

Autonomic nervous system

Seminar handout · Official questions 1-6 · Organisation and anatomy

Core idea

- **ANS:** largely involuntary control of **smooth muscle, cardiac muscle and glands**; also regulates metabolism.
- **Homeostasis:** keeping internal conditions within appropriate limits.
- **Sympathetic:** adjusts circulation, energy use and stress responses; active at rest too.
- **Parasympathetic:** supports digestion, secretion, voiding and cardiac slowing.
- **Enteric:** local sensory, integrating and motor circuits in the gut wall.
- **CNS (central nervous system) control:** hypothalamus, brainstem and spinal circuits; influenced by higher brain centres and emotions.

Two neurons connect CNS to organ

CNS → preganglionic neuron → **ganglion** → postganglionic neuron → **organ**

- **Preganglionic:** cell body in CNS; axon travels to ganglion; usually lightly myelinated.
- **Postganglionic:** cell body in ganglion; axon reaches organ; usually unmyelinated.
- **Ganglion:** neuronal cell-body cluster outside CNS. **Nucleus:** such a cluster inside CNS.
- **Somatic motor pathway:** one neuron from CNS to skeletal muscle; no autonomic ganglion.

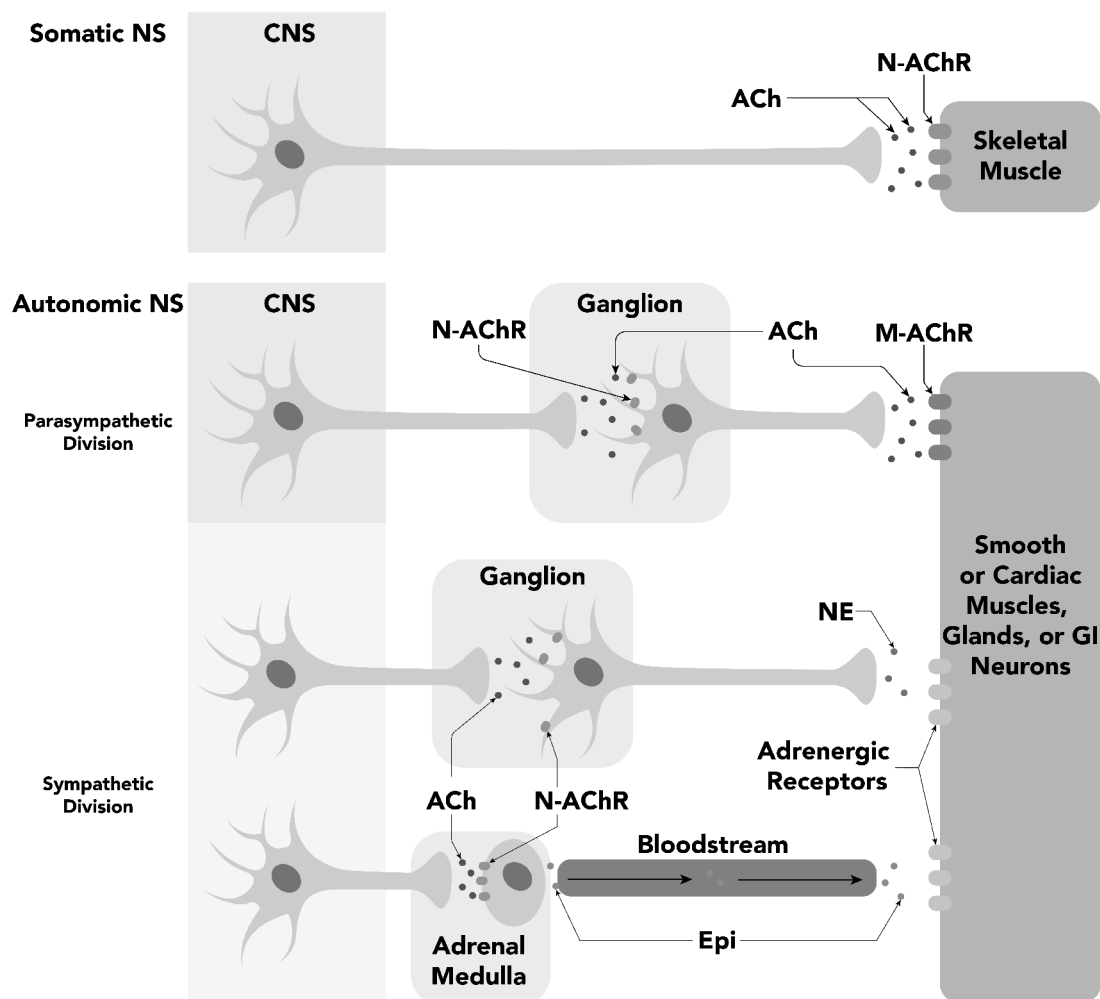
Feature	Sympathetic	Parasympathetic
Origin	Thoracolumbar: T1-L2 spinal cord; lateral-horn region.	Craniosacral: brainstem and S2-S4 spinal cord.
Ganglia	Paravertebral: chains beside spine. Prevertebral: near major abdominal arteries.	Near organ, or intramural: within its wall.
Fibre lengths	Usually short preganglionic; long postganglionic.	Usually long preganglionic; short postganglionic.
Distribution	Viscera, most vessels, sweat glands and skin.	Cranial nerves III, VII, IX, X; pelvic splanchnic nerves S2-S4.

