IN VIVO MEASUREMENTS FOLLOWING EXPOSURE TO ^{133}XE

AND ASSOCIATED DOSE ASSESSMENT PROCEDURES

Frances Fry
Atomic Energy Research Establishment Harwell
Graham Tyler
Experimental Reactor Establishment, Dounreay

ABSTRACT

Information is presented on the retention and distribution of ^{133}Xe in the human body as determined by in-vivo counting. Calculations by other workers have shown that for exposure to ^{133}Xe gas the critical dose is that to the skin. It is shown here, with reference to three cases, how measurements of the body content of ^{133}Xe made by in-vivo counting can be used to estimate skin doses.

By reference to actual recent cases attention is drawn to problems caused by ^{133}Xe intakes in the interpretation of external contamination and plutonium-in-lung measurements.

INTRODUCTION

^{133}Xe is produced in the fission of ^{235}U with a total yield (direct and by chain) of about 7%. It decays with a half life of 5.27 days to stable ^{133}Cs by the emission of beta particles of maximum energy 0.34 MeV, gamma rays of energy 81 KeV are emitted in 35.5% of the disintegrations and caesium K X-rays of 30 KeV energy are emitted following 56% of disintegrations. Xenon, a noble gas, which is present in the atmosphere with a partial pressure of about .8 mN/m^2, is in general chemically inert but appears to combine specifically with haemoglobin (1).

^{133}Xe has been used extensively in medical science for the investigation of lung function; information on the procedures and computations of the retention of ^{133}Xe when used for this purpose have been presented by Matthews et al (2). Measurements of the distribution and retention of ^{133}Xe in the body following both experimental and accidental inhalation in a laboratory manufacturing ^{133}Xe for medical application have been reported by Venner and Devell (3). Guillot (4) has reported on retention experiments with ^{133}Xe, ^{131m}Xe, ^{125}Xe and also stable xenon isotopes. The International Commission on Radiological Protection (5) state that the primary hazard from ^{133}Xe is from external radiation from submersion in a cloud and calculations of internal doses arising from inhalation of radioactive noble gases presented by Whitton (6) confirm this.

Because it is a gas it is possible for ^{133}Xe to leak from nuclear reactor fuel elements into operating areas. Whilst the reactor is operating any ^{133}Xe will usually be accompanied by other fission product noble gases notably ^{88}Kr ($t_{\frac{1}{2}}$ 2.8h) whose daughter product ^{88}Rb is a solid emitting energetic beta particles; the leak will therefore usually be rapidly detected by conventional filter paper air samplers. Because of its longer half life however ^{133}Xe may be released a day or so following reactor shut down virtually without the ^{88}Kr. If the release is a slow one which leads to more or less uniform contamination of the air in the reactor containment conventional external radiation monitor-

ing will adequately detect and assess the hazard. Sometimes however the release may be very localised and although when the activity is dispersed throughout the operating area the resultant radiation level is very low, significant doses may be received by individuals in the immediate vicinity of the release as we will show later.

THE UPTAKE AND RETENTION OF ^{133}XE IN THE HUMAN BODY FOLLOWING INHALATION

As stated by Matthews et al (2) the uptake of ^{133}Xe into the body is a function of its solubility in blood. However this apparently straightforward situation is complicated by the presence in the atmosphere of naturally occurring stable xenon. The solubility of a gas in a liquid is a function of the partial pressure of the gas and since the normal atmosphere contains xenon at a partial pressure of about 8 mN/m^2 the body will be saturated with xenon at this partial pressure.

We have made measurements of the retention of ^{133}Xe in persons who have been exposed to ^{133}Xe in a reactor environment. Some had been exposed to low level uniform concentrations during reactor operating periods and some to small localised clouds of high concentration following reactor shut down. Measurements made from 1 to a few hours following cessation of exposure to low level uniform concentrations showed the half life of ^{133}Xe excretion to be about 2 hours. Measurements on persons exposed to small high concentration clouds were made over a longer period up to about 80 hours after exposure and showed retention curves very similar to those reported by Venner and Devell (3), viz an initial rapid excretion phase lasting about 4 hours during which the half life is less than 1 hour followed by a slower elimination rate with half life of about 6 hours for the remainder of the period.

