C J Mieny, U Mennen: Principles of Surgical Patient Care - Volume II

Chapter 10: Abdominal Emergencies

Chapter 10.1: Peritoninitis and Intra-Abdominal Abscess

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Intra-abdominal sepsis, including peritonitis and abscess formation, presents serious and potentially life-threatening events in the management of surgical patients with significant resultant morbidity and mortality. Twenty percent of patients presenting with generalized suppurative peritonitis and over 50% of high-risk patients with postoperative intra-abdominal sepsis die despite intensive treatment. Major recent advances have occurred in the conceptual understanding of the pathogenesis, diagnostic methodology, antibiotic therapy and application of non-invasive technology in intra-abdominal sepsis. As a consequence, the management of intra-abdominal infection requires a careful interdisciplinary approach, with close collaboration between surgeon, physician, radiologist, bacteriologist and anaesthetist.

This chapter outlines the relevant anatomy and physiology, pathogenesis, clinical features, diagnostic approach and management of primary and secondary bacterial peritonitis and intra-abdominal abscess.

Anatomy

The peritoneum is the larges serous membrane in the body and consists of a single layer of mesothelial cells supported on a connective tissue base. The parietal peritoneum lines the anterior, lateral and posterior abdominal walls, the undersurface of the diaphragm and the pelvis and is reinforced by transversalis fascia. The visceral peritoneum is reflected onto the intra-abdominal viscera, mesentery and omentum, creating a closed cavity except for the open end of the fallopian tubes. Although the parietal and visceral surfaces are part of the same membrane, the distinction is relevant with regard to differences in sensory innervation. The peritoneal cavity forms the potential space between the parietal and visceral layers and is divided into the general peritoneal cavity or greater sac, and the lesser sac which communicates via the foramen of Winslow. The anatomic surface area of the peritoneum in adults is about 1.7 m^2 and approximates the total body surface area.

The parietal peritoneum has a somatic afferent nerve supply and is sensitive to all forms of stimuli. Irritation of the parietal peritoneum produces sharp, well localized, discriminate pain with tenderness, involuntary guarding and rigidity of the abdominal muscles if the stimulus is sufficiently intense. The parietal peritoneum on the undersurface of the diaphragm is supplied centrally by the phrenic nerves and peripherally by lower intercostal nerves. Irritation of the peritoneum over the central portion of the diaphragm results in referred pain in the distribution of the cutaneous branches of the third, fourth and fifth cervical nerves over the shoulder region.

The visceral peritoneum receives afferent innervation only from the autonomic nervous system and is relatively insensitive to tactile, thermal and chemical stimuli.

Stimuli from the visceral peritoneum characteristically are poorly localized and are perceived as a dull pain. The visceral afferent nerves have no receptors to mediate pain and temperature, but are sensitive to bowel ischemia and distention or traction on the mesentery.

Physiology

The peritoneum consists of a single surface layer of mesothelial cells supported on a basement membrane and a deeper well-vascularized connective tissue layer containing collagen and elastic fibres, fat cells, reticulum cells and macrophages. The peritoneal cavity contains less than 50 mL of clear fluid consisting of water, electrolytes and solutes derived from interstitial fluid and plasma. Normal peritoneal fluid has a specific gravity of less than 1016 and less than 3 grams per mL of protein, predominantly albumen. Fibrinogen is not present and the fluid will not clot. Much of the peritoneal membrane acts as a passive, semi-permeable barrier to the bidirectional diffusion of water and most solutes. Normal peritoneal fluid contains less than 3000 cells per cubic mL with 50% lymphocytes, 40% macrophages, a few eosinophils, mast cells and occasional desquamated mesothelial cells. Bacteria are absent. Peritoneal fluid has minimal antibacterial activity, mediated both via the complement system and the lymphocyte population in the fluid. The number of granulocytes is significantly increased in the presence of inflammation. Peritoneal aspiration may be of value for culture and chemical analysis to facilitate the diagnosis of inflammatory conditions, tumours or intraperitoneal trauma. The principal route of absorption and clearance of fluids and particulate matter from the peritoneal cavity is by lymphatics. Reverse flow is prevented by one-way valves within the thoracic lymphatic system. Experimentally, particulate matter including red cells and bacteria, is recoverable from the thoracic lymph within 6 minutes and from the blood within 12 minutes after intraperitoneal injection. Absorption of fluid by the diaphragmatic lymphatics produces a cephalad flow of peritoneal fluid which is promoted by increased respiratory movement. The rapid peritoneal clearance of particulate matter and fluid, functions as the essential first line of defence following initial peritoneal contamination.

Definition

Peritonitis is the acute inflammatory response of the visceral and parietal peritoneum to bacterial, chemical, radiation or foreign body injury. The two major clinical categories of peritonitis are based on aetiology:

- **Primary peritonitis** is an infection of the peritoneum occurring de novo without obvious intra-abdominal pathology.

- **Secondary bacterial peritonitis** is a purulent inflammation of the peritoneum due to contamination following a complication of a pre-existing primary intra-abdominal process such as perforated peptic ulcer, ruptured appendix or a disrupted anastomotic suture line.

Secondary bacterial peritonitis is more common and of greater surgical significance than primary peritonitis; accurate differentiation between the two types is crucial for optimal management.

Primary Peritonitis in Children

Primary peritonitis is uncommon, accounting for less than 1% of all cases of peritonitis and predominantly affects girls under 10 years. The bacteria involved are primarily pneumococci and Group A streptococci. Less commonly, gram-negative bacilli and Group B streptococci are found. A useful diagnostic feature in differentiating the usually monobacterial spontaneous peritonitis from the polymicrobial flora of secondary peritonitis is the single species of bacteria cultured. About one half of cases of pneumococcal peritonitis occur in children with nephrosis. The causative bacteria gain access to the peritoneum by a haematogenous route or, uncommonly, via the fallopian tubes.

The clinical manifestations are those of acute diffuse peritonitis. A pre-existing upper respiratory infection, pneumonia or otitis media may mask the initial onset of peritonitis. Severe, generalized abdominal pain is the predominant symptom. High fever, often with chills, irritability, vomiting and diarrhoea, is common. On examination, the child appears ill, pyrexial with a temperature of 40 °C and an elevated pulse rate. The abdomen is diffusely tender and ascites with shifting dullness may be demonstrated. Bowel sounds are hypoactive or absent. The white blood count reveals a leucocytosis of 20000-40000/mm^3 with a polymorphonuclear predominance and a shift to the left. Heavy albuminuria is present in cases superimposed on nephrosis. Blood culture is usually positive for streptococci or pneumococci. A peritoneal aspirate under local anaesthesia with an immediate gram stain is important to differentiate primary peritonitis in which surgical intervention is contra-indicated from peritonitis secondary to a perforated appendix where prompt surgery is necessary. If only cocci are seen on the smear, conservative therapy is appropriate. A mixed bacterial flora on the smear demands an exploratory laparotomy.

Primary Peritonitis in Adults

Spontaneous bacterial peritonitis (SBP) is an infective complication caused by enteric organisms and usually occurs in alcoholic cirrhotic patients with ascites. Characteristically, the patient presents with fever, abdominal pain, reduced or absent bowel sounds and encephalopathy in the presence of gross ascites. The presentation may be subclinical and subtle and the only clinical manifestation in a known cirrhotic may be unexplained deterioration in liver function, progressive encephalopathy or diminishing renal function. A diagnostic ascitic tap is necessary to determine the presence of bacteria, neutrophils, protein, glucose and pH of the ascitic fluid. Gram stains are positive, however, only in one-third of culture proven cases. Careful aerobic and anaerobic cultures isolate a single organism in 80% of patients with SBP; the most frequent organisms recovered are *E. coli* and pneumococci. Anaerobes are virtually never isolated. The ascitic fluid WBC is generally > 500/mm^3 with more than 75 neutrophils/mm^3. A peritoneal acidosis with a pH of less than 7.3 is due to lactic acid production by the infecting organisms. While the bacterial infection may be effectively treated with suitable antibiotics, the prognosis is poor and mortality exceeds 80% due to progressive liver decompensation, renal failure, encephalopathy and haemorrhage.

Secondary Bacterial Peritonitis

Secondary bacterial peritonitis is an acute suppurative inflammatory process of the peritoneal cavity arising as a consequence of:

- primary disease of the abdominal viscera
- penetrating or blunt trauma
- previous intra-abdominal surgery.

Aetiology

Secondary peritonitis may complicate almost any abdominal condition including inflammatory, traumatic, obstructive or neoplastic processes (table 10.1.1). Perforation following appendicitis or diverticulitis is the most common cause of acute suppurative bacterial peritonitis. Delayed treatment of duodenal and gastric perforations are further common causes of bacterial peritonitis. Gangrene of the bowel, either from strangulation or ischaemia, remains an important cause of peritonitis. Pelvic peritonitis often accompanies pelvic inflammatory disease, especially if infection of the fallopian tubes is severe, but seldom progresses to frank generalized peritonitis. Penetrating or blunt abdominal trauma may cause contamination of the peritoneal cavity and subsequent widespread inflammation. Some degree of inflammation follows every surgical procedure within the peritoneal cavity. Due to inevitable minor contamination from room air, poor surgical technique, glove perforation or spillage during resection or manipulation. Such contamination is nearly always contained by the protective mechanisms within the peritoneal cavity, but gross contamination, virulent organisms or immune compromise may result in clinically significant peritonitis.

Pathogenesis

The initial local responses of the peritoneum to contamination or injury are vascular dilatation, hyperaemia and increased capillary permeability, followed by transudation of fluid from the vascular and interstitial spaces. Fluid containing opsonins, polymorphonuclear leucocytes and macrophages accumulates in the involved tissues and the free peritoneal space. Phagocytosis of bacteria and foreign material is the major local peritoneal defence mechanism and the peritoneal exudate contains antibodies and complement which enhance aggregation, adherence and engulfment of micro-organisms, in addition to promoting chemotaxis and diapedes of white cells. The peritoneum loses its normal sheen and acquires a dull ground-glass appearance due to deposition of fibrinogen. The release of tissue thromboplastin converts fibrinogen to fibrin. Normal peritoneum, however, has a fibrinolytic capacity due to plasminogen activator present in mesothelial cells. Injury to the peritoneum produces an inhibition in fibrinolytic activity which permits the development of fibrinous adhesions that wall off and surround the inflamed area or injury. This abundant fibroblastic exudate aids specific humoral and cellular immune defence mechanisms in localizing the inflammatory process. Further restriction of the inflammatory process is due to peritoneal and omental tissue adaptation with adhesions between loops of bowel and the parietal wall with migration of the greater omentum into the area of inflammation to further compartmentalize the infective process. The regional lymphatic circulation provides an important peritoneal defence mechanism by absorption of bacteria through the lymphatic system present beneath the diaphragmatic mesothelium.

Bacteria removed via the lymphatics are filtered by the thoracic lymph nodes or, once in the systemic circulation, are eliminated by the reticulo-endothelial cells in the liver and spleen.

Table 10.1.1. Aetiology of Secondary Bacterial Peritonitis

1. Inflammatory

1. **Inflammatory**
a. *Intestine*
   - Diverticulitis with perforation
   - Appendicits
   - Meckel's diverticulitis
   - Necrotizing enterocolitis
   - Ulcerative colitis
   - Amoebic colitis
b. *Visceral*
   - Salpingitis
   - Empyema of gallbladder
   - Necrotizing pancreatitis
   - Liver abscess
2. **Strangulation obstruction**
a. *Small bowel*
   - Closed-loop adhesive obstruction
   - Hernia: internal, external
   - Volvulus with strangulation

   - Intussusception
b. *Colon*
   - Volvulus: sigmoid, caecal
   - Closed loop obstruction (neoplasm, diverticulum)
3. **Trauma**
a. *Blunt rupture of viscus: stomach, small bowel, colon*
b. *Penetrating*
c. *Iatrogenic*
   - endoscopy/biopsy
   - anastomotic disruption
4. **Perforation**
a. *Neplasms: gastric, colon carcinoma*
b. *Foreign body*
c. *Duodenal, gastic ulcer*
5. **Vascular**
a. *Mesenteric embolus*
b. *Ischaemic colitis.*

If the peritoneal defences are able to contain the inflammatory process, the disease may end by resolution. A second possible outcome is localization of the process with contained suppuration and abscess formation. If the peritoneal and systemic defence

mechanisms are unable to localize the inflammation, generalized suppurative peritonitis occurs. The factors favouring spread of the inflammatory process are:

--> overwhelming or continued bacterial contamination
--> virulence and synergistic bacterial action
--> presence of foreign bodies or adjuvant factors deleterious to resolution of the inflammation.

Continuing contamination of the peritoneal cavity occurring with free anterior perforation of a duodenal ulcer or perforation secondary to an obstructive colonic carcinoma results in rapid dissemination of bacteria throughout the peritoneal cavity. The volume and rate of spill are critical factors. Intra-abdominal spread of contamination is promoted by the peristaltic movement of the viscera and by diaphragmatic motion producing a cyclic pressure differential between the subphrenic space and the peritoneal cavity. The subphrenic area is contaminated by movement of bacteria into the potential space by the negative suction of the diaphragm during respiration. In addition, the effect of gravity in the recumber patient allows fluid to drain into the most dependent spaces.

A mixed bacterial flora is usually found in acute suppurative peritonitis after secondary contamination from the intestinal tract. The most common organisms are aerobic coliforms, in particular *E. coli, Proteus, Klebsiella,* and aerobes including *Bacteroids,* anaerobic cocci and *Clostridia* (table 10.1.2).

Table 10.1.2. Bacteriology of Secondary Peritonitis

Aerobic Bacteria

--> E. col	60
--> Streptococci	28
--> Enterobacter/Klebsiella	26
--> Proteus	22
--> Enterococci	17
--> Pseudomonas	8
--> Staphylococci	7
--> Candida	2

Anaerobic Bacteria

--> Bacteroides	72
--> B. fragilis	38
--> Eubacteria	34
--> Clostridia	17
--> Peptostreptococcus	14
--> Peptococci	11.

All these bacteria are potentially lethal pathogens in the human. The mixed aerobic-anaerobic faecal flora commonly found in peritonitis frequently acts synergistically. Particular combinations such as *E. coli* and *B. fragilis* produce a mortality rate significantly greater than the mortality rate of each organism alone. The mortality rate

resulting from peritonitis is in part a dose-dependent phenomenon correlating directly directly with the number of pathogenic bacteria introduced into the peritoneal cavity. In duodenal perforations, few bacteria are cultured initially, but bacterial proliferation increases significantly after six hours. Perforation of obstructed distal small bowel or colon, however, results in immediate gross contamination. The site of the gastrointestinal perforation and, by inference, the number of bacteria escaping from the lumen are therefore critical determinants in the risk of subsequent infection. Not all organisms cultured from the intraperitoneal fluid, however, have equivalent virulence. *E. coli* and *B. fragilis* are regarded as the major pathogens in intraabdominal sepsis since they are found most often in positive blood cultures.

Certain adjuvant substances increase the lethality of peritonitis, ie, the presence of necrotic tissue in the peritoneal cavity significantly enhances the risk of continuing infection. The virulence of contaminating micro-organisms is further enhanced by mucus, bile, barium sulphate and haemoglobin. This characteristic is attributed to the detrimental effect of the adjuvant on defence mechanisms, especially phagocytosis. The co-existence of haemoglobin and bacterial inoculum within the peritoneal cavity produces an increased susceptibility to the micro-organisms, since clearance of bacteria from the peritoneal cavity is inhibited by haemoglobin. Haemoglobin specifically inhibits intraperitoneal chemotaxis of neutrophils, phagocytosis and intracellular killing of bacteria. Haemoglobin also interferes with lymphatic absorption of bacteria and exerts and additional local toxic effect after degradation.

Pathophysiology

Peritonitis produces profound physiological alterations both locally within the abdominal cavity and also systemically affecting the cardiovascular, respiratory, renal and neuro-endocrine system.

Fluid Shifts

The peritoneum reacts to inflammation by vascular dilatation, hyperaemia and exudation of fluid from the vascular space into the free peritoneal cavity, the loose connective tissues beneath the mesothelium of viscera and mesenteries as oedema fluid and into the lumen of the atonic dilated gastrointestinal tract. This translocation of water, electrolytes and protein into the sequestrated third space effectively removes fluid from the body economy. The rate of fluid loss is proportional to the surface area of peritoneum involved in the inflammatory process and with extensive peritonitis, may reach 4-6 L in 24 hours.

Ileus

Generalized peritonitis produces an inhibition of intestinal motility with resultant dynamic ileus. Distention of the bowel with unabsorbed fluid and gas, aggravated by relative mural ischaemia if intraluminal pressure exceeds capillary perfusion pressure, permits bacteria to penetrate the mucosal barrier and enter the vascular compartment.

Endocrine Response

Peritonitis results in a major systemic stress producing a vigorous response from the pituitary-adrenal axis. Adrenal medullary secretion of catecholamines is in large part responsible for the vasoconstriction, tachycardia and sweating accompanying the initial response to peritonitis. Aldosterone secretion increases as a response to hypovolaemia and further aggravates potassium loss and sodium retention. Release of antidiuretic hormone results in renal conservation of water which may exceed sodium retention with consequent dilutional hypotonicity of plasma sodium.

Cardiovascular System

Loss of extracellular fluid volume depletes central venous return, lowering cardiac output and increasing heart rate. Compensatory vasoconstriction results in an increased total peripheral resistance to maintain blood pressure and cardiac and cerebral perfusion pressures. Decreased perfusion and oxygenation to the splanchnic bed, kidneys and inactive muscles result in anaerobic glycolysis with progressive accumulation in lactic acid.

Metabolic acidosis is aggravated by decreased renal clearance, secondary to reduced renal perfusion. If acidosis progresses, depression of cardiac contractility further decreases cardiac output.

Respiratory System

Demands on the respiratory system increase significantly in peritonitis, with a decrease in total respiratory capacity. Abdominal distention secondary to ileus causes an elevation and restriction of diaphragmatic movement. Respiratory restriction, fatigue and inefficient respiratory effort diminish ventilatory volume with ensuing atelectasis which may progress to ventilation-perfusion imbalance, intrapulmonary arteriovenous shunting and peripheral hypoxaemia.

Kidneys

Renal changes induced by peritonitis are primarily a reflection of hypovolaemia, reduced cardiac output and increased secretion of aldosterone and antidiuretic hormone. Renal blood flow, glomerular filtration and urine volume are reduced. Aldosterone promotes sodium retention and antidiuretic hormone results in increased reabsorption of water from the distal tubules with a further decrease in urine output.

Protein catabolism progresses during the duration of peritonitis and serum albument concentrations decrease with further loss into the peritoneal cavity. Hepatic glycogen stores are depleted. The net effect of these metabolic changes in the body results in a significant energy deficit.

Clinical Features

The predominant symptom in peritonitis is abdominal pain aggravated by any movement, including respiration. Perforation of a peptic ulcer with massive contamination

of gastric contents produces sudden onset of epigastric pain with rapid spread to involve the entire abdomen corresponding to spread of acid-pepsin throughout the peritoneal cavity. The spread of pain from lesions such as a perforated appendix or a diverticular abscess is more gradual corresponding to progression of bacterial inflammation. Anorexia, nausea and vomiting commonly accompany peritonitis. Patients may complain of feeling feverish, sometimes with rigors, and of thirst and decreased urine output. Other symptoms of adynamic ileus produced by the peritonitis are the inability to pass faeces or flatus and abdominal distention.

Physical Findings

Patients characteristically lie quietly in bed with the knees flexed and frequently shallow respiration. With advancing infection, respiration becomes more rapid as the demand for tissue oxygen utilization increases and acidosis worsens. Respiration is primarily intercostal since diaphragmatic movement worsens pain. Body temperature elevation in peritonitis is usually 39 °C to 40 °C but may reach 42 °C in overwhelming sepsis. Subnormal temperatures are seen in the early stages of chemical peritonitis and late in septic shock, where progressive peripheral hypothermia is a grave sign. Immunosuppressed patients do not show a marked febrile response; fever is usually higher and more spiking in character in younger, healthier patients than in the elderly. Increasing tachycardia with weak peripheral pulses reflects the circulatory effects of both hypovolaemia and toxaemia. With progression of peritonitis, blood pressure may be significantly reduced due to septic shock.

Abdominal examination elicits generalized tenderness with reflex guarding of the adominal muscles. Extensive abdominal hyper-resonance due to gaseous bowel distention may be demonstrated by percussion. Pneumo-peritoneum from a ruptured hollow viscus may produce decreased liver dullness. Bowel sounds are audible on auscultation in the initial stages of peritonitis, but with spread of inflammation the silen abdomen of adynamic ileus supervenes. Rectal and vaginal examinations are an essential part of the diagnostic procedure and must not be omitted in evaluating the patient with signs of peritonitis. Anterior tenderness on pelvic examination, or the presence of a mass in the rectouterine and rectovesical pouch may provide an important clue to pelvic sepsis. Palpation of the cervix with excitation tenderness suggests the presence of inflammation in the uterus and adnexae, while examination of material on the glove may give a clue to the etiology of the process.

Diagnostic Studies

A leucocytosis between 15000 and 20000 per mm^3 is usual in peritonitis with the differential count showing a polymorphonuclear predominance and shift to the left. Blood chemistry shows variable degrees of haemoconcentration and dehydration. Acidosis, both metabolic and respiratory, is present in severe and late cases. Urinalysis serves to exclude infection of the urinary tract as a source of abdominal pain. An increased serum amylase may indicate pancreatitis or a surgically correctable cause of hyperamylasemia including perforated duodenal ulcer or strangulated small bowel.

The X-ray findings of peritonitis may be non-specific with features of a paralytic ileus with distention of both small intestine and colon. Inflammatory exudate and oedema

of the bowel wall produces widening of the space between adjacent bowel loops and peritoneal fat lines and psoas shadows are obliterated. Free air may be visible beneath the right diaphragm and occasionally beneath the left hemidiaphragm. An intra-abdominal abscess may be detected by extraluminal airfluid levels or by the mottled soap-bubble appearance of an anaerobic abscess. In advanced cases with bowel necrosis, intramural gas may be seen in the intestinal wall. Portal vein gas is a late sign of bowel infarction in adults and occurs in infants with necrotising enterocolitis.

Management of Bacterial Peritonitis

Every patient with acute peritonitis is potentially critically ill and requires the rapid institution of physiological monitoring and aggressive treatment. The essential elements of management in sequence are fluid resuscitation, antibiotic administration, decompression of the gastrointestinal tract and, when appropriate, exploratory laparotomy. The patient with established peritonitis requires repeated systematic clinical evaluation with frequent determination of blood pressure, pulse, central venous pressure, and urine output with specific gravity. In older patients and in younger patients with cardiovascular compromise, a Swan-Ganz pulmonary artery balloon catheter should be inserted to determine pulmonary artery and wedged pulmonary artery pressures. A Foley catheter is essential to determine hourly urine output and a nasogastric tube should be inserted for decompression of the stomach to avoid vomiting and further progression of intestinal distention. Laboratory tests should be obtained for haematocrit, blood count, electrolytes, urea and creatinine, amylase and liver function. Respiratory function is assessed with repeated arterial blood gas determination, while in critically ill patients, a radial arterial line provides access for arterial samples for blood gas analysis and a constant mean arterial pressure record.

Hypoxaemia is a frequent complication in diffuse peritonitis due to increased permeability of the pulmonary vasculature with transudation of fluid into alveoli and decreased oxygen exchange and is treated by increasing inspired oxygen using a 40% ventimask and assessing response with regular blood gas analysis. Endotracheal intubation and positive pressure respiration with a volume-controlled pressure respiration are indicated if the patient remains hypoxic.

A critical caveat in the treatment of peritonitis is the frequent underestimation of prior fluid loss. Replacement with crystalloid solutions should take into account:

--> maintenance fluid requirements
--> ongoing loss via the nasogastric tube
--> prior fluid loss including dehydration from prolonged vomiting
--> third-space loss into the peritoneal cavity and bowel lumen.

Elevation of the serum haematocrit and blood urea are useful indicators of the degree of dehydration and third-space loss. The primary goals of fluid management are the maintenance of a normal blood pressure and the establishment of an adequate urinary output approaching 30 mL per hour. Rapid initial fluid replacement should include crystalloid supplemented by colloid colutions and is monitored continuously against central venous and pulmonary artery wedge pressures. If the haematocrit is low in the septic patient, blood transfusion is an appropriate addition to crystalloids, both for replacement and correction of anaemia.

Parenteral antibiotic administration should commence before surgery and should anticipate both aerobic and anaerobic flora with provision for the full bacteriological spectrum. The parenteral route is important to ensure adequate antibiotic tissue levels during surgery. The aim of antibiotic therapy is to reduce both the systemic and local infectious complications of peritonitis. The best current results are achieved by initial empiric triple antibiotic therapy designed to eliminate the three major groups of commonly isolated bacteria including coliforms, enterococci and anaerobes. Aminoglycosides are bactericidal to most facultative gram-negative enteric organisms and are indicated in peritonitis caused by these bacteria. Plasmid-mediated resistance to aminoglycosides is common among gram-negative organisms and the therapeutic agent may require changing in persistent or recurrent infections. Aminoglycosides are excreted in the urine and patients with impaired renal function require reduced dosages to prevent renal and ototoxicity. The volume of distribution and rate of excretion is variable in individual patients and dosage requirements should be based on trough and peak levels to minimize complications after intravenous administration. In patients with significant renal impairment, third-generation cephalosporins provide an effective substitute for aminoglycosides. Most anaerobes, including *Bacteroides* species, are resistant to aminoglycosides. Metronidazole is the antianaerobic agent of choice. Enterococcus is a frequent isolate in peritonitis and is an important synergistic partner with anaerobes in experimental peritonitis. Enterococcus is usually sensitive to ampicillin and is advisable treatment in patients with secondary bacterial peritonitis. No precise criteria exist for the length of antibiotic therapy in intraperitoneal sepsis and a period of seven to ten days is usually adequate while clinical improvement continues. Breakthrough bacteraemia during administration of appropriate antibiotics is presumptive evidence of residual or recurrent infection, provided nosocomial infection has been excluded.

