dysfunctions such as aphasia, hemineglect, and memory disturbance, and more rarely hemiparesis.[2–5]

In our patients the symptoms were mild and transient. The first patient had blurred vision on the left side for 5 days and in the second patient, the visual field defect lasted for a few minutes, although CT or MRI disclosed the corresponding lesions of the LPCA occlusion.

The LPCAs mainly originate from the ambient segment of the PCA trunk or its cortical branches. The number of twigs averaged 4.0 (range 1–9).[1] When two or more branches are present, anterior branches penetrate the choroid fissure to supply the choroid plexus of the inferior horn of the lateral ventricle. Posterior branches supply the choroid plexus in the trigone of the lateral ventricle and far anteriorly in the third ventricle. They also supply the pulvinar, medial, and lateral geniculate bodies, fornix, and midbrain tegmentum.[1 6] Many anastomoses between the branches of the LPCA and anterior choroid artery are present at the anterior third of the temporal horn as are many connections between the LPCA and medial posterior choroid arteries at the level of the foramen of Monro.[1]

Infrequent occurrence of occlusion of the LPCA is speculated to be due to such multiple branches and rich anastomosis. The number of arteries and development of anastomosis vary. Such ample anatomical variation in the supplying territory of these arteries may explain the wide range of severity and duration of symptoms of both our patients and others reported. Our patients represent the milder form of the clinical features of infarction of the LPCA, which has not been highlighted. The LPCA occlusion may present only with such minor complaints. Because LPCA occlusion may be prodromal signs of more serious brain ischaemia,[5] we need to know a wide range of the clinical symptoms related to occlusion of the LPCA.

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Neurostimulation of the ventral intermediate thalamic nucleus alleviates hereditary essential myoclonus

Therapy in hereditary essential myoclonus (HEM), a disabling movement disorder, is difficult in most cases, especially in regard to the myoclonic syndrome.[1] This is the first report on the amelioration (around 65%) of HEM by high frequency deep brain stimulation of the ventral intermediate thalamic nucleus (VIM) in a 61 year old male patient with medically intractable HEM.

The jerky movement disorder began in his neck and shoulders around the age of 6 and gradually progressed, especially in the proximal right arm, leading to a severe reduction of dexterity and inability to write. He was unable to continue working as a janitor when he was 55.

On examination he presented mainly oscillating, "lightning", irregular, jerky, asynchronous movements of his forehead, neck, proximal right arm, less severe of the left arm, and discreetly of the upper trunk, which was exaggerated by walking, writing or drinking and could be elicited by loud acoustic stimuli. The disorder improved at rest and disappeared in sleep. Additionally, he had a slight torticollis to the left, superimposed by inconstant no-no myocloni and a discrete action tremor of both hands, all of which complies with the HEM criteria as outlined by Quinn.[1] The remaining neurological status was unremarkable.

Four of the six children of the patient had possible myoclonus syndrome. Additionally, two of four siblings of our index patient were definitely affected by the movement disorder, whereas the parents seemed to be unaffected (a full account of the family history will be provided elsewhere, manuscript in preparation).

Alcohol consumption markedly attenuated the movement disorder with a clear rebound phenomena after alcohol withdrawal, leading to chronic alcohol misuse. A latent suicidal syndrome with regard to the exhausted medical and social perspectives complicated the situation.

Extensive drug treatment trials (including monotherapy or combination therapy with the following agents: apomorphine, both oral and intrathecal baclofen, cervical application of botulinum toxin, carbamazepine, carbidopa, clonazepam, clonidine, clozapine, fluphenazine, haloperidol, 5-hydroxytryptophan, levodopa, lithium, paroxetine, piracetam, primidone, propranolol, tiapride, trihexyphenidyl, and valproate) had no effect on the myoclonic syndrome except for a mild improvement under diazepam.

Electroencephalography, including back averaging of the EEG activity preceding spontaneous jerks, polymyography, and somatosensory evoked potentials of the median nerve, cranial CT, MRI, and a lumbar puncture did not show any abnormalities.

In this therapeutically hopeless situation, which was complicated by a latent suicidal syndrome secondary to the lacking therapeutical options, unilateral deep brain stimulation of the left VIM (essentially as outlined in Benabid *et al*[2]) was performed after informed consent of the patient, for three reasons: Firstly, VIM stimulation is considered to be a "low risk" and effective therapy in patients with drug refractory essential tremor, which is also characterised by alcohol attenuation.[2] Secondly, there are anecdotal reports on the improvement of myoclonic disorders by thalamatomy.[3] Thirdly, in the absence of a therapeutical effect the electrode could have been removed or switched off.

