Nordic Congress of Clinical Neurophysiology

7-9th of May 2015
Hotel Marienlyst, Helsingør, Denmark
Webseite: www.nccn2015.dk

Scientific programme

Thursday May 7th

09:00 - 17:30 Registration
10:00 - 11:00 Coffee in the exhibition area
11:00 - 11:05 Welcome
11:05 - 12:30 New neurophysiological methods in epilepsy

Chairmen; Martin Fabricius, Department of Clinical Neurophysiology, Rigshospitalet, Denmark
Sandor Beniczky, Department of Clinical Neurophysiology, Filadelfia, Denmark

11:05 - 11:10 Introduction (chairmen)
David Krýsl, Sahlgrenska University Hospital, Gothenburg, Sweden.

11:30 - 11:45 EEG - source imaging in Epilepsy.
Göran Lantz, Neurology Clinic, University Hospital, Geneva, Switzerland and Lund University Hospital, Sweden

11:45 - 12:00 MEG - source imaging.
Sandor Beniczky, Department of Clinical Neurophysiology, Filadelfia, Denmark

12:00 - 12:10 Source imaging, discussion.

12:10 - 12:30 ContinuousEEG.
Martin Fabricius, Department of Clinical Neurophysiology, Rigshospitalet, Denmark

12:30 - 14:00 Lunch, coffee in the exhibition area and poster session
14:00 - 15:30 Channelopathies and neurophysiological investigations

Chairman; Christian Krarup, Department of Clinical Neurophysiology, Rigshospitalet, Denmark.

14:00 - 14:30 Channelopathies in peripheral nerve.
Christian Krarup, Department of Clinical Neurophysiology, Rigshospitalet, Denmark

14:30 - 15:00 Channelopathies in muscle.
Werner Z’Graggen, Universitätsklinik für Neurochirurgie, Bern University Hospital, Switzerland

15:00 - 15:30 Channelopathies in pain.
Kristin Ørstavik, Section of Clinical Neurophysiology, department of neurology, Oslo University Hospital, Norway

15:30 - 16:00 Coffee in exhibition area

16:00 - 17:30 Free communications

16:00 - 16:15 Chronic post stroke central pain: Increased success rate of chronic epidural motor cortex stimulation using somatotopic, navigated repetitive TMS for patient selection and implant.
Magnus Thordstein

16:15 - 16:30 Impact Of Magnetoencephalography On The Decision Making By The Danish Multidiciplinary Epilepsy Surgery Team
Lene Duez

16:30 - 16:45 Altered motor axon voltage-gated Na+ channel function during aging in mice
Mihai Moldovan

16:45 - 17:00 Microneurography recordings from C-nociceptors in patients with erythromelalgia-like pain and Nav 1.9 variants
Inge Petter Kleggetveit

17:00 - 17:15 Cortical spreading depolarizations are associated with metabolic derangement in spontaneous intracerebral hemorrhage
Christian K. Friberg

17:17 - 17:30 Electroencephalography and delirium in critical illness
Rikke Malte Nielsen
17:45  Meet in the hotel lobby
18:00  Welcome reception at Kronborg with guided walk.

**Friday May 8th**

**08:30 - 17:00**  Registration

**09:00 - 10:00**  **Keynote lecture:**  *Single fiber EMG - historical landmarks and present developments.*
*Erik Stålberg, Institute of Neuroscience, Clinical Neurophysiology, Uppsala, Sweden*

**10:00 - 10:20**  Coffee in exhibition area

**10:20 - 11:50**  **Neuromuscular investigation in the critically ill patient**
Chairman;  *Clarissa Crone, Department of Clinical Neurophysiology, Rigshospitalet, Denmark.*

**10:20 - 10:50**  **Critical illness myopathy/polyneuropathy:**
Pathophysiology and diagnostic procedures during early course and follow up
*Susanne Koch, Department of Anesthesiology and Intensive Care Medicine, Charité Campus Virchow-Klinikum, Berlin, Germany.*

**10:50 - 11:20**  **Bench to bedside project on intensive care unit muscle wasting:** Underlying mechanisms and specific interventions.
*Lars Larsson, Department of Physiology & Pharmacology, Dept. of Clinical Neuroscience, Karolinska Institute, Sweden*

**11:20 - 11:50**  **Optimizing testing methods and collection of reference data for differentiating critical illness polyneuropathy from critical illness myopathy:**
Humberto Skott, Department of Physiology & Pharmacology, Dept. of Clinical Neuroscience, Karolinska Institute, Sweden

**11:50 - 13:10**  Lunch, coffee in the exhibition area and **poster session**

**13:10 - 14:30**  **Neurophysiological methods for intraoperative monitoring**
Chairman;  *Birger Johnsen, Department of Clinical Neurophysiology, Aarhus University Hospital, Denmark.*
13:10 - 13:30 **IOM in glioma surgery:**

Hans Axelson, Department of Clinical Neurophysiology, Uppsala University Hospital, Sweden

13:30 - 13:50 **IOM in brainstem surgery:**

Pål Gunnar Larsson, Division of Surgery and Clinical Neuroscience, Oslo University Hospital, Norway

13:50 - 14:10 **Intraoperative neurophysiology in intraspinal surgery:**

Jonas Persson, Department of Clinical Neurophysiology, Karolinska University Hospital, Stockholm, Sweden

14:10 - 14:30 **Intraoperative monitoring of scoliosis surgery.**

Ralf-Peter Michler, Department of Clinical Neurophysiology, St. Olav’s Hospital, University Hospital of Trondheim, Norway

14:30 - 15:00 Coffee in exhibition area

15:00 - 16:00 **Nerve and muscle Ultrasound and MRI**

Chairmen;  
Erisela Qerama Montvilas, Department of Clinical Neurophysiology, Aarhus University Hospital, Denmark

Nils Wolfram, Department of Clinical Neurophysiology, Glostrup, Denmark

15:00 - 15:20 **What can you see on neuromuscular imaging?**

Michel Court Payen, Gildhøj Private Hospital, Denmark

15:20 - 15:40 **Nerve and muscle ultrasound in neuromuscular disorders - Experiences and results of prospective studies:**

Erisela Qerama Montvilas, Department of Clinical Neurophysiology, Aarhus University Hospital, Denmark

15:40 - 16:00 **Experiences of combined electrophysiology and High Resolution UltraSound in the examination of peripheral nerves:**

Nils Wolfram and Janus Kaufmann, Department of Clinical neurophysiology, Glostrup, Denmark
16:00 - 17:00 **Parallel hands-on sessions:**
1) Neuromuscular ultrasound.
   *Nils Wolfram, Erisela Qerama*
2) Threshold tracking in channelopathies.
   *M. Moldovan, W. Z’Graggen, H. Tankisi*

19:00 Gala dinner

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**Saturday May 9th**

08:30 - 11:00 Registration

09:00 - 10:00 **Keynote lecture; Brain basis of social interaction.**
*Riitta Hari, Brain Research Unit, Aalto University, Finland*

10:00 - 10:30 Coffee in exhibition area

10:30 - 12:30 **Neurophysiological methods in sleep and aging**
Chairman; *Benedikte Wanscher, Department of Clinical Neurophysiology, Glostrup, Denmark*

10:30 - 10:45 REM sleep Behaviour Disorder: An early sleep disease marker for late neurodegeneration.
*Poul Jennum, Department of Clinical Neurophysiology, Glostrup, Denmark*

10:45 - 11:00 Electromyographic activity in REM sleep behavior disorder.
*Rune Frandsen, Department of Clinical Neurophysiology, Glostrup, Denmark*

11:00 - 11:15 Prepulse inhibition is associated with attention, processing speed, and 123I-FP-CIT SPECT in Parkinson’s disease.
*Marielle Zoetmulder, Department of Clinical Neurophysiology, Glostrup, Denmark Denmark*

11:15 - 11:30 Sleep stability and transitions in patients with idiopathic REM sleep behaviour disorder and patients with Parkinson’s disease.
*Julie AE Christensen, Department of Clinical Neurophysiology, Glostrup, Denmark*

11:30 - 11:45 The Copenhagen longitudinal study of male cognitive aging and fMRI and EEG measures of cognitive decline.
*Martin Lauritzen, Department of Clinical Neurophysiology, Glostrup, Denmark*
11:45 - 12:00 Selective attention modules auditory and visual steady state responses.
Krisztina Benedek, Department of Clinical Neurophysiology, Glostrup, Denmark

12:00 - 12:15 Neurophysiological correlates of illusions: Visual perceptive network study.
Anna Horwitz, Department of Clinical Neurophysiology, Glostrup, Denmark

12:15 - 12:30 Neocortical gamma oscillations in epilepsy.
Krisztina Benedek, Department of Clinical Neurophysiology, Glostrup, Denmark

12:30 Sandwiches in exhibition area and goodbye

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Stereo-EEG: the Göteborg experience

David Krysl, Bertil Rydenhag, Kristina Malmgren
Sahlgrenska University Hospital, Göteborg, Sweden

Introduction: Stereoencephalography (SEEG) uses stereotactically implanted intracerebral electrodes to test hypotheses about origin and propagation of epileptic seizures. It allows exploration of deep cortical regions and reconstruction of seizure pattern in 3D. SEEG candidates are commonly MR-negative or have electroclinical syndrome discordant with lesion location. In Göteborg, SEEG was first performed in 2013/04 and is now important part of epilepsy surgery program.

Methods: Review of approach to SEEG in Göteborg and presentation of patients investigated by SEEG since 2013/04.

Results: 6 adults (age 20-40) and 4 children (age 4-9) underwent SEEG. 4 adults presented as TLE with concerns about extent of epileptogenic zone or multilobar (fronto-temporal) involvement. Two adults presented as frontal lobe epilepsy (FLE), one was later reclassified as possible medial parietal case. One child presented as TLE+ (TPO), and 3 as FLE. SEEG was well tolerated, no major complications were observed. Minor complications (without deficits) included one epidural hematoma caused by orthogonal frontoorbital electrode and one bleeding during electrode insertion. Reduction in number of electrodes due to poor bone quality was unexpectedly needed in the youngest child. SEEG lead to cortical resection in 3/4 children and 4/6 adults. All three operated children and 3/4 adults are seizure-free post-op (follow-up 0,5-1 year).

Conclusions: SEEG is an effective and relatively safe method for investigating complicated patients with refractory focal epilepsy. Its successful use is heavily dependent on the comprehensiveness of pre-implantation hypotheses. Its use is not limited to adults, but it may be technically difficult in small children. Given the complexity of patients referred for SEEG, short-term seizure freedom in 6/7 operated patients is encouraging. SEEG may also bring evidence in favor of conservative approach that helps to avoid unsuccessful attempts at resective surgery.
In presurgical investigations of patients with pharmaco-resistant epilepsy different non-invasive investigations are performed before proceeding to either invasive recordings or directly to surgery. Among the different non-invasive techniques for localizing the epileptic focus are the electromagnetic methods (based on EEG or MEG recordings) and the hemodynamic methods (PET, SPECT, fMRI). These classes of investigations are complementary in the sense that the electromagnetic methods have a better temporal resolution (down to a few milliseconds) whereas the hemodynamic methods (notably fMRI) have a better spatial resolution. One such non-invasive technique for localizing the epileptic focus is Electrical Source Imaging (ESI). This technique is used to 3-dimensionally localize the source of interictal epileptiform activity from high density EEG recordings.

In this presentation a workflow for the ESI investigations will be suggested. The importance of using a sufficient number of recording electrodes as well as the importance of which timepoint of the spike is chosen for the analysis will be demonstrated. Different evaluation studies demonstrating the usefulness of the technique will be presented and the possibility to combine ESI with other non-invasive neuroimaging techniques will be discussed.
MEG - source imaging
Sandor Beniczky (Danish Epilepsy Centre and Aarhus University)

Several strategies are available for solving the inverse solution: determining the location in the brain of the cortical areas generating EEG and/or MEG signals. This presentation reviews the principles behind the various source imaging strategies and reviews the published evidence for the diagnostic accuracy and clinical utility.

BACK TO PROGRAM
cEEG

Martin Fabricius, dep.of Clinical Neurophysiology, Rigshospitalet, Copenhagen.

