A decompressive thoraco-lumbar laminectomy was performed, and the radiological appearances were confirmed; the conus and cauda equina was swollen and diffusely infiltrated by pale, amorphous tissue. Biopsy specimens were taken from the conus and a nerve root. Histological examination of these revealed the presence of granulomata containing multinucleate giant cells and *Schistosoma mansoni* ova. A diagnosis of schistosoma myeloradiculopathy was made.

The patient's neurological state was unchanged following the operation, and therapy with schistosomicidal drugs was started. This comprised a combination of praziquantel (40 mg per kg as a single dose), oxamniquin (15 mg per kg for two days) and niridazole (15 mg per kg for seven days). Within two days of starting therapy, the power of his legs improved as did his urinary function. At that stage, he returned to Nigeria.

Myeloradiculopathy due to *Schistosoma mansoni* infection is uncommon, but well documented. The clinical features were first described in 1917⁴ and have been recently reviewed by Scrimgeour and Gajdusek.² They typically comprise a flaccid paraplegia with some disturbance of sensory function occurring predominantly in males up to six years after exposure to the schistosomes. The myeloradiculopathy is usually caused by a granulomatous intramedullary mass extending to involve the cauda equina. The myelographic² and CT myelographic appearances of schistosomal involvement of the conus have been described elsewhere.^{5,6} However, there have been no detailed reports of the CT myelographic appearances of the nerve roots, these appearances being verified at operation. A pre-operative diagnosis of schistosomal myeloradiculopathy is often difficult to establish. Many of the investigations are non-specific, and even the specific serological tests may be misleading. The radiological findings described here, particularly following CT myelography may lend considerable support to a diagnosis of schistosomal myeloradiculopathy, and it may be that the need to carry out a decompressive laminectomy and biopsy will be obviated, except in those cases who either have a complete block shown by myelography, or continue to deteriorate despite adequate therapy. Treatment is usually by a combination of decompressive surgery and the administration of schistosomicidal drugs, which act by killing the adult female worms, so leading to a cessation of ova production and consequently a subsidence in the inflammatory response. Initially, antimony compounds were used,

but these proved to be disadvantageous because of their high toxicity and their need to be administered via intra-venous or intramuscular routes. Currently, praziquantel and oxamniquin are the drugs of choice for *Schistosoma mansoni* infection, both of which are relatively safe and can be administered orally.⁷

We thank Dr JP Hall and Dr W Squier for their assistance regarding the management of this case, and Dr JP Silver (Stoke Mandeville hospital) for kindly referring the patient.

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References

- Wakefield GS, Carroll JD, Speed DE. Schistosomiasis of the spinal cord. *Brain* 1962;**85**:535-52.
- Scrimgeour EM, Gajdusek DC. Involvement of the central nervous system in *Schistosoma mansoni* and *S. haematobium* infection. A review. *Brain* 1985;**108**:1023-38.
- Herskowitz A. Spinal cord involvement with *Schistosoma mansoni*. *J Neurosurg* 1972;**36**:494-8.
- Ferguson AR. Some notes on bilharziasis. *J R Army Med Corps* 1917;**29**:57-65.
- Acute Schistosomiasis with transverse myelitis in American students returning from Kenya. *JAMA* 1984;**252**:1116, 1119, 1123.
- Case records of the Massachusetts general hospital. Case 21. *N Engl J Med* 1985;**312**:1376-85.
- Efthimiou J, Denning D. Spinal cord disease due to *Schistosoma mansoni* successfully treated with Oxamniquine. *Br Med J* 1984;**288**:1343-4.

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Alzheimer-type dementia and Down's syndrome: solubility of neurofibrillary tangles is related to duration of dementia

Sir: Neurofibrillary tangles and senile plaques are characteristic cerebral lesions of Alzheimer-type dementia and elderly Down's syndrome.¹ The solubility of tangles in protein solubilising agents such as sodium dodecylsulphate (SDS) has been reported to vary considerably between brains.² We have

examined the possibility that this variation in solubility reflects ageing of tangles by studying the relationship between tangle solubility and patient age and between tangle solubility and duration of dementia.

Eighteen brains with severe Alzheimer-type dementia pathology from twelve cases of Alzheimer-type dementia (age range 56-82 years, median 70 years) who had clinical evidence of dementia and six cases of Down's syndrome (age range 56-67 years, median 60 years) who had shown behavioural changes indicative of dementia, were studied. One gram of temporal cortex was homogenised in 2.0 ml distilled water and aliquots further homogenised with distilled water (control), 0.2 M NaOH, 1% SDS or 5% SDS, with β -mercaptoethanol (0.1%) included in the SDS solutions. The samples were diluted to 6.0 ml with water and tangles and plaque cores separated by centrifugation at 8000 g for 5 minutes. The pellets were stained with congo red, dispersed in 1.0 ml water and applied to a 1.0 ml glass after counter. Tangles and plaque cores recognised by their green/red birefringence and distinguishable by their morphology were counted in fifty 1 μ l divisions of the counter. Neuritic plaques broke up during homogenisation and were unrecognisable in the counter. Differences between Alzheimer-type dementia and Down's syndrome cases and between sexes were tested using the Wilcoxon Rank Sum test and associations between counts and other variables tested using Spearman rank correlation.

