CHAPTER 5

The Pancreas

SCOPE

The pancreas, a gland in the abdomen that regulates glucose (blood sugar) metabolism and digestion, is vulnerable to a variety of injuries. The organ's location allows it to be compressed against the spine during severe abdominal trauma. Pancreatic injury caused by bicycle handlebars or child abuse is a highly lethal form of blunt trauma in children. The pancreas is also subject to penetrating trauma such as gunshot or stab wounds. Most pancreatic injuries are treated by surgical repair, which strives to preserve as much of the gland as possible. Moroever, the stress of an injury and its surgical treatment can cause pancreatitis (inflammation of the pancreas), even if the organ has not been traumatized. Other causes of pancreatitis include blockage of the gland's drainage by obstruction of the pancreatic duct, and the toxic effect of alcohol consumption. The diagnoses of pancreatic injury and acute or chronic pancreatitis are difficult to make, but improved visualizing techniques and new diagnostic laboratory tests are refining diagnoses and treatment.

SYNOPSIS

§ 5.00 Introduction

The pancreas is a multifunctional gland of the digestive system: its secretions include enzymes that digest proteins, carbohydrates and fats, and its hormones regulate the metabolism of glucose (blood sugar). Although the pancreas is well protected deep within the abdominal cavity, the gland can be injured in cases of penetrating trauma or of severe blunt injuries. A consequence of injury is pancreatitis, which may be acute or chronic. A variety of

diagnostic modalities, including blood tests, x-rays and scanning techniques can be used to evaluate pancreatic disease. Surgical treatment is necessary for a severely damaged gland, but care must be taken to preserve all the tissue that can be salvaged. Nonsurgical treatment has its place in treating pancreatic disorders, but surgery generally gives the best results.

§ 5.10 Anatomy

The pancreas, a pinkish, glandular organ has a retroperitoneal (referring to the zone behind, and outside of, the membrane that lines the abdominal cavity) location in the abdomen, behind and just below the stomach. (*See Figure 5–1.*) In an adult male, the organ typically weighs 100 grams (g) and measures 14 to 18 centimeters (cm) long, 2.0 to 9.0 cm wide, and 2.0 to 3.0 cm thick. The pancreas varies in size and weight, at different stages of life and under different conditions of a patient's health. An adult female has a pancreas weighing approximately 85 g; in a newborn, the weight of the gland is 5.0 g.

§ 5.11 Location and Traumatic Injury

The pancreas lies horizontally at the level of the second lumbar (referring to the low back) vertebra (one of the segments of the spinal column), and extends from the duodenum (the first part of the small intestine) on the right side of the abdomen, to the spleen on the far left. (*See Figure 5–2.*).

Pancreatic trauma or pancreatitis (inflammation of the pancreas) may result in the organ's enlargement or atrophy (wasting away). Because of its position in the abdomen, the pancreas can be compressed against the spine and injured during blunt or penetrating trauma.[1] Young children are particularly susceptible to pancreatic injury because they have not yet developed sufficient musculature to anticipate blows to the abdomen. Even a seemingly trivial blow to the abdomen can push a child's pancreas against the hard vertebral column and cause life-threatening pancreatic damage.

[1] *See also* ch. 1A and ch. 1B.

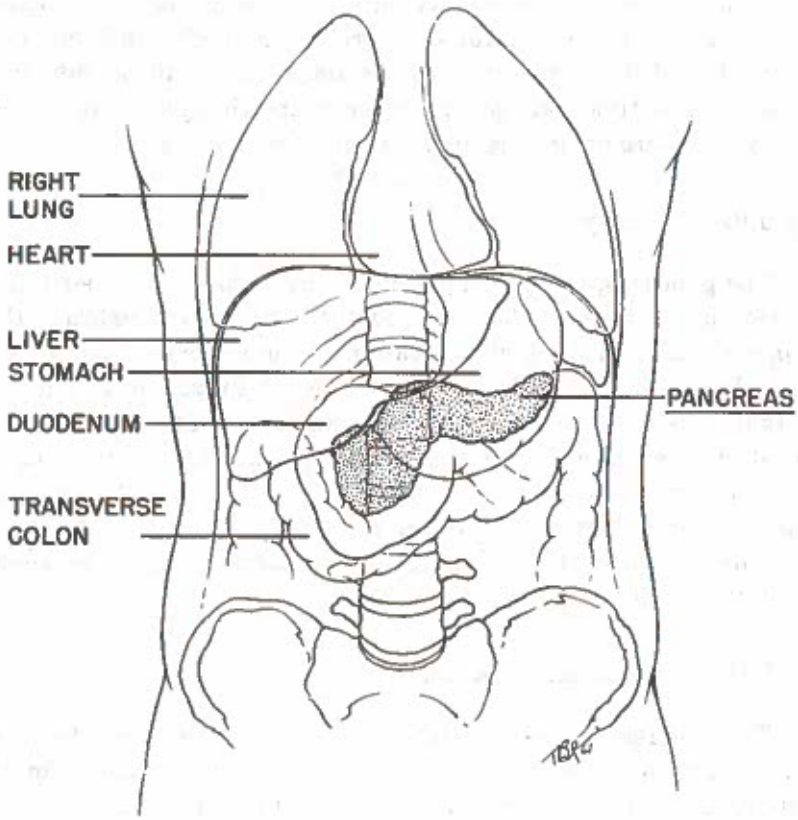

Fig. 5–1. An anterior view of the abdominal region, showing the position of the pancreas relative to the locations of other major internal organs.

§ 5.12 Divisions of the Pancreas

The pancreas can be divided superficially into four regions: head, neck, body and tail. The head, where the greatest mass of the organ is concentrated, fits snugly into the C-shaped curve of the duodenum. The neck of the pancreas thins slightly from front-to-back before continuing into the body and tail, which terminates bluntly at the spleen.

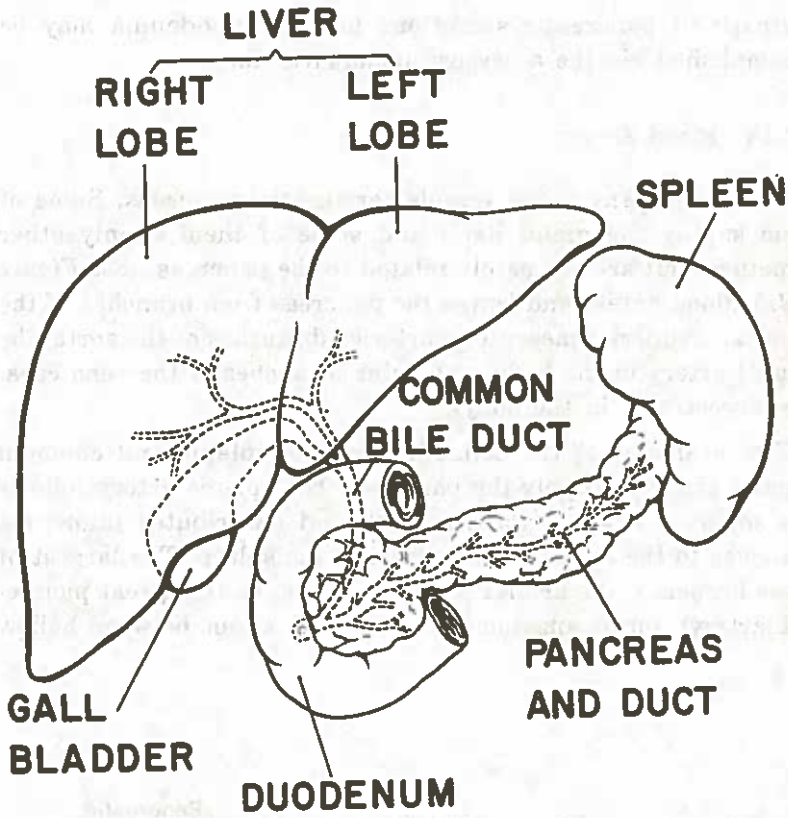

Fig. 5–2. The anatomical relationships of the liver, pancreas, gallbladder and duodenum. The main portions of the biliary drainage system are shown as dashed lines.

§ 5.13 The Pancreatic Duct System

The pancreatic duct (the drainage channel for pancreatic secretions), runs from the tail to the head, where it empties into the common bile duct (the channel that drains bile from the liver and gallbladder). Through the common bile duct, bile and pancreatic juice are secreted into the duodenum. (*See Figure 5–3.*) This association becomes particularly significant when gallstones form in the gallbladder, and eventually pass into the common bile duct, which becomes blocked. The accumulation of bile backs up to the pancreas, which becomes inflamed. These gallstones also can obstruct the pancreatic duct and cause pancreatitis. Additional

drainage of pancreatic secretions into the duodenum may be accomplished via the accessory pancreatic duct.

§ 5.14 Blood Supply

There are many blood vessels serving the pancreas. Some of them supply the gland itself and some of them supply other structures but are intimately related to the pancreas. (*See Figure 5–4.*) Blood enters and leaves the pancreas from branches of the celiac and superior mesenteric arteries (branches of the aorta, the largest artery in the body) and veins (branches of the vena cava, the largest vein in the body).

Two branches of the celiac artery, the splenic and common hepatic arteries, supply the pancreas. The splenic artery follows the superior edge of the pancreas and contributes numerous branches to the organ before reaching the spleen. The largest of these branches, the arteria pancreatica magna (the great pancreatic artery), forms anastomoses (communications between hollow

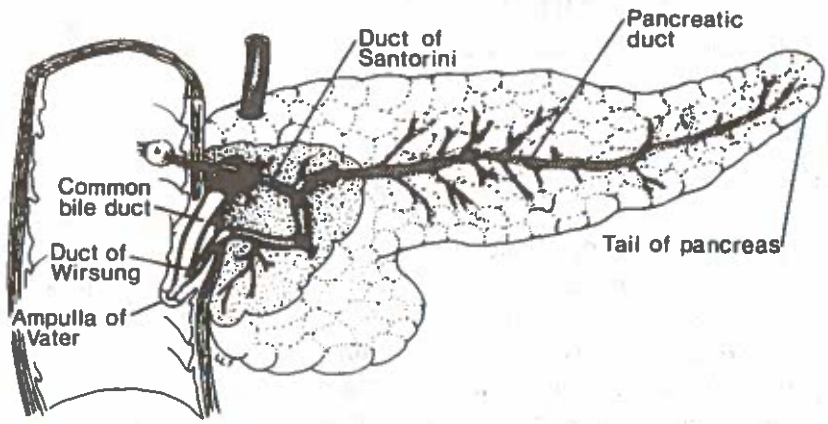

Fig. 5-3. The duct system of the pancreas.

