related complex present diagnostic difficulties. The lymphadenopathy in this group may result from a treatable infection or neoplasm. To our knowledge these are the first reported cases in which ultrasound guided percutaneous biopsy has been done in such patients. We performed Tru-Cut biopsy rather than fine needle aspiration because this technique is capable of yielding a tissue specific diagnosis so that lymphomas may be characterised by their nodal architecture and immunohistological features.

Percutaneous ultrasound guided biopsy is a simple method of obtaining tissue in patients with symptoms resulting from infection with HIV. It may remove the need for laparotomy, which has many disadvantages, including the use of considerable resources to provide appropriate precautions for the larger number of staff at risk of contact with infected blood. With this technique a skilled operator can achieve millimetric precision in placing the biopsy needle. In our experience of over 200 abdominal biopsies it is a safe technique even when the needle crosses bowel or includes bowel wall in the biopsy specimen (unpublished data). Furthermore, it is cheap and puts only the operator at risk of needlestick injury.

We thank Dr S G Semple for referring patients to us.

1 Lindgren PG. Percutaneous needle biopsy. A new technique. *Acta Radiol (Diagn)* 1982;**23**:653-6.
2 Abrams DI. AIDS-related lymphadenopathy: the role of biopsy. *J Clin Oncol* 1986;**4**:126-7.
3 Godley MJ. AIDS and lymphadenopathy. *Br J Surg* 1986;**73**:170-1.

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Effect of stanozolol on itching in primary biliary cirrhosis

In primary biliary cirrhosis itching can be debilitating and sometimes responds poorly to treatment. Norethandrolone, which controls itching associated with cholestasis, though it worsens jaundice, is no longer available in Great Britain. Stanozolol, a C_{17} substituted derivative of testosterone, causes cholestasis[1] and is not recommended in patients with pre-existing liver disease. We nevertheless gave stanozolol to five patients with primary biliary cirrhosis to see whether it could be used to control itching.

Case reports

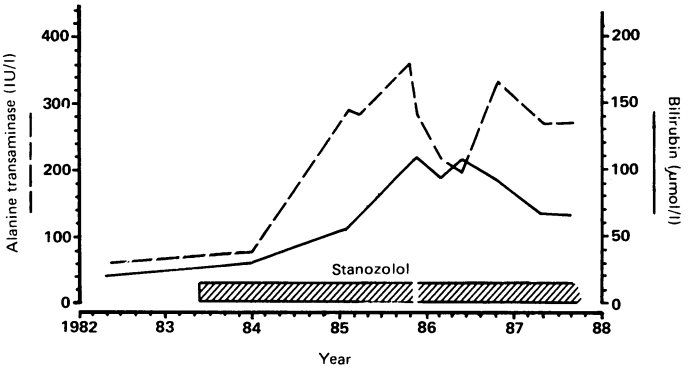
Case 1—A 57 year old woman had generalised itching that did not respond to chlorpheniramine and cholestyramine. Her liver disease was stable, but the pruritus had made her depressed. Treatment with stanozolol 5 mg daily was started, and within the first week the itching resolved completely. It returned nine days after stanozolol was stopped because the bilirubin concentration was high but then resolved when she resumed stanozolol. After two years the bilirubin concentrations had fallen to 60 μmol/l. Itching remained completely controlled. The peak concentration of bilirubin during treatment was 103 μmol/l (normal range 0-17 μmol/l).

Case 2—A 53 year old woman presented with itching, malaise, and pigmentation of her skin. Results of tests of hepatic function remained stable, but itching did not respond to cholestyramine. Desperate for relief, she started stanozolol 5 mg daily. Symptoms improved dramatically, but results of biochemical tests deteriorated: bilirubin concentration rose from 25 to 122 μmol/l. Stanozolol was given on alternate days, but this was inadequate to control the pruritus. She restarted daily treatment and although jaundiced was free from itching.

Case 3—A 52 year old woman had pruritus that was uncontrolled by cholestyramine and started taking stanozolol 5 mg daily. Itching improved, but six weeks later she had pain in the right hypochondrium and rapidly increasing jaundice (bilirubin concentration 300 μmol/l). An endoscopic retrograde cholangiogram showed a normal biliary system. Stanozolol was stopped. The bilirubin concentration fell to values seen before she started taking stanozolol, but after 18 months' gradual deterioration she died of hepatic decompensation.

