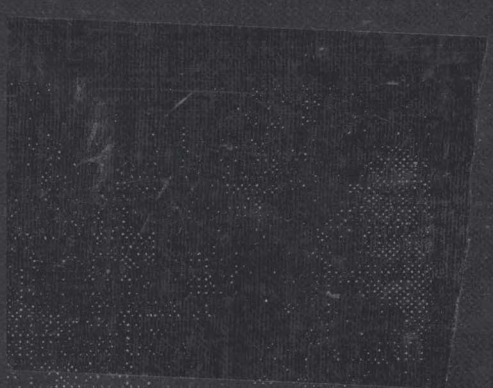
Maingot's Abdominal Operations

Volume II



Ninth Edition

Maingot's Abdominal Operations

Volume II

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Notice: Our knowledge in clinical sciences is constantly changing. As new information becomes available, changes in treatment and in the use of drugs become necessary. The authors and the publisher of this volume have taken care to make certain that the doses of drugs and schedules of treatment are correct and compatible with the standards generally accepted at the time of publication. The reader is advised to consult carefully the instruction and information material included in the package insert of each drug or therapeutic agent before administration. This advice is especially important when using new or infrequently used drugs.

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Rodney Maingot
(1893–1982)

Rodney Maingot was born in Trinidad. He spent his medical student days at St. Bartholomew's Hospital Medical School, London, and qualified in 1916. He joined the Royal Army Medical Corps and served as Captain in Egypt and Palestine, being twice mentioned in despatches. On demobilisation he returned to St. Bartholomew's to continue his surgical training and gained his FRCS in 1920. He was appointed to the Consultant staff of the Royal Free Hospital, London, which he served for many years with great distinction. In addition, he was Consultant Surgeon at the Southend General Hospital, where his Saturday clinics and operating sessions attracted visitors from all over the world. During World War II he served as Regional Consultant to the Emergency Medical Service.

Rodney Maingot's fame was as a surgical teacher and he was particularly interested in the abdominal cavity. Biliary surgery was his particular metière, but he also made great contributions to the surgery of hernia and was especially proud of his "Keel repair" for large incisional hernias. He lectured with distinction, and his clear, beautifully illustrated talks took him to many parts of the world. He was particularly well known and popular in the United States. His reputation was spread even more widely through his numerous textbooks, characterised by their clear writing, superb illustrations, meticulous production, and detailed, indeed encyclopaedic, knowledge.

Rodney was especially proud of his *Abdominal Operations*. The First Edition appeared in 1940; it boasted 1385 pages and, apart from short contributions by two internists (Dr. R.S. Johnson on postoperative chest complications and Dr. L.T. Bond on sternal puncture), the whole massive work was entirely the effort of this remarkable man. Some of the figures by Miss Pauline Larivière, a pupil of Max Brödel, live on today.

A Second Edition appeared in 1948. Now Rodney had collected eight contributors: five from the United Kingdom, two from the United States, and one from Australia. For the Third Edition, in 1955, there were now 24 contributors. Those from the United States included such famous names as Brunschwig, DeBakey, Cooley, Dragstedt, Harrington, and Pack. The United Kingdom contributors included two future Presidents of the Royal College of Surgeons—Russell Brock and Cecil Wakeley. The succeeding editions contained increasing numbers of contributors whose names formed a veritable *Who's Who* of international surgery. The Seventh Edition, published in 1980 when Rodney was in his ninth decade, found him still actively concerned with editing and writing this monumental work, as well as carrying out an extensive and personal correspondence with his numerous contributors all over the world.

The last few months of his life were passed in poor physical health but he remained in full mental vigour right until the end. Shortly before he died, I visited him with David Stires of Appleton-Century-Crofts. He fully realised that he would never live to see the Eighth Edition, nor indeed to have the strength even to undertake the task. The fact that *Abdominal Operations* was not only to continue but was to bear his name, gave him immense pleasure and satisfaction.

