AAPM REPORT NO. 50

Fetal Dose from Radiotherapy with Photon Beams

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REPORT OF AAPM RADIATION
THERAPY COMMITTEE TASK GROUP 36

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Fetal dose from radiotherapy with photon beams: Report of AAPM Radiation Therapy Committee Task Group No. 36

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Approximately 4000 women per year in the United States require radiotherapy during pregnancy. This report presents data and techniques that allow the medical physicist to estimate the radiation dose the fetus will receive and to reduce this dose with appropriate shielding. Out-of-beam data are presented for a variety of photon beams, including cobalt-60 gamma rays and x rays from 4 to 18 MV. Designs for simple and inexpensive to more complex and expensive types of shielding equipment are described. Clinical examples show that proper shielding can reduce the radiation dose to the fetus by 50%. In addition, a review of the biological aspects of irradiation enables estimates of the risks of lethality, growth retardation, mental retardation, malformation, sterility, cancer induction, and genetic defects to the fetus.

Key words: radiation therapy, fetus

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I. INTRODUCTION

Each year in the United States approximately 4000 pregnant women require treatment for a malignancy. The most common tumors are lymphomas, leukemias, melanomas, and tu-

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mors located in the breast, uterine cervix, and thyroid. Radiation therapy is often a treatment of choice for these patients.

Each pregnant patient presents a unique set of circumstances that the physician must evaluate before deciding whether and how to treat her. Ideally, the chosen treatment should control the tumor and give the fetus the best chance for a normal life. The challenge is to achieve the optimum balance between the risk and the benefit.

This report describes techniques and presents data that can aid the medical physicist who needs to plan and execute the radiation therapy of a pregnant patient using photon beams. The out-of-beam data presented in this report were measured by members of this task group to provide dose estimates that are both consistent and comparable. In general, these collected data agree well with other published data. Electron beam therapy is not considered specifically, even though the same type of measurements and shields could be used for these treatments. Brachytherapy is not discussed because shielding usually is not an option and the dose to a fetus can be estimated by calculation.

This report also summarizes the biological effects of fetal irradiation. The effects of radiation on the fetus are not fully understood and cannot be predicted with certainty in a particular case. However, the severity and frequency of adverse effects increase with total dose. Because it is impossible to eliminate all radiation to the fetus during radiation therapy, the best advice is to plan the treatment regimen to reduce the dose to the fetus as low as is reasonably achievable, thereby reducing the potential risk.

Adequate shielding of the fetus during radiation therapy requires a commitment by the medical physicist and the radiation oncologist to have the needed resources. Treating pregnant patients requires advanced consultation among the patient's radiation oncologist, medical oncologist, obstetrician, and medical physicist. This planning procedure often results in construction of equipment not available commercially. Even in large institutions, radiation therapy departments may see only one or two pregnant patients per year. An efficient allocation of resources may be for all pregnant patients in a geographic region to receive radiation therapy at one institution. For other institutions, the best management of a pregnant patient would be referral to an institution where she can receive treatment under optimal conditions.

II. PHYSICAL BASIS OF DOSE OUTSIDE PHOTON BEAMS

The principal sources of dose outside a treated volume are (1) photon leakage through the treatment head of the machine, (2) radiation scattered from the collimators and beam modifiers, and (3) radiation scattered within the patient from the treatment beams. For higher-energy (>10 MV) photon beams there is an additional contribution from neutrons emanating from the treatment head, neutrons produced from photoneutron interactions in the patient, and radioactive isotopes produced in photoneutron interactions.

The relative contributions of collimator scatter, head leakage, and patient scatter to peripheral dose have been investigated.[1-7] Fraass and van de Geijn[1] found that collimator scatter plus leakage is of the same order of magnitude as patient scatter and that for some machines the leakage component can vary by a factor of 2, depending on the collimator angle. Kase *et al.*[4] also indicate that near the beam the collimator scatter contributes 20% to 40% of the total peripheral dose and that leakage becomes the main contributor at greater distances from the field edge. Greene *et al.*[3] found that collimator scatter was the dominant component of the peripheral dose. As discussed by Fraass and van de Geijn,[1] it is important to know the magnitude of the leakage and the components of collimator scatter that contribute to the dose outside the field because these components can be reduced by placing a lead shield over the critical area.

The use of wedges and other beam modifiers can increase the peripheral dose. Published data[6-8] show that wedges increase the dose near the beam by a factor of 2 to 4. The use of lead shielding devices can increase the peripheral dose by a factor of 2 to 5.[7,8] As with head leakage and collimator scatter, these components can be reduced by shielding the critical area.

A. Total dose outside beams

Figures A1 through A7 in Appendix A show measured values of the peripheral dose-the dose outside of treatment beams-in tissue-equivalent phantoms (water or polystyrene) for several radiation therapy machines. (These measurements do not include contribution from photoneutrons, as discussed in Sec. II B below.) Measurements were made without special shielding or blocking devices. The beams include ^{60}Co gamma rays, and 4-, 6-, 10-, 18-, and 25-MV x rays for field sizes from 5X5 to 25X25 cm^2 at depths from 2 to 15 cm. These and other published data[1-5,7] are summarized below.

The most important determinant of the peripheral dose is the distance from the radiation field edge, with the dose decreasing approximately exponentially with distance from the field edge. In Fig. A1, our measurements for a given depth and field size show that the peripheral dose for photons from 4 to 25 MV is the same order of magnitude and is qualitatively similar. In contrast, the peripheral dose from ^{60}Co at distances greater than 10 cm from the field edge is considerably higher because of a larger amount of head leakage. Several sets of measured data[1,4,6] agree with our data in Appendix A. However, peripheral doses calculated by Keller *et al.*[5] show that the total dose outside the beam decreases as energy increases.

Most published data[1,4,6] show that the change in the peripheral dose with depth is small. Our data also show a small change in dose with depth: e.g., Figure A2 shows similar doses at depths ranging from 2 to 15 cm for a 6-MV beam with a field size of 10X10 cm^2. However, other published data[9] show a greater change in dose at depth for a ^{60}Co unit.

The peripheral dose increases as field size increases; this effect is more pronounced closer to the beam edge and is due to the scatter within the patient from the treatment beam. Figures A3 through A1 show the change of dose with field size for 4-, 6-, 10-, 18-, and 25-MV photon beams; similar data were reported previously.[1,4]

B. Photoneutron contamination

As mentioned above, incidental neutrons are produced by linear accelerators where photons are generated by electrons with energies greater than 10 MeV. The walls of the waveguide, the x-ray target, filters, collimators, and the patient are all potential sources of photoneutrons.[10-12] In and near the treatment beam, the contribution of neutrons to the total dose is small; at greater distances from the beam, the total dose is much smaller but the percent of neutrons in the total dose may be as high as 40%. The contribution of photoneutrons to total dose increases as the megavoltage is increased from 10 to 20 MV but remains approximately constant above 20 MV. Although the relative biological equivalence of neutrons is controversial, there are radiobiological data that suggest that the quality factor for late effects may be as high as 20.[13]

The National Council on Radiation Protection[14] considers the risk of long-term biological effects of incidental neutrons from linear accelerators to be negligible for most patients. Data that deal specifically with the risk to the fetus do not exist; however, it is prudent to treat a pregnant patient with photons generated by electrons less than 10 MeV if this modality is adequate to treat the tumor.

In summary, the dose outside a beam is a function of the distance from the beam edge and the field size and depends on primary radiation energy and depth within the patient. Although variation exists among machines, the major components of the out-of-beam dose within 10 cm of the beam edge are typically scatter off the collimator and scatter from the useful beam within the patient. In the region 10 to 20 cm from the field edge, collimator scatter decreases so that the major component of the out-of-beam dose is scatter within the patient; however, collimator scatter and head leakage also contribute to the dose. At about 30 cm, scatter in the patient and head leakage are approximately equal, and beyond that point, head leakage predominates. Scatter from special blocking devices, such as wedge filters, increases the dose near the beam edge by a factor of 2 to 5. Measurements to separate the dose components in a formal way are not necessary, but the medical physicist should be aware that the out-of-beam dose includes radiation from several sources, some that can be reduced by shielding (head leakage and collimator scatter) and some that cannot (scatter within the patient).

III. TECHNIQUES TO ESTIMATE AND REDUCE FETAL DOSE

Planning of radiation therapy should be based on estimates of the size and location of the fetus at the beginning of treatment as well as the expected change during the course of therapy. Points of dose estimation should be selected that will reflect the range of dose throughout the fetus. Three points commonly used are the fundus, symphysis pubis, and umbilicus (or midpoint between the fundus and symphysis pubis), as shown in Fig. 1. Other points may be of clinical interest depending on the orientation and size of the fetus. Figure 2 shows the height of the fundus during various stages of gestation.

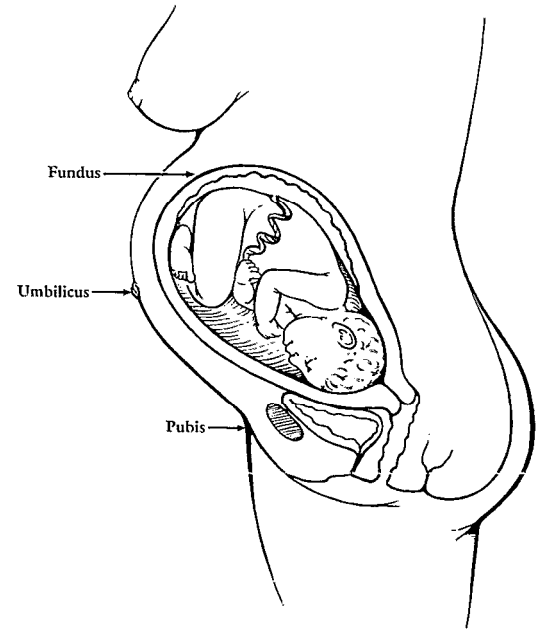

FIG. 1. Points of dose estimation: the fundus. umbilicus (or midpoint). and symphysis pubis.

The dose to the fetus can be reduced in two ways: by modifying the radiation therapy technique and by using lead shielding devices.

A. Modification of treatment techniques

The first and simplest step in reducing dose to the fetus is for the medical physicist and radiation oncologist to modify the usual treatment technique, by changing field angles, reducing the field size, choosing a different radiation energy, etc. The peripheral dose can be reduced further by treating the patient so that the lower collimator defines the field edge nearest the fetus. Also, trimmers should be in the lowest position where this is an option.

In addition, it is common practice to increase the field length during double-exposure portal filming. Because it is desirable to maintain fetal dose at as low a level as possible, it is important not to expose sensitive areas when performing this technique.

B. Use of special shields

1. Design of shields

The design of any shielding device must allow for treatment with anterior, posterior, and lateral fields, above the diaphragm and on the lower extremities. Constraints due to weight usually make it more difficult to treat with oblique fields while using special shielding.

