liver function tests and arterial blood gases were normal. Radiotherapy was administered for presumed neoplasm. Mental functions again failed accompanied by right hemiparesis. No changes were discernable by CT scan and asterixis could not be elicited. The patient's course terminated in severe dementia.

Since the original delineation by Adams and Foley,\textsuperscript{10–12} asterixis has been an accepted sign of metabolic encephalopathy of exogenous or endogenous origin. Uraemia, hepatic failure, hypotoniaemia, hypercapnea and drugs have been implicated.\textsuperscript{2} The pathophysiology of the motor disturbance is, however, uncertain. Leavitt and Tyler have suggested that failure of integration of proprioceptive impulses promotes the motor disturbance.\textsuperscript{13} Shahani and Young suggest that dysfunction occurs in a central motor system for the maintenance of sustained muscle contraction.\textsuperscript{2} Unilateral asterixis has been described as a consequence of structural lesions in the brain. Vascular events in the mesencephalon,\textsuperscript{8} thalamus\textsuperscript{4} and internal capsule,\textsuperscript{9} stereotactic thalamotomy,\textsuperscript{9} and parietal lobe lesions\textsuperscript{8,11} have been associated with unilateral asterixis of the contralateral extremity. Discrete lesions in mesencephalic, thalamic and parietal lobe structures may disrupt somatosensory impulse integration, attention to stimuli and facilitation of motor activity, thus producing unilateral asterixis.\textsuperscript{8}

To the best of our knowledge, the unusual synchrony of loss of muscle tone observed in this case and the association of asterixis with obstructive hydrocephalus have not been previously described. The patient reported by Tarsey \textit{et al.} did not show asterixis in the period preceding ventricular peritoneal shunting for communicating hydrocephalus.\textsuperscript{2} Presumably, intraventricular pressure was normal in their case. Bilateral alternating asterixis in this patient was thought secondary to biventricular hydrocephalus caused by an anterior midline haemorrhage, possibly of neoplastic origin. Dexamethasone, cimetidine, or phenytoin, however, may have acted in concert with hydrocephalus to yield this sign. Patients recovering from unilateral stereotactic thalamotomy for Parkinson's disease may show asterixis contralateral to the lesion when challenged with phenytoin.\textsuperscript{9} Biventricular obstructive hydrocephalus may produce asterixis because intracranial hypertension affects brain functions globally as in metabolic encephalopathy. Alternatively, pressure on nearby brain structures, particularly the thalamus, may cause bilateral focal disturbances of the ascending reticular activating system.\textsuperscript{18} The contribution of plateau waves and pulsatile arterial pressure to the origin of this sign is unknown.

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\textbf{References}


\textbf{Letters}

\textbf{Matters arising}

\textbf{Subacute sensory neuropathy}

\textit{Sir}: The interesting case of neuropathy associated with Hodgkin's disease described in your journal by Sagar and Read\textsuperscript{1} is difficult to classify. They reported the case under the title "subacute sensory neuropathy", yet their patient's illness progressed to prevent walking within three weeks, remained unchanged for four weeks and then improved coincidentally with treatment of the Hodgkin's disease. The course of the illness and the slowed motor conduction (25 m/s in the legs) surely resemble the Guillain-Barré syndrome\textsuperscript{2} more closely than subacute sensory neuropathy\textsuperscript{3} despite the predominance of sensory involvement. The well recognised association between Guillain-Barré syndrome\textsuperscript{3} and Hodgkin's disease\textsuperscript{4} may be due to depression of cell-mediated immune responses which protect the body from herpes infections or autoimmune processes or both. So long as the nosological limits of Guillain-Barré syndrome\textsuperscript{2} and subacute sensory neuropathy\textsuperscript{3} remain poorly defined, disputes about diagnoses like this will continue. It would be preferable to discard the old terms and describe the disease in as much detail as investigations permit. Sagar and Read's case would become "acute demyelinating motor and sensory neuropathy". This description would at least have the merit of accuracy, and could be abbreviated to ADMSN.

\textbf{References}

Subacute sensory neuropathy.

R A Hughes

*J Neurol Neurosurg Psychiatry* 1982 45: 858
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