Radiation Risks of Medical Imaging: Separating Fact from Fantasy

During the past few years, several articles have appeared in the scientific literature that predict thousands of cancers and cancer deaths per year in the U.S. population caused by medical imaging procedures that use ionizing radiation. These predictions are computed by multiplying small and highly speculative risk factors by large populations of patients to yield impressive numbers of “cancer victims.” The risk factors are acquired from the Biological Effects of Ionizing Radiation (BEIR) VII report without attention to the caveats about their use presented in the BEIR VII report. The principal data source for the risk factors is the ongoing study of survivors of the Japanese atomic explosions, a population of individuals that is greatly different from patients undergoing imaging procedures. For the purpose of risk estimation, doses to patients are converted to effective doses, even though the International Commission on Radiological Protection warns against the use of effective dose for epidemiologic studies or for estimation of individual risks. To extrapolate cancer incidence to doses of a few millisieverts from data greater than 100 mSv, a linear no-threshold model is used, even though substantial radiobiological and human exposure data imply that it is not an appropriate model. The predictions of cancers and cancer deaths are sensationalized in electronic and print public media, resulting in anxiety and fear about medical imaging among patients and parents. Not infrequently, patients are anxious about a scheduled imaging procedure because of articles they have read in the public media. In some cases, medical imaging examinations may be delayed or deferred as a consequence, resulting in a much greater risk to patients than that associated with imaging examinations.

© RSNA, 2012

[1] From the Department of Radiology (W.R.H., M.K.O.), Section of Nuclear Medicine (M.K.O.), Mayo Clinic, 725 11th St NW, Rochester, MN 55901. From the 2011 RSNA Annual Meeting. Received December 16, 2011; revision requested February 2, 2012; revision received February 18; accepted March 16; final version accepted April 13. Address correspondence to W.R.H. (e-mail: whendee@mcdw.edu).

© RSNA, 2012
The use of medical imaging to depict and help diagnose illness and injury and to guide therapeutic interventions into disease and disability has expanded greatly during the past 2 decades. Today, imaging is ubiquitous in health care, and patients with a wide spectrum of afflictions benefit from imaging procedures. As two snapshots, computed tomographic (CT) examinations in the United States increased from 26 million in 1998 to more than 70 million in 2008, and nuclear medicine procedures increased from 12 million to almost 20 million during the same period (1). Image-guided interventional procedures have shown a similar rapid rise, as have ultrasonography and magnetic resonance examinations. The rapid rise in the utilization of medical imaging is very good news, because it implies that imaging procedures are continuously being developed and used in new and expanded ways for the benefit of patients. Today, medical imaging is essential to the care of most patients in the United States, and a similar dependence is apparent in developed countries around the world.

Many imaging modalities deploy ionizing radiation, and, as a consequence, the exposure of patients to radiation has increased as medical imaging has expanded. In the early 1980s, the yearly per capita radiation dose was 3.6 mSv averaged over the U.S. population. Medical radiation contributed only 0.54 mSv to this annual dose, with the remainder coming from radon, soil, construction materials, and cosmic rays. In 2006, medical radiation contributed 3 mSv to the annual dose, raising the per capita dose to 6.2 mSv averaged over the U.S. population (1). The medical and total doses to an average individual in the U.S. population in the early 1980s and 2006 are compared in Figure 1. The increase in average per capita radiation dose reflects technologic advances and increased applications of medical imaging that have the potential to benefit more patients each year.

The increased exposure of patients to medical radiation has caused some authors to predict thousands of radiation-induced cancers and cancer deaths in the U.S. population in future years. In 2007, Brenner and Hall (2) estimated that in the future 1%–2% of all cancers in the United States will be caused by CT studies, and Berrington de González et al (3) predicted in 2009 that 29,000 additional cancers and 14,500 cancer deaths will be caused by CT examinations each year. These predictions, and several others like them (4–6), raise some fundamental questions: (a) What are the data that led to these numbers, and how dependable are these data? (b) Just how firm or speculative are these predictions, and how much attention should be given to them? The exploration of these questions is the intent of this article. The questions are important because the popular press recognizes the sensational nature of the predictions and exploits it in electronic and print media. This sensationalism provokes anxiety in patients and families (7), which may make them reluctant to agree to imaging procedures that would very much be in their best interests.

