Case report: A 34-year-old married male presented to our out-patient department with 2 years history of episodic loss of vision and a one-month history of disturbed behaviour and restlessness. There was no other contributory family or past history. For 2 years he had been having episodic loss of vision, for which he had been on steroids with poor compliance. He had been off steroids for six months prior to consultation. For one month before consultation he had been singing, over-confident, restless, talking excessively, and not sleeping adequately. He was brought in because he was unmanageable at home. He was in hospital for a period of 45 days. Physical examination revealed only light perception in the right eye, with vitreous haemmorhages. With his left eye, vision was 6/12 and fundal examination revealed peripheral gliosis and haemmorhages. A right lateral rectus palsy was present.

Mental status examination revealed increased psychomotor activity, pressure of speech, grandiose delusions of ability and identity, impaired social judgement, intact cognitive functions, and absent insight. He was treated symptomatically with phenothiazines, and the episode remitted about 3 weeks after discharge. Subsequently, he had another similar episode after one year which remitted after 5 weeks; this also required admission.

Routine investigations were within nomal limits: ESR = 24; STS non-reactive; LE cells negative; Rh factor negative; CSF cells nil; sugar 50 mg%; protein 28 mg%; globulins negative. ENMG showed motor nerve involvement in the left lower limb with myelin axon. EEG was within normal limits, and CT scan was normal. Muscle biopsy taken from the gastrocnemius was normal. In the biopsy of the sural nerve there was a minimal increase in the peri and endoneural connective tissue, with a marginal fall-out of myelinated tubules on the K. Pal stain. This was suggestive of an ischemic process.

The aetiology of Eale's disease is not known, although tuberculin hypersensitivity is most widely agreed upon (Dastor & Udani, 1966). Haarr, (1964) has pointed out that the histological appearances of vasculitis resemble those of erythema nodosum.

The diagnostic possibilities include reactive excitement, which is unlikely as the patient did not satisfy criteria for diagnosis of reactive psychosis. The two illnesses could have occurred due to a rare unreported association. The third possibility is of secondary mania (Krauthamer & Klerman, 1978) with Eale's disease. The possibility of steroid psychosis seems unlikely because of the gap of five months between cessation of steroid intake and onset of psychosis. It would be interesting in this case to regard vasculitis as the cause of all three defects; namely, in the encephalon, the peripheral motor nerve, and in the eye.

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Fluvoxamine and Hepatic Function

SIR: Fluvoxamine maleate is a recently introduced antidepressant, marketed as a specific 5-HT reuptake inhibitor (*Drug and Therapeutics Bulletin*, 1988). Caution in prescribing fluvoxamine is recommended for patients with renal or hepatic impairment. I would like to draw your attention to a possible serious side-effect of fluvoxamine therapy.

Case report: A 57-year-old office worker was admitted to hospital for assessment of low mood and suicidal ideation. He had become increasingly preoccupied and withdrawn over the previous three months and had had to stop work because of anxiety symptoms. There was no significant past medical or psychiatric history. There was no history of alcohol abuse from the patient, and this was confirmed by his wife and children. On admission his physical examination and initial investigations, including ECG and chest X-ray, were normal (mean cell volume 90 fl and gamma-glutaryl transferase 50 U/litre). He was started on fluvoxamine (100 mg b.d.). He was maintained on this dose for 18 weeks. There was no significant improvement in his mood, and he remained anxious and preoccupied. A repeat blood screen after 3 weeks of therapy showed a γ-GT of 176 U/litre. When this was repeated the γ-GT had remained elevated at 167 U/litre. The fluvoxamine was discontinued. Abdominal examination and ultrasound confirmed an enlarged liver with echo characteristics of fatty change. After stopping fluvoxamine therapy his liver function tests gradually returned to normal. Five weeks after ceasing fluvoxamine his γ-GT was back to 59 U/litre.

It seems most probable that fluvoxamine was responsible for the deterioration in hepatic function, with the possibility that it was responsible for the observed fatty change. This is the first such reported

case with this possible side-effect (Committee on the Safety of Medicines, Private Communication).

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DRUG AND THERAPEUTICS BULLETIN (1988) **26**, No. 3.

A Short Sharp Course of Steroids for the Patient with Depression

SIR: Since I had become afflicted with late onset asthma, a few weeks ago my GP gave me a short sharp course of steroids which had a quite remarkable effect. The combination of easier breathing and the euphoria was a most pleasant experience, and the uplift became apparent in 24 hours. Looking back, one can see that it was the stimulating effect of the iproniazid group of drugs used in the treatment of tuberculosis that drew attention to these agents as possible antidepressant medicaments. The snag about these drugs is that there is a tiresome latent period before any noticeable beneficial uplift occurs. If I were still in practice, I would like to try the effect of giving the depressed patient a short sharp course of steroids alongside the antidepressant agents, to see if this would cut short that period of waiting. Has this idea ever been tried? There is a precident for this humane kind of therapy: a short-acting barbiturate is given as a preliminary anaesthetic to cut out the unpleasant feelings that occur with the slow induction to oblivion of longer-acting anaesthetics. I am old enough to have had the experience of both methods of induction, and I know which one I prefer.

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A HUNDRED YEARS AGO

The Opium Habit

The thoughtful and epigrammatic lecture upon Coleridge by Mr. Leslie Stephen, delivered recently at the Royal Institution, once more reopens the vexed question of the influence of opium upon imaginative work. His opinion may be summed up in the words "the pity of it", since he considers that "opium ruined the power of will, never very strong, and any capacity he may have had – and his versatility was perhaps incompatible with any great capacity – for concentration on a great task." In another place he speaks of Coleridge's life affording a proof that "opium-eating is inconsistent with certain homely duties." While admitting these propositions in the main, Mr Leslie Stephen appears to have arrived at the true estimate of the author when he says that he "cannot think that Coleridge ever worked with his mind clear," since so much of the wealth of imagery and brilliance of colouring seem to have had their origin in what was formerly regarded as the directly stimulant action of opium. This action, according to Schmiedeberg, should be considered rather as a disturbance and alteration of the equilibrium of the separate functions of the brain, the area for ideation remaining intact, while sensory stimuli are beginning to lose their effect. Schmiedeberg holds that the area for ideation has thus less stimulation and guidance from without, and accordingly springs into action on its own account. While most earnestly deprecating the employment of opium as a "mental stimulant" by those who live by literature, we none the less feel that between the lines of those poems by which Coleridge will be best remembered the influence of the opium habit can be traced in its earliest, most fascinating, and therefore its most dangerous stages. The ultimate collapse which results from its frequent employment is none the less certain, though in exceptional cases it may be delayed.

Reference

The Lancet, April 7 1888, 692.