

Internal Dosimetry Primer

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Abstract

The various ICRP and MIRD internal dosimetry models are similar in terms of their assumptions and defining equations. This similarity is obscured by differing terminology and notation, and these differences contribute to confusion in understanding these models. Emphasizing the definition of absorbed dose and using this definition to illustrate the MIRD and ICRP terminology and notation minimizes the confusion. The approach outlined in this article has been successfully utilized in the author's certification review courses.

Key Words

ABHP certification examination
Basic health physics principles
ICRP and MIRD models
Internal dosimetry

Introduction

Students preparing for both parts of the American Board of Health Physics Certification Examination are often overwhelmed and sometimes confused by the wealth and diversity of internal dosimetry models and terminology. International Commission on Radiological Protection (ICRP) and Medical Internal Radiation Dose (MIRD) methodologies are the most commonly used internal dosimetry models.

Contemporary internal dosimetry models began with the single compartment models of ICRP 2 and 10^[1-3] (ICRP 2/10). The MIRD Methodology^[4] and ICRP 26 and 30^[5,6] (ICRP 26/30) developed the concept of source and target organs. ICRP 60 and supporting publications including ICRP 66^[7,8] (ICRP 60/66) continue to refine the internal dosimetry methodology. In this article, we use the notation ICRP 2/10, ICRP 26/30, and ICRP 60/66 to refer to the defining internal dosimetry publications and supporting documents. Additional refinement is planned as part of the 2005/2006 ICRP recommendations and supporting publications.^[9]

In this article, the essential elements of internal dosimetry are presented. The presentation begins by defining the key elements of the MIRD and ICRP models in terms of the absorbed dose. With the key elements established, the MIRD and ICRP methodologies are presented in additional detail. This presentation has been successfully used in the author's certification review courses.

Overview of Internal Dosimetry Models

As an introduction to the MIRD and ICRP internal dosimetry models, we calculate the absorbed dose rate following the intake of radioactive material. If an isolated (single compartment) organ having a mass m , contains an activity $q(t)$ of radioactive material that emits radiation of energy E per disintegration, then the initial absorbed dose rate (\dot{D}_0) to this organ is:

$$\dot{D}_0 = k \frac{q(0)E}{m} \quad (\text{Eq. 1})$$

where k is a constant and $q(0)$ is the initial activity in the organ. If $q(0)$ is expressed in μCi , E in $\text{MeV/disintegration}$, and m in grams, then^[10]

$$k = 2.13 \text{ (rad/h)}(g\text{-dis/MeV-}\mu\text{Ci}) \quad (\text{Eq. 2})$$

The dose rate as a function of time t , can be written in terms of the initial absorbed dose rate:

$$\dot{D}(t) = \dot{D}_0 \exp(-\lambda_{\text{eff}} t) \quad (\text{Eq. 3})$$

where λ_{eff} is the effective removal rate from the organ and

$$\lambda_{\text{eff}} = \lambda_p + \lambda_b \quad (\text{Eq. 4})$$

In Equation 4, λ_p is the physical removal rate (disintegration constant) and λ_b is the biological removal rate. The removal rates are related to their respective half-lives (T) through the relationship:

$$\lambda = \ln(2)/T \quad (\text{Eq. 5})$$

The absorbed dose (D) is the integral of the dose rate with respect to time. Using Equations 1 and 3 leads to an expression for the absorbed dose:

$$D = \int_0^T \dot{D}(t) dt = \int_0^T \dot{D}_0 \exp(-\lambda_{\text{eff}} t) dt = \int_0^T k \frac{q(0)E}{m} \exp(-\lambda_{\text{eff}} t) dt \quad (\text{Eq. 6})$$

Equation 6 is simplified by recognizing that only the activity in the organ varies with time:

$$D = \frac{kE}{m} \int_0^T q(0) \exp(-\lambda_{\text{eff}} t) dt \quad (\text{Eq. 7})$$

Equation 7 can be compared to the basic equations for internal dose within the MIRD (Equation 8) and ICRP (Equation 9) methodologies:

$$\bar{D} = \tilde{A} S \quad (\text{Eq. 8})$$

$$H_{50,T} = 1.6 \times 10^{-10} \frac{\text{Sv-g}}{\text{MeV}} U_S SEE \quad (\text{Eq. 9})$$

where \bar{D} is the mean absorbed dose (rad), \tilde{A} is the total cumulated activity ($\mu\text{Ci-h}$), S is the mean dose per unit cumulated activity ($\text{rad}/\mu\text{Ci-h}$), $H_{50,T}$ is the 50-year committed dose equivalent (Sv), U_S is the number of transformations in the source organ over 50 years (trans), and SEE is the specific effective energy (MeV/g-trans).

