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BETA RAY BURNS OF HUMAN SKIN

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The development of the atomic energy program has resulted in a large increase in the number of persons exposed to radiologic hazards. With this increase it is essential that physicians and other scientists be acquainted with the dangers accompanying the handling of radioactive materials. Four cases of beta ray burns of the hands are reported here for the purpose of supplementing the present information in the existing literature,[1] and emphasizing the early clinical course, as well as the laboratory observations in such cases. It is hoped that, by calling attention to the early signs and symptoms of injury by beta radiation, overexposure will be recognized and hazardous operations discontinued before more damage is incurred.

All 4 men were injured while undertaking procedures of a like nature at the recent atomic tests at Eniwetok Atoll (United States Atomic Energy Commission's proving grounds). This work involved the handling of radioactive materials which emitted large amounts of beta and gamma radiation. A deviation in the prescribed procedure undertaken to expedite the operation resulted in the handling of these radioactive substances directly with rubber-gloved and sometimes with bare hands rather than with the remote handling devices that had been provided. Measurement of the dosage of beta rays received by the patients was not obtained at the time of exposure, but film badge and pocket ionization chambers worn by the men over the thoracic region showed readings for gamma rays varying from 1 to about 15 r. In view of the fact that the hands were much closer to the active material, it must be presumed that the amount of gamma radiation received by the hands was somewhat larger, probably by a factor of 10.

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From the Los Alamos Scientific Laboratory (Drs. Knowlton, Leifer, Hogness and Hempelman) and the Los Alamos Hospital (Drs. Blaney, Gill, Oakes and Shafer).

1. (a) Robbins, L. L.; Aub, J. C.; Cope, O.; Cogan, D. G.; Langohr, J. L.; Cloud, R. W., and Merrill, O. E.: Superficial Burns of Skin and Eyes from Scattered Cathode Rays, Radiology **46**: 1-23 (Jan.) 1946. (b) Raper, J. R.: Plutonium Project: Effects of Total Surface Beta Irradiation, ibid. **40**: 314-324 (Sept.) 1947. Low-Beer, B. V. A.: External Therapeutic Use of Radioactive Phosphorus. I. Erythema Studies, ibid. **47**: 213-222 (Sept.) 1946. Wilhelmy, E.: Ueber die Reaktion der Haut auf langwierige Röntgenstrahlen und Kathodenstrahlen, Strahlentherapie **55**: 498-523, 1936.

The major portion of the radiation consisted of beta rays with an average maximum energy of about 1 Mev. (million electron-volts). The ratio of beta counts to gamma counts in a glass-walled Geiger-Mueller type tube was about 6 to 1. The physical properties of beta rays of 1 Mev. maximum energy are such that approximately 90 per cent of the incident rays are absorbed by the first 3 mm. of tissue and 99 per cent are absorbed in 6 mm. of tissue.[2] Estimates of dosage indicated that these 4 men received from 3,000 to 16,000 rep (roentgen equivalent physical) to the outer surface of the skin.[3] The beta ray dosages below the surface of the skin would then be 300 to 1,600 rep at 3 mm. and 30 to 160 rep at a depth of 6 mm. In view of the fact that there was definite evidence that the whole body exposure did not exceed 15 r of gamma rays in any case, it was believed that there was minimal, if any, danger of important whole body effect, and the patients were treated primarily for local beta ray burns of the hands. This assumption was borne out in the course of the disease, in that at no time was there any significant evidence of other than local damage.

Since the amounts of radiation received and the clinical courses of the patients differ to some extent, each case is reported separately. Following is a brief synopsis of the clinical course of each patient. Prior to this episode these men had worked with radioactive materials but had had no overexposure to radiation and were all in good health.

REPORT OF CASES

CASE 1.—A white man aged 26 received 10 r of gamma rays to his whole body and about 5,000 to 10,000 rep of beta rays to his hands over a period of about sixty minutes. At the time of exposure he noted a tingling and itching of his palms, which he assumed to be due to excessive perspiration. Approximately two hours later he noted a stiffness of the fingers and a slight swelling of the hands. He reported to a physician thirty-four hours after exposure.[4] At this time there was slight swelling, erythema and tenderness of the second and third fingers of the right hand. On the left hand the volar aspect of all digits and the distal portion of the palm showed mild erythema and swelling. There was a blanched area over the proximal phalanx of the second finger of the left hand.

During the next twenty-four hours there was a definite decrease in the erythema and swelling of both hands. Beginning vesiculation was noted in the blanched area of the index finger of the left hand, and three days after exposure a definite vesicle with a surrounding area of erythema had formed.

