THE INVESTIGATION AND RECONSTRUCTION OF A SEVERE RADIATION INJURY TO AN INDUSTRIAL RADIOGRAPHER IN SCOTLAND

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Abstract

This paper describes a severe radiation injury received by a young industrial radiographer concerned with pipeline radiography. The reconstructions carried out to explain the incident are discussed together with the calculations made to predict the radiation doses at the site of the injury and the effective whole body dose. These results are compared to the clinical estimates of the radiation dose profiles at the injury and the estimates of radiation dose made by reference to the film badge worn by the worker and to the chromosome aberrations detected in the lymphocytes obtained in a blood sample.

The injury was observed on the upper left area of chest wall adjacent to the left nipple, involving a circular area of skin about 100 mm diameter extending in depth 30 to 40 mm to ribs and even heart muscle.

The worker associated this injury with an exposure to an open industrial radiography source housing containing a 25 curie iridium-192 source during a short car journey with the container open on the adjacent front seat and directed towards him. Evidence is presented to discount totally this explanation and the possible alternatives derived from consideration of the clinical estimates of the radiation dose profile are discussed in detail. The radiation doses at the site of the injury were thought to be up to about 20,000 rads. A brief statement is made of the medical treatment given.

Introduction

The purpose of presenting this paper is to report one of the most severe accidents involving exposure of an industrial worker to radiation which has occurred in Great Britain. The paper also brings to light the difficulties in attempting to explain an incident in retrospect when most of the information comes from the person concerned who cannot, or may not wish to, recall the exact circumstances.

Description of Incident

The young man concerned, aged 20 years, commenced employment on 25th August, 1969 as a trainee radiographer with a large company engaged in non-destructive testing. His initial training was carried out informally in discussion with other more senior radiographers over the first two weeks. Towards the end of the first week he carried out radiography and from the second week onwards he sometimes worked without direct supervision.

On 20th September he was provided with an iridium-192 source, strength 25 curies, contained in an industrial protective source housing, to radiograph a 24 inch gas pipeline. The source housing, figure 1, had been designed by the company themselves to the shielding requirements specified in British Standard, BS.4097, 1966. Provision was made for locking the source in the closed position but on this occasion no padlock was provided.

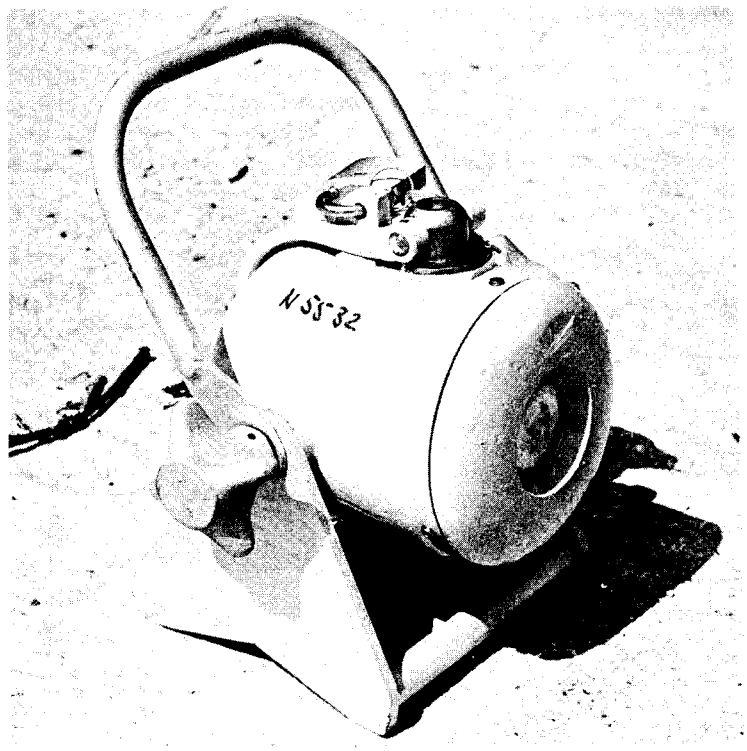

Figure 1. Protective Source Housing used for Industrial Radiography

He was the last radiographer to complete his work and was therefore left entirely on his own at the site. On finishing he loaded the exposed films onto the back seat of his car and placed the source housing on the front passenger seat.

At the completion of his journey a fellow worker noticed that the source housing was in the open position with the radiation beam directed towards the driving seat. It was estimated that he was exposed to the radiation in this situation for three hours. During this time his radiation monitoring film, not in a film holder, was in a wallet in his right hand hip pocket.

