Mind, Brain & Behaviour 1
Behavioural Neuroscience

The sensorimotor system

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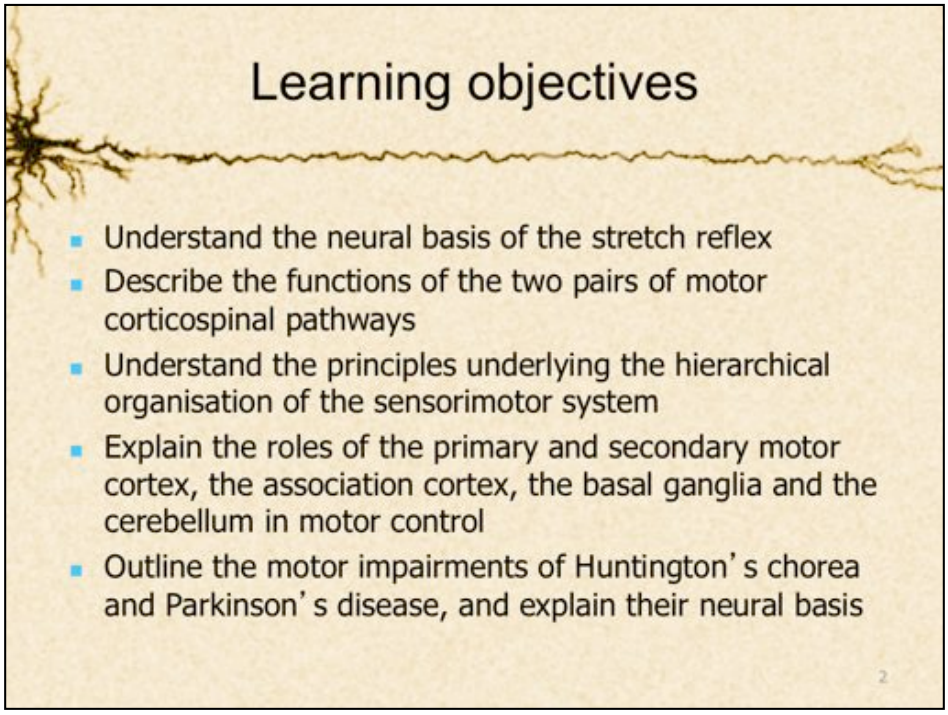
Learning objectives
Understand the neural basis of the stretch reflex
Describe the functions of the two pairs of motor corticospinal pathways
Understand the principles underlying the hierarchical organisation of the sensorimotor system
Explain the roles of the primary and secondary motor cortex, the association cortex, the basal ganglia and the cerebellum in motor control
Outline the motor impairments of Huntington's chorea and Parkinson's disease, and explain their neural basis
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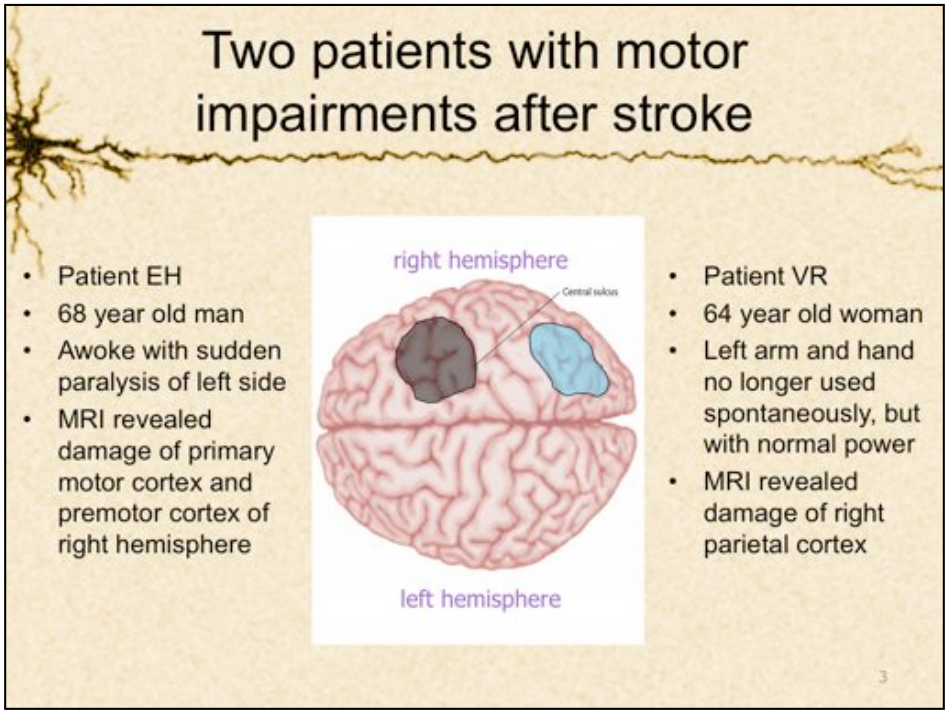


For most of us the ability to walk and move our limbs voluntarily is something we take for granted, partly because these activities seem to occur automatically, without the need for conscious control.

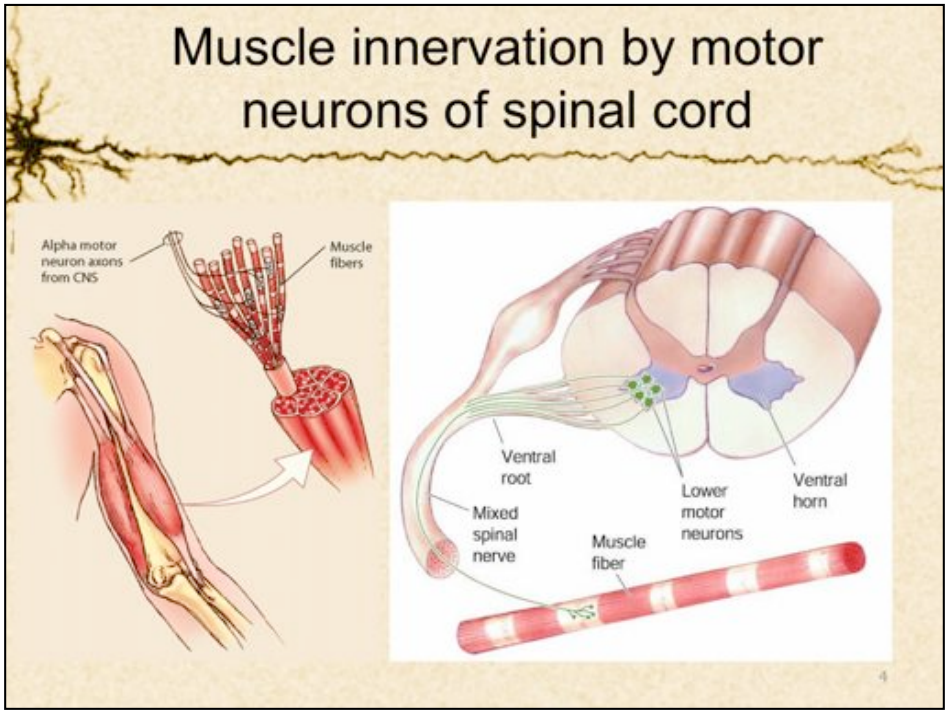
It is not until something goes wrong with the system that we realise how important movement is for everyday behaviour.

Let us consider two patients seen by a clinical neuropsychologist, both of whom had profound losses of **motor control** (i.e., the control of body movement) for limbs on the left side of their body.

Patient EH is a 68 year old man who awoke one morning to find he could not move his left arm or leg. He was admitted to hospital, at which time the examining neurologist guessed that EH had suffered a stroke. The neurological examination showed that even though EH could not make voluntary movements with his left limbs, he had preserved stretch reflexes (in fact his reflexes on the left were even stronger than those on the right). An MRI scan of EH's brain revealed a stroke-induced lesion of the primary motor cortex and premotor cortex of the right hemisphere.

