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— STUDY NOTES

Upper vs Lower Motor Neuron Signs

Faced with a weak or paralysed animal, the first question a clinician asks is: **is the problem in the lower motor neuron or the upper motor neuron?** The **lower motor neuron (LMN)** is the nerve cell that actually reaches the muscle — the 'final common path'. The **upper motor neuron (UMN)** lives entirely in the brain and cord and commands the LMN. Damage them and you get opposite pictures: an LMN lesion gives a **floppy, wasted, reflex-less** limb; a UMN lesion gives a **stiff limb with exaggerated reflexes** and little wasting. Read tone, reflexes and muscle bulk together and you can say *where* the lesion is — often before a single image is taken. This is the single most useful skill in the veterinary neurological exam.

INSIDE THESE NOTES

LEVEL
Vets & veterinary students

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Upper vs Lower Motor Neuron Signs

STUDY NOTES · BASIC SCIENCES · PHYSIOLOGY · UPDATED 2026-07-02

CONTENTS

LEARNING OBJECTIVES

After working through these notes you will be able to:

- ✓ Define the lower motor neuron (α motor neuron), the motor unit, and the upper motor neuron.
- ✓ Explain how the muscle spindle, the γ loop and the Golgi tendon organ set muscle tone and drive the stretch reflex.
- ✓ List the signs of LMN disease and explain the mechanism of each (including neurogenic atrophy).
- ✓ List the signs of UMN disease and explain the disinhibition ('release') mechanism.
- ✓ Contrast UMN and LMN lesions on tone, reflexes, atrophy, fasciculations and EMG.
- ✓ Localise a lesion from the limb pattern to a spinal cord region (C1–C5, C6–T2, T3–L3, L4–S3), and rule out the neuromuscular junction and muscle.

TL;DR

Faced with a weak or paralysed animal, the first question a clinician asks is: **is the problem in the lower motor neuron or the upper motor neuron?** The **lower motor neuron (LMN)** is the nerve cell that actually reaches the muscle — the 'final common path'. The **upper motor neuron (UMN)** lives entirely in the brain and cord and commands the LMN. Damage them and you get opposite pictures: an LMN lesion gives a **floppy, wasted, reflex-less** limb; a UMN lesion gives a **stiff limb with exaggerated reflexes** and little wasting. Read tone, reflexes and muscle bulk together and you can say *where* the lesion is — often before a single image is taken. This is the single most useful skill in the veterinary neurological exam.

AT A GLANCE

CORE QUESTION	Is the lesion in the lower motor neuron or the upper motor neuron?
LOWER MOTOR NEURON	The α motor neuron — cell body in the CNS, axon out to muscle; the 'final common path'
UPPER MOTOR NEURON	Neurons entirely within the CNS that command and inhibit the LMN
LMN LESION	Flaccid paresis · ↓ tone · ↓/absent reflexes · rapid severe atrophy · fasciculations
UMN LESION	Spastic paresis · ↑ tone · normal/↑ reflexes · little atrophy · no fasciculations
WHY REFLEXES RISE	The UMN normally inhibits the reflex arc; a lesion 'releases' it (disinhibition)
MUSCLE TONE	Held by slow (type I) motor units firing tonically, kept sensitive by the γ loop
LOCALISES WHERE	LMN signs = that cord segment/nerve; UMN signs = below the lesion
TWO MORE SITES	Rule out the neuromuscular junction and the muscle itself
PAYOFF	Tone + reflexes + atrophy pattern place the lesion in the nervous system

01 The one question that localises a case

- **The key decision:** is the lesion in the **lower** or the **upper** motor neuron? Get it right and you have localised the problem — often before imaging.
- **Two-neuron relay:** an **upper motor neuron (UMN)** in the CNS commands a **lower motor neuron (LMN)**; only the LMN reaches muscle.
- **4 sites can weaken a limb:** UMN · LMN · neuromuscular junction · muscle.