Surgical Treatment

Operative management of bacterial peritonitis is directed at the control of the source of contamination by closure or resection of the area of perforation, removal of the bacterial inoculum by suction or lavage, debridement of grossly necrotic tissue and prevention of recurrence of sepsis. For generalized peritonitis, a midline incision provides optimal access to all quadrants of the abdomen. A specimen of peritoneal fluid should be obtained for aerobic and anaerobic culture. All purulent matrerial and blood should be evacuated from the peritoneal cavity once the source of contamination has been controlled. Careful separation of loops of matted bowel may be uncover unsuspected intermesenteric abscesses. In generalized peritonitis, the sub-diaphragmatic, subhepatic and lateral peritoneal gutters and pelvis should be irrigated with warm saline. All gross foreign material including necrotic tissue, faecal residue, bloor or bile should be sucked out after irrigation since the virulence of peritoneal infections is enhanced by the presence of adjuvant foreign substances. In local abscesses such as contained appendix abscess, generalized peritoneal irrigation is unnecessary and undesirable after complete evacuation and drainage of the abscess cavity.

An alternative approach which has emerged is radical peritoneal debridement as a primary method of treatment in severe generalized peritonitis during which the entire peritoneal cavity is meticulously debrided of fibrin strands, blood clot and purulent membrane, followed by thorough irrigation with saline until the effluent is clear. While impressive results have been presented in patients treated with radical peritoneal

debridement, randomized clinical data have found no advantage using this technique over standard surgical treatment. Local intra-operative peritoneal irrigation with saline or antibiotic instillation have been used in several forms. Although peritoneal irrigation may spread bacteria throughout the peritoneal cavity, lavage does reduce the number of bacteria present, as well as diminishing the concentration of adjuvant substances. However, the mortality and infective complications of experimental peritonitis are not significantly reduced by saline irrigation alone. Residual saline left in the peritoneal cavity following irrigation dilutes bacterial opsonins and suspends bacteria in a fluid medium, decreasing phagocytosis and permitting bacterial proliferation. All residular fluid should therefore be aspirated upon completion, prior to abdominal closure. The use of intra-operative irrigation with antibiotic solution has, in a randomized study shown reduction only in infective complications without a reduction in mortality. Other studies have, however, been unable to show any advantage of intra-operative antibiotic irrigation over saline irrigation in patients with peritonitis who in addition received systemic antibiotics which achieve intraperitoneal levels comparable to serum levels. Patients with massive peritoneal contamination or with depressed host defences may benefit form further post-operative peritoneal lavage.

Drainage of the free peritoneal cavity in peritonitis is seldom effective since standard drains are rapidly isolated and sealed by omentum, exudate and loops of intestine. Drains may also act as a foreign body potentiating intraperitoneal infection and allowing external bacteria to enter the peritoneal cavity. Drains are effective only when their purpose is to evacuate an abscess cavity, establish controlled fistulae or offer a preferential drainage pathway after extensive surgery. Triple lumen sump drains which have inlets for both air and irrigation fluid, and a shielded, fenestrated outlet to which suction can be applied, provide more efficient drainage than corrugated or vacuum drains. Sump drains are therefore preferred when large, dependent cavities require drainage.

Controlled open drainage leaving the peritoneum open, without closing the abdominal wall, has been advocated as a technique in selected patients with gross contamination and abdominal wall defects. In this situation, polypropylene mesh (Marlex) is used to protect the bowel and prevent evisceration. Closure of the abdominal wound after surgery for peritonitis is best accomplished by approximating only fascia as a single layer mass closure using either monofilament synthetic suture such as Nylon or Prolene, or an absorbable polyglycolic acid (Dexon) suture. With gross contamination, the skin and subcutaneous tissues should be left open and packed with a fine mesh saline-soaked gauze. The gauze dressings are changed daily and, when a clean wound with granulation tissues is evident, usually within four to five days, the wound is closed with sterile adhesive strips. In selected cases, the use of retention sutures may be appropriate to avoid wound dehiscence and evisceration.

The ultimate outcome of secondary peritonitis depends on the patient's age, previous nutritional status, concomitant disease states including immunosuppression, and the source of sepsis. Important management factors determining prognosis include:

> --> avoidance of unnecessary delay before operative therapy
> --> the correct choice and administration of antibiotics
> --> nutritional and haemodynamic support.

Continued peritoneal contamination, septicaemia, fluid and electrolyte abnormalities and respiratory failure are the principal cause of death during the first week. Later causes are renal and hepatic failure. Stress ulceration, upper GIT bleeding and intestinal obstruction are further late causes of morbidity, in addition to being a mortality factor.

Intraperitoneal Abscess

Intraperitoneal abscesses may be either solitary or multiple and develop when a localized purulent fluid collection becomes walled off by the host tissue reaction in one of the potential spaces of the peritoneal cavity or between adjacent loops of bowel.

Etiology

Intra-abdominal abscess formation is due to:

- effective localization of the primary pathology such as an appendiceal or diverticular abscess

- a sequel or complication following generalized peritonitis

- intraperitoneal contamination secondary to external trauma or complicating previous surgery.

The four major intraperitoneal anatomic spaces in which abscesses commonly localize are:

- the right and left subphrenic spaces and the lesser sac

- the subhepatic space or Morrison's pouch

- the inermesenteric area, including the paracolic gutters and interloop areas

- the pelvis.

The increased tendency for peritonitis to resolve with the formation of a pelvic or subphrenic abscess reflects the anatomy of the peritoneal cavity with preferential drainage either into the pelvis by gravity or into the subphrenic spaces due to diaphragmatic movement.

Clinical Symptoms and Signs

Symptoms and signs may be variable due to the anatomy of the intraperitoneal spaces. Fever is the most common symptom and is intermittent at first becoming progressively higher and persistent with maturation of the abscess. Although localized sepsis is generally associated with a swinging temperature, the presence of fever is more significant than the pattern of the pyrexia. Fever may be absent in very young, elderly, malnourished, uraemic or immunocompromised patients. Fever may also be absent in patients with localized infections on antibiotics which suppress but do not eradicate infection. When antibiotics are discontinued, fever may recur. Overwhelming septicaemia

may present without pyrexia or with hypothermia. Rigors and temperature peaks above 39 °C are due to transient bacteraemia. Abdominal tenderness, pain and a palpable mass may be present only when the parietal peritoneum forms a portion of the abscess wall. Peritoneal signs with intermesenteric, subphrenic and pelvic abscesses are frequently absent. Although abdominal pain may occur, it is seldom well localized. Paralytic ileus is a frequent accompaniment and it may be localized or diffuse. Symptoms of nausea, vomiting and diarrhoea may be present. Pelvic abscesses may be heralded by tenesmus or urinary frequency and a tender mass may be palpable on rectal or vaginal examination. In the postoperative patient, persistent ileus, fever, leucocytosis, a falling platelet count and deteriorating liver function with low-grade hyperbilirubinaemia or pulmonary and renal insufficiency should initiate a persistent search for a hidden abscess. In patients receiving hyperalimentation, the development of persistent hyperglycaemia or acidosis may be due to an unsuspected abscess. The only indirect clinical findings with a subphrenic abscess may be pulmonary changes including a pleural effusion, elevation of the diaphragm and diminished basal breath sounds.

Diagnosis and Special Investigations

A leucocytosis greater than 15000 per mm^3 accompanied by a shift to the left with an elevated ESR is invariably present. Blood cultures may detect bacteraemia and identify enteric gram-negative bacilli, anaerobes or enterococci which are the organisms most commonly involved. Plain abdominal X-rays may show a soft-tissue mass, localized gas bubbles or an extraluminal air-fluid level. The chest X-ray provides indirect evidence of a subphrenic abscess with unilateral elevation and splinting of the diaphragm, pleural effusion and persistent basal atelectasis. Barium studies of the gastrointestinal tract may show organ displacement due to fluid collections or a spiculated mucosal pattern characteristic of surrounding inflammation and occasionally a sinus tract or extravasation of contrast from the bowel.

Radionuclide scintigraphy using gallium or indium are two isotopes potentially useful for imaging nonvisceral inflammatory processes. Gallium binds to lactoferrin, a protein present in neutrophils, after an intravenous injection, and accumulates at an inflammatory focus. Indium is lipophilic and binds to leucocyte cell protein. Indium-labelled cells migrate to an infective site where the isotope can be detected by a gamma camera. While both isotope studies may be applicable in selected patients, their non-specific and limited anatomic information has been superseded by more specific and anatomically accurate radiologic evaluation.

Ultrasonography and computerized tomography are currently the most useful investigations for the identification and localization of intraperitoneal abscesses. Ultrasonography offers a rapid, non-invasive method of detecting intra-abdominal abscesses without the risk of radiation exposure. Considerable operator skill is required for effective use of the technique in identifying small abscesses. The major technical limitations of ultrasound examination are poor penetration through bone and distended gas-filled bowel making evaluation of deeper structures inadequate. Potential practical scanning difficulties which may limit the skin area required for adequate transducer contact include large open wounds, recent incisions, bulky dressings, stomas and external appliances. The majority of abscesses appear as well-defined echo-free cavities that readily transmit ultrasound waves, although some abscesses have internal echoes which represent

debris within the cavity. Although real-time ultrasound scanning allows visualization of peristalsis and vascular pulsation within the abdomen, fluid-filled masses including abscess cavities, haematomas and loculated ascites may be difficult to differentiate. A logical extension now in general use is percutaneous ultrasound-guided aspiration of intraperitoneal collections with a fine 22-gauge needle allowing fluid to be retrieved for diagnosis and culture.

Computerized tomography (CT) currently provides the highest resolution with accurate anatomic localization and is the most useful modality for demonstrating multiple collections within the abdominal cavity. Oral contrast material, given prior to the procedure may facilitate the differentiation of fluid-filled intestinal loops from abscesses and other fluid collections. Intravenous contrast may be added to produce rim enhancement of a mass which is a characteristic feature of an abscess. A technical limitation of CT is the inability to fully differentiate haematoma from an abscess since the densities may be similar. In addition, a reduction in quality of the CT image may be produced by metallic surgical clips in the postoperative patient. Despite the use of a battery of diagnostic tests, the occasional patient with evidence of sepsis, persistent abdominal pain, leucocytosis and fever may not reveal the site, and in such patients, exploratory laparotomy still serves an important role.

Treatment

The initial resuscitation of patients with intraperitoneal abscess is similar to patients with diffuse peritonitis. Diagnosis and treatment may be delayed because of subtle clinical manifestations. Overt septicaemia, nutritional deficiency, fistulae and extra-abdominal complications including respiratory or renal failure may be present and will require specific attention.

Antibiotic therapy should cover the same spectrum as generalized peritonitis and should include both aerobic and anaerobic organisms.

The operative management of intraperitoneal abscesses requires:

--> effective drainage of the abscess
--> correction of the underlying cause when appropriate
--> careful exploration to exclude other abscesses within the peritoneal cavity.

In patients with a single accessible well-localized abscess, simple drainage through an extraperitoneal approach may be adequate. Localized appendiceal abscesses may be drained through a flank incision without contaminating the peritoneal cavity. Well-localized pelvic abscesses can be drained through the vagina and rectum. A transperitoneal approach should be used for patients who have multiple abscesses and require effective evacuation of all septic material, debridement of necrotic tissue and closure with drainage at the site of a gastrointestinal leak. The operation is best performed through a midline incision similar to diffuse peritonitis. The choice between the small extraserous operation and the larger transperitoneal procedure requires individualization and careful clinical judgment and no specific rules can be made.

The advent of percutaneous CT-aspiration and drainage has modified the various operative considerations: this technique, however, requires careful selection of suitable patients and definite localization by imaging of the abscesses. A drainage tract that will not transverse adjacent bowel is important. The precautions in selecting patients for percutaneous drainage are:

--> the presence of single non-loculated abscess cavity
--> a drainage route that does not traverse bowel, uncontaminated viscera, sterile peritoneum or pleura
--> no perforated viscus is present
--> highly viscous pus or thick necrotic debris is not present.

Percutaneous abscess drainage can be employed in critically ill patients as a temporising measure permitting definitive surgical treatment at a later stage. Despite the failures and complications which include fistulae, bacteraemia, bleeding and peritonitis, percutaneous drainage of abscesses has become an important addition to the therapeutic armamentarium of interperitoneal abscess in selected patients.

Despite recent advances in surgery and intensive care, mortality in patients with intra-abdominal sepsis remains distressingly high. Assessment of the variety of published treatment regimens is hampered by the uncontrolled nature of many of the studies. Objective validated scoring systems such as the sepsis score, the APACHE II Score and the surgical infection stratification system have been devised to aid audit and comparison between groups of patients.

The ultimate prognosis in patients with intraperitoneal abscesses is influenced by risk factors which include the presence of multiple, recurrent or lesser sac abscesses, positive blood cultures, multiple organ failure and age over 60 years.

Comment

Peritonitis and Intra-Abdominal Abscess

G A G Decker

In this comprehensive review of peritonitis and intra-abdominal abscess it is important to stress the need for early laparotomy in secondary bacterial peritonitis. Resuscitation should be done expeditiously while arrangements are being made to take the patient to the operating room. Modern anaesthesia has made surgery possible in these critically ill patients and any unnecessary delay must be avoided at all cost.

I still find it useful to note the colour and smell of the intraperitoneal fluid in secondary bacterial peritonitis. Green colourless fluid is usually indicative of a foregut or upper small-bowel perforation. Purulent fluid which smells faeculent because of the presence of anaerobic bacteria is indicative of an appendix abscess or colonic perforation.

Over the years I have adopted a policy of doing a laparotomy on most patients who have been transferred from other hospitals with abdominal complications requiring urgent treatment following a previous laparotomy. The decision to perform a second

laparotomy requires surgical judgement. Timely intervention can often interrupt the cascade of multiple organ failure. In the majority of cases one finds a localized collection of pus or a leaking suture line. Under no circumstances must an attempt be made to perform a second anastomosis if there is generalized peritonitis. The cut end to the bowel proximal and distal to the fistula must be exteriorized as an ostomy and mucus fistula respectively. Total parenteral nutrition, dedicated nursing and the dilligent attention of the stoma therapist has made it possible to keep patients alive even with a proximal jejunostomy. The continuity of the bowel can be re-established weeks later when the peritoneal cavity is sterile and the abdominal wound has healed.

One of the most appealing aspects of the open method of treatment of the septic abdomen is that it makes a virtue of a necessity in those patients in whom closure of the abdomen is technically impossible or in whom the respiration would be compromised by closing the abdomen under tension. The "sandwich technique" has been a major advance in the open management. Subsequent to the publication of this technique it has been improved by placing pieces of stoma adhesive on the skin right up to the edge of the abdominal wall defect. This protects the skin and also makes a more effective seal with the Opsite drape. It is not always necessary to suture the Marlex mesh to the edge of the fascia. If a larger piece of mesh is used the free edge can be tucked in between the abdominal wall and the underlying loops of the bowel. Vicryl mesh which is absorbable can be used if a second-look laparotomy is not being contemplated.

The problem of spontaneous bacterial peritonitis (SBP) and ascites has been reviewed by Reynolds. SBP is diagnosed if the ascitic fluid contains more than 250 polymorphonuclear (PMN) cells per mm^3. Antibiotic therapy should be commenced without waiting for the results of a positive culture. An elevated PMN count is not diagnostic of bacterial peritonitis in ascites due to cancer, tuberculosis or pancreatic disease. Ascitic fluid pH less than 7.35 and a lactate content more than 25 mg/dL have been considered superior to the PMN count in terms of specificity (defined as the proportion of patients without bacterial peritonitis on culture who had a negative test). This concept can be invalidated if it is possible to have SBP with elevated PMN count in the ascitic fluid but negative cultures. In the latter condition of neutrocytic ascites the elevated PMN count falls on repeat paracentesis when patients have been treated with antibiotics, which suggests that infection was present.

An ommission in the list of causes of secondary peritonitis is peritonitis due to perforation of a hollow muscular viscus occurring in renal failure patients receiving chronic peritoneal dialysis. Peritonitis should be diagnosed if two of the following criteria are present:

- abdominal tenderness
- cloudy dialysate with more than 100 polymorphonuclear leucocytes per mm^3
- micro-organisms in the peritoneal fluid.

If peritonitis is diagnosed management consists of continuing the peritoneal dialysis and adding heparin 500 IU/L and antibiotics to the dialysate. A cephalosporin alone or with an aminoglycoside is recommended. Treatment with antibiotics should continue for seven days beyond the first negative culture of the dialysate.

Comment

Peritonitis and Intra-Abdominal Abscess

P C Bauling

The indirect comparison by the authors of the peritoneal surface being of almost the same size as the body (skin) surface area needs to be underlined as this really brings home the potential extent and magnitude of the third-space fluid losses that can occur in a patient with acute suppurative peritonitis. Adequate fluid replenishment of volume contraction should be stressed. This is the key to the lowering of subsequent complications and organ failure. Remember the domino effect of organ failure so often mentioned in the past literature. Oliguria in a complex, critically ill patient remains the most reliable indicator of hypovolaemia regardless of the mean arterial pressure, central venous pressure and pulmonary wedge pressure readings (MAP, CWP, PWP).

The fact that macrophage migration is inhibited by excessive fluid in the peritoneal cavity, that "macrophages cannot swim" is relevant to the common practice of intra-operative irrigation of the peritoneal cavity at the conclusion of a procedure just prior to the closure. In my opinion it is important to leave the peritoneal cavity as dry as possible by suctioning and swabbing all fluid from the peritoneal cavity that can possibly be removed. This may be a preventative measure in avoiding subsequent peritonitis and abscess formation.

I make a plea for the application of the objective APACHE II scoring system to all septic cases and the need for prospective studes based on the APACHE system. All the answers to the different management options of peritoneal sepsis in severely ill patients are definitely not available yet. Whether open method, planned relaparotomy or continuous irrigation should be used, remains the choice of the individual surgeon. An interesting and opposing argument is put forward by Polk and Kinney.

The value of gallium scintigraphy in the detection of localized intra-abdominal sepsis is to be seriously questioned for the following reasons:

- The isotope is extremely expensive, approximately R400 per patient.

- The isotope is not available from the Atomic Energy Board on short notice; only on individual order and then several days later.

- The investigation may achieve some degree of accuracy only at 24-48 hours after gallium injection. Thus, the overall delay between deciding to do the investigation and obtaining an answer from the clinical nuclear physicist may be somewhere between five and seven days. Finally the specificity and accuracy of the investigation is not of the order to seriously challenge more rapidly available investigations such as CT scan or ultrasound.

Chpater 10.2: Obstruction of Hollow Muscular Viscera

D Pantanowitz

The term colic is described in the *Oxford dictionary* as "severe griping pain in the belly". It is actually derived from the greek word *kolikos* which means relating to the colon.

Colic develops from obstruction to the following involuntary or smooth muscular tubes:

--> the gut (small bowel, large bowel)
--> the urinary system (ureter)
--> the genital system (uterine tubes, uterus).

In spite of what is written in much of the surgical literature, biliary colic and renal colic are not by definition colicky pains, they are continuous pains. Only obstruction to smooth muscle tubes gives rise to colic, whereas obstruction to smooth muscle receptacles, ie, gall bladder, renal pelvis, urinary bladder, causes a continuous pain. The common bile duct contains virtually no muscle and obstruction to this tube also causes a continuous pain due to distension of the system.

With regard to the stomach which can be considered to be consisting of a sump (fundus) and a pump (antrum), pyloric obstruction is generally of a chronic nature (chronic duodenal ulcer or an annular constricting antral carcinoma) with no associated colic. Nevertheless, acute irritation of the stomach, ie, food poisoning from ingestion of the staphylococcal exotoxin, can cause violent gastric spasmodic contractions which may result in almost intolerable colic which is relieved by vomiting. True colic is accompanied by marked restlessness, occurs in paroxysms lasting a variable time and the abdominal wall is soft between the attacks of pain; in contrast to the patient with peritonitis who lies still, has continuous pain and in whom the abdomen remains rigid.

Colic (true or atypical) is a visceral pain, which is elicited by muscle spasm (true) or sudden luminal distension (atypical), proximal to the obstruction. This is a poorly localized, deep-seated and diffuse pain which may be vaguely localized to the organ from which it arises, or is referred to the dermatome supplied by the posterior root through which the autonomic (visceral) afferent impulses reach the spinal cord (convergence). Thus pain originating in the foregut (spinal segments T7-T9) is referred to the epigastrium. The reason for this is that because the mental image of the entire body is largely that of its surface (the growing child experiences touching and visualizing its skin), the visceral pain is referred to the appropriate dermatome because of the confluence or convergence of the two afferents into the same spinal neuron. Because the gut and its appendages are embryologically midline structures with bilateral innervation, the referred pain is felt in the midline. Pain originating in the midgut (T9-T11) is referred to the umbilical region, whereas hindgut pain (T11-L1) is felt in the hypogastrium.

Similarly, biliary pain may be vaguely localized to the right hypochondrium or is referred to the epigastrium. Renal pain is felt in the loin while ureteric pain is felt in the

loin radiating to the corresponding groin, this pain being unilateral as the urinary system has a unilateral innervation.

True colic originates in the gut. This is a gripping or cramping pain mounting to a crescendo which then remits and disappears completely. It is due to excessive muscle contraction proximal to the mechanical obstruction. This intestinal pain is better exemplified in the small bowel than the large bowel. True colic also occurs in the genital system, the uterine contractions of labour being a good example. The severity of the pain is directly proportional to the violence of the smooth muscle contraction, which is produced in an effort to overcome the obstruction.

Atypical colic is exemplified by biliary "colic". This term is a misnomer, because the pain here does not remit or disappear, ie, it is a continuous pain. The reason is that the smooth muscle in the biliary system is homologous to the muscularis mucosa of the gut, and thus has very limited contractile ability. There is no counterpart to the main muscle coats of the gut (which produce peristaltic movements) in the biliary system. The gall bladder is a receptacle rather than a propulsive structure, although it can contract weakly in response to cholecystokinin. In the common bile duct, the smooth muscle is virtually non-existent (and the orientation of the muscle cells that are present appear to be aligned longitudinally rather than transversely) thus virtually making this duct a purely passive conduit. Nevertheless, biliary pain may be exceedingly severe causing the patient to writhe in agony, and this is due to distension of the system proximal to the obstruction. This distension or stretching is responsible for the continuous pain.

Pain following obstruction to the flow of urine depends upon the site of the obstruction. Renal pain due to pelvi-ureteric obstruction and bladder pain due to acute bladder outlet obstruction are continuous pains (again the receptacle concept). Ureteric obstruction seems to have elements of both true colic (spasm) and atypical colic (distension). Thus ureteric colic is a continuous underlying pain (distension proximally) punctuated by exacerbations (smooth muscle contraction) but not true remission as in intestinal colic. The ureter does have a definite smooth muscle coat capable of contraction and peristalsis, in contrast to the biliary system.

The above discussion on the pathophysiology of certain types of visceral abdominal pain will now be followed by a detailed presentation on the aetiology and sequelae of obstruction to hollow muscular organs such as the bowel, biliary and urinary systems. Finally, certain important principles relating to acute and chronic intestinal obstruction will be emphasized.

Obstruction to Hollow Muscular Viscera

Aetiology

1. Lumen
    - Stones, foreign bodies, parasites, ie, *Ascaris lumbricoides.*
2. Wall
    - *Functional spasm,* failure of relaxation, segment of paralytic ileus
    - *Organic* congenital (atresia), traumatic (operative), inflammatory (diverticulitis, Crohn's disease, TB), ischaemic, neoplastic
3. Outside
    - *Compression* adhesions
    - *Displacements* hernia, torsion (volvulus), intussusception.

Sequelae

This is dependent on whether the obstruction is acute or chronic and is considered under the headings - proximal to, at the site of, and distal to the obstruction.

Acute (Complete) Obstruction

1. Proximal

--> Accumulation of contents

Abdominal pain; if bowel involved increased bowel sounds (borborygmi); reflex vomiting; progressive proximal distension which eventually paralyses the muscle wall resulting in disappearance of the colic (note that the gall bladder will not distend when there is a stone in the common bile duct due to the presence of fibrosis from prior inflammation).

--> Regurgitation of contents

Vomiting (bowel); jaundice (biliary).

--> Organ failure (back pressure)

Liver failure (secretory function ceases); renal insufficiency (excretory function ceases).

--> Secondary infection

This is a common complication and if this does not occur, and the epithelium is mucus secreting, ie, the gall bladder, a mucocele will form.

2. Site (depends on cause)

--> Stone

Haemorrhage, perforation, stricture.

--> Adhesion (fibrous band)

Pressure necrosis with perforation.

--> Strangulation

As the pressure of the compression force occludes the veins, congestion supervenes which is followed by arterial occlusion and gangrene.

3. Distal

--> Peristalsis continues

Empties organ distally.

Chronic (Incomplete) Obstruction

1. Proximal

--> Pain

Absent.

--> Compensatory hypertrophy

Thickened wall musculature; increased peristalsis; increased sensitivity to distension with further increased peristalsis

- *stomach* vomiting initially after big meals only, but later following all meals

- *urinary bladder* frequency of micturition, first noticeable nocturnally.

--> Failure dilatation

Hypertrophied muscle eventually fails resulting in accumulation of contents

- *stomach* frequency of vomiting decreases, but now vomits food eaten the previous day (succussion splash present)

- *urinary bladder* enlarged bladder with overflow incontinence

- *colon* constipation

--> Secondary infection

Fermentation and liquefaction of contents with consequent irritation of the wall of the viscus.

