DEATHS

There were eight deaths among the 77 patients (table III). Seven were elderly and seriously ill with either acute inflammatory disease or cancer. Two of these died of pulmonary emboli. The eighth was a 36-year-old man who died after a motor accident.

Discussion

Infections caused by *Bacteroides* should be treated early in order to diminish the incidence of morbidity and mortality (Bodner *et al.*, 1970; Ellner and Wasilaukas, 1971). Therefore bacteriological investigations should be carried out as soon as possible, and swabs should be cultured within three hours of being taken in order to prevent strict anaerobes from dying out. It was not possible to do this for all the specimens in this series. Specimens taken at operations out of laboratory hours are now being dealt with on an "on call" basis to avoid delay before culture. This has led to an increase in positive results and early reporting of sensitivity tests.

The strains of *Bacteroides* reported here were resistant to penicillin and ampicillin, therefore other antibiotics should be used to treat the infections. Our results confirm that clindamycin is the drug of choice as suggested by the in-vitro studies of Ingham *et al.* (1968), Kislak (1972), and the report of Tracy *et al.* (1972). Other antibiotics may be useful, however, especially erythromycin and lincomycin, which may be given parenterally as well as by mouth. Our in-vitro sensitivity patterns suggest that many of the infections might have responded to co-trimoxazole, but the use of the drug in this infection needs clinical evaluation.

It is difficult to assess the importance of *Bacteroides* as a cause of death in the patients who had concomitant serious disease. Among the septicæmic patients, however, it is perhaps worth noting that of the five treated with clindamycin all survived, whereas of four not so treated only one survived.

Septicæmia may affect any age group from the very young with appendicitis to the elderly with carcinoma. It is necessary to secure early blood cultures from patients—particularly those

where the source of infection is either the bowel or vagina. In this group it is probably worth while adding clindamycin or lincomycin to the antibiotics to be given empirically, as already suggested in *Lancet* (1973).

In this series there was no evidence of bleeding disorder said to be due to a heparinase produced by *Bacteroides* species (Gesner and Jenkins, 1961; Tracy *et al.*, 1972).

We wish to thank the many clinicians who made the clinical data available for us to review.

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MEDICAL MEMORANDA

Regression of the Forbes-Albright Syndrome after Pituitary Apoplexy

E. H. McLAREN, P. C. KEET

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Although pituitary tumours are known to undergo spontaneous infarction, the usual effect of this is pituitary apoplexy with acute necrosis of the gland and subsequent hypopituitarism. We report a case of the Forbes-Albright syndrome in which such an episode resulted in noticeable improvement in endocrinological function, with subsequent pregnancy, but later relapse.

Case History

The patient was a 42-year-old white woman with no significant past history. In 1966 she had an uncomplicated delivery of her fourth child. Lactation was suppressed with oestrogens and her periods returned. A year later she developed permanent amenorrhoea, intermittent galactorrhoea, and migrainous headaches. These continued for four years until the beginning of March 1971, when she suddenly developed a much more severe headache. On 7 April she awoke with a partial left ptosis. The headache and ptosis regressed spontaneously over seven weeks. A skull x-ray picture showed an abnormal pituitary fossa, and a lumbar air encephalogram showed a minimal filling defect in the chiasmatica presumably due to the presence of a pituitary tumour. Endocrine investigation at this time showed a protein bound iodine of 4.7 µg/100 ml triiodothyronine resin of 29% (normal 25-35%), and urinary gonadotrophin of > 2.0 mg I.R.P.2 HMG/24 hours (normal value for human menopausal gonadotrophin, international reference preparation No. 2 is 0.2-1.8). There was a normal cortisol but reduced growth hormone response to hypoglycaemia (table I). It was decided not to treat her at this time because of the risk of radiotherapy causing further pituitary gland necrosis after this pituitary apoplexy.

