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10.3.7 Radiation Jill Meara ESSENTIALS Ionizing radiation Ionizing radiation has sufficient energy to break chemical bonds and produce charged ions in living tissue. These changes might cause cell death, but breaks of both strands of a DNA molecule that do not kill a cell can be a precursor of cancer. Excluding medical exposures, natural radiation accounts for most human exposure, which produces health effects that might be (1) stochastic, where the probability of manifesting the effect depends on the radiation dose, including carcinogenesis and induction of heritable defects; (2) psychological, especially following accidental exposures; and (3) tissue reactions, occurring when sufficient cells are killed after exposure to radiation doses above a certain threshold, including the acute radiation syndrome (radiation sickness) and radiation burns. Prevention—legislative dose-limits prevent tissue reactions and reduce the risk of stochastic effects, although all doses should be kept as low as reasonably achievable. Nonionizing radiation Ultraviolet radiation affects primarily (1) the skin, causing sunburn in the short term and skin cancer in the long term; and (2) the eye, causing photokeratitis and photoconjunctivitis (arc eye, snow blindness) in the short term, and conjunctival and corneal disorders and cataracts in the long term. Information on the health effects of other types of nonionizing radiation (e.g. radiofrequency microwaves, and power-frequency electric and magnetic fields) is less robust, but controls are recommended to prevent those health effects that are established.

Introduction The term radiation applies to emissions in the electromagnetic spectrum. Only ionizing radiation is energetic enough to cause ionization of matter. There are natural sources of ionizing radiation, such as radon gas or cosmic rays, and manufactured sources, such as X-rays and radioactive

isotopes produced in nuclear reactors. Excluding medical exposures, natural radiation accounts for most human exposure. Some types of nonionizing radiation are also health hazards. These include radiant heat, ultraviolet radiation, radio waves, microwaves, and power-frequency electromagnetic fields. Historical perspective The dangers of ionizing radiation became apparent almost as soon as experiments with radioactive materials began. In the early 20th century, radiologists often calibrated their machines by the dose causing erythema on their hands. Many, including Marie Skłodowska-Curie and her daughter Irène Joliot-Curie, died of radiation-induced cancers. Despite universal exposure to natural background radiation, there is a general fear of ionizing radiation, especially that associated with nuclear power and nuclear weapons. The hazards of certain types of nonionizing radiation, such as sunburn and electrical discharge in thunderstorms, are well known, but the health of pioneers in nonionizing radiation research was not affected. Recently, the safety of power-frequency and radiofrequency fields—at the levels to which the public are exposed—has been questioned. However, hypotheses about possible long-term health effects, such as induction of cancer, lack biologically plausible mechanisms or confirmation by high-quality epidemiological studies.

Ionizing radiation: Mechanism of harm Atoms, radioactivity, and radiation Isotopes of some elements are unstable and undergo radioactive decay. The time taken for half of a given quantity to decay is called the half-life, which ranges from fractions of a second to thousands of years, depending on the particular isotope. The unit of radioactivity is the becquerel (Bq). 1 Bq equals 1 atomic disintegration per second. The natural radionuclide potassium-40 (⁴⁰K), with a half-life of 1.250×10^9 years, makes up 120 parts per million of all potassium on Earth. Since there is about 4000 Bq of ⁴⁰K in an average person, there are about 14 million radioactive disintegrations per hour from ⁴⁰K inside the average human body. Unstable isotopes (radionuclides) decay and release energy as subatomic particles (α - or β -particles) or γ -rays. X-rays are produced by bombarding a metal target with electrons in a vacuum.

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1710 Neutrons are produced during nuclear fission reactions. These vary in the extent to which they can penetrate the body and can interact with tissues and cells. α -Particles are densely ionizing and are stopped by the dead layer of the skin, but constitute a hazard if taken into the body. β -Particles can penetrate the body to the depth of a few centimetres; X- and γ -rays penetrate the body and, if not absorbed, pass through it. Lead shielding is needed to protect against X-rays and γ -rays. These properties of radiation affect the location and extent of cellular damage following exposure and dictate the protective methods required. Ionizing radiation has sufficient energy to break chemical bonds and produce charged ions in living tissue. Most of these changes are inconsequential, others can be repaired, but there is a finite probability that damage might cause cell death. Breaks of both strands of a DNA molecule might not kill a cell, but they are known to be a precursor of cancer.

