Transport of Radium Sulfate from the Lung and Its Elimination from the Human Body Following Single Accidental Exposures[1]

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IN THE PRESENCE of insoluble radioactive dust, it is not always possible to determine whether the permissible level in air should be governed by the radiation delivered to the lung and air passages or by the radiation delivered to the critical organ where ultimately most of the radioelement, mobilized from the lung but not excreted by the body, will be deposited. This perplexing situation is created not so much by lack of knowledge of the fraction deposited in the various parts of the respiratory tract (1–3) as by the scarcity of information on the rate of elimination of the particulate matter by the human lung. This statement applies not only to the recently discovered artificial radioelements but extends also to radium, which as early as 1926 was shown by Reitter and Martland (4) to gain access to the skeleton by way of the lungs.

In order to gather information on the subject, six individuals exposed to radium sulfate dust as a consequence of two industrial accidents were selected for study. Five persons (R., K., Ch., Ca., and S.) from the first accident (5) have been studied for a period of a year, through the collaboration of Dr. Eugene L. Saenger of the Cincinnati General Hospital; the sixth, technician G. from our laboratory, has been followed more closely for 250 days and will be under observation to the limit of our resources in sensitivity. In both instances the inhalation lasted for only a few minutes following the rupture of 50-mg. radium capsules containing approximately equal parts of $RaSO_4$ and $BaSO_4$.

This study is based on the following observations: (a) exhalation rate of radon, (b) measurements of gamma-ray activity from the whole body, (c) measurement of gamma-ray activity from the thorax, and (d) measurement of radium in the excreta. It has been possible to estimate from a and b the total-body burden according to accepted practice, and to gain by means of procedure c some information as to the localization of the $RaSO_4$. Owing to difficulties of a practical nature, a complete excretion study was feasible only in the case of our technician; only scattered data are available for the others.

THE BODY BURDEN

The quantity of radium in the body which produces radon at the rate exhaled by the host can be computed by the expression:

$$1 \ \mu g. \ \mathrm{Ra} \equiv 2.1 \ \mu\mu c. \ \mathrm{Rn \ per \ second}$$

This fraction does not contribute to the measured gamma-ray activity and it is, therefore, added to the latter in the estimate of the total radium burden.

After appropriate trapping of water, and through a continuous process of adsorption, all the radon contained in the air exhaled by a patient in ten- to twenty-minute periods was collected on charcoal. The radioactive gas was then transferred into suitable ionization chambers connected to an electrometer, and the calibration was performed by comparison with the ionization produced by known fractions of Rn liberated by standardized radium solutions. By such large sampling the sensitivity of detection may be increased considerably over the more conventional technic which utilizes only one

or two liters of air, and, for equal exhalation rates, the precision of the measurement is likewise improved.[2]

The gamma-ray activity of the whole body was measured, whenever possible, by a modification of the basic method originated by R. D. Evans (6) in his investigations on chronic radium poisoning. Whenever the Ra content of an individual was found too low to yield significant results by this method, the estimate was based on direct comparison with the more "active" individuals of the group, properly measured by the basic method, and rigidly seated in a "Standard" chair under reproducible conditions. Although the latter procedure is open to objections, on the ground that it is valid only when the patterns of Ra deposition in the body are identical, it was felt that errors were minimized by restricting the comparison to individuals who were exposed to Ra at the same time. An increase in sensitivity of a factor of about ten could be obtained with reasonable accuracy. The detecting instrument consisted of a NaI crystal (1.5 inches in diameter and 2.5 inches

long), attached to a 5819 photomultiplier provided with a linear amplifier and single channel pulse analyzer with variable band width.[3]

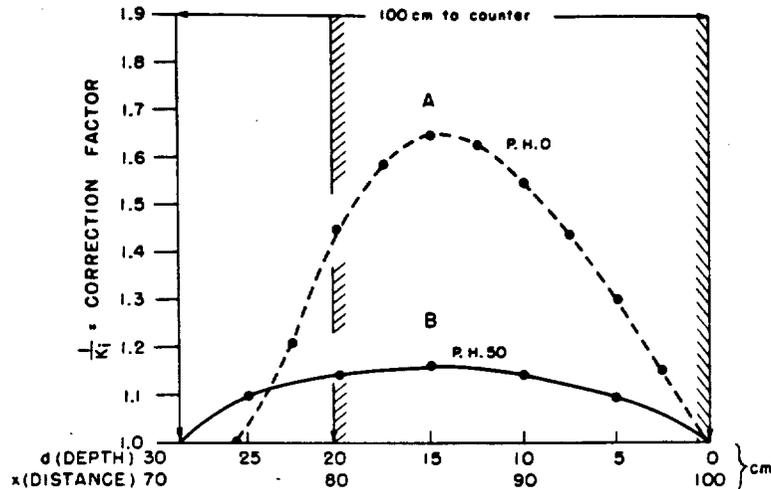Experiments were performed to utilize the spectrometric potentialities of this counter in order (a) to increase the ratio of the patient signal to the background noise accruing mainly from natural radioactivity in the surroundings and (b) to ascertain whether the estimates of radium content in any single individual would prove independent of the pulse size band selected for measurement. It was found that the signal (circa 500 counts per minute per microgram of Ra at 1 meter) to background noise ratio could be substantially improved by operating in a room lined with 1/8 to 1/4 inch of lead and by registering all pulses generated by electrons of energy between 30 to 370 kev.

