Chronic pancreatitis leads to significant morbidity and health care costs, in large part due to the pain that it produces. Investigations have found that chronic pancreatitis leads to significant impairments in health-related quality of life [1]. The most notable impairment is in the ability to perform usual daily activities due to compromises in physical and emotional well-being. This was found to correlate with an increased incidence of unemployment and early retirement in this population [2]. Due to this substantial impact on each patient and on society, it is important to recognize this disease and appropriately treat those patients afflicted with chronic pancreatitis.

Chronic pancreatitis is an end result of various types of insults to the pancreas that induce inflammation. The most common etiology in the Western world is alcohol abuse, accounting for 70% of the cases. The next most common cause is idiopathic. Obstruction of the pancreatic duct can also lead to chronic pancreatitis. This can be caused by stones, trauma, pseudocysts, tumors, or even anatomic variants (eg, pancreas divisum).

Other less frequent causes of chronic pancreatitis include hypertriglyceridemia, hereditary and tropical diseases, and systemic processes such as cystic fibrosis. Most idiopathic chronic pancreatitis is not caused by an underlying genetic alteration. Mutations in the cationic trypsinogen gene, cystic fibrosis conductance regulator gene, or the serine protease inhibitor kazal type 1 gene do not account for the pancreatitis in most idiopathic pancreatitis patients. Autoimmune pancreatitis is becoming increasingly recognized as an etiology of chronic pancreatitis. It has been found to occur all over the world, but the incidence is higher in certain regions, such as Japan. This entity can be suspected in patients with elevated serum IgG4 and characteristic imaging findings, but a pancreatic biopsy showing extensive lymphoplasmacytic infiltrate and dense fibrosis is necessary to confirm the diagnosis. It is important to identify patients who may have autoimmune pancreatitis, as they may have a high response rate to steroids.

Classically, chronic pancreatitis is thought to arise from an abnormality in the main pancreatic duct. However, in recent years the presence of small duct chronic pancreatitis has
become more appreciated, especially in those cases that are not associated with alcohol use. This entity is more subtle than large duct disease and may escape initial imaging, requiring more sophisticated diagnostic tools. It must be considered in patients who have signs and symptoms of chronic pancreatitis yet have no obvious ductal abnormality.

Due to the permanent structural damage that characterizes chronic pancreatitis, these patients are at risk for complications such as pseudocysts, malabsorption, diabetes, and mechanical obstruction of the duodenum and common bile duct. Other more rare complications include pancreatic ascites (which can lead to pleural effusions), splenic vein thrombosis (leading to regional portal hypertension), portal vein thrombosis, and splenic artery pseudoaneurysms. However, the most common and clinically challenging result of chronic pancreatitis is abdominal pain.

Pathophysiology
Chronic pancreatitis is characterized pathologically by patchy inflammation and fibrosis. The pathophysiology of chronic pancreatitis is multifactorial and not completely understood but predominantly stems from 2 events. First, a mechanical or functional obstruction of the pancreatic duct causes the pancreas to secrete an inadequate amount of bicarbonate. Obstructions may be due to tumors, strictures, sphincter of Oddi dysfunction, or stones. Second, digestive enzymes in the exocrine pancreas are prematurely activated. This leads to interstitial fat necrosis and hemorrhage. Other factors that may contribute to changes in chronic pancreatitis include antioxidant depletion and association with autoimmune disorders such as Sjögren’s syndrome, primary biliary cirrhosis, and renal tubular acidosis. Also, there may be underlying ischemia that may cause ductal hypertension, thereby compounding the problem.

There are several postulations regarding the etiology of abdominal pain. These include high pressures in the pancreatic ducts and parenchyma, perineural inflammation, and a deranged negative feedback mechanism. Recently, it has been suggested that abdominal pain may be due to trypsin activating nociceptive neurons through proteinase-activated receptor 2 [3].

CASE STUDY
Initial Presentation
A 48-year-old white man with a history of significant alcohol use presents to his primary care physician complaining of an 8-month history of epigastric abdominal pain that radiates to his back.

