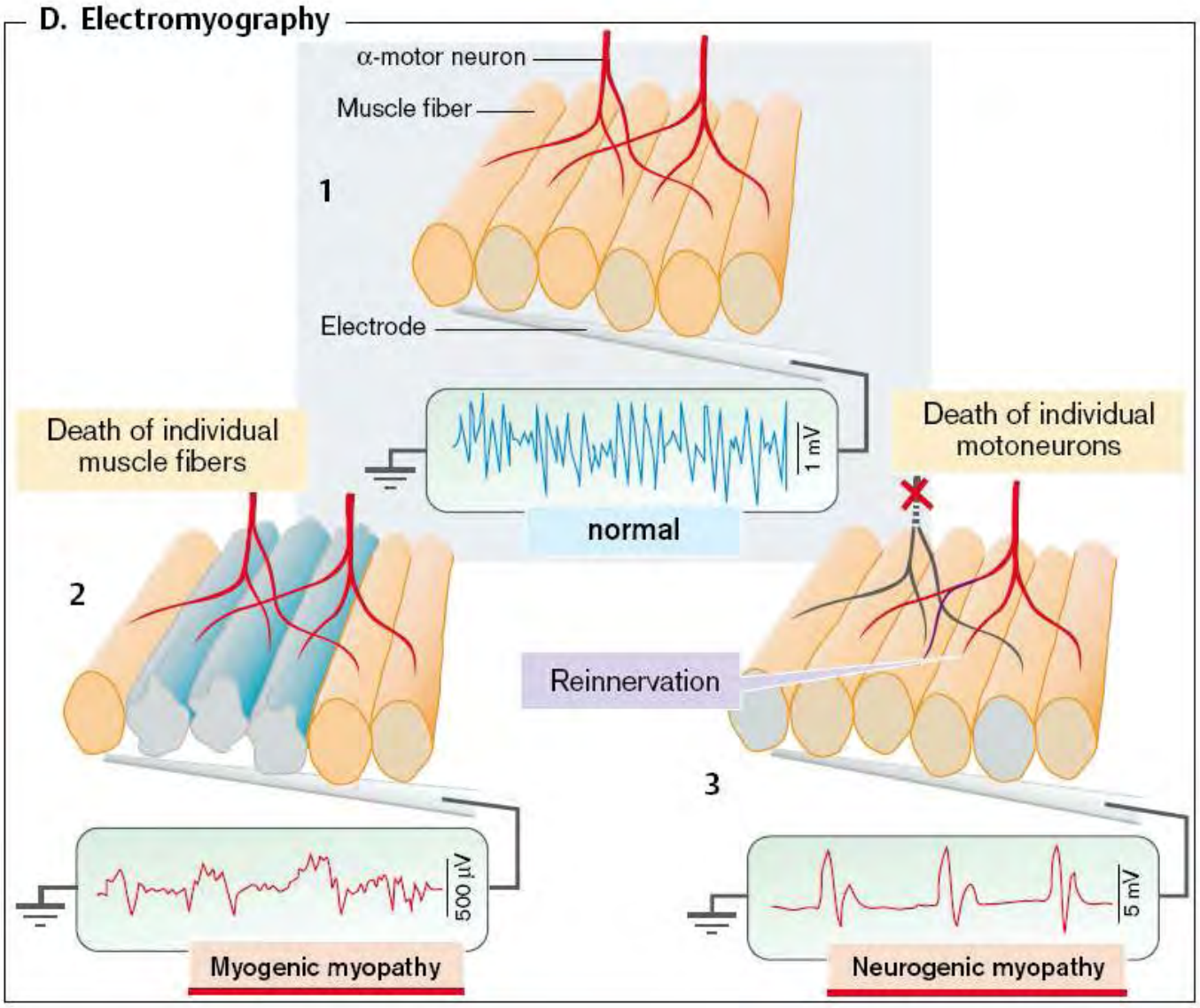
Interactive seminar - neurosciences

Q1/1 EMG – myopathies versus neuropathies

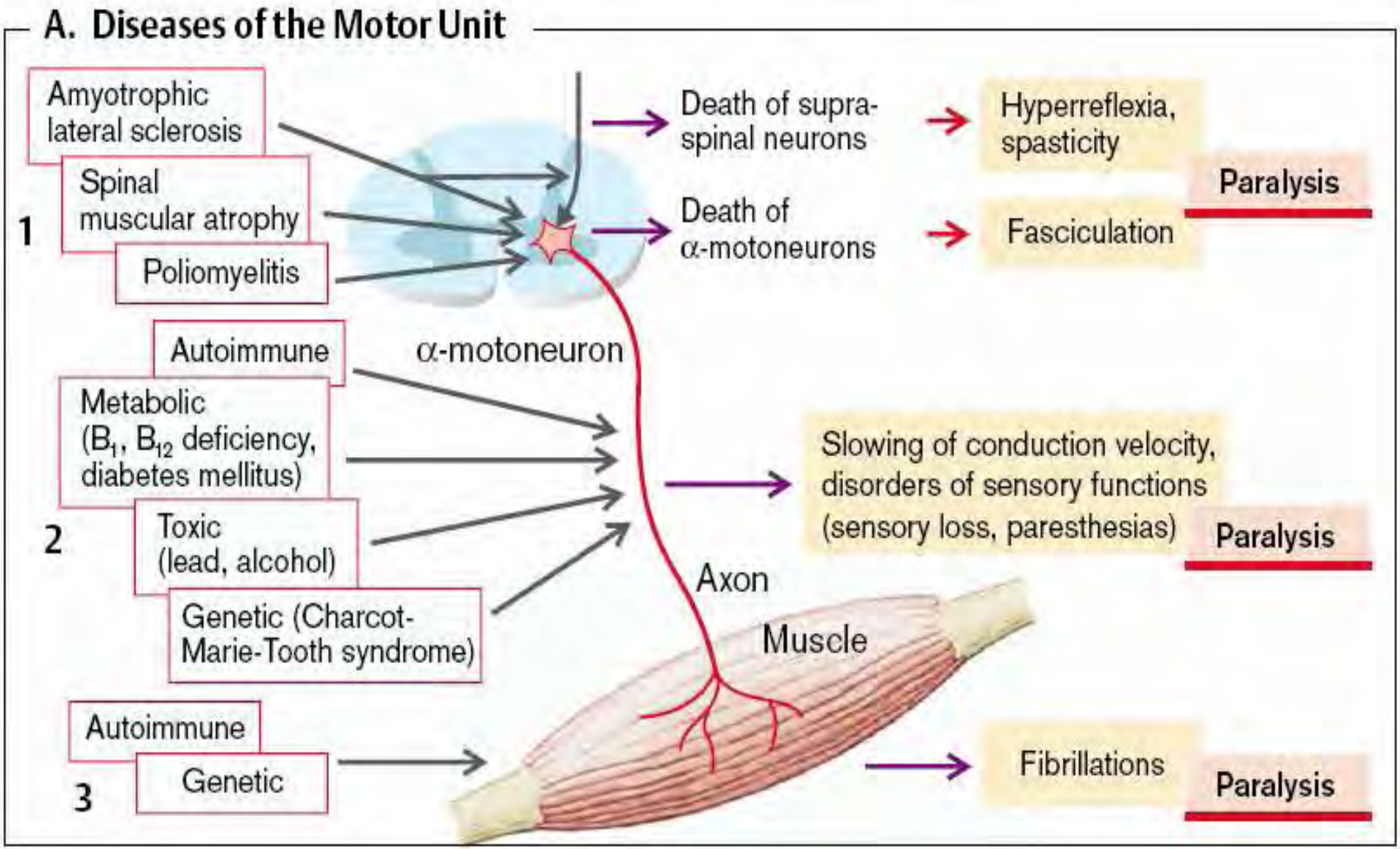


Interactive seminar - neurosciences

Q1/2 Neuropathies versus myopathies

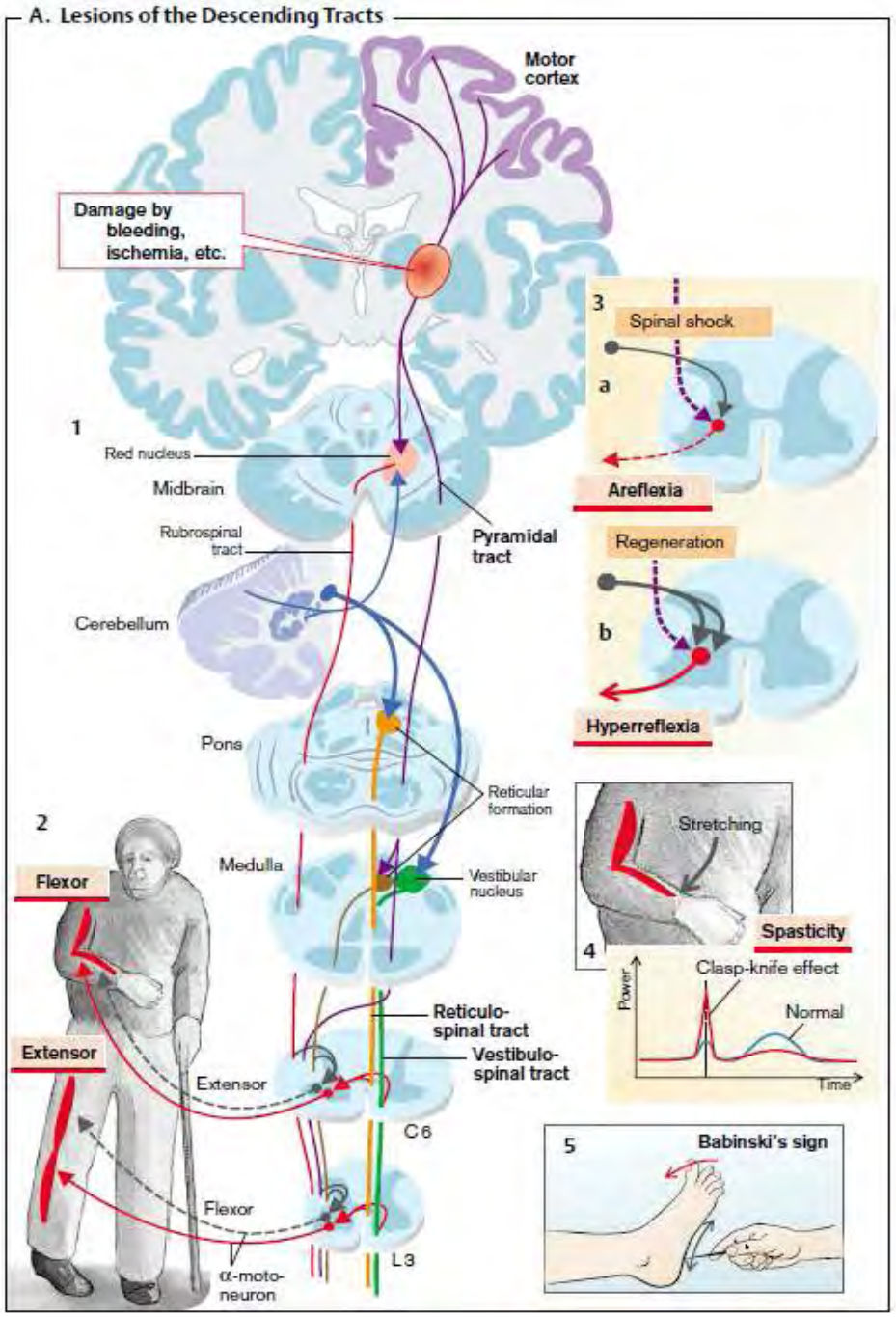
Clinical findings	Neuropathy	Myopathy
Muscle weakness	++	++
Loss of reflexes	+	0
Fasciculations (twitchings)	+	0
Sensory deficit	+	0
Abnormal reflexes (Babinski)	+	0

Q2/1 Lower motoneuron disorders



Q2/2 Upper motoneuron disorders

causes:
brain stroke,
tumor, infection,
demyelinization



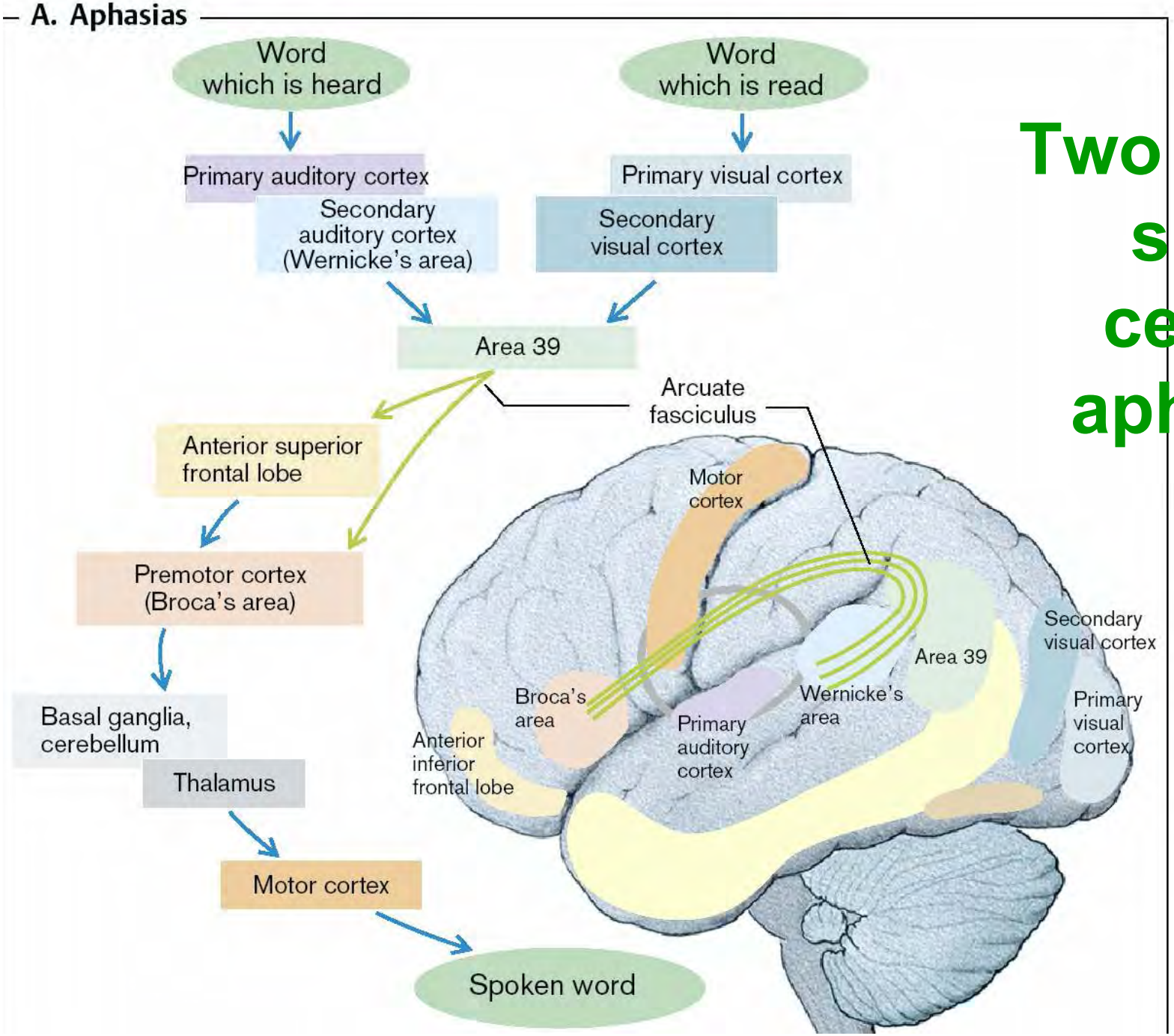
Q 3 Tremor

Tremor types

- Physiological tremor
- Essential tremor
- Parkinsonian tremor
- Orthostatic tremor
- Cerebellar tremor
- Holmes tremor
- Neuropathy-related tremor
- Substance-induced tremor*
- Palatal tremor
- Voice tremor
- Writing tremor
- Psychogenic tremor