Safety is the overriding consideration in the selection and design of equipment to reduce the dose to the fetus. Shielding, by necessity, involves the use of heavy materials and the medical physicist should carefully plan all aspects of the equipment to eliminate the possibility of injury to patients or

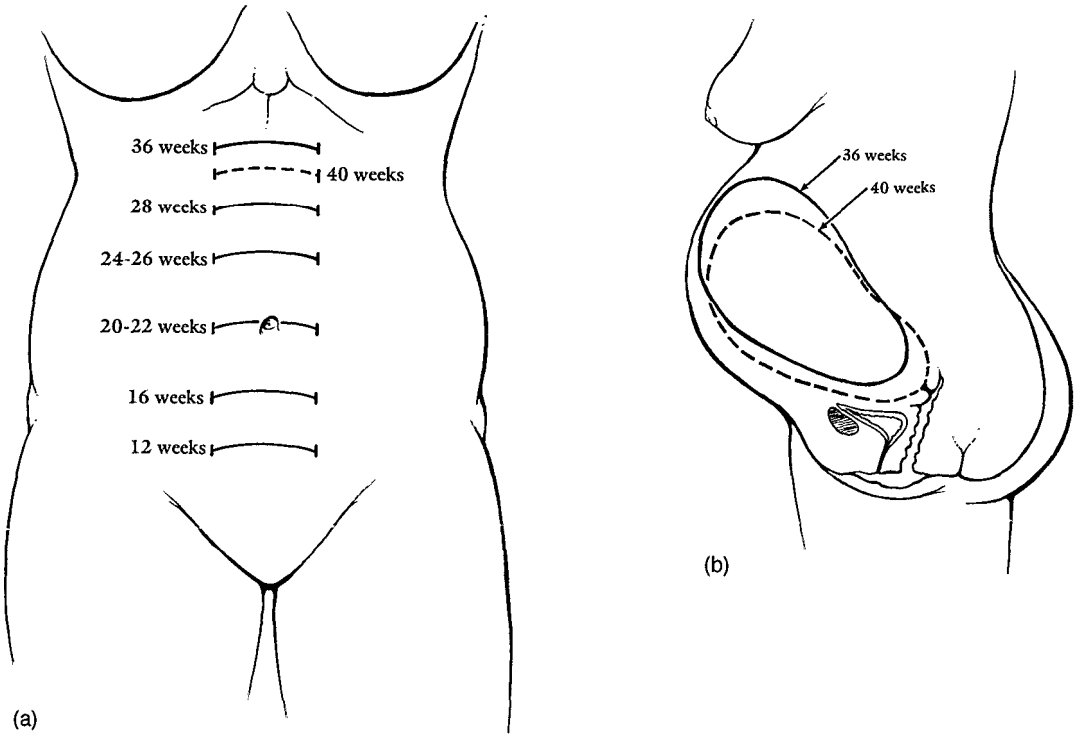

FIG. 2. (a) Anterior and (b) lateral views of height of the fundus at various times during pregnancy.

personnel. Methods of supporting the shielding material and the number of times and ways in which shields will be moved are important considerations.

Three types of shielding arrangements are described below, in terms of their complexity, ease of use, cost, and usefulness with other treatment aids.

a. Bridge over patient. The simplest shielding design consists of a bridge over the patient's abdomen that supports four to five half-value layers of lead (Fig. 3).[15] (Four to five half-value layers of lead is approximately 5 to 7 cm of lead or 6 to 8.5 cm of Cerrobend.) This type of shielding is shown in Fig. 4, with a phantom in position for measurement. Schematic diagrams of the shield are shown in Figs. 5 and 6. (Engineering drawings are available from The University of Texas M. D. Anderson Cancer Center, Department of Radiation Physics-544, 1515 Holcombe Blvd., Houston, TX 77030.)

For treatment of an anterior field, the patient lies in a supine position with the bridge and lead placed over the abdomen. To treat a mantle field, the superior edge of the lead is placed approximately 2 cm lower than the lower edge of the field to decrease the contribution of head leakage and collimator scatter. For the treatment of a posterior field, the

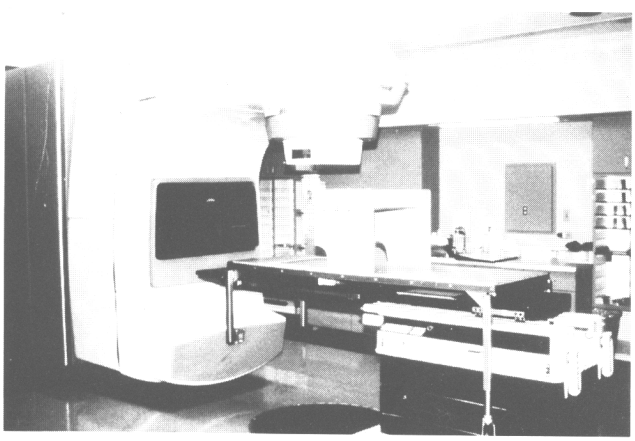

FIG. 3. Photograph of a bridge used to support shielding material.

FIG. 4. Photograph of shielding with phantom used for measurements.

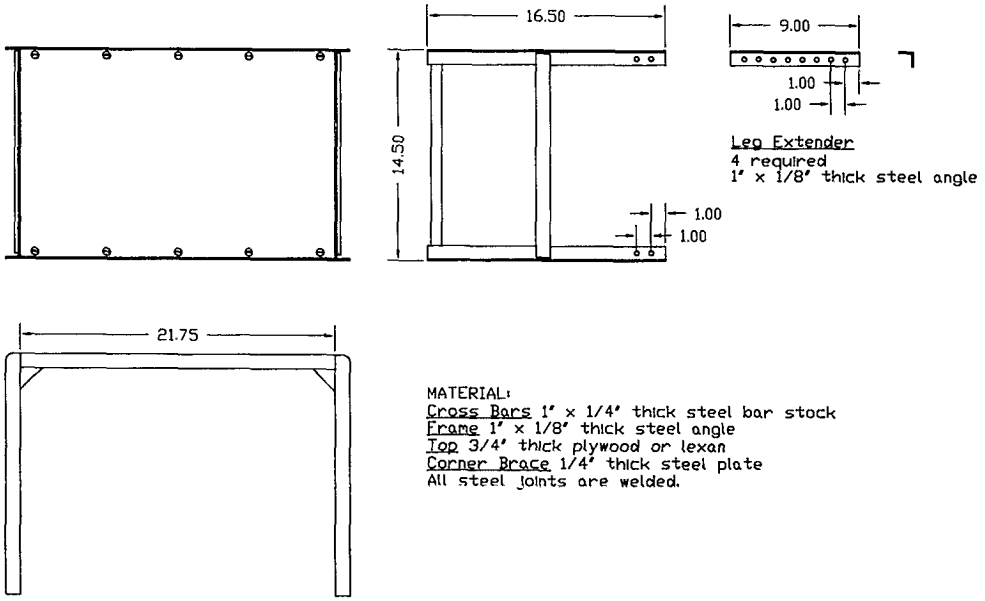

FIG. 5. Diagram, including measurements, of a bridge used to support shielding material.

patient lies prone on a false table top, with the bridge and lead placed over her.

The bridge shield can be constructed easily within a radiation oncology department at a modest cost of a few hundred dollars. One must take care to place the bridge so that it cannot slide off the edge of the couch or treatment table.

b. Table over treatment couch. Another shield design consists of a single unit that protects the fetus during treatment with anterior, posterior, and lateral fields. Typically, the single unit consists of a table that rests upon the treatment couch. A Mylar opening enables the treatment of anterior and posterior fields. The unit has a reinforced wooden bridge upon which lead can be placed on the top or sides, depending on field orientations (Fig. 7). Schematic diagrams are shown in Fig. 8. The bridge must be wide enough to straddle the entire abdominal area. Posterior fields are treated by rotating the gantry and raising the treatment couch. Because posterior fields can be treated with this design, lead must be placed

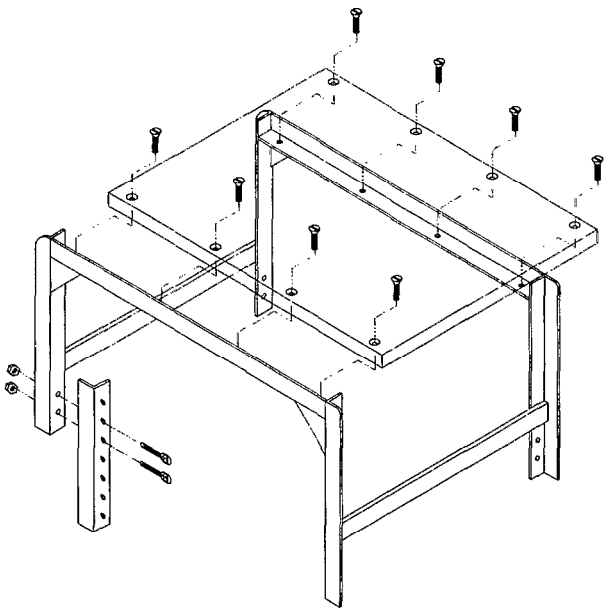

FIG. 6. Diagram of a bridge used to support shielding material.

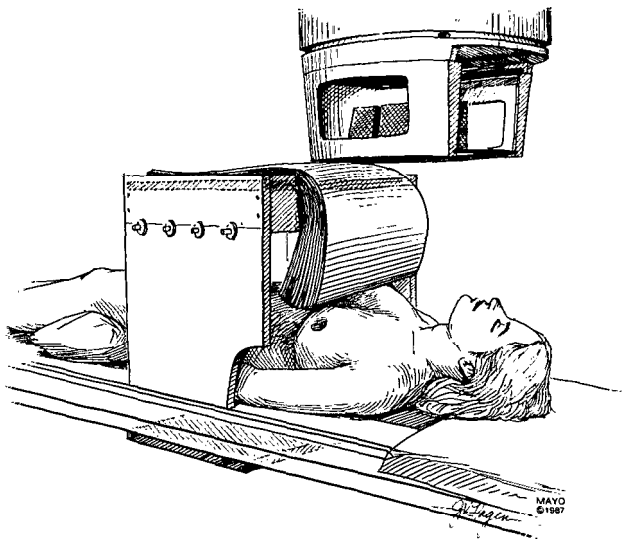

FIG. 7. Single-unit bridge, shown in Fig. 5, with patient in treatment position.

