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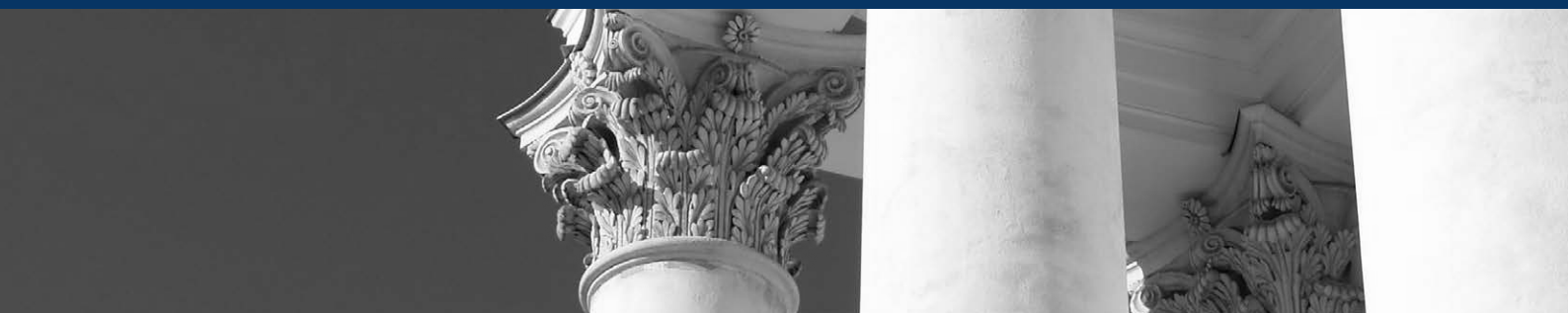
"IULIU HATIEGANU" UNIVERSITY
OF MEDICINE AND PHARMACY

DOCTORAL SCHOOL NEUROSCIENCE

PROGRAM

2018-2019 | SECTION 5

21 - 22 FEBRUARY 2019
"MULTIMEDIA" AUDITORIUM, "IULIU HATIEGANU" UMF CLUJ-NAPOCA
8 VICTOR BABES STREET | CLUJ-NAPOCA | ROMANIA



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President of Hungarian Neurological Society

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Department of Neurology, University of Debrecen, Hungary

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2018-2019

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COURSE PROGRAM

COURSE PROGRAM

THURSDAY, 21 FEBRUARY 2019

"MULTIMEDIA" AUDITORIUM, "IULIU HATIEGANU" UMF CLUJ-NAPOCA
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09:50 – 10:00	Welcome Address
10:00 – 10:45	Management of carotid stenosis: CEA vs STENT Natan Bornstein /Israel
10:45 – 11:30	Secondary stroke prevention Natan Bornstein /Israel
11:30 – 12:00	Coffee Break
12:00 – 12:45	Time is brain, TIA as an emergency Natan Bornstein /Israel
12:45 – 13:30	Post stroke depression Natan Bornstein /Israel
13:30 – 15:30	Session Break
15:30 – 16:15	Efficacy of stroke units Michael Brainin /Austria
16:15 – 17:00	Functional anatomy of the brain Michael Brainin /Austria

COURSE PROGRAM

17:00 – 17:30	Coffee Break
17:30 – 18:15	Post-stroke cognitive decline: intervention trials for prevention and treatment Michael Brainin /Austria
18:15 – 19:00	The global stroke epidemic: prevention is the main issue Michael Brainin /Austria

FRIDAY, 22 FEBRUARY 2019

"MULTIMEDIA" AUDITORIUM, "IULIU HATIEGANU" UMF CLUJ-NAPOCA

8 VICTOR BABES STREET | CLUJ-NAPOCA | ROMANIA

10:00 – 10:45	Bedside differential diagnosis of disturbance of consciousness Laszlo Csiba /Hungary
10:45 – 11:30	The use of neurosonological methods in pharma research Laszlo Csiba /Hungary
11:30 – 12:15	The diagnosis of brain death. Case presentations Laszlo Csiba /Hungary

INTERNATIONAL GUEST LECTURERS

NATAN BORNSTEIN
ISRAEL

EDUCATION

1970-73	University of Sienna, Medicine, Sienna, Italy
1973-79	Technion Medical School, Hifa, Medicine, MD, 1979

Date of receiving specialixation certificate: 11 September, 1984
Title of Doctoral dissertation: Dextran 40 in acute ischemic stroke
Name of Supervisor: Dr. Jacob Vardi

FURTHER EDUCATION

1978-83	Tel-Aviv University, Sackler Faculty of Medicine, neurology (residence), Israeli Board certified in Neurology, 1983
1979-83	Tel-Aviv University, Sackler Faculty of Medicine, Post graduate studies in Neurology
1984-87	Sunnybrook Medical Center, University of Toronto, M.R.C stroke, Fellowship

ACADEMIC AND PROFESSIONAL EXPERIENCE

1982-1995	Tel-Aviv University, Neurology, instructor
1991-present	European stroke Conference (ESC), Executive committee
1995-1999	Tel-Aviv University, Neurology, Senior lecturer
1995	Eliprodil CVD 715 clinical trial, Steering Committee
1995-1997	International Stroke Study (IST), Steering Committee
1995-1999	American Academy of Neurology, Member of the International Affairs Committee
1996	Asymptomatic Carotid Stenosis and Risk of Stroke(ACSRS), Advisory Committee
1996-present	The Mediterranean Stroke Society (MSS), President
1996-2002	EFNS, Management Committee
1997-2009	Israeli Neurological Association, Secretary
1999-present	Tel-Aviv University, Neurology, Associated Professor
2001- present	European Society Neurosonology and Cerebral Hemodynamics (ESNCH) Executive committee
2005-present	Neurosonolgy Research Group, Executive committee
2006-present	European Master in Stroke Medicine, Member of faculty
2006-2008	NEST II clinical Trial, Steering Committee
2006-present	SENTIS clinical Trial, Steering Committee
2006-present	CASTA Trial, Steering Committee
2006-present	Brainsgate clinical Trial, Steering Committee
2008- present	World Stroke Association (WSO), Vice president
2009-present	Israeli Neurological Association, Chairman
2009-present	European Stroke Organization (ESO), Member on the board of directors
2010-	NEST III clinical Trial, Steering Committee

PROFESSIONAL ACHIEVEMENTS- EDITORIAL BOARD

1991-present	Neurological Research Journal, Guest Editor
1991-present	STROKE, Member of the editorial board
1998-present	European Journal of Neurology, Member of the editorial board
1999-present	Journal of Cerebrovascular disease, Member of the editorial board
2000-present	Journal of Annals of Medical Science, Consulting Editor
2001-present	Journal of Neurological Science (Turkish), Member of the editorial board
2001-present	Acta Clinica Croatica, Member of the editorial Counsil
2003-present	Italian Heart Journal, International Scientific Board
2003-present	Journal of Neurological Sciences, Guest Editor
2004-present	Turkish Journal of Neurology, International Advisory Board
2005-present	Archives of Medical Sciences (AMS) , Member of the Editorial Board
2006-present	Journal of Cardiovascular Medicine, International Scientific Board
2006-present	International Journal of Stroke, Editorial Board
2006-present	Acta Neurologica Scandinavica, Editorial Board
2009-present	American Journal of Neuroprotection& Neurogeneration (AJNN) Member of the Editorial Board
2010	Neurosonology, International Editorial Board
2010	Frontiers in Stroke, Review Editor

PROFESSIONAL ACHIEVEMENTS- REVIEWER

1998-present	Lancet, Ad Hoc reviewer
1998-present	Diabetes and its complications, Ad Hoc reviewer
1999-present	Journal of Neuroimaging, Reviewer
1999-present	Journal of Neurology, Ad Hoc reviewer
2000-present	Neurology, Ad Hoc reviewer
2003-present	Israeli Medical Association Journal (IMAJ), Reviewer
2003-present	Acta Neurologica Scandinavica, Ad Hoc reviewer
2006-present	Journal of Neurology, Neurosurgery & Psychiatry, Reviewer
2010-	European Neurology, Ad Hoc reviewer

MEMBERSHIP IN PROFESSIONAL SOCIETIES

1977-present	Israeli Medical Association
1983-present	The Israeli Neurological Association
1985-present	Stroke Council of the American Heart Association (Fellow)
1986-present	American Academy of Neurology
1986-present	Neurosonology Research Group of the World Federation of Neurology
1987-present	Stroke Research Group of the World Federation of Neurology
1990-2008	International Stroke Society
1995-2008	European Stroke Council
1995-present	Mediterranean Stroke Society (MSS)
1998-present	European Neurosonology Society
2005-present	World Stroke Organization (WSO)
2008-present	Fellow of the European Stroke organization (FESO)

MICHAEL BRAININ
AUSTRIA

Professor and Chair, Department of Clinical Neurosciences and Preventive Medicine Danube University Krems, Austria.

He was co-founder of the national stroke unit network and founding president of the Austrian Stroke Society 2003-2006.

He was President of the European Stroke Organisation (2012-2014).
Currently he is the President of the World Stroke Organisation (2018-2020).
He is co-chair of the ESO-WSO 2020 Congress to be held in Vienna, Austria.

Dr. Brainin has led the WSO Education Committee 2008-2017 and was editor of the World Stroke Acedemy, a web-based learning platform for the WSO. He chairs the European Master's Program in Stroke Medicine since 2007. He has published more than 200 peer-reviewed papers, edited three textbooks on stroke, and has given more than 1.000 invited lectures. His scopus h-index is 46 and scopus citations are >15.000.

