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To the Editor:

Drs. Bovornkitti and Matangkasombut have raised some
questions regarding one or two features of the experimental
protocol and the interpretation of results which we re-
ported in Chest.

We generally agree with these investigators, but wish to
point out that the study being of a clinical nature, was de-
signed 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 fea-
tures of the results, and their interpretation, that have been
questioned.

The accepted use of PPD as a diagnostic aid for tubercu-
losis 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-
ties determined from the standardized 5TU preparation.

The preparations we used (Connaught Laboratories) were
further standardized in well-controlled studies using human
subjects.1-2 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 bioequivalent PPD preparations eliminates the need to
quantitate, in mg, the concentrations of PPD.

Both the American Thoracic Society3 and American Lung
Association4 have indicated that a positive skin test to
STU 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 reactions. Furthermore, when assessing reactivity
to 250TU in patients not responding to STU, 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 usu-
ally done within 10-15 days. This was the case for both the
STU and 250TU tests done on negative responders. We did
not note any "booster" effect following the second STU
tests. It is possible, however, that some of those patients
responding to 250TU, but not STU 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-
existen 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. Bovornkitti 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-

We agree completely with his comments on our use of
the Bazett 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-
ferent 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-
terization 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. Parisi, M.D.,
West Roxbury, Mass.

REFERENCE

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