Dementia scale. They did not reveal any symptom of aphasia, hemiparesis or other dyskinesia; only memory problem and/ or disorientation were present. In such patients, we could not find any asymmetric reduction, and the frontal reduction in cerebral blood flow was not observed.

2. We were previously unaware of the abstracts and paper. However, the paper does not discuss the differentiation of the patterns of regional reduction in cerebral blood flow among Alzheimer's disease, senile dementia Alzheimer type and multi infarct dementia. Also, our report appears original in the correlation between reduction in regional cerebral blood flow and degree of dementia in each demented group.

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# Radiation Exposure by Technitium-99m MAG<sub>3</sub>

**TO THE EDITOR:** According to Taylor and colleagues (1) the radiation exposure by technetium-99m ( $^{99m}$ Tc) MAG<sub>3</sub> for the kidneys of normal volunteers is 0.018 rad/mCi; the dosimetry calculations have been only briefly described (2). This value is 50% higher than the former given by this same team (0.012 rad/mCi) (3), that was not discussed in this paper.

Mallinckrodt Diagnostica (Netherlands) B.V., the commercial producer of [Tc]MAG<sub>3</sub> for Europe, indicates in the European Registration File an organ dose for the kidneys of 0.062 rad/mCi, that means the 3.4-fold (1) or 5.2-fold (3) value (all calculations followed standard MIRD methods). Surprisingly, there are no major differences between the analogous results for iodine-123 (123I) and iodine-131 (131I) orthoiodohippurate (OIH), respectively, by Taylor et al. and the European reference values (4). In comparison with [123I]OIH (0.020 rad/mCi) (4) Taylor et al. found significantly lower while Mallinckrodt found distinctly higher values for [99mTc] MAG<sub>3</sub>. These differences are considerable and should be proved by more related measurements with detailed information about technique and methods of calculation.

Our theoretic estimate showed the following results: the effective half-lives of the radiopharmaceuticals mentioned above at normal renal function are approximately equivalent (1,5) and the absorbed energy fraction of the gamma radiation is nearly the same for both nuclides. Furthermore, the part of nonpenetrating conversion electrons additionally generated during decay is comparable, too. But because of the essentially higher blood concentration of [99mTc]MAG3 compared with [123I]OIH (1,5), the activity level of 99mTc in the kidneys is higher than of 123I resulting in a higher organ exposure by [99mTc]MAG3 in normal functioning kidneys. In severely reduced renal function the radiation exposure may be less by [99mTc]MAG3 than by [123I]OIH because of the distinctly shorter physical half-life of 99mTc compared with 123I.

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**REPLY:** We appreciate Dr. Bubeck's letter regarding radiation dosimetry and especially his request for "more related measurements with detailed information about technique and methods of calculation."

Dr. Bubeck's letter focuses on the estimated dose to the kidneys from [99mTc]MAG<sub>3</sub> and notes that our estimates (1, 3) are one-third to one-fifth the estimate reported by Mallinck-rodt Diagnostica (Netherlands) B.V. in the "European Registration File." Our lower estimate was based on the kinetics of high performance liquid chromatography purified [99mTc] MAG<sub>3</sub> while the higher estimate was based on the kit formulation. The kinetics are slightly different (2,4). Probably more important than the kinetic differences are differences in the assumptions and the methods of calculation used in the two studies. These are briefly outlined below.

The MIRD method is quite universally adopted for dosimetry calculations and is probably applied correctly by all parties. The MIRD method requires, however, an estimate of the amount of activity in the source organ as a function of time and herein lies the challenge and potentially large discrepancies. When estimating the dose to the kidney, the radiation to the kidney from the bladder and other organs is practically insignificant compared to the radiation to the kidney from the kidney itself; for this reason we have to focus on the amount of activity in the kidney as a function of time. The estimates we reported (1-4) were based on Anger camera images acquired posteriorly over the kidneys and stored in a mini-computer at 20 sec per frame for 20 min. The activity in both kidneys as a function of time was estimated from regions of interest over the kidneys. The system was calibrated by imaging a kidney phantom (Alderson Research Laboratories) in a water bath with the center of the phantom at depths ranging from 5 to 8 cm. The calibration factor for each volunteer depended on his own kidney depth as estimated by the formulas of Tonnesen et al. (5). We note that the formulas of Tonnesen were supported by Tanasescu et al. (6) by comparison to ultrasound. We feel that this method of estimating the activity in the kidney is probably accurate to within 15%, especially when averaged over ten volunteers (as was done for our dose estimation). The weakness of this method is that Anger camera images were not extended beyond 20 min and the longer term activity in the kidney can be significant. In

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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 [99mTc]MAG<sub>3</sub>, [131I] and [123I]OIH, and [99mTc] diethylenetriaminepentaacetic acid dosimetry.

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### Swedish Thyroid Cancer Risk From Chernobyl?

et al. (1) present a study on the activity content of iodine-131 (<sup>131</sup>I) and of iodine-133 (<sup>133</sup>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 <sup>131</sup>I and <sup>133</sup>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<sup>4</sup> 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 <sup>131</sup>I. In a recent study on the incidence of thyroid cancer among 35,074 patients examined with diagnostic doses of <sup>131</sup>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 <sup>131</sup>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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