valproate with 1 g carbamazepine, and 2 m clonazepam a day) also did not help. The failure of the medical treatment and the frequency of up to 10 or more attacks a day made normal functioning impossible for the patient and therefore a surgical option was considered particularly as the attacks were mainly unilateral. With the consent of the patient a left posteroventral medial pallidotomy was carried out as in a previously described technique.[4] She made an uneventful recovery and had no neurological complications. Immediately after operation the attacks of exercise induced dystonia had ceased completely having occurred more than 10 times a day immediately before the operation even on walking 10–15 steps. At the end of 6 months follow up she had been attack free, apart from occasional minor spasms of her left foot on exercise, despite normal activities; anticonvulsant treatment was gradually being withdrawn.

This is the first example of the usefulness of pallidotomy in a patient with any form of paroxysmal dyskinesia. Pallidotomy and more recently pallidal stimulation are currently being used as surgical techniques for advanced Parkinson's disease in patients with complications of levodopa treatment.[5] These procedures are particularly helpful in abolishing the levodopa induced dyskinesias.[5] Pallidotomy has also been found beneficial in patients with generalised dystonia[6] and recently a unilateral pallidotomy was reported to produce bilateral benefit in one patient with tardive dyskinesia.[7]

Given the improvement in our patient a unilateral pallidotomy could be considered as a treatment option in patients with stereotyped paroxysmal attacks as occur in paroxysmal exercise induced dystonia and the other paroxysmal dyskinesias such as paroxysmal non-kinesigenic dyskinesias in which treatment is often unsatisfactory.[2] A bilateral procedure could be considered in patients with bilateral attacks such as patients with paroxysmal non-kinesigenic dyskinesias who are unresponsive to drug treatment as bilateral pallidotomy seems to improve patients with generalised dystonia.[6]

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Sudden appearence of invalidating dyskinesia-dystonia and off fluctuations after the introduction of levodopa in two dopaminomimetic drug naive patients with stage IV Parkinson's disease

Hyperkinesias (dystonia, dyskinesia) are, with fluctuating akinesias, the most debilitating disturbances appearing during the advanced course of Parkinson's disease.[1] The origin of these disturbances is controversial; as hyperkinesias are seen after long term treatment with levodopa or dopaminoagonist drugs many researchers think that these motor fluctuations could be prevented by dopaminomimetic drug restriction,[1] others think that hyperkinesias will appear anyway after enough years, independently of dopaminomimetic drug restriction—that is, the supersensitivity of striatal structures to external administration of dopaminomimetic drugs is an epiphenomenon of natural degeneration in Parkinson's disease.[2]

Ten years apart from one another we had the chance to observe two patients with Parkinson's disease with prevalent akinetic symptoms who came to us already in an advanced stage of Parkinson's disease, classified as stage IV according to the Hoehn and Yahr scale.[3] These patients had never been treated with dopaminomimetic agents (levodopa, dopaminoagonists), or amantadine or anticholinergic drugs, and both developed dyskinesias and motor fluctuations when levodopa was increased to the amounts commonly used in patients with stage III-IV Parkinson's disease treated for 6–10 years.

Patient 1 was a 76 year old man living in the inner mountainous part of central Italy. When he came to us he was incapable of rising from his bed; hypomimia, akinesia, flexed dystonic posture, and rigidity were rated 20 at motor examination with the unified Parkinson's disease rating scale (UPDRS),[4] modest 4–5 Hz tremor was present at the upper limbs, left and right intensity was rated 4 at the UPDRS, and utterances were feeble and incomprehensible. The total UPDRS score was 126 (SD 4).

It was possible to reconstruct his clinical history from relatives, and apparently his early stooped posture and akinetic disturbances had appeared at least 10 years before, but was considered to be due to severe arthrosis and was treated with salicilates. Brain MRI at admission was normal. Early treatment with 62.5 mg levodopa thrice daily+benserazide did not induce gastrointestinal intolerance but did not change his UPDRS score and was rapidly (4 days) increased to 250 mg levodopa four times daily+benserazide. Oromandibular dyskinesias, dystonic neck and trunk leftward rotations, and left leg dyskinesias were noticed 2 days after the 1g daily levodopa dosage was reached. Dystonic-dyskinetic movements appeared 20–30 minutes after the first (7 00 am) 250 mg levodopa+benserazide tablet, lasted through the day, and were painful, mostly in the afternoon.

