a detailed review of our series of several thousand scans has not been done, we have been impressed with the usefulness of the posterior view. Recently within a span of 1 week we encountered two patients with normal anterior views and either normal or only faintly suspicious lateral views on whom a definite defect was detected on the posterior scan.

**Example Case.** This 68-year-old male was admitted with weight loss, fever of unknown origin, and vague right chest pain. A liver scan (Fig. 1) revealed a large defect in the medial posterior portion of the right lobe seen conclusively only on the posterior view. At surgery a 10 × 15-cm "ball-like" mass was found involving the posterior right lobe near the midline. Biopsy yielded anaplastic adenocarcinoma.

This observation of the importance of the posterior view of the liver is not original to our laboratory, but we feel that it should be reemphasized at this time.

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**TECHNETIUM-LABELED RED BLOOD CELLS**

The concise communication entitled "Technetium Labeled Red Blood Cells" by Eckelman et al (J Nucl Med 12: 12, 1970) was read with great interest. The authors' suggestion that tin chloride functions as a reducing agent after the complexing of pertechnetate to hemoglobin is at odds with our experimental findings. Tin chloride with a tracer of 119mSn can be added to saline-washed red cells before the introduction of sodium pertechnetate. The tin can subsequently be chelated with EDTA and washed with several volumes of isotonic saline. It can be demonstrated that all of the tracer and presumably all of the stable tin has been removed. This procedure markedly enhances the complexing of pertechnetate to the red cell. One can reverse the binding efficiency of pertechnetate by re-exposing the saline-washed red cells to bubbling oxygen after the tin is added. That stannous ion remains with the cells after the EDTA washes is a moot question. The small amount of stannous ion necessary to reduce carrier-free technetium can escape measurement even in tracer experiments.

The point of concern to us in Weinstein's procedure is the preparation of stannous solution. Although care is taken to dissolve all the stannous chloride in HCl, the resulting citrate complex is not stable at pH 7.4 (2). Because the preparation of the stannous citrate solution could be critical, we prefer to add solid stannous chloride to an ACD solution and filter this solution directly into the suspension of the blood cells. Besides assuring sterility of the stannous solution, we also filter out any large (>0.22 micron) particles of undissolved tin.

With this point in mind, we do not feel that Weinstein's data are at odds with our postulated mecha-
nism. In our method, the technetium is added in a small volume and crosses the cell membrane as pertechnetate. Subsequent addition of the stannous ion results in a small fraction of the reducing agent also crossing the cell membrane leading to a reduction of the technetium which then binds intracellularly. Our extracellular technetium is removed in the saline washes as a technetium citrate complex. Although the exact mechanism of the reduction of technetium has not been clarified as yet, reduction of technetium with the red blood cells can be shown by the absence of a pertechnetate peak in gel chromatography studies of hemolyzed labeled red blood cells.

In Weinstein’s and Nouel’s procedures, the excess stannous ion is probably not removed completely so that subsequent addition of pertechnetate will result in reduction of both intra- and extracellular pertechnetate, resulting in a “mock” red-cell label. The technetium reduced outside the cells could be carried through the saline washes with the erythrocytes; e.g. as colloidal particle or as technetium bound to the cell membrane. It is difficult to predict the species or chemical form present without further data.

These theoretical considerations are supported by the biological data obtained with the two procedures. We have shown that cells altered by our excess stannous ion method are taken up by the human spleen (3). In addition to the excellent in vivo labeling stability in dogs and rabbits reported in our original article, we have since obtained in vivo stability data in human recipients. These data are presented in Table 1 and indicate that up to 2 hr the technetium activity is predominantly in the red-cell fraction of the blood, and that there is not a rapid loss of radioactivity from the cells into the plasma, as found with Weinstein’s procedure.

It is very difficult to compare Dr. Weinstein’s experimental work with our own because he presents no quantitative data. However, a few general comments on variation of the procedures can be made. We expect that oxygen and nitrogen would also affect our yields in the same manner as Weinstein’s. We did not report low yields with heparin as Weinstein states, but rather, following Mollison’s work for the analogous chromate labeling, we used only ACD, and stated so clearly. Like Weinstein we obtained higher labeling yields after purge of the cells with carbon monoxide as seen in Table 2 of our article, but did not make the procedure a part of our routine because, as stated in our article, it caused agglutination of the cells if carried out too long.

We feel that further work on the mechanism is in order to indicate more clearly whether the stannous ion is directly reducing the technetium or reducing an intermediate which, in turn, reduces the technetium. Further comparisons of the mechanisms of labeling by $^{51}$Cr and $^{99m}$Tc are also in progress.

**TABLE 1. DISTRIBUTION OF RADIOACTIVITY IN HUMAN BLOOD**

<table>
<thead>
<tr>
<th>Time after injection (min)</th>
<th>Percent $^{99m}$Tc in RBC</th>
<th>Percent $^{99m}$Tc in plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>99</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>96</td>
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<td>97</td>
<td>3</td>
</tr>
<tr>
<td>120</td>
<td>97</td>
<td>3</td>
</tr>
</tbody>
</table>

**REFERENCES**

1. **NOUEL JP, BRUNELLE P**: Le marquage des hématies par le technetium 99m. *La Presse medicale* 78: 73–78, 1970

**FURTHER OBSERVATION ON $^{131}$I-ROSE BENGAL CLEARANCE IN GILBERT’S DISEASE**

I would like to add a brief note to the article by Iio et al on the diagnosis of constitutional hyperbilirubinemias with $^{131}$I-BSP (1). Their patient with Gilbert’s disease had normal radiiodinated BSP and rose bengal kinetics when the test was performed 2 hr after breakfast. However, Felsher et al (2) recently demonstrated a reciprocal relationship between caloric intake and the level of indirect hyperbilirubinemia in Gilbert’s disease. They demonstrated a rapid rise in bilirubin level in response to fasting (less than 400 calories/day) with peak levels occurring at 48 hr. Therefore optimum conditions for detecting abnormal radiiodinated dye kinetics in Gilbert’s disease would be during the maximum stress of a 48-hr fast rather than shortly after breakfast.