cleidomastoids may still continue intermit-
tently 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 Hema-
chudha and Manutsathit, unpublished data).
Only one patient who received intensive
respiratory support and had severe hypo-
atremia had a seizure during the preter-
minal phase. Opiotothinos or convulsions
during hydrophobic spasms have been de-
scribed, 6 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 disappoin-
ting 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. 6
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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Matters arising

Peripheral neuropathy complicating pancre-
atitis

We were interested in the observations of
Gross et al. 8 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 charac-
terized by a confusional state. Within a few
days, a severe sensorimotor polyneuropathy
had led to quadriplegia. Nerve biopsy demon-
strated a very severe axonopathy (reported at
the VI Congrès International de Neuropath-
ology, 1970). The neuropathy disappeared
completely over the next few months, fol-
lowed by complete remission of the ence-
phalopathy.

Although all reported cases have had acute
pancreatitis, there are some notable differ-
ences 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 poly-
neuropathy". 1 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 iden-
tified before a causal link can be established.

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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
conducting at different velocities suggesting
that cooling was observed by small myelin-
ated and warming by unmyelinated fibres.
We provide objective evidence that disso-
ciated loss of warming and cooling sensa-
tion exists in diseases as seen in three patients
with peripheral neuropathy associated with
the acquired immunodeficiency syndrome.
Thermal thresholds 2 were tested using

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Peripheral neuropathy complicating pancreatitis.

J M Vallat and C Vital

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