His UPDRS scores were 72 (SD 3) from 8 00 am to 2 00 pm, 88 (SD 3) from 2 00 pm to 8 00 pm. Dyskinesia-dystonia scores were 11 (SD 2) from 8 00 am to 10 00 pm and 13 (SD 2) from 2 00 pm to 10 00 pm. Because dyskinetic-dystonic movements were not tolerated, the daily levodopa had to be reduced to 62.5 mg every 3 hours (total 375 mg/day). With this treatment UPDRS scores were 86 (SD 4); oromandibular dyskinesias and torsional dystonias were still present and rated 10 (SD 2). Bromocriptine up to 10 mg/day was not tolerated. During the next 2 years levodopa treatment could not be increased. His UPDRS scores was 89 at 2 weeks before his sudden death due to apparent cardiovascular complications with cardiac arrest. A postmortem examination showed anteroinferior myocardial infarction, and normal brain structures with depigmentation of the nigral structure. Mesencephalic structures were cut into horizontal 7 μm thick sections and stained with haematoxylin and eosin; three Lewy bodies were found in 109 identified pigmented cells and cell loss was about 86% compared with age matched controls and literature reports.[5]

Patient 2 was a 72 year old man from the same region of central Italy. He came to us akinetic and rigid, with a stooped posture and minor tremor of both upper limbs, and was confined to a wheelchair. Utterances were feeble and incomprehensible. His total UPDRS motor score was 93 for upper limbs, rest tremor was only 2. He had been incapable of walking during the past year, and spent his time on a chair, where he also slept. His relatives described the progressive deterioration in the past 10 years, from the stooped posture to progressive akinesia and language and walking deterioration. His disturbances were attributed to senescence-arthrosis, until comparison with other patients with Parkinson's disease living in the same region, prompted the neurological consultation. This patient was treated with increasing doses of levodopa+benserazide after a one week trial with 125 mg thrice daily had not changed his UPDRS score. A 1.5 g daily dose of levodopa (in 6 administrations) was reached in the next week. Oromandibular dyskinesias and leftward torsion dystonias appeared in the same week. He became able to walk unaided but tremor of the upper limbs was still present, at rest and during walking. His dyskinesias were uncomfortable, although not painful, and his stooped posture was only slightly modified (score 3 from 4). His UPDRS score during treatment was 57, tremor score was 4, and dyskinesia-dystonia score was 7 (SD 1). Treatment was then reduced to 500 mg levodopa+benserazide with 15 mg ropinirole (increasing in three weeks from 1.5 mg/day). With this treatment dyskinesias were reduced, UPDRS score for dyskinesias was 4 in the morning, 5 in the afternoon, and he was able to walk unaided in the morning. His UPDRS score was 65 (SD 4) in the morning and 79 (SD 2) in the afternoon. Brain MRI was normal.

In conclusion, both patients came to us with a levodopa responsive parkinsonism that had appeared, according to history reconstruction, at least 10 years before. Both could be considered at least in stage IV of the Hoehn and Yahr scale.[3] Both had never been treated with dopaminomimetic drugs or with other drugs currently used in the treatment of Parkinson's disease. In both patients dykinesias and dystonias, accompanied by motor fluctuations throughout the day, appeared in the first week. After that a levodopa dose able to modify the akinesia and rigidity scores was reached. Reduction of levodopa dosage in patient 2 and introduction of a dopaminoagonist improved dyskinesia but the total UPDRS score was higher than the score obtained with 1.5 g/day levodopa.

These findings favour the hypothesis suggesting that hyperkinetic fluctuations are not dependent on prolonged dopaminomimetic drug administration but on the natural

course of Parkinson's disease.[6] In favour of this viewpoint is the finding that MPTP

exposed parkinsonian patients had severe loss of dopaminergic neurons and developed dyskinesias rapidly after starting levodopa therapy.[7] Caveats about this conclusion must be placed, relative to the fact that both patients had prominently akinetic disturbances, and thus prevalent tremorigenic parkinsonisms might have different courses with different occurrences of complications during levodopa treatment.

