A Comparison of Local and Systemic Effects Following Contact and Flash Burns *

JAMES W. BROOKS, M.D., B. W. HAYNES, JR., M.D., W. T. HAM, JR., PH.D., FRED SCHMIDT, M.S., RAY WILLIAMS

From the Surgical Research Laboratories, Department of Surgery, and the Department of Biophysics, Medical College of Virginia, Richmond, Virginia and Walter Reed Army Institute of Research, Walter Reed Army Medical Center, Washington, D. C.

PREVIOUS REPORTS from this laboratory have shown the response of healthy adult mongrel dogs to a standard contact burn,[1] and the response when this thermal trauma was complicated by the addition of whole body irradiation.[2] The burns produced in these previous studies would probably be comparable with regard to extent, depth, and irradiation injury to a major proportion of the burn and/or burn plus whole body irradiation casualties following atomic detonation, anticipating that these burns would be due to secondary fires. However, a certain group would suffer thermal trauma as a result of direct exposure of unprotected human skin to the atomic flash. Such burns would constitute the so-called "flash burns." Since the process responsible for flash burn trauma differs from that encountered in the production of contact burns (i.e., burning clothing, hot water scalds, etc.) in many physical respects such as duration of exposure and temperature, it seemed advisable to investigate the biologic response to flash burns with regard to overall clinical course and local wound characteristics, so that these might be compared with previous experience involving contact burns, both in the clinic and in the experimental animal.

Studies reported in this investigation deal with the response of dogs to: (a) a 20 per cent deep second degree "flash burn" delivered in one second, (b) a 20 per cent deep second degree "flash burn" delivered in three seconds, (c) 100 r of whole body irradiation, (d) a 20 per cent deep second degree "flash burn" (one second) combined with 100 r of whole body irradiation, and (e) trauma as in (d) treated with penicillin.

Unprotected personnel exposed to an air burst within the lethal radius for ionizing radiation would be subjected also to overwhelming blast and thermal effects. Beyond this lethal radius would be found many potential survivors suffering from severe physical and thermal trauma combined with sublethal doses of ionizing radiation. We have confined our studies to this region in the belief that victims outside the lethal radius of ionizing radiation represent the group which would be benefitted most by therapy. The additional insult to the severely burned patient of moderate doses of gamma rays introduces complicating factors in burn therapy which deserve study.[2]

THERMAL ENERGY SOURCE

Our source of thermal radiation for flash burn studies has been described elsewhere in detail.[8] Briefly, a 24-inch Army search light focused the beam on a circular area of 20.7 cm^2. Thermal dosimetry was performed with a water calorimeter and exposure times were controlled and measured accurately by means of a special shutter

* Presented before the American Surgical Association, White Sulphur Springs, West Virginia, April 13, 1956.

This study was performed under Contract No. DA-49-007-MD 99, between the Medical Research and Development Division, Office of the Surgeon General, Department of the Army, and the Medical College of Virginia.

and electronic timing devices. Uniform thermal intensities of 8.0 cal/cm²/sec. were obtainable over the burn area. Exposure times of one second at this intensity delivered a dose of 8.0 cal/cm²; in those experiments where a thermal dose of 8.0 cal/cm² was delivered over a three-second interval, the thermal intensity was reduced to the appropriate value by defocusing.

Individual burns with a circular area of 20.7 cm² were distributed over the animal's surface area in sufficient number to involve 20 per cent of the total body area. Burns were distributed to insure an area of normal tissue between adjacent lesions. Total time required for producing each 20 per cent burn was 10 to 15 minutes.

WHOLE BODY IRRADIATION SOURCE

The roentgen ray apparatus used for whole body irradiation was a 1000 KVP tube.[4] Irradiation factors were as follows: F.S.D., 200 cm.; filtration, 2 mm. beryllium plus 1 mm. Al; dose rate, 17 r/min.; field, whole body; h. v. l., 12 cm. of tissue or 1.3 mm. Pb; effective wave length, 200 Kev. Dose in free air or within masonite phantoms was measured with Victoreen 25 r thimble chambers. Dogs were exposed on each side for approximately three minutes, and usual "baking" technic. It required about six and a half minutes to expose each animal to 100 r as measured in free air.

During atomic explosions exposure to ionizing and thermal radiation occurs simultaneously. Since this was not possible experimentally in the laboratory, we burned approximately 65 per cent of the animals first and then exposed them to x-rays; the other 35 per cent were irradiated first and then burned. No clinical differences were detected between these alternate procedures.

Observations. A complete blood count was done three times weekly for three weeks, then two times weekly for two weeks, and weekly until control levels were reached. A total of 3.0 ml. was withdrawn for each assay. Immediately after burning, whole blood samples were studied every two hours through the eighth postburn hour. These determinations included hemoglobin, hematocrit, RBC, WBC, differential, and plasma protein.

