LAPORAN AKHIR GERAN INSENTIF

NO GERAN:

USM/PPSP®/Ger.Peny.(14)

CAFLM
(COMPUTER AIDED FIXED LEARNING MODULE)

AMINAH CHE ROMLI

JABATAN FISIOLOGI
PUSAT PENGAJIAN SAINS PERUBATAN
UNIVERSITI SAINS MALAYSIA

ISI KANDUNGAN

BAHAGIAN I

LAPORAN

- BORANG LAPORAN AKHIR PROJEK PENYELIDIKAN JANGKA PENDEK

- LAPORAN AKHIR

1. Pengenalan
2. Objektif & Kaedah
3. Hasil
4. Perbincangan & kesimpulan
5. Carta aliran proses CAFLM

Semua laporan kemajuan dan laporan akhir yang dikemukakan kepada Bahagian Penyelidikan dan Pembangunan perlu terlebih dahulu disampaikan untuk penelitian dan perakuan Jawatankuasa Penyelidikan di Pusat Pengajian.

USM JP-06

BAHAGIAN PENYELIDIKAN
UNIVERSITI SAINS MALAYSIA

Laporan Akhir Projek Penyelidikan Jangka Pendek

1) **Nama Penyelidik:** Aminah Che Romli

Nama Penyelidik-Penyelidik Lain:
(*Jika berkaitan*)

2) **Pusat Pengajian/Pusat/Unit:** Jabatan Fisiologi PPSP

3) **Tajuk Projek:** Penyeliaan modul bantuan pembelajaran secara komputer. (Computer Aided Fixed Learning Modul - CAFLM)

4. (a) **Penemuan Projek/Abstrak**

(*Perlu disediakan makluman diantara 100-200 perkataan di dalam Bahasa Malaysia dan Bahasa Inggeris, ini kemudiannya akan dimuatkan ke dalam Laporan Tahunan Bahagian Penyelidikan & Pembangunan sebagai satu cara untuk menyampaikan dapatan projek tuan/puan kepada pihak Universiti.*)

FLM (Fixed Learning Modul) telah dijadikan bahan untuk dimasukkan ke dalam program CAFLM (Computer Aided Fixed Learning Module) di mana isi kandungannya dipindahkan dari poster ke dalam komputer. Segala isi kandungannya masih lagi dikekalkan.

Modul-modul yang berkaitan dengan Fisiologi telah dipilih dan dibahagikan mengikut tajuk tertentu sepertiSistem Saraf Pusat, Sistem Pencernaan dan Sistem Endokrin. Proses menyiapkan CAFLM dilakukan dengan menaip semula teks, melukis serta mengedit gambar yang terkandung dalam poster FLM.

(Rujuk carta aliran proses CAFLM)

(b) **Senaraikan Kata Kunci yang digunakan di dalam abstrak:**

Bahasa Malaysia	**Bahasa Inggeris**

5. **Output Dan Faedah Projek**

(a) **Penerbitan** (*termasuk laporan/kertas seminar*)
(*Sila nyatakan jenis, tajuk, pengarang, tahun terbitan dan di mana telah diterbitkan/dibentangkan*)

Hasil projek

Berikut ialah tajuk-tajuk FLM yang telah di-masukkan ke dalam komputer:

1. Sistem saraf pusat
2. Sistem pencernaan
3. Sistem endokrin

(b) **Faedah-Faedah Lain Seperti Perkembangan Produk, Prospek Komersialisasi Dan Pendaftaran Paten**
(*Jika ada dan jika perlu, sila gunakan kertas berasingan*)

(c) **Latihan Gunatenaga Manusia**

i) Pelajar Siswazah:

ii) Pelajar Prasiswazah:

iii) Lain-lain: Kakitangan mendapat pendedahan dan kemahiran dengan kerja-kerja yang berkaitan dengan projek ini.

6. **Peralatan Yang Telah Dibeli:**

1. Epson black cartridge SO20 - 189
2. Epson colour cartridge SO20 -191

UNTUK KEGUNAAN JAWATANKUASA PENYELIDIKAN UNIVERSITI

PROF. MADYA ZABIDI AZHAR MOHD. HUSSIN
Dekan
Pusat Pengajian Sains Perubatan
Universiti Sains Malaysia
16150 Kubang Kerian,
Kelantan.

TANDATANGAN PENGERUSI
JAWATANKUASA PENYELIDIKAN
PUSAT PENGAJIAN
fn:borang/adlinaimc/nak

PENGENALAN

CAFLM (Computer Aided Fixed Learning Module) merupakan satu kaedah pembelajaran FLM secara komputer. Oleh kerana terdapat beberapa kelemahan pada poster FLM yang lama seperti masalah kekurangan poster FLM yang timbul akibat pertambahan pelajar yang berlaku pada setiap tahun, maka pihak kami memikirkan satu pendekatan bagi menangani masalah tersebut.

Bagi projek ini segala isi kandungan FLM yang terdapat pada poster masih lagi dikekalkan dan berdasarkan kepada poster FLM tersebut dilakukan beberapa penambahbaikan iaitu melukis semula poster yang kabur atau rosak sebelum dimasukkan ke dalam komputer.

. Sebagaimana yang kita sedia maklum kebanyakkan pelajar sekarang lebih suka menggunakan internet untuk mendapatkan maklumat tertentu. Oleh itu pendekatan pengajaran dan pembelajaran secara ini dapat membantu pelajar membuat rujukan serta ulangkaji tanpa mengira waktu dan tempat, jika dibandingkan dengan penggunaan poster FLM yang dihadkan masa kegunaannya iaitu hanya pada waktu pejabat sahaja. Pelajar juga boleh mencetak nota yang dikehendaki melalui CAFLM ini.

Di samping itu, CAFLM dapat memudahkan kakitangan akedemik membuat pengubahsuaian dari masa ke semasa.

OBJEKTIF

1. Mewujudkan satu sistem FLM yang lebih teratur.
2. Membantu menambahbaikan sistem pembelajaran FLM secara komputer.
3. Mengurangkan kos perbelanjaan bagi penyediaan FLM.

KAEDAH

1. Poster FLM yang ingin dimasukkan ke dalam komputer dikumpulkan.
2. Proses semakan dilakukan dengan menyenaraikan semula tajuk-tajuk dan pemberian kod dilakukan bagi setiap tajuk berkenaan contohnya CNS 01 bagi tajuk pertama dalam sistem CNS, dan Eye 01 bagi tajuk pertama dalam sistem penglihatan.
3. Seterusnya semakkan dilakukan ke atas isi FLM oleh pensyarah yang terlibat.
4. FLM dibahagikan kepada dua iaitu poster yang bergambar atau berajah dan poster FLM yang mengandungi teks sahaja.
5. Kesemua teks ditaip semula atau discan dan dimasukkan kedalam folder yang dilabel sebagai "TEKS".
6. Gambar serta rajah dimasukkan ke dalam satu folder yang dilabelkan sebagai "GAMBAR".
7. Selepas proses semakkan serta pembetulan kedua-dua teks dan gambarajah digabungkan dan dimasukkan kedalam folder yang berasingan mengikut tajuk masing-masing.
8. Seterusnya folder tersebut dihubungkan antara satu sama lain dengan menggunakan perisian 'Netscape Composer' .

HASIL

Berikut merupakan hasil bagi projek CAFLM dan senarai tajuk FLM yang telah dimasukkan ke dalam komputer.

CAFLM
(COMPUTER AIDED FIXED LEARNING MODULE)

PENGENALAN

Sistem CAFLM ini adalah salah satu usaha dari Jabatan Fisiologi untuk meningkatkan sistem pengajaran ke arah penggunaan IT. Sistem ini menggantikan sistem FIX LEARNING MODULE (FLM) lama yang menggunakan kaedah pembentangan poster-poster.

Matlamat sistem CAFLM ini diujudkan antaranya adalah untuk :

- Memudahkan pelajar medapatkan maklumat FLM melalui kaedah komputer.
- Pelajar boleh menggunakan sistem ini samada semasa pembelajaran atau di luar masa pembelajaran.
- Pelajar boleh mengatur masa belajar sendiri khususnya untuk FLM.

SISTEM SARAF PUSAT	SISTEM PENGLIHATAN	SISTEM PENCERNAAN	SISTEM PERNAFASAN	SISTEM PERKUMUHA

SISTEM SARAF PUSAT

Modul 1 Factors regulating cerebral circulation.
Modul 2 Diagram of vasomotor reflexes.
Modul 3 Theory of pain.
Modul 4 Endogenous pain control mechanisms.
Modul 5 Basal ganglia.
Modul 6 The role of basal ganglia in posture and voluntary movements.
Modul 7 Extrapramidal system.
Modul 8 The sequences of singnaling changes that produces the reflex action.
Modul 9 Diagram of nerve.
Modul 10 Nervous control of micturition.
Modul 11 Physiology of micturition instructions.
Modul 12 Propagation of nerve impulse.
Modul 13 The strength-duration curve.
Modul 14 Axoplasmic transport.
Modul 15 Pathway for pain and temperature.
Modul 16 Qualities with pain and pain pathway.
Modul 17 The spinothalamic pathways.
Modul 18 Spinocerebellar pathways.
Modul 19 Somatic sensation- an overall view.
Modul 20 Principle of convergence.
Modul 21 Pathway for conscious proprioception.
Modul 22 Integumentary sensorria.
Modul 23 Gamma reflex loop.
Modul 24 Pathways for nonconscious proprioception.
Modul 25 Cerebral circulation
Modul 26 Diagram of the parasymphathetic (craniosacral) division of the autonomic nervous system.
Modul 27 Classification of nerve fibers.
Modul 28 Mylination of axons.
Modul 29 Cross section of a peripheral nerve showing myelinated and unmyelinated (arrow) fiber.
Modul 30 Cellular organization of peripheral nerves.
Modul 31 Nerve injuries causing bladder dysfunction.
Modul 32 Segmental demyelination.
Modul 33 Degeneration and regeneration in a mylenated nerve fiber.
Modul 34 Primary myelinopathy.
Modul 35 Segmental demyelination.
Modul 36 General pathology of peripheral nerves.
Modul 37 Toxic sensory neuronopathy.
Modul 38 Toxic distal axonopathy.
Modul 39 Class 1 acute nerve injury (eg. compression).
Modul 40 Class 2 nerve injury.
Modul 41 Degenaration and abberant regeneration in (class 3) nerve injury.
Modul 42 Muscle spindle.
Modul 43 Cerebral cortex.
Modul 44 Cutaneous distribution of major peripheral nerves.
Modul 45 Spinothalamic tracts.

SISTEM PENCERNAAN

Modul 1 Gastrointestinal tract
Modul 2 Secretion and digesion
Modul 3 Absorption
Modul 4 Motility
Modul 5 Control of small intestine

SISTEM ENDOKRIN

Modul 1 Hypothalamic hormones and factors regulating adenohypophysial Function
Modul 2 Caracteristics of hypothalamic releasing hormones
Modul 3 Anterior pituitary
Modul 4 Hypothalamic and pituitary hormones
Modul 5 The typical arrangment of negative feedback in the regulation of a Hypothalamus- Pituitary- target gland axis
Modul 6 Neural and endocrine interrelation
Modul 7 Regulation of growth hormone secretion
Modul 8 Patterns of growth hormone secretion
Modul 9 Mechanism of growth hormone action
Modul 10 Regulation of Antidiuretic hormone secretion and thirst

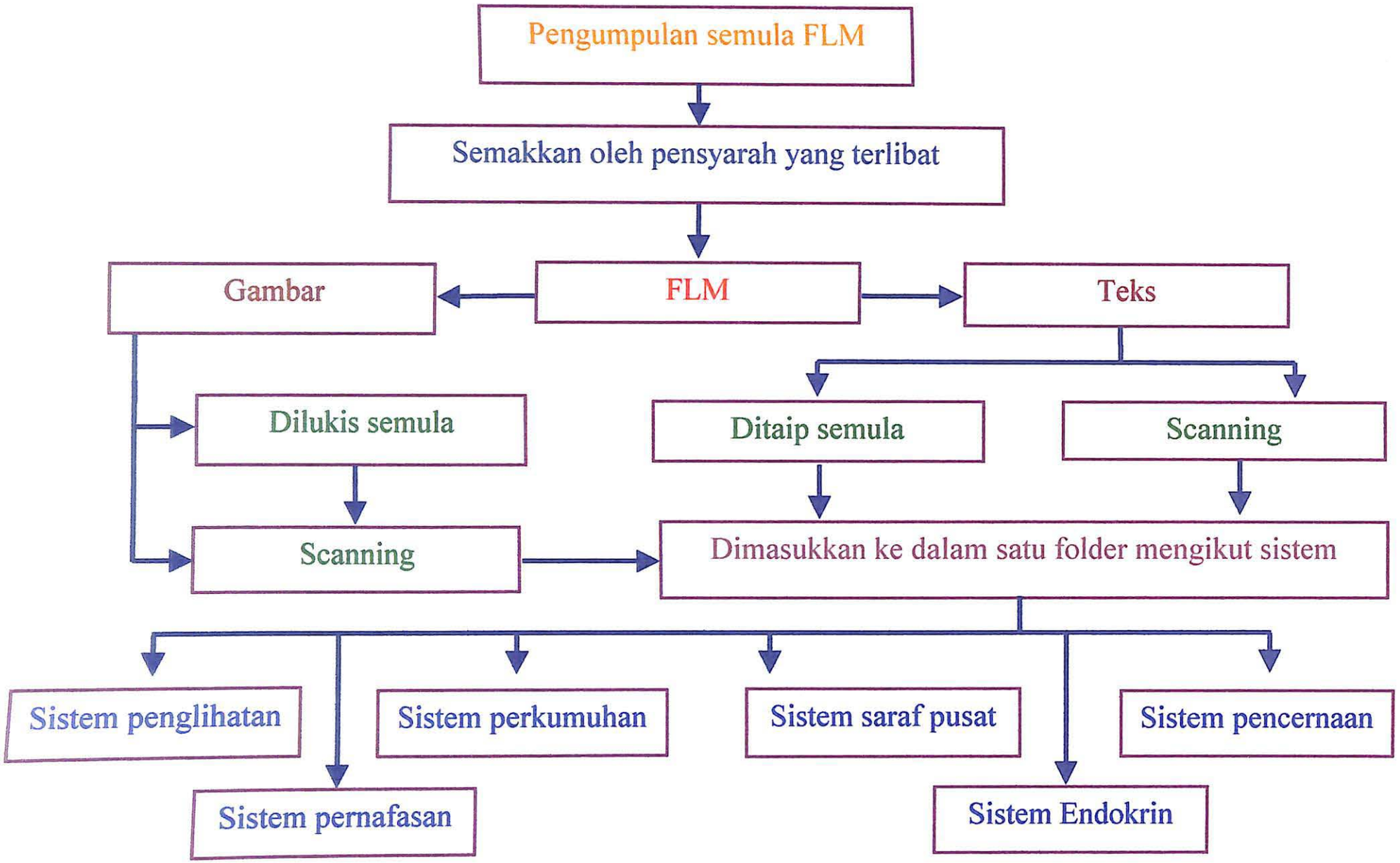
PERBINCANGAN

Isi kandungan FLM yang telah di masukkan ke dalam komputer ini perlu disemak semula untuk memantapkan lagi qualitinya serta mutu persembahan bagi CAFLM. Proses menyiapkan CAFLM mengambil masa yang agak lama kerana berlaku beberapa masalah seperti kehilangan poster FLM yang asal, kerosakkan pada alat scanner dan beberapa masalah yang berlaku selepas proses menyusun serta mengedit semula fail yang telah di masukkan ke dalam komputer.

KESIMPULAN

Kesemua tajuk FLM yang dipilih telah dimasukkan kedalam komputer. Selain daripada tajuk pilihan tersebut terdapat beberapa tajuk lain yang perlu di masukkan ke dalam CAFLM bagi melengkapkanya. Antaranya ialah sistem kardiovaskular sistem pembiakkan dan sistem otot rangka. Untuk kegunaan pelajar kesemua tajuk yang telah dimasukkan ke dalam komputer ini perlu disambungkan melalui internet.

Carta aliran proses CAFLM



BAHAGIAN II

HASIL PROJEK CAFLM

- ❖ Sistem Saraf Pusat
- ❖ Sistem Pencernaan
- ❖ Sistem Endokrin

SISTEM SARAF PUSAT

SISTEM SARAF PUSAT

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Menu Utama

SISTEM SARAF PUSAT

Modul 1 Factors regulating cerebral circulation

I.EXTRINSIC FACTORS

(a) Systemic blood pressure . Fluctuation in systemic arterial blood pressure in the healthy young individual have very little if any effect on cerebral blood flow. Cerebral blood flow will be maintained with fluctuations in systolic blood pressure between 200 and 50 mmHg . A fall in systolic blood pressure below 50 mmHg, may be accompaied by a reduction in cerebral flow. However, because more 02 is extracted, consciousness is usually not impaired. Cerebral blood flow may also decrease if systolic blood pressure rise above 200 mmHg or distolic blood pressure rise above 110 to 120 mmHg .The range of blood pressure fluctuations beyonds which cerebral blood flow is affected is narrower in individuals with arteriosclerosis of cerebral vessels.

b) Blood viscosity , cerebral blood flow is inversely propotional to blood viscosity in man. A reduction in blood viscosity , as occurs in anemia, will increase cerebral blood flow on the other hand , an increase in viscosity , as occurs in polycythemia will decrease cerebral blood flow.

c) Vessel lumen minor reduction in the lumen of carotid and vetrebral arteries are without effect on cerebral circulation. The vessel lumen must be reduced by 70 % to 90 % before a reduction in cerebral circulation occurs.

II . INTRINSIC FACTORS

[a) Autoregulation the single most important factor controlling cerebral circulation is a phenommenon of autoregulation by which cerebral vessel adjust their diameters to maintain a constant flow despite alteration in perfusion pressure. Thus, cerebral blood vessels constrict in response to an increase in intraluminal pressure and dilate pressure and dilate in response to a reduction in intraluminal pressure this phenomenon is particularly useful in shunting blood from healthy regions where intralumial pressure is higher to ischemic regions where a reduction in blood flow has occured, resulting in a reduction in intraluminal pressure. Autoregulation operates independently of, but synergistically with other intrinsic factors such as biochemical changes. The mechanism of autoregulation is poorly under stood.

(b) Biochemical factors several biochemical factors regulate cerebral circulation. Carbon dioxide arterial pco2 is a major factor in the regulation of cerebral blood flow. Hypercapnia (high pco2) produces marked vasodilation and an increase in cerebral blood flow. The reverse occurs in hypocapnia (low pco2). Thus, inhalation of C02 increases cerebral blood flow, whereas hyperventilation decreases cerebral blood flow. Under normal conditions, it is estimated that a change of 1 mmHg in pco2 will induce a 5% change in cerebral blood flow.

The control of cerebral blood flow by c02 is mediated via the cerebrospinal fluid bathing cerebral arterioles. The pH of the cerebrospinal fluid (CSF) reflects the arterial pco2 and is also influenced by the level of bicarbonate in the CSF.

The effect of co2 on cerebral blood flow is important in dampening the effects of tissue pco2 in areas of brain ischemia. The increase in cerebral blood flow in such areas helps to wash out metabolically produced c02 and

thus re-estabishes homeostasis of brain pH. oxygen moderate changes in arterial p02 do not alter cerebral blood flow. However, more marked changes in arterial p02 alter cerebral blood flow in a manner which is the reverse of that described for pco2. Thus, low p02 will increase cerebral blood flow and high p02 will decrease cerebral blood flow. Although the exact mechanism of this effect is not known, it is believed to be independent of changes in pco2 .

NEURAL FACTORS

Sympathetic supply sympathetic innervation of conducting vessels is amply documented from the cervical sympathetic chain. Stimulation of the sympathetic system produces vasocontriction and a decrease in cerebral blood flow. The effect is greater in the internal carotid artery system than in the vertebral basilar system.

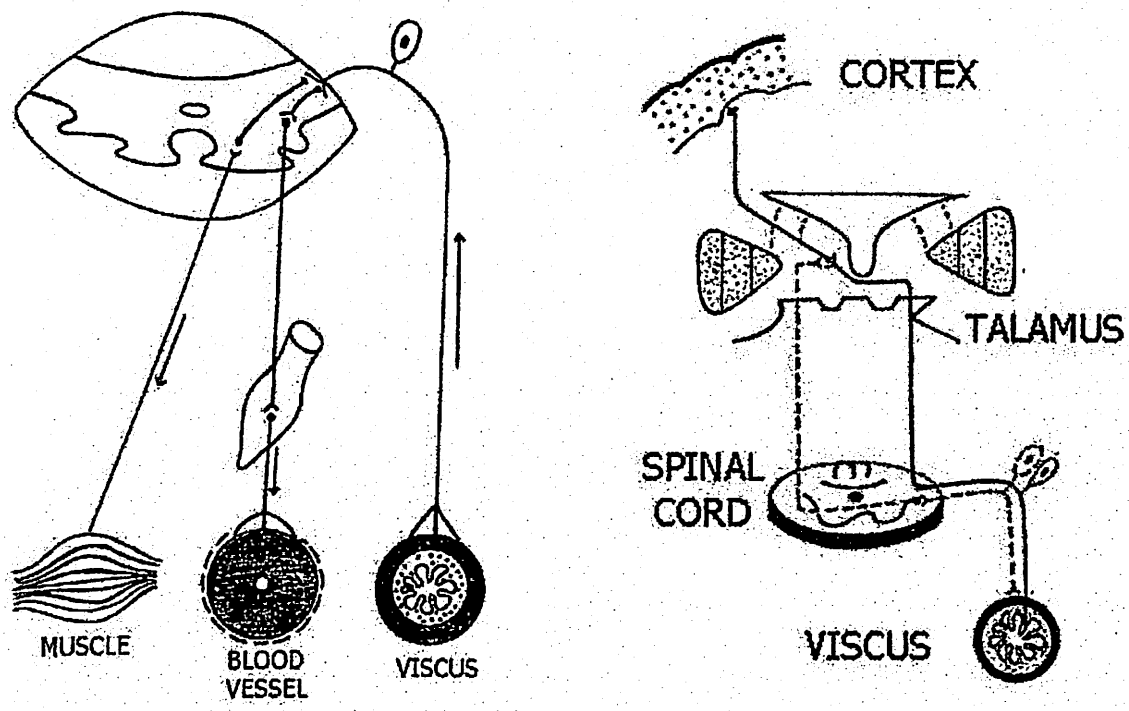
(b) Parasympathetic supply parasympathetic nerve fibers in the regulation of cerebral ciruclation is yet to be found. Thus, neural factors in the regulation of cerebral blood flow are of minor importance when compared with the biochemical factors.

Cerebral blood flow in coma

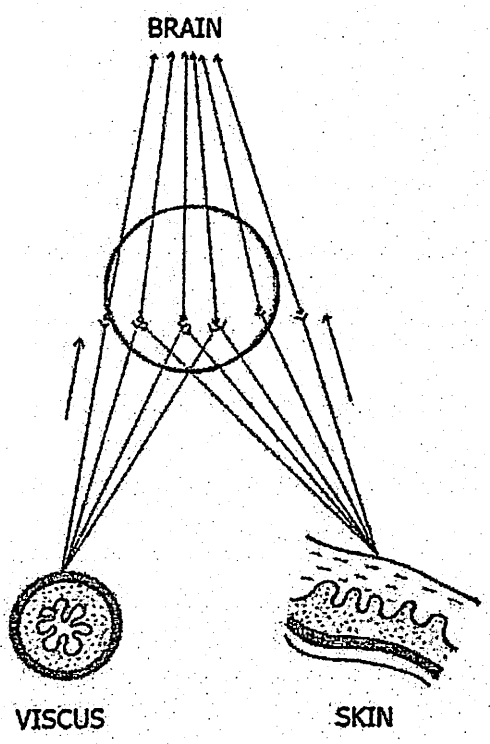
The cerebral blood flow is severely reduced in states of unconsciousness.

SISTEM SARAF PUSAT

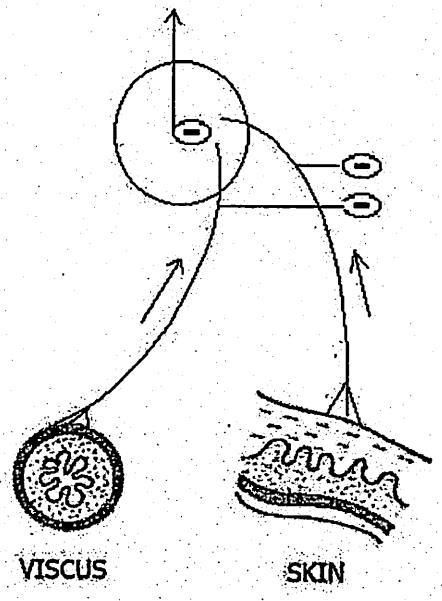
Modul 2 Diagram of vasomotor reflexes



Muscle rigidity and vasomotor reflexes in visceral disease are caused by the excitement of polysynaptic reflex arcs at the spinal cord level.



Cutaneous hyperasthesia in visceral disease occurs because activity in afferent pathways from the diseased viscus lowers the threshold for cutaneous sensory impulse at a common pool of neurons in the CNS.



Afferent pathways from skin and viscera converge on common neurones in spinal cord and thalamus.

SISTEM SARAF PUSAT

Modul 3 Theory of pain.

Prior to the "gate control theory of pain" proposed by Meizack and Wall in 1965, there were two main theories to explain pain mechanisms.

Specificity theory.

According to this theory, pain is a specific modality with its own specific receptors, peripheral fibers, and central pathways. Since its introduction, there has been mounting evidence against this theory.

Pattern theory.

The pattern theory proposed that receptors stimulation elicits a certain pattern of responses which reflects the quality , intensity and duration of the stimulus. These complicated pattern are fed into the central nervous system, which deciphers them and initiates the appropriate response.

Gate control theory.

According to this theory, there is a gating mechanism in the posterior horn of the spinal cord (lamina iii of rexed). This gating mechanism modules activity in spinal cord neurons that give rise to the central tracts for pain (t-cells) and thus increases or decreases the flow of impulses from the periphery into the central nervous system. The gating mechanism in the posterior horn is influenced by two types of inputs from the periphery.

One input is via a small (a - delta or c) fiber system which is continuously active , thus keeping the gate open. This system has a facilitatory role in pain mechanisms and acts to enhance the effect of incoming impulses. The second input is via a large (a beta), thickly myelinated fiber system which fibers in response to a stimulus both system project upon neurons in lamina iii of rexed, which is considered to be the modular center for pain in the spinal cord. The thin fibers inhibit and the thick fibers facilitate neurons in this lamina. Both fiber system also project upon neurons in this lamina. Both fiber system also project upon neurons in rexed laminae v to viii (t-cells). Their action here is purely facilitatiory. Futhermore, neurons in lamina iii of rexed have a presynaptic inhibitory effect on both the small and large fibers systems projecting upon laminae v to viii.

The gate control theory may be summarized as follows:

The ongoing activity which precedes a stimulus is carried solely by the small fiber system and tends to keep the gate open and ready to receive new impulses.

A superimposed peripheral stimulus will activate both the small and large fiber system. The discharge from the latter initially fibers the tract neurons (t-cell) in laminae vto viii through the direct facilatory route. Then partially closes the gate through its action on lamina iii neuron (facilitation of pressynaptic inhibition).

If the stimulus is prolonged , the large fiber system adapts, resulting in a relative increse in small fiber system adapts, resulting in relative increase in small fiber system activity, the gate opens , further increasing activity in laminae v to viii.

The gating mechanism in the spinal cord is under the control of the brain. In this way , brain mechanism concerned with attention , emotion and memory can influnce pain impulses in the spinal

cord.

Present concepts

The present status of our understanding of pain mechanism can be summarized by the following observations.

The pain receptor status is not specific for pain stimuli but is specialled. Below a certain threshold, anoxious (painful) stimulus will elicit responses from all types of receptors.

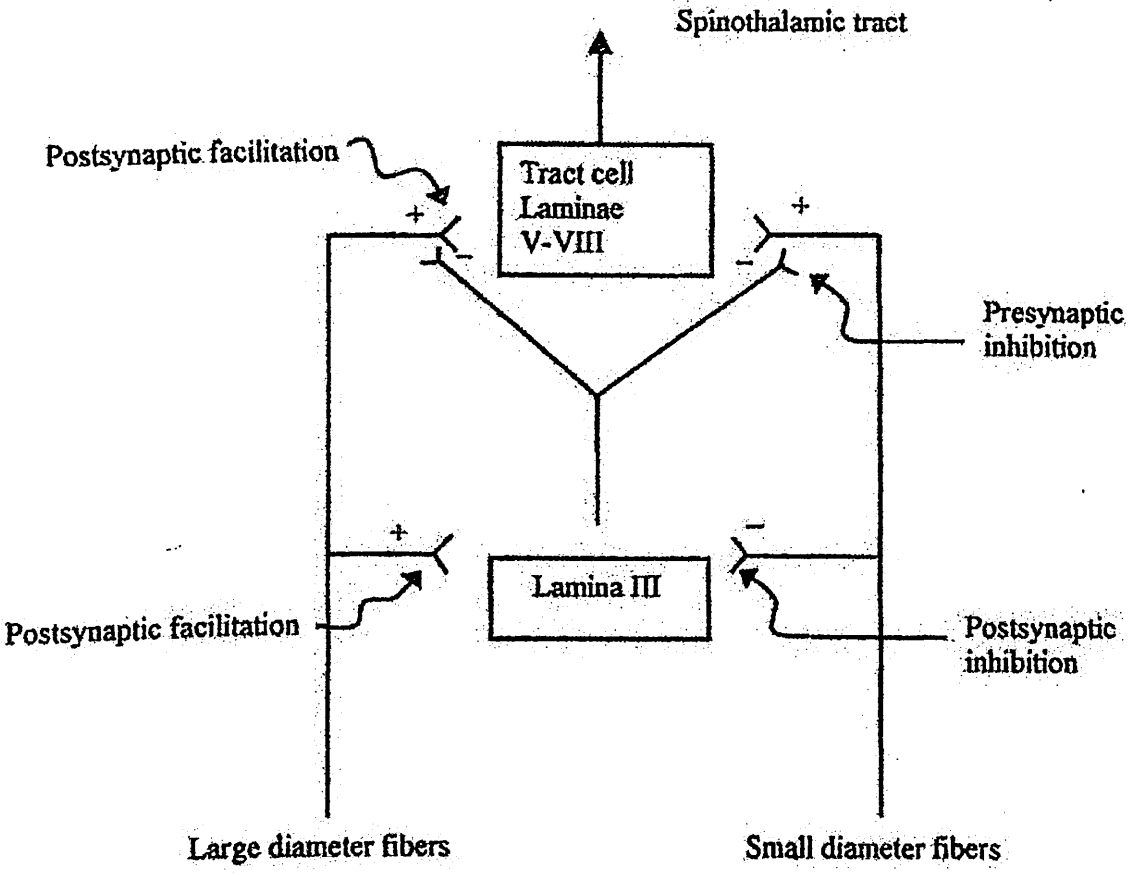
Pain impulses are conducted via the small a- delta fibers and c- fibers (peripheral nerve) the larger a- beta also play a role in pain mechanism.

The spinal cord posterior horn is a center for integration of pain mechanisms. It integrates activity arriving from the periphery with that arriving from cortical and subcortical regions.

Pain impulses are conducted in the neuraxis via two major pathways. These are a direct (neurospinothalamic and trigeminothalamic) pathway and a multisunaptic (spinoreticulothalamic) pathway.

The direct ascending pathway project upon posterior nuclei of the thalamus (vpl and vpm), whereas the multisynaptic pathways project upon the intralaminar nuclei of the thalamus.

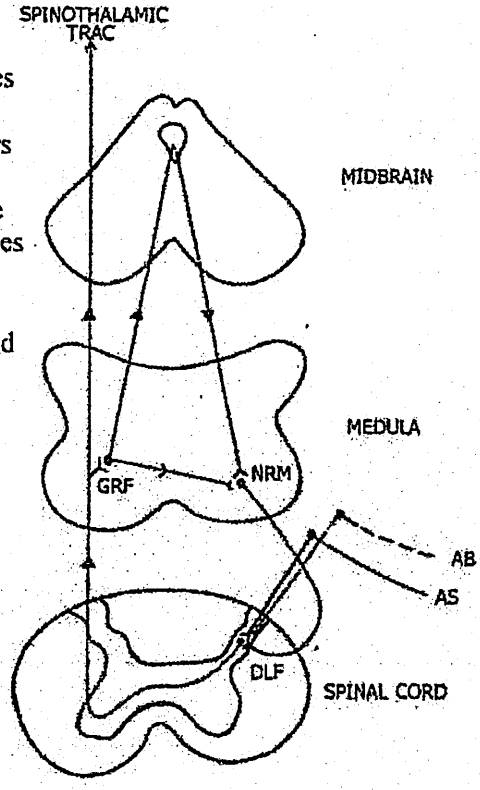
The cortical projection areas include the primary somasthetic cortex of
The parietal lobe, the frontal cortex, and the limbic lobe. The parietal cortex is concerned with the appreciation of the sharp,well-localized type of pain,
The frontal cortex is concerned with intellectual
Appreciation and affective reaction, and the limbic lobe is concerned with the memory of painful experience and the emotional reaction to pain. Pain can be appreaciated at subcortical levals, primarily in the thalamus.


Spinothalamic tract
Postsynaptic facilitation
Tract cell
Laminae
V-VIII
Presynaptic
inhibition
Lamina III
Postsynaptic facilitation
Postsynaptic
inhibition
Large diameter fibers
Small diameter fibers

SISTEM SARAF PUSAT

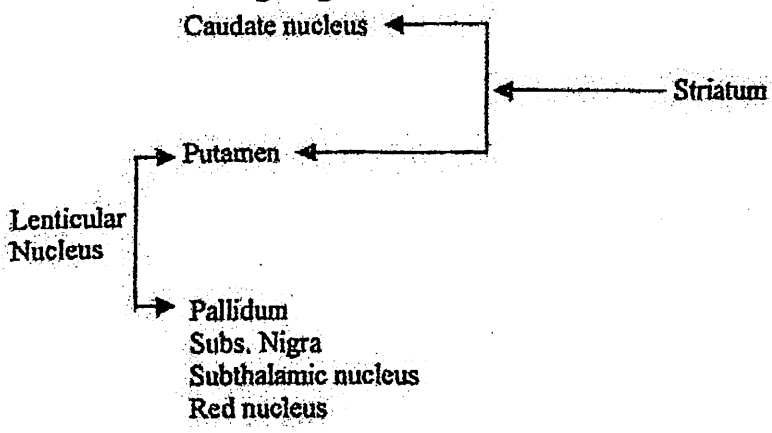
Modul 4 Endogenous pain control mechanisms.

Electrical stimulation in the periaqueductal grey matter produces longlasting analgesia in man and experimental animals. The periaqueductal grey matter is moderately rich in opiate receptors and enkephalin while naloxone, a morphine antagonist, blocks the electrically produced analgesia. It therefore seems probable that opiates activate an efferent brainstem system that suppresses pain at a segmental level. The descending pathway seems to be mainly in the dorsolateral fasciculus of the spinal cord (Figure) since lesions in this region reduce or abolish analgesia produced by opiates or electrical stimulation of the periaqueductal grey matter. The serotonin-rich midline raphe nuclei of the medulla also appear to be involved in the descending pathway although serotoninergic descending pathways through the dorsolateral fasciculus also exist. There is anatomical and physiological evidence to suggest that, in addition to receiving inputs from the opiate-sensitive brainstem structures, the midline raphe nucleus of the medulla also receives major though indirect somatosensory inputs from pain pathways, possibly by way of the reticular formation1.



ISTEM SARAF PUSAT

Modul 5 Basal ganglia.



