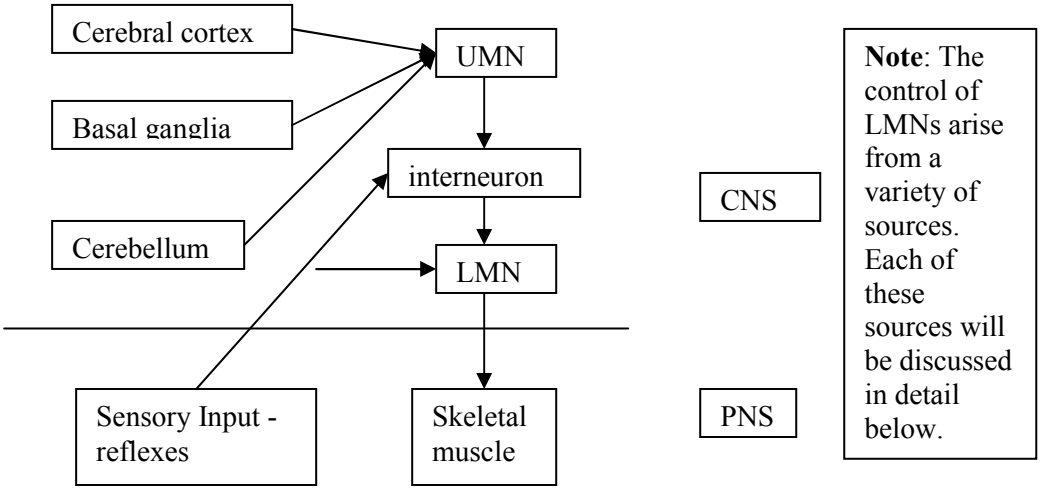
Lecture 19 – Somatic motor systems

Somatic (voluntary) motor systems



Motor unit: one LMN and its muscle fibres (Nolte 5th Ed pp 449)
A motor unit is defined as a combination of 1 LMN and all of the muscle fibres it innervates (i.e.: axon terminal branches innervate 1 muscle fibre). There are 3 types of muscle fibres, each corresponding to a particular function. Red fibres have an abundance of mitochondria – contract weakly and slowly – but as able to do so for long periods of time. Fast fatigue resistant fibres can contract fast and can do so for moderate lengths of time. Fast fatigueable (white) are larger fibres that have little mitochondria and can only contract for short periods of time.

All of the muscle fibres in a single motor unit are of the same type. There are about 10-10,000 fibres per motor unit – depending on the level of adjustment required.

Neuromuscular junctions (Notes)
All LMNs use ACh as their neurotransmitter. Once released from the terminal axons, they bind to nicotinic ACh receptors on skeletal muscle fibres. These receptors are ligand-gated ion channels, meaning – once the neurotransmitter binds to it – then it activates the opening of Na+ and K+ channels. What happens once the neurotransmitters have bound to their receptors? If the ACh is still present, then it will cause unlimited skeletal muscle fibre contraction. Therefore, in the synaptic cleft there is an enzyme called acetylcholinesterase, which breaks down ACh therefore stopping further AP's from being generated.

Lower motor neurons in spinal cord (Nolte 5th Ed pp 449)
A thing to note is that LMNs are located in the spinal cord and in the brain stem (as part of the cranial nerve nuclei). LMNs in spinal cord are located in the ventral horn, where axons leave in ventral roots. The ventral roots join the dorsal roots just distally from the spinal cord to become spinal nerves, which exit via the IV foramen. The arrangement of LMNs in the ventral root is one of interest. LMNs innervating proximal muscles are located medially. LMNs innervating flexors are located more dorsally.

LMNs in spinal cord always innervate muscles on the **ipsilateral side.**
LMNs in the brainstem (Notes)
LMNs are located in cranial nerve nuclei associated with III, IV, V, VI, VII, IX, X, XI, XII. Note **CN VIII** is missing (i.e.: largely sensory nerve). All of the LMNs arising from these cranial nerve nuclei innervate ipsilateral muscles with **one exception** (i.e.: LMNs arising in trochlear nucleus innervate contralateral superior oblique muscle).

<u>Control of LMNs</u>

Control of LMNs via sensory input → Reflexes

Reflexes: muscle stretch reflex (Nolte 5th Ed pp 234 Fig 10-9)
LMNs can, to an extent, be controlled by reflex pathways – mediated by sensory input. The muscle stretch reflex is the simplest of these reflex pathways. It is a monosynaptic reflex pathway – utilising an afferent limb directly synapsing onto an efferent limb. The primary sensory neuron (afferent limb) is a Ia fibre that is stimulated upon muscle stretch (detects muscle spindly activity) – and the impulse travels through the dorsal horn and synapses onto an alpha motor neuron located in the ventral root – which elicits a motor response.

