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**COMBINED EFFECTS OF TOTAL BODY X IRRADIATION  
AND RADIANT ENERGY THERMAL BURNS:  
I. STUDY OF BLOOD COAGULATION**

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**U.S. NAVAL RADIOLOGICAL DEFENSE LABORATORY**

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ENERGY THERMAL BURNS; I. STUDY OF BLOOD COAGULATION

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Health and Biology

Technical Objective  
AW-6

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U. S. NAVAL RADIOLOGICAL DEFENSE LABORATORY  
San Francisco 24, California

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22 October 1953

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ABSTRACT

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The effects of high intensity radiant energy thermal burns, total body X irradiation, and of combinations of thermal burns and total body X irradiation upon the blood coagulation mechanism has been investigated in rats.

Total body X irradiation at a dose of 500 r produces a rapidly developing thrombocytopenia and a defective clotting mechanism as measured by Lee-White and heparin clotting times. At a dose level of 100 r a brief thrombocytosis is followed by a transient mild thrombocytopenia.

The high intensity radiant energy thermal burns alone produce a brief fall in the level of circulating platelets followed by a sustained thrombocytosis.

The combined effects of thermal burns and total body X irradiation consist of the same initial fall of platelets seen in burns alone followed by the same degree of thrombocytopenia seen in animals receiving total body X irradiation at the particular dose given. However, the clotting defect, as measured by the Lee-White and heparin clotting times, is not as severe in the combined injury animals.

The observed defective coagulation mechanism fails to account for the reported enhanced mortality seen in combined injury animals. However, the thrombocytopenia and defective clotting mechanism does permit the recognition of casualties in which burns or other traumata are complicated by the occurrence of superimposed total body X irradiation.

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INTRODUCTION

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The development of atomic weapons and of other uses of nuclear energy has created the distinct possibility of overwhelming numbers of casualties. That the injuries resulting from the use of an atomic weapon would not be simply due to the effect of ionizing radiation, but would result from a combination of injuries including thermal burns, ionizing radiation, wounds, and fractures is quite evident from the experience of Nagasaki and Hiroshima. Brooks<sup>1</sup> has presented a clear analysis of the problem of burn casualties in the zone closest to the hypocenter of a nuclear detonation, emphasizing the important role ionizing radiation plays in prognosis and choice of treatment. There has already accumulated in the literature a large body of reports of the effects of ionizing radiation alone, but as yet only a limited number of studies have appeared documenting the effects of a combination of thermal burns and ionizing radiation. Brooks<sup>1</sup> has reported experiments with dogs which show that a distinct increase in the mortality from either trauma alone can be expected if combined with the other injury. Similar studies in mice by Parr<sup>2</sup> have shown a similar increased mortality from combined thermal burns and total body X irradiation. In this Laboratory<sup>3,4</sup> studies with rats in both hot water and radiant energy thermal burns has shown the same enhanced mortality from concomitant total body X irradiation.

As part of a large study of the combined effects of high intensity radiant energy burns and total body X irradiation, a series of observations have been made of blood coagulation. This study includes observations of the modified Lee-White (silicone) clotting time, heparin clotting time, blood platelet concentration, and prothrombin time.

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METHODS

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Animals

Female rats of the Sprague-Dawley strain, weighing initially between

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140 and 180 g and approximately 6 weeks old, from the Laboratory colony were used in all experiments. They were housed in individual wire cages and fed Purina Chow and water ad lib. The animals were divided into seven experimental groups as follows: Group I—control (C); Group II—500 r total body X irradiation (X<sub>5</sub>); Group III—100 r total body X irradiation (X<sub>1</sub>); Group IV—4 strip burns plus 100 r (B<sub>4</sub>X<sub>1</sub>); Group V—2 strip burns plus 500 r (B<sub>2</sub>X<sub>5</sub>); Group VI—2 strip burns (B<sub>2</sub>); and Group VII—4 strip burns (B<sub>4</sub>). Hereafter the animals of a given group will be referred to frequently by the designation in parenthesis (C, X<sub>5</sub>, B<sub>4</sub>X<sub>1</sub>, etc.).

### Depilation

All animals were clipped and depilated using a commercial strontium sulfide depilatory according to the method of Kuhl, et al.<sup>5</sup> Approximately 72 hr following depilation the animals were anesthetized with pentobarbital sodium and were treated according to the experimental procedure. Control rats were depilated and anesthetized only.

