

Neuroscience

Curated preclinical study track (not a medical degree, no licensure claim). Original explanations and practice only, following the reviewed course handout.

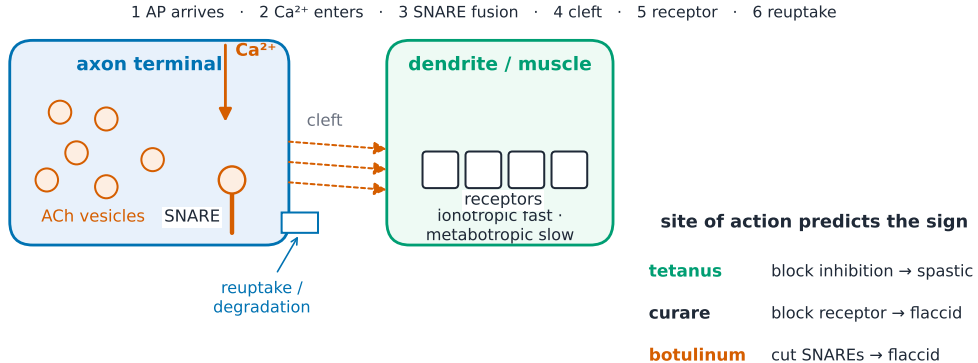
Traces and lesions

- Units 9.1-9.4 trace the wiring (synapses, sensory, motor, special senses); 9.5-9.7 build on it (autonomic, higher functions, localisation).
- Every tract is learned forward (trace it) and backward (localise along it).

Neurons, synapses and neurotransmitters: the decision point

- 1 Steps** AP $\rightarrow$ Ca^{2+} channels open $\rightarrow$ SNARE fusion $\rightarrow$ cleft $\rightarrow$ receptor $\rightarrow$ **reuptake** or degradation; the NMJ releases $\sim 5,000$ - $10,000$ molecules per vesicle and ~ 50 - 300 vesicles per impulse, 3-5 times what threshold needs.
- 2 Receptors** Ionotropic open channels in ms (nicotinic, GABA-A, AMPA/NMDA); metabotropic act through G-proteins in s (muscarinic, adrenergic).
- 3 Toxins** Botulinum cuts SNAREs $\rightarrow$ flaccid; curare blocks the receptor $\rightarrow$ flaccid; tetanus blocks inhibition $\rightarrow$ spastic: site predicts sign.

Synaptic transmission with its drug and toxin targets

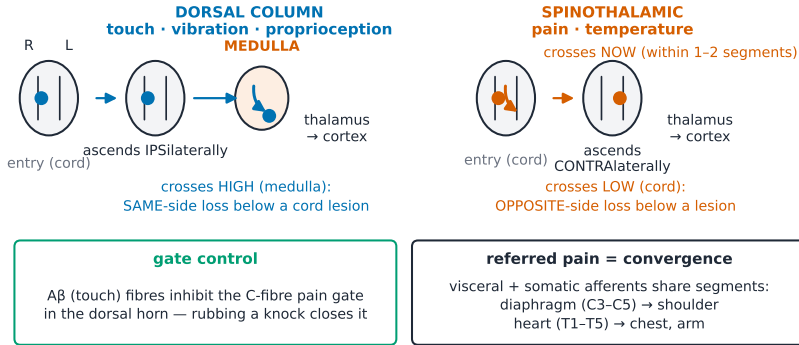


Six steps of transmission, each a drug or toxin target.

Somatosensory and pain pathways: two crossings

- 1 **Dorsal column** Fine touch, vibration, proprioception ascend IPSilaterally and cross in the MEDULLA - cord lesions hit them on the same side.
- 2 **Spinothalamic** Pain and temperature cross within 1-2 segments in the CORD - opposite-side loss below a lesion.
- 3 **Gate and referral** $A\beta$ fibres close the gate on C-fibre pain; convergence refers diaphragm (C3-C5) to the shoulder and heart (T1-T5) to chest and arm.