Studies of the response of the counter to the presence of Ra embedded at different depths in either Presdwood or water phantoms indicated that, on account of

Fig. 1.   Variation of the scattering correction factor K_1 with depth of Ra source in a Presdwood phantom of thickness t = 20 cm. and 30 × 30 cm. portal area.   Curve A: Band width 30–370 kev.   Curve B: Band width above 370 kev.
The distance, x, between the detector crystal and the back of the phantom is kept fixed at 1 meter;   depth, d, is measured from the distal surface of the phantom.
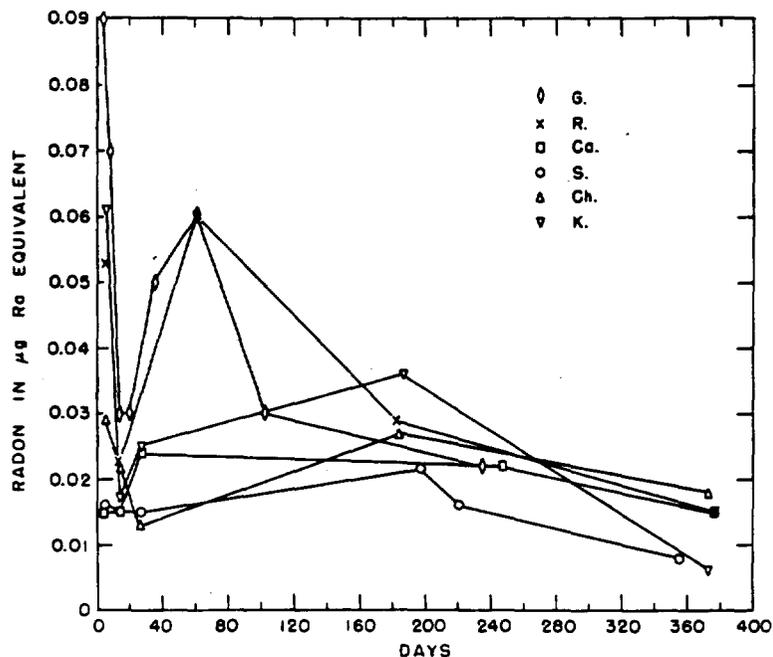
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[2] Details of the technic will be published elsewhere.

Fig. 2. Exhalation rates of radon, in μg. of Ra equivalent, plotted as a function of time after accidental exposure.

gamma-ray scattering, the counting rate depended in a complex manner on the depth of the source, on the thickness of the phantom, and on the pulse height band of the analyzer. When the distance from the back of the phantom to the counter was kept fixed at 1 meter, the counting depth dose curve, per microgram of Ra, could be expressed as

$$K_1 N_4 x^{-2} e^{-\mu d}$$

where

μ = coefficient of absorption per centimeter of phantom (broad beam)

d = depth of source in phantom in centimeters

x = source to counter distance in meters

N_4 = net counting rate due to 1 μg. at 1 meter in air

K_1 = a scattering correction factor which depends on d and on the pulse height band selected for measurement

The variation of K_1 with d for two pulse height widths is illustrated in Figure 1.

Following Evans's analysis, the Ra burden in any individual can be written as:

$$\mu g. = K(1 - t) (N_1 N_2 / N_4^2 e^{-\mu t})^{1/t}$$

where

t = ratio of the average thickness of patient to the distance of the counter to the distal surface of the patient

N_1 = net reading of patient facing counter when placed on arc of a circle 1 meter in radius, the center of which is at the counter

N_2 = same as N_1 but with patient facing away from counter

The factor K is calculated by the expression $K = (K_1 K_1')^{1/t}$, where K_1 and K_1' are the scattering correction factors corresponding to depths d and $(t-d)$ respectively; they can be read off Figure 1 for the particular phantom thickness illustrated. The variation of K with depth of the source is rather small, especially if the pulse height band above 370 kev. is used. This, however, is not always possible if the gamma activity is low, since the ratio of the signal to background noise is then considerably smaller. It is preferable in these instances to risk a greater, but definitely limited, uncertainty in K in order to gain adequate statistical accuracy in determining N_1 and N_2.
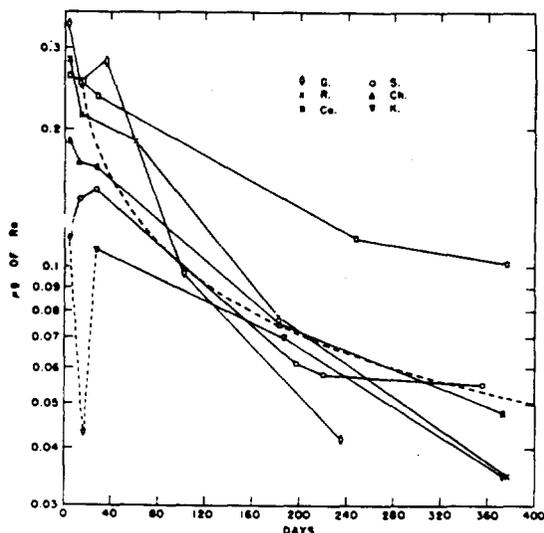
Fig. 3. Total Ra burden in the body (log scale) estimated from both Rn and gamma-ray activity measurements. Dashed curve: theoretical fractional retention of soluble salts, equation (2).

