

ball) were accurately identified. His tendon reflexes were slightly increased on the right and there was a Babinski's sign. His right upper limb showed a slight pronator drift, slowing of rapid alternating movements (pronation/supination), and fine finger tipping. Writing was impaired and hardly readable. The finger to nose test was slightly dysmetric on the right. There was no ataxia on the heel to knee test. Superficial and deep sensation were intact. His gait was slightly unsteady with a tendency to fall to the right.

At discharge after three weeks the dysarthria had almost resolved and he was not so clumsy. Writing was still difficult and tendon reflexes were more pronounced on the right side.

T1 and T2 weighted MRI on day 7 documented infarction of the central two thirds of the left ventral cerebral peduncle, extending as a small strip into the left tegmental region at the pontomesencephalic junction (figure). On day 7 HMPAO-SPECT showed symmetric and homogeneous tracer uptake in both cerebellar hemispheres (right:left ratio 0.97).

Transcranial magnetic stimulation (Magstim 200 S) was performed on days 4 and 20. Compound muscle action potentials were recorded using special surface electrodes from both halves of the tongue, both sides of the orbicularis oris muscle, and both abductor digiti minimi and tibial anterior muscles. A detailed description of the

method and data from normal subjects have been previously reported.⁴ Central conduction time to the right arm was delayed, whereas latencies to the left arm and both lower limbs were normal. On day 4 responses of the tongue to left hemispheric stimulation were absent on the right and delayed on the left. Responses of the right orbicularis oris muscle were also absent. On day 20 the central conduction time to the right arm had improved up to an abnormal side difference (3.5 ms; upper limit 2.6 ms). Responses of the tongue and orbicularis oris muscle were restored. Median nerve somatosensory evoked potentials on day 4 were within the normal range.

Our patient had the characteristic features of dysarthria-clumsy hand syndrome described by Fisher.¹ As shown by MRI the lesion affected the central two thirds of the left ventral cerebral peduncle at the location of the pyramidal tract. Functional involvement of the left corticospinal and corticobulbar tracts was confirmed by clinical findings and transcranial magnetic stimulation. Central conduction times ameliorated with clinical improvement, suggesting that clinical signs and transcranial evoked potential abnormalities were due to the same acute lesion. As sensory function (normal intraoral superficial sensation and intraoral stereognosis) was unimpaired and somatosensory evoked potentials were within normal limits, an afferent disturbance could be ruled out. Additional impairment of the cerebellum was excluded by MRI. Crossed cerebellar diaschisis disclosed by SPECT has been reported with lesions affecting the upper half of the basis pontis and is attributed to cerebropontocerebellar tract involvement.⁵ In our patient, however, HMPAO-SPECT showed symmetric tracer uptake in both cerebellar hemispheres, making such involvement highly unlikely. His clumsiness could be attributed to impairment of the corticospinal tract as shown clinically, electrophysiologically, and by imaging investigations. A disorder of the corticofacial and corticohypoglossal pathways mediating the articulatory movements of the orbicularis oris and tongue muscles therefore remains the only explanation for dysarthria.

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Transient alien hand syndrome: is this a seizure or a transient ischaemic attack?

The alien hand syndrome is usually persistent but has been reported as a paroxysmal phenomenon attributable to epilepsy. In our patient the alien hand syndrome formed the main complaint in a transient ischaemic attack.

The patient was a 62 year old right handed Portuguese man with arterial hypertension, first detected in 1980, and Leriche syndrome. He was a heavy smoker (about 60 pack/years) and used to take moderate amounts of spirits and beer. On 18 July 1994, on awakening, he felt as if his wife was touching his right forearm. Astonished and worried, he saw his own left arm and hand making automaton-like gestures, of which he was fully aware but could not consciously inhibit. His left hand would repeatedly grope for and grasp his right hip, trunk, and arm, coming up and down and picking his clothes. Sometimes, the arm tended to abduct with some forward displacement. For short periods he could control the unwanted activity by holding the left arm by his side; however, the abnormal behaviour pattern recurred every time he released the arm.

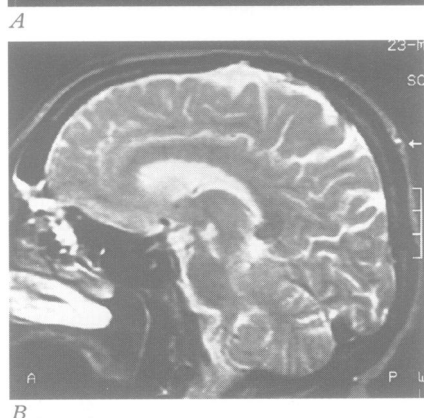
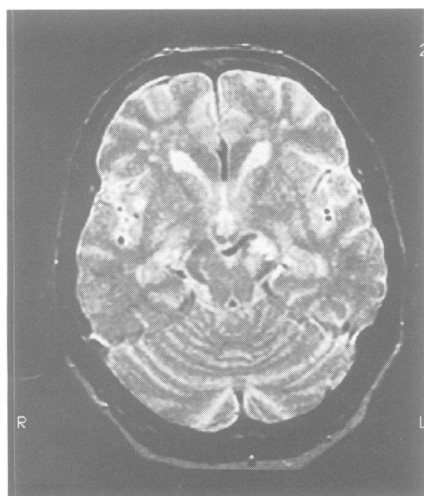
Trying to stand up, the patient noted that he could hardly move his left leg. It dangled on attempts to walk, although he could transfer some weight to it. Facial mimicry seemed to be preserved. At that time, the patient realised that his left side (including face and trunk) felt "heavy" and insensible to touch and pain.

