WHAT EVERY MEDICAL OFFICER SHOULD KNOW ABOUT THE ATOMIC BOMB

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IV. Evaluation of the Five Atomic Explosions

Employment of the bomb. The atomic bomb is primarily a strategic weapon, and the choice of target and method of employment require the evaluation of a number of factors. Thus far, five atomic bombs have been detonated, three of them under test conditions. The one factor that makes an atomic bomb detonation different from the detonation of any other type of weapon is the nuclear radiation produced. All high-explosive weapons produce high temperature and high blast pressure, and the only difference in these respects between atomic and conventional weapons is the increased magnitude of the blast and thermal effect produced by the atomic bomb. However, no other weapon devised to date is capable of releasing nuclear radiation.

The first bomb was set off under experimental conditions from a tower near Alamogordo, New Mexico, on 16 July 1945. The second bomb was dropped, 6 August 1945, on the city of Hiroshima from a B-29 bomber. Over 4 square miles of the city were instantly and completely devastated; 66,000 people were dead or missing and 69,000 were injured. On 9 August another B-29 dropped an atomic bomb on Nagasaki, totally destroying 1.5 square miles of the city. The number of persons dead and missing in Nagasaki was 39,000, and 25,000 more were injured. The fourth atomic bomb was dropped by a B-29 on target vessels assembled in Bikini lagoon on 1 July 1946, and the fifth was detonated underwater on 25 July 1946. Test animals placed in various locations on the target vessels yielded important data on the bomb effects. This work was under the supervision of the Naval Medical Research Center.

Action of the bomb. When a mass of fissionable material equal to or greater than a critical size is assembled, a violent detonation will occur. The subcritical masses of fissionable material must be brought together rapidly in such a manner that a chain reaction and detonation will occur. The bombardment of each fissionable nucleus by neutrons results in the formation of two fragments known as fission products. All nuclei do not split into the two types of fragment; therefore, many radioactive substances (fission products) are liberated. The sum of the masses of these fission products will not equal the original mass of the split nuclei. The difference between the fission products formed and the original mass represents the mass of the nuclei that has been converted into energy in the form of blast, heat, light, x-rays, gamma rays, and released nuclear particles.

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The detonation of the atomic bomb generates a crushing wave of high pressure. The bomb also liberates an enormous quantity of electromagnetic radiations and neutrons. The electromagnetic radiations include infrared, visible light, ultraviolet, x-ray, and gamma radiation. Thereafter, the fission products formed emit gamma rays and beta particles. The unfissioned bomb residue emits alpha particles. Substances bombarded by neutrons released at detonation, which become radioactive by induced radioactivity, may also emit nuclear particles and gamma rays. A large fraction of the gamma rays is emitted in the first flash of the atomic explosion. Neutrons also accompany this reaction. The range of neutrons is negligible at 1,000 yd. because of their absorption in the air. In an underwater burst, greater absorption occurs, resulting in induced radioactivity of the sea water. Of the constituents of seawater, only sodium is of any significance, and even this element is hazardous for only a limited period because of its short half-life (14.8 hours).

At detonation, practically all of the lethal gamma radiation is released, and the remaining small fraction of the total dose is given off by the resultant fission products that rise rapidly in the bomb cloud. The column of radiating fission products and combustion material rapidly rises into the air and begins to mushroom out when the temperature of the column is equal to the temperature of the surrounding atmosphere. The climatic and meteorologic conditions will govern the diffusion, dispersion, and radiation activity of the cloud. The fissioned and unfissioned material in an airburst will be distributed in the atmosphere; while in a subsurface waterburst, the adjacent water, ships, and land facilities in proximity to the detonation will be seriously contaminated. Fission products in the cloud may be dispersed as fine particles of varying size, and, depending on many factors, a shower of the radioactive material will fall on nearby areas. The fission products, therefore, present a continuing health hazard for a considerable time as an aftermath of the explosion. In general, regardless of the technique of bomb detonation, radioactive materials emitting alpha and beta particles and gamma rays will be encountered. The radioactivity of these substances will range from a few seconds to years. Violent changes in temperature, strong magnetic or electric fields; and drastic chemical interactions have no effect on the rate of transformation or emission characteristics of the radioactive substance. If an element is radioactive, it will decay normally according to its inherent half-life.

In the underwater detonation of the bomb, thousands of tons of water rise in a column, a few thousand feet in the air, followed immediately by a rapidly moving mass of water, constituting the base surge. The turbulent waters contain a high percentage of the fission products and unfissioned residue. Immediately at detonation and for a short period thereafter an enormous amount of radiation is emitted. The falling column of water and mist, depending on wind conditions and depth of detonation, contains a high percentage of the fission products and unfissioned residue that can contaminate an area of several square miles for a considerable period.

The emission of infrared, visible, and ultraviolet light occurs a few milliseconds after the explosion. The ball of fire in the airburst grows rapidly in
size. As it grows, its temperature and brightness decrease. Several milliseconds after the initiation of the explosion, the brightness of the ball of fire is several times the brightness of the sun. Most of the infrared and ultraviolet radiation is given off after the point of maximum intensity. The ball of fire rapidly expands from the size of the bomb to a radius of several hundred feet at one second after the explosion. Thus, the infrared and ultraviolet radiation comes in two bursts—an extremely intense one lasting a fraction of a millisecond and a less intense one of much longer duration lasting several seconds.

The heat from the flash in an airburst occurs in a short time, and, since there is no time for any cooling to take place, the temperature of a person’s skin can be raised 50° C. by the flash of infrared and ultraviolet rays in the first millisecond at a distance of over 4,000 yards. People may be injured by flash burns at even greater distances. Gamma radiation danger does not extend nearly so far, and the neutron danger zone is still more limited. High skin temperatures result from the first flash of high intensity infrared and ultraviolet and are probably as significant for injuries as the total doses that come mainly from the second, more sustained, ball of fire.

Effectiveness against personnel. For personnel in the open, within one-half mile of zeropoint of the airburst detonation, death would occur almost instantaneously or within a few hours from the blast, heat, and radiation effects. Within a radius of one-half mile and one mile from zeropoint, some persons would die instantly, while a majority would receive varying degrees of injury. Ordinary houses and structures would suffer complete destruction or extensive damage and fires would be widespread. Outside a radius of one mile and within a radius of two miles from zeropoint, personnel would suffer injuries from flash burns and indirect blast effects. Outside a radius of two miles and within a radius of four miles, personnel would be injured by flying fragments and suffer superficial wounds. Structures would be half or partially destroyed within this radius. In an airburst explosion 70 percent of those exposed would suffer from trauma, 65 percent from burns, and over 35 percent from radiation.

