
A Report of the American College of Cardiology Task Force on Expert Consensus Decision Pathways

Developed in Collaboration With Mended Hearts

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**ABSTRACT**

The stimulus to create this document was the recognition that ionizing radiation-guided cardiovascular procedures...
are being performed with increasing frequency, leading to
greater patient radiation exposure and, potentially, to
greater exposure for clinical personnel. Although the
clinical benefit of these procedures is substantial, there is
concern about the implications of medical radiation
exposure. The American College of Cardiology leadership
concluded that it is important to provide practitioners
with an educational resource that assembles and inte-
ports the current radiation knowledge base relevant to
cardiovascular procedures. By applying this knowledge
base, cardiovascular practitioners will be able to select
procedures optimally, and minimize radiation exposure
to patients and to clinical personnel.

Optimal Use of Ionizing Radiation in Cardiovascular
Imaging: Best Practices for Safety and Effectiveness is a
comprehensive overview of ionizing radiation use in
cardiovascular procedures and is published online. To
provide the most value to our members, we divided the
print version of this document into 2 focused parts. Part
I: Radiation Physics and Radiation Biology addresses
the issue of medical radiation exposure, the basics of radia-
tion physics and dosimetry, and the basics of radiation
biology and radiation-induced adverse effects. Part II:
Radiological Equipment Operation, Dose-Sparing Meth-
odologies, Patient and Medical Personnel Protection
covers the basics of operation and radiation delivery for
the 3 cardiovascular imaging modalities (x-ray fluores-
copy, x-ray computed tomography, and nuclear
scintigraphy).

PREAMBLE

This document has been developed as an Expert
Consensus Document by the American College of Cardi-
ology (ACC) in collaboration with the American Society of
Nuclear Cardiology, Heart Rhythm Society, Mended
Hearts, North American Society for Cardiovascular Imag-
ing, Society for Cardiovascular Angiography and In-
terventions, Society for Cardiovascular Computed
Tomography, and Society of Nuclear Medicine and Mol-
ecular Imaging. Expert Consensus Documents are inten-
ded to inform practitioners, payers, and other interested
parties of the opinion of ACC and document cosponsors
concerning evolving areas of clinical practice and/or
technologies that are widely available or new to the
practice community. Topics chosen for coverage by expert
consensus documents are so designed because the evi-
dence base, the experience with technology, and/or clin-
ical practice are not considered sufficiently well
developed to be evaluated by the formal ACC/American
Heart Association practice guidelines process. Often the
topic is the subject of considerable ongoing investigation.

Thus, the reader should view the Expert Consensus
Document as the best attempt of the ACC and document
cosponsors to inform and guide clinical practice in areas
where rigorous evidence may not yet be available or evi-
dence to date is not widely applied to clinical practice.

To avoid actual, potential, or perceived conflicts of
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a chair with no relevant RWI. Authors with relevant RWI
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disclosed in Appendixes A and B, respectively. Addition-
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to this document—is available online. Disclosure informa-
tion for the ACC Task Force on Clinical Expert Consensus
Documents is also available online, as is the ACC disclosure policy for document
development.

James L. Januzzi, MD, FACC
Chair, ACC Task Force on Expert Consensus Decision Pathways

1. INTRODUCTION

1.1. Document Development Process and Methodology

1.1.1. Writing Committee Organization

The work of the writing committee was supported
exclusively by the ACC without commercial support.
Writing committee members volunteered their time to
this effort. Conference calls of the writing committee
were confidential and attended only by committee
members and ACC staff.

The writing committee consisted of a broad range
of members representing 9 societies and the following
areas of expertise: interventional cardiology, general
cardiology, pediatric cardiology, nuclear cardiology,
nuclear medicine, electrophysiology, cardiac computed
tomography, cardiovascular imaging, and the consumer patient perspective. Both a radiation safety biologist and physicist were included on the writing committee.

This writing committee met the College’s disclosure requirements for RWI as described in the Preamble.

1.1.2. Document Development and Approval
The Writing Committee convened by conference call and e-mail to finalize the document outline, develop the initial draft, revise the draft per committee feedback, and ultimately sign off on the document for external peer review. All participating organizations participated in peer review, resulting in 21 reviewers representing 299 comments. Comments were reviewed and addressed by the writing committee. A member of the ACC Task Force on Expert Consensus Decision Pathways served as lead reviewer to ensure that all comments were addressed adequately. Both the writing committee and the task force approved the final document to be sent to the ACC Clinical Policy Approval Committee. This committee reviewed the document, including all peer review comments and writing committee responses, and approved the document in November 2017. The Heart Rhythm Society, North American Society for Cardiovascular Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Cardiovascular Computed Tomography endorsed the document in January 2018. This document is considered current until the Task Force on Expert Consensus Decision Pathways revises or withdraws it from publication.

2. PURPOSE

2.1. Document Purpose
This print-published document is part 1 of an abbreviated version of a larger, more comprehensive document that is published concurrently online. The online version contains additional technical detail for readers who wish to understand a topic in greater depth. The online published document, in addition to covering the topics in the 2 print-published documents in greater depth, also covers additional topics not covered in the print-published documents including: 1) dose reduction strategies; 2) operator education and certification; 3) quality assurance; and 4) patient radiation tracking.