Blood volumes were done on all animals prior to burning, at six hours postburn, and weekly thereafter. The Evans Blue Dye (T-1824) technic was used, one sample being withdrawn ten minutes after injection of the dye. Blood and wound cultures were taken daily beginning the third day through the 21st day post trauma.[7]

Sodium pentobarbital was given intravenously in doses of 20 mg/Kg. body weight immediately prior to burning. The effect of this anesthetic on the blood picture during the subsequent eight to ten hours has been discussed previously.[1] For details of animal care and preparation as well as determination of surface skin area, the reader is referred to previously published data.[1]

(A) *Flash Burns Alone: 20 Per Cent Body Surface, 8 cal./cm². in 1 second.* A total of 50 dogs (20 males and 30 females) were studied in this group; 30 were studied until wound healing was complete, while the remaining 20 dogs were sacrificed at 24 hours, being used mainly to study the hemoconcentration phase and plasma loss by blood volume determinations. The mortality was 6.6 per cent (two animals) in the 30 dogs which were not sacrificed until wound healing was complete; there were no deaths prior to sacrifice at 24 hours in the other group. Death was due apparently to a gamma and/or beta streptococcus septicemia as shown by blood cultures[7] and terminal bronchopneumonia with small lung abscesses. Death occurred on the ninth and tenth days in the two animals. This is compared to a death rate of 13.2 per cent in the series of 38 dogs studied following a similar burn produced by the contact method reported previously.[1] Death in this latter group had occurred between the fifth and 14th days.

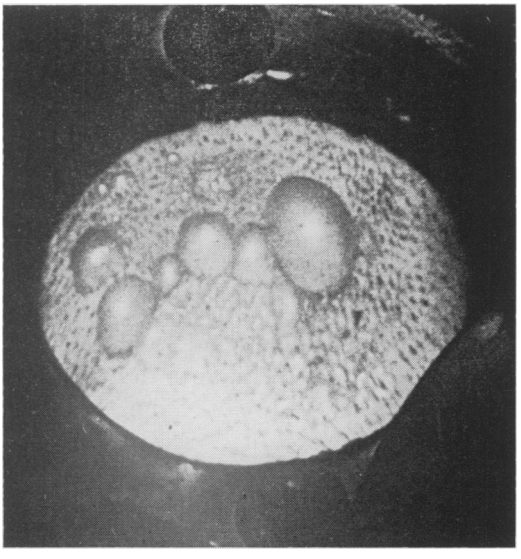

FIG. 1. Photograph taken from high speed moving picture of a flash burn produced with the 24" Army searchlight. Incident thermal energy was 8.0 cal./cm.^{2}/sec. and time was one second. This photograph was taken just prior to shutter (seen in lower right hand corner) closure. The vaporization of dermal moisture as seen by the large blisters is striking. Lack of definition in lower central portion is due to steam from previously ruptured blisters. All blisters rupture prior to closure of shutter. These blisters are not analogous to those seen in 2° burns, for these are due to vaporization of dermal moisture by the sudden intense heat of the thermal flash and not extravasated fluid in damaged skin. This apparently aids greatly in the development of a "dry wound" from the very beginning.

Therapy. The animals were given food and water as desired. However, no attempt at dietary, fluid, blood, or antibiotic therapy was made at any stage of the pre- or postburn study period.

Clinical Course. In contrast to the animals studied following a similar contact burn, these animals did not appear clinically as ill as did those who received the contact burn. Rectal temperatures showed an average elevation of 1 to 2.5° F. above a control of 101.0° F. during the second through the 12th days following injury; this was somewhat higher than that found in the contact burn group.

Following a flash burn of the magnitude given in this study an eschar is formed on the burned surface. (Figures 1 and 2 show photos and photomicrographs of the burn lesions as compared to the contact burns.) This firm, leathery feeling skin is elevated as a plaque above the surrounding unaffected tissues with the resultant edema involving the lower layers of the epidermis and dermis; whereas, in the lesion of a contact burn, the wound feels soft and doughy, with evidence of serum leaking through skin abrasions. No such soft appearance is seen in the flash eschar. This initial eschar persists throughout the subsequent days of tissue breakdown and repair. It actually forms a protective coat in intimate association with the line dividing normal and injured skin. It would seem that gross infection of the wound is minimized since these flash burn wounds showed markedly less purulent drainage than contact burn wounds at all stages following burning, although wound cultures were taken and showed essentially the same bacteria in both types.[7] In the contact burn, large collections of purulent material would elevate the dead eschar from the underlying raw surfaces. This did not occur in the flash burn since the eschar seemed to be more firmly fixed and thus appeared to limit overwhelming local wound infection. Healing of the flash wound was usually complete by four weeks with the eschar acting as a protective dressing for epithelization from deep hair follicles and wound edges in a lesion that did not show extensive suppuration. Usually, by the time the eschar had disappeared, the wound had healed almost completely. This was not true in the contact burns where the eschar was usually off by ten days, leaving a raw, poorly granulated surface which healed some six weeks later. Similar local findings with flash burns have been reported in pigs by Pearse [6] and Hogg.[5]

The animals' weight gradually decreases with a low at 21 days postburn, approximately 25 per cent below control values; return to control weights is gradual and almost complete by eight weeks.

