Journal of Occupational Medicine

SPECIAL SUPPLEMENT

MARCH, 1961

Acute Radiation Death Resulting from an Accidental Nuclear Critical Excursion

From the Los Alamos Scientific Laboratory, University of California, Los Alamos, New Mexico. This work was done under the auspices of the U.S. Atomic Energy Commission.

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The sections are by different authors who discuss those facets of the case with which they are most familiar. There are some areas of repetition, since each author was encouraged to include all details pertinent to his presentation.

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—THOMAS L. SHIPMAN, M.D.
Health Division Leader
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Description of the Accident and Subsequent Events

Thomas L. Shipman, M.D.

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Fig. 1 shows the solvent-treating tank and the assumed position of chemical operator K* just prior to the accident. He stood on the short stepladder shown in the photograph, looked

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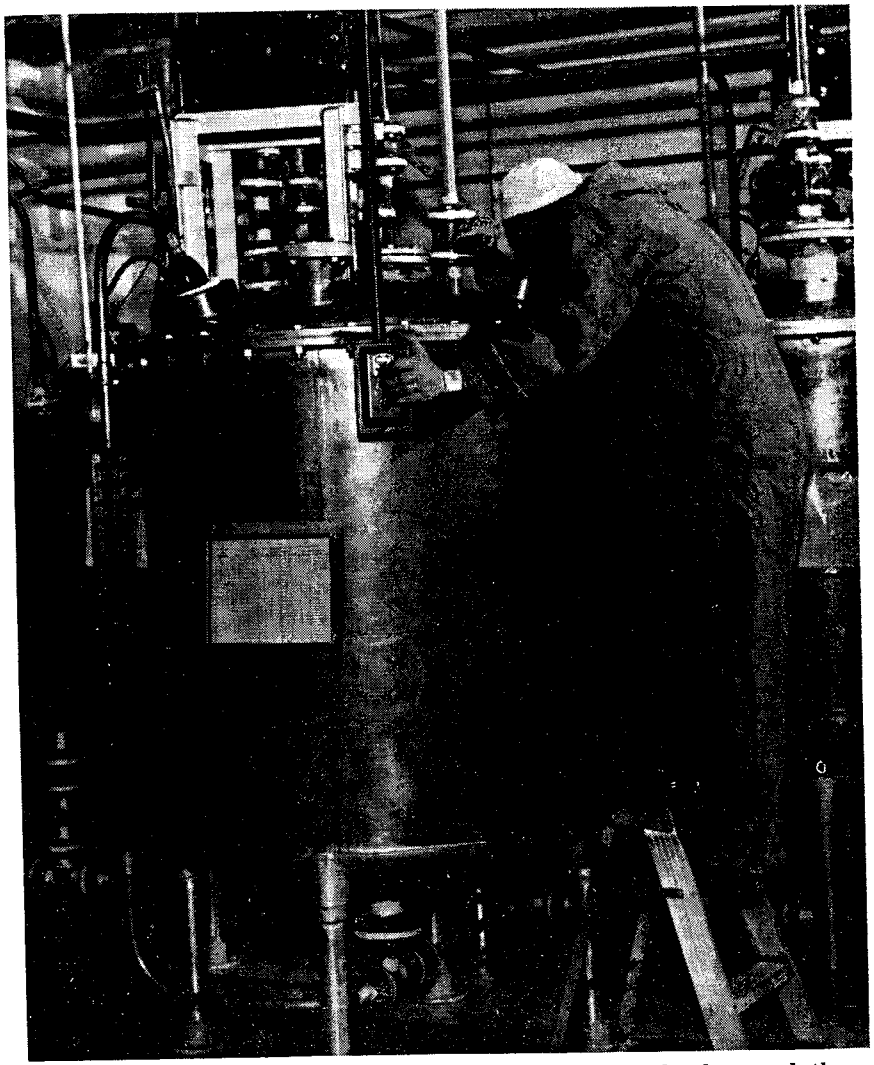

Fig. 1—The tank in which the critical excursion took place and the assumed position of the victim of the accident, K.

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By this time K could not stand unaided; so after he was showered the two other men placed him on the floor of an adjacent airlock, where he lay essentially unconscious. R went to summon assistance, and D returned to Room 218 to see whether the system was properly shut down. He spent one or two minutes in the room, and undoubtedly passed several times within 2 or 3 ft. of the tank, which was still intensely radioactive. Finding nothing out of order, he returned to the airlock.

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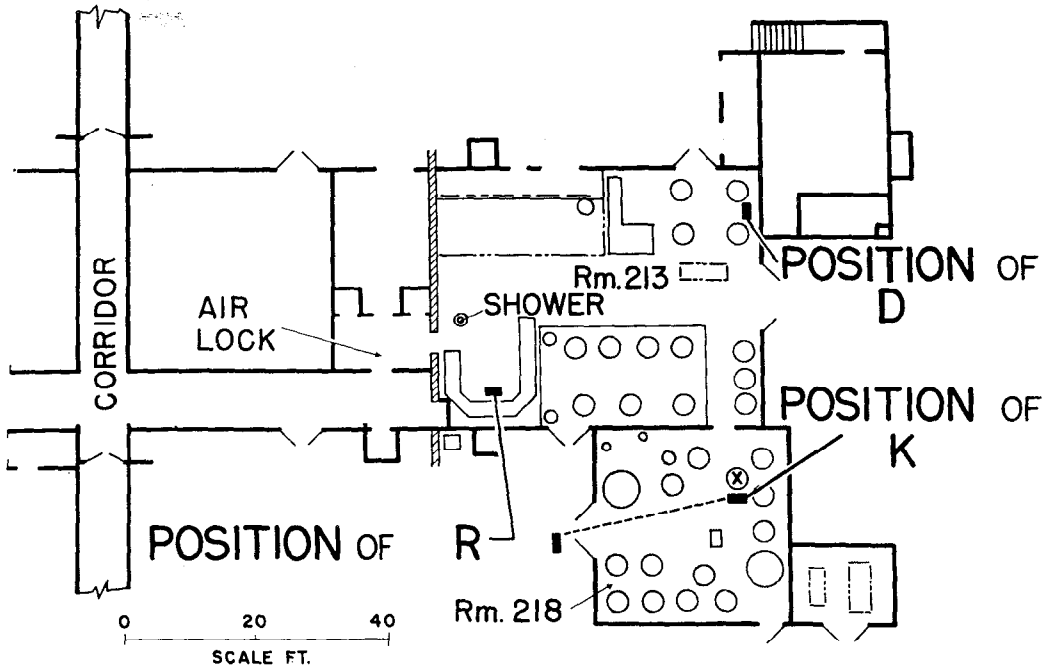

Fig. 2—Floor plan of rooms 213-218 DP West where the accident occurred. The circles represent chemical treatment tanks and the one marked with an X is that shown in Fig. 1.

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Between 4:40 and 4:45 p.m., 5 to 10 minutes after the accident, supervisors and monitoring staff converged on the area in answer to R's call for help. They found no widespread alpha activity (considered the principal problem), and it was not until 4:52 p.m. that the area monitoring supervisor first detected the presence of gamma activity. With its discovery, the entire area was evacuated.

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Precise calculation of the doses received by the victims of the accident required much time, but it was apparent within perhaps two hours that K had almost certainly received a massive supralethal dose of several thousand rads, and that D and R had received doses that were significant but far below lethality. In order to make the discussions that follow more understandable, the final figures are given here. K's dose varied from approximately 12,000 rads to the upper abdomen to less than 100 rads to the lower extremities. This might be averaged to approximately 4,500 rads of whole-body radiation. D's dose was approximately 130 rads, and that of R was about 35 rads. All these figures include dosage from neutrons and gamma rays.

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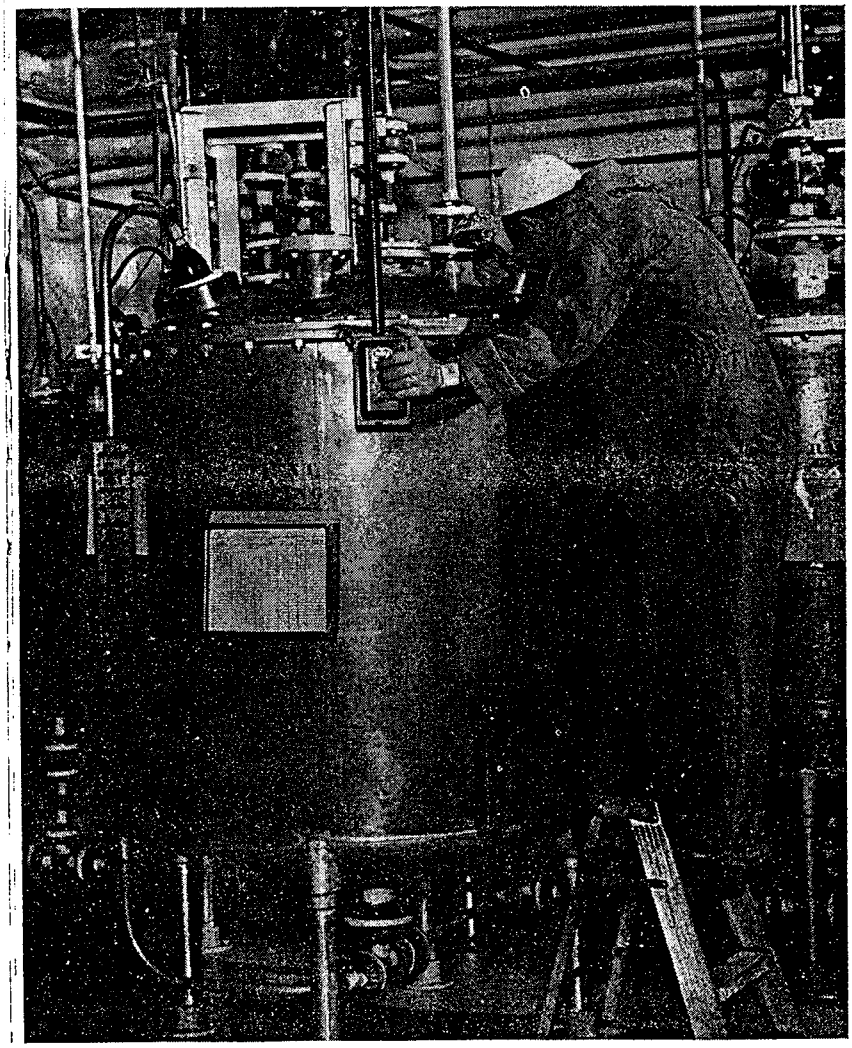

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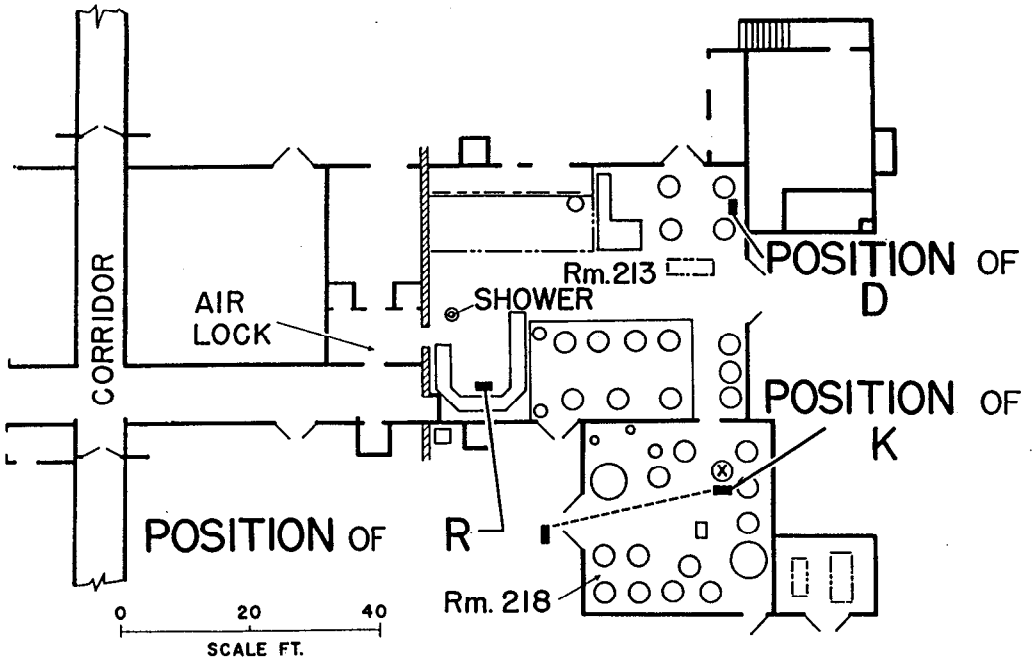

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SECTION II

Clinical Course of Case K

C. C. Lushbaugh, M.D.
With the collaboration of Robert S. Grier, M.D., John S. Benson, M.D., and Donald F. Petersen, Ph.D.

IN RETROSPECT, the clinical course of patient K can be divided into four rather distinct periods, differing in duration, symptomatology, and response to supportive therapy. The first period (lasting about 20 to 30 minutes) was characterized by his immediate physical collapse and mental incapacitation, which progressed to semiconsciousness and severe prostration (see Section I). The second period (lasting about one and one-half hours) began with his arrival by stretcher at the emergency room of the hospital and ended with his transfer from the emergency room to the ward for further supportive therapy. This second period was characterized by such severe cardiovascular shock that death seemed imminent during the whole time. During this period he seemed to be suffering severe abdominal pain. The third period was about 28 hours in length and was characterized by enough subjective improvement to encourage continued attempts to alleviate his anoxia, hypotension, and circulatory failure. The fourth period began with the unheralded onset of rapidly increasing irritability and uncooperativeness, bordering on mania, followed by coma and death in approximately two hours. The entire clinical course lasted 35 hours from the time of radiation exposure to death.

When the patient was seen first in the Los Alamos Medical Center emergency room, he was semiconscious and incoherent, retching, vomiting, and hyperventilating. His skin was cold, dusky reddish-violet, and his lips and oral mucous membranes were extremely cyanotic. The conjunctivae were so hyperemic that they appeared to have been injured by a welder's arc. He was immediately wrapped in warmed blankets and surrounded by hot water bottles. His blood pressure and radial pulse were at first unobtainable, but his apical heart rate was 160 per minute (just audible by stethoscope). His lungs seemed to be clear to auscultation. He had shaking chills and coarse purposeless movements of his extremities and torso, which necessitated restraint by the nursing staff.

About 10 minutes after arrival in the emergency room, he had an attack of propulsive, watery diarrhea which contained some large well-formed stools (later radioassayed, see Section V). At this time his blood pressure was successfully determined at 80/40 and his pulse rate as 160 per minute. A 30 ml sample of blood was obtained for laboratory and dosimetric measurements (see Sections V and VI). External monitoring with a portable survey instrument showed a body surface gamma-ray reading of 15 mr/hr. His feces and vomitus at this time were also sufficiently radioactive to give a demonstrable gamma-ray reading on the survey instrument.

He was given 25 mg of Thorazine, 0.4 mg of atropine, and 60 mg of sodium luminal, but his extreme anxiety and restlessness were not resolved until after receiving 50 mg of Demerol parenterally. An intravenous infusion of 5% dextrose in physiologic saline containing 20 ml of 10% calcium gluconate was started with the intention that this fluid would be replaced by blood just as soon as a cross-match could be completed. The calcium gluconate appeared to be responsible for cessation of his widespread muscular tremors. The clinical impression of profound shock was supported by laboratory findings of an 18 gm hemoglobin and a 58% hematocrit (Fig. 3). The first cross-match was rejected because of positive Coombs' reactions in both major and minor fields, and blood transfusion was deferred until additional cross-matches could be performed. Whole blood, however, was never given because, as shown in Fig. 3, he developed no anemia, and after the first six hours he had relatively stable hemoglobin and hematocrit values. The additional cross-

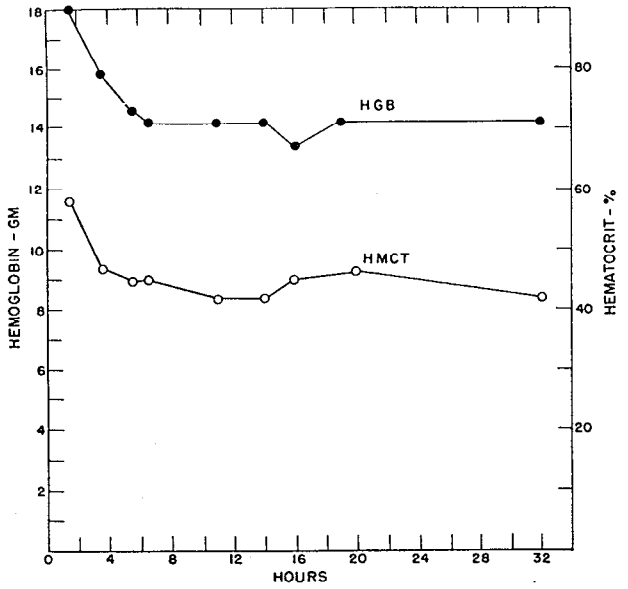

Fig. 3—Changes in hemoglobin and hematocrit following exposure (Case K).

matching continued to be complicated by positive Coombs' reactions with both his serum and his cells. This finding was explained later, when it was found that his own cells were agglutinated by Coombs' serum in the absence of foreign plasma, indicating the presence of isoantibodies on his own cells and free in his serum. This explanation then allowed the use of plasma in the treatment of his shock. More definitive studies of the nature of his antibody and serum proteins were made later (see Section III). Five determinations of his serum bilirubin over the first 25 hours failed to show a significant rise in either the direct or the indirect reaction, suggesting that hemolysis, if any, was negligible.

As coherence slowly returned to the patient, he complained of severe abdominal cramps, but neither increased nor abnormal bowel sounds could be heard. Although nausea continued and he occasionally retched and vomited, he seemed considerably improved. He was transferred to a room, where 1 hour and 40 minutes after the accident (6:16 p.m.) he was placed in bed on shock blocks and in an oxygen tent. Preliminary reports from the radioassays of the first blood sample estimated that his total exposure had been massive and probably supralethal.

As soon as he was resting quietly, he was examined thoroughly. The only positive finding, other than his continuing hypotension (85/45), tachycardia (140), and intense dermal and conjunctival hyperemia, was a temperature of 103°F rectally (Fig. 4). The examination revealed normal ocular fundi, normal cranial and peripheral nerves, normal but cyanotic mucous membranes, clear lungs, and sore abdominal muscles. Both hands and forearms seemed swollen. Although his erythema was most intense over the anterior surface of his face, thorax, abdomen, genitalia, and upper half of the thighs, its full extent was not delineated because it extended posteriorly and inferiorly without sharp borders. He complained of his mouth being dry but was not allowed to drink until several hours later. Forty milligrams of vasopressor amine* was added to his infusion without appreciably improving his blood pressure (Fig. 5). Fig. 5 also shows the relationship between the patient's systolic blood pressure, measurable fluid losses, accumulated

* Wyamine and Levophed were used.

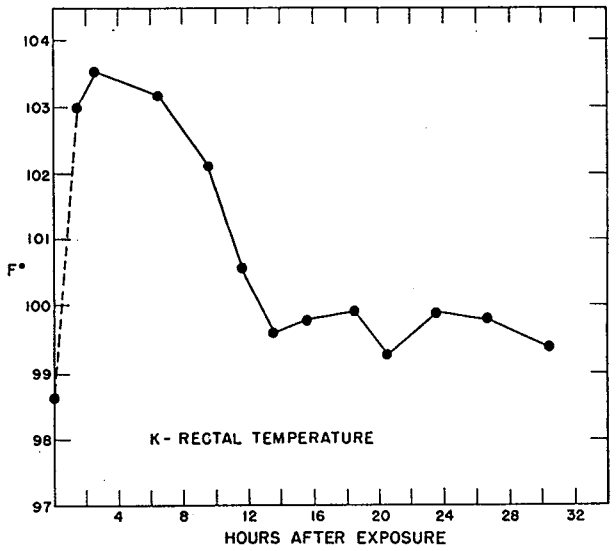

Fig. 4—Changes in rectal temperature following exposure (Case K).

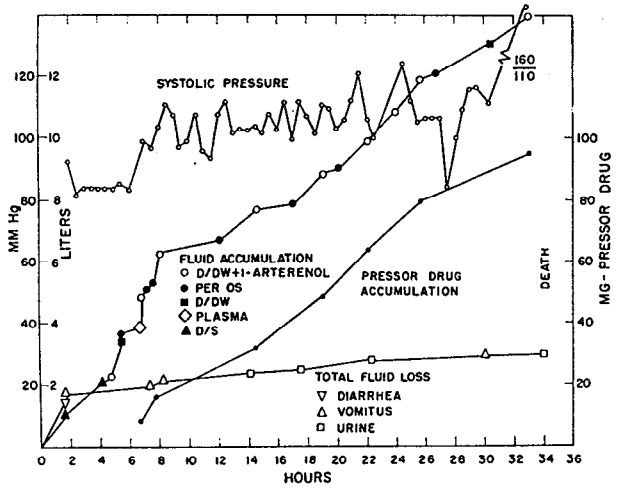

Fig. 5—Composite graph of systolic blood pressure, amount of fluid and vasopressor (levarterenol) administered, and amount of fluid lost via oral, urinary, and fecal routes.