- *stomach* gastritis with oedema which aggravates the obstruction and damages the mucosa thus decreasing acid production

- *urinary bladder* cystitis with increasing frequency and ascending infection

- *colon* stimulates mucosa to secrete mucus resulting in alternating bouts of constipation and diarrhoea.

2. Site (depends on cause)

--> Stone

Haemorrhage or stricture.

--> Neoplasm

Annular constriction which obstructs early while the fungating type obstructs late.

Acute on Chronic Obstruction

The chronic obstruction (incomplete) becomes acute (complete) when solid material suddenly obstructs the narrowed lumen.

Intestinal Obstruction (IO)

It is important to remember that this is a surgical disease requiring operative intervention as a general rule. There are certain well-defined exceptions to this rule:

--> patient mecially unfit for laparotomy - the surgeon feels that the risk of operating is more than the risk of treating the obstruction conservatively (rare)

--> where laparotomy is likely to be unsuccessful such as in diffuse carcinomatosis of the peritoneal cavity

--> in patients who have had numerous previous operations for adhesive obstruction (a reasonable figure seems to be at least three)

--> faecal impaction

--> early postoperative IO where patient has not regained normal bowel function after the laparotomy - this is often due to a combination of flimsy soft fibrinous adhesions and weak peristaltic activity

--> where conservative treatment is effective in treating the IO such as a rectal tube for sigmoid volvulus and antihelminthic therapy for *Ascaris lumbricoides* (round worm)

--> where during preparation of the patient for laparotomy, the patient unexpectedly decompresses by passing flatus.

Mechanical IO may be classified into a number of categories:

--> acute (complete, often involving small bowel), chronic (incomplete, often involving large bowel) and acute on chronic

--> simple or strangulated

--> single site or closed loop obstruction.

The term *ileus* is used by some to include mechanical IO, but it should preferably only be used to refer to intestinal paralysis (paralytic or adynamic ileus). Ileus may be classified into local (short segment ileus which may itself be the obstacle in a mechanical IO) or general. The aetiology of *paralytic ileus* is best divided into intra- and extra-abdominal causes.

Intra-abdominal (local or general) Ileus

--> Intraperitoneal
- accidental or operative trauma
- chemical peritonitis (bile, urine, pancreatic juice)
--> Extraperitoneal
- excessively distended bowel above a mechanical IO
- progressive hollow visceral myopathy
- traumatic retroperitoneal haematoma (fractured spine)
- retroperitoneal visceral disease (pancreatitis, ureteric stone)
--> Vascular
- visceral arterial occlusive disease
- mesenteric venous thrombosis

Extra-abdominal (general ileus)

--> Metabolic
- toxic (uraemia, cholaemia)
- biochemical (K+, Na+, Mg++, Ca++ DEPLETION)
- hypoxaemia
- diabetic coma
- porphyria
--> Neurogenic
- reflex sympathetic overactivity (fractured spine)
--> Drugs
- ganglion-blockers.

Ileus must be actively prevented and treated by removing the cause and treating the patient with intravenous fluids and nasogastric (NG) suction. Neglect of an ileus may have undesirable consequences for the patient such as raised intra-abdominal pressure from bowel distension and continued vomiting with its metabolic sequelae.

Normal bowel movements consist of mixing (low-pitched sounds of short duration due to isolated contractions) and propulsive (low-pitched sounds of prolonged duration due to peristaltic waves) activity. The typical tinkling sounds of distended loops of bowel filled with fluid and gas that are found in a paralytic ileus are due to movement of these loops caused mainly by the piston action of the diaphragm. This movement results in an overflow of fluid from one loop into another (dripping effect causes a tinkling sound).

In patients with resistant ileus where excessive sympathetic activity may be an underlying cause, a regimen to reverse sympathetic bowel inhibition by alpha and beta adrenergic blockade and to augment parasympathetic motor drive by cholinergic stimulation, ie, the Neely-Catchpole regimen, may be reqired in additon to the usual measures.

A variety of local ileus, usually referred to as pseudo-obstruction or colonic ileus, is an important condition to consider as it presents as a large-bowel obstruction. The cause of this pseudo-obstruction may be:

--> Ogilvie's syndrome (carcinomatous infiltration of the retroperitoneum)
--> fractured spine (reflex sympathetic overactivity)
--> electrolyte imbalance (particularly low K+ and Mg++)
--> hypoxaemia (obstructive or restrictive pulmonary disease)
--> uraemia.

Mechanical IO can be excluded by contrast enema or colonoscopy; the latter route may also be used to decompress the colon. Treatment is again to eliminate the cause of this variety of megacolon. Surgical intervention is indicated only if caecal tenderness develops or if the proximal colon dilates up to >8 cm, simply requiring caecal exteriorization.

Acute Mechanical IO

Here again the surgical approach will be considered - proximal to, at the site of and distal to the obstruction.

Proximal

The small bowel distends with gas and fluid. The gas consists of approximately 70% swallowed air (mainly nitrogen, some oxygen and CO_2), 20% diffusion from blood into the gut (again mainly nitrogen, some oxygen and CO_2) and 10% from bacterial fermentation (methane, hydrogen disulphide and CO_2). Large quantities of isotonic fluid are lost from the interstitial space and the intravascular compartment into the lumen, because of a disruption to the bidirectional flux of salt and water, with the nett effect largely into the lumen. The intraluminal pressure begins to rise and this:

--> impair venous drainage, resulting in oedema of the wall and fluid loss from the serosal surface into the peritoneal cavity and accentuates the loss into the lumen

--> may increase the susceptibility to perforation, as the tension is directly proportional to the diameter, for any given intraluminal pressure (law of Laplace).

There is also decreased removal of fluid and gas as onward propulsion is prevented and thus colonic reabsorption ceases. Another factor of importance is the multiplication of bacteria (coliforms, anaerobes such as clostridia and bacteroides, and faecal streptococci) which accelerate the fermentation process with the production of faeculent material (brown and foul-smelling small-bowel content that is not faeces). There is no absorption of bacterial toxins is simple IO as viable bowel wall is impermeable to the toxic molecules.

The clinical manifestations and metabolic efects of IO depend upon the site and the duration of the block. Proximal small-bowel IO causes earlier and more vomiting with less distension. Large losses of Na+, K+, H_2O, HCl occur producing dehydration, hypovolaemia, hyponatraemia with a hypokalaemic alkalosis. The vomitus initially consists of food (gastric contents) and finally faeculent material (small-bowel contents). Clinically there will be colic with increased borborygmi, these being less pronounced the higher the IO. Thus in summary a high small-bowel IO is more of a problem of fluid and electrolyte loss than of distension.

Distal

Distal small-bowel IO is associated with less vomiting and more distension. The colic is typical, with intermittent crescendo pain usually lasting a minute or two, with total pain-free interval. The diagnosis should be made early when there is colic plus increased borborygmi. Distension, vomiting and constipation are late features when the diagnosis is all too obvious and the patient is already compromised. The distension is central abdominal and on auscultation of the abdomen, the heart sounds are clearly audible. As the distension increases, the pain alters from colic to a constant visceral pain as the venous circulation becomes compromised, followed by arterial circulatory insufficiency as the intra-luminal pressure rises further. This will lead to necrosis and wet gangrene on the antimesenteric border of the gut with concomitant perforation. Radiographs of small-bowel IO show distended loops on the supine plate (applies especially to the jejunum where the "stacked-coin" or "step-ladder" appearance is seen and is due to the valvulae conniventes running transversely right across the bowel) and fluid levels on the erect plate. As the distension becomes more severe, the bowel wall muscle becomes paralysed and the colic disappears (silent abdomen) which must not be mistaken for a primary ileus.

In **colonic IO** vomiting is most unusual. Again colic may be present, but not the classical colic of a low small-bowel IO. The distension is more peripheral than central, and gross distension may cause elevation of the diaphragm with respiratory insufficiency and compression of the vena cava with concomitant diminished cardiac filling and cardiogenic shock. Here radiographs show dilated colon (the natural markings do not run right across the bowel as do the valvulae conniventes).

Closed-Loop IO

This occurs where there is more than one site of obstruction and it is seen typically in large bowel IO where the ileocaecal valve is competent. As the massively distended colon cannot decompress proximally into the ileum, the caecum bears the brunt of the distension, causing pain and tenderness in the right iliac fossa. As the intraluminal pressure continues to rise, the venous, followed by the arterial circulation, becomes compromised, leading to necrosis, gangrene and perforation of the caecum.

Site

In simple IO a foreign body or a band adhesion may cause direct pressure necrosis and perforation. But the main problem at the site of obstruction is strangulation, where external pressure constricts the venous circulation which in turn compromies the arterial input, again resulting in necrosis, gangrene and perforation. This is an emergency and the diagnosis of strangulation is made by noting the following features:

--> change in the character of the pain (persistent local pain is superimposed onto the colic because the peritoneum becomes irritated by the strangulated loop) and for the same reason local guarding and tenderness develop

--> blood found in vomitus, NG aspirate and stool

--> palpable mass due to engorged bowel wall

--> exudation of blood into the peritoneal cavity causing a bloody ascites and an ileus (silen abdomen)

--> the compromised bowel results in a pyrexia, tachycardia and leucocytosis

--> hypovolaemic shock due to blood loss into lumen, wall and peritoneal cavity, and this shock is out of proportion to the fluid loss.

It should be noted that strangulation is more common in closed-loop IO. A sustained increase in intraluminal pressure will result in strangulation without an external pressure source such as unyielding hernial rings and adhesive bands or the deformity or twisting of the mesentery as in volvulus or intussusception. Where the strangulated loop of bowel is not lying free in the peritoneal cavity (external strangulated hernia), local signs of peritonitis are absent.

There is additional lethal element to strangulation and that is the presence of bacteria, necrotic material and blood within the affected loop, which now becomes permeable to the luminal toxic material which is consequently absorbed causing toxaemia and septic shock.

Finally, there is a special variety of strangulation without prior IO, and that is where the blood supply of the gut is compromised due either to visceral arterial block or mesenteric venous thrombosis. The diagnosis can be made only by the presence of severe abdominal pain, usually without any abdominal physical signs a marked leucocytosis and a metabolic acidosis.

Distal

The bowel empties beyond the obstruction initially, to be followed by absolute constipation.

Chronic (Incomplete) Mechanical IO

Whereas acute IO is most commonly due to adhesive bands and external hernia, chronic IO is usually due to a neoplasm of the large bowel slowly occluding the lumen. In chronic IO the colic is vague (more a discomfort than true pain) and there is marked muscle hypertrophy proximally, resulting in a thickened bowel wall with increased peristalsis (visible peristalsis with audible prolonged low-pitched sounds or borborygmi). When the hypertrophy fails to compensate for the incomplete obstruction, the dilatation increases and secondary infection supervenes in the distended colon. The consequent fermentation results in irritation of the mucosa with excessive mucus secretion and liguefaction of the colonic contents. The constipation then alters to an alternating constipation (few days) and diarrhoea (few hours), and finally complete constipation supervenes.

Principles of Management

Conservative measures help to control the "internal environment" of the body, but cannot correct the cause of the disease which requires surgical treatment.

Conservative Treatment

The two components are decompression of the distended proximal bowel and correction of the body fluid and electrolyte imbalance. Decompression is achieved with simple NG suction in order to deflate the distension which:

- facilitates the subsequent surgery

- prevents aspiration pneumonia

- allows the diaphragm to descend which improves respiratory function and venous return to the heart.

If the gastric contents are too thick for NG suction, a stomach tube should be passed after the anaesthetist has placed a cuffed tube to protect the airway.

The fluid and electrolyte deficit is corrected by using replacement solutions for the losses and maintenance solutions for normal daily requirements. A simple "rule of thumb" is to administer 2 L for simple IO, 4 L when the patient is clinically dehydrated and 6 L when shock supervenes (at least half the requirements given as normal saline). Obviously the fine adjustment is dictated by the patient's clinical condition, renal function and urine output, CVP monitoring and blood biochemical parameters. Particular care must be taken when replacing K+ which should never exceed 40 mmol/L and 40 mmol/hour, with a urine output of at least 1 mL/minute.

Operative Treatment

The principles of management are as follows:

--> Examine the caecum initially - if it is collapsed the obstruction is in the small bowel, whereas if it is distended the obstruction is in the colon.

--> Care must be taken not to handle the distended colon too vigorously as it will rupture, the surgeon should preferably locate the distal colon (often collapsed) and proceed proximally to locate the obstruction.

--> Treat the cause of the IO, ie, adhesive bands, hernia, foreign body, obstructing colon carcinoma, etc.

--> If strangulation is diagnosed, operation becomes urgent and following a short period of intensive resuscitation (about an hour), the cause of strangulation must be relieved. This may allow a compromised region of bowel to recover and thus avoid a resection. If the viability of the bowel is doubtful, it should be placed in a warm saline pack for about 10 minutes and then inspected for mesenteric pulsation, motility and colour. If still in doubt a qualitative fluorescein test can be carried out: 1 gm of fluorescein is injected into a peripheral vein over 1 minute and the bowel insprected under UV light - the presence of fluoresceine indicates that the blood supply to the bowel is satisfactory and that it is viable. A Döpler probe applied to the surface of the bowel may also indicate a return of intestinal blood flow, but this has not proved to be reliable. If still unsure, a "second-look" procedure should be undertaken the following day.

--> With a colonic closed-loop obstruction, the caecum must be decompressed surgically (caecal exteriorization and not a tube caecostomy) to prevent caecal rupture and a faecal peritonitis. The colon obstruction (usually a carcinoma) can be removed at the same time or subsequently.

--> Closure can be facilitated by deflating massive distension using either a Miller-Abbott type tube or a needle placed carefully to avoid peritoneal contamination (the needle hole can be oversewn if necessary).

Conclusions

The following important points require reemphasis:

--> True colic (gut) is due to proximal smooth-muscle contraction; atypical colic (biliary, renal) presents as a continuous pain and is due to proximal distension; ureteric colic is a combination of both.

--> Generally obstructed tubes cause colic, while obstructed receptacles (bladders) cause a continuous pain (the exception is the bile duct which is a tube that contains very little muscle and thus distends proximally to cause a continuous pain.

--> IO is a surgical disease with a mortality of about 5% (simple IO).

--> Strangulation carries a high mortality (about 20%) and must be promptly treated.

--> If a patient with an adhesive IO suddenly improves on conservative treatment (passage of flatus, scaphoid abdomen, disappearance of colic), operation is no longer necessary. The NG suction presumably allows the kink in the bowel at the point fixed by adhesions to straighten out.

--> The higher the IO the less the distension, but the worse the fluid and electrolyte deficit. The lower the IO the greater the distension, but the dehydration is less. Thus the urgency for rehydration is greater in the former, while the urgency for operation is greater in the latter.

--> Must differentiate between paralytic ileus and mechanical IO. This is easier if there is a total or generalized ileus (small and large bowel filled with air, shown on radiographs in a patient with a silent abdomen) than if there is a localized ileus (the paralysed segment acts as the mechanical IO, and the diagnosis may be made only at laparotomy).

--> The condition of pseudo-obstruction or colonic ileus must be considered in all cases of large-bowel IO.

--> Finally, to diagnose IO early, the clinical features are colic with increased bowel sounds (distension, vomiting and constipation appear later in the course of the disease).

Comment

Obstruction of Hollow Muscular Viscera

C J Mieny

Although I agree with the emphasis placed on the dangers of strangulation in mechanical intestinal obstruction there are selected cases in whom non-operative treatment may be indicated. Certainly, patients with partial obstruction, early postoperative ileus obstruction, recurrent adhesive obstruction or obstruction secondary to metastatic intra-abdominal malignant disease may warrant initial attempts at non-operative treatment with intestinal decompression and intravenous fluids.

Most surgeons advocate early surgery for small bowel intestinal obstruction and regard a trial of tube decompression only as a source of delay in surgical treatment which results in higher mortality and morbidity. Those who stress an early surgical approach report a high incidence of strangulation when compared with those advocating a conservative approach. Several reports have pointed out that early recognition of obstructed bowel with strangulation plays a crucial role in determining morbidity and mortality. However, no consistent pathognomonic findings for the diagnosis of strangulation have been described. Traditionally such recognition is based on the presence of one or more of the classic signs of vascular compromise including continuous abdominal pain, as opposed to colicky pain, fever, tachycardia, signs of peritoneal

irritation, leucocytosis, hyperamylasaemia and metabolic acidosis. Retrospective studies have indicated that these individual parameters cannot be relied upon for the diagnosis of strangulation. Stewardson et al have suggested reliance on certain specific combinations of parameters.

Sarr et al have evaluated the diagnostic capability of preoperative recognition of intestinal strangulation in a prospective study. Numerous parameters fro the history, physical examination, laboratory findings as well as the surgeon's clinical judgement were analysed individually and in combinations. Although the classical clinical findings that have been proposed to be of use for the identification of strangulation were marginally more prevalent in the group with strangulation obstruction, when each parameter was analysed individually, it lacked sensitivity. Predictive values were also so low that they had no discriminant capacity.

Leucocytosis was more prevalent in patients with strangulation but this difference was not statistically significant. The sensitivity, specificity, predictive value and efficiency of the white blood count as a sole determinant of strangulation was too low to be of diagnostic value. Even when all the classic signs of strnagulation obstruction were combined and analysed discriminantly, no combination proved simultaneously sensitive, specific, highly predictive and efficient as a determinant of strangulation. Clinical judgement by experienced clinicians proved disappointing, the sensitivity being only 48%.

Increases in the phosphate concentrations in the peripheral blood as well as urinary clearance of phosphate in patients with necrotic bowel and dogs with devascularized intestinal segments, have been reported. In the prospective study neither phosphate concentrations nor creatinine phosphokinase proved reliable for predicting the presence or absence of strangulation.

These conclusions were based on patients with complete small bowel obstruction and therefore do not necessarily apply to the management of patients with partial obstruction. If selected patients with incomplete obstruction are treated conservatively it must be remembered that should improvement not occur within a reasonable time operative treatment should not be delayed because a preoperative diagnosis of strangulation cannot be made or excluded reliably by any known clinical parameter, combination of parameters or by experienced clinical judgement.

Chapter 10.3: Gastrointestinal Haemorrhage

P C Bornman

Gastrointestinal haemorrhage is a common cause of emergency admissions in large community hospitals (50-150 per 100000 population per year). The overall mortality is low (5-10%). In the small but increasing proportion of elderly patients, mortality figures may rise to 40% and 60-70% in those who bleed while in hospital for other reasons. Most of these deaths are related to continued bleeding or a rebleeding episode, requiring massive blood transfusion or the need for surgical intervention. The management of these patients requires a multidisciplinary approach with close co-operation between physician and surgeon. The prognosis of gastrointestinal haemorrhage depends to a large extent on

the underlying pathology and it is therefore important to establish the cause soon after admission to hospital.

This chapter outlines the aetiology, general approach and the medical and surgical treatment of the common conditions that may require active intervention. For convenience, upper and lower intestinal haemorrhage will be discussed separately.

Upper Gastrointestinal Haemorrhage

Upper gastrointestinal bleeding is by far the most common cause of gastrointestinal haemorrhage and is defined as bleeding coming from lesions proximal to the D-J flexure.

Incidence and Aetiology

The development of forward-viewing fibreoptic panendoscopy has made it possible to identify the source of bleeding in over 90% of cases. Table 10.3.1 shows a world-wide estimate of the relative frequencies of bleeding lesions. The spectrum of lesions varies geographically, and is influenced to a large extent by the population served by the admitting hospital and the timing of endoscopy. In most series peptic ulcer disease causes approximately 50% of bleeds while oesophageal varices and Mallory-Weiss tears rarely exceed 10%. The decline in the incidence of stress ulceration in patients with severe trauma, burns (Curling's ulcer), head injuries or multisystem failure is of interest. The explanation for this remains uncertain, but improved intensive care may have played an important role.

Table 10.3.1. Endoscopic incidence of causes of acute upper gastrointestinal haemorrhage

Oesophagus

--> Oesophageal varices	5-20%
--> Mallory-Weiss tear	5-15%
--> Oesophagitis	5-10%
--> Neoplasm, prosthesis, vascular malformation	< 5%
--> Oesophageal ulcer	< 3%

Stomach

--> Peptic ulcer	15-20%
--> Erosive haemorrhagic gastritis	10-20%
--> Neoplasm	< 5%
--> Stomal or anastomotic ulcer	1-5%
--> Hiatal hernia	< 2%
--> Vascular malformation	< 2%

Duodenum

--> Peptic ulcer	20-25%
--> Erosive duodenitis	5-10%.

Management

History and Clinical Examination

A careful history and clinical examination may disclose the probable source of haemorrhage. It must be stressed, however, that one-third of patients bleeding from peptic ulcer will have no history of dyspepsia and conversely 40% of patients with dyspepsia will not be bleeding from an ulcer. Furthermore, salycilate ingestion is more often associated with bleeding from a peptic ulcer than from erosions, and in known cases of portal hypertension with oesophageal varices one-third will be bleeding from another site.

The initial clinical assessment is helpful in identifying the important subgroup of patients who are likely to die from bleeding. These are patients over sixty years old, and those with cardiac, pulmonary, hepatic or renal disease. Also of prognostic importance is the severity of the bleeding episode as manifested by haematemesis, a low haemoglobin (< 8 gm%), shock (BP < 100 mm Hg) and the need for massive blood transfusion (> 6 units).

Resuscitation and Further Management

Ideally, all patients at risk should be admitted to a high-care unit and be looked after by a team of medical and surgical gastro-enterologists. There should be no delay in establishing an intravenous line and in shocked patients a central venous pressure line and urinary catheter are mandatory. Baseline haematological and biochemical investigations must be performed without delay plus blood cross-matched for transfusion. A hepatitis screen and basic coagulation factors should be requested in patients with suspected oesophageal varices.

Resuscitation is accomplished with standard crystalloid and colloid solutions until blood is available. It is important to note that patients with liver disease should under no circumstances be given sodium-containing crystalloid solutions. Secondary hyperaldosteronism prevents these patients from managing a sodium load, and administering of such solutions will lead to deteriorating liver function, and increase their ascites. They should only be resuscitated with blood and 5% dextrose, and when necessary given fresh-frozen plasma to replenish clotting factors.

Resuscitation must be adequate to ensure the early detection of rebleeding. Not infrequently further bleeding is erroneously diagnosed in patients who have simply not been adequately resuscitated and who continue to pass melaena stools from their initial bleeding episode.

The role of a nasogastric tube is controversial. Benefits include the possible early identification of recurrent or continued bleeding, the demonstration that a patient whose aspirate contains clear bile has stopped bleeding, and the prevention of further bleeding with gastric lavage. On the other hand, a nasogastric tube is uncomfortable, and in elderly patients it increases the risk of aspiration pneumonia. It must also be stressed that the tube, unless irrigated at regular intervals, tends to block and recurrent bleeding may pass unnoticed. Furthermore, the effectiveness of continued lavage with alkalis in preventing recurrent bleeding, othen than in the stress ulcer situation, remains to be established. On balanace the routine use of a nasogastric tube is not advised.

Diagnostic Investigations

Endoscopy or Barium Meal?

Total endoscopy is the preferred investigation for acute upper GI bleeding and it has largely replaced the barium meal as an emergency investigation. It has been shown to be more accurate than a barium meal in determining the bleeding site, particularly with regard to mucosal lesions such as erosions and gastritis. But perhaps more important is its ability to demonstrate stigmata of recent bleeding (blood clot on the lesion or a visible vessel in the base of an ulcer crater). Such stigmata not only indicate the site of bleeding when more than one lesion is present, but identify patients who are particularly likely to bleed again. The finding of a clean ulcer, however, does not necessarily mean that it has not bled and in the absence of other lesions it remains the most likely source of bleeding. The value of stigmata of recent bleeding in planning management strategy will be discussed under peptic ulcer.

The timing of endoscopy is important. Urgent endoscopy is indicated when oesophageal varices are suspected or when there are signs of continuing haemorrhage. Endoscopy in these situations is best undertaken in the operating theatre, with the patient anaesthetized and the airway protected by an endotracheal tube. However, most patients have stopped bleeding by the time of admission to hospital. Endoscopy can then be delayed for approximately 12 hours (usually until the next morning's routine endoscopy list). The patient will by then be fully resuscitated and the stomach clear of blood, facilitating an accurate endoscopic assessment. Follow-up endoscopy may be indicated during the same hospital admission if recurrent bleeding is suspected or when the initial endoscopy was unsatisfactory. It must be stressed that even an experienced endoscopist may fail to identify an ulcer high on the posterior gastric wall or the rare small superficial ulcer, Dieulafoy's gastric erosion. Other rare causes such as haemobilia, haemosuccus pancreaticus (bleeding from the pancreatic duct) and an aorto-duodenal fistula may also be overlooked.

Angiography and Isotope Scanning

Angiography is indicated in the small number of patients who continue to bleed and in whom endoscopy and barium studies have failed to reveal a likely site. Its success, however, requires active bleeding at the time of the procedure. The bleeding site cannot be demonstrated unless the bleeding rate is about 1-2 mL/min or when lesions such as a false aneurysm, tumour, or vascular malformation produce a readily identifiable "blush". Lesions which may be demonstrated in this way include: diverticular disease and angiodysplasia of the colon, those causing haemobilia and haemosuccus pancreaticus, arterio-venous malformations and tumours in the small bowel, and aorto-duodenal fistulae.

The routine use of isotope scanning to select patients for angiography has recently been advocated. However, its reported success rate varies greatly and its value depends to a large extent on the expertise available at the institution where it is performed.

It is important to stress that diagnostic investigations should never delay active treatment in the small group of patients who continue to bleed and in whom resuscitation has failed to stabilize the patient. These patients should be taken to the operating theatre,

where endoscopy can be performed under general anaesthesia before embarking on surgery. This will avoid inappropriate surgery, particularly in patients bleeding from oesophageal varices.