Patient VR is a 64 year old woman who one day stopped using her left arm during her daily activities. Her daughter found her struggling to open a jar with her right hand, and noticed that her left hand was dangling limply by her side. When she suggested to VR that she hold the jar steady with her left hand, her mother looked down by her side with a surprised look, raised her left hand, grabbed the jar and promptly twisted the lid off. During the day there were many similar incidents. It was as if VR had forgotten that she had a left arm at all, even though when her attention was drawn to it she seemed able to use it normally. Frightened by these strange symptoms, VR's daughter took her mother to hospital. After undergoing an MRI scan, a large lesion was found in VR's right parietal lobe, the result of a recent stroke.

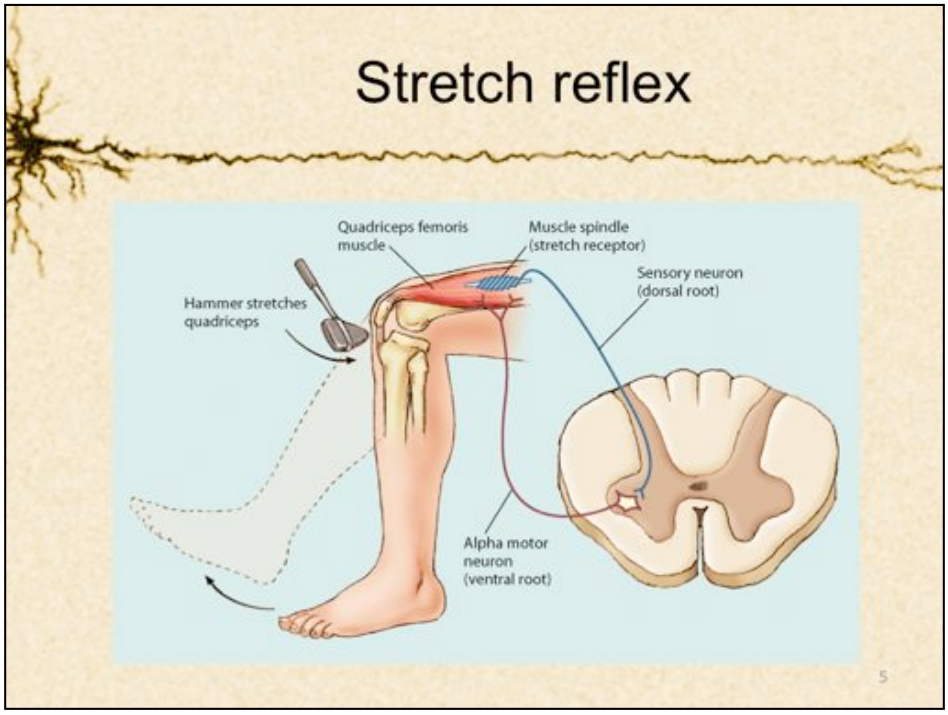
These two cases illustrate two different failures of motor control. To understand the basis for these failures, we need to consider which parts of the human CNS are involved in sensorimotor control, and how these areas perform the complex processes involved in motor control.



Movements are controlled by muscles that contract in response to neural signals from efferent **motor neurons** projecting from the spinal cord. Motor neurons exit the spinal cord via the **ventral root** and terminate on individual muscle fibres. An action potential in a motor neuron triggers the release of a neurotransmitter, **acetylcholine**, which stimulates muscle fibres to change their length accordingly.

A single motor neuron typically innervates many muscle fibres; when the motor neuron fires all the muscle fibres contract together. The group of fibres innervated by a single motor neuron is called a **motor unit**. Motor units with the fewest muscle fibres, such as those in the face and hands, permit the greatest degree of selective motor control.

Many skeletal muscles fall into one of two categories: **extensors** and **flexors**. Flexors act to bend or 'flex' a joint, whereas extensors act to straighten it. These two categories of muscle often act antagonistically (i.e., in opposition), as is the case for the biceps and triceps muscles in the upper arm.

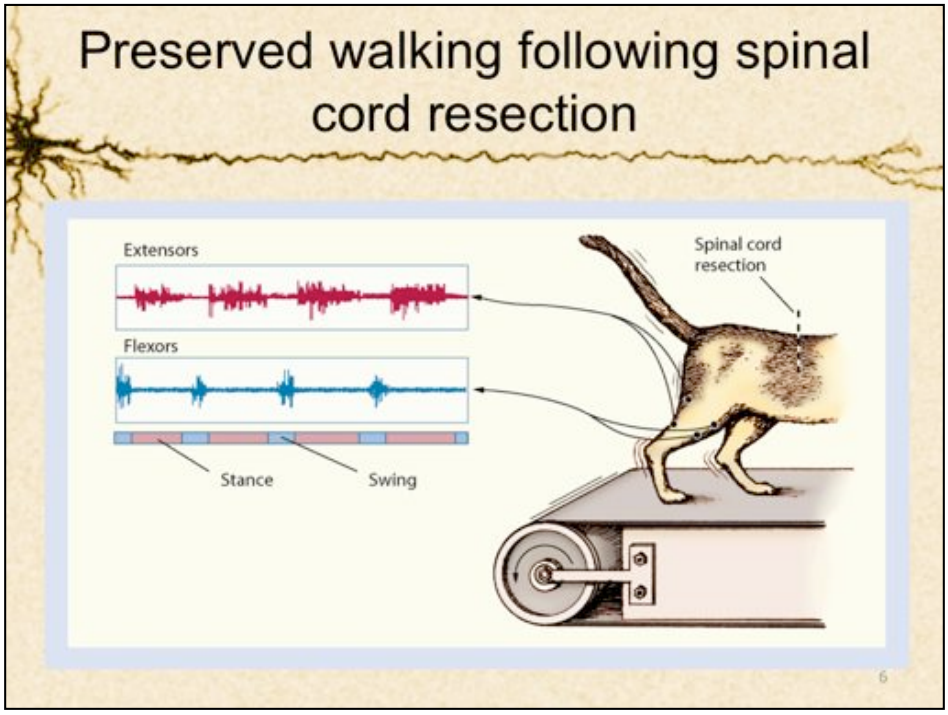


Spinal motor neurons receive input from a variety of sources. One such source is the sensory receptors located within the muscles themselves. The activity of skeletal muscles is monitored by receptors called **muscle spindles**. These provide information to the CNS regarding muscle length.

When a muscle is unexpectedly stretched, as occurs when a hammer is used to tap beneath the patella (kneecap), the muscle spindles convey information back to the spinal cord via the dorsal roots. The axons of **spindle afferent neurons** synapse directly with the motor neurons, which increase their activity in order to return the muscle to its original length. This results in a brisk contraction of the quadriceps muscle, which causes the lower leg to extend. This circuit forms a simple **reflex arc**.

When a doctor elicits the patellar tendon reflex just described the effects are readily noticeable. But the functional significance of the reflex is more subtle. The role of such simple stretch reflexes is to compensate for any perturbation by external forces and thus maintain the intended position of the body. Thus, for example, when someone bumps into you from behind or brushes your arm while you're carrying a hot cup of coffee, the stretch reflex compensates automatically and prevents you from falling over or spilling your drink.

