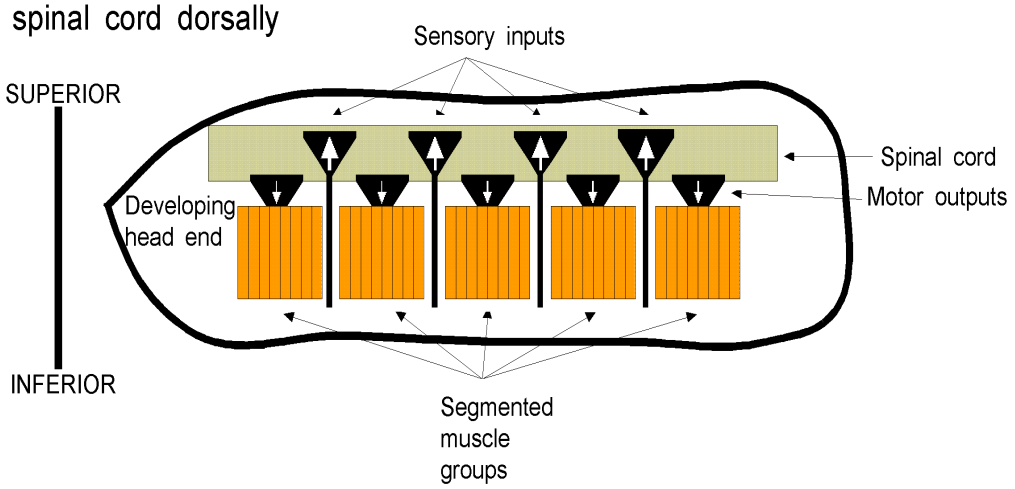
THE NERVOUS SYSTEM

The nervous system, along with the endocrine system, responds to changes in the external or internal environment. The nervous system is the co-ordination system for *rapid* actions.

INITIAL EVOLUTION OF THE NERVOUS SYSTEM

To coordinate the initial rod shaped organisms some form of communication system would have been required. Chemical messengers that spread by diffusion might suffice initially but, for rapid responses, a nervous system would be better and, probably at much the same time as the rod organisms were being evolved in a segmental fashion, a midline coordinating structure (the spinal cord) developed. Vertebrates eventually evolved from such segmented ancestors (possibly flatworms). Segmentation was useful to allow sequential contraction of the muscle groups (myotomes), for defensive reflexes and for propulsion in water or on land. The development of similar segments also minimized the need for genetic information - all that was required was a genetic instruction "repeat this basic pattern n times." Presumably vertebrates living on the seafloor were governed by gravitational considerations, and because muscles which conferred mobility work best if attached to a rigid structure like the backbone, and because muscle groups became ventrally suspended from the backbone, it made sense for the motor output to skeletal muscles to be ventrally situated in the spinal cord. The sensory nerves would mostly serve the outside of the rod-like animal and would have been dorsal to avoid pushing through the muscle groups (Fig. 1).

Figure 1. The probable reason for motor outputs to come out ventrally from the spinal cord and for sensory inputs to enter the spinal cord dorsally

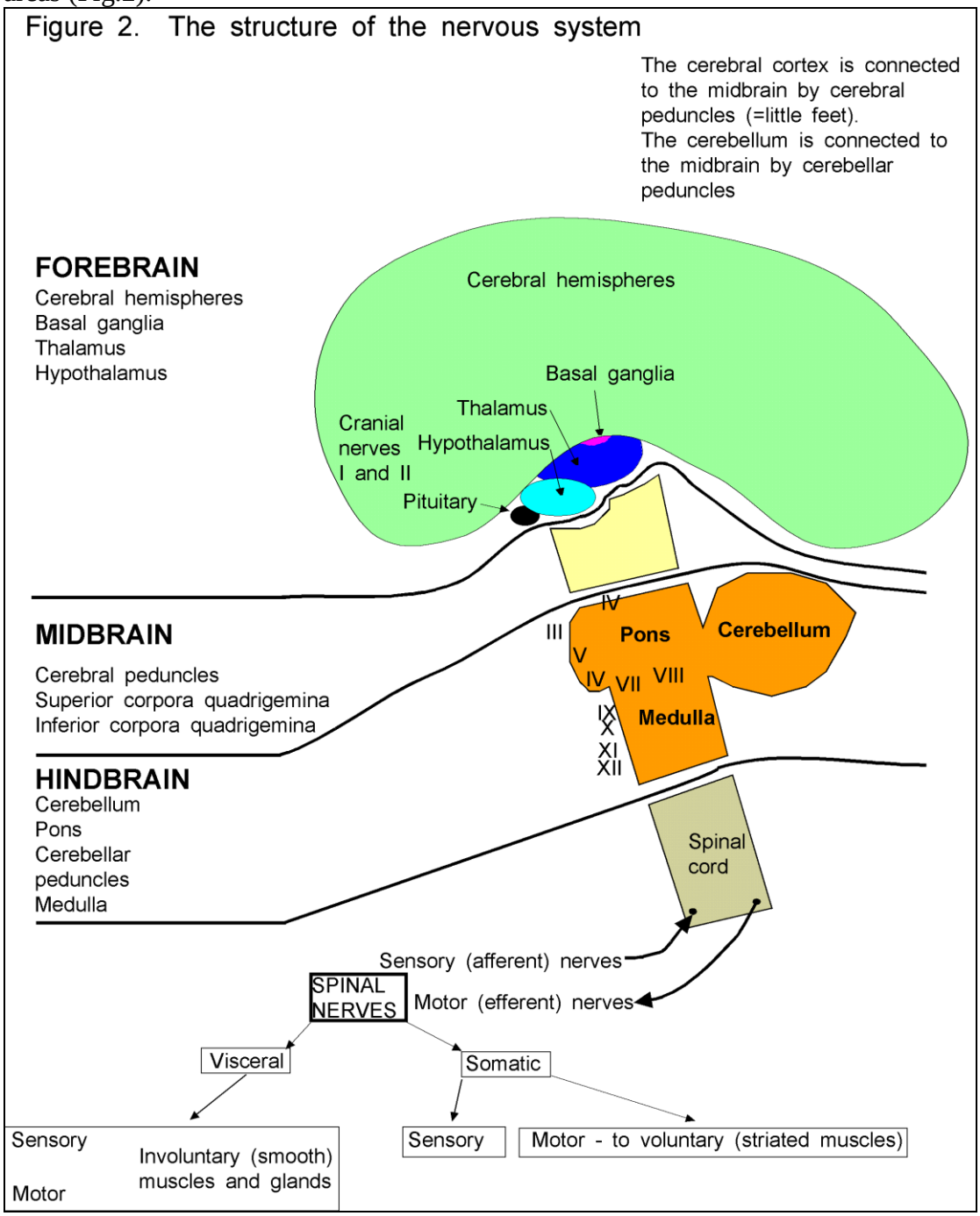


The Primitive organism

To move along the seabed/ground the muscles must be inferiorly situated and so its motor nerve supply is best also situated inferiorly. The sensory supply then "has to be" superior in situation and, In addition, most threats will come from above so it makes sense to have the sensory apparatus situated superiorly

Initially there was a neural tube, the front of which then divided into three functional areas (Fig.2).

Figure 2. The structure of the nervous system



Three important early features in mammalian evolution were:

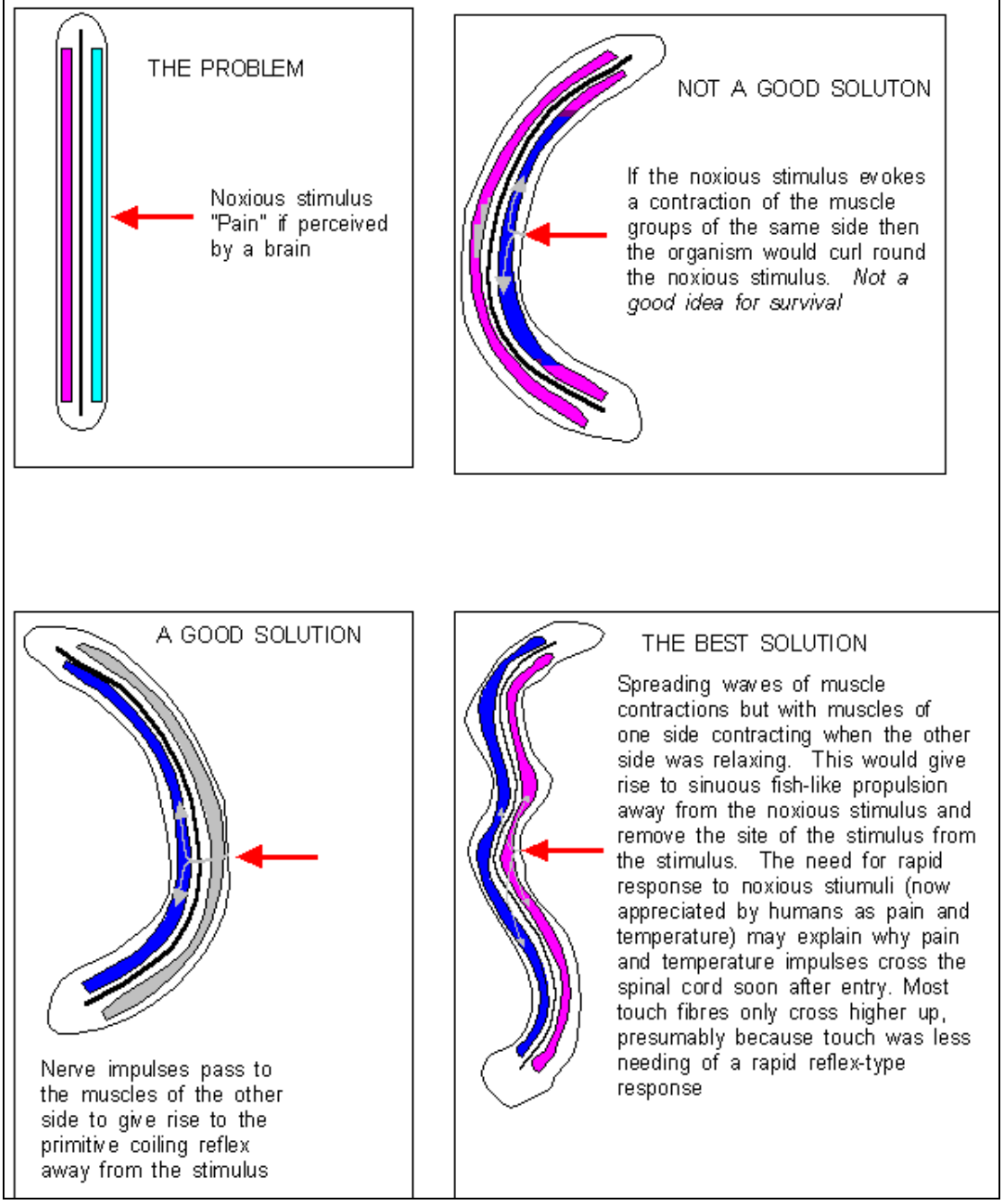
- bilateral symmetry (humans are mostly symmetrical)
- segmentation (to allow sequential contraction for swimming purposes)
- cephalization (a major focalisation of organisation in a head)

Nearly all nervous pathways, except cerebellar and vestibular pathways,cross the midline either in the spinal cord, the brainstem or in the brain. There seem to be three possible explanations.

Firstly the primitive coiling reflex to noxious stimuli (Fig. 3) required that nerve fibres crossed the midline. Impulses from the sensory nerve fibres had to be

conducted to the motor neurones on the immediate opposite side of the spinal cord to allow the primitive rod-like animal to bend away from the noxious stimulus.

Figure 3. A possible explanation of why nerves reporting noxious stimuli, particularly pain and temperature,cross the spinal cord soon after entry.



Secondly stereoscopic sight using two eyes (which allows accurate distance appreciation) would have offered great evolutionary advantages. Integration of the information from the two eyes demands that information crosses the midline (Figs. 4a, 4b and 4c).

Figure 4. Advantages of an optic decussation

Figure 4A. The result if no decussation occurs (this situation does not occur in any vertebrate).

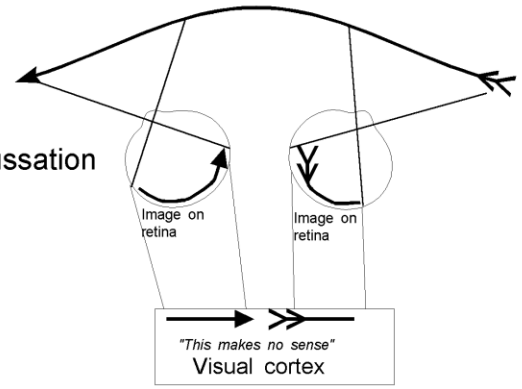


Figure 4B. Complete decussation (as in sub-mammalian vertebrates) Panoramic but not integrated.

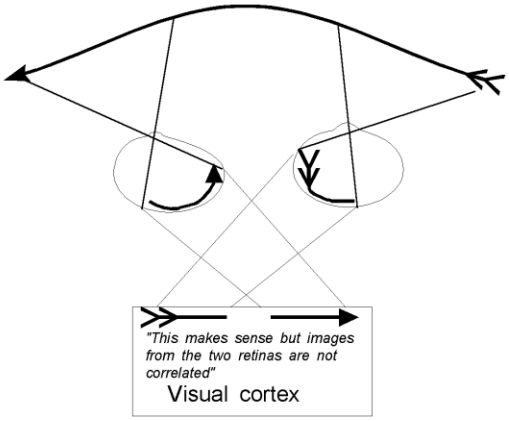
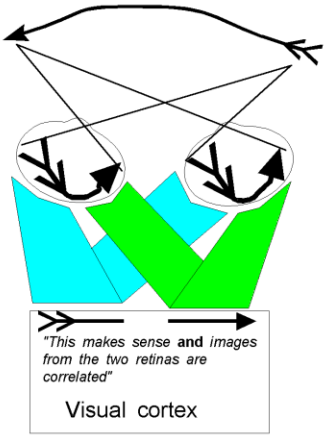


Figure 4C. The human situation. Corresponding images from both eyes are superimposed which assists distance evaluation

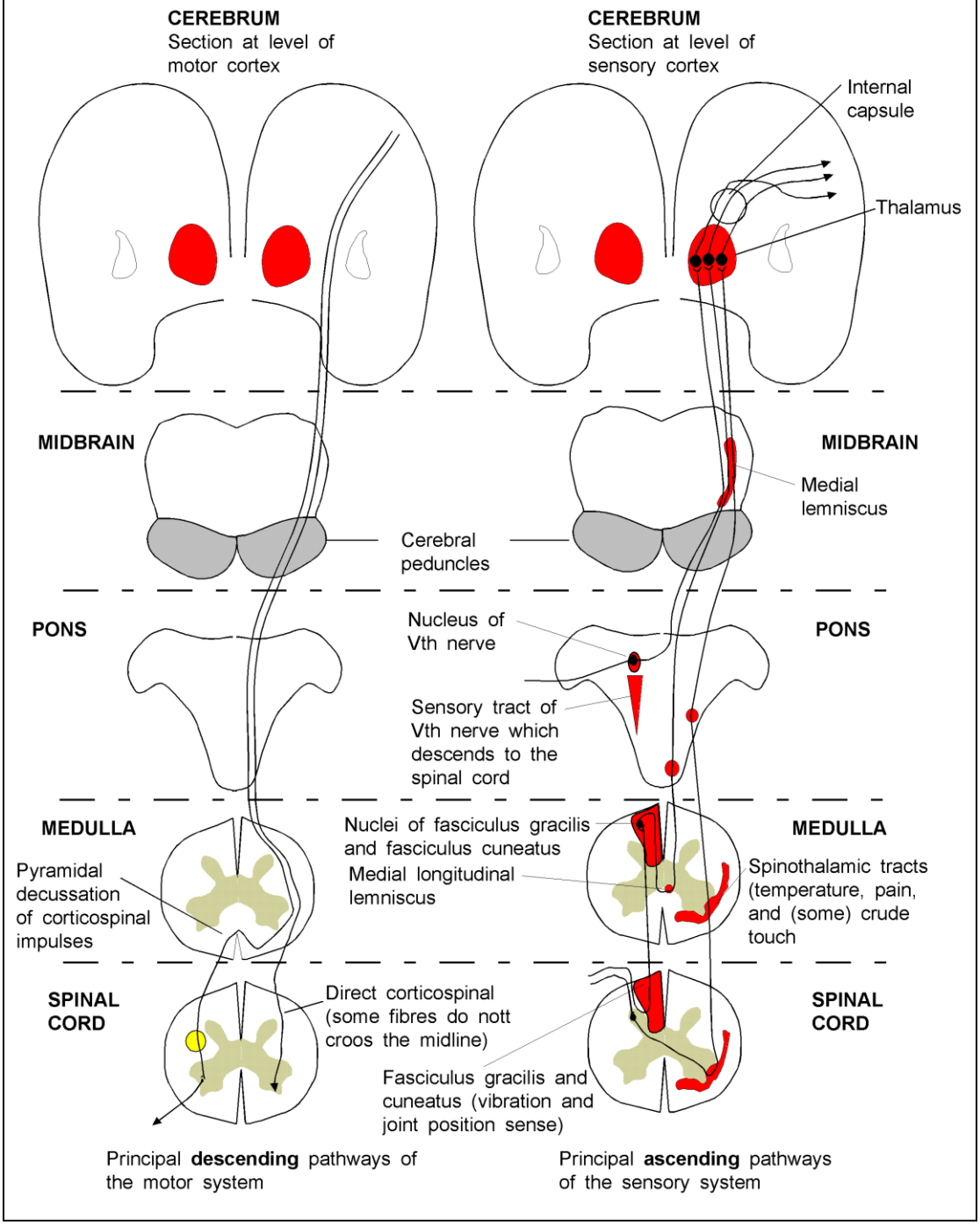


A threat to the right of the head is appreciated by the left hand side of each retina and information from one eye has to cross the midline (in the optic chiasma) and the visual input from both retinas is coordinated in the left occipital cortex (Fig. 4c). Thus if there is to be a coordinated response, then a quicker and more efficient response to the threat demands that the right side (specifically the right arm) be moved. To move the right arm the left motor cortex has to initiate action and to do this information from the left occipital cortex has to cross the midline.

Thirdly swimming or walking requires coordination of the two sides and this requires that nervous information crosses the midline.

The basic structure of the human nervous system is illustrated in Fig. 2. In anatomical terms the central nervous system comprises the brain and spinal cord (Fig. 5) whilst the peripheral nervous system comprises 12 pairs of cranial nerves and 31 pairs of nerves arising from the spinal cord.

Figure 5. The ascending sensory and descending motor pathways of the central nervous system (sagittal section of the cerebrum and cross sections of the midbrain and spinal cord)



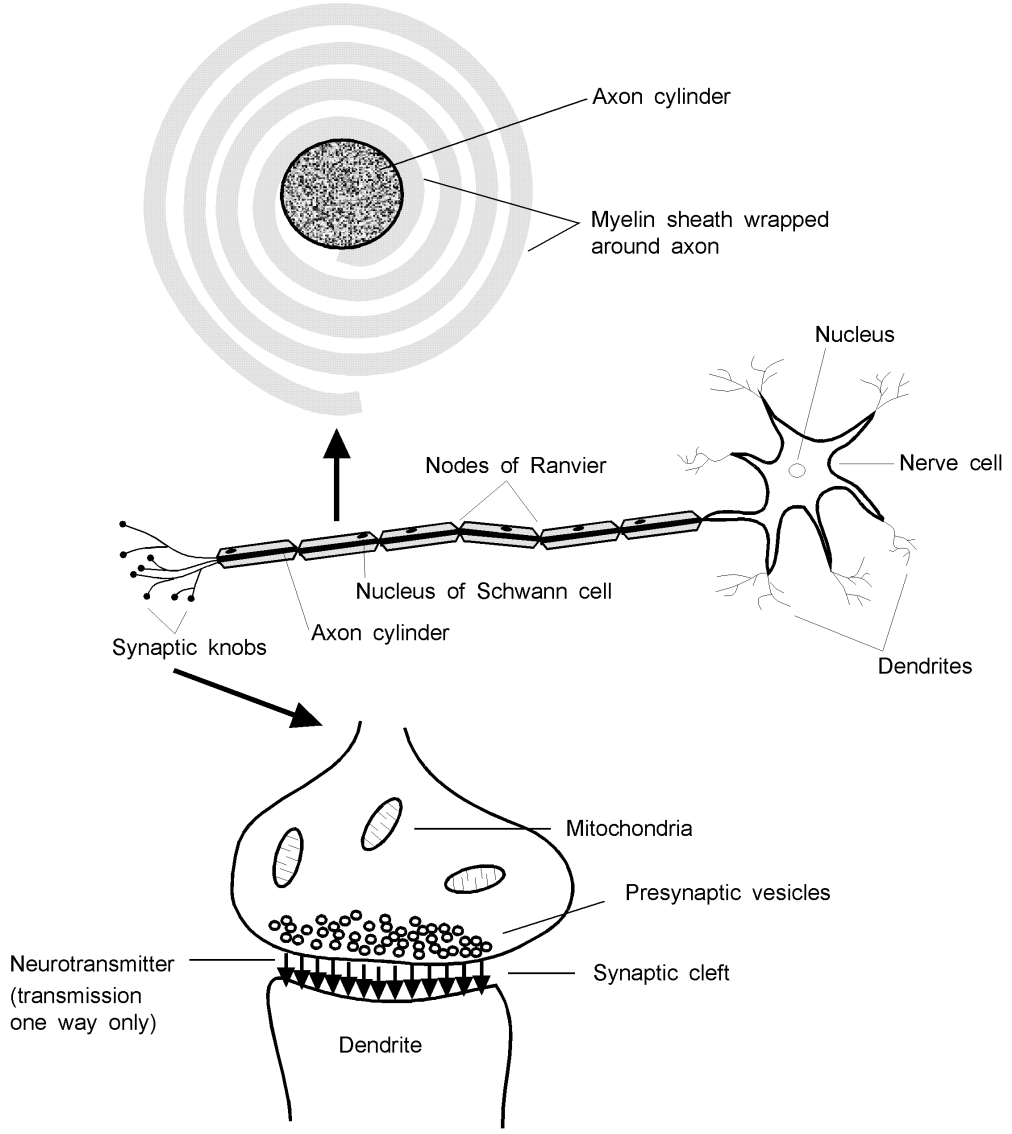
The brain and spinal cord are surrounded by three membranes, called the meninges. The outer layer is the dura mater, the middle the arachnoid mater (interior to which is the cerebrospinal fluid) and the inner pia mater. Blood or infection in the cerebrospinal fluid causes meningeal irritation which results in neck stiffness.

The capillary blood vessels supplying the central nervous system are anatomically and functionally different from capillaries elsewhere in the body. They constitute the blood-brain barrier which prevents certain large molecules and certain drugs from crossing into the central nervous system. On electron microscopy the CNS endothelial cells are joined by tight junctions which are impermeable to large molecules. The are fenestrations (holes) between endothelial cells outside the CNS.

NERVES IN GENERAL

Nerve cells (Fig.6)

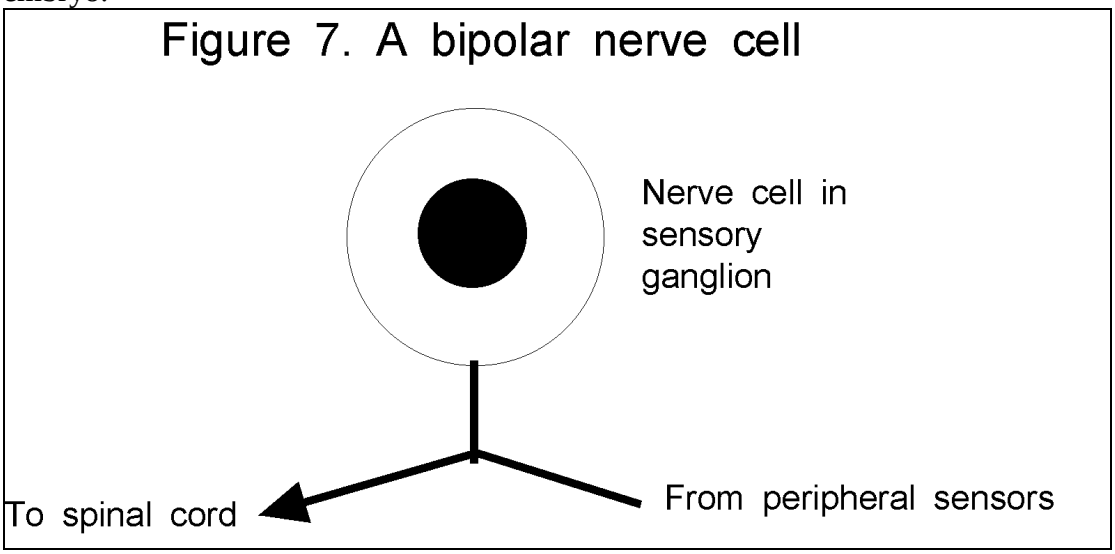
Figure 6. The structure of (medullated) nerves



Axon (=hillock): an outgrowth, usually a single outgrowth, from a nerve cell which conducts impulses along the nerve cell to the other nerve cells or their dendrites. Synonymous with nerve fibre
Dendrite (=tree): the branching process of a nerve cell which conducts impulses to synapses on other nerve cells
Decussation: nerve fibres which cross the midline in bundles of fibres **(commisures** pass to the same side)
Ganglia (=knotlike) collections of nerve cell bodies outwith the central nervous system (except the basal ganglia which are within the central nervous system). They are the seat of visceral motor reflexes which affect some internal organs
Neurone: nerve cell, axon, and dendrites
Nerve fibre: axon and its myelin sheath.

