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## FATAL RADIATION SYNDROME FROM AN ACCIDENTAL NUCLEAR EXCURSION\*

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THE acute radiation syndrome will almost surely be encountered from time to time as accidents occur in the rapidly expanding nuclear-energy industry. Nuclear power is already becoming economically competitive with conventional sources.<sup>1</sup> In view of the present magnitude of these activities it is remarkable that there have been so few accidents already, a testament to the care that has been exercised in safety control of highly hazardous undertakings during the past twenty years or so.

The first fatal accidental nuclear excursions occurred in 1945 and 1946 at Los Alamos.<sup>2</sup> One of the victims died nine days, and the other twenty-four days after exposure. The whole-body dosages were estimated respectively to be 1350 and in excess of 297 rads.<sup>3</sup> The next accident occurred at the Boris Kidric Institute at Vinca, Yugoslavia.<sup>4,5</sup> Six men were exposed, and 1 died. All these men received bone-marrow transplants in the fifth week,<sup>6</sup> but it is not entirely clear just what this contributed to the recovery of the surviving subjects. The patient who died received an estimated 640 rads of whole-body radiation, and the rest less than 600.<sup>3</sup> The most recent and perhaps most thoroughly studied accident was at Los Alamos in 1958, when an operator received an estimated total dose of 3900 to 4900 rads and died thirty-four and three-quarters hours later.<sup>7</sup> All these accidents have resulted from attainment through error of a critical mass and a resulting nuclear chain reaction. When fissionable substances, such as an isotope of uranium ( $U^{235}$ ), become confined in a sufficient mass and appropriate environment so that their fast neutrons are slowed to the point where their capture by fissionable atoms becomes significantly probable, a vicious circle of fission by capture and further release of neutrons is set in motion. This is a nuclear chain reaction. Some of these lethal or near lethal accidents have caused a

single burst of neutrons lasting only a few microseconds; others may have involved resurgent bursts as the responsible solution has boiled up and receded only to reach its critical mass again.

The present account concerns a fatal accident in a  $U^{235}$  recovery plant. The patient was cared for in a large general hospital by a staff that had had no previous experience with a similar problem. The patient survived for forty-nine hours after receiving ten to twenty times a lethal radiation exposure. He probably received the heaviest radiation dose of any victim of a nuclear accident, and this was the first fatality in private industry. Many medical and administrative difficulties were faced, and similar ones will be encountered at the time of any similar accident in the future.

### CASE REPORT

Late in the afternoon of July 24, 1964, a 38-year-old married father of 9 was pouring a "dirty" mixture containing  $U^{235}$  from a polyethylene cylinder 12.5 cm. in diameter and 120 cm. high into a tank 63 cm. in diameter containing sodium carbonate. A critical volume was attained by the new geometry near the completion of this operation, and a nuclear excursion occurred. The patient recalled a flash of light and was hurled backward and stunned, but did not lose consciousness. He immediately ran from the building to an emergency shack 200 yards away, discarding his clothing as he ran. There he was joined by 4 other occupants of the plant who had been alerted by the radiation alarm system. Almost at once the patient complained of abdominal cramps and headache, vomited, and was incontinent of diarrheal stool, which according to his colleagues was bloody. He was wrapped in warm blankets and taken to a nearby hospital (but not admitted) and transferred at once to the Rhode Island Hospital. He arrived at 7:49 p.m., 1 hour and 43 minutes after the accident.

He was taken at once to an isolated section of the emergency receiving service. He was complaining of severe abdominal cramps, headache, thirst and chilliness, and was perspiring profusely. He was incontinent of brownish but nonbloody diarrheal stool. Physical development and nourishment appeared excellent. The blood pressure was 160/80, the pulse 100 and regular, the respirations 20, and the temperature 100.4°F. Skin color and turgor were normal. The pupils were round, regular and equal, and reacted to light and in accommodation. The optic fundi appeared normal. The patient had transient difficulty in enunciating words. The neck was supple, and the lungs were clear to auscultation. The heart was not enlarged, there were no murmurs, and the quality of the sounds was good. The abdomen was rigid.