The radiologic hazard. In general, any radioactivity that remains in the area as fission products or induced radioisotopes will constitute a hazard. Fission products from the airburst bomb may be dispersed in the ground or spread out over wide and diffuse areas, depending on the technique employed in the detonation. Consequently, the degree and extent of residual radioactivity would depend on the height of detonation, climatic and meteorologic conditions conducive to the showering of the products on a specific area, and the nature and composition of the terrain. For example, because of the height of the detonation, certain prescribed areas of the bomb crater might remain hazardous. Also, because of the composition of the ground, dust particles intermixed with fission products might rise in the cloud. Many of these “dust particles” might also become radioactive as a result of neutron bombardment released at detonation and thus contribute to the hazard.

When the bomb is detonated over a modern city that contains countless thousands of items composed of iron, zinc, copper, and other “neutron capture materials,” it is possible that many of the elements within the effective neutron
range may become radioactive for a considerable period. The half-lives of some of these common elements and the radiations emitted are listed in Table I. Therefore, objects or material that might survive the detonation, such as medical supplies containing sulfur or arsenic, should be handled with caution until the degree and extent of the induced radioactivity is determined. In some cases, it is possible that fission products also are present and are adhering to the material. In an underwater burst the main hazard, following detonation, will be the result of the deposition of a large percent of the fission products in the water and on nearby objects. In addition, radioactive sodium is formed by the action of neutrons on the sea water. Some of the more persistent and hazardous fission products of U–235 are listed in Table II.

**Table I**

*Partial List of Some Common Radioisotopes that may be Produced by Neutrons Released at Detonation*

<table>
<thead>
<tr>
<th>Radioisotope</th>
<th>Half-life</th>
<th>Radiation</th>
</tr>
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<tbody>
<tr>
<td>Sodium-24</td>
<td>14.8 hours</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Sulfur-35</td>
<td>87.1 days</td>
<td>Beta</td>
</tr>
<tr>
<td>Calcium-45</td>
<td>180 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Iron-59</td>
<td>47 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Cobalt-60</td>
<td>5.3 years</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Copper-64</td>
<td>12.8 hours</td>
<td>Beta</td>
</tr>
<tr>
<td>Arsenic-74</td>
<td>16 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Gold-199</td>
<td>3.3 days</td>
<td>Beta, gamma</td>
</tr>
</tbody>
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**Table II**

*Partial List of Fission Products of U–235*

<table>
<thead>
<tr>
<th>Fission product</th>
<th>Half-life</th>
<th>Radiation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strontium–89</td>
<td>53 days</td>
<td>Beta</td>
</tr>
<tr>
<td>Strontium–90</td>
<td>25 years</td>
<td>Beta</td>
</tr>
<tr>
<td>Yttrium–91</td>
<td>57 days</td>
<td>Beta</td>
</tr>
<tr>
<td>Zirconium–95</td>
<td>65 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Columbium–95</td>
<td>35 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Ruthenium–103</td>
<td>42 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Ruthenium–106</td>
<td>1 year</td>
<td>Beta</td>
</tr>
<tr>
<td>Cadmium–115</td>
<td>44 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Cesium–137</td>
<td>33 years</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Barium–140</td>
<td>12.8 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Cerium–141</td>
<td>28 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Cerium–144</td>
<td>275 days</td>
<td>Beta</td>
</tr>
<tr>
<td>Neodymium–147</td>
<td>11 days</td>
<td>Beta, gamma</td>
</tr>
<tr>
<td>Europium–155</td>
<td>2 years</td>
<td>Beta, gamma</td>
</tr>
</tbody>
</table>

The radiologic hazard can be divided into two phases. The first phase includes the immediate or prompt release of any ionizing particles or radiations caused by the explosion during the period of visible flash of the bomb. These prompt ionizing radiations include beta particles, neutrons, x-rays, gamma and alpha particles from unfissioned bomb residue, and the ionizing radiations from fission products. After the flash of the bomb has subsided, a matter of a few seconds, the delayed phase of the radiologic hazard is of importance. The hazard here is from fissioned and unfissioned material and from radioactive elements induced by neutrons from the explosion. The nature and persistency of the second phase depends on the technique of detonation. In addition to the phase of the radiologic hazard, the protection problem depends on whether the radiation concerned is external or internal to the body. Alpha particles, for example, present no external hazard; but if they are inhaled and become fixed in the bone, depending on the amount, the results may be lethal. Although very little can be done to protect personnel in the open within the lethal range at the instant of detonation, a few points in connection with the second phase may be useful. A comparison of radiations is given in table III.

The relative protection against gamma radiation by shielding, in order of effectiveness, is given by lead, iron, concrete, earth, water, and air. Using the gamma radiation from radium as an illustration, a 5 in. thickness of concrete gives about the same protection as a 1 in. layer of lead. Where no shielding is available, "distance" is the best means of protection. It should be noted that neutrons pass through lead with extreme ease, but are readily absorbed by hydrogenous materials and boron.

The flash burn. At detonation, the flash burns from infrared and ultrared caused a higher percent of casualties than the radiologic effect, because of the increased range of the flash. Light shades of loosely fitting clothing, antiflash cream, and protection of the entire body surface will reduce the percent of casualties. Protection by these means will not reduce the effects of burns produced by secondary fires in buildings or facilities. The problem here is to minimize the amount of inflammable material as far as practicable. In this

<table>
<thead>
<tr>
<th>Nature</th>
<th>Description</th>
<th>Range</th>
<th>Ionizing power*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha</td>
<td>Helium nucleus (2 protons and 2 neutrons)</td>
<td>0.1</td>
<td>10,000</td>
</tr>
<tr>
<td>Beta</td>
<td>Electron emitted from nucleus</td>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>Gamma</td>
<td>Electromagnetic radiation from nucleus</td>
<td>1,000</td>
<td>1</td>
</tr>
</tbody>
</table>

*Note: For each ion pair formed by a gamma ray, 10,000 ion pairs are formed by an alpha particle.
connection, materials that ignite easily should be avoided in the design of equipment intended for military operations. Flash burn is not a serious factor in an underwater detonation.