Predictions of the effects of low doses of ionizing radiation should disclose all of the limitations in the current state of knowledge about low-dose radiation effects. The argument that it is better to err on the “safe” side in predicting health effects can distort the public’s perception of the risk of low doses of radiation. After the Chernobyl nuclear reactor accident in 1986, for example, 15 million people in Belarus, Ukraine, and Russia exhibited psychosomatic disorders that were not attributable to physical effects induced by radiation exposure (8–10). Instead, the disorders were linked to the popular belief that any amount of radiation, no matter how minuscule, can cause bodily harm.

Data Sources

Several epidemiologic studies during the past 6 decades have attempted to document the health consequences of exposure to low levels of ionizing radiation. Data sources for these studies can be divided into four categories: atomic bomb survivors in Hiroshima and Nagasaki (Radiation Effects Research Foundation...
The RERF program has followed 120,000 survivors of the atomic bomb blasts, including 93,000 who were in Hiroshima or Nagasaki when the explosions occurred, and 27,000 residents who were not in the cities at the time of the explosions. The latter individuals received no radiation exposure and are usually excluded from studies of health effects in the exposed populations. Both sexes and all ages are included in the RERF data. The average dose to the exposed individuals is estimated to be 200 mSv, with the following approximate dose distributions: 0–5 mSv, 37,000 subjects; 5–100 mSv, 32,000 subjects; and 100–2000 mSv, 17,000 subjects (13). The RERF data provide statistically significant evidence of an increased incidence of various types of cancer in Japanese survivors receiving whole-body doses of 100 mSv or more. At dose levels greater than 100 mSv, there is little disagreement in the scientific community about the detrimental effects of instantaneous radiation exposures to the Japanese survivors. At less than 100 mSv, it is not possible to identify an increased incidence of cancer with any degree of statistical confidence compared with the normal incidence of cancer in the exposed populations.

It is a challenge to extrapolate health effects in the Japanese population to the possible health consequences of low-level exposure to radiation from medical imaging procedures. Cancer incidence in Japan today is very different from cancer incidence in the United States. For example, breast cancer in women is approximately three times more prevalent in the United States than in Japan, whereas stomach cancer is approximately 10 times more prevalent in Japan than in the United States (14). In addition, cancer rates in the Japanese population in the 1940s were probably different from those in Japan today. Exposures from medical imaging are from X-rays and gamma rays of relatively low energy, often administered intermittently as a consequence of multiple procedures, whereas the atomic blasts exposed Japanese residents instantaneously to high-energy gamma rays, neutrons, and charged particles. The Japanese survivors were exposed to whole-body radiation and to radioactive fallout, whereas medical exposures are confined (with the exception of nuclear medicine) to external irradiation of specific regions of the body. Food in Hiroshima and Nagasaki was limited, and much of the population was malnourished and of compromised health, which may have amplified the effects of the radiation. The bombs created hazards for the population in addition to radiation, including intense...
heat and pressure, fire, flying debris, and psychologic terror. After the bomb blasts, medical care was extremely limited, and many people died of injuries and exposures that would have been survivable under better circumstances. These factors make the Japanese survivors very different from patients undergoing medical procedures in the United States and compromise the relevance of the extrapolation of health effects from one population to the other.

Other than the RERF data, most of the population studies have revealed no or much smaller demonstrable health effects of radiation exposure (12). The few that have shown some effect (eg, increased thyroid cancer in children exposed in utero downwind of Chernobyl, increased likelihood of cancer in persons receiving multiple doses of radiation from an extended series of medical procedures) are associated with relatively high radiation doses to specific organs (15,16). Studies of 500,000 occupationally exposed workers in the nuclear industry over many years even demonstrated reduced cancer in the exposed individuals, a result termed the “healthy worker effect” and attributed to the arguable possibility that the exposed population is in better health than the population at large (17,18). The BEIR VII report largely excludes all of these studies from its analyses on the basis that they are unsuited to the development of population-based risk estimates.

