RECENT PAST AND NEAR FUTURE ACTIVITIES OF ICRP COMMITTEE 2*

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The presence of this paper on the program is, I am sure, related to the publication last year of the long-awaited report of ICRP Committee 2 on "Limits for Intakes of Radionuclides by Workers" (1). I will spend most of my time discussing that report, but I think it also appropriate to say something about the committee itself.

RECENT PAST

It has been my privilege to serve as a member of Committee 2 since 1970, and for some years prior to that as a member of its Task Group on Plutonium and Related Elements. Even before 1970, the Committee was at work on the revision of its earlier report on "Permissible Dose for Internal Radiation", which was published as ICRP Publication 2, in 1960 (2). By 1973, the last year of Karl Morgan's tenure as chairman of the committee, the revision was in a draft form covering the major items which eventually appeared last year in Publication 30. The year 1974 marked the beginning of Jack Vennart's tenure as chairman, and was the year the Main Commission adopted its policy of summing risks by means of weighted organ dose, as a replacement for the "critical organ concept." This decision removed the last technical obstacle to completing the report, and we began to actually believe our annual prediction that the report would be published "next year." In fact, publication had to await the issuance of the Commission's basic "recommendations" as included in Publication 26 (3), and the completion of voluminous machine calculations.

I have mentioned the names of Karl Morgan and Jack Vennart, who served so capably as chairmen of Committee 2. I must also acknowledge the major contribution of our late colleague, Geoffrey Dolphin who served as Secretary of the Committee during compilation of the final drafts of Publication 30. The contributions of Norman Adams and Michael Thorne, who assisted the Secretariat in putting the data together, must also be acknowledged. But above all, Walter Snyder must be remembered for the unstinting and patient ministration of his vast knowledge of internal dosimetry to the Committee and to the Committee's publications. I must name one other person-Mary Rose Ford-who assumed the calculational chore after the death of Dr. Snyder in 1977, and averted a catastrophe that everyone feared.

I would not like to leave the impression that Committee 2 was concerned, for the 20 years since 1960, only with the preparation of Publication 30. A supplement to Publication 2 was published in 1964 as a part of ICRP Publication 6 (4). Dosimetry models for the gastro-intestinal tract (5,6) and for the respiratory tract (7) were published in 1966 by task groups of Committee 2. A joint task group of Committees 1 and 2 prepared, "A Review of the Radiosensitivity of the

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Tissues in Bone," which was published in 1968 as ICRP Publication 11 (8). Publication 19, "The Metabolism of Compounds of Plutonium and Other Actinides," appeared in 1972 (9). Publication 20, "Alkaline Earth Metabolism in Adult Man," followed in 1973 (10). The monumental "Report of the Task Group on Reference Man," ICRP Publication 23 (11), appeared in 1975, another memorial to the chairman of that task group, Walter Snyder. Other unpublished task group reports have been important in the development of Publication 30.

The current, and recent past membership of Committee 2 is shown

in Table 1.

Table 1. Membership of ICRP Committee 2

Current	Recent Past*
J. Vennart, Chmn. R.C. Thompson, Vice Chmn. W.J. Bair L.E. Feinendegen M.R. Ford** A. Kaul** C.W. Mays J.C. Nenot** B. Nosslin** P.V. Ramzaev C. Richmond** N. Veall**	G.C. Butler B. Chr. Christensen G.W. Dolphin M. Dousset M. Izawa W. Jacobi J. Lafuma J. Liniecki L.D. Marinelli W.G. Marley K.Z. Morgan P.E. Morrow J. Müller V. Shamov W.S. Snyder C.G. Stewart

^{*}Since work on Publication 30 began in 1967

NEAR FUTURE

Only Part 1 of Publication 30 has thus far appeared in print, and this includes limits and metabolic data for the radioisotopes of only 21 elements (Table 2). Limits for an additional group of 31 elements (Table 3) are in the final stage of compilation and will be soon published as Part 2 of Publication 30. Michael Thorne, for the Committee, has compiled the metabolic data for an additional 18 elements and is working on 25 more. These will be considered by the Committee at its meeting next week, in Brighton, England, and will eventually be included in a Part 3 of Publication 30. Perhaps a Part 4 will be required to complete the elements.