History and Physical Examination
Although the pain is always present, it tends to intermittently worsen, such as after meals. The pain is occasionally associated with nausea and vomiting. He has tried acetaminophen for the pain but has not found relief with this. When the pain is particularly bad, he finds that taking oxycodone, which was previously prescribed after a tooth extraction, has taken the edge off the pain. Physical examination reveals normal vital signs and tenderness in the mid-epigastrium. Laboratory studies show normal serum amylose and lipase values and normal liver function tests. An abdominal computed tomography (CT) scan shows dense calcifications in the pancreatic parenchyma and dilation of the main pancreatic duct to 9 mm (normal, < 5 mm).

- What is the differential diagnosis?

There are several disorders that must be excluded prior to making the diagnosis of chronic pancreatitis. The main one is pancreatic cancer, which can present similarly with epigastric pain, weight loss, and jaundice. Pancreatic cancer may be suggested by a long or sharply demarcated pancreatic duct stricture and a high CA-19-9, but the sensitivity and specificity of these are not high. Complications of chronic pancreatitis, such as pseudocysts and obstruction of the duodenum or pancreatic ducts, can also present with similar pain. Acute pancreatitis also must be considered. The remainder of the differential diagnosis includes gallstone disease, peptic ulcer disease, and irritable bowel syndrome.

In addition, we have found that many patients referred to us for management of chronic pancreatitis do not indeed have this but rather are found to have a motility disorder of the stomach or small bowel. These 2 disorders are often difficult to distinguish from one another. Gastroparesis has been found to have a higher prevalence in patients with small duct pancreatitis [4], but it also exists independently in patients who are initially thought to have chronic pancreatitis. Some patients may have an underlying motility disorder that should not be overlooked. The treatment for this motility disorder is very different than that of chronic pancreatitis; therefore, it is important to have a low threshold to obtain a gastric emptying study to evaluate epigastric pain. This should especially be considered when the diagnosis is questionable and improvement is not seen with pancreatic enzyme supplementation. Gastric emptying studies are cost-effective, simple, and widely available. The similarities and differences in the presentations of these 2 disorders are summarized in Table 1. Similarities include abdominal pain located in the epigastrium and of a postprandial nature. However, the patient population usually differs in that chronic pancreatitis is often present in older males and alcoholics, while motility disorders tend to be seen in younger women and diabetics.
The diagnosis of chronic pancreatitis may be straightforward if there is large duct disease, which has abnormal results on most testing. However, diagnosing small duct disease can be more obscure. A systematic approach can facilitate diagnosis in these cases. This consists of beginning with laboratory data and radiologic imaging. The patient may need to then undergo endoscopic procedures to reveal the abnormality. As a rule, abnormalities in function develop earlier than abnormalities in structure [5]. Specifically, our extensive clinical experience indicates that at approximately 60% damage to the exocrine pancreas, the secretin test turns positive, but only at above approximately 75% damage can endoscopic retrograde cholangiopancreatography (ERCP) detect the abnormality [6]. Features of large duct and small duct chronic pancreatitis are summarized in Table 2.

Laboratory

Often physicians begin by obtaining amylase and lipase levels in patients who are suspected of having chronic pancreatitis. While these are elevated in acute pancreatitis, they are usually normal in chronic pancreatitis. It may be useful to check a serum trypsinogen level or a fecal elastase, which are abnormal in severe chronic pancreatitis, especially if the patient presents with diarrhea or steatorrhea.

Although only available at select centers, the most sensitive and specific test of pancreatic function is the secretin stimulation test. This is the gold standard, being the most sensitive and specific test for chronic pancreatitis [8]. In this test, the patient is given an infusion of intravenous secretin, which normally causes the pancreas to secrete bicarbonate. An orally or endoscopically placed tube collects duodenal fluid to check for the amount of bicarbonate secreted. A peak bicarbonate concentration of less than 80 mEq/L indicates pancreatic exocrine insufficiency. It may be necessary to perform an endoscopic ultrasound (EUS) or ERCP for diagnosis, but we have found that starting with a secretin stimulation test may be more cost-effective. Not only is it less expensive than an

