

1. **Implementation of Home Video-EEG-Telemetry at King's College Hospital (KCH) London. F. Brunnhuber, D. Amin, Y. Nguyen, S. Goyal and M. Richardson. (Kings College Hospital, London, UK).**

Aim: We present KCH's experience of the first 50 home video-telemetry (HVT) patients.

Methods: HVT met the MRC definition of a complex intervention, and we used its guidance to evaluate the process of piloting, evaluating, developing and implementing this new clinical service.

Results: Our feasibility study (n=5) with a test re-test design found no difference in the quality of recording or clinical yield between inpatient video-telemetry (IVT) and HVT. The pre-implementation study (n=8) showed an excellent patient satisfaction. We also discuss the findings of the main stakeholder survey (consultants and technicians). Our economic modelling demonstrates a clear financial superiority of HVT over IVT.

Discussion: Our findings show that diagnostic video-telemetry for seizure classification and poly-somnographies can be carried out safely at the patients' home and poses no security risks for staff and can be effectively integrated into the service offering of a tertiary care centre as a routine home or community-based procedure. Community or home-based MDTs (multidisciplinary team meetings) is an unexpected advantage of HVT and has the potential for future development for patients with complex conditions. HVT was preferred by our patients over IVT and provides a means of reaching out to some of the most vulnerable patient groups (children, patients with learning difficulties or other mental health problems), who would not otherwise benefit from this investigation or only under great difficulties. We hope to encourage other clinical neurophysiology departments and epilepsy centres to take advantage of our experience and consider adopting and implementing HVT, with the aim of a nationwide coverage.

2. **Electroclinical Features of Voltage-gated Potassium Channel (VGKC) Complex Antibody Associated Encephalitis in Children. S. Tang<sup>1</sup>, Z. Agirre-Arrizubieta<sup>2</sup>, N. Mullati<sup>3</sup>, J.P. Lin<sup>1</sup>, Y. Hacoheh<sup>4</sup>, A. Vincent<sup>4</sup>, M. Lim<sup>1</sup> and S. Goyal<sup>1</sup>. (<sup>1</sup>Evelina Children's Hospital, London, UK, <sup>2</sup>East Kent Hospitals University NHS Foundation Trust, UK, <sup>3</sup>King's College Hospital, London, UK and <sup>4</sup>University of Oxford, Oxford, UK).**

Objective: We report the electroclinical characteristics of four children with voltage-gated potassium channel (VGKC) complex antibody associated encephalitis.

Methods: Retrospective review of the electro clinical presentation of children positive for VGKC complex antibody (>100pM/L).

Results: The electro-clinical features of 4 previously healthy children (3 female, 1 male) aged 6 to 11 years presenting with seizures following a febrile prodromal illness are detailed in the table below. The seizures were resistant to first line medication in all cases and progressed to clusters of up to 15 seizures a day or complex partial status epilepticus. 2 patients demonstrated bilateral basal ganglia changes on MRI that have not been previously described. Follow up EEG was normal in 2 but showed residual focal slowing in two. None of the patients received immunotherapy. At follow up (> 6m) 3 were seizure free, 2 had behavioural problems and mild cognitive impairment.

Conclusions: VGKC Ab encephalitis in children can present without limbic involvement with an acute onset of complex partial seizures. Our cases demonstrate that spontaneous remission of seizures and reduction of antibody levels occur even without immunotherapy.

3. **Glissandi: Transient Fast Electrographic Oscillations of Steadily Increasing Frequency, Explained by Temporally Increasing Gap Junction Conductance. M. O. Cunningham<sup>1</sup>, A. Roopun<sup>1</sup>, I.S. Schofield<sup>2</sup>, R. Whittaker<sup>2</sup>, R. Duncan<sup>3</sup>, A. Russell<sup>3</sup>, A. Jenkins<sup>2</sup>, C. Nicholson<sup>2</sup>, M.A. Whittington<sup>1</sup>, R.D. Traub<sup>4</sup>. (<sup>1</sup>Institute of Neuroscience, Newcastle University, <sup>2</sup>Royal Victoria Infirmary, Newcastle upon Tyne, UK, <sup>3</sup>Institute of Neurological Sciences, Southern General Hospital, Glasgow, UK and <sup>4</sup>IBM T.J. Watson Research Center, Yorktown Heights, NY, U.S.A).**

We describe a form of very fast oscillation (VFO) in patient electrocorticographic (ECoG) recordings, prior to ictal events, in which the frequency increases steadily from ~30-40 Hz to >120 Hz, over a period of seconds. We dub these events "glissandi" and describe a possible model for them. 4 patients with epilepsy had presurgical evaluations (with ECoG obtained in two of them), and excised tissue was studied *in vitro*, from 3 of the patients. Glissandi were seen spontaneously *in vitro*, associated with ictal events; using acute slices of rat neocortex; and they were simulated using a network model of 15,000 detailed layer V pyramidal neurons, coupled by gap junctions. Glissandi were observed to arise from human temporal neocortex. *In vitro*, they lasted 0.2 to 4.1 seconds, prior to ictal onset. Similar events were observed in the rat *in vitro*, in layer V of frontal neocortex, when alkaline solution was pressure-ejected; glissandi persisted when GABA<sub>A</sub>, GABA<sub>B</sub>, and NMDA and AMPA receptors were

blocked. Non-alkaline conditions, prevented glissando generation. In network simulations, it was found that steadily increasing gap junction conductance would lead to the observed steady increase in VFO field frequency. This occurred because increasing gap junction conductance shortened the time required for an action potential to cross from cell to cell. The *in vitro* and modeling data are consistent with the hypothesis that glissandi arise when pyramidal cell gap junction conductances rise over time, possibly as a result of an alkaline fluctuation in brain pH.

