Somatosensory System

Study Objectives

- *To define* adaptation, adequate stimulus, coding, sensory receptors including taste and smell, molecular receptors, receptor potential, stimulus transfer, types of sensory nerve fibres, conduction velocity, and threshold stimulus.
- *To describe* skin receptors, articular receptors, nociceptors and central pathways, the effect of chordotomy, thalamic surgery, and prefrontal lobotomy.
- *To draw* Hills force-velocity curve and the voltage-duration curve for nervous stimulation.
- *To calculate* one variable from relevant information's given.
- *To explain* cortical somatotropic and columnar organisation, the control of taste and smell, the control of nociceptive transmission (gatecontrol), central analysis, central pain, headache, referred pain, allodynia, causalgia, hyperalgesia, trigeminal neuralgia, thalamic syndrome, phantom limb pain, hyperalgesia, and Brown-Sequards syndrome.
- *To use* the concepts in problem solving and case histories.
- *Principles*
- *Critical empiricism. In brain research any scientific observation presupposes a theory that can be falsified. Theories that fail to be falsified in repeated scientific projects are temporarily acceptable. This philosophy is generally applicable.*
- *Sherrington's integration law. The integrative action of the nervous system unifies separate organs to form an individual personality.*

Essentials

This paragraph deals with *1. Sensory receptors and nerve fibres, 2. Blood-brain barrier and CSF, 3. Regeneration of nervous tissue, 4. Sensory pathways, 5. Central opiate receptors, 6. Taste and smell.*

1. Sensory receptors and nerve fibres

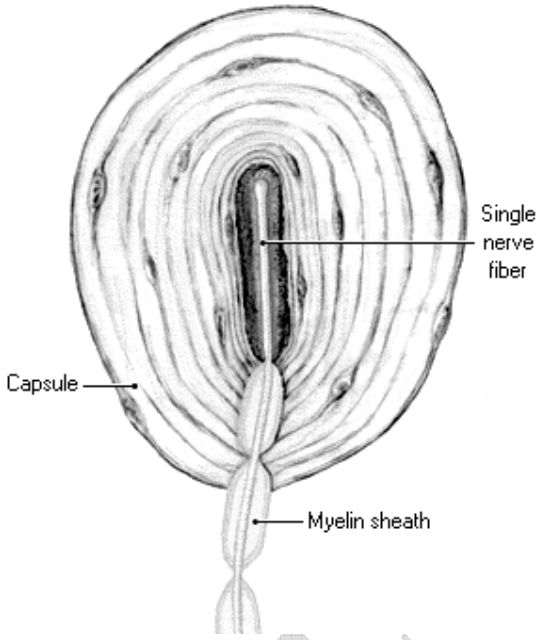
Sensory receptors are either *neurons* in the case of vision, smell and cutaneous senses, or *modified epithelial cells* in the case of vision, auditory, vestibular, smell and taste senses. The special sensory receptors for vision, hearing and balance.

Some sensory receptors have characteristics similar to the well-known *plasma membrane receptors*. Plasma membrane receptors consist of a protein or glycoprotein molecule, an ion channel or a specific enzyme (G-protein).

The stimulation of a receptor elicits a receptor potential (*generator potential*) that is graded continuously with stimulus intensity. When the stimulus is strong enough to reach the threshold, action potentials (APs) are fired. In neurons the stimulus intensity is coded by the frequency of action potentials.

Sensory receptor systems are *biological transducers* with a dynamic range of *up to* 10^{12} in the most sensitive organ, the *ear*. The *threshold* is the reciprocal of the sensitivity. The *threshold* is the weakest stimulus to which the receptor will react. The *sensitivity* of a sensory receptor is greater the smaller its threshold stimulus is.

The Pacinian corpuscles in the skin are gigantic receptors (almost 1 mm long) consisting of concentric layers like onion-scales in the microscope. There is a single axon in the axis of each corpuscle. Stimulus intensity is coded by the single axon. Compression deforms the axon and depolarises the membrane by opening of Na^+-channels.



