nearly 1:1; among African-Americans it was 3:1. Do the authors have an explanation for the disproportionate involvement of African-American females?  

3. A mortality of 10% attributable to sarcoidosis was estimated in an earlier study from this institution. Is it correct to infer from the statement "no patients were considered 'lost to follow-up'" that there were no deaths in 3,645 at-risk patient years of the study? If so, how do the authors account for the change in outcome?  

4. Although the group composition was well matched demographically, no information was provided concerning group allocation by stage or disease duration, both of which substantially influence prognosis. Can the authors provide this information?  

5. The authors employ the terms "relapse," meaning recrudescence of symptoms, and "remission," meaning resolution of symptoms. What do these terms signify in patients in the "Spontaneous Group" who have trivial or no symptoms? Why, for example, would one choose to assign the term "relapse," rather than "progression," to a previously asymptomatic patient whose disease was worsening?  

6. I employ the same usage as Badrinas et al, who distinguish "recurrence," meaning the reappearance of sarcoidosis following complete spontaneous clinical and radiographic resolution, from "relapse," meaning recrudescence of latent disease following partial or complete CST-induced remission. The former appears to be a rare event; identified in 1 of 210 patients reported by Romer, and in 0 of 56 patients reported by Reich and Johnson. Do the authors agree with this distinction?  

Finally, I should like to make four comments:  

1. There is an inconsequential error in the computed percentage (25%) in the sentence "In the British Thoracic Society study, 5 of 25 = 25%. It should be 5 of 25 = 20%."  

2. The interesting speculation that the authors provide to account for the superior course experienced by the untreated patients—corticosteroid treatment...contributed to the propensity for relapse—is supported among persons with recently diagnosed sarcoidosis not only by the cited reports of Eule et al and Izumi but also by an earlier study from their institution. In that study, a randomized double-blind trial of prednisone, a course of 15 mg daily for 3 months was administered to 83 patients; 21 of 46 persons with stage II or III disease received prednisone; 25 were untreated controls. After a mean observation period of 5.3 years, clinical, spirometric, and radiographic evaluation showed a more favorable outcome in the stage II and III untreated group (treated/untreated): definite improvement, 45%/44%; progression, 38%/16%; persistent radiographic abnormality, 71%/60%. These differences did not achieve a 0.1 level of significance.  

Two patients, whose allocation was not specified, died of sarcoidosis. Can the authors provide their allocation? The outcome of these three studies may not be generalizable to the constituent population of this report whose disease was, for the most part, chronic. The recently published British Thoracic Society report (cited by the authors), which demonstrated a small long-term advantage in the treated cohort, involved patients similar in age and therefore probably similar in disease duration.  

3. The authors state: "The purpose of highlighting these two groups [induced and spontaneous] was to explore the possible impact of corticosteroid therapy by comparing two groups of stable, asymptomatic patients with sarcoidosis, only one of which had been treated" [italics added]. This appears to be at variance with the earlier statement that the induced group was treated for "compelling symptoms," and that the spontaneous group had either "asymptomatic radiographic abnormalities or initial symptoms that were not judged of sufficient severity to warrant corticosteroid treatment."  

4. The author's hypothesis that severity of symptoms (and consequent need for CST) accurately predicts an adverse course is supported by Kolek, who observed the converse, a highly favorable course among 1,500 patients with sarcoidosis who were either asymptomatic or had trivial symptoms. Whichever hypothesis one accepts as the most probable—that severe symptoms are an accurate marker of prognosis or that CST adversely affects the course of the disease in some individuals—and the two are not mutually exclusive, the lesson seems clear: CST should be employed with restraint, if at all, in persons with trivial or no symptoms. What do the authors advocate for those patients who are either symptomatic or who are asymptomatic but demonstrate clear evidence of radiographic or physiologic progression?  

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REFERENCES  


To the Editor:  

Thank you for the opportunity to reply to the letter from Dr. Reich.  

Our proportion of patients judged to require corticosteroid therapy (65%) reflects, we believe, a referral pattern to a tertiary institution and to a renowned clinician (H. L. Israel). Our impression is that many of these patients presented with difficult, puzzling, or severe cases of sarcoidosis. We do not believe that this represents an adverse selection of patients; it represents a non-random selection. In addition, the decision to treat was made by a single physician, reflecting an identified source of bias. However, we believe the advantage of the single physician decision point is that it resulted in a consistent bias.  

The ratio of females to males present in this study was observed from referral patterns among patients who were seen at multiple visits over a 4-year period. This observation allows for hypotheses ranging from matters of disease prevalence to cultural values to selection bias; we leave the generation of these or other hypotheses to the reader.  

