We disagree with the statement that the presence of a pneumothorax precludes a second attempt at biopsy. In fact, the presence of a pneumothorax following the initial procedure often renders a poorly defined lesion much more visible at fluoroscopy.

An additional point not raised by the authors and which we feel is of value following a percutaneous lung biopsy is to place the hemithorax biopsied in a dependent position. This reduces both alveolar size, as well as the alveolar-to-pleural pressure gradient in the region surrounding a pneumothorax. This should decrease the rate of pneumothorax formation and permit sealing of the puncture site. We attempt to maintain our post-biopsy patients in this position as far as is possible for a period of approximately 12 hours following the procedure.

Eugene L. St. Louis, M.D.; Robert Hyland, M.D., F.C.C.P.; Robin R. Gray, M.D.; Michael Hutcheon, M.D., F.C.C.P.; Donald Jones, M.D., F.C.C.P.; and Harvey Grosman, M.D., The Wellesley Hospital, University of Toronto, Toronto, Ontario, Canada

REFERENCES

To the Editor:

We believe the comments by St. Louis and colleagues regarding percutaneous needle biopsy of lung lesions merit response. Anyone who has performed over 700 such procedures obviously has extensive experience that should be shared. Following completion of the study we recently reported in Chest, we have also begun to use a smaller bore 20 gauge Rotex needle with the anticipation of fewer pneumothoraces. We have yet to encounter implantation metastasis with any needle.

We perform over one-half of our procedures on an outpatient basis and therefore have found it necessary to formulate a practical plan to manage these patients. This includes a second needle pass when necessary, and forego the delayed chest roentgenogram. We do routinely follow-up these individuals and have found it distinctly rare for someone to develop a clinically symptomatic pneumothorax who does not either have one on the initial post-biopsy roentgenogram or clinical symptoms of one at the time of their release. We also believe there should be an increased incidence of pneumothorax with the second needle pass. Table 1 of our study shows this, but it is not a statistically significant observation.

We are pleased St. Louis et al agree with our premise that the depth of needle penetration is the single dominant variable in producing a pneumothorax. In our discussion, we postulate it may be the crossing of additional tissue planes in the deeper penetrations, but did not make an actual estimate of the pleural surfaces, as demonstrated by the figure of St. Louis et al.

One of our criteria for needle biopsy of the lung is a cooperative patient and we attempt to minimize the potential for involuntary motion during the procedure both by patient education to include breathing technique, and mild sedation as required. By use of a cardiac cradle, we control patient movement throughout the procedure. Therefore, we believe this is a factor that is controlled and is not a variable in our recent series.

We prefer not to re-attempt a biopsy in the presence of an obvious pneumothorax. While the lesion may be more clearly defined in a partially collapsed lung, we hesitate to add further to the already present morbidity of the situation and create the necessity of tube thoracotomy.

The observation of St. Louis et al of placing the biopsied hemithorax in a dependent position makes sense for the reasons he states, although it has not been objectively evaluated in humans. We incorporate this into our current technique whenever practical.

Robert H. Poe, M.D., F.C.C.P., Associate Professor of Medicine, and Michael C. Kalloy, M.D., Assistant Professor of Medicine, University of Rochester School of Medicine and Dentistry, Rochester, New York

To the Editor:

I cannot see how Figure 1 in Dr. Krongrad's article (Chest 1984; 85:107-13) illustrates a patient who "may eventually require treatment with a cardiac pacemaker" as "the cardiac rhythm decreases." There is nothing in the four strips shown to presage bradyarrhythmic problems: the junctional rhythm during sleep occurs frequently in normal young individuals, while the "marked sinus arrhythmia" is a simple misinterpretation of what appear to be junctional premature complexes.

In the context shown, I would only contemplate pacemaker therapy for the more likely possibility of intractable supraventricular tachycardia.

George Nikolic, M.D., Director, Intensive Care Unit, Woden Valley Hospital, Woden, Australia

REFERENCE

To the Editor:

Dr. Nikolic objects to the suggestion that patients with transposition of the great arteries who manifest junctional rhythms after the Mustard operation may eventually require pacemaker therapy. The objection is based on the premise that "junctional rhythm during sleep occurs frequently in normal young individuals." I would agree with Dr. Nikolic that junctional rhythms indeed occur frequently in young individuals, but I would like to alert physicians who take care of young patients following a Mustard operation that in the context of transposition of the great arteries such junctional rhythms are most often due to sinus node injury rather than a normal phenomenon.

Indeed, the tracing given in our Figure 1 is from a patient who eventually required pacemaker therapy when ambulatory electrocardiography revealed a heart rate of 14-19/min over extended periods of time during his adolescent years.

In general, our experience is that patients who manifest a sick sinus syndrome following a Mustard operation most often require pacemaker therapy for bradyarrhythmias rather than therapy for the bradycardia-tachycardia syndrome.

I believe Dr. Nikolic's letter is important because it re-emphasizes the issue of sick sinus syndrome in patients following the Mustard operation and allows to indicate on its very early manifestations.

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