Another potential source of information on the effects of radiation exposure is patients who received relatively high doses of radiation during medical procedures. How RERF data compare with data from patients can be determined from BEIR VII (Section 7: Medical Radiation Studies) (12). Figures 2 and 3 show modified versions of figures 7-1 and 7-2 taken from tables 7-2 and 7-3 of the BEIR VII report. These figures summarize the results of various studies that document increased cancer incidence in the lung and breast from radiation administered usually for therapeutic purposes.

Figure 2 depicts the ERR of lung cancer per gray of absorbed dose as a function of the organ dose reported in each study, and Figure 3 depicts the EAR of breast cancer per gray of absorbed dose as a function of the organ dose reported in each study. (Definition and discussion of EAR and ERR are in the Risk Models section.) In a perfect world, all studies would yield similar values for the ERR per gray and EAR per gray. Superimposed on the graphs are values of ERR per gray and EAR per gray derived from the RERF data, the value selected by the BEIR VII committee, and weighted averages based on the medical radiation studies. (Results were weighted by the number of cases reported in a study, because studies with small numbers had the largest statistical errors.) The BEIR VII values were weighted heavily in favor of the RERF data, even though the total numbers of cases reported for lung and breast cancer in the medical radiation studies exceeded those reported in the RERF data (1855 vs 1264 for lung cancer and 2284 vs 278 for breast cancer). For
both cancers, the BEIR VII values were considerably higher than those from the medical radiation studies. That is, medical radiation studies that are closer in both ethnicity and dose levels to those of the patient population undergoing medical imaging yield risk factors that are substantially lower than those reported from the RERF data. These findings challenge the validity of extrapolating health effects from the Japanese survivors to those for patients undergoing medical imaging.

### Linear No-Threshold Hypothesis

Cancers caused by radiation cannot be differentiated from cancers that occur spontaneously in a population and hence cannot be identified as radiation induced. All that one can do is to determine if there is an increased frequency of cancer incidence and death in an exposed population. RERF data provide statistically significant evidence of increased cancers in Japanese survivors who received doses of 100 mSv and higher, with the cancer incidence appearing to increase linearly with dose. At less than 100 mSv, an increase in radiation-induced cancers, if any, is too small to be distinguishable from cancer incidence due to all causes. Consequently, a model must be deployed to extrapolate from radiation-induced cancers at doses greater than 100 mSv to a hypothetical and much smaller number of cancers induced by doses of a few millisieverts delivered during medical imaging.

Various models for extrapolating cancer risk to low doses of radiation are illustrated in Figure 4. The model used most widely is the LNT model. This model is not chosen because there is solid biologic or epidemiologic data supporting its use. Rather, it is used because of its simplicity and because it is a conservative approach (ie, if it is not correct, then it probably overestimates the risk of cancer induction at low doses) (19). For the purpose of establishing radiation protection standards for occupationally exposed individuals and members of the public, a conservative model that overestimates radiation risk is preferred over a model that underestimates risk.

The LNT model for radiation effects first appeared in the 1920s in Hermann Muller’s publications of genetic mutations in *Drosophila* (fruit flies) induced by exposure to x-rays. Muller was awarded the 1946 Nobel Prize in Physiology or Medicine, and in his acceptance speech (20), defended the use of the LNT model for the mutagenic effects (mutagenesis) of x-rays. At that time, there was substantial evidence that the LNT model was inappropriate for x-ray-induced mutations and that a threshold appeared to exist below which mutations did not occur. Muller ignored this evidence in his acceptance speech, as documented by Calabrese (21).

In 1956, the first report was issued from the National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation (BEIR I report). At that time, genetic mutations were thought to be the major consequence of radiation exposure, primarily because of Muller’s studies. The BEIR committee engaged Muller as a consultant to help model these radiation effects in humans. At Muller’s urging, the committee adopted the LNT model to describe the possible genetic effects of radiation at low doses.

Subsequent study of the Hiroshima and Nagasaki populations over many years has revealed no genetic effects of radiation in the offspring of survivors. However, increased cancer incidence has appeared in the survivor population after receipt of instantaneous whole-body doses greater than 100 mSv. Subsequent BEIR committees have extended the LNT model from mutagenesis to carcinogenesis (the induction of cancer) at low doses without solid biologic or epidemiologic justification. In fact, there is evidence that the LNT model of radiation-induced carcinogenesis conflicts with current understanding of the biologic mechanisms of radiation injury at cellular and mammalian levels (22–24). A recent report (25) suggests that exposure of individuals to low-dose radiation may elevate the immune response and thereby protect the individuals from cancer. Nevertheless, the LNT model has gained acceptance over the years as a predictor of cancer risk at low doses of ionizing radiation.