Laparotomy and Peri-Operative Endoscopy

A careful laparotomy may reveal the bleeding source in the small but important subgroup of patients where no diagnosis was made preoperatively. The following conditions may be encountered:

--> Unsuspected liver cirrhosis with bleeding oesophageal varices missed on endoscopy due to shock (with collapse of the variceal channels) or to excessive blood in the oesophagus.

--> Postbulbar duodenal or small gastric ulcers.

--> A distended gallbladder and bile duct may be a clue to haemobilia, and chronic pancreatitis may be a pointer to haemosuccus pancreaticus.

--> Aorto-duodenal fistula, small-bowel tumours and arterio-venous malformations.

--> Colonic bleeding.

A gastrostomy is indicated when no obvious lesion can be identified and when there is a strong suspicion of a gastric lesion. The incision should be placed approximately 1.5 cm from the lesser curve to avoid technical problems should a gastrectomy become necessary. The stomach is cleared of all clots and washed with saline to enable meticulous inspection of the mucosa. Special care should be taken to exclude small ulcers in the upper part of the stomach as well as fundal varices. The surgeon should proceed to a pylorotomy when no lesion is found in the stomach. This will allow inspection of the distal part of the stomach, the duodenum and the ampulla of Vater. It is mandatory to mobilize the third and fourth part of the duodenum fully when an aorto-duodenal fistula is suspected. The detection of small-bowel and large-bowel pathology can be extremely difficult particularly in cases of diverticular disease and haemangiomatous lesions. It must be stressed that the site of blood within the GI tract is a poor guide to its source. Transillumination of the bowel is a useful technique but per-operative panendoscopy is frequently required. The latter has been used successfully to identify rare causes of bleeding particularly in the small bowel and the colon after rapid intra-operative bowel preparation.

Definitive Treatment

Bleeding from lesions such as oesophagitis, Mallory-Weiss tears and erosions of the stomach and duodenum are usually self-limiting and the treatment is entirely conservative. These lesions rarely rebleed and in the absence of endoscopic stigmata of recent haemorrhage, patients may be discharged from hospital once the diagnosis is made.

The treatment of patients bleeding from peptic ulceration or oesophageal varices is more complex and will be discussed in greater detail.

Peptic Ulceration

Medical Treatment

The choice of medical treatment lies between high doses of antacids, mucosal protecting agents and H2 receptor antagonists and more recently somatostatin. However, there is little evidence that these drugs will reduce the incidence of rebleeding. Their use should be seen merely as the commencement of definitive treatment of the underlying cause.

Only a small proportion of patients require emergency surgery for continued bleeding and of those who have stopped bleeding, only 15-20% will rebleed while in hospital (usually within 48 hours of admission).

Rebleeding is the most important cause of death (tables 10.3.2, 10.3.3) particularly in elderly patients who are haemodynamically compromised. Risk factors for rebleeding include those patients who present with red haematemesis, shock on admission and endoscopic stigmata of recent bleeding (table 10.3.4). The vexed question in patients at risk of rebleeding is whether a more aggressive surgical approach will reduce the high mortality associated with rebleeding. Several reports including a recent randomized study seem to support such an aggressive approach. This, however, must be weighed against the post-operative morbidity and mortality in an often elderly population with associated diseases who might respond to a conservative approach. Individual risk factors such as shock and endoscopic stigmata of recent bleeding do not predict recurrent bleeding accurately enough to justify early (prophylactic) surgery. But newer criteria such as the combination of shock on admission with endoscopic stigmata (which predicts rebleeding in 79% of cases) and the use of scoring systems or regression equations may help reduce the number of unnecessary operations. A more aggressive surgical approach may be justified in such patients. The decision to operate is often difficult in elderly patients with incidental medical illnesses and should therefore be made on an individual basis by any experienced team of surgeons and physicians (table 10.3.6).

Surgery

The surgical option in patients with a bleeding duodenal ulcer has been simplified by the recognition of the effectiveness and low risk associated with vagotomy and drainage with under-running of the bleeding points. This operation is also recommended in the high-risk patient with a gastric ulcer, particularly when the site of the ulcer will lead to a difficult gastrectomy. For more gastric ulcers, Billroth I gastrectomy remains the operation of choice. Success in emergency surgery for bleeding peptic ulcer is measured in terms of survival and not necessarily by long-term recurrence-free rates. The overall mortality for emergency surgery for bleeding peptic ulcer is about 10% but rises steeply with increasing age and delay in treatment.

Table 10.3.6. Indications for surgery in peptic ulcer

Absolute Indications

Exsanguinating haemorrhage	Emergency
Arterial spurter associated with shock	Emergency
Associated perforation	Emergency

Strong Indications

Combination of:	
--> Age over 60 years	Within 48 hours
--> Shock (Hb < 8 gm%; > 6 units of blood)	Within 48 hours
--> Stigmata on endoscopy or	Within 48 hours
--> Any age plus:	Within 48 hours
Second episode of bleeding associated with shock	Urgent
Continued transfusion requirement	
> 3 units/24 hours after resuscitation	

Relative Indications

Less severe second bleed	Semi-elective
Past history of bleeding	Semi-elective
Chronicity meriting elective surgery	Semi-elective.

Non-Operative Methods for Control of Bleeding

A number of endoscopic methods of treating bleeding peptic ulcer have been used since the advent of fibreoptic endoscopy. The most commonly used methods today employ thermal therapy with electro-coagulation (monopolar, electro-hydrothermal or bipolar), heater probe and laser photo-coagulation. Recently injection therapy for non-variceal lesions has also been advocated. Although good results have been reported in uncontrolled studies, few prospective controlled trials have shown a survival benefit. The disadvantages of endoscopic control of bleeding include:

--> the need for an experienced endoscopist
--> problems in achieving access to the bleeding site
--> a lack of evidence for cost effectiveness, particularly when laser equipment is used.

Nonetheless, there is a great need for further development in this field in order to avoid surgery in high-risk patients.

The role of angiographic embolization in cases with bleeding peptic ulcer is limited because it is potentially dangerous and the rich collateral blood supply of the stomach and duodenum often precludes effective embolization. There is also a real risk of bowel infarction when large vessels are embolized. This method is, however, extremely useful in

patients presenting with haemobilia and haemosuccus pancreaticus. Its use in the management of colonic bleeding will be discussed under lower gastrointestinal haemorrhage.

Oesophageal Varices

The management of acute bleeding from oesophageal varices is based on medical supportive measures, the replacement of blood and clotting factors, the administration of vitamin K, the use of vaso-active drugs to reduce the portal venous pressure and lactulose to empty the bowel of altered blood to prevent encephalopathy. H2 receptor antagonists have been shown to be of no benefit and the use of beta-blockers is still under review.

In most centres vasopressin or one of the newer more potent durgs such as glypressin is used to lower the portal pressure; this reduces recurrent bleeding. It is important to note that vasopressin not only causes constriction of the splanchnic arteriolar bed but also of the coronary arteries and in addition may cause cardiac arrhythmias. This drug is therefore contraindicated in patients with ischaemic heart disease. Glypressin, however, acts selectively on smooth muscle without the cardiac effects of vasopressin. Both drugs constrict the vascular bed and increase the risk of fluid overload. The effectiveness of these drugs is also limited by the phenomenon of tachyphylaxis. It is recommended that they be administered by continuous intravenous infusion rather than by a bolus injection or by selective intraarterial infusion. Their value in stopping bleeding is limited and their use should not delay endoscopy or the placement of a balloon for tamponade when there are signs of continued bleeding. In experienced hands the Sengstaken-Blakemore tube is very effective in controlling an acute bleeding episode. The placement and maintenance of this tube requires considerable experience and patients are best treated in an intensive care unit where complications such as aspiration, or displacement causing airway obstruction, can be treated expeditiously.

Both vasoconstrictive agents and balloon tamponade only provide temporary control of bleeding. After stopping these drugs or removing the Sengstaken-Blakemore tube, about 60% of patients will rebleed. More definitive treatment is therefore required. The options today includew injection sclerotherapy, devascularization operations or port-systemic shunting. Any form of surgery during the acute bleeding episode is associated with a prohibitive mortality and should be avoided if at all possible. In principle, the simplest form of treatment should be used and injection sclerotherapy has been shown to be a most effective non-operative method. The rigid oesophagoscopy technique which was originally used has not largely been replaced by the fibreoptic endoscopy method. Fibreoptic endoscopy is readily available in most centres, it is safe, and in selected cases sclerotherapy can be performed at the time of the initial diagnostic endoscopy. Repeated sclerotherapy sessions are required to prevent recurrent bleeding and today only a small percentage of patients requiring emergency surgery for ongoing bleeding.

Lower Intestinal Haemorrhage

Aetiology

The colon is the source of most cases of lower intestinal bleeding and diverticulosis is by far the commonest cause of massive haemorrhage. Rarer causes

include angiodysplasia, carcinoma of the colon, polyps, colitis, rectal ulcers and occasionally haemorrhoids. In the small bowel, lesions such as tumours, arteriovenous malformations, ileal ulcers and Meckel's diverticulum are rarely incriminated.

Management

The approach to the patient with massive lower intestinal haemorrhage is along similar lines to upper gastrointestinal haemorrhage. Most of the patients can be managed conservatively with spontaneous cessation of bleeding. The choice of investigations depends to a large extent on the severity of the bleeding episode and clinical suspicion as to the underlying cause. Most patients will undergo a careful proctoscopy and rigid sigmoidoscopy to exclude local anorectal lesions. Gastroduodenoscopy should be carried out when a bleeding source in the stomach or duodenum is considered. Tarry stools are not always present in patients bleeding from the upper GI tract and patients with brisk upper GI haemorrhage may pass fresh blood per rectum. However, in this situation patients are usually shocked. The value of lower GI endoscopy, including colonoscopy, is greatly limited by the presence of blood. A barium enema is invariably unhelpful and barium in the colon will preclude the subsequent use of mesenteric angiography. Therefore all tests except proctoscopy and rigid sigmoidoscopy should be delayed until the colon is clear of blood. Angiography is reserved for patients who continue to bleed or when there is recurrence of massive bleeding. Appropriate time of angiography is difficult because most patients stop bleeding spontaneously and because its success depends on the presence of either active bleeding at the time or the demonstration of lesions such as angiodysplasia, arteriovenous malformations or tumours of the small bowel. In most series angiography will demonstrate the bleeding site in approximately half of the cases. Selective intra-arterial vasopressin has been shown to be effective in stopping bleeding from diverticular disease but the risk of complications such as bowel infarction should limit its use to poor-risk patients. Recently, radio-isotope scanning (99m technetium sulphur colloid and 99m technetium-labelled red cells) has been advocated as screening test prior to angiography. However, the value of these non-invasive tests is limited by their inability to show the nature and the exact location of the bleeding site.

Exact location of the bleeding site greatly facilities surgery in these patients. Most of the lesions causing lower intestinal bleeding cannot be localized easily at laparotomy. Furhtermore, knowing the location of the bleeding site in patients with diverticulosis and angiodysplasia allows the surgeon to perform a relatively safe limited resection rather than an extensive colectomy with an ileo-rectal anastomosis.

As in the case of upper gastrointestinal haemorrhage, the decision to operate and the timing of surgery is critical. Most patients stop bleeding spontaneously but unnecessary delay in surgery and massive blood transfusion in elderly patients should be avoided.

Comment

Gastrointestinal Haemorrhage

H H Lawson

A clear distinction between "acute" and "chronic" ulceration is very useful as it helps a great deal in understanding the pathogenesis and the management of the whole problem of upper GIT bleeding. Essentially, in acute ulceration there is no deformity of the stomach or duodenum, and the diagnosis can only be made endoscopically. The disease is limited mainly to the mucosa. In chronic ulceration there is usually deformity and the diagnosis can be made on endoscopy or barium meal examination. (The disease involves much more than only the mucosa.)

Acute Ulceration

--> **Proximal gastric disease** (confined more or less to the acid/pepsin secreting part of the stomach) occurs with shock and sepsis (? A-V fistula, ? ATP defect). On endoscopy there are multiple small ulcers/haemorrhages, limited to the proximal stomach. If surgery is necessary, theoretically a proximal gastrectomy should be done. A total gastrectomy is usually advised, if only for technical reasons.

--> **Distal gastric/duodenal ulcer disease.** In Cushing's (head injury) or Curling's ulceration (burns) the ulcers are much larger than in the proximal variety; they also occur after drugs, especially cortisone. Possibly this represents a mucous abnormality. On scope they are limited to the duodenum. Treatment is by standard distal gastrectomy.

Total gastric disease. The so-called "acute superficial haemorrhagic gastritis" is often associated with drugs of the non-steroidal anti-inflammatory group. Literally on endoscopy the stomach looks as though it were a sponge dripping blood. Multiple ulcers are present, but can be seen microscopically only. Treatment is by total gastrectomy.

--> **Vagotomy and acute ulceration.** It is a lesser procedure than total gastrectomy, but vagotomy has been shown to have only a temporary effect on blood flow reduction. However, it may have a place when combined with such drugs as hydrogen ion receptor antagonists, or even the newer drugs that affect ATP mechanisms such as omeprazole.

--> **"Chronic ulceration".** Undersewing of the bleeding gastric ulcer is receiving more and more attention in the literature.

Lower Intestinal Bleeding

We have had good results from scanning with the appropriate isotope, because of course the scan can be repeated after a number of hours, whereas the angiogram is very much a "one shot affair". We have also had the good fortune to see the odd Meckel's diverticulum on a scan.

Comment

Gastrointestinal Haemorrhage

N J Laage

A major principle in the management of patients with gastrointestinal haemorrhage is to distinguish between bleeding originating from the upper versus the lower GI tract. Haematemesis localizes the source of bleeding proximal to the ligament of Treitz. With a history of melaena or haematochezia some doubt ofter remains as to the exact site of bleeding. Under these circumstances it is usually helpful to confirm objectively the presence and nature of blood with inspection of stool and nasogastric aspirate.

Controversy regarding the exact timing of endoscopy in upper gastrointestinal haemorrhage should be set aside, and the goal should be to make an early accurate diagnosis on which to base therapeutic regimens and prognosis. Urgent endoscopy is indicated in patients who had an incident of massive haemorrhage, evidence of continuing haemorrhage or rebleeding and when oesophageal varices are suspected. We do not perform endoscopy in these circumstances under general anaesthesia, but deliberately omit pharyngeal anaesthesia to reduce the risk of aspiration.

In approximately one-third of patients with upper gastrointestinal haemorrhage, bleeding will be massive, continuing or recurring with resultant formidable mortality. The benefits of early and accurate diagnosis must be sought in these patients by detecting stigmata of recent bleeding as predictors of further bleeding, on which to base therapeutic decisions.

Endoscopic stigmata of bleeding in peptic ulcer patients are classified as follows (with the approximate risk of continuing or rebleeding indicated in parenthesis):

--> signs of active bleeding:
- arterial spurter (90%)
- visible vessel of ooze from beneath clot (50%)

--> signs of recent bleeding:
- adherent clot or black spot (25%).

Endoscopic stigmata of bleeding cannot be recommended as the sole means of selecting patients for urgent surgery, but the associated risk of bleeding should be judged in the specific clinical setting. The mortality in patients older than 60 years with rebleeding is formidable and should be avoided. Shock as parameter is associated with rebleeding in 70% of patients. Gastric ulcers tend to rebleed significantly more than duodenal ulcers. With an aggressive management policy based on these guidelines, peptic ulcer mortality rates of 2-3% have been reported with operative rates around 40-50%.

The choice of operation for bleeding peptic ulcer should be carefully weighed after considering the operative risk, anatomical factors and available surgical expertise. With good surgical judgement it is possible to attain optimal control of bleeding as well as long-term cure without increasing the risk of operative mortality. For the good-risk patient

with duodenal ulcer who is haemodynamically stable, truncal vagotomy (antrectomy), figure-of-eight suture ligation of the bleeding vessel with non-absorbable suture material is recommended, kif a safe resection and anastomosis can be performed. A Billroth I reconstruction is preferred but under certain anatomical circumstances a Billroth II or Roux-en-Y reconstruction may be safer alternatives. Total vagotomy suture ligation of the bleeding vessel (pyloroplasty) is reserved for the high-risk patient but in the event of duodenal narrowing a gastroenterostomy is recommended as the drainage procedure. Prepyloric and pyloric ulcers should be managed as duodenal ulcers. Distal gastric resection including the ulcer with Billroth I reconstruction is recommended in the good risk gastric ulcer patient, and for the difficult high-lying gastric ulcer a Pauchet procedure, excising the lesser curve to include the ulcer, may have to be performed. Only in the extremely high-risk patient should ulcer excision be followed by truncal vagotomy and pyloroplasty be considered. Besides operative control of the bleeding site from recurrent ulcer following previous surgery, subtotal gastrectomy is the usual approach if previous vagotomy has been undertaken, and truncal vagotomy if the patient had an initial gastrectomy.

Endoscopic therapy emerged on the horizon as alternative treatment for many high-risk patients with high-risk lesions, and is sure to find wider clinical application especially since most bleeding patients are submitted to early diagnostic endoscopy.

Injection sclerotherapy as part of the routine management protocol for acute variceal bleeding has made a major impact in reducing mortality related to the initial incident, from 50% to 25%.

Mention should be made again that as much as 5% of acute lower gastrointestinal haemorrhage may occur from a duodenal ulcder or aortoduodenal fistula without haematemesis and should be appropriately investigated.

Before the colonoscopy and arteriography era, patients with unexplained lower GI bleeding were frequently submitted to subtotal colectomy and ileoproctostomy with unacceptable functional results, since most of these patients were elderly with compromised sphincters. These investigative techniques have prompted a change in approach by accurate localization of the involved segment preoperatively.

When double-contrast barium enema shows only diverticular disease as the most likely cause of bleeding, colonoscopy will detect additional lesions in 40% of patients (angiodysplasia 5%, colitis 10%, cancer 10% and polyps 15%).

Radiation damage to the large bowel can result in troublesome bleeding which may be very difficult to manage. Ischaemic colitis must also be considered as a cause of haemorrhage in the elderly and may be associated with infarcted bowel.

In the cases of Meckel's diverticulum a confident diagnosis can usually be made by a technetium scan.

Endoscopic therapy may be appropriate in certain circumstances of lower gastrointestinal haemorrhage using the polypectomy snare, electrofulguration techniques or laser therapy.

Chapter 10.4: Trauma of the Abdomen: Blunt and Penetrating

L W Baker

Introduction

Most surgeons, either during their training or in their surgical practice, are called upon to manage patients who have sustained trauma to the abdomen. Irrespective of the nature of the trauma or whether it is confined to the abdomen or forms part of a multi-system injury, the management can be challenging and even with clear guidelines the surgical treatment often requires clinical judgement which comes only with experience. Abdominal trauma as seen at King Edward VIII Hospital, the teaching hospital of the University of Natal, is less common than head injury and trauma to the chest. Although not always available, a clear history of the mechanism of the injury and knowledge of the patterns of trauma are helpful in anatomical localization and determination of the type of injury that may be expected in a specific organ.

Missed injuries at the initial assessment may have serious consequences for the patient and it is therefore vital that all patients receive frequent careful clinical assessment, preferably by the same observers, so that delayed presentation of injury to a specific organ will be detected at the earliest possible time and the appropriate management instituted. The surgical principles involved in the management of injury to some organs are clearly defined, in others there is controversy or bias, which is based on inadequate prospective data or clearly defined surgical principles, and for this reason injury scores for the patient as a whole and for individual organs are being widely used, but at present lack standardization.

Accepted Principles

Resuscitation

All patients should clearly have an adequate airway, be adequately ventilated and receive prompt and adequate blood volume replacement. Initially the nature of the replacement fluid is of lesser importance than the volume and no patient should be allowed to die from inadequate volume infusion. Healthy US Marines survived on a haematocrit of less than 10% and the most efficient for oxygen transport is 25% although 30% is considered to be the lower limit in the general population because cardiac output increases to maintain the level of tissue oxygen and this may not be available in older patients.

After resuscitation careful and repeated clinical examinations are of vital importance, will indicate early changes in the patient's condition and provide guidelines for timeous surgical intervention.

Routine laparotomy for penetrating trauma between the nipples and the upper thighs is undertaken in some units, therefore out attitude to stab wounds is considered very conservative. Laparotomy is performed when there is an indication of penetration of an organ as demonstrated by tenderness distant from the injury site, the development or peritonism, the presence of bleeding from the gastrointestinal tract or blood loss which

requires control. On prospective analysis in patients with proven penetration, this regimen has been shown to be safe and to limit the number of "unproductive" laparotomies. This is defined as a laparotomy at which there may be some pathology such as blood in the peritoneal cavity but where no surgical procedure is necessary for its correction. A small number of patients require delayed operation but with careful observation this does not increase morbidity. This policy has subsequently been supported by the experience at Baragwanath Hospital.

Diagnostic peritoneal lavage in penetrating trauma has led to unnecessary laparotomy because of the presence of blood in the lavage fluid and is not used. The management of gunshot injuries which are, in civilian practice, usually due to low-velocity missiles, is more aggressive and laparotomy almost routine if the passage of the bullet is suspected to have transgressed the abdominal cavity. Although the local tissue injury can be managed as for stab wounds, the path of the bullet is not predictable and more organs on average are damaged in the individual patient which leads to a higher morbidity and mortality in these injuries.

Although the indications for laparotomy are frequently present and clear in blunt trauma, peritoneal lavage is of value in patients with doubtful signs and particularly if the patient is unconscious. The performance of the open peritoneal lavage should be liberal rather than restrictive. The standard criteria of 100000 red blood cells per millilitre of fluid indicates significant bleeding and 500 white blood cells per mL are taken to indicate bowel penetrating. Although the latter is regarded as an absolute criterion for perforation, the occasional false positive has occurred.

Prophylactic antibiotics should be prescribed to cover gram-negative and anaerobic organisms at the time of admission to hospital if perforation of the bowel is suspected. The combination of early resuscitation, early antibiotic administration and surgery as soon as possible after the traumatic incident, have been shown to be associated with a low morbidity and mortality.

Laparotomy

There are advantages in acute and complicated trauma to have the patient in the modified lithotomy Trendelenburg (Lloyd-Davies) position. An assistant can be placed between the legs from which position access to the left upper quadrant is improved for the right-handed surgeon and access to the perineum is available in rectal and pelvic injuries. Utilization of this position is encouraged and is not time-consuming if used as a routine.

All patients undergoing operation for trauma require multiple peripheral venous access sites. This is particularly important when there is massive trauma or with intra-abdominal or thoracic bleeding. It is too late to gain access when the patient has been prepared and draped. A central venous line is not an acceptable route for massive fluid administration.

In the unstable patient a long midline laparotomy from xiphisternum to symphysis pubis is necessary to allow adequate access to control bleeding. Patients with inferior vena caval injuries including torn hepatic veins which have been tamponaded by intra-abdominal pressure, undergo rapid collapse on opening the peritoneal cavity and require

rapid volume and blood replacement. Under these circumstances it is necessary to tamponade venous bleeding by pressure and control the aorta at the diaphragmatic hiatus until circulatory volume has been re-established. Bleeding should then be controlled surgically prior to the management of perforated colon injuries followed by small bowel and solid visceral damage. Missed injuries of the colon and of the small bowel have a high morbidity and mortality and careful inspection of bowel for further possible injuries on the mesenteric aspect of the bowel, particularly in the colon, will be rewarded. Intra-operative prograde colone lavage has identified five such unsuspected colon injuries.

It should be routine practice to lavage the peritoneal cavity with large volumes of saline preferably heated to 38 °C until the lavage fluid is clear. The value of leaving an appropriate amount of antibiotic (tetracycline) in the last litre of lavage fluid has not been clearly established but it may be beneficial.

It is standard surgical practice to close the abdominal wall either as a monolayer or in two layers. More important than the number of layers is the suture interval which should be 1 cm or less and the suture bite which should be 1 cm or greater. The suture-to-wound length ratio should be at least 4 to 5 to one. There is no demonstrable difference in the quality of wound healing following continuous or interrupted suture techniquesx. The general tendency, however, is to pull the sutures too tightly which strangulates tissues. The suture material should be monofilament to avoid bacterial colonization of the interstices of woven or braided sutures.

Drains

Drains should be used in a closed system. Some negative pressure is desirable and this can be achieved by having the drain dependent. Drains should emerge through separate independent muscular wounds and not through the laparotomy (fascial) wound. Open drains of any variety lead to an increased wound and peritoneal infection rate. Prospective evaluation of drains used in association with colon injuries indicate that they are neither of great value nor harm. There is, however, a definite indication for their use with pancreatic and duodenal injuries where post-surgical fistulae are a sufficiently frequent occurence and in this situation the drain should be soft-walled and of adequate size, or multiple.

Principles in Contention

Organ Failure

Major trauma due to the presence of shock and sepsis, is frequently followed by various degrees of multiple systems organ failure. Adult respiratory distress syndrome (ARDS) is the most common. The part played by intra-abdominal sepsis, although it is a frequent component in this entity, is not clear nor are the indications or the timing for repeal laparotomy. Failure of the patient's condition to improve within 48 hours, usually in association with a falling platelet count or thrombocytopenia, constitutes a strong indication. Deterioration of the blood gas values, a fall in the haemoglobin level and the number of platelets has also been a helpful but not entirely reliable combination in defining the need for repeat laparotomy and peritoneal toilet. When pus or turgid fluid is found and lavaged from the peritoneal cavity, there is frequently but by no means

routinely, an improvement in the organ failure. Relaparotomy should always be accompanied by peritoneal lavage and culture of the fluid.

Postoperative Abscess

Whether an abscess if present, and where it is located, presents a difficulty. Indium labelled white blood cells, ultrasound examination and particularly computerized axial tomography (CAT) may all be useful but none is completely reliable. Drainage of the purulent collection is desirable and out choice is for surgical drainage but percutaneous drainage controlled by either ultrasound or CT scanning has been shown to produce comparable results and is probably the method of choice for some abscesses where surgical access would be complicated and where an anaesthetic is best avoided.

