LAPORAN AKHIR GERAN INSENTIF

NO GERAN:

USM/PPSP®/Ger.Peny.(15)

CAFLM
(COMPUTER AIDED FIXED LEARNING MODULE)

MOHAMAD SALLEH ABD HAMID

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

Bahagian II

Hasil Projek CAFLM

1. Sistem Pernafasan
2. Sistem perkumuhan
3. Sistem Penglihatan

BAHAGIAN I

LAPORAN

- BORANG LAPORAN AKHIR PROJEK PENYELIDIKAN JANGKA PENDEK

- LAPORAN AKHIR

1. Pengenalan
2. Objektif & Kaedah
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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:** Mohamad Salleh Abd. Hamid

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

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

3) **Tajuk Projek:** Penyediaan Modul 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 Module) telah dijadikan bahan untuk dimasukkan ke dalam program CAFLM (Computer Aided Fixed Learning Modul) 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 seperti Sistem Pernafasan, Sistem Pencernaan dan Sistem Penglihatan. 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 dimasukkan ke dalam komputer.

1. Sistem pernafasan
2. Sistem penglihatan
3. Sistem perkumuhan

(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:**

Scanner (Acer 3300U) - RM300.00

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

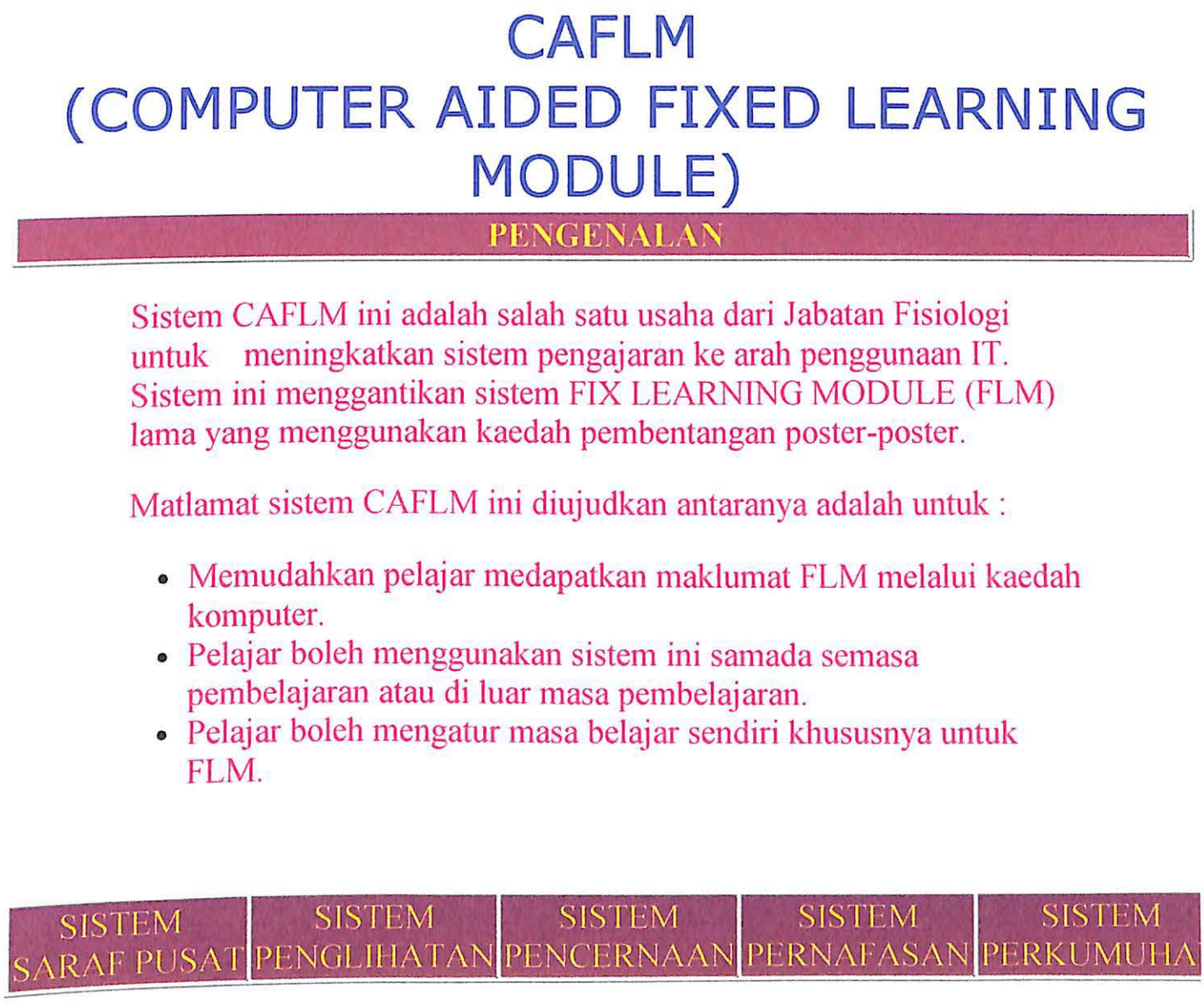
1. Mewujudkan satu sistem FLM yang lebih teratur.
2. Membantu menambahbaikan sistem pembelajaran FLM secara komputer.
3. Mengurangkan kos perbelanjaan bagi penyediaan FLM.

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.



SISTEM PERNAFASAN

Modul 1 Respiratory Failure.
Modul 2 Ventilation Of The Lungs.
Modul 3 Respiratory Airways.
Modul 4 What is cough?
Modul 5 Respiratory control
Modul 6 Control of respiration.
Modul 7 Postoperative Hypoxaemia.
Modul 8 Fetal Respiration.
Modul 9 Physiopathology Of Obesity In Relation To Respiratory Physiology.
Modul 10 Acute Pulmonary Embolism.
Modul 11 Acid-base disorders.
Modul 12 Clinical Signs Of Hypoxia.
Modul 13 Chronic Obstructive Pulmonary Disease And Chronic Hypoventilation.
Modul 14 Pleural Fluid, Pulmonary Interstitial Fluid, Pulmonary Edema.
Modul 15 Respiration.
Modul 16 The Pulmonary Circuit: Some Basic Physiologic Considerations.
Modul 17 Pulsus Paradoxus Is A Valueable Clinical Sign In Severe Asthma.
Modul 18 The Lung.
Modul 19 Behavioral Distribution Of V/Q Inequality.

SISTEM PERKUMUHAN

Modul 1 Physiology of the kidney and body fluids.
Modul 2 Countercurrent Multiplication by the Loop of Henle.
Modul 3 Countercurrent exchange by the vesa recta.
Modul 4 Recent modifications of the countercurrent hypothesis by Stephenson and Kokko and Rector.
Modul 5 Action of angiotensin II.
Modul 6 Clearance of inulin.
Modul 7 Urea clearance.
Modul 8 Creatinin clearance.
Modul 9 PAH clearance.
Modul 10 Acidification of urine.
Modul 11 Tubular maximum.
Modul 12 Production of a concentrated urine.
Modul 13 Overview of renal system.
Modul 14 Pathways by which sodium and water excretion are decreased in response to severe sweating.
Modul 15 Pathway by which the GFR is decreased when plasma volume decreases.
Modul 16 Summary of the processes occuring in urine formation.

SISTEM PENGELIHATAN

Modul 1 The eye.

Modul 2 Retina : distribution of rods, cones, dark sensitivity and visual acuity.
Modul 3 Visual adaptation.
Modul 4 Inversion of visual field upon the retina.
Modul 5 Intra ocular lens (IOL)
Modul 6 Eye accommodation.
Modul 7 Visual acuity.
Modul 8 The blind spot.
Modul 9 Visual fields.
Modul 10 Perimetry.
Modul 11 Projection of the different parts of the retina on the cerebral cortex.
Modul 12 Visual pathways as seen from the base of the brain.
Modul 13 Optical defects and refractive anomalies.
Modul 14 Colour vision.
Modul 15 Colour blindness.
Modul 16 Response of the three photopigments to light of different wavelengths.
Modul 17 Accommodation.
Modul 18 Pathway for accommodation reflex.
Modul 19 Movements of the eye.
Modul 20 Diagram of the course of the anatomic fibres to the eye.
Modul 21 The cell of retina.
Modul 22 General character of human aqueous homur(Expressed Relative to Plasma)
Modul 23 The aqueous humor.
Modul 24 Light reflex.
Modul 25 Diagrammatic representation of the cells in the visual pathways.
Modul 26 Structurs of photoreceptors.
Modul 27 The cell of retina.
Modul 28 Electrical activity of retina.
Modul 29 Recoding of the activity of a single ganglion cell.
Modul 30 Responses of a concentric cell in the retina to a monochromatic stimulus.
Modul 31 Light stimulus.
Modul 32 Receptivefield.
Modul 33 Retina, Control of eye movements, Fondues oculi & Iris, lens and ciliary body.
Modul 34 Organization of the visual field.
Modul 35 Highly schematic diagram of the visual projections to the cortex.
Modul 36 Projection of the retinas upon the lateral geniculate nucleus.
Modul 37 The intraocular fluid.
Modul 38 Intraocular pressure.
Modul 39 Optical defects and refractiue amomalies.
Modul 40 Eye and the retina.
Modul 41 Physiology of the central visual pathway receptive fields.
Modul 42 Visual field projects upon the retina normal.
Modul 43 The responses of retinal ganglion cells to colored light.

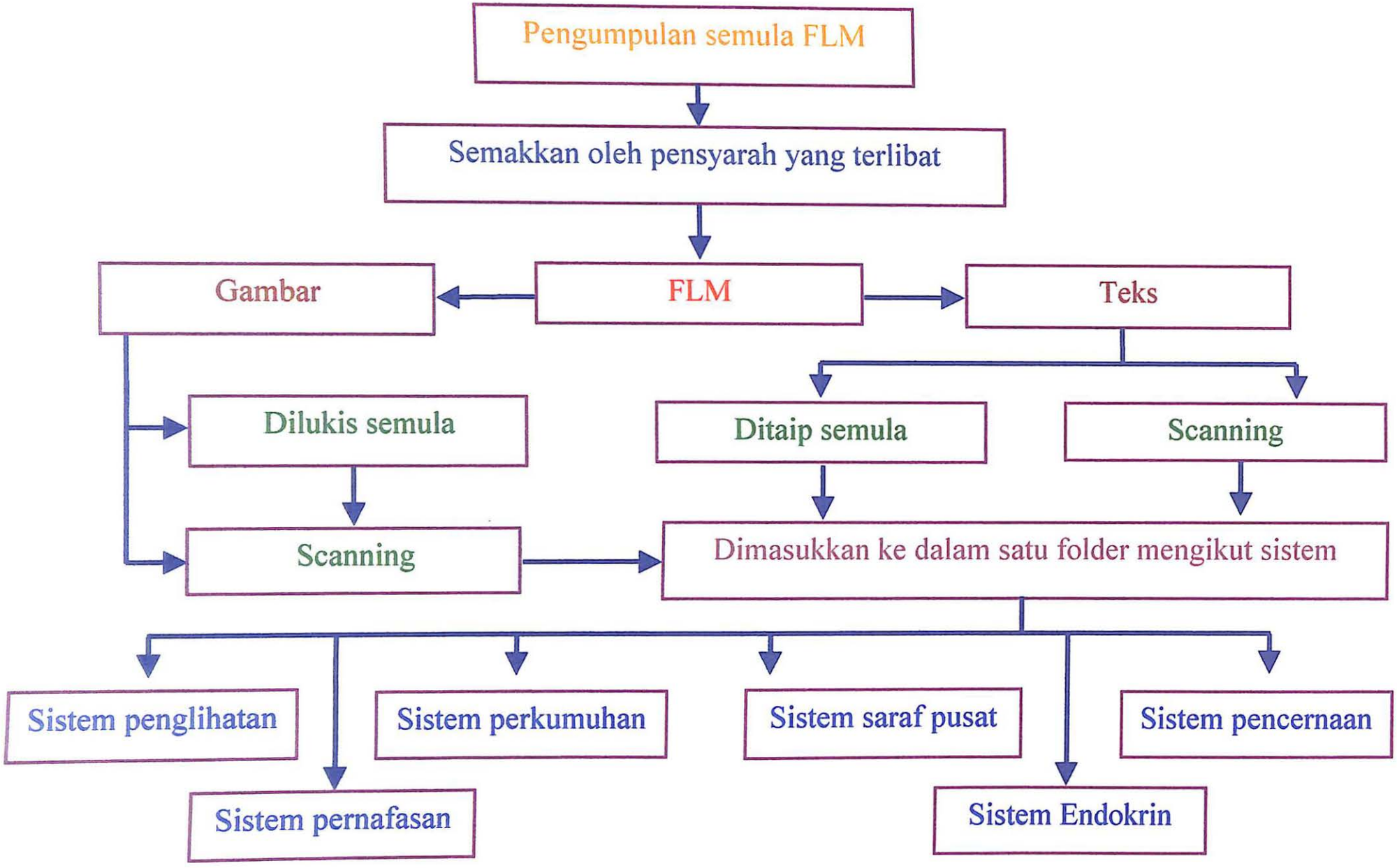
PERBINCANGAN

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 Pernafasan
- ❖ Sistem Perkumuhan
- ❖ Sistem Penglihatan

SISTEM PERNAFASAN

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Menu Utama

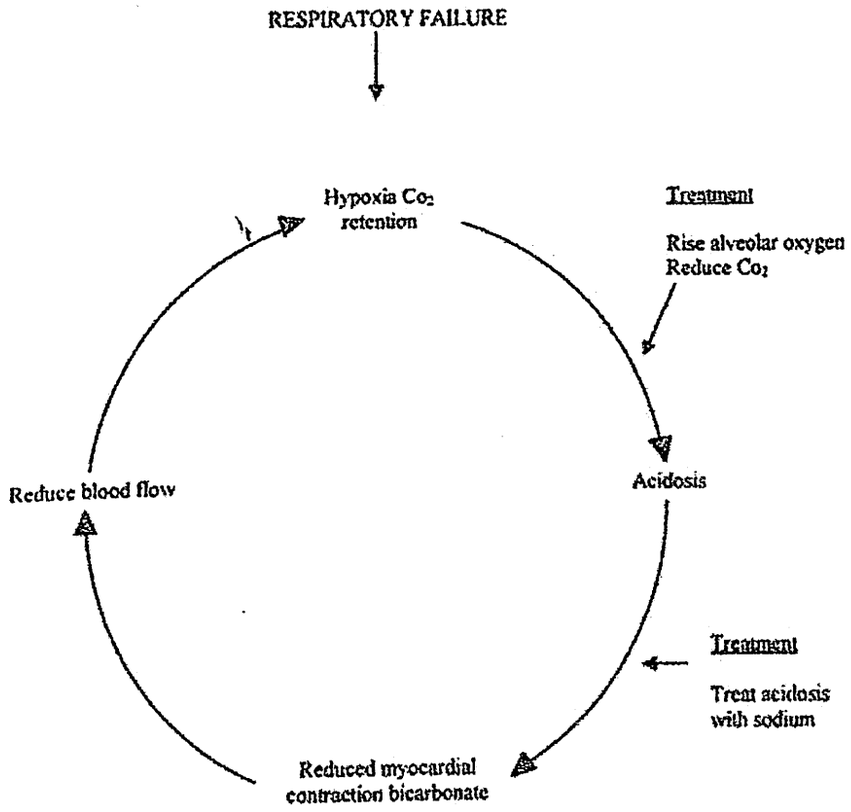
SISTEM PERNAFASAN

Modul 1 Respiratory Failure.

Effect Of Hypoxia And Hypercapnia

Respiratory failure from any cause result in hypoxia and possibly carbon dioxide retention. The effects of the latter are not as serious as hypoxia, although the respiratory acidosis produced will further aggravate hypoxia. If this persists, a chain of events will ensure, which, if not broken, will result in death. Fortunately, most organs can function as long as the.. oxygen saturatio does not fall below 50%, but below this level cerebral, renal and hepatic failure result. Hypoxia must be treated urgently and patients on the brink of respiratory failure treated energetically and observed meticulously.

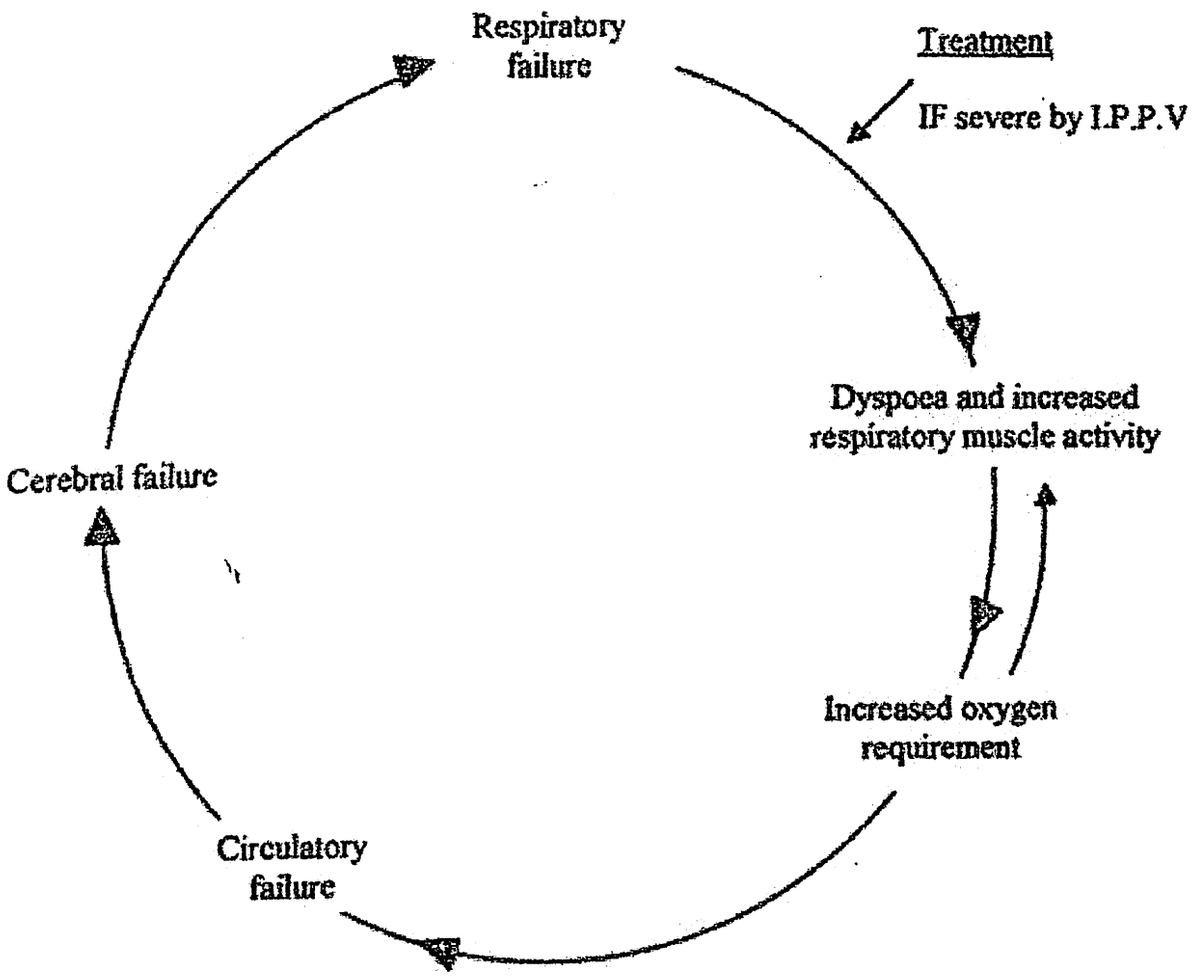
The consequence of hypoxia is metabolic acidosis. Anaerobic metabolism of pyruvic acid to produce energy allows the accumulation of lactic acid. This acidotic state leads to reduced cardiac contraction and reduced blood flow. This in turn leads to further hypoxia. The respiratory acidosis from carbon dioxide retention will be a further strain on any compensating mechanisms. It can be seen that not only will oxygen be required, but if the hypoxia has existed for any length of time, sodium bicarbonate or other chemical buffer will be required to neutralise the acids. If untreated this cycle of events will end fatally.



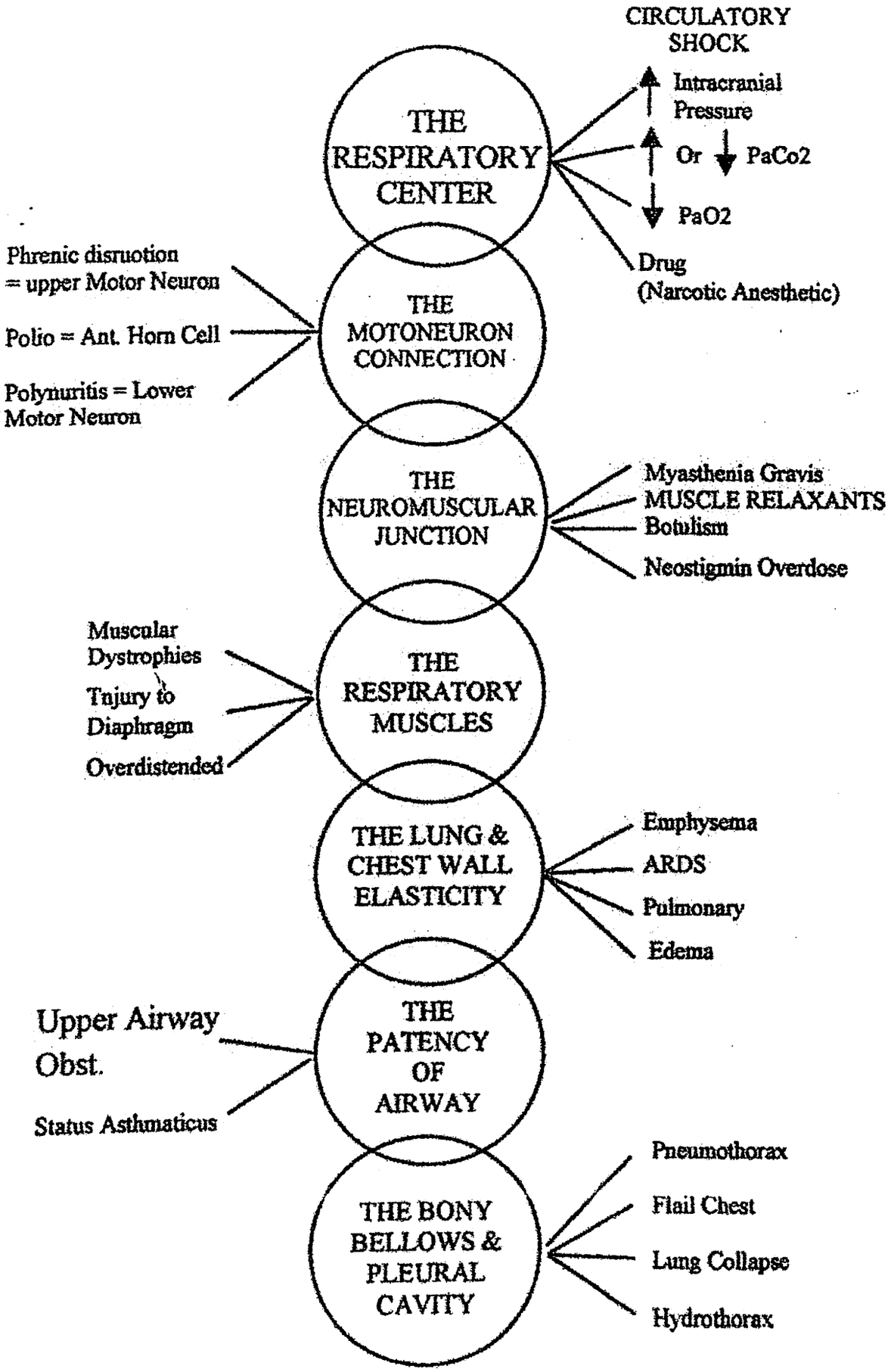
Ventilation In Excess Of Perfusion

In this case, blood gases approach alveolar gas concentration. Oxygen tension cannot be raised without adding oxygen to the inspired mixture as the blood will be saturated for that pressure of oxygen. The carbon dioxide may be normal or possibly raised. This situation does not occur in isolation clinically. It is however, present in addition to ventilation defects, for example in pulmonary embolus and emphysema. Added oxygen in metered concentration is of help, but if the condition is severe, ippv will be required.

It is important to remember that the cause of respiratory failure may be multi-factorial. Blood gases are the best guide to the line of treatment required. For example if the pCO2 low, added oxygen in carefully measured amounts is all that is required provided that the patient is not fatiqued. The increase work called upon by respiratory distress increases the oxygen requirements and puts an extra load on the respiratory and circulatory systems. This work is required to overcome resistance of narrow airways, lung stiffness and mucus obstruction. This work load, which may be responsible for 40-50% of oxygen consumption, will setup a cycle of events which may only further aggraviate the situation.



The interconnected anatomic-physiologic links that are necessary for maintenance of normal alveolar ventilation and some diseases affecting them that can cause acute respiratory failure.


CIRCULATORY SHOCK
Intracranial Pressure
Or
PaCo2
PaO2
Drug (Narcotic Anesthetic)
THE RESPIRATORY CENTER
Phrenic disruotion = upper Motor Neuron
Polio = Ant. Horn Cell
Polynuritis = Lower Motor Neuron
THE MOTONEURON CONNECTION
Myasthenia Gravis
MUSCLE RELAXANTS
Botulism
Neostigmin Overdose
THE NEUROMUSCULAR JUNCTION
Muscular Dystrophies
Tnjury to Diaphragm
Overdistended
THE RESPIRATORY MUSCLES
Emphysema
ARDS
Pulmonary Edema
THE LUNG & CHEST WALL ELASTICITY
Upper Airway Obst.
Status Asthmaticus
THE PATENCY OF AIRWAY
Pneumothorax
Flail Chest
Lung Collapse
Hydrothorax
THE BONY BELLOWS & PLEURAL CAVITY

SISTEM PERNAFASAN

Modul 2 Ventilation Of The Lungs

Introduction

The primary function of the lungs is to add oxygen to, and remove carbon dioxide from the blood. In order to do this, air in the lungs is equilibrated with blood as the gases diffuse across the alveolocapillary membrane. For efficient gas exchange to take place, adequate ventilation and perfusion are necessary. Ventilation occurs in cycles of inspiration and expiration, air coming in during inspiration and leaving during expiration.

Mechanics Of Respiration

Air moves from a region of higher pressure to one of lower pressure. Therefore, for air to move in and out of the lungs, a pressure difference between the atmosphere and the alveoli must be established.

Under normal circumstances, inspiration is accomplished by causing alveolar pressure to fall below the atmospheric pressure. Atmospheric pressure is conventionally referred to as "0 cm Hg" when we discuss the mechanics of breathing, and so lowering of alveolar pressure below atmospheric pressure is known as negative pressure breathing. [The use of the ventilator is an example pf positive pressure breathing]. Normal, quiet expiration is brought about by elastic recoil of the chest wall.

Generation Of Pressure Ingredients

When the muscles of inspiration contract, the chest wall is expanded, the volume of the alveoli is increased, and the alveolar pressure is lowered [Remember Boyle's Law!]

The pressure that distends the alveoli is called the transmural pressure gradient (transmural = across the wall). This is conventionally calculated by subtracting the outside pressure (the intrapleural pressure) from the inside pressure (the alveolar pressure). (Fig. 1)

The intrapleural pressure (the pressure in the thin space between the visceral and parietal pleura) is slightly subatmospheric, even when no inspiratory muscles are contracting. This negative intrapleural pressure (a.k.a. negative intrathoracic pressure; -3 to -6 cm H_20) is caused by the mechanical interaction between the lungs and chest wall (the lungs exert an inwards-elastic recoil of the alveoli and the chest wall opposes by outward- elastic recoil). The intrapleural pressure thus becomes more negative during inspiration. The transmural pressure gradient is increased, and air flows into the alveoli. [The volume of air flowing in a single breath during quiet inspiration is the tidal volume. This is normally about 0.5 litres, but may vary with age and training].

The intrapulmonary pressure (alveolar pressure), which is equal to the atmospheric pressure at the end of expiration, becomes negative during inspiration; as air flows in, it rises until equilibrium is reached between the alveoli and the atmosphere. As the chest wall recoils during expiration, the pressure rises, forcing air out, until equilibrium is again reached. (Fig. 2).

Inspiratory muscles :

* Diaphragm (75% of tidal volume)
* External intercostals
* Accessory muscles of inspiration (exercise, coughing/sneezing, pathological states e.g. asthma)-sternocleidomastoid

Expiratory muscles :

(passive-elastic recoil)

Active expiration (exercise, speech, singing, coughing, sneezing, pathological states e.g chronic bronchitis)-muscles of abdominal wall, external and internal oblique, transverse abdominis, internal intercostals

Resistances To Airflow

1) Elastic resistance (elastic recoil) - 65%
2) Non-elastic - airway resistance - 28 %
- viscous resistance - 7 %

1) Elastic resistance

* Presence of elastin, collagen etc.
* Surface tension at the air - liquid interface [This is lowered by surfactant]
* Higher resistance allows less expansion of the lungs, and less air entry i.e. the compliance is lower. [Compliance = change in volume per unit change in pressure; about 0.2 l/cm water] . In the absence of resistance, the distance travelled (lung expansion or change in volume) should be proportionate to force (change in pressure), and the work done (ventilation of lungs; WD = force xdistance) should also be proportionate to the pressure changes. However, because of the resistance offered, the rate of increase in lung volume is less. (Fig.3)

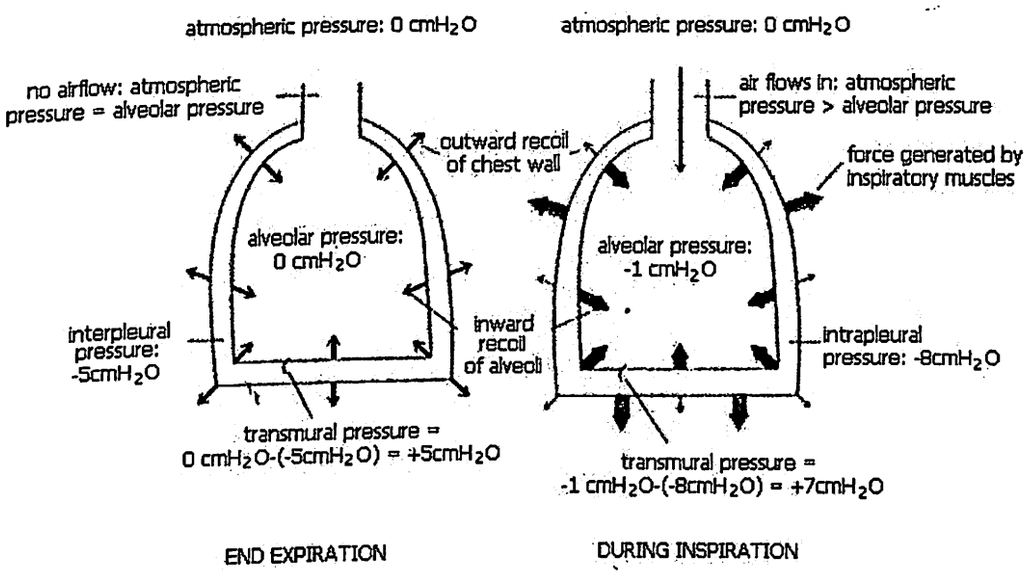


Figure 1 : Representation of the interaction of the lung and chest wall. Left: At end expiration the muscles of respiration are relaxed. The inward elastic recoil of the lung is balanced by the outward elastic recoil of the chest wall. Intrapleural pressure is – 5 cmH_2O; alveolar pressure is 0. The transmural pressure gradient across the alveolus is therefore 0 cmH_2O – (-5 cmH_2O), or cmH_2O. Since alveolar pressure is equal to atmospheric pressure, no airflow occurs. Right: During inspiration, contraction of the muscles of inspiration causes intrapleural pressure to become more negative. The transmural pressure gradient increases and the alveoli are distended, decreasing alveolar pressure below atmospheric pressure, which causes air to flow into the alveoli.

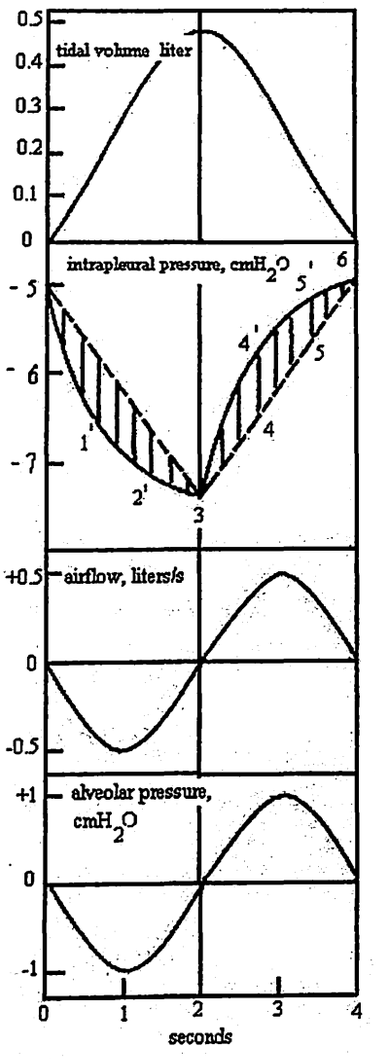


Figure 2 : Volume, pressure and air flow changes during a single idealized respiratory cycle. Described in text. (Reproduced with permission from Comroe, 1974: Physiology of Respiration, 2d ed., Chicago, Year Book Medical Publishers.)

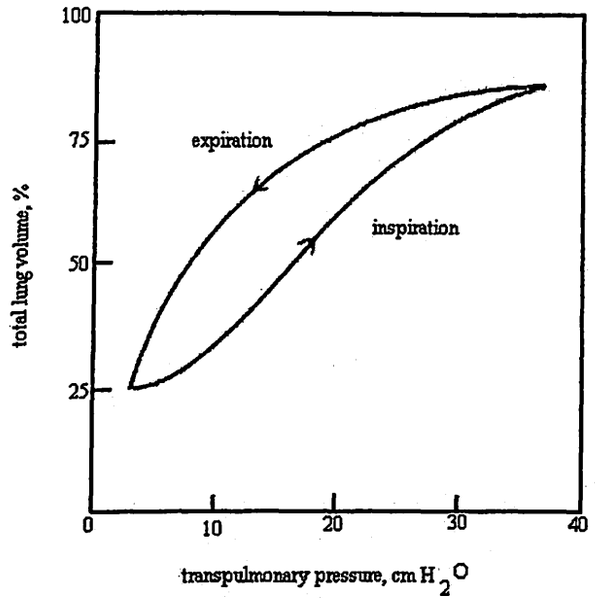


Figure 3 : Pressure-volume curve for isolated lungs.

2) Airway resistance

The inflow of air is resisted by the repiratory tract due to:

(a) the inertia of the respiratory system (negligible)

(b) frictional resistance of the lung - chestwall surface and chestwall = negligible)

(c) frictional resistance of airways (80% of pulmonary resistance)

(d) pulmonary tissue resistance (20% of pulmonary resistance) [increased in pulmonary sarcoidosis and fibrosis]

25 - 40% of airway resistance is contributed by the upper airways (nose, nasal turbinates, oropharynx, nasopharynx, larynx). Resistance is higher when one breathes through the nose than through the mouth.

Although the smallest airways should offer the most resistance due to their smaller diameters, they are arranged in parallel (lower resistance). Thus, under normal conditions, the greatest resistance to airflow resides in the medium- sized bronchioles.

Control Of Bronchial Smooth Muscle

The smooth muscle of the airways (from the trachea down to the alveolar ducts) is under the control of the autonomic nervous system. Parasympathetic cholinergic effects are bronchoconstriction and mucus secretion. Beta adrenergic effects are bronchodilation and reduction of mucus secretion; alpha stimulation causes bronchoconstriction. Under normal conditions, parasympathetic effects predominate. Circulating adrenergic transmitters may also be important in causing bronchodilation.

Table 1: Active Control of The Airways

Constrict	Dilate
Parasympathetic stimulation	Symphathetic stimulation ($beta_2$ receptors)
Acetylcholine	Circulating $beta_2$ agonists
Histamine	Nitric oxide

Leukotrienes
Thromboxane A_2
Serotonin
Alpha-adrenergic agonists
Decreased PCO_2 in small airways
Increased PCO_2 in small airways
Decreased PO_2 in small airways

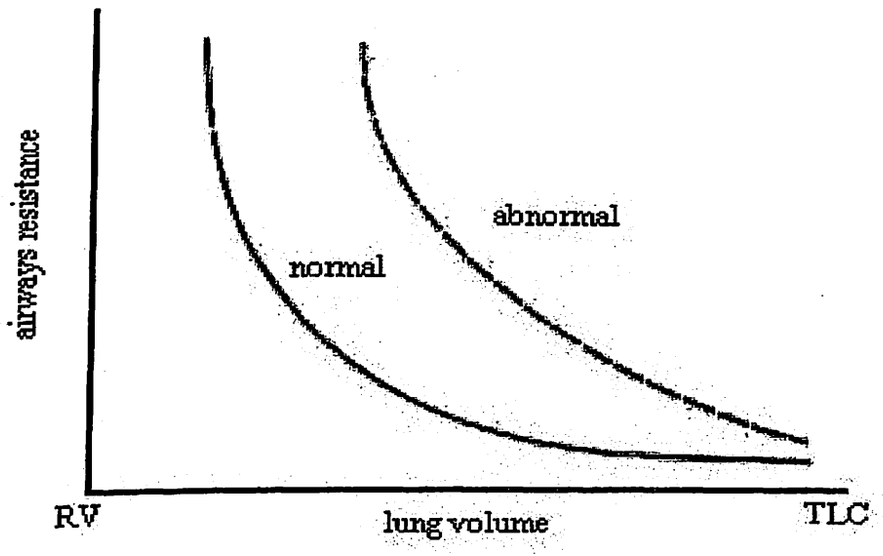


Figure 4 : Relationship between lung volume and airways resistance. Total lung capacity is at right; residual volume is at left. Solid line = normal lung; dashed line = abnormal (emphysematous) lung. (Reproduced with permission from Murray, 1972.)

Many substances can affect the calibre of the bronchioles (Table 1). [Note that histamine and leukotrienes, which are related to infection, cause bronchoconstriction].
Cool air causes bronchoconstriction , which explains bronchoconstriction seen in exercise (exercise - induced bronchoconstriction) and the fact that asthmatic attacks are more frequent in the early mornings. Furthermore, the sensitivity of the bronchioles to the above-mentioned factors is greater in the early mornings than in the evenings.

Lung Volume And Airway Resistance
Airways resistance decreases with increased lung volume (Fig.4).This is because:
(1) inspiration results in lowering of intrapleural pressure, causing the transmural pressure gradient to become more positive; the small airways become distended, and resistance falls [Remember Poiseulle's Law, which relates resistance to the fourth power of the radius!]
(2) of the traction on the small airways by the alveoli (Fig.6).

Air Turbulence
Airflow in the respiratory tract is laminar. However, turbulences may be set up whenever the incoming air meets with a constriction, or a sudden increase in calibre. Turbulent airflow will also resist the incoming of air. (Fig.5)

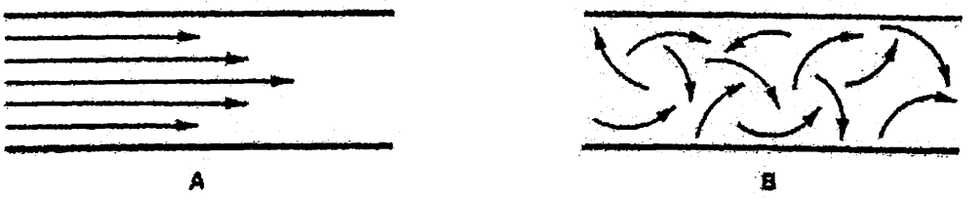
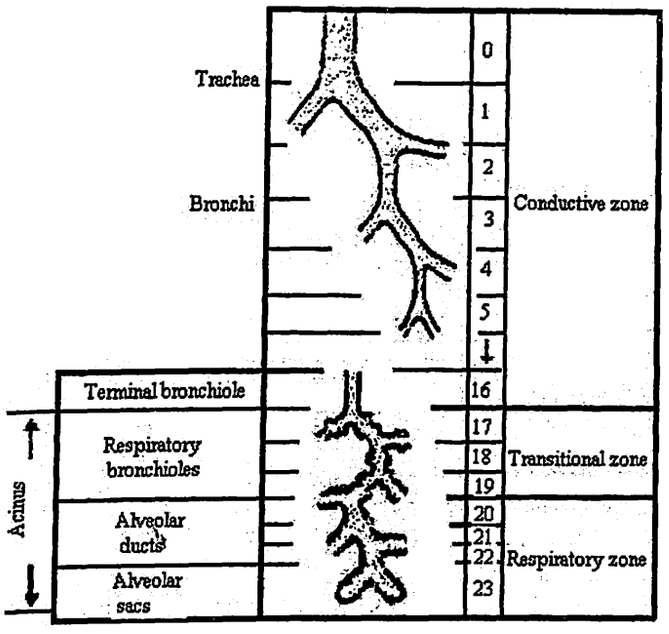
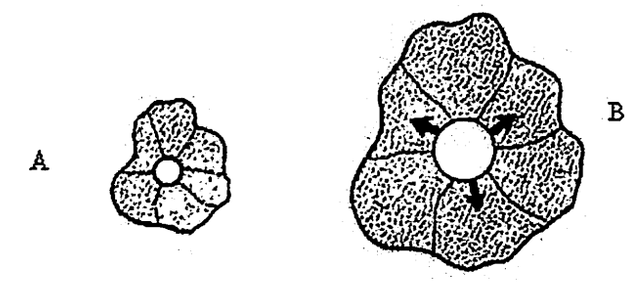


Fig. 5 : Airflow patterns. A, Laminar flow. B, Turbulent flow.



Diagrammatic representation of conductive, transitional and respiratory zones of the airways and components of the acinus. Arterisk designates order of generation of branching.



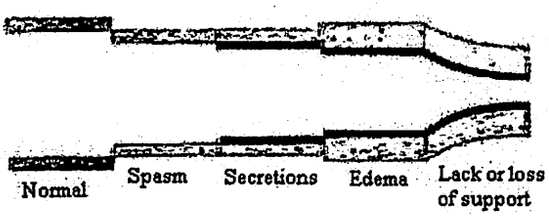


Figure 6 : Alveolar radial traction. Airway before inspiration, A, and on inspiration, B. Air filling each of the alveoli causes traction on the airway wall, thereby increasing the airway's circumference.

Diagrammatic representation of conditions affecting airway. (From Slonim, N.B., and Hamilton, L.H: Respiratory Physiology, ed. 3, St. Louis, 1976< The C.V Mosby Co.)

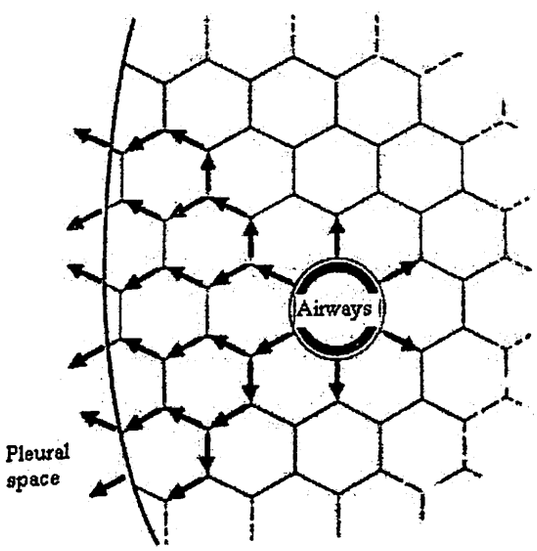


Figure 6a : Airway tethering by the surrounding alveoli. At higher lung volumes there is more tension in the alveolar septal walls. This tension is transmitted to the airways passing through the lung parenchyma, tending to increase their diameter and prevent their collapse (mechanical tethering). Hence, higher expiratory flows can be achieved at high lung volumes.

Assessment Of Airways Resistance

Airway resistance is increased during expiration because of the dynamic compression and intrapleural pressure (Fig. 7). Measurement of airways resistance during expiration is important in conditions like emphysema, chronic bronchitis and asthma.

Forced Vital Capacity

Definition

Vital Capacity (VC): volume of air expired after a maximal inspiration to the total lung capacity.

FVC - volume of air expired by forced expiration after inspiring to the total lung capacity.

The part of the air most sensitive to changes in expiratory airways resistance is the first second of expiration. This is designated FEV_1. Normally, 75-80% of vital capacity is expelled during the first second. It is reduced in obstructive lung disease, but may be normal in restrictive lung disease. (Fig.8)

Peak Expiratory Flow (PEF)

The relation between flow and volume is depicted in flow-volume curves (flow rates plotted against lung volume for expiratory efforts at different intensities). At high volumes the airflow is effort-dependent (Fig.9). Thus, maximal flow-volume curves are used to diagnose obstructive and restrictive lung disease. PEF is reduced in both conditions, for different reasons. It is reduced in restrictive lung disease due to reduced total lung capacity (caused by elevated alveolar elastic recoil); in obstructive lung disease, airway closure occurs at relatively high lung volumes.(Fig.10)

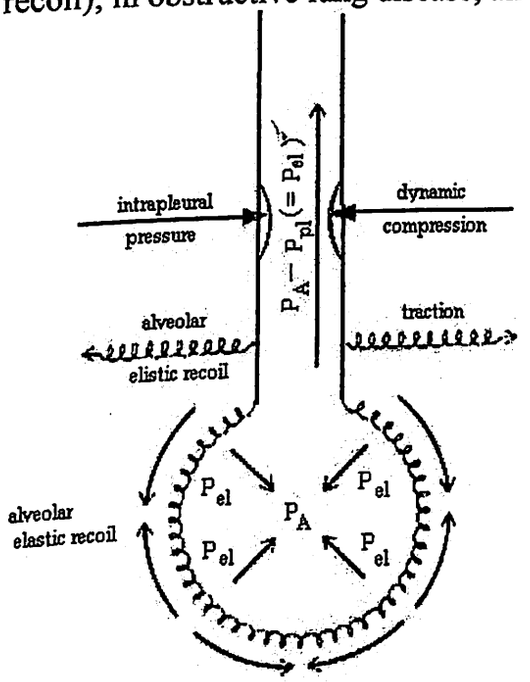


Figure 7 : Reprensentation of the effects of alveolar elastic recoil on airflow during a forced expiration. When dynamic compression occurs, alveolar elastic recoil helps to oppose it by traction on the small airways. The alveolar elastic recoil pressure becomes the effective driving pressure for airflow from the lung.

