tell a patient the result of a diagnostic test is a controversial ethical issue and has been debated in the radiological literature. In regards to this specific incident, they are mistaken on two accounts. First, I was not performing the ultrasound in a vacuum, but was aware of the relevant events leading to my contact with the resident who referred her. She asked me to perform the ultrasound when she knew her patient had an unfavorable sonographic result and that she knew she was being referred to us for an ultrasound for further evaluation. Second, I knew that the referring physician would further discuss the problem with her immediately afterwards. I immediately discussed the ultrasound results with her by phone and she promptly returned to discuss the situation further. To have told her such unfavorable news and then to send her away without being reasonably certain she would have contact with her referring physician would have been improper, but this was not the case.

On the broader issue, the argument of Drs Rokey, Rolak, and Vick is based on the premise that disclosure of such information is harmful to the patient. While this may be true in some cases, it is not a universal axiom. Patients in general are less passive about their medical care than in the past, and a paternalistic physician-patient relationship may no longer be the rule. A patient has the right to ask a question of the physician, be it the primary physician or a consultant. By telling the patient, the radiologist is not attempting to usurp the role of, or exclude, the primary physician. We always immediately inform the primary physician and arrange with him or her to see the patient straightaway for further explanation and discussion.

The suggestion also was made that rather than lie or disclose results, the consultant should tell the patient that a review of the data is necessary prior to issuance of a final report to the patient's physician. This certainly is not the truth regarding the diagnosis in this case, and to have done so would have been deceitful. While the results of imaging studies often have to be combined with other information before reaching a firm diagnosis, this is not true for some abnormalities, such as anencephaly, where the sonographic diagnosis is straightforward.

I certainly agree that a physician should not harm the patient. However, the telling of results such as in this case does not necessarily constitute harm. If a patient who is well-informed and cognizant of why the examination is being done asks a direct question of the radiologist, I believe the patient is entitled to a reasonable and truthful answer. To evade or refuse to answer a patient who believes that you have important information may cause more distress and harm than the truth.

Douglas L. Brown, MD
Memphis, Tenn.


ASA
To the Editor.—I suppose you might be mildly annoyed to have your remarks attributed to the American Manufacturers Association. You will understand my annoyance at finding that your abstractor has invented the American Standards Association as the authority for classification of Physical Status, instead of the American Society of Anesthesiologists, as properly attributed by Gluck et al. Perhaps in calling their error to attention you will permit yet another correction. The ASA Physical Status is not an anesthetist's evaluation of risk but rather of global function. A patient with coronary artery disease would be assigned the same Physical Status for either a bunionectomy or bypass grafting. The risks of the operation obviously are different. The ASA Physical Status is a comprehensive and significant risk factor but is the operation. Since sicker patients often require more dangerous surgery than healthy patients (a tautology I admit), Physical Status does vary with risk.

Interestingly enough, the same two errors, misrepresentation of definition and attribution, were made by Lee Goldman and the editors of the New England Journal of Medicine, for which Goldman apologized. Since physical status and mortality have been found by many other studies to closely correlate, it is becoming inevitable that our classification will be regarded as one of risk, I suppose. It is just this success that prompts this plea for proper publication of parentage.

Theodore C. Smith, MD
Riverside, Ill


In Reply.—Dr Smith is correct.

Back in October 1977, in the article by Goldman et al published in the New England Journal of Medicine, ASA was translated to American Surgical Association. The error was pointed out by four members of the American Society of Anesthesiologists in a brief poem, which ended with this moving anesthetic couplet:

To us belongs the classification of risks involved in operation.

This assertion was clouded by a letter from Fels and Owens, who pointed out that the classification of the American Society of Anesthesiologists was never intended to include the variable "operative risk." Dr Goldman did indeed apologize, noting that the switch from ASA to American Surgical Association had been made by the editors and that whatever the founders' intentions, he and other authors had used the classification to estimate operative risk.

In February 1986, JAMA, until then blameless, switched ASA in a letter from Chen et al to American Standards Association. JAMA published a notice of correction ("Incorrect Expansion is what this sort of trick is called") in June 1986. (Coincidentally this appeared on the same page as a letter from a self-confessed nitpicker.)

We publish Dr Smith's letter to put the record straight; to republish the strict constructionist view of the classification; to re-locate the anesthesiologists; and to indicate that this is probably going to be one of those errors we make because general medical journals have a collective, irrational, and incorrigible urge to misrepresent ASA.

Drummond Ronnie, MD


The Epstein-Barr Virus and Chronic Fatigue Syndrome
To the Editor.—Based on eight years of clinical experience and having performed many thousands of Epstein-Barr virus (EBV) serological assays with emphasis on IgG anti-early antigen I cannot agree with the conclusion of Hellinger et al that the antibody to EBV early antigen is not helpful in the clinical evaluation of patients with chronic fatigue syndrome. The initial suspicion of a direct etiologic link between EBV and chronic fatigue syndrome has yielded to the probability that EBV reactivation is a secondary phenomenon related to a primary, transient immune dysfunction, the cause of
which is currently unknown.