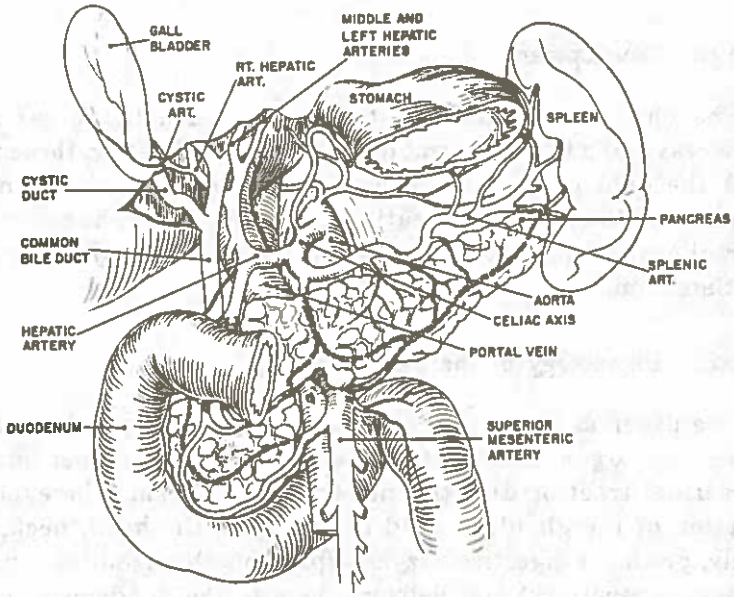

Fig. 5–4. The blood supply of the liver and the pancreas. The splenic artery follows the superior edge of the pancreas and contributes numerous branches to the organ before reaching the spleen.

organs, including blood vessels) with other branches of the splenic artery and contributes to the marked vascularity of the pancreas. The common hepatic artery gives off the gastroduodenal artery, which branches to supply the head of the pancreas and adjacent duodenum with blood. The superior mesenteric artery contributes branches (including the transverse pancreatic artery) to the head and tail of the pancreas.

The intimate relationship between the blood supply of the pancreas and that of the duodenum and spleen becomes particularly significant if the arterial circulation to these organs is compromised during abdominal surgery. If the blood flow to the duodenum or spleen is cut off, the pancreas also suffers. Likewise,

blocking or severing the arteries to the pancreas will affect the duodenum and spleen.

§ 5.15 Development

The embryonic gland has two parts, a small, dorsal (top) pancreas and a large, ventral (bottom) one. Ordinarily, these fuse, and the duct of the ventral component becomes the terminal portion of the main pancreatic duct. Frequently, however, the dorsal channel persists as the accessory pancreatic duct, or duct of Santorini.

§ 5.20 Physiology of the Pancreas

The pancreas has both exocrine and endocrine glandular functions. The organ delivers its secretions either via a duct into the intestinal tract or directly into the blood steam. The exocrine portion of the gland, located primarily in the head, neck, and body, produces digestive enzymes (proteins that facilitate specific chemical reactions) and delivers them to the duodenum via the pancreatic duct. The endocrine portion of the gland, located primarily in the tail region, produces hormones (derivatives of either proteins or fats, each affecting a specific target organ or tissue) and delivers them to the body via the bloodstream. Despite its wide range of functions, the pancreas is not indispensable to life. Parts of the pancreas and even the entire gland can be removed, though the latter procedure is performed only when the benefits of removal far outweigh the effects of losing pancreatic exocrine and endocrine functions.[2]

§ 5.21 Exocrine Functions

The pancreatic digestive enzymes produced by the acinar (referring to a tubular structure consisting of a single layer of secretory cells) cells in the pancreas are important in fat, carbohydrate, and protein digestion. The enzymes are also important in neutralizing the acidic digestive contents from the stomach so that the intestinal enzymes can perform under optimal conditions.

[2] *See* § 5.54 and § 5.83(1)(d) *infra*.

The most important pancreatic enzymes are protease (protein splitting enzyme), amylase (starch splitting enzyme), and lipase (fat splitting enzyme).

(1) Pancreatic Protease

Trypsin and chymotrypsin (pancreatic proteolytic enzymes) are produced as the inactive precursors trypsinogen and chymotrypsinogen. When they reach the duodenum, they are activated by enterokinase (an intestinal enzyme). This enables them to hydrolyze (to chemically cleave a molecule, with the uptake of water, as H and OH, at the site of the split) proteins into their simpler components (short chains of amino acids).

(2) Pancreatic Amylase

Amylase is responsible for breaking down complex starch molecules into simple sugars (sugars such as glucose, which contains six carbon atoms in a ring or a chain). Pancreatic amylase is responsible for digesting 60 percent of the carbohydrate ingested as starch. The remaining fraction is hydrolyzed by salivary amylase. Amylase can be an important indicator of pancreatic damage. Elevated levels of amylase in the blood, urine, or other fluids may indicate pancreatic disease or trauma.

(3) Pancreatic Lipase

Pancreatic lipase is the most important enzyme for fat digestion. If the pancreas is removed or malfunctioning, some proteins and carbohydrates can be digested, but most ingested fat will be eliminated in the feces. This condition is called fat malabsorption, and is marked by the occurrence of greasy, foul-smelling diarrhea.[3]

§ 5.22 Endocrine Functions

The endocrine function of the pancreas is performed by discrete clusters of cells called the islets of Langerhans. The islet cells

[3] *See* § 5.82 *infra.*

produce the hormones insulin, glucagon and gastrin. (*See Figure 5–5.*)

(1) Insulin

Insulin is produced by the g (beta)-cells of the islets of Langerhans, and is essential for the metabolism of carbohydrates (sugars and starches); the hormone facilitates the absorption of glucose by the brain, liver and muscles. This reaction is enhanced in the presence of potassium (the absorption of which is facilitated in the presence of glucose). Insulin lowers blood levels of glucose immediately after a meal by increasing the amount of glucose collected and stored in the liver. A deficiency of insulin causes diabetes mellitus.[4]

(2) Glucagon

Glucagon, which is produced by the Langerhans g (gamma)-cells, rapidly breaks down the starch glycogen into its glucose subunits, and thus, in contrast to insulin, increases the blood glucose level. Abnormally high levels of blood sugar (hyperglycemia) in a nondiabetic patient may indicate pancreatic disease or injury.

(3) Gastrin

Gastrin is produced in the g (delta)-cells of the islets of Langerhans, but most of this hormone comes from the G cells of the gastric (referring to the stomach) antrum (the last part of the stomach) and the proximal (the near part of; the upper end) part of the duodenum. Gastrin circulates in the blood and stimulates the glands of the stomach to produce gastric juice (an enzymatic fluid important in protein and, to a lesser extent, fat and carbohydrate digestion).

§ 5.30 Types of Injury Sustained by the Pancreas

The pancreas is subject to several types of injuries.[5] It can be pierced by a knife or bullet, or it can be compressed against the

[4] *See also* Attorney's Textbook of Medicine, ch. 74 (Matthew Bender, 1985).

[5] *See also* Attorney's Textbook of Medicine, ch. 228A (Matthew Bender, 1985).

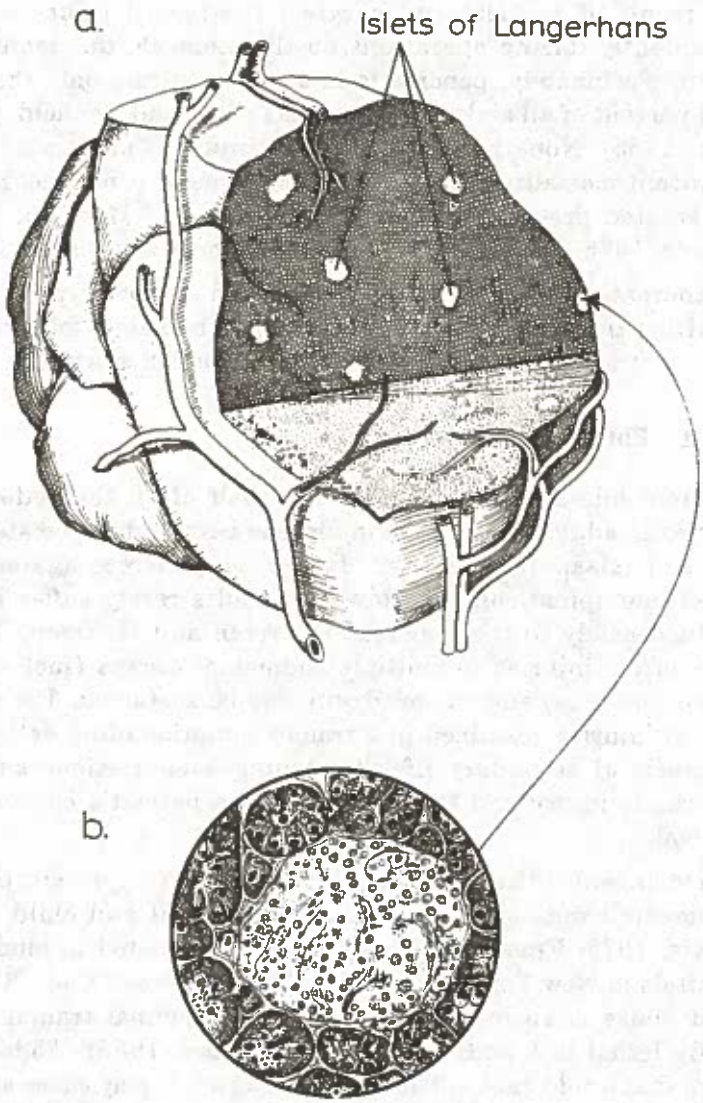

Fig. 5-5. Representations of the types of tissue in the pancreas: above, the pancreas in cross section, showing the distribution of the islets of Langerhans. Below, an islet appears as it would under a microscope, as a sharply circumscribed zone of pale cells.

spine by a steering wheel in an automobile accident. Iatrogenic (the result of medical and surgical treatment) injury occurs infrequently during operations on the stomach, duodenum, or spleen. Fortunately, pancreatic injuries constitute only three to eight percent of all abdominal injuries (White and Benfield, 1972; Sako, 1955). Nonetheless, pancreatic injury is associated with significant mortality (16 percent to 20 percent) if not recognized and treated promptly (Jones, 1978; Babb and Harmon, 1976; Heitsch, 1976; Bach and Frey, 1971; and Jones and Shires, 1971).