The BEIR VII report applies the LNT model to doses as high as approximately 3000 mSv. When viewed over such a large dose range, the LNT model appears at first glance to be a reasonable model for estimation of risk. However, medical imaging uses much smaller doses compared with the doses analyzed in the BEIR VII report. At doses delivered at medical imaging, there is no direct evidence that the LNT model is an accurate predictor of cancer risk.

In 2007, Preston et al (14) published a review of the RERF data. In this report, they compared cancer incidence in the populations exposed in Hiroshima and Nagasaki with that of residents of the cities who were not in city at the time of the bombings. This study was published after the BEIR VII report. Cancer incidence as a function of dose
was presented in Table 4 of the Preston et al study and is shown in a semilogarithmic plot in Figure 5, in which the not-in-city data are designated as background dose. Colon cancer is depicted because it is commonly used as a cancer indicator in the Japanese population. The data reveal that the incidence of colon cancer is not increased in the Japanese survivors who received doses less than about 100 mSv. The data are more consistent with a threshold-quadratic model of radiation-induced cancer than with an LNT model. In fact, Preston et al (14) noted that a threshold model for radiation-induced cancer incidence fits better than an LNT model, although the difference was not statistically significant.

### Table 1

<table>
<thead>
<tr>
<th>Examination</th>
<th>Effective Dose (mSv)</th>
<th>Range in Literature (mSv)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td>2</td>
<td>0.9–4.0</td>
</tr>
<tr>
<td>Neck</td>
<td>3</td>
<td>...</td>
</tr>
<tr>
<td>Chest</td>
<td>7</td>
<td>4.0–18.0</td>
</tr>
<tr>
<td>Chest for pulmonary embolism</td>
<td>15</td>
<td>13–40</td>
</tr>
<tr>
<td>Abdomen</td>
<td>8</td>
<td>3.5–25</td>
</tr>
<tr>
<td>Pelvis</td>
<td>6</td>
<td>3.3–10</td>
</tr>
<tr>
<td>Three-phase liver study</td>
<td>15</td>
<td>...</td>
</tr>
<tr>
<td>Spine</td>
<td>6</td>
<td>1.5–10</td>
</tr>
<tr>
<td>Coronary angiography</td>
<td>16</td>
<td>5.0–32</td>
</tr>
<tr>
<td>Calcium scoring</td>
<td>3</td>
<td>1.0–12</td>
</tr>
<tr>
<td>Virtual colonoscopy</td>
<td>10</td>
<td>4.0–13.2</td>
</tr>
</tbody>
</table>

Note.—Reprinted, with permission, from reference 26.

### Dose Descriptors

A major problem in estimating the cancer risk of medical imaging is to relate the doses delivered to specific organs during imaging to the cancer risks predicted in the BEIR VII report from RERF data for Japanese survivors receiving whole-body doses. Frequently, this relationship is attempted by expressing doses from imaging procedures in terms of effective doses, as depicted in Table 1. The effective dose is computed by multiplying the dose to each irradiated organ in a patient by a radiation weighting factor (unity for x-rays and gamma rays) and by a biologic weighting factor specific for the organ and summing the products for all exposed organs to yield the effective dose. The effective dose is defined as the dose which, if delivered uniformly to the whole body, would produce the same health consequences as those caused by a dose delivered to one or more specific organs.