On the assumption of an average depth $d \simeq 5$ cm. we have calculated: $K = 0.7$ as the scattering factor for pulse height band 30–370 kev. and $K = 0.93$ for pulse sizes above 370 kev. Confidence in this procedure has been established by measurements on cases of chronic Ra poisoning, in test subjects to whom known amounts of Na^{24} were administered orally, and in a balance study performed with one of our cases (vide infra).[4]

The exhalation rates of Rn, expressed in equivalent micrograms of Ra, are plotted in Figure 2. It will be noticed that all but 2 of the cases show a sharp decrease within 15 days of the exposure, followed by an increase, and later by a more moderate decrease. It is realized at this time that the exhalation of Rn should have been followed

[4] In a group of 6 Ra cases, the maximum discrepancy obtained by the use of the two pulse heights was ±11 per cent for any one individual and less than 3 per cent on the averages for the groups.

In the experimental group given Na^{24}, all measurements yielded consistent results within 10 per cent of the actual amount given, although the weights of the volunteers ranged from 96 to 176 pounds. Moreover, although measurements were made from one minute to thirty-six hours following ingestion, the computed amount remained constant despite the considerable diffusion of the radioelement within that time. A more detailed evaluation of the method will be presented elsewhere.

more closely during the 15- to 180-day interval as exemplified by the curve obtained from our technician (G.). Although there exists a possibility that, in the Cincinnati cases, the initial decrease in Rn exhalation might reflect in part the decrease in Rn content of the laboratory air in the first two weeks following the accident, there is much more reason to suspect that this decrease is due instead to the excretion of radium dust initially swallowed and dissolved in the gastrointestinal tract. This view is supported by the fact that similar behavior was exhibited by Rn exhaled by our technician (who was removed from any Rn contamination immediately following the second accident) and in particular by his excretion pattern (vide infra).

THE TOTAL-BODY BURDEN

The sum of the fractions yielded by Rn and gamma-ray activity measurements is shown in Figure 3 as a function of time after exposure. Since it was not feasible to proceed with the measurements on the first day of the accidents, it is impossible to compare directly the retentions with those investigated by Norris and Kisieleski (7), Hurst (8), and Norris et al. (9) in experimental animals and in man following injection of $RaCl_2$. It is possible, however, to compare the slope of the curves. It will be recalled that these investigators found that the rate of excretions dq/dt could be related to the injected amount, q_0, by an expression of the following type

$$dq/dt = -Aq_0(t + 1)^{-b} \qquad (1)$$

where A and b are constants. Upon integration, the retention, q, becomes

$$q = q_0 \left\{ 1 + \frac{A}{1 - b}[1 - (t + 1)^{1-b}] \right\} \qquad (2)$$

If in the last equation values of the constants are assumed in fair accord with the data on $RaCl_2$ injected in animals and man ($A = 0.50$, $b = 1.50$), the dotted line in Figure 3 is obtained. By inspection, it can be ascertained that the slopes are roughly similar but that in some

cases the retention of inhaled RaSO₄ will be less than that expected from injection of soluble Ra salts. We shall have an opportunity to return to this subject later in this paper.

Of practical interest are the ratios of Rn to total Ra found in this group. They are shown in Table I for those cases in which measurements of total gamma-ray activity by the absolute method were possible and hence more reliable. It should be noted that they are lower than the values re-

ported in cases of chronic Ra poisoning many years after the date of administration (6, 9). Therefore, shortly after an inhalation accident, the burden is likely to be underestimated severalfold if the current practice of assuming the chronic partition coefficient of 0.5 for Rn is adopted, but the error would be smaller if the exposure had lasted for several years. Except for venturing that the ratio of Rn to Ra is low because some of the Ra is retained in the non-emanating form of RaSO₄, we are un-
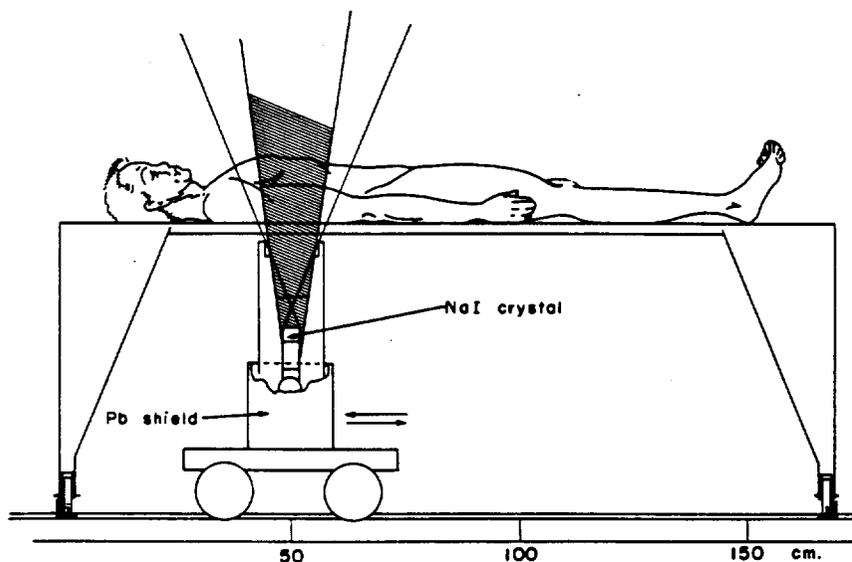

Fig. 4. Arrangement for the investigation of localized sources of gamma-ray activity (scanning measurements).