Pancreatic injury usually is classified as two types: blunt (resulting from an automobile accident or abdominal fall or blow) and penetrating (resulting from knife or bullet wounds).[6]

§ 5.31 Blunt Trauma

Automobile accidents cause nearly half of all the abdominal injuries in adults. Pancreatic injury can result when the steering wheel or misapplied seat belt forces the pancreas against the unyielding spinal column. However, adults rarely suffer injury confined solely to the pancreas (Warrren and Hoffman, 1976). More often, injuries to multiple abdominal organs (such as the spleen and liver) and the head will also be sustained. The multitude of injuries sustained in a trauma situation often delays the diagnosis of secondary life-threatening complications such as pancreatic injury and thus decreases the patient's chances for survival.

In younger children, blunt abdominal trauma is typically caused by bicycle handlebars, playground equipment and child abuse (Slovis, 1975; Fraser, 1969). One study conducted in municipal hospitals in New York City and Philadelphia found that although child abuse is an infrequent cause of abdominal trauma, it is highly lethal in a pediatric patient (Cooper, 1988). Abdominal blows that would cause little trauma in an adult may cause serious injury in a child. Unlike adults, children seldom suffer multiple organ damage in pancreatic injury caused by blunt trauma (Dahman and Stephens, 1981; and Otherson, 1968). This fact can make diagnosing pediatric pancreatic injury even more difficult.

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[6] *See also* Attorney's Textbook of Medicine: Manual of Traumatic Injuries, ch. 1 (Matthew Bender, 1989).

§ 5.32 Penetrating Trauma

Penetrating trauma to the pancreas is isolated in very few cases. In both children and adults, other organs will most likely also be affected. If, however, injury is confined solely to the pancreas, a patient's chance of survival is greater than if the other organs are affected. The fewer organs that are damaged, the greater the patient's chance of survival. The type of missile that causes the injury therefore has a direct impact on the patient's survival: shotgun bullets damage more organs than pistol bullets, which injure more organs than knives.

§ 5.40 Diagnosis of Pancreatic Injury

In an abdominal injury where there is multiple organ damage, pancreatic injury is considered a possibility — especially if the site of the trauma is located in the upper left quadrant of the abdomen or lower left chest.[7] Though diagnosing pancreatic injury is difficult because the symptoms vary greatly, combined findings from the physical examination, laboratory tests, peritoneal lavage (flushing the abdominal cavity with liquid), laparotomy (surgical opening of the abdomen), and diagnostic imaging techniques can be utilized to diagnose the pancreatic damage.

§ 5.41 Physical Examination

The approach to diagnosing pancreatic injury depends on the type of injury that is sustained (penetrating or blunt) and the cause of the injury (shotgun, pistol, knife).

(1) Penetrating Trauma

Penetrating wounds from gunshots are relatively easy to diagnose, as the path of the missile can be traced from entrance to exit, and requires only a minor physical examination before surgical correction. Patients with shotgun wounds almost always undergo a laparotomy (exploratory surgery of the abdominal cavity) so that the full extent of the damage can be evaluated and corrected.

[7] *See also* ch. 1A.

Patients who have suffered abdominal penetrating trauma and who show no alarming clinical signs will usually undergo a peritoneal lavage (flushing of the abdominal cavity with sterile salt water, which is instilled and removed via needle puncture);[8] the results of the lavage dictate whether or not a laparotomy needs to be performed (Toledo-Pereyra, 1985).

(2) Blunt Trauma

Pancreatic injury following blunt trauma is much more difficult to diagnose than damage due to penetrating wounds. Moreover, the physician who examines a child must confront the possibility of child abuse.

(a) Adult Patients

Adult patients who are not unconscious or intoxicated can be questioned about recent abdominal trauma which they may or may not recall. In a vast majority of cases, other viscera (abdominal organs, especially the hollow ones) also are injured and clinical symptoms of multiorgan damage will be very obvious (Warren and Hoffman, 1976). In rare cases of injury confined solely to the pancreas, there will be little or no abdominal pain or a physical exam will reveal only limited tenderness in the pancreatic region.

(b) Children

In children, diagnosing pancreatic injury following blunt trauma is even more difficult than in adults, because unusual circumstances may have caused the incident, and because children have less ability to communicate articulately than do adults. Especially troublesome in this regard is child abuse. One study of the extent of abdominal trauma due to child abuse found that more than half of the children whose cases were reviewed came from families where episodes of child abuse had been previously documented by authorities (Cooper, 1988).

Although one may have a high index of suspicion for a case of child abuse because of the physical evidence, the history available from the parents or patient may be inadequate or false. For example, parents may fail to give an accurate account of the events

[8] *See* § 5.43 *infra.*

causing the trauma for fear of recriminating action. Moreover, if the trauma was caused while the child was under someone else's supervision, parents may lack the knowledge of the events causing the trauma. In addition, children are even more afraid than their parents or guardians to give an accurate account of events.

As a result of fear or lack of knowledge, parents may fail to bring their child to the hospital until life-threatening signs are very obvious and, unfortunately, too late to correct. This delay in seeking treatment, combined with an inaccurate or incomplete patient history, contributes to the high mortality rate seen in pediatric patients suffering from abdominal trauma, and serves to complicate the already difficult task of diagnosing and treating pancreatic injury.

Diagnosing pancreatic trauma in a child, therefore, requires careful attention to detail. A child with a pancreatic injury will usually have pain in the upper left abdominal quadrant (the zone of the abdomen superior to the navel and to the left of the midline) which seems out of proportion to other physical findings. The child usually vomits, and has a fever and a distended abdomen. Unlike the pancreatic injuries occurring in adults, those in children are not associated with multiple organ injuries.

§ 5.42 Laboratory Tests

Laboratory tests are not always helpful in diagnosing early pancreatic injury. Serum amylase levels may or may not be elevated. Some patients have elevated serum amylase levels following blunt injury but never show any evidence of pancreatic injury (Olsen, 1973; Moretz, 1975). Other patients who sustain pancreatic injury will show no rise in serum amylase prior to operation.

§ 5.43 Peritoneal Lavage

Peritoneal lavage, the insertion of a hypodermic needle through the abdominal wall to infuse and withdraw fluids, is used to determine the presence of pancreatic injury in cases of blunt abdominal trauma. Approximately 2.0 liters of sterile saline solution are infused through the needle into the abdominal cavity.

The saline is withdrawn about 30 minutes later, and the fluid is inspected for blood and pancreatic enzymes that might indicate abdominal hemorrhage and pancreatitis.

§ 5.44 Diagnostic Imaging Techniques

Imaging of the pancreas can enhance the diagnosis of pancreatic injury but does not usually provide direct evidence of pancreatic injury.

(1) Plain X-Ray Films

Chest and abdominal x-rays will not yield distinct findings of pancreatic injury but can be helpful in revealing associated organ injury (Toledo-Pereyra, 1985). Abdominal x-rays which reveal tiny gas bubbles in the region between the right twelfth rib and upper lumbar vertebrae might indicate retroperitoneal rupture of the duodenum. Free air under the diaphragm indicates perforation of the digestive tract. Both of these findings suggest associated retroperitoneal hemorrhage and possible pancreatic damage.

(2) Ultrasonography

Ultrasonography is a noninvasive imaging technique that uses sound waves to visualize structures inside the body. Ultrasonography has limited value in investigating pancreatic trauma. While ultrasonography can visualize an inflamed or enlarged pancreas and can help diagnose some of the ensuing complications of pancreatic injury, such as a pancreatic pseudocyst, it is not helpful in the initial investigation of a traumatized pancreas that may not have yet changed its size or shape.

(3) Computed Tomography

Computed tomography (CT or CAT; an integration of multiple scanning images to form pictures of different levels within a patient's body) is a visualizing technique that achieves a better evaluation of the pancreas than ultrasonography but still only has limited value in the initial evaluation of pancreatic trauma. Like ultrasonography, CT may demonstrate changes in other organs

but should not be totally relied on to diagnose pancreatic injury (Cook, 1986; and Federle, 1981). In addition, CT scans are expensive and expose the patient to ionizing (having the capacity to produce electrically charged forms of atoms) radiation.

§ 5.50 Treatment of Pancreatic Injury

Surgical treatment for pancreatic trauma is nearly always indicated. The severity and location of pancreatic injury determines the exact surgical technique (Anane-Sefah, 1975; and Belasgarem, 1976). Based on these determinants, surgical treatment can be divided into five categories: minor wounds, main pancreatic duct wounds, pancreatic tail wounds, pancreatic head wounds, and combined duodenopancreatic (involving the duodenum and the pancreas) wounds.[9]

§ 5.51 Minor Wounds

During laparotomy, local wounds to the pancreas may be treated by suturing the edges of the lacerations. Care usually is taken to oversew the edges and not approximate them: it is better to have a small amount of pancreatic juice harmlessly leaking into the body cavity than for a a pseudocyst to form as a result of approximating the edges of the wound.[10]

§ 5.52 Main Pancreatic Duct Wounds

In cases where the pancreatic duct has been cut through (transected) either entirely or partially, a portion of the pancreas usually is removed or resected at the point where the pancreatic duct was injured. The cut end of the pancreas is then either sutured or stapled to prevent leakage. It is important to remember that in rare instances, the pancreatic duct may be cut through even though the surface of the pancreas appears intact.

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[9] *See also* Medical Malpractice: Guide to Medical Issues, ch. 34 (Matthew Bender, 1986).

[10] *See* § 5.62 *infra*.

§ 5.53 Pancreatic Tail Wounds

Injuries of the neck, body, and tail of the pancreas usually are treated by distal resection (removal of the neck, body, and/or tail). The cut end of the pancreas is then either sutured or stapled to prevent leakage.

§ 5.54 Pancreatic Head Wounds

Injuries to the head of the pancreas are potentially more serious than injuries to the body, neck, or tail of the organ. Because there are more enzyme-producing cells in the head of the pancreas, a larger volume of secretion can result if the head is injured. Additionally, trying to correct the damage by removing an extensive portion (80 to 90 percent) of the pancreas may result in endocrine or exocrine deficiency — especially in young people.[11] Extensive removal should only be performed in diabetic patients, in unstable patients, and in patients who have extensive injuries and cannot tolerate a long operation.

