arousal threshold, or better alpha power threshold and sleep fragmentation.

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REFERENCES

Transudate vs Exudate: Genug!

In 1972, Light et al developed criteria for the diagnostic separation of transudates and exudates. The authors developed three criteria, with a pleural effusion categorized as exudative if it met one or more of them: (1) a pleural fluid:serum protein ratio >0.5; (2) a pleural fluid:serum lactic dehydrogenase (LDH) ratio >0.6; or (3) a pleural fluid LDH >200. The upper limits of normal for serum LDH in their laboratory was 300, so a pleural fluid LDH >200 was also greater than two thirds the upper limit of normal for the laboratory.

In this issue of CHEST (see page 1503), Vives and colleagues report on the pleural fluid characteristics of 195 patients with pleural effusion of known etiology. The purpose of their study was to compare the accuracy of the criteria of Light and colleagues for categorizing a pleural effusion as an exudate with several alternative criteria. The authors come to the conclusion that the criteria of Light et al remain the most clinically useful and accurate diagnostic standard available.

The study by Vives et al in this issue of CHEST is one in a long series of attempts to improve upon the criteria of Light and coworkers. Different authors have looked at the pleural fluid cholesterol and pleural fluid:serum cholesterol ratio,2,4 pleural fluid:serum bilirubin ratio,5 serum-pleural fluid albumin gradient,6 and different manipulations of pleural fluid protein and LDH levels.4 When the dust has settled, it appears that none have been superior to the criteria of Light et al.

The fact that the criteria of Light et al have held up so well is not surprising. There are several reasons why they have stood the test of time. First, the criteria were developed and then modified by looking at diagnostic variables in a group of 150 patients with pleural effusions in whom the etiology of the effusion had been definitively determined. Second, protein and LDH each has separate physiologic import. The pleural protein concentration reflects increased “leakage” into the pleural space or decreased clearance, and the pleural fluid LDH reflects pleural inflammation. Third, the criteria as used by Vives and colleagues (using an LDH level greater than two thirds the upper limit of normal rather than an absolute pleural fluid LDH level >200) avoid all absolute values. As pointed out by the authors, this use of ratios eliminates much of the between-lab variability that would otherwise occur; a lab whose protein determinations run high and a lab whose protein determinations run low will each have different absolute values for all determinations, but both will have almost identical pleural fluid:serum protein ratios. Finally, when one looks at sensitivity and specificity, the criteria of Light et al maximize sensitivity over specificity in the diagnosis of an exudative effusion. This is consistent with the concept that it is better to occasionally misdiagnose a transudate as an exudate than it is to mistakenly characterize an exudate as a transudate.

Only one important sophistication has been added to the criteria of Light et al over the years. Following diuretics, the characteristics of a transudative effusion, particularly from congestive heart failure, may be altered enough for pleural fluid chemistries to shift
from transudative over into the “exudative” zone. Combining the criteria of Light et al with this knowledge of when a “transudate by etiology” can turn into “an exudate by chemistries” allows the clinician extremely high diagnostic acumen.

This study by Vives and coworkers demonstrates one of the values of a good negative study; it can tell us when “enough is enough.” Enough! It is time to accept the criteria of Light et al and to move on. It is time to stop focusing our efforts on the elusive goal of finding an ever more perfect technique of separating effusions into exudates and transudates, and instead, to concentrate our collective efforts on the frontiers which still exist in our understanding of the pleural space. Several areas beg for attention.

We need to find characteristics that are specific for different etiologies of exudative effusion. There is now a moderate amount of literature to support the use of adenosine deaminase (ADA) levels for the diagnosis of tuberculous effusion, although its use warrants further refinement. In fact, Vives and colleagues used ADA levels in their diagnostic criteria. Immunocytometry has been used to diagnose lymphoma in an otherwise idiopathic exudate. Doubtless, other etiology-specific chemistries or cell markers can and will be developed as diagnostic tools for exudative effusions.

We need to be able to better define the criteria for “complicated” parapneumonic effusion (a term which implies that the effusion should be drained with a chest tube to control infection or prevent fibrothorax). Even Light has changed his criteria over the years in an attempt to more accurately predict a need for chest tube insertion. A complicated parapneumonic effusion, whether it progresses to frank empyema or “just” localization, carries with it the potential for significant morbidity and cost. Because of the potentially serious consequences, we agree with the perspective that if in doubt, it is better to insert a chest tube too soon rather than too late. Nevertheless, chest tube insertion is not completely benign. It is an invasive, uncomfortable procedure which by its very nature increases morbidity, cost, and length of hospital stay. The issue of complicated parapneumonic effusion is complicated by the fact that there are certain considerations apart from pleural fluid chemistries which may affect the decision about pleural drainage. If imaging studies demonstrate that a parapneumonic effusion is becoming localized, chest tube insertion may be warranted regardless of fluid chemistries. Microbiology can affect the decision. One might insert a chest tube for a complicated parapneumonic effusion in a patient with an anaerobic or Gram-negative pneumonia but hold off on a patient with pneumococcal pneumonia and the same pleural fluid chemistries. The optimal approach to parapneumonic pleural effusion probably involves an intersection of etiology, chemistries, and imaging which has yet to be defined.