Case 4—This 78 year old woman, whose pruritus caused insomnia, developed diarrhoea while taking cholestyramine. She started stanozolol 5 mg daily with dramatic effect, but her jaundice worsened (bilirubin concentration rose from 39 to 150 μmol/l) and the drug was stopped. Two months later, when the bilirubin concentration had fallen to 68 μmol/l, her itching was again unbearable. Stanozolol on alternate days was insufficient, and the dose was increased to 5 mg daily despite the probability that her jaundice would worsen.

Case 5—A 47 year old woman suffered severe skin and vaginal itching despite treatment with antihistamines, cholestyramine, ultraviolet irradiation, and antifungal agents. Her general health and results of biochemical tests were stable. Close to suicide, she agreed to try stanozolol 5 mg daily. Her pruritus resolved completely within one week. Treatment on alternate days subsequently controlled her itching. The serum bilirubin concentration had risen to 46 μmol/l (from 19 μmol/l) after one year.



Serum bilirubin concentration and alanine transaminase activity before, during, and after treatment with stanozolol in a patient with primary biliary cirrhosis (case 1).

Comment

In most patients with primary biliary cirrhosis itching is mild and tolerable, occasionally requiring antihistamines or cholestyramine. In some patients, however, it can be severely debilitating both physically and psychologically: we treated only these patients with stanozolol. All showed dramatic resolution of pruritus within days of starting stanozolol, but one stopped the drug because of increasing jaundice.

Stanozolol increased serum bilirubin concentrations about threefold (figure), and there was a parallel rise of alanine transaminase activity. As liver biopsies were not repeated we do not know whether these increases represented histological deterioration. There was, however, no associated clinical deterioration, and serum albumin concentrations remained stable, suggesting that hepatic synthesis of albumin was unimpaired. Androgenic side effects were not noted. We commend the use of stanozolol in patients with debilitating itching associated with cholestasis for whom norethandrolone would previously have been prescribed. Before it is recommended for more general use, however, controlled study is required.

1 Everly RS, Triger DR, Milnes JP, Low-Beer TS, Williams R. Severe cholestasis associated with stanozolol. *Br Med J* 1987;**294**:612-3.

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Zinc deficiency in children with dyslexia: concentrations of zinc and other minerals in sweat and hair

Developmental dyslexia is estimated to affect about 10% of 10 year olds. Studies of mineral concentrations in hair have found that children with impaired learning and those with behaviour disorders tend to have higher concentrations of toxic metals, especially copper, lead, and cadmium. Although animal studies have shown that zinc is essential for brain development and function,[1] controversy has arisen about the extent and severity of zinc deficiency in clinical practice because a simple, reliable, and sensitive test is not yet in routine use. Sweat zinc concentrations are decreased in zinc deficient states. They may be a more useful guide to clinical zinc deficiency than either hair or serum concentrations.[2] Therefore we compared concentrations of minerals in sweat and hair in children with dyslexia and a control group.

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Subjects, methods, and results

Children aged 6 to 14 were recruited from those attending remedial classes at the Dyslexia Institute and the Hornsby Learning Centre. They were paired with their non-dyslexic schoolfriends, who were matched for age, sex, social class, and environment and had no obvious allergies, illnesses, or behaviour disorders. Seven pairs of girls and 19 pairs of boys volunteered. Eighteen of the 26 dyslexic children had a family history of dyslexia.

Sweat free of cells was collected from the skin of the children's backs on 7×4 cm absorbent patches backed with polythene during 60 minutes of passive sweating. Particular care was taken to avoid any contamination.[3] Hairs up to 4 cm long were cut from the occipitonuchal region as close to the scalp as possible and washed.[4] Ten trace minerals in sweat and 18 in hair were analysed with a Pye Unicam PU 9000 atomic absorption spectrophotometer.

Zinc concentration in sweat was significantly lower in the children with dyslexia, being 66% that in the control children (table). In 25 of the 26 pairs the child with dyslexia had a lower sweat zinc concentration than the control. In the remaining pair the control child had the lower concentrations of zinc in both sweat (4·4 μmol/l) and hair (1·4 mmol/kg). He also had the highest concentration of lead in hair in the control group (65·2 μmol/kg), while his zinc deficient

Concentrations of minerals in sweat and hair from 26 dyslexic children and matched controls