He is Senior Editorial Consultant for ‚Stroke', Associate Editor of the European Journal of Neurology and member of the editorial boards of Neuroepidemiology, International Journal of Stroke, The European Stroke Journal and The Journal of Neurological Sciences. He received several awards, honorary doctorates and honorary memberships from scientific societies.

LÁSZLÓ CSIBA
HUNGARY

Professor of the Department of Neurology at the University of Debrecen, Hungary since 1992.

- visiting scientist in the Max-Planck Institute for Neurological Research in Cologne (1981-83),
- one year in Kure City, Japan (1986)
- half year in Toulouse (INSERM, France).
- He is the founder of Hungarian Neurosonological Society (1994),
- honorary member of Austrian Stroke Society,
- visiting professor of Belgrade, Cluj/Kolozsvár, Targu Mures/Marosvásárhely, Novi Sad/Újvidék University and Israel Association of Neurology.
- Editorial board member of "International Journal of Stroke", "LAM" "Neurosonology (Japan)" "Frontiers in Stroke" (associate editor"), "Neurosonology and Cerebral Hemodynamics", "Emergency Medicine Search and Rescue Journal", "European J of Stroke"
- Past president of the Hungarian Stroke Society,
- Corresp. member of Deutsche Gesellschaft für Klinische Neurophysiologie und Funktionelle Bildgebung
- Between 2009 and 2013 he was the president of European Society of Neurosonology and Cerebral Hemodynamics.
- Since 2015 he is the president of Hungarian Neurological Society.
- Since 2016 corresponding member of Hungarian Academy of Sciences.
- He was awarded with the prize of European Stroke Conference, Eur. Neuroson. Soc. Cer. Hemodyn., Batthyány-Strattmann Prize (Ministry of Health), Francis Crick Award, Szentgyörgyi Award (Ministry of Health) and Lazarevics prize (Serbian Neurol Soc) for his activity in stroke care, education and research.
- His students elected him 7x "Teacher of the Year" and the "Faculty prize" has been donated him due to his outstanding educational activity.
- The President of Hungarian Republic awarded him the Knight's Cross of Republic (for outstanding educational and clinical work).
- His department hosted two times the Stroke Summer Course of the European Stroke Organisation.
- He has published 250 papers on stroke, stroke risk disease, neurosonology and arteriosclerosis.
- His book ("Dissect me, please") had two Hungarian editions and has been translated on German, English, Rumanian and Serbian language.

DAFIN F. MUREȘANU
ROMANIA

Professor of Neurology, Senior Neurologist, Chairman of the Neurosciences Department, Faculty of Medicine, "Iuliu Hatieganu" University of Medicine and Pharmacy Cluj-Napoca, President of the European Federation of Neurorehabilitation Societies (EFNR), Co-Chair EAN Scientific Panel Neurorehabilitation, Past President of the Romanian Society of Neurology, President of the Society for the Study of Neuroprotection and Neuroplasticity (SSNN), member of the Academy of Medical Sciences, Romania, secretary of its Cluj Branch. He is member of 17 scientific international societies (being member of the American Neurological Association (ANA) - Fellow of ANA (FANA) since 2012) and 10 national ones, being part of the executive board of most of these societies. Professor Dafin F. Muresanu is a specialist in Leadership and Management of Research and Health Care Systems (specialization in Management and Leadership, Arthur Anderson Institute, Illinois, USA, 1998 and several international courses and training stages in Neurology, research, management and leadership). Professor Dafin F. Muresanu is coordinator in international educational programs of European Master (i.e. European Master in Stroke Medicine, University of Krems), organizer and co-organizer of many educational projects: European and international schools and courses (International School of Neurology, European Stroke Organisation summer School, Danubian Neurological Society Teaching Courses, Seminars - Department of Neurosciences, European Teaching Courses on Neurorehabilitation) and scientific events: congresses, conferences, symposia (International Congresses of the Society for the Study of Neuroprotection and Neuroplasticity (SSNN), International Association of Neurorestoratology (IANR) & Global College for Neuroprotection and Neuroregeneration (GCNN) Conferences, Vascular Dementia Congresses (VaD), World Congresses on Controversies in Neurology (CONy), Danube Society Neurology Congresses, World Academy for Multidisciplinary Neurotraumatolgy (AMN) Congresses, Congresses of European Society for Clinical Neuropharmacology, European Congresses of Neurorehabilitation). His activity includes involvement in many national and international clinical studies and research projects, over 400 scientific participations as "invited speaker" in national and international scientific events, a significant portfolio of scientific articles (190 papers indexed on Web of Science-ISI, H-index: 20) as well as contributions in monographs and books published by prestigious international publishing houses. Prof. Dr. Dafin F. Muresanu has been honoured with: „Dimitrie Cantemir" Medal of the Academy of The Republic of Moldova in 2018, Ana Aslan Award 2018 - "Performance in the study of active aging and neuroscience", for the contribution to the development of Romanian medicine, National Order "Faithful Service" awarded by the President of Romania in 2017; "Iuliu Hatieganu" University of Medicine and Pharmacy Cluj-Napoca, Faculty of Medicine, the "Iuliu Hatieganu Great Award 2016" for the best educational project in the last five years; the Academy of Romanian Scientists, "Carol Davila Award for Medical Sciences / 2011", for the contribution to the Neurosurgery book "Tratat de Neurochirurgie" (vol.2), Editura Medicala, Bucuresti, 2011; the Faculty of Medicine, "Iuliu Hatieganu" University of Medicine and Pharmacy Cluj-Napoca "Octavian Fodor Award" for the best scientific activity of the year 2010 and the 2009 Romanian Academy "Gheorghe Marinescu Award" for advanced contributions in Neuroprotection and Neuroplasticity.

ABSTRACTS

MANAGEMENT OF CAROTID STENOSIS CEA VS. STENT

NATAN BORNSTEIN
Director of Neurological Division, Sackler school of Medicine, Tel-Aviv University, Israel

Symptomatic severe carotid stenosis (>70%) carries a high risk of subsequent stroke of about ~ 30% over 2 years. Carotid endarterectomy (CEA) was proved to reduce the risk of stroke significantly, with Relative Risk Reduction (RRR) = 65% and Number Needed to Treat (NNT) = 6 if performed safely (perioperative S&D =5.8%) and should be executed within 2 weeks of TIA or minor stroke (NASCET & ECST). For carotid stenting to replace CEA we need to know the comparative safety, durability and efficacy of the procedure. Only a few randomized, controlled studies comparing CEA and stenting were conducted (CAVATAS, SAPPHIRE, EVA-3 and SPACE) with inconclusive results. There are still several ongoing studies (CREST in the USA and ICSS in Europe and Australia). Until more data will be available carotid stenting should be performed only in a selected group of patients with specific indications like: re-stenosis of the CEA, post neck radiation, inaccessible lesion for CEA and contra-indications for CEA.

SECONDARY STROKE PREVENTION

NATAN BORNSTEIN
Director of Neurological Division, Sackler school of Medicine, Tel-Aviv University, Israel

Patients with TIA or ischemic stroke carry a risk of recurrent stroke between 5 and 20% per year. In patients with TIA or ischemic stroke of noncardiac origin antiplatelet drugs are able to decrease the risk of stroke by 11-15% and the risk of stroke, MI and vascular death by 15-22%. Aspirin is the most widely used drug. It is affordable and eff ective. Low doses of 50-325 mg aspirin are as eff ective as high doses and cause less gastrointestinal side effects. Severe bleeding complications are dose-dependent. The combination of aspirin with slow release dipyridamole is superior to aspirin alone for stroke prevention (ESPS-2 and ESPRIT1). Both studies have shown approximately 20%-24% relative risk reduction (RRR) of stroke and death. Clopidgrel is superior to aspirin in patients at high risk of recurrence by about 8.7% RRR (CAPRIE2). The combination of aspirin plus clopidogrel is not more eff ective than clopidogrel alone but carries a higher bleeding risk (MATCH3 and CHARISMA4). None of the antiplatelet agents is able to signifi cantly reduce mortality. The recent results of the PRoFESS trial 5, 6 showed no diff erence between clopidogrel and aspirin with slow release dipyridamole in secondary stroke prevention.

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2. Lancet 1996;348:1392-1339
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4. N Eng J Med 2006;354(16):1744-6
5. Cerebrovasc Dis 2007;23:368-380
6. N Engl J Med 2008;359:1238-51

TIME IS BRAIN, TIA AS AN EMERGENCY

NATAN BORNSTEIN
Director of Neurological Division, Sackler school of Medicine, Tel-Aviv University, Israel

Transient Ischemic Attack (TIA) should be considered as an emergency and work-up has to be done within 24 hours like acute unstable angina pectoris. It is known that about 23% of stroke are preceded by TIA.Several studies have shown that the risk of subsequent stroke in the fi rst 2 weeks after a TIA is about 1% per day. In 2 published well conducted studies, EXPRESS (P. Rothwell) and SOS_TIA (P. Amarenco) it was shown that very early management in a TIA clinic will reduce the risk of subsequent stroke by 80% at 3 months. Therefore, work-up evaluation has to be performed with in 24 hours in a dedicated organized structure. Several stroke registries reported that carotid stenosis is the cause of embolic stroke in about 25%-30% of all ischemic strokes. Current guidelines recommend immediate intervention either by carotid endarterectomy (CEA) or stenting (CAS) in patients with symptomatic carotid stenosis greater than 50%. Carotid duplex is a reliable, non-invasive, accessible tool for evaluation of carotid stenosis with very high level of accuracy. Therefore, carotid duplex should be the fi rst line tool for rapid evaluation of every patient with TIA in order to detect a potential treatable carotid stenosis for stroke prevention. It is recommended to establish an "Acute TIA clinic" equipped with immediate accessible Duplex device to enable rapid evaluation of the carotid system in order to detect potential treatable carotid stenosis

POST STROKE DEPRESSION

NATAN BORNSTEIN
Director of Neurological Division, Sackler school of Medicine, Tel-Aviv University, Israel

Stroke is a major cause of long-term physical, cognitive, emotional and behavioral disability. There is poor recognition of the emotional burden after stroke. Depression is abnormal and considered as "emotional distress". Post -stroke Depression (PSD) is the most frequent non-cognitive neuropsychiatric complication affecting up to a third of all ischemic stroke patients. PSD is associated with increased mortality, poorer functional recovery and lower quality of life. Despite its great clinical relevance the relationship between stroke, depression and cognitive impairment remains relatively unexplained and the awareness of . The potential mechanisms of PSD are either neuroanatomical caused by lesions in the frontal areas, or directly affecting neural circuits involving mood regulation, or as a result of psychological adjustment required by the disease. There is controversy regarding the appropriateness of diagnosing depression in the setting of an acute stroke. Geriatric Depression Scale (GDS) is the most widely used.
Regarding treatment there is insufficient randomized evidence to support the routine use of antidepressants for the prevention of depression or to improve recovery from stroke.
The approaches to management should be multidisciplinary including nurses and allied health staff.

