

APPLIED CARDIOVASCULAR PHYSIOLOGY

The **cardiovascular reflexes** Cardiac reflexes are fast-acting reflex loops between the heart and central nervous system (CNS) that contribute to regulation of cardiac function and maintenance of physiologic homeostasis. As with most homeostasis questions it is useful to structure your response into



Cardiac receptors are linked to the CNS by myelinated or unmyelinated afferent fibers that travel along the vagus nerve. Cardiac receptors can be found in the atria, ventricles, pericardium, and coronary arteries. Extracardiac receptors are located in the great vessels and carotid artery. Sympathetic and parasympathetic nerve input is processed in the CNS. After central processing, efferent fibers to the heart or the systemic circulation will provoke a particular reaction. The **baroreceptor reflex** is the most important cardiac reflex and maintains second to second control of BP via a negative feedback loop. Long term it acts via a set point which may be elevated in settings such as essential hypertension. Changes in blood pressure are monitored by circumferential and longitudinal stretch receptors located in the carotid sinus and aortic arch. The CPU is the NTS in the cardiovascular centre of the medulla. The effectors are the autonomic nervous system. **Chemoreceptor Reflex** -Chemosensitive cells are located in the carotid bodies and the aortic body. These cells respond to changes in pH status and blood oxygen tension. The **Bainbridge reflex** is elicited by stretch receptors located in the right atrial wall and the cavoatrial junction. An increase in rightsided filling pressure sends vagal afferent signals to the cardiovascular center in the medulla. These afferent signals inhibit parasympathetic activity, thereby increasing the heart rate. The purpose of this reflex is to ensure the extra blood arriving at the heart is pumped out. The **Bezold-Jarisch reflex** responds to noxious ventricular stimuli sensed by chemoreceptors and mechanoreceptors within the LV wall by inducing the triad of hypotension, bradycardia, and coronary artery dilatation. The **Cushing reflex** is a result of cerebral ischemia caused by increased intracranial pressure. Cerebral ischemia at the medullary vasomotor center induces initial activation of the sympathetic nervous system. Such activation will lead to an increase in heart rate, blood pressure, and myocardial contractility in an effort to improve cerebral perfusion. As a result of the high vascular tone, reflex bradycardia mediated by baroreceptors will ensue. The **oculocardiac reflex** is provoked by pressure applied to the globe of the eye or traction on the surrounding structures. Stretch receptors are located in the extraocular muscles and their activation results in increased parasympathetic tone. - See Miller 7th edition for further details

Shock is defined as inadequate tissue perfusion resulting in tissue damage. It is usually further classified according to the physiological cause into four categories. **Hypovolaemic shock** is due to a **decrease in the circulating blood volume**, most commonly due to haemorrhage, but gastrointestinal or non sensible losses may also cause this state. **Low resistance shock** is a result of the a **dramatic decrease in total peripheral resistance**, this may be seen in spinal cord injuries, anaphylaxis and most commonly excessive inflammatory response in sepsis. **Cardiogenic shock** is due to **decreased cardiac output** from an **intrinsically cardiac cause**, normally due to a large MI, myocarditis, or valvular disease. **Obstructive shock** is due to a **decrease in cardiac output due to obstruction** from an cause **extrinsic to the heart**, such as cardiac tamponade, tension pneumothorax or PE.

Cardiovascular response to shock is similar for all types of shock, however the underlying pathology will determine the degree of success to the physiological response. Note this information has already been covered in responses to 1000ml of blood loss. **Blood pressure is maintained** by baroreceptor response and sympathetic activation resulting in increased CO (preload, contractility and HR) and reduced run-off (increased TPR), Renin-angiotensin-aldosterone system activation, Chemoreceptors, Vasopressin, Adrenergic release by the adrenal medulla augments sympathetic response and finally there is increased thirst. **Blood redistribution is also an important temporiser**; decreased renal perfusion (which in combination with the RAAS system and Vasopressin release also decreases urine output), decreased muscle perfusion, maintained cerebral perfusion and a net reabsorption of interstitial fluid due to starling forces. **Delayed responses** in the setting of shock due to blood loss include hepatitic synthesis of plasma proteins, increased erythropoietin and subsequent erythropoiesis will lead to a reticulocyte count peak at day ten.