We would caution Dr. Reich or any other reader against making inferences from our study about prevalence or incidence. Our patients were selected in part by demonstrated interaction with their physician on at least two occasions over the 4-year period. Therefore, patients who had been seen previously but who had died were, by definition, not included in this study.
Despite having read the article four times, Dr. Reich must have missed our Table 1 and page 627. Table 1 demonstrated, in the bottom row, that disease duration in terms of months from diagnosis was not different among groups. On page 627, we note in the second paragraph that similar proportions of patients with radiographic types 1, 2, and 3 received treatment with corticosteroids, although fewer patients with type 0 radiographs had symptoms that required treatment.

We agree with Dr. Reich that our use of the term relapse may be unusual or nonstandard when applied to the spontaneous remission group. We tried to use a single term to denote the appearance of symptoms severe enough to warrant treatment, following a sustained period without such symptoms. Indeed, we could have termed this "progression" in the spontaneous group rather than "relapse." We do agree that recurrence or relapse following complete spontaneous clinical and radiographic resolution is an unusual event. In fact, this represents one of the main points we were trying to make.

Unfortunately, no other data were available from the 1973 study cited by Dr. Reich.

Dr. Reich asks what we would advocate for those patients who are either symptomatic or who are asymptomatic but who demonstrate clear evidence of radiographic or physiologic progression. Our approach is to treat the symptomatic patients with low doses, ie, 15 to 20 mg/day, of prednisone for 6 to 12 months. Those who are asymptomatic but who demonstrate radiographic progression alone do not treat routinely. For those patients who demonstrate clinically significant physiologic progression by pulmonary function studies, we do advocate a trial of corticosteroids at the above dosage.

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REFERENCE


Predictive Value of Pco2 Gap in Infants

To the Editor:

We were interested by the study of Duke and colleagues1 of the difference between tonometer saline and arterial carbon dioxide tension (Dco2) as a predictor of outcome. We have recently studied 62 critically ill adults to assess the usefulness of the gastric intramucosal to arterial carbon dioxide gradient (Pico2-PaCO2), among other parameters derived from gastric tonometry, as a predictor of outcome. Patients were entered into the study within 6 h of admission to our ICU and measurements were taken at 0, 12, and 24 h. At no time was there a significant difference in the Pico2-PaCO2 between those who survived and those who died. The study had a power of 90% to detect a 1 standard deviation difference between the two groups. The area under the receiver operating characteristic curves for Pco2 gap at 0 h as a predictor of ICU mortality and 30-day mortality were 0.54 and 0.57.2 In view of the striking difference between our data and those of Duke and colleagues, we would be interested to know more details of their study to help us account for the difference. Although data were collected from the time of commencement of extracorporeal support, only those data collected during weaning were presented. We would be interested to know whether there was a significant difference in Dco2 between survivors and nonsurvivors earlier in their clinical course, as these data would be more comparable to ours.

We would also be interested to know the time between the measurements during weaning and death in those children who died. Splanchnic ischemia is likely to occur as part of the dying process, and if the time interval between measurement of Dco2 and death was short, the high predictive value may be a reflection of this.

In a separate study we have also looked at the predictive value of lactate in the first 5 days following resuscitation from septic shock in adults. We found that lactate is a predictor of outcome, especially after 48 h, a finding similar to that of other studies of both septic and nonseptic shock.3-5 Surprisingly, Duke and colleagues did not find this to be the case in their patients. It is not discussed in the paper, and we would like to know the authors’ views on why there is this discrepancy.

Finally, unrelated to our research, we would like to know whether and how metabolic acidosis was treated in these children. We are surprised that the pH was normal in otherwise apparently very ill children, particularly in view of the raised lactate levels. Clearly, treatment of metabolic acidosis, particularly with bicarbonate, may reduce the usefulness of base excess as a prognostic marker.

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REFERENCES


To the Editor:

Drs. Gomersall and Joynt make several important points about the relative predictive value of measures made by gastric tonometry. We, too, have recently studied other groups of critically ill children in our ICU, comparing the predictive power of gastric tonometry to other measures. In 90 children after cardiac surgery we found that although low gastric intramucosal pH (pHi) or a high Dco2 predicted important adverse events in the postoperative period, three other variables: the duration of cardiopulmonary bypass; the admission values of mean arterial pressure; and blood lactate were the earliest independent outcome predictors.1 Dco2 and pHi added little to the predictive power of these simpler and less expensive measures. In another study of 30