9. Acute pancreatitis

Acute pancreatitis seems to be increasing in incidence, possibly because of the wide use of alcohol and the aging of the population. The incidence of gallstones is high in the elderly. In hospitals with large indigent populations, roughly two-thirds of the cases of acute pancreatitis are associated with alcoholism, whereas in private institutions two-thirds of the cases are associated with gallstones. The passage of small stones into the duodenum is probably an important causal factor, since stones have been demonstrated in the stools of patients with pancreatitis even though major obstruction of the ampulla of Vater by a stone is not commonly encountered. After these two major inciting factors (alcohol and gallstones), about 10 percent of cases can variously be attributed to trauma, hyperlipidemia, hyperparathyroidism, and drugs such as thiazide diuretics. In most reports, roughly 10 percent of cases cannot be classified according to cause.

It has been stated that the common failure to diagnose acute pancreatitis correctly is due to a failure to consider its possibility in the individual case, but even when the condition is thoroughly considered and discussed, a mistaken diagnosis frequently results. A special consideration of the symptoms is therefore all the more necessary.

To understand and remember the symptoms, one should recollect
the anatomy of the pancreas and the pathology of the disease. The gland lies in the retroperitoneal tissues in close relationship with the celiac plexus and ganglia. The head is surrounded by and slightly overlaps the duodenum; the body lies in front of the first lumbar vertebra, while the tail reaches the left loin and lies against the hilum of the spleen. There are still many points in the pathology of pancreatitis that are not settled, but there is a preponderance of evidence to show that the acute forms of inflammation may lead to severe, widespread hemorrhage into the gland and surrounding tissues, with subsequent disorganization of its substance and liberation and activation of its ferments. Pancreatic enzymes cause extensive destruction of retroperitoneal tissues. If a major pancreatic duct has been disrupted, a collection of pancreatic juice forms, bounded by whatever tissues happen to be in the area, and is called a pseudocyst. Less commonly, there is a free communication of the disrupted duct into the peritoneal cavity, giving rise to pancreatic ascites. Bacteria probably play no role in the early onset of the pancreatic inflammation but tend to cause secondary abscesses in the retroperitoneal about two weeks after the beginning of the attack. If the patient lives long enough, a part or the whole of the pancreas may become gangrenous, but this is very rare.

Acute pancreatitis seldom occurs before the age of twenty and is more common in stout people.

Symptoms

We describe a severe case. In milder cases the symptoms are correspondingly less acute.

The symptoms of acute pancreatitis are rather variable—a fact that explains the conflicting accounts of the disease published by individual observers. The one or two absolutely pathognomonic signs are rarely present because they are found late in the course of the disease; the more constant features must be carefully considered together before a diagnosis can be determined.
Acute Pancreatitis

Scess early in an attack because of fever and leukocytosis, only to find none.

Tenderness. Local epigastric tenderness is a constant finding.

Rigidity. Epigastric rigidity is by no means constant. It is true that soon after the onset there may be boardlike rigidity of the epigastric muscles, but when the patient is examined there is often a relatively lax abdominal wall. This point should be emphasized, since extreme muscular rigidity was at one time thought to be characteristic.

Symptoms and Signs Due to Enlargement of the Pancreas

Epigastric Tumor. Sometimes the pancreas may be palpable as a transversely placed tumor in the epigastrium. The fact that the patient is sometimes very stout and the occasional presence of rigidity often make the detection of a mass difficult.

Jaundice. Slight jaundice is found in about half the cases. Since frequently, if not usually, there are no obstructing gallstones, the most reasonable explanation for the jaundice is that the common duct is compressed by the swollen head of the pancreas.

Obstructive Vomiting. True obstructive vomiting of feculent or bilious material is very rare, but I have personal knowledge of one such case. At the operation, the swollen pancreatic head was definitely obstructing the duodenum. Obstructive vomiting must be distinguished from the more common reflex vomiting mentioned above.