Intraoperatively and in the subsequent 12 months deep brain stimulation (2.8 V, 130 Hz, 90 μs) produced a pronounced and perstistent reduction of the jerks and the action tremor of the right arm and to a lesser extent of the jerks of the neck and the trunk. The improvement could be postoperatively quantified by a roughly 65% decrease in myoclonus score (table).[4] In practical terms, this was reflected by the use of the right hand for handwriting for the first time after 15 years of incapacity. Interestingly, the optimal stimulation parameters corresponded to those used in deep brain stimulation for essential tremor.[2] The mild dystonic symptoms (slight cervical dystonia) were not affected by DBS with different stimulation parameters. Preoperative medication comprised 60 mg diazepam a day and could be reduced to 10 mg diazepam a day at 12 months postoperatively.

Stimulation of the VIM is an established neurosurgical method for the treatment of pharmacologically intractable essential tremor, although its mechanisms of action remain to be elucidated.[2] It is interesting to note that both diseases (HEM and essential tremor) may be improved by alcohol intake, which could hint at overlapping pathophysiological mechanisms. Importantly, it has been reported that low frequency stimulation (2 Hz-5 Hz) of the VIM in patients with Parkinson's disease may even trigger mycloni.[5] However, we have been unable to aggravate the myoclonic syndrome in our patient by low frequency stimulation (2 Hz-5 Hz), which may point to different mechanisms of actions of stimulus induced myocloni in Parkinson's disease versus HEM.

In conclusion, this case report suggests that neurostimulation of the VIM may be an effective therapeutic alternative for medically intractable HEM and suggests that the thalamus may be a myoclonus sensitive site.[6]

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Myoclonus rating scores

	Action induced myoclonus	*Stimulus induced myoclonus*	*Myoclonus at rest*	*Dis-ability score*	*Global assessment*	*Sum total*	*Sum (%)*
Preop	50	34	18	11	3	116	100
DBS-off	36.5	24	2.5	9	3	75	65
DBS-on	29	4	2.5	5	1.5	42	36

Myoclonus rating scores preoperatively, on and off deep brain stimulation (DBS) 10 months postoperatively, modified according toTruong and Fahn.[4] Postoperative ratings were obtained by an observer (TT), who had been blinded to the stimulation status ("on" or "off").

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Invasive intracranial aspergillosis secondary to intranasal corticosteroids

Intranasal corticosteroids are widely available; we describe a serious and previously unreported complication of their use. A 41 year old Asian woman was admitted acutely, after two generalised seizures. She described a 2 year history of anosmia, nasal stuffiness, and rhinorrhoea which was diagnosed 12 months previously, by both her general practitioner and an ear, nose, and throat surgeon, as allergic rhinitis, with patch testing confirming allergy to house dust mite II, although no imaging was performed at this time.

This was treated with the topical nasal steroid spray Flixonase (fluticasone propionate 50 mcg/spray). The remainder of the history was unremarkable and she had not been back to Asia for 6 years.

General examination was normal and there were no focal neurological signs, although she was rather confused and disinhibited.

Full blood count; urea and electrolytes; liver function tests, serum calcium; random blood glucose; thyroid function tests; serum angiotensin converting enzyme; blood cultures; cytoplasmic pattern (c-) antineutrophilic antibodies (ANCA) and perinuclear (p-) ANCA; an ECG; and chest radiography were all normal.

An EEG showed only diffuse slowing, consistent with a postictal state. Brain CT showed an ill defined area of reduced attenuation in the right frontal lobe, with no contrast enhancement, and a soft tissue mass in the paranasal and sphenoid sinuses, which was eroding through the base of the skull.

Brain MRI (fig 1) showed swelling of the right frontal pole with oedema and a small, intensely enhancing, soft tissue mass in the floor of the anterior fossa, which was probably in communication with the soft tissue mass in the paranasal sinuses.

A biopsy of the posterior ethmoid region showed granulomatous change with fungal hyphae seen and *Aspergillus fumigatus* was grown on culture. Cultures for tuberculosis were negative and HIV type 1 and 2 serology was negative.

Anticonvulsant therapy with modified release carbamazepine (carbamazepine MR) was started and the nasal spray was stopped. She was commenced on intravenous amphoterecin B (250 mcg/kg/24 hours), which within 2 days led to renal impairment and had to be discontinued.

Itraconazole syrup (200 mg twice daily) was substituted and adjusted according to serum concentrations, and the therapeutic range (4 hours postdose 5–15 mg/l) was achieved with a dose of 300 mg twice daily.

Over subsequent weeks she improved and became less confused and disinhibited, although the anosmia remained. On review 8 months later she remained well and a repeat MRI (fig 2), although still showing residual disease, showed a marked reduction in its extent and in particular a dramatic decrease in the size of the subfrontal lesion.