### Method of Total Body X Irradiation

The four groups exposed to X irradiation were treated in a 250 KVP Westinghouse therapy unit operating at 15 ma, with 0.5 mm Cu and 1 mm Al filter at a skin to target distance of 40 in. and with a dosage rate of 23 r/min, HVL 1.5 mm Cu. Dose rates on a particular day were calculated from several measurements in air made with a Victoreen chamber.

### Method of Administering Radiant Energy Thermal Burns

The four groups which were burned were treated by positioning the anesthetized rats on a rotary turntable behind asbestos insulated rectangular apertures measuring 2.5 by 12 cm by means of an elastic cloth sling. A Mitchell-Nelson carbon arc background projector focused to a usable spot 3.5 cm in diameter was the energy source. The turntable rotated past the focused beam at a speed of one revolution every 42 sec. Such treatment yields an exposure time of approximately 0.4 sec with a total energy delivery of 9.0 cal/sq cm as measured by a black body radiometer, neglecting scattering and reflection. The visible strip burn resulting from a single exposure was slightly overlapped for each succeeding exposure to produce a contiguous burn. The 2 strip burns (B<sub>2</sub> and B<sub>2</sub>X<sub>5</sub>) gave mean burn areas which were 15 per cent of total body area and the 4 strip burns (B<sub>4</sub> and B<sub>4</sub>X<sub>1</sub>) gave mean burn areas of 25 per cent of total body area. Histological examination revealed that these burns are essentially equivalent to the characteristic white burn described by Sheline.<sup>6</sup> The burns were applied to the back and sides of the animals and did not interfere with excretory functions nor seriously impair ambulation.

Animals in the B<sub>4</sub>X<sub>1</sub> and B<sub>2</sub>X<sub>5</sub> groups were first given total body X irradiation and then burned approximately 20 to 60 min later.

Observation Days

A single animal from each group was used for determinations performed on the following days after injury: Days 1, 3, 5, 8, 11, 15, 21, and 30. On each of these days the animals were anesthetized with subcutaneous pentobarbital sodium and, after a determination of the circulating blood and red cell volumes by the P<sup>32</sup> method of Hevesy and Zerahn,<sup>7</sup> blood samples were drawn by cardiac puncture through the opened chest. The animals were sacrificed and subjected to complete autopsy.

Replications

The experiment was run in six replications, each starting at 3 week intervals so that at each post injury day in each experimental group there were six animals with certain exceptions where the number was reduced to four or five.

Randomization

The rats were assigned to individual groups immediately after depilation by a random number table. The selection of a particular rat from a particular experimental group for determinations on any given observation day was made by selecting the lowest numbered surviving member of that group.

Blood Sampling Technique

The following technique was scrupulously followed in the drawing of samples for both clotting times and platelet counts. A clean, dry siliconized 5 ml syringe and 21 gauge non-siliconized needle was used to draw approximately 5 ml of blood from the exposed right ventricle. The cardiac puncture must be clean and the withdrawal free of air bubbles and interruptions. The final 1.0 ml of blood drawn in the syringe was discharged into a 10 ml Erlenmeyer flask containing heparin for other determinations. Then 0.4 and 0.6 ml blood were placed into two clean siliconized stoppered 13 by 100 mm test tubes. These were placed in a water bath at 37 C for the determination of the modified Lee-White clotting time. Next, exactly 1.0 ml of blood was discharged into a 25 by 100 mm screw cap vial containing 0.002 mg of heparin. The remaining blood in the syringe, approximately 0.5 to 2.0 ml, was added to the blood in the initial Erlenmeyer flask for use in other determinations. By a separate, clean cardiac puncture made immediately following the first withdrawal, using a clean siliconized 2 ml syringe and a clean non-siliconized 23 gauge needle that had been previously rinsed and filled with Rees-Ecker diluting fluid, approximately 0.5 to 1.0 ml of blood was removed and transferred to a dry, siliconized 10 ml Erlenmeyer flask. This blood was immediately used without agitation to pipette the sample for platelet count.

