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ANESTHESIOLOGY UPDATES

for Postgraduates



Sampa Dutta Gupta

JAYPEE

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4

Cardiovascular and Respiratory Physiology

Anupam Goswami

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Knowledge of the principles of cardiovascular and respiratory physiology is the foundation for the practice of anesthesia. This chapter reviews few aspects of cardiovascular and respiratory physiology for the understanding of both its scientific significance and its practical applications to patient management.

RESPIRATORY PHYSIOLOGY WITH ANESTHETIC IMPLICATIONS

- Anesthesia causes impairment in pulmonary function, whether the patient is breathing spontaneously or is ventilated mechanically after muscle paralysis
- Impaired oxygenation of blood occurs in most subjects who are anesthetized
- Lung function remains impaired postoperatively, and clinically significant pulmonary complications can be seen in 1–2% after minor surgery in up to 20% after upper abdominal and thoracic surgery.

Lung Volumes during Anesthesia (Fig. 1)

- Functional residual capacity (FRC) is reduced by 0.8–1.0 L by changing body position from upright to supine, and there is another 0.4–0.5 L decrease when anesthesia has been induced
- Anesthesia *per se* causes a fall in FRC despite maintenance of spontaneous breathing

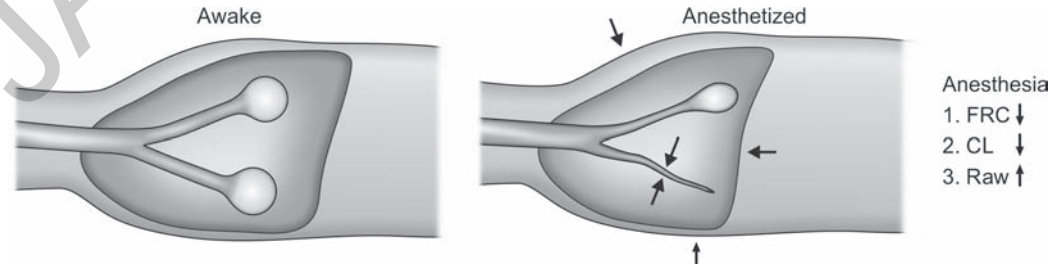

Fig. 1 Airway closure during anesthesia

- End-expiratory lung volume is thus reduced from approximately 3.5–2 L, the latter being close or equal to RV
- The decrease in FRC occurs regardless of whether the anesthetic is inhaled or given intravenously
- Muscle paralysis and mechanical ventilation cause no further decrease in FRC
- The average reduction corresponds to around 20% of awake FRC and may contribute to an altered distribution of ventilation and impaired oxygenation of blood
- The decrease is related to loss of respiratory muscle tone, which shifts the balance between the elastic recoil force of the lung and the outward force of the chest wall to a lower chest and lung volume
- Maintenance of muscle tone, as during ketamine anesthesia, does not reduce FRC
- Magnitude of FRC reduction is related to age and body weight, as obese patients demonstrate a much higher decrease in FRC
- The FRC increases with age if weight and height remain unaltered over the years.

Mechanism of FRC Reduction during Anesthesia

- Atelectasis
- Increased expiratory muscle activity
- Air trapping in distal airway
- Cephalic displacement of diaphragm
- Decreased outward chest wall recoil
- Increasing lung recoil
- Increased thoracic blood volume
- Atelectasis appears in approximately 90% of all patients who are anesthetized
- It is seen during spontaneous breathing and after muscle paralysis and whether intravenous or inhaled anesthetics are used
- 15–20% of the lung is regularly collapsed at the base of the lung during uneventful anesthesia
- It may also be mentioned that after thoracic surgery and cardiopulmonary bypass, more than 50% of the lung can be collapsed even several hours after surgery
- The amount of atelectasis decreases toward the apex, which is mostly spared
- Obese patients showing larger atelectatic areas than lean ones do
- Atelectasis is independent of age, with children and young people showing as much atelectasis as elderly patients
- COPD patients show much less amount of atelectasis
- There is a positive correlation between atelectasis and amount of shunting
- It is likely that it is a focus of infection and can contribute to pulmonary complications.

Prevention of Atelectasis during Anesthesia

Positive End-expiratory Pressure

- The application of 10 cm H_2O positive end-expiratory pressure (PEEP) will consistently reopen collapsed lung tissue
- Shunt is not reduced proportionately, and arterial oxygenation may not improve significantly by PEEP
- The persistence of shunt may be explained by a redistribution of blood flow toward more dependent parts of the lungs when intrathoracic pressure is increased by PEEP
- Increased intrathoracic pressure will impede venous return and decrease cardiac output
- Lung recollapses rapidly after discontinuation of PEEP. Within 1 minute after cessation of PEEP, the collapse is as large as it was before the application of PEEP.

