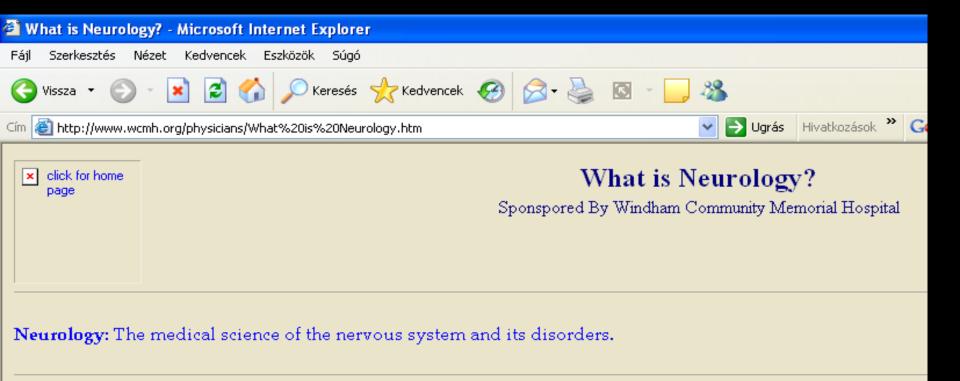
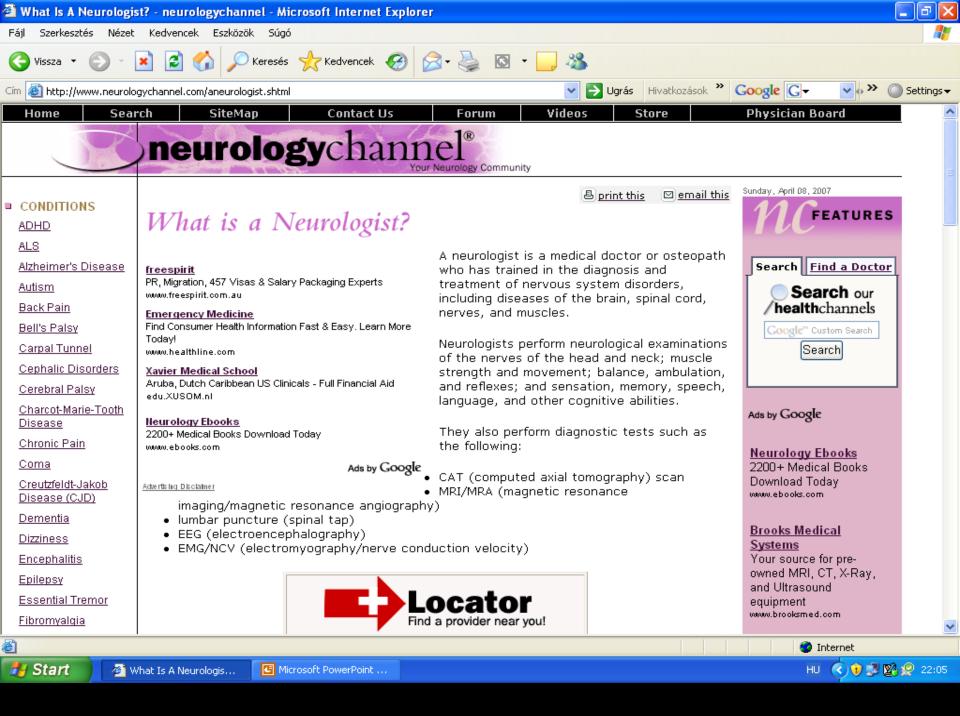
### Introduction into Neurology

Daniel Bereczki
Department of Neurology
Semmelweis University
BUDAPEST





<u>Lou Gehriq's Disease</u>	Education
<u>Lyme Disease</u>	
<u>Meningitis</u>	<ul> <li>Four years of premedical education in a college or university</li> <li>Four years of medical school resulting in an MD or DO degree (doctor of medicine or</li> </ul>
<u>Migraine</u>	doctor of osteopathy degree)
<u>Movement Disorders</u>	<ul> <li>One year internship in either internal medicine or medicine/surgery</li> <li>At least 3 years of specialty training in an accredited neurology residency program</li> </ul>
<u>Multiple Sclerosis</u>	
<u>Multisystem Atrophy</u>	Residency
<u>Myasthenia Gravis</u>	Residency programs accredited by the <u>Accreditation Council for Graduate Medical Education</u>
<u>Myopathies</u>	(ACGME) provide supervised experience in hospital and ambulatory care settings as well as
<u>Nervous System</u>	educational conferences and research trainings.
<u>Tumors</u>	  After completing residency training, neurologists may enroll in a fellowship program to develop
<u>Neurofibromatosis</u>	expertise in a subspecialty such as stroke, dementia, or movement disorders.
<u>Neuropathy</u>	
Normal Pressure	Board Certification
<u>Hydrocephalus (NPH)</u>	
<u>Parkinson's Disease</u>	After completing the educational requirements, medical doctors may seek certification from the <u>American Board of Psychiatry and Neurology</u> (ABPN), a member of the <u>American Board of</u>

The ABPN offers additional certification in the following fields:

Medical Specialties (ABMS).

