Correspondence

A CROSS CANADA SURVEY OF NEUROLOGISTS ON CONTROVERSIAL ISSUES IN EPILEPSY MANAGEMENT

To the Editor:

We have read with interest the special report "Epilepsy and Driving" by Andermann et al[1] and have found it to be an excellent general review of a complicated issue. It reviews the literature for the world as well as laws for different countries and finally those for each of the Canadian provinces. In the broad context many different scenarios arise for which one gets vague guidance from individual provincial guidelines[2] on determining the fitness for driving. In an attempt to get a clear concensus on how neurologists approach certain potentially controversial driving issues Dr. Bruni and I[3] did a survey. It was a broad survey on many issues but within it were questions that dealt specifically with driving related issues. In 1984 all neurologists in Canada were surveyed. The response rate was only 32% and thus we felt we could only look upon the results as showing an interesting trend as opposed to bringing a clear concensus to some of the thorny issues. Some relevant responses were: Only 8% automatically report all their patients with epilepsy to the provincial Motor Vehicles Branch. Reasons for not doing so included "not a watchdog", I am not a policeman", "not required", "it is the patient's responsibility", "I only report the real dangers", "most can be trusted", "only those in *my* opinion", etc. As Andermann et al summarize, in the majority of provinces it is obligatory to report patients with epilepsy.

Fifty-three percent of neurologists felt it could be safe for patients with simple partial seizures with full motor control and 50% of the neurologists felt that those with "auras" could probably also be allowed to drive. Provincial guidelines are remarkably vague on the definition of seizures and don't give clear direction to physicians on how to handle this situation. The phrase "in your opinion" is there in most instances allowing the physician the latitude to exercise their clinical judgment. Qualifying phrases or prudent advice received from this survey included "These partial seizures would not affect vision, would not be followed by obtundation or cloudy states or loss of consciousness and one ideally would like a witnessed description of the simple partial seizure or aura".

Eighty-one percent of neurologists felt that patients with alcohol withdrawal seizures should not drive and 60% felt that these individuals should be reported to the Motor Vehicles Branch in contrast to only 8% of neurologists who would routinely report people with epilepsy. Currently in Saskatchewan and Quebec such individuals are restricted from driving and B.C. is about to enact similar guidelines.

What started out to be a rather simple project, intended to get a clear concensus on some of these more contentious issues turned out to be a far more complicated matter. It quickly became apparent that there is no such thing as "an average case". Clearly the neurology community wanted flexibility to make recommendations for each individual case rather than being "hand-tied" by arbitrary guidelines that may be punitive or needlessly restrictive.

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PERIODIC COMPLEXES IN CREUTZFELDT JAKOB DISEASE AND SLEEP

To the Editor:

Bilaterally synchronous periodic complexes (BPC) are seen in various disorders including Creutzfeldt Jakob disease (CJD), subacute sclerosing parencephalitis (SSPE), hypoxic encephalopathy, Binswanger disease and lipidoses.[1] Mechanisms involved in production of BPC remain unknown. We report 3 patients with CJD in whom BPC disappeared during sleep but reappeared on arousal.

Patient 1: This 69 year old female presented with memory loss, ataxia and incontinence of 4 months duration. Examination showed dementia, bilateral choreoathetosis, ataxia, spasticity and occasional myoclonus. She died 8 months later and diagnosis of CJD was verified at autopsy.

EEG showed background slowing and diffuse arrythmic delta. Di or triphasic BPC were seen every 600 milliseconds. During spontaneous drowsiness and sleep, BPC disappeared or diminished with suppression of amplitude. Following 2 mg. of intravenous diazepam, BPC disappeared for approximately 180 seconds and the patient appeared asleep during this period. On arousal, BPC reappeared.

Patient 2: This 62 year old patient presented with a three month history of progressive dementia, ataxia and incontinence. Examination also showed myoclonic jerks, spasticity and rigidity. She subsequently developed cortical blindness and dysphagia. Evoked potentials and CT scan of brain were normal. She died approximately a year after the onset of symptoms. Autopsy was refused.

EEG on admission showed diffuse arrythmic delta activity and diphasic BPC which occurred every 200 milliseconds with amplitude varying between 100 and 150 microvolts. BPC disappeared during spontaneous and chloral hydrate induced sleep but reappeared on arousal. Intravenous diazepam induced sleep and suppression of BPC for several minutes. Intravenous naloxone - which in opiate free individuals produces drowsiness[2] - produced similar suppression of BPC.

Patient 3: a 68 year old, female presented with dementia, gait apraxia and aphasia of two months duration. Examination also showed bilateral dystonic posturing and myoclonic jerks. Her neurological condition rapidly deteriorated and she died two months after initial admission.

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Autopsy confirmed evidence of CJD.

Initial EEG showed periodic diphasic complexes predominantly in the left hemisphere; they occurred every 1000 milliseconds and disappeared during sleep. Several weeks later, BPC became generalised but the relation to sleep - wake cycles could not be determined because of vegetative state.

