COMPARISON OF SYSTEMIC PLUTONIUM DEPOSITION ESTIMATES FROM URINALYSIS AND AUTOPSY DATA IN FIVE WHOLE-BODY DONORS

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Abstract—The systemic deposition of $^{239,240}$Pu was determined by postmortem radiochemical analysis of the tissues from five whole-body donors to the United States Transuranium Registry (USTR). All were males with intakes typically occurring many years prior to death. The postmortem radiochemical results were compared with estimates of systemic deposition made with 13 different biokinetic models using urinary excretion data obtained during life. In general, estimates made with older biokinetic models were severalfold greater than those obtained from radiochemical analysis of the tissues. For all five cases, agreement within a factor of two with the tissue analysis results was obtained with two of the biokinetic models evaluated: the Langham power function model as modified by Leggett and Eckerman and the two compartment exponential model proposed in ICRP Publication Nos. 19 and 30.

INTRODUCTION

Since its inception in 1968, the United States Transuranium Registry (USTR) has been collecting and analyzing tissues obtained at autopsy from voluntary donations from persons with a known history of exposure to Pu. To date, five whole-body donations have been received and radiochemically analyzed for $^{239,240}$Pu (McInroy et al. 1989, 1991). As direct postmortem measurements of the distribution and content of Pu in the tissues of donors to the USTR become available, these data can then be compared with estimates made indirectly with the aid of various biokinetic models. This paper compares the systemic deposition of Pu measured by postmortem radiochemical analysis of tissues with estimates made from 13 different biokinetic models based on urinary excretion in five whole-body donors to the USTR. Systemic deposition refers to deposition in the tissues via the bloodstream as opposed to deposition of an insoluble bolus at the site of a wound or via inhalation (Norwood 1972).

Typically, in-vivo estimates of systemic Pu deposition are calculated from the measured Pu content of urine samples, collected either on a routine basis or following a known or suspected acute exposure, with the aid of a specific biokinetic model. This deceptively simple procedure is fraught with great potential for error; some of the more obvious significant sources of error include the inherent accuracy and applicability of the model, the measurement uncertainty for Pu in urine, the representativeness of the urine sample (e.g., 24 h vs. spot), individual variability, the route of entry, physicochemical form, and, perhaps most significantly in the case of chronic exposure, the time after intake used to calculate the fractional excretion value. Except in the case of an acute accidental exposure, the specific time of intake and other important parameters may not be known. The combined effects of these uncertainties may be sufficient to result in errors of an order of magnitude or greater.

MODELS FOR ESTIMATING URINARY EXCRETION

Since its discovery nearly a half century ago, the metabolism and distribution of Pu has been extensively studied in animals and, to a lesser extent, in humans. The first biological studies were made with rats during 1944 and indicated that “the new element was biomedically similar to radium” (Stannard 1988). The early studies identified skeleton as the principal site of deposition, with a residence half-time estimated to be in excess of 6 mo. The data from the animal studies were quickly applied to the occupational health physics need of relating the urinary excretion of Pu to the intake and deposition of Pu. By March 1945, urinalyses were being performed on Manhattan District personnel in an attempt to relate Pu excretion with deposition (Langham and Healy 1973).
Over the years, numerous different biokinetic models for Pu have been put forth in the literature, including those of Langham et al. (1950), Healy (1957), Beach and Dolphin (1964), Durbin (1972), Rundo et al. (1976), Parkinson and Henley (1981), Leggett (1984, 1985), Jones (1985), and Leggett and Eckerman (1987). Most of these are based on the original model proposed by Langham et al. (1950), which was derived from actual human data. Between April 1945 and July 1947, 18 hospitalized persons thought to be terminally ill were injected with known amounts of soluble Pu and excreta collection carried out (Russell and Nickson 1951; Durbin 1972; Stannard 1988). After death, the tissues of some of these patients were analyzed for Pu content. From these experiments, Langham and his coworkers at Los Alamos, in conjunction with the Atomic Energy Project of the University of Rochester School of Medicine and Dentistry, empirically derived a power function excretion model for Pu in humans (Langham et al. 1950). The model was based on urinary excretion observations for the first 138 d postinjection from 12 of the injection cases, plus additional excretion data out to 1750 d (approximately 5 y) obtained from experience with the control of occupational exposure associated with processing large amounts of Pu, and gives the percentage daily urinary excretion of an acute intake of Pu, \( Y(u) \), on day \( t \) after intake as:

\[
Y(u) = 0.2 t^{-0.74}.
\]

The original Langham model has been widely used to predict in-vivo deposition of Pu from urinary excretion data. It has been reevaluated and refined numerous times over the years by various investigators and still serves as the basis for several of the models currently used in operational health physics practice to estimate the systemic burden of Pu.