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REPOSITORY DDE-FORRESTAL

COLLECTION MARKEY FILES

BOX No. 30F6

FOLDER CRITICALITY MACHINES  
SOURCE ACCIDENTS

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TABLE 1. Summary of the Therapeutic Agents Employed in the Symptomatic Treatment of the Patient.

COMPOUND	DOSE	DOSES ADMINISTERED	RATIONALE	EFFICACY
Diphenhydramine	100.0 mg.	2	Intestinal hypermobility & vomiting	Good
Morphine	12.0 mg.	8	Pain & irritability	Good
Chloramphenicol	3.0 gm.	4	Antibiotic prophylaxis (?)	? Effectiveness
Neomycin	1.0 gm.	11	Stabilization of intestinal tract	? Effectiveness
Methylprednisolone	40.0 mg.	6	Adrenocortical failure (?)	Some value
Dimenhydrinate	50.0 mg.	2	Intestinal hypermobility & vomiting	Good
Levarterenol	8.0-12.0 mg.	9	Hypotension	Helpful
Metaraminol	200.0 mg.	1	Hypotension	No value
Mannitol	12.5 gm.	2	Osmotic diuretic	No effect
Hypertensinogen	3.0 micro-gm.	1	Hypotension	Unknown (patient died)

No masses were palpable. Muscle tone was normal, and peripheral pulses were full. There was no edema. The reflexes were intact.

After a brief initial examination 100 mg. of diphenhydramine was given intramuscularly, and 12 mg. of morphine subcutaneously. Because of continued restlessness 15 mg. of morphine was administered again 1 hour after the initial dose, and a good effect was obtained. A review of medications appears in Table 1. Blood samples were immediately drawn for routine studies and for chemical and radiation analyses.

A cannula was placed in a vein in the left ankle, and plasma was started. Because of vomiting and abdominal distress a Levin tube was inserted, but aspiration revealed only gastric juice without blood. A Foley catheter was then passed to monitor accurately the urinary output. Neomycin, 1 gm., was given every 4 hours through the Levin tube, and 2 gm. of chloramphenicol was begun intravenously with dextrose and saline solution in an effort to prevent infection if the patient survived long enough for effects of bone-marrow deprivation to become apparent. By 2 hours after admission the blood pressure had fallen to 130/60, and the pulse had risen to 90. Vomiting and diarrhea had ceased.

By 4 hours the blood pressure had dropped to 85/40, and the pulse had risen to 110. Methylprednisolone (40 mg.) was added to the intravenous infusion, and the systolic blood pressure rose to 100. Levarterenol, 8 mg., was begun at this time along with methylprednisolone intravenously to maintain the systolic blood pressure between 95 and 100. By 9 to 10 hours after admission the patient reported that he felt well. There was no other evidence of adrenal failure. The temperature had risen to 102°F. The left hand and forearm, which had held the container, and were nearest the reaction, became edematous and red. Conjunctivitis and periorbital edema appeared on the left. He was alert and co-operative, and spent the time reading and talking. Visual acuity for newsprint seemed normal.

X-ray study of the chest approximately 16 hours after the accident suggested some hilar congestion (Fig. 1), but the lungs remained free of rales, and he was not dyspneic. The hand and forearm became painful, and pain from venospasm of the leg caused by the infusion of levarterenol became severe and continued to be a troublesome symptom. The flow of urine diminished.

The patient was quite comfortable but slightly restless on the morning after exposure. The edema of the hand and forearm was increasing, and the fingers were moved with difficulty (Fig. 2). The neck veins were not distended. The respirations were 24 to 28. The blood pressure was maintained with norepinephrine, and this constituted a major therapeutic problem throughout the remainder of the course. A trial of metaraminol failed, as did hypertensinogen, but the latter was only used preterminally.

The condition of the patient had deteriorated by the morning of the 2d day. He was restless, fatigued and apprehensive and had become more dyspneic. The left hand



FIGURE 1. X-Ray Film of the Chest Taken with a Portable Apparatus Sixteen Hours after the Accident.

and forearm were badly swollen and livid, and there was massive edema of the upper arm. The conjunctivitis and blush of the left side of the face had increased. The lungs were free of rales. Vision had diminished to a point where he was unable to read 1-inch type, but he could still distinguish faces.