The 10^{11} nerve cells and their processes are the building blocks of the nervous system. Nerve cells, once dead, cannot be replaced. Each nerve cell body has one or more dendrites which collect impulses from other nerve cell bodies or from sensory receptors. Axons are long outgrowths from nerve cells which transmit impulses *away from* the nerve cell. Axons may branch at their terminations and impact upon other nerve cells or their dendrites. In the central nervous system such impulses can be excitatory or inhibitory. In some nerve cells a single process emerges which splits into two in a T-shaped manner - (pseudo) unipolar nerve cells. For example the cells of the sensory spinal ganglia lateral to the dorsal surface of the spinal cord have neurones which have sensory gathering nervous fibres extending peripherally and a nerve fibre extending centrally (Fig. 7). Nerve cells do not divide except in the embryo.

Figure 7. A bipolar nerve cell



There are four types of nerve fibres:

- Myelinated with neurolemma sheaths (mostly in peripheral nerves)
- Myelinated with no neurolemma sheaths (in brain and spinal cord)
- No myelination but a neurolemma sheath (autonomic nerves and fine efferents of corticospinal nerves)
- No myelination and no neurolemma sheath (the gray matter of brain and spinal cord)

There are three types of glial cells within the CNS, astrocytes, oligodendrocytes and ependymal lining cells. These support neurones structurally and metabolically. In the peripheral nervous system neurones are supported by Schwann cells which ensheath axons. In the CNS the oligodendrocytes produce myelin which ensheaths CNS axons. Whilst all axons have Schwann cells surrounding them the speed of propagation along axons is enhanced if the axons are wrapped up with a myelin sheath by Schwann cells (Fig. 6). This "plastic covering of the electric wire" is interrupted at intervals by Nodes of Ranvier. Myelinated axons appear white (constituting the white matter of the brain and spinal cord) whereas unmyelinated axons appear gray (constituting the gray matter (neuronal cell bodies) of the brain and spinal cord). White matter conducts impulses but gray matter processes and interprets. The cortex of the cerebral hemispheres and cerebellum are mostly infolded sheets of nerve cells (gray matter). Areas of the brain called nuclei are (more or less) spherically shaped collections of functionally related gray matter in the central nervous system. Multiple sclerosis produces sensory and motor symptoms and signs caused by patchy demyelination "plaques" in medullated axons "white matter" of the central but not the peripheral nervous system.

There are 31 pairs of spinal nerves - eight cervical, 12 thoracic, five lumbar, five sacral and one rudimentary coccygeal. Peripheral nerves are formed by the unification of the dorsal (sensory) and ventral (motor) roots. In the cervical and lumbar regions these roots join to form the cords of the brachial and lumbrosacral plexuses and from these arise mixed sensory and motor peripheral nerves. The motor fibres supply voluntary (striated muscle). Autonomic nerves arise from sympathetic plexuses to supply cardiac muscle, smooth muscle of the gut and blood vessels, and the salivary and some other glands.

Depending on which roots are predominantly affected there may be:

- Segmental muscle wasting
- Reflex loss
- Dermatomal pattern sensory loss (Fig. 8a, 8b for peripheral nerve losses and Fig. 8c for the likely explanation of the actual dermatome distributions)
- Radiating root pain (localization often poor)
- Tingling
- Oversensitive skin (hyperaethesiae)

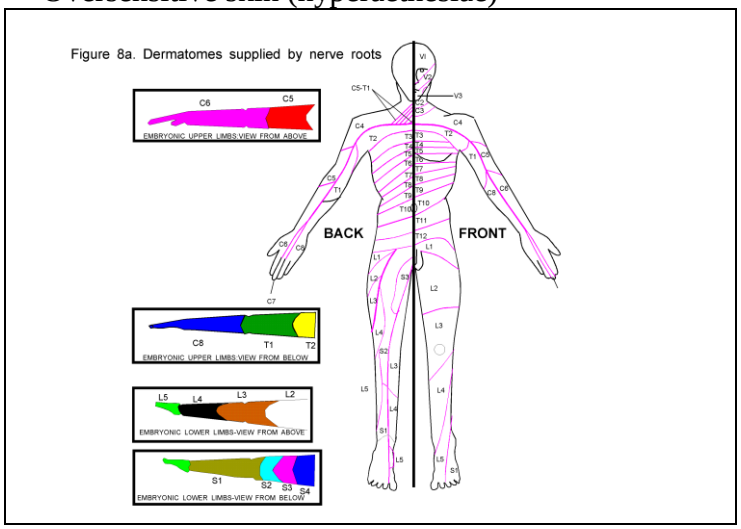


Figure 8b. Areas of skin supplied by peripheral nerves

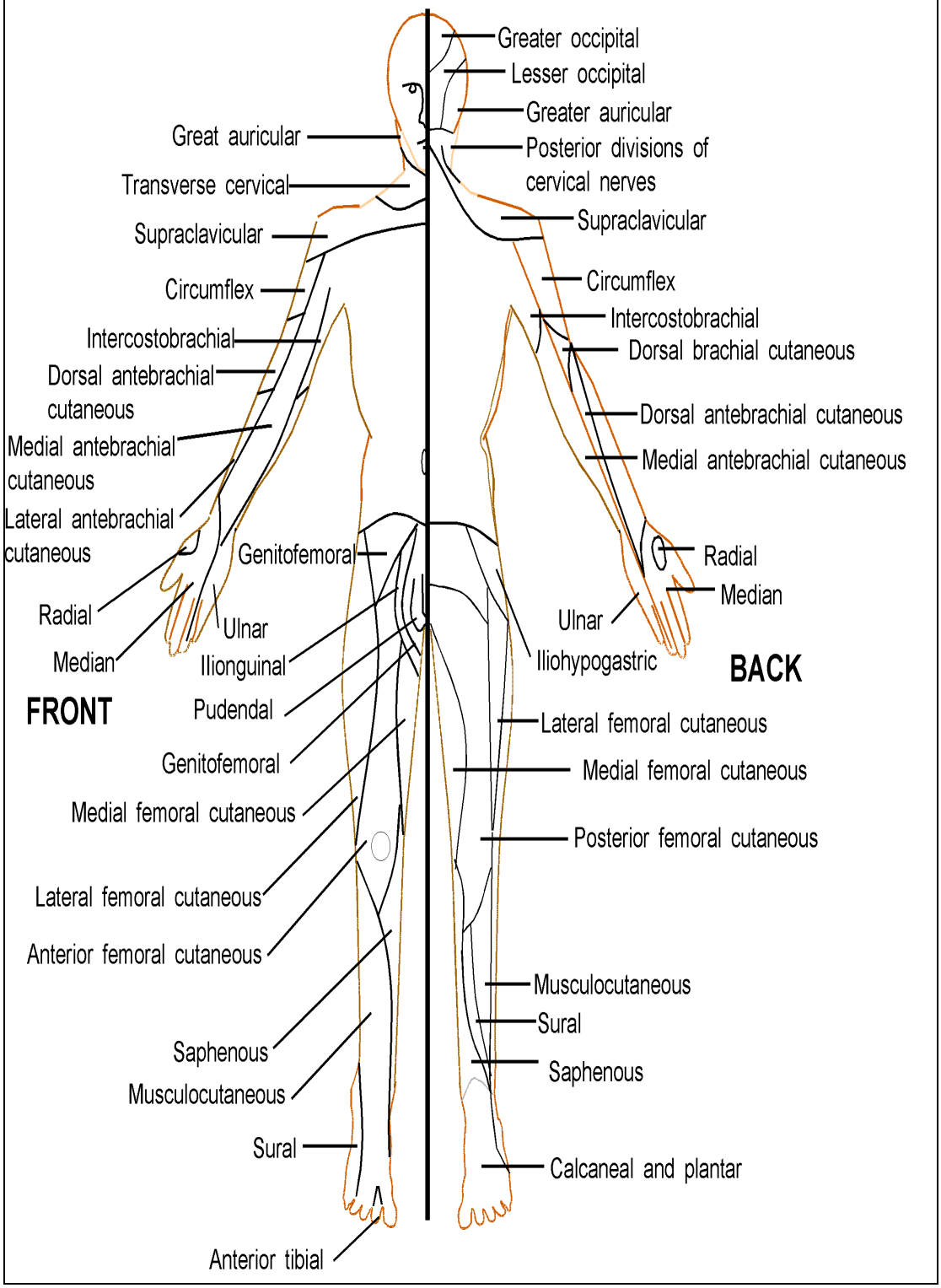
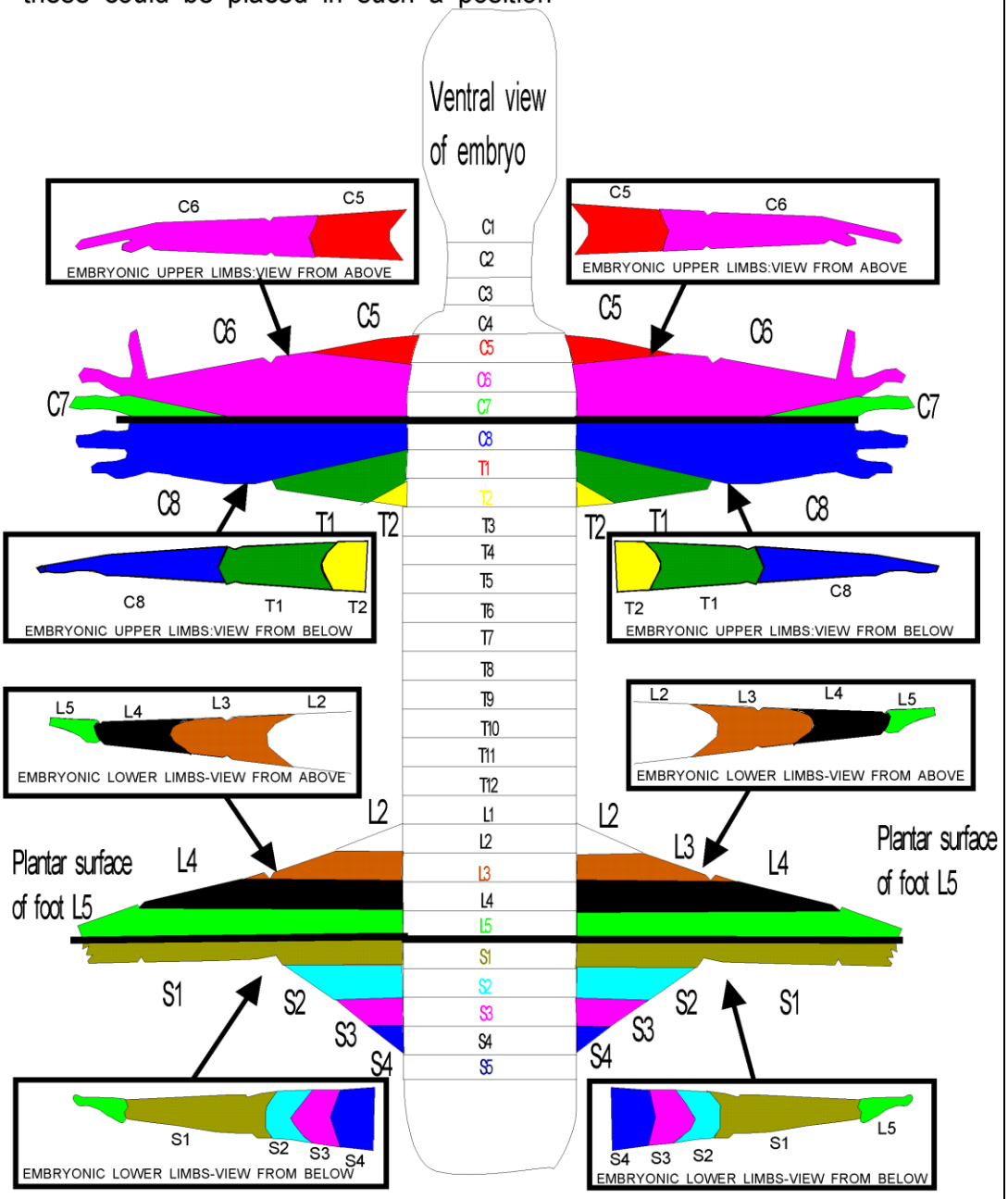


Figure 8c. The embryonic explanation for dermatome distribution. In clinical practice it helps if you can imagine the patient in the "embryonic position." This is easier to do for the upper limbs as these could be placed in such a position



During growth the lower limbs move through 90 degrees to point anteriorly and at the same time rotate so that the plantar surfaces of the feet face downwards

If nerve root problems are caused by pathologies that tether the root then anything which moves the root (movements, coughing, straining) will exacerbate symptoms, especially pain. Depending on the pathology there may also be signs of spinal cord compression. If the nerve roots leaving the end of the spinal cord (conus and cauda equina) are involved then bladder sensation may be reduced and there may be difficulty voiding with a poor stream (motor) and rectal function may be similarly affected leading to incontinence and faecal soiling.

Synapses (=junction)

The junction between a "sending" axon and a "receiving dendrite or nerve cell is a synapse (Fig.6). Synapses are only found in the central nervous system or in autonomic ganglia and only conducts information in one direction. Some axons terminate with a single synapse whilst others may have thousand of terminations. A typical motor neurone in the spinal cord has about 10,000 synaptic contacts. Whether the receiving nerve cell responds usually depends on the number of inputs from other axons that it is receives. The more synapses that impinge on a nerve cell or its dendrites the more modifying influences there are on the final output of the receiving nerve cell. This is the basis of the integrated neural networking that forms the brain.

Frequent use of particular synapses in certain situations may result in the establishment of a regular response pattern. In the central nervous system this constitutes a basis for learning (consciously or unconsciously). Synapse damage leads to forgetting.

Nerves pass on information by putting out chemicals - neurotransmitters - at their synaptic endings. There are four main groups of neurotransmitters (Fig. 6):

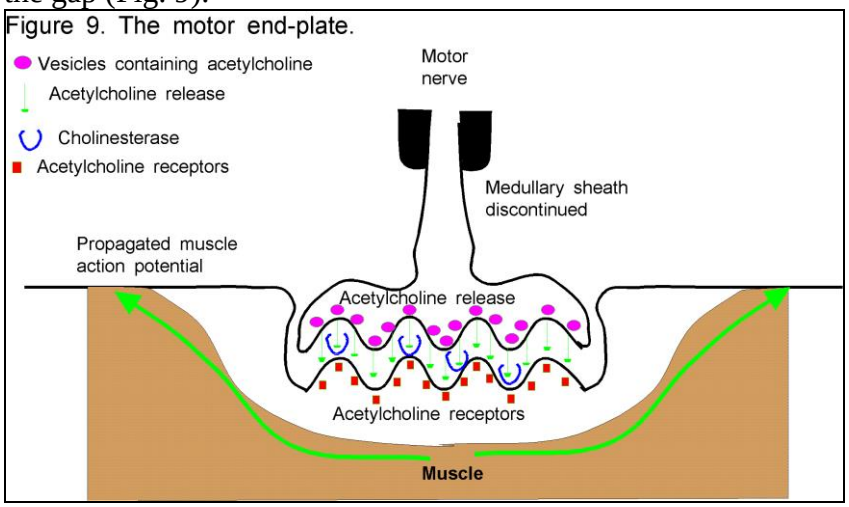
- Acetylcholine (in cholinergic neurones)
- Amines Noradrenaline (in noradrenergic neurones)
  Serotonin (an important transmitter in sensory channels relevant to emotions)
  Dopamine (in the motor systems, limbic system and in the hypothalamus)
- Amino acids Glutamic acid (always excitatory)
  Gamma aminobutyric acid (always inhibitory)
- Peptides Enkephalins
  Endorphins

These transmitters can be reabsorbed, broken down, or competitively inhibited or totally blocked before they can act. Nitric oxide (a gas) has recently been shown to be a neurotransmitter.

Neuromuscular junctions

When a motor nerve arrives at its muscle it loses its myelinated sheath and the axon forms a pre-synaptic terminal (motor end plate). Acetylcholine is released to bridge the gap (Fig. 9).

Figure 9. The motor end-plate.



When sufficient acetylcholine accumulates the muscle fibre depolarizes and an action potential spreads across the muscle to cause contraction. Acetycholine release from the pre-synaptic membrane is enhanced by calcium and its action is limited by destruction by an enzyme, cholinesterase, within the neuromuscular junction. Problems may arise if acetycholine release is modified, its action blocked, or if acetyl choline is broken down by cholinesterase. The final result is muscle weakness.

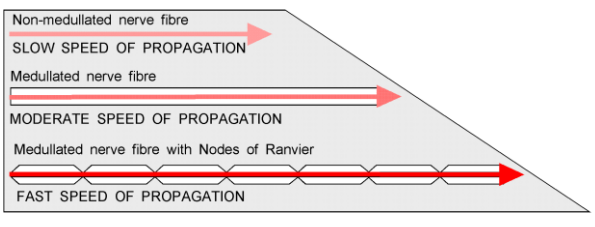
Antibodies may damage the acetylcholine *receptor* and prevent acetylcholine binding causing poor depolarisation of nerve impulses to muscle which leads to muscle fatigue and weakness on repeated use (myasthenia gravis). This may be focal (often causing drooping of the eyelids), or may affect other brainstem-derived cranial nerves or may be more widespread. In the Eaton-Lambert syndrome there are antibodies to calcium channels in the pre-synaptic terminals leading to failure of acetylcholine *release* (and perhaps also in the autonomic nerves to cause parasympathetic failure). Often the weakness improves on repeated use.

With botulism the toxin binds to the terminals of nerves such that acetylcholine cannot be released and there is widespread muscle paralysis and autonomic dysfunction.

Nerve impulses are electric waves of depolarisation which propagate along axons. Normally sodium is pumped out of the axon whereas potassium is kept at a high concentration inside the axon. This causes a 60 mVolts positive charge *outside* the axon. If the axon is excited sodium briefly enters the axon, the inside briefly becoming 40 mvolts positive, and a current flows ahead and initiates further sodium penetration "down the line" and thus a wave of depolarisation travels along the axon. An axon either transmits an impulse or not, "all or nothing," and information conveyed depends on alterations in the frequency of impulses plus the nature and situation of the sending nerve cell body or receptor. On arrival centrally the sensory inputs are allowed to progress depending on their density and timing from the periphery. This wave of depolarisation is followed by a refractory period in which the nerve is absolutely or relatively resistant to the passage of other impulses. After conducting an impulse the larger nerve fibres recover in over one millisecond (up to 1,000 vibrations per second can be appreciated). In practice most sensory fibres can conduct 300-400 impulses per second and motor fibres can conduct at 100-150 impulses a second. If the axon is unmyelinated then the wave flows continuously to its destination but if there are nodes of Ranvier the wave jumps from node to node, increasing the velocity of conduction. Unmyelinated axons can conduct as slowly as 50 cms per second whereas myelinated fibres can conduct at about 100 metres per second (Fig. 10).

Figure 10. Speed of propagation of nerve impulses

Strictly speaking, nerve impulses are not conducted along the nerve but rather depolarisation occurs at right angles to the axis of the nerve and it is the wave of depolarisation that travels along the nerve



Within limits, the larger the diameter of the axons, the faster the conduction

In some situations there can be pain appreciation without touch (from the bladder for example) but in other situations touch becomes pain if afferent nerves convey an excessive number of similar impulses.

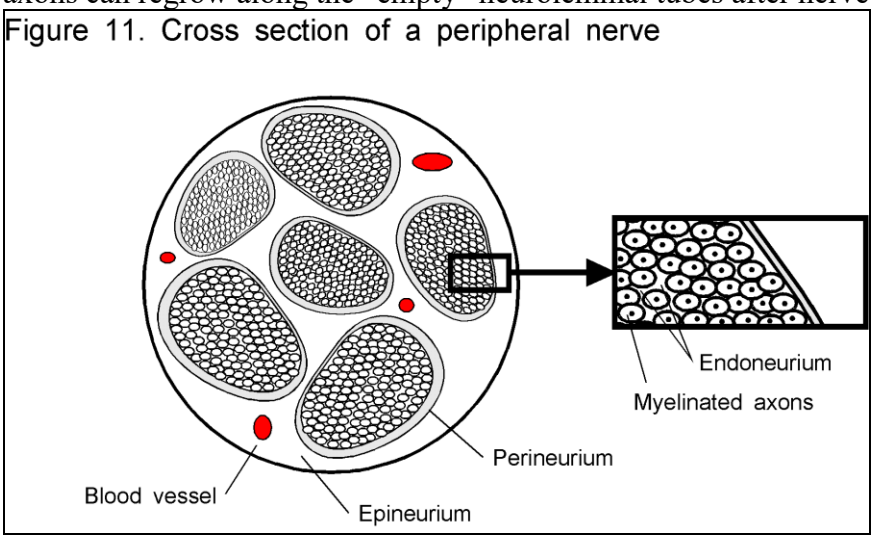
Within limits, the larger the diameter of the axon the quicker the impulse conduction. Large nerve fibres mostly transmit touch, pressure, two point discrimination, and joint position sensation but usually not pain and temperature. Smaller fibres (which are more likely to be affected by diabetes) mostly transmit pain and temperature but not other modalities (thus neuropathic foot ulcers caused by loss of pain sensation may result). Axons normally only transmit away from the nerve cell. Nerves that carry important messages tend to have myelinated axons and a minimum of synapses to allow rapid conduction (for example in the corticospinal tract).

Nerve cell bodies in the central nervous system, usually in the cortex, can give rise to unusual, paroxysmal, electrical activity which may spread to cause epilepsy, the manifestations of which depend upon the initial area of gray matter involved and the extent of spread. Localized seizure discharges in the frontal lobe motor cortex produce a localized jerking of skeletal muscle on the contralateral side but generalized epilepsy produces tonic (continuous contraction) and then clonic (jerking) contraction of all skeletal muscle along with loss of consciousness. Epileptic foci in areas of the brain that do not normally cause skeletal muscle action may present in other ways (temporal lobe epilepsy for example may present with autonomic sensations of taste, smell or déjà vu with loss of awareness without loss of consciousness.

Peripheral neuropathy

Peripheral nerves are surrounded by neurilemma (Fig. 11), groups of axons by perineurium, and groups of groups (the nerves) are surrounded by epineurium which also contains the nourishing blood vessels. The nerve fibres of brain and spinal cord have no neurolemma and are incapable of regeneration whereas peripheral nerve axons can regrow along the "empty" neurolemmal tubes after nerve damage.

Figure 11. Cross section of a peripheral nerve



The nutrient-providing blood vessels may be damaged by metabolic disorders (such as diabetes mellitus) and cause axonal degeneration or demyelination. The longer the nerve then the more cumulative low grade damage, explaining why symptoms and signs of neuropathy (nerve damage without inflammation) often begin in the feet. Nerve compression often causes both axonal degeneration and demyelination.

Neuropathy may affect individual nerves or nerves (mononeuropathy) or all nerves (generalized neuropathy). In either motor symptoms may include:

- Weakness
- Wasting
- Fasciculation (twitching of small areas of muscle caused by spontaneous electrical activity in the motor unit so that individual muscle fasciculi contract)

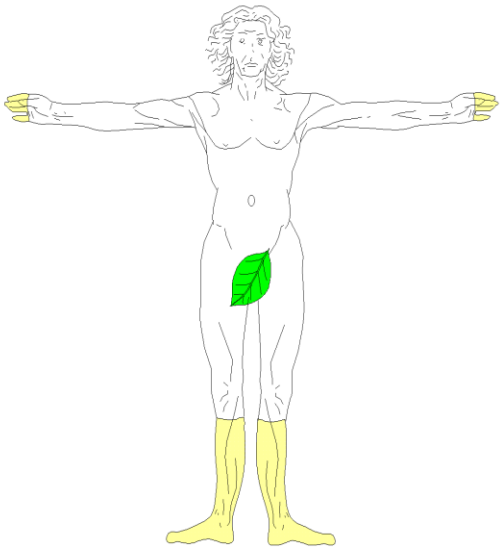
In either sensory symptoms may include:

- Loss of sensation (but only in the area exclusively supplied by an individual nerves in a mononeuropathy)
- Pain
- Tingling

With a generalized sensory neuropathy there will be glove and stocking sensory loss (Fig. 12). Reflexes may be reduced by damage to either sensory or motor nerve fibres or both.

Figure 12. Glove and stocking sensory loss in peripheral neuropathy

= Areas of diminished (not necessarily completely absent) sensory appreciation which suggests diffuse damage to all nerves - but the cumulative damage affecting the longer nerves first



Peripheral nerves usually contain a mixture of sensory (afferent= to carry to) and motor (efferent= to carry away) fibres. If a peripheral nerve is cut then the result usually include:

- Flaccid paralysis of the muscles receiving motor innervation, followed by muscle wasting and contractures
- Loss of cutaneous sensation in the areas exclusively supplied by the nerve (most cutaneous areas exhibit overlap of cutaneous sensation)
- Loss of reflexes (if there are reflexes associated with the nerve concerned)
- Loss of sympathetic and parasympathetic responses (causing initial vasodilatation, lack of sweating and piloerection)

The cell bodies of the motor nerves (motor horn cells) may be affected in isolation within the spinal cord (polio specifically damages the anterior horn cells of the lower motor neurone) to produce lower motor neurone signs.