The effective dose is a useful concept for developing radiation protection standards and setting dose limits for occupationally exposed individuals. It is not intended for epidemiologic studies or predictions of risk to exposed individuals. Unfortunately, effective dose is often used exactly in this unintended manner to predict cancer incidence and death in populations exposed to medical procedures. As the International Commission on Radiological Protection has stated in publication 103 (27):

**Effective dose is intended for use as a protection quantity. The main uses of effective dose are the prospective dose assessment for planning and optimization in radiological protection, and demonstration of compliance with dose limits for regulatory purposes. Effective dose is not recommended for epidemiological evaluations, nor should it be used for detailed specific retrospective investigations of individual exposure and risk.**

### Risk Models

As noted previously, the BEIR VII committee gave great weight to the RERF data. In addition, RERF personnel were recruited to assist the committee in analyzing cancer incidence and mortality and in developing risk models described in BEIR VII. In their publications, RERF personnel have emphasized the limitations in estimates of radiation risk at low doses. For example, Pierce and Preston have noted that at levels less than 100 mSv, assessing cancer risks “...greatly strains any epidemiological investigation since, within the scope of a study, cancer rates may vary to at least that degree due to other risk factors correlated with the exposure under investigation” (28).

The BEIR VII committee uses two risk models as the foundation for estimating the likelihood of radiation-induced cancer. These models are the ERR model and the EAR model. The ERR is the rate of disease in the exposed population divided by the rate of disease in an unexposed population minus 1.0, and the EAR is the rate of disease in an exposed population minus the rate of disease in an unexposed population. Risk factors from these models are then incorporated into a final risk model, the lifetime attributable risk (LAR) model, to compute a risk estimate for the likelihood of radiation-induced cancer over...
the lifetime of individuals exposed to ionizing radiation. It is this LAR model that has been used to predict cancer incidence and deaths in populations of individuals exposed to medical radiation. A large number of limitations and uncertainties underlie these predictions.

An illustration of the limitations in LAR estimates is seen in Table 2 (table 12-5A from the BEIR VII report). This table displays estimates of LAR based on the ERR model and the EAR model. Given that both models are based on the same data, one might anticipate reason agreement between them. As shown in Figure 6, this is not the case. For example, in a population of 100,000 people exposed to 100 mGy, the LAR based on the ERR model predicts 25 stomach cancers, whereas that based on the EAR model predicts 280 cancers. Conversely for prostate cancer, the ERR-based LAR predicts 190 cancers, whereas the EAR-based LAR predicts six. Clearly one or both models are in error. Because of the paucity of data, unfortunately, it is not possible to determine which model is more accurate. The BEIR VII committee resolves the differences between EAR and ERR models by combining estimates from them by using the following expression: LAR = p ✕ LAR (ERR) + (1 − p) LAR (EAR), where p is determined by the views and opinions of the committee.

We do not fault the path taken by the BEIR VII committee. The dearth of solid data on the effects of low levels of ionizing radiation and the complexity of the limited data that are available make the task of BEIR VII an undeniable one. At every step in the process, BEIR VII had to make assumptions about factors that could profoundly influence the final results. These assumptions are evident from even a cursory review of the BEIR VII report.

### Risk Estimates

Estimates of LAR of cancer in specific organs are provided in the BEIR VII report for a dose of 0.1 Gy (100 mGy) delivered to a population of 100,000 individuals of mixed ages and both sexes. A sample of these estimates is reproduced in Table 2 for the purpose of illustrating the wide range of values of each estimate encompassed by what is termed a subjective 95% confidence interval. For example, the LAR for liver cancer in female patients (predicted cancers per 100,000 persons exposed)

<table>
<thead>
<tr>
<th>Cancer Site</th>
<th>Male Patients</th>
<th>Female Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LAR Based on Relative Risk Transport</td>
<td>LAR Based on Absolute Risk Transport</td>
</tr>
<tr>
<td>Stomach</td>
<td>25</td>
<td>280</td>
</tr>
<tr>
<td>Colon</td>
<td>260</td>
<td>180</td>
</tr>
<tr>
<td>Liver</td>
<td>23</td>
<td>150</td>
</tr>
<tr>
<td>Lung</td>
<td>250</td>
<td>190</td>
</tr>
<tr>
<td>Breast</td>
<td>510 (not used)</td>
<td>460</td>
</tr>
<tr>
<td>Prostate</td>
<td>190</td>
<td>6</td>
</tr>
<tr>
<td>Uterus</td>
<td>66</td>
<td>47</td>
</tr>
<tr>
<td>Ovary</td>
<td>470</td>
<td>350</td>
</tr>
<tr>
<td>Bladder</td>
<td>32</td>
<td>No model</td>
</tr>
<tr>
<td>Other</td>
<td>1400</td>
<td>1310&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sum of site-specific estimates</td>
<td>1550</td>
<td>1250</td>
</tr>
</tbody>
</table>

Note.—Reprinted, with permission, from table 12-5A from reference 12. Data are number of cases per 100,000 persons of mixed ages exposed to 0.1 Gy. Data in parentheses are subjective 95% confidence intervals. DDREF = dose and dose rate effectiveness factor.