**Summary**

Air-burst atomic bombs will produce lethal effects over an area of two square miles and measurable effect over an area of seven square miles as a result of the prompt gamma radiation emitted at the time of detonation. The residual radioactivity is of little importance except in the area close to the center of a low-altitude explosion. In an underwater detonation, radioactive fission products and un fissioned material will be spread by the cloud and base surge over a large area. The gamma radiation from these materials will be lethal to exposed personnel more than two miles downwind, and serious contamination will result at much greater distances. This contamination will provide a serious hazard for an indefinite period. Prompt evasive action at the time of the detonation will permit the reduction of casualties, and orderly evacuation and re-entry procedures will undoubtedly pay great dividends in minimizing the effects.

**V. Fundamentals of Radiation Pathology**

The pathologic effects of radiation can best be presented by outlining the early and late changes in (1) tissue cells, (2) organ systems, (3) total body irradiation, and (4) internal radiation by radioactive materials introduced into the body either accidentally or therapeutically. Sensitivity of the various body tissues has been well established, and has been expressed largely as the relation of one tissue to another. Table I shows the relative sensitivities as indicated in two studies.

<table>
<thead>
<tr>
<th>Relative Radiosensitivities of Various Body Tissues Listed in Decreasing Order</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desjardins*</td>
</tr>
<tr>
<td>Lymphocytes</td>
</tr>
<tr>
<td>Granulocytes</td>
</tr>
<tr>
<td>Epithelial cells</td>
</tr>
<tr>
<td>(a) Basal cells of secretory glands</td>
</tr>
<tr>
<td>(b) Basal cells of testes and ovarian follicles</td>
</tr>
<tr>
<td>(c) Basal cells of skin and gastrointestinal tract</td>
</tr>
<tr>
<td>(d) Alveolar cells of lungs; bile ducts</td>
</tr>
<tr>
<td>(e) Tubules of kidneys</td>
</tr>
<tr>
<td>Endothelial cells</td>
</tr>
<tr>
<td>Connective tissue</td>
</tr>
<tr>
<td>Muscle cells</td>
</tr>
<tr>
<td>Bone cells</td>
</tr>
<tr>
<td>Nerve cells</td>
</tr>
</tbody>
</table>


Reactivity of the tissues in terms of energy or actual ionizing effect from a quantitative standpoint is somewhat less definite. Variation in response to ionizing radiation has been indicated in numerous studies, but becomes of particular interest in total body radiation, since in this circumstance the variation is not only a question of death or survival over a relatively broad range of radiation dosage, but also manifests itself as well by variations in organ responses, presumably by an equally wide range in symptoms and clinical findings. The effects of ionizing radiation are considered at present to be similar for all types of radiation—alpha, beta, gamma, x-ray, and neutron sources—when equal amounts from the standpoint of energy and time relationship are absorbed in the tissues.

*Tissue cells.* There is no satisfactory indication of any tissue effect of radiation other than destruction. In prolonged exposures of animals to tolerance and slightly higher levels, survival rates were higher in the exposed groups than in the controls. This same tendency was noted in weight curves. The exposed animals showed weights consistently above those of the controls, mostly from abdominal fat. This was considered not to be a castration effect. From a morphologic standpoint, however, the purely destructive effect has been emphasized in a recent report by Bloom.  

It is well to keep in mind that it is unlikely that all tissue has been subjected to the ionizing action of radiation. Microscopically, any one or all of a number of cellular changes may be observed, such as:  
(1) changes in staining characteristics, usually an increase in eosinophilic properties;  
(2) increased granularity, usually of cytoplasm;  
(3) vacuolation of a variable degree;  
(4) swelling of cellular components;  
(5) distortion of cellular structures;  
(6) cytolysis (loss of definitive borders);  
(7) pyknosis;  
(8) changes in Golgi's apparatus;  
(9) reduction in mitotic activity;  
(10) production of abnormal mitoses;  
(11) chromosomal changes (fragmentation, clumping);  
and  
(12) increased refractile neutral red staining bodies within leukocytes seen by vital staining methods. These changes are found in conditions other than radiation, and although highly suggestive are not specific. Alterations in the noncellular tissue may include intercellular edema, swelling and hyalinization of colloid, and swelling and fragmentation of elastic tissue. A more direct approach to the cellular changes is found in observations on cellular viscosity, ciliary action, phagocytosis, cellular secretions, and a few enzyme systems that can be demonstrated. Alterations in these processes have been described following radiation.

Initially no changes may be found. Alterations in viscosity and slightly increased acidophilic staining properties are among the earliest findings. Cessation of mitoses and destruction of lymphocytes may occur in a matter of hours or less. Vascular dilatation and edema may follow, and, in the case of larger doses, actual necrosis of tissue cells may occur, again depending on the relative sensitivity. These represent only the readily demonstrable changes, and are certainly an inadequate and relatively crude index of the tissue alterations.

In small or moderate doses recovery may occur with no residual lesion, may show the frequent pattern of repair by fibrous tissue replacement, or, in other instances, may show the pattern of repair characteristic of the organ. There is no indication that the features of repair are specific or characteristic for any or all types of radiation. References to "radiation fibroblasts" and "radiation dermatitis" lead one to assume that these are peculiar to radiation injury, although such is not the case; but these designations are useful in evaluating tissue damage and probable etiology. The recovery stage in terms of tissue repair is often a matter of months or years and, in the case of repeated or continuing exposure, becomes a much more important problem.

The late effects, in most instances involving repeated exposure to radiation, are well established and include: (1) atrophy and ulceration of the skin, telangiectasia, fibrosis, and vascular occlusion, which were early recognized as radiation effects; (2) carcinoma of the lung, which in the Schneeberg mines was considered to be due to the radioactive material present in the inspired air; (3) bone sarcoma developing in persons ingesting radium; (4) carcinoma of the skin as a late effect of repeated exposure to x-rays; (5) leukemia, which has an increased incidence in those exposed to repeated radiation; and (6) other effects such as genetic variation and shortening of the life span.

**Lymphoid tissue.** Changes in the lymph nodes have been described by many investigators. Relatively small doses produce in a short time nuclear degeneration of lymphocytes and some distortion of the germinal centers. Congestion, swelling, and slight inflammatory cellular infiltration may occur. Mitoses are not seen until regeneration becomes active. Continued cellular degeneration is followed by increasing and active phagocytosis by large macrophages. Erythrophagocytosis occurs in addition to the phagocytosis of nuclear and cellular fragments. The inclusion of red blood cells in the macrophages is an early finding, the significance of which is not well understood. Repair following small doses is rapid and apparently complete. Somewhat greater doses result in a marked reduction in lymphocytes, leaving an almost empty reticular stroma with the persistence of a few small lymphocytes and a few larger reticulum-type cells associated with the germinal centers. Repair may take place, if the destruction has not been too great, apparently from the remnants of such centers, often with definite irregularity in the size, shape, and pattern of the lymph node. If the damage has resulted in almost complete destruction, the area may consist of more or less condensed stroma and loose connective tissue containing a few scattered lymphocytes. Such areas are said to offer no resistance to lymphoid circulation.