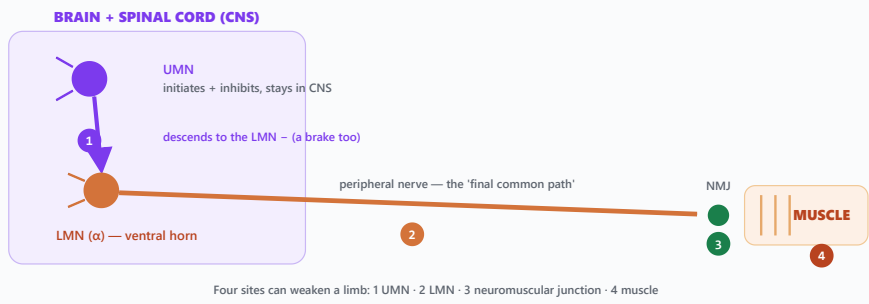

Fig 1 — UMN (in the CNS) → LMN → muscle; the four lesion sites.

02 The lower motor neuron = the final common path

- **LMN = the α (alpha) motor neuron:** cell body in the ventral horn (or brainstem), axon out to muscle — the **'final common path'** every command (reflex or voluntary) must pass through.
- **Motor unit** = one α motor neuron + all the fibres it drives. **Force is graded** by **recruiting units smallest-first** (the size principle) and by raising firing rate.
- **Slow (type-I) units** = small, fatigue-resistant, postural; **fast (type-II)** = large, powerful, for bursts.

03 Muscle tone & the stretch reflex (the γ loop)

- **Tone is active:** slow (type-I) motor units firing tonically at a low, steady rate — set by the cord and adjusted by the brain.
- **Muscle spindle** (in **parallel**, senses length) → **Ia** sensory fibre → **monosynaptic** excitation of the **same muscle's** α neuron = the **stretch reflex** (knee-jerk). Also inhibits the antagonist (reciprocal inhibition).
- **γ (gamma) loop:** γ neurons contract the spindle's poles so it stays taut as the muscle shortens (**α–γ coactivation**) — a built-in error-corrector.

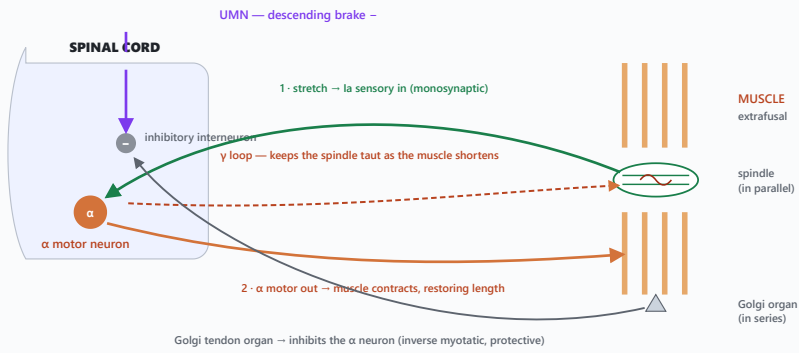

Fig 2 — The stretch reflex circuit: spindle (parallel) → α monosynaptically; the γ loop keeps it sensitive; the Golgi tendon organ (series) inhibits; the UMN brakes.

04 The reflex family

- **Golgi tendon organ** (in **series**, senses **tension**) → **inhibits** its own muscle (inverse myotatic) — protective against tendon rupture.
- **Withdrawal (flexor) reflex:** a noxious stimulus flexes the limb + inhibits its extensors, before conscious awareness.
- **Reflex types:** segmental (knee-jerk, L4–L6), intersegmental (cutaneous trunci), suprasegmental (postural). Each tests a known stretch of the nervous system.

05 Signs of LMN disease (everything Lost)

- **Flaccid** paresis / paralysis (floppy) — no command reaches the muscle.
- **Reduced muscle tone** (reflex loop broken).
- **Reduced or absent reflexes** (hyporeflexia / areflexia) — the arc's motor limb is gone.
- **Rapid, severe (neurogenic) atrophy** — visible within days (protein breakdown outpaces synthesis).
- **Fasciculations** + abnormal **EMG** within days (denervation).