Maingot's introduction to the first edition had a first paragraph consisting of one sentence:

> This book is intended to present detailed consideration of the technique of modern abdominal operations.

This aim, we hope, lives on today.

Harold Ellis

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Preface

Under the editorship of Rodney Maingot, *Abdominal Operations* achieved, over a period of 45 years and seven editions, a pride of place in the libraries of general surgeons throughout the world. It was our privilege to take over as editors for the eighth edition, but rapid advances in abdominal surgery have made a new edition necessary after only four years.

In updating these volumes, we have recruited a number of new contributors replacing those who have retired or who are, sadly, no longer with us. Our new authors are all surgeons in active clinical practice who have been chosen because of their internationally acknowledged expertise in their specialized fields. The majority are drawn, once again, from the United States and the United Kingdom, but also represented are Australia, Holland, Hong Kong, Israel, South Africa, and Switzerland. We are indebted to our contributors, both old and new, for their splendid efforts.

The general format of the book remains unchanged. Descriptions of the techniques of the major abdominal operations within the repertoire of the general surgeon persist as the nucleus. However, we have not merely produced another surgical atlas: rather, we have attempted to synthesize a complete expression of the science and art of abdominal surgery. We have included concise accounts of modern diagnostic procedures, relevant pathologic anatomy, pre-operative assessment, indications for and choice of operation, post-operative care and complications and their management. The majority of the text and illustrations has been carefully revised. Where possible, overlap and repetition have been minimized. What had previously been considered as individual chapters have, wherever possible, been fused into broader topics with a cohesion that parallels the surgeon's interest. Indeed, the number of chapters have been reduced in this edition from 94 to 81.

This edition is directed to sophisticated students of surgery, whether in training or in practice where the learning process continues. As editors, we hope that we have satisfied the desires and needs of our audience.

Harold Ellis
Seymour I. Schwartz

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SECTION VIII

Appendix and Colon

42. Appendix

Harold Ellis

HISTORICAL NOTE*

The first appendicectomy was performed by Amyand, surgeon to Westminster and St. George's Hospitals and Sergeant Surgeon to George II. In 1736, he operated on a boy aged 11 years who had a right scrotal hernia accompanied by a fistula. Within the scrotum was found the appendix, perforated by a pin. The appendix was ligated and all or, more likely, part of it, removed, with recovery of the patient.

In 1755, Heister recognised that the appendix might be the site of acute primary inflammation. He described an autopsy on the body of a criminal who had been executed and wrote:

> When about to demonstrate the large bowel, I found the vermiform appendix of the caecum preternaturally black. As I was about to separate it, its membranes parted and discharged two to three spoonfuls of matter. It is probable that this person might have had some pain in the part.

In 1824, Loyer-Villermay gave a presentation to the Royal Academy of Medicine in Paris, entitled "Observations of Use in the Inflammatory Conditions of the Caecal Appendix," in which he described two examples of acute appendicitis leading to death. In both cases the appendix was found at autopsy to be black and gangrenous, whereas the caecum was scarcely involved. Three years later these observations were confirmed by Melier. Unfortunately, at this stage the pathologic picture became obscured. The writings of Husson and Dance in 1827, Goldbeck in 1830, and, most powerfully of all, Dupuytren in 1835 developed the concept of inflammation arising in the cellular tissue surrounding the caecum; it was Goldbeck who invented the term "perityphlitis," which did much to delay the progress of the understanding of this disease.

The first textbook to give a description of the symptoms that accompanied inflammation and perforation of the appendix was published by Bright and Addison in 1839. The terms "typhlitis" and "perityphlitis" remained in use until the end of the nineteenth century. It was Fitz, professor of medicine at Harvard, who in 1886 gave a lucid and logical description of the clinical features and described in detail the pathologic changes of the disease; he was also the first to use the term "appendicitis." He wrote:

> In most fatal cases of typhlitis, the caecum is intact whilst the appendix is ulcerated and perforated. The question should be entertained of immediate opening. If any good result is to arise from such treatment it must be applied early.

