correlation between the two, suggest that the serum ferritin concentration partly reflects the ferritin load within the synovium. This explains the apparent anomaly, occasionally found in patients with rheumatoid arthritis, whereby serum ferritin concentrations are raised with disproportionately low marrow iron stores. We postulate that in these cases the reticuloendothelial cells within the synovial membrane, stimulated by the inflammatory process, actively take up iron and store it as ferritin, in direct competition with the bone marrow. Here, because of some as yet unspecified block in the release of iron from intracellular ferritin inflammatory states, it is no longer freely available for haemoglobin synthesis.[4]

Thus concentrations of ferritin in synovial fluid may reflect reticuloendothelial activity within the synovial membrane, and these observations merit further study.

We thank Dr M Thompson, department of rheumatology, Royal Victoria Infirmary, Newcastle upon Tyne, for his help with this study.

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(*Accepted 17 June 1980*)

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Galactorrhoea after withdrawal of bromocriptine

We describe a young woman with Parkinson's disease who developed transient galactorrhoea and hyperprolactinaemia after withdrawal of treatment with bromocriptine.

Case report

A 21-year-old housewife presented in 1975 with signs of Parkinson's disease limited to the right side. There was no history of encephalitis or treatment with drugs and no relevant family history. Results of the following investigations were normal: skull radiography, electroencephalography, isotope brain scan, computed tomography, slit-lamp examination of the cornea, and serum caeruloplasmin concentration.

In October 1976 she began taking levodopa and carbidopa. In August 1977 she developed involuntary movements of both legs, and her treatment was changed to benzhexol alone. While taking this she became increasingly hypokinetic, and in November 1977 began taking bromocriptine, 5 mg three times daily then 10 mg three times daily after one month.

There was considerable improvement in the hypokinesia without dyskinesia and she remained well until the last quarter of 1979, when she began to show the "on-off" phenomenon in addition to developing signs of Parkinsonism on the left side. Treatment with bromocriptine was stopped on 6 December 1979 and she continued taking benzhexol alone. Three weeks later she noted enlargement of the breasts and could express milk easily from both. Her menstrual periods remained normal. Serum prolactin concentration, first measured on 28 January 1980, was raised at 1950 mU/l (normal range 80-390 mU/l). During the next three months the galactorrhoea resolved spontaneously, and the serum prolactin concentration returned to normal (293 mU/l on 5 March 1980). When the basal prolactin concentration had fallen to 435 mU/l there was a large increase in prolactin concentration after the intravenous injection of 200 μg thyrotrophin-releasing hormone (table).

Tomograms of the pituitary fossa showed no evidence of pituitary tumour. The following were normal: serum total thyroxine concentration, basal serum thyrotrophin concentration, the response of serum thyrotrophin concentration to intravenous thyrotrophin-releasing hormone, basal concentrations of serum gonadotrophins and the response of these to intravenous gonadotrophin-releasing hormone, and diurnal variation in plasma cortisol concentration. An eight-hour water-deprivation test gave a normal result.

Response of serum thyrotrophin and prolactin concentrations to intravenous injection of 200 μg thyrotrophin-releasing hormone

Time after injection (min)	Thyrotrophin (mU/l)	Prolactin (mU/l)
0	2·7	435
20	12·6	4368
60	11·6	2246

Comment

Bromocriptine inhibits the secretion of prolactin from the anterior pituitary. There have been no other reports of hyperprolactinaemia after stopping treatment with bromocriptine for Parkinson's disease. In patients with hyperprolactinaemia, however, serum prolactin concentrations may return to pretreatment values when bromocriptine is withdrawn.

The occurrence of galactorrhoea and hyperprolactinaemia in our patient three weeks after stopping treatment with bromocriptine therefore raised the possibility of an underlying lesion, such as a prolactinoma, that had been masked by giving bromocriptine. Radiography of the pituitary fossa, however, showed no evidence of a tumour; the response of serum prolactin concentration to intravenous thyrotrophin-releasing hormone was not in keeping with a prolactinoma[1]; and the spontaneous resolution of the hyperprolactinaemia was inconsistent with any underlying abnormality of prolactin secretion.

Dopamine inhibits the secretion of prolactin, and brain dopamine is depleted in Parkinson's disease. Serum prolactin concentrations in patients with untreated idiopathic Parkinson's disease, however, are normal or only slightly increased,[2] suggesting that hypothalamic dopaminergic neurones are relatively unaffected by the disease. Galactorrhoea that persisted for five years has been described in one case of classical postencephalitic Parkinson's disease,[3] but the assay for serum prolactin was not then available. The aetiology of our patient's Parkinson's disease is uncertain, but the spontaneous resolution of the hyperprolactinaemia is inconsistent with any association between the high prolactin concentrations and Parkinson's disease itself. We therefore suggest that the transient hyperprolactinaemia in our patient represented an otherwise undescribed rebound phenomenon after withdrawal of bromocriptine.

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(*Accepted 30 June 1980*)

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Correction

Nocturnal enuresis and the buzzer alarm: role of the general practitioner

An error occurred in this article by Dr G C Close (16 August, p 483). In the footnote the address of N H Eastwood and Son Ltd, makers of the Eastleigh alarm, should have read "70 Nursery Road, London N14 5QH."

Br Med J: first published as 10.1136/bmj.281.6242.716-a on 13 September 1980. Downloaded from http://www.bmj.com/ on 19 April 2024 by guest. Protected by copyright.