Table I—Summary of Therapeutic Agents Employed in the Symptomatic Treatment of Case K

Compound*	Dose	Doses Administered	Rationale
Atropine	0.4 mg	1	Intestinal hypermotility
Thorazine	25 mg	1	Apprehension
Phenobarbital	120 mg	7	Irritability and insomnia
Hydeltrasol (prednisolone)	20 mg	6	Adrenal cortical failure (?)
Calcium gluconate	20 ml	1	Muscular tremors
B complex	5 ml	1	Coenzyme depletion
Demerol (meperidine)	50 mg	4	Epigastric pain
Wyamine (mephentermine)	7.5 mg	3	Hypotension
Aramine (metaraminol)	30 mg	2	Hypotension
Levophed (levarterenol)	8 mg	6	Hypotension
Compazine (perchlorpromazine)	5 mg	1	Apprehension
Regitine (phentolamine)	5 mg	1	Vascular spasm
Alidase (hyaluronidase)	150 units	1	Local edema
Sparine (promazine)	100 mg	1	Apprehension
Morphine	10 mg	2	Pain and irritability
Nalline	10 mg	1	Morphine antagonism

* Arranged in order of first administration.

fluids, and accumulated amounts of vasopressor substances administered during the entire course of the disease. The abrupt declines in systolic pressure seemed to occur when the rate of flow of the infusion was decreased. Failure of the hemoglobin and hematocrit to decline with progressive fluid administration was difficult to interpret at the time and, in fact, encouraged continuation of his fluid therapy. A number of other medications were used in the attempt to control K's clinical course. A complete summary of the therapeutic agents employed in the symptomatic management of the patient is shown in Table I, where the specific drugs are tabulated in order of their first use, together with the rationale.

K's rectal temperature varied between 103 and 103.5°F for the first six hours and then fell precipitously to normal, where it remained for the duration of his life (Fig. 4). This high initial temperature and its maintenance for six hours were considered in keeping with his suspected massive dose of radiation and were considered to be prognostically grave.[1, 6] Three hours after exposure he was resting quietly, but his radial pulse could still be determined only with difficulty and cyanosis developed whenever he was taken out of the oxygen tent. An electrocardiogram taken approximately 18 hours after exposure (Fig. 6) showed a sinus tachycardia (110/min) and findings consistent with electrical preponderance of the left ventricle. In retrospect (see Section IV, paragraphs on myocardial pathology), these findings were probably best explained as being due to decreased electrical contribution from the right ventricle, resulting in apparent left ventricular preponderance.

The patient was unable to void until 14 hours after the accident (Fig. 5). The results obtained by clinical laboratory test on the first two blood specimens (Table II) reflected this anuria, which was probably a major factor in his rising serum electrolytes, nonprotein nitro-

Table II—Changes in Serum and Urinary Constituents Following Acute Gamma-Neutron Exposure (Case K)

Hours After Exposure	Serum							Urine
	Na (mEq/l)	Cl (mEq/l)	K (mEq/l)	CO_2 cap (mEq/l)	NPN (mg %)	BUN (mg %)	Uric Acid (mg %)	Uric Acid (mg %)
1½	145	113	5.2	.....	41	13	4.8	.....
3½	148	116	5.4	.....	46	20	11.6	.....
5½	148	.....	....	.....	44	33	11.4	.....
6½	.....	.....	....	.....	48	....	16.2	.....
10½	148	.....	....	.....	53	....	17.2	.....
14	.....	.....	....	.....	54	25	.....	258
16	.....	.....	....	.....	54	....	14.4	.....
17	.....	.....	....	.....	....	....	.....	173
19	.....	.....	....	.....	62	42	11.8	.....
20	.....	.....	....	.....	51	20	16.2	.....
22	.....	.....	....	.....	....	....	.....	135
25	143	199	5.1	15	68	43	.....	.....
Normal range	135-145	99-108	3.5-5	22-30	25-30	10-15	2-5	10-200

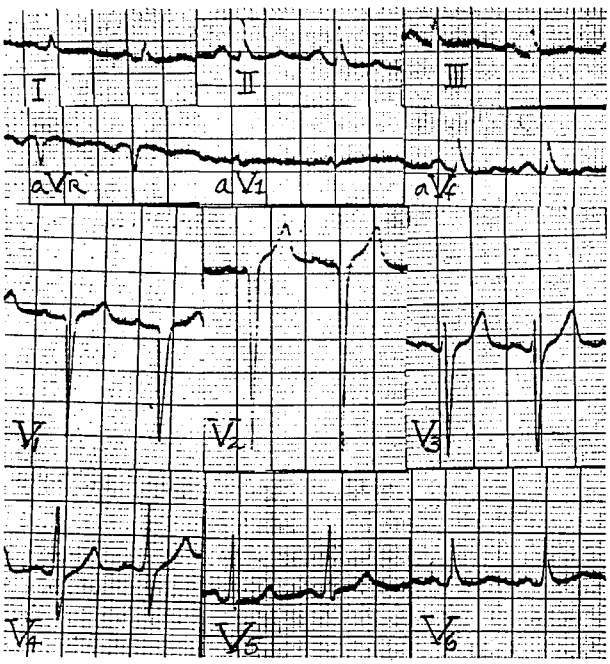

Fig. 6—Electrocardiographic study made about 18 hours after exposure (Case K). Description: Sinus tachycardia. Rate, 110 per minute. PR interval, 0.16 second. QRS duration, 0.09 second. Q-T interval, 0.32 second. Mean QRS axis, plus 60° with transition at V_1. Mean T axis, plus 60°, with T waves upright V_1 through V_6. Absent R wave at V_1 and rudimentary R wave at V_2 with a prominent S wave at V_2.

Impression: Borderline record due to QRS configuration at V_1 and V_2. Rudimentary R waves with a prominent S wave at V_2 may be associated with electrical position or left ventricular hypertrophy or occasionally as residual evidence of anterior wall myocardial infarction. Excluding electrical position, this finding is consistent with electrical preponderance of the left ventricle as commonly seen in anterior infarction or left ventricular hypertrophy. In this case, the electrocardiographic findings are not diagnostic but are consistent with decreased electrical contribution from the right ventricle, resulting in apparent left ventricular preponderance.

gen, blood urea nitrogen, and serum uric acid. (See Section IV, where necropsy findings of early widespread hematologic cell death indicated that much of this rise was a prerenal azotemia.)

A total of 10 blood samples and 3 urine specimens were obtained for diagnostic and radiobiological studies. The results of those studies which contributed to K's medical care are given in Table II. These results showed severe nitrogen retention and an increased production of nitrogenous waste products. When considered together with his continued hypotension, these results led to a rate of intravenous fluid administration which, in retrospect, may appear to have been excessive (Fig. 5). At the time, however, maintenance of adequate blood pressure seemed fundamental to prolongation of life and in particular to promoting renal function. In Section III, results of additional biochemical and hematological studies are tabulated and discussed.

Of all the various determinations made during the course of the illness, changes in white cell count were found to be the simplest and best prognostic indicator of severe irradiation. The virtual disappearance of lymphocytes from the peripheral circulation within six hours of exposure was considered a grave sign (Fig. 7). The clinical course of the patient's illness and associated laboratory findings (Table II), including morphologic changes, differed from those seen in heavily irradiated animals[9] only in the initial hyperpyrexia and absence of terminal hypothermia and specific late neurologic signs of central nervous system disturbance. These differences could have resulted from the supportive therapy, which the animals did not receive.

Twenty-four hours after the accident, a sternal bone marrow biopsy was performed. The marrow appeared watery rather than bloody, and no excessive bleeding occurred. Examination of smears from the marrow (and later fixed sections) showed an almost completely acellular, edematous, hemorrhagic fatty tissue. The finding, together with the rapid onset of lymphopenia, supported the conclusion that a supralethal dose had been received. Accordingly, arrangements were made to obtain as soon as possible fetal and cadaver marrow for transfusion, but the patient's early death precluded its use.

During the course of his second evening,

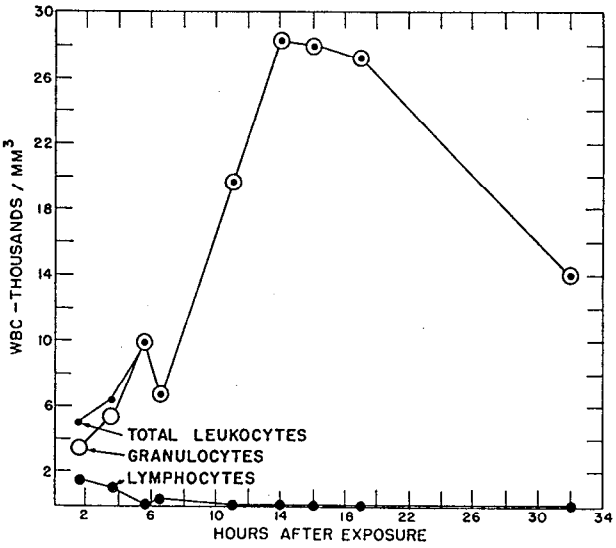

Fig. 7—Peripheral blood leukocyte changes following exposure (Case K).

difficulty was encountered in controlling pain from recurring abdominal cramps, and the patient became increasingly restless in spite of repeated doses of Demerol (50 mg) and sodium luminal (120 mg). He finally became so difficult to control that the intravenous infusions infiltrated extravascularly and were inadvertently interrupted. About 30 hours after the accident (at which time his temperature had been steady at 99.8°F for several hours), he began to sweat profusely and complained of severe diaphragmatic pain, his color became ashen and his pulse irregular. After a 45-minute interval, during which time his nurse had been unable to determine his blood pressure, it was found to be 160/110. Shortly thereafter, he began thrashing about so violently that it was difficult to keep him either in the oxygen tent or in bed, and the intravenous infusions were again disrupted. In spite of administration of oxygen by mask, his lips and nails became cyanotic and his respirations shallow. He again became incoherent and practically unmanageable about 32 hours after the accident. One hundred twenty milligrams of luminal and 10 mg of morphine given intramuscularly quieted him in about 20 minutes. Even with oxygen administration, however, his respirations became slower (18 to 20 per minute, declining slowly to less than 10 per minute) and his pulse was unobtainable. His heart tones were quite distant, slow, and irregular becoming progressively weaker and slower; they suddenly stopped 34¾ hours after the radiation exposure.

Acknowledgments

The technical assistance of Bessie Armstrong, Dorothy Hale, and Lora Belle Hughes is gratefully acknowledged, as well as aid from the Los Alamos Medical Center laboratory and nursing staffs and Los Alamos Scientific Laboratory staff. We wish also to express our appreciation to Lawrence E. Lamb, M.D., Chief of Internal Medicine and principal cardiologist at the Brooks Air Force Base School of Aviation Medicine, for aid in interpretation of the electrocardiograms.

SECTION III

Clinical Pathology and Biochemistry

Donald F. Petersen, Ph.D.
With the collaboration of C. C. Lushbaugh, M.D.

IN VIEW of the massive radiation exposure sustained by K, it was radiobiologically important to investigate a number of biochemical processes not directly related to his clinical management. In most cases, these analyses were conducted simultaneously and under identical analytical conditions with material collected from the less heavily irradiated patient D and from normal subjects. Understandably, the number of additional biochemical investigations performed in this case was severely limited by the small amount of analytical material available. Table III summarizes the details of collection and preservation of samples used in the studies described below. For the most part, significant changes in the biochemical parameters which were studied occurred only in the heavily irradiated patient, K.

Table III—Material Available from Case K for Analysis

Urine		*
Voided 6:30 a.m. 12/31/58		150 ml*
Voided 10:00 a.m. 12/31/58		180 ml*
Voided 2:40 p.m. 12/31/58		260 ml*
From urinary bladder at autopsy 6:30 a.m. 1/1/59		3 ml
Feces		
Formed stool obtained in emergency room approximately 5:20 p.m. 12/30/58		36.4 gm
From diarrhea 5:25 p.m. 12/30/58		64 gm
Blood		
Serum and/or plasma†	6:10 p.m. 12/30/58	
	8:00 p.m. 12/30/58	
	10:00 p.m. 12/30/58	
	3:15 a.m. 12/31/58	
	8:30 a.m. 12/31/58	
	11:15 a.m. 12/31/58	
	12:30 p.m. 12/31/58	
	5:15 p.m. 12/31/58	
Serum obtained at autopsy	6:30 a.m. 1/1/59	
Post mortem clots		

* These samples constituted total voidings during survival time.

† All materials not used immediately were frozen and stored at −20°C in sealed containers.

Alterations in Serum Constituents Following Acute Gamma-Neutron Exposure

Serum Enzymes—Radiation-induced increases in glutamic oxalacetic transaminase (GOT) activity have been reported in sera of irradiated experimental animals and patients receiving localized roentgen therapy.[10] A notable feature of our previous studies was the rapid onset of detectable change in serum enzyme content following exposure. However, no correlation between the dose administered and the magnitude of the change could be demonstrated and, owing to rapid destruction of circulating enzyme by the hepatic parenchyma, GOT measurements were considered unsuitable as a quantitative indication of the extent of radiation injury.[11] Nevertheless, these studies did demonstrate a consistent increase in a number of serum enzymes after exposure to x-rays and gamma-neutron irradiation, and it seemed desirable, therefore, to ascertain the effect of a massive dose of gamma-neutron irradiation on serum enzyme levels in man.

Glutamic oxalacetic transaminase, glutamic pyruvic transaminase (GPT), lactic dehydrogenase (LDH), and malic dehydrogenase (MDH) assays were performed by modifications of existing methods,[12-15] based on the spectrophotometric measurement of the rate of disappearance of reduced diphosphopyridine nucleotide from the reaction mixture at 38°C. All results have been corrected for temperature to facilitate comparison with clinical data where assays are conventionally carried out at room temperature. Measurements of serum lactic and malic dehydrogenase were discontinued because of limitations in available analytical material. Results of these analyses are summarized in Table IV. K's serum GOT activity was approximately twice normal in the earliest sample obtained one and one-half hours after irradiation. Subsequent analyses showed a gradual increase to six times normal during the succeeding 23 hours. Serum GPT activity did not exceed

*Table IV—Serum Enzyme Content**

Sample	Hours After Exposure	SGOT	SGPT	LDH	MDH
Case K	1½	53	31	676	310
	3½	42	29	.....	.....
	5½	55	29	.....	.....
	10½	101	32	.....	.....
	16	55	25	.....	.....
	19	116	24	.....	.....
	25	160	37	.....	.....
	Post mortem	429	58	.....	.....
Case D	4	17	44	604	136
	17	23	38	.....	.....
Control	....	26	24	388	113
Normal range		14-35	8-30	200-600	50-104

* Assays based on disappearance of reduced diphosphopyridine nucleotide at 340 μ, 38°C, corrected to reaction rate at room temperature for comparision with conventional clinical values.

Unit = decrease in optical density of 0.001/min/ml of serum.

normal levels before death, but both enzymes exhibited characteristic postmortem increases. These data are in contrast with negative findings reported after smaller doses of radiation sustained in the Oak Ridge Y-12 accident[6] but are in agreement with our previous experience with monkeys exposed to fission neutrons.[11] K's values for serum enzyme activity were undoubtedly modified by retention of most of the large volume of intravenous fluids administered. Since it has been shown[11, 16] that these serum enzymes are in equilibrium in the total extracellular fluid space rather than in the circulating fluid volume, actual serum enzyme values were probably one-third higher than the values obtained. Serum lactic and malic dehydrogenase values were also slightly elevated in the initial serum sample in comparison with the control value determined simultaneously. No significant abnormalities in serum enzyme content attributable to radiation injury were noted in sample obtained from the second patient, D.

The hypothesis has been advanced that necrosis of irradiated cells results from destruction of proteolytic enzyme inhibitors and the subsequent action of liberated cathepsins.[17] Results of serum inhibitor assays, summarized in Table V, indicated that K's serum inhibitor content remained within normal limits during his survival time except for a brief interval approximately 19 hours after irradiation, when values increased to twice normal and then rapidly declined to normal levels. These data were in essential agreement with experimental results obtained in rats given 1000 r of total-body x-radiation, where a transitory rise in serum inhibitor titer occurred 48 to 72 hours after exposure.[17]

Table V—Serum Protease Inhibitor

Sample	Hours After Exposure	Inhibitor Units
1	1½	43
2	3½	40
3	5½	50
4	10½	53
5	16	38
6	19	39
7	25	50
8	Post mortem	35
Normal range		37-65

Table VI—Serum Proteins 25 Hours After Exposure

Sample	Total Protein (gm %)	Albumin (gm %)	Globulin (gm %)	γ-Globulin (gm %)	A/G Ratio
Case K	5.2	3.4	1.8	0.4	1.9
Normal range	6-7.5	4.5-5	2-2.5	0.35-1.5	1.2-1.5

Serum Proteins—Electrophoretic examination of two serum samples obtained from Case K at 16 and 25 hours after exposure revealed only two abnormal features. The first was a shift of the alpha globulin peak away from the albumin peak, and the second was a slight diminution in the gamma globulin fraction. The significance of the alpha globulin shift was not readily apparent, but the reduction in gamma globulin has been observed repeatedly in experimental animals and man and appears to be a consistent effect.[2, 18, 19] Case D's serum electrophoretic pattern was not remarkably different from normal with the exception of a slight reduction in the gamma globulin fraction.

Total protein, albumin, globulin, and gamma globulin estimations (Table VI) indicated a general reduction in serum protein content, which was undoubtedly a contributory factor in the severe hypotension which persisted throughout the clinical course of Case K.

Serum Antibodies—The only antibodies demonstrable in K's serum at room temperature were anti-A and anti-B. A variety of techniques were used: routine "back typing" of the serum in test tubes, and checking for atypical antibodies against a panel of eight group O cells designed to demonstrate any one of 23 blood group factors (C, C^w, D, E, V, c, e, K, k, fy^a, fy^b, Js, Le^b, Jk^a, Jk^b, M, N, S, s, P, Lu^a, and Lu^b). The tests were carried out in saline, which was then converted to the indirect Coombs' test. The panel was also ficinized and then converted to the indirect Coombs' reaction after being exposed to K's serum. When group O cells were exposed to K's serum in the presence of group AB serum as a source of human complement in the cold (ice water), they gave a positive indirect Coombs' reaction. This was interpreted as being an example of the "incomplete" cold antibody described by Dacie.[20] There was insuf-

ficient serum to check whether this was a gamma or nongamma antibody.

Alterations in Urinary Constituents Following Acute Gamma-Neutron Exposure

As indicated in the discussion of renal effects in the preceding section, K was anuric for the first 14 hours after exposure (Table III). During the next nine hours, three urine samples were obtained, representing a total volume of 590 ml. The character of these specimens varied markedly: The first was dark, cloudy, and highly concentrated; the second and third were clear and had specific gravities approaching 1. Thus, although a pooled specimen represented a total collection time of 23 hours, direct comparison of analytical results with usual 24-hour collection values did not appear to be justified and values were, therefore, included which permitted comparison on the basis of concentration of the various constituents per unit volume.

Catechol Amine Excretion—K's failure to respond to massive doses of pressor amine was of particular interest. The total amount administered (*l*-arterenol bitartrate) was equivalent to 48 mg of *l*-norepinephrine over a period of 26 hours. Excretion of catechol amines in the urine of patients K and D was measured by established bioassay procedures.[21] The results of these estimations, which are summarized in Table VII, showed K's norepinephrine excretion to be four to five times greater than that determined for normal individuals. This increase undoubtedly reflected administration of *l*-norepinephrine in the infusion, but it is remarkably small in view of the massive dose. Since the pharmacologic response was limited despite the large dose, several mechanisms were considered. Either accelerated destruction in situ, diminished response of effector cells, or local extravasation due to venous incompetence, rather than excretion, must account for the apparent disappearance of active pressor amine. Pressor amine values on D's pooled specimen did not deviate appreciably from normal.

Urinary Amino Acids—Further evidence for a disturbance in nitrogen metabolism was indicated by marked increases in urinary alpha-amino nitrogen excretion in the initial specimens of both Case K and Case D (Table VIII). Quantitative estimations of amino acids appearing in the urine are as yet incomplete, but a preliminary report[22] indicates that they are in essential agreement with qualitative identification by one- and two-dimensional paper chromatography (Table IX). In agreement with a previous report,[6] serine was present only in trace amounts in the heavily irradiated patient,

Table VII—Urinary Excretion of Catechol Amines

Sample	Hours After Exposure	Epinephrine (μg/24 hr)	*l*-Norepinephrine (μg/24 hr)
K pooled	0-22	16.9	233.9
D pooled	0-15	5.8	66.7
Normal range		10-20	25-50

Table VIII—Urinary Amino Nitrogen

Sample	Hours After Exposure	Amino Nitrogen (mg %)
K_1	14	1.120
K_2	17	0.915
K_3	22	0.720
K pooled	0-22	0.860
D_1	4	0.577
D_2	15	0.800
D_3	72	0.766
$Control_1$	....	0.400
$Control_2$	....	0.426
Normal range		0.1-0.4

Table IX—Qualitative Identification of Urinary Amino Acids

Amino Acid*	Case K	Case D
Taurine	Prominent	Prominent
Aspartic acid	+	+
Serine	Trace	+
Glycine	+	+
Glutamic acid	+	−
Threonine	−	+
Alanine	+	+
σ-Aminoisobutyric acid	Prominent	Trace
Methionine	+	+
Phenylalanine	+	+
Isoleucine	+	−
Leucine	+	+
Norleucine	+	+

* Solvents: *n*-butanol-formic acid-water (75:15:10), *n*-butanol-propionic acid-water (61:31:8), phenol-water (saturated at 20°C).