The two ascending pathways and their crossing points

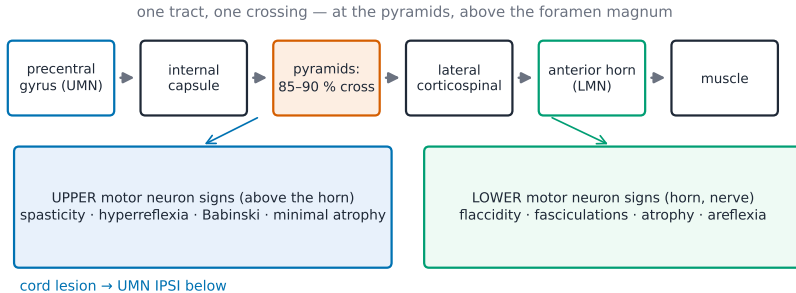


Dorsal column crosses in the medulla; spinothalamic crosses in the cord.

Motor systems: one tract, two neuron classes

- 1 Corticospinal** Precentral → internal capsule → pyramids (85-90 % decussate) → lateral column → anterior horn → muscle.
- 2 Signs** UMN: spastic, hyperreflexic, Babinski, minimal atrophy. LMN: flaccid, wasting, fasciculations, areflexia.
- 3 Computers** Basal ganglia scale amplitude (dopamine low → Parkinson; striatum lost → chorea); cerebellum times movement, ipsilaterally (dysmetria, **intention tremor**).

The corticospinal tract and the UMN-LMN fork



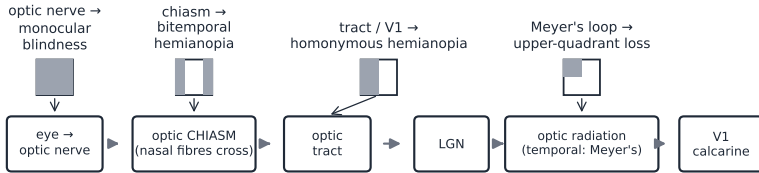
basal ganglia SCALE amplitude (dopamine low → Parkinson · striatum lost → chorea)
cerebellum TIMES movement, ipsilateral: dysmetria · intention tremor · dysidiadochokinesia

Precentral to anterior horn with one crossing at the pyramids.

Special senses: mapped pathways

- 1 Vision** Nerve → chiasm (nasal crosses) → tract → LGN → radiation → V1:
nerve = monocular, chiasm = bitemporal, behind = contralateral homonymous,
Meyer's loop = superior quadrant.
- 2 Hearing Tonotopy:** base high, apex low (~20-20,000 Hz); Rinne + Weber split
conductive (air lost, bone kept) from sensorineural (both reduced, Weber to the
better ear).
- 3 Balance** Three canals in orthogonal planes report rotation; otoliths report tilt and
linear acceleration.

The visual pathway and its field defects



cochlea is tonotopic

base → high frequencies · apex → low
young adult range ~20-20,000 Hz

Rinne + Weber

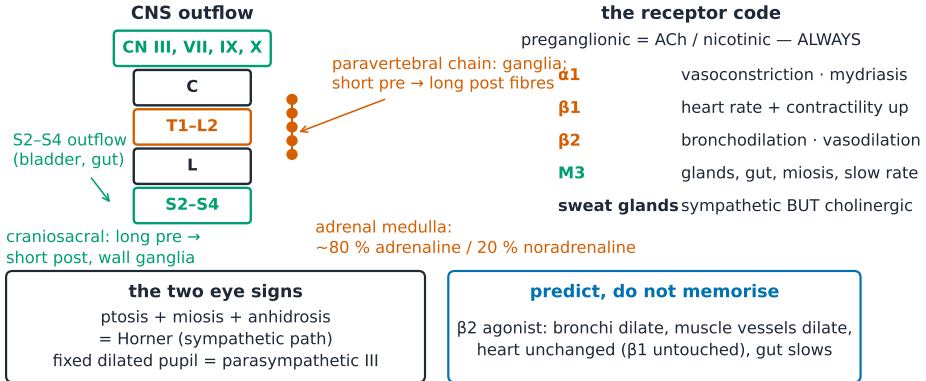
air > bone lost = conductive (middle ear)
both lost = sensorineural (cochlea/nerve)

Each lesion site stamps a signature field defect.