The evolution of the operative treatment of appendicitis proceeded significantly when Hancock in London successfully drained an appendix abscess in a female patient aged 30 who was in her eighth month of pregnancy. In 1848, he wrote:

> It may be premature to argue from the result of one case, but I trust that the time will come when this plan will be successfully employed in other cases of peritonitis terminating in effusion, which usually end fatally.

Parker of New York advocated earlier incision of appendix abscesses in 1867; after the publication of his paper, many similar accounts were published.

From the priority point of view, Shepherd showed that, in 1880, Tait of Birmingham operated on a patient with gangrenous appendicitis and removed the appendix, with recovery of the patient. Tait, however, did not record this case until 1890. Credit for the first published account of appendicectomy must go to Kronlein in 1886, although the patient, aged 17 years, died 2 days later. In 1887, Morton of Philadelphia successfully diagnosed and excised an acutely inflamed appendix lying within an abscess cavity. Two years later, McBurney in New York pioneered early diagnosis and early operative intervention and also devised the muscle-splitting incision named after him. Early intervention was still further popularised by the teaching of Murphy of Chicago. Both these surgeons pioneered the removal of the appendix before perforation had been allowed to take place.

It soon became evident that although the results of appendicectomy for the acutely inflamed unperforated appendix were satisfactory, the operative death rate for the later cases of perforated appendix with peritonitis was distressingly high. Ochsner in Chicago and Sherren at The London Hospital were both advocates in the early years of the twentieth century of conservative treatment in late cases. The discovery of antibiotics, fortunately, resolved the controversy between the schools of conservative and active surgery in such cases.

ANATOMY

The appendix arises from the posteromedial aspect of the caecum, about 2.5 cm below the ileocaecal valve. It is the only organ in the body that has no constant anatomic position; in fact, its only constant feature is its mode of origin from the caecum, where it arises from the site at which the three taeniae coli coalesce. It varies considerably in length, from 1 to 25 cm, but it averages 5 to 10 cm. The various positions

* For detailed accounts of the history of appendicitis, the reader is referred to the fascinating books by Sir Zachary Cope (1965) and Dr. Ralph Major (1944).

of the appendix are as follows: paracolic (the appendix lies in the sulcus on the outer side of the caecum), retrocaecal (the organ lies behind the caecum and may even be totally or partially extraperitoneal), preileal, postileal, promontoric (the tip of the organ points toward the promontory of the sacrum), pelvic (here the appendix dips into the pelvic cavity), and midinguinal (subcaecal). The retrocaecal position is the most common. Wakeley (1933), in an analysis of 10,000 cases at postmortem examination, gave the location of the appendix as follows: retrocaecal, 65.28 percent; pelvic, 31.01 percent; subcaecal, 2.26 percent; preileal, 1 percent; and right paracolic and postileal, 0.4 percent (Fig. 42–1).

