tral scotoma in both eyes. On the 6th day he was carried down to 4,700 m, but the follow-
ing day he could walk unaided. By the 18th
day, scotoma and impaired sensation disap-
ppeared, but his memory from the 3rd to the
5th day was lost. In February 1983 he was
examined at our university hospital, and no
abnormal findings were present in his optic
fundus.

The symptoms of high altitude sickness
have been well studied in the past.1-4 At
high altitude, mountaineers experience hypoxia, cold, hard exercise and, to a certain
extent, malnutrition. In the two afore-
mentioned cases, when they presented the
symptoms the altitude was around 5,900
meters (converted to standard height) and
the atmospheric pressure was about 50% of
that at sea level.5 The temperature several
days prior to their illness ranged from 0°C to
−10°C, but their clothes were sufficient to
protect them against the cold. They engaged
in activities under the same conditions as the
other climbing members. Until up to two
days before their symptoms developed, they
had been taking meals as usual and also
vitamin tablets every day. None of them had
any remarkable past history of diseases
involving peripheral nerves. In both cases,
they noticed their impaired sensation for the
first time only after they had regained con-
ciousness. This fact led us to suspect that
their symptoms appeared at the most crucial
stage of high altitude sickness. These series of observations suggested that the causes
of impaired sensory perception were related to
hypoxia and some adjunctive factors.

Locations and modalities of impaired sen-
sation were identical with the ischaemic neu-
ropathy caused by polycythæmia vera, namely, impaired perception of all sensory
modalities in the distal extremities with pre-
served power in all muscle groups.6

At high altitude, marked polycythæmia more than 7 × 10^9/mm³ could occur as the
result of acclimatisation.7 This polycythæmia can be exacerbated by dehydration
due to hyperventilation resulting in lowering
circulation in the capillary system.8 The
transient nature of these polyneuropathic
symptoms in our cases might be explained on
the basis of ischaemia in the peripheral
nerves.

KOUHEI ECHIZENYA
KUNIO TASHIRO
Division of Neurology,
Department of Neurosurgery,
University of Hokkaido,
School of Medicine,
North 15, West 7,
Kita-ku, Sapporo, 060,
Japan

References
1 Walton JN. Classification of the neuromuscular
280:175–84.
3 Wilson R. Acute High-Altitude illness in moun-
taineers and problems of rescue. Ann Int Med
4 Houston CS, Dickenson J. Cerebral form of high
5 Hackett PH, Rennie D, Levine HD. The inci-
dence, importance and prophylaxis of acute
6 Moore T. Unexpected effect of the peculiar shape
of the earth’s atmosphere. A physiological
study. Paper presented at the 6th Inter-
7 Winslow RM, Samaja M, West JB. Red cell func-
tion at extreme altitude on Mount Everest. J
8 Sili WE, Van Dyke DC, Winchell HS, et al. Early
erythropoietin, blood, and physiological
responses to serve hypoxia in man. J Appl

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Matters arising

Polyneuritis cranialis in Lyme disease
Sir: It was interesting to read the article
"Polyneuritis cranialis associated with Bor-
relia burgdorferi." We describe here a patient
with bilateral facial palsy due to Lyme
disease.

A 26-year-old man from the southern part of
New Jersey state was admitted to hospital
for left facial weakness of 2 days duration.
Three weeks prior to this admission the
patient had fever with chills for two days
which subsided without any specific therapy.
Subsequent to that he had bifrontal head-
aches and pain in the left mastoid region.
Two days prior to the admission he de-
veloped numbness of the left side of mouth,
tongue and the left eye. When he was seen in
the hospital he had slight neck stiffness,
dense lower motor neuron facial palsy and
diminished facial sensation on the left side.
A lumbar puncture was performed. The open-
ing pressure was 12 cm of CSF. The protein
was 41 mg/dl and sugar was 60 mg/dl. There
were 169 WBC/mm³; 95% were lymphocytes.
Routine bacterial and fungal cultures were
negative. A possibility of herpes encephalitis was considered. After 2 days he
developed right lower motor neuron facial pa-
lsy. A CT scan of the head with and without
contrast was normal. The antibodies for B. burgdorferi were negative.
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We increase in measurement subjects Parkinson of their frequency.2 The triad of neurologic manifestations of Lyme disease: meningitis, cranial neuritis and radiculoneuritis. Neurology 1985;35:47–53.