He did not work again with radioactive sources from this time until the onset of clinical symptoms.

Clinical Considerations

The clinical symptoms became apparent on 2nd October, 1969. He noticed for the first time a small white patch on his chest wall just below the left nipple. This patch was surrounded by a circular area 15 mm in diameter which gradually became inflamed. On 5th October he began to feel unwell. The chest area had started to swell, the reddening had extended to an area 100 mm in diameter, the central white patch had spread in size and a blister formed around its edge.

At this stage he sought medical advice. He approached his local general practitioner who, in view of his occupation, referred him to a consultant radiotherapist at the Institute of Radiotherapeutics, Western Infirmary, Glasgow.

By 8th October the injury had assumed an oval shape, bright fiery red in colour, about 100 mm in diameter, with a central spot, deeper and darker red in colour, 20-30 mm in diameter.

Other lesser injuries were also noted. There was a smaller, similar lesion about 20 mm in diameter over the sternum, a small lesion on the inside of the left wrist and blistering of the fingertips of the left hand. During the following week the central parts of both chest lesions vesicated and became ulcerated. Over the next few weeks the erythema decreased around the main injury, but the central ulcer extended to cover an oval area 100 mm x 90 mm developing a deep black scab in the centre, figure 2.

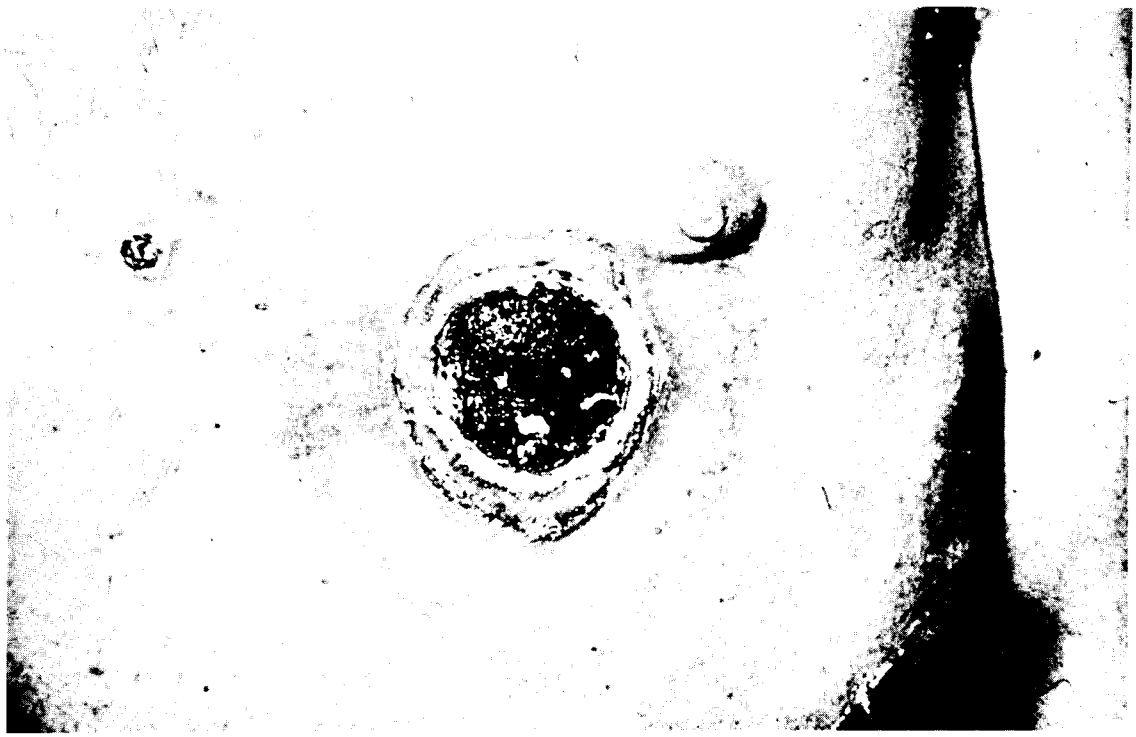

Figure 2. Appearance of main chest injury - December 1969.

The small sternal lesion scabbed and healed completely by the end of January 1970. The deep scab continued to develop over the main lesion however without any healing. He was admitted to Canniesburn Hospital, Glasgow in April 1970 with a view to surgery.

