Peripheral circulation II: Organ vascular beds

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Lecture slides

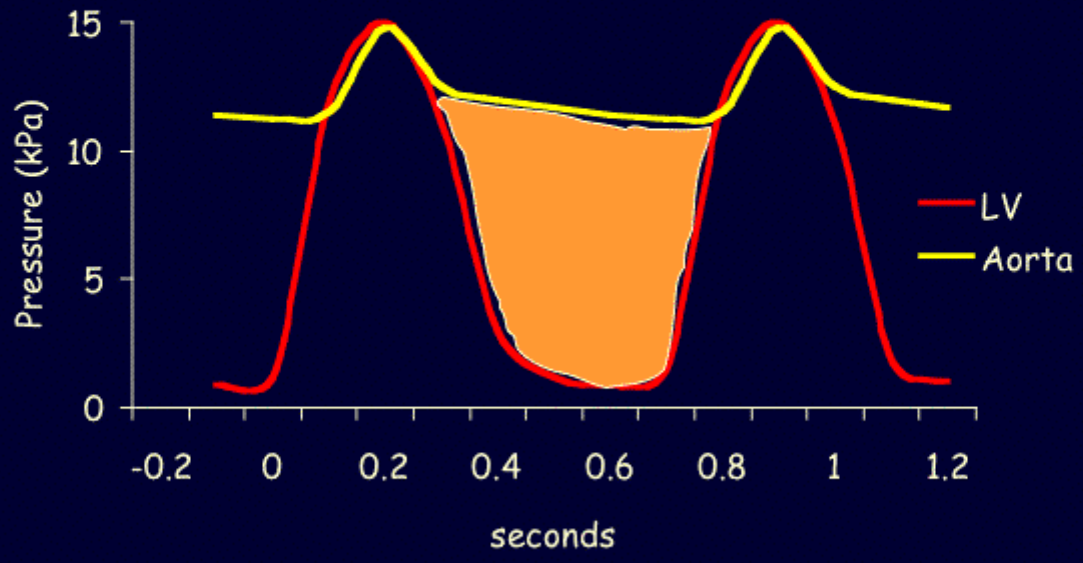
Coronary circulation

- R > L coronary artery:
  - ~ 50 % humans
- L > R coronary artery:
  - ~ 20 % humans
- R = L coronary artery:
  - ~ 30 % humans

Factors affecting coronary blood flow

- Aortic pressure
- Coronary arteriolar resistance ← myocardial metabolic activity (autoregulation)
- Extravascular compression
  - importance L > R (lower RV pressures)
  - maximal L flow in early diastole

Coronary flow "window of opportunity"



Counterpulsation

- Balloon in thoracic aorta
- Inflated during diastole
  - ↑ coronary pressure when resistance ↓
- Deflated in systole

♥ rate & coronary flow

- Changes in heart rate mainly by changes in the length of diastole
- Tachycardia:
  - ↑ proportion of time spent in systole

    vs.
  - ↑ metabolic needs (→ vasodilation)

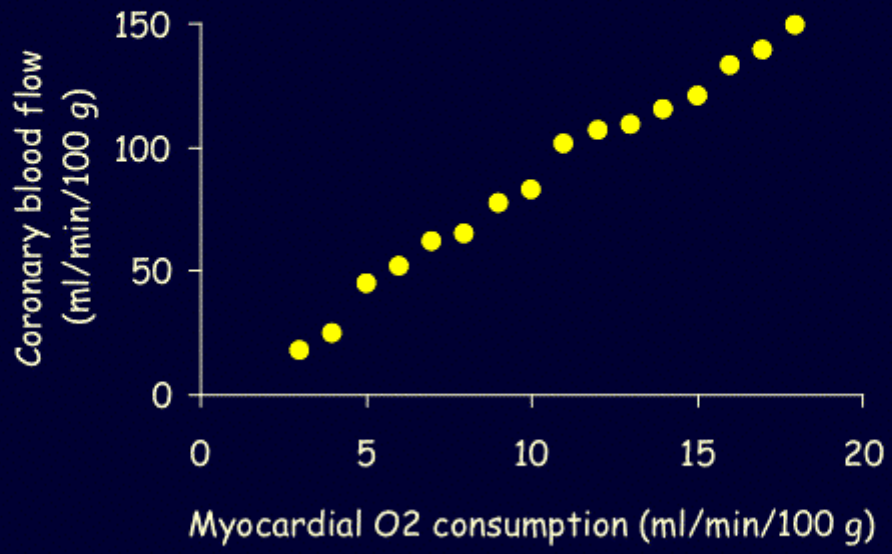
SNS stimulation

- Receptors:
  - α (vasoconstrictor)
  - β (vasodilator)
- Direct SNS effect: α vasoconstriction
- Overcome by vasodilation due to ↑ metabolic activity (local regulation dominant)