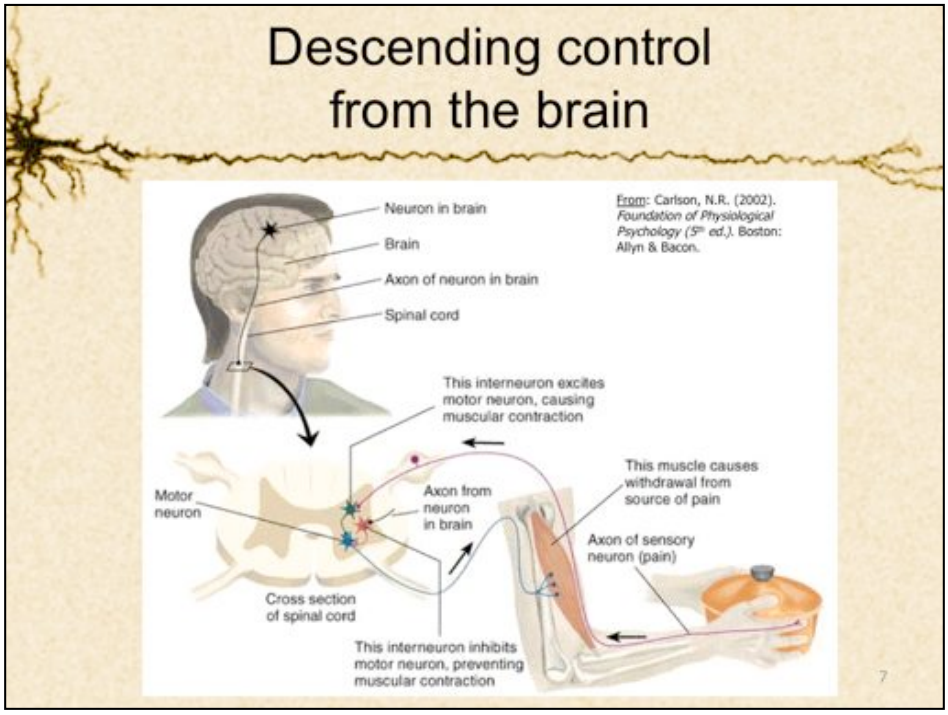
The patients EH and VR, introduced at the beginning of the lecture, both had strong patellar tendon reflexes, indicating that the motor and sensory neurons of the spinal cord remained intact.



Motor neurons in the spinal cord are capable of triggering quite complex movements of various muscle groups, without any controlling signals from the brain. This has been illustrated in experiments with cats, in which the spinal cord is surgically sectioned at a point just above where the spinal nerves subserving the hind legs are located. This effectively disconnects the lower motor neurons for the hind legs from the brain.

Despite the spinal cord section, the cats are still able to walk normally when placed on a treadmill, showing normal extensor and flexor movements of the hind legs.

This is a dramatic illustration of the **hierarchical organisation** of the motor system. Motor and sensory neurons within the spinal cord are able to control all of the complex patterns of muscle contraction required for walking, without any instructions from the brain. This leaves the brain free to control the more demanding aspects of motor control, such as determining precisely when to initiate particular actions, which effectors to use, and how to tailor movements to the specific environment in which the organism finds itself.

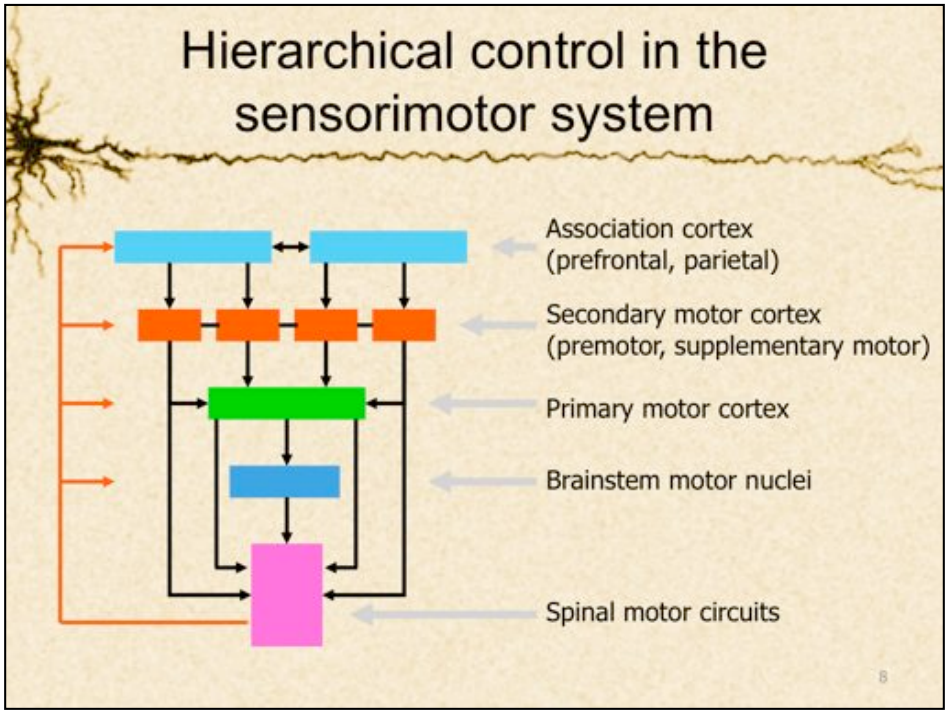


Of course, most purposeful actions are initiated and controlled voluntarily, and such actions depend upon signals generated by the brain that are conveyed to the muscles via the spinal cord.

You will recall from Lecture 2 that reflexes can be modulated by control signals from the brain.

In the example of carrying a hot casserole dish, the tendency to want to drop the dish comes from excitatory synapses on motor neurons in the spinal cord. But this excitation can be counteracted by inhibitory input from the primary motor cortex in the brain. The axons that descend from the primary motor cortex through the spinal cord form inhibitory synapses with lower motor neurons. These inhibitory synapses can prevent a muscle contraction from occurring by blocking action potentials in lower motor neurons. Similarly, excitatory inputs from the brain can trigger action potentials in lower motor neurons and initiate movements.

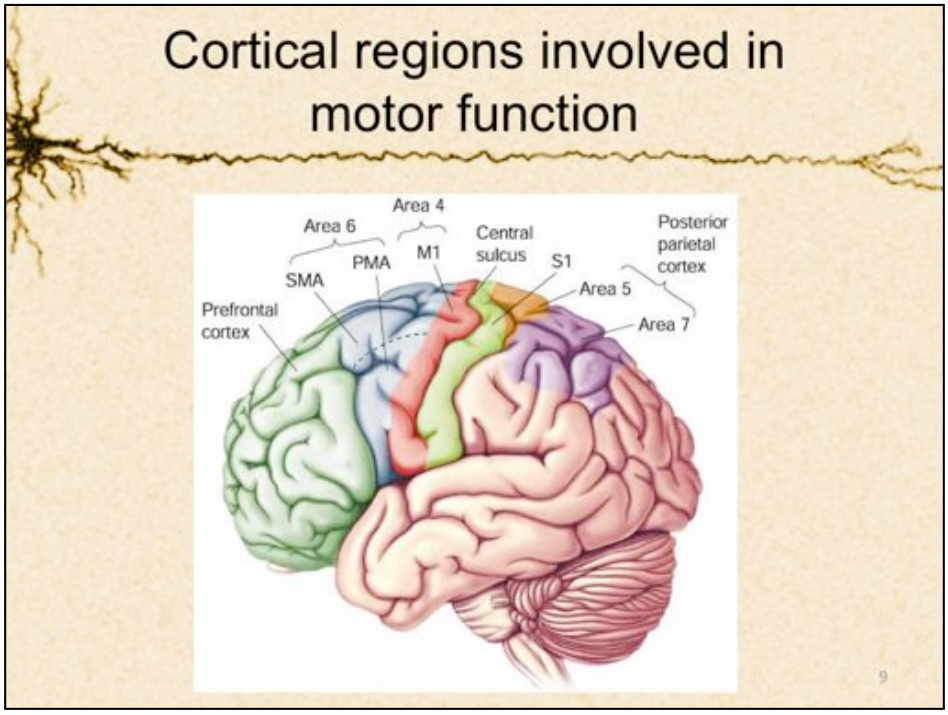
Recall that the stroke patient EH, whose right primary motor cortex and premotor cortex are damaged, is unable to make voluntary movements with his left arm and leg. Signals from the primary motor cortex are evidently needed for voluntary movement of the contralateral limbs. Patient VR, by contrast, is able to make normal limb movements when prompted, but fails to do so spontaneously. Her parietal lesion therefore seems to have affected her capacity to initiate movements internally.



The human sensorimotor system can be thought of as somewhat analogous to a large and efficient company, in which commands are issued in a **top-down** manner. The association areas (**prefrontal cortex** and **parietal cortex**) act as the president or general manager, specifying general goals rather than specific plans of action. Just like a general manager, the association cortex is not routinely involved in the details. This leaves the highest levels of control free to perform the most complex functions.

