1. The Sensitivity of Stimulated-SFEMG in the Paediatric Myasthenia by Two Methods of Data Analysis. V. Narwani, T. Tidswell, and M. Pitt. (Royal Free Hospital and Great Ormond Street Hospital, London, UK).

Aim: The diagnosis of paediatric myasthenia is supported by abnormal stimulated single-fibre electromyography (stimulated-SFEMG) from orbicularis oculi during facial nerve stimulation at 10Hz. This study re-assessed the sensitivity of stimulated-SFEMG for NMJ instability in a new patient cohort compared to clinical outcome.

Methods: Stimulated-SFEMG data from 167 children referred to Great Ormond Street Hospital (December 2008 to March 2011) were analysed with MCD and correlational algorithms; diagnosis, from clinical records, was defined as either myasthenia (genetic confirmation, ACh-R antibody positive or good response to treatment) or non-myasthenia (alternative diagnosis made).

Results: Outcome data was available for 74/167; 22 had myasthenia (11 genetically confirmed, 4 autoimmune and 7 treatment responders). MCD analysis was abnormal in 21/22 patients with myasthenia and 22/52 non-myasthenics; correlation analysis was abnormal in 22/22 patients with myasthenia and in 2/52 non-myasthenics.

Conclusion: Stimulated-SFEMG is a sensitive test for neuromuscular junction instability, with further evidence that a new, automated, correlation analysis method significantly reduces false positives.


A waves are commonly seen in demyelinating polyneuropathies, but the incidence of A waves in healthy subjects is uncertain, especially in the elderly. A-waves are thought to arise by either ephaptic transmission between adjacent motor axons or branched motor axons.

We retrospectively analysed nerve conduction studies in 182 healthy elderly subjects (359 nerves) to determine the incidence and distribution of A-wave latencies in the tibial and common peroneal nerves.

In the tibial nerve 24.9 % showed at least one A-wave. A-waves were present in only 7.8 % of the common peroneal nerves studied, a significant difference (P = < 0.001). The cause of this difference is unknown.

The distribution of the A-wave latencies in relation to the F-wave latencies was bimodal with peaks at approximately 20 and 55 ms. Review of the published data on S1 nerve root anatomy suggests that at least one of these peaks is caused by A-waves generated in close proximity to the nerve root exit from the spinal canal. Degenerative spine disease may cause subclinical compression of the S1 root giving it a predilection to generate A-waves.


The incidence and pathogenesis of neuropathic changes secondary to the construction of ipsilateral arteriovenous (A-V) shunts in patients with EKSD remain unresolved.

We have therefore prospectively examined radial, median, ulnar, peroneal and sural nerve conduction (between 30-32°C) over 4 years in 11 EKSD patients (age 38-79 years; 9 males) before construction of brachial or radial A-V shunts, in the immediate postoperative period and at intervals over the succeeding 4 years. 3 patients died during the course of the study. Of the remaining 8 patients, 3 had at least one shunt revision.

No patient developed novel ipsilateral neuropathic changes in the immediate postoperative period or up to 2 months postoperatively. 4 of the 8 surviving patients did show a longterm ipsilateral reduction of radial sensory potential amplitudes but all 4 patients had additional evidence of a progressive axonal neuropathy and the reduction was not disproportionate to that of the homologous radial nerve response. All 8 surviving patients showed evidence of an evolving peripheral neuropathy by the conclusion of the study. None of the study patients developed a novel, ipsilateral, focal median or ulnar nerve lesion over the 4 year study period.

The findings for this small cohort thus do not support the notion that A-V shunts are a potent factor in the development of immediate or longterm local neuropathic changes, even in the presence of coexistent peripheral neuropathy arising from the interplay of known neurotoxic factors in ESKD patients.

4. Deep Brain Stimulation of the Thalamic Centromedial Nucleus (CMN-DBS) in Epilepsy. L. Vico, G. Alarcón and A. Valentín. (Institute of Psychiatry, King’s College London and Clinical Neurophysiology Department, King’s College Hospital, London, UK).

Objectives: To evaluate CMN-DBS effects on seizure frequency and patient related outcome (PRO).

Methods: 5 patients with generalised epilepsy and 3 with frontal epilepsy (age 19-53 years) were implanted with CMN-DBS. Seizure frequency and the following PRO questionnaires were assessed before and after implantation: Liverpool Seizure Severity (LSSQ), Patient Weighted Quality of Life in Epilepsy-31 (QoL-31), and the Hospital Anxiety Disorders (HADS).
Results: 2 patients became seizure free, 2 remained with seizures but improved >50%, 2 showed no clear improvement and 2 are still in the blind 6 months period after DBS implantation. No patients with frontal epilepsy became seizure free (1 improved by >50%; 2 had no improvement in seizure frequency). PRO Questionnaires showed improvement in approximately 50% of patients.