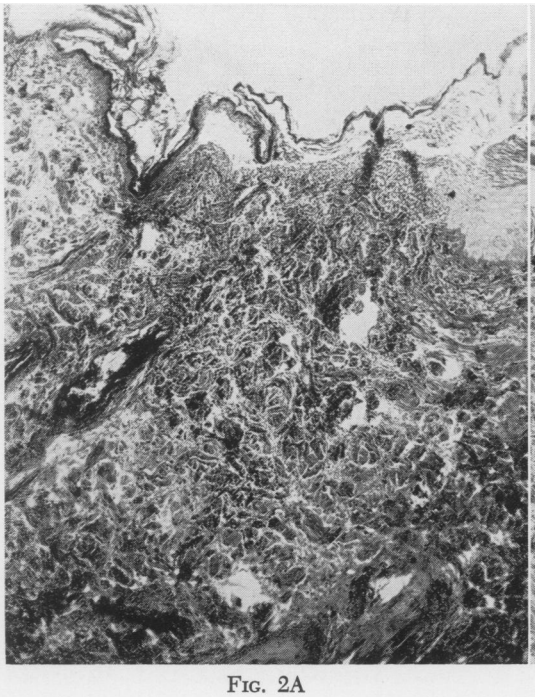

FIG. 2A

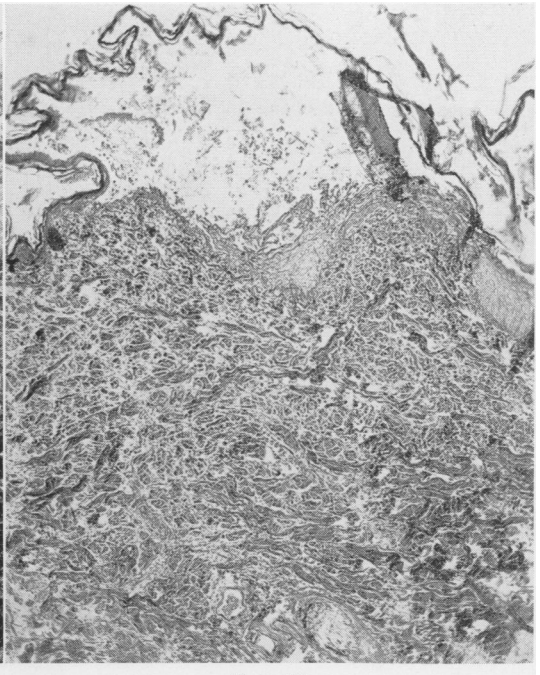

FIG. 2B

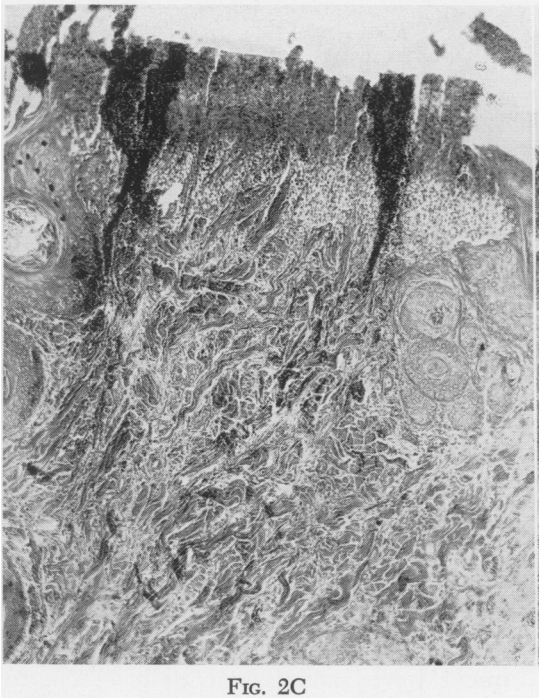

FIG. 2C

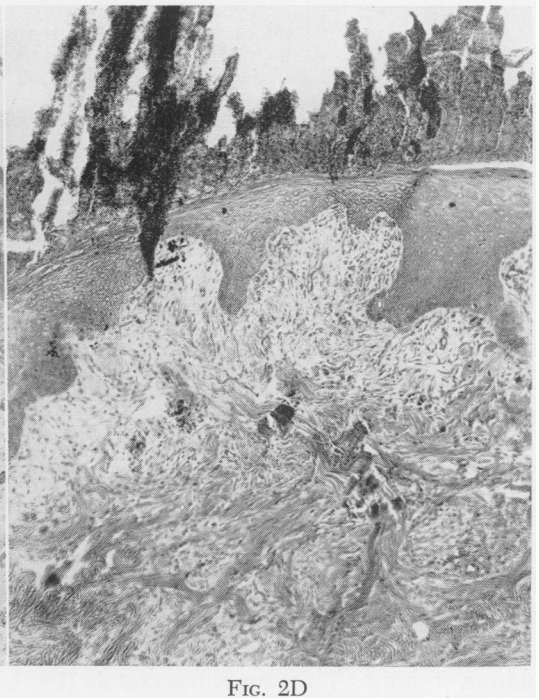

FIG. 2D

FIG. 2. a. Photomicrograph of contact burn at four days post burn illustrating comparable depth of dstruction to that seen in Figure 2 (b). Note more marked leucocytic infiltration and comparable line of demarcation of normal and burned skin as compared to Figure 2 (b). b. Photomicrograph of flash burn at four days postburn illustrating comparable depth of destruction to that seen in Figure 2 (a). c. Photomicrograph of contact burn at 12 days post-burn. Leucocytic infiltration and suppuration are still marked and epithelial advancement