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Letters to the Editor

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Axial T2 weighted (SE 2000/80) MRI shows an increased signal intensity in the right hemispheric cerebellar region without mass effect, corresponding to an ischaemic infarction in the territory of the posteroinferior cerebellar artery.

Physiologically, the vestibular pathways make contact with the ocular motor system, the spinal cord, and the vestibular cortex, contributing to the stabilisation of posture and perception of verticality and self motion.1.6 The tonic bilateral vestibular input builds up the actual central vestibular tone in the three major planes: horizontal or "yaw", sagittal or "pitch", and frontal or "roll".1.6 It seems that central pathways that mediate vestibular function in either of the three planes travel independently of each other, so that a specific lesion could cause a disorder restricted to one of them.1.6 The vestibular tone in the frontal or "roll" plane allows a correct perceptual, ocular, and postural alignment to the "gravitational vertical";1.6 an imbalance in this tone causes a lateral tilt with alteration in perception of verticality, head and body posture, alignment of the visual axes, or ocular torsion.1.6 Patients perceive the surroundings and their body as if they were tilted in the opposite direction to what the CNS erroneously computes as being vertical and try to adjust the visual objects and posture to it. Dietrich and Brandt showed that an alteration in the perceived verticality is not just the sensory consequence of the rotation of the eyes, as they can appear separately and are not proportional in degree.1 Furthermore, it is possible that not all the effects of tilt occur in one patient, and the perceptual disorder itself is the most sensitive sign of a vestibular tone imbalance in the frontal plane.1.6

Brainstem structures that mediate the vestibular tone in the "roll" plane include the vestibular nuclei and the interstitial nucleus of Cajal—perhaps the most rostral structure related to the control of vertical and torsional head and eye position. Both are connected by the medial longitudinal fasciculus, which crosses the midline in the pons.1.6 Visual vertical tilt is, then, ipsiversive to peripheral or pontomedullary lesions and contraversive to pontomesencephalic lesions and, in both cases, is usually associated with other tilt effects; in most rostral lesions it may be either ipsiversive or contraversive and is usually isolated.1.6 The role of the vestibular cerebellar structures with respect to the control of subjective verticality is not well known at the moment.2

Our patient's clinical findings suggest that he had an inclination of the internal representation of the gravitational vertical to his left and he tried to adjust both visual objects and posture to what he perceived as being vertical. It would have been interesting to assess whether there was ocular torsion, to define his clinical setting more exactly, but it makes no difference to interpretation as ocular torsion can be associated or not with perceptual or other tilt effects.1 Our patient showed a right hemispheric cerebellar ischaemic lesion, in a territory dependent on the posteroinferior cerebellar artery (PICA), with no mass effect and no brainstem or other alterations on MRI. The posterior and distal tilt was contraversive to the lesion. It is possible that an additional subtemporal lesion in the distribution of the PICA, not evident with clinical and imaging studies, produced the tilt effects in this case, because the more infratentorial arteries supply both brainstem and cerebellum and it is very difficult to differentiate the effects of cerebellar and brainstem lesions.2 But the tilt should then be ipsiversive, not contraversive, to the hypothetrical lesion. Therefore it is not likely that an associated medullary ischaemia could cause the tilt effects in our patient. A mesencephalic injury could cause this clinical picture but there were no other upper brainstem symptoms and MRI was normal at this level. A supratentorial disorder is unlikely because there were no MRI alterations and there were associated postural tilt effects. In this patient, we think that cerebellar dysfunction could be responsible for the tilt effects.

The present report confirms a previously hypothesised role for the cerebellar structures in the control of perception of verticality,1 and may contribute to a better knowledge of the pathophysiology and the topographic diagnosis of the central vestibular syndromes.

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Low striatal D2 receptor binding as assessed by [123I]IBSMPECT in patients with writer's cramp

Write's cramp is a form of idiopathic focal task specific dystonia. In accord with other studies on idiopathic and symptomatic dystonia, Tempel and Perlmutter suggested the presence of an abnormal striatohyalmeroc­
tidal drive in writer's cramp.1 In view of the
possible involvement of dopaminergic striatal receptors in dystonia, we measured the availability of striatal D2 receptors in patients with writer's cramp using \(^{[123I]}\)IBZM SPECT.

Ten consecutive right-handed patients (eight male and two female) were classified into four groups: (1) ten dystonic writer's cramp, (2) six dystonic writer's cramp depending on whether or not the symptoms appeared only during writing, (3) five writer's cramp dystonia, (4) six writer's cramp dystonia. All patients had written with neuroleptic, dopaminergic, or anticholinergic drugs or botulinum toxin. Hypokinesia, rigidity, and tremor were present in all patients. Bradykinesia of the hands was assessed with a pegboard test, measuring the time (s) required to insert eight pegs. Pegboard performance of patients was compared with that of 46 age-matched controls. Results of \(^{[123I]}\)IBZM SPECT were compared with 12 other age-matched controls from an earlier study.\(^7\)

A brain dedicated SPECT system, the Strichman Medical Equipment 810X, was used. Two hours after intravenous injection of \(^{[123I]}\)IBZM (185 MBq \(^{[123I]}\)IBZM \((45.5 	ext{ Ci kg}^{-1})\) in a Technical University, Eindhoven) and tomographic SPECT studies were performed. A maximum of 12 slices was made, starting at the orbitomeatal line and proceeding parallel to it (300 slices; interslice distance 5 mm). For analysis of specific striatal \(^{[123I]}\)IBZM binding, two slices with the highest striatal activity were summed and a template with fixed regions of interest for the striatum and occipital cortex was placed bilaterally on the summed image.\(^7\) The ratio of the striatal binding divided by the occipital binding quantifies specific binding.

The mean ages (table) did not differ among the three groups (t tests). Left and right \(^{[123I]}\)IBZM striatal : occipital ratios were significantly lower in patients than in controls.\(^7\) There was no significant asymmetry between \(^{[123I]}\)IBZM ratios for the hemispheres in patients or controls (repeated measures multivariate analysis of variance (MANOVA) tests involving side, group, and group by side: P > 0.05). The pegboard test results were similar in all patients with writer's cramp and controls in either hand (t tests), showing that the patients with writer's cramp did not have bradykinesia. There was no correlation between age or duration of disease and \(^{[123I]}\)IBZM ratios (Pearson's correlation coefficients). None of the variables differed between patients with simple and dystonic writer's cramp.

Our results suggest that the striatal dopaminergic system is involved in writer's cramp given that patients with writer's cramp have a significantly lower level of \(^{[123I]}\)IBZM binding than controls. Unfortunately, lack of an accurate measure of the severity of writer's cramp itself prevented us from studying the relation between severity of dystonia and \(^{[123I]}\)IBZM. Therefore, our findings are probably better explained by loss of D2 receptors on cholinergic striatal interneurons than D2 receptors on striatal spiny output neurons. The fact that the reduced availability of \(^{[123I]}\)IBZM binding in writer's cramp indicates disinhibition of the indirect pathway which might be expected to be accompanied by parkinsonism,\(^7\) in line with these findings, we conclude mean \(^{[123I]}\)IBZM ratio to be 1.43 (SD 0.16) in patients with definite hypokinetic-rigid symptoms due to multiple system atrophy or progressive supranuclear palsy,\(^7\) which is in the range as the values obtained in most of the present patients with writer's cramp (1.27-1.59).

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