To become a board-certified neurologist several requirements must be met.

Huntington's Disease

**Hydrocephalus** 

Periodic Limb

Restless Legs

Syndrome

Movement Disorder



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#### ABOUT THE FOUNDATION

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Debi Brooks, President & Co-Founder and Michael J. Fox, Founder

#### **EMAIL UPDATES**

To sign up for e-mail updates and join our mailing list, click here.

The Michael J. Fox Foundation for Parkinson's Research is dedicated to ensuring the development of a cure for Parkinson's disease within this decade through an aggressively funded research agenda.

Enormous progress toward finding a cure has been made on many neurological fronts, and scientists' understanding of the brain and how disease affects it has increased dramatically. The Foundation seeks to hasten progress further by awarding grants that help guarantee that new and innovative research avenues are thoroughly funded and explored.

Actor Michael J. Fox established the Foundation in May 2000 shortly after announcing his retirement from the ABC television show Spin City. In 1998 he publicly disclosed that he had been diagnosed with young-onset Parkinson's disease seven years earlier.

#### AUDITED FINANCIAL STATEMENT

To see our most recent audited financial statement, click here. (pdf file, 811 KB)

#### IRS 990 FORM

#### IN THIS SECTION

#### FAQS

Read commonly asked questions about the Foundation and how you can help us in our fight against Parkinson's disease.

Click here for more information

#### BOARD OF DIRECTORS

MJFF's Board of Directors is made up of leaders in science, business and entertainment. Their expertise in such varied fields helps to guide the Foundation in its pursuit of raising money for the sole purpose of funding Parkinson's research. Click here for more information

#### SCIENTIFIC ADVISORS

MJFF's Scientific Advisory Board is composed of a multidisciplinary group of leading Parkinson's researchers and clinicians from across North America. Its members are actively involved with all the strategic meetings, grant review sessions, and other activities that guide the Foundation's scientific and

### OUTLINE

- Learning requirements
  - What to study
  - Practicals
  - Exams
- Subject of neurology
- The neurological diagnosis
- Case presentations
- Patient presentation

### What to study?

- Textbook: show what you have to the tutor of your group to see if it is OK, e.g.
  - Mumenthaler
  - Neurology Neurosurgery Illustrated,
  - Walton, Victor-Adams, Netter, etc
- What is presented at the lectures
- What you are taught at practical classes
- Practical textbook of the Department
- E-learning material of the Department

## E-learning

- Individual study
- Interactive learning
- Self assessment at the end of chapters

 Use the same username and password as for other e-learning materials of Semmelweis University