There are a few surgical techniques that preserve the body and tail of the pancreas when the head has been traumatized. One technique removes the injured portion of the head and connects the cut end of the pancreas directly to the jejunum (the middle portion of the small intestine) so the pancreatic fluid can leak into the intestinal tract. Another method also removes the injured portion of the head, but then indirectly channels pancreatic fluid into the duodenum through a Roux-en-Y loop. Some surgeons see the latter procedure as unnecessary and, with the additional suture line the procedure requires, may increase the risk of infection or contamination (Brooks, 1983). Other techniques only drain the injured pancreas.

§ 5.55 Combined Duodenopancreatic Wounds

An injury to the pancreas and duodenum can pose great problems. The major objective in treating duodenopancreatic wounds is to divert the contents of the stomach from the damaged area. This can be accomplished by opening up the stomach,

[11] *See* § 5.20 *supra.*

suturing closed the pylorus (the opening between the stomach and small intestine), and diverting the contents of the stomach past the injured area to the jejunum through a gastrojejunostomy (surgical opening of the stomach and the jejunum). The pylorus will open spontaneously in a few weeks by which time the injured area will have healed.

§ 5.60 Complications of Pancreatic Injury

Complications of pancreatic injury include pancreatic fistulas, pseudocysts, ascites, and abscesses. Pseudocysts and pancreatic abscesses usually are associated with blunt trauma and may reflect an inadequate appreciation of pancreatic injury at the time of exploration. Some of these sequelae might occur after surgical repair of the organ.[12]

§ 5.61 Fistulas

Fistulas (pathological or iatrogenic communications between either two hollow viscera or an organ and the skin) can arise following a pancreatic operation where a pancreatic duct injury was overlooked or was inadequately treated during the procedure. Fistulas can also follow external drainage of a pancreatic pseudocyst when the pseudocyst fails to close properly. In rare instances, fistulas will develop following a needle biopsy (removal of tissue or fluid for the purpose of examination and diagnosis). This no longer seems to be a problem, however, since new techniques use very thin needles.

Fistulas usually close by themselves and require no operative intervention. Most fistulas close within six to twelve months, but some persist for over a year. If the output of pancreatic juice through the fistula is low, most patients can be managed at home through drainage, and by means of medical and nutritional therapy aimed at decreasing pancreatic secretion. The drainage usually consists of a catheter inserted in the fistula channel through which constant suction is maintained. Meticulous care of

[12] *See also* Medical Malpractice: Guide to Medical Issues, ch. 34 (Matthew Bender, 1986).

the skin surrounding the catheter is also vital since the pancreatic enzymes are strong enough to break down the skin.

In cases where unusually high fistula output causes severe dehydration and malnutrition, surgical intervention may become necessary. Fistulas in the tail of the pancreas may simply be resected. Fistulas in the head may be cut free to create an opening in the pancreas which is then connected either directly or indirectly to the jejunum.

§ 5.62 Pseudocysts

A pseudocyst (a space within body tissues that usually contains fluid, coagulated blood and cellular debris) is a classic complication of pancreatic injury, especially any form of trauma. Pseudocysts typically occur in the lesser omentum (a potential space between the layers of the peritoneum, located between and somewhat above the stomach and pancreas). (*See Figure 5–6.*)

(1) Pathophysiology

The initial lesion in pseudocyst formation is the digestion of peripancreatic tissues by enzymes released from an injured or diseased gland. This is accompanied by hemorrhage, with the accumulation of cellular debris and clotted blood. The protein in the blood clot has a strong affinity for water, which is drawn into the space from surrounding tissues. As a result, the cyst becomes progressively larger. Meanwhile, episodes of hemorrhage contribute additional clotted blood and cellular debris.

Pseudocysts differ from true cysts in that they are not surrounded by an epithelial (referring to the tissue that normally covers body surfaces and lines hollow organs) lining, but by fibrous tissue. (*See Figure 5–7.*)

(2) Symptoms and Diagnosis

The most common symptoms of pancreatic pseudocysts are pain, nausea and vomiting, weight loss, abdominal tenderness and a palpable mass in the upper left quadrant of the abdomen. Diagnosing the presence of pseudocysts is often difficult, since

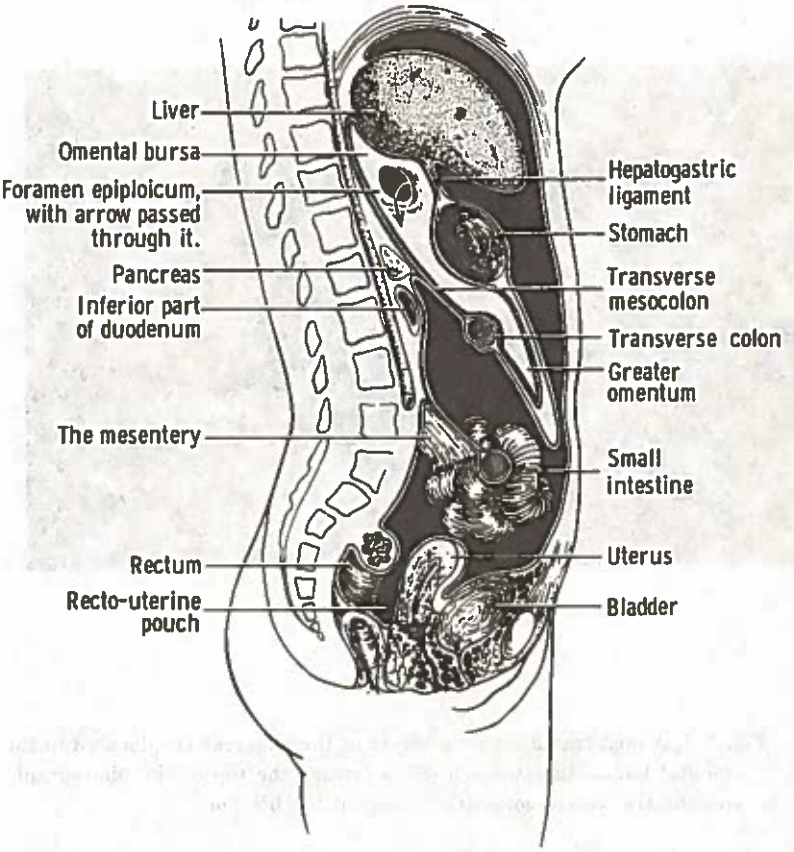

Fig. 5–6. A sagittal (side) view of the abdominal contents: pseudocysts form in the lesser omentum, a pouch like extension of the main peritoneal (intra-abdominal) cavity, the opening of which is the foramen epiploicum (epiploic foramen).

the symptoms are similar to the underlying disease. Ultrasonography and endoscopic retrograde pancreatography (ERP; radiographic visualization of the pancreas after a dye has been injected

Fig. 5–7. A post-traumatic pseudocyst of the pancreas (P), located in the omental bursa; the stomach (S) is toward the top of the photograph, and the transverse colon (C) is toward the bottom.

into the pancreatic duct via an endoscope) are the most reliable procedures for diagnosing pseudocysts.

(3) Treatment

Treatment of a pseudocyst is important, since one that ruptures or leaks may lead to pancreatic ascites (an accumulation of fluid within the abdominal cavity). Therefore, close monitoring of an early pseudocyst by physical exam, laboratory evaluation, and ultrasound are very important. If the pseudocyst appears to be enlarging (as indicated by the the symptoms' worsening), intervention becomes necessary. In patients who cannot tolerate surgery, external drainage and gradual repair are indicated.

§ 5.63 Ascites

Pancreatic ascites (the abnormal accumulation of pancreatic fluid within the body cavity) is most frequently a complication of chronic pancreatitis, but also can occur following pancreatic trauma. Ascites usually results from a leaking pseudocyst or disrupted pancreatic duct. Since the leak can dissect (spread by moving along the interfaces between layers of tissue) some distance from its source, a fluid collection may appear in the pleural cavity (the potential space between the lung and the chest wall), groin, or mediastinum (the zone in the middle of the chest).

Patients may have symptoms of increasing abdominal girth, weight loss, and mild abdominal pain. Paracentesis (introduction of a hypodermic needle into the body cavity and withdrawal of fluid) can be used in diagnosing pancreatic ascites. If ascites is present, fluids from paracentesis will have amylase levels above those in the blood and will also have a high concentration of total protein.

§ 5.64 Abscesses

Pancreatic abscesses are localizations of pus within or on the pancreas that usually are lethal unless drained quickly. These abcesses are most frequently associated with severe or postoperative pancreatitis and in patients who have undergone a laparotomy. After a laparotomy, a patient will either improve until the 14th to 21st day and then deteriorate rapidly, or the patient will fail to improve at all, despite continued treatment. The most typical clinical and laboratory features of a patient diagnosed as having pancreatic abscesses include fever, abdominal distention, leukocytosis (elevated white blood cell count), and a palpable abdominal mass.

The most important aspect of managing pancreatic abscesses is surgical drainage. Also, monitoring the respiratory status and electrolyte balance and preventing gastroduodenal bleeding are important.

§ 5.70 Acute Pancreatitis

Pancreatitis (inflammation of the pancreas) is a disorder in which, very simply put, the pancreas is digested by its own enzymes. Pancreatitis is classified as either acute or chronic. While it is often difficult to separate and classify the acute and chronic forms of pancreatitis, acute pancreatitis usually refers to an acute condition presenting with abdominal pains and raised levels of certain enzymes in the blood or urine. In acute pancreatitis, the pancreas returns to normal once these conditions are resolved and suffers no permanent structural or functional damage. Chronic pancreatitis, on the other hand, involves permanent structural or functional damage to the pancreas.

§ 5.71 Etiology

While the exact physiologic mechanisms leading to pancreatitis are still being studied, certain processes are commonly associated with the acute disease. Biliary tract disease and alcoholism account for the majority of patients who are diagnosed as having acute pancreatitis. In addition, other disorders including abdominal trauma can result in acute pancreatitis.