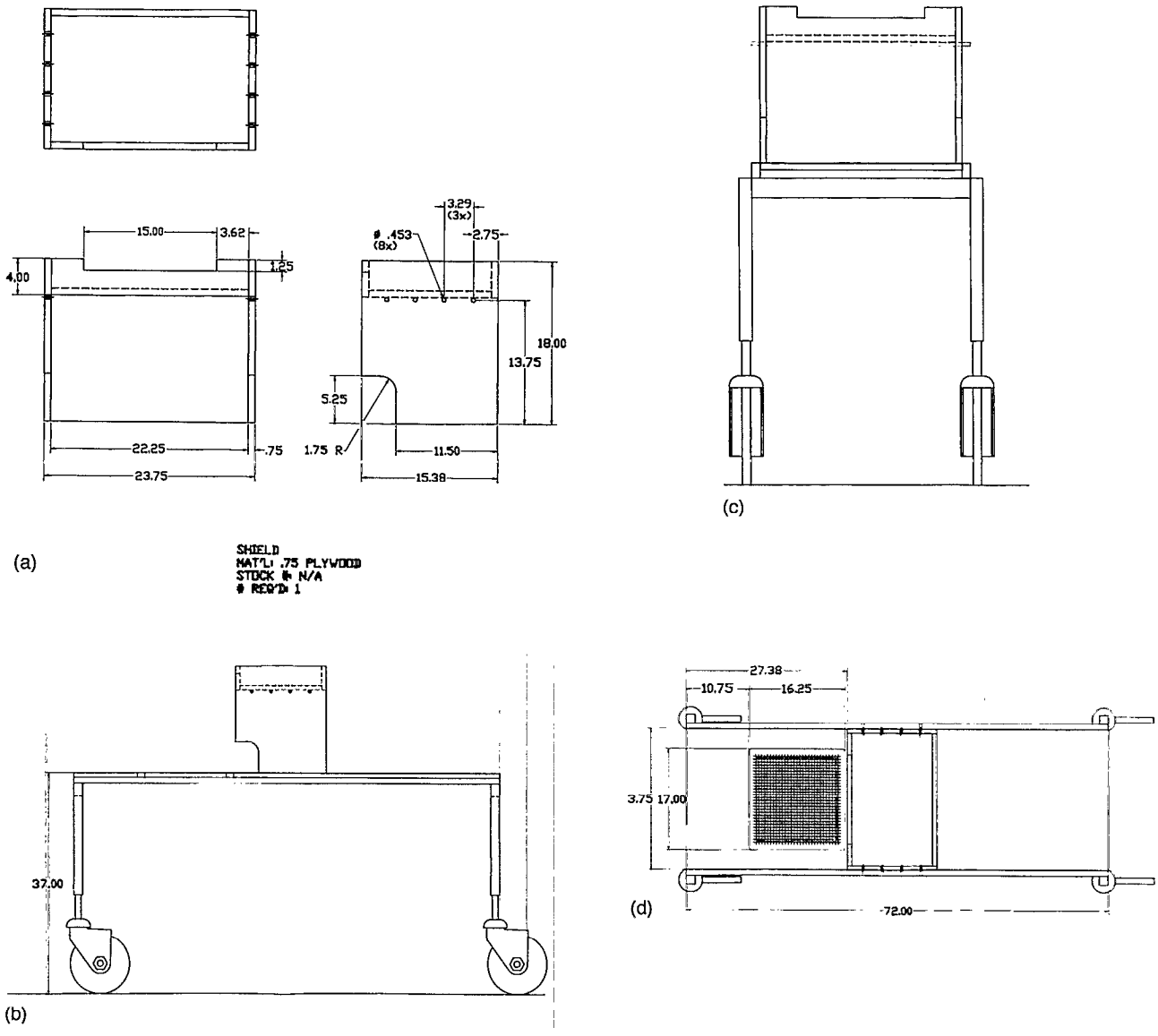

FIG. 8. (a) Diagram of a bridge used to support shielding material; (b) side view, (c) end view, and (d) top view of bridge on a cart used to transport it.

beneath the abdomen by means of a small shelf that contains lead sheets and is attached to the bottom of the table. Clinical use of this shielding has been published.[16]

The single-unit shield can be constructed for about $1000, with the largest expense being the purchase of a table that will straddle the treatment couch. The wooden bridge and lead can be easily added to the purchased table. The principal advantage of this design is that the patient can remain in the supine position for treatment of anterior, posterior, and lateral fields. The main disadvantage to this design is its weight. Typical use of this unit requires placing approximately 4 cm of lead over and beneath the abdomen in addition to the 4-cm lead block suspended vertically to reduce collimator scatter. The total weight of the table and lead, excluding the weight of the patient, may be approximately 200 kg, which exceeds the design limits (typically between 125 and 180 kg) of most commercially available treatment tables. As a result, additional support for the treatment couch should be used to ensure that no movement occurs during treatment. Another disadvantage of this shield is that the lead must be positioned properly each day, which increases the possibility of injury to the patient or technologist.

c. Mobile shields. Another design consists of single mobile shields for the anterior and posterior fields. A single anterior shield can be constructed such that it does not rest upon the existing treatment couch. Photographs of this shield are shown in Figs. 9 and 10 with a schematic diagram shown in Fig. 11. (Engineering drawings are available from the Mayo Clinic, Department of Therapeutic Radiology, 200 1st St., SW, Rochester, MN 55905.) The shield must be vertically adjustable (motorized) to allow for treatment at source to skin distances of 80-125 cm and must be easily movable by the staff. The weight of a unit with 4-cm lead blocks on the top, front, and sides will be approximately 200 kg.

At a cost of approximately $2500, this design is more expensive than the previous designs and requires substantial time for a machine shop to construct. The greatest advantage of this design is the ease with which the shield can be posi-

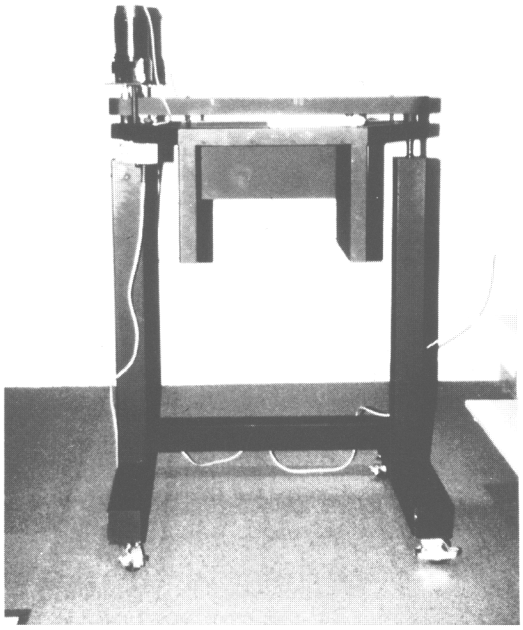

FIG. 9. Photograph of mobile support to hold shielding material independent of treatment couch.

tioned over the patient without having the weight of the shield on the treatment couch or requiring personnel to lift the lead blocks above the patient. In addition, this design is suitable for shielding critical organs during treatment of other patients.

For the treatment of posterior fields, a single posterior shield must fit between the existing treatment couch and head of the treatment machine, be vertically adjustable, and be easily moved and attached by personnel. The shield can be stored on a wheeled cart which is aligned with the treatment couch and then the shield is attached to the side rails of the couch and lifted from the cart. Bearings allow for easy positioning anywhere along the couch side rails. In addition, proper abdominal shielding may require angulation of the lead shield. A photograph is shown in Fig. 12, with schematic diagrams in Figs. 13(a) and 13(b). (Engineering drawings are available from the Mayo Clinic, Department of Therapeutic Radiology, 200 1st St., SW, Rochester, MN 55905.)

FIG. 10. Photograph of mobile shield positioned over treatment couch.

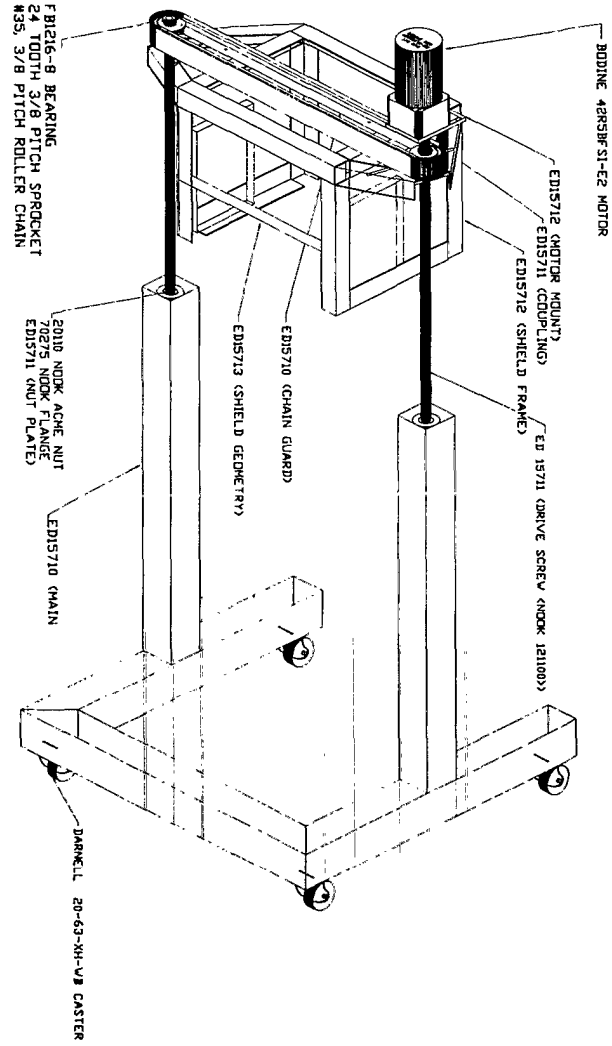

FIG. 11. Diagram of mobile support to bold shielding material independent of treatment couch.

2. Dosimetry with shields

The medical physicist is responsible for making the measurements necessary to estimate fetal dose before treatment. As a practical approach, the physicist should first estimate the dose to the fetus without special shielding. The total dose outside a beam can be measured in a phantom (water, polystyrene, or anthropomorphic) using an ionization chamber, diodes, or thermoluminescent dosimeters (TLDs). The phantom should simulate full-scatter geometry. The unshielded dose to the fetus can then be reduced with special shields as

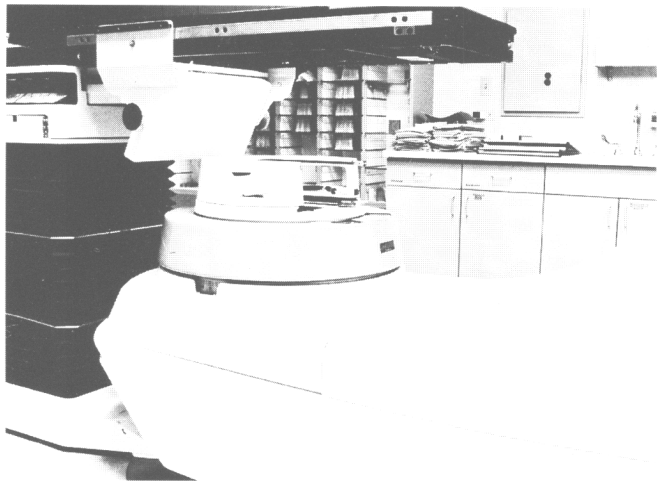

FIG. 12. Photograph of mobile support to hold shielding material.

discussed in the following section. Normally, the physicist would use the maximum amount of reasonable shielding, usually four to five half-value layers of lead.

Out-of-beam data and patient examples in this report can be used as guidelines but should not be used as the only means of estimating the fetal dose. Points of measurement should be sufficient to estimate the range of dose to the fetus and should include at least the fundus, symphysis pubis, and umbilicus (or midpoint) (Fig. 1). During treatment, dosimeters can be placed at these three points on the surface of the patient with appropriate build-up material to monitor fetal dose. Because the fetal dose cannot be measured directly, measurements on the surface of the pregnant patient compared with the same points on the phantom help to ensure that the phantom measurements are valid for the patient's treatment. Dosimeters on the patient also monitor the accuracy of the shield's placement each day.

The physician needs information regarding the fetal dose as related to gestational period. As the pregnancy progresses, the height of the fundus uteri increases with respect to the symphysis pubis (Fig. 2). Because wide variations may be expected in the location of the umbilicus, this point should be used only as a rough guide. However, the fundus and the symphysis pubis do delineate the extremes of the fetal position.