Vagus stimulation

- Slight vasodilation
  (local regulation still dominant)

Local metabolic regulation



O_2 supply to ♥ is flow limited

- Most O_2 extracted from coronaries during a single passage (O_2 extraction near maximal)
- Therefore: ↓ flow → ↓ O_2 delivery

Reduced coronary flow

- Not too strong and/or severe (e.g. quickly repaired coronary occlusion)
  → myocardial stunning (tempor. ↓ contractility)
  - Ca^{2+} overload in ischemia (pump dysfunction)
  - ROS in reperfusion
- Strong and/or severe
  → myocardial infarction (necrosis)
  - both mechanical & electrical impairment
  - O2 & substarte depletion, metabolite accumulation

Collaterals

- Not in normal human ♥
- Can develop with slow narrowing
- Artificial:
  - coronary bypass graft (e.g. from internal mammary artery)
  - coronary angioplasty (distention of occluded site with balloon catheter)

Tx of angina pectoris

- Coronary vasodilators (e.g. NO_2^-/NO_3^-)
- But:
  - arteries in ischemic regions already fully dilated by metabolic mechanism
  - vasodilation in OK areas can ↓ driving pressure in ischemic areas: CORONARY STEAL
- Angina pectoris treated only if coronary steal < pressure work reduction (↓ PVR, ↓ venous return due to peripheral arterio- & venodilation)

Cutaneous circulation

- Low metabolism
- Blood flow serves mainly thermoregulatory role

Numerous AV anastomoses

- Thick muscular layer
- Rich nerve supply
- No basal tone (can constrict maximally)
- No metabolic autoregulation
- Exclusively under SNS control (can close completely) → thermoregulation

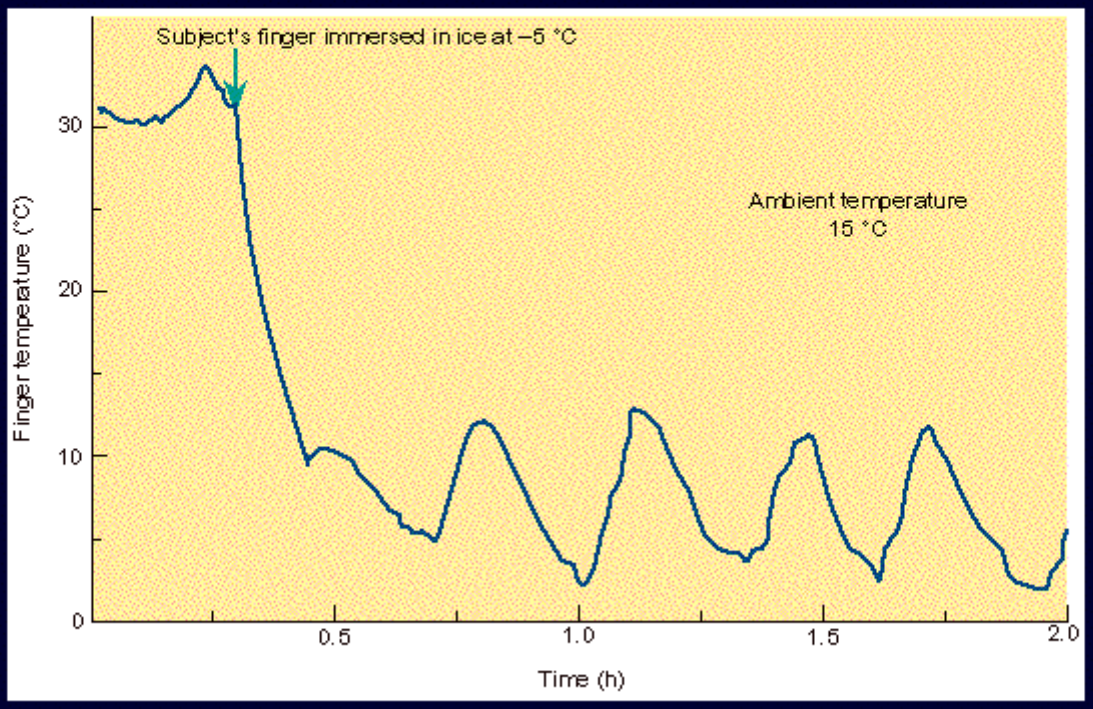
Other skin arterioles

- Some basal tone
- Dual control:
  - **SNS** (more important)
    - adrenaline & NA only vasoconstriction
    - but sweat gland activation (controlled by ACh SNS terminals) → skin vasodilation (bradykinin released from tissue fluid proteins by enzyme in sweat)
  - local metabolic & myogenic autoregulation
  - no parasympathetic nerves