On the fourth day the center of this vesicle was blackish blue. There was erythema of the volar surface of the second, third and fourth fingers of the left hand and slight swelling

2. Lapp, R. E., and Andrews, H. L.: Nuclear Radiation Physics, New York, Prentice-Hall, Inc., 1948, p. 178.

3. Joseph G. Hoffman of the Roswell Park Memorial Hospital, Buffalo, made the dosage calculations.

4. In cases 1, 2 and 3 the patients left Eniwetok immediately after exposure and were flown directly to Los Alamos, where they were seen by physicians on arrival. In case 4 the patient was not known to have been injured until the development of symptoms.

without erythema of the index finger and thumb of the right hand.

On the seventh day there were blanched and tender areas along the thenar aspect of the index finger and on the pad of the distal phalanx of the thumb of the right hand. By the eighth day these areas had become vesicular. From the eighth to the twenty-fifth day there was a continuous extension of

Fig. 1 (case 1).—Eleven days after exposure early vesicles were visible on the second fingers of both hands. (This photograph and those in subsequent figures were prepared by Elmer Donor of the Los Alamos Scientific Laboratory.)

the erythema of both hands, with vesicles forming two to three days after the onset of the reddening (figs. 1 and 2). By the twenty-fifth day, the erythema had disappeared and bulla formation had reached its maximum. At this time there was one large confluent bulla covering all four fingers and the distal portion of the palm of the left hand; a comparable bulla covered the right hand, including the fingers and palm.

Since the twenty-fifth day after exposure the clinical course has been one of constant slow improvement. By the twenty-eighth day drying of the bullae of both hands was noticeable, and on the thirty-second day most of these areas were dry.

On the thirty-ninth day the hands were surgically débrided with the patient under general anesthesia. At this time there was epithelization of both hands except in one area over the proximal phalanx of the second finger of the left hand, which appeared white and necrotic, and other smaller areas over the second, third and fourth fingers of both hands.

A second débridement was done on the fifty-third day, and at this time the only unepithelized areas present were over the second and third fingers of the left hand and a small area over the proximal phalanx of the second finger of the right hand. Further surgical débridements were carried out on the sixty-seventh and eighty-second days (fig. 3). Unhealed areas remained on the second and third fingers of the left hand and on the proximal phalanx of the second finger of the right hand.

Five months after exposure these lesions were still unhealed (fig. 4). The dimensions of all three lesions were about 1 by 2 cm., and there were white necrotic granulations in the center of each which bled easily on slightest trauma. The areas were surrounded by invaginating folds of thin, pink epithelium. There was minimal healing during the third, fourth and fifth months of the disease. Skin grafting of these lesions was performed during the seventh month with good results.[5] Skin grafting has also been carried out in an effort to replace the decidedly atrophic epithelium present in other injured areas, and further operative measures of this nature are anticipated. At the end of one year severe atrophy of the soft tissues underlying the sites of injury remains. There is moderate ankylosis of the phalangeal joints of the three most severely involved fingers.

Case 2.—A white man aged 27 received about 15 r of gamma rays to his thoracic region and an estimated 8,000 to 16,000 rep of beta rays to his hands over a sixty minute period. During this exposure he experienced a sensation of itchiness in both hands. Four hours later he noted a swelling of his left hand. He reported to physicians thirty-four hours after exposure, and at this time all the digits of the left hand were swollen and erythematous, the second and third fingers being most severely involved. There were no blanched areas on the left hand, and the right hand appeared perfectly normal.

During the third and fourth days, there was a definite decrease in erythema of the left hand and there were no symptoms referable to the right. From the seventh to the eleventh day erythema and tenderness developed over all four fingers and the distal portion of the palm of the left hand, and on the eleventh day tenderness and erythema was noted on the right index finger.

On the twelfth day vesicles began to develop in the erythematous areas. Up to the twenty-eighth day there was spreading of the erythema, with bullae eventually forming in all such

Fig. 2 (case 1).—Eighteen days after exposure there were large, tense, confluent bullae which followed the secondary erythema.

areas. By the twenty-eighth day the bullae covered the volar surfaces of all the fingers and the distal portions of the palms of both hands.

