Dr. Arcot J. Chandrasekhar: We have two very interesting patients with acute pancreatitis to discuss. Both of them demonstrate the pleuropulmonary complications of this disease. Mr. Skorton, will you please present the findings in first patient?

Mr. David Skorton: A 49-year-old white man was hospitalized because of ten days of upper abdominal cramping pain, nausea, and vomiting following excessive ingestion of alcohol. He had mild epigastric tenderness and hypoactive bowel sounds, and his liver was 15 cm in height by percussion. Physical examination of the chest revealed poor expansion on the right side, with dullness to percussion, decreased vocal fremitus, and diminished breath sounds in the right hemithorax.

The initial hemoglobin level was 11 gm/100 ml, the hematocrit reading was 33 percent, and the white blood cell count (WBC) was 16,400/cu mm, with a shift to the left. Levels of blood urea nitrogen, blood glucose, and electrolytes were normal. The patient's chest x-ray film showed a right-sided pleural effusion (Fig 1). The fluid was yellow in color, with the following values: WBC, 2,600/cu mm; hematocrit, 1.0 percent; total protein, 4.3 gm/100 ml; albumin, 1.9 gm/100 ml; lactic dehydrogenase, 175 international units (IU)/ml; and

![Chest x-ray film on admission, showing right-sided pleural effusion and basal plate-like atelectasis (confirmed on lateral film; not shown) (case 1).](image-url)
amylase, 1,830 IU/ml. Simultaneous serum values were as follows: protein, 6.6 gm/100 ml; albumin, 2.5 gm/100 ml; lactic dehydrogenase, 185 IU/ml; and amylase, 680 IU/ml (normal range, 40 to 100 IU/ml). The urinary level of amylase was 2,000 IU/100 ml.

One week later, a second thoracocentesis was performed, and 400 ml of fluid was removed. The chest x-ray films after removal of this fluid showed some residual effusion and plate-like basal atelectasis. The serum level of amylase remained elevated at 320 IU/ml after four weeks. There was significant improvement with intravenous administration of fluids, gastric suction, and therapy with tetracycline. A barium study of the upper gastrointestinal tract did not reveal any evidence of a pancreatic pseudocyst.

Dr. Chandrasekhar: Thank you. The second patient had a more complicated and protracted course:

Dr. James M. McKenna: A 52-year-old white man with one previous episode of pancreatitis was hospitalized with constant upper abdominal pain of two weeks' duration. The pain was relieved by sitting forward. The patient had lower substernal pain on swallowing, dysphagia, and mild exertional dyspnea for one week. Left pleuritic chest pain was present on the day of admission. The patient had lost 5.4 kg (12 lb) in the previous two weeks and one month prior to admission developed a cough productive of small amounts of brown sputum. The patient was a habitual drinker of alcohol and also smoked two packages of cigarettes per day.

Physical examination revealed a middle-aged thin man in distress from abdominal pain. Blood pressure was 90/72 mm Hg, the pulse rate was 106 beats per minute, the respiratory rate was 36/min, and the temperature was 37.1°C (98.8°F). The patient's neck veins were distended to just above the clavicles, with the thorax elevated to 30°. The apical impulse was palpated 11 cm from the midline, and upon percussion, the left cardiac border was located 14 cm from the midline. A loud fourth heart sound was heard, accompanied by a three-component pericardial friction rub. At the base of the left lung, there was dullness to percussion, egophony, and bronchial breathing. Moist rales were noted at the right base. The abdomen was tense, with moderate direct tenderness in the left upper quadrant and epigastrium, but without rebound tenderness. No bowel sounds were heard.

Upon admission, laboratory values for the blood were as follows: hematocrit, 40 percent; WBC, 17,400/cu mm; amylase, 634 IU/ml; albumin, 3.4 gm/100 ml; calcium, 9.3 mg/100 ml; serum lipase, 8.0 units/ml (normal less than 1.5 units/ml); lactic dehydrogenase, 220 IU/ml; arterial oxygen pressure (PaO₂), 45 mm Hg; arterial carbon dioxide tension (PaCO₂), 30 mm Hg; and bicarbonate, 22 mEq/L.