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Maintenance of Muscle Tone

- Use of an anesthetic that allows maintenance of respiratory muscle tone will prevent atelectasis from forming
- Ketamine does not impair muscle tone and does not cause atelectasis
- Another technique used in an attempt to restore respiratory muscle tone is pacing of the diaphragm. This was tested by applying phrenic nerve stimulation, which did reduce the atelectatic area.

Recruitment Maneuvers

- The use of a sigh maneuver, or a double Vt, has been advocated to reopen any collapsed lung tissue
- For complete reopening of all collapsed lung tissue, an inflation pressure of 40 cm H_2O is required
- Such a large inflation corresponds to a maximum spontaneous inspiration, and it can thus be called a VC maneuver.

Minimizing Gas Resorption

- Avoidance of the preoxygenation procedure (ventilation with 100% O_2) eliminated atelectasis formation during induction and subsequent anesthesia
- Preoxygenation can also be provided without producing atelectasis if undertaken with continuously increased airway pressure, as with continuous positive airway pressure (CPAP)
- Postoxygenation and airway suctioning at the end of surgery also promote atelectasis.

Compliance and Resistance during Anesthesia

- Static compliance of the total respiratory system (lungs and chest wall) is reduced on an average from 95–60 mL/cm H_2O during anesthesia
- Static lung compliance also decreases during anesthesia
- Resistance to total respiratory system and lung also increases during anesthesia.

Distribution of Ventilation during Anesthesia (Fig. 2)

In the upper part of the lung, the alveoli and airways are open (zone A). In the middle and lower parts of the lung, the airways are intermittently closed and impede ventilation (zone B), and in the bottom of the lung, the alveoli have collapsed (atelectasis, zone C). The corresponding ventilation-perfusion distribution as assessed by the multiple inert gas elimination technique can be seen in the *right panel*. Modes A and B correspond to a well-ventilated and perfused lung region and a region with intermittent airway closure, respectively, and are similar to what was shown in. In addition, there is a shunt that is caused by perfusion of the atelectatic zone (C) in the bottom of the lung.

Regional Blood Flow during Anesthesia

- A successive increase in perfusion down the lung, from the ventral to the dorsal aspect is evident during anesthesia in supine subject, with some reduction in the lowermost region
- PEEP will impede venous return to the right heart and therefore reduce cardiac output
- PEEP causes a redistribution of blood flow toward dependent lung regions.

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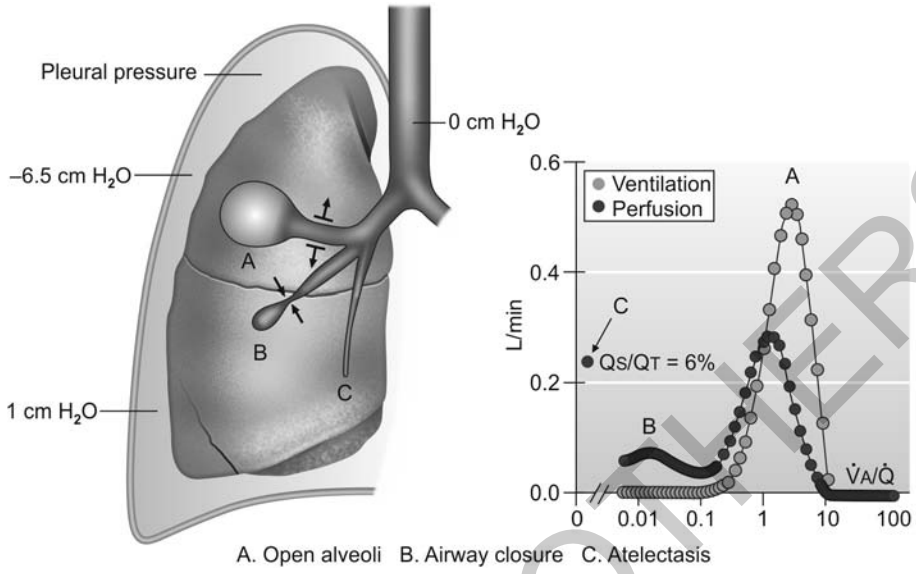

Fig. 2 Distribution of ventilation during anesthesia

Ventilation-perfusion mismatching during Anesthesia

- Both CO_2 elimination and oxygenation of blood are impaired in most patients during anesthesia
- The impeded CO_2 elimination can be attributed to increased dead space ventilation
- Anatomic dead space is unchanged, indicating that the "alveolar" dead space must have increased during anesthesia
- Increased dead space during anesthesia is not really dead space but poorly perfused lung regions that are signified by so-called high V/Q ratio
- Such high V/Q ratio can be explained by the tiny perfusion of corner vessels in interalveolar septa in the upper lung regions (where alveolar pressure may exceed pulmonary vascular pressure [zone I])
- The impaired CO_2 elimination is most easily corrected by increasing the ventilation and is seldom a problem in routine anesthesia with mechanical ventilation
- The impairment in arterial oxygenation during anesthesia is more severe at higher ages, obesity worsens the oxygenation of blood, and smokers show more impairment in gas exchange than nonsmokers do
- Venous admixture, as calculated according to the standard oxygen "shunt" equation, is also increased during anesthesia to approximately 10% of cardiac output.