EFFICACY OF STROKE UNITS

MICHAEL BRAININ
Clinical Neurology Danube University Krems, Austria

Organised stroke unit care is a form of care provided in hospital by nurses, doctors and therapists who specialise in looking after stroke patients and work as a co-ordinated team. An updated systematic review has confirmed significant reductions in death (3% absolute reduction), dependency (5% increase in independent survivors) and the need for institutional care (2% reduction) for patients treated in a stroke unit, compared with those treated in general wards. All types of patients, irrespective of gender, age, stroke subtype and stroke severity, appear to benefit from treatment in stroke units. These results have been confirmed in large observational studies of routine practice. Stroke units may also improve patients' quality of life, and improvements in outcome may persist for several years. Of available therapies in the acute phase of stroke (antiplatelet therapy, intravenous thrombolysis, stroke unit care), stroke unit care has the overall largest benefit because this principle of care may potentially be applied to all patients with acute stroke.

The core components of stroke unit care include

- Rapid medical assessment and diagnosis, and early assessment of nursing and therapy needs
- early management, consisting of early mobilization, prevention of complications, and treatment of hypoxia, hyperglycaemia, pyrexia and dehydration
- ongoing rehabilitation, involving coordinated multidisciplinary team care, and early assessment of needs after discharge.

Making an early diagnosis of stroke is crucial because a time-dependent deterioration occurs that is caused by oxygen depletion in the neural tissue that shows ongoing compromise of blood-flow. Without intervention this compromised area of the brain will develop into an infarct and cannot be rescued. This critical time, which enables us to perform recanalisation and reperfusion therapy is called therapeutic time window. If one quantifies the time factor of ischemia it has been estimated that up to two million neurons will be lost per minute which amounts to more than 30.000 neurons per second. Thus, it is important to recognize stroke as an emergency. Persons with stroke should be hospitalized and treated as soon as possible. In many countries there is a recommended chain of recovery which includes firstly the recognition of stroke, then the reaction towards stroke, then the response, the reveal and the treatment.

In some regions of the world these transport systems are well developed and the ambulance personnel regularly receives special training. Once the patient arrives in the emergency department it should be clear that an urgent triage and a priority code should be assigned to a stroke patient. Priority includes the setting up of an IV line, measuring blood glucose, performing routine biochemistry including blood count and performing standard ECG. Trained medical personal should perform an accurate clinical diagnosis and exclude mimics. Under ideal circumstances, the stroke team should be notified before the arrival of the patient and urgent clarification of the diagnosis preferably by usage of brain imaging as soon as possible should be thought for.

Thrombolytic therapy should be used by personnel trained in its use in a centre equipped to investigate and monitor patients appropriately. Currently thrombolysis is only approved for treatment within 4.5 hours of symptom onset. Thrombolysis requires admission of stroke to hospital and it cannot easily be given in small local hospitals. More recently, endovascular thrombectomy has become standard treatment for large thromboses in the M1 or 2 segment of the ACM which usually causes severe strokes with NIHSS values of 15 or more. This therapy can only be applied within 6 hours of onset and must be performed in specialized comprehensive stroke centers.

In the acute phase, aspirin is associated with a very significant reduction in acute ischemic strokes, as well as deaths (of any cause) and the combined end-point of death and further strokes. There is no significant access of intracerebral hemorrhages. Subgroup analyses showed that aspirin was beneficial in all types of ischemic strokes irrespective of age and gender. For every 1000 patients treated aspirin treatment avoids 9 deaths or stroke in the acute phase, 12 death and dependency, and an extra 10 patients make a complete recovery. Consequently, prompt treatment with aspirin should be considered for almost all patients presenting with suspected acute ischemic stroke.

Strategies to prevent further strokes should be initiated already when the patient is under early treatment for a first stroke. All patients with stroke (ischemic, hemorrhagic, and stroke of unknown cause) will benefit from modification of life style changes, in particular cessation of smoking, and blood pressure reduction with a diuretic and an ACE-inhibitor. Blood pressure reduction should not be started until after the acute phase.

Patients with ischemic stroke benefit from long-term use of antiplatelet therapy as well as from a statin if total cholesterol is >3.5 mmol/liter.

The structure and process quality of stroke units include that there is a seamless and constant observation of vital parameters including blood pressure, heart rate, temperature, breathing and other parameters. This adds to the direct observation of the patient by trained personnel to notice early changes in the state of consciousness, to recognize epileptic fits and extracerebral causes of clinical deterioration.

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FUNCTIONAL ANATOMY OF THE BRAIN

MICHAEL BRAININ
Clinical Neurology Danube University Krems, Austria

Localization of function within the brain was the starting point for clinical neurology. Only with Brodman the cartography of the cerebral cortex based on clinical observation and experiments had a lasting effect and was accepted as the basis of localization of function of the cortex. Controversies among great minds of neurology took place and had to wait for pathological confirmation which often took years. The dichotomy of localization and function was united in a model by Luria who laid the foundations of modern neuroplasticity. Neuroimaging enabled neurologists and neurosurgeons to localize function much more easily and speculations about functional models could be put to a test. Tailarach and his school in Marseille founded stereotactic localisation and laid this down in his famous atlas which allowed the exact location pinpointed in the depth of the hemispheres and enabled comparability among humans. Later on, based on modern imaging, brain atlases were developed to guide the clinician and researcher on the transversal, coronary, or sagittal cuts of CT and MRI images. Among the most notable ones was the CT/MRI Atlas of Hanna and Antonio Damasio developed as a guide for vessel anatomy and cortical Brodman areas, This is especially helpful for analysis of small groups of patients who have similar lesions or similar clinical deficits and can used in a semiquantitative way. Finally, Marsel Mesulam from Boston proposed a functional model of the human cortex which allows interpretation of function in health and disease alike. This combined usefulness allows the interpretation of cortical syndromes as disconnection disturbances and explains most neuropsychological syndromes on the basis of disconnected localisation of function, either intra- or interhemispheric. New methods of imaging such as fibre tract imaging or functional MRI confirm these models and visualize such disconnections. Several examples will be given, including the neuroanatomical basis of problem solving, the working mind of a calculating prodigy, or the neural basis of frontal lobe dysfunctions.

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POST-STROKE COGNITIVE DECLINE: INTERVENTION TRIALS FOR PREVENTION AND TREATMENT

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Previous randomized trials aiming at promoting recovery after stroke such as with levodopa, natural biologicals (Cerebrolysin) or SSRI's have been successful in showing improvement of motor recovery. But currently no established treatment exists for the preservation or restoration of cognitive status following stroke. Given the high frequency of delayed onset of cognitive deterioration following stroke it is surprising that large studies have yet to be performed. Single or combined drug interventions tested up to now were based on secondary outcome analyses and included antihypertensive drugs which showed only a modest effect on cognition in general and no consistent effect was shown for lipid lowering drugs. Combination of antiplatelet drugs have been tested in the SPS3 trial but showed no effect on cognitive outcomes. Life-style interventions include studies of a Mediterranean diet with extra virgin olive oil and nuts but while stroke occurrence can be reduced, no data on post-stroke cognition exist. The same applies for physical exercise programs which show good effects on physical fitness.

Ongoing registered stroke testing either drug and/or lifestyle interventions all are planned either for small sample sizes and /or a complex endpoint or combination of endpoints that are not likely to produce practice-changing results.
Multi-domain intervention studies are much more likely to be effective on cognition because they perform multiple risk factor management with lifestyle adaptation including diet changes with increase of drug compliance and adherence. Intensifying these interventions and to monitor them is crucial. The first comprehensive multi-domain intervention trial (ASPIS) has recently been terminated. The primary endpoint was a significant change of the z-score of 5 neuropsychologically assessed cognitive domains. While the overall result was neutral, a signal for change of dysexecutive function was seen and follow-up studies might have to consider this finding.

In the future, there is a need for including cognitive outcome measurements in all trials targeting the brain, to consider larger sample sizes, to harmonize assessment strategies, to focus on a high risk population, and to include biomarkers and imaging data for confirmatory analyses. Overall, it is crucial to aim for intervention intensities that create significant group differences.