slow. Islets from deep hair follicles show retarded growth and advancement. d. Photomicrograph of flash burn at 12 days near margin of burn showing overlying eschar with abundant healthy mounds of epithelium.

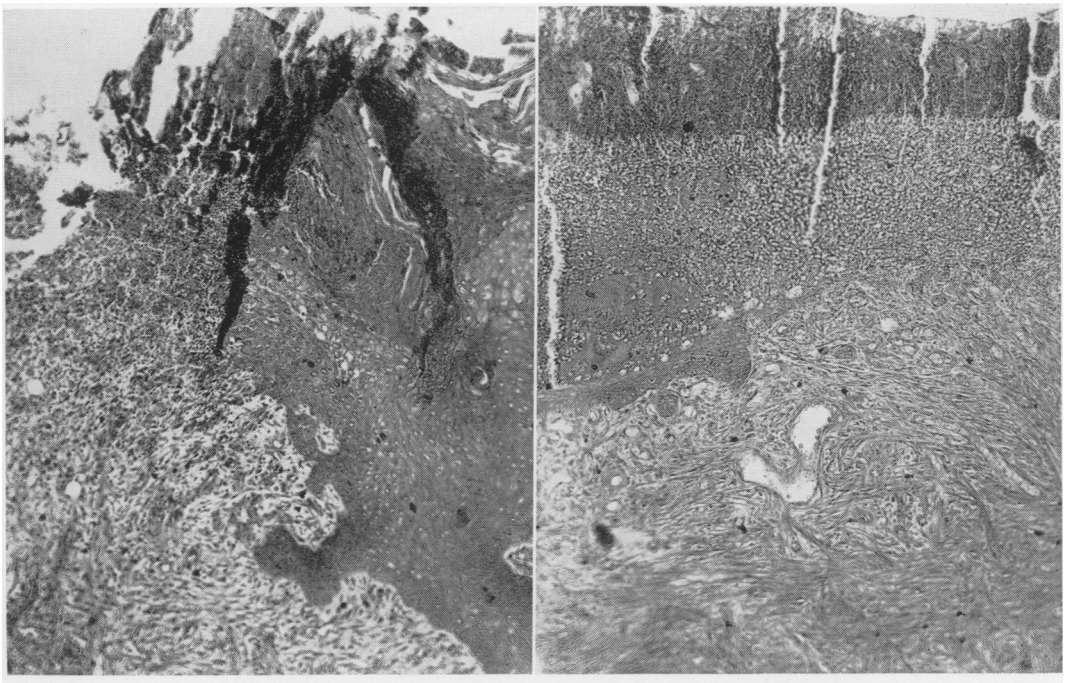

FIG. 2E FIG. 2F

FIG. 2. e. Photomicrograph of contact burn at 17 days. Note marked leucocytic infiltration and suppuration still present. More abundant epithelization at margin with slow advancement. This wound will be completely healed in 5–6 weeks. f. Photomicrograph of flash burn at 17 days with thick eschar and continued thin advancing epithelization. Very little inflammatory reaction is present, probably due to excellent sealing by eschar. This wound will be completely healed in four weeks.