Neural regulation

- Higher brain centers → SNS:
  - blushing in embarrassment or anger
  - blanching in fear

"Hunting phenomenon"



Raynaud's disease

- Vasoconstrictor hypersensitivity of fingers (& sometimes toes) to cold
- Finger ischemia in cold (tingling, numbness, pain)
- Blanching (no blood) → cyanosis (ischemia) → redness (spasm subsides)

Rosy color in cold

- Cold vasodilation (relieving cold vasoconstriction)
- Reduced O_2 uptake of cold skin (high Hb saturation)
- Cold-induced shift to the left of the Hb dissociation curve

Countercurrent heat exchange

- Major skin arteries run alongside veins
- In cold: heat taken up from arteries by cooled blood in veins before it reaches surface → heat conservation
- In heat: heat taken up by blood in the surface is given from veins to colder arterial blood → heat not taken up
  (+ ↑ temperature difference from skin to environment → heat dissipation)

Skeletal muscle circulation

- Blood flow (& capillary density):
  - red (slow-twitch, high oxidative) > white
  - exercise: ↑ up to 15-20x
    - reduction of resting asynchronous intermittent contractions of precapillary sphincters
    - mechanical squeezing by muscle contractions
      - no problem if intermittent
      - can limit tonic contraction

Regulation

- SNS (important because the largest vascular bed → great effect on total PVR)
  - prevails at rest
  - NA only vasoconstriction
  - adrenaline vasodilation @ low doses, vasoconstriction @ high
  - some ACh SNS vasodilation
- Local (important because of high metabolism)
  - prevails during exercise
  - high basal tone (→ large vasodilation)

Cerebral circulation

Unique features:

- contained within rigid structure → inflow/outflow dysbalances ↑ pressure
  - Cushing's phenomenon: ↑ systemic BP with ↑ intracranial pressure (e.g. tumor) - by ischemic stimulation of vasopressor center in medulla (helps maintain brain flow)
- Absolute requirement for adequate flow
  - least tolerant to ischemia
    - 5 sec ischemia → loss of consciousness
  - glucose-dependent
  - no contribution to total PVR regulation

Neural regulation of brain vessels

- Minimal importance (local mechanisms predominate)
- SNS (along carotid & vertebral arteries) - weak vasoconstriction
- Parasympathetic fibers from facial nerve - weak vasodilation