Her galactorrhoea and headache disappeared and she became pregnant two months after discharge on 9 July. Because of the possibility of pregnancy increasing the growth of her pituitary

Endocrine Unit, Departments of Medicine and Neurosurgery, Medical School, University of Cape Town, South Africa

E. H. McLAREN, M.B., M.R.C.P., Senior Registrar, Endocrine Service, Groote Schuur Hospital

P. C. KEET, M.B., M.MED. (SURG.), Consultant Neurosurgeon, Groote Schuur Hospital

tumour, the pregnancy was terminated and a normal fetus of about 14 weeks' gestation was removed by abdominal hysterotomy in August. After this, she had normal periods for five months until she once again developed amenorrhoea, headaches, and one episode of galactorrhoea.

In March 1972 she developed a further left ptosis not associated with an exacerbation of her headache. The ptosis persisted and she was readmitted for further investigation.

Physical examination showed nothing abnormal apart from a partial left-sided ptosis and partial left third nerve palsy. No milk could be expressed from her breasts. Secondary sexual characteristics were normal. Visual fields and fundoscopy were normal.

Investigation now showed a serum thyroxine of 8.7 µg/100 ml (normal 5.4-13 µg/100 ml), triiodothyronine resin of 28.7%, and total gonadotrophins of 1.5 mg I.R.P.2 HMG/24 hours. The cortisol and growth hormone response to hypoglycaemia remained unchanged (table I). The plasma thyrotrophin response to thyrotrophin-releasing hormone was normal (table II). Plasma prolactin was slightly raised and showed failure of suppression after a glucose load (table III) or stimulation by thyrotrophin-releasing hormone (table II). Tomography of the pituitary fossa showed increasing erosion of the posterior clinoids. Air encephalography showed a pituitary tumour with suprasellar extension (see fig.) and left carotid angiography confirmed extension into the cavernous sinus. She was treated with cobalt radiotherapy (total dose of 6,000 r) and after 1,000 r had almost complete regression of her third nerve palsy although her periods had not returned.

TABLE I—Growth Hormone (GH) and Cortisol Response to 0.1 U Soluble Insulin/kg given intravenously

Time (min)	July 1971			April 1972		
	Blood Sugar (mg/100 ml)	Cortisol (µg/100 ml)	GH (ng/ml)	Blood Sugar (mg/100 ml)	Cortisol (µg/100 ml)	GH (ng/ml)
0	74	21	0.4	64	14	1.2
20	57	17	0.2	42	14	2.3
40	22	16	2.0	22	12	0.9
60	41	25	5.5	32	27	2.7

TABLE II—Thyroid Stimulating Hormone (TSH) and Prolactin Response to 200 µg intravenous Thyrotrophin-releasing Hormone, April 1972

Time (min)	TSH (µU/ml)	Prolactin (µg/ml)
0	3.0	18.2
20	11.0	21.3
60	7.0	20.1

TABLE III—Glucose Tolerance after 50 g Oral Glucose, April 1972

Time (min)	Blood Sugar (mg/100 ml)	Growth Hormone (ng/ml)	Prolactin (ng/ml)
0	72	0.8	22.6
30	126	1.0	22
60	124	0.9	19.8
90	127	—	—
120	74	0.9	21.6

Lumbar air encephalogram showing pituitary tumour with suprasellar extension.

Lazarow (1963). Prolactin was measured by a mixed heterologous immunoassay (Jacobs *et al.*, 1972). Cortisol was measured as fluorogenic corticosteroids by the method of Mattingly (1962).

Comment

This patient presumably had a prolactin secreting tumour which underwent spontaneous infarction. Rushworth (1971) proposed that pituitary apoplexy is defined as occurring when a patient with a pituitary tumour develops a characteristic headache associated with either sudden meningeal irritation, loss of vision, or ophthalmoplegia.

There seems little doubt that improvement of her endocrine status resulted from this episode, as she became pregnant after four years of amenorrhoea followed by the appearance of normal periods when the pregnancy was terminated.

We have not been able to find any reported cases of improved pituitary function after pituitary apoplexy, although it is described after radiotherapy in this syndrome (Ioanitiu *et al.*, 1970).

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METHODS

Growth hormone and thyroid stimulating hormone were measured by a modification of the double antibody method of Morgan and