Instructions then get passed down through the chain of command, to senior executives, section managers, supervisors and finally the workers who have final responsibility for manufacturing and distributing the goods. Areas of secondary motor cortex (the **premotor** and **supplementary motor areas**) are involved in programming specific **patterns of movements**. The **primary motor cortex** in each hemisphere is the point of departure from which sensorimotor signals from the brain are conveyed to the **brainstem** and **spinal cord**.

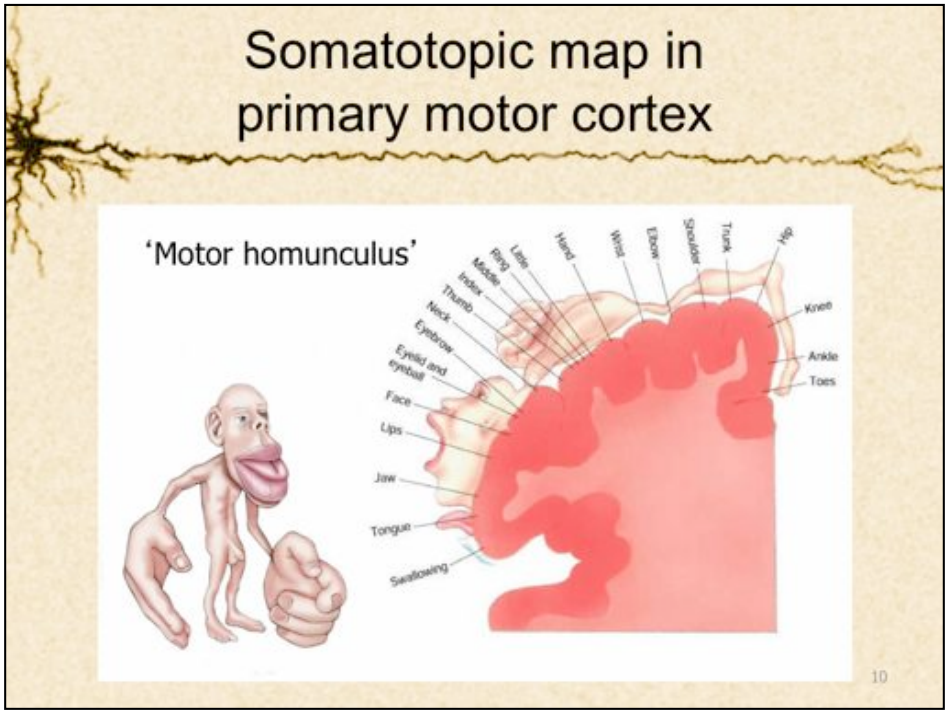
This **hierarchical (layered) organisation** involves both top-down and bottom-up communication. If a problem arises with one of the workers this is conveyed back up the chain of command to the higher levels, whose responsibility it is to resolve any problems, just as sensory feedback from the muscles and tendons is monitored by the CNS in case adjustments are required.



At the cortical level there are several key structures involved in sensorimotor control. We have already seen that the prefrontal and parietal cortex act as the company president or general manager, and exert the highest levels of control. Recall that patient VR fails to make movements with her left arm and hand unless she is prompted to do so by someone else (such as her daughter). In VR's case the general manager that would normally create goals for action has been lost due to her right parietal damage, and so she is reliant on an external agent to 'create the goal' for her. Importantly, once the intention for action is triggered VR performs the required movements completely normally.

The next level down in the chain of command involves the secondary motor areas, the supplementary motor area and premotor area. These areas are responsible for programming specific patterns of movement into complex sequences of behaviour. Once they receive instructions on the general goal of an action from the prefrontal and parietal areas, they determine the most appropriate movements to accomplish this goal efficiently.

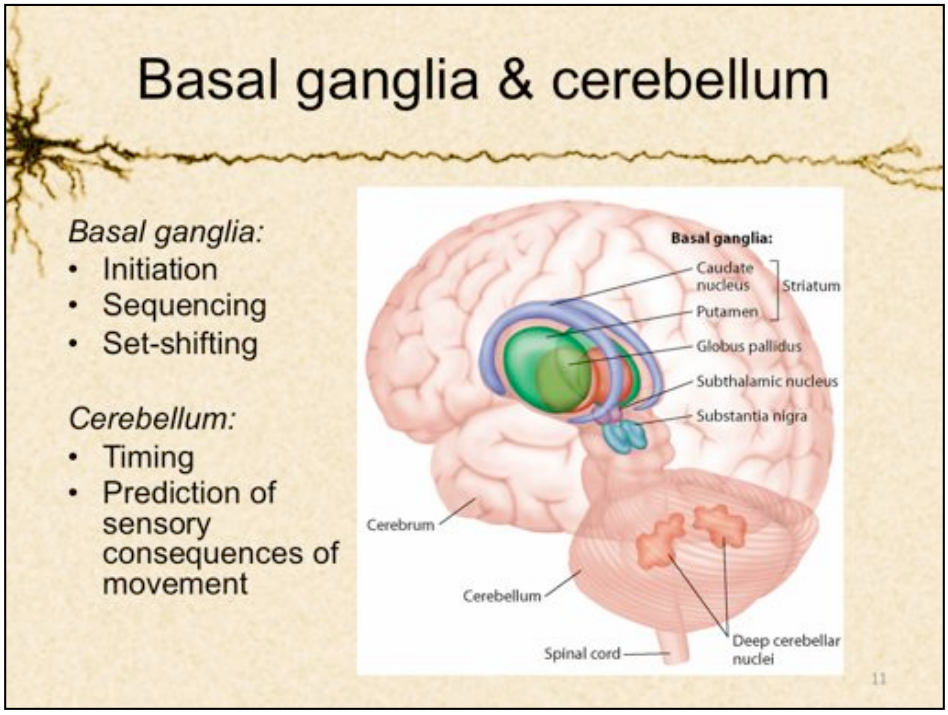
The next level down in the chain of command is the primary motor cortex, which you will recall has a **somatotopic organisation**.



Each region along the strip of cortex of the **precentral gyrus** represents muscles of a particular body part, as illustrated by the '**motor homunculus**'. Note that most of the primary motor cortex is devoted to those body parts that are capable of making fine movements, such as the mouth, lips, tongue and hand.

It should be remembered that the primary motor cortex receives sensory feedback, via somatosensory cortex in the postcentral gyrus, from the muscle spindles.

The primary motor cortex is the final cortical centre from which motor commands are issued to the spinal cord. Damage to this region in one hemisphere, as in patient EH, results in an inability to perform voluntary movements using the limbs of the opposite side of the body. The most prominent symptom of such **hemiplegia** is weakness of the contralesional limbs.



