Bayer et al and in one reported by Lerner. In Bayer’s review of 27 documented cases of Group D enterococcal meningitis, all but two had CSF leukocyte counts of less than 200/cu mm.

As in endocarditis, mortality is greater in enterococcal disease. Bayer et al reported 33% mortality in their review of enterococcal meningitis. In our review of Sbovis meningitis, six of seven patients had an excellent clinical response to antibiotics and mortality was 14%. Most cases of Sbovis meningitis (like endocarditis) respond to penicillin alone, whereas enterococcal disease may require intravenous or even intra- thecal aminoglycoside therapy.

Sbovis meningitis may be misdiagnosed as enterococcus if appropriate laboratory algorithms are not followed. Specific bacterial diagnosis allows the clinician to choose appropriate antibiotic therapy and to search for underlying gastrointestinal disease.

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References

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Recurrent vertigo: cochlear-vestibular interaction

Sir: Recurrent vertigo is a commonly encountered clinical problem. Fortunately most of these patients can be satisfactorily classified even if no specific treatment is available. There are, however, patients who do not comfortably fit established diagnostic categories leading to suggestions for additional “disease entities” such as benign recurrent vertigo, recurrent peripheral vestibulopathy, and disabling positional vertigo. The last of these is believed to be due to a specific aetiology of microvascular compression causing “hyperactivity” of the vestibular nerve. Surgical therapy has been suggested for this latter condition.

A patient seen recently did not fit any of the above categories but did have a syndrome suggesting abnormal conduction between auditory and vestibular nerves. The possibility of microvascular compression had been considered but surgery was deferred. The patient was subsequently treated with phenytoin with excellent resolution of his symptoms. This case suggests that anticonvulsant therapy is indicated in some patients with recurrent vertigo.

A 72 year old man noted the onset of tinnitus in the left ear 6 years previously and in the right ear 3 years previously. An acute attack of imbalance lasting half an hour occurred in 1979, followed by recurrent symptoms of a sensation of imbalance daily since that time. The sensation of imbalance was not rotational nor was it a sensation of fainting. There was a definite sensation of movement that might produce a fall but without a consistent direction. Symptoms of imbalance were not related to movement or position but were predictably produced by specific sounds. The sound of chewing lettuce for example would produce symptoms chewing other food would not. Sounds of similar frequency composition would also produce symptoms; however, loud sounds of other frequencies would not. Coughing, sneezing and pressing the ear had no effect. A left ear plug provided partial relief from symptoms produced by external sounds. There was a history of chronic atrial fibrillation but not other heart disease, hypertension nor diabetes. The only medication being used was 10 grains (650 mg) of aspirin per day.

Neurological examination was within normal limits including Hallpike tests. Fissula testing with Frenzel’s lenses was negative. Studies obtained in 1981 showed bilateral mild high frequency hearing loss with good discrimination and caloric studies showed a 29% left unilateral canal paresis. BAER showed a prolonged latency of wave III with an increased I-III interval with right ear stimulated and no reproducible responses with left-sided stimuli. CT scanning with posterior fossa emphasis with and without contrast was normal as was a posterior fossa pantopaque study.

Therapy was begun with phenytoin 300 mg/day and within 2 weeks symptoms were markedly improved with decreased dizziness and tinnitus and ear plugs were no longer required. The aspirin was continued in light of the patient’s age and history of atrial fibrillation even though transient ischaemia was not considered a likely explanation for his symptoms. Repeated attempts to decrease phenytoin dosage resulted in an increased frequency of attacks.