### E-learning





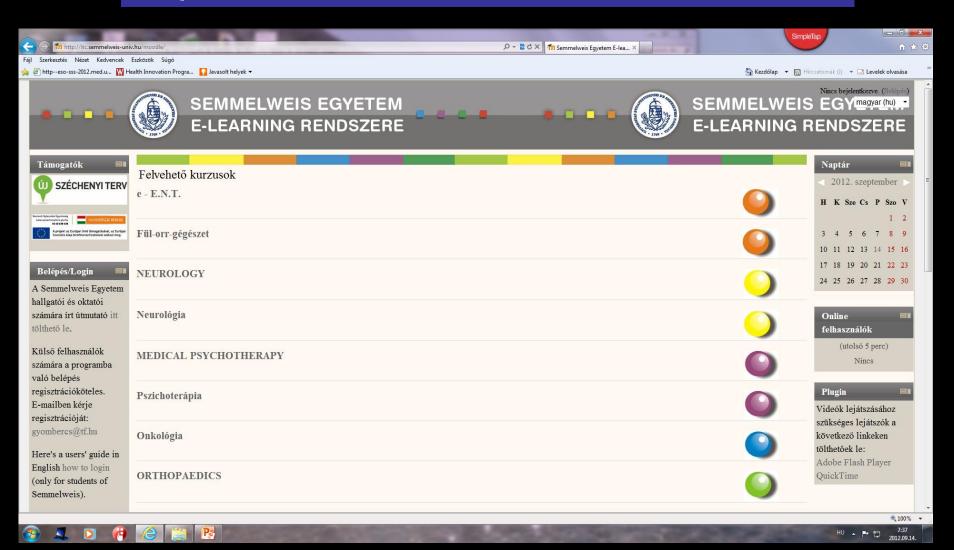
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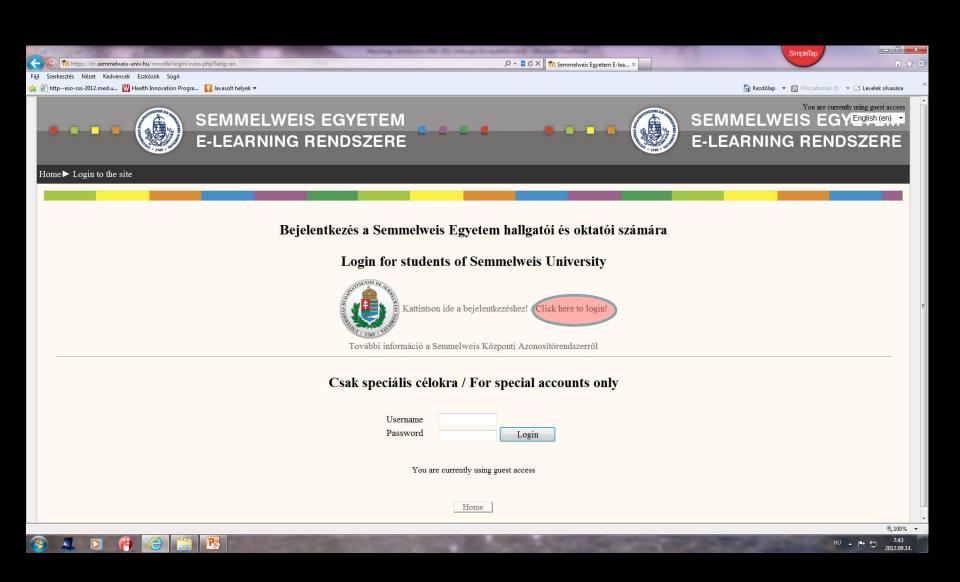