From this point the blood pressure could be maintained only with increasing difficulty. The heart increased in rate, and the sounds became tic-tac in quality. Six hours before death the patient became extremely restless and disoriented, the urinary output ceased, and the blood pressure could no longer be obtained. He died 49 hours after the accident.

A detailed account of the pathological findings on post-mortem examination will be published separately. The following summary was prepared by Dr. Herbert Fanger, pathologist to the Rhode Island Hospital:

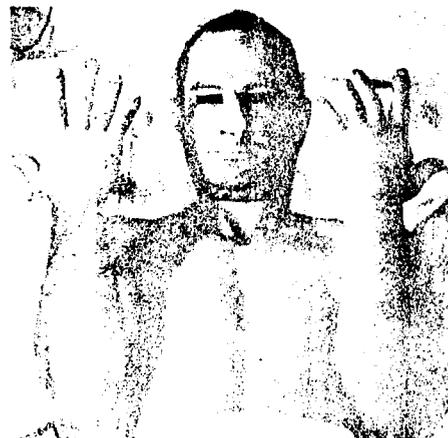


FIGURE 2. Appearance of the Patient Approximately 24 Hours after the Accident, Showing the Edema of the Left Hand and Forearm.

TABLE 2. Changes in Serum Constituents after Acute Gamma-Neutron Exposure.

INTERVAL AFTER EXPOSURE	BLOOD UREA NITROGEN	CREATININE	URIC ACID	SODIUM	POTASSIUM	CARBON DIOXIDE	CHLORIDE
hr.	mg./100 ml.	mg./100 ml.	mg./100 ml.	milliequiv./liter	milliequiv./liter	milliequiv./liter	milliequiv./liter
	Normal value 0-17	1.0-2.0	3.5-5.0	135-145	3.5-5.0	26-28	100-106
3	18	1.3	9.3	140	3.7	21	104
15	37	2.0	16.9	148	4.3	17	121
27	45	—	16.4	—	—	—	—
44	59	2.7	15.5	136	4.7	13	105

There was interstitial edema of the subcutaneous tissues of the left forearm, hand, anterior abdominal wall and chest wall. The trachea and esophagus were acutely inflamed. There was bilateral hydrothorax and slight hydropericardium and ascites. The lungs showed interstitial and intra-alveolar edema, and there was subserosal edema of the stomach and intestines and severe submucosal edema of the transverse and descending colon.

Acute pericarditis involved chiefly the anterior right atrium and the adjacent upper right ventricle. Interstitial myocarditis and periaortitis of the ascending aorta were also present.

The liver showed passive congestion and focal fatty metamorphosis, and there was acute pancreatitis, with patchy necrosis of the acini and infiltration by polymorphonuclear neutrophils.

The spleen, lymph nodes, thymus and intestinal tract were depleted of lymphocytes, but the reticulum cells of the follicles were retained. The bone marrow was aplastic. Only a few cells of the hematopoietic system remained.

The brain showed minimal changes. Rare foci of microglial proliferation were found, and a few oligodendroglia were swollen.

There was interstitial edema in the testes and rare necrosis of spermatogonia.

A radioautograph of the skin of the left hand disclosed the presence of radiation-emitting substance in the keratin layer suggestive of spray contamination.

The principal blood chemical measurements appear in Table 2. There was a gradual rise in blood urea nitrogen and creatinine concentrations. The uric acid concentration was elevated within 3 hours of exposure, at a time when the blood urea nitrogen concentration was normal, and remained at a high level throughout. Plasma electrolytes were normal except for a single elevation of chloride. The carbon dioxide content was reduced, but it is not known whether this was due to respiratory alkalosis or metabolic acidosis.

There was minimal evidence of liver damage. The serum glutamic oxalacetic transaminase activity rose from a normal value of 13 units at 2 hours to a borderline value of 40 units at 15 hours and a final value of 50 units at 44 hours. Serum bilirubin concentrations, thymol turbidity and alkaline phosphatase were within normal limits. Prothrombin activity was 50 per cent of normal at 15 hours and 47 per cent at 44 hours. No abnormality of the serum proteins was detected.

