We presented this concept at the Society of Nuclear Medicine Annual Meeting in June 1996 (*2*) and published an article titled "Quantification of Left Ventricular Function with Thallium-201 and Technetium-99m-Sestamibi Myocardial Gated SPECT" (*3*). We are pleased that Germano et al.'s (*1*) article confirms our observations that were submitted earlier but delayed in publication.

Again, we are happy to learn that another research group has confirmed these results. We believe that gated SPECT with ^{201}Tl is both effective and reliable. Therefore, clinical sites that prefer ^{201}Tl for myocardial perfusion imaging can perform gated SPECT and obtain useful functional information that was once thought to be possible only with ^{99m}Tc-sestamibi.

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Is Technetium-99m-MIBI a Relevant Tracer to Tumor Response to Chemotherapy of Bone Lesions?

TO THE EDITOR: We do not agree with Taki et al.'s (*1*) conclusion that ^{99m}Tc-MIBI detects bone and soft-tissue lesions and assesses tumor response to chemotherapy comparable to ^{201}Tl. Technetium-99m-MIBI accumulation in tumors is modulated by blood flow, cell viability and the level of permeability glycoprotein (Pgp) expression (*2*). Technetium-99m-MIBI is a well-documented transport substrate for Pgp, which is induced by overexpression of the multidrug resistance (MDR) gene, which in turn is a major cause of chemotherapy resistance and failure (*3–5*). In MDR gene cells, a high concentration of Pgp on the cellular membrane induces a rapid excretion of ^{99m}Tc-MIBI; ^{99m}Tc-MIBI accumulation by MDR tumor cells remains low. This low ^{99m}Tc-MIBI uptake can help assess MDR gene overexpression (*6*).

Table 2 in Taki et al.'s (*1*) article clearly indicates such a possibility. Patients 10 and 12 had decreased ^{99m}Tc-MIBI tumor uptake postchemotherapy (−5% and −19% compared to prechemotherapy), which is consistent with MDR development. Uptake ratio of ^{201}Tl did not decrease (+21% and +1%), and the tumors did not respond to chemotherapy. Patients 23 and 25 seem more complex: ^{99m}Tc-MIBI uptake ratios increased after chemotherapy, although the tumors were nonresponsive (Table 2) (*1*). Thallium-201 uptake, however, decreased only moderately after chemotherapy (−18% and −3%, respectively), indicating residual tumor viability.

We conclude that the observation of ^{99m}Tc-MIBI uptake decrease after chemotherapy is consistent with either weakening of tumor viability or induction of MDR.

Technetium-99m-MIBI provides more information than ^{201}Tl in assessing tumor response to chemotherapy.

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Time for a Change?

TO THE EDITOR: Perhaps those of us in nuclear medicine should consider following the example of the American College of Physicians (ACP) and the American Society of Internal Medicine (ASIM). The ASIM was founded by some internists who did not believe that ACP adequately addressed the socioeconomic issues of medical practice, in view of ACP's great interest in education.

The American College of Nuclear Physicians (ACNP) was formed because it was believed by some nuclear medicine physicians that the Society of Nuclear Medicine (SNM) did not adequately address the practice concerns of physicians, being interested primarily in research and education. There remain some areas of duplication of efforts, although many important activities have been coordinated or combined.

ACP now appears to be close to joining forces with the ASIM. The members and leadership have concluded that there is really no longer a reason to have two different organizations. They want internal medicine to speak with one voice. In a survey of the 100,000 members of ACP and the 20,000 members of the ASIM, informing them of the possibility of a merger, reaction ran about 5 to 1 in favor of the merger. It might be interesting to poll members of both groups on their opinion about the desirability of a possible merger of SNM and ACNP, a merger, not a takeover of ACNP by SNM.

Not the least of the advantages of a merger of SNM and ACNP is the beautiful and luxurious building SNM now owns in Reston, VA.

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Gene Radiotherapy; Gene Targeted Versus Targeted by Gene Product

TO THE EDITOR: The editorial by Larson et al. (*1*) describes a very interesting and promising approach for in vivo detection of the expression of a gene delivered into tumor cells. In referring to a personal communication from one of the coauthors, the writers suggest the use of this approach also to deliver higher doses of therapeutic radiation to cells expressing a "marker gene." This method, which the authors call "gene-targeted radiotherapy," if demonstrated to actually work, is potentially extremely powerful. We, however, have concerns about the terminology they used to describe their approach.

The application of the methods of molecular biology have recently become more evident in nuclear medicine. This symbiosis can be extremely productive and result in significant progress in both tumor imaging and radiotherapy. As often happens in a new field, the terminology has not been standardized and different approaches are sometimes called by the same name. For example, we also have described a type of "gene-targeted radiotherapy" in an interview and article written by Kotz (*2*).

For the last 4 years, we have been developing our approach for targeting Auger-electron emitters (AEs) to specific genes using triplex-forming oligonucleotides (TFOs) as delivery molecules. This method combines the