* Due to coffee, tea, alcohol, medications (stimulants, neuroleptics, antidepressants, anti-convulsants, cyclosporine A), neurotoxins (heavy metals, insecticides, herbicides, solvents)

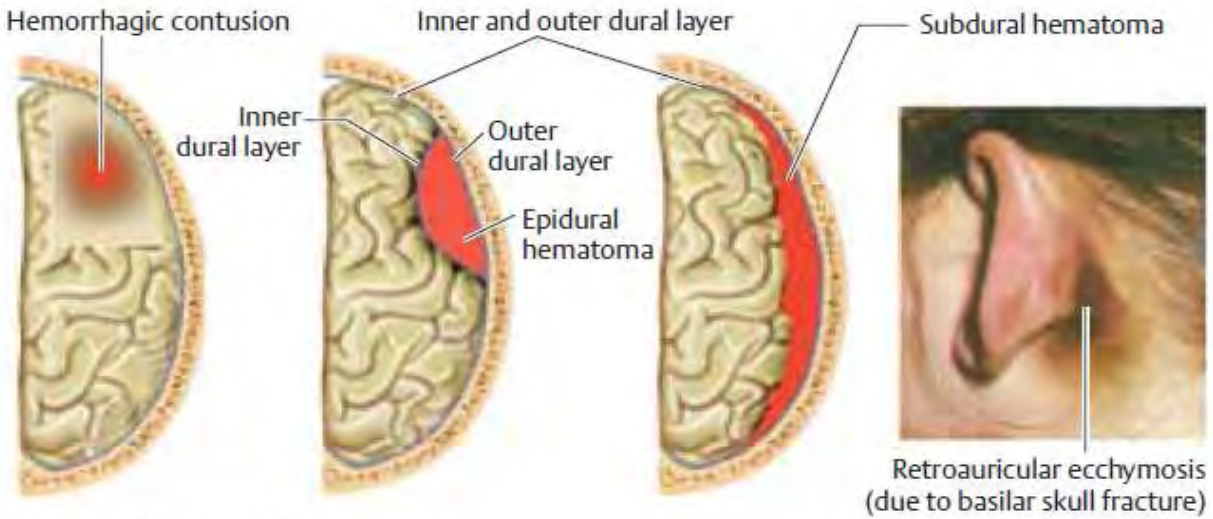
Q 4/1
Two major speech centers, aphasias



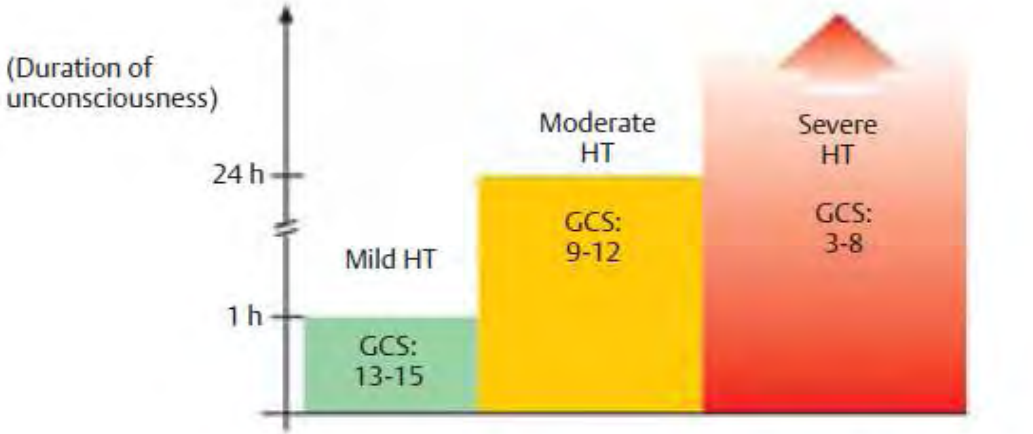
Type	Spontaneous speech	Repetition of words	Language comprehension	Finding words
Broca's aphasia	abnormal	abnormal	normal	impaired
Wernicke's aphasia	fluent (at times logorrhea, paraphasia, neologisms)	abnormal	impaired	impaired
Conduction aphasia	fluent, but paraphasic	markedly impaired	normal	abnormal, paraphasic
Global aphasia	abnormal	abnormal	abnormal	abnormal
Anomic aphasia	fluent	normal, but anomic	normal	impaired
Achromatic aphasia	fluent	normal, but anomic	normal	impaired
Motor transcortical aphasia	abnormal	normal	normal	abnormal
Sensory transcortical aphasia	fluent	fluent	abnormal	abnormal
Subcortical aphasia	fluent	normal	abnormal (transient)	abnormal (transient)

Q 4/2, speech centers, aphasias

Q 5 Intracranial hemorrhage

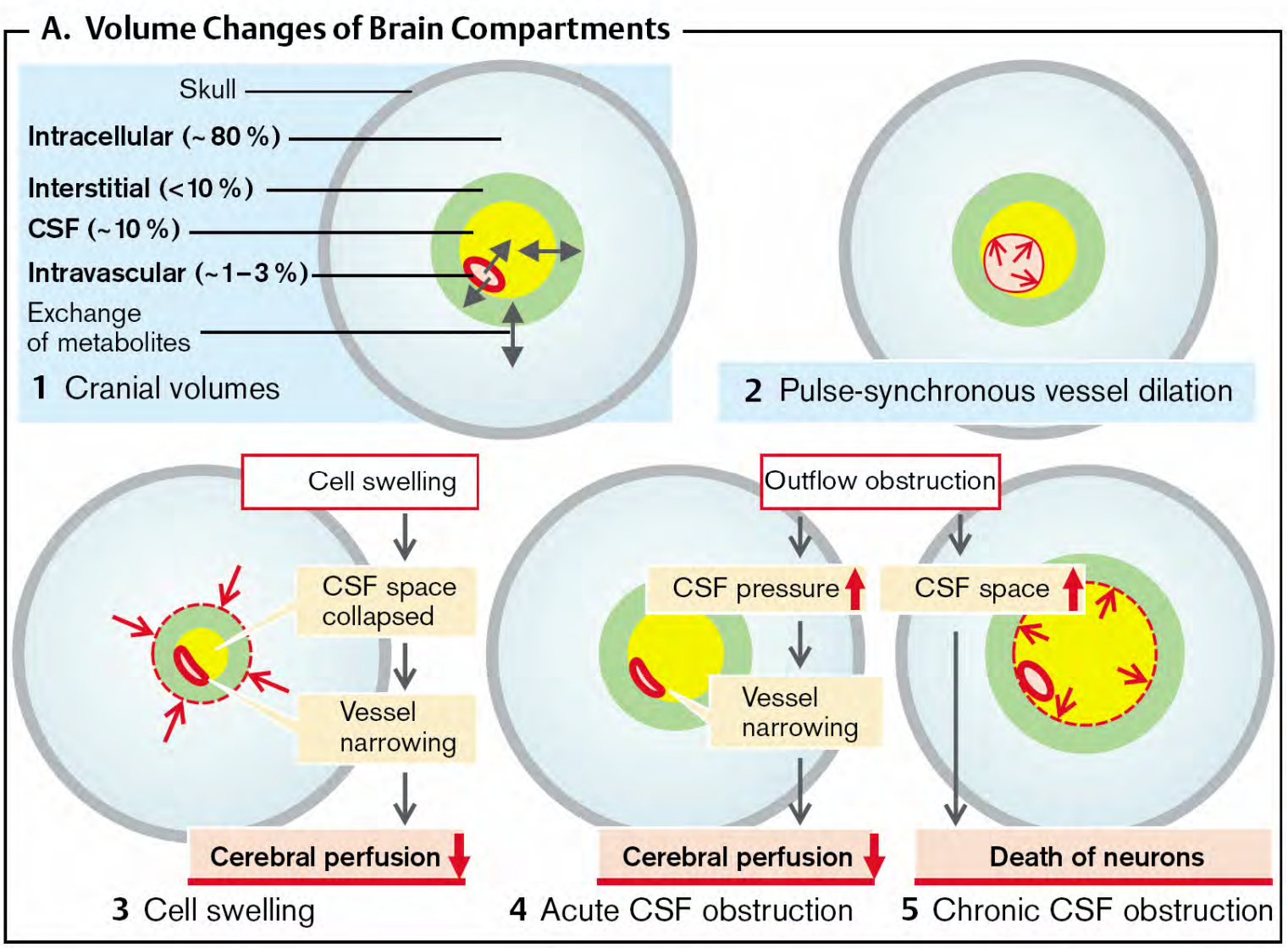


Traumatic intracranial hematoma

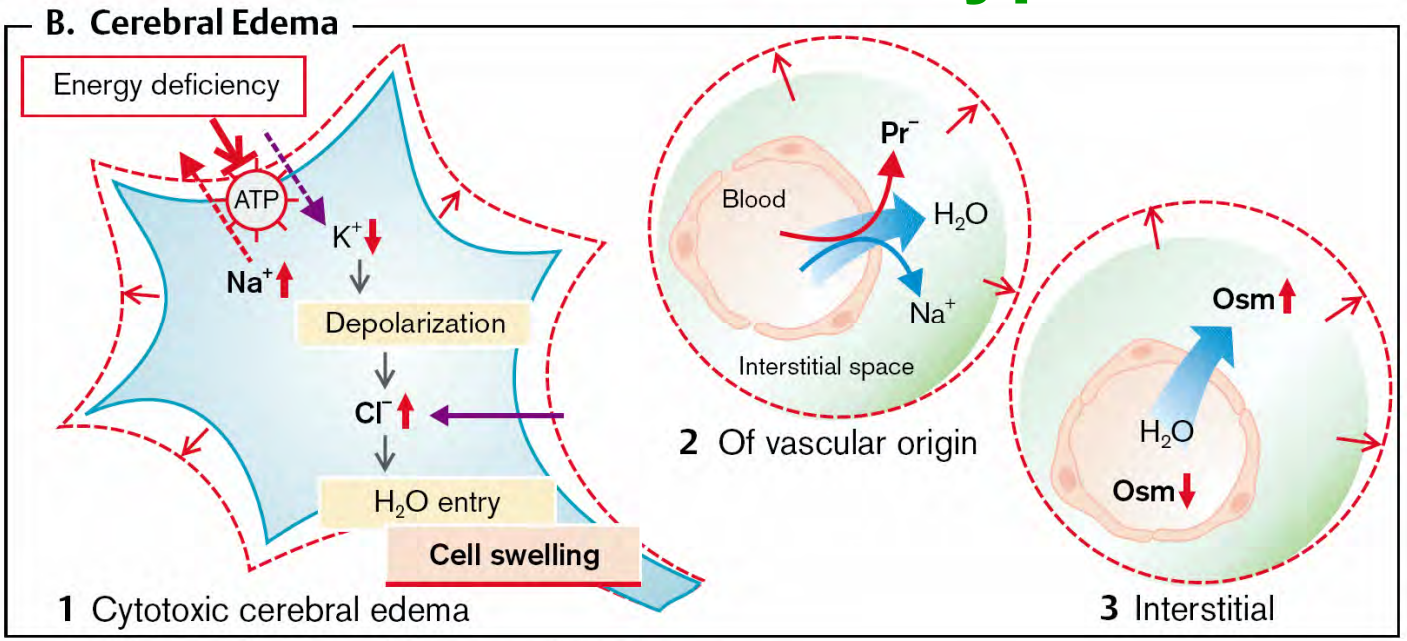


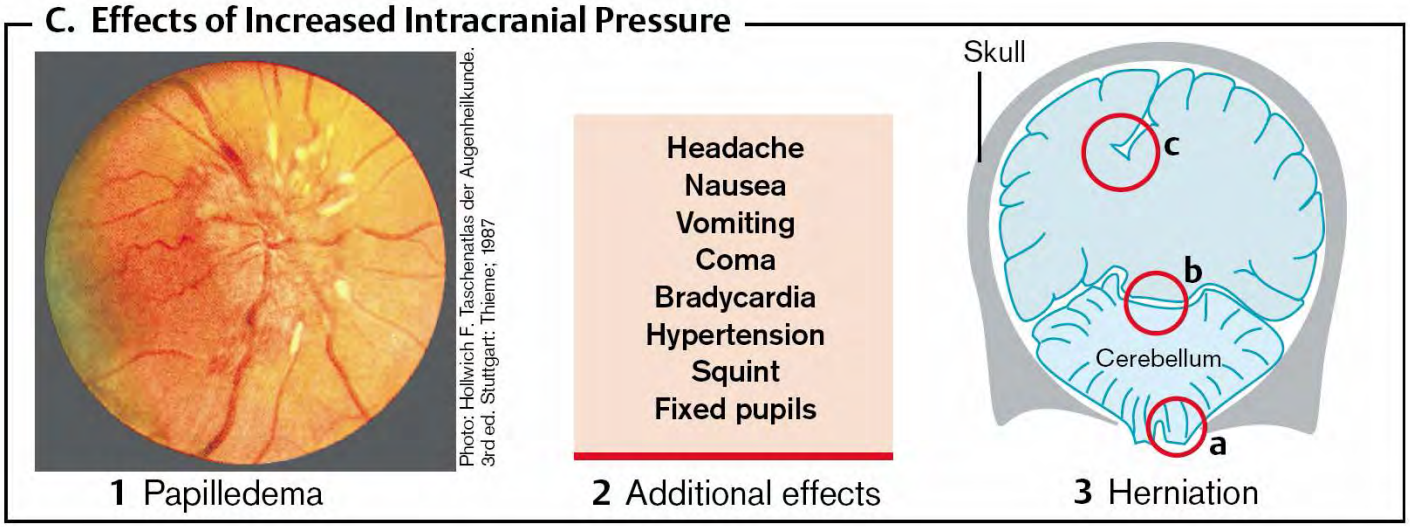
Classification of head trauma (HT) **by Glasgow Coma Scale** (GCS)

