Hemodynamic and Metabolic Effects of Ringer's Lactate Solution in Hemorrhagic Shock

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RECENTLY, there has been considerable interest in the use of Ringer's lactate solution or buffered saline in the treatment of hemorrhagic shock and burns and for patients undergoing surgical procedures. Although it has been recognized for some time that after moderate hemorrhage a satisfactory blood volume could be restored by large volumes of isotonic saline solution,[11, 13, 14] the present enthusiasm for this approach to fluid therapy has been brought about by two developments. One was the finding by Shires and his group that functional extracellular fluid volume as defined by the rapidly equilibrating portion of the radiosulfate dilution curve was decreased both in hemorrhagic shock[16] and during and after operative procedures.[19] The other was the finding by a number of groups[4, 10, 18, 22] that survival in experimental hemorrhagic shock was improved by the use of Ringer's lactate solution or isotonic saline in addition to return of some or all of the shed blood. Fogelman and Wilson[6] also suggested the use of Ringer's lactate solution in addition to blood for traumatic shock. In addition, Pruitt *et al.*[12] demonstrated adequate maintenance of blood volume for twenty-four hours with buffered saline in normal, young men after 10–20% of blood volume was removed. In their studies, buffered saline was given in a replacement volume equaling four times the volume of red cells removed plus 1.3 times the volume of plasma removed. Of some concern in the replacement of large hemorrhages with non-red cell containing fluids is the reduced oxygen carrying capacity of the blood and its possible effects on cell metabolism. This was studied with dextran and blood by Drucker *et al.*,[5] who found that the metabolic responses to blood volume replacement with either was comparable both in degree and in rapidity of correction. The feasibility of the addition of lactate when blood lactate is already elevated has also been questioned. The following study was carried out to evaluate and compare the effects on blood flow, oxygen consumption and excess lactate production after hemorrhagic shock treated with Ringer's lactate solution alone, Ringer's lactate solution plus blood, and the shed blood alone.

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Method

Beagle type dogs weighing 8–14 Kg. were anesthetized with pentobarbital sodium, 25 mg./Kg., which was supplemented as necessary, and a cuffed endotracheal

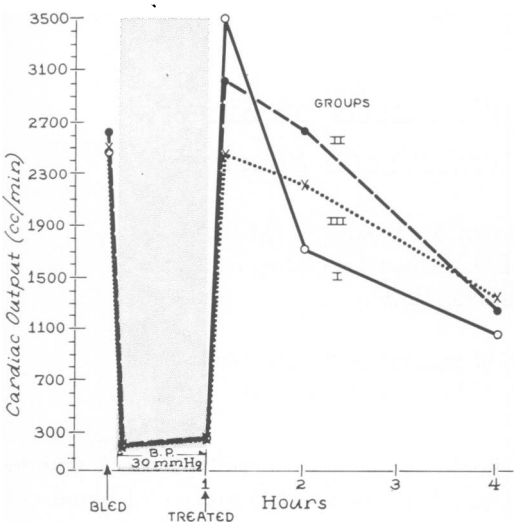

FIG. 1. Mean values of cardiac output (cc./min.).

tube was inserted. Both femoral arteries were cannulated: one for arterial pressure measurement with a Statham transducer and for arterial blood samples, and the other for bleeding into a heparinized plastic reservoir. A femoral vein was cannulated for fluid infusion. A catheter was inserted into the pulmonary artery via the external jugular vein for mixed venous blood samples. The endotracheal tube cuff was inflated and the tube connected to a Collins type spirometer filled with 100% O_2. The animal was allowed to breathe spontaneously from the spirometer. A soda-lime CO_2 absorber and circulating fan to eliminate dead space other than the endotracheal tube completed the system. Nitrogen washout was completed during an initial half hour control period. Anesthesia was kept at a light level so that arterial P_{CO_2} was within normal limits, and arterial P_{O_2} was usually well above 500 mm. Hg. Heparin, 5 mg./Kg. was given. The spirometer tracing, mean and pulsatile arterial pressure and pulmonary artery pressure were recorded on an Offner recorder. Continuous O_2 consumption was determined from the slope of the calibrated spirometer tracing. Arterial and mixed venous blood samples were analyzed for pH, P_{O_2} and P_{CO_2} with an Instrumentation Laboratory apparatus. Arterial and venous O_2 and CO_2 contents were determined by the method of Peters and Van Slyke. Blood lactate was determined by a modification of the enzymatic method of Horn and Bruns [8] and pyruvate was determined by a modified method of Segal et al.[15] Excess lactate was calculated by the formula of Huckabee.[9] Microhematocrits were measured on all blood samples. Cardiac output was calculated by the Fick principle from total O_2 consumption and arteriovenous O_2 content difference.