Signs due to extravasation of blood

Echymosis. In hemorrhagic pancreatitis, some of the blood may find its way along the tissue planes and become evident as a green or greenish-yellow area in one or both loins. This was first noted by Grey Turner, who also, in one patient, saw a similar discoloration around the navel (sometimes called Cullen’s sign). This sign, when found, is pathognomonic of retroperitoneal hemorrhage, but it never occurs until two or three days after the onset of the disease. It usually signifies the presence of a rather severe form of the disease.

Other Symptoms and Signs

Cyanosis. This symptom has been noted in a considerable number of cases. It is best observed in the face and extremities but has sometimes been present in the skin of the abdomen.

Tachypnea and dyspnea. These are often noticeable. It is reasonable to suppose that a partial inhibition of the diaphragmatic movements, owing to the contiguous inflammation, may account, at any rate in part, for the cyanosis and dyspnea. A left pleural effusion is very common and likewise contributes to these symptoms, as does the sometimes extreme abdominal distention. Some have suggested that circulating lipases may destroy pulmonary surfactant and hence produce atelectasis and pulmonary failure.

It should be remembered that in the later stages of acute pancreatitis, a more general abdominal condition results; blood-stained fluid collects in the peritoneal cavity, distention supervenes, and there may be persistent high fever. It is very difficult to diagnose such cases without a very accurate previous history of the case. Very rarely indeed, subcutaneous areas of fat necrosis may develop in pancreatitis.

Laboratory Findings

Hyperglycemia and glycosuria. These are sometimes found and in any case of acute abdominal pain should raise the question of pancreatic disease.

Increase in the serum and urinary amylase. The liberation of the pancreatic ferments leads to an increase in the amount of amylase in the blood and the urine.
The serum amylase is less than 200 Somogyi units per 100 ml of serum in about one-third of cases; between 200 and 500 units in one-third; and greater than 500 in the remaining third. If an elevation is present, it is usually found within the first forty-eight hours, returning to normal at that time even if the disease remains active. However, inasmuch as the amylase content of the serum may be significantly raised in patients suffering from cholesystitis, high intestinal obstruction, acute renal insufficiency, perforated ulcer, and other conditions, the serum amylase is not as reliable a test for acute pancreatitis as it was at one time supposed to be. While determination of the urinary amylase improved the diagnostic accuracy somewhat, it, too, fell short of the mark. Recently it has been demonstrated in cases of pancreatitis that there is a discordant elevation in clearance of amylase by the kidneys in comparison with the creatinine clearance so that the ratio of amylase:creatinine clearance exceeds 5:1. Great hopes were held for this test but, as is so often the case, many exceptions have been found and high ratios have already been reported in burn patients and in individuals with diabetic ketoacidosis. It is probably of no greater value than a serum or urinary amylase or lipase test.

Hyperlipidemia. It is thought that primary hyperlipidemia may account for some cases, but so-called secondary hyperlipidemia is much more common. A lipemic serum in a seriously ill patient with abdominal pain almost always means pancreatitis.

Other laboratory findings. Albuminuria is common. If methemalbumin is detected in the serum of a patient suffering from acute pancreatitis, it is a case of hemorrhagic pancreatitis. Leucocytosis is extremely frequent.

Diagnosis

Acute pancreatitis is most commonly mistaken for a perforated gastric or duodenal ulcer. The less acute cases may be misdiagnosed as appendicitis, while those cases with distention may easily be regarded as examples of intestinal obstruction. Acute cholecystitis and biliary colic may also simulate the symptoms of pancreatitis.