The slough was excised from the lesion revealing an area of necrotic tissue about 50 mm in diameter, which it was also decided to remove. During this

operation, the radionecrosis was found to involve parts of the fourth and fifth ribs. Damage had also occurred to the pericardium and an area of radionecrosis 10 mm in diameter was observed on the heart muscle. The surgery was completed with a thoraco-abdominal flap. The appearance on discharge from Canniesburn Hospital is shown in figure 3.

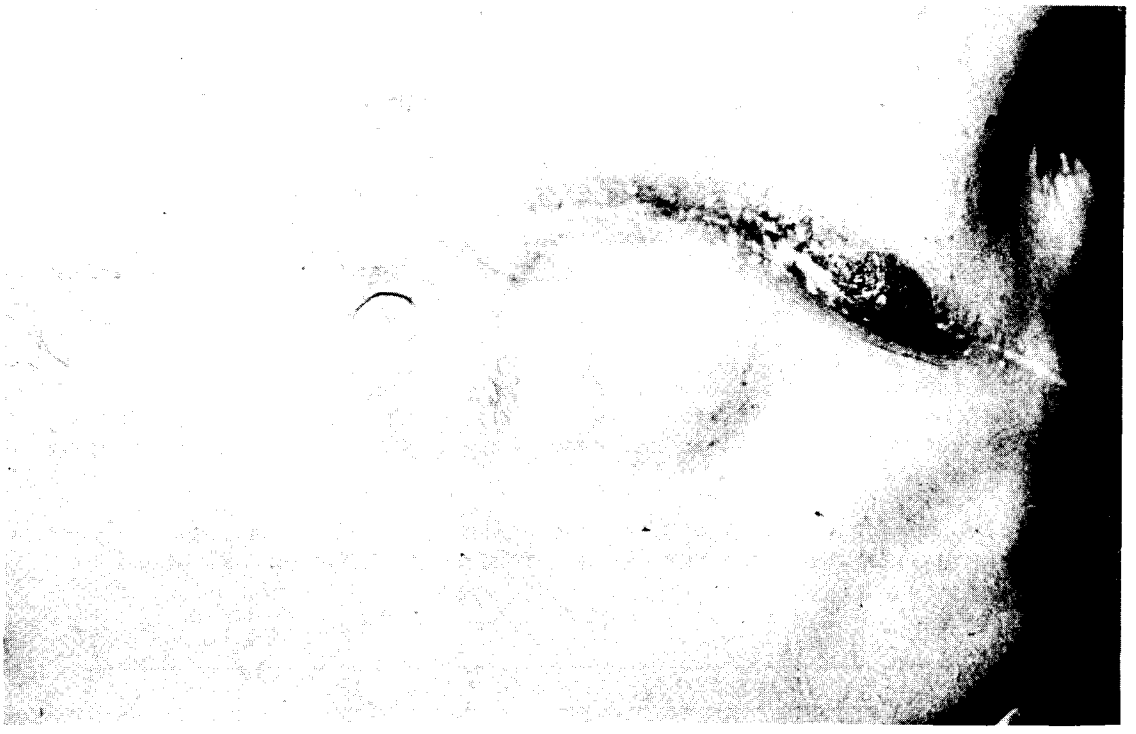

Figure 3. Appearance of main chest injury after surgery - October 1970.

Some postulations of radiation dose can be made on the basis of the clinical symptoms, their speed of progression and by comparison with tissue damage in radiotherapy. It is reasonable to assume that the injuries were due to single short exposures.

(1) The area of skin of the main lesion must have received a dose in excess of 2,000 rads but not greater than about 20,000 rads.

(2) The skin immediately surrounding the lesion where there was no erythema must have received a dose less than 800 rads.

(3) The area of observed damage to the heart muscle must have received a dose of about 2,000 rads.

(4) The area of skin of the smaller chest lesion, wrist and fingertips must have received a dose not much less than 1,500 rads.

(5) The original radiation exposure had occurred about 7 to 10 days prior to the reported onset of symptoms.

Physical Dose Estimates

Car Incident

Details of the car incident were obtained by careful questioning of the radiographer and a reconstruction carried out in October 1969. A water filled phantom was used to simulate the body to measure the attenuation by tissue for iridium-192 gamma radiation.