The spleen is less sensitive than the lymph nodes and regenerates less completely. A similar cycle of changes occurs. Loss of the lymphocytes may result in condensation of the stroma, and an accentuation of the reticular and sinusoidal pattern occurs. Regeneration, if it takes place, may show considerable irregularity in the cellular forms. Phagocytosis is active, and quantities of pigment may be present in the spleen after recovery. As in the lymph nodes, regeneration appears to proceed from the remaining reticulo-endothelial elements. The heavy accumulations of pigment have been interpreted as
evidence of excessive blood destruction, or failure of splenic tissue to dispose of the material, or both. The thymus shows changes of a similar nature, although phagocytosis and disposition of pigment are not seen as in lymph nodes and spleen.

**Bone marrow** is more resistant to radiation than lymphoid tissue. Destruction of cells appears to involve both the immature granulocytic and erythrogenic forms. Regenerative changes are seen early, within the first week. Pigment deposits, eosinophils, and plasma cells may appear. With particularly heavy irradiation, almost complete loss of cellular elements may occur, with only a few reticulum cells and perhaps an occasional focus of erythropoietic cells. The marrow in such cases possesses a peculiar gelatinous appearance, grossly, with a deceptive red coloration arising from red blood cells within dilated vessels or dispersed extravascularly. Such marrow may regenerate adequately, or may result in an aplastic marrow with variable amounts of connective tissue. In the case of ingested radioactive material, any stage of hyperplasia or aplasia may be found, depending on intensity and distribution.

The peripheral blood picture does not indicate in adequate fashion the processes occurring in the marrow. For example, the apparent paradox occurs in which a hyperplastic marrow is present with a relatively low count in the peripheral blood, which is found in conditions other than radiation effect. In these cases there is usually some lack of maturation within the marrow. This introduces the question that has been asked a number of times in the literature: What factors determine whether a given hematopoietic system, when subjected to repeated demands, stimulation, or insults, will respond by hyperplasia or aplasia? Warren cites the histories of two chemists working with radioactive substances over a period of years in the same laboratory. They observed no protective measures and died within five days of each other—one of aplastic anemia, the other of myelogenous leukemia. One can observe such cases clinically and encounter stages at which one is unable to indicate whether the case will progress to leukemia or to an aplastic anemia. Again this is not a situation peculiar to patients exposed to irradiation. Several well-known characteristics of radiation are shown by the marrow. One is the destructive effect on tissues elsewhere in the body, when exposure is limited to a relatively small area, another is the cumulative action of radiation. In successive exposures, the radiation necessary to show definite effects becomes less, and the periods necessary for recovery become longer. This has been expressed in the term “percentage recovery” for certain exposure.

**Gonads.** The reaction of the cellular elements of the seminiferous tubules to radiation varies. There is evidence to indicate that the primary spermatocytes are the more sensitive, contrary to the general statement that more primitive cells are more sensitive. Next in order of disappearance are the spermatogonia, small spermatocytes, spermatids, and spermatozoa, with the Sertoli’s cells remaining and proliferating to replace the germinal epithelium.

In other instances the spermatogonia, the most immature germinal cells, have been observed to be the only ones persisting. The interstitial cells have been generally regarded as resistant to radiation. The ovaries are less sensitive than the testes. Maturing follicles have been described as the most sensitive portion, and corpora lutea as relatively resistant. In mice, development of ovarian tumors following irradiation in the tolerance levels has been described.

**Gastrointestinal tract.** Edema and degenerative changes in the epithelial cells occur early. Subsequent changes may include hyperemia, hemorrhage, cellular changes progressing to necrosis, often with a thick superficial fibrin membrane, and subsequent ulceration. Mitotic figures and atypical cellular forms are seen within a week and are considered to be regenerative in nature, although closely resembling degenerated cells. These early epithelial changes in the gastrointestinal tract have been linked with the profound toxic changes. Connective tissue areas of the walls of the gastrointestinal tract show edema and myxomatous and hyaline changes, the same areas often containing bizarre connective tissue cellular forms. Later effects include fibrosis, atrophic changes in the mucosa such as reduction in the number of glands, and in the gastric mucosa a reduced number of chief cells. Ulceration is a relatively frequent occurrence after an extended period.

**Respiratory organs.** Pulmonary tissue is considered moderately sensitive to irradiation. A transient pneumonitis occurs, without apparent late effect. No significant changes have been described in the bronchial system.

**Skin.** The essential features include an early erythema occurring within a few hours to a few days, disappearing within a period of days, followed by a second occurrence of erythema ten days to four weeks later. This second episode represents the culminating pathologic change in the connective tissue and vascular bed of the corium, in contrast to the more direct injury to epithelial cells resulting in the early erythema. Pigmentation, epilation, and ulceration may follow with destruction of dermal glandular structures. Atrophy, hyperkeratosis, and telangiectasia may develop after repeated small doses without the preceding clinical manifestations and with the possibility of malignancy. The histologic picture is characteristic. The epithelium is thin, with obliteration of rete pegs. Irregular acanthosis may be present with cellular abnormalities. The corium shows dilated vascular spaces, atrophic skin appendages, dense and hyalinized collagen with variable basophilia, and reduced or absent elastic tissue.

**Other organ systems.** The epiphyseal region of infants and children is particularly reactive to radiation. In the eye, radioconjunctivitis occurs with moderate doses and may be followed by keratitis. Lenticular opacity occurs in young eye tissues with moderate doses, as compared with the greatly increased doses necessary in mature lenticular structures. Tissues that have not been discussed are generally in the less reactive range and undergo few changes except in massive localized exposure. To this group belong nerves, heart, liver, pancreas, bone, and muscle.

**Total body-irradiation.** Doses used commonly, such as the erythema dose, approach or exceed the lethal dose when applied to the entire body. It is of
considerable interest to define the changes at various levels of total body irradiation, and a certain clinical experience is available, as well as numbers of animal studies. Early and rather striking changes have been described in the gastrointestinal tract of animals dying of total body irradiation, with relatively slight changes elsewhere. Survival for a longer time places the organism in a period in which vascular damage and hemorrhagic phenomena are outstanding. The generalized destruction of hematopoietic tissue is a major factor at this and later stages. The findings at later stages are those of severe infection without adequate cellular response, and presumably without adequate resistance.

