axis usually = CO, SV, stroke index, stroke work
  - X axis usually = LVEDV, LVEDP, PCWP
- Curve also known as ventricular function curve ← in vitro measurement

- Starlings law of heart = energy of contraction is ∝ to initial length of cardiac mm fibre up to a point
- This is defined by myocardial sarcomere length (~2.2um) & ratchet mechanism:
  - Immediate rise in contractile force – due to overlapping actin filaments & myosin bridges
  - Delayed response (over mins) - ↑ systolic calcium flux – due to stretch sensitive Ca channels
- Starling mechanisms function:
  - Balance outputs of R & L ventricle:
    - 1% difference in RV > LV output over 30mins ⇒ ↑pulmon blood from ~600mls to ~2100mls ⇒ severe pulmon oedema
    ← RV output > LV ⇒ ↑LVEDV ⇒ starling mechanism ⇒ ↑LV output
  - Valsalva Manoeuvre – causes fall in SV
  - In upright exercise: if CVP rises contributes to ↑SV
  - Mediates postural hypotension
  - Mediates arterial hypotension following haemorrhage

Guyton Cardiac Function Curve
- = in vivo curve
- Displays pivotal role of CVP in regards to CO
- Shows how CVP raised the CO by means of Starling mechanism provided HR & MAP are unchanged
- Differs from Starling vent function curve in that is affected by changes afterload & contractility ie in vivo
  ← a ventricular function curve shows only Frank Starling relationship ie isolated mm strip (in vitro)

- Homometric regulation = changes of contractility not due to change in fibre length
  ← ie autonomic system, drugs, ischaemia
- heterometric regulation = changes in CO due to mm fibre length changes

Afterload
- afterload = impedance to vent ejection
- function of:
  - SVR
  - Laplace law: ventricular wall tension
    - T = P.R/wall thickness
      ← : ↑intrathoracic pressure seen with IPPV ⇒ ↓afterload via ↓R & ↑wall thickness
  - aortic root compliance
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Heart as a pump

- aortic valve dysfunction
- for LV = MAP during systole; for RV = mean pulmonary arterial pressure (during systole)
- MAP = CO x SVR (chief site of resistance = arterioles)

NB:
- isotonic cardiac contraction = against a fixed afterload (in vitro only)
- auxotonic = contraction against a changing afterload (happens in vivo)

Effect of Afterload
- afterload is reflected by ↑myocardial wall tension (T)
- ventricular hypertrophy ⇒ ↓wall tension by ↑ing wall thickness
  ← explained by La Place’s Law:

for thin walled spheres or tubes:

\[ P = 2T/r \]

can rearrange: \[ T = P \times r / 2 \]

For thick walled sphere or tube:

\[ \text{Tension} = \text{wall stress (S)} \times \text{wall thickness (w)} \]

∴ wall stress = \[ \frac{P \times r}{2w} \]

- thus wall stress ↑ed by:
  - ↑pressure in chamber (afterload)
  - ↑radius (dilated heart)
- thus wall stress ↓ed by:
  - ↑wall thickness (LVH)
- it is the ↑ed wall stress which ↑s myocardial O2 demand

Myocardial Contractility

- Starling mechanism = intrinsic regulation of contractile strength
- ∴ myocardial contractility = extrinsic or change in contractile energy not due to fibre length changes
- inotropic state = contractility

Positive Inotropic Factors
- Positive inotropic effect on heart ⇒ shifts length-tension curve UP & LEFT eg:
  - Symp n.s. =
    - ↑norad release at symp nerve terminals
    - circulating catecholamines ie norad, adrenaline, dopamine
      - act on \( \beta_1 \) receptors ⇒ ↑cAMP
  - Drugs:
    - Xanthines eg caffeine & theophylline –
      - Phosphodiesterase inhibitor ⇒ ↓breakdown of cAMP
    - Digoxin – inhibitory effect on NaK ATPase ∴ indirect inhibition of NCX pump
    - Angiotensin 2
    - ↑extracellular Ca
    - thyroxine
  - Force frequency relation:
Vent extrasystole condition next beat to be stronger
Due to ↑Ca [in]
- ↑HR –
  - small incr in contractility
  - not very much in vivo
  - if HR ↑ed the 1st beat is weaker than following beats with progressive ↑in strength
  - = Bowditch rate effect
due to ↑size of calcium influx

