Goldman Equation:

\[ V_m = 58 \log \frac{p_K[K]_o + p_{Na}[Na]_o + p_{Cl}[Cl]_i}{p_K[K]_i + p_{Na}[Na]_i + p_{Cl}[Cl]_o} \]

Membrane permeable to
- \( K \) \((pK = 1.0) - \) membrane most permeable to \( K \) at rest
- \( Na \) \((pNa = 0.04)\)
- \( Cl \) \((pCl = 0.45)\)

Ion channels may be open at resting membrane potential and make the membrane permeable to ions.

The \( Na^+/K^+ \) pump contributes to the resting membrane potential:
- **Indirectly** by maintaining unequal concentrations of \( Na^+ \) and \( K^+ \) across the cell membrane.
- **Directly**, because it is electrogenic. It pumps 3 x \( Na^+ \) out for 2 x \( K^+ \) in.

The indirect contribution is more significant and important than the direct pumping
Ionic Basis of Action Potentials
Neuromuscular Junction:

The neuromuscular junction is the synapse between the axon terminal of a motor neuron and the membrane of a muscle fibre.

Mammalian skeletal muscles are twitch muscles. Each muscle fibre is innervated by a single motor axon.

A single presynaptic AP results in a single postsynaptic AP. This is different to the CNS.

Epp = end plate potential.
It is a special kind of EPSP.

Neuromuscular transmission is mediated by release of ACh from motor axons.

ACh activates nicotinic ACh-receptors (ligand-gated cation channels) on the postsynaptic muscle fibre membrane.

Activation of nicotinic receptors causes depolarisation of the muscle fibre membrane (“end-plate potential”), which triggers a muscle AP. This is excitation-contraction coupling.
Mechanisms of Contraction in Skeletal Muscle
Circulatory Systems:

Most invertebrates have open circulations.
Vertebrates and cephalopods have closed circulations.

Closed Circulation
• Blood entirely contained within system of vessels – does not bathe tissues directly.
• Blood carried to the depths of each tissue by discrete vessels.
• Blood remains in discrete vessels as it passes through each tissue.
• Blood returns to the heart from each tissue by way of discrete vessels.

Open Circulation
• Blood discharged from heart into discrete vessels, but then exits vessels to bathe tissues directly.
• Blood flows through lacunae (small spaces between tissue cells) and sinuses (larger spaces between cells).
• Blood returns to the heart via tissue spaces (sinuses) rather than well-defined veins.

Open system: ↓ pressure, ↑ volume (blood continuous with extracellular fluid)
Closed system: ↑ pressure, ↓ volume (blood separate)

Closed circulation permits rapid adjustments of the circulation in response to tissue demands, and can sustain high metabolic rates.
Blood Vessels:

Veins have larger lumens and thicker walls than arteries due to more pressure. More tunica media (muscle) in arteries than veins. All blood vessels are lined with a single layer of epithelial cells, called endothelium.

Tissue Perfusion:

Tissue perfusion maintains adequate blood flow through the capillaries.

\[
\text{MABP} \approx \text{CO} \times \text{TPR}
\]

\[
\text{MABP} = \text{mean arterial blood pressure}
\]

\[
\text{CO} = \text{cardiac output}
\]

\[
\text{TPR} = \text{total peripheral resistance}
\]

Arterial pressure is maximum at systole, minimum at diastole. (MABP \approx \frac{2}{3} \text{ diastolic} + \frac{1}{3} \text{ systolic}). Pressure decreases from arterial to venous end of the system.

TPR depends on viscosity (haematocrit) and vascular resistance. Vascular resistance increases with increased length of vessel and decreased diameter. Significant changes in TPR can be achieved by small changes in vessel diameter. \(\downarrow\) diameter = \(\uparrow\) resistance = \(\downarrow\) flow rate (cm/s) – so maximum exchange is possible.

MABP must not be allowed to vary too much, so TPR can compensate for changes in CO. Vasomotor tone = constant resting level of muscle contraction due to nervous stimulation.
The Baroreceptor Reflex:

This is a negative feedback reflex. Arterial baroreceptors are stretch receptors found in areas where blood pressure is highest (carotid sinus, aortic arch).

Blood being forced out of the aorta causes a slight stretch. Increased pressure $\rightarrow$ increased stretch $\rightarrow$ increased baroreceptor activity.

Reflex works on a short-term basis. It stabilises to the MAP.

Increased firing $\rightarrow$ $\downarrow$ sympathetic output $= \downarrow$ norepinephrine release.

This causes:

- Vasodilation of arteriolar smooth muscle $\rightarrow$ decreased peripheral resistance.
- Decreased force of contraction $\rightarrow$ decreased cardiac output.
- Decreased heart rate (via SA node) $\rightarrow$ decreased cardiac output.

All result in decreased MAP.
Gas Transport
Ion Regulation in the Kidney