At about this time the erythema subsided. The bullae dried up by the thirty-fifth day (fig. 5), and part of the dead epidermis sloughed spontaneously. Surgical débridement on the thirty-ninth day disclosed a layer of thin epithelium covering most

5. The plastic surgery was performed on this patient and other patients by Dr. J. Barrett Brown of Barnes Hospital, St. Louis.

of the burned areas (fig. 6). When a second débridement was performed, on the fifty-third day, a small unhealed area remained on the second finger of the right hand and on the second and third fingers of the left hand. On the sixty-seventh day a third débridement was performed, and at this time both hands were entirely epithelized. The new epithelium was thin and had a tight, shiny appearance, as can be seen from figure 7

Fig. 3 (case 1).—Eighty-two days after exposure there was an ulcer over the first phalanx of the second finger with a covering of slightly vascular, fibrotic granulation tissue. Superficial desquamation was also evident over the other fingers and palm

Five months after exposure, there was a secondary breakdown of the epidermis over the most heavily exposed areas with cracking and drainage of serous fluid. Skin grafting of the palmar surfaces of both second fingers was performed during the sixth month with good results. By the twelfth month skin had been grafted onto the volar aspects of all fingers and the distal half of the palm of the left hand to replace injured, atrophic epithelium. By this time there was some loss of mobility of the digits of the left hand (despite efforts of active and passive manipulation during the course of disease) and considerable loss of underlying soft tissue.

CASE 3.—A white man aged 26 received 4.5 r of gamma rays to his whole body and about 5,000 to 10,000 rep of beta rays to the left hand in about a forty minute period. He first noted symptoms of swelling and stiffening of his left hand about six hours later. When he was examined forty hours after exposure there was a mild erythema and edema of the left hand. The right hand was free of signs and symptoms and remained so during the entire course of the disease. The generalized erythema and edema of the left hand decreased up to about the third day, at which time a secondary erythema appeared over the volar aspect of the index finger.

On the fourth day there were circumscribed dark red areas over the volar aspect of each phalanx of the index finger and the middle and distal phalanges of the third finger. From the seventh to the fourteenth day these five areas acquired a dark bluish color and became vesicular in character. By the fifteenth day these vesicles began to coalesce, and a new vesicle appeared on the proximal phalanx of the third finger. At this time the fourth and fifth fingers had become erythematous. On the seventeenth day erythema was noted over the distal heads of the second and third metacarpals. By the twenty-eighth day a confluent bulla including all four fingers and the heads of the second and third metacarpals was present. The entire reaction reached a maximum on the thirty-second day, at which time all erythematous areas, including one on the lateral margin of the distal phalanx of the thumb, had become bullous. On the thirty-fifth day the bullae had begun to dry and desquamate.

Surgical débridement was performed on the thirty-ninth, fifty-third and sixty-seventh days (fig. 8) with the patient under general anesthesia, and at each operation further healing was observed. On the sixty-seventh day whitish necrotic areas over the second and third fingers corresponding to the location of the initial bluish vesicles seen on the fourth day remained.

One hundred and sixteen days after exposure tendons were visible in both of the ulcerated areas (fig. 9). The tendon visible in the lesion on the second finger appeared to be partially necrotic. There was little, if any, healing of the lesions during the third and fourth months of the disease. By the fifth month there was definite improvement of the lesions (fig. 10), but from a functional point of view the second and third fingers of the left hand showed only slight joint mobility. During the next seven months skin was grafted first over the unhealed areas on the second and third fingers and later over the volar aspects of the remaining fingers of the injured hand to provide a more functional epithelium. Despite efforts of active and passive manipulation throughout the course of the disease, the phalangeal joints of the index finger are immobile and there is moderate limitation of flexion of the third finger. The soft tissue of both of these fingers is decidedly atrophic.

CASE 4.—A white man aged 27 received about 1 r of gamma rays to his whole body and an estimated 3,000 to 4,000 rep of beta rays to his left hand in about a sixty minute period. This patient's exposure occurred approximately two weeks prior

Fig. 4 (case 1).—One hundred and fifty-eight days after exposure there was still an unhealed ulcerated area over the first phalanx of the second finger with atrophy and flexor contraction of the digit

to that of the other 3 men. He had no symptoms until eleven days after exposure, at which time he noted redness and swelling of the skin over the volar aspect of the proximal phalanges of the second, third and fourth fingers of the left hand. The patient reported to a physician on the thirteenth day, at which time there was a bluish discoloration beneath the skin in the

aforementioned areas but no vesicle formation. This was not recognized as a radiation injury at this time, because the patient gave a history of carrying an exceedingly heavy lead container with his left hand by means of an iron cross bar. Mechanical injury was thought to be the etiologic factor. By the fifteenth day, however, vesicles had developed in all of the erythematous areas and it became apparent from the course

Fig. 5 (case 2).—Thirty-five days after exposure there was much spontaneous desquamation over the burned areas with a tanned appearance of the proximal portions of the palms.

that this was a radiation injury. The erythema had spread to the middle phalanges of the second and third fingers, and there was a bluish discoloration over the head of the second metacarpal. On the nineteenth day vesicles had formed over the middle phalanges of the second and third fingers.