The chest x-ray film (Fig 2) revealed bilateral pleural effusions. A left thoracocentesis was performed, yielding yellow pleural fluid with a WBC of 3,600/cu mm, with 90 percent lymphocytes and 10 percent neutrophils. An electrocardiogram showed elevation of the ST segments in leads 1, 2, aVL, V₅ and V₆ compatible with early acute epicardial injury or pericarditis, or both. A transient multifocal atrial tachycardia and a short run of atrial flutter with varying atrioventricular conduction were recorded soon after admission.

During the ensuing two weeks the abdominal pains persisted, along with elevated values for the serum concentration of amylase, lipase level, and WBC, and hypoalbuminemia. One month after admission, while still on nasogastric suction, the patient had an exacerbation of his abdominal pain. Tachypnea (initially at 36 breaths per minute and diminishing to 28 per minute) persisted for more than three weeks with a concomitant PaCO₂ of approximately 30 mm Hg. This hyperventilation was accompanied by bilateral basal rales and hypoxemia, both of which persisted for a month. The PaO₂ fell to as low as 54 mm Hg, despite inspired oxygen concentrations of 85 to 90 percent, and was associated with a generalized alveolar infiltrate (Fig 3). Serositis was evident from the persistent pericardial friction rub and recurrent left pleural effusion, which required four thoracocenteses of 500 to 600 ml on each occasion. Hemoptyis developed five days...
FIGURE 3. Chest x-ray film taken a few days after admission. There is generalized alveolar infiltrate (case 2). After admission and recurred intermittently for three to four weeks. Five weeks after admission, the basal rales cleared, with an increase in \( \text{PaO}_2 \) to 58 mm Hg while the patient was breathing room air. The ST-segment elevation disappeared after five days, but T-wave changes in leads 1, 2, aVL, and V\(_1\) to V\(_4\) were noted one month after admission.

Tests of pulmonary function were done when the patient’s condition was stable, seven weeks after admission, and follow-up tests were obtained at three and nine months (Table 1). The chest x-ray film obtained three months after the onset of the illness showed complete resolution of parenchymal and pleural findings.

Therapy consisted of nasogastric suction, intravenous administration of fluids with electrolytes, albumin, amino acids, and glucose, and supplemental oxygen.

Dr. Chandrasekhar: The pleuropulmonary complications of acute pancreatitis can be classified into four categories, as follows:

1. Pleural effusion
2. Diffuse pulmonary injury (adult respiratory distress syndrome)
3. Pancreatic pseudocyst
4. Non-specific; secondary to acute abdominal process
   a. Basilar infiltrates; pneumonitis
   b. Aspiration abscess
   c. Atelectasis
e. Elevated diaphragm; immobile diaphragm
f. Pleural reaction

The first patient had pleural effusion and focal basal atelectasis; the second patient had diffuse pulmonary injury in addition to pleural effusion. Mr. Skorton will review the pleural manifestations of pancreatitis.

Mr. Skorton: Pleural effusion is reported to occur in 4 to 17 percent of patients with pancreatitis and is usually painless, without any pleuritic features. The effusion occurs three times more frequently on the left side than on the right, but occasionally it is bilateral. The effusion is often hemorrhagic. Concentrations of amylase are elevated in the pleural effusion and in the serum, but the concentrations in the pleural fluid are higher and remain elevated after the serum levels return to normal. Rarely, the pleural effusion may be the sole initial manifestation of pancreatitis. One may also have a sympathetic

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Observed</th>
<th>Predicted</th>
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<tbody>
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<td></td>
<td>11/73</td>
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<tr>
<td>Lung volumes</td>
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<td>Maximal voluntary ventilation, L/min</td>
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<td>Maximal midexpiratory flow rate, L/sec</td>
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<td>FEV(_1) as percent of forced expired volume</td>
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<td>57</td>
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<tr>
<td>Diffusion</td>
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<tr>
<td>Dsb, ml/min/mm Hg</td>
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<td>15</td>
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CHEST, 71: 2, FEBRUARY, 1977

PLEUROPULMONARY COMPLICATIONS OF PANCREATITIS 199
pleural effusion with low fluid levels of amylase as a nonspecific sequela of subphrenic inflammation.

