

OBSERVATIONS ON THE ACCIDENTAL EXPOSURE OF A FAMILY TO A SOURCE OF COBALT-60

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BOX NO 3 OF 6

FOLDER LUSHBAUGH MEXICAN ACCIDENT
COBALT-60

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I. INTRODUCTION

This publication deals with the observations carried out on a family of five persons, and on a fetus, who were accidentally exposed to ionizing radiation emitted by a capsule of cobalt-60, of 5 curies, used for industrial purposes.

The doses, the time of exposure, the manifestations of the disease, and the damage produced were of varying gravity in each one of the persons, whose ages were between 2-1/2 and 57 years. Four of the patients died as a result of the accident, and among them one was pregnant. The surviving patient is asymptomatic at the present time and is under constant observation.

All the patients were treated in different hospitals and clinics of the IMSS (Mexican Institute of Social Security) so that this study is the result of a collaborative effort. Some aspects of the accident are still being analyzed and will be reported later.

This study has a particular interest because the exposed persons received total-body irradiation in a prolonged and fractionated manner; the greater part of other reported radiation accidents have been due to short and intense total-body exposures.

The calculation of the doses received by each of the patients was difficult because they were moving about, so that the distance between them and the radioactive source varied constantly. Another important factor, which was difficult to define, was the time of exposure, so that it was necessary to reconstruct, as closely as possible, the daily life of each one of the persons.

The clinical and hematologic study of the first members of the family who showed the disease was not done in a systematic manner, because at the time the cause of their disease was not known.

The postmortem studies of the persons dying were also incomplete for the same reason; no autopsy was done on the first patient of the four dying, and in two of the other three cases the autopsy studies were incomplete.

II. HISTORICAL REVIEW

The first references on the damaging effects of accidental radiation exposure on the human being go back to the 16th century, almost 400 years before the existence of radioactivity was known.⁵⁸

It is known that some of the workers in the mines of the Erz mountains in Germany developed a disease with characteristics similar to those of lung cancer.⁴⁷

In November 1895, Roentgen discovered X rays, and two years later the Curies discovered the radiations from radium. Since that time, it has been known that electromagnetic waves can produce damage of different type and magnitude on the human being.

In 1900 [redacted] described the lesions he noted in his wife as a result of her work with radium salts,⁴ as well as the lesions she suffered in a small area of the skin of the forearm that was in contact with radioactive material.

[redacted] developed, / over a period of several years, an aplastic anemia as a result of the fractionated and multiple exposure to ionizing radiation.

The industrial use of thorium salts in the fabrication of luminous dials for watches, gave rise to fatal accidents due to the deposit of radioactive material in the interior of the body.⁴⁸

As X rays were used more widely for diagnoses and therapy, more evidence was collected, indicating that their use was attended with risks. At the present time, there are many bibliographic references on the untoward effects occurring when the human being is exposed to ionizing radiation.^{11,16}

The occupational exposure to fractionated irradiation is capable of producing severe damage, and in certain cases death. In 1900, several cases were reported of lesions due to X rays. In 1903, [redacted] reported on the

blood changes due to radiation. In 1910, malignant changes were reported on the skin of persons exposed constantly to X rays. In 1922 it was estimated that approximately 100 physicians had died because of radiation damage.^{18,58} All these cases were due to damage produced by prolonged and fractionated exposure.

In 1942 the first chain reaction was achieved,⁵⁸ and three years later atomic bombs were exploded in Hiroshima and Nagasaki. Man's ingenuity had released the radiation energy from the disintegration of the atomic nucleus, and there was occasion to study its damaging effects on the human body. These studies showed that the disease due to total-body irradiation can be acute or chronic, and that there are also intermediate stages.

Since 1945 because of the use of nuclear reactors in several countries, several accidents have taken place with exposure to parts of or to the whole body of workers and scientists.

In accidents occurring in 1945 and 1946 in the reactor of the Los Alamos Scientific Laboratory, USA, there were two fatalities in persons aged 26 and 32, in whom death occurred at the 9th and 24th day after the accident. The following symptoms were described: subcutaneous edema, blister formation, paralytic ileus, shock, jaundice, hemorrhages, fever, diarrhea, and stomatitis.²⁹

In 1954 came the report of several cases of radiation syndrome in the inhabitants of the Marshall Islands, caused by exposure to a radioactive rain resulting from nuclear experiments carried out at a distance.^{17,18,44}

In 1955 Dr. Guskova and Dr. Buisogolov published the nuclear reactor accident that occurred in the USSR in the year 1953. Two persons received total-body irradiation. Both survived. The authors reported a new observation, namely that the brain waves registered by the electroencephalogram

changed drastically during the first weeks, coming back to normal 36 days after the exposure.⁵⁴

In June 1958 an accident occurred in the Y-12 plant in Oak Ridge, Tennessee, USA, in which eight persons were exposed to gamma and neutron irradiation from uranium. The duration of exposure was only a few seconds. The five persons who were closest to the radiation source received a dose that varied from 236 to 365 rads* to the whole body; the other three persons, farther removed from the source, received doses of 23 to 69 rads. No deaths occurred.^{2,3}

In October 1958, an accident occurred in a nuclear reactor in Vinca, Yugoslavia, where six persons were exposed to gamma and neutron radiation, receiving doses from 350 to 640 rads to the whole body. The person receiving the largest dose died 32 days after the accident.⁵²

In December 1958, in an accident in the Los Alamos Scientific Laboratory, USA, a person received a high dose of radiation, from plutonium, in a time interval of approximately 30 seconds. The calculated dose was between 3900 and 4900 rads to the whole body. The patient died 34 hours after the accident.⁵⁶

* (r) roentgen: the traditional ionic dose of ionizing radiation, defined for radiations up to 2 Mev.

Rad (Roentgen absorbed dose): The unit of dose of absorbed energy in matter, for ionizing radiation of any energy. One rad equals one r multiplied by f.

f is a factor that depends on the energy of the radiation and on the atomic number of the absorbing material.^{34,37}

In our calculations the factor f was taken as 0.98 for the primary radiation.

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In May 1960 nine workers in a military installation in Lockport, New York, USA, were exposed during 20 to 120 minutes to X rays of an energy of 145 to 155 kilowatts in an accident occurring in a radar tower. Three patients received total-body irradiation; the others received radiation to only parts of the body; none died.³¹

These accidents provided detailed information on the total-body radiation syndrome, and they support the idea that a dose of 400 to 600 r given in a single exposure is fatal in the majority of cases.⁵⁹

Mathe's communication on the group of Yugoslav scientists, who were accidentally exposed in 1958, contains an important analysis of clinical and therapeutic considerations, especially in reference to the use of bone-marrow transplants.^{36,49}

The accident described in the present report is different from the other ones because five members of a family were exposed during a long period of time, four months approximately, to the gamma radiation of a cobalt-60 source, 5-curie, that was accidentally left in the house where the family lived. The disease manifested itself under different aspects in all the cases; it varied from the acute to the chronic form, depending on the magnitude of the dose received.

III. RADIOPATHOLOGIC CONSIDERATION

To make clear the nomenclature used in this report, it has been thought that the following concepts should be set forth:

1. The radiation exposure can be single or fractionated, short or prolonged, and to the total body or only to part of it.

Short exposure means an exposure received in a short period, for instance those that occur in accidents around nuclear reactors. Prolonged exposure is that received in a continuous or repetitive manner during long periods, months or years, as the occasional exposure in an X-ray department. On dealing with short exposures, a dose of 50 rads or more is arbitrarily considered to be a high dose.⁶¹

2. The radiation syndrome can be considered as acute or chronic, but intermediate forms can occur, depending on several factors; the magnitude of the dose is one of the most important ones.

Acute exposure to the whole body from penetrating radiation, as X rays, gamma rays, and accelerated particles, happens rarely in these times. The syndrome in its most acute form was observed in the Japanese people after the atom bomb explosions in Hiroshima and Nagasaki in August of 1945, and it was called "atomic bomb disease." The absorbed dose is the main parameter determining the degree of biological response. It is known that a dose of 0 to 25 rems* produces no objective signs; 25 to 30 rems cause slight hematologic changes; with 50 to 100 rems the hematologic changes are well apparent, but there is no visible damage; 100 to 200 rems cause visible and permanent damage; a dose of 450 rems or higher is probably fatal.⁵⁸

* Rem (rad equivalent man): It is not a physical unit, since it takes into account biological factors. It is defined as: One rem equals one rad multiplied by RBE.

RBE (radiobiological efficiency) is a factor that depends on the type of ionization (linear energy transfer). In our calculations RBE was taken as one.

Doses of 300 to 500 rems are considered as a lethal dose (LD_{50}) for man. This dose produces an acute and fatal disease within 30 to 60 days in 50% of the exposed persons. The acute disease is manifested by nausea appearing in a few hours, vomitus, headaches, weakness, dizziness, anorexia, tachycardia, diarrhea, irritability, insomnia, and prostration.^{2,9,17,24,28,31,39,52,56}

This initial phase is followed frequently by a period of well being, although during the latent time the tissue damage progresses. Afterwards some of the initial symptoms reappear and new ones are observed: leukopenia, hemorrhages, infections, and ulcerations of the oral mucosa. These symptoms are due to the interplay of three main alterations: (a) tissue necrosis due to the direct effect of radiation; (b) hemorrhages caused probably by a combination of damage to the capillary walls and to changes resulting from the lesions in the hemopoietic tissues; (c) secondary infections due to depression of leukocytes and possible interference with the mechanisms of defense against infection.^{9,39}

The length of the latent period is inversely proportional to the dose received; it is shorter the higher the dose.

With higher doses, of the order of 2,000 to 30,000 rads, the signs and symptoms are more pronounced and the survival is only three or four days.⁹ With doses above 30,000 rads, death usually occurs faster, almost immediately.

Those surviving doses around the LD_{50} recover slowly and may show sequelae, such as permanent alopecia, dermatosis, cataracts, and sterility.^{43,54,58} Furthermore they may develop leukemia; this has been shown in the Japanese population surviving the explosion of nuclear bombs; the frequency of the cases of leukemia is increasing in the population of Hiroshima and Nagasaki progressively along a radius leading to the center of the sites where the explosion took place.

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The chronic form of the total-body radiation syndrome is manifested by late changes:⁹ blood alterations, such as anemia, purpura, and leukemia; increase in the frequency of degenerative diseases; increase in the frequency of cancer; retardation of growth and development in children; increase in the frequency of cataracts; alteration in fertility; genetic effects and shortening of the life span.

The acute syndrome results almost always from short and intense exposures; the chronic syndrome is due to fractionated and multiple exposures and it also appears in survivors of short but not very intense exposures.

With doses of radiation less than the LD₅₀, the disease takes a subacute form. The symptoms are of lesser intensity and appear less suddenly. The latent period is longer and death is less likely. It was studied on the inhabitants of the Marshall Islands who, in 1954, were exposed to radioactive rains from nuclear experiments (Refs. 9, 17, 18, 43).

3. The radiation damage can be immediate, early, and late; systemic or localized; permanent or reversible. It is not possible to establish a precise distinction between early and late damage, but from a clinical point of view, it is considered that early damage is that observed during the first 60 days after exposure, and late damage is that appearing after this time.⁵⁷

Whether the tissue damage will be permanent or reversible depends upon the sensitivity of the tissues and upon their capacity for recovery, as well as upon the dose received. The damage is more likely to be permanent if the tissue affected has greater sensitivity and if the dose is high.

The patients studied in this investigation experienced a prolonged and fractionated exposure to the whole body. Moreover, one received intense total-body irradiation. One developed the acute form of the radiation syndrome, and the others showed the subacute and chronic forms. The damage observed was early in three cases and early and late in the other two.

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IV. DESCRIPTION OF THE ACCIDENT

Many obstacles of different nature had to be overcome in reconstructing the pertinent facts, so that some of the details have not been fully established, and their interpretation cannot be considered as final. The main facts in chronological order were as follows:

March 21, 1962. On this date the persons to be described moved to a house in which there was a lead container that held a capsule of ^{60}Co of approximately 5 curies. The source was left to the family to keep and watch without any of the members of the family knowing exactly what the container was and what it contained. This material was in Site 4 of Fig. 1.

Without their knowing how, the capsule was taken out of the container between March 21 and April 1, 1962. Sometime during this interval, the son, E.E.P., 10 years old, happened to find the capsule in the yard, and he kept it probably in the left front pocket of his trousers.

April 1, 1962. It is believed that M.C.E., the mother of the child, took the capsule from the pocket of the trousers and put it in the drawer of a piece of furniture in the kitchen (Site 3 Fig. 1).

April 17, 1962. A.I.G., the mother of the survivor, came to live in the house. At that time she noted for the first time the blackening of the glass tumblers that were kept in the piece of furniture where the capsule was (Site 3 Fig. 1).

April 29, 1962. The boy E.E.P. died in the Hospital de la Raza.

July 19, 1962. The wife, M.C.E., died in the Gynecological Hospital of the National Medical Center.

July 22, 1962. The capsule was withdrawn from the house by its owners.

August 13, 1962. One of the physicians of Clinic 19 of the Mexican Institute of Social Security suspected for the first time that the symptoms showed by the family could be due to radiation damage.

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August 18, 1962. The girl M.E.E.C. died in the Hospital de la Raza.

August 20, 1962. The husband J.E.I. and his mother, A.I.G., were admitted to the Oncological Hospital of the National Medical Center.

September 6, 1962. J.E.I. was discharged from the Oncological Hospital.

October 15, 1962. A.I.G. died in the Oncological Hospital.

In Table 1 the identification data of the cases are summarized.

V. CALCULATION OF THE DOSES RECEIVED BY THE VICTIMS OF THE ACCIDENT

1) Introduction. A reconstruction was made, by physical means and theoretical calculations, of the radiation dose received by the members of the family. The theoretical calculations were made by means of the physical information known about the capsule of ^{60}Co and the plans and geometric conditions of the site of the accident. It must be stressed that the calculations indicate only the approximate upper and lower limits of the possible doses. All minute details were noted, including those that had little immediate utility but that served later for the confirmation of some of the results.

2) Uncertainty factors that made the calculation difficult. The main difficulty in the estimation of the dose was to know for how long and at what distance from the source were the persons, since the dose varies ^{directly} inversely with the square of the distance and/with the time of exposure.

In the case of the boy, E.E.P., there was the additional difficulty of knowing exactly where he kept the capsule during the time that it was in contact with him. It is supposed that the boy found the capsule in the yard of the house, near the container, and put it in the front left pocket of his trousers a few days before April 1, 1962; this, however, could not be confirmed. Nobody knows, up to this time, whether the capsule stayed there or whether the boy put it sometimes in other places. There are two different

statements about the site where the child left his clothing during the night; Sites 1 or 2 of Fig. 1. The only confirmation that the child had the capsule in his pocket is the clinical datum of the necrotic lesion, similar to a burn, in the left thigh, compatible with a radiation lesion.

The stay of the capsule in the drawer of the piece of furniture in the kitchen (Site 3, Fig. 1) from April 1, 1962, could be confirmed only by the blackening, during the exposure, of the glass tumblers that were placed near this piece of furniture.

About the girl M.E.E.C., we can say that the distance between her and the capsule varied between centimeters and a few meters, because she stayed very close to her brother a great deal of the day, except for the time when the boy was in school. This happened before April 1.

The other adult persons kept a distance of the order of meters in relation to the capsule, except for some brief moments during which they were near the boy before April 1 and afterwards near the piece of furniture in the kitchen.

The capsule was withdrawn from the container on an undetermined date probably between March 21 and April 1. During this time, it was not possible to know exactly the hours of irradiation to the persons because they, as well as the capsule, were in motion. After April 1, when the radioactive source was in the drawer of the piece of furniture in the kitchen, the uncertainty was reduced because from then on we had to deal only with the distance and the time that the persons were in relation to the piece of furniture, which was always on the same site.

The dose is a linear function of time so that the maximum possible error of dose depends also linearly on that factor, but at short distances the variations are notably great.

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As it will be shown later, the capsule did not emit radiation in a uniform and spherical manner around its transverse axis; for this reason it is necessary to take into account the position of the capsule, that is to say, the angle that its transverse axis made with its surroundings. The maximum possible error is of the order of 50% for a 90-degree sector. The probability that the persons were always in this sector in relation to the capsule is minimal, because, since the capsule has cylindrical shape, it tends to have its longitudinal axis perpendicular to the direction of the movement of the drawer.

In the drawer there were several metallic objects that absorbed part of the radiation from the source. This percentage of absorbed radiation is impossible to determine at the present time.

Another uncertainty has to do with the scattered radiation received by the persons, which depends on the geometry of the site; this dose could not be reconstructed at the site of the accident but had to be carried out in a similar site.

The absorption of radiation by the walls and by the objects of the home presented little uncertainties in these calculations.

3. Specifications of the ^{60}Co capsule. The source that occasioned the accident was a capsule of ^{60}Co as shown in Fig. 2. It was imported for industrial use in a container supplied by the commercial firm that sold the capsule. For greater ease the capsule was changed to a different container, built by the owner of the source; moreover, the capsule was additionally covered with brass 1 mm thick in the longitudinal direction and 2 mm in its ends.

According to the certificate of the commercial firm, Atomic Energy of Canada, Limited, the capsule had 5 curies on August 3, 1961 (Fig. 3), and an average yield of 6.78 ± 0.34 r/h m. Multiplying the decay factor for 9 months,

0.900, and taking into account the 30% maximum possible error of the measurement according to the certificate, 0.21 r/h m, one obtains a dose rate for the primary beam, on May 3,* 1962, of 6.297 r/h m. The calculation was made for this day, because it was ^{midway} between the 22nd of March and the 22nd of July, 1962.