Postoperative Bleeding

Recurrent bleeding in the postoperative period while uncommon, may occur in two circumstances. Firstly, if the patient is hypotensive during operation, a major bleeder may remain undetected and unligated and bleeding will commence when resuscitation has been achieved and the blood pressure returns to acceptable values. Care should be taken at laparotomy to avoid this situation, and re-exploration should be undertaken expeditiously if bleeding occurs and the clotting factors are normal. Secondly, sepsis and the need for massive transfusion may lead to the presence of a bleeding diathesis. Estimation of the platelet count, prothrombin time, the partial thromboplastin time, thrombin time and fibrinogen level will identify the presence of the most common disorders found in surgery. A deficiency in platelet function rather than depletion of numbers is more usual. Thromboelastography alone or with the above may be diagnostic and helpful in assessing the response to treatment. Replenishment of labile clotting factors which will include fibrinogen is the only valid reason for the administration of expensive freeze-dried plasma. Under these circumstances the decision to re-explore the patient is more difficult but it may be necessary to ensure that a controllable bleeding vessel is not the cause and in any event, packing of a defined cavity may be helpful in the control of oozing.

System Injuries

Gastrointestinal Tract

Once the diagnosis of an injury to the gastrointestinal tract is made, laparotomy is required. Full laparotomy is necessary to identify all injuries to the bowel and other viscera. Control of bleeding is the first priority. Injuries to the colon should then be managed to preven faecal soiling of the peritoneal cavity. The edges of the wound in the bowel should be trimmed to create a well-vascularized margin prior to repair. Bowel that has been injured by blunt trauma, by an explosive device or by a high velocity missile, requires resection and anastomosis or endostomy and mucous fistula. Because it may not be possible to identify the extent of the cavitation injury, relaparotomy in 24 or 36 hours is necessary if there is any doubt. The technique of repair of the bowel in either one or two layers using absorbable or non-absorbable sutures is a matter of personal preference. Injuries missed at the initial laparotomy have a high morbidity and mortality because of the lack of physical signs in the postoperative period. The difficulties are exaggerated by sedation and induced paralysis to allow for ventilatory support in the intensive care unit.

Stomach and Small Intestine

These are the simplest injuries to repair. Perforation of the stomach with spillage of gastric contents, particularly into the pleural cavity, causes sudden death for reasons unexplained, in a small number of patients. Antibiotics are routinely given for penetrating trauma to these organs, but whether this is necessary has not been prospectively determined.

Colon

Injuries to the colon and rectum are widely recognized as being the most dangerous injuries within the abdomen due to contamination of the peritoneal cavity with faecal organisms. These injuries should be dealt with as a priority after the control of bleeding. Appropriate antibiotic cover should be instituted as soon as possible after admission to hospital. The most appropriate surgical procedure for the management of colon injuries is not clearly established.

There is still a large body of opinion supporting the use of exteriorization of the injury as a colostomy on the basis that the repaired wound in the colon is likely to disrupt in the postoperative period particularly in the presence of a high faecal load with disastrous results if placed intraperitoneally. Primary closure of an injury with proximal colostomy is therefore an irrational procedure because it leaves a suture line intraperitoneal in the presence of faecal contents in the bowel. These two procedures are the legacy of the instruction issued during the 1939-45 war that a colostomy should form part of the management of any colon injury. There is no evidence, from a number of studies supported by postmortem findings, that an intraperitoneal colon suture line disrupts.

Intraperitoneal Primary Closure

The main issue at present in the management of colon injuries is to determine whether intraperitoneal primary closure is not the appropriate procedure for almost all civilian colon injuries (destructive or high-velocity wounds being excluded). This is in defiance of the principles established from the experience in the 1939-1945 war. There have, however, been many improvements in patient care since 1945 in respect of resuscitation, the use of appropriate antibiotics and the performance of early surgery. Evidence is accumulating that intraperitoneal primary closure is a satisfactory procedure in most patients. Three hundred and ninety patients have had this procedure in a prospective evaluation and certainly in the last 172 patients the mortality, morbidity and hospital stay in these lower risk patients were better than for exteriorized closure. The mortality rate was 4% overall and only 2% died after 48 hours. In these patients there was no evidence of failure of the repair as determined at autopsy. The mortality was half that for the patients who had had exteriorization of the primarily sutured colon who were, however, considered to be at higher risk.

Contraindications to intraperitoneal primary closure at present are:

--> delay greater than 12 hours from injury to operation

--> faecal contamination

--> doubtful viability of the colon at the site of injury due to haematoma or a destructive injury

--> associated diaphragmatic penetration or renal injury.

These contraindications are being modified to assess the role of intraperitoneal primary closure in high-risk patients.

Exteriorization of the Primarily Sutured Colon

Exteriorization of the primarily sutured colon with early return, "dropback", first described by Mason in 1945, achieves all the requirements of a colostomy in that the suture line is outside the peritoneal cavity. This procedure has not gained universal acceptability probably because of the variable success achieved in avoiding breakdown of the suture line on the exteriorized loop, resulting in a high colostomy rate. To achieve a high success rate of early return or "dropback" requires attention to technical details which are well described. Avoidance of a colostomy has been achieved in 75% of more than 400 patients who have had this procedure performed as the only alternative to intraperitoneal primary closure in patients with high-risk injuries. It is associated with a shorter hospital stay than for colostomy and has less morbidity. It avoids a second admission to hospital for colostomy closure and in our experience is certainly a superior alternative to creating a colostomy at the site of injury.

Rectum

A rectal injury should be suspected in all patients with a penetrating injury between the iliac crest and mid-thigh, particularly if the injury is caused by a gunshot and crosses the midline. Less commonly the rectum is injured by bony spikes in a severe pelvic fracture.

All rectal injuries, whether they appear to be small or large or above or below the peritoneal reflection, have a high potential for morbidity and mortality and should be treated according to the well-established protocol consisting of five requirements as there is no prospective evidence of any other superior regimen:

--> The patients should all have a diverting colostomy.

--> Theinjury in the bowel wall should be closed after debridement of the edges.

--> The faecal content of the rectum should be evacuated by lavage from above until the effluent is clear.

--> Drainage of the soft tissues, particularly below the peritoneal reflection, is essential and preferably through the perineum. This should be performed even if it is not possible to close the wound in the bowel.

--> Appropriate antibiotics covering gram-negative aerobes and anaerobes are also essential. The Lloyd-Davies position is a requirement for this protocol and should be used routinely.

Colon Lavage

Although lavafe of the rectum is an essential part of the management of rectal injury, prograde colon lavage prospectively and randomly allocated to patients with colon injuries in 391 patients did not show an improvement in mortality or septic complications, but it did identify five patients with perforations that would otherwise have been missed, thus saving at least significant morbidity, if not mortality. Patients who had successful early return of their sutured and exteriorized colon after prograde colon lavage had a significantly shorter hospital stay.

Splenic Injuries

The spleen is the organ most frequently injured in association with blunt abdominal trauma. Encouragement for splenic conservation has been provided by the appreciation of its immunological importance over the last two decades. It has been made possible by the finding that adequate splenic healing can occur after partial splenectomy or repair of splenic substance. Conservatism is being further encouraged by the fact that it is now possible to identify damage to the spleen by non-invasive technology. Non-operative management is being advocated in children on the basis that associated injuries particularly to bowel are less common than in adults. This is not, however, substantiated by reports from major trauma centres.

The basis for the conservatism in children is the fact that from 1-3% will develop post-splenectomy infection following removal of a normal spleen. The mortality ranges between 30% and 50%.

In blunt trauma, injury to the spleen should always be considered as a possibility and more particularly if the trauma involves the left upper quadrant, the left flank, and is associated with fractures of the left lower ribs.

Diagnosis may be assisted by displacement of the gastric shadow from the abdominal wall on a plain film of the abdomen or chest. It may be more strongly confirmed by an isotopic liver/spleen scan, by ultrasonography, CT scan or angiography, or digital subtraction angiography. In adults, associated visceral injury requiring operative repair is claimed to be present in 20% of patients. Therefore the risk of missing a serious associated injury or seriously delaying appropriate treatment requires that laparotomy should be performed for a clinical diagnosis of ruptured spleen.

Repair should not be attempted if the spleen is pulped or if the injury is at the splenic hilum. Numerous mechanisms of repair, such as partial resection and repair of the splenic capsule, compression of the splenic tissue using catgut or liver needles, wrapping the spleen in an absorbable mesh bag and the use of fibrin sealant, have all been successful. If no doubt, our dictum is "there will be no credit for saving a spleen and losing a patient".

Penetrating injury to the spleen which has stopped bleeding may well unwittingly be treated conservatively, particularly in stab wounds, if there are no indications for laparotomy. If an injury is discovered at laparotomy an attempt at repair should be made.

It is not clear whether the splenic bed should be drained but it is advisable to do so if there is any oozing or any doubt about possible injury to the tail of the pancreas. A closed drain, removed as soon as it is obvious that there is no drainage of blood or pancreatic secretions, should be used.

Liver Injuries

The liver is the most commonly injured organ in penetrating abdominal trauma and second to spleen in blunt abdominal trauma. Severe injuries, for which isolation of the hepatic blood flow is required, occur in approximately 1% of all liver injuries.

Diagnosis of liver injuries is usually not difficult since signs of blood loss are the most common findings. Penetrating injuries requiring laparotomy usually present little diagnostic challenge. Injuries resulting from blunt trauma are diagnosed by the presence of blood on paracentesis or peritoneal lavage. Non-invasive diagnostic tests play only a minor role.

The majority of low-velocity penetrating injuries, either stab or gunshot wounds, when found at laparotomy, have usually stopped bleeding and require no further management. It is our policy to place a tube drain in the subhepatic space or adjacent to the site of penetration. The rationale for the drain is predominantly to detect a leak of bile postoperatively and, to a lesser extent, a recurrence of bleeding. In practice this is uncommon and the necessity for the drain could be challenged. If bleeding continues after compression of the liver, either with packs or manually for an adequate period (15 minutes) the wound must be explored rather than the placement of deep sutures which may control the bleeding by pressure but lead to the formation of an intraparenchymal haematoma or abscess which may progress to haemobilia.

Following blunt trauma simple lacerations without surrounding necrotic tissue may similarly be managed by drainage only. The value of manual compression or compression with laparotomy packs producing "hepatic tamponade" while dealing with other extrahepatic injuries, cannot be too strongly stressed even for severe stellate and multiple lacerations. If bleeding continues and is not controlled by occlusion of the vessels in the porta hepatis (Pringle manoeuvre), isolation of the hepatic blood supply, as described under "Retroperitoneal haematoma", should be undertaken. Once control is obtained, finger fracture should be used to expose the depths of the liver injury during which identification and ligation of blood vessels and bile ducts should be performed with removal of necrotic liver tissue. The resultant raw area of liver should be covered with an omental pedicle and this will aid local haemostasis. Large areas of destroyed and crushed liver tissue require removal.

Hepatic artery ligation, either right, left or common hepatic artery, has been claimed to reduce bleeding and is an acceptable procedure providing the gallbladder is also removed.

The ultimate challenge in the management of hepatic trauma is the control of bleeding from torn hepatic veins or damage to the portal vein in the subhepatic space. While the placement of an aorto-caval catheter is theoretically attractive, survival in the clinical circumstances requiring this procedure is seldom recorded. Following hepatic

artery ligation or resection of a major part of the liver, hypoglycaemia, hypoalbuminaemia, thrombocytopaenia and coagulopathies must be anticipated and treated. Drains are necessary after liver resection because of the increased risk of a bile leak.

Renal Injuries

Surgical Anatomy

The approach to renal trauma depends on the appreciation that the renal arteries are end arteries and that occlusion or disruption of an artery results in an ischaemic infarct. The arterial supply to the kidney is segmental and clearly defined and easily identifiable on a good quality arteriogram. Previously surgical ligation of the involved branch was necessary but interventional radiology with embolization has simplified the management of bleeding from penetrating trauma.

Haematuria following penetrating trauma is best investigated initially by a renal arteriogram and embolization of the damaged vessel by the radiologist in consultation with the surgeon. Excretory urography may indicate extravasation of urine but is not of assistance in the definitive management of the condition. Even large defects in a kidney due to low-velocity buller wounds do not require treatment per se, do not require operation or nephrectomy, and healing will usually occur. It is important to follow the renal function and blood pressure of the patient for a period of at least six months to ensure that hypertension due to partial ischaemia is not overlooked.

The approach to blunt trauma can also be conservative and the use of a single film following a bolus dose of intravenous contrast will provide evidence of the functioning of both kidneys or the absence of function on the injured side. The presence of some function on the traumatized side will exclude avulsion of the renal pedicle, a surgical emergency requiring reconstruction of the renal artery and vein. It is doubtful whether reconstruction of the pedicle after the lapse of one hour warm ischaemic time leads to any satisfactory function.

Urgent exploration of a flank haematoma due to blunt renal trauma is necessary only if there is bleeding which requires continuous transfusion of blood or is a threat to life. If there is no renal function on the urgent excretory urogram and exploration is undertaken for the management of other injuries, then nephrectomy should be performed on the injured side.

The management of the associated renal injury in retroperitoneal haematoma is discussed in that section. By adopting these policies Angorn was able to achieve a nephrectomy rate of 10% which is lower than recorded elsewhere in the literature, and there was no hypertension in the patients who had had arterial interruption or embolization.

Duodenum and Pancreas

It is difficult in blunt trauma to determine the presence of an injury to either the pancreas or duodenum or both retroperitoneal organs. In posterior penetrating trauma a high index of suspicion will encourage an aggressive search for such an injury. Peritoneal

lavage may not identify an injury to either of these structures because of their retroperitoneal situation. In a small number of patients straight X-ray of the abdomen will show bubbles of gas in the retroperitoneum, but these may be overlooked initially unless carefully searched for. Gas surrounding the upper pole of the right kidney is more obvious. A repeat supine film in 24 hours will frequently demonstrate this gas and a gastrograffin study will identify a duodenal perforation. Computerized tomography considerably improves diagnostic ability for both pancreas and duodenal injury but in most areas this modality of investigation is not easily available to trauma surgeons.

If laparototomy is performed because of a suspected injury, wide exploration of these structures in the retroperitoneum is essential.

Duodenum

Duodenal submucosal haematoma is an uncommon occurrence in children which leads to duodenal obstruction and which can be identified by a gastrograffin study. It can be treated conservatively and will resolve in two to three weeks. If discovered at laparotomy the haematoma in the duodenal wall should be evacuated and followed by primary closure of the seromuscular layer. If possible the mucosa should not be opened.

"Tidy" injuries due to penetrating or blunt trauma may be closed primarily after debridement of the wound edges, preferably transversely, after mobilization of the duodenum. It is important tocheck that there is no corresponding hole on the opposite side from the obvious injury. A small to moderate tissue defect in the duodenal wall, approximately equal to the diameter of the jejunum, may be sealed by using an adjacent jejunal loop sutured to the defect in the wall to provide a serosal patch. If there is any doubt about the viability of the duodenal wall, a lateral duodenostomy using a large-bore T-tube, is desirable.

If a secure closure cannot be made, a pyloric exclusion using a polydioxinone or non-absorbable purse-string suture followed by gastroenterostomy should be performed. The gastrostomy for the exclusion is used as the site of anastomosis. This procedure will reduce the morbidity and time of healing in this type of injury and it is considered to be preferable to the Berne diverticulation of the duodenum, which involves gastric resection in addition. We believe that it is important to drain all duodenal injuries because of the potensity for leaks to occur. In complicated injuries, gastric decompression and a jejunal feeding tube may be passed through a de Pezzer catheter for later enteral feeding, should complications arise. A needle catheter feeding jejunostomy has been found to be a suitable alternative for parenteral feeding.

Pancreas

In blunt pancreatic injury ultrasonography and computerized tomography facilitate the diagnosis of a swelling or haematoma in the retroperitoneum. Blunt trauma from the right side usually damages the head of the pancreas and often injures the duodenum as well; frontal injury will split the body of the pancreas over the vertebral column and left-sided injury will damage the tail of the pancreas and probably the spleen. Tidy injuries of the pancreas can be closed and drained. Untidy crushing injuries to the left of the superior mesenteric artery and vein should be resected with closure of the proximal end of the

remaining pancreas. The main duct should be identified and individually ligated if possible, but this is often difficult in the presence of a pancreatic haematoma.

The more complicated Rouc-en-Y loop drainage of the remaining head and tail, or closure of the head on the proximal side and drainage of the tail on the distal side cannot be supported. The morbidity and mortality arise from traumatic pancreatitis, retroperitoneal sepsis and fistula formation and each additional anastomosis adds to the possibility of these leaks. Where there is a viable but crushed head of pancreas this should be left in situ. If possible the capsule should be approximated and closed; drainage should be instituted. Sump suction drains are claimed to be superior but we have experience with tube drains where a negative pressure is created by the siphon effect which is equivalent to the height of the patient above the closed system drainage bottle. Pancreaticoduodenectomy in the trauma setting has a high mortality and should be performed only as a last resort when the injury has performed the pancreaticoduodenectomy and removal of the tissues is really a toilet procedure.

Closed drainage with wide-bore multiple soft tubes should be instituted. In a prospective but non-randomized study morbidity and mortality was higher in the abscence of drains in pancreaticoduodenal injuries.

Laparotomy

At exploration for diagnosis and management of pancreaticoduodenal injuries, wide exposure is necessary. The duodenum and head of the pancreas should be reflected so that both anterior and posterior aspects of the organs can be visualized. The tail of the pancreas must aslo be examined. If adequate exposure is not obtained, reflection of the right colon from the right iliac fossa towards the left hypochondrium will provide good exposure as described by Cattell and Braasch. Intraoperative pancreatography is advised by some to identify the main duct but this is usually not available and certainly non-visualization of the ducts does not appear to have caused morbidity. It is not possible to comment on its value.

Dead or devitalized pancreatic tissue should be carefully removed and haemostasis obtained by fine diathermy or accurate clamping and ligation of vessels. Large bites of pancreatic tissue are likely to lead to acute pancreatitis.

Complications

Fistulae are likely to occur from both the pancreas and the duodenum. Duodenal fistulae have a higher morbidity and mortality than pancreatic fistulae of which the vast majority will heal without surgery within six weeks, particularly if the patient is maintained on total parenteral nutrition.

Somatostatin is reputed to "switch off" the fistulae but we have not had the opportunity of comparing this substance with total parenteral nutrition. When a fistula persists for longer than six or eight weeks, it should be drained into a Roux-en-Y jejunal loop.

Abscesses occur and require identification and drainage which may result in the formation of a fistula which is treated on its merits.

Retroperitoneal Haematoma

Retroperitoneal haematoma, which are divided into three types, may be caused by either penetrating or blunt trauma and their management remains controversial:

--> type 1 arises from injuries to central structures, pancreas, duodenum, aorta, vena cava and occasionally portal veins

--> type 2 from injury to the lateral retroperitoneal structures, kidneys, ureters, suprarenal glands, ascending and descending colon

--> type 3 from injuries to iliac vessels, pelvic veins, ureters, rectum and from pelvic fractures.

A widely held view suggests that all retroperitoneal haematomata other than those due to venous bleeding in the pelvis, should be explored. On the basis of a recent analysis of 106 consecutive patients with retroperitoneal haematoma found incidentally at laparotomy, we support this contention for type 1 haematoma but the experience indicates that unnecessary exploration in type 2 haematomata may have serious consequences for the patient. With penetrating trauma it may be lifesaving to explore type 3 haematomata. The desirability for exploration or observation will be determined in types 2 and 3 by the nature of the trauma.

Type 1 - Central Haematoma

Because of the vital structures situated in this location all haematomata, irrespective of the nature of the trauma, should be explored. The principles in identifying injury to the pancreas and duodenum have already been discussed and this includes adequate and careful exploration by mobilization of the right colon and the duodenum and pancreas. Damage to the aorta, and particularly the vena cava, may come as a surprise on exploration of a haematoma with escape of a torrent of blood and should be avoided by prior control of these vessels. Penetrating injuries in this region, particularly following gunshot injuries, have a high mortality. In 17 out of 22 patients it was necessary to explore the haematoma as part of the resuscitation procedure to control bleeding from the inferior vena cava, the aorta or the portal vein. There were 14 deaths (68%) in the 22 patients with haematoma due to penetrating trauma and this constituted 70% of all the deaths from retroperitoneal haematoma.

Active bleeding from the aorta, the renal arteries, the inferior vena cava or portal vessels should be compressed to stop the bleeding while control is obtained proximally and distally; vascular repair should proceed once the circulating blood volume has been re-established or preferably slightly exceeded. When a stable haematoma is found at laparotomy it is usually possible to determine whether the damage is to veins or to arteries. The aorta should be exposed as it enters the abdomen through the diaphragm by mobilization of the oesophagus to the left as for truncal vagotomy. A muscular crux over the aorta can be teased apart using artery forceps, the aorta identified between the index

and middle finger of one hand and a long vascular clamp applied in the sagittal plane over the fingers without further mobilization. Back bleeding from the distal aorta is usually minimal under these circumstances.

To obtain proximal control of the vena cava, the left lobe of the liver should be mobilized by division of the falciform and left triangular ligament to expose the central tendon of the diaphragm. This is then opened anterior to the vena cava to expose the vessel within the pericardium, where it may be clamped without further mobilization. The inferior vena cava should also be exposed, encircled, and clamped in its infra-renal portion, care being taken not to injure the lumbar veins.

The opening to the lesser sac should be identified so that the portal triad can be controlled at any time if necessary.

Control of these vessels will allow repair of torn hepatic veins and has been found to be more satisfactory than the attempted use of an arterio-caval catheter which is favoured by those who are completely at ease with cardiac bypass. There is high mortality of penetrating injuries in this region.

Type 1 retroperitoneal haematoma following blunt trauma also requires exploration in order to repair the duodenum and to deal adequately with a damaged pancreas.

Type 2 - Lateral Haematoma

The great concern in the management of patients with posterior penetrating injuries is that an injury into the colon will be overlooked because of the lack of physical signs. Prospective evaluation in 50 patients indicated that this is not a clinical problem. Careful observation and awareness of the possible consequences of a missed injury are essential if the indications for laparotomy are to be based on clinical examination. If there is haematuria and laparotomy is considered necessary for suspected injury ot organs other than the urinary tract, a preoperative aortogram with selective renal angiography should be performed to determine the presence and nature of the injury. Damage to the renal vasculature should be controlled by arterial embolization. If no vascular damage is identified and a stable perinephric haematoma is found at laparotomy, it should not be explored. The colon may be mobilized to ensure that this viscus has not been penetrated.

A stable haematoma surrounding the kidney as an incidental finding at laparotomy, should also not be explored. If bleeding from the renal tract occurs after laparotomy this should again be controlled by embolization of the appropriate branch of the renal artery. In a series of 73 patients 32 did not have exploration of their stable perinephric haematoma and there were no related complications in the 30 surviving patients. The two deaths were not related to the management of the retroperitoneal haematoma.

Fifteen patients who had preoperative excretory urography or renal angiography with an identifiable lesion were all confirmed at operation and appropriately handled by repair in nine and partial or total nephrectomy in six. In the remaining 26 patients with a stable haematoma exploration, often for no clearly stated reasons, identified no significant lesions were found and exploration was therefore regarded as unnecessary. Significantly the three deaths in these patients were the result of sepsis from colon injuries in

association with injuries to other organs. Similarly following blunt trauma, if a stable perinephric haematoma is found at laparotomy undertaken for other indications, exploration is not advised.

Type 3 - Pelvic Haematoma

The nature of the trauma divides the patients into those in whom exploration is desirable following penetrating trauma and those in whom it should be undertaken as a last resort or only for clear indications following blunt trauma.

In the same study all 11 patients with penetrating trauma required exploration, four for active bleeding and seven for a clearly identifiable pulse defect.

Following blunt trauma, usually in association with a fractured pelvis, a stable pelvic haematoma found at laparotomy should not be explored. If exploration cannot be avoided, control of bleeding should be attempted:

    --> first by repair of major vessels
    --> then by ligation of identifiable bleeding vessels
    --> followed by ligation of both internal iliac arteries
    --> and finally by packing.

This may be ineffective in the presence of the "compressible" rectum in which event abdomino-perineal resection has been advocated as a life-saving solution followed by packing. Where blood loss in association with a fractured pelvis is the dominant problem and there is no indication for urgent laparotomy, control should be attempted by arterial embolization at angiography. Stabilization of the pelvis by an external fixator is claimed to be highly effective in producing cessation of bleeding and should be the next procedure. Failure of embolization and fixation should be followed by surgical exploration and the procedures already mentioned. The MAST suit has been advocated and although it is accepted as a mechanism of stabilization during transport to hospital, it is not regarded as a method of control of bleeding for hospitalized patients.

Where effective packs have been placed, there is little justification for removing these at 24 hours if there are no signs of infection. A patient who has been stable for 48 or 72 hours probably has less chance of rebleeding and greater chance of survival following a rebleed if the packs are removed at this time.

Pelvic Injuries

Patients who have sustained a pelvic fracture have been subjected to considerable violence, and associated organ injuries within the pelvis and in other areas are likely. If there has been a crush injury to the pelvis, an associated rupture of the diaphragm should be suspected. A passenger who has sustained injuries in a motor-vehicle accident must be assessed for injuries from a seat belt and blunt injury to the chest, including myocardial contusion which may not be evident.

At the site of the accident, severe pelvic injury is probably the most important indication for the application of a MAST suit since it will provide stabilization to the

pelvis and by this mechanism, as well as compression, limit venous bleeding. The most important injuries associated with a fracture of the pelvis are rupture of the male membranous urethra, rupture of the urinary bladder, disruption of the pelvic venous plexus, associated soft-tissue injuries and, less frequently, rupture or penetration of the rectum.