This document covers radiation physics, radiation dosimetry and its determinants, and radiation harm. The document’s purpose is to provide a comprehensive information source about ionizing radiation use in cardiovascular procedures. The writing group has assembled this information to assist cardiovascular practitioners to provide optimal cardiovascular care when employing ionizing radiation-based procedures. The goal is to enhance cardiovascular practitioners’ ability to select the optimal imaging technique for a given clinical circumstance while balancing a technique’s risk and benefits, and to apply that technique optimally to generate high-quality diagnostic images of greatest clinical value and minimal radiation exposure.

2.2. The Radiation Safety Issue
Cardiovascular procedures that employ ionizing radiation have great value for diagnosis and treatment of properly selected patients with known or suspected cardiovascular disease. However, ionizing radiation has molecular-level detrimental effects on tissue, with potential for injury both to patients and to exposed medical personnel. It is desirable to minimize radiation exposure both to patients and to medical personnel while achieving optimal benefits to health. This principle requires that clinicians employ judicious use and conduct of radiation-employing procedures.

Currently, cardiovascular diagnostic and therapeutic procedures are a major source of patient exposure to medical ionizing radiation, accounting for approximately 40% of total medical radiation exposure (exclusive of radiation oncology) (1,2). Among occupationally exposed healthcare workers, interventional cardiologists and clinical electrophysiologists are among the most highly exposed, and there is potential for exposure to support personnel as well (3,4).

2.3. The Need for Physician Radiation Safety Education
Cardiovascular specialists have a responsibility to:
1. Apply knowledge of the radiation safety knowledge base to make appropriate case selection choices.
2. Conduct radiation-assisted procedures optimally, minimizing exposure to patients and personnel.

There is evidence that many cardiovascular specialists who order and conduct radiation-employing procedures are not fully informed about the radiation doses that accompany the procedure or the associated health implications for their patients and for themselves (5,6).

2.4. Appropriateness of Medical Radiation
The balance between a procedure’s risk and benefit determines its appropriateness. The hazard associated with ionizing radiation is a potentially important determinant of a procedure’s risk-benefit relationship. Physicians who either order or conduct such procedures need to:
1. Know the magnitude of a patient’s risk associated with a procedure’s radiation exposure.
Despite these engineering refinements, the patient radiation doses that accompany these procedures remain substantial and, for the most part, are at the upper range of radiation-based diagnostic studies. Medical professionals should be aware of the radiation dose that these studies deliver to patients. In addition, within a particular type of study, the radiation dose can vary substantially depending on image acquisition protocol and patient characteristics. For reference, the commonly performed cardiovascular diagnostic studies and their radiation dose ranges are listed in Table 1. Note that the doses delivered by x-ray computed tomography (CT) and nuclear cardiology can vary substantially depending on particulars of image acquisition protocols.

Patient radiation dose ranges (in millisieverts) are listed for the 3 principal radiation-based cardiovascular imaging studies: x-ray fluoroscopy, x-ray computed tomography, and nuclear cardiology. Individual procedure categories are further subdivided according to types of image acquisition protocols. Note that for a particular procedure category, the dose can vary considerably depending on image acquisition protocol and, within a given image acquisition protocol, procedure conduct and patient characteristics.

However, augmented capabilities have led to increased utilization levels, necessarily accompanied by greater radiation exposure both at the individual and population levels.
levels. In addition, increasing complexity of cardiovascular interventional procedures requires longer fluoroscopic times and, accordingly, larger radiation exposures.

Natural background radiation averages 3.0 millisieverts (mSv) per person per year in the United States—equivalent to 150 posteroanterior chest radiographs (a posteroanterior chest-x-ray dose is 0.02 mSv; combined posteroanterior and lateral is 0.06 mSv) (8). At the population level, between 1987 and 2006 estimated per person total medical radiation exposure grew from 0.6 mSv/year (0.2 × background) to 3.2 mSv/year (1.07 × background) (9). Consequently, currently, patients are receiving, on average, more radiation from medical sources than from natural background sources. 2006 is the latest year for which compiled data are available. (The National Council on Radiation Protection is currently compiling contemporary data—expected availability 2019—and it is likely that current average medical exposure will be found to have increased further.) The 2006 medical exposure is equivalent to 160 posteroanterior chest x-rays per person per year. Risks associated with this exposure must be weighed in relation to the health status benefits achieved by these procedures.

Physicians who are invasive cardiovascular procedure operators are among the most highly occupationally exposed healthcare workers. Measurements of interventional cardiologist operator exposure using current equipment and protection practices demonstrate an exposure range of 0.2 to >100 microsieverts (μSv) per procedure with a per-procedure average of 8 to 10 μSv (10). Thus, an active interventional cardiologist performing 500 procedures/year employing current technology may be expected to receive, in addition to background exposure, a dose of as much as 10 mSv/year or, in a most extreme scenario, 300 mSv over a 30-year active professional career.

The potential implications of this level of medical radiation exposure are summarized in Table 2.

### 4. THE MANY MEASURES OF RADIATION

#### 4.1. Radiation Exposure and Dose Metrics

Ionizing radiation exposure and dosimetry are not easily characterized by simple metrics. For clarity, in this document the interaction of radiation with tissue will be characterized from the perspective of 5 inter-related frames of reference: exposure, absorbed dose, equivalent dose, effective dose, and injected dose. For this document’s purpose, these metrics have specific meanings as defined in the following text:

**Exposure:**
Radiation exposure refers to the presence of ionizing radiation at the location of the exposed tissue. The typically used measure of radiation quantity is air kerma, which is the amount of energy released by the interaction of the radiation with a unit mass of air. Its unit of measure is the gray (Gy) (J/kg). One Gy is the quantity of radiation that when interacting with 1 kg of air releases 1 J of energy.

