for it signals reversible damage whereas the gluco-
ate and the tetracyclines react only to substantial 
reductions in blood flow, severe enough to indicate 
possibly permanent damage.

For all $^{99m}$Tc complexes tested, there was a thresh-
old level in bloodflow reduction below which 
concentration of the tracer was the same as in histolog-
ically normal regions distant from the infarct. At a 
critical point in bloodflow reduction, the concentra-
tion of the radiotracer began increasing in a linear 
form as blood flow was reduced further. This 
threshold point varied with the radiopharmaceutical. 
The very small reduction in flow with $^{99m}$Tc-diphos-
phonate would suggest that this tracer was very sen-
sitive to changes resulting from bloodflow reduction, 
sufficiently sensitive so that ischemic, reversibly dam-
agecd tissue might result in increased tracer concen-
tration. In fact, it is possible that for $^{99m}$Tc-dipho-
phonate no threshold exists at all since the variability 
in blood flow throughout the normal left ventricle is 
approximately 15% (10). On the other hand, the 
myocardial concentration of $^{99m}$Tc-tetracycline and 
$^{99m}$Tc-glucopentate increased only with substan-
tial reductions in flow, suggesting that these tracers 
concentrate only after significant cellular damage has 
occurred.

Therefore, if there exists a clinical need to deter-
mine the size of a frankly infarcted area, $^{99m}$Tc-
tetracycline or glucophenone would be the tracers of 
choice, at least in the current animal model. In an 
acute coronary episode the diphosphonate, reacting 
also to minor degrees of ischemia, would include 
some regions that might recover.

ACKNOWLEDGMENT

This work was supported in part by USPHS Grants HL 
17739, GM 18674, GM 01910, and GM 02201.

REFERENCES

and sizing of acute myocardial infarcts with $^{99m}$Tc(Sn)-tetracycline. 
method for radionuclide imaging of acute myocardial in-
fraction in humans. Circulation 50: 540-546, 1974
and localization of experimental myocardial infarction 
4. Fink/Bennett D, Dworkin HJ, Lee Y-H: Myoc-
dardial imaging of the acute infarct. Radiology 113: 449, 
1974
Visualization of acute myocardial infarction by the radio-
6. Braunwald E, Maroko PR: The reduction of infarct 
size—an idea whose time (for testing) has come. Circula-
tion 50: 206-209, 1974
myocardial perfusion after intracoronary injection of ra-
8. Mallory GK, White PD, Salcedo-Salgar J: The 
speed of healing of myocardial infarction: A study of the 
pathologic anatomy in seventy-two cases. Am Heart J 18: 
647-671, 1939
chemical method for morphologic diagnosis of early stages 
10. Cannon PJ, Dill RB, Dwyer EM: Regional myoc-
dardial perfusion rates in patients with coronary artery dis-

ERRATUM


Under the heading “Relationship between TAR and $\Phi$” the paragraph beginning “TAR is ex-
tensively used for dose . . .” should read as follows:

“TAR is extensively used for the dose computations in beam therapy (4). For point isotropic 
sources, the tissue-to-air ratio can be defined as

$$\text{TAR} = \frac{\text{Dose to a small mass of tissue in phantom from}}{\text{a point isotropic source}}$$

Dose to the same mass of the tissue in free space from the same source

Consequently, Equations 1 to 4 appearing on page 492 should be redesignated as 2 to 5, respectively.