including those that could have been transmitted by the bite gave negative results. These investigations included a complement fixation test for Q fever, malarial fluorescent antibody tests, schistosomal and filarial complement fixation tests, the rapid plasma reagin test and *Treponema pallidum* haemagglutination test, and a search of blood for trypanosomes. Serum was then examined for an extended range of antibodies to West African viruses (ETWB). Antibodies against Le Dantec virus were found in significant titre (1/16). No other viral antibodies were detected.

Comment

The question of a causal relationship between the insect bite, the infection, and the subsequent Parkinsonism must remain problematic, but there can be little doubt that the febrile illness after the bite occurred when the Le Dantec virus infection was acquired, for the patient had never been to West Africa and had had no other incident so far as he could recall during which he had been bitten while on a ship that had been to West Africa. Many viruses can cause encephalitis, and involvement of the basal ganglia is well recognised in certain viral infections, particularly those with Japanese B, St Louis, and Murray Valley viruses. It is particularly notable, however, that in the *International Catalogue of Arboviruses*¹ Le Dantec virus, on being inoculated into suckling mice, is reported to have produced "severe lesions of encephalitis with important destruction of the neurones of the cerebrum (cortex, thalamus, hypothalamus) less acute in the spinal cord. Perivascular cuffing with mononuclear cells. No myositis." In view of this there was probably a causal relationship between the bite with the infection it conveyed and the subsequent development of Parkinsonism.

The case is notable in focusing attention on the need for vigilance in containing viral infections that could be transmitted by insects carried on transport arriving from Africa. Secondly, it provides suggestive information that Le Dantec virus, like certain other viruses, is capable of damaging the basal ganglia of the brain.

¹ *International Catalogue of Arboviruses*, ed T O Berge, 2nd edn, p 426. US Department of Health, Education and Welfare, Washington, 1975.

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Cardiac arrhythmia after mushroom ingestion

Mushroom poisoning is relatively uncommon in Britain. Ingestion of the bulb agarics with their lethal "amanita toxins" account for most of the reported deaths but serious reactions may occur with other species, which contain a variety of toxins.¹ We describe here the reaction that may follow ingestion of the edible black-spored mushroom *Coprinus atramentarius* (inky caps) and alcohol.

Case report

A 37-year-old man became ill after eating wild mushrooms which he and his wife had picked, fried, and eaten on the same day. Two hours after the meal the patient drank three pints of beer and almost immediately felt unwell. His face flushed, a blotchy red rash appeared over the upper half of his body, and his hands and face swelled. He became breathless, sweated profusely, and was nauseated. During the three hours in which the reaction was severe he vomited six times.

The medical history was unremarkable and he had never complained of palpitations. He had not taken any drugs. There was no family history of heart disease or thyrotoxicosis.

On admission his face was flushed and there was peripheral vasodilatation, but the swelling of his face and hands had settled. His temperature was

37.5°C. He had an irregular pulse with a tachycardia of 120 beats/min. Blood pressure was 110/80 mm Hg. The heart sounds were normal with no audible cardiac murmurs. The remainder of the examination was normal. The admission electrocardiogram (ECG) showed sinus tachycardia with frequent supraventricular ectopic beats.

Twelve hours later the patient felt better but he was in atrial fibrillation with an apex beat of 150 beats/min. His ECG was monitored and he remained in atrial fibrillation for 60 hours before spontaneously reverting to sinus rhythm. The arrhythmias did not recur.

Full blood count; chest radiographs; and serum electrolyte, cardiac enzyme, and protein-bound iodine concentrations were normal. Serial ECGs confirmed the arrhythmias but were otherwise non-specific.

The patient's wife ate the same mushrooms without drinking alcohol and remained well. The mushrooms were identified as *Coprinus atramentarius*.

Comment

Toxic reactions after ingesting *Coprinus atramentarius* are uncommon. Features of the reaction which have been reported include flushing, swelling and rash of the face and hands, tachycardia, hypotension, dyspnoea, nausea, vomiting, and shock. While these effects are unpleasant, recovery, even from severe reactions, usually occurs spontaneously within 24 hours without complications.²

The reaction occurs only when alcohol is taken with or up to 24 hours after ingesting cooked mushrooms.¹ Its intensity is directly related to the quantity of mushrooms and alcohol consumed and the time interval between the two events.

The effects are similar to those that occur when a patient taking disulfiram drinks alcohol. Simandl and Franc³ have reported isolating tetraethylthiuramdisulfide (disulfiram) from *Coprinus atramentarius*, although this has not been confirmed.