In contrast to all other EBV serological markers, which reflect only previous infection, anti-EBV antibody does rise and fall with clinical activity of the signs and symptoms used to define the chronic fatigue syndrome. Although not diagnostic, it is a useful therapeutic marker when initially found in significant elevation (ie, >1:50). This occurs in approximately 70% of patients fulfilling the definition of chronic fatigue syndrome. Therefore, the finding of patients by Hellinger et al who are negative for anti-early antigen yet fulfill the chronic fatigue syndrome definition is not surprising.

Since seronegative and seropositive patients show similar disease, it is evident that EBV is not the primary etiologic agent. However, it is not reasonable to discard a marker that is useful in the majority of cases given the absence of any other more definitive laboratory test. Current emphasis in our laboratory is on demonstrating a functional immune deficiency using a flow cytometric adaptation of passive hemagglutination blastogenesis. Yet, elevated anti–early antigen will remain as evidence of the effect of the immune dysfunction, since reactivation of a dormant herpes virus is not hard to conceive in such an environment.

William J. Herrmann, Jr, MD Memorial City Medical Center Houston

To the Editor.—Hellinger et al1 state that “the validity of the chronic mononucleosis syndrome and the appropriate means for its diagnosis have not been established.” I would like to add a proviso.

Since there were no data presented in this study that either prove or disprove the “validity” of the syndrome itself, the authors should refrain from drawing such a conclusion. This article only addresses the value of Epstein-Barr virus serology in the diagnosis of chronic mononucleosis, also known as chronic fatigue syndrome. Others have reached similar conclusions in the past.2

These results could be interpreted to show that the testing available today is simply not sensitive enough to help diagnose this illness. They also suggest that laboratory testing for chronic fatigue syndrome, in itself, is no substitute for careful history taking, examination, and differential diagnosis.

George R. Reiss, MD Chicago


In Reply.—We agree with Dr Hermann that the cause of elevated antibody to EBV early antigen in some patients with chronic fatigue remains unexplained. However, the results of our study showed that measurement of antibody to EBV early antigen, as it is being used by clinicians in the evaluation of patients with chronic fatigue, was of no practical value. The symptoms, physical examination findings, laboratory evaluation, and subsequent course of patients with chronic fatigue were unrelated to antibody to EBV early antigen.

We agree with the major content of the letter by Dr Reiss. We note that the sentence to which he refers begins, “In our opinion. . . .” With what is now known, and unknown, about EBV, EBV serologies, and chronic fatigue, we believe that measurement of antibody to early antigen is of little practical value in the evaluation of patients with chronic fatigue. In our estimation, the burden of proof is on those who believe EBV can cause a chronic fatigue syndrome to prove such and to provide a straightforward means of diagnosing it.

Walter C. Hellinger, MD Randall S. Edison, MD Mayo Clinic Rochester, Minn

Prophylactic Lidocaine in Acute MI

To the Editor.—MacMahon et al recently reported the effect of prophylactic lidocaine in the therapy of acute myocardial infarction, noting that, even today, we do not have enough evidence to support such use of prophylactic lidocaine. In their extensive reference list, the authors’ earliest relevant reference is from 1967, in which Gianelly et al1 suggest the possible use of lidocaine to prevent ventricular arrhythmias in the coronary care unit setting.

In the same year, our detailed report in another journal dealt with the prophylactic use of procainamide and certain other intramuscular drugs in patients before admission to the hospital with coronary diagnoses. The primary point of our lengthy report and subsequent commentary2 was to indicate that it was not possible to recommend any single-drug therapy in the prophylactic treatment of acutely ill coronary patients. Rather, we pointed out that one probably must use a combination of agents, including those that deal with ventricular fibrillation, atrioventricular block, heart failure, cardiogenic shock, and massive infarction, so as to counteract the possible morbidity effects of the infarction.

Yet, 21 years later, MacMahon et al propose a study to learn if prophylactic lidocaine alone can improve survival in patients with acute myocardial infarction. On the conclusion of such a study, the unfortunate result might be that prophylactic drug therapy would be found not to serve a useful role in the myocardial infarction therapeutic regimen.

At this time, 2 decades after our original report, it must be emphasized that no single pharmaceutical agent can yield the dramatic results that MacMahon et al would hope to obtain from the proposed study. Can prophylactic drug therapy serve a useful role in present-day coronary care given the large therapeutic armamentarium? To learn the answer, we must address all of those factors that are likely to play a role in the survival of the patient, not simply that of ventricular arrhythmia.

As more is learned about the lethal coronary occlusive process, it remains clear that severe chest pain, bradycardia, hypotension, cerebral spasm, hypercoagulable states, heart failure, and other factors also can play key roles in coronary survival. It is this multifactorial approach—not simply the prophylactic treatment of ventricular irritability—that must be the objective in any study such as that proposed by MacMahon et al.

Philip I. Hersberg, MD Needham, Mass


To the Editor.—The excellent overview by MacMahon et al on prophylactic lidocaine in suspected acute myocardial infarction includes one fascinating quotation. The authors state: "With regard to survival after discharge from the hospital, nonfatal VF [ventricular fibrillation] following AMI [acute myocardial infarction] appears to be associated with a worse prognosis." However, this was not the case in the GISSI trial, since an episode of primary ventricular fibrillation was shown to be a negative predictor of survival only in the hospital phase.