5.CVS Response to Function

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CVS Response to Changes in Posture

Supine ⇒ Erect

CVS Challenges

• ↓ in MAP: = due to ↓ CO due to ↓ VR
  └ venous pooling of blood in the lower extremities
  effect occurs within seconds, but not immediately.

• Hydrostatic effects on CPP:
  o brain is ~ 30 cm higher than level of the heart in the erect position (as opposed to the supine)
  o ↓MAP at level of brain of ~ 22 mmHg
  o effect = immediate.
  "NB: ↓MAP at brain level is offset by a similar:
    o ↑ CVP venous side (brain circulation is like an inverted U-tube) as well as on the
    o ↓ CSF pressure.
  • CPP is further augmented by an increase in VR from the brain to the heart in the erect position

• Summary: the main challenge to the CVS (and the brain circulation) is ↓MAP caused mainly by ↓VR
  ⇒ ↓CO.

The CVS response

• baroreceptor reflex mechanism:
  o ↓ MAP ⇒ sensed by carotid (mainly) and aortic baroreceptors ⇒ ↓ traffic up to NTS ⇒ via
    medullary control centre ⇒ ↑ SNS outflow and ↓ PNS outflow.
  o The ↑ SNS outflow causes: [ remember: MAP (minus RAP) = CO x SVR ]
    ▪ [↑preload] peripheral venoC ⇒ ↑ VR ⇒ ↑ CO ⇒ ↑ MAP
    ▪ [↑afterload] peripheral vasoC ⇒ ↑ SVR ⇒ ↑ MAP (slight ↓ in SV due to afterload increase, but net
      effect = ↑ MAP)
    ▪ ↑ cardiac contractility ⇒ ↑ CO ⇒ ↑ MAP
    ▪ ↑ Heart rate ⇒ ↑ CO ⇒ ↑ MAP
  o

 NB: Baroreflex ⇒ vasoconstriction = more effective than venoconstriction to restore MAP
  """"not to be confused with the vascular function curves where vasoconstriction shifts the curve more up than what
  vasoconstriction rotates it downwards"

• Cerebral pressure autoregulation: a.k.a. the myogenic mechanism:
  o effective at maintaining a constant cerebral blood flow within a MAP range of 50 – 150 mmHg.
  o It effects this by changing the CVR.
  o Onset is not immediate though.

\[
\frac{\text{MAP} - (\text{CVP or ICP})}{\text{CVR}} \quad \leftarrow \quad \text{arterial baroreflex}
\]

\[
\text{CBF} \quad \leftarrow \quad \text{pressure autoregulation}
\]

• Activity: Mm pump further augments VR
  "in conjunction with the one-way valves in the veins to prevents further venous pooling

Overview of CVS Response

• Baroreceptor & cerebral autopressure reg effective in normal people to prevent fainting when standing
  from supine.
• If the arterial baroreflex is blunted, ⇒ syncope
  "eg elderly and diabetic autonomic neuropathy"
• The standardized valsalva test can be used to check the integrity of the baroreflex
**Shock**

- Shock = inability of circulation to ensure adequate O2 delivery to the body tissues
- Types:
  - **hypovolaemic**: - haemorrhagic (loss of all blood components)
    - loss of plasma (burns)
    - loss of fluids + electrolytes (D+V’s, ↑ sweating etc)
    - Internal (3rd spacing; eg ascitis, ileus, pancreatitis)
  - **distributive**: - septic
    - anaphylactic
    - neurogenic (including sympathectomy of a SAB)
    - vasodilator drugs,
    - acute adrenal insufficiency
  - **cardiogenic**: - pump failure (AMI)
    - dysrhythmia (tachy or brady)
    - acute valvular dysfunction / rupture of ventricular wall or IV septum
  - **Obstructive**: - tension pneumothorax
    - massive pulmonary embolus
    - pericardial disease (tamponade, constriction)

- DO2 = CO x CaO2
- ∴ whenever discuss shock must consider all factors influencing CO:
  - preload
  - afterload
  - conreactility
  - HR

**Hypovolaemic Shock**

- Very common
- Causes both:
  - ↓CO via volume loss ⇒ ↓preload ⇒ ↓CO ⇒ ↓MAP
  - ↓CaO2

**Resp Response**: Severe ↓MAP ⇒ hypoxia/hypercarbia/acidosis ⇒ periph chemoreceptor stimulation ⇒
- ↑SNS &
- hyperventilation
  - in attempt to defend CaO2