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### Platelet Count

Standard Thoma red cell counting pipettes, standard Levy hemocytometer chambers, and Rees-Ecker formalin-citrate mixture as the diluting fluid as recommended by Tocantins<sup>8</sup> have been used. Once pipetted, samples were shaken for 3 min and then set aside. Prior to counting, the pipettes were reshaken for 3 min and the chamber was then filled and placed in a moist, covered Petri dish for 20 to 30 min to allow settling of formed elements. To assure an accurate count, scrupulous cleanliness of the hemocytometer and cover glass were essential. As an additional check, on each day of counting a blank of the diluting fluid was drawn up and counted. The small number of platelet-like bodies were counted and this "background" was subtracted from the measured platelet count to give the "true" platelet count. The background correction approximated 10,000 to 40,000/cu mm. In an initial evaluation of the platelet measurements, and occasionally throughout the experiment, duplicate pipettings were taken and checked for accuracy of replication. The level of accuracy was approximately 5 to 10 per cent. An occasional source of difficulty was gross clumping of platelets which led to falsely low counts because of the impossibility of counting accurately the platelet aggregations. This difficulty could be recognized on inspection and counts on these animals were discarded. Clumping most often followed difficulty in obtaining a clean cardiac puncture. All platelet count manipulations and platelet counts were performed by the same individual (W.M.D.) to assure identical technique.

### Modified Lee-White (Silicone) Clotting Time

The first tube (containing 0.4 ml of blood) in the water bath was tilted at one min intervals until the tube could be inverted. When this point was reached the second tube (containing 0.6 ml of blood) was similarly tested. Where very long clotting times were encountered 5 min intervals were used. The actual clotting time in minutes was measured from the time when blood first was aspirated into the syringe until the second tube had clotted. This modification of the test differed from other versions of the coagulation time<sup>9</sup> in the use of both siliconized glassware and two tubes.

### Heparin Clotting Time<sup>10</sup>

The vial containing 0.002 mg of heparin, to which one ml of blood had been added, was mixed by gentle swirling and placed in the water bath at 37 C. It was tested at 5 min intervals for clot formation by tilting. The heparin clotting time was recorded as the time from sample withdrawal to formation of a solid coin-shaped clot in the bottom of the vial.

### Prothrombin Time

The method of Quick<sup>11</sup> was used with siliconized glassware.

RESULTS

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Circulating Platelet Measurements

Table 1 shows the mean platelet counts of the rats of the various experimental groups on the different days of observation. In Figs. 1 and 2 these same mean platelet counts are graphed together with the simultaneous mean Lee-White and heparin clotting times. Several interesting findings are apparent. The burned rats (Groups B<sub>2</sub> and B<sub>4</sub>) show an initial small but definite fall in level of circulating platelets for 1 to 3 days followed by a sustained elevation of the platelet count for the remainder of the 30 day period. No essential difference is seen between rats receiving the burns of two different severities. The rats receiving 100 r total body X irradiation (Group X<sub>1</sub>) shows an initial slight elevation in level of platelets for 3 to 5 days followed by a fall to levels well below control values. This fall is maximal at Days 8 and 11 and is then followed by a rise to sustained normal levels. In contrast, the rats receiving 500 r total body X irradiation show a rapid and continuing decrease in the level of circulating platelets over the initial 8 day period of observation so that by the end of the period circulating platelets have almost entirely disappeared. Levels on Days 8 and 11 are consistently below 100,000 platelets/cu mm of blood. On Day 15 the platelets have begun to reappear and two of six animals show levels above 100,000/cu mm. At the end of the 30 day period, platelet levels had reached normal. For the animals receiving combined total body X irradiation and thermal burns (Groups B<sub>2</sub>X<sub>5</sub> and B<sub>4</sub>X<sub>1</sub>) the graphs of levels of circulating platelets are almost identical in pattern to those of total body X irradiation alone, with two exceptions. These animals in the initial 1 to 3 day post burn periods show the initial fall of platelets associated with burns alone. Animals in Group B<sub>2</sub>X<sub>5</sub> following the period of maximum depression show slightly higher levels than the X<sub>5</sub> group on Day 21, but levels were still distinctly below normal on Day 30. The animals in the B<sub>4</sub>X<sub>1</sub> group on Days 15 and 21 show platelet levels which are generally higher than those of the X<sub>1</sub> group.

Lee-White Clotting Time

In Figs. 1 and 2, the graphs show the changes in the Lee-White clotting times for the various groups on the different observation days. Of interest is the slight shortening of the clotting time to be observed in the burn groups on Days 5 through 30 coincidental with the observed elevation of platelet counts in these groups. The X<sub>1</sub> group shows a slight prolongation of the clotting time on Days 8 and 11 but are otherwise within the normal range. The X<sub>5</sub> group animals show a prolongation of the clotting time on Day 5 and for Days 11 through 21 when the times are in excess of 120 min. The clotting times on Day 30 in this group are normal at the same time

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that platelet concentrations have returned to normal. The B<sub>4</sub>X<sub>1</sub> group shows an initial slight prolongation of the clotting times on Day 1, but thereafter, despite changes in the level of the circulating platelets, the mean clotting times fall within the normal range. The B<sub>4</sub>X<sub>5</sub> group shows a progressive increase in the mean clotting times after Day 3 but mean values above 120 min are observed only on Days 11 and 15. The mean clotting times on Day 21 are only slightly elevated and are normal on Day 30.