The hematologic findings in the peripheral blood are listed in Table 3. There was a moderate trend toward hemoconcentration as the illness progressed. The total white-cell count of the peripheral blood was elevated at the initial

examination at 2 hours, and increased to 46,000 cells per cubic millimeter at 38 hours. Lymphocytes constituted 5 per cent of the white cells at 2 hours and 1 per cent at 15 hours, and were not seen in subsequent blood smears. These findings are shown graphically in Figure 3. There were no noteworthy changes in the platelets.

The bone marrow was examined twice. The 1st aspiration was from the sternum at 4 hours, and the 2d from the ilium at 40 hours. The results appear in Table 4. The findings in the 1st aspiration were virtually normal, and a normal complement of lymphocytes was seen. There seemed to be a tendency to a shift toward maturity of the erythroid elements, and a few unidentified cells with irregular pink-stained granules and pyknotic nuclei were found. The specimen at 40 hours was entirely different. The aspirate was fluid. Almost all the cells were mature granulocytes with "toxic" granules, and some of these were in various stages of disruption. The megakaryocytes had disappeared.

Three electrocardiograms were obtained. These appear in Figure 4. The 1st, taken at 22 hours, was within normal limits. The axis was +30. There was a suggestion of peaking of the P waves in the diaphragmatic leads. The 2d, taken at 39 hours, showed persistently prominent P waves, diminished QRS voltage, lowered T waves and a shift in the QRS axis toward the right to +55°. There was slightly more clockwise rotation in the precordial leads. The final tracing, taken at 44 hours, showed still further diminution in the QRS voltage and lowering of the T waves. The changes seemed consistent with some degree of progressive acute strain of the right side of the heart and correlated with the dyspnea and pulmonary congestion and edema exhibited by the patient. Since there was no significant pericardial collection at autopsy the lowered QRS voltage may have been due to acute hyperinjection of the lungs produced by the pulmonary congestion. It is not clear how much of this may be attributed to radiation-induced changes in the pulmonary circulation, radiation damage to the heart and circulatory overload from intravenous therapy. The last was probably not a prominent factor since most of the fluid intake was accounted for by output plus a reasonable allowance for insensible water loss, and there was also hemoconcentration rather than hemodilution. The fluid balance appears in Table 5.

#### RADIATION CONSIDERATIONS

Shortly after admission to the hospital a survey of the body surface was made for gamma emission. The reading at 2 feet from the face and upper chest

TABLE 3. Changes in Hematologic Findings after Acute Gamma-Neutron Exposure.

INTERVAL AFTER EXPOSURE	HEMOGLOBIN	HEMATOCRIT	WHITE-CELL COUNT	DIFFERENTIAL COUNT			PLATELETS	TOTAL EOSINOPHIL COUNT	RETICULOLOCYTE COUNT
				NEUTROPHILS	LYMPHOCYTES	MONOCYTES			
hr.	gm./100 ml.	%		%	%	%	%	%	
2	15.5	43.5	17,750	92	5	—	168,000		
15	12.9	37.0	20,150	95	1	4	155,000		
21	13.7	38.5	28,800	100	0	0	175,000	5	
27	16.3	47.0	36,000	100	0	0	220,000	0.4	
38	17.5	51.5	46,000	100	0	0	170,000	0.3	

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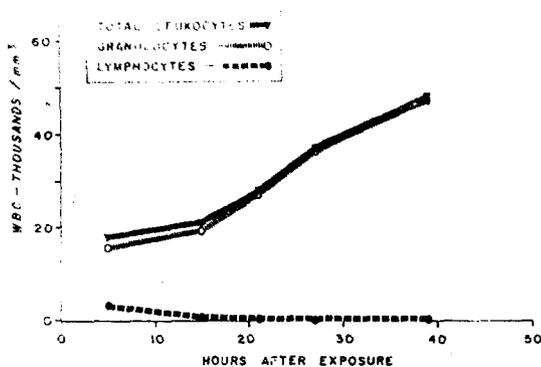


FIGURE 3. Leukocyte Changes in the Peripheral Blood after Exposure.

was 40 milliroentgens per hour, 18 milliroentgens per hour at the same distance above the mid-portion of the body, and 10 milliroentgens per hour above the feet. Shortly thereafter the patient was given a thorough bath on a plastic sheet, and this materially reduced the readings. Measurements were repeated immediately after death and disclosed the following, in milliroentgens per hour: head, 7.5; face, 3.5; chest, 2.5; abdomen, 5.0; right arm, 2.5; pubis, 4.0; thigh, 2.5; and feet, 1.1. Thus, at the time of death an attendant could have worked within 2 feet of the patient's head for nine hours to have received the maximum allowable radiation exposure for one week. This estimation, of course, does not take account of possible crosscontamination from alpha-emitting and beta-emitting isotopes on the skin and hair or in the excreta, which was clearly the significant hazard.