**CVS response**

- can be classified by **time**:
  - Immediate:
    - **Sensors**:
      - Arterial baroreflex – [biggest response]
        - aim to restore CO & MAP to normal
        - ↓MAP sensed in carotid & aortic baroreceptors
      - ↓volume sensed by low pressure sensors of atria & large veins
      - hypoxia/hypercarbia/acidosis sensed by periph chemoreceptors
    - **Effect- predominantly of ↑SNS & ↓PNS via baroreceptors**:
      - venoC: ↑VR ⇒ ↑CO ⇒ ↑MAP
      - vasoC: ↑SVR ⇒ ↑MAP
        - widespread sparing only brain & heart
      - ↑HR: ↑CO & ↑MAP
        - in severe shock also see initial tachycardia ⇒ transient brady ⇒ back to tachy
        - ↑unmasking of vagal tone to help clotting
• ↑contractility: ↑CO ⇒ ↑MAP
  o Intermediate:
    ▪ Autotransfusion: Interstitial fluid move to intravascular (reversal of Starling forces)
      \[ \text{Up to 1000 ml fluid /hr can be moved intravascular via this mechanism.} \]
    ▪ Mobilization of reserve volumes: splanchnic/liver mainly
    ▪ Decreased renal blood flow – via ↓MAP
      o (normally ~25% CO)
      o initial +ve effects:
        o efferent vessels constricted > afferent
        o ↓ renal plasma flow ⇒ ↓GFR ⇒ filtration fraction ↑ed
        o ↑ed Na retention
          \[ \text{⇒ ↓UO which serves to preserve circulating volume} \]
      o late –ve effects:
        o azotemia ie nitrogen waste products retained ⇒ ↑Urea & creat
        o ARF
    ▪ Further redistribution of CO: ↓ muscle flow, ↓ skin flow
    ▪ ↑ Muscle pump activity of legs ( restlessness) ⇒ ↑VR
    ▪ ↑ ADH release ( from volume receptor input) ⇒ water retention
    ▪ ↑ Thirst + other behavioural responses
    ▪ ↑ renin/angiotensin/aldosterone mechanism
    ▪ ↑ adrenaline from adrenal medulla
  o Delayed (post haemorrhage) – aim to restore components lost in blood
    ▪ 12 – 72 hrs :
      o plasma volume restored to normal
      o Albumin replaced rapidly from extravascular stores
    ▪ Days:
      o plasma proteins and enzymes: ↑ liver synthesis
    ▪ Days to weeks:
      o RBC’s: ↑EPO from kidneys ⇒
        o reticulocytes peak day 10 days (norm ~1% retics in blood)
        o mature RBC’s back to normal 4 – 8 weeks.
      o Other: PLT’s, WBC’s
    • Can also be classified by severity:
      o mod shock ⇒ ↓pulse pressure
        ▪ due to diastole caused by catecholamines ↑ing vascular tone
        ▪ ⇒ ↓discharge baroreceptors ⇒ ↑symp tone ⇒ ↑VC & ↑HR
      o severe shock ⇒
        ▪ ↓mean pressure
        ▪ tachy⇒brady⇒tachy
        ▪ widespread VC – spares only brain & heart vessels
        ▪ kidneys – initial positive changes but then –ve acute failure
    • Any inadequate perfusion to tissues ⇒
      o ↑anaerobic glycolysis ⇒ lactic acid accumulation
      o low/mod levels of lactic acid excellent fuel for heart/CVS system
        \[ \text{but tipping point ⇒ acidosis} \]
    • lactic acidosis ⇒
      o ↓myocardial contractility
      o ↓vascular response to catecholamines ie ↑ed VD
      o toxic to CNS ⇒ coma
Coordinated Response to Exercise

- muscular exercise requires 3 tasks from circulation:
  - ↑ pulmon flow – to enhance gas exchange
    - ↑ ed RV output
  - ↑ ed flow thru working mm
    - ↑ ed LV output
    - local vasoD
  - maintain stable bp
    - controlled vasoC in non active tissues

- other issues need addressing:
  - energy production & utilisation
  - temp reg
  - fluid shifts
  - acid base changes/compensation

- exercise can be
  - static – isometric
  - dynamic – isotonic

Cardiac Output

- CO ↑ by x5 ie 5 l/min to 25 l/min
- Heart = demand led pump:
  - ↑ ed demand set by exercising mm effecting ↑ VR
- ↑ VR caused by:
  - venoC (↑ VR)
  - vasoD (↓ SVR)
  - mm pump of limb muscles (need intact venous valves)
  - thoracic pump:
    - ↓ ITP & ↑ abdo pressure with ↑ ed inspiration
    - ↑ RR & ↑ depth of insp in exercise enhances effects
    - -ve effects of expiration prevented by venous valves
  - ↑ myocardial contractility
  - ↑ HR
  - diversion of blood from non active tissue (splachnic & renal circulations)
  - local metabolites in exercising mm ⇒ arteriolar dilation ⇒ ↓ SVR ⇒ ↑ CO ⇒ ↑ blood flow to exercising mm ⇒ ↑ VR