	Study group	Median (range)	Dyslexic/control: Geometric mean	Dyslexic/control: 95% Confidence interval†	p Value (Wilcoxon matched pair test)
Concentrations in sweat					
Zinc (μmol/l)	Dyslexia	5·4 (3·2-7·8)	0·66	0·59 to 0·75	<0·0001***
	Control	8·0 (4·4-10·3)			
Chromium (nmol/l)	Dyslexia	74·0 (23·1-88·5)	0·88	0·81 to 0·96	0·001**
	Control	78·8 (34·6-100·0)			
Copper (μmol/l)	Dyslexia	10·8 (6·7-17·7)	1·10	1·03 to 1·18	0·014*
	Control	9·5 (7·5-17·1)			
Nickel (μmol/l)	Dyslexia	0·98 (0·65-3·17)	1·10	1·00 to 1·22	0·069
	Control	0·89 (0·68-3·39)			
Sodium (nmol/l)	Dyslexia	21·0 (19·0-24·0)	1·04	1·00 to 1·09	0·077
	Control	20·5 (16·0-24·0)			
Manganese (nmol/l)	Dyslexia	54·6 (38·3-78·3)	0·95	0·88 to 1·03	0·216
	Control	58·3 (47·4-76·5)			
Magnesium (nmol/l)	Dyslexia	220 (170-270)	1·00	0·95 to 1·04	0·670
	Control	210 (170-270)			
Lead (mmol/l)	Dyslexia	106 (68-179)	1·17	1·05 to 1·30	0·017*
	Control	82 (53-203)			
Cadmium (nmol/l)	Dyslexia	10·7 (8·0-20·5)	1·15	1·04 to 1·28	0·032*
	Control	9·8 (6·2-16·9)			
Aluminium (mmol/l)	Dyslexia	370 (259-593)	1·02	0·91 to 1·14	1·000
	Control	370 (222-741)			
Concentrations in hair					
Zinc (mmol/kg)	Dyslexia	2·2 (1·2-3·8)	0·99	0·87 to 1·14	0·879
	Control	2·4 (1·4-3·2)			
Chromium (μmol/kg)	Dyslexia	16·0 (10·0-22·3)	0·87	0·81 to 0·93	0·002**
	Control	18·0 (15·0-27·7)			
Copper (mmol/kg)	Dyslexia	0·24 (0·17-0·46)	1·16	1·04 to 1·31	0·009*
	Control	0·20 (0·17-0·39)			
Nickel (μmol/kg)	Dyslexia	7·2 (4·4-10·7)	0·99	0·93 to 1·06	0·476
	Control	7·3 (3·6-12·4)			
Sodium (mmol/kg)	Dyslexia	4·6 (1·8-42·7)	0·94	0·71 to 1·24	0·899
	Control	5·1 (1·5-14·2)			
Manganese (μmol/kg)	Dyslexia	20·0 (14·6-43·7)	0·94	0·85 to 1·03	0·179
	Control	21·8 (10·9-49·1)			
Magnesium (mmol/kg)	Dyslexia	1·0 (0·5-6·6)	0·93	0·68 to 1·23	0·282
	Control	0·9 (0·4-7·1)			
Cobalt (μmol/kg)	Dyslexia	4·5 (2·4-6·8)	0·92	0·79 to 1·08	0·347
	Control	4·7 (1·9-8·8)			
Calcium (mmol/kg)	Dyslexia	10·2 (4·2-18·0)	1·09	0·89 to 1·35	0·409
	Control	10·0 (3·3-18·6)			
Potassium (mmol/kg)	Dyslexia	1·2 (0·2-9·1)	0·83	0·60 to 1·15	0·559
	Control	1·5 (0·3-12·0)			
Iron (mmol/kg)	Dyslexia	0·5 (0·3-0·8)	0·92	0·81 to 1·05	0·253
	Control	0·5 (0·3-0·8)			
Selenium (μmol/kg)	Dyslexia	26·6 (16·4-43·0)	1·02	0·92 to 1·14	0·590
	Control	26·6 (20·3-40·5)			
Phosphorus (mmol/kg)	Dyslexia	5·7 (3·5-7·1)	0·99	0·90 to 1·08	0·829
	Control	5·5 (4·0-7·2)			
Lead (μmol/kg)	Dyslexia	26·3 (14·0-74·3)	1·24	1·05 to 1·45	0·016*
	Control	20·5 (11·6-65·2)			
Cadmium (μmol/kg)	Dyslexia	0·84 (0·36-2·85)	1·31	1·08 to 1·59	0·004**
	Control	0·71 (0·36-1·87)			
Aluminium (mmol/kg)	Dyslexia	0·13 (0·05-0·36)	1·12	0·93 to 1·37	0·119
	Control	0·10 (0·04-0·45)			
Mercury (μmol/kg)	Dyslexia	1·7 (0·3-3·5)	1·18	0·95 to 1·47	0·045*
	Control	1·3 (0·4-2·2)			
Arsenic (μmol/kg)	Dyslexia	1·1 (0·5-1·7)	0·92	0·77 to 1·10	0·324
	Control	1·2 (0·5-3·2)			

