were stored at -18°C until assayed using materials supplied by the National Institute of Arthritis and Metabolic Diseases, USA. Expressing the results as percentages (mean $\pm$ SEM) of the plasma prolactin value obtained when blood was spun immediately after venepuncture, at 2 days at 37°C the value in the men was $116 \pm 16\%$ and in the women $140 \pm 21\%$ of the initial value: at 5 days at 37°C the mean value in men was $77 \pm 12\%$ and in women $228 \pm 59\%$ ($P < 0.005$) of the initial value—that is, the apparent plasma concentration in the women more than doubled. Haemolysis occurred to a similar degree in both sexes. When the samples were deliberately haemolysed by adding distilled water the apparent plasma concentration fell by an amount greater than could be accounted for by the water added. Haemolysis might therefore have contributed to the apparent fall in plasma prolactin seen in the men at 5 days but could not have accounted for the rise seen in the women. When blood was incubated for 5 days at 4°C the mean plasma prolactin concentration remained steady in both sexes: at 22°C values intermediate between those at 4°C and 37°C were obtained.

When samples were incubated for only 6 hours no changes in plasma prolactin were seen at 4°C and in most of the 19 persons no substantial changes occurred at 22°C or 37°C . There was, however, a tendency for those with high initial plasma prolactin levels to show a fall at 37°C over 6 hours and for those who had low initial plasma levels to show a rise. That raises the possibility that the presence of cells may have some sort of buffering action, releasing prolactin into the plasma when initial levels are low and taking up the hormone when initial levels are high. Occasionally, even with a 6-hour incubation, changes in apparent plasma prolactin values were substantial. These are illustrated in the table, which shows the findings in a man who had early morning attacks of asthma. Samples were taken at 10 am on three separate occasions at least five days apart. On the days when it was used the isoprenaline had been inhaled 2–6 hours previously.

Plasma prolactin concentrations ($\mu\text{g/l}$) before and after 6 hours' incubation of whole blood in an asthmatic patient sampled on three separate occasions

Incubation temperature ($^{\circ}\text{C}$)	Asthma + isoprenaline		No asthma no isoprenaline		No asthma isoprenaline	
	Before	After 6 h	Before	After 6 h	Before	After 6 h
4	11	19	22	22	21	19
22	11	22	22	21	21	41
37	11	26	22	21	21	40

Discussion

These results are of obvious importance for those studying human prolactin. Our main conclusions are: (1) blood samples for prolactin estimation should normally be spun immediately, though possibly in some individuals extra information may be obtained by incubating an aliquot at 37°C for six hours before centrifugation; (2) when carrying out receptor studies on human tissue care should be taken to wash out all blood; (3) prolactin associated with blood cells may sometimes provide a better guide than plasma prolactin to tissue prolactin levels; and (4) drugs may influence the association of prolactin with cells in the blood.

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Iatrogenic femoral neuropathy

A widespread use of anticoagulants has resulted in several complications, particularly bleeding from mucus membranes and haematoma formation. Neurological complications include subarachnoid haemorrhage, subdural haematoma, intracerebral or cerebellar haemorrhage,¹ spinal epidural haematoma,² and neuropathy associated with muscle haematoma.³ This report describes a patient on anticoagulant therapy who developed a femoral neuropathy after spinal manipulation.

X-ray appearance of displaced right ureter.

Case report

The patient, a 55-year-old man, received warfarin sodium after myocardial infarction in March 1973. In January 1974 he developed acute left sciatica and consulted an osteopath, who performed three spinal manipulations. After the last of these, the patient developed a dull pain in the right iliac fossa, relieved by hip flexion. The pain increased in severity over four days and his right leg became weak.

On admission to hospital he had definite tenderness in the right iliac fossa associated with a palpable intra-abdominal mass, about 10 cm in diameter, extending up from the right inguinal ligament. The right iliopsoas and quadriceps femoris muscles were very weak, knee jerk was absent. Sensation was impaired over the medial lower thigh. A thrombotest result was 12% (range for adequate anticoagulation 8–13%). An intravenous urogram showed displacement of the right ureter towards the midline (see fig).

Iliopsoas haematoma with femoral neuropathy was diagnosed and the warfarin was stopped. The haematoma resolved over 10 days. With physiotherapy power improved, but at follow-up 18 months later (August 1975) the right thigh was wasted (mid-thigh diameter 4.5 cm less than left side), hip flexion was normal, but knee extension was still moderately weak. The knee jerk was present but diminished and there was no sensory deficit.

Discussion

Femoral neuropathy after spontaneous haematoma formation in patients on anticoagulant therapy is uncommon but well recorded.³ It has also been described in haemophilia⁴ and after ruptured aortic aneurysm.⁵ The nerve is formed within the psoas muscle, descends between psoas and iliacus supplying them, and passes beneath the inguinal ligament to supply quadriceps femoris, with a cutaneous branch to the medial aspect of the thigh. With an iliopsoas haematoma the nerve is stretched and probably rendered ischaemic, although no pathological material has been examined. A further possible mechanism would be the infiltration of blood between the nerve fibre bundles.

In this patient the preceding spinal manipulation was most likely to have precipitated the haematoma in a patient with an iatrogenic haemorrhagic diathesis. The case history thus serves as a reminder of the hidden risks of long-term anticoagulants and a warning that manipulation in such patients is also not without appreciable risk.

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