### Table 1. Comparison of Chronic Pancreatitits and Gastroparesis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Chronic Pancreatitis</th>
<th>Gastroparesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main symptom</td>
<td>Abdominal pain</td>
<td>Abdominal pain</td>
</tr>
<tr>
<td>Other symptoms</td>
<td>Nausea, vomiting</td>
<td>Early satiety, nausea, vomiting</td>
</tr>
<tr>
<td>Location of abdominal pain</td>
<td>Epigastric</td>
<td>Epigastric</td>
</tr>
<tr>
<td>Character of pain</td>
<td>Postprandial (after 15–30 min, radiating to back</td>
<td>Postprandial (immediately), burning, crampy</td>
</tr>
<tr>
<td>Abdominal examination</td>
<td>Tenderness</td>
<td>Tenderness, distention</td>
</tr>
<tr>
<td>Etiologies</td>
<td>Alcoholism, cystic fibrosis, idiopathic, hypertriglyceridemia</td>
<td>Diabetes, scleroderma, hypothyroidism, post-vagotomy, idiopathic</td>
</tr>
<tr>
<td>Relieving factors</td>
<td>Eating smaller and less fatty meals, possibly leaning forward or sitting upright</td>
<td>Eating smaller and less fatty meals</td>
</tr>
</tbody>
</table>

### Table 2. Comparison of Large Duct and Small Duct Chronic Pancreatitis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Large Duct</th>
<th>Small Duct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex predominance</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>Diagnostic findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secretin stimulation test</td>
<td>Abnormal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Serum trypsinogen</td>
<td>Often abnormal</td>
<td>Typically normal</td>
</tr>
<tr>
<td>Diffuse pancreatic calcifications on imaging</td>
<td>Frequent</td>
<td>Infrequent</td>
</tr>
<tr>
<td>ERCP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Progression to steatorrhea</td>
<td>Frequent</td>
<td>Rare</td>
</tr>
<tr>
<td>Pain treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreatic enzymes</td>
<td>Poor to fair response</td>
<td>Good to excellent response</td>
</tr>
<tr>
<td>Surgical procedures</td>
<td>Sometimes helpful</td>
<td>Usually not indicated</td>
</tr>
</tbody>
</table>

ERCP = endoscopic retrograde cholangiopancreatography. (Adapted with permission from reference 7.)

• What is the appropriate workup to confirm suspected chronic pancreatitis?
CHRONIC PANCREATITIS

ERCP, but a secretin stimulation test also has fewer complications. Also, up to 30% of patients who have small duct disease can have ERCP results that are normal or have only subtle changes but have abnormal secretin stimulation tests. ERCP changes may be limited to side branch ectasia, although there is often discordance between endoscopists in readings. Furthermore, these changes can be normal in elderly patients [7]. Although aging affects the pancreatic ducts, it does not affect the secretin test.

Steatorrhea is a classic marker for exocrine pancreatic insufficiency. To investigate this further, fecal fat quantitation may be done by measuring fat excretion over 72 hours in a patient on a 100-g fat diet. A result of more than 7 g of fat excreted per day indicates malabsorption or maldigestion. However, this is essentially impractical. It is both difficult to ensure a 100-g fat diet and to properly collect the sample.

Radiology

Some findings on radiologic imaging may suggest chronic pancreatitis. These include pancreatic calcifications, ductal dilatation, pseudocysts, and atrophy of the pancreas. About one third of patients with chronic pancreatitis have pancreatic duct calcifications present on plain film. Abdominal ultrasound has a sensitivity of 60% to 70% and a specificity of 80% to 90% for chronic pancreatitis [9]. CT scan has a sensitivity of 75% to 90% and a specificity of 85% [10].

Endoscopy

Although secretin stimulation is overall a better test than EUS or ERCP, there are some cases in which these radiographic tests may identify an abnormality not found on the secretin stimulation test. Therefore, these diagnostic modalities should be used in combination if possible. The characteristic ERCP findings for chronic pancreatitis are beading of the main pancreatic duct and ectatic side branches. Strictures and stones are also characteristics of chronic pancreatitis noted on ERCP.