As shown in the diagram of Fig. 4, the radiation distribution from the source was not spherically symmetrical around its transverse axis because of the original and additional shielding. The absorption by the coverings reduced the dose rate by 4.5%, taking into account the half-value layer of brass, which is 2.3 cm; therefore, one obtains a dose rate of 6.014 r/h m for May 3 of this year. This value was taken as a base for all the calculations of the dose that were carried out.

4. Plans of the house, areas of radiation, and scattered radiation.

The plan of the house is shown in Fig. 1. It is a reconstruction made by direct measurements. The dimensions of the furniture were taken from the original ones that are in the house of the survivor. The piece of furniture in the kitchen, where the capsule was kept from April 1, 1962 on, did not exist at the time the investigation was carried out, and its dimensions had to be reconstructed by means of photographs, (Fig. 5.)

The house is located at the corner of a lot of 35 x 40 meters, Fig. 6, and is made with "porous" walls. The nearest house is at the distance of 30 meters. The family slept in two beds, as shown in Fig. 1, with the initials of the names, and took their meals on table (M) in the kitchen in front of the cabinet (T). The container that had lodged the capsule was at the site marked with the number 4 in Fig. 1.

Areas of irradiation: the areas drawn on the plan of Fig. 1 indicate the distances from the source, and should not be taken as being isodose

* Translator's note: probable typographical error. May 23 is the midway date.

curves, because radiation absorption was not taken into account. This was done to simplify the statistical work. The doses in each area were calculated theoretically for the primary radiation beam and are shown in Table 2. The diminution of intensity in areas 6, 7, 10, and 11 in this table were caused by the inner wall, 16 cm thick, made of "porous" material, and were calculated according to its half-value layer.

As can be seen in Figs. 1 and 7, a corner of the oven partially covered a vertical sector in areas 2, 3, 4, and 5. The six sheets of iron that cover the oven have together a thickness of 4.5 mm and absorbed 13% of the initial irradiation for these areas.

This protective factor was good to an average height, from the floor, of 1.2 m for the sites in front of the oven and sink. (See lines L-1 and L-2 in Fig. 1, and Fig. 7.)

The areas Z-1 to Z-7 are centered on point 3 of the plan in Fig. 1, which is the site where the capsule was located after April 1, 1962. The zones Z-9, Z-10, and Z-11 are drawn around point 1 of the same figure.

The windows of the house were not taken into account as radiation absorbers in making the calculations.

If the capsule had been on Site 2 (Fig. 1) instead of Site 1, the difference in exposure to the persons would not have been very great during their stay in bed during the night.

Scattered radiation: This radiation, due to the presence of walls and objects in the house, was reconstructed experimentally under the same conditions as during the accident. The obtained values are averages of all the rooms; no correction for distance from the source is necessary here. This scattered radiation is a mixture of various energies with an average quality corresponding to a half-value layer of 5 mm of copper, and it is estimated that it increased the dose by 125 milliroentgens/hour in the

kitchen, and 50 milliroentgens per hour in the bedroom. This small addition to the total dose contributed especially to the surface dose, while the dose in depth was due mainly to the direct beam.

5) Calculations of the dose received by the victims of the accident.

After the dose rates had been ascertained, the absorbed dose was calculated by trying to determine, as closely as possible, how long each person stayed in the field of radiation during the total time of exposure, and the position of each of the persons in relation to the site of the capsule. To this end it was necessary to establish in the first place a schedule of the family life and also of the activities of each member of the family. This schedule was obtained by means of an exhaustive sociological study, which was carried out through several sources of information: repeated questioning of the survivor, relatives, friends and neighbors, who by having visited the accidentally exposed family and by knowing their way of life, were able to remember some happenings and dates. Information also was obtained from the Department of Justice of the Republic, which carried out a legal investigation, from / photographs of the house taken when the accident was discovered, / from the original certificate of the capsule, and also by perusing the clinical histories of the patients.

Of all this information, the most reliable is considered to be that provided by the survivor. Information obtained from other persons was considered only when the same answers were obtained from more than one witness.

Table 3 is only one of many similar tables that were constructed to represent the activities of each of the members of the family.

Tables like this one were drawn for each of the days between the date when the family moved to the house and the date when the capsule was withdrawn, namely from March 21 to July 22, 1962.

In the column corresponding to each member of the family are specified "type days" designated with the letters E, B, C, etc. For each patient, a type day represents a schedule of certain activities. When the activities in a particular day were different from the norm this was designated as an "exceptional" day. Table 4 is an example of the calculation of the maximum exposure dose for a "type" day of patient M.C.E. During the investigations it was necessary to take into account the time changes of the activities of the family.

Because of the difficulties encountered during this study, it was necessary to make two calculations: one, taking into account all the factors resulting in a maximum possible exposure, and another in which the minimum dose that could have been absorbed was calculated.

One can see in Table 4 the estimate made of the time spent by M.C.E. in each of the areas indicated in Fig. 1. By multiplying these times by the dose rate in each area (Table 2) one obtains the doses in each area expressed in milliroentgens, which are also indicated in Table 4. By adding the doses in each area one obtains the total exposure for a "type" day in milliroentgens.

In Table 5 one finds the doses for each type day in monthly intervals, with two entries for each type day. The figure above is the number of the same type days in a month, and the lower figure is the result of multiplying the number of these days by the daily dose corresponding to such type days.

To calculate the dose contributed by scattered radiation for each person, it was necessary to calculate the time that each person stayed in the bedroom (R) or kitchen (C), because as previously mentioned the dose was different for these two sites. In Table 6 is indicated the number of

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hours that patient M.C.E. spent in each of these sites (C or R) for each type day in each month. This is an example of similar tables constructed for the other persons.

The total number of hours of stay in each site during a month was multiplied by the dose rate: 125 mr/hr in the kitchen and 50 mr/hr in the bedroom. This way one can obtain the monthly dose due to scattered radiation, which is indicated in the last column of Table 6.

Total dose: The beginning of exposure had to be decided somewhat arbitrarily, because it is not known when the capsule was withdrawn from the container. It is known, however, that it was taken out from the pocket of the child E.E.P. on April 1, 1962. Based on circumstantial evidence, surmising when it could have been easier for the child to take the capsule away, it was decided to make March 25, 1962, the beginning of exposure. All the calculations and graphs are based on this date, in order not to increase still more the difficulties of calculations.

In Tables 7, 8, 9, 10, and 11 are the total maximum and minimum doses for each member of the family. On the left side is the exposure dose contributed by the direct beam; in the central part of the tables is the contribution due to scattered radiation; and on the right of the table the total dose. This is expressed in roentgens and also in rads between parentheses.

The greatest part of the radiation had an energy of 1.3 Mev, which is absorbed at its maximum approximately 5 mm under the skin, because at this depth electronic equilibrium is reached. Several organs of the body received only a fraction of this dose, owing to absorption of the radiation in the depth of the body.

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At the foot of Tables 7, 8, 9, 10, and 11 are found the average doses received in 24 hr. For this calculation only the days during which the patient was irradiated were taken into account. For this reason the number of days is different from those in Fig. 8 in which the number of days is greater, for reasons of approximation.

Discussions of the results based on Fig. 8. As is well known the radiation effects are closely related to the dose rate. To facilitate the correlation with dose rate, Fig. 8 was drawn, which is based on Tables 7, 8, 9, 10, and 11. The abscissa of this graph is the time of exposure, and the ordinate the dose received. The day, March 25, 1962, was taken as the first day of exposure.

The child, E.E.P., not only had a high dose, but also the gradient is very sharp initially; this was because E.E.P. was exposed to irradiation by contact until April 1; after this date, the curve has a gradient similar to the curves of the other members of the family. The great difference between the maximum and minimum dose curves for E.E.P. is that in comparison with irradiation at a distance, irradiation by contact gives a very high dose rate, and small differences in the exposure time make a great difference in the total amount of radiation absorbed. In this case, therefore, the distance between the maximum and minimum curve represents the uncertainty about the time of exposure by contact. For this reason, we considered these curves only hypothetical ones. The doses received by E.E.P., taking into account the foregoing considerations were maximum 5165 rems, and minimum 2940 rems.

The slope of the curves for M.E.E.C. (Case 3) is similar to the curves for E.E.P. during the time between March 25 and April 1, 1962, because the children played almost always together in the yard. After the first of April the increment is more slow because the girl played outside,

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whereas the increment for M.C.E. was greater because she was busy in the kitchen, which is where the capsule was located. After the arrival of the mother of the survivor, A.I.G. (Case 4) and after the death of E.E.P., the slope of the curve for M.E.E.C. again is greater, because the girl stayed around closer to the adults; in other words she was closer to the radiation source. On the day when the source was withdrawn from the house, M.E.E.C. had received a maximum dose of 1,872 rems and a minimum dose of approximately 1,373 rems.

The curves for M.C.E. (Case 2) are flatter than one would think. This is because the mother of the survivor, A.I.G. took care of the household chores in the kitchen, near the radiation source, while M.C.E. was a little farther away from it because she felt sick, owing probably to the amount of radiation already received when A.I.G. arrived, as well as owing to the pregnancy. The doses received were maximum, 2,930 rems, and minimum 1,995 rems.

Considering the fetus it should be interesting to distinguish between the dose received in the superior and inferior parts of the body of M.C.E.; the doses were different when she was in areas 2, 3, 4, and 5 (Fig. 1) because of the absorption of radiation by part of the oven, so that the pelvis of M.C.E. received an approximate maximum dose of 2,767 rems and a minimum dose of 1,897 rems. The thorax and head received a maximum dose of 2,935 rems and a minimum dose of 1,998 rems. The difference therefore amounts to 5.4%. If the absorption at a depth of 10 cm in the body is taken as 60%,^a one obtains/fetal maximum dose of 1,661 rems and a minimum dose of 1,147 rems.

The dose curves for A.I.G. have a greater slope in the beginning because she took care of the chores that M.C.E. would have done.

On July 22, 1962, when the source was withdrawn from the house, A.I.G. had received a maximum dose of 2,897 rems and a minimum dose of 1,818 rems.

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Because J.E.I. had regular work hours outside the house, he received the smallest dose. It is interesting to observe that the maximum dose of M.E.E.C. coincides with the average dose for J.E.I. Because M.E.E.C. died and J.E.I. (Case 5) survived, one has to give greater weight to the minimum dose curve for J.E.I. and to the maximum dose curve for M.E.E.C. It is also coincidental that the lower curve for A.I.G. corresponds to the upper curve for J.E.I.; this might indicate that the upper curve for J.E.I. is not very acceptable. According to Table 11, J.E.I. received a maximum dose of 1,716 rems and a minimum dose of 994 rems.

6) Dosimetry studies on the blackening of glass objects during the accident. Several glass objects, as tumblers and pitchers, that were kept in the piece of furniture where the ^{60}Co source was, got darker until they became the color of coffee (Fig. 9).

This blackening of glass due to irradiation has been described in the literature and has been used for dosimetry for high doses (Refs. 5, 40, 41, and 55). We compared the blackening of the glass tumblers irradiated during the accident with the blackening obtained in similar tumblers, which were irradiated with a ^{60}Co source with controlled exposure doses. Measurements were made with a densitometer with blue Kodak filter (Wratten 47).

Although many experiments were done along these lines for several months, no acceptable results were obtained, mainly because the glass factories, even in the same run, use several mixtures of glass in the melting. For this reason, for the same exposure, different degrees of blackening were obtained in tumblers of the same type and same fabrication number. The measurements made six months afterward, to study the discoloration of the tumblers, gave variable results (Fig. 9A), which did not permit recalculation of the time of stay of the capsule in the piece of furniture in Fig. 5, for several reasons:

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(1) the tumblers were not on the same site all the time; (2) the distance between the capsule and the tumblers varied between 14 and 16 cm, which gives an error of 84%, according to the reconstruction of the piece of furniture (Figs. 9B and 3); the discoloration of the tumblers depends especially on temperature changes, and it is not known whether hot drinks were served in those tumblers, or whether they were washed with hot water, which would cause important variations in the color.

Other investigations with fixed objects with respect to the source, such as glass and plastic objects, did not give positive results, and the same can be said for the study of the discoloration of the glass fiber that was in the oven as an insulator, because on several occasions the oven was turned on.

VI PRESENTATIONS OF CASES

1. Case 1, E.E.P., son, student, male, 10 years old, was admitted to the hospital on April 16, 1962. It is surmised that between March 21 and April 1 he kept the ⁶⁰Co capsule in the front left pocket of his trousers, during a time interval of unknown duration. . It is calculated that he received a total-body irradiation dose of 5,165 rems maximum and 2,940 rems minimum in a period of 22 days.

On March 25, 1962 he fell and developed a small lesion on the left thigh. Eight days afterward an area of erythema appeared on this site, measuring 5 cm in diameter, with a blister in its center, that by the next day broke, draining some bloody material. Afterward he showed fever, anorexia, vomiting, and bloody diarrhea. On April 7 an abscess was observed on the inner aspect of the upper half of the left thigh, located in the center of the erythematous lesion, which looked like a second degree burn; this lesion extended to the region of the scrotum. He was treated with warm, wet compresses. The lesion became very painful and progressed until its center became necrotic.

On admission to the hospital the patient was in poor general condition, with intense pain in the left thigh and some listlessness. The skin of the hypogastric and inguinal region, left thigh, scrotum, and prepuce was erythematous, edematous, warm, and showing areas of superficial ulceration and necrosis. The skin of the thigh was hard and black; there was purulent secretion from the penis and scrotum; there was a urinary fistula in the anterior aspect of the lower third of the penis, and micturition was painful.

A hemogram on April 16 showed hemoglobin 8.95 g %; hematocrit 27%; segmented neutrophils 66%; bands 4%; eosinophils 1%, monocytes 4%, and lymphocytes 25%. Roentgenograms of the pelvis and left thigh taken on

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April 18 showed nothing abnormal. On the same date a urinalysis showed pH 6, albumin 2 +, bilirubin 3+, epithelial urate and cylindrical casts, and leukocytes.

Course in the hospital: The patient was given wide-spectrum antibiotics, intravenous fluids, topical treatment, and vitamins. The patient, however, remained febrile and the lesions kept on getting worse until they took the appearance of a deep abscess in the left thigh, which later extended to the right thigh. The urethral fistula became infected. A hemogram on April 24 showed hemoglobin 6.15 g%; hematocrit 20%; leukocytes 600/mm³; segmented neutrophils 36%; and lymphocytes 64%.

From April 25 on he had nausea and vomiting. On April 27 he became semicomatose and had Cheyne-Stokes respirations, fever of 40.15°C, and bloody diarrhea. At the same time the patient showed intermittent contractions of the upper extremities with clonic movements of the thumb. On April 28 he bled from the thigh lesions, and he was given a transfusion of 200 ml of blood; the patient entered into coma and died on April 29, 1962, with cessation of breathing and heart beat.

No postmortem examination was done. Four months afterward the body was taken out of the grave, but the tissues were in an advanced stage of autolysis and it was not possible to do a microscopic study. The coronor's report indicated that in spite of the long time in the tomb, the cadaver showed no signs of putrefaction, and with practically no bad odor.

2. Case 2, M.C.E., wife, 27 years old, with a 5 1/2 month pregnancy, was admitted as an emergency on July 18, 1962, because of some hematuria. She was exposed to total-body irradiation from March 25 to July 18 of that year, receiving a maximum dose of 2,930 rems and a minimum dose of 1,995 rems.

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She had had three pregnancies and two deliveries. A hemogram during one of her past pregnancies had shown hemoglobin 11.35 g %; red blood cells 4,300,000/mm³; hematocrit 40%; leukocytes 10,300/mm³; lymphocytes 28%; monocytes 6%; segmented forms 62%; and bands 4%. Urinalysis had shown albumin 0.20 g/liter.

Approximately two months after her last menstrual period, which had occurred on January 28, 1962, the patient started complaining of anorexia, nausea, and vomiting; at that time the family moved into the house. During the month of April the patient showed in addition chills, probably low grade fever, night sweats, insomnia, all the symptoms becoming more accentuated six days before admission to the hospital. Between April 16 and 29 she had noted blackening of her nails. Since early May she had shown intermittently petechiae and spontaneous ecchymosis. In the middle of May she showed hyperemesis. On May 31 a hemogram showed hemoglobin 9 g %; hematocrit 27%; and leukocytes 2,100/mm³. Approximately 20 days before admission she had noted exacerbation of the ecchymosis and also bleeding from the gums. On July 7 she complained of bilateral diffuse low back pain. On July 12 she sustained a small trauma in the left leg, which later on became infected. On July 15 she had massive hematuria, without dysuria, and in addition fever and increase in the intensity of the lumbar pain, which from then on was localized especially on the left side. On July 17 it was noted that she was extremely pale, with tachycardia of 130/min, and a blood pressure of 90/70. A hemogram showed hemoglobin 4.2 g %; hematocrit 14%; and leukocytes 100/mm³. She was referred to the gynecologic hospital for admission.

On admission the patient was in poor general condition, stuporous, pale, with a tachycardia of 140/min, and hypotensive, with a blood pressure of 90/70. On the skin surface there were numerous ecchymoses, and pain could be

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elicited by palpation in the flanks, lumbar regions, and also on the left lower abdominal quadrant. On the skin of the middle third of the left leg there was an ulceration. Rumpel-Leede's test was positive. Laboratory examination showed hemoglobin 4.5 g %; hematocrit 13%; bleeding time more than 5 min; coagulation time 9 min; and prothrombin concentration 100%.

