Quantity of Tin in Bone Imaging Agents

Krishnamurthy and colleagues (*1*) report that tin content has no effect on the performance of bone imaging agents, and recommend therefore the use of low-tin agents to avoid interference with subsequent brain scans. One factor they did not study, which may prove pertinent, is the handling of the vial prior to administration. The air sensitivity of these compounds is well known. In their report a separate vial was used for each patient; but when a multidose vial is handled in the average clinic, opportunity for inadvertent admission of air may be much greater. The reducing capacity of excess tin should provide a margin of protection against this contingency. It may be hasty to condemn the high-tin agents without looking into this possibility.

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REFERENCE

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Reply

Russell (above) brings out an important point about inadvertent introduction of air into a multidose vial. One may speculate that excesses of tin in a low ratio kit would safeguard against air being inadvertently introduced into the vial. However, there is no proof of this and the manufacturer of both the kits caution against introducing air into the multidose vial even inadvertently, meaning that excess tin in a low ratio kit may not be a protection against air introduction into the vial.

When multiple doses (three to five) were drawn carefully from both high and low ratio kit vials, there was no subjective difference in the quality of bone images. We did not intend to condemn the low ratio kits, as the problem of excess tin would arise only if technetium-99m pertechnetate is used for any clinical studies. As pointed out in our paper, the pertechnetate is being replaced more and more by Tc-DTPA or Tc-glucoheptonate and the problem of excess tin may not be real clinically. If technetium-99m pertechnetate has to be used, then a high ratio agent (low tin) is preferred. On the other hand, the real need for excess tin in the low ratio vials is not clear.

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Increasing Incidence of Hypothyroidism within 1 yr after Radioiodine Therapy for Toxic Diffuse Goiter

Drs. Von Hofe et al. (*1*) come to the conclusion that the incidence of hypothyroidism after radioiodine therapy for diffuse toxic goiter is increasing. This may or may not be true, but their published data are insufficient to support that conclusion. The problem lies in the complete uncertainty of the thyroidal radiation dose. The authors state that their plan of therapy was to administer 10 mCi of I-131, regardless of gland size or radioiodine uptake. In their table the range of 24-hr radioiodine uptakes is listed as 16–83%. If we assume a half-time of 6 days for thyroidal radioiodine and a gland weight of 40 g, the dose range to the thyroid resulting from their ranges of uptake is 3,600–19,000 rads (*2*). Depending on the actual thyroid weights and turnover times associated with a particular uptake value, the actual dose variations in the series might be more or less but certainly highly significant. Although the relation between thyroidal radiation dose and clinical outcome may not be simple, it has been shown (*3–5*) that, at the very least, hypothyroidism increases as the dose increases within the above dose range. It might be argued that clinical practice does not require the effort of detailed dose estimates in view of their uncertainties. The clinical results might be acceptable without such calculations, but dosimetry is necessary if one wishes to draw any sort of scientific conclusions about dose-related phenomena. This is not to say that explicit dose calculations are sufficient to describe the dose-response problem, since other factors such as gland size can affect the sensitivity of the thyroid to radiation (*6*), but because the individual response to radioiodine is beset by a number of variables, the failure to control as important a factor as dosimetry only further confuses the question.

Similarly, the conclusion that thionamide pretreatment protects against hypothyroidism must be supported by dosimetry information. It is conceivable, for instance, that all pretreated patients had a lower radioiodine uptake at time of therapy and thus actually received lower thyroidal doses.

The fact that previous studies cited by Von Hofe et al. reported total mCi doses rather than rads merely reflects previous shortcomings and does not justify a conclusion based on comparison with such studies. A comparison of series in which dose effects are observed obviously must be compared on the basis of dose delivered to the thyroid.

It would be useful if the authors would retrospectively calculate the radiation doses in their series in terms of rads to the thyroid or, failing the availability of turnover data, in terms of μCi/g taken up by the thyroid or, in the absence of weight estimates, in terms of total mCi to the gland.

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