An early recognized major deficiency of the Langham model is that it is based on excretion of soluble Pu that has reached the blood or transfer compartment and hence does not account for the deposition of a nearly insoluble quantity of Pu in the respiratory tract or at a wound site. Accordingly, Healy (1957) proposed modification of the Langham model to account for an initially insoluble deposit of Pu, based on the assumption that a constant fraction of the Pu in the reservoir was released per unit time. This resulted in the following equation:

\[
Y(E) = 0.2 \lambda Q_0 \int_0^R e^{-A(R - t)^{-0.74}} dt,
\]

in which \( t \) was considered as the time in days since the Pu was absorbed into the bloodstream; \( \lambda \), the rate of solubilization or transfer from the reservoir to the bloodstream; \( A \), the total rate of removal from the reservoir, including solubilization, and in the case of the respiratory tract, mucociliary clearance; \( Q_0 \), the initial amount of Pu deposited in the reservoir; and \( R \), the time in days between the initial intake and the urine sample. The term \( Y(E) \) is not the same as the \( Y(u) \) in eqn (1) and elsewhere in this paper but refers to the fraction of the original deposition of Pu in the reservoir rather than the systemic fraction. Since eqn (2) is not integrable in simple form, graphical means of solution were proposed by Healy and subsequently simplified and improved by Wilson (1967) and Nelson (1969, 1972), who also adapted the model for use with ICRP lung model (Nelson 1967).

The Langham data were also reevaluated by Beach and Dolphin (1964) to distinguish between the initial rapid excretion rate of the injected citrate and the lower excretion rate of metabolized Pu. The resultant two-compartment equation was obtained:

\[
Y(u) = 0.410 e^{-0.67t} + 0.16t^{-0.68}.
\]

This was then combined with the Healy modification to yield a slightly nonintegrable equation for which a graphical solution was provided. As is true of the Healy equation, it was necessary to know or assume a rate of release from the site of entry to the rest of the body. For inhaled Pu, this was accomplished by using the constants provided by the International Commission on Radiological Protection (ICRP) lung model, originally put forth in 1965 (ICRP 1966).

Several investigators, most notably Lawrence (1962) and Snyder (1962), evaluated discrepant results obtained with the Langham model and developed computer programs to minimize these errors or uncertainties but did not produce a new or significantly altered model. Similarly, Robertson and Cohn (1964) reanalyzed the Langham data with the Brookhaven Merlin computer and produced a slightly modified version of the original power function equation:

\[
Y(u) = 0.193t^{-0.721}.
\]

In 1972, more than two decades after the introduction of the original Langham model, Durbin carried out a meticulous, in-depth reexamination he appropriately called "A new look at the old data" (Durbin 1972). In her evaluation, Durbin considered the physiological status of the cases as well as relevant animal data and found that the excretion over a specific time interval was best described by an exponential relationship, deriving a number of excretion constants from which the following five-component exponential model could be constructed:

\[
y(u) = 0.0041e^{-0.078t} + 0.0012e^{-0.126t} + 0.00013e^{-0.0165t} + 0.00005e^{-0.0023t} + 0.000012e^{-0.000174t}.
\]
A serious deficiency of the Langham model was its apparent overestimation of systemic deposition at long times postexposure. This was identified by Hempelmann et al. in their 27-y follow-up of selected Manhattan Project Pu workers (Hempelmann et al. 1973) and partially quantified by Voelz et al. (1976, 1979) who noted that long-term estimates of deposition based on urinalysis tended to be high by a factor of five or more. Confirmation and better quantification came from a follow-up study by Rundo and his coworkers at Argonne National Laboratory of three of the original Pu injection subjects who had survived their illnesses (Rundo et al. 1976). The Rundo team observed that urinary excretion rates at 10,000 d postinjection were approximately an order of magnitude greater than predicted by Langham’s equation and cautioned against using the Langham model to determine systemic deposition at periods beyond 5–10 y postexposure. While the Rundo team did not propose a model per se, they did fit, by least squares analysis, three-compartment equations to the two cases for which adequate data were available. The equations were quite different for the two cases. For one case, the equation was a combination of a power function, exponential, and linear terms, which the authors noted was not meant to be predictive. For the other case, the following three-component exponential equation was obtained:

\[
Y(u) = 0.341e^{-0.319u} + 0.0578e^{-0.0832u} + 0.00147e^{-0.0000829u}. \tag{6}
\]

It is a tantalizing and clearly implied simplification to take quite literally the statement of Rundo et al. (1976), reiterated a few years later (Rundo 1981) in a further analysis of the data, to the effect that urinary estimates at 10,000 d postintake are approximately 10-fold greater than predicted by either the Langham or Durbin models, and one should simply adjust estimates of deposition at long times after intake made with either model downward by a factor of 10 and compare these results with data from postmortem radiocchemical analysis of tissues.