Three theories regarding the pathogenesis of pleural effusion in pancreatitis have been reviewed by Kaye. The first and simplest theory is based on the transport of amylase-rich fluid from the blood across the capillaries that supply the pleural space. The high concentration of amylase in the serum produces a concentration gradient, along which the amylase-rich fluid diffuses. This theory fails to explain how an effusion initially develops and how a higher concentration of amylase develops in the pleural fluid. On the other hand, if the capillaries supplying areas adjacent to the pancreatic inflammation (i.e., the diaphragm) are more permeable than usual, then fluid rich in enzymes could leak into the pleural space along a concentration gradient. Early in the course of pancreatitis, equal concentrations of amylase could exist in the serum and pleural space, but a diminished absorptive capacity of inflamed pleural membranes would result in delayed clearance of amylase from the pleural compartment, leading to subsequent higher levels in the pleural fluid.

A second theory is based upon an assumption that enzyme-rich ascitic fluid reaches the thoracic cavity by way of the esophageal hiatus or other normal anatomic communications; however, the esophageal hiatus actually communicates with the mediastinal or subpleural space, and not with the pleural space proper. Penetration of enzyme-rich fluid into the pleural space would require increased permeability or actual digestion of the pleural membranes. Direct perforation or formation of an actual fistula between the abdominal and thoracic cavities can occur. Although not frequently invoked as an explanation for the pleural effusion, cases have been reported in which such a communication was demonstrable at necropsy. Burrowing pancreatic pseudocysts could follow either of these two anatomic courses and leak directly into the pleural space. The final theory is based on the existence of a lymphatic plexus in and around the diaphragm that connects the abdominal cavity with the mediastinum and subpleural space. When there is an intrabdominal inflammatory process, such as pancreatitis, the lymphatic plexus transfers the enzyme-rich fluid to the mediastinal and subpleural areas. This fluid then leaks into the pleural space because of increased permeability. The theory of the lymphatic plexus, therefore, explains the relative predominance of left-sided effusion, although in our first case the effusion was purely right-sided. Dissecting pancreatic pseudocyst or aberrant location of pancreatic tissue to the right of the midline could explain this phenomenon. We had no evidence for pancreatic pseudocyst in this instance. Since the bulk of the pancreas is on the left side, most of the lymphatic plexus is also on the left. This would favor a left-sided pleural effusion.

Dr. Chandrasekhar: Thank you, Mr. Skorton. Dr. McKenna, would you please comment on the diffuse pulmonary damage that can occur in acute pancreatitis and bring us up to date on current thoughts on the pathogenesis of such pulmonary injury.

Dr. McKenna: The diffuse pulmonary disease commences from two to seven days following the onset of symptoms of pancreatitis and occurs primarily in the more severely ill patient with persistent hypocalcemia. There is an increase in temperature, often with a further escalation of the serum level of amylase. Initially, the patients are tachypneic and later progress to develop profound dyspnea, cyanosis, and hypoxemia, with a PaO₂ of 44 to 45 mm Hg. Alveolar ventilation is increased, resulting in reduced values for PaCO₂ except terminally, when the PaCO₂ rises. The x-ray film shows a bilateral, generalized alveolar pattern of pulmonary infiltration. These clinical and radiologic abnormalities persist to a varying degree for one to four weeks. This diffuse pulmonary involvement, one of the causes of the adult respiratory distress syndrome, occurs in 18 percent of patients with acute pancreatitis and carries a mortality of 50 percent; however, both the severity and the frequency of pulmonary parenchymal malfunction may be underestimated. In a study of patients with an apparently normal subjective, clinical, and radiologic pulmonary status within 48 hours of the time of diagnosis of pancreatitis, the PaO₂ was less than 75 mm Hg in 23 of 40 cases. The pathologic changes in the lung vary from simple pulmonary edema to alveolar atelectasis with marked congestion and hemorrhage to interstitial pneumonitis.