The Pacinian corpuscle (1 mm long) is a vibration detector.

The depolarisation generates a graded *receptor potential* forcing a current towards the first node of Ranvier with maintained stimulus. The receptor potential rapidly decreases (rapid adaptation), because the adequate stimulus is alterations in the deformity rate (vibrations in the range 150-300 Hz). At the first node of Ranvier, a propagating *action potential* along the axon is released, provided the generator potential is sufficiently large . The Pacinian corpuscle is located in the deeper layers of the skin and connective tissue. The afferent fibres are thick (Type A or II), and they lead the signals to synapses in nucleus gracilis and cuneatus of the spinal cord.

Perception of *taste, heat and angular acceleration* are described by *power functions* or *transfer functions* with n just above 1, whereas *hearing and smell* are described by functions with n lower than 1. The only sensation with a particularly large value of n (about 3) is *pain,* which is why pain is felt so severe with increasing stimulus intensity!

However, the power law and other types of curve fitting with transfer functions have hardly improved our understanding of sensory modalities.

Both *conduction velocity* and *size* is used in classification of nerve fibres. The fibres are divided into types A, B and C, based on the three main *conduction velocities* shown in the record of the *compound action potential* from a mixed nerve. Type A, B and C refer to phases in the combined action potential. Type A fibres are the fast conducting myelinated fibres (thick fibres subdivided into , , , and), type B are preganglionic sympathetic fibres, and type C are the small, unmyelinated fibres. Another classification is based on the *thickness of the axons* (I-IV). The size classification became necessary, when A - fibres were separated into two subgroups: Ia and Ib.

In Box 3-1, the velocity classification (A-C) is written first, and the size classification (I-IV) is given in parentheses.

Classification of nerve fibres			
e		meter m	on
		- -	m per s
	fibres		

	fferents (Ia)		
	rgans (Ib)		
	d pressure		
	muscle spindles		
	ssure, temperature		
	onic		
	ch, heat		

The A (I) fibres are *motor -fibres* and *proprioceptors* from the annulospiral endings of muscle spindles (Ia) and from Golgi tendon organs (Ib).

The A (II) fibres conduct discrete touch and fine pressure signals from cutaneous tactile receptors.

The A (II) fibres are motor fibres to muscle spindles. They have their origin in the spinal cord.

The A (III) fibres transfer pain sensations, decline in skin temperature as well as crude, passive touch and deep pressure.

 The B fibres (III) are autonomic preganglionic fibres.

 The C fibres (IV) are unmyelinated and lead *pain, touch* and *signals* from heat receptors from the skin. The C fibres have no myelin sheath.

Sensory receptors in the nervous system are classified as *exteroceptors* (located on the body surface), *proprioceptors* (located in muscles, tendons and joint capsules), *interoceptors* (located in the viscera), and *telereceptors* (stimulated by events far from the person).

Cutaneous receptors are exteroceptors (Fig. 3-3). Pacinian and Meissner corpuscles are rapidly adapting (dynamic) touch velocity detectors in glabrous skin. In hairy skin, hair-follicle receptors are velocity detectors (they adapt rapidly). Meissner corpuscles are located in the papillae of the hairless skin such as fingertips, lips and clitoris. Merkels discs and Ruffini-end organs are slowly adapting (static) touch intensity detectors both in hairless and hairy skin. Merkels discs are found in elevated dome corpuscles in hairy skin (up to 50 Merkel discs in a corpuscle of 0.5 mm in diameter). These so-called *Iggo dome receptors* are extremely sensitive and transmit touch signals to a single nerve fibre. In this way, even weak tactile stimuli can create sensation in the CNS.

Thermoreceptors are also exteroreceptors. We have cold-receptors just below the skin surface (200 nm deep). Cold receptors respond to changes in temperature. Heat receptors are also located in the skin. The location of certain heat and cold points in the skin is determined by bringing a thin hot or cold object in contact with the skin. *Thermoreceptors* react to temperature changes. *Cold receptors* and *heat receptors* in the skin are located close to the surface. Both types of receptors are also located in the deep tissue and in the CNS. Both types of receptors *discharge spontaneously* at normal temperature, *dynamically* when skin temperature is changing rapidly, and *adapt slowly*.