With a perforated ulcer, general abdominal rigidity is constant in the early stages after perforation, while in pancreatitis the abdomen may be softer, and any rigidity is usually limited to the epigastric zone. In his original paper, Fitz very accurately wrote that the symptoms of acute pancreatitis were those of an epigastric peritonitis. In a case of perforated ulcer, the symptoms are usually more widespread. Pain on top of the shoulder is frequently felt when an ulcer perforates; with pancreatitis, such pain is rare; when present, it is felt on top of the left shoulder. Bilateral lumbar pain, cyanosis, fever to 102°F or 103°F, and slight jaundice would favor pancreatitis, while absence of liver dullness in the axillary line would definitely indicate perforated ulcer. Hyperglycemia, glycosuria, hyperlipidemia, or an increased serum or urinary amylase would point to pancreatitis.

Appendicitis is generally distinguishable if careful attention is paid to the history of onset and the order of symptoms. The vomiting and pain are both less severe in appendicitis, and there may be definite local symptoms in the right iliac fossa. With acute cholecystitis and biliary colic, tenderness is felt more in the right hypochondrium, and there may be a definite history of previous attacks. The laboratory tests may help to determine the condition, but it must be remembered that cholecystitis and pancreatitis may coexist.

When distention has supervened, pancreatitis is difficult to distinguish from the late stage of peritonitis and intestinal obstruction unless positive laboratory tests and a very clear history point to the correct diagnosis.

Perforation of a duodenal diverticulum, although not common, may give rise to a clinical picture indistinguishable from that of acute pancreatitis. The pain and tenderness are usually more localized to the right upper quadrant. Films may help to distinguish the condition by demonstrating gas bubbles in the right retroperitoneal area.

A CT scan is now one of the most helpful radiographic studies. The diagnosis of acute pancreatitis can be made with confidence when an
enlarged edematous pancreas with some associated retroperitoneal fluid is seen on the CT scan.

**PSEUDOCYST: A COMPLICATION OF ACUTE PANCREATITIS**

This complication of acute pancreatitis should be suspected if, after having come through the early phase successfully, the patient does not progress rapidly to full recovery. There is usually persistent fever, low-grade abdominal pain—especially after eating, anorexia, and occasional vomiting. A mass is sometimes palpable in the epigastrium. The serum amylase level, rather than returning to normal, as in the usual case, may remain persistently high. A CT scan will show a well-defined cystic collection whose resolution or progression is best determined by serial CT examinations. Whether a pseudocyst is infected or sterile can only be determined by aspiration of the fluid under CT or ultrasound guidance. Some pseudocysts will resolve spontaneously, while others require drainage.

**"PANCREATIC ABSCESS"**

This term is a misnomer because the condition is neither an abscess in the true sense of the word nor is it confined to the pancreas. Rather, there is extensive destruction of the peripancreatic and retroperitoneal fat accompanied by secondary infection, usually with gram-negative rods. These retroperitoneal tissues do not coalesce into an easily drained abscess cavity but require active debridement.

The clinical setting and findings are identical to those described above for pseudocyst except that the CT scan shows multiple poorly perfused areas of pancreatic and peripancreatic tissues without the well-defined collection seen with a pseudocyst. The diagnosis should be considered in any patient with acute pancreatitis who has persistent abdominal pain and fever. Prompt diagnosis is extremely important because the condition is uniformly fatal without surgical intervention.

In cases of nonresolving acute pancreatitis, the diagnosis is established with certainty if gas can be detected in the retroperitoneum by either a plain film or CT scan, if there is persistent bacteremia without any other cause, or if CT-guided aspiration of the peripancreatic and pancreatic areas yields bacteria (Plate 6B).

Some surgeons have advocated debridement when pancreatic and peripancreatic necrosis without superimposed infection is present. The diagnosis of necrosis alone (in absence of infection) has been based on poorly perfused areas shown on a contrast-enhanced CT scan. Whether these areas are in fact necrotic pancreas, and whether an operation should be undertaken for them, has not been clarified, although on occasion operation is indicated in a patient with uninfected necrosis whose general condition is deteriorating.
10. Cholecystitis and other causes of acute pain in the right upper quadrant of the abdomen

Severe pain arising in or chiefly localized to the right hypochondriac region is usually due to one of the following conditions:

