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PHYSIOLOGY · STUDY GUIDE

Upper vs Lower Motor Neuron Signs

How to Tell Them Apart and Localise the Lesion

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?

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IN SHORT

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.

KEY POINTS

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**.

01 The one question that localises a neuro case

An animal is carried into the clinic unable to walk. Before reaching for a single test, veterinary neurology hangs on one decision: **is the lesion in the lower motor neuron or the upper motor neuron?** Get that right and you have already narrowed the problem from "somewhere in the

nervous system" to a defined region of the spinal cord or brain. It is the most useful single concept in the neurological examination, and it rests on a simple piece of anatomy that every clinician can carry in their head.

Every command to a skeletal muscle travels a **two-neuron relay**. The **upper motor neuron (UMN)** starts in the brain or brainstem and runs down inside the central nervous system; it never touches muscle. It hands its command to the **lower motor neuron (LMN)**, whose axon leaves the cord in a peripheral nerve and actually reaches the muscle. So a weak limb can fail at one of **four places**: the UMN, the LMN, the **neuromuscular junction** where nerve meets muscle, or the **muscle** itself. Most of this article is about telling the first two apart — because they produce strikingly opposite pictures — and the last section returns to the other two sites, which can masquerade as lower-motor-neuron disease.

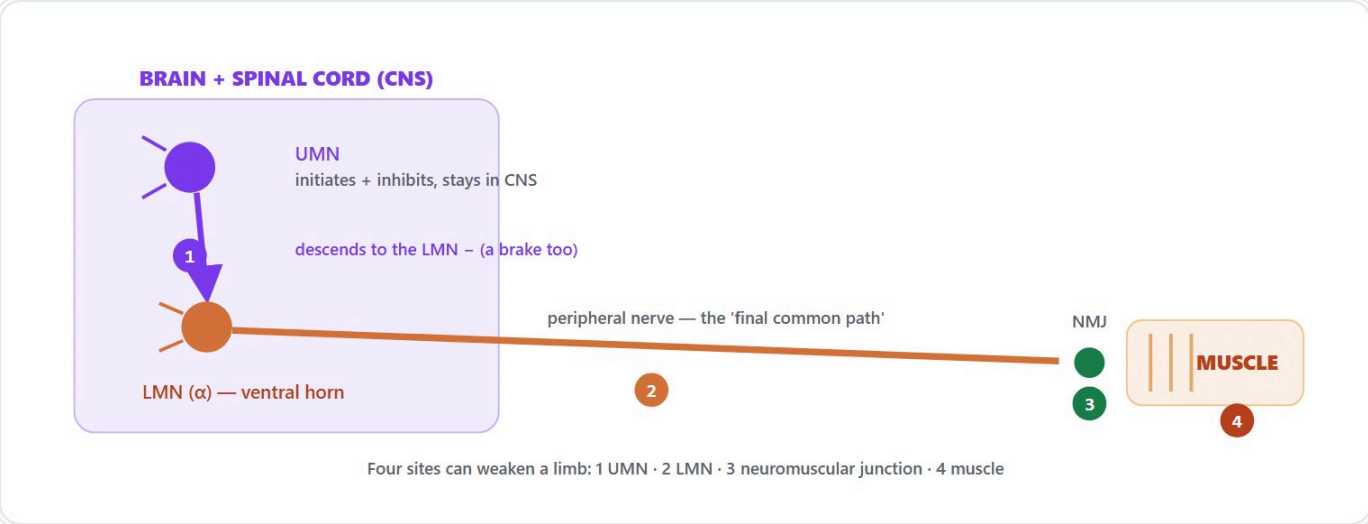


Fig 1 — The motor relay. A UMN (inside the CNS) commands the LMN, whose axon runs out through a peripheral nerve to the muscle. A weak limb can fail at the UMN, the LMN, the neuromuscular junction, or the muscle.

02 The lower motor neuron: the final common path

The **lower motor neuron is the α (alpha) motor neuron** — the cell whose body sits in the **ventral horn** of the spinal cord grey matter (or in the motor nuclei of the brainstem, for the muscles of the head), and whose axon carries the nerve impulse out through a peripheral nerve to the skeletal muscle it drives. It is the **"final common path"**: every influence on that muscle — a spinal reflex, a voluntary command from the cortex, a postural adjustment from the brainstem — must funnel through this one neuron to reach the muscle. Cut it, and the muscle is deaf to *all* of them. It is also the only neuron in the whole chain that leaves the central nervous system, which is exactly why its diseases look so different from those of the UMN.

The unit of action is not the single fibre (fiber) but the **motor unit**: one α motor neuron plus *all* the muscle fibres it innervates. When that neuron fires, every fibre in its unit contracts together, and all the fibres of a unit are the same functional type. Units vary enormously in size — a muscle for fine work such as an extraocular or a digit muscle may have only a handful of fibres per neuron, while a big postural muscle may have many hundreds. This is how the nervous system grades force. It **recruits units in size order** (the **size principle**): the smallest, most fatigue-resistant units switch on first for delicate low-force work, and progressively larger units are added as more power is demanded, so force climbs smoothly rather than in lurches. On top of recruitment, the CNS can raise the **firing frequency** of active units, fusing individual twitches into a smooth, stronger contraction. Together these two dials let a muscle span forces that differ by factors of thousands.