Hypoxic Pulmonary Vasoconstriction

- Several inhaled anesthetics have been found to inhibit HPV
- No such effect has been seen with intravenous anesthetics
- Nitrous oxide does not have any significant effect upon HPV.

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Factors Increasing Pulmonary Vascular Resistance

- Reduction in alveolar PO_2 (HPV)
- A rise in CO_2, acidosis
- Epinephrine, dopamine, histamine, 5-HT
- Lung collapse.

Factors Reducing Pulmonary Vascular Resistance

- Increased cardiac output by recruiting capillaries
- Acetylcholine and cholinergics
- Isoprenaline
- Prostacyclin.

Effects of Anesthetics on Respiratory Drive

- The inhalation anesthetics, opioids and sedative-hypnotics are all respiratory depressant
- Opioids and inhalation agents depress the ventilatory response to hypercarbia
- Opioids decreases respiratory rate and increases tidal volume, but minute ventilation is reduced
- Volatile agents and barbiturates increases respiratory rate but decreases tidal volume
- Anesthesia also reduces the response to hypoxia, but less than hypercarbia
- Attenuation of the hypoxic response may be attributed to an effect on the carotid body chemoreceptor
- The reduced ventilatory response to CO_2 during anesthesia is due to impeded function of the intercostal muscles.

Regional Anesthesia and Lung Function

- The ventilatory effects of regional anesthesia depend on the type and extension of motor blockade
- With extensive blocks that include all of the thoracic and lumbar segments, inspiratory capacity is reduced by 20% and expiratory reserve volume approaches zero
- Diaphragmatic function, however, is often spared, even in cases of inadvertent extension of subarachnoid or epidural sensory block up to the cervical segments
- Arterial oxygenation and carbon dioxide elimination are well maintained during spinal and epidural anesthesia
- Closing capacity (CC) and FRC relationship remain unaltered
- Ventilation-perfusion ratio remains unaltered during epidural anesthesia in all lung regions.

Pneumoperitoneum and Lung Function

- CO_2 pneumoperitoneum may cause hypercapnia and acidosis
- The direct effects of carbon dioxide and acidosis lead to decreased cardiac contractility, sensitization of the myocardium to the arrhythmogenic effects of catecholamine, and systemic vasodilatation
- Pneumoperitoneum causes decreased FRC and VC, formation of atelectasis, reduced respiratory compliance, and increased peak airway pressure
- Shunt is reduced and arterial oxygenation is mostly improved during CO_2 pneumoperitoneum
- CO_2 may enhance HPV, which helps in efficient redistribution of lung blood flow.

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CARDIOVASCULAR PHYSIOLOGY WITH ANESTHETIC IMPLICATIONS

Blood Pressure

Blood pressure = Cardiac output × peripheral vascular (arteriolar) Resistance (PVR). Cardiac output = Stroke volume × Heart rate.

So, blood pressure = Stroke volume × heart rate × peripheral arteriolar resistance.

Factors Determining Blood Pressure

- Factors determining stroke volume (stroke volume determines mainly the systolic blood pressure).
- Factors determining heart rate.
- Factors determining peripheral vascular (arteriolar), resistance (PVR determines mainly the diastolic blood pressure).

- *Factors determining stroke volume:*
  - *VEDV (ventricular end-diastolic volume).* Which is dependent on (a) Venous return, which in turn depends on position of body, skeletal muscle pump, and respiratory pump (preload), (b) Blood volume.
  - *Strength of myocardial contraction (inotropicity).*
  - *Ventricular afterload.*
- *Factors determining heart rate*—depends on autonomic nerve stimulation, circulating chemicals, activity and exercise, emotional stress, gender, age, body temperature, and baroreceptor reflex.
- *Factors determining peripheral arteriolar resistance*—constriction and dilatation of the arterioles are the main determinants.

Control of Blood Pressure

The cardiovascular center (CVC) is a collection of interconnected neurones in the medulla and pons.

CVC receives, integrates and coordinates inputs from:
- Baroreceptors (the primary mechanism)
- Chemoreceptors
- Higher centers in the brain.

CVC—via the autonomic nervous system (ANS) to the heart and blood vessels enables rapid response to changes in blood pressure.

Factors Modifying Systolic Blood Pressure

Systolic blood pressure is an interaction between stroke volume and distensibility (compliance) of the arterial system which decreases on ageing resulting in isolated/disproportionate systolic hypertension in the elderly.

The role of baroreceptors and chemoreceptors in the control of arterial blood pressure are shown in Flow charts 1 and 2.