If a peripheral nerve is damaged the neurone fibres degenerate proximally, usually up to the proximal node of Ranvier, and distally. The Schwann cells (Fig. 6) then remain, surrounding an empty tube. The intact proximal neurone then grows distally, hopefully along its previous tube, at a rate of about one mm a day. If a nerve is cut but the two ends remain approximately opposed an axon may regrow along the wrong tube and this can cause partial or altered sensation or false motor innervation leading to persisting dysfunction.

Malignant tumours of nerves are rare but benign tumours are not uncommon, either in isolation (as neuromas, notably acoustic neuromas) or as part of a more generalized problem (e.g. neurofibromatosis). Generalized peripheral neuropathy can be post-infective, metabolic, or related to malignancy.

- diabetes mellitus
- vitamin deficiency (notably vitamin B12 or B1)
- infections (including leprosy and HIV)
- toxic (including alcohol)
- drug induced
- hereditary
- vasculitis
- compression at certain points: wrist (median nerve), elbow (ulnar nerve), supper arm (radial nerve), head of fibula(lateral popliteal nerve), groin (lateral cutaneous nerve of thigh) and buttock (sciatic nerve).

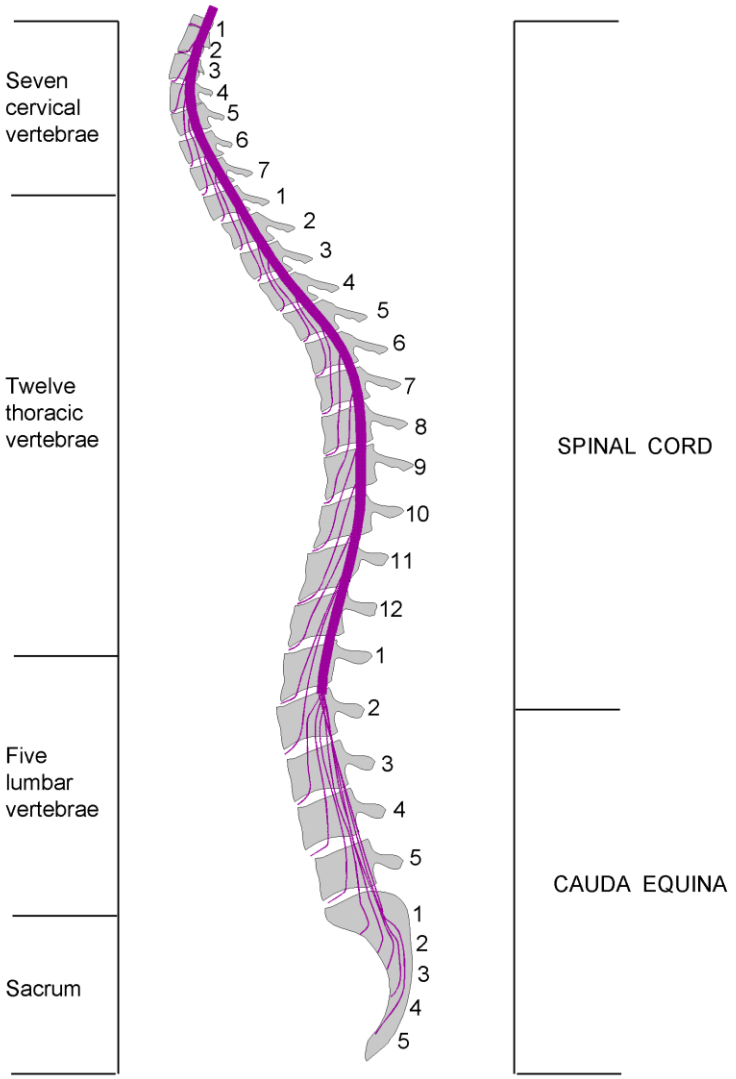
THE SPINAL CORD

Groups of functionally similar axons are called tracts, fascicles, peduncles, or lemnisci. The first part specifies the site of origin, the second the destination e.g. corticospinal = from the cortex to the spinal cord

The spinal cord extends from the foramen magnum to the lower border of the first lumbar vertebra (Fig. 13). The nerve roots leave the cord at an oblique angle and thus the site of the cord damage may be higher up the spine than the root number would suggest.

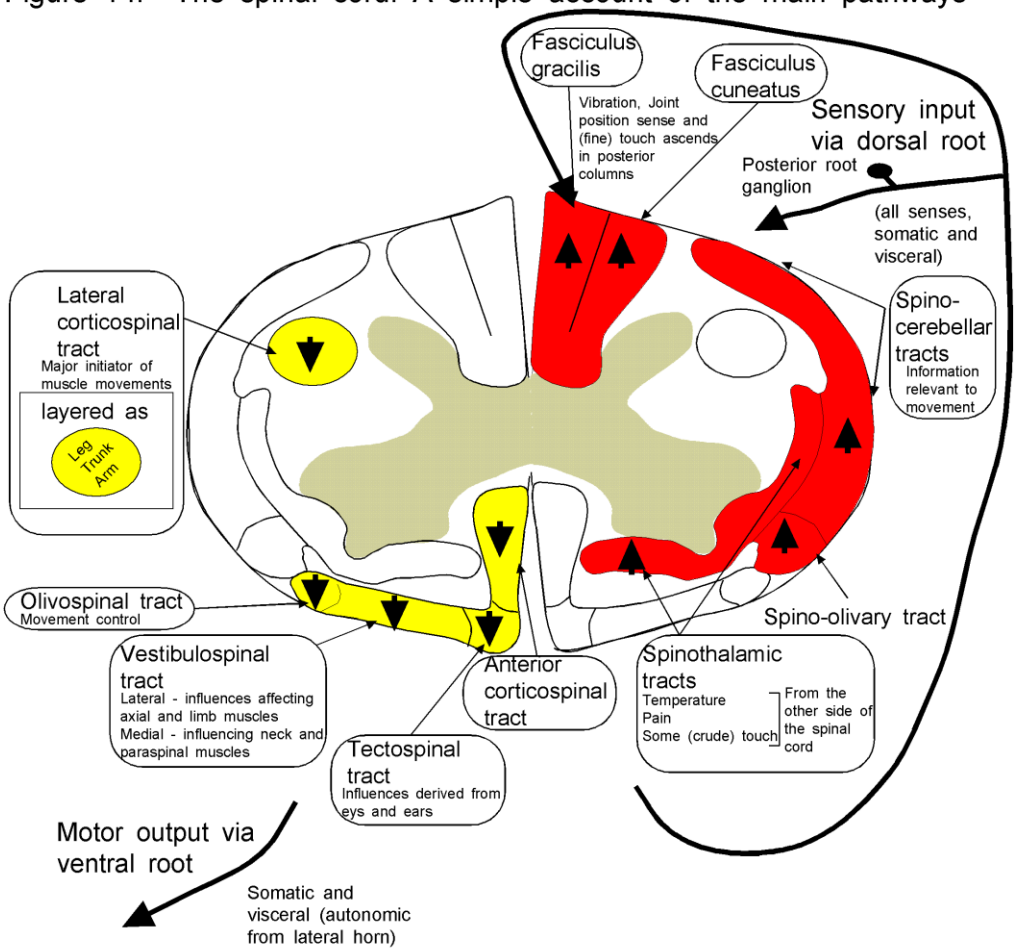
Figure 13 Spinal nerve roots and their relationship to numbered vertebrae.

Nore that the root number is determined by the site of exit from the spinal cord but, because of the oblique descent of the nerves exiting the spinal cord, the vertebra-related level of pathology may be quite distant. Also note that there are seven cervical vertebrae but eight cervical nerves



In the spinal cord, from dorsal to ventral, the inputs are somatic sensory and visceral sensory (Fig. 14, right side in red), and the outputs are visceral motor and somatic motor (Fig. 14, left side in yellow).

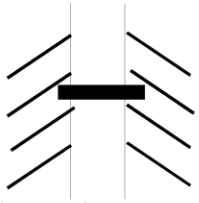
Figure 14. The spinal cord. A simple account of the main pathways



Complete spinal cord transection (Fig. 15) causes:

- Loss of all sensory modalities below the lesion
- Complete flaccid paralysis below the lesion
- Inability to pass urine because of a flaccid bladder and constipation
- Lower motor neurone signs at the level of the lesion
- Sensory impairment in the area exclusively supplied at the level of the lesion (there is overlap)
- Reflexes reduced or absent at the level of the lesion (and possibly below this at the time of the transection
- Upper motor neurone signs below the level of the lesion

Figure 15. The results of complete spinal cord transection



In the acute stage (spinal shock) there is:

Complete loss of sensation and power in spinal segment at and below transection

Loss of all reflexes

Bladder and rectum are paralysed (often there is urinary retention as the internal sphincter of the bladder retains its function)

Loss of arterial tone (and thus low blood pressure) if the transection is above the level of the sympathetic outflow (TI - L2)

Cold blue limbs as immobility leads to lack of muscle pump to return venous blood

Later:

The bladder regains the power of contraction

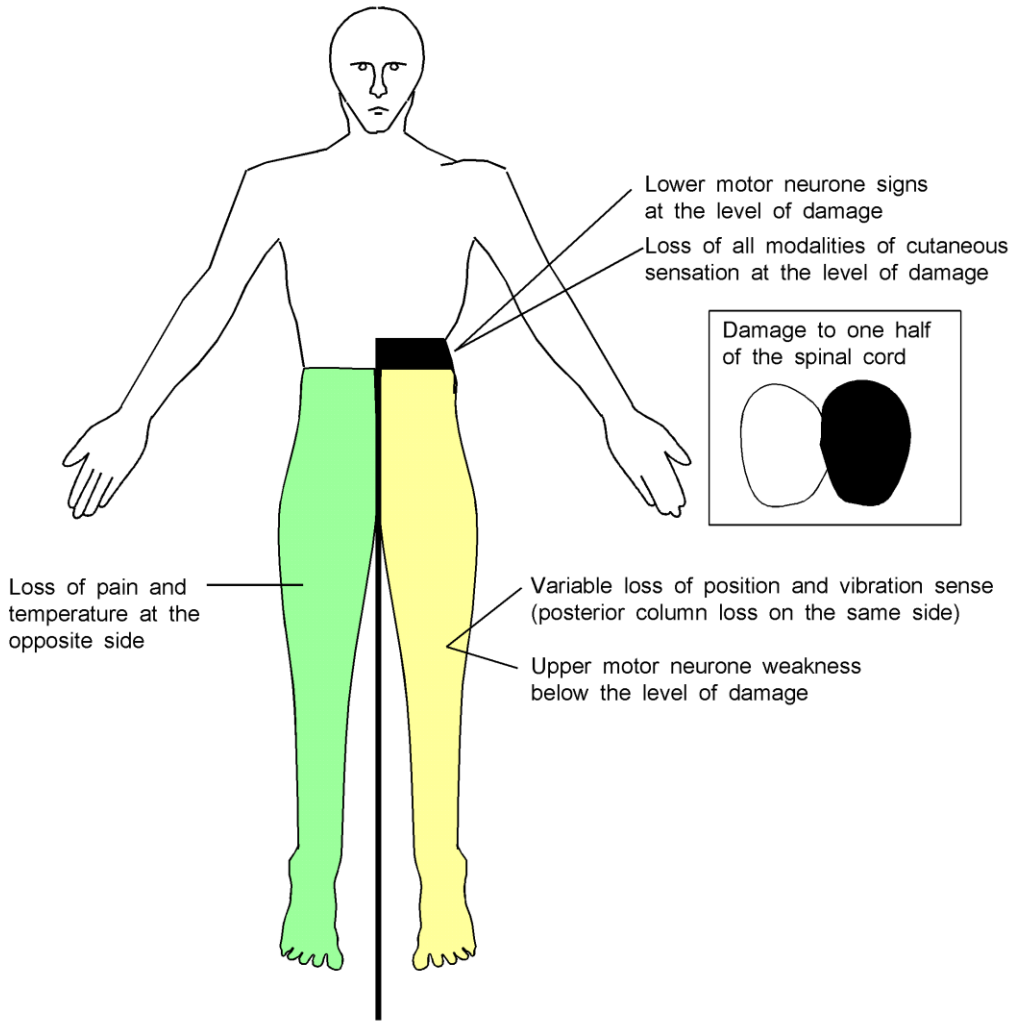
Muscle tone returns but less than prior to the transection as gamma efferent activity is lost

Flexor tone often exceeds extensor tone, thus there is paraplegia in flexion

Hemisection of the spinal cord (Fig. 16) produces:

- Loss of sensation and motor innervation and reflex activity at the level of the hemisection on the same side
- Upper motor neurone type paralysis of muscles below the hemisection on the same side
- Loss of pain and temperature on the opposite side
- Loss of joint position and vibration sense on the same side

Figure 16. The features of hemisection of the spinal cord - the Brown-Sequard syndrome



Because the spinal cord axons cannot usefully regenerate established changes are permanent.

Basic structure and function of the spinal cord

Figure 14 illustrates the anatomy of the spinal cord. In early quadrupeds impulses needed for fast communication to coordinate movement, mostly of the tail (and thus assist balance), were notably:

- vestibulospinal (for equilibrium)
- reticulospinal, olivospinal (part of the extrapyramidal tract controlling movement)

- rubrospinal (basically reflecting influence of corpus striatum and perhaps cerebellum, mostly to distal limb muscles)
- tectospinal (derived from visual information)

These pathways still persist in man but the anatomical tail has been replaced by the "physiological tail" of the cerebellum. The final common pathway to striated muscle is provided by anterior horn cells and their (lower) motor neurones which supply skeletal muscle fibres. The force of muscle contraction depends on the number of muscle fibres activated and the frequency of nerve impulses received.

The corticospinal (pyramidal) tract mostly crosses the midline in the medullary decussation (Fig. 5) developed later in mammals. It is the only uninterrupted descending pathway from the forebrain. Most of the descending motor pathways have to cross superior to the cervical part of the spinal cord so that the upper limbs receive appropriate motor instructions from the contralateral cortex. Some corticospinal fibres do not decussate in the medulla but pass caudally as the anterior corticospinal tract and cross the midline at the level of "their" anterior horn cells. The descending tracts (which are more medially situated than most of the ascending tracts (Fig. 14) influence the final common pathway of output from the anterior horn cells. Motor nerves which drive fine movements have fewer muscle fibres to innervate (for example in the muscle that move they eye there is a one nerve to one muscle ratio). About half of the descending fibres drive the arms and about one third drive the legs (reflecting the relative complexity of actions). It must be stressed that neither structure nor function is as precisely localized as these diagrams may suggest.

Sensory nerves feed into the *dorsal* columns of the spinal cord, the motor nerves leave from the *ventral* columns and the visceral output leaves from the lateral horn of the spinal cord between these two. This scheme persists in the medulla in a modified fashion. Somewhat similar nerve outgrowths occur in the head segments to form trigeminal, facial, glossopharyngeal and vagus nerve sensory ganglia (link).

Somatic: bodily, usually referring to skeletal muscle Visceral: refers to the inner organs

Joint position sense, vibration sense, and some (fine) touch ascend the posterior columns of the spinal cord (Fig. 14) without crossing the midline to the medulla where they cross the midline (Fig. 5) in the medial lemniscus. Some (crude) touch ascends in the anterior spinothalamic after crossing the midline.

Pain and temperature information crosses the midline shortly after entry into the spinal cord and ascend in the lateral spinothalamic columns (Fig. 14). These spinothalamic ascending fibres rejoin their initially non-crossing sensory partners in the thalamus.

Some diseases, such as syringomyelia, damage the center of the spinal cord by causing a cavity (syrinx) which impairs or abolishes the crossing pain and temperature sensation but leaves the non-crossing touch sensation intact (such patients usually unknowingly burn themselves by touching hot objects). This dissociated sensory loss may be confined to the arms and hands because the crossing fibres from the legs are situated more laterally than those from the hand and are thus away from the central

damaging syrinx. Syringomyelia usually occurs in the cervical cord producing wasting of the small muscles of the hand (caused by involvement of the anterior horn cells) and loss of pain and temperature in a "cape" distribution, often extending up the back of the head and often down to involve the hands..

Spinal cord problems

- Problems caused by an inadequate blood supply usually cause death of the relevant part of the cord. The anterior spinal artery supplies the anterior two thirds of the cord and the two posterior spinal arteries supply the posterior cord. Haemorrhage in or around the cord is unusual
- Inherited degenerations of parts of the cord can occur, often affecting specific tracts and their nuclei in the brain or brainstem
- Spina bifida, a condition in which the arches of the vertebrae fail to fuse, mayl cause spinal cord signs
- Inflammation (myelitis) usually is caused by viruses although in the past tabes dorsalis (caused by syphilis) used to affect the dorsal roots (to cause shooting "lightning" pain) and posterior columns (to cause loss of joint position sense, hence the characteristic stamping gait). Syphilis could also cause masses, gummata, that presented as space occupying lesions, or meningovascular damage, or meningoencephalomyelitis
- Deficiency of vitamin B12, usually associated with pernicious anaemia, produces subacute combined degeneration of the cord with changes in the posterior and lateral columns and signs of both peripheral nerve damage and spinal cord degeneration
- External or internal trauma may damage the cord directly or, by secondary pressure, by interrupting blood flow
- Tumours in the cord itself are rare (problems are more likely to be caused by extrinsic compression)

SENSATION AND REFLEXES

Detectors can be:

- Exteroceptive (reporting from the external environment) and include touch, pressure and, pain plus distance sensation of vision, hearing and smell
- Interoceptice (reporting from the internal body organs)
- Proprioceptive (=to take one's own) sensors report position of the body in space and bodily movements

There is a large variety of detectors. There are distinct detectors for:

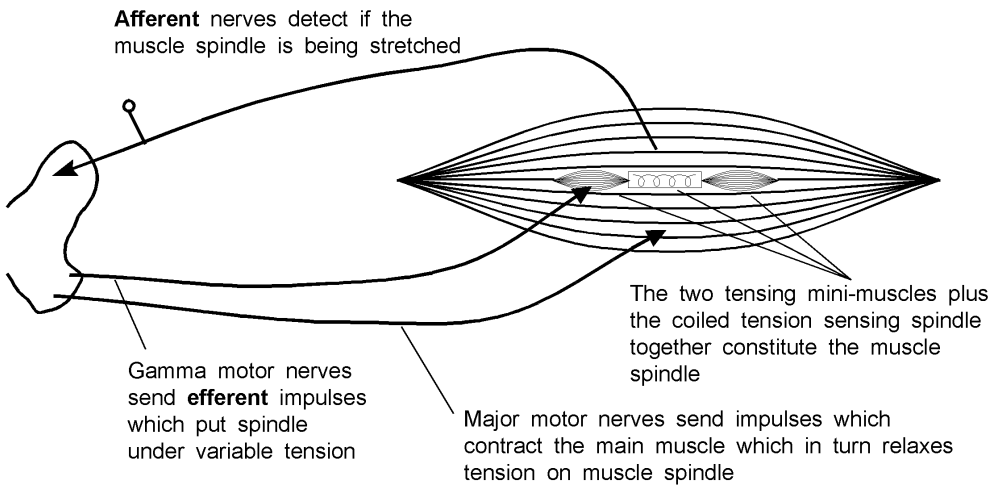
- Touch
- Noxious stimuli (pain if perceived by the cerebral cortex)
- Temperature
- Chemical stimuli
- Mechanical stimuli (stretch etc.)
- Vibration (the forerunner of hearing which probably evolved from tactile sense organs which reported on the frequency of touching).

There are three proprioceptive detectors. These are:

- Muscles spindles (Fig. 17) which detect muscle tension
- Joint receptors

- Tendon receptors

Figure 17. The muscle spindle mechanism for regulating muscle tone



Information is also gathered about the degree of contraction of muscles and their position in space. The semicircular canals of the ear also have a proprioceptive function. All detectors (except olfactory cells) pass impulses onto their sensory neurones.

Irritation of a sensory nerve at any point along its course will generate centripetal impulses along the nerve which will be interpreted and localized as if they had come from the detector. If pain is perceived (wrongly) to be coming from the peripheral detector it is known as "referred pain." Noxious stimuli can be reacted upon reflexly but *appreciation* of pain has to involve the cerebral hemispheres. If a sensory nerve is cut, spurious impulses generated at the site of the cut may give rise to a central appreciation of pain in the area supplied by the nerve (in the case of amputees giving rise to "phantom limb pain").

Pain from solid viscera such as the liver, spleen or lungs (which lack pain detectors) is usually caused by stretching of their capsules or from stretching of associated tissues. Hollow viscera such as the gut or ureters possess pain-reporting stretch receptors. In general pain from viscera passes centrally along with sympathetic nerves, pass uninterrupted through the sympathetic trunk, and/or thereafter run in the posterior nerve roots along with somatic pain fibres. This shared somatic and visceral input and processing explains why (visceral) heart pain is referred to the (somatic) arms.

Inflammation causes pain by liberating various substances that lower the threshold for impulse production in relevant nerves or detectors. It is possible that some pains can inhibit other pains. Superficial pain caused by rubbing the shin can dampen down the more severe boney pain caused by a kick to the shin. Acupuncture increases descending inhibitory impulses in the spinal cord (as well as inducing morphine-like substances, endorphins) whereas morphine itself activates descending inhibition of

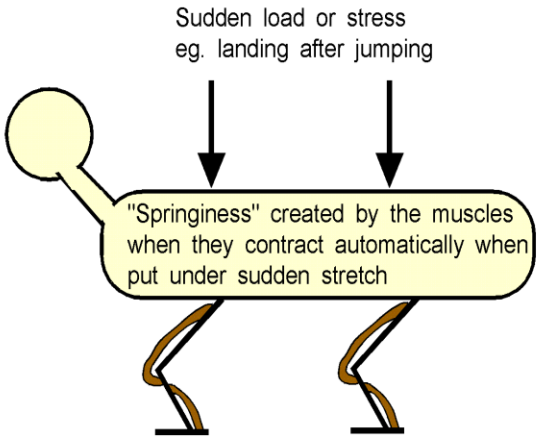
sensory nerve imulses. *Dependence* on opiate drugs depends upon the drug acting as a pseudotransmitter which blocks the action of the usual pain inhibiting neurotransmitters. Hence withdrawal of the opiate causes pain.

There are more general senses than those possessed by humans! Some animals can sense electrical or magnetic fields, and no doubt the lateral line sensation of fish is different to any human sense.

REFLEXES

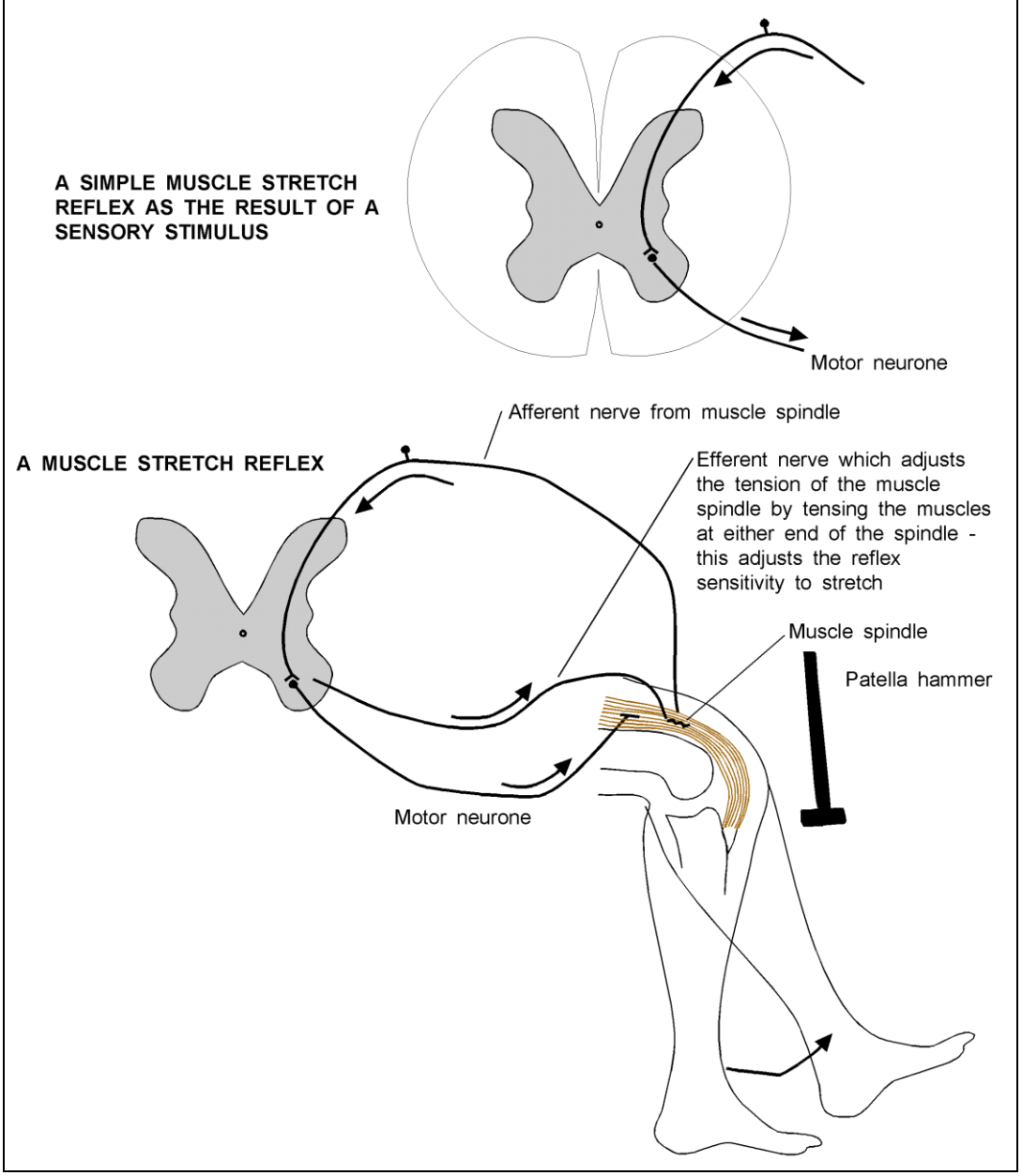
A reflex is an involuntary, rapid, stereotyped response to a specific stimulus. Skeletal muscle reflexes developed in land based animals as a response to gravity so the muscle length and/or strength (and thus posture), could be maintained (Fig. 18).