Two refinements matter clinically. First, the fibre types recruited follow the same logic: **slow (type I) units** are small, oxidative and fatigue-resistant — ideal for the continuous, low-level work of holding a joint against gravity — while **fast (type II) units** are large, powerful and quickly fatigued, held in reserve for sprinting or leaping. Second, although the α motor neuron is the classic LMN, the small **γ (gamma) motor neurons** that supply the muscle spindle, and the autonomic motor neurons, are lower motor neurons too; but the great majority of the clinical signs of "LMN disease" are explained by loss of the α motor neuron and its final common path.

03 Muscle tone and the stretch reflex: the γ loop

A limb at rest is not limp. It carries a low, steady background contraction — **muscle tone** — and that tone is *active*, produced by slow (type I) motor units firing at a low, almost constant frequency for long periods. Tone is not just a property of the muscle; it is continuously set by reflex machinery in the cord and adjusted by the descending pathways from the brain. Understanding that machinery is the key to reading the neurological examination, because **tone and reflexes are exactly what change — in opposite directions — in UMN and LMN disease.**

At the heart of it lies the **muscle spindle**, a stretch receptor built from a small bundle of specialised **intrafusal** muscle fibres wrapped in a capsule and lying **in parallel** with the ordinary force-producing (extrafusal) fibres. Because it is in parallel, stretching the muscle stretches the spindle. Sensory endings coiled around the non-contractile middle of the intrafusal fibres fire in proportion to how much, and how fast, the muscle is lengthening. Those **Ia sensory fibres** run into the cord and make an **excitatory, monosynaptic connection directly onto the α motor neurons of the very same muscle** — the only true monosynaptic reflex in the body. Stretch the muscle and it contracts straight back to length. This is the **stretch (myotatic) reflex**, and the patellar (knee-jerk) tap is simply the version clinicians can elicit most reliably, because the patella acts as a pulley that turns a tendon tap into a clean lengthwise stretch of the quadriceps. The same Ia fibres also drive inhibitory interneurons onto the *antagonist* muscle's motor neurons — **reciprocal inhibition** — so the joint is free to move.

There is a problem the spindle must solve, and its solution is elegant. When the muscle shortens during a contraction, the spindle lying in parallel would go slack and stop reporting length — it would be blinded exactly when feedback is needed. The **γ motor neurons** prevent this: they innervate the contractile *poles* of the intrafusal fibres, and by contracting those poles they take up the slack and keep the spindle's middle taut. Because γ activity is switched on *together* with the α command (**α–γ coactivation**), the spindle stays sensitive across the whole range of muscle length. This also gives the system an automatic error-corrector: if a limb meets an unexpected load and falls short of the intended movement, the spindle is stretched, the stretch reflex fires, and extra contraction is added — a "servo-assist" that works rather like power steering.

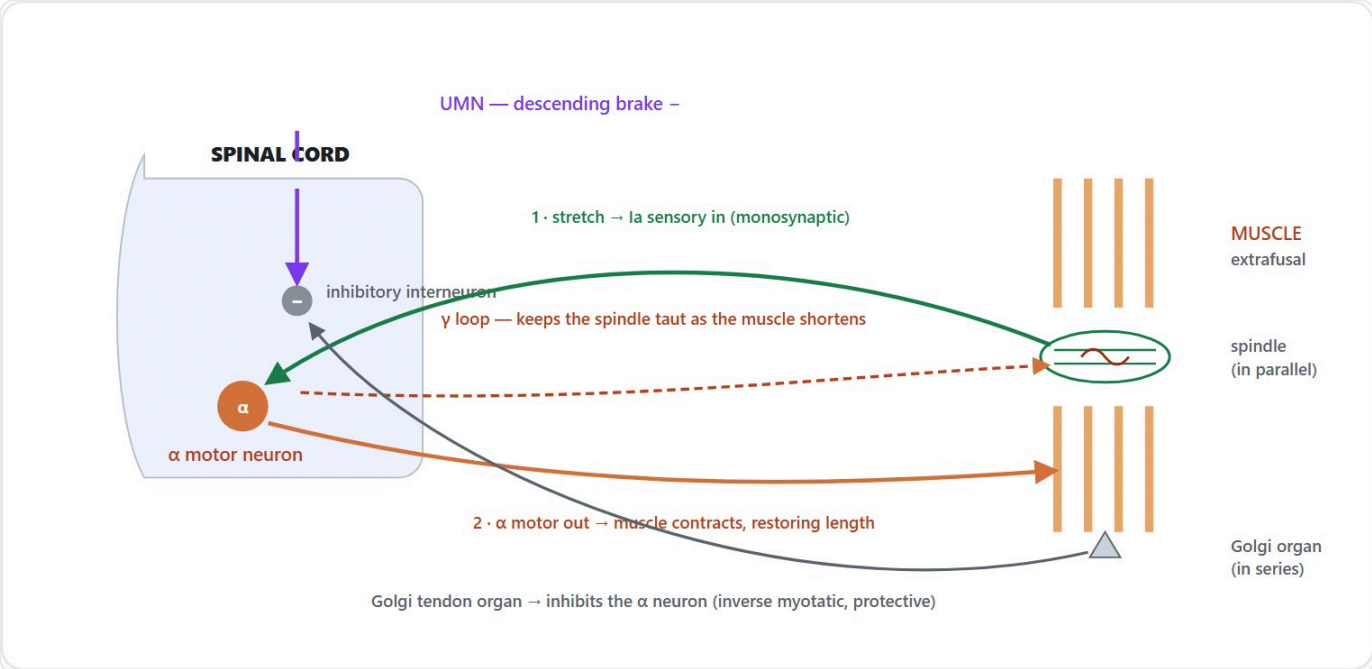


Fig 2 — The stretch reflex circuit. A muscle spindle (in parallel) drives the α motor neuron monosynaptically back to the same muscle; the γ loop keeps the spindle taut as the muscle shortens; the Golgi tendon organ (in series) drives protective inhibition; and the upper motor neuron feeds in a descending inhibitory (braking) input.