### Heparin Clotting Times

Figs. 1 and 2 show graphically the mean values for the heparin clotting times in the various experimental groups on the different observation days. The control mean values show a greater day-to-day fluctuation than did the Lee-White clotting times. The heparin clotting times bring out better than did the Lee-White clotting time the initial period in the burn group and combined injury group when clotting times are slightly prolonged in association with a slight fall in platelet levels. Otherwise, the curves for the heparin clotting time in the various groups mirror very closely the observed changes in the Lee-White clotting time.

### Prothrombin Times

The mean prothrombin times for the various experimental groups at the different observation points are all in the range of 17 to 20 sec. These values do not differ significantly from the control values.

### Degree of Correlation between the Level of Blood Platelets and Measurements of Clotting Time

In an effort to compare the relative usefulness and reliability of these different determinations of blood coagulation, the three independent determinations on individual animals have been used to calculate the correlation coefficient ( $r$ ) for any two different determinations for the entire experimental series, for the control group, and the B<sub>4</sub> group. The results of these calculations are shown in Table 2.

The good degree of correlation for the control series between any two estimations of blood coagulability indicates that no single measurement has advantages over another and the same physiological variables are probably being evaluated. However, the correlation between platelet counts and either clotting time is seen to be relatively better in the control group than the experimental groups. No explanation for this can be advanced at present.

These observations of the blood coagulation mechanism following thermal burns, total body X irradiation at two levels of dosage, and thermal burns combined with total body X irradiation at the same two levels of dosage reveal a number of interesting points. Our observations in animals receiving total body X irradiation in a dose of 500 r alone show the pronounced rapidly developing thrombocytopenia which is expected at this dosage level. The changes in Lee-White clotting time and heparin clotting time vary in an inverse manner to the change in platelet levels. This is in agreement with a large body of observations on the effect of various levels of total body X irradiation in different animal species. Cohn<sup>12</sup> in careful study of the hemorrhagic syndrome in rats following 400 r total body X irradiation showed that the predominant feature of the defect was the thrombocytopenia and that defect could be most accurately measured quantitatively by means of the heparin clotting time and electrical resistance measurements of blood, using the method developed by Rosenthal and Tobias.<sup>13</sup> They emphasize the finding of a normal Lee-White clotting time (unsiliconized glassware) despite the severity of the defect as measured by other tests. The rate of clot retraction as determined by electrical resistance measurements correlated well with platelet levels. Furthermore, the presence and degree of post-irradiation hemorrhage was correlated with the defective coagulation mechanism and was maximal at 16 days.

Rosenthal<sup>14</sup> made a careful study of the semi-quantitative changes in the bone marrow of rats subjected to 700 r total body X irradiation. No apparent change occurred in the megakaryocytes in the first 24 hr but in the subsequent 3 to 5 day period they gradually decreased and were absent from Days 7 to 15. After Day 15 a few megakaryocytes were seen to reappear but only in the animals showing beginning increases in total marrow cellularity. Rosenthal<sup>15</sup> also demonstrated the striking thrombocytopenia in rabbits subjected to X irradiation over a range of 200 to 1000 r. This was associated with diminished clot retraction. However, in the rabbit as contrasted with other species no conspicuous hemorrhagic response was seen. They found no evidence of circulating anticoagulants.

Jackson<sup>16</sup> has reported that the abnormal blood coagulation, spontaneous purpura, and thrombocytopenia occurring following 600 r total body X irradiation in dogs were closely related. They observed normal prothrombin times and failed to find any conclusive evidence for a circulatory anticoagulant substance. They concluded that the post-irradiation clotting defect is largely due to the observed decrease in the number of circulatory platelets. Cronkite<sup>17</sup> in observations on goats and swine exposed at the atomic bomb tests at Bikini attributes the hemorrhagic syndrome to a

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combination of a vascular defect plus the thrombocytopenia. They found prolonged whole blood coagulation times only in fatally irradiated animals. Certain observations in some animals suggested heparinemia but it was not a consistent or convincing finding.

