hyperactivity of the renin-angiotensin-aldosterone system and angiotensin insensitivity as secondary causes.

Furthermore, the depressor response to the antagonist indicates that arteriolar smooth muscle in a patient with Bartter's syndrome still responds to endogenous angiotensin, and that the renin-angiotensin system plays an important part in regulating the blood pressure in this syndrome.³ Of particular interest were the significant changes in the blood pressure response to this antagonist before and after the indomethacin treatment. Similar agonistic (pressor) responses to this or another antagonist (Saralasin) were observed in various types of hypertension with suppressed PRA.^{4,5} It therefore seems likely that even in Bartter's syndrome the antagonistic effect of this antagonist to natural angiotensin largely depends on the levels of endogenous circulating angiotensin II.

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(Accepted 31 May 1977)

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Congenital adrenal hypoplasia and gonadotrophin deficiency

With early diagnosis and replacement therapy patients with congenital adrenal hypoplasia now survive into childhood and adolescence. In a recent report a boy with this condition was found to have a luteinising hormone (LH) deficiency with low concentrations of follicle-stimulating hormone (FSH), which increased only moderately after stimulation with luteinising hormone-releasing hormone (LH-RH).¹ We report a second case of a boy with congenital adrenal hypoplasia associated with deficiency of both LH and FSH.

Case report

The patient was initially diagnosed as having congenital adrenal hypoplasia at the age of 2 weeks when he presented with a salt-losing crisis. No abnormal steroids were found and there was no response to corticotrophin.² Subsequently he was successfully treated with glucocorticoid and mineralocorticoid replacement therapy and his growth and development were normal. He was the fourth child of healthy parents and had three sisters, all of whom had developed normally in puberty.

At 13 years 5 months of age he was referred for assessment of delayed puberty. He was a pigmented boy with normal genitalia. Height was 151 cm (25th centile), weight 53.2 kg (75th centile), and bone age 10.8 years. There were no signs of pubertal development. Initial investigations were directed at establishing normal glucocorticoid concentrations by replacement therapy to suppress the concentrations of adrenocorticotrophic hormone, which had become very high. Normal plasma cortisol concentrations with a normal diurnal rhythm were achieved with 30 mg hydrocortisone daily given in divided doses (20 mg being given in the morning together with fludrocortisone 0.1 mg). Thyroid function was normal, and the hypothalamopituitary axis showed normal responsiveness of thyroid-stimulating hormone and prolactin to intravenously administered thyrotrophin-releasing hormone and of growth hormone to insulin-induced hypoglycaemia. An intravenous LH-RH test (100 µg) produced no response in concentrations of LH and FSH, both remaining below 1 U/l over 90 minutes. After subcutaneous injection of LH-RH 100 µg daily for one week a further intravenous LH-RH test (100 µg) produced a minimal response in LH and FSH concentrations. Peak concentrations of FSH and LH were 1.2 and 5.8 U/l respectively. After three injections of human chorionic gonadotrophin 2000 IU on alternate days

baseline plasma testosterone concentrations rose from 0.7 to 15.3 nmol/l (0.2-4.4 ng/ml) (normal range in men 9-24 nmol/l; 2.6-6.9 ng/ml).

At 14 years 2 months (bone age 11.2 years) he was started on twice-weekly intramuscular human chorionic gonadotrophin 1500 U to induce puberty. Over the next 10 months he grew 7.9 cm, secondary sex characteristics appeared, and his bone age advanced to 15.5 years. A repeat intravenous LH-RH test (100 µg) still showed no response in LH and FSH to stimulation.

Comment

It seems more than coincidental that two boys with a rare adrenal condition should also have disorders of the gonadotrophin axis. Presumably both had adequate concentrations of LH-RH or its equivalent in utero, unless they depended solely on placental gonadotrophin, since sexual differentiation was normal. Timing of the onset of puberty through the activation of the hypothalamopituitary gonadal axis is not yet understood, though adrenal steroidogenesis may play a part.³ Plasma dehydroepiandrosterone and androstenedione concentrations rise some time before the onset of puberty, suggesting that the adrenals are needed to prime the hypothalamus. This would be a reasonable explanation in these two cases. A curious feature, however, is that boys with Addison's disease go into puberty normally. Since the only difference between congenital adrenal hypoplasia and Addison's disease is the time of onset, the hypothalamic priming must take place early in life and the rise in adrenal androgens reactivate an already established mechanism.

Whatever the mechanism of the association between gonadotrophin deficiency and congenital adrenal hypoplasia, children with the condition should be tested early and treated appropriately to keep their pubertal development in line with that of their peers.

Requests for reprints should be sent to Dr C G D Brook.

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(Accepted 12 May 1977)

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Thread sutures seen on gastroscopy: do they cause ulcers or indigestion?

Black silk has been used to suture the anastomosis in gastric operations since Billroth's first partial gastrectomy in 1881. These sutures may occur in recurrent ulcers, but do the sutures cause the ulcers or the ulcers expose the stitch? Sutures without ulceration are also seen on endoscopy; it is not known if they cause symptoms or if they should be removed.

Patients, methods, and results

Two hundred and forty patients referred because of dyspepsia or vomiting after surgery for peptic ulcer were studied endoscopically. Eight patients had sutures situated in a chronic ulcer measuring from 0.7 to 2.5 cm. Thirty-one had a suture alone with no ulcer and 63 a chronic ulcer alone with no suture. Three had a gastric ulcer.

Pentagastrin tests were performed on the eight patients with sutures in an ulcer. These patients were matched for age (within five years), sex, type of operation, and interval since that operation (within two years), with eight patients having ulcers without sutures and eight having sutures without ulcers. Their peak acid outputs (PAO) are shown in the table. The mean PAO of patients with ulcers with or without sutures were not significantly different, but they were significantly higher than those of patients with sutures alone ($P < 0.01$ using Wilcoxon's matched pairs test).