Figure 18. The (striated) muscle reflexes and posture maintenance



Skeletal muscle stretch reflexes depend upon sudden stretching of the coiled tension sensing muscle spindle cells within striated muscle. Clinically a striated muscle is abruptly stretched, usually by hitting its tendon with a soft hammer. The normal response is for the muscle to contract briefly. The muscle spindle cells send sensory information of rapid stretch to the relevant segment(s) of the spinal cord and, after transmission to the anterior horn cells an involuntary contraction of the stretched muscle results (Fig. 19).

Figure 19. The simplest version of the muscle stretch reflex. The reflex arc involves two neurones. In practice many other descending fibres impinge and modify anterior horn celll output. Additionally skeletal muscle reflexes depend upon reciprocal relaxation of antagonist muscles (that would otherwise block movement of the reflexly contracting muscle)



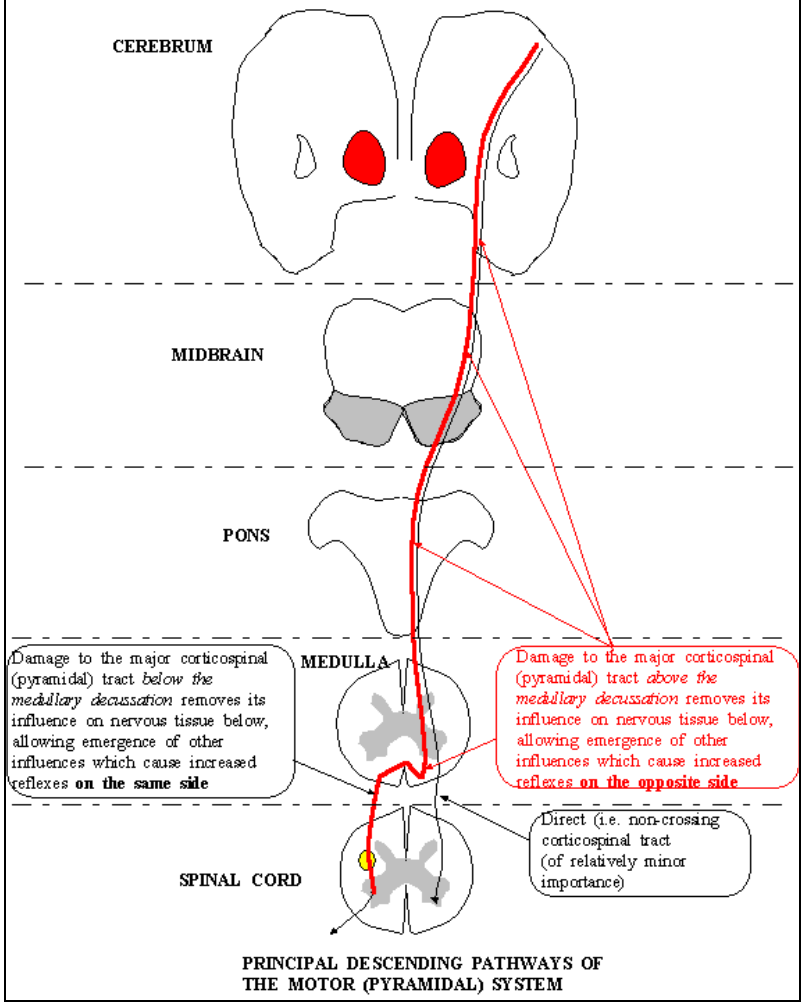
At its simplest, the spinal reflex could involves a sensor, a sensory (afferent) neurone, one synapse to an anterior horn cell with its motor neurone and an effector mechanism. In practice there are one or more (internuncial) neurones. This allows extra inputs to the motor neurones to provide variability and flexibility. As this involves only two or three motor neurones (out of 10^{11} neurones) this network is highly significant. The more nerve cells (axons, dendrites and synapses) that are involved in a reflex, the more that reflex can be modified.

To maintain posture the various striated muscles have to be kept at varying levels of contraction (tone). To do this the tension of muscle stretch receptors is varied by

gamma efferent impulses. Firing of gamma efferents stretches the muscle spindle sensing apparatus and, if sufficient numbers of tension sensing detectors are stretched then the whole muscle contracts to relieve the tension on the spindle sensory apparatus (Fig. 17). At rest muscles have a background resting tone by unconscious regular firing of anterior horn cells (if a group of anterior horn cells fire simultaneously then muscle twitching or tremor results).

Muscle stretch reflexes are normal, exaggerated, reduced, or absent. Exaggerated reflexes are characteristic of upper motor neurone damage. Whilst relaying in the spinal cord the reflexes are usually altered by local mechanisms or by descending (usually inhibitory) influences which usually inhibit the motor response and thus muscle contraction. If these descending inhibitory influences are interrupted by damage above the medullary decussation the reflexes of the opposite side limbs are released from descending inhibition and are increased and muscle tone is also increased (Fig. 20).

Figure 20. Damage to the corticospinal fibres allows mechanisms to come into play which increase the reflexes, the side depending on the site at which the corticospinal tract is damaged.



If such a hypertonic muscle is put under sustained stretch (for example stretching the triceps by flexing the elbow) then a protective complete collapse of muscle tone may occur - the claspknife reflex. A damaged reflex arc at a definite spinal cord level will interrupt reflexes at that level.

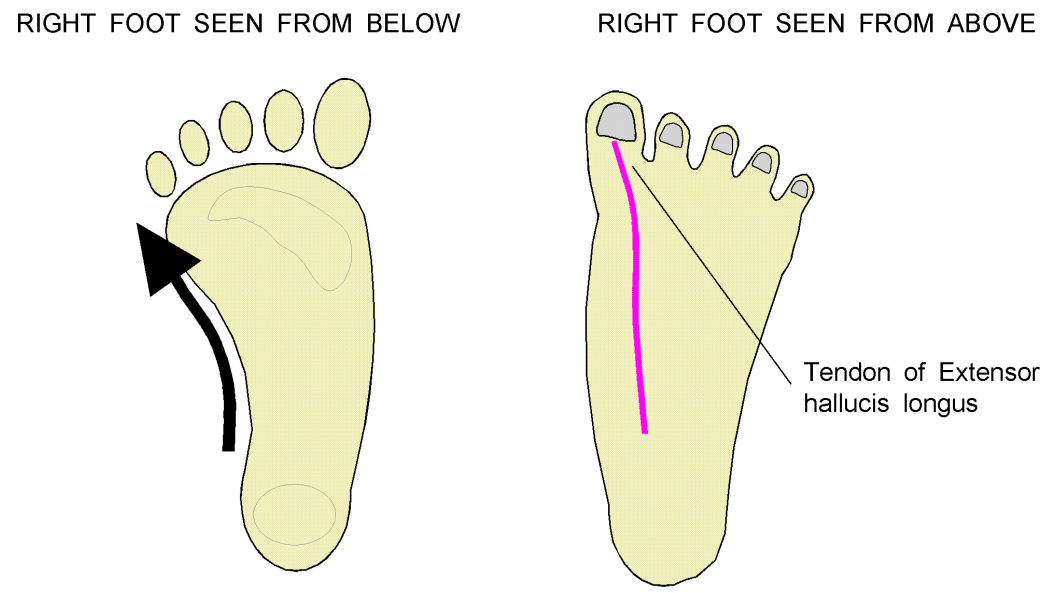
If a normal reflex can be elicited then the following all must be present and functioning normally:

- Peripheral detector (muscle spindle)
- Sensory innervation
- Spinal cord at that level
- Descending influences acting on that level of the spinal cord
- Motor innervation
- Ability of muscle to contract

Withdrawal reflexes to noxious stimuli occur at a spinal or medullary level and are not usually amenable to conscious control. Nearly all of the routine unconscious movements of the body depend on reflexes of one sort or another. For example sneezing, vomiting, and coughing all may be purely reflexive which conscious effort cannot suppress.

The plantar reflexes (Fig. 21)

Figure 21. The Babinski response



The outer border of the medial side of the foor is stimuated with a blunt object that is stroked from near the heel to the base of the little toe. The crucial observation is the first response of the Extensor hallucis longus, the muscle that moves the big toe towards the head. If its first observable response is to contract then the response is extensor. If relaxed, flexor

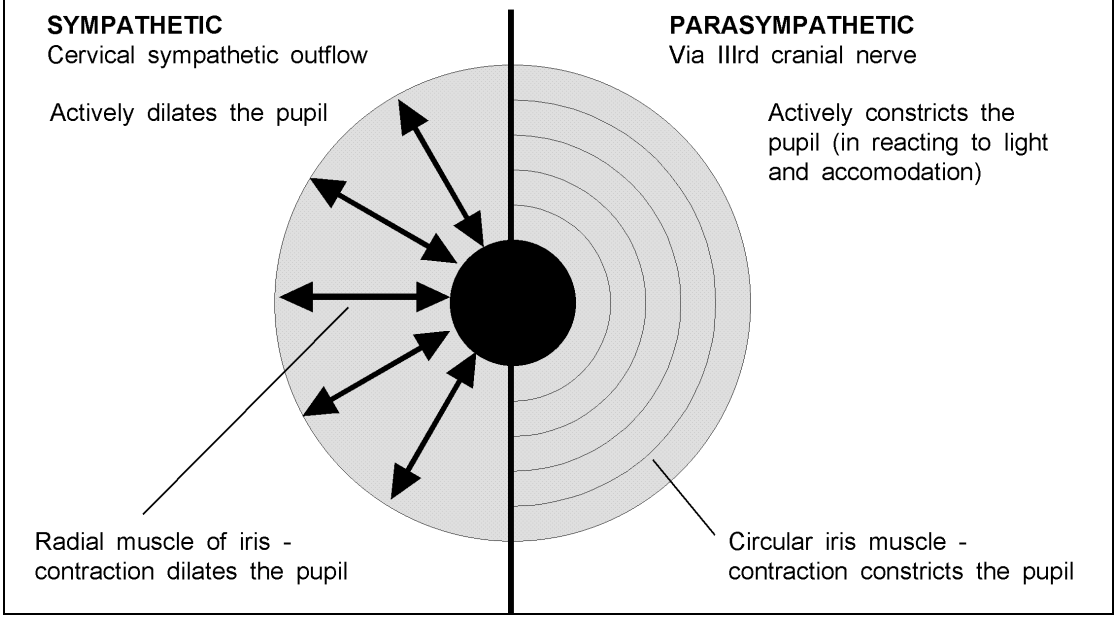
The normal plantar response is an a flexion of the great toe when the lateral border of that foot is stimulated from heel to great toe. A pathological (Babinski) response, signifying a disturbance affecting the relevant corticospinal tract is extension of the great toe caused by contraction of extensor hallucis longus (looking for contraction of this muscle or movement of its tendon may be more useful than observing the response of the toe). In most mammals and human neonates there is a defensive

reflex flexion of the leg in response to painful stimuli. This flexion reflex includes contractions of all muscles which shorten the limb and this shortening response includes (despite its anatomical name) contraction of extensor hallucis longus. As the corticospinal tract matures in the human, this reflex changes, with flexion of the great toe becoming dominant to produce the normal flexor plantar responses. If there is corticospinal damage then the more primitive response may re-emerge to produce extensor plantar responses.

In evolutionary terms the flexion withdrawal with upgoing plantars was a primitive protective reflex to avoid injury. Later, as a walking or swinging life in the trees evolved, our ape-like ancestors evolved a more appropriate local flexion "grasping" response when the sole of the foot was stimulated which almost certainly included flexion of the great toe as well. If our central nervous system becomes more primitive because of damage (or merely by being asleep) then the more primitive response emerges. The concept that when we awake and get up in the morning we are repeating, neurologically speaking, our ancestors' climb into the trees is appealing! Which of us would have discovered this response? Presumably Babinski stroked his patients all over before happening on this response!

Pupillary light reflex (Fig. 22)

Figure 22. The Anatomy and physiology of pupilloconstriction and pupillodilation



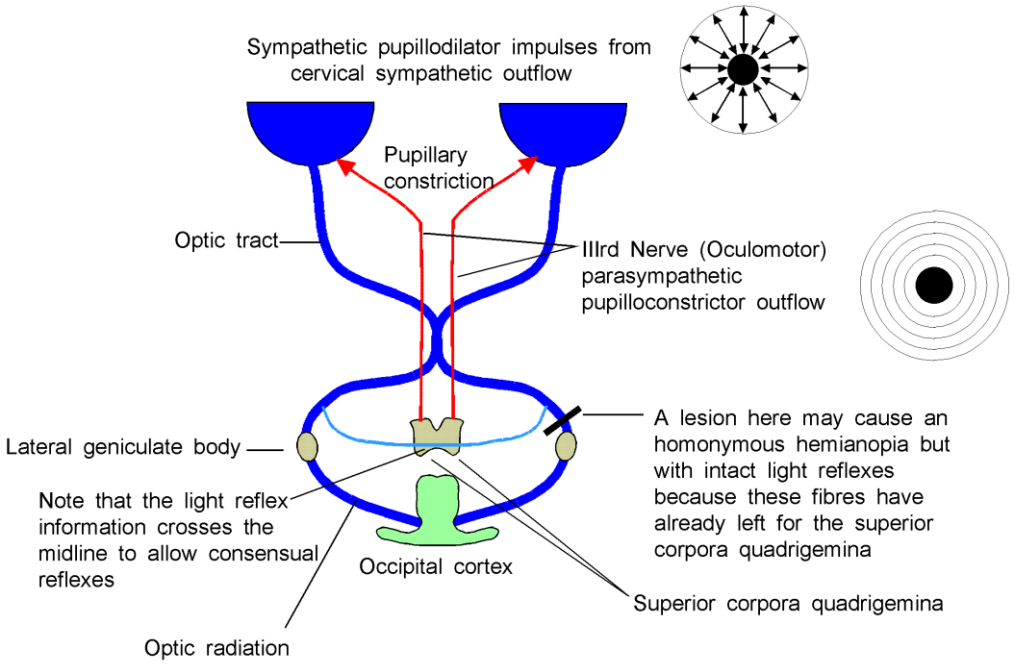
When one eye is exposed to a bright light both pupils constrict. Loss of this reflex implies a problem at a site or sites in the reflex pathway which comprises (Fig.23):

1. The retina
2. The II (Optic) nerve
3. The Optic chiasma
4. A small portion of the Optic tract to the lateral geniculate body

5. The superior corpora quadrigemina and the Edinger-Westphal nuclei (where information crosses the midline, explaining the normal contriction of both pupils to a light shone in one eye)
6. The pupilloconstrictor part of the III nerve nuclei
7. The III nerve which carries with it parasympathetic nerve fibres which constrict the pupil

Figure 23. The anatomy of the pupillary light reflexes

Midbrain at the level of the superior corpora quadrigemina

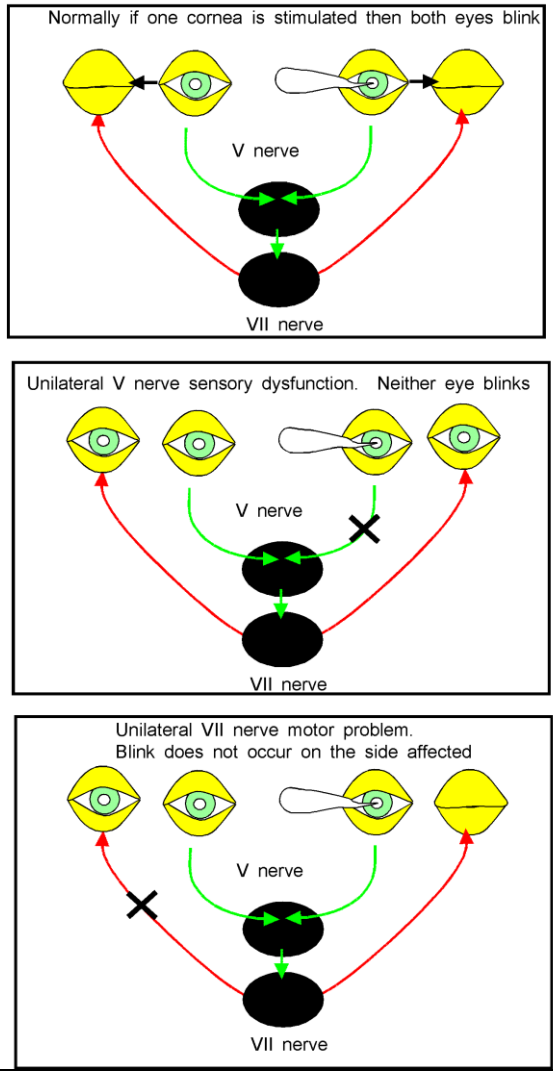


If there is damage to any of the first four sites then the pupillary reactions *on both sides* will be impaired or absent. If there is damage at any of the last three sites then the pupilloconstriction will be impaired or absent on the side affected. Predictably bilateral damage to the occipital cortex may result in the patient being blind but with intact pupillary reflexes.

Corneal reflex.

Corneal sensation is via the V nerve and the motor supply to blink the eye is by the VII nerve. Normally when the cornea is irritated on one side then both eyes blink. If there is V nerve damage on one side then when the cornea of that side is irritated then *neither* eye will blink. If there is VII nerve damage on one side then blink will not occur on that side no matter which cornea is irritated (Fig. 24).

Figure 24. The corneal reflex. Differences between sensory and motor impairments



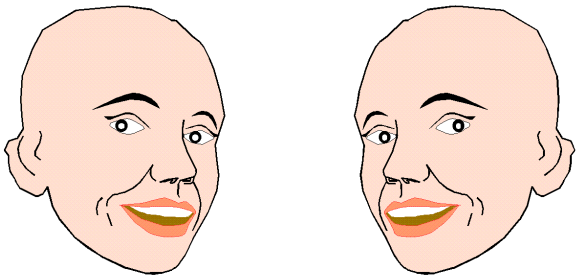
Glabella tap reflex

If the area just above the bridge of the nose is tapped from above (to ensure that the tapping does not cause a visually induced reflex blinking) then both eyes blink, but normally only for the first two or three taps. This reflex protects the eyes from a possible noxious stimulus. With late Parkinson's disease and in some patients with severe cerebral degeneration the blinking continues in response to each tap no matter how many taps are given.

Oculocephalic "Doll's eye" reflex

With a patient lying on his back, the eyes tend to look upwards if the neck is flexed or rotated. This reflex relies on vestibular input and the ability of the extra-ocular eye muscles to move the eyes. This reflex can be used to test eye movements in patients with impaired conscious levels (Fig. 25).

Figure 25. The doll's eye reflex



The pupils remain looking straight ahead on turning the head to either side or when flexing or extending the neck

Oculovestibular reflex

Irrigation of the external auditory meatus with hot or cold water causes nystagmus (and possibly associated vertigo). This is a test of the vestibular apparatus, the VIII nerve and their nuclei in the pons.

Jaw jerk

If the muscles that close the jaw are rapidly stretched (by tapping on the chin) there is a reflex jerk. Both sensory and motor aspects are served by the V nerve. If the jerk is pathologically brisk then there has been bilateral damage above the V nerve nuclei in the brainstem (importantly a positive jaw jerk implies pathology above the level of the neck).

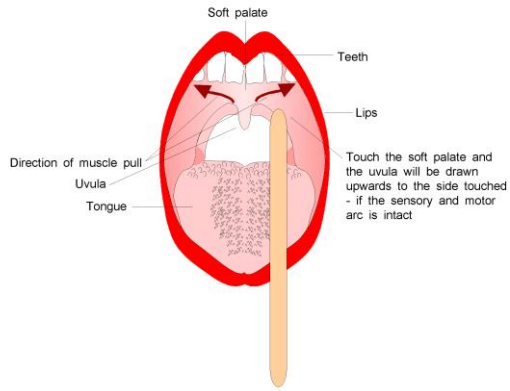
Sucking, rooting and snout reflexes of infancy

In infancy if an object contacts the lips there is a sucking action of the lips, tongue and jaws. A rooting reflex occurs when the head turns to allow the lips to pursue a tactile sensation just lateral to the mouth. A snout reflex is a pouting of the lips if the centre of closed lips is tapped. These reflexes may reemerge if there is severe central nervous system damage.

Palatal reflex

On stimulation of the palate the uvula is pulled upwards towards the normal side (Fig. 26). The sensory nerve is V and the motor X.

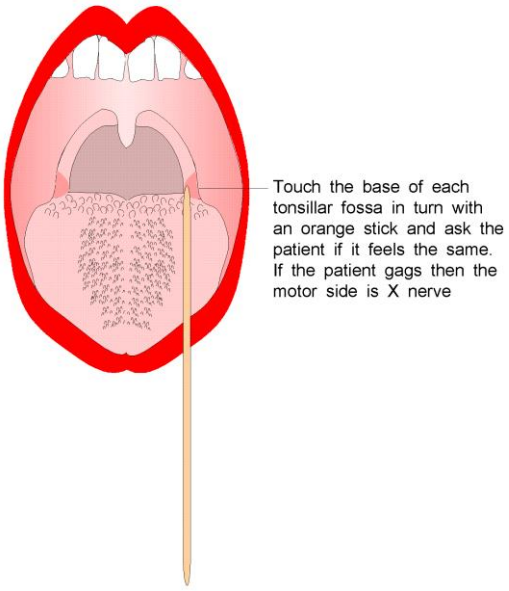
Figure 26. The palatal reflex (sensory V, motor X)



Pharyngeal "gag" reflex

On stimulation of the posterior pharynx there is constriction of the pharynx. The sensory nerve is IX and the motor nerve is X. Normally when the pharynx is stimulated on one side then both side constrict. If there is IX nerve damage on one side then neither side will constrict when the pharynx of that side is stimulated, and if there is X nerve damage on one side then constriction will not occur on that side no matter which side of the pharynx is stimulated (Fig.27).

Figure 27. The pharyngeal reflex (sensory IX, motor X)

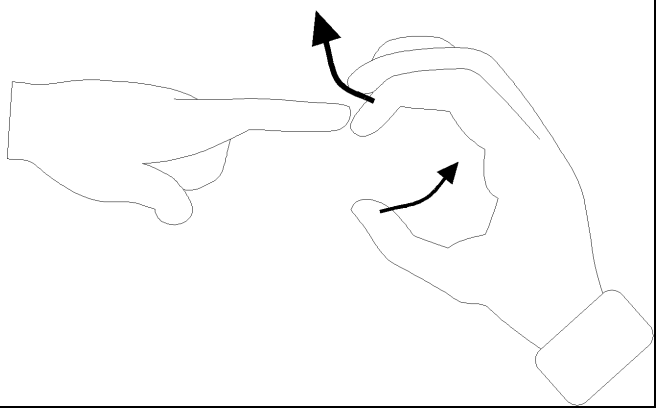


Hoffmann's reflex

If semiflexed fingers are suddenly stretched by tapping the distal (concave) tips then the finger flexors contract and, if the contraction is abnormally brisk, would suggest corticospinal problems. Hoffmann's reflex is an additional adduction and flexion of the thumb when eliciting finger jerks (Fig. 28).

Figure 28. The Hoffmann reflex

The flexor muscles of the index or middle finger are rapidly stretched when the pressure on the tip of the index finger is suddenly released. If there is an upper motor neurone lesion affecting these muscles then an exagerated muscle stretch reflex is evoked, and the reflex spreads to involve other muscles, notably those that cause the thumb to move towards the palm



Usually this reflex is elicited by flexing a distal phalanx on the middle phalanx and allowing it to flick to achieve a straight finger. This causes a sudden stretching of the finger flexor and the reflex response spreads to involve muscles other than those

stretched (usually only those sharing a root value). This spreading "radiation" of a reflex response may be found in cortocospinal lesions.

Inverted reflexes

Spinal cord damage at a specific level interrupts the reflex arc relevant to that level, but stops descending inhibitory influences from affecting the level below, which therefore exhibit enhanced reflexes. When there is damage at C5,6 level an attempt to elicit the biceps reflex (C5,6) by percussing the biceps tendon, fails but the slight stretching of the triceps (C7,8) caused results in an exaggerated contraction of triceps which extends the elbow joint contrary - an "inversion" - to what is expected.