Reflexes: withdrawal reflex (Nolte 5th Ed pp 235 Fig 10-11)
Painful stimuli elicit a withdrawal reflex. This type of reflex is initiated by cutaneous receptors and involve the whole limb. This type of reflex is constantly held in an inhibited state by descending influences such that only noxious stimuli produce the reflex. 1) Since an entire limb is involved – then the sensory input to the spinal cord is over several spinal segments – so that different flexor muscle groups are excited. 2) Interneurons are involved in this reflex pathway – hence it's a polysynaptic reflex pathway.

Primary sensory neurons pick up noxious stimuli → travel to spinal cord → ascend/descend along funiculi of spinal cord → enter dorsal horn at appropriate levels → synapse with interneurons → synapse with alpha motor neurons → elicit muscle contraction. Note reciprocal inhibition also occurs (i.e.: excitatory interneurons synapse with alpha motor neurons, but inhibition interneurons synapse with alpha motor neurons → reciprocal inhibition). Also note: interneurons may travel to contralateral side (crossed effects) to excite muscles of other side → therefore able to balance ourselves (reciprocal inhibition also involved here).

Interneurons (Notes)
Interneurons can be inhibitory/excitatory. They receive input directly from primary sensory neurons, and also from UMNs → pattern generators (cerebral cortex, basal ganglia, cerebellum etc). They are located in the ventral/dorsal horn.

Control of LMNs via pattern generators → UMN systems
Pattern generators (Nolte 5th Ed pp 452)
These are networks of interneurons in the brain stem and spinal cord that act as pattern generators for rhythmic movements such as breathing & walking.

Different descending pathways (tracts) can influence LMNs
UMN systems influencing spinal LMNs (Netter Plate 151)
So far we have covered reflexes and pattern generators as inputs to LMNs. What about UMN systems (descending pathways)? Yes, these are main influences originating from the cerebral cortex and include the following tracts:

- Corticobulbar, Corticospinal, Rubrospinal, Tectospinal, Recticulospinal (pontine & medullary) & Vestibulospinal tracts (medial/lateral).

Locate these tracts in Netter Plate 151 and determine which funiculi they are in.

Location of UMN tracts in the spinal cord (Netter Plate 151)
Lateral column: lateral CST, rubrospinal tract
Anterior column: medullary reticulospinal tract, vestibulospinal tract, tectospinal tract, pontoreticulospinal tract, anterior CST

UMN systems: Corticospinal tract (Nolte 5th Ed pp 248/455 Fig 10-22)
The cell bodies of primary motor neurons are located in the primary motor cortex (& **nearby areas)**. Cell bodies are usually located more medially in this cortical area. Fibres descend through the posterior limb of the internal capsule → cerebral peduncle in the mid brain → basal pons → medullary pyramids. Here, about 85% of fibres cross the midline in the decussation of pyramids to the lateral funiculus of spinal cord. This becomes the **lateral corticospinal tract**. About 15% of the fibres do not cross the midline at caudal medulla but continue to descend ipsilaterally in the anterior funiculus of spinal cord as **anterior corticospinal tract** and cross the midline (anterior white commissure) at the same level as interneurons/LMNs they supply.

CORTICOSPINAL TRACT PATHWAY

UMN systems: Corticobulbar (corticonuclear) tract (Nolte 5th Ed pp 461 Fig 18-17)
The term corticobulbar refers to cortex → brainstem. This tract is commonly used to describe the pathway taken by motor fibres innervating the cranial nerve nuclei. Most of these tracts synapse on interneurons in the reticular formation, which consequently synapse on motor neurons.

The oculomotor, trochlear and abducens nuclei receive no input from the corticobulbar tract. Thus the corticobulbar tract influences: trigeminal, facial, hypoglossal motor nuclei, nucleus ambiguus, and spinal accessory nucleus.

The cell bodies of primary motor neurons are located in primary motor cortex. Cell bodies are generally located more laterally in this cortical area. Fibres travel through the genu of the internal capsule (immediately anterior to CST) → cerebral peduncle → basal pons (give off fibres to both sides) → medulla (give off fibres to both sides). Generally, the corticobulbar tract reaches the level of the nucleus it is meant to innervate and gives off bilateral branches innervating the nuclei on both sides. The major exception to this is the innervation of facial motor nucleus. **i.e.:** motor neurons innervating lower ½ - 2/3 of face receive input from contralateral corticobulbar tract, while motor neurons innervating upper 1/3-1/2 face receive bilateral input from corticobulbar tract.

CORTICOBULBAR TRACT PATHWAY

UMN systems: Origin of corticospinal and corticobulbar tracts (Nolte 5th Ed pp 457 Fig 18-11/12)
Only about a 1/3 of the corticospinal fibres arise in the primary motor cortex (precentral gyrus of frontal lobe). The remainder of the fibres come from adjacent areas (i.e.: primary somatosensory cortex – post-central gyrus).