Possible causes for the pulmonary changes in acute pancreatitis are listed in the following tabulation:

<table>
<thead>
<tr>
<th>Physical factors</th>
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<tbody>
<tr>
<td>Aspiration</td>
</tr>
<tr>
<td>Fluid overload and hypoproteinemia</td>
</tr>
<tr>
<td>Shock</td>
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<tr>
<td>Diffuse intravascular coagulation</td>
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<tr>
<td>Fat embolism</td>
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<tr>
<td>Pharmacologic factors</td>
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<tr>
<td>Phospholipase (lecithinase) A</td>
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<tr>
<td>Trypsin; elastase; chymotrypsin</td>
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<td>Kinins</td>
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<tr>
<td>Immunologic factors</td>
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<tr>
<td>Complement activation</td>
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<tr>
<td>Leukocyte aggregation</td>
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</table>

With respect to the physical factors causing pulmo-
nary changes in acute pancreatitis, the aspiration of gastric secretions may be related to vomiting, nasogastric intubation, and obtundation. The absence of vomiting and obtundation decreases the significance of this factor in our case. Fluid overload from overcorrection of the initial hypovolemic hemocen­trated state and from perioperative fluid replace­ment may be etiologic factors, particularly when aggravated by the hypoalbuminemia of pancreatitis. The effects of fluid overload may be exaggerated by a myocardial depressant factor and its potent negative inotropic effect, which is enhanced by hypocalcemia. Congestive heart failure may have been contributory in this patient, as he had an elevated central venous pressure, tachycardia, and prominent atrial and ventricular gallop rhythms after intravenous administration of fluids. A majority of the reported cases with diffuse pulmonary parenchymal damage due to pancreatitis were in shock. Although our patient was hypotensive on admission for an unknown period of time, he responded promptly to administration of fluids without untoward renal sequelae and probably did not have profound shock.

Severe pancreatitis is often complicated by diffuse intravascular coagulation, which can have pulmonary sequelae. In a series of 22 autopsies of patients with diffuse intravascular coagulation, pulmonary involvement was found in 50 percent and followed three different patterns: (1) fibrin thrombi obstruct­ng vessels of varying sizes; (2) formation of hyaline membranes; and (3) pulmonary hemorrhage syndrome. The latter was evident clinically as dyspnea of sudden onset, hemoptysis, rales, wheezes, pulmonary edema, and a homogenous pulmonary infiltrate. The clinical course of our patient resembled this third pattern, the pulmonary hemorrhage syndrome. Unfortunately, supporting studies of coagulation were not performed.

Fat embolism is a well-recognized cause of adult respiratory distress syndrome and may be associated with pancreatitis. There is peripheral digestion and stress mobilization of fat with increased free fatty acidemia. Peltier demonstrated experimentally the local toxicity of neutral fats and free fatty acids and suggested that hydrolysis of fat emboli by lipase-rich lungs contributes to the pulmonary damage. In our second patient the serum level of calcium and the hematocrit reading were decreased, and he was febrile, but these are nonspecific changes, especially in the absence of other evidence of fat embolism.

Let us now turn to pharmacologic factors causing pulmonary changes in acute pancreatitis, the most important one being phospholipase A. The concentration of this pancreatic enzyme is elevated tenfold in acute pancreatitis, paralleling elevations in serum concentrations of amylase and lipase.

