pharmacy and will therefore already have a basic understanding of physiology and pharmacology, much of which he gives in unnecessary detail. Terms such as "local anaesthetic" and "EEG" do not need to be defined for them. There is much unnecessary repetition of material, and large numbers of typographical errors, some of them serious and misleading. Surprising errors of fact include the claim that imipramine and amitriptyline cause diarrhoea. The clinically unavailable 4-aminopyridine has 12 lines of text devoted to it, yet the particular danger of dopamine-receptor antagonists in young children does not receive the emphasis it deserves. Basic pharmacological mechanisms are often presented in a confused way, particularly the autonomic system, and some important clinical facts such as the interaction between tricyclic antidepressant drugs and direct sympathomimetic amines are omitted. This book cannot really be recommended as a scientific basis for drug treatment in children.

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**Letter to the Editor**

**Pancreatitis and inguinal swelling**

Sir,

The case report (Dennison and Royle, 1984) describing a patient with acute pancreatitis presenting with an inguinal swelling provides a good illustration of the ability of leaking pancreatic juice to track widely in the retroperitoneal tissues, particularly on the left side. This occurs also in chronic pancreatitis where spontaneous duct disruption may give rise to pancreatico-pleural fistulae or mediastinal pseudocysts by the tracking of pancreatic juice either through the diaphragmatic hiatus or behind the arcuate ligaments (Cameron, 1978). Downward drainage through the inguinal canal does not appear to have been reported previously in this condition.

A 55-year-old man, previously a heavy drinker, presented in 1982 with a long history of recurrent epigastic pain. He had had acute pancreatitis 9 years previously and a right inguinal hernia repair 5 years ago. Physical examination was normal.

Three months later he returned complaining of a painful swelling in the left groin of 1 week's duration. On examination he had marked left inguinal oedema and tenderness extending into the penis and upper scrotum. Four weeks later he was found to have tense ascites which had accumulated rapidly over 4-5 days. Over this same period, his inguinal swelling had disappeared. The ascitic fluid had an amylase content of 10,000 u/l whereas his serum amylase was 1990 on the same day. A diagnosis of pancreatic ascites was made and in an effort to reduce pancreatic exocrine secretion, he was treated with nasogastric aspiration and intravenous fluid replacement, and later by paracentesis and spironolactone.

Two months later, he had no detectable ascites but a tense left hydrocele had arisen during the preceding week. This was aspirated and, interestingly, the amylase content was 1450 u/l in comparison with a serum amylase of 745 at this time. Since then he has had no recurrence of his ascites or hydrocele. During the following year he developed diabetes and a funicular left indirect inguinal hernia, not communicating with the vaginal sac, when a left varicocele was also noted.

It is postulated that the inguinal oedema resulted from tracking of pancreatic fluid retroperitoneally into the left inguinal canal via the internal ring, which was enlarged by the presence of an undetected indirect inguinal hernia. Decompression of this retroperitoneal fluid collection by spontaneous rupture into the peritoneal cavity resulted in the disappearance of the inguinal signs and the development of pancreatic ascites. Retroperitoneal and inguinal fibrosis may have then been responsible for the subsequent hydrocele and varicocele formation by occlusion of the lymphatic and venous drainage respectively, from the testicle.

Yours faithfully,

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**Acknowledgment**

I am grateful to Mr C. H. Talbot for permission to report this case.

**References**


Pancreatitis and inguinal swelling.

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