TABLE I: MEASUREMENT PATTERN OF CASES R., CA., AND G.*

Days (1)	Name (2)	Rn (3)	Body γ-Ray (4)	Total Radium (5)	Rn/Total Ra (6)	Calculated Extra Skeletal Ra (7)	γ-Lung (8)	Ratio (9)
61	R.	0.060	0.128	0.188	0.32	0.102	0.045	2.26
182	R.	0.029	0.049	0.078	0.37	0.037	0.023	1.61
27	Ca.	0.024	0.211	0.235	0.10	0.201	0.113	1.78
247	Ca.	0.022	0.094	0.116	0.19	0.085	0.036	2.36
376	Ca.	0.015	0.087	0.102	0.15	0.081	0.027	3.00
3	G.	0.09	0.248	0.338	0.27	0.210	0.245	0.86
13	G.	0.03	0.222	0.252	0.12	0.209	0.194	1.08
35†	G.	0.05	0.232	0.282	0.18	0.211	0.102	2.07
35‡	G.	0.05	...	0.200	0.25	0.111	0.102	1.09
102	G.	0.03	0.067	0.097	0.31	0.054	0.029	1.86
235	G.	0.022	0.02	0.042	0.52	0.011	0.010	1.10

* Quantities in columns 3, 4, and 8 were measured directly. Column 7 was calculated as difference between values of Column 5 and skeletal radium. The latter was assumed to be given by the Rn values (Column 3) divided by the partition coefficient Rn/Ra total = 0.7 found by Norris *et al.* (9) as an average in cases of chronic Ra poisoning. In Column 9 are the ratios of the values of Column 7 to Column 8 (see text).

† Measurements based on Rn and gamma-ray activity.

‡ Estimate based on measurements of burden at three days less intervening excretion.

able to explain its relative constancy in the first two cases shown in Table I. Its tendency to reach higher values in the third case, as the time interval between accident and observation increases, could be attributed to redistribution and ultimate deposition in bone. Although par-

looked at a circular section of the chest, 15 cm. in diameter at the height of 10 cm. above the table. Travel of the table and counter along two mutually perpendicular axes allowed exploration of any region of the body. Typical traces of the activity detected along the axis of the body are
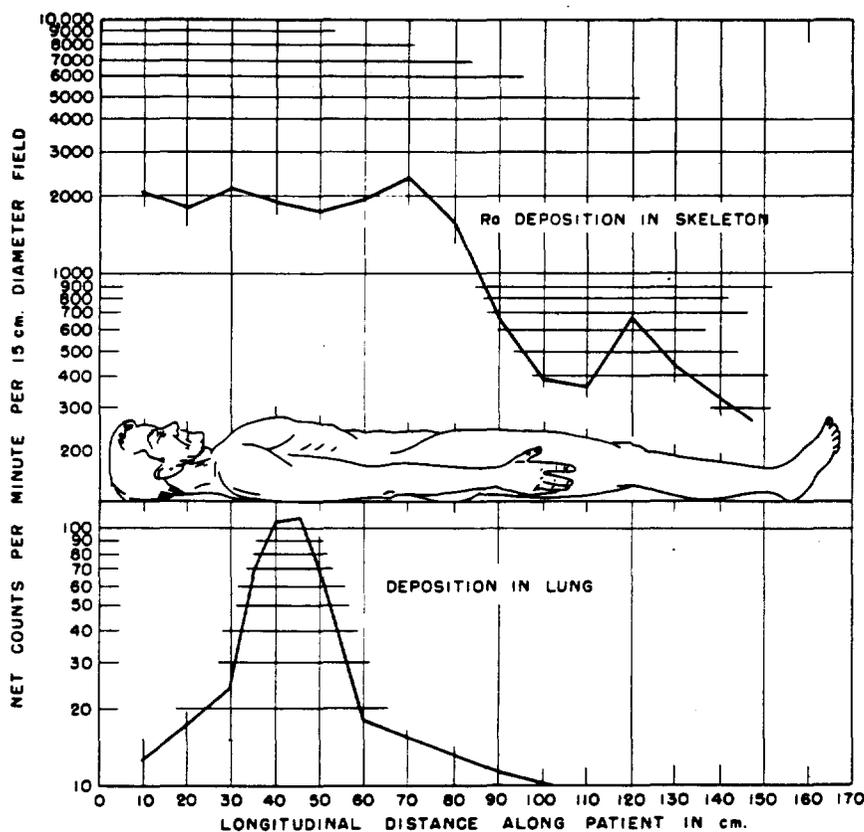

Fig. 5. Typical gamma-ray scanning curves. *Above:* Ra deposition in the skeleton 25 years after injection of RaCl2. *Below:* Deposition in the lung in case R. 61 days following accidental inhalation of RaSO4.

ticle size of the salt may play a role in the data presented here, we are in no position to evaluate its importance since, due to the accidental nature of the exposure, no special study of this type was possible at the moment in which inhalation took place.