Phospholipase A is a lecithinase and splits one fatty acid of the phospholipid, lecithin, the principal constituent of surfactant, to form lyssolecithin. When administered intravenously, phospholipase localizes predominantly in the lungs. If phospholipase A is infused intravenously, it produces tachypnea, hypoxia, hypercap­nia, and a decrease in the activity of pulmonary surfactant. Lysoproteins, including oleic acid, have independent deleterious effects. Oleic acid is the prototype free fatty acid used to produce experimental fat embolism. Lyssolecithin disrupts the phospholipid layer of membranes, resulting in hemolysis. Trypsin, elastase, and phospholipase A cause hemolysis and, thus, are possible etiologic agents responsible for the diffuse intravascular coagulation of pancreatitis. Acidosis and shock are additional potential causes of diffuse interstitial coagulation.

Release of histamine is another mechanism of pulmonary injury by phospholipase A and also by trypsin. The persistent elevation of the serum concentration of amylase in this patient indicates a concurrently high level of phospholipase A, a likely major mediator of his diffuse pulmonary disease.

The kinins increase vascular permeability and cause hypotension. Elevated blood concentrations of pancreatic kallikrein or trypsin or both occur in pancreatitis, and both are capable of generating kinins from various kininogens in blood. Although the major portion of the kinins are removed from the circulating blood by the lungs, the kinins cause pulmonary vasoconstriction and even spasms of the pulmonary veins. This venous hypertension may aggravate loss of fluid into the lungs. Kinins may act indirectly by causing release of histamine, and conversely, histamine may activate the kallikrein system, so that multiple causes for an increase in vascular permeability are at work simultaneously. The bronchospastic action of the kinins may adversely affect pulmonary function further, by causing ventilation-perfusion abnormalities, and fluid dynamics may be affected by release of vasopressin (antidiuretic hormone) by kinins.

Finally, let us consider the immunologic factors causing pulmonary changes in acute pancreatitis. Lepow claims that trypsin can cleave the C3 component of complement to produce its activated form and can serve as a starting point for the latter part of the complement cascade. Complement products, C3a and C5a, the anaphylatoxins, are highly active in causing increased permeability apart from their leukotactic and other effects. One final possible mechanism contributing to the diffuse pulmonary changes in acute pancreatitis is the liberation of complement products.
involvement of pancreatitis is pulmonary edema due to leukoagglutinin reaction of white blood cells in transusions of whole blood, a form of replacement commonly employed in the therapy for severe pancreatitis.

In summary, the hitherto underrecognized diffuse pulmonary involvement, a form of the adult respiratory distress syndrome, occurs with significant frequency in acute pancreatitis. Possible causes for this diffuse pulmonary damage are (1) the ever-present risk of aspiration, especially with a nasogastric tube in place; (2) fluid overload, compounded by the negative inotropic effect of myocardial depressant factor; (3) hypotension, which is common in severe pancreatitis; (4) embolization with fat, or thrombi from diffuse intravascular coagulation; (5) the indirect effects of altered pulmonary capillary permeability induced by histamine, kinins, and complement anaphylatoxins; and (6) leukoagglutinin reactions to transusions with whole blood, which are often needed. Fluid overload and the effects of phospholipase A would appear to be the primary agents in our second patient, although diffuse intravascular coagulation and aspiration cannot be ignored.

**Dr. Chandrasekhar:** Dr. Cugell, will you comment on the pulmonary function data and blood gas levels of the second patient?

**Dr. David W. Cugell:** During the acute phase of the patient’s illness, arterial blood gas levels showed a persistent reduction in PaO₂, despite the hyperventilation that is apparent from the reduced PaCO₂. That the cause for the low PaO₂ was a mismatch in the distribution of air and blood through the lung was corroborated later when the PaO₂ was only 54 mm Hg, despite inspired oxygen concentrations between 80 and 90 percent. Both the clinical course and the inability to maintain an adequate PaO₂, despite high inspired oxygen concentrations, are characteristic of the adult respiratory distress syndrome. Some weeks later, when the patient was much improved but still hospitalized, studies of pulmonary function showed a substantial reduction in lung volume compartments, modest impairment of expiratory air flow (as determined by several tests), a significant reduction in retractive force at total lung capacity (TLC), and normal values for pulmonary compliance over the normal range for tidal volume. The single-breath carbon monoxide diffusing capacity (Dsb) was reduced out of proportion to the decrease in lung volume. Subsequent studies at three and nine months following the acute illness demonstrated some improvement in lung volumes but no improvement in expiratory velocity or in Dsb. Whether or not the impairment in diffusion is permanent is still not certain. In other types of reversible diffuse parenchymal disease, the diffusing capacity may remain reduced for as long as a year, with subsequent improvement, despite a normal-appearing chest x-ray film.