1. Cholecystitis, with or without rupture
2. Biliary "colic" (a misnomer)
3. Inflamed or leaking duodenal ulcer
4. Hepatitis

But one always needs to exclude:

1. Appendicitis
2. Renal pain or colic
3. Pleurisy or pneumonia

The gallbladder and cystic duct may be regarded as a vermiform tube that has a dilated extremity and opens through the common bile duct into the duodenum. In certain respects, therefore, the gallbladder is analogous to the cecal appendix. Further, it is common for a stone to stop up the cystic duct just as a concretion may occlude the lumen of the appendix. The chief difference between the two structures lies in the fact that fecal material is present in the cecal appendix but not in the gallbladder, though *Escherichia coli* is frequently found in the latter. In addition, the muscular wall of the gallbladder and common bile duct is sparse, so that vigorous contractions do not occur as they do in the ureter or in the intestine.

Acute cholecystitis

**PRODROMAL STAGES**

The usual attack of acute cholecystitis begins with an episode of biliary colic (see Chap. 12). Since this pain is almost always without paroxysms or with only very minimal ones, one should not be misled by the term "biliary colic" or by the patient with *steady* pain. An attack of biliary colic may follow one of four courses:

1. It may subside over four to six hours because the calculus that caused the attack has either dropped back into the gallbladder or has passed out of the common bile duct.
2. Acute cholecystitis may supervene, often after an interval of several hours of freedom from pain.
3. A chronic hydrops of the gallbladder may form after impaction of the stone in the cystic duct provided that infection is not present and the mucosa of the gallbladder does not undergo necrosis.
4. Jaundice may occur transiently if the stone has passed into the duodenum or does not remain critically obstructive of the common bile duct. If it does not pass and partial obstruction of the common bile duct persists, then cholangitis may ensue.

It is to be emphasized that an *episode of biliary colic causes steady, non-paroxysmal pain* and that it may be the forerunner of any of the above-mentioned events. *Pain arising from a stone in the cystic duct or common bile duct is essentially the same because of identical segmental
innervation of these structures. Consequently, during the acute painful attack itself, the surgeon is rarely, if ever, able to predict which course of events outlined above will ensue. Occasionally acute cholecystitis occurs in the absence of gallstones or obstruction of the cystic duct, but this accounts for only about 5 percent of cases. Acalculous cholecystitis is most often observed in an otherwise seriously ill patient who is usually being given intravenous hyperalimentation.

SYMPTOMS, SIGNS, AND DIAGNOSIS

The main symptoms and signs are as follows:

1. Pain
2. Vomiting or nausea or complete loss of appetite
3. Jaundice (sometimes)
4. Fever
5. Palpable or visibly distended gallbladder
6. Tenderness and rigidity

Pain. The pain of biliary colic is described in Chapter 12. One point bears emphasis here: its constant, unrelenting nature and its very frequent midline epigastric position often mislead the physician. The acute pain usually disappears within four to six hours. After a pain-free interval of several hours, it returns, but now in the right hypochondrium and with a different character, that of a constant ache clearly aggravated by a motion, cough, or sneeze. The secondary pain is clearly that of peritoneal irritation and is analogous to the shift from visceral to somatic pain in acute appendicitis.

Vomiting. Anorexia, nausea, and vomiting are not only common in biliary colic but usually persist when acute cholecystitis supervenes. Vomiting is usually rather frequent and is bilious, not feculent, in character. Vomiting is occasionally completely absent.

Differential Diagnosis

Jaundice. A slight tinge of icterus is quite common in acute cholecystitis, even in the absence of stones in the common bile duct. Occasionally its presence is helpful in differentiating acute cholecystitis from a perforated ulcer. The more severe the attack of acute cholecystitis, the more likely the presence of icterus. Serum bilirubin levels of 3 to 4 mg percent are not at all uncommon in uncomplicated acute cholecystitis, and I have encountered one case that reached 11 mg percent.