Continuous EEG (cEEG) is a key instrument for diagnosing and treating non-convulsive seizures and non-convulsive status epilepticus. Furthermore non-epileptic motor manifestations are common in unconscious patients in the intensive care unit and may be distinguished from true seizures by means of cEEG. The analysis requires a considerable man-power from trained EEG-interpreters, but may be performed on-line by remote-access. The cEEG trace is evaluated by direct inspection as well as by means of quantitative EEG measures such as amplitude integrated EEG and power spectrum trend analysis. Bedside staff must be trained to enter events into the EEG software such as changes in medication and clinical events, to allow an optimal assessment. Current dialogue with the attending neurologists and intensivists is very important. Recordings will be presented to demonstrate typical cases as well as pitfalls.

BACK TO PROGRAM
Channelopathies in peripheral nerve

Christian Krarup, Department of Clinical Neurophysiology, Rigshospitalet and University of Copenhagen, Denmark

Nerve fibre function is dependent on the concerted activation and inactivation of a number of ion-channels integrated in the axolemma. In myelinated fibres, the different channels are segregated at the node of Ranvier and at the internode. The homeostasis of ion concentrations is furthermore dependent on various ion pumps and exchangers that maintain the axon membrane potential. Abnormalities of ion channels and pump function may cause cramps, paraesthesia, pain, sensory loss or weakness and may eventually lead to nerve fibre degeneration.

Channelopathies may be hereditary or acquired. Hereditary channelopathies may affect Na⁺ or K⁺ channels and include symptoms localized to the peripheral or the central nervous system or both. Acquired disorders that affect ion channels directly by blocking or indirectly through metabolic changes are more common, as for example autoimmune blocking of K⁺-channels in motor fibres. Furthermore, changes in ion-channel distribution may have functional implications in for example chronic demyelinating neuropathies and in regenerated fibres. Studies in hereditary motor and sensory neuropathies have also shown that aberrant ion-channels, usually not present at the node of Ranvier, may become expressed, and such ectopic channels may cause disturbed function confirming that transcriptional changes may lead to channelopathies.

Conventional nerve conduction studies are not well suited to unravel changes in function associated with ion-channel or axon membrane dysfunction. NCS may show loss or slowing of fibres but complementary studies of axonal excitability have become increasingly useful in the understanding of dysfunction of nerve fibres that are still able to conduct action potentials. These methods use threshold tracking to assess changes in membrane potential and thus gain insight into ion-channel functions. By combining different conditioning potential changes, the accommodation associated with activation of ion-channels localized at the node of Ranvier and at the internode can be assessed.

BACK TO PROGRAM
Channelopathies in muscle

Werner Z'Graggen, Departments of Neurology and Neurosurgery, University Hospital, Bern, Switzerland

Muscle fibers, like nerve fibers, are complex electrical organs, in which the accurate control of membrane potential, and the interplay of multiple voltage-dependent ion channels, have essential roles in normal physiological function. The number and diversity of diseases of striated muscle greatly exceeds the number of symptoms and signs by which they express themselves clinically; thus, different diseases share certain common symptoms. The typical diagnostic sequence in a patient with suspected myopathy consists of taking patient history, clinical examination, laboratory investigation and needle electromyography followed by muscle biopsy or genetic testing. Clinical and laboratory examinations provide often rather unspecific findings. Also qualitative and quantitative electromyography have a limited sensitivity to reveal the cause of myopathy or to characterize the type of channelopathy.

We have developed a method of recording muscle velocity recovery cycles, with the aim of facilitating the diagnostic process in patients with suspected myopathy. A muscle action potential is followed by early and late depolarizing afterpotentials, which can be assessed by their effects on the conduction velocity of a second muscle action potential. After the refractory period, the early afterpotential gives rise to an increase in velocity (early supernormality), which is highly sensitive to the muscle resting potential. The late afterpotential is augmented by multiple conditioning stimuli and thought to reflect potassium accumulation in the t-tubules.

Muscle velocity recovery cycles provide the clinical neurophysiologist with a quick and virtually painless method of obtaining information about human muscle membrane properties in vivo. Recently, the method has been used to demonstrate distinct changes in different myopathies including channelopathies. For example it has been shown, that chloride channel dysfunction causes characteristic alterations in muscle velocity recovery cycles in myotonia congenita and the myotonic dystrophies.

BACK TO PROGRAM
Channelopathies and pain.  
Kristin Ørstavik, MD, PhD. Section of Clinical Neurophysiology, Department of Neurology, Oslo University Hospital  

Ion-channels are crucial for conveying pain sensation in the peripheral and central nervous system. In later years the research regarding the role of ion-channels in chronic pain has been intensified. Our knowledge of possible mechanisms for neuropathic pain has been broadened due to the discovery of certain hereditary sodium-channel mutations that leads to rare chronic pain conditions such as primary erythromelalgia, while other mutations leads to an insensitivity for pain. Even in some patients with presumed idiopathic polyneuropathy, mutations of sodium-channels with a possible critical role in the generation and conduction of action potentials in nociceptors have been reported. In primary erythromelalgia patients, multiple different mutations have been identified in the gene coding for the voltage gated sodium channel subtype 1.7 (Nav1.7). Experimental animal studies have among other findings shown that this leads to hyperexcitability of nociceptors. Direct microneurographic recordings from small nerve-fibers has enabled us to study axonal properties of nociceptors in healthy subjects and how these may change in patients with painful disorders. C-nociceptors in healthy humans show a characteristic activity-dependent slowing of conduction velocity. The observation of a changed pattern of such activity-dependant slowing could reflect one aspect of the underlying ion-channel pathology in primary erythromelalgia. The analyses of mechanisms in such rare disorders might help us understand more about pathological pain including the role of ion-channel pathology.

BACK TO PROGRAM
SFEMG - historical landmarks and present developments

Erik Stålberg, Dept Clin Neurophysiology, University Hospital Uppsala Sweden

In late 50ties Ekstedt and myself started at the Department of Pharmacology Uppsala University for a PhD thesis and the topic given to us was on muscle fatigue. We started with strain gauges, integrated surface EMG recordings, ischemia and so on. In order to get more information from the muscle we looked for other electrophysiological recording parameters and were inspired by Buchthal’s multielectrode. We used some of those, but thought that smaller recording surfaces perhaps could reveal more details. After lots of testing, SFEMG electrodes were constructed by ourselves containing 1-14 very small recording surfaces, each with a size smaller than the muscle fiber. The obtained signals caused considerable headache. Which is the generator, can it be just ONE muscle fiber? This was against the prevailing concept of organization of muscle fibers within the motor unit (MU) in small bundles of fibers of 10-30 fibers, a subunit. We tested to see if our signals were generated by many or just one muscle fiber by using small multielectrodes to study volume conduction, injected sodium citrate intramuscularly to activate individual muscle fibers, induced slight curarization that supposedly should have caused a disintegration of the signal if composed of many fibers, and made recordings in MG, where single fiber signals should block together in an all-or-none fashion. The conclusion after some years, that included a couple of consultations with Buchthal in Copenhagen who was our primary opponent, was that the signals really come from individual fibers. 1 Another conclusion was that the organization of muscle fibers in a MU are arranged randomly, across the MU territory. During this work two separate phenomena were described. First the jitter phenomenon, which is a variability in transmission time in individual motor endplates. Its physiology was supported by intracellular recording by Elmquist and Quastel. The jitter phenomenon was closely studied both with voluntary and electrical stimulation regarding physiology (the effect of temperature, long term activity, response to drugs, ischemia, anesthetic agents) and methods for quantitation. After some time the jitter was introduced as a routine method more or less universally for the diagnosis of MG.

The other parameter of the single muscle fiber was its propagation velocity. 2 This varied from one discharge to the other depending on activity during the immediately preceding 1000 ms. This variability was due to the “velocity recovery function” (VRF), which followed a very strict mathematical law and was interpreted to be an ion channel phenomenon. This has recently been taken up for further excitability studies by Bostok et al. In addition the velocity decreased during continuous activity. With SFEMG recordings we also looked at the local organization of muscle fibers in a given MU, called “Fiber Density” (FD). This is abnormal in reinnervation but also in some myopathies.

During the development and later clinical implementation of SFEMG, computer techniques have been essential for practical application of jitter analysis, which actually was the first application of computers in EMG. Initially we had a film camera in front of a time interval counter showing numbers, and each sweep triggered a frame in the camera. All data were then inserted manually on punch cards, which were processed in the university computer, (IBM 1640, 16 Kb). Later, time measurements were made with PCs, and today all EMG manufacturers have implemented software for jitter analysis.

Over the years a number of nerve-muscle disorders have been explored with SFEMG3 and many observations have explained what we see in conventional EMG (jitter, VRF, change in propagation velocity with fatigue).

With more strict rules regarding the use of disposable material in medicine, including EMG electrodes in some countries, we have been forced to find a replacement for the SFEMG electrode to measure jitter. After practical and theoretical testing we have found the smallest available “facial” concentric needle electrode to be a workable alternative for jitter measurements, but not for fiber density. A multicenter study to obtain new reference values has just been completed. The jitter is somewhat lower in this reference material than the previously developed reference limits for SFEMG, however the diagnostic sensitivity for MG has been shown to be similar to that of SFEMG.

SFEMG has also been applied to study not only neuromuscular transmission but also the jitter in single anterior horn cells in vivo (via the H-reflex), F-responses, multisynaptic reflexes (blink & flexion
reflexes) and central transmission during cortical stimulation. A phenomenon called the “axon reflex” has been used to study the terminal motor nerve branches using electrical stimulation. The so-called “Complex Repetitive Discharges” (CRD) have been explained from SFEMG studies as a sign of hyperexcitability with ephaptic transmission between muscle fibers. In summary, jitter measured by SFEMG is an established clinical technique, which so far is the most sensitive electrophysiological parameter to detect disturbed n-m transmission in vivo. It should be stressed, however, that increased jitter is a sign of disturbed transmission at the neuromuscular junction, which is not specific for MG, since jitter is seen in early reinnervation, during ischemia, and probably during electrolyte disturbances and other conditions. SFEMG has also helped understand a number of phenomena seen in conventional EMG and is a valuable tool to study single cell activity elsewhere than in the muscle fiber. Improved technical facilities in EMG equipment have made the technical part easier, but SFEMG still requires special skill of the operator.

Reference List

BACK TO PROGRAM
Critical illness myopathy / Critical illness polyneuropathy: pathophysiology and diagnostic procedures during early course and follow up

Susanne Koch, Department of Anesthesiology and Intensive Care Medicine, Charité Campus Virchow-Klinikum, Berlin

Objective: Neuromuscular dysfunction in critically ill patients is attributed to either critical illness myopathy (CIM) or critical illness polyneuropathy (CIP). We investigated the impact of early, electrophysiological, differential diagnostic on clinical prognosis. Immobilization, systemic inflammation and impaired glucose metabolism are major risk factors, but the underlying pathophysiology is uncertain.

Methods: Critical ill patients (Sequential Organ Failure Assessment score > 8 on 3 consecutive days within 5 days in ICU) were investigated: Electrophysiological studies including direct muscle stimulation and threshold tracking; successive muscle biopsies to assess histology, glucose metabolism and inflammation response. Unilateral electrical muscle stimulation was performed.

Main Results: CIM is more frequent; CIP protracts ICU discharge and hampers complete recovery. Nonexcitable muscle membrane predicts muscle weakness, showing preferential type II fiber atrophy. Decreased synthesis and increased degradation of myosin heavy chain was found in CIM. Failing Glucose transporter-4 (GLUT-4) translocation to the sarcolemmal membrane has been proved in CIM and cannot be abolished by insulin treatment, whereas electrical muscle stimulation increased repositioning of GLUT-4. Interestingly, skeletal muscle contributes to general inflammation, showing up-regulated serum amyloid A1 expression. Membrane depolarization of motor nerves is a general feature of critically ill patients, whereas CIM/CIP patients show reduced membrane excitability and a “paradoxical” reduced potassium current over the membrane.

Conclusion: Our studies shed light on the pathophysiology of CIM / CIP, describing the central role of membrane inexcitability, immobilization, glucose metabolism and systemic inflammation.
Bench to Bedside Project on the Intensive Care Unit Muscle wasting: Underlying Mechanisms and Specific Interventions

Lars Larsson (lars.larsson@ki.se), Department of Physiology and Pharmacology, Department of Neuroscience, Clinical Neurophysiology, Karolinska Institutet, Stockholm, Sweden

Objective. Impaired muscle function accompanies intensive care unit (ICU) patients with negative consequences for recovery from primary disease and weaning from the respirator. Accordingly, there is urgent demand for novel treatments and preventive measures, requiring research on the underlying mechanisms. This project is focused on the acquired critical illness myopathy (CIM) and the ventilator induced diaphragm muscle dysfunction (VIDD). Novel experimental models and methods for analyses of muscle tissue have been used, giving us an unprecedented opportunity to (a) address the causative factors and underlying mechanisms, and (b) develop effective treatments.