Thus, it would appear that in the dog, flash burns of the same apparent depth of destruction as contact burns are less severe clinically, show less gross suppuration, and heal more rapidly, apparently as a result of the thick, hard eschar protecting the raw undersurface.

Blood Volumes. Using T-1824 (Evans Blue Dye) blood volumes were done on all experimental animals prior to burning. The procedure was repeated on 38 animals at six hours postburn. This revealed an average loss of 14 ml. of plasma per kilogram of body weight or 28 per cent of the control plasma volume. This corresponded to the level of greatest hemoconcentration as seen by serial hemoglobin, hematocrit, and RBC determinations. Following a comparable contact burn, 18 ml. of plasma was lost at six hours postburn or 29 per cent of the control plasma volume. Since the 4 ml. difference in plasma loss per kilogram body weight is not significant, it is assumed that plasma loss into the injured tissues is comparable in the contact and flash lesions of equal depth. Thus, fluid replacement would be handled in a similar fashion for both types of burns during the shock phase.

Blood volume studies in the weeks following initial injury reveal an expanded plasma volume 25 per cent above the control level at seven days postburn being the maximum expansion noted. This gradually returns toward normal during the subsequent weeks and is essentially at control levels by the fifth week post burn. The red cell mass and total circulating hemoglobin gradually decline with the lowest value at 21 days post burn when they are an average of 28 per cent below control values. There

is a gradual climb in these values during the subsequent weeks, but they had not reached normal or control values by the eighth week post burn.

Peripheral Blood. The hemoglobin, hematocrit, and red blood cell count paralleled each other, showing essentially the same picture as that observed with the contact burn. In the immediate period post burn there was a gradual increase of all values to an average peak of 20–23 per cent above control values at six hours after burning. This corresponds to the point at which plasma volume determinations indicated a 28 per cent loss of plasma volume (14 ml./Kg. body weight). The maximum anemia occurs about the 21st day post burn; during the first three days the red cells disappear rapidly to 10–15 per cent below control values. Thereafter, the decline is more gradual to the minimum of 20–25 per cent below control values on the 21st day. The recovery toward control levels is very gradual and not complete by the eighth week post burn.

The maximum leucocytic response following injury occurs on the tenth day when the white blood cell count is elevated approximately 115 per cent above control levels. However, in the flash burn animals the clinical appearance of the burn is not nearly so severe as seen in the contact burn and the leukocyte response is not as great nor as prolonged.

The plasma proteins show a decrease from normal immediately following burn trauma and remain low through the 14th day postburn when they gradually rise, but do not reach control levels by the eighth week.

Blood and Wound Cultures. A complete account of the bacteriologic studies done is in publication and may be referred to for details.[7] A total of 20 dogs were studied with 239 blood cultures and 185 wound cultures. Blood cultures were obtained from alternate femoral veins, sterile precautions being taken to avoid skin contamination, and were studied from the third through the 21st day postburn. Wound cultures were taken at the same time, but usually were discontinued at an earlier date due to the dry eschar making further cultures unobtainable.

Of the 239 blood cultures, 24 per cent showed the presence of bacteria, gamma streptococci and micrococcus albus being predominant. Usually only one type of organism was isolated from each blood specimen. In only one instance was beta hemolytic streptococcus isolated from the blood cultures. This was in an animal that died subsequently. The highest percentage of positive blood cultures occurred between the seventh and tenth days postburn.

In a similar group of contact burns studied previously, the blood cultures showed a 78 per cent incidence of positive blood cultures for the gamma streptococcus and no positive cultures for the beta hemolytic streptococcus.

Micrococci and/or streptococci were isolated from all 185 wound cultures taken in the flash burn group. Nearly every burn showed the presence of alpha hemolytic or gamma streptococci, but 23.8 per cent of all wound cultures showed beta hemolytic streptococci despite the fact that this organism was isolated only once from the blood cultures.

Cultures obtained from contact burns showed the presence of beta hemolytic streptococci in nearly all wounds, giving a much higher incidence than with flash burns, but no beta hemolytic streptococci were found in the blood stream.

The wound and blood cultures studied tend to support the clinical impression that these flash burns show less suppuration and less local evidence of infection. This probably accounts for the lowered blood stream invasion, lowered mortality, more rapid local healing, and earlier overall recovery from the flash burn wound. We believe that this is due largely to the initial eschar formation which acts as a barrier to

local bacterial invasion and growth and helps prevent blood stream invasion.

(B) *Flash Burns Alone: 20 Per Cent Body Surface, 8 cal./cm². in 3 sec.* A total of 12 dogs (seven males and five females) was studied in this group. There was one death on the 19th day postburn, apparently from distemper. The mortality rate was 8.3 per cent. All phases of this study paralleled those seen in the (A) group. The peak hemoconcentration was at six hours with an average loss of plasma from the circulating blood stream of 25–30 per cent or 16 ml. of plasma per kilogram body weight.