It has been concluded by Cronkite<sup>18</sup> from this large body of observations that the effect of the X irradiation in sufficient dosage is mediated through the destruction of practically all megakaryocyte activity and platelet production. With the known limited lifespan of platelets these rapidly disappear from the peripheral blood. Their reappearance in the blood in the second and third weeks has been shown to be correlated with the reappearance of megakaryocytes in the bone marrow.

In contrast to the group of animals receiving 500 r, the animals receiving irradiation at the dosage level of 100 r show a brief elevation on Days 1, 3, and 5 of the platelet count, suggesting increased release of platelets followed later by a brief period (Days 8 and 11) of depression of platelet levels. This would be expected from the degree of bone marrow depression produced by 100 r total body X irradiation. The platelet levels promptly return to normal levels in the subsequent period (Days 15, 21 and 30). Again, in general the changes in Lee-White clotting time and heparin clotting time mirror in an inverse degree the changes in level of circulating platelets.

In the burned animals, no essential differences were observed in animals receiving the burns of two different degrees of severity. Both groups show an initial fall in platelets lasting 1 to 3 days followed by sustained elevation of blood platelet levels. This suggests that during or immediately after the burn process, platelets were being utilized and/or destroyed. Either this process is self-limited or, after this period, increased platelet production occurs to counteract this process. It cannot be excluded, but it seems unlikely, that in the initial period there may be a period of decreased platelet production. If this were so, the platelet count would not be expected to fall immediately after the burn, but rather after a several day "lag" period, as in the animals receiving 500 r irradiation. There are only limited studies of the changes in blood platelets following burns but it has been shown that thrombocytosis does occur frequently following surgical trauma, fractures, burns, and a variety of other traumata as reported by Tocantins.<sup>19</sup>

In the group of animals receiving both traumata, a combination of these effects is observed. In the initial 1 to 3 day observation period platelets show a fall which is approximately to the same level as in the animals receiving burns only. Subsequent to this period the animals show a pattern which is nearly identical with that of the animals receiving total body X irradiation alone. There is no evidence of a more rapid disappearance of platelets in those animals as compared with those receiving a comparable dose of total body X irradiation alone. This suggests again that,

beyond the third day, there is no increased utilization and/or destruction of platelets as a result of the burn. The evidence is inconclusive but suggests that in those animals surviving, the recovery process, which begins on or about Day 15, proceeds at approximately the same rate as in those animals receiving the comparable dose of total body X irradiation alone. It is of considerable interest that, despite the fact that the platelet levels are comparable in the X<sub>5</sub> and B<sub>2</sub>X<sub>5</sub> groups and in the X<sub>1</sub> and B<sub>1</sub>X<sub>1</sub> groups for given observation days between Days 5 and 21, the defect in blood coagulation as measured by the Lee-White clotting time and heparin clotting time is not as severe in the animals receiving the combined injury as in those receiving irradiation only. This finding cannot be readily explained. There are no reports in the literature of comparable studies of blood coagulation in animals receiving a combination of thermal burns and total body X irradiation.

On the basis of our findings the coagulation mechanism has shown no essential differences among these groups despite the observed<sup>1, 2, 3, 4</sup> increased mortality in the combined injury group as compared with the irradiated-only group. The mortality and survival pattern described by Alpen<sup>3, 4</sup> shows an enhanced mortality both in the initial 72 hr period following burning and in the later period when deaths from irradiation alone are expected. During both of these periods the mean levels of circulating platelets and mean clotting times are essentially identical in the combined injury and the irradiated animals.

Our observations of platelet counts and both modified Lee-White and heparin clotting times have been analyzed for degree of correlation. The calculated correlation coefficients are interpreted as showing no inherent advantage to the measurement of any one variable as a measure of defective blood coagulation. Since all these determinations are not technically difficult but do require careful attention to detail and accurate replication of technique, the use of any one or two of these measurements will supply the necessary information in any study of either irradiated and/or burned animals.

Our selection of these three particular measurements was made with a view to their possible practical use in the evaluation of casualties. It is apparent that they are not necessarily the best measures of the extent of post-irradiation coagulation defect. Both Rosenthal and Cohn have been impressed with the usefulness of electrical resistance measurements of blood. However, at present, this test must remain an investigative rather than a clinical laboratory tool. The heparin clotting time, which measures the prolongation of the clotting time produced by a measured amount of added heparin, is a more sensitive test than the standard or modified Lee-White clotting time, but is in the long run subject to the same errors, occurring as a result of poor venepuncture or faulty technique in measurement. This increased sensitivity sharply limits its usefulness as compared with the Lee-White clotting time because of its

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susceptibility to technical defects. In our hands, it was only slightly better correlated with the level of circulating platelets than the modified Lee-White clotting time.

