However, immediately after an operation, the surgeon, the cardiologist, and the patient would like to prognosticate regarding the long-term benefits of the procedure. In addition, if a less than optimal procedure has been performed, there is always the possibility of additional surgery to correct the problem. For these reasons, a minimal number of investigative studies are justifiable.

For diagnosing localized cardiac injury (infarction) following surgery, no procedure has been shown to be superior to the electrocardiogram. Certainly no procedure is safer or less expensive. It is therefore most unlikely that any technique will replace this one as a basic method for the recognition of myocardial infarction. Additionally, the stress ECG allows early assessment of improvement in myocardial perfusion.

The myocardial-specific enzymes (such as the MB isoenzyme of creatine phosphokinase) provide a specific indication of myocardial necrosis. Thus, if the concentrations of these enzymes are elevated following surgery and ECGs suggest an infarction, a diagnosis of perioperative infarction is reasonably established. If the concentrations of the myocardial-specific enzymes are elevated in the absence of electrocardiographic evidence of infarction, then one must assume that either a nontransmural localized or a global cardiac injury has occurred. The severity of this local or global injury can be appreciated from the clinical status of the patient (presence or absence of congestive heart failure) and the degree of elevation of the concentrations of myocardial specific enzymes.

It would therefore seem that clinical observation, the ECG, and the levels of the myocardial-specific enzymes are adequate to diagnose significant myocardial damage in most situations. If stress testing is added, the ECG also gives early indication as to the success or failure of the operation in terms of correction of deficits in perfusion. To date, no new procedure, including scanning for the myocardial infarct, has been shown to add significantly to this basic armamentarium; however, scanning of the infarct may provide useful information when the electrocardiographic findings are unclear. For that reason, there is probably a place for these scans, not as routine postoperative procedures, but as additional studies when the clinical, electrocardiographic, and enzymatic findings are equivocal. This would be analogous to the manner in which postoperative coronary arteriograms have been utilized in the past.

All diagnostic procedures that are performed add significantly to the cost of the perioperative care. In these times when such strong demands are being placed on the medical profession to lower the cost of medical care, it would seem prudent to discourage the routine use of any study that is not of definite benefit. Therefore, it would appear that the basic test of clinical observation, the ECG, and the determination of the levels of myocardial-specific enzymes are optimal now (and likely to remain so for the foreseeable future) for the diagnosis of success in surgery for myocardial revascularization.

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Empyema or Abscess?  
Is Ultrasound a Diagnostic Aid?

The differentiation of pleural empyema from intrapulmonary abscess is of therapeutic importance. In both cases, treatment consists of antibiotics and drainage. In the former, drainage is usually established with chest tube thoracostomy and in the latter with postural maneuvers. The mistaken insertion of a chest tube into an intraparenchymal abscess carries the risk of empyema, pneumothorax, bronchopleural fistula, and hemorrhage. Such complications do not invariably develop, especially if the abscess is surrounded by significant pleural and parenchymal fibrosis; however, in most circumstances, it is generally recommended that a chest tube not be inserted into a pulmonary abscess.

Using standard roentgenographic techniques, it is frequently very difficult to differentiate a pulmonary abscess in close proximity to the pleura from a pleural empyema. The presence of an air-fluid level does not help, since a loculated empyema might contain air due to gas-forming organisms, a loculated pneumothorax, a loculated bronchopleural fistula, or introduction of air through the wall of the chest (eg, thoracocentesis). Friedman and Hellekant have described roentgenographic characteristics which will aid in this differential diagnosis. In addition, bronchography, sinography, and tomography are other radiologic procedures that are sometimes required. In this issue of Chest (see page 330), Adams and Kolodny have suggested another tool that might also be useful in this difficult diagnosis.
Up until now, the application of ultrasound to pleural and parenchymal pulmonary diseases has consisted primarily of the identification and localization of pleural fluid. Adams and Kolodny have now demonstrated that an intrapulmonary collection of fluid might also be identified as an echo-free space. In addition, the intraparenchymal fluid can be differentiated from pleural fluid by two characteristics. The first, and most reliable, is the symmetric motion of the proximal and distal interfaces of the echo-free space during tidal breathing and hyperventilation. This characteristic was present in five of eight cases of intrapulmonary fluid and was absent in 12 of 12 cases of pleural fluid. If would be important to know how many of the 12 cases of pleural fluid represented freely moving transudates and whether the same criteria will apply to loculated pleural exudates with significant peripleuroparenchymal fibrosis. It is this latter situation which is most difficult to differentiate from a juxtapleural intrapulmonary abscess and in which ultrasonic false-positive results might be expected.