Proprioreceptors, located in muscles, joints and joint capsules, are mechanoreceptors (muscle spindles, Golgi-receptors, Pacinian and Ruffini corpuscles, and free nerve endings). The Ruffini mechanoreceptors are also called *joint receptors*, because they are located in ligaments, tendons and *articular capsules*. They provide information for the CNS concerning articular movements, movement velocity and joint position. Joint receptors of the proximal joints are particularly sensitive. The static and dynamic receptors inform the CNS about the position and movement of the joint, respectively. These receptors enable us to sense the position of the joint with great accuracy.

Accommodation of sensory receptors or *adaptation* is a progressive decrease in firing frequency despite maintained depolarization. The frequency of action potentials from stimulated receptors fall, although the stimulus is maintained at constant strength. Accommodation or adaptation occurs, when a proportion of the voltage-gated Na^+-channels is rapidly *inactivated* by depolarisation, which also opens K^+- channels. This makes the cell more refractory to stimulation. Accommodation can also be caused by a hyperpolarization induced by gradual activation of Ca^{2+} -dependent K^+ -channels.

Pain- and cold-receptors, Merkel discs and Ruffini-end organs adapt extremely slowly and incompletely. Joint receptors, smell-taste-receptors, muscle spindles, carotid sinus- and pulmonary stretch receptors, and the optic nerve, all adapt somewhat better.

Hair-follicle receptors, Meissner corpuscles and Pacinian corpuscles adapt rapidly, just as many free nerve endings.

Nociceptors or *nocireceptors* (pain receptors) are responsive to stimuli that potentially cause injury. Nociceptors are free nerve endings of two types. The *fast adapting* A fibre mechanical nociceptors (group III) are high-threshold, finely myelinated afferents that originate superficially in the skin. The *slowly adapting* C-polymodal nociceptors (group IV) are unmyelinated afferent fibres that originate in the deeper cutaneous tissue, and respond to various mechanical, thermal and chemical stimuli. In the spinal cord nociceptive afferents synapse with secondary neurons in lamina I and II. These sensory neurons ascent in the spinothalamic tracts.

The fast adapting pain through group III fibres is bearable (acute, sharp, stinging, somatic pain), compared to the slowly adapting unbearable pain (diffuse, burning, prolonged secondary, visceral pain) through group IV fibres.

When nociceptors become sensitised (ie, more responsive), their thresholds are reduced, thus causing *hyperalgesia* (ie, hypersensitivity to pain). Many substances such as bradykinin, histamine, leucotrienes, prostaglandins, serotonin, and K+ that are often released near damaged or dying cells sensitise nociceptors. K^+ *activates* the nociceptors. Substance P is also released from polymodal nociceptors through an axon reflex with antidromal signal transduction in afferent group IV fibres, causing hyperalgesia, vasodilatation and increased capillary permeability. Glutamate may be co-released with *substance P* from the polymodal C-fibre terminals.

The *gate-control hypothesis* of pain states that pain transmission is suppressed by innocious signals in thick myelinated afferents (group II), whereas the pain sensation is enhanced by signals in thin afferents. Inhibitory interneurons of the lamina II in the dorsal horn of the spinal cord perform the gate-control through a special type of presynaptic inhibition called primary afferent depolarization (PAD), and the receptors on the cell body of the secondary neuron is the gate. The gate control hypothesis explains why innocuous signals, mediated by large myelinated afferents, can inhibit pain mediated by thin myelinated afferents.

The *adequate stimulus* is the stimulus, for which the receptor has a lower energy threshold than for other stimuli (ie, the stimulus to which the receptor is most sensitive). The adequate stimulus for pain receptors is mechanical deformation, extreme temperature or tissue damage. The *sense impression* depends on the site in the brain which receives the sensory signal (ie, *central analysis*) and on the receptor localisation (ie, *peripheral analysis*). This is how different neurons transmit different types of sensations, even though they may transmit the same electrical signals.