K, while excessive amounts of taurine were contained in the specimens of both patients. These data thus offer further confirmation of observations originally made by Kay and Entenman[23] in experimental animals, which indicated a marked increase in taurine excretion following relatively small doses of radiation. The prominence of taurine in the early urine samples of Case D, the less heavily irradiated patient, was the only significant biochemical change observed. Beta-aminoisobutyric acid was readily detected in the urine of K but was barely detectable in the urine of D.

Urinary DNA Metabolites—The appearance of beta-aminoisobutyric acid in the urine of irradiated patients has been interpreted as a reflection of accelerated destruction of deoxyribonucleic acid (DNA) in radiosensitive cells.[24, 25] Increases in total cysteine-sulfuric

Table X—Urinary Cysteine-H_2SO_4 Positive Material

Sample	Hours After Exposure	Mg %
K_1	14	110.0
K_2	17	39.0
K_3	22	28.0
D_1	4	47.5
D_2	15	30.0
D_3	72	43.0
D_4	96	41.0
D_5	120	45.0
$Control_1$	....	44.5
$Control_2$	....	43.0
$Control_3$	....	25.0
$Control_4$	....	42.0
$Control_5$	....	42.0

acid-reacting material[26] have been reported in animals following irradiation,[27] which presumably also reflect a radiation-induced defect in DNA metabolism. Results of measurements of total Dische-positive substances in urine specimens of K and D, as well as a number of normal subjects, are shown in Table X. The first urine specimen voided by K contained approximately twice the amount of cysteine-sulfuric acid-positive material found in normal individuals, while all subsequent samples, as well as voidings from patient D, fell within normal limits. Paper chromatography of a pyridine ethanol (v/v) eluate of urine adsorbed on activated charcoal indicated that the major component of the Dische-positive material in K's urine was deoxycytidine. This conclusion was further substantiated by rechromatography of the ultraviolet-absorbing material corresponding in R_f to deoxycytidine, together with authentic tritiated deoxycytidine of high specific activity.[28] A single spot of ultraviolet-absorbing material containing the radioactive marker confirmed the identity of the compound.

Urinary Creatine and Creatinine—Contrary to recent reports of creatinuria in irradiated experimental animals,[29] no appreciable amounts of creatine were found in the urine of Case K. The analyses in Table XI, performed by the method of Taussky,[30] indicated a greater amount of creatinine in K's first voiding than in subsequent specimens, but the amount contained in the pooled specimen, which approximated a 24-hour collection, was within normal limits. The fact that serum creatinine did not increase appreciably indicated that the aberration in creatine metabolism noted in irradiated animals did not occur during the brief survival time of Case K. A small amount of creatine was found in the 72-hour voiding from D, but urinary creatinine content was normal.

Table XII—Urinary Leucine Aminopeptidase Activity

Sample	Hours After Exposure	β-Naphthylamine (μg/ml/2 hr)	β-Naphthylamine (mg/24-hr sample)
K_1	14	0.52	.....
K_2	17	0.01	.....
K_3	22	0.004	.....
K pooled	0-22	.....	21
Normal range		.....	50-175

Leucine Aminopeptidase—Leucine aminopeptidase is an exopeptidase secreted by the intestinal mucosa, which occurs normally in serum and urine and increases markedly in the urine of patients with metastatic carcinoma involving the bowel.[31] Since the dose of ionizing radiation sustained by K was sufficiently high to anticipate considerable intestinal damage, urinary leucine aminopeptidase activity was determined to ascertain whether such injury would also produce alterations in amount of enzyme excreted. Calculations, based on data shown in Table XII for a pooled sample for the 22-hour period during which voidings were obtained, indicated a normal amount of enzyme activity. However, it was found that virtually all the leucine aminopeptidase activity was in the initial specimen, voided 14 hours after exposure. Since the mode of excretion of the enzyme is not known and the identity of the urinary and intracellular enzymes has not been definitely established, these data are reported only as isolated observations. No attempt was made to interpret their significance in the acute radiation syndrome.

Table XI—Creatine and Creatinine

Sample		Hours After Exposure	Creatine (mg %/sample)	Creatinine (mg %/sample)	Creatine (gm/24 hr)	Creatinine (gm /24 hr)
Urine	K_1	14	0.0	436	.....	.....
	K_2	17	4.0	237	.....	.....
	K_3	22	0.0	217	0.01	1.66
Urine	D_1	4	0.0	208	.....	.....
	D_2	15	6.0	254	.....	.....
	D_3	72	20.0	206	0.249	1.56
Normal range			.....	.....	0.000	1.0-2.0
Serum	K_4	10½	.....	2.0	.....	.....
	K_5	16	.....	2.0	.....	.....
	K_6	19	.....	2.1	.....	.....
Normal range			.....	1.0-2.0	.....	.....

Acknowledgments

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SECTION IV

Gross and Microscopic Pathology and Neuropathology

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THREE hours after the death of patient K, a complete necropsy examination was made.

Gross Pathology

External Appearance

The body was that of a middle-aged, male adult weighing 172 lb. and measuring 71 in. in length. The only external evidences of injury were numerous needle puncture marks in both anticubical fossae and in the large veins of the forearms and lower legs, and two recently sutured, 2-cm long, surgical incisions over the manubrium sterni. Both forearms bore blue tatooed designs. There was no erythema of the anterior abdominal wall, and the conjunctivae were no longer hyperemic as they were at the time of admission to the hospital.

Internal Organs

Abdominal Cavity—The subcutaneous tissues and muscles were quite wet, and there was about 250 cc of clear, thin fluid in the abdominal cavity. The peritoneal surfaces were smooth and glistening except those of the first loop of the jejunum, the gastric pyloric bulb, and the surfaces of the left lobe of the liver. These areas were dusky-rose in color and contained minute, petechial hemorrhages. Such hemorrhages were also present in the epigastrium and in the parietal peritoneum. The domes of the diaphragms were at the fourth rib on each side. There was no excessive gaseous distention of the bowel. The majority of the intestines were small in diameter except for the hyperemic first portion of the jejunum. The stomach was moderately dilated, containing about 800 cc of gray, mucinous, gastric contents devoid of food or other recognizable debris.

Pleural Cavity—The lungs collapsed as the chest was opened. The pleural spaces contained less than 10 cc of fluid. The anterior portions of both lungs were well aerated, while the posterior areas were atelectatic. There were no pleural adhesions.

Pericardial Cavity—The pericardial cavity contained about 75 cc of cloudy yellow fluid which contained fibrin that clotted on standing. The right side of the heart was dilated and filled with blood. The left heart was in systole. The pericardium and epicardial fat over the right ventricle which lay in the midline, just above the xiphoid process, contained numerous petechial and small ecchymotic hemorrhages. These hemorrhages were also present in the right auricle and that portion of the right atrium which was most anterior. The distended right auricle appeared to contain thrombosed blood.

Mouth and Pharynx—The oral mucous membranes were normal in appearance and showed no hemorrhages.

Plate I—Photomicrographs of histologic sections obtained at necropsy

A. Femoral bone marrow island (hematoxylin-eosin, ×1,250) consisting of macrophages containing yellow hemosiderin, metamyelocytes, myelocytes, normoblasts, and erythroblasts. No necrotic cells or debris are present. The most primitive and most mature cells are no longer demonstrable.

B. Femoral bone marrow (Prussian-blue reaction, ×330) showing reticular macrophages loaded with bright blue irregular masses of iron among the pink-staining myelogenous cells and orange-staining erythrocytes.

C. Peripheral pulmonary alveoli (Prussian-blue reaction, ×200) containing masses of clumped macrophages filled with ingested material and erythrocytes which stain positively for iron.

D. Chromatin debris in a cortical follicle of a mediastinal lymph node (hematoxylin-eosin-azure, ×320).

E. Thymus gland (hematoxylin-phloxine, ×260) showing small focus of remaining lymphocytic debris, and epithelial-like groups of reticular endothelial stromal cells.

F. Typical remnant of a lymphoid follicle in the spleen (hematoxylin-eosin-azure, ×330) showing the apparent hypertrophy of the central reticular macrophages.

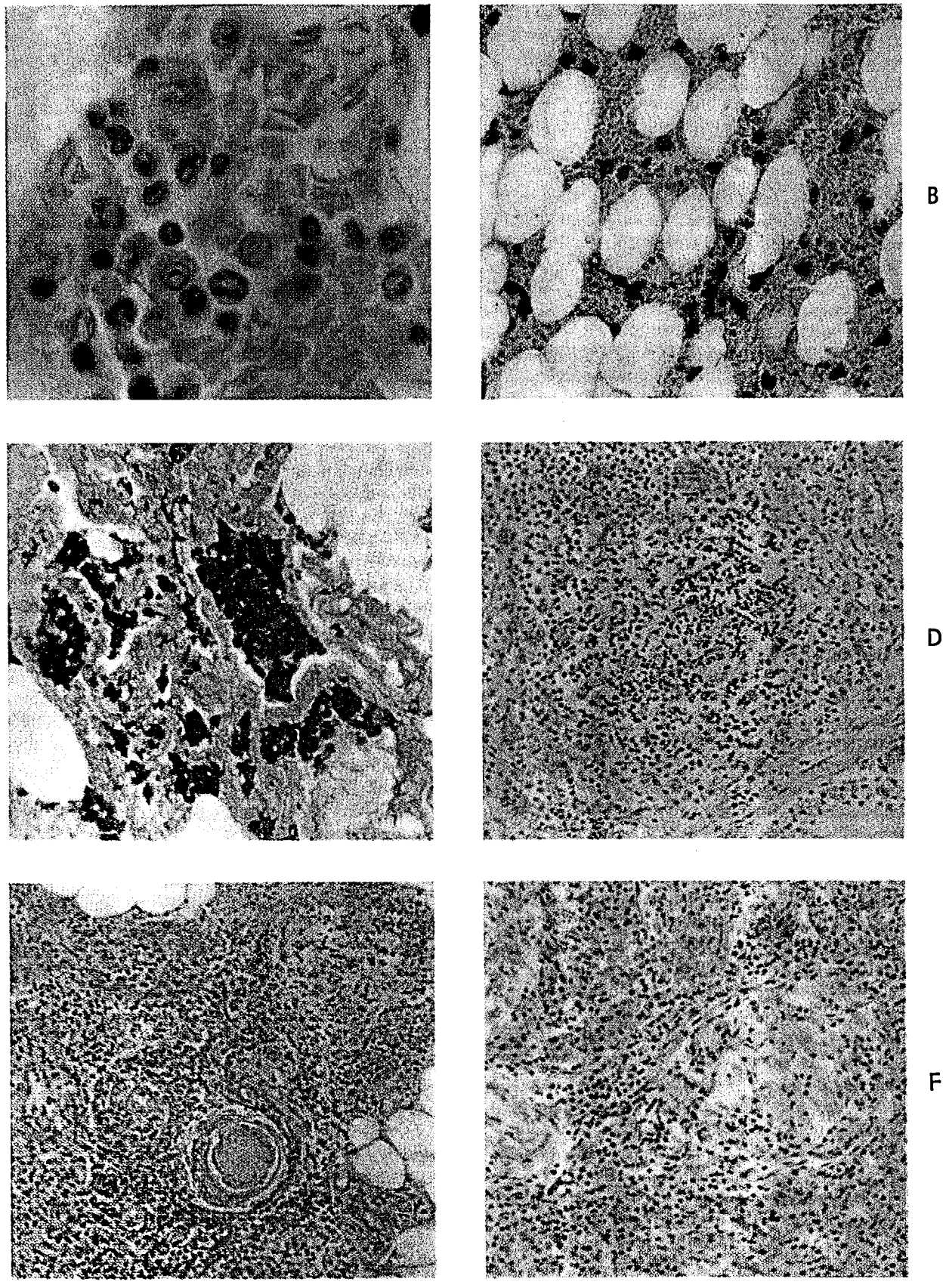

Plate I—See description at left.

Thyroid and Thymus—The thyroid was normal in size, shape, and consistency. There were some small lymph nodes associated with it which appeared grossly to be normal. The site of the thymus was filled with fat in which there could be seen some minute remnants of thymic gland. There were no hemorrhages in the thymic fat or other mediastinal structures.

Trachea and Main Bronchi—The mucosa of the trachea and main bronchi appeared edematous, but these tubes did not contain frothy edema fluid and no such fluid could be expressed from the lungs. The tracheobronchial lymph nodes were anthracotic. One in the left hilus contained a 5-mm-diameter, round, calcified nodule. There was a similar calcified nodule under the pleura of the left upper lobe in the intralobular fissure. Besides their black color, these hilar nodes were conspicuous only because of their soft, fleshy consistency.

Lungs—The right lung weighed 400 gm; the left lung, 450 gm. Their posterior portions were atelectatic, while their anterior portions were well aerated. There were no areas of consolidation or hemorrhage. The left lung contained the Ghon complex described above. The lung parenchyma contained irregular anthracotic blotches (up to 2 cm in size) uniformly distributed throughout.

Heart—The emptied heart weighed 400 gm. The tricuspid, pulmonary, and aortic valves were normal. The mitral valve showed slightly more than moderate thickening of the free margins, and some shortening and thickening of the chordae tendinae were seen. The right ventricle averaged 4 mm in thickness; the left, 18 mm. The right ventricle and atrium were dilated. The auricle of the right atrium contained thrombosed blood between the pectinate muscles, and there were many pericardial and intramuscular linear petechial hemorrhages in the right atrium and right ventricle. The left ventricle did not have these hemorrhages except in the interventricular septum, where they were most prominent just below the aortic valves. The coronary blood vessels showed minimal atherosclerosis and no thrombosis. The aorta contained only a moderate amount of atherosclerosis in its lower third. The great veins were normal.

Liver—The liver weighed 1,950 gm. It was acutely congested, as evidenced by its dark color and by extremely prominent central areas which gave its cut surfaces a typical nutmeg pattern. The peritoneum over the left lobe of the liver was slightly opaque, and there were small areas of hemorrhage under it. On section, these areas appeared to be continuous with the vascular spaces of the liver.

The gallbladder was distended with about 50% more bile than is normal. The bile, however, was easily expressed through the papilla of Vater and was green, thin, and mucinous. There were no gall stones.

Pancreas—The pancreas appeared normal throughout its length.

Spleen—The spleen weighed 116 gm. Its capsule was wrinkled, and the organ was exceptionally flabby. On the cut surfaces, the connective tissue trabeculae were unusually prominent and the pulp, while bloody, appeared to be much more fluid than usual. No lymphoid aggregates could be discerned.

Gastrointestinal Tract—The stomach was distended with slightly less than 1 liter of thin, mucinous, gray fluid with a typical gastric odor. The peritoneum over the antrium was dusky-rose and contained many hemorrhages, while under this area the mucosa anteriorly and posteriorly also contained numerous petechial hemorrhages. These hemorrhages were seen only within a radius of 5 cm from the pylorus. The mucosa of the duodenum and jejunum appeared pale and edematous except in the region of the first 10 cm of the jejunum, which was moderately dilated and had a hyperemic mucosa. There were, however, no grossly discernible ulcerations in this or any other portion of the intestinal tract. The jejunum became progressively smaller in diameter caudally, so that the ileum and colon were quite contracted and free of fecal material and intestinal chyme other than mucus. The appendix appeared to be atrophic. The mesenteric lymph nodes were small and difficult to discern from the surrounding fat, since both were pinkish-yellow.

Adrenal Glands—Both adrenals were normal in size and shape. They weighed (with a small amount of fat) 7 gm each. Surfaces made by cutting failed to reveal any abnormality. The cortices appeared normally lipid-rich.

Kidneys—The right kidney weighed 130 gm; the left, 140 gm; and the capsules stripped with ease. The parenchymal blood vessels were hyperemic. The surfaces made by cutting bulged slightly so that the edges could not be completely approximated. The markings of the cut surfaces were those of grossly normal kidneys.

Gastrourinary Tract—The renal pelves, ureters, and urinary bladder were normal in appearance. The urinary bladder was contracted and contained only about 3 cc of yellow, cloudy urine. The lining of the bladder was thrown up into folds and contained no hemorrhages.

Generative Organs—The prostate was normal in size, although there was a hypertrophic

median bar. This median bar was knobby and had a mulberry-like appearance.

The testes were normal in size but small. The testicular tubules could be teased apart easily.

Skeletal and Muscular System—There were no gross abnormalities of the bone other than the recent surgical biopsy of the manubrium sterni. The bone marrow appeared hemorrhagic and devoid of its normal, fleshy appearance.

The muscles were dark red and much more moist than normal. Rigor mortis was exceptionally strong and the muscles more contracted than usual. The muscles and subcutaneous tissues of the arms and thorax showed extensive rigor mortis and swelling, but this was not evident in the lower portion of the torso and the lower extremities (five hours post mortem).

Cranial Cavity—The brain stretched the dura mater tightly and was congested and swollen. It weighed (including the dura) 1,600 gm. The gyri were flattened and the sulci obscured. The blood vessels were prominent. There was a small pressure conus in the region of the uncus hippocampi. A section of the brain revealed congestion of the white matter of the cerebrum and, in the parietal lobe, several prominent red-tinged streaks along the course of the vessels.

The pituitary gland was quite hyperemic, fleshy, and soft in appearance.

Microscopic Pathology

Fixation of the hematopoietic tissues was obtained with Maximow's Zenker-formol solution, of the brain and other neurological tissues with neutralized formalin, and of the other organs with mercuric chloride formalin solution. In addition to hematoxylin and eosin, Maximow's hematoxylin-eosin-azure stain, a Prussian-blue stain for iron, the periodic acid Schiff reaction, and Verhoeff's elastic tissue stain were used in the histologic preparations.

Bone Marrow—Sections were obtained from marrow in the proximal femur, vertebral bodies, and sternum. The marrow specimens from these areas were histologically quite similar, but the staining of the femoral marrow was best since no decalcification was necessary. A search for mitoses failed to reveal a single one in any of the sections. Similarly, there were no cells of either the myelocytic or the erythropoietic series which showed any destruction in situ (Plate I *A*). Bone marrow islands contained more red cells than nucleated cells, and there was a pronounced decrease in polymorphonuclear leukocytes and primitive cells in particular. Eosinophilic myelocytes were prominent along with the very primitive reticuloendothelial macrophages which contained frequent irregular-sized blobs of gold pigment and many pyknotic cell remnants. The Prussian-blue stain for iron made these cells stand out prominently, since the gold pigment and the diffuse brown cytoplasm of these cells now stained positively blue (Plate I *B*). Occasional megakaryocytes were found to have degenerate, pyknotic nuclei, but nuclear debris were never found freely in any of the sections. When present, such debris was always inside the reticular histiocytes. The vertebral marrow showed slightly severer depletion than the femoral marrow, as many of the spaces formerly filled by cells were found here to contain protein-rich edema fluid. The majority of the nucleated cells here were erythro- and normoblasts. As in the femoral marrow, no mitotic activity was present, and the primitive hemocytoplasts in all series were conspicuously difficult to find.

Lungs—There were occasional areas of alveoli containing thin, protein-containing fluid. The lungs were poorly expanded, and there were many areas of focal atelectasis interspersed with areas where the alveoli were ballooned by emphysema with the walls being ruptured. Many areas were found where there were relatively huge atrial cavities. In the alveoli near the periphery of the lungs and in the atelectatic alveoli adjacent to the bronchi and interlobular septa there were many macrophages collected in masses containing yellowish-green granular debris, some of which still had the form and appearance of erythrocytes. These macrophages stained positively for diffuse and particulate iron (Plate I *C*). They were occasionally found associated with macrophages containing carbon pigment in the interstitial tissue. In addition, the alveolar septa contained an abnormal number of polymorphonuclear leukocytes. There was so much iron present in the macrophages that it was difficult to believe this picture resulted from the radiation. No gross hemorrhages were found. The fact that this man had had pneumonitis in the past was evidenced by epithelization of many of the peripheral alveoli, and many of the macrophages were associated with this epithelium. On the other hand, it was in these areas that one saw degenerating granulocytes, lymphocytes, and extravasated red cells being ingested by the macrophages. Many of the iron-laden macrophages were in the bronchioles and bronchi, apparently in the process of being coughed up. Sections containing pleura showed damaged (pyknotic and crenated) serosal nuclei and many pyknotic and degenerating lymphocytes and granulocytes in the subpleural connective tissue.

