Z A G A D N I E N I A B I O F I Z Y K I W S P Ó Ł C Z E S N E J

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THE EVOLUTION OF RADIATION PROTECTION STANDARDS *

Rontgen was 50 years old in 1895 when he discovered, on November 8th, the X-rays, as he called them, although in many countries of the world they were, and are still, known as Rontgen rays. The news of the discovery of X-rays reached London on January 6th, 1896, and between January 7th-13th the first X-ray photographs in this country were taken. In April there appeared the forerunner of the British Journal of Radiology called the "Archives of Clinical Skiagraphy". These were edited by a man who later became a well-known radiologist, Sidney Rowland. The first volume contained some good X-ray photographs taken by Rowland himself and, of particular interest, a series of cinema-radiographs, taken by Dr. John McIntyre of Glasgow, showing the movement of frogs legs. In June 1897 there was the first meeting of the newly formed Rontgen Society, the forerunner of the British Institute of Radiology, and perhaps more significant, and indicative of the considerable contribution which physicists, notably in Britain, have made to the development of the subject, the president was Silvanus Thompson.

In that year Thompson delivered his presidential address to the Rontgen Society and in it he reviewed the discoveries of Rontgen and other workers. It is a record of achievement in the short period of two years that can hardly be equalled elsewhere. It is surprising to realise that, by 1897, double coated X-ray film had been used in an attempt to increase photographic density for a given exposure, that intensifying screens had been tried out but temporarily abandoned because of trouble with grain effect, that McKenzie Davidson had devised a method of localisation of foreign bodies, that dermatitis and epilation had been observed and that aluminium filters were already being used to remove the

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longer wave-length components of the X-ray spectrum and so to reduce the liability to X-ray burns.

As early as 1898 the newly formed Rontgen Society started to collect information on the injurious effects of X-rays although it was not until 1915 that the Society's recommendations for the protection of X-ray workers were published. These recommendations simply referred to enclosing the X-ray tube in a protective shield, to covering fluorescent screens with lead glass and to providing lead gloves and aprons. There was no concept yet of a level of exposure to which workers could be subjected without harm, the "tolerance dose" as it later became known. The last sentence of the recommendations may interest you "the hand or any portion of the body of the operator should never be used to test the hardness of quality of the X-ray tube".

In those days there were no kilovolt meters let alone dosemeters and the kV of the tube was assessed by the contrast, seen on a fluorescent screen, between bone and soft tissue while output was judged from the brightness of the screen. About the time the recommendation quoted was made the device shown in Fig. 1 was

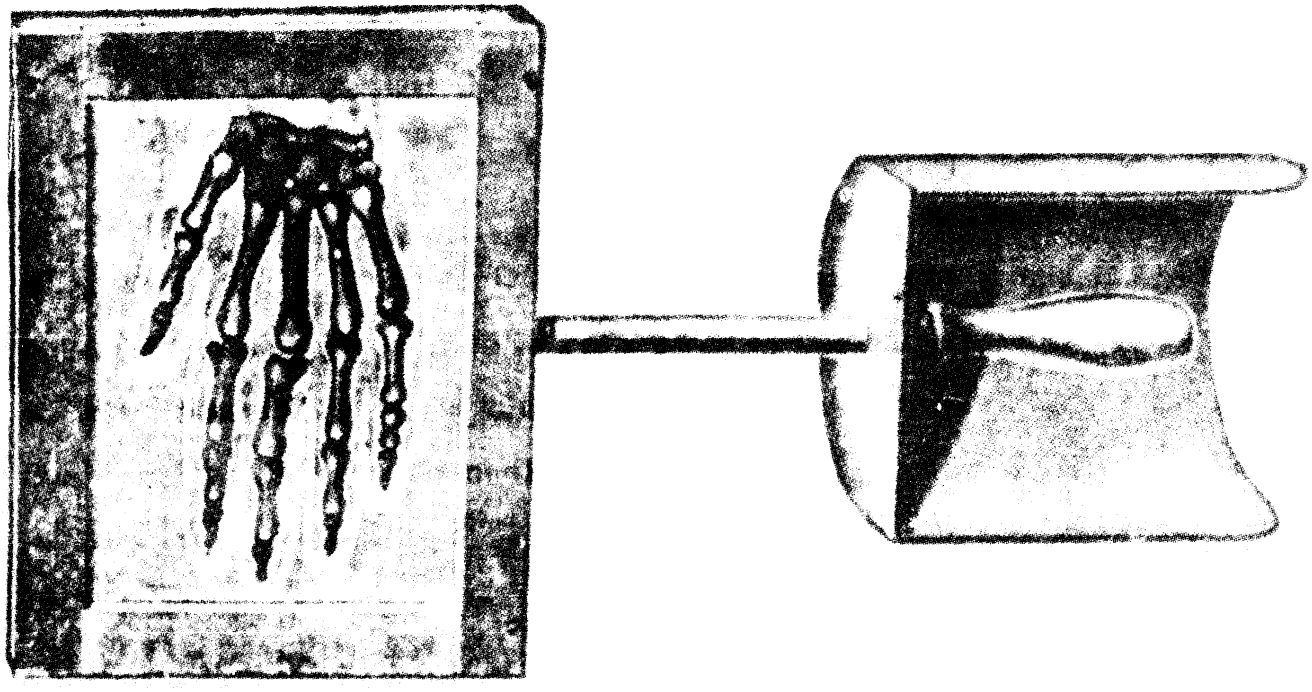


Fig. 1. Effect of X-rays on soft tissue and bones (Courtesy Mould, R. F. "History of X-rays and Radium")

introduced. It consisted of the bones of a hand mounted in aluminium, the chosen thickness of which gave an attenuation similar to soft tissue. The device had, as will be seen, a handle shielded from radiation.

Table 1

Historical landmarks: Radiation injuries

1895 (DEC 28)	ANNOUNCEMENT OF DISCOVERY OF X-RAYS	1901	GUINEA-PIG KILLED BY X-RAYS WITHOUT "BURN"
1896 (JAN)	X-RAY DERMATITIS OF THE HANDS	1902	CANCER DEVELOPED IN CHRONIC X-RAY ULCER
(MARCH)	SMARTING OF THE EYES	1903	BONE GROWTH INHIBITED IN ANIMALS
(APRIL)	EPILATION WITHOUT DERMATITIS		STERILIZATION OF MALE RABBITS & GUINEA-PIGS
1897 (FEB)	23 CASES OF X-RAY DERMATITIS REPORTED	1904	CHANGES PRODUCED IN BLOOD OF RABBITS
	CONSTITUTIONAL SYMPTOMS REPORTED		LEUKOPENIA CAUSED BY X-RAYS IN MAMMALS
1898	PARAPLEGIA & SPASMODIC MUSCULAR CONTRACTION	1905	LYMPHOCYTE SHOWN AS MOST SENSITIVE LEUKOCYTE
	ROENTGEN SOCIETY (BRITISH) STARTED TO COLLECT INFORMATION ON INJURIOUS EFFECTS OF ROENTGEN RAYS	1906	BONE MARROW CHANGES DEMONSTRATED
		1910	TUMORS PRODUCED IN SKIN OF RAT
1899	DEGENERATION OF VASCULAR ENDOTHELIUM REPORTED	1911	94 CASES OF X-RAY INDUCED HUMAN TUMORS
		1912	ANEMIA FOUND IN TWO X-RAY WORKERS