**Absorbed Dose:**
Absorbed radiation dose is a measure of the energy that radiation deposits in an exposed tissue through interactions with its molecular constituents. It differs from exposure in that the radiation present at a given location does not deposit all of its energy there. The fraction of its energy that a given radiation exposure will deposit in the exposed tissue varies with the type and energy of the radiation, the tissue composition, and the exposure duration.

Absorbed dose is a measure of the intensity of energy deposition (energy deposited per unit mass of tissue) and is expressed in Gy–joules of energy deposited per kilogram of tissue.

**Equivalent Dose:**
Different types of ionizing radiation cause varying degrees of tissue injury for a given absorbed dose. Equivalent dose is a construct used to account for differences in tissue injury caused by different radiation types. X-rays and gamma rays are the benchmarks against which particle radiation types such as protons, neutrons, and beta particles are compared. To adjust for this variability, each radiation type is assigned a radiation weighting factor by which the absorbed dose (in Gy) is multiplied to yield a measure of the expected tissue injury caused by that dose. The unit of measure is the sievert (Sv), which is the absorbed dose in Gy multiplied by the radiation weighting factor. All radiation types used in cardiovascular medicine have a radiation weighting factor of 1.

**Effective Dose:**
Effective dose is a measure of the estimated potential for a stochastic biological effect (such as cancer induction) caused by a particular absorbed radiation dose. In medical

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**TABLE 2** Potential Consequences of Patient and Medical Personnel Radiation Exposure

<table>
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<tr>
<th>Category</th>
<th>Description</th>
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<tr>
<td>Individual Patient</td>
<td>Although many individual patients receive little or no medical radiation exposure, some receive lifetime doses in excess of 100 mSv. Doses in excess of 100 mSv are associated with a detectable increased cancer risk.</td>
</tr>
<tr>
<td>Population</td>
<td>Increased total exposure incurred by total population of patients has the potential to increase the population incidence of cancer and other radiation-related disorders.</td>
</tr>
<tr>
<td>Occupationally Exposed Workers</td>
<td>Occupationally exposed physicians and support staff may receive doses as large as 10 mSv per year over a career that may span 30–40 years. The implications of this level of exposure at the level of the individual practitioner are uncertain.</td>
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radiation exposures, absorbed dose is typically not uniform throughout all tissues. For x-ray imaging, dose is concentrated in the body region being examined and varies with depth from the beam entrance port. For nuclear imaging, dose is concentrated in the tissues that most avidly take up the tracer or are involved in its elimination.

Effective dose is the sum of the equivalent doses received by each organ, with each organ equivalent dose multiplied by a coefficient that reflects that organ’s sensitivity to a stochastic effect. The unit of effective dose is also the Sv. The Sv has the same unit as the Gy (J/kg).

The connection between effective dose and absorbed dose is that an effective dose of 1 Sv (which may be concentrated in only a few organs) confers the same estimated stochastic risk that would be caused by a uniform total absorbed body dose of 1 Gy of radiation that has a radiation weighting factor of 1.

Different tissues have different sensitivities to radiation-induced effects. In the effective dose construct, each tissue is assigned a tissue-weighting factor that specifies its sensitivity to radiation effects. To calculate the effective dose in Sv, each exposed tissue’s equivalent dose is multiplied by its tissue-weighting factor yielding that tissue’s contribution to the overall risk. The contributions to risk from all exposed tissues are summed yielding total risk, which is expressed as the effective dose in Sv.

**Injected Dose:**
Injected dose describes the quantity of radioactivity injected into a patient for a nuclear scintigraphy study (expressed in millicuries). The relationship between an injected dose and the previously described dose parameters is complex and is discussed in Part II: Radiological Equipment Operation, Dose-Sparing Methodologies and Protection.

**4.2. Challenges in Relating Radiation Exposure and Dose to Risk of Detrimental Effects**
Detrimental effects of radiation exposure typically present weeks to years following exposure. In addition, many detrimental effects, principally cancer, have a large background frequency. This complicates the attribution of an effect in a particular subject to prior radiation exposure.

**4.3. Types of Ionizing Radiation Used in Medical Imaging**
Radiation in cardiovascular imaging consists of photons with energy >10 kiloelectron volts (keV) (x-rays and gamma rays) and positrons. The physical effect of such radiation is to eject electrons from atoms forming ions and free radicals. This is the basis for the term “ionizing radiation.” The resulting ions and free radicals react with tissue molecules, damaging them.

**4.3.1. X-Rays and Gamma Rays**
X-ray and gamma ray photons travel at the speed of light and have no mass and no charge. Their electromagnetic energy ranges from a few electron volts (eV) to millions of electron volts (MeV). X-rays used in x-ray fluoroscopy and x-ray CT have a photon energy spectrum between 30 and 140 keV. Thallium-201 releases photons primarily in the 68-80 keV range, similar to diagnostic x-rays. Technetium-99m releases photons primarily in the 140 keV range.

**4.3.2. Positrons**
Positrons are positively charged electrons. They have mass and charge. When they travel through a medium, their electrostatic charge causes them to interact readily with electrons in the medium, leaving a trail of ionization. Consequently, they have a very short mean free path in tissue (6 to 7 mm, with a maximum of 15.2 mm). Positrons are annihilated by colliding with an electron of a constituent atom releasing two 511 keV gamma ray photons that travel in opposite directions. These high-energy photons are minimally attenuated in tissue, and the majority reach the imaging detector. Rubidium-82 is the most commonly used positron emitter for myocardial perfusion imaging. Nitrogen-13 ammonia is used less frequently for this purpose. Fluorine-18 deoxyglucose is used in cardiology for metabolic imaging and to detect myocardial sarcoid and other inflammatory conditions.