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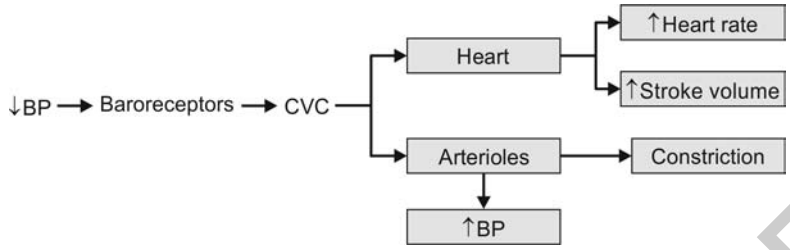
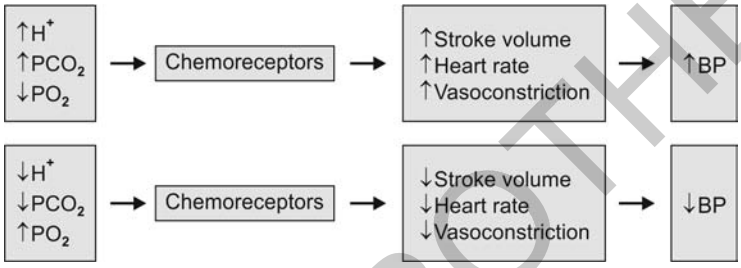
Flow chart 1 The role of baroreceptors in the control of arterial blood pressure



Flow chart 2 The role of chemoreceptors in the control of arterial blood pressure



Factors Modifying Diastolic Blood Pressure

Diastolic blood pressure is principally determined by cardiac afterload. SVR is a major contributor to diastolic BP. Diastolic BP itself is a determinant of coronary arterial filling and both duration of diastole and diastolic blood pressure have a bearing on the adequacy of coronary perfusion.

Measurement of Blood Volume

^{51}Cr-radiolabeled red cells are injected into the circulation, and blood volume may be calculated from:

$$\text{Blood volume (mL)} = \frac{\text{Quantity of test substance}^{**}\text{ injected}}{\text{Concentration of test substance}^{**}\text{ observed}}$$

(**Test substance is labeled red cells)

When radiolabeled albumin or Evans blue is injected initially, it will reside in plasma, its volume may be calculated from:

$$\text{Plasma volume (mL)} = \frac{\text{Quantity of test substance}^{**}\text{ injected}}{\text{Concentration of test substance}^{**}\text{ observed in plasma}}$$

(**Test substance is radiolabeled albumin or Evans blue)

$$\text{Blood volume (mL)} = \text{Plasma volume} \times \frac{100}{100 - \text{hematocrit}}$$

Fick Principle

The Fick principle states that the amount of a substance taken up by an organ (or by the whole body) per unit of time is equal to the arterial level of the substance minus the venous level multiplied by the blood flow (Q). It can be employed to calculate blood flow, or to calculate VO_2 (O_2 consumption).

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To derive the difference in O_2 content, simultaneous arterial (CaO_2) and mixed venous (CvO_2) samples are measured.

$$Q = \frac{VO_2}{(CaO_2 - CvO_2)}$$

Mixed Venous O_2 Saturation

Mixed venous O_2 saturation (SvO_2) is the percentage of oxygenated mixed venous blood which may be measured photometrically at the tip of a pulmonary artery (PA) catheter.

It is increased in sepsis with peripheral shunting, cyanide toxicity, hypothermia, sampling on wedging of a PA catheter (erroneously collecting pulmonary venous blood).

It is decreased in anemia, low cardiac output, arterial O_2 desaturation, increased O_2 consumption.

O_2 Flux/Content

- Oxygen flux (mL) = CO × CaO_2
- O_2 content (mL/dL blood) = (1.31 × Hb × saturation/100) + 0.02 PO_2

Arteriovenous O_2 Difference

This is the difference in oxygen tension between the arterial and venous circulations, reflecting the oxygen consumption of an organ or of the whole body.

Oxygen Consumption

- O_2 content = 1.31 × (Hb × Sat/100) + 0.02 PO_2
- For CvO_2, pulmonary arterial sample from a PA catheter tip without wedging is required
- For CaO_2 arterial sample is required
- Q (CO) is obtained from calorimetric measurement (PAC)
- VO_2 (O_2 consumption) is calculated from Fick's equation.

$$Q = \frac{VO_2}{(CaO_2 - CvO_2)}$$

Diminished VO_2 is the earliest pathophysiological event in shock, and usually precedes the hypotension.

Ejection Fraction (EF) of Left Ventricle

$$EF = \frac{\text{Stroke volume}}{\text{Left ventricular end-diastolic volume}}$$

Measured by Doppler at echocardiography: Normal > 0.55 or 55%

Starling's law of heart: Stroke volume is proportional to left ventricular end-diastolic volume/pressure/length of the myofibril (Fig. 3).