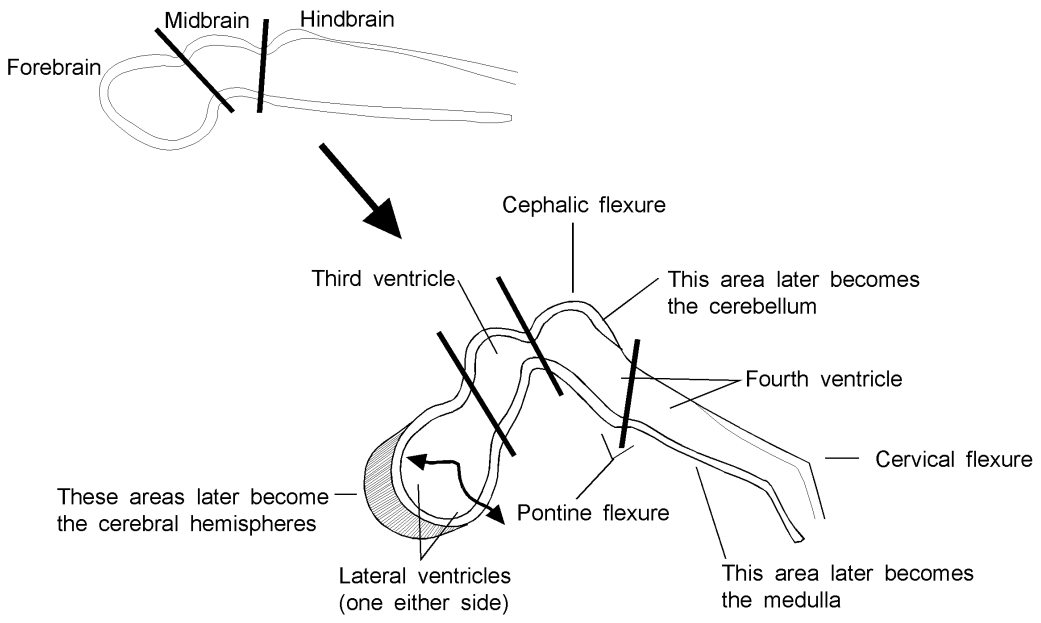
In principle every muscle that can be suddenly stretched should show a reflex contraction. Numerous other reflexes are described each of which can be used to confirm intact sensation, sensory nerves and motor nerves, spinal cord and muscle.

THE BRAIN (SOME FURTHER CONSIDERATIONS

The human brain is the most complex device known to man. It contains 10^{10}-10^{12} neurones, each of which can have up to 100,000 synapses impinging upon them. It is estimated that 3,000 million impulses reach the brain every second.

Figure 29 is a schematic view of the embryonic brain which in essence begins as a hollow tube. Three expansions occur at the cephalic end of the neural tube which then go on to form the fore, mid, and hind brain. The rest of the neural tube develops into the spinal cord.

Figure 29. The development of the mammalian brain



The basic central nervous system pattern is that the fore and midbrain deal with information about the distant environment (in our ancestors this was information gained by means of sight or smell). The hindbrain, notably the medulla, processes information about the immediate environment (with information about temperature,

touch, taste and balance) for quick reflex responses which occur before the cortex is informed.

The brain receives information and decides whether to:

- Accept it and/or
- Process it and/or
- Store it and/or
- React reflexely and/or
- Communicate to other parts of the brain, especially to the cerebral cortex if appreciation is to occur.

Headache

- There are no pain receptors in brain tissue. There are pain receptors in the meninges and possibly in the ependymal blood vessels. Pain receptors are found in arteries, veins, striated muscle around the head, paranasal sinuses, eyes and teeth. Thus headache caused by problems within the brain may be associated with:
- Space occupying lesions
- Stretched blood vessels
- Changes in intracranial pressure
- Infection or bleeding into the cerebrospinal fluid which affects the meninges
- Blood vessel dysfunction (migraine or accelerated hypertension)
- Blood vessel inflammation (arteritis)

Recurrent episodic headache may be caused by muscle tension, sinusitis, migraine or hangovers. Eye strain is a rare cause of headache.

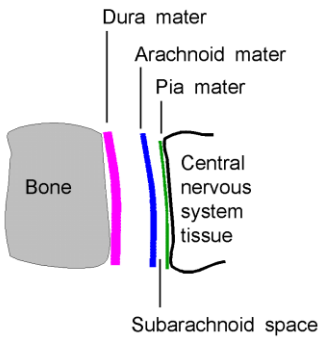
The ventricles

There are four ventricles (= small cavities), two laterally in the cerebral hemispheres and two midline (the third and fourth).

The meninges

There are three lining layers outside the central nervous system. The innermost is the pia mater which covers the nervous tissue, the middle layer is the arachnoid mater (inferior to which is the cerebrospinal fluid) and the dura mater which covers the inner surface of the bony skeleton surrounding the nervous system (fig. 30).

Figure 30. The meninges.
The lining layers outside the central nervous system



There is approximately 125 mls of clear and colourless cerebrospinal fluid (the fluid removed at lumbar puncture) in the subarachnoid space, the ventricles and in the central canal of the spinal cord. About 200 ccs of cerebrospinal fluid is formed daily by the vascular choroid plexus in the lateral ventricles and flows via the cerebral aqueduct into the 4th ventricle and exits via holes in the roof of the medulla into the subarachnoid space (Fig. 31). The cerebrospinal fluid is secreted by the choroid plexus of the lateral ventricles, absorbed by arachnoid villi in the superior sagittal sinus and is replaced several times each day. The cerebrospinal fluid acts as a supporter and "pressure buffer" of the central nervous system. Communication between ventricles and subarachnoid space is via a medial and two lateral foramina (= windows).

Bacterial meningitis

Infection may enter the cerebrospinal fluid as it is a place where invaders are, initially at least, at an advantage because immunoglobulins and complement (link) are normally absent. With bacterial meningitis the conscious level is often impaired as there is also an encephalitis (inflammation of brain tissue). With meningitis caused by tuberculosis there may be space occupying abscesses or large lesions called tuberculomas. Healing is by fibrosis (known as gliosis within the brain) and this, rather than the infection itself, may later cause problems including constriction of cranial nerves or blockage of blood or cerebrospinal fluid pathways, the latter to cause hydrocephalus (excessive accumulation of cerebrospinal fluid).

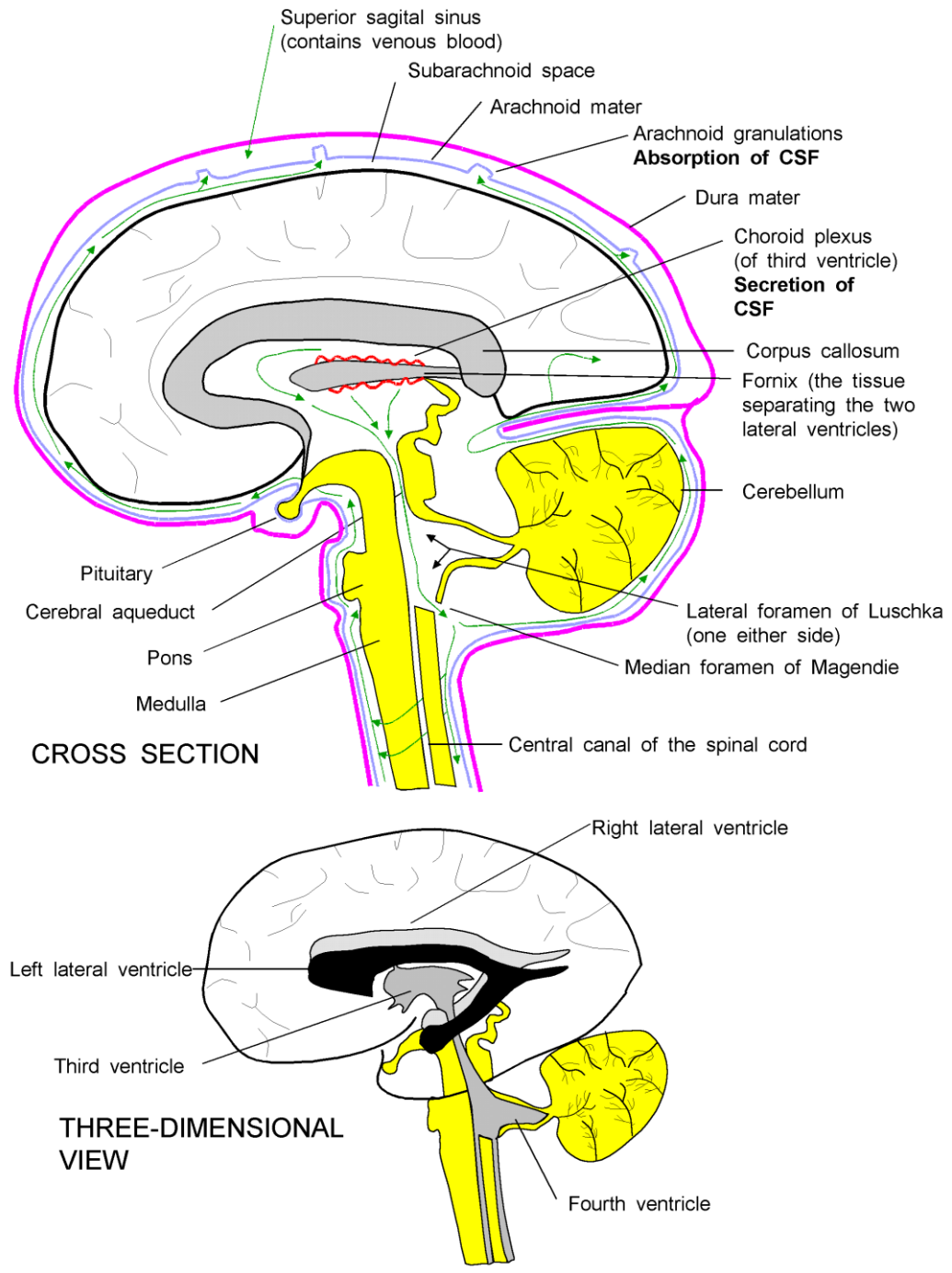
The forebrain

The forebrain comprises the two cerebral hemispheres (which grow at the most cranial part of the neural tube with a cavity in each which later becomes the lateral ventricles) the limbic system, the thalamus, the hypothalamus and basal ganglia.

The forebrain started evolving at the same time as filter feeders became active seekers of food, an activity requiring smell, hearing and vision. The olfactory bulbs lie beneath the anterior ends of the cerebral hemispheres. Smell, a chemical sensing of the environment, was a dominant early sense of multicellular life forms.

Caudal to the forebrain is the midbrain (in which the central cavity is the called the cerebral aqueduct) which mostly contains ascending and descending nerve tracts. Caudal to the midbrain is the hindbrain which comprises the medulla, pons and cerebellum. The fourth ventricle passes into the central canal of the spinal cord (Fig. 31).

Figure 31. Formation and flow of cerebero-spinal fluid (CSF)

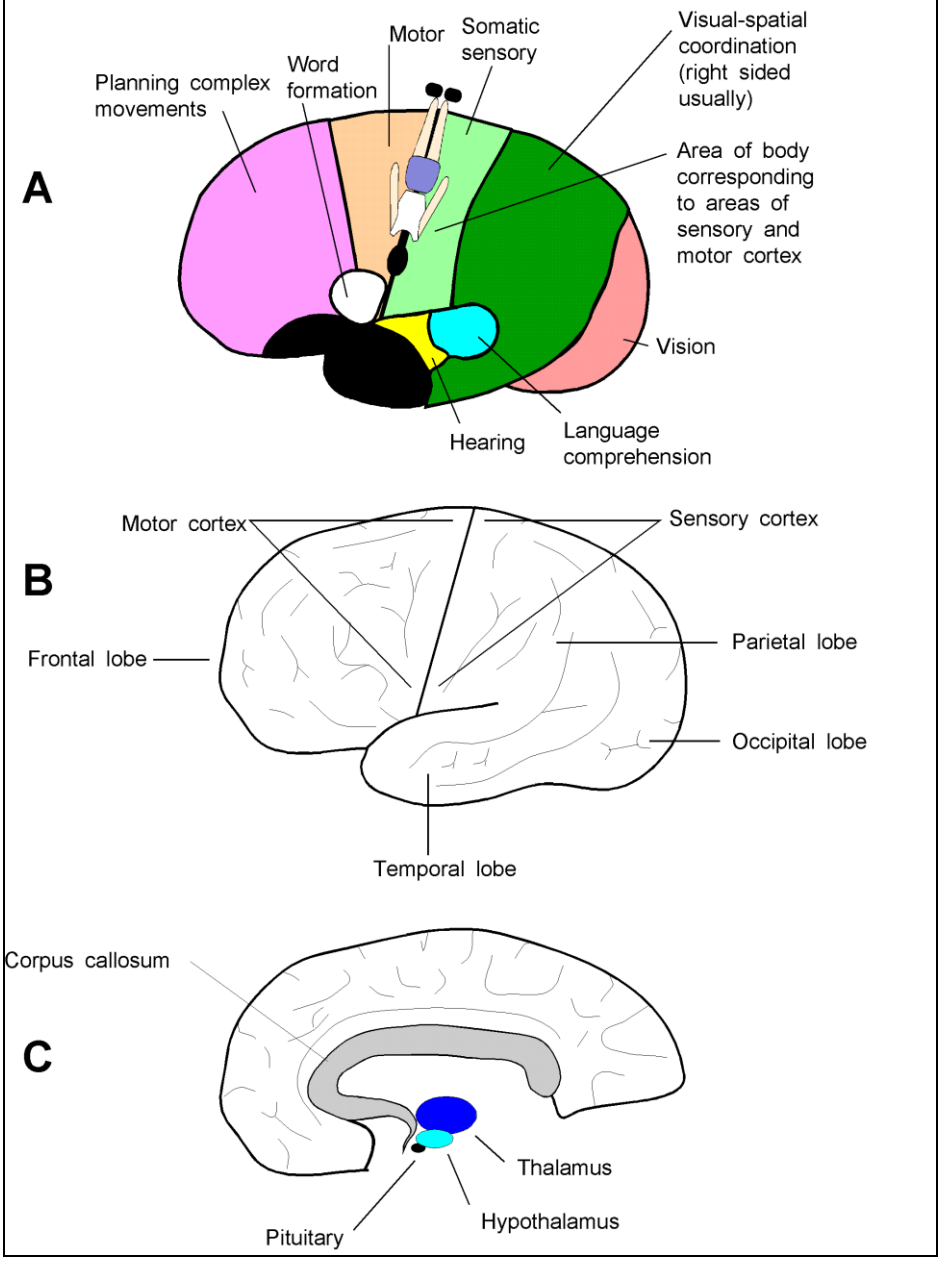


The cerebral cortex

The cerebral cortex (Fig. 32) is the convex surfaces of the cerebral hemispheres, which contains nerve cells and is about two millimeters thick with gyri (= folds) and sulci (= grooves) which increase the surface area. About 70 percent of all neurones in the central nervous sytem are in the cortex. Classically the cortex is divided into four regions, the frontal, temporal, parietal and occipital lobes. The cortex provides conscious thought and some (albeit limited) higher control for the primitive urges and reflexes of our distant ancestors.

Figure 32. A. and B An external view of the brain as seen from the left showing the structure and some functions of the cerebral cortex

C. A view of the brain as if sectioned along the midline



The *frontal* lobes control behaviour, intellect, mood responsiveness, and appropriate bladder and bowel function. Frontal lobe damage may cause a contralateral hemiparesis (paresis=lack of movement), inappropriate behaviour, a dulling of intellect, blunted emotional responses, and incontinence or a tendency to pass urine or stool inappropriately.

The *parietal* (=pertaining to the walls of a cavity) lobes provides cortical type sensation, visiospatial coordination, recognition, and movement. Cortical type sensation is the ability of the cortex to integrate all the various sensory stimuli to identify objects or the texture of objects placed in the hand, or numbers drawn on the

hand. The visual appreciation fibres, after passing through the optic chiasma (Fig. 23) spread out through the parietal and temporal lobes. A lower quadrantic visual field defect may result if the upper fibres of the optic radiation are damaged. Parietal lobe damage may cause defects in cortical type sensation, visiospatial incoordination, failure to recognize familiar objects, and other sensory dysfunctions. A lower quadrantic visual field defect may also occur if the upper fibres (those in the parietal lobe) of the optic radiation are damaged.

The *temporal* lobes provide some memory functions, and the temporal lobe of the dominant hemisphere provides sensory speech appreciation. An upper quadrantic visual field defect may result if the lower fibres of the optic radiation are damaged (Fig. 23). Temporal lobe damage may cause a sensory dysphasia and an upper quadrantic visual field defect may result if the lower fibres (those in the temporal lobe) of the optic radiation are damaged.

The *occipital* lobes provide appreciation of vision. Occipital lobe damage may result in loss of vision in one or both visual fields. The light reflexes are preserved (Fig. 23).

The motor cortex is in front of the major fissure and sends motor impulses to the anterior horn cells in ventral areas of the spinal cord (Fig 14) and to certain cranial nerves. The sensory cortex is behind the major fissure and receives input from the dorsal spinal cord and from relevant cranial nerves. All sensory nerves are connected directly or indirectly to the limbic system to integrate emotional responses.

The cortex is responsible for conscious initiation of movements, thinking, learning, and reasoning. Memory is widely distributed within,the hippocampus and the cerebral cortex being major sites. Memory obviously involves conditioning of certain pathways and of synapses. Interestingly short and long term memory are functionally differentiated (the former being particularly affected in dementia). The higher mental functions (which differentiate most of us from other primates) are mostly provided by our cerebral cortex, and include prediction of consequences, problem solving, abstract thinking, creative thinking, and our level of self awareness. However most of the body's cells can function in the short term without any input from the cerebral cortex. A cerebral cortex is not essential for life - some primitive animals have a minimal cortex and decapitated chickens can run around, albeit briefly.

Both hemispheres send down commands to initiate movements on the opposite side, but each hemisphere has certain other separate responsibilities. The dominant hemisphere (usually the left in right handed people) is responsible for:

- Sensory reception and initiation of motor output affecting the opposite side of the body
- Speech
- Writing
- Drawing
- Recognition
- Reading
- Calculation
- Analysis over time

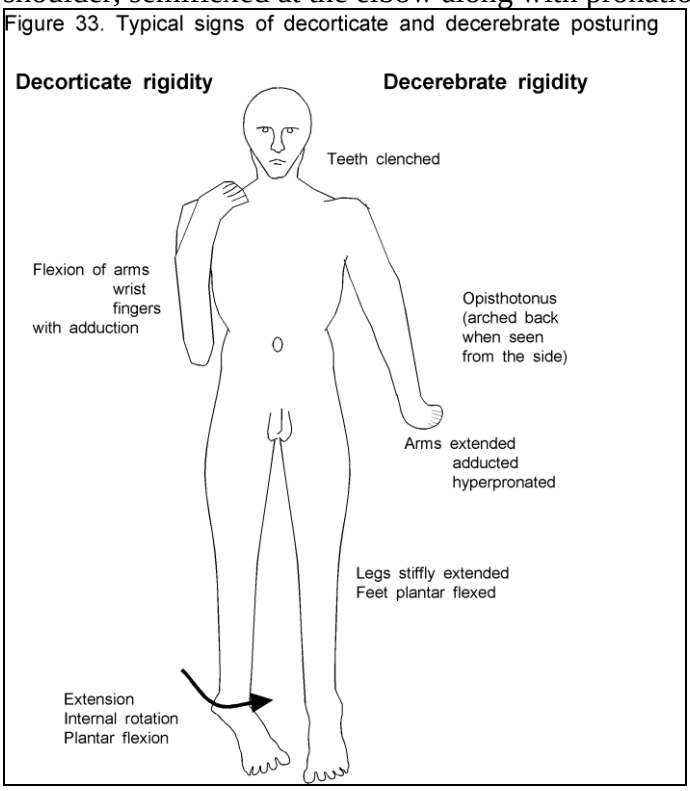
The non-dominant hemisphere is responsible for:

- Sensory reception and initiation of motor output affecting the opposite side of the body
- Visiospatial recognition and integration
- Artistic ability

If there is damage to the corticospinal tract (cortex downwards to, but not including, the anterior horn cell) then in the limbs affected (on the opposite side above the medullary decussation, on the same side below) tone is increased because of interruption of descending inhibitory influences to muscles affected, the reflexes are increased, there is little muscle wasting (the muscles are tense) and the plantar response is extensor. During sleep the corticospinal tracts are mostly inactivated, presumably to prevent us acting out our dreams

Decerebrate posture (Fig. 33) is caused by brainstem damage which results in unrestrained gamma efferent activity and generalized muscle contraction results. *Decorticate rigidity* causes the legs to be extended and the arm to be adducted at the shoulder, semiflexed at the elbow along with pronation of fingers and wrist.

Figure 33. Typical signs of decorticate and decerebrate posturing



The thalamus (Fig. 2)

The thalamus (= inner chamber) is essentially a relay station between cortex and the more caudal nervous tissues. At and above the thalamus all sensory modalities from each region of the body are brought together again for onward transmission for integrated processing.

Hypothalamus (Fig. 2)

The hypothalamus is physically and functionally between the spinal cord and the cerebral hemispheres. It has seven main functions:

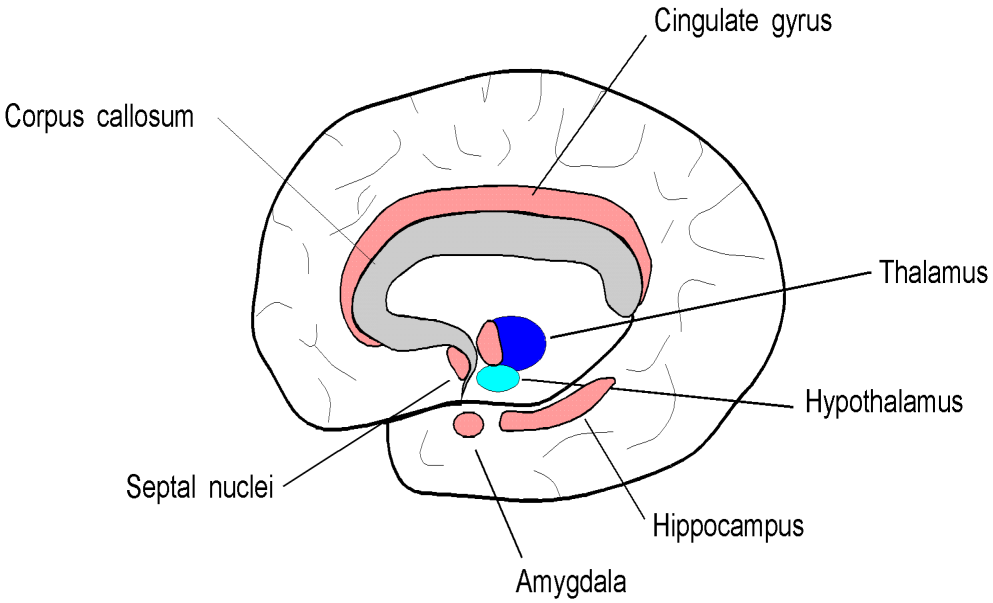
- Correlation of taste, smell, and visceral functions

- Control of the autonomic nervous system, including aspects of vasomotor, respiratory, pupillary, piloerection, genital function, bladder, gastric secretion, gut motility and bronchoconstriction
- Appetite
- Growth, via growth hormone
- Water and electrolyte balance via antidiuretic hormone
- Control of the pituitary hormonal secretion
- Body temperature

Limbic system (=border)
The limbic system surrounds the brainstem and comprises the hippocampus (an area extending throughout the length of the floor of the inferior horn of the lateral ventricle), the amygdala (=almond), the cingulate (=girdle) gyrus, and part of the anterior thalamus (Fig. 34).

Figure 34. The limbic system.

The definition of the tissues covered by this term is somewhat variable but is usually taken to include the amygdala, the cingulate gyrus, the septal nuclei, the hippocampus, and part of the anterior thalamus - all in



It is responsible for the autonomic components surrounding emotional behaviour, learning and motivation. It can in unusual circumstances even initiate movement - it is better and more convincing than the conscious efforts of the cortex at making us smile, laugh or cry. For example patients with hemiparesis can use limbic initiation of movement (rather than the corticospinally paralyzed facial muscles) to smile. Patients with Parkinson's disease who are immobile can use the limbic system to initiate faint motor responses if terrified.

The hindbrain (Fig. 2)
Modification of muscle movement is carried out by the cerebellum and the forebrain basal ganglia. Under normal circumstances neither initiates movements.

The cerebellum (=little brain) developed to integrate inputs from the vestibular apparatus and lateral line organ of fish. The cerebellum comprises two lateral hemispheres and a central area (the vermis) and receives inputs from the labyrinth of the ear and proprioception (muscle) sense organs of the trunk, along with input from touch, sight and hearing (but not smell). The cerebellum is a timing device which achieves motor coordination by temporal sequencing of nerve impulses initiated elsewhere thereby causing reciprocal relaxation or contraction of opposing muscles and by damping down oscillations. The cerebellum has an important role in regulation of gamma efferent activity (Fig. 17). The cerebellum is thus important for balance. Interestingly as the tail (an anatomical balancing organ) of our ancestors regressed at much the same time as the cerebellum (a physiological balancing organ) developed. The lateral hemispheres of the cerebellum (the neocerebellum) is mostly responsible for the distal limb muscles whereas the central vermis is mostly responsible for trunk muscles. The cerebellum stores up memory of specific sequences of movements.