In recent years, EUS has been increasingly utilized and found to be quite helpful in the diagnosis of chronic pancreatitis. It requires a highly specialized gastroenterologist but may be as sensitive as ERCP [11]. Ductal findings seen in chronic pancreatitis are echogenic or irregular walls, stones, stricture, dilation, and visible side branches. Parenchymal findings suggestive of chronic pancreatitis include echogenic strands or foci, calcifications, lobular contour, and cysts. If the patient has 4 or more of the above findings on EUS, chronic pancreatitis is considered likely [12].

Diagnosis and Follow-up

The patient returns for a follow-up visit in 2 months after having undergone EUS, which confirmed chronic pancreatitis by demonstrating pancreatic duct stones as well as a pancreatic stricture, dilation of the main pancreatic duct, and parenchymal calcifications. The patient continues to have abdominal pain that is causing him to miss increasingly more days at his work as a construction worker.

- What lifestyle modifications and medical treatments should be recommended to this patient?

The treatment of chronic pancreatitis is focused on minimizing exacerbating factors and relieving symptoms. The first step should be encouraging patients to abstain from all alcohol intake. Patients should also be instructed to eat small, low-fat meals. Because the cardinal symptom in this disease is abdominal pain, most of the treatments are aimed at pain control. One study showed that smokers had less pain relief with treatment than their nonsmoking counterparts with chronic pancreatitis [13]; hence, it may be worthwhile to strongly encourage smoking cessation also. Management options for pain control range from pancreatic enzyme supplementation to surgery. It has been theorized that the natural course of chronic pancreatitis leads to burnout of the pancreas, at which point pain tends to decrease or diminish. However, this has not been found to hold true. Of note, the treatment suggestions below are generally in accordance with recommendations made by the American Gastroenterological Association.

Pancreatic Enzymes

The reason for administering pancreatic enzymes to patients with abdominal pain secondary to chronic pancreatitis is to effect pancreatic suppression via feedback inhibition of pancreatic exocrine secretion. Normally, cholecystokinin (CCK) promotes release of enzymes from the pancreas and pancreatic serine proteases deactivate CCK-releasing factor [14]. Serine proteases (trypsin, chymotrypsin, and elastase) are generally depleted in chronic pancreatitis. Thus CCK-releasing factor is not catabolized and CCK continuously stimulates the pancreas. Supplementation with pancreatic enzymes can inhibit pancreatic enzyme release, thereby allowing the pancreas to rest and decreasing pain. However, it has been shown that patients who benefit most from enzyme supplementation are those with less advanced disease, women, and patients with idiopathic chronic pancreatitis. In our experience in people with small duct disease, pancreatic enzymes have been shown to be 70% effective in pain control. They are only 25% effective in those with large duct disease, such as the majority of chronic pancreatitis patients who have alcoholic disease [15]. For this reason, if a patient with small duct disease fails to improve after a 1-month trial of pancreatic enzymes, the clinician must perform a gastric emptying study to determine if the administered pancreatic enzymes
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CASE-BASED REVIEW

are being emptied into the proximal small bowel where the feedback mechanism is operative.

Pancreatic enzyme supplementation is a reasonable treatment to begin in a patient with significant pain that has not improved with dietary modification. Viokase 16 has 16,000 U of lipase, 60,000 U of protease, and 60,000 U of amylase per tablet. This medication may be started at 4 tablets with each meal and at bedtime. It is not enteric coated and therefore must be given with a proton pump inhibitor or H₂ receptor blocker to prevent gastric acid from inactivating it. Enteric-coated enzymes are not as effective in decreasing pain as nonenteric-coated enzymes. The enteric-coated preparations do not consistently deliver their enzymes into the proximal small bowel, the site of feedback regulation [16]. The pancreatic enzymes can be discontinued once the patient is pain-free for 6 months. Approximately half of patients taken off pancreatic enzymes will have a relapse of pain but do well once the medication is restarted.

Pancreatic enzyme replacement is not only helpful in the treatment of pain but also for steatorrhea by facilitating weight gain and resolution of diarrhea. In this case, the enteric-coated preparation is given because fat absorption takes place throughout the small intestine. Two capsules of a potent high-lipase preparation may be given before meals. Thus, nonenteric-coated enzymes are preferred for treating pain, and enteric-coated enzymes are preferred for treating malabsorption (steatorrhea).