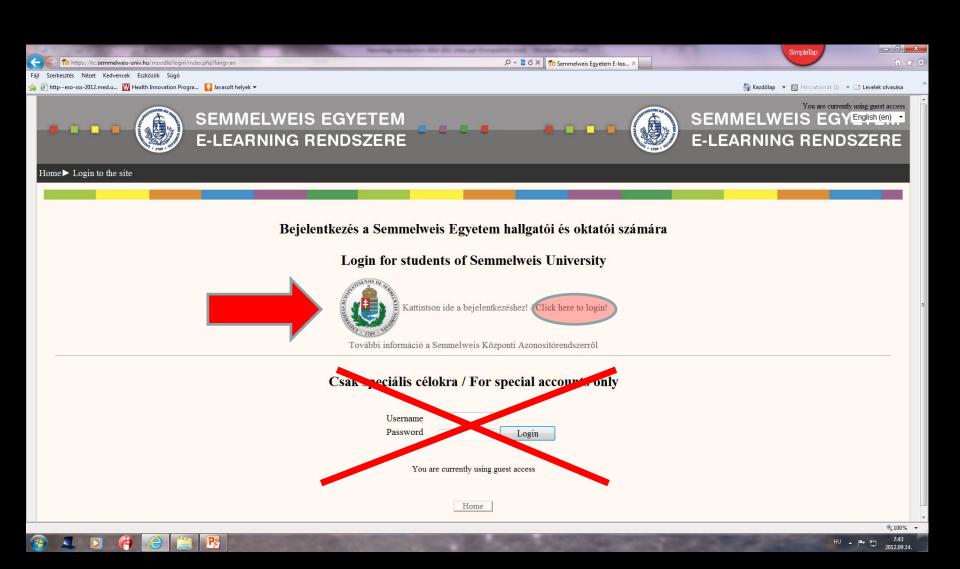


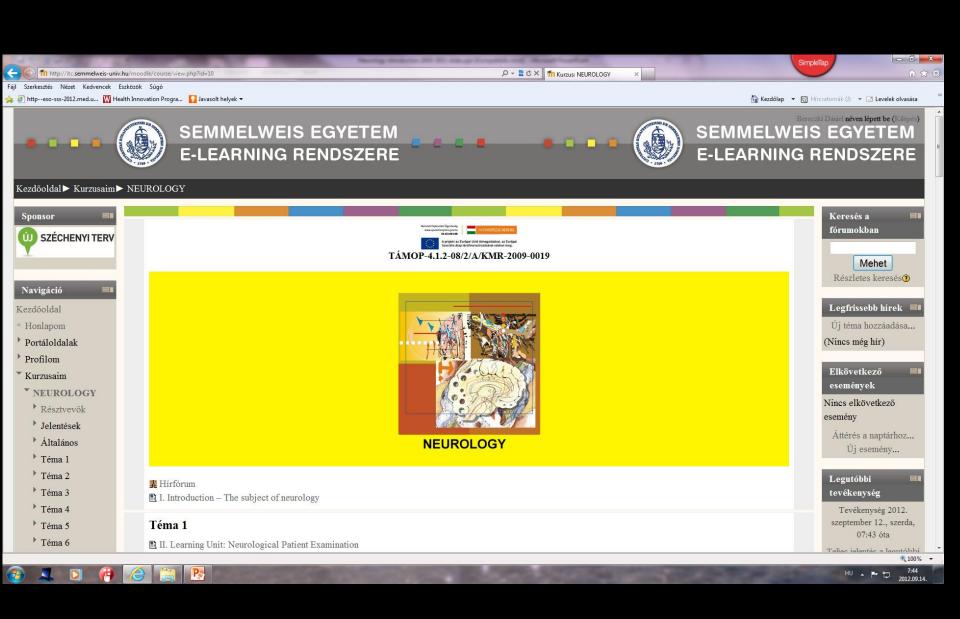
www.semmelweiskiado.hu/

### http://itc.semmelweis-univ.hu/moodle/









Lesion of cortical eye movement centers and of the descending fibers causes contralateral horizontal gaze palsy. Lesion of the pontine gaze center causes spalateral horizontal gaze palsy. For further details, refer to the recommended sources.

#### Symptoms of oculomotor nerve lesion

The nerve exits the brainstem between the superior cerebellar artery and the posterior cerebral artery; therefore an aneurysm on these vessels may lead to oculomotor nerve lesion. The external (partial=without the involvement of parasympathetic pupillary function) oculomotor nerve lesion seen in diabetic patients is caused by ischemia. On the affected side, ptosis is present, and the eye is deviated laterally and downward because the intact abducens and trochlear nerves pull the eye in this position. The patient complains of diplopia. If the parasympathetic fibers are also affected, the pupil is dilated, the direct pupillary light reflex and the accommodation reaction are lost. This is a complete oculomotor nerve lesion (Fig. 5)



Fig.

The consensual light reflex can be elicited from the abnormal, wide pupil, but no consensual light reflex is seen on the abnormal side when the intact eye is illuminated.

Ptosis may be unilateral or bilateral. Unilateral ptosis is caused by oculomotor nerve lesion. Causes of bilateral ptosis include 1) congenital ptosis; 2) chronic progressive ophthalmoplesia; 3) myasthenia gravis; 4) central lesion of the oculomotor nucleus.

In case of circulatory insufficiency of the brainstem, the axons of the oculomotor nerve may be damaged before exiting, at the base of the midbrain. If the ischemia affects the corticospinal pathway or the red nucleus, then contralateral hemiparesis or contralateral intentional tremor also develops in addition to the ipsilateral oculomotor nerve lesion (Weber's and Benedict's syndromes) (go to the video).

Inflammation or cellular infiltration on the basal part of the brain (bacterial meningitis, syphilis, tuberculosis, meningeal carcinomatosis) may also damage the nerve.

Herniation of the temporal lobe due to space occupying lesions causes dislocation of the cerebral peduncle, which damages the nerve as well (see below).

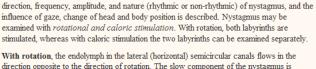
#### Symptoms of trochlear nerve lesion

When the trochlear nerve (4th cranial nerve) is damaged, the affected eye's movement is





In peripheral nystagmus, how is the direction of nystagmus determined?



Examination of nystagmus: the eyes are first observed in resting position, then during

following eye movements in all four directions, in both sitting and supine positions. The

With rotation, the endolymph in the lateral (horizontal) semicircular canals flows in the direction opposite to the direction of rotation. The slow component of the nystagmus is opposite to the direction of rotation, therefore it is in the same direction as the flow of endolymph; the quick component beats in the direction of rotation. When rotation is stopped, the direction of post-rotational nystagmus is inverted, thus the quick component now beats opposite to the direction of rotation. The direction of deviation and past-pointing is the same as that of the slow component.

