by cell separation was more than 96.5%. In euthyroid patients only faint thyroid uptake was noted.

The thyroidal Tc-99m uptake reaches a plateau shortly after its i.v. administration, remains there approximately 1 hr, and then falls gradually (2). It is reported that in hyperthyroid patients 20-min thyroid uptake of pertechnetate ranges from 3 to 36% (3). Carotid-thyroid transit time of pertechnetate is also much shorter than in euthyroid or hypothyroid patients (4). We suggest that in hyperthyroidism a significant fraction of injected pertechnetate might be trapped in the thyroid within several transit times before it could label circulating RBCs, and that this will eventually obscure the cervical great vessels. Moreover, free pertechnetate released from the thyroid might degrade the blood pool image to some extent several hours after the injection. In hyperthyroidism, in vivo labeling of RBCs therefore seems inadvisable.

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REFERENCES


Reply

Dr. Ueno’s observation is certainly true: in hyperthyroid patients there is uptake of Tc-99m by the thyroid when our in vivo RBC labeling protocol is used. This is to be expected because the RBC labeling process is not instantaneous, requiring approximately 4 min for completion. This indeed makes the visualization of the cervical great vessels more difficult, so if one is interested in that particular area one should be aware of the thyroid problem. However, the suggestion that “in hyperthyroidism, in vivo-labeling of RBCs seems inappropriate” appears unwarranted. From the theoretical point of view one cannot expect a very high percentage of activity to concentrate in the thyroid, because the supply of free TcO\(_4^-\) to the thyroid is dwindling rapidly after the first passage due to the in vivo labeling protocol. Our own clinical experiences, as well as the illustration submitted by Dr. Ueno himself, prove that the blood-pool visualization is perfectly acceptable even in the presence of a hyperfunctioning thyroid. The rate of TcO\(_4^-\) release from the gland is unable to alter the images in any significant way. Besides, any released pertechnetate may contribute to the RBC labeling process.

As far as the salivary and gastric uptake is concerned, we are not aware of any interference with the interpretation of blood-pool scans. Concerning the choroid-plexus shadow, it is most unlikely that this is due to free pertechnetate, but rather to its blood pool. In addition, an interesting suggestion has recently been formulated linking the choroid plexus image with a direct effect of tin (1).

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REFERENCE


Comparison of 19-Iodocholesterol and 6-Iodomethylcholesterol as Adrenal Scanning Agents

In a recent publication (1), Couch and Williams reported inability to visualize adrenal glands in Sprague-Dawley rats by scintillation scanning with pure \(^{131}I\) 19-iodocholesterol. They attribute previous successful adrenal visualizations to the presence of the isomer of 6-iodomethylcholesterol as a contaminant of labeled 19-iodocholesterol preparations.

We have had considerable experience in the preparation of \(^{131}I\) 19-iodocholesterol and recently have been analyzing our products by means of high-performance liquid radio-chromatography. The reverse-phase column technique affords unequivocal separation and quantitation of the labeled iodocholesterol isomers, in contrast to TLC methods. In our hands, typically, 19-iodocholesterol containing 0.3% of the 6-isomer yields \(^{131}I\) 19-iodocholesterol containing barely detectable amounts of the labeled 6-isomer. Since the clinical experience with our pure product has been entirely satisfactory, as reported at the 24th Annual Meeting of the Society in Chicago (2), we do not believe that attribution of bioactivity to contamination by the 6-isomer is valid.

Scintillation scanning with each of the pure 1-131 labeled isomers in a very limited number of Sprague-Dawley rats in our laboratory gave results not unlike those reported by

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