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THE GLOBAL STROKE EPIDEMIC: PREVENTION IS THE MAIN ISSUE

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Today, there is an increase in stroke mortality which is most dramatic in low and middle income countries. If we include prevalence rates and the overall burden of the disease, dementia and stroke combined are by far the most burdening diseases globally. Moreover, in countries with aging populations the increase is also seen due to demographic changes.

More recently, several studies have shown that a decrease of incidence rates is possible by improving modifiable risk factors, mostly of life style. For example, The Global Burden of Disease Study and the Interstroke Study both report that the burden of stroke is strongly influenced by modifiable risk factors and up to 90% of stroke occurrence can be explained by these risk factors. Conversely, a major reduction of incidence might be expected if behavioral and metabolic risk factors are managed appropriately. Recently, environmental factors (indoor and outdoor air pollution and lead exposure) have been recognized as major risks. Air pollution alone explains 30% of the stroke risk burden globally.

Prevention on a population scale can only be effective if large programs are established that target not only high-risk persons but aim also at medium risk and low risk persons. The WHO led initiative of reducing the NCDs (non-communicating diseases such as heart disease, cancer, diabetes, stroke and cardiopulmonary disease) can only become effective if the prevention issues are carried across diseases and are not only focused on one specific illness. This NCD Alliance has published a WHO Global Action Plan 2013-2020 which aims at reducing the NCD burden by 30% in 2030 (30 by 30). Regional assessments of the effectiveness of such initiatives show that in some world regions this may be reached but in others the targets will be missed if additional efforts are not made.

NEUROSONOLOGICAL METHODS IN PHARMA TRIALS

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Age, hypertension, hyperlipidemia, smoking, alcohol influence the IMT (should be less than 1 mm in healthy persons). It was proven with prospective trials, that the IMT could be decreased by appropriate treatment. The positive effect of statins, antihypertensive drugs have been proven. Some statins and antihypertensive drugs resulted in significant decrease of IMT thickness after one or 2 years therapy while others did not have beneficial effect. Besides, the majority of trials detected significant positive correlation between the reversal of IMT and the risk of vascular events. The advantages of CIMT are: a./ measures the effect of a new drug onto the IMT, b./ the results of a CIMT trial (needs much shorter time than a morbidity-mortality trial) may be decisive to start or refute a long-lasting and expensive morbidity trial on drugs aimed on atherosclerosis.

The transcranial Doppler (TCD) is also useful for evaluation of the efficacy of pharmacotherapy not only in subarachnoidal bleeding but also in ischemic stroke risk patients. The microembolic signals are accepted surrogate marker for future stroke risk and have been used to show treatment efficacy in different clinical conditions (TIA, carotid stenosis, carotid endarterectomy, coronary bypass surgery and during some intravascular interventions). The randomized trials successfully evaluated the effect of different anticoagulants (e.g. heparin vs. LMWH), mono vs dual antiplatelet therapy in stroke or stroke risk patients.

With the help of continuous monitoring of cerebral blood velocity (e.g.MCA), you can evaluate the efficacy of t-PA and new fibrinolytic agents in acute ischemic stroke patients.

The impairment of dilatative capacity of brain arteriolas can be also measured noninvasively, without radiation hazard, by transcranial Doppler. The positive or negative effect of different pharmacotherapies (statins, antihypertensives, antiparkinson drugs, NSAIDS, diuretics etc.) on cerebral vasomotor reactivity could be estimated.

Conclusion: both extra-and intracranial US methods proved their usefulness in the evaluation of pharmacological interventions. They are attractive techniques due to their low cost, non-invasive characteristics, excellent time resolution and unique abilities (e.g. embolus detection, continuous non-invasive cerebral blood velocity monitoring).

BEDSIDE DIFFERENTIAL DIAGNOSIS OF ACUTE DISTURBANCES OF HYPNOID TYPE OF CONSCIOUSNESS

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1. Introduction

This lecure discusses the hypnoid (sleep-like) types of disturbances of consciousness. This patient has closed eyes similar to a sleeping healthy person. The vegetative state will be not discussed.

2. The severity of hypnoid types of disturbances of consciousness (HDC).

This large group of consciousness disturbances does not involve only the coma, but it contains also the state of somnolence and stupor too. All patients suffering from any type of hypnoid unconsciousness are alike sleeping people, who are healthy otherwise. If not provoked by pain or any other stimulus, the patient is lying with his/her eyes closed.
Somnolence: the patient can be woken up easily, but sleeps away right after being left alone.
Stupor (also known as sopor): the patient seems to be sleeping abnormally deeply, but reflects with motor or verbal responses for motor or verbal stimuli.
Coma (from a greek word meaning deep sleep): The patient does not react for strong pain stimuli, but both the spontaneous breathing and circulation are intact.

In the everyday practice, the terms somnolent-stuporous and stuporo-comatose are also widely used for indicating the severity, but cannot be used for quantitative assessments. Instead, we use different coma scales to record the level of consciousness. The lowest possible score of Glasgow Coma scale 3, while the highest is 15 (fully awake person).

Table 1. The Glasgow Coma Scale

Eye-opening	Spontaneous	4
	In response to voice	3
	In response to painful stimuli	2
	No response	1
Motor response	Obeying command	6
	Localizing response	5
	Withdraws	4
	Abnormal flexor response	3
	Extensor posturing	2
	No response	1

Verbal response	Oriented	5
	Confused conversation	4
	Inappropriate speech	3
	Incomprehensible speech	2
	No verbal response	1

A recommendation published in 2010 compared the scales, which are commonly used to assess the disorders of consciousness. The use of Coma Recovery Scale-Revised (Table 3) is recommended with minimal limitations. The total score range is between 0-23, and the assessment takes 25 minutes.

Table 2. The most frequently used scales in the assessment of consciousness disturbances

CRS-R Coma Recovery Scale-Revised
CLOCS Comprehensive Levels of Consciousness Scale
CNC Coma/Near-Coma Scale
FOUR Full Outline of UnResponsiveness Score
GCS Glasgow Coma Scale
GLS Glasgow-Liege Coma Scale
INNS Innsbruck Coma Scale
LOEW Loewenstein Communication Scale
RLS85 Swedish Reaction Level Scale-1985
SMART Sensory Modality Assessment Technique
SSAM Sensory Stimulation Assessment Measure
WHIM Wessex Head Injury Matrix
WNSSP Western Neuro Sensory Stimulation Profile

Table 3. The Coma Recovery Scale-Revised

1. Auditory function scale
    4 Consistent movement to command
    3 Reproducible movement to command
    2 Localization to sound
    1 Auditory startle
    0 None

2. Visual function scale
    5 Object recognition
    4 Object localization: reaching
    3 Visual pursuit
    2 Fixation
    1 Visual startle
    0 None

3. Motor function scale
    6 Functional object use
    5 Automatic motor response
    4 Object manipulation
    3 Localization to noxious stimulation
    2 Flexion withdrawal
    1 Abnormal posturing
    0 None/Flaccid

4. Oromotor/Verbal function scale
    3 Intelligible verbalization
    2 Vocalization/Oral movement
    1 Oral reflexive movement
    0 None

5. Communication scale
    2 Functional: accurate
    1 Non-functional: intentional
    0 None

6. Arousal scale
    3 Attention
    2 Eye opening without stimulation
    1 Eye opening with stimulation
    0 Unarousable

3. The examination of patients suffering from HDC

The reliable data on medical history and risk factors are very important.
Information should be collected not just about the previous diseases, such as hypertension, diabetes, cardiovascular or hematological disorders, depression, fever, but the medications too. Life habits are very important (medication? alcohol/drug consumption? problems in the private life or at work?).

It should be questioned if the family members experienced any change in the patient's behavior, difficulty in his/her verbal communication and self-expression, or the signs of lightheadedness. Did the patient complain about numbness, visual problems or double vision? Did he/she experience any trauma? Painful stimuli should be used to assess the severity of the consciousness disorder.

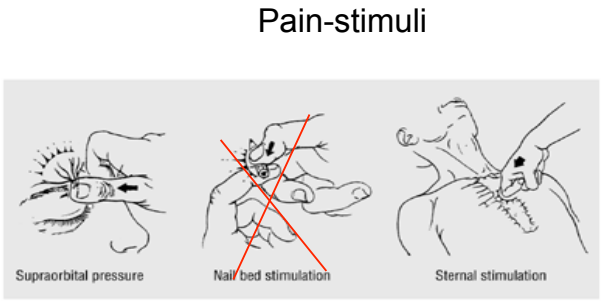

Figure 1. Painful stimuli applied either on the supraorbital area, sternum or on both mastoid processes. The symmetrical or asymmetrical movements of the extremities can be assessed.

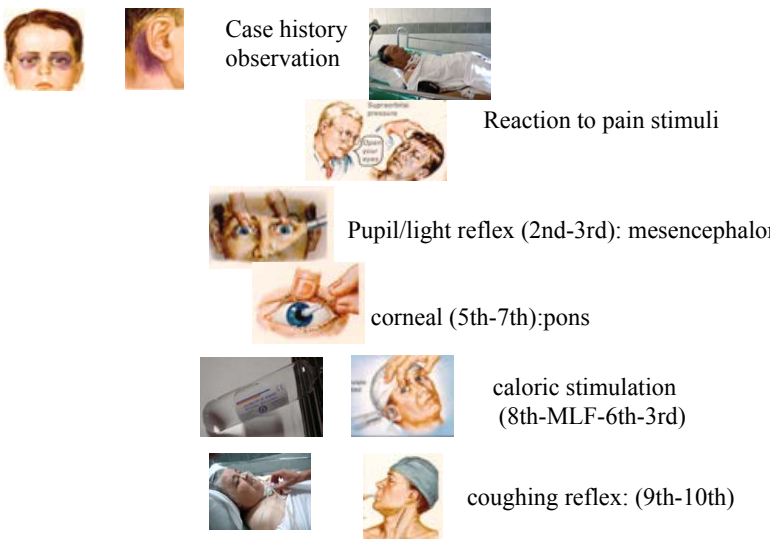

Figure 2. The most important steps of neurological examination in HDC.

