

## The 57th American Epilepsy Society Annual Meeting

5-9 December, 2003; Boston, US

Most delegates arrived just 24 hours before the airport was closed by the heaviest December snowstorm for over 20 years. This forced them with various degrees of enthusiasm to attend the conference as it was the only event that could be reached without walking outside in blizzard conditions. We got away as the snow started thawing to watch the Ottawa Senators, a Canadian Ice Hockey Team play the Boston Bruins, in which the electronic advertising and blaring commentary were as entertaining as the match itself, not to mention skimpily clad ice maidens who cleared the ice of debris every few minutes – of more appeal to MM than EC!

There was a mixture of educational programmes and scientific presentations. Taxonomy continues to tax many clinicians. A new patient-orientated epilepsy classification has been developed at the Cleveland Clinic Foundation according to epileptogenic zone, seizure type(s), etiology, severity, and related medical conditions. Unlike the International League classification, all patients can be categorised in this 5-dimensional classification which they have found more useful in clinical practice. However, cases continue to be identified that defeat existing classifications. Twelve affected individuals from four families showed that clinical features of juvenile myoclonic epilepsy and idiopathic photosensitive epilepsy overlap; 50% of individuals with visual aura had myoclonic jerks, although visual aura is characteristic of IPOE and myoclonus of JME.

A retrospective study of 857 patients with status epilepticus from Richmond, Virginia found that 60% of cases were African Americans (over-represented). Their mortality was lower (22%) than in Caucasians (33%) and this may partly be due to different causes; more drug withdrawal and head injury. The authors speculated there may also be some biological factors that are worthy of exploration.

The Mayo clinic has been applying the ketogenic diet to a wide variety of adults with epilepsy and achieved a 50% seizure reduction in 73%. Metabolic changes did occur with a rise in cholesterol levels and reductions in

magnesium and selenium. Changes in phosphorus and potassium were also seen. Most patients lost weight. Given the similarities, it was only a matter of time before someone tried the Atkins diet and indeed ketosis was achieved in 3 of 5 patients from Baltimore who tried it. Two became seizure free for 2-4mths at the time of writing.

The difficulties of the diagnosis of epilepsy and complexities of multiple pathology in the elderly combine to delay diagnosis in this patient group. The Florida group found that for 159 people aged between 59-96, clinicians were able to identify GTCS reasonably quickly, but focal epilepsy took a long time to diagnose and associated cardiovascular disease delayed diagnosis, with a mean time to diagnosis for the whole group of 1.7yrs. The word needs to be spread: not all paroxysmal events in the elderly are TIAs, and an open mind and a good history are the best investigative tools.

Hyperventilation during the EEG is well established for childhood absence epilepsy. In a Brazilian study of 102

patients with intractable focal epilepsy 23.5% had their typical seizures during hyperventilation; 18 of 63 with TLE, 4 of 6 with multilobar epilepsy but only 1 of 20 with FLE. The induced seizures peaked at 4 minutes of hyperventilation and decreased thereafter – we need to push our patients

harder. This proved a cost effective and safe means of evaluating focal epilepsy as the time spent on video-EEG monitoring can be significantly reduced.

It is widely believed that prolonged GTCS are associated with foetal hypoxia and occasional fetal death but that partial seizures probably do not significantly affect the foetus. A 46 year old woman was described with a cavernous haemangioma manifesting maternal tachycardia and fetal bradycardia during a focal seizure. There were limited motor manifestations and no increase in uterine contractions to explain the foetal bradycardia, suggesting that the seizure may have triggered maternal dysautonomia and brought about fetal cardiac deceleration. This is worthy of further study.

The medical community is taking on Tony Blair's mantra of "education, education, education"! The National Sentinel Clinical Audit of epilepsy related death (UK) highlighted poor support and education for patients during complex treatment regimen and access to specialist advice for patients and general practitioners. A telephone advice service run by the Epilepsy Specialist Nurse (ESN) at Queen Square is aimed to address some of these longstanding inadequacies. It has improved continuity of care and reduced morbidity and has proved cost effective for both patients and the health service.

The American Epilepsy society sponsors a programme known as TELE Consults in epilepsy for Allied Health Care Professionals. The programme is free to those interested and further details can be obtained on line at [www.aesnet.org](http://www.aesnet.org).

Clinical trials can be a little bit like elections; everybody has a reason for saying that they won no matter how awful their result seems to be, but not in this veterans administration study. They compared carbamazepine 600mg with lamotrigine 150mg and gabapentin 1200mg in the

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Boston in the snow

treatment of 593 patients with a first seizure over the age of 60 (570 men). Clinicians were allowed to titrate up the dose if necessary. The average number of co-medications was 7! The primary outcome variable was retention which is generally considered the best composite measure of adverse effects and efficacy. Lamotrigine (about 50% retention) and gabapentin (about 40%) were both significantly better than carbamazepine (<30%). The study is sufficiently large and the results sufficiently convincing that I think this one paper is enough to change practice.

"Why are all my seizures at night?" is a common question. Diurnal variation of seizures in rats appears to be the same as in humans, even though they are nocturnal creatures, so the simple explanation of a relationship to brain activity and the sleep-wake cycle seems incorrect. My reply to patients will have to be changed to a more honest "I don't know"!

One snowed-in morning of the conference was devoted to temporal lobe epilepsy. The neuroimaging was beautiful, stunningly detailed anatomical pictures with 4.5 Tesla magnets. But two talks devoted to basic mechanisms probably carried the most important message of the conference. Interest has grown in recent years in fast rhythms: Gamma are 30-80Hz and are probably physiological or pathological depending on circumstances; ripples are 80-200Hz and fast ripples are >200Hz. Ripples and fast ripples seem to depend on axo-axonal interactions, gap junctions, ephaptic transmission and inhibito-

ry circuits. Fast ripples may be detected in the EEG hours before a seizure and are probably responsible for the electrodecremental response before some seizures. This in itself may prove clinically useful. March Dichter described how in models of TLE and in human slice preparations, these ripples appear in small hyperexcitable islands of cells in the entorhinal cortex and subiculum before any changes are seen in the hippocampus. Robert Sloviter discussed networks in TLE and presented data that hippocampal granule cell hyperexcitability correlates with hilar cell loss and restoration of inhibition correlates with mossy fibre sprouting. Seizures precede mossy fibre sprouting suggesting this is not an epileptogenic mechanism after all. Taking these lines of evidence, these researchers argued that the entorhinal cortex may be crucial in epileptogenicity and that changes in the hippocampus may be important in seizure expression but are secondary. They cite the frequency of dysplasia in the temporal lobes in patients with TLE as supportive evidence. If true, this will lead to a major re-think of mechanisms in TLE, which is likely to have far-reaching consequences.

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