The modified Lee-White (silicone) method gives a coagulation time which is definitely prolonged as compared with the standard test in ordinary glassware. This prolongation appears to result from the modification of the surface to which the blood specimen is exposed. Since by careful technique a fairly uniform surface is obtained and since this prolongation of the time increases the sensitivity of the test, we recommend the modified Lee-White clotting time as a useful method. It still is subject to the same errors inherent in the other measurements of clotting time due to faulty sampling technique.

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SUMMARY

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This study of the blood coagulation mechanism reports the findings in rats subjected to high intensity radiant energy thermal burns, total body X irradiation, and the combination of thermal burns and total body X irradiation, and includes observations of modified Lee-White clotting time, heparin clotting time, blood platelet concentration, and prothrombin time.

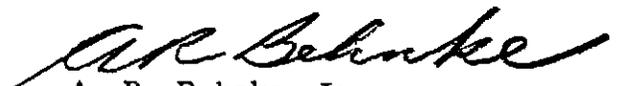
The total body X irradiation in a dose of 500 r alone produces a rapidly developing thrombocytopenia and defective clotting mechanisms as measured by Lee-White and heparin clotting times. At a dose level of 100 r a brief thrombocytosis is followed by a transient mild thrombocytopenia, which is reflected in changes in both the Lee-White and heparin clotting times.

The high intensity radiant energy thermal burns produce a brief fall in the level of circulating platelets followed by a sustained thrombocytosis.

The combined effects of thermal burns and total body X irradiation consist of the same initial fall in platelets seen in burns alone followed by the same degree of thrombocytopenia seen in animals receiving total body X irradiation at the particular dose given. In this pattern no significant differences are observed between the animals receiving the total body X irradiation alone and the combined thermal burn and X irradiation, with the exception that the coagulation defect, as measured by the Lee-White clotting time and heparin clotting time, is not as severe in the animals receiving the combined injury.

Despite the enhanced mortality seen in animals receiving the combined traumata it is apparent that the observed defective coagulation mechanism fails to account for this increased mortality. From a clinical point of view, however, the observed thrombocytopenia and defective clotting mechanism does permit the recognition of casualties in which thermal burns or other traumata is complicated by the occurrence of superimposed total body X irradiation and the degree of thrombocytopenia is correlated with the dose of total body X irradiation.

Approved by:



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For the Scientific Director

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TABLE 1

Platelet Counts of the Various Experimental Groups on the Different Days of Observation

Experimental Group <sup>(a)</sup>		Platelet Counts (no./cu mm x 10 <sup>-3</sup> ) on Various Days after Exposure							
		1	3	5	8	11	15	21	30
Control	Mean	741 <sup>(b)</sup>	741	737 <sup>(b)</sup>	703	782	845	851	771
	SD	107	73	131	210	71	131	137	104
X <sub>1</sub>	Mean	667	906	886	601	490	899	838	757
	SD	187	187	164	102	99	143	107	65
X <sub>5</sub>	Mean	803	738	380	22	8	87	383	719
	SD	124	164	98	32	12	62	315	166
B <sub>4</sub> X <sub>1</sub>	Mean	534	855	989	521	605	1,031	1,063	802 <sup>(b)</sup>
	SD	145	180	148	203	205	249	213	143
B <sub>2</sub> X <sub>5</sub>	Mean	645	641	319	29	28	91	557	618 <sup>(c)</sup>
	SD	205	73	84	26	31	173	408	110
B <sub>2</sub>	Mean	636 <sup>(b)</sup>	819	901	1,026	830	942	939	746
	SD	101	104	102	148	139	99	182	159
B <sub>4</sub>	Mean	664	631 <sup>(b)</sup>	1,000	947	1,048 <sup>(b)</sup>	1,027	935 <sup>(c)</sup>	926
	SD	112	136	143	159	214	234	111	183

(a) Six animals constituted each experimental group except where noted.

(b) Five animals in group.

(c) Four animals in group.

