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EFFECTS OF OCCUPATIONAL AND ENVIRONMENTAL EXPOSURES TO IONIZING RADIATION

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INTRODUCTION

The injurious effects of ionizing radiation were recognized soon after the discoveries of x-ray and radium. Eight years after his first exposure during a series of experiments with x-rays Thomas Edison's assistant died of metastases from carcinoma of the skin induced by the radiation exposure. A distressing number of the early workers with x-rays and radium died or were severely injured by excessive radiation. The sequence of excessive exposure, radiodermatitis and cancer became all too familiar not only among radiologists but also among some patients too heroically treated.

Heavy mortality in research workers with radium (Madame Curie herself died of radiation-induced aplastic anemia), medical specialists in its use and radium chemists called attention to the danger of another type of radiation, that due to internal absorption of radioactive materials and damage from these internal sources. Full awareness of this type of danger did not come, however, until Harrison Martland,¹ medical examiner for Essex County, New Jersey, called attention to the dangers to the luminous dial painters who tipped their brushes with their lips and ingested small amounts of radium and mesothorium. Martland's studies were followed by the brilliant work of Evans² who laid the foundation for the present ongoing study of internal contamination with radioactive materials at the Argonne National Laboratory of the Atomic Energy Commission.

As ionizing radiation became more and more important in medicine, as the dangers became better appreciated, larger numbers of patients were

exposed to greater or less degrees of radiation, and the need for radiation protection became better recognized. An important step was the establishment by radiologists and the radiation industry of a National Committee on Radiation Protection (now the National Council on Radiation Protection and Measurements) which for years established informal but widely recognized standards for protection of radiation workers and later of potentially exposed general populations. Similar organizations have been established in other countries. Their activities are coordinated by an International Committee. Radiation standards for the Federal Government are the responsibility of the Federal Radiation Council. Federal agencies most immediately concerned are the Bureau of Radiological Health in the Department of Health, Education and Welfare, a related group in the Environmental Protection Agency and the Atomic Energy Commission. State governments are responsible for standards and regulations regarding potential radiation hazards. These largely follow the recommendation of the national bodies.

With World War II and the international race in search of controlled atomic energy large numbers of persons were for the first time occupationally exposed to a wide range of radioactive materials. Very fortunately, the potential occupational and public health hazards were appreciated from the start, thanks to earlier experience with therapeutic or occupational exposures, and adequate standards were laid down for the protection of those involved.

The atomic explosions at Hiroshima and Nagasaki revolutionized warfare as they tipped the balance overwhelmingly in favor of the offense. The efficiency and cheapness of destruction was increased by a factor of

several thousand over that of other weapons. Deaths of thousands by radiation, the dramatic surrender of Japan, the availability of hitherto unknown man-made radioactive sources ranging from atomic reactors to radioactive isotopes, led to a great surge of interest in radiation effects and to the new fields of radiobiology, nuclear medicine and health physics. These and other factors which made ionizing radiation of much greater importance in our daily lives, as for example the growth of nuclear power, altered the attitude of the general public from disinterest to the present almost unduly heightened concern.

It is the duty of forensic medicine to detect evidences of radiation injury so that hazards may be prevented or controlled, that those injured may be helped to whatever extent possible, as well as adequately compensated if deserved, that late complications may be avoided through knowledge of the progress of lesions, and that false compensation claims may be detected and rejected.

SOURCES OF RADIATION

Natural—From his beginning, man has been exposed to small amounts of background ionizing radiation, half of which on the average is of cosmic origin (the sun and radiation from outer space) and about half of which is of terrestrial origin (chiefly from elements in the radium-thorium series in granitic rocks and sands and some naturally-occurring ^{40}K). The yearly average dose of whole-body radiation in the United States is about 100 millirads/year. In a few naturally high radioactive areas such as Kerala in India and southeast Brazil the level is about ten times as high. Relatively high levels in the United States are found in

the Colorado plateau and, owing to increased cosmic radiation due to altitude and to chemical makeup of rocks and soil there, are over three times as high as at sea level.³

Minute traces of ^{14}C , too small to be readily detected, are present in foods, air and water. Since the ^{14}C content of living things is in equilibrium of that of their environment, archeologists have been able to make practical use of this fact by determining the ^{14}C content of retrieved organic substances, comparing the level found to the current atmospheric level and so permitting estimates of age.⁴

Man-made--The chief man-made sources of radiation are diagnostic x-ray apparatus, clinical nuclear medicine, radiation sources used in industry, and therapeutic radiation. It is estimated that the genetically significant dose to the United States population from medical and dental sources is about 100 mR/yr.³ Improvements in technique are leading to reductions in this annual average. In general, man-made devices for the production of ionizing radiation emit radiation only when electric power is applied. However, portions of cyclotrons or other complex devices may remain radioactive because of neutron absorption, the time depending also on the physical properties (neutron capture cross-sections) of the exposed materials. Also, radioisotopes may be sources of exposure, depending on physical characteristics and degree of control.

