Performing Thoracentesis

To the Editor:

I read with interest the article "Limited Utility of Chest Radiograph After Thoracentesis" by Petersen and Zimmerman in a recent issue of CHEST (April 2000). Although I agree that performing a routine chest radiograph following a thoracentesis is needless in most patients, I was surprised by the number of pneumothoraces that occurred during the performance of thoracentesis, because, in my hands, pneumothorax occurs very rarely (< 0.025%).

Upon review of the technique described in the article, I advise that during a thoracentesis procedure the tubing attached to the angiocatheter should be utilized in order to ascertain (using the tubing itself as a manometer) what the fluid pressure is in the chest. If the fluid pressure is 0 or even negative, then the removal of fluid will result almost certainly in a pneumothorax, or at worse shock, because one cannot have “a negative space.” However, if the fluid pressure is positive, it is very likely that the removal of fluid will not result in a pneumothorax. (In a patient who is borderline, I often recheck the pleural pressure of fluid intermittently during thoracentesis to make sure that I do not remove “too much.”) The patient at this point (when the fluid pressure is 0) usually complains of a dull chest pain, which is a reflection of a negative pressure in the chest.

I also was concerned about the authors’ disparaging comment about the use of vacuum bottles in performing thoracentesis. I have found that the use of vacuum bottles is an excellent adjunct to the performance of thoracentesis. I have observed that if there is foam at the top of the fluid, then this means that the vacuum is still present; when there is no foam or little foam, there is no vacuum present, which can be indicative of a pneumothorax. Moreover, if a significant pneumothorax is found, I recommend a needle thoracostomy as opposed to chest tube insertion because often the pneumothorax is, at best, transient, when it is caused by such a small needle, as opposed to one induced by trauma.

In closing, I wish to add that the evaluation of fremitus with the stethoscope is a much better test than listening to breath sounds in someone with a pneumothorax. The absence of fremitus is easier to ascertain, whereas the finding of “decreased breath sounds” is sometimes a very difficult physical finding to reproduce. Figures 1 and 2 illustrate a thoracentesis performed with the manometer technique.

William H. Fee, Jr., MD
Franklin, PA

Correspondence to: William H. Fee, Jr., MD, Physician’s Office Building, 150 Prospect Ave, Franklin, PA 16323.

REFERENCE


Clavicle Tapping and Auscultation as an Alternative to Chest Percussion When Performing Thoracentesis

To the Editor:

Thoracentesis is a common procedure in medical practice. Chest percussion, however, seems to have become a lost art among medical trainees. Unfortunately, chest percussion remains an important component of assessment when performing thoracentesis at the bedside, as it allows for the identification of the pleural fluid meniscus, and therefore, the procedure site. The importance of the accurate identification of the superior-most aspect of the pleural effusion when performing thoracentesis cannot be overstated as inaccurate assessment may needlessly increase the risk of procedural complication, particularly for pneumothorax. The response of many medical centers to the loss of this physical diagnosis skill by clinicians seems to be an increase in the use of ultrasonography and the relegation of thoracentesis to radiologists.

CORRESPONDENCE TO:
William H. Fee, Jr., MD
Physician’s Office Building, 150 Prospect Ave, Franklin, PA 16323.
In recent years, we have developed an alternative approach when teaching to our housestaff trainees thoracocentesis that does not rely on chest percussion, namely, clavicle tapping with posterior chest auscultation. Stated simply, this technique takes advantage of the sound transmission characteristics of the inflated lung and the loss of sound transmission caused by the interposition of a layer of fluid between the air-filled lung and the chest wall. A steady tapping of the clavicle anteriorly by the examiner, who is positioned posteriorly reaching over the ipsilateral shoulder, generates a repeating sound that is well-transmitted through the lung to the posterior chest wall and that is readily appreciated via a stethoscope. The stethoscope then is moved slowly inferiorly from the apex to the base with each appreciated tap to the level below which no further conducted sound can be appreciated. This place represents the level of the superior aspect of the pleural effusion.

In our experience, this technique correlates well with chest wall percussion performed by experienced examiners, is readily taught and learned, and has increased the comfort of our housestaff with thoracocentesis.