The CNS discards more than 99% of all incoming signals as irrelevant.

The visual system is an example of a *specific information line* for a certain modality of sensation. The neurons in the retina, the optic nerve, the lateral geniculate nucleus, and the visual cortex describe just such a dedicated neuronal pathway. The specific information line through which the signal is conducted determines the way in which a suprathreshold stimulus is perceived (eg, pressure applied on the eye will be perceived as light).

The auditory system also forms a *specific or labelled line* all the way from receptor to cortex. In all cases the specific region in the cerebral cortex, where the nerve fibre ends determines the modality of sensation.

Now, where is the sense interpretation localised and what is its intensity?

1. Coding in the sense organ is *peripheral analysis,* which is based on the peripheral location of the receptor. External energy is transformed to a *receptor potential* that triggers APs in afferent nerve fibres. Peripheral analysis depends upon the location and the special structure and sensitivity of the receptor. The pattern of firing of APs is the only

possible variable for coding information in a single neuron. Examples of firing patterns are *on-off* patterns with mean frequencies, *off-on* patterns, *transient* patterns or *adaptation*, *long-lasting* patterns, firing with latency etc.

2. *Central location coding in the CNS* is termed *central analysis*, which is related to the sense impression.

2. Blood-brain barrier and CSF

The blood-brain barrier consists of *tight junctions* between the endothelial cells of the capillaries in the CNS and of neuroglia. This barrier only allows extremely small or hydrophobic molecules to pass into the brain. The cerebral microcirculation consists of *strong arterioles* that can constrict to carry a high arterial pressure without brain oedema.

Many large molecules cannot pass from the blood to the cerebrospinal fluid (CSF) across the choroid plexus, a tight junction barrier that is called the *blood-cerebrospinal fluid barrier.*

The *blood-CSF barrier* of the choroid plexus allows some large molecules to pass from the blood to the CSF.

The blood-brain and the blood-CSF barriers exist in all areas of the brain, except in the so-called *circumventricular organs* (hypothalamus, the pineal gland, and the area postrema). These discrete organs have highly fenestrated capillaries that are easily penetrated by large and small molecules as well as ions. The circumventricular organs are located close to essential control centres in the hypothalamus and brain stem regions regulating respiration, blood glucose concentration, and extracellular fluid osmolality.

The two brain barriers are almost impermeable to large molecules such as plasma proteins, but highly permeable to CO_2, oxygen, water, alcohol, anaesthetics, hallucinogens, and other lipophilic substances. The blood-brain barrier is almost completely impermeable to water-soluble molecules, electrolytes such as H^+, whereas CO_2 passes through the barrier to the medullary chemoreceptors.

Humans produce 500 ml of CSF daily. The total CSF volume is only 1/3 of the daily production. Most of the 500 ml of CSF is produced in the *choroid plexuses* in the four brain ventricles, and the remaining is produced across the blood-brain barrier.

The ventricular system and the central spinal channel are covered with *ependyma*. The absorption of CSF takes place through the *arachnoidal granulations*, which protrude, into the *sinus sagittalis*. The rate of absorption is directly related to the pressure in the cranial cavity - in particular the CSF- pressure. Proteins can pass through large holes in the endothelial cells. The CSF is separated from the brain cells by the *thin* pia mater. Substances that enter the CSF can easily diffuse into the brain interstitial fluid. Drugs that cannot pass the *blood-brain barrier* can enter the brain through pia mater, when infused into the CSF.

The CSF passes from the lateral ventricles (I and II) through the *foramen of Monroe* into the third ventricle (III), through the *aquaduct of Sylvius*, the fourth ventricle (IV), and out into the subarachnoid space through the *foramina Luschkae & Magendie*.

The normal CSF-pressure in a supine person is up to 10 mmHg (1.3 kPa) or 136 mm of water.