Study tip Spindle = *in parallel*, senses *length*, excites the same muscle. Golgi tendon organ = *in series*, senses *tension*, inhibits the same muscle. Getting those two straight explains most of the reflex exam.

04 The reflex family: tendon organ, withdrawal and reflex types

The stretch reflex is only the simplest member of a family. Sitting at the muscle–tendon junction, **in series** with the muscle, is the **Golgi tendon organ**, which senses **tension** rather than length. When the muscle contracts hard, the tendon organ fires and — through inhibitory interneurons — damps down its own muscle's α motor neurons. This **inverse myotatic reflex** is protective: it prevents a contraction from becoming forceful enough to tear the tendon from bone. Notice the neat symmetry: because the spindle is in parallel, a contraction *reduces* its firing; because the tendon organ is in series, the same contraction *increases* its firing.

A second everyday reflex is the **withdrawal (flexor) reflex**. A noxious stimulus to the foot excites interneurons that fire the limb's flexor motor neurons and simultaneously inhibit its extensors, so the paw is pulled away — and it happens before the animal is consciously aware of the pain. In a standing animal this is coupled to support from the opposite limb so balance is kept. Clinically, the withdrawal reflex is invaluable because, like the knee-jerk, it tests a specific segment of the cord and its nerves: a present withdrawal tells you the local reflex arc is intact, whatever the brain above is doing.

Reflexes are classified by how much of the nervous system their arc spans. A **segmental** reflex is confined to one small region of the cord — the patellar reflex, occupying only the L4–L6 segments, is the classic example. An **intersegmental** reflex runs its circuitry up or down several segments, as in the cutaneous trunci ("panniculus") reflex, where a skin pinch far back travels forward to twitch the skin muscle. A **suprasegmental** reflex also loops through the brain, as the postural vestibulospinal reflexes do. This anatomy is precisely why testing reflexes localises disease: each reflex is a live check on a known stretch of the nervous system.

05 Signs of lower motor neuron disease

Because the LMN is the only route to the muscle, its loss produces a **stereotyped, unmistakable set of signs** whatever the underlying cause — the muscle is simply disconnected from the nervous system.

- **Flaccid paresis or paralysis.** With no impulse reaching the neuromuscular junction the muscle cannot contract, so the limb hangs **floppy** ("flaccid"). If only some of a nerve's axons are lost — or the muscle is served by more than one spinal nerve — the result is weakness (paresis) rather than complete paralysis.
- **Reduced muscle tone.** Tone depends on a working reflex loop and on tonic firing of the α motor neuron; break that neuron and the loop collapses, so the limb goes limp and offers no resistance to passive movement.
- **Reduced or absent reflexes.** The α motor neuron is the motor limb of every reflex arc, so the knee-jerk and withdrawal reflexes become weak or disappear (**hyporeflexia / areflexia**) — even though the spindle may still be sensing the stretch perfectly well.
- **Rapid, severe atrophy.** A denervated muscle wastes fast — visibly, **within days** — because losing its nerve tips the balance of protein turnover sharply toward breakdown (the ubiquitin–proteasome and autophagy pathways are switched up). This **neurogenic atrophy** is far quicker and deeper than the slow atrophy of simple disuse.
- **Fasciculations and EMG changes.** Diseased or denervated motor units discharge on their own, producing fine visible twitches (fasciculations) under the skin; on an **electromyogram** abnormal spontaneous activity appears within a few days — a useful early confirmation of denervation.

In short, an LMN limb is **floppy, wasted and reflex-less**. Because peripheral nerves usually carry sensory fibres alongside motor ones, there may also be sensory loss in the same territory — but that is not itself a cardinal LMN sign.

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

The **upper motor neuron** is any neuron that lives **entirely within the CNS** and shapes the activity of the lower motor neuron. Voluntary movement is *initiated* by UMNs in the motor cortex, but the descending system is far broader than one pathway, and in domestic mammals its make-up differs importantly from the human textbook picture. Its axons descend in several named tracts, conventionally split into a **lateral system** that governs the distal, skilled, largely flexor muscles of the limbs and a **medial system** that governs the axial and proximal, largely extensor muscles of posture.

- **Corticospinal (pyramidal) tract** — the only *direct* projection from cerebral cortex to the cord, responsible in primates for the most dexterous voluntary movement. Most of its fibres cross at the pyramidal decussation (about 75% in the dog, up to 90% in primates). Crucially, in most veterinary species this tract is comparatively **minor**, so a lesion above the decussation causes not a dense hemiparesis but only subtle contralateral signs — a proprioceptive placing deficit, a toe that drags.
- **Rubrospinal tract** — from the red nucleus of the midbrain; the chief *lateral* pathway in quadrupeds, driving skilled movement of the distal limb. In domestic mammals it does much of the work the corticospinal tract does in primates.
- **Reticulospinal tracts** — from the reticular formation of pons and medulla; the principal controllers of antigravity **muscle tone**, with an excitatory (pontine) and an inhibitory (medullary) component, and the pathway that pre-sets posture just before a voluntary movement.
- **Vestibulospinal tracts** — from the vestibular nuclei; they fire the antigravity extensors to keep the animal upright when balance is disturbed.
- **Tectospinal tract** — orients the head toward a sudden sight or sound.