4. Bedside differential diagnosis of patients suffering from acute HDC.

The normal conscious state requires the intact function of the reticular formation located in the brainstem and the intact function of the supratentorial region activated by the reticular formation. These facts help us to understand the differential diagnosis of consciousness disorders. Many agents can harm the supratentorial region directly or indirectly, but an isolated brainstem lesion can also cause coma (e.g. occlusion of the basilar artery affecting the brainstem reticular formation).

It is essential to understand that whatever damages the supratentorial region, HDC develops only if the acute pathological process has diffuse/extended effect on the supratentorial area.
(Accepting this postulate it is easy to understand why an acute viral encephalitis or barbiturate intoxication will result in disturbance of consciousness while a large territorial cerebral infarct without mass effect will not alter the consciousness).
With other words: an acute supratentorial focal ischemia or bleeding without mass effect (without midline shift, without increased pressure etc.) will NOT result in HDC, but an acute event with space occupying effect can be accompanied with HCD. Last but not least, all of our statements are valid only for acute events!
A slowly growing tumor or subdural hematoma does not necessarily result in disturbance of consciousness except if the CT reveals space occupying/mass effect with midline shift etc.

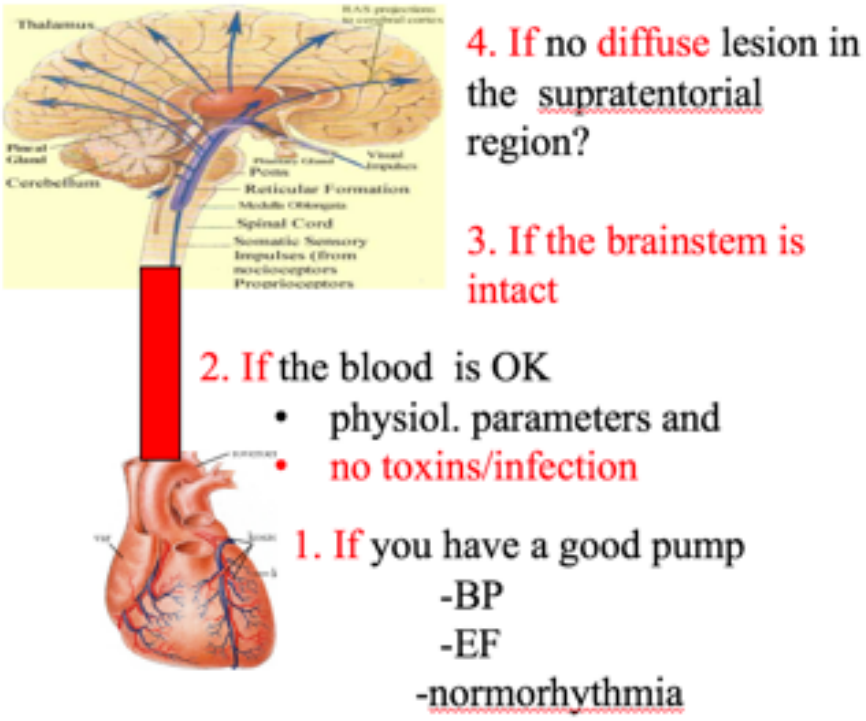

Figure 3. Acute external and internal factors (four groups) resulting in hypnoid type of disturbance of unconsciousness. You are alert if..

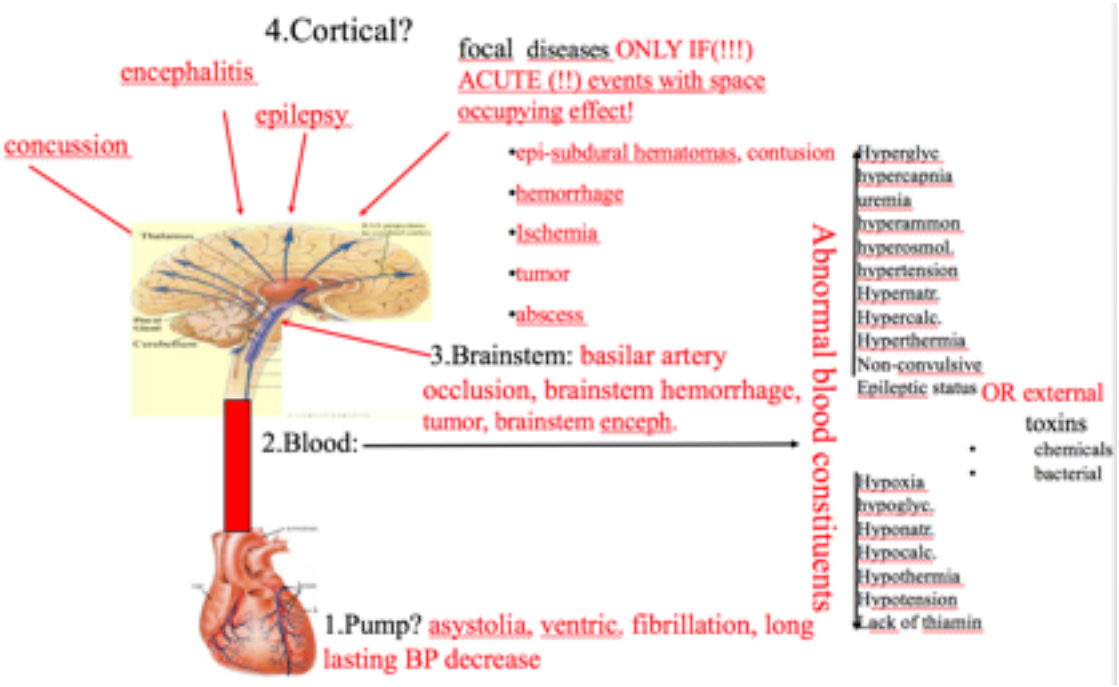

Fig.4 The most common diseases resulting in HDC

The therapy depends on the causative agent, so we do not provide further details about it. In case of basilar occlusion, desobliteration is indicated, if intoxication, detoxification etc.
Important! If CT or MRI reveals ischemia or hemorrhage without any mass effect, and the patient has normal conscious state, but hours later he/she shows the signs of any consciousness disturbances, such as somnolence, the hemorrhage is likely to have increased suddenly, or malignant ischemic cerebral edema could have developed, especially if the patient has normal laboratory result, did not experience fever, and did not get any sedatives. In these cases imaging should be performed again!

4. Prognosis

The prognosis depends on the causative agent, the patient's age and general condition. According to the survey of the American Academy of Neurology, patients suffering from coma caused by cardiac arrest (hypoxia and ischemia) are likely to have poor prognosis after 3 days, if any of the followings is present:

- absent pupillary, corneal, coughing reflexes, absent reaction to caloric reflex test,
- absent or extensor motor responses (evidence A), the presence of seizures or myoclonus status epilepticus (evidence B),
- Bilateral absence of the cortical component of the SSEP predicts a poor outcome (evidence B)
- burst-suppression pattern or generalized epileptiform activity are associated with poor outcome (evidence C).
- serum neuron-specific enolase (NSE, only this marker has verified diagnostic value!) levels >33 µg/L at days 1 to 3 predict poor outcome (evidence B),
- There are insufficient data to support or refute whether the measurement of intracranial pressure, the oxygenisation of the brain, and neuroimaging (MRI, CT)are indicative of poor outcome or not.

5. Summary

In case of consciousness disturbances the following examinations are essential:
1. Analysis of the detailed medical history including the information obtained from family members.
2. General physical examination, neurological examination, and the examination of external injuries, including:
   - responses to painful stimuli,
   - the isocoria of the pupils, eye positions and movements, the presence or absence of pupillary, corneal, and coughing reflexes,
3. The treatment of consciousness disturbances can be operative or conservative depending on the causative agents.