06 The upper motor neuron = the brain's descending control

- **UMN = neurons entirely within the CNS** that command and modulate the LMN; they never reach muscle.
- **Descending tracts:** corticospinal / pyramidal (fine control; **minor** in vet species → only toe-drag / placing deficits), **rubrospinal** (**dominant** for skilled distal movement in quadrupeds), reticulospinal (antigravity **tone**), vestibulospinal (balance), tectospinal (orienting).
- **Key role:** the UMN largely **inhibits (brakes)** the spinal reflexes. **Decussation** → brain lesion = signs on the **opposite** side; cord lesion = **same** side, below the lesion. Cerebellum times movement; basal nuclei select it.

07 Signs of UMN disease (everything Up) — and why

- **Spastic** paresis below the lesion (stiff, not floppy); **increased tone**; **normal to exaggerated reflexes** (hyperreflexia).
- **Little atrophy** (LMN intact → slow disuse only); **no fasciculations**, normal EMG.
- **Why: disinhibition** — the UMN normally brakes the reflex; a lesion removes the brake so the reflex is 'released'.
- **Watch:** acute **spinal shock** can transiently depress reflexes; a T3–L3 lesion can give **Schiff–Sherrington** forelimb extension (not forelimb UMN disease).

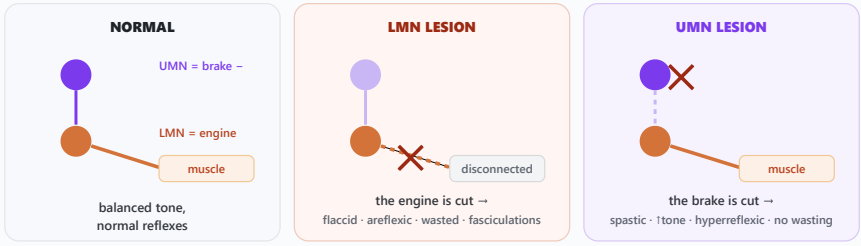

Fig 3 — An LMN lesion cuts the engine; a UMN lesion cuts the brake.

08 UMN vs LMN — side by side

FEATURE	LMN LESION	UMN LESION
Paralysis	Flaccid (floppy)	Spastic (stiff)
Muscle tone	Reduced	Increased
Reflexes	Reduced / absent	Normal to exaggerated
Atrophy	Rapid & severe (days)	Mild & late (disuse)
Fasciculations	Present	Absent
EMG	Abnormal (denervation)	Normal

- **LMN = Lost** (Low tone, Lost reflexes, muscle melts). **UMN = Up** (tone up, reflexes up, muscle kept). Floppy vs stiff.

09 Localising by spinal cord region

- **LMN signs localise to that segment / nerve. UMN signs appear BELOW the lesion** → their upper edge marks the level.
- **The four regions: C1–C5** (UMN all four limbs) · **C6–T2** (LMN fore, UMN hind) · **T3–L3** (normal fore, UMN hind — commonest, TL disc) · **L4–S3** (LMN hind).
- **Bladder:** lesion above sacral = **UMN bladder** (distended, hard to express); sacral lesion = **LMN bladder** (flaccid, easily expressed, overflow).

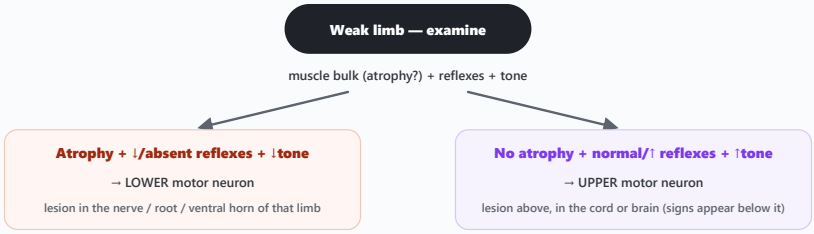

Fig 4 — Atrophy + lost reflexes → LMN; no atrophy + brisk reflexes → UMN higher up.