Methods. Novel experimental ICU (ExICU) models together with analyses of muscle tissue from ICU patients have been used to study the mechanisms underlying the muscle wasting and impaired function of limb, respiratory and craniofacial muscles in time-resolved studies. Gene/protein expression, protein-protein interactions, mitochondria structure/function and regulation of muscle contraction at the muscle fiber level have been analyzed.

Results. Long-term exposure to immobilization and mechanical ventilation in the rat ExICU model induced an identical muscle geno/phenotype as in ICU patients with CIM, suggesting that the complete mechanical silencing unique for ICU patients is an important factor underlying CIM. Mild passive mechanical loading reduced the decrease in muscle fiber function in both experimental and clinical studies, supporting early physical therapy in immobilized and mechanically ventilated ICU patients. Pharmacological intervention with a chaperone co-inducer had strong positive effects on mitochondria and muscle fiber function in the diaphragm, providing proof-of-concept for the translation of this intervention to clinical studies.

Conclusion. Specific mechanisms and muscle type specific differences have been unravelled and positive outcomes of specific interventions have been documented. This will facilitate the translation of laboratory findings into practical clinical knowledge; leading to useful therapies and improved outcomes for patients in need of mechanical ventilation and critical care.

Methods: The effects of 5 days CMV on diaphragm muscle fibre size and function were investigated in young and old F344 Brown Norway hybrid rats. The experimental groups were deeply sedated, pharmacologically paralyzed and extensively monitored 24 hours per day. BGP-15 was given in systemically by intravenous infusion. After 5 days, the mid-costal parts of the diaphragm were dissected, chemically skinned and prepared for contractile measurements. Cross sectional area (CSA) absolute force (P0) and specific force (absolute force/CSA) were measured at the single muscle fibre level.

Results: An ageing-related hypertrophy and decline in specific force were observed in control fibres. A compensatory hypertrophy was observed in response to 5 days CMV in both young and old animals. Systemic administration BGP-15 had a significant positive effect on diaphragm muscle fibre specific force in the young, restoring it to control values, but not in the old. The strong effect of BGP-15 on muscle fibre function in the young was paralleled by a significant up-regulation of HSP 70, but this effect was absent in the old.

Conclusions: Significant age-specific differences were observed in control diaphragm fibres and in the response to 5 days CMV. Further, BGP-15 had a strong positive effect on diaphragm muscle fibre function and HSP expression after 5 days CMV, but this effect was restricted to the young animals.

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Optimizing testing methods and collection of reference data for differentiating critical illness polyneuropathy from critical illness myopathy

Humberto Skott, MD, Karolinska Universitetssjukhuset, Huddinge, Neurofysologiska Kliniken C1-78 141 86 Stockholm, Sweden

INTRODUCTION: Acquired muscle weakness in critically ill patients in the ICU is a major cause of morbidity and mortality. In severe acute quadriplegic myopathy, muscle fibers are electrically inexcitable; in critical illness polyneuropathy the excitability remains normal. Conventional electrodiagnostic methods do not provide the means to adequately differentiate these.

OBJECTIVE: To further optimize methodology for the study of critically ill ICU patients and to create a reference value database in healthy controls.

METHODS: Different electrophysiologic protocols were tested to find techniques as robust as possible and with sufficiently reproducible results for clinical diagnostic applications.

RESULTS: Many parameters show large test-retest variability within the same healthy subject. Reference values have been collected and described as a basis for studies of weakness in critical illness.

DISCUSSION: With these parameters described and quantified, we can now apply these to actual patients with weakness in the setting of critical illness.
IOM in glioma surgery

Hans Axelson

The main goal of glioma surgery is to achieve maximal cytoreduction while obtaining optimal functional outcome. For this purpose, there is an increasing demand for intraoperative neurophysiology (ION) in addition to other assistive pre- and intraoperative technologies. Like any other diagnostic tool, ION may produce false positive “alarms”, with the risk of reducing the extent of resection and long-term survival. On the other hand, false negative results may instill a false sense of security which may lead to permanent neurological deficits from the surgery. Obviously, all attempt should be made to use standardized ION techniques while bearing in mind the lack of “high-evidence” support for some of the procedures.

In this presentation, I will describe some of the established ION methods used in glioma surgery and share some of our experience in this context. Basically, the ION procedures are divided into mapping and monitoring techniques. Cortical mapping is the first step where the neurophysiologist aid the surgeon to find a suitable cortical entry point for tumour resection by excluding that “eloquent” cortical areas is in the harm’s way. For tumours near the primary motor cortex it may be sufficient to establish the location of the central sulcus by phase-reversal SEP whereas planned surgery near or within cortical language regions requires electrical stimulation of those regions in the awake patient. As tumour resection proceeds, it is important to continuously control the integrity of cortical areas and their subcortical projections with for instance MEP monitoring from contralateral muscles. In the awake patient, the functional status is typically monitored by clinical motor-, language- or cognitive tests in addition to some of the standard ION monitor procedures. Not to forget, is that subcortical pathways can be located by electrical stimulation in the resection cavity (subcortical mapping) which may produce a “no-go” result for further resection.
IOM in brainstem surgery:

Pål Gunnar Larsson, Dr. philos., Seksjonsleder Klinisk nevrofysiologi, Nevrokirurgisk avdeling, Oslo Universitetssykehus

The human brainstem is very complex and this makes neurosurgery in the area difficult. Due to the compact nature and the complex organization it may be difficult for the neurosurgeon to navigate and to avoid damage to the locale structures. To monitor and to help localizing structures, a set of clinical neurophysiological methods are available. The cranial nerves leave the central nervous system in the brainstem area. Except the Olifactory Nerve, there are clinically available methods for monitoring all cranial nerves. The optical nerves may be monitored by continuous flash-VEP and acoustical nerve by AER. The other cranial nerves may be monitored by means of spontaneous EMG recording, and most of them may also be localized by stimulation procedures e.g. needed when the nerve roots are embedded in tumor tissue. Passing nerve tracts may be monitored both by SEP, MEP and direct stimulations. MEP may even be used on the cranial nerves, but there are challenges. Available methods and challenges will be discussed and practical experiences will be conveyed.
Intraoperative neurophysiology in intraspinal surgery

Jonas Persson, jonas.persson@karolinska.se

During surgical procedures in the spinal canal there is a potential risk for damage to the spinal cord or nerve roots which could result in postoperative neurologic dysfunction. To prevent or minimize this hazard, various electrophysiological techniques are used intraoperatively.

For monitoring of spinal cord motor function, motor evoked potentials (MEPs) are generated by transcranial electrical stimulation over the motor cortex and recorded from appropriate muscles (muscle MEPs, mMEPs) or asynaptically from the corticospinal tracts (direct wave, D-wave). Recording of mMEPs are often used in combination with D-wave recording to enable a graded evaluation of the motor function integrity.

To evaluate the functional integrity of the lemniscal sensory pathway, which is of outermost importance in spinal cord surgery during e.g. posterior myelotomy, somatosensory evoked potentials (SEPs) are recorded transcranially over the sensory cortex following electrical stimulation of peripheral nerves (tibial nerves most often used in lower limbs and median or ulnar nerves in upper limbs). If the dorsal median raphé of the spinal cord is difficult to identify by anatomical landmarks, SEPs from the lower limbs can be recorded selectively from the gracile fasciuli for identification of the physiological dorsal midline of the spinal cord.

In addition to spinal cord surgery there are surgical procedures in the spinal canal at the level of conus medullaris/cauda equina where electrophysiological monitoring and mapping can be used. Recording of free-running EMG during surgical manipulation and recording of compound muscle action potentials (CMAPs) during focal electrical stimulation of various structures in the operative field are done from relevant myotomes for identification of nerve root elements.

Monitoring of genitourinary and anorectal functions (by e.g. pudendal SEPs, bulbocavernousus reflex) could also be of importance during surgery in this area.

References:


Intraoperative Monitoring of Scoliosis Surgery

Ralf Peter Michler, St. Olavs Hospital Trondheim

The Scoliosis Research Society defines scoliosis as a lateral deviation of the normal vertical line of the spine by at least 10 degrees. There are three categories; congenital, neuromuscular and idiopathic scoliosis. The most common, form idiopathic scoliosis, is present in 2% to 4% of children older than 10 years. Surgical intervention is indicated in patients where the curvature is over 40-50 degree.

Neurologic impairment, particularly paraparesis or paraplegia are infrequent but potentially devastating complications of scoliosis surgery. The survey of 7885 scoliosis surgeries showed that spinal cord injury occurred with a frequency of 0.72%. (Scoliosis research society 1975).

The greatest risk of spinal cord injury occurs during distraction of the spine, and injury may result from excessive elongation of the spinal cord by curve correction or by ischemic injury of the anterior spinal artery or its feeding radicular vessels, causing spinal cord ischemia.

Possible factors associated with higher risk of neurologic complications of scoliosis surgery are congenital scoliosis, scoliosis combined with hyperkyphosis, severity of the curve (Cobb angle over 90 degree), patients with congenital scoliosis, neuromuscular scoliosis, combined approach and decreased spinal cord perfusion due to hypotension and hemorrhage.

The “wake up” test was the first method to control the integrity of the motor tract in spinal surgery but with many limitations. The “wake up” test gives only information of spinal cord function at a single point in time, and the reversal of anesthesia confers other risks.

From the 1970s somatosensory evoked potential monitoring (SEP) has been used and a large multicenter survey by Nuwer in 1995 affirmed the efficacy of SEP monitoring in prevention of neurologic deficits. Since SEPs only monitor the neural conduction through the dorsal columns, false negative measurements happened and patients woke up paraplegic despite the absence of an intraoperative SEP signal change.

In the 1990s Deletis, Epstein and colleagues introduced motoric transcranial electrical stimulation monitoring (MEP) to intramedullary spinal cord tumor surgery. MEP was soon adopted in scoliosis surgery, and numerous studies have confirmed its efficacy.

MEP monitoring is more directly relevant for detecting motor pathway injury, and MEP deterioration often occurs before and sometimes, without SEP change (Mac Donald et al 2007). Nevertheless, congruent MEP/SEP deterioration increases diagnostic certainty and suggests a more severe transverse cord compromise. In addition, reversible SEP deterioration without MEP change has been reported and implies selective dorsal column compromise (Tome-Bermejo et al 2014, Mac Donald 2007) There is a critical window for intervention. Significant alteration of SEP and MEP signal, and recovery within 15 min. are usually not associated with postoperative deficits. (Nuwer et al 2008).

The anesthesiologist and surgeons are able to intervene in a variety of ways when IOM raises warnings: Elevating of the mean blood pressure to > 90mm Hg, increasing the oxygen concentration, measuring arterial blood gas for metabolic abnormality or low hemoglobin, irrigating the wound with warm saline solution, discontinuing spinal instrumentation, removing or adjusting grafts and hardware, and if the evoked potentials fail to return, perform a wake up test.

There is general consensus in SEP intraoperative interpretation. A decrement in amplitude over 50% is used as a warning criterion. Over 10% increment in latency is also used as a warning criterion, but latency changes are quite common and less significant.

Different warning criteria have been proposed in MEP interpretation, ranging from changes in thresholds that elicit muscle MEPs, amplitude variation, or simply the presence or absence of responses. Presence of MEPs excludes paralysis, and disappearance is a strong predictor of new weakness, with few false positives. (MacDonald 2014)

Disappearance is a major criterion, mandating restorative efforts. Marked amplitude reduction could be a moderate criterion prompting restorative efforts, but may increase false positives. (MacDonald 2014)
The D waves are generated by direct activation of the axon of fast conducting fibres of the cortico-spinal tract. Combined recording of D wave and muscle MEPs is a powerful tool in spinal cord tumor surgery. The D wave amplitude remaining above 50% of baseline appears to predict long-term recovery of any early weakness, whereas irreversible over 50% reduction correlates with permanent motor deficit. In scoliosis surgery the amplitude changes up to 70% (Ulkatan et al., 2006). This might be due to alterations of the distance between the spinal cord and the epidural recording electrode. Therefore, there is no established criterion for scoliosis surgery. Because of the high incidence of false positive results, the D wave is not very useful in IOM in scoliosis surgery.

To assess thoracic pedicle screw placement, testing of the pedicle screws may be performed. Triggered thresholds below 6 mA should alert the surgeon to suspect a medial or inferior wall breach.