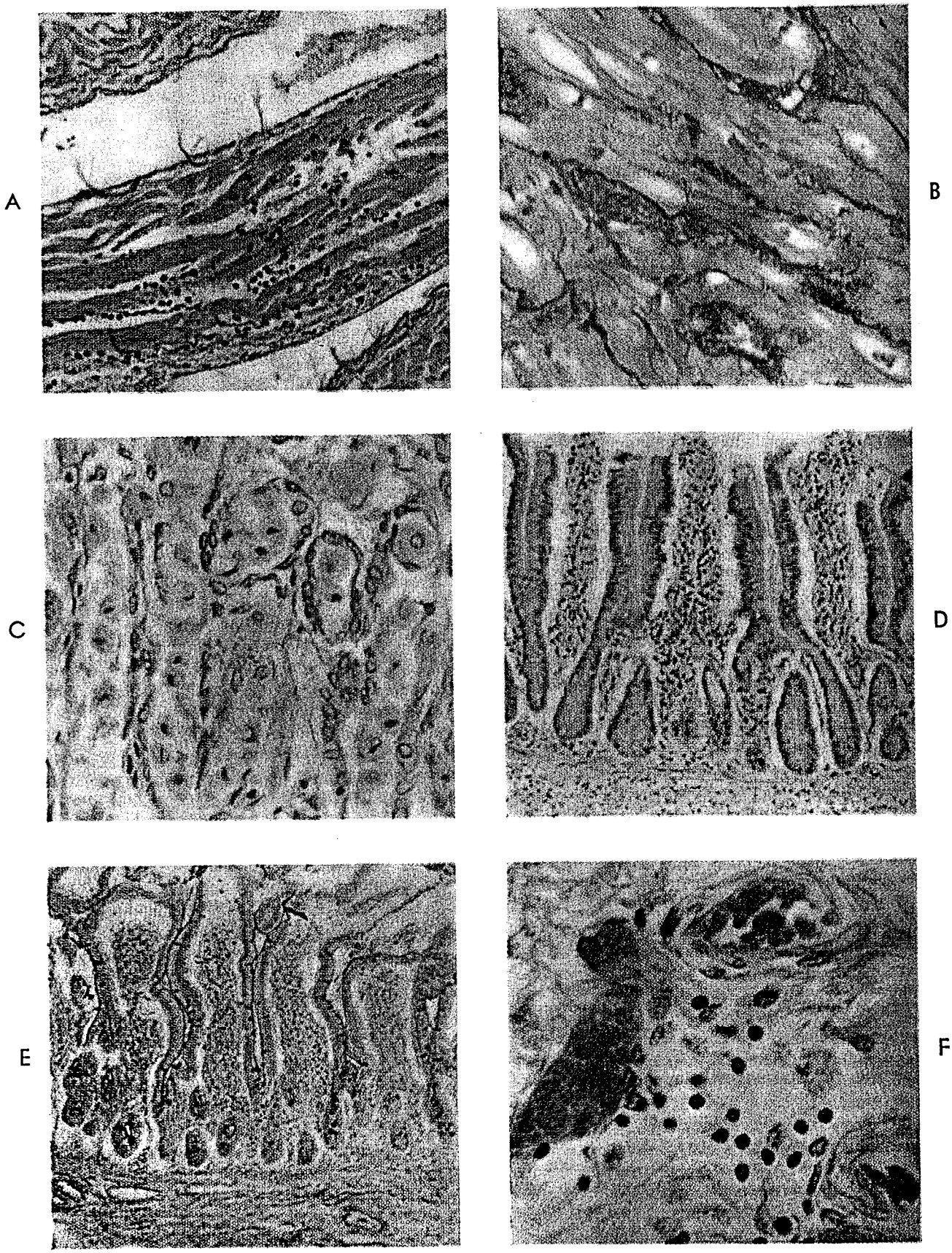

Plate II—See description at right.

Tracheobronchial Lymph Nodes—Mucus in the bronchi contained desquamated columnar epithelial cells, macrophages laden with iron, and a few exudated granulocytes. Whether blood was present ante mortem could not be determined. The peribronchial lymph nodes contained carbon dust in macrophages in areas of increased collagenous connective tissue. The iron stain revealed foci of iron-containing macrophages. These foci were usually in or around areas of anthracosis. The cortical sinuses contained macrophages, but very little stainable iron, although some dark blue blobs were present. In another lymph node from the hilus, iron-laden macrophages were found most prominently in the peripheral sinus and the perifollicular sinuses.

Lymph Nodes (in general)—The lymph nodes in general were markedly atrophic owing to the recent loss of lymphocytes. The architecture was quite recognizable, and the reticulum and the sinusoidal macrophages were particularly prominent. There was a variable amount of lymphocytic debris present; a periaortic node from the celiac axis showed the least amount of lymphocytic debris but the greatest amount of atrophy. The reticular centers of follicles were not prominent, and there was no apparent increase in sinusoidal phagocytosis, although some leukocytes were present. Many of the remaining lymphocytes were small and pyknotic, although many normal-appearing lymphocytes were present. Relatively more debris was found in the lower abdominal lymph nodes, together with an occasional reticular center. Occasional nodes from this region contained large numbers of leukocytes in the sinuses. Lymph nodes from higher up in the mediastinum and lower down in the abdomen showed follicles rich in chromatin dust (Plate I *D*).

Thymus—The thymus consisted of thin strands of reticular cells with occasional large Hassall's bodies. Either there were very few lymphocytes before the accident or, as is more likely the case, since macrophages containing vestiges of cellular debris could be seen, the lymphocytes present at the time of the accident were destroyed and removed. The lymphocytes still present at the time of death were extremely pyknotic, and many peculiar "explosion figures" were seen which typified continuing rhexis. Some of the reticular cells were hypertrophied and arranged in epithelial-like clumps (Plate I *E*).

Spleen—The lymphoid follicles were greatly depleted. They were now composed of large reticular cells, a few scattered lymphocytes, the majority of which were normal in appearance, and numerous large macrophages containing vacuoles (Plate I *F*). The red pulp was composed of islands of erythrocytes, among which were many pyknotic white cells. The sinusoids, however, were far from being completely distended, and there were many areas in the pulp where there were no red cells other than a few apparently dead ones mixed among degenerating leukocytes. The reticuloendothelial cells in these areas were large and often filled with red and white cell debris. The stain showed such macrophages staining positively for iron, but not to the same extent as in the bone marrow and lungs. In addition, in the relatively bloodless areas of the pulp there were diffuse, fine reticular-like fibers which stained positively for iron.

Appendix—The appendiceal mucosa was hyperchromatic, and the lamina propria was lymphoid-poor. The submucosal follicles contained large reticular centers in which a few of the reticular histiocytes still contained chromatin dust. These centers were surrounded by pyknotic, crenated lymphocytic nuclei. The mucus of the lumen was free from fecal debris but contained masses of degenerating leukocytes. An occasional vein of the mesoappendix contained fibrinous leukocytic clots.

Heart—The serosal cells of the pericardial surface were swollen and bead-like with minute strands of fibrin adhering to them. The connective tissue bands between the fat cells were widened by abundant protein-rich edema fluid. There was fibrinoid necrosis of the small collagenous fibers immediately beneath the serosa, where granulocytes had accumulated in minute foci, often around small arterioles. These ac-

Plate II—Photomicrographs of histologic sections obtained at necropsy (continued)

A. Interstitial accumulation of granulocytes between myocardial muscle fibers in the base of the right ventricular wall (Verhoeff's elastic connective tissue stain, ×330).

B. Swollen Purkinje myocardial fibers from the right atrial appendage showing perinuclear vacuolation and interfibrillar edema (Verhoeff's stain, ×500).

C. Gastric mucosa (PAS stain, ×650) from anterior wall of stomach showing intraglandular sloughing of pyknotic parietal cells.

D. Duodenal-jejunal mucosa (hematoxylin-eosin, ×210) showing loss of epithelium of villous tip, decreased cellularity of the lamina propria, absence of mitoses, and absence of signs of epithelial necrosis.

E. Lower jejunal mucosa (PAS stain, ×125) showing edema of villa and disruption and beginning intraluminal loss of the epithelium.

F. Leptomeninges of the cortex (hematoxylin-phloxine, ×450; AFIP 905226-15) showing perivascular exudation of lymphoid cells and leukocytes, and activated histiocytes containing blood pigment.

cumulations of leukocytes were also found in the pericardium immediately adjacent to the myocardium and in the interstitial connective tissue of the right myocardium itself, along with diffuse irregular hemorrhages (Plate II *A*). These exudates were oriented in a hit-or-miss fashion and were most frequent in sections from the base of the right myocardium, but infrequent in sections from the apex and left ventricle. Interstitial hemorrhages were common among the muscle fibers of the right auricular appendage and atrium.

The myocardial fibers did not show loss of striations or any other degenerative signs, but the Purkinje fibers of the right atrium were unusually swollen in appearance (Plate II *B*). The nuclei of these fibers were swollen and irregularly enlarged, misshapen, and surrounded by large clear spaces, which were interpreted as perinuclear accumulations of edema fluid. Sections obtained from the same areas of the heart of a man of similar age, muscular activity, and history of accidental sudden death failed to show this remarkable swelling at all. Interstitial edema was present throughout the entire myocardium and seemed to explain the unexpectedly heavy weight of the emptied heart (400 gm). The blood of the myocardial vessels was low in leukocytes, but the thrombi between the pectinate muscles of the right auricle and atrium were composed principally of granulocytes and fibrin.

Liver—The histologic appearance of the liver was quite different from what was anticipated from its gross appearance. The central areas did not show much atrophy, and the engorgement seen grossly was apparently lost during fixation. The iron stain showed relatively little iron deposition. There was a yellow-brown pigment in the central hepatic cells which failed to stain positively for iron. The hepatic cells contained fine granules of iron, but the most strongly positive areas were found in occasional macrophages located in the midcentral zones. No macrophages were found which stained as positively for iron as those of the bone marrow and lungs. Aside from the early central atrophy, there was no evidence of primary or secondary hepatic cell injury.

Kidneys—The convoluted tubules showed a slight amount of cytoplasmic sloughing, while in the medulla, occasional tubules were filled with eosinophilic, cytoplasmic debris.

Gastrointestinal Tract—Esophagus: The esophageal epithelium was covered with a thin layer of poorly stained erythrocytes. There was a well-defined basal, glomerular, and squamous layer. A search along the basal layer showed that no mitoses were present. There was an occasional vacuole containing a lymphoid-like cell in the epithelium, while the basal cells appeared to be differentiating into squamous cells. The nuclei were swollen and more vesicular than normal, and even the most basal of these cells appeared to have developed intercellular bridges which were accentuated by intercellular edema. In the subepithelial connective tissue, there was an increase in the numbers of lymphoid and polymorphonuclear cells. An occasional lymphoid follicle was present containing chromatin dust which was both free and in macrophages. There was a reduced number of normal-appearing large and medium-sized lymphocytes in these follicles. There was no true arteritis or phlebitis. The smooth muscle coats were edematous. The blood in some of the smaller vessels of the periesophageal mediastinal connective tissue stained opaquely as though it were congealed. In these vessels there was seen an occasional leukocyte undergoing rhexis.

Stomach: The gastric mucosa was, in general, well preserved. The most superficial portions showed foci of autolytic changes. In the best-preserved areas, many acid-secreting eosinophilic chief cells could be found free in the lumen of the glands. These sloughed cells usually had crenated, pyknotic nuclei, although the nuclei were occasionally swollen and vesicular (Plate II *C*). These changes were seen in the stomachs of the heavily irradiated monkeys as one of the earliest signs of heavy radiation exposure.[32] The zymogen cells appeared to be unaffected. The subperitoneal connective tissue was extremely edematous. In this edematous fluid, there were occasional leukocyte-like cells rimmed by dark blue to purple, opaque material, irregular in outline which gave them a "fried egg" appearance. There was an increase in leukocytes in the connective tissue here, and occasional veins were occluded by fibrin and leukocyte masses. The blood vessels themselves did not appear to be chemotactic. Sections from the posterior gastric wall did not show a similar necrosis of the parietal cells.

Duodenum: The most superficial layers of the duodenal mucosa were missing. Fragments of epithelial strips were lying free in the lumen, and occasionally a well-preserved villus with epithelium could be found. Such villi showed a loss of small lymphocytes from the connective tissue core. There was shrinkage of the core with condensation of the reticular cells and histiocytes (Plate II *D*). Scattered among these cells were occasional pyknotic remnants of lymphocytes and granulocytes. Such remnants could also be seen in the overlying epithelium, which

contained occasional mucous vacuoles. These mucous vacuoles were found also in the deepest cells of the crypts. No mitoses were present here. Outside of the pyknotic nuclear remnants, the only other change in the crypts consisted of nuclear ballooning. Necrosis of the crypt epithelium was not found. Brunner's glands showed cells which were occasionally without a nucleus or with a distorted and pyknotic one. Most, however, appeared unaffected.

Jejunum: The section of jejunum was taken from the area of subperitoneal hemorrhage and edema. The extravasated red cells could be seen diffusely through the subperitoneal edematous connective tissue, and there had been a slight leukocyte exudation. In addition, in and around some of the smaller blood vessels leukocytic, nuclear debris could be found along with leukocytic nuclei elongated in the process of diapedesis. Some of these vessels were occluded by opaque masses of erythrocytes in which it was difficult to delineate individual red cells. The Meissner-Auerbach plexus and the sympathetic nerves were quite edematous. The jejunal mucosa was better preserved than in the duodenum. Villi could be seen which were still covered completely by epithelium (Plate II *E*). No mitoses were present in the crypts. The nuclei showed ballooning. A few examples of intraepithelial nuclear debris could be found, but the crypts filled with debris as found in heavily irradiated animals were not found here. Pyknotic leukocytes and lymphocytic remnants were present in the lamina propria, which was relatively cell-poor and shrunken. The epithelial cells of the extrusion zone of the tip of the villus were lower and broader than those along the side of the villus. True early squamous metaplasia was seen best in the necks of the crypts.

Lower Jejunum: Similar changes were seen.

Ileum: The omental fat showed no changes. The lymphoid tissue of the villi was markedly depleted. The villi were, in general, shortened and there was an apparent increase in mucous goblet cells. No mitoses were found. An occasional crypt contained cellular debris.

Pancreas—The pancreas showed normal-appearing islets and acini. An occasional necrotic cell in the pancreas proved, on close examination, to be leukocytic in origin.

Prostate—Some of the prostatic glands were hyperplastic and showed a squamoid metaplastic type of epithelium which, in some areas, was definitely transitional.

Pituitary—The pituitary blood vessels were quite hyperemic, and in the region of the stalk there were areas of extravasated red blood cells.

Brain and Spinal Cord—In general, the changes in the meninges were few. There was conspicuous engorgement of vessels in the meninges, brain substance, and choroid plexuses. In several sections substantial activation of arachnoidal histiocytes was observed. Here and there, some of the histiocytes had become macrophages and, on rare occasion, macrophages were found filled with broken-down blood pigment (Plate II *F*). A few neutrophilic leukocytes were found in the arachnoidal meshes. In the meninges no vasculitis was observed except for a few small and large mononuclear cells in the adventitia. Most of these were regarded as activated histiocytes rather than lymphocytes. No collagen damage was observed except possibly in occasional short stretches of the arachnoidal membrane, beneath which the arachnoidal trabeculae were condensed or swollen and displayed increased eosinophilia.

Conspicuous in the brain substance was focal evidence of an edematous process perivascularly, especially in the cerebral white matter, and occasionally in the gray matter. This was manifested by porosity of perivascular brain tissue. No coagulated edema fluid was found in the brain, apparently since only low-molecular fluid had escaped from the blood stream. The prominent red-tinged streaks noted grossly in the cerebral white matter proved microscopically to be due to the presence of low-molecular fluid and erythrocytes in the perivascular white matter. Tissue sponginess, denoting an edematous process, was noted here and there in lamina I of the cerebral cortex, and sometimes in lamina II.

In the brain substance no vasculitis was observed, but the adventitia of vessels, chiefly veins, contained mononuclear cells. Such cells were usually few in number. Vessels thus affected were found chiefly in the cerebral white matter where some of them contained plugs of leukocytes or plugs of leukocytes in clotted plasma. A fair number of leukocytes in several vessels exhibited karyorrhexis and other cytologic evidence of damage.

Occasionally in the cerebral cortex a necrotic nerve cell was found. In the white matter of the parietal lobes an extraordinary number of oligodendroglia were pyknotic. The cytoplasm of numerous astrocytes (or oligodendroglia) in the corpus callosum was rather copious, which was considered of a reactive nature. No alterations were observed in the white matter proper.

The choroid plexuses of all ventricles were studied. The only abnormality found consisted of one small group of mononuclear cells in the base of the choroid plexus of the fourth ven-

tricle. Here, occasional macrophages contained broken-down blood pigment. The ependyma was spared except for local loosening of the subependymal matrix in the floor of the fourth ventricle (in the region of the dorsal nucleus of the vagus nerve bilaterally), and except for a rather large, old glial scar in the wall of the lateral ventricle.

The cerebellar cortex was unaffected except for the tonsil; it was edematous, and a fair number of the granule cells were poorly stained. No pyknosis of granule cells was observed. This edematous process was thought to be related to circulatory disturbances consequent to pressure of the tonsil against the lower brain stem, secondary to brain swelling. Some of the cells of the dentate nucleus appeared unduly shrunken.

Hemorrhages were fairly common throughout the brain but were small and usually confined to the perivascular space. They were most common in the hypothalamus and in the medulla oblongata, especially near the ventricular system. Because of lack of cellular reaction perivascularly, they were regarded as an agonal phenomenon.

In summary, the microscopic evidence indicated circulatory disturbance in the brain, manifested by generalized cerebral swelling perivascular transudates and hemorrhages. There was no appreciable vasculitis, but reaction on the part of the histiocytes was very evident in the arachnoidal meshes and, in less measure, about vessels in the meninges and brain. Lymphocytes also contributed to the infiltrates, including the one found in the choroid plexus. The brain swelling and edema could be explained by the right cardiac failure and resulting cerebral passive congestion and anoxia.

Anatomical Diagnoses

The anatomical diagnoses which summarized the gross and histologic findings were as follows:

Acute myocarditis, right atrium and ventricle; severe intracellular myocardial edema.

Acute cardiac dilatation, right atrium and ventricle (clinical right heart failure).

Acute pericardial fibrinous effusion, inflammation and edema.

Thrombosis of the right auricular appendage.

Acute hepatic central congestion.

Cerebral edema; slight cerebellar pressure conus; diffuse radiovasculitis, agonal perivascular cerebral hemorrhages; parietal oligodendroglial pyknosis.

Acute radioatrophy of spleen, lymph nodes, thymus, and bone marrow; lymphorrhexis in lymph nodes and thymus.

Peripheral leukocytosis and lymphopenia.

Acute radiohemachromatosis of macrophages of bone marrow and lungs.

Petechial hemorrhage (and radiovasculitis) of epigastrium, pericardium, left lobe of liver, jejunal and antral peritoneum, antral mucosa, pericardium, right atrial, ventricular, and interventricular septal myocardium.

Acute, moderate gastric distention.

Radionecrosis of gastric parietal cells of anterior gastric wall.

Acute upper jejunal distention.

Acute, generalized mitotic radiosuppression seen in gastrointestinal tract, skin, esophagus, bone marrow, lymph nodes, and testes.

Acute jejunal and ileal radioenteritis.

Moderate distention of gallbladder.

Early benign prostate hyperplasia, median bar type.

Fibrocalcareous complex of Ghon.

Minimal pulmonary anthracosis and anthracosis of peribronchial and hilar nodes.

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SECTION V

Special Studies

Wright H. Langham, Ph.D.
With the collaboration of Donald F. Petersen, Ph.D.,
R. Gordon Gould, Ph.D., Harry Foreman, M.D., F. N. Hayes, Ph.D.,
Marvin Van Dilla, Ph.D., Morris F. Milligan, Ph.D., William D. Moss, B.S.,
Dean D. Meyer, B.S., and others

A NUMBER of special biochemical studies, whole-body counts, and gamma-ray spectral measurements were performed to collect additional information on effects of high doses of radiation and to provide data for dose calculations and for comparison with similar data obtained from past cases of serious exposure. In the case of K, the number of studies that could be carried out was limited by the condition of the patient, which affected the amount of antemortem biological material available for study. Prior to death, the patient voided only three times, with a total urinary output of only 590 ml. The biological samples available for study were as shown in Table XIII.

No concerted effort was made to carry out special biochemical studies on the other personnel involved in the accident, since the exposures were all relatively low. Some measurements were made, however, on Case D, who received an exposure of about 130 rads (most of which resulted from his entering the room soon after the accident had occurred).