The question of excretion of Pu at long times after intake was squarely addressed by Parkinson and Henley (1981) who proposed the following four-component exponential model:

\[
Y(u) = 0.19e^{-0.272u} + 0.023e^{-0.0237u} + 0.0052e^{-0.00303u} + 0.000086e^{-0.000019u}, \tag{7}
\]

noting that at 10,000 d postintake, the estimated urinary excretion would be fourfold greater than that calculated from the Langham model, which would result in a fourfold reduction in the estimated systemic deposition. Although no direct reference to human excretion data was made, Parkinson and Henley indicated that their model was developed from excretion data from a few exposed individuals but with some emphasis placed on Case HP-6, an injection case that was also used by Durbin (1972) and Rundo et al. (1976) in their analyses. They attempted to achieve a better fit of the data with the then current ICRP model (1972).

The problem of the validity of previous urinary excretion models at long times after intake was rigorously evaluated by Jones (1985), who incorporated the excretion data collected by Rundo et al. (1976) at 10,000 d postinjection to obtain a four-component exponential model for urinary excretion (eqn 8) with the same general form, albeit different coefficients, as that of Parkinson and Henley:

\[
Y(u) = 0.475e^{-0.558u} + 0.0239e^{-0.442u} + 0.00855e^{-0.00380u} + 0.0000142e^{-0.0000284u}. \tag{8}
\]

In a note added in proof, Jones cautioned that reconsideration of the additional data coupled with some newly available urinary excretion data at times 300–400 d postintake indicated that the model given in eqn (8) would underestimate urinary excretion at 1,000–10,000 d postintake by 20–30% and correspondingly overstate uptake by the same fraction.

Recently, Leggett (1984, 1985), in contrast to simply fitting equations to existing data, derived a mechanistic model for the retention and excretion of systemic Pu that was applicable to exposures at any adult age. He characterized the fractional retention \( R(t) \) as opposed to the percentage daily excretion, \( Y(u) \) after injection into the bloodstream, in terms of the following explicit four-component exponential:

\[
R(t) = 0.012e^{-0.693t} + 0.02e^{-0.0288t} + 0.042e^{-0.0028t} + 0.926e^{-0.0000216t}. \tag{9}
\]

Since eqn (9) includes both urinary and fecal excretion, it cannot be readily converted to \( Y(u) \) unless the relative amounts excreted via the feces and urine are known. Once a suitable estimate of the urine to fecal excretion ratio has been selected, eqn (9) can be appropriately adjusted to consider only urinary excretion. Leggett, in conjunction with Eckerman (Leggett and Eckerman 1987), subsequently tabulated the urinary excretion for what he termed a base case, viz. namely 45 y of age at time of injection. Leggett and Eckerman also reexamined the original Langham equation in the light of autopsy and other data that had been acquired subsequently and modified the original equation by the addition of a time-dependent term to correct for the overestimation of the deposition at long times after intake as shown in the following equation:

\[
Y(u) = 0.2(1 + 0.0008t)^{-0.74}. \tag{10}
\]

The ICRP has twice published comprehensive examinations of the metabolism of Pu recommending certain parameters that can be used to develop an excretion equation for Pu. In the first of these, originally put forth in 1972 and reiterated in 1979 (ICRP 1972, 1979), the initial fractionation of Pu is taken as 45% to liver with a residence half-time of 40 y, 45% to skeleton with a residence half-time of 100 y, and 10% to early excretion and
other tissues. Ignoring the small early excretion component, a two-compartment exponential retention function for the percentage of systemic Pu remaining at time \( t \) can be derived from the ICRP parameters:

\[
R(t) = 45e^{-0.000047t} + 45e^{-0.000091t}.
\]  