Two points from this anatomy pay off at the cage side. First, a large part of what the descending system does is **inhibit** — it holds the spinal reflexes in check, which is exactly why an animal can consciously override a withdrawal reflex to walk across a hot surface. Keep that braking role in mind; it is the key to the whole UMN picture. Second, because most motor tracts cross the midline, a *brain* lesion tends to show its signs on the **opposite** side of the body, whereas a *spinal cord* lesion shows them on the **same** side, below the level of the damage. Refining all of this in the background are the **cerebellum**, which constantly compares the movement intended with the movement achieved and corrects the timing, and the **basal nuclei**, which help select the wanted movement and suppress the unwanted — neither touching the muscle except through the lower motor neuron.

07 Signs of upper motor neuron disease — and why they differ

When a UMN lesion strikes, the lower motor neuron below it is still perfectly intact — it has simply lost its commander. So the muscle stays connected, but its control is now unbalanced, and the picture is almost the **mirror image** of LMN disease:

- **Spastic paresis** below the lesion — weak, but **stiff** rather than floppy.
- **Increased tone.** With the descending brake removed, the reflex loops run hot and the limb becomes hypertonic.
- **Normal to exaggerated reflexes (hyperreflexia).** The reflex arc is untouched *and* disinhibited, so it over-responds.
- **Little muscle atrophy.** The LMN still feeds the muscle, so there is no neurogenic wasting — only slow **disuse** atrophy much later.
- **No fasciculations, and a normal EMG** — the muscle is not denervated.

The engine of that whole contrast is **disinhibition**. The UMN normally acts as a **brake** on the spinal reflex; when a lesion removes the brake, the reflex is "released" and becomes exaggerated, and tone climbs. So the very finding — a brisk reflex — that is impossible in LMN disease is a *signature* of UMN disease.

Two clinical wrinkles are worth knowing. In the first hours to days after a severe, acute cord injury the expected hyperreflexia may be absent — a period of **spinal shock** in which reflexes below the lesion are transiently depressed before they recover and become exaggerated; do not let it fool you into diagnosing LMN disease. And a lesion of the T3–L3 cord can produce **Schiff–Sherrington** posture — rigid extension of the *forelimbs* — which looks alarming but is not forelimb UMN disease: the forelimbs retain normal strength and the sign simply reflects loss of ascending inhibition from the damaged region.

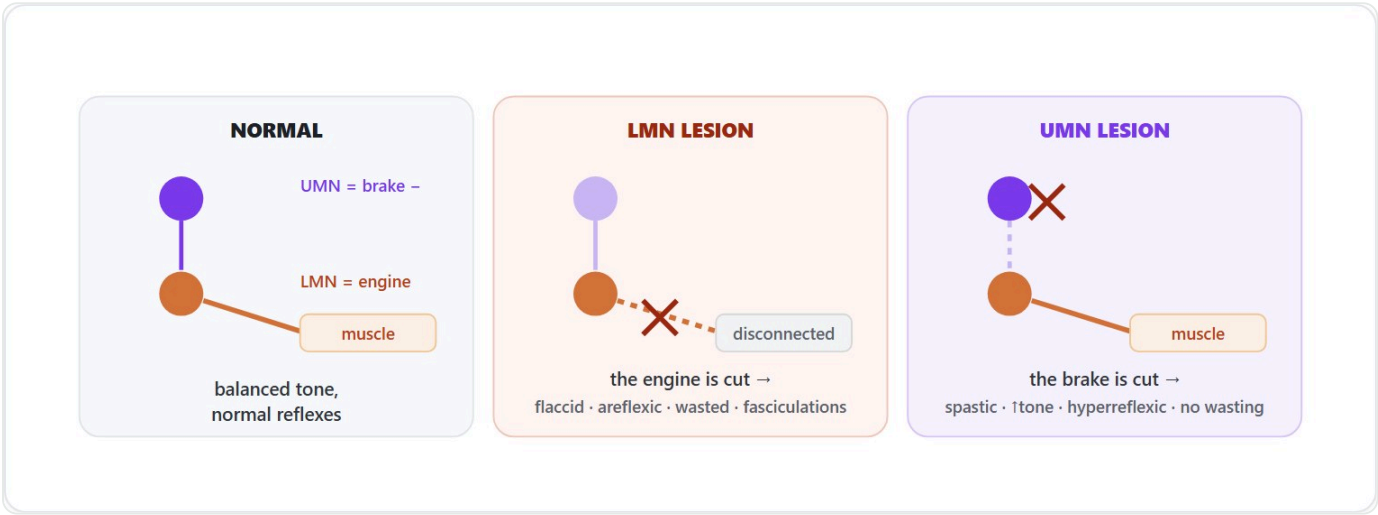


Fig 3 — Why the signs differ. Normally the UMN brakes and the LMN drives the muscle. An LMN lesion cuts the engine (flaccid, areflexic, wasted); a UMN lesion cuts the brake, releasing the reflex (spastic, hyperreflexic, no wasting).

08 Proprioception, ataxia and the bladder — what tone and reflexes miss

Tone and reflexes answer *which motor neuron*. They do not answer three questions that often matter more at the cage side, and the first of them usually gives you the earliest warning of all.