Measurement of Induced Sodium-24 Activity

Blood—The first samples of blood were drawn from K at 6:10 p.m., and from D at 8:30 p.m. They were counted immediately for induced Na^{24} activity and for comparison with similar measurements made on victims of previous Los Alamos criticality accidents.[1] Five milliliters of blood was counted in a sodium iodide crystal well counter which, when calibrated against a Na^{24} standard, had an efficiency of 8.1%. Corrected to zero time, K's blood showed a Na^{24} activity of 5.47 mμc/ml. From his hematocrit of 58% and a serum sodium concentration of 3.33 mg/ml, his serum sodium specific activity was estimated as 3.9 mμc/mg. A similar measurement using a 9½ × 6 in. sodium iodide crystal spectrometer gave a Na^{24} blood concentration of 5.13 mμc/ml and a serum sodium specific activity of 3.66 mμc/mg (average 3.77 mμc/mg). The specific activity of D's serum sodium measured by the same

Table XIII—Biological Samples Available for Study (Case K)

Urine	
Voided 6:30 a.m. 12/31/58	150 ml
Voided 10:00 a.m. 12/31/58	180 ml
Voided 2:40 p.m. 12/31/58	260 ml
From bladder at autopsy, 6:30 a.m. 1/1/59	3 ml
Feces	
Formed stool 5:20 p.m. 12/30/58	36 gm
Diarrhea fluids 5:25 p.m. 12/30/58	64 gm
Second diarrhea 7:30 p.m. 12/30/58	Not available
Blood	
6:10 p.m. 12/30/58	30 ml
8:00 p.m. 12/30/58	20 ml
10:00 p.m. 12/30/58	20 ml
3:15 a.m. 12/31/58	20 ml
8:30 a.m. 12/31/58	20 ml
11:15 a.m. 12/31/58	20 ml
12:30 p.m. 12/31/58	20 ml
5:15 p.m. 12/31/58	20 ml
6:30 a.m. 1/1/59	Post mortem
Tissues (for analysis)	
Heart	371 gm
Lungs	854 gm
Stomach	203 gm
Jejunum	169 gm
Ileum	91 gm
Colon	160 gm
Skeletal muscle	199 gm
Liver	619 gm
Kidneys	252 gm
Spleen	108 gm
Thymus	25 gm
Pulmonary lymph nodes	5.2 gm
Adrenal	3.5 gm
Rib	19 gm
Sternal bone	122 gm
Vertebral bone	180 gm
Sternal cartilage	40 gm

Table XIV—Serum Sodium Specific Activities of Los Alamos Criticality Accident Victims (at t = 0)

Case*	Serum Na S. A. (mμc/mg)
1	0.49†
2	0.03
3	1.99†
4	0.36
6	0.19
7	0.10
8	0.05
9	0.04
10	0.03
D	0.01
K	3.77†‡

*Case numbers are those in the paper by Hempelmann et al.[1]

†Terminated fatally.

‡Average of crystal spectrometer and sodium iodide well counter determinations.

methods was = 0.01 mμc/mg, which is approximately the lower limit of detection. The radioactivity in the blood was characterized as Na^{24} both by its 2.76-mev gamma-ray energy and its half-life (= 14.5 hr).

A comparison of the serum sodium specific activities with those of previous criticality accident cases is shown in Table XIV. These data were the first laboratory indications that K had received a supralethal exposure. Assuming the same ratio of serum sodium activation to radiation dose as occurred in Cases 1 and 3, K's serum sodium value indicated an exposure that was approximately eight times the dose that had been fatal in 24 days to Case 1 and approximately two times the dose that had proved fatal in 9 days to Case 3. D's serum sodium value indicated relatively little exposure to the prompt radiation, indicating that the majority of his exposure resulted from his entering the room after the accident had occurred.

G. S. Hurst, of the Oak Ridge National Laboratory, was able to give a preliminary estimate of the dose received by K based on his experiences during and subsequent to the Y-12 accident.[33] His estimate of the fission neutron dose was based on his observations that a burro exposed to 48 rads of fission neutrons in a mock-up experiment showed 2.9×10^{-1} mμc Na^{24}/ml of blood. Assuming that the accidents were comparable and that the blood sodium concentrations in the burro and in man were the same, the neutron dose received by K was estimated as

$$\frac{5.47 \times 48}{0.29} = 900 \text{ rads}$$

Assuming a gamma-to-neutron ratio of 3 (observed in the mock-up of the Y-12 accident), the estimated gamma dose was 2,700 rads, giving a total exposure of 3,600 rads.

Table XV—Total-Body Sodium-24 Measurements (t = 0) on Personnel in Building at Time of Accident

Initial*	Occupation	Sodium-24 Activity (μc)
K	Chemical operator	293†‡
D	Chemical operator	1.1‡
		1.1
R	Chemical operator	0.60
L	Security inspector	0.15
M	Security inspector	0.14
Z	Operator supervisor	0.14
X	Chemical operator	0.05
U	Monitor	0.04
N	Chemical operator	<0.02
O	Chemical operator	<0.02
W	Group leader	<0.02
S	Monitor	<0.02
P	Security inspector	<0.02
Q	Security inspector	<0.02

*Designations of individuals are the same as those used in Section VII

†Post mortem, after correcting for Na^{24} loss in urine and feces prior to death

‡Measured by gamma spectrometer; all other measurements in whole-body liquid scintillator

Similarly, the neutron radiation dose received by D was estimated at 2.6 rads. In his case, use of a gamma-to-neutron dose ratio of 3 to obtain prompt gamma exposure was not justified. Serum sodium specific activities were not measured on any of the other personnel in the building at the time of the accident.

Whole-body Sodium-24 Measurements—All persons in the building at the time of the accident were counted for gross whole-body gamma activity in the whole-body liquid scintillation counter within three hours after the accident. D was also counted in the whole-body gamma-ray spectrometer, as was the body of K. No whole-body measurements were made on K prior to death because of his condition and the fact that his body Na^{24} activity immediately after the accident exceeded the capacity of the counters. The Na^{24} measurements (corrected to $t = 0$) on all personnel are shown in Table XV.

A gamma-ray spectrometer measurement on Case D indicated conclusively that the principal activity measured in the various personnel was Na^{24}. His spectra, taken at 4 and 70 hours after the accident, showed a 15% loss of Na^{24} during the interval, which is in good agreement with the value of 13% predicted from whole-blood Na^{22} studies on humans conducted by Richmond.[34]

The value reported for K was corrected for

loss of 1 μc of Na^{24} in urine, feces, and diarrhea fluid prior to death. Because of the high Na^{24} activity of the body, it was not possible to make the spectral measurements with the 9½ × 6 in. sodium iodide crystal spectrometer, and it was necessary to substitute a 3 × 3 in. crystal and make the measurements at a distance of 46½ in. from the sternum. The counting system and geometry were then calibrated by giving a known amount of Na^{24} to six subjects varying in weight from 125 to 214 lb and counting them in exactly the same position. From the calibration curve of weight versus counts and K's weight, it was possible to estimate his Na^{24} activity rather accurately. Corrected for excretion and for radioactive decay, his body Na^{24} activity at the time of the accident was estimated at 293 μc. The application of blood and body Na^{24} measurements to neutron dose calculations is shown in Section VI.

Sodium-24 Measurements on Autopsy Specimens (Case K).—Since sodium is largely extracellular, soft tissues having about the same amount of extracellular fluid would be expected to contain approximately the same amount of sodium. Exceptions are the lymph nodes and blood, which contain relatively larger amounts of extracellular fluid, and muscle, which contains a large amount of intracellular but a relatively small amount of extracellular fluid. Bone, however, contains more sodium than do the soft tissues; about 50% of this sodium is incorporated in the bone mineral and is unexchangeable. The measured Na^{24} activity in the autopsy specimens from K (corrected to time of the accident) are shown in Table XVI. All samples were measured with the large sodium iodide crystal gamma spectrometer, which was calibrated with a known Na^{24} standard.

Division of the total Na^{24} activity of the soft tissues by the gross soft tissue weight gives a Na^{24} activity of 2.93 mμc/gm. If the sodium space is taken as $\frac{Na^{24}/gm\ tissue}{Na^{24}/gm\ serum}$, K's sodium space is estimated as 24.7%, which agrees with the value of 25% reported by Hevesy.[35] Assuming the specific activity of the total-body sodium was the same as that of serum sodium (3.77 mμc/mg), K's total-body sodium content would be

$$\frac{2.93 \times 10^5\ m\mu c}{3.77\ m\mu c/mg \times 10^3\ mg/gm} = 78\ gm$$

which is considerably lower than the 105-gm value given for the standard man. That K's estimated total-body sodium was low cannot be explained on the basis of Na^{24} excretion, since excreta were measured and contained only 1 μc of activity. Part, but not all, of the discrepancy perhaps may be explained by the fact that the neutron dose to the chest and upper portion of the body was higher than to the lower extremities, and compact bone such as the femur is known to have a higher sodium content than highly trabeculated bones such as rib and vertebra.[36] Because of the inequality of dose distribution, the specific activity of nonexchangeable bone sodium in the compact bone of the extremities may have been lower than that of the serum. Again assuming equilibration between serum and tissues sodium specific activity (a reasonable assumption for soft tissues), the total sodium content of the various tissues can be estimated from Table XVI. The estimated values for most tissues are considerably lower than reported in the literature.[37, 38]

The estimated Na^{24} content of the skeleton (minus marrow) was 19% of that of the total body, which is considerably lower than the 30% reported by Edelman.[39] Again, the cause of the discrepancy may, at least in part, lie in the inequality of neutron dose distribution.

Table XVI—Sodium-24 Concentration in Autopsy Specimens (Case K)

Tissue or Organ	Sodium-24 Activity* (mμc/gm)	Tissue Sodium Concentration		Total Sodium-24‡ (μc)
		Estimated (mg/100 gm)	Reported (mg/100 gm†)	
Muscle	1.54	41	65-105	51.65
Skeleton	7.14	190	300	79.08
Blood (whole)	5.31	141	175	31.88
Serum	12.27§	316	300-330	....
Large intestine	3.57	95	160	1.12
Small intestine	5.75	153	160	7.18
Stomach	3.31	88	130	1.03
Liver	2.48	66	120-150	4.84
Lungs	3.62	96	190-240	3.95
Lymphoid tissue	7.94	211	....	6.19
Kidneys	4.36	116	165-220	1.18
Heart	3.57	95	80-160	1.43
Spleen	3.15	84	120	0.37
Adrenals	4.51	120	....	0.06
Thymus	3.28	87	....	0.03
Balance	4.15**	110	....	86.65**
Total-body calculated	3.55	95	....	277
Total-body measured	3.76	110	....	293

*Corrected to time of accident

†References 37, 38

‡Where organ and tissue weights were not available, calculations were made on basis of the standard man[37]

§Corrected for specific gravity of 1.023

**Sodium-24 in balance taken as average of soft tissue analyses.

Miscellaneous Gamma-Ray Spectrometer Measurements

Gamma-ray spectral measurements were made on all excreta, which included urine, feces, and diarrhea fluid passed at the times and in the amounts shown in Table XIII. No gamma activity other than Na^{24} was found in the urine, and the total urinary activity amounted to 0.3 μc. The formed stool passed at 5:20 p.m., 45 minutes after the exposure, contained 0.1 μc Na^{24}. The diarrhea fluid passed 5 minutes later contained 0.5 μc Na^{24} and identifiable amounts of 38-minute Cl^{38}. A third gamma activity was detected also, which corresponded to 2.6-hour Mn^{56}. Manganese-55 has a 13-barn thermal neutron cross section, and the daily intake is normally about 5 mg. The activity found in the diarrhea fluid was believed to have resulted from activation of Mn^{55} in the lumen of the bowel at the time of the accident.

One amalgam filling obtained at autopsy was found to emit gamma rays with energies corresponding to 46-day Hg^{203} and 270-day Ag^{110}. The filling weighed 0.5395 gm and contained 0.1958 gm (36.3%) of silver and 0.2917 gm (54.07%) of mercury.

Phosphorus Analyses of Blood and Urine

Determinations of P^{32} specific activity were carried out on two blood samples and all three urine specimens from K and on five urine specimens from D.

Starting 72 hours after the accident, the beta activity of whole urine and blood plated directly and measured with an end-window Geiger counter was identical with the P^{32} activity as shown by the decay curve, with a half-time of 14.5 days.

Phosphorus was also isolated from a number of samples as reduced phosphomolybdic acid in isobutanol solution by the Berenblum and Chain procedure.[40] A number of the values obtained by evaporating the isobutanol solution to dryness checked well with those from whole dried urine or plasma but in some cases were much lower. Urine samples to which P^{32} was added also gave low results. A possible explanation is that the P^{32} may form volatile phosphate esters during the drying procedure; experiments to test this hypothesis are planned. The data collected by the isobutanol extraction procedure were not used except where results by other methods were lacking.

Analyses for total phosphorus were done by both the Berenblum and Chain and the standard Fiske and Subbarrow methods.[41] Specific activity values, calculated from the counting rates and phosphorus content of serum, red cells, and urine, are given in Table XVII. The specific activity of urinary phosphorus was much greater than that of serum. This observation was first noted by Perley, Langham, and Hoffman[42] in experimental animals and was documented for other criticality accident victims in the report of Hempelmann, Lisco, and Hoffman.[1]

Two possible explanations may be considered: (*A*) About 90% of the phosphorus in the body is in the bones, and because of nonuniformity of neutron dose, etc., it may not have the same average specific activity as the serum and soft tissue phosphorus. Consequently, immediately after exposure, the higher specific activity phosphorus in body fluids and soft tissues may be excreted in the urine before equilibration with less active bone phosphorus could take place. (*B*) The Szilard-Chalmers effect may play a role also, as suggested by Hempelmann et al. If half the P^{32} atoms formed from P^{31} are converted into P^{32+3} ions by recoil, they would presumably form phosphite salts, which may be excreted preferentially by the kidney before being metabolized to phosphates.

The urine samples from D had so little activity that the results from counting small samples were not statistically reliable. Large samples (100 to 200 ml) of all urine specimens except the first were treated with NH_4OH and the alkaline precipitate (free from K^{40}) was

Table XVII—Blood and Urine Inorganic Phosphorus and Phosphorus-32 Determinations (Cases K and D)

Time After Exposure (hr)	Urine Phosphorus		Serum Phosphorus	
	Total (mg %)*	S. A. (mμc/mg P)	Total (mg %)*	S. A. (mμc/mg P)
Case K				
3½	....	....	14†	0.83†
35 (post mortem)	....	....	23	0.16
14	5	152	....	....
17	3	121	....	....
22	42	4.3	....	....
Case D				
4	111	0.03	....	....
16	147	neg.‡	....	....
64	71	neg.‡	....	....

*Normal inorganic phosphorus in urine is 50 to 100 mg %; in serum 3.5 to 5 mg %.

†Phosphorus in red cells run on this sample, total phosphorus 70 mg % (normal 70 mg %), specific activity of cell phosphorus was 0.11 mμc/mg.

‡100 ml of urine showed a count only slightly above background.

Table XVIII—Urinary Phosphorus Specific Activities in Criticality Accident Victims

Case	Sampling Time (hrs)	Specific Activity (mμc/mg P)
1*	17.4	2.97
2	17.4	0.13
3	5.3	9.65†
4	3.1	1.84
6	2.2	0.37
7	1.2	0.51
8	2.2	0.07
9	1.2	0.04
10	2.2	0.04
D	4	0.03
K	14	152†

*Case numbers refer to Hempelmann.[1]

†Serum phosphorus specific activity was measurable only in Case 3 (0.35 mμc/mg) and Case K (0.82 mμc/mg), both of which terminated fatally.

counted. The results indicated negligible activity.

A comparison of the urine phosphorus specific activity of K and D with that of other criticality accident victims at Los Alamos is shown in Table XVIII. These data merely emphasize the apparent lack of any significance of urinary and serum phosphorus specific activity measurements with regard to evaluation of criticality accidents. It is interesting but puzzling that the P^{32} concentration (P^{32}/ml) in K's urine appeared to decrease exponentially, with a half-time of ~4 hours.

Neutron-Induced Phosphorus-32 in Sulfur-Rich Tissues (Case K)

Tissues with a high sulfur-to-phosphorus ratio afford the possibility of estimating the fraction of neutron dose resulting from neutrons with energies in excess of 2.5 mev by determining the P^{32} produced by the $S^{32}(n,p)P^{32}$ reaction. Samples of hair, xiphoid cartilage, and testis were wet-ashed with nitric acid, and aliquots of the digests were used for total phosphorus,[40, 41] sulfur,[43] and P^{32} determinations. The method of Berenblum and Chain[40] was found to be particularly useful for separation of P^{32} and Na^{24} activities. Results of these measurements are summarized in Table XIX. Application of the data to the estimation of incident fast neutron dose and dose distribution is shown in Section VI. Fingernails and toenails could be used also to estimate incident fast neutron dose to the extremities, but samples of these tissues unfortunately were not obtained.

Plutonium Analyses of Tissues (Case K)

The victim of this fatal accident had spent a total of approximately 6 years of an 11½ year employment period working as a plutonium process operator. Because of his long potential exposure to this material, tissue samples were obtained at autopsy and analyzed for Pu^{239}. Since death resulted from an overdose of radiation involving no plutonium contamination, the conditions of the accident did not influence the plutonium content found in his tissues and organs.

Table XIX—Fast Neutron-Induced Phosphorus-32 Activity in Sulfur-Rich Biological Materials (Case K)

Material	mg P/g	mg S/g	P/S Ratio	Phosphorus-32 d/m/mg S (t = 0)	Calc. Dose (rads)
Hair	0.063	45.50	0.001	24.0	2600
Xiphoid cartilage	0.260	9.26	0.028	31.0	3000
Testis	1.900	17.90	0.160	Background	....

Table XX—Employment History of Case K

Period	Occupation	Time (days)
6/17/46 to 1/24/49	Pu^{239} recovery operator	947*
1/24/49 to 6/4/54	U^{235} recovery operator	1,987
6/4/54 to 6/8/55	Resigned—no exposure	369
6/8/55 to 12/31/58	Pu^{239} recovery operator	1,297*
Total plutonium exposure time		2,244

*Between first and second plutonium exposure periods, exposure conditions had been greatly improved.

These data are of interest because they provide for the first time limited information on *(a)* the efficacy of industrial hygiene and engineering practices in plutonium-processing operations; *(b)* body distribution of plutonium in an individual exposed primarily by inhalation to low-level plutonium aerosols over a prolonged period; and *(c)* reliability of estimates of body burden from urinary excretion data when exposure has been primarily via inhalation.

Plutonium Exposure—K's employment and job assignment history is shown in Table XX. During the first period of plutonium exposure (6/17/46 to 1/24/49), his work consisted of chemical processing involving plutonium nitrate solutions, plutonium oxalate, and occasionally plutonium fluorides. During the second period (6/8/55 to 12/31/58), his work consisted largely of liquid-liquid extraction of plutonium under greatly improved exposure conditions.

Detailed exposure records were kept during the periods when the employee was working with plutonium. These records included plutonium air concentrations in the processing rooms; the operator's nose swipe counts done several times a week up to the end of 1955, and frequently but irregularly thereafter; daily hand counts; and frequent plutonium assays of 24-hour urine specimens. Average plutonium

Table XXI—Average Concentration of Air-Borne Plutonium in Areas in Which K Was Assigned

Year	Room 308 (d/m/M³)	Room 313 (d/m/M³)	Room 308 (d/m/M³)	Room 413 (d/m/M³)	Rooms 212, 213, 218 (d/m/M³)
1946	6	16	188	15	
1947	11	25	77	98	
1948	Not used	24	35	69	
1949	Not used	19	30	72	
1949-1954	Worked with enriched uranium				
1955					3
1956					3
1957					4
1958					4

*Table XXII—Record of High Nose Swipe Counts**

Date		Right/Left Nostril (c/m)
1946	7/29	189/320
	7/30	87/70
	9/5	149/19
	9/25	55/4
	11/1	61/15
	12/10	57/68
	12/30	164/106
1947	1/21	102/61
	4/1	91/135
	7/7	144/40
	10/3	120/78
1948	2/10	0/59
	4/26	86/3
	6/9	33/50
	6/10	244/72
	7/2	72/1
	8/2	65/0
	12/1	50/38
After 1948		None

*Technique consisted of rotating a piece of dampened filter paper on an applicator in each nostril, after which the paper was unrolled, flattened, dried and counted. Fifty counts per minute was arbitrarily taken as the limit of significance.

air concentrations to which the individual was exposed are presented in Table XXI. The instances when his nose swipe counts went above 50 c/m (arbitrarily chosen as the limit of significance) are listed in Table XXII, and his urine assay record is given in Table XXIII. No tabulation of hand counts is given, since they were without exception below significant levels. Results of fecal analyses were not available. Because of the analytical and sampling difficulties involved, fecal analyses are not a routine practice at the Los Alamos Scientific Laboratory.

There were no specific accidents to which the individual's exposure could be attributed. However, as might be expected, a number of minor mishaps occurred during the period of employment. The detailed record of such minor incidents is summarized in Table XXIV.

Plutonium in Tissues and Organs—At the time of autopsy, tissues were taken specifically for plutonium assay. The specimens taken and their plutonium content are shown in Table XXV. The assays were carried out using the alpha-track counting method.[44] Two independent analyses of aliquots from each tissue sample, ribs, sternum, vertebrae, lungs, liver and lymph nodes, were made by the analytical section of the Los Alamos Industrial Hygiene Group and also at the Hanford Atomic Products Operation,* where they were analyzed independently by both the Biological Laboratory and the Bioassay Group. The values given in Table XXV are averages and standard deviations for the

*We wish to express our appreciation to our colleagues of the Biology Laboratory and Bioassay Group of the Hanford Atomic Products Operation for cooperation in supplying independent analyses of the tissue samples.