Nuclear--Uranium mines and mills provide a potential hazard through the associated radon and daughter products. Uranium has some toxicity as an absorbed chemical poison, injurious to renal tubular cells, and has but little radioactivity unless enriched. Carcinoma of the lung has been induced among uranium miners, particularly those who also smoke cigarettes.

The transuranic elements, particularly plutonium, are especially hazardous because of their emission of alpha particles.

Nuclear power generating plants and other reactors normally produce as wastes three types of radioactive substances: 1) Gases, such as tritium, ^{85}Kr , ^{131}Xe , ^{133}Xe and small amounts of the several radioisotopes of iodine, chiefly ^{131}I , in vapor form. These are appropriately collected, diluted and discharged to insure safety both of reactor power station personnel and potentially exposed public; 2) Liquids, such as small amounts of liquid wastes from leakage of primary coolant water from a reactor or from other substances exposed to neutron flux radioactivity. Among important radioactive liquid contaminants are tritium, ^{59}Fe , ^{90}Sr , ^{131}I and ^{137}Cs . These liquids are largely contained, in part diluted and discharged; 3) Solids. The chief potential hazards are particles such as inert dust particles which have adsorbed radioisotopes on their surfaces or in very rare instances particles of plutonium which give off alpha radiation. Reactor fuel rods accumulate radioactive fission products as they are used. Hence, used fuel rods are more potentially dangerous than the fresh rods. In addition, they contain varying amounts of plutonium, a transuranic element emitting alpha particles, which has been shown to be extremely potent in producing cancer in animals. Such lesions in man have not yet been reported. An atomic fuel reprocessing plant may have particularly complex problems in radiation protection due to the many types of radioactive elements present.

Solid wastes are largely substances that have been exposed to neutron flux with induction of radioactivity within them by neutron capture as might occur in metal from the reactor core or substances contaminated by

radioactive materials. Radioactive wastes, liquid and solid, are contained with adequate shielding and shipped to controlled disposal sites.

Packaging is such that in transport accidents the radioactivity should be contained.

Under the established standards the contribution of atomic power plants to the average whole-body dose to the population of the United States is about 1/33,000th of the dose received from natural background.⁵ An argument used by the opponents of nuclear power to delay its utilization is that a major catastrophe may occur due to a run-away reaction⁶ with generation of great local heat ("melt-down") and liberation of large amounts of a variety of radioisotopes. Specifically, a loss of coolant has been hypothesized. The probability of such an event is extremely small and has been compared to that of a meteor striking a populated area of the earth.

Radiopharmaceuticals are now widely utilized in the United States and may contribute about one percent of the average annual whole-body dose of radiation to the population. These are usually transported by truck or airplane and are so packaged as to protect the handlers and the public.

Among the man-made sources that may contribute to accidental occupational exposure are devices for radiologic examination of industrial fabrication such as welds, castings or pipes. Here such radioisotopes as ⁶⁰Co, ¹³⁷Cs and others are used as sources.

Fallout from Nuclear Tests--Radioactive materials have been added to the biosphere through surface and atmospheric nuclear tests and rare accidental venting of underground tests. These fission products are usually in too low a concentration to have detectable pathologic effects.

In such rare events as, for example, the passage of a radioactive cloud over Rongelap in 1954 following a nuclear test explosion both gaseous and particulate materials were present. Radioisotopes of iodine were the most important contributors to dose, and because of the avidity of the thyroid gland for iodine more damage was done to this than other organs.

If an atomic explosion is sufficiently powerful to project radioactive material into the upper atmosphere and the stratosphere, world-wide distribution of the material may occur. The first studies of the nature and distribution of radioactive fallout were made by the Manhattan Project of the United States following the Trinity Test in 1945. Subsequently Japanese and Americans studied fallout after the explosions at Hiroshima and Nagasaki. Since then world-wide studies have been repeatedly carried out with detailed measurements of radioactivity. A very wide range of studies have been made of the distribution of the radioactive materials, their entry into the biosphere, and particularly the food chains. The results of these studies have been summarized by Reports of the United Nations Scientific Committee on the Effects of Atomic Radiation,^{7,8} Biological Effects of Atomic Radiation,⁹ Biological Effects of Ionizing Radiations¹⁰ and Medical Research Council of the United Kingdom.¹¹ In spite of the many tests of atomic explosive devices, world-wide distribution of radioactive fallout makes only a slight contribution to the total dose of ambient radiation.