With cerebellar damage there may be:

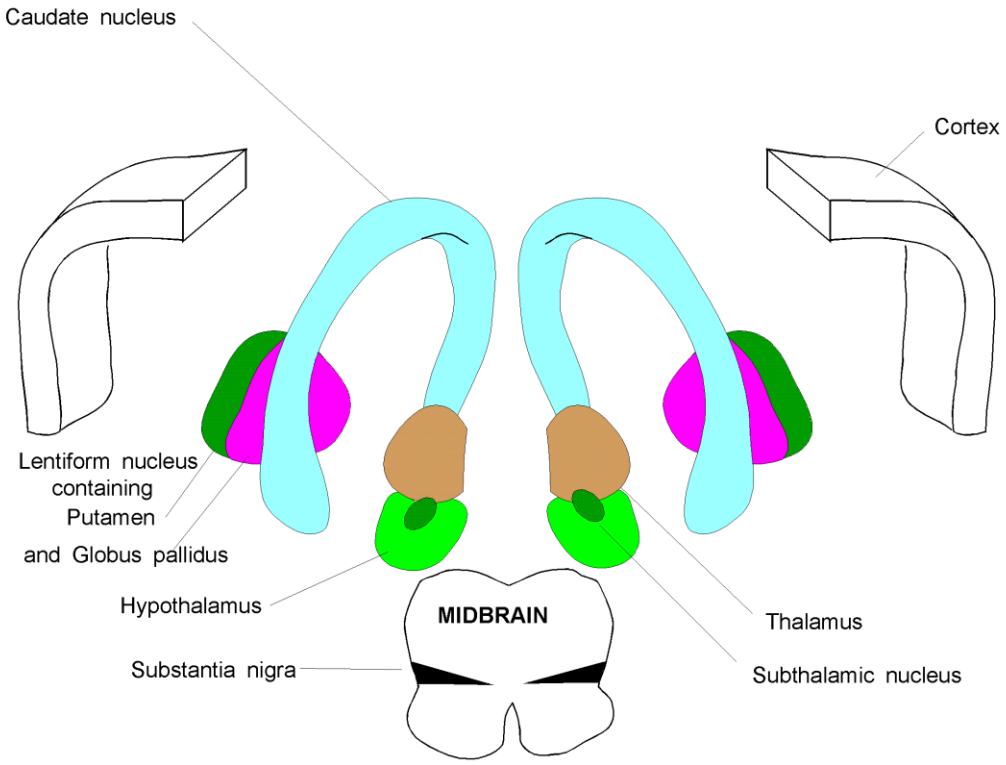
- Low muscle tone (hypotonia)
- Inaccuracy of movement with clumsiness, broken-up non smooth movements (dysynergia) and/or inappropriate force, past pointing (dysmetria), and intention tremor which develop or worsen when a limb is being used
- Coarse jerking eye movements (nystagmus)
- Unsteady gait (ataxia)
- Scanning or staccato speech (a tremor of speech)
- Incoordination of articulation (dysarthria)
- Incoordination of swallowing (dysphagia)
- Vertigo

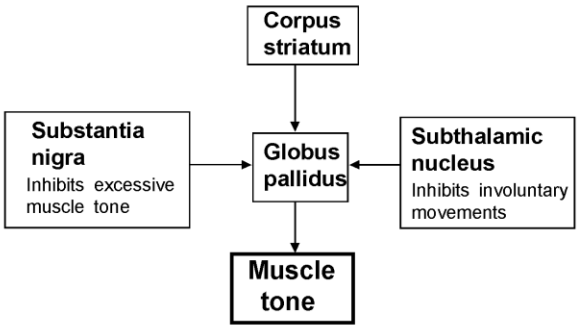
The basal ganglia

The basal ganglia (basal = at the base of the cerebral hemispheres), also loosely known as the extrapyramidal system (although the cerebellum should, but usually is not, included in this term) contain the basal nuclei (Fig. 35):

- The corpus striatum
- The amygdala (= almond shaped) an association area for olfaction
- The globus pallidus (= ball + pale)
- The caudate (= tailed) nucleus which arches over the rest
- Putamen (= shell)
- The subthalamic nucleus
- The substantia nigra

Figure 35. The basal ganglia and associated tissues. The extrapyramidal.system modifies the gamma efferents to the muscle spindle organs.





The basal ganglia collectively contribute to the smooth action of striated muscle groups and are dominated by the cortex which initiates non-reflex movements. The basal ganglia receive inputs from the reticular formation, the vestibular nuclei, optic and auditory reflex sites and the cerebellum. The basal ganglia are responsible for automatic control of posture whereas the cerebellum is responsible for modifying movement. Signs of basal ganglia dysfunction, are typified by Parkinson's disease in which there is "accelerated aging" of dopaminergic neurones in the sustantia nigra, causing:

- Tremor which is most marked at rest and reduced when the limbs are moved
- Reduced or absent ability to initiate movement (hypokinesia or akinesia respectively)

- Rigidity of cogwheel (rigidity + tremor) type
- Muscle stretch reflexes are normal
- Postural changes: flexion of neck, spine, arms and legs causing forward leaning and hence a rushing "festinant" gait as those affected try to catch up with their centre of gravity

The basal ganglia usually do not develop dysfunctions caused by an inadequate blood supply, but in contrast parts of the cerebellum (or its brainstem connections) may suffer ischaemic damage. Alcohol may damage the cerebellum, particularly the midline vermis.

The brainstem (Fig. 2)
The brainstem comprises the midbrain, pons and medulla. Within the brainstem all ascending fibres are arranged so that sensory inputs from one side of the body end up on the opposite side, and most descending fibres also cross (one exception is the anterior corticospinal tract which only crosses in the spinal cord at the level of their anterior horn cells).

The midbrain (Fig. 5) contains the internal capsule, a tract mostly derived from the motor cortex which passes downwards in between the basal ganglia, separating the caudate from the putamen (Fig. 35), then as the pyramidal system, the decussation in the medulla (and finally into the ventral spinal cord where the fibres synapse with the anterior motor horn cells of peripheral nerves). Most of the internal capsule comprises the corticospinal, corticopontine, and corticobulbar tracts.

The pons contains ascending and descending tracts and some nuclei, and has major connections with the cerebellum.

The medulla organises basic functions including:
- heart rate
- blood pressure
- breathing
- swallowing

All cranial nerves except I and II have stations in the brainstem. The brainstem is like a spagetti junction and, in addition, there are several nuclei therein. The enlargement of the brainstem in mammals took place because the ability to coordinate visual, cutaneous and proprioceptive information was advantageous. Not surprisingly a large number of symptoms and signs may result from brainstem dysfunction. These include:
- Strokes (sudden onset of focal neurological signs)
- Double vision (diplopia)
- Changes to facial sweating (sympathetic involvement)
- Facial weakness
- Difficulty in articulating words (dysarthria)
- Difficulty in swallowing (dysphagia)
- Limb paralysis
- Impaired conscious level
- Vertigo

- Death

CONNECTING ROUTES IN THE CENTRAL NERVOUS SYSTEM

Medial longitudinal fasciculus (Fig. 5)

The medial longitudinal fasciculus is the oldest and most constant tract in the central nervous system and is largely confined to the brainstem although it may reach the caudal spinal cord (as the medial vestibulospinal tract). Its main functions are 1) to connect the motor parts of the nuclei of the nerves that drive eye movements and the balance (vestibular) part of the VIII nerve and 2) on each side, to connect the motor parts of the V and VII nerve to the motor parts of the IX and X nerves to harmonize chewing, swallowing and speech.

The medial lemniscus (=ribbon)

Ascending fibres carrying joint position sense, vibration sense and fine touch end at the gracile and cunneate nuclei in the medulla (Fig. 5) and cross the midline where they join up with the ascending pain, temperature and crude touch to constitute the medial lemniscus which then carries all impulses from the skin and muscle of the opposite side. Other information routed via the medial lemniscus includes:

- sensory fibres of the V nerve, which have descended into the cervical cord also cross the midline and eventually join the medial lemniscus
- some fibres from the Xth nerve (mostly respiratory information)
- some fibres from the VII and IX nerves (mostly taste)
- Some fibres from the VIII nerve (mostly vestibular)

The conjoined ascending fibres reach the thalamus from where they can be relayed to the cortex.

Reticular formation

The reticular (=netlike) formation is a collection of irregular columns of nervous tissue which functionally extend from the medulla to the midbrain and pons.

The reticular formation remains independent of domination by any one system but rather is a final common pathway conveyance system which allows specific impulses to be disseminated throughout the central nervous system and provides a linkage between most parts of the brainstem.

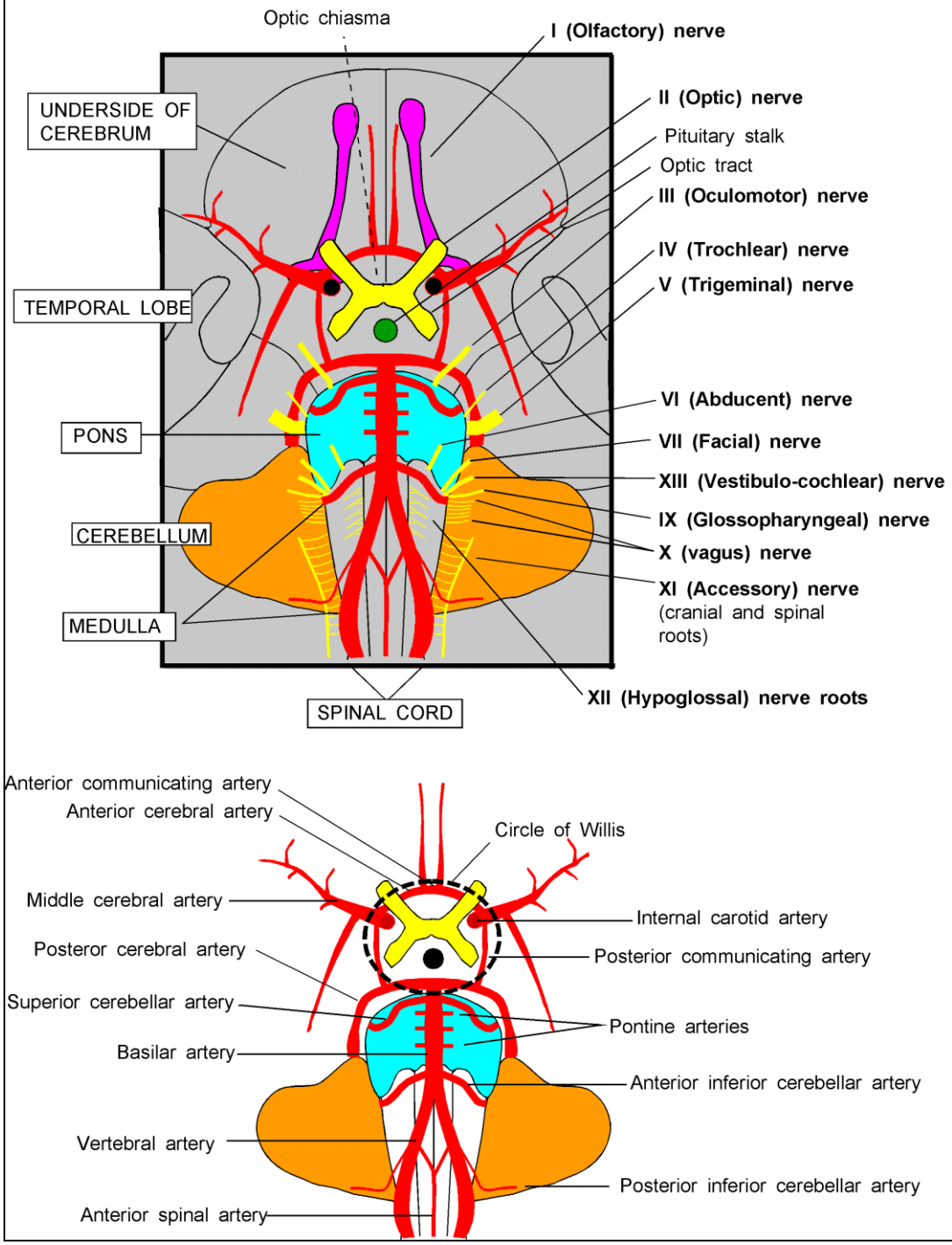
The *ascending* reticular formation receives inputs from almost all parts of the body and is responsible for maintenance of consciousness, sleep, attention and arousal and. Sleep consists of a deep component with slow synchronous waves (on electroencephalogram) and a paradoxical component with fast asynchronous waves. The latter is associated with rapid eye movement and occupies 20 percent of sleep in man, 15 percent in rodents, 0.5 percent in birds and none in reptiles. The *descending* reticular formation conveys rhythmic motor patterns such as walking to the reticulospinal and vestibulospinal tracts.

Figure 36 details the blood supply to the central nervous system and the origin of the cranial nerves.

CRANIAL NERVES (Fig. 36)

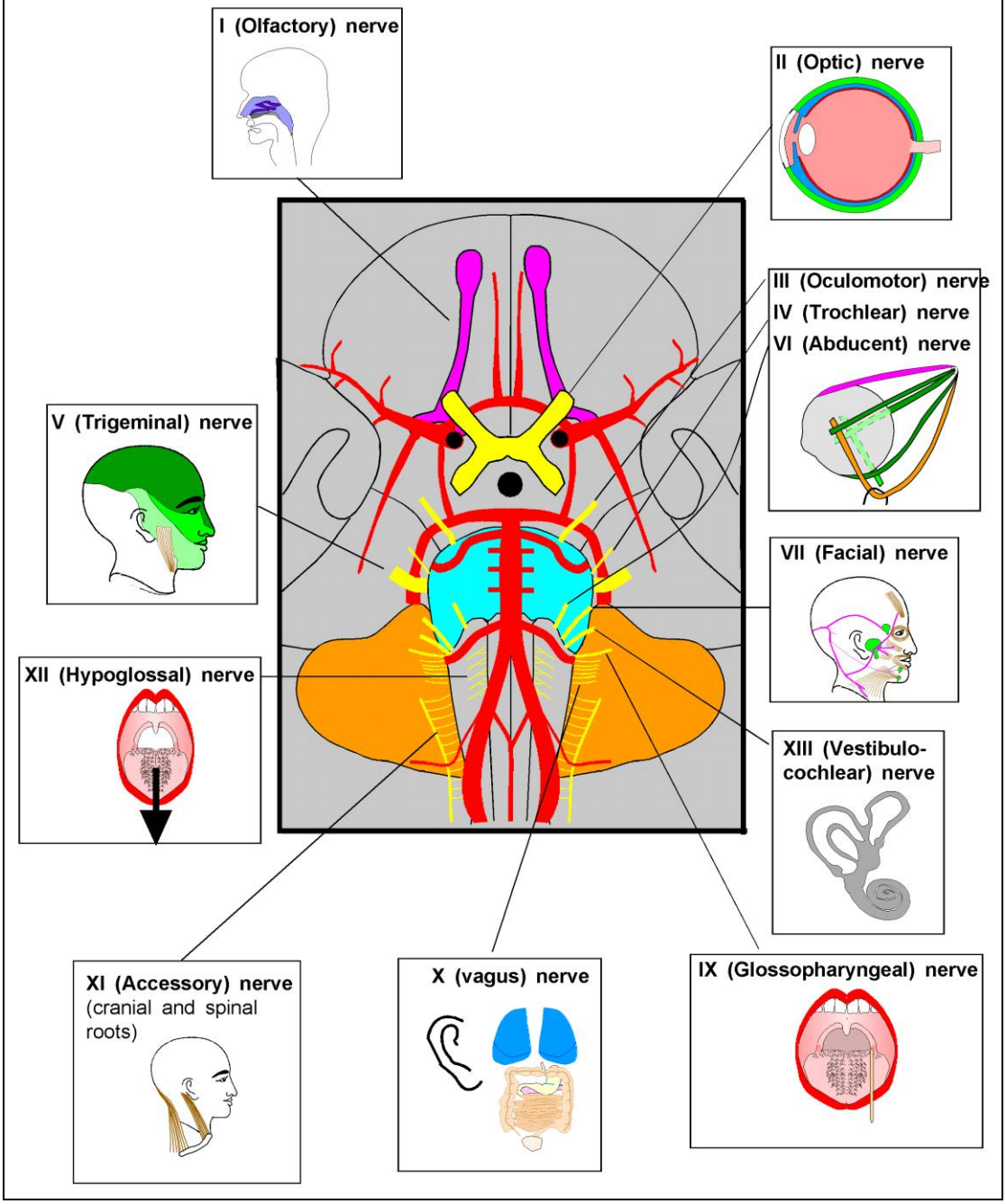
There were originally thirteen cranial nerves in our primitive ancestors. Number 0, the vomeronasal nerve does not exist in humans (despite the claims of various perfume manufacturers and the hopes of some of those who use perfumes) and was used to detect airborne sex hormones (pheromones).

Figure 36. The blood supply to the brain, also showing the site of origin of cranial nerves



Humans have 12 pairs of cranial nerves (Fig 37)

Figure 37. The twelve pairs of cranial nerves



In evolution each cranial nerve was specialized for specific functions, whilst other components were lost. Cranial nerves tend to have dedicated functions, unlike spinal nerves which are all essentially similar in structure and functional abilities. The motor cells of cranial nerves are in nuclei. Although conscious initiation for some cranial nerve functions comes from the cerebral cortex,all the complex integration for brainstem cranial nerves is performed automatically and usually unconsciously. Although we actively look at the environment, papillary accommodation, light reflexes and eye movements are all automatic.

The sensory nerve cells of cranial nerves are outside the brain in ganglia on the trunks of nerves, or in or on the peripheral nerves associated with sensing organs themselves. These cranial nerve ganglia are equivalent to the sensory ganglia of the dorsal roots of the spinal cord.

I. Olfactory nerve. Smell. There are no ganglia as the axons are in effect extensions of the sensory cells themselves. Damage causes unilateral or bilateral loss of smell.

II. Optic nerve. Eyesight and the sensory arm of pupillary responses to light (the motor pupillodilator responses are mediated via the sympathetic nerves which travel along blood vessels, and the pupilloconstrictor responses are mediated via the parasympathetic carried with the IIIrd nerve). The optic tectum (=a rooflike structure), the superior corpora quadrigemina in mammals, is situated (dorsally) in the roof of the midbrain and is the correlative center for initiating or modifying reflex motor responses to light. Unilateral II nerve dysfunction causes:

- Reduction or loss of vision
- Loss of sensory component of pupillary light reflexes
- Visual field impariment
- Reduced pupillary reactions to accommodation

II nerve dysfunctions may be caused by:

- Infections
- Degenerations
- Occlusions of arterial or venous blood vessels
- Inflammation
- Demyelination
- Papilloedema (caused by raised intracranial pressure or by optic neuritis)
- Lens corneal or aqueous opacities
- Blood diseases and toxic reactions

III, IV, VI nerves. Oculomotor (=eye +move), **Trochlear** (=pulley) **and Abducent** (=to lead away) **nerves** (Figs. 38 and 39).

Figure 38. Nerve supply to the eye muscles.
The right eye as seen from above

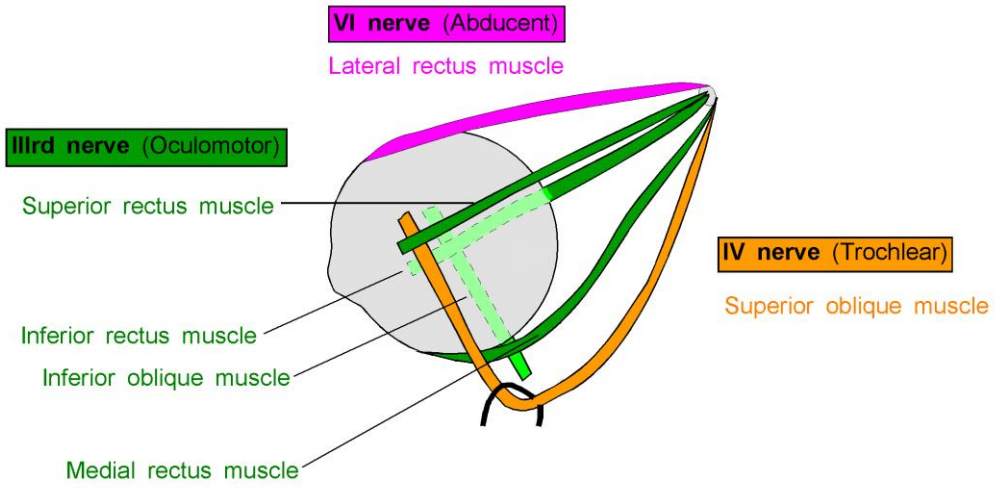
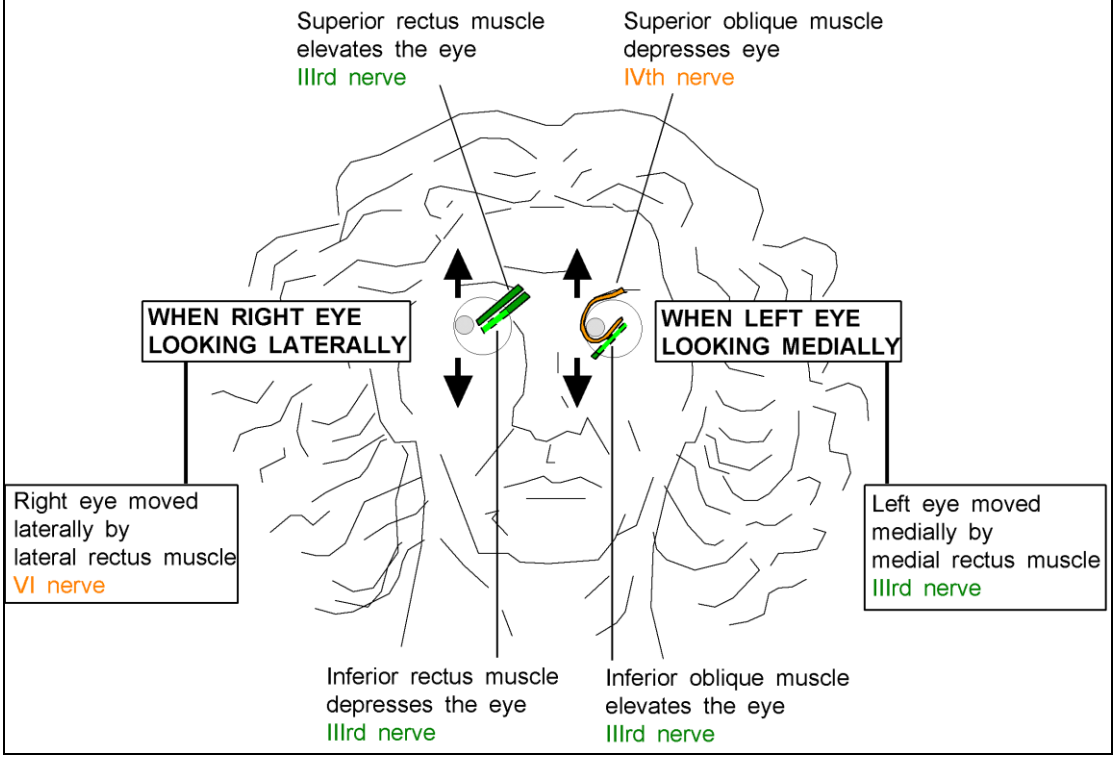
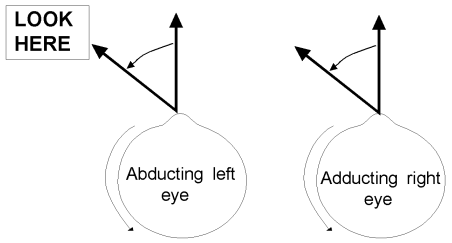


Figure 39. The extrinsic eye muscles and their actions



The IV nerve only provides motor innervation for the superior oblique muscle and the VI only motor innervation for the abducent muscle. The III nerve provides motor innervation for the rest of the muscles used to move the eye - the medial, inferior and superior rectus muscles and the inferior oblique muscle (Fig. 40). The III nerve also supplies the smooth muscle of the ciliary apparatus, the circular constrictor muscles of the iris and the striated muscle which elevates the upper eyelid.