Urethra

Damage to the membranous urethra is common in association with fractures to the pubic rami. Injury is almost certainly present if there is blood at the urethral meatus, if the patients wishes but is unable to pass urine, and if there is a distended bladder. Complete rupture will be identified by a high-riding prostate gland on rectal examination. If there is any doubt, a urethrogram should be performed. A very gentle attempt to pass a urethral catheter is acceptable although it is unlikely to pass even in the presence of a partial rupture. Not infrequently the anterior wall of the urethra is intact and a urethral catheter may be passed by an experienced operator on an introducer by hugging the anterior wall of the membranous urethra. If a catheter can be inserted without any iatrogenic damage, there is a chance for partial rupture of the urethra to heal primarily without further surgical intervention. If there is complete rupture of the urethra, or it has not been possible to pass a catheter, a percutaneous suprapubic catheter should be inserted at least until the patient has been fully assessed and the general condition and the circulatory volume have been stabilized.

Urethral repair is not an easy surgical procedure and there is considerable controversy among urologists whether railroading a catheter in the acute situation, early primary repair at the time of initial surgery or delayed repair of the urethra between eight weeks and six months, is the appropriate procedure. As the repair of the urethra is not a lifesaving procedure our preference is for delayed repair. As the urethra is a delicate structure with a propensity for stricture formation, delicate manipulation is as essential when performing urethrography as it is in attempting to pass a catheter.

Bladder

Rupture of the bladder is not as easily recognized as rupture of the urethra but it should always be suspected in the presence of a pelvic injury. It may, however, occur after any deceleration injury and particularly after interpersonal violence when the person may have been kicked or hit by a brick or knobkerrie. Rupture of the full bladder is common. A urethral catheter should be passed routinely in all major injuries. The appearance and volume of the urine can be assessed to recognize injury and as a measure of successful resuscitation. The presence of haematuria or a small volume should lead to radiological assessment of the bladder as soon as the patient is sufficiently stable to be moved to the radiology department or preferably during resuscitation and assessment if the equipment is available in a trauma unit. Rather than using any specific volume of contrast medium it is important to distend the bladder with the solution until the patient has discomfort or until the distended bladder is clearly outlined on the X-ray film. A volume in excess of 300 mL of contrast may be required.

It is important not to miss a rupture of the bladder. Haematuria is not always present. The total volume of contrast solution instilled may be returned and be misleading

unless the bladder is distended. The presence of contrast amongst loops of bowel or in the extraperitoneal space will establish the diagnosis.

Although the urine may initially be sterile it will soon become infected in the peritoneal cavity or in the soft tissues, especially in the presence of a urethral catheter. This sequence of events carries a high morbidity and mortality. Late identification of the pathologt by the demonstration of swelling in the suprapubic area, lower abdomen and upper thighs, a raised urea and potassium and a lowered sodium indicating that peritoneal dialysis of the urine has taken place, should be recognized as a failure of initial assessment.

Rupture of the bladder, either intraperitoneal or extraperitoneal, is an indication for surgical intervention. The wall of the bladder should be repaired in two layers using absorbable sutures on the mucosal aspect. The choice still appears to be either plain or chromic catgut. This must be the only surgical indication for the use of plain catgut. There is a tendency to treat extraperitoneal rupture of the bladder by urethral or suprapubic drainage or both without closure of the defect in the bladder. Extravasated urine into the pelvic cellular tissues will be contaminated by the presence of a urethral catheter and sepsis may supervene. It is not possible to estimate on cystography how much urine has been extravasated into the tissues. Repair and urinary drainage is therefore still the general recommendation for extraperitoneal as well as intraperitoneal bladder rupture despite the fact that some patients with extraperitoneal rupture have recovered, without morbidity, on urinary drainage only.

Rectal Injury

Injury to the rectum in the presence of pelvic fracture is usually due to penetration by a spicule of bone which requires removal and the defect in the rectal wall requires surgical repair and a diverting proximal colostomy, lavage of the faecal content of the rectum, drainage of the site of injury and antibiotic cover, as discussed above. Vascular injury has also been discussed under type 3 retroperitoneal haematoma.

Soft-Tissue Injuries

Injuries to the soft tissues may occur in association with pelvic fracture or as an independent entity. There is an erroneous but recurring tendency to attempt debridement and closure of all wounds particularly if bone has been exposed. This is appropriate if the injuries are obviously of an incised nature and there has been no contamination with road dirt or soil, the tissues are not bruised and ischaemic and are unlikely to be contaminated by faeces.

Wounds which are in the region of the perineum, the upper thighs posteriorly, the buttocks or the anus, or if a "degloving" injury has occurred, should undergo rigorous debridement of any dead tissue and be left open. A diverting iliac colostomy should be formed to prevent recurrent contamination of the wounds by faeces.

The initial and repeat debridement of necrotic tissue and dressings should be done in the operating theatre under anaesthesia, if possible at 48 hours intervals until they are suitable for delayed primary or secondary closure, they may be allowed to close by

secondary intention. There were six deaths in 27 patients with compound pelvic injuries where these principles had been breached. Three patients died acutely from haemorrhage and the other three died later from sepsis. The latter three had had their wounds closed.

Pregnant Uterus

Injury to the non-pregnant uterus is very unusual and occurs only with transvaginal instrumentation or occasionally with bullet wounds. This also applies to the pregnant uterus up to the end of the first trimester.

The more advanced the pregnancy, the greater is the potential for both penetrating and blunt injury. Late in pregnancy the large uterus might aggravate the effects of hypovolaemia by reducing venous return through compression of the inferior vena cava when the patient is in the suptine position. To avoid compression of the vena cava, the patient should be positioned in the left lateral decubitus position because hypoxia of the foetus may be severe when maternal hypoxia is only minimal, high levels of oxygenation should be provided for the patient. While the uterus is not liable to rupture, placental separation is not uncommon and may aggravate blood loss from this source. Maternal blood volume in late pregnancy is increased by about 30% but there is a reduction in the red cell mass and the haematocrit level is usually in the region of 35%. In late pregnancy cardiac output is increased by 30-40% over normal values, but during hypovolaemia the uterine blood flow may be diminished by selective uterine artery constriction to as little as half, and this may jeopardize survival of the foetus. If a woman is injured late in pregnancy the foetus should be considered in the treatment plan.

In general, priorities of management are the same as for other victims with multipel systems injury and usually the most effective measures to save the life of the mother will also contribute to the survival of the unborn foetus.

The enormous increase in pelvic blood supply in late pregnancy exposes the patient with a pelvic fracture to severe haemorrhage. In addition pregnancy is a contraindication to the inflation of the abdominal compartment of the MAST suit.

If the foetus is in danger of hypoxia, Caesarean section may be indicated. About 50% of the foetuses deliver over the period of 28 weeks can be expected to survive.

The Diaphragm

The diaphragm provides a fibromuscular, mobile division between the thoracic and abdominal cavities. During full expiration and inspiration the dome moves between the level of the 7th and 12th ribs and may be injured by penetrating wounds to either cavity. Abdominal organs move up with the diaphragm and may therefore be injured by high thoracic wounds during expiration.

Blunt injury causes disruption of the diaphragm by forcing abdominal viscera into the chest usually rupturing the left dome. The liver is thought to absorb forces thus protecting the right dome.

An injury to the diaphragm should be looked for during a laparotomy. It will therefore be diagnosed more frequently under circumstances where routine laparotomy is performed for penetrating trauma. Some will be missed if patients undergo selective exploration. Close attention must be paid to the appearance of both domes on X-ray. Blurring of the diaphragmatic margin may be indicative of an injury.

Gas shadows which appear to transgress the diaphragm, if laparotomy is not to be performed for injury to other organs, must be investigated by a contrast study that includes the whole length of the gastrointestinal tract, stomach, small intestine and colon. On occasion, penetration is confirmed by omentum protruding through thoracic penetrating wound. A distended stomach herniated through a diaphragmatic wound may be impossible to distinguish from a large pneumothorax on the chest X-ray. Passage of a nasogastric tube or a small amount of contrast may show the stomach to be in the chest. On occasion it is discovered because a carefully placed intracostal drain does not function satisfactorily.

The frequency of missed injuries following conservative management, even with careful assessment, is unknown, but patients with strangulation of either stomach or colon, and less frequently small intestine, do present. The extent of abdominal organs in the chest is usually grossly underestimated by the chest X-ray and frequently includes the spleen.

Surgical repair of the diaphragm in penetrating trauma should be performed with a non-absorbable suture from within the cavity that is indicated by the trauma. Bleeding from the injured margin may be troublesome and should preferably be controlled by suture ligation or alternatively by figure-of-eight sutures.

Delayed repair, in the presence of a strangulated hernia, should initially be by laparotomy as resection of abdominal organs is best performed through that approach. Gangrene of the contents carries a high morbidity from empyema, particularly if there is perforation into the pleural cavity. It is difficult to lavage the pleural cavity satisfactorily through a small hole in the diaphragm. Gross contamination is best handled by a thoracotomy, careful lavage, chest drainage and antibiotics.

Occasionally, in blunt trauma, bursting of the diaphragm may leave a friable margin which is less easily repaired. If it is torn from its peripheral attachment, it must be re-attached by pericostal sutures.

While there is little morbidity or mortality from repair of the diaphragm, there is significant morbidity from missed injuries.

Miscellaneous

Organs commonly injured have been dealt with. However, injury to any structure may occur, particularly with penetrating wounds. The path of the weapon or missile should be carefully followed to its end. It is easier in knife wounds than in bullet wounds where it may be impossible.

--> Injury to the gall bladder is usually managed by cholecystectomy although closure and cholecystostomy have been shown to be safe.

--> Transection of the common bile duct is sutured over a T-tube.

--> Division of the ureter is best repaired over a ureteric stent brought out proximally.

--> Rarely, the cisterna chyli will be injured and probably missed at the initial procedure.

In these difficult circumstances the best care will be provided by the most experienced person available. The advantage of a trauma unit is the ready availability of staff experienced in a diversity of emergency situations.

Comment

Abdominal Trauma

D Pantanowitz

The approach to abdominal trauma at Baragwanath is generally very similar to the one outlined in this chapter. However, there are certain differences which will be discussed under four sections:

--> the conservative approach to abdominal trauma
--> major liver trauma involving the hepatic venous trunks and the retrohepatic vena cava
--> renal trauma
--> membranous urethral trauma.

The Conservative Approach to Abdominal Trauma

This was originally proposed by Saftan in 1960 and the concept was reinforced by Stein in 1968. But the large trials were conducted from 1984 onwards by Demetriades et al at Baragwanath, and this was subsequently confirmed by Huizinga et al in 1987. Essentially, Demetriades showed that peritoneal penetration, and even evisceration or air under the diphragm on chest X-ray, are not in themselves indications for laparotomy. He feels that the decision to operate should be based primarily on the abdominal physical signs, viz guarding, tenderness and rigidity. This applies to all penetrating trauma, but excludes high-velocity bullet wounds, which have not been studied at Baragwanath. We thus manage low-velocity bullet wounds in a similar manner to a penetrating stab wound. With regard to diagnostic peritoneal lavage, we are in agreement with Baker's approach, which is to avoid this investigation unless the patient cannot communicate properly, ie, head injury, alcoholic intoxication.

Injury to the Hepatic Venous Trunks and the Retrohepatic Vena Cava

We do not use catheter-shunting (arterio-caval shunts) for these injuries. Our approach to this type of injury is to obtain "hepatic tamponade" by either manual compression or packing (often both), thus allowing for adequate volume replacement and resuscitation. Only once the blood pressure returns to around 100 mm Hg systolic is the

liver blood supply controlled by clamping both the supra and infra hepatic vena cava above the renal vein, and the relevant portal pedicle structures (portal vein and hepatic artery). The liver can with stand the induced anoxia for at least one hour, provided that the shock has been adequately managed during the compression/packing phase. Now the damaged vessels can be repaired by displacing the liver to the relevant contralateral side for adequate exposure, or by resectional debridement using finger fracture to get to the "business area" of the liver injury.

Renal Trauma

We place emphasis on the intravenous pyelogram or excretory urogram (EU). In a patient with macroscopic haematuria, or with microscopic haematuria together with renal angle tenderness, signs of peritoneal irritation or fractures of the lumbar transverse processes, we obtain an urgent EU. If there is intrarenal extravasation (capsular tear or minor laceration of the collecting system), we treat conservatively. If there is extrarenal extravasation (major injury to the collecting system), exploration and repair is indicated. If there is delayed or absent function on EU, a renal pedicle injury must be suspected and angiography is required. In this situation, an intimal flap is not infrequently the cause of the arterial occlusion, and immediate vascular repair is required to prevent renal insufficiency and the development of reno-vascular hypertension.

Membranous Urethral Trauma

This is diagnosed by the presence of blood at the external meatus in a patient who has difficulty voiding or acute retention. This injury may follow a fracture of the anterior pelvic bony ring and is due to distraction of the prostate from the urogenital diaphragm (accounting for the high-riding prostate on PR). No attempt should be made to pass a urethral catheter as this may convert a partial into a complete tear. The diagnosis is confirmed by urethrography.

We treat this injury by suprapubic cystostomy with later attention to the stricture. Once the haematoma has settled, a voiding cysto-urethrogram (VCU) is performed via the suprapubic catheter. If there is minimal extravasation at the site of injury, the catheter is kept open for about seven to ten days and the VCU repeated. If there is not further extravasation, the suprapubic is clamped and the patient made to void. The catheter is removed following normal voiding.

With gross extravasation, the suprapubic is allowed to drain for about 14 to 16 days, after which a panendoscopy is performed to inspect the injury. If a filiform can negotiate this area to enter the bladder, an optical urethrotome is introduced and the area of stenosis incised at three positions (12, 3 and 9 o'clock). If a filiform cannot be passed, the bladder is filled via the suprapubic. The suprapubic tract is then dilated using a 7/10 Lister sound. This sound is advanced into the bladder and passed retrograde down the prostatic urethra. Now the optical urethrotome is introduced into the urethra and by manipulating the Lister sound, the site of obstruction is incised down to the sound. The urethrotome is then advanced into the bladder ("railroading"), thus restoring urethral continuity. Once a silastic urethral catheter is placed in position (using a "guideline" pulled through via the urethrotome to which the catheter is attached), a Foley is then placed via the suprapubic tract, and both catheters are allowed to drain freely. After two

weeks, the urethral catheter is removed, the suprapubic is clamped and the patient made to void. If voiding is satisfactory, the suprapubic remains clamped and a panendoscopy is repeated after a further week. If there is no stricture, the suprapubic is removed. If the stricture has recurred, it is again incised (urethrotomy for stricture may be repeated as required).

Comment

Abdominal Trauma

E J Theron

Introduction, Resuscitation and Evaluation

In the initial assessment of the abdominal injury patient it is our policy to encourage the casualty officer and trauma surgeon to maintain a
very high index of suspicion of possible intra-abdominal injury, especially in the multiple injury patient. Laparotomy is usually performed in patients with stab wounds of the abdomen.

However, in the stable patient with no signs of intra-abdominal blood loss, leakage from injured viscera or peritoneal irritation, the wound is sutured and the patient admitted and monitored carefully for 48 hours. Probing and local exploration of the wound yields very little information, is mostly unreliable and should therefore not be used. Gunshot and other missile wounds involving the abdominal cavity should always be managed by early laparotomy after resuscitation.

Diagnostic peritoneal lavage for penetrating injury is not used in out unit except in cases of penetrating injury of the lower chest, where penetration of the diaphragm is a possibility. Diagnostic peritoneal lavage for blunt trauma is performed for the indications listed in the text. Computerized tomography (CT) should also be considered in selected cases as an early investigation in centres with this facility. Blunt abdominal injury with haematuria is the ideal indication in the stable patient for early CT with contrast, instead of IVP only. Early CT scanning could effectively evaluate injuries of solid viscera, spot retroperitoneal haematomas and diagnose leakage from hollow viscera.

Laparotomy

The use of Lloyd-Davies position is no doubt of value, especially by providing easier access to the upper abdomen.

Before closure of the contaminated abdomen, lavage fluid is used to cleanse the abdominal cavity, with antibiotics added to the last volume of fluid in cases of severe contamination. Mass closure technique of abdominal wounds as described in the text is used routinely at out institution. Suction and closed-system drains rather than open drains are used in most cases.As a compromise a Penrose corrugated drain covered by a colostomy bag is a useful alternative.

Principles in Contention

--> Organ failure and abdominal sepsis/abscess

In the event of infection and sepsis of the peritoneal cavity intervening during the subsequent management, the surgeon may resort to the open treatment method. The abdominal cavity is thoroughly cleansed by lavage of several litres of warmed saline, the laparotomy wound is then approximated with nylon tension sutures leaving a gap of 2 cm, the bowel loops are covered with plastic sheeting and abdominal swabs. The sutures are not tied, but twisted with artery forceps which remain in the dressings. The patient is taken back to the operating theatre evewry 24-48 hours for re-exploration by untwisting the sutures, the cavity explored and cleaned.

The laparotomy wound is eventually closed by a running mass suture technique; the skin is not sutured.

System Injuries

--> Gastrointestinal tract

Various studies reported in the literature have shown that single-layer suture repair with one of the recently developed synthetic sutures may give improved results; this has become the accepted technique in many centres, and can be recommended. As far as the routine use of antibiotics is concerned, we would like to emphasize that antibiotics in our institution are administered per indication only, ie, mainly for colonic perforations where soiling and extended time lapse are complicating factors. This policy, we believe, should reduce the early development of resistant strains.

- Colon

Exteriorization of sutured colon is an excellent procedure for lesser injuries of the colon. Contraindications of exteriorization and of intraperitoneal primary closure as mentioned should, however, be adhered to carefully to prevent serious problems during management.

- Rectum

The very important rectal injuries and their management as outlined by the author, is in our opinion the safest protocol to follow. The Lloyd-Davies position is imperative under these circumstances, and early repair of rectal wall defects and the sphincter mechanism as far as possible, is strongly recommended. Lavage of the distal rectosigmoid segment should be continued until the anal effluent is clear.

- Spleen

Injuries to the spleen are managed conservatively as far as possible. Preoperative assessment of the injury by CT scan could also help the surgeon to decide what line of treatment to adopt. Splenic conservation is best achieved by full mobilization of the

spleen, and application of a vascular clamp to the pedicle in order to evaluate and repair defects or to perform partial splenectomy.

- Liver

CT scanning has again been a very useful non-invasive investigation in our hands, especially as a follow-up procedure in conservative management. Wide exposure and extensive mobilization of the liver remain the mainstays in the surgical management of severe liver trauma. Removal of completely devitalized liver tissue and firm packing with large numbers of abdominal swabs to effect tamponade and control haemorrhage is done at the initial surgery. Re-opening of the abdomen after 12-24 hours under fully controlled circumstances, with definitive surgery, is then a much safer procedure.

- Kidney

Renal injuries and their management have been a controversial subject for some time. Although arteriography is indicated early in renal injury, it could be a problem in some institutions where this facility is not readily available. Single film bolus dose intravenous urogram should then be resorted to first. In an actively bleeding kidney arteriographic evaluation with immediate embolization is the accepted treatment method of choice. Where this is not available, the urgent excretory urogram in theatre before opening the abdomen is strongly recommended to establish bilateral renal function. CT with contrast, if available, has become an important preoperative investigation.

- Duodenum and Pancreas

Pancreatic and duodenal injuries are best assessed by CT scanning, but again should only be used in stable patients. At laparotomy, a suspected duodenal injury should be examined thoroughly and full mobilization of duodenum is imperative. One must, however, be aware of the extensive attempts at mobilizing a contused duodenum where damage to frail blood supply may lead to necrosis or impaired healing of bowel.

We have never resorted to diverticulization in severe duodenal trauma, because the operation involves a partial gastrectomy. However, pyloric exclusion with purse-string suture closure of the pylorus and gastroenterostomy can be recommended. Roux-en-Y drainage of the proximal end of the distal pancreas with closure of the proximal end of the distal pancreas with closure of the proximal side near the duodenum is another option in treating severe lacerations of the neck of the pancreas. Whipple's procedure should not be embarked upon in severe trauma of the head of the pancreas and duodenum because of the high mortality. It is, however, very important to fully mobilize the pancreas and duodenum to evaluate injury to these areas adequately.

- Retroperitoneal Haematoma

Retroperitoneal haematoma is to be explored only in the central and upper areas, unless special indications are present. Types I and III haematomas often arise from structures where surgical repair and control of bleeding are a challenge to the surgeon, and should be approached with all due respect. Penetrating injuries to the back with the possibility of intra-abdominal or retro-peritoneal injury should be managed with great

concern. These patients should always be admitted and evaluated clinically and radiologically at regular intervals to prevent serious complications and mortality.

- Pelvic Injuries

Injuries to the urethra should never be catheterized, because of possible further damage to the ruptured urethra. Under these circumstances the surgeon should resort to suprapubic catheter placement. Urethrograms when indicated should be done as a sterile procedure under low pressure, so that leakage will be minimized as this may give rise to an increased risk of fibrosis.

If total disruption of urethra becomes evident, immediate primary repair is performed; if incomplete rupture is shown, then conservatism and careful follow-up is recommended. Railroading is condemned and should not be used except under special circumstances. At our institution, however, it is recommended that such patients be investigated by intravenous urography with voiding cysto-urethrogram in the first instance. A retrograde urethrogram may, however, then become necessary.

Penetrating injuries to the diaphragm, especially from lower thoracic stab wounds, could pose a severe problem and under these circumstances we recommend peritoneal lavage which, if positive, would indicate damage to the diaphragm necessitating laparotomy. False negative results may occur in up to 20% of cases, but we feel that this investigation remains a valuable asset in this diatgnostic dilemma.

Injuries to the gall bladder in our experience may break down after primary suture, and we would advise cholecystectomy or if this was not possible at least temporary cholecystostomy through the defect. Through-and-through injuries of the gall bladder are common and should be looked for carefully, also in the bed of the gall bladder.

The surgeon in training should be encouraged to approach the perplexing problem of abdominal injury with enthusiasm and responsibility, so as to achieve a successful outcome in the management of these patients.

Chapter 10.5: Medical Conditions Simulating Acute Abdominal Emergencies

D F du Toit

Introduction

It is important that the attending surgeon be aware that patients admitted to hospital as surgical emergencies could be suffering from an important underlying medical condition that may mimic or simulate a surgical condition, in particular an acute abdominal emergency. It must be remembered that laparotomy itself carries risks and some disorders are adversely influenced by anaesthetics and operations. The surgeon must be sure that he is not operating on a patient with an undiagnosed and potentially serious medical disorder which has been misinterpreted as a surgical emergency. Pain referred to the abdomen from another part of the body, ie, the chest or spine, must always be considered in the differential diagnosis of upper abdominal pain. A careful examination of the chest and its contents is imperative in order to exclude important intrathoracic pathology such as acute

coronary thrombosis, pneumothorax and acute pneumonia with diaphragmatic pleurisy that mimic abdominal pain.

Although medical conditions closely imitate the symptoms and signs of intra-abdominal pathology in adults (table 10.5.1) and children (table 10.5.2), it must be remembered that acute medical and surgical conditions can occur simultaneously.

Table 10.5.1. Medical Conditions Stimulating Acute Abdominal Emergencies

Respiratory

--> Pneumonia with diaphragmatic pleurisy
--> Pulmonary embolism and infarction
--> Acute spontaneous pneumothorax

Cardiac

--> Acute myocardial infarction
--> Acute pericarditis

Metabolic and Endocrine

--> Acute porphyria
--> Hyperlipidaemia
--> Diabetic ketoacidosis
--> Acute adrenal crisis
--> Familial Mediterranean fever
--> Haemochromatosis
--> Lead intoxication
--> Uraemia

Gastrointestinal

--> Gastroenteritis (viral, bacterial, protozoa)
--> Amoebiases (hepatic, colonic)
--> Acute hepatitis
--> Ascariasis

Blood Disorders

--> Sickle cell anaemia
--> Haemophilia
--> Acute haemolytic crises
--> Polycythaemia vera
--> Henoch-Schönlein purpura

Nervous System

--> Herpes zoster
--> Nerve root compression (spinal osteoarthritis)
--> Tabes dorsalis
--> Spinal cord tumour

Other

--> Acute rheumatic fever
--> Polyarteritis nodosa (vasculitis)
--> Acute epidemic pleurodynia (Bornholm disease)
--> Disease of hip joint
--> Congestive cardiac failure and hepatomegaly
--> Renal conditions (pyelitis), urinary tract infections
--> Acute tonsillitis
--> Acute poliomyelitis
--> Typhoid fever
--> Malaria
--> Tuberculosis peritonitis
--> Food poisoning
--> Dorsal or lumbar vertebra osteomyelitis.

Thoracic Diseases

Intrathoracic causes of pain may be referred to the abdomen and be localized by the patient in the lateral abdominal region (segments Th9-L1) below the rib margin or centrally in the epigastrium (segments Th6-9).

Pneumonia and pleurisy can produce abdominal pain and rigidity. Aggravation of the pain by breathing suggests pleural irritation and that the pain is referred from the chest. Referral of the pain to the corresponding side of the lower chest wall and upper abdomen usually indicates involvement of the diaphragmatic surfaces of the pleura by the inflammatory process. Upper abdominal tenderness and even rigidity are due to referred pain resulting from involvement of the parietal pleura over the outer aspects of the diaphragm. History and physical examination will often reveal cough, chest pain, dyspnoea, fever, tachycardia, tachypnoea, diminished respiratory excursion, a pleural rub and signs of consolidation. A chest X-ray usually confirms the diagnosis. Pulmonary embolism and infarction may also occasionally produce abdominal pain due to irritation of the lower six thoracic nerves following pleural involvement.

Spontaneous pneumothorax is a rare cause of abdominal pain, pleural pain and shortness of breath being the principal symptoms.

In healthy persons the diagnosis is usually made easily and confirmed on chest X-ray which shows air in the pleural space accompanied by varying degrees of collapse of the lung.