(1) Biliary Tract Disease

Biliary tract disease (any disorder associated with the gallbladder and bile ducts) is most commonly associated with acute pancreatitis (White 1966; Kelly, 1976; Dixon, 1970; Acosta and Ledesma, 1974). In particular, cholelithiasis (gallstones in the gallbladder or bile ducts) is seen frequently in patients suffering from acute pancreatitis. Some studies found that more than 50 percent of non-alcoholic patients diagnosed with acute pancreatitis had gallstones (Kelly, 1980 and Imrie, 1980). Calculi (abnormal concretion of materials found in ducts, hollow organs, and cysts) appear to lodge in the common duct and obstruct the flow of fluids from the biliary and/or pancreatic duct. (*See Figure 5–8.*)

(2) Alcohol

Binge drinking of alcohol can sometimes precipitate an attack of pancreatitis hours after consumption. However, it is often not

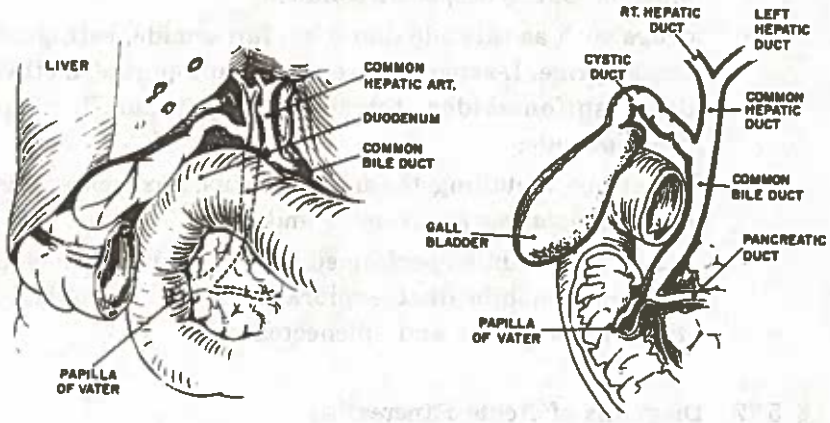

Fig. 5–8. A detailed representation of the major branches of the biliary tree.

clear whether these attacks are of the acute or chronic variety. Although the importance of alcohol is greatest in chronic pancreatitis where excessive alcohol consumption over a prolonged period of time has caused permanent damage to the pancreas, the first attack of inflammation in an otherwise asymptomatic patient usually is classified as acute. This is because the status of the gland prior to the attack is rarely known. Thus, unless evidence of chronic injury can be documented, the first attack of pancreatitis is conventionally classified as acute.

(3) Other Disorders

Other disorders have been associated with a small percentage of cases of acute pancreatitis. These include the following:

- blunt or penetrating abdominal trauma;
- tumors originating in the duodenum, bile duct, or head of the pancreas;
- metabolic disorders such as hypercalcemia, hyperlipidemia and hyperparathyroidism;
- drugs such as thiazide diuretics, furosemide, estrogens, azathioprine, L-asparaginase, 6-mercaptopurine, methyldopa, sulfonamides, tetracycline, pentamidine and procainamide;
- infections including the mumps virus, coxsackievirus, and *Mycoplasma pneumoniae* and
- surgical procedures performed near the pancreas including common bile duct exploration, sphincteroplasty, distal gastrectomy and splenectomy.

§ 5.72 Diagnosis of Acute Pancreatitis

Diagnosis of acute pancreatitis is often difficult because signs and symptoms are so variable. Also, the position of the pancreas deep within the abdomen makes physical examination difficult and radiologic findings imperfect. However, combined findings from the clinical presentation, physical exam, patient history, laboratory studies and diagnostic imaging techniques make diagnosis possible.

(1) Clinical Presentation

The primary symptoms of acute pancreatitis are abdominal pain, nausea and vomiting. Abdominal pain occurs in almost every case of acute pancreatitis (Olsen, 1974). Located primarily in the epigastric (the upper-middle abdomen) region, the pain often radiates straight through to the back. Five characteristic features of pain usually are evident in acute pancreatitis:

- the onset of pain is sudden and reaches maximum intensity within a few minutes;
- the pain is often severe (10 on a scale from 1 to 10);
- the pain is very steady and fluctuates little in intensity;

- the pain persists for many hours and usually for several days; and
- patients are unable to find a position that provides relief.

These characteristics of pain can help differentiate acute pancreatitis from other acute abdominal conditions. For example, a gradual onset of pain suggests mesenteric infarction. Pain that fluctuates in intensity is more characteristic of partial small-bowel obstruction. Pain that responds to conventional dosages of narcotics and subsides within 24 hours may be caused by biliary colic.

Some degree of nausea is experienced by patients suffering from acute pancreatitis; and, if the condition is severe, vomiting will persist indefinitely and culminate in dry heaves. The vomit almost never contains fecal material as would occur in a small-bowel obstruction (Go, 1986). In addition, vomiting in acute pancreatitis does little to alleviate pain and may only be relieved by insertion of a nasogastric tube. Vomiting in peptic ulcer disease, on the other hand, ceases after perforation.

(2) Physical Exam

Other signs of acute pancreatitis that may be revealed upon physical examination include tachycardia (increased heart rate), lowgrade fever, mildly distended abdomen with marked tenderness, hypotension (abnormally low blood pressure) and mental aberrations. These symptoms can also indicate the prognosis of a patient. While tachycardia is normal in mild acute pancreatitis, a tachycardic patient who is restless, cold and clammy has a poor prognosis. A temperature greater than 101 ° Celsius (C) suggests a complicating infection. Hypothermia (abnormally low body temperature) is observed only in extremely ill patients and is a poor prognostic sign.

(3) Patient History

Obtaining a detailed history is very important in diagnosing acute pancreatitis. Questions regarding alcohol consumption, previous attacks of biliary colic, recent abdominal trauma, drug ingestion, and operative procedures associated with an increased risk of pancreatitis can help diagnose acute pancreatitis.

(4) Laboratory Studies

Laboratory procedures can aid in the diagnosis of acute pancreatitis; they are rarely used as the sole diagnostic tool. Routine blood studies, serum amylase, serum lipids, and serum lipase may all help diagnose acute pancreatitis.

(a) Routine Blood Studies

Hemoglobin (the oxygen-carrying portion of red blood cells) may be elevated early in the course of pancreatitis due to the loss of plasma (the fluid component of blood not containing blood cells) in the area of the inflammation. Plasma loss decreases the volume of the blood and thus causes the blood cells to become more concentrated. Such red blood cell concentration is not seen with other acute abdominal conditions and usually signifies an acute pancreatic condition.

Hyperglycemia. Hyperglycemia (an abnormally high level of blood glucose) is common in moderate to severe attacks of pancreatitis where there is no history of diabetes mellitus. In moderate cases of acute pancreatitis, the hyperglycemia will disappear within a few days. However, hyperglycemia will persist in severe acute pancreatitis where there is extensive damage to the pancreas.

Hypocalcemia. Hypocalcemia (an abnormally low level of blood calcium) usually is not evident before the diagnosis of acute pancreatitis is made. However, a blood calcium level that drops suddenly should suggest acute pancreatitis in a previously undiagnosed patient. A marked fall in blood calcium indicates severe pancreatic damage and usually is a poor prognostic sign.

(b) Serum Amylase

Hyperamylasia or an increased blood level of amylase in a patient suffering from abdominal pains is a reasonably good indicator of acute pancreatitis.

(c) Serum Lipids

In one study, high serum triglyceride levels were reported in 20 percent of all patients diagnosed as having acute pancreatitis (Warshaw and Lee, 1980). However, other reports found an incidence of only five percent (Greenberger, 1966). Further

studies will be required to show the exact role of serum lipids in diagnosing acute pancreatitis.

(d) Serum Lipase

High levels of serum lipase has sometimes been used as an indicator of acute pancreatitis over the years. However, there seems to be little advantage over serum amylase indicators. As techniques for accurately measuring serum lipase improve, the role of serum lipase in diagnosing acute pancreatitis may become more significant.

(5) Diagnostic Imaging Techniques

Abdominal and chest x-rays usually are enough to evaluate the condition of the pancreas and other abdominal structures. Ultrasonography and gastrointestinal examinations can aid in determining the cause of pancreatitis, but a CT scan rarely is necessary in cases of uncomplicated acute pancreatitis.

(a) Abdominal X-rays

An x-ray of the abdomen usually is taken of any patient suspected of having pancreatitis. This is done in order to provide some positive evidence to support the diagnosis, and to rule out the possibility of other acute abdominal conditions such as bowel ischemia (compromised blood supply to the intestine) or perforation. Both ischemia and perforation of the bowel display symptoms and serum enzyme levels nearly identical to those of acute pancreatitis. The presence of free air in the peritoneal cavity on the radiograph indicates perforation of the gut; thickenings or gas in the bowel wall, or gas in the portal vein indicates bowel ischemia.

In addition, these x-rays may help distinguish between attacks of acute pancreatitis and chronic pancreatitis. If calcifications (deposits of calcium carbonate in the tissues) are present on the radiograph, this strongly suggests the patient is suffering an acute attack of chronic pancreatitis.

Duodenal ileus (acute duodenal obstruction caused by a dysfunction of the nervous system) is a relatively unusual finding in abdominal x-rays but, if found, supports the diagnosis of acute

pancreatitis. A more common radiologic finding in acute pancreatitis may be distension of the lesser sac (a cavity in the membrane lining the abdominal cavity and enveloping the organs). This distention forces the stomach forward in the abdomen and pushes the horizontal portion of the large intestine (or the transverse colon) down in the abdomen, away from the stomach. Normally, the transverse colon intimately relates to the lower portion of the stomach.

(b) Chest X-rays

Chest x-rays may reveal conditions associated with acute pancreatitis. Chest x-rays that show pleural fluid on the left side (left-sided pleural effusion) in a patient without any evidence of lung abnormalities nearly always raises the suspicion of acute pancreatitis. However, pericarditis (inflammation of the membrane surrounding the heart) may also show this finding.

(c) Gastrointestinal Examinations

Patients whose symptoms mimic a peptic ulcer or an intestinal malignancy will often be referred for gastrointestinal or GI examinations—usually a barium contrast exam.

(d) Ultrasonography

Two studies performed in the early 1980s showed that ultrasonography adequately visualized the pancreas in 60 to 70 percent of the cases so its value in visualizing the pancreas itself was limited (McKay, 1982; and Silverstein, 1981). Ultrasonography appeared most useful in determining the cause of pancreatitis. The gallbladder was visualized in 70 percent of the patients, and the ultrasonography findings were 93 percent accurate in visualizing the presence or absence of gallstones. However, because newer techniques of ultrasonography have proven superior to the ultrasound scanners of the late 1970s and early 1980s, it has become standard practice in diagnosing pancreatitis. Ultrasonography is also valuable in detecting other maladies that present symptoms similar to pancreatitis (such as pancreatic cancer) and in visualizing complications of pancreatitis (such as pancreatic pseudocysts, abcesses, and ascites).

