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cortical origin were extremely rare in our experience of more than 40 rabies patients (by Hemachudha and Manutsathit, unpublished data).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

Table *Thermal thresholds and nerve conduction studies in peripheral neuropathy associated with AIDS*

Patient	Thermal thresholds °C			Nerve conduction studies			
	Site	Hot	Cold	Ulnar SAP	Sural SAP	Med pl SAP	C per MCV
1	Wrist	0·1	0·1	4·0*	11·2	0*	45
	Ankle	0·8	3·8*				
2	Wrist	1·5*	0·3	3·2*	0*	0*	46
	Ankle	4·2*	0·3				
3	Wrist	0·1	0·1	18·4	0*	0*	37
	Ankle	4·2*	0·5*				
Normal values[2,3]	Wrist	<0·4	<0·3	>6·0	6–41	1–8	37–62
	Ankle	<2·8	<0·3				

The right side was tested in all 3 patients. Skin reference temperature 34°C. Abbreviations: * = abnormal; SAP, sensory nerve action potential (μV); MCV = maximal motor conduction velocity (ms ^{1}); Med pl = medial plantar nerve; C per = common peroneal nerve.

a commercially available device (Medelec: Triple T).

Case 1. A 34 year old homosexual man had noted numbness in his fingers and difficulty in doing up buttons for 6 weeks. He had noted an increased sensitivity to painful stimuli in his legs for 3 months. He had been HIV antibody positive for 3 years, had *Pneumocystis carinii* pneumonia 17 months before and had received bleomycin (270 mg total) and vincristine (3 mg total) for Kaposi's sarcoma during the last year. He was on zidovudine, ketoconozole and cotrimoxazole. Neurological examination revealed no motor deficit with intact deep tendon reflexes. Pinprick and light touch were impaired in his fingertips and toes and he had hyperpathia and hyperaesthesiae to mid thigh and mid forearm. Proprioception and vibration sense were intact. Thermal thresholds and nerve conduction studies are shown in the table.

Case 2. A 31 year old homosexual man had a 4 week history of numbness and tingling in all his toes and the little finger in his left hand. This had become painful after 2 weeks. He had been HIV antibody positive for 3 years, had *Pneumocystis carinii* pneumonia 27 months before and CMV pneumonitis diagnosed 2 weeks before. He was on zidovudine and foscarnet. Neurological examination revealed normal power and intact deep tendon reflexes with no deficit of light touch, pinprick, proprioception or vibration sense. The result of thermal thresholds and nerve conduction studies are in the table.

Case 3. A 33 year old homosexual man complained of pain and tingling for 3 months that had begun in his feet and gradually spread to his knees. He had been HIV antibody positive for 18 months, had CMV oesophagitis a year before and was taking acyclovir and ketoconazole. Neurological examination revealed no motor deficit and bilaterally absent ankle reflexes. Light touch and pinprick were absent to the ankle and impaired to the knees and vibration sense was absent to the knees bilaterally. Proprioception was normal. The results of thermal thresholds and nerve conduction studies are seen in the table.

All three patients had electrophysiological evidence of peripheral neuropathy. Cases 1 and 2 had exclusive impairment of either cooling or warming thresholds respectively. Case 3 had a disproportionate abnormality of warming threshold. There was no evidence of local cutaneous lesions, myelopathy or encephalopathy. The aetiology of the peripheral neuropathy in these cases is not clear.

It is of interest to note the association of spontaneous pain with the loss of warmth sensation and hyperpathia with the loss of the sensation of cooling. Although clinicians have long known that hot and cold sensations may be affected in different degrees[4] the differential involvement of cooling and heating seen in these patients provides objective evidence that these modalities are conveyed separately. Such a dissociation was mentioned by Jamal *et al*[5] in a variety of neuropathies but not discussed. Whether in our cases the pathology has affected differentially the receptors, the nerve fibres or both is not clear and further studies may provide more information.

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References

1 Fowler CL, Sitzoglow K, Ali Z, Malonen P. The conduction velocities of peripheral nerve fibres conveying sensations of warming and cooling. *J Neurol Neurosurg Psychiatry* 1988;**51**:1164–70.
2 Jamal GA, Hansen S, Weir AI, Ballantyne JP. An improved automated method for the measurement of thermal thresholds. 1. Normal subjects. *J Neurol Neurosurg Psychiatry* 1985;**48**:354–60.
3 Guiloff RJ, Sherratt RM. Sensory conduction in medial plantar nerve. *J Neurol Neurosurg Psychiatry* 1977;**40**:1168–81.
4 Gowers WR. *A Manual of Diseases of the Nervous System*. Philadelphia: P Blakiston, 1888:32.
5 Jamal GA, Weir AI, Hansen S, Ballantyne JP. An improved automated method for the measurement of thermal thresholds. 2. Patients with peripheral neuropathy. *J Neurol Neurosurg Psychiatry* 1985;**48**:361–6.

Fowler replies:

When a temperature testing device which can measure thresholds for warming and cooling separately is used in neuropathic patients, the finding of a differential loss affecting one thermal modality more severely than the other is common. In general we have found more marked impairment of perception of warming than cooling in diabetics. In some patients with mild diabetic neuropathy who have erectile dysfunction as the only manifestation of small fibre neuropathy, perception of warming on the sole of the feet may be absent while perception of cooling remains within normal limits.[1] Also in diabetics with plantar ulcers occurring in the context of severe generalised neuropathy, we have shown a more severe abnormality in warming perception than cooling on the feet, hands and face.[2] Indeed, using our particular testing system,[3] such a pattern is almost the rule in diabetics; only in patients with Fabry's disease have we observed the converse, a more severe abnormality for cooling than warming.

However, some caution should be exercised in the interpretation of such results. In our paper on the extent of small fibre neuropathy in diabetics with plantar ulcers[2] we point out that selective damage to unmyelinated fibres may not be the only explanation for relatively high thresholds of warming: that the perception of cooling is a