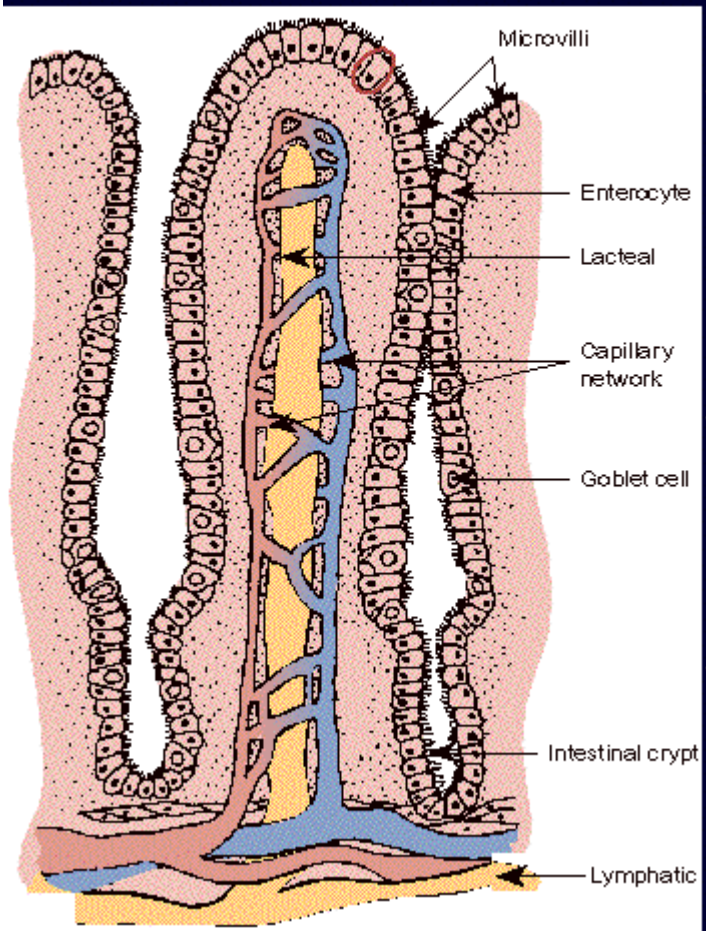
Local regulation of brain vessels

- Hypoxia
- Very sensitive to CO_2 (vasodilation via changed pH)
- H^+ cannot cross blood-brain barrier
  → cerebral vasodilation by:
  - local CO_2/pH changes
  - blood CO_2
  - not blood pH (if = CO_2)

Brain flow autoregulation

- Excellent between 60 and 160 mmHg
- Below 60 mmHg: syncope
- Above 160 mmHg: cerebral edema

Intestinal circulation

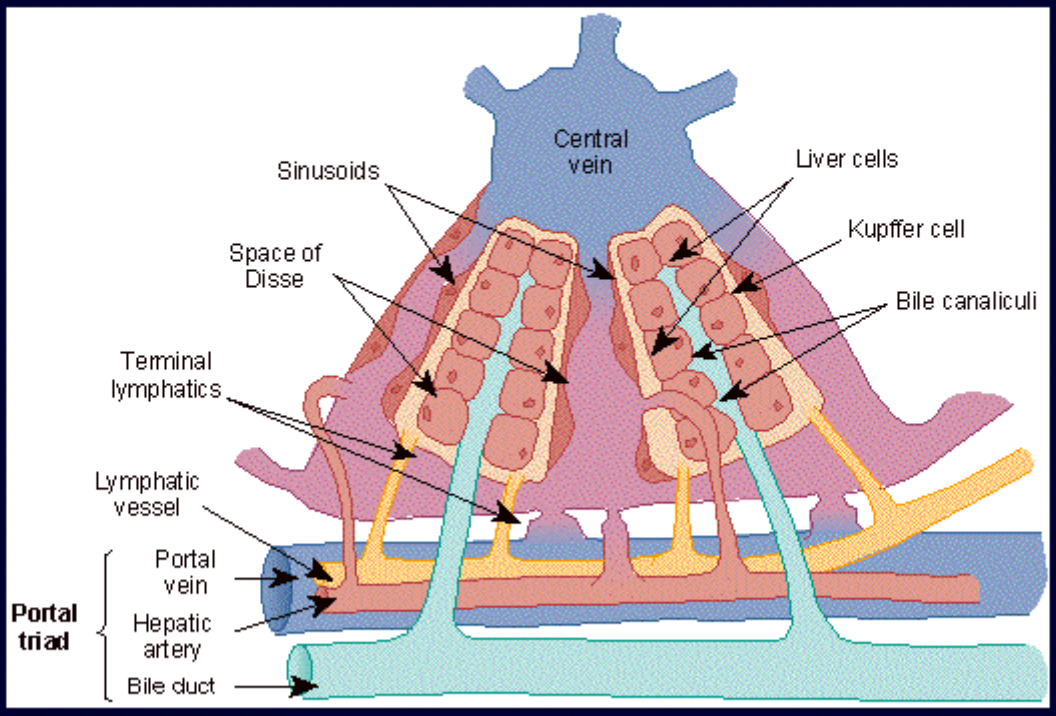


- Countercurrent O_2 exchange (shortcut for O_2 from arteries to veins)
- Flow needed to carry away nutrients, not to bring O_2
- Necrosis of villi with severe flow reduction (→ bacteremia)

GIT flow regulation

- Neural:
  - prominent (blood shunted away when needed elsewhere)
  - SNS (NE, α vasoconstriction; β receptors much less expressed)
- Autoregulation less developed
  - functional hyperemia (after meal)
  - gastrin & cholecystokinin ↑ GIT blood flow
  - vasodilation by digestion products (glucose, FFA)