BRAIN DEATH

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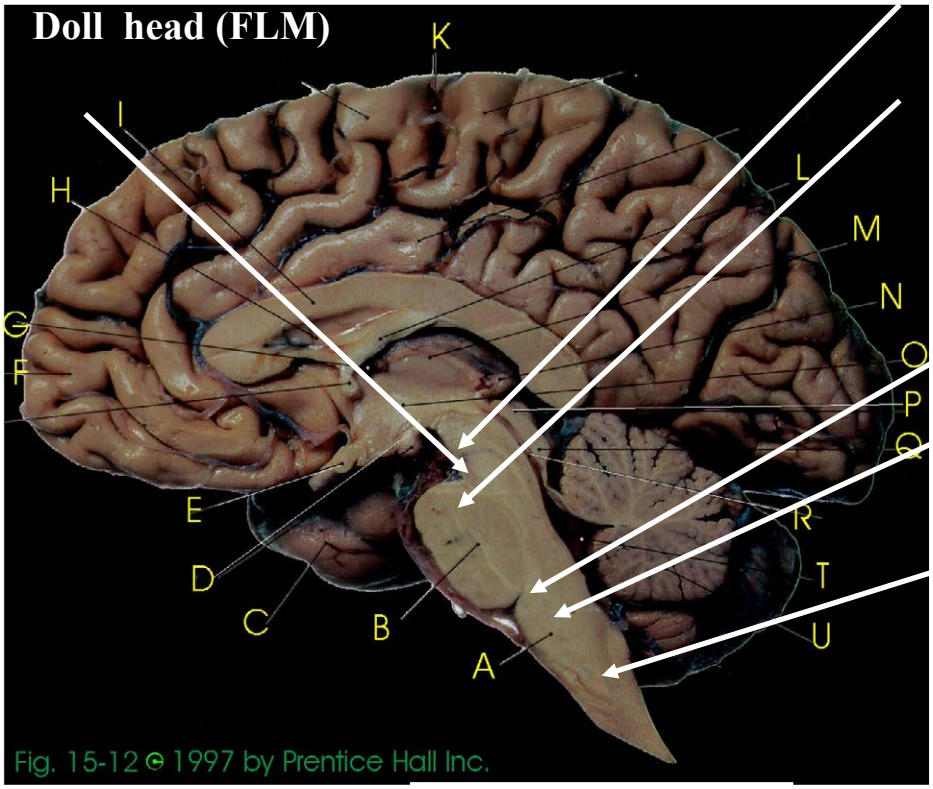
MOST IMPORTANT ASPECTS
- no exclusion criteria (e.g. hypothermia, intoxication)
- lack of all brain activity
- irreversibility

EXCLUSION CRITERIA
- intoxication
- medication? (4x half time)
- neuromuscular blockade
- shock
- metabolic or endocrine coma
- hypothermia (bellow 35 °C)
- infectious CNS disease

CONFIRMATION OF BRAIN DEATH
- Based on clinical investigations and disease course
- Ancillary instrumental investigations

BRAIN DEATH=BRAINSTEM DEATH



Pupil, light reaction

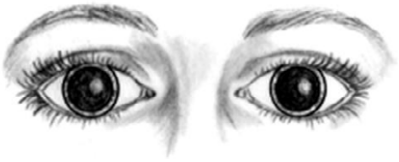
Corneal reflex (V,VII)

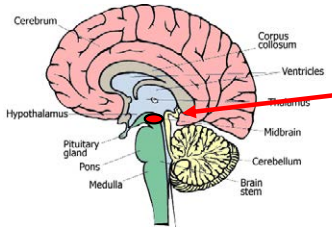
vestib. reaction

Coughing test

Spontaneous breath (apnoe test)

DILATED PUPILS





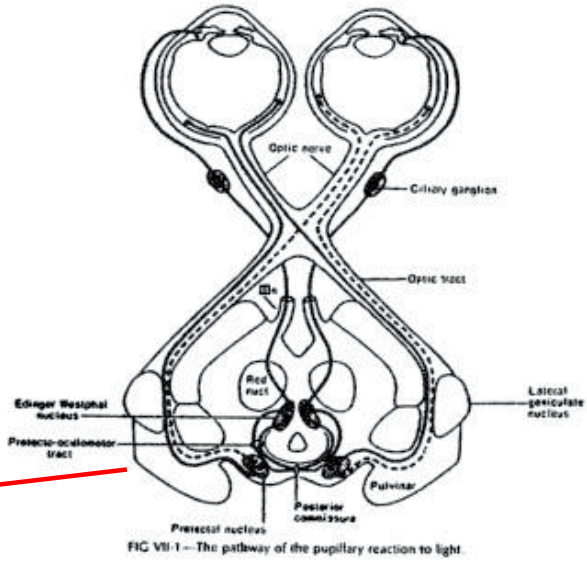
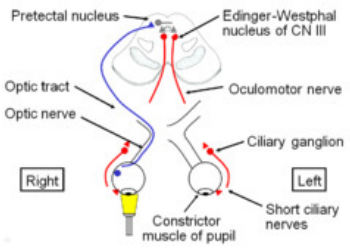


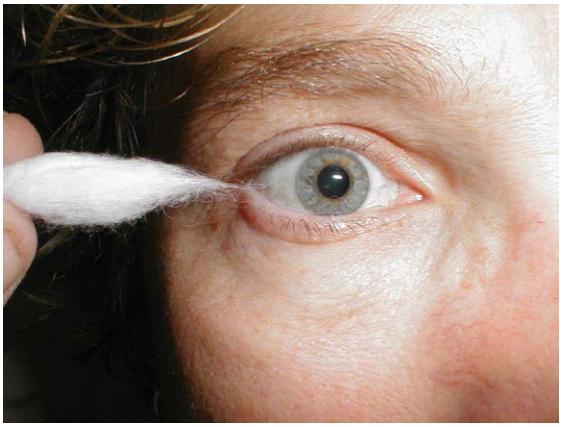
FIG VII-1—The pathway of the pupillary reaction to light.

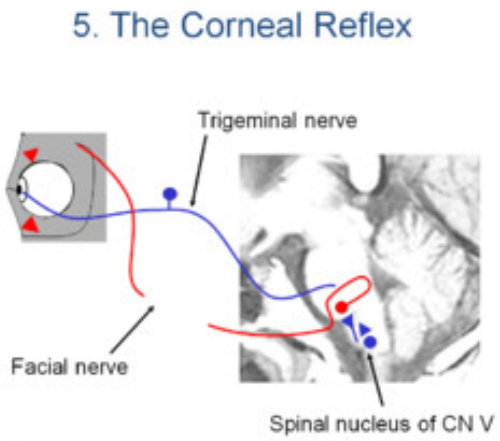
PUPILS

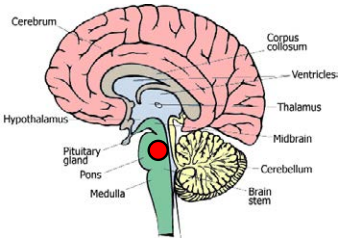
- dilated,
- no light reaction
- misleading:
  - shrinkage of cornea
  - surgery



BILATERAL LACK OF CORNEAL REFLEX

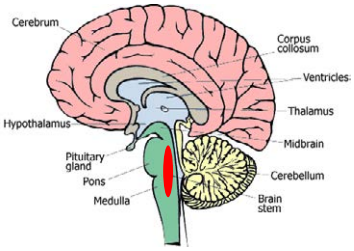






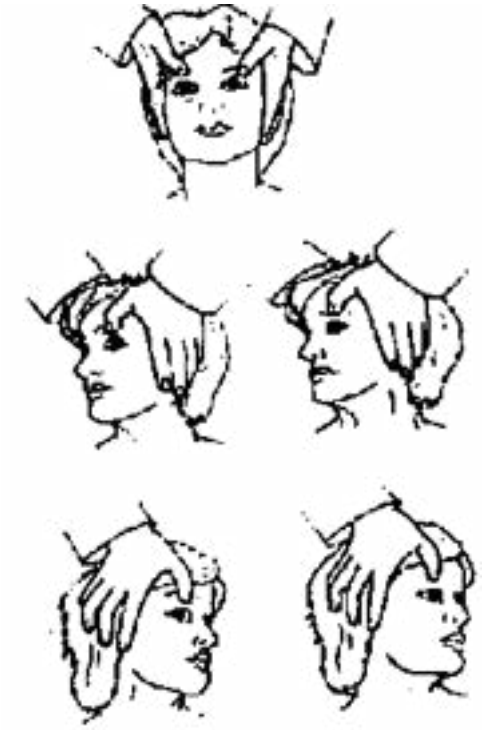
LACK OF PAIN REACTION (TRIGEMINAL STIMULATION)

- grimace
- palpebral closing
- moving
- increased pulse or BP



DOLL HEAD PHENOMEN

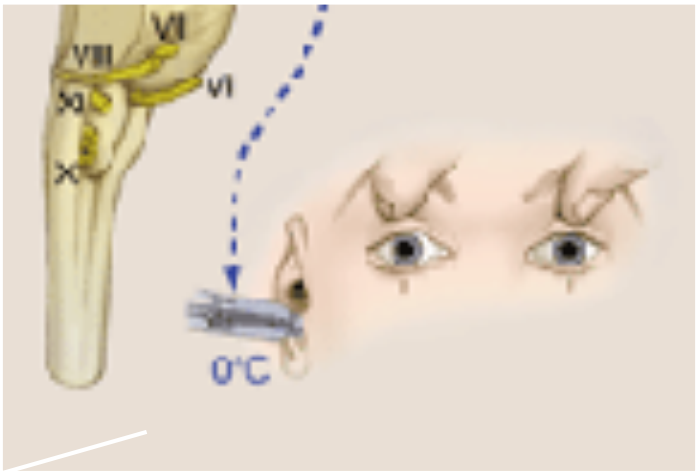
- Control:sudden head movement results in contralateral compensatory movement

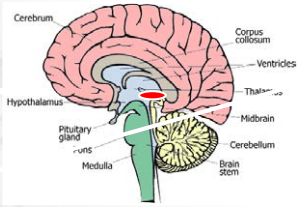


- Brain death:the eyes passively follow the head-movements

VESTIBULO-OCULAR (CALORIC) STIMULATION

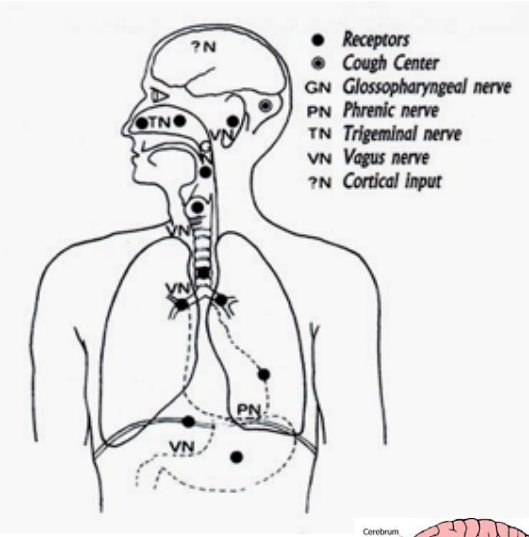
- ice water or chlorethyl
- control:ipsilateral slow deviation of both eyes and quick jumping to contralateral side
- brain death:no movement
- 5 min between sides