Proprioception fails first. Conscious proprioception runs in the superficial dorsal part of the white matter, which makes those fibres the most vulnerable to external compression. In a slowly compressive lesion — a disc, a tumour (tumor) — the sequence is almost always **proprioceptive deficits, then paresis, then loss of superficial pain, then loss of deep pain**, and it recovers in reverse. That is why **postural reaction testing** (paw positioning, hopping, hemiwalking) is worth more than any single reflex: a dog that knuckles or is slow to replace a turned-over paw has a lesion even though it is still walking. Deep pain, at the other end of the sequence, is the last to go and the one that carries the prognosis.

Ataxia tells you where, not how badly. Ataxia is incoordination *without* weakness, and there are three kinds worth separating. **Proprioceptive (general) ataxia** comes from the spinal cord or dorsal columns: a wide-based, swaying, scuffing gait with delayed postural reactions, and it accompanies UMN signs. **Vestibular ataxia** comes with a head tilt, nystagmus and a tendency to fall or roll to one side. **Cerebellar ataxia** is the odd one out: exaggerated, high-stepping *hypermetria* and an intention tremor, with **strength fully preserved** — the cerebellum modulates movement rather than generating it. A patient that is ataxic but not weak is telling you to look above the cord.

The bladder splits the same way the limbs do, and getting it wrong is dangerous. A lesion *cranial* to the sacral segments produces a **UMN bladder**: the detrusor cannot contract to order while the urethral sphincter keeps or increases its tone, so the bladder becomes large, firm and genuinely difficult to express, and it can over-distend to the point of detrusor damage or rupture if nobody empties it. A lesion in the **sacral segments or cauda equina** produces an **LMN bladder**: flaccid, easy to express, dribbling overflow urine constantly, with a lax anal tone and reduced perineal reflex alongside it. Both need active management, but only one of them will burst.

For vets Tone has names worth using in the record: **hypertonia** with hyperreflexia is the UMN picture, **hypotonia** with hyporeflexia or areflexia is the LMN one. A **crossed extensor reflex** — the opposite limb extending when you pinch a toe — is a UMN sign and is always abnormal in a recumbent patient. In dogs, **intervertebral disc disease (IVDD)** is by far the commonest cause of the T3–L3 UMN picture, and lumbosacral disease the commonest cause of the cauda equina one.

09 UMN vs LMN, side by side

Lay the two lesions next to each other and the whole examination falls into place. Read across the row you are testing — tone, reflexes, muscle bulk — and the pattern points to the culprit.

FEATURE	LOWER MOTOR NEURON LESION	UPPER MOTOR NEURON LESION
Paralysis	Flaccid (floppy)	Spastic (stiff)
Muscle tone	Reduced	Increased
Reflexes	Reduced / absent	Normal to exaggerated
Muscle atrophy	Rapid & severe (neurogenic, days)	Mild & late (disuse only)
Fasciculations	Present	Absent
EMG	Abnormal (denervation) within days	Normal
Localising value	Marks that segment / nerve / root	Signs appear below the lesion

For vets The tidiest rule of thumb: **LMN = everything Lost** (Low tone, Lost reflexes, muscle melts away). **UMN = everything Up** (tone up, reflexes up, muscle preserved). Floppy vs stiff.

10 Localising the lesion by spinal cord region

The reason this split matters so much is that it tells you **where** to look. The lower motor neurons for a given limb live at a specific run of spinal segments, so **LMN signs localise to that segment (or its nerve or root)**. Upper motor neuron signs, by contrast, appear **below** the lesion — so their *upper edge* marks how far up the cord the problem sits. Put the limbs together and the lesion falls out. Clinicians divide the cord into four functional regions, each giving a distinctive pattern:

- **C1–C5** — UMN signs in *all four* limbs (the lesion is above every limb's motor neurons).
- **C6–T2** (the cervicothoracic enlargement, which houses the forelimb motor neurons) — *LMN* signs in the forelimbs but *UMN* signs in the hind limbs.
- **T3–L3** — normal forelimbs with *UMN* hind limbs; this is the commonest region and the home of the classic thoracolumbar disc.
- **L4–S3** (the lumbar enlargement) — *LMN* signs in the hind limbs, often with a flaccid tail and bladder.

The same UMN-versus-LMN logic extends to the bladder, a distinction that changes nursing care. A lesion *above* the sacral segments gives a **UMN bladder** — distended, firm and difficult to express, because the sphincter has lost its descending inhibition. A lesion *of* the sacral segments or their nerves gives an **LMN bladder** — flabby, easily expressed and prone to overflow leakage. Reading the bladder alongside the limbs sharpens the localisation still further.