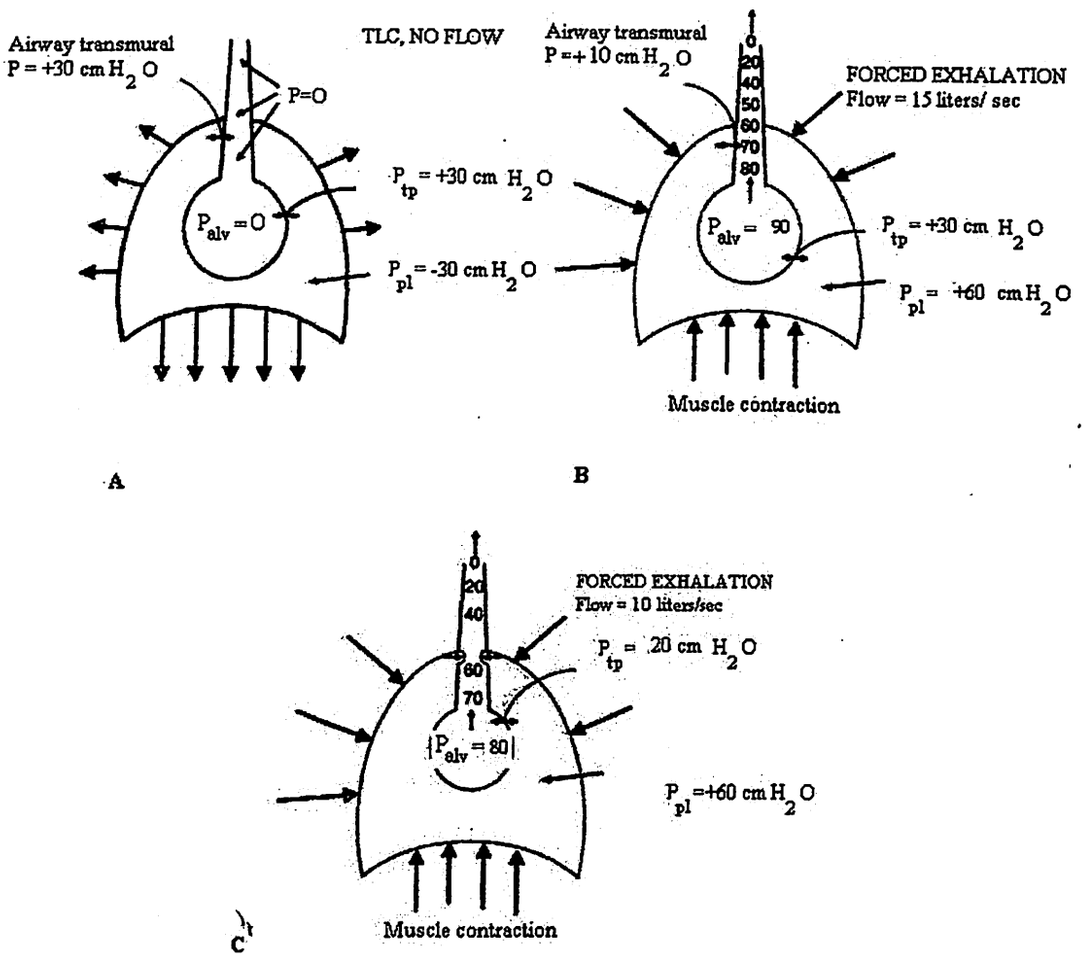


Figure 7a : A, End respiration, before the start of exhalation. B, At the start of a forced exhalation. C, Expiratory flow limitation later in a forced exhalation. Expiratory flow limitation occurs at locations where airway diameter is narrowed as a result of a negative transmural pressure. See text for further explanation.

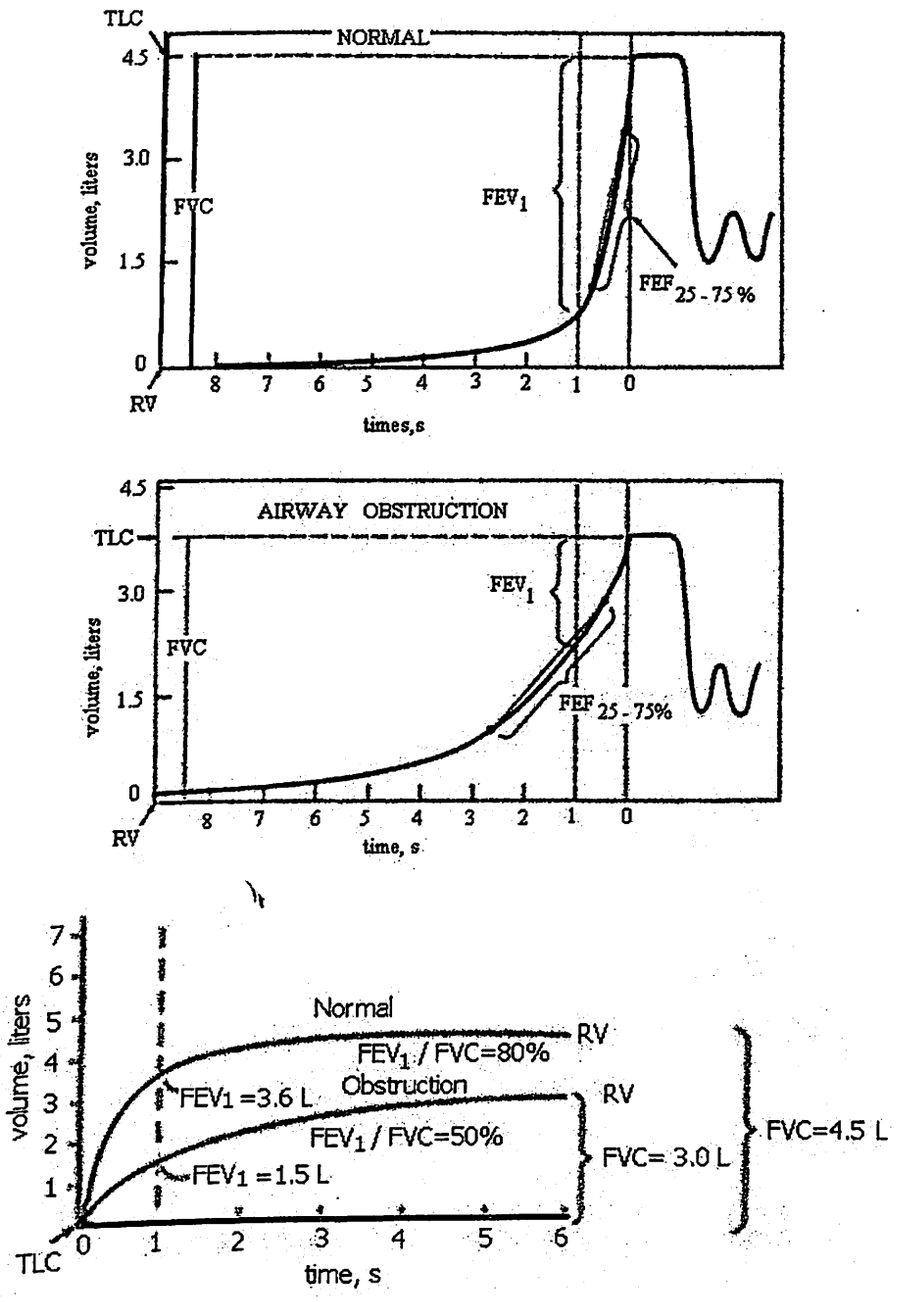


Figure 8 : Force vital capacity (FVC) maneuver using a spirometer. (See Fig. 3-4 for a diagram of a spirometer.) Upper trace: FVC from a normal subject. Middle trace: FVC from a patient with obstructive disease. FEV_1 = forced expiratory volume in the first second; $FEF_{25-75\%}$ = forced expiratory flow between 25 and 75 percent of the forced vital capacity. Bottom traces: Similar curves obtained from a more commonly used rolling seal spirometer. Note that the TLC is at the bottom of the curves and the RVs are at the top. The time scale is from left to right.

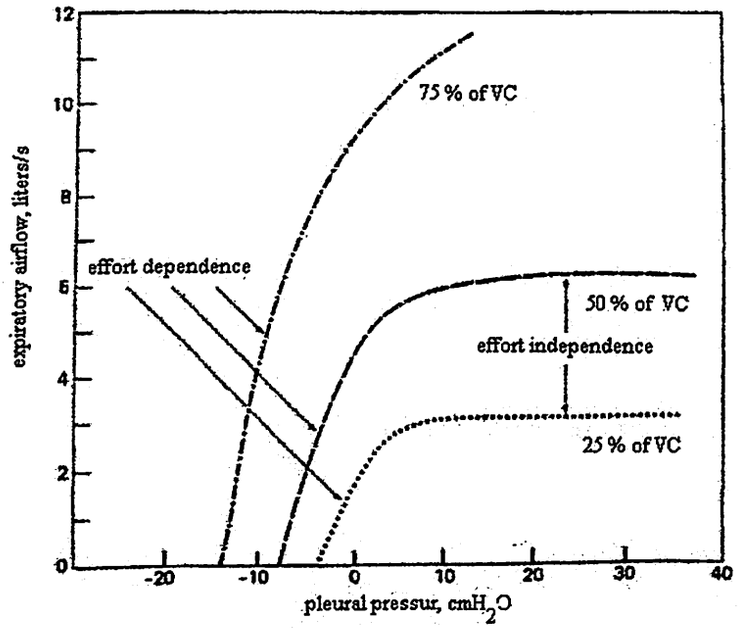


Figure 9 : Isovolumetric pressure-flow curves at three different lung volumes: 75 percent, 50 percent, and 25 percent of the vital capacity.

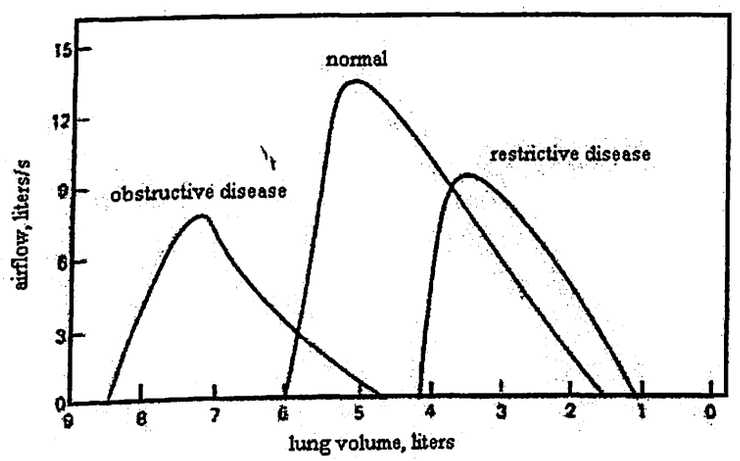


Figure 10 : Maximal expiratory flow-volume curves representative of obstructive and restrictive diseases.

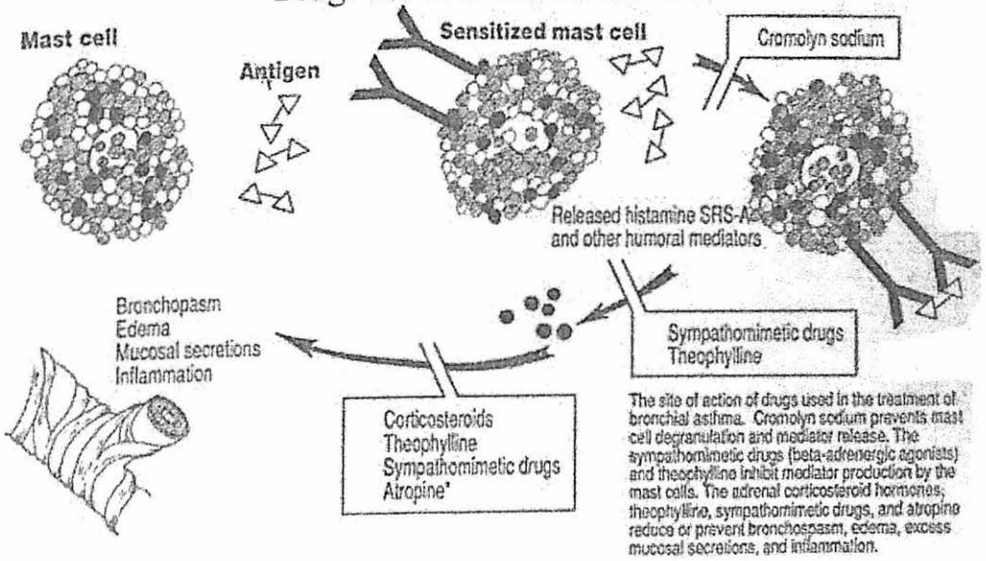
SISTEM PERNAFASAN

Modul 3 Respiratory Airways.

FUNCTIONS OF THE CONDUCTING ZONE OF THE AIRWAYS
1. Provides a low-resistance pathway for air flow; resistance is physiologically regulated by changes in contraction of airway smooth muscle and by physical forces acting upon the airways.
2. Defends against microbes and other toxic chemicals and foreign matter; cilia, mucus, and phagocytes perform this function.
3. Warms and moistens the air.
4. Phonates (vocal cords).

Neural and chemical influences on airway smooth muscle:	
Airways constricted by	Airways dilated by
Histamine Parasympathetic nerves Decreased carbon dioxide Some eicosinoids Irritants	Epinephrine Increased carbon dioxide Some eicosinoids
Physical influences 1. Airways are held open by transpulmonary pressure and lateral traction. They open more during inspiration and may collapse during forced expiration. 2. Airways may be occluded by mucus accumulation.	

Drugs use in bronchial asthma.



The site of action of drugs used in the treatment of bronchial asthma. Cromolyn sodium prevents mast cell degranulation and mediator release. The sympathomimetic drugs (beta-adrenergic agonists) and theophylline inhibit mediator production by the mast cells. The adrenal corticosteroid hormones, theophylline, sympathomimetic drugs, and atropine reduce or prevent bronchospasm, edema, excess mucosal secretions, and inflammation.

Pulmonary Function Tests

Lung volumes and capacities		
Volume	Symbol	Measurement
Tidal Volume (about 500 ml at rest)	TV	Amount of air that moves into and out of the lungs with each breath.
Inspiratory Reserve Volume (approximately 3000 ml)	IRV	Maximum amount of air that can be inhaled from the end of normal inspiration
Expiratory Reserve Volume (approximately 1100 ml)	ERV	Maximum volume of air that can be exhaled from the resting end-expiratory level
Residual Volume (approximately 1200ml)	RV	Volume of air remaining in the lungs after maximum expiration. This volume cannot be measured with the spirometer; it is measured indirectly using methods such as the helium dilution method, the nitrogen washout technique, or body plethysmography
Functional Residual Capacity (approximately 2300 ml)	FRC	Volume of air remaining in the lungs at end-expiration (sum of RV and ERV)
Inspiratory Capacity (approximately 3500 ml)	IC	Sum of IRV and TV
Vital Capacity (approximately 4600 ml)	VC	Maximum amount of air that can be exhaled from the point of maximum inspiration
Total Lung Capacity (approximately 5800 ml)	TLC	Total amount of air that the lungs can hold; it is the sum of all the volume components after maximal inspiration. This value is about 20% to 25% less in females than in males.
Maximal Voluntary ventilation	MVV	Maximum amount of air that can be breathed in a given time
Forced Vital Capacity	FVC	Maximum amount of air that can be rapidly and forcefully exhaled from the lungs following full inspiration. The expired volume is plotted against time.
Forced Expiratory Volume achieved in 1 sec	FEV1.0	Volume of air expired in the first second of FVC.
Percentage of forced vital capacity	FEV1.0/ FVC%	Volume of air expired in the first second, expressed as a percentage of FVC.
Forced midexpiratory flow rate	FEF25-75%	The forced midexpiratory flow rate determined by locating the points on the volume-time curve recording obtained

		during FVC corresponding to 25% and 75% of FVC and drawing a straight line through these points. The slope of this line represents the average midexpiratory flow rate.
Forced inspiratory flow rate	FIF25-75%	FIF is volume inspired from RV at the point of measurement. FIF2575% is the slope of a line between the points on the volume pressure tracing corresponding to 25% and 75% of the inspired volume.

EFFECT OF BREATHING PATTERNS ON ALVEOLAR VENTILATION

Subject	Tidal Vol. ml/Breath x	Frequency, Breaths/min =	xMinute Ventilation ml/min	Anatomic dead-space Ventilation ml/min	Alveolar Vent., ml/min
A	150	40	6000	150 x 40 = 6000	0
B	500	12	6000	150 x 12 = 18000	4200
C	1000	6	6000	150 x 6 = 900	5100

SISTEM PERNAFASAN

Modul 4 What is cough?

Cough is a sudden expulsion of air from the lungs. It is a normal mechanism of cleansing the tracheobronchial tree in healthy individuals. It usually stems from the accumulation of excess secretions in the mucous membranes of the larynx, trachea and bronchial tree. As such, the cough may be looked upon as the ultimate defense mechanism for keeping the tracheobronchial tree clear, being invoked - when other defense, such as the beating of cilia and the steady upword flow of the mucous sheet coating the epithelium, have been overwhelmed.

Describe the mechanism of cough.

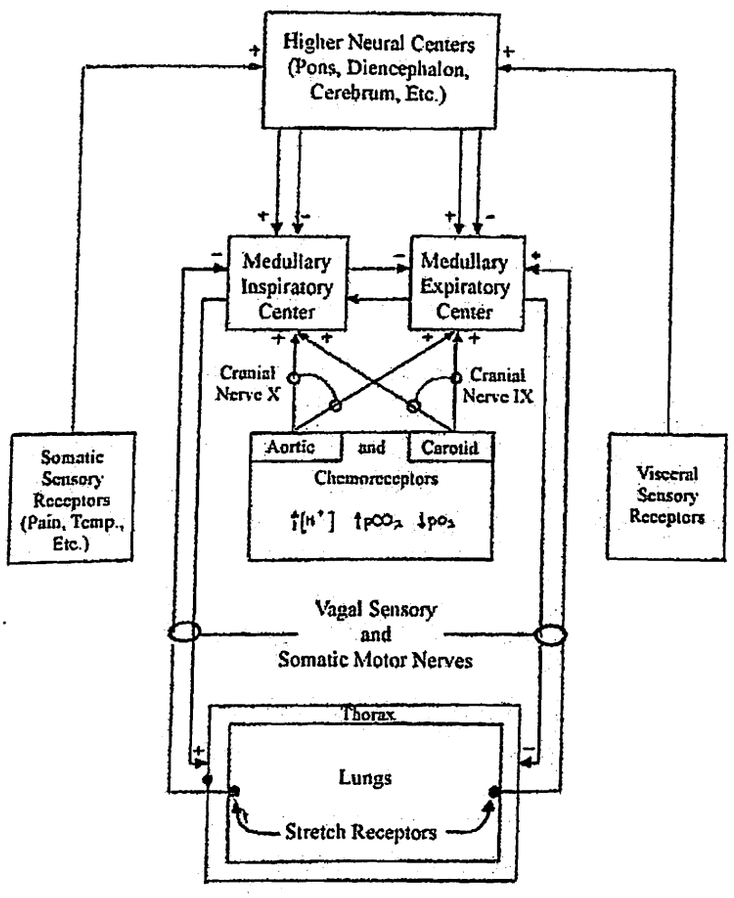
Cough reflex is initiated by any physical or chemical irritation of the pharyngeal, laryngeal or tracheobronchial epithelium. The cough receptors or the effector sensory end-organs situated in the subepithelial tissue are thus stimulated. From these receptors the impulses travel primarily along the vagus and glossopharyngeal nerves to the cough centre in the medulla. The impulses are then relayed through the efferent nerve fibres to involved muscles (diaphragm , intercostals, pectoral, scalenel, etc.) to comple the reflex arch.

Mechanically, a cough is preceded by a deep inspiration, glottic closure and compression of gas within the thorax by the expiratory musculature. A sudden opening of the glottis and a rapid expulsion of the compressed thoraxic gas ensues. Intrapulmonary pressures may approach 300 mm Hg above atmospheric pressure. The resultant dynamic airway compression aids maximal flow, which commonly exceeds 500 liters/min. A sheering stress is thus produced along the tracheobronchial epithelium that dislodges material in the airways. Coughing at high lung volumes tends to empty larger airways; coughing at lower lung volumes, the smaller airways.

Normal and abnormal patterns of breathing.

- Eupnea: normal breathing-repeated rhythmic inspiratory-expiratory cycles without inspiratory or expiratory pause; inspiration is active and expiration passive.
- Hyperpnea: increased breathing; usually refers to increased tidal volume with or without increased frequency. May or may not be related to increased metabolism.
- Polypnea, Tachypnea: increased frequency of breathing.
- Hyperventilation: increased alveolar ventilation in relation to metabolic rate (i.e. decreases alveolar PCO2 to less than 37 torr).
- Hypoventilation: decreased alveolar ventilation in relation to metabolic rate (i.e. permits alveolar PCO2 to rise above 43 torr).
- Apnea: cessation of respiration in the resting expiratory position.
- Apneusis: cessation of respiration in the inspiratory position.
- Apneustic Breathing: apneusis interrupted periodically by expiration: may be rhythmic.
- Gasping: spasmodic inspiratory effort, usually maximal brief and terminating abruptly: may be rhythmic or irregular.
- Cheyne-Stokes Respiration: cycles of gradually increasing tidal volume followed by gradually decreasing tidal volume.
- Biot's Respiratory: originally described in patients with meningitis by BIOT (Lyon Med 23:571, 561. 1876) as irregular respiraration with pauses: to day it refers to sequences of uniformly deep gasps, apnea, then deep gasps.

Regulation of pulmonary ventilation:



SISTEM PERNAFASAN

Modul 5 Respiratory control.

The site of the central inherent rhythmic activity which initiates the appropriate skeletal muscle activity effecting lung ventilation has not been identified. However, integrity of the ventromedial and dorsilateral regions of the medulla is important for the generation of rhythmic activity. These two centres which are reciprocally innervated have been called the inspiratory and expiratory respiratory centers respectively.

There are also additional centres in the pons which influence the pattern of ventilation and interplay between these leads to rhythmic continuous inspiratory and expiratory activity with no perceptible pause during any phase of the cycle. This activity is termed eupnoea.

Characteristic patterns of breathing have been described in animals following the destruction or stimulation of these centres. Ablation of the pneumotaxic centre (upper pons) is associated with ventilatory slowing and apneusis, whereas an increase in depth and rate of ventilation occurs with stimulation. Gasping occurs after ablation of the apneustic centre (lower and middle pons) and ventilation is abolished by destruction of the medullary respiratory centres.

The reticular activating system influences the pontine and medullary respiratory centres. An increase in the neuronal traffic in this system (electrical stimulation, arousal from sleep, and carbon dioxide build up) increases ventilation. Sleep, sedatives, narcotic analgesics (ondine's curse – see below), and general anaesthesia reduce activity in the system and there is an associated reduction in ventilation.

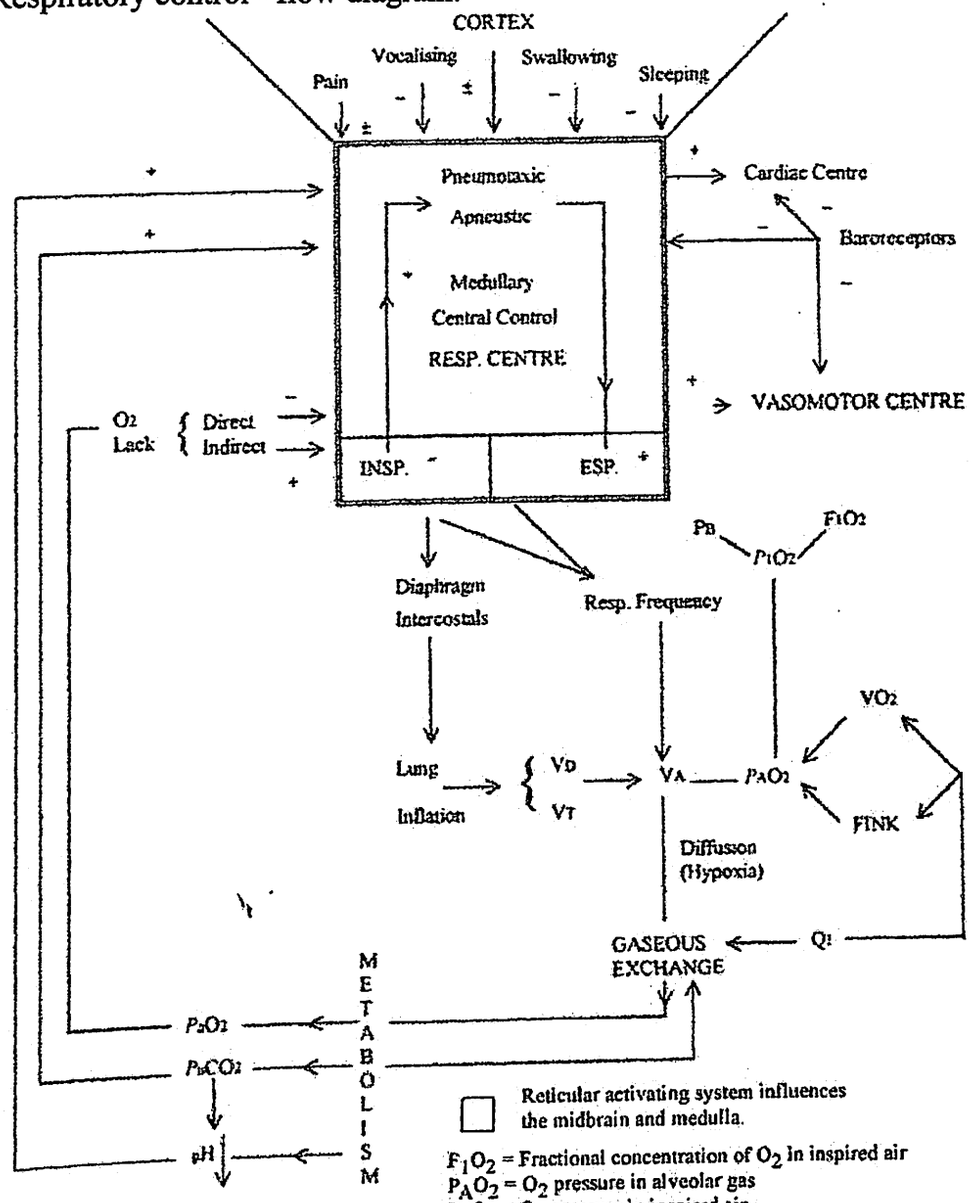
Ondine's curse (of mythical origin) described a condition in which the automaticity of breathing is lost and therefore describes a ventilatory state comparable with that observed following an overdose of some synthetic opiates, associated with bulbar poliomyelitis and following surgery involving the brain stem, i.e. a failure to breath in the absence of external vocal commands.

Stimulation of stretch receptors as lung inflation occurs, is associated with an increase in vagal discharge which inhibits inspiration, and allows expiration to occur (Hering Breuer Reflex). The importance of this reflex for normal human breathing has yet to be determined.

The role of the vagus nerve is also in dispute but classically it has been held that following vagal transection there is an increase in the depth of breathing and a decrease in respiratory rate. However, if the vagus nerve is intact, sudden deflation of the lung stimulates inspiration. Alternatively a sudden inflation of the lungs may be followed in neonates by a transient inspiratory effort this is known as head's paradoxical reflex.

Speech, sleep, swallowing, vomiting, sneezing, hiccoughing, pain and volition all modify the basic respiratory established by neuronal interaction.

Respiratory control - flow diagram:

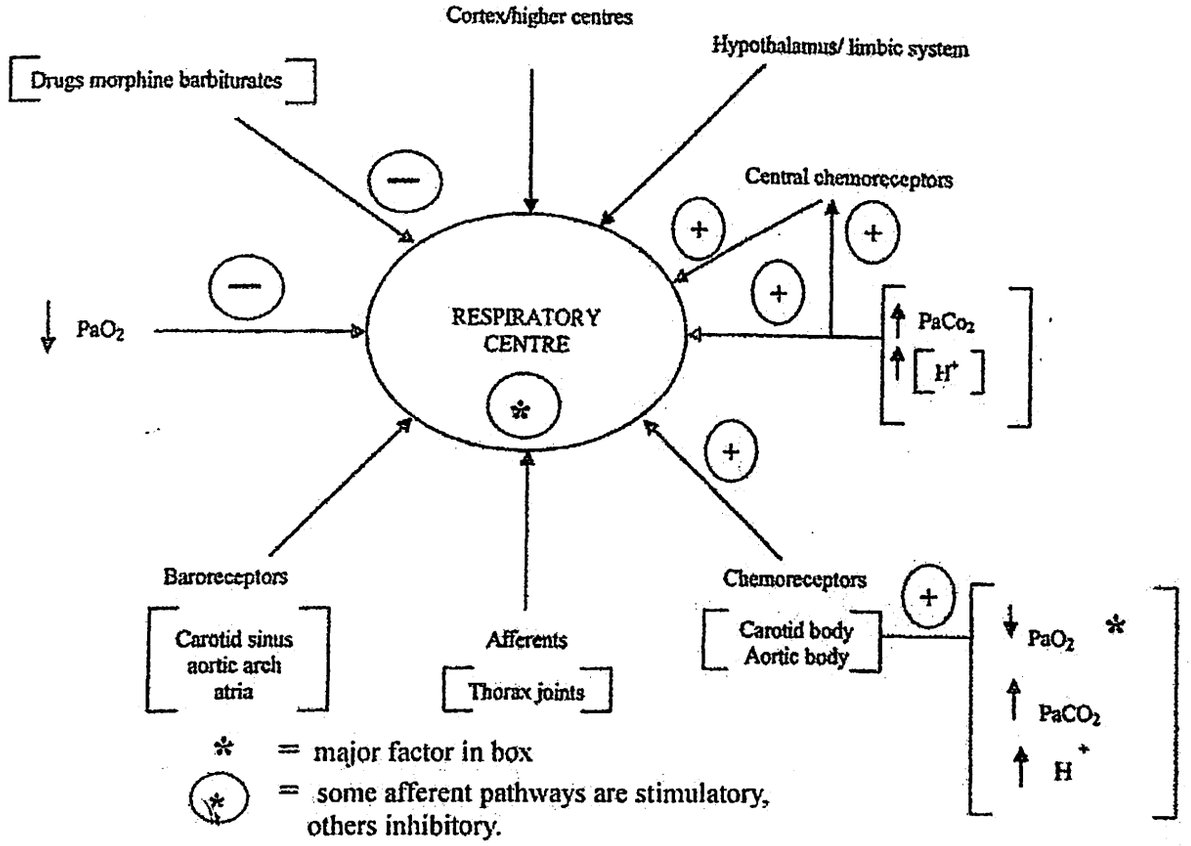


Reticular activating system influences the midbrain and medulla.

F_IO_2 = Fractional concentration of O_2 in inspired air
P_AO_2 = O_2 pressure in alveolar gas
P_IO_2 = O_2 pressure in inspired air
Q_T = total cardiac output/min.
V_A = alveolar ventilation/min.
V_D = volume of dead apace
VO_2 = O_2 consumption/min.
V_T = tidal volume

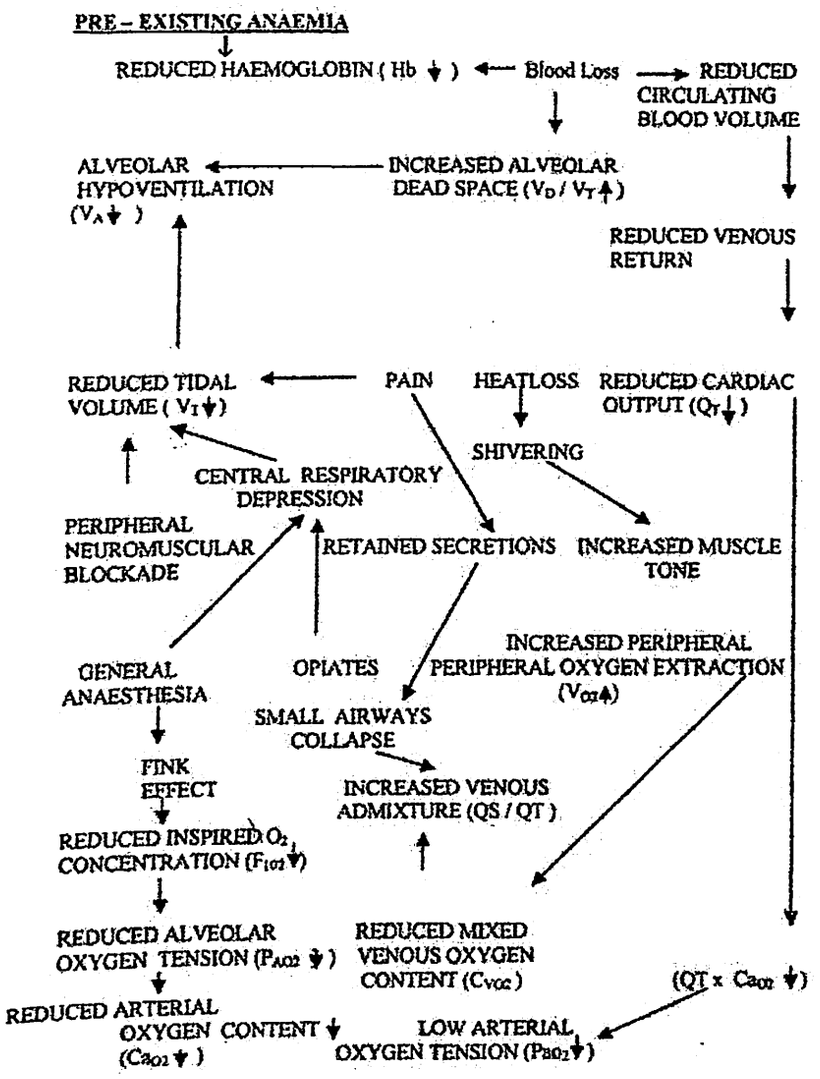
SISTEM PERNAFASAN

Modul 6 Control of respiration.



SISTEM PERNAFASAN

Modul 7 Postoperative Hypoxaemia.



Steps should be taken all through the anaesthetic to minimise postoperative hypoxaemia. This applied particularly to the elderly and to those with pre-existing respiratory disease, especially if there is sputum retention. Retained secretions must be cleared, and tracheobronchial toilet may be facilitated by humidification of the inspired gases. Aspiration by means of a small-bore suction catheter via the endotracheal tube should be performed. Small airway blockage and collapse will aggravate ventilation/perfusion abnormalities by increasing venous admixture and thus lead to further hypoxaemia. Tendency to small airway collapse may be reduced by a relatively large tidal volume, positive end-expiratory pressure (peep) and occasional aspiration of the tracheobronchial tree.

At the termination of general anaesthesia, an adequate tidal volume must be guaranteed by complete reversal of neuromuscular blocking agents and of any central respiratory depression. The airway must be patent. The blood volume and haemoglobin concentration should approximate to normal. Increased muscle tone, caused by either shivering or pain, leads to increased oxygen extraction at the periphery and thus further hypoxaemia. In order to avoid this, attention must be paid to control of the body temperature and relief of pain. Heat loss can be minimised by wrapping the patient in a space blanket. In relieving pain it is necessary to give potent narcotic analgesics and these inevitably lead to a degree of respiratory depression. A fine balance needs to be struck between all these factors.

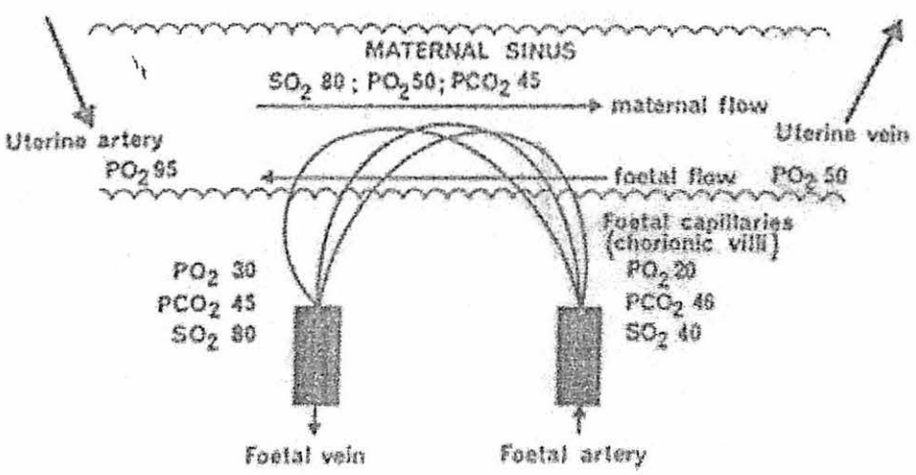
SISTEM PERNAFASAN

Modul 8 Fetal Respiration.

The palcenta is a feto-maternal organ with maternal sinuses through which maternal blood flows from uterine arteries to uterine veins. Fetal capillaries in the chorionic villi dip into the maternal pools and gas exchange between the 2 discrete circulations takes place. The fetal capillary networks (cotyledons) are arranged approximately in parallel and are surrounded by blood in the maternal sinuses or intervillous spaces. Pulsatile jet of blood from the maternal spiral arteries perfuse the intervillous spaces, and a counter-current flow between the maternal and fetal circulations (not very efficient in man) is established. This crestes a counter-current exchange system to facilitate gas, nutrient and waste-product exchange in the placental circulation.

Transplacental Gas Exchange Near Term.

The 0_2 diffusion gradient is about 30 mmHg because diffusion is hindered by cells in chorionic villi (less permeable to 0_2 than alveolar cells in the lung), and consequently 0_2 equilibrium between the blood of fetal vein and maternal sinus is not attained. Nutrients and waste-product pass through fetal capillaries. Maternal vein-fetal vein P 0_2 difference is~20mmHg, and differences of this order are maintained even during administration of 0_2 to the mother (P 0_2 in fetal vein rises only slightly); during uterine contractions in labour; and during altitude hypoxia (provided there are no maternal adverse effects). Transplacental gas exchange is determined largely by the rate and distribution of the maternal and fetal blood flows so that gas exchange is chiefly flow limited rather than diffusion limited.



Key: The fetal and maternal circulations flow in opposite directions (an oversimplification of a very complex system).

SO_2 = per cent of saturation with O_2

Pressures are in mmHg

Effects Of Labour On Fetal Blood P 0_2 , PC 0_2 , pH and Placental Perfusion

These parameters are relatively stable before the onset of labour and remain so during the 1st and 2nd stages of labour (a slight clinically unimportant fall in P 0_2 may occur during 2nd stage).

The mean P 0_2 in the uterine venous blood in pre-labour and during labour is steady, but it increases within minutes after birth (e.g. from 50 to 80 mmHg) as 0_2 is not now delivered to the fetus.

Rhythmic uterine contractions decrease, and relaxations increase, maternal placental perfusion so that the mean perfusion is relatively stable.

During normal parturition, therefore, fetal asphyxia does not occur, but it is present in varying degree immediately after birth until neonatal respiration commences and becomes functionally adequate.

If the maternal placental blood flow decreases then fetal hypoxia, hypercarbia and acidosis can occur.

SISTEM PERNAFASAN

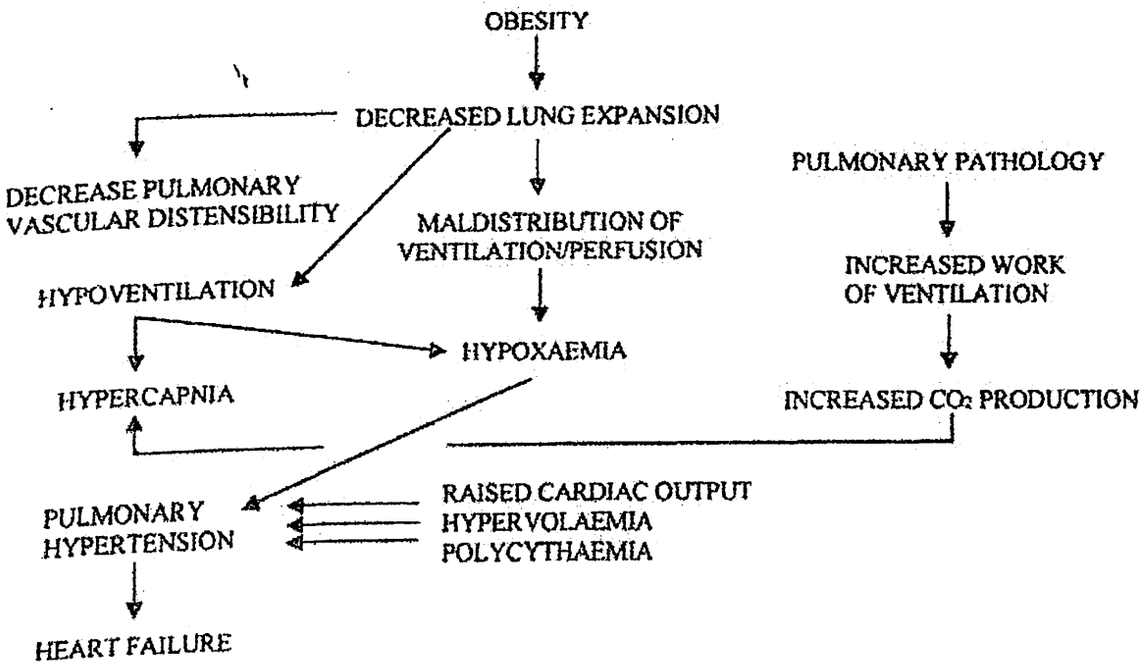
Modul 9 Physiopathology Of Obesity In Relation To Respiratory Physiology.

Obesity.

Obesity is probably the commonest metabolic problem encountered. It occurs at all ages but particularly from middle age onwards. The condition constitutes a very real hazard during anaesthesia and postoperatively, mainly because of the extra burden thrown on the respiratory and cardiovascular systems. It is obviously of benefit to the patient to postpone operation for some weeks if possible, and encourage drastic weight reduction by a low calorie diet. Unfortunately, however, such patients often present as acute emergencies.

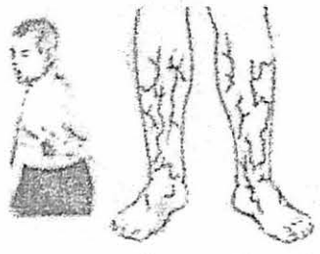
The disturbed physiology compounds the problem for the anaesthetist. Veins are always inadequate. The airway is difficult to maintain because of the relative shortness of the neck and the fullness of the tissues surrounding the air passages, which also makes poor cardiac performance, and the excess fat in the peritoneal cavity further embarrasses ventilation when the patient is prone. The use of the trendelenberg and lithotomy positions exaggerate an already complicated picture. High pressures have often to be used when ventilating the patient mechanically and this in turn may further reduce cardiac output. The postoperative period is similarly fraught with hazards from the same causes. Here ventilation may be further embarrassed by the pain of the operation site, and the use of narcotic analgesic results in respiratory depression. Hypoxaemia is common and attention must be paid to the ventilation and oxygenation of the patient.

Physiopathology of obesity:

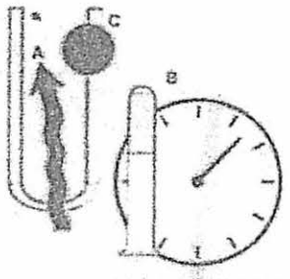


SISTEM PERNAFASAN

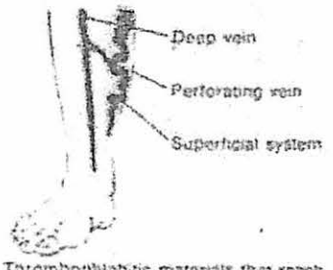
Modul 10 Acute Pulmonary Embolism.