Table XXIII—Results of Periodic Plutonium Urine Assays for Entire Period of K's Employment

Date	d/m/24-hr Specimen	Date	d/m/24-hr Specimen
1946 8/9	1.2	1955 8/1	1.2
9/19	4.7	8/12	0.6
12/18	1.7	8/19	0.0
1947 4/18	0.7	9/30	0.7
5/23	0.7	11/14	0.7
6/26	0.7	12/27	0.6
7/30	0.0	1956 2/9	0.0
8/27	1.0	4/5	0.7
10/2	1.5	4/30	0.0
11/7	0.8	6/8	0.4
12/8	4.0	7/20	0.6
1948 1/13	1.0	8/23	0.5
2/13	0.0	9/25	0.0
3/19	0.0	10/24	0.0
4/22	2.0	11/23	0.0
6/23	3.7	12/17	0.1
7/22	0.0	1957 1/31	0.23
8/19	0.8	2/28	0.68
9/20	3.0	4/12	0.22
10/26	2.0	5/14	0.12
11/22	2.5	6/14	0.11
12/21	0.0	7/15	0.03
1949 1/24	Uranium	8/19	0.00
1/31	2.0	9/20	0.20
7/14	1.2	10/22	0.21
1950 2/8	0.8	10/31	0.39
9/1	0.1	11/14	0.51
1951 2/28	0.8	1958 1/10	0.00
9/4	0.7	2/21	0.65
1952 5/19	0.3	3/25	0.51
12/14	0.0	5/7	0.25
1953 9/4	0.0	6/19	0.55
1958 9/4	Off uranium	7/30	0.49
6/18	0.0	9/15	0.79
1955 6/8	Back on plutonium	11/28	0.47

Table XXIV—Summary of Minor Exposure Incidents of Case K during Period of Employment

Date	Type of Incident	Potential Exposure
8/26/46	Minor laceration on thumb	No alpha activity detected
12/30/46	Helped clean up spill of plutonium solution	Air concentration 52 d/m/M³ for 8-hr day; nose count 164/106 d/m
4/22/47	Received nitric acid burns on arm	No record of contamination
9/3/47	Small cut on hand, received while making material transfer	No alpha activity detected
12/14/47	Puncture wound on right hand from a screw point on an instrument panel	No contamination reported
3/17/49	Helped clean up spill of enriched uranium	No air concentration or nose swipe data recorded
4/22/50	Exposed to fumes of oxides of nitrogen	No contamination involved
8/10/53	Helped clean up spill of enriched uranium	Room air concentration 8 d/m/M³; nose count 4 d/m
8/9/55	While adding caustic to a plutonium and americium solution, materials sprayed out of reaction vessel; operator was wearing face shield and respirator	No body contamination detected; nose count 22/28 d/m; room air concentration 75 d/m/M³
11/25/58	Maintenance work involving removal of plutonium-contaminated insulation from an evaporator-condenser	Nose swipe count 0 d/m; room air concentration 1070 d/m/M³; operator was wearing Wilson 800 respirator

four independent analyses. The standard deviation for the lymph nodes includes variation in sample, as well as variation in analysis, since different lymph node samples were used in the independent determinations. The highest plutonium concentration was found in the pulmonary lymph nodes (125 ± 57 d/m/gm), followed by the liver (9.9 ± 1.4 d/m/gm), lungs (4.8 ± 0.6 d/m/gm), and then vertebrae (2.1 ± 0.6 d/m/gm). The average of all bone samples was 1.4 ± 0.7 d/m/gm, for which the standard deviation includes the variation among the different bone samples plus the analytical variation.

The total organ and body content, calculated from the data in Table XXV, are shown in Table XXVI. The estimated total body burden was 0.018 μc.

As a result of changes in production methods, the Pu^{238}/Pu^{239} ratio of plutonium processed at the Laboratory increased considerably between 1946 to 1948 and 1955 to 1958. Determination of the Pu^{238}/Pu^{239} ratios in bone, lymph node, liver, and lung samples indicated bone and lymph nodes had the lowest ratio (corresponding generally to material produced in 1946 to 1948), and lung samples had the highest (corresponding roughly to more recently produced material). The Pu^{238}/Pu^{239} ratio in the liver was intermediate.

Discussion—The data in Tables XXI and XXII show that all high plutonium air concentrations to which the employee was exposed and all high nose swipe counts recorded

Table XXV—Plutonium Concentration in Autopsy Samples (Based on Actual Analytical Data)

Organ or Tissue	Gross Weight at Autopsy (gm)	Plutonium Concentration (d/m/gm wet wt.)
Psoas muscle	....	0.01
Kidneys	270	0.05
Heart	400	0.06
Cartilage	....	0.14
Spleen	116	0.18
Sternum	....	1.0 ± 0.2
Ribs	....	1.2 ± 0.3
Vertebrae	....	2.1 ± 0.6
Lungs (minus major bronchii)	850	4.8 ± 0.6
Liver	1,950	9.9 ± 1.4
Bronchial lymph nodes		125 ± 57*

*Includes variation in different lymph node samples, as well as analytical variation.

occurred during his earlier period of exposure. It is very likely that most of his plutonium burden was accumulated during this period. The record summarized in Table XXIV shows that there were no specific incidents to which his plutonium exposure could be attributed. The summary is given principally to point out that close attention was given to all potential modes of exposure and thereby to emphasize the certainty with which a contaminating accident can be ruled out as the source of the subject's plutonium burden. It is most likely that the body burden, in this case, resulted from chronic inhalation exposure over a long period to a low-level plutonium-contaminated atmosphere. The

above speculations regarding time and mode of exposure are supported also by the indication that the Pu^{238}/Pu^{239} ratio in lymph nodes and bone appeared to correspond to that of plutonium being processed during the early period of the subject's exposure.

Three different urine assay procedures were used during the 12-year period over which the urine data shown in Table XXIII were collected. Each change resulted in somewhat greater reliability of the data. In 1957, the Hanford alpha-track counting procedure was adopted.[44] Urine assays from this time on have considerably higher reliability than previously. Even during 1957 and 1958, however, there was considerable variation in the assays, which is probably due both to analytical limitations and to normal physiological fluctuations. The employee's systemic plutonium burden was estimated from the urine assays using empirical equations derived from human excretion data.[45] Following an acute exposure occurring at known time, the retained plutonium body burden (D_R) is given by the expression

$$D_R = 435\ Ut^{0.76} \qquad (1)$$

in which U is the plutonium (c/m, d/m, μc) in a 24-hour urine sample collected days after the time of exposure. D_R is given in the same units used to express U. Since this equation is applicable to relatively acute exposure occurring at known time, it is necessary in protracted exposure cases to assume an effective time of exposure which, to a first approximation, may be taken as the midpoint of the work period. Following chronic invariant exposure to plutonium, the total systemic intake (D_t) is given by the expression

$$D_t = \frac{130 \times m \times U}{(n + \frac{1}{2})^{0.26} - (n - m + \frac{1}{2})^{0.26}} \qquad (2)$$

in which m is the duration of exposure (days), and U is the average d/m/24-hour urine sample taken n days from the beginning of exposure. D_t is the systemic exposure and it is necessary to subtract the amount excreted in order to obtain the amount retained (D_R). However, since only about 10% of a plutonium burden is excreted in 10 years,[45] such a correction is insignificant in most cases. The above expressions indicate the determination of body burden from a single 24-hour urine assay. Analytical limitations of the assay methods and normal physiological variations in urinary plutonium excretion make such estimates completely unreliable, and in practice it is better to use the average of several consecutive assays (even though they may be weeks or months apart).

Because of the nature of the exposure, neither of the above equations is specifically applicable to the case under consideration. Application of Equations 1 and 2 to the average of all urine assays run during 1949 to 1951 (during which time there was no exposure to plutonium) gives 0.033 and 0.031 μc, respectively, for the employee's body burden at that time as a result of his first period of exposure. During 1957 and 1958, frequent urine assays were run using the more sensitive and reliable alpha-track counting method. Estimations based on Equations 1 and 2 (assuming all his exposure occurred during the earlier work period, and using the average of the 1957 and 1958 urine assays) gave 0.034 and 0.031 μc, respectively, for the body burden at the time of death. The latter estimates may be high, since they are predicated on the assumption that the entire body burden was accumulated during the earlier work period and that exposure during the second period made no contribution to the 1957 and 1958 average urine assay value. That this assumption is approximately correct is borne out by the estimates of burden at the end of the employee's first period of exposure. A method of estimating plutonium body burden employing IBM 704 Fortran programming of all urine assay data has been developed by Lawrence.[46] His estimate of the subject's body burden at time of death was 0.019 μc. The burden derived from tissue analyses (Tables XXV and XXVI) was 0.018 μc, which is 45% of the maximum permissible level of 0.04 μc.[47]

The above agreement between body burden from tissue analyses and estimated burden from urine assays is so very satisfactory that it is undoubtedly fortuitous. Determination of body burden from tissue analyses is subject to considerable sampling uncertainty. Since it is not possible to analyze the entire body, it is neces-

Table XXVI—Total Body Burden Estimated from Tissue or Organ Weight and Plutonium Content

Organ or Tissue	Weight (gm)	Total Content (d/m)
Liver	1,950	1.930×10^4
Skeleton (average)	10,000	1.400×10^4
Lungs (minus bronchii)	850	0.408×10^4
Pulmonary lymph nodes	10	0.125×10^4
Muscle	30,000	0.030×10^4
Heart	400	0.002×10^4
Spleen	116	0.002×10^4
Kidneys	270	0.001×10^4
Remaining tissues*	26,400	0.026×10^4
Total	70,000	3.924×10^4 (0.018 μc)

*Assuming plutonium content of the remaining tissues is the same as that of muscle.[45]

sary to analyze aliquots of the various organs and, on the assumption of uniform distribution and representative sampling, calculate the total body content from the organ weights. This assumption is particularly uncertain with regard to the skeleton, as shown by the analytical data for sternum, ribs, and vertebrae (Table XXV).

Some uncertainty in the body burden estimated from urine assays may be expected also from the uncertainty in the exponents of the time parameters in Equations 1 and 2. When t = 10 days, a 10% error in the exponent of t (Equation 1) would make an 18% error in the body burden estimated from the urine assay. The error would increase with time, approaching a factor of about 2 at t = 3,500 days. Indication that the exponent of t in the basic urinary excretion equation[45] may be in error (by about 10%) in the direction resulting in overestimation of the body burden was obtained recently by reestimation of the plutonium burdens of workers exposed at the Laboratory during 1944 and 1945 who had received no subsequent exposure. When estimated from a urine sample collected approximately 10 years after termination, their systemic burdens were, in most cases, about a factor of 2 higher than the estimates based on urine samples collected during or immediately after exposure. Most of the cases, however, were believed to be inhalation exposures, and it is possible, although seemingly unlikely, that their systemic burdens did increase as a result of absorption of plutonium deposited in the lungs and lymph nodes.

The distribution of plutonium in the various tissues and organs was somewhat surprising. Approximately 50% of the body burden was found in the liver and 36% in the skeleton (Table XXVI). One hundred fifty days after intravenous injection of Pu^{+4}-citrate in man, approximately 65% of the administered dose was found in the skeleton and 22% in the liver.[48] Rat experiments show that the relative concentration of plutonium in bone and liver following intravenous injection is dependent on the chemical form and valence state of the material administered.[49] It is quite possible, in the present case, that the partitioning of plutonium between the liver and skeleton was influenced both by the chemical or physical nature of the plutonium and by the route of exposure. The respiratory route of exposure was undoubtedly responsible for the high plutonium concentrations found in the lungs and pulmonary lymph nodes.

Some interesting qualitative speculations regarding relative deposition and clearance rates of plutonium in various tissues may be drawn from the Pu^{238}/Pu^{239} ratios in bone, lymph nodes, liver, and the lungs. The Pu^{238}/Pu^{239} ratios of the plutonium deposited in bone and lymph nodes appeared to correspond with that of plutonium produced in 1946 to 1948 (which was the subject's earlier and greater period of exposure). The Pu^{238}/Pu^{239} ratio of the material deposited in the lungs, however, appeared to correspond to that of more recent production. The above observations suggest (*a*) a relatively rapid clearance rate for plutonium in the lungs compared to that in bone and lymph nodes, and (*b*) that a relatively small percentage of the material deposited in the lungs must migrate to these tissues. Since the Pu^{238}/Pu^{239} ratio of material in the liver was intermediate between that deposited in the bone and lymph nodes and that in the lungs, the rate of clearance from the lungs to the liver must be relatively fast and the retention time in the liver must be longer than in the lungs.

The biologically effective dose rate to the various tissues of the subject may be estimated from the observed plutonium concentrations (Table XXV) and the following expression derived from the Recommendations of the International Commission on Radiological Protection[50]:

$$R = \frac{C \times \Sigma E(RBE)n}{2.8 \times 10^{-3}}$$

In this expression, R is the dose rate in rem per week, C is the plutonium concentration in μc per gm, and $\Sigma E(RBE)n$ is an effective energy term weighted for the RBE of alpha particles and the nonhomogeneity of energy distribution and absorption in the tissue. The weighted energy terms for plutonium in soft tissue and bone are 53 and 270, respectively. Calculated in the above manner, the pulmonary lymph nodes were subject to the highest biologically effective dose rate ($\backsim$1 rem per week), the liver next (0.08 rem per week), followed by the bone and lungs (0.06 and 0.04 rem per week, respectively). The estimated alpha radiation dose to the lymph nodes may be considerably too high because of their shrinkage in mass as a result of the acute radiation dose ($\backsim$10,000 rads of fast neutrons plus gamma rays incident to the chest) received during the criticality accident.

No definitive conclusions can be drawn from a single case, but these observations again bring up the troublesome questions of choice of the critical organ following chronic inhalation exposure, the size of the critical tissue volume, and the relative sensitivity of various tissues to chronic radiation damage.

SECTION VI

Dosimetric Calculations

Payne S. Harris, M.D.

THERE were a number of complications connected with evaluation of doses to personnel involved in the accident. These ranged from the geometric and moderator characteristics of the source to the movement of personnel in the vicinity of the source after the accident. As has been usual in these situations, the standard dosimeter coverage was inadequate and, in some cases, absent.

The personnel involved can be considered in two groups. One group consists of those intimately associated with the accident and includes only three individuals, K, D, and R. This portion of the dosimetry report is concerned only with the exposures of these three persons. They received the highest and most significant doses. The second group consists of all other individuals who were in the area at the time of the accident and those who entered it afterward. The estimates of doses received by the personnel in the second group are given in Section VII.

Physical Characteristics of the Source

The critical reaction occurred in a 225-gal stainless steel tank containing approximately 130 gal of material. A schematic diagram of the tank is shown in Fig. 8. The critical reaction occurred in the upper layer of solution (labeled solvent). There was 3.27 kg of plutonium dissolved in this layer of organic solvent, which consisted of a tributyl phosphate-kerosene mixture. The aqueous phase below contained only 40 gm of plutonium, while the total solids ("heel," etc.) contained only 60 gm. Apparently, when the stirrer was started, the material in the organic phase was brought into a critical configuration in the horizontal center of the tank slightly below the center of the organic layer. The geometric level of the critical reaction was determined by linear measurement of the long-lived induced gamma activity in a stainless steel baffle located inside the tank.

There was apparently enough heating by the reaction and dispersal by continued stirrer operation to prevent any oscillation of the system such as occurred in the Oak Ridge accident.[51] Thus the whole event consisted of a single supercritical reaction. The total fissions that occurred were determined subsequently by standard radiochemical analysis and found to be $\backsim 1.5 \times 10^{17}$. Under these conditions, the pulse width was probably less than 200 μsec. This undoubtedly caused a rapid temperature increase in the solution, which in this time period would cause quite a thermal shock. The tank, in fact, was found to have moved $\frac{3}{8}$ in. on one of its supports.

The general position of a chemical operator in performing the standard operation is indicated by the posed photograph shown in Fig. 1 and in Fig. 8. A number of operators were questioned and demonstrated this standard technique. The schematic outline shown in Fig. 8 gives the most likely dimensions, distances, and conditions prevailing at the time of the accident. In no case during demonstration of the technique was the frontal plane of an operator's body found to be closer than 6 in to the tank. Thus thermal shock could not be transmitted directly to the chest, abdomen, etc. From the dimensions shown in Fig. 8, it can be seen that the area of the xiphoid was actually closer to the essentially unmoderated critical reaction center than any other portion of the body. Also, the geometry was such that inverse square could materially change the dose by a factor of 2 over the average body thickness of 30 cm without any consideration of absorption. Finally, it can be seen that the moderator mass varied from essentially zero to greater than 70 cm of H_2O over the exposure profile. These considerations certainly made the dose distribution of both neutrons and gamma rays completely anisotropic as far as the operator K was concerned. It is, therefore, possible to consider only average dose to the body and incident dose to specific areas.

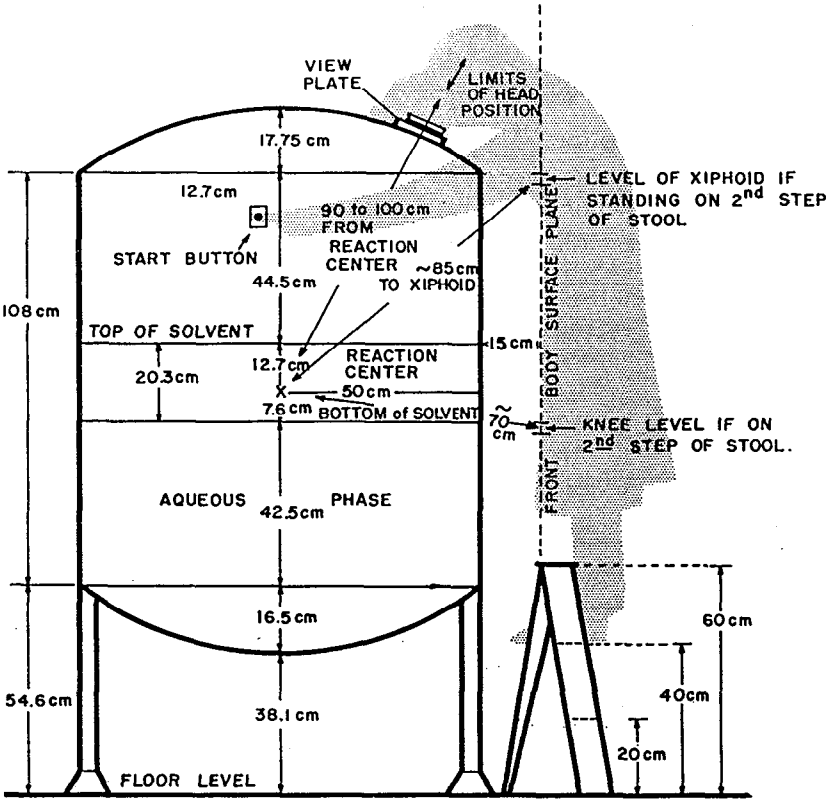

Fig. 8—Schematic diagram of solvent-treatment tank set-up.

Neutron Dose

The neutron dose to the operator K is of primary interest. This portion of his total exposure was due almost exclusively to fast rather than thermal neutrons. The incident neutron dose may be expected to vary from point to point because of position with respect to the source and the varying thickness of moderator. The depth dose may be expected to vary also with respect to both geometry and absorption.

Several measurements were available from which the fast neutron component of K's dose could be estimated. These included measurements of blood and total body Na^{24} activity (see Section V), serum sodium specific activity, induced activity in materials in his pockets or clothing, and specific induced activities in selected tissues. The applicability of all such measurements to neutron dose estimation is predicated on the assumption that thermal neutrons did not cause an appreciable amount of the total activity measured. This assumption seems justified on the basis that the thermal neutron fluxes were low or that the activation cross sections were small, or a combination of the two.

If one accepts the above assumption, two uncertainties remain in the estimation of fast neutron dose from Na^{24} measurements. One is the estimation of the total amount of Na^{23} (fixed plus exchangeable) in the body. Surprisingly, this is not well known, although 105 gm is the value given for the standard 70-kg man.[50] Total-body activity measurements apply to total-body sodium, whereas specific activity measurements of blood or serum sodium apply primarily to the exchangeable fraction. The second uncertainty is the variation in induced Na^{24} activity per unit of neutron dose as a function of source spectrum change. It has been postulated[51] that the activity per unit dose is a slowly changing function of spectrum. This assumption implies that the mass and moderating ability of the irradiated object are enough to wipe out any significant spectral effect.

An experiment was performed using a Los Alamos prompt critical assembly (Godiva II) as a fission neutron source to check the second uncertainty. Varying thicknesses of polyethylene moderator were interposed between the source and a point of measurement, and the neutron dose was measured. Afterward, an 18-liter polyethylene jug filled with a 1.29% $NaHCO_3$ solution in water was positioned at the same point and the induced Na^{24} activity measured as a function of moderator thickness. The results of the measurements are shown in Table XXVII and indicate that the induced activity per unit of neutron dose is not a strong function of spectral degradation under these conditions of exposure. Induced Na^{24} measurements can be used, therefore, to estimate the average fast neutron dose to the exposed subjects.

Estimation of Average Fast Neutron Dose—The total-body sodium activities can be used to estimate the average fast neutron dose by comparison with the burro experiments carried out in connection with the Oak Ridge National Laboratory's Y-12 criticality accident.[51] In these experiments, a burro was exposed to 48 rads of fission neutrons and induced Na^{24} activity measurements carried out. Assuming the sodium concentration in the human is the same as that in the burro (comparison of blood concentrations indicate that this assumption is reasonable), the average total-body fast neutron dose may be estimated from total body

Na^{24} measurements using the following expression

$$Rad_S\ (ave) = \frac{Act_S}{Act_B} \times Rad_B$$

where Act_S = activity in the subject in $\mu c/kg$; Act_B = activity in the burro in $\mu c/kg$; Rad_B = dose to the burro; and Rad_S is the dose to the subject. The Na^{24} activity in the burro was found to be 0.236 $\mu c/kg$, following a total neutron dose of 48 rads. In a similar manner, the average fast neutron dose to the exposed subjects can be estimated from blood Na^{24} activity and specific activity of the serum sodium. The burro exposed to 48 rads had a blood Na^{24} activity of 0.29 $m\mu c/ml$ and a serum sodium specific activity of 0.187 $m\mu c/mg$ (assuming a 50% hematocrit).