*p<0·05, **p<0·005, ***p<0·0001.
†Obtained from matched pair *t* 95% interval for mean difference in log concentration. Intervals below 1·0 indicate lower concentrations in dyslexic children; intervals above 1·0 indicate higher concentrations in dyslexic children; intervals including 1·0 indicate no significant difference between dyslexic children and controls at the 5% level of significance.
Conversions—Zinc: 1 μmol=65·4 μg. Chromium: 1 nmol=52 ng. Copper: 1 μmol=63·5 μg. Nickel: 1 μmol=58·7 μg. Sodium: 1 nmol=23 ng. Manganese: 1 nmol=54·9 ng. Magnesium: 1 μmol=24·3 μg. Lead: 1 mmol=207·2 mg. Cadmium: 1 nmol=112·4 ng. Aluminium: 1 mmol=27 mg. Cobalt: 1 μmol=58·9 μg. Calcium: 1 mmol=40·1 mg. Potassium: 1 mmol=39·1 mg. Iron: 1 mmol=55·8 mg. Selenium: 1 μmol=79 μg. Phosphorus: 1 mmol=31 mg. Mercury: 1 μmol=200·6 μg. Arsenic: 1 μmol=74·9 μg.

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partner (sweat concentration 5·5 μmol/l) had the highest concentration of lead in hair in the dyslexic children (74·3 μmol/kg), suggesting that an environmental agent was a factor. The children with dyslexia also tended to have lower chromium and higher copper, lead, and cadmium concentrations in both sweat and hair and a higher mercury concentration (measured only in hair) than the controls.

Comment

This study shows clear evidence of an association between dyslexia and low concentrations of zinc in sweat. As in previous studies, higher concentrations of copper, lead, and cadmium and no differences in zinc concentrations were found in hair from dyslexic children compared with controls. In a study of adults one of us (SD) found that when sweat zinc concentration is very low hair zinc concentration may be low, normal, or high and serum zinc concentration may be in the low normal range; when supplementation was given serum zinc concentrations became normal within a day or two but sweat zinc concentrations took longer to increase to the normal range.[2]

Ward *et al* studied concentrations of 37 elements in placental tissue from obstetrically normal births and showed similar mineral imbalances of (lower zinc and higher lead and cadmium concentrations), which were related to reduced head circumference in neonates.[5] In animals zinc deficiency during pregnancy can cause learning impairment, behaviour disorders, and immune dysfunction persisting for several generations. Brains of animal offspring deprived of zinc have shown increased concentrations of catecholamine and copper and random permanent microscopic abnormalities in the hippocampus, which is essential for working memory.[1] Zinc deficiency in either parent before conception may possibly contribute to familial dyslexia. Prospective studies of zinc concentrations and the effects of supplementation before conception, during pregnancy, and in childhood are urgently needed.

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1 Stanstead HH. Zinc: essentiality for brain development and function. *Nutr Rev* 1985;**43**:129-37.
2 Davies S. Effects of oral zinc supplementation on serum, hair and sweat zinc levels in 7 subjects. *Sci Total Environ* 1985;**42**:45-8.
3 Howard J. Zinc status and sweat levels. *Clin Chem* (in press).
4 Passwater RA, Cranton EM. *Trace elements, hair analysis and nutrition*. New Canaan, Connecticut: Keats, 1983:291-303.
5 Ward NI, Watson R, Bryce-Smith D. Placental element levels in relation to fetal development for obstetrically "normal births": a study of 37 elements. Evidence for effects of cadmium, lead and zinc, on fetal growth, and for smoking as a source of cadmium. *International Journal of Biosocial Research* 1987;**9**:63-81.