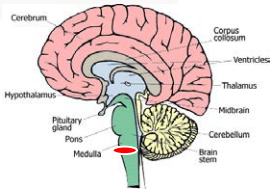




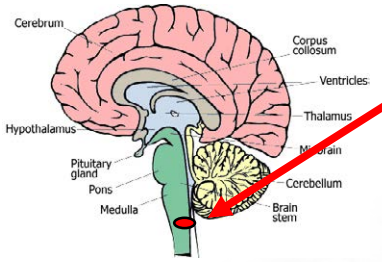
COUGHING REFLEX

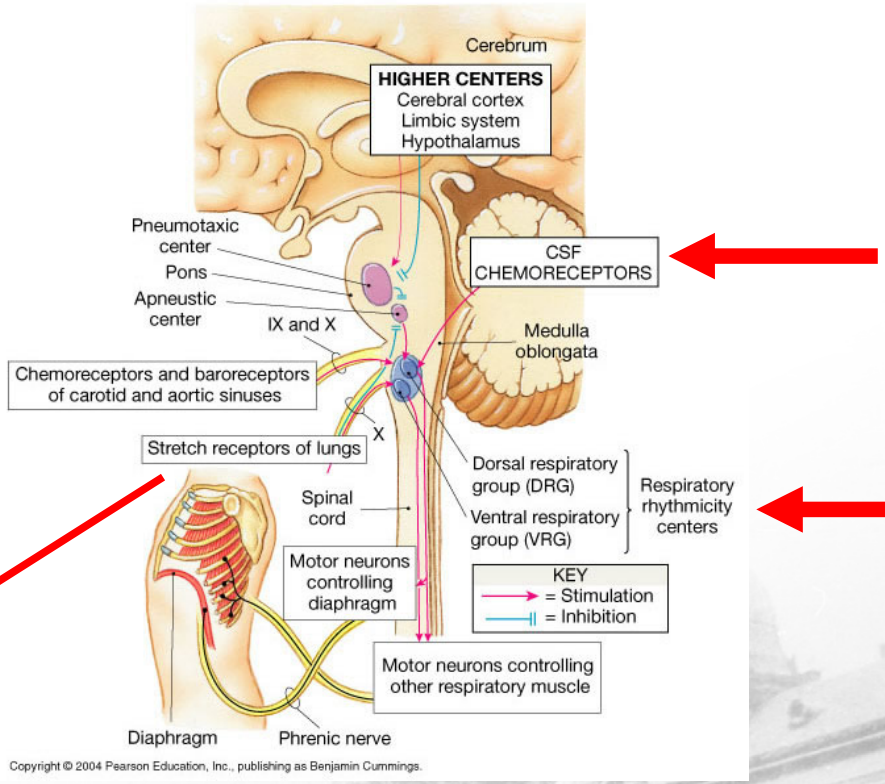
- tube movement
- deep suction
  - coughing?
  - pulse or BP changes?





LACK OF SPONT. BREATHING:APNOE TEST





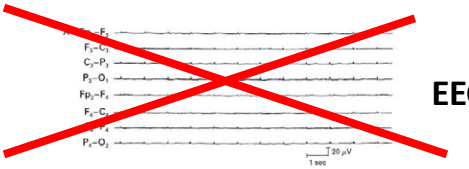
APNOE TEST

- arterial canule
- 1,0 Fi02 ventilation (10 min)
- removal from artef. ventil. 6 1/min 02 into tube
- observation of chest
- repeated arterial blood gas samples
- brain death: if arterial pC02 higher then 60 Hgmm but no return of spontaneous breath
- then back to artef. ventil.

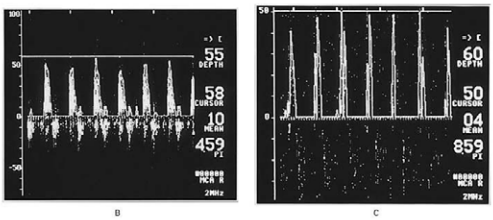
IRREVERSIBLE

- observation of disease course:permanent lack of reflexes/reactions
  - if elder than 3 y and primary brain damage: 12 h
  - secondary brain damage 72 h
  - from 5 weeks till 3 y in both cases 24 h
  - neonates:in both cases 72 h
- or:clinical investigations+ ancillary instruments: no follow up time

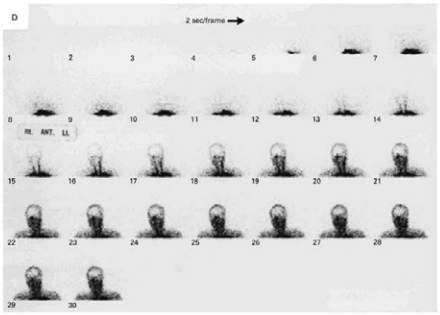
ANCILLARY METHODS



EEG

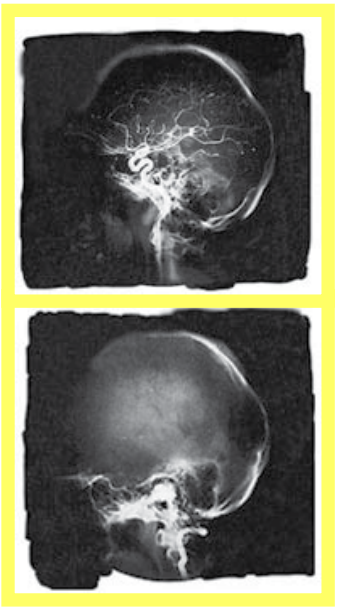


TCD



Brain scintigraphy

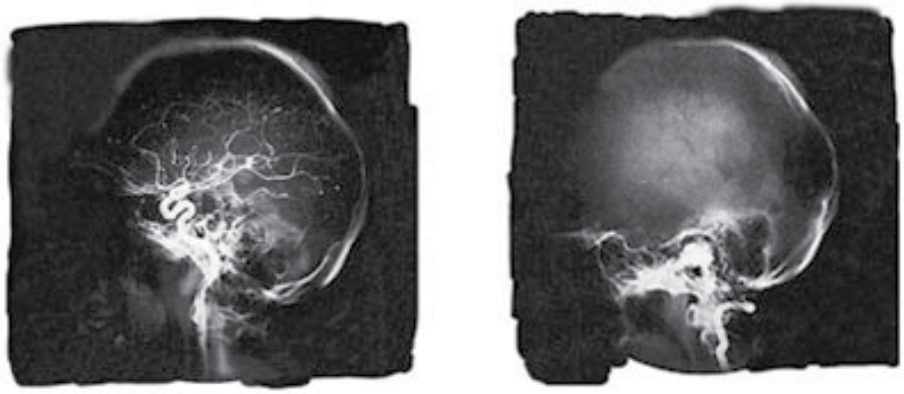
Angiography



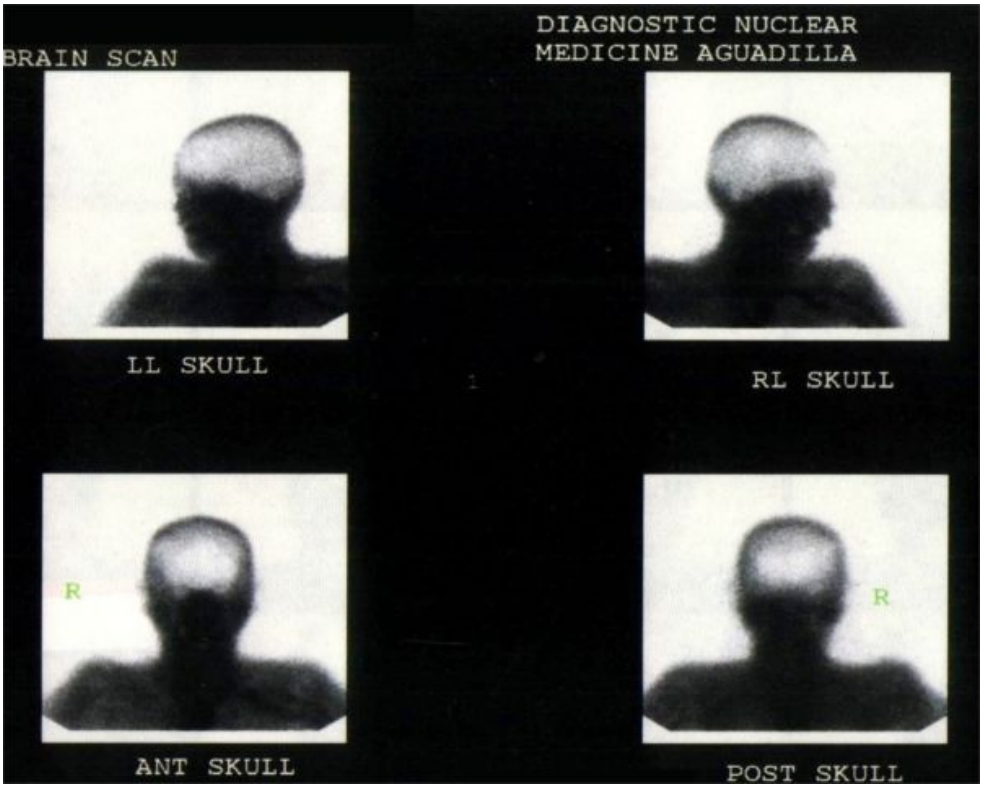
ANCILLARY

- Hungary:
  - angiography (DSA)
  - SPECT
  - TCD (sensit: 70-100%)
- elswhere:EEG, SSEP, CTa
-
-