DISTRIBUTION OF ^{133}XE IN THE BODY FOLLOWING INHALATION

We have made measurements of the distribution of xenon-133 in the body (1) after exposure for several hours to low-level contamination and (2) after exposure to a small cloud of high concentration. Profile curves, obtained one day after exposure, are shown in figure 1. Some differences between the curves may be attributed to differences in the scanning techniques. Curve (1) was obtained by scanning with a collimated detector above the supine subject whereas a more finely collimated detector, located under the body, was used for scan (2). The 'depressions' in counting-rate in scan (2) could be due to absorption of the 80 KeV gamma-rays in bone. The 'depressions' in the counting-rate from the chest could be due to attenuation in the ribs and the decrease in counting rate at about 110 cm could be attributed to absorption in the pelvis. These effects would not be so marked with the wider-angle collimator particularly as it was used above the subject. The maxima of the distributions occur at 60-80 cm from the top of the head and could indicate accumulation of xenon-133 in liver. Neither plot shows the large depression in the chest region noted by Venner and Devell (3) in a scan made some hours after an accident inhalation.

CALCULATION OF SKIN DOSE FROM IN-VIVO MEASUREMENTS

The critical tissue dose from exposure to ^{133}Xe is that to the skin. If however the ^{133}Xe is released in the form of a small cloud normal external radiation monitoring devices will often not give a correct indication. Under those circumstances however it is possible to calculate a skin dose from

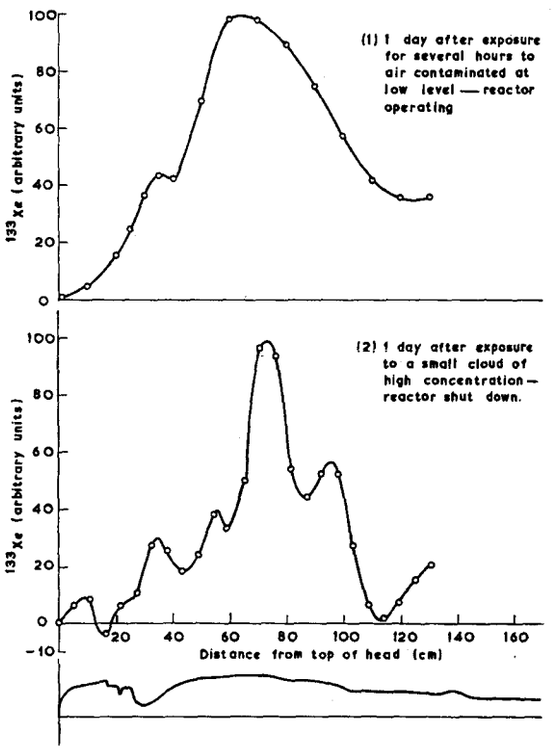

FIG 1 DISTRIBUTION OF ^{133}Xe IN TWO SUBJECTS

in-vivo measurements. Although the calculation will be subject to considerable error it should be sufficiently accurate to decide whether or not an over exposure had occurred. This is illustrated by the following cases.

Case 1

A technician L, removing a thermocouple from a shut down reactor, was working at a glove box containing a mixture of reactor blanket gas and purging argon at a pressure just above atmospheric when a glove was snagged and torn releasing some of the gas mixture. In vivo measurements were carried out on the technician and his assistant who was standing about 2m away at the time of the incident. These both showed ^{133}Xe but the intake by the assistant was only 1% of that of the technician, confirming that only a small cloud of gas had been involved. In vivo measurements were made on the technician over the period 4-75 hrs after the exposure showed a retention pattern similar to that obtained by Venner and Devell (3) after controlled inhalations; for the period 4-10 hrs the fall was more rapid by about a factor of 2. By extrapolation and interpolation of the retention curve we estimated a body content of about 9 mCi of ^{133}Xe at 1 hr and 300 μCi at 10 hours after exposure. The Venner and Devell plot (3) shows that for controlled inhalations the retained amounts of the inhaled activity at those times are 4% and 0.5% respectively. Applying these factors to our estimated body content we obtained estimates of 200 mCi and 60 mCi respectively for the initial inhaled amount. In view of the more rapid fall from 4 hours to 10 hours in our case we chose the higher figure as being a more correct estimate (perhaps even a little on the low side). If the activity was breathed in during some unknown but short period of time, t hours (it is not necessary to know this time), whilst the subject was breathing at the standard man rate of 1 m^3 per hour, the activity concentration in the cloud would have been 200/t mCi/m^3. On the assumption that the radius

of the small cloud of gas was about equal to the range of the beta particles (80 cm) conventional calculation showed the beta dose rate to a plane in the centre of the cloud to be 20/t rem/hr.