Implications: The failing heart will generate less stroke volume for the same end-diastolic volume in comparison with a nonfailing heart. If compliance of the heart is assumed to be constant then stroke volume is proportional to the end-diastolic pressure.

If mitral valve functions normally then: LVEDP = LAP = PCWP . SV ∞ PCWP.

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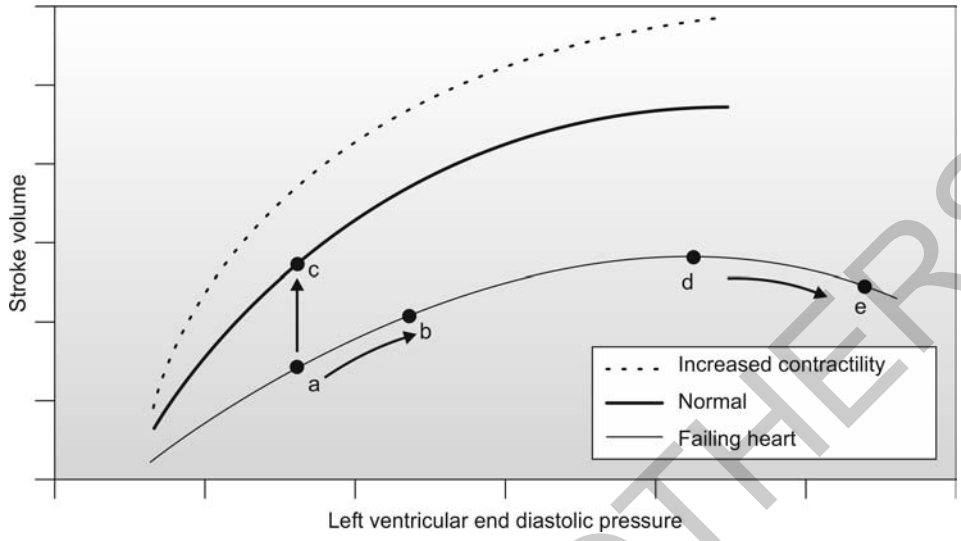

Fig. 3 Starting's law of heart

Starling Forces in Capillaries

P_c = Capillary hydrostatic pressure (varies from artery to vein)

P_{if} = Interstitial hydrostatic pressure (usually 0/constant)

Π_p = Oncotic pressure due to plasma proteins (28 mm Hg/constant)

Π_{if} = Oncotic pressure due to interstitial proteins (3 mm Hg/constant)

Net filtration = $(Pc - P_{if}) - (\Pi_p - \Pi_{if})$

Starling Forces and Blood Loss

If patient loses blood acutely, then the capillary hydrostatic pressure falls especially at the venous end. Balance is no longer maintained, the net pressure into the capillaries increases and fluid is retrieved into the circulation from the interstitium until P_c is restored. Flow chart 3 shows physiology response to hemorrhage.

1. What are the factors that regulate peripheral resistance?

Ans. A fast acting mechanism, controlled by three mechanisms:

(1) Intrinsic, (2) Extrinsic, (3) Paracrine.

Extrinsic mechanism is controlled through several reflex mechanisms, most important is baroreceptors reflex, *chemoreceptors reflex and other vasomotor reflexes.*

Baroreceptors are receptors found in carotid sinus and aortic arch and are stimulated by changes in BP. *Chemoreceptors reflex*: Chemoreceptors are receptors found in carotid and aortic bodies are stimulated by chemical changes in blood mainly hypoxia ($\downarrow O_2$), hypercapnia ($\uparrow CO_2$), and pH changes. *Other vasomotor reflexes* include *atrial stretch receptor reflex where, with increase in* venous return lead to stimulation of atrial stretch receptors cause reflex vasodilatation and lowering of BP.

Thermoreceptors: Present in skin/or hypothalamus when exposure to heat cause vasodilatation, when expose to cold cause vasoconstriction.