The Various Kinds of Ionizing Radiations--Ionizing radiation reacts with matter by the addition or removal of electric charges from atoms or molecules that are ordinarily neutral, thus converting them to

electrically-charged ions. These several types of ionizing radiation are listed below.

- 1) Alpha rays are positively-charged particles which are the nuclei of helium atoms. They penetrate poorly but ionize densely, having high linear energy transfer.
- 2) Beta rays are electrons, negatively-or positively-charged and are moderately penetrating.
- 3) Gamma rays are a type of electromagnetic radiation of short wavelength, highly penetrating.
- 4) X-rays are a form of electromagnetic radiation similar to gamma rays and are highly penetrating.
- 5) Neutrons are uncharged but highly penetrating particles, basic elements in the nuclei of atoms.
- 6) Cosmic rays are a mixture of highly accelerated heavy particles (stripped elemental nuclei) with different energies, highly penetrating, and are of galactic or solar origin. Most of these are excluded from the earth by the Van Allen layers.

Levels of Radioactive Materials Normally Found in Man¹²—The average ^{226}Ra content of the skeleton of a standard man is about 30 pCi. This represents 80 to 85 percent of the total radium in the body. The concentration of ^{226}Ra /gram of body ash is constant throughout life. The ratio of radium to calcium in body ash is 0.016. The natural skeletal content of ^{232}Th is 10 pCi. The ^{210}Pb content is about 270 pCi. The average total potassium content of the body of a standard man is 140 grams; 0.1 percent of this is the radioisotope ^{40}K . The total dose from this radioactive element is about 11 mR per year to bone, somewhat more to soft tissues.¹³

As a result of atmospheric nuclear weapons testing a few atoms of many of the longer-lived radioisotopes are present in the body of most members of the world's population, but in insignificant amount so far as effect is concerned. Among those which can be readily detected are ^{90}Sr and ^{137}Cs . If a body or skeleton does not contain minute traces of either of these isotopes death had probably occurred prior to 1954, when large-scale weapons testing began. $^{90}\text{Strontium}$, metabolized like calcium, tends to enter into the crystalline structure of bone and is retained selectively to a considerable degree. The average adult body burden in New York is currently about 1 nCi.¹⁴ The levels of ^{137}Cs on the other hand tend to be in equilibrium with those in the environment.

Population Dose Rates to Critical Tissues from Various Sources—The relative average contribution of the different sources to radiation of the gonads, the bone-forming cells, the bone marrow and whole body of man are shown in the accompanying table.

Table 1

Dose Rates in Millirads/year to

Source of Radiation	Gonads	Bone-forming Cells	Bone Marrow Cells	Whole Body
Natural	95	96	90	102
Medical and dental	11-45	--	32	73
Occupational	--	--	--	0.8
Nuclear power	--	--	--	0.003

These figures refer to contributions to average population doses. Obviously, some individuals such as patients requiring radiation therapy, seriously ill patients requiring multiple diagnostic x-ray studies, and possibly some occupationally-exposed persons may exceed these average levels.^{10,15}

The permissible occupational dose level is arrived at by the formula $N \times 5 \text{ rem} = \text{permissible dose}$ where N is the number of years of age. Occupational exposure is not permitted under 18 years of age. No unfavorable results have been detected in those who have maintained the occupational safeguards.¹⁶

Radiation Damage to Cells—Ionization can be produced in almost any element of the cell from the all-pervasive water to complex proteins; hence, every aspect of cellular activity may be damaged. If the damage is slight, there may be a latent period before it becomes apparent. Living tissue is not inert, however, so that there is some degree of repair of radiation damage. This principle is used clinically in protraction of therapeutic radiation to reduce its injurious effects on normal tissues.

A major early effect is damage to protein and DNA as well as to various enzymes. Since these are controlling mechanisms in the cell, the effect of radiation is usually much greater than would be expected from the mere caloric energy transfer involved. A particularly important effect is the retardation or inhibition of cell division. This is often accompanied by structural changes in chromosomes. This chromosomal damage resulting in fragmentation, translocation or deletion may well be the key factor in damage to the cell.