The second criterion is less reliable. It consists of the presence of discontinuous echoes at least 2 cm in depth, representing overlying lung proximal to the fluid. It was absent in three of eight cases of pulmonary fluid (false-negative) and present in two of 12 cases of pleural fluid (false-positive).

Both criteria require the application of subjective assessment. The proximal and distal interfaces of intrapulmonary fluid might not be perfectly symmetric with hyperventilation. The allowable amount of asymmetry before placing the fluid in the pleural cavity might vary among observers and depend upon experience and interpretation. The judgment that certain echoes proximal to the fluid are discontinuous, rather than continuous, requires a degree of subjective assessment and might also be influenced by technical factors.

The article by Adams and Kolodny implies that the mere presence of an echo-free space does not necessarily localize fluid to the pleural cavity. The characteristics of motion of the interfaces, along with other clinical and radiographic criteria, must be evaluated prior to making that assumption, with its attendant therapeutic implications.

The article by Adams and Kolodny extends the use of ultrasound beyond the pleural space. It provides an additional noninvasive tool which might help with difficult therapeutic decisions. It should stimulate further investigation into the usefulness of ultrasound in differentiating pleural from pulmonary densities.

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References


Pulmonary Infiltrates in Acute Leukemia

Since acute leukemia is a disease of the circulating blood cells, it is not surprising to find multiple organs of the body infiltrated by leukemic cells. Prior to the introduction of effective chemotherapy, leukemic infiltrations were observed in many organs at postmortem examination. However, these infiltrates seldom interfered with normal organ function and, thus, were of little clinical significance. For example, in an autopsy series of 420 patients with acute leukemia, leukemic infiltrates were identified in the hearts of 156, yet only 20 of these patients had clinical symptoms. In a prospective autopsy study of the lung in 50 consecutive patients with acute leukemia, 66 percent were found to have leukemic infiltration, but only two patients had symptoms which could be associated with these infiltrates.

Pulmonary leukemic cell infiltration may be alveolar, peribronchial, perivascular or subpleural. The degree of pulmonary infiltration correlates with the level of circulating leukemic cells in the peripheral blood. Leukostasis in pulmonary vessels has been observed, usually in patients with leukocyte counts of greater than 100,000/cu mm. These lesions are similar to those arising in the brain in patients with high leukocyte counts, a complication which often terminates fatally. Patients with extensive pulmonary infiltrates, such as those reported in this issue by Prakash, Divertie, and Banks (see page 345) have been reported sporadically. In these patients, the pulmonary complication predominates and is manifested by dyspnea, cyanosis, hypoxemia and abnormalities of chest roentgenograms. Usually, the diagnosis of acute leukemia is established easily because the patient has a high leukocyte count with circulating abnormal cells. However, a few patients have presented with leukemic pleural effusions and normal peripheral blood.

In the past, patients with symptomatic leukemic infiltrates in the lung usually died of respiratory failure. Antileukemic agents available at that time were not able to cause rapid destruction of leukemic cells and reverse this process quickly. Both arabinosyl cytosine and the anthracycline antibiotics (daunorubicin, Adriamycin) promptly destroy leukemic cells and, thus, the respiratory failure can be reversed.

Prakash and colleagues have emphasized correct-