The pathogenesis of BPC in CJD is unknown but several hypotheses have been proposed. Traub and Pedley[3] proposed that fusion of dendritic processes may lead to electrotonic coupling between neurons and this in turn might lead to large aggregates of cells, firing synchronously to produce BPC. They suggested that breaks in the neuronal and dendritic membrane, which have been shown to occur in CJD,[4] might lead to electrotonic coupling. They also argued that the cerebral cortical origin of BPC in CJD is supported by the lack of BPC in Kuru, a disorder in which spongiform changes occur mainly in the cerebellum and brain stem.[5] Szirmai et al[6] studied a single patient in whom diazepam produced suppression of BPC and proposed that the drug depressed the rhythmicity of a deep thalamic pacemaker responsible for BPC in CJD. Similar findings with diazepam and methylphenidate were subsequently reported in another patient by Rossini et al[7] and these workers hypothesized that lack of intracortical inhibition produced BPC. Our present findings in 3 additional cases suggest that mechanisms involved in the production of BPC in CJD are also linked to the reticular activating system and its diffuse cortical projection fibers.

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YOUNG AGE PARKINSONISM AND COMMON DRUGS

To the Editor:

Secondary parkinsonism can be a complication of a wide variety of chemical and metabolic insults.

Six young patients with parkinsonism, seen in the past five years, who had taken large amounts of common prescription or proprietary drugs are reported. In the same time only one female, age 42, with the disease and no drug history has been seen. If these observations are not unique there may be a causal relationship between these substances and the disease and the purpose of this letter is to raise this possibility.

Patient 1: A female bank teller, aged 25, began taking a dextroamphetamine anorexiant, tablet size unknown, three to four times a day, for the next six years and then replaced it with diethylpropion (Tenuate®) for the next three years with simultaneous daily diazepam, dose unknown. All drugs were stopped in 1975. In 1986 (age 43) she developed left hemiparkinsonism followed by some parkinsonian facial features and rigidity of both legs. She responded well to antiparkinsonian medication.

Patient 2: A female administrative assistant born in 1947 became sensitive to housedust and pollen in 1957. Each spring and summer for the next 10 years she used xylometazoline HCl nosedrops (Otrivin®) and spray for nasal congestion and hayfever. Chlorpheniramine (Chlor-Tripolon®) was also used. In 1963 a dextro-amphetamine anorexiant (dose unknown) was taken daily until 1973 when all medications were stopped. In 1983 a right-sided resting tremor of the hand and arm began and by 1986 the tremor involved the right leg and there was awkwardness and dragging of the leg. Physical examination revealed asymmetrical generalised parkinsonism.

Patient 3: A company manager, born in 1930, became an alcoholic in 1948 and continued drinking until 1972. Since then he has continually used xylometazoline HCl (Otrivin®) as a nasal spray several times a day. A 20 ml spray bottle lasts approximately one week. In 1983 the right hand became tremulous and in 1985 a diagnosis of right hemiparkinsonism was made. Appropriate investigations were normal and various combinations of antiparkinsonian medications were ineffective. He continues the daily use of the nasal spray.

Patient 4: A female clerk-typist, born in 1950, suffered from frequent, severe, migraine headaches. In 1979 she was started on a combination of acetylsalicylic acid, caffeine, butabarbital, plus 30 mgms of codeine (Fiornal®). She took four to 10 per day plus acetaminophen with 15 mgms of codeine, 40-60 tablets per day for the next four or five years. There were sporadic additions of diazepam 2-5 mgms, three or four per day, and regular daily dimenhydrinate (Gravol®) 150-180 mgms per day for nausea and large amounts of cascara for constipation. All medications were stopped in 1984. In 1986 she complained of a progressive limp due to weakness and stiffness of the left leg and six months later found her typing abnormal. Examination revealed predominantly left-sided parkinsonism that responded to levodopa-carbidopa medication and enabled her to return to work with good typing skills and marked general improvement. She has remained well with no change in symptoms or medication since then.

Patient 5: A male high school teacher, born in 1945, complained of weakness and dragging of the left leg in 1986. By the end of 1987 generalised signs of parkinsonism were evident. He had taken acetaminophen 300 to 600 mgms for insomnia for the past 20 years and had combined this with diphenhydramine (Sominex®) 20 mgms per day from 1976 to 1986. The response to antiparkinsonian medication was poor. He continues with the nightly medications.

Patient 6: A male machinist, born in 1934, developed a chronic E. Coli urinary tract infection in 1977. This was treated with trimethoprim-sulfamethoxazole (Septra®) half a tablet twice a day until 1984. He then became aware of a fine resting tremor in the left thumb and some left hand loss of dexterity and seborrheic scaling on the left side of the face. Idiopathic parkinsonism was assumed to be the diagnosis and not treated at his request. A year later the urinary tract symptoms recurred, the trimethoprim-sulphamethoxazole was replaced with norfloxacin which was effective. Within three months the tremor and left hand clumsiness had disappeared and the seborrheic crusting was no longer evident. Eighteen months later there is no evidence of urinary tract infection or parkinsonism.

There may be no relationship between the parkinsonism of these six patients and these common drugs. However, the