**4.4. Relationships Between Exposure and Absorbed Dose**
Medical radiation exposures occur in 2 ways:

1. Exposure from an external radiation beam (x-ray fluoroscopy and x-ray CT)
2. Exposure from radioactive decay within the subject (nuclear scintigraphy)

**4.4.1. Measures of Exposure From External Beams**
For external radiation beams, the absorbed dose is determined by the total incident exposure, the properties of the incident radiation, and the volume of tissue exposed.

Air kerma (“kinetic energy released in material”) is the standard unit of measure for x-ray beam exposure. It is an energy intensity measured in Gy. 1 Gy = 1 J of energy released per kilogram of absorbing material. The metric “air kerma” is used because the measurement is made using air as the absorbing material.

**Absorbed Dose From an External Beam**
Radiation absorbed dose, as distinguished from exposure, is an energy intensity, the concentration of radiation energy actually deposited in the exposed tissue. Not all radiation energy that impinges on a tissue is absorbed. Some radiation (a variable quantity depending on both
radiation and tissue characteristics) passes through the tissue without interacting with it, depositing no energy (it is this radiation that contributes to image formation). Radiation absorbed dose is also measured in Gy. 1 Gy = 1 J of energy deposited per kilogram of irradiated tissue.

External beam energy deposition in tissue is not uniform. X-ray radiation is attenuated exponentially as it passes through tissue, decreasing by approximately a factor of 2 for each 5 cm of tissue. The incident beam air kerma is a good measure of dose at the body surface, but structures deep to the body surface receive smaller doses.

**X-Ray Fluoroscopy Kerma-Area Product: Incorporating the Exposed Tissue Volume**

The risk of radiation harm is related both to the intensity of the radiation dose, and also to the quantity of tissue that receives the dose. Kerma-area product (KAP) is the product of the beam’s kerma and its cross-sectional area incorporating the volume of tissue irradiated. This concept is particularly important in x-ray fluoroscopy, as imaging field sizes vary leading to very different KAPs.

**X-Ray Computed Tomography Kerma-Length Product: Incorporating the Exposed Tissue Volume**

CT delivers radiation to a patient in a manner quite different from that of projectional imaging or fluoroscopy. The dose is distributed more uniformly around the patient.

The total dose delivered by a CT examination is the measured kerma multiplied by the axial length of the scan. A variety of dose metrics for x-ray CT are derived from this model.

### 4.4.2. Exposure From Radionuclides

Unlike external beam exposures, radionuclide exposures come from radioactive decay within the subject. Exposure is determined by the activity administered, the tracer distribution, the tracer elimination rate, and the tracer’s time-activity relationships.

### 4.5. Estimating Effective Dose

The effective dose construct assigns each organ/tissue a weighting factor that reflects the tissue’s sensitivity to radiation-induced stochastic risk. The calculation of effective dose involves estimating each organ’s actual equivalent dose (in Gy). Each organ dose is adjusted by multiplying it by the organ’s tissue-weighting factor. The organ sensitivity-adjusted individual organ doses are summed to yield a total effective dose (in Sv) (11).

For a chest exposure, absorbed dose is concentrated in the skin, mediastinal structures, lungs, breast, and thoracic bone marrow. Doses to these organs contribute the largest components to the effective dose calculation. Smaller quantities of scattered radiation expose the abdominal viscera and upper neck. As these organs would receive smaller exposures, their contribution to the effective dose calculation would be smaller.

The International Commission on Radiation Protection (ICRP) published the most recent organ sensitivity estimates in 2007 in ICRP Publication 103 (12). These estimates are listed in **Table 3**.

### 4.6. Synopsis of Measures of Radiation Exposure and Dose

The existence of the many different measures of radiation exposure and dose has the potential to cause confusion leading to misapplication of units of measure. **Table 4** contains a synopsis of the principal metrics described in this section. It should be noted that, because effective dose in the table is based on gender- and age-averaged tissue weighting factors, (not accounting for the fact that children and females are more sensitive), its practical value is in comparing the effects of different exposures rather than in estimating an individual’s stochastic risk.

### 5. HOW RADIATION CAN HARM PEOPLE

#### 5.1. Mechanism of Radiation-Induced Biological Effects

Radiation-induced tissue injury is due to molecular alteration caused by particles or photons that have sufficient energy to induce ionization. Atoms ionized by radiation are frequently chemically unstable and transform...
5.2. Types of Radiation-Induced Health Effects

Radiation-induced health effects are divided into 2 groups that differ in mechanism, the nature of effects, relationship to absorbed dose, and time between exposure and manifestation.

5.2.1. Tissue Reactions (Formerly Called Deterministic Effects)

Tissue reactions are caused by radiation-induced injury to structural and functional molecules in cells. Cell necrosis will occur if the amount of molecular damage exceeds the cell’s ability to repair itself and maintain function. Tissue reactions only become macroscopically evident if a threshold radiation dose is exceeded, causing a sufficient fraction of an exposed tissue’s cells to malfunction or necrose. A dose below the threshold dose may cause unapparent cellular injury but will not cause a detectable reaction (13).