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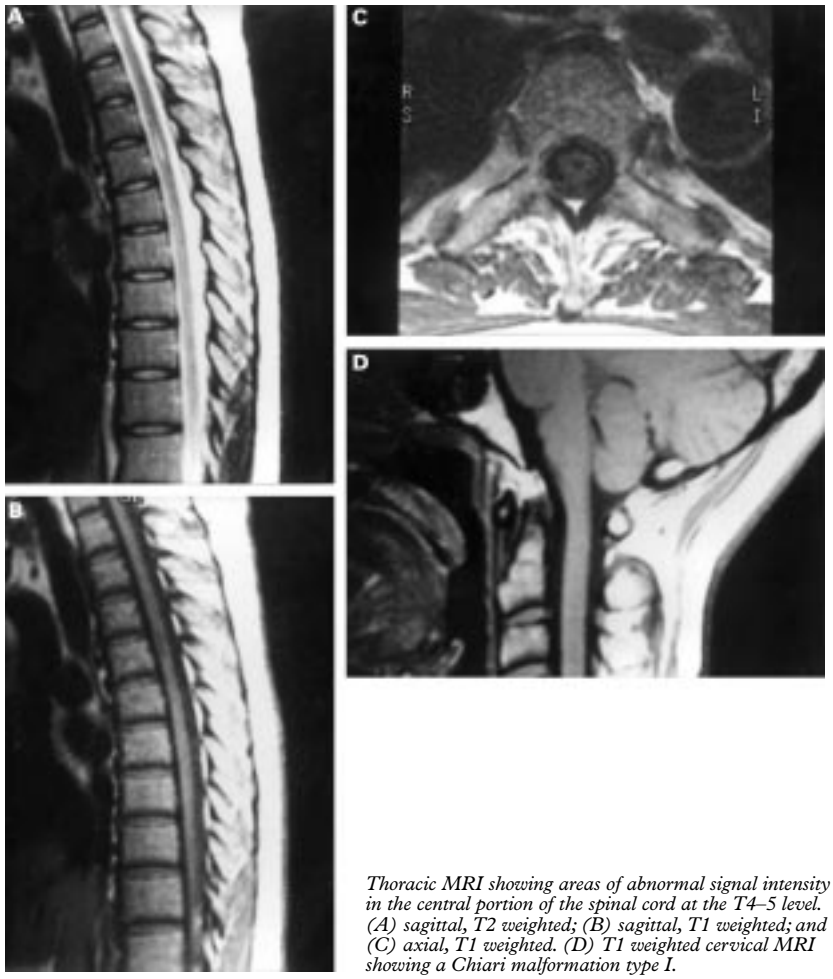
Reversible hydromyelia in a synchronised swimmer with recurrent thoracic girdle pains

Synchronised swimming is considered a low injury competitive aquatic sport for all ages, although stress related symptoms such as knee or shoulder pain are common.[1] We report a case of recurrent thoracic girdle pains in a professional instructor of this sport due to reversible hydromyelia.

The patient was a 40 year old woman with 15 years experience as an instructor of synchronised swimming. She was in good health up to 15 December 1996 when she developed insidious left thoracic pains 2 days after an underwater exhibition performance that was longer and more strenuous than usual. The pains spontaneously disappeared over the next 10 days. On 6 February 1997, she again experienced similar thoracic girdle pains 2 days after prolonged lessons and a week later came to our hospital. The pain was dull and increased intermittently, in particular when she turned over in bed at night. On examination she was alert, afibrile, and normotensive. Neurological examination disclosed no abnormalities in her cranial nerve, motor, sensory, and autonomic functions. There was no nuchal rigidity. Routine laboratory findings for blood, urine and CSF were all normal. Bleeding and whole blood clotting times were normal. Tests for oligoclonal bands and myelin basic proteins of CSF were negative. Thoracic cord MRI detected areas of abnormal signal intensity (high in the T2 weighted and low in the T1 weighted images with no enhancement by Gd-DTPA) in the central portion of the spinal cord at the T4–5 level (figure A, B, C). The intensities were linear-elliptic in the sagittal plane, and small and round in the axial plane, indicative of hydromyelia (central canal dilatation). Myelography and MRI of the cervical and lumbar cord and of the brain showed no abnormalities except for a Chiari malformation type I (figure D). Only a recommendation not to strain or hold her breath was given, and the pains resolved spontaneously over the next 10 days. Two months later follow up MRI of the thoracic cord showed the absence of the initial abnormal finding.



Thoracic MRI showing areas of abnormal signal intensity in the central portion of the spinal cord at the T4–5 level. (A) sagittal, T2 weighted; (B) sagittal, T1 weighted; and (C) axial, T1 weighted. (D) T1 weighted cervical MRI showing a Chiari malformation type I.

Synchronised swimming requires flexibility, kinesthetic awareness, and aerobic conditioning. Few acute injuries occur in the participation of this sport, but overuse injuries such as knee pain associated with the eggbeater kick and shoulder pain associated with sculling are becoming more common.[1] Therefore the thoracic pain in our patient might have been wrongly diagnosed as having a musculoskeletal origin.

The combination of breath holding and the performance of compulsory figures, such as those that involve hyperextension of the spine, can markedly raise intrathoracic and intracranial pressures during prolonged underwater performances, and may cause dangerous hydromyelia (central canal dilatation) due to changes in the CSF dynamics, especially in those who have a Chiari malformation.[2] We have no idea why this hydromyelia developed at the upper thoracic level.

We conclude that synchronised swimming rules should not encourage prolonged underwater performances and unnatural compulsory figures, and that prior checks for risk factors such as a Chiari malformation should be made.

The lumbar puncture, myelography and follow up MRI were done at the Department of Neurology, the National Institute of Neuroscience Kohnodai Hospital, Ichikawa, Chiba. We thank Dr Takeshi Sato and his colleagues for their expert help.

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Barium carbonate intoxication: an electrophysiological study

Barium carbonate is an uncommon poisoning agent in India. This whitish coloured powder is available over the shelf from the