be generally advocated, however, it would be necessary to study larger groups of patients and controls to determine the sensitivity and specificity of the test. Ironically, the diagnostic ("gold") standard against which the test results would be weighed would be clinical evaluation with reproduction of symptoms with hyperventilation. In the final analysis, therefore, I believe that the diagnosis of HVS will continue to rely primarily on the clinician's high index of suspicion and willingness to subject patients suspected of having HVS to the simple procedure of forced hyperventilation in order to reproduce symptoms.

We agree completely with Dr Kummer's assertion that a psychogenic stimulus is the underlying force that triggers the response of hyperventilation. Our experience also indicates that correction of the overbreathing may be very difficult in a significant portion of these individuals, especially if the breathing pattern has been established for a long time. This is not surprising, since a chronic pattern of hyperventilation would allow the respiratory behavior to become deeply ingrained into the patient's habit patterns. In addition, the secondary gain provided by friends and relatives would furnish an additional stimulus for an individual to cling to his or her symptoms. Many of these patients, therefore, require specific therapy aimed at the correction of the faulty breathing habits, as well as therapy directed at the underlying psychologic disorder, before the symptom complex can be adequately controlled.5,6

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Antimycobacterial Antibodies in Tuberculous Pleural Effusions: Reliability of Antigen 60

To the Editor:

We read with interest three recent articles in Chest1-3 regarding the detection of specific antibodies against mycobacterial antigens in the diagnosis of pleural tuberculosis (PTB). The authors of all three articles found this technique efficient, and we would like to report our experience in this field.

Recently, we had the opportunity to communicate part of this experience with the use of an enzyme-linked immunosorbent assay (ELISA) to detect specific immunoglobulin (Ig) G to mycobacterial antigen 60 (A60) in serum and in pleural fluid." This antigen, purified from the cytoplasm and cellular membrane of Mycobacterium bovis BCG by Cocito and Vanlinden in 1986,7 possesses a thermostable macromolecular structure including free lipids, lipo-polysaccharides, and lipoproteins. It is, therefore, a generic mycobacterial antigen, not a specific one from M tuberculosis. Some European investigators are now using the ELISA of antibody to this antigen for the serodiagnosis of tuberculosis, especially since this test is commercially available (Anda-TB, Bio-Sell).8-9 With this method, the absorbances are translated into units from the graph obtained with positive reference sera of 1, 2, and 16 units.

We have studied 18 serum and pleural fluid samples from patients with PTB confirmed by means of positive culture results in pleural fluid or biopsy specimens or both and/or the existence in the biopsy specimen of granulomas with caseous necrosis. As a control group we studied 33 patients whose pleural effusion was related to a variety of diseases (19 malignant neoplasms, seven cases of transudation, five cases of pneumonia, one case of systemic lupus erythematosus, and one pulmonary thromboembolism). The serum and pleural fluid from patients with PTB had a higher level of anti-A60 antibodies than those from nontuberculous patients. Median serum values were 459.83 for patients with PTB and 107.42 for control patients (p<0.01). For patients with PTB, the median pleural fluid value was 273.61; the corresponding value for control patients was 42.60 (p<0.001).

Unlike Levy et al,3 we have not found any significant difference among the subject categories of the control patients. In our population, a cutoff for a positive ELISA test was established at 150 units in pleural fluid and 235 units in serum. This gives a sensitivity of 50 percent in pleural fluid and 55 percent in serum; a specificity and positive predictive value of 100 percent in both samples; negative predictive values of 78 percent and 80 percent, respectively; and efficiency of 82 percent and 84 percent, respectively. Our results are significantly better than those reported by Murate et al10 (sensitivity, 22.6 percent; specificity, 94.9 percent; positive predictive value, 77.8 percent; efficiency, 62 percent). However, we think, as Levy et al10 do, that measurement of pleural fluid antibody levels does not offer much more information in the diagnosis of PTB than serodiagnosis does. This is consistent with the conclusion that there is no local IgG production and that the level of anti-A60 IgG antibodies in pleural fluid is probably due to passive diffusion. However, this hypothesis does not explain why Van Vooren et al found higher levels of IgG and IgA anti-F32 antibodies in pleural fluid than in serum.

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To the Editor:

Dr Caminero and his co-workers report their experience with a generic mycobacterial antigen. The assignment of a cut-point value has allowed specificity and positive predictive values of 100 percent,

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which would vary according to the arbitrary definition of their cut point. We are encouraged by their agreement that pleural fluid antibody levels do not offer any advantage over serodiagnosis. Areas of the world with a higher prevalence of tuberculosis (such as Spain) will have inherently greater diagnostic reliability because of the relative lack of confounding atypical mycobacterial infection.