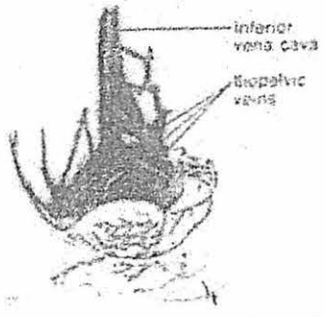
1. Suspect pulmonary embolism when acute cardiopulmonary symptoms or signs occur in the presence of one or more factors that underlie or predispose to the development of deep vein thrombosis



2. The basic underlying mechanisms in venous thrombosis are: vedous stasis (A), hypercoagulability (B), injury to the vascular endothelium (C)

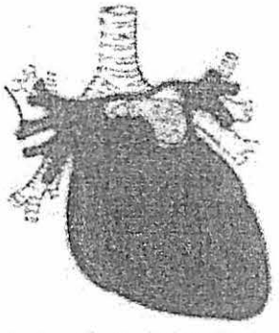


3. Thrombophlebitic materials that reach the lung originate in the deep veins of the lower limbs in about 90% of cases, but clinical evidence of the thrombotic process is usually detectable in fewer than 50% of cases

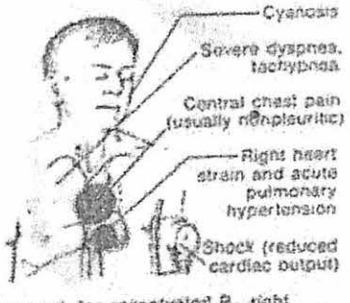


4. Other sources include the above and the veins of the upper extremity. Septic emboli usually arise from the pelvic veins in pelvic sepsis or from the right heart in bacterial endocarditis

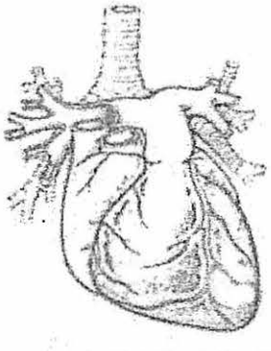
5. In virtually all cases, embolism is accompanied by the development of acute symptoms. Dyspnea and tachypnea are almost always present, while others, more variable, may present in one of five patterns, depending upon the size and number of emboli



6. The "central catastrophe" syndrome due to sudden occlusion of a major proportion of the pulmonary vascular bed, usually by a "saddle" embolus at the pulmonary bifurcation

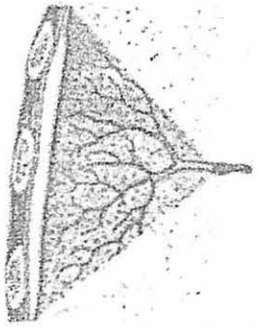


7. Look for accentuated P_2, right ventricular gallop, fixed splitting of P_2, and neck vein distention. Chest film is usually clear, with hilar enlargement and cardiomegaly. ECG may reveal acute right heart strain or only evidence of ischemia

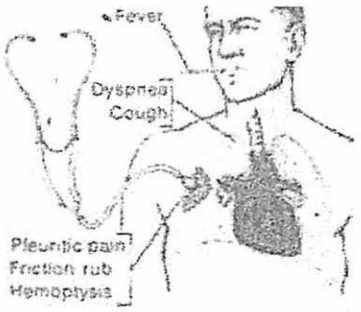


8. The "unexplained dyspnea" syndrome due to embolic occlusion of one or many pulmonary vessels, producing ischemia *without* infarction

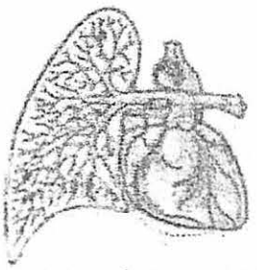
9. The salient feature is dyspnea, at times with wheezing. X-rays may show hemidiaphragm elevation and discoid atelectasis. ECG usually nonspecific. Chest pain occasionally occurs and may be central or pleuritic



10. The "pneumonia-pleurisy" syndrome results from infarction (ischemic necrosis) of one or more of the embolized areas

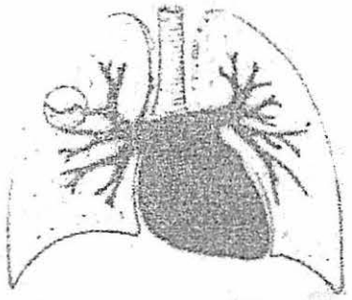


11. Look for parenchymal consolidation on chest film and the presence of pleural effusion

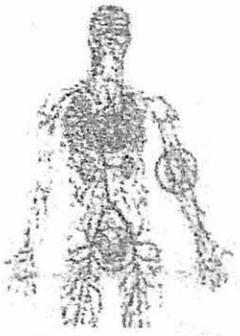


12. The "chronic pulmonary hypertension" syndrome results from recurrent showers of small emboli that lead to chronic pulmonary hypertension by virtue of sufficient precapillary pulmonary vessel obstruction

13. Typical clinical findings: exertional dyspnea, weakness, exertional syncope, central chest pain resembling angina, and signs of pulmonary hypertension (accentuated P_2, pulmonic ejection murmur at times). Auscultation of the lungs is negative, but note cardiomegaly, bilateral hilar enlargement with clear lung fields on chest film. ECG usually reveals right ventricular strain

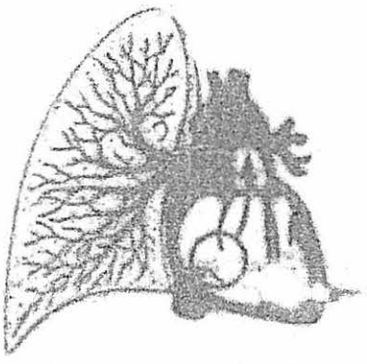


14. The "pulmonary sepsis" syndrome, bland type: a bland infarct cavitates and becomes secondarily infected, resulting in lung abscess. It is imperative that the thromboembolic etiology be recognized in order that appropriate management of this problem may be pursued



15. The "pulmonary sepsis" syndrome, septic type: emboli are septic from the start. They may arise from septic pelvic thrombophlebitis, right-sided endocarditis, contaminated indwelling IV catheters or A-V shunts

16. Findings include fever, cough, purulent sputum, leukocytosis, positive blood and sputum cultures, and x-ray evidence of multiple areas of necrotizing pneumonia, often cavitary

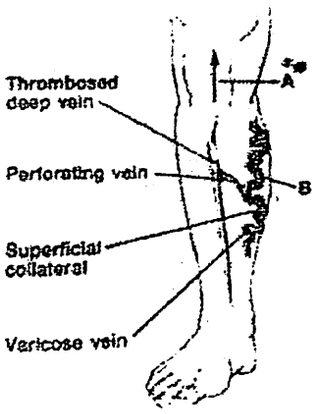


17. The presence of a heart murmur or evidence of septic thrombophlebitis will help identify the underlying cause

18. Since most pulmonary emboli originate from clots in the lower limbs, consider undertaking diagnostic tests to ascertain their presence or absence in this area in many cases of suspected pulmonary embolism. Approaches include Doppler ultrasound, impedance plethysmography, contrast venography, radionuclide venography, radioactive fibrinogen, and fibrinogen scintigraphy

Figure 1.

POSTOPERATIVE CALF THROMBI: IMPORTANT CONSIDERATIONS



The Problem

Twenty percent of postoperative thrombi extend into the major veins (A) and may embolize to the lungs. Some result in chronic venous insufficiency (B), which give rise to recurrent pain and swelling or ulceration. Others result in recurrent venous thrombosis

The Solution

Prevention of venous thrombosis. The routine use of effective prophylaxis in patients undergoing elective general surgery could prevent 4000 to 8000 postoperative deaths annually in the United States and prevent many more cases of disabling chronic insufficiency syndromes

Methods

Primary prevention

1) Heparin
2) Physical measures
3) Platelet suppressants
4) Dextran
5) Oral anticoagulants

Early detection of subclinical venous thrombosis by screening high-risk patients

Figure 4.

PULMONARY EMBOLISM AS A CAUSE OF DEATH IN POSTOPERATIVE PATIENTS

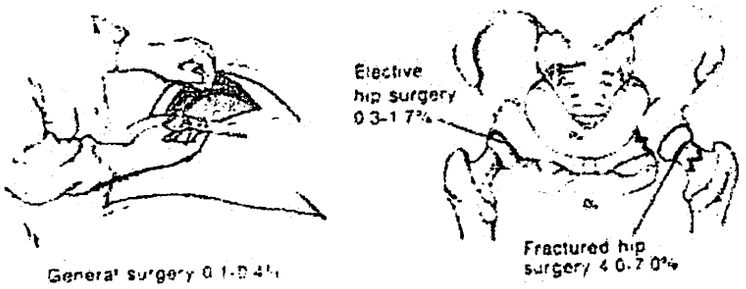


Figure 2.

SISTEM PERNAFASAN

Modul 11 Acid-base disorders.

Respiratory acidosis

Causes:

Acute

1. Narcotic overdose eg. Morphine
2. Sedatives : eg : barbiturates
3. Alcohol
4. Anaesthesia
5. Pulmonary oedema
6. Severe pulmonary infection
7. Bronchial obstruction
8. Atelectasis
9. Pneumothorax
10. Abdominal distension
11. Poliomyelitis

Chronic

1. Compensatory to metabolic alkalosis
2. Corpulmonale
3. Kyphoscoliosis
4. Multiple pulmonary embolism
5. Bronchiectasis
6. Emphysema
7. Pulmonary fibrosis

Respiratory alkalosis

Causes:

1. Hyperventilation anxiety
2. Encephalitis
3. Brain tumour
4. Intracranial surgery
5. Hypermetabolic conditions:
    a. Sepsis
    b. Hyperthyroidism
6. Salicylate poisoning
7. Mechanical hyperventilation
8. Compensation to metabolic acidosis

Pathophysiology

PARAMETERS	NORMAL RANGE	ACIDOSIS	ALKALOSIS
pH	7.35 - 7.45	↓	↑
PCO2	35 - 45mmHG	↑	↓
HCO3	23 - 28mmol/L	± ↑	± ↓
Base Excess	± 2	-	+

Pure Acute Respiratory Acidosis:
pH = 7.24
PCO_2 = 84mm Hg.
Actual Bicarbonate = 34 meq/L
(HCO_3)
TCO_2 Content = 36.52 meq/L
Base Excess = + 2
Pure Acute Respiratory Alkalosis:
pH = 7.51
PCO_2 = 28 mm Hg.
Actual Bicarnonate = 18 meq/L
(HCO_3)
TCO_2 Content = 18.74 meq/L
Base Excess = + 2.5
Compensation
Acute respiratory acidosis -- metabolic alkalosis:
1. Blood buffers react with carbonic acid and form more basic salts.
2. Compensation by kidneys
   a. Increased secretion and excretion of hydrogen ions
   b. Ammonium formation is stimulated and ammonium ions are excreted
   c. Retention of bicarbonate and excretion of chlorides
   d. Mono-hydrogen phosphate is converted to dihydrogen phosphate and excreted
3. Shift of electrolytes
   a. Hydrogen and sodium ions move from extracellular fluid (e.c.f.) Into intracellular fluid (i.c.f.)
   b. Potassium moves from intracellular fluid to extracellular fluid.

Compensation
Acute respiratory alkalosis -- metabolic acidosis:
1. Blood acids react with bicarbonates and form more acidic salts.
2. Compensation by kidneys
   a. Decreased secretion and excretion of hydrogen ions
   b. Supress ammonium formation
   c. Retention of chlorides and excretion of bicarbonates
3. Shift of electrolytes:
   a. Hydrogen and sodium ions move from intracellular fluid into extracellular fluid
   b. Potassium moves from extracellular fluid to intracellular fluid

SISTEM PERNAFASAN

Modul 12 Clinical Signs Of Hypoxia.

Symptoms of oxygen want.

The signs and symptoms of oxygen want are similar to those of drunkenness and inebriation. In other words the effects of oxygen lack and alcohol are similar. In regards to the central nervous system one first notes a mild exhilaration often manifested by argumentativeness and boisterousness. There then follows emotional impairment and later impairment in judgment which sometimes finally results in complete delirium. There is like wise incoordination and particularly psychomotor incoordination.

An interesting psychological experiment has been conducted by the army air corps. This consists in testing future pilot in atmospheres of 12% oxygen. In the pre-test period candidates are selected who are considered anxiety personalities and are segregated for statistical purposes from the group of normal individuals. When the anxiety subjects were exposed for a period of about 12 minutes to an atmosphere of 12% oxygen, approximately 25% collapsed and approximately an equal number showed some impairment in emotional stability and judgment. Of the normal or those considered normal by usual psychic tests none collapsed and only 18% showed any impairment in judgment.

Sensory symptoms exhibited are as follows. Most patients exhibit mild to severe head aches. Often precordial pain is present, typical of the type that occurs in anginal patients.
Finally lassitude and malaise are common features in the anoxic patient.

Gastrointestinal symptoms consist essentially of nausea, retching and vomiting.

SYMPTOMS OF HYPOXIA

GENERAL	SIMILAR TO DRUNKENNESS
PSYCHIC	HIGHER CENTERS FIRST IMPAIRED. PSYCHOMOTOR AND EMOTIONAL. JUDGEMENT POOR. BOISTEROUS, ARGUMENTATIVE- DELIRIOUS
SENSORY	HEADACHE, MALAISE, LASSITUDE. PRECORDIAL PAIN.
GASTROINTESTINAL	NAUSEA, RETCHING, VOMITING.
MUSCULAR	INCOORDINATION, CONVULSIONS, FLACCIDITY.
EXPERIMENTAL	12% OXYGEN. NORMALS- 18% IMPAIRMENT, NO COLLAPSE. ANXIETY CASES- 50% IMPAIRMENT, 25% COLLAPSE.

Cardiovascular Signs.
The pulse rate offers a more accurate index of hypoxemia. The increase in pulse rate occurs in circumstances either of lowered arterial oxygen tension ($Pa0_2$) or of lowered oxygen content. Hence pulse responses differ from the respiratory response to anoxemia. The pulse rate also increases at reduced arterial oxygen tensions which do not cause a respiratory response. Usually the degree of tachycardia encountered is directly proportional to the reduction of arterial oxygen saturation.

As hypoxemia becomes severe (levels not encountered clinically) and is prolonged, the pulse may slow and become full and bounding. This, however, is not constant nor a reliable sign except in the late stags.

The diagnosis of hypoxemia may often be substantiated by the therapeutic test. This relies on the observation that the pulse rate will decrease by 10 or more beats per minute within a minute or two of the start of oxygen therapy.

The mechanism of pulse changes due to hypoxemia is probably two fold. When oxygen tension is low, the chemorecptors are probably stimulated to produce reflex tachycardia. This does not preclude direct stimulation of cardiac centers by hypoxemia which may also occur. When there is a decrease in oxygen content but tension is normal, the tachycardia which occurs is probably produced through changes in circulation. Specifically, there is peripheral vasodilation due to tissue hypoxia followed by lowered peripheral resistance and lowered arterial blood pressure. Tachycardia is the produced reflexly through pressoreceptor mechanisms.

Blood pressure changes are relatively insignificant in clinical situations of hypoxemia. Generally, there is a slight increase in both systolic and diastolic pressure with moderate hypoxemia (arteriao saturation 80% or better). However, exposure of a vein to a 10% oxygen atmosphere produces systolic changes of 6 to 12 mm Hg and diastolic changes of 0 to 3 mm. In many individuals blood pressure falls. With severe hypoxemia (arterial oxygen saturation less than 80%), there may be greater elevations in systolic pressure but diastolic invariably falls. Such changes may occur promptly, but if the hypoxemia is prolonged, the blood pressure falls markedly and shock supervenes.

Cardiocirculatory Signs Of Hypoxia:
Mechanism of pulse changes.
1. Low oxygen tension:
   - stimulates chemoreceptors
   - response is reflex tachycardia.
2. low oxygen content:
   - causes tissue hypoxia - vasodilation.
   - lowered peripheral resistance – stimulates pressoreceptors.
   - response is reflex tachycardia.
3. possible stimulation at cardiac centers.

Respiratory Signs.
Contrary to common belief the respiratory response to hypoxia is neither marked nor significant. Not until a 16% oxygen atmosphere is breathed does respiration increase and the minute volume then increase 7%, while breathing an atmosphere of 10% oxygen the minute volume increases only 17%. Only when there is severe active hypoxia does a vigorous hyperpnea occur. The change in minit volume is brought about through an increase in both rate and depth. Initially, the rate shows a periodic increase but after prolonged hypoxia there is depression rates amplitude is irregularly increased at first by later shows a slight but constant increases.

The mechanism of respiratory stimulation is the chemoreceptor system of the carotid and aortic bodies. These receptors respond only to hypoxemia when the tension of the oxygen (the Pa02) is reduced below normal. Thus, low arterial oxygen content which would occur in hemorrhage, anaemia or carbon monoxide poisoning will not stimulate chemoreceptors and increase respiratory exchange.

RESPIRATORY SIGNS OF HYPOXIA

OXYGEN PER CENT	MINUTE VOLUME
16	7% INCREASE
10	17% INCREASE
PATTERN OF RESPONCE INCREASE IN BOTH RATE AND DEPTH FIRST RATE INCREASES BUT LATER IS SLOWED. AMPLITUDE IS IRREGULARLY INCREASED AT FIRST BUT LATER IS CONSTANT.	
MECHANISM CHEMORECEPTORS	
STIMULUS LOWERED PaO_2 (OXYGEN TENSION) LOWERED OXYGEN CONTENT (SHOCK-ANAEMIA) NO CHANGES.	

SISTEM PERNAFASAN

Modul 13 Chronic Obstructive Pulmonary Disease And Chronic Hypoventilation.

COPD is the most common cause of chronic hypoventilation. Three features are responsible.

1. Underventilation of well-perfused alveoli (low VA/Q)
2. Low alveolar ventilation, made worse by the wasted ventilation of poorly perfused alveoli.
3. Increased CO_2 production, due to excessive work of breathing.

Hypoxemia

Abnormal Gas Exchange And Hypoxemia

There are only common causes of Hypoxemia

1. Hypoventilation
2. Ventilation / perfusion mismatch.
3. Shunt.

The effect of O_2 therapy depends on the cause of hypoxemia.

Hypoventilation

Hypoventilation with room air causes hypercapnia and hypoxemia.

The severity of hypoxemia is dependent on the level of P_ACO_2 (P_ACO_2 is about equal to P_aCO_2).

O_2 Therapy For Hypoxemia Due To Acute Hypoventilation

If pure acute hypoventilation (e.g muscle paralysis or overdose of sedatives) is the cause of hypoxemia, only a small increase in F_1O_2 is needed to bring PaO_2 to normal. Although O_2 therapy always is among the initial treatment measures, O_2 therapy alone is not adequate in such a clinical situation. An adequate treatment program will include some means of increasing alveolar ventilation . Mechanical ventilation often is necessary. The alveolar PCO_2 will fall, making room for O_2 molecules, the P_AO_2 will rise without using an O_2-enriched atmosphere.

HYPOXEMIA Due To Ventilation/Perfusion Mismatch.

Ventilation/perfusion mismatch describes the common problem in which some units are well perfused but hypoventilation (regional hypoventilation).....mech.

Effect Of Therapy On Hypoxemia Due To VA/Q Mismatch.

Hypoxemia due to VA/Q mismatch alone is readily repaired by O_2 therapy (fig.). Because.

Hypoxemia due to VA/Q mismatch-Effect of 100% O_2 breathing.

1. N_2 is washed out of all units.
2. All pulmonary capillary blood encounters high P_AO_2
3. $P_{(A-a)}O_2$ is normal.

HYPOXEMIA Due To Shunt

Shunt describe perfusion of nonventilated alveoli. Capillary blood perfusing such collapsed alveoli is not modified by its journey and has the same gas content as systemic venous blood.

Cause	HYPOXEMIA	Respon to high F_1O_2
Hypoventilation		Corrected
VA/Q mismatch		Corrected
Shunt		Not corrected

Effect Of O_2 Therapy On Hypoxemia Due To Shunt

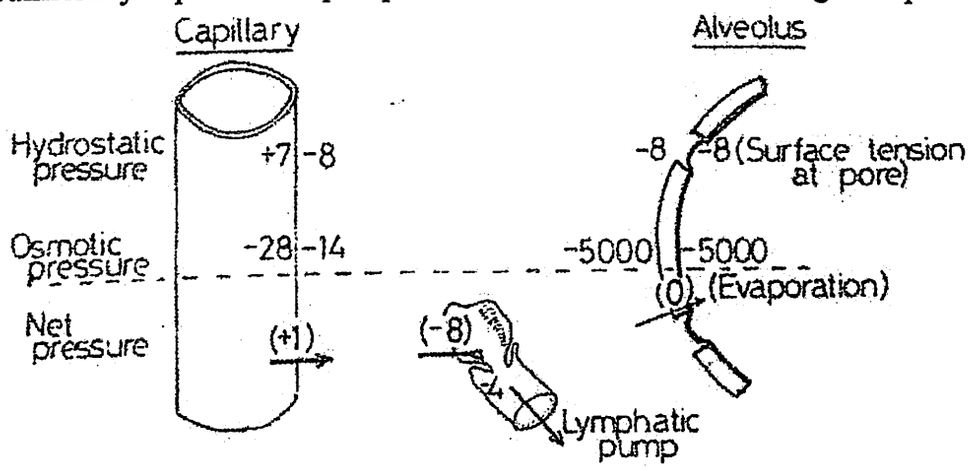
The enriched O_2 atmosphere has no contact with the pulmonary capillary in shunt units and has little effect on PaO_2.

Interstitial free fluid pressure	-8 mm Hg
Total inward pressure	20mm Hg

The net mean filtration pressure at the pulmonary capillary membrane is

Total outward force	+21 mm Hg
Total inward force	-20 mm Hg
Net mean filtration pressure	+1 mm Hg

This net filtration pressure causes a slight continual flow of fluid from the pulmonary capillaries into the interstitial spaces, except for a small amount that evaporates in the alveoli. This fluid from pulmonary capillaries is pumped back to the circulation through the pulmonary lymphatic system.



Pressure causing fluid movement

Pulmonary Edema.
Any factors that causes the pulmonary interstitial fluid pressure to rise from the negative range into the positive range will cause sudden filling of the pulmonary interstitial spaces and in more severe cases even the alveoli.

What are the factors that must be overcome before positive interstitial fluid pressure can occur and cause edema.
1)The normal negativity of the interstitial fluid pressure of the lungs.
2)The lymphatic pumping of the fluid out of the interstitial spaces
3)The increased osmosis of fluid into the pulmonary capillaries caused by decreased protein in the interstitial fluid when the lymph flow increases.

For pulmonary edema to occur, the pulmonary capillary pressure normally must rise to a value at least equal to the plasma colloid osmotic pressure. In the human beings, who normally has a plasma colloid osmotic, pressure of 28 mm Hg, one can predict that the pulmonary capillary pressure must rise from the normaly level of 7 mm Hg to over 28 mm Hg to cause pulmonary edema, giving a safety factor against edema of about 21 mm Hg.

Q.Why doesn't the fluid normally present in the interstitial space floods the alveoli?

A.This does not happen because of the negative interstitial fluid pressure of approximately 8 mm Hg; which continually tends to pull fluid inward through the alveolar membrane.

What Is The Mechanism For Keeping The Alveoli Dry.
Pulmonary capillaries have very large slit-pores between the adjacent endothelial cells. Ions such as Na+, Cl- and K+ as well as crystalloid molecules such as glucose, urea can pass through these large capillary pores with ease.

Alveolar epithelial membrane contains no such large openings. Therefore the ions and molecules in the pulmonary capillaries can cause osmotic pressure effects, at the alveolar membrane.

Example: when water enters the alveoli, the high concentration of the different dissolved substances in the pulmonary interstitial fluid causes almost instantaneous osmosis of the water from the alveoli into the interstitial fluid and the fluid is then absorbed into the pulmonary capillaries because of the colloid osmotic pressure of the plasma.

In a person who drowns in fresh water, enough fluid can be absorbed from the alveoli into the blood within 2 to 3 minutes to cause fibrillation of the heart because of dilution of the blood electrolytes.

Apart from osmosis, small amount of fluid can also be moved from the alveoli into the interstitial spaces as a results of suction by the negative pressure in these spaces.

So in sea-water drowing, the saline solutions, the ions of which prevent its osmosis into the interstitial fluid, moves slowly from the alveoli into the interstitial spaces because of the negative interstitial pressure.

Pulmonary Interstitial Fluid Dynamics:
1) Pulmonary capillary pressure is very low, approximately 7mm Hg.
   (systemic capillary pressure is 17mm hg)
2) Interstial free fluid pressure in the lung interstitium is -8mm Hg.
3) The pulmonary capillaries are relatively leaky to protein molecules so that the protein concentration of lymph leaving the lungs is relatively high, averaging 4 gm%
4) Rate of lymph flow from lungs is also very high, because of continuous pumping motion of the lungs.
5) The interstitial spaces of the alveolar portions of the lungs are very narrow.
   (minute spaces between the capillary endothelium and alveolar epithelium).
6) Alveolar epithelia are not strong enough to resist very much positive pressure. They are probably ruptured by any positive pressure in the interstitial spaces greater than atmospheric pressure (0 mm Hg), which allows dumping of fluid from the interstitial spaces into the alveoli.

Pressures causing fluid movement is as follows

Forces tending to cause movement of fluid outward from the capillaries and into the pulmonary interstitium:

Capillary pressure	7 mm Hg
Interstial fluid colloid osmotic pressure	14 mm Hg
Total outward pressure	21 mm Hg

Forces tending to cause absorption of fluid into the capillaries:

Plasma colloid osmotic pressure	28 mm Hg

SISTEM PERNAFASAN

Modul 14 Pleural Fluid, Pulmonary Interstitial Fluid, Pulmonary Edema.

Physiology Of Pleural Fluid Secretion And Absorption

Pleural Fluid:

The lungs are covered with a serous membrane known as the visceral pleura; the walls of the chest cavity are lined with another serous membrane known as the pariental pleura. The fluid secreted by these two serous membranes is resposible for the surface tension which holds the lungs and the chest wall very close together. This small amount of fluid found between the visceral pleura and parietal pleura is called the pleural fluid. The fluid is continually produced and continually reabsorbed into the lymphatics. Basically this pleural fluid is a capillary filtrate.

The parietal pleura which lines the chest wall has capillaries with pressures in them similar to those throughout the body.

The visceral pleura covers the outer surface of the lungs, and the hydrostatic pressure in its capillaries in considerably less than that found in the pariental pleural capillaries nearby. This is because the blood in the visceral pleural capillaries comes from the pulmonary arteries and right ventricle, rather than the left ventricle and the systemic
circulation.

As the right ventricle in thinner than the left ventricle and has less muscle mass, it will not be able to generate the contractile force that the left ventricle can. This causes the hydrostatic pressure in the visceral pleural capillaries to be about one-third as great as those in the pariental pleural capillaries.The osmotic pressures in both sets of capillaries will be the same. This means that the forces operating at the two sets of capillaries are such that some fluid which is filtered and leaves the parietal pleural capillaries will be absorbed in the visceral pleural capillaries. Thus, the forces which causes this flow of filtrate from parietal to visceral capillaries are responsible for the formation of the pleural fluid and for keeping the visceral pleural surface of the lungs close to the parietal pleural surface of the chest wall.

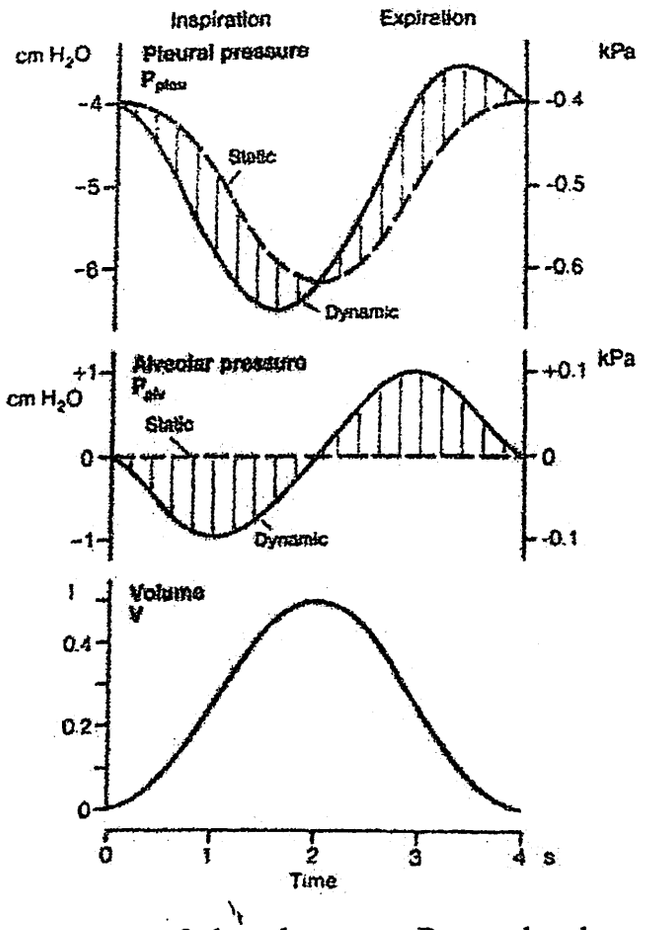
There are also lymphatics present in both the visceral and pariental pleura, which return protein and filtrate to the circulation.

The pleural fluid seal between the lungs and chest wall is important because it
1) forces the lungs to move, to expand and contract with the movement of the chest wall.
2) reduces the friction between the surface of the lungs and the chest wall.

If the pleural fluid seal is broken, as it often is when the chest is punctured the lungs, or lung will collapse to a much smaller volume.

Pleural Effusion:

An excess of pleural fluid is called a pleural effusion. A pleural effusion, like edema, can be caused by anything that upsets the normal balance of forces at the capillaries and the lymphatics.
Examples are: -
- inflammatory conditions which increase capillary permeability
- tumour or infections (which block and reduce flow through the lymphatics)
- increased pulmonary capillary pressure (such as can occur in heart failure).



Time course of pleural pressure P_{pleu} , alveolar pressure P_{alv}, and respired volume v during A breathing cycle. The dashed lines show the pressure that would be found if respiration encounted only elastic resistances. Because viscous resistances are also present, P_{pleu} and P_{alv} become more negative during inspiration and more positive during expiration (small thin arrows).

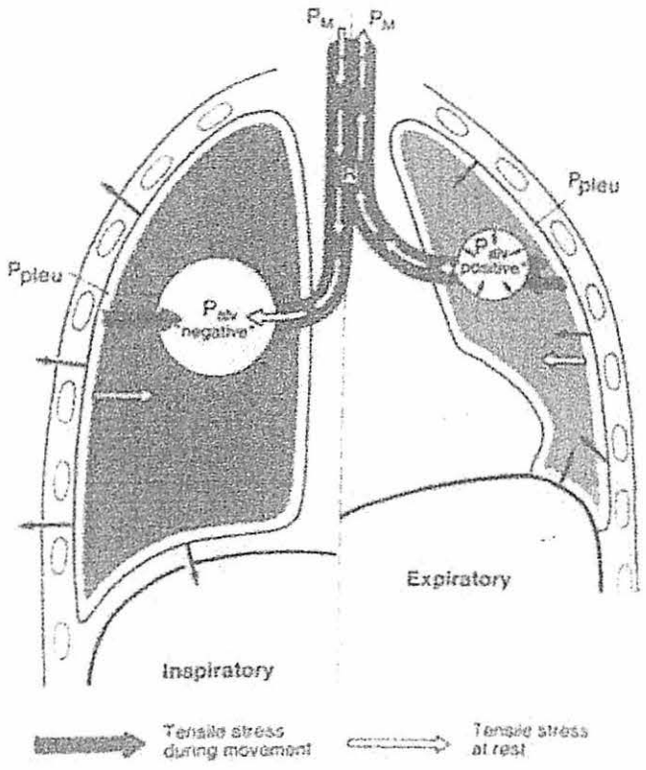
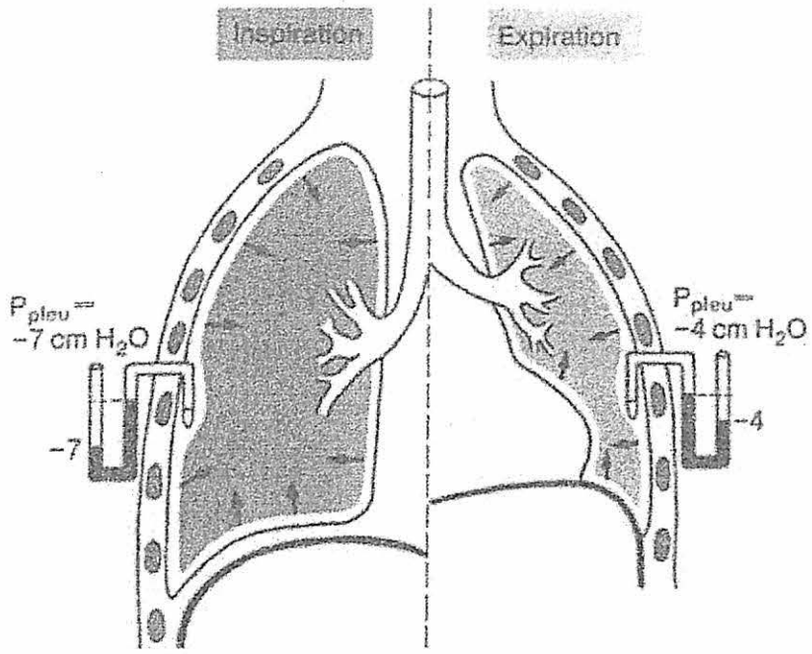


Diagram to explain the changes in pleural (P_{pleu}) and alveolar (P_{plv}) pressure during inspiration (left) and expiration (right).
Pm, mouth pressure; R, airway resistance.

The resulting pressure changes during a respiratory cycle are shown.

If only the elastic resistance of the lung were to be overcome during respiration, the alveolar pressure p alv would remain zero over the entire cycle and the pleural pressure would follow the dashed curve $P_{pleu\ (stat)}$.

But because of the additional viscous resistances P_{alv} becomes negative in the inspiration phase and positive in the expiration phase. By adding this curve to that for $P_{pleu\ (stat)}$ one obtains the dynamic pleural pressure $P_{pleu\ (dyn)}$. It is evident here that in order to overcome the viscous resistances, $P_{pleu\ (dyn)}$ must always be somewhat smaller than $P_{pleu\ (stat)}$ during inspiration and some what larger during expiration.



Pressure-Volume Relations In The Breathing Cycle:

During a breathing cycle the pleural and alveolar pressure change in a regular way. The relationship between the two are indicated as follows:

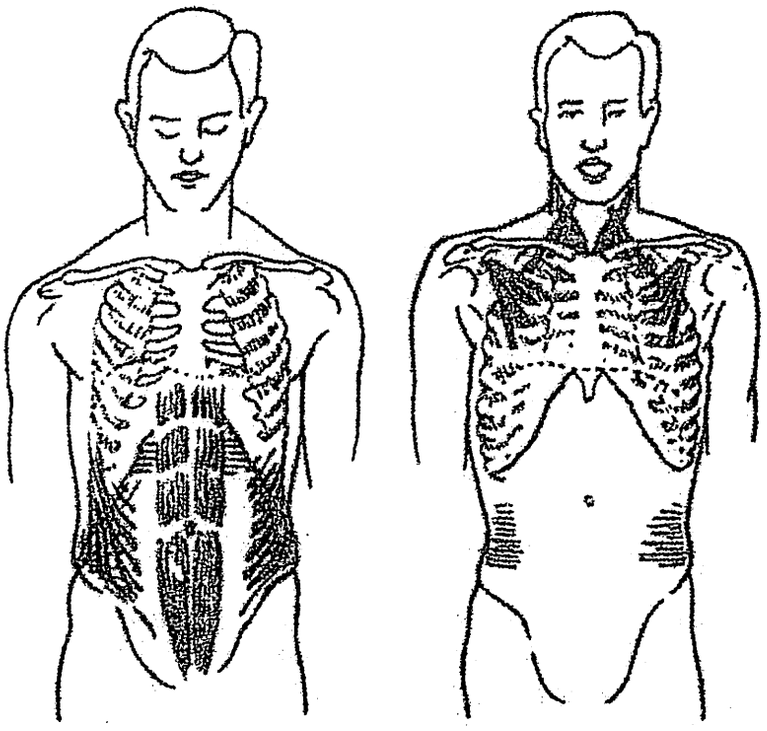
When the thorax is briefly at rest, as it is at the transition from inspiration to expiration the only force exerted on the pleural spaces is the elastic recoil force of the lung, which causes a "negative" pleural pressure, $P_{pleu\ (stat)}$

The alveolar pressure $P_{plv\ (stat)}$ is zero in the resting thorax because the alveoli are in communication with the mouth, so that the pressure can equilibrate.

During normal respiratory movements the situation is more complex. This is shown in the diagram.

In the diagram, the alveolar space is represented by a large bubble. The black arrows indicated the directions of movement. The red arrows, the directions in which the tensile forces act. During inspiration (left) the airway resistance R prevents the air from flowing rapidly enough into the enlarged alveolar space. Therefore the pressure in the alveoli must fall, becoming negative with respect to the outside pressure. This decrease in alveolar pressure affects the pleural space and makes the pleural pressure still more 'negative' . The movement - dependent pleural pressure $P_{pleu\ (dyn)}$ is thus the sum of the static pleural pressure $P_{pleu\ (stat)}$ and the momentary alveolar pressure P_{plv}.

During expiration the situation is reversed. P_{plv} becomes positive and reduces the negativity of $P_{pleu\ (stat)}$.



Accessory respiratory musculature. Left: Accessory muscles for expiration; Right: Important accessory muscles for inspiration.

Pleural Pressure:

The pleural pressures during breathing is shown. The elastic forces in the lung, which pull in the direction of the arrows, cause the pressure in the interpleural space to be "negative" with respect to the outside of the body. This is demonstrated by an appropriately positioned manometer.

The close contact between the surface of the lung and the inner surface of the chest wall is maintained only as long as there is no opening into the interpleural space. If the chest wall or the lung is injured so as to let air into the space the lung collapses - its elastic and surface tensions pull it together towards the hilus. Filling of the space between the pleurae with air is called pneumothorax.

The collapsed lung, having lost contact with the thorax wall, can follow the respiratory movements only incompletely or not at all, so that effective exchange of gases is impossible.

SISTEM PERNAFASAN
Modul 15 Respiration.

Respiratory muscles.
During quiet breathing the intercostal musculature and he diaphragm normally suffice to change the shape of the thoracic cavity.

The inspiratory muscles of the rib cage are the:
(1) external intercostals
(2) the intercartilaginous part of the internal intercostals and
(3) diaphragm (innervated by phrenic nerve from c3 – c5).

The expiratory muscles under normal conditions comprises only the internal intercostals.

Accessory Muscles Of Respiration:
When more respiratory work must be done – particularly when breathing is difficult and the subjective feeling of shortness of breath (dyspnea) develops - accessory muscles can supplement the muscles regularly used for breathing.

The accessory inspiratory muscles include all those that are attached to the-pectoral gidle, the head or the spine and can lift the ribs. These are:
(i) major and minor pectorals
(ii) the scalness
(iii) the sternocleidomastoids and
(iv) parts of the seratus muscles

In order for these to be employed in respiration, their points of origin must be fixed by other muscles or stabilized in some other way.

The main accessory expiratory muscles are the abdominal muscles that pull the ribs down and compress the abdomen forcing the vicera and the diaphragm upward.

SISTEM PERNAFASAN

Modul 16 The Pulmonary Circuit: Some Basic Physiologic Considerations.

Pulmonary Hypertension.

By virtue of this great vascular capacitance, large volumes of lung can be removed without causing pulmonary hypertension in patients free of severe obstructive lung disease.

⇧

Resistance, which is a function of the cross sectional area of the perfused vascular bed, is low because of the bed's ability to expand and accommodate increases in flow by "recruiting" vessels not perfused at rest. Pressure is thus maintained according to the equation: Pressure = Flow X Resistance.

⇧

This is explained by the low resistance of the pulmonary vascular bed.

⇧

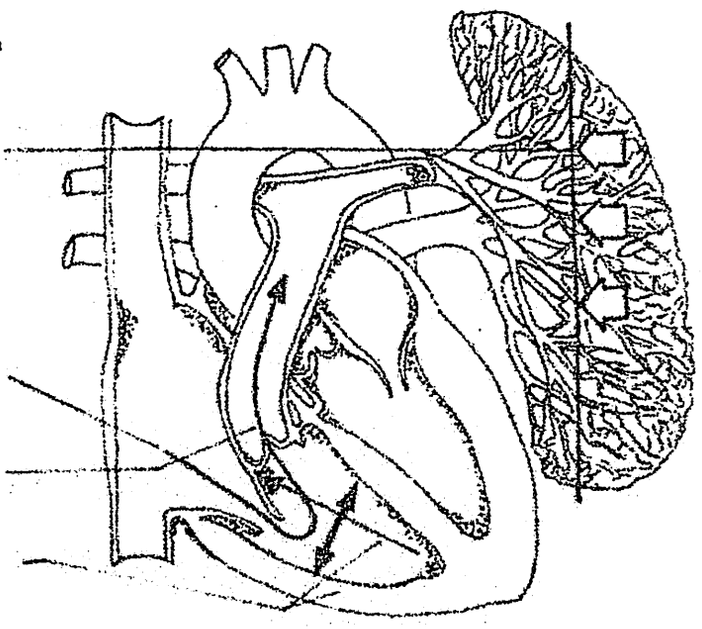
Normal pressure is not significantly increased even by marked increases in flow, as during exercise.

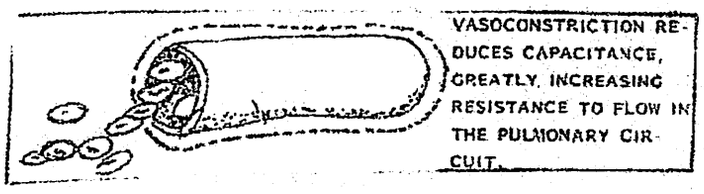
⇧

Normally, the pulmonary circulation is low-pressure circuit (mean pulmonary artery pressure < 20 mm Hg).

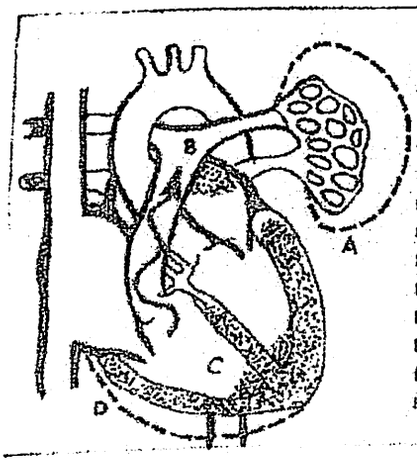
⇧

The right ventricle has a thin free wall and a large internal surface area compared with the left ventricle making it much less able to tolerate pressure overload.

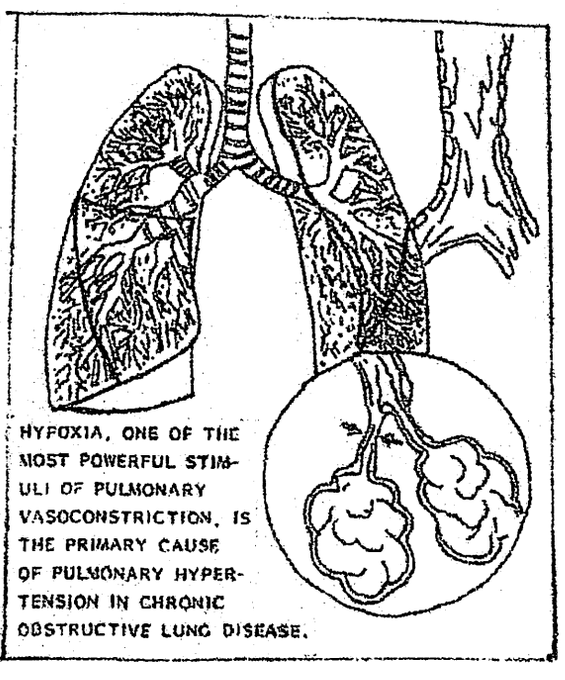




VASOCONSTRICTION REDUCES CAPACITANCE, GREATLY INCREASING RESISTANCE TO FLOW IN THE PULMONARY CIRCUIT.



IN SUMMARY, HYPOXIA ⟶ VASOCONSTRICTION ⟶ REDUCTION OF THE PULMONARY VASCULAR BED (A) ⟶ INCREASED RESISTANCE TO PULMONARY BLOOD FLOW AND PULMONARY HYPERTENSION (B) ⟶ RIGHT VENTRICULAR PRESSURE OVERLOAD (C) ⟶ EVENTUAL RIGHT HEART FAILURE (D).

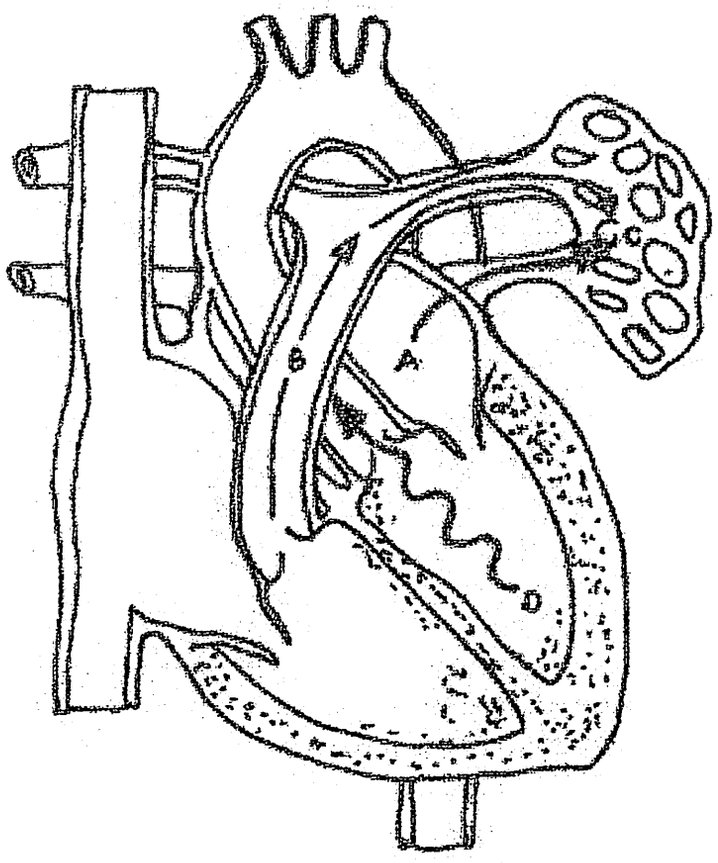


HYPOXIA, ONE OF THE MOST POWERFUL STIMULI OF PULMONARY VASOCONSTRICTION, IS THE PRIMARY CAUSE OF PULMONARY HYPERTENSION IN CHRONIC OBSTRUCTIVE LUNG DISEASE.