It is concluded from the clinical and laboratory data on Groups (A) and (B) that no difference can be detected between flash burns delivered in one second and those delivered in three seconds. This observation conflicts with our experimental findings on human volunteers, where a decrease in exposure time (thermal dose held constant) resulted in a more severe burn lesion. It is felt that a difference would have become apparent in the dogs had we been able to decrease the exposure time to a half second or less as we did with the human volunteers.[3]

(C) *100 r Whole Body Irradiation Alone.* A total of 20 dogs (11 males and nine females) were studied in this group with no mortality. Each animal received the 100 r whole body irradiation while under sodium pentobarbital anesthesia and was studied subsequently for 40 days, the studies including all factors previously mentioned in the other groups.

The hemoglobin fell quite gradually to a low of 15 per cent below control values on the 17th and 18th days post irradiation. There was a gradual climb to control levels by the 35th day. Hematocrit and red blood cell count values did not drop lower than ten per cent below control values and also rapidly regained control levels. A similar small, but not significant, drop was seen in the red cell mass with little to no change in the plasma volume.

The most significant change was in the white blood cell count which fell to levels of 28 to 42 per cent below controls between the sixth and 24th days post irradiation. A return to control levels was attained by the 40th day following irradiation. There was an early drop in the lymphocytes with return to relatively normal differential counts in seven to ten days.

Clinically, these dogs showed no evidence of illness in their activity, temperatures, weight changes, or appetite.

Blood cultures studied on ten dogs (six males and four females) of this group daily for three weeks revealed two positive cultures for micrococcus albus out of 140 cultures, an incidence of 1.42 per cent positive cultures.

Since there were no wounds to culture on these animals, we elected to use them prior to the whole body irradiation as subjects to study the possible origin of the beta hemolytic streptococci found so frequently in our burn wound. Accordingly, 150 cultures were obtained on 25 control dogs from five areas—(1) the mouth, (2) the nose, (3) the skin or fur, (4) the anus, (5) the genitalia. It was found that 40 per cent of the animals harbored the beta hemolytic streptococci in one of the above listed culture sites. The most common sites were the anal and genitalia regions. Alpha and gamma streptococci and the micrococci were found at all sites in practically every animal studied.

(D) *Deep Second Degree 20 Per Cent Body Surface Burn (One Second) Combined with 100 r Whole Body Irradiation.* A total of 30 dogs (14 males and 16 females) were studied in this group with a mortality of 37.0 per cent. Death occurred between the fifth and sixth days in all but one dog who died on the 37th day. Death was attributed in four animals to a beta hemolytic streptococci septicemia and in one animal to a septicemia with clostridium perfringens, micrococcus albus, and gamma streptococci. In the remaining six deaths

