throughout the life of the person. In the social mileu of our country, it would be a stigma making marriage and jobs difficult. We can recount many children in whom shunt surgery was advised but was refused by the family and on follow up the child was normal or near normal. Such children would have been subjected to unnecessary shunt with its attendant complications and stigma.

3. It is quite clear that hydrocephalus in TBM does arrest in a proportion of patients. What is not clear is (i) What clinical or radiological features in the patient predict arrest of hydrocephalus (ii) whether shunt in such cases improves outcome and (iii) what clinical and radiological features (apart from just stage of disease) predict a favourable response to shunt surgery. I think these are questions that urgently need to be answered. Till then, it may be best to give a trial of medical treatment and shunt surgery be kept for those with TBMH who fail to improve on medical treatment.

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Reply

1. It is correct that antitubercular therapy is also responsible for the beneficial effect in these patients, and we have not claimed

otherwise. We feel that it is not justified to wait in a patient who has neurologic deficit (with hydrocephalus) or in the case of a drowsy patient. The shunt procedure itself takes care of the hydrocephalus related symptomatology, and the continuing antitubercular therapy treats the meningitis. To differentiate the beneficial effect of antitubercular therapy from shunt by stratifying the duration of antitubercular therapy would require a much larger patient population, and should be done as a prospective study wherein issues like compliance can be better kept track of. The above cannot be expected from our retrospective review of 37 patients. The policy of purely expectant antitubercular treatment is followed by us only in patients with TBMH having headache, or other signs of raised intracranial pressure but without neurologic deficits or alteration in sensorium, and it is true that in some such patients we may be able to avoid need for shunt eventually. We do not follow the policy of shunt placement in ALL TBMH cases, as has wrongly been interpreted. This has been clearly brought out in the discussion section.

2. It is well-known that shunts done for TBMH have poorer results and higher complications. However, we do not feel that patients with a neurologic deficit or alteration in sensorium should be managed expectantly on ATT. The 62% and 40% good outcome seen in our study in Grade 2 and Grade 3 patients, respectively points to this fact. The remaining patients did not improve despite continuing ATT, pointing to the fact that one should aggressively treat these patients with all means available and not have unrealistic expectations from ATT alone. The social stigma and related issues are

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- worth considering only in Grade 1 cases, whom we also treat expectantly, and shunt only if they fail to improve on follow up. It is better to endure social stigma than a permanent deficit.
- 3. We agree that it is important to identify parameters that may predict high probability of improvement following ATT alone, but at the same time, treatment for each patient having TBMH should be individualized and take into account all available clinical, laboratory and radiologic data. Advocating a universal policy

of shunt only after trial of ATT for all TBMH patients would lead to even patients with deficits and altered sensorium being managed expectantly on A TT for a variable length of time (according to the policy of the treating physician), which is potentially disastrous.

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