Origins and routes to remember

- **Sympathetic chain:** fibres may synapse at entry, ascend/descend, or pass through to a prevertebral ganglion.
- **III:** pupil / near focusing. **VII:** tears, submandibular and sublingual saliva. **IX:** parotid saliva.
- **X, vagus:** much of thoracic and abdominal viscera. **S2-S4:** distal bowel and pelvic organs.
- **Fibre length follows ganglion position.** Ganglion near organ = long first axon.
- **Neural versus hormonal:** nerves usually act rapidly and locally; circulating hormones reach many tissues. Responses still require the right receptor.

Autonomic pathways

Practise the labelled chains · The course slides explicitly include an ANS summary drawing



Daniel Walsh & Alan Sved (2019) · CC BY-SA 4.0 · Greyscale print rendering; labels unchanged.

Figure key: ACh = acetylcholine; NE = NA = noradrenaline; Epi = adrenaline.

N-AChR = nicotinic receptor (Nn in ganglia; Nm in muscle); M-AChR = muscarinic receptor; GI = gastrointestinal.

Draw and label	Essential sequence
Parasympathetic	CNS → ACh / Nn ganglion → ACh / muscarinic organ.
Usual sympathetic	CNS → ACh / Nn ganglion → NA / α or β organ.
Sweat exception	Sympathetic chain, but final transmitter is ACh ; eccrine gland receptor M3 . Add this branch yourself.
Adrenal medulla	ACh / Nn → chromaffin cell → adrenaline + NA in blood . No ordinary postganglionic axon; figure emphasises adrenaline.
Somatic comparison	One neuron → ACh / Nm → skeletal muscle.

- **Label:** CNS boundary, ganglion, pre/postganglionic fibres, transmitter, receptor and target.
- **Cholinergic** = uses/responds to ACh. **Adrenergic** = responds to adrenaline/NA. These terms do not identify a branch by themselves.

Which receptor does what?

Official topics 2.2 and 3.2 · Know receptor, location and effect together

Receptor	Mechanism	Key location / response
Nn	Cation channel	Both autonomic ganglia; adrenal chromaffin cells. Depolarisation / excitation.
Nm	Cation channel	Skeletal-muscle end plate. Excitation → contraction.
M2	Gi; K ⁺ channels also open	Heart: slower SA-node firing and AV conduction ; reduced atrial contractility.
M3	Gq	Gland secretion; smooth-muscle contraction in bronchi, gut, bladder and eye. Endothelium: NO release → adjacent muscle relaxes.
α1	Gq	Vessels, radial iris, bladder outlet: smooth-muscle contraction.
α2	Gi	Presynaptic terminals: less NA release. CNS: reduced sympathetic outflow. Pancreatic β cells: less insulin secretion.
β1	Gs	Heart: faster rate/conduction, stronger contraction. Kidney juxtaglomerular cells: renin release.
β2	Gs	Bronchial and selected vascular smooth muscle: relaxation. Circulating adrenaline is important.
β3	Gs	Bladder detrusor: relaxation for storage. Adipose tissue: fat mobilisation / thermogenic signalling.