Measuring radiation risk Acute cell damage depends on the energy imparted by the radiation. The mean energy absorbed per unit mass of tissue (absorbed dose) is measured in gray (Gy). 1 Gy is equal to 1 joule (J) deposited per kilogram of tissue. Radiation and tissue-weighting factors are used to convert the absorbed dose in Gy to an effective dose in sieverts (Sv). This allows external and internal exposures from all types of ionizing radiation to be integrated into one dose, on the basis of equality of stochastic risk. The United Kingdom average annual individual natural background radiation dose is 2.3 mSv. The typical dose from an anteroposterior chest radiograph is 0.02 mSv and that from an abdominal CT scan is 10 mSv. Health effects of exposure to ionizing radiation There are three types of health effects associated with exposure to ionizing radiation: stochastic effects, psychological effects, and tissue reactions.

- In stochastic effects, the probability

of manifesting the effect depends on the radiation dose and include carcinogenesis and induction of heritable defects. Radiation-induced cancer is clinically and pathologically indistinguishable from idiopathic cases. Risks at low-radiation doses are extrapolated from animal, experimental, and epidemiological studies at higher doses assuming a linear no-threshold model. This implies that there is no 'safe' radiation dose, but very small exposures convey very small risks. The absolute cancer risk per unit of radiation dose (risk coefficient) is estimated to be 5.5%/Sv. Recent data suggest that cardiovascular system damage might also be a stochastic effect of radiation, with a similar risk coefficient as for cancer induction.

- Psychological effects are found especially following accidental exposures. Readers are referred to the literature on risk communication.
- Tissue reactions (also called deterministic effects) occur after exposure to radiation doses above a certain threshold, when sufficient cells are killed. These include the acute radiation syndrome (radiation sickness) and radiation burns (Fig. 10.3.7.1). Radiation accidents are rare and the initial symptoms of radiation sickness are nonspecific, resembling influenza or food poisoning, so physicians might be involved in diagnosis and treatment before the true cause is appreciated. Patients might present to a range of different medical settings. For example, the theft of a caesium-137 (^{137}Cs) radiotherapy source in Goiânia, Brazil, led to 50 people being overexposed, and resulted in four deaths. Many people and large areas of land and property were contaminated before the true cause of the incident was appreciated. Clinical features of radiation-induced tissue reactions

External exposures, either whole body or partial, do not render patients radioactive and thus pose no radiation risk to medical attendants. If the patient has ingested or inhaled radioactive materials, or has wounds containing them (internal exposure), they and their waste products can pose a persisting radiation or contamination hazard to other people. Decontamination of radioactive material on skin or clothing is often straightforward, but should not take precedence over life-saving procedures. If contamination is suspected, contact a radiation-protection expert for monitoring and avoid spread of material. Stable iodine can be used to block uptake of radioactive isotopes of iodine. Chelating agents, such as ethylenediamine tetraacetic acid, and ion-exchange resins, such as Prussian blue, can be used to enhance excretion of certain internal radionuclides, such as ^{137}Cs and actinides. Partial-body exposures, especially of the extremities, might not be accompanied by systemic disease if the equivalent whole-body dose does not reach the symptom threshold. Symptoms of Fig. 10.3.7.1 Mature radiation burn showing central necrosis and annular epilation. Reproduced with permission from The Radiological Accident in Lilo, International Atomic Energy Agency, Vienna (2000). © IAEA, 2000.

10.3.7 Radiation burns include erythema, oedema, dry and wet desquamation, blistering, pain, necrosis, and gangrene. There are no pathognomonic features, but margins of ulcers might show epilation. Radiation burns can extend deep into the soft tissue, increasing fluid loss and risk of infection. Skin injuries evolve slowly, usually over weeks to months, can become very painful, and are resistant to treatment.

Acute radiation syndrome The acute radiation syndrome is a rare (handfuls of cases per year worldwide), multiphasic illness. The prodrome of high exposure to external ionizing radiation is sudden anorexia, nausea, and vomiting, headache, fatigue, fever, and diarrhoea, sometimes with erythema and itching, usually lasting 24–48 h. The timing of onset, severity, and duration of prodromal symptoms depend on the radiation dose. After a latent period of apparent recovery, effects of the killing of cells—especially stem cells—appear. Severity depends on the radiation dose. The main clinical features are:

- haematopoietic syndrome, at whole-body radiation doses exceeding 1 Gy—significant reductions in blood cell counts, infection, haemorrhage, and anaemia
- gastrointestinal syndrome at whole-body radiation

doses around 6 Gy—breakdown of the integrity of the gut wall leading to massive fluid and electrolyte loss and ingress of pathogens • radiation pneumonitis and the cerebrovascular syndrome (at doses exceeding 20 Gy)—respiratory failure, hypotension, and major impairments of cognitive function • radiation burns if the skin dose exceeds 20 Gy If the patient survives this phase, recovery is likely. High radiation doses can also lead to permanent sterility. Several triage categories have been published, relating the severity and time-course of symptoms and signs to prognosis. Although the threshold radiation dose for symptoms is approximately 1 Gy, lymphocyte dosimetry can detect acute doses down to about 100 mGy. Patients who also have conventional injuries have a worse prognosis. Without medical treatment, an acute dose of approximately 4 Gy is likely to be fatal within 60 days in 50% of those exposed. Doses over 10 Gy are likely to be fatal sooner, despite treatment. Similar doses over longer periods (days, weeks, and so on) might cause less severe symptoms as the body has time to repair the damage and the main concern in such patients may be the stochastic risks.

Clinical investigation This includes full history, examination, cytogenetic and regular blood tests. The estimated radiation dose is needed to predict the clinical course of the patient and plan treatment. This dose should be revised as treatment progresses because the heterogeneous nature of accidental exposures makes the scale of radiation damage difficult to estimate. Vomiting less than 2 h after acute exposure indicates a dose of at least 3 Gy. However, there is considerable individual variation and vomiting is not invariable, even at high doses. Prodromal symptoms last for more than 24 h with doses exceeding 6 Gy. The pattern of fall in blood levels of lymphocytes, granulocytes, platelets, and red cells depends on radiation dose. For pure γ -field exposures, the dose (between 0.1 and 10 Gy), follows first-order kinetics and can be estimated by multiplying the lymphocyte depletion rate constant by 8.6. Chromosome aberration assays, mainly dicentrics (chromosomes with two centromeres) in lymphocytes or other chromosomal abnormalities detected by fluorescence in situ hybridization, can be used to give a more precise estimate of whole-body dose. These assays can be used for several years after exposure.

Treatment of acute radiation syndrome Good clinical care ensures the best chance of recovery, provided that some stem cells have survived the radiation exposure. Early treatment of associated conventional injuries is important. Routine monitoring should include daily full blood counts, and blood cultures and other infection screens, especially in febrile patients. As a rule of thumb, patients with an estimated dose of 2 Gy or more should be observed in hospital and monitored for onset of acute radiation syndrome, but not all will require intensive treatment. Patients with doses of more than 4 Gy should be presumed to be developing acute radiation syndrome. Early arrangements should be made for specialist treatment. The mainstays of treatment are:

- symptomatic treatment (e.g. early wound closure, antiemetics, analgesics, and fluid replacement)
- early cytokine (colony stimulating factor) therapy
- avoiding infection by barrier isolation (or reverse isolation) with strict environmental control, oral feeding with cooked food only and meticulous hand and nail hygiene, skin, and hair disinfection and minimization of invasive procedures
- supporting affected organs until surviving stem cells multiply and repopulate the relevant organ/tissue (e.g. consider gastrointestinal decontamination, antibiotics, blood, and platelet transfusions).

Avoid antacids, proton pump inhibitors, and H₂ blockers to maintain gastric acidity; use sucralfate to avoid stress ulcers Bone marrow transplants have not been proven to be beneficial. There is weak evidence for erythropoiesis stimulating agents and haematopoietic stem cells having benefit. Haematopoietic syndrome Reverse barrier nursing and topical treatments to decrease bacterial/ fungal colonization should be used. Intravenous lines should be kept to a minimum and sited to decrease infection risk. Febrile neutropenic patients should be given broad-spectrum antimicrobials. Established infections should be treated as for other patients with

neutropenic sepsis. Early use of antifungal agents or antiviral drugs might be required to prevent late mortality. Gastrointestinal syndrome Use supportive therapy to prevent infection and dehydration. 5-hydroxytryptamine-3 (5HT₃) receptor antagonists should be used prophylactically if whole-body dose exceeds 2 Gy. Diarrhoea should be treated with antidiarrhoeals, fluids, and electrolytes. Prophylactic antibiotics should be considered. Food with a low microbial content might minimize infection risks. Enteral feeding should be used if possible.