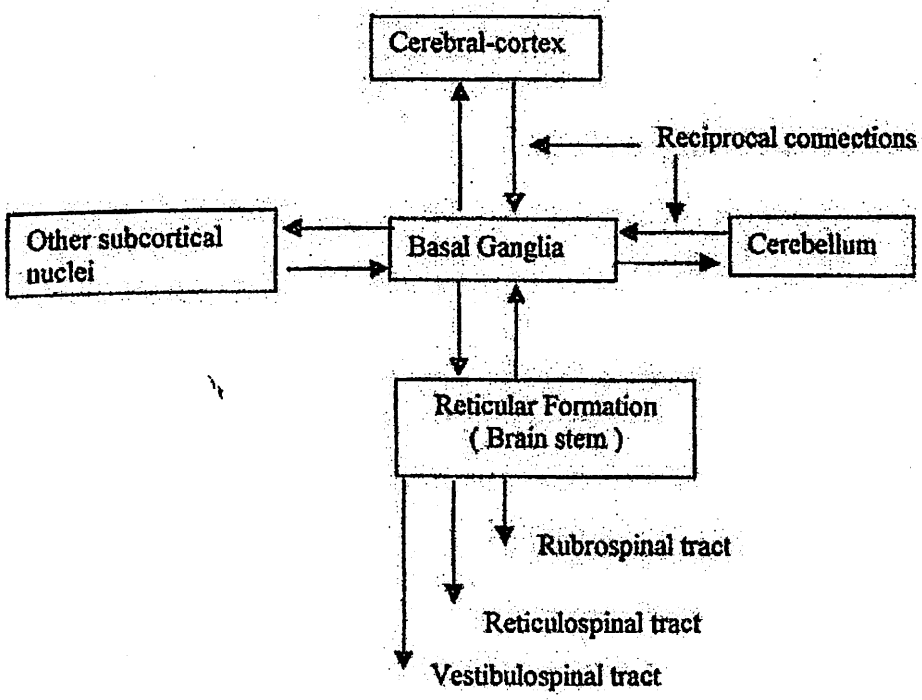
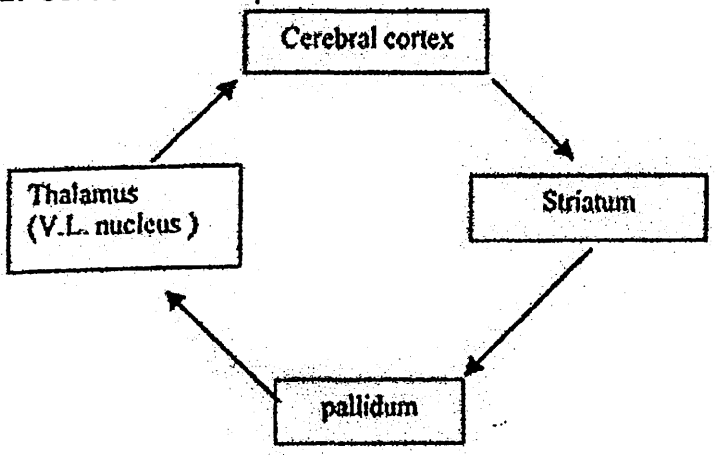


Diagram showing neural connections of Basal Ganglia with other structures of the brain.

SISTEM SARAF PUSAT

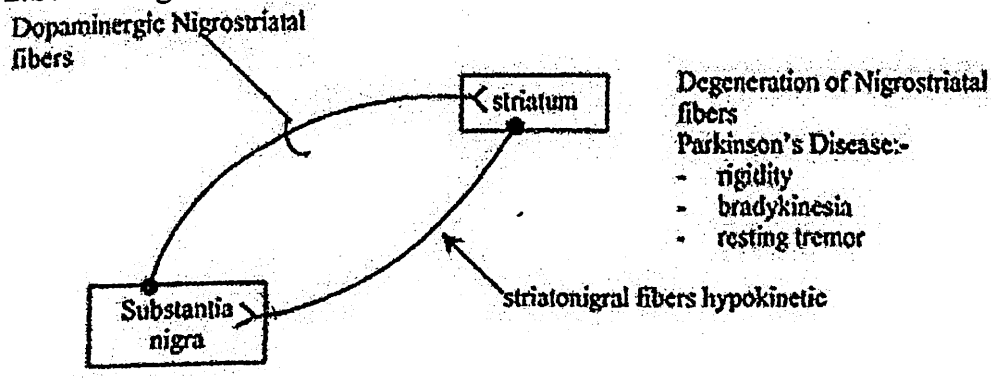
Modul 6 The role of basal ganglia in posture and noluntary movements.

1. Cortico-striato-pallido-thalamo-cortical:



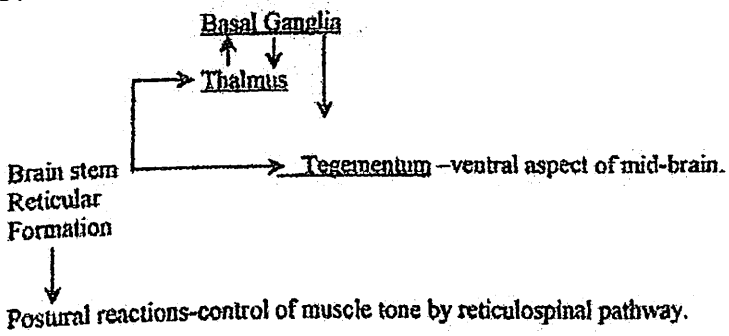
Function:
- postural adjustments for voluntary movements
- programming of ramp voluntary movements

2. Striato-nigro-striatal:



Degeneration of Nigrostriatal fibers
Parkinson's Disease:-
- rigidity
- bradykinesia
- resting tremor

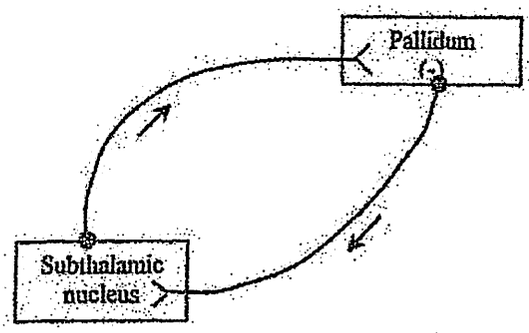
3. Pallido-thalamo-striatal, pallido-tegmental.



4. Pallido-subthalamo-pallidal.

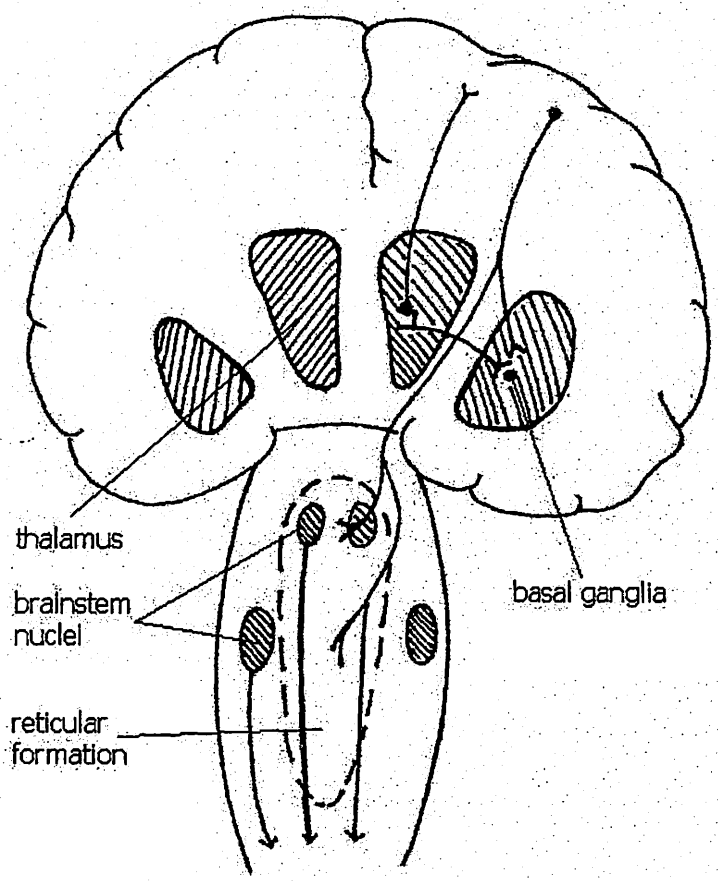
Damage of the subthalamic nucleus :

- Hemiballismus → (contralateral)
" sudden onset of involuntary movements".
- Hyperkinetic type.



SISTEM SARAF PUSAT

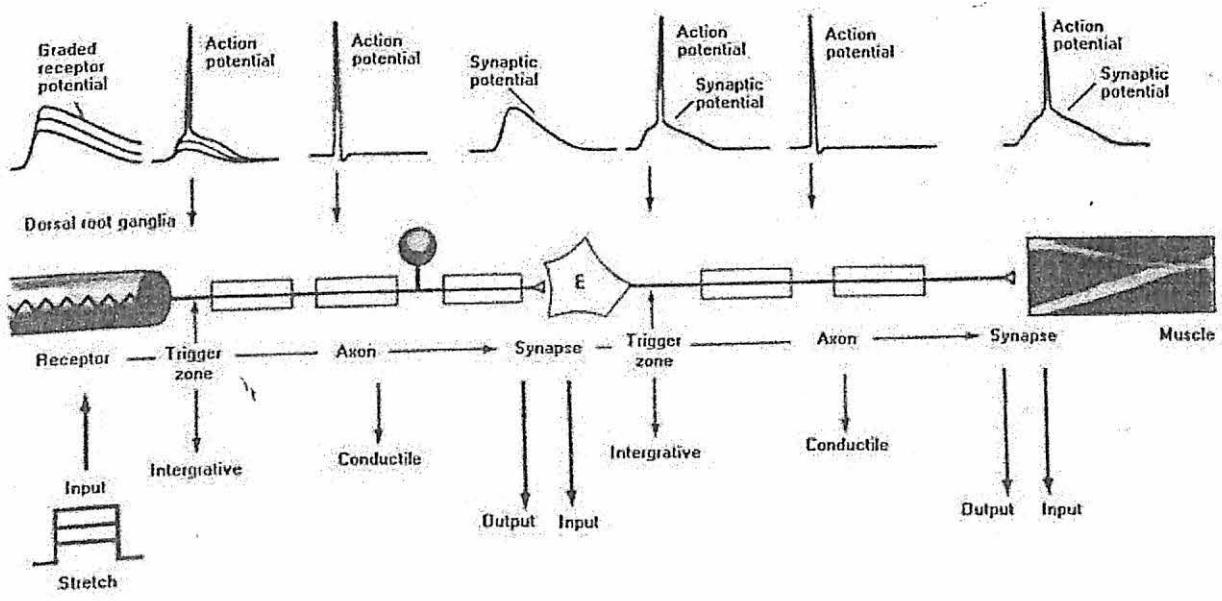
Modul 7 Extrapramidal system.



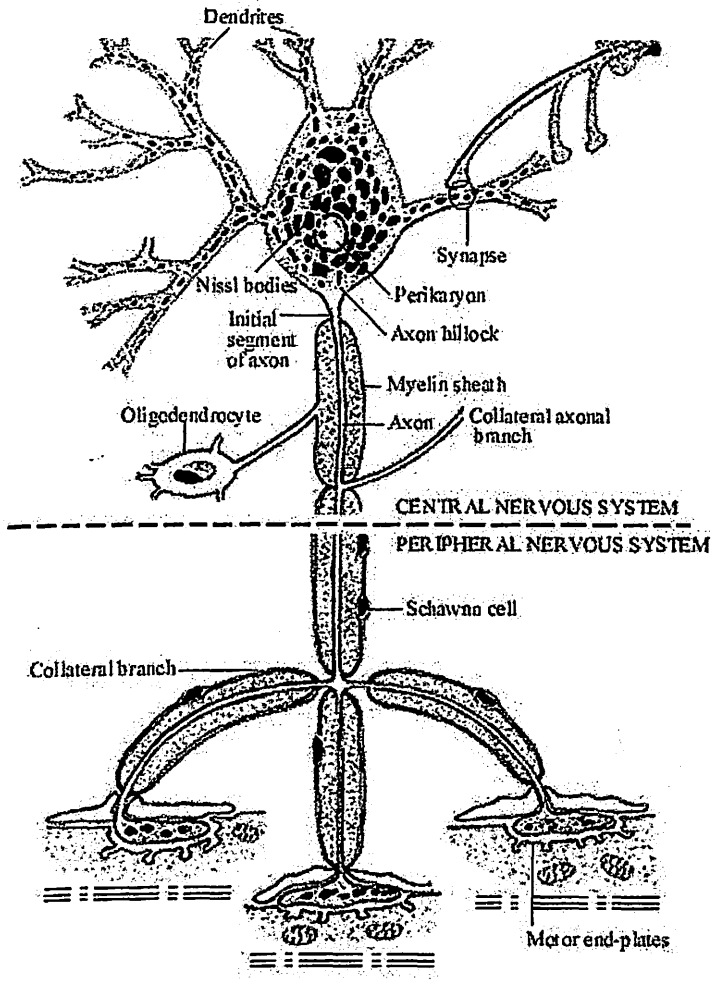
SISTEM SARAF PUSAT

Modul 8 The sequences of singnaling changes that produces the reflex action.

Grade stretch of muscle produces a graded receptor potential that propagates passively to the trigger zone at the first node of ranvier. If the potential is sufficiently large, it will trigger an action potential that will propagate actively and without fail along the axon, the conductile component of the neuron, to the terminal region. At the terminal the depolarization produce by the action potential gives rise to a secretory potential that leads to release of transmitter substances. The transmitter diffuse across the synaptic cleft and interact with receptor molecules on the external membrane of the post synaptic cell to initiate a synaptic potential. The synaptic potential that propagates to the terminal of the motor neuron and leads ultimately to a synaptic potential in the muscle that initiates an action potential producing a contraction.



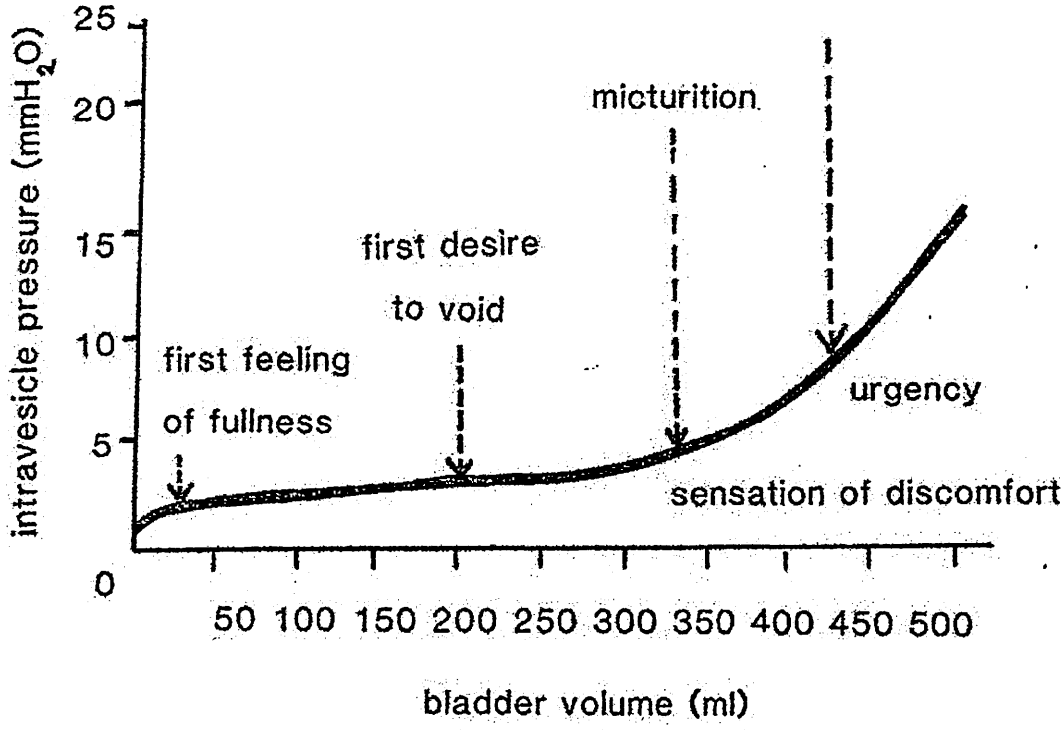
SISTEM SARAF PUSAT
Modul 9



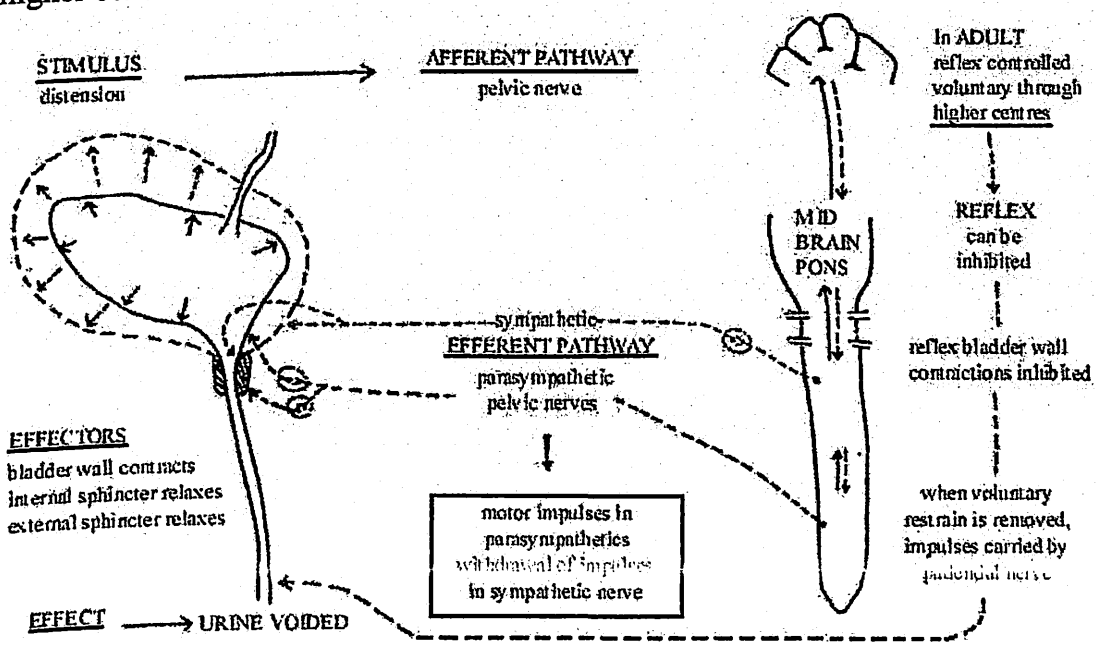
SISTEM SARAF PUSAT

Modul 10 Nervous control of micturition.

Urine is formed continuously in the kidneys. It collects in the urinry bladder which expands till about 300cc. When full, the desire to micturate is experienced.



Micturition is essentially a reflex with centres in the spinal cord. This reflex can be controlled by higher centres.



When bladder is empty and beginning to fill,

Activation of _____ } relaxation of _____
Inhibition of _____ } contraction of _____

When bladder is full and voiding is to occur,

Activation _____ } relaxation of _____
Inhibition _____ } contraction of _____

Urination can occur solely by reflex action not involving higher centers.
Filling ⟶ reflex contraction of bladder wall and relaxation of sphincters.
Seen in infant with no voluntary control over the sphincters.

ISTEM SARAF PUSAT

Modul 11 Physiology of micturition instructions.

1. Read the objective listen below.
2. This FLM will help you RECALL the PHYSOLOGICAL of MICTURATION.
3. Each section of this module represents one important aspect on the physiological of urine formation.
4. Try to answer all question BEFORE looking at the answers.
5. Go through the objectives again and check if you have achieved them.

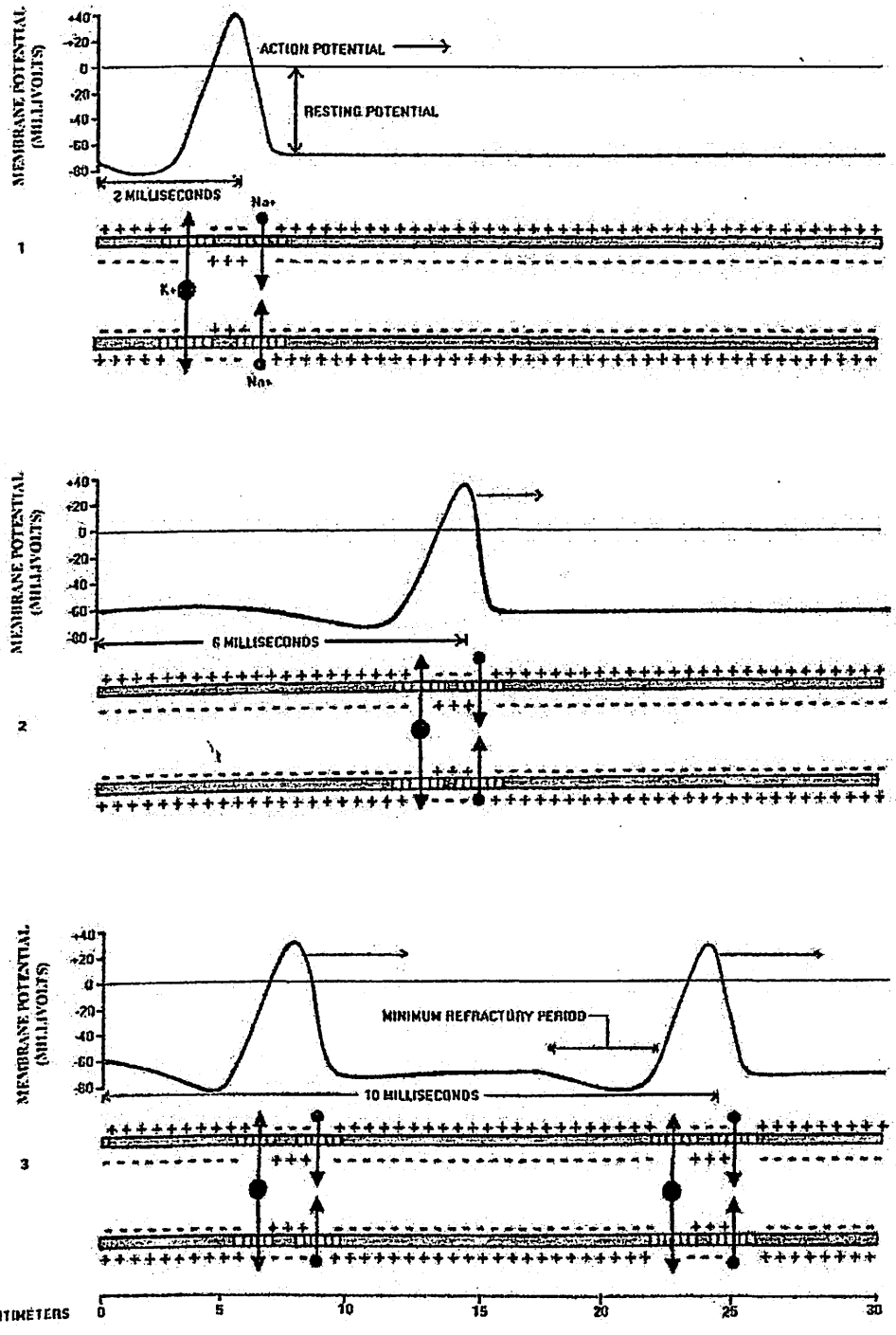
Understanding the process of micturition will help you understand the pathophysiology of bladder dysfunction. If you are in doubt, DON'T FORGET TO ASK.

Objectives:

1.Describe the nerve supply of the bladder and their relationship to micturition
2. Describe the nervous control of micturition.
3. Outline the types of nerve of injuries that can cause bladder dysfunction.

SISTEM SARAF PUSAT

Modul 12 Propagation of nerve impulse.



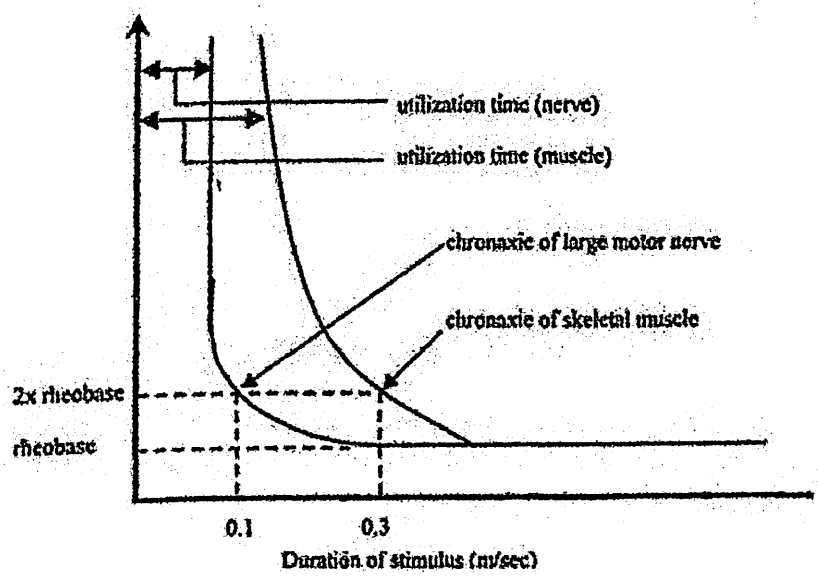
Propagation of nerve impulse along the axon coincides with a localized inflow of sodium ions (Na+) followed by an outflow of potassium ions (K+) through channels that are "gated" or controlled, by voltage changes across the axon membrane. The electrical event that sends a nerve impulse travelling down the axon normally orginates in the cell body. The impulse begins with a slight depolarization or reduction in the negative potential across the membrane of the axon where it leaves the cell body. The slight voltage drifts open some of

the sodium channels, shifting the voltage still further. The in-flow of sodium ions accelerates until the inner surface of the membrane is locally positive. The voltage reversal closes the sodium channel and opens the potassium channel. The outflow of potassium ions quickly restores the negative potential. The voltage reversal known as the action potential, propagates itself down the axon (1,2). After a brief refractory period, a second impulse can follow (3).

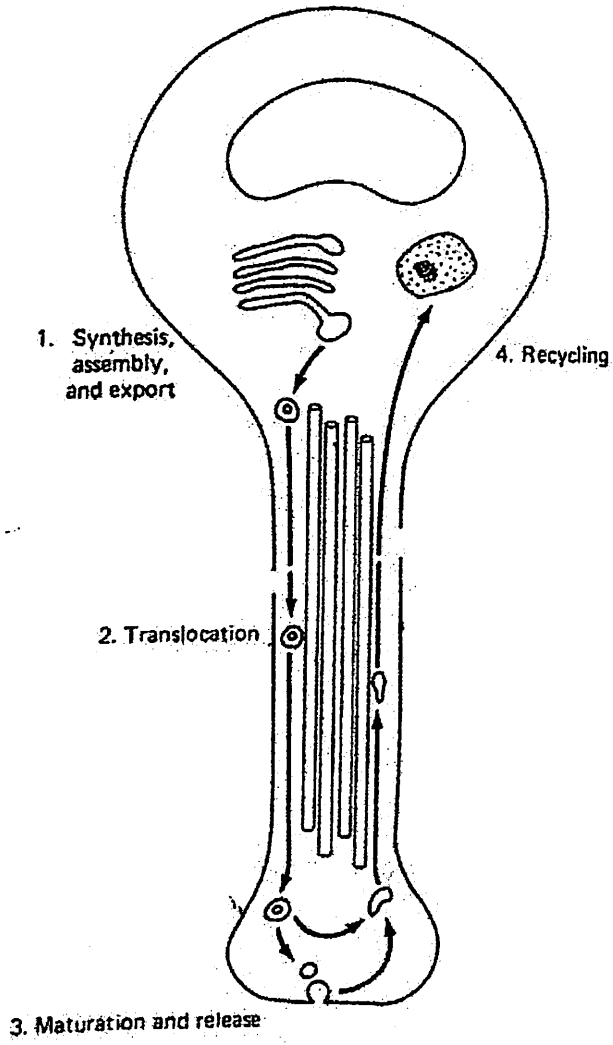
SISTEM SARAF PUSAT

Modul 13 The strength-duration curve.

The relationship between the strength of the stimulus and its duration is an important one, which is shown in the strength-duration curve. It can be seen that a certain minimum duration, the utilization time, is needed for even the strongest stimulus to voke a response. The weakest stimulus that will excite the tissue, provided it is applied for a long enough time, is called the rheobase. In order to compare excitability of different fibers, a stimulus of twice the strength of rheobase is used to test the length of time needed to excite a response. This time is know as chronaxie. Time chronaxie of nerve fibers varies according to tee size and type of the fiber (as well as with its ability to accomodate), but it serves as a rough indicator of excitation time. It is used a clinical device to determine whether the innervation of a muscle is intact. If it is intact, the chronaxie will be compound because of the difference in excitability between the nerve and muscle fibers, if the nerves rave been destroyed, a simple chronaxie will be obtained, corresponding only to the muscle fibers .The strength-duration relationship applies only to currents that rise to peak intensity rapidly. Slowly rising currents sometimes fail to fire the nerve because the nerve in some way adapts to the applied stimulus, a process called accommodation.



The strength-duration curve shows that a minimum duration (the utilization time) is essential for even the strongest stimulus to evoke a response the chronaxie of a large motor nerve is considerably less than that of skeletal muscle, demonstrating the greater excitability of the nerve.



The life history of synaptic vesicles and other membranous organelles involved in synaptic transmission. Proteins and lipids are synthesized and incorporated into membranes within the endoplasmic reticulum and golgi apparatus of the neuron's cell body. Organelles are then assembled from these components and exported from the cell body into the axon where they are rapidly moved towards terminals by fast axonal transport. Some of the material is deposited along the axon to maintain the axolemma. Synaptic visicles and their precursors reach the neuron's terminals to participate in the exocytotic release of transmitter substances. The membranes of synaptic vesicles are used many times over in the release process. At random, a small amount of the membrane becomes degraded by lysosomes and this material is returned to the cell body by fast retrograde axonal transport. The degraded membrane is partly recycled; its residue is progressively accumulated in large, end-stage lysosomes that are characteristic of neuronal cell bodies.

SISTEM SARAF PUSAT

Modul 14 Axoplasmic transport.

Axoplasmic transport

Axoplasmic transport is the movement of perikargally (cell body) material into axons and dendrites. This mechanism supplies the dendrites with protein and other constituents necessary for structure and functional intergrity.It also carries neurotransmitters and the enzymes needed for their metabolism. Since axoplasmic transport supports such functions, its b reakdown can lead to pathology.

In mammalian nerve fibers, isotope labelled studies show the presence two types of transport:
(i) A slow axoplasmic transport at 0.5 - 3 mm/day know as axoplasmic flow and
(ii) A fast axoplasmic transport at about 400 mm/ day which is known as axonal transport.

Anterograde (or orthograde) axonal transport is the transport of materials away from the cell body.

Retrograde axonal transport is the transport or returning worn out materials from terminals to the cell body, either for degradation or for restoration. The rate is about one half to two thirds that of orthograde transport.

Fast transport

Using ligation or crush techniques, studies have shown acetlycholinesterase, norephinephrine, including a wide variety of polypeptides, proteins, enzymes are all transported by fast axonal transport.

It is dependent an oxidative phosphorylation and a local supply of ATP is needed along entire length of the fiber for energy.

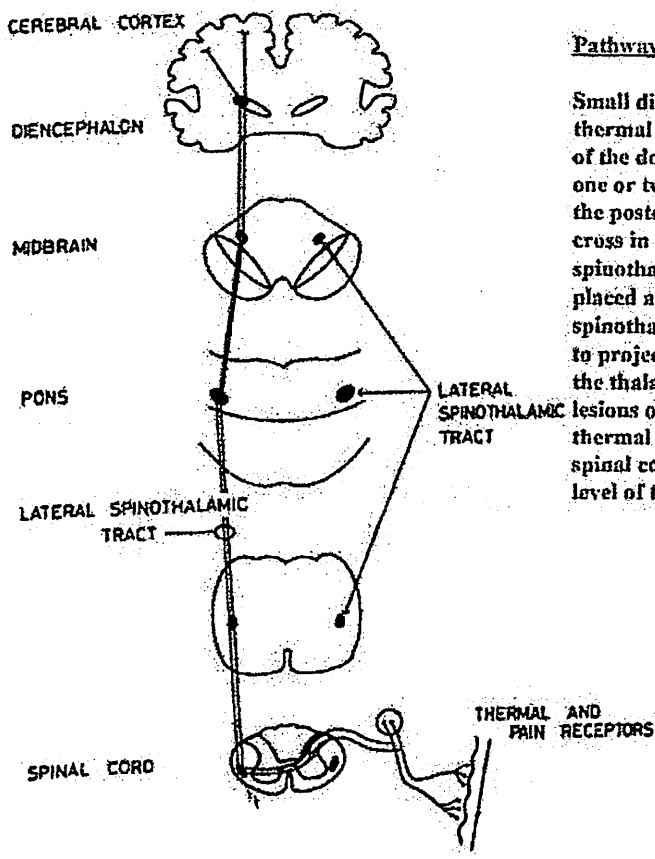
Slow transport

Phospholipids and phosphoproteins are transported by slow axonal transport. Amino acids too are transported by this mechanism.

Some neurotropic viruses, tetanus toxin and certain viruses (rabies/polio) reach the central nervous system by ascending from peripheral nerve terminals to cell body by grade transport.

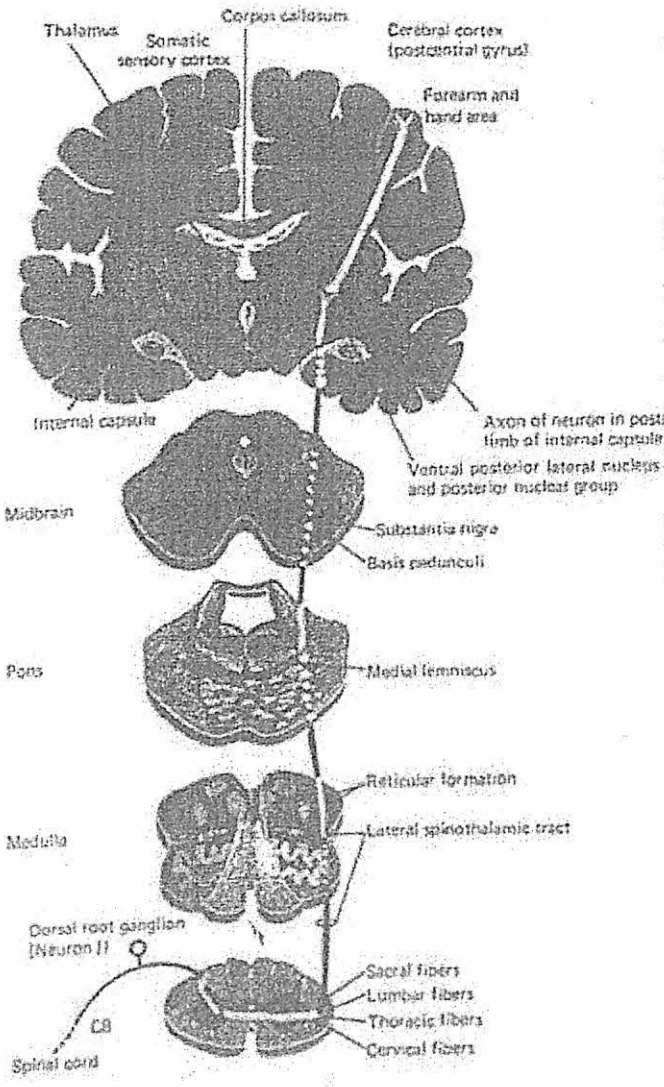
SISTEM SARAF PUSAT

Modul 15 Pathway for pain and temperature.



Pathway for pain and temperature

Small diameter, thinly myelinated fibers conveying pain and thermal sensations enter the spinal cord via the lateral division of the dorsal (posterior) root. Within the spinal cord they ascend for one or two segments and project upon neurons in several laminae in the posterior horn. From tract neurons in laminae v to viii, axons cross in the anterior white commissure and form the lateral spinothalamic tract in the lateral fun iculus. Sacral fibers are laterally placed and cervical fibers are more medially placed in the tract. The spinothalamic tract ascends throughout the spinal cord and brain stem to project upon neurons in the ventral posterolateral nucleus (VPL) of the thalamus. Axons of VPL neurons project to the somasthetic cortex lesions of the spinothalamic tract result in diminution or loss of pain and thermal sense contralateral to the lesion. when the tract is affected in the spinal cord, the sensory deficit begins one or two segments below the level of the lesion.



