L9 Neural Regulation of Blood Vessels

All blood vessels have symp → constriction
Descending symp nerves have tonic excitatory activity = blood vessels are always slightly constricted (decreased activity → dilation)

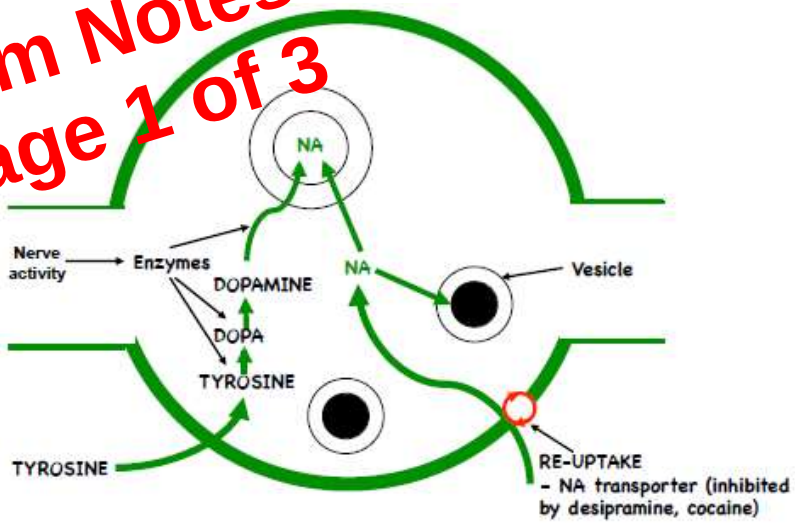
- Autonomic nerves use co-transmitters
- Sympathetic vasoconstrictor: NAd, ATP + NPY (neuro peptide Y)
- Sympathetic dilator:
    - ACh, + vasoactive intestinal peptide (VIP) + NO
        - Skin in some regions
- Parasympathetic cholinergic: ACh + VIP + NO
    - Cerebral, coronary, reproductive tissue

Arrangement of sympathetic nerve fibres
- Paravascular nerve bundles send out branches to form perivascular network with varicosities
- Perivascular nerve fibres are in adventitial-media border; don't enter the VSM
- Effect of nerve activity on VSM must be conducted from cell to cell

Varicosity
- Granular vesicles contain NA, ATP
- Large opaque vesicle contain NPY
- Sites of release of transmitters
- Release is "en passant"
    - When action potential passes through varicosity as it travels through along nerve fibres
    - Increased Ca^{2+} leads to release a bit of NAd

Biosynthesis of NAd
- Mainly made in varicosity
- Tyrosine from the blood stream
- Usually re-uptake to stop its effect

Release of NAd + termination of action of NAd
- Act mostly on α_1 receptors (sometimes on α_2)
- Have pre-synaptic α_2 receptors – helps control the release of NAd
- NAd can:
    - Diffuse in to blood stream
    - RE-UPTAKE



α_1 receptors all along proximal arterioles (the bigger ones)

α_2 receptors on distal arterioles – their constrictor influence is more easily blunted by local dilator influences (metabolites)

NAd binds to α_1:
1. Can activate PLC and produce IP_3 → binds to receptors on sarcoplasmic reticulum → Ca^{2+} released → increased $[Ca^{2+}]$→ contraction of VSM
2. Open voltage gated channels on VSM → depolarisation → sarcoplasmic reticulum release Ca^{2+}

NAd binds to α_2:
1. Closes K^+ channels → depolarisation → more Ca^{2+} influx through voltage gated channels