**Noradrenaline effects**
- =most impot ionotrope
- NA released from symp nerve terminals in vent wall:
  - binds to B₁ receptors on myocytes (Gₛ linked) ⇒
  - activates a prot – cAMP – prot kinase A – calcium channel phosphorylation sequence ⇒
  - ↑inward calcium current during the plateau of the AP ⇒ ↑intracellular calcium stores ⇒ more forceful systolic contraction
- In addition:
  - sarcoplasmic reticulum calcium uptake pumps are accelerated ⇒ shorter systole
  - This is beneficial as it preserves diastolic filling time (impt in tachycardia)
    - This property (ie relative shortening of systole) is called *lusitropy*.
- Thus the effect of NA is a more *forceful* and shorter systole, which results in following:
  - ↑ed bp: Ventricular pressure ↑s more rapidly in the isovolumetric phase
  - ↑ed Ejection fraction: - velocity of contraction and shortening are enhanced by NA.
    - EF is sometimes used as an indirect index of contractility
  - Stroke volume:
    - Transient ↑ as EF rises, BUT
      - then limited by the concomitant ↓ in EDV as well as ↑MAP.
  - ↓ed Systolic duration: (lusitropy) – shorter ejection time does not significantly curtail SV, because the velocity of shortening is increased. (ie more forceful contraction).

**** **** ****
- Negative ionotropic ⇒ DOWN and to RIGHT
  - Para symp = vagal tone via Ach
  - ↑CO₂, ↓O₂
  - ischaemia via acidosis & hypoxia
  - drugs:
    - BBs & CCBs
    - Barbituates
    - Many anaesthetic drugs
  - heart failure – intrinsic depression:
    - ? cause but thought:
      - ↓regulation of B receptors
      - ↓Ca liberation from Sarcoplasmic reticulum
Heart rate

- CO = SV x HR
- Any change in HR usually occurs as part of integrated response to a ↑↓MAP
- HR under autonomic control:
  - Symp ⇒ ↑HR
  - Parasymp ⇒ ↓HR
- Heart = demand pump ∴ any change in HR will only effect CO if assoc with change in demand from tissues
- Tissues set their demand by means of total venous return ⇒ ↑↓tendency for VR ⇒ Starling mechanism ⇒ adjustment of CO
  - ∴ VR = CO

Isolated Changes in HR

- Eg pacing, atropine
  - Will see surprisingly little change in CO (within limits)
    - cos see an inverse change in SV for any change in HR
- Isolated ↑HR:
  - artificial pacing rate ⇒ ↓diastole but not systole ⇒ ↓ed vent filling time
  - transfer of blood from venous side to arterial side ⇒ ↓EDP & ↑MAP both of which ↓SV
    - at a threshold actually start seeing ↓CO due to ↓↓ed vent filling time
- isolated ↓HR:
  - initially CO will remain constant:
    - ↑HR offset by ↑vent filling time ⇒ ↑SV
  - at threshold will see a ↓CO as ventricles max filled ∴ ↓ing HR ⇒ ↓ing CO
O2 Consumption

- O2 consumption determined by:
  - intramyocardial wall stress (surrogate for afterload)
  - contractile state of heart
  - HR

- Ventricular work = SV x mean arterial pressure
  - thus LV work x7 more than RV

- Pressure work requires ↑↑o2 consumption compared to volume work ie afterload > preload
  - incompletely understood
  - why angina is more common AS : AR