TABLE 2

Calculated Correlation Coefficients (r) for Different Determinations of Blood Coagulation

Determination	Entire Series (r)	Control Group (r)	B <sub>4</sub> Group (r)
Platelets and Lee-White C. T.	-0.757	-0.891	-0.877
Platelets and Heparin C. T.	-0.684	-0.899	-0.776
Lee-White C. T. and Heparin C. T.	+0.872	+0.930	+0.946

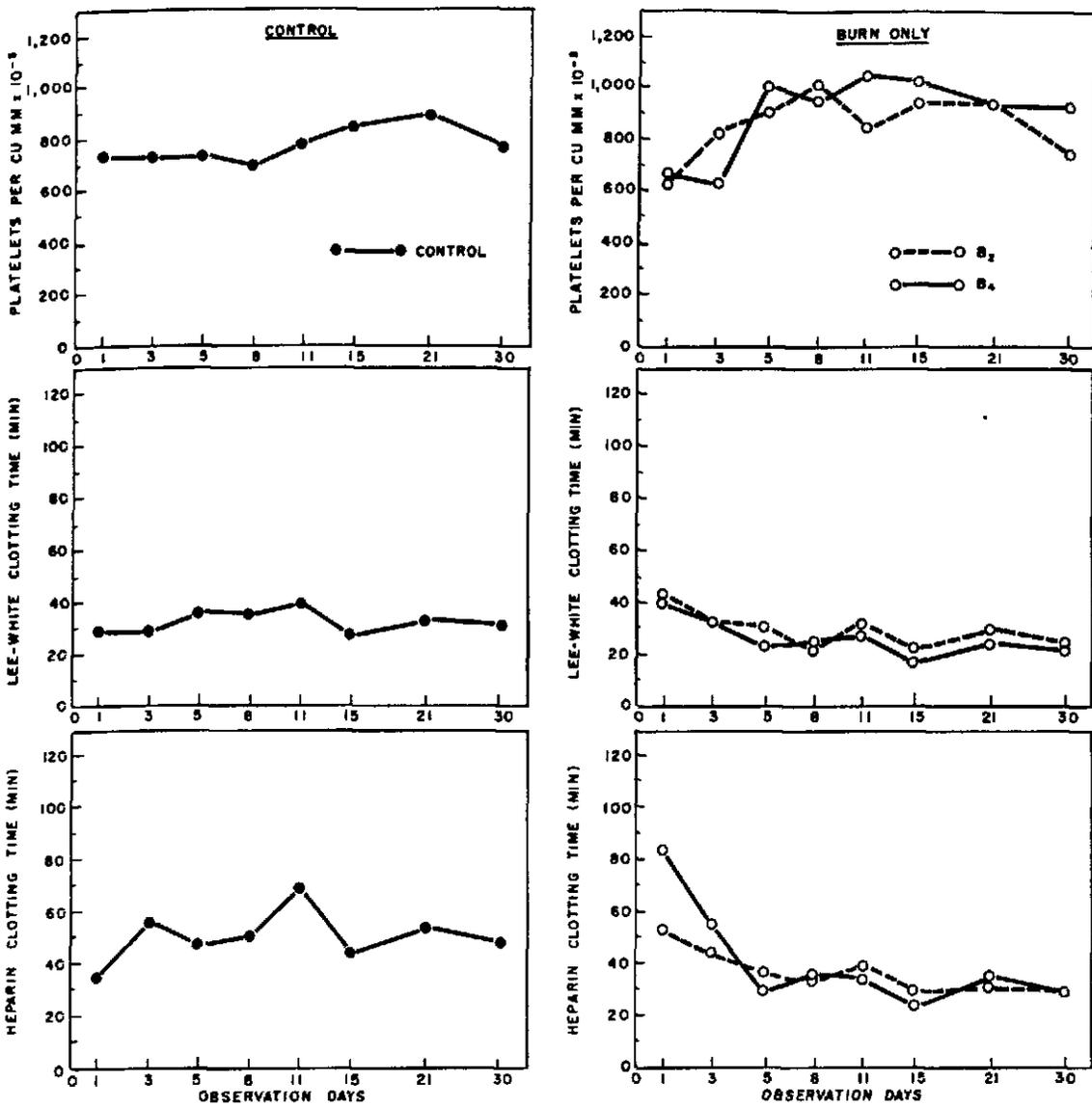


Fig. 1 Measurements of Blood Platelets, Lee-White Clotting Time, and Heparin Clotting Time in the Control Group and Burn Groups (B<sub>2</sub> and B<sub>4</sub>). Values are the mean of determinations for a given observation day

