our first published estimate of dose to the kidney (*3*), we simply fit the trailing portion of the kidney curve to a single exponential and extrapolated that fit to infinite time. We did not feel that this extrapolation was optimal, however, and the second estimate used a different method (*4*). The plasma curve over the first 20 min was deconvolved from the kidney curve to derive an impulse response function for the kidney. Plasma data had been collected for 3 hr and the impulse response function of kidney was reconvolved with all 3 hr worth of plasma data to generate a 3-hr kidney curve that was finally extrapolated with a single decaying exponential. As noted, this second method led to an estimated kidney dose 50% higher than the first, but our data do not support the Mallinckrodt Diagnostica estimate that is yet another factor of 3 higher.

We are pleased to note that Dr. Bubeck finds approximate agreement with our reported value of 0.025 rad/mCi to the kidneys from [^{123}I]OIH and he cites for confirmation the 1978 work of Roedler et al. (*7*). It is important to note that the same authors (*8*) reported a much higher dose of 0.065 rad/mCi to the kidneys from [^{123}I]OIH in 1973 and it was these higher dose estimates (*8*) that were unfortunately transcribed into NCRP report No. 70 (*9*) and into a recent article on pediatric OIH dosimetry (*10*).

In conclusion, we agree with Dr. Bubeck on the importance of detailed dosimetry information regarding technique, assumptions and methods and we are preparing a detailed comparison of [^{99m}Tc]MAG_3, [^{131}I] and [^{123}I]OIH, and [^{99m}Tc] diethylenetriaminepentaacetic acid dosimetry.

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Swedish Thyroid Cancer Risk From Chernobyl?

TO THE EDITOR: *J Nucl Med* 1988; 29: 1719, Strand et al. (*1*) present a study on the activity content of iodine-131 (^{131}I) and of iodine-133 (^{133}I) in the thyroid glands of 18 autopsied subjects from southern Sweden following the accident of Chernobyl in 1986. They estimated that the increase in dose equivalent to the thyroid for the whole population of southern Sweden due to the released ^{131}I and ^{133}I will be <0.1 mSv. The authors suggest that this radiation exposure may increase the incidence of thyroid cancer of 0.1% during a period of 25 yr.

We disagree with their conclusion mainly for two reasons. First, they used the previous UNSCEAR risk figure of 50–150 cases per 10^4 manSv for radiation induced thyroid cancer during a period of 25 yr. UNSCEAR believes that the cancer risk from gamma rays at low radiation doses and dose rates is lower than the values assessed for high doses and dose rates. A reduction factor would therefore be needed to calculate this risk and in the 1986 and 1988 UNSCEAR reports the appropriate range for the reduction factor is assumed to be between 2 and 10.

Second, no epidemiologic study has observed any clearly increased cancer risk after exposure to low to intermediate doses of ^{131}I. In a recent study on the incidence of thyroid cancer among 35,074 patients examined with diagnostic doses of ^{131}I (mean 1.92 MBq) between 1951 and 1969 no increased risk for thyroid cancer was observed with a mean follow-up period of 20 yr (*2*). The study supported the notion that the carcinogenic potential of internally deposited ^{131}I is much less than external x-rays or gamma rays.

The average radiation dose to the thyroid in that study was 500 mSv. It is therefore unlikely that a mean dose of <0.1 mSv to the thyroid gland would result in any increased thyroid cancer risk.

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