How to explain the mechanism

- **Nicotinic:** ACh directly opens an ion channel → inward net positive charge → depolarisation.
- **Muscarinic / adrenergic:** G-protein-coupled receptors (GPCRs); activate intracellular signalling rather than a channel within the receptor.
- **Gs:** adenylyl cyclase activated → **cAMP increases.**
- **Gi:** adenylyl cyclase inhibited → **cAMP decreases.** Other G-protein actions can alter ion channels.
- **Gq:** phospholipase C → **IP3 + DAG.** IP3 releases stored Ca²⁺; DAG helps activate protein kinase C.
- **Second messenger:** intracellular signal, e.g. cAMP. **Kinase:** enzyme adding phosphate groups to proteins.
- **Muscarinic families:** M1/M3/M5 use Gq; M2/M4 use Gi. For organ effects, prioritise M2 and M3.

Why one transmitter can have opposite effects

- **ACh:** Nn excites ganglion cells; M2 slows the heart. Different receptors.
- **cAMP:** promotes cardiac activity but favours smooth-muscle relaxation. Different cellular machinery.
- **α2:** inhibiting transmitter release is not the same as directly relaxing the target organ.

Answer pattern: receptor → signalling → target tissue → response. Never infer the effect from the transmitter name alone.

Sympathetic versus parasympathetic

Official topics 2.3 and 3.3 · Compare actions, not just branch names

Target	Sympathetic / catecholamines	Parasympathetic
Heart	β1 : faster rate/conduction; stronger contraction.	M2 : slower rate/conduction; strongest direct effects in nodes/atria, weaker in ventricles.
Pupil	α1 : radial muscle contracts → dilation (mydriasis).	M3 : circular sphincter contracts → constriction (miosis).
Lens	Not the main active mechanism for near focusing.	M3 : ciliary muscle contracts → lens rounds for near vision.
Airways	β2 : bronchodilation; mainly circulating adrenaline.	M3 : bronchoconstriction; more gland secretion.
Vessels	α1 : constriction. Less sympathetic tone permits dilation. Adrenaline can dilate selected beds via β2 .	Most systemic vessels lack substantial direct parasympathetic supply.
Gut	Generally less motility/secretion; tighter sphincters; less blood flow.	Generally more motility/secretion via ENS; coordinated sphincter relaxation.
Saliva / tears	Can stimulate relatively protein-rich saliva.	Copious watery saliva; tear secretion.
Bladder	β3 : detrusor relaxes. α1 : outlet contracts → storage.	M3 : detrusor contracts; coordinated outlet relaxation → voiding.
Skin / sweat	Eccrine sweating: ACh / M3 . Arrector pili: α1 → goosebumps.	No opposing parasympathetic supply to eccrine sweat glands.
Fuel / kidney	Mobilises glucose/fat; can suppress insulin. β1 : renin release.	Supports digestion; vagal input can increase pancreatic enzyme and insulin secretion.
Sexual function	Emission/ejaculation, with somatic muscle activity.	Erection through local NO -dependent vasodilation.

Eye: explain rather than memorise

- **Both iris muscles contract**: radial fibres pull open; circular fibres squeeze closed.
- **Near accommodation**: ciliary contraction → zonules loosen → lens rounds → stronger refraction. Different from changing pupil size.

Bladder: wall versus outlet

- **Storage**: relaxed wall + closed outlet. **Voiding**: contracted wall + relaxed outlet.
- **External urethral sphincter**: skeletal muscle, somatic pudendal nerve; must relax during voiding.
- **Do not equate receptors with innervation**: human airway **β2** effects do not require dense direct sympathetic supply.
- **Endothelial M3** can release NO; this does not mean every vessel has parasympathetic innervation.