It is perhaps not surprising that no pronouncement was made on acceptable levels of exposure since there existed, as yet, no agreed system of X-ray dosimetry. Between 1919 and 1921 the number of deaths of radiation workers from carcinoma and aplastic anaemia began to mount (Table II) and gave rise to widespread uncertainty and anxiety as to the efficacy of the various techniques and materials used for the purpose of protection against ionising radiation. This led to the formation of the British X-ray and Radium Protection Committee, the world's first, which issued its preliminary report in 1921. Again it dealt simply with measures to protect the worker from exposure to radiation and made no comments on a level of exposure which might be acceptable. It did, however, consider establishing a tolerance dose in terms of a specifiable and reproducible biological standard with the hope that ultimately this biological standard could be expressed

in physical units. There was, at this time no agreed physical unit of X-ray intensity nor was there any accepted method of measurement. Then, and as was proved many years later, the most promising method of measurement seemed to be that of ionisation produced in air, and if, as was thought, ionisation was the link between the radiation energy falling on a medium and the effect it produced then the ionisation produced per unit mass, or better still, the energy absorbed from the radiation per unit mass, seemed to provide an appropriate and convenient unit of dose.

When this problem was first approached it was the general opinion that what was required was a unit of energy absorbed in tissue. Since, however, tissues have different compositions an arbitrary choice would have to have been made for standardisation purposes and, with the state of knowledge sixty or seventy years ago, it seems most unlikely that agreement would have been reached in this choice. In any case measurement of the energy absorbed in tissue presents grave difficulties and, although over a period of more than half a century we have come round again to wishing to measure in this way, many of the difficulties still remain. A much simpler way, and the one still widely used, was to measure the ionisation in a gas. Since air and soft tissue are composed of atoms which, because of their comparable atomic numbers, behave similarly in regard to the absorption of radiation of various energies, the energy absorbed in air was chosen as the quantity to be measured which, it was felt, could be closely related to the biological effect.

It was not until 1928, at the first International Congress of Radiology in Stockholm, that a definition of the unit of X-ray quantity, to be called the "Rontgen", was adopted. It read as follows "the Rontgen is that quantity of X radiation which, when the secondary electrons are fully utilised, and the wall effect of the chamber is avoided produces, in one CC of air at $0^{o}C$ and 760 millimeters of mercury pressure, such a degree of conductivity that one ESU of charge is measured at saturation current". The unit with various adaptions, but still retaining the basic concept, is in use to-day.

However, looking back before that event - experience in the early days of X-ray therapy indicated that there was a threshold for the production of reddening of the skin, or erythema, and that below a certain dose, no effect was seen. This interpretation

of X-ray effects, implying that there was a threshold below which no effects were produced, became deeply embedded in radiological thought and lead to the concept that a tolerance dose existed, exposure at levels below which could be considered safe. Despite difficulties, produced by the absence of a system of dosimetry, several attempts were made to define such a tolerance dose, a very early one being due to Rollins. He described a tolerance dose as that which would fog photographic plates in 7 minutes, irradiation at a level less than this being not of harmful intensity. This corresponds to something like 10 Rontgens per day which is twice the annual exposure we now consider permissible! In 1920 two workers Seitz and Wintz introduced the term "Unit Skin Dose", this being the dose which would induce a first degree erythema a relationship which led others often to refer to it as the "erythema dose".

In 1925 the first suggestion of recognition of protracted exposure related to delayed effects was introduced when Mutscheller put forward the possibility of estimating a dose which an operator could tolerate for a long period of time without ultimately suffering injury. On the basis of observations on workers in several establishments, where he thought the installations were particularly well designed from the protection point of view, he proposed that it was entirely safe if an operator did not receive, in one month, a dose exceeding one hundredth of an erythema dose or a thousandth of it in three days. Also in 1925, Sievert adopted one tenth of an erythema dose per year as the safe dose. This was close enough to Mutscheller's figure of one hundredth of an erythema dose per month. A number of other estimates of this sort were made and are given in Table 2.

Since all these workers made their assessments independently and, bear in mind, none could claim to have observed any effect at levels above $\frac{1}{100}$ of an erythema dose per month it is surprising that their estimates come so close to one another. All the evidence available was that people in good installations showed no effects so having made some measurement those investigating the matter divided it by 10 for safety - thus we have, as Lauriston Taylor once observed, "a large array of philosophy built on observing nothing"!

It was, of course, realised that, in the form which Mutscheller expressed the tolerance dose, the definition could not be readily used and it was decided therefore to try to express the

Table 2

Estimation of tolerance dose

Date	Proposer	Days for $\frac{1}{1000}$ erythema dose	Equivalent (calculated) γ per day
1902	Rollins	Photographic	10
1925	Mutscheller	3	0.2
1925	Sievert	3	0.2
1926	Solomon	0.3	2.0
1927	Netherlands Protection Board	15.0	0.04
1928	Barclay and Cox	3.5	0.17
1928	Kaye	5	0.12
1931	U.S. Advisory Committee		0.2
1932	Failla (Ra γ rays)	30.0	0.1
1932	Stenstrom	3.7	0.16
1934	British X-ray and Radiation Protection Committee (also later that year I.C.R.P.)		0.2

erythema dose in terms of some physical unit. There was in use at that time, in some parts of the world, a unit designated R, and with proved later not to be terribly different from the Rontgen (r) once it was defined. In 1927 Kustner in Germany, as a result of an enquiry made by circular letter to a number of radiotherapists in which he asked them to give the R value, which they found for the erythema dose, stated that the average value was 550 R. When the International Commission on Radiological Units (I.C.R.U.) defined the Rontgen, in 1928, the erythema dose became equal to six hundred rontgens. Thus the Mutscheller tolerance dose became six r in one month or about 0.2r in one day. This figure was not adopted officially in Britain until the Fourth Revised Report of the British X-ray and Radium Protection Committee in June 1934, and it was subsequently accepted by the International Commission on Radiological Protection (I.C.R.P.) in July of that year.

So forty years elapsed before we were in a position to quantify properly a relationship between exposure to radiation and its effects. The value of 600r for the erythema dose was reaffirmed following the redefinition of the Rontgen in 1937 and it was the value in use up to, and including, the period of the last war.