A few hours after admission it was observed that the patient developed ecchymoses at the sites of injections and also persistent bleeding on withdrawing the needle after intravenous infusion. She was given a transfusion of 800 ml of fresh blood, and on July 19 laboratory tests showed hemoglobin 4.7 g %; hematocrit 19%; red blood cells showing anisocytosis and anisochromia; leukocytes 200/mm³; platelets 12,000/mm³; bleeding time 20 min; coagulation time 12 min; prothrombin concentration 100%; prothrombin consumption less than 10%; blood retraction absent; urea and glucose normal. The urine had a pH of 7; a density of 1.015; 2 to 4 leukocytes per high-power field; numerous erythrocytes; hemoglobin 4+; and albumin 0.60 g/liter. The urine culture showed Candida albicans.

On the same day she developed fever, vomiting of dark green material, dizziness, listlessness, chills, and she continued having hematuria without dysuria or oliguria; no fetal activity could be detected. At 1430 she lost consciousness and her tachycardia increased with gallop rhythm; for this reason she was given ouabaine intravenously; however, the tachycardia persisted and she died with heart arrest.

An autopsy was performed. The patient was also reexamined later on by removing the body from the grave and like the previous case, the cadaver showed no evidence of putrefaction.

Macroscopic findings. External appearance: There was postmortem mottling over the posterior surface of the body. Pupils were dilated. The

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gums contained a small amount of clotted blood. All over the skin surface were numerous rounded spots, not elevated, red-blue, distributed predominantly over the upper and lower extremities. On the middle third of the left leg was an ulceration measuring 2 cm long. The external genitalia had a small amount of clotted blood. The skin and mucosal surfaces were intensely pale. In the pleural cavities were numerous petechiae measuring up to 5 mm in diameter. The mediastinal lymph nodes were enlarged, deep red, and slightly hard. In the pericardial cavity were numerous hemorrhagic spots, slight flattening of the right ventricle over the septum, and petechiae in the epicardium, especially in its posterior aspect (Fig. 10).

The peritoneal cavity showed distention of the stomach,^{and} the small and the large intestine, with petechiae on the serosal surfaces. The intra-abdominal and intraperitoneal lymph nodes were enlarged, hard, and red. The uterus was enlarged, soft, with a smooth outer surface, deep red in its inferior aspect, near the cervix, where the uterus was thinner. Inside it there was a dead fetus, with the dorsum to the right and in cephalic position (Figs. 11 and 12).

Brain: Weight 280 g. The cerebrospinal fluid was plentiful and hemorrhagic; the dura showed no alterations; the brain hemispheres were symmetrical. There was a hemorrhagic area in the parietal lobes, at the level of the interhemispheric incisure over the roof of the third ventricle (Fig. 13).

This hemorrhage was limited almost exclusively to the gray matter (Fig. 14). The brain convolutions were normal, although slightly flattened. In the inferior aspect of the left cerebellar hemisphere there was an area of soft consistency, caused by the presence of a hemorrhage, predominantly cortical, which extended to a depth of less than 1 cm (Figs. 15 and 16). The inferior aspect of the brain, which bulged moderately, showed a depression at the border of the occipital foramina.

The hypophysis was slightly enlarged. The midbrain and medulla showed no alterations.

Microscopically there were subarachnoidal and parenchymal hemorrhages in the brain in the sites previously mentioned, as well as in the cerebellum. The brain hemorrhage was massive; in addition hemorrhages were found around the small vessels in the periphery of the lesion.

The hypophysis showed a notable increase in basophilic cells, severe congestion, and cysts that contained a little acidophilic material, looking like colloid. There were also calcific deposits in the pars nervosa, proliferation of the intima of the capillaries in the meninges, with striking diminution of their caliber, and also interstitial hemorrhages in the meninges, and edema of both brain hemispheres.

Tongue: The papillae were a little prominent, and the surface was whitish with little deposit. Microscopically there was slight atrophy and parakeratosis of the mucosa and of the papillae.

The tonsils were small and atrophic. The larynx was pink and had numerous petechiae. The cervical part of the esophagus had a white appearance and there were numerous scattered petechiae. The mucosa of the trachea was whitish with red areas and a small quantity of mucus. The thyroid weighed 12 g and showed no alterations. The parathyroids were small, light brown, and showed no alterations.

Lungs: The right one weighed 200 g, and the left one 180 g. They were partially collapsed, with numerous scattered small hemorrhages over the lung surfaces, more abundant in the posterior surface. On cut sections, the lungs had a deep red color and were increased in size and consistency. Microscopically the findings were focal emphysema, acute congestion, small recent parenchymal and subserosal hemorrhages filling some of the alveoli.

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Heart: Weight 180 g. A few petechiae in the myocardium were distributed irregularly on its surface (Fig. 10).

Mammary gland: Microscopically there were very few lobules if one takes into account the pregnancy; there was practically no secretion in the acini and ducts (Figs. 17 and 18). The better preserved acini showed little secretion and the lumen of the ducts was collapsed. There was also increased fibrous interstitial intralobular tissue, with considerable edema. The interlobular stroma showed increase production of collagen and a few cells, similar to what one observes in some ^{cases of} mastitis. In the intralobular edematous stroma . were plasmocytes and fibroblasts in great numbers, without a clear-cut inflammatory response; these plasma cells were scattered or in small groups in the stroma. There were some degenerative changes in the epithelial acinar cells, and probably abortive mitoses in some of them. The small blood vessels showed irregular thickening of their walls, of hyaline type.

Esophagus: The mucosa was whitish ^{but} with petechiae, which were more apparent near the cardia, where there were a few superficial ulcerations. The stomach was distended, containing mucous material and clumps of red color, appearing hemorrhagic; the mucosal folds were prominent with numerous petechiae. The small intestine contained some light green mucous-like material; there were petechial hemorrhages in the submucosa scattered irregularly, but less abundant than in the stomach. The large intestine had some mucosal and subserosal petechiae. Microscopic examination, in spite of autolysis, demonstrated marked depletion of the lymphoid tissue, and petechial hemorrhages (Fig. 19 and 20).

The pancreas was normal. Gallbladder and bile ducts showed no alterations. The liver weighed 1,520 g and appeared normal. Microscopically

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there was a slight thickening of the walls of the veins in the portal spaces, with a homogeneous fibrinoid appearance. The parenchymal liver cells showed fine granulations in the cytoplasm, dark brown, probably hemosiderin. Those cells closest to the portal spaces had most of the pigment.

Spleen: Weight 200 g./hard. ^{It was} The capsule was thin and deep red; it could be cut with ease, and the cut surface was deep red as well, with diminution in the number of malpighian corpuscles, which were difficult to recognize. Microscopically there was severe atrophy of the malpighian corpuscles, which were very scanty and had no germinal centers; they could be recognized only by the central arterioles of the corpuscle. Another striking observation was that the sinusoidal spaces, with endothelial cells, plasma, and reticular cells, were easily identified. There was no inflammation, no extramedullary hemopoiesis, and no hemorrhages.

Kidneys: The right weighed 140 g and the left 160 g. The shape was normal, the consistency was hard in some places and soft in others. The external surface had a deep-red color, more intense on the right than on the left. The superior part of the right ureter was also deep red. On cut surface, a large clot was seen in the right kidney that filled the pelvis and extended into the right ureter. Microscopic examination revealed acute congestion of the glomeruli and parenchyma; there were no vascular and tubular lesions; numerous tubules, however, contained blood. There were no submucosal hemorrhages in the right renal pelvis.

The adrenal glands weighed 4 g each. Their size was decreased and the consistency was hard. The fascicular zone of the cortex was prominent, the glomerular zone was thin, and in certain areas almost absent. The cytoplasm of the cells of the two internal zones was vacuolated, showing moderate degenerative signs, which in some places resulted in the disappearance of the

cells with rupture of the basal membrane, especially in the reticular and fascicular zone. The lipid content was, however, apparently normal.

The urinary bladder contained scant, bloody fluid and a few petechial hemorrhages in the mucosa. Microscopically there were submucosal hemorrhages with rupture of the mucosa in some areas.

The fallopian tubes showed no alterations. There was no right ovary (history of oophorectomy, Fig. 11). The left ovary, weighing 8 g, showed on cut surface two corpora lutea, the larger one measuring 7 mm in diameter. In addition, several small cysts containing a serous yellowish fluid were found (Fig. 21). On microscopic examination there were two nests of degenerating, ectopical decidual cells near the cortex. The stroma contained multiple corpora albicans of different sizes and degrees of hyalinization. Fibrinoid degeneration was observed in the walls of some blood vessels. There were two small corpora lutea where the cells were separated from each other owing to their small number; the space between them was filled with a finely granular, slightly acidophilic material similar to the cytoplasm of luteinic cells; these showed marked alterations in the nucleus as well as in the cytoplasm. The nuclei were oval or many-faceted; some were pyknotic or with irregular distribution of chromatin; in the better preserved cells a nucleolus could easily be seen, and the membrane was irregular and ill-defined. The cytoplasm showed diffuse borders; it was slightly acidophilic and granular without vacuoles. There were small foreign-body granulomas, probably caused by suture material.

Uterus: Weight 2, 120 g. The shape was globular with a smooth, shiny, external surface. In its cavity was a male fetus in cephalic presentation inside a thin, translucent-walled amniotic sac (Fig. 11). The placenta was implanted in the fundus and firmly adherent to the inner wall

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membranes were of the uterus (Fig. 12). The decidua and whitish and measured 1 cm in thickness. The cervical canal was long and narrow, with a large amount of mucus and a few pseudopapillomatous structures with cysts (Fig. 22).

On microscopic examination the myometrium showed the changes associated with pregnancy, and the endometrium was made up of a spongy decidua, with different degrees of degenerative changes. Near the myometrium were a few areas of inflammatory infiltration with mononuclear cells, predominantly plasmocytes. There were fine fibrinoid deposits in the walls of some of the myometrial venules; one of the small veins in the endometrium was thrombosed by decidual cells. The endocervix showed considerable hyperplasia and cystic glandular dilatation, as well as an infiltrate made up of mononuclear cells, almost all of which were plasmocytes.

Placenta: It had numerous villi with a single layer of trophoblastic elements. In general, the blood vessels were normal in numbers but with important degenerative changes (Fig. 23), consisting of thickening of the walls, focal fibrinoid necrosis, striking diminution of their caliber, and occlusion in some of them. The edema in the wall of some of the vessels had dissociated their fibers, which gave them the concentric appearance of "onion-skin." The stroma of the villi was very edematous and contained fibroblasts with irregular nuclei, many of which had fibrinoid necrosis (Fig. 24). The blood vessels in the thinner villi were collapsed and altered by the edema; larger blood vessels showed interstitial edema, with great dissociation of their muscular fibers, and in some of them there was proliferation of the intima with diminution of their lumen (Fig. 24). There were large areas with subchorionic fibrinoid deposits. There were frank degenerative changes in the decidua. No hemorrhages were observed.

The aorta showed a few yellowish, flat, nonindurated plaques, which were more abundant in the thoracic than in the abdominal region. The vena cava and its branches showed no alteration. The axillary, carotid, and inguinal lymph nodes were increased in size and deep red.

Bone and bone marrow: The bones showed no alteration. The bone marrow in all areas examined was extremely pale. On microscopic examination, the cellular elements were greatly diminished in numbers, showing no indication of maturation. There were almost no megakaryocytes.

Peripheral nerves showed no alterations.

In summary, the most important microscopic findings were cerebral and cerebellar hemorrhages, 3+; also in kidneys, 1+, in lungs/¹⁺ and urinary bladder 2+; absent lymphopoiesis in spleen; generalized granulocytopenia; ovarian depletion; and marked bone-marrow hypoplasia.

The probable causes of death were bone-marrow aplasia, multiple hemorrhages, and toxicity due to massive tissue destruction.

The fetus: It had the following macroscopic characteristics: Whole, well-formed, male, weighing 500 g; it measured 27 cm from the occiput to the calcaneus, with moist, flaccid, pink skin, with the exception of the face, which was cyanotic (Fig. 25).

Serosanguineous fluid leaked out when the cranial cavity was opened; the dura had no alterations; the pia showed vascular congestion. The brain lacked gyri and sulci. The base of the cranium was normal. No microscopic alterations were seen in the hypophysis.

The pericardium was moist, smooth, and shiny without alterations; it contained no fluid. The heart had a normal appearance; Botallo's foramen of the heart was patent; and microscopic examination showed no important alterations.

The pleural cavities contained no fluid. The lungs were not retracted; the right one had three lobes and the left one two; the external surface was pink, and on cut section the lung had a compact appearance. There were no alterations in the pleura. On microscopic examination the alveoli, although numerous, were covered by cuboid or cylindric epithelium, which even showed nuclear mobilization. The blood vessels and bronchi had no alterations. The thymus showed few Hassall corpuscles, and in general, there were regressive phenomena, interstitial edema, and numerous mature lymphocytes (Fig. 26).

The spleen had no malpighian corpuscles and was made up almost exclusively of red pulp; the arterioles showed thickening and apparent edema in their walls; no fibrinoid deposits were observed.

Pancreas: A very small fragment of tail, adherent to the spleen, had the fetal appearance with loose stroma in which several mononuclear cells, lymphocytes, lymphoblasts, and polymorphonuclears were observed. The rest of the pancreas was normal. Mature lymphocytes were found in a small lymph node, but there were practically no young forms or reticular cells.

In the esophagus there was moderate mucosal and submucosal infiltration by polymorphonuclears. No microscopic changes were found in the stomach or small and large intestines.

The adipose tissue was of the adult type, without any fetal fat.

Because at the time it was not known that there had been a radiation accident, no histopathologic studies were made of the liver and bone marrow of the fetus.

III. Case 3, M.E.E.C., girl, age 2 1/2 years, was seen for the first time on May 2, 1962, for an itching macropapular skin rash, localized to the extremities, not accompanied by fever. She had a past history of intolerance

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to some foods. She was exposed to total-body irradiation, probably from March 25 to July 22 of that year. She received approximately a maximum dose of 1,872 rems and a minimum dose of 1,373 rems; she was given local treatment and an antihistaminic. She returned on July 6 because she had evacuated Ascaris lumbricoides. The skin lesions had not changed with the prescribed treatment.

On July 11 laboratory analysis showed abundant Ascaris lumbricoides eggs in the stool; urine, pH 6.5, density 1.010, a small number of red and white cells, numerous bacteria, and traces of hemoglobin.

The hemogram showed hemoglobin 7 g %; hematocrit 21%; leukocytes 3,500/mm³, lymphocytes 18%; monocytes 2%; neutrophils 74%; and eosinophils 3%*. She was given antihelminthic treatment.

By the middle of July the patient developed weakness, anorexia, nausea, vomiting, fever, cough, diarrhea at times bloody, petechiae, ecchymosis, and little bloody lesions in the borders of the tongue.

On August 1, when she was admitted to the hospital, she was pale, and had blood in the oropharynx, ecchymoses in the cheeks and thigh. The skin seemed to be hyperpigmented, with a color similar to that seen in persons who have been long exposed to the sun. The labia majora of the vulva were blackened, with edema and blisters.

She remained febrile, with increase in the number of petechiae and ecchymoses, and developed microscopic hematuria and bronchial pneumonia. On August 17 a bone-marrow aspiration showed aplasia. She died on August 18, 1962.

A hemogram on August 16 had shown hemoglobin 6.6 g %; hematocrit 25%; leukocytes 2000/mm³; lymphocytes 88%; monocytes 3%; eosinophils 3%; segmented neutrophils 6%; plasmocytes 5%* sedimentation rate 64 mm; and toxic granulations in the neutrophils.

* Translator's Note: Possible typographic error in numbers?

On August 9 the hemogram showed hemoglobin 7.35 g %; hematocrit 25%; leukocytes 2300/mm³; lymphocytes 50%; monocytes 3%; segmented neutrophils 42%; and bands 3%.*

August 10: hemoglobin 5.8 g %; leukocytes 1800/mm³; lymphocytes 73%; monocytes 3%; and segmented neutrophils 24%.

The postmortem study was done on August 19, 1962. The body was in good state of nutrition; the skin was pale; the hair had been closely shaved, but there was no alopecia; eyebrows were normal; the left conjunctiva had a hemorrhagic area measuring 3 mm in diameter in its outer aspect. There was dilatation of both pupils, cyanosis of the lips, and there were superficial encrusted ulcerations in the lips. The gums were pale with ecchymoses. Brownish fluid exuded through the anal orifice. There were petechiae on the skin of the face, neck, anterior aspect of the thorax, arms, forearms, right inguinal region, and in the anterior aspect of the thighs and legs.

The scalp showed ecchymoses in the frontal, occipital, and temporal regions. There were ecchymoses in the dura, along the superior longitudinal sinus. There was congestion and edema of the brain. Microscopically there were small areas of demyelination, more marked around the blood vessels. There was moderate congestion, and a great amount of melanotic pigment was present in the arachnoid plexus; there were numerous glial cells surrounded by a clear space that separated them from the other glial cells and also from the neighboring nervous tissue. There were a few small areas of gliosis. There were a few petechiae, and some neurons showed degenerative changes, due probably to hypoxia and autolysis.

In the neck the lymph nodes were slightly increased in size and consistency; on cut surface they appeared hemorrhagic. In some of them there was pronounced congestion with hemorrhage and deposits of blood pigments,

* Translator's Note: Possible typographic error in numbers?

taken up by phagocytes. There was no evidence of germinal centers. The reticular cells were markedly hyperplastic (Fig. 27). There was lymphocytic infiltration of the capsule, with apparent breakage through it in some places; there was also chronic inflammation and fibrosis of the neighboring adipose tissue, with hemorrhages. Atypical cells, suggestive of lymphoblasts, were present. There were numerous plasmocytes, and also some lymphocytes, but these were diminished in numbers.