This case appears to be unique. The history does not fit the common syndrome of benign positional vertigo nor are there sufficient features to suggest a Meniere’s syndrome. The absence of spontaneous vertigo since 1979 also makes diagnosis of benign recurrent vertigo or recurrent peripheral vestibulopathy unlikely. The major symptom of sound induced vertigo suggests an abnormal cochlear vestibular interaction. This effect is often labelled as “Tullio’s phenomenon”, although there is some inconsistency as to whether this term should include only a movement of the visual environment induced by sound or include any cochlear vestibular interaction. The usual implication, however, is that the effect is mechanical rather than electrical coupling occurring as a result of trauma or scarring in the middle ear. In this case the effect was clearly pitch or tone related and not related to volume of sound, making a mechanical explanation unlikely. It is more likely that the interaction was electrical at either the nerve or the brainstem level. Similar nerve to nerve ephaptic transmission has been demonstrated in “dystrophic” mice and postulated as a mechanism in the pain of causalgia.

It cannot be determined with certainty in this case whether the interaction was at the level of the 8th nerve or brainstem. The absence of any central signs or symptoms, the increased I-III interpeak BAER latency on the right as well as a unilaterally depressed caloric would however favour a peripheral (nerve) origin for this patient’s symptoms.

Recently microvascular compression of the 8th nerve has been postulated as a cause of both vertigo and tinnitus and indeed surgical therapy has been reported to improve symptoms in many of these patients. Unfortunately there is no mention of trials of anticonvulsant therapy for these patients.
Indeed anticonvulsants are often not mentioned as part of the medical armamentarium in treating vertigo. In contrast to the situation with 8th nerve dysfunction, patients with 5th nerve dysfunction (tic douxureux) are usually offered intensive therapy with anticonvulsive medication prior to surgical considerations.

The response to phenytoin in this patient, while clinically gratifying, does not provide insight as to the pathogenesis of his vertigo. The situation is akin to that in trigeminal neuralgia where both peripheral and central mechanisms have been proposed. Indeed this disorder shares with trigeminal neuralgia features of a trigger, paroxysmal response, cranial nerve involvement, and response to phenytoin. This case demonstrates that similar trials of anticonvulsant therapy are indicated in patients with other irritative 5th nerve dysfunctions.

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References

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Normal sural nerve morphometry in acute uraemia

Sir: We have reviewed the literature, and so far as we are aware, in only seven reported cases of acute uraemia were the sural nerves histologically studied. Of the five cases reported by Dinn and Crane, four had no clinical evidence of peripheral neuropathy, of whom two revealed segmental demyelination and remyelination in their sural nerves. The cases without histological evidence of neuropathy had uraemia of sudden onset and short duration. In a separate study published in the same year, myelin abnormalities were also observed in three cases, including one without clinical neuropathy. The latter case underwent biopsy 4 days after onset of uraemia and recovered completely in 2 weeks.

Recently, we had the opportunity to examine two acute uraemic cases, initially seen by nephrologists, as part of a longitudinal study for screening neuropathy in uraemia.

Fig. The clinical course of the two cases of acute uraemia.

Case 1 was a 20 year old male who developed acute uraemia due to volume depletion after 4 days of high fever and several bouts of vomiting. The patient recovered completely after 10 days, and did not require dialysis. Sural nerve biopsy, after obtaining informed consent, was done on the second day of the uraemia state (fig).

Case 2 was a 33 year old female who developed acute uraemia due to cardiogenic shock after suffering from acute myocardial infarction. She succumbed after 4 days from progressive increase in creatinine levels and oliguria. The sural nerve was obtained during necropsy, 3 hours after death (fig). Both cases did not have clinical neuropathy after systematic evaluation and grading. Furthermore, nerve conduction studies of at least four nerves in the extremities did not reveal peripheral neuropathy. The sural nerves were fixed in 3% glutaraldehyde and osmicated. Morphometric studies included teased fibre preparations of 100 myelinated fibre samples, internodal length and fibre diameter plotting, transverse epon embedded toluidine blue sections for light microscopy and myelinated fibre histograms employing a Zeiss TGA analyser followed by a programmed calculation. Likewise, electron microscopic studies were done, including histograms of the unmyelinated fibre populations. In all the methods employed no definite evidence of axonal degeneration or myelin abnormalities were obtained in either case.

It is of particular importance to make
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