After five to 10 minutes the automaton-like movements, especially those of the hand, decreased. Displacement of the left arm persisted to a lesser degree for a further 10 minutes. Volitional control of the arm and hand was then possible, without any residual weakness. There was parallel recovery of leg strength and face and trunk sensation. Arm and leg hypaesthesia gradually recovered in the next eight hours.

One hour after disease onset his blood pressure was 220/110 mm Hg. Brain CT showed focal hypodensities in the left internal capsule and right occipital cortex. He was discharged in the same evening, after blood pressure control and complete neurological recovery. He was advised to stop smoking and abstain from alcohol and was started on oral hypotensive medication and acetylsalicylic acid (200 mg/day).

Three weeks later full cognitive and neurological examination was normal. General examination showed reduced distal leg pulses with bilateral femoral bruits. Laboratory analyses disclosed dyslipidaemia and slight polycythaemia. Electrocardiograms and a transthoracic echocardiogram were normal. Two EEG studies (including one after sleep induction with diazepam) were normal or yielded non-specific results.

Duplex ultrasonography disclosed bilateral plaques on carotid bulbs. The right sided lesion was extensive with possible ulceration. Brain MRI confirmed old ischaemic lesions in the left internal capsule, right occipital lobe, and the left cerebellar hemisphere. Magnetic resonance angiography of the brain showed normal blood flow through both carotid and vertebral arteries and the basilar artery. Cervical MRA showed a focal signal void immediately beyond the origin of the right internal carotid artery, suggesting slight narrowing of its proximal segment.



(A) Axial section on MRI (TR 2080 ms, TE 90 ms) showing area of increased signal intensity at the central two thirds of the left cerebral peduncle, extending as a small strip into the left tegmental region at the pontomesencephalic junction. (B) Left parasagittal section.

He had no additional neurological symptoms during the next 13 months. He has stopped smoking and drinking alcohol. Arterial blood pressure and the lipid profile have returned to normal.

Leiguarda *et al*¹ reported four patients with frontal and parietal lesions in which abnormal complex movements akin to the alien hand syndrome were present in brief episodes, sometimes followed by focal and generalised seizures. Our patient was very different, having multiple risk factors for stroke, Leriche syndrome, and three previous cerebrovascular lesions. The described episode had an abrupt onset and gradual recovery, featuring some of the most typical symptoms of reversible ischaemic dysfunction.

The pattern of abnormal movements and the pronounced leg weakness in our patient is suggestive of damage to the medial frontal lobe.^{2,3} The typical syndrome after midline lesions has a different presentation, with much intermanual conflict,³ the abnormal behaviour of the affected hand always being triggered by volitional movements of the normal hand.⁴ Sensory changes have already been described in patients with alien hand syndrome.^{1,5} We speculate whether the ipsilateral hypaesthesia (thalamic?) somehow contributed to the pattern of abnormal movements of the arm, as in patients with parieto-occipital lesions.⁵

Reversible reduction of blood flow to the medial frontal cortex responsible for the formation of motor programmes (plus thalamoparietal region?) should now be regarded as another cause for the transient alien hand phenomenon.