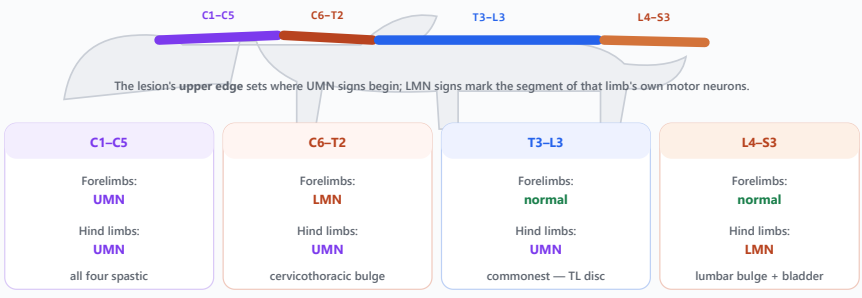

Fig 5 — The limb pattern maps to one of four cord regions (C1–C5, C6–T2, T3–L3, L4–S3).

10 The other two sites: NMJ & muscle

- **Neuromuscular junction: myasthenia gravis** (fatigable; improves with an anticholinesterase), **botulism** / **tick paralysis** (flaccid, LMN-like), hypocalcaemia (ruminants).
- **Muscle:** inflammatory + inherited myopathies (incl. dystrophin dystrophy).
- **Separating tests:** nerve won't drive but **direct muscle stimulation contracts** → nerve/junction; improvement with an **anticholinesterase** → NMJ; EMG separates denervation from myopathy.

11 Two textbook cases

- **LMN:** dog with ascending flaccid paralysis of all four legs, widespread atrophy, absent reflexes, **deep pain intact** → diffuse LMN disease = **polyradiculoneuritis ('coonhound paralysis')**, ventral nerve roots.
- **UMN:** dachshund with weak hind legs, NO atrophy, normal reflexes, normal front legs → **UMN lesion, T3–L3 cord** = **thoracolumbar disc herniation**.

Fig 6 — Diffuse LMN disease vs a focal UMN cord lesion — read off tone, reflexes and atrophy.

RED FLAG

Rapidly progressive paralysis, loss of deep pain, or failing respiration (diffuse LMN disease — polyradiculoneuritis, tick paralysis, botulism) is a neuro emergency: stabilise, protect ventilation, refer.

> *Read tone, reflexes and muscle bulk together and four vague possibilities collapse to a region of the nervous system — often before any imaging.*
>
> — after Cunningham 6e Ch 6–10 & PDA 3e Ch 4 & 8.

KEY TERMS — QUICK GLOSSARY

Lower motor neuron (LMN)	The α motor neuron — cell body in the CNS, axon out to skeletal muscle; the final common path.
Upper motor neuron (UMN)	A neuron lying entirely within the CNS that controls (excites and inhibits) the lower motor neuron.
Final common path	Every motor command — reflex or voluntary — must pass through the α motor neuron to reach muscle.
Motor unit	One α motor neuron plus all the muscle fibres it innervates; the smallest gradable unit of contraction.
Size principle	Motor units are recruited smallest-first, giving fine control at low force and power as demand rises.
Muscle spindle	A stretch receptor of intrafusal fibres lying in parallel with the muscle; signals length and rate of stretch.
γ (gamma) motor neuron	Innervates the spindle's contractile poles, keeping it taut and sensitive as the muscle shortens (α–γ coactivation).
Golgi tendon organ	A tension sensor in series with the muscle at the tendon; drives protective inhibition (inverse myotatic reflex).
Flaccid vs spastic	Flaccid = floppy, low tone (the LMN picture); spastic = stiff, high tone (the UMN picture).
Hyporeflexia / areflexia	Reduced or absent reflexes — the LMN pattern (the reflex arc's motor limb is gone).
Hyperreflexia	Exaggerated reflexes — the UMN pattern (loss of descending inhibition).
Neurogenic atrophy	Rapid, severe muscle wasting after its LMN is lost (visible within days).
Fasciculations	Visible twitches of denervated motor units — a lower motor neuron sign.
Disinhibition ('release')	Loss of the UMN's normal braking of spinal reflexes, causing hyperreflexia and increased tone.

Schiff–Sherrington	A T3–L3 cord lesion causing rigid forelimb extension with normal forelimb strength — not a UMN sign in the forelimbs.