**Internal radiation by radioactive substances** does not involve any differences from the tissue reactions described, other than those associated with localization and intensity. The action of radioactive substances internally depends on (1) the activity of the substance ingested, whether an alpha, beta, or gamma emitter, and the duration of its activity; and (2) behavior in the body—rate of excretion, affinities for certain tissue, and its course of localization. For example, radioactive sodium-24, which is highly diffusible in the body, gives the pathologic picture of total body irradiation from an external source. The localization of many of the radioactive materials in relation to bone has intensified their effect. The lesions in radium poisoning may be used as an example—bone necrosis, particularly in the jaw, destruction of marrow with variable hyperplastic and aplastic changes, and the incidence of malignancy in the form of bone sarcoma. The amount of radioactive isotopes required to produce bone sarcomas, lymphomas, and the like in animals is practically identical with that required to produce perceptible effects in the peripheral blood.

**VI. Pathologic Anatomy of Radiation Effects of Atomic Explosion**

Before considering the radiation effects on the systems of the body, it is important to consider the relationship of lesions and time of death. In Japanese patients dying within two weeks after exposure there was histologic evidence of radiation in the bone marrow, gonads, gastrointestinal tract, and skin that was not manifested clinically. In the group dying in the third to sixth weeks, bone marrow changes predominated, while neutrophenic ulcers and hemorrhagic symptoms were very common. The general nutritional state declined. Gross changes were at the peak. Those dying in the third and fourth months showed beginnings of recovery in bone marrow and hair regeneration. Testicular and connective tissue changes remained evident. There was an increase in the number of emaciated patients. The poor nutrition was not based entirely on shortage of food. Intestinal lesions and other factors played an important part.

**Skin.** The quickly visible changes in Japanese affected by an atomic bomb were the pigmented areas that appeared in the first few weeks and persisted. These had such sharply demarcated outlines that they were considered as flash burns. Whether very soft, nonpenetrating gamma rays played a role has not been determined. Development of what we have recognized as ionizing ray skin burns was not seen. There were a few early cases of bullous
edema that may have been from gamma rays. Epilation appeared mainly on
the scalp, occasionally more on one side than the other; in the axilla in 16
percent; in the pubic region in 12 percent; and in the eyebrows in 8 percent.

Microscopically the hair follicles showed distinct changes both in the
epidermal and dermal coats. Early specimens were not obtained, but in the
fourth week the internal root sheaths could not be identified, the external
sheath (continuous with the malphigian layer of the epidermis) being con­
tinuous with the hair shaft. Vascularity of the papillae was reduced, and the
adjacent epithelium was atrophic. Pigment was irregularly clumped. The
dermal coat showed thickening both of the inner hyaline membrane and the
cellular fibrous layer. In pushing the base of the hair toward the surface a
continuous shrinking in the bottom of the follicle occurred until regeneration
took place with new cells over the papillae in a manner similar to ordinary
hair replacement. There was also atrophy of the sebaceous glands, but this
was also present when old hairs were replaced in the normal individual.

Some of the sweat glands were small, their cells occasionally vacuolated
and pyknotic, and the basal membranes thickened. Evidence of radiation on
the skin was not definite. Third degree flash burns could also be expected to
have some radiation effect, but interpretation was difficult. At the edge of the
burn area there was hyperpigmentation in basal cells and chromatophores.
Some thinning of epidermis, hyperkeratosis, ironing out of papillae, and
hyperpigmentation of basal cells were found in the scalp. Vascular and collagen
changes were minimal.

Pituitary. Large basophilic cells with much cytoplasmic vacuolation
appeared in 25 percent of the males dying in the third to sixth weeks. Because
cells of this type are found in mammals after castration, they are known as
“castration cells.” In the second and third months large basophils were
found, only a few being vacuolated.

Adrenals. In the first two weeks there was a loss of lipoid in the cortex,
but in the next months the cortex progressively lost its orange-yellow color
and was distinctly thin. Microscopically, most cells were granular rather than
foamy, and the atrophy was most marked in the outer zona glomerulosa, con­
trary to what was expected. When foam cells were present, they were usually
in the inner layer. The medulla was normal.

Heart. Epicardial petechiae were found within the first two weeks, and
there was microscopic evidence of some perivascular and rare muscle edema
in the myocardium. These changes continued to be present during the second
month when myocardial hemorrhages were also seen. After the second month
no distinct irradiation changes were found.

Lungs. Only the slight perivascular edema of the pleura that appeared in
the first two weeks might be a primary radiation effect. Hemorrhagic and
necrotizing pneumonia were common after the first weeks, as secondary lesions.

Genitourinary system. Except for hemorrhagic manifestation, there were no
primary lesions in the kidneys and ureters. In the hemorrhagic stage of the
radiation disease, mucosal hemorrhages in the bladder might result in necro­
tizing ulceration without evidence of leukocytic infiltration. The prostate and
seminal vesicles were not remarkable, except for a rare neutropenic necrosis and the presence of a few spermatozoa that were morphologically normal in spite of the irradiation.

The testes showed intense changes in almost every cadaver. As early as the fourth day when the parenchyma had a normal appearance grossly, the histologic sections presented marked injury of the germinal epithelium, numerous cells of which were necrotic and free in the tubules and even in the rete testis. The number of mitoses was small. Sertoli's cells were increased in number. Mature spermatozoa were found even in later specimens with no spermatogenesis. Apparently uninjured spermatozoa appeared in the seminal vesicles. In the second month gross examination revealed little. A few necrotic germ cells remained, but most had disappeared, and phagocytic or infiltrating inflammatory cell activity was absent. A few bizarre cells still approximating the basal membrane appeared to be spermatogonia. Sertoli's cells were more numerous. The tubules had started to shrink. At this time also the interstitial cells of Leydig were so prominent that some interpreters considered them hyperplastic. Some of the small interstitial vessels showed the most marked vascular change of any part of the body. Beneath the distinct thin endothelium was an eccentrically located mass of eosinophilic, homogeneous, refractile material that almost occluded the lumen. This change was often best seen near the tunica albuginea and was present also in the third and fourth months. The interstitial tissue was less, but still prominent. The basement membranes were quite thick, wavy, and acellular. The tubules, more atrophic at this stage, were often hyalinized. Elsewhere Sertoli's cells had replaced the germ cells, which were rare. In the third and fourth months the state of nourishment was poor and specimens from the Dachau prison camp in Germany have been described as showing similar testicular changes.