Hormonal output and blockers

Official topics 2.4, 2.2 and 3.2 · Mechanisms, not treatment instructions

Adrenal medulla = modified sympathetic ganglion

- **Chromaffin cells:** neural-crest-derived, modified postganglionic cells.
- **Preganglionic ACh** → **Nn** → **excitation / Ca^{2+} entry** → **catecholamine secretion into blood**.
- Secretes mainly **adrenaline**, plus NA; no ordinary postganglionic axon.
- **Medulla:** catecholamines. **Cortex:** steroids, e.g. cortisol/aldosterone.

Compare	Noradrenaline / NA	Adrenaline
Usual role	Local sympathetic transmitter.	Circulating adrenal hormone.
Receptors	Strong $\alpha/\beta 1$; weak $\beta 2$.	α and β ; prominent $\beta 2$.
Shared effects	Direct cardiac stimulation; fuel mobilisation; α -mediated vasoconstriction.	
Key difference	Strong vasoconstriction; little bronchodilation.	$\beta 2$ bronchodilation and dilation in some vascular beds.

- **NA may slow the measured heart rate:** pressure rises → baroreflex increases vagal activity, overriding direct $\beta 1$ stimulation.
- **Adrenaline depends on concentration:** lower concentrations favour $\beta 2$ vasodilation; higher concentrations bring stronger α vasoconstriction.

Block the receptor: predict what is lost

Antagonist	Target	Main consequences
Atropine / scopolamine	Muscarinic	Less secretion/sweating; slower gut; dilated pupils, impaired near vision; urinary retention; usually faster heart.
Prazosin	$\alpha 1$	Vasodilation; weaker pressure support on standing.
Phentolamine	$\alpha 1 + \alpha 2$	Vasodilation; also removes inhibition of NA release.
Propranolol	$\beta 1 + \beta 2$	Less cardiac activity/renin; blocks airway relaxation too.
Metoprolol	Preferential $\beta 1$	Mainly cardiac effects; selectivity is not absolute.
Hexamethonium	Nn	Ganglion block: both autonomic branches affected.
Curare-type blockers	Nm	Skeletal-muscle paralysis; somatic, not muscarinic blockade.

- **Agonist activates:** phenylephrine $\alpha 1$ → constriction; clonidine $\alpha 2$ → less central sympathetic outflow; salbutamol $\beta 2$ → bronchodilation; isoproterenol → β stimulation.
- **Antagonist blocks:** removes existing signalling; effect depends on baseline tone.
- **Atropine blocks sympathetic sweating** because its target is muscarinic. It spares Nn/Nm.
- **Neostigmine inhibits ACh breakdown:** more ACh action at muscarinic and nicotinic sites; not receptor blockade.