* Linear estimate based on ERR models shown in table 12-2 with no DDREF adjustment.
*<sup>1</sup> Linear estimate based on EAR models shown in table 12-2 with no DDREF adjustment.
*<sup>2</sup> Estimates obtained as a weighted average (on a logarithmic scale) of estimates based on relative and absolute risk transport. For sites other than lung, breast, and thyroid, relative risk transport was given a weight of 0.7 and absolute risk transport was given a weight of 0.3. These weights were reversed for lung cancer. Models for breast and thyroid cancer were based on data that included Caucasian subjects. The resulting estimates were reduced by a DDREF of 1.5.
*<sup>3</sup> Including uncertainty from sampling variability, transport, and DDREF. Sampling uncertainty in the parameters that quantify the modifying effects of age at exposure and attained age is not included except for the all solid cancer model.
*<sup>4</sup> Includes thyroid cancer estimate based on ERR model.
*<sup>5</sup> Includes breast cancer estimate based on EAR model.
*<sup>6</sup> Estimates based on model developed by analyzing life span study incidence data on all solid cancers excluding thyroid cancer and nonmelanoma skin cancer as a single category (table 12-1).
posed to 0.1 Gy) is 12 with a 95% confidence interval of 1 to 130. The LAR for radiation-induced prostate cancer in male patients is 44 with a 95% confidence interval of less than 0 (maybe radiation hormesis?) to 1860. Confidence intervals this wide undermine the meaningfulness of predictions of cancer incidence derived from LAR estimates.

The adjusted LAR estimates in Table 2 are decreased by a factor of 1.5, which is referred to as the dose and dose rate effectiveness factor. This factor is an assumption used to correct for possible reduced effects of radiation dose when the dose is small or delivered at a low dose rate, thereby permitting cellular repair of injury to occur.

The BEIR VII committee was careful to point out the limitations and uncertainties of its risk estimates. The committee states that “because of the various sources of uncertainty it is important to regard specific estimates of LAR with a healthy skepticism, placing more faith in a range of possible values.” It states further that the “…range of plausible values for lifetime risk is consequently labeled a ‘subjective confidence interval’ to emphasize its’ [sic] dependence on the opinions of the committee in addition to direct numerical observation” (12) (BEIR VII, Section 11, page 279). Unfortunately, many articles that use the BEIR VII report to forecast cancer incidence and deaths from medical studies fail to acknowledge the limitations of BEIR VII and accept its risk estimates as scientific fact rather than as a consensus opinion of a committee.

Often a risk estimate of 5% per sievert is used as an approximate predictor of cancer incidence in all organs for a population of individuals exposed to ionizing radiation. Articles in the scientific literature that use this predictor stimulate sensational articles in the electronic and print public media that create anxiety in patients and parents. For the reasons described previously, the accuracy of this risk estimate is highly suspect. Use of the 5% per sievert predictor of cancer incidence (or any other numeric predictor of radiation-induced cancer incidence at low doses) must be considered highly speculative at best.

Estimates of radiation-induced cancer incidence and death from medical imaging are computed at times with the assumption that the age distribution of the exposed individuals resembles that of the population at large. This assumption is invalid, because older patients undergo the bulk of imaging examinations. Older patients are at substantially reduced risk for cancer induction for several reasons, including their limited expected lifetimes. The age factor for medically exposed individuals lowers the risk substantially compared with risk estimates without consideration of patient age (12).

Many patients who undergo medical imaging procedures have an illness that shortens their life expectancy. These patients are at reduced risk of cancer induction by radiation because they will not survive long enough for the cancer to materialize (29). This comorbidity problem reduces the risk of radiation-induced cancer averaged over the entire patient population.