Bornholm disease (epidemic pleurodynia) is usually caused by group B Coxsackie viruses and presents as an acute febrile infectious disease often associated with sharp, spasmodic pain overlying the intercostal region of the chest or the abdomen. Patients with abdominal pain may experience paroxysms of muscle spasm. The majority of patients are less than 30 years old of either sex and more than one family member may be affected at the same time during an epidemic. The hallmark of the disease is the spasmodic nature of the pain and during a paroxysm splinting of the chest or abdominal muscles may be present. The pain, not unlike that of myocardial ischaemia, is usually aggravated by coughing, turning or any other movement. The pain is usually bilateral, associated with localized tenderness to pressure and in some cases hyperaesthesia of the overlying skin. The condition should be considered a possibility in the absence of auscultatory findings in the chest and a negative chest X-ray. It is reported that despite the presence of abdominal pain and spasm, signs of peritoneal irritation are often absent. In some cases it is very difficult to exclude an acute abdomen as the condition can closely mimic abdominal pathology such as appendicitis, pancreatitis, acute cholecystitis and perforated peptic ulcer which may result in laparotomy.

The pain of acute myocardial infarction may be referred to the epigastric region and the condition may mimic perforation of a peptic ulcer, acute cholecystitis or acute pancreatitis. The patient may regard the pain as "indigestion" but on careful enquiry the pain is usually felt over the lower sternum and is described as a vice-like constriction. In a severe case the patient may be grey and cyanosed, sweating, distressed and collapsed, with a low blood pressure and feeble pulse. In most cases the pain is severe and continuous and the tachycardia and hypotension is not usually adequately explained by the essentially negative abdominal findings. Myocardial infarction should be strongly considered in any patient presenting with severe, prolonged chest pain of a gripping nature with radiation to the arms, throat and epigastrium, with changes in heart rate and blood pressure. The diagnosis of myocardial infarction is confirmed by typical ECG abnormalities and supported by enzyme changes. As the mortality of the condition is highest during the first day it is imperative that the condition be considered as a possible cause of abdominal pain. Abdominal operations in the presence of an acute myocardial infarction are associated with a prohibitive mortality due to cardiogenic shock, persistent arrhythmias and ventricular failure.

Patients with pericarditis with involvement of the diaphragmatic portion of the pericardium may present with left shoulder-tip pain and/or epigastric pain associated with true rigidity of the abdominal wall, but unaccompanied by other signs of abdominal disease. Other features include sternal or praecordial pain increased by movement and respiration and the presence of a pericardial rub. Patients with congestive cardiac failure and severe hepatomegaly may present with abdominal pain which does not cause true rigidity of the abdominal wall. Other distinguishing features of cardiac failure include distended neck veins, cardiomegaly, peripheral oedema, triple rhythm, basal crepitations and hepatomegaly.

Spinal Conditions

The pain of Herpes Zoster (Shingles) may mimic acute appendicitis or cholecystitis and in the pre-eruptive stage the diagnosis can be difficult. However, the diagnosis is straightforward after three or four days with the appearance of the vesicular eruption. In

the pre-eruptive phase the pain which may be intermittent or constant, is sharply delimited by the segmental distribution fo the affected nerves. During this stage tenderness or hyperaesthesia may be detected along the dermatome affected. Involvement of the lower thoracic intercostal nerves usually produces a bandlike pain and paraesthesia over the area of the abdominal wall supplied by the nerves. The pain is of nerve root distribution and stops in the midline. Often only unilateral epigastric pain is experienced.

The gastric or visceral crises of Tabes dorsalis, now rarely seen, may mimic intrinsic visceral disease. The manifestations of lightning pain, the finding of the Argyll-Robertson pupil and the reduced ankle jerks should alert the surgeon to the possibility of the condition. An acute abdomen may occur in a tabetic subject and in these cases suppurative appendicitis or a perforated peptic ulcer may go undiagnosed. Therefore operation should be advised if the local signs are constant and signs of peritoneal irritation develop.

Other conditions that may mimic an acute abdomen include meningitis, anterior poliomyelitis, acute osteomyelitis of the dorsal or lumbar vertebrae, Potts' disease of the spine, spondylitis and osteoarthritis of the spine.

Endocrine and Metabolic Disorders

In the pre-comatous phase the diabetic may present with severe abdominal pain which may simulate acute appendicitis or a perforated peptic ulcer. The diagnosis can be rapidly confirmed by testing the urine for glucose and determining the blood glucose levels by haemaglucotest or by conventional laboratory methods. Of importance is that diabetics are as likely to develop an acute intra-abdominal emergency as anyone else. If the diabetes is the cause of the abdominal symptoms, the condition is usually self-limiting and rapidly improves with effective treatment of the diabetic state. If the abdominal signs do not subside or progress despite optimum treatment of the diabetes, a severe underlying pathological abdominal condition which may need urgent surgical intervention must be considered.

Colicky abdominal pain is often the initial or most prominent symptom of a patient with acute intermittent porphyria. Although abdominal tenderness and distension may be present, the abdomen usually remains soft. The attacks of acute pain may simulate acute appendicitis or intestinal obstruction. Other features include peripheral neuropathy, muscle weakness, paralysis, psychiatric abnormalities and seizures. The condition may be precipitated by a variety of drugs including barbiturates and may become manifest during pregnancy or during fasting. The diagnosis is confirmed by the detection of an excess of aminolaevulinate (ALA) and porphobilinogen (PBG) in the urine (Watson-Schwartz test). During acute porphyria the Watson-Schwartz test is always strongly positive. Porphyria should always be considered in the presence of unexplained abdominal pain as the condition has been mistaken for acute cholecystitis, peptic ulcer, appendicitis, intestinal obstruction and pancreatitis. Variegate porphyria (porphyria cutanea tarda or South African genetic porphyria), an hereditary condition, usually manifests with chronic cutaneous lesions, abdominal pain and neuropathy. As in other forms of porphyria, barbiturates, sulphonamides and general anaesthetics may be aggravating or precipitating factors. Typical findings in variegate porphyria is the increased amounts of copro- and protoporphyrin in the bile and faeces. As in acute intermittent porphyria, elevated urinary

levels of ALA and PBG are present in acute attacks. Other metabolic and endocrine conditions that may mimic an acute abdomen include acute adrenal insufficiency due to adrenal haemorrhage or infarction, haemochromatosis, lead intoxication, uraemia and hypercalcaemia.

Haematological Disorders

Patients with coagulation defects such as Haemophilia, Christmas disease and von Willebrand's disease may present with acute abdominal pain resulting from extraperitoneal or retroperitoneal haematoma, bleeding within the psoas sheath or mesentery which may mimic acute appendicitis. In some cases differentiating features between intra-abdominal haemorrhage with haematoma formation and acute appendicitis may be extremely difficult emphasizing the importance of obtaining a detailed history and performing repeated examinations of the abdomen. Haemophiliacs may present with acute abdominal pain due to intussusception caused by haematoma development in the bowel wall.

Agonizing episodes of severe abdominal pain and fever simulating acute appendicitis and other urgent abdominal disorders are typical in black patients with sickle cell anaemia, a hereditary type of chronic haemolytic anaemia. In some patients the condition can mimic an acute abdomen very closely with tenderness and rigidity of the abdomen often associated with an elevated leucocyte count in excess of 20000/mm^3, the latter often found in patients with sickle cell anaemia without acute symptoms. Acute abdominal symptoms may follow intramural haematoma formation and gross bowel mucosal haemorrhage in patients with Henoch-Schönlein purpura, an allergic vasculitis seen in children associated with streptococcal infections. Other important features of the condition include purpura or ecchymosis of the skin, joint pain and renal involvement. The condition may rarely present with an acute abdomen due to underlying intussusception following haemorrhage within the bowel wall.

Other conditions that may be complicated by abdominal pain include acute haemolytic anaemia with a crisis, systemic lupus erythematosus, intra-abdominal haemorrhage and rectus sheath haematomas in patients on anticoagulation treatment and patients with polycythaemia vera, resulting in splenic infarction and mesenteric thrombosis.

General and Other Intraabdominal Diseases

Renal tract conditions including urinary tract infections such as acute pyelonephritis and cystitis are important causes of abdominal pain, particularly in the paediatric age group.

Liver diseases that often present with acute right upper hypochondrium pain include amoebic liver abscess and acute infective hepatitis.

Infective causes include typhoid fever, cholera, amoebiasis and tuberculosis, yersinia and campylobacter enteritis. Collagen diseases such as periarteritis nodosa may present with acute abdominal pain and be confused with appendicitis. Patients with hereditary angioneurotic oedema and eosinophilic disease of the gut may also present with intermittent episodes of abdominal pain.

Parasitic infestation with ascariasis (bolus obstruction) and patients with malaria in the tropics may present with severe abdominal pain. In the latter condition the diagnosis is confirmed by the clinical presentation, especially the type of fever, together with examination of a blood smear.

Food poisoning and acute gastroenteritis, particularly in children, may closely mimic an acute abdomen. In the paediatric age group acute tonsillitis and associated mesenteric adenitis remains important in the differential diagnosis of acute appendicitis.

Patients from the Mediterranean or of Middle East origin suffering from familial Mediterranean fever, an inherited disease of unknown etiology, frequently present with abdominal symptoms indistinguishable from an acute abdomen and many have undergone a laparotomy, some more than once. Usually no abnormalities are found at laparotomy. Other features of the disease include self-limiting attacks of fever accompanied by abdominal pain, pleuritis and arthritis. A late complication is the development of amyloidosis.

Other non-surgical causes include hyperlipidaemia (types 1 and 5) and following black widow spider (*Lactrodectus mactans*) bites which produce muscle spasm, abdominal pain and rigidity suggesting an acute abdominal emergency. Heroind addicts occasionally present with an acute non-surgical abdomen which may mimic an acute surgical catastrophe. Difficulty may be experienced in differentiating between genuine and feigned abdominal symptoms when dealing with this notoriously unreliable group of patients.

Paediatric Causes

The problem of abdominal pain in children (table 10.5.2) is often a difficult and complex subject.

The commonest non-organic causes of abdominal pain in otherwise well infants, are wind and evening colic which maybe mild in nature or severe enough to cause rhythmical screaming attacks which may be confused with an underlying intussusception of the bowel.

In older children important medical conditions mimicking an acute abdomen include parasitic infestation (ascariasis, amoebiasis, urinary tract infection, food poisoning, gastroenteritis, rheumatic fever and lobar pneumonia with referred pain to the abdomen due to pleurisy).

Necrotising enterocolitis, a complication observed in infants who have been subjected to stress from a variety of conditions, usually presents with abdominal pain and distension with the passage of bloody diarrhoea. Intramural gas on abdominal X-ray is a typical finding. The condition may mimic other abdominal pathology, and surgical exploration is indicated if pneumoperitoneum or suspected peritonitis develops.

A rare cause of abdominal pain in children is haemorrhagic necrosis of the caecum or surrounding bowel (typhlitis), a condition occasionally seen in patients with end-stage leukaemia or aplastic anaemia and in children with leukaemia receiving chemotherapy.

Table 10.5.2. Medical Conditions Stimulating Acute Abdominal Emergencies in Paediatric Surgical Practice

--> Wind and evening colic
--> Necrotizing enterocolitis
--> Typhlitis (as with leukemia, aplastic anaemia)
--> Gastroenteritis
--> Ascariasis
--> Lobar pneumonia and pleurisy
--> Mesenteric lymphadenitis (tonsillitis)
--> Urinary tract infection
--> Rheumatic fever
--> Acute nephritis
--> Infectious hepatitis
--> Glandular fever
--> Anaphylactoid purpura
--> Sickle cell anaemia crises
--> Infectious diseases (prodromal stage)
    - measles
    - poliomyelitis
    - brucellosis
    - typhoid fever
--> Food poisoning.

Comment

Medical Conditions Simulating Acute Abdominal Emergencies

P Perdikis

The author quite rightly stresses the importance of a full history, thorough examinations and continual awareness of the medical conditions which mimic the acute surgical abdomen. However, in spite of this, surgeons are still led into operating on these problems.

In 68% of abdominal emergencies a definite diagnosis is made, in a further 16% diagnoses such as spastic colon, mittelschmerz and mesenteric adenitis are suggested while 16% leave hospital without any diagnosis being made. Twenty-four per cent of acute abdomens are due to medical causes. Where a laparotomy is done in which there is no firm radiological or biochemical support, 20% result in an unnecessary operation. The author has not suggested how these figures can be improved. It has been shown that the computer may improve diagnostic accuracy of a consultant's opinion, albeit only by 5-8%, but perhaps with a better input into the computer these results may further be improved. Ultrasonography has greatly improved emergency diagnosis in upper abdominal crises. It certainly has helped in assessing emergencies in females and there is evidence that ultrasonography can improve the diagnosis of acute appendicitis. Laparoscopy too has aided the surgeon in the diagnosis of pelvic and appendiceal pathology and recently it has been used in right hypochondrial pain in establishing the diagnosis of perihepatitis due to chlamydial infection in the Curtis Fitz-Hugh syndrome which is much more common than

previously thought. Needle aspiration has been of tremendous aid in the diagnosis of primary and starch peritonitis as well as pancreatitis.

There certainly is a risk in operating on the elderly with a host of medical conditions, but this risk also applies to the younger age groups who may be suffering from potentially lethal conditions such as acute myocarditis or rheumatic fever when presented to the surgeon. Operating on cirrhotic patients, both electively and under emergency conditions, carries a prohibitive risk. In non-shunt procedures mortality figures are quoted in the region of 30%. To date there has been no prospective study evaluating immediate and delayed morbidity of the negative laparotomy.

The author has given an extensive listing of causes giving rise to the acute medical abdomen. However, the mechanisms whereby the various groups of conditions give rise to these symptoms are not always clear. In many the mechanisms are easily understood but in others like tabetic crisis or diabetic ketoacidosis they are not clear.

In considering the aetiological agents, it should be stressed that the infective intraluminal agents form the biggest group of surgical mimers and perhaps it should be in this direction that future research, particularly rapid viral identification, should be directed. Primary peritonitis genesis causes 2.19% of all abdominal emergencies and 10% of paediatric abdominal emergencies.

Chapter 10.6: Alimentary Tract Stomas

L C J van Rensburg

Alimentary tract stomas, whether the anatomical site is the oesophagus or colon, have the same principle in common, ie, an opening of a hollow viscus either to the exterior of the body (ie, colostomy) or between two hollow organs (ie, gastroenterostomy). The purpose of the stoma is variable. It could be for decompression (colostomy), used for feeding (jejunostomy) or part of a practised definitive procedure (gastroenterostomy). Alimentary tract stomas connected to the body exterior may be by the tube (tube jejunostomy) or by a direct muco-cutaneous method (permanent end-colostomy). Certain stomas will require collection and measurement of the luminal content (tube duodenostomy); in others the luminal content is merely collected to be discarded (end-colostomy). Stomas can be temporary (loop transverse colostomy) or permanent (end-colostomy after abdomino-perineal resection).

Classification

Several classifications are acceptable:

Anatomical Location	Procedural

Anatomical Location

External
--> Oesophagostomy
--> Gastrostomy
--> Duodenostomy
--> Cholecystostomy
--> Jejunostomy
--> Ileostomy
--> Caecostomy
--> Appendostomy
--> Colostomy

Internal
--> Oesophagogastrostomy
--> Oesophagojejunostomy
--> Gastroenterostomy
--> Cholecysto
    - gastrostomy
    - jejunostomy
--> Choledocho
    - gastrostomy
    - duodenostomy
    - jejunostomy
--> Duodenojejunostomy
--> Jejunojejunostomy
--> Ileocaecostomy
--> Ileocolostomy
--> Ileorectostomy
--> Colocolostomy

Procedural

--> Feeding
    - oesophagostomy
    - gastrostomy
    - jejunostomy (duodenostomy)
    - ileostomy
--> Decompression
    - oesophagostomy
    - gastrostomy
    - duodenostomy
    - cholecystostomy
    - jejunostomy
    - ileostomy
    - colostomy
--> Definitive
    - oesophagogastrostomy
    - oesophagojejunostomy
    - oesophagocolostomy
    - gastro-enterotomy
    - cholecysto-enterostomy
    - entero-enterostomy
    - entero-colostomy
    - entero-rectostomy

Temporary or Permanent Stomas

Temporary
--> Oesophagostomy
--> Gastrostomy
--> Cholecystostomy
--> Duodenostomy
--> Jejunostomy
--> Ileostomy
--> Caecostomy
--> Loop colostomy
--> End-colostomy

Permanent
--> Gastrostomy
--> Ileostomy
--> Colostomy
--> Definitive procedures
      - oesophagogastrostomy
      - oesophagojejunostomy
      - oesophagocolostomy (bypass)
      - gastro-enterostomy
      - cholecysto-enterostomy
      - entero-enterostomy
      - entero-colostomy
      - entero-rectostomy.

Oesophagostomy

There are three main indications for oesophagostomy, namely:

--> congenital oesophageal atresia
--> oesophageal perforations
--> certain cases of carcinoma.

In type I congenital oesophageal atresia there is a long atretic segment making primary anastomosis impossible. A temporary proximal end cervical oesophagostomy is performed for later interpositional reconstruction. In certain injuries of the oesophagus, mainly long lacerations and delayed diagnosis, a diverting oesophagostomy is indicated. Where the injury involves the thoracic oesophagus, the laceration is repaired via thoracotomy, the oesophagus distal to the laceration ligated and the cervical oesophagus mobilized to fashion a side-oesophagostomy. The oesophagus is carefully mobilized in continuity being careful to preserve its blood supply. A 5 cm longitudinal incision is performed and the mucosa and muscle sutured to the subcutaneous fascia and skin with interrupted 4-0 non-absorbable sutures.

Occasionally a diverting end-oesophagostomy is performed in carcinoma of the oesophagus. In the rare situation where the carcinoma has perforated, oesophagectomy is performed leaving the cervical oesophagus as a temporary stoma before reconstruction.

Gastrostomy

Temporary

Temporary tube gastrostomy is used mainly to feed the patient and secondly to decompress the stomach.

The Stamm gastrostomy is most commonly used because of its simplicity and safety. The site chosen is the mid anterior point of the stomach. A small stabwound is made for insertion of a 20-26 Fr, Malecot, de Pezzar or Foley catheter. Two concentric

purse-string sutures are used to invert the stomach wall around the tube. The tube is brought out through the abdominal wall in the left upper quadrant. The stomach wall close to the tube exit is sutured to the peritoneum thus avoiding retraction of the stomach and peritoneal soiling. Feeding can be commenced the following day. Tube irrigation to dislodge mucus and food residue is advisable on a regular basis. The tube is simply removed when indicated.

Witzel Gastrostomy

In this operation a tunnel is made on the anterior wall of the stomach ostensibly to reduce soiling from the tube.

Marwedel's Gastrostomy

A modification of the Witzel method. A 5 cm incision is made through the seromuscular layer of the anterior wall of the stomach. At the lower end of the incision a small opening is made through the mucosa for insertion of the tube and fixed by a catgut suture. The tube is buried in the tunnel by interrupted sutures.

Permanent Gastrostomy

The standard method is to bring a tube of stomach wall onto the surface of the abdominal wall with the mucosa forming the inner layer of the tube. Unlike tube gastrostomy this mucus fistula will not close and can be used for feeding for a long time.

Janeway Gastrostomy

A flap of stomach wall is formed with its base on the greater curvature. The stomach defect is closed in two layers with the sutures continued into the flap converting it into a tube. The tube is brought through a stabwound onto the abdominal wall and a mucocutaneous suture line fashioned. The tube should be large enough to accomodate an 18-20 Fr tube and the opening through the abdominal wall adequate to avoid constriction of the tube.

Beck-Jianu Gastrostomy

A tube is fashioned from the greater curvature of the stomach. A long curved incision is made on the greater curve of the stomach to create a tube with the opening at the pyloric end and the part entering the stomach at the upper end of the greater curvature. The tube is brought onto the abdominal wall as with the Janeway gastrostomy.

Spivack Method

This gastrostomy was devised as a leak-proof method with an internal walve. An anterior gastric tube is created and a collar of stomach at the base of the tube is inverted by interrupted sutures.

Indications for Gastrostomy

- Temporary

Feeding of patients undergoing oesophago-pharyngeal surgery or for postoperative decompression of the stomach instead of a nasogastric tube.

- Permanent

Permanent swallowing disability because of stroke, cerebral palsy or permanent loss of the cervical oesophagus.

Enterostomy

Either jejunostomy or ileostomy.

Indications

- Temporary

Enterostomy feeding, as a decompression vent proximal to an anastomosis, ie, Park's poch procedure; as a diversion of small-bowel content, ie, acute toxic megacolon in ulcerative colitis or amoebic colitis. After injuries of the small bowel or (R) colon.

- Permanent

End-ileostomy after proctocolectomy. As a conduit, ie, external choledochojejunostomy.

Methods

As a temporary measure the skin incision is usually in the left upper part of the abdomen.

Witzel Technique

- A loop of proximal jejunum 20 cm from the ligament of Treitz is isolated and controlled by soft bowel clamps.

- A 3-0 chromic catgut purse-string suture is placed on the antimesenteric aspect.

- A small incision is made through the centre for placing a catheter.

- The suture is tied and the catheter buried in a serosal tunnel with interrupted sutures.

- The tube is brought out through the abdominal wall and the loop of jejunum secured to the parietal peritoneum to avoid soiling and volvulus formation.

Stamm Technique

The tube is placed through the centre of two 3-0 chromic catgut purse string sutures. The rest of the procedure is similar to the Witzel technique.

The tube is simply removed at the desired time allowing the fistula to close within three or four days.

Needle Catheter Jejunostomy

- A large bore needle (16 g) is tunnelled through the subserosa of the isolated loop before entering the lumen.

- A catheter is threaded through the needle and the needle withdrawn.

- The jejunum is fixed to the parietal peritoneum and the catheter exteriorized.

- This method is used for enteral feeding and not for decompression.

End-Ileostomy

- Temporary

One example is high-velocity gunshot wounds of the right colon where resection of the injured bowel is performed followed by an end-ileostomy and transverse colon mucous fistula. Further indications are toxic megacolon and amoebic colitis, as an exclusion procedure. In some patients with caecal carcinoma perforation where resection is performed and an ileostomy and transverse colon mucous fistula preferred to primary anastomosis because of sepsis endangering anastomotic leakage.

Technique

The loop should be adequate in length to reach the skin usually in the right iliac fossa conveniently placed for attachment of ileostomy bags (see Brooke's ileostomy technique).

Permanent Ileostomy

Siting of the stoma is most important and must not be done too close to the umbilicus, the iliac crest, rib margin or so low down that hip flexion could dislodge the appliance.

Preoperative marking of the ileostomy site by placing the appliance in various positions is essential.

Indications

Ulcerative colitis after proctocolectomy and to a lesser extent for Crohn's disease. In familial polyposis after proctocolectomy.

There are two types of ileostomy, transperitoneal and extraperitoneal (Brooke's ileostomy).

Technique

- Transperitoneal ileostomy

Recommended for granulomatous bowel disease and where it is difficult to strip the peritoneum from the abdominal wall due to previous surgery. A disc of skin 2 cm in diameter is excised. A similar disc of subcutaneous tissue is similarly removed. The opening through the muscle can be circular or cruciate. In delivering the terminal ileum through the defect, there should be adequate length (4-6 cm protrusion) and no torsion of the loop. The mesentery of the ileum is sutured to the transverse abdominis muscle with a few interrupted sutures to avoid prolapse. The rest of the mesentery is attached to the parietal peritoneum closing the lateral gutter and further fixation of the loop.

- Extraperitoneal technique

An extraperitoneal tunnel is created from the opening in the abdominal wall beneath the parietal peritoneum. The ileum is fixed to the abdominal wall with interrupted sutures to avoid prolapse. The peritoneal defect is closed over the existing loop.

In both types of ileostomy the distal end of the ileum is reverted upon itself and the margins sutured to the skin. The ileostomy appliance is fitted immediately and should be carefully placed to avoid excoriation of the skin.

Continent Ileostomy (Kock)

With sphincter-saving operations and pelvic pouch procedures the vogue for continent ileostomy has changed. It is contraindicated in Crohn's disease, children and patients with low intelligence. The procedure can be performed at the time of proctocolectomy for ulcerative colitis or as a conversion of an end-ileostomy.

In principle a 30 cm U-loop of terminal ileum is sutured together and opened to create a pouch. Distal to the loop a 15 cm segment of ileum is used to create an inverted nipple for continence. The pouch is drained by catheter postoperatively for four weeks before its removal. The patient is taught to empty the pouch by catheter three to four tinmes per day.

Complications of End-Ileostomy

--> stenosis and retraction
--> fistulae
--> prolapse
--> small-bowel obstruction
--> recurrent disease (Crohn's disease)
--> the technical complications of bleeding and improper placing should be avoided.

Loop Ileostomy

Indications

Toxic megacolon to divert the faecal stream, colonic obstruction in preference to a caecostomy, to protect an ileo-anal anastomosis, trauma, and where an end-ileostomy is difficult to construct due to a short mesentery and in obese patients.

Technique

- The loop is brought to the exterior through a disc-like opening as for end-ileostomy at a suitable site in the right lower abdomen.

- A rod is placed through the mesentery to support the loop.

- The loop is opened and the stoma is sutured with interrupted sutures to the skin.

- The rod is removed after four to seven days.

- Closure of the loop usually requires resection.

Caecostomy

Caecostomy has been condemned as a poor operation and there is severe criticism of its use to protect a distal anastomosis. There are three main indications, namely for:

- pseudo-obstruction of the colon where the caecum is very distended

- caecal volvulus after derotation

- injuries of the caecum.

Tube Caecostomy

This type of caecostomy has the problems of tube blockage and dislodgement of the tube with peritoneal contamination.

Technique

--> The caecum is isolated from the rest of the abdominal cavity with packs. With severe distension of the caecum needle decompression is advised.