**Analgesics**

If a trial of appropriate pancreatic enzyme supplementation has not been beneficial, analgesics are the next step. Acetaminophen or nonsteroidal anti-inflammatory drugs may first be tried. If these are not helpful, we recommend using tramadol or propoxyphene. More potent narcotic medications are frequently prescribed to chronic pancreatitis patients, but these often initiate a dangerous cycle for the patient, ultimately doing them more harm than good. It is in most patients’ best interest to use nonnarcotic modalities for pain treatment. Tramadol and propoxyphene do not impair gastric emptying, whereas potent narcotic preparations do, making treatment with pancreatic enzymes problematic.

**Octreotide**

Although studies looking at octreotide as a treatment for chronic pancreatitis have been inconsistent, our clinical experience using this medication in refractory patients has been promising. Octreotide is a somatostatin analog. It strongly inhibits pancreatic secretions. It also significantly lowers CCK levels, which may explain its role in decreasing pain. Octreotide given at 200 µg subcutaneously 3 times per day was shown to be better than placebo in a multicenter, double-blind study [17].

**Antioxidants**

The formation of free radicals may lead to inflammation of the pancreas. Hence, studies have evaluated antioxidants as a possible treatment for chronic pancreatitis. One study suggested that the combination of vitamins C and E, methionine, and selenium might be beneficial [18]. Another study concluded that chronic pancreatitis patients had significantly decreased micronutrient intake due to diet modification secondary to pain [19]. However, antioxidant supplementation is not a well-established treatment for this disease.

**Cholecystokinin Antagonists**

Supplementation of pancreatic enzymes can inhibit the release of CCK, as indicated above. However, another place in the cycle that can be targeted to produce therapeutic benefits is direct inhibition of CCK. Proglumide is a nonspecific CCK-receptor antagonist that has been found to help quell pancreatitis pain [20]. Loxiglumide is a more selective agent. This CCK-A receptor antagonist was also found to clinically improve pain in pancreatitis [21]. Use of this agent for 4 weeks led to up to 58% clinical improvement versus 34% improvement in the placebo group. These agents are not yet used in clinical practice, but they are promising prospective treatments.

**Psychiatric Medications**

Tricyclic antidepressants and gabapentin have been used in treatment of pain in pancreatitis, as they do have a role in altering the perception of pain. They work by modifying neural transmission. There are no controlled trials to prove their efficacy, but anecdotal experience supports their use. If a component of underlying psychological disease is suspected, referral to a psychiatrist or psychologist who is trained in chronic pain management should be done.

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**What endoscopic interventions are available?**

Endoscopic treatment is recommended when pain cannot be controlled with analgesics and the other conservative measures outlined above. Interventions on endoscopy include dilation of pancreatic duct strictures, stone extractions, pancreatic duct sphincterotomy, and lithotripsy. Endoscopic placement of pancreatic stents should be considered if there is a dominant stricture in the main pancreatic duct. If there is diffuse dilation of the main pancreatic duct without a stricture, endoscopic therapy is usually not successful. One large study using an intention-to-treat analysis showed that 65% of patients treated with a variety of endoscopic methods experienced pain relief up to at least 5 years after the procedure [22], but this was not a placebo-controlled study. A group looking
CHRONIC PANCREATITIS

at collective data reported that 50% to 85% of patients treated with endoscopic therapy had improved pain at 15 to 25 months [23]. Again, however, there are no placebo-controlled trials with endoscopic therapy for chronic pain in patients with chronic pancreatitis. In addition, there is a risk of damaging the duct with the procedure, which can further complicate the disease. This modality should be reserved for centers that specialize in this.

Extracorporeal shock wave lithotripsy can be helpful because pancreatic duct stones are found in 22% to 60% of patients with chronic pancreatitis. These can obstruct the outflow of pancreatic secretions and cause increased intraductal pressure. This, in turn, can lead to tissue hypertension and even ischemia. Lithotripsy fragments these stones, which allows for improved results of endoscopic therapies. It is predominantly used outside of the United States. Pain relief in up to 91% of a select patient population has been demonstrated [24]. However, there are no controlled trials to date that have proven this to be efficacious.