After control blood samples were drawn, each animal was bled into the reservoir at a rate of 5 cc./Kg./min. to a mean arterial pressure of 30 mm. Hg. This was main-

TABLE 1. *Average Cardiac Outputs ± 1 S.D.*

Condition	Group I Lactated Ringer's Alone			Group II Lactated Ringer's & ½ Shed Blood			Group III Shed Blood Alone		
	(cc./min.)	(cc./Kg.)	(% of control)	(cc./min.)	(cc./Kg.)	(% of control)	(cc./min.)	(cc./Kg.)	(% of control)
Before bleeding	2,482 (±618)	227 (±59)	100	2,635 (±328)	196 (±58)	100	2,490 (±627)	238 (±89)	100
60 min. at 30 mm./Hg	312 (±120)	28 (±12)	13	262 (±76)	19 (±3)	11	264 (±71)	22 (±24)	11
10 min. after fluids	3,564 (±1,377)	331 (±45)	145 (±49)	3,026 (±708)	231 (±49)	136 (±83)	2,462 (±703)	204 (±57)	95 (±38)
60 min. after fluids	1,734 (±837)	161 (±88)	71 (±30)	2,638 (±632)	203 (±49)	118 (±78)	2,237 (±775)	185 (±64)	85 (±34)
180 min. after fluids	1,079 (±403)	98 (±39)	45 (±14)	1,272 (±211)	98 (±18)	57 (±32)	1,370 (±286)	114 (±26)	54 (±21)

tained for one hour with the reservoir open. At the end of this time, further blood samples were drawn. According to our previous experience, this period of shock with this preparation should not produce irreversibility and survival should approach 100% if only the shed blood is returned. The animals were then divided into three groups. In Group I, six animals received Ringer's lactate solution in a volume determined by the hematocrit and volume of shed blood according to the formula developed by Moyer and associates.[4, 7] This equalled four times the volume of red cells lost and 1.3 times the plasma lost as shed blood into the reservoir. The Ringer's lactate solution, or Hartman's solution, was a commercially available preparation (Abbott Lab.) containing Na-130 mEq./L., K-4 mEq./L., Ca-3 mEq./L., Cl-109 mEq./L. and lactate 28 mEq./L. The pH was 6.5. The solution was given at a rate of 2.5 cc./Kg./min. with the average time of administration of one hour. Prior to the beginning of infusion, the reservoir was closed. Blood samples were drawn at 10 minutes, 1 hour and 3 hours after the end of the infusion. The cannulae were then removed, the vessels ligated, the incisions closed and the animal returned to its cage. Arterial blood was drawn for control lactate and pyruvate levels immediately after arterial cannulation because of the known effects of prolonged anesthesia and trauma on these values.

TABLE 2. *Oxygen Consumption in cc./Kg. ± 1 S.D.*

Condition	Group I L. R. Alone	Group II L. R.+1/2 Shed Blood	Group III Shed Blood Alone
Before bleeding	7.1 (±0.75)	6.3 (±0.74)	7.4 (±0.44)
60′ at 30 mm./Hg	4.4 (±1.27)	3.5 (±0.43)	3.7 (±0.69)
10′ after fluids	7.6 (±0.96)	7.0 (±0.72)	7.9 (±0.70)
60′ after fluids	7.4 (±1.30)	6.5 (±0.48)	7.9 (±1.18)
180′ after fluids	7.4 (±0.86)	7.4 (±1.58)	9.3 (±1.66)