### Conclusions

No prospective epidemiologic study with nonirradiated control subjects has quantitatively demonstrated adverse effects of radiation at doses less than about 100 mSv. A recently published retrospective cohort study (30) demonstrated an increase in leukemia and brain cancer in children who underwent multiple CT scans at ages younger than 15 years, with an excess absolute risk of 0.83 excess case of leukemia and 0.32 excess case of brain cancer in 10,000 children receiving 10 mGy from a CT scan. Children are recognized as particularly susceptible to radiation injury, and care should always be exercised to keep dose as low as possible while consistent with acquiring needed diagnostic information. It is essentially impossible to accurately predict cancer incidence and death in a population of individuals exposed to doses below about 100 mSv. Virtually all imaging procedures, including CT and nuclear medicine examinations, deliver doses to
patients well below 100 mSv when they are properly conducted. Hence, predictions of cancer incidence and death from medical imaging procedures lack supporting data and are highly speculative. In the future, it may become possible to make more accurate predictions of cancer induction (or its absence) at low doses through improved understanding of cellular mechanisms of cancer, better criteria for identifying cancer precursors at the cellular and molecular levels, more relevant epidemiologic data on cancer risk from large registries of patients exposed to medical radiation, and studies of subpopulations of individuals (eg, persons with ataxia telangiectasia) who are at increased risk of cancer after radiation exposure. At this time, these advances seem rather distant.

Because predictions of cancer incidence and death in populations exposed to doses less than 100 mSv are highly controversial, the Health Physics Society has taken the following position (31): “The Health Physics Society recommends against quantitative estimation of health risks below an individual dose of 5 rem (50 mSv) in one year, or a lifetime dose of 10 rem (100 mSv), above that received from natural sources. For doses below 5–10 rem (50–100 mSv) risks of health effects are either too small to be observed or are nonexistent.”

The American Association of Physicists in Medicine, an organization of more than 7000 medical physicists responsible for the quality and safety of medical imaging and radiation therapy, approved in December 13, 2011 the following statement concerning the risks of medical imaging (32):

The American Association of Physicists in Medicine (AAPM) acknowledges that medical imaging procedures should be appropriate and conducted at the lowest radiation dose consistent with acquisition of the desired information. Discussion of risks related to radiation dose from medical imaging procedures should be accompanied by acknowledgment of the benefits of the procedures. Risks of medical imaging at patient doses below 50 mSv for single procedures or 100 mSv for multiple procedures over short time periods are too low to be detectable and may be nonexistent. Predictions of hypothetical cancer incidence and deaths in patient populations exposed to such low doses are highly speculative and should be discouraged. These predictions are harmful because they lead to sensationalistic articles in the public media that cause some patients and parents to refuse medical imaging procedures, placing them at substantial risk by not receiving the clinical benefits of the prescribed procedures.

AAPM members continually strive to improve medical imaging by lowering radiation levels and maximizing benefits of imaging procedures involving ionizing radiation.

Highly speculative articles that predict cancer incidence and death in populations receiving relatively small doses of radiation from medical imaging are not without their own health risks. These articles receive considerable media attention because they emphasize hypothetical cancer risks of imaging procedures without acknowledgment of the benefits that the procedures provide to patients. Governmental agencies, institutions, and medical groups spend millions of dollars each year to safeguard against low levels of radiation—funding that is diverted from other more pressing needs. This distorted emphasis does induce one risk in many patients—namely anxiety about imaging procedures that causes some patients and parents to delay or defer necessary imaging procedures. The negative health consequences of deferred imaging examinations undoubtedly far outweigh any risks of having the procedures performed.

This article does not contend that medical imaging procedures should be conducted without concern about the dose delivered to patients. The authors support efforts such as Image Gently (33) and Image Wisely (34) to use only enough radiation to acquire needed diagnostic information. The authors believe in three principles: to keep radiation doses as low as reasonably achievable (or ALARA), to keep medical procedures as safe as reasonably achievable (or ASARA), and to keep medical benefits as high as reasonably achievable (or AHARA).

Disclosures of Potential Conflicts of Interest: W.R.H. No potential conflicts of interest to disclose. M.K.O. No potential conflicts of interest to disclose.

References