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1712 Treatment of radiation burns Wound contamination is treated by gentle wound cleansing and debridement. Wastes arising should be treated as contaminated. Care should be taken not to break intact skin and introduce internal contamination. Systemic corticosteroids are now not recommended without a specific indication. Radiation burn treatments include: topical steroids, hyperbaric oxygen, pentoxifylline with oral vitamin E, wet dressings, alginates, hydrocolloids, and anti-inflammatory agents. Growth factors have been used to foster granulation and epithelialization. Wide excision, surgical repair, and skin grafting might be necessary by surgeons experienced in the management of chronic vascular injury. Systemic mesenchymal stem cells have been used, but need further evaluation. Combined injury Surgical correction of life-threatening and other major injuries should be carried out as soon as possible (within 36–48 h); elective procedures should be postponed until late in the convalescent period (45–60 days), following haematopoietic recovery. Surgical wounds and traumatic lacerations tend to heal more slowly in irradiated tissues. Health effects of exposures to

nonionizing radiation Ultraviolet radiation Ultraviolet radiation primarily affects the skin and the eye. The short-term skin effect is sunburn, with erythema and oedema. In some people, sunburn is followed by increased production of melanin (suntan) but this offers only minimal protection against further exposure. Acute ocular exposure to ultraviolet radiation can lead to photokeratitis and photoconjunctivitis (arc eye, snow blindness, and so on). The most serious long-term effect of ultraviolet radiation is induction of skin cancer. Nonmelanoma skin cancers, mainly basal cell carcinomas and squamous cell carcinomas, are common in white populations but are rarely fatal. The overall incidence is difficult to assess because of underreporting, but is likely to exceed 100 000 cases per year in the United Kingdom. The incidence of malignant melanoma, which is much more likely to be fatal, has increased substantially in white populations for several decades causing about 2150 deaths/year in the United Kingdom. Chronic exposure to solar radiation causes photoageing of the skin, characterized by a leathery, wrinkled appearance and loss of elasticity. Suberythral quantities of ultraviolet radiation are beneficial in stimulating vitamin D synthesis in the skin. Vitamin D has been associated with several musculoskeletal and nonmusculoskeletal health outcomes, including hypotheses about protection from cancer. Overall, there is insufficient evidence on vitamin D and nonmusculoskeletal health outcomes to set adequate vitamin D intakes from dietary or UV sources over and above those established to prevent musculoskeletal disease. Repeated ocular exposure is a major factor in corneal and conjunctival diseases, such as climatic droplet keratopathy, pterygium, and, probably, pinguecula. Cumulative exposure to ultraviolet radiation is a major cause of cortical cataracts, but its importance in the general population remains uncertain. Immune responses Exposure to ultraviolet radiation can suppress immune responses by complex mechanisms, but the significance for human health and response to vaccinations is uncertain. Radiofrequency electromagnetic waves The widespread adoption of radiofrequency microwaves in wireless technology, including mobile phones and wi-fi, has led to concerns about adverse health effects. High exposure to radio frequencies can cause thermal burns. There is no evidence that there is significant risk to the general public from exposure to radiofrequency

radiation or from use of micro/radiowave appliances. However, these are new technologies and a cautious approach is appropriate because of the lack of scientific evidence. Public Health England (PHE) recommends that children should use mobile phones only for important calls. Power-frequency electric and magnetic fields There are concerns that power-frequency electric and magnetic fields might have adverse effects on health even at levels below those required to interfere with nerves through induced fields and currents. The evidence is controversial. However, epidemiological studies had shown a consistent statistical association—not necessarily indicating causation—between unusually high background magnetic fields in homes and/or residential proximity to power lines and increased risk of childhood leukaemia (possibly 2–5 attributable cases per year in the United Kingdom). This prompted the International Agency for Research on Cancer to classify power-frequency electric and magnetic fields as ‘possibly carcinogenic’. In March 2004, the United Kingdom Health Protection Agency (now PHE) recommended that the government should consider precautionary protection from power-frequency electric and magnetic fields. More recent studies have failed to confirm this association. Static magnetic fields Head movements in static magnetic fields stronger than 2 T can cause symptoms such as vertigo, nausea, a metallic taste, and phosphenes (seeing light without light entering the eye). Humans undergoing MRI (magnetic resonance imaging) are exposed to static magnetic fields exceeding 2 T. There are insufficient data to indicate long-term health effects of exposures to static electric and magnetic fields. Stronger fields should be used with care in controlled or experimental situations with more rigorous patient monitoring. Limits have also been advised for switched gradient and radiofrequency exposures from MRI. FURTHER READING Bennett P, et al. (eds) (2010). Risk communication and public health, 2nd edition. Oxford University Press, Oxford. Dainiak N, et al. (2011). First global consensus for evidence-based management of the hematopoietic syndrome resulting from exposure to ionising radiation. Disaster Med Publ Hlth Preparedness, 5, 202–12.

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