--> Two purse-string sutures are inserted for securing a large Malecot or Foley catheter.

--> The caecal wall is attached to the parietal peritoneum to prevent leakage and abdominal soiling. The tube should be securely fixed to the skin and requires frequent washouts to avoid blockage.

--> On removal of the tube, the caecal fistula gradually closes.

Exteriorization Technique

--> The caecum is approached through a muscle cutting incision in the right iliac fossa.

--> The peritoneum is opened to reveal a bulging caecum. Needle decompression may be required.

--> Seromuscular sutures are inserted between the caecum and the peritoneum before the caecum is opened.

--> The caecal wall is then sutured with interrupted sutures to the skin.

Colostomy

Indications

--> Colonic obstruction due to congenital abnormalities such as rectal atresia and Hirschprung's disease, chronic diverticular disease with stenosis, inflammatory bowel disease, endometriosis, neoplasms - mainly constricting carcinomas, radiation injury, colonic ischaemia.

--> Perforations of the colon due to inflammatory bowel disease, diverticular disease, ulcerative colitis, Crohn's disease and ischaemic colitis.

--> Colonic injuries, where primary closure is inadvisable such as severe destruction of the colon, serious concomitant injuries, and massive faecal contamination.

--> As a definitive part of an operation such as an abdomino-perineal resection or temporary loop colostomy to protect a distal anastomosis.

--> Miscellaneous indications are severe perineal injuries and sigmoid volvulus.

The colostomy is either temporary or permanent. A loop colostomy is usually temporary and defunctioning. A permanent colostomy is usually an end-colostomy. End-colostomies, on the other hand, can also be temporary, for example where complete defunctioning is necessary and the two ends are brought out at different sites, or associated with a Hartmann's procedure where an end-colostomy is fashioned to be anastomosed to the closed rectal stump as a later procedure.

Transverse Colostomy

This procedure is usually a temporary colostomy. The indications are for colonic obstruction, trauma, disruption of a distal colonic anastomosis or to protect an anastomosis.

Technique

--> The general principles of location of the stoma are important.

--> The loop can be brought to the exterior through either the right or left split rectus muscle. The left side and the left part of the transverse colon is theoretically better as the stool is more formed and because of the relative fixity of the splenic flexure, prolapse occurs less often.

--> The omentum is freed from the colon to permit exteriorization without tension.

--> A rubber tube or Penrose drain is passed through the mesentery close to the bowel wal to deliver the loop.

--> A plastic rod or a short length of rubber or latex tubing is passed through the mesentery to prevent retraction of the loop.

--> The colon is opened longitudinally and the bowel edges sutured to the skin.

--> The colostomy ring and bag is applied.

--> The rod is removed once the oedema has settled.

Sigmoid Colostomy

Indications

--> carcinoma of the rectum
--> congenital abnormalities
--> perineal trauma
--> Crohn's disease
--> problematic fistulae in ano (rarely).

The technique is similar to that of transverse colostomy.

End-Colostomy and Mucous Fistulae

Although a well constructed loop colostomy could be non-functioning, two separate openings are more reliable. In rectal atresia it is advisable to perform either a transverse or sigmoid colostomy by dividing the colon and bringing out the proximal loop as an end-colostomy and the distal loop as a mucous fistula. The same technique is used for severe colonic trauma following gunshot wounds or vascular disruption of the colonic blood supply where primary anastomosis is contraindicated. The injured colon is resected and the distal loop either suture-closed or exteriorized as a mucous fistula.

End-Colostomy

The usual indication for an end-colostomy is after an abdomino-perineal resection for carcinoma of the rectum. Again it is important to have marked the stoma site on the abdominal skin prior to surgery to secure the ideal position.

Technique

--> The sigmoid colon is divided so that the blood supply is not jeopardized and that the length is adequate, thus avoiding too short a loop or redundancy.

--> A disc of skin and subcutaneous fat is removed.

--> A cruciate incision is made in the rectus sheath, the rectus muscle is split longitudinally followed by an adequate opening in the peritoneum. The opening must admit two fingers and 3-0 chromic catgut or polyglycolic acid sutures are placed through the full thickness of the bowel and skin.

--> The colostomy ring and bag is applied at the end of the operation.

General Principles

The location of the stoma is very important in the management of the colostomy. Sites to be avoided are the groin, the waistline, iliac crest, skin folds and abdominal scars. The build of the patient must be considered. A large pendulous abdomen needs extra care in siting the stoma.

Cholecystostomy

Indications

The main indications are acute cholecystitis and cholangitis. In the exceptional case of gallstone pancreatitis where choledochotomy is not possible, cholecystostomy may be performed. In the elderly and indigent patient and where there are real technical difficulties, cholecystostomy is a safe option rather than cholecystectomy. This procedure can be done under local anaesthetic.

Technique

The abdominal incision can either be a right subcostal, paramedian or midline incision.

--> The fundus of the gall-bladder is packed off from the rest of the abdominal cavity to prevent peritoneal soiling.

--> The fundus is punctured by a wide-bore needle attached to suction tubing to aspirate the bile.

--> A cholecystostomy is performed to remove stones from the gall-bladder.

--> A large Foley or Malecot catheter is inserted into the gall-bladder and the opening closed either by interrupted sutures or a purse-string suture.

--> The cholecystostomy tube is brought out through an incision in the abdominal wall and fixed to the skin with a suture.

A tube cholecystogram may be performed before removing the tube to establish whether resiudal stones are present in the gall-bladder. The tube should not be removed before the tenth postoperative day.

Choledochostomy

For practical purposes choledochostomy means drainage of the common bile duct by T-tube. Transhepatic biliary drainage is a separate entity.

Indications

The main indication is for removal of stones from the common bile duct. It may also be necessary for the removal of biliary parasites such as *Ascaris lumbricoides* or bloodclot in cases of haemobilia. Cholangitis, acute pancreatitis, biliary strictures and bile duct carcinoma are further indications for choledochostomy.

Technique

This may vary but the usual technique is as follows:

--> Once the common bile duct has been exposed, two stay sutures of fine material (ie, 3-0 chromic catgut) are inserted about a half a centimetre apart on the anterior surface of the duct before opening the duct between the sutures.

--> The duct is opened longitudinally, the opening being large enough for the purpose of exploration of the duct and to facilitate drainage. The commonly used T-tube is made from latex and comes in various sizes catering for the surgeon's choice. The average size used is either a 16Fr or 18Fr gauge.

--> The length of the intrabiliary segment must be carefully assessed before insertion. This segment can be modified by excising the back wall, by a longitudinal slit or an opening opposite to where the limb is inserted.

--> The choledochostomy is closed either above or below the exit of the T-tube limb with either continuous or interrupted sutures.

--> The T-tube is brough out through the abdominal wall through an incision and secured to the skin by suture.

--> The bile is collected in a bag and measured daily.

--> The tube is irrigated if necessary and is usually removed on the tenth postoperative day, preceded by a T-tube cholangiogram.

Duodenostomy

For practical purposes duodenostomy is seldom other than a tube duodenostomy. The tube can be brought out through the end of the opened duodenum or through the wall of the duodenum.

Indications and Technique

One of the main and valuable indications is in the management of the "difficult" duodenal stump after partial gastrectomy for peptic ulcer where closure of the stump is difficult or impossible to close. There are two options. Where the stump cannot be closed or if a closure could lead to stump leakage, the duodenostomy tube can be inserted into the end of the duodenum and the duodenal wall closed around the tube, or the duodenum can be closed but because of the risk of suture disruption and leakage, the tube is inserted through a separate duodenostomy in the second part of the duodenum. The duodenostomy tube must not be too large and the insertion into the duodenum must be snug to avoid leakage. A suitable tube is a 14Fr Foley catheter; the bulb should not contain more than 2 mL of water to avoid obstruction of the duodenum. The tube is brought onto the abdominal wall through an incision in the right upper abdomen and secured to the skin by suture.

The next common indication is duodenal injuries, either isolated or in association with pancreatic trauma. The principle is to decompress the injured duodenum and to divert bile and pancreatic juice to allow healing of the sutured duodenum or to create a controlled fistula. The site of the duodenostomy could be above or below the injured bowel. The tube size is either a 14Fr or 16Fr of soft consistency such as latex or silastic where a long period of drainage is anticipated. Meticulous aftercare is necessary to avoid blockage of the tube, leakage and skin irritation.

Stoma Therapy

The role of a stoma therapist is now well established in most large institutions. The task of the therapist involves conselling the patient and explaining all the aspects of the stoma about to be performed by the surgeon. The psychological preparation is as important as the teaching and demonstration of self-management of the stoma. Modern appliances have eliminated many of the unpleasant and unsatisfactory elements of ill-fitting "bags" used in the past. The stoma therapist is instrumental in marking the most ideal site for the stoma, whether it be a colostomy or ileostomy, and is actively involved in the postoperative care.

Comment

Alimentary Tract Stomas

N J Laage

In constructing a stoma the following surgical principles should apply:

--> The stoma must be sited correctly.
--> The bowel must not be stretched to reach the body surface.
--> The mucosa should be sutured to skin to prevent stricture.
--> No spaces should be left which may lead to visceral obstruction and strangulation.

Spontaneous stomas may occur in many disease processes and examples of internal stomas include:

--> bilio-enteral following acute cholecystitis
--> gastrojejunocolic following gastroenterostomy
--> stomal ulceration
--> gastropancreatic as a consequence of complicated pancreatic pseudocyst
--> enterocolic in malignancies and colovesical in diverticular disease.

External stomas may occur in association with:

--> amoebiasis
--> Crohn's disease tuberculosis
--> and may also follow penetrating trauma.

Anastomotic breakdown is unfortunately a fairly common cause of iatrogenic stomas and the services of the stomatherapist are invaluable under these circumstances. Examples of stomas created inadvertently include gastroileostomy instead of gastrojejunostomy following subtotal gastrectomy resulting in severe metabolic consequences, and following segmental colon resection with closure of the proximal segment as a Hartmann and exteriorizing the distal segment as a colostomy with severe obstructive consequences.

With the development of stapling techniques and the colo-anal sleeve anastomosis, the outlook for many patients with rectal cancer has changed, thus avoiding a permanent colostomy. Total colectomy, mucosal proctectomy and pelvic pouch procedures with ileo-anal anastomosis also eliminate the need for a stoma in patients operated on for ulcerative colitis and familial polyposis.

Comment

Alimentary Tract Stomas

H H Lawson

In the end-type ileostomy such as may be used in the emergency situation following high velocity injuries, a peritonitis develops on the exposed serosal surface. This could lead to stenosis, and all the problems of so-called ileostomy diarrhoea. The beauty of the Brooks type ileostomy is eversion, which not only avoids this peritonitis, but also creates the adequate length on which the stoma therapist depends.

Care of the Skin

It may well be that a stoma therapist is not available. In the absence of the stoma therapist koryagum powder, and even old-fashioned aluminium paint can be very helpful pending the arrival of the stoma therapist.

Chapter 10.6: Appendix

Rehabilitation and Practical Management of Patients With Intestinal Stomas

Priscilla J d'E Stevens

The key to successful stoma surgery lies both in impeccable surgical technique and in the provision of adequate emotional and practical support for patient and family. All patients undergoing stoma construction either as an elective or emergency procedure, require the service of an informed stomaltherapy nurse as part of the multidisciplinary team. Her role includes the provision of pre-operative counselling and orientation, stomal siting, post-operative practical management and education, discharge planning and ongoing community and outpatient clinic care.

Preoperative Counselling and Orientation

Patients come from a wide range of social, cultural and religious backgrounds and each requires an individualized approach, involving a close family member or friend. Orientation methods include education on the normal anatomy and physiology of the gastrointestinal or genito-urinary tract, with a simplistic description of the passage of food and liquid between mouth and anus or kidney and urethra. By utilizing models and diagrams it is possible to delineate the part of the anatomy compromised by disease or injury and demonstrate the surgical plan and the site of the faecal or urinary diversion. Few patients have sufficient insight into the way their bodies function and therefore visual aids showing the normal healthy stoma and colour and site are helpful in creating awareness and allaying postoperative fears. When indicated, the prospective stoma patient may benefit from meeting a well-matched rehabilitated ostomate with a similar stoma, condition and social background.

Questions and concerns regarding sexuality, diet, prognosis, and lifestyle should be openly discussed. An introduction to simplistic pouching and skin care and the knowledge

that ongoing practical care and support will be available following discharge from hospital go far in helping a patient to accept the prospect of an intestinal diversion.

Stoma Siting

This aspect of preoperative preparation is vital to the success of the procedure. A poorly placed stoma can lead to leakage, odour, skin breakdown and a host of emotive and psychosocial problems. To achieve the ideal site acceptable to both patient and surgeon, the patient must be examined in the lying, sitting and standing position in order to note the changing body contours.

Criteria for Siting Intestinal Stomas

The chosen site should avoid:

- costal margins
- waistline
- umbilicus
- surgical incision
- obvious abdominal folds and creases
- scars
- anterior iliac crests.

Use a skin-protective wafer with a central hole and place on the proposed site to ensure the skin surface covered does not encroach the wound, or umbilicus. Once satisfied, mark the site with a skin pen.

Special Considerations

- Obese patients

Note the pendulous abdomen in the standing position - choose a site where the patient can see the stoma, let the patient sit to ensure that it is marked on the summit of any fat folds.

- Patients for radiotherapy

When radiotherapy is planned in intra-abdominal malignancy, place the stoma in a more lateral site and higher on the anterior abdominal wall to avoid as much of the radiation field as possible. Keep to the criteria for all stoma siting - keep the stoma within the rectus abdominis muscle.

- Wheelchair-bound patients - orthopaedic/neurological pathology

Site the patient in the wheelchair or wearing any braces or external appliance to ensure that the site does not encroach or compromise. Note any scoliosis or kyphosis and place the stoma in the most convenient site for patient self-management. This may mean siting on the opposite side to the conventional.

Practical Management of Faecal Stomas

A wide range of products exists for the practical management of intestinal stomas.

Pouches

Ostomy pouches are plastic, odour proof and disposable. They can be opaque or non-opaque, closed or drainable, one or two piece. The adhesive materials are variable - with the majority being hypo-allergenic. Some pouches have pre-cut apertures; others can be cut to the appropriate size. Integral skin-protective wafers of washers are available in the majority of pouches, especially in the drainable ranges, to ensure peristomal protection. Closed pouches are ideal for ostomates with a well-formed stool and are removed once or twice daily.

Drainable pouches are left in place for as long as they are leakproof and comfortable, usually three to five days, and the stool is drained from the pouch using a releasing clip. Two-piece equipment enables the adhesive base to be left in place and either a closed or drainable pouch affixed as required. This system is ideal for patients with very sensitive skin or for those receiving radiotherapy, as the base can be cut back and the pouch angled away from the treatment area.

Postoperative Pouching

It is advisable to use a drainable system with peristomal skin protection until the stool pattern is firmly established. A closed system is preferable for patients with well-formed effluent. Two-piece equipment ensures easy access to the stoma.

Flatus Patches

Many of the closed-type colostomy pouches have a built-in charcoal flatus patch allowing flatus to be spontaneously filtered and deodorized. Patches can also be affixed to pouches but should not be placed on drainable pouches as they only remain odourproof for approximately twenty-four hours.

Skin-Protective Wafers

These are made from pectin and carboxymethyl cellulose. Their function is to protect skin from the adhesive of pouches and to provide a firm base as additional security. Allergenic reactions to adhesives or excoriation from faecal contamination is managed by placing a skin-protective wafer over the peristomal skin.

Stoma Pastes

Pastes are used as fillers when irregular skin surfaces compromise continence. A washer of paste around leaking stomas in conjunction with appropriate skin protection will often solve problems. Spirit in the paste compounds may cause a temporary stinging sensation when applied to raw skin.

Stoma Powders

These are ideal for insufflating onto traumatized or bleeding stomal mucosa. The powder forms a protective gel until healing takes place. It may also be lightly dusted onto broken skin.

Convex Inserts and Convex Faceplates

These are used to promote a "pouting" of flush stomas - especially when ileal diversions have not been adequately fashioned as a spout. "Sliding" proximal loops in defunctioning loop ileostomies are especially difficult to manage as skin excoriations and leakage occur unless the active loop is encouraged to protrude into the pouch. Similarly, ileal loop urinary diversions require an adequate spout to avoid leakage of urine.

Belts

The majority of drainable and urinary pouches provide an integral belting system for use in extreme activity or to provide added security especially in difficult or high output ostomates.

Stomal Complications

The first problem is dermatological and the most common causes of sore skin are:

Faecal Contamination

This complaint is a result of effluent especially from the ileum and right-sided colostomies. The usual reason is application of the wrong diameter pouch - allowing faeces to be in contact with peristomal skin for a prolonged period, or leaking equipment due either to poor application, poor siting or surgical construction.

Retracted stomas with resulting skin creases require intensive stoma care or on occasions reconstruction or resiting.

Allergy to the Adhesive of the Pouch or Adhesive Tape

An obvious distribution of the erythematous or excoriated skin will give the clue to this distressing condition. Immediate removal of the offending adhesive and application of a protective skin wafer with a one or two-piece pouching system usually rapidly clears the problem. The offending adhesive may be found in several of the available pouches, in highly sensitive cases two-piece equipment with no other adhesive than that of a methyl cellulose wafer will solve the problem.

Fungal and/or Bacterial Infections

High-output stomas with leakage onto the skin can present a major management problem. Should fungal overgrowth occur, dermatological opinion should be sought and the use of topical anti-fungal and anti-inflammatory agents are sometimes prescribed. The

problem of adherence of equipment is maximal when any topical ointment is required. The use of a belt will give added security.

Mechanical Trauma

Patients who usually have formed stools and weal closed pouches remove their pouch once or twice daily. More frequent removal of any type of pouch will result in a damaged epidermis. Drainable pouches of a one or two-piece kind should be recommended to all patients with frequent stooling habits.

Surgical Complications

Paracolostomy Hernia

This relatively common complication rarely requires surgical intervention unless intermittent obstructive symptoms are manifested. Supportive belts, individually prescribed, should be put on in the morning while in the recumbent position with the hernia reduced. The pouch is outside a precut hole in the belt to allow the effluent to pass easily.

Prolapse

This is most commonly seen in loop colostomies of the transverse colon. Apart from being distressing to the patient, they are best conservatively managed. The most important consideration is to ensure that the pouch diameter is regularly monitored and increased to cope with the changing bulk of the stoma. Should a purse-string be considered, the patient must be regularly seen to ensure that the prolapse does not come through the suture and become irreducible. A patient who is severely distressed by the complication and who has to keep the stoma, may be offered an alternative division of the stoma, or conversion to a Hartmann procedure.

Retraction

Inadequate mobilization of the bowel at the time of surgery, especially in obese patients, leads to a major problem for the ostomate. Faecal contamination, especially on the inferior border of the peristomal skin, leads to excoriation and lichenification of chronically burnt skin. Incontinence is common and complex stoma care includes stoma pastes, convex face plates, belts and protective wafers. In suitable patients irrigation may be a solution - but many patients are reluctant or unable to resort to this. If deemed surgically practical, revision and adequate mobilization is the most satisfactory resolution of the problem.

Peristomal or Stomal Granulation

Topical mucosal granulations can become troublesome and bleed readily if rubbed - the use of electro-cautery to the lesions is the most effective treatment. The stoma should be coated liberally with stoma powder after the treatment and healing will take place once the lesions have dropped off. Stitch granulomas can be topically treated with a silver

nitrate stick and an application of Orabase placed around the stoma to prevent faecal contamination.

Stenosis

- Cutaneal stenosis: If a finger cannot be introduced cutaneally, local revision is required.

- Fascial stenosis: Troublesome intermittent winds and difficulty with the passage of faeces should always be investigated by a digital examination. If a finger cannot be advanced through the deep fascia, sometimes gentle dilation with Hegar dilators over a period of time will relieve the problem. Failing this, surgical intervention is necessary.

Bolus Obstruction

The possibility of bolus obstruction in ileostomy patients or ostomates with a fascial narrowing is high. A good history and gentle digital examination will reveal the bolus proximal to the fascial opening. It is advisable to stop oral intake and gently insert a soft rubber catheter and instil small amounts of warm water in an attempt to break up the bolus. This is the *only* time instillation of fluid is recommended in ileostomy patients. Common bolus offenders include bran, nuts and some fibrous fruits and vegetables.

Stomal Separation - Peristomal Sepsis

In cases of total separation immediate surgical intervention is required with refashioning of the stoma. Partially separated stomas with adequate protection from intra-peritoneal soiling should be managed with two-piece equipment to facilitate regular observation and care to the peristomal area. Topical stoma powder and paste may be placed into the defect and regular toilet performed until granulation has taken place. For peristomal sepsis, a similar technique is used with appropriate agents.

Ischaemia

Every surgeon and stomaltherapy nurse should carefully examine the postoperative stoma during the first 48 hours to ensure viability. Post-operative pouching should be non-opaque and if the stoma is in any way dubious it should be placed in a two-piece system to allow regular close observation. To assess the degree of ischaemia, a glass tube can be passed through the stomal or and a torch shone down the tube. However, a paediatric proctoscope with a light source is more efficient. Sloughing of the compromised mucosa takes place during the postoperative period and some stomas are left flush to the skin with a possibility of stomal stenosis if not dilated.

Colostomy Irrigation

A sigmoid colostomy is managed by natural evacuation or by irrigation. This latter technique is reserved for patients who have suitable home circumstances, are dextrous and motivated and have enjoyed regular bowel habits prior to surgery. Training by a stomaltherapy nurse should commence approximately six weeks after stomal construction,

after the perineal wound has healed. Irrigation is performed daily at a regular time for four weeks, and thereafter every second day.

Equipment

--> A water bag with attached connecting tube with a regulating clamp and a cone for insertion into the colostomy.

--> A face plate with an open-ended sleeve and a belt for attaching it to the abdomen.

Method

The water bag is filled with 1000-1500 mL of warm tap water. The bag is elevated to shoulder height and the water run through the tubing to eliminate any airlock. The patient may sit or stand. The pouch is affixed to the face place and placed over the stoma and held to the body by the firmly applied belt. The long open-ended pouch is placed into the toilet bowl and the patient inserts the lubricated cone into the stoma in the direction of the lumen. The water is slowly run in for five to seven minutes or until distension and discomfort dictate. On removing the cone, the upper open end of the bag is closed with clips and the faeces and water run through the bag into the toilet. The return can take anything from twenty minutes to an hour. Once completed, a small security pouch is placed over the stoma.

Potential Problems

--> Difficulties with the flow of water may be due to:

- an airlock caused by insufficient elevation of the water bag or incorrect positioning of the cone

- colostomy stenosis or paracolostomy hernia

--> Difficulties with faecal return:

- colonic obstruction

- paracolostomy hernia

--> Evacuation between irrigations:

- insufficient volume of water introduced

- not more than 2000 recommended

- dietary indiscretions, ie, increased alcohol intake.

Preparation of the Colon for Investigation or Colostomy Closure

Oral Preparation

All colostomy patients receiving oral bowel preparation should wear a skin-protective wafer and a long drainable pouch prior to receiving the appropriate aperient.

Distal Loop Preparation

While using the colostomy irrigation technique, let the patient sit on the toilet and instill warm tap water via the colostomy irrigation cone into the distal loop; encourage the patient to pass any retained faecal material from the rectum. In obstructed patients who require a second-stage resection, some water may return up the distal loop and pass into the toilet via the open-ended sleeve. Patients who have had barium studies require colostomy irrigation immediately following the procedure to avoid later barium impaction. Should this have occurred, olive oil is instilled twenty four hours prior to the washout.

Diet and Stomas

All ostomates are encouraged to eat their normal diet, although foods inducing excessive flatus or intestinal hurry are identified. Constipation may be avoided by adequate ingestion of high fibre and fluids. Persistent constipation or diarrhoea may have an organic cause and obviously require investigation.

Management of Intestinal Urinary Diversions

Urinary stomas may be fashioned from ileum, jejunum or colon. In each case, it is essential that the stoma protrudes from the abdomen as a spout and not flush with the abdominal wall.

Preparation of the patient includes:

--> full orientation
--> counselling
--> siting
--> adequate bowel preparation as an enteric anastomosis is necessary.

Special Siting Considerations

The conventional site for a urinary stoma is in the right lower quadrant, as for ileostomy. However, note must be taken of possible problems associated with previous radiotherapy fibrosis, compromised ureteric length and non-function of a kidney. Patients with neurogenic bladders may have orthopaedic deformities or be wheelchair-bound. It is essential to site such patients in the appropriate orthopaedic support belt or brace so that continence will not be compromised.

Postoperative Pouching

A two-piece urostomy pouch and base cut to fit snugly to the stoma should be attached to a closed-drainage system to hang by the side of the bed. The ability to remove the pouch from the base at will allows for observation of the spout, removal of excessive mucus and flushing of the ureteric splints if required.

Ureteric Stenting

The ileo-ureteric anastomosis is splinted at surgery using two feeding tubes of approximately 10F-12F. It is advisable to cut each protruding end at a different angle in order to identify right and left-sided drainage, ie, one cut oblique and one straight across. Should either become blocked, gentle flushing with a syringe of sterile water will remove mucus or blood clot. The stents remain in situ for seven to ten days depending on the surgeon's discretion. In the event of a ureteric leak developing, urine will be noted to drain from the left-sided abdominal drainage site. Insertion of a Foley catheter into the stoma with inflation of the balloon and gentle suction with a Drager suction unit should be established until the drainage ceases.

Monitoring of Urinary Stomas

Regular *calibration* of urinary diversions is recommended both as a prophylaxis and means of early detection of stenosis. This may be accomplished digitally or by means of a Hegar dilator.

Collection of Urine Samples for Bacteriology

Samples should be taken directly from the urinary conduit and never from the pouch. Clean the stoma with an antiseptic solution and using a sterile technique. Insert a urinary catheter into the conduit and collect the sample into a sterile container.