Hepatic vasculature



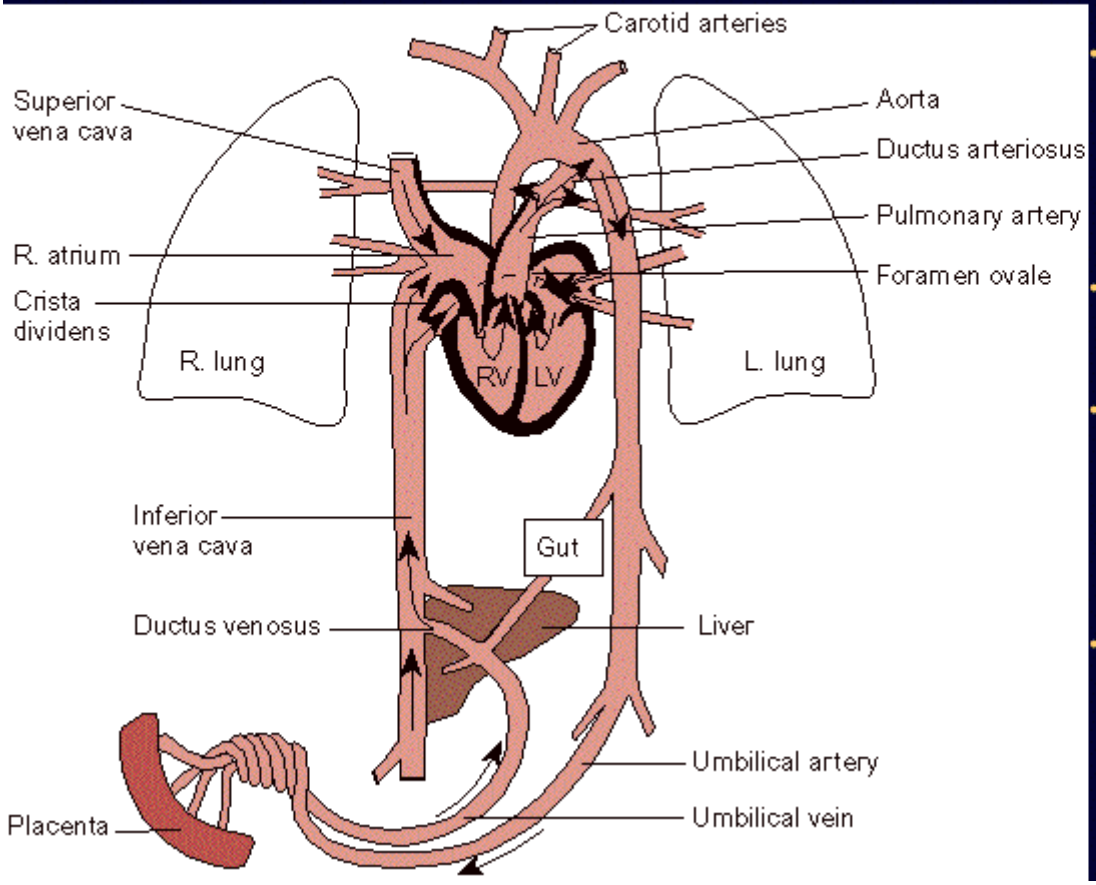
Hepatic circulation

- Hepatic blood flow ~ 25% CO
  - 3/4of that via portal vein
    - little O_2
    - mean pressure ~10 mmHg → small driving pressure gradient
      - increases in hepatic (and central) venous pressure easily transmit upstream → liver edema → transudation to peritoneal cavity (ascites)
        (also with ↑ portal resistance due to fibrosis in cirrhosis)

Regulation of liver circulation

- Autoregulation
  - not in portal system
  - hepatic arterioles autoregulate
- SNS
  - constriction of resistance vessels in portal venous & hepatic arterial systems
  - constriction of capacitance vessels more important (blood reservoir)
    - liver contains ~ 15% of all blood
    - 50% of that can be rapidly expelled by SNS