The secretion of fluid by the choroid plexus depends on the active Na^+-transport across the cells into the CSF. The electrical gradient pulls along Cl^-, and both ions drag water by osmosis. The CSF has lower [K^+], [glucose], and much lower [protein] than blood plasma, and higher concentrations of Na^+ and Cl^-. The production of CSF in the choroid plexuses is an active secretory process, and not directly dependent on the arterial blood pressure. The CSF is separated from the brain cells by the extremely thin *pia mater*. All natural substances that enter the CSF can easily diffuse into the brain extracellular fluid.

CSF leaves the four ventricles through the roof of the 4th ventricle, traverses the subarachnoid space, and is reabsorbed into the blood of the venous sinuses via the arachnoidal villi. The *absorption* here is directly related to the CSF pressure in the cranial cavity. Large holes through the endothelial cells allow proteins to enter the blood.

3. Regeneration of nervous tissue

Severe injury to nervous tissue causes cell death. Neurons are postmitotic cells. For this reason lost neurons cannot be replaced.

There is, however, considerable capacity for regeneration of axons in the peripheral nervous system. Both growth and maintenance of axons require the *nerve growth factors* (NGF). NGF is an essential survival factor for neurons outside the CNS - in particular sensory neurons. NFG binds to receptors belonging to the insulin receptor family (*tyrosine kinase family*).

When a motor axon has been severed, the cell body undergoes **chromatolysis**. This is a neuronal reaction, where the rough endoplasmic reticulum (the *Nissl bodies*) becomes active. The Nissl bodies accumulate proteins required for repair of the axon. The *axonal reaction* is an attempt to repair the fibre by production of new protein structures that are transported along the axon. Therefore, proteins distend the rough endoplasmic reticulum. The axon and the myelin sheath distal to the injury die and are phagocytized. The neuroglial Schwann cells that had formed the myelin remain alive. This is the so-called *wallerian degeneration* named after Waller.

The Schwann cells proliferate and form long rows along the pathway previously occupied by the dead axon. The severed axon regenerates along this pathway, and *growth cones* may eventually reinnervate the target organ.

Neurological injury probably involves excessive *glutamate receptor stimulation* as a common pathway.

Glutamate is the most important of the *excitatory amino acids* (EAAs) in the spinal cord and the brain. Glutamate stimulates the family of EAA-receptors including AMPA-, NMDA-and metabotropic receptors. NMDA means N-methyl-D-aspartate. - Effective glutamate antagonists are applied in clinical studies of pain.

The inhibitory amino acids, GABA and glycine, and the monamines and endogenous opiods inhibit the second-order neurons of the spinothalamic tract.

Fast axonal transport of organelles in the cytosol occurs as rapidly as 0.4 m per day. At this rate synaptic vesicles can travel along the motor axon from the spinal cord to a patient's foot within three days. Fast axonal transport of enzymes and organelles occurs on microtubuli in the axons, and is not interrupted by resting periods in cell compartments outside the transport system (Fig. 3-8). Oxidation of glucose in the mitochondria provides ATP for the Na^+-K^+-pump and for transport filaments and microtubules embedded in the axonal cytoplasm.

Slow axonal transport occurs as diffusion of cytosolic proteins and organelles such as mitochondria. This transport occurs at a rate 100 times more slowly than fast axonal transport. Organelles or enzymes are stored in different cell compartments on their way or their direction of transport reverses.

Axonal transport can be *anterograde*, when it occurs in the direction from the soma to the axonal terminals. Axonal transport can also be *retrograde*, when it occurs in the opposite direction. Here vesicles are degraded by lysosomes, when returned to the soma. A typical example of slow transport is the transfer of the many mitochondria towards the terminal of an axon.

In the CNS, fast neurotransmission is *inhibitory* or *excitatory*. In the neuromuscular junction, *each signal* is always excitatory and sufficient to trigger a muscular contraction. In the neuromuscular junction, acetylcholine is the only neurotransmitter, whereas in the CNS there is a large variety of neurotransmitters.