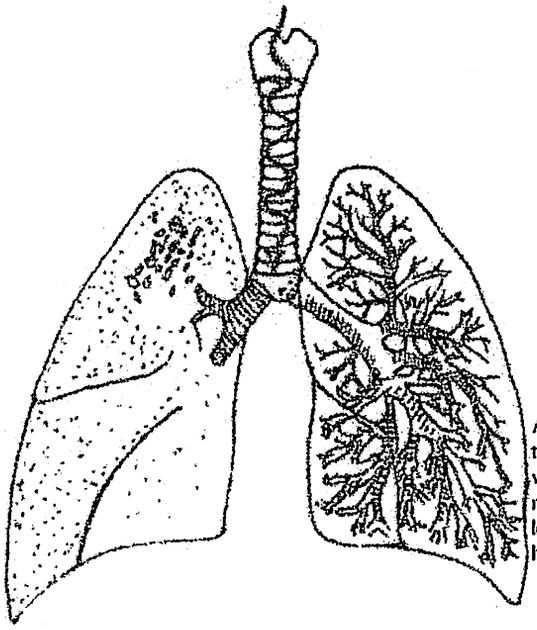
Pathologic Mechanisms Of Pulmonary Hypertension

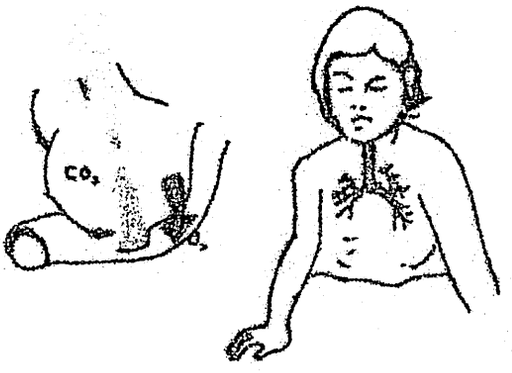
Passive.

The most common cause of right heart failure is left heart failure. Elevation of left atrial pressure (A) requires that pulmonary artery pressure (B) also increase to maintain forward flow through the lungs (C). The most common cause of elevated left atrial pressure is left ventricular failure. In the absence of left ventricular failure, left atrial pressure may be elevated by mitral stenosis or, more rarely, by a left atrial myxoma.



Vasoactive.

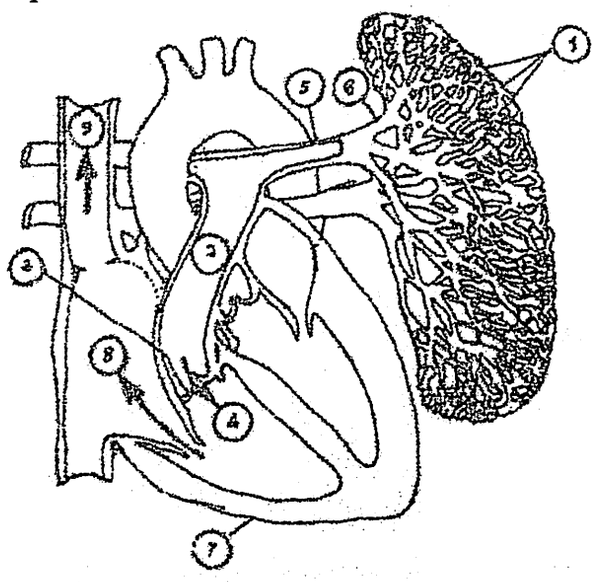




A wide range of stimuli cause pulmonary artery vasoconstriction among the most powerful being hypoxia. Those diseases, therefore associated with clinically significant hypoventilation (Pickwickian syndrome, neuromuscular diseases with respiratory depression, severe kyphoscoliosis, etc.) may lead to pulmonary hypertension, as may hypoxic vasoconstriction in chronic obstructive lung disease.

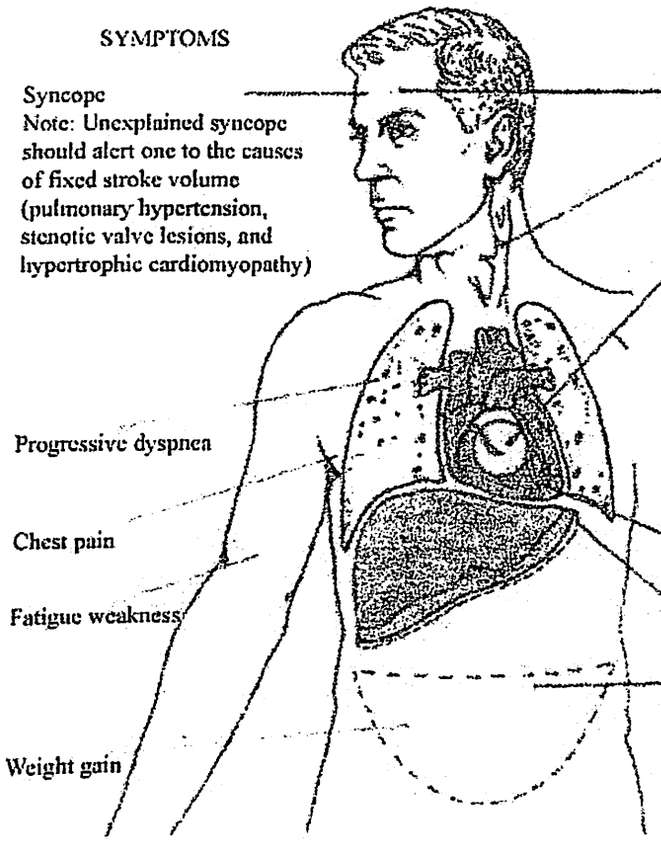
Obliterative
(with or without vasoconstriction)
Pulmonary thromboembolism mechanically obstructs pulmonary arteries. In addition, it may trigger biochemical changes leading to vasoactive pressure effects. Interstitial lung disease, collagen disease and severe chest deformities likewise reduce the pulmonary vascular bed, but because of the large

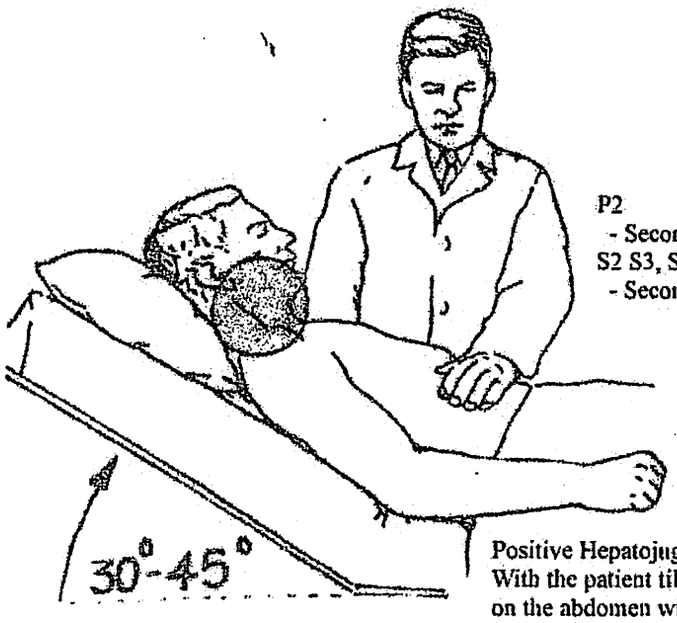
vascular reserve do not usually cause significant pulmonary hypertension unless a vasoactive component is associated.



Physical Findings In Pulmonary Embolism.
Multiple emboli (1) reduce the pulmonary vascular bed, increase resistance to flow, and lead to increased pressure in the pulmonary circuit (2). P_2 is accentuated (3); further pressure elevations may produce a regurgitant murmur at the pulmonary valve (4). Systolic murmurs may be heard over the pulmonary artery due to flow phenomena around an embolus (5); partial obstruction of peripheral branches (6) may produce bruits. Gallops indicate right heart strain (7). Under conditions of severe pressure elevation and poor cardiac reserve, a tricuspid insufficiency murmur (8) may appear, with evidence of increased venous pressure (9) and prominent "v" waves in the neck.

Clinical Features Of Pulmonary Hypertension: A Checklist



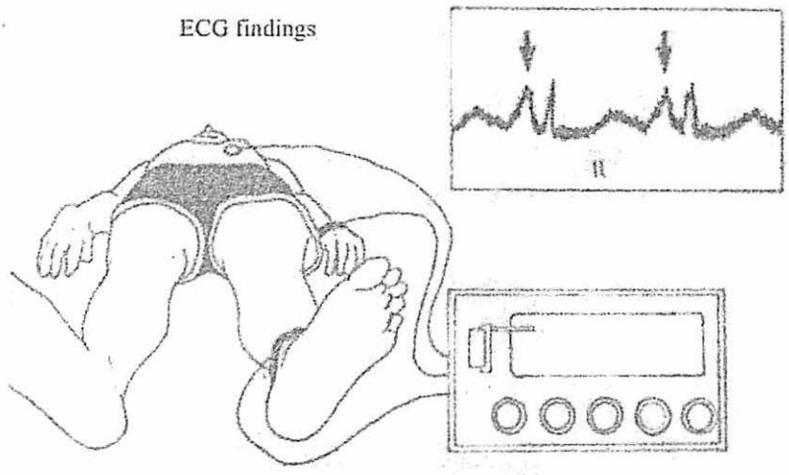


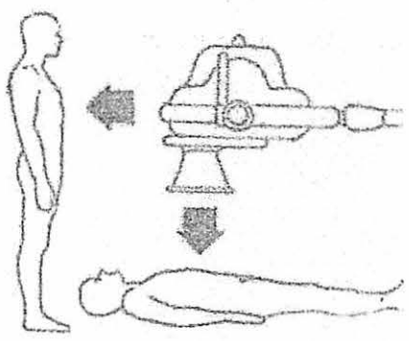
SIGHNS

P2
- Second Pulmonary Sound
S2 S3, S4
- Second, Third, Forth Heart Sounds

Positive Hepatojugular Reflex:
With the patient tilted in recumbency, pressure on the abdomen will accentuate jugular distension and pulsation if right ventricular failure is present.

ECG findings





·Increased P amplitude; rightward P axis, "P pulmonale"
(tall, pointed P waves in II, III, AVF)
·Right axis deviation; right ventricular strain
·Right ventricular hypertrophy patterns (R>S in V1; S in V5 and V6)
·Incomplete RBBB
RBBB - right bundle branch block

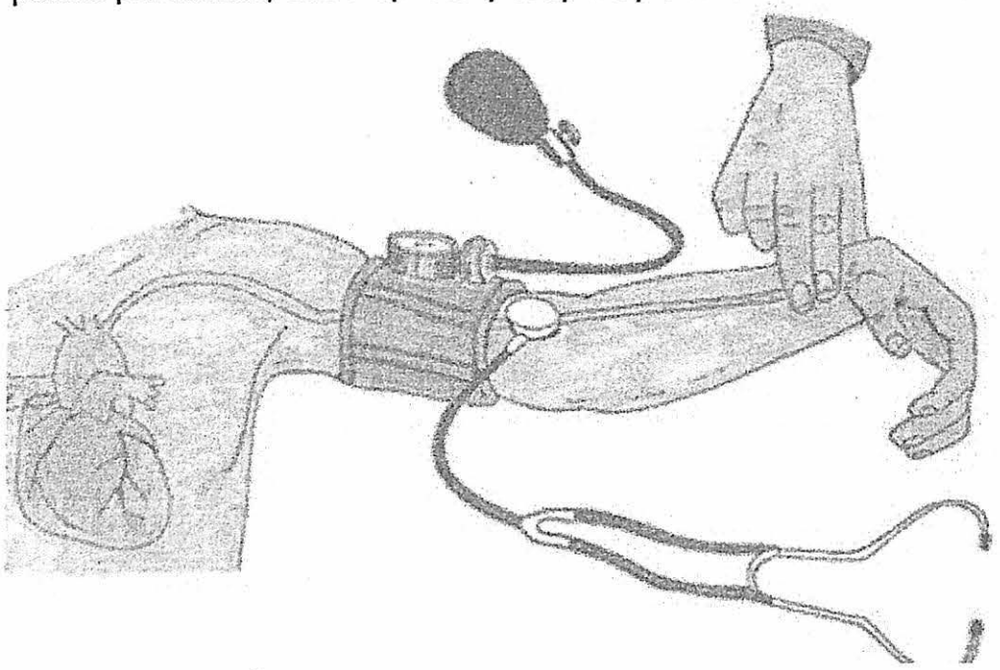
Chest x-ray findings

·Enlarged main pulmonary artery
·Enlarged right inerlobar artery
·Enlarged left descending artery

SISTEM PERNAFASAN

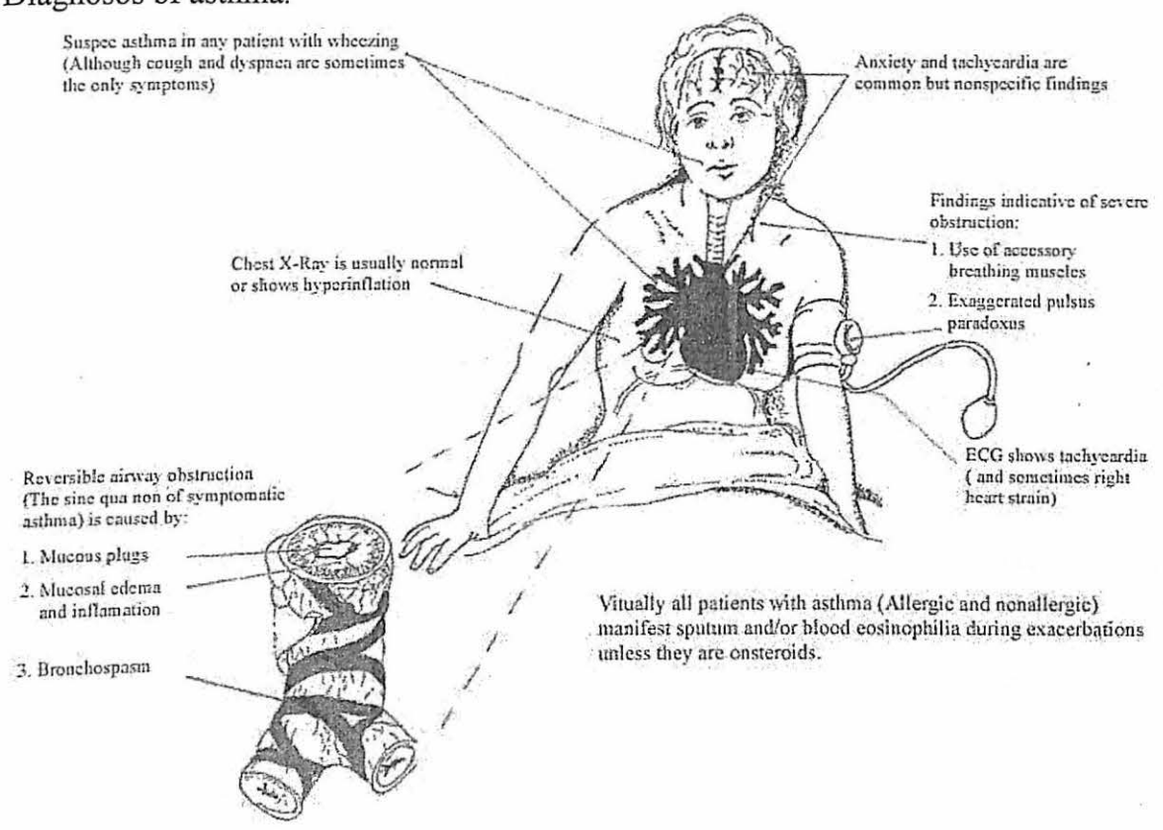
Modul 17 Pulsus Paradoxus Is A Valueable Clinical Sign In Severe Asthma.

This sign isn't really a paradoxical phenomenon, but rather an exaggeration of a normal response. Systolic blood pressure normally drops less then 5 mm Hg on deep inspiration; in pulsus paradoxus, this inspiratory drop may exceed 10 or even 20 mm Hg.



When the sign was first introduced, it was regarded as the exclusive hallmark of pericardial constriction. It is now also a useful diagnostic indicator in asthma. The more severe the asthma, the greater the inspiratory decrease in pulse amplitude.
(Note: pulsus paradoxus may also accompany pulmonary embolism, myocardiopathy, or hypovolemia).

Diagnosos of asthma.



Remember: Because physical and other findings do not reliably assess the degree of airway obstruction, pulmonary function tests are necessary for objective measurement.

SISTEM PERNAFASAN

Modul 18 The Lung.

I am John's right lung, and I claim the privilege of speaking since I am slightly larger than my partner in the left side of his chest. I have three lobes - sections - while the left has only two.

John would be surprised if he could see me. He thinks of me as a kind of hollow, pink bladder hanging in his hest. I'm not like that at all. I am not hollow – if you cut through me, I would look something like a rubber bath sponge. And I am not pink. I was when john was a baby. Now a quarter of a million cigarettes plus thousands of millions of breaths of dirty city air later. I am slate-grey, mottled black.

There are three separate, sealed compartments in John's chest: one for me, one for the left lung, one for his heart. I hang loosely in my compartment, filling it completely, and weigh a little over a pound. I have no muscles and hence play a passive role in breathing. There is a slight vacuum in my compartment, so when John's chest expands, I expand. When John exhales, I collapse. It is simply a recoil mechanism. Should John puncture his chest wall in an accident so that my vacuum is broken, I'll hang closely, doing no work, until healing takes place and the vacuum is re-established.

Take a look at my architecture. John's windpipe divides at its lower end into two bronchial tubes - one for my partner. Then branching begins in me - like an upside-down tree . First the larger bronchi, then the bronchioles, one hundred of an inch in diameter. These are simply air passages. My real work is done in my alveoli - grape - like bunches of minute air sacs. I have some 250 million of these. Flattened out they would probably cover half one tennis court.

Each alveolus is covered with a cobweb of capillaries. Blood is pumped by the heart into one end of a capillary. Red cells pass through single file - passage taking about second – and a remarkable this takes place. Through the gossamer membrane of the capillary wall the cells diffuse their cargo of carbon dioxide into my alveoli. At the same time, my cells pick up oxygen going the other way. It's a kind of gaseous swop snop - blue blood flowing in at one end of the capillary, emerging red at the other.

John's more important body organs - notably the heart - are under automatic control. Most of the time this is true of me, too, though I am under voluntary control as well. As a child, John had temper tantrums and would sometimes hold his breath until he turned a faint shade of blue.

His mother worried - unnecessarily. Long before he got into any real trouble, automatic respiration would take over.

My automatic breathing control is in the medulla oblongata - the bulge where the spinal cord joins the brain. It's an amazingly sensitive chemical detector. Labouring muscles burn oxygen rapidly and pour out waste carbon dioxide. As it accumulates, the blood becomes

slightly acid. The respiratory control center detects this instantly - and orders me to work faster. Let the levels rise high enough as when John does heavy exercise, and it orders deeper breathing as well one's "second wind".

Sitting up, John needs approximately 16 quarts of air a minute: walking requires 24; running 50. Lying in bed, John requires only about eight quarts a minute. To take this in he breathes about 16 times a minute - a pint of air each time. (This only partially inflates me. I can hold eight times as much). Even so, not all of that one-pint breath reaches me, one-third of it shuffles aim lessly in and out of the wind-pipe and other air passages.

I like my air just about as moist and warm as that in a tropical swamp. Producing this very special air in the space of a few inches is quite a feat. The same tear glands that bathe John's eyes, plus other moisture-secreting glands in his nose and throat, produce as much as a pint of fluid a day to humidify my air. Surface blood vessels along the same route wide open on cold days, closed on warm days - take care of the heating job.

There is an almost endless list of things that can cause me trouble. Each day, John breathes in a variety of bacteria and viruses. Lysozyme in the nose and throat; a powerful microbe slayer, destroys most of these. And those that slip into my dark, warm, moist passages – a microbial happy hunting ground - I can usually handle. Phagocytes patrol my passages mid destroy invaders.

Dirty air of course, is my biggest challenge. Other organs lead sheltered, protected lives, but for all practical purposes I am outside John's body-exposed to environmental hazards and contaminants. I am really quite delicate, and it's a wonder I am able to survive at all, having to deal with such things as sulphur dioxide, benzopyrene and nitrogen dioxide. Since some of them actually melt nylon stockings, you can guess what they do to me.

My air-cleaning process - such as it is - begins with hairs in the nose, which trap large dust particles, sticky mucus in nose, throat and bronchial passages acts as fly -paper to trap finer particles. But the real cleaning job falls to the cilia. These are microscopic hairs - tens of millions of them - along my air passages. They wave to and fro, like wheat in the wind, about 12 times a second. Their upward thrust sweeps mucus to the throat, where it can be swallowed.

If John could watch my cilia under a microscope, he'd see that if cigarette smoke or badly contaminated air is blown on them, the wind-in-the-wheat field action stops. A temporary paralysis sets in. Let this irritation continue long enough and the cilia wither and cie, never to be replaced.

After 30 years of smoking, John has lost most of his cilia, and mucus-secreting membranes in his air passages have thickened to three times normal size. John is in actual danger or drowning. If enough mucus drops down into my air sacs, it halts breathing just as effectively as a lungful of water. One thing saves John from this: his noisy) inefficient smoker's cough, which has replaced the quiet efficiency of the cilia. John might remember that it's the only cleaning method left to me - and be cautious about taking cough-suppressing drugs.

Much of the time, John is asking me to breathe real garbage. Some of the particles clog my smaller passages, and some actually sear my tissues. The fragile walls of my alveoli lose elasticity. They don't collapse the way they should when I exhale. (thus it is possible to breathe in but not out). Carbon dioxide is trapped in them, and they can no longer contribute oxygen to the blood or extract

waste carbon dioxide. The result is emphysema-a fearsome trial that breath represents a fight for survival.

Although John doesn't know it, this has already happened to a few millions of my alveoli. Since John has about eight times the lung capacity he needs for desk work, he still has plenty in reserve. But lately he has noticed that even a small amount of exertion brings on breathlessness. I'm warning him.

John should heed the saying, "if you are aware that you have lungs, you are already in trouble", and take better care of me. This means giving me better air to breathe. The big thing, of course, would be to give up smoking. A little more exercise and more sensible eating would be in order. Any general body exercise - climbing stairs, walking, jogging, sports - forces me to breathe more deeply, which is all to the good. And there are exercises for me alone. Ordinarily, the best breathing is deep breathing - more air at a slower pace. John could practice abdominal breathing, the way babies and opera singers do it: not by inflating the manly chest, but by dropping the diaphragm down then air is sucked into even my deepest alveoli.

John's breathing will also be helped if he sleeps in a warm bedroom with the door ajar to let in fresh air. Cold air from an open window is likely to make him cough. A few times each day, John could also give me a housecleaning. He thinks that with a normal exhalation I'm empty by no means. Let him blow out all the air he can via his mouth. Then if he will purse his lips, he can do quite a lot more blowing. If he does this while smoking, he will see something that should make him pause: smoke trailing out enough his pursed lips that would normally be left in me to stagnate.

It all adds up to this: most of my neighbouring organs can absorb an enormous amount of abuse without complaint. I can't. Nature hasn't equipped me with all the defences I really need in today's world. That's why a variety of lung diseases have reached epidemic proportions.

John, take heed!

(Chapter from "The Body And How It Works" by JD. Ratcliff, Reader's Digest home doctor library).

SISTEM PERNAFASAN

Modul 19 Behavioral Distribution Of V/Q Inequality.

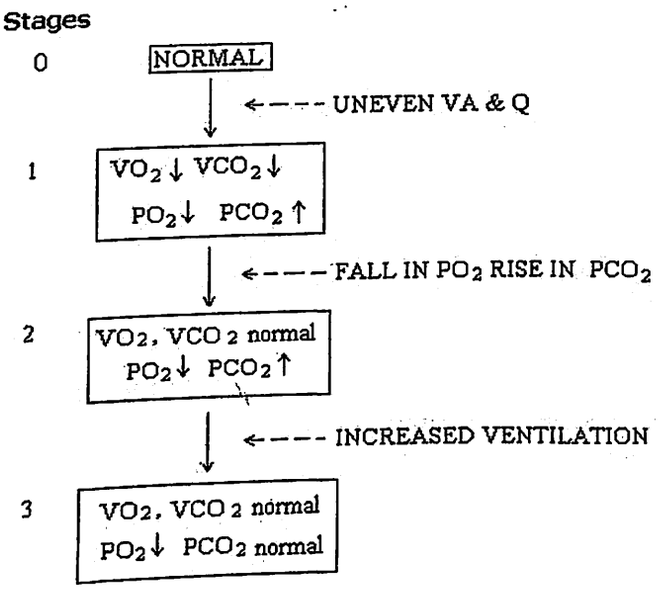
Some insight into the way in which the diseased lung copes with the imposition of ventilation-perfusion inequality can be obtained by analysing the behaviour of theoritical distribution of V/Q ratios.

1.Imagine a lung which is uniformly ventilated and perfused and transfering normal amounts of O_2 and CO_2

2.Now suddenly the matching of ventilation and blood flow is suddenly disturbed (everything else remain constant).

WHAT HAPPENS TO GAS EXCHANGE?

3.To answer this, it is useful to identify conceptual "stages" in the development of V-Q inequality and the corresponding changes that occur in gas exchange.



a. Initially, we have a normal lung, no uneven ventilation and blood flow (STAGE 0)

b. Then some kind of disturbances cause mismatch of ventilation and blood flow.
This results in - Fall in O_2 uptake
- Fall in CO_2 output
- A fall in PO_2 - In arterial blood
- A rise in PCO_2 - In arterial blood

STAGE 1:
This situation is only transient because the lung must restore O_2 uptake and CO_2 output to normal to satisfy the demands of the metabolising tissues. This is done by a fall in PO_2 and a rise in PCO_2 in arterial and mixed venous blood.

c. This fall in mixed venous PO_2 increases the amount of O_2 that can be loaded by the blood in its passage through the pulmonary capillaries. Similarly this process assist in unloading CO_2 giving rise to the situation seen in STAGE 2. Here O_2 uptake and CO_2 return to normal. However, there is an abnormally low $PO_2$and high PCO_2 in the arterial and mixed venous bloods.

d. As STAGE 2 is not constant, further change usually occur. The central chemoreceptors normally do not like to see the rise in arterial PCO_2 and therefore increase ventilation to the alveoli until the arterial PCO_2 is brought back to normal. This in a way increases arterial PO_2 to some extent giving rise to STAGE 3. Here it shows:

- normal O_2 uptake
- normal CO_2output
- normal arterial PCO_2

BUT - a reduced arterial PO_2

NOTE:
a. These conceptual stages are useful to identify these so-called stages of gas exchange, but they do not occur sequentially.
b. Some patients do not make the transition from STAGE 2 to STAGE 3, or having made it, revert from STAGE 3 to STAGE 2. These are generally patients who have a very high work of breathing, often because their airway resistance is high and they apparently elect to increase their arterial PCO_2rather than accept the penalty of increasing their ventilation. Furthermore, the initial disadvantage of a fall in arterial pH is soon lost as the kidneys retain bicarbonate resulting in a fully compensated respiratory acidosis. Nevertheless, this is usually a relatively late stage in lung disease with a corresponding poor prognosis and the term 'respiratory failure' is often reserved for this situation.

QUESTION:
Why does the increase in ventilation to the alveoli in the transition from STAGE 2 to STAGE 3 correct the raised arterial PCO2 but not return the arterial PCO2 to its normal level?

ANSWER:
This is due to the different shapes of the O_2& CO_2 disassociation curves.
CO_2 dissociation curve is so nearly linear in its working range, that an increase in ventilation to a lung with substantial V-Q inequality continues to be effective in eliminating more CO_2.
With reference to O_2 dissociation, due to its very nonlinear curve, the increase in ventilation typically only results in modest gain in arterial PO_2. In essence this is because the high V/Q ratios which are operating high on the O_2dissociation curve are unable to make up for the depressing effect on the arterial PO_2 of the low V/Q ratio units.

Conclusion: V/Q inequality does not intefere with CO_2 elimination.

SISTEM PERKUMUHAN

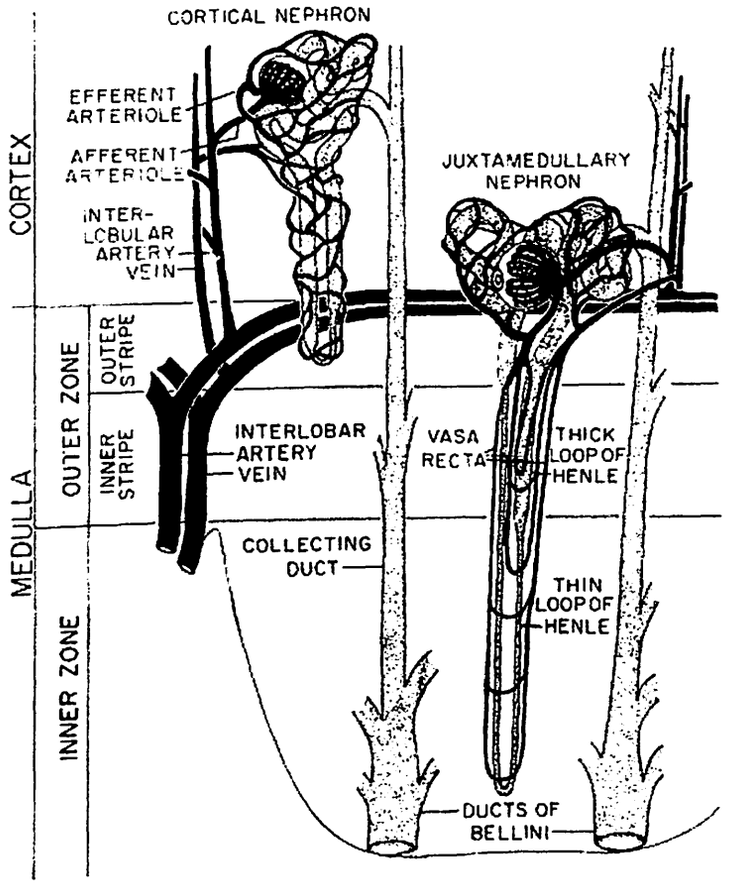
SISTEM PERKUMUHAN

Modul 1 Physiology of the kidney and body fluids.
Modul 2 Countercurrent Multiplication by the Loop of Henle.
Modul 3 Countercurrent exchange by the vesa recta.
Modul 4 Recent modifications of the countercurrent hypothesis by Stephenson and Kokko and Rector.
Modul 5 Action of angiotensin II.
Modul 6 Clearance of inulin.
Modul 7 Urea clearance.
Modul 8 Creatinin clearance.
Modul 9 PAH clearance.
Modul 10 Acidification of urine.
Modul 11 Tubular maximum.
Modul 12 Production of a concentrated urine.
Modul 13 Overview of renal system.
Modul 14 Pathways by which sodium and water excretion are decreased in response to severe sweating.
Modul 15 Pathway by which the GFR is decreased when plasma volume decreases.
Modul 16 Summary of the processes occuring in urine formation.

Menu Utama

SISTEM PERKUMUHAN

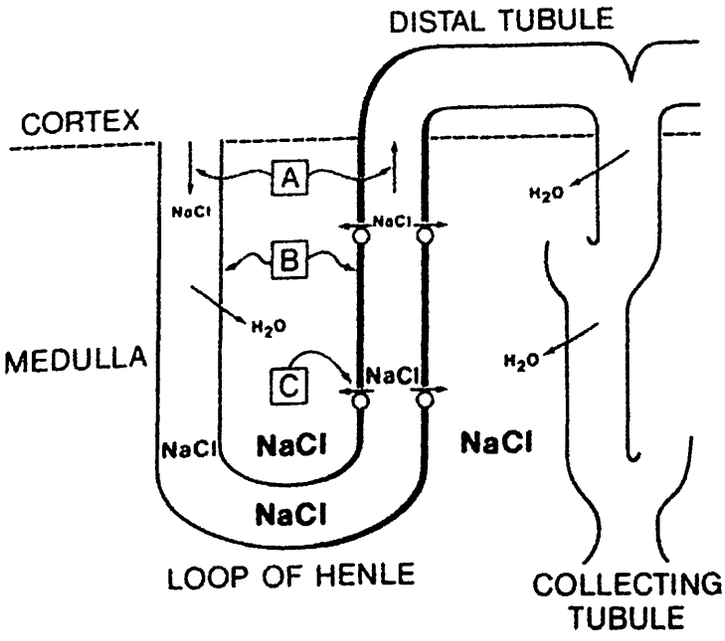
Modul 1 Physiology of the kidney and body fluids.



Comparison of the blood supplies of cortical and juxtamedullary nephrons.

SISTEM PERKUMUHAN

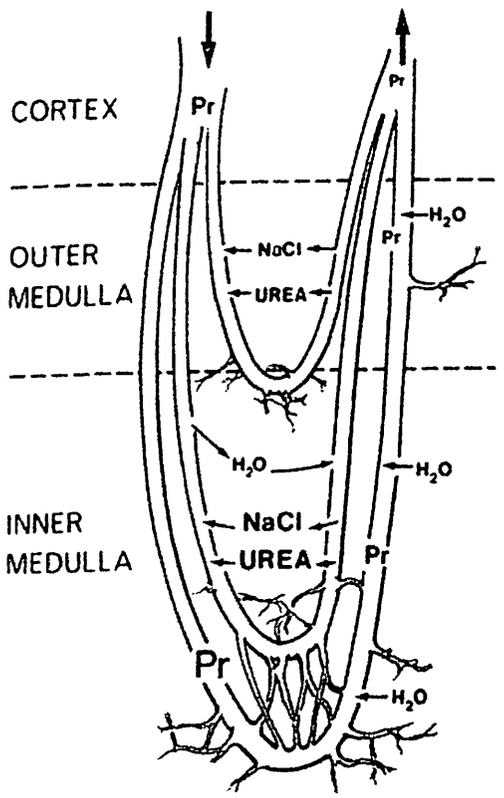
Modul 2 Countercurrent Multiplication by the Loop of Henle.



According to the countercurrent hypothesis there are three requirements for the loop to act as a multiplier: (A) counter-current flow; (B) difference in epithelial permeability; and (C) a source of energy. The thickened lining of the ascending limb and first part of the distal tubule indicates a water-impermeable epithelium, in contrast to the water-permeable descending limb. The source of energy is supplied by active reabsorption of sodium chloride. Note that the progressive increase in sodium chloride concentration (indicated by the size of type) in the medulla osmotically extracts water from the descending limb and collecting duct.

SISTEM PERKUMUHAN

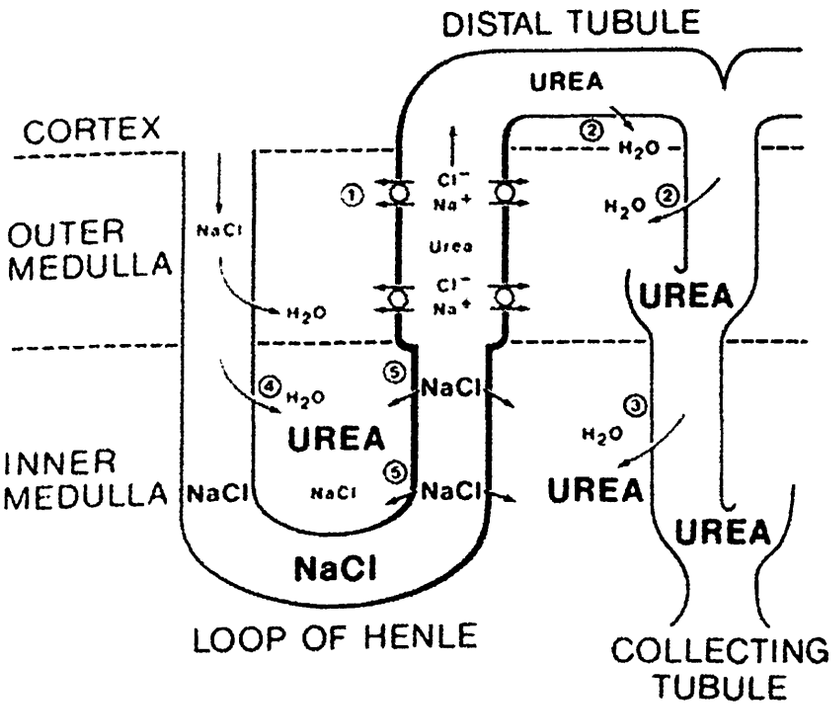
Modul 3 Countercurrent exchange by the vesa recta.



The medullary circulation, unlike the loops of henle, consists of a network of channels with main thoroughfares (the vasa recta) and branch connections. Pr denotes plasma protein. The size of type indicates the relative concentrations of each solute with respect to its location in the medulla but not necessarily with respect to other solutes. The progressive rise in the concentration of sodium chloride and urea in the medullary interstitium is due to the loop of henle and collecting tubule. Since the capillaries are permeable to sodium chloride and urea, these solutes enter descending vasa recta and leave ascending vasa recta. This transcapillary exchange helps "trap" the solutes in the medulla. Conversely, water leaves the descending vasa recta, causing the plasma protein concentration to increase. In the ascending vasa recta the sum of oncotic (that due to plasma protein) and osmotic (that due to nonprotein solutes) pressures results in capillary fluid uptake. Thus, water reabsorbed from the collecting tubule and henle's descending limb is removed from the medulla and returned to the general circulation. Vasa-recta function in a dual capacity, trapping solute and removing water, to preserve the hyperosmolality of the renal medulla.

SISTEM PERKUMUHAN

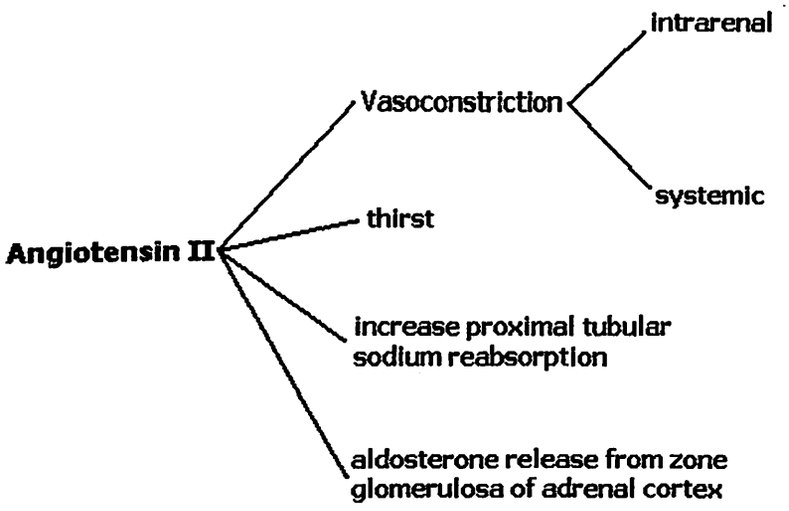
Modul 4 Recent modifications of the countercurrent hypothesis by Stephenson and Kokko and Rector.



Both the thin ascending limb in the inner medulla and the thick ascending limb in the outer medulla, as well as the first part of the distal tubule, are impermeable to water, as indicated by the thickened lining. In the thick ascending limb, active chloride reabsorption, accompanied by passive sodium movement (1),renders the tubule fluid dilute and the outer medullary interstitium hyperosmot1c. In the last part of the distal tubule and in the collecting tubule in the cortex and outer medulla, water is reabsorbed down its osmotic gradient. (2), increasing the concentration of urea that remains behind. In the inner medulla both water and urea are reabsorbed from the collecting duct. (3), some urea re-enters the loop of henle (not shown). This medullary recycling of urea, in addition to trapping of urea by counter-current exchange in the vasa recta (not shown), causes urea to accumulate in large quantities in the medullary intesstitium (indicated by the large type), where it osmotically excracts water from the descending limb. (4), and thereby concentrates sodium chloride in descending limb fluid. When the fluid rich in sodium chloride enters the sodium chloride permeable (but water permeable) thin ascending limb, sodium chloride moves passively down its concentration gradient. (5), rendering the tubule fluid relatively hypo-osmotic to the surrounding interstitium.

SISTEM PERKUMUHAN

Modul 5 Action of angiotensin II.



SISTEM PERKUMUHAN

Modul 6 Clearance of inulin.

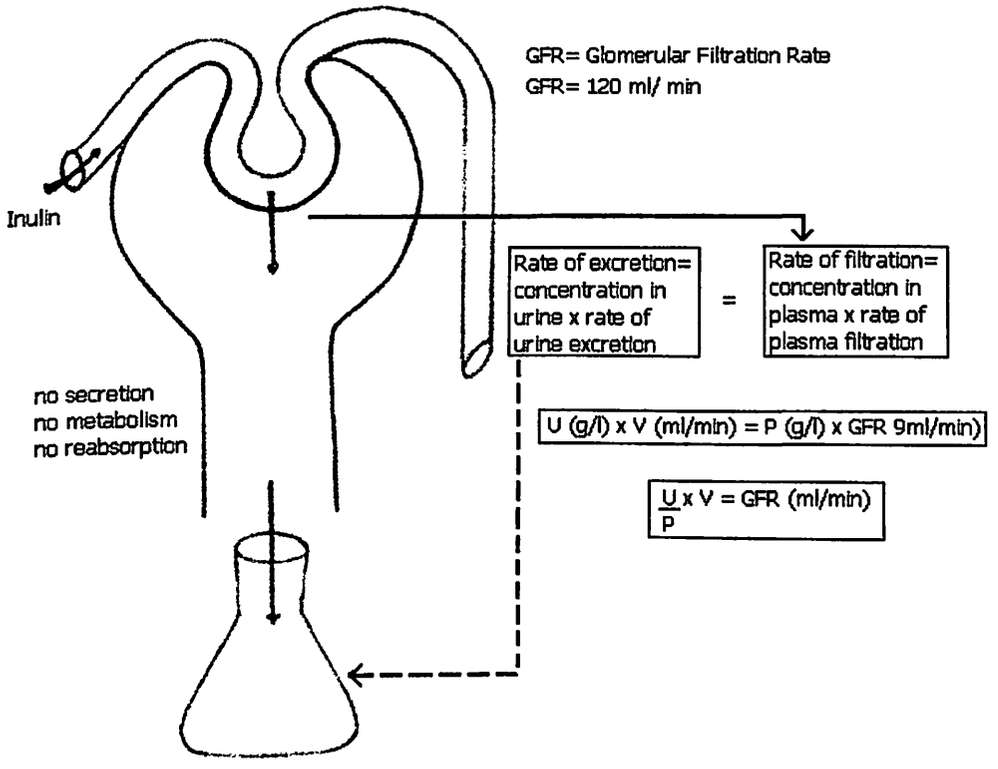
Measurement of glomerular filtration rate.

To measure GFR the indicator substance used must be:

1. inert
2. does not influence renal function
3. enters tubules by FILTRATION only
4. is not reabsorbed or secreted

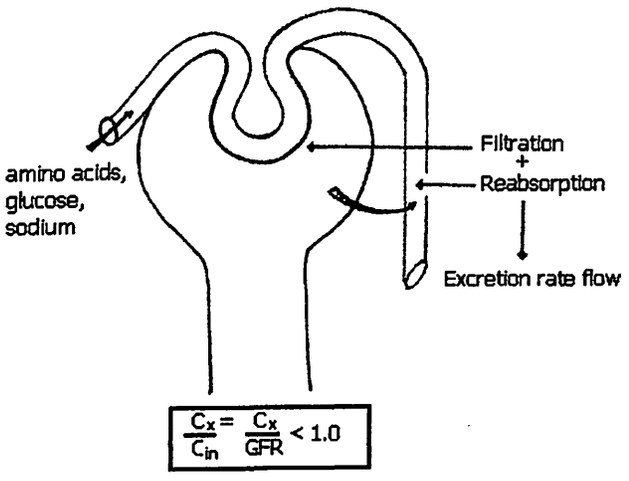
Such substance is INULIN. Thus inulin clearance = Glomerular Filtration Rate

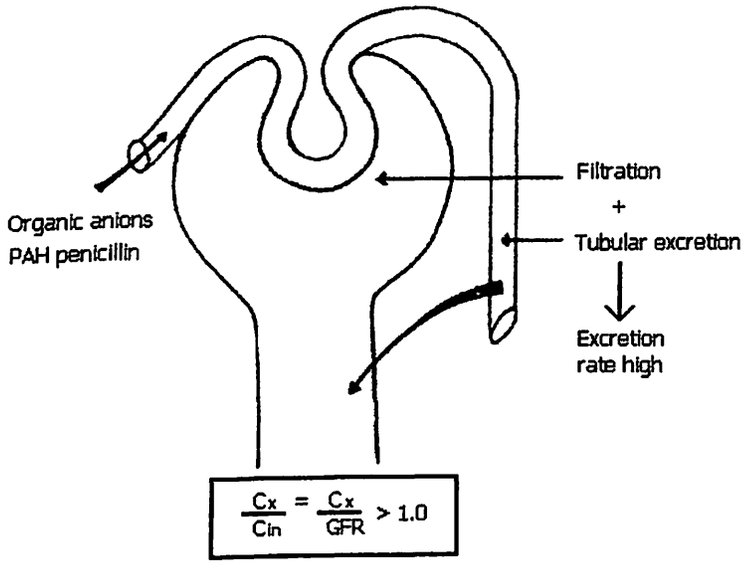
GLOMERULAR FILTRATION RATE :



CLEARANCE RATIOS RELATIVE TO FILTRATION :

The clearance of any substance (Cx) can be compared to the GFR by its clearance ratio.