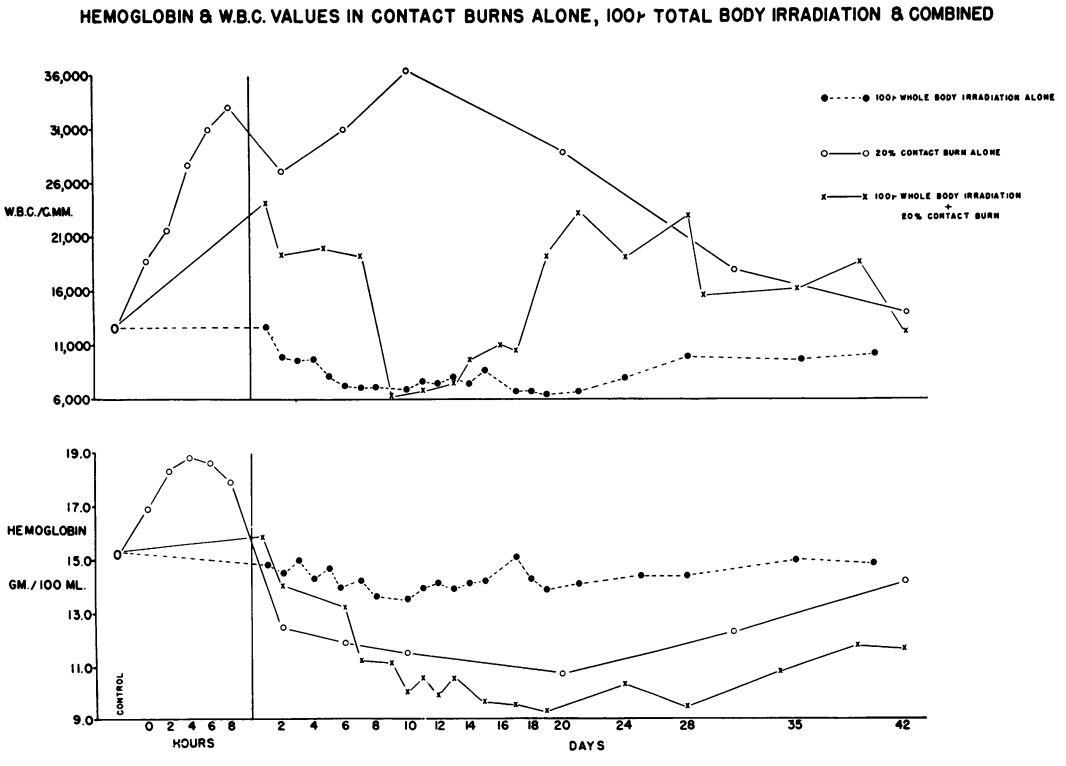

FIG. 3. This graph is a summary of all the animals studied in each group indicated in the contact burn groups of experiments. Study period was 70 days but graphs end at 42 days where trend toward control values is definite.

no blood cultures had been obtained and autopsy showed only bronchopneumonia and occasional small pulmonary abscesses.

The mortality rate in this combined injury group is much less than that observed with the contact burn combined with 100 r of whole body irradiation where it was 73 per cent. This can probably be explained on the basis of the decrease in local wound infection with subsequent less blood stream invasion.

No significant change or difference could be detected in the wounds of those animals living long enough to complete wound healing as compared to non-irradiated dogs. The rate of healing was the same and the gross appearance of the burns unchanged. Their clinical course as regards weight and temperature changes was the same as in Group (A). This same similarity has been noted previously in the contact burn animal whether or not whole body irradiation was given.

Blood volume studies showed a 25 per cent reduction of plasma volume at 6 hours (13 ml. plasma per kilogram body weight). Subsequent blood volumes indicate an expanded plasma volume at seven days 30 per cent above the control level. This gradually returns to normal figures by the fifth postburn week. The red cell mass and total circulating hemoglobin decline gradually to a low at about the 35th postburn day which is 29 per cent below the control values. A gradual rise begins at this point and is not back to control levels by the eighth week.

The hemoglobin, hematocrit, and red blood cell counts are similar in pattern to those found in the non-irradiated animals with the exception that they remain at lower levels for a longer period of time and return more slowly. Return to control

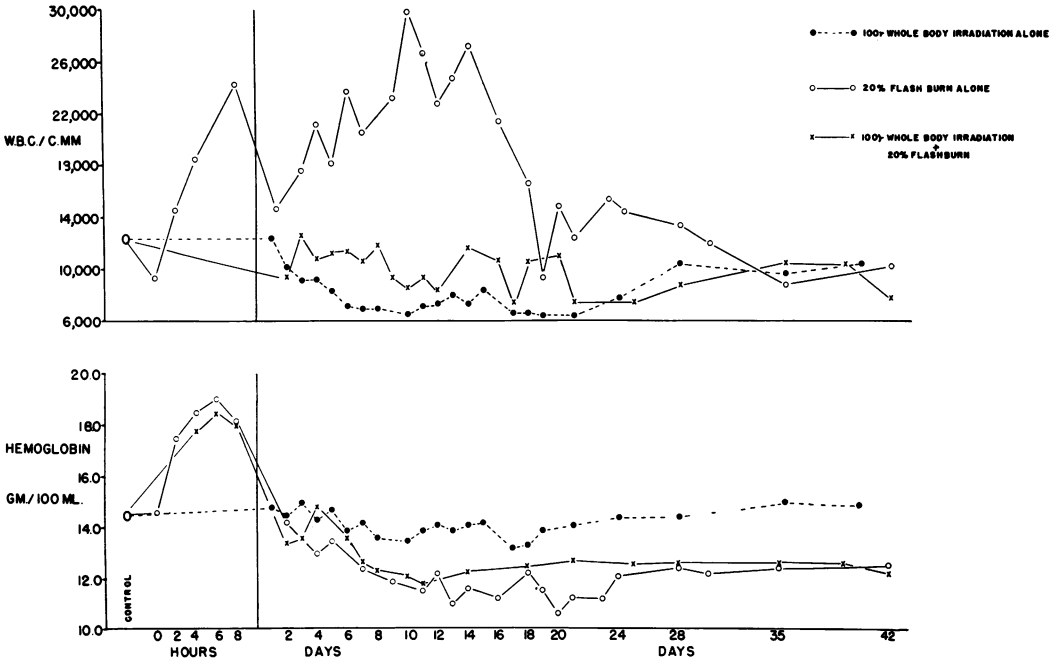

FIG. 4. This graph is a summary of all the animals studied in each group indicated in the flash burn group of experiments. Study period was 70 days but graphs end at 42 days where trend toward control values is definite.

values is not complete by the eighth week and levels are lower than were seen with the flash burn alone at this period of recovery.