Note that the primary motor cortex is not the only one involved in motor control, other areas such as: premotor cortex (directly anterior to primary motor cortex) and supplementary motor cortex (medial surface of hemisphere – just anterior to primary motor cortex representation of foot – Fig 18-11).

Damage to cortical UMNs or their axons: motor cortex or CST/CBT damage (Notes, Nolte 5th Ed pp 460-461)
Corticospinal system
Any damage to motor cortex (involving CST) or axons of CST means → symptoms on contralateral side of body if lesion is above decussation of pyramids (i.e. CST is before decussation). If below, then symptoms ipsilateral to lesion.

Corticobulbar system
The corticobulbar tract supplies motor neurons of CN nuclei bilaterally (except for motor neurons supply lower ½-2/3 of face – receives input form contralateral corticobulbar tract). Thus any lesion to this tract will mean – there will be mild bilateral symptoms (i.e.: only mild symptoms because corticobulbar tract of other side also inputs bilaterally). In addition – the lower ½-2/3 of face contralateral to side of lesion will be paralysed (i.e.: no bilateral innervation).

UMN systems: Rubrospinal tract (Nolte 5th Ed pp 452 Fig 18-6)
The rubrospinal tract is one of the principal alternate routes for the mediation of voluntary movement. Primary motor cell bodies located in red nucleus (rostral midbrain) – axons cross to other side of midbrain (i.e.: cross midline) immediately → descends in lateral pons, medulla → travels in lateral funiculi of spinal cord alongside lateral CST → synapse with interneurons/LMNs.

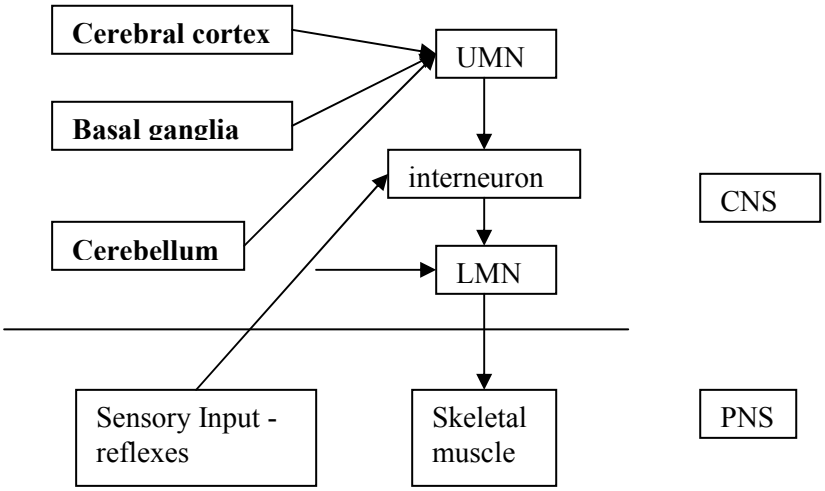
UMN systems: Tectospinal tract (Nolte 5th Ed pp 453 Fig 18-6)
Primary motor cell bodies are located in the superior colliculus, where their axons cross (midbrain). Fibres travel in the medial longitudinal fasciculus → medial pons → medulla → descend along the anterior funiculi of cervical spinal cord where LMNs for this system are located. The tectospinal tract is thought to be involved in reflex turning of the head in response to visual/sound stimuli.

UMN systems: Vestibulospinal tracts (Nolte 5th Ed pp 361 Fig 14-28)
Cell bodies are located in the vestibular nuclei, which is also involved in vestibular functions (as discussed in earlier lecture). Axons of these cells descend into the spinal cord, travelling in the ipsilateral anterior funiculi. There are two main pathways: the LVST & MVST. The LVST arises in the lateral vestibular nucleus and sends its axons ipsilaterally to interneurons/motor neurons located in the ipsilateral ventral horn of spinal cord. The MVST arises in the medial vestibular nucleus and sends its axons to both sides → these travel with MLF to supply interneurons/motor neurons on both sides.