Up to this time the so called tolerance dose had been thought of only in terms of radiation falling on the skin but several

workers, notably Nayneord, emphasized that both the dose to critical tissues at depth in the body and the total quantity of energy absorbed in the body, what Mayneord called the integral dose, were of considerable importance both physically and clinically. Clearly for a given dose on the surface the amount of radiation reaching depths in the body varies with the penetrating power of the radiation and the integral dose, a measure of the total energy absorbed by the body, per unit surface dose will increase with the energy of the radiation (as well, of course, as with the area irradiated). For example, for whole body irradiation the integral doses are as follows:

40 KV -	13,000	gram rads per rad
200 KV -	46,000	" " " "
Radium Gamma rays -	59,000	" " " "

So a reduction in tolerance dose values was contemplated as higher X-ray energies became available. The outbreak of the second world war caused suspension of the activities of I.C.R.P. and of its sister the (I.C.R.U.) and further progress in estimating the "tolerance dose" was made by individual national bodies and notably by the British X-ray Radium and Protection Committee and its successor the Medical Research Council Tolerance Doses Panel.

By the end of the war, besides X and gamma rays, other radiations such as neutrons and heavy charged particles had to be considered. By then, too, we were aware that one of the more sensitive indicators of radiation exposure of individuals is changes in blood count levels and, based on observation of these changes and other studies related to carcinogenic effects, particularly the induction of leukeamia, it was concluded that an exposure, at the depth of the blood forming organs (approximately 5 centimeters) of 0.05r per day would be tolerable. This is about 0.3r per week skin dose which was the value finally adopted by I.C.R.P. in 1950.

The possibility of the production of genetic effects by radiation had now come to be realised and, on the data then available, it was concluded that exposure of the whole population at the level of 0.3r/week would produce a seven fold increase in mutations. However, if only a small proportion of the population were to be exposed as was then the case, there would only be a very slight increase in hereditary abnormalities in the population as a whole.

Consideration of genetic effects led to the fundamental change from the idea of a "tolerance dose" to the concept of a "maximum permissible dose". This came about because effects, such as genetic ones, were considered to be irreversible and further that such effects could be induced by the smallest doses so that there could be no threshold, and therefore no tolerence dose below which no effect is produced.

These changes heralded the introduction of a number of new features.

1) the permissible exposure values were based on quantitative information concerning biological effects, notably changes in the blood picture.

2) the changes took account of the fact that more penetrating radiations were now available and therefore that, for a given surface exposure, more radiation was reaching sensitive tissues,

3) some effects were irreversible and so there was no question of tolerating a dose of radiation but rather of determining a level at which the consequences were at an acceptably low value.

4) for the first time genetic effects were taken into account and it was considered that, if only a small proportion of the population were exposed at levels of 0.3r per week, the possible increase in mutations introduced would only very slightly increase the natural incidence.

The variety of radiations now available called for rethinking on units of measurement. Although the necessary information for its practical use was not available until 1956 the unit the rad (Table 3) was proposed by the I.C.R.U. in 1953 together with the so called "RBE dose", which became the rem (Table 4), and these were introduced into the 1953 recommendations of I.C.R.P.

In 1956, while the maximum permissible dose remained at 0.3r/week it was required that it should not average more than 0.1r/week, mainly to bring the cumulative exposure up to the age of 30 years, in which period most reproduction occurs, down to a lower level. Two further changes were made at this time which took the emphasis away from levels of exposure in the short term to consideration of cumulative dose. The maximum permissible exposure was expressed as the level per year, 5 rems being the value, rather than per week as hitherto, and a limit was placed on the cumulative dose over a working life by saying that the dose should not exceed the value D given by $D = 5 (N - 18)$ rems,

T a b l e 3

Absorbed dose and the rad

Absorbed dose – a concept of more general application than exposure – is a measure of the total energy deposited by any kind of ionising radiation per unit of mass of material at some location of interest. Its unit, the rad, is equal to 10^{-2} joules per kilogram. In general, the energy deposited by radiation depends on the nature of the absorbing material, and the units of dose are normally qualified as rads-in-air, rads-in-tissue, etc. In soft tissue, and for a wide range of X- and gamma-ray energies, a good rule of thumb is that an exposure of one roentgen will result in an absorbed dose of about one rad.

N being the age in years. This change, which took account of the requirement for limitation for dose received up to the age of 30 mentioned above, was introduced because of information coming forward about the incidence of leukeamia in survivors of the Japanese atomic weapon explosions which indicated that the dose of 750 rems which would be received by a radiation worker exposed at 0.3 rems per week for a working life of 50 years was too high and that the life-time dose should be reduced to about 200/250 rems. Although the overall limit of 5 rems per year is still with us substantial detailed changes in the I.C.R.P. recommendations have been made over the past 25 years.

Over that time we have moved from the idea of limit based on a threshold effect, as envisaged in the original tolerance dose, to growing awareness of probablistic, non threshold effects such as carcinogenesis. Until recently, limits were set which recognised the non-threshold nature of the effects but the limits were applied only to the whole body and to one or two special organs, like the blood forming organs. Moreover the limit for the whole body was the same as for some of its parts which seems illogical.

Table 4

Dose equivalent and the rem

The effect of ionising radiations on living tissue is not simply proportional to absorbed dose. It may also depend, for example, on the spatial distribution of energy released along the track of each ionising particle, which in turn depends on the type and energy of the radiation concerned. To cater for such variations, each type of radiation is assigned appropriate weighting factors to convert the absorbed dose in rads into its biological dose equivalent, whose unit is the rem.

The one most frequently used is the so-called quality factor, which takes account of the differences in linear energy transfer mentioned above. For X-rays, gamma rays and beta rays, the quality factor is unity and hence dose equivalent in rems is the same as absorbed dose in rads. Other radiations have much greater biological effectiveness. For fast neutrons, the quality factor can be as high as 11 and for alpha particles as high as 20.

In its most recent report on such limits, I.C.R.P. 26, the Commission recommends a procedure which takes account of the total risk attributable to the exposure of all tissues irradiated.

In previous recommendations the Commission stated that there should be some limitation on the rate at which the dose equivalent may be accumulated, the age-related formula being an example of an approach to this, but it now believes that it is sufficient to set annual dose equivalent limits and it does not recommend any further restrictions, either on the instantaneous rate, or on the rate on which a dose equivalent may be accumulated. The exceptions to this are the occupational exposure of women of reproductive capacity and of pregnant women.

In the report, the Commission also points out that their assumption about the proportionality between dose and response means that, for non-threshold effects, it would be justifiable to consider the mean dose over all cells of uniform sensitivity in

a particular tissue or organ. This use of the mean dose has practical advantages in that the significant volume can usually be taken to be that of the organ or tissue under consideration. However, when the radiation of the tissue is non-homogeneous the use of the mean dose over the tissues ceases to be strictly valid if doses to individual cells differ more widely than the range of doses over which the dose response relationship for the tissue can be regarded as linear. An important example of this may be the irradiation of the lung by radioactive particles. However, the Commission believes that, for late somatic effects, the absorption of a given quantity of radiation energy is ordinarily likely to be less effective when due to a series of hot spots than when uniformly distributed because of the effect of high doses in causing the loss of reproductive capacity or the death of cells. Available epidemilogical evidence supports this conclusion. Thus, for particulate radioactive sources within the tissue, to assess the risk by assuming homogeneous dose distribution would probably over-estimate the actual risk.