THE LUNG BURDEN

The localization of Ra in the lung was studied by scanning the thorax with a scintillation counter according to the scheme illustrated in Figure 4. The NaI crystal, provided with a lead collimator,

shown in Figure 5; they illustrate the different patterns provided by skeletal and pulmonary deposition.

The most complete set of readings obtained in a single individual of our group (G.) is shown in part in Figure 6. It will be noticed that transverse scanning yields curves wholly comparable with those obtained by longitudinal scanning and that in this case the Ra must have been concentrated originally in a rather small volume (~300 c.c.) centered at the level of 4th or 5th thoracic vertebra. The latter state-

ment is self-evident when the half-width[5] of the curve at 7 days after exposure is compared with the half-width obtained by moving a point source at mid-point of a 20 × 30 × 30-cm. phantom of Presdwood. Because of the long counting times required to yield estimates of reasonable precision,
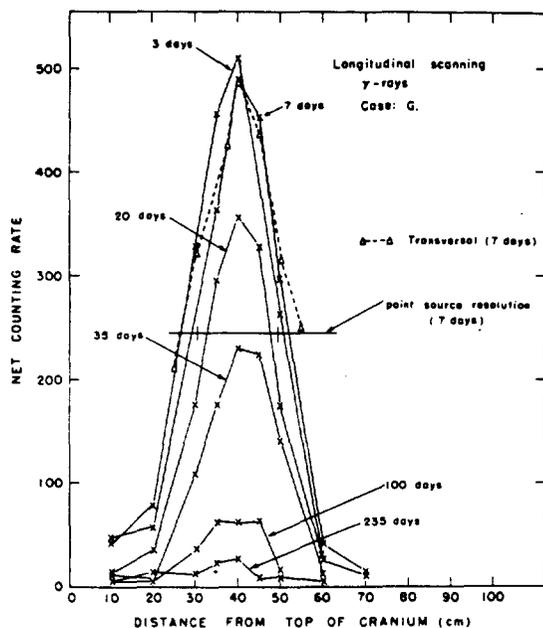

Fig. 6. Gamma-ray scanning curves along lontigudinal axis of subject G.

not all cases were scanned to the same extent. Instead, a few regions around the field of maximum activity were surveyed for periods as long as a year. To account for changes in counter sensitivity, the gamma-ray activity was compared with that of a known amount of Ra located as a point source on the collimator axis in a phantom 20 cm. thick at a height equal to half the thickness of the person's chest. This procedure was deemed justified, since equality in readings obtained with the patient in the prone and supine positions indicated that the deposits were roughly symmetrical with respect to the median anteroposterior plane. By integration of the area under the in vivo scanning curves,[6]

[5] Width of the curve at a height equal to one-half the maximum height.

[6] More correctly volumes under curved surfaces, approximately conical in shape.
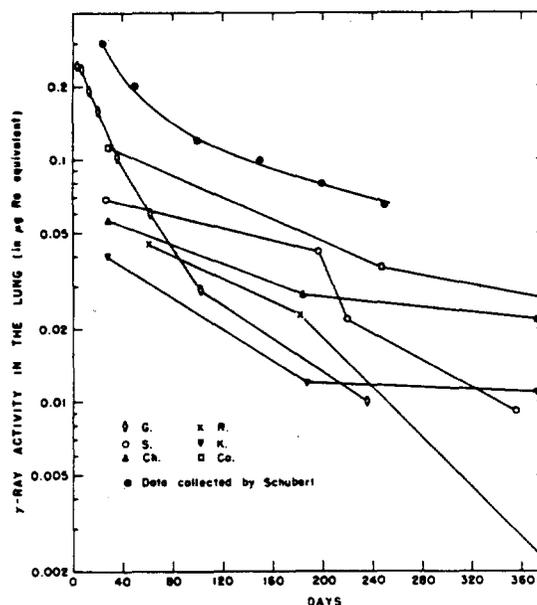

Fig. 7. The time variation of the total gamma-ray activity in the lung, expressed in µg. of Ra equivalent. Data collected by Schubert (11).

and by comparison with areas under analogous curves obtained by scanning the Ra source, it is possible to arrive at fair estimates of the total gamma-ray activities in the lungs in terms of a Ra source in equilibrium with its decay products. These are shown in Figure 7, plotted on a semilogarithmic scale as a function of time.

Although these data do not carry per se unambiguous meaning on Ra metabolism—since they could be influenced by an indeterminate change in the Rn-emanating fraction of the lung deposit—they are, nevertheless, of direct significance in the field of radiation protection.[7]

It will be noted that the decrease is not always exponential and that the half-lives vary from 32 to 140 days within 6 months after exposure. The average is 118 days with a root mean square deviation of ±25 per cent.