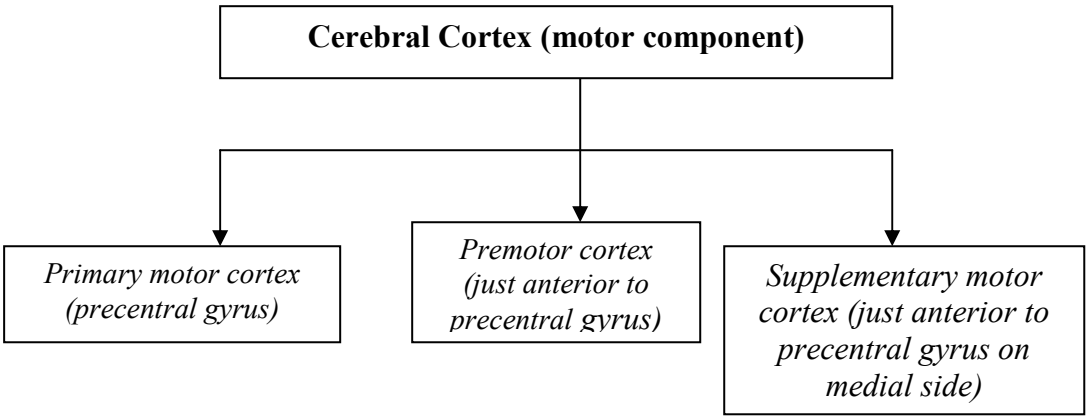
These tracts are responsible for maintaining/adjusting posture in the event of head/body tilts.

UMN systems: Reticulospinal tracts (Nolte 5th Ed pp 276, Fig 11-16)
Cell bodies are located in the pontine and medullary reticular formation. Pontine fibres descend ipsilaterally with MLF; medullary fibres descend ipsilaterally in the anterior funiculi. Axons travel to cervical spinal cord – sometimes even more caudally. The reticulospinal tracts are the major alternate route to voluntary movement, and also regulate sensitivity of spinal reflexes.

Control of UMN systems (Notes, above diagram, Nolte 5th Ed pp 456 Fig 18-11)



As you can see, from the above diagram, the influences of LMNs are from various UMN tracts, already discussed. What influences UMN systems? These influences come from cerebral cortex, cerebellum, and basal ganglia. The latter two will be discussed in coming lectures. The motor component of cerebral cortex can be divided into the following:



Decorticate rigidity (Notes)
Refer to Derek's powerpoint slides for picture of decorticate rigidity. Lower limbs are fully extended & upper limbs are fully flexed. This posture occurs as a result of loss of corticospinal tract, loss of cortical inputs to UMN systems, and loss of cerebellar input.

Decerebrate rigidity (Dorlands Medical Dictionary – under 'rigidity')
As defined: "rigid extension of animals legs as a result of decerebration. In humans, it presents due to lesion in the upper brainstem". De-cerebration means – taking out the cerebral input. In humans this would occur if there is midbrain lesion. The vestibulospinal and reticulospinal tracts are intact (i.e.: only begin at pons/medulla area). Corticospinal/Rubrospinal/Tectospinal tracts are lost (i.e.: these arise at midbrain or above level). All cortical and cerebellar inputs to UMN systems are lost. Upper and lower limbs extended.

Damage to LMNs vs UMNs (Notes)
Symptoms to LMN damage:

- Severe atrophy of muscles (i.e.: LMN supply trophic factors to skeletal muscles, loss of LMN means no trophic factors → muscle wasting)
- Weakness / paralysis (i.e.: due to inability to move freely, sometimes cannot move at all)
- Hyporeflexia or areflexia (i.e.: reflexes are mediated by LMNs innervating muscle groups – absence of this input means little or no reflex)

- Hypotonia (i.e.: muscle is always maintained via LMN input, loss of input means loss of tone)

Symptoms of UMN damage:

- Some / no atrophy of muscles (i.e.: LMN intact, but not excited as often due to UMN damage)
- Spasticity (i.e.: sudden, violent, involuntary muscular contraction – due to ↓ UMN input)
- Hypereflexia (i.e.: loss of UMN input, means LMN synthesis more receptors for ACh → more chances of reflexes occur)
- Hypertonia (i.e.: ↑ resistance to movement, maybe because tonic inhibition no present from UMN systems)

Some diseases affecting LMNs and/or UMNs (Notes)

Various diseases can affect function of LMNs and UMNs. These are: strokes (ischaemia, haemorrhagic), tumours (compression of tracts), polio (polio virus infects LMNs of anterior horn), MS (autoimmune reactions towards myelin protein – affects CNS tracts), motor neurone disease / amyotrophic lateral sclerosis (lesion of lateral CST)

What happens to muscle when LMNs die? (Notes)

Remember 1 motor unit = 1 LMN and all muscle fibres it innervates. So if single LMN dies – then you would expect that set of muscles to become paralysed. But, what actually happens is that the remaining LMN take over the innervation of these muscles.

Remember that a LMN innervates a selected type of muscle fibres (slow/red, fast fatigue resistant, fast fatigable/white). In the above case, as the remaining LMN take over – they convert the previously denervated muscle to its own type (i.e.: “its own” = LMN’s own).

LMN lesions results is fibrillations (i.e.: spontaneous firing of motor units) & fasciculations (i.e: spontaneous muscle contraction).