There was atrophy of the mucosa of the tongue and mouth, as well as severe congestion of the connective tissue stroma in the submucosa. The submaxillary glands were normal. The thyroid appeared normal macroscopically, but under the microscope it was moderately hypoplastic, and in some places it still had^a/fetal appearance; there was congestion and abundant interfollicular connective tissue stroma with very few follicles containing colloid.

In the left supraclavicular region was an area of ecchymosis measuring 2.5 cm in diameter.

Thorax: Petechiae were found in the parietal pleura. Axillary lymph nodes were increased in size and consistency and were hemorrhagic. There was no hydrothorax and the lungs were distended. The right lung weighed 100 g and had petechial hemorrhages in the visceral pleura, as well as small areas of ecchymoses. The left lung weighed 75 g and had a similar appearance. Under the microscope there was severe congestion and small areas of atelectasis next to other areas with emphysema. In one of the cut surfaces there was a triangular area of hemorrhage, with the base toward the pleura, with fairly well-preserved alveoli, some of which had a hyaline covering that was refringent and fibrinoid, similar to hyaline membrane (Fig. 28). The heart, weighing 175 g, showed areas of ecchymoses and petechiae in the pericardium and myocardium.

Abdominal cavity: There was intestinal distention due to gas; the mesenteric lymph nodes were slightly increased in size and consistency; there were petechiae in the mucosa of the stomach, small and large intestines, and peritoneum. In the terminal portion of the large intestine there was a severe hemorrhage that extended into the muscular and serosal layers, especially at the level of the rectum. On microscopic examination there was autolysis, severe submucosal hemorrhages, numerous bands of fibrous scar tissue in the mesentery, with small groups of lymphocytes and plasma cells, and slight fibrosis in some of the lymphoid follicles. The liver was congested and weighed 500 g; microscopically it showed autolysis, acute congestion, very pale erythrocytes, parenchymal liver cells with cytoplasm finely vacuolated, mononuclear lymphocytes and plasmocytes in the portal spaces. The spleen weighed 50 g, and on cut surface showed no malpighian corpuscles. There was autolysis and precipitation due to formaldehyde; over the capsule there were deposits of mucoid material and numerous bacterial colonies, which were also found in the parenchyma. The malpighian corpuscles had disappeared, only the arterioles remaining, although a few lymphocytes still persisted here and there. The architecture of the corpuscle was apparently normal. Autolysis and subserosal petechial hemorrhages were found in the gallbladder. The pancreas was normal.

The adrenals were uniformly hemorrhagic; microscopically there was interstitial hemorrhage that involved the two inner zones of the cortex, with frank destruction of many cells while others showed degenerative changes that could not be distinguished from autolysis; in some areas of the medulla, around the blood vessels, there were numerous plasma cells, some of them with atypical features.

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The kidneys weighed 60 g each; they had ecchymoses in the capsule, renal pelvis, and ureters; they showed autolysis and probably cloudy degeneration of the epithelium in the proximal tubules. The urinary bladder was distended and had small hemorrhages in its mucosa.

The bone marrow in the sternum, ribs, and vertebral bodies was extremely pale.

The probable causes of death were bone-marrow hypoplasia, and multiple hemorrhages, especially in the gastrointestinal tract and adrenal glands.

IV. Case 4A.I.G. mother, 57 years old, hospitalized on August 18, 1962. It is surmised that the patient was exposed to total-body irradiation from April 20 until July 22 of the same year. It was calculated that she received a maximum dose of 2,827 rems and a minimum dose of 1,518 rems.

In the last days of the month of April, 1962, she started complaining of feeling cold, and she noticed that the fingernails took a deep blue-black tinge. During the first days of July she started complaining of asthenia, dyspnea, and diarrhea, and she manifested ecchymoses, epistaxis, bleeding from the gums, and bloody sputum.

On August 13 a blood count showed hemoglobin 4.9 g %; hematocrit 15%; leukocytes 2,000/mm³; lymphocytes 44%; monocytes 12%; neutrophils 36%; and eosinophils 8%. Twelve hundred milliliters of blood were transfused between August 13 and August 18.

At the time of her admission she had clinical signs of purpura and hemorrhage, petechiae, epistaxis, ecchymoses, and bleeding gums. The nails of hands and toes showed the previously described blackening (Fig. 29). The blood pressure was 160/110. There were no other positive physical signs. A blood count on admission showed hemoglobin 10.6 g%; hematocrit 33%; leukocytes 1,300/mm³; marked thrombocytopenia; lymphocytes 54%; monocytes 12%;

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neutrophils 32%; and eosinophils 2%. On August 20 a bone marrow aspiration showed aplasia. Urinalysis showed an acid pH, density of 1.014, and microscopic hematuria. Determination of prothrombin in plasma and serum, BSP retention, and serum bilirubin were normal.

The patient was isolated to try to prevent infection; however, during the first few days of hospitalization she showed all the signs of pneumonitis, which was confirmed by a chest roentgenogram; in addition she was found to have a slight enlargement of the heart. The pneumonitis resolved itself rapidly with wide-spectrum antibiotics. During the last week of September she showed pustules, several millimeters in diameter in the anterior aspect of the thorax, which disappeared partially after treatment with local applications of antibiotic ointments. At that time she also had a period of diarrhea lasting five days, without fever, which subsided easily with antibiotics and symptomatic treatment.

During her hospitalization she received corticosteroids and synthetic androgens for her pancytopenia, and Rauwolfia serpentina for her mild hypertension; the blood pressure became normal and stayed so throughout the rest of her hospitalization.

Several bone-marrow aspirations, taken from different areas, kept on showing bone-marrow aplasia. New blood tests showed a prothrombin consumption of 65%, BSP retention of 28%, and persistence of her microscopic hematuria.

During her last week in the hospital, she complained of intermittent chest pain, localized in the anterior aspect of the right hemithorax, radiating to the sternum and epigastrium, accompanied by a slight, nonproductive cough, and without fever. Electrocardiograms on October 10 and 11, 1962, showed changing patterns of subepicardial repolarization of the anterolateral area of the left ventricle, which were interpreted as indicating myocardial ischemia.

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Blood and urine cultures on October 13 were negative. She was given massive doses of antibiotics, but her symptoms persisted. She died suddenly on October 15, 1962. Table 12 shows the hematologic data of this patient. The autopsy was carried out in the Pathological Medical Service of the Department of the Federal District.

Microscopic findings. External appearance: moderate rigor mortis; dependent lividities in the back of the head and neck and extremities; numerous ecchymotic areas over the skin surface, measuring up to 8 cm in diameter.

Multiple petechiae, irregularly distributed, were found all over the skin surface; eight superficial ulcerations measuring from 2 to 7 mm in diameter, covered by a deep blue material, were located in the right pectoral region and below the right breast; microscopically, the ulcerated areas showed loss of epithelium, and deposit of a fibrinoid material, under which there was an inflammatory infiltrate made up of mononuclear cells, predominantly plasma cells, which tended to be localized around the small blood vessels of the skin (Figs. 30, 31).

In the areas without ulcerations, the epidermis was atrophied, thinned, and showing moderate hyperkeratosis. These atrophic changes were also observed in the skin appendages. There was, in addition, a perivascular infiltrate in the dermis, which was less dense than under the ulcerations.

The vulva showed a tumefaction on the left side, measuring 6 cm in length, which involved the labia majora and minora, the lateral vaginal wall, and extending also under the skin to the inguinal area. This tumefaction was elastic, with well-defined and hardened borders. The skin covering it was extremely thin, friable, desquamating, and hyperpigmented. There was an ulceration measuring 2 mm in diameter, through which on pressure

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one could obtain serosanguineous fluid. The cut surface at this level showed necrotic adipose tissue, which contained pus; the lesion was not encapsulated. There was a small hemorrhage. This lesion did not involve bone, and communicated with the fistulous opening described above. In the right inguocrural area there was an old surgical scar, approximately 4 cm long, which followed the direction of the crural arch.

The abscess of the left vulvar region was the only place where purulent material could be found; it was localized in the fat tissue, and it was made of multiple small circumscribed and coalescent abscesses. The pus contained the following elements: polymorphonuclears, macrophages, cellular debris, and fibrinoid material. Around this lesion there were areas of fat necrosis (Figs. 32 and 33), hemorrhages, and a scanty number of isolated fibroblasts, without evidence of fibrosis. The walls of some blood vessels in this region showed inflammation, edema, and thickening, with proliferation of the intima, which sometimes were responsible for thrombosis (Figs. 34 and 35).

In some of the smaller vessels there were subendothelial deposits of eosinophilic material, similar to that observed in the splenic and renal arterioles (Fig. 36).

The skin covering the abscess showed moderate hyperkeratosis, and a tendency of the epidermis to become separated between the granulosa and malpighian layers, as well as between the malpighian layer and the basal layer, forming blisters of variable sizes. Many of the cells in the malpighian and the basal layers had a ballooned, pale cytoplasm, which simulated a great vacuole, pushing the nucleus to the periphery. The hair follicles in the region were scanty and atrophied, and only remains of sweat glands were found. There were no sebaceous glands in the slides studied (Figs. 37-39).

In the scalp were hemorrhagic infiltrates, the largest one measuring 6 cm in diameter localized in the left frontal region immediately under the muscle. The brain showed no alterations.

The eyelids were soft and edematous with the sclerae slightly congested. Both eyes showed the same alterations: the corneal epithelium was desquamating, there was hyaline degeneration under the conjunctivae in the nasal and temporal sides, which involved also the peripheral portion of the cornea. On the temporal side there were a few small calcifications. In the cortical zone of the lens, in its anterior aspect, were slight degenerative changes, consisting of tumefaction and fragmentation of the fibers, interpreted as bilateral cortical cataracts (Fig. 40). In the posterior portion of the choroid there was slight congestion, as well as cystic degeneration in the peripheral portion of the retina, somewhat more pronounced on the temporal side.

The thyroid gland weighed 40 g and showed no gross changes; microscopically it had an irregular shape, with small follicles some of which had a normal amount of colloid, others showing very little of it, the acini being collapsed. There was moderate congestion, and areas of inflammatory infiltration made up of plasma cells and a few lymphocytes.

The trachea contained abundant bloody material, and the mucosa was reddish.

The lungs barely retracted, due in part to adhesions to the parietal pleura, mainly on the right side. One hundred and fifty milliliters of yellow fluid were obtained from the left pleural cavity. The right lung weighed 890 g and the left one 740 g; they were large and their fissures were adherent by means of loose fibers; in some areas the pleurae were thickened and had a white-yellowish color. The rest of the pleurae were

transparent and normally shiny. The crepitation of the lungs was diminished, their color was a deep red; the left inferior lobe had a greater consistency than the superior lobe. The bronchial mucosa was congested, with abundant mucoid and hemorrhagic material. On cut surface, the pulmonary parenchyma was hard and oozed a great deal of a deep red hemorrhagic fluid. The bronchi showed moderate cylindrical dilatation of their walls. The pulmonary artery had a few yellowish plaques, which covered less than 25% of its surface. Both lungs had similar findings: they were drenched with fresh blood owing to coalescent parenchymal hemorrhage; very few alveoli contained air and others were distended or emphysematous; the hemorrhagic fluid contained almost exclusively erythrocytes without polymorphonuclears or lymphocytes; some alveoli contained only edema fluid, and in these sites there was a great number of macrophages with vacuoles. There was fibrinoid necrosis of the alveolar walls, and also under the epithelium of the terminal bronchioli; this was a homogeneous eosinophilic material, which reminded one of the hyaline membrane that is observed in the newborn and in some pneumonias (Figs. 41 and 42).

Around the larger bronchi, and in the interalveolar spaces, there was a mononuclear cell infiltration, some of the cells resembling plasma cells, and others atypical lymphocytes, as those found in the spleen and lymph nodes.

Some of the secondary bronchi were filled with a mixture of mucus and blood. The greatest part of the epithelial cells of the bronchi, bronchioli, and alveoli were exfoliated and atypical (Fig. 43).

The heart weighed 425 g. It was hard and slightly enlarged. There were 100 milliliters of yellowish fluid in the pericardial cavity; on the pericardium and epicardium there was a great amount of yellowish fat. The left ventricular wall was hypertrophied, measuring 1.6 cm in thickness; the

aorta and the aortic valve contained atheromatous plaques and there was atherosclerosis of the coronary arteries; there was also a scar measuring 2 x 1 cm in the septum.

In the mediastinum there were slightly enlarged gray-pink nodes. On cut surface they showed a great amount of anthracotic pigment. The germinal centers of the lymph nodes had disappeared, and there was also a relative decrease in the number of mature lymphocytes; there were also cells similar to those described in the spleen (lymphoblasts and plasma cells) and also some atypical cells; the reticular cells were well preserved. The lymph nodes were severely congested, and there was moderate hyperplasia of the free reticular cells, localized in the sinusoids (Figs. 44-46).

The bone marrow in the ribs and sternum was pale, yellowish, ^{and} resembling a honeycomb; the bone marrow in the vertebral bodies, however, was red. The histologic sections of the sternal bone marrow showed almost complete absence of bone-marrow elements, and increased adipose tissue; only in a few sites were small nests of myeloid cells and plasmacytes present (Figs. 47 and 48). There were no megakaryocytes.

The bone marrow of the ribs was not much more cellular than that in the sternum; it was also greatly hypoplastic. There was erythrophagia in some cells; megakaryocytes were not found, and erythrocytes were very scarce (Figs. 49 and 50).

The areas of greater cellularity were in the vertebral bodies; even here, however, the bone marrow was very hypocellular. This is the only place where a cell was seen resembling a megakaryocyte, although showing signs of degeneration (Figs. 51 and 52).

There were hemorrhages and petechiae in the muscles of the anterior abdominal wall.

In the digestive tract, the esophagus showed an ulceration measuring 1 cm in diameter, located 8 cm above the level of the cardia. The stomach, small intestine, and large intestine had numerous petechiae. In the descending colon, numerous diverticula were found, measuring from 0.5 to 1 cm in diameter. All the intestine was moderately distended, and there were congested blood vessels.

The liver weighed 1,400 g and was bound to the diaphragm by loose bands. On cut surface a small amount of blood oozed out, and the lobular pattern was faintly visible. Only minimal alterations were found, the most important one being passive congestion, Grade II.

The gallbladder was small, with intact walls; it contained a stone measuring 2 x 1.5 cm, which on being broken appeared crystalline and green. The bile ducts were normal.

The spleen weighed 50 ^g/₂ and had a normal appearance except for being slightly enlarged. It was blue and soft. On cut surface there was slight bulging of the red pulp over the capsule; the white pulp was absent. The follicles were absent, but their original site could be recognized by the presence of arterioles. At the site of the malpighian corpuscles, a great deal of a homogeneous acidophilic, fibrinoid, refringent material was located beneath the endothelium (Figs. 53-55).

There was congestion of the red pulp with pale erythrocytes, and small areas of hemorrhage (Fig. 52). There were atypical cells, probably from the reticulum, as well as erythrophagia and moderate hemosiderosis, due to macrophages containing blood pigment.

The pancreas was normal.

The right kidney weighed 150 g, and the left one 160 g; both had persistence of the fetal lobulations. The capsule could be easily stripped,

* Translator's Note: Possible typographic error?

the surface was finely granular, the consistency was normal, and the color was deep red. On cut surface the corticomedullar junction was very evident and the cortical zone was deep red as has been already mentioned. In the inferior pole of the right kidney there was a contracted zone, triangular, measuring 1.5 cm, of fibrous appearance. In both kidneys there were petechiae. The adrenals showed no abnormalities. In the small vessels were subendothelial deposits of a material similar to that described in the splenic vessels and in the vessels of the abscess (Figs. 56 and 57).

Although some of the glomeruli were hyalinized, and in others there was moderate thickening of the capillaries (Fig. 58), the majority of them showed no important changes. In many of the healthy looking glomeruli, however, ~~increased~~ permeability to red blood cells was evident in the space between the Bowman capsule and the capillary tufts (Fig. 59), and there was also blood in the tubules [Fig. 60].

There was no interstitial hemorrhage. There were alterations in the epithelium of the tubules, suggestive of cloudy degeneration, but this was difficult to distinguish from autolytic changes, although in some sites there was rupture of the basal membrane, as well as numerous hyaline and red cell casts.

In general, both kidneys showed signs of cellular atrophy, with irregular areas of interstitial fibrosis, and a certain degree of arteriosclerosis (Fig. 58).

The most important microscopic changes were bone marrow aplasia, with minimal signs of regeneration; generalized depletion of the germinal centers and lymphocytes in the lymphoid tissue; multiple and small hemorrhages in kidneys, spleen, and skin; massive hemorrhage in the lungs.

Cause of death. Anoxia due to massive pulmonary hemorrhage, bone marrow aplasia with anemia, agranulocytosis, and thrombocytopenia.

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V. Case 5, J.E.I., survivor, 30 years old, was admitted on August 20, 1962. He was exposed to total-body irradiation from March 25 until July 22 of the same year. The estimated dose was maximum 1,716 rems, minimum 994 rems.

This man was employed as a printer. In May 1962, he noted weakness, fatigability, and blackening of the fingernails exclusively (Fig. 61); this discoloration progressed throughout the subsequent months. He had complained occasionally of fever and headaches. He did not show any signs of bleeding.