Van Vooren and co-workers\(^\text{1}\) adjusted IgG levels to IgG (0.5 g/L) or IgA (1 g/L) concentration before assaying specific antimycobacterial activities. We adjusted levels for total protein content.\(^\text{2}\) This difference in method may explain their finding of higher levels in pleural fluid than in serum in some of their patients. Their study is also compromised by having only five patients in the series, which may have led to sampling error.

We remain convinced that there is no local IgG production and that the level of antimycobacterial antibodies in pleural fluid is due to passive diffusion.

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"Advocate's Disease": An Etiologic Approach

To the Editor:

The article entitled "Advocate's Disease" by Dr Spodick\(^\text{1}\) and the accompanying response by Dr Baffes,\(^\text{2}\) which appeared in the May 1990 issue of Chest, were both thought provoking and enlightening. However, I believe that both Drs Spodick and Baffes are, at least in part, incorrect, and the article and accompanying response deserve further comment. There would seem to be no "malpractice crisis" as described by Drs Spodick and Baffes, but rather a litigation crisis. Medicine is but one of the multitude of professions and businesses with increasing numbers of suits and increasing amounts of awards to plaintiffs. Medical malpractice has been prominent in litigation for the reason so aptly stated by Dr Baffes: "There is too much money involved."

There can be little doubt that medical litigation represents a financial and professional loss to the medical profession. Furthermore, the problem also represents an increasing liability to the patient, who eventually bears, at least in part, the financial burden of medical malpractice litigation. In this age when increasing demands are being placed on the medical profession to reduce the costs of health care, the additional burden of malpractice litigation is neither desirable nor affordable. Therefore, discussions regarding strategies to reduce medical malpractice litigation are both timely and appropriate.

The time has come for the medical profession to reexamine its passive role in medical malpractice litigation. Physicians must accept several premises in order to actively participate in medical malpractice litigation:
1. Physicians have the right to be protected from frivolous and inappropriate suits.
2. Such suits harm the reputation of the individual physicians and the profession.
3. Frivolous suits are a financial burden to the individual physician and to the profession as a whole (through higher medical malpractice premiums).
4. Unwarranted suits increase the cost of medical care by both direct and indirect means (practicing "defensive" medicine).
5. Physicians are probably best able to judge the appropriateness of medical care, which is increasingly technical and complex.
6. Minimal help will be obtained from the business community, which is struggling with its own litigation crisis.
7. Help is unlikely to come from the legal community.

In this context, there are several actions that the medical profession could take through organizations such as the American College of Chest Physicians to decrease unwarranted medical malpractice litigation:
1. The College could make available expert physicians to review the appropriateness of medical care who are willing to testify in court. These witnesses should be volunteers and should be willing to serve for a fee commensurate with their loss of time from professional activities.
2. The College could provide lists of expert legal counsel to physicians.
3. The College could encourage physicians to seek damages from members of the legal community who file inappropriate and frivolous lawsuits.
4. The College and its members could provide education to the public regarding medical malpractice litigation not only through individual contacts but also through the lay press.
5. The College could explore alternatives to medical malpractice litigation, such as binding arbitration.
6. Last, and most important, physicians are a community of professionals dedicated to "reaching out to their fellow human beings." Physicians must not only be willing to reach out to their patients but also must reach out to each other. Only through such cooperation will the principles on which the medical profession was built continue, and only thus will medicine survive in a form that is professionally satisfying to its practitioners and beneficial to its patients.

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Hemoptysis: The Third-World Perspective

To the Editor:

I read with great interest the recent article by Drs Haponik and Chin,\(^\text{3}\) which outlined the practicing physicians' perspectives on diagnosis and management of patients with hemoptysis.

Hemoptysis is a frightening and serious presentation of pulmonary and cardiac diseases. The source of the hemorrhage in pulmonary diseases is mostly from bronchial blood vessels, which are often tortuous, hypertrophic, and dilated.\(^\text{4}\) In some cases the hemorrhage is from the pulmonary arterial system, from Rasmussen's aneurysms, or even from intercostal arteries and other vessels supplying the lungs.\(^\text{3}\)

Although chronic bronchitis and lung cancer are the most common causes of minor, major, and massive hemoptysis in the developing countries, pulmonary tuberculosis (PTB), with its chronic sequelae, such as bronchiectasis, broncholithiasis, and recurrent secondary pulmonary infections, is the most common cause of pulmonary