Celiac Nerve Blocks

Nerve blocks can be performed percutaneously or endoscopically. Historically, this is done with alcohol, but more recently steroids have been used instead to prevent irreversible nerve injury. Although relief of pain can be achieved, it is often short-lived and multiple sessions are necessary. A study that compared the modes of delivery found that EUS-guided blocks produced better and longer lasting pain relief than those that were CT-guided, an average of 10 weeks versus 4 weeks, respectively [25]. In another study performed by the same group, EUS-guided celiac block was done by injecting bupivacaine and triamcinolone on each side of the celiac plexus [26]; 55% of patients reported significant improvement in pain, but this decreased to 10% at 24 weeks. This study also included a cost comparison between EUS ($1200) and CT ($1400) techniques and found that EUS was less expensive. However, those who were younger than 45 years and those who had prior surgery for chronic pancreatitis did not find improvement. There is also some suggestion that narcotic-dependent patients do not get much benefit from these nerve blocks [27]. Celiac plexus nerve blocks should be reserved for patients who are refractory to more conservative measures.

Endoscopic Intervention

The patient returns for follow-up 3 months after undergoing an ERCP and being started on Viokase 16 at 4 tablets with each meal and at bedtime as well as omeprazole 20 mg per day. On the ERCP, stones were swept out of the main pancreatic duct, and a stent was placed at the site of the stricture. The patient was admitted to the hospital several hours after completion of the ERCP with significant acute worsening of his abdominal pain and vomiting. He was treated for post-ERCP pancreatitis with bowel rest, intravenous fluids, and morphine. His pain subsided and he was discharged home after 5 days, able to tolerate his usual diet. Since then, the patient has had significant improvement of his chronic pain and is able to function at work again.

• When is referral to a surgeon appropriate?

Surgery should be considered in patients who fail medical and endoscopic treatments. Surgical techniques used in the treatment of chronic pancreatitis include surgical denervation, decompression and drainage operations, resection procedures, and autologous islet cell transplantation.

Thoracoscopic bilateral splanchnicectomy is minimally invasive. Nociceptive fibers of pancreatic origin are transected as they course through the thoracic region. A large study analyzing this technique found that initially pain scores were significantly improved but that half of the patients had recurrent pain at 5 years [28]. A more recent (but much smaller) study followed patients an average of 15 months and found this technique to improve patients’ quality of life. In particular, it demonstrated pain relief in 60% of patients with chronic pancreatitis pain, decreased pain medication use in 53% (several patients were able to discontinue narcotics), and mood improvement in 55% of patients. There was also a decrease in hospital admissions in 95% and a decrease in the number of days spent in the hospital in 75% [29].

Patients are eligible for a decompression or drainage surgery if they have a significantly dilated main pancreatic duct (conventionally defined as > 6 mm). The most common of these surgeries is a variant of the Puestow procedure (lateral pancreaticojejunostomy), which was the first surgical treatment accepted as being effective for pain management in chronic pancreatitis. According to the American Gastroenterological Association technical review [30], most series have concluded that these drainage procedures lead to pain relief in 80% of patients and that it can be done with very low morbidity and mortality [31–35]. However, they show that 40% of patients have recurrence of their pain by 2 years after the surgery [33,36,37]. An advantage of this type of surgery compared with others is that there is enough remaining pancreatic tissue so that the function is not compromised.

When the pancreatic duct is not dilated, drainage procedures are not indicated due to technical difficulties that limit their usefulness. Rather, these patients may be candidates for surgical resections. This entails removal of a portion of the pancreas, usually the head or tail. The classical Whipple procedure is a pancreaticoduodenectomy. Currently, variations of this procedure are being done to preserve the pylorus.
Duodenum-preserving pancreatic head resections are increasingly being done and have good long-term outcome data. The American Gastroenterological Association has reviewed surgeries for chronic pancreatitis and concluded that distal pancreatectomy does not have much success unless the disease is mostly isolated to the body and tail [30]. On the other hand, 85% of patients who undergo a pancreatic head resection achieve pain relief, even if the disease courses into the distal pancreas. It has been shown that the quality of life and nutritional status are excellent in patients who have undergone a Whipple procedure [38]. Total pancreatectomy may be performed if the patient fails a partial pancreatic resection. However, even with this major surgery, most patients continue to have pain.

