eye would be expected to increase the binocularly perceived distance of an object moving left to right, and this is just what is observed. The magnitude of the change in disparity caused by a filter is approximately proportional to the speed of the moving object.

The Pulfrich effect can be demonstrated spontaneously (ie, without a neutral density filter) in patients with multiple sclerosis because of the delayed optic nerve conduction seen after unilateral optic neuritis in multiple sclerosis.³ The detrimental impact of the Pulfrich effect on sporting performance with the use of a neutral density filter has previously been described.⁴ The Pulfrich phenomenon is normally seen in patients with clinical signs of optic neuropathy, such as an afferent papillary defect or colour vision abnormalities. Here, we report the case of a patient who presented with the solitary complaint of reduced sporting performance without any other associated symptoms or clinical signs.

A 33-year-old man presented to the accident and emergency department with problems playing squash. Over a 1 month period, he had noticed difficulty hitting the squash ball. If the ball was hit to his right side he returned it with ease. However, if it was placed to his left side he missed it consistently. He had no other visual symptoms and had no significant past medical history.

On examination, his visual acuity was 6/4 in both eyes. He had no relative afferent pupil defect, no manifest or latent deviation. His ocular range of movement was normal with no evidence of internuclear ophthalmoplegia. He showed normal coordination and cerebellar function. Colour vision (100 Hue Test, total error score right eye = 20, left eye = 36) and visual fields were also normal. At this point a Pulfrich-like phenomenon was suspected. A pattern electroretinogram (ERG) and pattern visual evoked potential (VEP) were therefore performed.

The pattern ERG was normal but the pattern VEP showed an abnormal P100 between 126–132.5 ms in the right eye with a normal P100 in the left eye (fig 1) indicative of conduction delay within the right optic nerve. An MRI brain scan was then performed. FLAIR image sequence revealed two discrete lesions in the deep white matter on the right hemisphere and one lesion on the right anterior visual pathway. The findings were consistent with demyelination. Referral to the neurologist was arranged to decide on the need for further treatment.

In order to improve this patient's symptoms it was decided that reversal of the Pulfrich effect could be beneficial. VEP measurements were repeated using neutral density filters of incremental values placed in front of the normal eye. The VEP was brought to approximate the abnormal eye (fig 1). With increasing neutral density filters, the VEP amplitude was almost unaltered but the latency progressively increased. This filter was then prescribed in a pair of squash goggles. Further review showed an improvement in his sporting performance.

There was a 41 ms delay between the VEP of this man's eyes. The delay would result in the ball being apparently in a different spatial location for each eye. At 20 m/s this would correspond to a spatial difference of 82 cm. For fast moving ball games it is therefore easy to appreciate why unilateral delays in optic nerve conduction can create significant hand-eye coordination problems which would not be apparent for stationary targets. The increased difficulty in hitting the ball on the left side

with a right sided pathology may relate to the fact that on this side the ball would be misjudged such that it would be appear to be further away than it was and so would be played late. On the right side, conversely, it would appear closer and hence would be played earlier but with a greater chance of connecting with the ball.

In this patient the only symptoms related to reduced sporting ability. Other tests performed in the clinic were all normal. Specialised electrophysiological testing and MRI imaging were required to determine an organic basis for his unusual presenting complaint. This case highlights how a simple theoretical concept can be applied to a clinical situation and the need to consider Pulfrich-like symptoms as a possible indicator of demyelinating disease even in the presence of otherwise normal clinical examination.

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Braille alexia: an apperceptive tactile agnosia?

Alexia for Braille reading is rarely reported.^{1–6} A further case is presented in which the clinical features may give some insight into the neurobiological mechanisms.

Neurological consultation was requested 17 days after apparently uncomplicated coronary artery bypass grafting in a 73-year old right-handed woman who complained that she could no longer read properly. Blind from birth because of anophthalmia, she learned to read Braille at age 7 years using her left index finger, or middle finger if the index finger became "tired" (the right hemisphere may have better discrimination for complex spatial patterns^{4,7}). She was a proficient Braille reader, normally reading 8–10 book chapters per day; however, on initial recovery from the operation, she could not read at all. Matters improved over the following days but her reading was still much slower than preoperatively (1–2 book chapters/day) and she reported making errors when reading, necessitating re-reading, although she had no difficulty understanding what she read. Past medical history was unremarkable, aside from a right carotid endarterectomy performed several months previously.

On examination, her spoken language was fluent with no evidence of motor or sensory aphasia. There was no left-sided sensory neglect or extinction, and no finger agnosia. Testing stereognosis in the left hand, she was able to identify a pen, ring, paper clip and watch, but was slow to identify a key, could not decide on the denomination of a coin (50p, heptagonal or 10p, circular) and thought a £1 coin was a badge, although she identified this immediately with the right hand. Two point discrimination was 3 mm on the pulp of the right index finger (minimum spacing possible between tines) but 5 mm on the pulp of the left index finger. As she had never learned letters or Arabic numerals, it was not possible to test for graphaesthesia.

MRI of the brain showed a few punctate high signal hyperintense lesions on T₂ weighted and FLAIR sequences in the subcortical white matter, thought to be ischaemic in origin, including one subjacent to the right motor cortex in the region of the internal watershed between the anterior and middle cerebral artery territories (fig 1). In addition, there was marked acquired global brain atrophy, including the occipital lobes.

