

Myocardial Dosimetry of ^{99m}Tc -Pyrophosphate

Identification of myocardial infarcts by radionuclide imaging using ^{99m}Tc -Sn-pyrophosphate (PP_1) or related compounds is under active investigation (1-3). As with any new procedure involving ionizing radiation, complete dosimetry should be performed. The purpose of this article is to estimate by calculation the absorbed radiation dose to the myocardium for infarctions of various sizes, assuming an injection of 15 mCi of PP_1 .

The classic equation for absorbed dose using the concept of absorbed fraction is

$$D(v_1 \leftarrow v_2) = \bar{A}_2 / M_1 \Sigma_i \Delta_i \phi_i (v_1 \leftarrow v_2), \text{ rad}, \quad (1)$$

$$\bar{A}_2 = A \cdot T \cdot F \cdot M_2, \quad \mu\text{Ci-hr}, \quad (2)$$

where A is the initial activity injected, F is the fractional uptake (assumed to be constant for this calculation) per unit mass of source volume, M_2 is the mass of the source volume v_2 , T is the mean life of radionuclide, 8.64 hr (assuming an infinite biologic half-time), M_1 is the mass of the target volume v_1 , ϕ_i is the absorbed fraction of the i^{th} component of radiation, and Δ_i is the equilibrium dose constant of the i^{th} component of radiation.

The dose to the myocardium was divided into two components, the dose to the normal heart tissue and the dose to the infarcted tissue. Each of these components was calculated assuming irradiation from five sources: (A) normal heart tissue, (B) infarcted heart tissue, (C) the skeleton, (D) the bladder, and (E) a uniform body distribution. The total dose to the myocardium is then given by Eq. 1, summed over all of these contributions.

In calculating the dose to normal heart tissue from irradiation by normal heart tissue, the value for F is chosen as $4.9 \times 10^{-6}/\text{gm}$ on the basis of animal studies (4). In addition, M_1 is chosen to be equal to $M_2 = 600 \text{ gm}$. The values of Δ for the K x-rays, gamma rays, and conversion electrons from technetium are 3.1, 264.4, and 36.2 $\text{gm} \cdot \text{rad}/\text{mCi} \cdot \text{hr}$, respectively.

Values for the absorbed fraction ϕ are obtained by assuming the heart to be a thick ellipsoid with axes of 1:0.667:1.333. Brownell et al. (6) have tabulated values of ϕ for various ellipsoid masses. The energy of the x-rays/gamma rays is considered to be the average of the energies of all the x-rays/gamma rays, weighted by their respective numbers per disintegration. The average gamma-ray energy is 140.5 keV, which results in a ϕ_γ (nor \leftarrow nor) of 0.106 (where nor = normal). This value, corrected for backscattered radiation (Table 12, Ref. 5), becomes 0.127. The average energy of the x-rays is low (18.73 keV) and extrapolation of the published data gives a value of 0.808 for ϕ_x (nor \leftarrow nor) after correction for backscatter.

Substitution of the assigned values into Eq. 1 yields a dose of 0.046 rad for normal heart tissue irradiating normal heart tissue. The source volume for this calculation actually included both normal and infarcted masses and the estimate is therefore high. The magnitude of the error would depend on the geometry and mass of the infarct, but it is estimated the error would be only 10% high for a 100-gm infarct in a 600-gm heart.

The photon dose resulting from the infarction's irradiation of normal tissue was computed from integration of specific absorbed fractions (6) using a HP9830A calculator. For an 8-gm infarction (inf), located at the apex of the heart, the dose contribution would be

$$D_p (\text{nor} \leftarrow \text{inf}) = 0.001 \text{ rad}. \quad (3)$$

The size and configuration of the infarct was selected to simplify the computer calculation.

It has been suggested (7) that out of the total amount of radionuclide injected, approximately 50% is taken up by the skeleton, about 40% by the bladder, and the remaining 10% is more or less uniformly distributed throughout the body. These values are substituted into Eq. 1 with $F \times M_2$ set equal to the appropriate fractional uptake; this yields 0.052 rad from the skeleton and 0.001 rad from the bladder.

In calculating the dose to the heart from a uniform body distribution, the fraction of dose to the heart from uniform distribution within the heart was subtracted from the total, thus avoiding redundancy. Also skeleton and bladder contributions were removed from this calculation. Uniform body dose contribution was 0.014 rad.

The total dose to the heart tissue from irradiation outside the heart is 0.067 rad. The total dose to the normal heart tissue is then

$$D_{\text{total}} (\text{normal} \leftarrow) = 0.114 \text{ rad}. \quad (4)$$

Calculations similar to those shown above were performed in order to estimate the dose to the infarcted heart tissue. Because such a dose is strongly dependent on the infarct mass, however, calculations were carried out on three sizes of infarcts: 2, 50, and 100 gm.