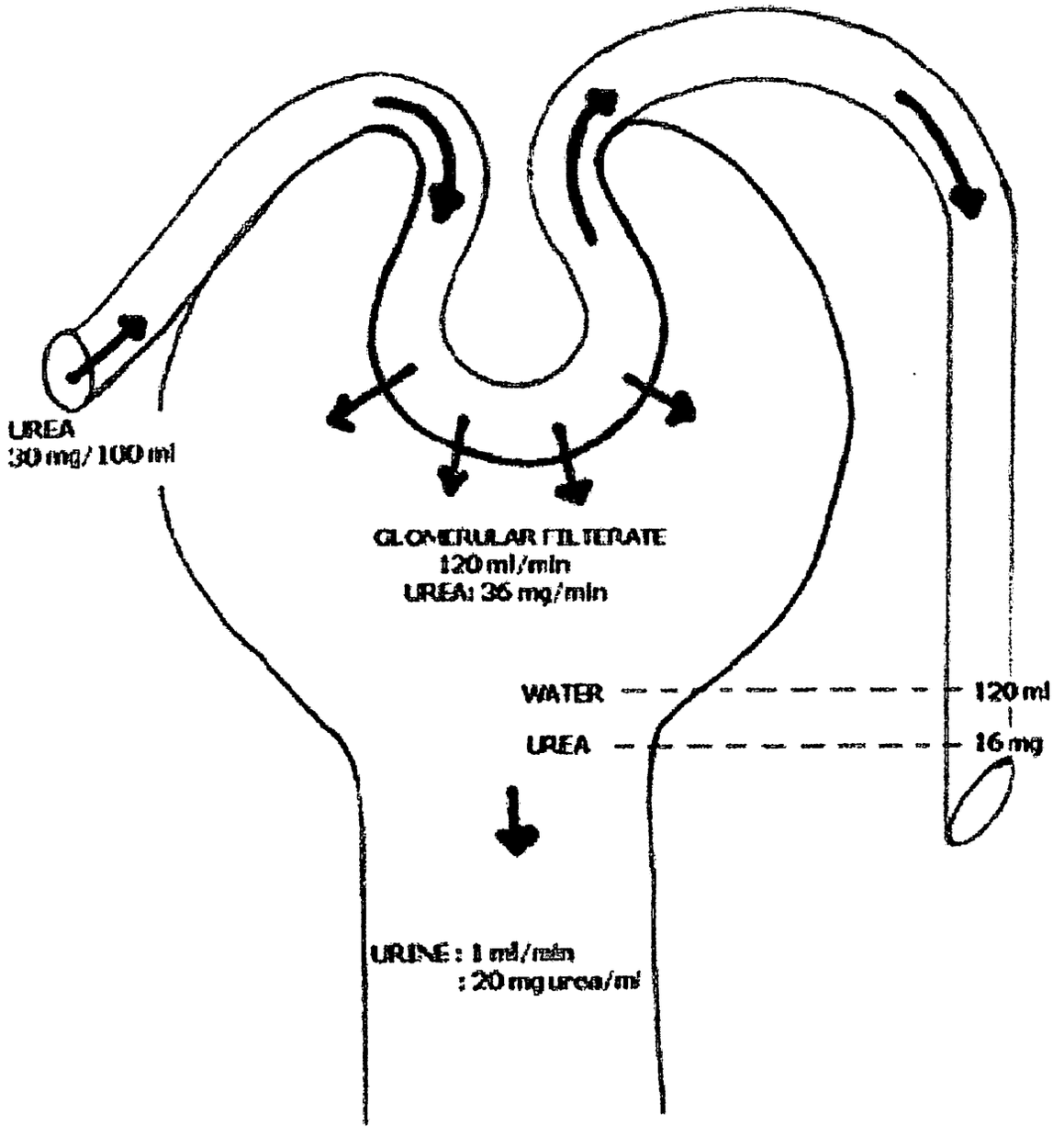


Clearance ratio of < 1.0 : the substances has been filtered and _____.
Clearance ratio of > 1.0 : the substance has been filtered and _____.
Clearance ratio of = 1.0 : the substances has been but not _____.

SISTEM PERKUMUHAN

Modul 7 Urea clearance.

Urea, like inulin, is filtered from plasma into the glomerulus.
Unlike inulin, some urea diffuses back into thr blood stream from the tubules.
Urea clearance is used as a test for renal function.



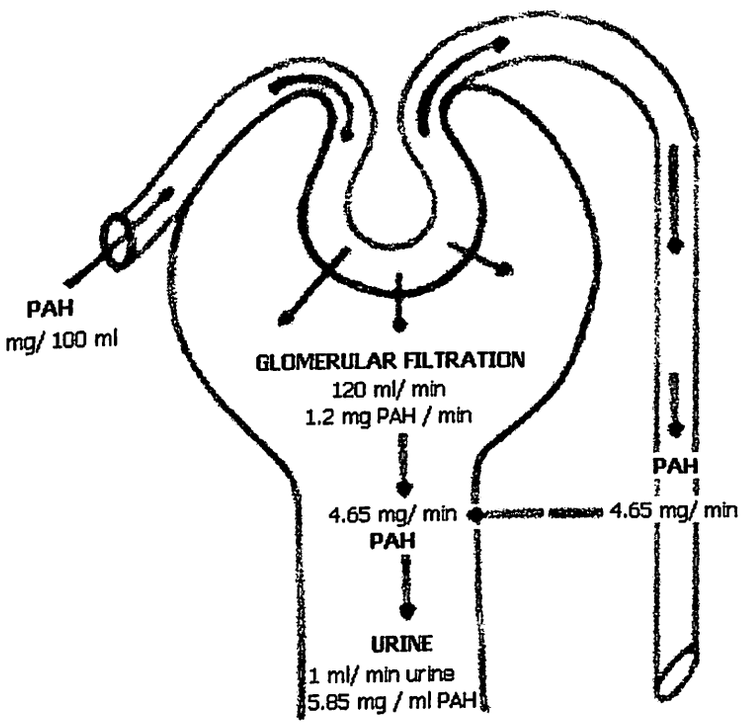
Calculate the urea clearance.
What is the clearance ratio of urea?
What does the ratio indicate?

SISTEM PERKUMUHAN

Modul 9 PAH clearance.

1. FICK PRINCIPLE: Ratte of removal of a test substance from blood is measure of blood flow to the organ.
2. Any substance which is removed from plasma during a single passage through the kidney can be used to measure RENAL PLASMA FLOW (RPF).
3. Para amino hippurate (PAH) is effectively filtered at the glomerulus and also secreted by the tubular cells. It is used to measure RPF.

PAH is an index of glomerular filtration as well as tubular function.



Calculate the RPF using the figures given above.

SISTEM PERKUMUHAN

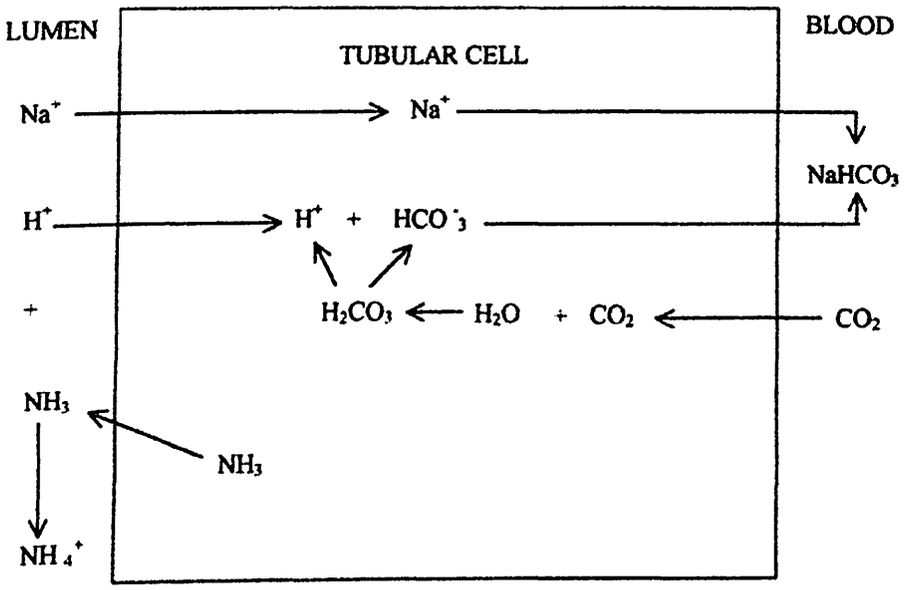
Modul 10 Acidification of urine.

Under normal condition, acid producing foods predominate over alkali producing food. Free H+ also results from production of HCL by gastric cells.

The body thus has to maintain an average pH (7.4) in the face of forces which tend to lower the pH of ECF.

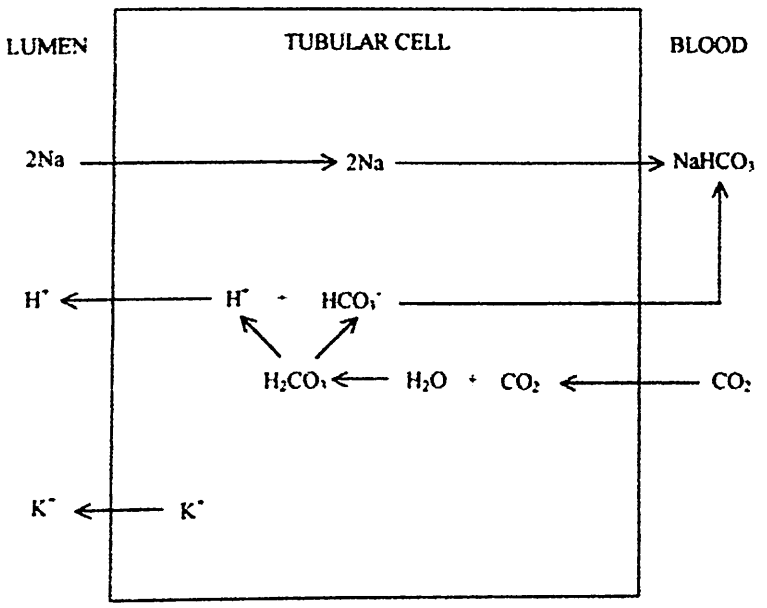
RENAL METHODS OF INCREASING H+ SECRETION AND CONSERVING BASE :

A. Secretion of NH4



NH3 is derived from the deamination of amino acids and glutamine.
It diffuses into the tubular lumen where it reacts with H+ to form NH4.

B. Secretion of potassium



1. H and K move out in exchange for 2 Na molecules.
2. Urine leaving the distal tubule has been acidified but is still hypotonic.

SISTEM PERKUMUHAN

Modul 11 Tubular maximum.

1. TUBULAR MAXIMUM: maximum rate of absorption or secretion of a substance in renal tubules in mg/min.
2. Depends on:
   i. transport systems
   ii. associated carrier proteins and enzymes
3. Once Tm is exceeded, the substance will be found in urine.

The Tm values of any substances can be found by utilizing INULIN CLEARANCE to measure GFR.

Tm can then be calculated by:

Urine concentration (A) x urine flow (V) = (plasma concentration (A) x GFR) – Tm

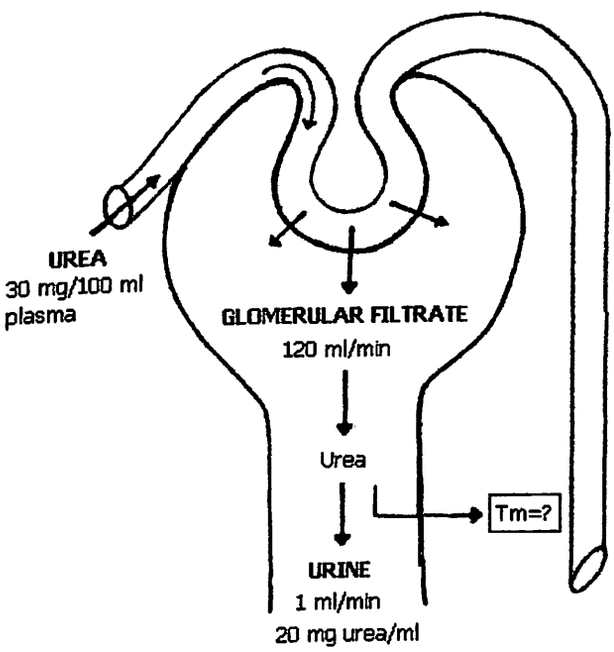
Amount of A appearing in urine (UxV) = filtered load (GFR or C) – amount of absorbed by tubular cells (Tm)

Some Tm values:

· Tm for absorbed substances: glucose - 350
plasma protein - 30

· Tm for secreted substances: PAH - 80
Creatinine - 16

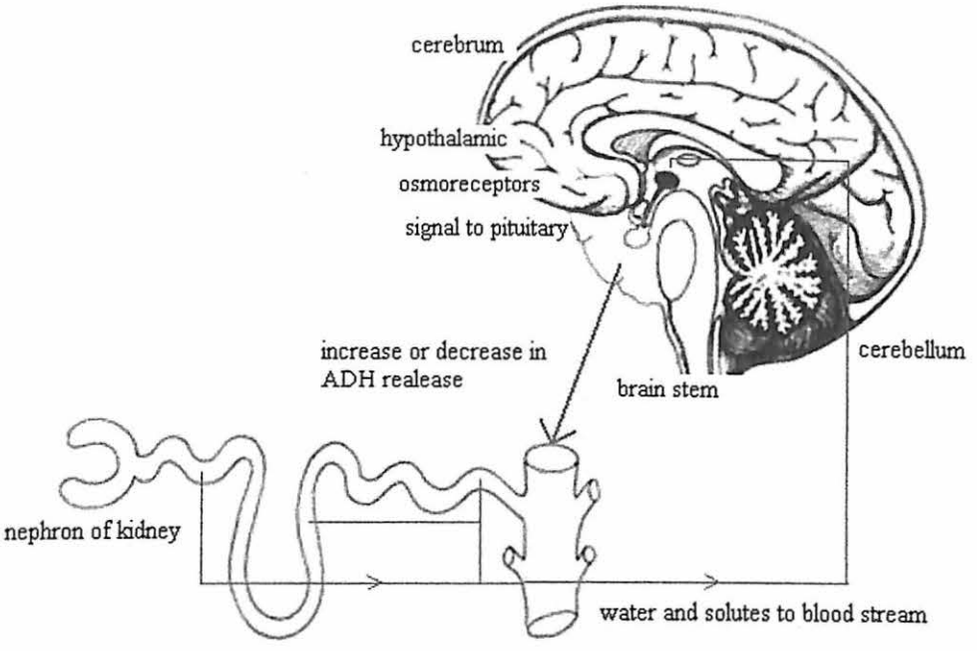
Clinical significance: reflects the functional mass of proximal tubular cells.



Calculate the Tm using the figures given above.

SISTEM PERKUMUHAN

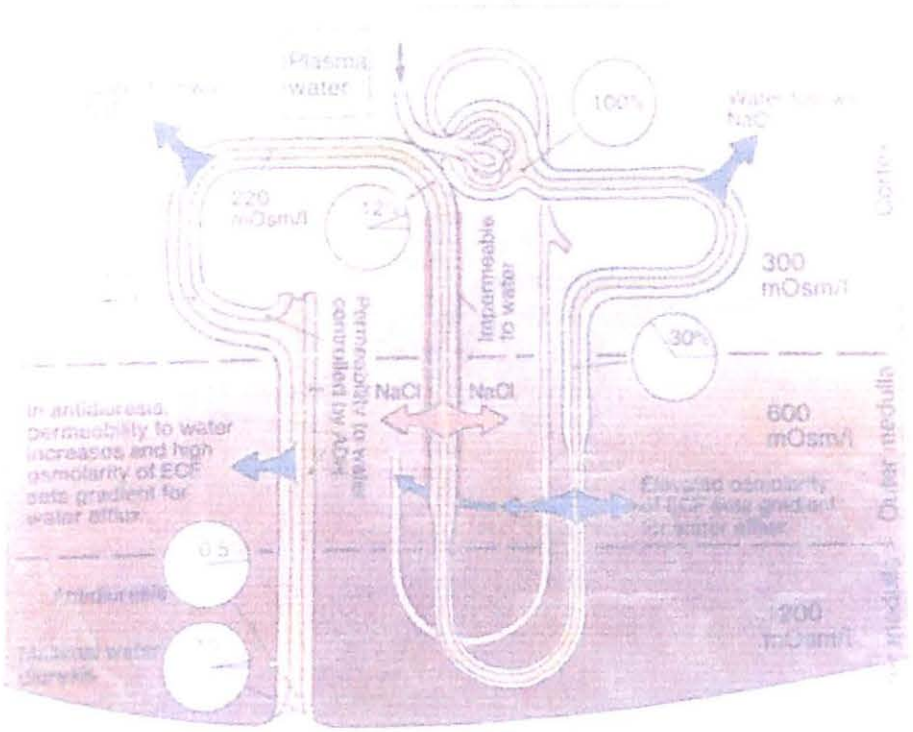
Modul 12 Production of a concentrated urine.



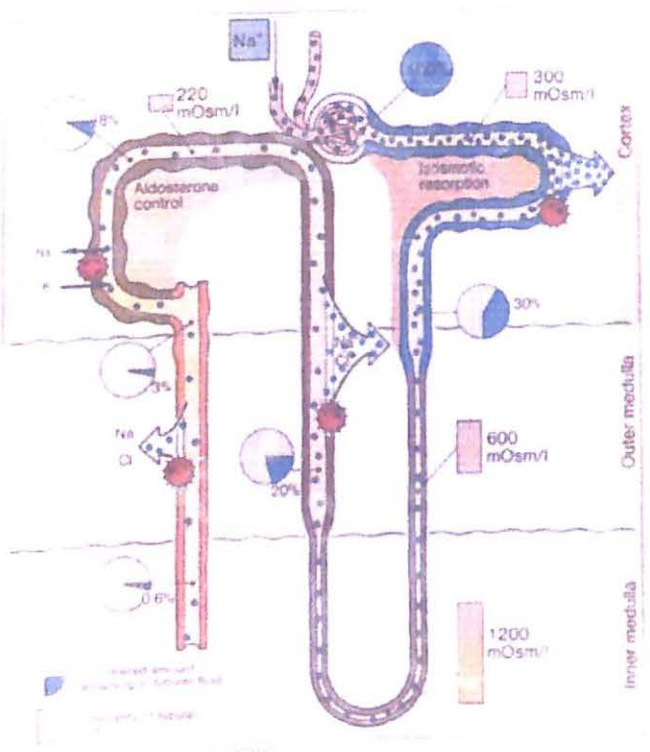
1. The collecting tubules receive HYPOTONIC solution from the distal tubule.
2. These collecting tubules run through the medulla towards the tips of the pyramid.
3. REMEMBER: in the medulla, the countercurrent multiplier creates an increasingly hypertonic interstitium around
   the collecting tubules. There is a tendency for water to leave the tubule by osmosis but this is not permitted unless
   the tubule become permeable to water in the presence of ADH.
4. ADH is produced in the hypothalamus and is stored and release from the posterior lobe of the pituitary. It increases
   permeability of collecting tubules to water, thus allowing its passage into the interstitium (see above).
5. As fluid pass through the collecting tubules it loses more and more water under the influences of ADH. Thus the urine
   excreted will become HYPERTONIC.

SISTEM PERKUMUHAN

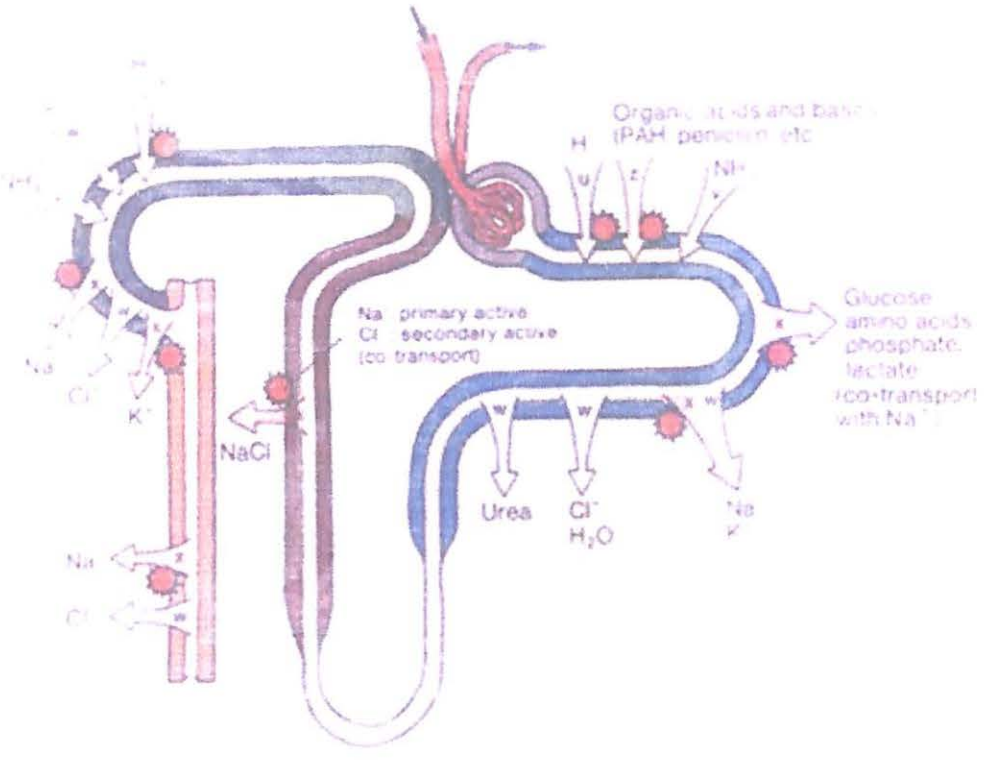
Modul 13 Overview of renal system.



A Fluxes of water in the nephron



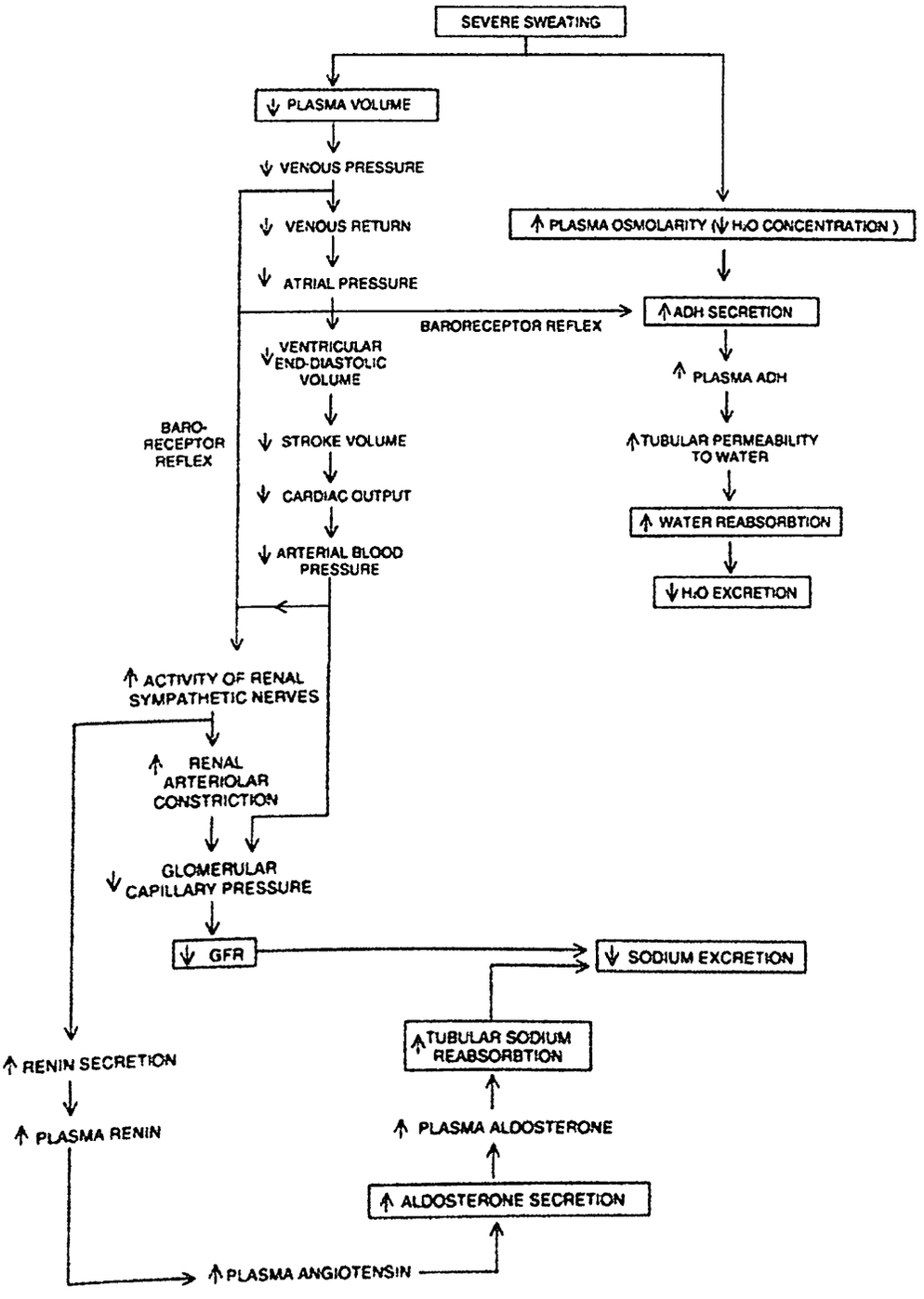
B Fluxes of Na+ in the nephron



C Locus of transport processes in the nephron (simplified)

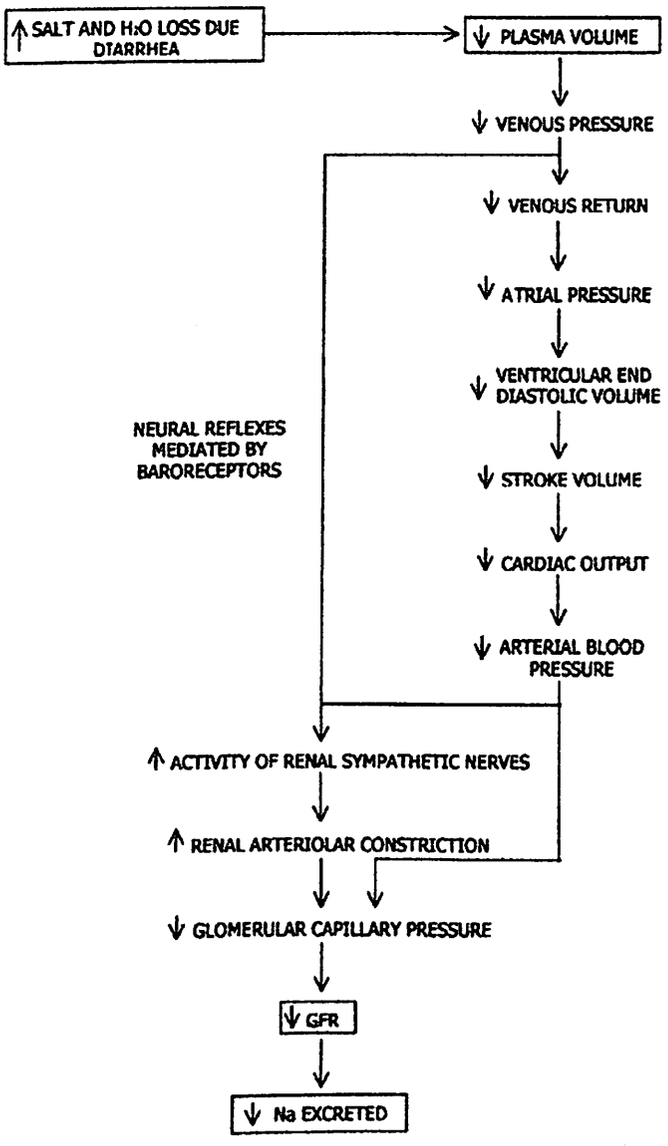
SISTEM PERKUMUHAN

Modul 14 Pathways by which sodium and water excretion are decreased in response to severe sweating.



SISTEM PERKUMUHAN

Modul 15 Pathway by which the GFR is decreased when plasma volume decreases.



The baroreceptors which initiate the sympathetic reflex are probably located in large veins and the walls of the heart, as well as in the carotid sinuses and aortic arch.

SISTEM PERKUMUHAN

Modul 16 Summary of the processes occuring in urine formation.

PROCESS	WHERE OCCURING	MAIN FORCE RESPONSIBLE	RESULT
Filtration	A 1 _____	B Blood pressure, opposed by: 1. _____ 2. _____ 3 _____	C Formation of fluid _____ tonic to plasma but with _____ protein concentration.
Tubular Reabsorption Secretion	D 1. _____ 2 _____ 3 _____ 4. _____ 5 _____	E _____ transport _____ transport	F. _____ of physiologically important solutes 1. _____ of waste product 2. _____ of urine
Acidification	G 1. _____	H. _____ transport and exchange of _____ for acid.	I. 1. _____ of H^+ 2. _____ of base.
Countercurrent Multiplier Exchanger	J. 1. _____ 2. _____	K. Multuplier — _____ transport Exchanger — _____	L. Creates conditions for _____ tonic urine.
ADH mechanism	M. 1.	N. _____ permeability of _____ tubules to _____	O. Forms _____ tonic urine

SISTEM PENGLIHATAN

SISTEM PENGELIHATAN

Modul 1 The eye.
Modul 2 Retina : distribution of rods, cones, dark sensitivity and visual acuity.
Modul 3 Visual adaptation.
Modul 4 Inversion of visual field upon the retina.
Modul 5 Intra ocular lens (IOL)
Modul 6 Eye accommodation.
Modul 7 Visual acuity.
Modul 8 The blind spot.
Modul 9 Visual fields.
Modul 10 Perimetry.
Modul 11 Projection of the different parts of the retina on the cerebral cortex.
Modul 12 Visual pathways as seen from the base of the brain.
Modul 13 Optical defects and refractive anomalies.
Modul 14 Colour vision.
Modul 15 Colour blindness.
Modul 16 Response of the three photopigments to light of different wavelengths.
Modul 17 Accommodation.
Modul 18 Pathway for accommodation reflex.
Modul 19 Movements of the eye.
Modul 20 Diagram of the course of the anatomic fibres to the eye.
Modul 21 The cell of retina.
Modul 22 General character of human aqueous homur(Expressed Relative to Plasma)
Modul 23 The aqueous humor.
Modul 24 Light reflex.
Modul 25 Diagrammatic representation of the cells in the visual pathways.
Modul 26 Structurs of photoreceptors.
Modul 27 The cell of retina.
Modul 28 Electrical activity of retina.
Modul 29 Recoding of the activity of a single ganglion cell.
Modul 30 Responses of a concentric cell in the retina to a monochromatic stimulus.
Modul 31 Light stimulus.
Modul 32 Receptivefield.
Modul 33 Retina, Control of eye movements, Fondues oculi & Iris, lens

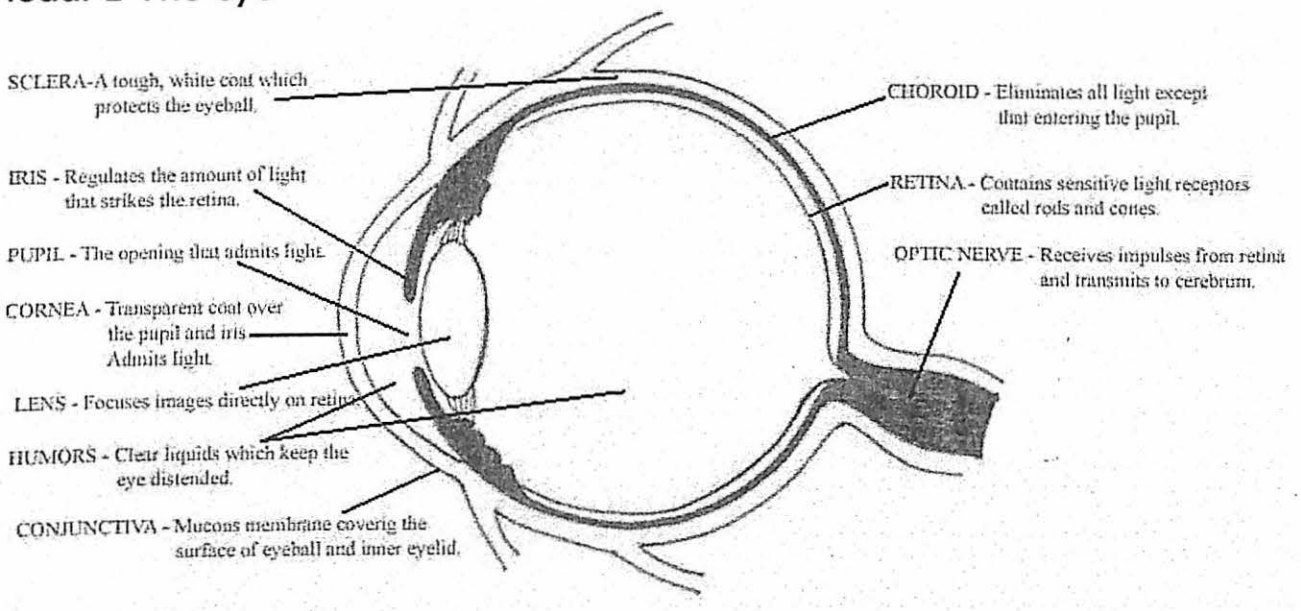
and ciliary body.

Modul 34	Organization of the visual field.
Modul 35	Highly schematic diagram of the visual projections to the cortex.
Modul 36	Projection of the retinas upon the lateral geniculate nucleus.
Modul 37	The intraocular fluid.
Modul 38	Intraocular pressure.
Modul 39	Optical defects and refractiue amomalies.
Modul 40	Eye and the retina.
Modul 41	Physiology of the central visual pathway receptive fields.
Modul 42	Visual field projects upon the retina normal.
Modul 43	The responses of retinal ganglion cells to colored light.

Menu Utama

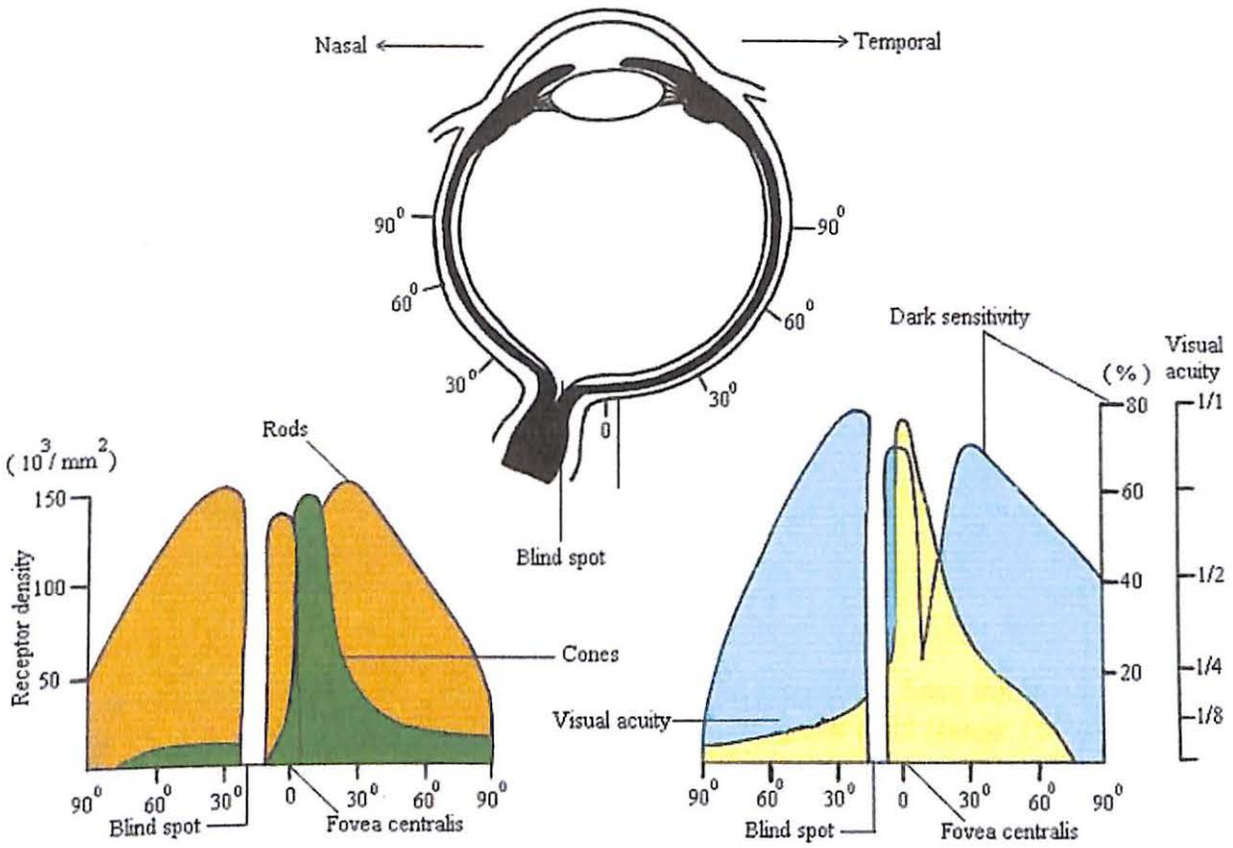
SISTEM PENGELIHATAN

Modul 1 The eye



SISTEM PENGELIHATAN

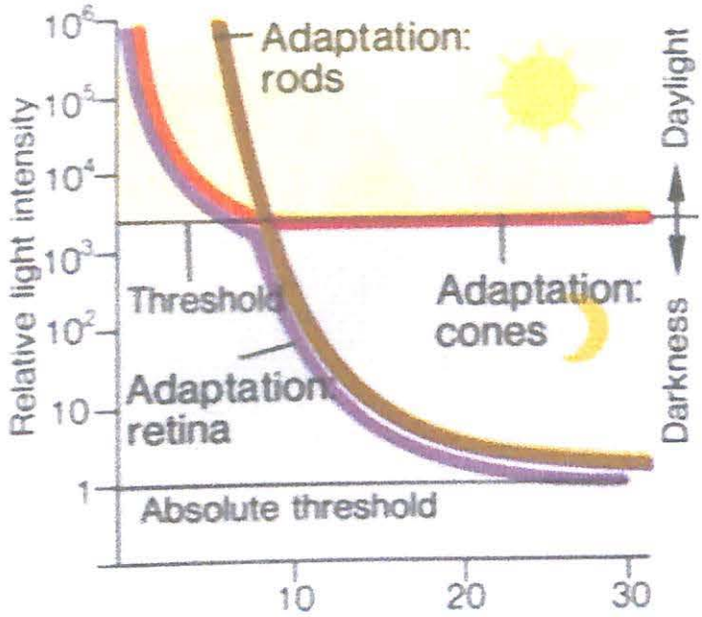
Modul 2 Retina : distribution of rods, cones, dark sensitivity and visual acuity



The retinal receptors are the light-sensitive rods and cones. The are not uniformly distributed in the retina: the fovea contains exclusively cones; the density of cones declines rapidly as the distance from the fovea increases. The rods are greatest in number 200 from the foveal centre. The fovea is capable of the greatest visual resolution because the signals of its cones have the lowest convergence towards the visual centers of the cortex. The fovea is therefore the visual center of the eye. The gaze adjusts to the foveal axis for the most precise visual inspection. Visual acuity declines as the image becomes distant from the fovea (right) because the convergence of the signals of the peripheral receptors is higher. At the optic disc, which subtends an angle of 3° at a distance of 15° from the fovea, there is no visual capacity at all; this is the blind spot because it contains no receptors. After dark adaptation, the distribution of visual acuity changes and corresponds to the distribution and convergence of the rods. Thus, cones are specialized for discrimination of color in bright light (photopic vision) and rods for discrimination of form (black and white) in poor light (scotopic vision). Visual acuity is sacrificed for dark adaptation.

SISTEM PENGELIHATAN

Modul 3 Visual adaptation



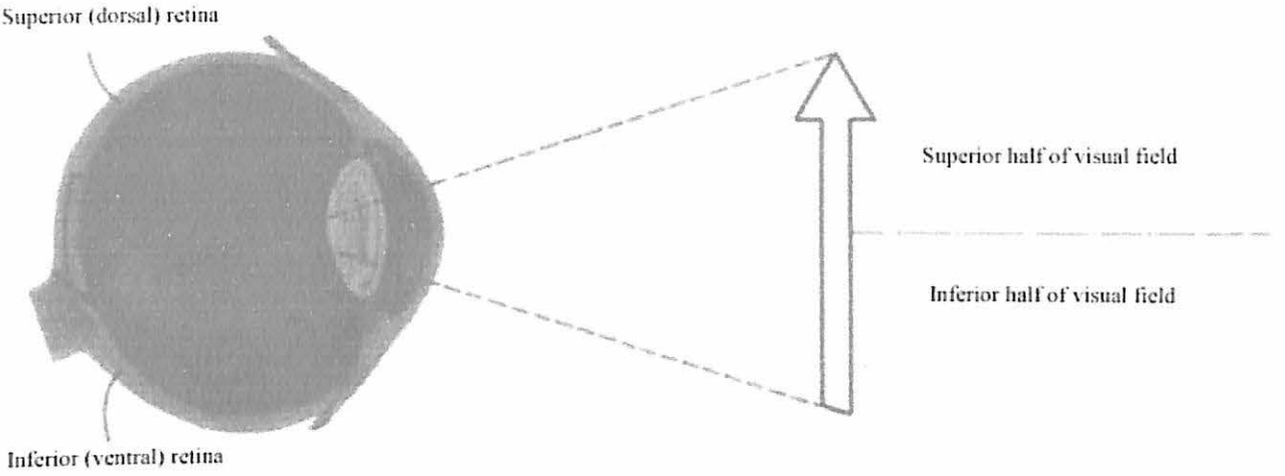
Adaptior to light

The eye is capable of responding to a wide range of light intensities from the low intensity of a distant star to the glare of the summer sea shore or a sunlit snow field (range 100 to 1011) response to such a broad range requires special mechanisms for adaption. For example, in going from daylight into a darkened theater, the eye is unable to perceive the low intensity light signals because they are temporarily below the retinal threshold. Important changes must occur before it is possible for the crude low luminance (scotopic) vision to operate optimally; maximum adaptation may require up to 30 min. The minimum light that can be detected after dark adaptation is the absolute visual threshold. For the first few minutes of adaptation, the threshold declines rapidly (1o min) and then more slowly to the absolute threshold (20 to 30 min). At c. 2000 times the assolute threshold, the bend in the curve represents transition from cone receptors to rod receptors purple curve. The transition level is the threshold for daylight (cone) vision. In night blindness or nyctalopia, the transition does not occur, and it is possible to record the adaptation curve for cones alone. Red curve)

Dark adaptation is a lowering of the visual threshold. Full adaptation requires c. 20 to 30 min and depends to a large extent on the regeneration of visual pigment (rhodopsin) in the receptor cells. Since vitamin A is involved in the synthesis of visual pigments, vitamin A deficiency is associated with various visual abnormalities. Chief among these is nyctalopia or night blindness in this state, there is not only a deficiency of visual pigments but also anatomic changes in the rods and cones and progressive degeneration of the neural layers of the retina.

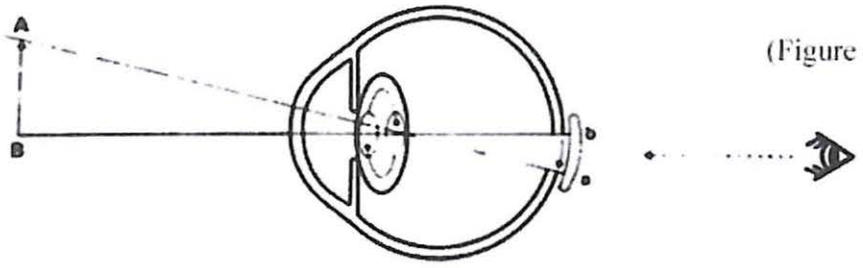
SISTEM PENGELIHATAN

Modul 4 Inversion of visual field upon the retina



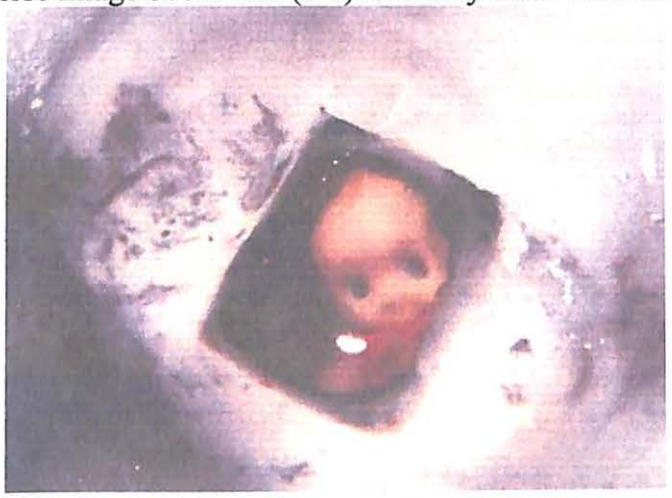
SISTEM PENGELIHATAN

Modul 5 Intra ocular lens (IOL)



(Figure 1)

After the Spring IOL is placed in the bag, the posterior wall of the eye is removed and the bared surface of the vitreous is covered with a plain soft contact lens. A doll (A-B) is placed in front of the eye. From behind, a reverse image of the doll (b-a) is clearly observable in the window.

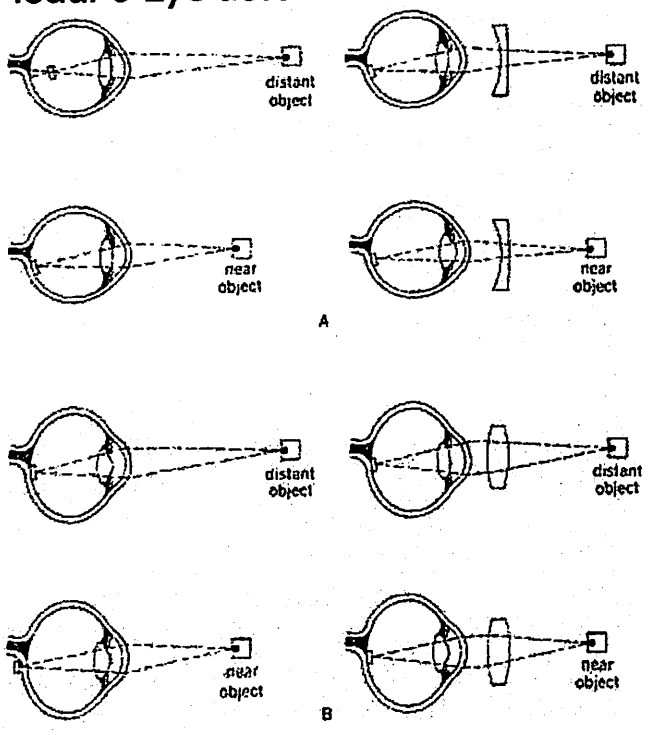


(Figure 2)

Actual image of (b-a) in Figure 1. A reverse image of the doll which is placed in front of the eye is clearly observed in the window.