**Dr. Chandrasekhar:** Thank you. The fourth category of pulmonary complications of pancreatitis (including basilar infiltrates, atelectasis, elevated diaphragm, nonspecific pleural reaction, etc) are extremely common in pancreatitis. These are nonspecific and hardly unique to pancreatitis and may be observed with the acute abdomen, regardless of the cause. Similar complications occur with splenic abscesses, but with greater frequency. In pancreatitis the elevated diaphragm may be secondary to localized peritonitis and transitory paresis, to atelectasis secondary to hyperventilation because of pain, or to aspiration following vomiting and the presence of a nasogastric tube. Plate-like atelectasis at the right base, with subsequent clearing, was noted in the first patient. Pulmonary embolism has to be carefully evaluated because of the increased incidence in pancreatitis. Although pneumonitis in patients with pancreatitis is usually secondary to aspiration, there have been cases when an inflammatory pancreatic pseudocyst ruptured through the diaphragm and established a pancreatic bronchopleural fistula. When this happens, the pneumonitis is secondary to pancreatic enzymes and subsides only after drainage of the pseudocyst.

Now let us turn our attention away from the respiratory system and consider the development of the pericardial rub in the second patient. Dr. Craig will comment on this aspect of the second case.

**Dr. Robert Craig:** Recent reviews of acute pancreatitis make little or no mention of pericardial or cardiac complications. Pericardial effusion is found infrequently at autopsy, and only rarely has it been described during clinical episodes of acute pancreatitis. When present, pericardial effusions are usually associated with pancreatic ascites or hemorrhagic pleural effusion; and pericardial fat necrosis has been implicated, similar to the peripheral fat necrosis in acute pancreatitis, but a normal pericardial biopsy in one patient with acute pancreatitis and sudden pericardial tamponade refutes that view. In our patient a peritoneal-to-pericardial fistula was probably present. Direct transport of peritoneal fluid to the pericardial space via the lymphatic vessels, as we mentioned previously in connection with transport from the peritoneum to the pleura, seems the most likely route.

Although not a feature of the present case, elec-
trocadiographic abnormalities indistinguishable from acute myocardial infarction occur in patients with acute pancreatitis, despite normal coronary arteries seen at postmortem examination or visualized by selective coronary angiograms. The pathophysiology of the electrocardiographic disturbances is probably related to circulating proteolytic enzymes producing sublethal myocardial damage with a resultant potassium leak. This is supported by the finding that necrosis of cardiac and skeletal muscles can be induced experimentally by the intravenous infusion of proteolytic enzymes. Electrocardiographic abnormalities of depolarization occur in dogs with experimentally produced pancreatitis. Arrhythmias have also been associated with pericardial involvement of pancreatitis, and this was underscored by the supraventricular tachyarrhythmia in our patient.

**Audience Question:** Is an elevated concentration of amylase in the pleural fluid pathognomonc of pancreatitis? Aren't you disturbed by the prolonged elevation of the serum concentration of amylase?

**Dr. Chandrasekhar:** Elevated serum concentrations of amylase can be seen in a variety of conditions, but the concentration of amylase in the pleural fluid is elevated only in pancreatitis, ruptured esophagus, and occasionally in malignant pleural effusions due to bronchogenic and pancreatic carcinoma. In a ruptured esophagus the effusion is always on the left side, the fluid tends to be purulent, and the elevated concentration of amylase is of salivary origin, due to leakage of swallowed saliva into the pleural space. In bronchogenic carcinoma the elevation is secondary to the salivary type of amylase.