Large amounts of radiation will produce significant changes. As little as 22 rads of whole-body radiation may produce detectable but probably insignificant chromosomal abnormalities. More extensive changes are induced by doses above 200 rads. Chromosomal changes may be detected in man by a study of cultured lymphocytes from the peripheral blood. Fragmentation or deletion of chromosomes is a nonspecific type of injury.

The presence of dicentric chromosomes or ring chromosomes is strongly suggestive of radiation injury. Radiomimetic drugs such as nitrogen mustard may produce chromosomal aberrations similar to that seen following radiation but do not cause the more localized types of tissue and organ damage seen following radiation.

The structural changes in chromosomes evidence damage to DNA. If these changes occur in cells of germinal epithelium hereditary effects may be manifest. These are beyond the scope of the present chapter. If the damage has been done to somatic cells a wide variety of changes may result ranging from prompt cell death, death of cells after several cell divisions have taken place to varying degrees of impairment of cell function.

One of the best indices of exposure to damaging amounts of radiation is the presence of quantitative and qualitative chromosomal abnormalities in cultured circulating lymphocytes. The chromosomes may be studied by appropriate treatment and cultivation of circulating lymphocytes in whole blood. Of the many observed forms of chromosomal damage, the occurrence of such translocations as dicentric chromosomes and the presence of ring forms are particularly characteristic of radiation. In fact, the absence of such changes should raise serious question as to the accuracy of a history of antecedent radiation. Some of the radiomimetic drugs may induce similar changes. A wide variety of mutagenic substances can cause fragmentation or loss of chromosomes.

The cytoplasm of cells may also be altered, showing chiefly vacuolization in the acute stages of injury and later increased evidence of maturation or senescence. Electron microscopic study of radiation-damaged cells shows in addition to nuclear damage vacuolar cytoplasmic changes, dilated cisternae of the endoplasmic reticulum and swelling of mitochondria.¹⁷ These changes, however, are not specific for radiation damage.

Radiation often leads to premature maturation and aging of cells, as evidenced by accumulation of end products such as pigments or keratin granules. The absence or slowing of mitosis may lead to the formation of giant cells with multiple or large or bizarre nuclei.

Genetic Effects—Ionizing radiation is one of the most potent means known for inducing genetic change, as Muller¹⁸ pointed out. It is assumed in planning protection against ionizing radiation that there is no dose below which genetic change may not occur and that any increase in exposure of a population will induce proportional increase in genetic mutations. On this basis a dose of one R to a thousand members of a population would have the same hereditary effect as would a dose of 100 R to ten members. An excellent discussion of genetic changes in man is presented in the Reports of the United Nations Scientific Committee on the Effects of Atomic Radiation.^{19,20} Also of value is the comprehensive report of Reel and Schull²¹ on pregnancy termination among the irradiated survivors of Hiroshima and Nagasaki.

Somatic Effects—Somatic effects are determined by the combination of a variety of physical and biologic factors such as the volume of tissue irradiated, the sensitivity of its constituent cells, the dose, the dose

rate and the type of radiation. The broad experience gained by therapeutic radiation has shown that very large doses aggregating thousands of R can be delivered to a very limited portion of the body without undue general deleterious effect, whereas whole-body radiation is but poorly tolerated. The lethal dose of whole-body radiation for 50 percent of a given population is about 450 rem (roentgen equivalent man). Large doses of whole-body radiation may lead to life shortening in both animals and man. Localized radiation may result in local atrophy or ulceration and ultimately neoplasia.

Rapidly growing tissues, such as those of the embryo or child, are specially susceptible to radiation injury. The effects in infancy and childhood and on the growth of bone, teeth and soft parts have recently been discussed by Dawson.²² The epiphyseal cartilage cells are particularly sensitive to radiation. Damage to the epiphyseal plate interferes with growth of bone. It may under some circumstances produce osteochondromas. The iatrogenic aspects of radiation carcinogenesis have been presented by Warren.²³

Irradiation in utero may lead to abortion if the dose is over 200 rem or may cause a wide variety of congenital anomalies. Details on these have been given by Russell,²⁴ Rugh,²⁵ Warren,²⁶ Kučerová²⁷ and Kato.²⁸