In considering the limitation of exposure for protection purposes the Commission have examined both stochastic and non-stochastic effects. Stochastic effects are those for which the probability of an effect occurring, rather than its severity, is regarded as a function of dose without a threshold. Non-stochastic effects are those for which the severity of the effect varies with the dose and for which a threshold may, therefore, exist. So in a sense we now have both the concepts of a maximum permissible exposure and of a tolerance dose.

At the low doses involved in radiation protection the main stochastic effect is carcinogenesis. Examples of non-stochastic effects are the induction of cataract of the lens of the eye and non-malignant damage to the skin.

The aim of radiation protection then should be to prevent detrimental non-stochastic effects and to limit the probability of stochastic effects to levels considered acceptable.

However, most decisions about human activities are based on some, often explicit, form of balancing of costs and benefits and, in recent years, in radiation protection it has become possible to formalise such decision-making procedures, although not always to quantify them. Sometimes too, we have to establish dose equivalent limits in situations where the benefits and detriments

are not both received by the same members of the population. For such reasons the Commission has recommended a system of dose limitation which should meet the following criteria:

1. No practice shall be adopted unless its introduction produces a positive net benefit.

2. All exposure should be kept as low as reasonably achievable (ALARA principle), economic and social factors being taken into account.

3. The dose equivalent to individuals shall not exceed the limits recommended for the appropriate circumstance by the Commission.

The first two of these are the most important, the dose equivalent limit requirement being a "back-stop" marking the dividing line between acceptable and unacceptable risks.

The absorbed dose D is not by itself a sufficient quantity by which to predict either the severity or the probability of detrimental effects and the quantity Dose Equivalent is used where the dose equivalent H at any point in tissue is given by $H = DQN$.

"N" is introduced to take account of any modifying factors which may be discovered such as dose rate, fractionation, distribution, etc., and is the product of these. At present the value assigned to N is one. Q is the quality factor.

Radiobiological considerations

The exact relationship between a dose of radiation received by an individual and a particular biological effect which is induced is complex and, for radiation protection purposes, we must of necessity make simplifying assumptions. There is radiobiological evidence for assuming that for low LET radiation the dose response curve generally increases in slope with increasing dose and dose rate for absorbed doses ranging up to a few gray (1 Gy = 100 rads). Such a response can be represented by an expression such as:

$$E = aD + bD^2,$$

where E is the effect, D is the dose and a and b are constants. A variety of effects obey a relationship of the form but the relative values of the parameters a and b vary from one observation to another. The quadratic term predominates at high absorbed doses, generally above 1 gray, and at high absorbed dose rates

of the order of 1 gray per minute, but the linear term comes to predominate as the dose and dose rate are reduced and it is for this reason that the Commission make the basic assumption that, for stochastic effects, within the range of exposure conditions usually encountered in radiation work, a linear relationship without threshold between dose and the probability of an effect exists.

The main late somatic effect of radiation on man is cancer. No clinical distinction can be made between cancers induced by radiation and those occurring naturally in the population and considerable difficulties are involved in attempting to establish estimates of risk of producing cancer in different tissues. However, a wide range of experiments with animals as well as observations on man provide convincing evidence that ionising radiation can cause cancer. Many types have been produced and the evidence is so extensive that it is reasonable to assume that all tissues, or very nearly all, are susceptible.

Until recently, however, there has been little direct evidence to show that moderate levels of radiation can increase the incidence of tumours in any of the tissues of man in which cancer is relatively common under normal conditions, for example, the stomach, colon, rectum, breast, uterus and bladder. The most detailed evidence relates to the induction of leukaemia.

In earlier recommendations the Commission has said that, in circumstances in which more than one organ of the body is exposed, a particular organ or tissue irradiated is likely to be of greatest importance to the health of the individual either because of the dose it receives or because of its sensitivity to radiation or because of the importance to health of any damage that results. This tissue or organ was referred to as the critical one and dose limitation for the individual was determined by the dose equivalent limit for that tissue or organ. The concept did not permit the summation of detriment according to the relative radiosensitivity of all the irradiated tissues and the Commission now recommends a procedure which takes account of the total risk attributable to the exposure of all tissues irradiated.

To do this it is necessary to specify the organs and tissues that have to be considered because of their susceptibility to radiation damage, the seriousness of such damage and the extent to which the damage could be treatable. Some of the values for

such risk factors, which I shall be discussing shortly, are age and/or sex dependant, e.g. breast cancer and hereditary effects. Then too, the risk factors for the occurrence of malignancies must be smaller in older persons because of the long latent periods involved. The mean latent period for leukaemia, is about 10 years but for most other forms of cancer it is 20 years. In fact the variations from the average value for all ages and both sexes are not large, and so, for protection purposes, sufficient accuracy is obtained by using a single dose equivalent limit for each organ or tissue for all workers regardless of age or sex.

Recommended dose equivalent limits

It is believed that non stochastic effects will be prevented by applying a dose equivalent limit of 0.5 Sv (50 rem) in a year to all tissues except the lens of the eye for which it has recommended that the limit be 0.15 (15 rem). This latter limitation is to prevent the possible occurrence of changes, which, while not themselves detrimental to vision, could develop, without further exposure, to cause deterioration of vision. The limits apply irrespective of whether the tissues are exposed singly or together with other organs.

In considering stochastic effects, in many practical situations the exposure of the body may not be uniform so the Commission's recommended dose limitation is based on the principle that the risk should be equal whether the whole body is irradiated uniformly or whether there is non-uniform irradiation. The

T a b l e 5

Relevant risk factors, representing the stochastic risk resulting

Tissue	Risk factor - Sv^{-1} (rem^{-1})
Gonads	0.4×10^{-2} ($\times 10^{-4}$)
Breast	0.25×10^{-2} ($\times 10^{-4}$)
Red bone marrow	0.2×10^{-2} ($\times 10^{-4}$)
Lung	0.2×10^{-2} ($\times 10^{-4}$)
Thyroid	0.05×10^{-2} ($\times 10^{-4}$)
Bone	0.05×10^{-2} ($\times 10^{-4}$)
All other	0.5×10^{-2} ($\times 10^{-4}$)
	1.54×10^{-2} ($\times 10^{-4}$)

Commission introduces, therefore, the quantity effective dose equivalent, H_E, as the sum of the dose equivalents in individual organs so that if $\sum_T W_T H_T = H_E$ the conditions will be met. W_T is a weighting factor representing the proportion of the stochastic risk resulting from tissue T to the total risk when the whole body is irradiated uniformly. H_T is the annual dose equivalent in a tissue T. H is the recommended annual dose equivalent for uniform irradiation of the whole body, namely 50 mSv (5 rem). The relevant risk factors are shown in Table 5 and are obtained as follows.