The sensory system transmits signals from sensory nerve receptors in the body. The nerve receptors are located in the skin, muscles, tendons, joints and viscera. The signals are transferred to the CNS by a pathway of first, second, third, and higher-order neurons. The third and higher order neurons are located in the *thalamus* and the *cortex*. The cell body of the first order afferent neuron is located in the dorsal root or in the cranial nerve ganglia. The signals pass through the spinal cord, the brain stem, and the thalamus before reaching the cerebral cortex.

4. Sensory pathways

Several sensory tracts and pathways synapse in the *nuclei of the thalamus* (the spinothalamic tracts). The *somatosensory thalamus* is a relay station for most sensory modalities. The sensory inputs are processed in somatotopic areas of the thalamus, and are then transferred to appropriate cortical areas. The *somatotopic organisation* is maintained all the way to the cortex.

The *reticular activating system* (RAS) of the brainstem is involved in arousal acting in concert with the thalamus.

The *spinothalamic tract* conveys pain and temperature (lateral tract), and also crude passive touch (ventral tract). The first-order neurons are afferent A fibres (III) which have cell bodies in the spinal ganglia. Second-order neurons cross immediately to the opposite side of the spinal cord, and ascend in the lateral and ventral spinothalamic tract.

Pain and temperature reach the thalamus in the lateral spinothalamic tract (in the lateral funiculus). The second-order axon terminates in the *somatosensory thalamus* (the ventral posterior lateral nucleus and the central lateral nucleus). The third-order neurons pass from the somatosensory thalamus via the *thalamocortical fasciculus* to the *somatosensory cortex* or the *primary sensory cortex* with the *sensory homunculus.* Some third-order neurons also pass to the somatic sensory area II of both hemispheres.

Proprioception and active tactile signals are transmitted through sensory nerve fibres to the spinal cord. Primary afferent fibres ascend in the *dorsal columns* all the way to the medulla oblongata. These primary axons synapse with second-order neurons in the gracile and the cuneate nuclei. These second order neurons cross the midline in the medulla, and ascend in the medial lemniscus to end in the somatosensory thalamus. The *medial lemniscus pathway* transmits proprioception and fine tactile senses.

The *spinothalamic tract* is the most important pathway for *pain*. The *second order neurons* of the spinal tracts have their cell bodies in the lamina I, II and V of the spinal cord. These cells receive excitatory signals from nociceptors in the skin, muscles and viscera. The action potentials from the nociceptors are conducted along the axon to the spinal cord and release neurotransmitters such as the excitatory amino acid, glutamate, and different neuropeptides. When these neurotransmitters bind to the receptors on the postsynaptic membrane of the secondary neurone, they increase the permeability to small ions, and excite secondary, postsynaptic neurons. The secondary neurons of the spinothalamic tract projects mainly to the *contralateral thalamus* by crossing over immediately through the anterior commissure to the opposite side of the spinal cord within the incoming segment.

5. Central opiate receptors

The *endogenous analgesia system* is a pain control system descending from brainstem to the spinal cord.

As an example, this system may explain why a runner who twists his leg during a competition may finish the run before he really feels the pain. As soon as he has passed the goal and stop running the pain often becomes severe, and he cannot run at all.

The cell bodies of the neurons belonging to this system are located in the *periaqueductal grey area* of the midbrain, the *periventricular areas*, locus coeruleus, and the areas surrounding the *aqueduct of Sylvius*. Signals from these cell bodies reach the medullary *raphe magnus* nucleus and the medullary *nucleus reticularis gigantocellularis* with nucleus reticularis *paragigantocellularis* lateralis. The nuclei transmit signals via the *descending pain-suppressing pathway* in the dorsolateral column to a *pain inhibitory complex*. Stimulation or increased tone of the analgesia system can suppress strong pain signals entering the spinal cord through the dorsal spinal horn. These regions contain *opioid receptors*. There are at least 4 types of *central opiate receptors* and their subtypes: **u** for morphine-like drugs, and for enkephalins, and the non-selective *-receptors*.