Figure 40. Integration of nerve supply to the eye muscles (as seen from above)



Only the VI nerve is required for the left eye to abduct

V1 IV III

III IV VI

The III and, to a lesser extent, the IV nerve are required to adduct the right eye

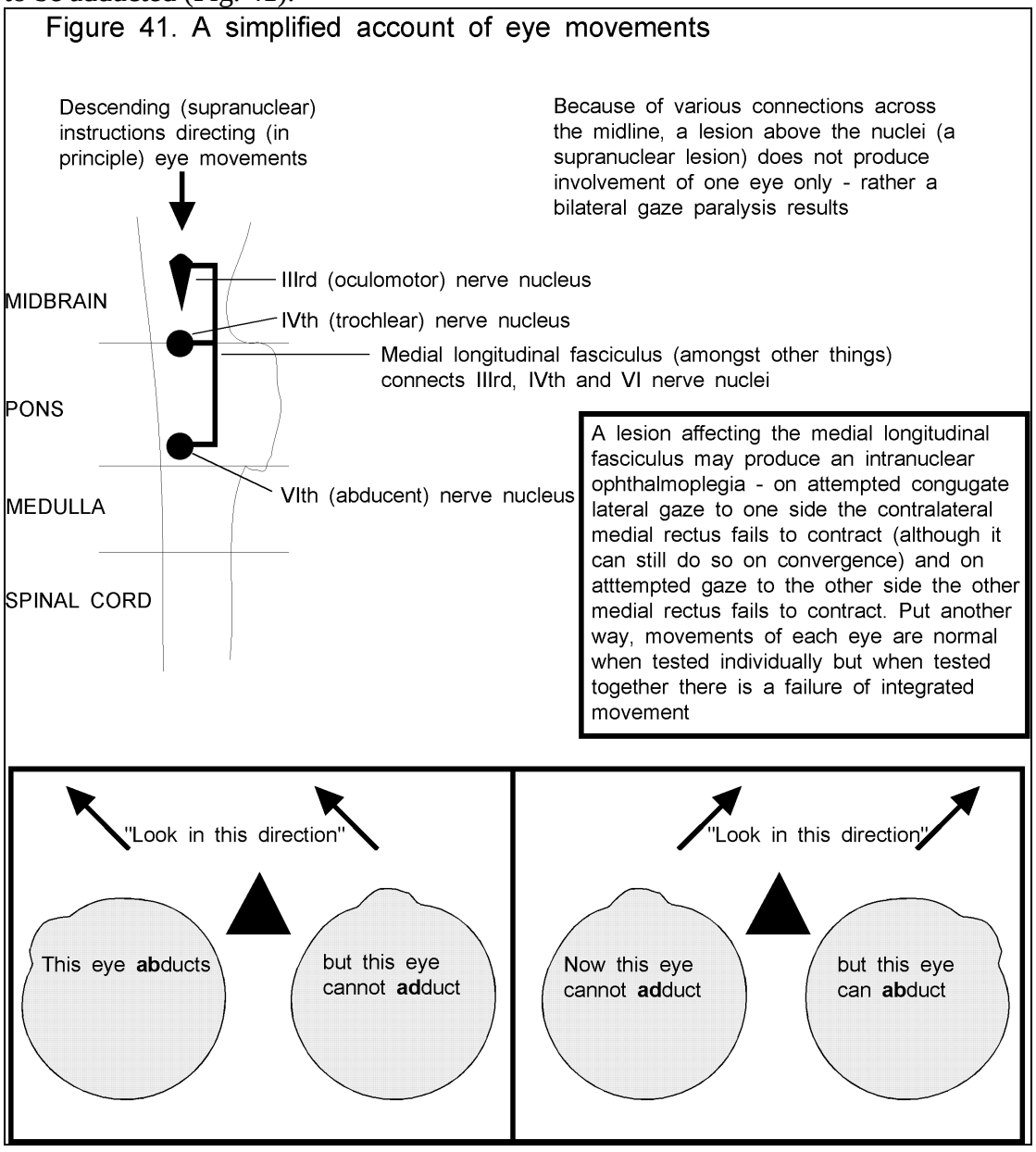
The III nerve muscles pull the eye inwards (as well as up or down)
The IV nerve muscle pull the eye downwards (as well as inwards)
The VI nerve muscle pulls the eye outwards

To integrate these activities there must be 1) a central coordinating nucleus and/or 2) the nuclei of each side must have directly crossing communications in the pons or 3) either the adducting or abducting nerves must cross the midline. Option 3) is what happens with the adducting nerves III and IV crossing but a bit of 2) also occurs

The III and IV nerve both cross the midline within the pons. The III nerve nucleus is dorsal, in close relation to the medial longitudinal fasciculus. The IV nerve nucleus is, unusually for a motor nerve, fairly dorsal and crosses the midline to exit the medulla *dorsally* (unlike any other somatic motor nerve).

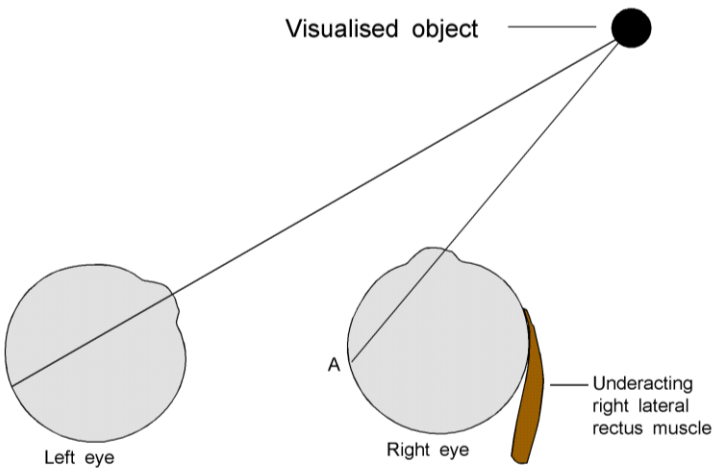
The instructions for eye movements descend into the brainstem and cross the midline near the nuclei of the III, IV and IV nerves and there are communications to the medial longitudinal fasciculus *of the other side* to allow simultaneous abduction of one eye and adduction of the other eye. If this communication is interrupted a curious condition (intranuclear opthalmoplegia) occurs in which the movements of each eye are full when tested independently, but if looking together *to either side* there is abduction of the eye that needs to abduct but failure of adduction of the eye that needs to be adducted (Fig. 41).

Figure 41. A simplified account of eye movements



If there is double vision (diplopia) the false image, the image from the eye at fault, is the more peripheral (Fig. 42).

Figure 42. Identification of the eye at fault if there is diplopia

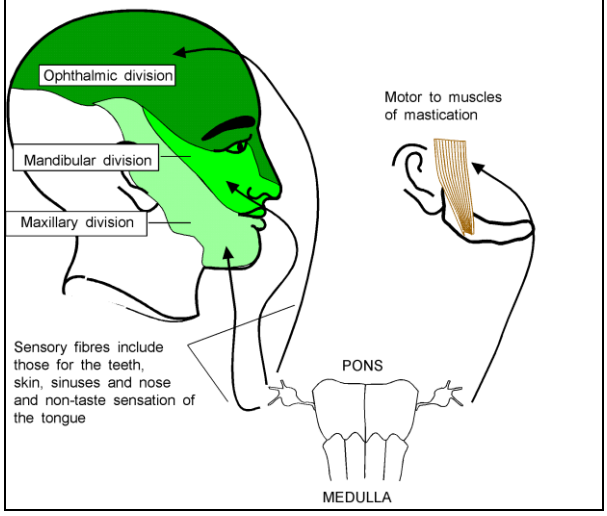


Because one eye is malpositioned the brain receives two images. Point A in the right eye normally receives light from more peripheral objects. This causes the brain to interpret the right eye's image as being the peripheral image. This false image is always the more peripheral image and, by obscuring each eye in turn, the eye at fault (the eye that produces the peripheral image), can be identified. A much more simple method is to give the patient a pair of spectacles with one lens green and the other red and ask which is the more peripheral image.

V. The Trigeminal nerve (Fig. 43). The V nerve, using its ophthalmic, mandibular and maxillary branches, is the principal cutaneous sensory nerve of the face and head. To achieve integration with the sensory inputs from the upper cervical nerves some V nerve fibres descend into the cervical cord. The sensory parts of the maxillary and mandibular branch innervate:

- the nasal mucosa
- the hard and soft palate
- teeth
- anterior two thirds of the tongue (via the chorda tympani which travels with the VII nerve)
- the cornea
- the buccal mucosa

Figure 43. The three branches of the Vth (Trigeminal) nerve



The sensory ganglion that serves all of this is the Gasserian ganglion. The motor supply is to the masseters. In addition to the abnormalities above V nerve dysfunction may cause loss of facial sensation loss of the corneal reflex, weak jaw clenching and jaw jerk abnormalities.

VII. Facial nerve. The VII nerve innervates the muscles of facial expression and certain other muscles, carries taste sensation to the anterior two thirds of the tongue (for part of its course) and some nerves which stimulate salivation. Dysfunction causes:

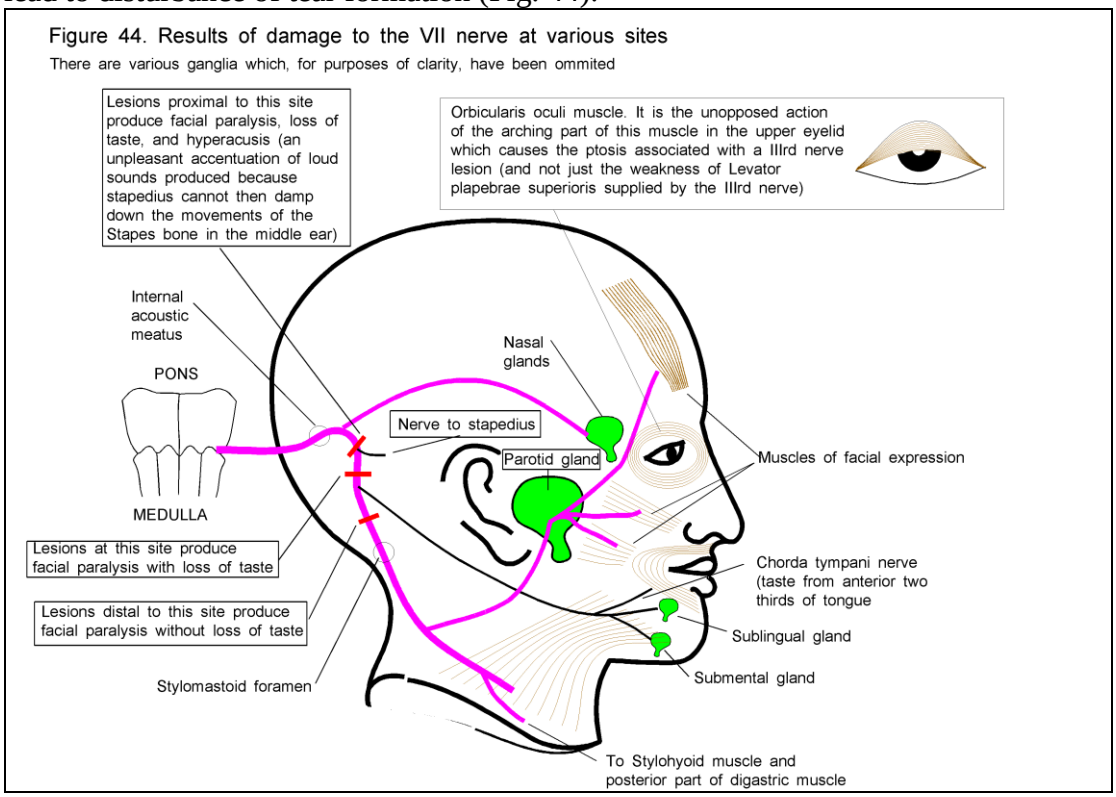
- Weakness of facial muscles
- Impaired hearing
- Loss of taste on anterior two thirds of tongue

A lower motor neurone VII nerve lesion produces drooping of the upper and lower face, whereas an upper motor neurone lesion (often associated with hemiparesis) only affects the lower half of the face because the upper part is bilaterally represented in the cortex.

If the dysfunction is in the facial canal (proximal to nerve to stapedius) then hyperacusis (loudness) results. Dysfunction proximal to the geniculate ganglion will lead to disturbance of tear formation (Fig. 44).

Figure 44. Results of damage to the VII nerve at various sites

There are various ganglia which, for purposes of clarity, have been ommited



VIII. Auditory nerve. The auditory nerve provides hearing and innervation of the balance apparatus of the ear. Dysfunction may cause impaired hearing, vertigo (link), nystagmus, or tinnitus. Further details are mentioned under Special Senses (see later).

IX. Glossopharyngeal nerve. This nerve provides:

- sensation from the back third of the tongue, fauces, palate and upper pharynx
- secretory impulses for the parotid gland

- information from the pressure and chemical receptors in the neck
- motor impulses for the stylopharyngeus muscle which, together with palatopharyngeus (X Nerve) elevates the uvula

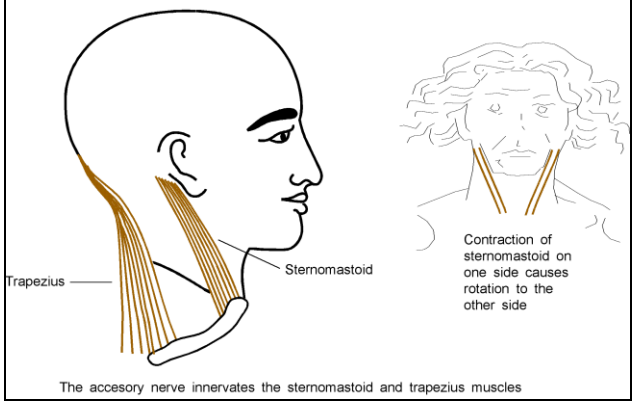
Dysfunction of IX and/or X produces abnormalities of:

- Articulation
- Phonation
- Gag reflexes
- Taste reduction on posterior third of tongue and sensation impairment of pharynx

X. Vagus nerve. This nerve innervates muscles providing the motor side of articulation, phonation, and the gag reflex. If there is a unilateral X nerve lesions then the uvula is pulled to the opposite side. This is brought out by asking the patient to say "Ah." The X nerve supplies most of the viscera.

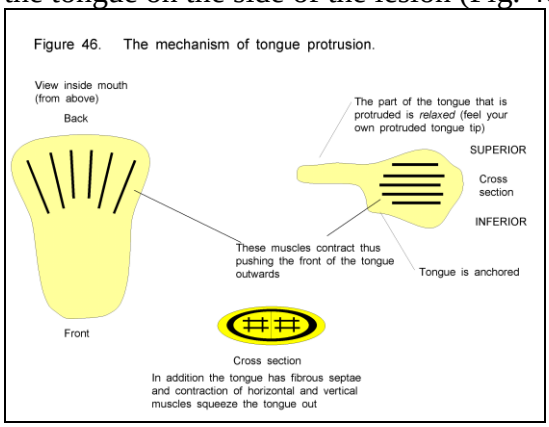
XI. Accessory nerve. The IX nerve was originally known as the *spinal* accessory nerve and provides motor innervation for the trapezius and sternocleidomastoid muscles. The XI nucleus, sensibly enough, may descend as low as the 6th or 7th spinal segment, so that it can integrate with the origin of other nerves that supply neck muscles. Dysfunction of XI produces impaired shrugging of shoulders and neck rotation (Fig. 45).

Figure 45. Functions of the accessory nerve - rotation of the head and shrugging of the shoulders



XII. Hypoglossal nerve. This nerve mediates protrusion of the tongue. The original nucleus was dorsal in situation but has become more ventral so that it is near the medial longitudinal fasciculus and the sensory (visceral, tactile and taste) centres of the medulla which influence its activity. Dysfunction produces impaired protrusion of the tongue on the side of the lesion (Fig. 46).

Figure 46. The mechanism of tongue protrusion.



The pituitary

The pituitary is the important junction between the central nervous system and endocrine system. Pituitary tumors may produce bitemporal visual field loss by pressing upwards on the optic chiasma, optic atrophy by pressing on the optic nerve, and ocular nerve palsies (if very large) by pressing on the III, IV or VI nerve by invasion of the cavernous sinus lateral to the pituitary.

AUTONOMIC NERVOUS SYSTEM (Fig.47)

Figure 47a. The autonomic nervous system

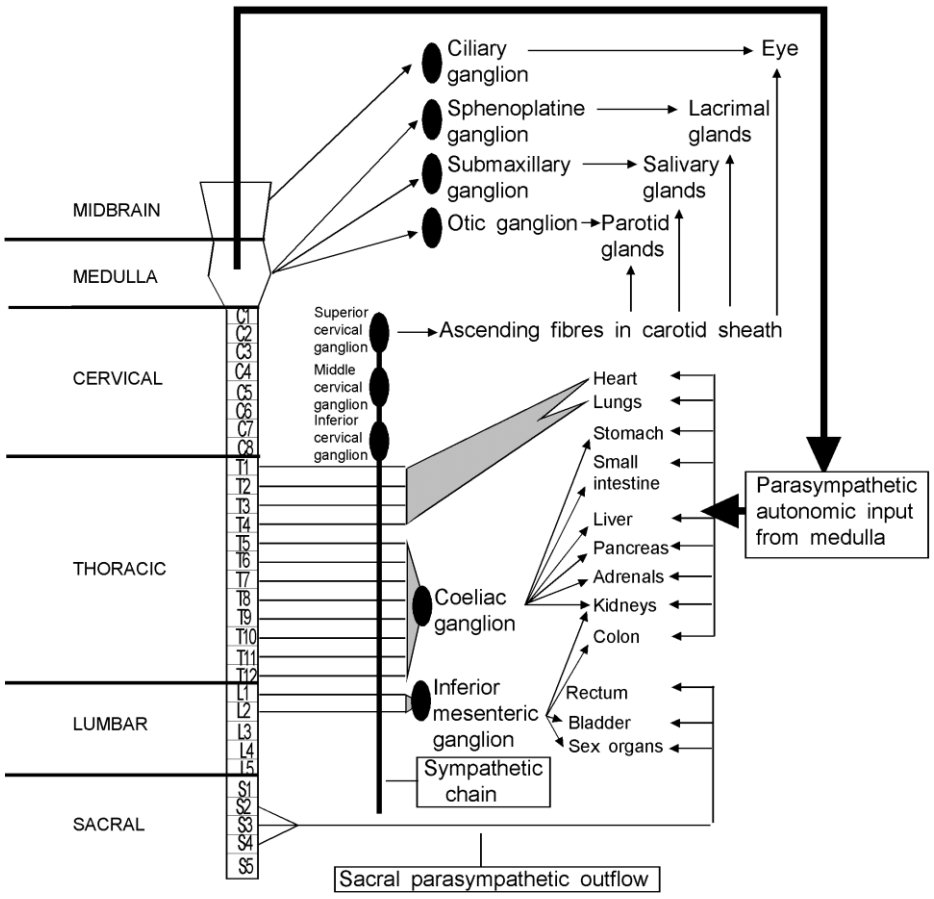
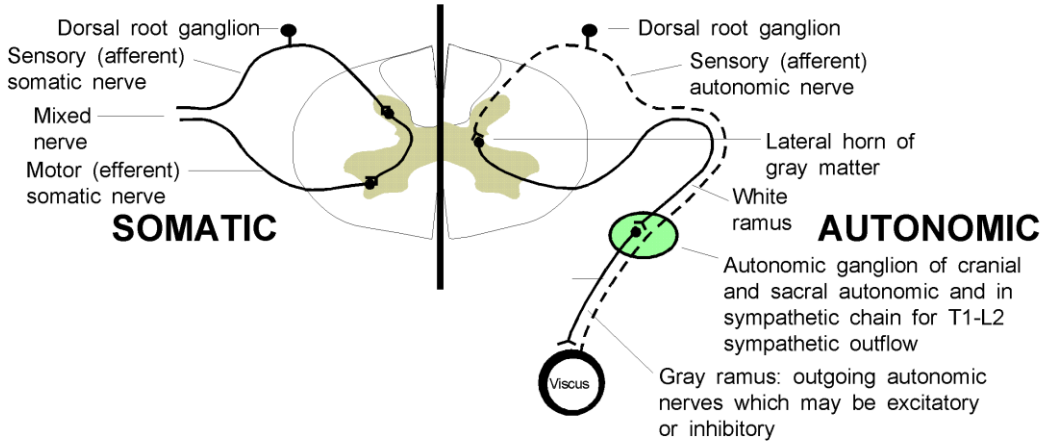


Figure 47b. The somatic and autonomic input and outputs from the spinal cord compared.



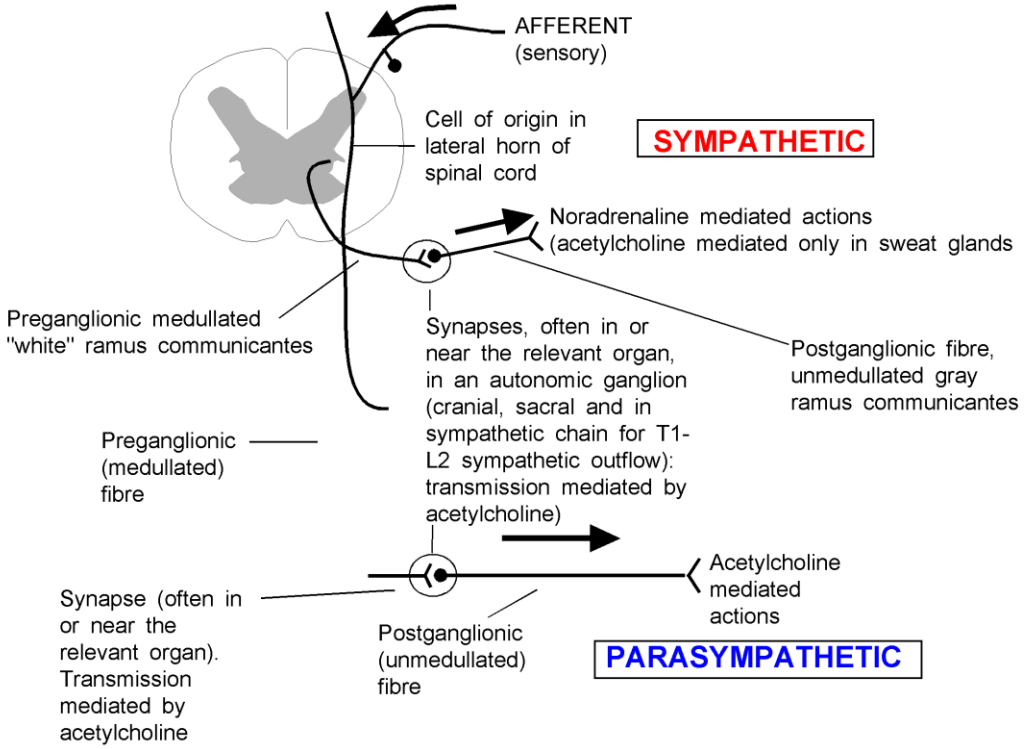
The autonomic (=self + law) nervous system co-ordinates involuntary automatic, unconscious bodily functions including those involving the smooth muscle of the gut, bladder, blood vessels, glands and striated muscle of the heart all of which have intrinsic activity which needs to be modified. It is also essential for function of the sexual organs in the male (link).

The human autonomic nervous system has two, usually counterbalancing, arms - the sympathetic and parasympathetic (Figs. 48 and 49). Both the sympathetic and parasympathetic have sensory inputs and motor outputs.

Figure 48. The reciprocal sympathetic/parasympathetic innervation of some organs

ORGAN	SYMPATHETIC ACTION	PARASYMPATHETIC ACTION
The pupils	Dilates	Constricts
The bronchi	Dilates	Constricts
The heart	Increases rate	Decreases rate
The gut	Decreases motility	Increases motility
The gut sphincters	Constriction	Relaxation

Figure 49. The sympathetic and parasympathetic autonomic systems



Broadly speaking the sympathetic arm elicits extra awareness, excitement, and alarm "the flight and/or fight response" by causing:

- A fast heart rate
- Pupillary dilatation

- Bronchodilatation
- Sweating (the only sympathetic nerves that use acetylcholine for transmission)
- Piloerection “gooseflesh”
- Cessation of digestion

The sympathetic axons have their cells of origin in the lateral horns of the spinal cord. and emerge as myelinated fibres (known as white rami communicantes) which synapse in ganglia (in the sympathetic chain) and leave the ganglia as unmyelinated nerves (known as gray rami communicantes). The sympathetic motor fibres do not relay in the sympathetic chain but within plexuses local to the tissues, for example by the gut (the coeliac, superior mesenteric, inferior mesenteric, and hypogastric plexuses). As evolutionary developments occurred the sympathetic system extended to the head and to the peripheral blood vessels and the glands, whereas the parasympathetic remained largely confined to the viscera and eyes.

The parasympathetic system in contrast is quietening, soothing, and supports background functions such as digestion, defaecation, micturition and heart rate.

The visceral parasympathetic sensory fibres convey information about:
- Baroreceptors in the aortic arch, common carotid arteries and the pulmonary arteries
- Chemoreceptor in the aortic and pulmonary bodies
- Stretch receptors in the lungs
- Certain cardiac and gut receptors

The parasympathetic motor nervous system has a cranial and sacral outflow. The cranial nerve parasympathetic outflow is via the:
- III nerve using the ciliary ganglion. The circular muscle of the iris contracts to constrict the pupil
- VII (+V) causes secretion from the submandibular and sublingual glands via the submandibular ganglion
- IX (+V) causes secretion from the parotid gland via the otic ganglion
- X provides motor impulses for control of heart rate, bronchoconstriction, gastric juice production, and gut and gut derived organs as far as the transverse colon (below this the sacral output takes over) via ganglia near the innervated tissue.