Q 6/1 Intracranial hypertension

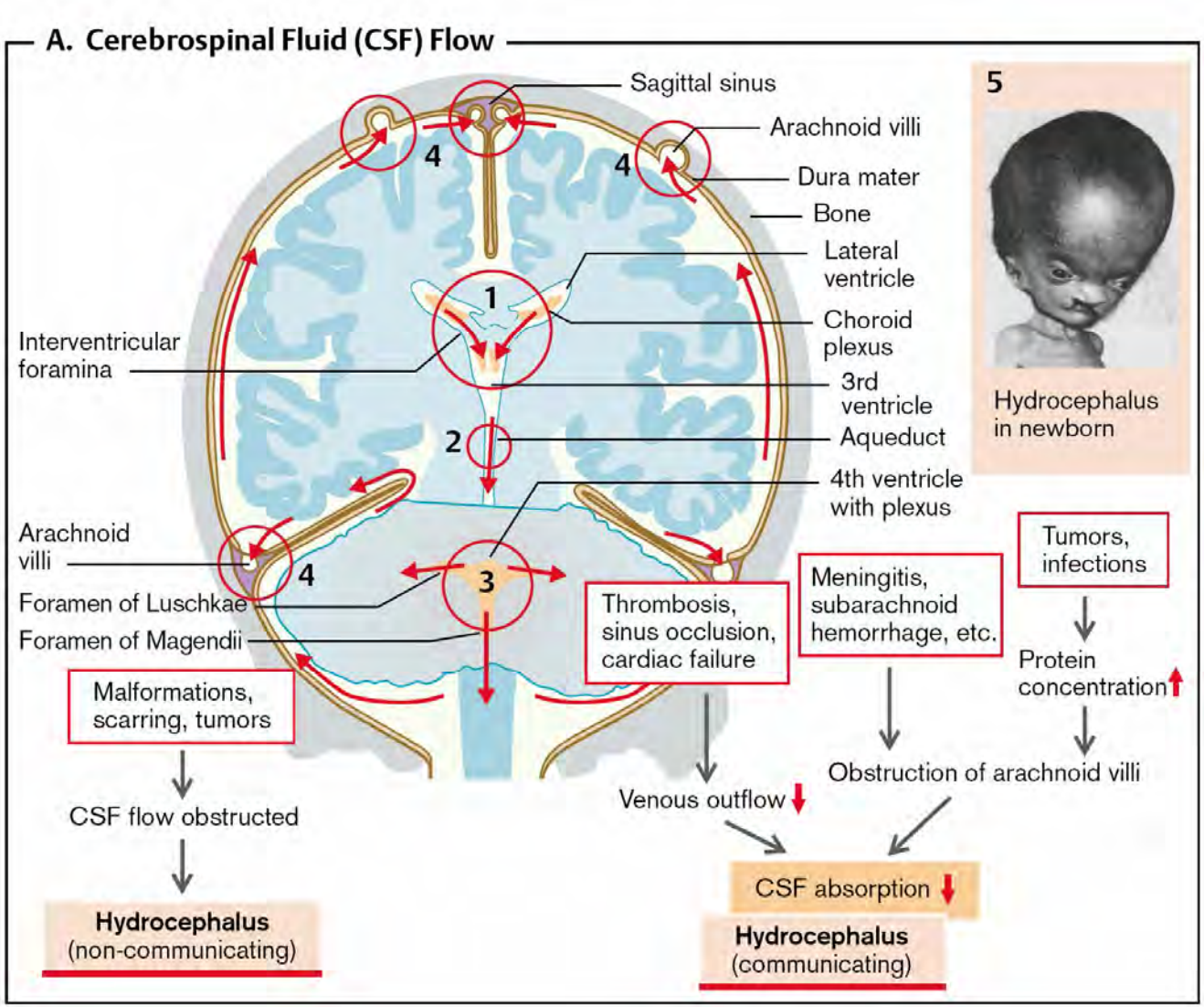


Q 6/2 Intracranial hypertension





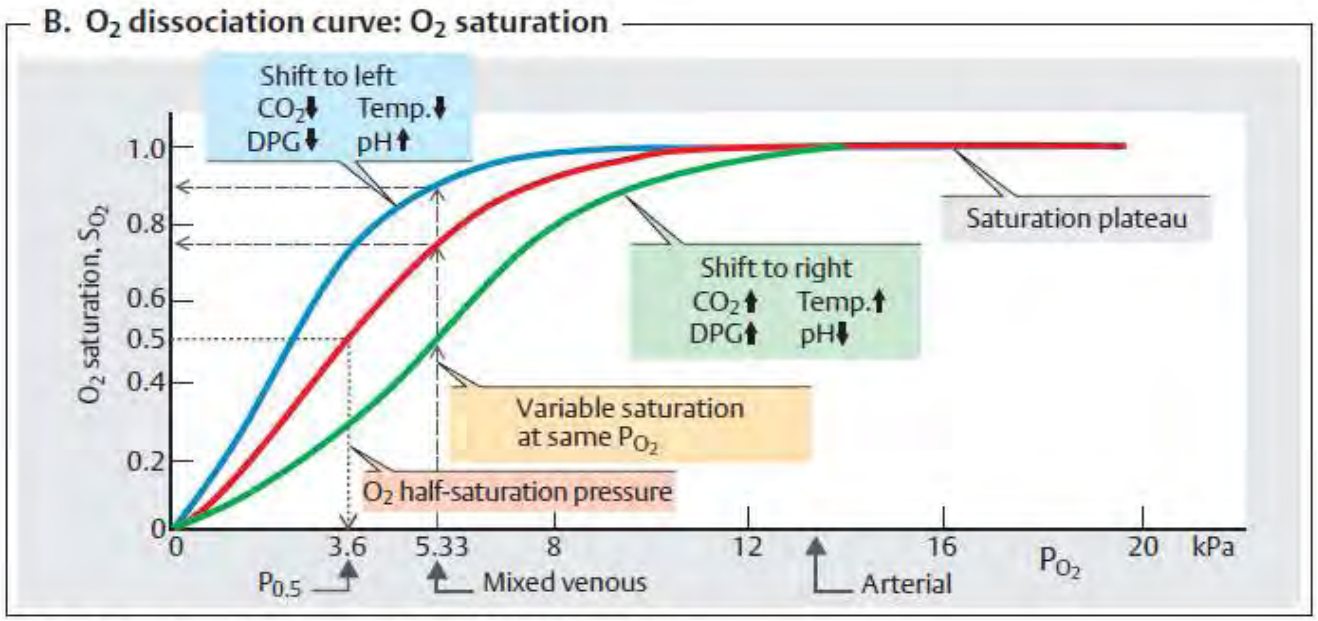
Q 7
Hydro-cephalus

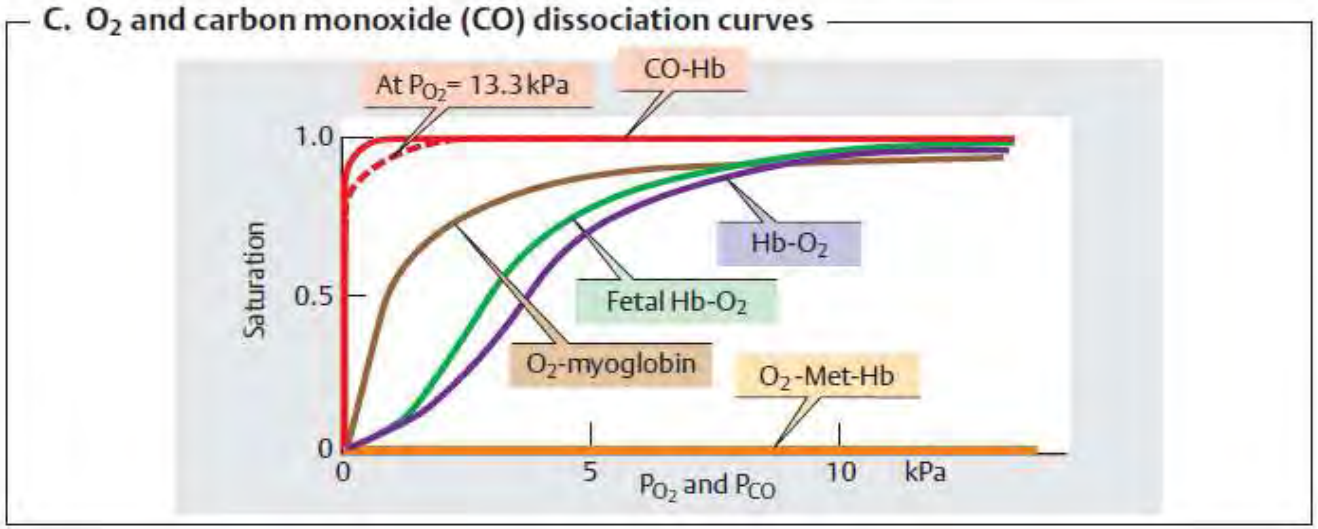


Q 8/1 Acute carbon-monooxide poisoning

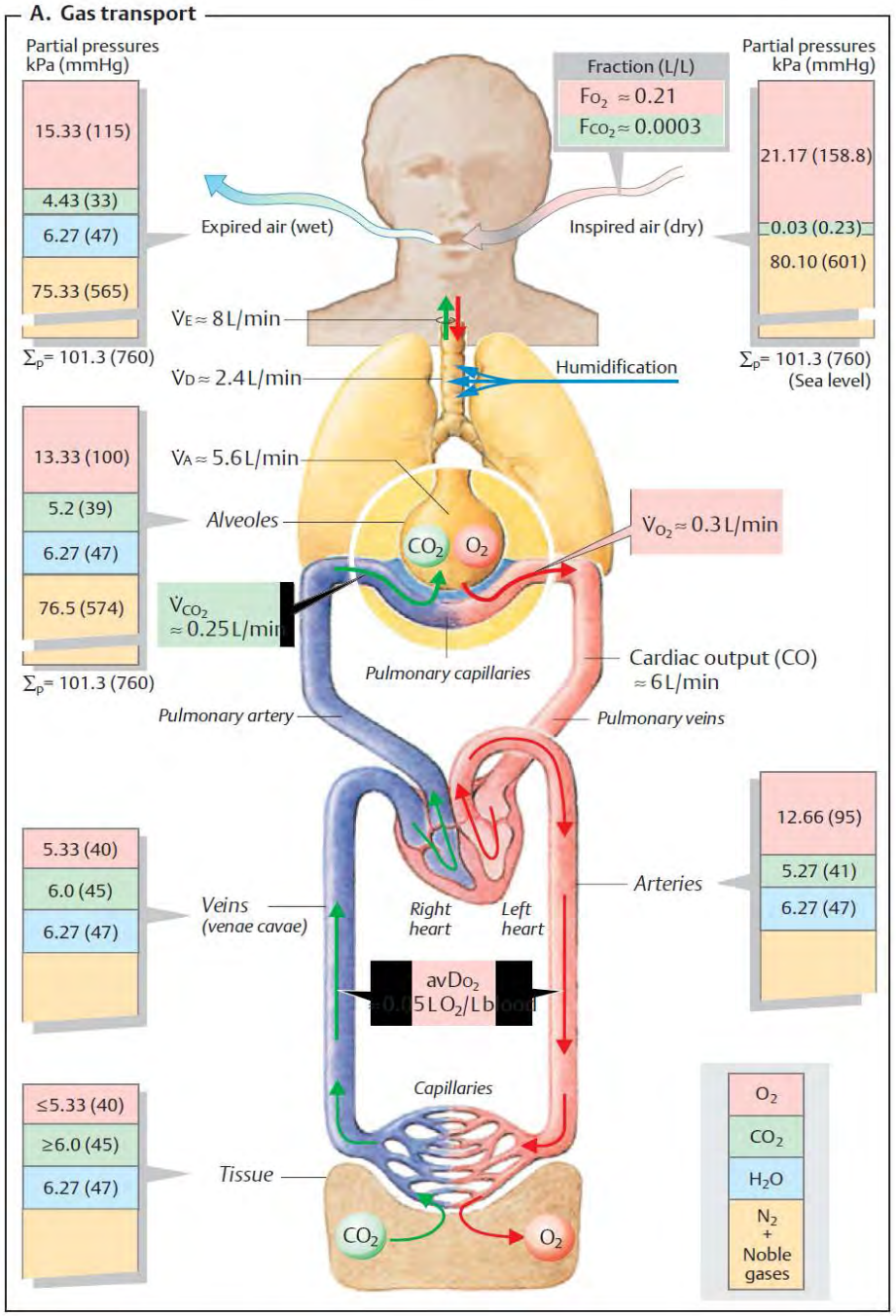
Concentration	Symptoms
35 ppm (0.0035%)	Headache and dizziness within six to eight hours of constant exposure
100 ppm (0.01%)	Slight headache in two to three hours
200 ppm (0.02%)	Slight headache within two to three hours; loss of judgment
400 ppm (0.04%)	Frontal headache within one to two hours
800 ppm (0.08%)	Dizziness, nausea, and convulsions within 45 min; unconsciousness within 2 hours
1,600 ppm (0.16%)	Headache, tachycardia, dizziness, and nausea within 20 min; death in less than 2 hours
3,200 ppm (0.32%)	Headache, dizziness and nausea in five to ten minutes. Death within 30 minutes.
6,400 ppm (0.64%)	Headache and dizziness in one to two minutes. Convulsions, respiratory arrest, and death in less than 20 minutes.
12,800 ppm (1.28%)	Unconsciousness after 2-3 breaths. Death in less than three minutes.