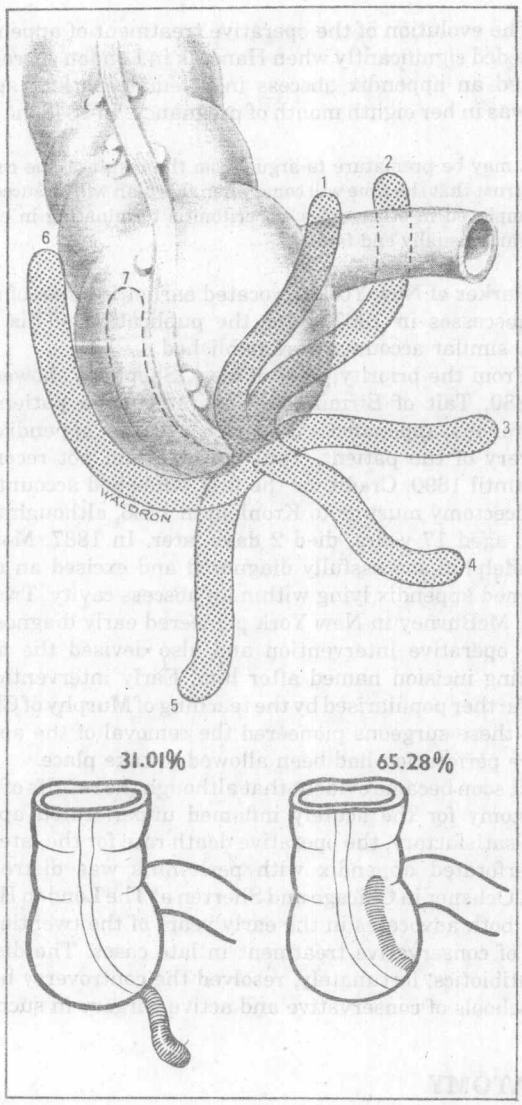

Figure 42–1. *Top:* Various positions that the appendix can occupy: (1) preileal; (2) postileal; (3) promontoric; (4) pelvic; (5) subcaecal; (6) paracolic or precaecal; (7) retrocaecal. *Bottom Left:* Location of the appendix in Wakeley's series of 10,000 cases (pelvic or descending position). *Bottom Right:* Location of the appendix in Wakeley's series of 10,000 cases (postcaecal and retrocaecal).

(*Source: Adapted from Wakeley C, 1933, with permission.*)

Williamson et al (1981) found that of 105 retrocaecal appendices removed at operation, 12 (11.4 percent) extended retroperitoneally. In this position the appendix may extend upward as far as the kidney, and indeed in 2 of these 12 cases, the patient experienced pain in the right flank.

The appendix may be situated in the left lower quadrant of the abdomen in cases of transposition of the viscera. Here the clue may be the observation that the patient has dextrocardia; I have correctly diagnosed and operated on such a case. A particularly long appendix may also extend over into the left side of the abdomen and, if inflamed, produce left iliac fossa pain. In cases of malrotation of the bowel, where the caecum fails to descend to its normal position, the appendix may be found in the epigastrium, abutting against the stomach or beneath the right lobe of the liver.

Robinson (1952), in reporting a case of congenital absence of the appendix, was able to collect only 68 other examples, a figure sufficiently indicative of the great rarity of this condition.

Duplication of the appendix, a subject well reviewed by Khanna (1983), is an anomaly of extreme rarity: fewer than 100 cases have been reported. Wallbridge (1962) classified duplication of the appendix into three types. Type A comprises partial duplication of the appendix on a single caecum. Type B has a single caecum with two completely separate appendices. This is further subdivided into type B1, which is also called "birdlike appendix" because of its resemblence to the normal arrangement in birds, where there are two appendices symmetrically placed on either side of the ileocaecal valve, and type B2, in which one appendix arises from the usual site on the caecum, with another, rudimentary appendix arising from the caecum along the line of one of the taeniae coli. In type C there are two caeca, each of which bears an appendix. Tinckler (1968) described a unique case of a triple appendix, associated with a double penis and ectopia vesicae.

Embryologically, the appendix is part of the caecum, of which it forms the distal end and which histologically it closely resembles, with the exception that it contains an excess of lymphoid tissue in the submucous layer. The mesentery of the appendix is contiguous with the lower leaf of the mesentery of the small intestine, and it passes behind the terminal ileum. The appendicular artery runs in the free border of the mesentery of the appendix and is a branch of the ileocolic artery (Fig. 42–2). This represents the entire arterial supply of the organ, and therefore thrombosis of this artery in acute appendicitis inevitably results in gangrene and subsequent perforation. This is in contrast to acute cholecystitis, in which the rich collateral blood supply of the gallbladder, from its bed in the liver, accounts for the comparative rarity of gangrene.