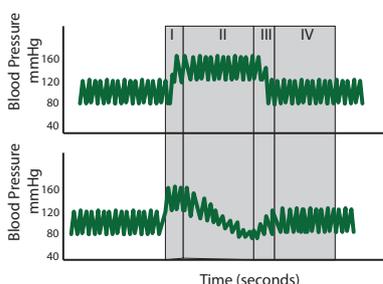
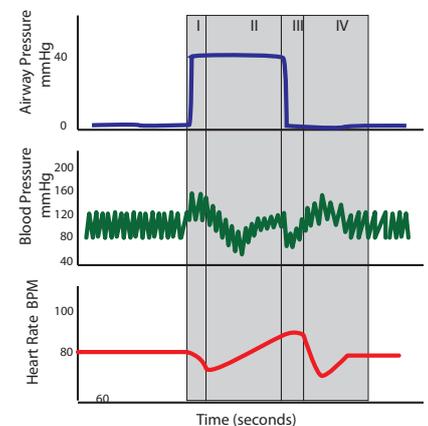
Normal aging Myocardial changes include: A **decrease** in the resting and maximum **cardiac index**, the **maximum heart rate**, diastolic compliance, **sensitivity to catecholamines** and the **number of functioning myocytes**. There is an accumulation of pigment within the myocytes and **increase** in contraction and relaxation time (**cardiac cycle**) of the heart. **Peripheral vascular changes** include: An **increase in total peripheral resistance** as well as **decreases in arterial and venous compliance** (less windkessel effect), and capillary density in some tissue beds. A **blunted baroreceptor response mechanism** due to decreased stretch in the carotid and aortic bodies and impaired response due to impaired sympathetic responses (less noradrenaline and adrenaline sensitivity). **Haemodynamic consequences** of the above include: **increased pulse pressure (windkessel)** and **mean arterial pressure, increased afterload** due to raised blood pressure.

Cardiovascular effects of IPPV and PEEP. (from respiratory section) IPPV and PEEP cause a reduction in cardiac output by **reducing venous return to the right atrium** because of increased intrathoracic pressure. With normal inspiration there is negative intrathoracic pressure which acts as a pump to draw blood into the chest from the major veins and this is abolished with positive intrathoracic pressure. The reduction in RV filling leads to less LV filling and therefore preload, which is exacerbated in hypovolaemic states. Furthermore the **increased airway pressures lead to increased pulmonary vascular pressures and this increases RV strain**. It is noted that whilst PEEP may improve PaO2 the decrease in CO may actually lead to decreased O2 delivery to the tissues (remember that $DO = CO(\text{sats} \times \text{Hb} + \text{dissolved O}_2)$). There is some **increase in peripheral vascular resistance to counter the decreased CO** but this is often inadequate. Interestingly, in heart failure patients the reduced RV filling may actually improve function by moving an overloaded RV to a more favourable position on the starling curve.

Cardiovascular changes during pregnancy include; **Upward and leftward cardiac displacement**, aortocaval compression after 20/40 with associated maternal hypotension and decreased uteroplacental flow (hence left lateral positioning), Placental blood flow of 625 mLmin⁻¹, **30-50% increase in circulating volume** (mediated by aldosterone, oestrogen and progesterone) ~12% **increase in HR**, 25% **increase in stroke volume**, 30-40% **increase in cardiac output**, 20-40% **decrease in TPR** due to the placental circulation (low resistance running in parallel with other systemic organs), 5-10 mmHg decrease in diastolic pressure at 12-20/40, **increase in renal plasma flow and GFR (75% and 50% respectively)**, During labor: **300-500 mL autotransfusion with uterine contractions** and ~ 25% increase in cardiac output.

Valsalva Manoeuvre was first described by the Italian Anatomist Antonio Valsalva in the early 18th century. It refers to the cardiovascular responses to forced expiration against a closed glottis for a duration of ten seconds or more. There are four main phases described in the diagram below.

- Phase I** Increased intrathoracic pressure causes a **brief increase in preload** as the pulmonary vasculature empties to the LA/LV. This causes an increase in CO and therefore blood pressure and a **fast baroreceptor driven decrease in heart rate**.
- Phase II** The increased intrathoracic pressure causes **reduced venous return which leads to decreased preload and CO**. As blood pressure now falls the **baroreceptor response is an increase in HR and TPR**. The increase in TPR means that the diastolic pressure will be increased more than the systolic and as a result the **pulse pressure narrows**.
- Phase III** The intrathoracic pressure now drops, **transiently decreasing preload and CO** as blood pools in the pulmonary vasculature. There is a transient decrease in blood pressure.
- Phase IV** As CO returns to normal, the residual increase in TPR (and probable venous constriction) means that there is an **overshoot** and blood pressure spikes (**with associated fast baroreceptor decrease in HR**) before **stabilising**.



In **heart failure** most mechanisms are blunted and the pressure on the aorta leads to an increase in blood pressure and a characteristic 'square wave' pattern of blood pressure changes.

In **autonomic dysfunction** the TPR is not increased which impairs the response of phase II and removes the cause of the overshoot in Phase IV.