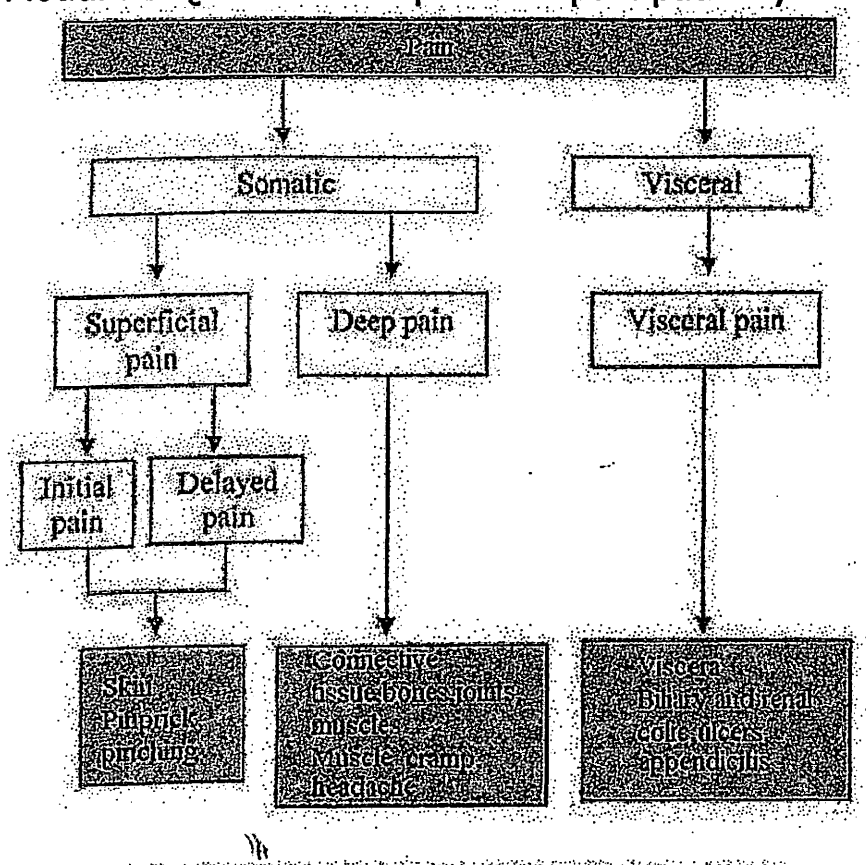
The anterolateral system mediates pain and temperature sense

The anterolateral system mediates pain and temperature sense the anterolateral pathway crosses in the spinal cord and their cell of origin (in the dorsal horn) are postsynaptic to the primary afferent fibers. In addition, the incoming afferent fibers ascend one or two segments before synapsing on cells that project to the brain stem or thalamus.

The major termination of the pathways in the anterolateral columns is in the thalamus. This is the spinothalamic tract. The anterolateral pathways also project to the lower brain stem and the midbrain. The termination in the lower brain stem is rather diffuse and is also called the spinoreticular tract because it terminates in a region of the central core of the brain stem known as the reticular formation. the termination in the gray matter of the midbrain in the region surrounding the aqueduct of sylvius called the mesencephalic periaqueductal gray, is thought to be important in endogenous mechanisms for pain inhibition.

SISTEM SARAF PUSAT

Modul 16 Qualities with pain and pain pathway.



Pain qualities (white boxes). The localization of each quality is indicated (gray boxes), with example of each specific forms of pain.

Qualities of pain.

Pain can be categorized in terms of a number of qualities, defined either by the site of origin of the pain or by its nature.

The major subdivision of the modality pain is that into the qualities somatic pain and viceral pain.

Somatic pain.

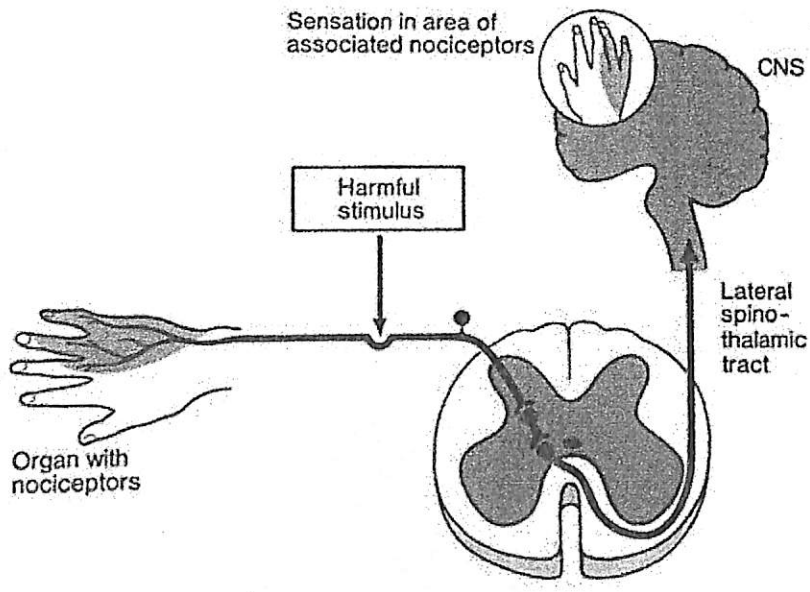
If somatic pain pain originates in the skin it is called superficial pain if it cames from the muscles, bones, joint or connective tissue it is called deep pain. Superficial and deep pain are thus (sub) qualitites of somatic pain.

If superficial pain is produced by piercing the skin with a needle, the subject feels a sharp "flash" of pain, a readily localizable sensation that fades a way rapidly when the stimulus stops. This initial pain is often followed, particularly at high stimulus intensities, by a delayed pain with a latency of 0.5- 1.0 sec. The delayed pain has a dull (burning) character, is more difficult to localize and dies out more slowly, a good example is the pain felt in response to squeezing of on interdigital fold.

The most familiar example of deep pain is headache probably the commonese form of pain experienced by humans. Deep pain is dull in nature, is poorly localizable as a rule and tends to radiate into the surroundings.

Visceral pain.

This pain, too, tends to be dull or diffuse in character and it resembles deep pain in that similar autonomic responses accompany it. It is remarkable that when the viscera are exposed under local anesthesia they can be squeezed or cut without pain as long as the parietal peritoneum and the mesenteric roots are not stimulated, but rapid or extensive stretching of the hollow organs dicits severe pain. Spasmo or strong contractions of smooth muscle are painful, especially when they are associated with inadequate circulation eon (ischemia).



Pain pathways: fast and slow fibers.

There is a fast pathway for pain along small a fibers conducting from 3 to 2o m /sec. These reach the somatosensory cortex to give rise to a pricking pain.

Slow impulses travelling along the thinner c fibbers at 0.5 to 2m/sec end in the reticular activating system and the thalamus, resulting in generalized slow, burning pain. These pain signals are poorly localized and additive, so that they become increasingly disturbing if ignored. This mechanism ensures the activation of the reticular activating system itself, which regulates the arousal and protective responses of the organism.

Response to pain.

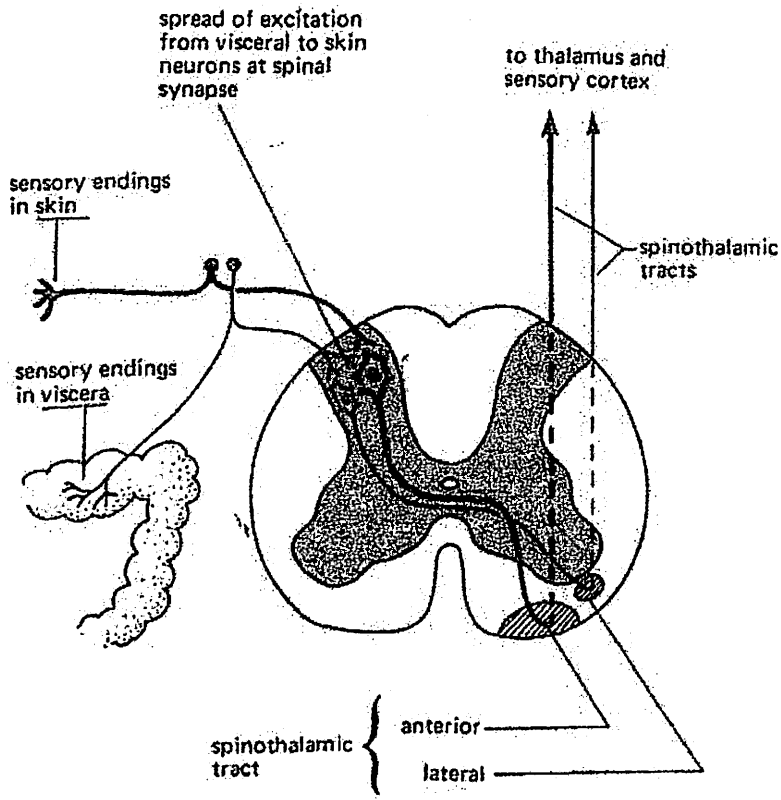
The individual response to pain varies widely, and the differences are more likely central, rather than being differences in receptor threshold.

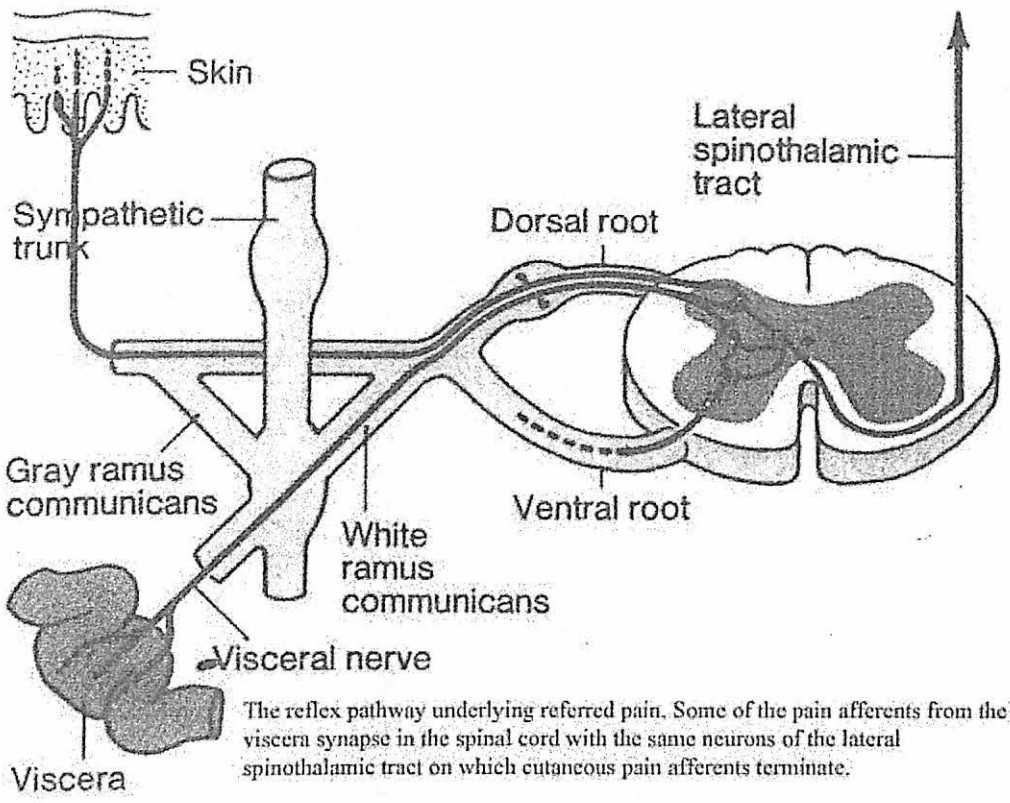
Reffered pain.

Reffered pain is pain initiated in one of the visceral organs and felt else where in the body, commonly in the skin.it is believed that the referral of pain occurs when visceral pain fibers are so severely stimulated that they activate synapses in the spinal cord and thalamus that normally only receive impulses from skin. This is interpreted by the somatosensory cortex as pain coming from the skin.

A well known example is that of pain originating at the heart but appearing to come from the chest and a narrow strip along the medial aspect of the left arm, because the relationship between dermatomes and internal organs (innervation from the same spinal segment) is known, such referred pain is an important aid to diagnosis

The production of referred pain probably occurs as shown in figures. Some of the pain afferents from the skin and the internal organs that enter a given spinal segment are connected with the same neurons of origin of the spinothalamic tract. Excitation of such cells is interpreted as pain at the periphery because this interpretation is usually appropriate in the body's experience. When an internal organ is diseased, there is often a further consequence of the convergence of noczceptive afferents from the organ and the associated dermatome upon neurons in the pain pathway –hyperesthesia (over sensitivity) of the skin in the dermatome. The reason is that the excitability of the spinal interneurons is increased by the visceral impulses so that a stimulus to the skin causes greater than normal central activity (facilitation)



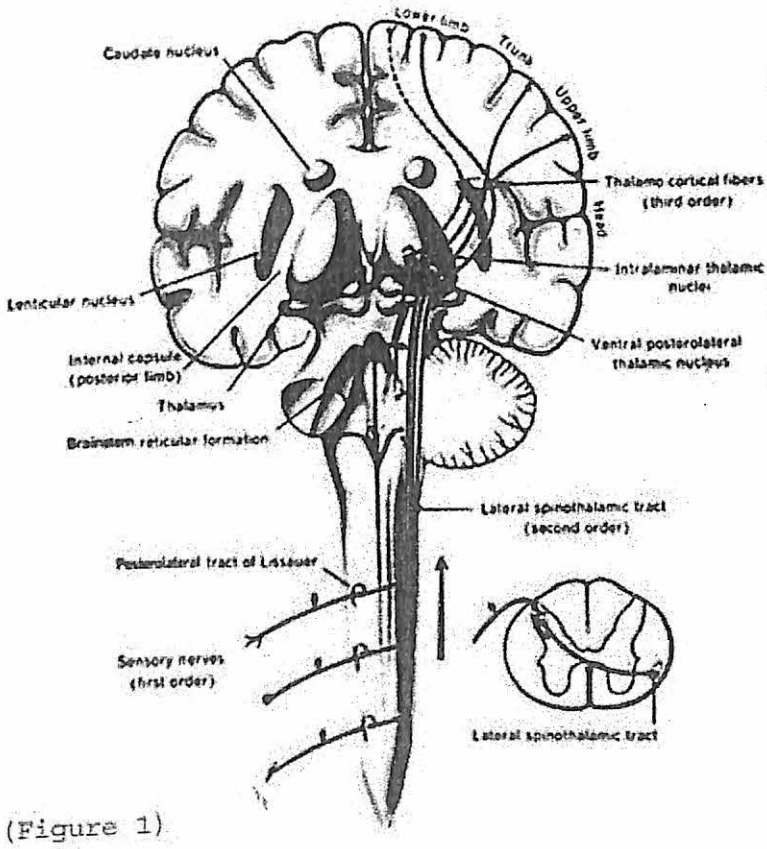


The reflex pathway underlying referred pain. Some of the pain afferents from the viscera synapse in the spinal cord with the same neurons of the lateral spinothalamic tract on which cutaneous pain afferents terminate.

One explanation of referred pain is on the basis of spread of excitation from neurons from the viscera to neurons that receive pain impulses from the skin. Consequently, both visceral and skin sensations travel along spinothalamic pathways for skin. Subsequently interpretation by the cortex that the pain originated from the skin.

SISTEM SARAF PUSAT

Modul 17 The spinothalamic pathways.



(Figure 1)

Pain and temperature pathway. The direct spinothalamic pathway comprises :

(1) Neurons of the first order with cell bodies in the spinal ganglia.

(2) Neurons of the second order with cell bodies in the posterior horns and, with axons that decussate and ascend as the lateral spinothalamic tract to the thalamus and

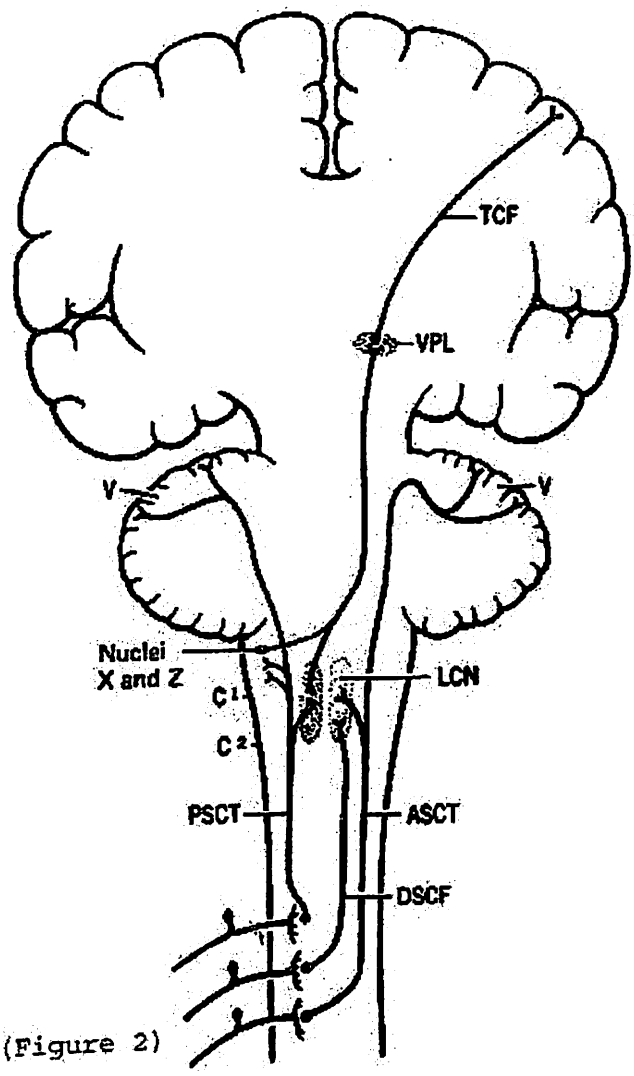
(3) Neurons of the third order with cell bodies in the thalamus and with axons that project to the cerebral cortex.

The indirect spinothalamic pathway is composed of :

(1)Neuron of the first order.

(2)Sequence of several neurons of the second order that project through the brainstem reticular formation to the intralaminar thalmic nuclei and

(3)Neurons of the third order with cell bodies in the thalamus and with axons that project to the cerebral cortex (the latter is represented in broken line because course is not precisely known). Note interneurons in the relay nuclei.



(Figure 2)

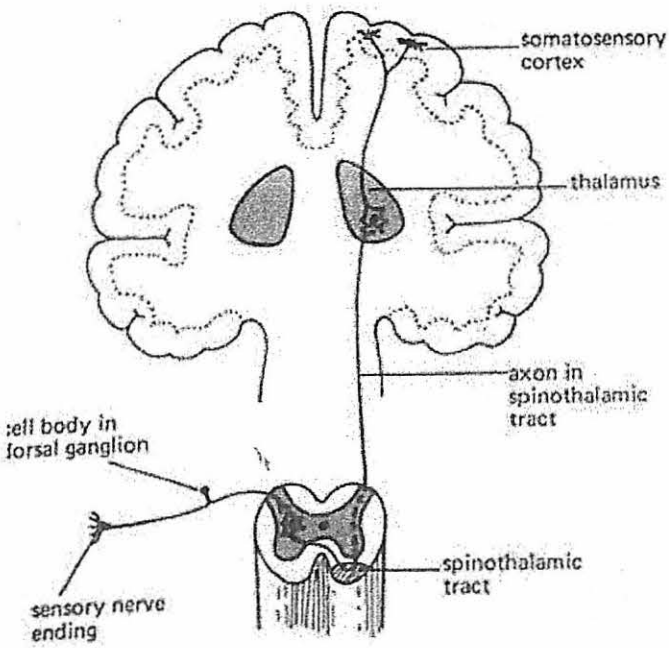
The ascending, tracts of the spinothalamic system consist of
(1) The lateral spinothalamic tract (neospinothalamic tract, NSTT).
(2)The spinoreticulothalamic pathway (paleospinothalamic tract, PSTT), and
(3) The anterior spinothalamic tract.

Interneurons (i) stand between central terminations of the peripheral nerves (PN) and the ascending tracts, including the, spinoreticular tract (SRT) and lateral spinothalamic tract.

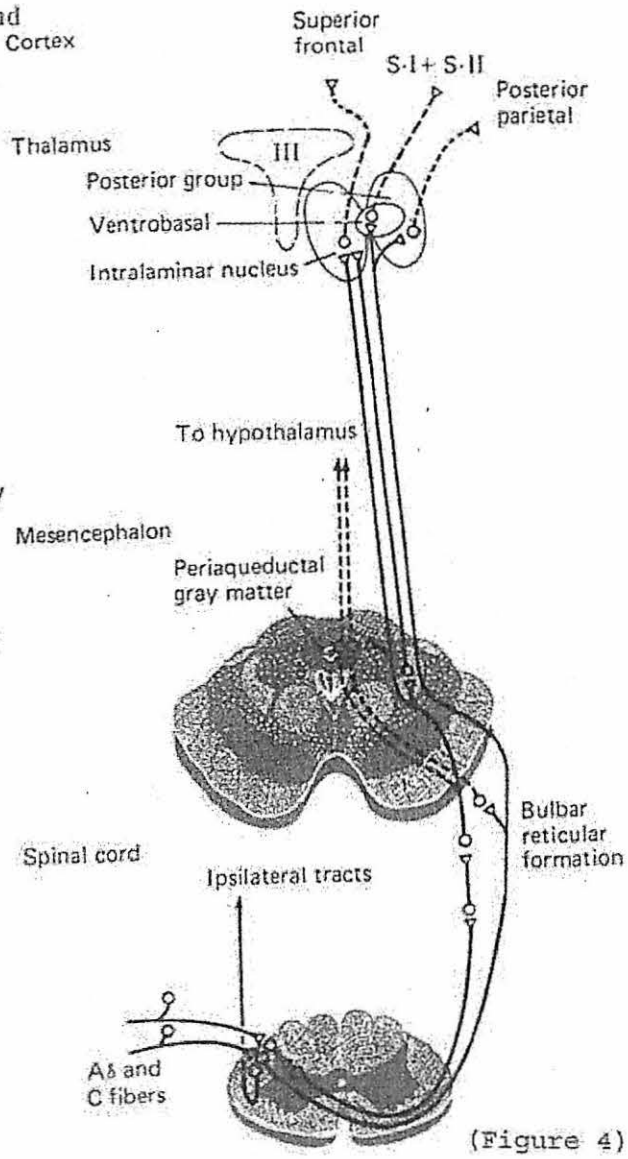
(The paleospinothalamic tract includes interneurons located in the brainstem reticular formation (RF). The thalamic nuclei integrated within the spinothalamic pathways, include the intralaminar nuclei (IL), parafascicular nucleus (PF), posterior thalamic region (PTR) and ventral posterior lateral nucleus (VPL). Influences from the thalamus are projected by thalamocortical fibers (TCF).

Figure 3: This slow pathway for pain, temperature, and sexual sensations involves sensory fibers that synapse directly with dorsal horn cells in the spinal cord, then cross over and travel up the spinothalamic tracts to the thalamus. From the thalamus, axon ascend to synapse in different regions of the sensory cortex.

Figure 4: The anterolateral system of spinothalamic, spinoreticular and spinotectal fibers, which convey information about pain to broad regions of the brain stem and diencephalon.



(Figure 3)



(Figure 4)

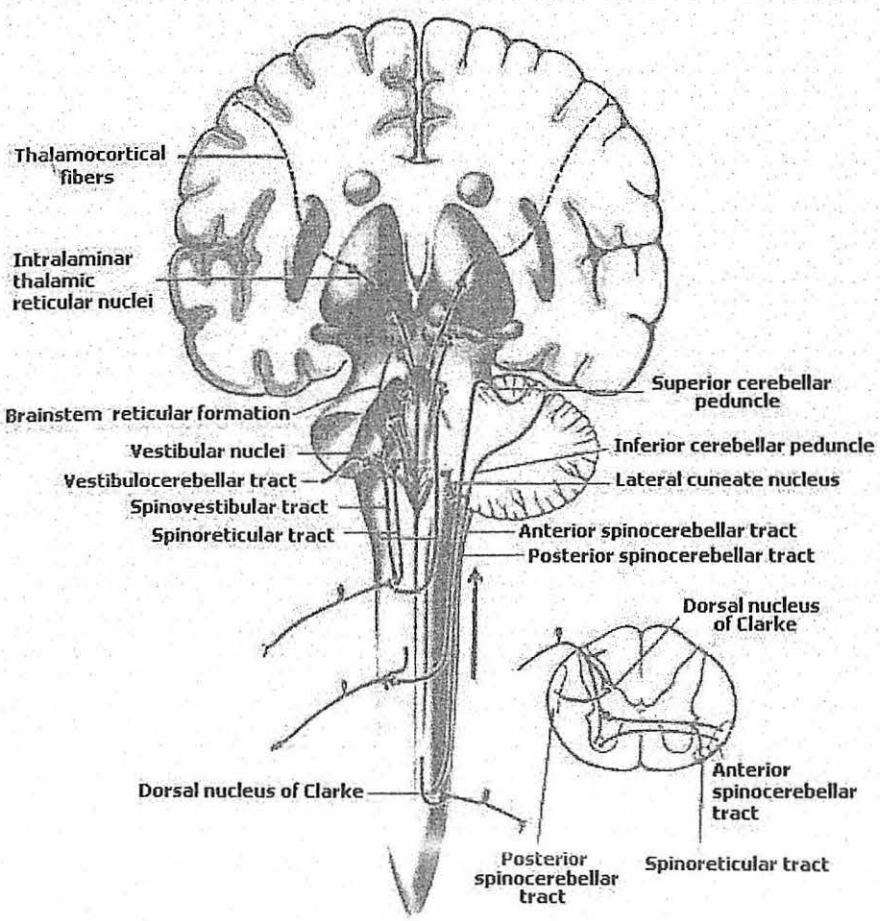
SISTEM SARAF PUSAT

Modul 18 Spinocerebellar pathways.

Receptors in the extremities and body wall, especially those in the muscles and tendons, are continually monitoring the immediate status or muscle contraction and the momentary degree of tension within the tendon. The resulting "unconscious" proprioceptive information from the muscles tendons and joints is projected to the cerebellum via four pathways whose fibers terminate as mossy fibers in the cerebre llar cortex.

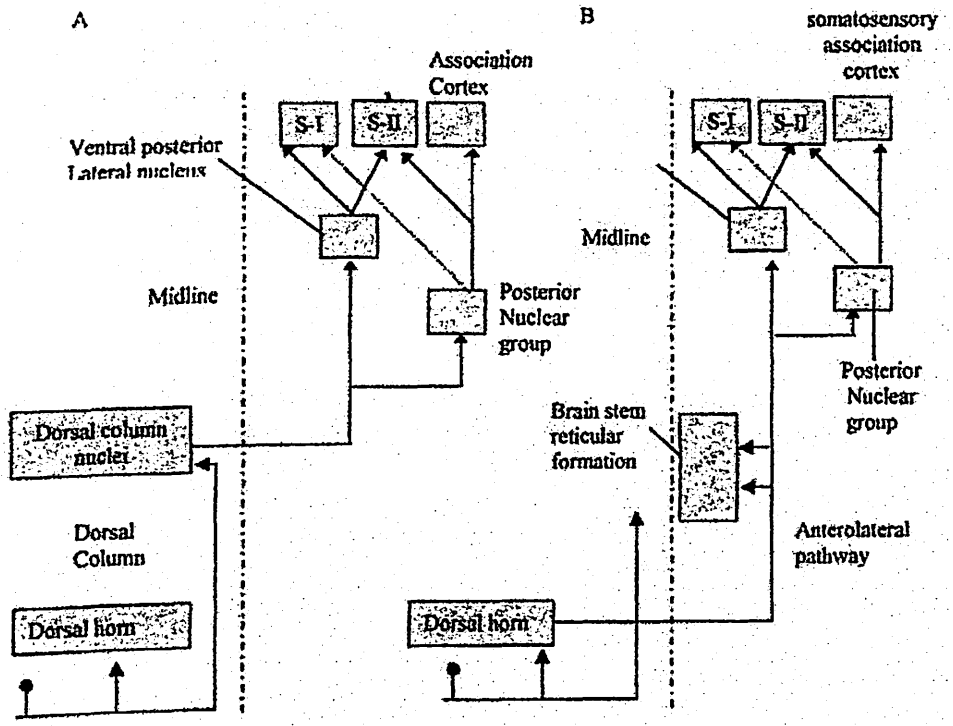
Same input to the cerebellum comes from exteroceptive receptors in the skin. The posterior and anterior spinocerebellar tract convey these influences from the lower extremities and the caudal half of the body, while the cuneocerebellar tract and the rostral spinocerebellar tract convey influences from the upper extremities and upper half of the body.

The spinocerebellar tracts include (1) the uncrossed posterior spinocerebellar tract that passes through the inferior cerebellar peduncle and terminates in the palecerebellum, and (2) the crossed anterior spinocerebellar tract that passes through the superior cerebellar peduncle and terminates in the paleocerebellum. The spinovestibular tract includes fibers that terminate in the vestibular nuclei. The spinoreticular tract is composed of crossed and uncrossed fibers that terminate in some brainstem reticular nuclei.



SISTEM SARAF PUSAT

Modul 19 Somatic sensation- an overall view.



Afferent information from the body is relayed to the cerebral cortex by two major ascending system: The dorsal column-medial lemniscal system and the anterolateral pathway (see fig A,B). These systems, which serve different functions, converge in the thalamus , (see table below). The ventral posterior lateral nucleus and the posterior lateral nucleus and the posterior nuclear group mediate the projection to the somatic sensory corties, which include S-I , S-II and the somatic sensory association area. In the somatic sensory corties, afferent information further processed somatic perception.

TABLE Major Ascending Somatic Sensory Systems

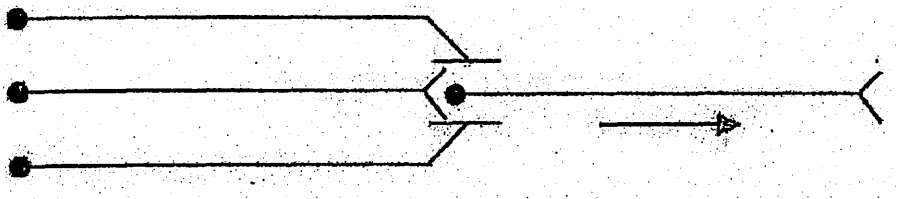
Ascending system	Submodalities	Location in spinal cord	Level of decussation	Brain stem terminations	Cerebral terminations
Dorsal column Medial Lemniscus	Touch–pressure Vibration Position sense–kinesthesia	Dorsal column	Medulla	Ventral posterior lateral nucleus and Posterior nuclear group of thalamus	Primary and secondary somatic sensory cortices and somatic sensory association area
Anterolateral	Pain Thermal Crude touch	Anterolateral column	Spinal cord	Brain stem reticular formation Ventral posterior lateral nucleus and posterior nuclear group of thalamus	Primary and secondary somatic sensory cortices and somatic sensory association area

SISTEM SARAF PUSAT

Modul 20

Principle of convergence.

Each neuron of the central nervous system is excited and inhibited by the synaptic activity of many other neurons, on its dendrites and cell body. This receptive portion of the neuron is the focus for the convergence of the activity of, tn many cases, literally thousands of other neurons. The intergrated reactivity op the excitatory and inhibitoty synapses on the receptor portion of the neuron may result in the generation op an action potential in the axon of the neuron.



Principle of convergence. several presynaptic neurons synapse with one postsynaptic neuron.

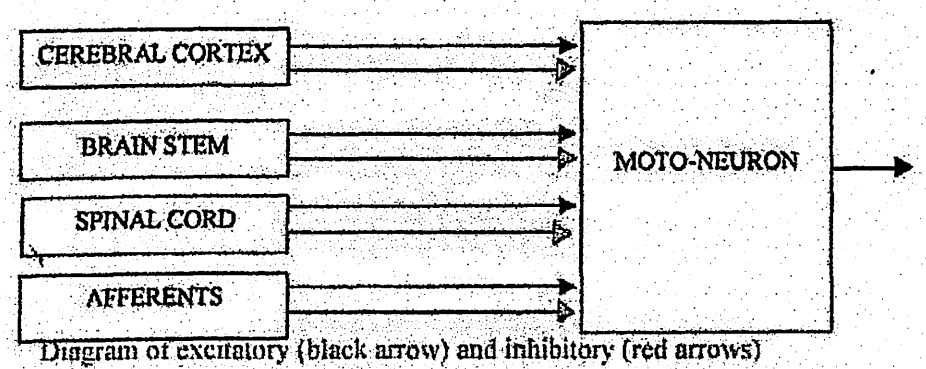
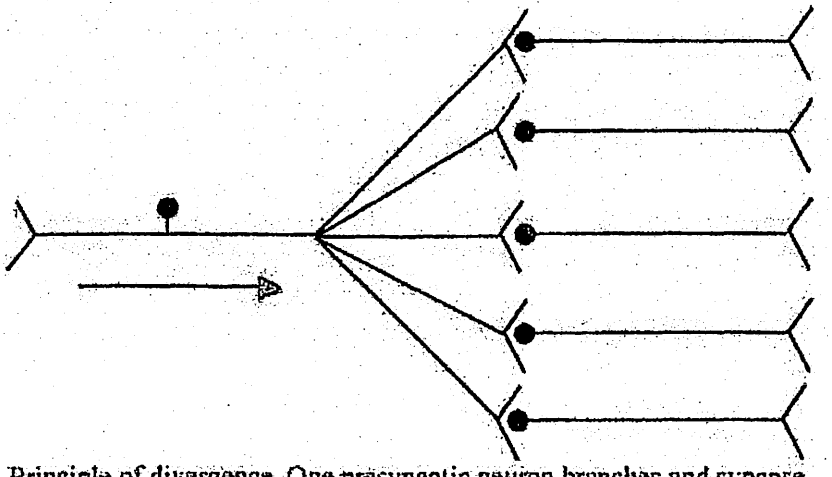


Diagram of excitatory (black arrow) and inhibitory (red arrows) affe rents converging onto a motoneuron. The motoneuron forms the final common pathway of all motor reflexes.

Principle of divergence.

Each neuron synapses with many other neurons. An afferent nerve cell that is stimulated by peripheral receptors terminates in the central nervous system by arboriz ing into many branches, each branch having synapt ic contact with tme receptive port ion of many other neurons. The oppurtunity of a neuron to excite or to inhibit numerous, other neurons by the divergence of its axonal terminal branches is a fundamental principle underlying the activity of the central nervous system.



Principle of divergence. One presynaptic neuron branches and synapse with several postsynaptic neuron.

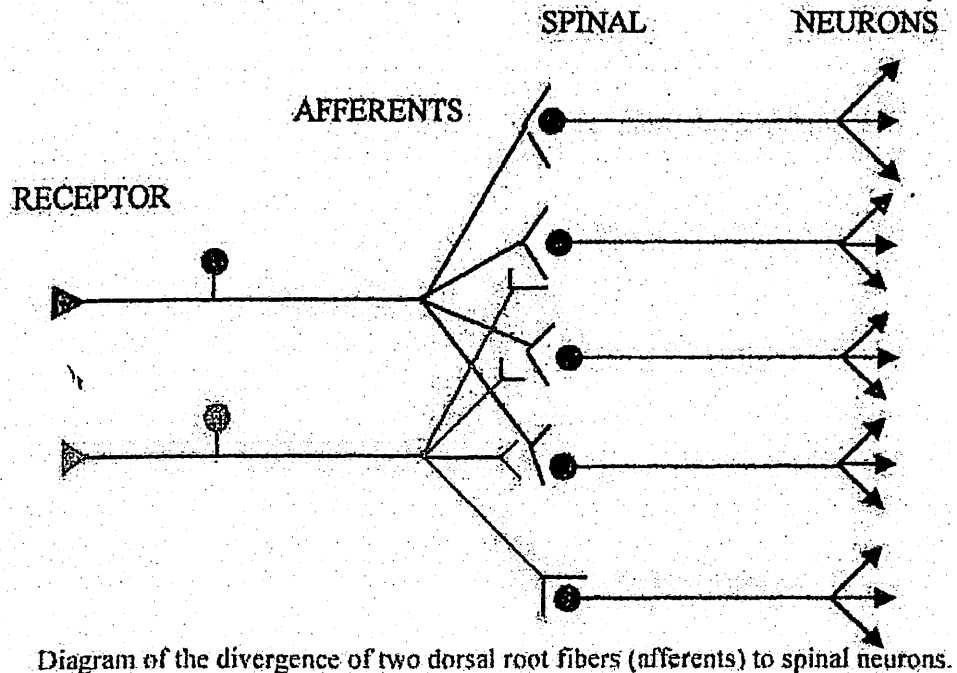


Diagram of the divergence of two dorsal root fibers (afferents) to spinal neurons. The axons of these neurons in turn branch into a large number op collaterals.

Facilitation.

Facilitation (literally, "making easy") is the phenomenon wherein a normally subliminal (subthreshold) stimulus form a presynaptic neuron "primes" a postsynaptic neuron so that another subliminal stimulus can evoke a dischar of the postsynaptic neuron. In brief, the first stimulus has facilitated the postsynaptic neuron.

