movements.[17]

In considering the possible aetiologies of this patient's chorea, there is no evidence to support any of the typically considered causes of chorea in an adult, such as Huntington's disease, systemic lupus erythematosus, polycythaemia, cerebral infarcts or haematomas, Wilson's disease, recent neuroleptic ingestion or other iatrogenic, metabolic, or toxic causes. Hemiballismus, with its typically abrupt onset and unilateral features, is unlikely, especially in view of the absence of focal findings on the MRI scan. There was also no history of a previous choreiform disorder or of rheumatic heart disease. The temporal association of the transient choreiform disorder and the recent Group A beta haemolytic streptococcal pharyngitis reported in this patient suggests that this may be the first reported case of Sydenham's chorea beginning in late adulthood.

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Rapid development of basal ganglia calcification caused by anoxia

Sir: Basal ganglia calcification occurs in a number of conditions. It is not known as to how long it takes for the calcification to occur. We recently encountered a patient in whom basal ganglia calcification was demonstrated on sequential computed tomography (CT) scan after only 9 days.

A 58-year-old woman had a history of diabetes mellitus for 23 years for which she was treated with insulin. She had an operation for cataract under local anaesthesis. Two days after the operation, she developed sudden onset of dyspnoea and orthpnoea, and was referred to our department. On physical examination her temperature was 36·4° C, blood pressure was 118/64 mmHg, and respiratory rate was 40 breath/min. She was intubated, and ventilatory support was begun. Chest examination revealed dullness to percussion and diminished breath sounds at the base of the left chest. There were bronchovesicular breath sounds in the bilateral lower chest. Abdomen was normal. On neurological examination, she was stuporous, and with intermittent spontaneous movements in all her extremities. The pupils were 3 mm in diameter, equal, and reactive. Full doll's eye responses were obtained both horizontally and vertically. She had intact gag and ciliospinal reflexes. Deep tendon reflexes were brisk bilaterally with absence of ankle jerks and extensor plantar responses. Arterial blood gas levels (room air) revealed a PaO_2 of 49 mmHg, $PaCO_2$ 38 mmHg; pH 7·16 a repeat blood gas determination while the patient breathed 6l/min nasal oxygen revealed a PaO_2 54 mmHg. The white blood cell count was 6400/mm.[3] Blood sugar was 86 mg/dl. Serum Ca, P, Mg, Al-p, and parathyroid hormone levels were normal.

A chest radiograph revealed massive enlargement of the cardiac silhoutte and pulmonary oedema.

Next day, the arterial blood gas levels continued to show remarkable hypoxoemia and metabolic acidosis. She was comatose and quadriparetic, but remained responsive to noxious stimuli and could say a few words. The pupils were moitic with light responses. Intermittent spontaneous movements in the extremities persisted and plantar responses were extensor bilaterally. It was noted for the first time that if her neck was laterally rotated she developed a sudden jerking flexion of both shoulders and extension of the arms at the elbows. The movement lasted less than 1 second. The EEG was isoelectic. Later that day, she developed a decerebrate posture; other neurological signs being unchanged, one could still produce movement of the arm with neck rotation. There was occasional movement of the neck towards extension when it was passively and suddenly flexed. The movement varied from moment to moment with neck motion. During that time, she sustained two cardiopulmonary arrests with successful resuscitation. Intermittent spontaneous movement and decerebrate posture with an occasional few words of spontaneous speech persisted unchanged until 11 days after the onset, when the blood pressure progressively failed and the heart stopped. Necropsy was not performed. An initial CT scan at admission was normal. A CT scan 9 days later showed extensive basal ganglia calcification (fig).

Microscopic basal ganglia calcification was independently reported by Virchow[1] and Bamberger[2] in 1855. Fritzche[3] first described the radiographic appearance in 1935. In 1939, Eaton *et al*[4] noted the association of calcification of the basal ganglia with hypoparathyroidism, and in 1944, Sprague *et al*[5] reported its occurrence with pseudohypoparathyroidism. It has since been described in association with many pathological conditions. In our case, it is likely that the underlying cause of basal ganglia calcification was anoxia.

The vulnerability of the basal ganglia, particularly of the globus pallidum, to anoxic injury has long been recognised.[6] It is rare for the corpus striatum to suffer this type of anoxic injury without concomitant pallidal injury.