Tissue reactions typically exhibit dose-related severity and occur with a time delay (typically 4 to 8 weeks) between exposure and the appearance of tissue injury. Above the threshold dose, a greater dose causes more extensive injury to a greater fraction of cells in proportion to the dose.

Skin injury is the most common tissue reaction observed in cardiovascular imaging. It occurs almost exclusively from x-ray fluoroscopic exposures. Other tissue reactions include cataract formation, bone necrosis and, in the heart, damage to myocardium, cardiac valves, and coronary arteries. In addition, if a fetus incurs sufficient cellular injury at critical stages of organogenesis, development will be impaired (14).
5.2.2. Stochastic Effects: Cancer

Stochastic effects are caused by radiation-induced damage to a cell’s genetic material that reprograms the damaged cell’s deoxyribonucleic acid (DNA) into dysfunctional operation. The principal stochastic event of clinical importance is radiation-induced cancer.

Stochastic effects differ from tissue reactions in their dose relationship. Whereas tissue reactions exhibit dose-related severity and have a definite dose threshold, stochastic events, in contrast, have a probabilistic relationship to dose. They are not known to have a dose threshold and do not have a quantitative dose-related severity. Radiation-induced cancer either does or does not occur (or may not present within the subject’s lifetime). A single critically located DNA damage event can create an oncogene (15). This is the theoretical basis for the concept that there is no threshold dose below which stochastic risk is zero (16).

5.2.3. Stochastic Effects: Heritable Effects in Offspring

Theoretically, radiation injury to DNA in germ cells could cause a clinically important mutation that would not affect the exposed individual, but would be transmitted to that individual’s offspring. Such effects have been demonstrated in animal models but have not been observed in humans with statistical significance (17).

5.3. Tissue Reactions: Dose-Effect Relationships

5.3.1. Skin Injury

The most common radiation-induced tissue reaction is skin injury at the beam entrance port (typically on the patient’s back) following an x-ray fluoroscopic examination. Skin entrance port injuries are rectangular, reflecting the beam shape. These injuries vary in severity from erythema to desquamation to ulceration and necrosis.

Skin injury typically appears 4 to 8 weeks following the exposure. In extreme cases, the ulceration can become confluent and full thickness necrosis of skin may develop, exposing underlying fat, muscle, and even bone (Figure 1). The skin injury threshold dose is variable, as is the relationship between dose and injury severity. A procedure’s cumulative air kerma can be used to estimate a patient’s skin injury risk.

![FIGURE 1](image.png)

An example of full thickness skin necrosis (underlying muscle and fat are exposed) caused by a large-dose x-ray fluoroscopic procedure (90 minutes of fluoroscopy time). Note the rectangular area of skin discoloration surrounding the area of skin necrosis. The injury is on the left side of the subject’s back indicating that the exposure was conducted in the right anterior oblique projection (17). (This image is available on the U.S. Food and Drug Administration Web site and is in the public domain.)

<table>
<thead>
<tr>
<th>TABLE 5 Radiation-Induced Skin Injuries—Relationship of Severity to Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Single Exposure Dose Range (Gy)</strong></td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>0-2</td>
</tr>
<tr>
<td>2-5</td>
</tr>
<tr>
<td>5-10</td>
</tr>
<tr>
<td>10-15</td>
</tr>
<tr>
<td>&gt;15</td>
</tr>
</tbody>
</table>

Adapted from Balter et al. (13).
General guideline values for the ranges of threshold values for a single first-time exposure for absorbed doses associated with degrees of skin injury severity are tabulated in Table 5. Injury thresholds for a subsequent exposure are lower.

Fluoroscopic entrance skin doses vary greatly because of variations in procedure complexity and duration and variations in patient radiological characteristics. Skin dose is strongly affected by the patient’s characteristics and procedural techniques. Body habitus is the most important patient characteristic. Larger patients require a greater skin entrance port dose. Dose is also determined by equipment calibration and imaging protocol settings.

The prototypical patient at risk for a skin injury is an obese diabetic who has undergone 1 or more long-duration procedures within the past several months.

5.3.2. Bone Injury
In addition to skin injury, on occasion, incident radiation can cause necrosis of superficial bones such as ribs. Although the dose to bone needed to cause osteonecrosis is greater than the dose that causes skin necrosis, bone’s high calcium content imparts a greater capacity to absorb x-ray photons, causing a greater absorbed dose to bone.

5.3.3. Cataracts
The single dose threshold that will cause vision-imparing cataracts in humans is not well characterized but is believed to be on the order of 500 mGy with a minimum latency of approximately 1 year (18). Cataracts are also increasingly being observed in physician operators with long career experience. This area is currently a subject of ongoing study.

5.3.4. Tissue Reactions: Managing Skin Injuries
Less-severe degrees of skin injury have the potential to heal if managed with good supportive dermatological care.

The cornerstone of optimizing the outcome of a skin injury is mechanical protection of the affected skin. X-ray injured skin is fragile. Mechanical trauma to the skin can aggravate the injury. Dressings and other mechanisms that help the patient avoid applying pressure or friction to the affected area is important.

Early recognition of a radiation-induced skin injury is essential to initiate protection and early treatment. The inherent delay of weeks between exposure and the initial signs of skin injury may interfere with recognition of the cause, delaying appropriate treatment. The best strategy to facilitate prompt recognition is to warn the patient, family, and primary care physician of the skin injury potential. The 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention state that it is a Class I recommendation for all patients who receive an air kerma at the interventional reference point $>5$ Gy to be counseled about the possibility of a skin injury and instructed how to react to the earliest signs should they occur (19).