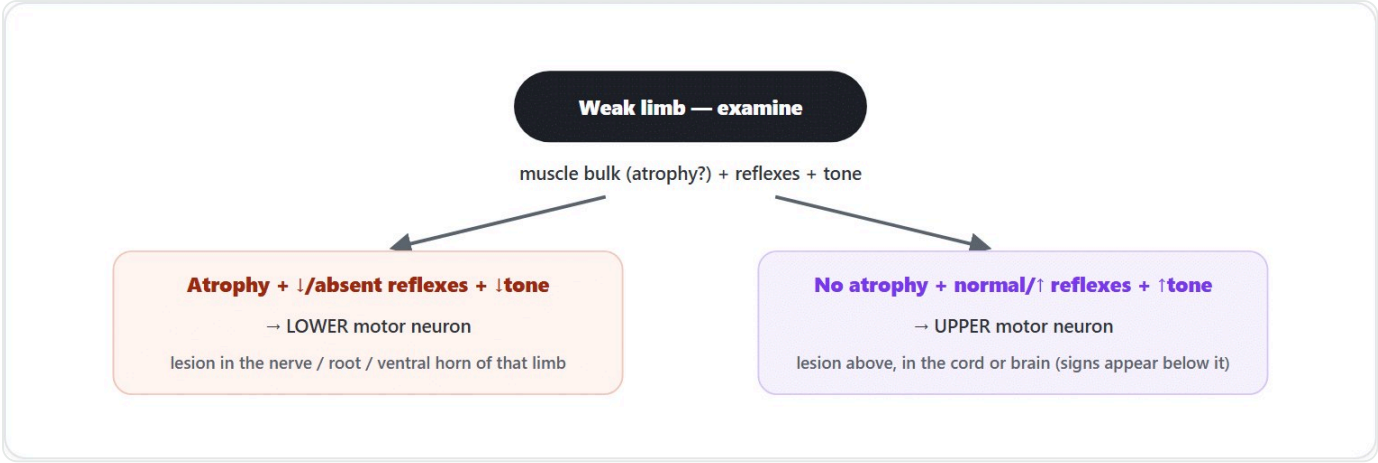


Fig 4 — The localisation shortcut: at a weak limb, check muscle bulk, reflexes and tone. Atrophy with lost reflexes points to the lower motor neuron of that limb; no atrophy with preserved or exaggerated reflexes points to an upper motor neuron lesion higher up.

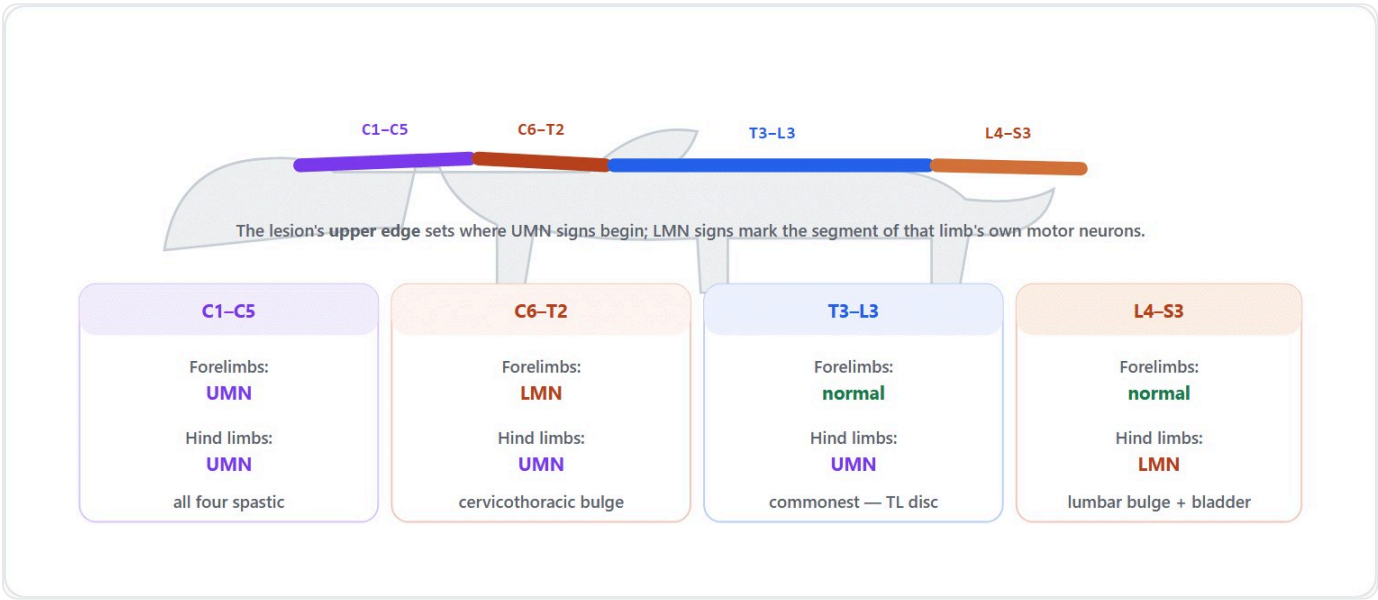


Fig 5 — Localising by cord region. The limb pattern maps to one of four regions: C1–C5 (all four UMN), C6–T2 (LMN forelimbs, UMN hind), T3–L3 (normal forelimbs, UMN hind), and L4–S3 (LMN hind limbs).

11 The other two sites: neuromuscular junction and muscle

Remember that a weak limb has **four** possible sources, not two. Beyond the UMN and LMN, disease of the **neuromuscular junction** or of the **muscle** itself can weaken an animal, and both tend to mimic an LMN picture — weakness, sometimes with reduced tone — so they must be actively ruled in or out.

At the **neuromuscular junction**, **myasthenia gravis** (autoimmune loss of acetylcholine receptors) causes weakness that *fatigues* with use and recovers with rest; **botulism** and **tick paralysis** block transmitter release and cause a flaccid, ascending, LMN-like paralysis; and in ruminants profound **hypocalcaemia** (milk fever) impairs junctional transmission enough to cause recumbency. Primary diseases of the **muscle** — the inflammatory and inherited myopathies, including the dystrophin muscular dystrophy seen in some dogs — weaken the effector organ directly.