Once there is less than 20% of pancreatic tissue remaining, the pancreas cannot function adequately. These patients can experience fat malabsorption and diabetes. If there is enough disease present, significant gland dysfunction can occur even when the pancreas appears normal on imaging studies. Because of exocrine or endocrine insufficiency experienced with total pancreatectomy, autotransplantation of a portion of the pancreas or of islet tissue has been investigated. This is done by crudely separating islets from the pancreas and infusing them into the portal vein. This has resulted in normoglycemia for up to 7 years post-transplantation [39]. However, the data are not all positive; this is still an experimental surgery that may be considered as a last resort in patients who have failed medical, endoscopic, and other surgical procedures [40,41].

No matter which surgery is performed for severe chronic pancreatitis, pancreatic enzyme therapy should be employed to enhance absorption and preserve nutrition. All too often patients are not treated with pancreatic enzymes even though they have severe enough damage to require surgery, and patients are not put on enzymes until they declare themselves with diarrhea or significant weight loss. If one waits until these patients become truly symptomatic, it is often hard for such patients regain their weight.

**SUMMARY**

Chronic pancreatitis is a lifelong disease that results from injury to the pancreas. Although alcohol is the most common cause, there are many other etiologies for this. It is important to have the diagnostic tools to identify patients who have small duct disease, as the diagnosis for this is more obscure than that of large duct disease. It is also becoming more evident that other disorders, such as gastroparesis, can mimic chronic pancreatitis. Originally thought to be a disorder limited to diabetics, we now see a growing population of others, such as young women, who have gastroparesis masquerading as chronic pancreatitis. Physicians should have a low threshold to obtain a gastric emptying study in patients who are likely to have this, because the treatments are very different. Health care dollars can be used more effectively if gastroparesis is identified early. If gastroparesis is not likely and chronic pancreatitis is the probable diagnosis, we recommend beginning with a plain radiograph or CT of the abdomen. The next diagnostic step is an EUS or a secretin stimulation test if available. If this is negative, proceeding to ERCP is a reasonable next step. Once chronic pancreatitis is confirmed, the treatment modalities range from pancreatic enzyme supplementation and analgesics to endoscopic and surgical treatments. The goal of these treatments is abatement of pain and prevention of complications, such as diabetes and fat malabsorption. It is critical that a proper diagnosis of chronic pancreatitis be made before such patients are sent to pain clinics where narcotics are often prescribed. If such a patient does not have chronic pancreatitis but rather has a motility disturbance, it is inappropriate to prescribe narcotics, which will make the situation worse by delaying gastric emptying.

**References**

CHRONIC PANCREATITIS

Clinical Approach to Chronic Pancreatitis

DIRECTIONS: Each of the questions below is followed by 4 possible answers. Select the ONE lettered answer that is BEST in each case and circle the corresponding letter on the answer sheet.

1. The most accurate test to detect small duct chronic pancreatitis is
   (A) Computed tomography (CT) scan
   (B) Endoscopic retrograde cholangiopancreatography (ERCP)
   (C) Secretin intubation test
   (D) Magnetic resonance cholangiopancreatography

2. Which of the following is usually normal in patients with alcohol-induced chronic pancreatitis?
   (A) Serum lipase
   (B) CT scan
   (C) Stool fat
   (D) ERCP

3. Which of the following pain medications does not decrease smooth muscle motility?
   (A) Meperidine
   (B) Morphine
   (C) Fentanyl transdermal
   (D) Tramadol

4. For the treatment of abdominal pain secondary to chronic pancreatitis, the most appropriate initial therapy is
   (A) Endoscopic stenting
   (B) Nerve blockage via endoscopic ultrasonography
   (C) Nonenteric-coated pancreatic enzymes
   (D) Sphincteroplasty

5. Which of the following is NOT characteristic of small duct chronic pancreatitis?
   (A) Progression to steatorrhea
   (B) Normal CT scan
   (C) Chronic abdominal pain
   (D) Normal serum trypsinogen
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   ______________________________________________________
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