The reconstruction showed that the closest distance from the source to the car driver was 0.4 m at the level of the left hip. The estimated three hour exposure time gave an average whole body dose of 45 rads calculated by determining integral dose in body tissue and averaging over the whole body. The maximum dose estimated was 215 rads to the left hip. The absorbed dose calculated at the surface of the right hip gave 7 rads in water. This can be directly compared with the absorbed dose of 7.5 rads in water recorded by the film badge exposed during this incident in a wallet in the radiographer's right hip pocket.

At this early stage the good agreement between the calculated dose at the site of the film badge and that recorded by the film gave a degree of confidence in the reconstruction. Medical opinion had not yet ruled out thermal burns as the cause since the symptoms had not fully developed and the severity of the chest injury was not realised. It was impossible for the incident as described to give rise to the very localised radiation burn on the chest or damage to the wrist and fingers, consequently further radiological investigation was suspended.

Main Chest Injury

Due to circumstances outwith the control of the authors, medical treatment continued but no further radiological assessment took place until May 1970 by which time it was clear that there had been heavy exposure in addition to that occurring in the car.

After full development of the symptoms, the dimensions and radiation dose profile of the main injury derived from the degree of biological damage are shown in figure 4.

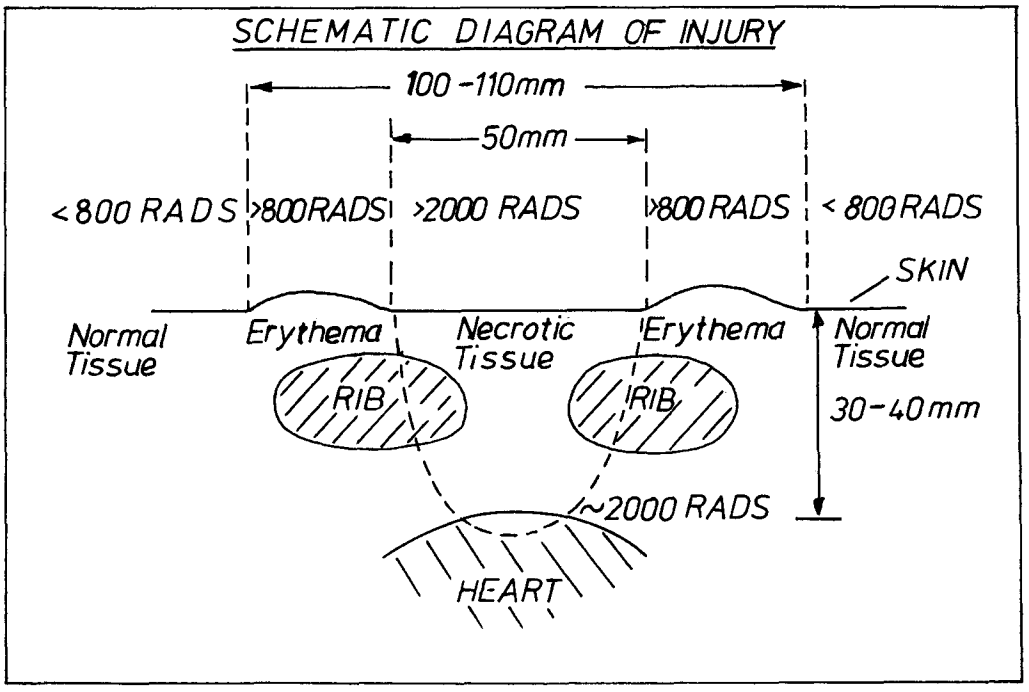

Figure 4. Schematic diagram of main chest injury showing dimensions, and estimates of dose based on the clinical symptoms.

Since the exposure had probably taken place about the time of the car incident, and to be consistent with the availability of sources to the radiographer, the 25 curie iridium-192 source was used to reconstruct the radiation dose profile for the chest injury.

Two possibilities were considered, first that the source had been removed from the source housing and was therefore unshielded and secondly that the source was mounted inside the source housing in the "exposed" position.

Radiation dose profiles were calculated for both cases at a number of source to skin surface distances and were normalised to deliver 2,000 rads at a depth of 30 mm of tissue, i.e. the heart muscle and are shown in figures 5 and 6.

Comparison of these calculated dose profiles with the estimated doses required to cause the observed biological damage of the injury, shows that the necessary conditions of exposure are met either with the unshielded source at a distance of 10 mm from the surface of the skin for about 12 minutes or the collimated source in the source housing, with the housing in contact or close to the surface of the skin for about 19 minutes.

In both cases at greater source skin distances, the radiation dose profiles are too flat to have caused the observed damage. For distances closer than 10 mm from the unshielded source, the dose to the centre of the lesion would have been too high to be consistent with the speed of development of the injury, or if the exposure time was reduced the dimensions of the injury would have been smaller than observed.