The examination separates these sites with a few simple tests. If a nerve will not drive a muscle but **direct electrical stimulation of the muscle still makes it contract**, the fault lies in the nerve or the junction, not the muscle. If a suspected junctional weakness **improves with an anticholinesterase drug** (edrophonium), that points to the neuromuscular junction, as in myasthenia gravis. And the **EMG** distinguishes the denervation pattern of nerve disease from the changes of a primary myopathy. Fitting these two extra sites into the checklist is what turns "the leg is weak" into a specific anatomical diagnosis.

12 Putting it together — the clinical workflow

The workflow, then, is simple and powerful. Look at the limb and judge whether it is **floppy or stiff**; test the **reflexes** and note whether they are depressed or brisk; assess **muscle bulk** for rapid atrophy; combine the limbs to place the lesion in the cord; and, where the picture could be junction or muscle, run the tests that separate them. From four vague possibilities you arrive at a region of the nervous system, often before any imaging.

Two textbook cases make it concrete. A dog with **ascending flaccid paralysis of all four legs, widespread atrophy and absent reflexes but intact deep-pain sensation** has diffuse **LMN** disease — classically **polyradiculoneuritis ("coonhound paralysis")**, a lesion of the ventral nerve roots; deep pain is preserved because the dorsal (sensory) roots are spared. A dachshund with **weak hind legs, no atrophy, normal-to-brisk reflexes and normal front legs** has a **UMN** lesion in the T3–L3 cord — the classic **thoracolumbar disc herniation**. Same examination, opposite patterns, different part of the animal — and a different plan for each.

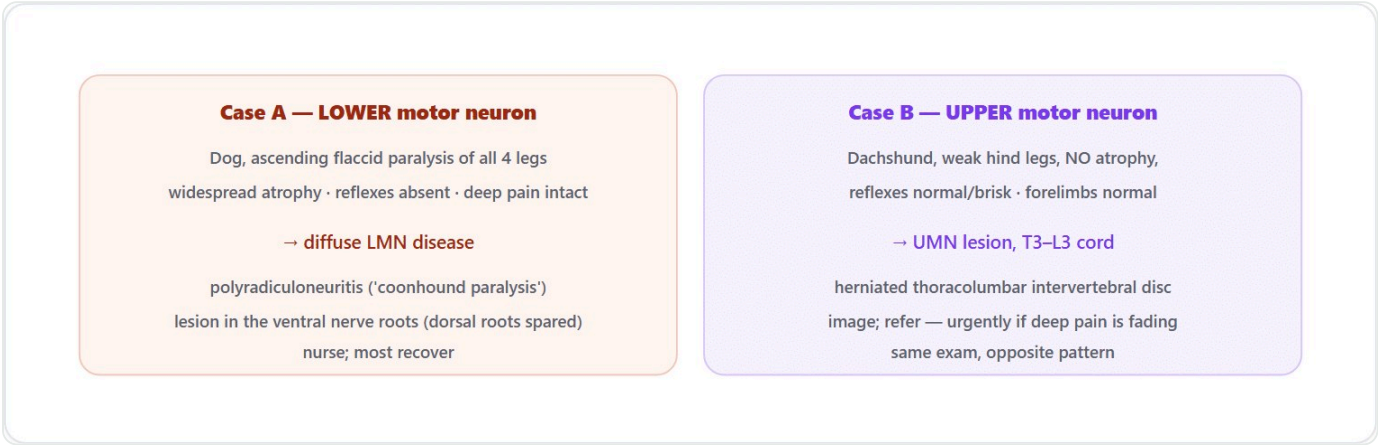


Fig 6 — Two textbook localisations: diffuse LMN disease (coonhound paralysis, ventral roots) versus a focal UMN cord lesion (a dachshund thoracolumbar disc) — read straight off tone, reflexes and atrophy.

Red flag

Rapidly progressive paralysis, loss of deep-pain sensation, or failing respiration (diffuse LMN disease such as polyradiculoneuritis, tick paralysis or botulism, or a severe cervical UMN lesion) is a neurological emergency — stabilise, protect ventilation, and refer without delay.

13 Quiz: test yourself

Twenty-one questions across the final common path, tone and reflexes, the two lesion patterns, localisation by cord region and the junction and muscle. Answers are beneath each question — cover them and work down.

1. A limb is weak. At which four places can it be failing?**Answer:** The **upper motor neuron**, the **lower motor neuron**, the **neuromuscular junction**, or the **muscle** itself.2. Why is the lower motor neuron called the final common path?**Answer:** Because **every** influence on that muscle — spinal reflex, voluntary command, postural adjustment — must funnel through this one neuron to reach it. There is no other route.3. What is a motor unit, and what is true of its fibres?**Answer:** One motor neuron and all the fibres it supplies. They contract **together**, and all fibres in a unit are the **same functional type**.4. What produces resting muscle tone?**Answer:** It is **active**, not passive: **slow (type I) motor units firing at a low, almost constant frequency**, set by reflex machinery in the cord and adjusted by descending pathways.5. What does the Golgi tendon organ reflex do?**Answer:** The **inverse myotatic reflex** is protective — it prevents a contraction becoming forceful enough to **tear the tendon from bone**.6. Why does an LMN lesion make a limb flaccid?**Answer:** Tone depends on a working reflex loop and on **tonic firing of the α motor neuron**. Break that neuron and the loop collapses, so the limb goes limp and offers no resistance to passive movement.7. Why does an LMN lesion sometimes cause paresis rather than paralysis?**Answer:** If only **some** of a nerve's axons are lost — or the muscle is served by **more than one spinal nerve** — enough supply survives to leave weakness rather than complete loss.8. Where do most upper motor neuron fibres cross, and in what proportion?**Answer:** At the **pyramidal decussation** — about **75% in the dog**, up to **90% in primates**.9. Why does a lesion above the decussation give only subtle signs in most veterinary species?**Answer:** Because the corticospinal tract is **comparatively minor** in them. You get a proprioceptive placing deficit or a dragging toe, not a dense hemiparesis.10. A large part of what the descending system does is inhibit. What does that explain?**Answer:** It **holds the spinal reflexes in check** — which is why an animal can consciously override a withdrawal reflex, and why removing the brake makes reflexes run hot.11. Why is a limb below a UMN lesion hypertonic with brisk reflexes?**Answer:** The lower motor neuron is **perfectly intact** — it has simply lost its commander. With the descending