^{**}Appointed subsequent to final drafting of Publication 30, Part 1.

Table 2. Elements included in ICRP Publication 30, Part 1 (with atomic number)

1	Hydrogen	38	Strontium	53	Iodine	90	Thorium
15	Phosphorus	40	Zirconium	55	Cesium	92	Uranium
25	Manganese	41	Niobium	58	Cerium	94	Plutonium
27	Cobalt	42	Molybdenum	84	Polonium	95	Americium
36	Krypton	52	Tellurium	88	Radium	96	Curium
	• •					98	Californium

Table 3. Elements to be included in ICRP Publication 30, Part 2 (with atomic number)

9	Fluorine	26	Iron	45	Rhodium	76	Osmium
11	Sodium	29	Copper	47	Silver	77	Iridium
16	Sulfur	30	Zinc	48	Cadmium	79	Gold
17	Chlorine	35	Bromine	49	Indium	80	Mercury
18	Argon	37	Rubidium	54	Xenon	82	Lead
19	Potassium	39	Yttrium	56	Barium	83	Bismuth
20	Calcium	43	Technetium	61	Promethium	93	Neptunium
24	Chromium	44	Ruthenium	75	Rhenium		

For each "Part" of Publication 30, there will be issued a "Supplement". These supplements will be reproduced directly from computer printouts, and will tabulate the data employed in arriving at the recommended values of ALI and DAC. These data are necessary for the derivation of limits for exposure to mixtures of radionuclides, and to particles varying from the one micrometer diameter assumed for the tabulated ALI's and DAC's. Finally, there will be published a separate report tabulating the radionuclide decay schemes employed in deriving these limits. Much remains to be done before we have finished with Publication 30, but another year or two should see its completion.

While Committee 2 has, in the past, dealt only with problems of internal exposure, since November of 1977 its official title has been "Committee 2 on Secondary Limits," and it is the intention of the Main Commission that Committee 2 should have the responsibility for derivation of secondary limits for external exposure as well as for internal exposure. Committee 3, which formerly dealt with external exposure, is now Committee 3 on Protection in Medicine. However, the Main Commission has acknowledged that, for the immediate future, Committee 2 will be fully concerned with the preparation of secondary limits for internal irradiation.

Aside from the completion of Publication 30, identified future activities of Committee 2 in the area of internal exposure include the following. A task group of Committee 2, with Dr. Nosslin as chairman, is preparing a report on Dose to Patients from Radiopharmaceuticals. This effort is not concerned with establishing limits, but only with estimating patient dose from unit intake; questions of philosophy and medical ethics lie in the domain of Committee 3. Committee 2 will also be concerned with the improvement of internal dosimetry models, in particular those concerned with bone and lung. The bone model of Publication 30 does not take account of the burial of surface deposited radionuclides, and the lung model is applicable only to inhaled particulates. Finally, the Committee will address

itself to the establishment of radionuclide exposure limits for members of the public. The exact manner of formulating these limits has not been determined; however, it has been agreed that an exhaustive appraisal of the internal dosimetry of all radionuclides at all ages is impracticable, and that the approach will involve the application of a correcting factor to the occupational limit, this correcting factor probably being different for different radionuclides.

ICRP PUBLICATION 30, PART 1

Let me now return to a more detailed consideration of Part 1 of Publication 30 (1). I will concentrate on specific aspects of the Publication, which seem most important to its understanding and application. A most obvious change in Publication 30 is the absence of "Maximum Permissible Concentrations (MPC) or Maximum Permissible Body Burdens (MPBB), to which we had become accustomed in Publication 2. They are replaced by Annual Limits on Intake (ALI) and Derived Air Concentrations (DAC); the DAC being equivalent to the old MPC for air, but renamed to avoid the connotation that it should never be exceeded. The new limits are expressed in SI units, without even parenthetical microcuries.