4. **A National Census of Intensive Care Sedation Monitoring – Current Practice in England. S. Freilich and M.J. Shaw. (St Georges Hospital, London, UK).**

**Aims:** To determine improvements in sedation monitoring practices in England following the UK's Intensive Care Society<sup>1</sup> sedation guidance of 2007.

**Methods:** A telephone based questionnaire, developed and validated at St Georges Hospital, was applied to the 237 ITUs in England between July and October 2011.

**Results:** 185/237(78%) of ITUs completed the survey. 150/185(81%) have a written sedation policy, 184/185(99.5%) utilise a sedation scoring system, mainly the Richmond Scale (63%). 133/185(72%) assess sedation at least 2 Hourly, 52/185(28%) use Bispectral Index (BIS) monitors, but 14/52 did not use BIS on patients with pharmacologically induced paralysis. 135/185(73%) of the nurses interviewed felt that medical guidance provided for sedation depth was at least adequate.

**Conclusions:** Compared to reference data prior to the 2007 guidelines<sup>2</sup>, improvement was seen in the practice of sedation scoring. However, there remain a number of areas for future improvements. These include encouraging the development of local written sedation policies, setting sedation depth targets for patients, and increasing the frequency of sedation scoring in the minority of ITUs where sedation assessments were less than 6 hourly. At this time, BIS technology is not frequently used in England.

**Refs:**

<sup>1</sup>Intensive Care Society Sedation Guidance 2007  
[http://www.ics.ac.uk/intensive\\_care\\_professional/standards\\_and\\_guidelines/sedation\\_guidelines\\_2007](http://www.ics.ac.uk/intensive_care_professional/standards_and_guidelines/sedation_guidelines_2007) ;

<sup>2</sup>Rechscreiter et al. (2008) *Critical Care* 12:R152 (doi:10.1186/cc7141)

5. **The Effects of Gyral Geometry on the Size of Magnetic Motor Cortical Evoked Potentials. S.R. Jaiser, D. Soteropoulos, M.O. Cunningham, S.N. Baker and M.R. Baker. (Institute of Neuroscience, Newcastle University, Newcastle upon Tyne, UK).**

Computational models of transcranial magnetic stimulation (TMS)-induced fields have

traditionally employed half-planes or spherical shell approximations of cortical geometry. Recently, more complex models have applied finite element methods to a volume mesh based on high resolution human MRI data. These have shown that gyral geometry can focus electric fields induced by TMS; field strength increases by ~50% when the induced current is perpendicular to the gyral axis. These findings might explain the effects of TMS coil orientation on the size of motor cortical evoked potentials (MEPs) and predict that MEP size should not be affected by coil orientation in lissencephalic primates, such as marmosets.

We tested the effect of TMS coil (flat figure-of-eight) orientation in 6 marmosets during anaesthetic induction for unrelated terminal experiments. Anaesthetic regimens included alfaxone (n=2), alfaxalone+sevoflurane (n=2), alfentanil+sevoflurane (n=1) and propofol (n=1). MEPs were only recordable under alfaxalone anaesthesia (propofol and sevoflurane abolished MEPs), and in one of these animals, experiments were aborted for technical reasons. EMG was recorded via implanted stainless steel wire electrodes from the forearm flexor and extensor muscle compartments. In the single marmoset from which a complete dataset was obtained, MEP size was dependent on coil orientation (AP, PA, lateral) despite the absence of gyri. While gyral orientation may focus the induced electric field at the cortical level, we conclude that other major mechanisms contribute to coil-orientation specific effects on MEPs.

6. **Assessing Seizure Susceptibility Using Visual Psychophysical Tests. R.G. Whittaker<sup>1,2</sup>, J.C. Read<sup>2</sup> and A.J. Trevelyan<sup>2</sup>. (<sup>1</sup>Department of Clinical Neurophysiology, Royal Victoria Infirmary, Newcastle upon Tyne, UK and <sup>2</sup>Institute of Neuroscience, Newcastle University, Newcastle upon Tyne, UK).**

**Introduction:** Recent work suggests particular subclasses of interneurons play a critical role in restraining epileptiform activity<sup>1,2</sup>. Intriguingly, certain visual stimuli interact in a remarkably similar way to suppress neuronal firing. Interneuron pathology may therefore cause both increased seizure risk and measurable deficits in visual processing. We test this hypothesis by examining visual responses in subjects with forms of mitochondrial disease associated with an increased risk of epilepsy using visual stimuli in which inhibition plays a key role<sup>3</sup>.