Samples of blood, urine, feces and hair and a gold ring were studied in the Physics Research and Thyroid Research Laboratories of the Massachusetts General Hospital. All samples were measured with an NaI(Tl) well detector and a 512-channel gamma-

ray spectrometer. The spectrum from the blood, urine, feces and vomitus corresponded exactly to Na<sup>24</sup> in that the primary peak was at 1.37 MeV, and conformed exactly to an authentic sample of Na<sup>24</sup>. The spectrum from the hair was much more complex and indicated contamination with several fission products. The gold ring yielded a spectrum characteristic of Au<sup>198</sup> and no evidence of other activation products.

A comparison of the Na<sup>24</sup> content of the plasma sample at sixteen and a half hours and authentic Na<sup>24</sup> standards was made by integration of the total counts in the 120 channels that included both the gamma peaks. Background was subtracted, and sufficient counts accumulated to give 1 per cent counting statistics. The result was extrapolated to the time of the accident and indicated a plasma assay of 0.0264 microcurie per milliliter. A plasma sample drawn at seven hours was analyzed similarly and showed good agreement.

Precise calculation of the radiation dose received by the patient would depend on exact knowledge of the geometry of the accident and of the neutron and especially the gamma-ray spectrum resulting from the excursion. Also, one would need to know the ratio of gamma rays to neutrons. If one assumes that this accident was similar to the Y-12 accident at Los Alamos, it can be estimated from the Na<sup>24</sup> content of the plasma that the patient received a neutron dose of approximately 2200 rads and a gamma-ray dose of 6600 rads, or a total whole-body dose of 8800 rads. This is probably between ten and twenty times the average lethal dose.

Specimens of urine, feces and vomitus were thinly dried on planchettes and counted in a high-efficiency, very-low-background gas-flow beta counter after time had elapsed for complete disappearance of the short-lived Na<sup>24</sup>. Counting efficiencies were corrected by measurement of an authentic standard of P<sup>32</sup>. Serial counts were made over a three-week period, and the resulting curves fitted by eye. The decay constants were consistent in each count with the presence only of P<sup>32</sup>. The P<sup>32</sup> concentration of the four-hour urine sample was calculated to be 0.0062 microcurie per milliliter.

TABLE 4. Bone-Marrow Differential Count.

DIFFERENTIAL	NORMAL RANGE	4 1/2 HRS. AFTER EXPOSURE	40 HRS. AFTER EXPOSURE
	%	%	%
Polymorphonuclears	16-27	7.5	91.5
Bands	16-27	3.5	3.5
Metamyelocytes	2-15	10.5	
Myelocytes	2-15	10.0	
Promyelocytes	0-5	1.5	
Basophils	0-2	0.5	
Eosinophils	0-7	2.0	
Lymphocytes	5-20	12.0	
Monocytes	1-7		
Reticulum cells	1-5		
Primitives	0-4		
Blasts	0-5		
Plasma cells	0-5	0.5	
Clasmatocytes	0-1		
Normoblasts	0-5	9.5	
Late erythroblasts	5-30	11.0	
Early erythroblasts	1-15	17.0	0.5
Proerythroblasts	0-1		

TABLE 5. Fluid Balance.