With caloric stimulation, warm water is injected into the ear, which causes an ampullopetal flow of the endolymph in the lateral semicircular canal and a nystagmus beating in the direction of the stimulus. With cold stimulation, the direction of nystagmus is towards the opposite side. Caloric stimulation is suitable for determining whether the nystagmus is caused by a lesion to the vestibular organ. Caloric stimulation on the side of lesion produces no nystagmus, and causes no change in any on-going nystagmus.

The Romberg's test is used to differentiate peripheral and central vertigo.

#### Description of normal findings

Whispered words are well heard on both sides. Weber test is normal. Rinné test is positive on both sides. No nystagmus. No swaying in Romberg's test. No past-pointing in Bárány's test. No deviation when walking with eyes closed.

#### Nystagmus

Nystagmus is an involuntary, rhythmic eye movement with a slow and a quick component, occurring in the presence of dysfunction of the vestibular, cerebellar and the eye movement control system. Based on the relation of the quick component and the direction of gaze, nystagmus of peripheral vestibular origin may be of 1st degree if the nystagmus appears only when looking in the direction of the quick component, 2nd degree if it appears already when looking straight ahead (go to the video), and 3rd degree if the nystagmus is present in any direction of the gaze. The direction of the quick component of the nystagmus may be horizontal, vertical (go to the video), oblique or rotatory. Nystagmus of peripheral vestibular origin is rhythmic. The slow component results from the activity of the intact side, the quick component is a compensatory restoring saccade produced by the brainstem. Undulating nystagmus is irregular, no slow and quick components can be differentiated.

#### Physiological nystagmus types

a.) Induced nystagmus of labyrinthine origin: physiological nystagmus resulting from the stimulation of the semicircular canals. It can be elicited by rotation, and by cold/warm and galvanic stimulation of the peripheral vestibular system. In the lateral semicircular canal, ampullopetal flow of the endolymph (towards the ampulla) induces nystagmus beating in the opposite direction, whereas ampullofugal flow (from the ampulla) induces nystagmus beating in the same direction.





Peripheral vestibular syndrome







### Exams

- End of semester (first time in schoolyear 2007/2008 due to the credit system)
  - Practical exam (1-5)
  - Requirements:
    - What you learned on the practical classes
    - What you heared on classroom lectures
    - Departmental textbook
- End of year exam
  - Practical exam
  - Written test in the e-learning system
  - Option to improve in oral exam

### Neurology

- Central nervous system
  - Brain
  - Spinal cord
- Radices, plexus, nerves
- Neuromuscular junction
- Muscles

### Borderzones

- Internal medicine
- Neurosurgery
- Psychiatry
- ENT
- Ophthalmology
- Urology
- Dermatology
- ETC

### Tasks

- To have in mind the possibility of a neurological disease based on
  - Anamnesis (history)
  - Physical exam
- Diagnostic plan (decide on ancillary investigations)
- Come to a diagnosis
- Determine steps of treatment
- Plan follow-up

## Frequent neurological disorders

- Cerebrovascular disorders
- Tumors
- Epilepsy
- Multiple sclerosis
- Parkinson syndrome
- Dementias
- Headache

- Trauma
- Metabolic disorders
- Developmental disorders
- Inflammatory diseases
- Neuropathies
- Chronic pain syndromes

# Tasks in general practice

- Take the history and perform exam.
- Consider a neurological disease.
- Answer the 4 questions.
- Organize diagnostic procedures.
- Decide on treatment.
- Educate and help relatives of patient

# Taking the hisory

- Not enough time.
- What exactly mean the patient and the relative on the complaint?
- Do you suspect a neurological disease?
- Is there an emergency?

### What to consider at history

- Age
- Clarifying the symptoms
- Mode of onset and progression
- Chronological sequence of events
- Value of negative information
- Exclude irrelevancies
- Drugs
- Heteroanamnesis (interviewing relatives)

# Problems with history

- Time
- Missing data
- Misunderstandings

"If a neurologist were in a group of people, stranded on a desert island, and if he were to be bereft of sight, arms and legs, but was still able to speak and hear, he would be able to take a history..... By the time the history is complete, the physician should be three-quarters of the way towards diagnosis, and, if he is not, then there is something wrong with the way in which it has been taken."

