patients with systemic lupus erythematosus and antiphospholipid antibodies.

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1 Levine SR, Welch KMA. Cerebrovascular ischaemia associated with lupus anticoagu-

2 Beck RW, Cleary PA, Anderson MM, et al. A randomized, controlled trial of cortico-

3 Drows AA, Fertis CA; Peris GS, Moutoumpous HM. Unusual eye manifesta-

4 Herranz MT, Rivier G, Khamashu M, Hughes GRV. Epilepsy associated with antiphospholipid antibodies in systemic lupus erythematosus [abstract]. Fifth Inter-


8 Asherson RA. Subungual splinter haemor-

9 Oppenheimer S, Hoffbrand BI. Optic neuritis and myelopathy in systemic lupus erythe-

10 Hess DC, Sheppard JC, Adams RJ. Increased immunoglobulin binding to cerebral endothelium in patients with antiphospholipid antibi-

Cocaine induced chronic tics

Since the early 1980s, cocaine misuse has rapidly become more frequent with many users chronically taking large doses of this drug. It has been increasingly associ-

Cocaine administration induces chronic tics, and the diagnosis of cocaine-induced tics is based on the presence of characteristic movement disorders that persist for more than 2 months after the use of the drug. The tics are caused by the excitatory effects of cocaine on dopaminergic neurotransmission, and they may be exacerbated by psychological and environmental factors. Unlike medication-induced tics, cocaine-induced tics are often associated with other neurological symptoms, such as dyskinesias and cognitive impairments. The long-term effects of cocaine use on the central nervous system are not fully understood, but they may include permanent neurodegeneration and brain atrophy. The prevalence of cocaine-induced tics is difficult to estimate, but it is likely to be high among individuals who use cocaine chronically.

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aggravate pre-existent movement disorders such as Tourette's syndrome and chorea, or provoke tics in patients with attention deficit hyperactivity disorders. These amphetamine mediated phenomena are similar to those induced by cocaine except for the stereotypic dyskinesia. In regard to the NAergic system, there were no biochemical abnormalities in the cortex and basal ganglia. Clonidine, an α2-adrenergic agonist, reduced tics, however, through a direct effect or indirectly by modulating mesolimbic neurons containing dopamine. CSF MHPG in our case was paradoxically high, showing a complex imbalance between neurotransmitter systems that is only partially detected by CSF metabolites. Some findings (cited by Lang) reported variable implications of opioid and GABAergic systems in the pathogenesis of Tourette's syndrome. Opiate antagonists may lessen tics, whereas withdrawal of chronic opiate treatment may worsen this condition. There are some responses to benzodiazepines (clonazepam). Implications of other neurotransmitter systems are cited in the literature but these findings are not consistent. Further controlled studies using SPECT or PET, CSF biogenic amines, possibly concentrations of cocaine in blood or CSF, and neuropsychological testing results are needed to better identify deficiencies after cocaine misuse.

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A 65 year old ex-policeman was admitted with a three day history of falling to his right whenever he attempted to sit, stand, or walk. He was normal until a day before the onset of this neurological event when he had complained of a mild generalised throbbing headache. The next morning he noticed difficulty in getting out of bed and needed support to even sit erect. He was unable to stand or walk without support. Whenever he attempted to do so he leaned heavily to his right and fell over. He had never experienced such an event in the past. There was no history of drugs that could produce extrapyramidal syndrome or ataxia. He gave no history of head trauma. He was not a diabetic and was not hypertensive.

On admission his blood pressure was 140/80 mmHg and his heart rate was 90 beats/min. The cardiovascular system was normal. There were no external injuries. He was conscious and well orientated to his surroundings. Speech and memory were normal. His pupils were normal in size and reacted equally well to light. He had a mild drift of the outstretched right arm. Power of the other limbs was normal. Muscle tone was normal in all limbs. The deep tendon reflexes were hyperactive on the left side. The Babinski's reflex was flexor on both sides.

His tendency to fall to his right was obvious and striking. He was unable to sit, stand, or walk without support. When asked to rise from a recumbent posture he would grasple at the cot rails with his left hand and struggle to do so. When supported he could sit erect for a few seconds but gradu-
ality leaned to his right and diagonally back-
wards. When helped to stand erect, he would fall in the same direction. Supported walking was possible for only a few steps and was terminated by the falling attack. Another striking feature was that he made no postural adjustments to overcome such falls and barely expressed concern about them (figure; left).

He had no evidence of hemianesthesia or visual field cut. Bedside tests for sensory neglect were negative. His right arm was underused in motor tasks. This was out of proportion to the mild weakness of that limb, indicating the presence of motor neglect as well as the pyramidal lesion. He had no features of cerebellar, vestibular, or peripheral nerve disorder.

A clinical diagnosis of “ease of falling” syndrome was made. A plain and contrast enhanced CT of the head surprisingly showed a large subdural haematoma in the left frontoparietal region (figure; right). The haematoma was isodense with the cortex and compressed the ipsilateral subcortical structures and lateral ventricle and produced a shift of the midline structures to the opposite side. There was no evidence of damage to the underlying brain. Chert radio-

graph, BCG, and carotid Doppler studies were normal. Blood chemistry was normal. Serological tests for syphilis were negative.

A burrhole was made on the left side of the skull and 250 ml of altered blood was evacuated under local anaesthesia. The result was dramatic. The patient could sit erect without support in the immediate postoperative hours. Detailed evaluation was carried out over the next 24 hours and photographically documented. He could sit erect, stand, and walk by himself without any tendency to fall. The outstretched right arm showed no drift. The pyramidal signs and motor neglect disappeared.

The “ease of falling” syndrome has become well characterised through the studies of Masdeu and Gorelick, Awerbuch et al., and Labadie et al. Isolated cases with similar features had already been reported by Fisher and Cole in 1965 and by Fisher in 1979 and 1982. As noted by all these authors the falls are a contralateral slow tilting motion either laterally or diagonally backwards. The patient shows lack of awareness and does not make postural adjustments to avoid the fall. Criteria require that the patient should exhibit such falling events in the absence of significant hemiparesis, hemianaesthesia, cerebellar ataxia, vestibular dysfunction, proprioceptive loss, and peripheral nerve disorder. Our patient qualifies for the diagnosis of this syndrome.

All previously reported cases had an intracerebral lesion affecting either the putamen, pallidium, or the thalamus, Lacunar infarcts and haemorrhages are the only lesions that have produced this acute

“Ease of falling” syndrome associated with subdural haematoma

The “ease of falling” syndrome refers to acute onset contralateral postural deficits secondary to acute lesions of the unilateral basal ganglia. Previously reported cases had intracerebral lesions affecting the basal ganglia. The present case is unique as the patient developed the syndrome secondary to an extracerebral lesion in the form of a subdural haematoma.

(left) The patient falling to his right and diagonally backwards. Note the lack of concern about the fall; (right) CT showing a large left frontoparietal subdural haematoma.