Three months later, she had still not returned to her previous level of reading fluency. A working diagnosis of Braille alexia due to an apperceptive tactile agnosia was made, of uncertain aetiology: acute onset and partial recovery strongly suggested a vascular event, supported by the structural brain imaging appearances, although neurodegenerative disorders first manifesting with acute postoperative language problems have been reported.⁸

Previous reports of Braille alexia are rare, and clinically heterogeneous with respect to early or late onset of blindness, premorbid fluency of Braille reading and presence or absence of additional neurological signs such as aphasia and hemiparesis, these differences reflecting lesion location and aetiology (vascular, neoplastic, neurodegenerative).^{1–6} Sensory limb symptoms, but without clinical or neurophysiological correlate, have been reported.⁵ In those cases undergoing neuroimaging, structural correlates have included bilateral occipital infarctions⁵ and selective calcarine atrophy in a patient with visual hallucinations,⁶ findings which correlate with functional imaging

Figure 1 Axial MRI of the brain (FLAIR) showing hyperintense white matter signal change subjacent to the right motor cortex, and marked parieto-occipital brain atrophy.

studies showing that visual cortical areas process tactile information in Braille readers.⁹ Dissociation between verbal and musical alexia in Braille has been reported following left middle cerebral artery infarction in a professional organist blind from childhood.³

Braille alexia, the tactile homologue of pure alexia (alexia without agraphia), a category specific visual agnosia, may result from disruption of different, possibly overlapping, psychoperceptual mechanisms, some analogous to those postulated in pure alexia.¹⁰ It may reflect problems integrating tactile information over the temporal or spatial domains, associative forms of agnosia (or tactile simultanagnosia). A frontal-parietal network may contribute to the integration of perception with action over time,¹¹ and right hemisphere lesions may be associated with impaired integration of spatial information from multiple stimuli.¹² Alternatively, Braille alexia may reflect perceptual impairment, an apperceptive form of agnosia. As Braille characters are close to the limits of normal perceptual resolution, impaired light touch perception following damage to the primary sensorimotor cortex or its connections may result in degraded tactile identification and slowed Braille reading speed.

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Acute myelopathy selectively involving lumbar anterior horns following intranasal insufflation of ecstasy and heroin

We report a patient who developed acute myelopathy after intranasal insufflation of amphetamines and heroin. The functional prognosis was very poor; after 4 months, she remained paraplegic. MRI imaging showed selective T2 hyperintensity and intense enhancement confined to the spinal anterior horns and lumbar nerve roots and plexus. This unique MRI pattern, together with neurophysiological data, suggests that the pathological process at the first primary affected spinal anterior horns (SAH), conditioning motoneuron cell death, and then nerve roots and lumbar plexus as a consequence of wallerian degeneration

Case report

A 17-year-old girl was admitted to the emergency department in a drowsy state and unable to walk after an overdose of intranasal insufflated heroin and amphetamines. After a few hours, drowsiness progressed to stupor, and progressive weakness in all four limbs, mainly involving the lower limbs, developed. At that time, laboratory data showed massive rhabdomyolysis (creatinine phosphokinase 36 880 mg/dl) with acute renal failure (ARF), and hepatic failure; medical therapy was promptly started. The patient's past medical history was unremarkable except for habitual use of amphetamines (ecstasy) and cannabinoids since the age of 12 years. The previous week she had insufflated heroin about once a day; the previous night she reported a double dose of heroin consumption, and a high dose (approximately 1 g) of intranasal insufflation of amphetamines. The next day the patient was alert and cooperative but complained about diffuse muscle pain and tenderness, prevailing

in the lower limbs, where weakness rapidly worsened to flaccid, areflexic paralysis.

Urinary chromatography detected amphetamines (3-4-methylenedioxymethamphetamine (MDMA) 7680 ng/ml, 3-4-methylenedioxymphetamine 2000 ng/ml) and opiates (>2000 ng/ml, morphine 228 ng/ml). CSF protein content was slightly increased with no intrathecal Ig synthesis; glucose, chloride and cells were within the normal range. All virological analyses, including HIV, were negative. Rhabdomyolysis related ARF completely normalised by day 10, with resumption of spontaneous diuresis; from that time on, no more myalgias were reported. Muscle weakness promptly improved in the upper limbs to normal strength while flaccid paralysis persisted in the lower limbs. The patient had no sensory symptoms or signs, and no sphincteric abnormalities were observed.

Motor evoked potentials recorded from foot muscles were absent from day 9 onwards; both sural nerve sensory action potentials (SAPs) and tibial somatosensory evoked potentials (SSEPs) were normal. On day 16, electromyography revealed complete unexcitability of the leg motor nerves, together with fibrillation potentials without any voluntary electromyographic activity in all lower limbs and paraspinal muscles innervated by L3–S1 roots. Quantitative sensory test, skin sympathetic reflex and urodynamic test were all within the normal range.

MRI performed at the beginning of symptoms was negative. One month later, MRI showed selective T2 hyperintensity of the anterior horns and signal alteration of lumbar nerve roots, associated with volume increase and intense gadolinium enhancement (fig 1).

Intravenous high dose steroid therapy was started with no clinical benefit. MRI signal alteration and enhancement of the nerve roots almost completely disappeared while signal alteration of the anterior horns remained, although it was reduced in intensity. Four months later, the patient was still paraplegic.

Figure 1 MRI imaging. (A) Post-gadolinium (standard dose) T1 weighted MRI image, coronal view: linear enhancement along the nerve roots is evident. (B) T2 weighted MRI image, coronal plane: widespread T2 hyperintensity along nerve roots is more evident on the right side. (C) Post-gadolinium (standard dose) T1 weighted MRI image, axial view: selective anterior horn intense enhancement is seen. (D) T2 weighted MRI image, axial plane: selective anterior horn T2 hyperintensity is noted.