cleidomastoids may still continue intermittently in both forms if the patient is carefully observed.

Our patient had symptoms compatible with paralytic rabies. However, the initial presentation with seizures resulted in delay in diagnosis. Clinical seizures of cortical origin were extremely rare in our experience of more than 40 rabies patients (by Hemachudha and Manutsathit, unpublished data). Only one patient who received intensive respiratory support and had severe hyponatremia had a seizure during the preterminal phase. Opistothonos or convulsions during hydrophobic spasms have been described,[9] but not as an initial presentation. Attempts to make a diagnosis of human rabies in life by immunofluorescent testing for rabies virus antigen in corneal or salivary smears, or from nuchal skin or brain biopsy, and efforts to detect antibody to rabies virus in the serum or spinal fluid, were all disappointing during the early clinical stage of the disease.[6] Antibody to rabies virus by RFFIT was demonstrated in this case 9 days after the onset of disease. The presence of rabies antigen of comparable amount in both neurons and glial cells was surprising. Study of rabies virus distribution in six other patients (four encephalitic and two paralytic cases) showed neuron to be the almost exclusive target of infection (Manuscript submitted). Inclusion bodies in astrocytes have been regarded as an uncommon finding on light microscopy.[10 11] Only 17% of human rabies cases reported by Tangchai *et al*[10] were found to have inclusion body positive astrocytes. These were mostly in the floor of the third ventricle, paraventricular area and brainstem. It is not known whether seizure activity can be induced by the presence of virus in glial cells.

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References

1 Hemachudha T. Rabies. In: Vinken PJ, Bruyn GW, Klawans HL, eds: *Handbook of Clinical Neurology*. Amsterdam: Elsevier Science Publishers, 1989 (In Press).
2 Hsu SM, Raine L, Fanger H. The use of avidin-biotin peroxidase techniques: comparison between ABC and unlabelled antibody (PAP) procedures. *J Histochem Cytochem* 1981;**29**:577–80.
3 Houff SA, Burton RC, Wilson RW, *et al*. Human-to-human transmission of rabies virus by corneal transplant. *N Engl J Med* 1979;**300**:603–4.
4 *Morbidity and Mortality Weekly Report*. Human-to-human transmission of rabies via a corneal transplant-France. 1980;**29**:25–6.
5 *Morbidity and Mortality Weekly Report*. Human-to-human transmission of rabies via corneal transplant-Thailand. 1981;**30**:473–4.
6 Hemachudha T, Phanuphak P, Sriwanthana B, *et al*. Immunologic study of human encephalitic and paralytic rabies. Preliminary report of 16 patients. *Am J Med* 1988;**84**:673–7.
7 Phaupradit P, Manutsathit S, Warrell MJ, *et al*. Paralytic rabies: some unusual clinical presentations. *J Med Assoc Thailand* 1985;**68**:106–10.
8 Hemachudha T, Phanthumchinda K, Phanuphak P, Manutsathit S. Myoedema as a clinical sign in paralytic rabies (letter). *Lancet* 1987;**i**:1210.
9 Kaplan C, Turner GS, Warrell DA. *Rabies. The Facts*. Oxford University Press 1986.
10 Tangchai P, Yenbutr D, Vejjajiva A. Central nervous system lesions in human rabies—a study of 24 autopsy cases. *J Med Assoc Thailand* 1970;**53**:471–88.
11 Perl DP. The pathology of rabies in the central nervous system. In: Baer GM, ed. *The Natural History of Rabies*. New York: Academic Press, 1975:236–72.

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Matters arising

Peripheral neuropathy complicating pancreatitis

We were interested in the observations of Gross *et al*.[1] In 1970, we reported a case of a 30 year old sportsman who presented with acute pancreatitis, which was treated by surgery. Immediately after the operation, the patient developed encephalopathy characterused by a confusional state. Within a few days, a severe sensorimotor polyneuritis had led to quadriplegia. Nerve biopsy demonstrated a very severe axonopathy (reported at the VI Congrés International de Neuropathologie, 1970). The neuropathy disappeared completely over the next few months, followed by complete remission of the encephalopathy.

Although all reported cases have had acute pancreatitis, there are some notable differences between our case and that reported by Gross *et al*. The neuropathy in our patient appeared within a few days of the onset of the pancreatitis, although in other cases, the first signs of neuropathy were only observed some weeks after the pancreatitis. Our patient was not diabetic, and was not taking metronidazole or receiving parenteral nutrition. This would tend to rule out an aetiology involving significant vitamin deficiency. These conditions of onset also rule out the so-called "critically ill polyneuropathy".[2] In addition, it is noteworthy that the acute pancreatitis in our patient was accompanied by involvement of both central (transient encephalopathy) and peripheral nervous systems. We feel that a peripheral neuropathy may, in some circumstances, result from an acute pancreatic lesion. However, further cases will need to be identified before a causal link can be established.

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References

1 Gross MLP, Fowler CJ, Ho R, Russel RCG, Harrison MJG. Peripheral neuropathy complicating pancreatitis and major pancreatic surgery. *J Neurol Neurosurg Psychiatry* 1988;**51**:1341–4.
2 Bolton CF, Laverty DA, Brown JO, Witt NJ, Hahn AF, Sibbgald WJ. Critically ill polyneuropathy: electrophysiological studies and differentiation from Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 1986;**49**:563–73.

The conduction velocities of peripheral nerve fibres conveying sensations of warming and cooling

Sir: Evidence that in man the sensations of warmth and cooling are conveyed by different fibres is indirect.[1] Fowler *et al*[1] using reaction times to supramaximal heat and cold stimuli in normal controls recently demonstrated that the two modalities were conducted at different velocities suggesting that cooling was subserved by small myelinated and warming by unmyelinated fibres. We provide objective evidence that dissociated loss of warming and cooling sensation exists in diseases as seen in three patients with peripheral neuropathy associated with the acquired immunodeficiency syndrome. Thermal thresholds[2] were tested using