Q 8/2 Acute carbon-monooxide poisoning

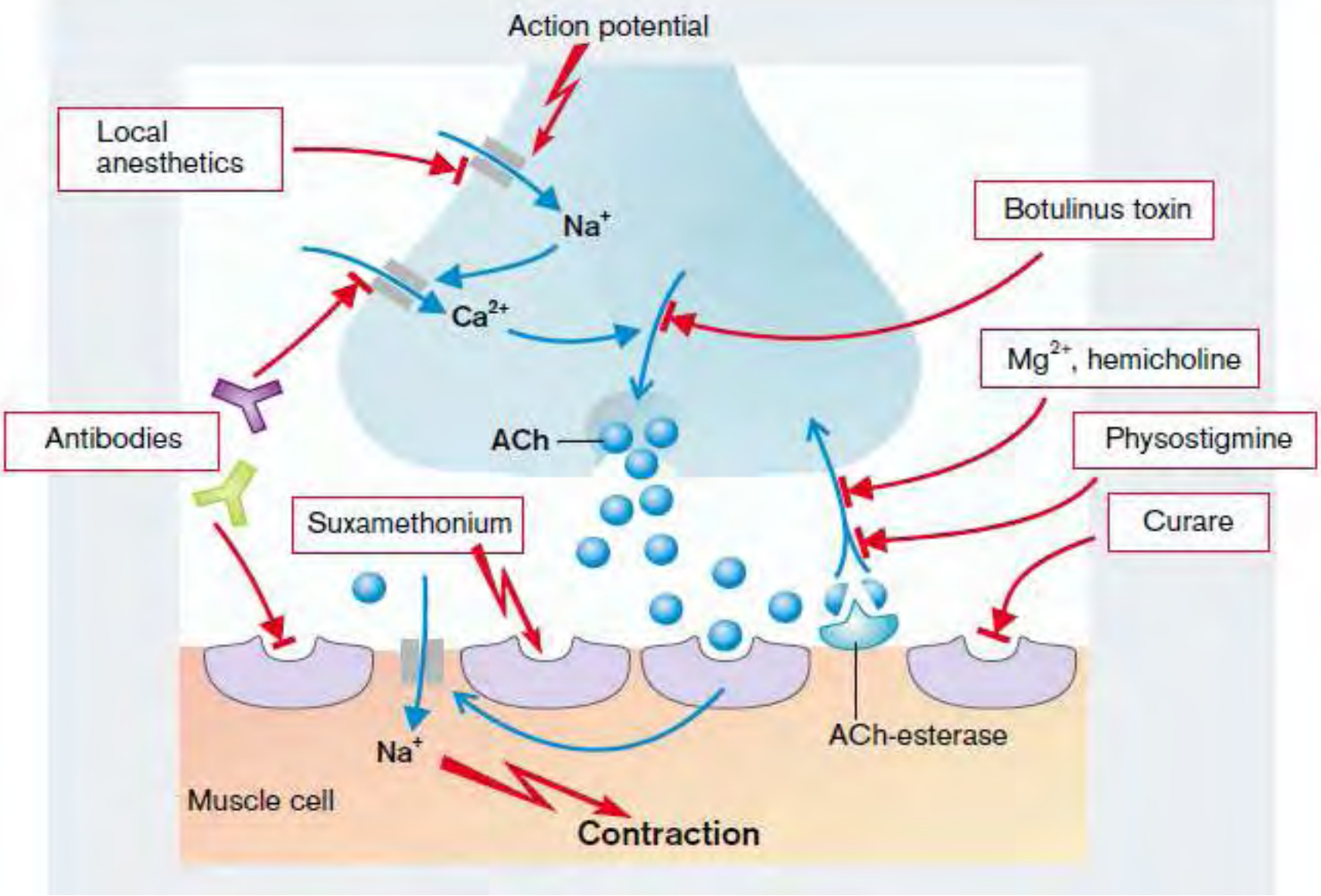




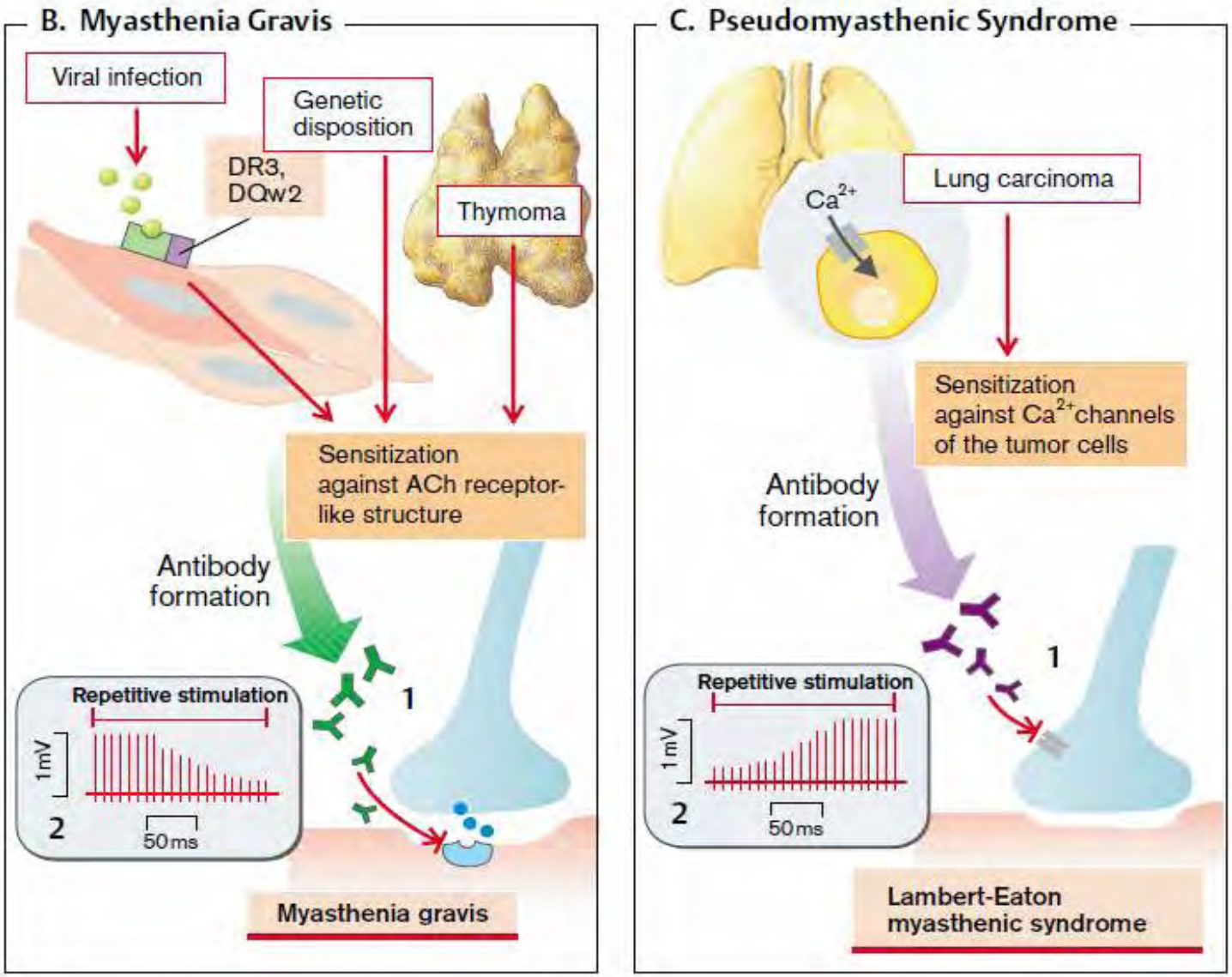
Q 8/3 Blood gases



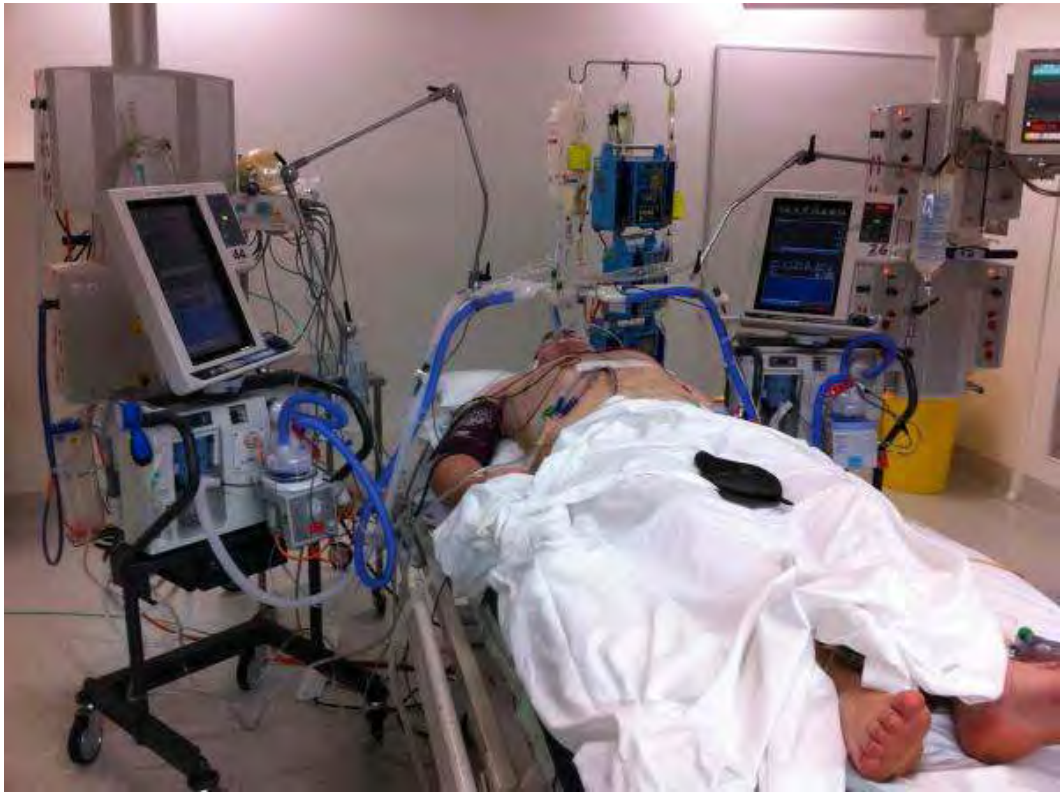
Q 10/1
Disorders of Neuromuscular transmission



Q 10/2
Disorders of Neuromuscular transmission

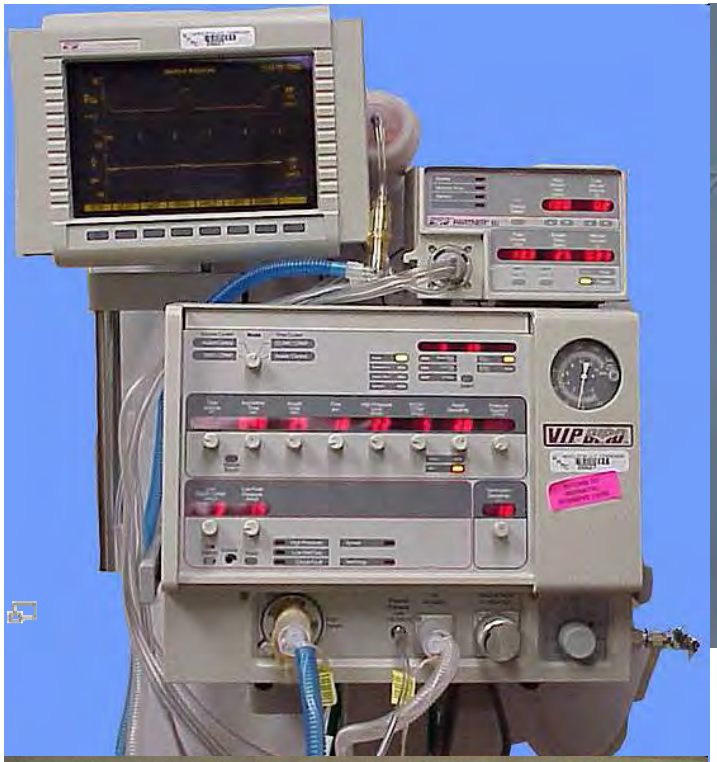


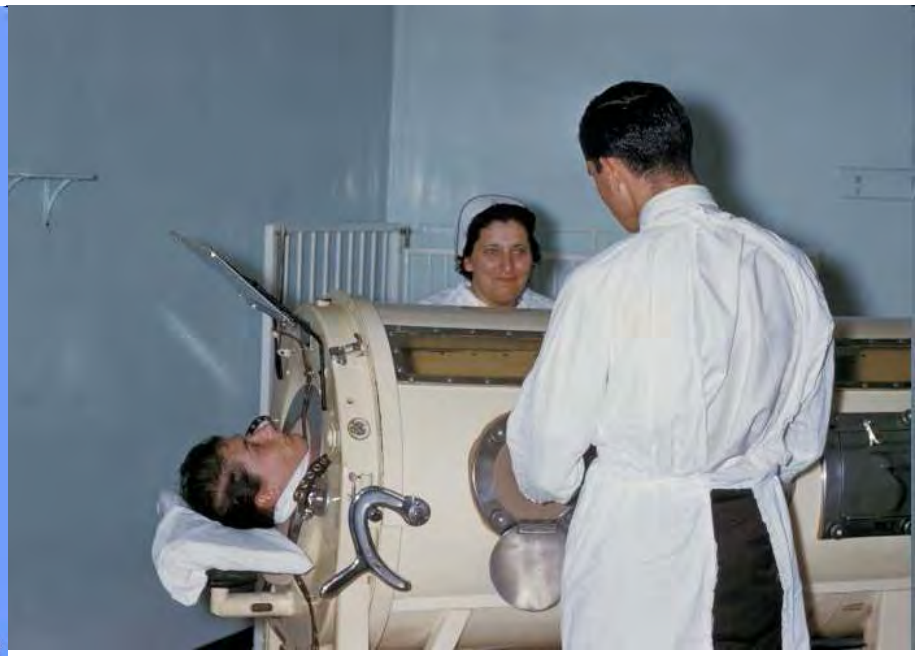
Q 9 poly-radiculo-neuritis Guillain-Barre

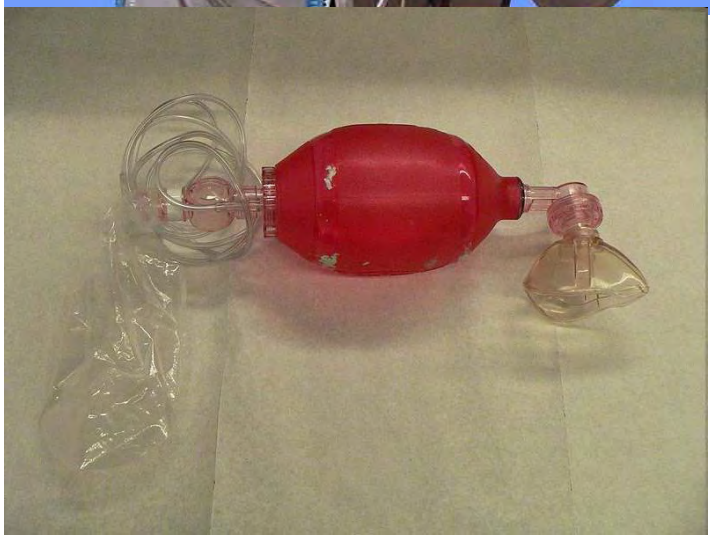


- **acute inflammatory demyelinating polyneuropathy**
- **disorder of the peripheral nerves**
- **attack of the myelin sheath of nerves by antibodies or white blood cells**
- **rapid onset of ascending paralysis**
- **begins with weakness and/or abnormal sensations of the legs and arms**
- **breathing muscles may be so weakened**
- **following gastrointestinal or respiratory viral infections**