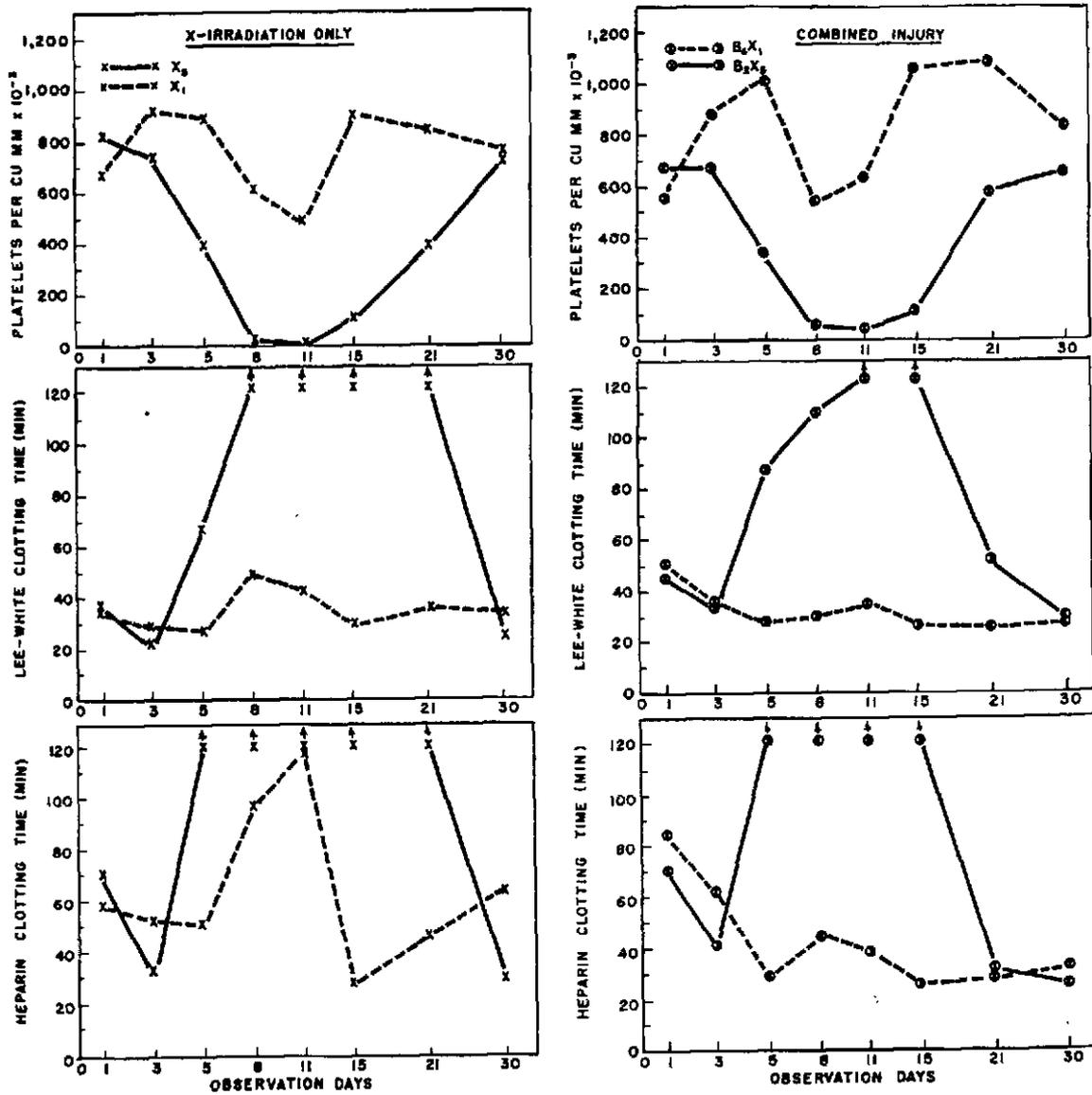


Fig. 2 Measurements of Blood Platelets, Lee-White Clotting Time and Heparin Clotting Time in the Total Body Irradiation Groups (X<sub>1</sub> and X<sub>5</sub>) and in the Combined Injury Groups (B<sub>4</sub>X<sub>1</sub> and B<sub>2</sub>X<sub>5</sub>). Values are the mean of determinations for a given observation day

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  3. Thermal radiation - Pathological effects
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The observed defective coagulation mechanism fails to account for the reported enhanced mortality seen in combined injury animals. However, the thrombocytopenia and defective clotting mechanism does permit the recognition of casualties in which burns or other traumata are complicated by the occurrence of superimposed total body X irradiation.

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