The average whole body dose was calculated at 15 rads for both cases.

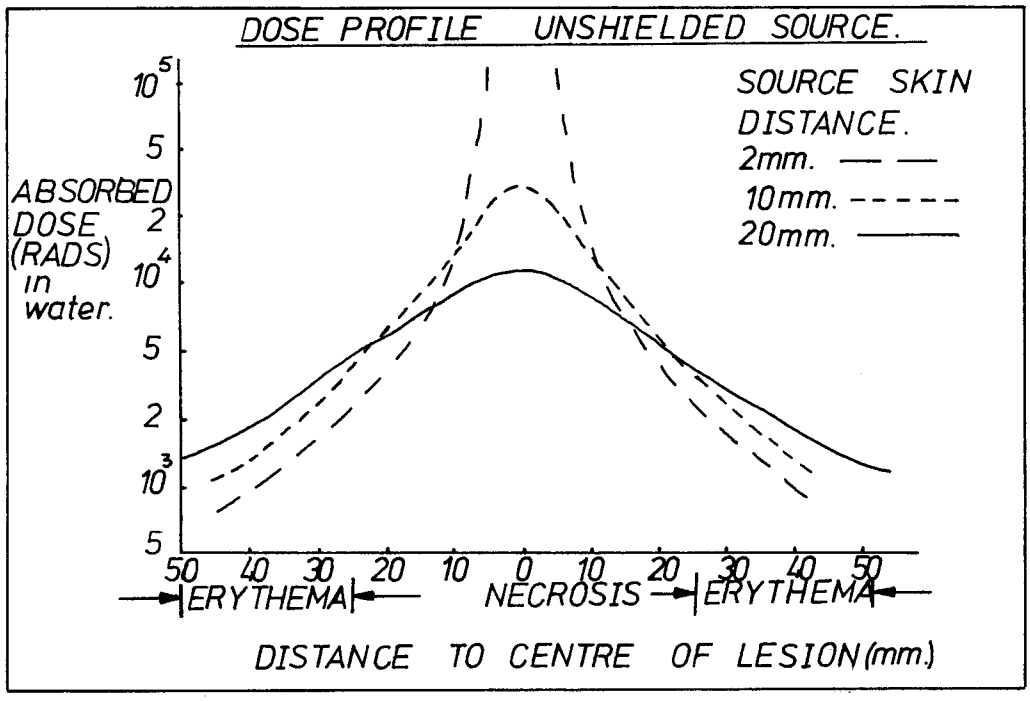

Figure 5. Surface dose profiles for the unshielded source normalised to a dose of 2,000 rads at a depth of 30 mm of tissue in the centre of the lesion.