Q 11 Artificial ventilation/ Iron lung







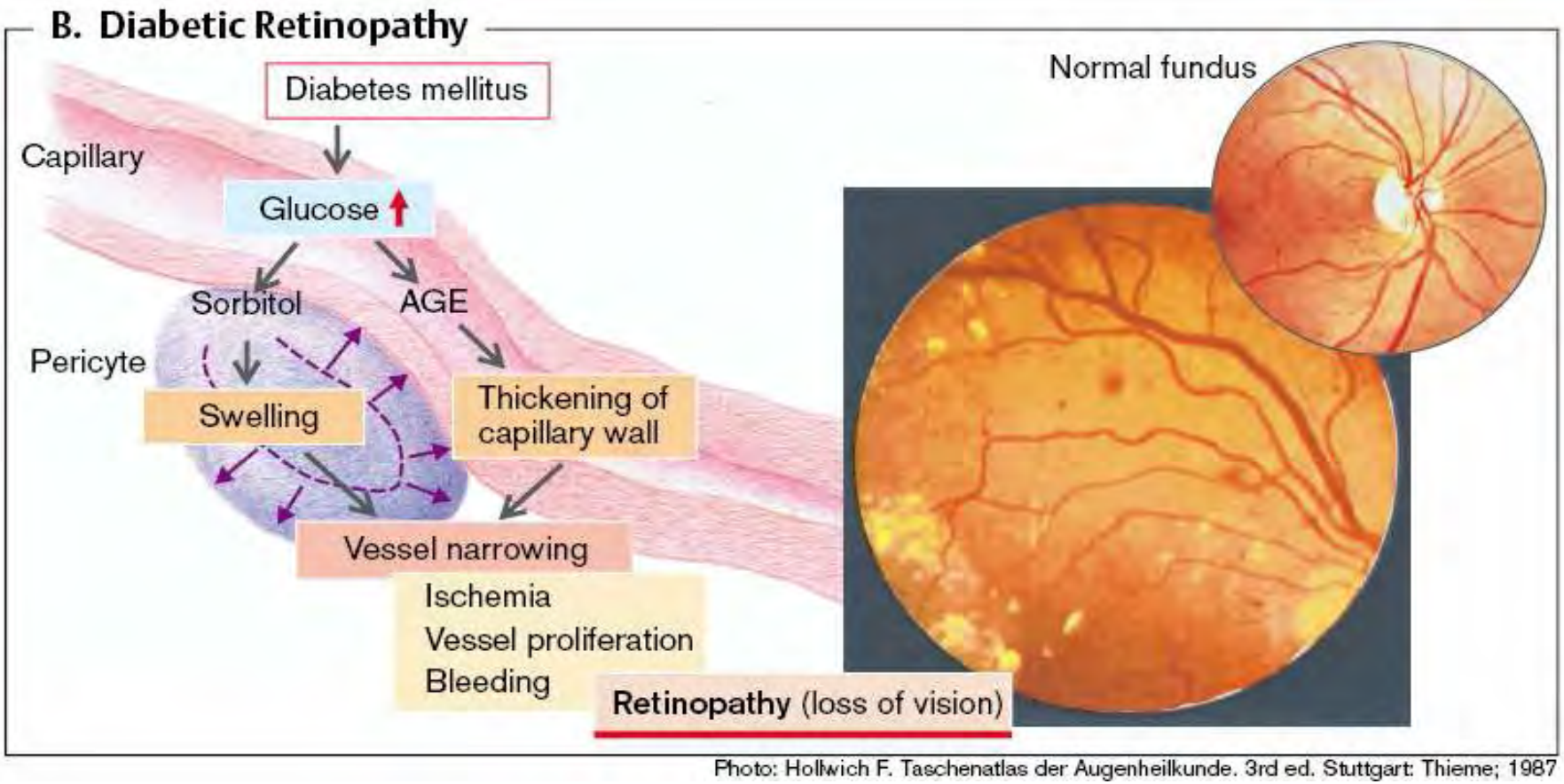
Ventilation can be delivered via:

-Hand-controlled ventilation such as: Bag-Valve-Mask Resuscitator Continuous-flow or Anaesthesia (or T-piece) bag

-A mechanical ventilator.

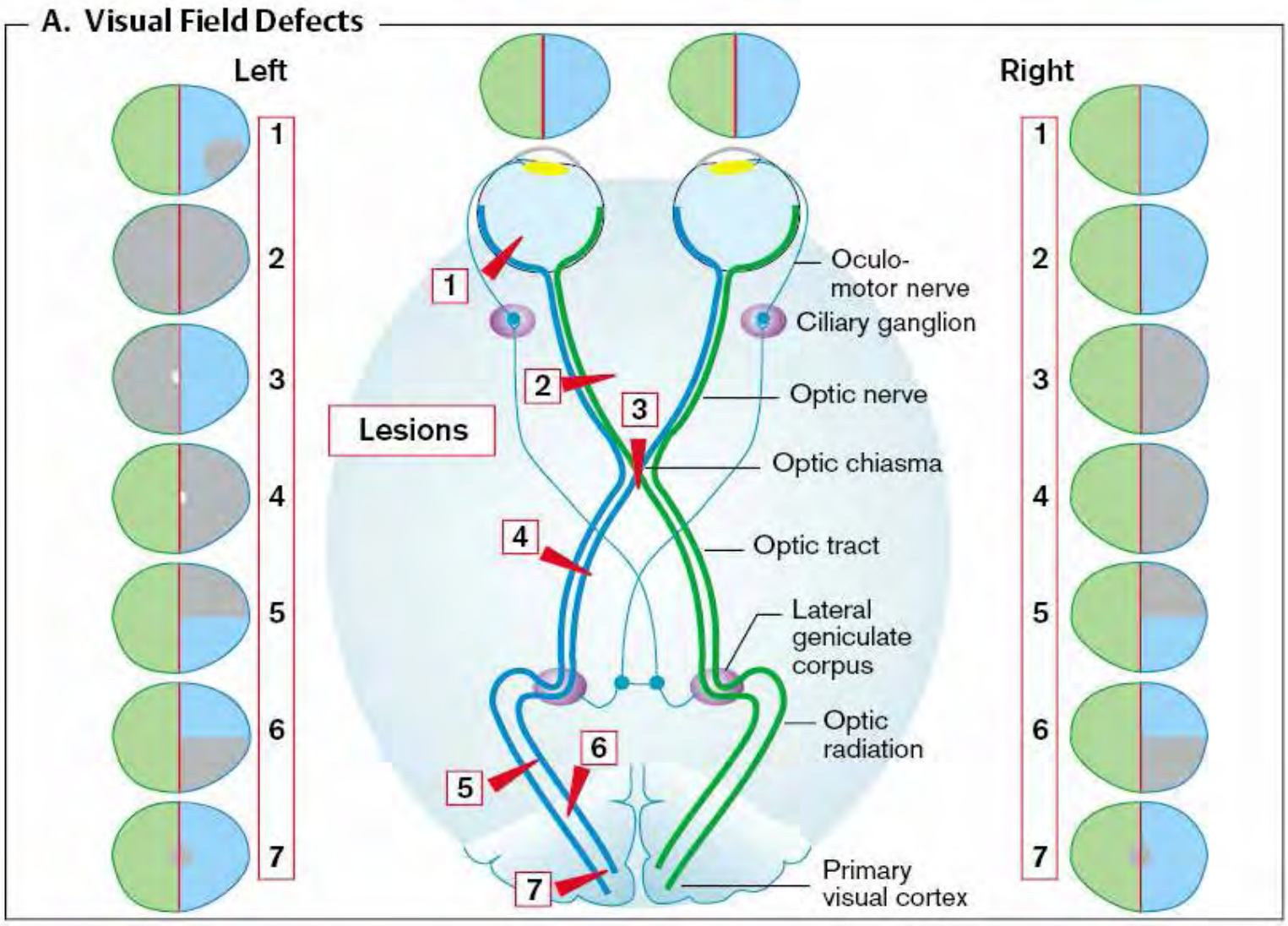
-Iron lung is a historical type of mechanical ventilator

Q12 Diabetic retinopathy



AGE – advanced glycation end products

Q 13/1 Visual field defects - scotomas



Q 13/2
Functional classification of vision impairment

1 normal vision 6/6

2 low vision worse than (<) 6/18

(on the best eye with corrective lenses)

3 (practical) blindness

< 3/60

or narrowing of visual angle less than < 10*10^{o}

other norm < 6/60, < 20*20^{o}

4 *amblyopia*

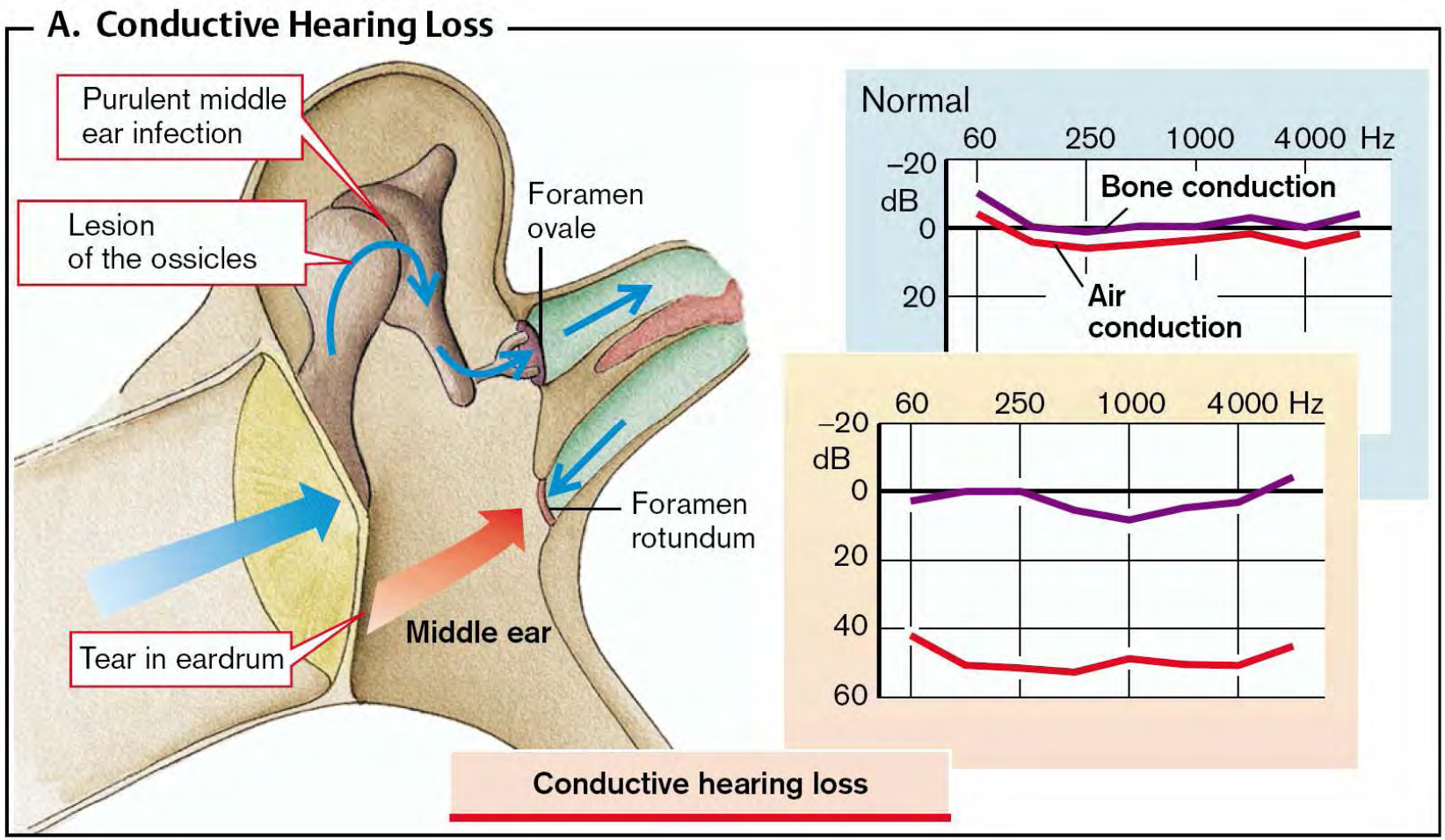
Q 13/3 Causes of blindness

A ordered by frequency in the developed countries:
1 diabetes: retinopathy, 2 glaucoma, 3 senilní poruchy,
4 injuries, 5 others

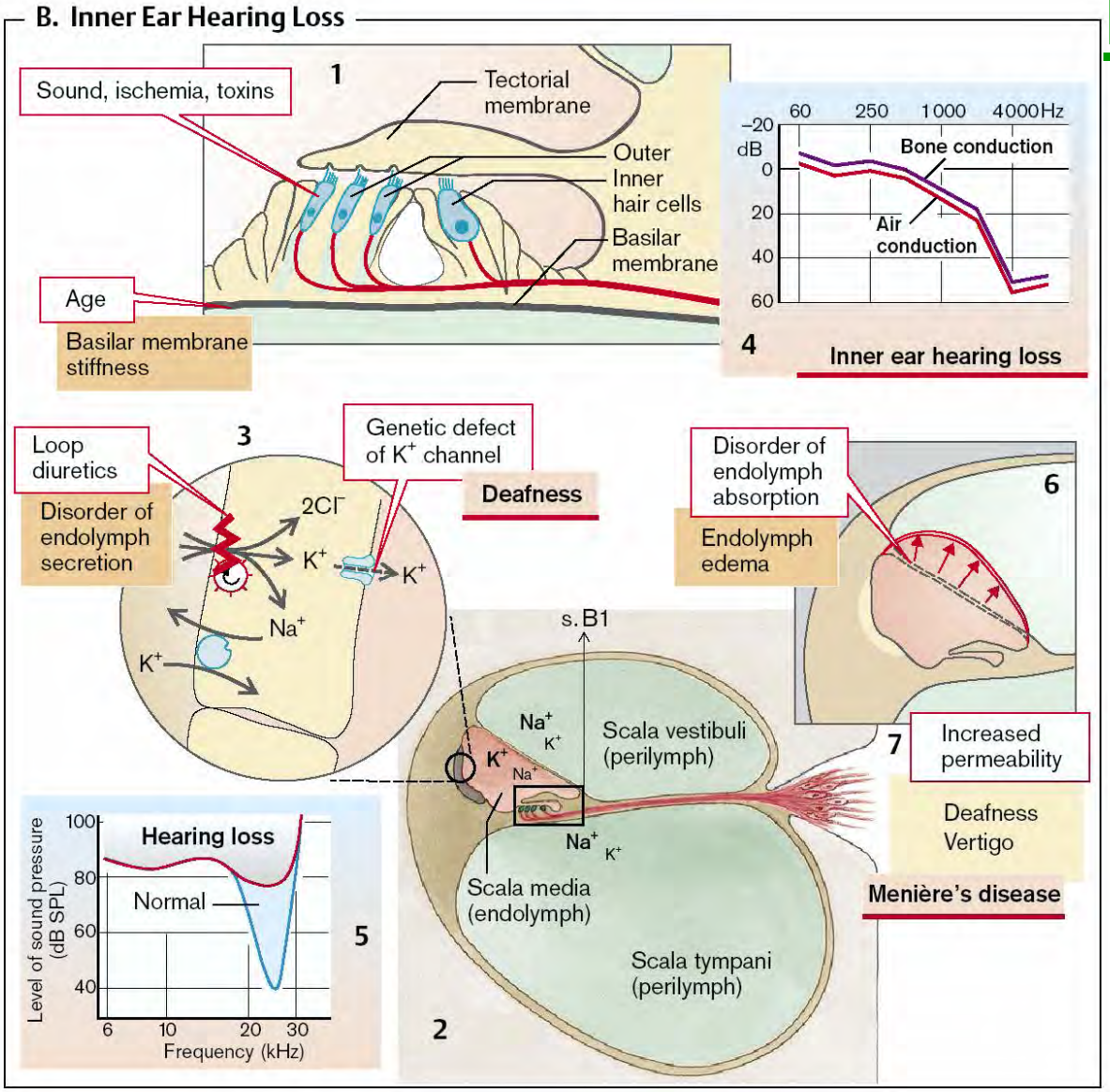
B ordered by frequency in the third world countries:
1 trachoma (chlamydia trachomatis), 2 onchocercosis (onchocerca volvulus),
3 xeroftalmia (vit. A avitaminosis), 4 cataract, 5 glaucoma,
6 injuries, 7 senile macular degeneration, 8 diabetic retinopathy
9 genetic causes, 10 neurologic causes