5.4. Stochastic Effects: Radiation-Induced Cancer

Note: Considerable additional detail for this section is provided in the online version of this document.

Radiation-induced cancer is potentially the most important consequence of medical radiation exposure. It is an important determinant of a cardiovascular procedure’s risk-benefit relationship and an occupational hazard to healthcare workers who work in a radiation environment.

5.4.1. Stochastic Effects: Attribution Challenges
It is difficult to attribute a particular cancer to medical radiation exposure. The large background cancer prevalence (the lifetime risk of developing cancer is roughly 46% (16) and the risk of developing fatal cancer is about 23%) and the latent period (2 years to decades) between exposure and presentation present challenges to efforts to construct evidence-based models that relate dose to risk.

Population-based studies have demonstrated a statistical association between leukemia and other childhood cancers in children exposed to large medical radiation doses (20,21). Pearce et al. (21) found a 3.18-fold increase in incidence of leukemia in a large cohort of children exposed to a mean dose of 51 mGy from CT scanning. In a cohort of 674 children who underwent cardiac catheterization with a mean follow-up of 28.6 years (12,978 patient-years), Modan et al. (20) found a 4.75 times increased risk of malignancies, with a 6.3 times increase in lymphomas, and a 4.9 times increased risk of melanoma.

5.4.2. Stochastic Effects: Risk Metrics
At the population level, stochastic risk can be quantified as an increased cancer incidence in an exposed population compared with the background incidence in a comparable unexposed population. This risk is measured using by 2 related but different metrics:

1. **Excess relative risk.** The rate of disease in an exposed population divided by the rate of disease in an unexposed population minus 1.0.
   a. Excess relative risk is a ratio derived from the disease incidence in exposed and unexposed populations.
2. **Excess absolute risk.** The rate of disease in an exposed population minus the rate of disease in an unexposed population.

Excess absolute risk is an incidence.

### 5.4.3. Stochastic Risk: Dose-Risk Relationships

Understanding of dose-risk relationships in humans is derived from epidemiological studies of exposed human populations. These studies have clearly identified a dose-related risk for cancers including both leukemias and solid tumors. The LSS (Life Span Study), conducted by the Radiation Effects Research Foundation in residents of Hiroshima and Nagasaki, provides some of the best quantitative data relating dose to future cancer risk (22).

**Stochastic Risk: Qualitative Dose-Risk Relationships**

Most models derived from epidemiological data find a linear relationship between dose and increased future cancer risk, with no dose threshold below which there is no risk. This is the basis of the “linear-no threshold” theory, which is the basis for the concept that radiation exposure should always be minimized (ALARA: “As Low As Reasonably Achievable”) (16).

Children and young adults are more sensitive to radiation and, accordingly, for a given exposure have a greater risk of radiation-induced cancer than the elderly. Children born with congenital heart disease are at greater risk, compared with other children, for increased radiation exposure given their ongoing need for cardiac catheterization and other radiation-based procedures. In addition, because radiation-induced cancer has a latent period for induction, young people are more likely to live long enough for a stochastic event to present.

The Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation of the National Research Council has examined a number of statistical models that relate incremental cancer risk to absorbed radiation dose for individual solid organ cancers and leukemia. These models also incorporate important patient characteristics including age and gender. The models were published in the 2006 report, Biological Effects of Ionizing Radiation (BEIR) VII (16).

The models have several common features that are of pragmatic importance.

1. **Risk has a graded relationship to total dose.**
2. **Excess cancer incidence is statistically detectable in population studies at a dose of 100 mSv in adults and in smaller doses in children (23–25).**
3. **Dose-response risk for solid organ cancer correlates loosely with the organ’s intrinsic mitotic activity. The most radiation-sensitive solid organs are: lung, female breast, colon, bladder, and thyroid.**
4. **Hematopoietic tissues have a higher dose sensitivity and a shorter latent period for leukemia induction than solid organs have for primary cancers.**
5. **Women have greater risk and a steeper dose-risk relationship than men. Some, but not all, of this difference is attributable to breast sensitivity.**
6. **Risk and dose-risk relationships have a strong relationship with age, with subjects younger than 30 years of age having greater dose sensitivity (20). Beyond age 30 years, dose sensitivity is less strongly age-related (26).**
7. **The length of the latent period for clinical presentation of an induced cancer decreases the importance of radiation-related risk for elderly patients who have limited natural life expectancies.**

**Stochastic Risk: Quantitative Dose-Risk Relationships**

The quantitative relationship between radiation exposure and increased cancer risk has implications both for a patient undergoing a medical procedure and for occupationally-exposed healthcare workers.

**Background Cancer Risk in the Overall Population**

An unexposed subject’s lifetime risk of developing solid cancer or leukemia is approximately 46% and lifetime risk of cancer mortality is approximately 23% (16).

**Incremental Cancer Risk Attributable to Patient Medical Radiation Exposure**

The BEIR VII models calculate coefficients that estimate the excess relative risk and excess absolute risk per Sv of exposure. Because subject age and gender are important risk determinants, different age ranges and genders have different coefficients (larger coefficients for younger subjects and females).

The lifetime attributable risk for cancer incidence and mortality is the percent of exposed patients who are projected to develop a cancer attributable to an exposure. **Figures 2 and 3** display the model-predicted incidence and mortality estimates for a whole-body 100 mGy (100 mSv) exposure (a moderately large, but plausible, medical exposure dose). The impact of gender and age at exposure is highly evident. Children age 15 years and younger are projected to have incremental incidence rates in the range of 2% for males and 4% for females (27). In older patient groups, the predicted incremental rates are substantially smaller, but not negligible, with
smaller gender differences than in the pediatric age range.