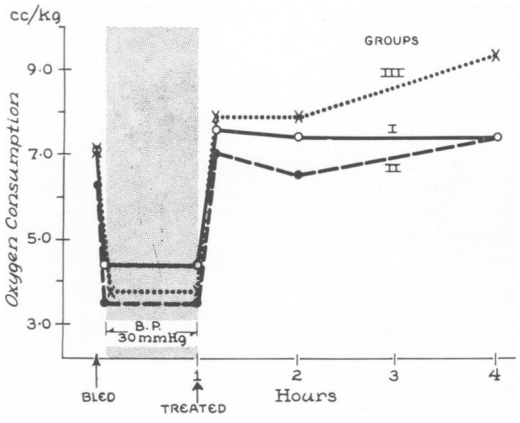

FIG. 2. Oxygen consumption.

The second group of six animals received Ringer's lactate solution in the same amount as determined for the Group I animals and at the same rate. Ten minutes after the Ringer's lactate infusion, blood samples were drawn. After this, one half of the shed blood was returned at a rate of 50 cc./min. Blood samples were drawn after 1 hour and 3 hours and the animals returned to their cages.

In the third group of six animals, after 1 hour of hypotension at 30 mm. Hg, the shed blood in the reservoir was returned at a rate of 50 cc./min. and blood samples were drawn at 10 minutes, 1 hour and 3 hours after the end of the infusion. The animals were then returned to their cages.

All animals were observed for 72 hours or longer, and if they survived for this period they were considered permanent survivors.

Results

The prebleeding control values were comparable for each of the three groups of animals. The amount of blood removed to maintain a pressure of 30 mm. Hg for an hour was 60.9 cc./Kg. ± 5.6 cc. for Group I, 64.1 cc./Kg. ± 4.3 cc. for Group II and 58.7 cc./Kg. ± 15.1 cc. for Group III. If blood volume is estimated to be

TABLE 3. *Mean Arterial Blood Pressure in mm./Hg ± 1 S.D. and Total Peripheral Vascular Resistance* $\frac{\text{mm./Hg Arterial Pressure}}{\text{Cardiac Output in L/Min}}$

Condition	Group I		Group II		Group III	
	B. P.	Resistance	B. P.	Resistance	B. P.	Resistance
Before bleeding	143 (±9.3)	60.3	149 (±14.8)	64.5	160 (±14.2)	62.2
60′ at 30 mm./Hg	30	107	30	120	30	122
10′ after fluids	98 (±9.8)	30.7	118 (±12)	40.6	132 (±16.2)	66.4
60′ after fluids	96 (±12.4)	61.8	130 (±15)	51.5	136 (±16.2)	66.4
180′ after fluids	103 (±25.4)	103	134 (±18.7)	106	137 (±15.9)	103

8% of body weight, the Group I animals bled an average of 76% of estimated blood volume, Group II, 78%, and Group III, 69%. In Group I, an average of 150 cc./Kg., or 1635 cc. of Ringer's lactate solution was given to each animal. In Group II, 161 cc./Kg., or 2,111 cc. were given. This equaled 2.46 times the volume of blood lost in Group I and 2.51 times the volume of blood lost in Group II.

Cardiac output (C.O.) changes are shown in Table 1 and Figure 1 as the means for the three groups. The decreases in C.O. at 30 mm./Hg were the same in each group. The increase 10 minutes after Ringer's lactate solution was greater and well beyond the control value for a short period of time in Groups I and II. At 60 minutes after infusion C.O. was higher in Group II, but this group received fluid in addition to blood. Although blood flow tended to be higher in Groups II and III at 180 minutes, these differences were not significant. C.O. in all groups had fallen to about half the control value at this time.

