Clinical Signs of Pancreatitis
Clinical Signs of Pancreatitis

Steven M Penny, BS, R.T.(R), RDMS

After completing this article, the reader should be able to:
- Discuss the vital functions of the pancreas.
- Describe the underlying physiologic alterations that lead to acute and chronic pancreatitis.
- Summarize the clinical signs of acute and chronic pancreatitis.
- Explain the etiology, diagnosis, and medical management of pancreatitis.
- Identify and distinguish the imaging characteristics of pancreatitis as seen on computed tomography and ultrasonography.

The pancreas is a complex organ that performs several vital functions, including the production of important hormones. Radiologic technologists must be familiar with normal pancreatic anatomy and physiology in order to perform functional imaging procedures in their daily practice. In addition, technologists must appreciate how any deviation from normalcy can disrupt homeostasis. This article reviews pancreatic function and discusses basic pancreas imaging. In addition, acute and chronic pancreatitis are covered, including the role of medical imaging in diagnosis and treatment.

This ASRT Directed Reading Classic was originally published in Radiologic Technology, July/August 2012, Vol. 83/No. 6.

Visit www.asrt.org/store to purchase other ASRT Directed Reading Classics.
Other pancreatic enzymes include the proenzymes trypsinogen, chymotrypsinogen, procarboxypeptidase, proelastase, kallikreinogen, and prophospholipase A and B. Unlike amylase and lipase, these enzymes are inactive until they reach the duodenum. The duodenum produces duodenal enteropeptidase, which converts trypsinogen into its active form, trypsin. Trypsin facilitates the activation of other proenzymes.

When partially digested food from the stomach reaches the duodenum, cells release the hormone cholecystokinin into the bloodstream. Cholecystokinin concurrently triggers the contraction of the gallbladder, relaxation of the sphincter of Oddi, and the release of pancreatic juices by the acinar cells. The acinar cells release digestive enzymes into tiny ducts scattered throughout the pancreatic parenchyma. These ducts act as tributaries that ultimately deposit the juices into the main pancreatic duct (duct of Wirsung). Once in the duct of Wirsung, the enzymes reach the hepatopancreatic ampulla, also referred to as the ampulla of Vater (see Figure 2), where they mix with bile from the liver and empty into the proximal portion of the duodenum through the sphincter of Oddi. Some individuals have an accessory duct (the duct of Santorini) in the pancreas. The accessory duct has a separate minor sphincter opening into the duodenum, typically located 2 cm proximal to the sphincter of Oddi.

Table 1

<table>
<thead>
<tr>
<th>Enzymes Produced by the Pancreas (Exocrine)</th>
<th>Enzymes Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amylase</td>
<td>Converts starch to sugar</td>
</tr>
<tr>
<td>Lipase</td>
<td>Converts fats to fatty acids and glycerol</td>
</tr>
<tr>
<td>Nuclease</td>
<td>Breaks down nucleic acids</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Neutralizes stomach acid</td>
</tr>
<tr>
<td>Trypsin, chymotrypsin, carboxypeptidase, elastase, kallikrein</td>
<td>Break down proteins</td>
</tr>
<tr>
<td>Phospholipase A and B</td>
<td>Convert phospholipids to fatty acids</td>
</tr>
</tbody>
</table>

The acinar cells produce more than 2 L of pancreatic enzymes (also referred to as pancreatic juices) per day. These pancreatic juices assist digestion once food enters the duodenum (see Table 1). Although the pancreas produces several digestive juices, amylase and lipase play a critical role in digesting food and provide critical clinical information concerning pancreatic function.

The acinar cells secrete amylase and lipase in their active form, which allows the body to use the enzymes instantly to aid digestion. Amylase converts starch to sugar. In addition to the pancreas, the salivary glands and — in small part — the ovaries, intestines, and skeletal muscles also produce amylase. Lipase transforms fats into fatty acids and glycerol. Because lipase is produced primarily by the pancreas, abnormal lipase laboratory findings are more specific in identifying pancreatic dysfunction. However, in the presence of diseases such as pancreatitis, lipase often rises more slowly than amylase.

Unlike the liver, which has its own true capsule, the pancreas is not enclosed by a sac and therefore can leak harmful digestive juices freely into the peritoneal spaces when inflamed or injured.
Oddi. Food within the duodenum mixes with pancreatic enzymes and bile, and digestion proceeds.