These data are displayed graphically in Figures 2 and 3.

5.4.4. Incremental Cancer Risk Attributable to Radiation Exposure for Occupationally Exposed Healthcare Workers

Occupationally exposed healthcare workers typically incur very small doses on a daily basis that can accumulate over time into a significant exposure. Healthcare workers in x-ray environments employ protective garments. Consequently, their exposures are heterogeneous for different body parts. Healthcare workers in nuclear cardiology incur exposure when handling radioactive materials and are at risk of exposure from radiopharmaceutical spills or accidents.

There are few observational human data that assess cancer risk from long-term daily small exposures. Most of the available data comes from studies of nuclear plant operators (28). These data have not identified an increased cancer incidence in this cohort of occupationally exposed workers.

Applying the BEIR VII models to dose levels and occupational exposure durations that are typical for healthcare workers working in a medical radiation environment calculates a small but measurable increase in future cancer risk. Example findings from 2 extremes of exposure include:

1. A very low dose (1 mGy/year) throughout life such as one might experience living at high altitude. This would result in a lifetime incremental exposure of 80 mGy that would confer an incremental cancer mortality risk of 0.33% in males and 0.50% in females.
2. An extreme occupational dose for a person working in an x-ray fluoroscopic environment for his/her entire adult working life (16 mSv/year for 40 years = 640 mSv). This would confer an incremental cancer mortality risk of 1.70% in males and 2.39% in females.

**Implications of Occupational Exposure in Healthcare Workers**

The ALARA principle applies both to patients undergoing radiation-employing procedures and healthcare workers who conduct them.

Based on the risk estimates, the current recommended exposure limits for occupationally exposed workers published by the ICRP are in Table 6 (12).

It should be noted that the ICRP standards (Europe) are more stringent than the National Council on Radiation Protection standards (United States). Historically, standards have become more stringent over time. Consequently, the most stringent standards are presented.

**Implications of Fetal Radiation Exposure**

The human embryo and fetus are more sensitive to radiation effects than adults. This phenomenon has implications for the impact of radiation exposure both to patients and to occupationally exposed workers who are known to be or who may be pregnant.

Knowledge of the effects of ionizing radiation on the human embryo and developing fetus is derived from...
multiple sources, including the Hiroshima, Nagasaki, and Chernobyl experiences as well as radiation of pregnant experimental animals (29). Detrimental radiation effects include embryonic death, fetal malformations, impaired fetal development (particularly neurological), and increased risk of future cancer (16,30,31). The type of event and the dose-risk relationship for them is variable throughout the stages of pregnancy and is summarized in Tables 7 and 8 (32–34).

The principal risk of radiation exposure to the early embryo during the blastogenesis phase of development is intrauterine death, which would be experienced as failure to establish a pregnancy. Exposure during the organogenesis phase has the potential to cause fetal malformations. Later exposure during the fetogenesis phase can cause growth retardation and impaired neurological development, and can potentially increase the fetus’ future cancer risk.

In considering these risks, it is important to link the risk to threshold radiation doses. This knowledge base has been summarized by the Centers for Disease Control and Prevention (35). In this document, dose ranges are expressed in Gy rather than in Sv, as the Sv construct is not applicable to embryos and fetuses.

The increased childhood cancer risk caused by fetal radiation exposure is less well characterized, and whether fetal radiation exposure might confer a lifelong increased cancer risk is not known. Estimates of childhood cancer risk are summarized in Table 8. The available data indicate minimal detectable childhood risk at fetal doses <50 mGy but increased risk at doses >50 mGy.

A general synthesis of the fetal radiation dose data indicates that fetal doses <50 mGy (as distinguished from maternal exposures to other body regions) are not associated with a detectable increase in frequency of

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**TABLE 6**  Recommended Exposure Limits for Occupationally Exposed Workers

<table>
<thead>
<tr>
<th>Total body</th>
<th>20 mSv/yr averaged over defined periods of 5 yrs with no individual annual exposure to exceed 50 mSv.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lens of the eye</td>
<td>100 mSv/5 yrs (20 mSv/yr)</td>
</tr>
<tr>
<td>Skin</td>
<td>500 mSv/yr</td>
</tr>
<tr>
<td>Hands and feet</td>
<td>500 mSv/yr</td>
</tr>
</tbody>
</table>

Adapted from the International Commission on Radiological Protection (32).