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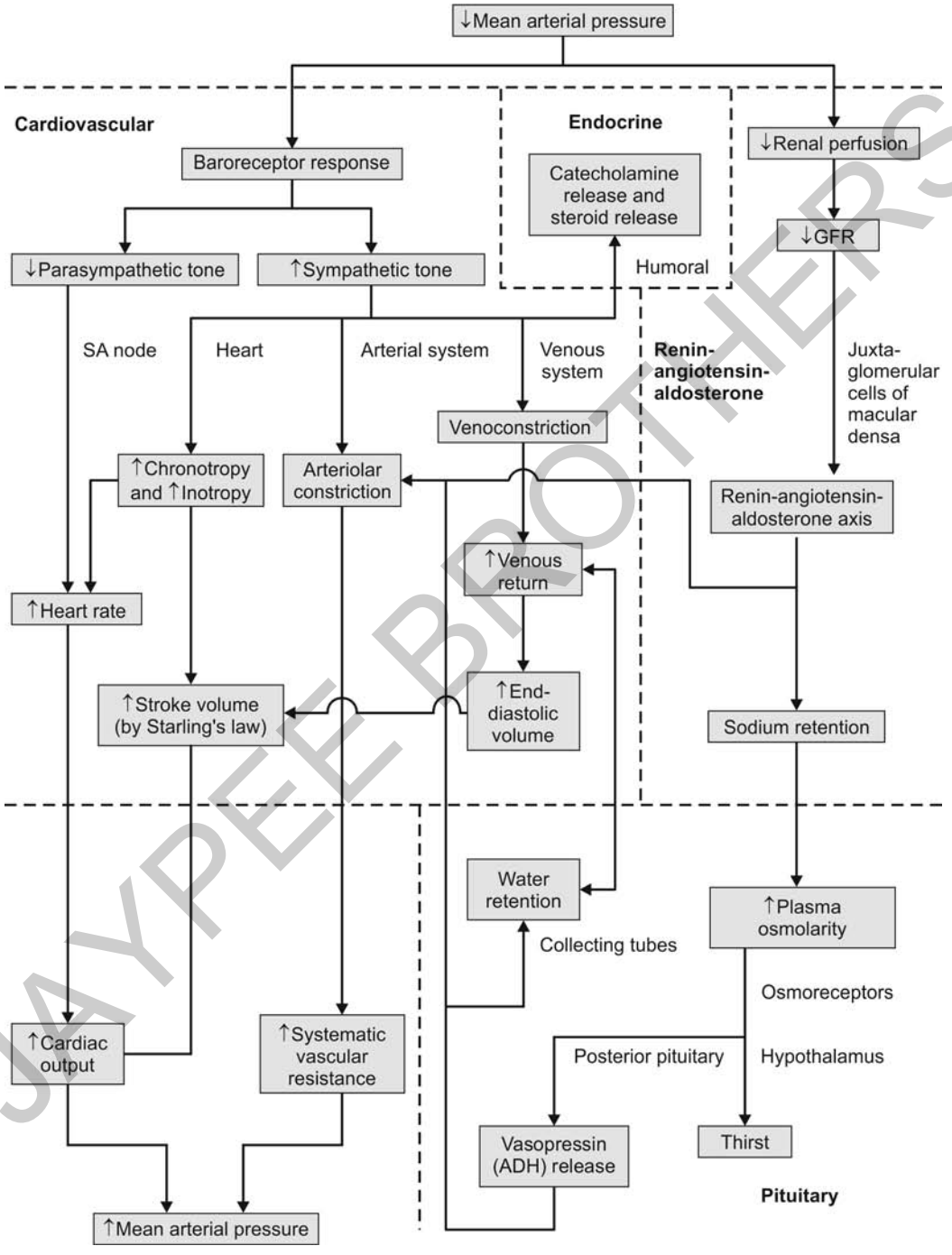
Flow chart 3 Physiological response to hemorrhage



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Pulmonary receptor: Vasoconstriction *with* lung inflation

Among hormonal agents, noradrenaline cause vasoconstriction, adrenaline cause vasoconstriction (except in skeletal muscle). Angiotensin II cause vasoconstriction, vasopressin lead to vasoconstriction.

Autoregulation of Blood Flow

Autoregulation is the ability of an organ to control its own blood supply independently of neural and hormonal influences. There are two main mechanisms whereby it is mediated:

- Fall in arterial pressure resulting in a reduction in blood flow, accumulation of metabolites thus causdilates terminal arterioles restoring blood flow.
- Myogenic response involving local neural reflexes in response to stretch at the level of the 1st order arteriole.

Valsalva Maneuver

The valsalva maneuver (Fig. 4) is forced expiration against closed glottis.

Effects:
There is increase in intrathoracic pressure and decrease in venous return.
Normal person maintains mean arterial pressure by increasing heart rate and systemic vascular resistance, and demonstrates on release transient hypertension and bradycardia.

Phases:
Phase I: Increase in intrathoracic pressure and MAP.
Phase II: Decreased venous return and MAP.
Phase III: Overshoot as vasoconstriction and increase in heart rate persists.
Phase IV: Return to normal.

The normal valsalva response is absent or abnormal in autonomic dysfunction, in particular in autonomic neuropathy in DM and after sympathectomy.