### The neurological record of a patient

- Personal data
- History (taken from and by)
- Internal exam
- Neurological exam
  - Meningeal signs and signs of injury
  - Cranial nerves
  - Motor system
  - Sensory system
  - Reflexes
  - Co-ordination
  - Vegetative functions
  - Psychiatric condition
- Summary
- Opinion (probable diagnoses)
- Diagnostic plan
- Followup

### The 4 questions to answer

- 1. Is there a neurological disease?
- 2. If yes, where is the lesion?
- 3. What pathological conditions may cause a lesion at this site?
- 4. In this patient which of these conditions is the most likely to be present?

# If you suspect a neurological disease

- Think over what to do with the patient.
- Can you take the responsibility to treat this patient?
- Is it necessary to send the patient to a neurologist?
- How urgent it is?

# Organizing the diagnostic procedures

- Is it an emergency?
- If yes, where to send the patient?
- If not, how far can I get in the diagnostic process?
- What ancillary investigations to ask for, and from whom?
- Where do they perfom these investigations?
- If the appointment is at a distant time, is it safe to wait?

### Organizing the care of the patient

- I reached the diagnosis myself or with a help of a specialist.
- Who determines the therapy?
- Is there a need for pharmacological or other treatments?
- Who may prescribe certain drugs?
- Shall I prescribe original or generic drugs?
- How frequently shall I check the patient?
- What to do during checkup exams?
- When shall I send back the patient to a neurologist?
- Shall I send to an outpatient service or to hospital?

### Education of relatives

- Is it needed to involve relatives
  - When taking history?
  - When deciding on treatment options?
  - When organizing long term care?
- Pick the proper person from the relatives.
- Keeping contact with the relatives.

# Ancillary invstigations to confirm or refute the suspected diagnosis

- Methods examining structure
  - X-ray, CT, MRI, ultrasound
- Imaging methods examining function
  - fMRI, SPECT, PET, ultrasound
- Electrophysiological methods
  - EEG, ENG, EMG, evoked responses
- Examination of the cerebrospinal fluid
- Immunological, genetic and molecular biological investigations
- Cytology and pathological investigations
- Consultations with other specialities

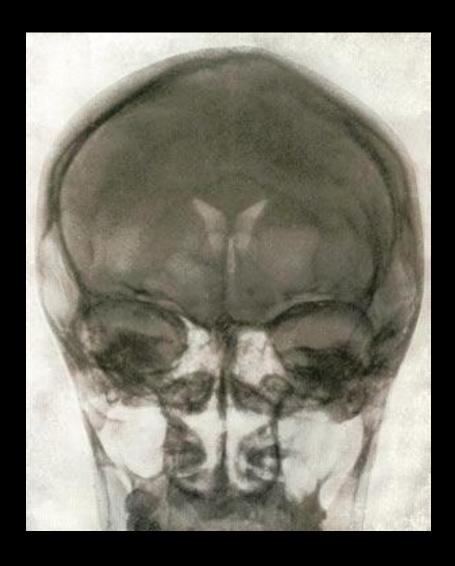
# Old methods (until mid 80-ies)