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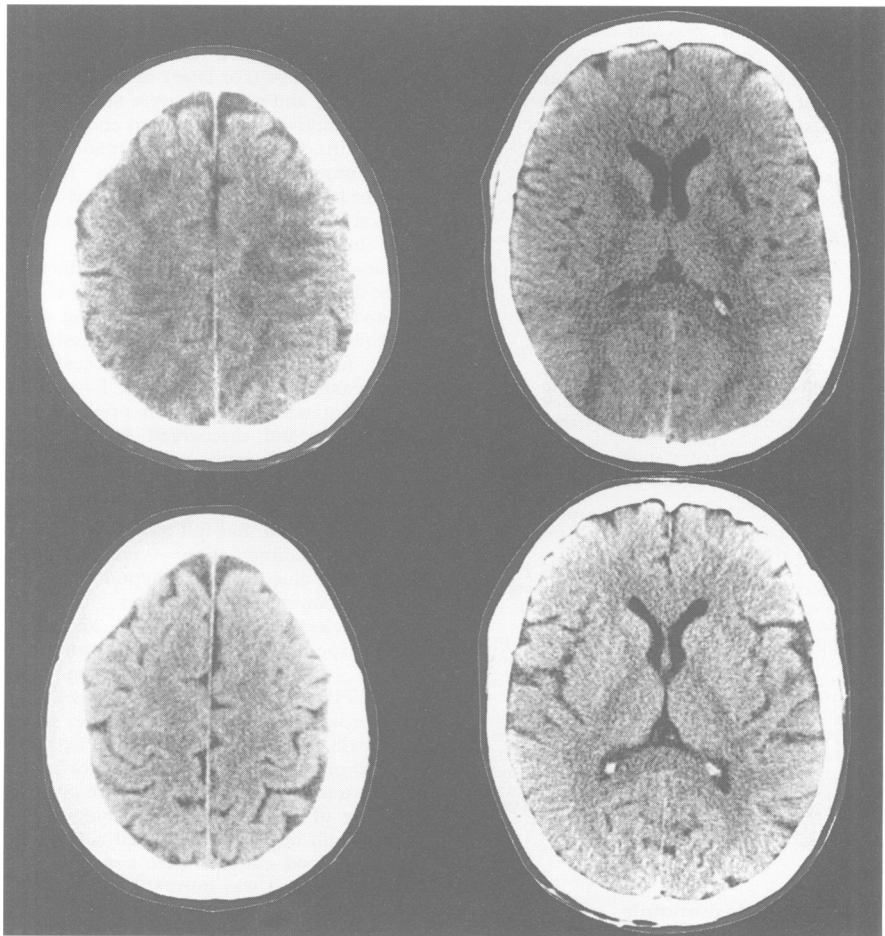
Brain water intoxication and CT hypolucencies in ovarian hyperstimulation syndrome complicating gametes intrafallopian transfer (GIFT) and tubal pregnancy

The ovarian hyperstimulation syndrome is a recognised complication of hormone treatment for assisted conception, and includes

serositis with ascites and hydrothorax in its most severe form.¹ Our patient had probable syndrome of inappropriate antidiuretic hormone secretion (SIADH) complicating ovarian hyperstimulation syndrome and ectopic (tubal) pregnancy. The patient was a 36 year old university educated bank clerk with ampullary tubal occlusion. Four weeks after gametes intrafallopian transfer (GIFT) procedures and hormonal treatment with FSH (150 UI for 10 days) and GnRH (10 000 UI) pregnancy tests were positive and the patient developed serohaematic ascites, bilateral hydrothorax, and severe orthopnoea with tachypnoea and hypotension, due to severe ovarian hyperstimulation syndrome. She underwent laparotomy; a tubal pregnancy was recognised and aborted. She was then transferred to the intensive care unit and treated with blood transfusion (red cells were 1 880 000/ml), methylprednisone, glucosate, and isotonic saline solutions. Blood pressure was maintained with dopamine. Concentrations of Na⁺ were 133-147 mEq/l for four days, during which time liquid balance was negative (-300 to -600 ml), creatinine clearance was normal, serum creatinine was 0.7 mg/dl, blood urea nitrogen 11 mg/dl, serum osmolarity 327 mmol/l, urinary osmolarity 630 mmol/l, and urinary specific gravity 1017. On the fifth day she had two tonic-clonic seizures treated with diazepam. She became confused and lethargic; Na⁺ concentrations were 134 mEq/l two hours before the seizures and 118 mEq/l one hour after. Fluid balance was 1000 ml positive. On the same day creatinine clearance

was normal, serum osmolarity was 306 mmol/l, urinary osmolarity 807 mmol/l, urinary specific gravity 1038. 30 mEq/l Na⁺ were given with demeclocycline and furosemide.

A CT performed on the same day was normal. Concentrations of Na⁺ increased to 130 mEq/l in the next 12 hours, but she became stuporous, then comatose (Glasgow coma scale 4; pupils 2 mm; presence of light reflex). An EEG showed diffuse 2-4 Hz slowing. Creatinine clearance was again normal, serum osmolarity was 285 mmol/l, urinary osmolarity 860 mmol/l, and urinary specific gravity 1039. Parenteral phenobarbitone (3 mg/kg/day) was added to current treatment, including albumin, demeclocycline, furosemide, and isotonic solution to increase Na⁺ concentration at a rate of less than 5 mEq/l/day. Concentrations of Na⁺ were 131-137 mEq/l in the next five days, liquid balance was positive (+950 ml to +600 ml) until the 10th day, when another seizure (one minute duration) occurred. Intracranial pressure was 32 ml H₂O; CSF was normal. Creatinine clearance was normal, serum osmolarity 325 mmol/l, urinary osmolarity 800 mmol/l, and urinary specific gravity 1030. A CT performed on the same day showed non-homogeneous patchy areas of hypolucency (fig 1); her liquid balance became negative (-1200 ml) and remained so for the next 10 days (-1400 ml to -300 ml). Consciousness progressively improved; the patchy hypolucencies were not present on CT on the 17th day after admission to intensive care (figure). On the 20th day she



Top row: when the patient was comatose, five days after the first seizure, patchy areas of non-homogeneous hypolucencies are present, prominently in the right dorsal prefrontal, left central parietal, right parieto-occipital, right internal, and left external capsule. Bottom row: control CT 30 days later, when the patient was fully alert; no hypolucencies are present.