QUICK REVISION — REMEMBER THESE

1. Every motor command reaches muscle through a **two-neuron chain**: an **upper motor neuron (UMN)** in the CNS commands a **lower motor neuron (LMN)**, and only the LMN reaches the muscle.
2. The LMN is the **α motor neuron** — the **'final common path'**; damage disconnects the muscle from all control, reflex and voluntary alike.
3. One α motor neuron plus the fibres it drives is a **motor unit**; the CNS grades force by **recruiting units in size order** and by raising their firing rate.
4. Resting **muscle tone** is active — slow (type I) motor units fire tonically, and the **γ loop keeps the muscle spindle sensitive** at every length.
5. **LMN disease** gives a stereotyped picture: **flaccid** paresis, lost tone, **reduced or absent reflexes**, rapid severe (neurogenic) **atrophy**, and fasciculations.
6. **UMN disease** looks the opposite below the lesion: **spastic** paresis, increased tone, **normal-to-exaggerated reflexes** (hyperreflexia), and little wasting.
7. Reflexes are exaggerated in UMN disease because of **disinhibition**: the UMN normally brakes the spinal reflex, so losing it releases the reflex.
8. Reading **tone, reflexes and atrophy together localises the lesion** — and the same exam rules in or out the **neuromuscular junction** and the **muscle**.

MEMORY AIDS

LMN = Lost — **L**MN disease = everything **L**ost: **L**ow tone, **L**ost reflexes, muscle wastes fast. Floppy / flaccid.

UMN = Up — **U**MN disease = everything **UP**: tone **up** (spastic), reflexes **up** (hyper); the LMN is intact so muscle stays. Stiff.

Brake off — The UMN is the **brake** on the reflex. An LMN lesion cuts the engine (no reflex); a UMN lesion cuts the brake (reflex runs wild).

Below the lesion — UMN signs appear **below** the lesion — their upper edge marks the level. LMN signs mark **their own** segment.

Four regions — Limb pattern → cord region: C1–C5 (all UMN), C6–T2 (fore LMN/hind UMN), T3–L3 (hind UMN), L4–S3 (hind LMN).

TEST YOURSELF — ACTIVE RECALL

Cover the answers and try to retrieve each one from memory first — self-testing beats re-reading.

1. **What is the lower motor neuron, and why is it the 'final common path'?**
2. **What is a motor unit, and how does the CNS grade the force of a muscle?**
3. **How do the muscle spindle and the γ loop keep the stretch reflex working as a muscle shortens?**
4. **List the cardinal signs of LMN disease and the mechanism of each.**
5. **List the signs of UMN disease and explain why reflexes are exaggerated.**
6. **Why does muscle waste fast in LMN disease but not in UMN disease?**
7. **A dog has weak hind legs, brisk knee-jerks and no atrophy; the forelegs are normal. Where is the lesion?**
8. **A dog has flaccid tetraparesis with widespread atrophy and absent reflexes but intact deep pain. Where is the lesion?**
9. **Besides UMN and LMN, what two sites can weaken a limb, and how do you exclude them?**

ANSWERS

1. The α motor neuron (cell body in the CNS, axon to skeletal muscle). It is the final common path because every motor command — reflex or voluntary — must pass through it to reach the muscle.
2. A motor unit is one α motor neuron plus all the fibres it innervates. Force is graded by recruiting more units (smallest first — the size principle) and by raising their firing frequency.
3. The spindle lies in parallel with the muscle and signals length; when the muscle shortens it would go slack, so the γ motor neurons contract its poles (α–γ coactivation), keeping it taut and sensitive.
4. Flaccid paresis/paralysis (no command reaches the muscle), reduced tone (reflex loop broken), reduced/absent reflexes (motor limb of the arc gone), rapid neurogenic atrophy (denervated muscle wastes within days), and fasciculations (denervated units fire on their own).
5. Spastic paresis below the lesion, increased tone, normal-to-exaggerated reflexes, little atrophy and normal EMG. Reflexes are exaggerated because the UMN normally inhibits the spinal reflex arc; losing it disinhibits ('releases') the reflex.
6. LMN loss denervates the muscle, so rapid neurogenic atrophy appears within days. In UMN disease the LMN is intact, so the muscle stays innervated and only slow disuse atrophy develops much later.
7. A UMN lesion in the T3–L3 spinal cord (e.g. a thoracolumbar disc) — UMN signs in the hind limbs only, with normal forelimbs, place the lesion between the two limb enlargements.
8. Diffuse LMN disease of the ventral nerve roots — classically polyradiculoneuritis ('coonhound paralysis'); the intact deep pain reflects sparing of the dorsal (sensory) roots.
9. The neuromuscular junction and the muscle. If a nerve won't drive the muscle but direct muscle stimulation still makes it contract, the fault is in the nerve/junction; improvement with an anticholinesterase points to the neuromuscular junction (myasthenia gravis).