The infarcted area was assumed to be a flat ellipsoid with axes (1:3:8). This model for an infarct was selected because of published absorbed fraction data (8), and it seems to be reasonable, especially for large infarcts. For uptake by the infarct a value of $F = 4.7 \times 10^{-5} \text{ gm}^{-1}$, determined from animal studies, was chosen (4). The values of ϕ for the indicated infarct sizes, corrected for backscatter, were determined from Refs. 5 and 8.

The photon dose (x-rays and gamma rays) from infarcted tissue irradiating infarcted tissue becomes:

Infarct mass	D_p (inf \leftarrow inf)
2 gm	0.021 rad
50 gm	0.076 rad
100 gm	0.097 rad

The dose from electrons is independent of the infarct mass, and the calculated value is

$$D_e (\text{inf} \leftarrow \text{inf}) = 0.221 \text{ rad}. \quad (5)$$

The photon dose resulting from the normal heart tissue irradiating infarcted tissue is a reverse calculation of that used to obtain Eq. 3; the result for an 8-gm infarct is

$$D_p (\text{inf} \leftarrow \text{nor}) = 0.010 \text{ rad}. \quad (6)$$

The total dose to the infarcted muscle can therefore be considered to lie in the range of

$$D_{\text{total}}(\text{inf} \leftarrow) = 0.319-0.395 \text{ rad}, \quad (7)$$

where the precise value will depend on infarct size and uptake.

It has been shown (2) that often the distribution of uptake within infarcted muscle is nonuniform, with decreased uptake toward the center. This would have little or no effect on D_p (nor \leftarrow inf) or D_p (inf \leftarrow nor), but it would alter the total dose to the infarct from the sources within the infarct. The contribution to dose from photon irradiation would be reduced because more photons would escape the infarct volume without interacting. The electron dose would be increased toward the periphery. The average total infarct dose would be decreased over the case of uniform uptake, making our previous estimate conservative.

We note that several assumptions were made in performing the above dose calculations. In selecting a value for F from Ref. 4, two assumptions were necessary. First, from Ref. 3 it was noted that all human patients were scanned between 48 and 96 hr after onset; accordingly, the F value for animal uptake at 84 hr after onset was used. Second, it was assumed that this value of F determined at 1 hr after injection was a constant in time. The value chosen for F from Ref. 4 was further corrected to that for an average human by multiplying by the ratio of the estimated weight of the experimental animal (20 kg) to the weight of the human model (70 kg).

Finally, in all calculations the biologic half-time was assumed to be infinite and distributional uptake was assumed to be instantaneous. The assumption of infinite biologic half-time is incorrect for the bladder, but its contribution to the heart dose is so small that the error introduced by this assumption is negligible. The assumptions applied to the total-body, skeletal, and cardiac sources are believed to change the resulting calculated dose only slightly for different physiologic states in the patient.

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Biologic Half-Time of ^{125}I -Fibrinogen

We would like to comment on a recent paper (1) concerning the biologic half-time ($T_{1/2}$) of ^{125}I -fibrinogen in the detection of deep-vein thrombosis. Hardy and Newble calculated the $T_{1/2}$ of circulating ^{125}I -fibrinogen from precordial counting measurements in a small number of medical and surgical patients. Deep-vein thrombosis (DVT) in the legs was simultaneously diagnosed. We similarly believe that the $T_{1/2}$ of ^{125}I -fibrinogen can be useful in the diagnosis of DVT, but only when other clinical factors with significant influence on the $T_{1/2}$ are not present.

We have studied 153 surgical patients postoperatively with ^{125}I -fibrinogen. We suggest that the $T_{1/2}$ of ^{125}I -fibrinogen can be measured by a precordial counting technique, simultaneously with the detection of DVT in the legs. The purpose of our study was to examine possible influence of different clinical factors on the $T_{1/2}$. The diagnosis of DVT was correlated with the $T_{1/2}$ and the investigated clinical factors. Multiple regression analysis (Table 1) indicated the clinical factors that influence the $T_{1/2}$. The degree of the significant influence is expressed in percent of relative explanation. Our results may also indicate the occurrence of disseminated intravascular coagulation (DIC) in malignant diseases and that surgery as such also induces a DIC, showed by a shortened $T_{1/2}$. While blood transfusions had no significant influence on the $T_{1/2}$, significantly increased frequency of DVT was found in patients for whom the operation time had exceeded 100 min, compared with those who did not receive blood transfusions.

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TABLE 1. MULTIPLE REGRESSION ANALYSIS

Parameters	Relative explanation (%)
Operation time	10
Malignancy not removed and metastases	8
Infections	5
Deep-vein thrombosis	4
No significant parameters	3