Previous internal radiation exposure standards, as formulated in ICRP Publication 2, were based on limiting the dose equivalent received by the critical organ after a period of 50 years of continuous exposure, critical organ being defined as "that organ of the body whose damage by the radiation results in the greatest damage to the body" (2). The exposure standards derived in Publication 30, on the other hand, limit the annual effective dose equivalent commitment, thus differing in two respects from Publication 2: (1) the limit is on annual commitment rather than on ultimate realization, and (2) the limit is on effective dose equivalent, i.e., the sum of weighted organ or tissue dose equivalents. What are the implications of these changes?

The change from a dose rate achieved after 50 years of exposure to an annual dose commitment has no effect at all in a mathematical sense--one arrives at the same exposure limit by either approach. This is best illustrated graphically, as shown in Figure 1, where A, B, C, and D represent the dose in successive years resulting from the exposure in year n. For simplification, this illustration assumes that dose contributed beyond the fourth year is insignificant and that a steady-state total dose is therefore achieved after four years of constant exposure. It should be apparent from the graph that the annual dose commitment, $A_1+B_1+C_1+D_1$, is numerically identical to the total dose in the 50th year, $A_{50}+B_{49}+C_{48}+D_{47}$. Concern has been expressed, however, by those who must apply these limits, that controlling to an expressed annual limit on intake may prove more restrictive than the old practice of controlling to a fraction of a permissible body burden. Thus, for a radionuclide tenaciously retained in the body, like plutonium, an accidental exposure to several Annual Limits on Intake might be considered an "overexposure", and as such might limit the work status of the exposed individual in future years, even though the actual radiation dose, received or projected during any year, is never more than a small fraction of that allowed on an annual basis. It must be emphasized that such

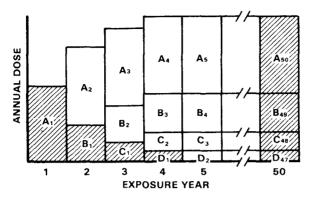


Figure 1. Illustration of dose commitment concept (see text)

an application of the dose commitment concept exceeds the intention of the ICRP, which employs it to calculate ALI's for the control of the environment in which people work, and not to determine the work status of exposed individuals.

The change from the "critical organ concept" to the concept of an effective dose equivalent, has more profound consequences. Instead of applying an assigned dose limit to a single organ or tissue considered critical, the total body limit is applied to the sum of doses received by all significantly exposed organs or tissues, each weighted in accord with its presumed contribution to the total risk of whole body exposure. This is certainly a more logical approach to limit setting and is, in fact, essential to a system based on the limitation Unfortunately, our knowledge of the biological parameters required to implement such a system is not entirely adequate, and the assumptions required because of that inadequacy result in uncertainties with the new system that are probably as large as the more obvious inaccuracies in the old system. And the calculational complexities introduced are formidable. The new system is, however, a more logically consistent one, and if we lack the information to implement it most effectively, it at least focusses attention on these short-I would only caution that our numbers are not as good as the refinement of the calculational procedures might suggest.

A vestige of the old "critical organ concept" still remains in Publication 30, in the guise of an overriding "non-stochastic" limit of 0.5 Sv (50 rem) per year, applicable to any organ or tissue except the lens of the eye, where the non-scholastic limit is 0.3 Sv (30 rem) per year. The weighted organ dose system of ICRP-26 would, in certain instances, allow doses to single organs in excess of 0.5 Sv (50 rem) per year, but this is prevented by the non-stochastic limit.