By the twenty-fifth day the process had ceased to advance and bullae covered the volar surfaces of all the fingers and the head of the second metacarpal. From this time on drying progressed rapidly, and by the thirty-second day the bullae were completely dry. There was desquamation of the burned surfaces by the forty-seventh day, with epithelization over all these areas. This patient has had no signs of residual damage or tissue breakdown over a period of one year except for a slight reddening of the palmar surface of the hand. There were no signs or symptoms referable to the right hand during the course of the disease.

GENERAL CLINICAL OBSERVATIONS

The clinical course of these 4 patients may be divided into four distinct phases, comparable to those described by Robbins and others[18] and corresponding to the time intervals described by Borak[6] for effects of roentgen rays. Phase 1 consists of the initial erythema and edema with blanching of areas which received the greatest amounts of exposure. This phase begins with the initial exposure, reaches a peak in about forty-eight hours and then subsides rapidly. There follows a period of relative absence of signs and symptoms for three to five days (phase 2: third day to sixth or eighth day), at the end of which time phase 3 begins. This phase is first manifested by the development of a secondary erythema which in some cases is complicated by extravasation of blood into the erythematous areas. These reddened areas become vesicular eight to twelve days after exposure. There follows a period of about two weeks during which the erythema spreads and vesicles and bullae are formed. At the end of this time (twenty-four to thirty-two days after exposure) the active disease process becomes arrested and the bullae begin to dry and desquamate. During the last part of phase 3 epithelization occurs over all areas where the vascular supply has not been seriously damaged. Phase 4 is the chronic stage of the disease. Those areas of skin in which there has been serious interference with the vascular supply remain unhealed. In these cases skin grafting was undertaken when it became evident that epithelization would be incomplete. An atrophic epidermis, which is probably permanent in nature and in which secondary epithelial structures (such as hair and sebaceous glands) are absent, is present in the less seriously damaged areas. Secondary ulcerations such as were present in these cases, as well as the hyperkeratoses, telangiectases and malignant degenerations[7] which may occur many years after exposure, are included in phase 4.

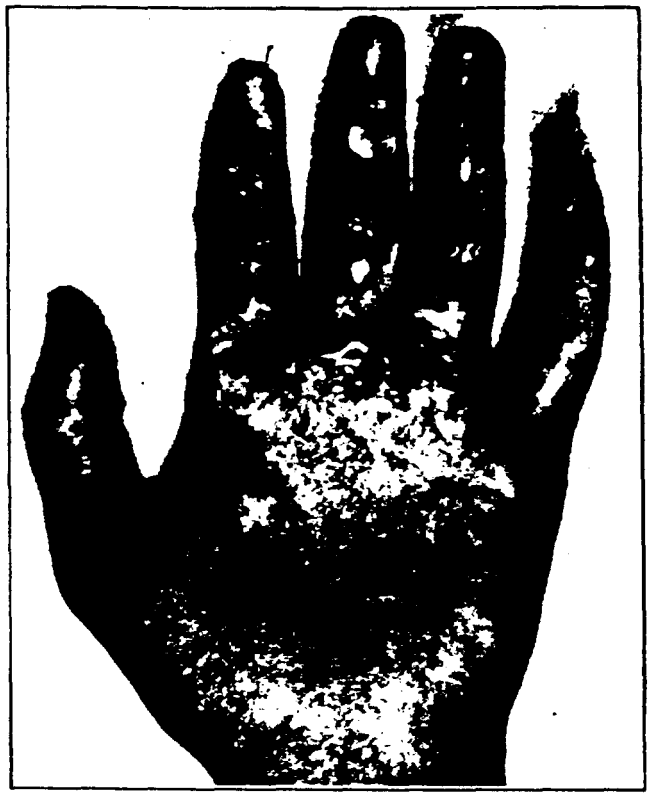

Fig. 6 (case 2).—Thirty-nine days after exposure exceedingly thin epithelium was found beneath the thick, dead skin which had been removed surgically. These areas were extremely pain sensitive.

The two men who probably received the largest amounts of radiation apparently noted a sensation of tingling and itching of the hands at the time of exposure. This suggests that intense beta radiation produces a sensory perception.