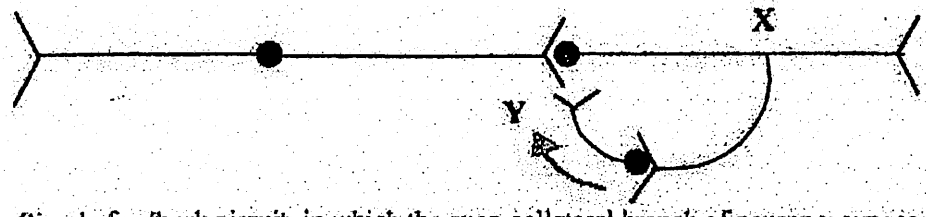
The start of a sprint race offers an anology. The starter's instruction of "get set" is the stimulus that "facilitates" the sprinters for the final stimulus of "go" many interactions of the central nervous system involve facilitation.

Recruitment.

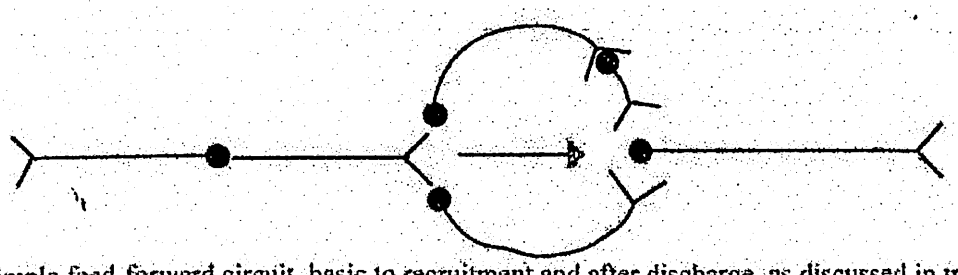
Recruitment (the recruiting of neurons) is a neurophysiologic phenomenan in which a response (action potential) is obtained only after a rapid succession of afferent stimuli is delivered. The complex of multiple chains intercalated between the stimulated sites (afferent neuron) and the responsive sites (efferent neurons) is critical to the phenomenan of recruitment.

Recruitment is a form of facilitation primary utilizing closed multineuronal circuits. The following is a simplified explaination of this complex activity. The impulses resulting from the first afferent volleys travel via short circuits (an open loop) to the motor pool. These initial volleys also activate closed multiple-chain circuits, but the resulting impulses arrive at the motor pool too late to summate with those traveling over the short circuits. However, these initial volleys transmitted through the closed multiple circuits arrive at the same time as the subsequent volleys transmitted with the short circuits. The resulting summation facilitates a response from the motor neurons on the motor pool. Recruitment is a form of facilitation obtained by repetitive stimulation. The complexities of the circuits capable of eliciting recruitment stagger the imagination.

(A volley - is the firing of a group of neurons)



Simple feedback circuit, in which the axon collateral branch of neuron x synapses with interneuron y, which, in turn, synapses with neuron x.

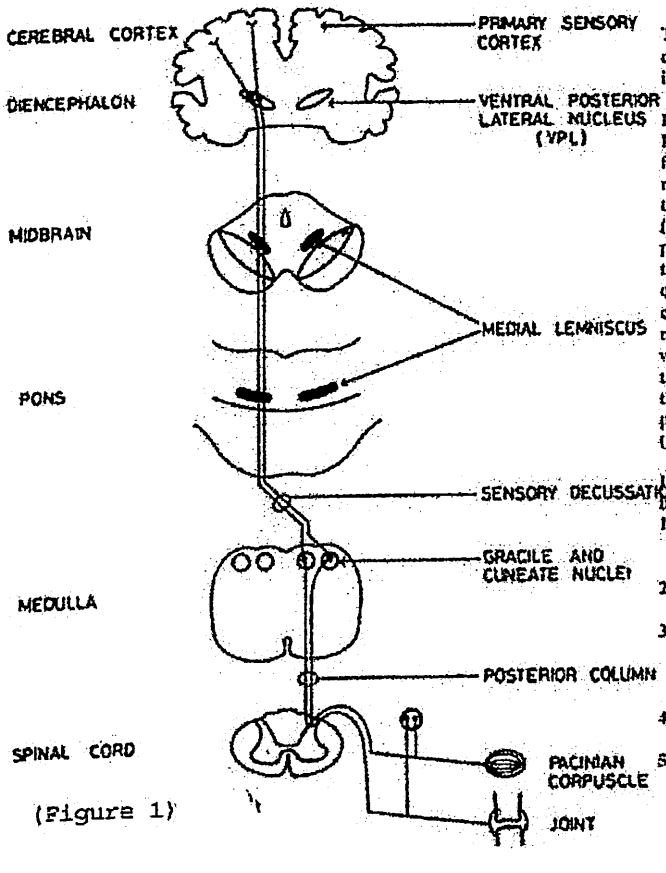


Simple feed-forward circuit, basic to recruitment and after discharge, as discussed in treat.

The response of same intact sense organs is more complicated because many sensory fibers emerge from them. Therefore, strong stimulation increases not only the frequency of discharge of individual afferent fibers but also the number of activated sensory units. The latter are more numerous when a strong stimulus is applied than a weak one is used. The process of bringing in several sensory fibers during stimulation has been aptly and picturesquely described as recruitment of sensory units, because it is something like recruiting soldiers for a given military task.

SISTEM SARAF PUSAT

Modul 21 Pathway for conscious proprioception.



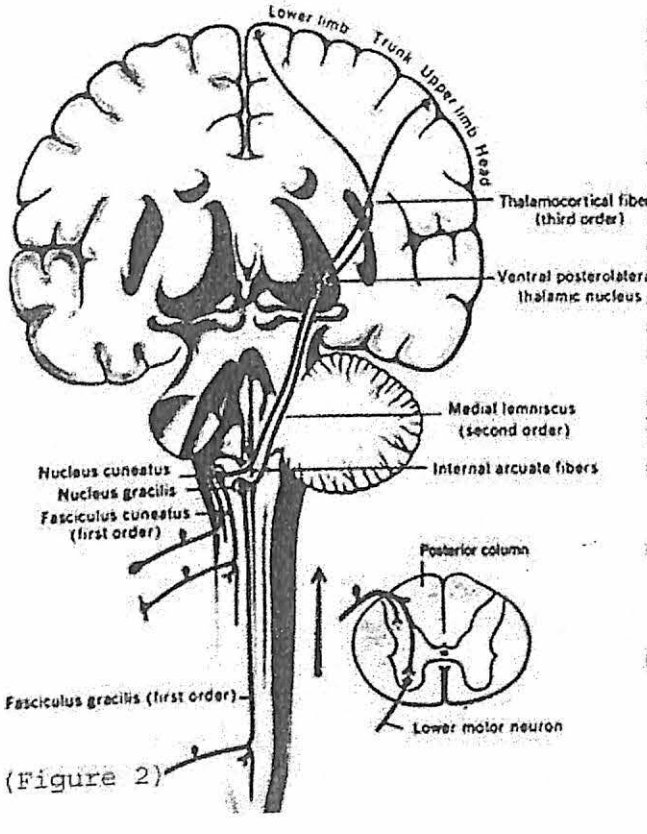
(Figure 1)

The pathway for kinesthesia (position and vibration sense) and discriminative touch (well-localized touch and two-point discrimination) is the posterior column-medial lemniscus system.

Receptors for this system are those in joint capsules and pacinian corpuscles. Impulses arising in the receptors travel via the thicly myelinated, large nerve fibers that enter the spinal cord as the medial divisinn of the posterior (dorsal) root and occupy the posterior funiculus of the spinal cord. Those arising below the sixth thoracic spinal segment form the medial part of the posterior funiculus (gracile tract) those arising above the sixth thoracic segment from the lateral part of the posterior funiculus (cuneate tract) fibers in the gracile and cuneate tracts project upon neurons in the posterior column nuclei of the medulla oblongata (nuclei gracilis and cuneatus). Axon of neurons in the posterior column nuclei (second order neurons) decussate in the tegmentum of the medulla oblongata (internal arcuate fibers) to form the medial lemniscus, which ascends throughout the medulla oblongata, pons and midbrain to terminate upon neurons of the ventral posterolateral nucleus (vpl) of the thalamus. Axons of neurons in this thalamic nucleus (third order neurons) project upon the terminal station of this pathway in the somasthetic (primary sensory) cortex of the parietal lobe.

Lesions of the posterior column-medial system are manifested clinically by the following signs.

1. Inability to identify position of the limb in space with eyes closed. Such patients abe unable to tell whether a joint is in a position of flexion or extension.
2. Inability to identify objects placed in the hands, such as keys or coins, from their shape, size and texture with eyes closed.
3. Loss of two-point discrimination. Such patients are unable to recognize two simultaneously applied stimuli to the brain when the two stimuli are separated by the minimal necessary distance for their proper identification as two stimuli.
4. Inability to perceive vibration when a vibrating tuning fork is applied to a bony prominence.
5. Inability to maintain steady standing posture when eyes are closed and feet are placed close together (romberg test). Such patients begin to sway and may fall when they close their eyes, which eliminates visual compensation.



(Figure 2)

Posterior column - medial lemniscal pathway.

This major system conveys information from mechanoreceptors in the skin, muscles tendons, and joints. (Their activity is subjectively perceived in basically three forms of sensibility: touch-pressure, kinesthesia (position sense) and vibratory sense. Touch-pressure is the sense resulting from deformation of the skin; kinesthesia includes the sense of position and movement of joints (angle-movement detectors). Vibratory sensed from meissner's corpuscles are called flutter-vibrations (30-40 hz), while recognition of those from deep structijres such as joints and bone is called the vibration sense. Vibratory sense (roughly 400 hz from pacinian corpuscles) is actually a sensing of rnpid, successive stimuli of tactile sense.)

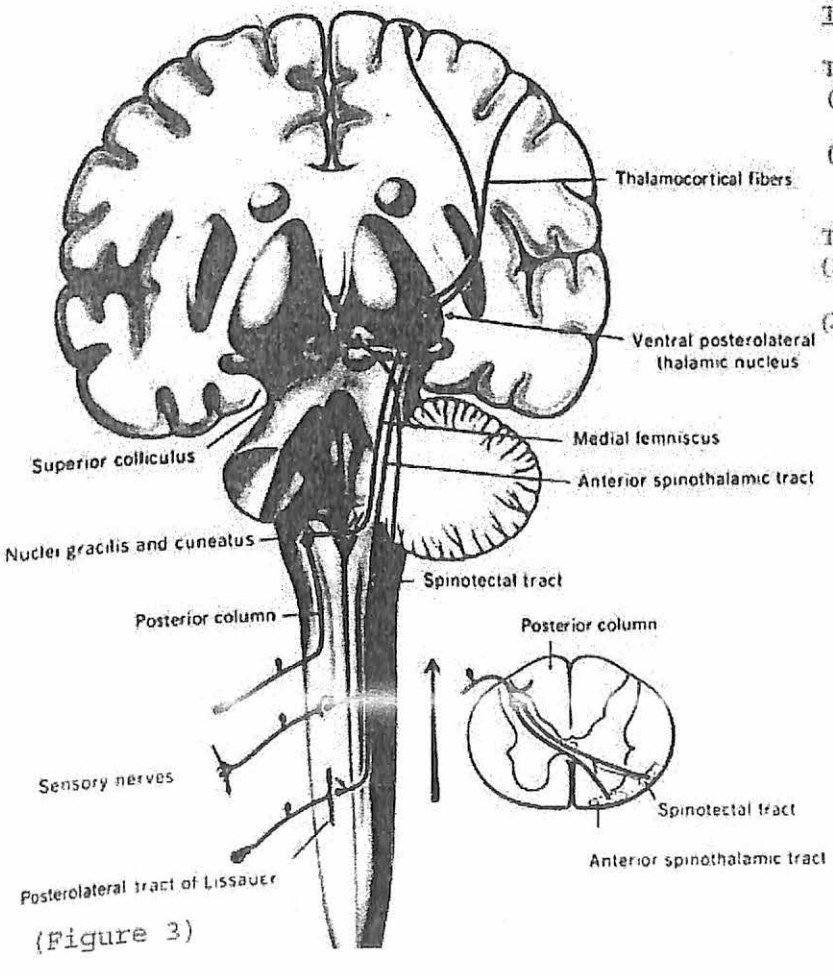
The posterior column-medial lemniscal

Pathway - is composed of:

(1) Neurons of the first order with cell bodies in the spinal ganglian and with axons that ascend in the posterior column to the nuclei gracilis and cuneatus;

(2) Neurons of the second order with cell bodies in the nuclei gracilis and cuneatus and with axons that decussate as the internal arcuate fibers in the lower medulla and ascend in the medial lemniscus to the thalamus; and

(3) Neurons of the third order with cell bodies in the thalamus and with axons that project to the cerebral cortex (post central gyrus). Collateral branches of the neuron of the first order pass to the posterior horn the anterior horn and the posterior column; the last are descending association fibers.



(Figure 3)

Touch pathways and spinotectal tract

The touch pathways include :

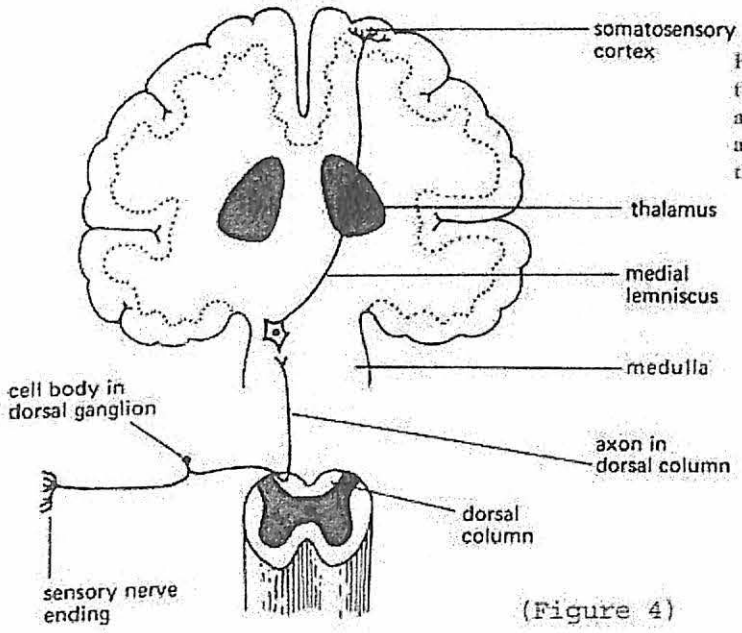
(1) the posterior column medical lemniscusthalamocortical pathway and

(2) the anterior spinothalamic tract-thalamocortical pathway.

The spinotectal pathway is composed of :

(1) neurons of the first order with cell bodies in the spinal ganglia, and

(2) neurons of the second order with cell bodies in the posterior horn with axons that decussate and ascend as the spinotectal tract to the superior colliculus.

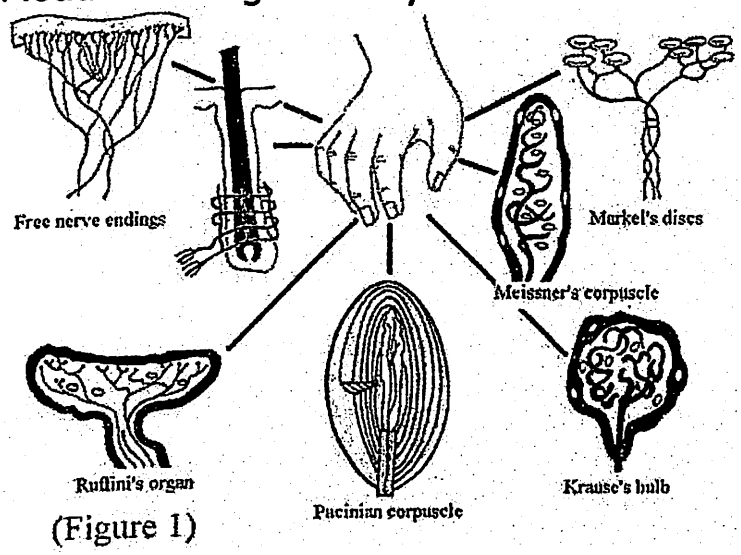


Fast pathway for touch and pressure sensations from the skin through the dorsal column of the spinal cord and the medial lemniscus, which is between the medulla and the thalamus. From the thalamus, axons ascend to the sensory cortex.

(Figure 4)

SISTEM SARAF PUSAT

Modul 22 Integumentary sensorria.



(Figure 1)

Pacinian corpuscle : deep pressure

A. Largest, most widely distributed encapsulated receptors; detects deep pressure. .
B. Concentric layers of connective tissue (separated by very thin cell layers) enclosing terminal nerve fiber within protoplasmic core.
C. Deep in dermis ; prominent in hand and foot, peritoneum, mesenteries, genitalia and walls of many viscera ; also in ligaments and joint capsules.

Rufini's organ : heat

A. Elongated, encapsulated receptors , nerve fibers branch and terminate within central syncytium , enclosed by connective tissue capsule, sense of heat.
B. Deep in dermis and subdermis.
C. Sensitive to temperatures exceeding that of normal body, particularly sensitive to temperature differentials.
D. Number varies greatly in different parts of body.
E. Generally much less numerous than cold receptors in any given area.

Krause's bulb :cold

A. Spherical encapsulated receptors , nerve fiber branches with convolutions in central syncytium, enclosed by connective tissue capsule, sense of cold.
B. Deep in dermis
C. Sensitive to temperature, lower than body temperature , particularly sensitive to temperature differentials.

Complex sensation

Intergumentary receptors are rarely stimulated singly;

1. Usually nature of stimulus is such that several different types of receptors are stimulated stimultaneously
2. Cerebral cortical associations produce broad spectrum of complex sensations from various combination of simple, basic sensations.

Free nerve ending ; pain

A. Simpleast least specialized of receptors, numerous branches with bare terminal ending, associated with pain.
B. Most widely distributed nerve ending , most numerous in skin but also in mucous membranes and muscles of visceral organs.
C. Extend into germinative layer of epidermis (see dyna-vue th 4.01)

Hair ending plexus, hair movement

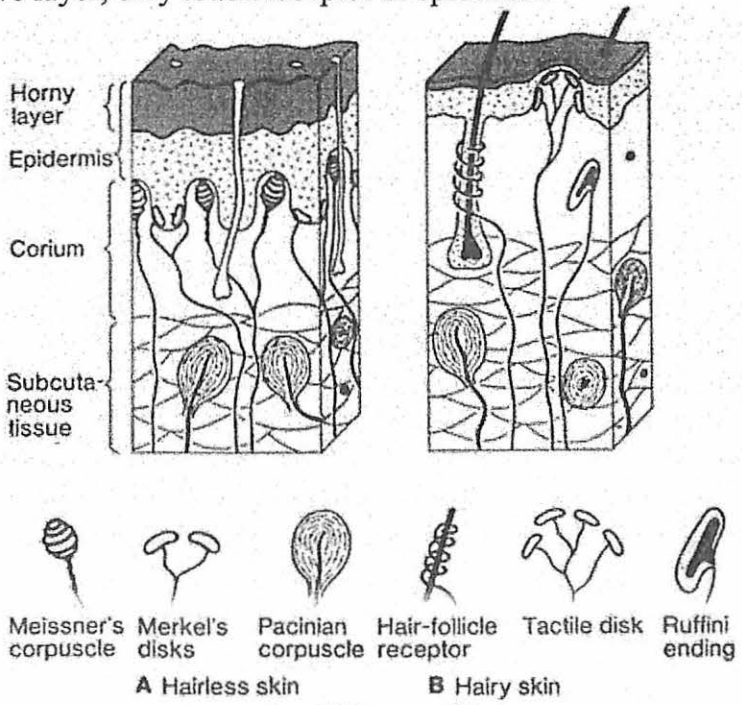
A. Neural Fiber Plexus, associated with hair root (see dyna –vue Th 4.01).
B. Movement of hair shaft stimulates awareness of hair movement

Meissner's corpuscle ,touch - light pressure.

A. Encapsulated receptors in dermal papillae, associated with sense of touch and light pressure.
B. Corpuscle consists of vertically-layered cells interspersed with nerve fibers, and enclosed by thin connective tissue sheath.
C. From 1 to 4 nerve fibers per corpuscle
D. Mainly in hairless portion of skin , especially in fingers, toes, hands and feet, also in lips and tip of tongue
E. Sense of touch and pressure very similar , but touch is the more localized in time and space (or position)

Merkel's discs : touch

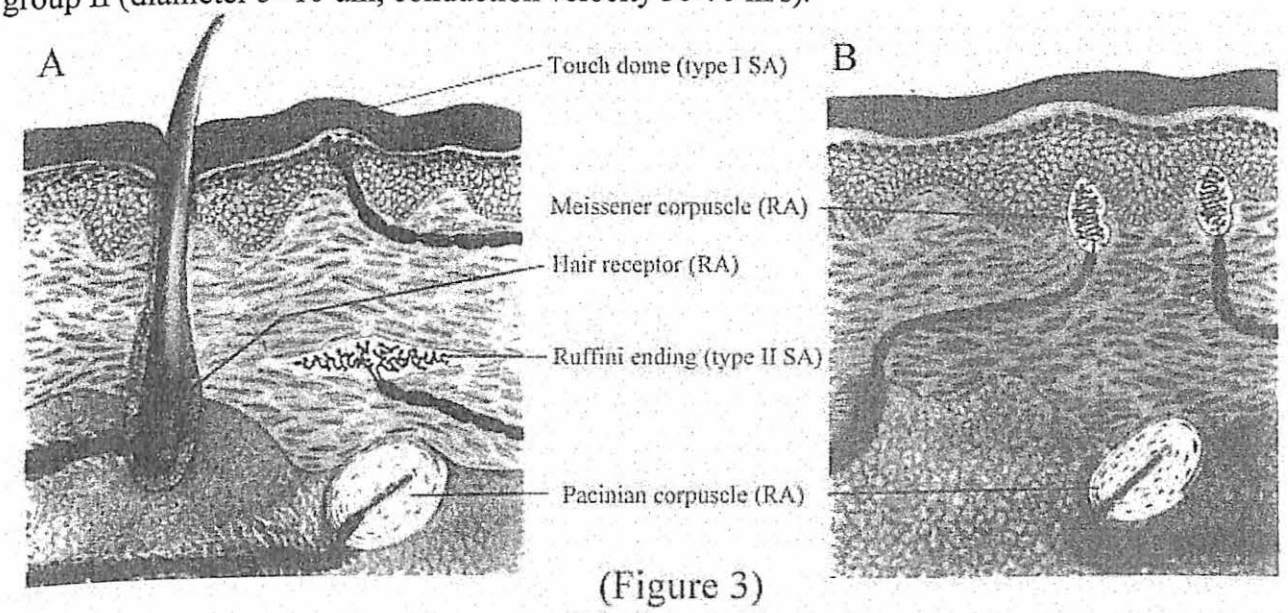
A. Multibranched nerve fiber with terminal discs on each branch, with single epidermal cell above each discs.
B. Single nerve fiber supplies many discs.
C. Pressure on epithelial cell stimulates discs below, interpreted as touch
D. Deep in germinative layer, only touch receptor in epidermis.



(Figure 2)

Schematic drawing of the structure and position of mechanoreceptors in hairless (A) and hairy (B) skin.

Pacinian corpuscles :-
Acceleration detector also called pc receptors also found in tendons and fascia of the muscles in the periosteum in the joints capsules and in the mesenteries.
Meissner corpuscles :-
Also called meissner's tactile corpuscles moderately rapidly adapting in hairless skin.
Hair follicles receptor :-
Velocity detectors in hairy skin.
Merkel's disks :-
Also called merkel cell complexes slowly adapting intensity detectors in hairless skin located in small groups in the lower most layers of the epidermis. Merkel cell complexes are also found in hairy skin, but lie in special dome corpuscles elevated above the surrounding skin surface, also called pinkus- iggotactile corpuscles.
Ruffini corpuscles:-
Slowly adapting intradermal receptors found in the deeper layers of the dermis of hairy and glabrous skin (hairless skin) All the mechonoreceptors are supplied by myelinated afferent nerve of group II (diameter 5- 10 um, conduction velocity 30-70 m/s).

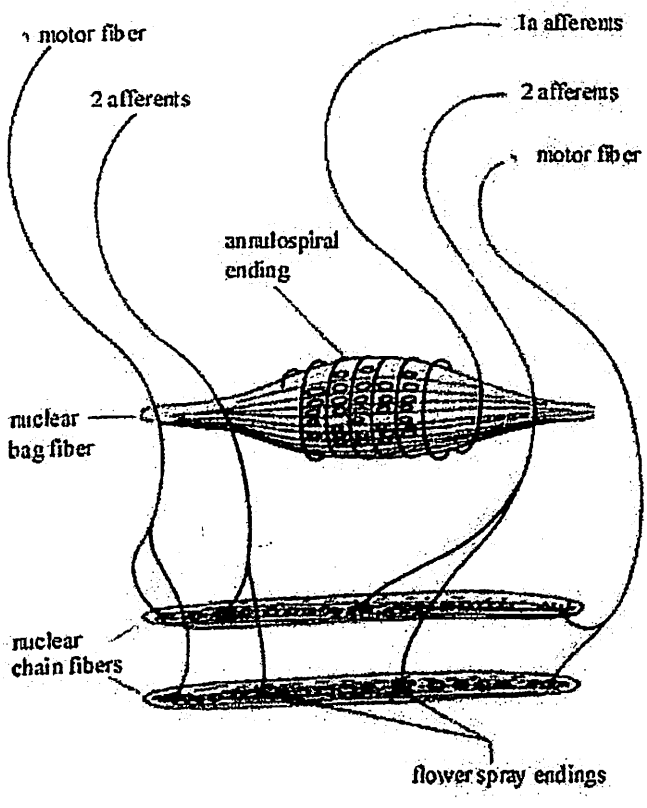


(Figure 3)

Morphology of mechanoreceptors of the hairy and glabrous skin.
A) Hairy skin, Rapidly Adapting (RA) receptors, the pacinian corpuscle, is located in the border region between the dermal and subcutaneous tissue. Other rapidly adapting receptors have fibers that are connected by free nerve endings at the based of the hair follides. The type I slowly adapting (Sa) receptors correspond to the touch dome. The type II slowly adapting receptors are located in the ruffini end-organ.
B) Glabrous skin (hairless skin).
Meissner corpuscles, which are rapidly adapting receptors, are located in dermal papillae. Pacinian corpuscles are located beneath glabrous skin as well as hairy skin.

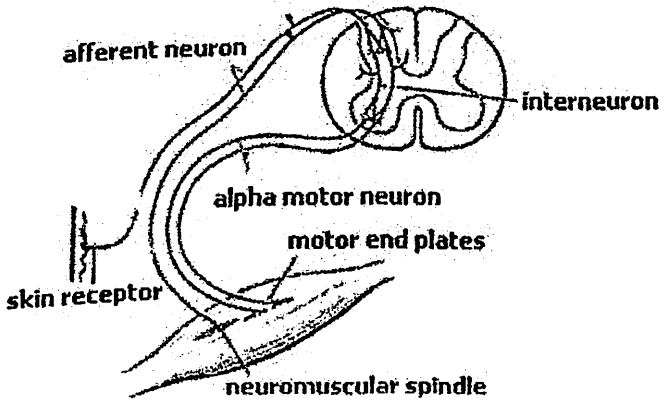
myelinated afferents) fibers. These fras activate interneuronal circuits with widespread polysynaptic connections within the spinal cord. The eventual effects of this spinal activity are (1) ixcitation of the alpha motor neurons to flexor muscles and (2) inhibition of the alpha motor neurons to the antagonistic extensor muscles.

The flexor reflex loop (see figure). This three-neuron disynaptic ipsilateral, intersegmental reflex is composed of a sensory receptor in the skin, afferent neuron, spinal intersegmental neurons, alpha motor neurons and voluntary muscles. This reflex can be facilitated by the "secondary sensory ending" (flower spray ending) of neuromuscular spindle, afferent neuron, spinal interneurons, alpha motor neurons and voluntary muscles.



The muscle spindle as an informant.

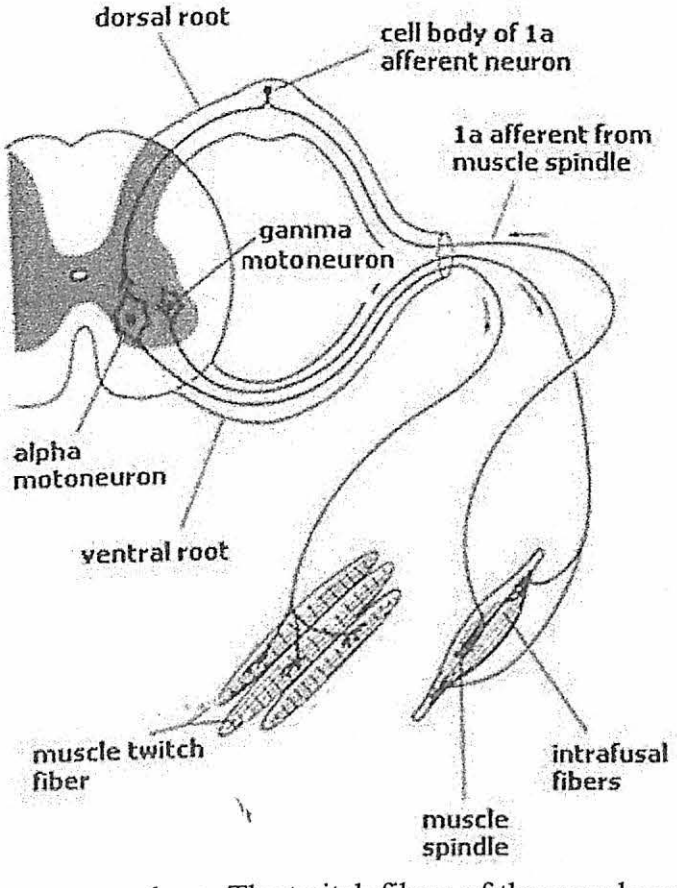
Muscle spindles are capable of relaying for more information than merely changes in length. Because of represence of another set of sensory nerve endings within the spindle, the secondary. (2) fibers, the spindles can also convey information concerning the rate at which a muscle is changing its length. The fibers form "flower-spray" endings around the ends of long, thin, intrafusal fibers, known as the nuclear chain fibers. The annulospiml endings of the la fibers wrap around the central region of a different type of intrafusal fiber, the nuclear bag fiber. There are several la and 2 nerves. (in addition of course, to the gamma motor fibers a simplified version of muscle spindle structure is shown.



Flexor reflex responses (protective or withdrawal reflexes).

The flexor responses are initiated by a heterogeneous group of afferents called fle or reflex afferents (fra' s) with receptors ending in the skin, muscle, joints and viscera. They include a delta and c pain fibers, group ii (secondary spindle afferents fibers and group iii and iv (free nerve endings of

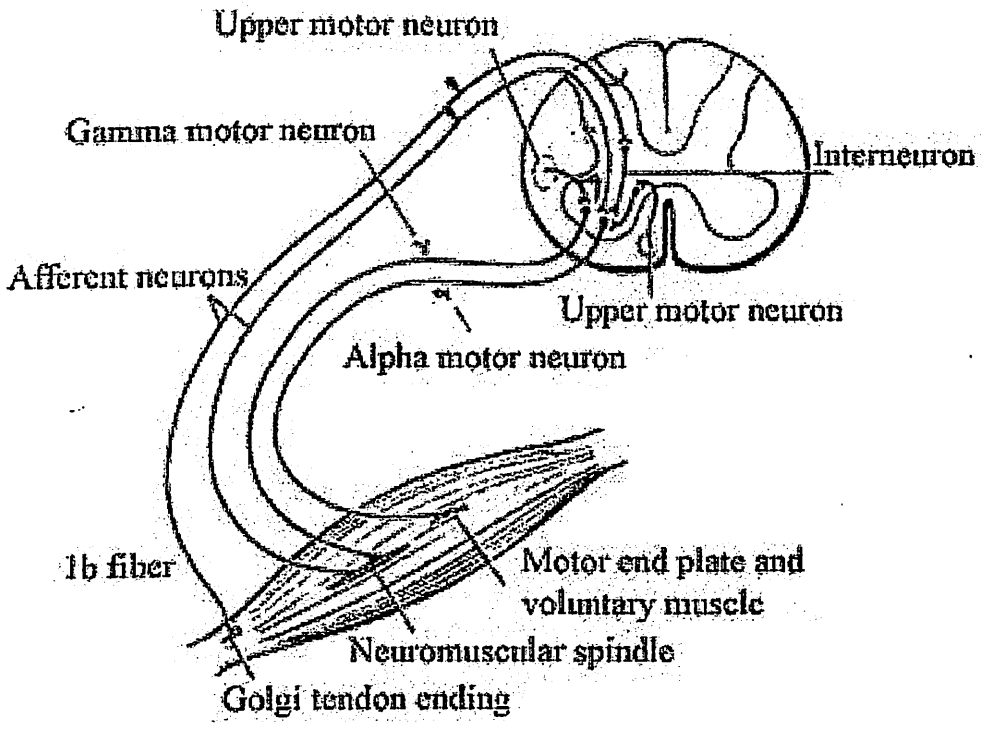
2) To preset the muscle to contract to lift a load and to make adjustments in contraction rate and force during the contraction.



The gamma loop. The twitch fibers of the muscle are innervated by the axons of the alpha motoneurons. The intrafusal fibers of the muscle spindle are innervated by the gamma motoneurons. The muscle spindle also innervated by la sensory fibers that synapse directly on the alpha motoneurons.

ISTEM SARAF PUSAT

Modul 23 Gamma reflex loop.



Gamma reflex loop.

As a muscle contracts, the neuromuscular spindles within the muscle become passively shorter. With this shortening there is a cancomitant reduction in the rate of spindle firing via ia afferent neurons to the alpha motor pool. With this rati reduction, the system is not able to maintain the continuous contraction of any muscle mass. Continous contraction of a muscle mass results when the muscle fibers are in various degrees of contraction in response to asynchronous volleys of alpha motor neurons (a volley is the firing of a group of neurons). This continous contraction is dependent upon the gamma reflex loop.

The figure shows gamma reflex loop and the golgi tendon endings. The gamma loop comprises (i) gamma motor neuron
(ii) neuromuscular spindle
(iii) afferent neuron (i a fiber),
(iv) alpha motor neuron, and
(v) voluntary muscle.

The upper motor neuron can facilitate the gamma motor neurons. The golgi tendon endings are involved in the loop composed of golgi tendon endings, afferent neuron, spinal intrasegmental neuron, and alpha motor neuron.

The gamma loop is the pathway that involves the alpha and the gamma neurons and the ia sensory fibers that connect them. the gamma loop is essential:

1) To set the general level of muscle tone precisely, and so to control posture.

SISTEM SARAF PUSAT

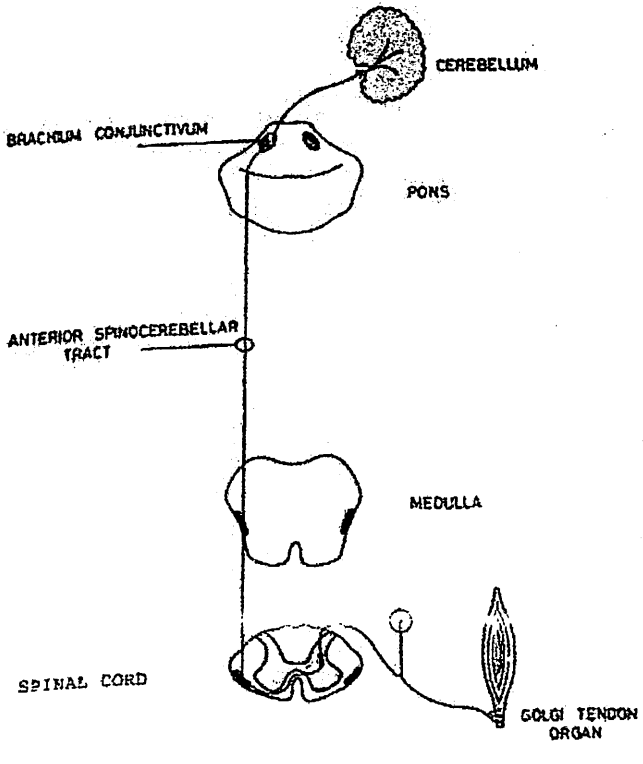
Modul 24 Pathways for nonconscious proprioception.