IOM does not eliminate all adverse neurologic events. Some neurological deficits can occur despite interventions in response to the warning, because there is nothing that can be done, such as acute anterior spinal artery thrombosis.

Monitoring only determines the spinal cord function at the moment of monitoring and delayed postoperative paraplegia which is often correlated with hypotension, can occur many hours after operation. Close postoperative observation is important.

Successful monitoring during scoliosis surgery requires good communication between the neurophysiologist and the anesthesiologist. Neuromuscular blocking agents depress MEP and should not be used. Inhalational anesthetics interfere particularly with MEPs and should be avoided. Total intravenous anesthetics like propofol and opiate attenuate MEPs to a much lesser degree and should be preferred.

It is recommended that the responsible person on the monitoring side should be an medical doctor with subspecialty education in clinical neurophysiology and additional education on intraoperative monitoring. (Sutter et al 2007).

Reliance on computerized information, without validation by trained and experienced monitoring professional can be dangerous. (Yingling 2011)

Conclusion:

Optimal intraoperative monitoring during scoliosis surgery entails both SEP and MEP recording.

Combination of SEP and MEP monitoring provides assessment of entire spinal cord functionality in real time.

There is strong evidence that multimodality intraoperative neuromonitoring is sensitive and specific for detecting intraoperative neurologic injury during scoliosis surgery.

IOM is a valuable tool to optimization of outcome in scoliosis surgery. Multimodal IOM is the Gold Standard in scoliosis surgery.

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BACK TO PROGRAM
What can you see on Neuromuscular Imaging?

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Ultrasound (US) is increasingly used for morphologic assessment of patients with nerve diseases as a complement to neurophysiological examination. Compared to MRI, US has following advantages: providing images with high resolution, multiregional (possible tracing of a long segment of a nerve, detection of polyneuropathy), interactive (with the clinical examination), comparative (quick examination of both sides), dynamic (nerve snapping at the elbow, US-guided needle aspiration or injection, needle biopsy, placement of EMG-needle), minimal artifacts from metal. In contrary to US, MRI provides visualization of nerves/muscles hidden by bony structures and detection of diffuse tissue edema. MRI is also better to detect small deep-situated nerves, and gives a better overview of large muscle groups.

The normal fascicular structure of nerves is well demonstrated on US. US can provide localization and direct visualization of nerve entrapment with nerve flattening at the site of compression, proximal (and sometimes distal) nerve swelling, nerve hyperemia (color Doppler examination). The cause of nerve entrapment can be established (retinaculum, fibrous band, tenosynovitis, bone, osteosynthesis, tumor, cyst, or anomalous muscle). Maximal nerve swelling can be measured (nerve area). Muscle atrophy and/or fat degeneration, secondary to nerve entrapment, can be demonstrated and may be the only finding in entrapment of very small nerves. Early muscle edema is not well demonstrated with US, but easy to see on MRI.

US may also be useful in patients with blunt or open trauma for detection of nerve transsection, neuroma and other lesions in the tendons and muscles. US may help detecting soft tissue tumors compressing nerves, arising from nerves (schwanomas), or ganglion cysts invading nerves (peroneal or tibial). MRI is required in patients with suspicion of malignancy.

US may have a role in primary myopathic disorders, neurogenic myopathies, inflammatory myopathies, muscle tumors, and traumatic muscle lesions.
Nerve and muscle ultrasound in neuromuscular disorders - Experiences and results of prospective studies.

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This oral presentation will focus on an overview of studies on ultrasound in neuromuscular disorders and will present cases, experiences and results of recent studies conducted in our department.

Since its first use in the 1950s for medical practice, ultrasound has become a sophisticated imaging modality in the hands of specialists to diagnose a broad range of clinical disorders. Ultrasound provides a fast real-time and dynamic imaging capability, less inconvenience for the patient and lower cost compared to other imaging modalities. In the last two decades, ultrasound has taken an increasing important place in the evaluation of neuromuscular disorders.

Studies on carpal tunnel syndrome (CTS) (1) and on ulnar nerve entrapment (UNE) (2) at the elbow have shown that enlargement and loss of echogenicity are two consistent changes on ultrasound that can distinguish diseased nerves from normal nerves. We have conducted a study on CTS and on UNE at the elbow and will present some key results from those studies. Moreover, we bring cases and experiences from the daily routine in our department where ultrasound is used as a supplement to the electrodiagnostic tests.

Ultrasound may be more sensitive than EMG at detecting fasciculations (3,4) probably because it samples a larger muscle region than needle EMG and is more tolerable than the EMG. We will present first results of an ongoing study on ultrasound of muscles especially of the tongue in patients with various neuromuscular disorders. Diseased muscle displays increased echogenicity and homogeneity, atrophy and loss of the bone shadow (3). Electrographic ultrasound (EUS) is a dynamic strain imaging technique and a promising technology to identify tissue stiffness. We will present results from an ongoing study on the value of EUS as a diagnostic tool in muscle atrophy seen in neuromuscular diseases.

1- Sonography in the diagnosis of carpal tunnel syndrome: a critical review of the literature.

2- Ulnar neuropathy at the elbow: follow-up and prognostic factors determining outcome.


4- Ultrasonographic detection of fasciculations markedly increases diagnostic sensitivity of ALS.
Experiences of combined electrophysiology and High Resolution Ultrasound in the examination of peripheral nerves

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A large amount of all patients referred to EMG/ENG examination of peripheral nerves end up with an unspecified diagnosis. Furthermore, electrophysiological findings do not always fit with the patients’ medical history and/or clinical examination.

To evaluate the extent to which High Resolution Ultrasound (HRUS) is contributive or non-contributive to electrophysiology in relation to final diagnosis, examinations combining electrophysiology and HRUS in 2014 were evaluated.

HRUS was used as an investigative tool when:

- medical history and/or clinical examination gave suspicion of other specific peripheral nerve injury as expected from electrophysiological findings alone (e.g. scar tissue, trauma, tumor) or

- medical history and/or clinical examination gave suspicion of localized peripheral nerve injury despite normal or unspecified electrophysiological findings.

HRUS findings were classified in different groups whether supplemental ultrasound was contributive or non-contributive to electrophysiology in relation to the final diagnosis.

HRUS is able to contribute to electrophysiological examination in order to localize or reveal further information in peripheral nerve injury.

A couple of examples will be shown in the lecture. BACK TO PROGRAM
Brain basis of social interaction

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Human social interaction is everywhere, effectively shaping our brains and minds. Although social interaction is among the most complex functions humans (and their brains) perform, the interaction often appears surprisingly easy. I will advocate the view that to fully understand human brain function we ultimately should be able to study interacting people in naturalistic conditions and to collect information from two brains at the same time, ending up in “two-person neuroscience” (2PN). This approach goes beyond the commonly applied “spectator science” that assumes that humans (and their brains) are reactive and that even complex social functions can be probed by presenting complex-enough stimuli that the subject just observes. However, in reality people are actively participating interactors who by their behavior dynamically affect the (social) stimuli they will receive at the next moment.

I will discuss our approaches and challenges in imaging of single brains under naturalistic conditions and in imaging two brains at the same time (“hyperscanning”). For 2PN purposes, we have built a setup for simultaneous MEG-to-MEG recordings between two distant laboratories and a 2-coil fMRI for imaging two subjects in the same magnet. Analysis of the 2PN-data is still extremely challenging. Most likely, we have not yet adopted the best experimental setups.

BACK TO PROGRAM
REM sleep Behavior Disorder: an early sleep disease marker for later neurodegeneration

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Parkinson’s disease and other alpha-synucleinopathies are some of the most disabling, mortal and common chronic and neurodegenerative disorders that affect middle age and elderly humans. Once fully developed there is only symptomatic treatment but no treatment which affect the serious disease course. Therefore early detection of alpha-synucleinopathy is imperative for potential disease modifying treatment.

Idiopathic Rapid eye movement sleep Behavior Disorder (iRBD) has recently been identified as a strong and early risk factor for later development of an alpha-syn’s with a conversion rate of more than 80% in 10 years. In addition, 60-100% of alpha-synucleinopathy patients suffer from RBD. iRBD is a parasomnia, characterized by loss of the normal REM sleep skeletal muscle atonia, resulting in motor/behavioral activity during sleep in relation to dream mentation. Therefore iRBD may be an early marker in the preclinical/premotor phase for alpha-synucleinopathies which is confirmed in neuropathological studies early identification of iRBD, increased understanding of disease mechanisms and development of sensitive diagnostic measures is central for future treatment-options, neuroprotective strategies, and preventing or postponing that patients with iRBD will develop Parkinson’s disease or other alpha-synucleinopathies.

We have shown that specific electrophysiological polysomnographic markers characterize RBD and PD patients, including: changes in macro sleep structure (loss of wake-sleep stability, sleep transitions), sleep microstructure (e.g. sleep spindles, K-complexes), muscle tone, eye activity and autonomic activity. Further we have found that changes in sleep pattern are diagnostic for diagnosing RBD/PD as compared to controls.

These findings may be important for identification of early phases of RBD/PD, understanding disease process, monitoring disease progression and improving diagnostic methods. Further the results may raise opportunity to identify candidates for disease intervention.
Electromyographic activity in REM sleep Behaviour Disorder.

Rune Frandsen

Objectives Rapid eye movement (REM) sleep behaviour disorder (RBD) is characterised by dream enactment and REM sleep without atonia. The evaluation of atonia is based on visual criteria but there is a need for more objective, quantitative measurements. We aimed to establish a method for determining the baseline and all other parameters in automatic quantifying submental motor activity during REM sleep.

Method Analysis of electromyographic (EMG) activity of the submental muscle in polysomnographies of 35 patients with idiopathic RBD (iRBD), 31 controls and 43 Parkinson's patients. Six adjustable parameters for motor activity were defined. EMG activity was automatically detected and quantified. 648 possible combinations of parameters defining EMG activity were compared for the ability to separate RBD patients from controls. After identification of the optimal method for separating RBD from controls, the parameters were used for PD patients.

Results Automatic baseline estimation improved characterisation of atonia during REM sleep. We found an optimised method for quantifying motor activity during REM sleep. The method was stable in iRBD and PD patients and can be used to differentiate RBD from controls with a sensitivity and specificity of 83%.

Conclusion We developed and applied a sensitive, quantitative, automatic algorithm to evaluate loss of atonia in RBD patients.

BACK TO PROGRAM
Prepulse inhibition is associated with attention, processing speed, and 123I-FP-CIT SPECT in Parkinson's disease.

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BACKGROUND:

Prepulse inhibition is a measure of sensorimotor gating, which reflects the ability to filter or 'gate' irrelevant information. Prepulse inhibition is dramatically altered in basal ganglia disorders associated with dysfunction in the midbrain dopaminergic system, and corresponding cognitive information processing deficits such as slowed processing speed. Parkinson's disease is characterised by the degeneration of the midbrain dopaminergic system and is associated with cognitive dysfunction, including slowed information processing. Although sensorimotor processes in Parkinson's disease have been extensively studied in relation to motor function, less is known about the potential role of sensorimotor processes in cognitive function.

OBJECTIVE:

We investigated the relationship between prepulse inhibition, cognition and nigrostriatal dysfunction, as measured with 123I-FP-CIT-SPECT scanning, in patients with Parkinson's disease.

METHODS:

38 Parkinson patients were assessed with prepulse inhibition, neuropsychological tests, and neurological investigation. A subset of these patients underwent 123I-FP-CIT-SPECT scanning.

RESULTS:

Patients with a higher level of prepulse inhibition performed better on cognitive measures tapping attention and processing speed than patients with a lower level of prepulse inhibition. Furthermore, there were significant correlations between prepulse inhibition and 123I-FP-CIT uptake in the striatum.

