By Adam Hollingworth

5.CVS Response to Function

Table of Contents

By Adam Hollingworth

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:
  - brain is ~ 30 cm higher than level of the heart *in the erect position* (as opposed to the supine)
  - ↓MAP at level of brain of ~ 22 mmHg
  - effect = *immediate*.

  ↳ NB **:** ↓MAP at brain level is offset by a similar:
    - ↑ CVP venous side (brain circulation is like an inverted U-tube) as well as on the
    - ↓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:
  - ↓ MAP ⇒ sensed by carotid (mainly) and aortic baroreceptors ⇒ ↓ traffic up to NTS ⇒ via medullary control centre ⇒ ↑ **SNS** outflow and ↓ PNS outflow.
  - 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**
  -

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

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

$$CBF = \frac{MAP - (CVP \text{ or } ICP)}{CVR}$$

← ← *arterial baroreflex*
← ← *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
    - Intermediate:
        - Autotransfusion: Interstitial fluid move to intravascular (reversal of Starling forces*)*
            ↳ *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
            - (normally ~25% CO)
            - initial +ve effects:
                - efferent vessels constricted > afferent
                - ↓ renal plasma flow ⇒ ↓GFR ⇒ filtration fraction ↑ed
                - ↑ed Na retention
                ↳ ↓UO which serves to preserve circulating volume
            - late –ve effects:
                - azotemia ie nitrogen waste products retained ⇒ ↑Urea & creat
                - 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
    - Delayed (post haemorrhage) – aim to restore components lost in blood
        - 12 – 72 hrs :
            - plasma volume restored to normal
            - Albumin replaced rapidly from extravascular stores
        - Days:
            - plasma proteins and enzymes: ↑ liver synthesis
        - Days to weeks:
            - RBC's: ↑EPO from kidneys ⇒
                - *reticulocytes* peak day 10 days (norm ~1% retics in blood)
                - mature RBC's back to normal 4 – 8 weeks.
            - Other: PLT's, WBC's
- Can also be classified by severity:
    - mod shock ⇒ ↓pulse pressure
        - due to diastole caused by catecholamines ↑ing vascular tone
        - ⇒ ↓discharge baroreceptors ⇒ ↑symp tone ⇒ ↑VC & ↑HR
    - 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 ⇒
    - ↑anaerobic glycolysis ⇒ lactic acid accumulation
    - low/mod levels of lactic acid excellent fuel for heart/CVS system
        ↳ but tipping point ⇒ acidosis
- lactic acidosis ⇒
    - ↓myocardial contractility
    - ↓vascular response to catecholamines ie ↑ed VD
    - 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

  - initially caused by ↓vagal output
  - later by ↑ed SNS output
- stroke volume:
  - ↑in non-linear way
  - big ↑in light/mod exercise; only small ↑ into severe exercise
  - reasons for ↑:
    - ↑VR & ∴ ↑LVEDV
    - ↑contractility ⇒ ↓LVESV
- blood pressure:
  - SBP can rise to 190-225mmHg 2nd to ↑ed CO
  - 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:
  - mm blood flow = 2-3ml/100g/min

    ↳ mediated by SNS constriction of arterioles
  - ~20% of CO – despite skeletal mm being ~40% of lean body mass
  - precapillary sphincters closed ⇒ diverts mm blood flow away from microcirculation to main channels
- @exercise see:
  - 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
  - ↑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:
  - must meet extra cardiac work
  - 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:
  - remains constant at all levels of ex ~50ml/100g/min
  - 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 5-10seconds 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:
  - 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
  - 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 symp ns stim to heart (cardiac symp nerve stim):
  - ↑MAP ⇒ ↓CVP both of which favour ↓ed SV (ie opposite of desired effect)
- in exercise:
  - [↑afterload] ↑ed MAP minimised by VasoD of exercising mms
  - [preload] ↓CVP minimised by:
    - periph venoC
    - mm & thoracic pumps encouraging VR
- in upright exercise SV can double due to:
  - [preload] ↑EDV (from ↑CVP)
  - [contractility] ↓End systolic volume – from ↑EF via ↑ed contractility

CO = VR

Ohms Law:

$$VR = \frac{MSP - RAP}{VVR}$$

MSP = mean systemic pressure ~ 7mmHg
RAP ~2-3 mmHg
VVR = venous vascular resistance
↳ Δ**P** ~ 5mmHg ie venous resistance is v low

c/f

$$LHCO = \frac{MAP - RAP}{SVR}$$

LHCO = L heart CO
Δ**P** ~ 88mHg

c/f

$$RHCO = \frac{MPAP - LAP}{PVR}$$

RHCO = R heart CO
Mean Pulmonary artery P ~ 15mmHg
LAP 5mmHg
Δ**P** ~10mHg