Before beginning any measurements it is important to gather as much information as possible with respect to the clinical treatment geometry to be used. In particular, SSD, field size, and the specific blocking to be used will aid in providing realistic dose estimations. In addition, measurements should be made with and without shielding; both sets of data demonstrate the effectiveness of the shielding.

Several phantom arrangements can be used to simulate the patient's treatment. An anthropomorphic phantom simulates the patient more correctly in regard to shape and anatomy. Diodes or TLDs may be placed at selected points in and out of the region representing the fetus. The upper torso of an anthropomorphic phantom coupled with a small-sized water or solid phantom that accommodates dosimeters or a

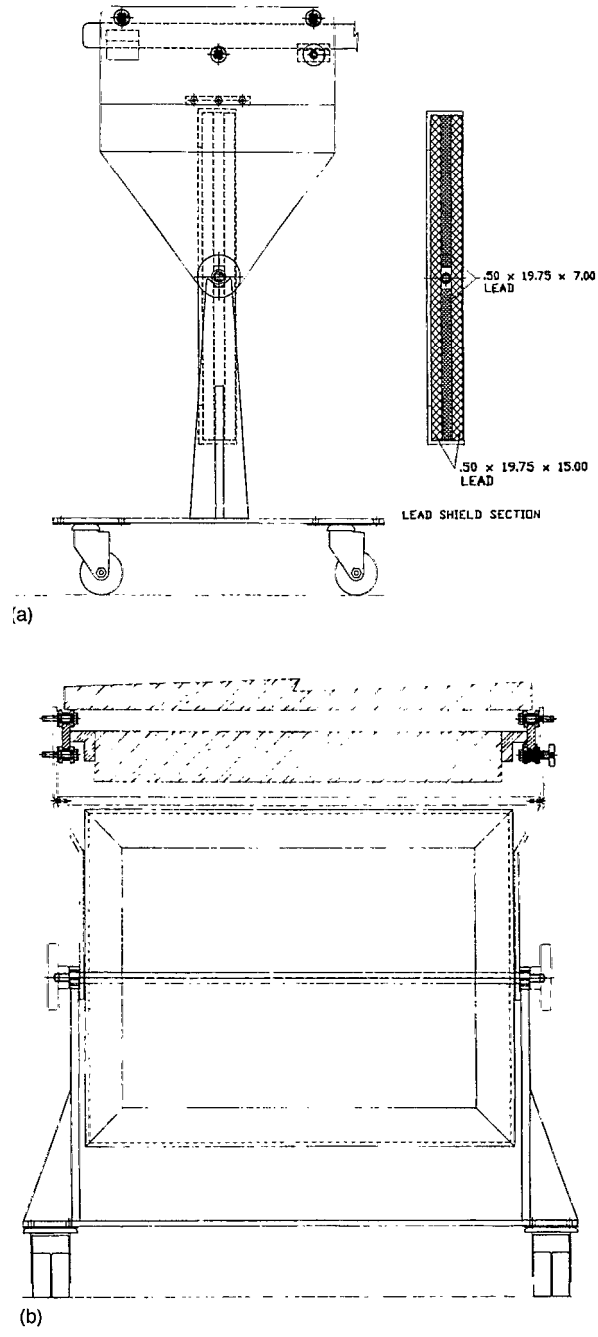

FIG. 13. Diagram of mobile support for shield with (a) support resting on trolley, (b) treatment table down and shields attached to sides of treatment table.

full-sized water phantom that can accommodate a larger variety of detectors can also be used for the simulation.

TLDs or other dosimeters may be used for both phantom and *in vivo* measurements.[17] However, the medical physicist needs to ensure that the chosen dosimeters can measure low doses with accuracy; this is particularly important for *in vivo* measurements because daily doses are very small. More sensitive dosimeters are needed to measure smaller doses in the phantom. In addition, if treatment is to be given with high-energy (>10 MV) photons that give rise to neutron production, lithium fluoride may produce spurious results, by being

TABLE I. Example 1: Pregnant patient with sarcoma in the left distal tibia.

Planned radiotherapy
Machine: Varian Clinac 600C, 6-MV photons at 100-cm target skin distance
Field configuration: Anterior and posterior, 7wX15h cm^2
Prescribed doses: 27.5 Gy to each field and 50 Gy to tumor
Therapy to be given over 5 weeks
Gestation stage at the beginning of therapy: 25 weeks
Shielding: None

Dose (Gy) to fetus for course of radiotherapy (10-cm depth)

	Point A Top of fetus	Point B Mid-fetus	Point C Pubis
Distance from nearest edge of field(s) to fetal points, cm	90	80	70
Dose to unshielded fetus	0.012	0.015	0.015
Dose to shielded fetus	******No shielding used******		

too sensitive to the neutron component. However, TLDs placed on the surface of a patient during treatment can monitor the fetal dose if the phantom irradiation includes the same surface points.

IV. EXAMPLES OF REDUCTION AND ESTIMATION OF FETAL DOSE

Tables I-III outline the radiation therapy of typical pregnant patients treated for three different cancers (sarcoma of the tibia. glioblastoma, and Hodgkin's disease). In each case, the fetal dose was measured in an anthropomorphic phantom using TLDs. These examples show that for most patients one can expect to reduce the fetal dose by 50% by using a reasonable amount of shielding.

V. BIOLOGICAL EFFECTS OF FETAL IRRADIATION

The principal effects of ionizing radiation on the mammalian embryo and fetus include lethal effects in the embryo, malformations, growth impairment. mental retardation. induction of malignancies, and hereditary defects. The frequency and magnitude of effects differ according to the absorbed dose, type of radiation, and gestational age at which exposure occurs, among other factors. Here the effects of irradiation are discussed in general terms and then are summarized for each of five major developmental phases at different human postconception (PC) time periods.

A. Radiation effects

1. Lethality

Little direct information exists about the lethal effects of radiation in early human pregnancy because of the uncertainty regarding the existence of a fertilized ovum during the first month following conception and the naturally high frequency of embryonic loss during the same time interval. Conclusions are drawn from experiments on animal cells and animals, *in vitro* and *in vivo,* particularly on rats and mice. Loss of viability is the main, if not the only effect, of irradiation during this period. Doses *in vitro* as low as 0.1 Gy may cause significant embryonic death at the time of maximum sensitivity before DNA synthesis begins in the fertilized cell.[18] The median lethal dose (LD_{50}) varies from 1 to 6 Gy through the mitotic cycle of the early mouse embryo.'" *In vivo* experiments in rats irradiated at 9 to 16 hours after mating showed a statistically significant increase in fetal

TABLE II. Example 2: Pregnant patient with glioblastoma.

Planned radiotherapy
Machine: Thcrac 6, 6-MV photons at 100-cm target skin distance
Field configuration: Right and left lateral, 17wX14h cm^2
Prescribed doses: 38 Gy to each field and 60 Gy (midline) to tumor
Therapy to be given over 6 weeks
Gestation stage at beginning of therapy: 13 weeks
Shielding: Three half-value layers, 4.5 cm lead blocks

Dose (Gy) to fetus for course of radiotherapy (10-cm depth)

	Point A Top of fetus	Point B Mid-fetus	Point C Pubis
Distance from nearest edge of field(s) to fetal points, cm	44	52	60
Dose to unshielded fetus	0.030	0.025	0.022
Dose to shielded fetus	0.015	0.013	0.011

TABLE III. Example 3: Pregnant patient with Hodgkin's disease.

Planned radiotherapy
Machine: Therac 6, 6-MV photons at 100-cm target skin distance
Field configuration: Anterior and posterior mantles
Prescribed doses: 40 Gy to anterior field, 13 Gy to posterior field, and 38 Gy to tumor
Therapy to be given over 6 weeks
Gestation stage at beginning of therapy: 34 weeks
Shielding: Five half-value layers, 6.7-cm lead blocks

Dose (Gy) to fetus for course of radiotherapy (10-cm depth)

	Point A Top of fetus	Point B Mid-fetus	Point C Pubis
Distance from nearest edge of field(s) to fetal points, cm	15.5	28.5	41.5
Dose to unshielded fetus	0.42	0.14	0.06
Dose to shielded fetus	0.17	0.04	0.02

mortality after exposure to 0.05 Gy, with earlier (embryonic) deaths observed after exposure to 0.10 to 0.25 Gy.[20] In these experiments the increased risk of embryo or fetal mortality was about 1.5% for doses on the order of 0.1 Gy, but there was no significant increase in the malformation rate.

Human data on fetal lethality primarily relate to pregnancy outcomes in women receiving large therapeutic radiation doses to the abdomen during the embryonic or organogenesis period (8 to 56 days PC). For example, 3.6 Gy[21] and 5 Gy[22] delivered during either period induced abortion in a large majority of cases.

2. Anatomical malformations

Neonatal malformations (congenital anomalies) occur sporadically in human and all other mammalian populations. The incidence varies among different populations and among social groups within populations. The principal cause is probably the incorrect interplay of many genetic factors. A widely accepted average incidence of malformation in live-born children throughout the world is 6%. An increased risk from exposure to ionizing radiation and many environmental teratogens has been observed, particularly during early organogenesis when organs are composed of a limited number of cells and are particularly vulnerable to damage that can adversely and irretrievably alter growth. The shape of the dose-response curve for these effects is unresolved and probably differs for different types of effects. Evidence suggests, however, that there is a threshold dose for many, if not all, developmental effects. Evidence, primarily from animal populations, shows that protracting the irradiation reduces the frequency of developmental effects.[23]

Most data on the malformation rate as a function of dose and dose rate relate to experimental animals, particularly mice. A comprehensive review appears in the 1986 report by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR).[24] Most dose-response curves relate to frequency and not to severity of the end point; severity is more difficult to quantify but generally increases with dose.[25] Dose-response curves are generally sigmoidal, and a threshold dose on the order of 0.1 Gy or greater may apply for most end points. Cell killing, which accounts for most malformations, is characterized by a shoulder in the cell survival curve for low linear energy-transfer radiation.[26] This shoulder represents a region of the dose-response curve in which incremental increases in dose have an increasingly greater effect. A typical dose-response curve illustrating this increasing effect is shown in the results of Tribukait and Cekan[27] (Fig. 14) for single doses of x rays delivered at 9 days PC in mice. The increase (statistically nonsignificant with $p > 0.05$) in the total number of malformations following doses of 0.125 Gy was 0.4% based on observations of 286 exposed and 291 nonexposed animals. The malformation rate doubled after doses of 0.4 Gy and rose steeply at higher doses, with 17% of animals malformed after exposure to 1 Gy.

Before World War II, human data on radiation-induced malformation concerned pregnancy outcomes in women receiving abdominal radiation therapy during the period of organogenesis. Human data differ from animal data in several respects. Malformation in humans frequently occurs after doses exceeding 0.5 Gy, but effects on the central nervous system (CNS), in particular small head size (SHS), dominate. (SHS is defined in the Japanese A-bomb survivor studies[28] as a head circumference that (a) in one or more examinations of children between the ages of 10 and 19 years was at least two standard deviations below the average for the age and sex of the patient in each city and (b) was on all previous and subsequent examinations at least one standard deviation below the average.) Brent" notes that in all reports of a morphologic malformation induced by radiation exposure in humans, the individual also exhibited either growth retardation or a CNS abnormality. This is particularly evident in Dekaban's review[30] of more than 200 published cases of women who received therapeutic pelvic irradiation, mostly in early pregnancy and usually with doses exceeding 2.5 Gy.