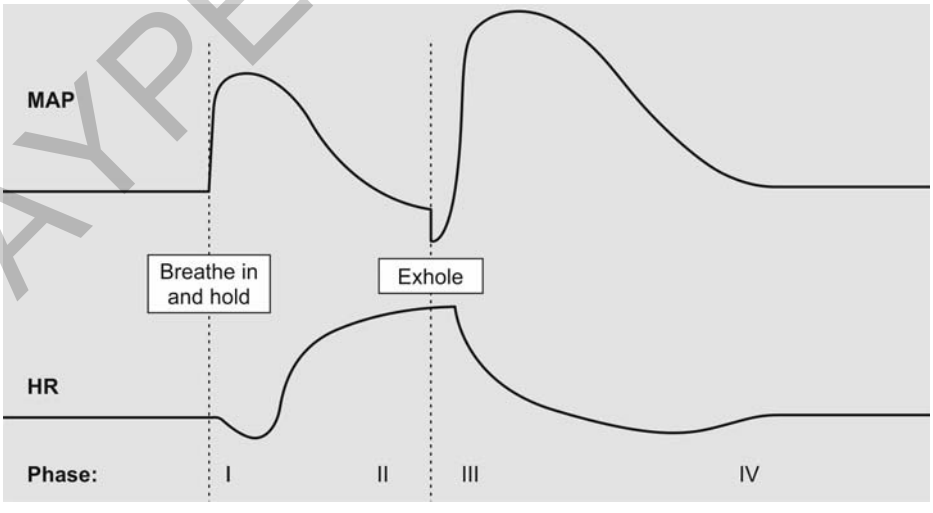

Fig. 4 Valsalva maneuver

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Cardiac Action Potential

Specialized cardiac myocytes in the SA node generate cardiac action potential (AP) which is transmitted through the heart via the conducting system causing normal myocytes to contract. The cells of the conducting system are modified myocytes classified as nodal, transitional and Purkinje myocytes and all are capable of spontaneous and rhythmic generation of APs. Any focus generating cardiac action potential at a higher frequency acts as the pacemaker. Normally, in sinus rhythm this is the SA node.

There is an electrical charge difference across the myocardial cell membrane in the resting/polarized state known as resting membrane potential (RMP). Potassium (K^+), sodium (Na^+), calcium (Ca^{2+}) ions are principally responsible for this. Concentration of (K^+) inside the cell is 151 mEq/L and 4 mEq/L outside. For Na^+ these are 144 mEq/L outside and 7 mEq/L inside, and for Ca^{2+} these are 5 mEq/L outside and less than 1 mEq/L inside. In the resting or polarized state the inside of the cell is negatively charged and outside is positive. The Na^+/K^+ pump establishes an increased Na^+ concentration outside and an increased K^+ concentration inside the cell. Both ions diffuse along their concentration gradient, K^+ coming outside while Na^+ diffusing inside the cell. For every 50–75 K^+ ions diffusing out, only one Na^+ diffuses inside, causing a deficiency of positive cations inside the cell creating a RMP of magnitude around –90 mV. As at rest the membrane is more permeable to K^+ than Na^+ the potential difference (Nernst equation) applies more to K^+ (–90 mv) than to Na^+ (+ 60 mv). It is the continuation of depolarization which distinguishes cardiac action potential (AP) from skeletal AP. This is due to the action of slow Ca^{2+} channels, allowing Ca^{2+} entry into the cell, maintaining a balance against K^+ diffusing outwards, causing a prolonged depolarization.

- Ca^{2+} is functionally related to β-receptors
- K^+ is functionally related to M2 receptors and β-receptors
- Changes in external K^+ affect the resting membrane potential level
- Changes in internal Na^+ affect the magnitude of the action potential.

Phases for Depolarization

Depolarization is the trigger for myocardial contraction.

- 0 = Fast depolarization, Na^+ inwards
- 1 = Early incomplete repolarization
- 2 = Plateau, slow Ca^{++} inwards, prolonging AP
- 3 = Rapid repolarization, K^+ outwards
- 4 = Electrical diastole, refractory period.

Pacemaker Cell Action Potential

Action potential, that is produced when a pacemaker cell depolarizes, is a slow response type AP, which is classified into phases (0–4):

Heart rate is altered by slope of the prepotential (phase 4), which is affected by:

- Sympathetic system—norepinephrine
- Parasympathetic system—Ach
- Temperature.

Difference Between the Pacemaker and Myocyte Action Potential (Figs 5 and 6)

Pacemaker has:

- Less negative phase 4 membrane potential
- Less negative threshold potential

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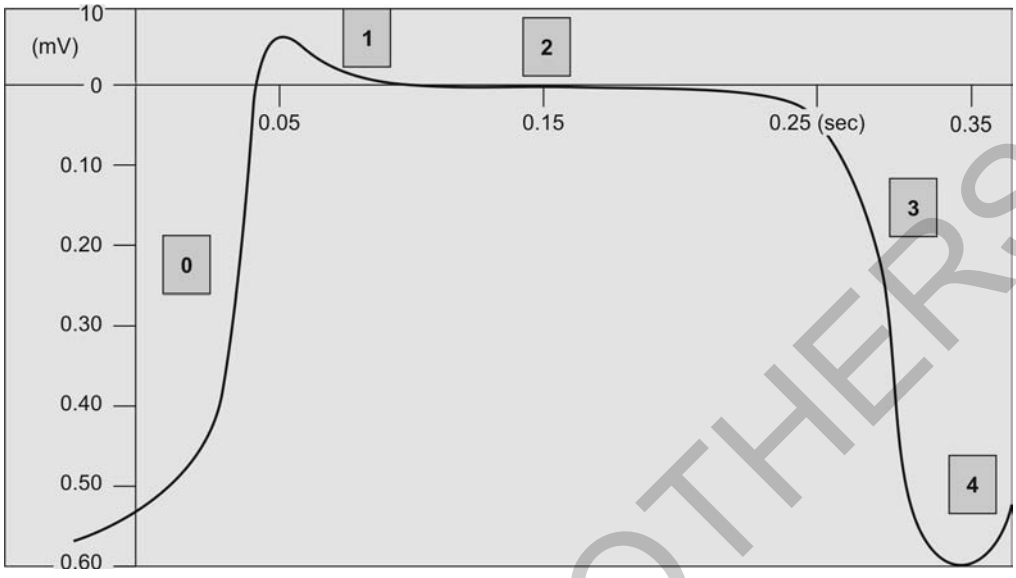