Comparing Equation 7 to Equations 8 and 9 leads to the explicit identifications summarized in Table 1. Table 1 illustrates that the ICRP and MIRD methodologies are essentially equivalent. With the exception of terminology, the major difference is in the upper limit of integration (i.e., $T = 50 \text{ y}$ for the ICRP and $T = \infty$ for MIRD). Additional discussion regarding the MIRD and ICRP methods is provided in References 4, 10, 11, and 12.

Table 1. Comparison of the MIRD and ICRP Models

Eq. 7 Term	Corresponding Quantities	
	MIRD	ICRP
D	\bar{D}	$H_{50,T}$
kE/m	S	$1.6 \times 10^{-10} \frac{\text{Sv-g}}{\text{MeV}} SEE$
T	∞	50 y
$\int_0^T q(0) \exp(-\lambda_{\text{eff}} t) dt$	\tilde{A}	U_S

Equations 7 – 9 and the comparisons of Table 1 illustrate the inherent consistency of the internal dosimetry models. With this basic consistency established, model specific aspects can be presented. These aspects should be periodically reviewed with regard to Table 1 to simplify and unify the presented concepts.

MIRD Methodology

The Committee on Medical Internal Radiation Dose of the Society of Nuclear Medicine developed a methodology to perform radiation absorbed dose calculations. These calculations are performed to assess the risks associated with the administration of radiopharmaceuticals for medical studies including imaging, therapy, and metabolic applications.

The MIRD technique is a computational methodology that facilitates absorbed dose calculations to specified target organs from radioactive decays that occur in source organs. The source organs contain the radioactive material, and the target is the organ in which the dose is calculated. The target and source organs can be the same tissue. In subsequent discussion, the terms tissue and organ are used interchangeably.

To specify the MIRD methodology, it is necessary to define several terms. The mean energy emitted per transition (Δ), in Gy-kg/Bq-s or rad-g/ μ Ci-h, is given as the product of the mean particle energy (E) in MeV or joules, and the number of particles per nuclear transformation (n):

$$\Delta = K E n \quad (\text{Eq. 10})$$

where K is a conversion factor. Within the MIRD methodology, particles are defined to be photons, beta particles, or positrons. These are the radiation types used most frequently in nuclear medicine procedures.^[4,10-12]

The cumulated activity or the total number of nuclear transitions occurring within the source organ from time $t = 0$ to time T is:

$$\tilde{A} = \int_0^T A(t) dt \quad (\text{Eq. 11})$$

The activity as a function of time is:

$$A(t) = A(0) \exp(-\lambda_{\text{eff}} t) \quad (\text{Eq. 12})$$

Using Equation 12, the cumulated activity is significantly simplified if the MIRD upper integration limit ($T=\infty$) is selected. In this case, the total cumulated activity is:

$$\tilde{A} = \frac{A(0)}{\lambda_{\text{eff}}} = \frac{A(0)T_{\text{eff}}}{\ln(2)} = 1.44T_{\text{eff}}A(0) \quad (\text{Eq. 13})$$

where

$$T_{\text{eff}} = \frac{T_p T_b}{T_p + T_b} \quad (\text{Eq. 14})$$

The initial activity in the organ, $A(0)$, is not the intake activity but only a fraction (f_2) of the total activity of the radionuclide in the body (q):

$$A(0) = f_2 q(0) \quad (\text{Eq. 15})$$

where f_2 is the fraction of the intake activity reaching the organ of interest.