The rapidly increasing availability of pleuroscopy with conscious sedation for the evaluation and management of selected exudative effusions offers great opportunity for earlier, more definitive diagnosis and management but brings with it many questions. How many thoracenteses and pleural biopsies should we do before moving on to pleuroscopy? Can we consistently reproduce a sensitivity and specificity with this technique in which the percentages are in the high 90s as has been initially reported for previously undiagnosed effusion? If we can exclude tuberculosis or diagnose it biochemically, is it worth doing pleuroscopy to look for occult malignancy which by its very presence in the pleural space would mean that it is metastatic and incurable? Can we find other less invasive diagnostic techniques to detect malignancy when cytology is negative?

The criteria of Light et al for the separation of transudates from exudates based on readily available chemistries are an important milestone in our diagnostic approach to pleural effusion. The results reported by Vives and coworkers in this issue of CHEST reaffirm for us that it is now time to move on. The pleural space still has frontiers, but they are not where they were 24 years ago.

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REFERENCES


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Continuous ECG Monitoring for Ischemia in the ICU

In the last 2 decades, monitoring techniques in ICUs have evolved considerably but mainly in respiratory and hemodynamic measurements. Oximetry has become routine; capnography, mixed venous oximetry, and even continuous cardiac output measurements are widely used. Monitoring by electrocardiography (ECG) for ischemia, however, has not kept pace.

There are substantial reasons to be concerned. Of the 27 million patients undergoing surgery annually in the United States, as many as 1 million may develop a cardiac complication, at an estimated cost of $20 billion.

Clinical studies have validated this concern. For example, in one study of 474 men with coronary artery disease (CAD) or at high risk for it, continuous monitoring of two bipolar leads in the perioperative period of noncardiac surgery demonstrated a 41% incidence of myocardial ischemia postoperatively. The ischemia on ECG correlated with a 9.2-fold increase in ischemic events (cardiac death, myocardial infarction, unstable angina). Almost all of the postoperative ischemia in this study was silent and would not have been detected without the continuous two-lead ECG monitoring.

Studies in continuously monitored patients with peripheral vascular disease undergoing vascular surgery have shown a similar incidence of ischemia in patients at risk. In one study, 176 patients were monitored preoperatively from inferior and lateral leads; 32 had 75 episodes of ischemia. Seventy-three of the 75 episodes were asymptomatic. Of the 32 patients with preoperative ischemia, 12 had postoperative cardiac events (myocardial infarction, unstable angina, ischemic pulmonary edema). Only 1 of the 144 patients without preoperative ischemia had an untoward postoperative event, suggesting postoperative ischemia might be confined to high risk groups. Another study in patients having peripheral vascular surgery demonstrated an astonishing 63.5% incidence of silent perioperative ischemia. Nine of 200 patients suffered a myocardial infarction, and there were 2 cardiac deaths. Infarctions here too correlated with silent ischemia time detected by continuous ECG monitoring. These studies clearly demonstrate a high incidence of silent ischemia and concomitant adverse outcome in patients with known vascular disease or at high risk for it.

Ischemia in these patients postoperatively was presumably related to pain, fluid balance, fever, catecholamine levels, or other postoperative stresses. More recently, continuous ECG monitoring has been applied to a different kind of stress—weaning from mechanical ventilation. Hurford et al., originally using thallium scintigraphy, documented a high incidence of myocardial perfusion abnormalities occurring or worsening in patients rapidly weaned from positive pressure to spontaneous ventilation. This group then applied continuous two-bipolar-lead ECG monitoring to 17 patients weaning from mechanical ventilation in a mixed population ICU. Six of 17 patients had ECG evidence of ischemia during the 24-h study period; only two episodes were clearly related to discontinuation or changes of ventilatory support. Patients with ischemia did, however, fail to wean more commonly, reflecting either myocardial dysfunction or possibly a “sicker” cohort identified by their vascular disease.

In this issue of CHEST (see page 1577), Chatila and colleagues extend this work. As in the earlier study, a mixed population of 93 patients with a high incidence of coronary disease was studied. Continuous ECG monitoring in this study detected 6.4% incidence of ischemia during weaning in the group. As might be expected, five of the six patients with ischemia had known CAD, and again half of the ischemic episodes were asymptomatic. Four of the six failed initial weaning attempts, although it is by no means clear that the failure to wean was causally related to ischemia.

What can we make of all of this? First, silent ische-