1. day: cisternali CSF sampling



2. day: percutaneous carotid angiography

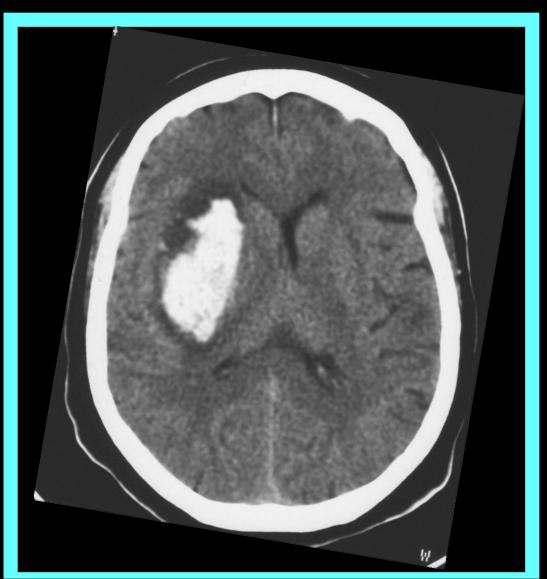




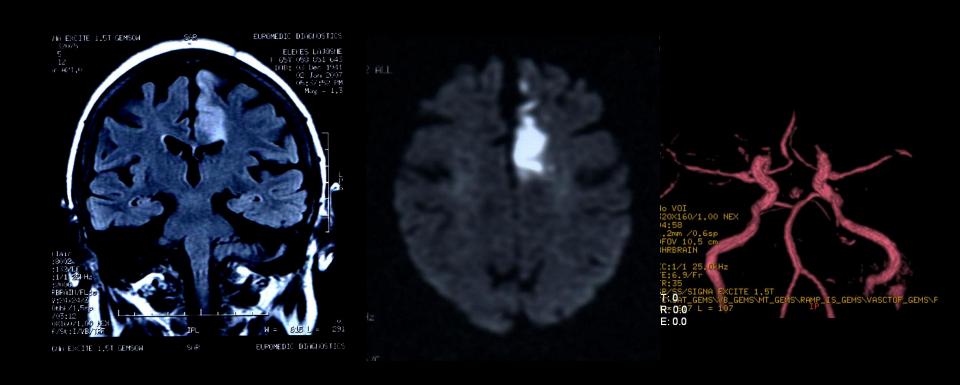
PNEUMOENCEPHALOGRAM

MR IMAGING

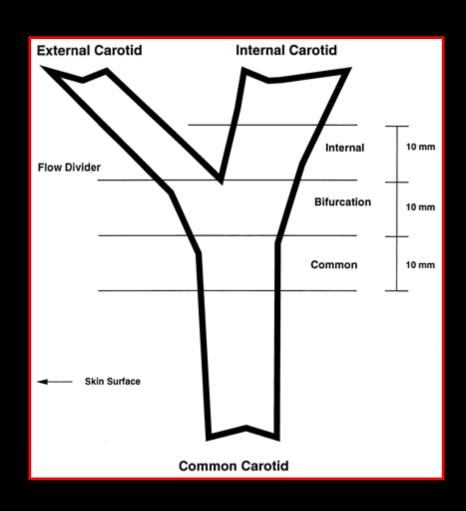
# Intracerebral hemorrhage



### Arteria cerebri anterior

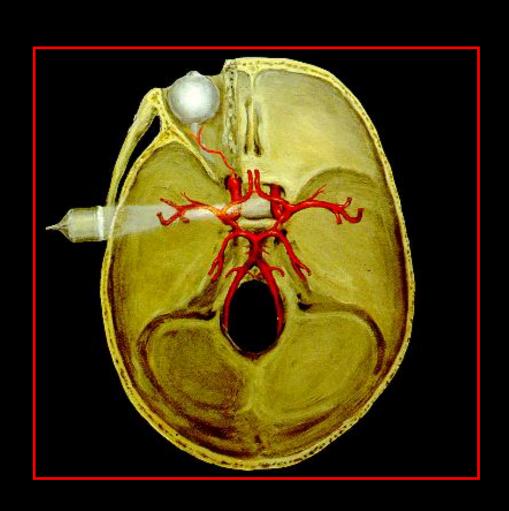


### **Carotid Ultrasound**

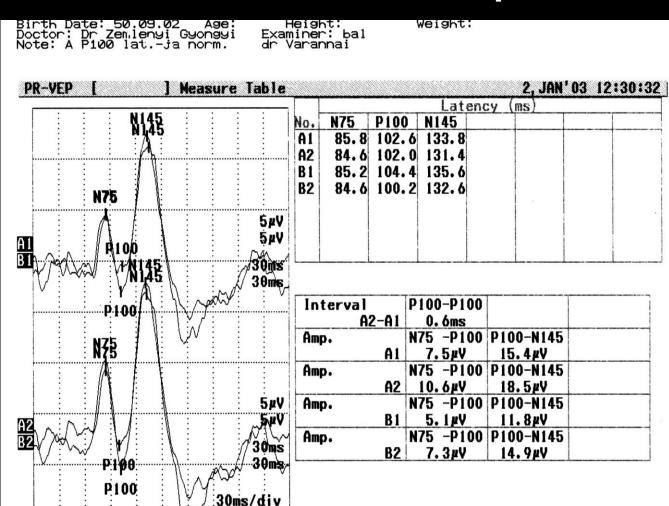




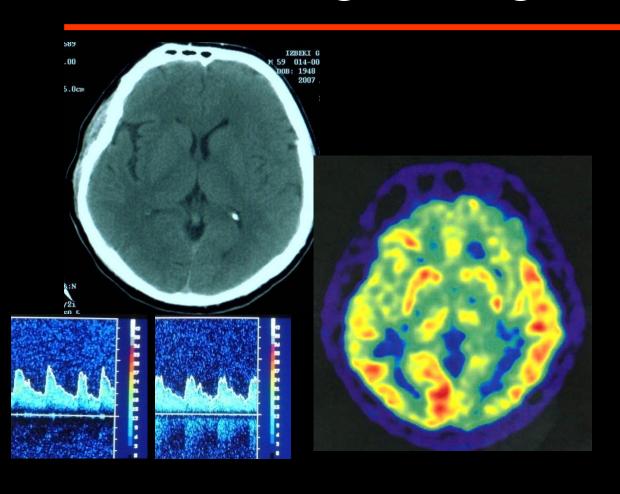
# **Transcranial Doppler**

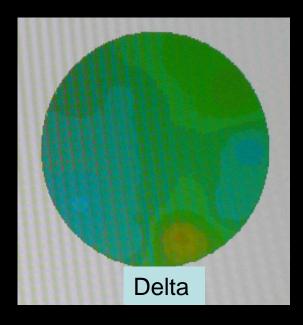


### Visual evoked response



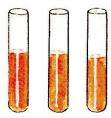
# Prolonged migraine aura





#### Cerebrospinal fluid

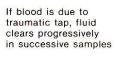
Three successive fluid samples collected. Shortly after or during bleeding, all 3 samples frankly bloody or orange



Later, on repeat tap, all 3 samples are xanthochromic (yellow) as a result of hemoglobin release or bilirubin formation

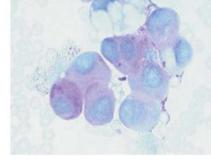




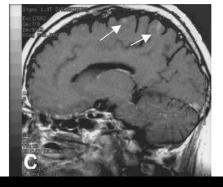


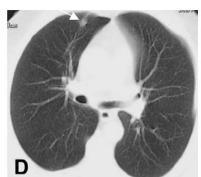