Conclusion: CMN-DBS appears to be effective in improving seizure control and PRO in patients with generalised epilepsy and possibly in frontal lobe epilepsy.


We hypothesized that brain pulsation whereby the distance from coil to cortex varies with the cardiac cycle could influence the amplitude of MEPs. Magnetic energy varies as the fourth power of distance and therefore a small change in distance will produce a large change in magnetic field strength. The time course of the pressure pulse during the cardiac cycle predicts that the brain would be closest to the coil 200-400ms after the QRS complex. In 8 healthy subjects, we used the QRS to trigger TMS at delays of from 50 to 750 ms whilst subjects maintained a constant contraction of the FDI muscle. Stimulus intensity was adjusted to produce an MEP of at least 1mV. The mean MEP amplitude at each delay was measured in each subject. To compare MEPs across subjects, MEPs were normalised to the maximum found in each subject. There were no significant differences in the amplitude of MEPs with respect to the cardiac cycle. We conclude that even though the brain pulsates, no effect can be detected in MEP amplitude as a result of this.

6. Efficacy of Standard Parameters for Transcranial Direct Current Stimulation (tDCS). J. Barnes¹, S.R. Jaiser¹, H.M. Lai², S.N. Baker¹ and M.R. Baker¹. (¹Institute of Neuroscience, Newcastle University. ²Department of Clinical Neurophysiology, Royal Victoria Infirmary, Newcastle upon Tyne, UK).

Transcranial direct current stimulation (tDCS) is a simple method of producing persistent changes in cortical excitability with potential therapeutic applications. In most studies a 1mA DC stimulus passed through the brain via two scalp electrodes for 10 minutes is sufficient to alter excitability.

To establish the reliability of these parameters, we tested both anodal and cathodal tDCS to motor cortex in 9 healthy subjects. Motor evoked potentials (MEPs) and 15-30Hz intermuscular coherence (IMC) were used to assess changes in motor cortical excitability. In 5/9 subjects anodal tDCS produced significantly increased MEPs (p<0.05) and non-significant increases in 15-30Hz IMC. Cathodal tDCS significantly reduced MEPs and 15-30Hz IMC in 5/9 subjects. In the remaining subjects tDCS had no significant effect on MEPs/IMC.

Based on our small study, standard published tDCS parameters are only effective at modulating cortical excitability in ~50% of healthy controls. We therefore urge caution in applying tDCS to non-motor cortex, without first verifying its effects on motor cortex in each subject.


Aim: Prolonged EEG techniques (ambulatory EEG or video telemetry) increases clinical episodes detection over routine EEG but the impact on clinical management is unknown. This study assessed the impact of prolonged EEGs on the diagnosis and clinical management of patients with suspected epilepsy.

Methods: 104 consecutive patients underwent prolonged EEG between November 2009 and February 2011. Clinical records from before and after the prolonged EEG were assessed, retrospectively, for changes in diagnosis or clinical management.

Results: A total of 81/104 patients (43 males, 57 females, age 38 ±13yrs), had sufficient clinical data; 36 patients had a change in diagnosis; 21 patients had EEG support for non-epileptic attack disorder (NEAD), and 24 patients had no change in diagnosis. Video telemetry was less likely to lead to diagnosis change than ambulatory EEG but more likely to support a diagnosis of NEAD (p<0.0001). Of these patients, 72/81 were assessed for modification of treatment, with 37 patients changing after prolonged EEG.

Conclusion: The use of prolonged EEG recordings has a high impact on either change in diagnosis or the clinical management of epilepsy.


REM Behaviour Sleep Disorder (RBD) is a parasomnia that occurs mainly in older age groups and is characterised by the enactment of dreams and an absence of REM sleep atonia. The diagnosis of RBD is important as it may herald the onset of a neurodegenerative disease such as Parkinson’s or Alzheimer’s disease. Quantification of REM EMG hyperactivity in RBD has hitherto relied on visual inspection but a more objective measure of EMG power might be obtained using power spectral analysis (PSA).