It should be noted that, although for two individuals the decrease in lung activity is distressingly small from about 6 months to a year, no account has been taken of the presence of extrapulmonary

[7] This statement becomes obvious when one considers that the disintegrations of Ra in the body, not followed by the exhalation of Rn, are, energetically speaking, about six times as effective as those that are.
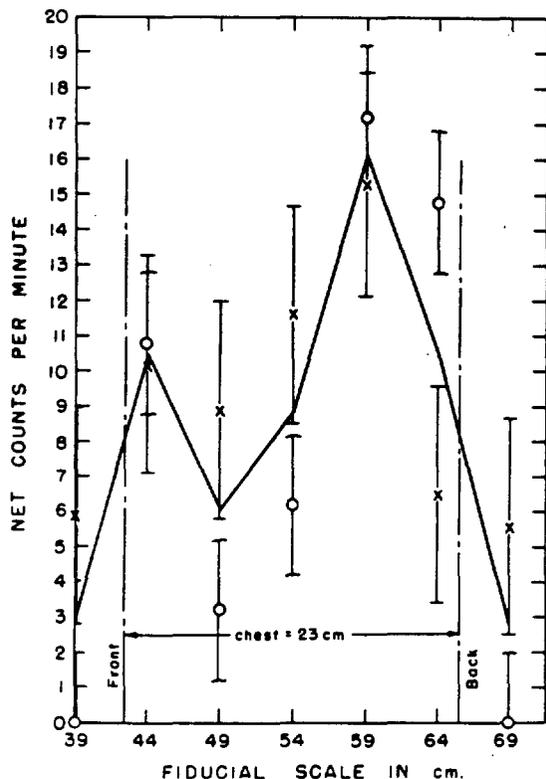
Fig. 8. Anteroposterior scanning curve of subject G., x at 264 days, O 241 days. Curve represents the average. Note rather large experimental error.

Ra within the field of view of the counter. A suitable correction for this could not be made in general because of the low activities involved. An attempt was made for the case of G. by scanning several times at a single level with the subject lying on his side and with his arms out of the field of the counter. Although the evidence (Fig. 8) is not complete, because the scanning was not repeated at various levels of the thoracic skeleton, it indicates, nevertheless, a certain correlation between counting rate and bone mass. Such correction could lower the values of Figure 7 by as much as one-third, beyond the time interval of 6 months. If this be true, the biological half-life of 120 days on the average would extend to as long as a year after inhalation.[8]

These findings, therefore, are reassuring from the standpoint of lung transport but

point rather critically to the need of establishing the concentration of inhaled deposits with a precision higher than that attainable with our scanning methods.

THE EXCRETION OF RADIUM

Circumstances of practical nature precluded significant measurements of excreta from the Cincinnati cases, but adequate data were obtained for the case of our technician (G.) through the collaboration of the bioassay laboratory of the Health Services Division.[9] Both feces and urine samples were frequently assayed for radium content by the technic described by Russell, Lesko, and Schubert (12). Although complete 24-hour collections were rarely possible, the radium content in the latter was estimated on the basis of a urinary daily output or 1,250 c.c. and a dry fecal sample of 30 gm. per day, in accordance with accepted physiological data (13, 14). The urinary excretion represented 2 to 8 per cent of the total. By integration of the total excretion curve for suitable intervals of time, and by the subtraction of the values thus obtained from the total-body burden measured at 3 days, it was possible to compute the retention at later times and to compare it with the total-body content as estimated by means of Rn and gamma-ray measurements. The results of the comparison are shown in Figure 9, where it can be seen that, except for the values at 35 days, the agreement between the two estimates is entirely satisfactory.[10]

For the purpose of discussion, the rate of total excretion is plotted in Figure 10 as percentage of body burden; it is compared with the values reported by Aub et al. (15) on a human case following inhalation of $RaCl_2$ dust. It will be noticed that, although in the latter instance excretion can be expressed satisfactorily as

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[8] This half-life is similar to that found by the Rochester group in the lungs of dog exposed to uranium oxide (10) and consistent with the data gathered by Schubert (11).

[9] We are indebted to Dr. L. S. Myers for these valuable data.