- basal o2 consumption 2ml/100g/min
  - much bigger than skeletal mm
- beating heart 9ml/100g/min
- ↑ in o2 consumption require ↑ in blood flow

Summary Effects on CO

- ∴ 4 determinants of CO:
  - HR
  - Cx
  - AL
  - PL
  - All these factors also determine Myocardial O2 demand
- But any change in single factor remarkable ineffective
- Need coordinated cooperative change in all factor to change CO substantially

Functional Factors Effecting CO Overall

- No change:
  - Sleep
  - Mod changes in environmental temperature
- Increase:
  - Anxiety ↑50-100%
  - Eating ↑30%
  - Exercise ↑ up to 700%
  - ↑temp
  - pregnancy
  - Adrenaline
• Decrease:
  • Lie to sit/stand ↓20-30%
  • Arrhythmias
  • Heart disease
**Ventricular Function Curves**

**Ventricular Pressure Volume Loops**

Normal P-V loop for LV:

- **All the information obtained from Vent P-V loop (favourite exam topic):**
  - EF
  - LVEDV – Note: this is number on x axis – NOT point B
  - Indices for afterload = Ea line
  - Indices for ventricular compliance (EDVP Line)
  - Contractility = Ees line
  - Stroke work (PxV = Joules) = area within loop
  - B-A = diastolic filling
  - C-B=isolvolumetric contraction
  - D-C = volume ejected
  - D-A= isovolumetric relaxation

**EDPV Line**

- P-V relationship during diastole (A⇒B) gives an idea of ventricular compliance
  - most important part of line is End Diastolic P-V line (EDPV)
- ΔP is plotted against ΔV ∴ talk about elastance (ie inverse of compliance)

- EDVP line as shown

**ESPV Line**

- Similar line to EDVP but drawn on top of ventricular function curve at End systole
- Aka endsystolic elastance of EEs line ie measure of contractility
Effect of Isolated ↑Preload on LV PV Loop

- Loop 1 = normal; loop 2 shows ↑ed preload
- Loop 2 has ↑ed stroke volume as wider
- Contractility & afterload are constant

Effect of Isolated ↑Afterload

- Index for afterload =
  - slope of line connecting LVEDV & End systolic point
  - aka effective arterial elastance (Ea line)
- In loop above this line parallel; in this loop clear ↑in afterload in loop 2
- Loop 2 has ↓ed SV 2nd to ↑ed afterload which manifests as earlier aortic valve closure

Note the decreased stroke volume for loop 2 (which has the increased afterload). The aortic valve is closing at a higher pressure so less volume is ejected during systole.
**Effect of ↑ed Contractility**
- LVEDV is same for both loops ie preload same
- Ea line is the same ie afterload same
- ESPV Line = index of contractility
- Loop 2 demonstrates ↑SV by mechanism of isolated ↑contractility \(\therefore\) ESPV ↑ed gradient

![Effect of Increased Contractility on LV Pressure-Volume Loop](image)

Note the increased stroke volume for loop 2 (which has the increased contractility).
* The increased slope of the end-systolic pressure-volume line is an index of the increased contractility.
* The end-systolic points of both loops lie on the same 'afterload line' so thereafterload is the same for the 2 loops.
* The LVEDV is the same for the 2 loops so the pre-load is the same.

**NB:** Above lines demonstrate differences in vitro. In body more integrated changes seen

**Effect of Systolic & Diastolic Dysfunction**
- L curve = systolic dysfunction:
  - ↓ed stroke volume
  - curve also implies afterload ↑ed by steeper Ea line (not drawn)
- R curve = diastolic dysfunction:
  - EDPV shifted up and left ie preload ↓ed due to ↑elastance \(\Rightarrow\) ↓SV

![Effect of systolic and diastolic dysfunction on the pressure-volume loop of the left ventricle](image)