Endogenous opioids are substances with opiate-like effects. These substances are naturally occurring in the nervous system (-endorphin, met-enkephalin, leu-enkephalin, dynorphin and many others). Endogenous opioids are derivatives of *three* large protein molecules encoded by three different genes. These mother-molecules are *pro-opio-melanocortin* (POMC)*, proenkephalin* and *prodynorphin.*

Enkephalins inhibit both type C and type A (III) pain fibres presynaptically in the dorsal horns. *Enkephalin* is the endogenous ligand for the -opiate receptors. Dynorphin has much higher affinity than morphine and is only found in small quantities close to the dynorphinergic **-**opiate receptors. *-endorphin* is present in the hypothalamo-hypophysary system.

Presynaptically located opiates inhibit depolarization of nerve terminals and reduce synaptic transmission. The purpose of pain is to protect the body from further or imminent harm.

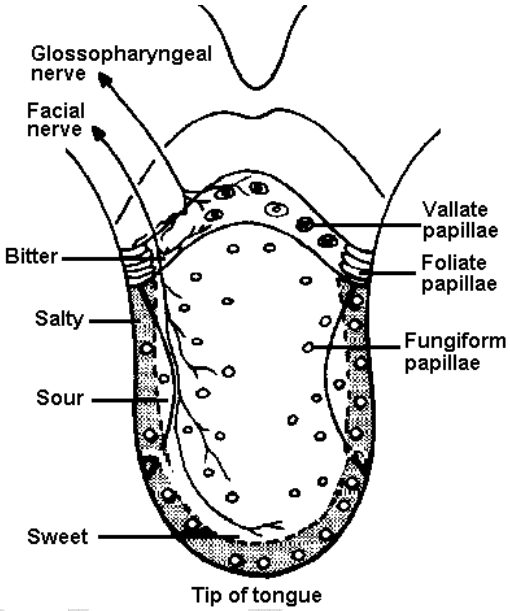
A special type of burning pain is provoked by noxious heat or by capsaicin (which contain a vanillyl-group) in chilli, paprica and pepper. These spices and heat stimuli seem to activate a vanniloid receptor subtype 1 in sensory nociceptors with

terminals in the dorsal horn of the spinal tract. Activation opens Ca^{2+}-channels and the Ca^{2+}-influx is probably involved in the burning sensation.

Gyrus cinguli has the highest density of *central opiate receptors*. Pyramidal cells are contacted by *opiate secreting interneurons* that inhibit arriving pain signals.

6. Taste and smell

The sensations from the anterior 2/3 of the tongue travel with the trigeminal nerve fibres, through the *chorda tympani* into the facial nerve (VIIth), and eventually reach the *solitary tract* of the *brain stem*. Taste signals from the back of the tongue and surrounding tissues are transmitted through the glossopharyngeal nerve (IXth) into the tractus solitarius. All taste fibres synapse in the *nuclei of the solitary tract* and the axons of these neurons project to the thalamus. From the thalamus third-order neurons reach the lower part of the *primary sensory cortex* in the postcentral gyrus



Taste buds and taste pathways from the tongue.

Acids evoke *sourness*, because H^+ stimulates special *H^+-receptors* in the taste buds. *Saltiness* is produced by the anions of inorganic salts. The *Cl^--receptor* is particularly effective in registering saltiness. Our taste buds at the base of the tongue also have *bitter-receptors* stimulated by many long-chain organic compounds. Many alkaloids (quinine, caffeine, and nicotine) also taste bitter. *Sweet-receptors* are stimulated by sucrose, glucose, lactose, maltose, glycerol, alcohol, aldehyde, ketone, and organic chemicals.

In the *upper nasal cavity* the mucous membrane is yellow and termed the *olfactory membrane*. It contains 100 million bipolar neurons called *olfactory cells*. They contain hairs or *olfactory cilia*. The olfactory cells are *smell receptors*. They work as telereceptors, and the smell pathways do not include the thalamic relay station and a neocortical projection area. Instead, the olfactory cells pierce the cibriform plate and synapse in the olfactory bulb. The olfactory tract then transmits the olfactory signals to the olfactory cortex at the surface of the temporal lobe. In the *limbic system*, olfactory information is correlated with feeding behaviour and emotional-motivational behaviour.