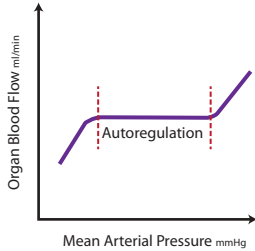


# REGIONAL VASCULAR SYSTEM

**Regional circulations** The resting cardiac output is 5-6 litres per minute and this may be divided up between the various organs in the systemic circulation. Flow is determined by the pressure change divided by the resistance. From this we know can derive that the **percentage of each organ blood flow is dependent on the organ vascular resistance** (as the pressure change from the arterial to venous side is constant across all organs). **Most of the resistance** in the systemic system **results from the arterioles**, therefore it is the state of **contraction and relaxation in the smooth muscle cells** of the arterioles which determines the distribution of blood to organs.

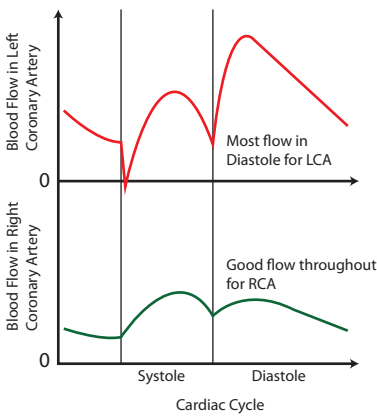
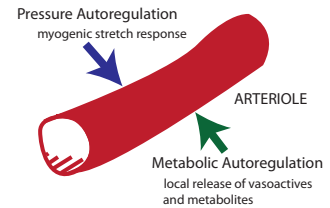
**Extrinsic control of blood flow** was covered in a previous summary. It is determined by the general formula flow = pressure/resistance. As such the factors which are important are the total peripheral resistance and cardiac output, coupled with the total circulating volume. The main extrinsic control of blood flow is therefore by **sympathetic control** (controlling vascular tone, HR and contractility), **parasympathetic control** (controlling heart rate and vasodilation) and **extrinsic hormonal control** (such as RAAS, ANP and ADH).



**Autoregulation** flow is locally controlled in certain vascular beds by a process known as autoregulation. This is defined as the ability of an organ to **maintain relatively constant blood flow in the face of variations in perfusion pressure**. Since flow = pressure/resistance, as the pressure changes the resistance also changes (usually via vessel relaxation or constriction) to maintain flow. The result is a characteristic autoregulatory curve seen adjacent. Outside the limits of autoregulation, flow is dependent on driving pressure. Autoregulation is seen in the **kidneys, brain and heart**. The skin and the lungs demonstrate minimal autoregulation.

**Intrinsic control of blood flow** Autoregulation of local blood flows is dependent on two mechanisms. **Pressure Autoregulation** refers to the **myogenic stretch response** that occurs in response to increases and decreases in pressure with vasoconstriction (in the setting of increased pressures to increase resistance and thus maintain stable flow) and vasodilation in low pressure settings.

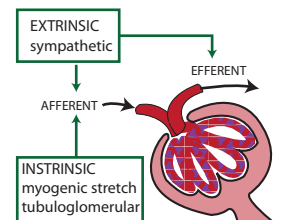
**Metabolic or Vasoactive Autoregulation** refers to the direct action of **locally derived metabolites and vasoactive substances** which cause local variations in flow according to requirements. This is not completely understood. Examples include vasodilation in the setting of increased buildup of  $CO_2$ ,  $H^+$  and **temperature** during exercise and the release of the potent vasoconstrictor **thromboxane  $A_2$**  by platelets to cause vasoconstriction in the setting of vessel damage.



**Coronary blood flow** At rest the heart receives approximately **200-250ml per minute of blood flow** which equates roughly to **5%** of the total CO. **Myocardial  $O_2$  consumption** is very high in the order of **8 mL  $O_2$  /min/100g** of tissue which is almost 20 times that of skeletal muscle. The main **determinants** of myocardial oxygen demand are **wall tension (30-40%), HR (15-25%), contractility (10-15%), basal metabolism (25%) and external work (10-15%)**. In order to achieve its high oxygen requirements the heart has two adaptations. Firstly it has a **very high density of capillaries** which enable extensive exchange opportunities. Secondly, myocardium **extracts up to 75% of  $O_2$**  compared to a whole body extraction of 25%. A result of this high extraction is that blood returned via the coronary sinus and thesian circulation has a markedly decreased  $PO_2$ . The coronary circulation has both parasympathetic and sympathetic innervation and does demonstrate local myogenic stretch control, however it is the **local metabolic control of flow that predominates** especially in exercise states. This is not well understood but postulated metabolites include NO, adenosine,  $H^+$ ,  $CO_2$  and reduced  $O_2$  tension. **Mechanical aspects** of the coronary blood flow are also of particular importance. Unlike most organs where driving pressure is the arterial pressure - venous pressure, in the heart the **external pressure** on the arteries is also considered. In this setting therefore, three pressures become important, during diastole it is the pressure at the aortic root (arterial) minus the pressure in the right atria (venous). In systole however the ventricle pressure becomes significant in a **starling resistor model**. It is now the pressure in the aortic root (arterial) minus the pressure in the ventricle wall (external pressure on the vessel). As a **result flow will often cease in early systole in the LCA**. The RCA maintains good flow due to the significantly lower ventricle wall pressures. See adjacent diagram.