*Figure 20-7. Effect of systolic and diastolic dysfunction on the pressure-volume loop of the left ventricle. Left: Systolic dysfunction shifts the isovolumic pressure-volume curve (see Fig 20-2) to the right, decreasing the stroke volume from b-c to b'c'. Right: Diastolic dysfunction increases end-diastolic volume and shifts the diastolic pressure-volume relationship upward and to the left. This reduces the stroke volume from a-b to a'c'. (Reproduced, with permission, from McPhee SJ et al. Pathophysiology of Disease. 2nd ed. Appleton & Lange, 1997.)*
Mechanical Energy & Work

- = a pressure volume area
- Area within the loop = stroke work (mechanical energy) done by heart during single contraction
- = external work of ventricle

• Isovolumetric contraction (upstroke):
  - no ejection & . no external work
  - energy expended to generate potential energy ⇒ converted to heat during diastole

• Potential energy:
  - Purple triangle (EDPV – ESPV – isovolumetric relaxation line)
  - = amount potential energy available during a contraction
    ∴ correlates well with heat generated during contraction
  - total mechanical work + heat generated = PVA of stroke work + potential energy
    ⇔ pressure volume area
  - PVA correlates well with O2 consumption (VO2) of myocardium during single contraction
  - Gross energy efficiency of heart ~15%:
    - Much of contractile energy goes into raising pressure rather than ejection
    - . ↓bp can improve SV in heart failure

• Metabolic energy source variable:
  - 2/3 FFAs
  - rest = glucose & lactate
  - heart is an omnivore

Vascular Function Curves

• Starling Curve (&Guyton Curve) = cardiac function curves which relate:
  - Change CVP – x axis (indep variable)
  - Change in CO – y axis (dependant variable)
    ∴ ie how changes in CVP effect CO
    ⇔ a compliance curve
  - Vascular function curve relates: (switch-a-roo)
    - Change in CO – x axis (indep variable)
    - Change in CVP – y axis (dependant variable)
      ∴ ie how changes in CO effect CVP
      ⇔ a elastance curve
• Impt points:
  o CO = 0 ie cardiac arrest:
    ▪ Pressure becomes an equilibrium throughout whole CVS ie not just venous
    ▪ This = Mean circulatory pressure (MCP) (or Pmc as in diagram)
    ▪ MCP ~ 7mmHg ie vascular system overfilled
  o Point A:
    ▪ @normal CO ~5l/min
    ▪ CVP = ~2mmHg
    ▪ = norm operating pressure of system
  o Break point of line below point A:
    ▪ ↑ing CO ⇒ ↓ing CVP up to a point
    ▪ here intravascular pressure falls < extravascular pressure ⇒ collapse of distensible large vessels
    ▪ CO now limited by VR & cannot ↑anymore
  o If a VFC is combined with a CFC then CFC axis orientation take precedence for convenience

Effect of Change in Blood Volume & Venous Tone
• Transfusion or venoconstriction ⇒ upward shift of curve ie ↑ed MCP
  ⇔ converse is true ie haemorrhage & venodilation ⇒ downward curve shift
• NB neither will affect position or shape of cardiac function curve!!

Effect of change in SVR (ie ↑afterload)
• MCP does not change – only ~3% of total blood is in the arterioles
• Vasodilation ⇒ larger CO (or VR) ⇒ anticlockwise rotation of VFC
• Vasoconstriction ⇒ ↓CO (or VR) ⇒ clockwise rotation of VFC
• NB change in SVR (VasoC or VasoD) will also effect cardiac function curve
Coupling Between Heart & Vasculature

- NB CO = VR ie both describe total flow around closed circuit
- Coupling can be demonstrated by plotting CFC & VFC on same graph (with CFC axis orientation) (Guyton)
- Intersection of 2 curves = equilibrium point at which CVS tends to operate