The total energy emitted by the source organ is the product of Δ and the cumulated activity. However, only a fraction (f) of this energy will be deposited in the target organ, which is the location of interest in the dose calculation. With these quantities and a knowledge of the mass of the target organ (m), the mean absorbed dose \bar{D} can be defined as:

$$\bar{D} = \frac{\tilde{A} \Delta f}{m} \quad (\text{Eq. 16})$$

The MIRD methodology also defines the specific absorbed fraction (F):

$$F = \frac{f}{m} \quad (\text{Eq. 17})$$

where f is the energy absorbed by the target divided by the energy emitted by the source. The specific absorbed fraction represents the mean target dose per unit energy emitted by the source. Therefore, the mean absorbed dose can be written as:

$$\bar{D} = \tilde{A} \Delta F \quad (\text{Eq. 18})$$

The MIRD methodology defines the mean dose to the target (T) per unit cumulated activity in the source (S) in $m\text{Gy}/\text{MBq}\cdot\text{s}$ or $\text{rad}/\mu\text{Ci}\cdot\text{h}$:

$$S(T \leftarrow S) = \frac{\Delta f}{m} = \Delta F \quad (\text{Eq. 19})$$

Equations 18 and 19 permit the expected MIRDOSE relationship to be written:

$$\bar{D} = \tilde{A} S(T \leftarrow S) \quad (\text{Eq. 20})$$

In Equation 20, the metabolic factors are contained in the \tilde{A} term, which depends on uptake by the source organ and biological elimination of the radiopharmaceutical by the source organ. The S factor represents the physical decay characteristics of the radionuclide, the range of the emitted radiations, and the organ size and configuration. If a standard anatomy is utilized, S can be calculated and tabulated for a variety of radionuclides and source-target combinations. MIRDOSE Pamphlet No. 11^[12] provides a tabulation of these S factors.

ICRP Methodology

The ICRP internal dosimetry models are based in part on evolving assessments of the biological effects of ionizing radiation. These assessments impact the selection of model organs/tissues and their weighting factors. The biological data and organ models lead to recommendations regarding occupational exposures. Each of these ICRP model aspects will be reviewed in subsequent sections of this article.

The specific ICRP recommendations have been incorporated into national and international regulations. For example, ICRP 26/30 form the basis for the US ionizing radiation regulations (10 CFR 20^[13] and 10 CFR 835^[14]), and ICRP 60/66 are the basis for current international regulations.

Biological Effects

The ICRP models should be viewed in their historical context. The models continue to evolve and incorporate available data regarding the biological effects of ionizing radiation.

A portion of the scientific basis for ICRP 26/30 and ICRP 60/66 are summarized in Table 2. ICRP 26/30 are based in part on the Biological Effects of Ionizing Radiation (BEIR) III Report.^[15] In BEIR III, the dose response relationships for both solid tumors and leukemia are assumed to have a linear-quadratic (LQ) relationship:

$$f(d) = ad + bd^2 \quad (\text{Eq. 21})$$

where $f(d)$ is the effect of the radiation exposure, d is the dose equivalent, and a and b are risk coefficients. BEIR III based its preferred age-specific cancer model on the absolute (additive) risk model:

$$r(d) = r_o + f(d)g(\beta) \quad (\text{Eq. 22})$$

where $r(d)$ is the number of cancers of a specific type in the population group, r_o is the natural incidence of the specific cancer type, and $g(\beta)$ is the excess risk function that contains the time dependence.^[10,15]

BEIR V^[16] forms a portion of the basis for ICRP 60/66. In BEIR V, the dose response model is linear (L) for solid tumors:

$$f(d) = cd \quad (\text{Eq. 23})$$

Table 2. Comparison of the Basis for Recent ICRP Models

ICRP Model	Basis	Dose Response Relationship ^a		Risk Model
		Solid Tumors	Leukemia	
26/30	BEIR III	LQ	LQ	Absolute
60/66	BEIR V	L	LQ	Relative

^a L = Linear and LQ = Linear-Quadratic

and linear-quadratic for leukemia. In Equation 23, c is a risk coefficient. In contrast to BEIR III, BEIR V uses a relative (multiplicative) risk model:

$$r(d) = r_o[1 + f(d)g(\beta)] \quad (\text{Eq. 24})$$

Both BEIR III and BEIR V assume the dose response models have no threshold. That is, any dose no matter how small, has an effect (detriment).

