Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Bronchoscopy In Hemoptysis

To the Editor:

We read with interest the review by Haponik et al summarizing clinicians’ perspectives on hemoptysis. However, we should like to take issue with his statement that our paper advocated the use of fiberoptic bronchoscopy without endotracheal intubation in patients with massive hemoptysis. On the contrary, three of our four patients underwent fiberoptic bronchoscopy through a large bore (number 8.5) endotracheal tube. We concluded that massive hemoptysis can be managed successfully utilizing the fiberoptic bronchoscope and with no morbidity, provided the airway is protected with an endotracheal tube. In fact, it appears that the majority (41 percent) of respondents favored this approach. A survey of pulmonologists and ENT surgeons in our community revealed that the majority favored the use of the flexible bronchoscope through a protected airway.

It is of interest that the initial submission of our manuscript to a thoracic surgical journal was rejected because, “Every one knows that massive hemoptysis should be managed with a rigid bronchoscope.” The paper by Haponik vindicates our position, although our data appears to have been misinterpreted by the authors. We should like to set the record straight.

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REFERENCES
1 Haponik EF, Chin R. Hemoptysis: clinicians perspectives. Chest 1990; 97:469

Effect of Converting Enzyme Inhibitors on Serum ACE Test

To the Editor:

Rodman and Lindenfeld recently reported a case of sarcoidosis-associated pulmonary hypertension successfully treated with corticosteroids. In the history, it is mentioned that the patient had been treated for congestive heart failure with angiotensin-converting enzyme inhibitor (type not mentioned). One year later, signs of sarcoidosis arose and a diagnostic workup was undertaken. As part of the workup, the angiotensin-converting enzyme (ACE) level was measured, and a normal level of 20 IU was obtained (normal 10 to 30). No further mention of converting enzyme medication is made.

It is now known that converting enzyme inhibitor medication does reduce serum ACE level to a degree that varies with the specific form of ACE inhibitor being administered. Enalapril causes extremely low (near zero) serum ACE levels that are unaffected by diluting the serum, by dialysis, or by storage of the serum sample. Captopril, on the other hand, causes inhibition of serum ACE that is gradually lost with storage of the serum sample (up to one month at 4°C), and may be totally reversed by an appropriate dilution of the serum or by dialysis with physiologic saline solution.

If the method of serum ACE assay employs an intrinsic dilution of serum greater than 1:8 and the patient is receiving captopril, the inhibitory effect of the medication will be circumvented. However, if the method resembles my modification of Cushman and Cheung’s assay, then captopril will reduce the serum ACE level unless the serum sample is diluted 1:8 prior to assay. The level obtained may otherwise fall within a “normal” range because of the loss of inhibition by captopril with storage.

Thus, the normal serum ACE level obtained by Rodman and Lindenfeld may not have been normal at all if the patient was receiving captopril and if the method of ACE assay was the one mentioned above, performed without pre-dilution of the serum.

A measure of serum ACE is extremely useful in the diagnostic workup of sarcoidosis and for following the clinical course of the disease. It is unfortunate that a valuable class of medication has the potential for interfering with the assay, but this potential interference is not insurmountable if the problem is recognized and appropriate corrective measures are taken in performing the assay. Unfortunately, if the patient is taking an enalapril-type medication and sarcoid disease is suspected, the only corrective measure is to discontinue treatment with enalapril and substitute captopril.

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REFERENCES

Arterial Oxygenation in Carbon Monoxide Poisoning

To the Editor:

I read with interest the report by Marius-Nunez describing a case of myocardial infarction due to carbon monoxide (CO) poisoning in a patient with normal coronary arteries. Myocardial ischemia