brake removed the reflex loops run hot.12. What is the rule of thumb that separates the two patterns?**Answer: LMN = everything Lost** — low tone, lost reflexes, muscle melts away. **UMN = everything Up** — tone up, reflexes up, muscle preserved.13. How does muscle atrophy differ between the two?**Answer:** LMN gives **rapid, severe neurogenic atrophy within days**. UMN gives only **mild, late disuse atrophy**.14. Which pattern shows fasciculations?**Answer: LMN**. They are absent in UMN disease.15. Why is postural reaction testing worth more than any single reflex?**Answer:** Tone and reflexes answer **which** motor neuron; postural reactions detect the lesion at all. A dog that knuckles or is slow to replace a turned-over paw has a lesion **even though it is still walking**.16. What does proprioceptive ataxia look like, and what accompanies it?**Answer:** A **wide-based, swaying, scuffing** gait with **delayed postural reactions**, from the spinal cord or dorsal columns — and it accompanies **UMN signs**.17. Why do LMN signs localise more precisely than UMN signs?**Answer:** The lower motor neurons for a limb live at a **specific run of spinal segments**, so LMN signs point to that segment, nerve or root. UMN signs appear **below** the lesion, so only their **upper edge** marks how far up the cord it sits.18. What pattern does a C1–C5 lesion give?**Answer: UMN signs in all four limbs** — the lesion sits above every limb's motor neurons.19. A dachshund has weak hind legs, no atrophy, normal-to-brisk reflexes and normal front legs. Where is the lesion?**Answer:** A **UMN lesion in the T3–L3 cord** — the classic thoracolumbar disc herniation.20. A dog has ascending flaccid tetraparesis, widespread atrophy, absent reflexes and intact deep pain. What is this?**Answer: Diffuse LMN disease** — classically **polyradiculoneuritis** ("coonhound paralysis"), a lesion of the ventral nerve roots.21. How do you separate a nerve or junction problem from a primary myopathy? **Answer:** If a nerve will not drive a muscle but **direct electrical stimulation of the muscle still contracts it**, the fault is in the nerve or junction. **EMG** distinguishes the denervation pattern from the changes of a myopathy.

14 Upper vs lower motor neuron signs — frequently asked questions

What is the difference between an upper and a lower motor neuron?

The lower motor neuron (the α motor neuron) runs from the CNS out to the muscle — the "final common path." The upper motor neuron stays entirely within the brain and spinal cord and controls the lower motor neuron; it never reaches muscle directly.

How do you tell a UMN from an LMN lesion?

By tone, reflexes and muscle bulk. LMN lesions give flaccid weakness, reduced or absent reflexes and rapid severe atrophy. UMN lesions give spastic weakness, normal-to-exaggerated reflexes and little atrophy.

Why are reflexes exaggerated in upper motor neuron disease?

Because the upper motor neuron normally inhibits (brakes) the spinal reflex. A UMN lesion removes that inhibition, so the reflex is "released" and over-responds — hyperreflexia — while tone rises.

What is the difference between a UMN bladder and an LMN bladder?

A **UMN bladder** follows a lesion cranial to the sacral segments: the detrusor will not contract but the urethral sphincter keeps its tone, so the bladder is large, firm and hard to express, and it is at real risk of over-distension. An **LMN bladder** follows a sacral or cauda equina lesion: it is flaccid, easy to express and leaks overflow urine continuously, usually with a lax anal tone. Both require the bladder to be emptied for the patient; the UMN one is the more urgent because it can rupture.

What is the earliest sign of a spinal cord lesion?

Loss of **conscious proprioception**, in almost every compressive lesion. The proprioceptive fibres sit superficially in the white matter and are squeezed first, so knuckling and delayed paw placement appear while the animal is still walking. The order of loss is proprioception, then motor function, then superficial pain, then deep pain — and function returns in the reverse order, which is why deep pain carries the prognosis.

What is the commonest cause of UMN signs in dogs?

Intervertebral disc disease. A Hansen type I extrusion in a chondrodystrophic breed — dachshund, French bulldog, beagle — typically hits the T3–L3 segment and produces the classic picture: normal or exaggerated pelvic limb reflexes, increased tone, no rapid atrophy, and thoracic limbs that are entirely normal. Trauma, fibrocartilaginous embolism and neoplasia produce the same UMN pattern from the same region.

Can I download these UMN vs LMN notes as a free PDF?

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When to refer

- 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.

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