Average fast neutron doses, estimated from the various Na^{24} measurements, are shown in Table XXVIII. The values for K's average body dose range from 835 to 968 rads.

The average fast neutron dose to K can be estimated also from the Godiva II experimental data (Table XXVII) using the Na^{24} values obtained for the 18-liter water phantom containing 1.29% $NaHCO_3$. With 6 to 9 in of moderator between the fission neutron source and the phantom, the induced Na^{24} activity was approximately 100 c/m/rad. Under this set of circumstances, the initial counting rate of the phantom was 5.575×10^3 c/m, with a dose of 57.1 rads. Calibration of the counting equipment indicated a sensitivity of 1.923×10^{-3} $\mu c/c/m$. Thus, the total activity induced in the phantom was 10.7 μc. The sodium concentration in the phantom was 3.53 gm/liter, while the value given for man[50] is 1.5 gm/kg, or 1.5 gm/liter based on a density of unity. K's total body Na^{24} activity was 293 $\mu c/kg$. The induced Na^{24} activity of the phantom was 10.7 $\mu c/18$ kg = 0.595 $\mu c/kg$. From these values, K's average neutron dose is estimated as

$$\frac{4.10}{0.595} \times \frac{3.53}{1.5} \times 57.1 = 926 \text{ rads}$$

which agrees satisfactorily with the doses estimated from total body and blood Na^{24} measurements and the serum sodium specific activity. Since there is no unequivocal reason for assuming that any one of the above estimates is more accurate than the others. K's average total body fast neutron dose is assumed to be the average of all the estimates, or ~900 rads. His tissue dose, however, definitely was not uniform and varied both with depth and with respect to moderator characteristics and geometry of the source.

Estimation of Incident Fast Neutron Dose—Estimates of fast neutron dose incident on different regions of K's body were made using other measurements. Samples of hair (which has a high sulfur content) were obtained, and the P^{32} activity produced by the $S^{32}(n,p)P^{32}$ reaction was measured. This is a threshold reaction for neutrons with energies greater than 2.5 to 3 mev. For a fission spectrum, the cross section is 0.230 barn; the cross section is about the same as that for the $P^{31}(n,\gamma)P^{32}$ produced by thermal neutrons. In hair, however, there is less than 1% as much phosphorus as sulfur present, and thermal neutron production of P^{32} from P^{31} may be neglected. The sulfur concentration in K's hair was found to be 45.6 mg/gm, and the P^{32} activity was 27 d/m/mg of sulfur. The fast neutron flux incident to the head was estimated from the relation

$$N = N^*/\lambda = \phi \frac{\sigma f A}{W}$$

or

$$\phi_S = \frac{N^* W}{\lambda \sigma f A}$$

where N^* = activity per gram of hair = 20.5 d/sec/gm; W = atomic weight of sulfur = 32; λ = decay constant for $P^{32} = 5.65 \times 10^{-7}$ sec^{-1}; σ = activation cross section = 0.230×10^{-24} cm^2; f = fractional concentration of sulfur in hair by weight = 0.0456; A = Avogadro's number = 6.02×10^{23} atoms; and ϕ_S = total fast

Table XXVII—Effect of Thickness of Moderator on Induced Sodium-24 in $NaHCO_3$ Water Solution

CH_2 Thickness (in.)	Sodium-24 (c/m/rad)
0	80
3	80
6	100
9	100

Table XXVIII—Average Total-Body Fast Neutron Dose from Body and Blood Sodium-24 Concentration and Serum Sodium Specific Activity

Subject	Body Sodium-24 ($\mu c/kg$)	Blood Sodium-24 ($m\mu c/ml$)	Serum Na S.A. ($m\mu c/mg$)	Dose Estimates: Body Sodium-24 (rads)*	Dose Estimates: Blood Sodium-24 (rads)†	Dose Estimates: Serum Na S.A. (rads)
K	4.10	5.3	3.77	835	880	968
D	0.014	0.016	0.01	2.8	2.6	2.6
R	0.007	....	....	1.4	....	....

*Assuming total body sodium concentration in man and the burro is the same.

†Assuming the blood sodium concentration in man and the burro is the same.

neutron flux seen by sulfur (n/cm²). Thus

$$\phi_S = \frac{20.5 \times 32}{(5.65 \times 10^{-7}) \times (0.230 \times 10^{-24}) \times 0.0456 \times (6.02 \times 10^{23})} = 1.78 \times 10^{11} \text{ n/cm}^2$$

The flux-to-dose conversion factor for neutrons above the sulfur threshold is 3.63×10^{-9} rad/n/cm², giving a dose estimate of ~650 rads incident to the region of K's head. With the source as indicated in Fig. 8, the total neutron flux in the head direction was essentially an undegraded fission spectrum. Under these conditions, a comparison with Godiva II measurements indicates that neutrons above the sulfur threshold deliver approximately 25% of the total neutron dose. The total fission neutron dose incident to the head was, therefore, about 2,600 rads. Extrapolation of neutron dose measurements performed with the Godiva II assembly indicates 1.5×10^{17} fissions would deliver a dose of 3,700 rads at a distance of approximately 100 cm from the source, the distance assumed in Fig. 8 as the most probable position of K's head during performance of the stirrer operation. Although the two estimates differ appreciably, it is not surprising since the position of the head was not known. For this reason, the estimate from the P^{32} measurement is considered the more likely value.

Consideration of the distances and source characteristics shown in Fig. 8 suggests that the upper abdomen in the region of the xiphoid cartilage probably received the maximum incident neutron dose. Although other regions of the body were closer, the amount of moderator between the source and body surface probably resulted in a considerably lower dose. A sample of K's xiphoid cartilage was analyzed also for sulfur, phosphorus, and P^{32}, and the results used to estimate incident fast neutron dose to this region of the body. The phosphorus-to-sulfur ratio was 0.028, and phosphorus capture of thermal neutrons could again be neglected.

The P^{32} activity was 4.7 d/sec/gm, and the sulfur fraction by weight was 0.00926. The incident neutron flux (ϕ_S), calculated in the manner shown previously, was 2.05×10^{11} n/cm². Using the same conversion factors and assumptions used previously, the incident fast neutron dose to K's upper abdominal area is estimated at about 3,000 rads. Extrapolation of neutron dose data from the Godiva II assembly gives an estimate of ~5,000 rads from 1.5×10^{17} fissions at the distance of the xiphoid (Fig. 8). The agreement between the two estimates is considered satisfactory when the uncertainty in K's position, the increasing amount of moderator between the source and surface, and the differences in the two sources are considered. The increasing amount of moderator would tend to decrease the fraction of dose due to neutrons above the sulfur threshold.

A sample of testicular tissue was analyzed also, but the P^{32} activity was below the level of detection. Failure to detect P^{32} in the testis supports the conclusion that moderation of the source spectrum by intervening water occurred with degradation of high energy neutrons to below the sulfur threshold. It may be concluded also that a significant absorption of thermal neutrons occurred, since the phosphorus-to-sulfur ratio in the testis was 0.11.

Metal buttons were removed from the coveralls worn by K at the time of the accident, and their induced activity was measured. The radiation emitted was identified as the annihilation radiation from Cu^{64}. Unfortunately, the buttons (which varied in activity by a factor of ~1.7) were not labeled as to location on the coveralls as they were removed. The activity of the various buttons, corrected to time of accident is shown in Table XXIX. Calibration of similar buttons taken from the same type of coverall was attempted during the Godiva experiments, designed to study the effect of moderator on induced Na^{24} activity in the 18-liter water-$NaHCO_3$ phantom. The activity induced in the buttons resulted from thermal neutron capture. However, a free-air measurement with Godiva II indicated that the thermal neutrons from Godiva itself could be neglected. When the buttons were activated on the front of the phantom with 3, 6, and 9 in of polyethylene moderator between the source and the buttons, the measurements indicated an essentially constant ratio of 0.17 rad/c/m. The incident neutron dose indicated by each button is shown in Table XXIX. These results show a variation of from 1,500 to 2,500 rads and are not inconsistent with the neutron dose consideration given previously.

Table XXIX—Cu^{64} Activity of Buttons from Coveralls Worn by K (corrected to time of accident)

Button Number*	Activity (c/m)	Incident Neutron Dose (rads)
1	11,100	1,900
2	13,500	2,300
3	11,300	1,900
4	9,800	1,700
5	8,730	1,500
6	14,500	2,500
7	9,480	1,600
Average	11,200	1,900

Position on coveralls not known

Gamma-Ray Dose

In many respects, estimation of K's gamma-ray exposure was more difficult than the estimation of neutron dose. Gamma doses to D and R were reasonably well known, since both were wearing film badges, but no film badge results were available for K, and the nature and geometry of the source were important in the estimation of his gamma dose by other means. The gamma-to-neutron dose ratio would be expected to vary from point to point because of the moderating conditions. Three different components contributed to the total gamma-ray dose. First was fission gamma rays; second, the dose due to capture gamma rays from the hydrogen in the moderator; and, third, that due to residual fission products and induced activities in the tank and its contents. As far as K was concerned, the third portion may be neglected. D and R (as will be seen later) received a major portion of their gamma-ray exposure from the residual fission products and associated activities.

Estimates of the gamma-to-neutron dose ratio in the area occupied by K may be made from the results of the Oak Ridge National Laboratory's mock-up experiment following the Y-12 accident.[51] These experiments indicated a dose ratio of approximately 3. Although a value of 3 may roughly apply to the upper portion of K's body, it certainly would not apply to the lower portion in the directions of large thicknesses of hydrogenous moderator. In the presence of significant amounts of hydrogenous moderator, the gamma-to-neutron ratio increases rather rapidly owing to hydrogen capture gamma rays.[52] Neglecting fission gamma rays, the capture gamma-to-neutron dose ratio increases rapidly from 0.5 at 20 cm of water shielding to 5 at 50 cm, and 20 at 100 cm. Application of these observations to the distances and source conditions shown in Fig. 8 suggests that the incident gamma-to-neutron dose ratio over K's body may have varied from 3 to 1 at the head and xiphoid region to 10 to 1 at the feet. In the regions of high ratios, however, the neutron dose was very significantly decreased owing to fast neutron moderation. Experiments indicate that the fast neutron dose relaxation length[63] is of the order of 10 cm in a water shield. Some build-up close to the source occurs, but one would estimate that the actual fast neutron incident dose to the lower portion of K's body was only a few per cent of the unmoderated value in the region of the head and xiphoid shown by the sulfur measurements. Therefore, even though the gamma dose to the lower portions of the body may have been 10 times as great as the neutron dose, the total dose to this area was undoubtedly only a fraction of that to the upper portions. From these purely theoretical considerations, an estimate of 3,000 to 4,000 rads as K's average total-body gamma-ray dose seems reasonable.

Table XXX—Transmission Data for Co^{60} Gamma Rays

Position	Dose Rate (mr/hr)*	Transmission Factor
Film badge rack	1.86	7.0×10^{-6}
R's station	4.23	1.6×10^{-5}
D's station	5.64	2.1×10^{-5}

*Ionization chamber readings from 20-curie source placed near the solvent-treating tank.

Located in a rack 137 ft from the center of the criticality excursion were 46 previously unexposed film badges. After the accident, these films showed an average gamma-ray exposure of 670 ± 90 mr. Calculations showed that only about 4% of the dose indicated by the film badges could have resulted from residual fission product gamma-ray exposure, which certainly constituted a negligible fraction of K's dose.

An experiment was carried out to measure gamma-ray transmission from the source to the film badge rack before the general geometric features of the area were disturbed; only the contents of the solvent-treating tank had been transferred. Using a Co^{60} source of approximately 20 curies placed at about 6 in from the outer wall of the solvent tank at a level approximating the level of the reaction, gamma-ray dose rates were measured at D's and R's positions and at the film badge rack. Measurements were made with 0.025-r ionization chambers and gamma rate meters, and agreement between the two methods was quite good. The results of these measurements are shown in Table XXX. Calibration of the Co^{60} source gave a dose rate of 4.4 r/min at a rate of 1 ft, which is in good agreement with the rated source strength of 20 curies.

Using a transmission factor of 7×10^{-6} and a reading of 670 mr at the film badge rack, extrapolation to a position 1 ft from a point source gave a dose rate of 4.4 r/min at a rate tank gave a total gamma dose of 9.57×10^4 rads. Similar extrapolations from D's and R's stations could not be made, since their film badges showed readings of 118 and 31.5 r, respectively, most of which was from fission gamma exposure accumulated during subsequent trips near the source. That this was the case is indicated by the gamma-ray transmission factors at their respective stations. These data indicate that D's prompt gamma dose was

approximately 3 times that measured at the film badge rack (or about 2 r), and R's dose was about 2.3 times the dose at the rack (or approximately 1.5 r).

Extrapolation of the total gamma dose at 1 ft from a point source at the center of the fission excursion (assuming no moderator effect) to the dose incident to K's head and xiphoid regions gives a value of ~10,000 r to the head and a slightly higher value to the upper abdominal area. Considering the nature of the data and the extrapolations involved, this is considered to be in reasonable agreement with the values of 7,800 and 9,000 rads derived from the incident neutron dose which were estimated from the P^{32} measurements, and a gamma-to-neutron dose ratio of 3 to 1. The source was not actually a point source, and certainly some of the gamma exposure to the film badges might have been from gamma-ray production in areas away from the immediate source. It is possible also that the observed gamma-ray transmission factors, which varied greatly from point to point because of intervening equipment, etc., were low compared to what actually was the case.

Another estimate of gamma-ray dose may be made strictly on theoretical grounds. Considering fission gamma rays only, the total prompt gamma energy release is variously quoted as from 7.5 to 12 mev per fission. Using 7.5 mev and 1.5×10^{17} fissions, the total gamma energy available is about 1.5×10^{12} ergs. At 100 cm, the energy flux would be 1.5×10^{7} ergs/cm^2. For gamma rays of intermediate energy, an energy flux of 3,000 ergs/cm^2 results in a dose of 1 rad. Therefore, the total fission gamma dose in the region of K's head would be around 4,500 rads and in the region of his xiphoid greater than 6,000 rads. These estimates agree within a factor of 2 with the 3 to 1 ratio of gamma-to-neutron dose, and in view of the complications this is perhaps as good as can be expected.

Table XXXI—Summary of Estimates of K's Radiation Exposure

Region and Conditions	Fast Neutron Dose (rads)	Gamma Dose (rads)	Total Dose (rads)
Head (incident)	2,600	7,800	10,400
Upper abdomen (incident)	3,000	9,000	12,000
Total body (average)	900	3,000-4,000	3,900-4,900

Total Dose (Case K)

As stated previously, both the fast neutron and the gamma-ray doses to K were completely anisotropic. It is possible, therefore, only to estimate his average total-body dose and total incident dose to the head and upper abdominal regions. In many respects, the neutron dose estimates were more satisfactory than those of gamma dose. The most reliable estimates of incident gamma dose to the head and upper abdominal areas, where the effects of the water moderator were minimum, are perhaps those based on a fast neutron-to-gamma dose ratio of 3. Taking the various factors into consideration, what seems to be the most reasonable estimates of K's radiation exposure are summarized in Table XXXI.

The RBE of fission neutrons for the production of lethality in the mouse is approximately 2.[54] The mouse, however, is thin compared to the neutron mean free path, and the neutron dose may be assumed to be isotropic throughout and over the entire body. Since these were not the conditions of K's exposure, it is virtually impossible to choose an appropriate RBE value for the fast neutron component of his dose. Thus, no attempt is made to estimate average total-body exposure in rems. It seems advisable to say only that his average total-body exposure was probably between 3,900 and 4,900 rads, of which about 900 rads was due to fission neutrons, and that a considerably greater portion of the dose was delivered to the upper half of the body than to the lower half.

SECTION VII

Health Physics Studies and Area Radiation Levels

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With the collaboration of Jerome E. Dummer, M.S., Dean D. Meyer, B.S.,
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THE COMPLEX of buildings at the Los Alamos Scientific Laboratory known as DP West is an area in which the principal health hazard is alpha radiation. In these buildings there are some other radiologic hazards, but the greater part of the work in the area involves plutonium. An outline of the principal structures is shown in Fig. 9. The accident occurred in Room 218 of Building 2, and the layout of this room is shown in detail in Fig. 2.

Whenever plutonium in kilogram quantities exists, the possibility of a critical excursion can never be zero. In this instance it was the considered opinion of supervision and also of the Laboratory's Criticality Safety Committee that adequate safeguards for all processes existed, that a critical mass of plutonium could not exist in any one place. For this reason the monitoring personnel assigned to the building were primarily concerned with incidents or accidents involving alpha contamination.

Detection of the Accident

The first indication of the accident outside its immediate vicinity was the activation of a criticality alarm in the north wing of Building 3, the only such alarm in the area. Additional alarms were on order but had not yet been installed. The workers in Building 3 could find no sign of trouble and assumed that the alarm had gone off accidentally for some unknown reason.

At Los Alamos there are eight continuous-recording gamma monitors about the community. Three of these instruments recorded the excursion. In each case the detector was in an unusually favorable position for observing the burst as compared to the other five. Distances from the accident were 3,250, 7,700, and 12,900 ft. Although some of the other stations were closer to the burst, they were well shielded by the terrain from the low-level radiation which was recorded.

The burst was also recorded on the background monitor located on the top of Omega West reactor, about 3,900 ft away in a deep canyon. A sensitive ion chamber for tritium monitoring located in Building 3 of DP West recorded the gamma radiation of the burst, as did a multiple-channel tritium monitor in the same area. All the recorders gave good evidence that only a single burst occurred.

The burst was also indicated on several non-recording instruments. Unusual activity in the alpha hand counters in the area was noted at about the time of the excursion. The activation of the criticality alarm in Building 3 was mentioned above. Approximately five minutes elapsed before this was reported to the area monitoring supervisor. He realized the significance of the report, and this realization subsequently disclosed the site and nature of the accident.

As proof that no significant radiation was received at residential areas, film badges which had been planted on Dec. 22, 1958, were developed on Jan. 5, 1959, and indicated no exposure. The minimum gamma exposure detectable by film badge is about 0.04 r. These films were located at 2,000, 4,700, 7,700, and 12,900 ft from the accident site.

Monitoring and Dosimetry of Personnel

Although film badges capable of recording exposures in the range of 1,000 r were routinely issued to nearly all employees involved in plutonium or uranium operations in this area, the wearing of these devices at all times was not required. Normally the only penetrating radiations to be expected were gamma radiations from fission products contaminating U^{235}, high-energy gamma from contaminants of U^{233}, and low energy x-rays from plutonium. When significant exposure to these radiations was not anticipated, individuals frequently did not remove their film badges from the storage rack,

which was located about 137 ft from the position of the excursion.

The practice of planting film badges in the buildings was not in general use. Films were planted only in vaults where appreciable quantities of fissionable material were stored.

Since a large number of persons involved in the initial appraisal of the accident were not wearing film badges, it was necessary to estimate their exposures. At the same time it was decided to estimate the exposure of everyone employed in the immediate area. A check was made of all persons having access to the site to determine their presence or absence at the time. All craft workers were found to have left the area by 4:30 p.m. on the day of the accident.

A personal interview was held with each individual who normally worked in the area, and with those persons who held badges which would permit their access to the area. In this interview it was determined if and when the person was wearing a film badge and what type of work had been done the previous week while the film badge was worn. Further, an account of the movements of these persons was obtained from 4:30 p.m. to 5:00 p.m. on the day of the accident.