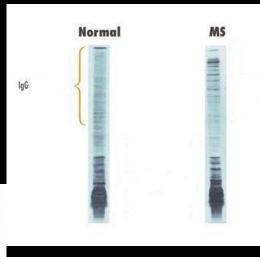




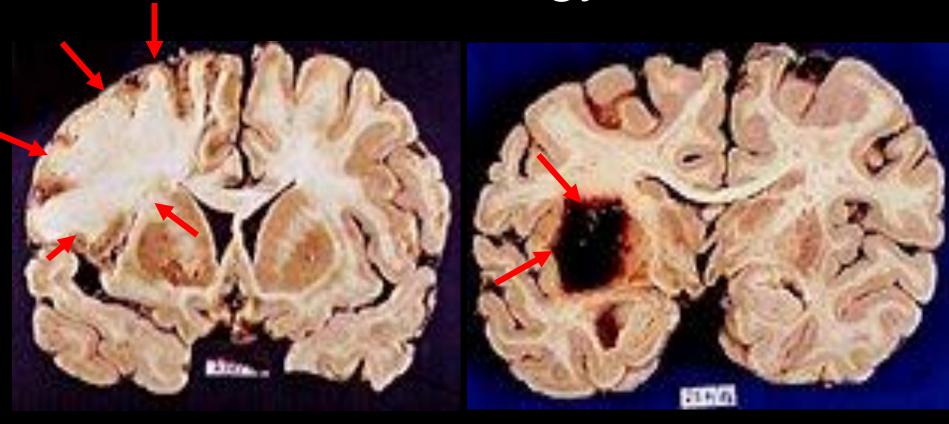




# CSF examination



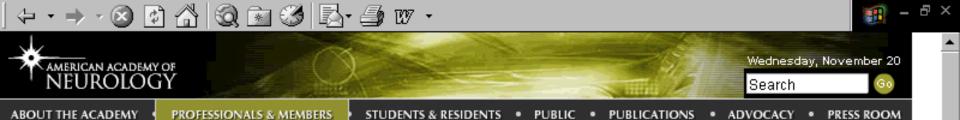
# Pathology



**ISCHEMIC STROKE** 

INTRACEREBRAL HEMORRHAGE

Hegedűs, 2001



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  Agents in Acute Stroke (.pdf).
- Parkinson's Disease Initial Treatment of Parkinson's Disease (.pdf).
- Dementia

AD can be reliably diagnosed; early diagnosis is possible and important. While AD is not curable, there are treatment and care options available today.

- Detection of Dementia-Mild Cognitive Impairment: (.pdf)
   AD and MCI differ from normal aging. Patients with MCI should be identified and monitored, as progression to AD is likely.
- <u>Diagnosis of Dementia</u> (.pdf)
   The clinical criteria for AD are reliable and valid, data (.pdf)
- Management of Dementia (.pdf)
   Cognitive and behavioral symptoms can be treated.
   Caregiver programs are effective. data (.pdf)
- Summary Version for Physicians, Summarizes all three