[10] The agreement at 35 days could be substantially improved if the assumption were made that this person consumed during the summer months less food than in the succeeding winter and hence that the dry fecal sample was 20 instead of 30 grams.
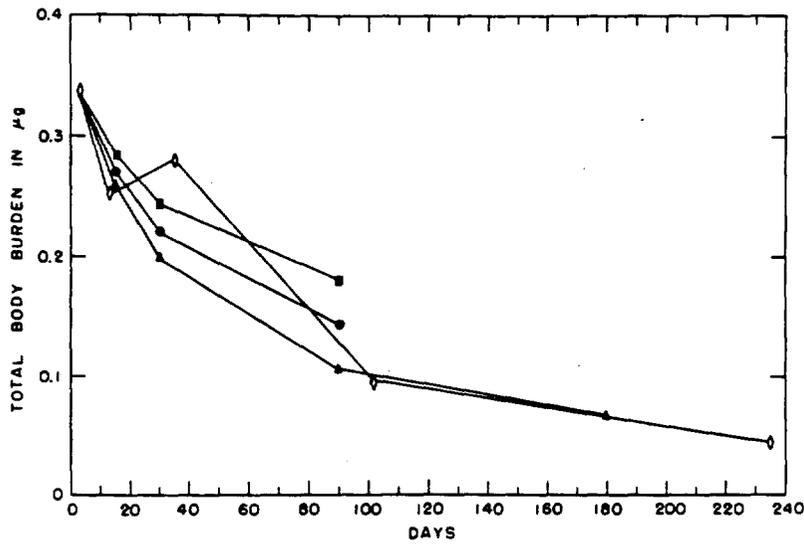
Fig. 9. Subject G. Comparison between estimates of body burden. ◊ = values obtained by direct measurements of Rn and gamma-ray activity. ◆, ●, and ▲ = values obtained by subtracting from the burden at 3 days the integrated total excretion. The latter was computed on the basis of 20, 25, and 30 gm. of dry feces per day and 1,250 c.c. daily urinary output. Date of accident June 13, 1952.
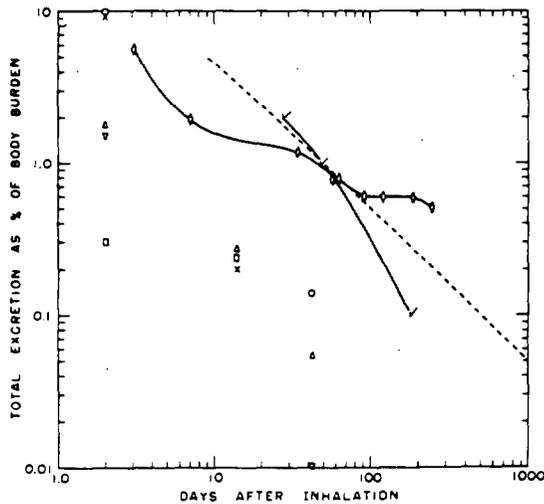

Fig. 10. The daily excretion expressed as per cent of body burden. ◊ Case G (RaSO₄ inhalation). ∇ Case RL. (RaCl₂ inhalation) (Ref. 15). The broken line represents the theoretical value based on a power function of time, equation (1). Other points: scattered data from Cincinnati cases according to key of Figure 2.
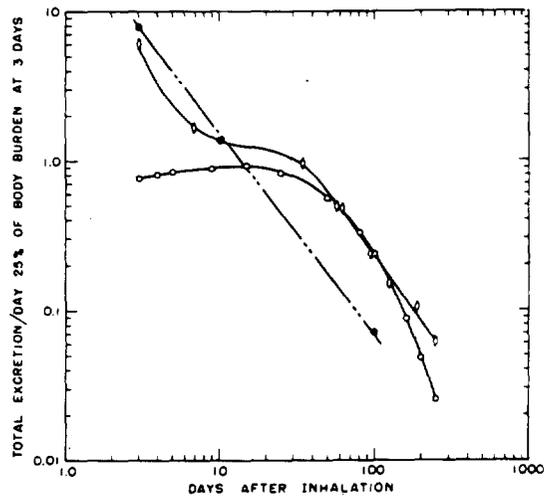
Fig. 11. Daily excretion expressed as per cent of Ra content 3 days following exposure. ◊ Case G. ● Average data of Hurst (8) and Norris (9) on dogs injected with RaCl₂. O the theoretical excretion curve, equation (3).

an inverse power function of time, this cannot be done following RaSO₄ inhalation. Our excretion data are also shown in Figure 11, where they are plotted as percentage of body burden at 3 days in order to compare them with similarly plotted results of Hurst (8) and Norris et al. (9) on

dogs injected with RaCl₂. The fair agreement in slope exhibited by the two curves for the first few days suggests that the initial excretion phase of G. could be ascribed to elimination of RaSO₄ ingested[11]

[11] Ingested either directly or as a result of swallowing the dust mobilized from the trachea and bronchial tree by ciliary action.

and subsequently solubilized by the gastro-intestinal tract. This view is consistent with known solubility of $RaSO_4$; it finds support, also, from (a) the fact that the Rn exhaled—presumably set free from the dissolved Ra in the gut—has been observed to decrease rapidly during this interval of time (see Fig. 2), and (b) the fact that this decrease in Rn (0.06 µg. Ra equivalent from the 3rd to the 13th day) represents 70 per cent of the measured decrease in body burden within the same interval.[12] A similar relationship between exhaled Rn and excretion has been noted to follow injection of soluble Ra salts in dogs, and it is suspected to occur also in man following similar circumstances (9).

The elimination rate found by us after 100 days, however, is much greater than that found for the case of inhaled soluble salt (Fig. 10) and seems to be sustained at these levels by the mobilization of $RaSO_4$ from the lung. Indirect support for this hypothesis may be found in the following analysis: if λ is the fraction of lung burden mobilized per day, the actual amount made available daily to the body will be equal to $\lambda q_0 e^{-\lambda t}$, if q_0 is the total amount of Ra deposited initially in the lung. If this amount is assumed to behave thereafter as soluble Ra salt, then it will be excreted according to an expression of the type $\lambda q_0 e^{-\lambda t} \times A(T-t+1)^{-b}$, where $(T-t)$ is the time interval between pulmonary release and excretion, and A and b are the empirical constants found to describe correctly the elimination of soluble Ra salts in man. It follows, therefrom, that at any time, T, after inhalation the excretion rate dq/dt will be given by an integral of the type

$$\frac{dq}{dt} = \lambda q_0 A \int_{t=0}^{t=T} e^{-\lambda t} (T - t + 1)^{-b} dt \quad (3)$$

We have evaluated this expression with $A = 0.5, b = 1.50, \lambda = 0.02$, and $q_0 = 0.248$ µg., the first two values in accordance with the data on man and the last two from

the lung activity of the particular individual in question (Fig. 7); the values expressed in per cent of the burden at 3 days are plotted in Figure 11 and are shown to be in reasonable agreement with the late phase of the experimental elimination curve.