At 6:00 p.m. most of the film badges issued to DP West personnel were collected and processed. This included 46 film badges which were not worn at the time of the accident but were located in the storage rack 137 ft from the burst. The highest readings were from the badges of D and R, who assisted K from the room following the nuclear excursion. Unfortunately, K was not wearing his film badge at the time of the accident. The average exposures and standard deviations of the exposures to the 46 films in the film badge rack were: 0.67 ± 0.09 r gamma; 0.23 ± 0.04 rem thermal neutrons; and 1.3 ± 0.21 rem fast neutrons. The fast neutron exposure was determined with Kodak's Personal Monitoring Film Type 2B, as designed by J. S. Cheka of Oak Ridge.[55] The thermal neutron and gamma exposures were evaluated by the method devised by Kalil.[56]

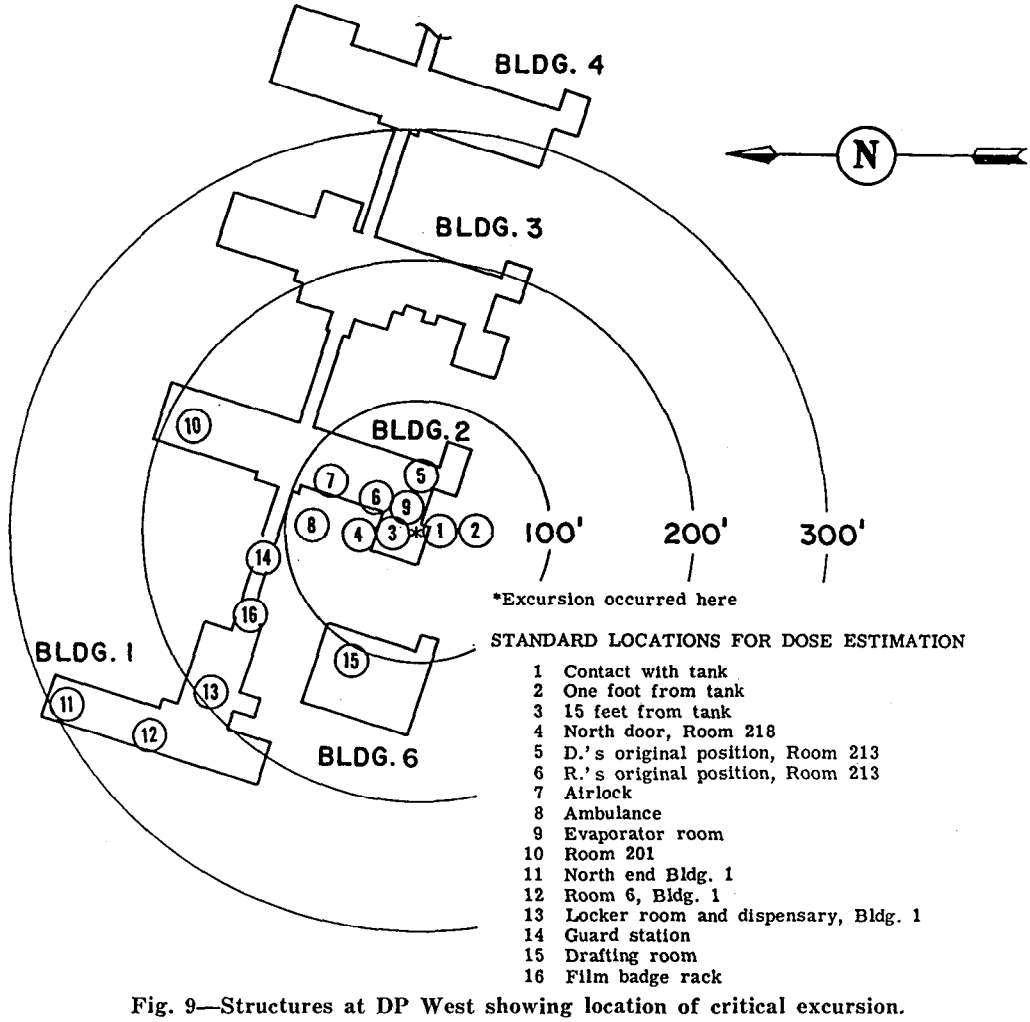

Fig. 9—Structures at DP West showing location of critical excursion.

Numerous field surveys were made with both G-M tube and ionization chamber instruments. Nearly 100 separate dose rate measurements were made and recorded between 4:40 p.m. on Dec. 30, and 3:30 p.m. on Jan. 1. In many cases dose rates measured at the same location did not decrease according to the $t^{-1.2}$ law, probably because of minor differences in location and differences in personal judgment in reading the instruments. For purposes of calculation the expected readings at specific locations of interest were calculated according to $t^{-1.2}$. The average of these values was used for dosage calculation in some instances.

Attenuation factors at a number of different locations were determined several days after the accident (Section VI). From these data, dose rates at various positions were related to the dose at the film badge rack.

No practical experiment was conceivable for determining the neutron attenuation. For all positions closer to the burst than the film badge rack, a simple inverse-square attenuation was assumed. However, nuclear-track films located about 270 ft from the burst indicated an absorption attenuation of 1/5 in addition to the inverse-square decrease. It was assumed, therefore, that the absorption attenuation increased linearly with distance beyond the film badge rack.

For lack of better data, it was assumed that the gamma dose fell inversely as the square of the distance with a similar absorption attenuation factor at 270 ft from the excursion, and that this attenuation increased linearly with distance beyond the film badge rack. There has been no opportunity to verify this assumption other than recognition of the fact that no gamma exposure was indicated on film badges located about 270 ft from the burst.

It was also determined from film badges that the average exposure to personnel in Room 201 of Building 2 was 0.23 r gamma with 0.14 rem fast neutron exposure and no thermal neutron exposure.

The effect of large-diameter sources on meter readings taken near the tank was computed. It was found that for a source 15 in in diameter, a monitoring instrument in contact with the tank (19 in from center of tank) would respond according to inverse-square predictions. For a source the size of the tank, i.e., about 38 in in diameter, the instrument response would be 1.5 times that predicted by simple inverse-square at the surface of the tank, about 1.1 times inverse-square predictions at 9 in from the surface of the tank, and in accordance with inverse-square prediction at 20 in from the tank. For the burst the small-diameter source was assumed, and for the residual gamma, the large source was assumed (due to the mixing action of the stirrer).

Table XXXII—Tabulation for Health Monitor U

Time	Location	Calculated Exposure (r)
4:35 p.m.*	At coverall check station	1.50
4:40 p.m.	Answered R's call for help	
4:41 p.m.	Checked K for alpha in air lock	0.07
4:44 p.m.	Entered Room 218 by north door	
	Assume: First 5½ min in location 3	2.46
	Assume: ½ min in location 2	6.52
4:50 p.m.	Left Room 218 to get gamma survey meter in Room 201	
4:52 p.m.	Arrived to hear "clear area call" and left area	
Total		10.55
Burst fast neutron (rem)		3.08
Burst thermal neutron (rem)		0.54

Film badge worn for entire operation indicated 9.3 r gamma

Total exposure 9.3 + 3.08 + 0.54 = 12.92 rem

*Time of burst.

Table XXXIII—Tabulation for Ten Individuals

Individual	Total Dose (rem)
S	12.46
T	8.18
U	12.92
C	1.09
W	6.83
X	10.12
Y	0.51
Z	10.24
R	53.74
D	133.54

By using the monitoring measurements at several positions close to the tank, integration of a $t^{-1.2}$ function, the total attenuation factor from the tank to the film badge rack determined with Co^{60}, and the effect of the 38-inch-diameter source of gamma, it was determined that 96% of the radiation exposure as recorded at the film badge rack was due to the burst plus one second thereafter, and 4% due to the residual gammas from the fission products. Although this is not the ratio of prompt gamma mev to residual gamma mev over the time period considered, Harris[57] reports that he has also observed that most of the gamma radiation detected by film badges is due to the burst plus one second and not due to the residual gammas.

Had every individual in the general vicinity been wearing a film badge at the time of the critical excursion, the task of recording all

Table XXXIV—Estimated Exposure of Case K, Assuming Three Possible Positions at Time of Burst

Time	Location	Chest at Contact (r)	Back When Chest Was at Contact (r)	Chest at 6 in (r)	Back When Chest Was at 6 in (r)	Chest at 12 in (r)	Back When Chest Was at 12 in (r)
4:35 p.m.	At tank	4×10^4	1.9×10^4	2.3×10^4	1.3×10^4	1.5×10^4	9.1×10^3
4:35 p.m.	Residual, 1 to 20 sec	2.5×10^3	1.2×10^3	1.4×10^3	8.1×10^2	9.5×10^2	5.7×10^2
4:33-35 p.m.	Remainder insignificant with respect to instantaneous exposures						
Total		4.25×10^4	2.02×10^4	2.44×10^4	1.38×10^4	1.6×10^4	9.67×10^3

personnel exposures would have been relatively easy. As has been mentioned above, this unfortunately was not the case. Many hours were spent interviewing a large number of workers and charting their movements on a minute-by-minute basis. In some cases conflicting statements had to be resolved. Eleven individuals were of particular interest even though some of them were some distance from the excursion at the time of the accident. The following listing indicates the positions of these people at 4:35 p.m., the time of the accident:

S, health Monitor	Bldg. 2 mezzanine, 200 ft from excursion
T, health Monitor	Dispensary, Bldg. 1, 160 ft from excursion
U, health Monitor	Check station, Bldg. 2, 95 ft from excursion
C, monitoring Supervisor	Office, Bldg. 1, 240 ft from excursion
W, group Leader	Office, Bldg. 1, 260 ft from excursion
X, chemical Operator	Room 201, Bldg. 2, 180 ft from excursion
Y, nurse	Office, Bldg. 1, 210 ft from excursion
Z, operation Supervisor	Drafting room, Bldg. 6, 100 ft from excursion
R, chemical Operator	Room 213, Bldg. 2, 36 ft from excursion
D, chemical Operator	Room 213, Bldg. 2, 43 ft from excursion
K, chemical Operator	Room 218, Bldg. 2, Beside excursion tank

The movements of these as well as many other people was plotted for each minute of the next 25 minutes. From these findings and from the attenuation studies mentioned previously, a dose was estimated for each individual. Table XXXII shows a typical tabulation for Health Monitor U who, it will be seen, was wearing a film badge. His exposure was also calculated from his movements, however, and the agreement of the two figures for gamma dose is quite acceptable. It will also be noted that neutron doses are listed in rem, as this is the standard method of reporting such exposures for record purposes even though the RBE factor may be open to some question.

Similar calculations were made for all other individuals in the vicinity, and Table XXXIII indicates their estimated doses. Exposures to all other personnel were lower than the ones in this table.

Using the factors previously mentioned, K's gamma dose from the burst and residual gammas was estimated for three possible positions at the time of the burst. In Table XXXIV estimates are given for his chest and back assuming that his chest was in contact with the tank, 6 in from the tank, and 12 in from the tank. It should be pointed out, however, that these calculations were based on the assumption that K was standing on the floor rather than on the stepladder. The latter assumption was used in the calculations in Section VI, which explains the difference in results. The best guess is that he *was* on the stepladder.

SECTION VIII

Report on Other Personnel Exposed

Thomas L. Shipman, M.D.

IN THE CASE of an accident such as the one described in this paper most of the interest is focused on the most heavily irradiated case. As shown in Section VII, however, a number of other individuals received measurable overexposures. Of these, only the two significant exposures will be considered here: Case D, whose dose was approximately 130 rads, and Case R, with a dose of approximately 35 rads. In both of these cases the majority of the exposure was to gamma rays rather than neutrons. In both cases the only significant findings are the alterations in the blood counts, and it is of interest to compare these with the changes observed in the Rongelap natives[58] and in the Oak Ridge cases.[6]

Case D

D, it will be recalled, was at work in the room next to the one in which K was exposed (Fig. 2). D's exposure from the critical excursion itself was negligible, a total of only about 3 rads. He did, however, enter the room of the accident twice, and in these visits he unquestionably passed within a few feet of the highly radioactive tank at least four times. His total dose was approximately 130 rads, neutrons and gamma.

D's blood chemistry has already been discussed in Section III. It may be stated that at no time did he show evidence of any signs or symptoms, objective or subjective, which could not be attributed to his concern for his fellow worker, K. He had no nausea or vomiting, no weakness. He developed no petechiae, no loss of hair, and his only apprehension was for K. Some months later he developed one transient respiratory infection and also underwent a full-mouth extraction of his teeth for an infectious process that antedated the accident.

The changes in his blood picture, however, were both interesting and dramatic (Fig. 10). D's preexposure blood counts were well known. Over a period of approximately 10 years he had had 24 blood counts done, all of them by the same group of technicians, and one of these technicians did all save a few of the postexposure counts.

Leukocytes—D's preexposure white blood cell count averaged 8,300/mm³, with extremes of 10,600 and 6,200. His absolute granulocyte count averaged 4,785, and his average lymphocyte count was 2,650/mm³. The first postexposure count, done about two hours after the accident, showed a rise in total leukocytes and in granulocytes to 10,900 and 7,200, respectively, but a drop in the absolute lymphocyte count to

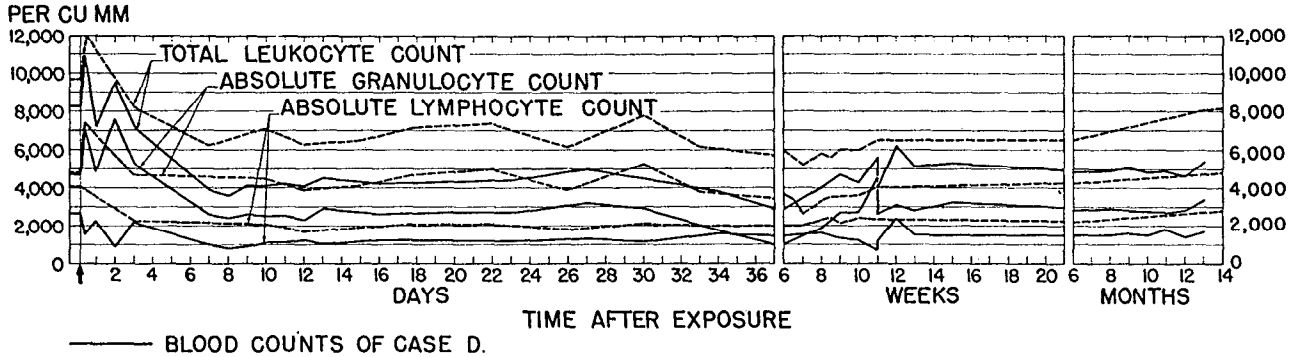

Fig. 10—Blood changes in Case D compared with those of the Rongelap natives.

1,463. During the next 48 hours the leukocyte and granulocyte counts fluctuated rather widely, the lymphocyte count showing a moderate fluctuation. On the morning of the third day after the accident, the total white cell count was 7,200, and the absolute granulocyte and lymphocyte counts were 5,328 and 1,224 respectively. All counts dropped progressively until the eighth day, at which time a low point seemed to be reached. On this day the total WBC was 3,600 and the granulocytes and lymphocytes fell to 2,484 and 864. This drop was followed by a slight rise, followed by a leveling off until about the end of the fourth week when the total white cell count and the granulocyte count seemed to drop again.

The blood counts taken on the 34th and 37th days were done at a laboratory in another state. It would appear, however, from a comparison with the cases of the Rongelap natives,[58] as well as other accident cases,[1, 2, 4, 5] that this drop at around the fifth week is to be expected, although the reason for this is not yet clear.

During the 11th week D had a mild influenza-like infection, with both respiratory and gastrointestinal symptoms. This cleared up without any treatment other than rest at home. From that time on there was surprisingly little change in the counts of any of the white cells through the 13th month. The final counts at the time of writing showed a total WBC of 5,400, a granulocyte count (absolute) of 3,402 and a lymphocyte count of 1,674. These final counts, however, all show an increase above the previous month, and it may be that recovery in the next year will be more pronounced.

Abnormal Cells—During the first five months after the accident, atypical lymphocytes were seen in almost every smear in percentages varying from 1 to 6. One binucleated lymphocyte was seen in the third month. There did not, however, seem to be any significant pattern in the occurrence of these cells. There was no significant or consistent shift in the ratio of segmented and band-type granulocytes.

Erythrocytes—The preexposure average number of red cells was 5.38 million, with an average hemoglobin level of 16.9 gm. Both showed a tendency to drop somewhat in the first two months, although this drop was neither dramatic nor consistent. The lowest levels noted in this period were 4.77 million and 15.3 gm. The average red cell count in the 13 months following the accident was 5.03 million, and the average hemoglobin level, 15.9 gm. No reticulocytes had been noted in any preaccident smears but some were seen in almost all subsequent smears through the third month. The largest number noted in any smear was 2.6%.

Table XXXV—Average Blood Levels for R

	Eight Years Preaccident	Thirteen Months Postaccident
RBC	5.19 million	5.15 million
Hgb	16.2 gm	16.4 gm
WBC	6,471	5,300
Granulocytes (absolute)	3,980	3,240
Lymphocytes (absolute)	1,870	1,570

Platelets—The number of platelets seemed to show a slight fall until about the 10th week, after which there was a return to normal. It is questionable whether this drop was of statistical significance, and it certainly would not be proper to compare our findings with those of other laboratories using other methods of counting platelets or even other technicians using the same method. In this one case, however, it appears that alterations in the platelet count were of no clinical significance.

Had it not been for previous experience with cases of radiation exposure in the same general dose range, D's alteration in blood counts would have been rather alarming. He followed, however, much the same course as previous cases, and his general state of well-being was entirely unimpaired. At the time of the accident he was 37 years old, tall, lean, and in unusually good physical condition. He was proud of his muscular vigor and firmly denied any sign of weakness or any other symptom, either subjective or objective.

Case R

As has been described, R received a total dose of approximately 35 rads, principally gamma radiation received when he entered the room in which the accident occurred in order to assist K. He passed twice within a few feet of the tank in which the reaction took place. His dose is quite compatible with these actions.

Studies on R were confined largely to those indicated from a clinical point of view. His blood findings are shown in Table XXXV, where it will be noted that his erythrocyte count dropped less than 1% on the average, while his average hemoglobin level actually increased about 1%. These changes are regarded as of no statistical significance.

The decrease in total leukocyte count, granulocyte count, and lymphocyte count are obviously of some significance, although his most recent counts are well within his preaccident range. Like D, R at no time showed any unusual signs or symptoms and he lost no time away from his work.

Discussion

The records for each of these men present food for speculation. On the chart showing the blood levels of D, there are also shown the average blood levels of the Rongelap natives over a similar period (Fig. 10). The native controls had levels slightly higher than D to start with and the Rongelap counts did not go as low as his, although their radiation doses were slightly higher, 175 rads as compared to 130 rads. But after more than a year neither the levels of the natives nor those of D had returned to their previous norms.

D received his exposure within a few minutes, while the Rongelap doses were spread out over a period of about 48 hours. It is quite possible that we have here an indication of dose rate dependence, although it would be hard to prove this statistically. Furthermore, the Rongelap natives undoubtedly had among them a number of people with chronic and acute infections which possibly had the effect of stimulating leukocyte formation. There is the suggestion in the case of D that all his white cell levels were slightly higher after his respiratory infection in the third month. His levels might have been higher yet had he had a number of mild infections.

The total white blood cell count is of little or no significance in the first 48 hours after exposure to radiation. The absolute lymphocyte count, on the other hand, falls promptly and steadily. Its rate and depth of fall may be used as of some prognostic significance. After 48 hours all leukocytes become fewer, reaching a low point in eight or nine days. After a period of apparent improvement they will again decrease at about the end of the fifth week. Complete return to normal, however, will be extremely slow.

The case of D, together with those of the Rongelap natives, indicates quite conclusively that individuals exposed in the 100- to 200-rad range will in all probability show little or nothing in the way of subjective symptoms. Impressionable patients may become nauseated and complain of weakness, but this is certainly not the rule.

R also presents some interesting points. It is possible to demonstrate a decrease in his leukocyte counts, but only because there existed a firm base line of previous counts. Prior to the accident his highest recorded white count was 9,000, his lowest 4,900. After the accident his highest was 7,200, his lowest 4,100. But without previous counts for comparison with a series of postexposure counts, it seems obvious that such routine hematology is of little diagnostic or prognostic value for exposures in this general range. More research in this area is naturally indicated, but at the present time routine blood studies contribute little to the clinical evaluation of exposures below 50 rads except to provide reassurance that the dose was not far larger than estimated.

On the night of the accident, both D and R were permitted to go home, although both were requested to report to the hospital for observation the following morning. There seemed to be no medical reason for hospitalizing them, and it was felt very strongly that such a course was important for the morale of the men and their families.

SECTION IX

Summary

Thomas L. Shipman, M.D.

THE PRECEDING discussions have presented in considerable detail the various facets of a nuclear criticality accident which resulted in the death of one man and substantial exposures to two others.

The critical excursion took place in a tank of solution from which plutonium was being recovered, and the number of fissions was calculated to be $\backsim 1.5 \times 10^{17}$. The average whole body dose to K has been estimated to be between 3,900 and 4,900 rads, with the incident dose to the upper abdomen calculated to be approximately 12,000 rads, neutrons plus gamma. Two other workers received total doses of approximately 130 and 35 rads, respectively, most of this being gamma radiation.

K went promptly into shock and was unconscious within a few minutes of the accident. Neither of the other men showed any signs or symptoms of injury other than alterations in their blood counts. By heroic medical treatment it was possible to get K out of his state of shock, but he died 35 hours after the accident. His clinical course and treatment are described in detail. The most dramatic clinicopathological changes were observed in the hemopoietic and urinary systems. Lymphocytes were not found in the circulating blood after the eighth hour, and there was virtually complete urinary shutdown despite administration of large amounts of fluids.

The gross and microscopic pathology are discussed in Section IV. The changes in the heart and in the gastrointestinal tract were pronounced, as were the changes in the bone marrow.

Section V describes a number of special studies which were carried out, and one of the most significant of these relates to the distribution of plutonium in the man's body. He had worked with plutonium for a number of years, although he had never had a significant overexposure. A surprisingly large percentage of the material was recovered from the pulmonary lymph nodes.

Sections VI and VII are concerned with the dosimetry of the fatal case and give in detail the various studies which were carried out to establish the dose levels of other workers less seriously involved. In the case of K it is clearly apparent that the dose varied widely in different parts of the body and even in different parts of the same organ, such as the heart or the stomach.

Cases D and R received doses of approximately 130 and 35 rads, respectively. The changes in their blood counts are given in Section VIII. It appears that the changes following exposures in excess of 100 rads are characteristic and of clinical significance, whereas doses below 50 rads produce blood changes which are too slight to be of any diagnostic value.

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