(e) Computed Tomography

Computed tomography is more specific in visualizing acute pancreatitis than ultrasonography.[13] Two studies conducted in the last decade showed that CT can evaluate changes in the pancreas in nearly all patients (Lawson, 1983; Mendez, 1980). The scan can provide information on the stage and the severity of the disease. Despite CT's success in visualizing the pancreas, the procedure is not indicated in most patients, since the diagnosis can be made with the less expensive techniques discussed above. A patient with obscure abdominal pain is a more likely candidate for CT than a patient with obvious acute pancreatitis. Additionally, CT might be indicated in a patient who appears to have acute pancreatitis but does not respond to standard medical therapy.

(6) Diagnostic Laparotomy

In a very small percentage of cases of acute pancreatitis, it is necessary to surgically explore the abdominal cavity to exclude the possibility of nonpancreatic diseases that mimic pancreatitis. If acute pancreatitis is found during a laparotomy, several procedures can be undertaken depending on what the surgeon finds. If cholelithiasis (gallstones) is found and the pancreatitis is very mild, removal of the gallstones may be undertaken to prevent subsequent episodes of pancreatitis. If severe gallstone pancreatitis is found, a drain usually will be constructed in the gallbladder to syphon its contents (cholecystostomy).

§ 5.73 Treatment of Acute Pancreatitis

Nonoperative treatment can be used to treat a majority of patients with mild to moderately severe acute pancreatitis. Operative measures are only indicated in a small percentage of cases to explore the abdominal cavity or to treat some types of pancreatic trauma. Treatment of acute pancreatitis can be categorized as specific and supportive. Specific (also termed "definitive") measures seek to limit the severity of the inflammation and prevent complications. Supportive measures treat complications as they arise.

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[13] *See* § 5.44(3) *supra.*

(1) Nonoperative Treatment

Specific nonoperative measures may include inhibition of pancreatic secretion, inhibition of pancreatic enzymes, and administration of antibiotics.

(a) Inhibition of Pancreatic Secretion

Inhibiting pancreatic secretion can be accomplished through nasogastric (insertion of a tube down the esophagus and into the stomach to collect gastric juices) suction and pharmacologic treatment. Nasogastric suction reduces vomiting and abdominal distention, and may reduce pancreatic secretion by decreasing the amount of contact the small intestine has with acidic gastric juices. Pharmacologic treatment to inhibit pancreatic secretion may include anticholinergics (drugs that inhibit the secretory activity of glands), glucagon, and cimetidine (an antihistamine that inhibits the production of gastric juice).

(b) Inhibition of Pancreatic Enzymes

Inhibiting the action of pancreatic enzymes may reduce the severity of acute pancreatitis. Aprotinin, an extract from bovine (referring to cattle) parotid (salivary) glands, may inhibit the action of trypsin and thus reduce the amount of pancreatic injury. Aprotinin has been researched extensively but is not currently recommended as a drug to treat acute pancreatitis.

(c) Antibiotics

The use of antibiotics in treating acute pancreatitis has sometimes been used as prophylaxis (prevention) against infection and to lessen the severity of pancreatitis. However, several studies show that antibiotic administration does not decrease the morbidity from infectious complications of acute pancreatitis (Ranson, 1978; Finch, 1976; Craig, 1975; Kodesch and Dupont, 1973; and Cogbill and Song, 1970). Antibiotics should be instituted when an infection does develop during pancreatitis.

(d) Supportive Nonoperative Treatment

Supportive nonoperative treatment is a very important aspect of treating acute pancreatitis as there are relatively few specific measures that effectively limit the severity of pancreatitis and prevent its complications. Most supportive measures involve

monitoring respiratory function, restoring and maintaining the volume of blood, replacing electrolytes such as calcium and magnesium, and providing nutritional support and pain relief.

Respiratory care is one of the most important supportive measures. Left-sided pleural effusions (the accumulation of fluid on the lungs) in addition to other respiratory complications can occur during acute pancreatitis. One study even associates acute respiratory distress syndrome (ARDS; rapid accumulation of fluid in the lungs associated with fluid replacement therapy in cases of massive trauma) with acute pancreatitis (Interiano, 1972).

(2) Operative Treatment

Operative measures are not often indicated in cases of acute pancreatitis. Occasionally, however, operative management of acute pancreatitis is necessary.

Specific operative measures may include biliary operations, pancreatic drainage, pancreatic resection, and peritoneal lavage.

(a) Biliary Operations

To limit the severity of the pancreatic inflammation, early biliary surgery may be necessary. This operation should only be performed if diagnosis of pancreatitis has been confirmed.

(b) Pancreatic Drainage

To prevent complications from occurring, some have suggested early operative drainage of the pancreas. However, one clinical study showed a significant increase in the frequency of sepsis (systemic infection) among patients who underwent early drainage (Ranson, 1974).

(c) Pancreatic Resection

In the past, removal of part or all of the pancreas (resection) was done in an attempt to alleviate the symptoms and complications of acute pancreatitis. Currently, resection is used primarily in cases of pancreatic trauma with associated injuries.

(d) Peritoneal Lavage

Peritoneal lavage (the introduction of a hypodermic needle through the abdominal wall to infuse and withdraw fluids) is

sometimes indicated in the treatment of acute pancreatitis. Numerous studies have observed a significant clinical improvement in patients treated with peritoneal lavage (Ranson, 1976; Bolooki and Gliedman, 1968). There are risks associated with the procedure, however. The most obvious, if infrequent, is damage to abdominal organs. The second risk is the possibility of aggravating respiratory insufficiency with the addition of two liters of fluid into the abdominal cavity. The second demands close monitoring of respiratory status during lavage.

Supportive operative measures are most important in treating the complications of acute pancreatitis. Complications such as paralytic ileus (nonmechanical obstruction of the bowel due to paralysis of the intestinal wall muscles) and duodenal or biliary obstructions usually resolve as the pancreatitis subsides and require no specific treatment. Pancreatic abscesses, on the other hand, are a serious complication that require prompt diagnosis and treatment. Pancreatic abscesses usually are lethal unless they are drained quickly.

§ 5.80 Chronic Pancreatitis

Chronic pancreatitis is characterized by permanent structural or functional damage to the pancreas even though the causes of the pancreatitis may be eliminated. Like acute pancreatitis, the chronic version presents with pain in the epigastric region. However, chronic pancreatitis is associated much more with alcohol consumption than with biliary tract disease.

§ 5.81 Etiology

Excessive alcohol consumption is considered the main cause of chronic pancreatitis in Western societies. Less common causes include hyperparathyroidism (an excessive production of the parathyroid glands' hormone, parathormone), hereditary pancreatitis and trauma. Acute pancreatitis is rarely a cause of chronic pancreatitis.

(1) Alcohol Consumption

At least two studies have documented a positive correlation between alcohol consumption and the risk of developing chronic pancreatitis (Dubec and Sarles, 1978; and James and Agnew, 1974). The risk of developing chronic pancreatitis also seems to increase with the duration of alcohol consumption. The period of intake can range from six to 17 years. Despite the alcohol abuse, there is a low correlation between chronic pancreatitis and cirrhosis of the liver.

(2) Hyperparathyroidism

Pancreatitis is felt to occur in four to seven percent of patients suffering from hyperparathyroidism. However, more recent studies observed pancreatitis in only 1.5 percent of patients with hyperparathyroidism (Bess, 1980). The hypercalcemic (an abnormally high serum calcium level) state produced by hyperparathyroidism has been suggested as the underlying cause of the pancreatitis.

(3) Hereditary Pancreatitis

Hereditary pancreatitis is an autosomal (carried by the chromosomes which do not determine gender) dominant genetic disease and affects males and females equally. Most persons affected are Caucasian with a northern European ancestry .

(4) Trauma

A blunt or penetrating blow to the abdomen or back may produce significant pancreatic damage. If trauma is the cause for chronic pancreatitis, it is important to recognize this fact immediately since the associated ductal constriction or dilation responds poorly to medical treatment.

§ 5.82 Diagnosis of Chronic Pancreatitis

Differentiating between the acute and chronic forms of pancreatitis can be difficult. However, findings from the clinical

presentation, patient history, laboratory studies, and diagnostic imaging techniques make diagnosis possible.

(1) Clinical Presentation

Pain is the most frequent symptom of chronic pancreatitis (Go, 1986). Located primarily in the epigastric region, it often radiates to the back. As in acute pancreatitis, the pain is continuous and usually lasts for several days. Patients who suffer from chronic pancreatitis and continue to drink alcohol may have intermittent attacks of severe pain or may eventually become pain-free. In the latter case, the clinical presentation will be dominated by secondary symptoms such as weight loss.

Weight loss is also a common primary symptom of chronic pancreatitis. In some cases, the weight loss occurs early in the progression of the disease and is due to decreased consumption of food resulting from the patient's fear of precipitating a painful attack. As the disease progresses, however, the weight loss is due to malabsorption (the failure of the intestine to absorb nutrients, especially fats or proteins, often presenting as persistent diarrhea).

Malabsorption occurs late in chronic pancreatitis and is evidenced by steatorrhea (fatty stool). This occurs because the extensive pancreatic damage halts the production of lipase.[14]

(2) Patient History

Patient history is very important in diagnosing chronic pancreatitis. Questions regarding alcohol consumption help to confirm the diagnosis.

(3) Laboratory Studies

In chronic pancreatitis, routine laboratory studies may be very unrevealing. For example, hyperamylasia (elevated serum amylase levels) is sometimes seen in a patient with chronic pancreatitis but is much more likely to be elevated in a patient with acute pancreatitis (Brooks, 1983). Therefore, diagnosing chronic

[14] *See* § 5.21(3) *supra.*

pancreatitis depends primarily on pancreatic function tests and imaging tests to detect structural abnormalities. The exact tests to use and in what order to use them are matters of controversy.

(4) Tests of Pancreatic Function

Tests for chronic pancreatitis measure exocrine secretion since this decreases with long term inflammation. These tests can be divided into two categories: noninvasive and invasive. The noninvasive tests measure the malabsorption that results from pancreatitis. The invasive tests involve stimulating the pancreas, then collecting its secretion for analysis. Although invasive tests are among the best measures of pancreatic function, collecting samples through gastrointestinal intubation (placing a tube through the esophagus and stomach into the duodenum) presents some difficulties.