It should be realized that this analysis cannot be expected to elucidate the actual mechanism involved in the transport of $RaSO_4$ from the lung. Thus, the exponential rate of disappearance, based on external activity of the lung, could represent either the rate of solubilization at the alveolar interface or rate of removal by macrophage transport. In the latter instance, the average life of the macrophage must be less than $1/\lambda \simeq 34$ days in order to explain both the rate of disappearance from the lung and the availability of the Ra in soluble form. This statement applies only if Ra particles released by one macrophage are not phagocytized *de novo*. Lack of complete data on elimination in the Cincinnati cases precludes any statement as to the general success of the previous analysis. We may say only that the similarity in slopes in the retention curve (Fig. 3) of R. and K. to that of the case just described makes it plausible that the elimination curves of these persons might have resembled those shown in Figures 10 and 11. Unfortunately, the few data available, shown by the scattered points in Figure 10 at 1 day, 2 and 6 weeks,[13] are at variance with this deduction and we are at loss to explain it.

We cannot refrain, however, from pointing out that if we assume that the skeletal Ra releases only radon, and this at the rate of 70 per cent of the Rn it produces, then it should be possible to calculate the bone deposition from the data of Table I. If this amount is subtracted from the body burden in each case, we obtain values which range from 1.6 to 3 times the total gamma-ray activity of the lung in cases R. and Ca., but more consistent with the

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[12] On the assumption of a dry fecal sample of 25 gm., instead of 30, it would account for the total excretion observed for the same period.

[13] We are indebted to Dr. D. S. Anthony for the values at 2 and 6 weeks.

SUMARIO

El Transporte del Sulfato de Bario del Pulmón y su Eliminación del Cuerpo Humano a Continuación de Exposiciones Fortuitas Aisladas

A 6 personas que experimentaron exposiciones fortuitas aisladas a polvo de RaSO₄ se las examinó durante el año siguiente en busca de exhalación de radón y de actividad de rayos gamma procedente del tórax y del cuerpo en conjunto. Obtuviéronse mediciones de radio en los excrementos de un sujeto durante unos 250 días. Las observaciones revelan que el patrón de dichas mediciones discrepa radicalmente del de las observaciones ejecutadas en casos humanos de envenenamiento crónico por radio. En particular:

(a) La proporción de Rn exhalado a la carga total de Ra es netamente más baja, sobre todo durante los primeros seis meses.

(b) El patrón revelado por el escrutinio de los rayos gamma muestra fácilmente la localización acentuada del elemento en los pulmones.

(c) Durante el primer año, la actividad de rayos gamma procedente del tórax disminuye con una semivida media del orden de 120 días.

(d) El patrón de eliminación observado en un solo sujeto se caracteriza por eliminación temprana del Ra ingerido y eliminación relativamente elevada en fechas subsiguientes, sostenida presuntamente por desprendimiento de la sal del pulmón.

Por lo tanto, dedúcese que los cálculos de la carga orgánica de Ra, basados exclusivamente en las mediciones ya del radón o de la excreción, son susceptibles de graves errores si se aplican a casos de exposición a polvos insolubles.

lung activity of case G. The latter result merely confirms the assumptions made in the mathematical analysis, but the first two would indicate that a considerable fraction of the total burden is present neither in the skeleton nor in the lung, and is not being easily excreted. Possible sites are the macrophage population or the reticulo-endothelial system of the whole body. Alternative explanations for the data in question could be based also on a relatively low emanating power of radium sulfate in the skeleton and on gross error in the estimates of either total body or lung content.

SUMMARY

Six persons who suffered single accidental exposures to RaSO$_4$ dust were examined during the following year for exhalation of radon, and for gamma-ray activity from the thorax and from the body as a whole. Measurements of radium in the excreta of one individual have been obtained for about 250 days. Our observations reveal that the pattern of these measurements is radically at variance with observations made on human cases of chronic radium poisoning. In particular:

(a) The ratio of exhaled Rn to total Ra burden is distinctly lower, especially during the first six months.

(b) The gamma-ray scanning pattern easily discloses the sharp localization of the element in lung.

(c) During the first year, the gamma-ray activity from the thorax decreases with an average half-life of the order of 120 days.

(d) The elimination pattern found in a single individual is characterized by early elimination of Ra swallowed and a relatively high elimination at later times, presumably sustained by release of the salt from the lung.

We conclude, therefore, that estimates of Ra body burden based solely either on radon or excretion measurements are likely to be seriously in error if applied to cases of recent exposure to insoluble dusts.

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