Despite having read the article four times, Dr. Reich must have missed our Table 1 and page 627.1 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 we 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. Comersall 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