SISTEM PENGELIHATAN

Modul 6 Eye accommodation



A-In the nearsighted eye, light ray from a distance source are focused in front of the retina. A concave lens placed before the eye bends the light rays out sufficiently to move the focused image back onto the retina. When near objects are viewed through concave lenses, the eye accommodates to focus the image on the retina.

B-The far sight eye must accommodates to focus the image of distant objects upon theretina. (The normal eye views distant objects with a flat , stretched lens.)
The accommodating power of the lens of the eye are sufficient for distant objects, and these objects are seen clearly. The lens cannot accommodate enough to keep image of near object focused on the retina, and they are blurred. A convex lens converge light rays before they enter the eye and allows the eye' s lens to work in a normal manner.

SISTEM PENGELIHATAN
Modul 7 Visual acuity

Tests for distant vision

Visual acuity is measured with Snellen's test types, a series of letters of varying sizes constructed so that the top letter is visible to the normal eye at 60 meters, and the subsequent lines at 36, 24, 18, 12, 9, 6 and 5 meters respectively. Visual acuity (V) is recorded according the formula V = d/D, where d is the distance at which the letters are read, and D the distance at which they should be read.

Visual acuities of less than 1/60 are recorded as counting fingers (CF), hand movements (HM), perception of light (PL) or no perception of light (no PL).

Tests for near vision

Visual acuity at the ordinary reading distance is assessed by using reading test types of varying sizes, the notation being based on the printers' point system. The smallest print is N5. The near vision is recorded as the smallest type which the patient can read comfortably.

Recording of visual acuity

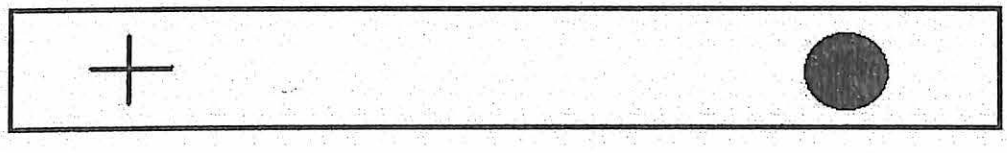
-Place Snellen's chart so that the letters are well illuminated.
-Stand at a distance of 6 meters from the test type (d = 6).
-Close one eye and read down the chart as far as you can and
-note the distance (D) written along
-Record Visual Acuity V = d/D

SISTEM PENGELIHATAN

Modul 8 The blind spot

If a small object is moved within the visual field until its image falls upon the optic nerve head, where there are no photoreceptors, it is no longer visible. Thus the normal visual field of each eye has a 'blind spot' or scotoma about'5 or 6~ in diameter situated some 15° lateral to the fixation point. We are usually quite unaware of the existence of this blind area as we may also be unaware of other scotomata resulting, for example, from local retinal damage; because image of an object falls on the retina in the other eye.

Determine your blind spot



Close the left eye with left hand. Hold the card with cross and black dot in your right hand so that black dot lies in the temporal field of vision. Keep the card at arms length and fix the vision on the cross. Gradua11y bring the card nearer to; eye so that black dot disappears. Keep on bringing the card nearer till ; it reappears. This gives indirectly the horizontal extent of the blind spot.

SISTEM PENGELIHATAN

Modul 9 Visual fields

When we look at an object, we not only see that object but also a number of other objects in the neighbourhood, more or less distinctly. The full extent of this vision is called the visual field. The field of vision is limited both by the area of the retina and by the margins of the orbit, nose and cheek. Hence the position of the eye is important. The extent of the visual field varies with the stimulus used. For example, the field is larger to large than to small objects, or to brightly illuminated than to dimly lit objects. The full extent of the visual field cannot be tested to coloured objects since colour sensitivity is mainly confined to the central or macular field, corresponding to the central part of the retina rich in cone cells. The rod cells, sensitive to white light, are more uniformly distributed across the whole area of the retina. Moving stimuli, on the other hand, are best perceived in the peripheral field since specialized movement-sensitive receptor cells are located mainly in the peripheral parts of the retina. This part of the retina • is also particularly responsive to the sudden appearance or disappearance of stationary visual stimuli, but the appreciation of the form of an object only poorly developed in this part of the field. Form and detail are best perceived in the central field and this aspect of visual function is tested by assessment of the visual acuity.

Mapping of field of vision

Confrontation tests.

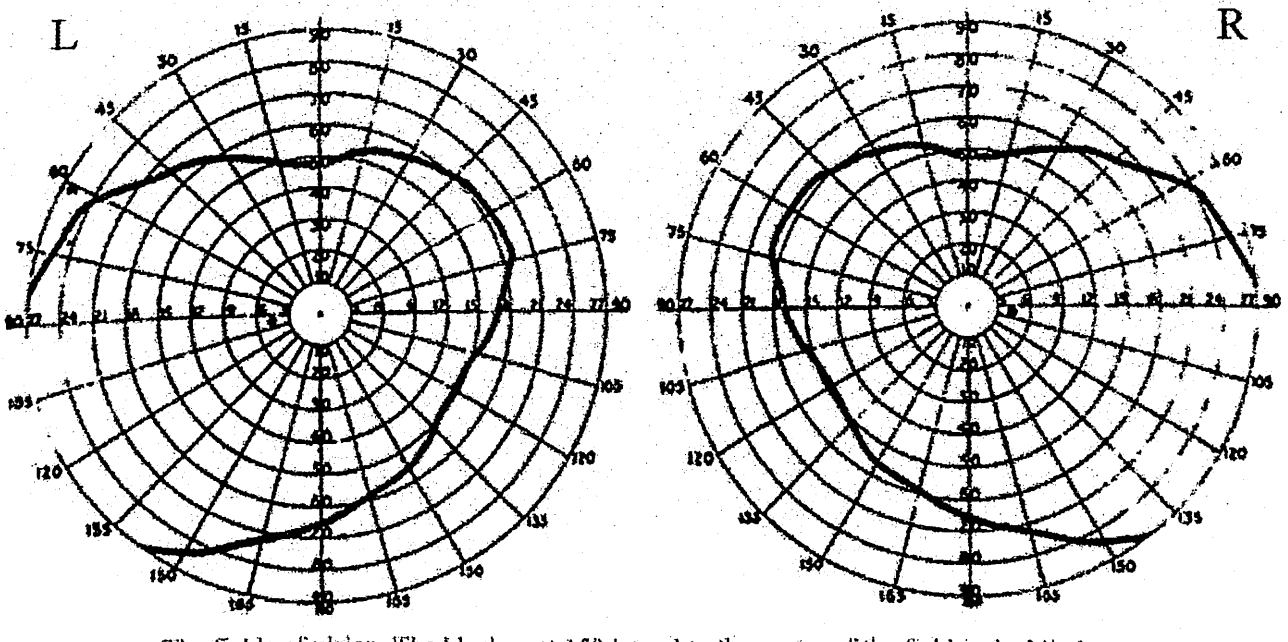
Seat yourself opposite the patient, at a distance of about 1 meter from him. If his right eye is to be tested, ask him to cover his left eye with his hand and look steadily at your left eye Cover your right eye with your right hand and look steadily at your left eye. Cover your right eye with your right hand and gaze steadily at the patient's right eye. In this way slight movements of the patient's eye, which would introduce error into the test, can be detected. Hold up your left hand in a plane midway between the patient's face and your own, at first at almost a full arm's length to the side. Keep moving the fingers of the hand and bring it nearer until you yourself can just perceive the movements of the fingers 'with the tail of your eye'. Ask the patient whether he sees the movements, telling • him meanwhile to be sure not to take his own eye off yours. If he fails to see the fingers, keep bringing the hand nearer until he does see them. Test the field in this fashion, in every direction - upwards, downwards, to right and to left using the extent of your own/ field for the purpose of comparison.

SISTEM PENGELIHATAN

Modul 10 Perimetry

A more accurate examination can be made by the perimeter. This consists of a semi-circular metal arc, rotatable about its centre, so as to describe a hemi sphere. A black cross marks this point on the inner side of the arc. This cross is used as a points of fixation. At the centre of curvature of the arc, is an adjustable chin rest on a pillar. The subject places his chin on this and its height is adjusted until the eye to be examined is on the same level as the fixation point. Behind the mounting stand is a large metal disc. Place the perimeter chart on the disc, making sure that it is correctly oriented. Hold it in position with the metal frame.

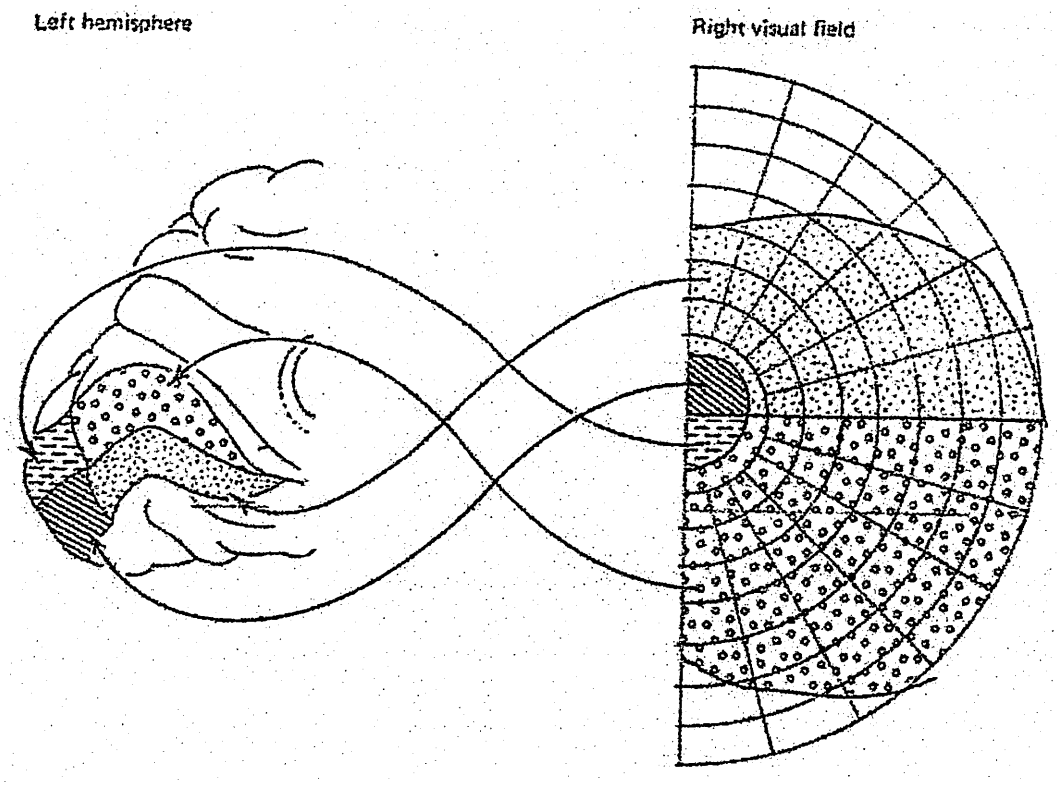
The object to be viewed is an illuminated spot. This can be directed at any point on the arc by rotating a knurled knob. Movements of this knob and rotations of the metal arc are communicated to a small metal spike behind the mounting stand. The subjects sits with his back to the light, places his chin on the chin rest and looks at the fixation point with the eye under examination and closes the other eye. The illuminated spot is directed at the outer end of the arc and brought inwards until it can just be seen by the subject who is gazing steadily at the fixation point. This is the edge of the subject's visual field. Record the position by bringing the disc forward so that the perimeter chart is pierced by the metal spike. Now rotate the arc through 15° (when it should click into position) and repeat the process. Continue altering the inclination of the arc by 15° step until the entire visual field has been plotted. Join up the dots on the chart to get the boundary of the field of vision and compare it with the field already printed on it.



The fields of vision. The black spot 15° lateral to the centre of the field is the blind spot.

SISTEM PENGELIHATAN

Modul 11 Projection of the different parts of the retina on the cerebral cortex

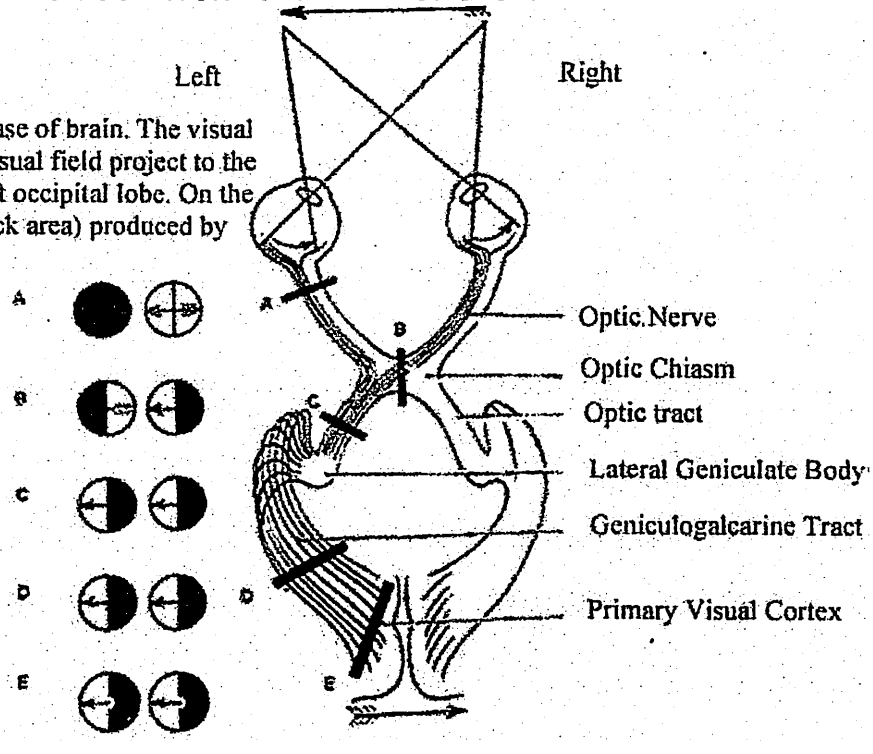


SISTEM PENGELIHATAN

Modul 12 Visual pathways as seen from the base of the brain.

Visual pathways as seen from the base of brain. The visual impulse from the right half of the visual field project to the left half of each retina and to the left occipital lobe. On the left are the visual field defects (black area) produced by lesions affecting :

A-Optic nerve
B-Optic chiasm
C-Optic tract
D-Optic radiation
E-Occipital cortex.



SISTEM PENGELIHATAN

Modul 13 Optical defects and refractive anomalies.

The lens system in a modern camera can produce a considerably better image than the dioptric apparatus of the eye. The physicist and physiologist Hermann V. Helmholtz (1821-1894) once wrote that if he should receive an optical instrument so carelessly constructed as the eye, he would send it back to the maker. The "physiological" deficiencies in focussing by the eye discussed here, however, are largely compensated by neuronal contrast mechanisms.

Astigmatism.

The corneal surface is not rotationally symmetric about the optical axis, for the vertical curvature is usually somewhat greater than the horizontal curvature. This discrepancy results in an angle-dependent difference in refractive power (astigmatism or astigmia). If the difference is no greater than 0.5 D, the condition is called "physiological" astigmatism.

D-diopter : unit of refractive power.

Spherical aberration.

The cornea and the lens of the eye. Like simple lenses, have shorter focall lengths in the peripheral regions than in the central part around the optical axis. The resulting spherical aberration causes blurring of the image. The smaller the pupils the more the peripheral rays are excluded and the less distortion is caused by spherical aberration.

Chromatic aberration and accommodation.

As do all simple lense, the dioptric apparatus refracts short-wavelength light more strongly than long-wavelength light (chromatic aberration). Therefore greater accommodation is required for sharp focussing of the red parts of an object than for the blue parts. It is because of this difference that blue objects appear to be further away than red ones at the same objective distance. The builders of gothic churches often exploited this physiological illusion in their stained-glass windows, by making the background blue and the fig ures other colors, so that one sees an apparent spatial separation between figures and background.

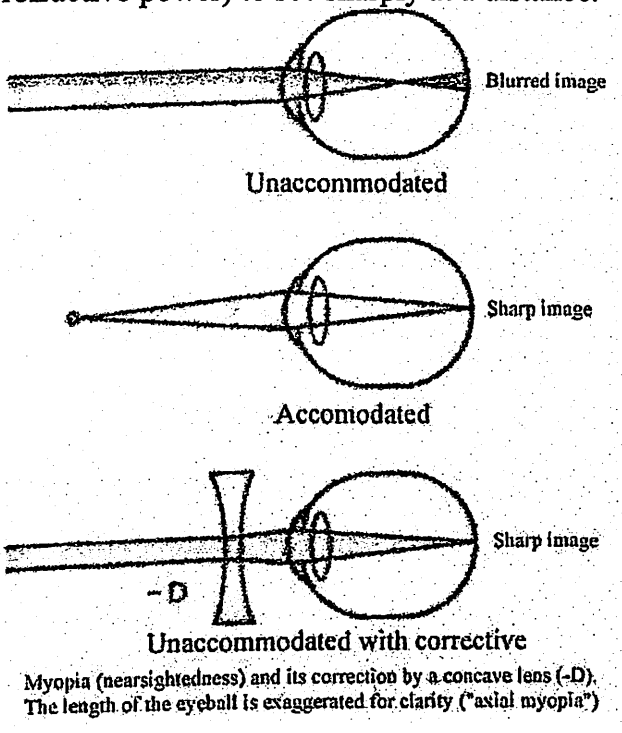
Stray light and clouding of the dioptric apparatus.

The lens and the vitreous body contain structural proteins and macromolecular substances in colloidal solution. Therefore a slight diffuse dispersion of the light occurs in the dioptric apparatus. But this stray light impairs visual perception only with very bright stimuli. Even in a healthy eye there are cloudy areas in the vitreous body. Visible against a white wall as small disks or irregularly shaped small gray spots. When the eye moves they seem to flit like gnats across the light background. In older people the water content of the lens can decline so greatly that the structure of the remaining material becomes condensed. Making the lens opaque (senile cataract). Removal of the lens enables these patients to see normally when they are fitted with spectacles having a strong convex lens (ca. + 13 d for long-distance vision).

Myopia.

The total refractive power of the dioptric apparatus of a normal, unaccommodated eye is 58.6 diopter. With this refractive power an infinitely distant object is focussed sharply on the retina when the distance between the pole of the cornea and the fovea is 24.4 mm. If the axial length of the eyeball is greater distant objects cannot be seen sharply because the plane of focus is in ront of the

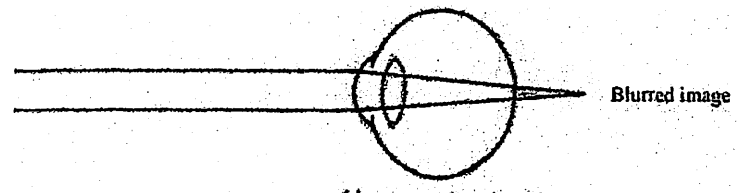
fovea (near-sightedness, myopia). A nearsighted person must wear with concave lenses (negative refractive power) to see sharply at a distance.



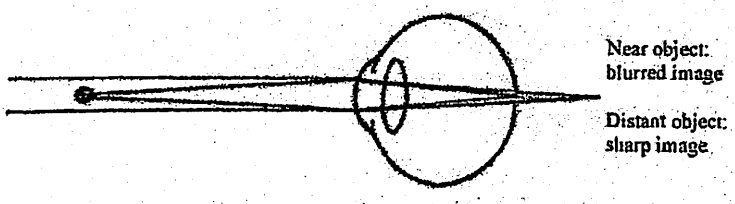
Myopia (nearsightedness) and its correction by a concave lens (-D). The length of the eyeball is exaggerated for clarity ("axial myopia")

Hypermetropia.

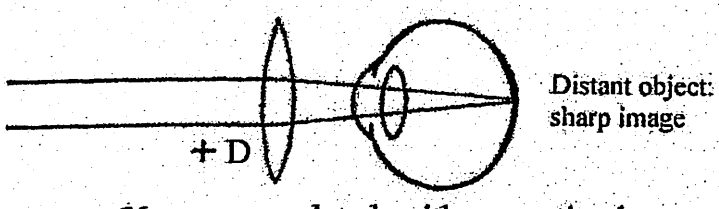
If the axial length is too short for the refractive power of the dioptric apparatus. A condition of "farsightedness" (hyperopia or hypermetropia) exists. The hypermetropic person can see distant objects clearly by nearaccommadating to some extent, but his accommodation range is not sufficient to allow sharp focussing of nearby objects. Convex lenses (positive refractive power) are required to compensate this defect.



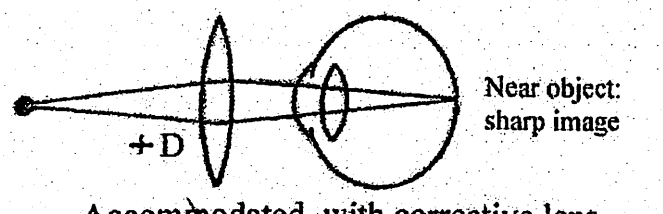
Unaccommodated



Accommodated



Unaccommodated, with corrective lens



Accommodated, with corrective lens

Hypermetropia (farsightedness) and its correction by a convex lens (+D)

through one eye and then the other, he will see that the apparent colours of things around him are very different according to which eye he uses. Again, the colour of a small object may appear quite markedly different if viewed against different, large, strongly coloured backgrounds. These effects do not disturb colour matches however much they change the subjective appearance.

Objects which seem to have the same colour when lit by one source of light may well not match when lit by a light of different spectral composition. The spectral composition of the light reflected from an object depends, of course, upon both the illumination and the spectral variation in reflectivity of the object.

Anomalous trichromats, comprising nearly 6 per cent of the male population, resemble normal subjects in that they require three primaries to match all colours by colour mixture, but they require them in abnormal proportions. The cause of anomalous trichromacy is unknown: anomalous trichromats may have abnormal pigments.

Colour-blind subjects, even when their colour-discrimination is very poor, are often unaware of their defect. In familiar situations they compensate for their defective colour-discrimination by an increased use of alternative clues based upon prior knowledge of the usual colours of objects that they recognise by their shape. It may only be on rare occasions, when these clues are absent, that the defect becomes apparent.

SISTEM PENGELIHATAN
Modul 14 Colour vision

Photopigments in cones

When a subject views a scene illuminated by white light of low intensity his visual acuity is low and he is unaware of any sensation of colour. However, when the illumination level is sufficiently raised objects can be seen in much greater detail and in their natural colours.

Vision at high luminance levels (phototopic vision) appears to be mediated by retinal cones (high acuity being achieved by using the densely packed mosaic of cones in the Lovea) while scotopic vision is mediated by peripherally sited rods,

Measurements of the absorption spectra of the outer segments of cones from primate and human retinae show that cones do not al contain the same pigment. The measured absorption spectra fall, into three separate groups probably corresponding to the presence of three different photo. pigments. None of the cones has an absorption spectrum like that of the rod pigment rhodopsin (with maximum absorption at about 500 nm).

The absorption spectra of two photopigments present in the human foveal cones have been measured in the living eye by the opthalmoscopic technique. the two foveal cone pigments detected in this way by Rushton have been called 'erythrolabe' (the more red-sensitive pigment) and 'chiorolabe' (the more green-sensitive one). The absorption spectra of these two pigments correspond to the two commoner types found in isolated cones cones containing the blue-sensitive 'cyanolabe' seem to be re relatively scarce and probably completely absent from the central fovea, The chemical nature of these pigments is unknown but it is probable that, like rhodopsin, the cone pigments are conjugated proteins.

Ultraviolet (UV) radiat1.on is normally invisible not because the retinal photopigments are insensitive to UV but because the lens contains a yellow pigment that prevents the UV from reaching the retina. People who nave had their lenses removed (because they have become opaque) can see UV quite well. Normal people may be aware of ultraviolet radiation as a vague haze because the lens pigment flouresces strongly

Basis of tcichromatic vision

It is now accepted that the trichromatic nature of normal colour vision is determined by the existence in the retina of three kinds of photo sensitive pigment molecules segregated in three different groups of cones. If there are just three different pigment, and if the nature of the effect on pigment molecule of absorbing a quantum of light is independent of its wavelength, which it is, then the trichromacy of normal colour vision is an inevitable consequence. Since the degrees of excitation of the three kinds of cone are the only independent variables, the sensation produced by a visual stimulus must be capable of description in terms of three quantities.

This is not to say that the perceived colour of a visual stimulus is entirely determined by its spectral composition. It is well known that the appearance of a coloured object is dependent upon the visual environment in which it is seen and the recent visual experience of the subject. If a subject places a coloured filter in front of one eye for a few minutes and then, after removing it, looks around first

SISTEM PENGELIHATAN
Modul 15 Colour blindness

Abnormalities of colour vision known as colour—blindness are common in men (about 8 per cent) but much less common in women (0.4 per cent). The common abnormalities are inherited as X.-linked recessive characters.

Colour-blind subjects fall into a number of fairly sharply defined categories. Monochromat are quite unable to distinguish colours at all: a monochromat can match any two lights simply by adjusting their intensities. Monochromat are of two kinds. Rod monochromats have a luminosity curve like that of normal dark-adapted subjects; they see very poorly in bright surroundings
are presumed to lack functional. cone mechanisms. Cone monochromats, on the other hand, appear to lack the rod mechanism: although their vision is more or less normal in bright surroundings, they see very badly when the illumination is reduced to scotopic levels. Monochromatism is rare, cone monochromatism exceptionally so.

Dichromats, who can match all colours with suitable mixtures of two primaries, are of three kinds. Protanopes and deuteranopes (each about 1 per cent of males) are often grouped together with the anomalous trichromats as 'red-green' blind. They have very little ability to discriminate colours at the red end of the spectrum, and thus confuse red, brown and green objects, though they can usually distinguish yellow objects by their higher reflecting power. Protanopes and deuteranopes differ from each other in the form of their photopic luminosity curves. Protanopes are relatively insensitive to red light and appear to lack the more red-sensitive pigment erythrolabe. Deuteranopes have luminosity curves similar to normal subjects and appear to have none of the green-sensitive pigment. The third kind of dichromatic vision is tritanopia. Tritanopes, who are as of female as male, are rare. They have normal colour discrimination at the red end of the spectrum, but they have little ability to distinguish blue from green. Tritanopes, sometimes called 'blue-blind', appear to lack the blue-sensitive pigment cyanolable.

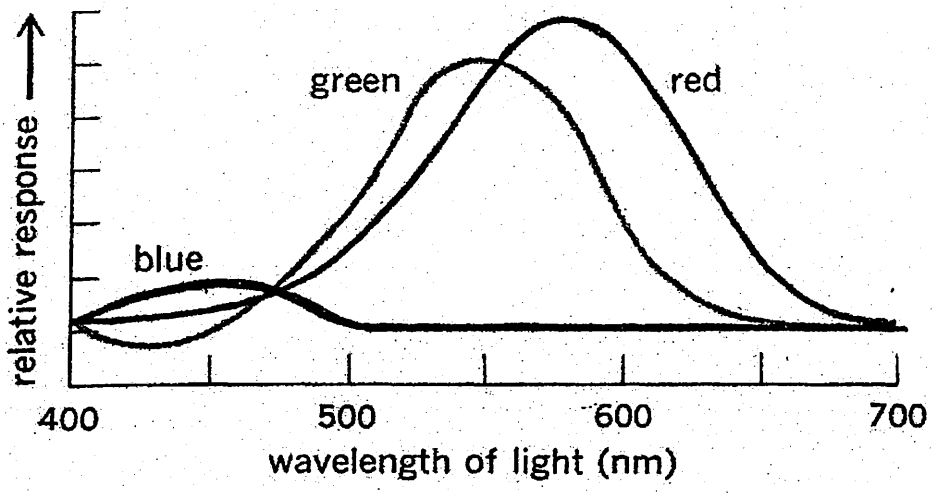
Test for colour blindness

The detection of colour-blindness is important in selecting people for jobs in which it is necessary to be able to distinguish coloured markings or coloured light signals. A convenient quick test consists of a set of 'pseudo-isochromatic plates', of which Ishihara's are probably most widely used. Each plate has an array of multi-coloured dots so that a letter or figure is formed by dots of one colour, other colours forming the back-ground. Some plates are designed to be read easily by the normal, but not by the colour-blind subject, which others can be read only by the colour-blind subject; some plates are interpreted differently by normal and colour-blind. This test is very efficient at separating the normal from the abnormal but it does not distinguish well between different types of abnormality. To decide whether a subject with a mild abnormality can safely be employed in a particular occupation, a special test designed to imitate the task that has to be performed is often used.

Though hereditary colour-blindness is very much commoner, defects of colour vision can also be acquired as a result of diabetes mellitus or disease of the retina, optic nerve or visual cortex. These acquired defects are usually accompanied by severe defects of visual acuity or of visual fields.

SISTEM PENGELIHATAN

Modul 16 Response of the three photopigments to light of different wavelengths.



SISTEM PENGELIHATAN
Modul 17 Accommodation

Consists of three associated events.
- convergence of optical axis.
- constriction of pupils.
- accommodation of the lens.

Occurs when gaze is transferred from a near object.
Images on the two retinae are blurred and disparate.
They do not fall on corresponding points.

Axes converge- images fall on corresponding ciliary muscle contracts:
- anterior surface of' lens bulges
- optical power increases
- objects brought into focus

Near point
Nearest point to eye an object may be viewed without blurring.
- 10 years old 7.0 cm
- 40 years old 20.0 cm
- Established presbyopia 40 cm.

Visual reflexes
Light reflex:
Bright light on retina
- pupillary constriction
- on same side - direct
- on opposite side - consensuai.

Reflex pathway:
- Photoreceptor
- Optic nerve

Relay in pre-tectal regions

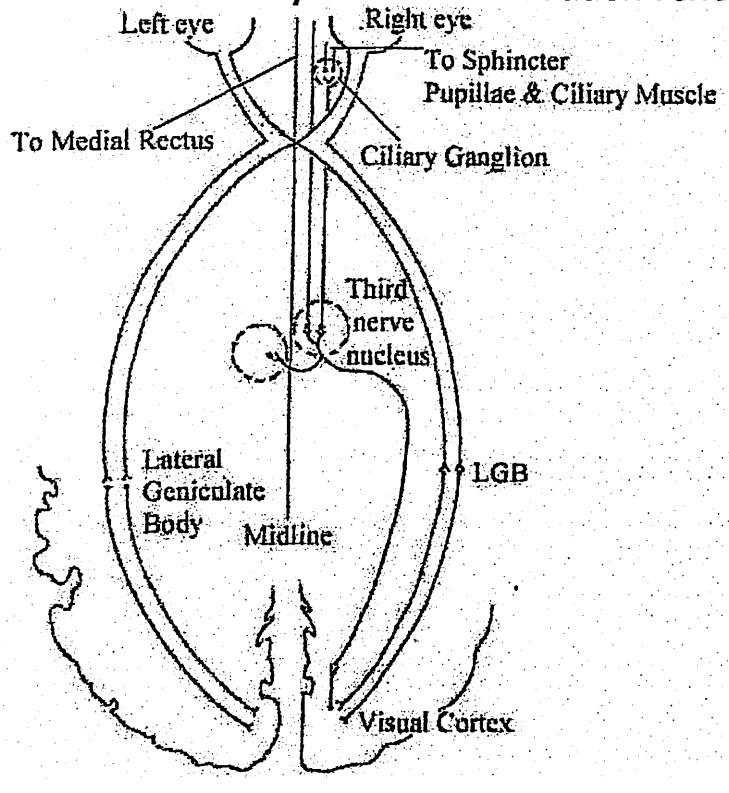
Fibers pass to edinger - westphal nucleus of each side.

Relay
- pre-ganglioruc fibers
- ciliary ganglion
- ciliarry nerve (post - ganglionic, parasympathetic)
- constrictor pupillae

Light reflex intact after visual cortex ablation abolished after lesions in pre-tectal region.
Constriction of accommodation unaffected by pre-tectal damage.

SISTEM PENGELIHATAN

Modul 18 Pathway for accommodation reflex.



SISTEM PENGELIHATAN

Modul 19 Movements of the eye

Movements of the eye:

Eye is moved by extra - ocular muscles.

Movement	Muscle	Innervation
Rotation of eye around vertical axes	Medial rectus Lateral rectus	III nerve VI nerve
Rotation of eye around oblique axes	Superior oblique Inferior oblique Superior rectus Inferior rectus	IV nerve III nerve

Muscle	Direction of eye movement
Lateral rectus	Abduction
Medial rectus	Adduction
Superior rectus	Elevation, plus adduction and rotation of upper part ofpupil towards nose
Inferior rectus	Depression, plus adduction and rotation of upper parts of pupil away from nose
Superior oblique	Depression; plus abduction and movement of upper part of pupil towards nose
Inferior oblique	Elevation, plus abduction and rotation of upper margin of pupil away from nose.

Control through midbrain nuclei (III, IV & VI nuclei)
Eyes move together
-conjugate movement
-convergence

Objective to produce retinal images on corresponding points.
Failure :

→ diplopia (double vision)

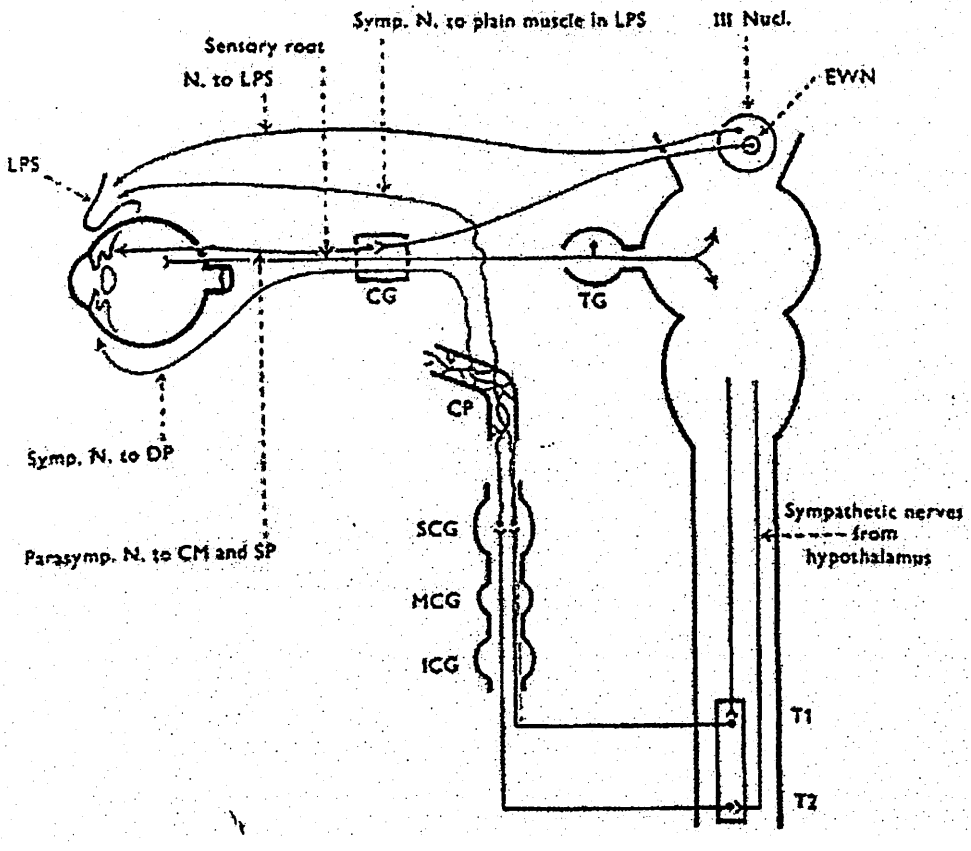
squint → double vision → supression of one image

The reflex pathways for constriction and dilation of the pupil

	Constriction
Stimulus	Bright light impinging on the retina
Receptors	Retinal nerve cells
Afferent neurons	Fibers in the optic nerves (second cranial) and optic tracts

SISTEM PENGELIHATAN

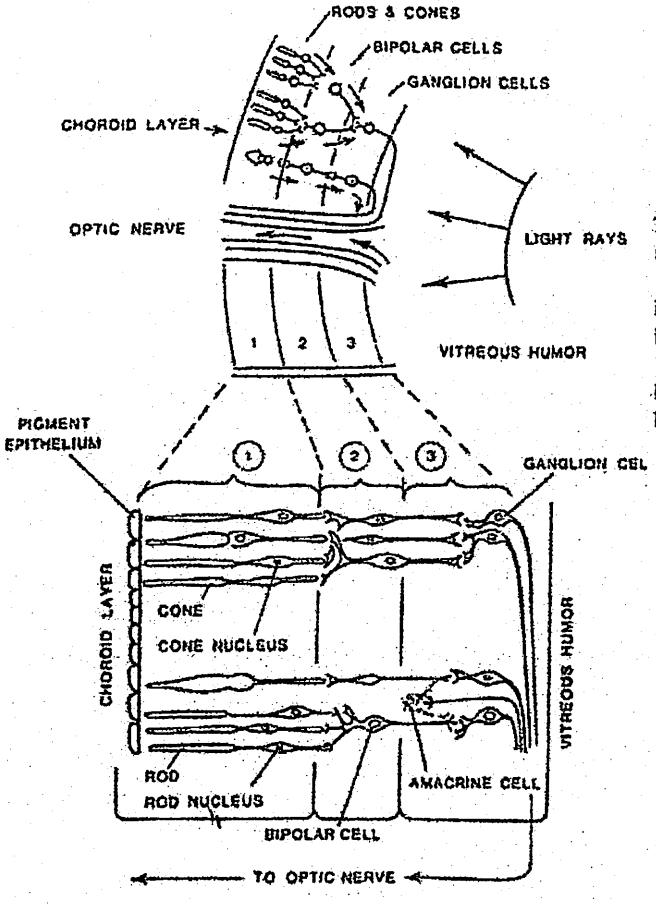
Modul 20 Diagram of the course of the anatomic fibres to the eye.



III Nucl.= oculomotor nucleus.
EWN= Edinger-Westphal Nucleus.
LPS= Levator Paipebrea Superioris.
CG= Ciliary Ganglion.
T1, T2= first and second thoracic segments of spinal cord.
SCG, MCG and ICG = Superio, Middle and Inferior Cervical sympathetic Ganglia.
TG = Trigeminal Ganglion.
DP = Dialator Pupilae.
SP = Sphincter Pupil.
CM = Ciliaris Muscle.
CP = Plexus on internal Carotid Artery.

SISTEM PENGELIHATAN

Modul 21 The cell of retina.



The retina has three cell layers from outside to inside : rods and cones,

i) The receptors cells (1)
ii) The bipolar cells (2)
iii) The ganglion cells (3)

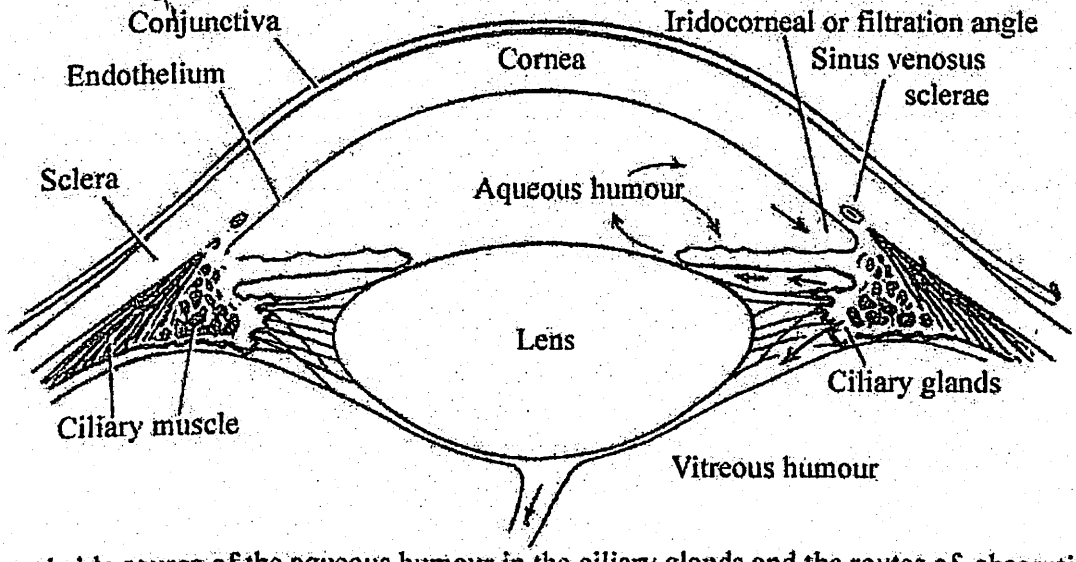
Note that the light rays have to pass through the inner layers to reach the receptor cells in the outer layer.

SISTEM PENGELIHATAN

Modul 22 General character of human aqueous homur

(Expressed Relative to Plasma)

1. Slightly hypertonic
2. Acidic
3. Marked excess of ascorbate (15 times more)
4. Marked deficit of protein (0.02 % in aqueous & 7 % in plasma).
5. Slight excess of:
   a. Chloride
   b. Lactic acid
6. Slight deficit of:
   a. Sodium (rabbit study)
   b. Bicarbonate
   c. Carbon dioxide
   d. Glucose
7. Other features
   a. Amino acids (variable concentrations)
   b. Sodium hyaluronate
   c. Coagulation properties
   d. Containing IgG



The probable source of the aqueous humour in the ciliary glands and the routes of absorption into the ciliary glands lie in the ciliary body, which is a ring-like structure. The suspensory ligaments of the lens.

SISTEM PENGELIHATAN
Modul 23 The aqueous humor

Functions
1.Regulation of Intra ocular pressure.
2.Maintains the shape of the eyeball and there by that the refracting surfaces.
3.Aqueous humor has important metabolic requirements in providing substrates and removing metabolites from the avascular cornea and lens. For example, the cornea takes glucose and oxygen from the aqueous and releases lactic acid and a small amount of carbon dioxide into the aqueous. The lens also uses glucose and generates lactate and pyruvate. In addition, it is reported that potassium and amino acide in the aqueous may be taken up by the lena, while sodium moves from the lens to the aqueous. The metabolism of the vitreous arid retina also appears to be associated with the aqueous humor in that substances such as amino acids and glucose pass into the vitreous from the aquenous.

Formation
The aqueous humour is formed continuously by the ciliary epithelium at a rate of 2 to 3 ml/min. It is derived from the plasma with in the capillary network of the ciliary processes. It is formed by the process of :
1.diffusion (lipid-soluble substances are transported through the lipid portions of the membrane proportional to a concentration gradient across the membrane).
2.ultrafiltration (water and water-soluble substances, limited by size and charge, flow through theoretical "micropores" in the protein of the cell membrane in response to an osmotic gradient or hydrostatic pressure)
3.secretion (water-soluble substances of larger size or greater charge are actively transported across the cell membrane.

The fate of the aqueous humour.
The aqueous humour formed in the posterior chamber passes forwards over the anterior surface of the lens, through the pupil enters the anterior chamber and then drains from the anterior chamber at iridocorneal angle in to the sinus venosus sclerae (SVS; Canal of Schlemm). The SVS is a thin walled vein that extends circumferentially round the sclera. It is so highly permeable that molecules as big as large proteins are able to penetrate its wall. Between the wall of the SVS and the anterior chamber are numbers of trabeculae or laminar plates, which are perforated with small holes. As the trabeculae lie in parallel, the holes become partly occluded as the plates come together, increasing the resistance of flow of the aqueous through them It is suggested that an increase in pressure of they aqueous distends the space between the plates, thereby lowering the resistance to drainage, a fall in pressure having the reverse effect. Such a mechanism could act as an autoregulatory process to keep the intraocular pressure constant.

The intra-ocular pressure
The mechanism described above provides resistance to flow and therefore creates a pressure within the eyeball that serves to maintain its shape. The pressure is constant throughout life at 16 mmHg (range 10 to 20 mmHg or 1.3 to 2.6 kPa).

Glaucoma
This is a disease of the eye in which the intraocular pressure is abnormally high. Any rise in pressure is transmitted through the vitreous humour and impedes the retinal circulation with a risk of atrophy

of the retina and consequent blindness.

Glaucoma is one of the commonest causes of blindness so its early diagnosis is important. A moderate increase in intraocular pressure maintained over a number of years will gradually induce blindness. An acute rise in pressure to about 60 to 70 mmHg causes considerable pair) and. may result in blindness in a few days. The major cause of glaucoma is 'an increased resistance to filtration and treatment is directed at relieving this. The secretion of aqueous may be temporarily reduced by drugs such as acetazolamide (Diamox), which is a carbonic anhydrase inhibitor and interferes with the production of bicarbonate and hence reduces the rate of .fluid formation"? The causes of increased resistance are not always known but increased pressure occurs after intra-ocular haemorrhage or infection. There are protective mechanisms for removing debris producec by such events. Large numbers of phagocyte are to be found on the trabecular surfaces and in the interstitial spaces near the SVS: these phagocytes ingest debris and reduce to smaller molecules, which pass easily into the SVS.

SISTEM PENGELIHATAN
Modul 24 Light reflex.

1. Examine each eye separately.
2. Make the subject sit in a slightly dark place, (this will dilate the pupils) which should be indirectly illuminated.
3. Ask him to look into the distance. (this will relax accommodation)
4. Shine a bright light into the eye to be tasted.
5. The pupil should contract immediately, then dilate again a little and after undergoing few oscillation, settle down
   to a smaller size.
6. Switch off the light. The pupil will dilate rapidly to its previous size.

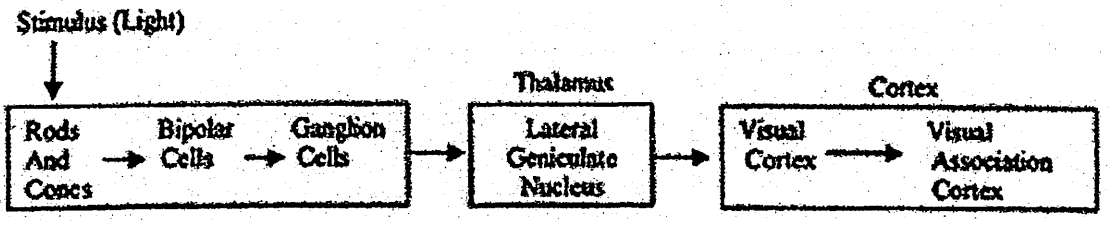
Consensual light reflex.
Put your hand between the two eyes of the subject. Shine bright light in one eye. Both pupils will constrict simultaneously.