The leukocytic response is quite different from that seen in the flash burn alone, but closely parallels that found in the contact burn plus whole body irradiation of 100 r. Instead of the leukocytosis of 20,000–30,000 seen in the flash burn alone during the first week these animals show total white blood cell counts closely paralleling the control value of 12,000 except between the ninth and 15th days when values around 8,000 are common, approximately 25–30 per cent below control values. Figures 3 and 4 give summary of peripheral blood studies in contact and flash groups.

Blood cultures were drawn on 20 dogs from the third through the 21st days post burn with a total of 221 such blood cultures; 32 per cent were positive for beta hemolytic streptococci. In a similar group of contact burns with 100 r whole body irradiation, 75 per cent of blood cultures were positive for beta hemolytic streptococci. In the flash burn plus 100 r whole body irradiation, 53 per cent of the wound cultures were positive for beta hemolytic streptococci while in the comparable group with contact burns plus 100 r whole body irradiation nearly all cultures from the wounds showed the beta hemolytic streptococci.

(E) *Combined Injury as in D Above with Penicillin Therapy.* A 20 per cent deep second degree flash burn plus 100 r whole body irradiation was given eight dogs. Twenty-four hours later they were given 900,000 units of Crysticillin® I.M. and this was repeated daily through the 17th day post trauma. One animal died on the eighth day post trauma, a mortality of 12 per cent. The remaining animals had completely

healed and all blood levels and weight had returned to control levels by the 28th day.

All phases of the study were much less severe and recovery uncomplicated in the seven dogs that survived. A similar picture was seen in the contact burn plus whole body irradiation when treatment was instituted with penicillin except that the recovery period was far longer—an average of 120 days. The treated contact burns had a mortality of 14 per cent.

No blood or wound cultures were studied in this group.

SUMMARY

Flash burns were produced experimentally in dogs and compared with burns of a comparable depth and extent produced by the contact method. Peripheral blood studies were similar in both groups. However, blood culture studies and mortality seemed to indicate a less fatal outcome in the flash burn group which was thought greatly influenced by the initial eschar formation in the flash burn, this eschar acting as a protective coat to prevent the purulent suppuration seen in contact lesions. It would appear that the overall clinical approach to flash and contact burns will be the same.

It is not the purpose of this report to advocate any form of therapy in burn wounds, but rather to point out local factors which influence to a marked degree wound healing and the systemic effect produced by varied sources of thermal energy which produce burns.

ACKNOWLEDGMENT

Technical assistance of Harry Mueller, Mrs. Evelyn Mueller, Fred Pierce, Leslie Ellis, Max Rittenburg is herewith acknowledged.

BIBLIOGRAPHY

1. Brooks, J. W., P. Robinett, T. L. Largen and E. I. Evans: A Standard Contact Burn. Surg., Gynec. and Obst., **93**: 543, 1951.
2. Brooks, J. W., E. I. Evans, W. T. Ham and J. D. Reid: The Influence of External Body Radiation on Mortality from Thermal Burns. Ann. Surg., **136**: 533, 1952.
3. Evans, E. I., J. W. Brooks, F. H. Schmidt, R. C. Williams and W. T. Ham, Jr.: Flash Burns in Human Volunteers. Surg., **37**: 280, 1955.
4. Ham, W. T., Jr. and E. D. Trout: Million-Volt Berillium Window X-Ray Equipment for Biophysical and Biochemical Research. Radiology, **55**: 257, 1950.
5. Hogg, L., J. T. Payne and H. E. Pearse: Experimental Flash Burns. Ann. of Pathol., **49**: 267, 1950.
6. Pearse, H. E. and H. D. Kingsley: Thermal Burns from the Atomic Bomb. Surg., Gynec. and Obst., **98**: 385, 1954.
7. Reid, J. Douglas, J. W. Brooks, W. T. Ham and E. I. Evans: The Influence of X-radiation on Mortality Following Thermal Flash Burns: The Site of Tissue Injury as a Factor Determining the Type of Invading Bacteria. Ann. Surg., **142**: 844, 1955.
8. Schmidt, Frederick H., R. C. Williams, W. T. Ham, J. W. Brooks and E. I. Evans: Experimental Production of Flash Burns. Surg., **36**: 1163, 1954.

DISCUSSION.—PRESIDENT BLALOCK: I feel sorry for those who did not stay to hear this final paper, because it has been excellent. I wish Everett Evans could have been here. I am so glad to see people like Dr. Willis Gatch, Pete Churchill and others of us who no longer are quite as young as we used to be, who still retain a great interest in what is new in surgery.