of their previous investigation and follow up. The control group comprised 530 normal children seen in hospital for reasons unrelated to the genitourinary tract (308 girls, 222 boys; age range 5-14, mean 8.8 years; 375 were aged 9 or under). Statistical analysis was with fourfold tables χ^2 test.

Twenty three patients (34%) with reflux but only 42 (8%) of the controls were enuretic ($\chi^2=42.7$, $p<0.001$) (table). There was no significant difference in the prevalence of primary nocturnal enuresis between children with different grades of reflux or between children with bilateral and unilateral reflux: 15 (31%) of 49 with grade I or II vesicoureteric reflux and eight (44%) of 18 with grades III-V were enuretic, and 15 (30%) of 50 children with unilateral and eight (47%) of 17 with bilateral vesicoureteric reflux were enuretic. The prevalence of enuresis was the same in the children with reflux aged 9 or under as in those aged 10-14 ($\chi^2=0.01$), but the difference in the prevalence of enuresis between the same age groups in normal children was considerable ($\chi^2=3.49$, $0.05<p<0.1$) (table). Eight (24%) of the 34 children who had vesicoureteric reflux and had had a recent urinary infection were enuretic compared with 42 (8%) of the 530 controls ($\chi^2=9.62$, $0.001<p<0.01$). Furthermore, none of the enuretic children who had reflux and had had a recent urinary infection stopped bed wetting after their infection had been treated. Enuresis was twice as common in children who had a longstanding infection (five of 13) as in those with a recent infection (eight of 34) ($\chi^2=3.57$, $0.05<p<0.1$).

Prevalence of primary nocturnal enuresis in children who had vesicoureteric reflux

	No of children	No (%) with enuresis
Children with reflux:		
Aged 5-9	49	17 (35)
Aged 10-14	18	6 (33)
Total	67	23 (34)
Controls:		
Aged 5-9	375	35 (9)
Aged 10-14	155	7 (5)
Total	530	42 (8)

Comment

Our results showed a fourfold increase in the prevalence of primary nocturnal enuresis among children who had vesicoureteric reflux compared with normal children. It is difficult to establish a direct causal relation between the presence of reflux and the occurrence of enuresis; some urodynamic studies, however, have shown that detrusor instability in children who have vesicoureteric reflux is quite common,⁴ and bladder instability also often occurs in enuretic children.⁵

In enuretic children who have vesicoureteric reflux the various stressful events that are related to their urological problem do not seem to be solely responsible for the higher prevalence of enuresis among them. The prevalence of enuresis remained high even in the group of children who had had only a recent urinary infection. Furthermore, the prevalence of primary nocturnal enuresis in children who had reflux was uninfluenced by the presence of a urinary infection.

The prevalence of primary nocturnal enuresis remained virtually unchanged in children who had reflux in the two age groups 5-9 and 10-14 years, unlike that in normal children. It would be interesting to discover whether the spontaneous resolution or surgical cure of reflux also has a therapeutic effect on enuresis.

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Extranodal lymphoma in a haemophiliac negative for antibody to HIV

Diseases associated with serum antibodies to human T cell lymphotropic virus type I and human immunodeficiency virus (HIV) include lymphoproliferative neoplasia.^{1,2} Among haemophiliacs treated with factor VIII concentrates the prevalence of seropositivity for antibody to HIV is high, and non-Hodgkin's lymphoma has been reported in patients with the antibody.³ We report the occurrence of extranodal non-Hodgkin's lymphoma in a haemophiliac given concentrate whose cell mediated immunity was impaired but who was negative for antibody to HIV.

Case report

A 50 year old patient deficient in factor VIII (<0.01 U/ml) was admitted with haemarthrosis of the right knee. He was taking ibuprofen 1200 mg daily and dihydrocodeine for chronic haemophilic arthritides. He had received 75 000 units of factor VIII concentrate in the previous four years. His bleeding episode was treated with transfusion of factor VIII concentrate, analgesia, and physiotherapy. He was negative for antibody to HIV by both enzyme linked immunosorbent assay (ELISA) and Western blotting. Tests for antibodies to human T cell lymphotropic virus type I also yielded negative results. Intradermal infection of both purified protein derivative and dinitrochlorobenzene yielded no reaction. The T lymphocyte ratio was 0.36 (T helper cells $0.7 \times 10^9/l$ and T suppressor cells $1.925 \times 10^9/l$).

Two weeks after admission he had a haematemesis and his blood pressure fell to 80/50 mm Hg. Three units of blood and 3000 units of factor VIII concentrate were transfused. Endoscopy showed superficial gastric ulcers; ibuprofen was stopped, and he was treated with factor concentrate and ranitidine.

Though there was no further gastric bleeding, his renal function deteriorated, having been normal on admission. Serum urea concentration on the day of the haematemesis was 32.3 mmol/l and creatinine 180 $\mu\text{mol/l}$; a week later creatinine had risen to 370 $\mu\text{mol/l}$. He was initially presumed to have acute tubular necrosis, and fluid overload was corrected with diuretics. Renal ultrasonography showed both kidneys to be enlarged. Management was complicated by chest infections, and on one occasion he suffered septicaemia and cardiopulmonary arrest, from which he was resuscitated. Fourteen days after his haematemesis serum creatinine concentration was 900 $\mu\text{mol/l}$. Renal biopsy was not performed because of the risk of bleeding: an inhibitor to factor VIII was detected six weeks after the onset of renal failure. Prednisolone 40 mg was then started, with a transient improvement in renal function (creatinine concentration 450 $\mu\text{mol/l}$). His condition deteriorated; and he was considered unsuitable for dialysis. He died of bronchopneumonia and septicaemia due to *Escherichia coli*.