Fever. Fever of about 101°F is common in acute cholecystitis. Higher fever or rigors, while occasionally observed in acute cholecystitis, are more likely to occur in cholangitis.

Palpable or visibly distended gallbladder. A palpable gallbladder has been reported in close to half of all cases of acute cholecystitis. The presence of a palpable gallbladder in a patient with a compatible history establishes the diagnosis of acute cholecystitis. A rough examiner who palpates too deeply will miss this finding because the patient will guard against the probe. I have often seen a distended gallbladder descend on deep inspiration while I was standing at the foot of the bed, with the patient in a good light, when the same turgid organ has been missed on palpation by an untrained, rough hand. Gentle palpation will often reveal a very hard mass slightly larger than a golf ball that cannot be mistaken for anything else once a few have been felt. Millions of dollars in diagnostic tests could be saved this way. With the rare exception of the tiny, shrunken intrahepatic gallbladder, years of operative experience indicate that the vast majority of gallbladders in acute cholecystitis are markedly distended and that, once anesthesia is induced, most are palpable. This suggests that if we train ourselves to be gentle, most gallbladders in acute cholecystitis will probably be palpable.

Differential Diagnosis

Cholecystitis must be distinguished from appendicitis, an inflamed or leaking duodenal ulcer, biliary colic, rupture of the gallbladder or bile duct, and hepatitis, but pleurisy must always be excluded (Fig. 21).
PAIN IN THE RIGHT UPPER QUADRANT

Hepatitis
Inflamed gallbladder
Periduodenitis (duodenal ulcer)
Pylitis (R)
Inflamed ascending appendix

Fig. 21. The differential diagnosis of cholecystitis.

Differential Diagnosis

The symptoms—pain, vomiting, constipation, fever—are very similar to those of appendicitis, but the site of localized pain is in the one case in the right hypochondrium and in the other in the right lumbar or iliac region. If any swelling is palpable, its continuity with or distinction from the liver is of prime importance in diagnosis. It must be allowed that there are some cases, especially in obese patients with a rather low-lying, inflamed gallbladder accompanied by local peritonitis and rigidity, in which a definite clinical differentiation from appendicitis with local abscess is almost impossible. A history of jaundice or biliary colic may be of assistance, or an account given of previous attacks suggestive of appendicitis might point to that disease. A CT scan will make this distinction by demonstrating an abscess cavity in association with an ileo-cecal phlegmon.

In some cases of acute cholecystitis, the initial pain may be felt in the epigastrum and may precede by several hours the pain and tenderness in the right upper quadrant of the abdomen. This is comparable to the epigastric or umbilical pain that may precede the right iliac pain and tenderness in cases of acute appendicitis. All patients suffering from acute epigastric pain unaccompanied by any other localizing symptom should be examined again within a few hours.

In difficult cases in which the gallbladder cannot be palpated, a radionuclide scan (HIDA) may be useful. Visualization of the gallbladder excludes the diagnosis of acute cholecystitis. Ultrasound examination of the gallbladder may demonstrate calculi (Plates 7A, B, 8A, B), but the degree of distention of the gallbladder and the thickness of the gallbladder wall are difficult to ascertain by ultrasound.

Case Report of the Author's Attack

(Z.C.)

The only previous abdominal crisis I had suffered was an attack of acute gangrenous appendicitis in 1907; an abscess formed and was drained, and later the stump of the appendix was removed at another operation.

For six months before my illness I occasionally had attacks of profuse sweating and increased pulse rate that woke me from sleep, but usually passed off within half an hour. They were not accompanied by any pain. A physician found my heart normal.