Breast

Information on radiation induced malignancy of the breast has only come forward in the past few years and it is of particular importance because of the proposals involving, for example, mammography, for regular examination of women to detect and identify lumps in the breast. The data available suggests that, during reproductive life, the female breast may be one of the more radio-sensitive tissues of the human body. There is information from three sources the first being in women exposed during the Japanese explosions. The second comes from studies of women who were suffering from tuberculosis and who were given repeated radiographic examinations to evaluate the completeness of artificial pneumothorax therapy. The third source is a series of women given X-ray therapy for acute post partum mastitis. There is now evidence that pre-menopausal women are more susceptible to radiation induced breast cancer than post-menopausal women while the female in general is more sensitive than the male. For young women the risk is 10×10^{-3} Sv^{-1} but is less above the age of 40, so the average risk is 5×10^{-3} Sv^{-1} and for both sexes 2.5×10^{-3} Sv^{-1}.

Red bone marrow

The red bone marrow is taken to be the tissue mainly involved in the causation of radiation induced leukaemia. An important series of observations on humans given radiotherapy for ankylosing spondylitis and others on the Japanese survivors of nuclear explosions indicate that the incidence of irradiation induced leukaemia reaches its peak within a few years, probably around 7, and returns to pre-irradiation levels after about 25 years. For radiation protection purposes the risk factor for leukaemia is 2×10^{-3} Sv^{-1}.

Lung

Although there is some evidence that external radiation can induce lung cancer in man most of our information on cancer of the lung has come from observations on miners working in uranium mines where they are exposed to high concentrations of radon and its decay products. It is perhaps important at this stage to point out that no cases of cancer of the lung, clearly attributable to radiation exposure, have been reported in people who have worked with radioactive materials, especially plutonium, in particulate form even though some of them were exposed to above current limits. (in one example 25 workers, 30 years ago, were exposed to about 25 M.P.B.B). This is the confirmatory evidence I referred to earlier which suggests that the hazard of particulate material in the lung is likely to be less than that of the same material distributed uniformly throughout the lungs.

The risk of lung cancer in man appears to be about the same as that for the development of leukaemia and so for radiation protection purposes the risk factor is 2×10^{-3} Sv^{-1}.

Thyroid

The thyroid is one of the organs on which fairly extensive information on radiation induced changes is available. Most of this comes from studies of children irradiated, mainly in the United States, for thymic enlargement which resulted in doses of the order of 100 rads to the thyroid. There is also information on people exposed at the atomic bomb explosions, while additionally many thousands of people have been treated for thyroid conditions using radioactive iodine often involving exposure of several thousand rads. Further information on the effects of internal exposure of the thyroid comes from the study of the incidence of thyroid cancer in the Marshall Islanders. For internally incorporated iodine 131, and other longer lived isotopes, the risk is much lower and it is conservatively estimated that it requires at least 10 times the dose of radiation from iodine 131 compared with X-rays to produce the same incidence of thyroid cancers.

The thyroid is one of the sites for which, when malignancy occurs, the prognosis is good and most of the radiation induced thyroid cancers have been found to be of the well differentiated type which has a good prognosis. The risk of developing either malignant or bening nodules in the thyroid in children is between 100 and 200×10^{-4} Sv^{-1} respectively after exposure to

external radiation. The adult is less prone than the child to developing either type of nodule by a factor which may be as much as 3. For the standard population with a median age of 35 the risk is taken as 100×10^{-4} Sv^{-1}. A mortality rate of 5% is assumed for malignant nodules giving as a risk 5×10^{-4} Sv^{-1}.

Bone

There is little information on the incidence of bone sarcoma following external radiation. The ankylosing spondylitic patients have been studied and there is no evidence of an increased incidence of cancer. However the interpretation of the data is uncertain for the follow up period has been short and the people concerned were already suffering from a clinical disorder at the time of irradiation.

The most extensive human data on radiation induced bone sarcoma is derived from long term studies on over 1700 people, mainly women, who were exposed to radium when employed, between about 1910 and 1930, as luminous dial painters. There are many difficulties in estimating the dose to skeleton from the deposition of radium. In most cases the initial bone content has had to be assessed from measurements of body content made up to 30 years after exposure while, additionally, the radium 226 to which some people were exposed contained an unknown amount of mesothorium (Ra228) with a half life of only 6-7 years.

The radiosensitive cells in bone have been identified as the endosteal and the epithelial cells. The risk coefficient obtained from the above studies is 100×10^{-4} Sv^{-1} for alpha radiation. The most recent value of the Q.F. for α radiation is 20 and so the risk coefficient for low LET radiation becomes 5×10^{-4} Sv^{-1}.

Gonads

Deleterious effects caused by irradiation of the gonads may be of three different types, tumour induction, impairment of fertility in the irradiated individual and hereditary effects in descendants.

No carcinogenic effects in these organs due to irradiation have been found and human gonads therefore appear to be of very low sensitivity in this context.

Radiation impairment of fertility differs between the male and the female. Relatively high doses are required to impair fertility either temporarily and, certainly, permanently and the difference

between the male and female comes in that, in the male, there is a constant replenishment of spermatozoa throughout adult life whereas in the female the production of new oocytes normally ceases in childhood and the number of such cells decreases progressively with age after the menarche and so impairment by radiation varies with age.

Radiation induced hereditary disease differs in no known way from hereditary disease which occurs spontaneously and it has been estimated that any hereditary detriment is likely to be a good deal less than the detriment due to somatic injury in the irradiated individuals.

The total risk spread over many generations is of the order of 200×10^{-4} Sv^{-1}. About half of this will occur in the first and second generations which are those of prime concern to the radiation worker, so the risk becomes 100×10^{-4} Sv^{-1}. Then productive life is about 30 years in 75, therefore risk becomes 40×10^{-4} Sv^{-1}.

All other tissues

In addition to the tissues discussed there are other tissues (e.g. stomach, liver, large intestine) for which there is evidence that radiation is carcinogenic at moderate doses. The risk factor is known to be low. For other tissues like muscle there is little, if any, evidence of tumour induction by radiation.

The combined risk of malignancy in all these remaining tissues is estimated not to exceed 5×10^{-3} Sv^{-1}.

Table 6

Weighting factors (W_T) - determining the effective equivalent dose

Tissue	W_T
Gonads	0.25
Breast	0.15
Red bone marrow	0.12
Lung	0.12
Thyroid	0.03
Bone	0.03
Remainder	0.30

The weighting factors (W_T) representing the proportion of the stochastic risk resulting from the irradiation of tissue T can be obtained from Table 5 by dividing the risk for each tissue by the overall risk for whole body exposure and W_T values are given in Table 6. These values are used in determining the effective equivalent dose as defined earlier.