There are significant differences between the BEIR III and BEIR V estimates. Table 3 illustrates the variation in both leukemia and nonleukemia (solid tumor) cancers. The solid tumors include respiratory, digestive, breast, and other cancer types. For leukemia, BEIR V leads to a factor of 4-5 greater risk. A similar increase of about 3-5 occurs for nonleukemia cancers if BEIR III relative risk models are considered.

Considerably larger factors of 11-19 occur for nonleukemia cancers if the BEIR III absolute risk model is compared to BEIR V's relative risk model. Because recent data^[16] support the relative risk model, these large factors do not appear to be reasonable. However, the BEIR VII Report^[17] is yet to be published and may offer a different view.

ICRP 26/30 and ICRP 60/66 Terminology

ICRP 26/30 and ICRP 60/66 utilize different terminology to describe similar quantities. Table 4 summarizes the terminology appropriate to each model. The specific terms are defined in subsequent sections of this article.

ICRP 26 and ICRP 60 Recommendations

Prior to reviewing specific ICRP internal dose formalism, the ICRP 26 and ICRP 60 recommendations are outlined. This is important because these recommendations and the internal dose formulation are closely related. The ICRP recommendations are based on the following two general principles:

- Preventing the occurrence of clinically significant radiation induced deterministic effects.
- Limiting the risk of stochastic effects to a reasonable level.

The National Council on Radiation Protection and Measurement (NCRP)^[18] also adopts these two general principles. In addition, the NCRP recommends that risk be limited over a working lifetime to be no greater than the risk of accidental death in a safe industry.

The deterministic effects may have a threshold. The term "deterministic effect" was introduced in ICRP 60. Deterministic effects include erythema, cataracts, impairment of fertility, and depletion of blood-forming cells in bone marrow. These effects only occur in irradiated individuals. Keeping the dose below the threshold for the deterministic effect can eliminate it. With deterministic effects, the severity of the effect varies with dose. ICRP 26 refers to deterministic effects as non-stochastic effects.

Stochastic effects include cancer and hereditary effects. These effects occur in the general population as well as in irradiated individuals. With stochastic effects, the probability of the effect increases with increasing dose without threshold.

With ICRP 26, these recommendations are implemented by limiting the effective dose equivalent and committed dose equivalent, and by establishing stochastic and non-stochastic annual limits on intake (ALIs). Considering the purpose of this article, the applicable recommendations are summarized in Table 5. In Table 5, the annual doses (deep dose equivalent), eye dose equivalent, and skin dose equivalent are evaluated at 1,000 mg/cm², 300 mg/cm², and 7 mg/cm², respectively.

Table 3. Lifetime Cancer Risk Estimates (Deaths per 100,000 persons) ^a

Cancer Type	Continuous Lifetime Exposure 1 mGy/year		Instantaneous Exposure 0.1 Gy	
	Male	Female	Male	Female
Leukemia				
BEIR III	15.9	12.1	27.4	18.6
BEIR V	70	60	110	80
BEIR V/BEIR III	4.4	5.0	4.0	4.3
Nonleukemia				
BEIR III (absolute)	24.6	42.4	42.1	66.5
BEIR III (relative)	92.9	118.5	192	213
BEIR V (relative)	450	540	660	730
BEIR V/BEIR III (relative)	4.8	4.6	3.4	3.4
BEIR V/BEIR III (absolute)	18.3	12.7	15.7	11.2

^a Derived from Table V.3 Ref. 10.

Table 4. Terminology Utilized in Recent ICRP Models

ICRP Model	Terminology	
	Organ Dose	Whole Body Dose
26/30	Committed Dose Equivalent ($H_{50,T}$)	Effective Dose Equivalent ^a (H_E)
60/66	Equivalent Dose (H_T)	Effective Dose (E)

^a US regulations use the term committed effective dose equivalent.