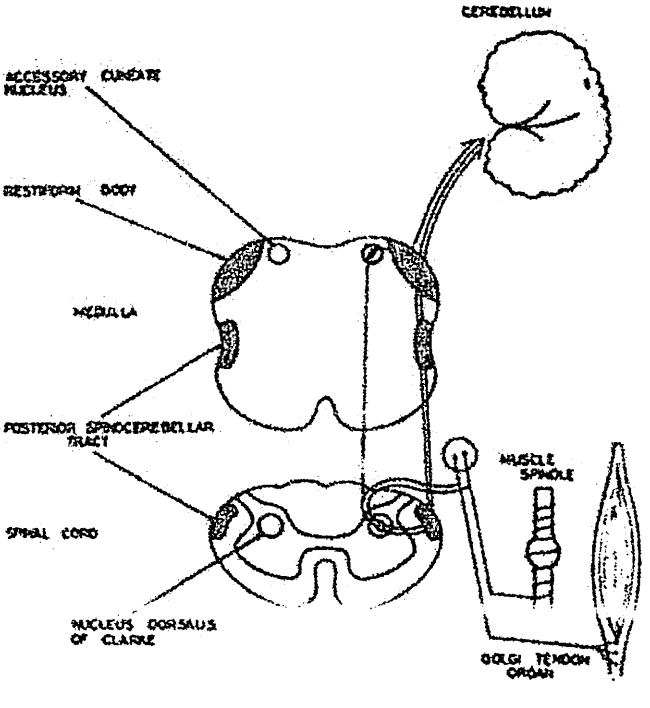
Nonconscious proprioception is mediated via the two spinocerebellar tracts these are the posterior (dorsal) and anterior (ventral).

The posterior spinocerebellar tract conveys impulses from the muscle spindle and golgi tendon organ. Such impulses travel via groups of ia, ib, and ii nerve fibers, enter the spinal cord in the medial, thickly myelinated, large diameter fiber portion of the posterior root, and project upon the ipsilateral nucleus dorsalis of clarke. Axons of neurors in this nucleus (second order neurons) form the posterior spinocerebellar tract, which ascends in the lateral funiculus of the spinal cord and the medulla oblongata to reach the cerebellum via the inferior cerebellar peduncle (restiform body).

The anterior spinocerebellar tract conveys impulses from the golgi tendon organ. Incoming fibers project upon neurons in the posterior horn of the spinal cord (laminae v to vii). Axons of neurons in these laminae decussate to the contralateral lateral fun iculus to form the anterior spinocerebellar tract, which ascends throughout the spinal cord., medulla oblongata, puns, and mid-brain, loops backward to join the superior cerebellar peduncle (brachium conjunctivum), and enters the cerebellum. The spinocerebellar pathways convey to the cerebellum information about activity of muscles and progress of motion for coordination of movement.

Lesion of the spinocerebellar pathways (as occur in hereditary spinocerebellar degeneration) result in incoordinate movement. such patients tend to walk with a wide base, stagger, and frequently fall.





SISTEM SARAF PUSAT

Modul 25 Cerebral circulation.

The internal carotid and vertebral arteries provide the total blood supply which is relatively constant during exercise, postural / gravitational changes, mental acuity and sleep regional blood flow may change in response to activity in parts of the brain.

CEREBRAL CIRCULATION

- is 750ml/min (measured by N_2O method-fick principle)
- SUPPLIES 45ml O_2 / min
  - 20% resting body heat
  - grey matter uses 15x more O_2 than white matter (w/w basis)
  - grey matter has rich capillary supply
- supplies brain, tissue, which with cerebrospinal fluid, is enclosed in rigid cranium; hence volume of total intracranial contents is nearly constant (and brain blood flow is relatively constant)
- when stopped for 10 sec. (eg. cardiac arrest) → unconsciousness
- when PaO_2 decrease to 35mm hg → unconsciousness
- when PaO_2 increased steeply (hyperbaric O_2) → vasoconstriction
- provides glucose as major metabolic energy sources (during starvation free fatty acids and ketones may be used
- is stabilised by baroreceptors in carotid sinus which monitor pressure of blood flowing to brain, and via pressor-reflexes regulate aortic blood pressure; hence, brain is protected from acute changes in blood pressure.

Regulation of cerebral blood flow.

$$F = \frac{\Delta P}{R}$$

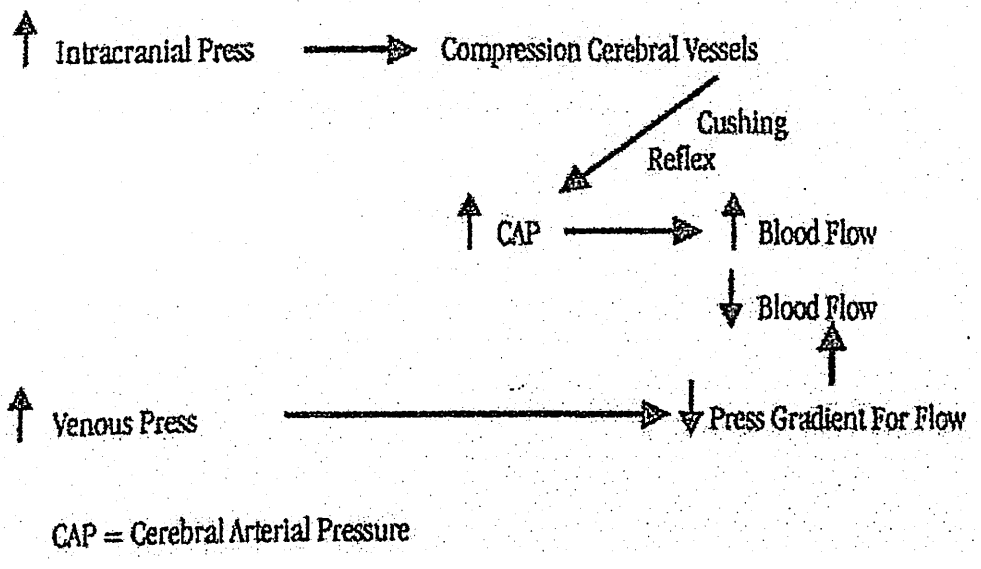
$$= \frac{\text{Mean aortic pressure} - \text{Internal jugular pressure}}{\text{Cerebral vascular resistance}}$$

$$= \frac{MAP - IJV}{CVR}$$

CVR — Due to →
- diameter of cerebral vessels
- viscosity of blood
- intracranial pressure (Ie. Pressure of brain tissue, csf and blood)

On assuming upright position, both arterial and venous pressures in brain decrease by same amount, hence perfusion pressure (and blood flow) are maintained steady.

Effects of intracranial pressure on cerebral blood flow.



CAP = Cerebral Arterial Pressure

Control.

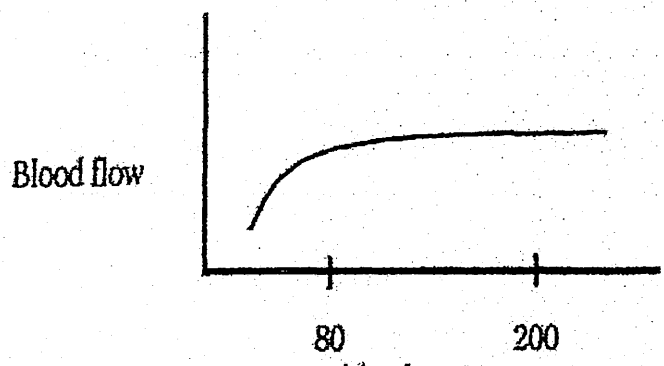
1. Autoregulation
   Main determinant is PACO2flow relatively steady over arterial blood pressure range 60-200mmhg. Anaesthetics may reduce brain metabolism and hence, brain blood flow
2. Sympathetic nervous system (noradrenaline - receptor)
   (constriction)
   Unimportant in cerebral vasomotor regulation
3. Parasympathetic nervous system (ach-muscarinic receptors (dilatation)
   Unimportant in cerebral vasomotor regulation

Autoregulation of circulation.

$$\text{Flow} = \frac{\text{Pressure}}{\text{Resistance}}$$

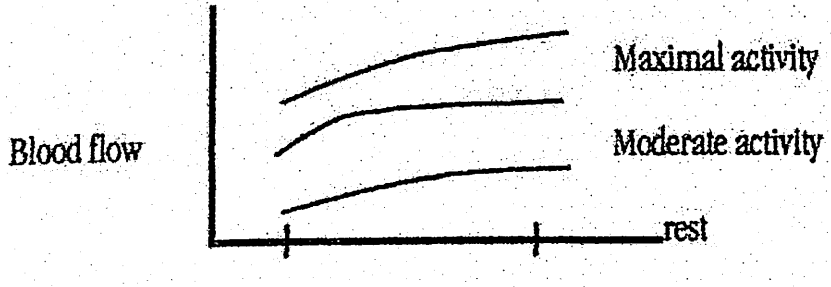
Tissue flow is regulated by intrinsic local changes in resistance of arterioles, metarterioles, and precapillary sphincters.
This tailors the flow to the tissue metabolic requirements.
Autoregulation is of particular importance in vessels of brain, muscle, heart, kidney, intestine and liver.

1. Tissues whose metabolic requirements are relatively constant, eg. brain



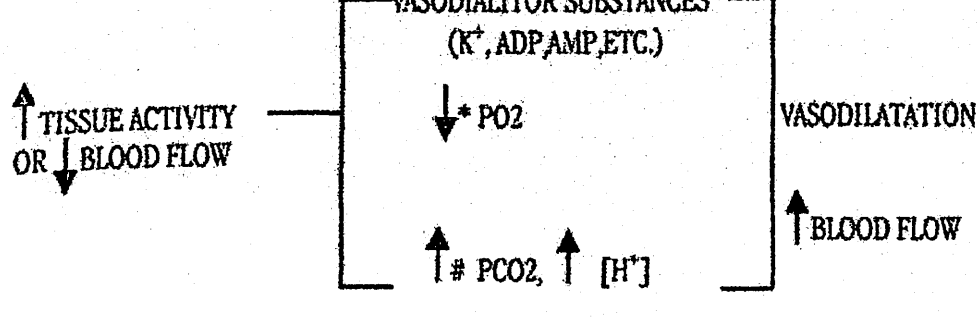
Flow-pressure relationship in tissues with relatively constant metabolic requirements.

2. Tissues whose metabolic requirements changes widely, eg. skeletal and cardiac muscle.

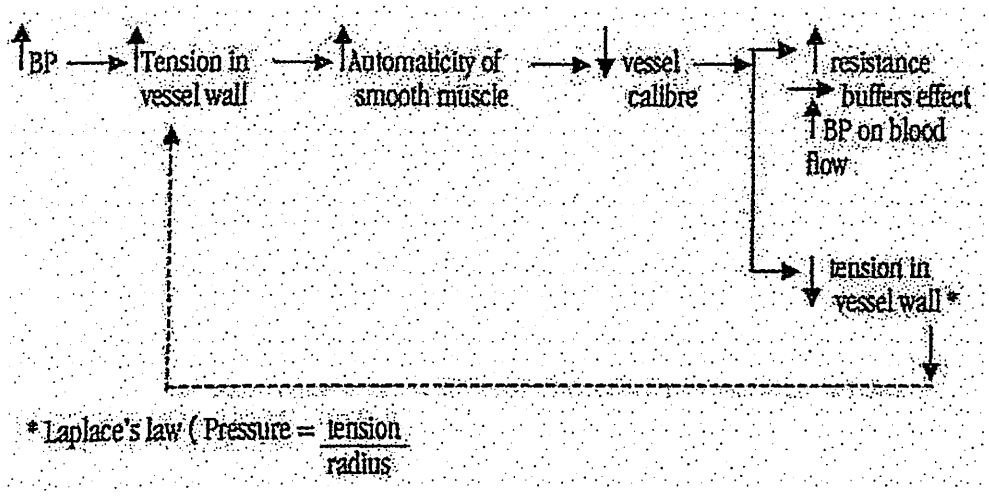


Flow-pressure relationship in tissues with changing metabolic requirements.

Mechanism of autoregulation.



* especially in muscle
# especially in brain (CO_2 diffuses through blood-brain barrier into interstitial fluid $\longrightarrow H^+ \longrightarrow$ dilatation)

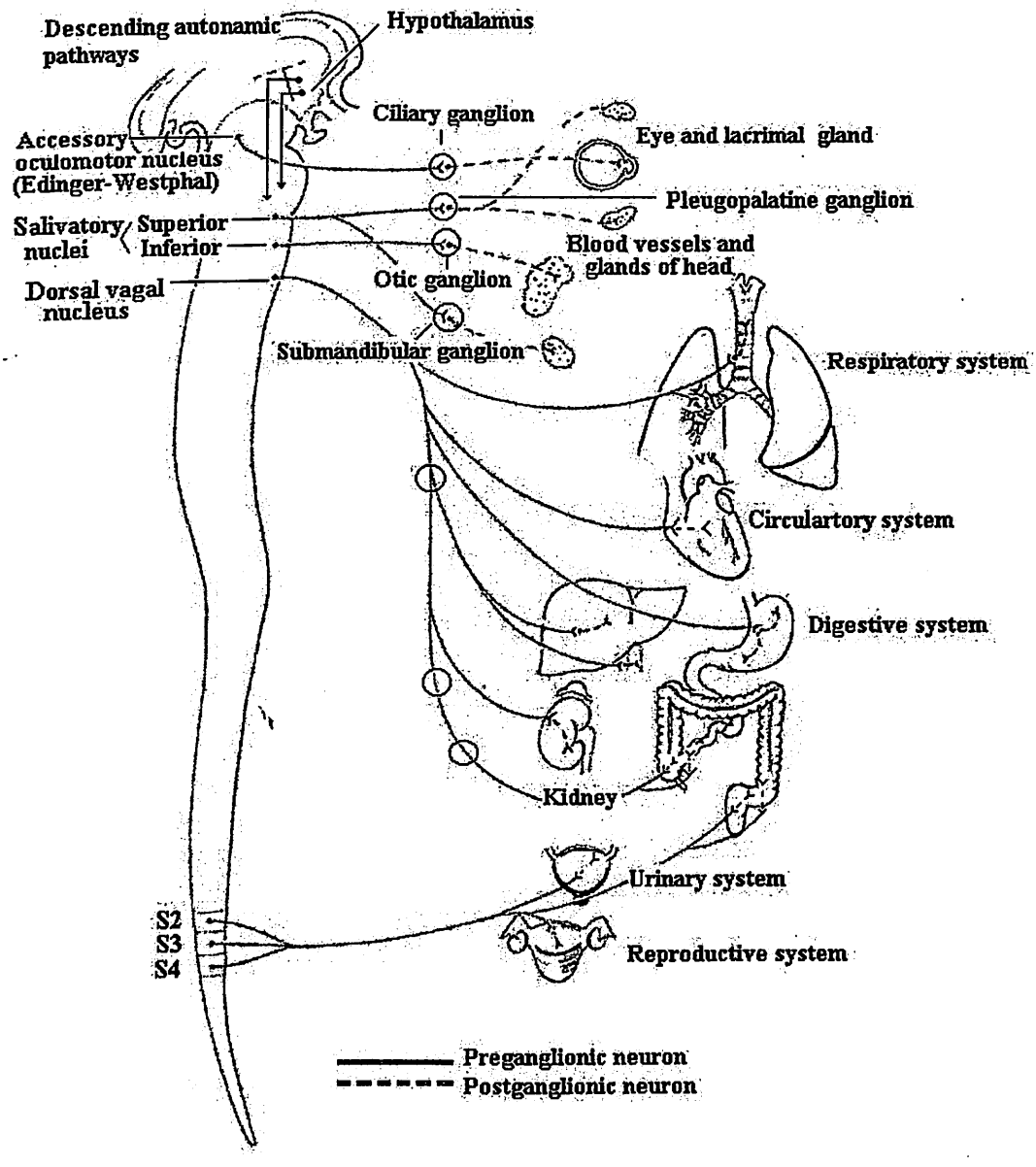


* Laplace's law (Pressure = $\frac{\text{tension}}{\text{radius}}$

SISTEM SARAF PUSAT

Modul 26 Diagram of the parasymphathetic (craniosacral) division of the autonomic nervous system.

The preganglionic and postganglionic neurons are cholinergic.



SISTEM SARAF PUSAT

Modul 27 Classification of nerve fibers.

1. Motor and sensory nerve.

Classification of nerve fibers by Erlanger/Gasser:

Fiber type	Function (examples)	Avg. fiber diameter (μm)	Avg. cond. velocity (m/s)
Aα	Primary muscle-spindle afferents, motor to skeletal muscles	15	100(70-120)
Aβ	Cutaneous touch and pressure afferents	8	50(30-70)
Aγ	Motor to muscle spindles	5	20(15-30)
Aδ	Cutaneous temperature and pain afferents	<3	15(12-30)
B	Syampathetic preganglionic	3	7(3-15)
C	Cutaneous pain afferents, sympathetic postganglionic	1 (unmyelinated)	1(0.5-2)

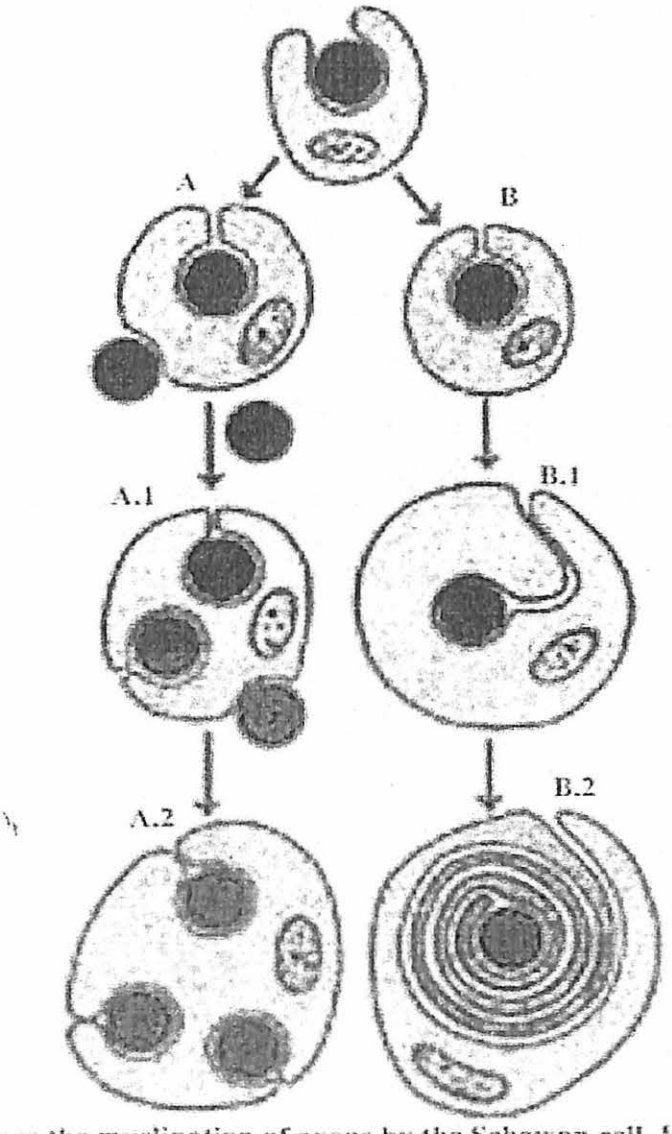
2. Sensory nerves.

Classification of nerve fibers by Lloyd/Hunt:

Group	Function (examples)	Avg. fiber diameter (μm)	Avg. cond. velocity (m/s)
I	Primary muscle-spindle afferents and afferents from organs	13	75(70-120)
II	Cutaneous mechanoreceptors	9	55(25-70)
III	Deep pressure sensors in muscle	3	11(10-25)
IV	Unmyelinated pain fibers	1	1

SISTEM SARAF PUSAT

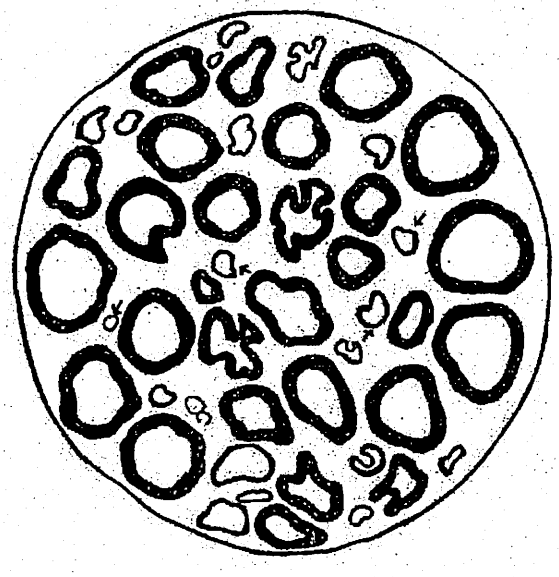
Modul 28 Mylination of axons.



Schemes to illustrare the myelination of axons by the Schawnn cell. In A, A1 and A2 a number of axons invaginate a Schawnn cell, such axons are said to be non-myelinated. In B a single investigates a Schawnn cell. In B1 early rotation of Schawnn cell is shown. In B2 the Schawnn Cell has rotated a number of times round the axis cylinder in such a way that the axon becomes surrounded by a number of double layers of the Schawnn Cell cytoplasm. B, B1 and B2, therefore, are successive stages in the development of the myelin sheath.

SISTEM SARAF PUSAT

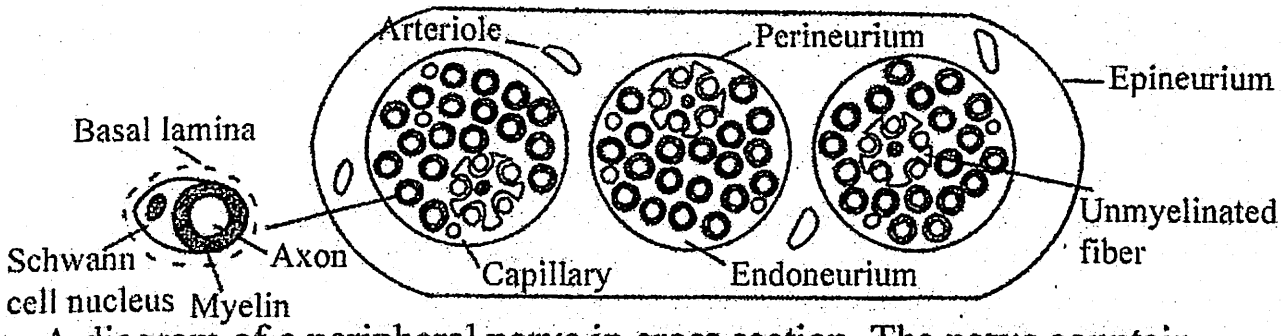
Modul 29 Cross section of a peripheral nerve showing myelinated and unmyelinated (arrow) fiber.



SISTEM SARAF PUSAT

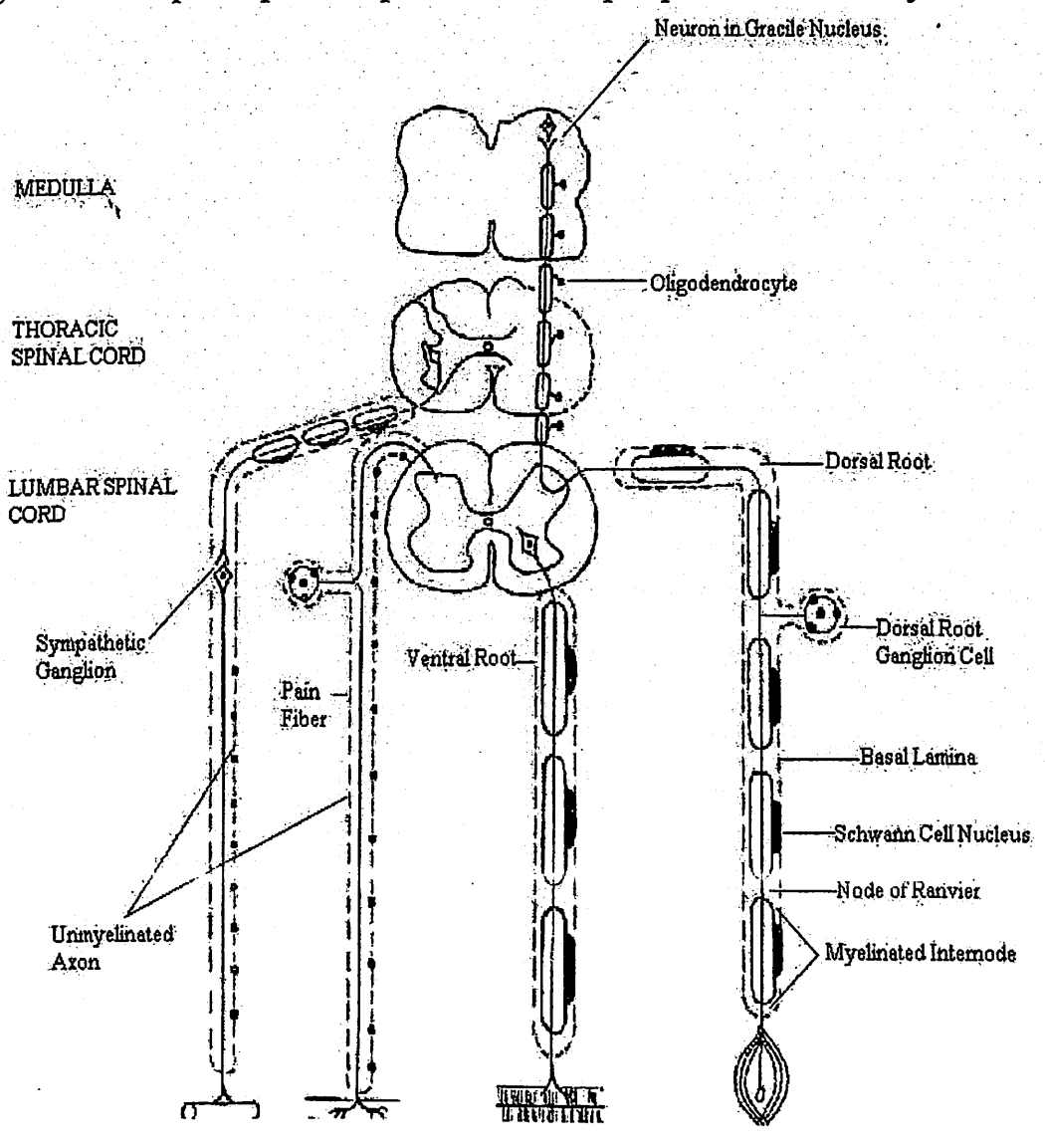
Modul 30 Cellular organization of peripheral nerves.

A. Peripheral nerve components.



A diagram of a peripheral nerve in cross section. The nerve conntain three fascicles. The figure on the left represents a high magnification of a mylenated axon in cross section.

B. A diagram of the principal components of the peripheral nervous system.



ISTEM SARAF PUSAT

Modul 31 Nerve injuries causing bladder dysfunction.

Nerve injuries produce 3 types of bladder dysfunction:

A. Atonic bladder

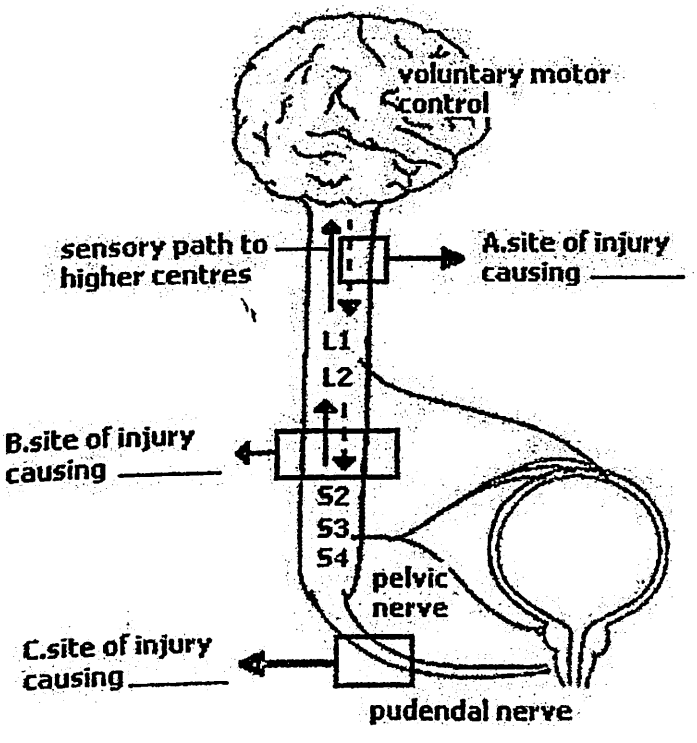
Interruption of sensory nerve supply results in loss of bladder tone. The bladder may become extremely distended with no development of an urge to urinate. It fills to capacity, then overflow.

B. Hypertonic bladder

Interruption of the voluntary pathways results in excessive tone, and small distensions create an uncontrolled desire to urinate.

C. Automatic bladder

Complete section of the cord above S1 causes automatic emptying in response to filling.



SISTEM SARAF PUSAT

Modul 32 Segmental demyelination.

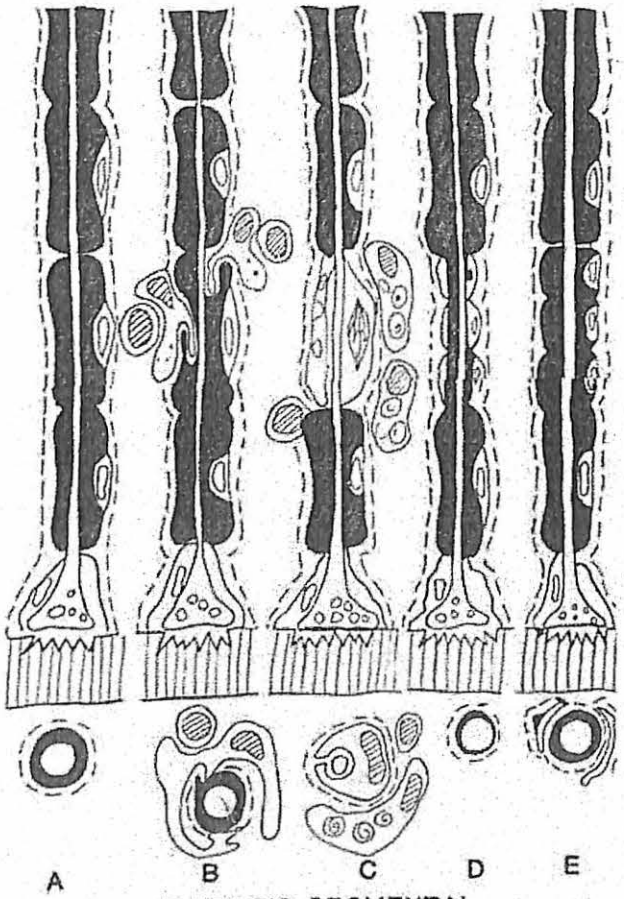


Diagram to summarize allergic segmental demyelination and remyelination.

A.Normal fibre.

B.Demyelination;macrophages are stripping the myelin sheath. Associated lymphocytes are also shown.

C. Schwann cell mitosis.

D,E. Remyelination.

SISTEM SARAF PUSAT

Modul 33 Degeneration and regeneration in a mylenated nerve fiber.

Schema of the wallerian degeneration and the regeneration in a severed peripheral myelinated nerve fibre. In these drawing are presented the endoneurium sheath in which the nuclei of the fibroblasts the myelin sheath in which the Schwann cell nuclei are surrounded by some cytoplasm (in dark grey). The neurofibrils are indicated within the axon.

i) General view indicating the location of the nerve transection. The part of the nerve within the rectangular line is presented
in higher magnification in the numbers (ii) up to (viii).

ii) 1st day after the section: swelling of the proximal axon stump end.

iii) 2nd day: the axon is shrunken, varicose and the neurofibrils have disappeared; there is a retraction of the myelin sheath
from the nodes and a proliferation of the endoneurium disappeared; there is a retraction of the myelin sheath from the
nodes and a proliferation of the endoneurium fibroblasts.

iv) 3rd day: vacuolization and fragmentation of the axon; fragmentation of the myelin; multiplication of the Schwann cells.
The myelin digestion starts at the periphery of the Schwann cells.

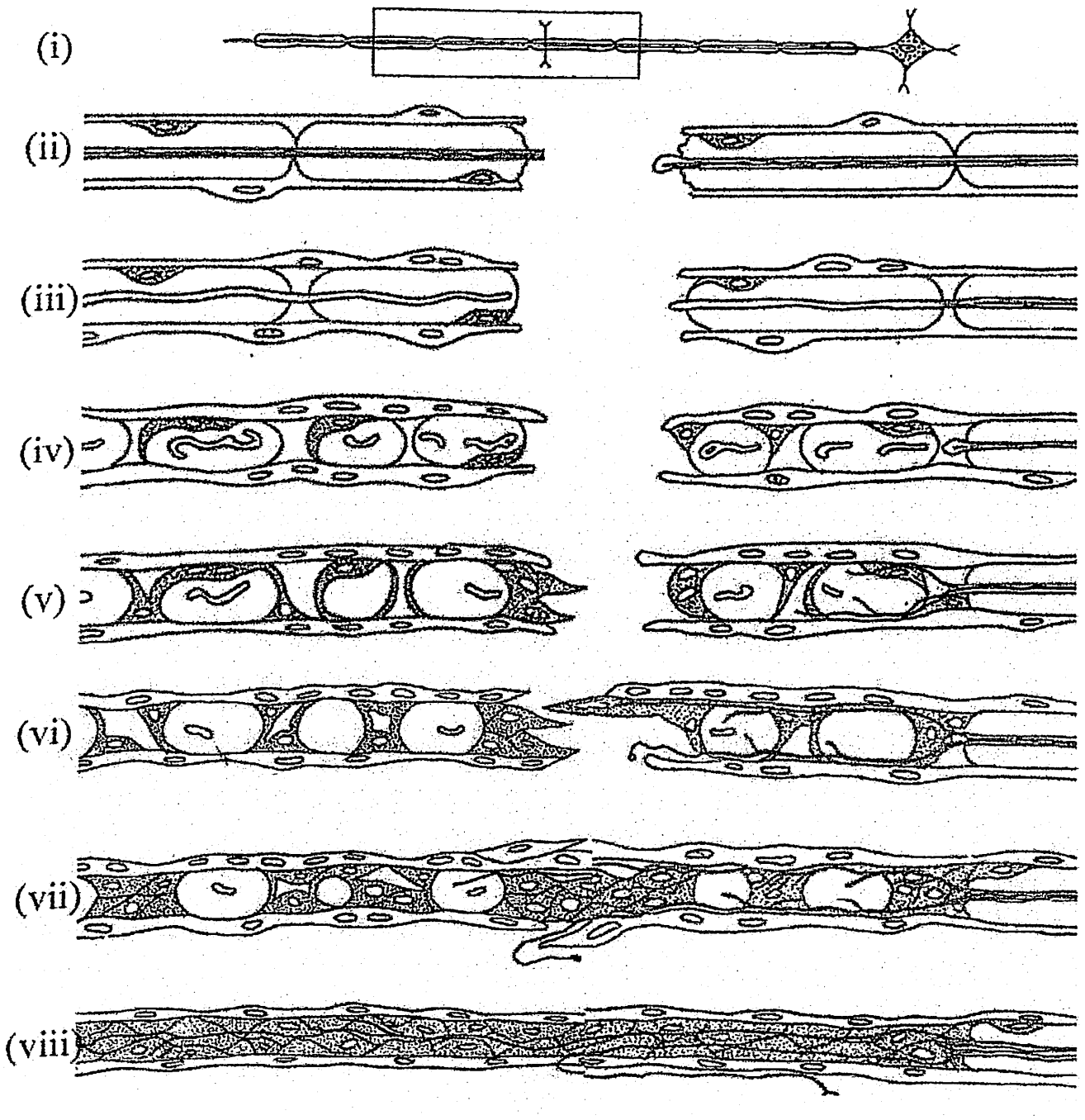
v) 8th day: the axon fragments disappear; the Schwann cells invade the gap between both. nerve ends; the myelin stains
Marchi positive (in light grey); regeneration is strarting with a fork-like outgrowth of neurofibrils from the proximal axon
stump. These fibrils grow along the periphery of the myelin ellipsoids.

vi) 12th day: the myelin fragments stain Sudan positive (in middle grey).

vii) 20th day: the gap between both nerve ends is closed by Schwann cells and fibroblasts. This is the so called band fibre or
Bungner band. Only one nerve fibre successfully grows into the peripheral stump. An other fibre has deviated into the
surroundings.

viii) 100th day: all axon and myelin fragments have disappeared. The diameter of the nerve is still less then nerve originally was.