At necropsy both kidneys were enlarged (250 g) with uniform pale cut surfaces. The corticomedullary junction was poorly defined, and there was no evidence of scarring or haemorrhage. Microscopy showed a diffuse, mainly cortical infiltrate of large lymphoid cells (figure), which were pleomorphic with convoluted nuclei and numerous mitoses. Results of immunocytochemical staining with common leucocyte antigen were positive. Fresh tissue was not available to define the lymphocyte infiltrate in more detail. There was no evidence of tumour infiltrate in the rest of the organs examined.

Comment

The presentation, site, and nature of the lymphoma in our patient were rare; to our knowledge this is the first report of an extranodal non-Hodgkin's

Renal cortex expanded by diffuse tumour infiltrate with relative sparing of glomeruli. Many tubules are atrophic; the rest show degenerative changes.

lymphoma in a haemophilic negative for HIV. Serum samples from 1983 and 1984 examined retrospectively also failed to show this antibody. All serum samples were negative for antibody to human T cell lymphotropic virus type I.

Histologically similar extranodal lymphomas have been reported in homosexual men negative for antibody to HIV but with inverted ratios of T helper to suppressor cells.⁴ The aetiology of the lymphomas in such patients is not clear. Congenital and other acquired immune defects are also associated with lymphomas. We previously reported abnormal cell mediated immunity in haemophiliacs with factor VIII concentrate and negative for HIV antibody.⁵ Such defects may have contributed to the pathogenesis of the tumour in our patient.

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Tinel's sign and the carpal tunnel syndrome

The diagnostic value of Tinel's sign in the carpal tunnel syndrome depends on (a) the wrist position—if dorsiflexed the median nerve becomes stretched and tightens the contents of the carpal tunnel—and (b) the mode of eliciting the sign—thumb pressure, fingertip, or patellar hammer percussion. Stretching the median nerve by the wrist flexion test has also been recommended. We assessed these techniques to determine the best way of eliciting Tinel's sign and its value.

Patients, methods, and results

Twenty seven patients (19 women, 8 men, aged 22-80 (mean 53)) were examined. Sixteen had unilateral and 11 bilateral carpal tunnel symptoms. Both hands of 14 control patients were also assessed. In all patients we examined both wrists with (a) hyperextension alone, (b) firm thumb pressure once over the carpal tunnel, (c) percussion (two fingers) six times, and (d) multiple percussion, 6-10 beats with a soft "Queen Square" tendon hammer over and proximal to the carpal ligament. A positive response was recorded when the patient complained of distal or proximal paraesthesia, and percussion ceased when this response was obtained. Paraesthesia within 60 seconds with the wrist acutely flexed (wrist

flexion test; Phalen's sign) was also assessed. All symptomatic patients and those controls with a positive Tinel's sign underwent electromyography of the median nerve.¹

In normal wrists repeated firm percussion with a Queen Square tendon hammer produced "awareness" only. In 31 wrists with a hyperaesthetic median nerve hammer percussion resulted in paraesthesia: in 27 this was felt distally, in three proximally, and in one in both directions. Of the 38 hands with symptoms of median nerve compression 33 had the diagnosis confirmed by electromyography. The responses to the various tests are shown in the table. Five paraesthetic hands (five patients) with normal median nerve conduction had a positive Tinel's sign: one patient gave a classical history of carpal tunnel syndrome but was improving when examined, another had thenar wasting and weakness and shoulder-hand syndrome secondary to frozen shoulder, and a third had neurogenic changes in abductor pollicis brevis with normal results on sensory studies.

Seven of the 33 symptomatic hands with electromyographically confirmed median nerve compression at the wrist did not give a positive Tinel's sign on any of the tests; but there was no relation between the severity or duration of symptoms and the absence of Tinel's sign. No patient had complete analgesia in the median nerve distribution. Four of the 14 control patients gave a positive Tinel's sign: two had peripheral neuropathy confirmed on electromyography (one diabetic, one uraemic), one had delivered a baby nine months earlier, and the fourth had been taking prednisone 15 mg daily for a year; these last two patients gave normal results on electromyography.

Comment

Extending the wrist to tense the contents of the carpal tunnel so that percussion is transmitted to the median nerve may be essential in obtaining Tinel's sign. Also, a broad based Queen Square hammer seemed better than smaller hammers or fingertip or thumb pressure because the whole transverse carpal ligament and several millimetres proximally could be percussed. A false neuroma is sometimes encountered above the carpal ligament, and tension or compression of that area might account for the paraesthesiae when the wrist is extended or flexed. Five of the symptomatic wrists with a positive Tinel's sign gave normal results on electromyography, but this is common, particularly in mild cases.^{1,2}

Failure to elicit Tinel's sign in electromyographically confirmed carpal tunnel syndrome may have been due to finger percussion,³ "gentle tapping" with a patellar hammer,⁴ or failure to appreciate that the carpal tunnel contents are compressed when the wrist is extended.⁵ We therefore conclude that percussing the extended wrist over and immediately proximal to the carpal tunnel using a Queen Square hammer is the simplest and best way to elicit Tinel's sign.

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Sensitivity of Tinel's sign elicited by different methods

Method of eliciting Tinel's sign	No tested	Positive sign	Positive electromyogram	Positive sign and electromyogram	Sensitivity† (%)	False positives (%)
Tendon hammer percussion*	38	31	33	26	79	16
Finger percussion*	38	19	33	16	49	16
Wrist flexion	32	11	27	9	33	18
Thumb pressure*	35	8	31	7	23	13
Hyperextension of the wrist*	38	2	33	2	6	0

*Performed with the hyperextended wrist.

†Sensitivity = positive sign with positive electromyogram × 100/positive electromyogram.