C overall incidence:
developed countries 0,2 %, worldwide 1 %,
some third world countries several %

Q 14/1 Conduction: through air and bone
Hearing loss: A. conductive, B. sensorineural



Q 14/2 Hearing loss: A. conductive, B. sensorineural



Silbernagl/Lang, Color Atlas of Pathophysiology © 2000 Thieme

Q 14/3 Pitchfork tests

Test	Principle	Norm	Conductive	Sensory-neural
Weber	PF on the vertex of the head	Non-lateral	Lateral to blocked side	Lateral to healthy side
Rinne	First on bone, then in the air	Positive	Indifferent	Positive
Schwabach	(subjective) Patient compared to examiner	Normal	Longer	Shorter

Q 14/4
Functional classification of hearing loss

(measured without hearing aid)

1 normal hearing (threshold about 4 phon)

2 hardness of hearing

(hearing aid may be indicated:
at the band 500 Hz - 2 kHz bilaterally
threshold rise of 35 - 40 dB,
speech audiometry –threshold rise of more than 35 dB
low comprehension of loud speech at less than 4 m)

3 (practical) deafness

(does not hear loud voice at the ear, own voice,
threshold rise of 75 - 80 dB)

4 *deaf-and-dumbness*

(speech was not rehabilitated after inborn deafness)

Q 14/5 Causes of hearing loss

- otosclerosis (in 0,5 - 1 % of elderly)
- conductive disorders
- hereditary and inborn disorders
- toxic damage
- meningoencefalitis
- profesional damage
- presbyakusia
- Menier’s disease

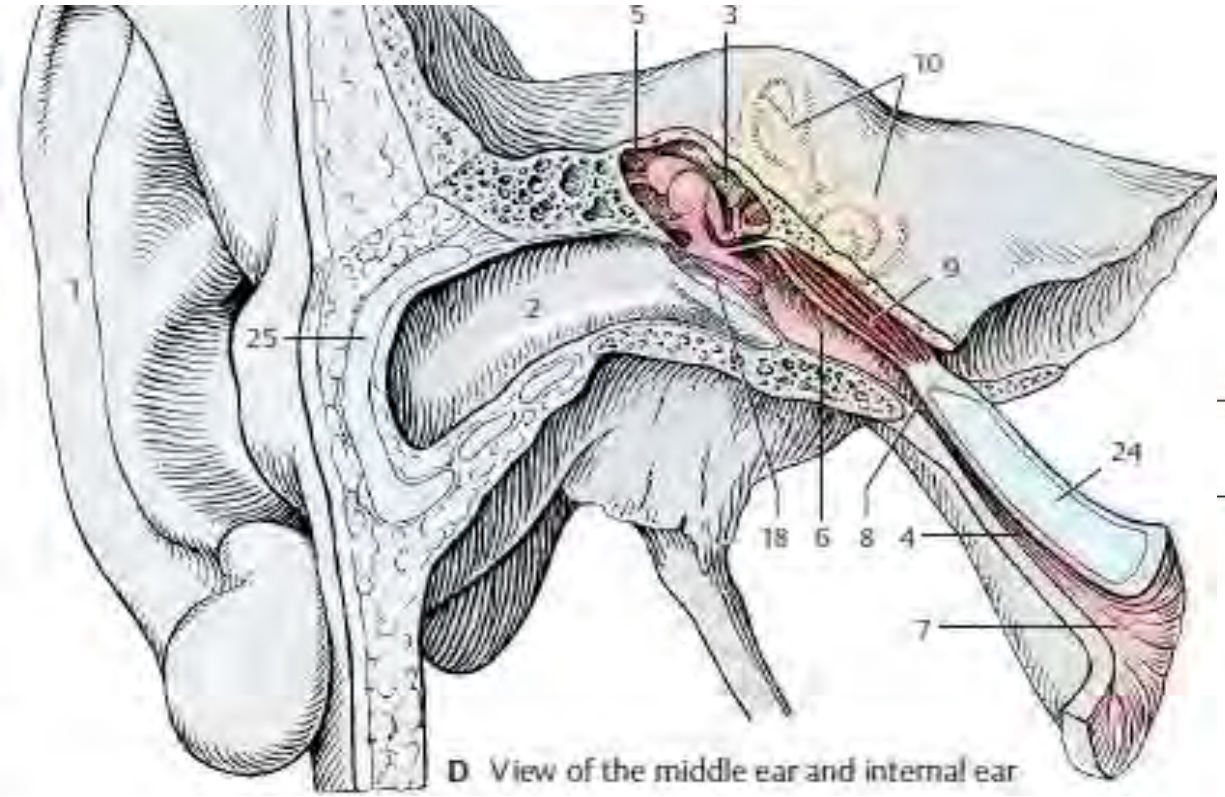
Q 15/1 Acoustic neurinoma

Acoustic neurinoma

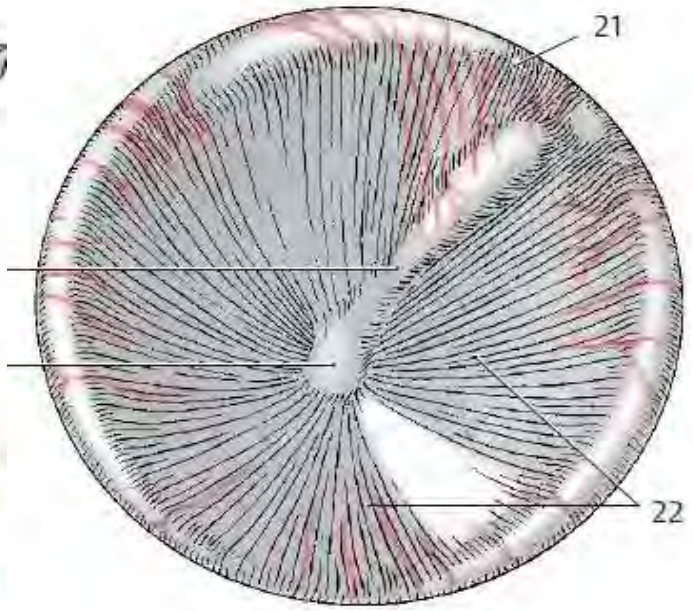
Represents about 8% of all intracranial tumors. It arises from the Schwann cells which invest the eighth nerve as it enters the internal auditory canal. 95% of these lesions originate within the auditory canal, and the other 5% arise from the nerve at its cerebellopontine angle course, proximal to the canal. Often bilateral in neurofibromatosis. Most acoustic neuromas arise from the superior vestibular branch of the eighth cranial nerve. The most noticeable radiographic change caused by these tumors is erosion of the superior and posterior lips of the porus acusticus.

Peritumoral edema can be seen in 30–35% of cases with larger lesions, and less frequently calcification, cystic change, and hemorrhage

Q 16 Acute otitis media



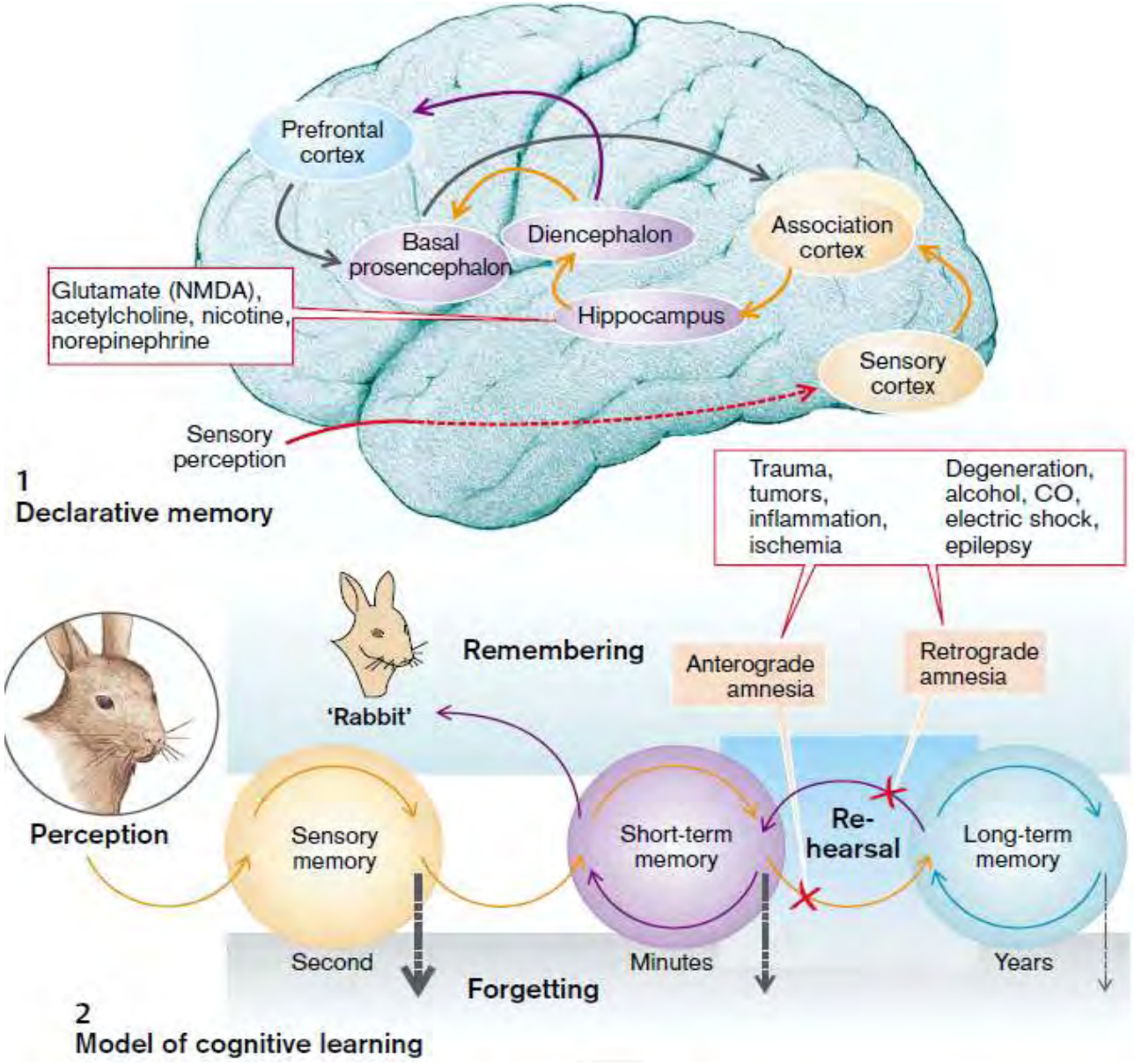
D View of the middle ear and internal ear



B Right eardrum

Children younger than seven are much more prone to otitis media due to shorter Eustachian tubes, which are at a more horizontal angle than in the adult ear. They also have not developed the same resistance to viruses and bacteria as adults.

Q 17 Memory



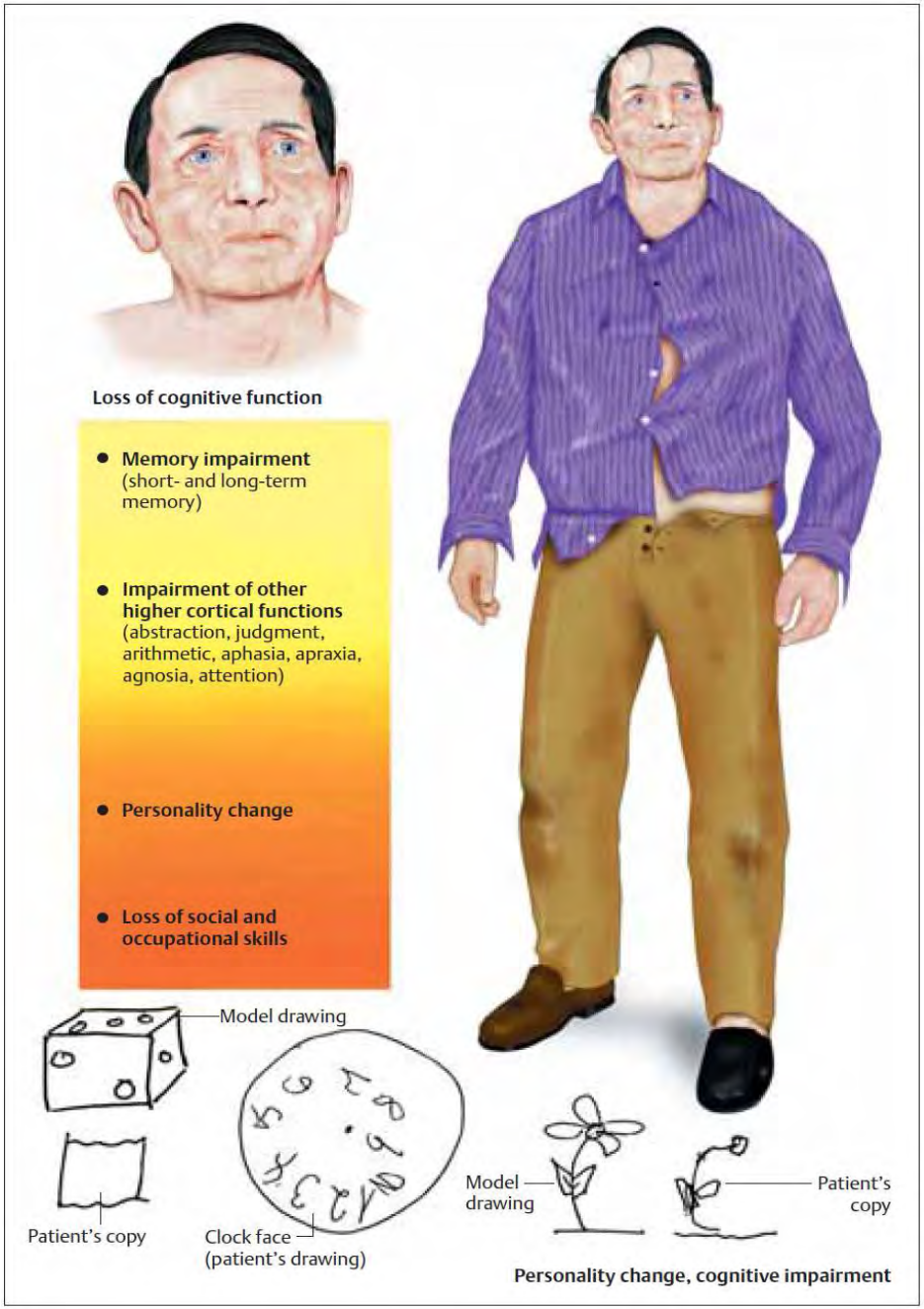
Memory
Short term
Mid-term
Long term

Amnesia
Anterograde
Retrograde

Disorders
(examples only)
Acute
-posttraumatic
Chronic:
-Alzheimer's disease
-Korsakoff syndrome

