CORRESPONDENCE

This department is provided for the publication of informal communications which are of interest because they are informative or stimulating, and for the discussion of controversial matters.

BLOOD SERUM TESTS

TO THE EDITOR:

With respect to the statement made by Sister Hilary Ross in her paper on the thymol turbidity and other so-called liver function tests in leprosy about observations which I have made (see p. 237 this issue), I have never published anything about them save one short paragraph and a chart in a discussion on tests of that type (*Proc. Staff Meet., The Clinic* **13** (1947) 76-85 (July)). I have, however, kept in contact with the work; and Mrs. Fredricks, in the laboratory at Kalaupapa, has carried on such work for those cases suggested by her physicians. In talking about the matter with Sister Hilary, when visiting Carville in 1947, I expressed the hope that changes in these serological findings might be correlated with the clinical changes which occur during sulfone treatment.

Since talking with Sister Hilary I have added my own adaptation (unpublished) of the colloidal mastic test as applied to serum as a substitute for MacLagan's colloidal gold or Ducci's colloidal congo red, and have found it an excellent confirmation of the other methods. More recently (*Gastroenterol.* **12** (1949) 394-409) I have added Kunkel's method for estimating gamma globulins, and at present it seems to be the method of choice; and it is so very simple.

All these so-called "liver function tests," in my opinion, depend on the reticuloendothelial system, whether the cells be the Kupffer cells of the liver, the reticular cells of the spleen and lymph glands, or the histiocytic bed of the dermis. In the first instance they are excellent indices of hepato-cellular irritation or damage; in the last one they indicate equally well the extent and severity of lepromatous leprosy, which Tilden has so well described as a reticuloendothelial disease.

I find it annoying to hear the professional leprologists argue and split hairs over the classification of leprosy. How many angels really *can* dance on the head of a pin? Isn't it about time that we all quit paying so much attention to macules and

anesthesias, nodules, tiny "snips" and almost as tiny biopsy specimens? Isn't it about time that, since the whole patient has leprosy, we paid more attention to the biochemistry of the patient as a whole? A negative snip and a biopsy might suggest tuberculoid leprosy, but if the biochemistry repeatedly indicated lepromatous leprosy, I would depend on the latter indication of the final outcome of the case.

For example, Sloan recently observed casually a boy at a Boy Scout meeting and investigated further. He found that the boy's father was a patient at Kalaupapa, admitted July 1, 1947, with biopsy and clinical findings of lepromatous leprosy. For social reasons the boy, who was diagnosed tentatively as tuberculoid leprosy, was admitted direct to Kalaupapa rather than being immediately placed on "temporary release" or admitted to Kalihi. At Kalaupapa he was studied further and continued to be classed as tuberculoid. A skin biopsy of April 1949 was reported by Dr. Tilden as follows:

"The epidermis is thin and shows nothing unusual. The upper part of the corium contains a few very small streaks of foci of lymphocytic infiltrate which are concentrated around blood vessels. The involvement is quite superficial in location and the lower part of the corium appears normal. Acid-fast stain has been carefully studied and no organisms could be found. Diagnosis: Non-specific histologic changes; no organisms present."

The biopsy apparently confirmed Dr. Sloan's clinical diagnosis of tuberculoid leprosy. But then the lad gave a negative Mitsuda reaction! Sloan talked his quandary over with me, and I suggested that serum studies might help him, so I was supplied with some fresh serum. Here are the results of the serologic tests: total proteins, 6.8 gm/%; total globulins, 2.8 gm/%; albumins, 4.0 gm/%; A/G ratio, 1.43. By our method and among our peoples, the normal ratio range is from 2.0 to 3.0. This case is definitely low.

The relatively high globulin and low ratio point toward active leprosy. Thymol units were 10.4. The way our colorimeter is calibrated, about 5.5 —possibly 6.0 units—is the upper limit of normal. The 10.4 units suggests a hyperactive reticuloendothelial system. The gamma globulins were 35.4 units, and our upper normal limit is 19.0—possibly 20.0 units. This again points to the reticuloendothelial system. The Hanger test (cephalin-cholesterol) was only 2+, but the indication remained unchanged. The serum colloidal mastic test gave readings of 5-4-2, and our upper normal limit is 2-1-0. The indication of reticuloendothelial hyperactivity seems confirmed. Clinically, this boy has tuberculoid leprosy; serologically, he has lepromatous leprosy. Take your choice. Time will tell. Up to August 1949 it has been silent. I'd be willing to bet on it.

It is time that the leprologists should have not only his detailed clinical findings, along with "snips" and biopsies, but also all the available serologic or biochemic data such as those with which Sister Hilary Ross has been working.

The Clinic E. A. FENNEL, M. D.
Honolulu, Hawaii *Pathologist*