**Cerebral blood flow** At rest the brain receives around **750ml of flow** which equates to approximately **15% of total CO**. It has a high myocardial consumption of **3-3.5 mL  $O_2$  /min/100g** which when calculated using the weight of the brain (1400g) results in around 50 ml of oxygen which is **20% total consumption** (remembering that total oxygen consumption is around 250ml/min). As always flow = pressure/resistance. In this setting it is the **cerebral blood flow (CBF) = cerebral perfusion pressure (CPP) / cerebrovascular resistance (CVR)**. The CPP is normally MAP - CVP, however in pathological states **where there is raised ICP a starling resistor model is set up**. In this setting ICP > CVP and therefore the CPP = MAP - ICP. Extrinsic nerve and hormonal control have little influence on CBF. The cerebral circulation demonstrates a very **well controlled autoregulation** normally within the range of **50-150mmHg**. It should be noted that in chronic hypertension this may be reset. The mechanism of autoregulation has not been fully elucidated, however it is believed to be primarily due to **myogenic stretch factors**. Local **metabolic factors** are significant also, but primarily from a **regional cerebral flow** perspective and may include adenosine, NO,  $H^+$ . The exceptions to this are arterial  $PCO_2$  which demonstrates an **almost linear relationship with CBF from 20mmHg-80mmHg**, and to a lesser extent  $O_2$  which when to oxygen content drops significantly (associated with a  $PaO_2$  of less than 50mmHg which is on the steep part of the dissociation curve) there is an increase in CBF. Above this point oxygen saturations have little influence.

**Renal blood flow.** At rest the kidneys receive approximately **1100 ml/min** of blood flow which equates to around **20-25% of blood flow**. The main resistors in the kidneys which modify the flow according to different pressures are the efferent and afferent arterioles. **Extrinsically** there is both neural and hormonal regulation. The kidneys have **extensive sympathetic innervation** and resistance is increased in response to carotid and aortic body stimulation via the medulla to increase resistance in the afferent and efferent arterioles and reduce flow. The **renin angiotensin aldosterone system** is also activated in low volume states to retain sodium and water which influences overall flow along with **ADH release**. **Intrinsically** the kidney demonstrates **autoregulation**, maintaining a renal blood flow within a systemic pressure range of **75-170 mmHg**. The intrinsic control of blood flow is mediated by **myogenic stretch mechanisms** and **tubuloglomerular feedback via afferent arteriole constriction**. Tubuloglomerular feedback involves the **macula densa** which releases **adenosine** if the renal perfusion pressure rises, and reduces production if the pressure falls. It may also release **NO** in response to a decreased perfusion pressure.



**Splanchnic (including hepatic) blood flow** the splanchnic system receives approximately **1250ml of the CO** which equates to approximately **25%**. Anatomically the blood is derived from the **coeliac, superior and inferior mesenteric arteries**. It is unique in that it is **partially in parallel** (incl gastric, spleen, pancreas, small intestine and colonic) and **partially in series**. The liver is the reason for this, as it receives **25% of its flow from the hepatic artery** and the **remainder from the portal system**. The hepatic system has the capacity to increase flow arterial or portal flow if the reciprocal is decreased. The **liver** maintains a tightly controlled **oxygen utilisation** due to very effective **variable extraction**, and uses approximately **50ml  $O_2$  per minute (20%)**. The splanchnic system is also an important reservoir with pooling occurring in the capacitance vessels of the mesentary, spleen and liver. Control of flow **extrinsically** is almost exclusively through **sympathetic activity**. Sympathetic activation results in increased venous constriction which increases the circulating blood volume, and increased resistance of the arterioles which diverts blood away from the digestive system to prioritised organs. Intrinsic blood flow control demonstrates **significantly less autoregulation** than other organs such as the kidney, brain and heart. **It is present however in the hepatic artery** in response to modulate the differences in pressures in the compared to the portal vein (100mmHg compared to 10mmHg) and to vary flow if portal return to decreased. **Metabolic control is important however in regional control of blood flow**, particularly in settings such as food ingestion and consumption with local release of hormones such as gastrin and cholecystokinin increasing flow to the digestive tract.