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To the Editor:

Drs. Bovornkiti and Matangkasombut have raised some 
questions regarding one or two features of the experimental 
protocol and the interpretation of results which we 
reported in Chest.

We generally agree with these investigators, but wish to 
point out that the study being of a clinical nature, was 
designed to identify some of the problems encountered by 
the physician using the PPD skin test reaction as an adjunct for 
the diagnosis of tuberculosis. We take this opportunity to 
clarify the experimental design and comment on those features 
of the results, and their interpretation, that have been 
questioned.

The accepted use of PPD as a diagnostic aid for tubercu- 
osis has resulted in the commercial distribution of stan- 
dardized preparations. Generally, these clinical reagents 
are prepared in large quantity and the 5TU dose standard- 
ized against PPD-S. The 250TU dose is made from dilu- 
tions of the stock material and represent calculated poten- 
cies determined from the standardized 5TU preparation. 
The preparations we used (Connaught Laboratories) were 
the further standardized in well-controlled studies using human 
subjects.1-3 Although we used a 5TU dose as an inter- 
mediate strength, we have not referred to the 250TU as second 
strength and furthermore feel that the practical usefulness of 
bi-equivalent PPD preparations eliminates the need to 
quantitate, in mg, the concentrations of PPD.

Both the American Thoracic Society4 and American Lung 
Association5 have indicated that a positive skin test to 
5TU PPD given intracutaneously is represented by indura- 
tion equal to or greater than 10 mm. Both published guide- 
lines also suggest that induration from 5-9 mm should be 
considered doubtful. Although we chose to call a skin test 
reaction negative when induration was less than 10 mm, we 
felt it necessary, for completeness, to identify the number of 
doubtful reactors. Furthermore, when assessing reactivity 
to 250TU in patients not responding to 5TU, any response 
suggests that the patient is not generally anergic, but his 
responsiveness to PPD remains 'questionable' since a mini- 
mal reaction could represent sensitivity to cross reacting 
contaminating antigens.

Lymphocytes for in vitro studies were obtained from all 
subjects prior to skin testing. Repeat skin tests were usual- 
ly done within 10-15 days. This was the case for both the 
5TU and 250TU tests done on negative responders. We did 
not note any "booster" effect following the second 5TU 
tests. It is possible, however, that some of those patients 
responding to 250TU, but not 5TU might be responding to 
a prior but heretofore latent sensitivity to PPD. Thus, any 
conversion following the 250TU dose could represent a 
booster phenomenon. Other possibilities are discussed.

Differences observed for total serum protein levels could 
very well reflect a difference in disease state, a variable 
which was not considered in our study. Other differences in 
serum protein values, obtained when the positive and nega- 
tive PPD responders were compared, demonstrated statistical 
significance, but it is doubtful that these differences are 
clinically significant and as such have little, if any, practical 
value. Nevertheless, a more complete analysis taking into 
account the disease state, age, malnutrition, alcoholism, co- 
existent diseases and any other variable known to influence 
serum protein levels should probably be pursued in order 
to resolve the possibility that some serum protein values 
might correlate with PPD skin test reactivity in patients with 
pulmonary tuberculosis.

We agree with Drs. Bovornkiti and Matangkasombut 
when they suggest that suppressor cells could be influential 
in abrogating cellular responsiveness. We wish, however, to 
point out once again that soluble serum factors, inhibiting 
both in vitro and in vivo responsiveness, have been de- 
scribed. In our discussion, we identify several known in- 
hibitors, both cellular and humoral, present in the systemic 
circulation of tuberculosis patients.

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Severity of Aortic Stenosis

To the Editor:

We would like to thank Dr. Spodick for his remarks and 
comments on our paper on the severity of aortic steno- 
is. (See page 122, January, 1981 Chest).

We agree completely with his comments on our use of 
the Baezett formula. Our goal was really not to establish 
rate-related normal values for T time, U time, etc, but 
rather to evaluate them by one recommended technique. 
For an extremely short interval such as the T time, dif- 
f erent methods of rate correction are unlikely to yield a 
practical difference in evaluating disease states.

We were very surprised that our Q-MP data correlated 
better than other measurements (although still poorly) 
with the severity of aortic stenosis. Noninvasive records 
were indeed read without definitive knowledge of cathe- 
erization results. We were interested in investigating this 
because Bonner et al1 had reported these results. Before 
commencing our study, we felt we would be unlikely to 
reproduce these data because of the variability that is 
commonly encountered in phonocardiograms. Hence, we 
also were surprised by our findings.

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Alfred F. Parish, M.D., 
West Roxbury, Mass.

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of aortic stenosis by phonocardiography and external 