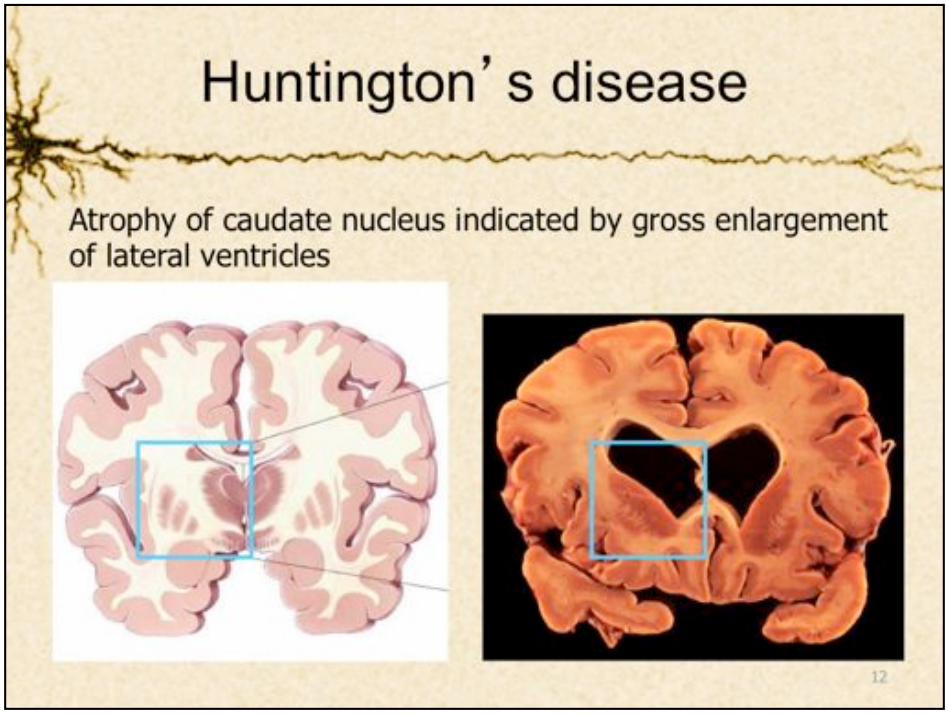
The **basal ganglia** consist of a complex network of interconnected nuclei: the **globus pallidus**, the **putamen**, the **caudate nucleus**, the **subthalamic nucleus** and the **substantia nigra** (Note: the word **ganglia** is plural; the singular is **ganglion**, meaning 'swelling'). The basal ganglia do not send any descending nerve fibres into the spinal cord, and so do not control muscle contractions directly; instead, they receive axons from several cortical areas, and transmit neural signals back to the cortex via the thalamus, suggesting that the basal ganglia play a **modulatory role in motor control**.

The basal ganglia are important in controlling motor behaviours that involve **sequencing of movement** (e.g., a series of finger hand and finger movements needed to play a simple melody on a piano). They are also crucial for the **initiation of voluntary movement**. An analogy for the functioning of the basal ganglia is that they select the most appropriate action from the set of possible responses available in any given situation.

The neurological disorders of **Huntington's chorea** and **Parkinson's disease** are characterised by dysfunction of the basal ganglia.

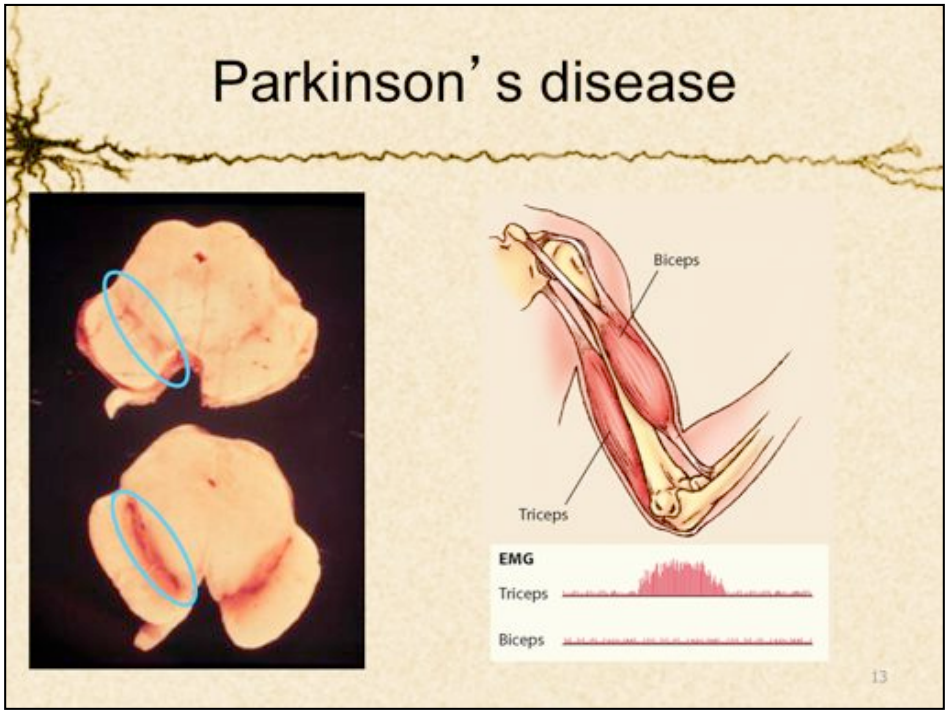
The **cerebellum**, which is situated below the occipital cortex, constitutes only about 10% of the total mass of the brain, but contains more than 50% of its neurons. The cerebellum receives axons from the primary and secondary motor cortex and from brainstem motor nuclei; it also receives sensory information from the somatosensory areas. It is thought that the cerebellum uses this motor and sensory information to **monitor and correct ongoing movements** that may depart from their intended course. The cerebellum is also thought to play a crucial role in aspects of **timing** of movements.

Lesions of the cerebellum disrupt a person's ability to control the direction, velocity, force and amplitude (extent) of voluntary movements; damage to the cerebellum also impairs a person's ability to adjust to changing conditions (e.g., changes in the surface or slope of the ground will cause problems with walking).



Huntington's disease (HD) is a progressive disorder that begins with degeneration of the caudate nucleus and rapidly spreads to other nuclei of the basal ganglia, and finally to the cortex. HD is an autosomal dominant genetic condition (the gene is located on chromosome 4), with onset of symptoms around the fourth to fifth decades on life. Most patients die within 12 years of disease onset.

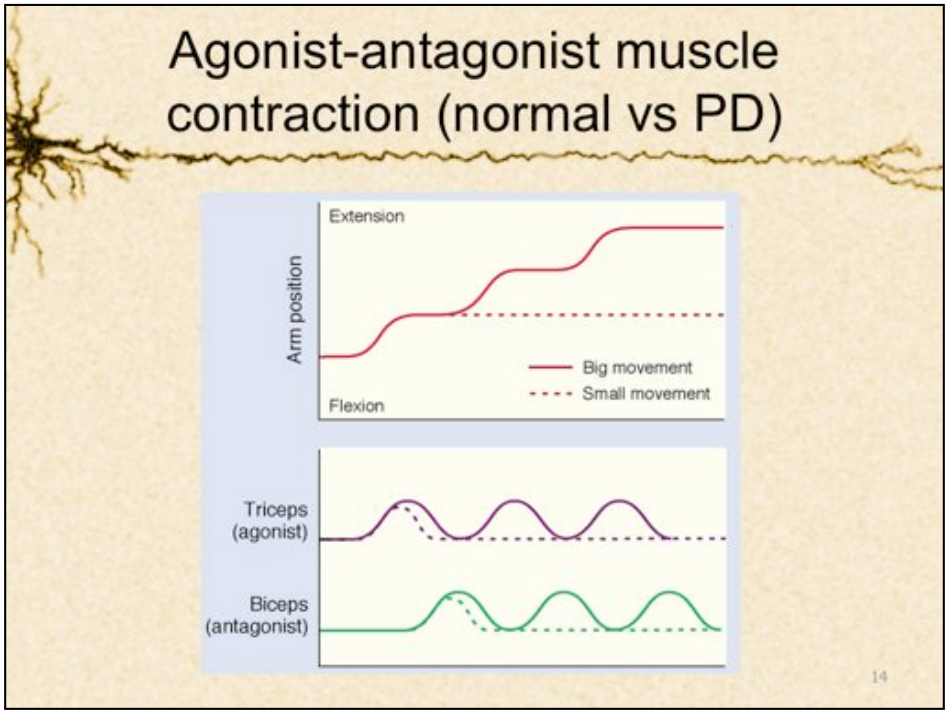
HD is initially characterised by **mental changes** (personality change, irritability, absent mindedness). Thereafter the patient shows a variety of **motor abnormalities**: clumsiness, problems with balance, and restlessness of the limbs. These involuntary movements, or **chorea** (which means 'dancing'), come to dominate the everyday life of the person afflicted with the disorder. The abnormal movements tend to consist of slow writhing or jerking of the arms, legs, trunk and head, which leads to contorted postures. In the 17th century many HD sufferers were executed as witches because it was believed they had been possessed by evil spirits.