On admission his blood count showed hemoglobin 14 g %; hematocrit 42%; leukocytes 2,200/mm³; lymphocytes 3%; monocytes 14%; segmented neutrophils 23%; bands 10%;* platelets 190,000. A sternal bone-marrow aspiration showed diminished cellularity ++; diminished megakaryocytes +; normoblasts 28.82%; young granulocytes 20.6%; mature granulocytes 41.76%; lymphocytes 7.05%; eosinophils 1.17%; blasts 0.6%. The patient was isolated to prevent infection. Treatment was started with corticosteroids and oxyxetholone. Because repeated blood counts were essentially normal and the patient was asymptomatic, isolation was discontinued and all medications were stopped. Table 8 shows the evolution of his hematologic data.

On September 5, 1962, another bone-marrow aspiration was performed, which showed normal cellularity, with normal numbers of megakaryocytes, although these showed slight alterations in the relation of the maturation of the nucleus and cytoplasm. The patient was discharged on September 6 in good condition. On September 17, he had an episode of bleeding from the gums on brushing his teeth; a blood count on that date showed slight thrombocytopenia, and the patient was hospitalized again and watched, without requiring any treatment. The platelets went back to normal levels. The blackening of the nails progressively diminished, shifting to the free border of the nail, as these were growing. By the end of November, the

* Translator's Note: 50 cells counted?

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blackening of the fingernails had disappeared. In February of 1963 the patient had bilateral basal pneumonitis, which was confirmed by roentgenograms. A culture from pharyngeal material was negative; he was treated with high doses of antibiotics, with resolution of the pneumonitis after a few days in the hospital.

During December, two examinations of the semen showed no spermatozoa. In May 1963, examination of the semen again showed no spermatozoa. In June of the same year, a biopsy of both testicles showed atrophy of the tubular epithelium and absence of interstitial cells (Fig. 62). The patient is still being observed frequently, and a report will be issued later on his clinical course.

VII. CLINICO-PATHOLOGIC COMMENTS

It is important to emphasize that one of the main differences between these cases and other reported radiation accidents (Refs. 2, 13, 17, 18, 31, 44, 52, 54, 56) is that the persons were exposed to fractionated and prolonged irradiation during several weeks, while in the accidents reported in the literature, the exposures were single and short, resulting in an acute or chronic radiation syndrome, according to the magnitude of the dose received.

That the tissue damage due to the radiation depends on several factors, among them the dose-time relation, is well-known. Case 1 received a maximum dose of 5,165 r to the whole body in 24 days, similar to the case described by Shipman⁵⁶, with the difference that the exposure in that case lasted only 30 seconds. Both patients died, ours 36 days after the start of irradiation, Shipman's 34 hours after exposure.

Comparing the dosimetry between the published accidents and that of this report is difficult. To say precisely what dose (and in what time) will cause death or recovery is also difficult. Case 1 developed the acute form of the disease, which was fatal with the dose mentioned; Case 5 received a dose between 984 and 1,716 r to the whole body in 119 days and recovered. The other three patients, receiving doses intermediate between those two, developed a subacute or chronic radiation syndrome, and eventually died.

Figure 8 shows the importance of the dose-time relation: the steeper the radiation dose curve, the graver the total-body radiation syndrome, and death occurs faster. Considering that the minimum dose received by Case 4, (1,818 r in 90 days) was fatal, and that the maximum dose received by Case 5, (1,716 r in 119 days) did not cause death, it can be surmised that, in adults, a dose intermediate between the two will be fatal when it is received in a time interval of 90 to 119 days.

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The following considerations are based on the clinical and pathologic findings in our cases. In Case 1 in which no autopsy was done immediately after death because the etiology of the disease was unknown at that time, the symptoms and evolution correspond to the acute total-body radiation syndrome (Refs. 2, 3, 9, 12, 17, 25, 28, 29, 36, 52, 56). The child received a high dose to the whole body in a relatively short time interval, 2,940 to 5,165 r in 24 days, including in this time an unknown period during which he received contact irradiation at the level of his left thigh: this occurred at the beginning of his exposure.

Because of this, the first symptoms consisted of erythema, which progressed rapidly to a lesion resembling a burn, and later on to wide necrosis, which involved the neighboring region of the external genitalia. The intense necrosis is easy to explain, because the amount of irradiation received in this region, when the child kept the capsule of ^{60}Co in the front left pocket of his trousers, must have been on the order of several thousand rads in a short time, probably a few hours or one or two days. Knowing that the dose rate of the source was 6.014 roentgens per hour at 1 meter, according to the inverse-square law, one obtains a dose rate at the distance of 1 cm of 60,140 r/hr. This explains the intense irradiation that the left thigh and neighboring regions must have received when the child was carrying the source in his pocket.

On the day after the appearance of erythema, the child showed the symptomatology characteristic of the acute radiation syndrome: anorexia, vomiting, fever, bloody diarrhea, and 1 week later weakness and listlessness; on the tenth day he again showed nausea and vomiting. These symptoms suggest lesions in the digestive system and central nervous system, due to radiation, and that is characteristic of the acute form. He died 12 days after the end

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of exposure, undoubtedly because of the lesions in these systems, for he showed semicomatose, Cheyne-Stokes respirations, high fever, bloody diarrhea, and intermittent contractions of the upper extremities. In other words, the nervous and intestinal manifestations of the acute radiation syndrome were present and caused his death.

Twenty-two days after the beginning of exposure, a blood count showed anemia; hemoglobin 8.95 g %; and hematocrit 27%. These values became lower 8 days later: hemoglobin 6.15 g; hematocrit 20%; a leukopenia of 600 leukocytes/mm³; and probably pancytopenia. These values indicated severe damage to the bone marrow, but the patient did not show a generalized bleeding tendency at any time, except in the necrosed area. The patient, therefore, probably did not have a severe thrombocytopenia, which is the common cause of death in persons who have received total-body irradiation if the nervous and digestive syndrome have not been so intense. In such a case there is a more prolonged evolution, which permits the hematologic syndrome to manifest itself, death occurring then almost always after the fourth week for doses near the LD₅₀ in 30 to 60 days (Refs. 21, 35, 38, 61).

As shown in Table 7, the child received a maximum daily dose of 676 rems to the whole body during the first 7 days of exposure, and later on a daily dose of 25 rems during 17 days. A dose of 600 rems is considered by many authors⁵⁸ as the medium lethal dose in 30 to 60 days, so that it is logical to consider this case one of acute radiation syndrome since he received a dose much higher than the LD₅₀ in a short time interval.

For the same reasons that an autopsy was not done immediately, no hematologic studies were done with the idea of studying the radiation syndrome. When the cadaver was exhumed 4 months after death, it was not possible to carry out the histopathologic studies necessary to determine the cause of

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death and to assess the degree of damage to the nervous system, digestive tract, and hemopoietic system, because of autolysis of the tissues. The fever that the patient showed during the evolution of his disease was due probably to secondary infection, which is a frequent complication in the radiation syndrome because of the marked leukopenia that these patients have.

When the cadaver was exhumed, no putrefaction was found and it had practically no odor, which is in agreement with the observation that radiation prevents putrefaction of living tissues for variable periods of time, according to the dose given. This fact has been used in industry for the preservation of meats.

In other radiation accidents (Refs. 6, 7, 13, 14, 19, 44) alterations similar to those seen in our cases 2, 3, and 4 were reported, such as bone-marrow aplasia with absence of megakaryocytes, thrombocytopenia, and generalized granulocytopenia, hemorrhagic tendencies, and generalized depletion of the lymphoid tissue, especially of mature lymphocytes. The final event in the three cases was massive hemorrhage, in Case 2 in the central nervous system, in Case 3 in the gastrointestinal tract, and in Case 4 in the lungs.

Case 2 received a total-body dose of 1,995 to 2,938 rems in 115 days of exposure; the calculated maximum daily dose was 25.5 rems. This case showed the symptoms of the radiation syndrome, but in lesser degree than in the previous case because the dose was lower and was absorbed in a longer time interval. The symptoms consisted of anorexia, nausea, and vomiting during the first week of exposure, although these symptoms can also be explained by her pregnancy. Later on, during the third week of exposure, she showed low-grade fever, night sweats, blackening of the fingernails, and later on, during the fourth week, hemorrhages appeared in intermittent fashion, petechiae and ecchymoses, indicative of thrombocytopenia due to bone-marrow

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damage. The thrombocytopenia appeared during the time that has been reported in most of the articles reviewed (Refs. 1, 18, 21, 32, 50, 61). The bone-marrow damage was substantiated by a blood count done on the eighth week of exposure, which showed anemia and 2,100 leukocytes.

Later on she showed massive hematuria and increased fever, signs of profound thrombocytopenia and of a probable secondary infection, which is frequent in these cases because of the leukopenia and the probable existence of diminished body defenses to infection, as related in Section III. At this time a blood count showed 100 leukocytes, 12,000 platelets, 4.2 g % hemoglobin, and a hematocrit of 14%.

Considering the evolution of this case, one would call it a subacute radiation syndrome (Refs. 9, 17, 18, 43), because she did not die as a result of the digestive-system damage, which the patient initially showed, but rather there was time for the hematologic syndrome to develop. This started in the fourth week and caused the patient's death by massive cerebral and renal hemorrhages in a period of 6 weeks after the first hematologic symptoms appeared.

The changes found in the mammary glands of this patient, consisting of reduced secretory activity in a woman 5 1/2 months pregnant, are difficult to interpret. In addition there were hyalinization of the stroma and discrete inflammatory foci. Unfortunately only one section of each mammary gland could be studied and this made interpretation of the status of the whole organ somewhat difficult. It seems possible, however, that these changes might have been due to the radiation received.

The degenerative changes found in the placenta were due probably to anemia and hypoxia, but one cannot rule out the possibility that they could have been due to radiation.

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Case 3 showed a clinical evolution similar to the previous one. She received a total-body dose of 1,373 to 1,872 rems (Table 9), in 99 days of exposure. The daily maximum dose was 89.9 rems during the first 7 days, and afterwards the daily maximum dose was 13.5 rems. The patient developed the subacute form of the radiation syndrome; she died 146 days after the beginning of the exposure, and 27 days after exposure had ended. The cause of death was hemorrhage in the adrenal glands and massive hemorrhage in the gastrointestinal tract, caused by bone marrow aplasia.

She showed systemic and digestive symptoms like those of the previous case; the bone-marrow damage was evident when the first blood count was taken, 11 weeks after the beginning of exposure. The count showed hemoglobin 7 g; hematocrit 21%; and leukocytes 3,500. The bone-marrow damage progressed and 17 weeks after the beginning of exposure, the blood count showed hemoglobin 5.8 g %, and leukocytes 1,800/mm³.

It is important to point out that this patient, 2 1/2 years old, received a maximum dose very similar to the minimum dose received by her mother (Case 2), and the girl lived one month longer. The blood counts also showed less damage to the hemopoietic system, in the beginning of her disease, than in Case 2.

The initial clinical symptoms in the girl were urticaria, and it is known that she expelled multiple Ascaris lumbricoides with the feces without any treatment, after she had been exposed to radiation for 5 weeks. It is probable that the radiation killed the intestinal parasites, and for this reason they were expelled easily; maybe the fast disintegration of these nematodes in the intestine, with the passage of the substance in the blood resulting from the disintegration of these worms, gave rise to a clinical picture of urticaria as an allergic manifestation. This possibility was

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entertained because it is known that frequently there is passage of dead intestinal parasites during the first weeks after starting a course of radiation therapy to the abdomen on patients with intraabdominal malignant tumors.

Case 4 received a dose of 1,818 to 2,897 rems to the whole body in 90 days of exposure. The maximum daily dose was 32 rems. She developed the chronic radiation syndrome, which caused her death due to bone-marrow aplasia, on the 178th day / ^{after} initiation of exposure, and 85 days after exposure had ended. Clinically in the beginning there was fatigability and blackening of the nails (Fig. 29), appearing about 15 days after the beginning of exposure. She did not show central-nervous-system or digestive-system symptoms, as is common in the subacute and acute forms of disease; rather she began having symptoms 70 days after the beginning of exposure, symptoms related to her bone-marrow damage. She developed asthenia, fatigability, dyspnea due to anemia, ecchymoses, epistaxis, bleeding from the gums, and bloody sputum due to thrombocytopenia. The first blood count, done when it was already known that there had been a radiation accident, showed the severe damage to the bone marrow. The hemoglobin was 4.9 g %; hematocrit 15%; white blood cells were 2,000 (Table 12). During this time she showed hemorrhagic purpura, which is in agreement with the low number of platelets found in the peripheral blood and with the studies on the bone marrow, which indicated aplasia.

The thrombocytopenia increased and the patient died by massive pulmonary hemorrhage in the tenth week after the end of exposure.

In the three cases described (No. 2, 3, and 4) the mechanism of hemorrhage was the severe thrombocytopenia that these patients had. Some authors, however, found that in Hiroshima and Nagasaki^{8,21} a number of cases showed hemorrhage, in whom the platelet values were not low enough to cause

hemorrhages. The mechanism of hemorrhage in irradiated patients who do not have severe thrombocytopenia has been explained by the presence of a substance in the blood with properties similar to those of heparin.¹ In some experiments,³⁵ however, no anticoagulants have been found in the blood of irradiated animals, and it seems that the hemorrhages are closely related to the number of circulating platelets.

In the three patients who died because of hemorrhages, bleeding occurred in different sites: central nervous system, lungs, and gastrointestinal tract. It is very difficult to determine which factor or factors determine in what organ the hemorrhage will be most severe.

In the three patients who were studied post-mortem, the white blood cells diminished constantly, without showing any signs of recovery. In particular, in Case 4, which was studied more thoroughly, the histologic sections from the sternal, costal, and vertebral bone marrow showed aplasia, with the exception of a few regenerative foci in the bone marrow of the vertebral bodies, although the patient lived 178 days. This lack of bone-marrow regeneration has not been reported in other accident cases where the exposure was short and intense. This may be explained because our patients received total-body irradiation in a prolonged and fractionated fashion, which prevented bone-marrow regeneration at any time.

In the three cases studied port-mortem there was generalized depletion of the lymphoid tissue, mainly of adult lymphocytes and of the germinal centers of lymph nodes; this was less pronounced in Case 3.

The blackening of the nails, in Cases 2, 4 and 5, has been considered to be due to changes brought about by the absorbed radiation on the mineral salt of the nail. In Case 5 this discoloration progressively disappeared as the nails kept on growing.

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The presence of homogeneous, acidophilic, refringent material, with a fibrinoid appearance, localized under the endothelium of the splenic and renal arteries, as well as in other organs, which was particularly apparent in Case 4, but was also evident in Case 2, may be due to arteriosclerosis. It could also be due, however, to small subendothelial hemorrhages caused by direct radiation effect on the blood vessels; the age of the patient of Case 2, 27 years, would seem to rule out arteriosclerosis as a cause for these findings.

In none of the cases studied by autopsy did we find the typical vascular lesions due to radiation, described in the literature. The only vessels with arteritis (proliferation of the intima and thrombosis), were in areas near the vulvar abscess in Case 4, and these lesions could be attributed to the inflammatory process as well as to the radiation.

The hemorrhage and pulmonary edema are sufficient to explain the presence of "hyaline membrane" on the walls of the alveoli and some of the terminal bronchioli, as in Cases 3 and 4. It is not possible, however, to rule out the possibility that this was a direct radiation effect on these organs.

In Case 4 alterations were found on the anterior aspect of the lens, which were interpreted as a bilateral, cortical cataract. These lesions, however, are not considered to be due to radiation, because the typical postradiation cataract starts in the posterior surface of the lens.¹⁵

The fetus, contrary to expectations, was found with practically no alterations. It showed only depletion of the lymphoid tissue, and absence of hemopoiesis in the spleen and lymph nodes. These changes, however, were less pronounced than those found at autopsy in the other cases. It is notable that the thymus was normal, only the Hassall corpuscles being diminished.

Although, unfortunately no histologic slides of the bones and liver of the fetus were available, it is worth noting that there were no hemorrhages at all, and its development was normal. It is thought that this fetus died in utero, probably owing to maternal anoxia, death occurring a few hours before the mother died.

The near normality of the fetus can be explained by what we know from experimental animals. If the embryo or fetus is exposed to radiation after the period of implantation of the ovum, the fetus may survive even though it may have sustained damage. The belief that abortion will result if the embryo is damaged by radiation is not true. The lack of anomalies of the fetus may be explained by taking into account that it was already 8 1/2 weeks old when the exposure was begun; it has been found that the period of organogenesis is the one of greatest potential danger to the embryo.⁵³ More than 20 abnormalities of development are known, especially in the central nervous system (mainly microcephalia and ophthalmic lesions), and these lesions are more likely to occur if the radiation was given from the second to the seventh week of pregnancy. It is considered, therefore (1) that the dose received by the fetus was perhaps not high enough to provoke its death (1,147 to 1,661 rems in 115 days); (2) that because the exposure started after the period of maximum organogenesis, the fetus showed no developmental abnormalities; (3) that there was moderate damage to the lymphatic and hemopoietic systems so that it seems that if the mother had not died the fetus probably would have been born alive, but with hypoplasia or aplasia of the bone marrow that would have caused an early death. The latter point is only supposition, because no observations were available on the status of the bone marrow. It is based on the surmise that if the mother developed bone-marrow aplasia, it is logical to suppose that the fetus would have shown it, too, since the dose received by both was similar.