Respiration and sleep in Parkinson’s disease

Sir: We have read with great interest the recent paper by Apps et al.1 on respiration and sleep in Parkinson’s disease. We were particularly intrigued by the finding of an increase in respiratory rate among the Parkinson subjects compared with control subjects while awake and during REM sleep. It would be important to know if any direct measurement of minute ventilation or end tidal CO₂ was performed, that is, whether the tachypnoea was associated with true hyperventilation. Also, we wonder if their subjects had pulmonary function testing, as restrictive lung disease is often accompanied by increased respiratory frequency.

The authors appropriately point out that one possibility for this finding is an alteration of ventilatory control in Parkinson’s disease. As there is a great deal of evidence that central catecholamines play a role in ventilatory drive,2 this is a very reasonable speculation.

We have recently studied ventilatory drive in a group of 14 patients with Parkinson’s disease (Hoehn and Yahr stages III–IV) and 11 age matched controls. All subjects had spirometry to rule out significant obstructive or restrictive lung disease. We did not observe changes in resting end tidal CO₂ or respiratory rate at rest in our group of Parkinson’s patients. However, using rebreathing methods for hyperoxic hypocapnia and isocapnic hypoxia3,4 we found an increased response in our Parkinson’s disease subjects to both hypocapnia and hypoxia.5 It is not yet clear whether this might be a central effect or whether dopamine metabolism in the carotid bodies of these patients is abnormal as well.

Further studies of ventilatory drive in Parkinson’s disease should be of considerable interest both to expand our knowledge of this disease and to elucidate further the role of catecholamines in respiratory drive in man.

References


Apps replies:

I myself have carried out studies of hyperoxic hypercapnic rebreathing in Parkinsonian patients and found a normal response to this diminished breathing though some of the patients had an end tidal CO₂ at the lower end of the normal range.

Sensorimotor neuropathy and cisplatin and adriamycin toxicity

Sir: The brief report by Pages et al.1 of a severe sensorimotor neuropathy developing in a subject treated with adriamycin and cisplatin raises a number of interesting mechanistic questions. As they note, the occurrence though transient of a motor component to the neuropathy was unexpected and is as yet inexplicable. Neither of these drugs penetrates the normal blood–brain barrier2 and this is shown by the low concentrations of cisplatin found in the CNS, although cisplatin may have access to peripheral nerve.3 Both drugs must readily enter the spinal ganglia, presumably through fenestrations in the vascular bed noted some years ago.4 Another possible route to motor nerves through their terminals is available,5 but this is likely to be very minor by comparison, although with the very high dose of cisplatin given (about twice the amount usually considered to be neurotoxic) this route could conceivably have become more important.

The well known damage to sensory nerve fibres encountered in cisplatin intoxication is a different, and perhaps more straightforward, matter. The reduction in numbers of myelinated and unmyelinated axons in the sural nerve biopsy of this reported case confirms this and would be anticipated to be due to severe damage to their cell bodies within sensory ganglia, if our recent experimental studies in rats have any relevance to the matter.6 While in this species it in fact has not been possible to reproduce the neuropathy, (for the animals die of non-neurological causes when the cumulative dose reaches only about 150 mg/m², which is about half the dose required to cause neuropathy in man), there is nevertheless unequivocal damage to nucleoli in a high proportion of sensory ganglion cells. This becomes visible within the first 24 hours of treatment with cisplatin and proceeds to segregation of the nucleolar constituents and later nucleolar fragmentation. Since nucleoli are the seat of ribosomal synthesis and nucleolar segregation is a sign of reduction or cessation of synthetic activity, it was not perhaps surprising to find that by the end of a week of treatment many ganglion cells showed severe reduction in Nissl material and conspicuous shrinkage of the whole cell. If the animals had not died from other causes, it is highly likely that cell death and/or axonal degeneration would have occurred, as these cellular events, while not precisely the same as those found with adriamycin in rat spinal ganglia, followed the same general sequence. Indeed, both drugs have somewhat analogous effects upon DNA and lead particularly to inhibition of RNA polymerase I activity,7,8 the polymerase concerned with ribosomal transcription. In cisplatin toxicity ganglion cells are randomly affected regardless of size, and since small neurons responsible for unmyelinated and thinly myelinated fibres are substantially more numerous than large neurons concerned with the more discriminatory aspects of sensation, it is not wholly surprising that cases of neuropathy should occasionally show very little in the way of sensory loss of the latter type. The sural nerve biopsy showed in this case a substan-