Autonomic nervous system: two limbs, one code

- 1 Outflow** Sympathetic T1-L2 (chain, short pre → long post; adrenal medulla ~80 % adrenaline / 20 % noradrenaline); parasympathetic craniosacral III, VII, IX, X + S2-S4.
- 2 Code** Preganglionic ACh/nicotinic always; $\alpha 1$ constricts, $\beta 1$ drives heart, $\beta 2$ dilates, M2 slows the heart, M3 secretes and moves the gut.
- 3 Eyes** Horner triad (ptosis, miosis, anhidrosis) = sympathetic lesion; fixed dilated pupil = III nerve.

Sympathetic and parasympathetic outflow

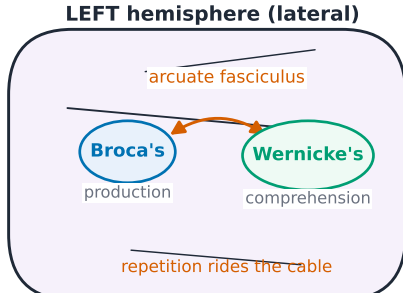


T1-L2 versus craniosacral, and the receptor code that predicts organ responses.

Higher functions: architecture predicts disorders

- 1 **Sleep** ~90-min cycles $\times$ 4-6: N3 delta (0.5-4 Hz, growth hormone) early; REM ~20-25 %, atonia, lengthens toward morning.
- 2 **Memory** Hippocampus episodic, striatum procedural, amygdala emotional, cerebellum conditioned - HM proved the dissociation.
- 3 **Language** Broca non-fluent, Wernicke fluent-empty, **arcuate fasciculus** = repetition destroyed (conduction).

Language areas and their connections



three aphasias, three sites

site	fluency	comprehension	repetition
Broca's	non-fluent, effortful	intact	impaired
Wernicke's	fluent, empty	impaired	impaired
conduction	fluent	intact	DESTROYED

*fluency names the front; content names the back;
repetition names the cable*

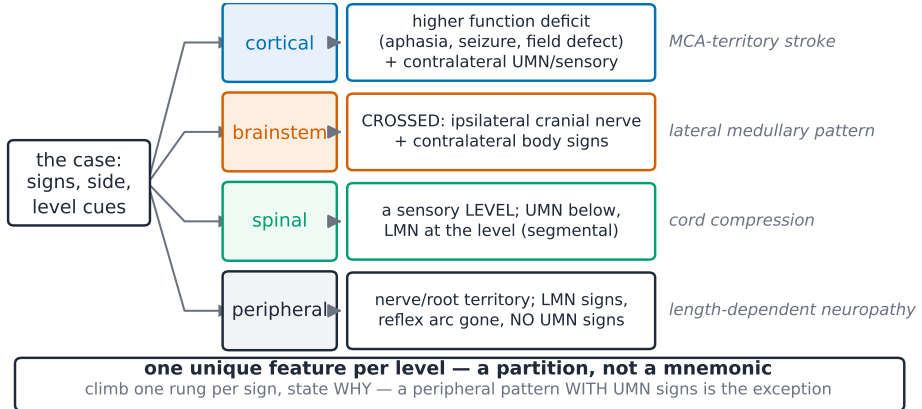
memory: hippocampus episodic · striatum procedural · amygdala emotional · cerebellum conditioned
sleep: ~90-min cycles × 4-6 · N3 delta early · REM ~20-25 % late

Broca produces, Wernicke comprehends, the **arcuate fasciculus** repeats.

Lesion localisation: the map is a partition

- 1 **Cortical** Higher function deficit (aphasia, seizure, field defect) + contralateral UMN/sensory signs.
- 2 **Brainstem** CROSSED: ipsilateral cranial nerve + contralateral body - the single most useful exam clue.
- 3 **Spinal / peripheral** Cord: a sensory LEVEL, UMN below, LMN at the level. Periphery: nerve/root territory, LMN signs, reflex arc gone, no UMN.

The lesion-localisation decision map



Cortical, brainstem, spinal or peripheral - one unique feature per level.

Check yourself

- Pain crosses where...? (In the cord, within 1-2 segments.)
- Cord lesion → UMN signs on which side...? (Same side - it crossed at the medulla.)
- Chiasm lesion → which field defect...? (Bitemporal **hemianopia**.)
- Aphasia with fluent speech, intact comprehension, dead repetition...? (Conduction - **arcuate fasciculus**.)