Case 5, the survivor, received a dose between 984 to 1,716 rems in 119 days; it was calculated that the maximum daily dose to the whole body was 16.2 rems (Table 11). He was the person receiving the least exposure. He showed a clinical picture with the characteristics of the chronic form of the total-body radiation syndrome, but he later recovered. Aside from the blackening of the nails, which has been previously commented upon, his symptoms were all due to bone-marrow hypoplasia, caused by radiation. On the thirty-sixth day after initiation of exposure he developed fatigability and blackening of the nails. One hundred and seventy-eight days after initiation of exposure he had a leukopenia of 2,000 and 190,000 platelets (See Table 13). The bone marrow at that time showed diminished cellularity and also diminished megakaryocytes. By the 204th day after beginning of exposure, 55 days after the end of exposure, he had already recovered, for the bone marrow at that time was reported as normal, except for discrete alterations in the megakaryocytes. Only on one occasion did he show slight bleeding from the gums; this occurred on the fifty-sixth day after the end of exposure, and coincided with a moderate thrombocytopenia. The discoloration of the nails disappeared 130 days after termination of exposure, owing to the normal growth of the nails. Testicular biopsies 22 and 46 weeks after the end of exposure showed absence of spermatozoa (Fig. 62), and this is in agreement with what is found in the literature.

The patient at the present is asymptomatic and he is being closely followed. His clinical course will be reported later, for it is likely that he may develop late radiation sequelae.

A summary of the doses received, times of exposure, some clinical data, and the date of death of the exposed persons are summarized in Fig. 63.

1006048

PRIVACY ACT MATERIAL REMOVED

FIGURES

- Figure 1. Plan of the family dwelling.
- Figure 2. Model of the cobalt-60 capsule.
- Figure 3. Photostat of the certificate of the cobalt-60 capsule.
- Figure 4. Diagram showing the asymmetric distribution of radiation around the transverse axis of the capsule.
- Figure 5. The piece of furniture (cabinet) in the kitchen; the capsule was kept in this drawer.
- Figure 6. Lot where the house was located.
- Figure 7. Elevation of the frontal view of the kitchen.
- Figure 8. Doses absorbed by the accident victims (rem).
- Figure 9. Blackening of the glass tumblers in comparison with nonirradiated tumblers.
- Figure 9a. Graph of the blackening curves for test glass tumblers irradiated with ^{60}Co .
- Figure 9b. Reconstruction of the piece of furniture (cabinet) of Fig. 5.
- Figure 10. Posterior aspect of the heart showing subepicardial petechiae.
- Figure 11. Uterus containing the fetus before opening of the membranes.
- Figure 12. External aspect of the fetus in the uterus.
- Figure 13. Hemorrhage on the surface of the brain.
- Figure 14. Coronal section of the brain showing the extension and depth of the hemorrhage.
- Figure 15. Inferior aspect of the brain showing a hemorrhage in the left cerebellar hemisphere.
- Figure 16. Hemorrhagic area in the cerebellum. Enlargement.
- Figure 17. Few lobules and discrete secretion in the mammary gland.
- Figure 18. Secretion and edema in the mammary gland.
- Figures 19 and 20. Absence and atrophy of the lymphoid tissue in the intestine.

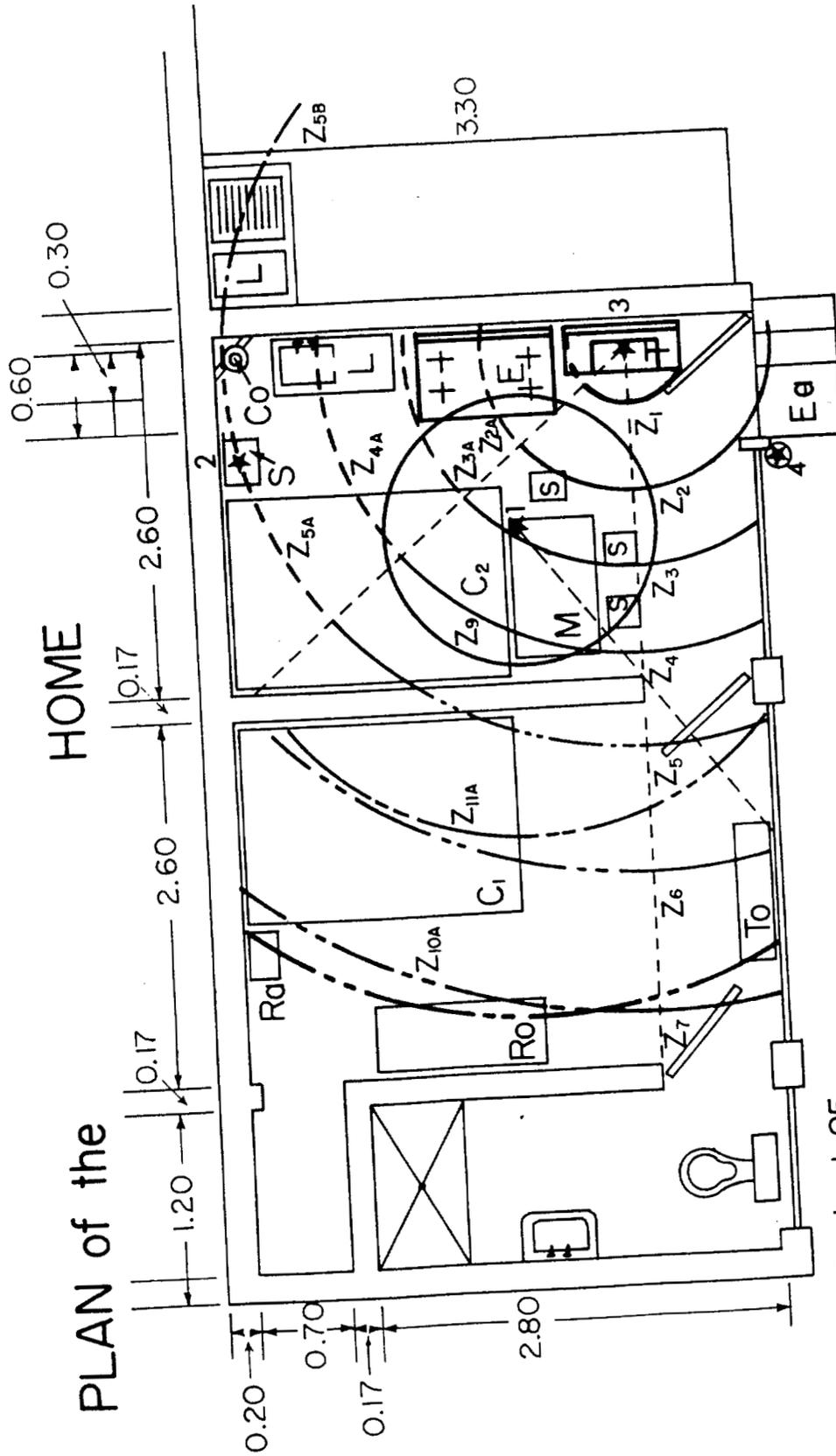
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- Figure 21. Left ovary and tube, showing a corpus luteum of pregnancy.
- Figure 22. Inner surface of the uterus showing its mucosa and the site of the placental implantation.
- Figure 23. Fine villi. There are no hemorrhages.
- Figure 24. Vascular changes in the placenta.
- Figure 25. External aspect of the fetus. Observe its normal development.
- Figure 26. Microscopic appearance of the fetal thymus. Regressive changes with few Hassall bodies and mature lymphocytes.
- Figure 27. Lymph node showing hyperplasia of the reticular cells and edema.
- Figure 28. Lung. There are areas of emphysema and atelectasis.
- Figure 29. Photograph of the patient's hands showing the blackening of the nails and pigmentation of the skin.
- Figure 30. Base of an ulcer resembling a first degree burn; absence of polymorphonuclears.
- Figure 31. Dermis under the ulceration with perivascular inflammation, made up almost exclusively of plasma cells.
- Figure 32. Abscess localized in the soft tissues of the left vulvar region, where one can observe a great number of polymorphonuclears, which were practically absent in other parts of the body.
- Figure 33. Enlargement of the previous photomicrograph. There are, in addition, macrophages and cellular debris.
- Figure 34. Blood vessel in the abscess, with secondary inflammation of its walls and organized thrombus.
- Figure 35. Enlargement of the previous figure.
- Figure 36. Acidophilic material in the subendothelium of a blood vessel in the abscess.

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- Figure 37. Skin of the area of the abscess with inflammation, fibrosis, bullae, necrosis, and degenerative cellular changes.
- Figure 38. Skin of the area of the abscess showing details of the epidermis with incipient formation of vesicles.
- Figure 39. Another area of skin showing the same changes as in the previous figure.
- Figure 40. Lens. Showing a cortical cataract.
- Figure 41. Lung showing hemorrhage and acidophilic material similar to hyaline membrane. Masson.
- Figure 42. Enlargement of the previous figure. Masson.
- Figure 43. Atypical alveolar cell and foamy macrophages, plasmocytes, and lymphoblasts. Masson.
- Figure 44. Lymph node showing disappearance of the germinal centers and hyperplasia of reticular cells.
- Figure 45. Lymph node showing congestion and absence of germinal centers and lymphocytes.
- Figure 46. Lymph node. There are lymphoblasts, plasmocytes, and reticular cells, and absence of mature lymphocytes.
- Figure 47. Sternum bone marrow. General view showing the meager cellularity.
- Figure 48. Enlargement of the few cellular areas of the previous figure.
- Figure 49. Rib. One can see the cellularity of the bone marrow.
- Figure 50. Enlargement of the previous figure, showing good conservation of reticular cells, and little variation in the morphology of the myeloid series.
- Figure 51. General view of the bone marrow in the vertebral body.
- Figure 52. Enlargement of the previous figure showing a cell that could be a degenerated megakaryocyte.

- Figure 53. Diminution of the follicles and small hemorrhages in the spleen.
Masson.
- Figure 54. Acidophilic material in the subendothelium of the arterioles
of the splenic follicles. Masson.
- Figure 55. Enlargement of the arteriolar lesion, surrounded by lymphocytes
and plasmocytes. Masson.
- Figure 56. Group of renal arterioles with subendothelial acidophilic
material. Masson.
- Figure 57. Enlargement of the previous figure. Masson.
- Figure 58. Glomerulus with discrete thickening of the capillary wall.
The proximal tubules show exfoliation of the epithelium and
autolytic changes. Arteriole with sclerosis.
- Figure 59. Glomerular and tubular hemorrhage in a normal appearing
glomerulus.
- Figure 60. Renal tubules distended with red blood cells.
- Figure 61. Blackening of the fingers of J.E.I. (Case 5), which was not so
pronounced as in A.I.G. (Case 4).
- Figure 62. Section of atrophic testis with absence of interstitial cells.
- Figure 63. Summary of the principal data of the presented cases.



PLAN of the

HOME

Scale: 1:25
 Height of ceiling: 2.40 meters

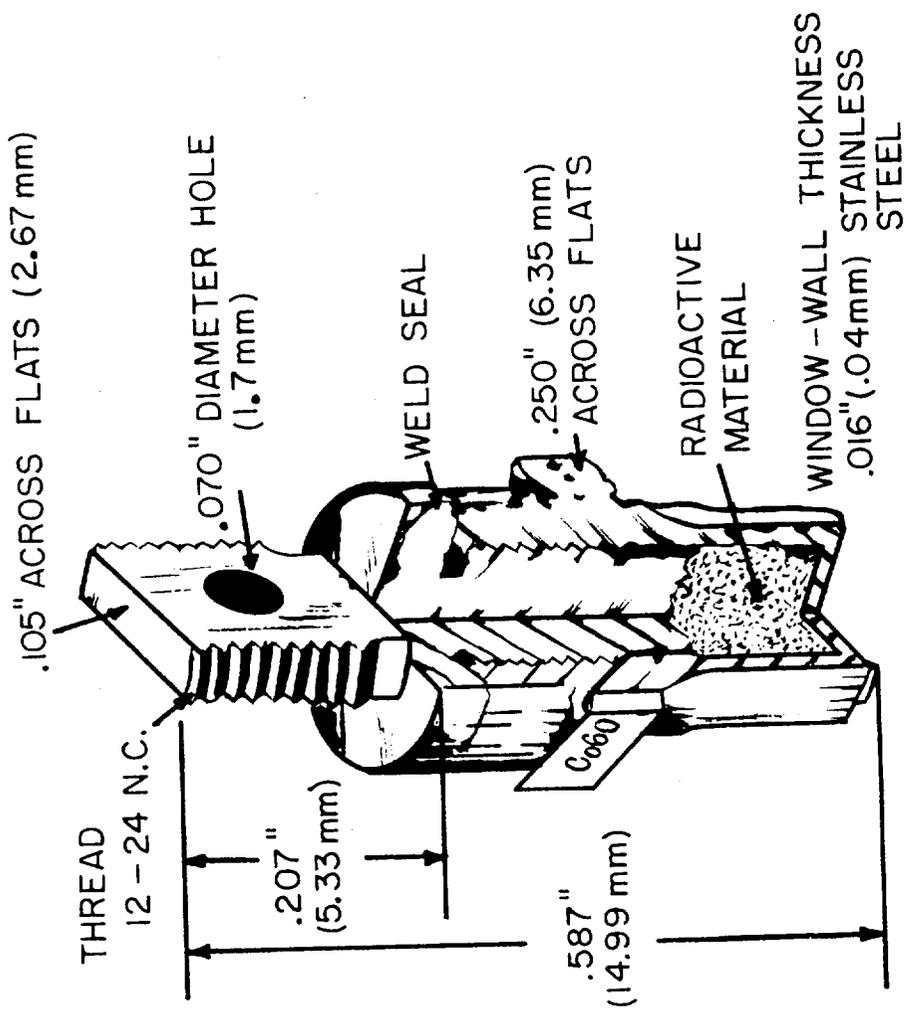
KEY

- Ea - Stairs
- T - Cabinet
- L - Sink
- S - Chair
- Ra - Radio
- M - Table

AREAS

- Area without absorption
- - - Area with absorption by the oven (11.3%)
- Area with absorption by the external wall (56%)
- Area with absorption by the internal wall (50%)