**Methods:** We studied 9 patients with MERRF (Myoclonic Epilepsy, Ragged Red Fibres), 3 with MELAS (Mitochondrial Encephalomyopathy, Lactic Acidosis and Stroke-like episodes) and 25 controls. Subjects were asked to report the direction of movement of high

contrast gratings. The suppression index (ie ratio of log10 duration threshold for large vs small stimuli) was compared between groups using student's unpaired t-test.

Results: We found a mean suppression ratio of 0.39 for controls, 0.31 for MELAS, and 0.26 for MERRF. These differences were not significant.

Conclusion: These initial results suggest that patients with mitochondrial disease may have reduced cortical inhibition compared to controls. We are continuing this study on more patients with mitochondrial disease and patients with all forms of newly-diagnosed epilepsy.

Refs: <sup>1</sup>Brain 2011 Jul;134(Pt 7):180 ; <sup>2</sup>J Neurosci. 2006 Nov 29;26(48):12447-55 ; <sup>3</sup>Neurosci. 2006 Nov;26(44):11403-12.

7. **Operation of A Myoelectric-Controlled Interface as A Potential Rehabilitation Therapy For Facial Nerve Paralysis - A Pilot Study.** C. Brewster<sup>1</sup>, H. Magill<sup>1</sup>, K. Nazarpour<sup>2</sup>, M. Lai<sup>2,3</sup>, D. Sainsbury<sup>3</sup>, M. Ragbir<sup>3</sup>, O. Ahmed<sup>3</sup>, and A. Jackson<sup>2</sup>. (<sup>1</sup>Newcastle University Medical School, Newcastle upon Tyne, UK, <sup>2</sup>Institute of Neuroscience, Newcastle University, Newcastle upon Tyne, UK and <sup>3</sup>Royal Victoria Infirmary, Newcastle-upon-Tyne, UK).

Introduction & Aims: Facial nerve palsy is a potentially disfiguring condition associated with weakness and synkinesis in muscles of facial expression. Biofeedback with electromyogram (EMG) activity of facial muscles could be a potential treatment. We have developed a novel biofeedback training method to assess flexibility of facial muscles compared to distal hand muscles.

Materials & Methods: 12 subjects without facial nerve palsy made repeated movements of a myoelectric cursor, receiving scores that reflected the proportion of time they remained within elliptical or circular targets. Cursor position was determined by smoothed, rectified EMG from three muscle pairs. Facial muscle combinations included a natural synergist pair (orbicularis oculi-zygomaticus major) and an unnatural pair (orbicularis oculi-mentalis). In the hand, abductor pollicis brevis and abductor digiti minimi were paired. In some trials visual feedback was withheld.

Results: The study has demonstrated that control accuracy could be improved over 4 blocks of 100 tasks. Although lower in facial muscle pairs ( $p < 0.001$ ) compared to hand, performance of all muscle pairs improved at similar rates. Withholding visual feedback decreased performance of all muscle pairs comparably.

Conclusion: The findings in all muscle groups tested suggest that operation of the myoelectric-controlled interface may improve

facial muscle control and, consequently, function in facial nerve palsy.

8. **EMG Evidence for a Neurotoxin in Hump Nosed Viper Venom.** E. M. Sedgwick, V. Weerasinghe, N. Senanayake and I. Gawaramma. (Faculty of Medicine, University of Peradeniya, Sri Lanka).

Hump nosed viper (Hypnale) envenoming is common in Sri Lanka. The effects are local inflammation with necrosis, a coagulopathy and renal failure. Neurologic manifestations are rare but ophthalmoplegia, EEG changes and respiratory failure have been reported. Flaccid paralysis and muscarinic features are seen in envenomed mice. The venom has a neuromuscular blocking effect on chick biventer cervicis muscle which reduces the effect of ACh and carbachol and is reversible by washing. The venom is therefore thought to act on the postsynaptic membrane. The venom is known to contain phospholipase as well as procoagulant enzymes.

We tested the possibility of a sub-clinical effect on the venom on 4 envenomed patients (male 74 yrs, 144 hours since bite; male 44 yrs, 96 hours since bite; female 33 yrs, 26 hours since bite and male 34 yrs, 4 hours since bite) by stimulated single fibre EMG of orbicularis oculi muscle using the method and normal data from Kouyoumdjian & Stalberg (Ref). Mean jitter was raised in all patients (63.1, 36.5, 31.3 and 34.8  $\mu$ sec respectively ; Normal mean 33.4  $\mu$ sec, ULN 21.6  $\mu$ sec).

None of the patients showed neurological signs and all made a full recovery.

The 74 year old male showed the most abnormality at 6 days after the bite. Twelve of the 48 fibres studied in him showed some neuromuscular blocking. The others showed no blocking.

We conclude that Hypnale venom contains a neurotoxin but its effects are normally subclinical. Clinicians need to be alert to this particularly in those of small body mass.

Ref : *Clin. Neurophysiol.* (2011) 122(3):617-622.