Fetal circulation



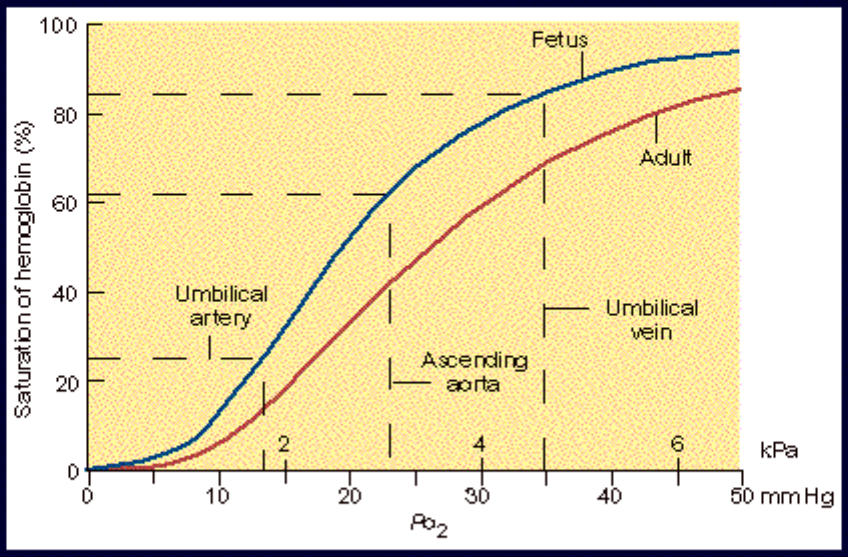
- ~50% placenta flow returns via ductus venosus
- rest through liver
- separate streams in IVC (crista dividens)
- stream from d.v. → foramen ovale → LV → carotids

High fetal pulmonary vascular resistance

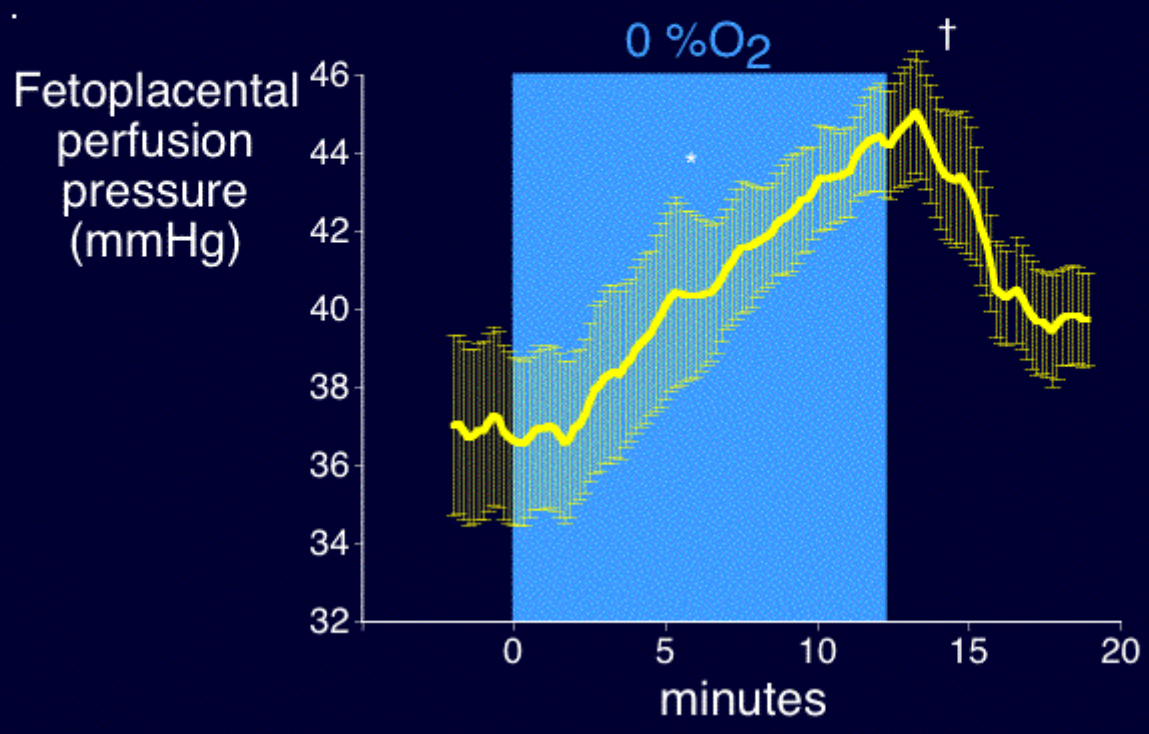
- Low O_2 → hypoxic vasoconstriction
- No ventilation → undistended, convoluted vessels

- Shunts ~90% of *CO* through ductus arteriosus (enters aorta distal to origin of carotid arteries)

Fetal Hb helps O_2 transfer in placenta



Hypoxic fetoplacental vasoconstriction



Birth

- Umbilical vessels closed by trauma (if not tied)
- Ductus venosus closes (mech. ??)
- ↑ CO_2 → breathing
- ↑ arterial pO_2 constricts ductus arteriosus (via ↓ vasodil. PGs, Bk; also K channels)