Let me now illustrate some of the previous discussion by considering, as an example, the derivation of the ALI for ingested Pu. For this exercise I have gathered in Table 4 information which will appear in the supplement to Publication 30, Part 1. Table 4 lists values of committed dose equivalent per Bg ingested, for the tissues

Table 4. Committed dose equivalent (H_{50T}) and weighted committed dose equivalent (W_TH_{50T}) per unit ingestion of soluble ²³⁹Pu in units of Sv/Bq, and derivation of annual limits on intake (ALI)

Tissue	^Н 50Т	W _T	W _Т H ₅ ОТ
Ovaries	2.6 X 10 ⁻⁸	0.25	0.6 X 10 ⁻⁹ 20. X 10 ⁻⁹ 63. X 10 ⁻⁹ 3.2 X 10 ⁻⁹ 27. X 10 ⁻⁹
Red Bone Marrow	17. X 10 ⁻⁸	0.12	
Bone Surfaces	210. X 10 ⁻⁸	0.03	
Lower Large Intest	sine 5.4 X 10 ⁻⁸	0.06	
Liver	44. X 10 ⁻⁸	0.06	

Non-stochastic ALI (bone surfaces):

210 X 10^{-8} Sv/Bq \approx 0.5 Sv/240 kBq

Stochastic ALI:

$$\Sigma_{\text{T}}W_{\text{T}}H_{50\text{T}} = 119.8 \text{ X } 10^{-9} \text{ Sv/Bq} \approx 0.05 \text{ Sv/} \frac{420 \text{ kBq}}{200 \text{ kBq}}$$

of significance. These values result from calculations based on metabolic models and dosimetric models which we will have more to say about later. The largest committed dose equivalent is calculated for the bone surfaces, which would have been considered the critical organ in the old system. We must still calculate a non-stochastic ALI for bone surfaces, which turns out to be 240 kBq; i.e., 240 kBq will deliver the non-stochastic committed dose equivalent limit of 0.5 Sy.

The committed dose equivalent values in Table 4 are multiplied by the weighting factors, W_{T} , as defined in ICRP-Publication 26, to give the weighted committed dose equivalents, which are summed to yield the effective committed dose equivalent. This sum is compared with the committed dose equivalent limit of 0.05 Sv for stochastic effects, leading to an ALI of 420 kBq. Since the stochastic limit is higher than the non-stochastic limit, the non-stochastic limit of 240 kBq is taken as the ALI for ingested soluble plutonium For many radionuclides, non-stochastic limits are controlling and we have, in fact, a limit still based on a single critical organ. We have in the process, however, considered all organs and tissues to insure that a summation of organ risks would not have led to a more restrictive value.

I thought it best to dwell at some length upon the preceding basic changes in philosophy, but it leaves us time to consider only briefly a number of other important changes.

Dose equivalent (H) is now defined as the product of absorbed dose (D), a quality factor (Q), and the product of any other modifying factors (N). Publication 2 employed a conceptually similar "RBE dose", which was the product of absorbed dose, relative biological effectiveness (RBE) and a relative damage factor (n), which applied only to non-radium alpha and beta emitters in bone. Though somewhat differently defined, the new quality factor serves the same function as the old RBE, and the old "n" factor might be considered a specific modifying factor which could be part of N. However, we no longer calculate average dose to bone, so the "n" factor is no longer needed. A significant change has been made in

the numerical value of the quality factor for alpha particles, which is now 20 rather than 10.

Dose equivalent commitment in a given "target" organ or tissue $(H_{50}T)$ is now calculated by taking into account the "crossfire" from radiations originating in all other significant "source" organs or tissues, as well as the radiation originating in the target organ itself. This is a complex process, some of the intermediate stages of which will be detailed in the supplements to Publication 30.