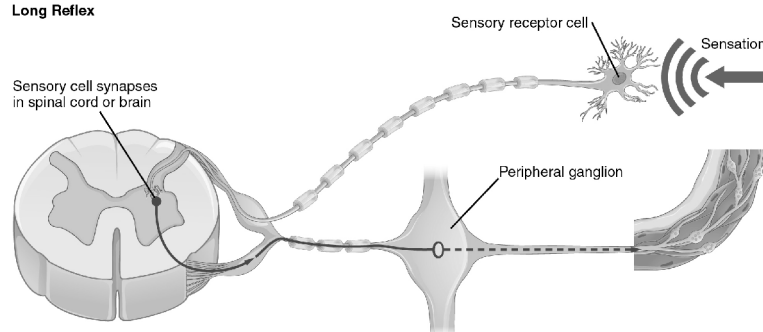
Local versus central reflexes

Official topic 4 · Intramural = in the organ wall; plexus = nerve network

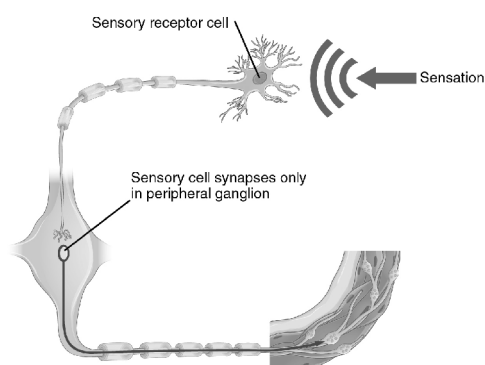
- **ENS:** sensory neurons + interneurons + motor neurons in gut wall; can generate local reflexes without CNS instructions.
- **Parasympathetic input** generally promotes digestion; **sympathetic input** generally suppresses it and constricts gut vessels.

Plexus	Location	Main role
Myenteric / Auerbach	Between circular and longitudinal muscle.	Motility / propulsion.
Submucosal / Meissner	In submucosa, near inner lining.	Secretion, mucosal function, local blood flow.

Long Reflex



Short Reflex



Practise drawing

- **Long:** sensor → afferent → CNS → autonomic efferents → target.
- **Short:** sensory input → local enteric circuit → target; no CNS loop required.
- Include arrows and label **sensory input, integrating centre and motor output**.
- Figure is schematic; actual enteric circuits include interneurons.

OpenStax College, Short and Long Reflexes · CC BY 3.0 · Greyscale rendering.

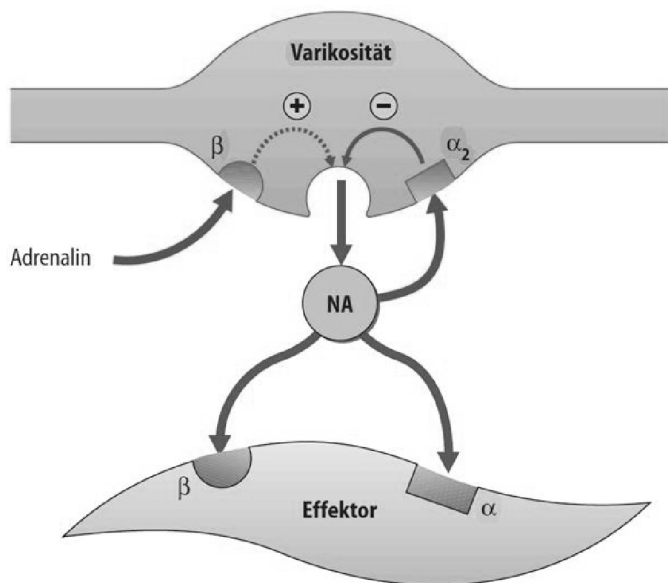
Peristaltic reflex

- Bolus stretches/stimulates gut → local sensory neurons activate enteric circuits.
- **Behind bolus:** circular muscle contracts. **Ahead:** relaxes → contents move forward.
- **Excitatory motor signals:** ACh and substance P.
- **Inhibitory motor signals:** nitric oxide (NO) and vasoactive intestinal peptide (VIP); relax smooth muscle. VIP also supports secretion.
- **Serotonin / 5-HT:** contributes to gut sensory signalling; released by mucosal enterochromaffin cells in response to local stimuli.

Independent is not isolated: the ENS can run local circuits, while the brain and autonomic branches modify them.

Control the amount released

Official topic 5 · Figure extracted from your actual seminar slides



What to draw

- Releasing nerve terminal above; target cell below.
- NA released towards target α/β receptors.
- Return arrow from NA to terminal α_2 , marked -.
- Adrenaline $\rightarrow$ terminal β , marked +.
- Keep terminal receptors separate from target receptors.

Jana Kivastik, Veg NS 2022 ENG, slide 20; figure attributed there to Schmidt / Thews. Original figure extracted; greyscale rendering.
Varikosität = varicosity; Adrenalin = adrenaline; Effektor = target cell.