6. Borak, J.: Radiation Effects on Blood Vessels (*a*) Erythema; Edema, Radiology **38**: 481-492 (April) 1942; (*b*) Inflammation; Degeneration; Suppression of Growth Capacity; Retrogression; Necrosis, ibid. **38**: 607-617 (May) 1942; (*c*) Telangiectasis, Effects on Lymph Vessels, ibid. **38**: 718-727 (June) 1942.

7. Henshaw, P. S.; Snider, R. S., and Riley, E. F.: Aberrant Tissue Development in Rats Exposed to Beta Rays; The Late Effects of P^{32} Beta Rays, Radiology **52**: 401-415 (March) 1949.

The initial erythema and edema seen in these patients in the first forty-eight hours may be due to a local release of histamine in the skin with a resulting dilatation and increased permeability of the superficial capillaries.[6] In the more heavily exposed areas there was a definite blanching, which may have been caused by direct damage to the capillaries, resulting in a vasoconstriction phenomenon or to thromboses with occlusion of the vessels. The secondary erythema which occurred after five to seven days may have been a vascular reaction to cells which were continuing to die over a period of several weeks. It has long been recognized that many cells which have been injured by radiation can survive for a considerable period of time, but when they attempt to divide at a later date are unable to do so and die.[7]

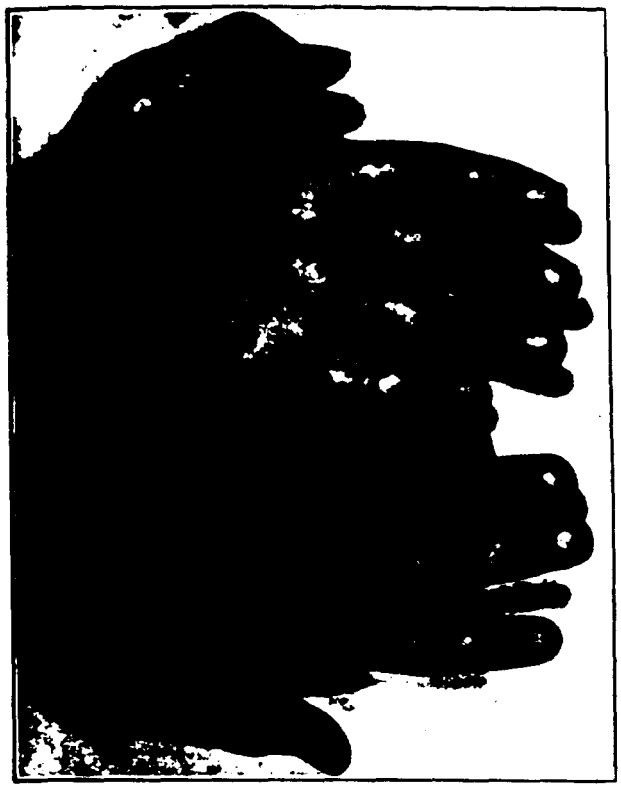

Fig. 7 (case 2).—Eighty-two days after exposure there had been complete epithelization of both hands, but the shiny, atrophic epidermis was evident especially over both index fingers. A secondary breakdown occurred in these areas at about the one hundred and sixtieth day.

The bluish discoloration of some of the erythematous areas was the result of extravasation of blood from the injured dermal plexus of capillaries. In the early stage these lesions appear indistinguishable from ordinary "blood blisters" and bruises. These hemorrhagic areas in all cases showed poor healing and in some cases nonhealing, probably as a result of the severe damage to the capillaries which must, in the process of repair, nourish the healing epidermis.

All areas of the skin which were involved in the secondary erythematous process developed vesicles. These were usually initially discrete and subsequently coalesced with the formation of large bullae, which in some cases were continuous over all five digits and most of the palm. The bullae were tense and frequently caused sufficient pain, despite generous sedation of the patient, to justify repeated drainage. Aerobic and anaerobic cultures of the vesicle fluid were negative except on one occasion, in 1 case, in which Staphylococcus albus was found, presumably a contaminant. Thus, the course was not complicated by secondary infection. The development of a cellulitis resistant to chemotherapy would have been seriously detrimental to the injured tissue. The vesicles began to dry at the end of phase 3 and were either spontaneously desquamated or removed by surgical debridement.

The new epithelium which was formed over the involved areas was in general thin and shiny, except in case 4, in which there appears to be complete healing. A blotchiness of the underlying vascular tissue, which probably represents minor vascular damage, was noted in this case.