Our case suggests that basal ganglia are not only highly susceptible to anoxic injury and consequent dystrophic calcification, but also that basal ganglia calcification can

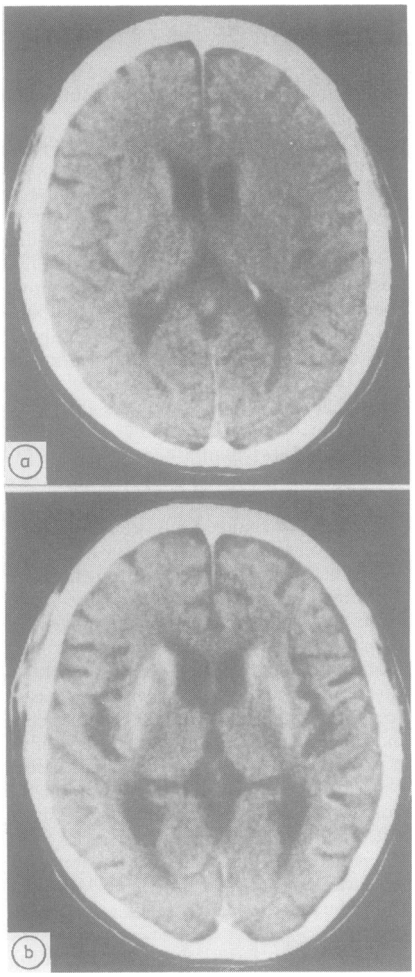

Fig (*a*) *Normal admission scan.* (*b*) *Nine days later. Extensive basal ganglia calcification.*

occur in as short a period as 9 days, much more rapidly than generally believed.

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An implant clamp for atlanto—axial fusion

Sir: Atlanto-axial arthrodesis was described by Gallie in 1939[1] and Brookes and Jenkins in 1978,[2] and atlanto-occipital fusion by Newman and Sweetnam, 1969.[3] There is an appreciable incidence of non-union and a risk of damage to the vertebral arteries and medulla or cervical cord. Mitsui in 1982[4] described a clamp to reduce and hold the atlas and the axis whilst fusion occurred. We report the use of a similar clamp in three cases. Case 1 was a man of 38 years with atlanto-axial instability following trauma, a Gallie graft failed but successful fusion followed the application of a clamp. Case 2 was a woman of 73 years with a non-union of the odontoid with spinal cord compression. Stable union was achieved. Case 3 was a girl with Down's syndrome aged 17 years who had progressive weakness in her legs. Radiographs showed anterior atlanto-axial subluxation. Reduction and fusion was achieved with some relief of the symptoms.

The device comprises two parallel horseshoe shaped clamps with hooked ends which grasp the laminae of the atlas and axis. Long series screws with lock-nuts held the clamp in position. The whole assembly was manufactured from stainless steel (BS 3531 Comp.B). The patient lies prone with the head on a headrest with or without skull traction. Through a midline incision the neural arches and spinous processes are prepared. The upper and lower hooks of the clamp are inserted seperately and reduction achieved by tightening the nuts on the screws. Cancellous bone grafts are inserted.

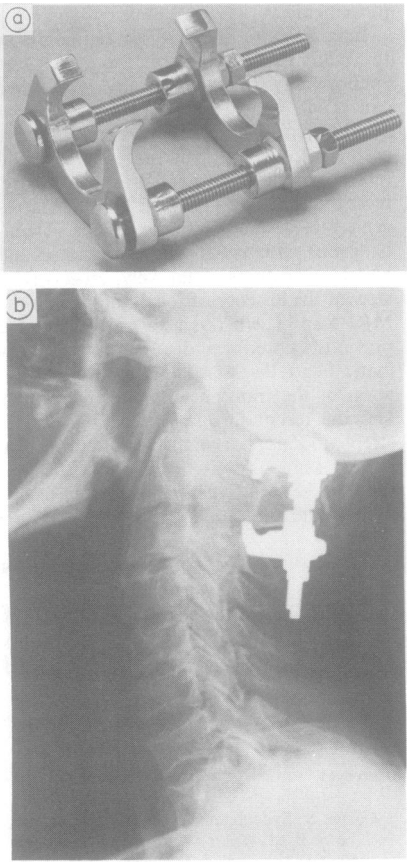

Fig *(A) The implanted clamp. (B) Lateral radiograph showing clamp.*

Clearly, the clamp is not suitable for fractures involving the posterior elements of either vertebra. We have no experience of its use in rheumatoid disease though Mitsui described its use in this situation. The clamp is effective and safe to insert and provides improved correction and stability compared to other methods of fusion.

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