The veins from the appendix drain into the ileocolic vein, which in turn empties into the superior mesenteric vein. A variable number of slender lymphatic channels traverse the mesoappendix to empty into the ileocaecal nodes.

ACUTE APPENDICITIS

Incidence

Acute appendicitis is the most common cause of the "acute surgical abdomen" in the United Kingdom, but because notifi-

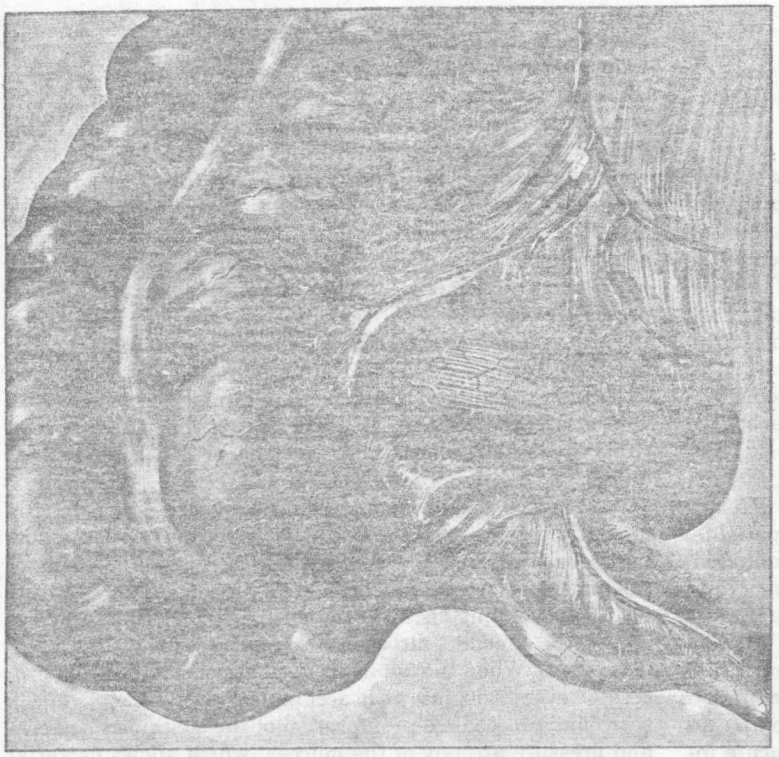

Figure 42–2. The appendix and its blood supply.

cation of the disease is not required, the exact incidence is not known.

The Hospital Inpatient Inquiry (Office of Population Censuses and Surveys, 1984), which estimates the total hospital inpatient discharges in England and Wales, gave the annual estimated total of acute appendicitis, based on a one tenth sample, as 50,115.

Pieper and Kager (1982), in a careful study from Sweden, estimated a yearly incidence of 1.33 cases of appendicitis per thousand of the male population and 0.99 per thousand of the female population (this difference is statistically significant, $P = .002$). In this study of 971 cases, the ages of the patients ranged from 1 to 89 years, with a median of 22 years. Twenty-five percent of the patients were younger than 14 years, and 75 percent younger than 33. Although these authors found no evidence of a fall in the incidence of acute appendicitis, other studies have indicated a steady decline in appendicitis and appendicectomy. Noer (1975) reported a decrease in the incidence of acute appendicitis from 1.3 per thousand to 0.5 per thousand over a 30-year period, from 1943 to 1972, in a study of a well-defined population in Norway.

Castleton et al (1959) reviewed 19 major hospitals in the United States and found that the total number of acute appendicectomies had dropped when 1941 figures were compared with those of 1956. This was not attributed to the increase in admittance at rural or community hospitals, since a study of 20 such hospitals also showed a decline in the number of appendicectomies. Palumbo (1959) also noted a decline in the number of cases of appendicitis at his Veterans Administration hospital from 1947 to 1958.