The effects on certain secondary skin structures in these patients were definite. Nail growth was retarded in all cases to about one half the normal rate (we served as controls), and there was a slight transverse depression in the nail which was formed at the time of exposure. There was a definite loss of hair from the backs of the fingers in case 2, but this was not noted in the other 3 cases. The dorsal surfaces of the fingers in cases 1, 2 and 3 showed an apparently permanent increase in pigmentation. During the first few days after exposure, there was an excessive amount of sweating of the palms, but after about a week there was a noticeable lack of perspiration which persisted for many weeks.

Fig. 8 (case 3).—Sixty-seven days after exposure there were deep ulcers similar to that in figure 5 and superficial desquamation of the palmar and digital surfaces.

There were definite changes in the capillary loops of the nail beds.[8] In the first five days an abnormal amount of dilatation and constriction of the loops was apparent. An increased tortuosity and in some capil-

6. Lasnitzki, I.: Effect of X Rays on Cells Cultivated In Vitro: Recovery Factor, Brit. J. Radiol. **16**: 61-67 (Feb.) 1943; Response of Cells In Vitro to Variations in X Ray Dosage, ibid **16**: 137-141 (May) 1943.

7. Todd, J. C., and Sanford, A. H.: Clinical Diagnosis by Laboratory Methods, Philadelphia, W. B. Saunders Company, 1943, pp. 653-654.

laries actual thrombosis with interruption of blood flow was observed. By ten to fifteen days there was a definite decrease in the number of capillary loops, and during the third and fourth weeks some of the remaining loops had formed collateral loops into the area of destroyed capillaries. At the end of four months, a decreased

Fig. 9 (case 3).—One hundred and sixteen days after exposure a close-up view showed the extent of the ulcers during the fourth month. Small hemorrhages could be seen in the fibrotic granulation tissue, and a shiny tendon surface was present in the lesion over the first phalangeal joint of the index finger.

number of capillaries remained and the collateral loops were apparent and functioning.

The erythema and vascular lesions were confined, for the most part, to the volar surfaces of the hands. The dorsal surfaces exhibited minor desquamation indicating significant irradiation of these areas, but there was a sharp line of delineation between the volar and dorsal surfaces. Another important feature of this series was the lack of damage to the right hands in cases 3 and 4 and the lesser reaction seen in right hands in cases 1 and 2. This can be explained by the nature of the operation, which involved more contact of the radioactive material with the left hand.

Although the temperature, pulse and respiration charts are not shown, there were slight elevations of temperature and pulse corresponding in time to the development of vesicles and bullae. The respiratory rate and blood pressure remained in the normal range throughout the illness.

LABORATORY DATA

The laboratory examinations in these cases showed significant changes in the total white blood cell counts, the absolute neutrophil counts, the refractive granules in the cytoplasm of the lymphocytes[10] and the sedimentation rate. The total white blood count is the average of counts obtained from both chambers of a hemocytometer. The differential count, used for the calculation of the absolute neutrophil and lymphocyte figures, was made in wet films stained supravitally with neutral red and Janus green. Two hundred cells were examined for each differential count, one hundred by each of two technicians.[11] To determine any quantitative change in the number of refractive granules in the lymphocytes, one hundred lymphocytes were examined, fifty by each of two technicians, and the average values used. The same technicians made all the differential counts and lymphocyte studies and, although there was constantly a slightly larger number of granules counted by one of the technicians, this possible source of error is constant throughout the data. The hemoglobin values were obtained from a standard Evelyn photometric hemoglobinometer. The percentage of reticulocytes and the platelet counts were determined in a wet preparation stained with brilliant cresyl blue, and at least 1,000 erythrocytes were counted. The sedimentation rates were obtained using a standard Wintrobe tube.

The total white blood count, absolute neutrophil and lymphocyte counts, sedimentation rate and lymphocytic granules are graphically presented for case 1 in figures 11 and 12. The hematologic responses of the other

Fig. 10 (case 3).—One hundred and sixty-five days after exposure the tendon surface was still visible through a thin layer of fibrous tissue

3 patients are not shown but were similar to that seen in case 1. The preexposure blood data as plotted are taken from approximately one year of routine blood studies and are plotted without reference to the time interval between counts. The refractive granules in the lymphocytes are plotted by two methods. The percentage of lymphocytes with six or more granules

10. Dickie, A., and Hempelmann, L. H.: Morphologic Changes in the Lymphocytes of Persons Exposed to Ionizing Radiations, J. Lab. & Clin. Med. 32:1048-1059 (Sept.) 1947.