Markham has observed reversible ECG changes in most patients during controlled reactions,⁴ while cardiac arrhythmias and myocardial infarctions have been described with toxic interactions⁵ between disulfiram and alcohol.

Because of the similarity between the two reactions cardiac dysfunction may be expected to occur after the alcohol-mushroom reaction, but it has not been reported previously. In our patient the arrhythmias occurred during and for a short time after the reaction and were unlikely to have been coincidental. In a healthy man the effects were self-limiting but in patients with cardiovascular disease the consequences may be more serious. If several hours lapse between the ingestion of the mushrooms and consumption of alcohol the connection between them may not be recognised and a possible cause of cardiac abnormality may be missed.

We thank Mr Thomas Boam, chief technician, Department of Botany, Sheffield University, for his advice.

¹ Buck, R W, *New England Journal of Medicine*, 1961, **265**, 681.

² Reynolds, W A, and Lowe, F M, *New England Journal of Medicine*, 1965, **272**, 630.

³ Simandl, J, and Franc, J, *Chemike Listy*, 1956, **50**, 1862.

⁴ Markham, J D, and Hoff, E C, *Journal of the American Medical Association*, 1953, **152**, 1597.

⁵ Macklin, E A, et al, *New England Journal of Medicine*, 1951, **146**, 1377.

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Male dyspareunia due to short frenulum: an indication for adult circumcision

The main indications for adult circumcision are a prepuce that cannot be freely retracted behind the glans, fibrosis of the prepuce orifice, recurrent dermatitis of the prepuce, persistent or recurrent balanoposthitis, paraphimosis, or suspected carcinoma of the penis. Patients are also referred because of inadequate hygiene and because

of pain. The latter is often caused by psychosexual problems with no obvious organic cause. There is, however, a group of patients presenting with dyspareunia who have a well-defined lesion—a short frenulum that becomes painfully tight on erection and that may be cured by circumcision.

The existence of a short frenulum is noted in few urological textbooks and then usually in relation to prepuce adhesions in the child.¹ The normal frenulum is a mucous membrane on the ventral surface of the glans penis, through which runs the frenular artery. A short frenulum may apparently be congenital, when it is of normal thickness, or secondary to recurrent infection or trauma, when it can be felt in the flaccid state as a thickened band. When the penis is erect the short frenulum restricts the prepuce and causes downward curvation of the glans.² During intercourse this means that there is an element of chordee and the abnormal position of the glans penis causes pain. Tension in the short frenulum itself is also painful and the band may be torn and then heal by fibrosis, thus aggravating the condition.

Case histories

A retrospective survey of adult circumcision performed at the Churchill Hospital under the care of three general surgeons in 1975 and 1976 showed that 48 had been performed (see table), with an age range of 16 to 68 years (mean 29). The commonest indication, not unexpectedly, was phimosis (25); but the problem posed by a short frenulum was found to be the next most common (10). The patients in the latter group were contacted and asked to fill in a questionnaire stating the reasons for their first seeking advice and the result of their operation. Nine replied stating that they had had no further problems, specifically noting that they had had no pain on intercourse since circumcision. The one who did not reply has not sought further advice about dyspareunia from his general practitioner.

Indications for circumcision in 48 patients 1975-6 and number with pain on intercourse

	Hygiene	Para-phimosis	Phimosis	Phimosis and balanitis	Balanitis alone	Short frenulum
No of cases ..	3	2	25	6	2	10
No with pain on intercourse ..			10	3		10

Comment

As may be seen from the table, male dyspareunia is not uncommon and attention has been increasingly drawn to it as a cause of difficulty with sexual relationships, notably by Masters and Johnson.³ In their chapter on male dyspareunia they cite phimosis and lack of personal hygiene as the main contributing causes. They also draw attention to the abnormal sensitivity of the glans penis to pH, for example, and emphasise the well-recognised problems associated with hypospadias and chordee. They do not mention the problem of a short frenulum.

Short frenulum was an important reason for male dyspareunia in this series. Circumcision apparently completely alleviated symptoms, with a correspondingly grateful patient. Complete or formal circumcision is necessary, however, for simple division or attempts at Z-plasty on the frenulum may leave residual scarring and therefore sufficient tightness to cause further symptoms. Short frenulum should be sought as a cause of dyspareunia in patients who do not have phimosis or questionable hygiene and who may erroneously be labelled as having psychosexual problems.

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¹ Campbell, M F, and Harrison, J H, *Urology*, 3rd edn, p 1536. London, W B Saunders, 1970.

² Campbell, M F, and Harrison, J H, *Urology*, 3rd edn, p 1835. London, W B Saunders, 1970.