Although, then, the Commission no longer proposes separate annual dose equivalent limits for individual tissues and organs irradiated singly the implied values of such limits may be obtained, if required, by dividing the dose equivalent limited

H_E (50 mSv/yr) by the relevant value of W_T and Table 7 shows the values so obtained. It will be seen that the implied limits for tissues of the lowest sensitivity are greater than the limit of 0.5 Sv (50 rem) for non-stochastic effects. Thus, when any of such tissues receives a much greater dose equivalent that all other tissues, non-stochastic rather than stochastic effects will be limiting.

T a b l e 7

Implied dose equivalent limits for individual tissues

Tissues	WT	Limit-Sv	(rem)
W.B.	1	0.05	(5)
Gonads	0.25	0.2	(20)
Breast	0.15	0.3	(30)
R.B.M.	0.12	0.4	(40)
Lung	0.12	0.4	(40)
	⟶	Limited by non-stochastic limit	
Thyroid	0.03	1.7	(170)
Bone	0.03	1.7	(170)
Any other 5 organs	0.06	0.8	(80)

I shall refer to the changes related to intake of radioactive materials later but it is worth noting now that, in this context, non-stochastic effects are limiting in a number of cases, and a practical example, would be when the gastro-intestinal tract is irradiated following the inhalation and/or ingestion of very insoluble compounds.

As before, for occupational exposure the Commission considers two categories which they now call working condition A and working condition B. The former describes the condition where the annual exposure might exceed 3/10th of the dose equivalent limits and latter where it will not exceed. In the former case individual monitoring and medical examination at appropriate intervals is required.

Special mention is made of women of reproductive capacity and women who are pregnant. There is now no restriction on the employ-

ment of the former it being stated that the circumstances under which women would be exposed are unlikely to result in the embryo receiving more than 5 mSv (0.5 rem) during the first two months of pregnancy. For pregnant women, and it is assumed that any pregnancy is unlikely to be of more than two months duration before being recognised, it is recommended that final exposure should be such that it is most unlikely that her annual exposure would exceed 3/10th of the dose equivalent limits.

When making comparisons with other safe occupations it should be realised that the level of risk representative of the safe occupation relates to the average risk for all workers in that occupation. The risk for individual workers varies with the job and is distributed around the average. A similar distribution of individual risks also occurs in radiation work. In many cases of occupational exposure where the Commission's system of dose limitation has been applied the resultant annual average dose equivalent is no greater than one tenth of the annual limit so the application of a dose equivalent limit provides much better protection for the average worker in the group than that corresponding to the limit.

In operational experience of uniform exposure of the whole body in large groups of workers where conditions meet the Commission's recommendations, including the annual dose equivalent limit of 50 mSv, it is found that the distribution of the annual dose equivalent usually fits a log normal function with an arithmetic mean of about 5 mSv and with very few exposures approaching the limit. In the example shown in Fig. 2, for the staff of the C.E.G.B. nuclear power stations in Britain, it will be seen that 50% of the workers are exposed at less than 1.4 mSv (0.14 rem) and none exceeded 40 mSv (4 rem).

Other occupations which are recognised as having high standards of safety are generally considered to be those in which the average annual mortality due to the occupational hazards does not exceed 10^{-4}. In most occupations fatalities, whether due to accidents or disease, are accompanied by a much larger number of less severe consequences whereas radiation exposure, controlled by the limits proposed, would be expected to cause very few injuries or illness to exposed workers other than malignant disease which, of course, corresponds to the fatality in other occupations. The application of the risk factors for radiation exposure discussed earlier, and assuming these mean doses for

occupational workers, means that the average risk in these radiation occupations is comparable with the average risk in other safe industries.

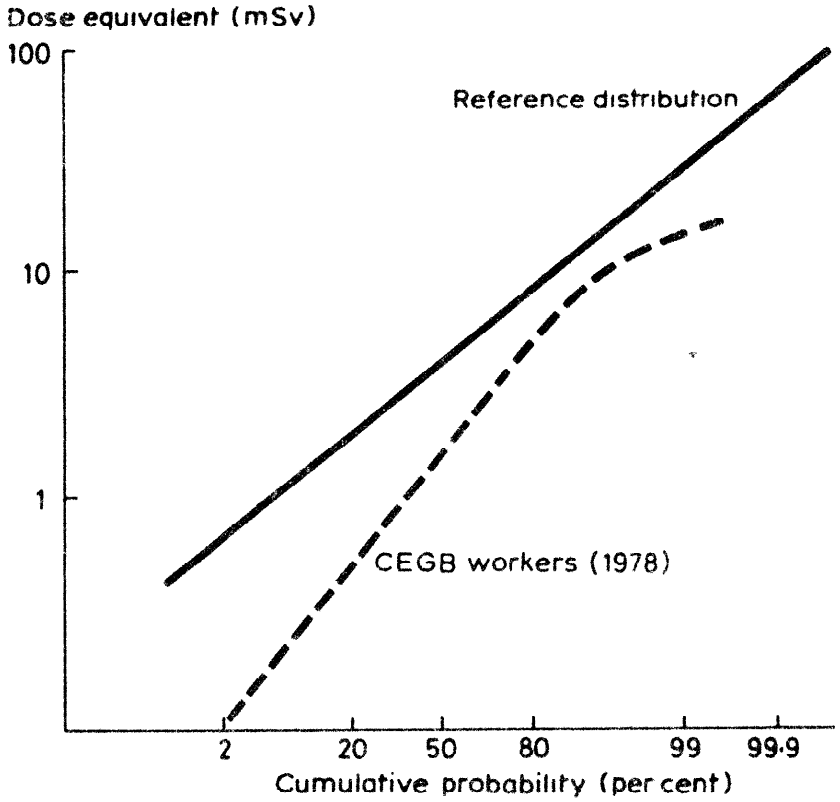


Fig. 2. Distribution of the annual dose of radiation (Courtesy Mayneord, W.V. and Wheatley, B.M. CEGB Research January, 1981)

Internal exposure

So far I have considered only exposure to external sources of radiation. However, more recently, the I.C.R.P. has produced its report No. 30 dealing with limits for intake of radionuclides by workers. This replaces I.C.R.P. 2 and the significant difference in the approach, in the 17 years which separates the two publications, is the replacement of concepts of a Maximum Permissible Concentration (M.P.C.) in air and water and a Maximum Permissible Body Burden (M.P.B.B.) by an Annual Limit on Intake (A.L.I.) from ingestion and inhalation.

Valuable though they were in the state of knowledge at the time M.P.L. and M.P.B.B. were misused. The M.P.L. was specified to prevent the reaching of an M.P.B.B. and although variations in the M.P.L. were permissible within limits it has been misused to imply a concentration which should never be exceeded while the M.P.B.B., which was the activity that would result in the body

after 50 years continuous exposure to the M.P.L., has also been misused to imply a limit on body radioactivity at any time.

As stated earlier for the non-uniform irradiation which results from the intake of radioactive material the logical view is taken that the risk in any year from all the tissues irradiated should not exceed that from 0.05 Sv (5 rem)of wholy body exposure.