---

**TABLE 7**  Estimates of Adverse Embryonic and Fetal Events as a Function of Fetal Radiation Dose

<table>
<thead>
<tr>
<th>Time Post Conception</th>
<th>Acute Radiation Dose* to the Embryo/Fetus</th>
<th>Blastogenesis (up to 2 wks)</th>
<th>Organogenesis (2–7 wks)</th>
<th>Fetogenesis (8–15 wks)</th>
<th>(16–25 wks)</th>
<th>(26–38 wks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.05 Gy (5 rads)</td>
<td>Noncancer health effects NOT detectable</td>
<td>Incidence of failure to implant may increase slightly, but surviving embryos will probably have no significant (noncancer) health effects</td>
<td></td>
<td>Incidence of major malformations may increase slightly</td>
<td>Growth retardation possible</td>
<td>Noncancer health effects unlikely</td>
</tr>
<tr>
<td>0.05–0.50 Gy (5–50 rads)</td>
<td>Incidence of failure to implant will likely be large,† depending on dose, but surviving embryos will probably have no significant (noncancer) health effects</td>
<td>Incidence of miscarriage may increase, depending on dose</td>
<td>Substantial risk of major malformations such as neurological and motor deficiencies</td>
<td>Growth retardation likely</td>
<td>Incidence of miscarriage will probably increase, depending on dose</td>
<td>Growth retardation possible, depending on dose</td>
</tr>
<tr>
<td>&gt;0.50 Gy (50 rads)</td>
<td>The expectant mother may be experiencing acute radiation syndrome in this range, depending on her whole body dose.</td>
<td>Incidence of miscarriage may increase, depending on dose</td>
<td>Substantial risk of major malformations such as neurological and motor deficiencies</td>
<td>Growth retardation likely</td>
<td>Incidence of severe mental retardation &gt;20%, depending on dose</td>
<td>Incidence of miscarriage will probably increase, depending on dose</td>
</tr>
</tbody>
</table>

*Acute dose: dose delivered in a short time (usually minutes). Fractionated or chronic doses: doses delivered over time. For fractionated or chronic doses the health effects to the fetus may differ from what is depicted here. †Both the gray (Gy) and the rad are units of absorbed dose and reflect the amount of energy deposited into a mass of tissue (1 Gy = 100 rads). In this document, the absorbed dose is that dose received by the entire fetus (whole-body fetal dose). The referenced absorbed dose levels in this document are assumed to be from beta, gamma, or x radiation. Neutron or proton radiation produces many of the health effects described herein at lower absorbed dose levels. ‡A fetal dose of 1 Gy (100 rad) will likely kill 50% of the embryos. The dose necessary to kill 100% of human embryos or fetuses before 18 weeks’ gestation is about 5 Gy (500 rads). ‡For adults, the LD50/60 (the dose necessary to kill 50% of the exposed population in 60 days) is about 3 to 5 Gy (300 to 500 rads) and the LD10(0 (the dose necessary to kill 100% of the exposed population) is around 10 Gy (1,000 rads). Reproduced with permission from the Centers for Disease Control and Prevention (32).
any adverse fetal outcomes. For external beam maternal exposures (x-ray fluoroscopy and x-ray CT), fetal exposures are substantially less than the exposure to the imaged or unshielded body region unless the uterus is directly in the imaged field. For occupationally exposed pregnant health care workers in x-ray fluoroscopy environments, proper shielding and practices should keep uterine exposures substantially below 50 mGy.

Specific recommendations for management of pregnant, possibly pregnant, or lactating patients are discussed in depth in the online version of this document.

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### REFERENCES


### TABLE 8

<table>
<thead>
<tr>
<th>Radiation Dose</th>
<th>Estimated Childhood Cancer Incidence†</th>
<th>Estimated Childhood Cancer Incidence‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>No radiation exposure above background</td>
<td>0.3%</td>
<td>38%</td>
</tr>
<tr>
<td>0.00–0.05 Gy (0-5 rads)</td>
<td>0.3%-1%</td>
<td>38%-40%</td>
</tr>
<tr>
<td>0.05–0.50 Gy (5-50 rads)</td>
<td>1%-6%</td>
<td>40%-55%</td>
</tr>
<tr>
<td>&gt;0.50 Gy (50 rads)</td>
<td>&gt;6%</td>
<td>&gt;55%</td>
</tr>
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</table>

Estimated lifetime cancer incidence (exposure at age 10 years). The right column tabulates the estimated lifetime incidence of cancer for the same exposure incurred at age 10 for comparison to the estimated childhood incidence from fetal exposure. Data published by the International Commission on Radiation Protection. Childhood cancer mortality is roughly half of childhood cancer incidence. The lifetime cancer risks from prenatal radiation exposure are not yet known. The lifetime risk estimates given are for Japanese males exposed at age 10 years from models published by the United Nations Scientific Committee on the Effects of Atomic Radiation.
33. ICRP. Biological effects after prenatal irradiation (embryo and fetus). ICRP publication 90. Ann ICRP. 2003;33.

**KEY WORDS** ACC Expert Consensus Document, nuclear cardiology, positron emission tomography, radiation, radiation risk, radiation safety, single-photon computed tomography, x-ray computed tomography, x-ray fluoroscopy

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**APPENDIX A. AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (RELEVANT):**

2018 ACC/HRS/NASCI/SCAI/SCCT EXPERT CONSENSUS DOCUMENT ON OPTIMAL USE OF IONIZING RADIATION IN CARDIOVASCULAR IMAGING: BEST PRACTICES FOR SAFETY AND EFFECTIVENESS, PART 1: RADIATION PHYSICS AND RADIATION BIOLOGY

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ABIM = American Board of Internal Medicine, ACC = American College of Cardiology, ACPC = Adults with Congenital Heart Disease and Pediatric Cardiology, AHA = American Heart Association, ASNC = American Society of Nuclear Cardiology, HRS = Heart Rhythm Society, NASCI = North American Society for Cardiovascular Imaging, SCAI = Society for Cardiovascular Angiography and Interventions; SCCT = Society of Cardiovascular Computed Tomography; SNMMI = Society of Nuclear Medicine and Molecular Imagery, UT = University of Texas.

APPENDIX C. ABBREVIATIONS

Gy = gray
Kerma = kinetic energy released in material
keV = kiloelectron volts
mSv = millisieverts
RWI = relationship with industry
Sv = sievert