The gamma dose rate from a cloud of this size would be several orders of magnitude lower and was ignored. When we eliminate our unknown exposure time (t) we obtain a skin dose of 20 rems. We were therefore able to say that, despite a film badge recorded dose of only 0.5 rem gamma and 0.4 rem beta, the technician probably received a dose to the skin of his face of more than the 13 week permitted dose (15 rem) but less than 1 year's permitted dose (30 rem) and appropriate administrative action was taken. The most significant internal organ dose from this inhaled quantity was calculated from the information given by Whitton (6) as 1.3 rem to the tracheal mucosa which is much less significant than the skin dose.

Case 2

Several men became internally contaminated with mixed fission products during removal of a fuel element two days after reactor shut-down.

The most highly contaminated man, subject G, had a body content of 12.8 μCi of ^{133}Xe $21\frac{1}{2}$ hours after the release. Measurements made during the following three days indicated that ^{133}Xe was being removed from the body with an effective half-life of 8.7 hours (biological half-life 9.3 hours): this is in agreement with Venner and Devell's retention curve at this time after inhalation (3).

By extrapolation of the retention curve we estimated a body content at 10 hours of 32 μCi ^{133}Xe. According to Venner and Devell's data (3), retention of ^{133}Xe at 10 hours is 0.5% and we therefore estimated the initial body burden as 6.4 mCi. It is probable that most of the intake occurred in a short period of time since the measured general air levels were high (>3 nCi/ml) for about an hour and then dropped to 0.6 nCi/ml and it is also probable that a cloud of much higher concentration existed close to the source for a shorter time. As before we assumed that subject G's initial body burden was acquired in a short period while breathing at 1 m^3/hr, then calculation of the β-dose rate at the centre of a cloud of gas, as above, showed that subject G may have received a skin dose of 0.6 rem. This is less than the 13-week permitted dose, but greater than the dose to any internal organ. The most significant internal dose was 40 mrem to the tracheal mucosa and the corresponding lung dose was 8 mrem (6).

Case 3

^{133}Xe was also identified in subject M, $2\frac{1}{2}$ hours after a release of mixed fission products which occurred during removal of a rig from an operating reactor. The total body content of ^{133}Xe at this time was 0.5 μCi. Several measurements made during the next few hours indicated that xenon was being removed from the body with a biological half-life of 2 hours. However, a further measurement made three days later suggested that there was some long-term retention greater than that predicted by the Venner and Devell retention curve. The faster clearance may correspond to removal of xenon from the water-containing tissues and the longer-term clearance may represent elimination from the less well-perfused fatty regions of the body.

The initial intake was estimated by extrapolating the measured value at $2\frac{1}{2}$ hours to 1 hour after intake, with the observed half-life of 2 hours, and

then applying Venner and Devell's figure of 4% retnetion at one hour. This suggests that subject M's initial intake was 20 μCi. The β-dose to the skin of the face was estimated to be 2 mrem and internal doses to body organs were negligible.

INTERFERENCE FROM INTERNALLY INCORPORATED ^{133}XE WITH HEALTH PHYSICS MEASUREMENTS

Internally incorporated ^{133}Xe gives rise to relatively small internal dose commitments as indicated in Case 1 above where the highest internal organ dose from an initial intake of 200 mCi was calculated as 1.3 rem. However incorporated ^{133}Xe in much smaller amounts can lead to misleading results being obtained from health physics measurements as is illustrated by the following two occurrences.

i. The technician referred to in Case 1 above monitored his body immediately after the incident and believed himself to be highly contaminated (about 200 x dwl). Several unsuccessful attempts at decontamination by showering were made before a health physicist was consulted who suggested that internally incorporated ^{133}Xe was the most likely cause; gamma spectrometry of a blood sample confirmed this. Later concurrent in-vivo counting and monitoring with a contamination probe showed that an internal content of 300 μCi gave rise to a counting rate at the surface of the body (using a thin walled, 30 mg/cm^2, Geiger Muller tube of dimensions 14 cm long and 1.5 cm diameter) of 10 cps equivalent to approximately 2 dwl of skin contamination.