- Curve shows how disturbances in equilibrium are dealt with:
  - ↑CVP:
    - CFC moved to point A
    - ↑ed CVP would ⇒ ↑CO ie point A ⇒ point B during next systole
    - As a result of ↑CO NET transfer of blood from venous to arterial side of circ ⇒ ↓CVP (due to VFC)
    - With each beat this transfer would be small ie point B ⇒ point C (not B ⇒ C1)
    - Due to ↓CVP next CO of next beat will also be ↓ed (due to CFC) ie point C ⇒ D
    - Point D is still above equilibrium ∴ heart will pump blood from venous to art circ every beat at a rate faster than blood will flow across peripheral capillaries back to venous circulation ∴ CVP continue to ↓ until equilibrium reached

Effect of ↑ed Contractility on Cardiac-Vascular Coupling
- Isolated ↑ed contractility due to symp stim
in vitro demonstration cos symp stim would also have effect on vasculature in vivo!

- equilibrium values will change:
  - CO shift up to point B (due to ↑ed contractility ie no change in CVP initially)
  - NET transfer of blood to art side ⇒ ↓ing CVP (B to C)
  - CO will continue to fall until reach new equilibrium (C to D)
  - at intersection of same VFC but with new CFC

Effect of Changes in SVR

- ↑SVR (vasoC) effects both CFC & VFC downwards
  - @equilibrium for an ↑SVR ⇒ ↓CO (point A to B)

Effect of Transfusion on Coupling

- VFC shifted parallel up to right
  - CFC not changed
  - CO & CVP are both ↑ed (point A to B)
  - the ↑CVP is reflected on the CFC by Starlings Mechanism ie moves along curve but does not change its shape
Coordinated Effect of Max Symp Activity (in Vivo)

- CFC is shifted up & left - ↑contractility overrides the ↑afterload effects
- VFC shifted:
  - Up – venoconstriction predominant
  - Slight rotation down – vasoconstriction = less predominant

Effect of Heart Failure

Heart as a pump - 22
By Adam Hollingworth

• Acute pump failure:
  o Mod to severe failure: CFC shifted right & down
  o Blood volume remains constant immediately
  o \( \therefore \) equilibrium (point A) will slide down normo-volaemia line
    \( \iff \) equilibrium point of will correspond to ↓ed contractility (point B or C)

• Chronic congestive Heart failure:
  o ↑blood volume ⇒ VFC shift upwards
    \( \iff \) 2\(^{nd} \) to aldosterone fluid retention & ↓GFR
  o CFC remains shifted down and right from pump failure (↓contractility)
  o \( \therefore \) mod heart failure – can maintain norm CO (point D) but with higher CVP
  o BUT with severe failure CO↓ing & CVP remains high

• Disparate changed in vent contractility:
  o Eg ant MI effecting LV only ⇒ ↓LV contractility but norm RV contractility
  o Initially LAP norm but ↓LV output
  o RV continue with norm output ⇒
    ▪ ↓ing RAP ⇒ ↓ing RV output
    ▪ ↑ing LAP ⇒ ↑LV output
    \( \iff \) both via CFC effects
  o continue until new equilibrium reached & 2 vent will have same outputs BUT ↑ed L sided pressures ⇒ ↑pulmon venous P which can lead to pulmon oedema

Athletes, Heart Transplant & Sympathetic Control of CO

• During exercise = ↑symp activity ⇒ ↑HR & ↑SV
  o In untrained individuals: bigger ↑HR; less ↑SV
  o In trained athlete: less ↑HR; bigger ↑SV
  o In heart transplant: no direct symp innervation thus
    ▪ Circulating catecholamines – small effect
    ▪ ↑EDV ⇒ ↑vent contraction ⇒ ↑SV
      \( \iff \) mm pump ⇒ ↑venous return
      \( \iff \) ↑Respiration ⇒ ↑venous pressure gradient
    ▪ ↑VD of arterioles in mm ⇒ ↓afterload ⇒ ↑CO