CONCLUSIONS:

Our results suggest that the level of prepulse inhibition is related to the efficiency of information processing in Parkinson's disease, and to the density of dopamine transporters in the striatum.
Sleep stability and transitions in patients with idiopathic REM sleep behavior disorder and patients with Parkinson’s disease

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Objective: Patients with idiopathic rapid eye movement (REM) sleep behavior disorder (iRBD) are at high risk of developing Parkinson’s disease (PD). As wake/sleep-regulation is thought to involve neurons located in the brainstem and hypothalamic areas, we hypothesize that the neurodegeneration in iRBD/PD is likely to affect wake/sleep and REM/non-REM (NREM) sleep transitions. Methods: We determined the frequency of wake/sleep and REM/NREM sleep transitions and the stability of wake (W), REM and NREM sleep as measured by polysomnography (PSG) in 27 patients with PD, 23 patients with iRBD, 25 patients with periodic leg movement disorder (PLMD) and 23 controls. PD patients were divided in those with RBD and those without, and iRBD patients were divided in those with major self-reported dream enactments and those with minor. Measures were computed based on manual scorings and data-driven labeled sleep staging. Results: Measures computed from the data-driven labeling showed significant differences. Patients with PD showed significantly lower REM stability than controls and patients with PLMD. Patients with iRBD had significantly lower REM stability compared with controls. Patients with PD and RBD showed significantly lower NREM stability and significantly more REM/NREM transitions than controls. No significant differences were found between PD with and without RBD, and between iRBD patients with and without major self-reported dream-enactments. The measures computed from the manual scorings lacked to show any significant differences. Conclusion: We conclude that W, NREM and REM stability and transitions are progressively affected in iRBD and PD, probably reflecting the successive involvement of brain stem areas from early on in the disease. Sleep stability and transitions determined by a data-driven approach could support the evaluation of iRBD and PD patients.
The Copenhagen Longitudinal Study of Male Cognitive Aging (CoLoSMA) explores how aging influences cognition and brain function in testpersons from the Metropolit 1953 Danish male birth cohort. CoLoSMA is a longitudinal study based on repeated IQ tests, and the selection of the study population is based on each person's IQ trajectory. The aim is to identify etiological factors for cognitive decline in otherwise healthy males, and to obtain fMRI and EEG data that can be used to substantiate a decline in cognitive function in order to be able to intervene before dementia sets in. Higher cognitive functions in mammals, such as perception and cognition, are critically dependent on synchronized neocortical network activity, especially activity in the gamma range (30 – 100 Hz), which is produced by repeated discharges of fast-spiking inhibitory interneurons that synchronize the activity of numerous pyramidal cells by rhythmic inhibition. The presentation will describe the design of the CoLoSMA study, review the literature on neocortical gamma oscillations in pre-MCI/MCI (MCI= mild cognitive impairment) patients, and present the data obtained so far with fMRI in this birth cohort. The fMRI data point to a disturbance in one of the brain's functional connectivity networks in a large group of males (n=100) with subclinical cognitive decline in late midlife. The alterations are similar to the pattern seen in patients with Alzheimer's disease and MCI, which suggests that physiological correlates of cognitive decline may be discovered by use of appropriate methodologies. We now seek to substantiate the abnormalities at the neuronal network level by examining evoked neocortical gamma oscillations as described in the following 2 presentations. Inclusion of test-persons from the Metropolit cohort in the EEG studies was initiated February 1, 2015 and data from these males are not included in the following talks that describes results from normal controls.
Selective attention modulates auditory and visual steady state responses

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Steady state evoked potentials (EP) are widely used in cognitive neuroscience and may be useful as predictors of cognitive decline. Cognition may be examined as rivalry for attention between two sensory stimuli. Rivalry entails competitive interactions at multiple neural sites in higher brain areas, and may provide insights into the neural bases of awareness and attention. This study examined the power spectrum of auditory steady state responses in the presence of a competing visual task. Steady state auditory or visual EPs were recorded in 15 healthy individuals in three consecutive stimulation paradigms that were presented in randomized order. First, the power of an auditory 45 Hz, amplitude-modulated (2 Hz carrier frequency) stimulation was assessed without competing inputs. Second, a visual image of a face/vase illusion was presented alternating with a grey background at a frequency range of 36 Hz. Third, the auditory and visual stimulations were presented together without special instruction as to which one to attend to. The response at the stimulus frequency was normalized to the response within the surrounding frequencies, and SNRs and power of the evoked responses were measured and compared. The results demonstrate that the auditory cortical responses at 45 Hz were markedly decreased by a competing visual stimulus. In comparison, rivalry for attention obtained by presentation of the complex, visuo-auditory paradigm resulted in a 3 fold increase of the power of the visual stimulation at 36 Hz. Auditory evoked responses were localised around, centro-temporal and centro-frontal areas, while the peak of the 36 Hz frequency elicited visual response was localised above the parieto-occipital cortex. Our findings indicate that without a goal directed attention the competition between auditory and visual processing is mainly driven by the visual stimulus during a complex sensory input. The low gamma activity evoked by this paradigm will be applied to study the test-persons in the study of cognitive decline.
Neurophysiological correlates of illusions: Visual perceptive network study

Anna Horwitz, Nelly Richard, Krisztina Benedek, Keng Wah Pang, Miki Nikolic, Martin Lauritzen.

Steady state visual evoked potentials (SSVEP) have been used widely in cognitive neuroscience, but the extent to which SSVEPs can be used to track cognitive function is unknown. The amplitude of the SSVEP response is enhanced in response to attention, working memory and in several other cognitive processes. The strongest local source of SSVEPs is located in the striate cortex and propagates by the combined activity of locally and broadly distributed sources to secondary visual cortical areas. The aim of the study was to assess variations in the SSVEPs during a visual task where the participants were shown a vase-face illusion interchanging with a grey background with a flickering rate of 2 Hz, 8 Hz and 36 Hz, and the evoked activity patterns for the vase-face illusion was compared with the power and spatial distribution elicited by simple checkerboard stimulation with alternations at the same three frequencies. Our study included 20 healthy individuals, mean age 35 years. Signal to noise ratios, and EEG power of the equivalent frequencies were calculated and compared statistically between checkerboard stimulation and the complex vase-face illusory image. SSVEP amplitudes were 3-5 times larger for the complex visual task as compared to checkerboard stimulation. The localization of the maximum power of the SSVEP response varied for different stimulation frequencies: 8 Hz stimulation elicited a response with maximum corresponding to the striate cortex, while the amplitude maximum at 36 Hz stimulation frequency was measured above the subdominant parieto-occipital areas. Different areas of activation of SSVEP during various stimulation points out the involvement of higher level cognitive processing during perception of illusory images, and higher stimulation frequencies elicits activity in association cortex known to be involved in cognitive processing. We suggest that SSVEP recordings may be useful to track changes in cognitive function.
Neocortical gamma oscillations in epilepsy

Krisztina Benedek, Martin Lauritzen

Neocortical gamma oscillations emerge from the precise synaptic interactions of excitatory pyramidal cells and fast spiking inhibitory interneurons, which release GABA at high rate. This presentation compares high frequency EEG activity preceding and during ictal recording of patients with idiopathic generalized epilepsy and frontal lobe seizures.

We used power spectral analyses of the EEG and calculated the time-frequency cross-coherence measures of neocortical gamma oscillations to search for interictal and ictal interregional rhythmic synchrony preceding and complementing clinical seizures. During primarily generalized seizures an abrupt rise of all frequencies was seen synchronously over both hemispheres. The rise in EEG gamma oscillations was short-lasting and decreased before activity at lower frequency ranges. Although interictal gamma activity was significantly higher in this population compared to non-epileptic patient’s recordings no changes were observed prior or after their clinical event. On the contrary in cases of frontal lobe epilepsy a gradual increase of high beta and gamma band was observed prior to their clinical event. This high frequency increase coincided with a decrease of delta activity.

Our data supports the hypothesis that gamma synchrony plays a key role in generating and maintaining a seizure. The increase in gamma oscillatory activity precedes clinical seizures in focal epilepsy, while it rises simultaneously with other frequencies during generalized seizures.

BACK TO PROGRAM
Chronic post stroke central pain: Increased success rate of chronic epidural motor cortex stimulation using somatotopic, navigated repetitive TMS for patient selection and implant

Magnus Thordstein

Introduction Neuromodulation for severe central pain using repetitive stimulation of motor cortices has been used for more than two decades, first via direct cortical stimulation (DCS) by implanted electrodes, later non-invasively mainly by repetitive transcranial magnetic stimulation (rTMS). The outcome at group level has been limited, though for some, the effects have been of large value. There is a need for selection of patients that are likely to benefit from this procedure. Based on a preliminary report indicating that rTMS could contribute to this, we aimed to address this problem. Methods 15 patients with severe central post stroke pain, refractory to conventional treatment were assessed. Somatotopically directed, placebo controlled, motor cortex stimulation with navigated rTMS (nrTMS) employing the published paradigm was used to identify those patients that were likely to respond to DCS in terms of self-reported, quantified measures of symptoms one week before and two weeks after the procedure. The optimal stimulation positions were transferred to an intraoperatively used neuronavigation system and verified by epidural stimulation. Here, an electrode was sewn to the dura mater. Patients reported symptoms as before, intermittently up to 12 months after the operation. Results At present, of the 15 patients evaluated, seven were rejected since they did not respond to nrTMS. Of the eight that did respond, four are waiting for- and four have had the procedure. Of the four with implant all report an, on the whole, positive, clinically meaningful effect. Discussion The value of Evidence Based Medicine is beginning to be reconsidered, pointing to the risk that the overall outcome for individuals and the health service may suffer from a lack of individualized treatment. The kind of procedure described above may considerably improve the outcome of this invasive procedure for severe central pain, making it sound from economical and ethical perspectives.

BACK TO THE PROGRAM
Impact Of Magnetoencephalography On The Decision Making By The Danish Multidiciplinary Epilepsy Surgery Team
Lene Duez

Objective: This prospective ongoing study investigates the impact of magnetoencephalography (MEG), as a non-invasive tool to guide the multidisciplinary epilepsy surgery team.

Methods: MEG (306 channels) was recorded in 33 consecutive patients with refractory focal epilepsy, referred for epilepsy surgery. All patients underwent conventional non-invasive presurgical evaluation. The Danish epilepsy surgery team evaluated the patients first blinded and two weeks later unblinded to MEG. At both sessions the multidisciplinary team determined the presumed localisation of the epileptogenic zone and decided on surgical or additional presurgical plans. The impact of MEG was divided into eight predefined categories: No change, change from operation not possible to implantation of intracranial electrodes, change form operation not possible to operation, change from implantation to operation, change from operation to implantation, change from operation to stop, change from implantation to stop, and change of implantation strategy.

Results: Two patients changed from operation not possible to operation, and 4 patients changed from implantation to operation. Four patients changed from operation not possible to implantation. In five patients the implantation strategy changed. One patient implantation was stopped. In the remaining 17 patients MEG had no impact. In total the decision was changed in 48% of the patients.

Conclusion: MEG makes epilepsy surgery possible in patients where conventional non-invasive presurgical evaluation did not localize the epileptogenic zone.

Key message: MEG influences the decision of the epilepsy surgery team.

BACK TO THE PROGRAM
Altered motor axon voltage-gated Na+ channel function during aging in mice

Mihai Moldovan

Membrane ion channel function of myelinated axons can be explored in vivo using clinically available nerve excitability testing by "threshold tracking" techniques. In contrast to the age related-changes in conduction studies, and the changes of nerve excitability during development, the age-related changes in human nerve excitability were found to be of smaller magnitude and, as such, are presumed to be of little consequence. Our aim was to specifically address membrane function in aging motor axons of C57Bl wild-type (WT) mice. We compared tibial nerve conduction and excitability measures by threshold-tracking in 12 months (mature) and 20 months (aged) wild-type (WT) mice. In aged WT there was an increased CMAP latency and an increased strength-duration time constant. Deviations during threshold electrotonus were reduced. Resting current-threshold slope and hyperpolarizing current-threshold slope were increased. Modeling indicated that aged WT were depolarized, had a larger inward rectification and increased capacitance. An increased Nav1.8 isoform expression was found by immunohistochemistry. The depolarizing excitability features were absent in Nav1.8 null mice, and they were counteracted in WT by the A-803467 Nav1.8 blocker. Our data suggest that an acquired ectopic Nav1.8 expression contributes to motor axon dysfunction during regular aging in mice. The altered VGSC expression with increased Na+ channels with slow closing kinetics like the NaV1.8 could lead to an increased Na+ load per impulse which could become neurotoxic during prolonged activity.
Microneurography recordings from C-nociceptors in patients with erythromelalgia-like pain and Nav 1.9 variants
Inge Petter Kleggetveit

Objective: The sodium channel Nav 1.9 is expressed in peripheral nociceptors and has recently been linked to human pain, but the exact role of Nav 1.9 for human nociceptor excitability is still unclear. Methods: C-nociceptors from two patients with late onset of erythromelalgia-like symptoms, including pain, signs of small fibre neuropathy and rare genetic variants of Nav 1.9 (N1169S, I1293V) were assessed by microneurography recordings of cutaneous C-nociceptors. Results: Compared to patients with comparable pain phenotypes, there was a tendency towards more activity-dependent slowing of conduction velocity in mechano-insensitive C-nociceptors. Hyperexcitability to heating and electrical stimulation were seen in some C-nociceptors, and other unspecific signs of increased excitability including spontaneous activity and mechanical sensitization were also observed. Conclusion: The findings may be compatible with increased C-nociceptor excitability based on increased Nav 1.9 function, but the functional roles of these genetic variants are still unknown.
Cortical spreading depolarizations are associated with metabolic derangement in spontaneous intracerebral hemorrhage

Christian K. Friberg

Objective: Recently, the clinical significance of cortical spreading depolarizations (CSDs) in secondary brain injury was described regarding subarachnoid and traumatic brain injury (ref). However, the significance of CSDs after spontaneous intracerebral hemorrhage (sICH) remains unknown.