The distribution and retention of radionuclides among and within the various source organs is determined by application of suitable metabolic and dosimetric models. These models, for most elements, have become considerably more complex during the interval between Publication 2 and Publication 30. General dosimetric models for the respiratory system, the gastrointestinal tract, for bone, and for submersion in a radioactive cloud, are described in Publication 30, Part 1. The model for the respiratory system is that developed by the Task Group on Lung Dynamics (7), as modified in ICRP Publication 19 (9). The model for the gastrointestinal tract is based on the model developed by Eve and Dolphin (5,6). The bone model estimates dose to red marrow and to bone surfaces, for cortical and trabecular bone; radionuclides being assumed to deposit either uniformly throughout bone or on bone surfaces. For most elements, detailed information on distribution within bone is not available. In the absence of more specific information it is assumed that: (1) alkaline earth radionuclides with radioactive half-lives greater than 15 days are uniformly distributed throughout the volume of bone, (2) radionuclides with radioactive half-lives of less than 15 days are uniformly distributed on bone surfaces, (3) radionuclides on bone surfaces are equally distributed between trabecular and cortical bone, and (4) radionuclides uniformly distributed throughout the volume of bone are present 20% in trabecular bone and 80% in corti-

Specific metabolic models are employed for each radionuclide, and these are not restricted to any particular mathematical form, although most are systems of first order differential equations with constant coefficients. More than one value for the absorption coefficient from the gastrointestinal tract and/or lung may be employed, to represent different compound forms; the respiratory tract model also provides for three different classes of compounds, based upon their assumed clearance rate from the lung. The metabolic models and briefly summarized supporting data are presented separately, for each element in Publication 30, just preceding the tabulated values of ALI and DAC.

Finally, let us look at an example of the actual limit values, as tabulated for plutonium. In Table 5, values are shown for $^{239}\text{Pu};$ Publication 30 lists values for 12 isotopes of plutonium--from ^{234}Pu to $^{245}\text{Pu}.$ Oral ALI's are listed for compounds exhibiting two absorption fractions, the value of 10^{-5} applying to oxides and hydroxides and the value of 10^{-4} applying to other commonly occurring compounds. It is indicated that the ALI's are based on a non-stochastic limit for irradiation of bone surfaces and that in the absence of such a limit the stochastic limit would have been the value shown in parentheses. Inhalation ALI's are listed for two compound classes, Class Y being applicable to plutonium oxide

Table 5. ALI (Bg) and DAC (Bg/ m^3) (40 h/wk) values for 239 Pu as listed in ICRP Publication 30

			Inhalation			
	Oral		Class W	Class Y		
	$f_1 = 1 \times 10^{-4}$	$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-3}$		
ALI	2 X 10 ⁵	2 X 10 ⁶	2×10^{2}	5 X 10 ²		
	(4 X 10 ⁵)	(3 X 10 ⁶)	(4×10^{2})	(6×10^2)		
	_ Bone surf.	Bone surf.	Bone syrf.	Bone surf.		
	[15 X 10 ⁵]*	[9 X 10 ⁶]*	[2 X 10 ²]*	[27 X 10 ²]*		
DAC			8 X 10^{-2}	2 X 10-1		
			[7 X 10- ⁻]*	[15 X 10 ⁻¹]*		

*Values derived from ICRP Publication 2.

and Class W to other commonly occuring compounds; the fraction absorbed from the gastrointestinal tract after clearance from the lung is different for the two classes. Again the ALI's are based on a nonstochastic limit. Values of the DAC are shown for the two compound classes.

As a matter of interest, I have listed in brackets, in Table 5, the values for ALI and DAC which one would obtain from the old limits of Publication 2. The DAC values are derived from the old $(MPC)_a$ values by a simple change of units. The ALI's are derived from the MPC values by multiplying by the assumed annual intake of water or air, and by changing units. It will be seen that, except for inhaled Class W compounds, where there is no significant change, all Pu limits have become more restrictive by a factor of about six. Similar comparisons, involving a representative isotope of each element, suggest that about 1/3 of Publication 2 limits have become more restrictive, by as much as a factor of 100; about 1/3 have become less restrictive, by as much as a factor of 25; the remaining 1/3 have changed by less than a factor of 2. REFERENCES

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