Release, feedback and clearance are different steps

- **Release:** action potential $\rightarrow$ depolarisation $\rightarrow$ voltage-gated Ca^{2+} channels open $\rightarrow$ Ca^{2+} entry $\rightarrow$ vesicle fusion / **exocytosis**.
- **Varicosities:** swellings along autonomic axons; release transmitter over an area. This is a diffuse **neuroeffector junction**, unlike a skeletal-muscle end plate.
- **Presynaptic:** releasing terminal. **Postsynaptic:** receiving cell.
- **Autoreceptor:** detects own transmitter. NA activates terminal $\alpha_2 \rightarrow$ Gi-related inhibition of Ca^{2+} -dependent release $\rightarrow$ less further NA. **Negative feedback.**
- **Heteroreceptor:** detects another signal. Course example: adrenaline activates terminal β receptors $\rightarrow$ facilitates NA release.
- **ACh clearance:** acetylcholinesterase breaks it down; choline is recycled.
- **NA clearance:** mainly reuptake into terminal; reused or metabolised, including by monoamine oxidase (MAO). Diffusion also removes it.

How sympathetic and parasympathetic signals interact

- **At the heart:** β_1/Gs raises cAMP; M_2/Gi lowers cAMP and opens K^+ channels $\rightarrow$ opposing effects on nodal activity.
- **At terminals:** vagal ACh can inhibit nearby sympathetic NA release; sympathetic neuropeptide Y can inhibit vagal ACh release.
- **Tonic control:** ongoing sympathetic activity constricts vessels; reduce it $\rightarrow$ dilation without needing parasympathetic input.
- **Complementary control:** both branches can stimulate saliva, with different secretion patterns.

Visceral afferents and the baroreflex

Official topic 6 · Explain the loop from stimulus to correction

Visceral afferents

- **Visceral:** internal organs. **Afferent:** towards CNS. **Efferent:** away from CNS.
- Report stretch, pressure, chemical changes and harmful stimuli; support reflex control of circulation, digestion and bladder.
- Mostly unconscious; some input produces fullness, nausea or pain.
- Cell bodies lie in **sensory ganglia**, not the autonomic ganglia relaying motor output.
- **Vagus is mixed:** visceral afferents + parasympathetic efferents; not every vagal fibre is a motor fibre.

On standing: restore pressure

1. **Standing:** venous return ↓ → filling / stroke volume ↓ → arterial pressure ↓.
 2. **Baroreceptors stretch less:** carotid sinus and aortic arch firing ↓.
 3. **Afferents reach medulla:** carotid sinus via **IX**; aortic arch via **X**.
 4. **Output changes:** sympathetic ↑; cardiac vagal ↓.
 5. **β1:** heart rate/contractility ↑. **α1:** arterioles constrict → resistance ↑; veins constrict → venous return supported.
 6. **Pressure recovers:** response opposes disturbance = negative feedback.
- **If pressure rises:** stretch/firing ↑ → sympathetic ↓, vagal ↑ → slower heart and less vasoconstrictor drive.
 - **Orthostatic hypotension:** pressure falls on standing; impaired autonomic compensation is one possible cause.

Quick oral checks

Question	Essential answer
Why does atropine reduce sweating?	Sympathetic eccrine nerves use ACh; glands have muscarinic receptors.
Does a ganglion blocker block one branch?	No: both use Nn. Effect depends on the organ's dominant resting tone.
How does gut muscle relax ahead of a bolus?	Enteric inhibitory motor neurons release NO/VIP.
Feedback versus reuptake?	α2 feedback limits new release; reuptake removes transmitter already released.

Course coverage and sources

- **Official scope:** 1 organisation (pp. 1-3, 7); 2 sympathetic (pp. 1-5); 3 parasympathetic (pp. 1-5); 4 ENS (p. 6); 5 presynaptic regulation / interaction (p. 7); 6 visceral afferents (p. 8).
- **Course:** Veg NS ENGLISH 2021.pdf, seminar questions; Jana Kivastik, Veg NS 2022 ENG.pdf; Silverthorn chapter 11, pp. 399-408. Figure credits accompany each drawing.
- **Haemodynamic cross-check:** DailyMed labels for norepinephrine and epinephrine. Drawing checklists are study aids, not a claim that every figure will be examined.