Methods: Patients with sICH meeting the inclusion criteria were enrolled in this prospective observational study as part of the COSBID (Co-operative Study on Brain Injury Depolarisations) initiative. Electroencephalography (EEG) was used to detect CSDs using AC and DC amplifiers. The duration of electrophysiological depressions of the EEG signal was calculated using the power integral (0.5-45Hz; 60 sec time constant decay). Depression duration was defined as the time between depression onset and the start of recovery.

Brain tissue oxygen tension (PbtO2), cerebral blood flow (CBF), cerebral metabolism (micro dialysis) and intracranial pressure were monitored in the region of perihematomal edema (PHE).

Results: 18 patients were analyzed. Hematoma evacuation (ICH volume: 54 [33-69] ml) was performed in 17 patients, 1 subject underwent craniectomy only. Patients were 60 (55-67) years old and 38% female. Monitoring time per patient was 10 (6-14) days. A total of 129 CSDs with 16 (10-29) minutes EEG depression were observed in 66.7% (n=12) of patients. 84% (N=15) of patients showed marked expansion of PHE by 25 (10-50) ml within 3-6 days after bleeding. Neuromonitoring probes were 35 (23-58) mm distant from the EEG strip. CSDs occurred in 73% (N=11) of patients with PHE expansion.

Interval between CSDs was 98 minutes (25-308). CSDs were directly associated with a significant decrease of PbtO2 (-4mmHg [-3;-7]; duration 10[5-23] minutes) in 68% (52/77), CBF changes in 95% (19/20). Metabolic derangement with an LPR>40 was observed in 80% (80/100) at the time of CSDs. PHE expansion was observed in all patients with spreading convulsions (N=2) and patients with repetitive CSDs occurring as clusters (N=3).

Conclusions: CSDs are common in sICH patients and associated with perihematomal PbtO2 decreases and metabolic derangement. Clusters of CSDs in particular seems to be associated with detrimental metabolic changes of the perihematomal brain tissue.
Electroencephalography and delirium in critical illness
Rikke Malte Nielsen

Objective: Delirium in critical illness is an acute brain dysfunction associated with significantly increased morbidity and mortality (HR 3.2;1 after 6 months). Severe cognitive sequelae seem to persist post-discharge. Delirium affects an estimated 80% of patients in the intensive care unit (ICU) but a vast majority remain undiagnosed. The pathophysiology is insufficiently elucidated, however associated with systemic inflammation, changes in cerebral blood flow and metabolism, altered neurotransmission, increased sympathetic activity and cortisol secretion during critical illness. Whether delirium can be verified by preceding continuous electroencephalographic recordings (cEEG) is unknown yet plausible as critical illness induces alterations in the blood-brain-barrier and cell membranes. Furthermore analysis of background rhythm frequency and relative theta-band-power in quantitative EEG has been suggested as a potential predictor of cognitive impairment in patients after cerebral infarcts. Methods: 77 critically ill patients admitted to a medical ICU are monitored with cEEG for 4-7 days. Sedation levels are assessed by RASS (Richmond Agitation and Sedation Scale). Screening for delirium is applied every other hour during daytime using the CAM-ICU (Confusion Assessment Method in the Intensive Care Unit). Upon every CAM-ICU score the preceding EEG is analysed in two minute epochs for pre-defined signature changes. Results: Data suggests generally increased theta- and delta-band power correlating with clinical delirium. Delta coherence peaks primarily in the occipital area whereas theta coherence peaks prefrontally and midline. Frontal Intermittent Delta Activity (FIRDA) occurs and seems to persist after clinical reconvalescens from delirium. Conclusion: Evaluating quantitative EEG for potential reliable predictive biomarkers could be valuable in diagnosing, taking precautionary measures, and further studying delirium as well as constitute a basis for future trials.
Abstract No: 3

Comparison of quantitative MUAP analysis obtained with two different decomposition systems: MultiMUP and EMGTools

Merle Ööpik

Objective: Quantitative Motor Unit Action Potential (MUAP) analysis includes measurements of mean duration, amplitudes and shapes of at least 20 MUAPs sampled at several sites in the muscle. Different MUAP analysis systems (MASs) use different algorithms for extraction of MUAPs. We compared MUAP analysis with two different MASs, Keypoint.NET (Natus) and EMGTools, both used in a clinical setup.

Methods: The same EMG signals were recorded over 5-sec epochs with both MASs from 39 muscles in 22 unselected patients (aged 39-91 years) examined by one of us (MÖ), who was blinded to the results. Both MASs presented the user with the detected MUAPs for editing, and duration was manually adjusted if required. In each muscle the mean duration and amplitude was compared with laboratory age-matched controls.

Results: Eleven different muscles were examined dictated by the clinical picture. The mean number of MUAPs per muscle before vs. after editing was 31/23 for MultiMUP and 47/37 for EMGTools. MUAP duration was normal in 32 and prolonged in 6 muscles in both MASs. In one muscle (3%), MUAP duration was slightly prolonged in MultiMUP, but normal in EMGTools. The mean difference in duration found at the systems was 0.8 ms (0-1.9 ms). In 14 muscles MUAP duration was longer in EMGTools (mean 0.7 ms (range 0.1-1.4)), and in 24 in MultiMUP (mean 0.8 ms, range 0.1-1.9). The mean MUAP amplitude was always higher in MultiMUP, the mean difference being 192 µV (range 18-681 µV). Conclusion: EMGTools detected more MUAPs in each muscle than MultiMUP. In 97% of muscles there was diagnostic agreement regarding MUAP duration between the two MASs. The lower mean amplitude and the higher number of muscles with shorter MUAP duration in EMGTools compared to MultiMUP, indicates that the former system detected more small MUAPs. This may be an advantage in the diagnosis of patients with myopathy.

Abstract No: 9

Short-segment nerve conduction studies and ultrasound of the ulnar nerve across the elbow – a study in healthy subjects

Hanne Rheder Ellegaard

Introduction: Ulnar nerve entrapment is diagnosed by nerve conduction studies. The diagnostic criteria include decreased conduction velocity (CV) across the elbow. Ultrasound showing an enlargement of the cross sectional area (CSA) has also been suggested for diagnosis. In patients short-segment nerve conduction studies are rarely performed due to unclear reference values. We aimed to examine the ulnar nerve with ultrasound and short-segments studies in healthy subjects.

Methods: We included 43 healthy subjects with no clinical signs or symptoms of ulnar nerve entrapment. Inching was performed and CV was calculated in the forearm and in segments of 2 cm across the elbow, starting from 7 cm proximal to the sulcus (between the medial epicondyle and olecranon) to 3 cm distal to the sulcus. Furthermore, the CSA was measured in the same segments, i.e. at 5 and 2.5 cm proximal to the sulcus, at the sulcus, at 2.5 and 5 cm distal to the sulcus, and the pisiform bone.

Results: In both nerve conduction studies and in ultrasound a significant difference at the sulcus was seen compared to the rest of the arm. CV across the sulcus was 37.27 m/s (SD 9.42), and was significant lower compared to the remaining segments. Mean CV in the remaining segments ranged from 63.97-69.95 m/s. CSA at the sulcus was 6.81 mm2 (SD 1.50), and was significant enlarged compared to the remaining CSA measurements, which ranged from 5.30-6.41 mm2.

Conclusion: We found a significant decrease in the CV across the sulcus and an enlargement of the CSA in the sulcus in healthy subjects with no signs or symptoms of ulnar nerve entrapment. This might be explained by the fact, that the ulnar nerve is more exposed at the sulcus than in the rest of the arm. This fact becomes important when normative values are determined for diagnosis.
**Quantitative electroencephalography reactivity in comatose neurosurgical patients.**

**Mads Qvist Ebbesen**

Objective: EEG-reactivity (EEG-R) to pain, auditory and visual stimuli is an important prognostic marker for comatose neurosurgical patients. The method for evaluation of EEG-R relies on a visual inspection prone to subjective interpretations and an objective quantitative method is desirable. In this study we investigate if it is possible to perform a quantitative evaluation of EEG-R and establish cut-off values.

Methods: Twenty-seven comatose patients at the neurosurgical intensive care unit had a 24-hour EEG-monitoring as screening for non-convulsive seizures or status. Standardized pain, auditory and visual stimulations in 30-second epochs were done. EEG-power in the delta, theta, alpha and beta frequency bands at each EEG electrode were calculated. The ratio between the power in a stimulation epoch and the power in the 30-second epoch of rest immediately before was calculated. EEG-R was measured as the average power ratio of four pain stimulation epochs. In order to establish the measurement error we calculated power ratios between four 30-second epochs and the 30-second epochs before.

Results: The prediction intervals for ratios at different electrodes and different frequency bands varied with lower limits ranging from 0.56 to 0.89 and upper limits from 1.15 to 1.57. The θ-band showed the highest variation. At the C3 electrode 11/27 patients showed significantly increased power in the delta-band during pain stimulation. Six showed increased power in the theta-band and two showed decreased power in the theta-band. Ten showed increased power in the alpha-band and 19 in the beta-band.

Conclusion: It seems possible to quantify EEG-R and establish reference limits in the different frequency bands. There are different patterns of EEG-reactivity in different frequency bands in different patients. Our study will further investigate whether there is correlation between EEG-R and prognosis of the patients.

**Ephapse from II-afferents to static gamma and beta efferents causes complex repetitive discharges**

**Juhani Partanen**

Objective: To study the origin and characteristics of complex repetitive discharges (CRDs). Methods: A number of CRD:s were recorded in routine ENMG studies and the variables were compared between CRDs in myofascial pain and CRDs in neuropathies. The propagation of CRDs was studied with multi-channel EMG with needle electrodes in parallel with muscle fibres in the given muscle. Results: There were no significant differences between CRDs in myofascial pain and CRDs in neuropathies. The multi-channel EMG pointed out that CRDs were mostly local and did not propagate to other EMG channels. However, some CRD:s with usually low frequency propagated to all channels as motor unit or fasciculation potentials do. Conclusion: CRDs were supposed to originate from intramuscular ephaptic circuit (1), driven by a spontaneously active denervated muscle fibre as a pacemaker, without any synapse. However, in that case the CRD potentials should propagate along the muscle fibres as motor unit potentials do. Denervated muscle fibres do also have a long refractory time (3), which prevents the high frequency of potentials, common in CRDs. The muscle spindles II-afferents branch widely in the juxtaequatorial regions of either side where they may intermingle with motor terminals (2). This creates an ideal circumstance for ephapse in favourable conditions, for example when K+ ions are accumulated in the periaxial space of the muscle spindle. We do not know the jitter values of the gamma efferent trail ending synapse, but these may be lower that the jitter values of the alpha efferent synapses.

**A prospective and blinded study of ultrasound in patients with neuromuscular disorders**

**Maria Thelin Johansson**

Objective: Today the standard of diagnosing Neuromuscular disorders (NMD) such as amyotrophic lateral sclerosis, involves electrophysiological measurement with needles and electricity. Detected by
electrophysiological measurements, fasciculations are a diagnostic feature of ALS. These brief, involuntary muscle twitches are also detectable using Ultrasound (US). The aim of this study is to investigate the clinical feasibility of US in diagnosis of NMD such as ALS. Methods: All consecutive patients referred to the Department of Clinical Neurophysiology at Aarhus University Hospital under the suspicion of motor neuron disease or ALS, polynuropathy and myopathy, are invited to participate in the study. There will be a follow-up at 3 months after baseline examination. The reference group will be a randomly selected group of healthy controls, age between 20 and 90 yrs. The US examination will follow a standardised schedule, and scanning will be performed on selected muscles in standardised positions. Results: We have so far invited 30 patients to participate in the study, and we have so far included 20 patients. Out of the 20 patients, the referring diagnosis was in seven cases ALS, eleven cases polynuropathy (pnp) and two cases myositis or myopathy. The electrophysiological diagnosis of these patients was: probable ALS (five patients), pnp (five patients). In eight patients the electrophysiological examination was normal and in two patients we found a mononeuropathy (one ulnar nerve and one peroneal nerve). Collected data from the US examination will later be de-identified and evaluated in a blinded fashion. Conclusions: We expected that 1) Fasciculations measured by US, are more frequent in patients with NMD’s compared to healthy controls. We expect to find this pattern clearly visualised by US. 2) Fasciculations are prominent in the genioglossus muscles, in patients suspected to have ALS with bulbar affect.