WHEN TO REFER OR ESCALATE

- Acute flaccid tetraparesis with widespread atrophy and absent reflexes — suspect diffuse **LMN disease** (polyradiculoneuritis, tick paralysis, botulism); monitor respiration and refer.
- Spastic paresis below a spinal level with preserved/exaggerated reflexes and no atrophy — a **UMN (spinal cord) lesion** (e.g. disc herniation); image and refer, urgently if deep-pain sensation is fading.
- Any rapidly progressive or markedly asymmetric paresis, or loss of deep pain — a neurological emergency; refer promptly.
- Fatigable weakness that improves with rest, or a positive response to an anticholinesterase — investigate the **neuromuscular junction** (myasthenia gravis).
- Focal atrophy following a single nerve's territory (a limb, or the tongue) — localise to that **LMN / nerve** and pursue the cause.

SOURCES

1. Klein BG. Cunningham's Textbook of Veterinary Physiology. 6th ed. St Louis: Elsevier; 2020. Chapter 9 — The Concept of Lower and Upper Motor Neurons and Their Malfunction (pp 93–96): the α motor neuron / final common path, LMN vs UMN signs, the disinhibition mechanism, and localisation cases.
2. Klein BG. Cunningham's Textbook of Veterinary Physiology. 6th ed. Elsevier; 2020. Chapter 6 — The Physiology of Muscle (pp 73–81: the motor unit, size-principle recruitment, fibre types, the EMG); Chapter 7 — The Concept of a Reflex (pp 82–85: the reflex arc, reciprocal inhibition, reflex types); Chapter 8 — Skeletal Muscle Receptor Organs (pp 86–92: the muscle spindle, γ motor neurons and α–γ coactivation, the Golgi tendon organ); Chapter 10 — The Central Control of Movement (pp 97–108: the descending tracts, medial vs lateral systems, corticospinal decussation and the rubrospinal-dominant quadruped, cerebellum and basal nuclei).
3. Sjaastad ØV, Sand O, Hove K. Physiology of Domestic Animals. 3rd ed. Oslo: Scandinavian Veterinary Press; 2016. Chapter 4 — The Nervous System (the α and γ motor neurons and final common path, the monosynaptic stretch reflex, muscle spindle and reciprocal inhibition, the Golgi tendon organ and withdrawal reflex, and the descending tracts and their inhibition of spinal reflexes) & Chapter 8 — Muscles (the motor unit and size-principle recruitment, muscle tone from tonic type-I firing, denervation atrophy and the EMG).
4. de Lahunta A, Glass E, Kent M. Veterinary Neuroanatomy and Clinical Neurology. 5th ed. Elsevier; 2021 — the standard clinical reference for the UMN/LMN sign comparison, the four cord-region localisation scheme (C1–C5, C6–T2, T3–L3, L4–S3), spinal shock and the Schiff–Sherrington phenomenon, and the UMN vs LMN bladder.
5. Lorenz MD, Coates JR, Kent M. Handbook of Veterinary Neurology. 5th ed. Elsevier; 2011 — lesion localisation from the neurological examination and the disorders of the neuromuscular junction and muscle (myasthenia gravis, botulism, tick paralysis, myopathies).

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