The people who were *in utero* during the atomic bombing (A-bombing) of Hiroshima and Nagaskai also have provided a major source of information on significant increases in the frequency of SHS following irradiation during the first half of pregnancy. The incidence of SHS was much higher in

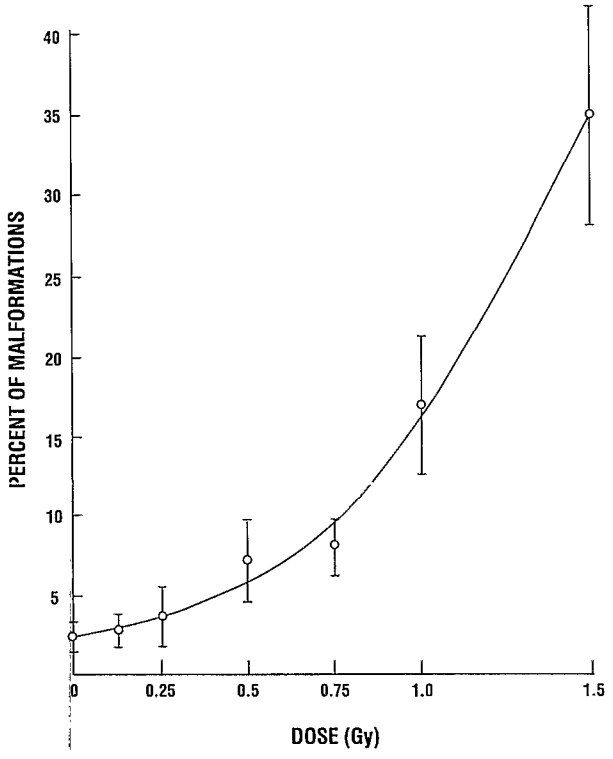

FIG. 14. Dose-response curve for all malformations in C3H mice following fetal irradiation at 9 days PC with 250 kV x rays. Error bars refer to ±1 standard deviation. (The curve is plotted and standard deviations calculated from data provided by Tribukait and Cekan, Ref. 37.)

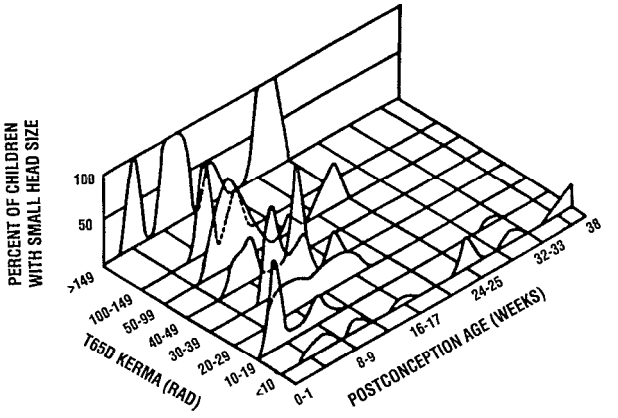

FIG. 15. Percent of children with small head size irradiated at various dose levels (kerma, T65D), during the period 2 to 11 weeks PC. During the period 2 to 11 weeks PC, incidence vs air kerma is approximately 100% for doses ≥ l.5 Gy, 21% for doses of 0.25 Gy, and 17% for doses of 0.15 Gy. (After Miller and Mulvihill, Ref. 31.)

Hiroshima, and Fig. 15 shows the distribution of observed cases by PC age and free-in-air kerma.[31] With the T65DR dosimetry system" used in this 1976 study, a significant excess in the risk of SHS occurred at kerma ranging between 0.10 and 0.19 Gy. The risk from radiation exposure is evidently greatest during the embryonic period, smaller during the second trimester, and even smaller during the third trimester of pregnancy. The incidence of SHS among the A-bomb survivors for all doses combined was 28% for those exposed between 4 and 13 weeks PC but only 7% for those exposed during the remainder of gestation.[31]

Otake and Schull[28] used the new DS86 dosimetry to re-evaluate A-bomb survivor data. The excess risk of SHS, with or without severe mental retardation (SMR), was analyzed by PC age at exposure and by estimated uterine-absorbed doses (not dose equivalent). As children, these people were classified as severely mentally retarded if they were unable to perform simple calculations, to make simple conversation, to care for themselves, or if they were completely unmanageable or had to be institutionalized.[28] For irradiation at PC ages of 0 to 7 weeks and 8 to 15 weeks, the incidence of SHS is well-fitted by either a linear or linear-quadratic dose response, with an excess risk of about 40% after exposure to 0.5 Gy. The increase is significant ($P < 0.05$) for doses between about 0.1 and 0.5 Gy but not for doses between 0.01 and 0.09 Gy. In the dose range of 0.01 to 0.09 Gy (mean dose 0.05 Gy) there were 3 SHS cases observed with 1 case expected among 43 subjects exposed at ages 0 to 7 weeks PC and 1 case observed with 0.6 cases expected among 45 subjects exposed at ages 8 to 15 weeks PC. The data are also compatible with a threshold dose of a few centigrays (95% confidence intervals of 0 to 0.13 Gy for those exposed 0 to 7 weeks PC and 0 to 0.10 Gy for those exposed 8 to 15 weeks PC). In the total cohort with doses up to about 2 Gy, among those exposed to radiation 8 to 15 weeks PC, SMR occurred in 12 of 29 children with small heads; however, SMR did not occur in 17 children with small heads who were exposed 0 to 7 weeks PC. For doses less than 1 Gy received at a PC age greater than 15 weeks, there was no significant elevation of SMR or SHS.

3. Severe mental retardation (SMR)

Japanese A-bomb survivors in Hiroshima and Nagasaki provide the primary source of information on SMR for persons exposed *in utero*.[33] This survivor cohort of 1544 exposed people included 30 subjects with SMR compared with 13 expected among the unexposed controls. The risk per gray from exposure between 8 and 15 weeks was four times greater than that for exposure between 16 and 25 weeks PC. Figure 16 shows the relation between the incidence of SMR and the DS86 uterine dose for all subjects irradiated between 8 and 15 weeks PC and between 16 and 25 weeks PC with the omission of four cases that probably were unrelated to radiation exposure (two subjects with Down's syndrome, one subject with a retarded sibling, and one subject with Japanese encephalitis). In the 8- to 15-week PC group there were two cases at doses < 0.1 Gy and one case in the dose range of 0.1 to 0.5 Gy, whereas in the 16- to 25-week PC group there were no cases in the dose range of 0 to 0.99 Gy.

In contrast to the SHS-sensitive period, SMR was not observed among the Japanese A-bomb survivors who were exposed *in utero* before day 56 PC and was observed only after exposure between days 56 and 175 PC (weeks 8 to 25). Dekaban's report,[30] however includes detailed case reports

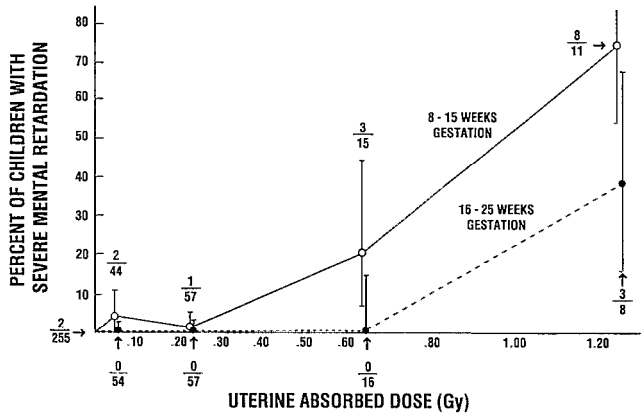

FIG. 16. Percent of children with severe mental retardation, excluding Down's syndrome, in those irradiated prenatally in Hiroshima and Nagasaki, including 90% confidence limits. The numbers above or below the data points arc the incidence/cohort size for the mean DS86 doses received by each cohort. (After Otake, Yoshimaru, and Schull, Ref. 33.)

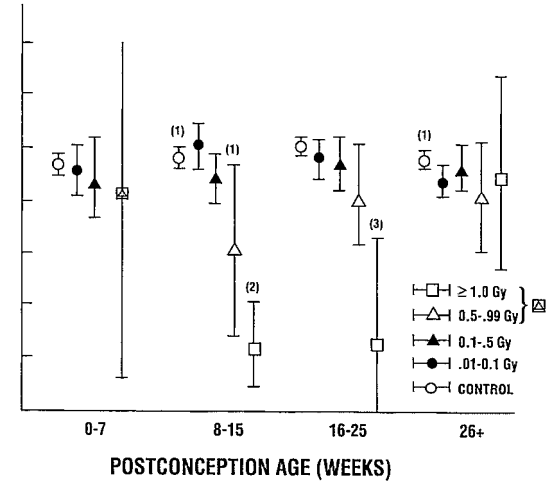

FIG. 17. Mean IQ scores by PC age and fetal dose, including 95% confidence limits. Numbers in parentheses are the count of several retarded cases included in the data, defined as children with IQ scores of ≤ 64. (After Schull and Otake, Ref. 25.)

on 9 of 26 subjects with SMR following therapeutic irradiation at gestational ages between 4 and 8 weeks.

Figure 16 demonstrates a threshold of radiation dose of about 0.65 Gy for SMR induced in the group exposed between 16 and 25 weeks PC. For the critical 8- to 15-week PC group, the data without the exclusion of the Down's syndrome cases can be fitted adequately with linear, linear-quadratic, and quadratic dose-response models. The risk of SMR after exposure to 0.1 Gy is 4% with the linear model, but is 10 times smaller with the quadratic model (i.e., 0.4%), and four times smaller with the linear-quadratic model.[33] However, when the two Down's syndrome cases are eliminated, the data are also consistent with a dose threshold of 0.39 Gy (95% confidence interval 0.12 to 0.60 Gy) for the grouped data. With the threshold model, the risk above the threshold of 0.39 Gy is 0.74 per gray.

The issue of the presence or absence of a threshold in the 8- to 15-week PC period cannot be resolved with current epidemiological or experimental information." Environmental factors, including malnutrition and disease which followed the devastation created by the A-bombs, may have affected the incidence of SMR and confounded the question. Nevertheless, because SMR, like malformation, appears to be a phenomenon arising from multicellular damage, it is often regarded as a deterministic effect rather than a stochastic effect in which case a threshold is likely.

Another indicator of damage to the cerebral cortex is the effect of prenatal radiation exposure on the intelligence test scores of children. Schull and Otake[25] analyzed this indicator for children aged 10 to 11 who were exposed *in utero* in Hiroshima and Nagasaki (Fig. 17). The substantial drop in IQ in those subjects who were exposed at gestational ages of 8 to 15 weeks or 16 to 25 weeks PC confirms the risk of SMR at doses greater than 0.5 Gy. A progressive downward shift in IQ of about 30 points per gray occurs in the group exposed at 8 to 15 weeks PC; however, the drop is not statistically significant for doses less than 0.1 Gy.