Radiation damage to various tissues and organs may result in a wide variety of lesions, but all tend to have certain common properties. Since the cells and tissue have varying degrees of sensitivity to radiation, usually more severe the more rapidly the tissue proliferates,²⁹ there tends to be selective loss of certain structures. Thus, radiation of the

skin will produce loss of hair follicles while the erector pili muscles remain intact. Because the endothelial cells of capillaries and small vessels are quite sensitive, vascular damage is usually an important component of radiation injury and is shown early by swelling of endothelial cells or even necrosis with thrombosis and later the development of large endothelial cells with irregular nuclei together with hyaline or fibrous thickening of vascular walls. Telangiectases may be prominent. A third common lesion is increased intercellular substance, chiefly collagen but sometimes elastic as well. The collagen usually shows some degree of hyalinization. Surviving fibrocytes and at times other cells may show giant and distorted nuclei.

Irradiation of bone may result in two types of change. Osteolytic changes occur if the dose inhibits osteoblastic activity but is not high enough to kill osteoclasts (sometimes resulting from a dose of several hundred rads or more). The more common response is increased density or sklerotization of bone where the dose has been sufficient (usually several thousand rads) to kill osteoclasts and to damage but not destroy osteoblastic function so that dense bone continues to be laid down without resorption or remodeling. Irradiated bone is always more brittle than is normal bone.

Criteria of Injury Caused by Radiation—There are several types of criteria for identifying injuries caused by radiation. In any case of suspected injury by radiation a detailed history of exposure and estimate of dose and physical factors are helpful.

1) Localized radiation—Acute localized lesions at some time within the first two months appear first as edema of the skin, usually accompanied

or followed by epilation and loss of ability to sweat. In the more heavily irradiated cases (doses of thousands of rads) erythema or even blistering occur, and may be followed by dry desquamation, moist desquamation and necrosis with ulceration in order of increasing dose. These changes are frequently accompanied by symptoms of itching or pain.

Desquamation and ulceration may appear if mucous membranes are involved. Very rarely the dose may be high enough to produce necrosis. This is usually sharply demarcated by the limits of the field of exposure.

It is a general rule that because of the latent period in development of radiation effects that an early radiation lesion rarely looks as serious as it is. This is the reverse of injury due to another physical agent, cold, where the initial frostbite damage tends to appear more extensive than is ultimately the case. After months or even years delayed lesions may appear. Radiation dermatitis is extremely persistent and usually painful with patchy keratoses and foci of ulceration. The irradiated epithelium may ultimately give rise to epidermoid carcinoma or to basal cell carcinoma. Very rarely malignant change will occur in the connective tissue.

Among the milder late changes are hyperpigmentation or depigmentation. On histologic examination the epithelium tends to be hyperkeratinized and the normal cellular orientation lost. The sweat glands, sebaceous glands and hair follicles will show varying degrees of atrophy or may be absent. The fibers of the arrectores pilorum tend to be relatively spared. Collagen is increased in amount and tends to be hyalinized. Focal necrosis with fibrin deposition may occur. Elastic tissue is also increased. Fibrocytes are decreased in number, and those

present may have giant and distorted nuclei. If the lesion is of short duration there is dilatation of capillaries with swollen endothelial cells, sometimes even endothelial necrosis with fibrin formation in the lumen. In older lesions telangiectases of varying degrees may be present, and some arterioles show hyaline thickening of their walls.

Irradiated tissue does not repair readily after trauma. It is also susceptible to infection which may be intractable owing to impaired circulation and reduced viability of the irradiated cells or their dependents.

2) Generalized radiation--Such radiation is rarely experienced except in the event of a radiation accident or due to the blast of an atomic weapon. With doses of 5000 rads or over there is prompt generalized erythema accompanied by disorientation promptly followed by coma. Practically no pathologic effects will be apparent as death occurs before recognizable lesions develop. If the patient can be kept alive by supportive therapy for a few hours, profound leukopenia will develop. With lower doses, from 200 to about 5000 rads, the characteristic evidences of the acute radiation syndrome will be found,³⁰ their severity increasing with dose.

The course of the uncomplicated cases is quite distinctive. With whole-body exposure of 600 rads and more, there will be prompt nausea and vomiting followed by prostration and rapidly developing and persistent leukopenia. In the next few days there will be a rising sustained fever with diarrhea with death within 14 days. Ulceration of lymphoid tissue, such as tonsils, and a variety of purpuric manifestations may appear as death approaches. If the radiation is less severe (about 400 rads) nausea and vomiting may develop transiently a few hours after exposure to reappear

some days later. A mild lymphocytopenia may develop with a fluctuating total white blood cell count, low after a few days. Easy fatiguability persists. In about three weeks epilation and purpura may appear with bleeding from various body orifices. Oropharyngeal ulcerations are common. Diarrhea and fever appear. There is severe leukopenia with multiple infections, general sepsis and breakdown of healing wounds.

