Matters arising

Sir: We read Henderson *et al*'s, recent article (*J Neurol Neurosurg Psychiatry* 1983;46:437-9) with interest. Indeed Raspe is buried at Muckross. However, this is in County Kerry: Killeggy Churchyard (unmarked grave) in the parish of Killarney. The parish register has his date of demise as 19 November, 1794; his occupation as "miner"; and his cause of death as "fever". Perhaps the authors were thinking of McIlroy's (one 'l') supposed place of birth¹ when they placed Raspe's remains in County Donegal. In fact McIlroy was born in Belfast.²

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References

- ¹ Pallis CA, Bamji AN. McIlroy was here, or was he? *Br Med J* 1979;2:973-5.
- ² Copy of Birth Certificate lodged with Editor.

Progressive opticatropy and sensorineural hearing loss due to chronic glue sniffing

(*J Neurol Neurosurg Psychiatry* 1983; 46:349-51)

Sir: The case report by Ehyai & Freeman was suitably brief and of some clinical interest, but the absence of any information about the solvents used by the patient and any critical evaluation of the quoted reports prompts me to comment here.

The individual was "sniffing glue" and "had a strong smell of glue on his breath". He was also said to have shown gradual improvement with "no toluene exposure". No analyses of the materials used by this individual were offered and there is no experimental evidence available that toluene is responsible for the syndrome associated with this practice. It is imprecise and unhelpful to say that "he had a smell of glue, etc." for the glue itself is not sniffed; it is the solvent that is inhaled for its effects. The critical question is, what is the solvent? We are now confident that n-hexane and certain of its analogues will induce filament accumulations within axons that impair the flow of materials along the axons to the periphery and thus induce Wallerian degeneration. This occurs principally in large diameter and longer fibres. In shorter, smaller axons no degeneration is seen though functional

changes, probably secondary to transport problems, may be encountered. In true hexacarbon intoxication it is remarkable how closely the symptomatology is linked to long, large fibres, even though all parts of the nervous system show filamentous accumulations within axons.¹

To argue by analogy from this clearly very specific (or almost specific, since carbon disulphide does exactly the same thing) intracellular mechanism to another chemical of entirely different structure should not be done without some supporting evidence. Unfortunately the clinical cases quoted do not help. Thus, while Grabski's² case certainly sniffed toluene and was very fastidious about its quality, we have no objective information about the nature of the cerebellar damage if any. Again, in the case of Escobar and Aruffo³ Escobar admits in a personal letter that this case may have inhaled any number of solvents and his nutritional state was not optimal.

Indeed, it is rare, I understand, for pure solvents to be used in these preparations. Similar criticisms can be raised against the other reports quoted.

Lest the impression becomes widespread that we know the toxicological basis of cerebral and/or cerebellar damage that follows repeated solvent inhalation, with all that this implies, it should be emphasised that without any analysis of the materials used or other indications of its nature the changes observed cannot yet be ascribed specifically to one substance or another.

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References

- ¹ Cavanagh JB. The pattern of recovery of axons in the nervous system of rats following 2, 5 hexanediol intoxication: a question of rheology? *Neuropath Appl Neurobiol* 1982;8:19.
- ² Grabski DA. Toluene sniffing producing cerebellar degeneration. *Am J Psychiatry* 1961;118:461.
- ³ Escobar AC, Aruffo C. Chronic thinner intoxication: clinicopathological report of a human case. *J Neurol Neurosurg Psychiatry* 1980;43:986.

Freeman and Ehyai reply:

Our patient described his toxic exposure to us in great detail on several occasions, years apart, without significant variation. He purchased glue of various brands at hobby shops. Several times each day, he

squeezed some into a bag, held the bag to his nose and mouth, and breathed the material until he obtained the desired mental changes. On the first day of each of his hospitalisations, an obvious odour was present on his breath. This smelled to us like the odour of glue, as when one uses glue to make model airplanes. When we examined the patient after two or three days in the hospital, this odour was no longer present.

For the reasons detailed in the paper, we conclude that the patient's neurological deterioration resulted from this repetitive behaviour, which is probably most simply described as "sniffing glue". The patient denied exposure to other solvents and his forthright description of glue use makes us believe him.

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