The sacral motor parasympathetic travels with nerves derived from S2,3,4 (the same derivation as the somatic component of the pudendal nerve). The sacral parasympathetic:
- Supplies motor impulses to smooth muscle of descending colon and anus
- Inhibits the internal anal sphincter (the external sphincter is under voluntary control
- Helps coordinate micturition
- Provides vasodilator impulses to the external genitalia producing erections

The autonomic nervous system is also responsible for transporting most visceral pain impulses (visceral = emanating from organs in the body cavities). Such visceral

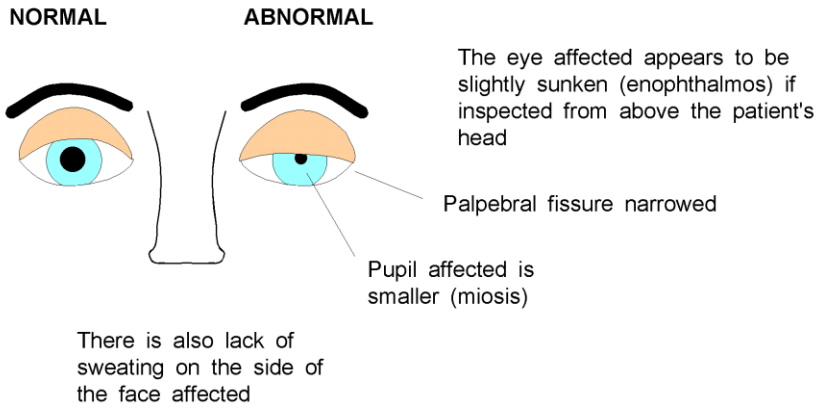
pains are poorly localized, tend to be diffuse, and may be referred elsewhere (and may be appreciated as though it had come from a somatic nerve of same root value.

Symptoms that may result if autonomic nerves are damaged include:

- A burning type pain (causalgia)
- Poor blood pressure control with postural hypotension
- Reduced sweating
- Reduced exocrine secretion by some glands
- Bladder and bowel dysfunction

Horner's syndrome (Fig. 50) is a specific autonomic syndrome which comprises a unilateral constricted pupil, a slight ptosis, an apparently slightly sunken eye (enophthalmos) and lack of facial sweating on the side affected caused by interrupted sympathetic supply to the eye. The relevant sympathetic impulses descend from the hypothalamus into the brainstem and emerge within the anterior roots of C8, T1, and T2, pass into the cranial sympathetic nerve truck and thereafter ascend on the internal carotid artery to the eye. Damage anywhere in this pathway will produce Horner's syndrome.

Figure 50. Horner's syndrome



SPECIAL SENSES

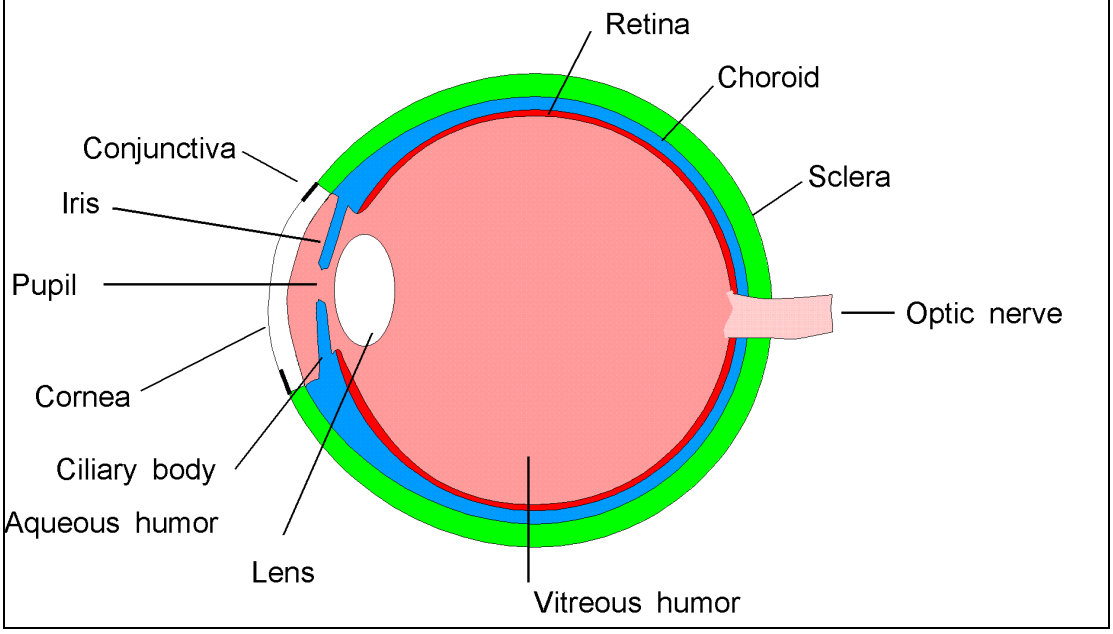
Animals, unlike plants, tend to seek out rapid interaction with the environment. For example plants respond to light but animals look, and some plants respond to vibration but animals *listen.* Often detector organs of animals, particularly the eyes and ears, are able to "tune in" to events of interest. Indeed some animals send out specifically targeted impulses to which their specifically tuned detectors can respond - porpoises and bats probably "see" by echolocation.

The special senses are, sight, hearing, taste, and smell. All are situated in the head.

In evolutionary terms the organs of special sense, smell, hearing and vision, were all useful for detecting food at a distance and presumably evolved with the integrative centers at the head end of the nervous system so that purposeful action could follow detection.

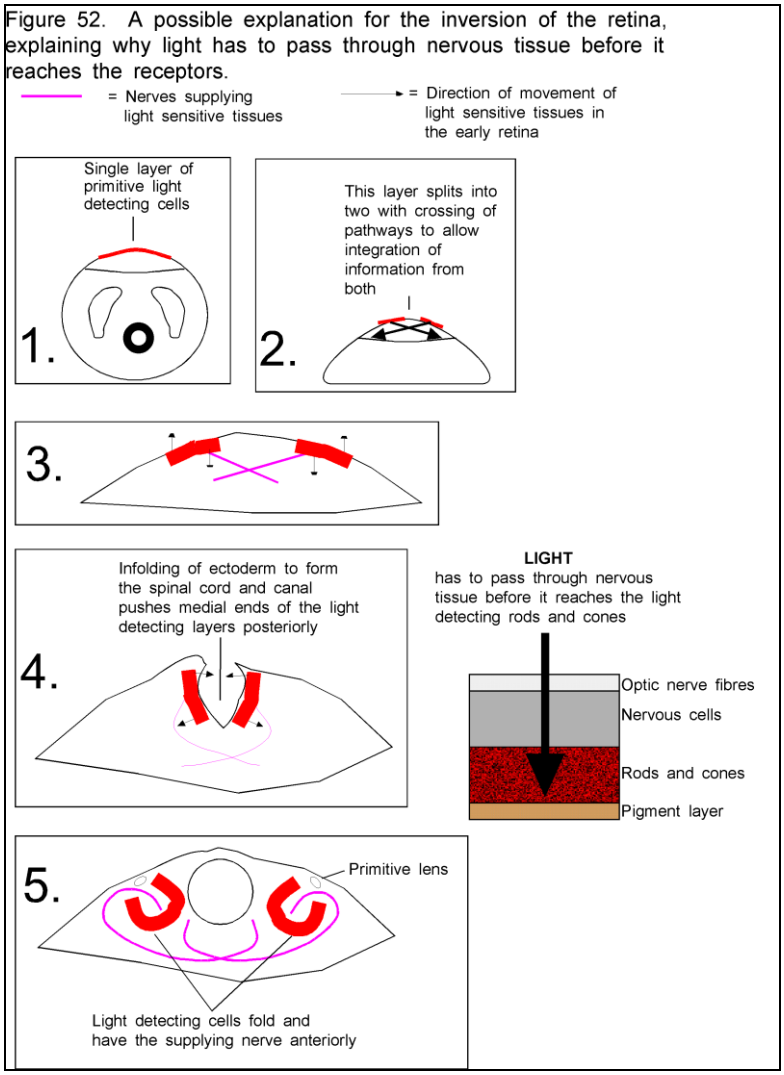
The eye (Fig. 51)

Figure 51. **The anatomy of the eye**



In embryological terms part of the brain migrates outwards and form a cup, the inner surface of which will form the retina and the rim of which will form the iris. The eye is often said to be a perfect design for its job. This is not so. The flaws are:

- The nervous tissue is in front of the light receptors so that light has to pass through nervous tissue before hitting the light receptors (Figure 52)
- There is a large blind spot where the Optic nerve enters the eye
- We can only see clearly what we look at directly – peripheral vision is vague
- The blood vessels run on the surface of the retina



The lens is derived from ectoderm and the sclera, choroid, cornea and ciliary apparatus derive from mesenchyme. In humans eyes are paired, allowing stereoscopic vision.

The retina is derived from the lining of the third ventricle and it appears that the light detectors in the retina, the rods and cones, are modifications of cilia.

There are two types of photoreceptor cells, rods and cones. There are about 120 million rods which:

- Are all alike
- Contain rhodopsin
- Are more able to respond to low level light and movements in the visual field than cones
- Do not respond to colour

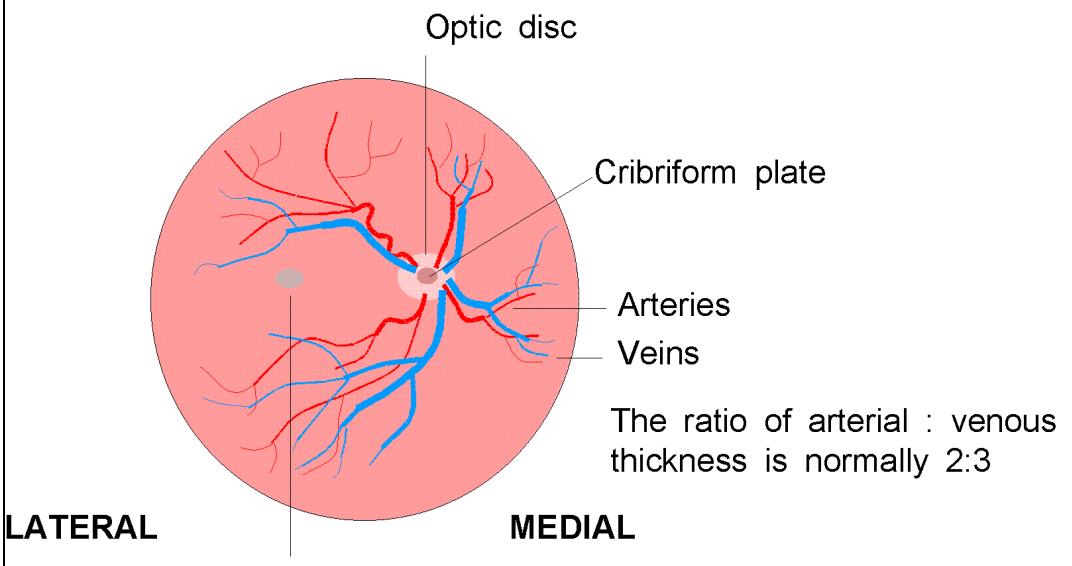
There are about six million cones which:

- Are used for fine discrimination
- Respond to light of different wavelengths (colour)

- Each contain one of three visual pigments, each to responding to short, medium or long wavelengths

Humans have a macula, an area of maximum acuity, which contains cones and very few rods. The central macular depression is the fovea (=a pit) centralis on which images of interest are focused (Fig. 53).

Figure 53. The (right) optic fundus



The peripheries of the retina contain rods and few cones whereas the fovea contains only cones. The visual object of maximum interest is focussed onto the fovea for maximum definition. It takes 0.02 of a second for retinal impulses to reach the visual cortex: this time is prolonged if there is dysfunction of myelinated nerves (as may occur in multiple sclerosis).

Curiously the light has to travel through nerve tissues of the retina before it reaches the receptors. The probable explanation of this apparent inefficiency is shown in Figure 52.

The sclera is a firm yet flexible housing for the eye. The sclera merges with the cornea, which partially bends light before the lens performs the major role in the focusing of images onto the retina. Humans focus by changing the shape of the lens by using the radial muscle to stretch the lens (Fig. 22) which has to return to its previous shape by virtue of its intrinsic elasticity (which declines with age).

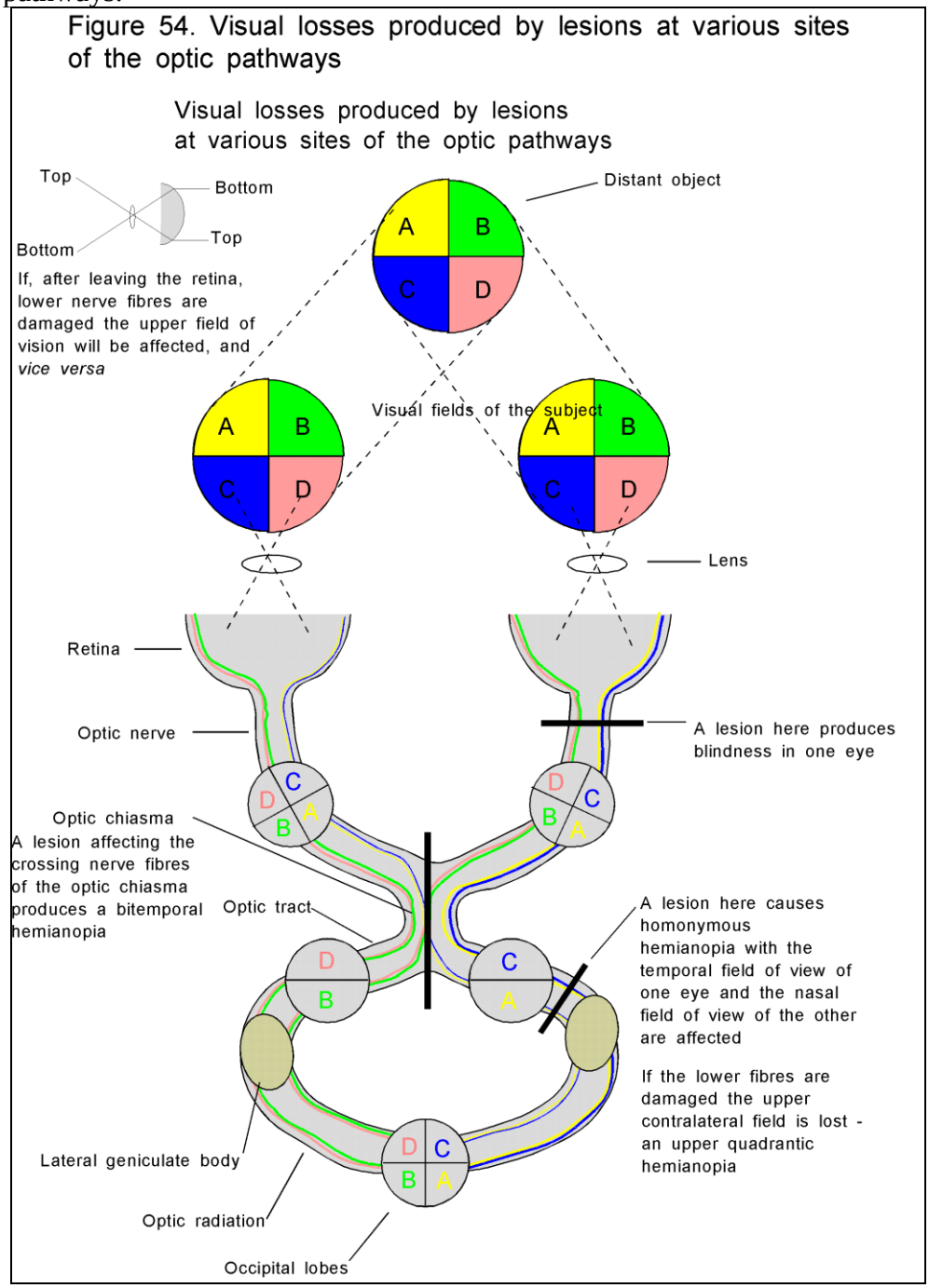
The iris surrounds the pupil and, by contraction of its circular or radial muscle attempts to ensure that a constant amount of light reaches the retina. Accordingly the pupils are small in bright light and large in dim light.

The aqueous humour (the fluid in the anterior chamber) is formed by the ciliary body. The aqueous humour is drained (into the canal of Schlemm) in the filtration angle. If this angle is too acute the canal cannot drain the humour and closed angle glaucoma (glaucoma = grayish blue, describing the appearance of the cornea in this disorder) results from raised intraocular pressure.

The conjunctivae (=to connect) is a thin transparent membrane which can be considered to be part of the skin which lines the inner lids and the exposed areas of the eye to protect them against the elements.

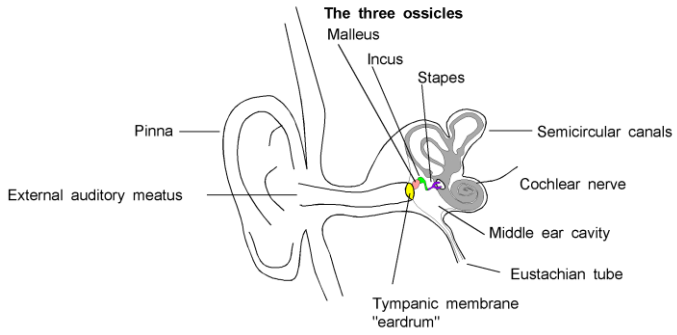
Figure 54 illustrates the visual losses produced by damage at various sites in the optic pathways.

Figure 54. Visual losses produced by lesions at various sites of the optic pathways



The ear (Fig. 55).

Figure 55. The ear and associated structures



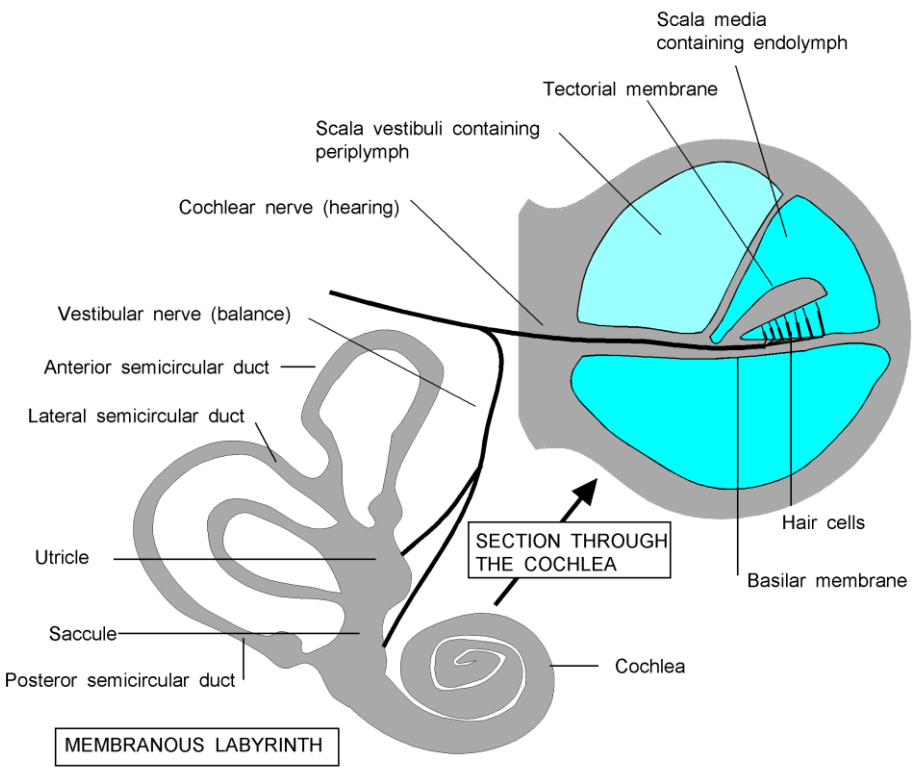
Amphibians first dragged themselves out of the sea onto the land, probably from estuary shores, 200-300 million years ago. Initially their heads were in contact with the ground and they used bone conduction for hearing. Later the head and body were lifted off the ground which encouraged the development of other hearing mechanisms with part of the lateral line of our fish ancestors sinking into the head to become a new organ, the ear, (presumably this new organ developed in the head presumably because it was away from the jogging influence of the limbs). Sound appreciation is thus a modification of aquatic "tactile vibration sense."

Vibration sense is a "crude hearing" relayed by all general somatic sensory nerves. If someone has complete nerve deafness then vibration sense can still be appreciated by trigeminal innervation (Beethoven rested a ruler, one end of which was held in his teeth, on his piano to "hear" his playing when he became deaf)

The ear has to collect sound waves varying in frequency between 20-20,000 cycles per second, funnel them toward the sensory detectors, "concentrate" the sound by mechanical means and then convert this concentrated movement via vibrations in the various ear lymphs into nerve impulses for onward transmission to the brain. The sensitivity of the system is so great that the two ears together can localise sound using the delay in the time taken for sound to travel the distance of the head diameter and the slight differences in volume. The scale used for measuring volume (decibels) is logarithmic (each change in 10 units represents a tenfold change in volume). The ear's range of useful volume appreciation is 1-140 decibles – a range of about 10^{13}.

The inner ear (Fig. 56) is made up of the utricle (=little leather bag), saccule (=small sac), the semicircular canals, and the vestibule (which is responsible for hearing).

Figure 56. The simplified anatomy and neurological connections of the ear



The outer ear focuses air borne vibrations onto the tympanic (= drum) membrane. These vibrations are then greatly amplified by three middle ear ossicles (= small bones), the malleus (= hammer), incus (= anvil) and stapes (= stirrup). The stapes depresses the oval window and creates a pressure wave in the tube containing perilymph (the scala vestibuli) which then doubles back on itself (as the scala tympani) with the scala media (which bears the spiral organ of Corti) in between. The tubes are coiled to form the cochlea (= snail shell). Pressure waves in the scala vestibuli are transmitted across the thin membrane to the endolymph of the scala media and from there to the basilar membrane of the scala tympani which bears the hair cells which detect vibrations in the fluid.

To maintain orientation and balance, the effects of acceleration, deceleration, rotation, and gravity have to be assessed. Vision became the predominant modality for orientation in our ancestors, and to do this stable retinal images had to occur, and to do this the head had to be stabilized. To achieve this the hair cells in the semicircular canals of the ear are utilized in two ways to detect changes in acceleration or rotation.

Firstly, the semicircular canals comprise three loops, each lying in different planes and each filled with endolymph. At one end of each canal there is a swelling, the ampulla (= flask) within which there are sensory cells, the crista (= crest), bearing the hair cells which detect movement in the endolymph. Interestingly the orientation of the semicircular canals is such that acceleration in a horizontal plane causes a flow of endolymph such that the brain thinks that the body is moving upwards. This is why pilots flying in mist have to rely on their instruments if they are not to crash into the ground: if they cannot use sight for orientation their ears tell them they are going

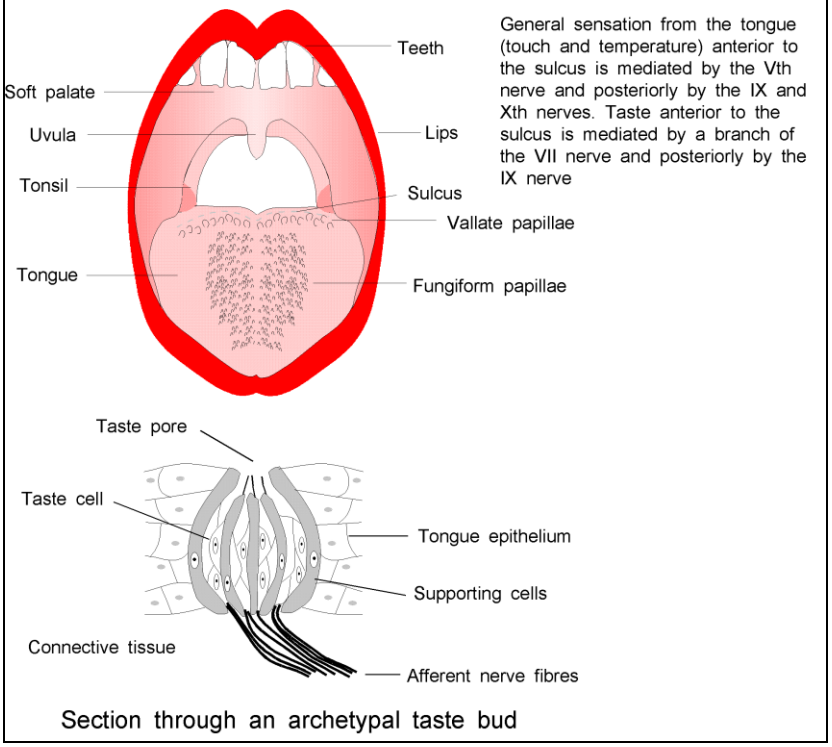
slightly upwards and they therefore react by flying slightly downwards, ultimately into the ground. When flying in fog pilots *have* to rely on their instruments and not their sensation of what is horizontal.

Secondly, to assess orientation when there is no head movement there are small speckles of calcium carbonate (otoliths) that are found in the otolithic membrane of the utricle and saccule. Gravity acts on otoliths and hair cells which assess the direction of the pull of gravity. Movement of the perilymph will also affects the otoliths but to a lesser extent.

Taste and smell

Appreciation of food depends upon the detection of dissolved substances by chemical detectors on the tongue (Fig. 57).

Figure 57. Distribution and structure of taste buds



Section through an archetypal taste bud

Smell depends on detectors, situated at the top of the nose, of airborne substances which have to be dissolved before they can be smelt. Smell is thus a tasting of airborne substances. In our marine ancestors taste probably developed first - in the sea everything was dissolved and not airborne - and smell developed later when the land was invaded. Information about taste is conveyed to the medulla, then to the thalamus and then to the cerebral hemispheres. Each taste bud is most responsive to one of the four basic tastes - sweet, sour, salt and bitter. Umami is now recognised as a fifth basic taste.