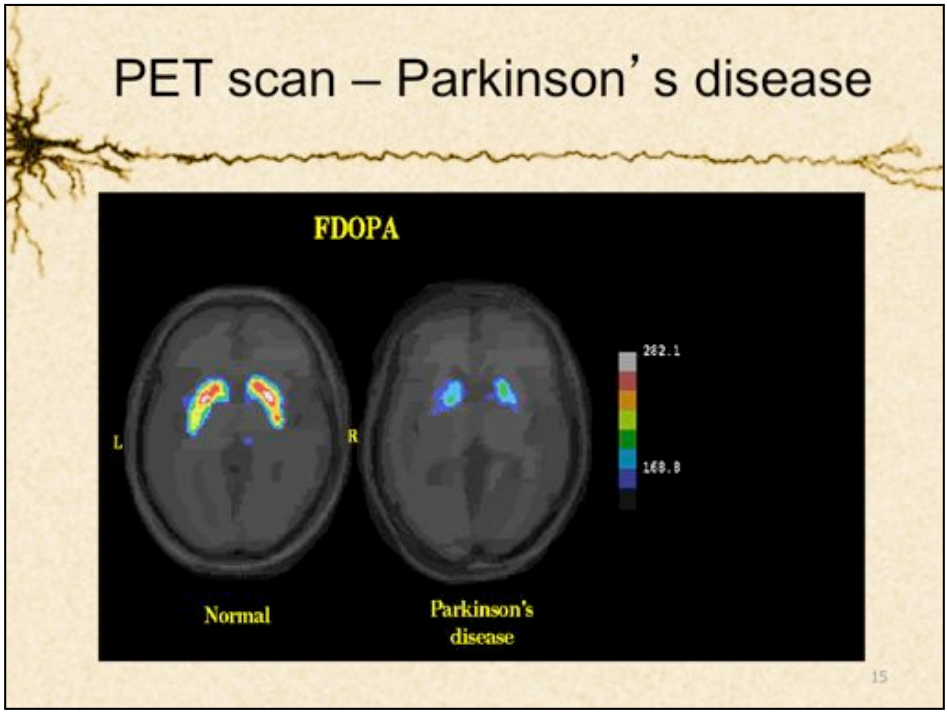
Parkinson's disease (PD) is characterised by degeneration of **dopamine-producing neurons** in the **substantia nigra** ('black substance'), which is located in the brainstem. Although PD can develop after encephalitis or drug abuse, most cases are **idiopathic** (with no known cause). The symptoms of PD are due to loss of **dopaminergic axons** originating in the substantia nigra and projecting to the putamen and globus pallidus. You will recall from Lecture 3 that PD is treated with L-dopa, which is synthesised into dopamine by neurons of the putamen and basal ganglia.

PD has several symptoms:

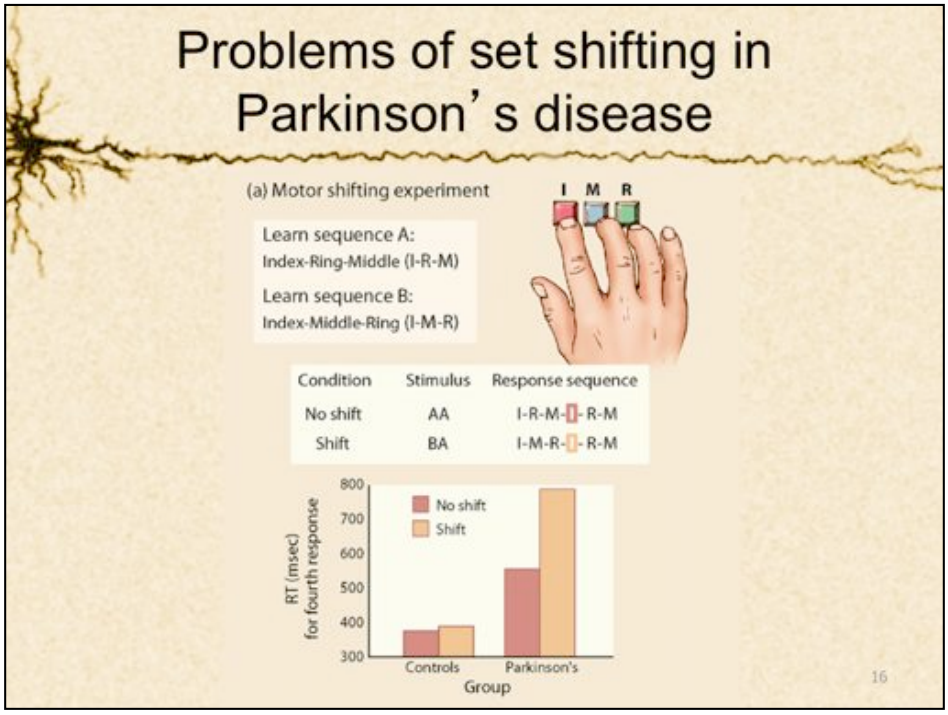
1) **Motor tremor** (shaking), which is present when the limb is at rest but disappears once a movement is initiated.
2) **Rigidity** of the body and limbs, which occurs due to simultaneous contraction of agonist and antagonist muscles.
3) **Festinating gait** (small, shuffling steps with a stooped posture).
4) **Hypokinesia** (difficulty initiating movements).
5) Problems in **sequencing** and **set shifting**.



PD patients may have problems varying the force required to perform limb movements, producing a series of small bursts of agonist and antagonist muscles rather than scaling a single agonist burst to reach the desired goal.

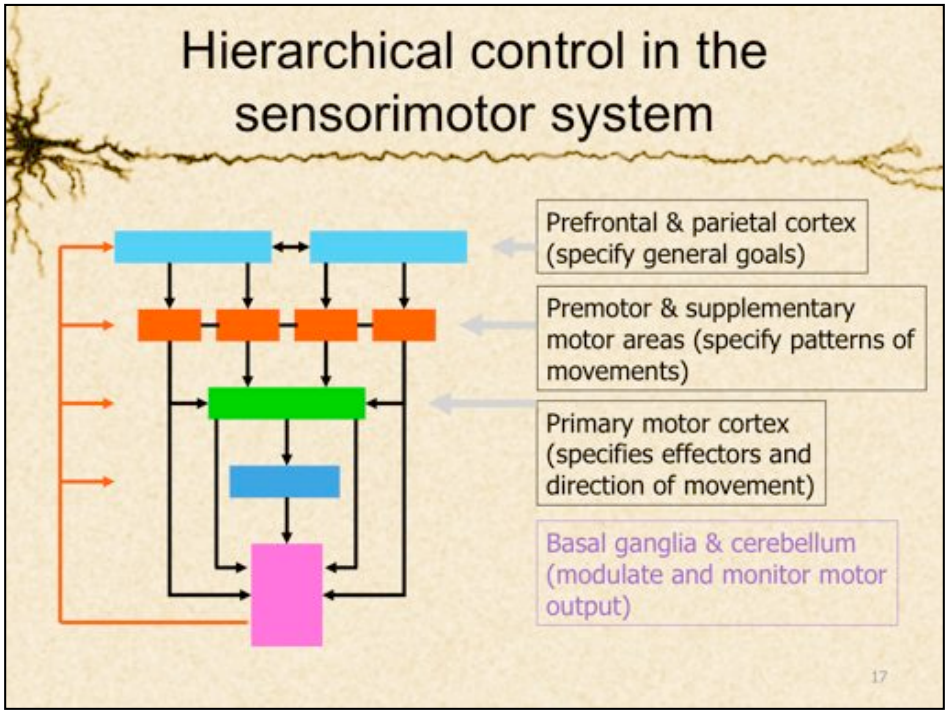


PET scans performed in patients with PD have confirmed that the putamen and globus pallidus have abnormally low levels of the neurotransmitter dopamine.



PD patients exhibit problems in **set shifting** (i.e., shifting from one kind of activity to another) for both motor and cognitive tasks.

In a motor shifting task PD patients and age-matched healthy participants were required to repeat a sequence of three finger movements according to a specified rule. They performed two sequences in a row, and these consisted of either the same or different finger movements. Even though the fourth movement involved the same finger in both the SAME and DIFFERENT sequences, the response time for this movement in the DIFFERENT sequence was markedly impaired (as indicated by the extremely long response times for the 'shift' condition in the PD group relative to healthy control participants.