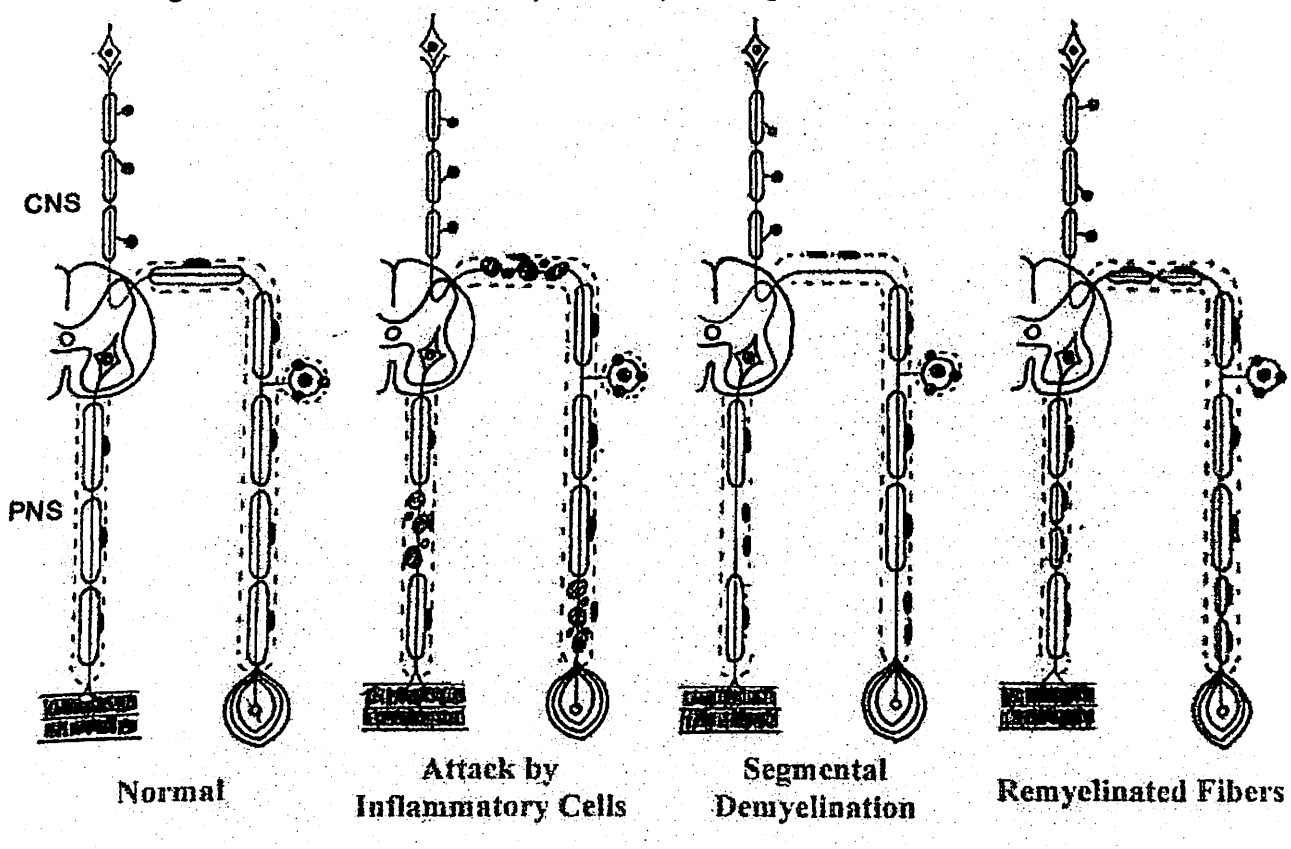
Although is reinnervation there still is no remyelination yet.


(i)
(ii)
(iii)
(iv)
(v)
(vi)
(vii)
(viii)

SISTEM SARAF PUSAT

Modul 34 Primary myelinopathy (e.g. Inflammatory)

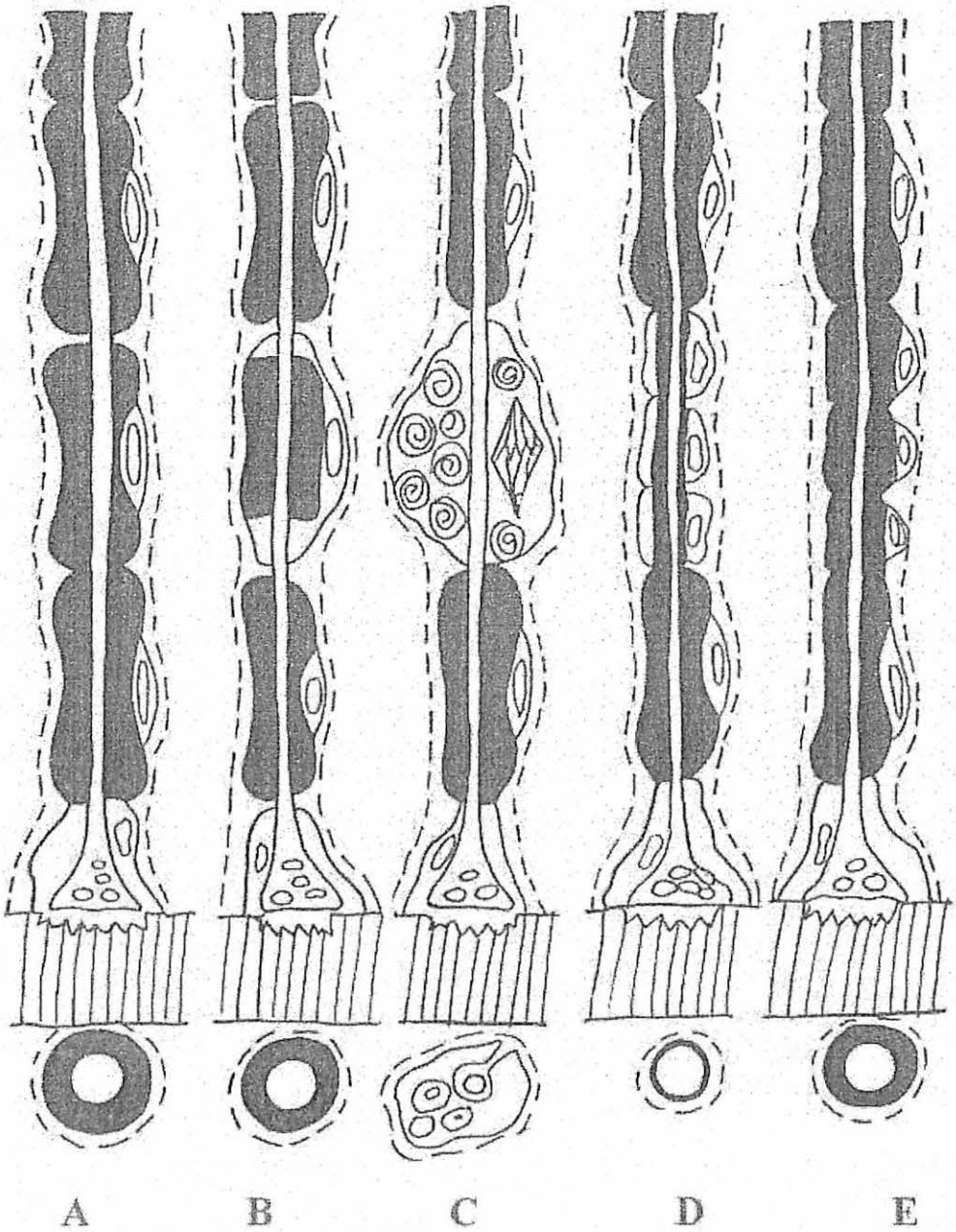
A diagram of the cardinal pathologic features of an inflammatory PNS myelinopathy. Axons are spared as is CNS myelin. Following the attack, the remaining Schwann cells divide . The denuded segments of axons are remyelinated, leaving them with shortened internodes.



SISTEM SARAF PUSAT

Modul 35 Segmental demyelination.

Diagram to summarize the events occurring in primary segmental demyelination and remyelination. A. Normal nerve. B. Early segmental demyelination; retraction of paranodal myelin with widening of the nodal gap. C.Destruction of myelin sheath and Schwann cell mitosis. D,E. Remyelination; intercalated short segments.

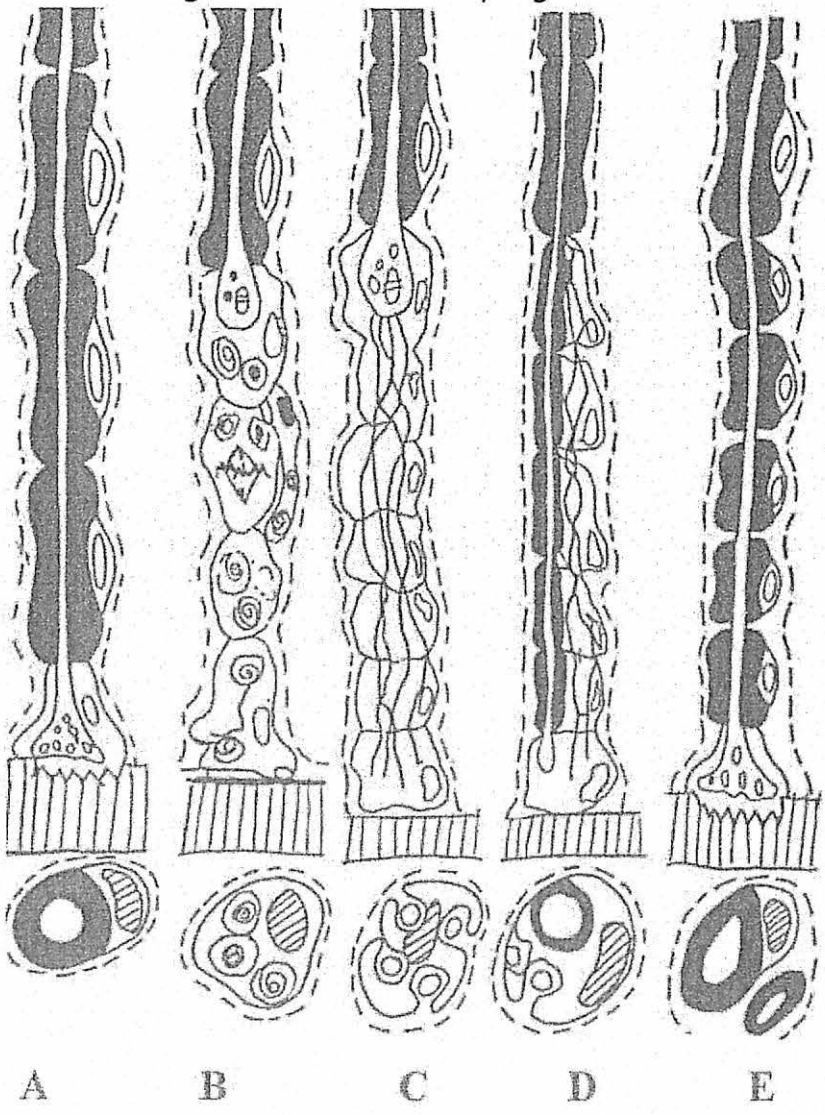


Primary segmental demyelination and remyelination

SISTEM SARAF PUSAT

Modul 36 General pathology of peripheral nerves.

Diagram,summarizing the events occurring during axonal degeneration and regeneration.
A.Normal nerve.
B.Seven days after axonal damage; Schwann cells containing axon and myelin debris have divided to form bands of Bungner. C.Axon sprouts grow from the swollen end bulb of the proximal axon.
D.An axon becomes myelinated.
E.Connection with the endorgan is re-established; regenerated internodes are short.

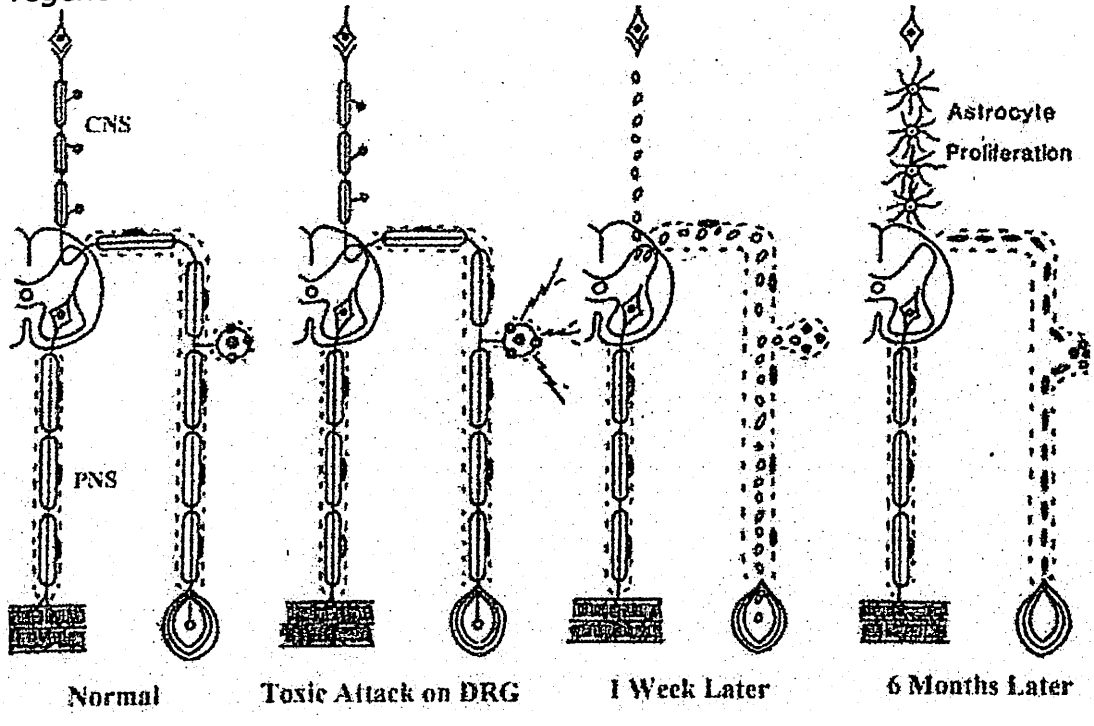


Axonal degenaration and regenaration

SISTEM SARAF PUSAT

Modul 37 Toxic sensory neuronopathy.

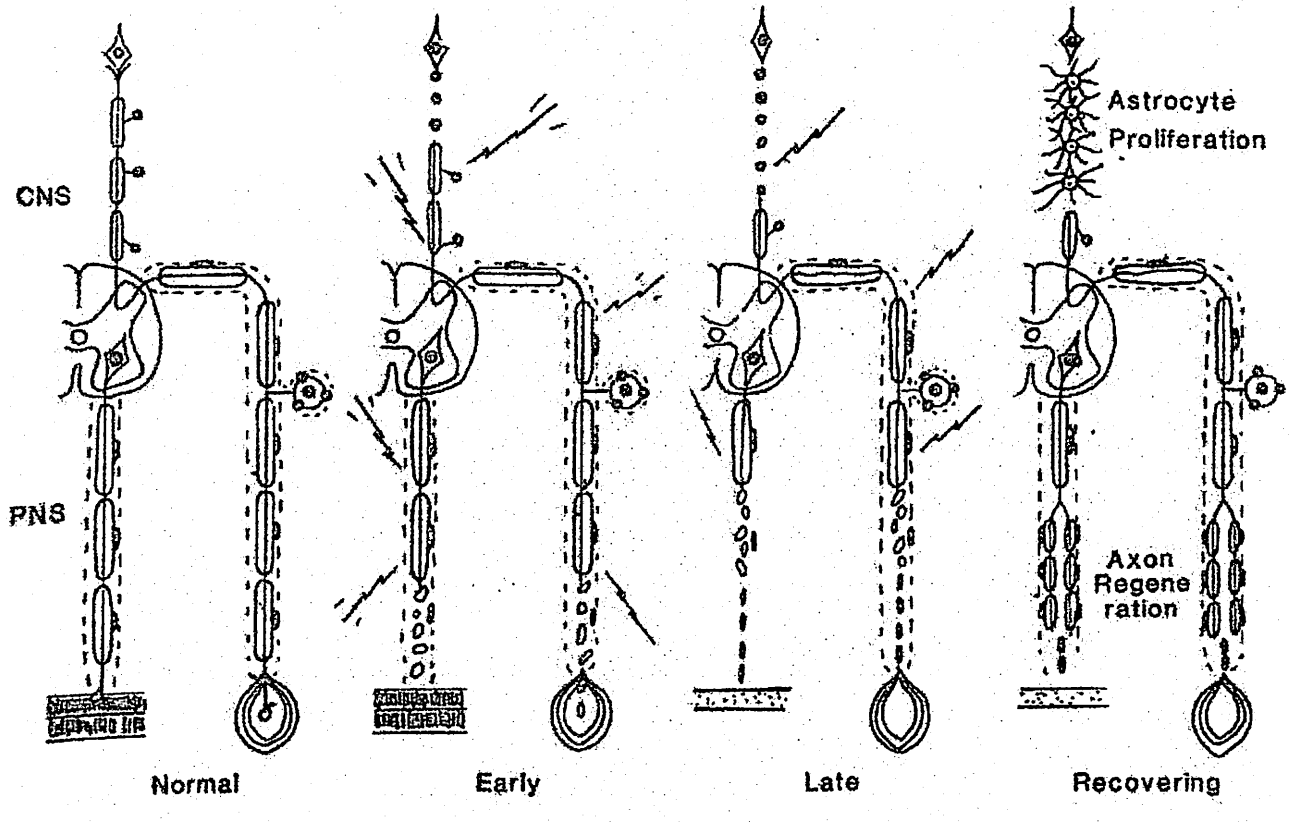
A diagram of the cardinal features of a rapidly involving toxic sensory neuronopathy. The jagged lines (lightning bolts) indicate that the toxin is directed at neurons in the dorsal root ganglion (DRG). Degeneration of these cells is accompanied by fragmentation and phagocytosis of their peripheral-central processes. The Schwann cells remain, there is no axonal regeneration.



SISTEM SARAF PUSAT

Modul 38 Toxic distal axonopathy.

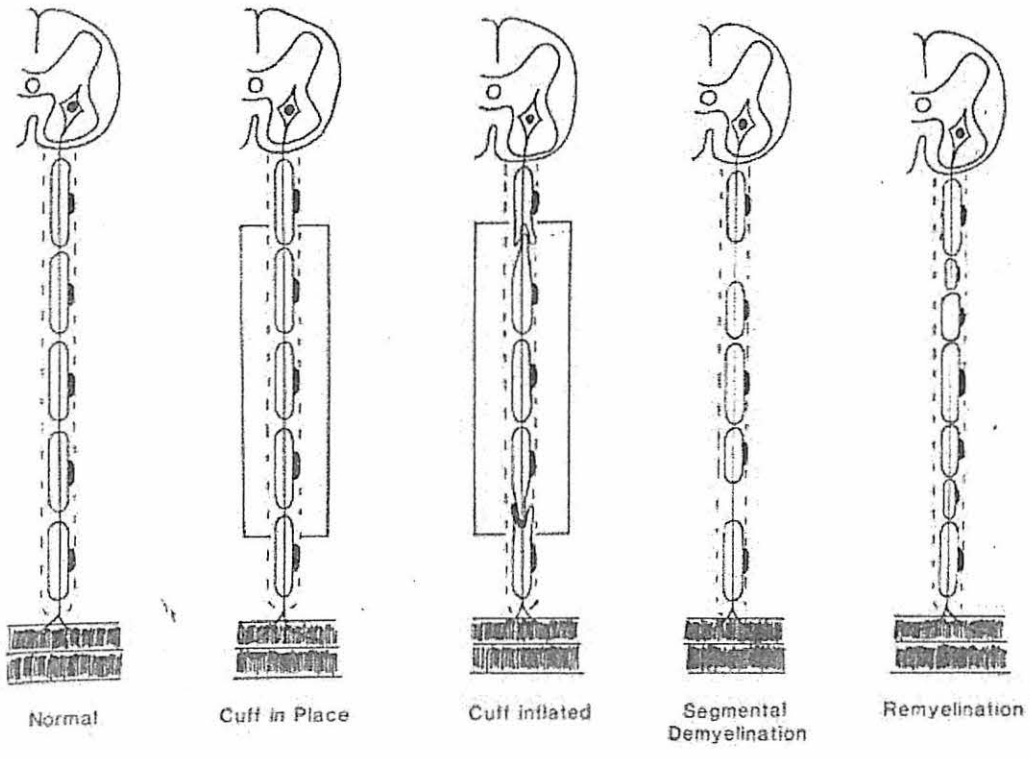
A diagram of the cardinal pathologic features of a toxic distal axonopathy. The jagged lines (lightning bolts) indicate that the toxin is acting at multiple site along motor and sensory axons in the PNS and CNS. Axon degeneration has moved proximally (dying – back) by the late stage. Recovery in the CNS is impeded by astroglial proliferation.



SISTEM SARAF PUSAT

Modul 39 Class 1 acute nerve injury (eg. compression).

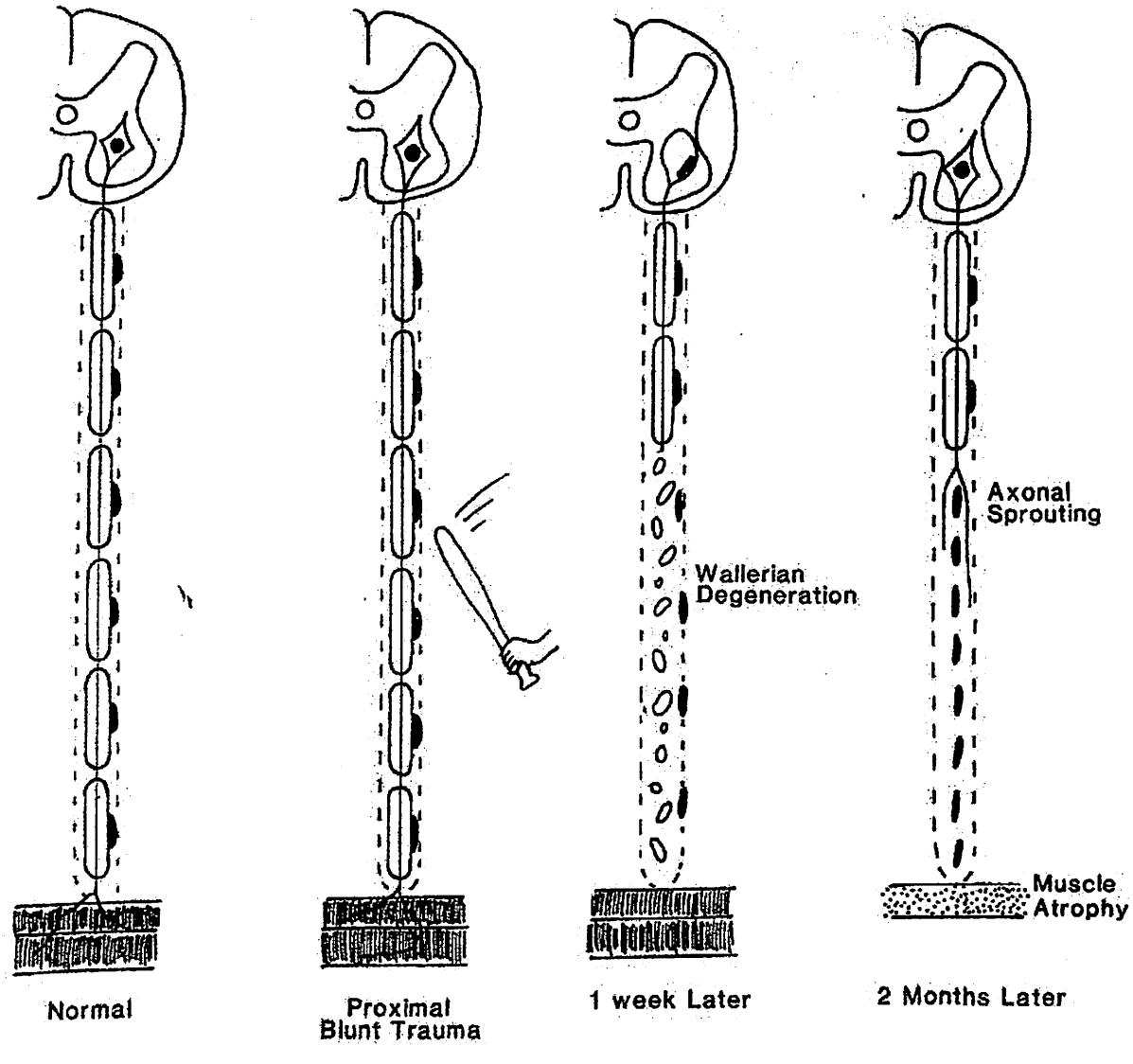
Class 1 (neurapraxia) nerve injury associated with compression by a cuff. Axon movement at both edges of the cuff causes intussusception of the attached myelin across the node of Ranvier into the adjacent paranode. Affected paranodes demyelinate. Remyelination begins following cuff removal and conduction eventually resumes. Conduction is normal in the nerve above and below the cuff since the axon has not been damaged.



SISTEM SARAF PUSAT

Modul 40 Class 2 nerve injury.

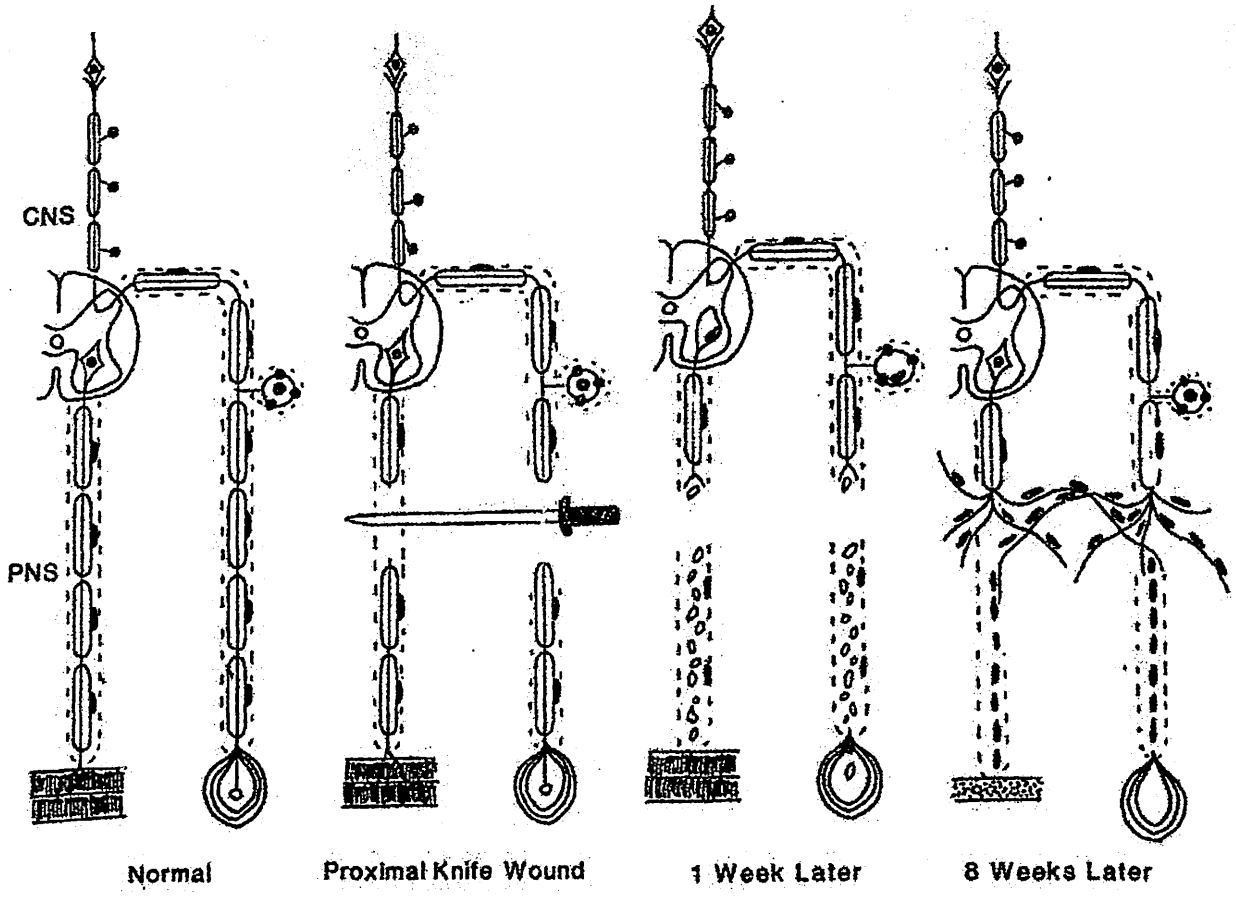
Class 2 nerve injury (axonotmesis) from a crush injury to a limb. Axonal disruption occurs at the site of injury. Wallerian degeneration takes place throughout the axon distal to the injury with loss of axon, myelin, and nerve conduction. Preservation of Schwann cell tubes and other endoneurial connective tissue ensures that regenerating axons have the opportunity to reach their previous terminals and, hopefully, re-establish functional connections.



SISTEM SARAF PUSAT

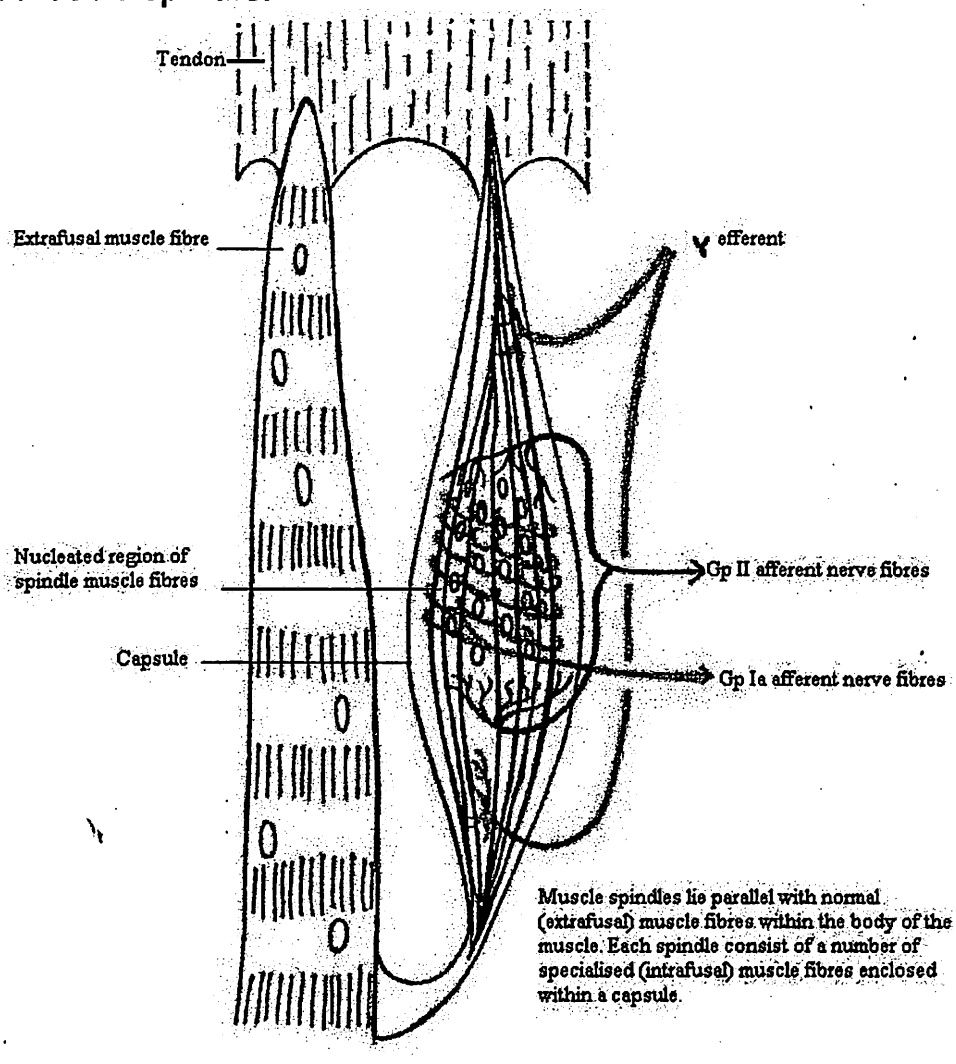
Modul 41 Degenaration and abberant regeneration in (class 3) nerve injury.

Class 3 nerve injury (neurotmesis) with severance of all neural and connective tissue elements. There is little hope of functional recovery without skilled surgery. Regenerating axons are entering inappropriate Schwann cell tubes (aberrant regeneration).

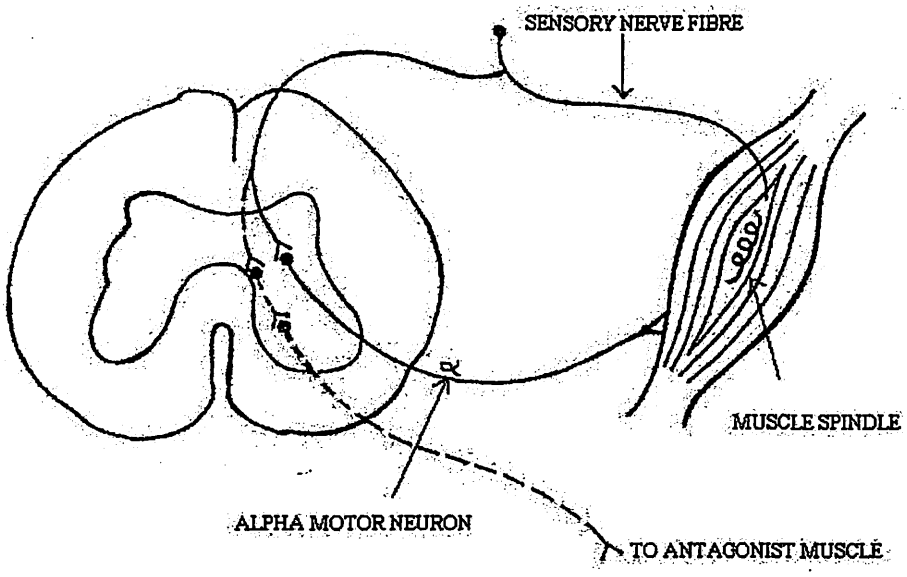


SISTEM SARAF PUSAT

Modul 42 Muscle spindle.



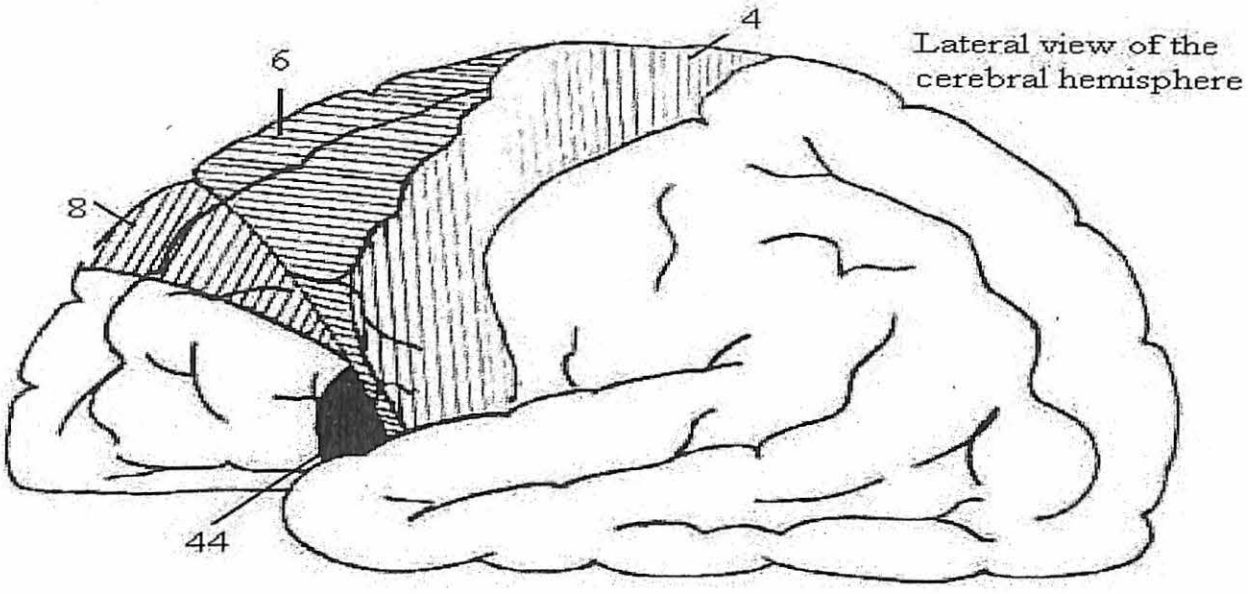
MUSCLE SPINDLE REFLEX



Impulses in the sensory nerve fibre of a muscle spindle produce muscle contraction via a nonosynaptic reflex-the stretch reflex.
These fibres also produce inhibition of motor neurons of antagonist muscles.