Abstract No: 17
Clinical Utility of Pelvic Floor Electrophysiology
Berit Sørensen

Clinical Utility of Pelvic Floor Electrophysiology Sørensen B1,2, Fuglsang-Frederiksen A1, Tankisi H1
1Department of Clinical Neurophysiology, Aarhus University Hospital, Denmark. 2Department of Neurology, Viborg, Aarhus University Hospital, Denmark. Objective: Since clinical neurological examination of the sacral nervous system is neither sensitive, nor specific, neurophysiologic tests of pelvic floor may have important implications in the diagnosis of pelvic floor disorders. The aim of this study was to examine whether pelvic floor electrophysiological tests contribute to diagnosis or initiated further examinations or treatment of pelvic floor disorders. Methods: Clinical information and pelvic floor electrophysiological data, of 18 patients consecutively referred to the Department of Neurophysiology, Aarhus University Hospital for assessment of possible pelvic floor disorders, were analysed retrospectively. The results of pelvic floor electrophysiological tests, including somatosensory evoked potentials (SEPs) of pudendus nerve, penilo/cliterocavernosus reflex (BCR), electromyography (EMG) of bulbocavernosus and external anal sphincter muscles were analysed. The clinical course of the patients and the influence of the electrophysiological results to clinicians’ decision for further investigations and therapy were reviewed from patient files. Results: Five of 18 patients had no abnormalities in any of the electrophysiology tests performed. Ten of the 18 patients had abnormal pelvic floor electrophysiology tests showing pudendal nerve neuropathy, and all 10 had delayed latencies or absent responses of the penilo/cliterocavernosus reflex. EMG of bulbocavernous and external sphincter ani muscles were abnormal in 6 and 8 patients, respectively. For 10 patients the electrophysiological assessment confirmed the referral theory that there was pathology of the pelvic floor, thus contributing to the diagnosis, initiate further examinations or treatments or both. For the remaining patients the assessment made it possible for the clinician, to change the diagnosis, initiate further examination, or treatment. Conclusion: Pelvic floor electrophysiology may contribute to the diagnosis of patients with symptoms of pelvic floor disorders. The penilo/cliterocavernosus reflex is suggested to be a sensitive test in diagnosing pelvic floor disorders. In this group of patients the pelvic floor tests had an impact on the diagnosis or further course for all the patients.
Abstract No: 18
**EEG during anesthesia**
Ville Jäntti

For obvious clinical reasons the anaesthesiologists have been obsessed to find an indicator of unconsciousness during general anaesthesia. The movement response based MAC value is now accepted as a misunderstanding, because it essentially measure spinal cord reflexes. In the 1990’s the idea of using EEG spectral edge, which decreases monotonously in deepening anaesthesia, was further developed to commercial indexes like BIS or Entropy. The development essentially involved compensation of the log-linear amplitude distribution of power spectrum with narrow band or logarithmic analysis. Two decades of intensive research followed, which ended in disappointment. The reasons were simple: in most of the studies the raw signal, EEG was not analyzed, and the researchers did not understand principles of EEG. Failure to understand the physiology, biophysics and signal processing of EEG at the same time when manufacturers refused to reveal the details of signal analysis resulted in a vast number of scientifically meaningful publications. Arousal reactions and epileptiform patterns rendered the indexes meaningless. Concepts like bispectrum and entropy only served commercial purposes. Different anaesthetics generate very different types of anaesthesia and these can be seen in their effect on reactivity as well as autonomic nervous system. Simultaneous recording of other physiological signals like ECG and functional measures like fMRI and PET are necessary to reveal these. Today it is understood that general anaesthesia does not necessarily mean unconsciousness: the patient may safely have cognitive function during operation like in local anaesthesia. This sets new challenges for anaesthesia research: it must develop into a part of cognitive neuroscience, cognitive anaesthesiology, and EEG will be an increasingly important tool in that development.

BACK TO PROGRAM

Abstract No: 19
**Normal material for duration of CMAP in hand muscles**
Steffen Birk

Objective: Some studies suggest that duration of compound muscle action potential (CMAP) is increased in demyelinating polyneuropaties [1] and critical illness neuro-myopathy (CINM) [2]. This parameter may thus play a role in the electrophysiological evaluation of these diseases. However, little is published regarding normal values for CMAP duration [2, 3], and hence the objective of the study was to establish our own normal values for this parameter in the abductor pollicis brevis (APB) and abductor digiti minimi (ADM) muscles. Methods: 41 participants were included: 28 data sets from patients previously included in a normal material in our department and 13 additional healthy participants. Demographic data: 16 male, 25 female. Mean age 41 years (range 18-81). No participants had symptoms or signs of polyneuropathy, carpal tunnel syndrome, ulnar nerve lesion or other neuromuscular disease. The median and/or ulnar nerve in the dominant extremity was electrically stimulated and the duration of the negative phase of CMAP of APB and ADQ was measured with surface electrodes. Height and weight was recorded. Multiple linear regression analysis was used to investigate effect of height, weight and age. T-tests were used to study differences between the sexes. Results: Mean CMAP duration was 5.0 ms ± 0.68 ms SD for APB and 5.3 ms ± 0.58 ms SD for ADM. The values were normally distributed. For APB, there was a small statistical effect of age, but the effect was quantitatively negligeable. No effects of height, weight or sex could be shown. Conclusion: The present normal values are comparable to previous studies with one study including 54 patients (APB mean 5.4 ms, range 3.0 – 7.8 ms; ADM mean 6.0 ms, range 5.0-7.7 ms) [2] and another including145 patients (APB mean 4.8 ms, ADM mean 5.1 ms) [3]. Further studies are needed to investigate utility of this parameter in the electrophysiological evaluation of demyelinating polyneuropathies and CINM.

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Abstract No: 20
Differential “set-shifting” behaviour in idiopathic generalized epilepsy syndromes
Steffen Birk

Objective: To evaluate specific aspects of executive function in patients with different types of idiopathic generalized epilepsy. Methods: 15 patients with juvenile myoclonic epilepsy were matched by age, sex and educational level with 15 healthy controls and 15 patients with other types of idiopathic generalised epilepsy. FMRI (3T) was performed with a computerised version of Wisconsin Card Sorting Test. Data were analysed in a event-related design with special emphasis on BOLD activation after negative feedback, which corresponds to set-shifting behaviour. Effects of group membership were analysed with single-factor ANOVA with clusterlevel threshold detection using Monte Carlo simulations. Reaction times and errors were recorded and classified. Results: Regarding behavioural data, juvenile myoclonic epilepsy patients had significantly longer reaction times and more errors than other idiopathic generalized epilepsy and healthy controls. When analysing BOLD activation after negative feedback, juvenile myoclonic epilepsy patients had significantly less BOLD activation than healthy controls in left medial frontal gyrus (BA6), in right prestriate (BA19), and in the left claustrum (in close connection to BA47 and BA13). Except in BA13/47, the difference in BOLD activation between juvenile myoclonic epilepsy and other idiopathic generalized epilepsy did not reach statistical significance. Conclusion: The performance data confirms impaired executive functions in juvenile myoclonic epilepsy compared to IGE. FMRI data show decreased BOLD activation in juvenile myoclonic patients in areas previously reported to be important in set-shifting behaviour. Differences between the two idiopathic epilepsy syndromes were less clear, possibly due to insufficient statistical power.

Abstract No: 23
Aetiology and Electrophysiological Classification of Polyneuropathies
Ajbanis Murtuzova

Objective: Electrophysiological classification of PNP may provide invaluable information about the aetiology. We aimed in this study to examine aetiologies and pathophysiological classification of patients referred for electrodiagnostic examination for PNP. Methods: Clinical information and Nerve Conduction Study (NCS) data of 291 patients consecutively referred by the Department of Neurology for assessment of PNP to the Department of Neurophysiology, Aarhus University Hospital, were analysed retrospectively. The aetiological causes and clinical diagnoses were reviewed from patient files. In all patients, bilateral peroneal and tibial motor NCSs, sural sensory NCS, and unilateral median and ulnar motor and sensory NCSs were done. The classification of PNP was first done as sensory, motor and sensory-motor and then further classified as axonal, demyelinating and mixed according to ESTEEM criteria (Tankisi et al, 2005). Results: In 130 patients, there were no signs of PNP. Of these, in 68 there was no etiological cause while in 14 small fiber neuropathy, in 15 radiculopathy or spinal stenosis and in 32 patients, metabolic or systemic disorders were found to cause the symptoms. In 161 patients, a sensory (21), motor (10) or sensory-motor (130) PNP was diagnosed. Sensory PNP were either axonal (17) or unclassified (4). Motor PNP were demyelinating (6) or unclassified (4). Of 130 sensory-motor PNP, 36 were demyelinating, 39 axonal, 21 mixed and 34 unclassified. The aetiologies of the 161 PNP patients were inflammatory (38), diabetic (23), hereditary (15), paraneoplastic (11), alcoholic (7), B12 deficiency (6), other (13) or idiopathic (48). Inflammatory PNP were mostly demyelinating (74%), idiopathic PNP axonal (50%) and diabetic PNP unclassified (52%); however in the other groups, pathophysiological classification of PNP were equally disturbed. Conclusion: Demyelination guides the clinicians towards inflammatory PNP. However, a broad spectrum of etiological factors should be checked in other classifications as well as patients showing normal NCSs if they symptoms of PNP.

BACK TO PROGRAM
Conclusion. Varicectomy may cause neuropathic complications but it rather infrequently gives rise to persistent neuropathic pain. Subjective sensory symptoms should be verified
Leena Puksa

Objective. The aim of the study was to investigate occurrence and risk factors of sensory nerve injuries and neuropathic pain in patients undergoing varicectomy, and to evaluate the diagnostic sensitivity of sensory neurography and clinical sensory tests in detecting the neuropathic complications.

Methods. 38 patients, 32 women (mean age 41.94, SD 11.44) and 6 men (mean age 39.17, SD 12.51) were prospectively studied before and within 30 days after varicectomy. The patients who showed neuropathic symptoms or signs were further investigated after 3 and 6 months. Symptoms were evaluated using a symptom chart drawing and Visual Analogue Scale (0-100) for pain. Clinical sensory tests included warm/cold discrimination using thermal rolls and tactile as well as mechanical pain detection thresholds measured with Semmes-Weinstein monofilaments and method of limits. Pain wind-up was tested with the monofilament giving the first slight sensation of pricking pain. Sensory neurography of the sural, saphenous, and superficial peroneal nerves was done bilaterally with standardized protocol.

Results. Five of the 38 patients (13.2%) reported postoperative neuropathic symptoms: neuropathic pain and altered sensibility: two patients within saphenous, one within superficial peroneal and saphenous, and two patients in all 3 studied nerve distributions. In 4 patients, sensory alterations and pain symptoms disappeared by 6 months after surgery. In one patient (2, 6%), neuropathic pain persisted after severe partial axonal saphenous nerve lesion. Neurography findings were in line with subjective symptoms and confirmed all nerve injuries (demyelinating), severity of the lesion was moderate/ severe). Clinical sensory tests were able to detect the nerve injuries out of 3 patients after surgical treatment, while neurophysiologic methods were normal and the patients were without neuropathic pain. Conclusion. Varicectomy may cause neuropathic complications but it rather infrequently gives rise to persistent neuropathic pain. Subjective sensory symptoms should be verified with neurophysiologic recordings as clinical sensory testing may remain falsely negative in these iatrogenic injuries.

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Abstract No: 27

Composite nerve conduction Z scores for studies of polyneuropathy
Kristian Bernhard Nilsen

Objectives. Nerve conduction studies are widely used to confirm clinical polyneuropathy and should be included as part of the definition of polyneuropathy for clinical research. Grading of abnormality and evaluation progression is difficult. Calculation of a compound Z-score by averaging Z-scores from single nerve conduction parameters may be helpful. We wanted to find the best subset of nerve conduction parameters to be used in studies of diabetic neuropathy.