Reaction to accomodation.
1. Pupillary constriction
2. Convergence
3. Increase in the curvature of the anterior surface of lens.

Accommodation reflex.
1. Make the subject sit in a shady place which is indirectly illuminated.
2. Hold up one finger close to subject's nose.
3. Ask him to look away at a distance.
4. Then ask him to look quickly at your finger.
5. The eyes will converge and pupils become smaller.

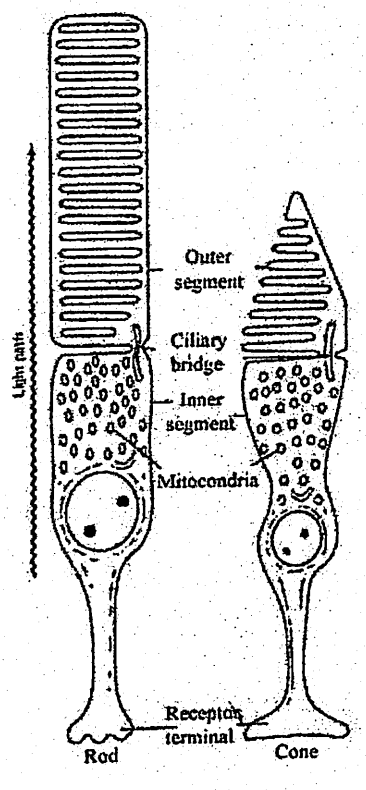
SISTEM PENGELIHATAN

Modul 25 Diagrammatic representation of the cells in the visual pathways.



SISTEM PENGELIHATAN

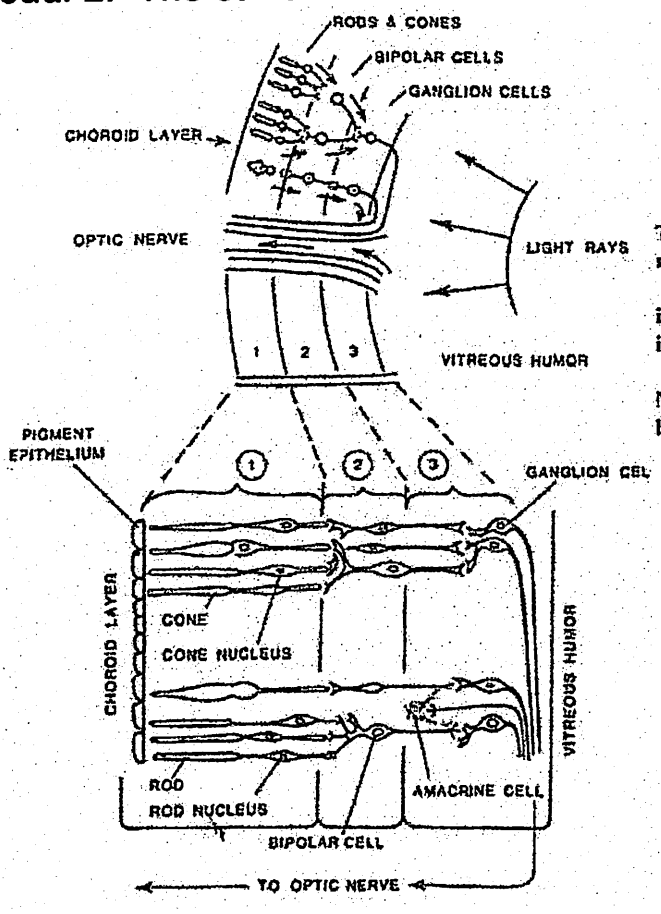
Modul 26 Structurs of photoreceptors



Structure of photoreceptors. Both rod and cone cells are differentiated into inner and outer segments connected by a ciliary bridge.
The inner segments of both cell types contain the nucleus and most of the biosynthetic machinery and are continuous with the receptors terminal, the membranous disc in the outer segments of rod cells (unlike those in cone cells) are nut
connected with the plasma membrane.

SISTEM PENGELIHATAN

Modul 27 The cell of retina.



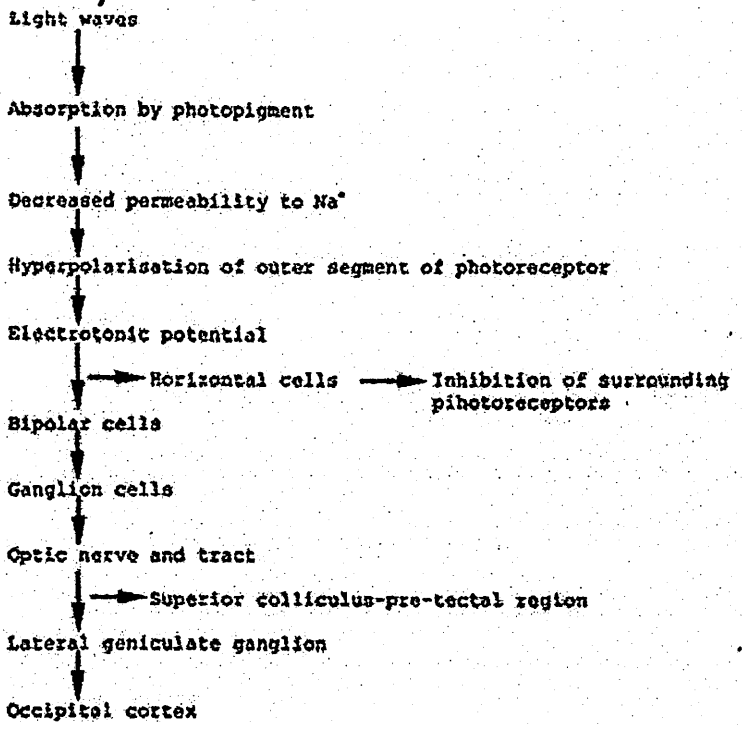
The retina has three cell layers from outside to inside : rods and cones,

i) The receptors cells (1)
ii) The bipolar cells (2)
iii) The ganglion cells (3)

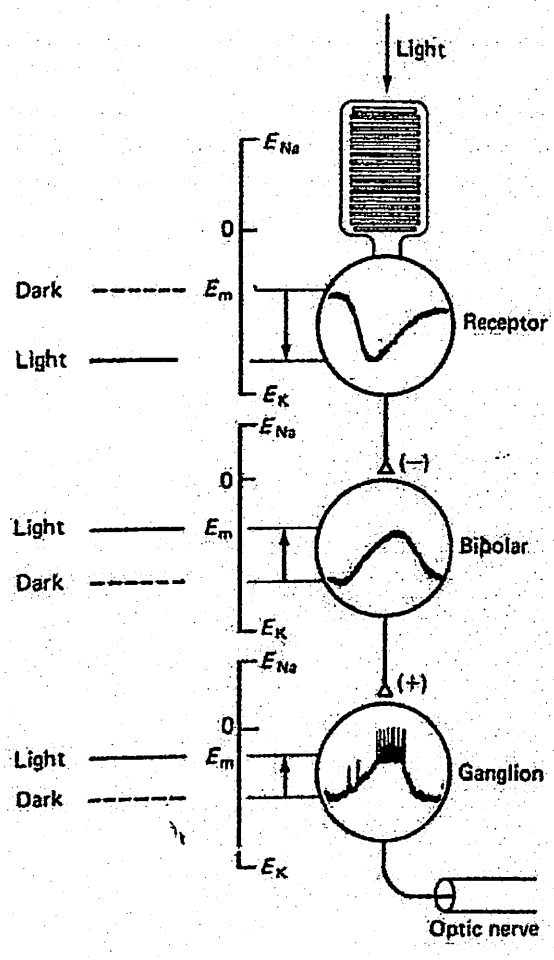
Note that the light rays have to pass through the inner layers to reach the receptor cells in the outer layer.

SISTEM PENGELIHATAN

Modul 28 Electrical activity of retina



Polarization of cells in the on-line retinal pathway involving a depolarizing bipolar cell.



In the dark a constant leakage of Na+ ions keeps the resting membrane potential of the photoreceptor relatively low .A flash of light (arrow) presented to the receptor results in decreased Na+ conductance and a net hyperpolarization. The light-induced receptor hyperpolarization decreases the release of inhibitory transmitter (-) at the receptor-bipolar cell synapse. This disinhibition of the bipolar cell produces an increase in the release of excitatory transmitter (+) at the bipolar-ganglion cell synapse se, resulting in excitation of the ganglion cell.

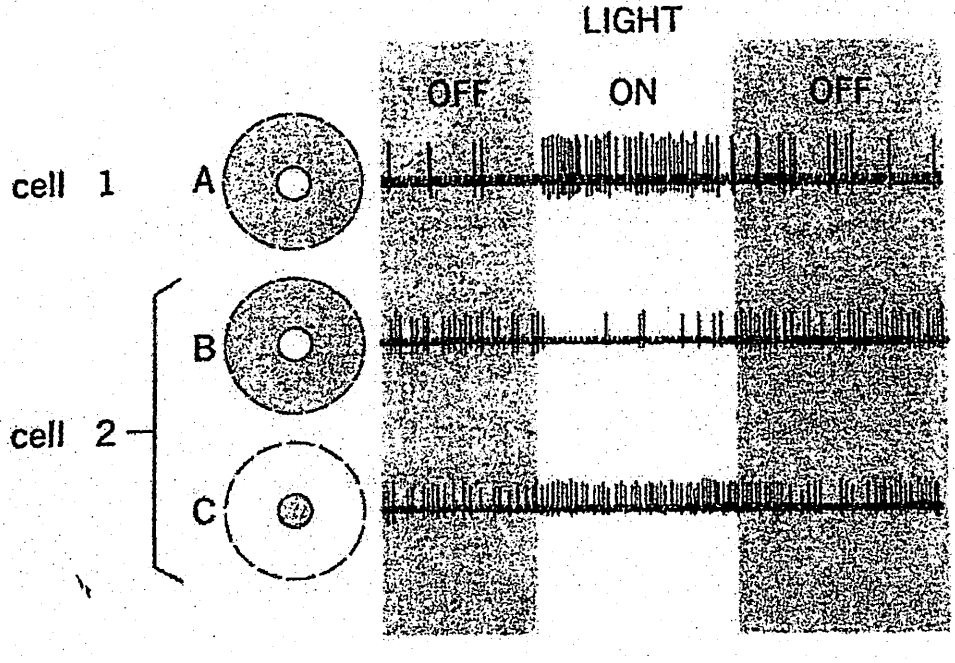
SISTEM PENGELIHATAN

Modul 29 Recoding of the activity of a single ganglion cell.

A- Response of an on-center ganglion cell (cell 1) which increased its activity when light stimulated the center of its
receptive field.

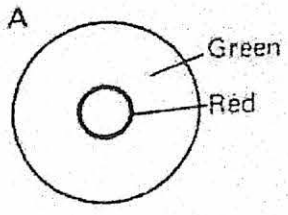
B-Activity of an off-center cell (cell 2) is suppressed when the center of its receptive field is stimulated.

C-The same off- center cell increase its activity when the light is restricted to the periphery.



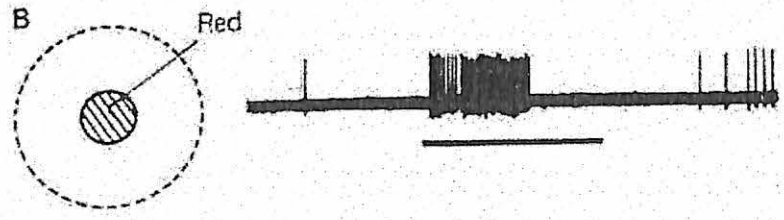
SISTEM PENGELIHATAN

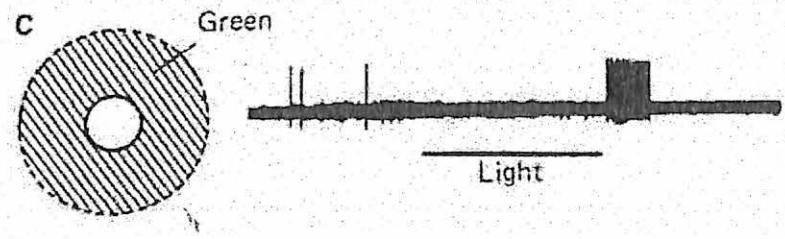
Modul 30 Responses of a concentric cell in the retina to a monochromatic stimulus.



A. The cell has a concentric red-green receptive field; the centre in excitary and sensitive to red; the surround is inhibitory and sensitive to green.
B. A red light shown on the center produces an excitatory "on" reponse.
C. A green light shown on the surround is inhibitory. There is on "off" discharge when it ceases.

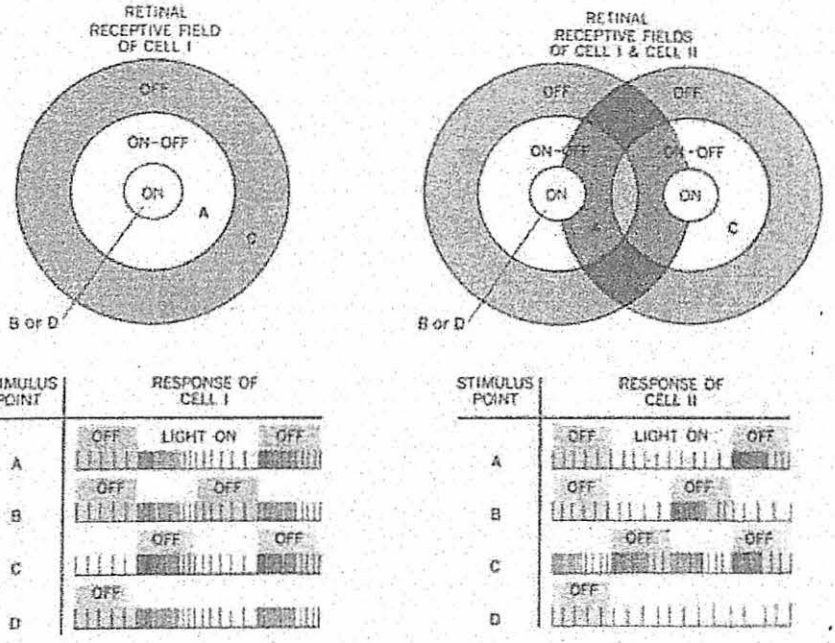
(Illumination is indicated by the bar below each record).





SISTEM PENGELIHATAN

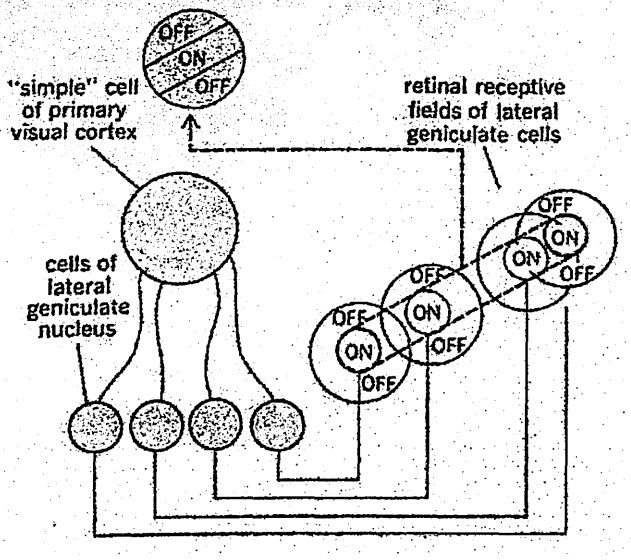
Modul 31 Light stimulus



Letters A, B, C and D on the receptive fields of the cell I and cell II represent points of light stimulus. A particular on –center ganglion cell (cell I) responds to the four different stimulus arrangements with only on pattern, therefore it conveys no specific information. The stimultaneous activity in two neurons discriminates between the four different event. In this case cell II alone provides specific information, but in other stimulus conditions cell II could fail to provide specific information, and activity of an additional neuron would be required.

SISTEM PENGELIHATAN

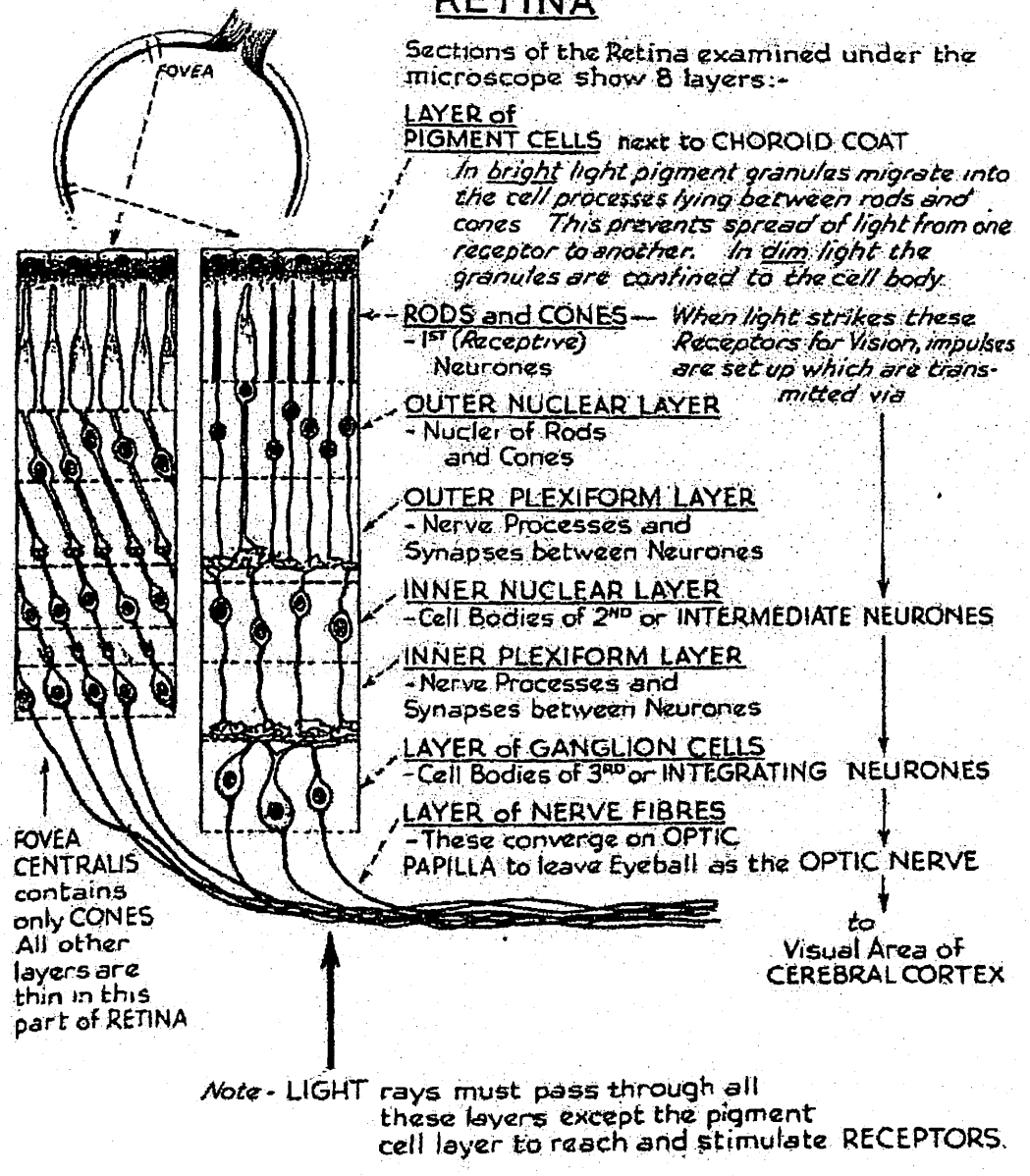
Modul 32 Receptive field



A possible way in which many on-centre retinal receptive fields could give rise to a slit-shaped receptive field in cortex.

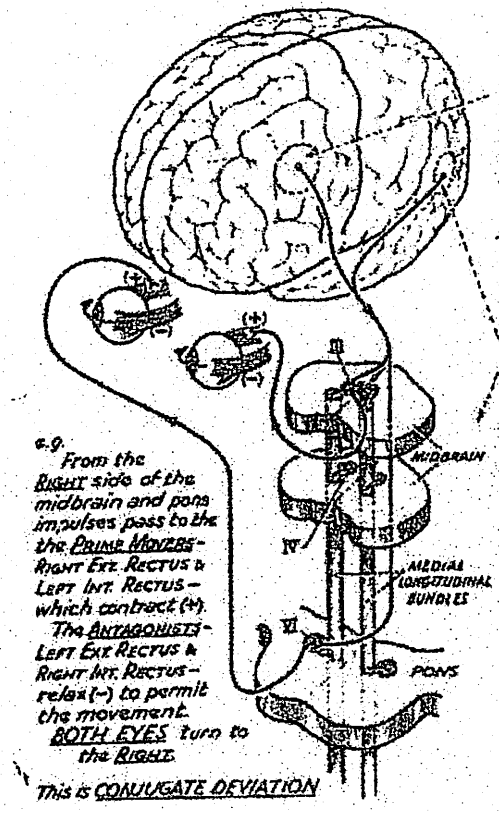
SISTEM PENGELIHATAN

Modul 33 Retina, Control of eye movements, Fondues oculi & Iris, lens and ciliary body



CONTROL of EYE MOVEMENTS

Both eyes normally move together so that images continue to fall on corresponding points of both retinae.



e.g. *From the RIGHT side of the midbrain and pons impulses pass to the the PRIME MOVERS - RIGHT EXT. RECTUS & LEFT INT. RECTUS - which contract (+).*
The ANTAGONISTS - LEFT EXT. RECTUS & RIGHT INT. RECTUS - relax (-) to permit the movement.
BOTH EYES turn to the RIGHT.
This is CONJUGATE DEVIATION

VOLUNTARY EYE MOVEMENTS are initiated in motor centres in FRONTAL LOBES

Impulses from one side of the Cerebral Cortex turn both eyes to the other side of Visual Field

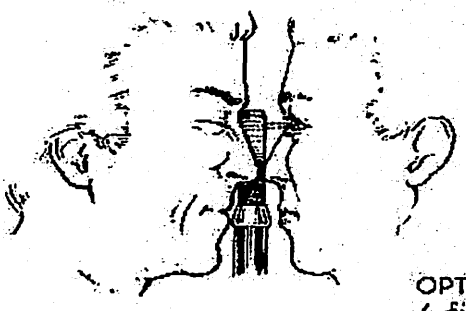
REFLEX EYE MOVEMENTS
Two Groups - (1) Those in response to Visual Stimuli. (2) Those in response to Non-Visual Stimuli.
In control of these are :-
CENTRES in OCCIPITAL LOBES:
CENTRES in MIDBRAIN and PONS which give rise to CRANIAL NERVES III, IV and VI.

Impulses from one side of the Midbrain and Pons turn eyes to the same side.

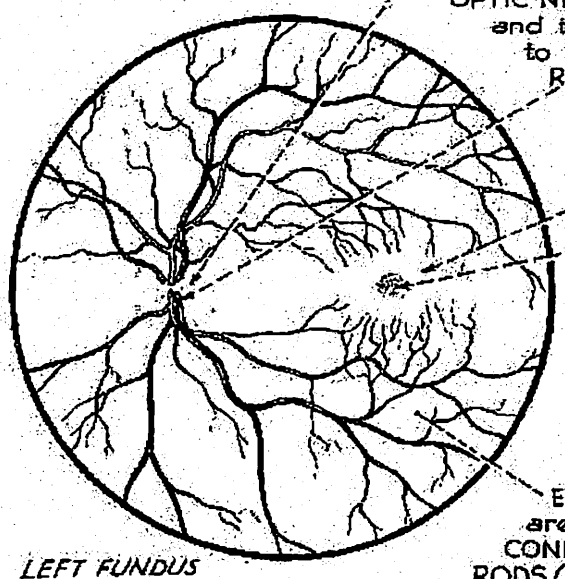
These centres are closely linked with each other and with HIGHER and LOWER centres in the Central Nervous System, so that the eyes are moved reflexly in response to many stimuli, e.g. loud noises or proprioceptive messages from vestibular organs.

FUNDUS OCULI

Part of the RETINA can be seen by means of an instrument – the OPHTHALMOSCOPE – which shines a beam of light through the PUPIL of the eye on to the RETINA.



The part of the Retina seen in this way is called the FUNDUS OCULI.



LEFT FUNDUS

OPTIC PAPILLA – or 'BLIND SPOT' – The nerve fibres from all parts of the retina converge on this area to leave the eyeball as the OPTIC NERVE. It has no RODS or CONES and therefore is not itself sensitive to light.

RETINAL BLOOD VESSELS enter or leave the eyeball here.

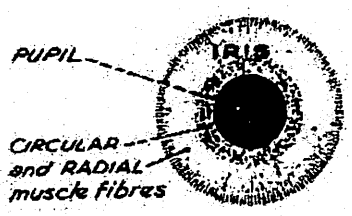
MACULA LUTEA – or 'YELLOW SPOT' – with

FOVEA CENTRALIS – area of acute vision – contains CONES only (the Receptors stimulated in BRIGHT and COLOURED LIGHT). When we look at an object the eyes are directed so that the image will fall on the fovea of each eye.

EXTRAFOVEAL part of Retina – area of less acute vision – CONES become fewer – RODS (the Receptors stimulated in DIM LIGHT with no discrimination between COLOURS) become more numerous towards the peripheral part of the Retina.

IRIS, LENS and CILIARY BODY

The IRIS is a muscular diaphragm with a central opening - the PUPIL.



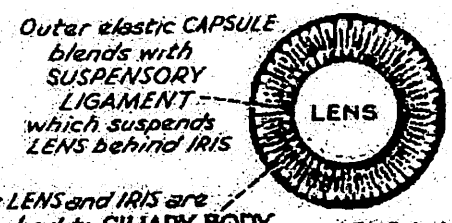
IRIS controls amount of LIGHT entering the EYE

CIRCULAR smooth muscle fibres - *SPHINCTER PUPILLAE* - contract to make Pupil smaller in bright light.

RADIAL fibres - *DILATOR PUPILLAE* - contract to make Pupil larger with change from LIGHT to DARK; NEAR to DISTANT vision (also with FEAR and PAIN)

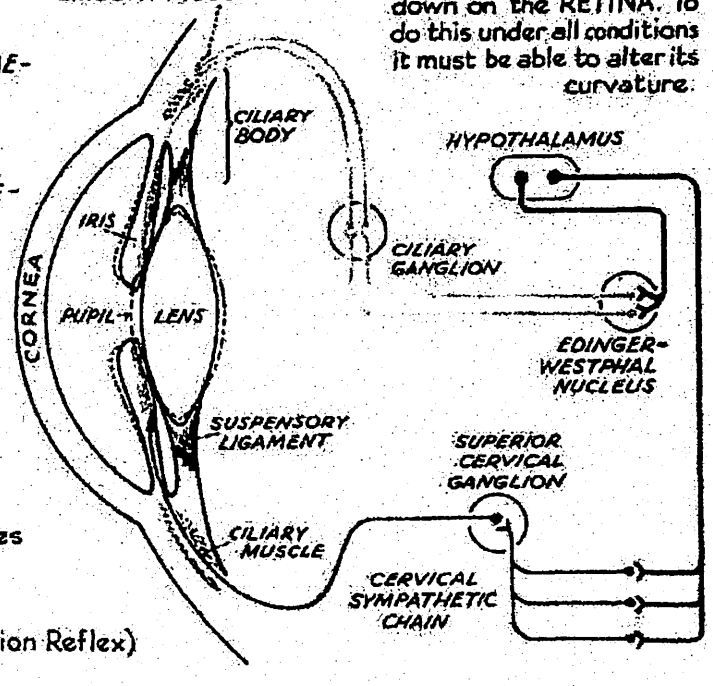
When CILIARY MUSCLE contracts, SUSPENSORY LIGAMENT is slackened. Tension on CAPSULE of LENS is relaxed. Anterior surface bulges forwards → LENS becomes more convex especially in its central part. This brings near objects into focus. (Accommodation Reflex)

The LENS is a transparent biconvex crystalline disc.



The LENS and IRIS are attached to CILIARY BODY which contains fibres of SMOOTH MUSCLE

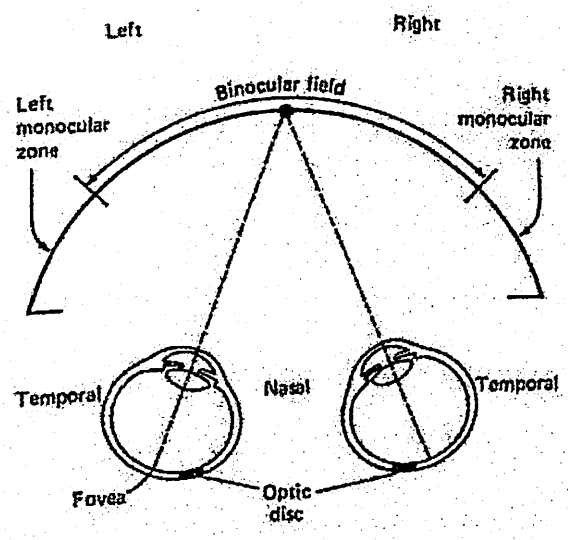
LENS brings LIGHT RAYS to a FOCUS upside down on the RETINA. To do this under all conditions it must be able to alter its curvature.



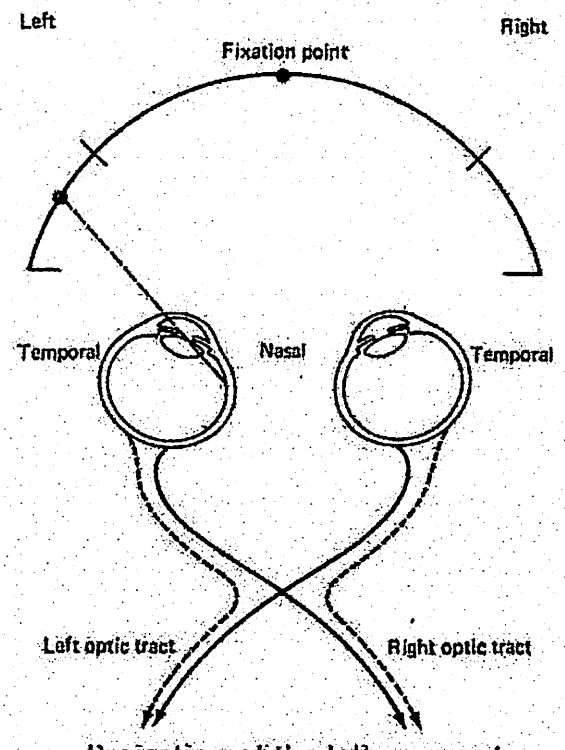
These changes are brought about reflexly. The ingoing impulses travel in the Optic nerves. The outgoing motor impulses travel in Parasympathetic to Ciliary Body and Sphincter Pupillae and in Sympathetic to Dilator Pupillae.

SISTEM PENGELIHATAN

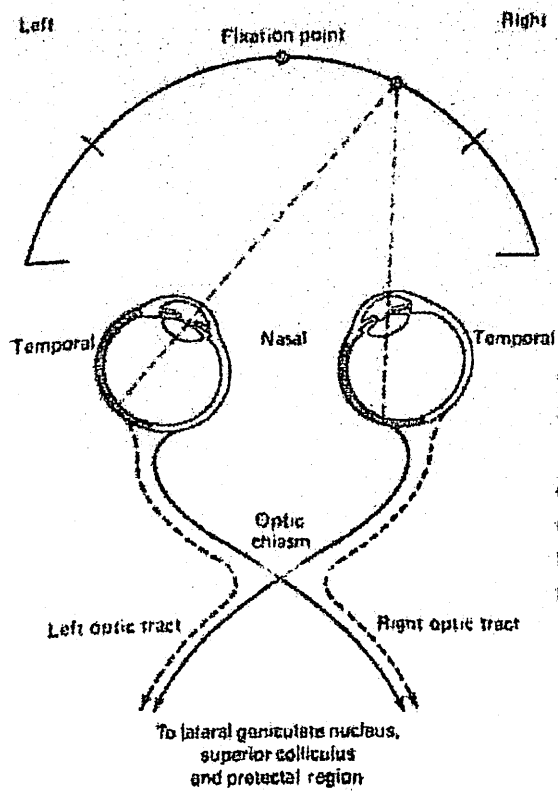
Modul 34 Organization of the visual field



Organization of the visual field. Light from the binocular zone strikes both eyes, light from the left or right monocular zone will strike only that eye. The regions of the retina are referred to as the temporal and nasal hemiretinas.



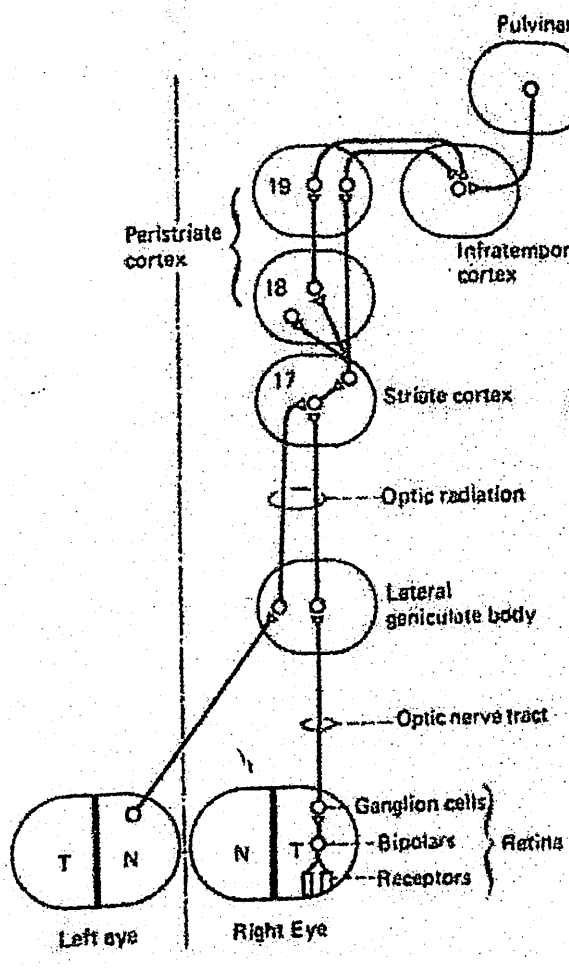
Projection of the left monocular crescent upon the retina.



Light from the rightbinocular field falls on the left temporal retina and the right nasal retina. Because fibers from the nasal retina of each eye cross to the opposite side at the optic chiasma, the left optic tract carries axons from the left temporal retina and the right nasan retina and therefore contains a complete representation of the right hemifield of vision.

SISTEM PENGELIHATAN

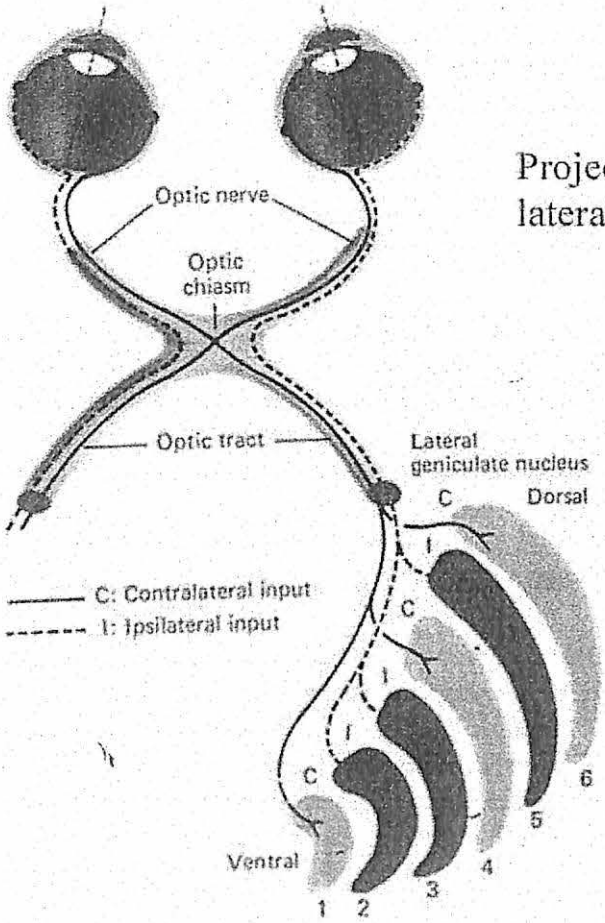
Modul 35 Highly schematic diagram of the visual projections to the cortex



The right hemiretina of each eye (the nasal hemiretina of the eye and the temporal hemiretina of the right eye) projects to the right lateral geniculate nucleus via the optic nerve and tract (the left hemiretinas project to the left lateral geniculate nucleus). From the lateral geniculate nucleus, neurons project via the optic radiations to the primary visual cortex (brodmann's area 17, or visual area 1). This area is also called the striate cortex. From the striate cortex, neurons project to the peristriate cortex (brodmann's area 18 + 19. Or visual areas II and III). Neurons from area 17 also project to the superior coliculus, and back to the lateral genicuate nucleus (another example of central of sensory input). From the peristraite cortex, neurons project to the infratemporal cortex. The infratemporal cortex also receives input from the pulvinar of the thalamus.

SISTEM PENGELIHATAN

Modul 36 Projection of the retinas upon the lateral geniculate nucleus.



Projection of the retinas upon the lateral geniculate nucleus.

A substantial number of the fibers in the optic tract terminate In the lateral geniculate nucleus, a knee-shaped structure in the posterior aspect of the thalamus. In this nucleus there is an orderly representation of the contralateral visual hemifield. Ganglion cells at different local in the retina project upon distinct visuotopic points in the lateral geniculate nucleus.

In primates, the lateral geniculate nucleus of the thalamus consists of six layers of neurons seperated by intervening layers of axons and dendrites. The layers are numbered from 6 most dorsally to 1 most ventrally. An individual layer in the neclues receives input from one eye only fibers from the contralateral nasal retina contact layers 6,4 and 1; fibers from the ipsilateral temporal retina contact layers 5,3 and 2. Thus, the complementary halves of the retina in both eyes each contact individual layers. In a topograptically ordered way, so that each layer contains a precise representation of the contralateral visual filed. These representation are stacked on top of one another in the layers of the nucleus, which contains six maps of the contralateral hemifield as a result.

SISTEM PENGELIHATAN

Modul 37 The intraocular fluid

The eye is filled with intraocular fluid which maintains sufficient pressure in the eyeball to keep it distended. This fluid can be divided into two portions, the aqueous humor, which lies in front and to the sides of the lens, and the vitreous humor, which lies between the lens and the retina. The aqueous humor is a freely flowing fluid, while the vitreous humor, sometimes called the vitreous body, is a gelationous mass held together by a fine fibrillar network. Substances can diffuse slowly in thevitreous humor, but there is little flow of fluid.

Aqueous humor is continually being formed and reabsorbed. The balance between formation and reabsorption of aqueous humor regulates the total volume and pressure of the intraocular fluid.

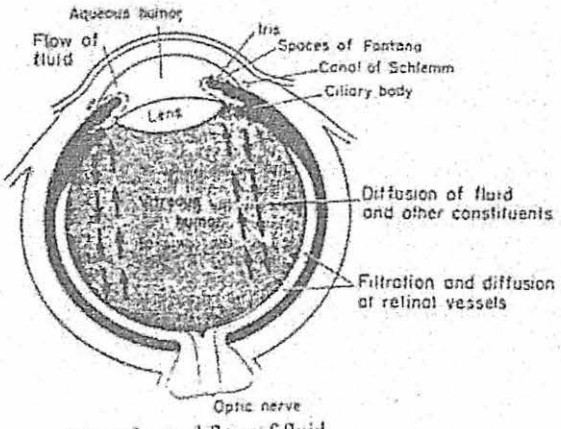
Formation of aqueous humor by the ciliary body

Aqueous humor is formed in the eye at an average rate of 2 to 3 cubic millimeters each minute. Essentially all of this is secreted by the ciliary processes, which are linear folds projecting from the ciliary body into the space behind the iris where the lens ligaments also attach to the eyeball. The fluid chambers of the eye can be seen in figure. Because of their folded architecture, the total surface area of the ciliary processes is approximately 6 square centimeters in each eye a large area, considering the small size of the ciliary body. The surfaces of these processes are covered by epithelial cells, and immediately beneath these is highly vascular area.

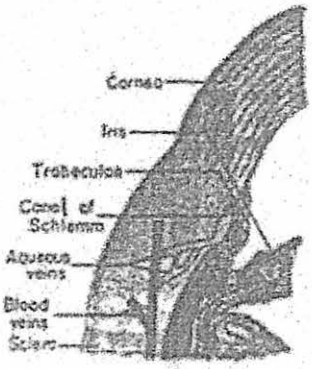
Aqueous humor is formed by the ciliary processes in much the same manner that cerebrospinal fluid formed by the choroid plexus. The postulated mechanism is the following: it is believed that the ciliary epithelium actively secretes sodium chloride, and probably bicarbonate ions into the spaces between the cells. This in turn causes osmosis of water into these spaces, and the resulting solution then oozes out of the surfaces of the ciliary processes. In addition, several nutrients are transported across the epithelium by active transport or facilitated diffusion; these include amino acids, ascorbic acid, and probably also glucose.

Outflow of aqueous humor from the eye

After aqueous humqr is formed by the ciliary processes, it flows, as shown in figure, between the ligaments of the lens, then through the pupil and finally into the anterior chamber of the eye. Here, the fluid flows into the angle between the cornea, and the iris and thence through a meshwork of trabeculae, finally into the canal of schlemm. The canal of schlemm in turn is a thin-walled vein that extends circumferentially all the way around the eye. Its endothelial membrane is so porous that even large protein molecules, as well as small particulate matter, can pass from the anterior chamber into the canal of schlemm. Even thouch. The canal of schlemm is actually a venous blood vessel, so much aqueous humor normally flows into it that is filled only with aqueous humor rather than with blood. Also, the small veins that lead from the canal of schlemm to the larger veins of the eye usually contain only aqueous humor, and these are called aqueous veins.



Formation and flow of fluid



Anatomy of the iridocorneal angle, showing the system for outflow of aqueous humor into the conjunctival veins.

SISTEM PENGELIHATAN

Modul 38 Intraocular pressure

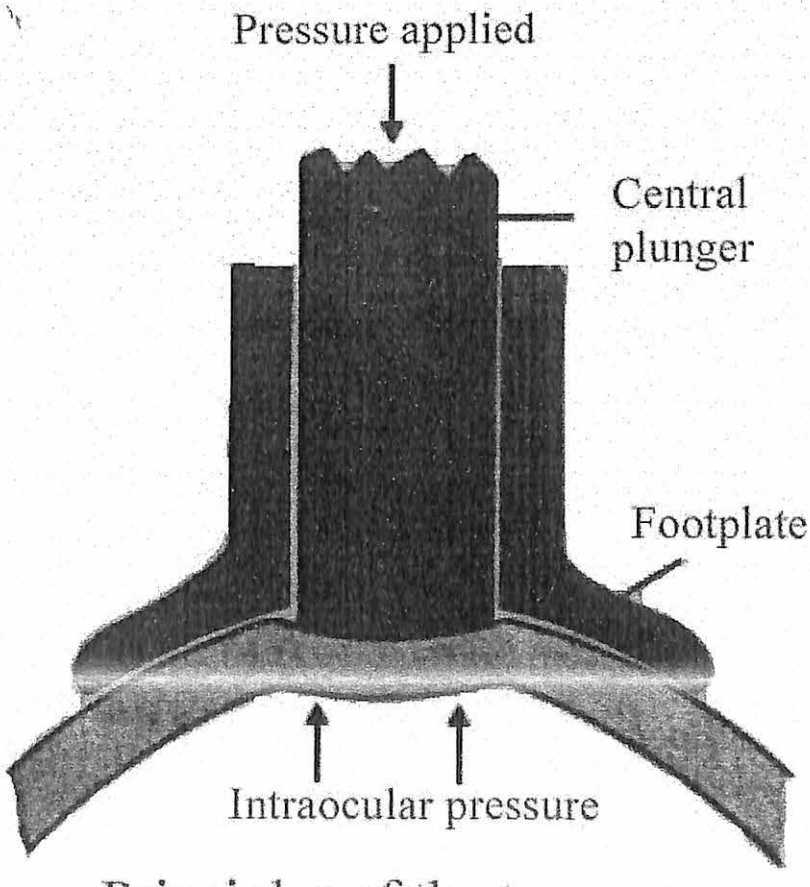
The average normal intraocular pressure is approximately 16 mmHg, with a range from 12 to 20.

Tonometry.

Because it is impractical to pass a needle into a patient's eye for measurement of intraocular pressure, this pressure is measured clinically by means of a tonometer, the principle of which is illustrated in figure. The cornea of the eye is anesthetized with a local anesthetic, and the footplate of the tonometer is placed on the cornea. A small force is then applied to a central plunger, causing the central part of the part of the cornea to be displaced inward. The amount of displacement is recorded on the scale of the tonometer, and this in turn is calibrated in terms of intraocular pressure.

Regulation of intraocular pressure.

The intraocular pressur of the normal eye remains almost exactly constant throughout life, illustrating that the pressure regulating mechanism is very effective. But the precise operation of this mechanism is not clear. The pressure is regulated mainly by the outflow resistance from the anterior chamber into the canal of schlemm, presumably the entrance of the fluid into the canal of schlemm are actually laminar plates that lie one on top of the others each of the plates is penetrated by numerous small holes. When the plates are compressed against each other, each successive plate partially blocks the holes in the next plate. An increase in pressure above normal is believed to distend the spaces between the plates and therefore to open the holes, thus causing rapid flow into the canal of schlemm and decrease of the pressure back to normal. On the other hand, a decrease in pressure below normal allows the plates to impinge upon each other, thus preventing fluid loss until the pressure rises again back to normal. Thus, this mechanism acts as an automatic feedback regulatory system for keeping the intraocular prssure at a nearly constant level day in and day out.



