into the sella. She received hydrocortisone replacement treatment for one year, and this was withdrawn before pituitary function tests were carried out. These showed normal cortisol and prolactin responses to hypoglycaemia induced with insulin (peak cortisol concentration 860 nmol/l (31 µg/100 ml), prolactin concentration 550 mU/l). Basal growth hormone concentration was 6 mU/l, with a subnormal rise to the combination of hypoglycaemia and administration of thyrotrophin releasing hormone (peak growth hormone concentration 9 mU/l). Responses of luteinising hormone and follicle stimulating hormone to luteinising hormone releasing hormone and of thyrotrophin stimulating hormone to thyrotrophin releasing hormone were absent.

Comment

Spontaneous infarction of the pituitary is well described in acromegaly.¹⁻³ In one review all patients had classical pituitary apoplexy except patients with diabetes mellitus, in whom infarction was silent.¹ Another review reported that 42 out of 93 patients found at the time of hypophysectomy to have haemorrhage from pituitary adenomas gave no history of apoplexy.³ Pituitary necrosis in postpartum haemorrhage was reviewed by Sheehan and Davis, who concluded that it resulted from ischaemia due to a fall in arterial blood supply with or without disruption to portal blood flow.⁴ They suggested that there may be special factors in the puerperium that predispose to pituitary necrosis as it is often associated with pre-eclampsia.

The case we report is the first to show infarction of a growth hormone secreting tumour after a prolonged period of hypotension due to haemorrhage. After pituitary apoplexy hormone function may range from being normal to completely absent. We propose that selective infarction of the tumour occurred as shown by the preservation of a normal pituitary-adrenal axis. As growth hormone concentrations were not fully suppressed after an oral glucose load residual tumour may have remained. The patient also developed intrasellar arachnoid herniation of cerebrospinal fluid. Previous reports have shown that this condition may coexist with pituitary tumours or necrosis.^{4,5} This case, documented by sequential computed tomograms, shows the development of the empty sella syndrome after necrosis of a pituitary tumour.

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Factors affecting day to day incidence of stroke in Nottingham

Mortality due to stroke is known to increase in winter and also shows a negative correlation with temperature in all seasons.¹ A time trend association with changing levels of air pollution has also been reported.¹ Published information does not, however, indicate whether this association is due mainly to a change in incidence of stroke or in the proportion of strokes that are fatal. We investigated the effect of meteorological changes on the day by day incidence of stroke in Nottingham in 1983.

Patients, methods, and results

Nottingham City and University hospitals together take all acute medical admissions for the city and surrounding area (catchment population 600 000-800 000). About 15% of patients with stroke are admitted directly to geriatric wards, but these were excluded from our study. We also excluded

(retrospectively) those who were found to have primary subarachnoid haemorrhage or transient ischaemic attacks and those whose neurological signs were caused by space occupying lesions. We did not attempt systematically to distinguish cerebral haemorrhage from infarction. Daily temperature readings were supplied by the Castle Museum, Nottingham, and records of smoke and gaseous pollution by the Nottingham Environmental Health Office.

Altogether 732 cases of stroke were registered over 51 weeks and the dates of onset of symptoms ascertained. A further 129 cases were excluded for the reasons given above. Taking daily total numbers of cases as the dependent variable, significant correlations were found with daily temperatures and levels of sulphur dioxide and smoke ($p < 0.01$) but not with other meteorological variables such as rainfall and hours of sunshine. Multiple regression analysis indicated that temperature had the strongest effect and that the independent contribution of levels of smoke and sulphur dioxide was negligible. Interposing a lag of one day between the dates of temperature reading and onset of stroke strengthened the association. Even so, only 2.4% of the measured variation in numbers of strokes could be explained by the temperature effect (approximate 95% confidence limits 0.3-6.6%).

Temperature may have influenced the chance of being admitted to hospital rather than the incidence of stroke. We therefore reclassified the cases of stroke by date of admission (rather than onset). We also examined for comparison a group of 464 patients admitted during the same period with upper gastrointestinal bleeding. As the table shows, the numbers admitted daily for this condition showed much weaker correlations with temperature than the figures for incidence of stroke.

Correlations with daily temperature readings

Dependent variable	r*	r ²	p value
No of strokes:			
By date of onset	-0.130	0.017	<0.01
By date one day after onset	-0.154	0.024	<0.01
By date of admission	-0.090	0.008	<0.05
No of cases of upper gastrointestinal bleeding:			
By date of admission	-0.065	0.004	>0.1

*Univariate correlation coefficient.

Comment

These data suggest a significant but small increase in incidence of stroke on cold days. Displacement of dates of onset by one day strengthens the correlation, but displacement by more than a week causes it to disappear; the effect thus seems to be a short term one. Substitution of dates of onset by dates of admission makes the association much weaker so we are confident that we have not simply measured changes in the proportion of cases admitted to hospital. Furthermore, no significant temperature effect could be shown on daily admissions for acute upper gastrointestinal bleeding, although longer term associations with temperature and seasonal variations are known to occur in this condition.²

Overall rates of death due to stroke in Britain and the USA have fallen dramatically over the past 30 years, and data from Rochester, Minnesota, suggest that this is mainly due to a decline in incidence.³ Although workers at the Mayo Clinic attribute this fall almost entirely to detection and treatment of hypertension,³ recent surveys have shown that this is unlikely to be the case in Britain,^{4,5} and alterations in diet and levels of air pollution are possible alternative explanations. Clearly, secular changes in mean temperature could not account for such a large fall either, and the weaker effect for smoke and sulphur dioxide than for temperature shown by our data is evidence against the pollution hypothesis.

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