

Finally, we would like to add a word of caution and remind the authors that it would be wise to avoid deriving the measure of “objective duration” of sleep benefit from three scales filled in at home by patients without any further instructions than to fill them at waking, and during best and worst before drug intake.

As the only dopamine agonist mentioned in this study is bromocriptine, we would be grateful to know what year the study was conducted in.

In any case, although “little is known about sleep benefit”¹ any study concerning this phenomenon should certainly attempt to increase knowledge and avoid a further increment of confusion.

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- 1 Bateman DE, Levett K, Marsden CD. Sleep benefit in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1999;67:384–5.
- 2 Högl BE, Gómez-Arévalo G, García S, *et al.* A clinical, pharmacologic and polysomnographic study of sleep benefit in Parkinson's disease. *Neurology* 1998;50:1332–9.
- 3 Factor SA, McAlarney T, Sanchez-Ramos JR, *et al.* Sleep disorders and sleep effect in Parkinson's disease. *Mov Disord* 1990;5:280–5.

Bateman replies:

The difference in results is due to a different definition of sleep benefit. Sleep benefit as defined in our paper refers to mobility as good as “on” on waking, which wears off over a variable period. Högl *et al* define sleep benefit as “self perceived mobility in the morning before drug intake as better than during the rest of the day.”¹

The purpose of the first part of our study was to verify the existence of sleep benefit as we had defined it, particularly in view of the findings of Högl *et al* that “patients with sleep benefit had a small improvement between night and morning” and “sleep benefit patients were clearly in the “off” state during baseline motor examination”. Our inpatient study showed that six out of the 16 “who were studied from the moment of waking” performed as well as when they were “on” due to medication. Subsequently they spontaneously turned “off” to an identical state to “off” after medication. Four patients could not be studied from the moment of waking as they awoke before the investigator! We clearly found that sleep benefit is as good as “on” after medication and wears off, not an intermediate stable state between “on” and “off” as Högl *et al* have defined it, by subsequently giving these patients their normal medication on the same day and monitoring their response by half hourly UPDRS and ADL scores.

Aware of the patient's misperceptions about sleep benefit, we wished to confirm as objectively as possible the findings from our outpatient questionnaire by using the ADL rating scales. The inpatient study showed a correlation between motor UPDRS “on” score and ADL “on” score of $r=0.72$, $t=4.42$, $p<0.001$ and sleep benefit ratings $r=0.62$, $t=3.35$, $p<0.01$. The ADL maximum score is 125. It consists of 25 items that can be rated on a five point scale. Dr P Brown, originator of the scale, suggested that a change of 12 would

be sufficient to confirm sleep benefit. As we were aware that sleep benefit, confirmed by personal observation, can represent a substantial change in motor performance, this criterion seemed reasonable. The correlation between motor UPDRS and ADL scores in our study was good, showing that the ADL scores are generally a reliable measure, although there will inevitably be exceptions.

Our study showed that sleep benefit as we defined it was generally a feature of patients with young onset Parkinson's disease. A 73 year old patient described in their paper, with disease onset at 62, would be unlikely to have sleep benefit as we defined it. Their paper, as their figure 2 shows, refers to a different phenomenon.

D E BATEMAN

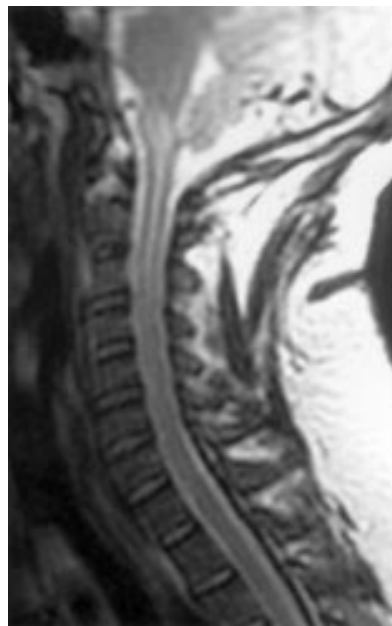
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Intracranial dural fistula as a cause of diffuse MRI enhancement of the cervical spinal cord

We read the recent short report by Bousson *et al*¹ on spinal MR findings in a patient with progressive myelopathy and intracranial dural arteriovenous fistula with great interest.

We recently had a 42 year old man admitted as an emergency with a 3 week history of stepwise altered sensation in both lower limbs ascending to the torso which progressed to weakness involving his legs and hands. Two years before this he had an episode of severe backache associated with a tight band of pain around the waist and significant bilateral leg weakness. Resolution occurred only after 4 months, when he was able to walk normally. On the current admission examination showed a spastic tetraparesis; there was minimally increased tone in the upper limbs, mild weakness of the small muscles of both hands, and marked pyramidal weakness of the legs with extensor plantar responses. He was unable to support his weight and was in



MRI of lower brain and spinal cord.

urinary retention. He had a sensory level at T5 although dorsal column function was preserved.

Brain and spinal cord MRI showed increased signal in the medulla extending into the upper cervical cord down to C4 (figure). Slightly prominent vessels were seen overlying the right cerebellar hemisphere and a varix was visible close to the torcula. There was no enhancement in the cord or medulla and no abnormal flow voids in the spinal veins. The changes were thought to represent a spinal cord infarct and in view of the “stuttering” course in his history we proceeded to cerebral angiography. This showed an arteriovenous fistula supplied by the left middle and posterior meningeal artery and both occipital arteries. Venous drainage was into prominent varices lying just to the left of the midline and in front of the transverse sinus and then on the transverse sinus itself.

After an unsuccessful attempt at embolisation via the arterial route, the fistula was occluded by packing the varix with Guglielmi detachable coils.

He made an uneventful recovery; after 2 months of intensive neurorehabilitation he recovered full function in his upper limbs and now has sufficient power in his legs to be able to walk with the aid of crutches.

This case emphasises that an intracranial arteriovenous fistula should be included in the differential diagnosis of increased signal on MRI of the cervical cord, even when dilated veins are not, as in this case, very apparent. Prodromal symptoms can occur and a careful history in a patient with ascending paraparesis and tetraparesis is essential. Endovascular occlusion at these fistulae can lead to useful improvement in neurological function.

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- 1 Bousson V, Brunereau L, Vahedi K, *et al.* Intracranial dural fistula as a cause of diffuse MR enhancement of the cervical spinal cord. *J Neurol Neurosurg Psychiatry* 1999;67:227–30.

Inverse relation between Braak stage and cerebrovascular pathology in Alzheimer predominant dementia

Goulding *et al*¹ carried out a preliminary retrospective postmortem analysis of 25 patients (13 men, 12 women, mean age 80.7 years) with the clinical diagnosis of Alzheimer-type dementia (only one with suspected multi-infarct dementia) and a 36.4% frequency of the ApoEε4 allele. Eighteen brains (89%) with neuritic Braak stage ≤4 had either additional cerebrovascular lesions (n=14), or Lewy bodies (n=1), or both (n=6), with a significant inverse correlation between cerebrovascular lesions and Braak stage. Forty eight per cent of the brains showed small focal infarcts, and only 20% disclosed “pure” Alzheimer's disease pathology. No association between the ε4 allele and any pathological variable was found. Based on these data, the authors emphasised the importance of screening for concomitant pathology in Alzheimer's disease, in which a cerebrovascular component has been suggested as an