PHYSIO

lec 1

- major mechanism for changing flow is by changing resistance (mainly arterioles)
- capillaries 2500 cm^2
- smooth parabolic profile of velocity in laminar flow
- korotkoff sounds caused by turbulence
- reynold's no. to see if laminar or not
- ^ normally 200-400 in vasc. system
- rises to a couple of thousands at prox aorta and PA due to rapid ejection
- anemia has high rey no. due to decreased viscosity and increased V
- thrombi inc viscosity & inc rey no.
- conductance is the exact reciprocal of resistance
- diameter plays the greatest role of all factors in determining rate of flow
- TPR in parallel less than single vessels
- parallel: brain, kidney, GI, skin, muscles and coronary circulations
- amputation leads to reduced total flow and conductance and increases TPR

lec 2

- increase in arterial pressure initiates compensatory vascular resistance
- arterial pressure norm 70-175 mmHg
- local autoregulatory mechanisms override effects of vasoconstrictors
- critical closing pressure: no flow at all due to collapsed vessels
- inhibition of sympathetic activity increases flow by two-fold or more
- veins are most distensible vessels (8x more than arteries)
- compliance = distensibility x volume
- compliance of veins is 24x that of arteries
- sympathetic stimulation increases pressure at each volume (both A+V)
- sympathetic control of vascular capacitance is very important in hemorrhage
- delayed compliance makes it possible to accommodate extra blood e.g. large transfusion
- MAP is 60% diastole 40% systole
- at very high HR MAP is closer to systole
- factors that effects pulse pressure: stroke volume and compliance
- transmission of pressure pulse in arteries is when pressure overcomes inertia, sending wavefronts of distension along the aorta
- the greater the compliance, the slower the velocity
- in aorta transmission of pulse pressure is 15x velocity of blood flow
- damping of pressure pulses depends on resistance and compliance

lec 3

- kidneys and adrenal glands have highest BF
- acute control by vasodilation or constriction
- vasodilators: ADP, CO2, adenosine, H+
- cyclic opening/closing of precapillary sphincters is called vasomotion
- reactive hyperemia: sudden increase of BF after removal of a block
- metabolic theory: great arterial P that has excess flow which provides too much O2 and nutrients and washes out vasodilators, which causes constriction
- myogenic theory: stretch of BV causes wall to contract which causes reactive constriction, decreasing BF
- ^ theory is inherent to smooth muscles & can occur in absence of neural or hormonal influences
- metabolic factors override myogenic mechanisms
- brain is dependent on CO2 & H+ conc.
- NO in angina and ED
- endothelin stimulated by damage to endothelium like HTN or trauma
- retrolental fibroplasia in premature babies who were in oxygen tents
- steroid hormones stop BV growth
- collateral BV formed after thrombosis of one of the coronary arteries
- small BV: inward eutrophic remodelling. no change in cross sectional area of vascular wall
- large arteries: hypertrophic remodelling + increase CSA
- in dialysis; renal artery has outward remodelling and increase CSA whereas venous side has outward hypertrophic remodelling
- NE is a more powerful vasoconstrictor
- angiotensin II constricts small arterioles to regulate arterial pressure
- vasopressin is more powerful than A-II. also called ADH
- bradykinin: arteriolar dilation + inc capillary permeability. regulates blood in inflamed tissues
- Mg causes powerful vasodilation
- H+ dilate arterioles
- anions (citrate and acetate) cause mild vasodilation

lec 4

- baroreceptor reflex restores Pa to its set point in a matter of seconds
- info from aortic arch baroreceptors are carried on vagus nerve
- vasoconstrictor sympathetic effect powerful in kidneys, spleen, intestines, and skin
- brain stem info integrated in nucleus tractus solitarius
- CV centres are tonically active
- C1 in anterolateral portion of upper medulla (vasoconstriction on arterioles, veins, venules)
- vasomotor tone: partial state of constriction
- vasodilator centre in anterolateral portion of lower half of medulla
- sensory area bilateral in nucleus tra... in posterolateral medulla and lower pons
- chemoreceptors for O2 found in carotid bodies near bifurcation of carotid A and in aortic bodies. sensitive to decrease PO2
- cushing reaction: increased intracranial pressure which decreases perfusion of brain, so medullary chemoreceptors direct outflow to increase TPR and dramatically increase BP

lec 5

- atria and PA have low-pressure baroreceptors
- bainbridge reflex: increase in HR due to increased pressure at atria
- RAAS activates when BP is low
- losartan is AT1 receptor blocker
- angiotensin II stimulates Na/H exchange in renal prox tubule
- A-II can act directly on kidney and arterioles
- gases are highly lipid soluble so they diffuse through endothelial cells of capillaries
- water soluble substances go between clefts of endothelial cells
- fenestrated capillaries in kidneys and intestines for protein passage
- filtration: net fluid movement out of capillary into interstitial fluid. opposite is absorption
- capillary hydrostatic pressure (Pc) is a force favouring filtration
- Pc is closer to arterial pressure than to venous pressure
- Pc declines along length of glomerular capillaries bc of filtration
- interstitial hydrostatic pressure (Pi) opposes filtration. normally it's 0 or slightly negative
- interstitial oncotic pressure favours filtration
- capillary oncotic pressure opposes filtration. determined by protein conc. of capillary blood
- magnitude of fluid movement is determined by hydraulic conductance (Kf) across capillary wall
- glomerular capillaries have highest Kf. cerebral are lowest
- Kf increased in capillary injury

lec 6

- inadequate BF = circulatory shock = inadequate CO
- circulatory shock that results from diminished cardiac pumping ability is called cardiogenic shock
- in excessive metabolic rates, normal CO is inadequate so it can result in circulatory shock
- arterial pressure is the principal measure of adequacy of circulatory function. however, it can also be misleading
- progressive stage without therapy is fatal
- haemorrhage is most common cause of hypovolemic shock
- CO and arterial pressure fall to 0 when 40-45% of total blood volume is lost
- in hemorrhage, arterial pressure is maintained better than CO due to sympathetic reflexes
- last-ditch stand is CNS ischemic response (second plateau, extreme stimulation of sympathetic reflexes)
- sympathetic stimulations does not cause significant constriction of cerebral or cardiac vessels
- angiotensin, ADH and reverse stress-relaxation require 10-60 mins to respond completely after haemorrhage
- progressive shock has positive feedback cycle
- vasomotor centre does not fail if arterial pressure remains above 30 mmHg
- capillary hypoxia does not cause capillary permeability until the late stages of prolonged shock
- endotoxin (released from G-ve bacteria in intestines) causes cardiac depression and septic shock
- liver mainly affected in progressive shock, then lungs and heart
- severe plasma loss: intestinal obstruction and severe burns
- shock can result without haemorrhage by extensive contusion
- loss of vasomotor tone results in massive dilation (neurogenic shock)
- septic shock is most frequent cause of shock related deaths in hospitals
- dextran solution to manage shock when plasma is unavailable
- sympathomimetic drugs are beneficial for neurogenic and anaphylactic shock
- head-down position promotes venous return, thereby increasing cardiac output
- cardiopulmonary resuscitation may restore normal cardiac rhythm after complete cardiac arrest