11. Technical assistance was given by Dorothy E. Poskonski and Ann C. Lorimer.

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is used, since control counts on this patient had been obtained by this method previous to exposure and also because the cases reported in the literature previously had been studied in this fashion.[10] The number of granules per lymphocyte is a less arbitrary method of reporting the observations and is now used routinely

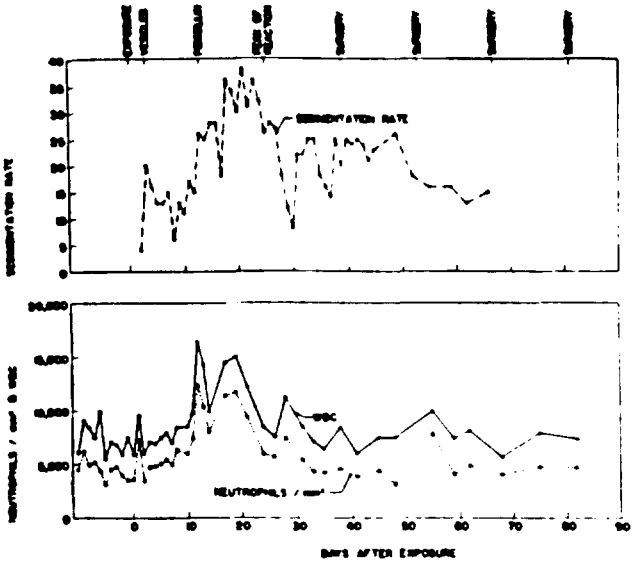

Fig. 11 (case 1).—Sedimentation rates, white blood cell counts and absolute neutrophil counts at various stages of treatment.

by this laboratory. In the preexposure portion of the curve showing the percentage of lymphocytes with six or more granules all the values have been averaged, because fewer lymphocytes were examined per count for these routine preexposure observations.

The response of the total white blood count, the neutrophil count and the sedimentation rate appeared to run parallel with the development of vesicles and bullae on the hands and the febrile response. Presumably the destruction of tissue which occurred during this time caused these hematologic changes. Thus, data on case 1 showed the severest formation of bullae and also the greatest changes in these blood tests.

There were no significant changes in the absolute lymphocyte count at any time after exposure. However, there was a definite morphologic change in these cells manifested by an increased number of refractive granules in the supravital preparations. This change was observed earlier than the increase in the neutrophils or the sedimentation rate. In case 2 the patient, who probably received more radiation than any of the others, had a lesser increase in refractive granules and a more rapid epithelization of the burned areas than occurred in cases 1 and 3. This suggests that the response of the lymphocytic granules may be related to the local disease of the hands and may possibly serve as an early indication of the seriousness of the burn, but further studies are needed to establish this point.

A rise in reticulocytes to 1 to 2 per cent in all cases is difficult to explain. These patients were at sea level for about ten days and returned to Los Alamos (elevation 7,400 feet). It appears unlikely that this increase in altitude caused a reticulocyte response, because 4 men all had lived at Los Alamos for at least one year before their short sojourn at sea level. The reticulocytosis is more likely due to an erythroid response in the bone marrow following the destruction of tissue.

The hemoglobin values and the erythrocyte and platelet counts at no time showed significant variations from normal. The prothrombin times, bleeding times and clotting times were determined frequently during the course of the illnesses and were always within normal limits. The absolute monocyte, eosinophil and basophil counts did not disclose significant variation from the normal.

Urinalyses always gave normal results, and tests for radioactivity in twenty-four hour urine specimens collected on the third day after exposure resulted negatively.

Determinations of serum proteins with albumin-globulin ratios, serum bilirubin, alkaline phosphatase, cephalin flocculation and blood urea nitrogen, as well as daily nitrogen balance studies, were all normal during the illnesses.

TREATMENT

The initial treatment consisted of immobilization, elevation and cooling of the involved hands with ice packs. The men were placed on a high protein diet with supplementary multiple vitamins and were given rutin 20 mg. three times a day, the latter because of the possible therapeutic effects in capillary damage.[11]

Penicillin therapy, 300,000 units (duracillin*) per day, was started in case 1 after drainage of the bullae with the concurrent development of a low grade fever and increase in sedimentation rate; in cases 2 and 3 it was started after the first debridement. This was considered to be prophylactic therapy because of the possible deleterious effects of concurrent infection.