(a) Committed dose equivalent

The absorbed dose from external irradiation is received at the same time as the exposure of the tissure or organ to the

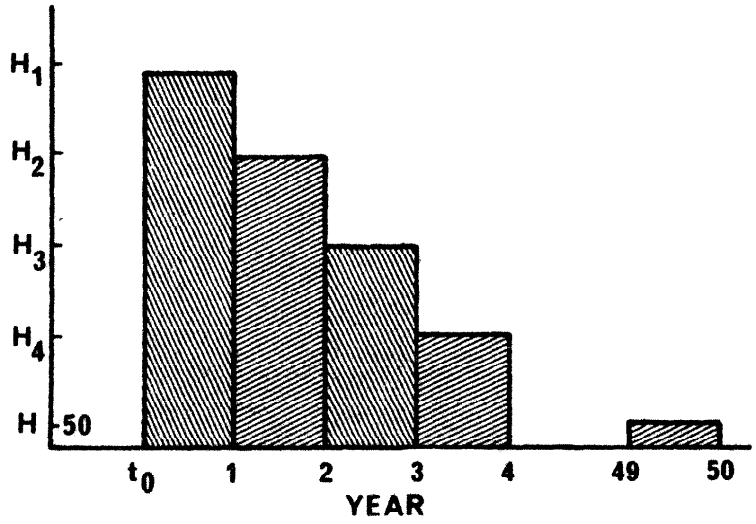


Fig. 3. The dose equivalent rate in a tissue as a function of time after an intake of a radionuclide

radiation field. But in the case of internal irradiation from incorporated radionuclides, the total absorbed dose will be delivered gradually, over a period of time, as the radionuclide decays. The time distribution of this absorbed dose rate will vary with the form and mode of intake of the radionuclide and with the tissue which it is incorporated. The Committed Dose Equivalent, which is the integrated dose equivalent in a particular tissue which will be received by an individual following an intake of radioactive material into the body has been defined by the Commission to take account of this time distribution of dose rate. The integration time has been set as 50 years after the intake, assume to correspond to a working lifetime.

Figure 3 illustrates, in histogram form, the dose equivalent rate in a tissue as a function of time after an intake of a radionuclide. The annual dose equivalents in tissue are represented by

H_1, H_2, ... H_{50} for each year after intake and the committed dose equivalent is given by the total shaded area under the histogram.

Committed Dose Equivalent then is defined as $H_{50} = \int_{t_0}^{t_0+50} H(t)dt$

(b) Dose equivalent limits

In making their proposals concerning D.E.L. the Commission explains as follows: "The Commission's dose equivalent limits for workers are intended to apply to the sum of the dose equivalents resulting from external exposure during one year and the committed dose equivalent from that year's intake of radionuclides... Similar principles apply to the dose equivalent limits for members of the public".

Thus the limits apply to the sum of internal and external exposures, but in practice it will often be found convenient to determine these separately and them sum them.

As experienced earlier the dose equivalent from external radiation is received at the same time as the exposure occurs, so the limit for a given year applied simply to the external radiation received during that year. However the dose equivalent from an intake of a radionuclide may be distributed over a number of years and so it is the committed dose equivalent which must be compared with the limit. For example if an individual takes in a radionuclide at the annual limit each year for 50 years then his dose equivalent during the 50th year would be equal to the dose equivalent limit. This (rather hypothetical) pattern is illustrated in Fig. 4 which illustrates that if the intake in each year does not exceed the A.L.J. then the maximum annual dose equivalent from that radionuclide always be less than will the dose equivalent limit even if intake occurred every year for 50 years.

Thus the A.L.I. is determined so that if D is the resulting committed dose equivalent over 50 years in a tissue and L is the D.E. to a tissue giving the same risk as a whole body exposure of 0.05 Sv (5 rem) then the sum of the ratios D/L for all tissue irradiated should equal unity. Note that there is now a restriction placed on the dose to all the tissues irradiated whereas in I.C.R.P.2 the restriction was placed only on the dose to a critical organ although a number of other tissues might also be irradiated.

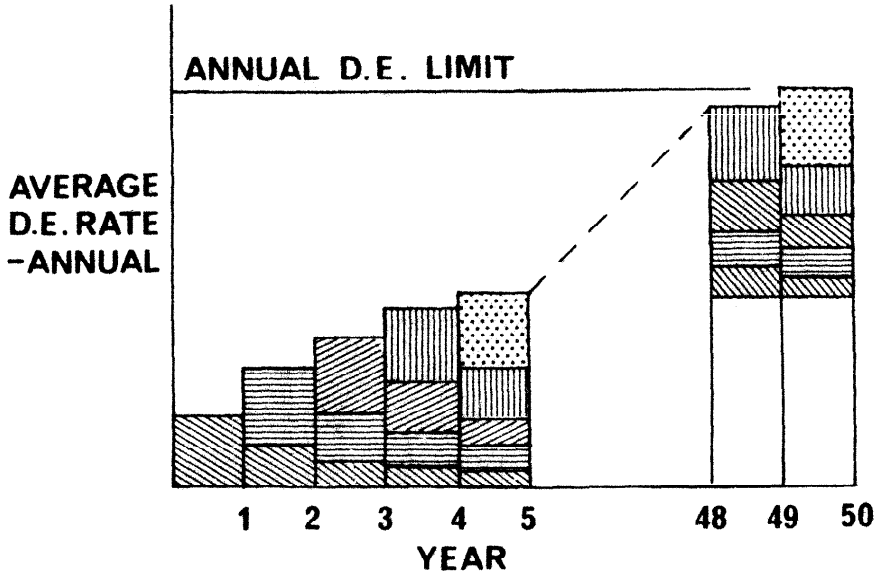


Fig. 4. Cumulative dose equivalent from an intake of radionuclide

Although the Commission's philosophy on dose limitation has, as we have seen, changed this has resulted in only small changes in the limits on intake of radioactive materials. The greater changes have come from improved knowledge about decay schemes and the metabolic behaviour of the materials concerned.

However, and bearing in mind that comparisons cannot be straightforward, of some 250 comparisons which can now be made about half of the new A.L.I.s are within a factor of 3 of former values, and the remainder are about equally divided above and below the old limits, though saying this, should not hide some important changes for individual radionuclides.