Such figures need cautious interpretation, because many studies do not differentiate between all cases of appendicectomy and those in which the diagnosis of acute appendicitis was confirmed.

Geographic Distribution

Appendicitis is most frequently observed in North America, the British Isles, Australia, and New Zealand and among white South Africans. It is rare in most of Asia, Central Africa, and among the Eskimos. When people from these areas migrate to the Western world or change to a Western diet, appendicitis becomes prevalent, suggesting that the distribution of this disease is determined environmentally rather than genetically. It is undoubtedly much more common among the meat-eating white races and relatively rare in those who habitually live on a bulk cellulose diet. Unexplained variations in incidence in various parts of England were found by Barker and Liggins (1981)—14.6 per 10,000 cases of acute appendicitis in the North compared with 10.4 and 10.6 in Central and Southern England, with no consistent socioeconomic variations. Again, these authors suggested that dietary differences might be contributory factors.

Many surgeons believe that there is a familial tendency in this disease that could be explained by an inherited malformation of the organ. However, the incidence of a large number of cases in the same family can equally be explained by the common nature of this disease. Andersson and colleagues (1979) compared 29 children between the ages of 5 and 15 years who had acute appendicitis with 29 control subjects. Twenty in the study group and four of the control subjects gave a history of appendicitis in parents or siblings.

Pathology

Cases of appendicitis are best classified as follows:

1. Acute appendicitis without perforation
2. Acute appendicitis with perforation
   a. With peritonitis
   b. With local abscess (appendix mass)

Acute appendicitis is not associated with any specific bacterial, viral, or protozoal invader. The bacteria in the inflamed organ are those of the normal bowel flora, suggesting secondary invasion of damaged tissue from the lumen of the bowel. A detailed study by Pieper and colleagues (1982) of the bacteria population of 50 inflamed appendices gave both aerobic and anaerobic isolates from all cases. Anaerobic bacteria were found more frequently than aerobic (141 versus 96 isolates). *Escherichia coli* was the most common aerobic bacterium (47 out of 50 patients). Ten patients also harboured other aerobic gram-negative rods, including *Klebsiella, Proteus,* and *Pseudomonas*. Enterococci (*Streptococcus faecalis* and *S. faecium*) were found in 15 patients, and other streptococci (*S. mitior, S. milleri,* and *S. salivarius*), in 21 patients. Of the anaerobic strains, *Bacteroides fragilis* predominated. Anaerobic gram-positive cocci were next in frequency, and *Clostridium perfringens* was cultivated from nine patients.

Examination of a series of fresh specimens of acutely inflamed appendices will show that types of inflammation fall into two groups. The first is a "catarrhal" inflammation of the whole organ, and the second is characterised by an obstruction of the appendix beyond which there is acute inflammation, distension with pus, and, in later cases, progression to gangrene and eventually perforation.

Catarrhal appendicitis is initially a mucosal and submucosal inflammation. In early cases, the appendix may appear normal externally or may merely show hyperaemia. When the appendix is slit open, however, the mucosa will be seen to be thickened, oedematous, and reddened; later it becomes studded with dark brown haemorrhagic infarcts, patches of grey-green gangrene, or small ulcers. Eventually the whole appendix becomes swollen and turgid, and the serosa becomes roughened, loses its healthy sheen, and becomes coated with a fibrinous exudate. The probable cause of this condition is bacterial invasion of the lymphoid tissue in the appendix wall, and indeed some cases are probably localised manifestations of a generalised enteritis. Because the lumen of the appendix is not obstructed, these cases rarely progress to gangrene, and in many instances the acute inflammatory attack will resolve spontaneously. In other cases, however, swelling of the lymphoid tissue in the appendix wall may lead to obstruction of the lumen and the condition may then proceed to obstructive appendicitis and gangrene. Even when the acute inflammatory process subsides, the appendix probably never regains its pristine state; adhesion formation and kinking of the appendix may lead to a final episode of acute obstructive appendicitis. It is interesting that an episode of gangrenous appendicitis may well be preceded by several milder and resolving attacks (Fig. 42–3).