- C - Bed
- E - Oven
- Ca - Heater
- To - Dressing Table
- Ro - Dresser

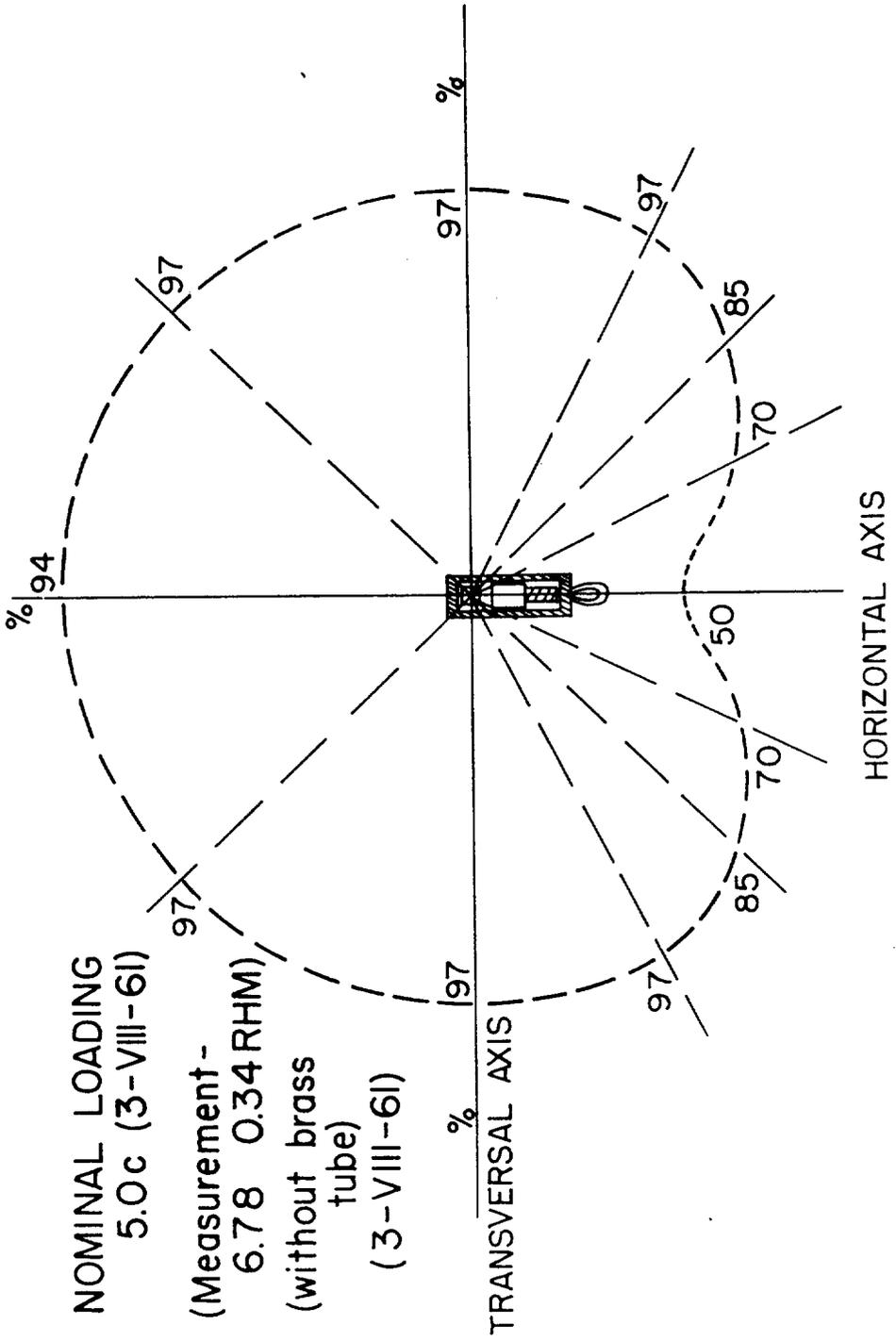


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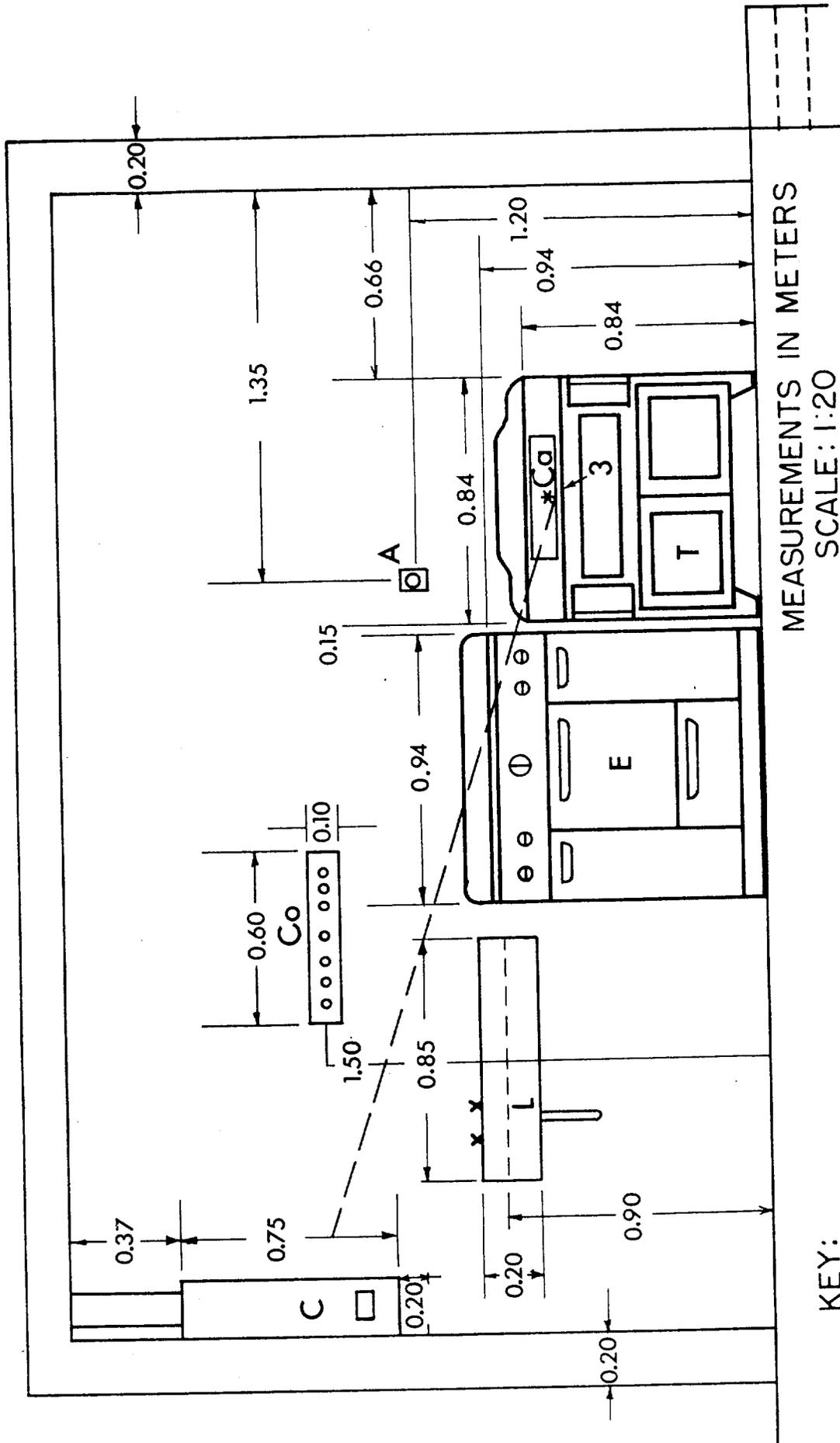
DISTRIBUTION OF RADIATION of a Type C-164 ^{60}Co Source (INSIDE A BRASS TUBE)

NOMINAL LOADING
5.0c (3-VIII-6I)
(Measurement -
6.78 0.34 RHM)
(without brass
tube)
(3-VIII-6I)



KITCHEN ELEVATION OF THE HOME

Location of Capsule in Drawer in Relation to Oven

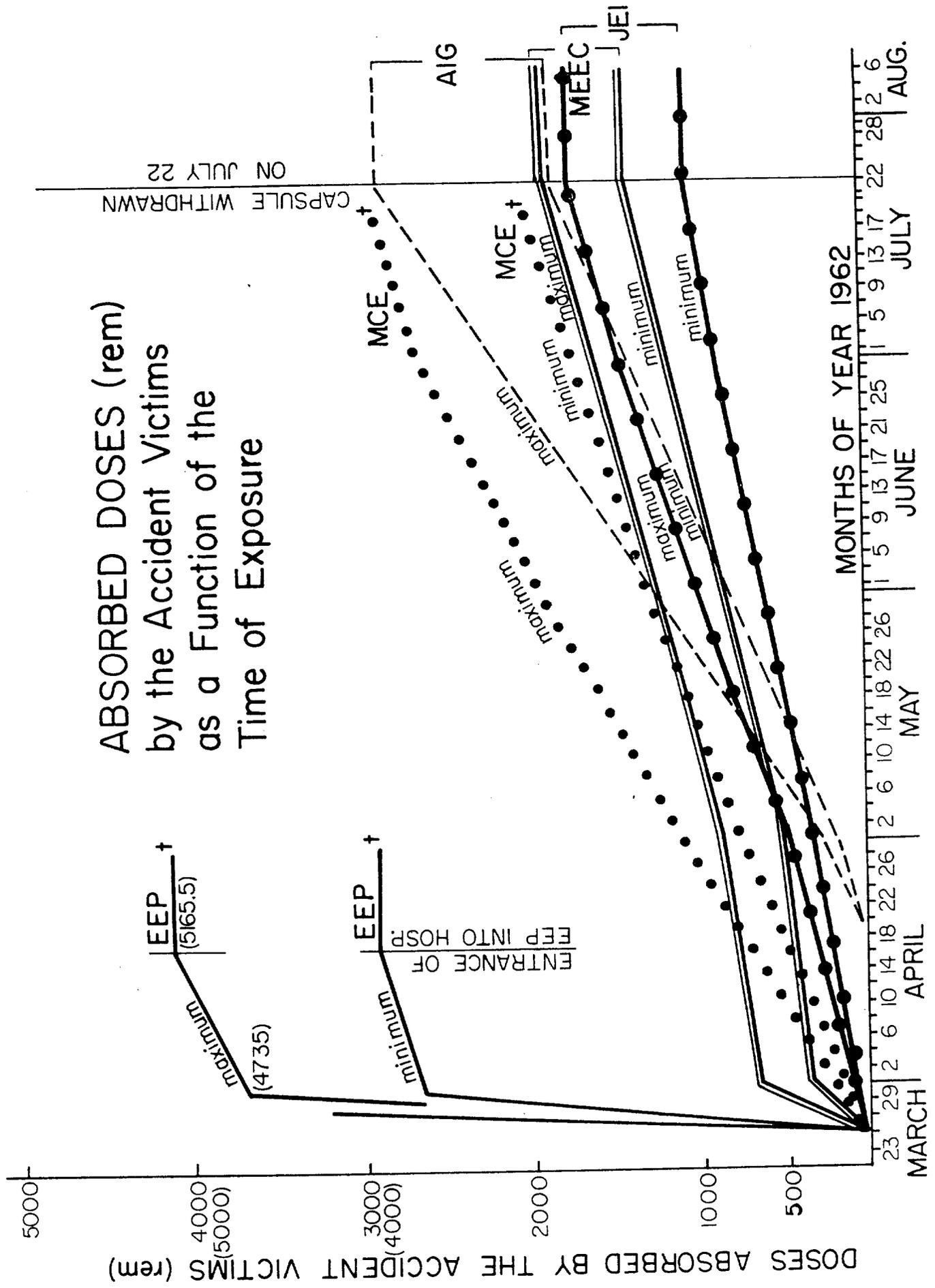


MEASUREMENTS IN METERS
SCALE: 1:20

- KEY:
- L- SINK
 - E- OVEN
 - T- CABINET
 - A- SWITCH
 - C- HEATER
 - Co- HANGER
 - Ca- POSITION OF CAPSULE (SITE 3)

1509001

ABSORBED DOSES (rem) by the Accident Victims as a Function of the Time of Exposure



DOSES ABSORBED BY THE ACCIDENT VICTIMS (rem)

EEP
(5165.5)

EEP
(4000)

ENTRANCE OF
EEP INTO HOSP

CAPSULE WITHDRAWN
ON JULY 22

AIG

MCE †

MEEC

JEI

MONTHS OF YEAR 1962

MARCH

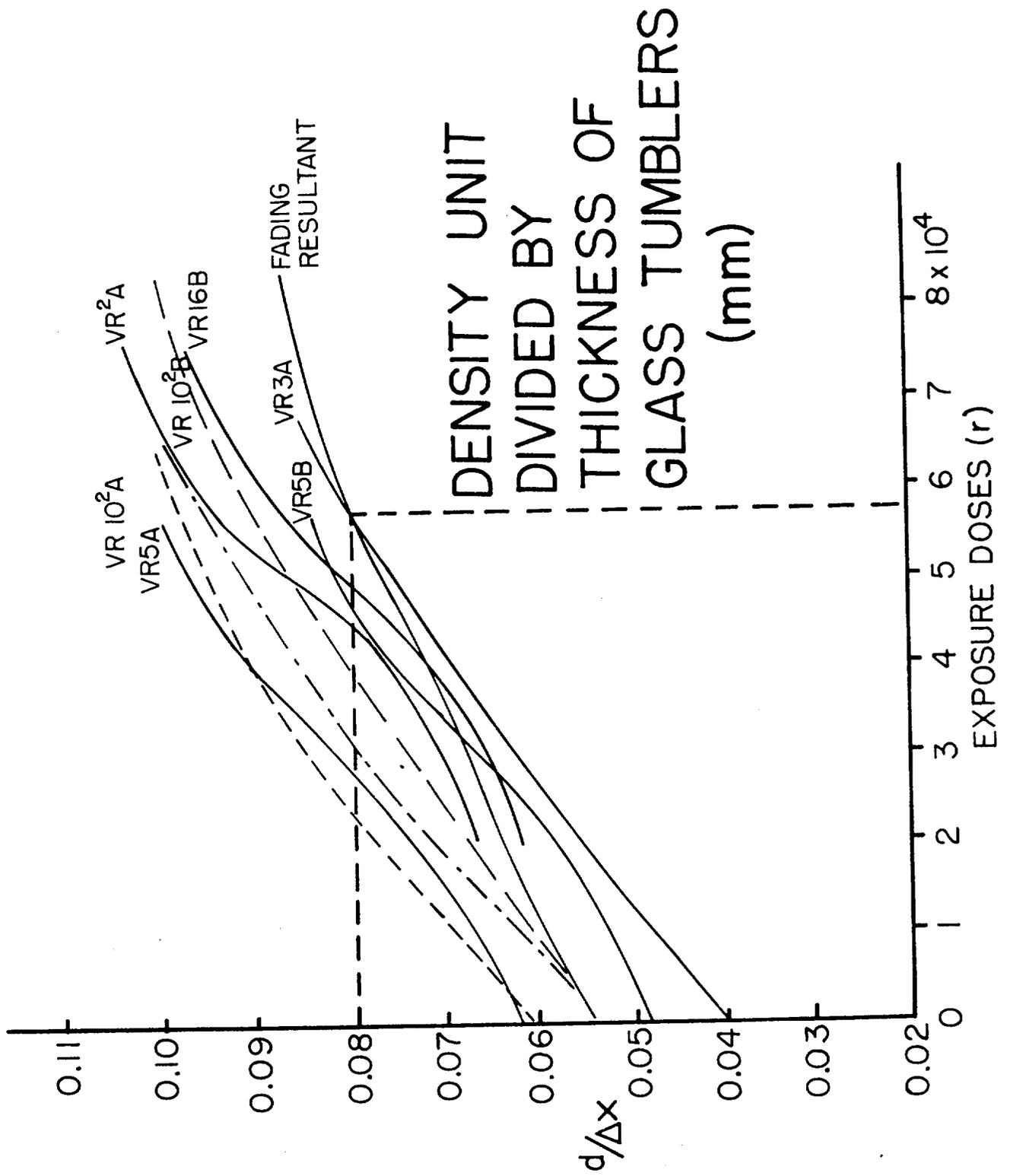
APRIL

MAY

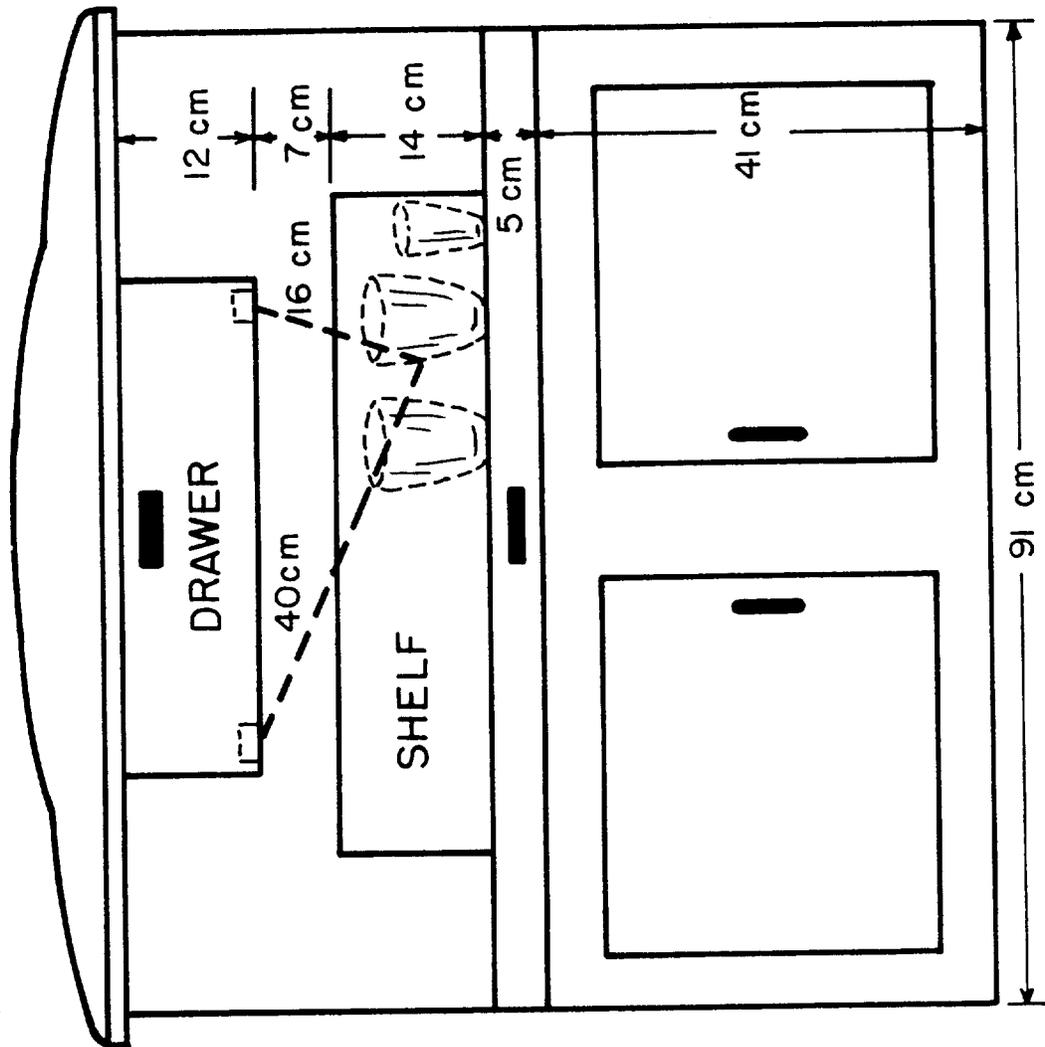
JUNE

JULY

AUG.



1006058



1006059

1006060

KEY TO GRAPH

 RADIATION SOURCE IN MOTION
(IN POCKET OF E.E.P.)