Changes in the ovaries were much less striking. Gross changes, except as part of the hemorrhagic phenomena, were absent, even to the presence of a well-developed corpus luteum of pregnancy seen about the end of the first month after irradiation. Histologically, primary ova were usually present and only occasional specimens had a few atresic primary follicles. The absence of developing follicles was usual. There were no corpora lutea and the "resting phase" of the endometrium reflected this. Amenorrhea was distinctly increased in Nagasaki, and a significant number of abnormal births and an increased death rate of the mothers in relation to distance from the explosion were found there.

Gastrointestinal tract. This tract was one of the first to show gross lesions. Even before hemorrhagic manifestations the cecum or colon, particularly, might present a widespread change marked by swelling, green and yellow-gray coloration, and induration of the mucosa, sometimes with a pseudomembranous effect, and with much submucosal edema. Later mucosal hemorrhage might institute another cycle of similar change in the stomach or intestine. This change might begin with ulceration of the mucosa at the site of the hemorrhage and progress to a pseudomembrane or deep ulcer. Again, in the third and fourth months an enteritis, usually in the large intestine but sometimes affecting the
small intestine and occasionally the stomach, might be the most prominent lesion. In the small intestines only the tips of the folds might first be involved. These looked at first as though they had been dipped in boiling water and then became green or yellow-gray. A few specimens of small intestine had a diffuse mucosal process. The large intestine in this late stage usually had a more widespread process that might extend from the ileocecal valve to the rectum. The thickened wall was characteristic. A pseudomembrane and ulceration were sometimes present so that the morphology was similar to that of bacillary dysentery. Much of the process here was not only an irradiation effect of the sensitive intestine, but also a result of the lowered local ability to cope with intestinal microorganisms and, probably more important, to the lowered antibiotic capabilities of the blood.

Microscopically the epithelium early contained extremely bizarre cells with giant hyperchromatic nuclei and multipolar mitoses. The swelling was seen to be from edema and the peculiar coloration from the absence of infiltrating leukocytes. Later, areas of mucosal ulceration with much fibrin, few leukocytes, and in the remarkably edematous submucosa quite a few histiocytes, a few lymphocytes, and occasional eosinophils were seen. Plasma cells of the lamina propria remained numerous.

Spleen. The lymphoid elements here reacted to radiation as in the nodes. Early spleens were usually small, but occasionally showed the early swelling reaction. On section, they were dark red and firm, the follicles were indistinctly seen, and the trabeculae were prominent. Besides the near absence of lymphocytes, large mononuclear cells were increased, and erythrophagocytosis and hemosiderin deposits were seen. In the second month the spleen was small and follicles were absent. There was a syncytial reticulum around the follicles in which the slight lymphocytic content of the organ was seen. Atypical large mononuclears were found in about 25 percent. Through the fourth month there was still some atrophy. Occasional germinal centers appeared, and the lymphocytic content showed evidence of recovery.

Lymph nodes. The high sensitivity of lymphoid tissue to ionizing radiation resulted in tremendous atrophy seen as early as the third day. Lymphocytes almost disappeared, leaving a lacy framework that was histologically spectacular. A similar picture was found in the tonsils and other lymphoid tissue. Changes in the germinal centers might be necrobiosis, but a departure from normal was not marked except when the germinal centers disappeared, as they did in three-fourths of those who died in the first two weeks. The early gross appearance of human nodes was not known, but bombed animals showed some enlargement, softening, and a paler color. By the second week, large atypical mononuclear cells, considered by one observer as lymphoblasts, appeared. These cells logically could be pathologic forms whose sensitive nuclear chromatin was deformed by the radiation. About the fifth week, the nodes were usually small and almost devoid of lymphocytes and germinal centers. Bizarre large cells were more numerous. Plasma cells, eosinophils, and mast cells, along with increased numbers of reticulum cells, were present. Lymphocytes were more numerous in the fourth month but were still reduced.
Bone marrow. The cellular picture of irradiated bone marrow was tremendously changed in the first week after the bomb explosion. There was almost total disappearance of blood-forming elements, excepting small islands of erythropoiesis, which were less sensitive. By the end of the week reticulum began to proliferate and differentiated first into lymphocytes and plasma cells rather than myeloid cells. This type of differentiation was predominant until the fourth week when myeloid differentiation was seen. Most marrows of those dying before six weeks were hypoplastic, but a few showed hyperplasia with arrest of maturation. Most of the fatal cases of the third and fourth months showed hyperplasia, which in the femur was grossly evident as pink marrow extending through from one-third to one-half of the shaft. In these the maturation defect decreased and more neutrophils were found in the peripheral blood and in infected tissues. A few of the older cases, however, showed aplasia with pink gelatinous femur marrow. Some grossly appearing hyperplastic marrows were really hypoplastic, the pink color coming from dilated blood vessels. Whatever the marrow picture, there was usually a profound leukopenia at some time in those dying in the first six weeks. Later leukopenia did not persist, and even those who died had leukocytosis except for the few that had aplastic marrows.

Miscellaneous. Only secondary hemorrhagic or necrotic changes were found in the brain. No changes were found in the pancreas, except for some mitoses in the islet cells. The presence of any irradiation effects in the liver is a moot point.

Secondary effects of radiation of reticulo-endothelial system. Hemorrhagic lesions and leukopenic necrosis affected the irradiated body about the end of the first month. The pharynx and its connections, the gastrointestinal tract, the respiratory organs, and the skin manifested both changes. In addition, particularly the urinary tract, mesothelial linings, muscles, and all soft tissues, showed petechiae, purpuric patches, or large ecchymoses. These changes were outstanding clinically. The severity depended on the location of the larger hemorrhagic lesions. Hemorrhages in the linings of the pharyngeal regions, of the bowel, or of the urinary tract gave signs externally. Large submucosal hemorrhages as well as petechiae appeared in the kidney pelves and in the bladder and sometimes in the ureters. Hemorrhages breaking through the epithelium of bacteria-laden surfaces often initiated the neutropenic ulcers, which in the pharynx were similar to acute agranulocytosis. Ulcers sometimes extended to the tongue, gums, buccal mucosa, lips, and even the skin to give the picture of noma. Such ulcers also began without hemorrhage. Bacteria ordinarily nonpathogenic might cause serious consequences through the loss of sufficient reticulo-endothelial reserves. Ulcers throughout the gastrointestinal tract were on a similar basis, as indeed, many of the diffuse mucosal changes might be. The necrotizing pneumonia appeared to be a part of this picture. There was little leukocytic reaction in these lesions, which overwhelmed the patient and led to death.