DATE	INTAKE ml.	OUTPUT ml.
7/24/64 (8 p.m.) to 7/26/64 (8 a.m.)	2000 (clear) 1770 (plasma)	1335 (urine) 2015 (Levin tube & vomitus)
	4575	3450
7/26/64 (8 a.m.) to 7/26/64 (7 p.m.)	2000 (clear) 500 (plasma)	urine (at 7 hr.) (Levin tube) (vomit)
	2500	200
Totals	7075	4190

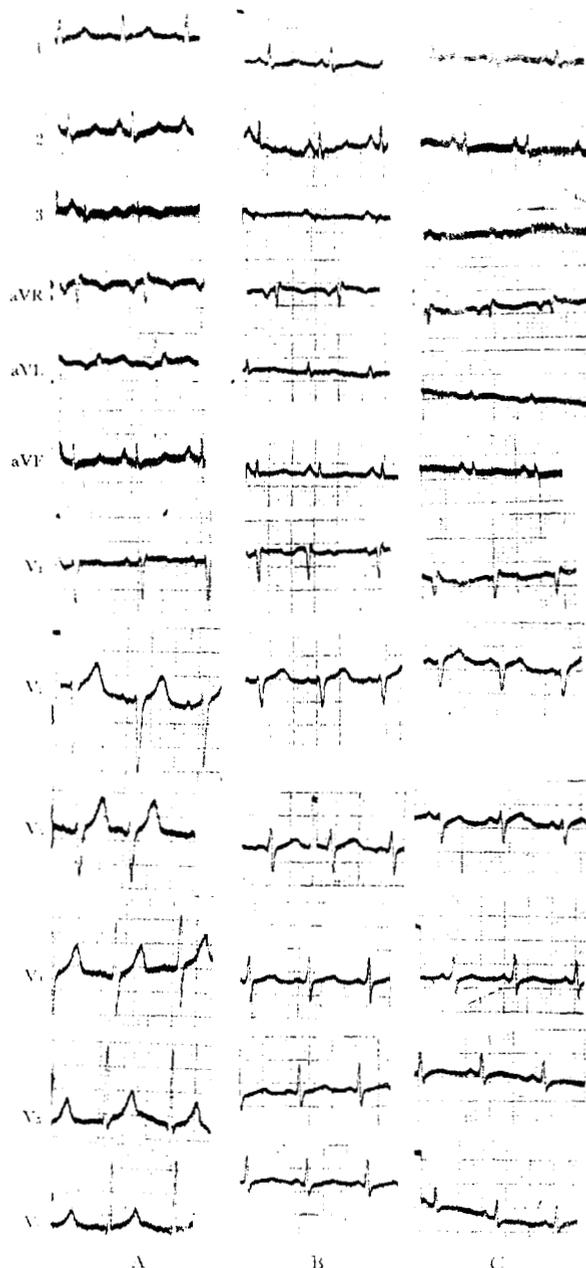


FIGURE 1. Serial Electrocardiograms Taken Twenty-two Hours (A), Thirty-nine Hours (B), and Forty-four Hours (C) after the Accident.

#### THE MEDICAL PROBLEM

It is apparent from the estimation of the dose of radiation received by this patient that no known form of therapy would have been effective in saving his life. The central problem was failure of the circulation. Not only was there failure to maintain an adequate blood pressure, and unsustained response to vasopressor agents, but also there was evidence of dilatation of the right side of the heart and venous

congestion. This aspect of the acute radiation syndrome has been encountered before and seems to have received little consideration.<sup>9</sup> It might be suggested that circulatory failure is due to vasoactive peptides released as a result of radiation-induced cell destruction. At all events cell destruction was indicated by the findings in the bone marrow and the rise in uric acid and transaminase in the blood. The possibility that the shock was neurogenic in origin cannot be excluded.

There have been virtually no studies on the acute radiation syndrome in man except for the exhaustive observations on the victims of the Los Alamos,<sup>2</sup> Vinco<sup>4,6</sup> and Oak Ridge accidents.<sup>8,10,11</sup> Countless studies have been made on the effects of radiation exposure of laboratory animals. The lethal radiation syndrome has been somewhat arbitrarily divided into the neurologic, gastrointestinal and hematologic forms. Beginning almost immediately after exposure, the patient may be disoriented, ataxic and delirious. This state usually clears within a few hours or days. Delirium and mania may occupy the last few hours or days before death. The shocklike state that may be seen immediately after exposure and may persist until death may have a neurogenic basis, but this is not clear. Initially, the gastrointestinal findings range from nausea to vomiting, cramps and explosive diarrhea, the last being an ominous sign. Gastrointestinal bleeding has not been prominent. If these symptoms clear abdominal discomfort and anorexia may take their place and persist for weeks. Ulcerations of the oral mucosa may be troublesome. As in the case reported above, the lymphocytes disappear from the blood of the heavily irradiated patient within a few hours, and the bone marrow becomes aplastic within a day or two. The neutrophil count in the peripheral blood may rise to 15,000 to 50,000 during the first two days, and then falls to very low levels. Hematocrit and platelets usually begin to fall only after ten days to two weeks.