We have therefore retrospectively analysed EMG activity in 10 patients with RBD...
Yawning as an Ictal Seizure Manifestation. A. Nicotra1,2, N. Khalil1, P. Owbridge2, M. Hakda3 and Y. Beitverda3.
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We observed an 87 year old man presenting with a history of decreased responsiveness and recurrent falls on a background of general physical decline over 9 months. On hospital admission, investigations included 24 hour ECG and routine blood pressure monitoring (no significant abnormalities) and brain CT (moderate degree of cortical atrophy, no space-occupying or infarct lesions).

A routine EEG was revealing and captured a clinical event. The EEG background showed a generalized excess of slow waves. With patient becoming unresponsive, a generalised decrement of ongoing EEG activities occurred and remained for the duration of the event. While unresponsive, clusters or single yawning was the most striking manifestation.

This is an exceptional case of prolonged seizure with impairment of consciousness and paroxysmal yawning. We hypothesize that this can be regarded as an autonomic seizure originating from diencephalic/brainstem structures, manifesting with yawning as an ictal phenomenon.

Further Support for Non-Genomic Hormonal Regulation of the Skeletal Muscle Chloride Conductance. J. Burge, S. Schorge and M.G. Hanna. (MRC Centre for Neuromuscular Disease, Queen Square, London, UK.)

The skeletal muscle chloride channel, CIC-1, regulates muscle fibre excitability by resisting deviations from the resting potential and by determining the cell’s input resistance at rest. Relatively little is known about the signalling pathways that control CIC-1 expression levels, nor about the pathomechanisms underlying phenotypic variability in low chloride conductance myotonia (Myotonia Congenita). Our group previously showed that progesterone, but not oestrogen shifts the voltage dependence of human CIC1 heterologously expressed in Xenopus oocytes, and that a hormone-induced voltage shift can exacerbate the effect of a Myotonia Congenita mutation. However, signalling pathways in amphibian oocytes are likely to be different from those in mammalian muscle. Here we present data from wildtype mouse flexor digitorum brevis showing that progesterone induces a right-shift of voltage dependence and reduction in amplitude of the endogenous skeletal muscle chloride current. Oestrogen exerts a qualitatively similar but less pronounced effect. This work provides further support for the idea that sex hormones contribute to the exacerbation of Myotonia Congenita that can occur during pregnancy.

Acute Oxaliplatin- Induced Neurotoxicity: Membrane Potential Changes with Natural Activity. S. Park1,², C. Lin1, A. Krishnan1, D. Goldstein3, M. Friedlander3 and M. Kiernan1. (1Neuroscience Research Australia & University of New South Wales; 2Institute of Neurology & Institute of Child Health, University College London, UK and 3Prince of Wales Hospital, Sydney Australia).

Oxaliplatin is utilized as first-line chemotherapy for colorectal cancer, but produces dose-limiting neurotoxicity, both acutely following infusion and chronically at higher cumulative doses. Axonal excitability studies were undertaken in 15 oxaliplatin-treated patients before and immediately after infusion to further determine mechanisms underlying acute neurotoxicity, which produces symptoms including cold-triggered fasciculations and cramps. Following oxaliplatin infusion, abnormalities developed in the recovery cycle with refractoriness markedly increased (P<.005). Excitability properties were assessed before and after maximal voluntary contraction of the abductor pollicis brevis. Following activity, axonal hyperpolarization developed, with proportional changes pre-and post-oxaliplatin in threshold (NS). However, recovery cycle parameters following activity were disproportionately enhanced post-oxaliplatin, with partial normalization of the recovery cycle (P<.05). Patients with the most abnormal recordings post-infusion demonstrated the greatest changes post-contraction. These findings suggest that oxaliplatin affects nerve excitability through voltage-dependent mechanisms, with
effects mediated through axonal Na\(^+\) channel inactivation, without significant alteration in membrane potential or Na\(^+/K\) pump function.


In stable incomplete male spinal cord injury (iSCI) subjects with severely compromised voluntary control of the sphincters the aberrant “guarding reflexes” can be monitored by recording the pudendo-anal reflex (PAR). This reflex is facilitated in control and iSCI subjects by single pulses of trans-cranial magnetic stimulation (TMS) (Craggs et al. 2007; *Neurourology & Urodynamics* 26: 614-615). In control subjects it can be further enhanced by a period of 5Hz repetitive TMS (rTMS).

In the present study we demonstrate that a short protocol reveals the degree to which single pulse TMS at an optimal preceding interval of about 30ms facilitates the PAR despite the progressive adaptation that occurs to repeated reflexes.

The protocol will now be used in a randomised cross-over trial of rTMS and sham stimulation to determine possible therapeutic benefit in iSCI by tapping into the neuro-plasticity of surviving corticospinal pathways to effect functional recovery in pelvic sphincters.