Obstructive appendicitis is the dangerous type, for the appendix becomes a closed loop of bowel containing decomposing faecal matter. The changes after the sudden blocking of the lumen of the appendix depend on the amount and character of the content distal to the obstruction. If the lumen is empty, the appendix distends with mucus to form a mucocele (Fig. 42–4). When the appendix becomes obstructed, the process of events begins with accumulation of normal mucus secretion, proceeds to proliferation of the contained bacteria and pressure atrophy of the mucosa, which allows bacterial access to the deeper tissue planes, and continues with inflammation of the walls of the appendix with vessel thrombosis, which, because the blood supply is an end-artery system, leads inevitably to gangrene and then to perforation of the necrotic appendix wall. On other occasions, bacterial invasion occurs through pressure erosion of a contained faecolith, which may discharge into the peritoneal cavity through the perforation.

The relationship between obstruction of the appendix and gangrenous appendicitis was demonstrated in 1914 by

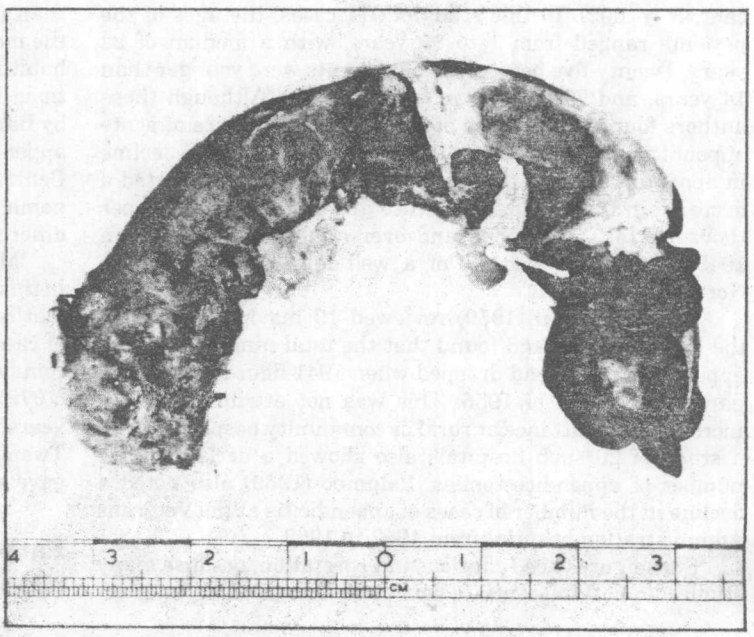

Figure 42–3. An acutely inflamed appendix; the distal half is gangrenous.

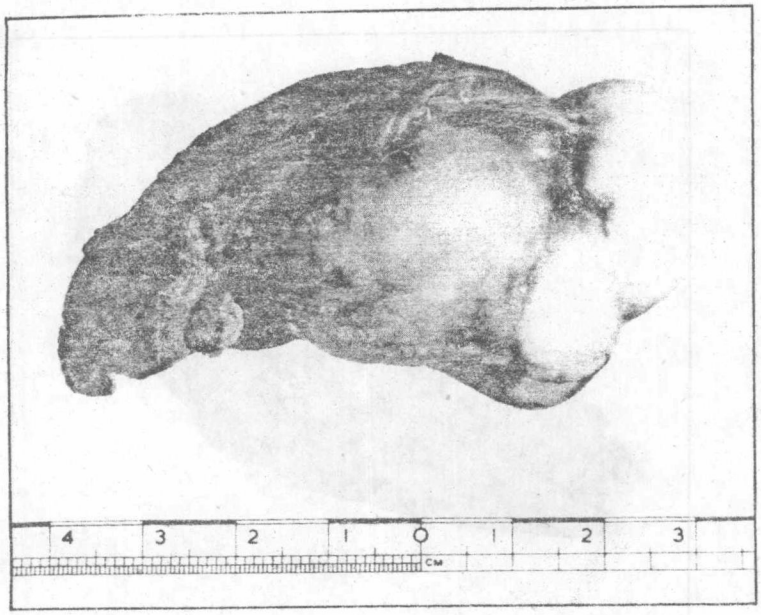

Figure 42–4. Mucocele of the appendix.