Equation (11) includes both fecal and urinary excretion, and hence some estimate of urinary excretion needs to be made in order to calculate a urinary excretion function. Voelz et al. (1979), in their 32-y follow-up of 26 Pu exposure cases, observed that while fecal excretion at 31-32 y postexposure was variable, fecal excretion averaged about 30-40% of the urinary excretion. A similar observation was made in two of the early injection cases at 10,000 d (27.4 y) postexposure (Rando et al. 1976). These data suggest that at long times postexposure, the urinary to fecal ratio is about 2. Durbin (1972), in her analysis of human data, indicated that at times greater than 100 d postexposure, the urine to fecal excretion ratio is 1.0 to 1.5. At earlier times, Durbin indicates the ratio is closer to unity and perhaps even less than unity very soon after intake. Assuming the ratio of urinary to fecal excretion is more or less constant with time and equal to 1.5, \( Y(u) \) can be obtained by adjusting eqn (11) to consider only urinary excretion and differentiating with respect to \( t \), to yield:

\[
Y(t) = \frac{d[R(t)\%]}{dt} = 0.0013e^{-0.000047t} + 0.00051e^{-0.000091t}.
\]  

A similar approach can be taken with the later ICRP report, which gave an initial fractionation of 50 and 30% and effective half-times of 50 and 20 y, respectively, for the skeleton and liver. The remaining 20% is vaguely described as "...excreted, or deposited in a variety of other organs and tissues" (ICRP 1986). Ignoring this remaining 20% yields the following equation:

\[
Y(t) = 0.0029e^{-0.000091t} + 0.0019e^{-0.000018t}.
\]

CASE REPORTS

Five USTR whole-body donors with a history of occupational exposure to Pu are included in this study; these cases have been briefly described elsewhere along with the initial results of the postmortem tissue radionuclide analyses for Pu (McInroy et al. 1989). Relevant information about each case is abstracted below.

**USTR Case 193.** The medical and work history of this case has been described in detail by McInroy et al. (1989, 1991) and has been identified as Case 2 in a previous study of the distribution of Pu in bone (Kathren et al. 1987). This individual was a 62-y-old Caucasian male, 1.73 m tall and 75 kg in weight, who retired in January 1982, about 1 y before his death from respiratory failure consequent to pneumonia in December 1982. He had been employed as a chemical engineer in Pu alloy research and other Pu work from 1945 until his retirement in 1982.

There was potential for moderate inhalation exposure to Pu from 1945-1957; no wounds or ingestions were recorded, but positive nasal swipes with >1 Bq of activity were obtained from this individual on 16 occasions before 1957. Exposure potential was probably significantly reduced in 1957 when he assumed supervisory responsibilities.

From 1945 through 1982, data from 177 urinalyses for Pu were available. About 50 of these were made prior to 1957, when the techniques for quantitating Pu in urine were not well refined and hence are of questionable validity. Six urinalyses were performed in the year prior to his retirement and averaged 2.6 ± 0.8 mBq 24 h⁻¹. Review of the overall urinalysis results suggests that exposure occurred during two time periods, with about 80% of his exposure incurred during 1945-1947 and the remainder from about 1965-1970.

**USTR Case 208.** This male Caucasian, age 69 y at time of death in April 1984 from a pulmonary embolus, was 1.78 m tall and weighed approximately 59 kg. He suffered a cerebral vascular accident (stroke) 8 y prior to his death and was physically inactive and retired from active employment after that event. He began work in 1946 as an operations foreman in a Pu reduction and dry chemistry operation. He incurred the potential for acute inhalation exposure to Pu during the period from May 1946 through March 1947, showing on one occasion (August 1946) positive nose wipes for Pu. A total of 92 Pu urinalyses were performed on this case, four during 1946 when his potential for exposure was greatest. The first of these yielded a negative result, while the other three showed increasing concentration, typically of a few mBq 24 h⁻¹. Urinalysis was discontinued for approximately 9 y until October 1955 and when restarted indicated levels of a few mBq 24 h⁻¹ through the time of his retirement in 1977. Overall, the urinalysis results are indicative of an exposure in the early years of employment, with perhaps some additional exposure about 1961. Urinalysis was discontinued with his retirement in early 1977 until 1982, when two analyses were made, with a single analysis performed in 1983, the year prior to death. These samples were elevated, averaging about 10 mBq 24 h⁻¹ that may have been an artifact associated with increased excretion attributable to wasting osteoporosis.