Table 5. Applicable ICRP 26 and ICRP 60 Recommendations

Dose Recommendation	Dose (mSv)	
	ICRP 26	ICRP 60
Annual	50 ^a	50 maximum ^b
Cumulative	none	100 over 5 years ^b 20 /y average ^b
Eye	150 ^c	150 ^d
Skin, Hands, and Feet	500 ^c	500 ^d

^a Effective dose equivalent
^b Effective dose
^c Committed dose equivalent
^d Equivalent dose

In ICRP 60, the restrictions on effective dose are sufficient to ensure the avoidance of deterministic effects in all body tissues except the lens of the eye and the skin. The limits for the eye and skin preclude deterministic effects. Therefore, only a stochastic ALI is needed in the ICRP 60/66 internal dosimetry formulation.

Calculation of Internal Dose Equivalents Using ICRP 26/30

Internal dose equivalents may be calculated in a variety of ways. These include the use of the annual limit on intake (ALI), derived air concentration (DAC), and SEE and U_S values.

Within the ICRP 26/30 methodology, the stochastic and nonstochastic recommendations for internal dose equivalents are developed in terms of the ALI. Following ICRP 26/30, the ALI is defined to be the largest value of intake that satisfies both of the inequalities of Equations 25 and 26. In Equations 25 and 26, ALI_S is the stochastic ALI and ALI_{NS} is the nonstochastic ALI:

$$ALI_S \sum_T w_T H'_{50,T} \leq 0.05Sv \quad (\text{Eq. 25})$$

for stochastic effects

$$ALI_{NS} H'_{50,T} \leq 0.5Sv \quad (\text{Eq. 26})$$

for non-stochastic effects

where w_T is the ICRP 26/30 organ/tissue weighting factor and $H'_{50,T}$ is specified as the dose per unit intake (Sv/Bq) which yields the correct units for the ALI. The organ/tissue weighting factors for ICRP 26/30 and 60/66 are summarized in Table 6.

ICRP 26/30 form the basis of the current domestic regulations embodied in 10 CFR 20^[13] for U.S. Nuclear Regulatory Commission licensees and 10 CFR 835^[14] for U.S. Department of Energy licensees. These regulations require the calculation of individual organ doses (i.e., the committed dose equivalent (CDE)) and the committed effective dose equivalents (CEDE) that are based on the risk of dose to the various organs/tissues included in the ICRP 26/30 model. The CDE and CEDE are calculated in terms of the intake (I) as follows:

Table 6. Weighting Factors for Recent ICRP Models

Organ or Tissue	ICRP 26/30	ICRP 60/66
Gonads	0.25	0.20
Breast	0.15	0.05
Red Bone Marrow	0.12	0.12
Lung	0.12	0.12
Thyroid	0.03	0.05
Bone Surfaces	0.03	0.01
Stomach	--	0.12
Colon	--	0.12
Esophagus	--	0.05
Bladder	--	0.05
Skin	--	0.01
Liver	--	0.05
Remainder	0.30 ^a	0.05 ^b

^a Five highest other organs.
^b Adrenals, brain, small intestine, spleen, kidneys, muscle, pancreas, upper large intestine, thymus, and uterus.

$$\begin{aligned}
 \text{CDE} &= H_{50,T} \\
 &= \frac{I}{\text{ALI}_{\text{NS}}} 0.5\text{Sv} \\
 &= 1.6 \times 10^{-10} \frac{\text{Sv} \cdot \text{g}}{\text{MeV}} U_{\text{S}} \text{SEE}(T \leftarrow S)
 \end{aligned}
 \tag{Eq. 27}$$

$$\begin{aligned}
 \text{CEDE} &= H_{\text{E}} \\
 &= \sum_T w_T H_{50,T} \\
 &= \sum_T \frac{I}{\text{ALI}_{\text{S}}} 0.05\text{Sv}
 \end{aligned}
 \tag{Eq. 28}$$

Equations 27 and 28 can also be rewritten in terms of the derived air concentration (DAC)

$$\text{DAC} = \text{ALI}/2400 \text{ m}^3 \tag{Eq. 29}$$

Calculation of Equivalent and Effective Doses Using ICRP 60/66

Within the ICRP 60/66 formalism, two new dose terms are introduced. These are the equivalent dose and the effective dose. The equivalent dose (H_T) is defined as:

$$H_T = \sum_R w_R D_{T,R} \tag{Eq. 30}$$

where w_R is the radiation weighting factor and $D_{T,R}$ is the average absorbed dose in tissue T due to radiation of type R . The ICRP 60/66 radiation weighting factors are provided in Table 7.