Timing of Changes

- start of exercise:
  - sudden ↑ CO then gradual ↑ to steady state
- sudden initial changes 2nd to:
  - cortical activity (motor area)
  - sensory nerve activity assoc with movement
  - mm/thoracic pump ⇒ ↑ VR
- slow changes to steady state 2nd to:
  - vasoD in mm
  - redistribution of CO
  - ↑ SNS
- @ end of exercise:
  - abrupt ↓ CO
  - exponential fall

CVS Changes

- HR changes:
  - ↑ linearly up to max ~200/min in young adult
o initially caused by ↓vagal output
o later by ↑ed SNS output

• stroke volume:
o ↑ in non-linear way
  o big ↑ in light/mod exercise; only small ↑ into severe exercise
  o reasons for ↑:
    ▪ ↑VR & ↓: ↑LVEDV
    ▪ ↑contractility ⇒ ↓LVESV

• blood pressure:
o SBP can rise to 190-225mmHg 2nd to ↑ed CO
o DBP may increase slightly or even fall 2nd to ↓SVR
  ▲ NET result ▲pulse pressure x2-3

• Baroreceptor reflex – reset to higher level in severe exercise

**Muscle Blood Flow**

• @rest:
o mm blood flow = 2-3ml/100g/min
  ▲ mediated by SNS constriction of arterioles
o ~20% of CO – despite skeletal mm being ~40% of lean body mass
o precapillary sphincters closed ⇒ diverts mm blood flow away from microcirculation to main channels

• @exercise see:
o relaxing of precapillary sphincters due to:
  ▪ ↓PO2
  ▪ ↑PCO2
  ▪ ↑H
  ▪ ↑temp
  ▪ ↑K
  ▪ ↑ADP in interstitial fluid
  ▲ result is ↑total blood flow to max 50ml/100g/min ie ↑x20 ~80-90% of CO
o ↑diffusion of O2 into mm cell & ↑total O2 uptake by up to x40:
  ▪ ↑delivery O2
  ▪ R shift of OHDC

• Static contraction: sig ↓mm flow ⇒ ↑pressure in mm
• Isotonic contraction good mm flow as flow occurs in relaxation

**Blood Flow to Other Organs**

• ↑coronary flow:
o must meet extra cardiac work
o mediated by:
  ▪ local metabolic autoreg
  ▪ circulating catecholamines stim B2
• ↓flow to GIT & kidney – SNS activity shifts flow to exercising mm
• ↑skin flow to help with heat loss (SNS mediated)
• cerebral flow:
o remains constant at all levels of ex ~50ml/100g/min
o but relatively much smaller % of ↑ed CO

**Summary CardioResp Control During Exercise**

• 1st ventilation ↑s keeping close proportion of:

  ↑VO2 + VCO2 ⇒ PaO2 + PaCO2 = normal

• near max intensity: V_A rises > VO2 ⇒ ↓ PaCO2
• 1st 5-10 seconds of exercise: ↑HR 10-15/min due to ↓ vagal tone, then steady ↑ing HR over 5-10min due to ↑ SNS output
  ↦ initial tachy under central command
• end of exercise: HR & V_A fall sharply initially then more gradual ↓
• during exercise:
  o baroreceptors reset to operate at higher bp ranges allowing ↑ ed HR, ↑ CO, ↑ MAP
    ↦ in moderate exercise this resetting compensates for ↓ SVR
    in more strenuous exercise need ↑ SNS to compensate
  o resp chemoreceptor reflexes also seem to reset:
    ▪ ↑ ed response to change in PaO2
    ▪ severe exercise: ↑ lactate (↓ pH) additional stimulus

**Cardiac & Vascular Function Curves**
• Exercise requires an ↑ CO & control of heart & vasculature
• If isolated symns stim to heart (cardiac symp nerve stim):
  o ↑ MAP ⇒ ↓ CVP both of which favour ↓ ed SV (ie opposite of desired effect)
• in exercise:
  o [↑ afterload] ↑ ed MAP minimised by Vasod of exercising mms
  o [preload] ↓ CVP minimised by:
    ▪ periph venoC
    ▪ mm & thoracic pumps encouraging VR
• in upright exercise SV can double due to:
  o [preload] ↑ EDV (from ↑ CVP)
  o [contractility] ↓ End systolic volume – from ↑ EF via ↑ ed contractility

**CO = VR**

**Ohms Law:**

\[
\frac{\text{MSP} - \text{RAP}}{\text{VVR}} = \frac{\text{MAP} - \text{RAP}}{\text{SVR}} = \frac{\text{MPAP} - \text{LAP}}{\text{PVR}}
\]

MSP = mean systemic pressure ~ 7mmHg

RAP ~ 2-3 mmHg

VVR = venous vascular resistance

⇒ ΔP ~ 5mmHg ie venous resistance is very low

c/f

LHCO = L heart CO

ΔP ~ 88mHg

c/f

RHCO = R heart CO

Mean Pulmonary artery P ~ 15mmHg

LAP 5mmHg

ΔP ~ 10mHg