4. Growth retardation

Growth retardation with reduced height and weight has been observed in mammalian, including human, offspring particularly when irradiated during organogenesis.[29,30,35] This effect is attributed to cell killing and is less likely to be produced by irradiation during the fetal stage and then only after doses exceeding 0.5 Gy.[30,35] The retardation may continue into adult life. Growth retardation was apparent 17 years later among the children irradiated *in utero* during the A-bombing,[35] for fetal irradiation occurring within 1500 m of the hypocenter (i.e., for T65DR average doses of about 0.25 Gy). No risk estimate is available for this effect, but there is probably a threshold on the order of 0.1 Gy.

5. Sterility

Sterility has also been observed in persons irradiated during the organogenesis and fetal phases of gestation." Because of their sensitivity in the immature state, both male and female gonial cells can be depleted through cell death in the late as well as the earlier stages of fetal development. Sterility in the male and loss of fertility in the female may therefore be produced by a fetus being exposed to a smaller dose of radiation (e.g., < 1 Gy) than that required to produce the same result in adults. No information on this possibility is available for the Japanese survivors exposed *in utero.*[36]

6. Cancer induction

Cancer may be induced in man following exposure to radiation *in utero.* Little is known about the relative risk of *in utero* irradiation as a function of PC age; the risk may be highest during the first trimester.[37] The risk of excess malignant disease in persons following prenatal irradiation has been estimated from the results of two categories of epidemiologic study. The first category comprises several case/control studies in which the frequency of prenatal radiation exposure of children dying from cancer was compared with

TABLE IV. Risk of cancer after *in utero* radiation exposure.

Source	Age range	Number with cancer	Number irradiated	Number of controls	Number irradiated	Rclativc risk factor	Cancer incidence/ 10 000 people per gray of fetal dose
Case/control studies following diagnostic x-ray exposure							
Oxford surveys							
Stewart & Kneale (39)	0 to 10	7649	1141	7649	774	1.56	572 (300 to 800)[a]
Bithell & Stiller (40)	0 to 10	7649	1141	7649	774	1.56	217
Muirhead & Kneale (41)	0 to 14	...	...	...	...	...	640 (410-1000)[a]
Twins							
Mole (33)	0 to 10	161	111	183	101	1.8	...
Harvey *et al.* (44)	0 to 14	31	12	109	18	2.4 (1.0 to 5.9)[a]	...
Rodvall *et al.* (45)	0 to 15	95	25	190	39	1.4 (0.8-2.5)[a]	...

Source	Age range	Number with cancer	Number irradiated	Number expected with cancer	Excess cancers	Dose (Gy)	Cancer incidence/ 10 000 people per gray of fetal dose
Cohort studies following A-bomb radiation exposure							
Jahlon & Kato (46)	4 to 10	1	1250	0.75	0.25	0.184	10.9[b]
Yoshimoto (47)	4 to 15	2	1263	0.73	1.27	0.184	54.6 (0 to 279)[a,b]
Yoshimoto (47)	4 to 39	13	920	5.9	7.1	0.279	223 (16 to 492)[a]

[a]Confidence Intcrval = 95%.
[b]Not significant.

that of-matched controls who were cancer-free children. The second category comprises prospective cohort studies of the observed cancer incidence (or mortality) in Japanese A-bomb survivors exposed *in utero* compared with that expected in the general population in Japan for the same age, sex, and year. Table IV summarizes the results of both types of study.

In the first case/control study, the Oxford survey of childhood malignancies conducted by Stewart *et al.*,[38] the children with cancer had a significantly higher probability of prenatal exposure, usually from pelvimetry of the mother conducted in the third trimester, with fetal doses on the order of 0.01 Gy. Stewart and Kneale[39] later concluded that the relative risk of cancer mortality before age 10 in exposed children was about 1.5, with an absolute risk of 570 deaths: 10 000 people per gray of fetal dose and about 50% of the excess deaths resulting from leukemia. A reevaluation of the fetal doses reduced this estimate to an absolute risk of 217 deaths: 10 000 people per gray of fetal dose.[40]

In a recent expansion of the original Oxford survey, the revised fetal doses gave a risk of excess cancer *incidence* in children before age 14 of 640 cases: 10 000 people per gray of fetal dose.[41] Although a similar association between childhood cancer and *in utero* x-ray exposure was found in several later case/control studies, the UNSCEAR report[24] and MacMahon,[42] in particular, expressed doubt about the causal nature of the association. One significant problem was the possibility that the association was biased by the characteristics of the small fraction of pregnant women selected for pelvimetric examination. To counter this argument, three case/control investigations were conducted on childhood cancer in twins in which the incidence of *in utero* exposure was considerably greater than that in singletons.[43-45] In all three studies there was an increased frequency of x-ray examination in twins who developed childhood cancer, and the deduced relative cancer risk following exposure was similar to that determined in earlier singleton studies. These findings considerably strengthen the evidence that these low x-ray doses *in utero* cause cancer.

In the cohort studies of the Japanese A-bomb survivors. an early investigation[46] of 1250 children exposed *in utero* found no excess of cancer during the first 10 years of life following an average fetal dose now estimated to he 0.184 Gy. In a recent follow-up, there were 18 cancer cases among 1630 children in the study period of 1950 to 1984 These 18 cases included two children diagnosed with cancer before age 15; the remaining patients were diagnosed as adults with adult-type cancer, of which only two cases were leukemia.[47] The absolute risk of childhood cancer, based on the two cases, is 55 cases: 10 000 people per gray of fetal dose (upper 95% confidence level of 279), which is considerably below estimates from the Oxford surveys (Table IV). For the entire follow-up period (through age 39), the absolute risk estimate for excess cancer incidence was 223 cases: 10 000 people per gray of fetal dose, which is also less than the Oxford survey risk estimates through age 14.[41] The Japanese cancer excess risk following *in utero* exposure is 6.57 cases: 10 000 people per gray of fetal dose *per year* over the 34-year follow-up period and is similar to the cancer mortality risk for the cohort study of survivors exposed in the first decade of life (6.2 deaths: 10 000 people per gray of fetal dose per year after 35 years of follow-up).[48] Patients in neither cohort study have reached the older ages when most cancers normally appear; therefore, it is likely that the lifetime risk of cancer in the former children comprising the cohort studies will eventually be greater than the risk to persons exposed as adults. Therefore a reasonable assumption for the lifetime risk of increased mortality from cancer following fetal exposure is that it will be similar to that projected for children exposed during the first decade of life. The latter risk, based on age extrapolation, is 1404 deaths:

TABLE V. Risk associated with irradiation during fetal development (After Brent, Ref. 29).

	Preimplantation	Organogenesis	Early fetal	Mid-fetal	Late fetal
Postconception time, days	0 to 8	9 to 50	51 to 105	106 to 175	>175
Postconception time. weeks	1	2 to 7	8 to 15	16 to 25	>25
Effects					
Lethality	+ + +	+	+	-	-
Gross malformations	-	+ + +	+	+	-
Growth retardation	-	+ + +	+ +	+	+
Mental retardation	-	-	+ + +	+	-
Sterility	-	+	+ +	+	+
Cataracts	-	+	+	+	+
Other neuropathology	-	+ + +	+	+	+
Malignant disease	-	+	+	+	+

- No observed effect.
+ Demonstrated effect.
++ Readily apparent effect.
+++ Occurs in high incidence.

10 000 people per gray of fetal dose or 14% per gray.[36] An effectiveness factor for the dose rate was not used because the A-bomb explosion resulted in a single acute dose.

7. Genetic effects

Genetic effects involve hereditary gene mutation and chromosomal damage that produce deleterious effects in future offspring over many generations. Because irradiation of the germ cells in a male or nonpregnant female introduces a genetic risk, the abdominal irradiation of a pregnant woman involves an additional risk to the progeny of the fetus. Because the number of future offspring of a fetus usually exceeds that of the irradiated parent, the genetic consequences of fetal irradiation have greater significance.

Genetic risks were extensively reviewed by the National Academy of Sciences (NAS),[36] UNSCEAR,[24] and the International Commission on Radiological Protection (ICRP).[49] The risk is commonly expressed as the gonadal dose that will double the spontaneous incidence of genetic aberrations in a mammalian population; this is known as the doubling dose. Although there is considerable uncertainty, the general agreement in the above reviews is that 1 Gy is a reasonable value for the doubling dose for low dose-rate. low-LET radiation. This value is primarily based on results from experiments with mice. The corresponding estimate for the number of radiation-induced genetic disorders for all generations following 1 Gy of fractionated or low dose-rate exposure of either parent is 100 incidents: 10 000 live-born children; this is a risk of 1% per gray. This risk was adopted by the ICRP in their recent recommendations for radiation protection[49] following a review of the estimates in the NAS and UNSCEAR reports noted above.

B. Summary of effects by gestational age (PC)

Table V qualitatively summarizes the gestational ages for the five periods of embryo/fetus development and the relative magnitude of risk associated with radiation exposure during each period. The absolute magnitudes of these risks in man are small and possibly nil for most of these effects for low doses on the order of 0.1 Gy. The accuracy of risk estimates improves at higher doses.

1. Preimplantation: 0 to 8 days PC

During the preimplantation period, the death of the embryo or early fetus is the principal effect of radiation exposure, with sensitivity being dependent on cell-cycle stage.[35] The maximum risk to the embryo/fetus of rodents suggests a 1% to 2% chance of early death after doses on the order of 0.1 Gy, corresponding to an LD_{50} of about 1 Gy.[20] Most animal studies of irradiation during this period do not show an increase in the risk of malformation.[20]

2. Embryonic period: 8 to 56 days PC

The principal risk during the embryonic period is malformation of specific organs, including neuropathology, particularly for irradiation during differentiation. In man, a major risk to the child is SHS in the child but without mental retardation. In Hiroshima the incidence of SHS was 17% for estimated air kerma of 0.1 to 0.2 Gy (T65DR dosimetry) received by subjects in the most sensitive developmental period of 2 to 11 weeks PC. However, in Nagasaki, there was no significant increase in SHS for air kerma less than 1.5 Gy.[31,35] A recent re-evaluation using the new DS86 dosimetry" concluded that the risk progressively increases with dose above a possible threshold of a few centigrays and is about 40% for a uterine-absorbed dose of 0.5 Gy. The incidence at doses below 0.1 Gy received during this period was not significant ($p > 0.05$). In addition, the incidence for any dose delivered after this period was not significant. Investigations on rodents suggest an effective threshold dose for malformation in the range of 0.05 to 0.25 Gy, depending on the time of irradiation. A risk of growth retardation with a similar range of threshold dose has also been observed in mammalian populations, including man. Further, there is a

possible late cancer risk of 14% per gray for an acute single dose to the fetus at 8 to 56 days PC, fractionation of the dose will probably reduce this risk.[14,36]

3. Early fetal: 56 to 105 days PC

SHS and mental retardation are the principal risks observed in children following *in utero* irradiation during the early fetal period. During the first part of this period, the risk of SHS is similar to that during the organogenesis phase[34] but this risk appears to decrease after week 11. The risk of mental retardation, which was not evident among the Japanese A-bomb survivors irradiated during the organogenesis phase, reaches a maximum level during the early fetal stage. The most conservative evaluation of the data suggests a risk of 40% per gray with a relationship proportional to fetal dose. The data are also consistent with a threshold of at least 0.12 Gy. Irradiation during this time also carries a demonstrated risk of growth retardation that is smaller than that for the organogenesis phase. When the fetal dose is about 1 Gy or greater, there is also a risk of sterility and a continuing risk, presumably with no threshold, of subsequent cancer.