 DAYS OF IRRADIATION

 DAYS WITHOUT IRRADIATION

I INITIATION OF CLINICAL SYMPTOMS

† DEATH

↓ DATE OF APPEARANCE OF:

A - ANOREXIA

NV - NAUSEA, VOMITING

D - DIARRHEA

DS - BLOODY DIARRHEA

F - FEVER

S - BLEEDING (DUE TO SKIN OR MUCOSAL
LESIONS, WOUNDS, OR HEMATURIA)

UN - BLACK NAILS

C - CYTOPENIA

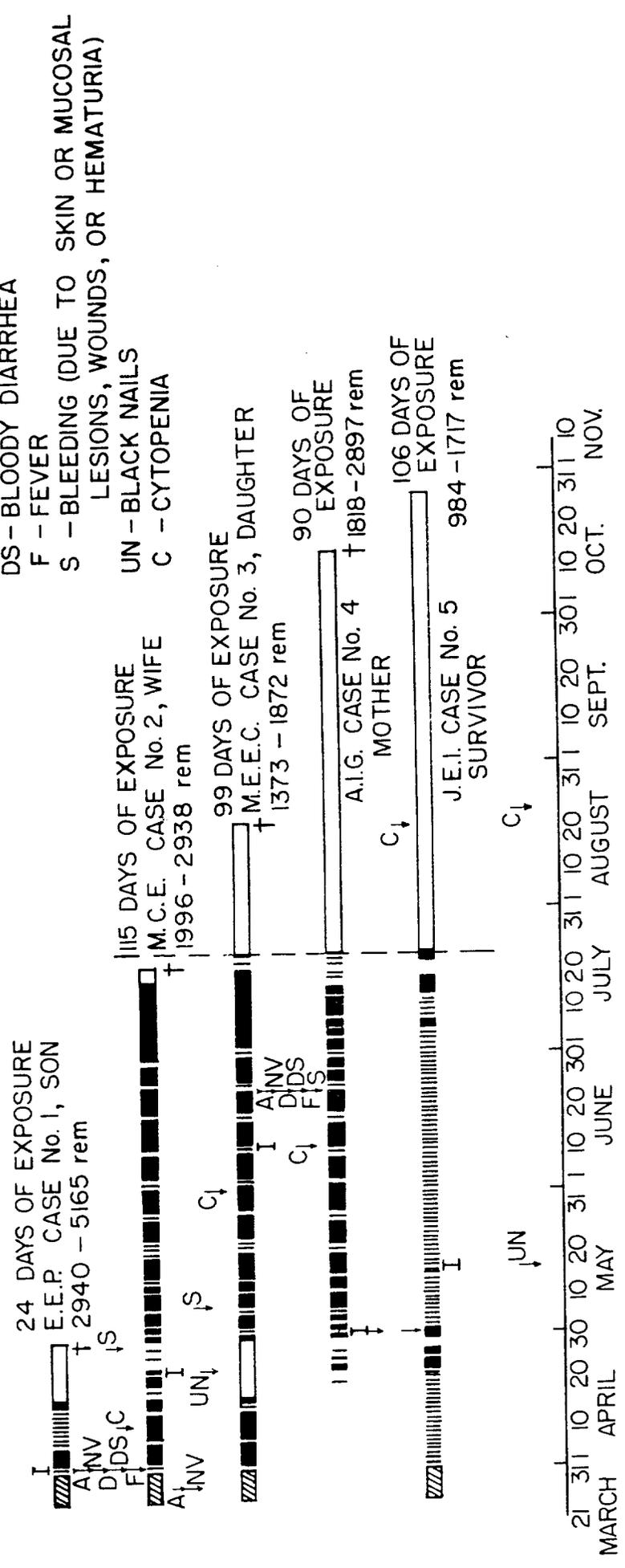


TABLE 1
IDENTIFICATION OF THE CASES

CASES	NAME	AGE	SEX	DATE OF FIRST CONSULTATION	DATE OF DEATH
Case 1 SON	E.E.P.	10 yr	M	7-IV-62	29-IV-62
Case 2 WIFE	M.C.E. fetus	27 yr 24 weeks of M gestation	F	21-IV-62	19-VII-62
Case 3 DAUGHTER	M.E.E.C.	2 1/2 yr	F	2-V-62	18-VIII-62
Case 4 MOTHER	A.I.G.	57 yr	F	13-VIII-62	15-X-62
Case 5 HUSBAND	J.E.I.	30 yr	M	13-VIII-62	Survivor

1006061

TABLE 2

AREAS, DISTANCES, AND EXPOSURE DOSE RATES
(The doses were calculated for the direct beam)

AREA	DISTANCE (Meters)	OBSERVATIONS	DOSES in' mr/h
Z _c	0.20	Direct contact	150,350
Z ₁	0.40		37,580
Z ₂	1.00		6,014
Z _{2a}	1.00	Absorption by approximately 4 mm of iron sheet of the oven (13% less than the initial dose)	5,230
Z ₃	1.60		2,350
Z _{3a}	1.60	Absorption by the oven (see before)	2,040
Z _{4a}	2.20	Absorption by the oven (see before)	1,080
Z ₅	2.90		715
Z _{5a}	2.90	Absorption by the oven (see before)	620
Z _{5b}	2.90	Absorption by the external wall of the house, porous walls 20 cm thick (56% less than the initial dose)	314
Z ₆	3.80		261
Z _{6a}	3.80	Absorption by the inner wall of the house, porous walls 17 cm thick (50% less than the initial dose)	208
Z ₇	4.80		261
Z _{7a}	4.80	Absorption by inner wall of the house (see above)	130
Z ₉	1.00		6,014
Z _{10a}	3.50	Absorption by the inner wall of the house (see above)	245
Z _{11a}	2.30	Absorption by the inner wall of the house (see above)	568

1006062

TABLE 3

ACTIVITIES OF THE MEMBERS OF THE FAMILY

MONTH	EEP	MCE	MEEC	AIG	JEI
1 Tuesday		Type Day G ₂	Type Day (excep)	Type Day (excep)	Type Day (excep)
2 Wednesday		Went to Clinic No. 19 Day type (excep)	Went to Clinic No. 19 Day type (excep)	Normal Day type B ₄	Day type F ₅
3 Thursday		Rest day Day type J ₂	Normal Day type A ₃	Market Day type (excep)	Normal Day type B ₅
4 Friday		Normal Day type D ₂	Normal Day type A ₃	Normal Day type B ₄	Normal Day type B ₅
5 Saturday	DIED ON APRIL 29	Movie Market Day type I ₂	Movie Market Day type (MCE) I ₃	Movie Market Day type (excep)	Movie Day type E ₅
6 Sunday		Visiting Day type H ₂	Visiting Day type L ₃	Visiting Day type B ₄	Visiting Day type E ₅
7 Monday		Normal Day type D ₂	Normal Day type A ₃	Normal Day type B ₄	Normal Day type B ₅
8 Tuesday		Normal Day type D ₂	Normal Day type A ₃	Normal Day type B ₄	Normal Day type B ₅

1006063

TABLE 4

EXAMPLE OF THE DOSE CALCULATION FOR A TYPE DAY FOR PATIENT M.C.E.

Timetable	Area	Activities for the day and observations	
5:30 - 9.00	6a	Slept	
9:00 - 9.30	2a	Breakfast	
9:30 - 11:30	5b	Washed laundry	
11:30 - 13:00	5a	Rested	
13:00 - 15:00	. .	Market or went shopping	
15:00 - 16:00	2	Ate	
16:00 - 18:00	6a	Rested	
18:00 - 18:30	2	Went with her husband to eat	
18:30 - 19:00	5a	Rested and helped her mother-in-law	
19:00 - 21:00	3	Ironed	
21:00 - 21:30	2	Dined	
21:30 - 22:00	3	Talked with mother-in-law	
22:00 - 5:30	6a	Slept	
.08	1	Near the cabinet	
<hr/>			
Total time in the respective areas (h)		Doses in the respective areas (mr)	Total dose for the day (mr)
2.00 in Z_2		12,028	
0.50 in Z_{2a}		2,615	
2.50 in Z_3		5,875	
2.00 in Z_{5a}		1,240	
2.00 in Z_{5b}		628	
13.00 in Z_{6a}		2,704	
.08 in Z_1		3,006	28,096 mr

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TABLE 5
CALCULATION OF THE MAXIMUM EXPOSURE DOSES FOR PERSON M.C.E.

Month	DOSES (r) ON THE TYPE DAYS												Monthly Dose (r)		
	Type A ₂	Type B ₂	Type C ₂	Type D ₂	Type E ₂	Type F ₂	Type G ₂	Type H ₂	Type I ₂	Type J ₂	Type K ₂				
March	5 140.7							1 25.7							166.6
April	11 377.4	1 44.7	1 28.1	10 257.5	1 23.5	1 32.9	3 77.3	1 14.4	1 26.8						882.3
May		3 134.1	14 393.3		2 47.0	1 32.9	5 128.6	3 43.1	3 40.3						819.3
June		3 134.1	11 309.1		1 23.5		5 128.6		10 134.2						729.4
July							1 25.7		12 161.0	4 59.2					246.0

1006066

TABLE 6
 CALCULATION OF THE MAXIMUM EXPOSURE DOSE BY SCATTER RADIATION FOR PERSON M.C.E.

		LENGTH OF STAY IN THE KITCHEN AND BEDROOM DURING THE TYPE DAYS IN HOURS (h)												
Month	Type	Type A ₂	Type B ₂	Type C ₂	Type D ₂	Type E ₂	Type F ₂	Type G ₂	Type H ₂	Type I ₂	Type J ₂	Type K ₂	Total Time (h)	Scattered Dose (r)
March	C	18.75							4.58				23.33	2.9
	R	50.00							11.00				61.00	3.0
April	C	88.88	8.08	7.08	48.30	5.83	11.08	13.74	2.33	11.00			196.32	2.5
	R	123.75	9.00	13.00	100.00	10.75	7.00	33.00	8.25	30.00			334.75	16.7
May	C		24.24	99.12		11.66	11.08	21.40	6.99	16.50			190.99	23.9
	R		27.00	182.00		21.50	7.00	55.00	24.75	45.00			362.25	18.1
June	C		24.24	77.88		5.83		21.40	55.00	55.00			184.35	23.1
	R		27.00	143.00		10.75		55.00	150.00	150.00			385.75	19.3
July	C							4.58					116.58	14.6
	R							11.00					235.00	11.8

TABLE 7

Doses for Case 1

TOTAL MAXIMUM AND MINIMUM DOSES FOR PERSON E.E.P.

Month	Exposure doses to direct beam r (rad)		Exposure doses of scattered radiation r (rad)		Dose absorbed (rem)	
	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum
March	4,809.0 (4,712.8)	2,711.9 (2,657.7)	24.0 (23.0)	17.0 (16.3)	4,735.8	2,674.0
April	400.7 (392.7)	240.9 (236.1)	38.6 (37.0)	31.3 (30.1)	429.7	266.2
May						
June						
July						
GRAND TOTAL	5,105.1 (rad)	2,893.8	60.1 (rad)	46.4	5,165.5 (rem)	2,940.2
7 days of exposure before 1-IV-62					Daily dose: maximum 676.54 rem minimum 382.00 rem	
17 days of exposure from 1-IV-62					Daily dose: maximum 25.27 rem minimum 15.65 rem	

1006067

TABLE 8

Doses for Case 2

TOTAL MAXIMUM AND MINIMUM DOSES FOR PERSON M.C.E.

Month	Exposure dose to direct beam r (rad)		Exposure dose to scattered radiation (rad)		Dose absorbed (rem)	
	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum
March	166.5 (163.2)	116.5 (114.1)	5.9 (5.6)	4.9 (4.7)	168.8	118.8
April	882.2 (864.5)	601.7 (589.6)	41.3 (40.0)	35.1 (33.7)	904.5	623.3
May	819.2 (803.0)	516.2 (505.8)	42.0 (40.3)	32.5 (31.2)	843.3	537.0
June	729.3 (714.7)	444.1 (435.2)	42.3 (40.6)	32.3 (31.0)	755.3	466.2
July	246.0 (241.1)	237.4 (232.6)	26.3 (25.2)	18.7 (17.9)	266.3	250.5
GRAND TOTALS	2,786.5 (rad) 1,877.3		151.7 (rad)	118.5	2,938.2 (rem) 1,995.8	
115 days of exposure			Daily dose: maximum 25.5 rem; minimum 17.3 rem			

1006068

TABLE 9

Doses for Case 3

TOTAL MAXIMUM AND MINIMUM DOSES FOR PERSON M.E.E.C.

Month	Exposure dose to direct beam r (rad)		Exposure dose to scattered radiation r (rad)		Absorbed dose (rem)	
	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum
March	628.7 (616.1)	319.2 (312.8)	13.8 (13.2)	9.7 (9.3)	629.3	322.2
April	176.8 (173.3)	147.9 (144.9)	16.6 (15.9)	10.6 (10.2)	189.2	155.1
May	365.8 (358.5)	304.8 (298.7)	32.3 (31.0)	28.9 (27.8)	389.5	326.5
June	354.5 (347.4)	295.9 (290.0)	31.7 (30.5)	28.6 (27.5)	377.9	317.5
July	272.4 (266.9)	231.0 (226.4)	19.9 (19.0)	26.3 (25.3)	286.0	251.7
GRAND TOTALS	1,762.2 (rad)	1,272.8	109.7 (rad)	100.1	1,871.9 (rem)	1,373.0
7 days of exposure before 1-IV-62		Daily dose: maximum 89.9 rem; minimum 46.0 rem				
92 days of exposure since 1-IV-62		Daily dose: maximum 13.5 rem; minimum 11.4 rem				

1006069

TABLE 10

Doses for Case 4

TOTAL MAXIMUM AND MINIMUM DOSES FOR PERSON A.I.G.

Month	Exposure dose to direct beam ^r (rad)		Exposure dose to scattered radiation ^r (rad)		Absorbed dose (rem)	
	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum
March						
April	193.9 (190.2)	138.9 (136.1)	10.4 (9.9)	7.3 (7.0)	200.1	143.1
May	973.2 (953.7)	622.8 (610.3)	52.1 (50.0)	43.3 (41.5)	1,003.7	651.8
June	965.0 (945.0)	599.6 (587.6)	46.3 (44.4)	39.7 (38.1)	989.4	625.7
July	689.5 (675.7)	381.7 (374.1)	29.9 (28.7)	24.7 (23.7)	704.4	397.8
GRAND TOTALS	2,764.6 (rad)	1,708.1	133.1 (rad)	110.4	2,897.2 (rem)	1,818.5
90 days of exposure		Daily dose: Maximum 32.2 rem; minimum 20.2 rem				

1006070

TABLE 11

Doses for Case 5

TOTAL MAXIMUM AND MINIMUM DOSES FOR PERSON J.E.I.

Month	Exposure dose to direct beam r (rad)		Exposure dose to scattered radiation r (rad)		Absorbed dose (rem)	
	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum
March	70.2 (68.8)	63.7 (62.4)	3.2 (3.1)	3.1 (2.9)	71.9	65.3
April	358.0 (351.0)	201.0 (197.9)	19.9 (19.1)	16.9 (16.2)	370.1	214.1
May	496.0 (481.1)	268.2 (262.8)	23.6 (22.6)	21.4 (20.5)	503.7	283.3
June	457.5 (448.3)	242.8 (238.0)	21.6 (20.7)	19.6 (18.8)	469.0	256.8
July	294.2 (288.3)	155.8 (152.7)	14.0 (13.4)	12.6 (12.1)	302.0	164.8
GRAND TOTALS	1,637.5 (rad)	913.8	78.9 (rad)	70.5	1,716.7 (rem)	984.3
106 days of exposure		Daily dose: Maximum 16.2 rem; minimum 9.3 rem				

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TABLE 12

HEMATOLOGIC DATA ON THIS PATIENT [A.I.G.]

Date	Hemoglobin g %	Leuc. per mm ³	N % Absolute	Lymph % Abs.	Platelets per mm ³	Blood Trans.	Cort. mg	And. mg
13-VIII-62	4.9	2,000	720	880				
18-VIII						1,200	P-30	
20-VIII	10.6	1,300	410	702			80	75
21-VIII	10.0	1,700	646	843			80	75
23-VIII	9.4	3,100					80	75
25-VIII	9.1	4,950	1,574	2,970			80	75
28-VIII	11.0	3,200	1,312	1,728		500	80	75
30-VIII	10.3	3,300	1,607	1,452	30,000		40	75
1-IX	10.6	3,400	1,564	1,564			40	75
4-IX	10.0	2,800					40	75
6-IX	9.8	3,200			35,000		40	75
8-IX	9.1	2,300	897	1,242			40	75
11-IX	8.6	1,440	1,440	520			40	75
15-IX	8.8	2,200			5,000		40	75
17-IX	8.0	2,250			6,000		40	75
18-IX	9.1	2,700	1,431	1,053	4,000		40	75
20-IX	7.9	2,450					40	75
26-IX	7.5	2,200					40	75
1-X	12.3	2,700	1,134	1,404	12,000		40	75
3-X	10.6	2,250	1,125	1,012	4,000	1,000	40	75
13-X	10.6	2,600			2,000		40	75

HB = Hemoglobin g %; Leuc. = leucocytes per mm³; N% Abs. = Neutrophils, absolute numbers; Lymph % abs. = Lymphocytes - absolute numbers; Platelets per mm³; Blood trans. = Blood transfusions; Cort. = Corticosteroids; And. = synthetic androgens.

TABLE 13
HEMATOLOGIC DATA FOR CASE 5 (J.E.I., Survivor)

Date	HB g %	Leucocytes per mm ³	N % Abs.	Lymphocytes % Abs.	Platelets per mm ³
21-VIII-62	14.0	2,200	1,452	132	190,000
23-VIII	14.6	4,150			
24-VIII	14.2	5,500			
28-VIII	14.6	3,200	1,824	1,280	
30-VIII	15.8	7,200	5,164	1,728	210,000
4-IX	15.6	4,900	3,577	1,275	235,000
6-IX	14.6	4,000			
13-IX	15.8	3,400			70,000
26-IX	15.3	2,900			126,000
1-X	14.2	3,100	1,953	961	110,000
6-X	13.4	3,200			172,000
13-X	14.6	3,800			
18-X	13.0	4,800	2,880	1,584	
23-X	13.8	2,450			
29-X	14.2	4,500	2,160	1,935	108,000
12-XI	14.6	4,100	2,050	1,681	102,000
23-XI	13.8	5,100	2,907	1,785	101,400
13-XII	15.3	5,400			190,000
19-XII	15.3	4,800			

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