**USTR Case 212.** This 56-y-old male Caucasian plumber, measuring 1.82 m and weighing 89 kg, died from acute left pneumothorax and associated complications of emphysema in 1984. During his employment, he was involved in two radiation incidents with the potential for internal deposition of Pu. The first occurred in August 1956 as a result of skin contamination and resulted in a relatively small deposition estimated by the operational health physics staff to be approximately 0.8 Bq of 239Pu. In March 1967, he suffered a contaminated wound to a finger that was treated by excision and chelation with DTPA. A total of 26.5 g of DTPA was administered in 47 treatments during the approximately 6-mo period following the injury. The initial deposition at the wound site...
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was determined by external counting and estimated as 22 kBq; this was reduced to an estimated 40 Bq following surgical excision and at the start of chelation therapy.

**USTR Case 213.** This donor was a 68-y-old male Caucasian measuring 1.72 m tall and weighing 64 kg at the time of his death from lung carcinoma in 1984. He began working with Pu in July 1946 as a chemist and had potential airborne Pu exposures until his retirement in January 1979. Five potential inhalation accident events were recorded between 1946 and 1967, including three occasions in 1946 and 1947 when positive nasal wipes for Pu were obtained. Seven in-vivo chest measurements were made between 1970 and 1979. All were below the minimum detection levels for 239Pu.

Nearly 200 urinalyses were performed for 239+240Pu during his employment. As is true for Case 193, those performed prior to 1957—about 40% of the total—are of questionable validity. Later results are quite good, however, and indicated chronic low-level exposure, with possible additional exposure during 1959, 1967-1968, and 1976. The last urinalysis was performed approximately 2 y prior to his death and measured 4.4 mBq 24 h⁻¹.

**USTR Case 242.** This 78-y-old Caucasian male was 1.80 m tall and weighed 85 kg at time of death in January 1987 of left ventricular hypertrophy and atherosclerosis. He began working with Pu in 1946 as a Pu chemical operator and had potential exposures to airborne Pu until his retirement in June 1972. Six accidents with potential for involvement were recorded during 1947-1948. All were exposures to airborne Pu except one, an acid burn in 1947 having a recorded surface contamination level of >40,000 cpm (counting efficiency unstated). One additional exposure to airborne Pu was recorded in 1958.

More than 200 urinalyses for 239+240Pu were performed over the course of his employment. Approximately half were performed prior to 1957 and, thus, are of questionable validity. The bioassay and work history data are suggestive of long-term chronic low-level exposure, with more than half of his exposure incurred in the first few years of employment. His final urinalysis was performed in 1975, some 3 y after employment ended and more than 10 y prior to death, and this measurement indicated a level of 11.5 mBq 24 h⁻¹.

**ESTIMATING SYSTEMIC DEPOSITION FROM URINARY EXCRETION MODELS**

The models discussed above can be used to estimate systemic deposition by simply dividing $Y(u)$, expressed as a fraction rather than a percent, into the Pu activity excreted on day $t$ after intake. Mathematically, this can be expressed by:

$$q_t = \frac{100A}{Y(u)} \cdot \int \frac{100A}{Y(u)} \cdot du$$

in the following example in which the systemic deposition is calculated for an individual at $t$ days after an intake of Pu.

The above technique, while conceptually simple, is deceptively so, for in practice, an individual may incur multiple exposures or be chronically exposed to unknown and variable levels. Moreover, the specific day of the exposure (i.e., intake), as well as its magnitude, is uncertain, and diurnal variation in urinary excretion of the individual can also affect the estimate. To minimize these potential sources of error, urinalysis data collected over a period of time is used, and they are sometimes subjected to analysis by sophisticated computer programs such as PUQFUA (Lawrence 1962, 1978; Snyder 1962), which perform multiple iterative and smoothing functions for a particular model. In this study, the calculations for each of the models listed in Table I and described above were performed manually, much in the manner that they would be done by an operational health physicist in the field, with com-

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puter verification of the hand calculations in a few select cases.

The basic technique was to plot urinary excretion ofPu as a function of time, fitting the individual data points to a smoothed curve by eye. A step increase in urinary excretion was considered to be a presumption of a fresh acute systemic deposition, or, in other words, an increment.

Occupational and exposure history data were used to verify that an acute exposure at that time was in fact reasonable. Since the urinary excretion of Pu at approximately the time of death was known for all cases, it was relatively easy to calculate the relative contribution of each of the two or three step-function increases typically observed in the urinalysis data to the urinary excretion ofPu at the time of death using the various models. Simplifying assumptions regarding the specific time and relative magnitude of exposure were made based on the available exposure and bioassay data for each case; for any given case the same assumptions were used with all models. For example, if examination of the available bioassay and other information on a case suggested a chronic low-level exposure or intake over a period of a few years, this complex intake situation was simplified by assuming that the chronic intake could be approximated by a single intake of equal magnitude occurring at approximately the midpoint in time of the chronic intake. Estimates made in this fashion compared very well with the more rigorous summation techniques for the chronic intake situation, which were carried out with the aid of a computer in a few select cases.