As there were no differences between results from the Alzheimer-type dementia

Fig Number of tangles insoluble in 1% SDS expressed as a percentage of total number of tangles vs duration of dementia.

and Down's syndrome cases, data from all cases were pooled for analysis. The number of tangles in the aqueous homogenates varied enormously from brain to brain (range 14–325, median 102/mg wet weight) and were not related to age, sex, duration of dementia or post-mortem interval. For each brain the number of tangles after extraction with SDS or NaOH was expressed as a percentage of the number of tangles in the aqueous (control) homogenate. The percentage of tangles insoluble in 1% SDS ($57 \pm 25\%$, mean $\pm$ SD), 5% SDS ($43 \pm 17\%$) and 0.2M NaOH ($37 \pm 22\%$) was not significantly related to age, sex, or post-mortem interval. There was, however, a significant negative correlation ($r = -0.57$, $p < 0.02$) between the percentage of tangles insoluble in 1% SDS and duration of dementia: that is, tangle solubility in 1% SDS increased with increasing duration of dementia (fig). This relationship was also significant ($r = -0.62$, $p < 0.05$) when only the Alzheimer-type dementia cases were considered. Duration of dementia was not significantly correlated with the percentage of tangles insoluble in 5% SDS or 0.2M NaOH. Plaque cores were essentially insoluble in all three agents.

A wide variation in tangle counts between Alzheimer-type dementia brains has been

reported using homogenates² and tissue sections.³ Although younger cases of Alzheimer-type dementia usually have more tangles than older cases,⁴ we observed no correlation between age and tangle counts, possibly because our cases were selected on the basis of severe pathology. The fact that the percentage of tangles soluble in 1% SDS increased with increasing duration of dementia suggests that in any one brain with severe Alzheimer-type dementia, tangles become more soluble and less stable with the passage of time. This enhanced solubility may reflect transformation of intracellular tangles to extracellular "ghost" tangles.⁵

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References

- Tomlinson BE, Corsellis JAN. Ageing and the dementias. In: Hume Adams J, Corsellis JAN, Duchen LW, eds. *Greenfield's Neuropathology*. London: Edward Arnold, 1985:958–66.
- Iqbal K, Zaidi T, Thompson CH, et al. Alzheimer paired helical filaments: bulk isolation, solubility and protein composition. *Acta Neuropathol (Berl)* 1984;62:167–77.
- Wilcock GK, Esiri MM, Bowen DM, et al. Alzheimer's disease. Correlation of cortical choline acetyltransferase activity with the severity of dementia and histological abnormalities. *J Neurol Sci* 1982;57:407–17.
- Tomlinson BE. Plaques, tangles and Alzheimer's disease. *Psychol Med* 1982;12:449–59.
- Rasool CG, Abraham C, Anderton BH, et al. Alzheimer's disease: Immunoreactivity of neurofibrillary tangles with anti-neurofilament and anti-paired helical filament antibodies. *Brain Res* 1984;310:249–60.

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Matters arising

Limb apraxia in patients with damage confined to the left basal ganglia and thalamus

Sir: We read with interest the important paper by De Renzi *et al*¹ on limb apraxia caused by a left basal ganglia and thalamic lesion. In a previous publication² we described in detail four cases of moderate/severe apraxia caused by small left subcortical strokes. In our series involvement of the basal ganglia was not a prominent finding. Instead the lesions appeared on the higher CT cuts, lateral to the body of the lateral ventricles, involving the periventricular white matter. Lesions in this location damage both longitudinal white matter tracts (superior occipito frontal and superior longitudinal fasciculus) and the anterior callosal radiations. In De Renzi *et al*'s cases 2

and 3, CT scans showed infarcts involving the white matter adjacent to the left ventricle. In case 4 the lesion also extended to upper CT slices, while in both haemorrhagic cases 1 and 5, longitudinal white matter tracts might be functionally disturbed by edema and pressure from the haematoma. Damage to the periventricular white matter causes apraxia by disconnecting the parietal from the frontal lobe and preventing visuokinaesthetic motor patterns from the parietal lobe from contributing to the premotor cortex in programming correct movements.

This comment does not imply denying a role to the basal ganglia on the execution of learned movements. In fact they are also important in gesture recognition,³ suggesting that spatio-temporal engrams of common and over-learned gestures may be stored on the basal ganglia.

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References

- De Renzi E, Faglioni P, Scarpa M, Crisi G. Limb apraxia in patients with damage confined to the left basal ganglia and thalamus. *J Neurol Neurosurg Psychiatry* 1986;49:1030–8.
- Kertesz A, Ferro JM. Lesion size and location in ideomotor apraxia. *Brain* 1984;107:921–33.
- Ferro JM, Martins IP, Mariano G, Castro-Caldas A. CT scan correlates of gesture recognition. *J Neurol Neurosurg Psychiatry* 1983;46:943–52.

De Renzi *et al* reply:

We agree with Dr Ferro's claim that white matter damage may cause apraxia by interrupting the tracts linking the posterior regions of the brain with the premotor cortex. We doubt, however, that this was the case in our patients. As indicated by Dejerine,¹ (figs 219–23), the arcuate fascicle runs above the level at which our CT scan upper cut was taken and is located between the external and the extreme capsule in the white matter underlying the sylvian operculum, namely, more laterally with respect