more than 3 SD below the expected level. Comparing his tests results with age and education corrected norms, he showed specific dysfunctions regarding speed of information processing, motor speed, visuomotor coordination, and tacto-perceptual functions. On these tests he performed between 2 SD and 4 SD below the expected level. There was also a discrepancy between verbal and non-verbal intellectual capacity with a pronounced decrease on the performance IQ score of 64 compared with his verbal IQ of 89. The motor tests and some of the sensory-perceptual tests showed a left-right discrepancy of more than 1 SD with right hand performance being better than the left. On tests for verbal intellectual functions, memory, and other higher cortical functions, he performed within the normal range.

The rehabilitation therapy was terminated after four months. At this time his general functioning was moderately improved, but objective signs on neurological examination were essentially unchanged. Re-examination 24 months after the onset of neurological symptoms showed unchanged clinical findings on neurological examination, whereas cerebral MRI showed regression of the cerebellar changes. Neuropsychological re-examination showed some improvement, with an Halstead impairment index of 0-6, an improvement of 1-5 SD from the initial examination. The improvement was most obvious in time and motor dependent tests, but was also evident in tests for non-verbal cognition and sensory perceptual functions. The left-right discrepancy seen initially on some tests was now less obvious.

The clinical and radiological findings in our patient are consistent with the previously reported cases of leucoencephalopathy after inhalation of heroin.2,4 Unlike most of the reported patients however, our patient managed to terminate his drug misuse and was accessible for follow up examinations. In the previously reported cases there has been a symmetric hypodensity of both cerebellar hemispheres on cerebral CT; in some cases CT has also shown hypodensity of cerebral white matter. Neuropathological studies have shown oedema of the cerebral and cerebellar white matter, and to a lesser degree also of the grey matter. This condition has been termed spongiform leucoencephalopathy because microscopic investigations have shown that the myelin sheaths were swollen and vacuolated, and some were totally destroyed. The axons were spared.2,4

Spongiform leucoencephalopathy after inhalation of heroin pyrolysat is a rare complication, and so far only 56 cases have been reported in English. For general epidemiological data on how and where of heroine intake is common, the condition has never been reported. In Europe it seems that when it occurs several cases are usually reported. This suggests that the etiology, which is still unknown, is related to the heroin batch and could perhaps be a toxic effect of one or more of the heroin additives. In our case we managed to obtain a sample of the heroin batch used by the patient for analysis. This has, however, been done in other reports, and none of the common heroin additives detected, such as caffeine, phenobarbitone, methaqualone, procaine, piracetam, and lignocaine, is known to cause this kind of encephalopathy.6

Spongiform leucoencephalopathy has so far been almost entirely related to inhalation of heroin. One reason may be that the dose inhaled by direct inhalation is much greater than the doses obtained by smoking or sniffing. Another explanation is that the process of heating creates a new compound from either the heroin or one of the additives, and which in turn causes the leucoencephalopathy. Because this leucoencephalopathy tends to occur as small epidemics it seems more likely that it is related to one of the irregularly occurring additives. Other modes of heroin intake do not seem to be associated with this condition, with the possible exception of a two and a half year old boy recently reported.7 In this case the mode of intake was unknown, but inhalation was considered improbable. With this exception it therefore seems that inhalation is essential for the development of this state.

The heroin inhaled by our patient originated, according to his own statement, mainly from the same batch. He shared the heroin with a friend, and he usually received greater doses than our patient to reach the same effect. The girl never developed any symptoms and showed normal progress on neuropsychological examination. This suggests that the etiology is not only related to toxicity of the heroin or its additives, but also to an individual disposition.

Severe neuropsychological deficits are seldom seen in ordinary opiate abuse. A previous study of seven patients receiving injections of high doses of pharmaceutical heroin for an average of 32 years, showed minor or no cerebral CT abnormalities, and only slight cognitive impairment in terms of reduced verbal memory function and speed of information processing.8 To our knowledge none of the previously reported cases of spongiform encephalopathy after inhalation of heroin have been neuropsychologically examined. Our patient had pronounced neuropsychological impairment not typically seen in heroin addicts. In south east Asia, in south east Asia, and coordination problems are fully consistent with the cerebellar abnormalities seen on MRI, the cognitive and sensory-perceptual deficits mimicking the original, and the verbal performance discrepancy and the neuropsychological test profile might indicate a relatively more severe affection of the right hemisphere. The sensory perceptual and cognitive deficits could be explained by affection of cerebral white matter.

Sixth nerve palsy from a CNS lesion in chronic inflammatory demyelinating polyneuropathy

Evidence of demyelination of the CNS has been found in half of the patients with chronic inflammatory demyelinating polyneuropathy (CIDP) as shown by MRI, but only a few patients showed evidence of CNS involvement.1 Sometimes a relapsing multifocal course resembling multiple sclerosis precedes CIDP.2 Involvement of cranial nerves is uncommon in CIDP, but cranial nerve impairment may be the first manifestation.3 Although an immune attack on nerve myelin sheaths is a likely explanation, a central cause should also be considered. We report a patient with CIDP who developed unilateral right sixth nerve palsy associated with a pontine white matter lesion.