Suture removal was achieved apparently successfully on endoscopy in

three patients with ulcers and sutures. In only one patient did the ulcer heal, but it recurred three months later without a visible suture. Sutures were removed from 18 patients without ulcers. In 17 symptoms improved initially, but in 13 symptoms had recurred within three months. Six of these patients were later selected for bile-diversion surgery. One year after this operation five of these patients were cured of their dyspepsia or vomiting, although four still had sutures visible at endoscopy.

Peak acid output (mmol/hour) in 24 patients matched in three groups; those with ulcers with sutures, ulcers alone, and sutures alone

Operation	PAO after pentagastrin (6 µg/kg)		
	Suture and ulcer	Ulcer alone	Suture alone
Vagotomy and pyloroplasty	14.7	36.1	5.1
Vagotomy and pyloroplasty	18.0	28.6	2.1
Vagotomy and pyloroplasty	22.2	20.7	2.6
Vagotomy and pyloroplasty	26.3	23.0	5.2
Vagotomy and pyloroplasty	20.5	31.2	6.0
Bilroth II partial gastrectomy	17.2	16.1	3.0
Bilroth II partial gastrectomy	24.1	27.0	0
Bilroth I partial gastrectomy	15.0	26.3	0
Average	19.7	25.5	3.1

In a prospective study of vagotomy and antrectomy all willing patients underwent endoscopy six to twelve months after the operation. Black silk was used for the seromuscular layer and catgut for the mucosal layer in all patients. Thirty-two patients volunteered for review endoscopy. Eleven had dyspepsia and in them sutures without ulceration were seen in six (55%). Twenty-one patients had no symptoms, and sutures were seen in 13 (65%).

Discussion

Sutures situated in an ulcer were found uncommonly in this as in other series.^{1,2} Of 71 patients with ulcers at or near the stoma, only eight had visible sutures. Patients with ulcers associated with sutures had dyspepsia and a high PAO, but those with sutures without ulcers had a low PAO and often no symptoms. Thus sutures with and without ulcers are of different significance.

If a recurrent ulcer and suture were found the suture was situated in the ulcer, and so the suture appeared to determine the site of ulceration. Nevertheless, we found that ulceration occurred only with a high acid output. Our results differ from those of Small *et al.*,¹ who found that the mean maximal acid output in six patients with sutures after Bilroth II partial gastrectomy was 11.9 mmol/h (range 0.3 to 36.2). Nevertheless, they did not always see an ulcer on endoscopy, and some patients with sutures without ulcers may have been included. Removal of the stitch endoscopically may heal the ulcer, but there is a high recurrence rate. Of 13 reported cases, only five remained free of dyspepsia after apparently successful stitch removal.^{1,3,4} As PAO is raised, treatment should be directed towards lowering acid output by surgery or cimetidine and not removal of the sutures. A larger matched sample is needed to determine whether ulcers occur with a lower acid output when sutures are present than when they are not.

Kasugai *et al.*⁵ reported symptomatic improvement after endoscopic stitch removal in 16 patients, but the length of follow-up was not given. We found that dyspepsia was relieved only transiently after removal of a stitch alone. The initial improvement was probably a "placebo effect." Sutures were found as frequently in patients without symptoms as with those with dyspepsia. Sutures without ulcers do not cause symptoms, and their removal is pointless.

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(Accepted 27 May 1977)

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Oleogranuloma of the rectum produced by Lasonil ointment

Oleogranuloma (lipid granuloma, oleoma, paraffinoma) of the rectum is a recognised, uncommon sequel of injection treatment for haemorrhoids. Presenting as a rectal tumour it may be mistaken for carcinoma of the rectum. We report a case of a lesion produced by a self-administered "pile" treatment which we consider has not been described.

Case report

A 57-year-old man presented with a history of discomfort which had become worse for three weeks and was associated with a sensation of a lump at the anus. He had had one episode of fresh bleeding per rectum but his bowel habit had been unchanged and he was otherwise well. Digital examination showed a firm plaque in the anterior rectal wall, five cm from the anal margin. The lesion occupied half of the rectal circumference and was mobile on the prostate. Sigmoidoscopy showed a normal mucosa to 20 cm and biopsies were taken from the plaque. A barium enema was also performed, which showed no abnormality in the colon. The histology report of the rectal biopsies described the lesion as a lipid granuloma (oleogranuloma) affecting the submucosa and parts of the mucosa. There was no evidence of malignancy and the overlying mucosa was normal (see figure).

Oleogranuloma of the rectum (H and E) × 125.

On further questioning the patient stated that he had not had injections for haemorrhoids. Nevertheless, he had used Lasonil ointment, a self-administered preparation with a nozzle attachment. On occasions he had experienced pain when administering the preparation. With the nature of the rectal lesion elucidated and the patient reassured, an expectant policy of management was pursued.

Discussion

Oleogranulomata have been reported as complications of injection treatment of haemorrhoids using 5% phenol in almond oil. They are uncommon (Goligher¹ reported that he had not seen a case) and may present several years after the injections have been performed. The lesions are induced by submucosal reaction to the vegetable oil in which the sclerosant is suspended. The patient may present with the sensation of a mass or the lesion may be an incidental finding. On palpation it is a submucous tumour, just above the dentate line, and on occasions may be annular or ulcerating.²

The histological appearance is that of a histiocytic and giant-cell reaction surrounding circular spaces, which represent the oil dissolved out in the processing of the slide. The process may affect both submucosa and mucosa and a macrocyst variety has been reported.³ The