ii. A laboratory worker who normally worked in a laboratory handling ^{239}Pu went into a reactor operating area to view an experiment on his way to keep an appointment for a ^{239}Pu-in-lung measurement. This measurement was made

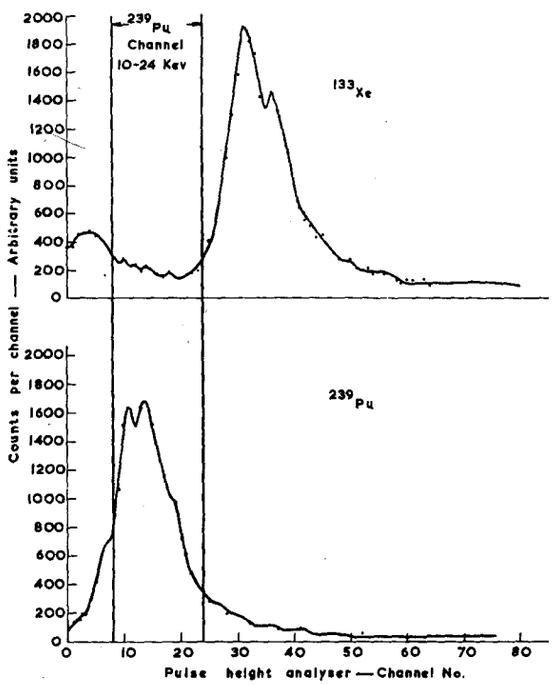

FIG. 2. PROPORTIONAL COUNTER X-RAY SPECTRA.

using a gas filled proportional counter of the type described by Taylor (7). The initial result, which was assessed by the total counting rate in the 17 KeV ^{239}Pu channel, caused some concern as it was equivalent to more than 100 times the maximum permissible lung burden. Examination of the spectrum from the counter however showed peaks at about 30 KeV and 5 KeV and ^{133}Xe was suspected.

A further count using a scintillation detector indicated a body content of about 0.6 μCi of ^{133}Xe and a repeat measurement with the proportional counter a few days later showed no activity in the plutonium channel. A plot of the spectrum from ^{133}Xe in the body as given by the proportional counter together with a spectrum of ^{239}Pu for comparison is shown in Figure 2.

CONCLUSIONS

In-vivo measurements made on men exposed to air contaminated with xenon-133 in nuclear reactor environments show retention patterns similar to those reported (2). Elimination of xenon-133 is a complicated function of time, indicating that many body compartments are involved in the uptake and retention. Uptake, retention and also distribution within the body may vary depending upon the partial pressure of the inhaled xenon. Profile scanning measurements suggest that the distribution within the body is different for the two modes of uptake discussed. The reasons for this are not readily apparent, but some of the differences between the two profile curves may be due to different scanning techniques.

The critical dose from exposure to ^{133}Xe is that to the skin but, as we have shown, the results of in vivo measurements of the body content and elimination rates may be used to calculate this dose.

^{133}Xe incorporated in the body in amounts which give rise to trivial doses of radiation can interfere with health physics measurements and health physicists for reactor areas should be aware of the possibilities.

REFERENCES

1. Schoenborn, B P, Bindong of xenon to horse haemoglobin. Nature 208, pp 760-763 (1965).

2. Matthews, C M E, Dollery, C T, Clark J C and West, J B, USAEC Symposium 6 Radioactive Pharmaceuticals, p567 (1966) Conf 65111.

3. Venner, L and Devell, L, Retention of ^{133}Xe after inhalation. Nordic Radiation Protection Conf. Oslo 2-4 October 1968.

4. Guillot, P. Nonlinear relationship between radio xenon inhaled activity and whole-body activity; radio xenon retention differs from stable xenon retention in man and rat. Presented at 2nd European Congress of IRPA Budapest, 3-5 May 1972.

5. Recommendations of the International Commission on Radiological Protection. ICRP Publication 2. Pergamon Press (1959).

6. Whitton, J T (Mrs), Doses arising from inhalation of noble gases. CEGB RD/B/N1274 (Dec 1968).

7. Taylor, B T, Health Physics 1969, Vol 17, pp 59-69.