Though noninvasive tests are not as specific or as sensitive as invasive tests, they are easier to perform and usually are indicated prior to invasive tests. If noninvasive tests are normal and imaging tests are negative, then invasive procedures are implemented.

(a) Non-Invasive Tests

The most common noninvasive procedures are the nitro blue tetrazolium-para-aminobenzoic acid (NBT-PABA) test and the pancreolauryl test. The NBT-PABA test is most sensitive in cases of severe chronic pancreatitis. Because NBT-PABA is broken down by the enzyme chymotrypsin in the intestine, the NBT-PABA test measures the amount of PABA in the urine and therefore indirectly indicates the amount of chymotrypsin secreted by the pancreas. A severe decrease of urine PABA corresponds to a severe decrease in chymotrypsin secretion and to severe pancreatitis. Unfortunately, in patients with mild chronic pancreatitis and with mild decreases in chymotrypsin, the test is not helpful.

In the pancreolauryl test, the amount of fluorescein (a dye that glows in the dark after being exposed to light; fluorescein is produced by the break down of pancreatic arylesterase, an enzyme) in the urine is measured. This value is an indirect indication

of pancreatic arylesterase secretion. A severe decrease of urinary fluorescein corresponds to a severe decrease in arylesterase secretion and to severe pancreatitis. As with NBT-PABA test, the pancreolauryl test is not helpful in diagnosing mild cases of chronic pancreatitis.

(b) Invasive Tests

Invasive tests employing secretin (a duodenal hormone that stimulates the production of pancreatic juice) and cholecystokinin (CCK; an intestinal hormone that stimulates contraction of the gallbladder) are most often used in conjunction with imaging tests to diagnose pancreatic disease when noninvasive procedures are inconclusive. Secretin and CCK can be given intravenously to stimulate pancreatic secretion. These secretions can then be collected by the gastrointestinal tube and examined for abnormalities resulting from chronic pancreatitis.

Invasive tests that employ the use of nutrients to stimulate pancreatic secretion are less specific than those that employ hormones, because nutrient stimulation must rely on hormonal secretion from the intestinal tract to stimulate pancreatic activity. However, nutrient stimulation is simpler, less expensive, and may produce fewer side effects than the use of hormones. A liquid meal containing certain ratios of fats, carbohydrates and proteins (commonly referred to as the Lundh meal) is sometimes used to stimulate pancreatic secretion but will give significantly more false negative results than hormone stimulation.

(5) Diagnostic Imaging Tests

Imaging techniques have become a major tool in diagnosing chronic pancreatitis. Changes in size or shape of the pancreas and pancreatic calcifications can be detected on an x-ray and confirm the diagnosis of chronic pancreatitis. Several types of imaging tests can be used: abdominal x-rays, chest x-rays, ultrasonography, computed tomography (CT) and endoscopic retrograde pancreatography. However, some of these tests are expensive and will not reveal early chronic pancreatitis. Therefore, the results of routine laboratory studies and conventional abdominal and chest x-rays will probably dictate what additional tests are needed.

(a) Abdominal X-rays

An abdominal x-ray is important in ruling out other acute abdominal conditions and in distinguishing between chronic and acute pancreatitis. An abdominal x-ray showing pancreatic calcification will confirm the diagnosis of chronic pancreatitis with a high level of confidence.

(b) Chest X-rays

Chest x-rays and abdominal x-rays may reveal abnormalities which occur in patients who have chronic pancreatitis and are having an acute attack of pain. However, they will not aid in differentiating between the acute and chronic form of the disorder.

(c) Ultrasonography

Ultrasonography is most useful in detecting diseases that mimic chronic pancreatitis such as hepatic (liver) and biliary disease, pancreatic cancer, and retroperitoneal lesions. An ultrasonogram's sensitivity in detecting chronic pancreatitis lies between 60 and 70 percent (Hessel, 1982; Cotton, 1980; Kunzmann, 1979; and Cotton, 1978). Ultrasonography may also reveal duct dilation which often occurs in chronic pancreatitis. Ultrasonography is less sensitive in detecting the presence of pancreatic calcifications.

(d) Computed Tomography

Computed tomography scans (CT) are 10 to 20 percent more sensitive than ultrasonography in visualizing chronic pancreatitis (Hessel, 1982; and Husband, 1977). CT scans display changes in the size and shape of the pancreas, reveal small lesions that cannot be visualized in abdominal x-rays and delineate pancreatic duct dilation. However, CT is expensive and exposes the patient to ionizing radiation. Therefore, it usually is recommended only when ultrasonography fails for technical reasons or if the diagnosis is uncertain (Go, 1986).

(e) Endoscopic Retrograde Pancreatography (ERP)

ERP (also referred to as endoscopic retrograde cholangiopancreatography or ERCP) is an invasive test that delineates the ductal system by the injection of a contrast medium (any material that absorbs x-rays) directly into the ducts. This provides unique

information about the ductal system that ultrasonography and CT cannot provide. Although ERP is very sensitive and specific, there are complications associated with this procedure, including acute pancreatitis. ERP usually is indicated for defining the ductal anatomy prior to operation, and to locate complications such as pancreatic pseudocysts and ductal leaks.

§ 5.83 Treatment of Chronic Pancreatitis

Chronic pancreatitis can be treated either by surgical means or with medications. Nonoperative treatment might also include altering the patient's diet by eliminating alcohol and fat.

(1) Operative Treatment

Seventy to 80 percent of patients can obtain relief from their pain through surgical intervention (Brooks, 1983). There are no standard operations for this disease; diagnosis often dictates what type of surgical intervention is best. Certain procedures, however, are more common than others. The three most common types of procedures include surgery of the biliary tract, pancreaticojejunostomy, and resection of the pancreatic head and body or body and tail (distal resection). Less frequently, a total pancreatectomy (removal of the entire pancreas) will be performed.[15]

(a) Operations on the Biliary Tract

Because biliary tract disease ranks second as a cause for chronic pancreatitis, most patients who have gallstone pancreatitis will be operated on sooner or later to stop the pancreatitis from recurring.[16] Prior to the operation, ERP may be indicated to define the ductal anatomy. At the time of operation, the gallbladder will probably be removed (cholecystectomy) and the entire common duct will be explored and evaluated.

(b) Pancreaticojejunostomy

In patients whose chronic pancreatitis is caused by intraductal plugs or stones, a portion of the pancreatic duct can be connected

[15] *See also* Medical Malpractice: Guide to Medical Issues, ch. 34 (Matthew Bender, 1986).

[16] *See* § 5.71(1) *supra*.

to the jejunum to drain properly. The connection may be direct or it may be accomplished via a Roux-en Y loop. (*See Figure 9–9.*)

(c) Subtotal Pancreatectomy

Subtotal pancreatectomy (resection of either the pancreatic head and body or the organ's body and tail) is indicated in patients with painful chronic pancreatitis associated with alcoholism where there is no ductal dilatation and where major parenchymal (referring to the characteristic, functional tissue of a gland or an organ) disease, fibrosis, and cystic disease are present.

The location of the lesions dictates the section of the pancreas to be removed. If the disease exists in the body and the tail, then a distal resection (removal of the body and tail) will be performed. If the disease exists primarily in the head and body, then a Whipple procedure (resection of the head and body) is the procedure of choice.

(d) Total Pancreatectomy

Total pancreatectomy (removal of the entire pancreas) is the ultimate therapy for chronic pancreatitis. However, while this procedure may offer relief from pancreatitis, the patient loses the endocrine and exocrine functions of the pancreas. Thus, the removal of the entire gland is only indicated where the benefits of removal far outweigh the effects of losing pancreatic endocrine and exocrine function. Another important consideration is the selection of patients for the operation. After the pancreas is removed, patients must be very self-disciplined to manage the diabetic condition that will result from removal of the pancreas. Unfortunately, self-discipline may not be evident in patients who are suffering from chronic pancreatitis as a result of alcohol abuse.

(2) Nonoperative Treatment

Nonoperative treatment for chronic pancreatitis seems to be of minimal effect. Discontinuing alcohol and fat ingestion is an important component of managing chronic pancreatitis. Also, antacids and anticholinergics are sometimes used to decrease the stimulation of the pancreas. Unfortunately, when these

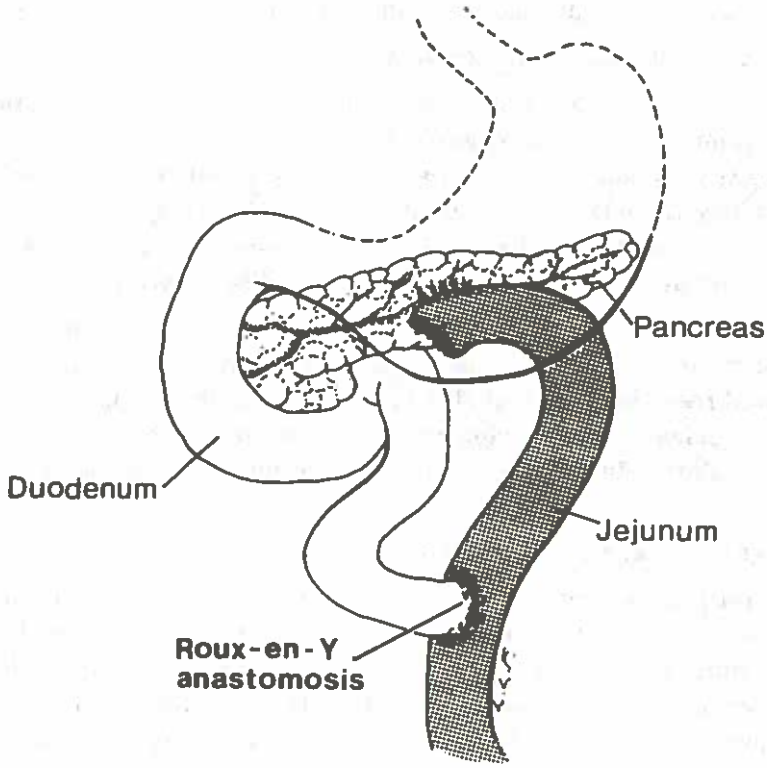

Fig. 5–9. A Roux-en-Y pancreaticojejunostomy.

interventions fail, patients are often incapacitated by a debilitating pain. In these cases, surgical treatment may be indicated for pain relief.