Members of the public

Radiation risks are a very minor fraction of the total number of environmental hazards to which members of the public are exposed. It seems reasonable therefore to consider the magnitude of radiation risks to the general public in the light of the public acceptance of other risks of every day life. From a review of available information related to risks regularly accepted in every-day life (Table 8) it can be concluded that the level of acceptability for fatal risks to the general public is an order of magnitude lower than for occupational risks. On this basis a risk in the range of 10^{-6} to 10^{-5} per year would be likely to

Table 8

Acceptability of fatal accident risk to the individual

Annual risk per person	
1 in 1,000	Unacceptable
1 in 10,000	Prepared to spend public money (police, fire precautions etc)
1 in 100,000	Warnings given (swimming, poison etc)
1 in 1,000,000	No worry Lightning strike
1 in 10,000,000	(Reactor accident)
1 in 100,000,000	Meteorite strike

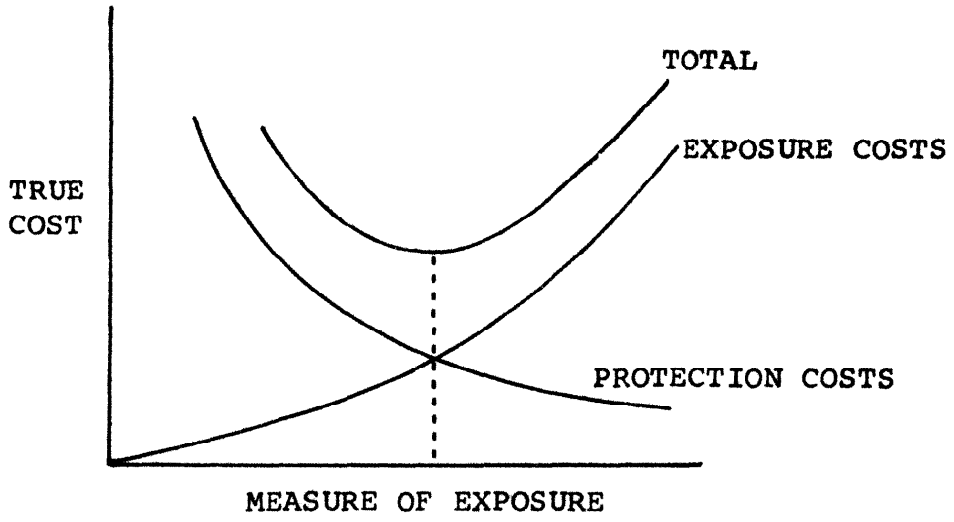
be acceptable to any individual member of the public. We saw earlier that the total stochastic risk from uniform whole body irradiation is of the order of 10^{-2} per Sv (10^{-4}/rem) and so assuming this risk would imply the restriction of the life-time dose to individual members of the public to a value corresponding to 1m Sv/year (100m rem per year)of life long whole body exposure.

In its previous recommendation the Commission made suggestions about population exposure, the limits being set at $^{1}/_{10}$th of those for occupational exposure. The main reason for this lower limit was the possible genetic consequences of exposure of large numbers of individuals. For this reason they set a population dose limit of 5 rem in 30 years. It has become clear that the average dose equivalent in a population is unlikely to reach values of even a small fraction of this 5 rem provided the practices exposing the public are few and cause little exposure outside the critical groups. This, together with the knowledge that genetic effects, while important, are unlikely to be of over-riding importance, is justification for not making new recommendations on dose limits for populations. Rather the Commission believes that each man-made contribution to population exposure should be justified by its benefits.

Cost-Benefit Approach

We have seen that a major objective of radiological protection is to reduce exposures to levels which are as low as reasonably achievable, economic and social factors being taken into account.

While the rationale behind this ALARA principle is attractive problems arise in its application in comparison with dose-equivalent limits which are conceptually simple. For this reason the Commission has recommended the use of Cost-Benefit Analysis to determine whether reduction in levels of exposure are reasonably achievable. Essentially this quantitative technique seeks to compare the harm from radiation against the resources available to reduce exposure to it.



If it is appropriate for Society to pay £5 x 10^5 to avoid one death from cancer and the risk coefficient is 10^{-2}/Sv then the cost of 1 man-Sv = £5 x 10^3

Fig. 5. Cost-Benefit analysis

Resources can be expressed in monetory terms and the analysis, therefore, requires a monetory valuation for radiation induced detriment. The ALARA principle will be satisfied when an increase in the cost of protection is balanced by a decrease in the cost of the detriment (Fig. 5).

In reaching an appropriate decision the costbenefit analysis shifts from consideration of total benefit to the change on the net benefit which could be involved in stipulating that some process be performed at one level of dose rather than another.

The Commission express the requirement mathematically as follows:

$$B = V - (P + X + Y),$$

where: B - net benefit,
V - gross benefit,

P - basic production cost,

X - cost of protection,

Y - detriment.

The optimum net benefit will be attained if

$$\left(\frac{dV}{dS} - \frac{dP}{dS} + \frac{dX}{dS} + \frac{dY}{dS}\right) = 0$$

With any given practice V and P can be considered as constant so the optimisation condition is fulfilled at a value S such that

$$\left(\frac{dX}{dS}\right)_{S'} = \left(\frac{dY}{dS}\right)_{S'}$$

As I said earlier the common measure, through which the consequences for people's health can be directly balanced against the costs of reducing their risk of occurrence is money value. Such a measure has been used for years in comparing risks to health e.g. from traffic accidents and the comparisons have invariably had to be carried out within constraints, usually the total money available, so that the allocation of money for one provision denies its use another.

Radiation, at the low doses associated with occupational exposure will not produce any non-fatal effects and therefore, comparisons with other occupations can be made on the basis of fatalities. (It should be noted, however, that "detriment" as used by I.C.R.P. can include other than health effects, e.g. denial of use of land or buildings, damage to the environment).

However, if we consider fatal consequences only we have to place a value on a human life so that we may determine the cost of a man-Sievert. The evaluation of a human life is extraordinarily difficult. At first sight one might say "go and ask life insurance companies". However, when a man insures his life he does so for the benefit of his dependents - the value could be very different if his only surviving relative is his mother-in-law compared with a situation where he has a wife and three children.

Sometimes implicitly we put a value on a human life by a particular action. Here is an example.

Some years ago there was concern in Britain at the number of farm workers being killed by overturning tractors and a law was passed to require the fitting of safety cabs with the following consequences:

Size U.K. tractor market	$= 0.5 \times 10^6$
Cost of a cab	= £50
∴ Cost of the legislation	$= £25 \times 10^6$
No. of lives saved	= 40/year
Average life of a tractor	= 5 years
∴ Total lives saved	= 200
∴ Value of a life	$= \frac{25 \times 10^6}{20} =$ £125,000

It is clear that for some time to come we are not going to be able to quantify all the components of a cost-benefit analysis and, as has been the case in other fields for many years, an element of judgement will continue to be involved especially where, as I have indicated before, the benefit may be derived by one section of the community and any detriment placed with another section.

Artykuł wpłynął do Redakcji 4 II 1983

Jack H. Martin

ROZWÓJ STANDARDÓW STOSOWANYCH DLA RADIOPROTEKCJI

S t r e s z c z e n i e

W pracy omówiono historię określenia dawki promieniowania jonizującego, tolerowanej przez człowieka. Przypomniano najważniejsze efekty radiobiologiczne oraz podano dopuszczalne dawki, a także współczynniki ryzyka dla poszczególnych tkanek. Przytoczono również metodę obliczenia kosztów związanych z zabezpieczeniem właściwej radioprotekcji.

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