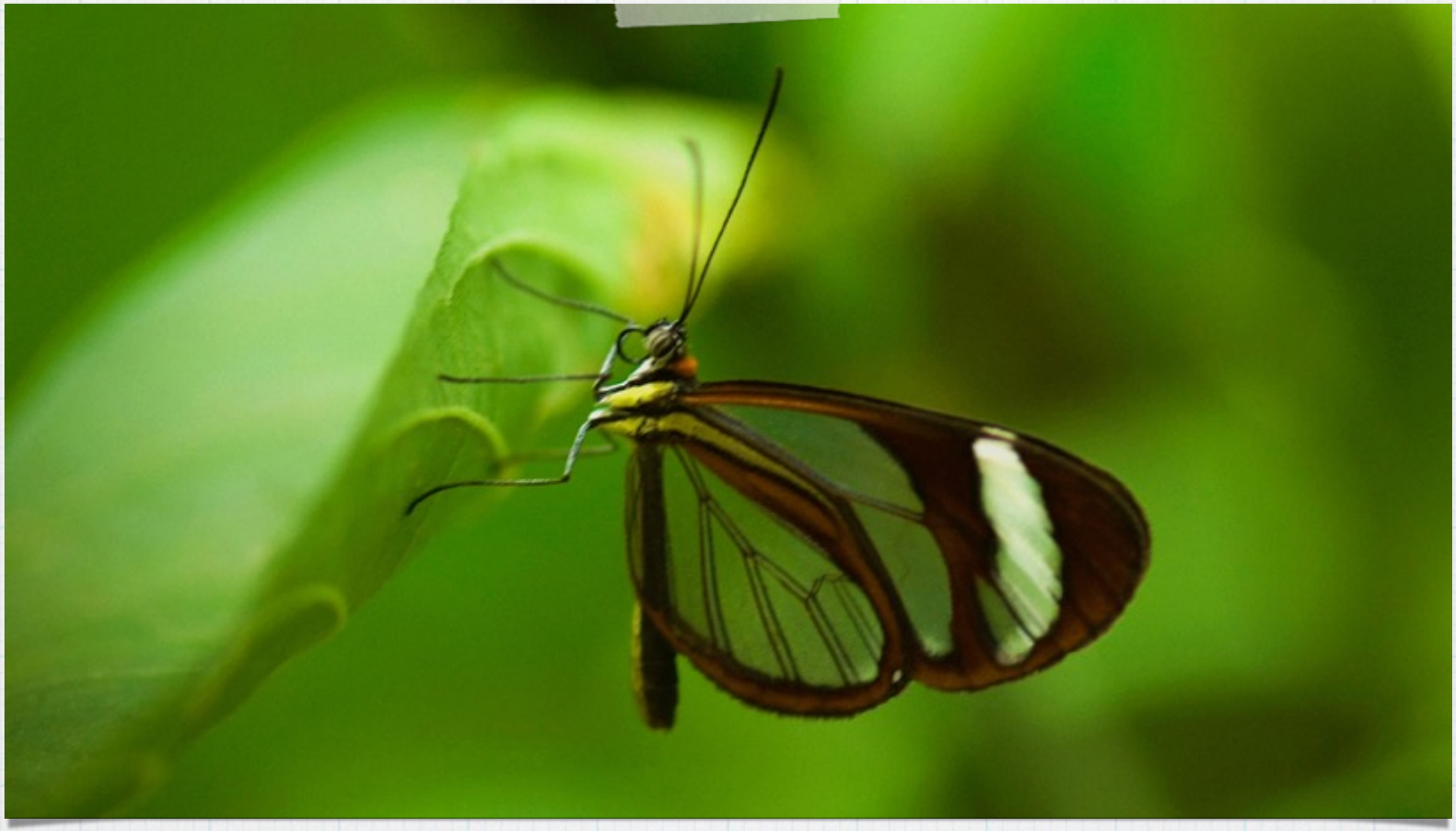

Physiology lecture 11

external effects on the circulatory system

The effects of gravity

* In the standing position - MAP at feet is 180 - 200mmHg, venous pressure is 90 mmHg
* Arterial pressure in the head is 60 - 75mmHg and the venous pressure is 0
* If an individual does not move in the upright position 300 - 500mL of blood would pool in the venous circulation - SV decreases, cerebral ischemia develops and LOC