§ 5.100 Bibliography

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Wynn, M., et al.: Management of Pancreatic and Duodenal Trauma. Am. J. Surg. 150(3):327–332, Sept. 1985.

§ 5.200 Abstracts

Feliciano, D.V., et al.: Management of Combined Pancreatoduodenal Injuries, Ann Surg, 205:673–679 June 1987

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Pancreatoduodenal injuries commonly occur as a result of penetrating wounds and are usually seen in patients who have other, multiple intra-abdominal wounds. The incidence of perioperative mortality is related to the severity of pancreatoduodenal injury as well as to the magnitude of the associated injuries. Late morbidity and mortality rates, however, may be associated with the type of surgical repair with the subsequent formation of fistulas leading to the breakdown of secondary repairs that results in postoperative hemorrhaging.

Other complications associated with operative treatment of pancreatoduodenal injuries include the formation of an intra-abdominal fluid collection or an abscess. Percutaneous drainage or reoperation is required in the treatment of these patients some of whom may progress to multiple organ failure resulting in death. Furthermore, formation of a duodenal fistula, which may either heal spontaneously or require reoperation to improve draining, has been reported postoperatively.

Several other sequelae associated with surgical treatment of pancreatoduodenal injuries have been reported to occur. These include acute respiratory failure, pneumonia, and atelectasis. Renal failure as well as the occurrence of intra-abdominal gastrointestinal and biliary fistulas are also commonly seen. It should be noted that, because of the large number of combinations of injuries to the pancreas and duodenum which may occur, there is no one particular form of operative intervention that is therapeutic for all patients.

Morel, P., and Rohner, A.: Surgery for Chronic Pancreatitis, Surgery, 101:130–135, February 1987

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The surgical treatment of chronic pancreatitis involves either resection or diversion, neither of which has proven to be more clinically beneficial than the other. It should be noted, however, that treatment with surgical resection is associated with a number of both early as well as late complications.

In this series, three patients who underwent a distal pancreatectomy expired during their hospital stay; one patient succumbed as a result of pulmonary embolism, another had septicemia, and the third patient experienced massive hemorrhaging of the bowel due to multiple erosions.

The postoperative morbidity rate for resection, however, was higher than that for diversion. Most of the local complications seen included the accumulation of intra-abdominal fluid that required drainage. Systemic complications which occurred included five cases of moderate pulmonary infections.

Reoperation was necessary in five patients due to the recurrence of symptoms. Two patients required biliary diversion following drainage of a cyst. A third patient required insertion of a splenorenal shunt to relieve thrombosis of the superior mesenteric vein. Two additional patients respectively underwent secondary cholecystectomy as well as resection of the distal pancreas.

There were several late deaths in this study that were attributable to the treatment method of choice for chronic pancreatitis. Seven patients died of general debilitation due to juaundice or pancreatic dysfunction, six patients died of nonpancreatic cancer, and two died of cirrhosis. Cholangitis with multiple liver abscesses developed in two patients who had been treated with biliary diversion, one alcoholic patient died of hypogycemia, and another patient expired following massive hemorrhage due to an anastomotic ulcer.

Taxier, M., et al: Endoscopic Retrograde Pancreatography in the Evaluation of Trauma to the Pancreas, Surg Gynec Obstet 150:1 (Jan) 1980

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Precise delineation of the extent and nature of a pancreatic injury is critical to proper operative or nonoperative treatment.

Endoscopic retrograde pancreatography had become readily available and may have an important role in the assessment of patients in whom pancreatic injury is suspected. Six patients who had blunt abdominal trauma and were thought to have disruption of the pancreas from the basis of this report. In five of these patients, pancreatic injury was demonstrated by endoscopic retrograde pancreatography and confirmed at laparotomy.

Five of the six patients had suffered blunt trauma in automobile accidents. The sixth patient had been struck across the abdomen by a pole in a go-cart accident. In two patients, the injury had occurred three and six years previous to admission.

In the first patient, endoscopic retrograde pancreatography visualized only the proximal portion of the main pancreatic duct with an abrupt cutoff in the region of the head of the pancreas. Extravasation of contrast material filled a 2.0 by 3.0 cm cavity. These findings were interpreted to be an obstructed main pancreatic duct with leakage of contrast material into the communicating pseudocyst. At operation, a pseudcyst was noted in the head of the pancreas with a two cm rent in the inferior border of the pancreas. A Roux-en-Y pancreaticojejunostomy was performed successfully.

Endoscopic retrograde cholangiopancreatography in the second patient showed the proximal five to six cm of the pancreatic duct to be opacified. An abrupt cutoff was noted at the junction of the head and body, with antegrade filling of the accessory duct along with flow of contrast material into the duodenum through the accessory papilla. At operation, a tense rock-hard pancreas was palpated, and a pancreatic fistula was resected. A Roux-en-Y pancreaticojejunostomy was created. Four months after operation, the patient was doing well.

In the third patient, endoscopic retrograde pancreatography was performed prior to scheduled laparototomy. Roentgenograms of the pancreas and bile ducts were normal and the patient was managed conservatively and discharged after an uneventful hospital course.

Pancreatography in the fourth patient showed an obstruction of the contrast agent in the pancreatic duct. Operative exploration revealed a transection of the distal part of the pancreas. A distal

pancreatectomy with oversewing of the proximal gland was performed with good results.

In the fifth patient, endoscopic retrograde pancreatography revealed opacification of the pancreatic duct. Preoperative angiography revealed a hypovascular mass in the region of the head of the pancreas. At operation, a pancreatic pseudocyst was drained through a cystographic stoma, and a distal pancreatectomy performed. The pathologic specimen showed chronic pancreatitis with fibrosis and atrophy; there was no long-term follow-up.

In the sixth patient, endoscopic retrograde pancreatography showed the main pancreatic duct to be tortuous and shortened. The findings were believed to be compatible with obstruction of the midbody of the gland. At laparotomy, findings included a recurrent pancreatic pseudocystand marked distal chronic pancreatitis with a dilated duct in the distal segment. A Roux-en-Y pancreatico-jejunostomy was created. At follow-up two months later, the patient was doing well.

Sims, E.H. et al.: Factors Affecting Outcome in Pancreatic Trauma, J Trauma 24:125 (February) 1984

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Pancreatic trauma still has a high death and complication rate. The number of deaths has been estimated to be between 17 and 32 percent, and the number of complications in most series is around 33 percent. In the series reviewed by the authors, the mortality rate was only 14 percent. They attributed this lower death incidence to the prompt transport of the patients to a well-equipped hospital.

It was possible to evaluate the outcome in 44 patients seen over a seven-year period. Six died and 12 developed fistulas (a penetrating wound or ulcer creating an abnormal passage between two body surfaces) and/or abscesses. Of the patients who died, four had colon injuries; one had a duodenal injury, and one had both colon and duodenal injuries.

The injuries in this series were caused by gunshot wounds (30 patients), stab sounds (seven patients) and blunt trauma (seven patients).

Since the patients with colon injuries also had the highest rate of intra-abdominal abscesses and fistuals, the authors concluded that these injuries are probably the most significant factor in the continuing high rate of morbidity and mortality in pancreatic trauma.

Cogbill, T.H., Moore, E.E., and Kashuk, H.L.: Changing Trend in the Management of Pancreatic Trauma, Arch Surg, 117:722, (May) 1982

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Injuries to the pancreas — a retroperitoneal organ filled with proteolytic enzymes which shares its blood supply with the duodenum, and is surrounded by major vascular structures — continue to challenge the knowledge and judgment of trauma surgeons. If such an injury is overlooked or underestimated it may lead to potentially dangerous complications. On the other hand, the authors warn that devastating consequences may result from inappropriately aggressive treatment of these injuries.

In 1975 the authors outlined an operative management program for pancreatic trauma. Since that time several additional techniques have been developed that seem to improve the general postoperative outcome. The current report emphasizes the role of these innovations when they were used over a three-year period in 44 patients operated on for pancreatic trauma. Drainage alone was used to treat 21 patients; by distal resection in nine; duodenal diversion in eight; and pancreatofuodenectomy in one. Active sump drainage was used in 27 cases and post-operatively early enteral feeding by needle catheter jejunosotomy was used in 24. Six patients did not survive the initial operation and two died postoperatively. There were pancreas-related complications in 13 patients.

Based on their comparison with their earlier study, the authors reached the following conclusion. Postoperative complications and mortality after pancreatic trauma are minimized by:

(1) Distal resection for perforations of the pancreatic body and tail with suspected ductal trauma:
(2) Duodenal diversion for concurrent pancreatic head and duodenal injuries;
(3) Active sump drainage for all but minimal pancreatic injuries;
(4) Early postoperative nutrition by needle catheter jejunostomy for patients with serious pancreatic trauma.

Jones, R.C.: Management of Pancreatic Trauma, Am J Surg, 150:698–704, December 1985

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Pancreatic injuries that can be associated with a high rate of complications are sometimes not recognized at the time of an initial operation. At the Parkland Memorial Hospital where the author practices, there are between 25 and 30 pancreatic injuries treated every year.

An analysis was made of 500 patients who sustained pancreatic trauma over a period of 35 years. The analysis covered the type and severity of the injury, diagnosis, location, method of management, complications , and mortality. The injuries were caused by penetrating trauma in 362 and blunt trauma in 138.

It was determined that the majority of patients who sustain penetrating blunt trauma to the pancreas can be managed with sump drainage, including those with gunshot wounds to the head of the pancreas. In two to three of patients with pancreatic injury, pancreaticodudenectomy may be indicated.

In the authors opinion, patients who require resection of 80 percent or more of the pancreas and do not have spenic injury should be considered for a Roux-en-Y anastomosis to the distal pancreas after ductal injury has been proved.

The best management for severe injuries to the body of the pancreas is pancreatectomy.

In this series, the mortality rate due to pancreatic injury has been less than 3.0 percent. It is rarely the cause of death. Few normotensive patients die. None of the patients in the series reported by the author who had an isolated pancreatic injury died.

The severity of the injury often dictates the appropriate treatment. For most pancreatic injuries, a conservative approach is indicated. This means a shorter operating time and less blood loss in unstable patients with multiple injuries. The most important thing for the surgeon to do is identify the ductal injury at the time of the initial operation and to institute surgical drainage.

COURTROOM MEDICINE—ABDOMINAL INJURIES

VOLUMES 10 AND 10A

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