Failing vision, restlessness and delirium in our patient suggested neurologic damage but could have been due in part to other circumstances. There were gastrointestinal symptoms but no authenticated evidence of bleeding. The lymphocytes disappeared from the blood, and the bone marrow was virtually destroyed, but the patient succumbed before bone-marrow failure had much reflection in the red cells or granulocytes of the peripheral blood. Thus, whereas the patient shared in the manifestations of the acute radiation syndrome as reported by others, the principal features were in the cardiovascular system. The initial treatment with antibiotics was probably unnecessary.

#### RADIATION SAFETY

Management of the victim of a nuclear accident imposes a formidable exercise in radiation safety.

This patient had heavy surface contamination of radiation-emitting isotopes. Furthermore, vomitus, sweat, urine and feces were all contaminated with radioactive sodium and  $P^{32}$ . Thus, there was the problem of radioexposure to the personnel caring for the patient, as well as the disposal of contaminated clothing, linen and radioactive excreta. Finally, there were the additional problems of significant skin contamination during intimate care imposed by a patient who was for a time delirious and unmanageable, as the patients often are.

Immediately upon arrival at the hospital this patient was sequestered in a section of the Emergency Ward to which access could be controlled. Admission to the area was denied to all except those directly involved in his care and in the management of radiation safety. A nursing station was established outside the door of his room, as well as an area for gowning and degowning for all those attending him, and a disposal area for monitoring and collecting all vomitus and excreta. All contaminated articles were disposed of either to the laboratory or into large plastic bags for subsequent disposal by radiodecay or through routine channels for radioactive waste.

Those attending the patient were required to wear caps, masks, gowns, plastic gloves and large paper "grocery" sacks tied over the feet. They discarded these into plastic bags upon leaving the area. Radiation safety was supervised by Mr. Ernest Resner, of the New York Operations Office of the Atomic Energy Commission, who arrived a few hours after his office was notified of the accident. The Atomic Energy Commission medical consultant arrived a few hours later. It is worth noting that consultants from the Commission are available at any time in the event of a nuclear accident.

#### OTHER EXPOSED PERSONNEL

Four persons were in the plant at the time of the excursion, but all were on a different floor level and 30 or more feet removed from the accident. The plant manager entered the plant thirty minutes after the accident with the shift supervisor and spent some time assaying the radiation-contamination levels and draining off the tank containing the residual uranium into critically safe containers. They and another employee were found to have been exposed to 30 to 50 rads of whole-body radiation when their film badges were examined on the following day. Apart from some apprehension they were free of symptoms and have remained so.

Several days later the plant manager, the shift supervisor and the third staff member who had appreciable exposure were measured in the whole-body radiation detector at the Massachusetts Institute of Technology by Dr. Constantine Maletsios. There was minimal but detectable  $Na^{24}$  in all 3. The

gold ring of 1 of them also had  $Au^{198}$  contamination. Ten-milliliter samples of urine from the plant manager and shift supervisor were concentrated and dried on planchettes and measured for beta activity for twenty-five days. The counting rates were low. The samples were corrected for detector sensitivity against an authentic  $P^{32}$  sample. The half-life of decay was forty-six days for 1 sample and fifty-five days for the other, but the logarithmic curves may not have been linear. These findings are taken to mean inhalation or ingestion of fission products while they were draining the tank in the heavily contaminated room where the accident occurred.