On 3 April 1969, I arose about 7 a.m., enjoyed a simple breakfast, and was up and about all the morning. Then, almost exactly at noon, I suddenly felt a dull severe, deep pain in the middle of the epigastrium. I lost all appetite for food and retired to bed. I wondered what might be the cause of the pain, and thought first of coronary thrombosis, but the pulse was normal in every respect, and there were no symptoms to support this diagnosis. My attention was then directed to the abdomen. I palpated the left side and the right iliac region without finding anything abnormal. In the right hypochondrium, however, a surprise awaited me, for in the normal position of the gallbladder was a rounded, tense, and firm swelling, about the size of a small golfball. It was not painful or tender. This absence of tenderness probably put all thoughts of acute cholecystitis out of my mind. I decided to phone an experienced medical practitioner, and he arranged to visit me that afternoon. As I rested in bed the epigastric pain became easier and I dozed a little on and off, and did not even once feel if the abnormal swelling had changed. The physician arrived later in the afternoon, examined the abdomen very carefully, but found no abnormal swelling in the gallbladder area, nor anywhere else in the abdomen. This greatly surprised me, but I was able to confirm that the swelling I had felt had now completely disappeared. Both pain and swelling having now gone it was agreed that there was no need for any special line of treatment.

I remained comfortable and free from pain for four to five hours. But about 9
p.m. I began to have pain in the right hypochondrium and tenderness in the same area. The pain increased in severity, so I phoned the physician, told him of the change, and asked him to pay me another visit on the morrow. The second visit was paid on 4 April. When the physician saw the position of the pain and tenderness, and found I had a slight rise of temperature, he promptly called into consultation a distinguished surgeon, who confirmed the diagnosis of acute cholecystitis and advised my immediate removal to hospital. When I arrived there the diagnosis was again confirmed by a surgeon of vast experience, and he advised immediate operation. This took place that same evening, and a diseased gallbladder, already necrotic in parts, and containing 15 pigment stones, was successfully removed. Convalescence was uneventful, and I was discharged on 18 April.

COMMENT

The course of events was probably as follows:

Bacterial action first attacked the mucosa, which responded by a copious secretion of mucus that distended the viscus and caused the epigastric pain. The distension may have been due to the inability of the weak muscle fibres in the wall of the viscus to push the sticky mucus up through the narrow opening of the cystic duct, or maybe a small stone had temporarily stopped up the opening. In either case lying horizontally in bed permitted the mucus to escape from the gallbladder, relieved the distension, and eased the epigastric pain. During the four to five hours of freedom from pain bacterial action slowly penetrated the whole thickness of the wall of the viscus, and thereupon irritation of the parietal peritoneum led to pain and tenderness in the right hypochondrium. The next stage would have been perforation of the wall of the gallbladder and diffuse peritonitis.

This experience taught me with regard to acute cholecystitis (1) that the first symptom may be epigastric pain, (2) that the first sign may be a distended but not tender gallbladder, (3) that for one reason or another the gallbladder may then be able to expel its contents and both swelling and pain may disappear, (4) that a period of four or five hours may then ensue in which the patient is free of symptoms, and (5) that for the first time pain may now be felt in the right hypochondrium and lead to tenderness on gentle deep palpation, and so to suspicion of the true diagnosis. This sequence is important and is similar to that pointed out many years ago by J. B. Murphy in cases of acute appendicitis. I do not know whether the nocturnal bouts of sweating and rise in pulse rate had any connexion with the presence of gallstones, but it may be of significance that since the operation a year ago I have not had a similar attack. Finally, I learned the truth of the saying that "one is never too old to learn."

BILIARY COLIC

CHOLANGITIS

It may be difficult or impossible to distinguish cholangitis secondary to a common duct stone from acute cholecystitis. In general, the patient with cholangitis tends to have a greater constitutional reaction, with higher fever and often rigors but with lesser degrees of local tenderness than the individual with acute cholecystitis. The degree of hyperbilirubinemia tends to be lower in patients with acute cholecystitis.