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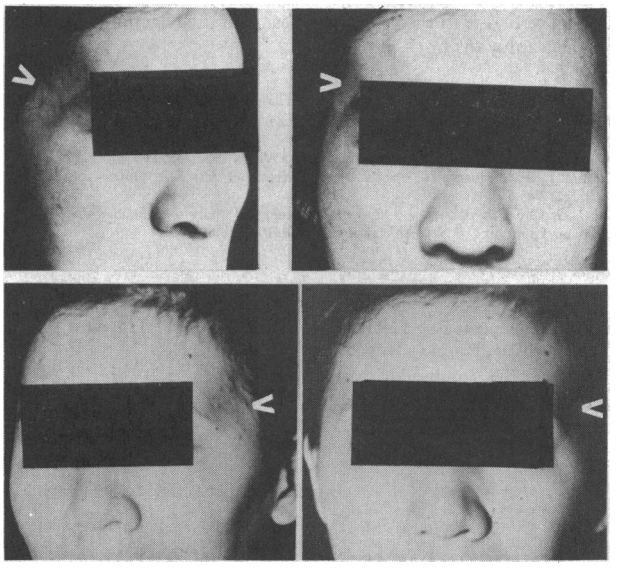
Pure trigeminal motor neuropathy

Among trigeminal sensory disorders findings related to trigeminal sensory neuropathy (also referred to as trigeminal neuropathy, isolated facial numbness or trigeminal neuritis),[1] trigeminal trophic syndrome,[2] and Raeder's paratrigeminal syndrome[3] have been reported. So far as we know pure trigeminal motor neuropathy—trigeminal motor paralysis unaccompanied by trigeminal sensory signs and without affecting other cranial nerves—has not been reported. This study investigated five patients with pure trigeminal motor neuropathy encountered over several years in our outpatient department.

Patients and methods

Five patients with pure trigeminal motor neuropathy were studied. The electromyography, electrically elicited blink reflex[4] and brain stem auditory evoked potentials[5] were examined with a DISA electromyograph. Computed tomography of the brain (4 mm thin slice on the brain stem) was performed by a Siemens Somatom DR3. *x* Ray films of the skull were also taken.

In case 1 a 30 year old man suffered from dull pain of the right cheek on chewing and right masticatory weakness a few days after a common cold. Afterwards muscle wasting in the right temporal area and cheek was noticed (figure). He denied a family history of pure trigeminal motor neuropathy or other



Wasting of masseter and temporalis muscles (arrowed) in cases 1 (top) and 4 (bottom).

forms of neurological disease. Neurological examination showed weakness and wasting of the right masseter and temporalis muscles. The jaw deviated slightly to the right when he opened his mouth fully. Corneal reflexes were active bilaterally, while sensation on the face and taste were intact. There was no anosmia, visual defect, ophthalmoplegia, nystagmus, ptosis, facial weakness, or disturbance of hearing, speech, or swallowing. No bruit was heard of the neck, orbit, or temporal areas and no motor, sensory, or reflex abnormalities were found in his extremities.

Similarly, in case 2 a 39 year old man complained of soreness of the right cheek and weakness of biting on the right side after a common cold. About eight months later an obvious wasting of the right temporal area and cheek was found. In case 3 a 20 year old man suffered from pain of the right cheek and right masticatory weakness after a common cold. Several months later muscle wasting in the right temporal area and cheek was noted. In case 4 a 22 year old woman had noted progressive weakness during mastication on the left side. About six months later muscle wasting in the left temporal area and cheek was found (figure). In case 5 a 24 year old man progressively suffered from left masticatory weakness. One year later he noticed muscle wasting in the left temporal area and cheek. Medical and family histories in cases 2-5 were non-contributory. Neurological examinations showed nothing abnormal except for definite wasting and weakness of the affected masseter and temporalis muscles.

Test results—In all five cases electromyography showed abnormal spontaneous activity, decreased recruitment pattern and chronic neurogenic motor unit potential changes in the affected masseter and temporalis muscles; these remained normal in the frontalis, orbicularis oculi, orbicularis oris, sternocleidomastoid, and tongue muscles. The electrically elicited blink reflex, brain stem auditory evoked potentials, computed tomograms and skull radiographs were all normal.

Comment

Since it is rare to have histological proof of a pathologic lesion in pure trigeminal motor neuropathy it seems advisable to accept clinical or laboratory evidence of a disorder of the trigeminal motor nerve as justification for use of the term pure trigeminal motor neuropathy. In our study electromyography showed evidence of trigeminal motor denervation. Electrically elicited blink reflex indicated normal functioning of the trigeminal sensory nerve and facial nerve. Brain stem auditory evoked potentials and computed tomography showed normal functioning of the acoustic nerve and ruled out brain stem lesions. The other cranial nerves were intact according to normal electromyography and clinical findings. Therefore, only the trigeminal motor nerve was affected in these cases.

The most common causes of trigeminal neuropathy are neoplasm, stroke,

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