Wilkie, who showed that acute appendicitis followed ligation of the appendix in the rabbit. Wangensteen and colleagues documented in 1937 that combined obstruction and bacterial infection resulted in acute appendicitis, whereas, if the appendix was first washed free of faecal material and then ligated, a mucocele developed as mucus continued to be secreted within the bacteria-free lumen of the appendix. These classic studies were elegantly extended by Pieper et al (1982), who showed that obstruction of the appendix in the rabbit with the use of a balloon catheter introduced via a caecostomy resulted, in 12 hours, in the inflammatory changes that histologically were similar in all respects to appendicitis in man.

The obstruction may be due to a large number of possible causes. Inflammatory swelling of the lymphoid tissue in the appendix wall may, as we have noted, occlude the lumen. Kinks and adhesions may result from congenital bands or from previous episodes of inflammation. One or more faecoliths are commonly found within the appendix lumen in the normal organ; in about two thirds of all gangrenous appendices a faecolith is found firmly impacted at the junction between the uninflamed proximal and the gangrenous distal part of the appendix. Other foreign bodies, such as food debris, worms, or even a gallstone, have been found to obstruct the appendix lumen.

Perhaps the rarest cause of obstructive appendicitis is strangulation of the appendix within a hernial sac. Thomas et al (1982) reported seven such cases. The most common hernia to be involved is the right femoral, and then the right inguinal, but cases have been reported of an acutely inflamed appendix within a left inguinal, an umbilical, an incisional, and an obturator hernia. The usual diagnosis is, of course, a strangulated hernia, and the correct diagnosis virtually has never been made before operation.

Appendiceal Faecolith

Faecal material is commonly present in both the normal and the inflamed appendix, and this should be differentiated from the true faecolith, which is ovoid, about 1 to 2 cm in length, and faecal coloured. Unlike ordinary faeces, the true faecolith shows a well-ordered lamination in section. Shaw (1965) showed that the great majority of these faecoliths are radiopaque and in 10 percent of cases of acute appendicitis contain sufficient calcium to be demonstrated on a plain x-ray film of the abdomen. In a study of 240 cases of acute appendicitis, in which the appendix specimen was x-rayed, faecoliths were demonstrated in 33 percent of cases. When a faecolith was present, 77 percent of the specimens were gangrenous, compared with 42 percent when there was no evidence of a stone (Fig. 42–5).

Effects of Perforation

The appendix may rupture at any spot, but most frequently the site of perforation is along the antimesenteric border. After perforation, a localised abscess may form in the right iliac fossa or the pelvis, or diffuse peritonitis may ensue. Whether the peritonitis remains localised or becomes generalised depends on many factors, including the age of the patient, the virulence of the invading bacteria, the rate at which the inflammatory condition has progressed within the appendix, and the position of the appendix.

It is usually stated that poorer localisation of the infection occurs in infants because the omentum of the child is filmy and less able to form a protective sheath around the inflamed appendix. A more likely explanation is that delays in diagnosis are more prone to occur in infants. Similar delays occur in the management of elderly persons. In the nonobstructed type of acute appendicitis, the disease is comparatively limited in its course, and there is ample time for inflammatory peritoneal adhesions to form. In contrast, in the acute obstructive form the rapidity of the process gives little time for defensive adhesions to develop before the sudden flood of infected contents. An appendix situated in the retrocaecal or pelvic location is probably more likely to form a local abscess than one in the preileal or subcaecal position.