In milder cases, those receiving from 200 to 300 rads, the early symptoms will be less apparent, often only fatiguability and mild nausea, followed by a period of apparent well-being. After about two weeks there may be malaise, sore throat, petechiae, diarrhea, loss of weight,³¹ and epilation, largely of the scalp.

It appears to rest largely on chance and intestinal bacterial flora as to whether the symptoms related to hematopoietic damage or to gastrointestinal injury will predominate. Components of each are always present.

In an atomic explosion radiation injuries may very well be complicated by burns, by trauma and by infection because of damage to immune mechanisms.

Most radiation lesions have in common the interplay of their three fundamental components--the death or retarded growth of cells or cell groups, change in intercellular substance with some loss of supporting mesenchymal cells, and damage to blood vessels with the sequelae of impaired circulation. If covering epithelium is seriously damaged, ulceration usually associated with low-grade and localized infection will result. The change in cellular substance is probably mediated through damage to the supporting cells. It is first shown by localized edema followed by hyalinization of the collagen. As time progresses the surviving mesenchymal

cells tend to show abnormal shapes with enlarged and often irregular nuclei. Irradiated blood vessels may show distorted endothelial cells. If much endothelial necrosis has occurred, there may be localized thrombosis. In later stages thickening of the vessel wall develops, largely through hyalinization of collagen. Some vessels may be completely occluded. Others, predominantly capillaries, become telangiectatic. There may be loss of smooth muscle cells of the media.

The relative contributions of these various types of lesion may vary considerably according to the radiosensitivity of the structures involved. If a given tissue is relatively radiation resistant as, for example, striated muscle or the heart, radiation effect will be largely evidenced by diffuse interstitial fibrosis with varying degrees of vascular occlusion. Some atrophy of muscle fibers is usually present and may be accompanied by increase in number of nuclei. If the irradiated tissue is relatively sensitive, however, as, for example, a lymph node, the bulk of the lymphocytes will be destroyed with persistence of stroma and some stem cells from which partial regeneration may occur.

Certain specific lesions should lead one to consider past radiation injury as a possible factor. Among these are retarded local growth as a shortened limb due to damage to the epiphyseal plate in a child, or an atrophic defect in musculature or subcutaneous fat, particularly if combined with a soft pliable telangiectatic region of skin. Hematopoietic disturbances such as persistent leukopenia or anemia may be an aftermath of hematopoietic damage. Chronic ulceration has already been mentioned. Trauma to a previously irradiated site, whether accidental or operative, heals slowly with impaired fibroblastic response. Such a wound is prone

to sluggish, persistent infection with gradually progressing ulceration. Localized epilation may be the result of radiation, particularly if the epilated region is also devoid of sweat and sebaceous glands.

There are other less obviously related effects, the most important of which is radiation carcinogenesis.⁶ There is overwhelming evidence that a variety of malignant tumors can be induced by radiation. However, these are not distinctive from spontaneous tumors of the same sites. Therefore, a causal relationship to radiation can usually be determined only by past history or epidemiologic techniques. The likelihood of origin from radiation is strengthened if there is evidence of radiation changes in the otherwise normal body tissues. Cancer of the lung in uranium miners, osteogenic sarcoma and carcinoma of the bony sinuses in radium dial painters, cancer of the skin and leukemia in radiologists, cancer of the thyroid in those irradiated in early life are among the lesions that have been long recognized.

The latent period between radiation and clinical detection of a radiation-induced tumor is very variable. In the Hiroshima/Nagasaki survivors radiation-induced leukemia reached its peak incidence about ten years after exposure. Cancer of the lung in the uranium miners appeared only rarely within 15 years of occupational exposure, and iatrogenic radiation-induced cancers have been reported 30 years or more after exposure. Although benign tumors have been produced in experimental animals by radiation, their occurrence in man is apparently rare.

There is some evidence of life shortening due to radiation aside from that incidental to induced carcinogenesis. This was noted among some of the early radiologists³² and followed repeated small accidental overdoses

estimated to have aggregated a total dose of hundreds or thousands of rads over long periods of time. This shortening may be due to increase in a variety of immediate causes of death, and the mechanisms are as yet ill understood.

There is no evidence of deleterious effect within present permissible occupational dose levels.

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