The effects of gravity

* Major compensations on assuming upright posture
    * Triggered by a drop in BP in the carotid sinus and aortic arch
        * Baro-receptor —> HR increases —> increased CO
        * Relatively little vasoconstriction peripherally but higher circulation levels of renin and aldostrone
        * Arterioles constrict

Standing and the cerebral circulation

* Arterial pressure drops 20 - 40 mmHg
* JVP also drops which reduces venous pressure and reduces ICP
* This decreases overall CPP which increases the partial pressure of CO2 and decreases PO2 and the pH in the brain tissue
* This actively dilates the cerebral vessels
* Because of this blood flow only declines about 20%

Fainting and gravity

* In a way it is a homeostatic mechanism - falling to horizontal position restores VR, CO, and CPP
* The effects of gravity are also inversely proportional to our blood volume

Exercise and the circulation

* Muscle blood flow
    * Blood flow at rest is low (2 - 4ml/100g/min)
    * When a muscle contracts it compresses the vessels in it if it develops more than 10% of maximal tension
    * When it develops more than 70% of maximal tension then blood flow stops
    * During contractions - flow is increases about 30 fold
    * Local mechanisms are the dominant mechanisms in mediating this large increase in blood flow

Exercise and the circulation

* Muscle blood flow
    * Fall in O2, Rise in CO2, rise in K and metabolites and an increased temperature all mediate this rise in blood flow
    * Dilation of arterioles and pre capillary sphincters opens up many more capillaries and decreases diffusion distance
    * Dilation also increases cross sectional area and therefore the velocity of flow also decreases
    * Decreased pH and increased temperature cause the Hb O2 curve to shift to the right and increase the dissociation from blood
    * Concentrations of 2-3 DPG increases and this further decreases the affinity for O2

Effects of exercise

* Increases HR, SV, SBP and MAP
    * Venoconstriction —> increases pre load
    * Increased action of muscle pump —> increased pre load
    * Increased resp rate —> Increased thoracic pump, increased venous return
    * Blood flow = pressure difference/resistance
        * so during exercise SBP increases, increasing P difference and therefore increasing blood flow
    * Adrenaline (sympathetic tone) + lactic acid (vasoactive metabolite) —> increased HR and skeletal vasodilation and constriction of non vital organs
    * Skeletal muscle increases from 20% at rest to 80 - 85% during exercise

Some important numbers...

* SV rises during exercise up until 40% of VO2 max, rising from 80 - 120ml/beat
* HR increases until VO2 max is reached, from 70 —> 200bpm
* Cardiac output increases until VO2 max is reached, rising from approximately 5L/min to 25 - 30 L/min
* The AV oxygen extraction rises from approximately 4mL of O2 per 100mL of blood at rest to 18mL of Oxygen per 100mL of blood
* VO2 in the average person is 37 - 48 ml/kg/min
    * Cardiac output is a major determinant of VO2
    * Declines with HR as age

SHOCK

* DEFINITION: Inadequate tissue perfusion with relatively or absolutely inadequate cardiac output
* 4 distinct pathophysiologies
    * Amount of fluid in vascular system is insufficient
        * Hypovolemic shock - Hemorrhage, trauma, burns, vomitting/diarrhoea
    * Relatively inadequate amount of fluid
        * Distributive shock - anaphylaxis, sepsis
* Can be due to inadequate pumping action of the heart
    * Cardiogenic shock - AMI, CCF, Arrythmia
* Inadequate cardiac output due to obstruction
    * Obstructive shock - PE, Tamponade, Tension PTx
* Neurogenic shock

Shock

* Stages of shock
    * Compensated
        * Tachycardia
        * RAAS system activated —> Na and H20 retention —>
        * Vasopressin secreted —> thirst
        * Neurogenic vasoconstriction
* Progressive shock
    * When the initial CV insult is so large the normal compensatory mechanisms cannot cope —> either iatrogenic support or desent into vicious cycle of decreased CO and BP —> decreased myocardial and cerebral perfusion —> decreased contractility and neurological BP control —> vasodilation and venous pooling, hypoxia and acidosis eating to deem and clotting and decreased VR —> decreased CO
* Refractory shock