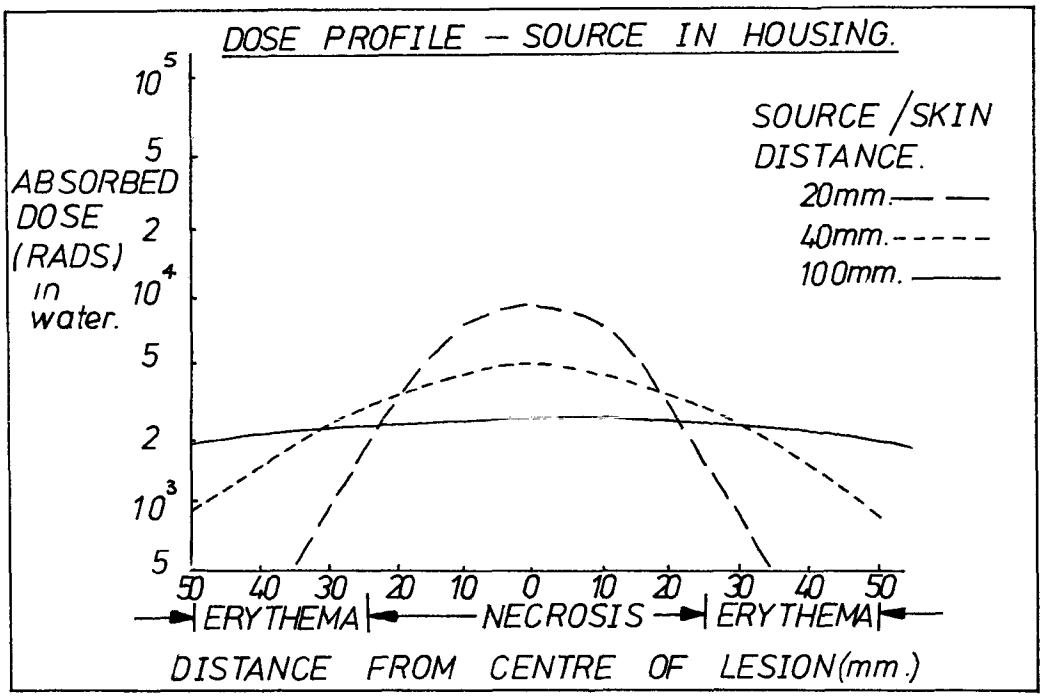

Figure 6. Surface dose profiles for collimated source in the source housing normalised to a dose of 2,000 rads at a depth of 30 mm of tissue in the centre of the lesion.

Biological Dose Estimates

Blood samples were taken in Glasgow and were sent in heparinized tubes to the National Radiological Protection Board, Harwell for chromosome aberration analysis. Lymphocytes from these samples were cultured for 48 hours by the mini-culture method[1]. Slides were prepared and 500 cells scored from each of three separate blood samples. The chromosome aberration yield is shown in table 1.

Date	Cells Scored	Damaged Cells	Dicentrics	Centric Rings	Acentric Aberration
December 1969	500	43	50	2	19

Table 1. Chromosome aberration levels in blood samples.

Only cells with 46 centromeres are included in these data[1] and in cells where one or more dicentrics were found associated fragments were also present.

The estimate of equivalent whole body dose based on the dicentric yield for this sample was 90 rads. This value was obtained from unpublished data on the dicentric yield following acute and chronic exposure of blood samples to cobalt-60 gamma radiation.

As the exposure must have been extremely non-uniform the distribution of dicentrics among cells was examined as suggested by Dolphin[2] and is given in table 2.

Date	Cells Scored	Normal	Distribution of Dicentrics				
			0	1	2	3	4
December 1969	500	457	476	20	3	0	1

Table 2. Distribution of dicentrics among scored cells.

In the first sample one cell was found with four dicentrics which is an unexpected finding at low dose levels. In calibration experiments a mean yield of four dicentrics per cell denotes a dose of about 1,000 rads and the observation of one cell in 500 with this amount of damage suggests that a small fraction of the body's lymphocyte population received a particularly high dose.

Discussion

Detailed questioning of the radiographer failed to reveal any suggestions as to the possible causes of the chest injury. Any intimate contact with a sealed source either in or removed from the protective source housing was categorically denied. It was therefore necessary to consider as a theoretical exercise what sources have been available to him and which of these could have been misused so as to cause the injury in question.

These considerations led to the two possibilities mentioned earlier concerning the large radiography source exposed either directly to the chest wall or with the source in the housing in contact or close to the chest wall. The calculations and observed damage did not allow any distinction to be made between these alternatives.

It is possible to make suggestions as to how these exposures might have taken place. These range from the unshielded source held close to but not in contact with the chest, or in the top breast pocket of a loose fitting shirt, to holding the source housing close to the chest or even lying down beside the housing.

The reconstruction of the car incident indicated good agreement between the expected film badge dose and the actual dose recorded lending a degree of confidence to the reconstruction and therefore in the estimate of average whole body dose of 45 rads. The contribution to this dose from the chest injury was about 15 rads yielding a total of 60 rads.

The estimate of whole body dose may be directly compared with that made by analysis of chromosome aberrations at 90 rads. This higher value may be expected due to the effect on the yield of dicentrics when small volumes of tissue are irradiated at higher doses.

Conclusions

The car incident undoubtedly occurred, contributing about 45 rads to the average whole body exposure. The chest injury probably added a further whole body exposure of about 15 rads.

The physical dimensions of the main injury appear to restrict the possible explanations to those discussed in this paper. All of the suggestions for the mechanisms of the exposure were completely denied by the radiographer.

The conclusions are somewhat unsatisfactory in that it is not possible to offer a firm explanation agreed by all parties.

Under these circumstances it is difficult to say what lessons can be learnt from this accident. Large radiography sources can be exceedingly dangerous if incorrectly used. It is unwise to make such sources freely available to untrained, unsuspecting persons and leave them almost completely unsupervised. Adequate instruction is vital if radiation injury is to be avoided and some form of certification of competence would go a long way towards solving this problem.

Acknowledgements

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