Case history. A 29-year-old man was at a distance of 0.7 km. from the explosion center. He was outdoors a few paces from a concrete building and
was struck by a falling roof that inflicted slight head and neck injuries. There was nausea on 6 August 1945, and on the same day he vomited about 25 times. Malaise, accompanied by anorexia, began on 6 August and lasted until 10 August. He again experienced malaise from 21 August until he died on 1 September. Anorexia appeared four days after the second onset of malaise. There was epilation and gingivitis on 21 August, which persisted. The gingivae began to bleed on 30 August. On 25 August tonsilitis and purpura were noted. Both of these symptoms lasted until death. There was a high fever between 24 August and the time of death; and there was a productive cough beginning on 25 August with a hemoptysis on 30 August. The urine examined on 29 August was positive for albumin and negative for sugar.

Sections of marrow in this patient were hyperplastic, showing vascular adipose tissue crowded by a large number of young myelocytes. Mature polymorphonuclear leukocytes and even stab cells were rare. There was an occasional megakaryocyte. Occasional cells were found in mitosis. A few small cells with shrunken nuclei, thought to be normoblasts, also were found. Other important lesions at necropsy were: petechiae of the skin; epilation of the scalp; focal necrosis of the pharynx, tongue, tonsils, and larynx; necrotizing gingivitis; and abscess in the region of the right mandibular joint; necrotizing and hemorrhagic aplastic pneumonia; and minute hemorrhages of the gastrointestinal tract, trachea, and renal pelvis.

VII. Detection of Overexposure to Ionizing Radiation

At present the potential sources of exposure to radiation include: (1) diagnostic and therapeutic x-ray units, (2) industrial x-ray machines, (3) radium and its degradation products, (4) cyclotrons, (5) the chain reacting pile, (6) radioisotopes produced by the pile that are being used in tracer studies, therapy, and as sources of heavy radiation for biologic systems, and (7) the atomic bomb and its fission products. It is apparent that the medical profession and public health authorities must take cognizance of the sources of exposure and endeavor to establish means of prevention and recognition.

Prevention is accomplished by careful measurement of radiation intensities by personnel film badges and radiation detection instruments whenever radiation may be present. Personnel should be followed closely for the presence of radioactive isotopes in nasal secretions, excreta, and on the skin. Where radioactive gases may exist, expired air should be monitored. In brief, overexposure to radiation should never occur. Since signs and symptoms are late, conditions conducive to excessive exposure should be detected by physical measurements before cellular damage occurs. In spite of this, protective regimens may fail, and in the advent of atomic warfare many will be overexposed to ionizing radiation regardless of precautions. Many earlier scientists learned of the hazards of radiation by tragic personal experience. The incidence of radiation burns, ulcers, and superimposed cancer in the early physicists and radiologists, the incidence of aplastic anemia in x-ray technicians, and the greater incidence of leukemia in radiologists point to the possible hazard of long, continued minimal radiation and potentially harmful cumulative effects.
The effects of overexposure may be acute or chronic. The exposures may result from any type of radiation externally or internally applied. The clinical picture will depend on the amount, rate of delivery, and depth of the dose. Acute overexposure may be defined as a single total body exposure of more than 50 r. delivered within a period of a few hours. The signs and symptoms that may develop vary with the penetrating ability of the radiation and the amount absorbed. If the skin receives a large amount of soft x-ray or beta radiation, anything from a slight erythema to massive vesicle formation and destruction of its full-thickness may develop. The injury will resemble thermal burns.

Similar cutaneous injuries can be caused by more penetrating radiations; but, in addition, other signs and symptoms such as diarrhea, nausea, vomiting, headache, anuria, purpura, and secondary infections largely caused by the leukopenia may develop. The latent period before the development of symptoms will vary with and be inversely proportional to the amount of radiation absorbed. The symptoms and signs will be directly proportional to the amount of radiation received up to the point that the latent period becomes so short that there is insufficient time before death for the entire picture to develop. The signs and symptoms of acute overexposure to penetrating radiation are variable. Although the best biologic index of overexposure to radiation is the blood, with the less penetrating external radiation the blood changes are less marked and may be absent.

The blood changes following acute exposures are fairly uniform if the exposure is over 100 r. The changes with smaller amounts of radiation may be missed if careful and repeated observations are not made at frequent intervals. There is, however, a uniform response to amounts over 100 r. that is roughly proportional to the amount received, up to a maximum response in the absolutely fatal dose range. The response is a prompt decrease in the total lymphocyte count that is detectable within a period of a few hours. The decrease attains a maximum within about seventy-two hours. Recovery may or may not occur, depending on the amount received. Another quite constant phenomenon is an initial neutrophilic leukocytosis caused by mobilization of the neutrophils and perhaps accelerated maturation and release from the bone marrow. It is reported by some workers that the leukocytosis does not occur with massive amounts of total body radiation (over 500 r.) in some species. The changes in the numbers of platelets and red blood cells and morphologic changes of the leukocytes are less certain and vary so much with the dose and the survival time that they will not be considered here. The acute blood changes can be summarized as follows: If no drop in the total lymphocytes is detectable in the first seventy-two hours, it can be stated with certainty that the exposure to radiation has been small and that serious illness will almost assuredly not occur.

The chronic overexposure to ionizing radiations presents an entirely different problem. The changes that occur are insidious and progressive. In fluoroscopists, radiochemists, or radium handlers the following may develop on the hands: (1) an increased brittleness and tendency to develop longitudinal ridges of the fingernails, (2) loss of integrity of the fingerprint by...
patches of atrophy, (3) impaired sensation, and (4) pigmentation. In general, as with the acute exposure, the blood is the best biologic index of overexposure to radiations. In order to evaluate the blood picture, some sort of norm for the average person must be established. This is most difficult, for the human blood is variable. Leukocyte counts of 4,000 to 16,000 are occasionally found in people who are in every other detectable respect perfectly healthy. The differential counts vary considerably with age and may remain abnormal for many months following infectious mononucleosis. Erythrocyte counts and hematocrit and hemoglobin determinations similarly vary widely. The time-honored normal values for hematological measurements probably include 80 percent of a given population within the upper and lower limits of the ranges given in standard textbooks. The 20 percent of normal individuals outside of this range will cause considerable consternation in a radiologic safety program.