These 3 exposed persons have been kept under close medical supervision since the accident. No overt evidence of radiation damage has appeared. Hemoglobin and total and differential white-cell counts and platelets have been normal. The plant manager, forty-three years of age, had a serum uric acid concentration of 7.6 mg. per 100 ml. three days after the accident. It was 9.2 mg. two days later, 7.3 mg. on the following day and 6.4 mg. per 100 ml. on the next day. The plasma bilirubin was 1.1 mg. per 100 ml. on initial determination, but subsequent values were normal. Alkaline phosphatase and transaminase assays were normal. The shift supervisor, thirty years of age, had a serum uric acid concentration on the third day after the accident of 8.1 mg., but subsequent values were below 5.5 mg. per 100 ml. These values are highly suggestive of radiation damage, but are of uncertain significance because the pre-exposure serum uric acid concentrations are not known. The serum bilirubin concentration was 1.7 mg. on the third day, 1.6 mg. on the fifth and 0.7 mg. per 100 ml. thereafter. Other tests of liver function were within normal limits.

#### ADMINISTRATIVE FEATURES

A serious nuclear accident generates enormous local interest and administrative problems not only because of the drama of the event but also because of the possibility of serious contamination by radiation, legal implications and special difficulties in management of this kind of patient. Fortunately, the accidents that have occurred to date have involved only 1 or a very few victims. One shudders to imagine the difficulties generated by an accident involving major exposure of several or more persons.

We were struck by the importance of establishing tight medical and administrative control at the earliest possible moment. One physician should be in full and acknowledged command of the medical care of the patient. Matters of radiation safety should be entirely in charge of the hospital radiation-safety officer (and every hospital within reach of a nuclear installation should have such a person on its staff). In addition, there should be an officer of the administration stationed at or near the point of entry to

the area where he can govern access, handle procurement, inform relatives and the press and take care of the countless administrative details inevitably accompanying such an occurrence. A serious problem contributing to the confusion is the tendency of professional and other personnel to remain in the area long after their authentic duties have been discharged. The administrative officer should make it his business to keep the area free of all except those engaged in their legitimate duties. Also, ideally he would be in a position to handle and fend off as many telephone calls and other interruptions of the attending staff as possible. If these simple but necessary procedures are followed danger of contamination will be minimized, and care of the patient enhanced.

**SUMMARY AND CONCLUSIONS**

The rapid growth of the industrial use of nuclear energy imposes an inevitable risk of radiation accidents. The first fatal nuclear excursion in private industry is described here. The victim succumbed forty-nine hours after receiving between ten and twenty times the lethal dose of neutrons and gamma rays. The principal clinical problem was that of maintenance of the blood pressure and the competence of the heart. The myriad medical and administrative problems that arose are discussed.

We are indebted to Drs. Gordon Brownell and Roger Rydin, of the Physics Research Laboratory of the Massachusetts General Hospital, who carried out the gamma-ray spectroscopy and dose estimation, to Dr. M. Alcala, of the Rhode Island Hospital, who performed the bone-marrow and other hematologic studies, to Drs. Thomas Forsythe and Stephen Frazer and the house staff and nursing staff of the Rhode Island Hospital for their skillful and devoted help in the care of the patient and to Dr. Roman W. DeSanctis, who interpreted the electrocardiograms independently.

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**REFRACTORY ANEMIA WITH ABNORMAL IRON METABOLISM\***

**Its Remission after Resection of Hyperplastic Mediastinal Lymph Nodes**

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**M**EDIASTINAL-lymph-node hyperplasia resembling thymoma was first recognized as an entity by Castleman in 1954,<sup>1</sup> although 3 cases of "enlarged inflammatory lymph nodes" had previously

been described in 1946,<sup>2</sup> in a series of 109 patients operated on for mediastinal tumors. Subsequently, Castleman, Iverson and Pardo Menendez<sup>3</sup> presented a series of 13 cases, including 2 that had previously been classified as thymoma.<sup>4,5</sup> The characteristic pathological features noted were germinal-center formation and marked capillary endothelial proliferation. These findings were consistent with hyperplasia and not neoplasia. Many similar cases have been reported.<sup>6-16</sup>

In none of the approximately 30 cases in the world literature was there an associated anemia. It is the purpose of this report to present a case of mediastinal-lymph-node hyperplasia associated with severe iron-loading anemia, fever, marked hyperglobulinemia, splenomegaly and bone-marrow plasmacytosis. Remission of all these abnormalities followed excision of the mediastinal mass. The patient was investigated intensively in an attempt to correlate the pathological findings with the clinical course.

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