INFLAMED OR LEAKING DUODENAL ULCER

In the case of a duodenal ulcer that is threatening to perforate or has even leaked slightly, the local findings may be similar to those of cholecystitis with local peritonitis, but a careful inquiry into the history will distinguish the two conditions. The pain that comes on about two and a half hours after meals and is relieved by taking food, the bringing up of "waterbrash" and acid eruptions, the attacks of flatulence, and possibly the occurrence of melena may give a clear picture of ulcer. If time permits, the diagnosis of ulcer may be confirmed by X-ray or endoscopy.

FREE RETROPERITONEAL PERFORATION OF DUODENAL ULCER

Retroperitoneal perforation of a duodenal ulcer may be attended by severe collapse at the onset, but the condition quickly localizes and leads to tenderness and swelling in the right loin. The perinephric tissues become edematous, and there may be frequency of micturition and even hematuria from the irritation of the renal pelvis. There is great pain on pressure at the erector-costal angle. The diagnosis is difficult, since a primary renal condition is likely to be suspected. It is, fortunately, a very rare condition.

BILIARY COLIC

Biliary colic (unassociated with inflammation of the gallbladder) is usually an antecedent of acute cholecystitis. The abdominal wall over the
gallbladder is soft and yielding, though there may be local deep tenderness. The pain is usually radiating, being felt especially in the right subscapular area or in the midline over the lower thoracic spine. It may also be felt on the left side, in which case there may be a complaint of a sense of constriction around the waist. This feeling of constriction, when present, is very characteristic of biliary colic. A subnormal temperature is more common than fever.

RUPTURE OF THE GALLBLADDER

Although rupture of the gallbladder is not infrequent, it is quite uncommon for generalized peritonitis to occur, because the perforation is usually very well confined in the form of a pericholecystic abscess. Such cases are indistinguishable from an ordinary acute cholecystitis except that the attack by that time has usually been present and unrelenting for at least five or six days.

With free rupture of the gallbladder or of one of the bile ducts into the general peritoneal cavity, there are usually the history and symptoms suggestive of biliary colic or cholecystitis, with a gradual extension of the painful area downward until the whole abdomen is tender; distention of the intestines increases, and there is tenderness on rectal examination. Free fluid may sometimes be demonstrated. Sometimes, free rupture of the gallbladder results from gangrene of the organ caused by occlusion of branches of the celiac artery. Such patients may have a history of prior abdominal angina.

HEPATITIS

In hepatitis there is tenderness all over the liver, including the lateral aspect, as ascertained by pressure in the lower intercostal spaces laterally, as well as in the right hypochondrium. This sign serves for diagnosis except in those cases where hepatitis coexists with the cholecystitis. The onset of pain is rarely abrupt in hepatitis.

MYOCARDIAL INFARCTION

PLEURISY

In right basal pleuropneumonia or diaphragmatic pleurisy fever is usually higher (104°F or 105°F), there may be an initial rigor, the right hypochondriac tenderness is more superficial, by gradual coaxing the fingers may be pressed well into the subhepatic region, and there should be signs—at any rate, fine crepitations—at the base of the right lung and pleura. Pain on top of the right shoulder is much more likely to be met with in diaphragmatic pleurisy than in cholecystitis. The occurrence of hemoptysis in any doubtful case would suggest either pneumonia or a pulmonary infarct.

MYOCARDIAL INFARCTION OR ANGINA PECTORIS

One of the most frequent noncardiac conditions with which a patient is admitted to modern coronary care units is biliary colic or early acute cholecystitis. The midline position of some cases of biliary pain, especially when located high in the epigastrium, occasionally makes the distinction very difficult. If a true myocardial infarction evolves, the electrocardiogram and serum enzyme changes, when followed serially, serve to distinguish the conditions. We have on occasion been unable to distinguish repeated attacks of angina pectoris, particularly when they occurred postprandially, from episodes of biliary colic. Even a stress electrocardiogram may fail to make this distinction. Suffice it to say that during myocardial infarction the abdominal examination is usually normal, but an electrocardiogram must always be taken in a patient with suspected cholecystitis.