4. Mid-fetal: 105 to 175 days PC

Irradiation during the mid-fetal period is not likely to induce gross malformations. During this period, SMR was observed with a threshold of about 0.65 Gy among the people irradiated *in utero* during the A-bombing. Some effects on human development, including SHS and growth retardation with reduced height and weight, can also be produced during this stage but only after doses exceeding 0.5 Gy.[28,30] However, there is a continuing risk of subsequent cancer development. The observed risks of sterility and neuropathology are' smaller than those associated with earlier irradiation."

5. Late fetal: more than 175 days PC

Apparently, during the final period of pregnancy the risks of malformation and mental retardation are negligible. The major risk quantitatively is probably subsequent cancer development. The data from the Oxford surveys of childhood cancer and similar case/control studies[35,37-42,44,45] mainly relate to diagnostic x-ray exposure of pregnant women in their third trimester. The extrapolated risk for lifetime cancer of 14% per gray for a single acute radiation dose and a smaller risk for fractionated radiation doses, as noted earlier, will apply. However, there is a continuing risk of growth retardation for doses exceeding 0.5 Gy.

C. Conclusions

The three effects that dominate the risk to a fetus following exposure to ionizing radiation are malformation (including SHS), SMR, and subsequent development of cancer. For fully developed organisms the risks are likely to be substantially reduced when the exposure is fractionated over a period of 4 to 6 weeks, the typical duration of therapy. For a rapidly developing embryo/fetus, however, there are periods of critical sensitivity, and irradiation during these periods will increase some specific risks. These risks can be considered initially and conservatively by assuming that the dose-response relationships for mental retardation and cancer induction are linear with dose and without threshold. A reasonable assumption for the risk of malformation is a threshold of 0.5 Gy and a 50% risk at 1 Gy of fetal dose; this risk becomes linear at doses greater than 0.05 Gy. The assumed risk of SMR would then be 0.4 per gray for exposure during gestation weeks 8 to 15. Assuming a cancer risk equal to the lifetime risk for a 5-year-old child[36] and a dose-rate reduction factor of 2,[49] the risk to the fetus is 7% per gray. A dose of 0.1 Gy would then produce a risk of 1:25 for SMR, 1:20 for malformation, and 1:140 for increased cancer mortality. The malformation risk (including SHS) and the risk of SMR are clearly dominant with these assumptions.

Less conservatively, a linear-quadratic dose response for SMR shows a risk of 1% at 0.1 Gy. Furthermore, the likelihood of a threshold dose greater than 0.1 Gy, and perhaps as high as 0.4 Gy, implies a zero risk in this range of fetal dose for the most critical 8- to 15-week PC group. Following a dose of 0.2 Gy, the reduction of IQ in this group would on average be 6 points, assuming a linear dose response. The risk of SHS does not appear to be linear with dose above an assumed threshold; more likely this risk is linear-quadratic in shape, as much of the animal data show (Fig. 14). The lifetime cancer risk at 0.2 Gy of about 1.5% (1:70) and a risk of malformation including SHS of about 5% may be the most critical risks. As currently evaluated, the genetic risk for future generations is considerably smaller than these somatic risks to the live-born child.

It should be evident that accurate specification of risks for fetal doses of 0.1 to 0.2 Gy is not possible with the present state of knowledge, particularly for fractionated radiation. Whatever the risk at 0.2 Gy or even larger doses, consideration should be given not only to the risk for the child but also to the maternal benefit from the therapeutic procedure. The conservative recommendation of a limit of 0.005 Gy during the nine months of gestation as guidance for maximum fetal dose from *occupational* exposure of the mother is not relevant because it ignores the issue of the medical benefit to the mother in the treatment context. The possibility of substantial fetal risks of malformation and SMR during the first and early in the second trimesters of pregnancy, however suggests that planning a treatment regimen around the critical period of gestation may be appropriate.

Table V is a guide to the PC periods in days and to the embryo/fetal dose range for which the risk of known effects is believed to be greater than 5%. Consideration should be given to the avoidance or reduction of radiation exposure during these periods. Reducing the fetal dose to less than 0.1 Gy substantively minimizes these risks. Table VI summarizes the risk to the fetus as a function of radiation dose; however, the user should recognize that this table is a simple guide which does not take account of different end points and stages of gestation.

TABLE VI. Summary of risk as function of dose.

Dose (Gy)	Risk
<0.05	Little risk of damage
0.05-0.10	Risk uncertain
0.10-0.50	Significant risk of damage during first trimester
>0.50	High risk of damage during all trimester

6. PROFESSIONAL CONSIDERATIONS

When a pregnant patient is to receive radiation therapy, the physician should carefully document aspects of the treatment that could affect the fetus, including the basis of a decision for or against therapeutic abortion. This information should be made a part of the patient's record and, at minimum, should include the following:

(1) Time of gestation. The estimate for the patient should be verified to the extent possible.

(2) Estimated dose to fetus. Measurements and/or calculations should be documented so that another physicist can verify the dose estimates. Details should include the geometry of shielding, if appropriate, and the point or points of dose estimation.

(3) Basis for recommendations to patient. Full reference should be made to this and other authoritative reports for dose determination and risk evaluation.

(4) Informed consent. The patient, after discussions with the physician, should be asked to sign a consent form. A typical consent form appears in Appendix B; however, any form should have approval of the institution's legal counsel before it is used. Topics that should be discussed with the patient include the following:

(a) anticipated dose to fetus resulting from therapy,

(b) comparison to fetal radiation dose owing to naturally occurring radiation,

(c) nondistinction between radiation-caused anomalies in the child and those that occur naturally,

(d) rate of natural occurrence of abnormalities,

(e) anticipated increase of risk of fetal abnormality resulting from radiation therapy to this patient,

(f) citations and statements from authoritative reports regarding recommendations for or against therapeutic abortion in various situations,

(g) written indication that the patient understands the discussion, with her signature, and

(h) presence of a credible witness to attest that the patient understood the discussion, with signature of the witness.

Determining the degree of detail appropriate for a discussion with a particular patient is difficult. A simple statement will not be correct and a complex discourse might not be understood.

VII. SUMMARY OF RECOMMENDATIONS

In collaboration with the radiation oncologist, a medical physicist should perform the following tasks as part of the management of a pregnant patient.

(1) Complete all planning as though the patient were not pregnant. If the fetus is near the treatment beam do not take portal localization films with open collimators and blocks removed.

(2) Consider modifications of the treatment plan that would reduce the radiation dose to the fetus by changing the field size and angle, selecting a different radiation energy, etc. For the field edge nearest the fetus, use trimmers in the lower position where this is an option. If possible, treat a pregnant patient with photon energies of less than 25 MV.

(3) Estimate dose to the fetus without special shielding using out-of-beam data measured in a phantom. Usually there are at least three points of interest: the fundus, the symphysis pubis, and a midpoint.

(4) Design and construct special shielding if the fetal dose without shielding exceeds acceptable limits. Usually four to five half-value layers of lead are appropriate.

(5) Measure dose to fetus in a phantom during simulated treatment, with shielding in place, adjusting radiation amount and location.

(6) Document the treatment plan, including special shielding, and discuss the planned treatment with all personnel involved in patient setup.

(7) Check all aspects of safety, including load-bearing limits of the treatment couch, support of shields, movement of shields, etc., to ensure that there will be no injury to the patient or to personnel.

(8) Be present at the time of initial patient setup and be available for consultation when the patient receives therapy to ensure that shielding is placed correctly and safely. Photograph the setup of each field at least once as part of documentation.

(9) Monitor fetal size and location throughout the course of radiation therapy and repeat estimates of fetal dose if necessary.

(10) Document the completion of treatment by estimating the total dose, including the range of dose, to the fetus during the course of therapy. Recheck the estimate to determine whether the documentation includes complete information regarding the phantom measurements and placement of special shielding.

(11) Consider referring the patient to another institution for treatment if equipment and personnel are not available for reducing and estimating dose to the fetus as described above.

APPENDIX A

The data presented in Figs. 18 through 24 are typical for modern radiation therapy machines. However, the dose outside a beam depends on the design and construction of the machine head and collimators. For this reason the out-of-beam dose may differ among machines of the same nominal type and energy. The specific machines used for the measurements in this report were as follows,

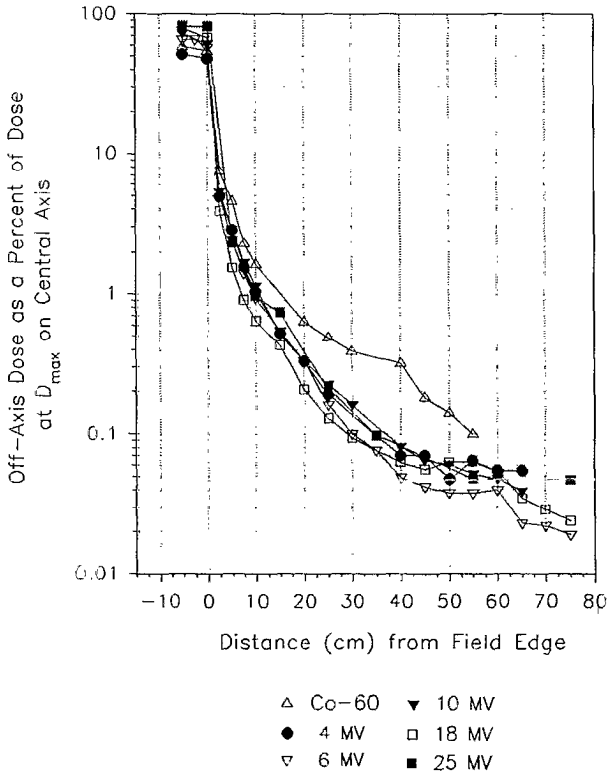

FIG. 18. Total absorbed dose in phantom from 10X10-cm^2 fields of ^{60}CO gamma rays and 4-, 6-, 10-, 18-, and 25-MV photons at IO-cm depth, normalized to 100% on the central axis at depth of maximum dose.

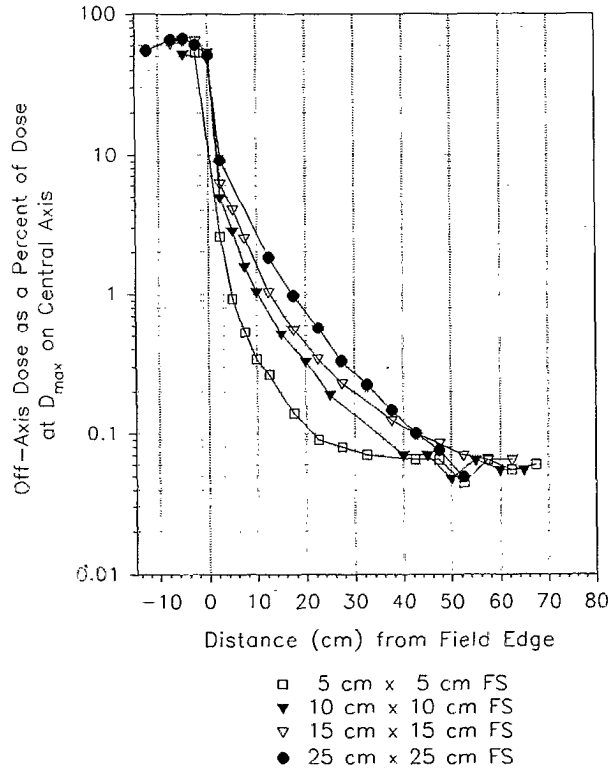

FIG. 20. Total absorbed dose in phantom from 4-MV photons for field sizes of 5X5, 10X10, 15X15, and 25X25 cm^2 at 10-cm depth, normalized to 100% on the central axis at depth of maximum dose.