Principles of the tonometer

Cleansing of the trabecular spaces and of the intraocular fluid.
When large amounts of debris occur in the aqueous humor, as occurs following hemorrhage into the eye or during intraocular infection, the debris is likely to accumulate in the abecular spaces, therefore preventing adequate reabsorption of fluid from the anterior chamber and sometimes causjng glaucoma, however, on the surfaces of the trabecljlar lates are large numbers of phagocytic cells. Also, immediately outside the canal of schlemm - between the canal and the trabecular plates is a layer of interstitial gel containing large numbers of reticuloendothelial cells that have an extremely high capacity for both engulfing debris and degrading it into small molecular substances that can then be absorbed. Thus, this phagocytic system keeps the trabecular spaces cleaned.

In addition, the surface of the iris and other surfaces of the eye behind the iris are covered with an epithelium that is capable of phagocytizing proteins and small particles from the aqueous humor, thereby helping to maintain a perfectly clear fluid. This epithelium can also transport many toxic substances out of the aqueous humor, thereby helping to maintain the chemical purity of the internal environment of the eye.

Glaucoma.
Glaucoma is one of the most common causes of blindness. It is disease of the eye in which the intraocular pressure becomes pathologically high, sometimes rising to as high as 70 mmhg. Pressures rising above as little as 25 to 30 mmHg. Can cause loss of vision when maintained for many years. And the extremely high pressures can cause blindness within days. As the pressure rises, the retinal artery, which enters the eyeball at the optic disc, is compressed, thus reducing the nutrition to the retina. This often results in permanent atrophy of the retina and optic nerve, with consequent blindness.

In essentially all cases of glaucoma the abnormally high pressure results from increased resistance to fluid outflow at the irido-corneal junction. In most patients, the cause of this is unknown, but in some it results from infection or trauma to the eye. As explained above, red blood cells, white blood cells, and tissue debris block the outflow of fluid,
thereby greatly increasing the intraocular pressure.

SISTEM PENGELIHATAN

Modul 39 Optical defects and refractiue amomalies

The lens system in a modern camera can produce a considerably better image than the dioptric apparatus of the eye. The physicist and physiologist Hermann V. Helmholtz (1a21-1894) once wrote that if he should receive an optical instrument so carelessly constructed as the eye, he would send it back to the maker. The "physiological" deficiencies in focussing by the eye discussed here, however, are largely compensated by
neuronal contrast mechanisms.

Astigmatism.
The corneal surface is not rotationally symmetric about the optical axis, for the vertical curvature is usually somewhat greater than the horizontal curvature. Tris discrepancy results in an angle-dependent difference in refractive power (astigmatism or astigmia). If the difference is no greater than 0.5 d, the condition is called "physiological"
astigmatism.

D-DIOPTER - UNIT OF REFRACTIVE POWER

Spherical aberration.
The cornea and the lens of the eye, like simple lenses, have shorter focall lengths in the peripheral regions than in the central part around the optical axis. The resulting spherical aberration causes blurring of the image. The smaller the pupils the more the peripheral rays are excluded and the less distortion is caused by spherical aberration.

Chromatic aberration and accommodation.
As do all simple lense, the dioptric apparatus refracts short-wavelength light more strongly than long-wavelength light (chromatic aberration). Therefore greater accommodation is required for sharp focussing of the red parts of an object than for the blue parts. It is because of this difference that blue objects appear to be further away than red ones at the same objective distance. The builders of gothic churches often exploited this physiological illusion in their stainedglass windows, by making the background blue and the fig uses other colors, so that one sees an apparent spatial separation between figures and background.

Stray light and clouding of the dioptric apparatus.
The lens and the vitreous body contain structural proteins and macromolecular substances in colloidal solution. Therefore a slight diffuse dispersion of the light occurs in the dioptric apparatus. But this stray light impairs visual perception only with very bright stimuli. Even in a healthy eye there are cloudy areas in the vitreous body. Visible against a white wall as small disks or irregularly shaped small gray spots. When the eye moves they seem to flit like gnats across the light background. In older people the water content of the lens can decline so greatly that the structure of the remaining material becomes
condensed, making the lens opaque (senile cataract). Removal of the lens enables these patients to see normally when they are fitted with spectacles having a strong convex lens (CA. + 13 D for long-distance vision).

Myopia.
The total refractive power, of the dioptric apparatus of a normal, unaccommodated eye is 58.6 diopter. With this refractive power an infinitely distant object is focussed sharply on the retina when the distance between the pole of the cornea and the fovea is 24.4 mm. If the axial length of the eyeball is greater distant objects cannot be seen sharply because the plane of focus is in front of the fovea (nearsightedness, myopia). A nearsighted person must wear glasses with concave lenses (negative refractive power) to see sharply at a distance.

Hypermetropia.
If the axial length is too short for the refractive power of the dioptric apparatus, a condition of "farsightedness" (hyperopia or hypermetropia) exists. The hypermetropic person can see distant objects clearly by near-accommadating to some extent, but his accommodation range is not sufficient to allow sharp focussing of nearby objects. Convex lenses (positive refractive power) are required to compensate this defect.

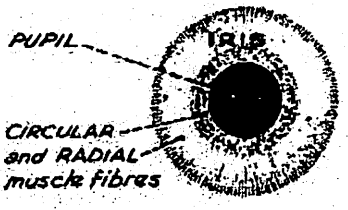
SISTEM PENGELIHATAN

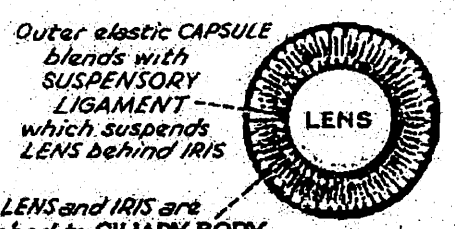
Modul 40 Eye and the retina

IRIS, LENS and CILIARY BODY

The IRIS is a muscular diaphragm with a central opening - the PUPIL

The LENS is a transparent biconvex crystalline disc.





IRIS controls amount of LIGHT entering the EYE

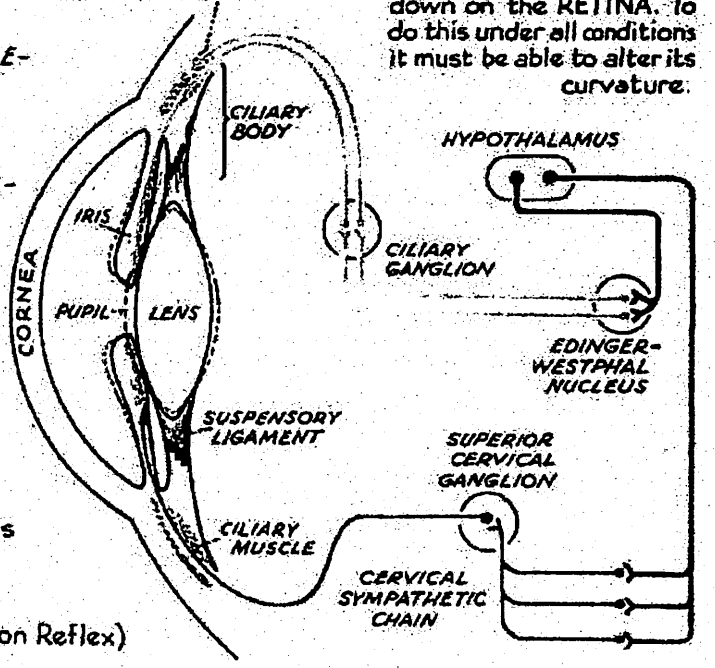
CIRCULAR smooth muscle fibres - *SPHINCTER PUPILLAE* - contract to make Pupil smaller in bright light.

RADIAL fibres - *DILATOR PUPILLAE* - contract to make Pupil larger with change from LIGHT to DARK: NEAR to DISTANT vision (also with FEAR and PAIN)

When CILIARY MUSCLE contracts, SUSPENSORY LIGAMENT is slackened. Tension on CAPSULE of LENS is relaxed. Anterior surface bulges forwards → LENS becomes more convex especially in its central part. This brings near objects into focus. (Accommodation Reflex)

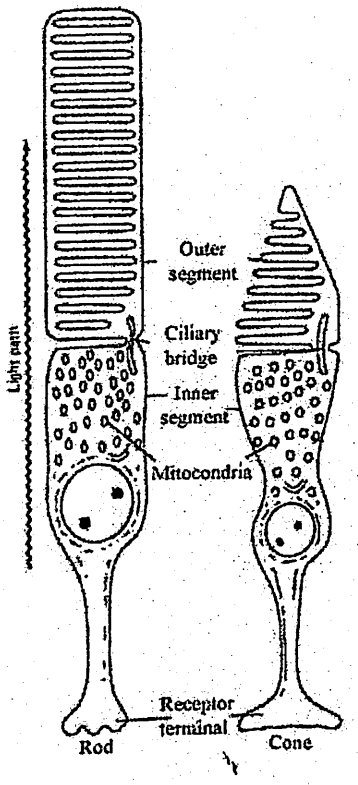
The LENS and IRIS are attached to CILIARY BODY *which contains fibres of SMOOTH MUSCLE*

LENS brings LIGHT RAYS to a FOCUS upside down on the RETINA. To do this under all conditions it must be able to alter its curvature.



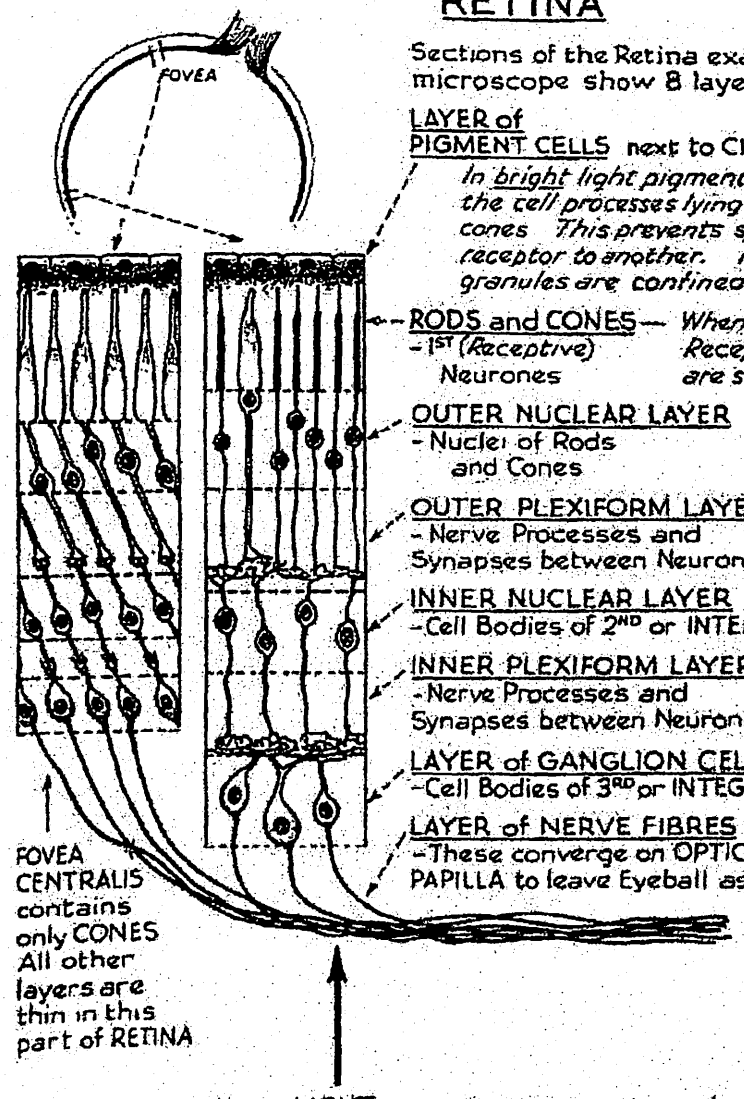
These changes are brought about reflexly. The ingoing impulses travel in the Optic nerves. The outgoing motor impulses travel in Parasympathetic to Ciliary Body and Sphincter Pupillae and in Sympathetic to Dilator Pupillae.

Structure of photoreceptors.



Structure of photoreceptors. Both rod and cone cells are differentiated into inner and outer segments connected by a ciliary bridge.
The inner segments of both cell types contain the nucleus and most of the biosynthetic machinery and are continuous with the receptors terminal, the membranous disc in the outer segments of rod cells (unlike those in cone cells) are nut
connected with the plasma membrane.

RETINA



Sections of the Retina examined under the microscope show 8 layers:-

LAYER of PIGMENT CELLS next to CHOROID COAT

In bright light pigment granules migrate into the cell processes lying between rods and cones. This prevents spread of light from one receptor to another. In dim light the granules are confined to the cell body.

RODS and CONES — 1ST (Receptive) Neurones

When light strikes these Receptors for Vision, impulses are set up which are transmitted via

OUTER NUCLEAR LAYER
- Nuclei of Rods and Cones

OUTER PLEXIFORM LAYER
- Nerve Processes and Synapses between Neurones

INNER NUCLEAR LAYER
- Cell Bodies of 2ND or INTERMEDIATE NEURONES

INNER PLEXIFORM LAYER
- Nerve Processes and Synapses between Neurones

LAYER of GANGLION CELLS
- Cell Bodies of 3RD or INTEGRATING NEURONES

LAYER of NERVE FIBRES
- These converge on OPTIC PAPILLA to leave Eyeball as the OPTIC NERVE

to Visual Area of CEREBRAL CORTEX

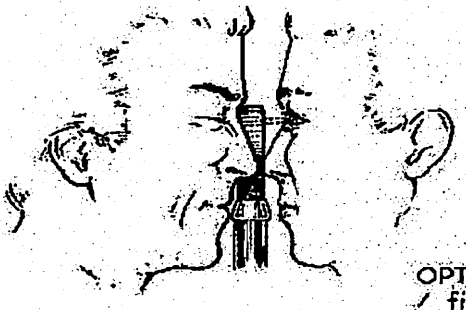
FOVEA CENTRALIS contains only CONES All other layers are thin in this part of RETINA

Note - LIGHT rays must pass through all these layers except the pigment cell layer to reach and stimulate RECEPTORS.

Polarization of cells in the on-line retinal pathway involving a depolarizing bipolar cells.

FUNDUS OCULI

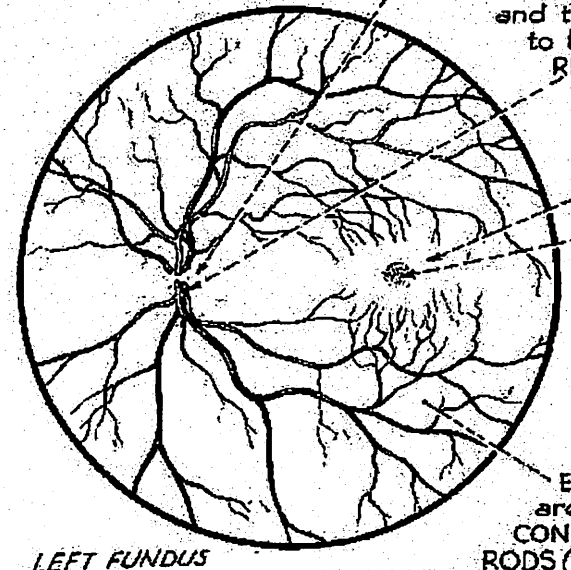
Part of the RETINA can be seen by means of an instrument – the OPHTHALMOSCOPE – which shines a beam of light through the PUPIL of the eye on to the RETINA.



The part of the Retina seen in this way is called the FUNDUS OCULI.

OPTIC PAPILLA – or 'BLIND SPOT' – The nerve fibres from all parts of the retina converge on this area to leave the eyeball as the OPTIC NERVE. It has no RODS or CONES and therefore is not itself sensitive to light.

RETINAL BLOOD VESSELS enter or leave the eyeball here.

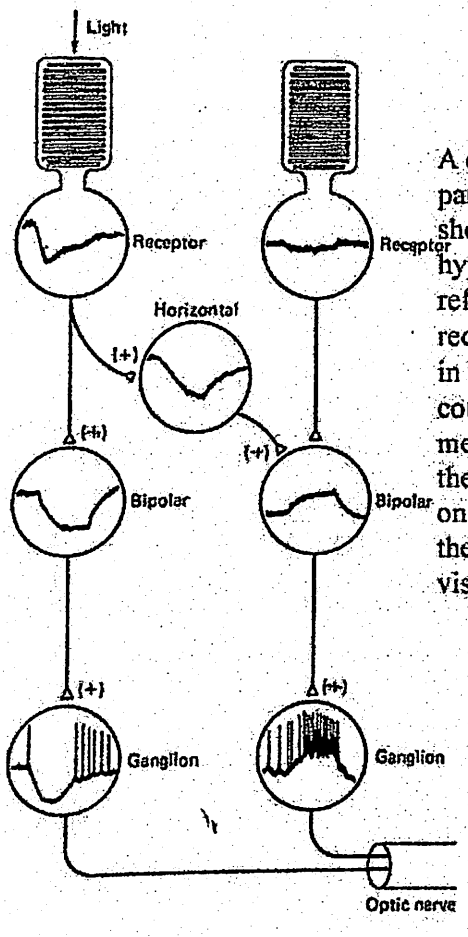


MACULA LUTEA – or YELLOW SPOT – with FOVEA CENTRALIS – area of acute vision – contains CONES only (the Receptors stimulated in BRIGHT and COLOURED LIGHT). When we look at an object the eyes are directed so that the image will fall on the fovea of each eye.

EXTRAFOVEAL part of Retina – area of less acute vision – CONES become fewer – RODS (the Receptors stimulated in DIM LIGHT with no discrimination between COLOURS) become more numerous towards the peripheral part of the Retina.

LEFT FUNDUS

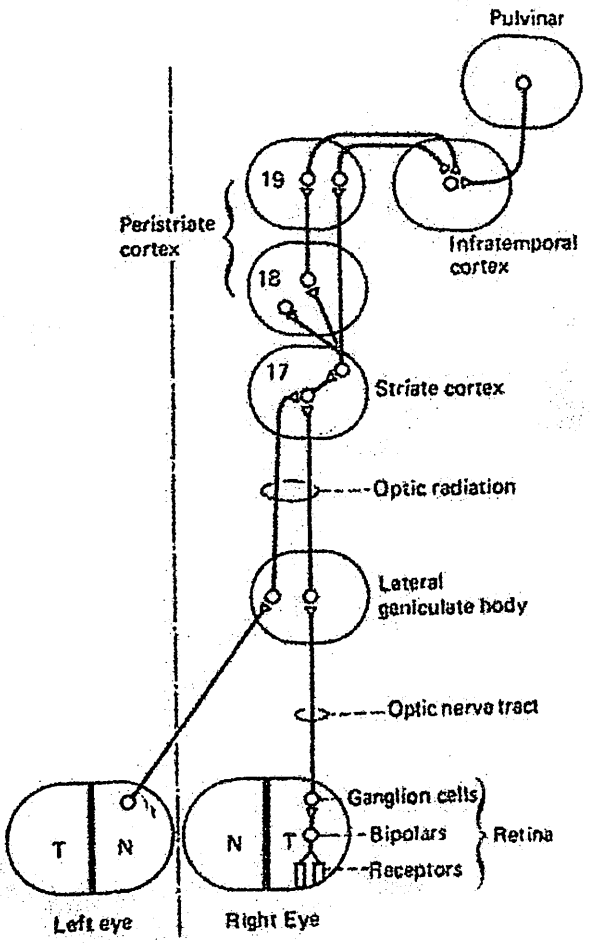
Polarization of cells in an antagonistic surround receptive field.



A direct on-line pathway is shown on the left, and an off-line pathway mediated by the laterally oriented horizontal cell is shown on the right. The flash of light (arrow) produces a large hyperpolarizing response only in the receptor directly beneath it, reflecting the relatively narrow receptive field found at the receptor level in many retinas. The small deflection observed in the distant receptor on the right may be due to electrical coupling between the receptor cells. The horizontal cell mediates an antagonistic surround receptive field at the level of the bipolar cells and stimulates the hyperpolarizing bipolar cell on the right. This receptive field organization is transferred to the ganglion cell, where its expression is utilized to accentuate visual contrast +, excitatory transmitter; -, inhibitory transmitter.

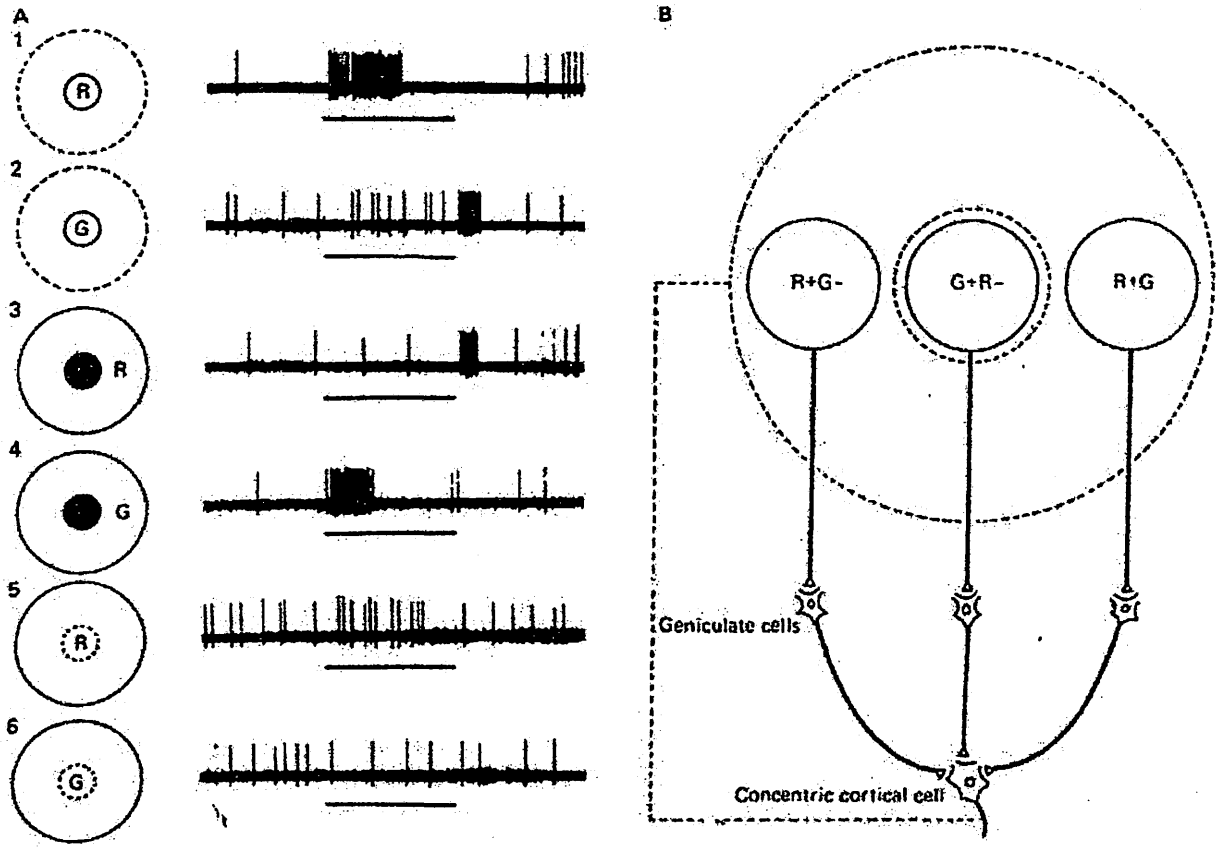
SISTEM PENGELIHATAN

Modul 41 Physiology of the central visual pathway receptive fields



Highly schematic diagram of the visual projections to the cortex.

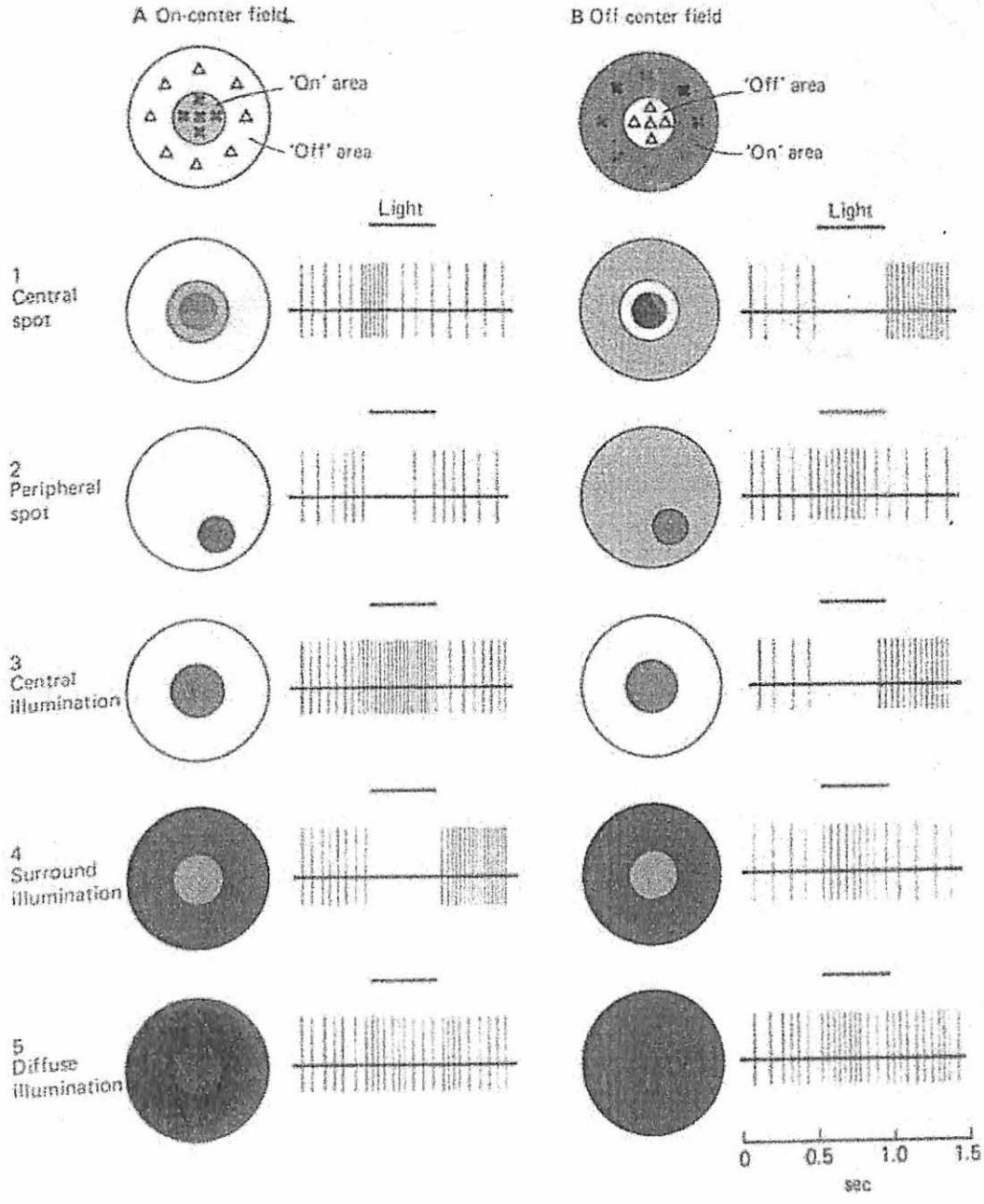
Color-coded cortical cells with concentric receptive fields.



(A). Responses of a light adapted concentric cell to monochromatic stimuli. The field center was 0.5° in diameter; the total diameter of the field was 8°. Stimuli were a 0. 5° spot, 0.5° inner diameter and - 8° outer diameter annulus, and an 8° spot. 1: A centered red spot produced an "on" outer diameter discharge. 2: A centered green spot produced an "off" discharge. 3: A red annulus evoked an "off" response. LI: A green annulus evoked an "on" response. 5, and 6: Larger monochromatic spots were without effect. The 1-sec. stimulus duration is indicated by the bar below each oscilloscope trace. Duration of each sweep was 3 sec.; wavelengths were 630nm for red and 500nm for green.

(B). Proposed synaptic mechanism to explain the response properties and receptive field organization of the concentric cell. Solid circles: receptive fields of the geniculate fibers; broken circles: center and surround of the concentric cortical cell' s field. A single geniculate afferent has a circular receptive field (in this case green on, red off). Which coincides with the cell' s field center. Encompassing in an annular fashion the field of the central efferent are the circular fields of a large group of geniculate fibers, only two of with are shown here. Fields of these units, which collectively form the surround of the cell' s field, are all of the same opponent-color type and are the same diameter. The central fiber and the surround group have the opposite opponent-color organization. All synapses are excitatory.

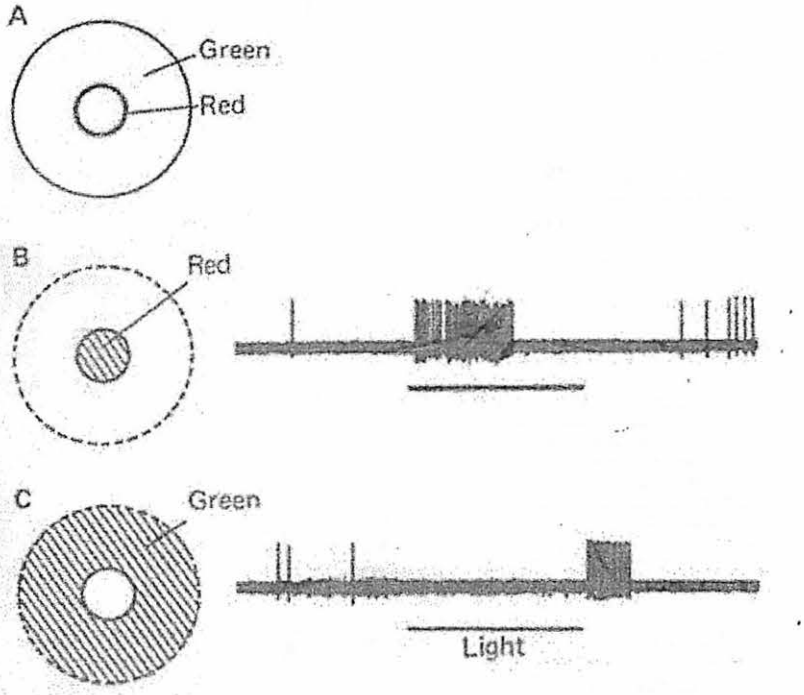
Responses of on-center and off-center retinal gang lion cells different types of illumination. (X excitatory zone, D inhibitory zone).



A) An on-center cell responds best to a spot of light shone onto the central part of its receptive field (1). Illumination (bar above records) of the surrounding area with a spot (2) or an annulus (4) of light reduces or suppresses the discharges and causes a response when the light is turned off. Diffuse illumination of the entire receptive field (5) elicits a relatively weak discharge because center and surround oppose each other' s effects.

B) A cell with off-center receptive field has its spontaneous firing suppressed when the central area of its. field is illuminated (1,3) and accelerated when the light is turn off. Light shone onto the surround of an off-center receptive field area excites (2,4).

Responses of a concentric cell in the retina to a monochromatic stimulus.



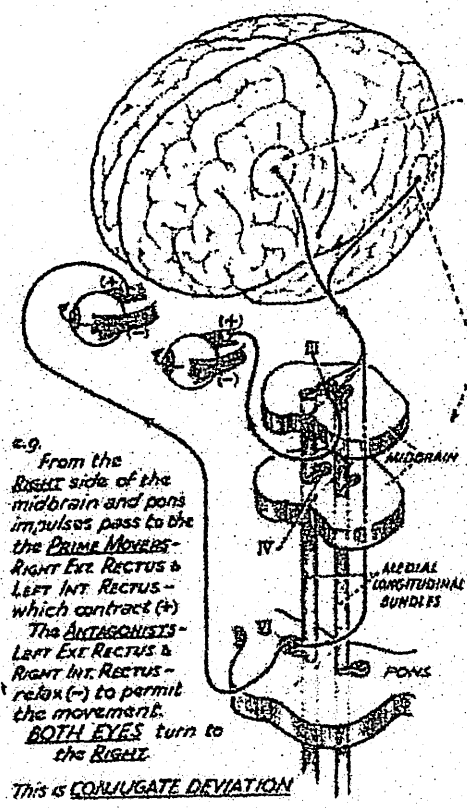
(A). The cell has a concentric red-green receptive field, the center is excitatory and sensitive to red; the surround is inhibitory and sensitive to green.
(B). A red light shown on the center produces an excitatory "on" response.
(C) A green light shown on the surround is inhibitory. There is an "off"discharge when it ceases.
Illumination is indicated by the bar below each record.

SISTEM PENGELIHATAN

Modul 42 Visual field projects upon the retina normal

CONTROL of EYE MOVEMENTS

Both eyes normally move together so that images continue to fall on corresponding points of both retinae.



VOLUNTARY EYE MOVEMENTS are initiated in motor centres in FRONTAL LOBES.

Impulses from one side of the Cerebral Cortex turn both eyes to the other side of Visual Field

REFLEX EYE MOVEMENTS

Two Groups – (1) Those in response to Visual Stimuli. (2) Those in response to Non-Visual Stimuli. In control of these are:-

CENTRES in OCCIPITAL LOBES:

CENTRES in MIDBRAIN and PONS which give rise to CRANIAL NERVES III, IV and VI.

Impulses from one side of the Midbrain and Pons turn eyes to the same side.

These centres are closely linked with each other and with HIGHER and LOWER centres in the Central Nervous System, so that the eyes are moved reflexly in response to many stimuli, eg loud noises or proprioceptive messages from vestibular organs.

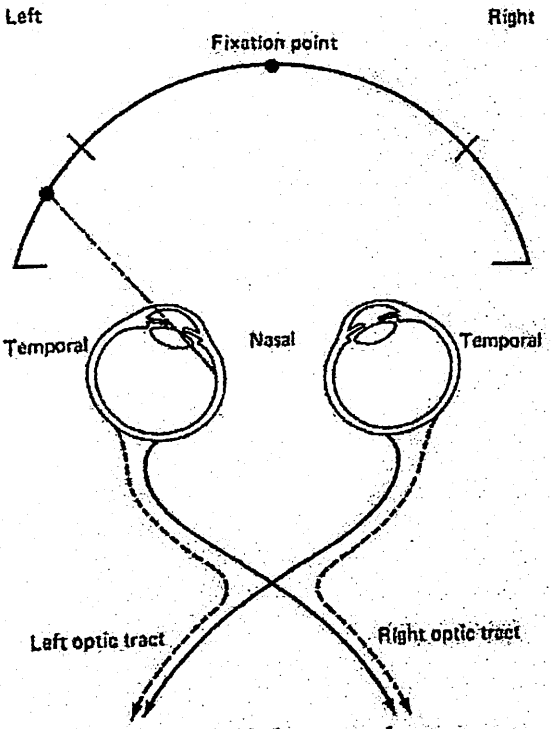
e.g.

From the RIGHT side of the midbrain and pons impulses pass to the the PRIME MOVERS – RIGHT EXT. RECTUS & LEFT INT. RECTUS – which contract (+)

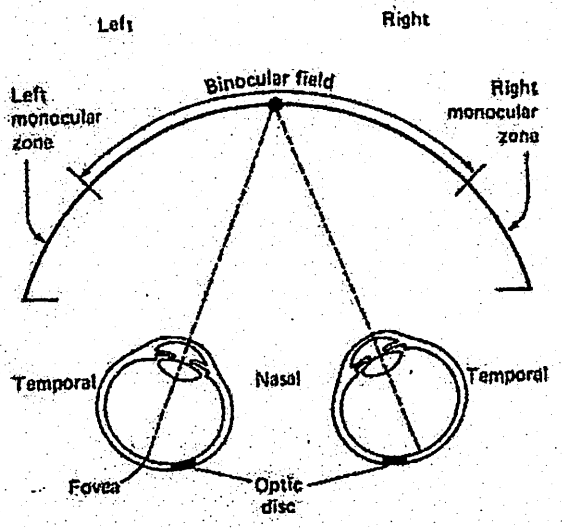
The ANTAGONISTS – LEFT EXT. RECTUS & RIGHT INT. RECTUS – relax (–) to permit the movement.

BOTH EYES turn to the RIGHT.

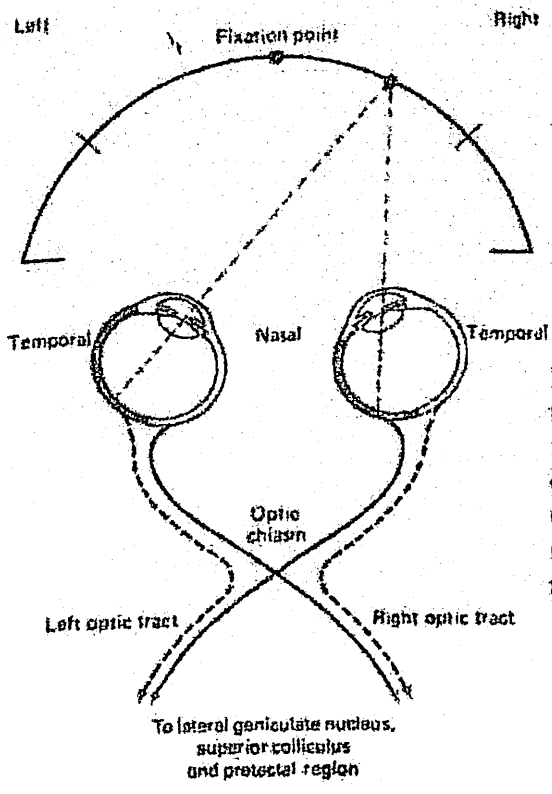
This is CONJUGATE DEVIATION



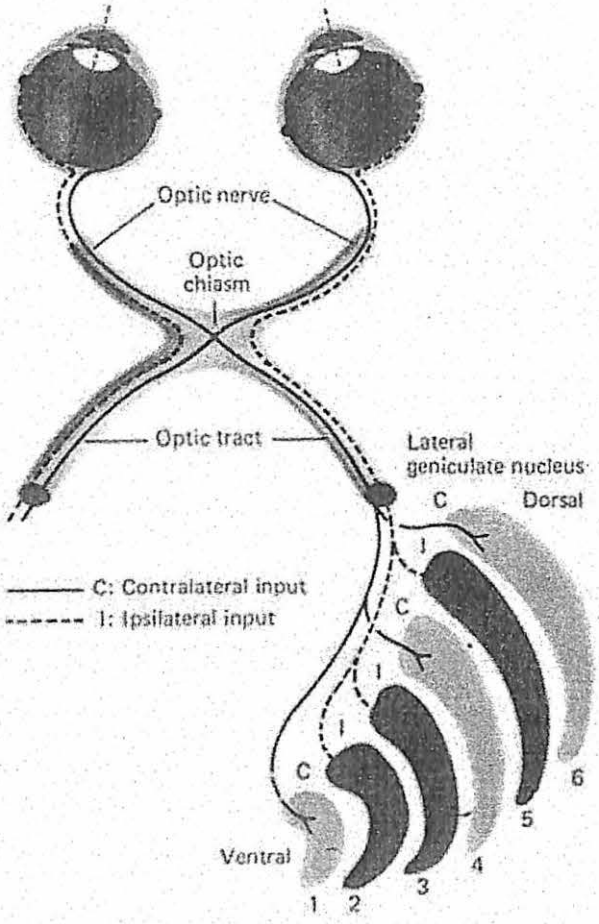
Projection of the left monocular crescent upon the retina.



Organization of the visual field. Light from the binocular zone strikes both eyes, light from the left or right monocular zone will strike only that eye. The regions of retina are referred to as the temporal and nasal hemiretinas.



Light from the rightbinocular field falls on the left temporal retina and the right nasal retina. Because fibers from the nasal retina of each eye cross to the opposite side at the optic chiasma, the left optic tract carries axons from the left temporal retina and the right nasan retina and therefore contains a complete representation of the right hemifield of vision.


Optic nerve
Optic chiasm
Optic tract
Lateral geniculate nucleus
C
Dorsal
I
C: Contralateral input
I: Ipsilateral input
C
I
I
C
6
5
4
3
Ventral
1
2

SISTEM PENGELIHATAN

Modul 43 The responses of retinal ganglion cells to colored light

Stimuli.

Behavioral measurements imply that the color vision of animals such as the rhesus monkey is similar to that of humans. The functional organization of the receptive fields of retinal ganglion cells in these animals, however, is more complex than the "on"- center or "off"- center pattern described above. In the lightadapted retina, as well as in the lateral geniculate body and the visual cortex of these animals, nerve cells can be found which have partially color-specific responses. Three classes of such cells are distinguished (fig. below).

The ganglion cells of the light/dark system react in qualitatively the same way ("on"- center or "off"- center properties) regardless of the wavelength of the monochromatic light stimulus, in the visible spectrum (~400-700 nm). The spectral sensitivity is no different in the center than in the periphery of the receptive fields of ganglion cells in this class.

The ganglion cells of the red/green system have in part a color-specific antagonistic rf organization. Monochromatic light stimuli in the red region of the spectrum elicit activation in the rf center and inhibition in the periphery, whereas monochromatic stimuli in the green region of the spectrum produce the reverse responses.

The ganglion cells of the yellow/blue system, when the rf center is stimulated monochromatically, are activated by yellow light and inhibited by blue: conversely, in the periphery a yellow light inhibits and blue light excites.

The retinal and geniculate neurons thus perform a transformation relevant to color vision. By way of the bipolar cells, horizontal cells and amacrines the signals from the three different cone types are directed so as to provide the ganglion cell layer with a neuronal system

For "achromatic vision" and two color-specific, antagonistic neuron systems. The latter constitute a four-color. Opponent system with the opponent color pairs yellow/blue and red/green.

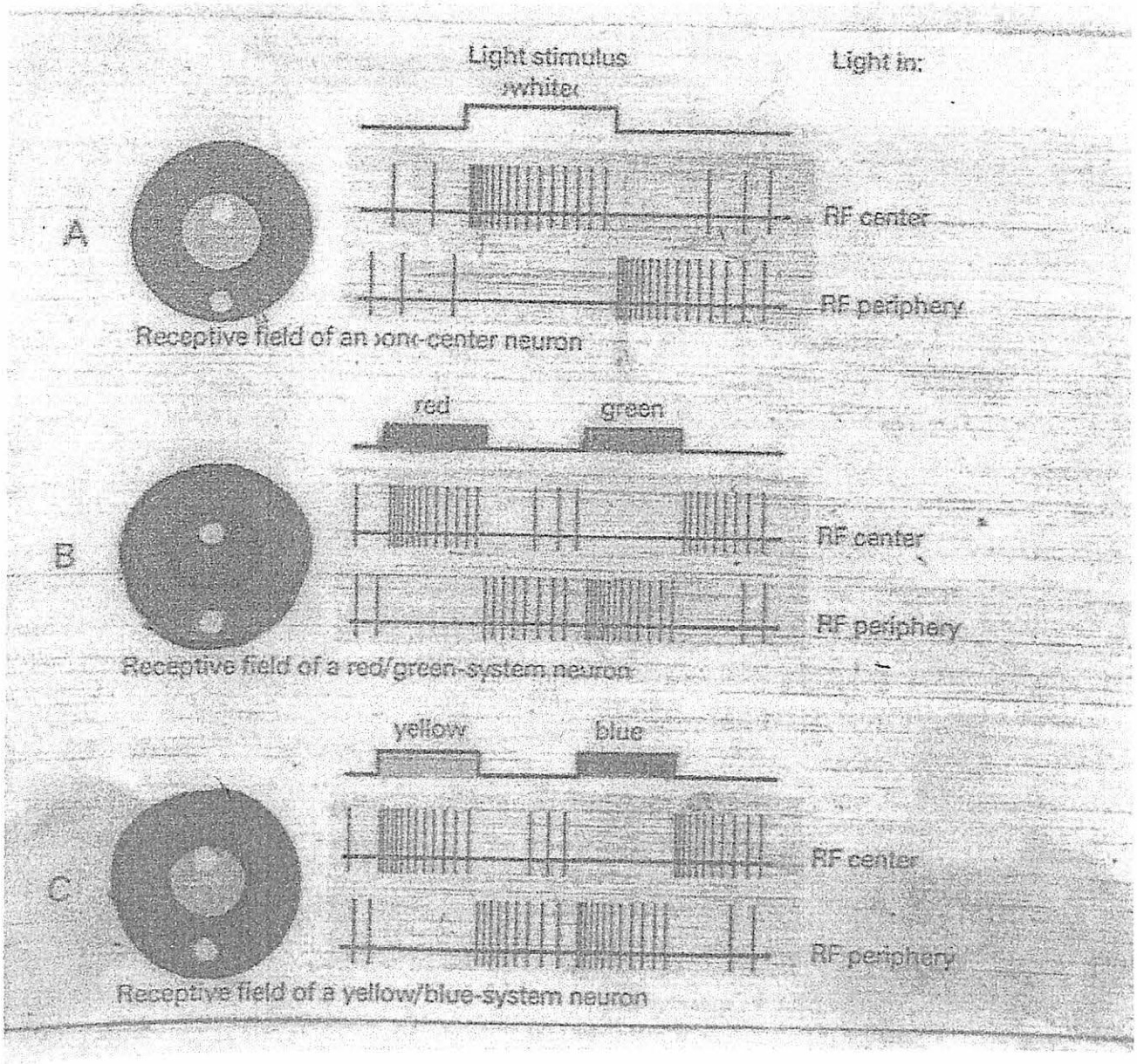


Fig. (A) - (C). Diagram of the spatial organization of three Receptive fields in the ganglion-cell layer of the retina and in the lateral geniculate of a mammal with color vision. (A) nerve cell in the light/dark system; (B) nerve cell in the yellow/blue system. In the color specific receptive fields (B and C) the center and periphery are antagonistically organized. (B) and (C) are highly schematized.