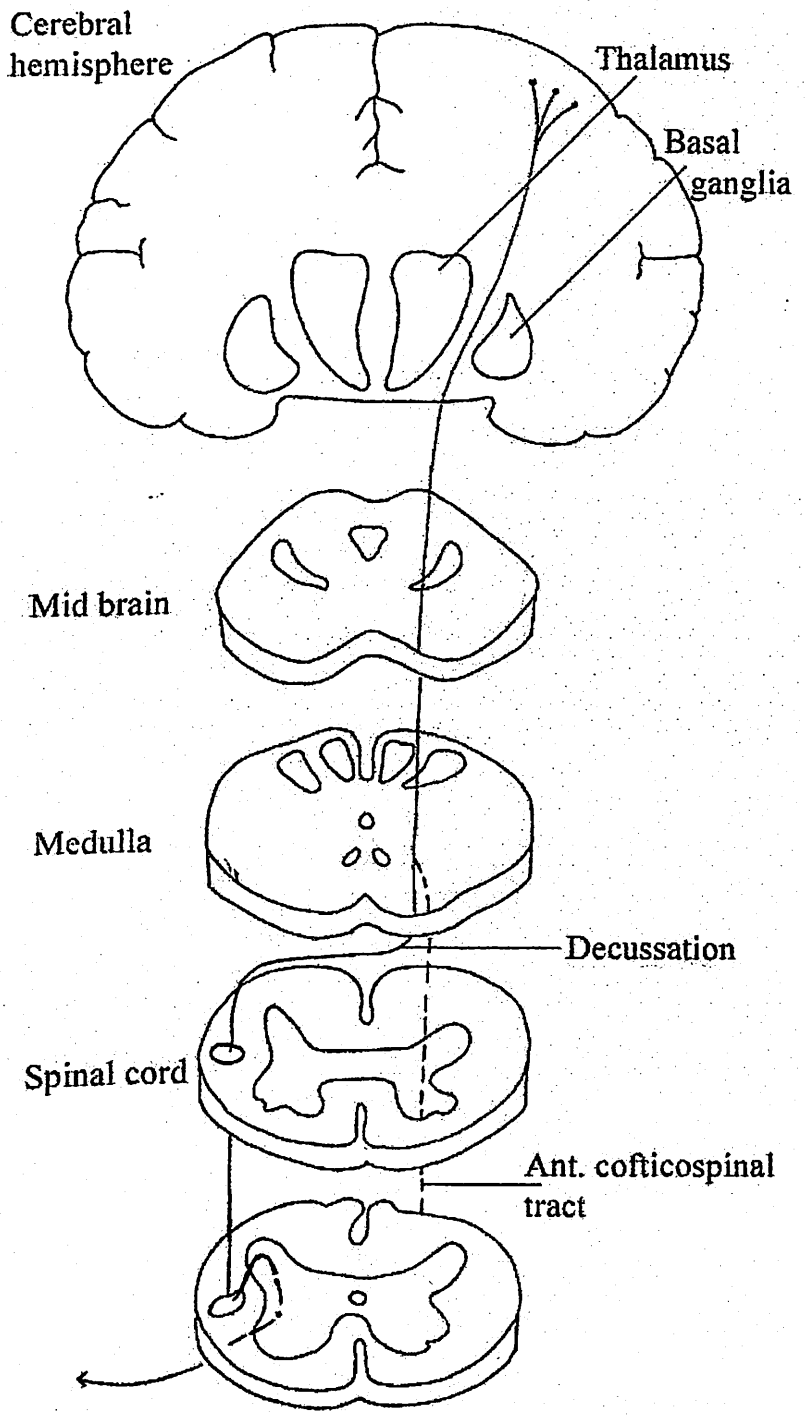
SISTEM SARAF PUSAT

Modul 43 Cerebral cortex.

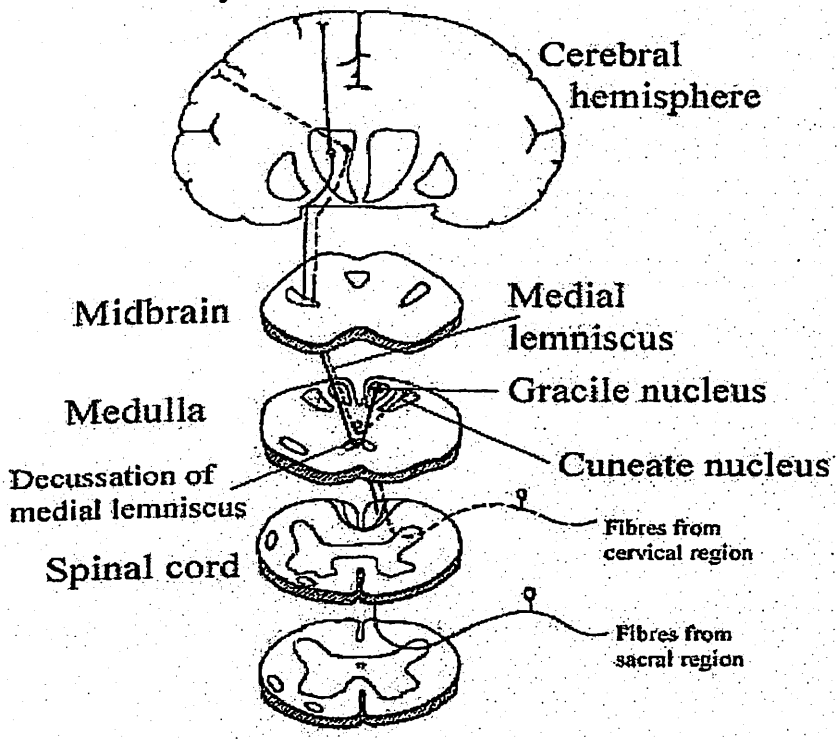


On the diagram above, indicate where the motor areas of the brain are.

Pyramidal system.

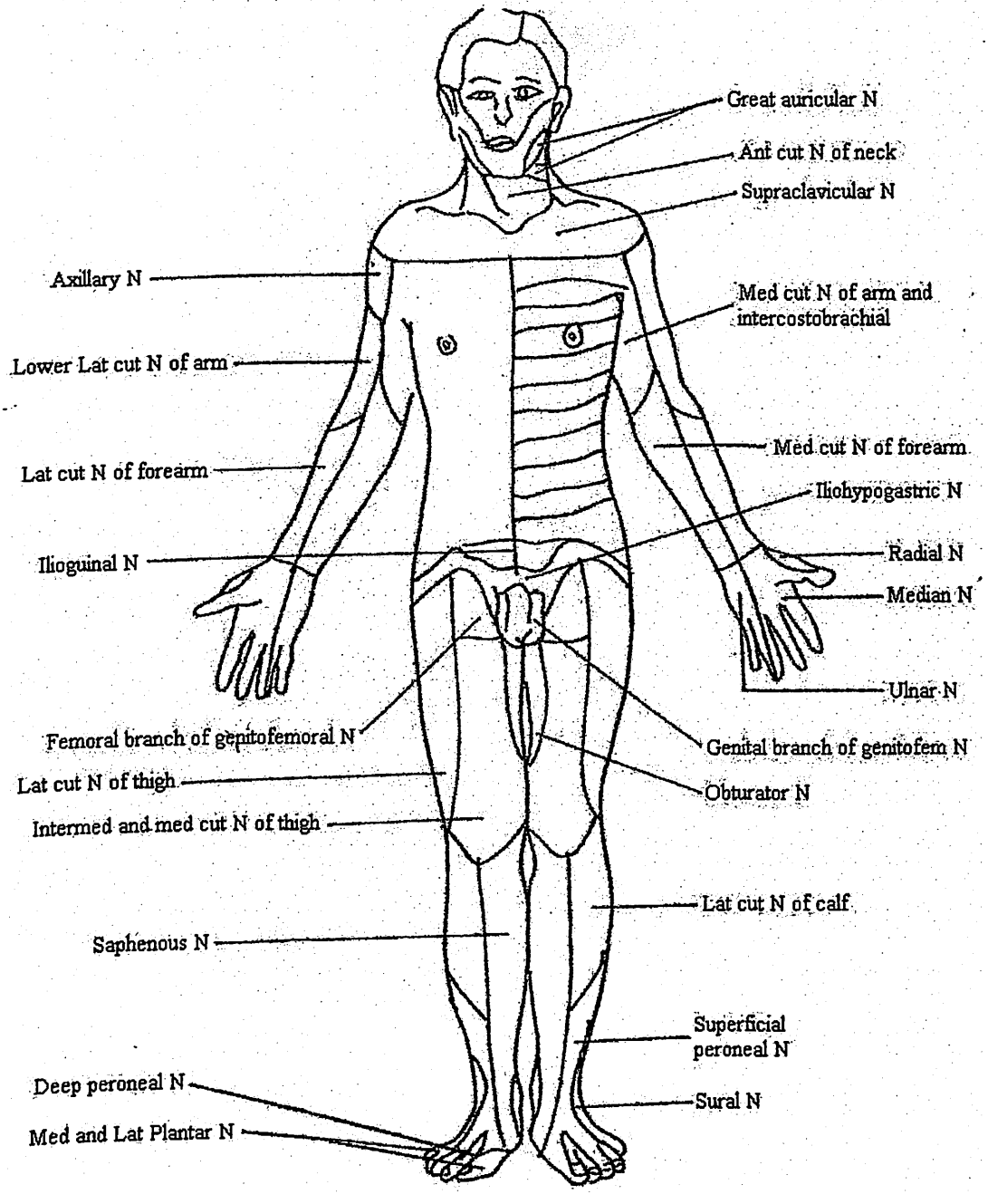


Dorsal column system.



SISTEM SARAF PUSAT

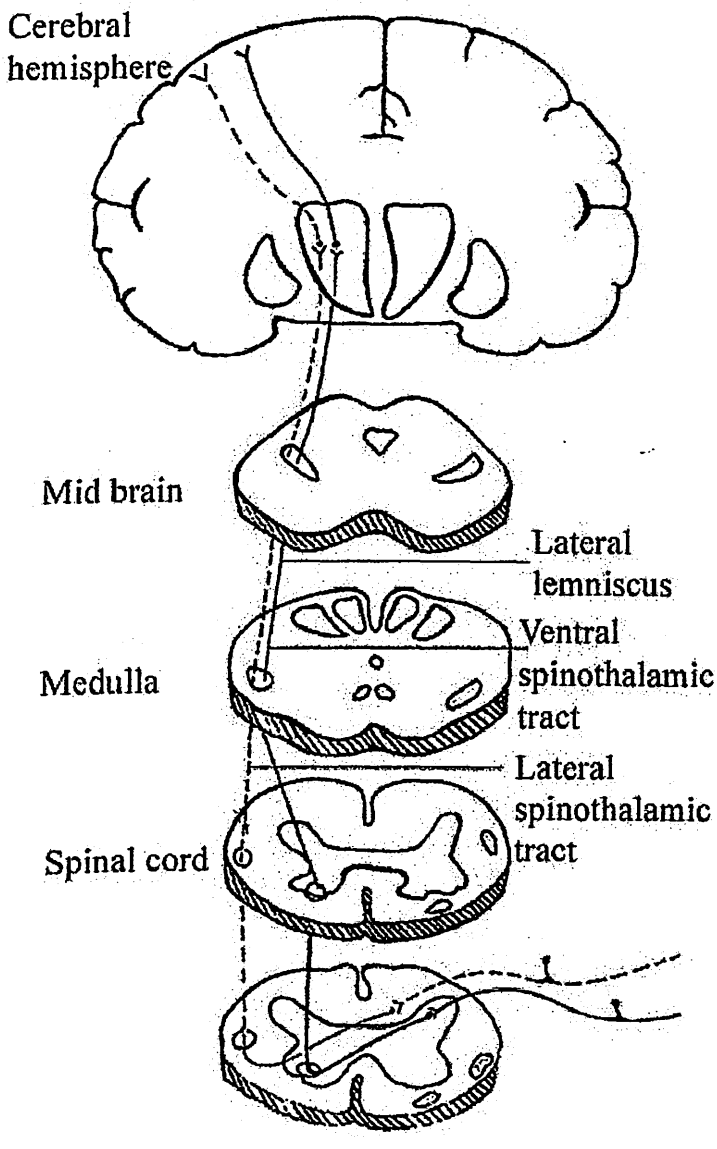
Modul 44 Cutaneous distribution of major peripheral nerves.



The distribution differs fromthat of spinal nerves.

SISTEM SARAF PUSAT

Modul 45 Spinothalamic tracts.



SISTEM PENCERNAAN

SISTEM PENCERNAAN

Modul 1 Gastrointestinal tract

Modul 2 Secretion and digesion

Modul 3 Absorption

Modul 4 Motility

Modul 5 Control of small ontestine

Menu Utama

Modul 1 Gastrointestinal tract.

The 4 main function of the GIT are :

1. Secretions

2. Digestion

3. Absorption

4. Motility/ excretion

SECRETIONS AND DIGESTION (1)

A. FUNCTION

Breakdown of food into simpler components (monosacehariclés, amino acids, free fatty acids) for absorption and assimilation.

B. SITES

(A) Mouth

(B) Stomach

(C) Small intestines

THE MOUTH

1.1 Saliva - produced by

i) Parotid gland

ii) Submaxillary gland

iii) Sublingual gland.

1.1.1 Composition of saliva

i) Enzymes- a amylase and Lingual lipase

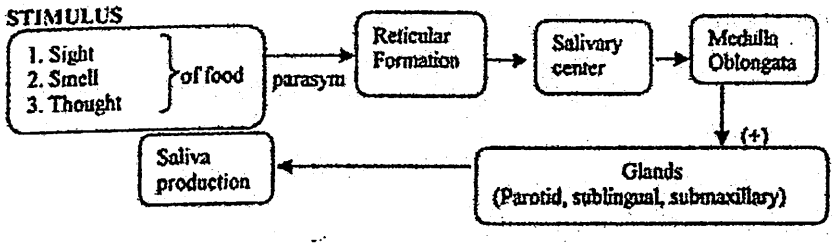
ii) Electrolytes

1.1.2 Volume of saliva : 0.5- 1.5 L/day (PH 6.4 - 7.0)

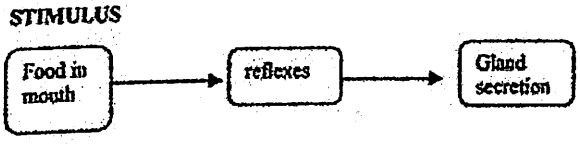
Little digestion of carbohydrate (a-amylase) and lipid (lingual lipase) occurs in the mouth.

1.2 Secretions controlled by 3 mechanisms

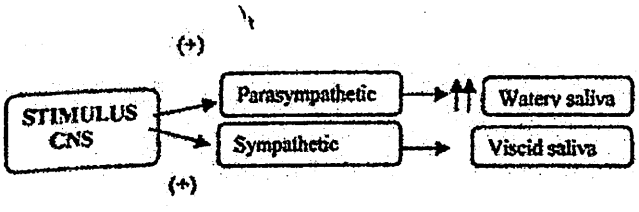
1.2.1 Conditional reflex (cephalic phase)



1.2.2 Local



1.2.3 Neural



The food is transported into the stomach via the OESOPHAGUS by PERISTALTIC MOVEMENT.

(Ref. FLM on MOTILITY Station 4)

Modul 2 Secretions and Digestion

THE STOMACH

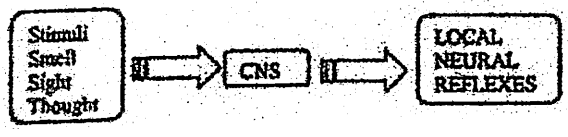
2.1 Function to

i) mix food

ii) add gastric juice (HCL , pepsinogen , gastric lipase) to the content of the stomach produce CHYME

2.2 Secretion of gastric juice is effected by 3 mechanisms

2.2.1 Cephalic phase of gastric secretion

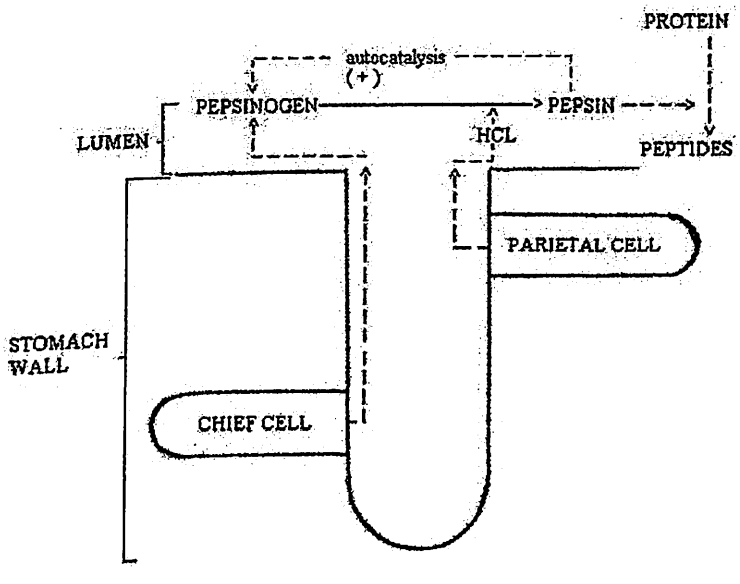


2.2.2 Hormonal

This involves a hormone called : Gastrin.

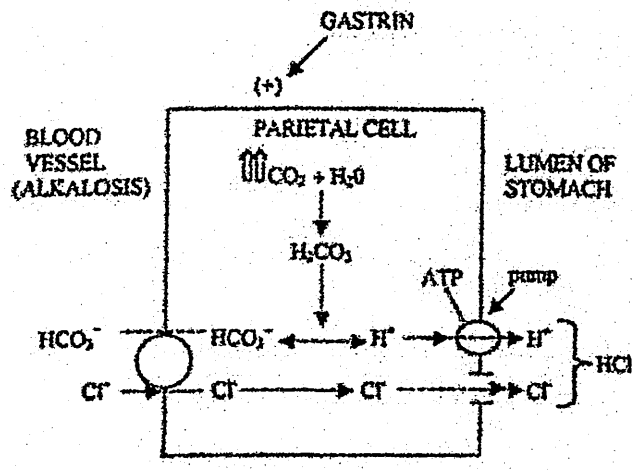
Gastrin is released by G cells and stimulates HCL and pepsinogen secretion as illustrated in (Station 2 e)

2.3 Mechanism of Pepsinogen catalysis by HCL



HCL catalyses the break down of Pepsinogen into Pepsin. Pepsin digests the proteins ⟶ PEPTIDES

2.4 Mechanism of HCL secretions - 3 (Cephalic, Gastric, Intestinal)



Gastrin stimulates the Parietal cell to ↑ metabolism → CO_2
CO_2 hydrated → $H_2CO_3^-$
$H_2CO_3^-$ split up into HCO_3^- + H^+
H^+ is pumped out into lumen of stomach
HCO_3^- into the blood in exchange for Cl^- which enters the lumen of the stomach

2.5 Neural : Local reflexes (ref FLM 2q)

THE SMALL INTESTINE

Functions to

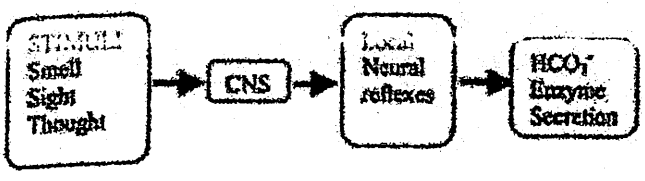
i) mix the chyme with alkaline secretions

ii) mix the chyme with secretion from pancreatic and bile duct cells (HCO3-)

iii) realease digestive enzymes from the pancreas.

3.2 Secretion of intestinal juices is effected by mechanisms

3.2.1 Cephalic phase of intestinal secretion



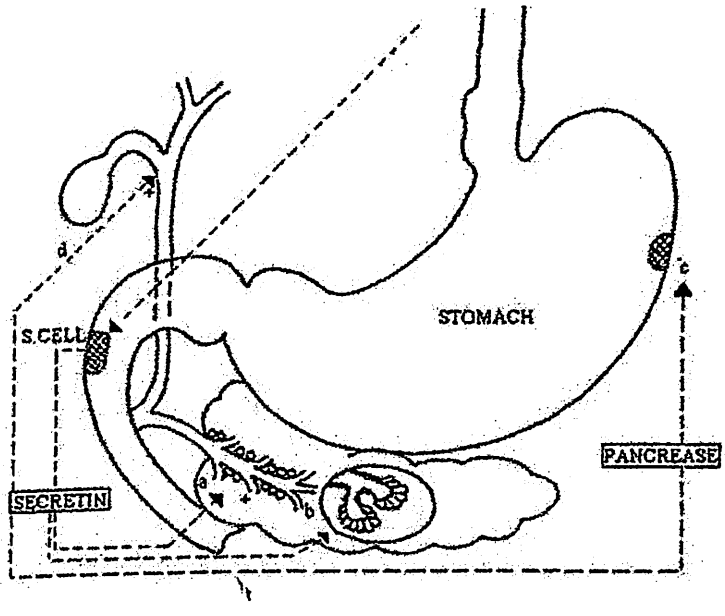
3.2.2 Hormonal

This phase involves hormones of both the secretin and gastrin families

STIMULI

(i) Acid in Small Intestines

(ii) Products of Pr - digestion (AA, Peptides)

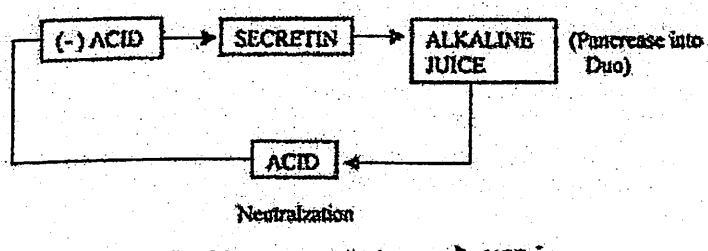


ACTIONS

(a) Stimulates Pancreatic Duct cells → HCO_3^-

(b) Augments CCK to produce Pancreatic Enzyme (Exocrine cells)

(c) ↓ gastric acid secretion by a NEGATIVE FEEDBACK MECHANISM as below.



(d) Stimulates cells of the common Bile duct → HCO_3^-

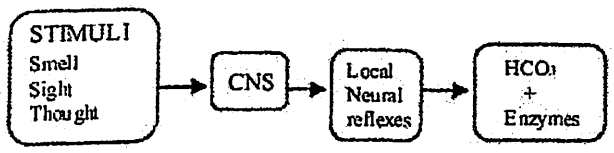
3.4 Neural : Local reflexes (refer FLM 2q)

THE SMALL INTESTINAL- hormones

CHOLECYSTOKININ (CCK)

3. It's secretion is effected by the same 3 mechanisms as for other hormones

3.1.1 Cephalic phase of release

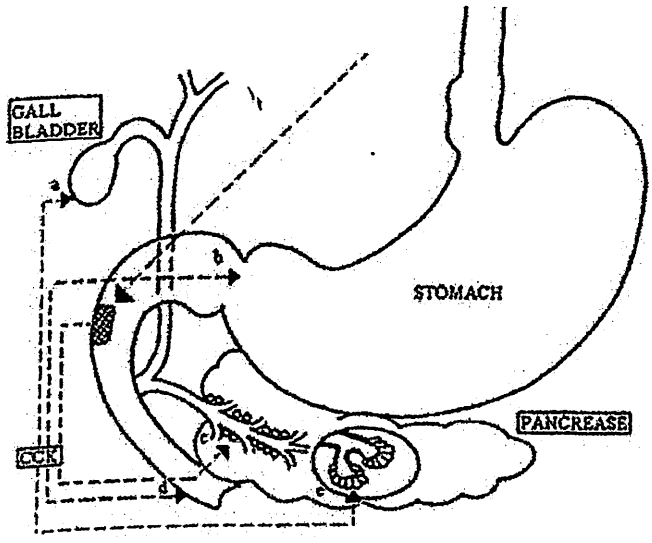


3.2.1 HORMONAL : Release and actions

STIMULI

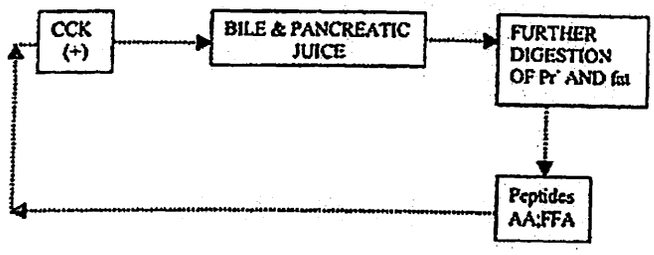
(i) Fatty Acids

(ii) Amino Acids/Peptides



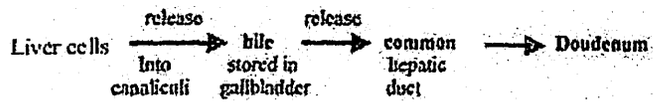
ACTIONS

(a) Contraction of gallbladder ⟶ Bile release for FAT digestion digestion
(b) Inhibition of gastric emptying
(c) Stimulation of pancreatic duct cell ⟶ HCO_3
(d) ↑ secretion of ENTEROKINASE ⟶ ↑ motility
(e) Stimulation of pancreatic juice ⟶ secretion of Pancreatic Enzymes
CCK acts by a POSITIVE FEEDBACK SYSTEM



Secretion and Digestion - Bile

PRODUCTION OF BILE



* 80-90 % from haemaglobin, Haemproteins

* 25 % Hepatic cytochrome P450

COMPOSITION : bile salts, bile pigment + other substances in an alkaline electrolyte solution.

Water ---------- 97.0 %

Bile salts ---------- 0.7 %

Bile pigment ----------0.2 %

* Cholesterol ----------0.06 %

Inorganic ----------0.7 %

* Inorganic salts----------0.15 %

* Lecithin ----------0.1 %

Fat----------0.1 %

* In solution due to the bile salts

Colour:-

Golden yellow due conjugates of bilirubin.

Tetrapyroles (metals, porphyrins)

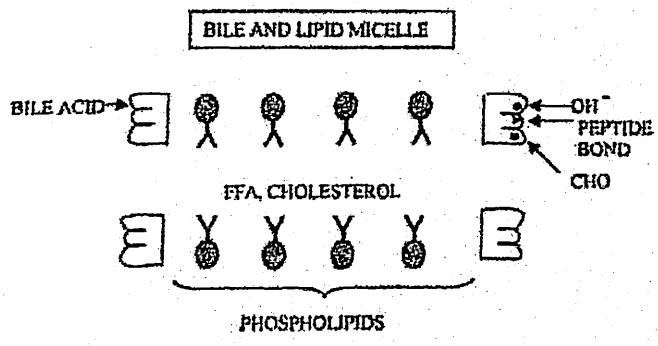
Bile pigments (biliverdin, bilirubin)

PROPERTIES OF BILE SALTS :

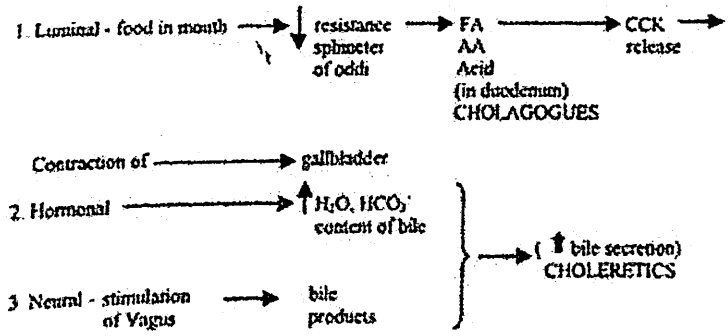
(i) ⬆ surface activity due:
(a) hydrophilic activity group side chain
(b) Lipophilic steroid nucleic

(ii) Amphipathic (i.e has hydrophobic and hydrophilic domains)

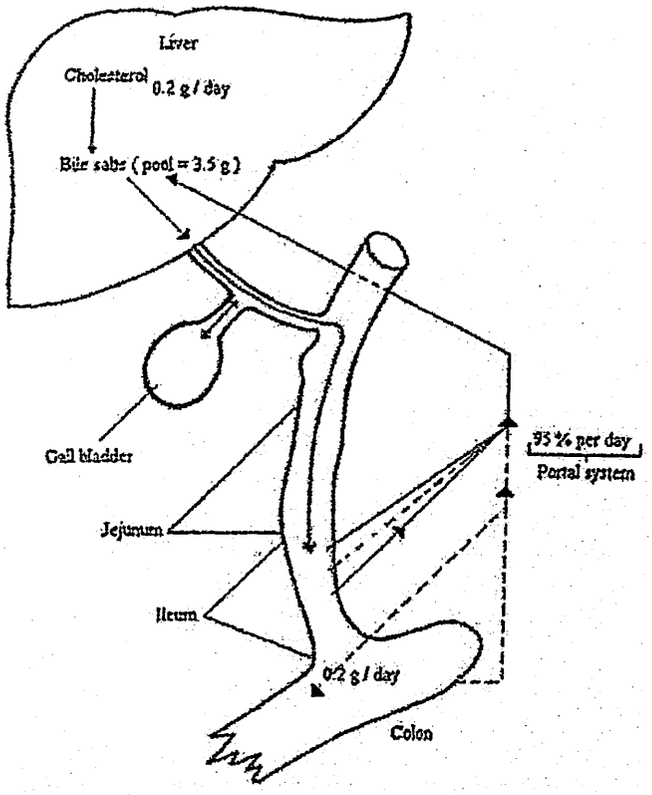
(ii) From micelle which has hydrophobic core and hydrophilic surface ⟶ Lipids in solution which are transported to the brush border of the intestines.



Regulation of biliary secretion:



Reabsorbed bile acids inhibit new bile secretion



Enterohepatic circulation of bile salts. The solid represent bile salts of hepatic origin, whereas the dashed lines represent bile salts resulting from bacterial action.

Cholelithiasis - Gallstones 2 types:

i) Ca++ bilirubinate stones

ii) Cholesterol stones

Predisposing factors:

i) Bile stasis

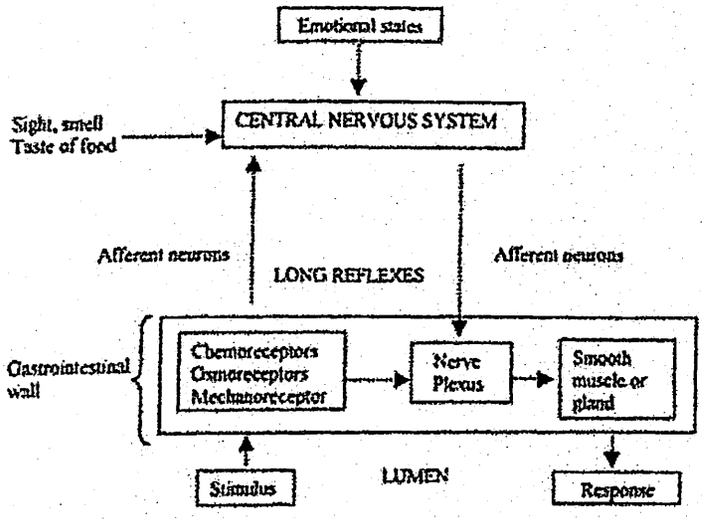
ii) Supersaturation with cholesterol (crystals + micells)

iii) mixture with nucleation factors (in supersaturated bile)

Neural regulatory mechanisms:

i) Enteric nervous system or short reflexes (local).
Involves the myenteric or submucous plexuses or both.
Can be influenced by the ANS

ii) Autonic Nervous System (ANS). Mediated via parasympathetic and Sympathetic system → CNS → motor
Influence
Secretory activity


Emotional states
CENTRAL NERVOUS SYSTEM
Sight, smell
Taste of food
Afferent neurons
LONG REFLEXES
Afferent neurons
Gastrointestinal wall
Chemoreceptors
Osmoreceptors
Mechanoreceptor
Nerve Plexus
Smooth muscle or gland
Stimulus
LUMEN
Response

Modul 4 Motility

Function : Movement of food from the mouth through

(1) The Oesophagus

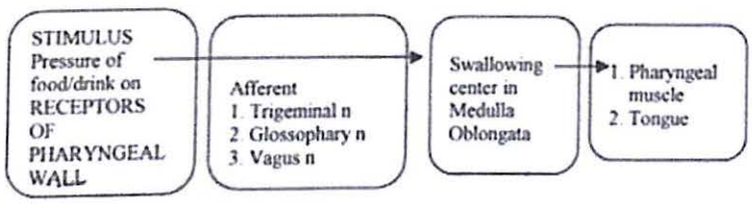
(2) Stomach

(3) Intestines (small and large) to the anus (Defaecation)

Movement Through The Oesophagus

(A) Swallowing (Deglutition)

Is a complex reflex



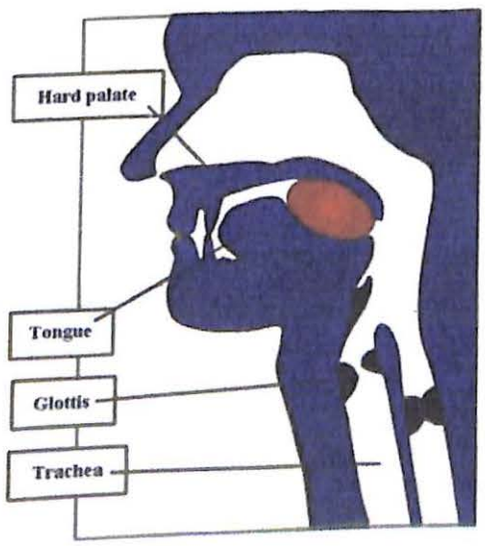
PHASE:

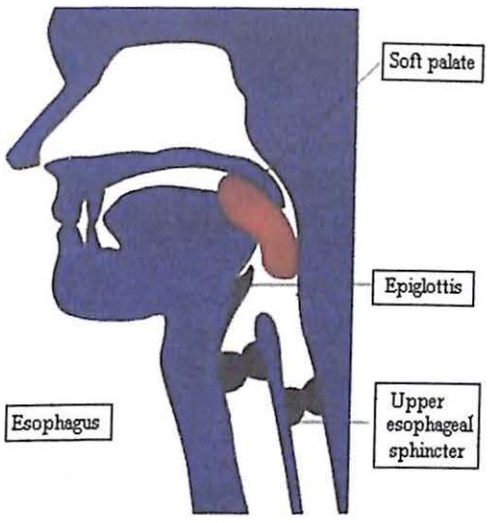
a) Voluntary

b) Involuntary

Voluntary phase

1. Elevation of soft palate ⟶ closure of nasopharyngeal passage

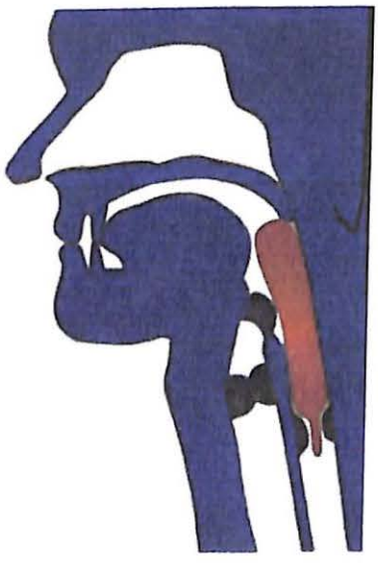




2. Pressure of food on pharyngeal wall ⟶ Receptor stimulation ⟶ impulses to swallowing center

(i) Inhibition of respiration
(ii) Raising the larynx
(iii) Closure of glottis

3. Tilting of epiglotis backwards as food is force backwards.



4. Involuntary phase

1. Relaxation of upper sphincter

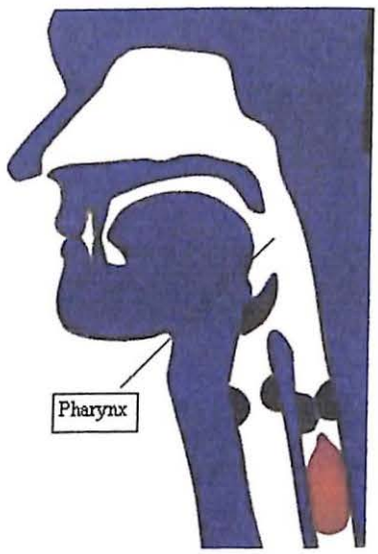
2. Food passes followed by closure

3. Glottis opens,breathing resumes

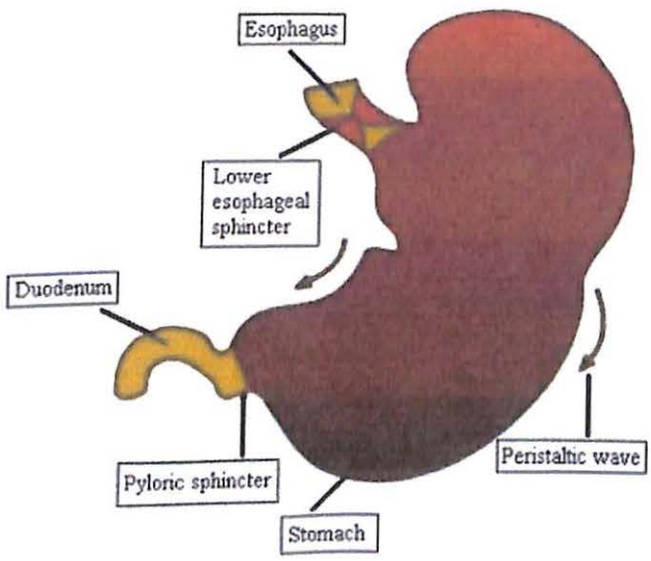
4. Food moved forward by PRESTALTIC WAVE

5. Lower sphicter opens

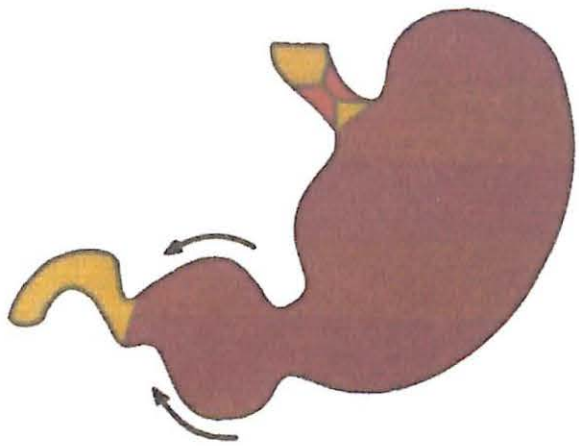
6. Food into stomach followed by sphincter closure.



