this considerably decreases the specificity(2). Hence using 400 nmol/L as cut off would reduce the specificity further. Furthermore, at least in the evolution of adrenal dysfunction due to primary adrenal insufficiency, stimulated cortisol values become abnormal earlier than basal cortisol values(4). Hence there is no reason to believe that it is different in secondary adrenal insufficiency, where the trophic action of ACTH is lost. In the absence of normal controls to substantiate the basal cortisol values, the authors should refrain from using the term "adrenal dysfunction" to describe patients with basal cortisol <400 nmol/L.

Hence, it is incorrect to classify a patient with "low" basal cortisol (<400 nmol/L) and normal post ACTH cortisol as having adrenal insufficiency. Obviously, such a state has no therapeutic implication. Further, it gives an impression that adrenal dysfunction is very common (9 out of 20 patients) in thalassemics who have received multiple transfusions.

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Reply

We thank Dr. Mathew John for his interest in our article(1). His comments are very meaningful. The following paragraphs address the suggestions/comments:

Regarding the 1 µg ACTH test, only freshly prepared dilution of the standard preparation was used and it was not stored for further use. The 30 min value was used in the 1µg test to define adrenal insufficiency, in accordance with the current recommendation, though the 60 min response was also assessed. In the solitary patient, who had failed the 1µg ACTH stimulation test, the basal and 30 min cortisol levels were 230 and 300 nmol/L, respectively.

We agree with the comment that the baseline cortisol values alone cannot be used to diagnose adrenal insufficiency. It is precisely for this reason that we carefully avoided the use of the term "adrenal insufficiency" in the concluding paragraph. Instead we designated it as a subtle abnormality of adrenocortical function.

Though the baseline cortisol cut off value of 400 nmol/L may appear arbitrary but it was based on published evidence on adrenal function during stress of illness or pharmacological stress. Stewart, *et al.* had shown that no patient with a morning cortisol value >14 mg/dL (~400 nmol/L) failed an insulin tolerance test(2). Similarly it has been shown that

random cortisol value below 13 mg/dL (~360 nmol/L), during severe stress is a predictor of increased mortality with potential benefit with glucocorticoid supplementation(3). Since thalassemics are chronically stressed with anemia, hypoxia and multiple organ dysfunction, we proposed that a baseline cortisol value of <400 nmol/L is inappropriately low for these patients. Any intermittent acute ilnness has the potential to precipitate adrenal insufficiency. Though adrenal insufficiency, by accepted criteria of stimulation tests, is less common in thalassemics, a high index of suspicion is warranted to diagnose and treat this condition in thalassemics in an appropriate clinical setting.

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Role of Shunt Surgery in Pediatric Tubercular Meningitis with Hydrocephalus

The authors(1) of "Role of shunt surgery in pediatric tubercular meningitis with hydrocephalus" have tried to delineate the indications and timing of shunt surgery in tubercular meningitis. However, we find an obvious fallacy with the conclusion drawn.

1. The authors have retrospectively analyzed records of 37 children with tubercular meningitis hydrocephalus (TBMH) all of whom underwent ventriculoperitoneal shunt surgery, correlating stage of disease at the time of surgery and outcome. They conclude that since children who were shunted in earlier stage did better, shunts should be performed in all children with TBMH as soon as they are diagnosed.

   It is well known that stage of the disease at the time of diagnosis is a strong prognostic indicator in TBM because it also determines the time when antitubercular therapy was started. Time of starting antitubercular therapy would thus act as a confounder/effect modifier as it is itself a predictor of outcome. The authors have not taken this into account. Although they mention the duration of antitubercular treatment in the group as a whole, they have not analyzed this with respect to outcome. To delineate the beneficial effect of shunt surgery and stage, one would have to compare outcomes in shunted vs nonshunted children stratified for stage. The better outcome in children who were shunted in earlier stages of TBM was probably because antitubercular therapy was also started earlier in these patients.

2. The authors themselves admit that shunt surgery in children has a high rate of complications. The shunt remains in place