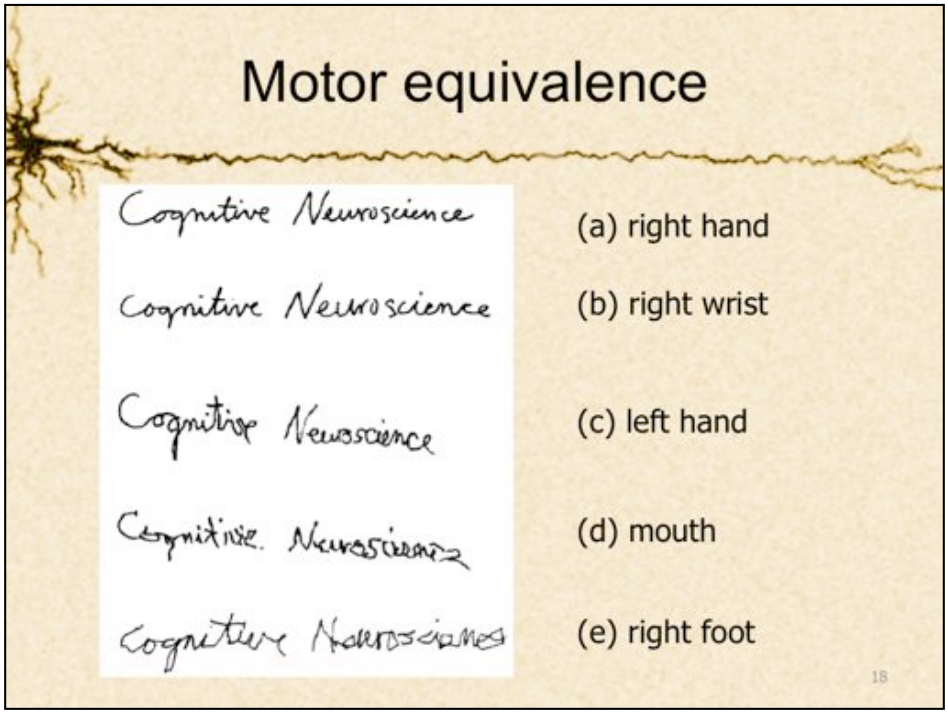
We have now seen that the human sensorimotor system is a complex, interconnected network of brain regions, each of which plays a specific role in controlling purposeful movements. The network is organised **hierarchically**, that is to say, it consists of a number of **levels** of control.

At the top level, the prefrontal and parietal cortex specify general goals for action (e.g., reaching out to pick up a cup of coffee), but they leave the details on how this is to be achieved to lower levels in the hierarchy.

At the next level down, the premotor and supplementary motor areas specify the particular patterns of movement that are required to achieve the desired goal (e.g., lift the right hand from the table, move the hand toward the cup, extend the thumb and fingers to provide an appropriately scaled grasp, close thumb and fingers around the cup handle, etc.).

At the next level down, the primary motor cortex selects the appropriate effectors (limbs) to be used, and controls the various muscle groups that are needed to achieve each component of the action. These instructions are sent to the relevant muscles via the brainstem and spinal cord.

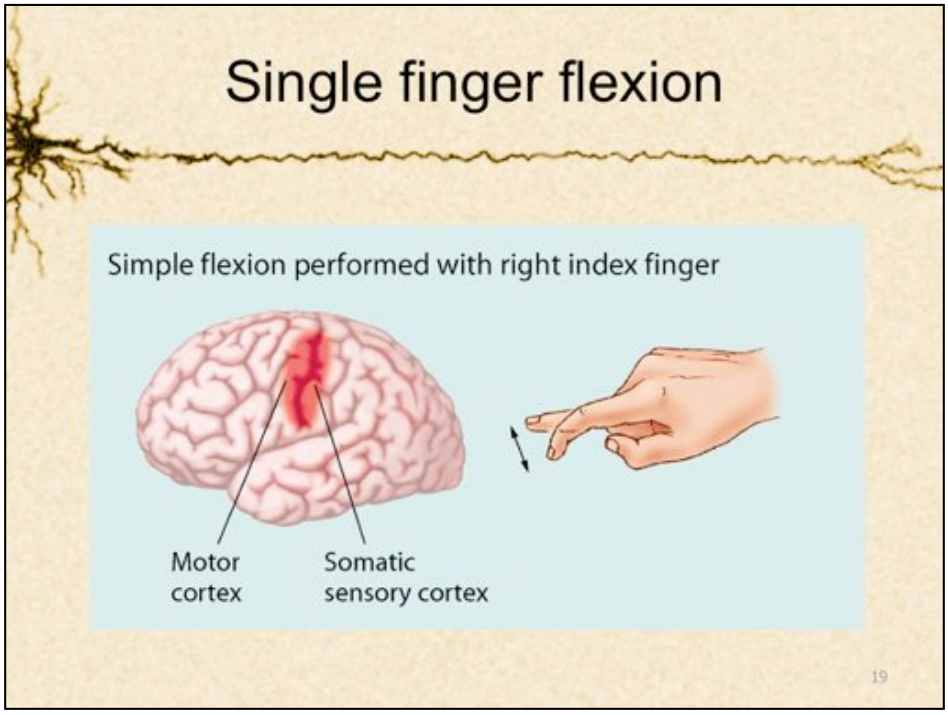
Throughout the planning and execution of actions, the basal ganglia are involved in modulating actions, and the cerebellum integrates sensory feedback from the skin, muscles and joints to correct any errors.



One compelling example of the hierarchical organisation of the sensorimotor system is the phenomenon of **motor equivalence**.

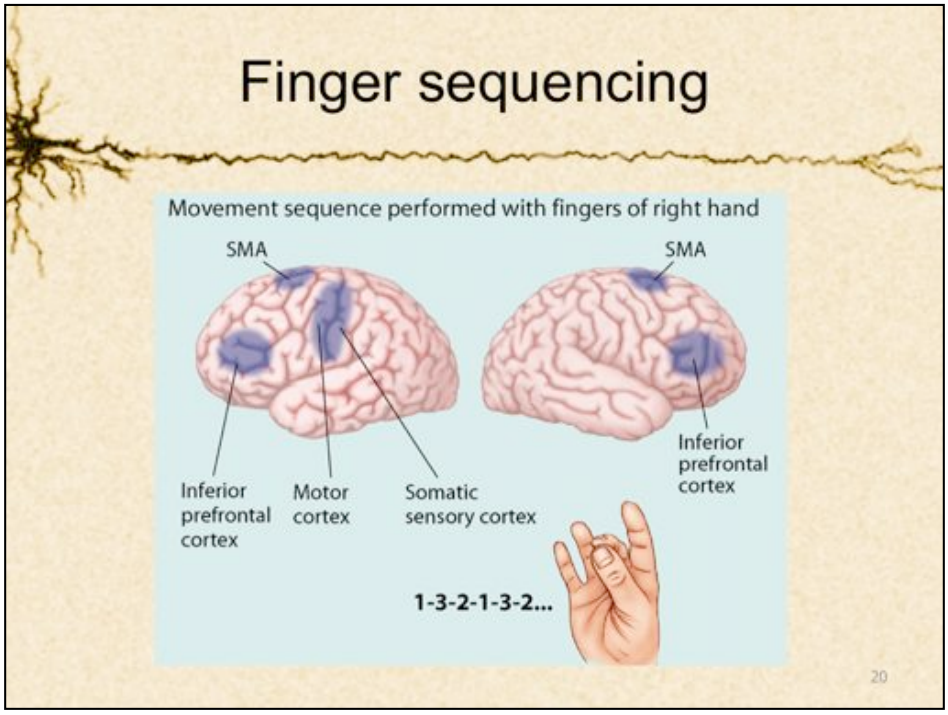
For most of us, handwriting is a skill that is highly overlearned and automatic. We normally have just one preferred hand with which we write, and few of us change this preference during our lifetime. It is tempting to think that the skill of handwriting is represented at one of the lower levels of the motor hierarchy, perhaps even in the hand region of the primary motor cortex contralateral to our preferred writing hand.