The endocrine cells of the pancreas are dispersed like islands of tissue between the clusters of the acinar cells, and thus, are referred to as the islets of Langerhans. Although their role is vital, they only constitute about 2% of the total pancreatic mass. The islets of Langerhans are composed of alpha, beta, and delta cells (see Table 2). The most significant endocrine function of the pancreas is to control blood glucose levels. The beta cells produce the hormone insulin. Insulin transports glucose to cells, stimulates glycogen formation, and ultimately reduces blood glucose levels. The alpha cells produce the hormone glucagon, which accelerates glycogen breakdown and causes blood glucose levels to increase (ie, the opposite effect of insulin). The delta cells create the hormone somatostatin, which acts as an alpha and beta cell inhibitor.

Congenital anomalies of the pancreas are uncommon. Agenesis is a malformation in which the dorsal pancreas fails to develop. Annular (ring-shaped) pancreas, ectopic (out of place) pancreatic tissue, and pancreas divisum are other clinically significant congenital anomalies. Pancreas divisum is the failure of the dorsal and ventral ducts to fuse, thereby producing a shortened main pancreatic duct that only drains the pancreatic head and not the entire pancreas. Consequently, the minor sphincter — a much smaller opening than the sphincter of Oddi — must take over most pancreatic drainage, which essentially leads to functional stenosis and the backup of pancreatic juices (see Figure 3). This anomaly can lead to both acute and chronic pancreatitis. Pancreas divisum is considered the most common anatomic variant of the pancreas. It occurs in 4% to 14% of adults; however, fewer than 5% of individuals suffer symptoms.

**Imaging the Pancreas**

Medical imaging can help provide early diagnosis, determine treatment options, and monitor pancreatic disorders. Computed tomography (CT) and ultrasonography play a vital role in the diagnosis of pancreatic irregularities. Other diagnostic examinations also are used, such as endoscopic retrograde Table 2

<table>
<thead>
<tr>
<th>Pancreatic Hormone</th>
<th>Produced by</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucagon</td>
<td>Alpha cells</td>
<td>Accelerates glycogen breakdown and causes blood glucose levels to increase</td>
</tr>
<tr>
<td>Insulin</td>
<td>Beta cells</td>
<td>Transports glucose into cells, stimulates glycogen formation, and ultimately reduces blood glucose levels</td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Delta cells</td>
<td>Inhibits alpha and beta cells</td>
</tr>
</tbody>
</table>

![Common bile duct](Common bile duct)  
![Minor sphincter](Minor sphincter)  
![Sphincter of Oddi](Sphincter of Oddi)  

**Figure 3.** Pancreas divisum. Note the abnormal arrangement of the pancreatic ducts.
cholangiopancreatography (ERCP), magnetic resonance (MR) imaging, MR cholangiopancreatography, and endoscopic sonography. For example, ERCP is most often used to diagnose pancreas divisum. CT is used to assess the pancreas when pancreatic abnormalities are suspected (see Figure 4). CT is superior to other imaging modalities because of the speed and accuracy at which it can provide high-resolution, diagnostic information. Unenhanced CT is used to analyze the size of the pancreas and evaluate surrounding structures. Although pancreatic imaging protocols vary among institutions, both oral and intravenous contrast agents help evaluate the abdomen and pelvis for interconnected pathology.

Oral contrast is administered 20 to 30 minutes prior to the examination and again just before the examination begins. Intravenous contrast should be administered at an adequate injection rate to obtain peak pancreatic enhancement. The pancreatic enhancement phase occurs between 40 and 70 seconds after intravenous injection of 150 cc low-osmolar contrast at a rate of 3 cc per second. Several authors recommend a 5-mm slice thickness. CT has few limitations for the diagnosis of pancreatic disorders; however, imaging patients with large body habitus can be challenging, and CT is often contraindicated for those with pre-existing renal dysfunction.

Ultrasonography often is used to screen the acute abdomen. Patients who present with clinical symptoms of pancreatic or biliary disease often are evaluated with sonography before scheduling more expensive and invasive procedures such as CT or ERCP. Technical imaging of the pancreas using ultrasonography depends on the skill and experience of the practitioner, the patient’s body habitus, and the accessibility of the pancreas with ultrasound (see Figure 5).

Sonographers must manipulate and correctly orient the transducer to the pancreas to obtain diagnostic images. The sonographer often offers a preliminary report, or assessment, during the interpretation phase of the examination under the guidance of a qualified physician. Frequently, the sonographer provides information about technical considerations, the sonographic appearance of anatomy, and the presence of any disease observed during the procedure. The normal sonographic appearance of the pancreas is less echogenic, or hypoechoic, than a normal liver. However, fat can infiltrate the organ, making its appearance brighter and more echogenic than the liver. Because the pancreas can blend in with adjacent abdominal fat and can be