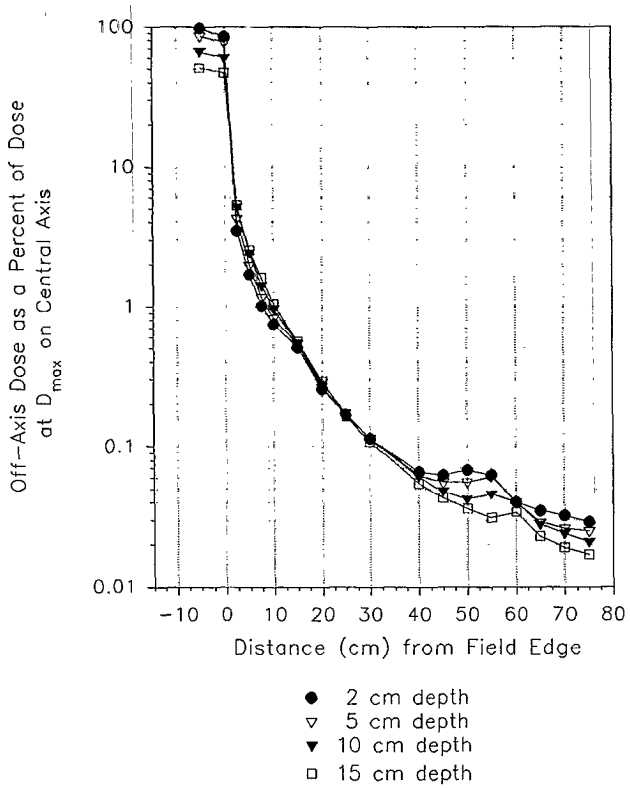

FIG. 19. Total absorbed dose in phantom from a 10X10-cm^2 field of 6-MV photons (Varian Clinac 2100C) at depths of 2, 5, 10, and 15 cm, normalized to 100% on the central axis at depth of maximum dose.

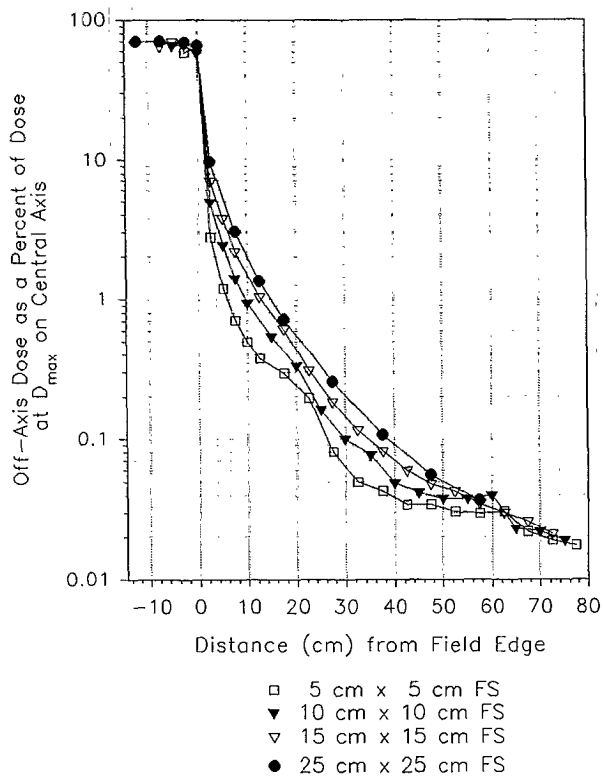

FIG. 21. Total absorbed dose in phantom from 6-MV photons for field sizes of 5X5, 10X10, 15X15, and 25X25 cm^2 at 10-cm depth, normalized to 100% on the central axis at depth of maximum dose.

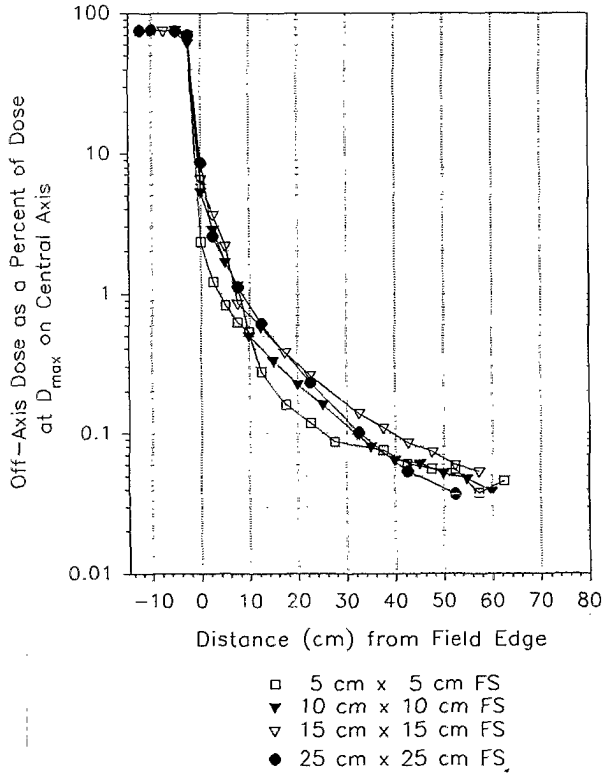

FIG., 22. Total absorbed dose in phantom from 10-MV photons for field /sizes of 5X5, 10X10, 15X15, and 25X25 cm^2 at 10-cm depth, normalized to 100% on the central axis at depth of maximum dose.

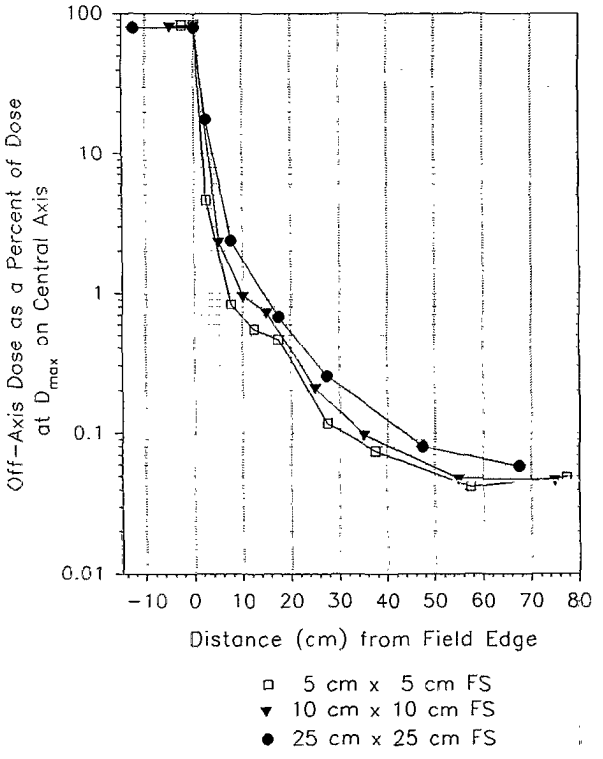

FIG. 24. Total absorbed dose in phantom from 25-MV photons for field sizes of 5X5, 10X10, 15X15, and 25X25 cm^2 at 10-cm depth, normalized to 100% on the central axis at depth of maximum dose.

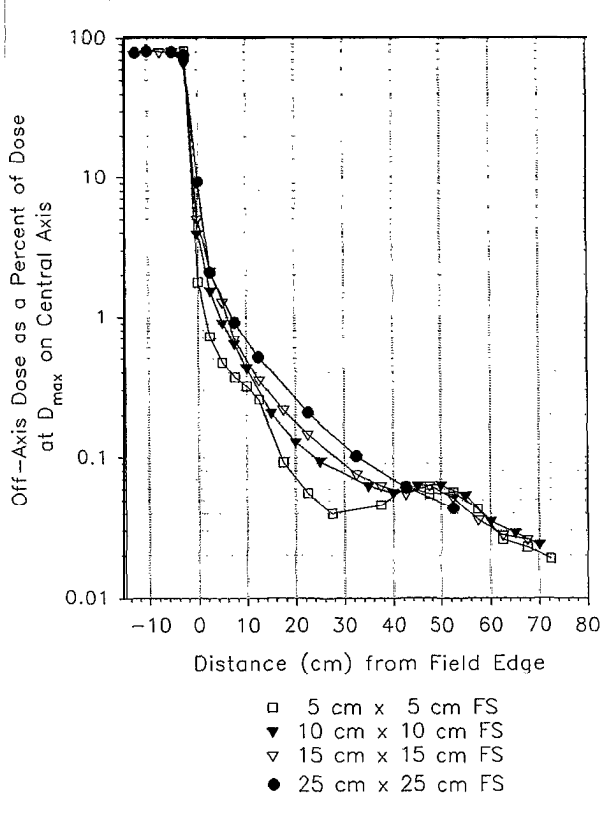

FIG. 23. Total absorbed dose in phantom from 18-MV photons for field sizes of 5X5, 10X10, 15X15, and 25X25 cm^2 at 10-cm depth, normalized to 100% on the central axis at depth of maximum dose.

Varian Clinac 2100C (6 and 10 MV)	S/N 004
Varian Clinac 2100C (6 and 18 MV)	S/N 008 and S/N 009
Varian Clinac 4	S/N 157
Varian Clinac 4/100	S/N 60
Siemens Mevatron 74	S/N 01358
Philips SL25 (6 and 25 MV)	S/N 5013
Therac 6	S/N and S/N 4
AECL Theratron 780 (cobalt)	S/N 35

S/N = Serial number.

All data in this Appendix were measured using TLDs or diodes. Phantoms used were either water or polystyrene.

APPENDIX B

DISCLOSURE AND CONSENT FOR RADIATION THERAPY

I hereby voluntarily request and authorize Dr._____ as my physician, and such associated technologists and health care providers as he/she may deem necessary, to treat my condition which has been explained to me as:

_____

I understand that my condition may be treated with external beam radiation therapy alone, with internal radiation implants alone, or with both. Also, radiation therapy may be combined with surgery and/or chemotherapy.

The nature and purpose of the proposed procedure, the alternative methods of treatment, and the risks and hazards if treatment is withheld have been explained to me by my physician. I understand that radiation can be harmful to my unborn child. There is a possibility of miscarriage and there is also the possibility that the child may not develop or grow in a normal manner as a result of these treatments. I have had an opportunity to discuss these matters with my physician and to ask questions about my condition, alternative methods of treatment, and the proposed procedure(s). I understand that no warranty or guarantee has been made to me as to result or cure.

I further authorize the taking of photographs or placing of tattoo or skin marks as necessary for treatment.

Patient/Other Legally Responsible Person (Signature)

Date Time

Witness

Address

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