Pathological Changes in a Jejunal Graft used for Œsophageal Reconstruction

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G F, female, aged 13.

History: Hæmatemesis necessitating blood transfusion when 2 days old but breast-fed without difficulty till 6 months. Subsequently, progressive dysphagia and regurgitation. At age of 10 years, stricture with ulceration demonstrated at 29 cm

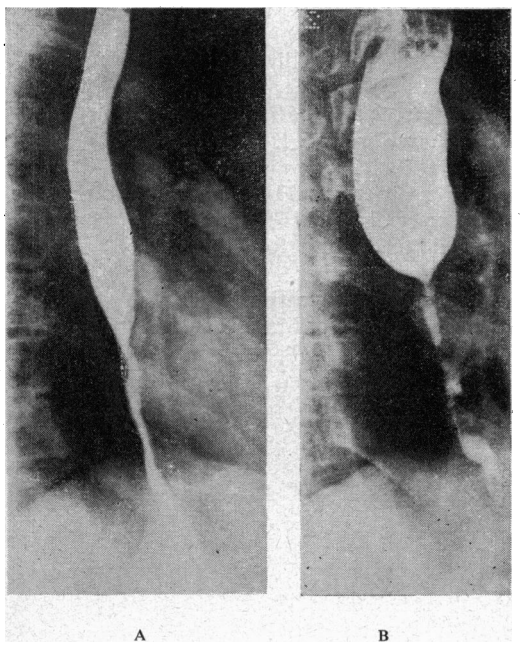

Fig 1 A, *original stricture.* B, *stricture in the jejunal graft*

(Fig 1A). Dilatations carried out at progressively shorter intervals. At age 13, child wasted and ill, and operation decided on.

Operation: Resection of stricture together with hiatus hernia. Jejunal graft 8 in. long implanted between stump of œsophagus above and stomach below – on pedicle of a single artery and vein. Graft extended to stomach which was then entirely below diaphragm and hiatus repaired by suturing together limbs of right crus and suturing lower end of graft to under side of diaphragm.

The preparation of this graft on its vascular pedicle was uneventful and no clamps were used. Jejunal continuity was restored by end-to-end anastomosis. The free length of jejunum was threaded up through a prepared hole in the mesocolon and lesser omentum and was anastomosed to the œsophageal stump. When this anastomosis had been completed, it was noted that the graft was blue and found that this was due to inadvertent rotation through 180 degrees of the graft on its single artery and vein. The anastomosis was undone, the rotation corrected, and top and bottom anastomoses completed. The period of graft cyanosis was less than 15 minutes. After untwisting, the gut was pink and healthy and its artery and branches pulsated. Healing was by first intention and the patient left hospital in two weeks.

Immediately following operation swallowing was almost normal, but within a month dysphagia recurred and progressed. Further X-rays, confirmed by œsophagoscopy, showed a stricture of the whole length of the graft (Fig 1B). Further operation was therefore decided on.

2nd operation: Resection with great difficulty of whole graft. Stomach brought up and joined to stump œsophagus. The jejunal graft macroscopically looked typical of Crohn's disease. Its walls were ¼ to ½ in. thick and the lumen was ulcerated throughout its length. The graft was embedded in enlarged and inflamed mediastinal glands and fibrous tissue.

Microscopically all lining mucosa of graft had vanished. Submucosa of graft was replaced by granulation tissue but without the focal patchy accumulations of lymphocytes seen in some cases of Crohn's disease.

There were no sarcoid-like changes consisting of histiocytes and giant cells present although scattered solitary giant cells were seen here and there. The mediastinal lymph nodes showed inflammatory changes but no histiocytes or giant cells.

A small leak developed at the œsophagogastric anastomosis on the tenth day with the formation of a localized mediastinal abscess, and the patient died suddenly on the fourteenth day from aspiration of vomited fluid.

Post-mortem: Confirmed the above findings. Otherwise the whole intestine, including the jejuno-jejunal anastomosis from which the graft was taken, was normal and there were no enlarged mesenteric glands.

Discussion: The changes in this jejunal graft were macroscopically those of Crohn's disease. Microscopically a typical case of Crohn's disease shows loss of the mucosa from ulceration, and œdematous granulation tissue replacing the normal structures of the bowel wall. By no means all show the classical lymphocytic accumulations nor the sarcoid-like areas with giant cells. In the present case, lymphocytes and sarcoid changes were not

present but the lesion was otherwise indistinguishable from regional ileitis.

This would appear to be the first time that Crohn-like changes have been reported in a jejunal graft, and the possibility arises that these changes were due to the temporary interference with the graft's blood supply. Other writers (Chiene 1869, Hawkins 1957) have drawn attention to similar lesions with jejunal stenosis from mesenteric artery occlusion, and in 1947 Spellberg & Ochsner confirmed what had already been recorded earlier, that similar lesions might result from non-penetrating injury to the small intestine.

This case appears to provide further evidence in support of a relation between vascular interference and intestinal granuloma.

REFERENCES
Chiene J (1869) *J. Anat. Physiol.* 3, 65
Hawkins C F (1957) *Lancet* ii, 121
Spellberg M A & Ochsner M D (1947) *Amer. J. med. Sci.* 213, 579

The following specimens were also shown:

(1) Suppurative Thyroiditis Presenting as Carcinoma of the Thyroid

(2) Hæmorrhage into an Adenoma of the Thyroid
Mr R V Cooke

Sturge-Weber Syndrome
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Mr P T Savage

(1) **Sarcoma of the Ascending Colon**
(2) **Carcinoma of the Rectum Associated with Basal Cell Carcinoma of the Peri-anal Region**
Mr M Ward (for Mr Clive Butler)

Leiomyosarcoma of the Small Intestine
Mr J I Burn (for Mr R H Franklin)

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