³ Masters, W H, and Johnson, V E, *Human Sexual Inadequacy*, p 289. London, J and A Churchill Ltd, 1970.

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Dry beriberi in a slimmer

In Europe beriberi is usually found among alcoholics, in whom it occurs mainly in the wet form.¹ We report here severe dry beriberi in a schoolgirl who reduced her weight by 44 kg in four months. To our knowledge slimmer's beriberi has not been reported before.

Case report

A 17-year-old schoolgirl with no personal or family history of neurological disease had been suffering from obesity for many years. After reaching 104 kg (at a height of 167 cm) in August 1976 she firmly decided to start a slimming diet planned by herself. In the beginning it consisted of one fruit (orange, mandarine, pear, or banana) and one egg every day. After a month she abandoned the eggs. Besides fasting she also restricted drinking. The strict diet was effective: after four months her weight had fallen to 60 kg. Having reached this goal, she started to take normal meals again from 10 December 1976. During the last days of fasting she had noticed slight weakness and numbness in the extremities. The symptoms worsened over a few days and culminated after a week in total paralysis of all four limbs. She was admitted to Oulu University Central Hospital on 13 December 1976, where she was bedridden.

She was found to have a severe distally aggravating polyneuropathy. The radial and peroneal nerves were the worst affected: she had wrist drop and foot drop. Tendon reflexes were absent in her feet. Slight one-sided facial paralysis was found, but this disappeared in a few days. Sensory neuropathy manifested itself in the form of a weakened posture and vibration sensibility in the distal parts of the extremities. Her mental state and functions were unaffected, and no other abnormal physical signs were found. Total amenorrhoea was an additional complication, but the cause was obscure because the patient also took oral contraceptives.

Various laboratory tests were performed to exclude known causes of polyneuropathy, but only thiamine deficiency could be found. The only other abnormality was slightly raised aspartate and alanine transaminase

Serum vitamin concentrations

Vitamin	Before treatment	During treatment	Normal values
Thiamine ($\mu\text{g/ml}$)	0.006	0.027	0.01*
Riboflavin ($\mu\text{g/ml}$)	0.035	0.07	0.06*
Pyridoxine ($\mu\text{g/ml}$)	0.02	0.08	0.02*
Vitamin B ₁₂ (pmol/l)	425		100-500
Vitamin A ($\mu\text{mol/l}$)	0.8	1.3	0.4-1.4
Folic acid (nmol/l)	8.6-5.8		6-22

*Mean value of 15 other hospital patients.

Conversion: SI to traditional units—Vitamin B₁₂: 1 pmol/l $\approx$ 0.14 ng/100 ml. Vitamin A: 1 $\mu\text{mol/l}$ $\approx$ 28 $\mu\text{g/100 ml}$. Folic acid: 1 nmol/l $\approx$ 44 ng/100 ml.

concentrations, which became normal eight weeks later. The cerebrospinal fluid was normal. No disturbances were seen on malabsorption tests, and serum pyruvate concentrations were normal two weeks after admission. Beriberi had been diagnosed clinically on admission and was confirmed by serum vitamin measurements (see table).

No gynaecological abnormalities were noticed, and excretion of female sex hormones was normal. Neuro-ophthalmological examinations showed no pathological signs (which would have been expected had the patient had Strachan's syndrome).² Psychological investigations showed average cognitive capacity without abnormalities of personality. An electroencephalogram (EEG) and electroretinogram (ERG) were normal, and measurement of visual evoked potentials (VEP) and quantitative EEG analysis gave normal results. Electroneuromyography showed considerable axonal degeneration in both peripheral motor and sensory nerves, and pathological delay was found in the partially affected facial nerve.

The patient was treated with multivitamin preparations and intensive physiotherapy. Both the motor and sensory symptoms responded to treatment but recovery was slow. In 10 weeks she could walk with crutches. After five months peroneal nerve functions were only slightly repaired and she still needed a walking stick. Lower extremity tendon reflexes remained inelicitable. The patient could not write until after three months, but after five months the functions of her hands were normal. Sensory disturbances appeared more persistent and were still pronounced after five months.

After five months EEG and ERG-VEP studies were normal. Electroneuromyography showed slight progress toward recovery in the motor nerves, but the sensory nerve functions had not improved at all.

Comment

The history suggested a nutritional polyneuropathy in this patient, and her clinical state, with distally aggravating polyneuropathy, agreed well with a diagnosis of dry beriberi. The diagnosis was confirmed by the results of serum vitamin measurements and by excluding other factors known to cause polyneuropathy. We did not