The effective dose (E) is defined as:

$$E = \sum_T w_T H_T \tag{Eq. 31}$$

Using Equation 30, the effective dose can be written as:

$$E = \sum_R w_R \sum_T w_T D_{T,R} = \sum_T w_T \sum_R w_R D_{T,R} \tag{Eq. 32}$$

Within ICRP 60/66, only one ALI is defined. The committed effective dose $E(50)$ is written as:

$$\begin{aligned}
 E(50) &= \frac{I}{\text{ALI}} 0.02\text{Sv} \\
 &= \sum_{T=1}^{12} w_T H_T(50) + w_{\text{remainder}} \frac{\sum_{T=13}^{22} m_T H_T(50)}{\sum_{T=13}^{22} m_T}
 \end{aligned}
 \tag{Eq. 33}$$

where $H_T(50)$ is the committed equivalent dose, m_T is the mass of the remainder tissue, and $w_{\text{remainder}} = 0.05$. In Equation 33, the first sum is over the 12 organs/tissues with assigned weighting factors (See Table 6) and the second sum is over the 10 remainder organs/tissues (i.e., adrenals, brain, small intestine, spleen, kidneys, muscle, pancreas, upper large intestine, thymus, and uterus). The right hand side of Equation 33 is applicable whenever one of the 12 organs with assigned weighting factors has the largest committed equivalent dose. In the exceptional case in which one of the remainder organs receives a committed equivalent dose in excess of the highest committed equivalent dose in any of the 12 organs for which a weighting factor is assigned, a weighting factor of 0.025 should be

Table 7. Radiation Weighting Factors^a

Type and Energy Range ^b	Radiation Weighting Factor
Photons (all energies)	1
Electrons and muons (all energies) ^c	1
Neutrons:	
< 10 keV	5
10 keV – 100 keV	10
> 100 keV – 2 MeV	20
> 2 MeV – 20 MeV	10
> 20 MeV	5
Protons, other than recoil protons (> 2 MeV)	5
Alpha particles, fission fragments, and heavy nuclei	20
^a All values relate to the radiation incident on the body or, for internal sources, emitted from the source. ^b The choice of values for other radiation types is discussed in Annex A, ICRP 60. ^c Excluding Auger electrons emitted from nuclei bound to DNA.	

applied to that remainder organ or tissue. A weighting factor of 0.025 should also be assigned to the average dose in the rest of the remainder, and the $E(50)$ equation has the form shown in Equation 34 (below), where $m_{T'}$ is the mass of the remainder tissue or organ in which the committed equivalent dose is calculated to be higher than in any of the 12 specified tissues/organs with assigned weighting factors and $H_{T'}(50)$ is the committed equivalent dose in that remainder tissue/organ.

The careful reader will note that the first term in Equation 33 contains no ALI subscript since the ICRP 60/66 formulation only utilizes a stochastic ALI. The 0.02 Sv (20 mSv) multiplier is a direct consequence of the Table 5 cumulative effective dose recommendation.

Model Dependence

Equations 25 – 34 and Tables 3 and 6 illustrate the model dependence of ICRP 26/30 and 60/66. The selection of the tissues, governed by a host of inherent model assumptions and historical data, dictates the dose result. An examination of Table 6 and the differences in the number of listed tissues, their associated weighting factors, and the treatment of the remainder illustrate the evolving nature of the ICRP internal dosimetry models.

$$E(50) = \sum_{T=1}^{12} w_T H_T(50) + 0.025 H_{T'}(50) + 0.025 \frac{\sum_{T=13}^{22} m_T H_T(50) - m_{T'} H_{T'}(50)}{\sum_{T=13}^{22} m_T - m_{T'}} \quad (\text{Eq. 34})$$

Conclusions

Using the concept of absorbed dose, the MIRD and ICRP internal dosimetry models are found to be quite similar. The ICRP general principles and the supporting biological effects of ionizing radiation publications affect the specific model formulations. With the exception of terminology, the ICRP methodology remains consistent with the definition of absorbed dose with model refinements being influenced by evolving assessments of the biological effects of ionizing radiation.

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