Methods. Standard nerve conduction studies on 4 motor and 5 sensory nerves were performed on 66 patients with type I diabetes mellitus (T1DM) with a median neuropathy impairment score of 10 (interquartile range = 12). Reference ranges were based on data from 366 healthy subjects, with available data varying between parameters. Composite scores were calculated as an average of Z-scores of the included variables. The composite scores that best distinguished between a separate set of healthy controls matched for age, sex and educational level (n = 28) and diabetic subjects were identified using Students t-tests and abnormality rates (sensitivity, specificity and accuracy).

Results. Twenty different composite scores were calculated, based on 2-86 variables. Sensitivity for polyneuropathy varied between 46% and 72%, with median prevalence of 69%. All composite scores differentiated patients from controls (5.1 < t < 8.1). A composite score based on conduction velocity discriminated best, while experience-based composite scores discriminated generally discriminated less. A composite score based only on peroneal nerve conduction velocity and sural nerve amplitude discriminated well. Conclusion. Prevalence of diabetic polyneuropathy varies greatly depending on the selection of investigated nerves in nerve conduction.
studies. Composite Z scores may be ideal for longitudinal studies of polyneuropathy. The selection of neurophysiological parameters may be of great importance when studying potential treatment effects.

Abstract No: 28

**Neurography, QST and CHEP in the diagnosis of neuropathic complications after laser and fibrovein treatment of varicose veins**

Satu Laaksonen

The exact incidence of nerve injury and neuropathic pain after surgical treatment of varicose veins remains unclear: earlier studies have been based on clinical examination or subjective questionnaires; incidence figures up to 25% have been reported for altered sensibility. The aim of our study was to detect neurological side effects after treatment of varicose veins and compare different treatment techniques with each other. 24 healthy patients were studied before and after 4 weeks of laser (8), fibrovein (1) or laser + fibrovein (15) treatment of varicose veins. If patients had symptoms / neurophysiological findings at 1st post operative study, the studies were repeated 3, 6 and 12 months after the operation. Assessment of subjective sensory symptoms were studied with pain and symptom chart, clinical sensory tests from both legs included warm/cold discrimination with thermal rolls from the operation area and contralateral site n. saphenus, n. suralis and n. peroneus superficialis distribution, neurography of these nerves were also bilaterally studied. Tactile detection threshold, tactile-pain detection and pain wind-up was studied from the same three distributions with Semmens-Weinstein monofilaments. Also sharp-blunt differentiation and QST that included included warm/cold, warm-pain and cold-pain detection thresholds were studied. Contact heat evoked potential (CHEP) were studied stimulating peroneus superficial and n. saphenus area bilaterally. Four (4) patients reported reversible subjective pain symptoms after treatment. Three (3) patients had symptoms after laser-fibrovein (3/15) treatment and one (1) after fibrovein treatment only. Preliminary results show that subclinical small fiber hypofunction was postoperatively observed in 2 laser treated and 2 laser+ fibrovein treated patients in QST. Laser treatment alone seems to be a safe method to treat varicose veins, irreversible heat induced nerve lesions were not seen.

Abstract No: 29

**Myopathic EMG changes in patients with Lambert-Eaton myasthenic syndrome.**

Clarissa Crone

Objective: Lambert-Eaton myasthenic syndrome (LEMS) is a debilitating but treatable disease, which often precedes a malignancy. The condition may clinically mimic myopathy and a few reports have described myopathic EMG changes in LEMS patients (pts) (1,2). These studies did however either include only very few pts (1) or did not report motor unit potential (MUP) duration or compare with normal values (2). The aim of the study was therefore to investigate if in LEMS pts myopathic EMG findings could be found using quantitative EMG (qEMG). Patients: 8 pts were included. All had experienced weakness, fatigue and dry mouth. A general proximal weakness (1-4+ MRC) was found in 7 patients. 6 pts had antibodies against voltage gated calcium channels and 6 were diagnosed with lung tumor. Muscle strength improved in all pts after 3,4 diaminopyridine (3,4 DAP) treatment. 7 pts showed reduced amplitude of CMAPs, a decrement during 3Hz stimulation and a clear increment during tetanic stimulation (50Hz) or after 10 s of maximal voluntary contraction (MVC). In one patient, who was not tested until after initiation of 3,4 DAP treatment, the CMAP amplitude was normal and no increment or decrement was seen. Methods: 20-30 different MUPs were collected in proximal arm and/or leg muscles with a concentric EMG needle electrode, and the duration of each potential was measured by marking start and end of the MUP at high amplification. The mean duration of simple as well as all potentials was calculated. The mean duration of simple potentials was considered significantly shortened if decreased by more than 20% from the mean of our laboratory’s own aged-matched normal
Conclusion: EMG abnormalities mimicking myopathy may often be found in patients with LEMS. Blocking of some, but not all nerve fiber terminals belonging to a motor unit may cause the shortened duration of MUPs. Our finding that MUP duration normalized during 3,4 DAP treatment in one LEMS pt supports this hypothesis.

Abstract No: 30

**CMAP duration in patients with critical illness myopathy**

Clarissa Crone

Objective: Many patients with sepsis and multi organ failure (MOF) develop tetraparesis while in intensive care unit (ICU). It has been debated whether the main organ of failure is peripheral nerve or muscle, but many recent results point towards a muscle dysfunction. The aim of the study was to measure the duration of compound muscle action potential (CMAP) in patients who were considered to have critical illness myopathy (CIM) in order to investigate if this measure may contribute to diagnosing CIM. Patients: 27 patients who had had septic shock and MOF and who developed severe tetraparesis in ICU were included. All had decreased peak-peak amplitude of CMAP of abductor pollicis brevis muscle (APB) and/or abductor digiti minimi muscle (ADM) and decreased duration of MUPs in brachial biceps and/or medial vastus muscle. None had signs of sensory polyneuropathy. The amplitude of the tibialis anterior muscle responses evoked by direct muscle stimulation (dMS) was below normal (3mV) and all responses were polyphasic in the 23 pts tested this way. Methods: The median and/or ulnar nerve was electrically stimulated and the duration and amplitude of the negative phase of CMAP of APB and/or ADM was measured with surface electrodes and compared with our normal values. Results: In 23 out of 27/26 patients an abnormal duration of APB/ADM CMAP was found. Mean duration of APB CMAPs in all pts was 11,3 ms (4,8-34,3 ms, upper normal limit 6,3 ms). Mean duration of ADM CMAPs in all pts was 10,3 ms (5,4-17,8 ms, upper normal limit 6,4 ms). The mean amplitude of all APB CMAPs was 1,0 mV (0.1-3,2 mV). The mean amplitude of all ADM CMAPs was 1,9 mV (0.1-3,6 mV). All CMAPS were monophasic. Conclusion: CMAP duration is prolonged in most patients with clinical and electrophysiological signs compatible with CIM. The prolonged duration may be caused by decreased muscle fiber conduction velocity, but a prolonged CMAP duration may also be seen in acute inflammatory demyelination neuropathy (AIDP). However, in the absence of demyelinating signs in nerve conduction studies a prolonged CMAP duration supports the CIM diagnosis. Future studies will show if measurement of CMAP duration is a useful tool for diagnosing and monitoring CIM and whether it may predict prognosis in critically ill patients with tetraparesis developed in ICU.

Abstract No: 31

**High-resolution ultrasound adds valuable information when diagnosing patients with symptoms of carpaltunnel syndrome**

Janus Kaufmann Lindqvist

Objective In 2014, about 48% of patients referred with the diagnosis of carpaltunnel syndrome (CTS) to Department of Clinical Neurophysiology, Glostrup Hospital, ended up with the diagnosis of CTS. 46% got the diagnosis Observatio. Here it is evaluated whether supplemental High Resolution Ultrasound (HRUS) contributed to diagnosing these patients by revealing specific pathological findings in relation to the patients’ medical history and/or symptoms.  Method Electrophysiological/ultrasound reports from 2014 were reviewed. Reports from patients referred with a diagnosis of CTS, who were investigated with a combination of electroneurography and HRUS, were rated.  Results In 2014, 94 patients were
referred with a diagnosis of CTS and were investigated with electroneurography in combination with HRUS. Of these, 34 showed classical electrophysiological findings of CTS. 29 were confirmed with ultrasonographic findings of increased wrist-to-forearm ratio and flattening beyond the transverse ligament (85%). 2 showed other specific changes with further information (traumatic neuroma) (6%), 3 showed increased wrist-to-forearm ratio but not flattening. Thirty (30) patients showed unspecific electrophysiological findings, which lead to the diagnosis Observation. Of these, 14 had findings of compression revealed by ultrasound (46,6%). 8 patients had other specific changes with further information (26,6%), 8 showed unspecific changes. The remaining 30 patients had normal electrophysiological findings also leading to the diagnosis Observation. In this group, 5 had findings of compression using ultrasound (17%), while 4 had specific changes with further information (13%), 21 were normal or showed unspecific changes.

Conclusion HRUS was able to show ultrasonographic signs of compression of the median nerve in the carpal tunnel in 32% of patients referred with the diagnosis of CTS when an electrophysiological examination was normal or showed unspecific changes.

Abstract No: 32

High-resolution ultrasound in post-surgery persistent carpal tunnel syndrome
Janus Kaufmann Lindqvist

Objective   Despite classical symptoms and/or electrophysiological findings of carpal tunnel syndrome (CTS) before surgery, some patients do not benefit from surgical carpal tunnel release. Here it is evaluated whether High Resolution Ultrasound (HRUS) is a valuable supplement to electrophysiology in case of suspected post-surgery persistent CTS. The question is: can HRUS reveal persistent compression of the median nerve, complete decompression, or other specific pathological findings corresponding to the patients’ medical history and/or symptoms. Method Electrophysiological/ultrasound reports from 2014 were reviewed. Investigations with combined electroneurography and HRUS referred with a suspected post-surgery persistent CTS were rated. Results In 2014, 22 patients with suspected post-surgical CTS were investigated with electroneurography in combination with HRUS. Of these, 10 patients showed electroneurographic changes typical for CTS or changes strongly suggesting CTS. Of these, 8 patients showed remaining transverse ligament and persistent compression of the median nerve. 1 patient showed full decompression of the median nerve and 1 patient showed unspecific changes. 8 patients showed unspecific electrophysiological changes, not diagnostic for CTS. Of these, 5 patients showed full decompression of the median nerve, 1 showed remaining transverse ligament and persistent compression of the median nerve, 1 was decompressed but showed scar tissue with focal compression of the nerve, and finally, 1 showed an additional site of compression more proximally at the level of the wrist. 4 patients had normal electrophysiological examinations, and in all of these HRUS showed full decompression. Conclusion HRUS was able to secure the differentiation of post-surgery persistent CTS in patients without benefit of release by visualizing whether remaining transverse ligament and persistent compression of the median nerve was present or if the median nerve

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Effect of Deep brain stimulation and medication on somatosensory function in Parkinson’s disease
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Objective: Deep brain stimulation (DBS) of subthalamic nucleus (STN) significantly alleviates cardinal motor symptoms and improves quality of life in Parkinson’s disease (PD). DBS and medication have been shown to affect sensory perception differently in PD patients. We used somatosensory evoked fields (SEF) to investigate this difference at the level of cortical processing. Methods: Nine DBS implanted PD patients were recruited. Magnetoencephalography (MEG) assessments were performed “OFF medication, ON DBS”, “OFF medication, OFF DBS” for each half hour until two hours and then “ON medication, OFF DBS”. For each hour, patient symptoms were evaluated
with the UPDRS-III. Repetitive median nerve stimulation was performed with current set at the level of an observable twitch of the thumb. Inter-trial phase coherence (ITPC) values, a measure of phase synchrony across trials, were calculated. Clustering-based univariate F-statistics was used to discern differences in phase synchronies. Results: We found significant differences in early high gamma (60-100 Hz) phase synchronisation between conditions. Contrasts revealed that the effect was mainly driven by differences between the DBS ON vs DBS OFF and DBS ON vs Medication ON conditions. The phase synchronies showed a differentiated effect between DBS and medication. This effect was observed on the contralateral hemisphere and was more frontal compared to the position of the parieto-central N20m component. There was no significant accompanying change in evoked power. Conclusion: Significant differences in gamma synchronisation during DBS and medication may reflect differences in mechanism although they have a similar effect on PD motor symptoms.