In March 1993 a 48 year old man developed tingling in his hands and feet followed by progressive weakness of the legs leading to walking impairment after three weeks. In June severe weakness of all limbs was present; tactile, vibratory, and pain sensations were decreased in the hands and feet, and there was areflexia. Analysis of CSF showed a raised protein content of 1·0 g/l (normal <0·4). Nerve conduction studies fulfilled the criteria for CIDP. In the sural nerve biopsy some axonal degeneration was found with a normal density of myelinated fibres (6048/mm²); CD3 positive T lymphocytes were scattered throughout the biopsy. The patient recovered gradually after a five day course of intravenous immunoglobulin treatment (IVlg 0·4 g/kg/day). In November 1993 his condition seriously deteriorated, to improve again after treatment with IVlg. In both relapses there were increased concentrations of mutant T lymphocytes in peripheral blood; values returned to normal during remission.4

In January 1995 he suddenly experienced diplopia while driving his car. In that position there was a complete paralysis of the left abducens nerve but no signs of a relapse...
of the neuropathy. After three months the diplopia had disappeared. The CSF showed 6 x 10^9 mononuclear cells and a protein content of 0.88 g/l; some oligoclonal bands were present, but the IgG index was normal. Brain MRI showed multiple abnormalities of perivenricular white matter. So, it is likely that enhanced intravenous administration of gadolinium, suggesting active CNS inflammation. In the pons an area of high T2 signal intensity was seen in the proximal part of the left abducens nerve, providing substantial evidence for a sixth nerve palsy of CNS origin (figure).

Although a vascular origin cannot be fully excluded, the distribution of the lesions was typical for plaques seen in multiple sclerosis. Moreover, the enhancing white matter lesions were present several weeks after onset of the sixth nerve palsy, making a coincidental vascular lesion unlikely. Lesions without enhancement may reflect older lesions. Nevertheless, multiple sclerosis could not be diagnosed, as other white matter abnormalities in the CNS were asymptomatic.

Previous studies have suggested that cranial nerve lesions in CIDP may be related to CNS lesions. However, MRI abnormalities were not more common in patients with cranial nerve palsies than in those without. The enhancing pontine lesion in our patient is suggestive of active CNS demyelination and therefore probably responsible for a central origin of the ocular palsy, which interestingly occurred independent of peripheral nerve involvement at that time. Previous information on white matter abnormalities in the CNS in this patient was not obtained as MRI is not a routine diagnostic procedure in CIDP. Yet these abnormalities may occur in a disproportionate number of patients.

There may be several explanations for the combined occurrence of central and peripheral myelin dysfunction. Firstly, coincident lesions seem unlikely, as so many patients with CIDP show evidence of involvement of central and peripheral myelin. Secondly, the process of demyelination in itself may lead to ongoing generalised demyelination through activation of cytokines. Finally, central and peripheral myelin may share common antigens which trigger an immune response. The occurrence of cranial nerve palsies in our patient stresses the relevance of MRI in patients with CIDP and cranial nerve dysfunction.

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Capture-recapture methods for precise measurement of the incidence and prevalence of stroke

Capture-recapture methods, originally developed for wildlife censuses, are increasingly being used in epidemiology to estimate the incidence and prevalence of disease in the population. The principle is that two or more censuses are taken, with the people or animals being identified and marked on each occasion, and knowledge of the degree of independence of these censuses is used to estimate the proportion of the total population that has been missed. This allows an estimate of the size of the total population under study. Capture-recapture results have been published for a wide range of medical conditions including myocardial infarction, but not yet for stroke. The method is attractive because maintaining community-based registers that are “as complete as possible” is an expensive exercise.

Using the capture-recapture method, at least two independent sources need to be identified. The number of cases that are common to both sources is calculated (m), as are the total numbers of cases from the first and second samples (M and n respectively). The equation for the capture-recapture estimate of the total number of cases, N, is given by the equation:

\[ N = \frac{(M + 1) (n + 1)}{(m + 1)} - 1 \]

LaPorte has used the capture-recapture method to estimate the number of cases of myocardial infarction among a defined population in Sweden, with the myocardial infarction registry as the first source (M = 5832) and the hospital discharge index as the second source (n = 6582), and the estimate is m = 4746. The capture-recapture estimate of 8088 cases implies that 5% of the cases of myocardial infarction had been missed by both sources of notification. The fact that cases may be missed by different sources does not imply that our estimates are likely to be lower for the older age groups. Our estimates for myocardial infarction would be higher in the under 75 years age group.

The number of deaths per year is calculated as the number of cases multiplied by the death rate. In our study and, as previously mentioned, the prevalence of new cases was calculated as the number of cases divided by the population at risk. This allows an estimate of the number of strokes per 1000 person-years. In the Oxfordshire Community Stroke Project, some 10 years earlier, we calculated a crude annual rate of 0.688 cases per 1000 person-years (aged under 75 years). The corresponding crude rate from the Oxfordshire Community Stroke Project, some 10 years earlier, was 0.93 with 95% confidence interval 0.82-1.04. The initial capture-recapture estimate of the total number of strokes, obtained using log linear modelling, was 1999, corresponding to 2.13 cases per 1000 person-years. This rate is more than twice that of the Oxfordshire study and, considering the thor-