The surgical debridements were performed in cases 1, 2 and 3 on the thirty-ninth, fifty-third and sixty-seventh days after exposure, with the patient under general anesthesia because of the extreme tenderness of the tissue. An attempt was made to clean off all necrotic tissue without damaging the newly formed epithelium. The lesions were covered with petrolatum gauze, and pressure dressings were applied. These dressings were kept on for about fourteen days. Gentle manipulation of the involved fingers was carried out

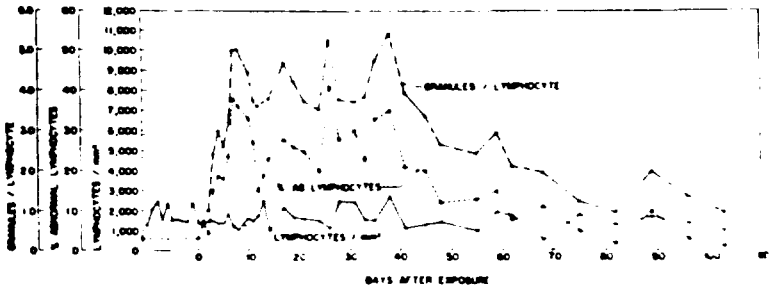

Fig. 12 (case 1).—Refractive granules in the cytoplasm of the lymphocytes at various stages of treatment.

whenever possible while the patients were under anesthesia and throughout the course of the disease. Skin grafting has been performed in cases 1, 2 and 3 with satisfactory "takes."[12] In all cases, phase 4 of the

12. Rickers, P. E., and Field, J. B.: Control of Hemorrhagic Syndrome and Reduction in X-Irradiation Mortality with a Flavanone, Science **107**: 16 (Jan. 2) 1948.

13. Brown, J. B., McDowell, F., and Fryer, M. P.: Surgical Treatment of Radiation Burns, Surg., Gynec. & Obst. **88**: 609-622 (May) 1949.

clinical course has actually just been entered and these persons will have to be followed closely for many years.

SUMMARY AND CONCLUSION

Four cases of beta ray burns of the hands have been reported. Although small doses of beta rays are not considered a serious external radiologic hazard, this form of ionizing radiation is seriously destructive in larger doses. The clinical response to such irradiation may be divided into four phases:

1. A sensation of tingling and itching at the time of exposure followed within a matter of hours by a slight erythema and edema (sometimes associated with blanching of the skin), which lasts for two to three days.

2. A latent, asymptomatic period of three to five days.

3. A secondary erythema, which is soon followed by vesicle and bulla formation. These vesicles dry and desquamate after about three weeks, leaving a new layer of thin epithelium.

4. A chronic phase characterized by atrophic epithelium and loss of secondary epidermal structure. Telangiectases, hyperkeratoses and malignant degenerations are potential complications which may develop over a period of time.

Sometimes, especially in persons receiving a lesser exposure, phase 1 and 2 may pass unnoticed or may be absent altogether. In view of the time lapse between exposure and phase 3, any exposure to beta radiation should be taken seriously.

The only significant hematologic findings were a neutrophilia, an increased sedimentation rate, an increase in the number of refractive granules in the cytoplasm of the lymphocytes and a low grade reticulocytosis. Liver function tests, nitrogen balance studies and urinalyses were consistently normal. Local treatment consisted of routine burn therapy with accessory chilling and later surgical débridement and skin grafting. General treatment involved the use of rutin, high protein diet, multiple vitamins and parenteral penicillin. Prognosis is dependent on the status of the vascular supply to the burned areas, especially as related to the skin grafting. Careful late follow-up studies are necessary in order that early malignant changes may be recognized.

Amebiasis for the Clinician.—Amebiasis means the presence in the human body of *Endamœba histolytica*. The other amebas which are found at times in the human body, with the possible exception of *Dientamœba fragilis*, are nonpathogenic. *D. fragilis* has been associated with severe diarrhea by Hakansson and others, although its actual pathogenicity has not been established. For the physician and the medical student it is necessary only to recognize *E. histolytica* with certainty. A suspected ameba then is identified either as a pathogen, *E. histolytica*, or as a nonpathogen, needing no further attention except as a suggestive guide that the patient has been exposed to fecal or other contamination and, hence, has been exposed to risk of ingestion of cysts of *E. histolytica*. It is desirable that every physician have access to a reliable laboratory for diagnosis of *E. histolytica*. Failing such access, he can for himself often make the diagnosis of the one known pathogenic ameba. Knowledge of its diagnosis and treatment is essential for every practicing physician, but is too often lacking. . . . In the past 10 years, research has introduced no feasible new methods in diagnosis and treatment. However, the literature reflects two new emphases that are important. The first is attention to X-ray findings, especially in the cecum. The second is control of secondary bacterial infections of amebic lesions with penicillin and sulfa drugs.—Alfred C. Reed, M.D., Department of Medicine, Stanford University School of Medicine, San Francisco, *Stanford Medical Bulletin*, August 1949.