The elevated serum concentration of amylase declines rapidly usually within a few days. The molecular weight of amylase is small (45,000), and the renal clearance is rapid. Prolonged elevations make one suspect a pancreatic pseudocyst. We searched for this by examination of the upper gastrointestinal tract in the first case, but no pancreatic pseudocyst was found. The other likely cause for a prolonged elevation of the concentration of amylase is a malignant neoplasm. Because the pleural effusion was on the right side, with no evidence for a pancreatic pseudocyst, and because the serum concentration of amylase was elevated for a long time, we obtained acrylamide gel electrophoresis of the amylase to determine which type of amylase was present in the serum. In both patients the amylase in the serum was of pancreatic origin. Subsequent followup of both cases showed no evidence for malignant neoplasm, and the clinical course was consistent with pancreatitis.

**Audience Question:** Is there any specific therapy for pulmonary complications of pancreatitis?

**Dr. Chandrasekhar:** Therapy for pulmonary complications is mainly supportive and symptomatic. The pleural effusion often responds to evacuation by pleural tap. If fluid is persistent and reaccumulates rapidly, one has to look vigilantly for a pancreatic pseudocyst which, if present, must be drained surgically. The diffuse pulmonary injury is managed as any other patient with adult respiratory distress syndrome; however, it is important to recognize this serious complication early, so that appropriate therapeutic measures can be taken. When there are factors that usually portend adult respiratory distress syndrome, such as severe shock, markedly depressed calcium levels, and persistently high levels of amylase, one should monitor the pulmonary status and blood gas levels closely. The nonspecific pulmonary complications, such as basal atelectasis, are best handled by good pulmonary physiotherapy to promote proper clearance of secretions. The hypoxia that follows pulmonary complications should be corrected by appropriate increments of inspired oxygen concentrations.

In summary, pulmonary complications are relatively frequent in patients with pancreatitis, and close attention should be paid to the pulmonary status of these patients. Complications secondary to acute pancreatitis occur as a result of the extravasation of pancreatic secretions into the abdominal and thoracic cavities. These include pancreatic pseudocyst, pleural effusion, and diffuse pulmonary injury. Nonspecific pulmonary effects include atelectasis, aspiration, and an elevated diaphragm. The spectrum of pulmonary complications is illustrated by the pleural effusion and plate-like atelectasis in the first patient and by the pleural effusion with diffuse pulmonary injury in the second patient. Pericarditis was an unusual additional complication in the second case.

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**Pleuropulmonary Complications of Pancreatitis 203**


AMERICAN COLLEGE OF CHEST PHYSICIANS
Presents
ALFRED A. RICHMAN ESSAY CONTEST
and the
ALFRED A. RICHMAN RESEARCH CONTEST

HISTORY
The Alfred A. Richman Contest was established by the Board of Regents of the American College of Chest Physicians in 1949. Since the first contest in 1950, over 700 medical students from 47 countries have submitted manuscripts.

In 1966, Alfred A. Richman, M.D., a Fellow of the College, established a fund to underwrite the contest. In recognition of his interest in undergraduate medical education and his generosity in establishing the fund, the Board of Regents voted to ascribe his name to the contest.

PURPOSE
To encourage and stimulate undergraduate medical students to explore and investigate problems relating to the disciplines of respiration and circulation.

INDIVIDUAL RECOGNITION
Cash awards will be presented to the undergraduate medical students selected by the judges for the best essay and the best original research paper on cardiovascular and pulmonary diseases.

$1,000 AWARDED TO THE BEST ESSAY
AND
$1,000 AWARDED TO THE BEST RESEARCH PAPER

Each winner will also receive a certificate of merit. Presentation of awards will be made at the 43rd Annual Medical College Recognition

The College of Medicine attended by each winner will receive a trophy inscribed with the name of the winner and the medical college. The trophy marks the college's high achievement in motivating and stimulating scientific inquiry in the realm of circulation and respiration.


IMPORTANT: All entries must be postmarked before midnight, May 31, 1977. For full details, please write:

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