Yet a simple experiment shows clearly that the actions required to produce written words are represented at a higher level in the motor hierarchy. When people are asked to write the same phrase using different effectors and different muscle groups, the final results are strikingly similar. This suggests that the motor programs used to produce written words are in fact represented at a relatively high level in the motor hierarchy, above that of the primary motor cortex.

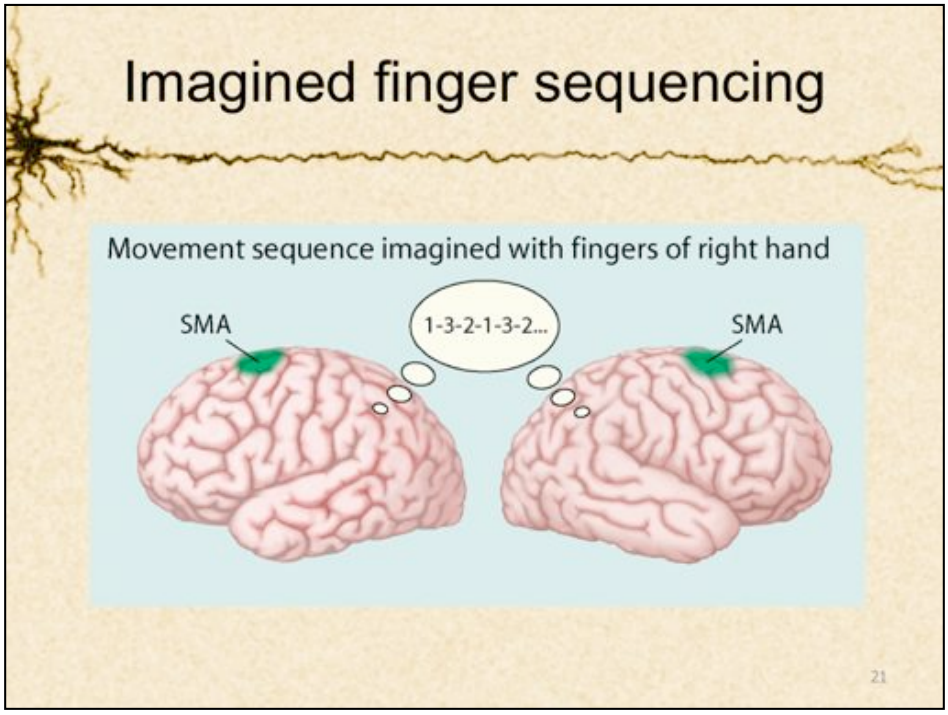


Studies using functional brain imaging have confirmed the general principle of hierarchical organisation of the human sensorimotor system. A study by Roland and his colleagues (1993) examined patterns of regional cerebral blood flow that correlated with particular motor actions involving the fingers of one hand. (Recall from Lecture 6 that changes in regional cerebral blood flow provide an index of the relative level of activity of neurons in corresponding areas of the brain.)

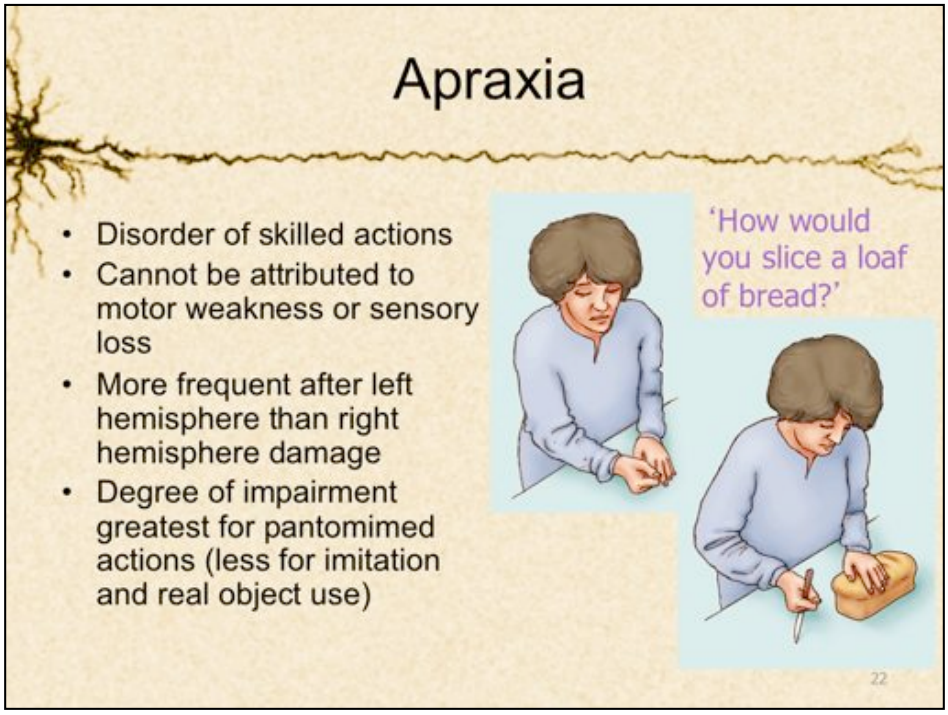
When participants performed flexion and extension movements with the index finger of their right hand, areas in the primary motor and somatosensory cortex of the left hemisphere were selectively active.



When the same participants were required to perform a sequence of finger movements that followed a specified pattern (index, ring, middle, index, ring, middle, etc.), increased activity was observed not only in the primary motor and somatosensory cortex of the left hemisphere, but also in the prefrontal cortex and supplementary motor areas in both hemispheres. Also active (but not shown in the figure) were the basal ganglia and cerebellum. Thus, the more complicated finger sequencing task recruited many more components of the motor hierarchy than the simple finger flexion task.



In a final experiment, Roland and his colleagues had participants simply **imagine** they were performing the finger sequencing task, without actually moving their fingers at all. Only the supplementary motor area (SMA), in both hemispheres, was selectively active during this imagined movement task. This pattern of brain activity is consistent with the notion that the SMA is involved in the programming of **patterns of movements**, rather than in their actual execution (which is carried out by the primary motor cortex and other structures lower down in the motor hierarchy).



Apraxia is a **disorder of skilled actions** that cannot be attributed to motor weakness or sensory loss. It is more common after damage to the left hemisphere than after damage to the right, and it is particularly associated with lesions of the prefrontal and parietal cortex, which constitute the highest levels of representation in the sensorimotor hierarchy.

Clinical tests for apraxia involve asking the patient to perform simple, everyday gestures and actions to verbal command, e.g., 'Show me how you would comb your hair', or 'Show me how you would use a key to unlock a door'. Patients with apraxia are impaired in performing these tasks, even though they have no difficulties in controlling the muscles involved. Often patients with apraxia will use their limbs as of they were the object (such as extending their index finger as if it were a key and pushing it forward into an imaginary lock. Sometimes patients will simply perform clumsy movements that do not resemble the desired action at all.

Patients with apraxia tend to have the most problems in **pantomiming** actions (i.e., performing actions in response to verbal commands); they may be better at **imitating** an action performed by the examiner, or when given an object and asked to indicate how it should be used.

Patients with apraxia resulting from parietal damage are not only impaired in producing skilled actions, but also in **recognising skilled actions** performed by others. It has been suggested that the left parietal cortex plays a central role in representing learned actions, particularly those involving gesture and tool use.

Summary

- Hierarchy of control in the motor system – initiation, goal specification, programming, execution
- Spinal control of reflexive movements
- Primary motor cortex – somatotopic organisation, population coding of direction
- Basal ganglia – initiation, motor sequencing, set shifting; disorders of motor control (HD, PD)
- Cerebellum – timing, monitoring of sensory consequences of movement
- Parietal and prefrontal cortex – control of skilled movement (apraxia)

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