The fungal genus *Aspergillus* causes a range of diseases including allergic; non-invasive; invasive and fulminant aspergillosis. The two commonest strains are *A flavus and A fumigatus*. Invasive aspergillosis is characterised by tissue invasion with *Aspergillus* hyphae and is most commonly seen in immunocompromised people, but there are a few case reports in apparently immunocompetent people.[1 2] It differs from allergic aspergillus sinusitis and allergic bronchopulmonary aspergillosis, which are immune mediated reactions to *Aspergillus* infection.

There are three possible routes of invasion into the CNS: haematogeonous, usually from the lung; direct spread from an area adjacent to the CNS, and iatrogenic introduction, usually after neurosurgical procedures.

The commonest route of spread is haematogeonous and the most frequent CNS manifestation is an intracerebral abscess, but it can also cause meningitis or meningoencephalitis; granulomas; cerebrovascular disease; vasculitis, or cranial nerve palsies[1].

Intracranial extension from an adjacent area is only seen in advanced cases, and extension through the skull base is rare[1].

The prognosis in this disease is very poor and invasive CNS aspergillosis is often a fatal disease regardless of the mode of therapy, with mortality upwards of 80%, and very few long term survivors of cranial and intracranial aspergillosis have been reported in the literature.[1–5]

Amphoterecin B is effective against *Aspergillus* but its toxicity and mode of administration limit its usefulness. Itraconazole is well absorbed orally, is comparatively non-toxic, and is effective against *Aspergillus*; however, serum concentrations need monitoring.[2 4]

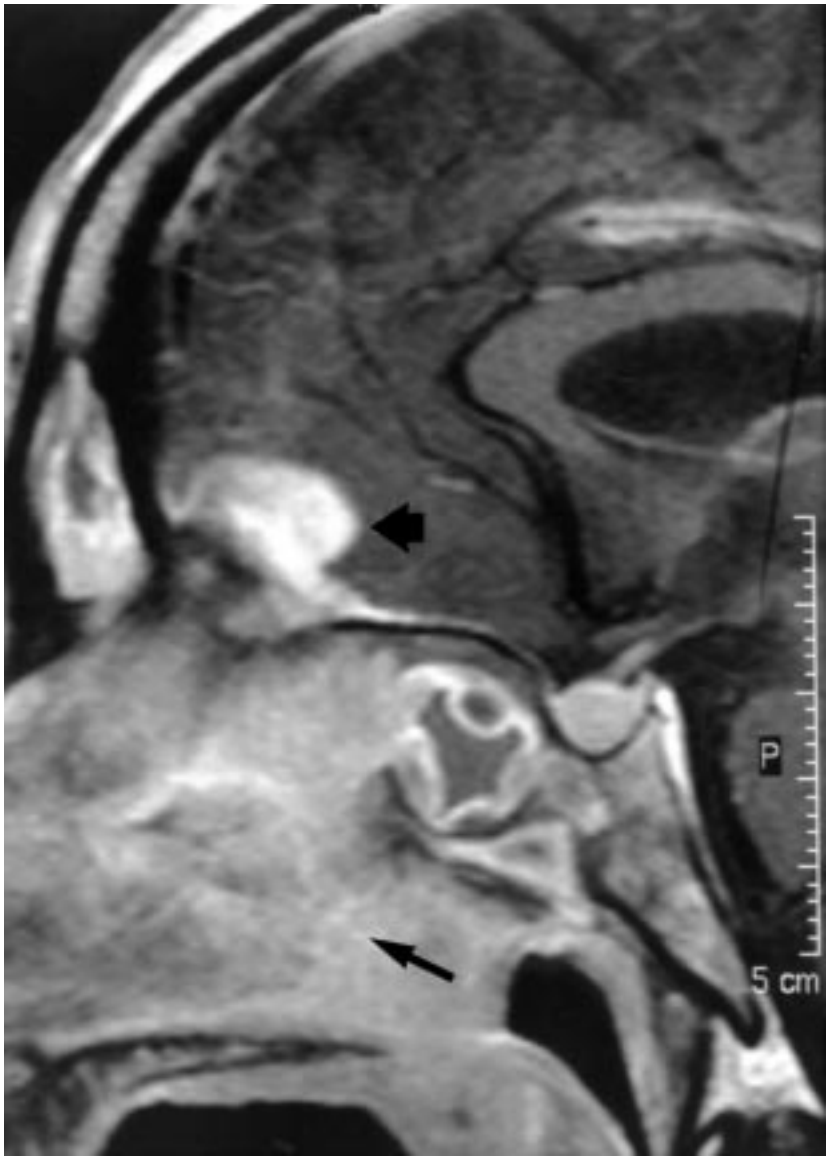

Figure 1 Gadolinium enhanced, T1 weighted, sagittal MRI, showing diffuse disease throughout the sinuses (thin arrow) and an enhancing lesion in the subfrontal area (thick arrow).