Function : Mixing of food with juices and grinding ⟶ CHYME



1 Distension of stomach wall initiates peristaltic contraction of body stomach ⟶ antrum



2 ⬆ antral contraction ⟶ closure of pyloric sphincter

Modul 5 Movement of Small Intestines

Function : Mixing of chyme with:
(i) Digestive juices
(ii) Exposure of digested products to large absorptive surface
(iii) Propulsive movement ⟶ aboral passage of chyme

Stimulus : Distention (stretch) of small intestines ⟶ contraction (mixing and propulsion).

Control of Small Intestinal Motility

The contractions are myogenic in origin and are influenced by:

A. Intrinsic plexus

B. Extrinsic plexus

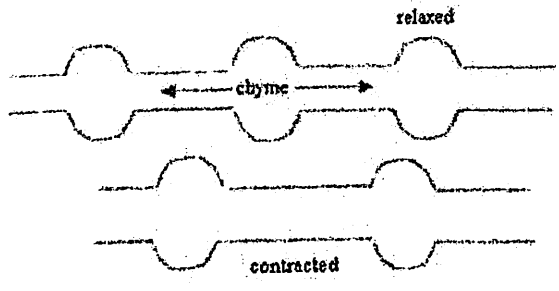
C. Hormones

MYOGENIC ELECTRICAL ACTIVITY
An autonomous basal electrical rythmn (BER) originate from the muscle wall.

(i) Slow oscillatory wave Generator potentials ⟶ spike potentials ⟶ contraction
(ii) Slow waves originating in the longitudinal muscle fiber ⟶ circular muscle fiber ⟶ circular muscle fiber ⟶ myenteric plexus
(iii)Slow waves follow a frequency gradient. (highest in duo 12-14 impulse/min.)
(iv) Vagotomy – no effect on autonomous basal electrical rhythm.

Rhythmic Segmentation

Contraction occurs rhythmically in functional segments of small intestine. In between rings the intestine ballons out. Moments later the ballooned zones contracted zones relax ⟶ mixing mainly.



These contraction are weaker than peristaltic contractions and are dependent (mainly) on the myenteric plexus (partially blocked by atropine)

Factors affecting it :

I Unaffected by denervation of extrinsic autonomic nerves

i. Parasympathetic activity ⟶ (+) Increase in contractions
ii. Sympathetic activity ⟶ (-) inhibition of contraction
iii. Contraction ↑ lymph flow

Peristaltic Movement

Mechanoreceptors present in mucosa are stimulated by stretch (or irritants) and are probably sensitized by locally present 5-HT or subt. P. (modulators)

Chyme (bolus) initiates wave of contractions which maybe preceded by a wave of relaxation (usually travels only a few cm. (Velocity 1-5 cm/sec) ; faster in proximal than in terminal intestines). Small intestine transit time 3-10 hr.

Peristalsis is dependent on the intrinsic myenteric plexus and is abolished by atropine, removal of the mucosa, or degeneration of the plexus.

MECHANISM OF PERISTALSIS

1. Entry of chyme into duodenum ⟶ distension ⟶ peristalsis/ rhythmic contraction.
2. Gastroenteric(gastro-ileal) reflex occurs with entry of food into stomach. Trigger; by distension of the stomach.

Neural Reflexes of Peristaltic Movement

Impulses of this reflex are conducted along intrinsic plexus ⟶ motility of the small intestine and may propel chyme through the ileo-caecal valve into caecum.
In case of peristaltic wave sweeps rapidly over large part of small intestine ⟶ colon : induced by irritants or distension.

Control of Small Intestinal Motility

The contraction are myogenic in origin and are influenced by:

A. Intrinsic plexus

B. Extrinsic nerves

C. Hormones

Intrinsic plexus

Rhythmic segmentation and peristaltic contraction are dependent (mainly) on local myenteric reflexes.The myenteric plexus mediates 4 types of impulses.

a. Cholinergic excitatory (mediator ACh)

b. Non-cholinergic exitory (mediator?5HT,?sub.P-)

c. Adrenergic inhibitory (mediator Nor adrenaline)

d. Non-adrenergic inhibitory (mediator ATP)

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Menu Utama

SISTEM ENDOKRIN

Modul 1 Hypothalamic hormones and factors regulating adenohypophysial function

Hormone	Predominant site in hypothalamus	Target pituitary hormone
Thyrotropin releasing hormone (THR)	Anterior perventricular	Thyrotropin (TSH) Prolactin Growth hormone (by Pathologically high levels of THR)
Gonadotrpin releasing hormone (GnRH)	Arcuate	Lutenization hormone (LH) Follicle stimulating hormone (FSH) Growth hormone (by pathologically high levels of GnRH)
Corticotropin releasing hormone (CRH)	Anterior perventricular	Adenocorticotropin (ACTH) Beta and gamma Lipoprotin Beta endorphins
Growth hormone releasing hormone (GHRH)	Arcuate	Growth hormone
Growth hormone inhibiting hormone (somatostatin)	Anterior perventricular	Growth hormone Prolactin Thyrotropin (TSH) Adrenocorticotropin
Prolactin inhibiting factore (PIF)	Arcuate	Prolactin Thyrotropin Growth hormone (at pathologically high levels of PIF)
Prolactin releasing factor (PFR)	Not known	Prolactin

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Modul 2 Caracteristics of hypothalamic releasing hormones

Secretion in pulses
Action on specific membrane receptors
Transduction of signals through calcium, membrane phospolipids and cAMP
Stimulation of release of stored hormones via exocytosis
Stimulation of synthesis of target hormone at transcription level
Modification of target hormone at post translation stage eg. Glycosylation
Stimulation of hyperplasia and hypertrophy of target cells
Modulation of effects by up-or down-regulation of their own receptors

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Modul 3 Anterior pituitary.

Cell (Cell population)	Hormones	Hormone characteristics	Main functions
Corticotroph (15 - 20%)	Adrenocorticotropin Beta lipotropin	A polypeptide hormone derived from a larger polypeptide molecule that also give rise to lipotropins and melanocyte stimulating hormone. Cells are mostly in the pars distalis of the pituitary	ACTH-Stimulates glucocorticoid and sex hormone secretion from the adrenal cortex Lipoprotein-weak lipolytic and opioid actions
Thyrotroph (3 - 5%)	Thyrotropin	A glycoprotein containing two subunits, alpha and beta. Alpha subunit is found in LH and FSH also. Cells are found mainly in the anteromedial part of the gland	Stimulation of all aspects of thyroid function : hormone synthesis, hyperplasia, hypertrophy, vascularisation
Gonadotroph (10 - 15%)	Luteinizing hormone Follicle stimulating hormone	Glycoprotein hormones with similar structures. Cells are scattered all over the gland and most secrete both hormones	LH-stimulates hormone production in Leydig cells, theca cells FSH-stimulates granulosa cells Sertoli cells
Somatotroph (40 - 50%)	Growth hormone	A single chain polypeptide hormone	Stimulates the production of IGFs and has direct actions on growth and metabilosm
Mammotroph (10 - 25%)	Prolactin	A single chain protein hormone. The percentage of mammotrophs increase during pregnancy lactation and oestrogen therapy	Stimulates milk production and inhibits gonadotropin secretion

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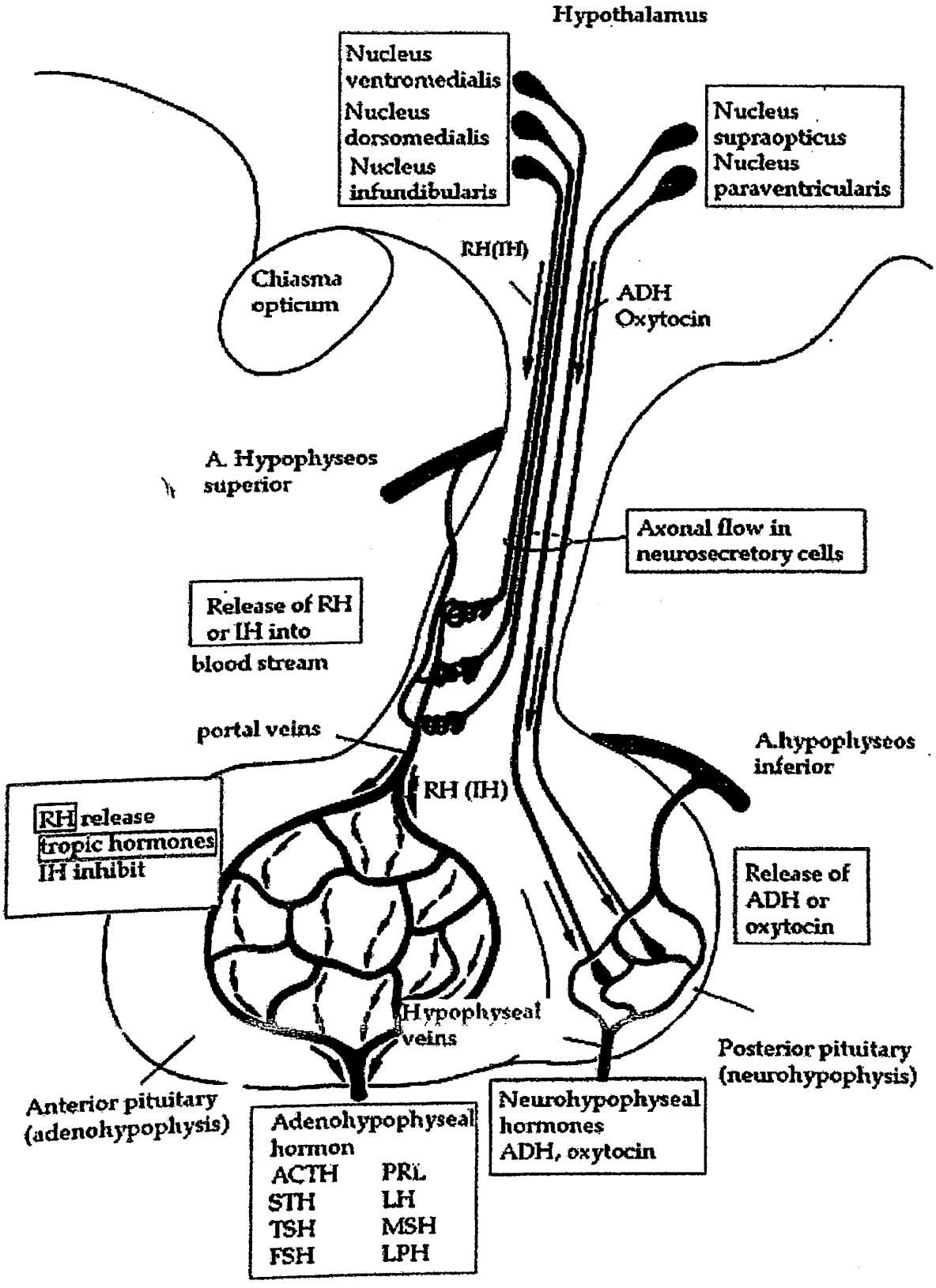
Modul 4 Hypothalamic and pituitary hormones

RH = releasing hormone
IH = inhibitory hormone



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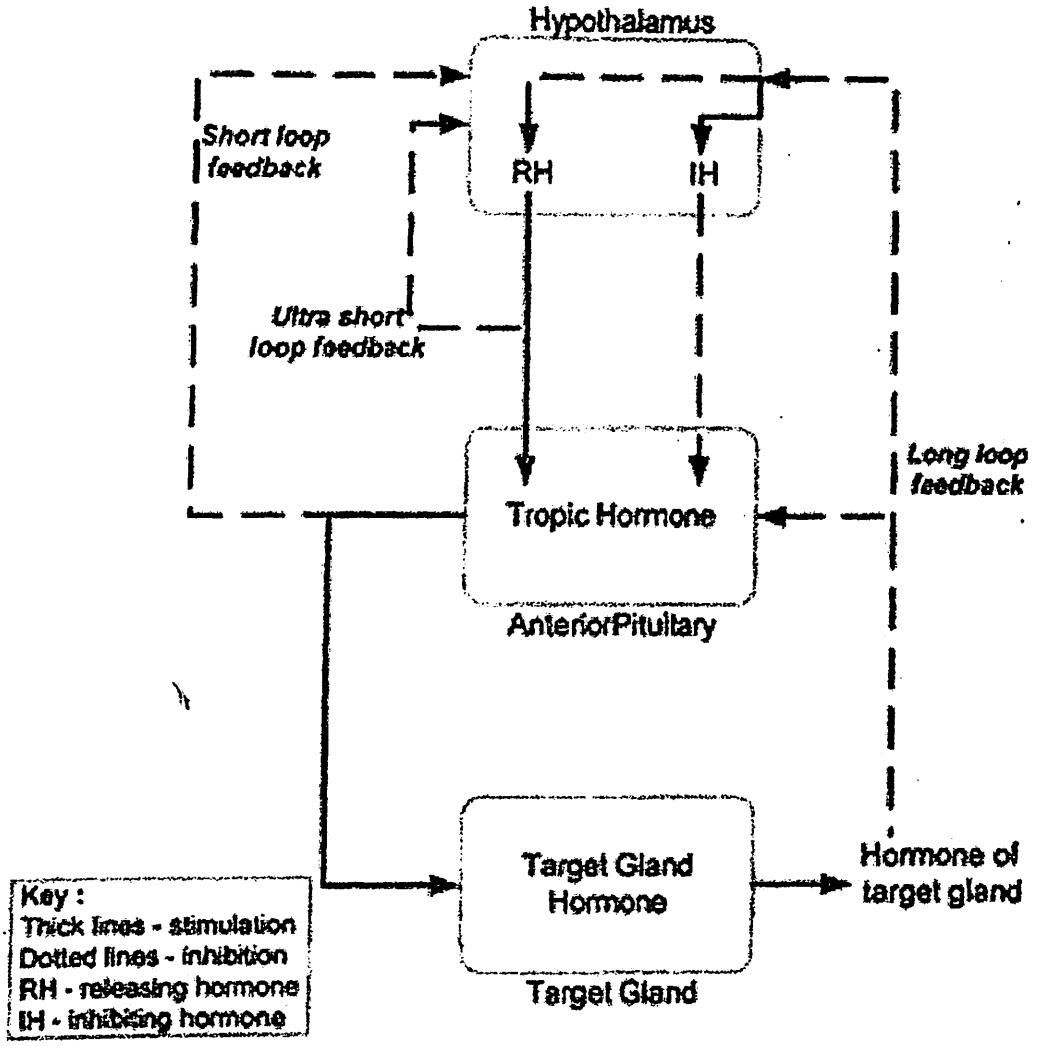
Modul 5 The typical arrangment of negative feedback in the regulation of a Hypothalamus-Pituitary- target gland axis



Exercise :

1. Name the relevant hormones involved in the feedback loop for each of the target glands under anterior pituitary control.
2. There are some execption to this pattern. For example with some hormones the long loop feedback becomes positive feedback under certain circumstances. Can you think of these hormones and the circumstances?

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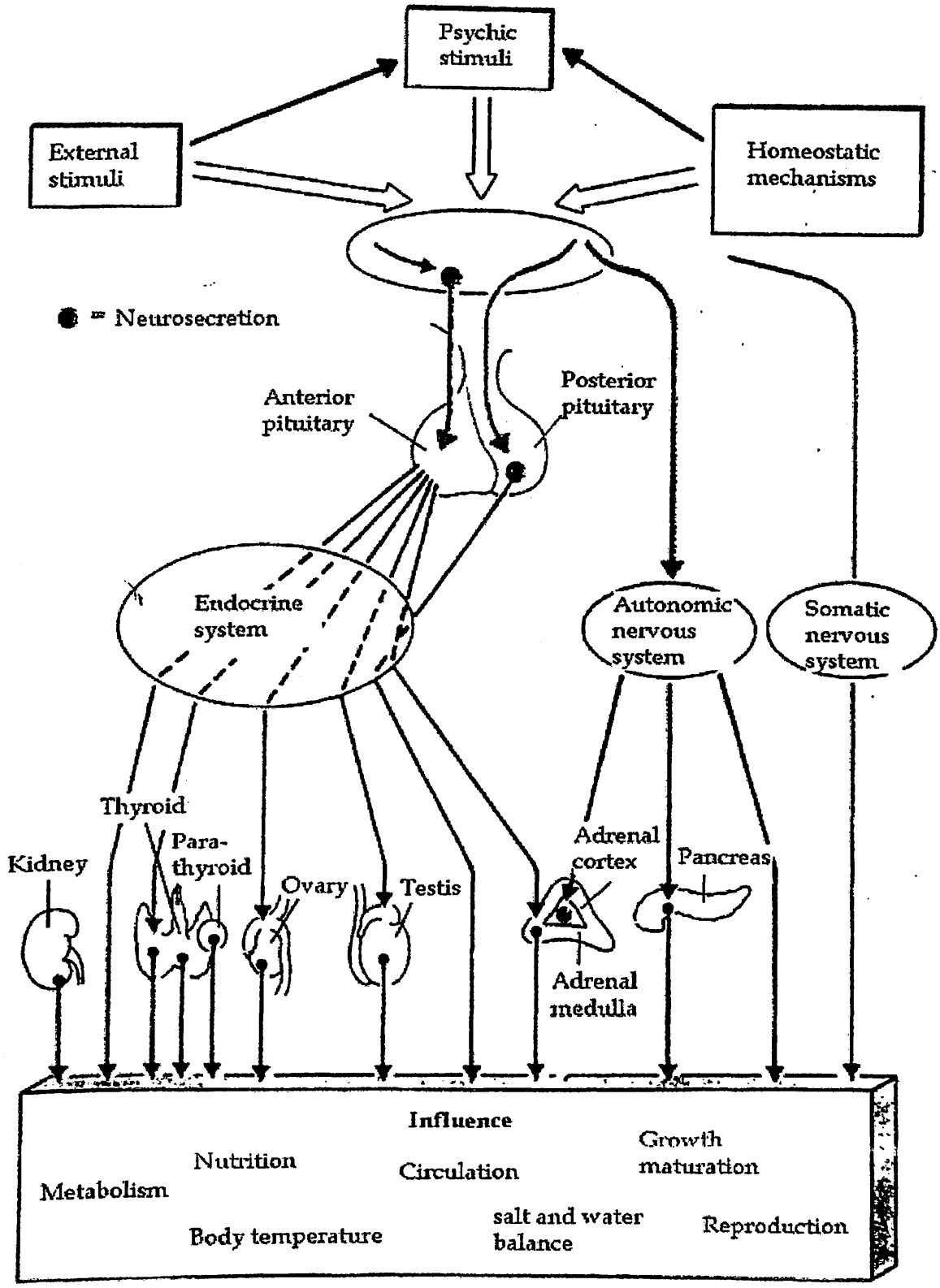
Modul 6 Neural and endocrine interrelation



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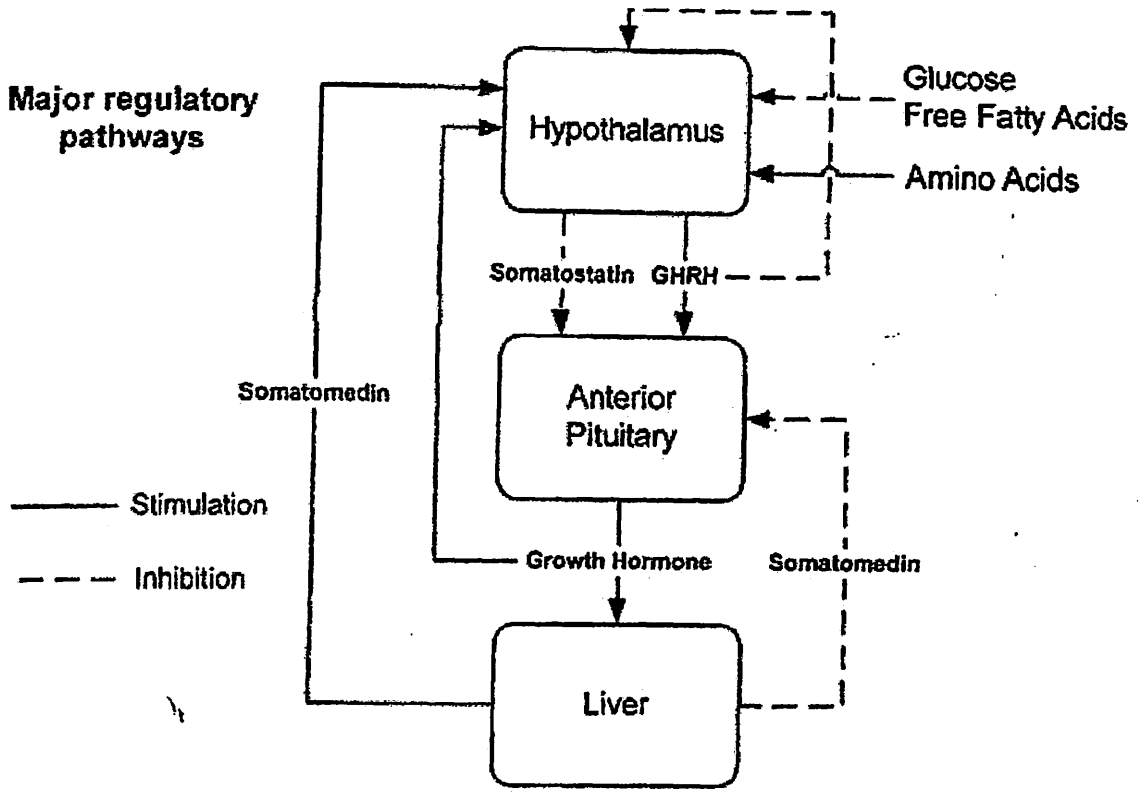
Modul 7 Regulation of growth hormone secretion



Factor influencing growth hormone secretion

Stimulated by	**Inhibited by**
GHRH	Somatostatin
Hypoglyceamia	Hyperglycaemia
Reduction in free fatty acids	Excess free fatty acids
Rise in amino acids (arginine)	Somatomedians
Fasting	Growth hormone
Starvation	Beta adrenergic agonists
Stage IV sleep Exercise	Cortisol
Stress	Ageing
Puberty	Obesity
Oestrogens	Pregnancy
Andorgens	
Dopamine	
Acetylcholline	
Serotonin	
Alpha adrenergic agonists	
Gammaamino butyric acid	
Encephalins	

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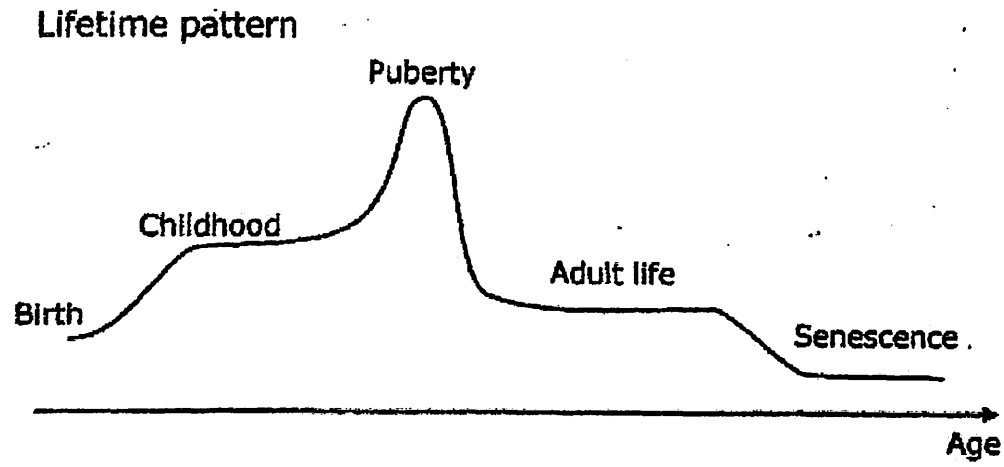
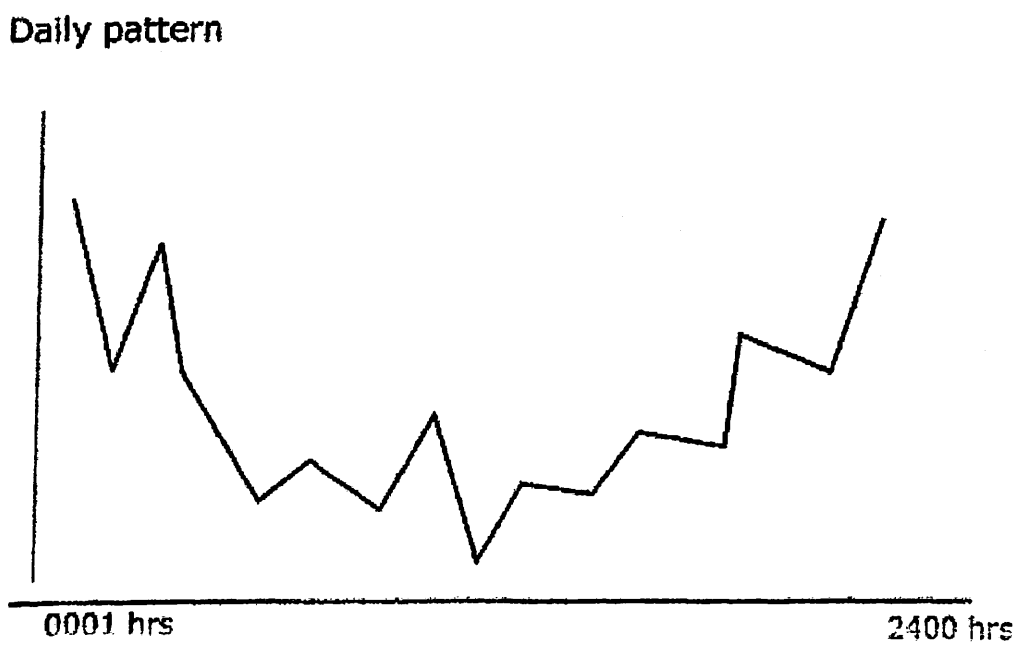
Modul 8 Patterns of growth hormone secretion





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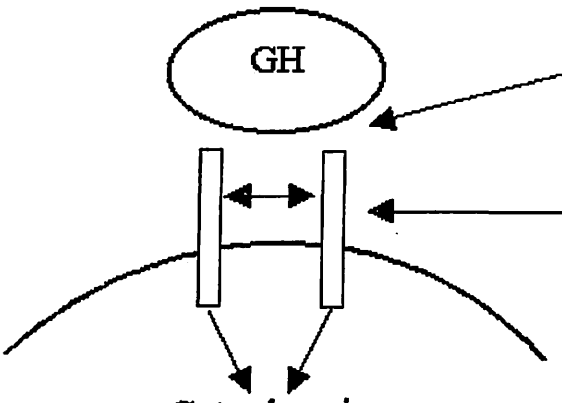
Modul 9 Mechanism of growth hormone action



1. One GH molecules binds to two adjacent receptors

2. This causes dimerzation of two receptors

Cytoplasmic Tyrosine Kinase

3. This result in ativation of the receptors which in turn activate tyrosine kinase

Phosphorylation of substrates

4. The next step is phosphorylation of substrates

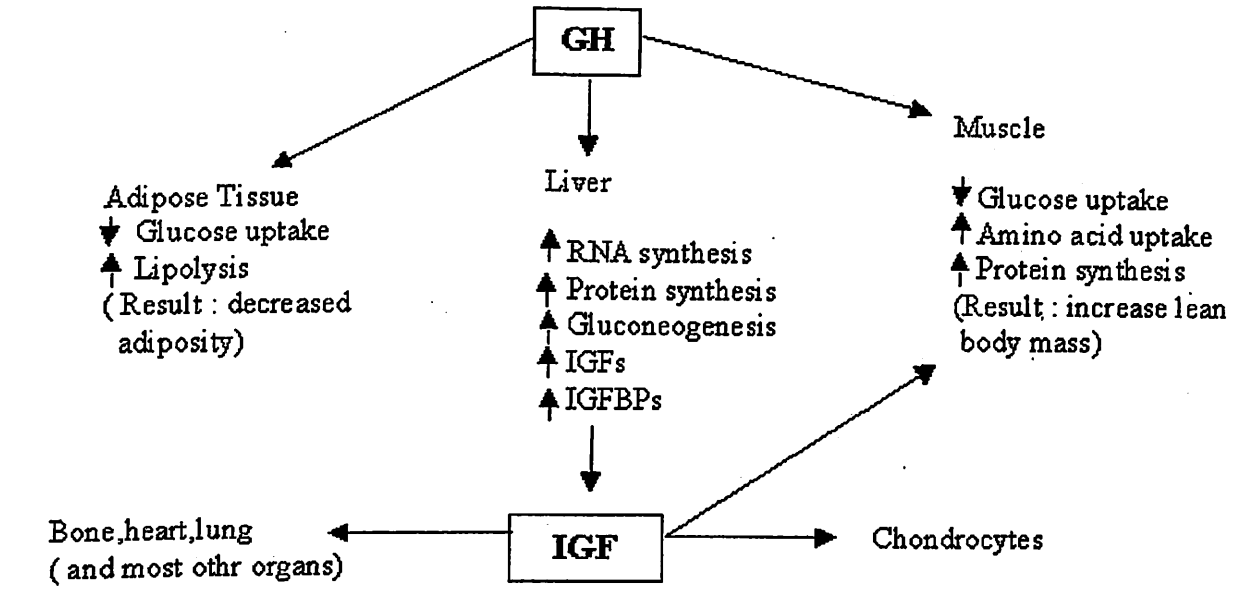
Activation of transcription factors

5. This result in activation of transcription factors

Modulation of gene expression

6. These factors modulate target genes eg IGF gene

Actions of growth hormone



- Protein synthesis
- RNA synthesis
- DNA synthesis
- Cell size and number

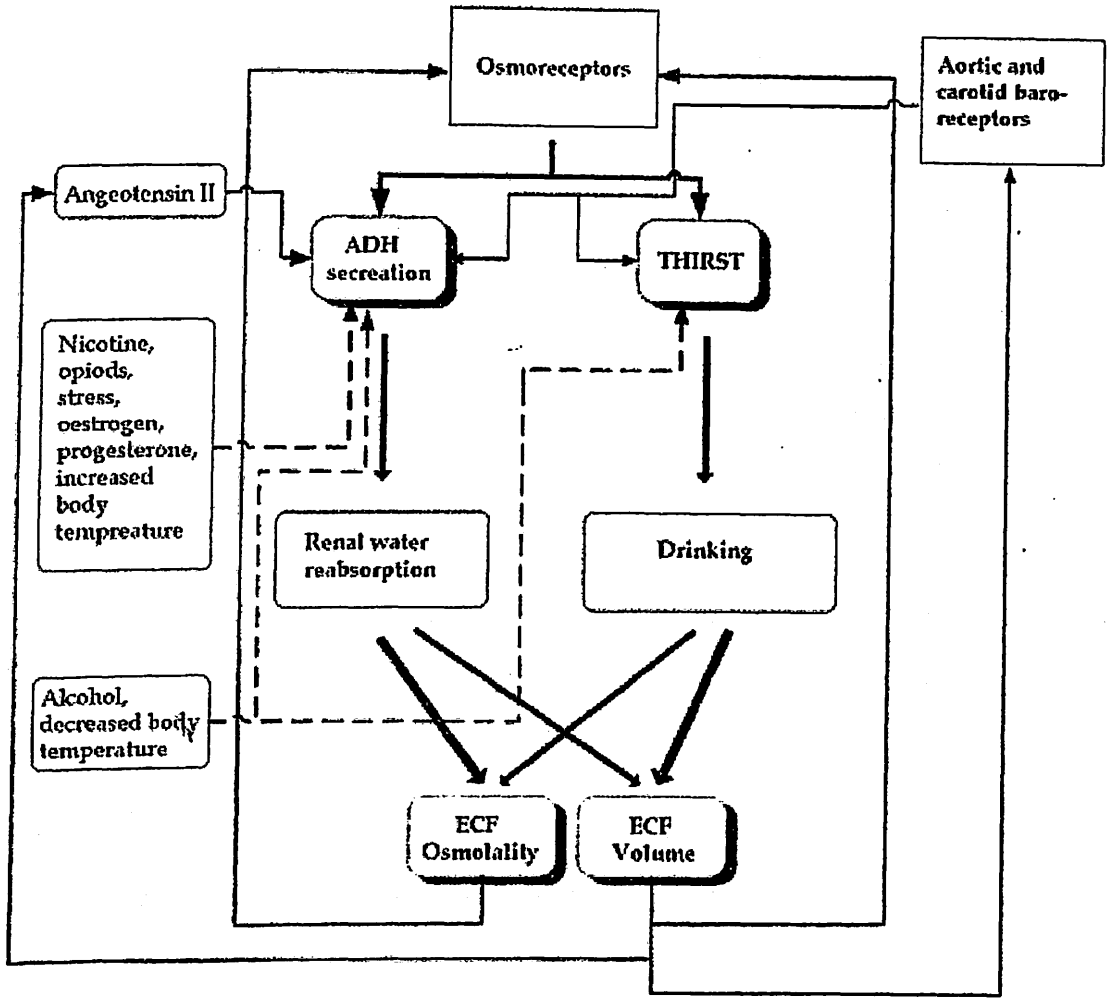
(Result : increase organ size and function)

- Amino acids uptake and protein Synthesis
- RNA and DNA synthesis
- Collagen
- Chondrotin sulphate
- Cell size and number

(Result : increase linear growth)

SISTEM ENDOKRIN

Modul 10 Regulation of Antidiuretic hormone secretion and thirst



The major regulatory mechanisms are in thick lines
Thin lines show regulatory mechanisms that are of second importance
Dotted lines are incidental factors that influence the ADH secretion (not important for day to day regulation)
Whether each factors/parameter stimulates or inhibits the subsequent factors is not indicated in this chart

Can you determine the type of influence (stimulatory or inhibitory) at each arrow