The presence of proteinuria and hyaline casts in the urine is nonspecific. Increased excretion of proximal tubular enzymes and beta-2 microglobulin too are only an epiphenomena and hence indicate aminoglycoside effect and not nephrotoxicity(4). Aminoglycosides and creatinine are both good markers of glomerular filtration rate. Hence, it is recommended that simple and inexpensive serum creatinine determinations combined with a limited number of serum aminoglycoside assays be used for monitoring drug efficacy and toxicity(4). The facilities for monitoring drug levels are virtually non-existent in a developing country like India. Thus, for the practicing pediatrician developing a strategy to reduce the risk of aminoglycoside nephrotoxicity requires appreciation of clinical risk factors besides the biochemical monitoring(4). There is a high risk of nephrotoxicity in presence of hypotension, volume depletion, acidosis, concomitant nephrotoxic drug therapy and hypokalemia. In these situations adequate corrective measures should be undertaken along with close biochemical and clinical monitoring. Correct dose adjustment according to renal function and use of lesser nephrotoxic aminoglycoside are the other therapeutic strategies. Last but not the least as highlighted by the authors, the need for a rational use of drugs can not be overemphasized.

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Reply

Eight patients receiving gentamicin showed evidence of nephrotoxicity. Five of these patients were receiving cephaloridine concomitantly. Other predisposing factors including hypotension, volume depletion and hypokalemia were seen in 5 patients. None of these patients had oliguria. Elevated levels of blood urea and creatinine were seen in all the cases.

Aminoglycoside nephrotoxicity is characterized by a variety of renal functional alterations. Reduction of the glomerular filtration rate, manifested by raised levels of serum creatinine, is the *final* manifestation of this disorder. Prior to renal excretory failure, subtle derangements predominated by proximal tubular abnormalities are seen. These include enzymuria, tubular proteinuria and urinary concentration defects. These represent nephrotoxicity and not merely an aminoglycoside effect(1), as suggested by Drs. Gulati and Sharma. Elevated levels of urine beta-2 microglobulin and proximal tubular enzymes are seen 4-5

days prior to significant elevation in serum creatinine(2). Sequential estimation of these products in the urine are useful in monitoring and predicting nephrotoxicity.

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BCG Test in Diagnosis of Tuberculosis

Velhal et al. in their study(1) stated that Tuberculin test is more specific than BCG test, even though BCG test is more sensitive. I wish to make it clear that inspite of several authors in India showing that BCG test is more sensitive than TT, none has bothered to evaluate specificity of this test. However, each author makes a statement that BCG test is less specific.

It is important to realize that in a disease like tuberculosis whose prevalence and incidence is high in the country, any diagnostic test has to be much more specific than sensitive. I, therefore, consider that advocates of BCG test must first define specificity of this test before they consider its utility in the diagnosis of active

tuberculosis. Nobody doubts higher sensitivity of BCG test; but that is not what we should be looking for.

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Reply

In the present study, we have been able to prove the sensitivity as well as specificity of BCG test among proved cases of childhood tuberculosis. However, no attempt was made to define only specificity of the test among the children who were proved to be absolutely free from tuberculosis infection as well as disease.

I agree to his comment that in a disease like tuberculosis, whose incidence and prevalence is very high, specificity of a test carries importance over sensitivity. We would also like to undertake a study to define only specificity among non-sensitized children.

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