Robert S. Crausman, MD, MMS, FCCP
Amanda R. Crausman
Brown University School of Medicine
Providence, RI

Correspondence to: Robert Crausman, MD, MMS, FCCP, Director, Internal Medicine Residency Program, Memorial Hospital of Rhode Island, 111 Brewster St, Pawtucket, RI 02860

Alveolar Hemorrhage Associated With Antiphospholipid Syndrome

To the Editor:

Santos-Ocampo et al (October 2000) reviewed seven patients with systemic lupus erythematosus (SLE) admitted to the hospital with episodes of alveolar hemorrhage (AH). Recently, we published a case report of multiple organ dysfunction with acute respiratory failure due to AH associated with antiphospholipid antibodies in a 42-year-old woman with a medical history of antinuclear antibodies-negative SLE and antiphospholipid syndrome (APS).

The term antiphospholipid syndrome was introduced to describe patients presenting with combination of recurrent arterial and venous thrombosis, recurrent fetal loss, often accompanied by thrombocytopenia, and elevations of serum antiphospholipid antibodies, eg, lupus anticoagulant (LA) and anticardiolipin antibodies. APS may present as a primary disorder in patients without other autoimmune diseases, or as a secondary disorder in SLE and other autoimmune diseases. An analysis of 29 studies yielded an average frequency of 35% for LA and 44% for anticardiolipin antibodies in SLE patients. The reported frequency of APS in patients with SLE ranges from 20 to 35%. The pulmonary manifestations of APS include multiple pulmonary emboli, major pulmonary arterial thrombosis or microthrombosis with or without capillaritis, pulmonary hypertension, and AH. The presence of antiphospholipid antibodies is not usually associated with hemorheologic manifestations, and such an event occurring in a patient with the APS is usually due to deficiency of another coagulation factor, severe thrombocytopenia or the presence of severe uremia or hepatic disease. The pulmonary vasculature, however, because of unknown factors, may be susceptible to hemorrhage.

In the report of Santos-Ocampo et al, one patient had a medical history of cerebral vascular accident from APS. We are interested to know if all patients were tested for antiphospholipid antibodies, eg, LA and anticardiolipin antibodies or anti-β2-glycoprotein I antibodies, and AH could also be the result of APS in some of the patients.

Franz J. Wiedermann, MD
Wolfgang Lederer, MD
Wolfgang Schobersberger, MD
The Leopold-Franzens-University of Innsbruck
Innsbruck, Austria

Correspondence to: Franz J. Wiedermann, MD, Department of Anesthesia and Intensive Care Medicine, Division of General and Surgical Intensive Care Medicine, The Leopold-Franzens-University of Innsbruck, Anichstrasse 35, A-6020 Innsbruck, Austria; e-mail: Franz.Wiedermann@ublk.ac.at

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Lost in the Labyrinth of End Points

To the Editor:

We feel that the attack on our article in CHEST (October 2000) by Kass and Bartter in their editorial is unjustified on the basis of the data that have been presented. If they had taken care to read the “Materials and Methods” section carefully, they would have clearly seen that the primary outcome variable was the effect on bronchial hyperresponsiveness to adenosine monophosphate challenge, which is a suitable surrogate for mast cell-mediated airway inflammation. On the basis of this end point, there was significant superiority of monotherapy with budesonide over formoterol after 4 weeks, amounting to a 2.5-fold difference. A 1 doubling dilution shift (ie, twofold) in bronchial hyperresponsiveness is usually taken as being a clinically relevant effect. Likewise, there was also a significant difference when comparing these two therapies in the suppression of exhaled nitric oxide, which is another airway inflammatory surrogate. We did not show any difference between combination therapy and budesonide monotherapy for either of these surrogate inflammatory markers, suggesting that any improvements in control with combination therapy are simply due to bronchodilatory activity. For Kass and Bartter to concentrate their editorial on end points of lung function such as FEV1 and peak expiratory flow, which are insensitive in patients with mild-to-moderate asthma, and which clearly were not chosen as the a priori primary efficacy variable, is to miss the whole rationale of the study, even though we clearly stated the emphasis on the inflammatory markers in the title and in the introduction. Indeed, it has been shown previously that the dose response for inhaled budesonide is much steeper for effects on bronchial hyperresponsiveness than on lung function in patients with mild-to-moderate asthma. To properly evaluate the effects on lung function would require hundreds of patients with more severe asthma, as has already been done in pharmaceutically sponsored multicenter studies.

It was interesting that our patients preferred the treatment regimens that contained formoterol, perhaps suggesting that they had acquired a taste for its rapid effect on airway caliber. We believe...