With the exception of the Healy modification to the Langham model, continuous release of activity to the blood from a deposition in the lung of Class Y material, or from activity at a wound site, was not assumed. In the one case (Case 212) in which chelation therapy was used following a percutaneous exposure, the period of enhanced urinary excretion ofPu was determined by eye from examination of the excretion curve. Urinalysis results during this period were not used.

### RESULTS AND DISCUSSION

Estimates of initial deposition of Pu using eqn (14) have been made for each case using the 13 biokinetic models for Pu described above, and these are given in Table 1. In Table 2, the estimated systemic deposition at the time of death, calculated with eqn (15), is presented for the various models except for the two with the continual release compartment. These can be compared with the postmortem measurement results, which are also given in Table 2. The two models with continual release compartments—i.e., the Healy model and that of Beach and Dolphin—do not permit calculation of systemic deposition per se but rather estimate the ultimate systemic uptake from a reservoir slowly releasing Pu from the site of initial deposition to the blood. For this reason, they are not included in Table 2.

In four cases, exposure was primarily via inhalation; in the other case (212), a contaminated wound was the primary source of exposure. Since in all cases the majority of the intake took place many years (typically two to three decades) prior to death, the deposition estimates with the various models at the time of death (Table 2) are for long times postexposure and accordingly are smaller than the estimates of initial deposition made with the same models (Table 1). In general, differences between the initial systemic deposition estimates [represented by $q_0$ in eqn (14)] and the estimated deposition at the time of death [represented by $q_0$ in eqn (15)] do not differ appreciably for the earlier biokinetic models such as those of Langham et al. (1950) but do differ significantly with the later models. In other words, the later models predict greater excretion, which is, of course, as they were intended to do. This is not surprising in view of recent reports by Moss and Gautier (1985) and Moss and Tietjen (1989), who reexamined the raw data on which the Langham model was based and observed that errors had been made that resulted in underestimation of the excretion at long times after intake, which would, of course, lead to overestimation of predictions of deposition based on the Langham model.

### Table 1. Estimated initial systemic deposition by various biokinetic models.

<table>
<thead>
<tr>
<th>Model/reference</th>
<th>Estimated initial systemic deposition (Bq)</th>
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<tr>
<td></td>
<td>Case 193</td>
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<tr>
<td>Langham et al. (1950)</td>
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<tr>
<td>Healy (1957)</td>
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<tr>
<td>Beach and Dolphin (1964)</td>
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<td>Robertson and Cohn (1964)</td>
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<td>Durbin (1972)</td>
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<td>Rundo et al. (1976)</td>
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<tr>
<td>Parkinson and Henley (1981)</td>
<td>330</td>
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<td>Leggett (1984, 1985)</td>
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<td>Jones (1985)</td>
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<td>100</td>
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* Model not applicable.
The real test is the comparison of predictions of systemic deposition at the time of death made by the various biokinetic models with the actual activity measured by postmortem radiochemical analysis of the tissues (Table 2). In general, the estimates made with the older models were severalfold greater than the tissue measurement values. Significantly better agreement with the postmortem results was obtained with more recent models; several models produced estimates of the systemic deposition within a factor of 2–3 of the radioassay results for all five cases, and two—the Leggett-Eckerman modification of the Langham model and the earlier ICRP two-component exponential model originally proposed in 1972 and reevaluated in ICRP Publication No. 30 (1979)—were within a factor of 2 in all cases.

It should be noted that the predictive ability of all the biokinetic models is quite sensitive to the accuracy of the urinary excretion data and to accurate knowledge of the specific time(s) and relative magnitude(s) of exposure.

It is well known that urinary excretion data are highly variable, and in particular, earlier results need to be considered carefully in view of the capabilities prevailing at the time they were obtained. Campbell and his coworkers (1972) reported marked differences in the sensitivity and reliability of radioisotope analyses performed at Los Alamos in the early years when compared with those performed more recently. Thus, the factor of two agreement of the Langham-Leggett-Eckerman and ICRP 19/30 models with the postmortem data should probably be considered excellent. Either model would be suitable for operational health physics purposes.

REFERENCES


Langham, W. H.; Healy, J. W. Maximum permissible body burden...