How are blood changes that may be caused by chronic overexposure determined? First, base line counts should be established on all who may conceivably be exposed. The counts should be made at monthly intervals. Notations on the occurrence of colds, infections, and other symptoms should parallel the blood records. Relative changes in the blood of a given individual may then more readily be detected. The following hematological criteria for presumptive evidence of overexposure to radiation are offered and have been based on standard normal values and possible changes that have been described in the literature: (1) a depression of the leukocyte count below 4,000; (2) an elevation above 15,000 with an absolute and relative lymphocytosis; (3) a relative lymphocytosis with a low total count that returns to normal following removal from exposure; (4) an increased mean corpuscular volume, a shift in Price-Jones curve to the right, and an increase in the mean corpuscular diameter; (5) a reticulocytosis over 2 percent; and (6) an erythrocyte count over 5.1 million/cu. mm. and hemoglobin over 18.0 gm. percent. If any of the above criteria develop in a person who has a definitely established base line and who is associated with radiation, it is presumptive evidence of overexposure to radiation until proved otherwise.

Many other phenomena have been suggested as hematological evidence of overexposure. Changes in blood coagulation, prothrombin times, platelets, and morphologic changes in leukocytes, such as toxic granules, basophilic staining, and vacuoles (the toxic triad), have all been offered. It is exceedingly difficult to evaluate the importance and the diagnostic value of those changes. The evaluation of chronic exposure of any given individual in terms of changes within the blood cannot be made with absolute certainty. The following procedure may yield helpful information: (1) Remove the suspect from all possible sources of radiation. (2) Study breath, excreta, and nasal swabs for the presence of radioactive isotopes by making differential radiation counts. (3) Study the blood at weekly intervals and compare with the base line counts. (4) Endeavor to eliminate other factors, such as infectious mononucleosis, infectious lymphocytosis, virus diseases, benzol poisoning, and heavy metal poisoning. (5) Examine others that may have been similarly exposed and com-
pare the base line mean leukocyte counts with the present mean counts for the group.

The fifth maneuver may yield more information than all the other blood changes combined. If a statistically significant difference in the means of the leukocyte counts of a group of people can be demonstrated during known chronic exposure as compared to the base line means, particularly if the difference shows a downward trend, it can be stated with some assurance that there has been chronic overexposure to radiation. The development of the above presumptive signs in the mean leukocyte counts for a group must be considered as evidence of overexposure until proved otherwise. The main bulwark of protection from radiation must remain physical control and measurement by established monitoring procedures.

VIII. Public Health Aspects of Atomic Explosion

It is hard to think of a group as other than made up of individuals. It is equally difficult to regard the individual without giving some consideration to the fact that he is a member of society. Public health is that branch of medicine that deals with the relationship of the individual to the community and of the community to the individual. At present the emphasis is shifting from the absence of disease to the presence of health.

In the event of an atomic explosion the medical officer will be called upon to assess the hazard and to advise the command accordingly. He will probably have the necessary physical findings supplied to him. The magnitude of the hazard will depend on many factors. The advice of not only the physicist but also the meteorologist, geologist, and oceanographer will be needed. In damp or rainy weather there is little dust, therefore ground contamination will not be as serious from an internal (inhalation) standpoint as it would be under dry conditions. In assessing the hazard, it must be kept in mind that external radiation is more easily dealt with than internal radiation. You can guard against external radiation, but you must prevent internal radiation. Decontamination of the skin, although at times difficult, is far easier than decontamination of the thyroid, lungs, or bone.

The common personnel monitoring devices are film meters, pocket ionization chambers, pocket electroscopes, and Geiger-Müller tubes. Area monitoring instruments include Geiger-Müller tubes, electroscopes, ionization chambers, film meters, and dust- or air-sampling devices. Let us assume that an area is contaminated. It may be contaminated with: (1) Alpha emitters. This will constitute a most serious hazard if such substances gain access to the interior of the body. There will be no external radiation hazard. (2) Beta emitters. This will constitute both external and internal radiation hazard, which is more serious per unit if internal. (3) Gamma emitters. Here again we must think of both external and internal hazard, which is more serious from a practical standpoint if external. (4) Contamination. Contamination will almost certainly not be limited to one of the above types of radiation.

Food. It must be assumed that all food found in the area is dangerous. The food may contain induced radioactivity. This is unlikely to be present in
dangerous quantities, because of generally unfavorable conditions and because of the short half-life of many substances. The medical officer will, however, probably be called on to give an opinion in these cases. Radioactive substances will most likely have been deposited on the food. In this case decontamination will be impractical or impossible. Canned or otherwise protected foods may be eaten only after careful inspection and most rigorous attention to detail in removing the food from the protecting agent. If it is necessary to bring food into the contaminated area, a high degree of laboratory precision, comparable to aseptic surgical technique, must be maintained in the handling of it. Smoking should not be allowed, as the handling of tobacco adds one more hazard.

Water. If possible, no water should be drunk in the area. If canteens are to be taken in, troops must be drilled in the matter of drinking without contaminating the mouth of the canteen by wiping. If larger amounts of water must be taken in, this greatly increases the hazard. The water in an area may be contaminated as a part of the general area contamination or may have become contaminated upstream. What can be done about decontamination? (1) Boiling is useless and may be harmful. It is unlikely that all contaminants will be volatile. Boiling will then serve only to concentrate and increase the contamination. (2) Storage, although useful for short-lived isotopes, is impractical for field operations and of little benefit for long-lived isotopes. (3) Filtration offers some promise and it is especially hoped that experimental work will point the way to practicable means of field application. (4) Chlorination and other chemical procedures are useless. (5) If we can combine precipitation and filtration, we may greatly reduce the load on precipitation. Here again, methods must be developed that are applicable to field use.

Prevention of dissemination by personnel is often of great importance. The underlying principles are always the same and may be illustrated by a discussion of the evacuation of an area. A decontamination center for area evacuation should be set up near the border of the contaminated area and all traffic in and out of the area controlled. Facilities must be provided for personnel entering the contaminated zone to remove all clothing, especially outer clothing, and change to overalls, hat, gloves, and boots. All food and tobacco should be left behind. Efficient monitoring is essential. On leaving the contaminated zone, personnel remove hat and gloves, wash face, neck, and hands thoroughly five times with soap and water, remove remaining clothing, and then soap and thoroughly wash entire body five times. The monitor located in a room between the shower and the uncontaminated side gives permission to go to the "clean" side and put on "clean" clothing. Laundering facilities for contaminated clothing must be provided. Shoes will present a difficult problem for evacuees leaving the area.

One may work with any amount of radioactive material if proper precautions are taken; but one cannot work with even the smallest amounts without proper precautions. Troops should learn to appreciate without hysteria the dangers of exposure.

(To be concluded)