Q 18 Memory

Dementia/ Alzheimer's disease



Q 19 Lobotomy

The Nobel Prize in Physiology or Medicine 1949
Walter Hess, Egas Moniz

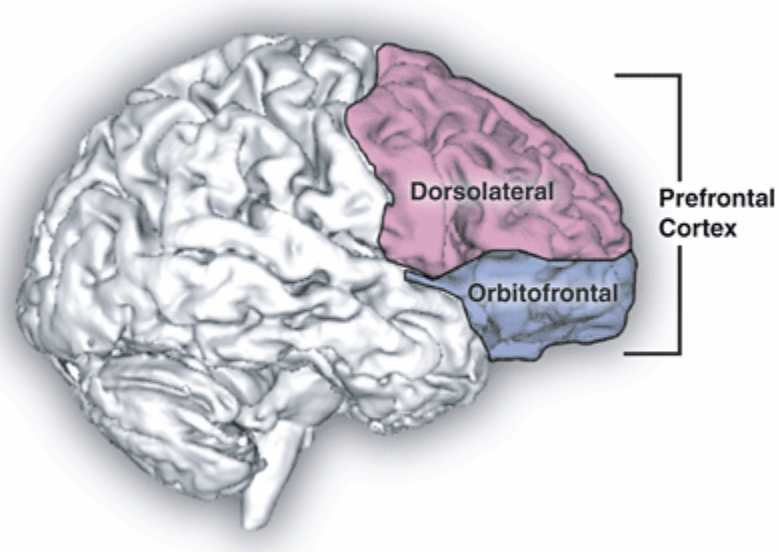
The Nobel Prize in Physiology or Medicine 1949

Walter Hess

Egas Moniz

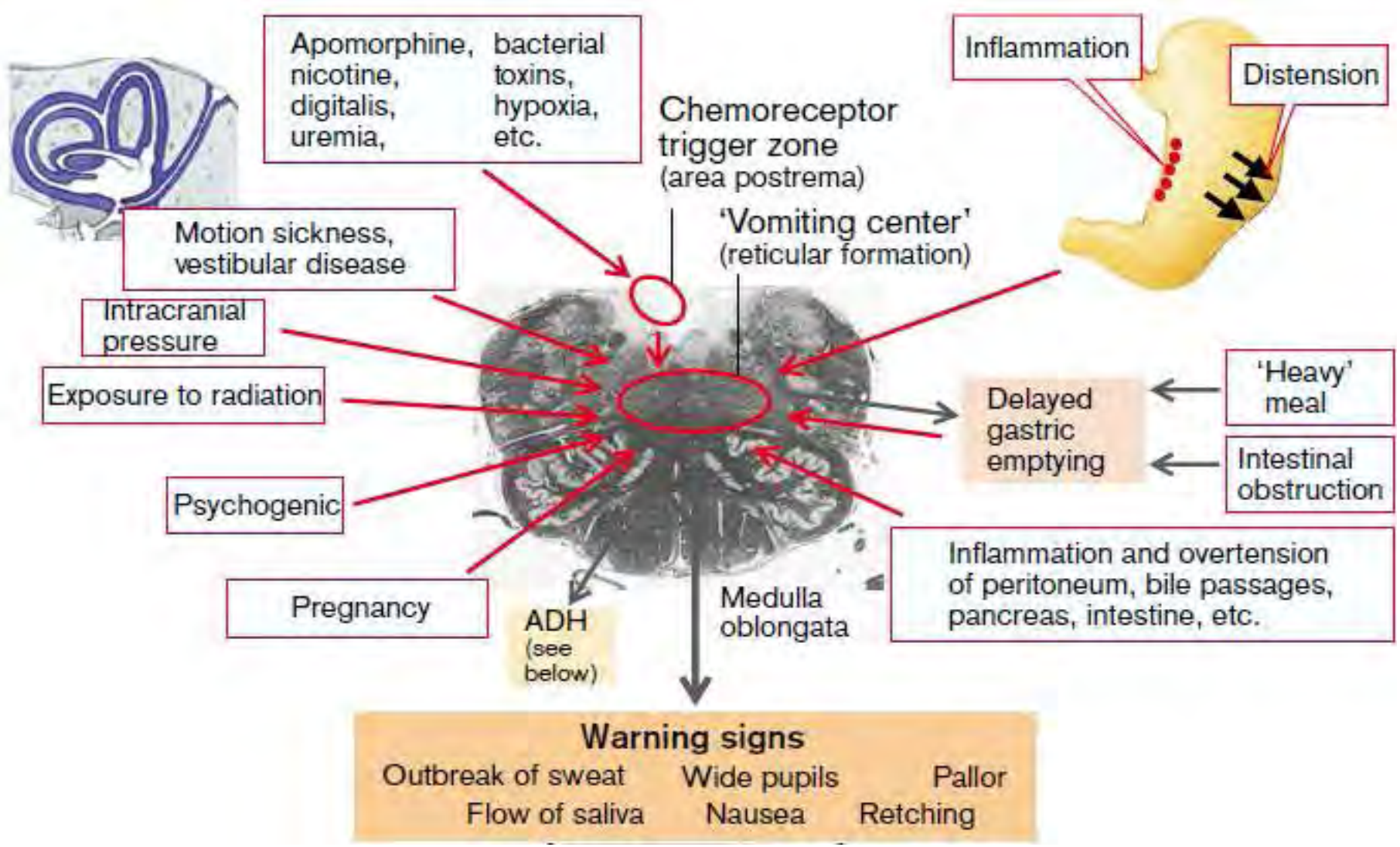
Walter Rudolf Hess

Antonio Caetano de Abreu Freire Egas Moniz



The Nobel Prize in Physiology or Medicine 1949 was divided equally between Walter Rudolf Hess *"for his discovery of the functional organization of the interbrain as a coordinator of the activities of the internal organs"* and Antonio Caetano de Abreu Freire Egas Moniz *"for his discovery of the therapeutic value of leucotomy in certain psychoses"*.

Q 20/1 Vomiting center/ causes of vomiting



Q 20/2 Causes of vomiting

1 Intracranial hypertension - irritation

2 Drugs – nicotine, apomorphine, etc

3 Kinetosis

4 Radiation diesease

5 Pregnancy

6 Psychogenic

7 Pharyngeal irritation

8 Local gastric irritation – food poisoning

9 Peritoneal irritation, ileus

10 Other internal organs – heart etc

Q 21/1 Migraine - phosphenes

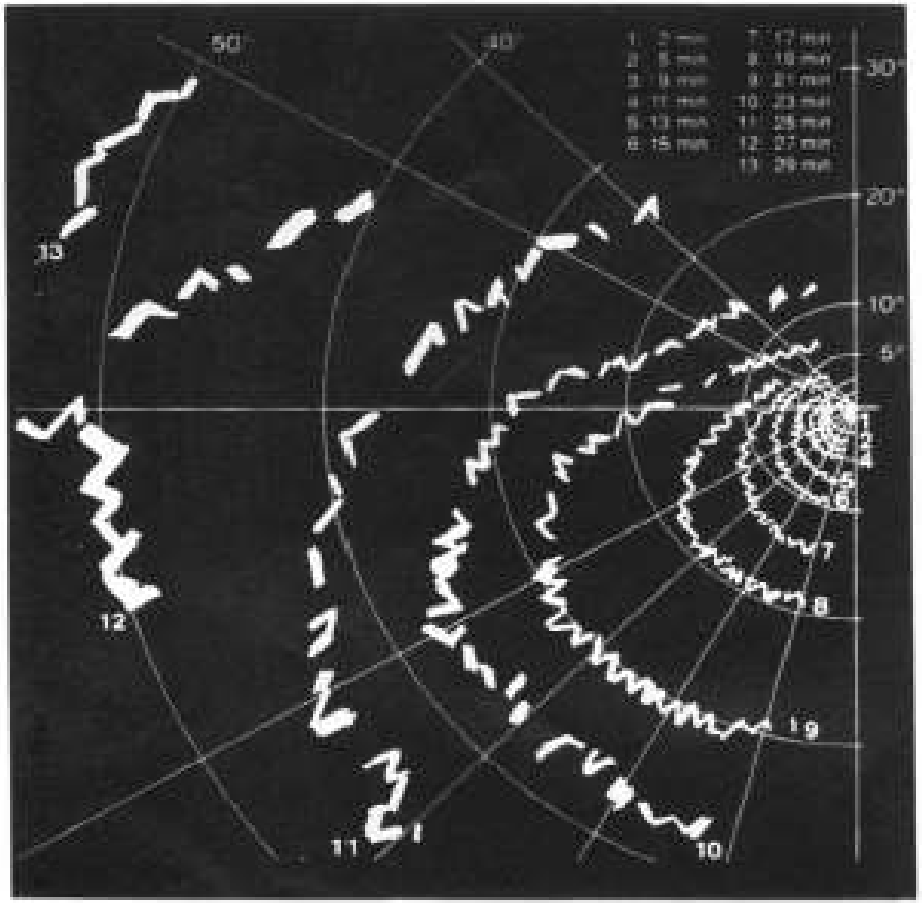
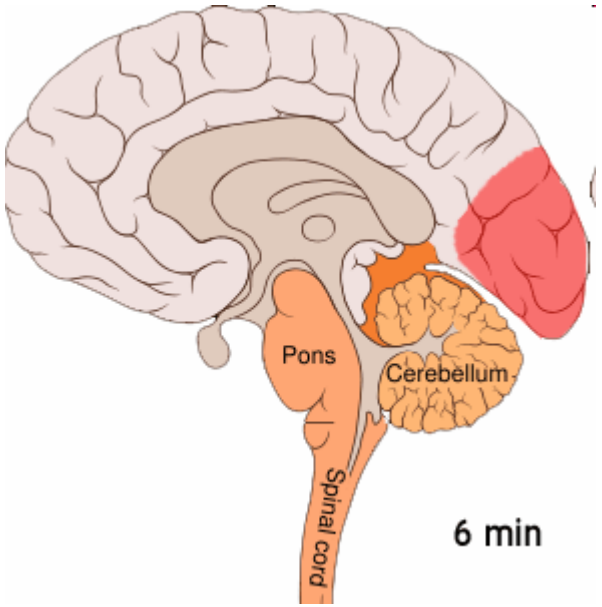


FIGURE 1. Photographic negative of a *migraine phosphene protocol.* The scintillating phosphene was progressing through the lower quadrant and part of the upper quadrant of the left visual hemifield. Thirteen drawings were made between 2 and 29 min after the phosphene appeared near the centre of the visual field. To evaluate the distance between the migraine phosphene and the centre of the visual field, several radii were drawn across the protocol. The angular distance from the fovea centre, computed in degrees of visual angle, is indicated by circles. Circles and radii were added to the protocol sheet after the observations were made. Observation distance, 34 cm.



Q 21/2 Migraine - scotoma

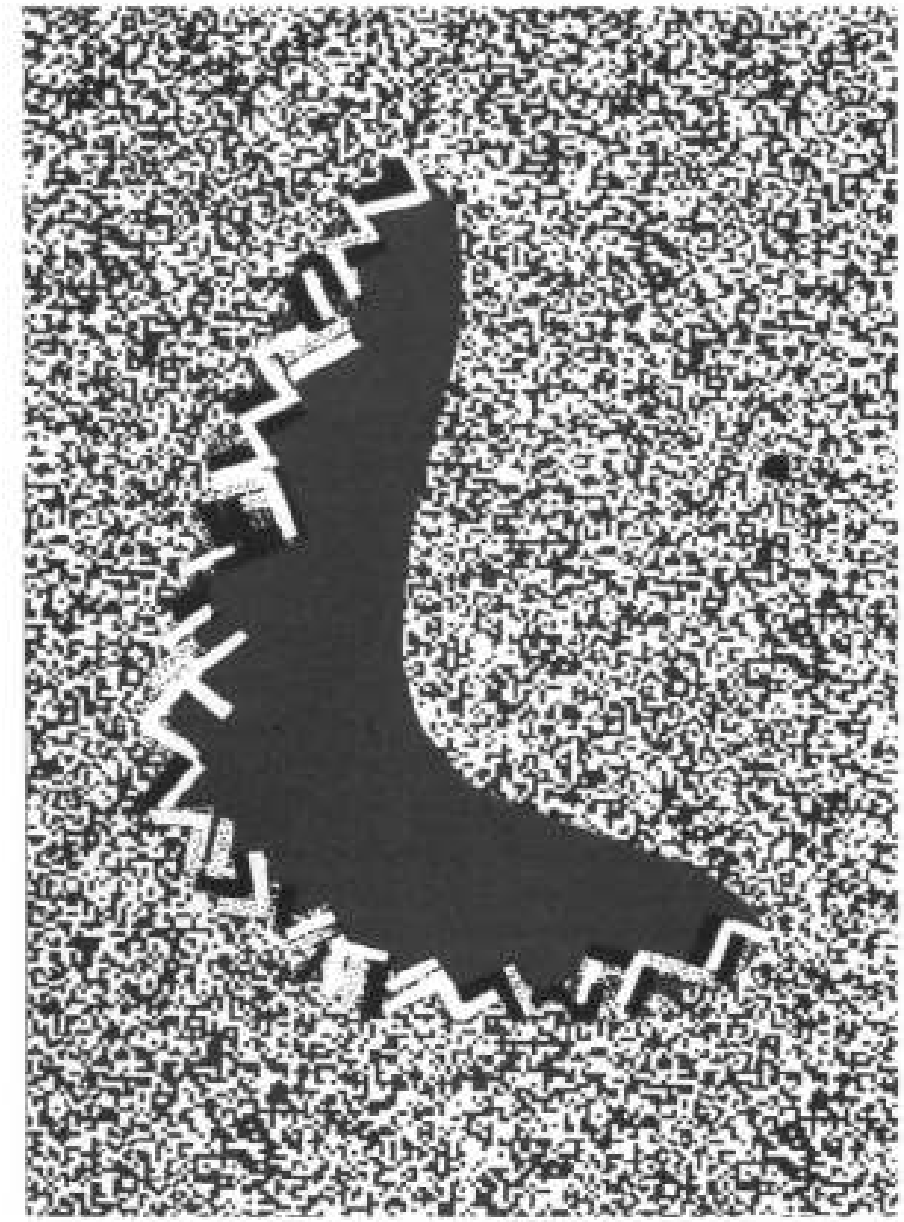
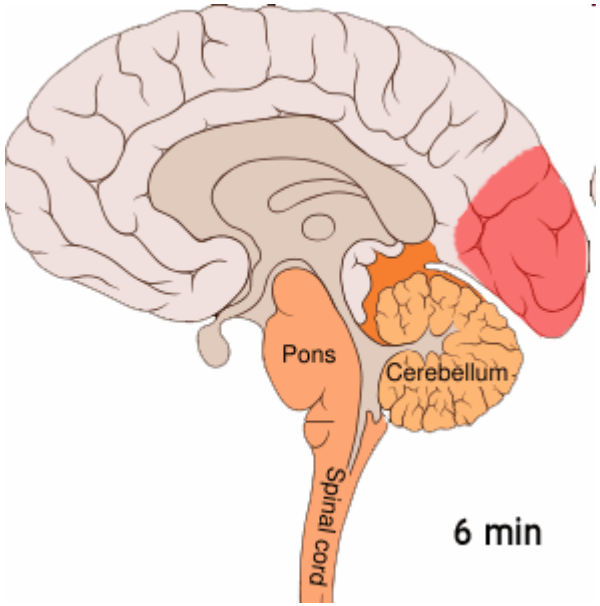


FIGURE 4. Illustration of a scintillating migraine phosphene and its trailing scotoma observed on a *dynamic random-dot noise pattern* (TV screen without program). The scotoma is perceived as a homogeneous neutral grey. Some of the phosphene particles (dotted) appeared in a pure red or green colour, some in deep black (Grüsser & Landis, 1991).