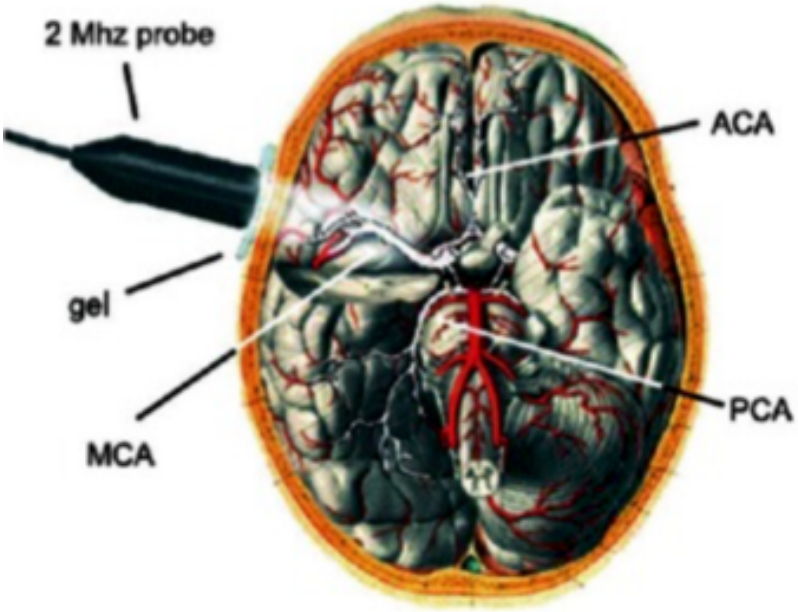
CEREBRAL ANGIOGRAPHY

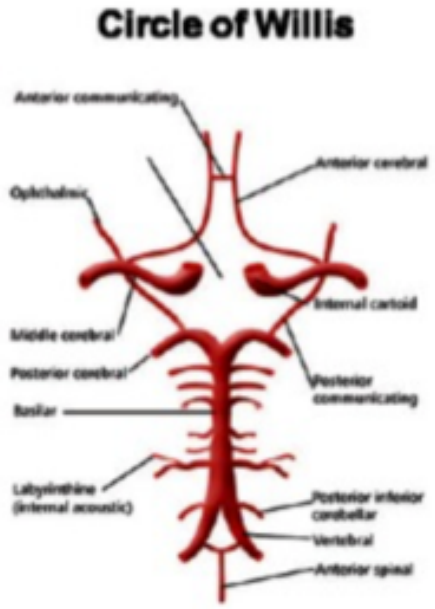


SPECT



TRANSMISSION OF US 2-3.5 MHZ FREQUENCY



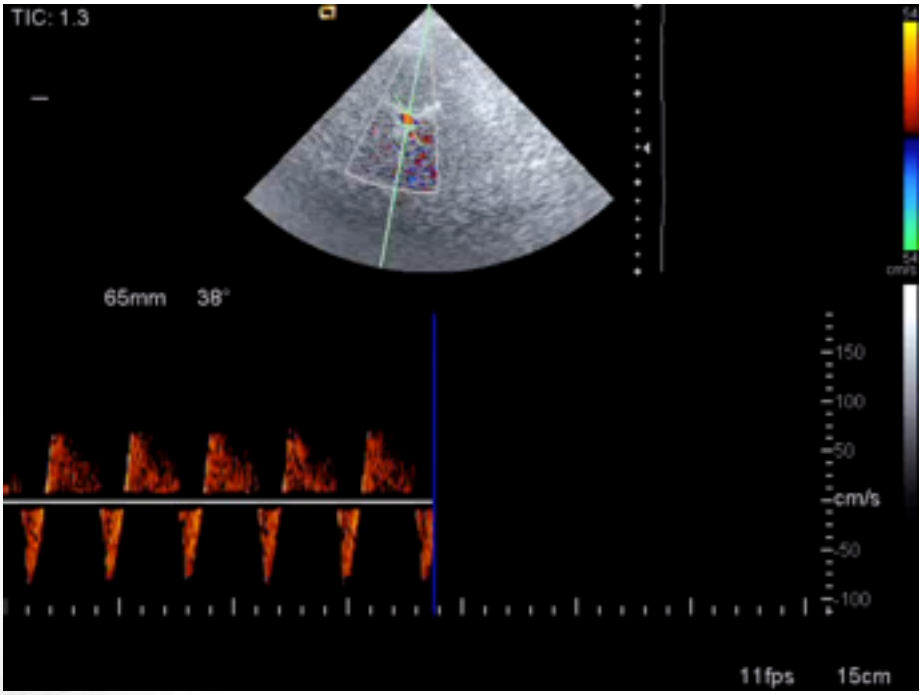


J Cardiovasc Echogr 2016:28(2):28-41.

WAVEFORMS

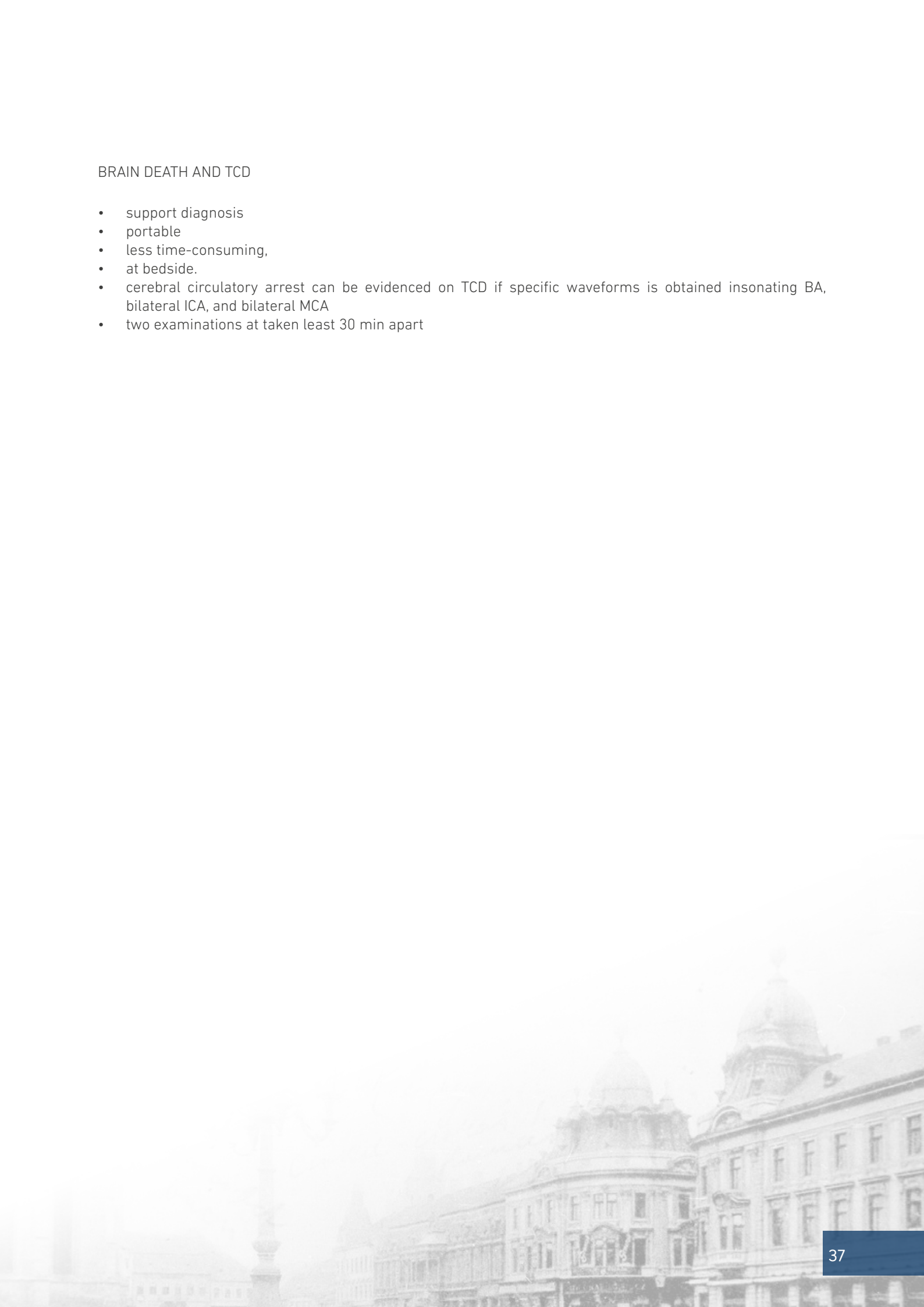
- oscillating waveform (equal systolic forward flow and diastolic reversed flow, i.e. zero net flow;
- or small systolic spikes of <200 ms duration and <50 cm/s PSV with no diastolic flow
- or disappearance of intracranial flow with typical signals observed in the extracranial circulation.

PENDEL FLOW AND SYSTOLIC SPIKES



BRAIN DEATH AND TCD

- support diagnosis
- portable
- less time-consuming,
- at bedside.
- cerebral circulatory arrest can be evidenced on TCD if specific waveforms is obtained insonating BA, bilateral ICA, and bilateral MCA
- two examinations at taken least 30 min apart



NOTES


UNIVERSITAS MEDICINAE ET PHARMACIAE
NAPOCENSIS