Fig. 5 Myocardial AP

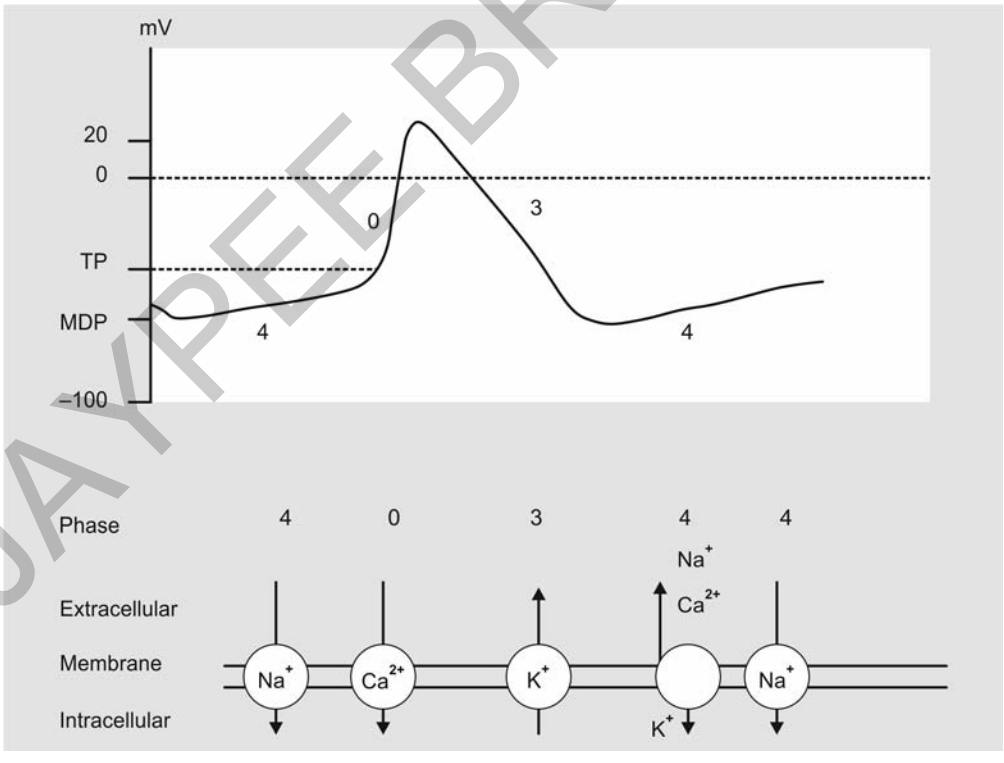

Fig. 6 Pacemaker AP

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- Spontaneous depolarization in phase 4
- Less steep slope in phase 0
- Absence of phase 2 (plateau).

Coronary Circulation

Coronary blood flow (CBF) occurs in diastole in the left ventricle, because intramyocardial vessels are compressed in high systolic pressure.

CBF is metabolically controlled, myocardial hypoxia is a potent dilator of the coronary arterioles.

In the right ventricle CBF occurs both during systole and diastole.

Coronary blood flow at rest is 250 mL/min. Myocardial O_2 consumption is 11 mL/100 g tissue/min (for skeletal muscle it is 8 mL/100 g tissue/min).

Coronary sinus (venous) PO_2 is very low as O_2 extraction is maximal, so increased demand can be met by increased flow only.

Increased heart rate decreases diastolic time and coronary blood flow.

Myocardial hypertrophy compresses vessels decreasing myocardial oxygen supply.

Changes in the Fetal Circulation

The key is the change in pressure induced by the sudden fall in pulmonary vascular resistance (PVR). Prior to birth the pulmonary vascular resistance is high, as the lung inflates and PVR falls, there is sudden reduction in right sided pressure, and pressure in left side exceeds that on the right. For this pressure change the foramen ovale closes, oxygenated blood flows retrograde in the ductus arteriosus and it closes.

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