Q 22 Glasgow coma scale

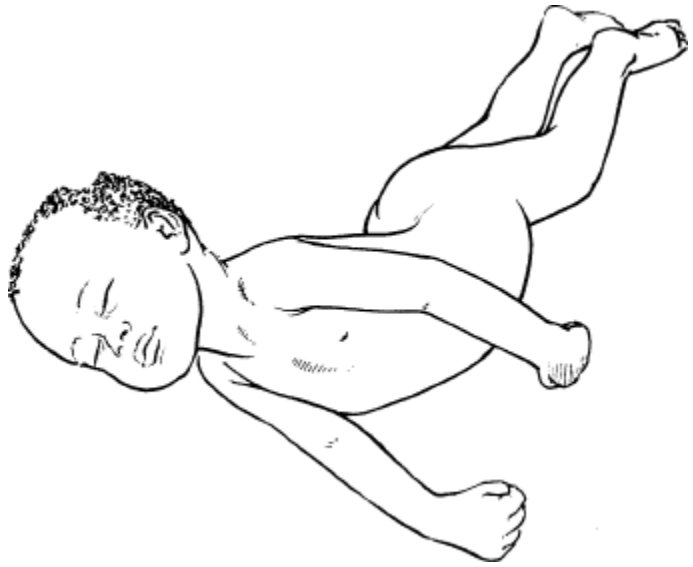
Glasgow Coma Scale

	1	2	3	4	5	6
Eyes	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

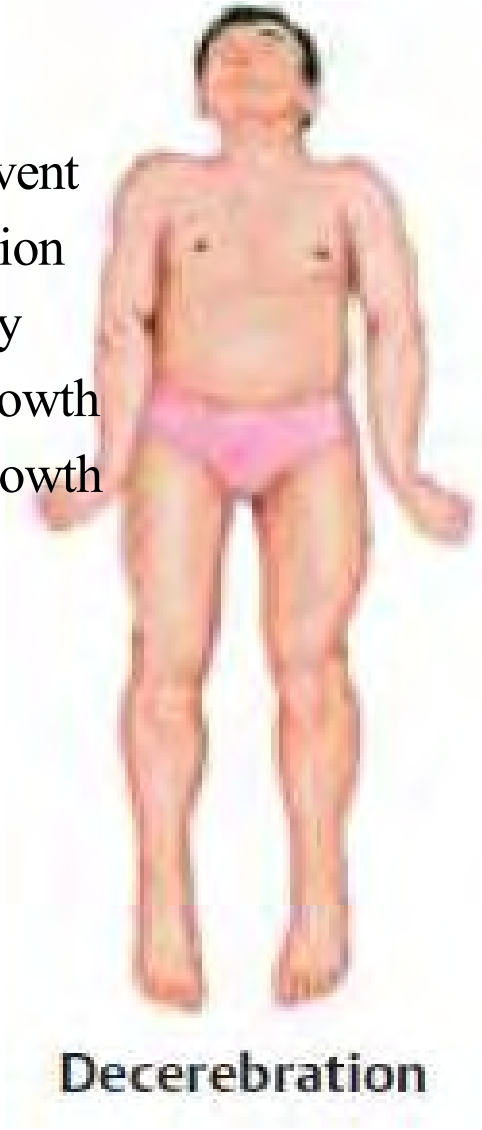
The scale comprises three tests: eye, verbal and motor responses. The three values separately as well as their sum are considered. The lowest possible GCS (the sum) is 3 (deep coma or death), while the highest is 15 (fully awake person).

Q 23 Spinal shock in man

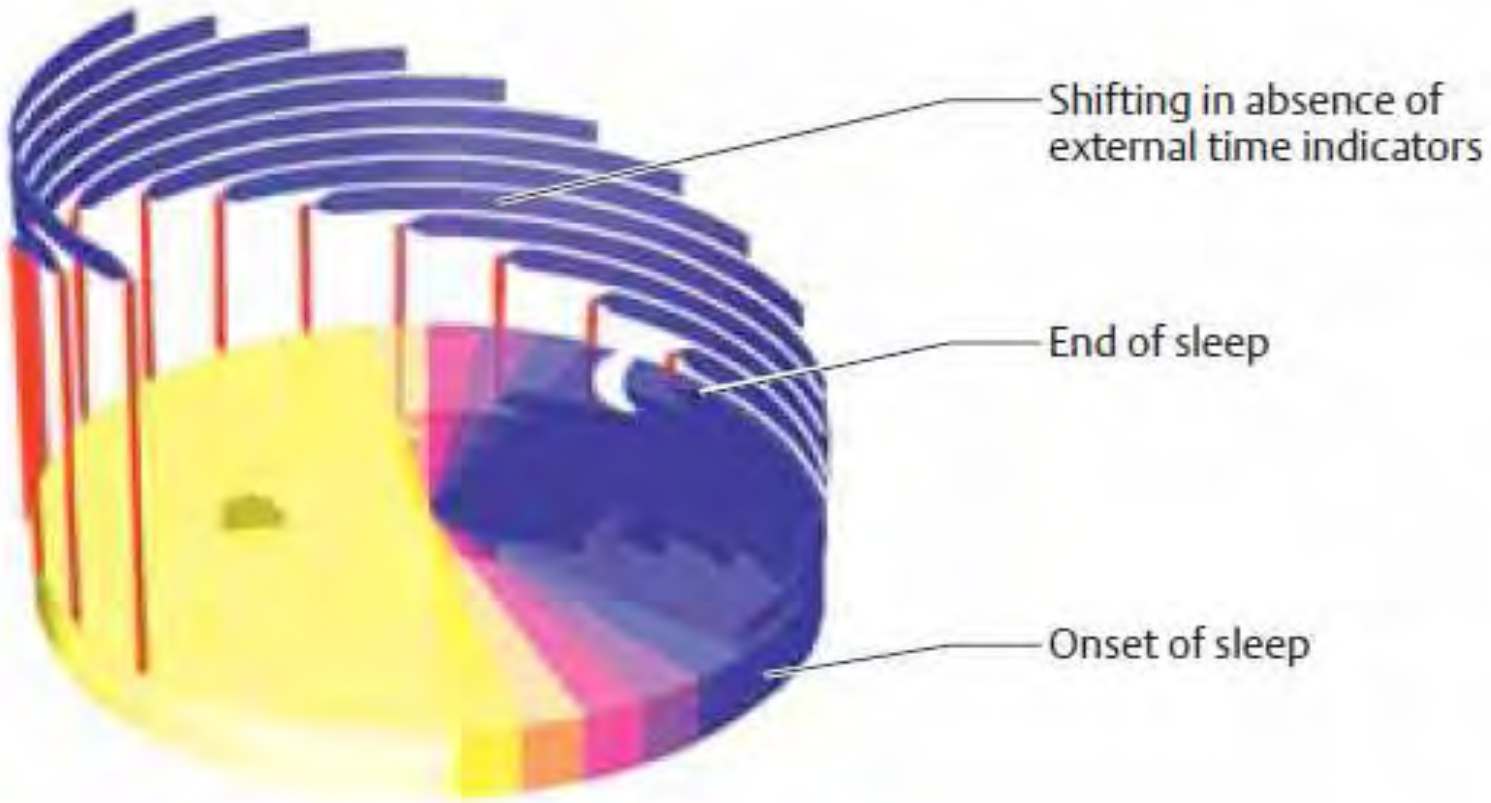
Phase	Time	Physical exam finding	Underlying physiological event
1	0-1d	Areflexia/Hyporeflexia	Loss of descending facilitation
2	1-3d	Initial reflex return	Denervation supersensitivity
3	1-4w	Hyperreflexia (initial)	Axon-supported synapse growth
4	1-12m	Hyperreflexia, Spasticity	Soma-supported synapse growth



In both meningeal irritation and spinal shock extensor systems take over flexor systems

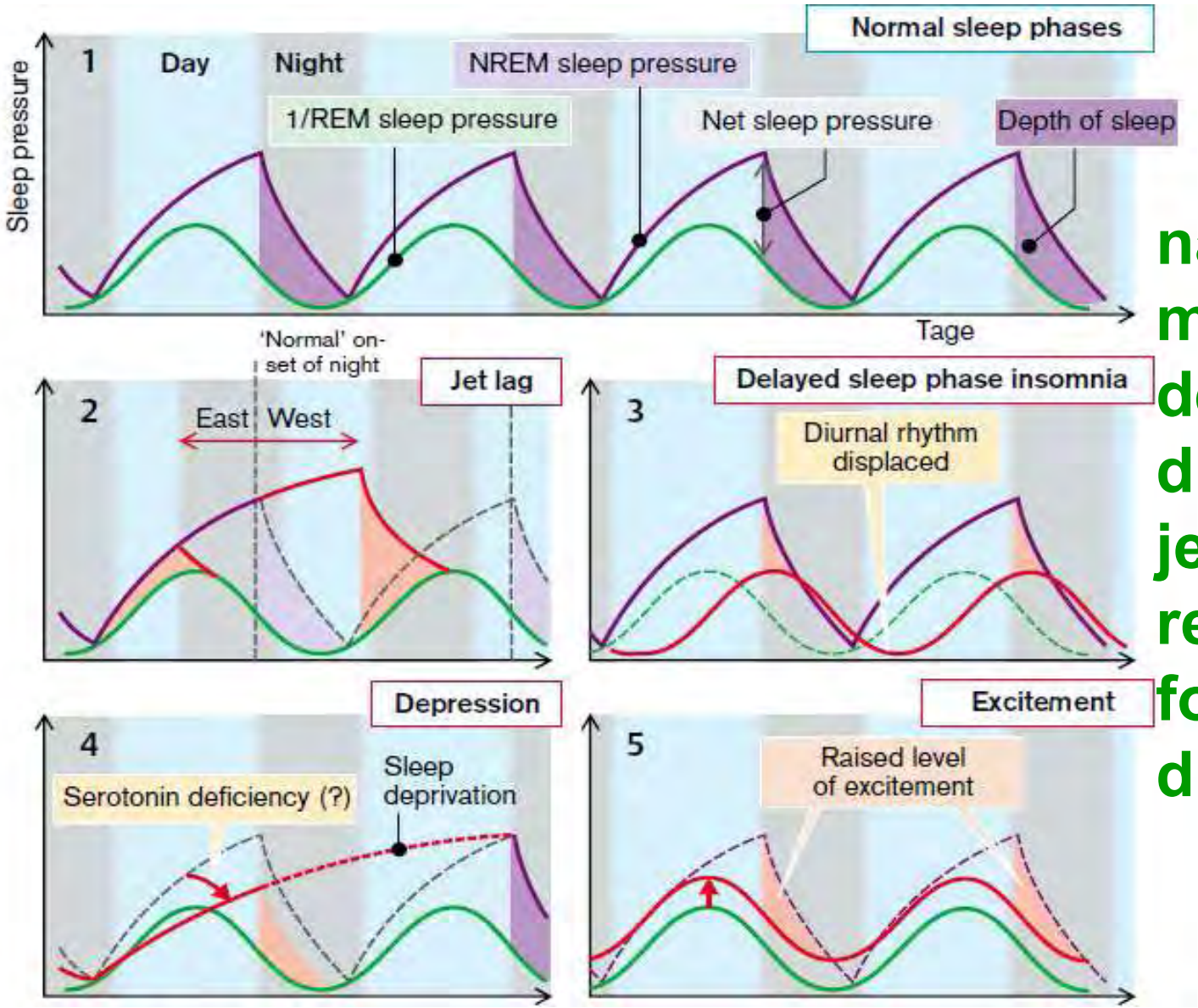


Q 24/1 Sleep/ wake cycle



Circadian rhythm

Q 24/2 Sleep/ wake cycle



narcolepsy, manic-depressive disorder, jet lag, … reticular formation disorders

Q 25 Criteria for cerebral death

TABLE 52–2. Criteria for Cerebral Death (Brain Death)

Prerequisite: All appropriate diagnostic and therapeutic procedures have been performed

Criteria (to be present for 30 minutes at least 6 hours after the onset of coma and apnea):

1. Coma with cerebral unresponsivity (see definition 1)
2. Apnea (see definition 2)
3. Dilated pupils
4. Absent cephalic reflexes (see definition 3)
5. Electrocerebral silence (see definition 4)

Confirmatory test: Absence of cerebral blood flow

Definitions

1. Cerebral unresponsivity—a state in which the patient does not respond purposively to externally applied stimuli, obeys no commands, and does not utter sounds spontaneously or in response to a painful stimulus.
2. Apnea—the absence of spontaneous respiration, manifested by the need for controlled ventilation (that is, the patient makes no effort to override the respirator) for at least 15 minutes.
3. Cephalic reflexes—pupillary, corneal, oculoauditory, oculovestibular, oculocephalic, ciliospinal, snout, pharyngeal, cough, and swallowing.
4. Electrocerebral silence—an EEG with an absence of electrical potentials of cerebral origin over 2 μV from symmetrically placed electrode pairs over 10 cm apart and with interelectrode resistance between 100 and 10,000 Ω.

(Adapted from A Collaborative Study by Ninos, NIH, 1977.)