resulting in a minute aneurysm; this is surrounded by granulation tissue, and the whole is infiltrated by polymorphonuclear and eosinophil cells. The basis of the lesion therefore appears to be a necrotizing arteritis, and a diagnosis of polyarteritis nodosa must be considered."

Post-operative progress was satisfactory, although on May 28 the patient had another severe attack of abdominal pain associated with lower abdominal tenderness, guarding, and a positive release sign. This second attack was treated by a short course of erythromycin, and he made an excellent recovery except for a continued diarrhoea. Sigmoidoscopy revealed "slight patchy reddening of the mucosa and one definite ulcer, with an irregular edge, rad base, but without a slough." By this time his leucocyte count was 14,800 per c.mm. (12% eosinophils). A left pectoral muscle biopsy was performed (Dr. R. D. Clay) to confirm the diagnosis, and this showed "extensive fibrinoid necrosis of the arteriolar walls, which were densely infiltrated with cells mainly of the polymorphonuclear type. Surrounding the necrotic walls were zones of epithelioid cells, plasma cells, lymphocytes, eosinophil cells, and more polymorphs. This surrounding cellular infiltration was very marked, extending in some areas for a short distance between the muscle bundles. The biopsy thus showed periarteritis nodosa in the acute stage.

The diarrhoea slowly ceased together with some vague abdominal pain of which he had complained. On June 21 treatment with cortisone, 25 mg. twice daily, was begun, and within a week he was stronger; the cortisone was then reduced to 25 mg. daily owing to oedema formation. After three weeks' treatment he was discharged home on the above dosage and continued to remain well apart from two attacks of asthma.

On July 29 he became unconscious after vomiting and was readmitted. The unconsciousness lasted only a few minutes and was followed by irrational and violent behaviour requiring heavy sedation. Later neck rigidity, a positive Kernig's sign, and bilateral early papilloedema developed; a diagnosis of subarachnoid haemorrhage was made and confirmed by lumbar puncture. The blood pressure was 190/145 mm. Hg. The dosage of cortisone was increased to 100 mg. daily for eight days and gradually reduced to 25 mg. twice a day. He recovered satisfactorily from this incident, his blood pressure returning to 140/95 mm. Hg. One further attack of abdominal pain occurred. This was suggestive of peritonitis, but was relieved by sedation. Apart from this incident and slight frontal headaches he recovered well from the subarachnoid haemorrhage. He was eventually discharged on September 20 to continue with cortisone, 25 mg. twice daily. Since then he has remained well, though at no time has his blood pressure been below 140/95 mm. Hg.

## COMMENT

Diseases of the abdominal viscera occupy first place in the minds of most surgeons, and it is only natural that they tend to be most commonly diagnosed in cases of peritonitis. Allen (1940) describes a case of abdominal periarteritis nodosa in which perinephric abscess, appendix abscess, and cholecystitis were considered before laparotomy was performed on a presumed diagnosis of penetrating peptic ulcer. Manges and Baehr (1921) thought that their case was one of suppurative retroperitoneal lymph nodes or perhaps a localized peritonitis in the lesser sac. It would appear that patients with abdominal periarteritis nodosa present with a short history of vague abdominal pain-in our case thought to be due to a peptic ulcer; they may also have pains in the muscles and joints and a history of sore throat. Eventually the abdominal symptoms become more marked and signs of peritoneal irritation develop, leading to surgical intervention. The patient usually has a high temperature-100° F. (37.8° C.) in our case—and a marked leucocytosis. Mullins (1946), in reporting 177 cases of periarteritis, list the most prominent symptoms as fever (81%), leucocytosis (73%), albuminuria (65%), abdominal pain (56%), and hypertension (53%). The presence of albuminuria and hypertension depends on involvement of the renal arteries; in our case there was a raised blood pressure which has remained constant, but no albumin or red blood cells have been found after repeated urinary examination.

Our thanks are due to Mr. J. D. Younghusband and Dr. R. G Moore for permission to publish this case.

A. G. NORMAN, F.R.C.S.
Formerly Senior Surgical Registrar.
P. S. WILKINS, M.B., Ch.B.
Medical Registrar,
St. Mary's Hospital, Portsmouth.

## REFERENCES

Allen, P. D. (1940). Arch. Surg. (Chicago), 40, 271. Logue, R. B., and Mullins, F. (1946). Ann. intern. Med., 24, 11. Manges, M., and Bachr, G. (1921). Amer. J. med. Sci., 162, 162.

## Operation for Midline Epigastric Incisional Hernia

Although the majority of incisional hernias following midline epigastric incision cause little discomfort and can be supported by a suitable abdominal belt, there are a number with wide divarication of the recti and ballooning of the hernia itself which, because they cause both mental and physical discomfort and in some cases exaggeration of preexisting chest symptoms, need surgical repair.

Such hernias can, in the main, be prevented by the careful use of unabsorbable suture, but the price is an incidence of stitch sinus which, although varying in the practice of individual surgeons, occurs in a few cases.

For the type of hernia described above I have used the following procedure with unfailing success in some 12 cases.

The scar is excised by an elliptical incision from xiphisternum to umbilicus and the skin on both sides undermined laterally to expose the peritoneum of the sac and the whole of both anterior rectus sheaths to their lateral edges.

The peritoneum is now repaired where it is deficient or weak and the exposed anterior rectus sheaths are dissected off the underlying rectus muscles to form two rectangular flaps, consisting of external and internal oblique aponeurosis, hinged on their medial margins—that is, along the whole lateral margin of each side of the hernial gap. It is important that these flaps should be raised without damage either to the underlying muscle or to the flaps themselves, and that they should include almost the whole width of the sheath. Bleeding, which is usually troublesome, should be dealt with carefully by ligation and suture.

The flaps are now turned over medially on their hinges and sutured with closely placed interrupted sutures of No. 3 plaited silk to the medial edge of the opposite rectus sheath, this being the original but split linea alba forming the hinge of the opposite flap. The security of these sutures is enhanced by folding under the lateral edge of each flap to obtain a double bite. The gap can always be filled in this way with two double-layered aponeurotic flaps—the suture line of each being protected by the intact hinge of its fellow.

The recti, relieved of the lateral pull of the obliques, tend to approximate slightly during the suturing, and this relief of lateral pull can be appreciated later when the repair is healed, when it can be seen that, on straining, the gap tends to close and the remaining deficiency is well controlled by the aponeurotic graft.

The suturing having been completed and haemostasis checked, the skin incision is closed without drainage.

Any subcutaneous serum is aspirated as soon as it forms during the post-operative period; drainage is avoided, to ensure healing by primary union.

ADDENDUM.—Since submitting this article for publication a description of this operation by Professor Charles Wells has been published (Ann. roy. Coll. Surg. Engl., 1956, 19, 316).

I thank Sir Heneage Ogilvie for his advice concerning the preparation of this article.

H. A. McDonald, F.R.C.S., Consultant Surgeon. Great Yarmouth and Gorleston General Hospital.