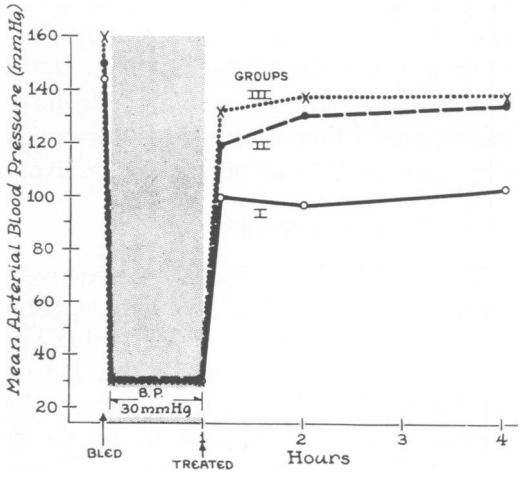

FIG. 3. Mean arterial blood pressure.

Mean changes in oxygen consumption for the three groups are shown in Table 2 and Figure 2. Oxygen consumption fell to the same degree in all three and returned to above the control values following vascular volume replacement regardless of the type of replacement. This increase beyond the control value could be considered indicative of repayment of an O_2 deficiency developed during the shock period. These increases beyond the control values were small, however, and were greatly influenced by small changes in the level of anesthesia late in the experiment. The high final value in Group III is believed to be due to lighter anesthesia in these animals at that time.

Blood pressure and peripheral vascular resistance changes are shown in Table 3 and indicate a higher blood pressure after

blood and Ringer's lactate solution plus blood than after Ringer's lactate solution alone (Fig. 3). At 180 minutes, blood pressure was below the control value in all groups. Peripheral vascular resistance increased to comparable levels in all groups during shock. It was decreased more by Ringer's lactate solution, but after 180 minutes there were no differences in the three groups.

Hematocrit changes are summarized in Table 4 and show considerable hemodilution with Ringer's lactate solution. At 180 minutes, hematocrit was still greatly reduced in Group I, was normal in Group II and was elevated in Group III (Fig. 4).

Arterial pH and blood gas tensions changes were much the same for each

TABLE 4. *Hematocrit Changes ± 1 S.D.*

Condition	Group I	Group II	Group III
Before bleeding	38.8±3.7	39.8±4.1	45.7±5.3
60′ at 30 mm./Hg	34.5±4.9	38.9±3.1	37.1±7.8
10′ after fluids	15.1±3.5	19.9±4.4	47.8±3.8
60′ after fluids	21.3±5.4	34.2±5.0	51.0±3.9
180′ after fluids	25.5±4.7	41.8±8.1	52.1±5.0

group (Table 5). The pH decreased to 7.04–7.07 after 60 minutes at 30 mm. Hg and rose progressively after fluid or blood replacement, reaching normal at 180 minutes in all animals (Fig. 5). It decreased initially after blood from the reservoir, but rose after the electrolyte solution. At 180 minutes, even though the pH was normal, there was persistent metabolic acidosis with respiratory compensation since arterial P_{CO_2} and CO_2 contents were decreased. Arterial P_{O_2} was maintained at high levels in all animals during the entire experiment. This ranged from 450–600 mm. Hg, average—550 mm. Hg. Arterial O_2 content varied with the hematocrit changes. There were marked decreases in O_2 content after hemodilution with Ringer's lactate solution. At 180 minutes, arterial O_2 content was 14 vol.% in Group I, 20 vol.% in Group II

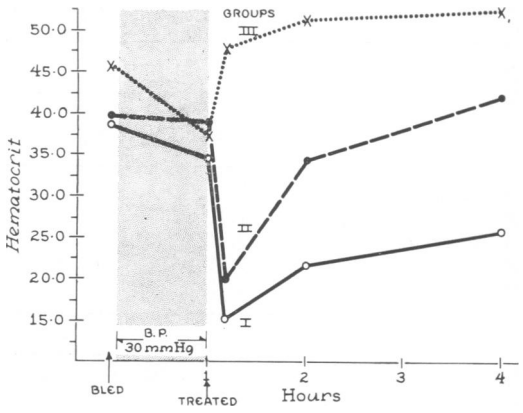

FIG. 4. Hematocrit.

and 25 vol.% in Group III. Venous O_2 content stayed extremely low in the Group I animals (4.5 vol.%) and returned to the control value in Groups II and III (14–17 vol.%) at 180 min. Arteriovenous O_2 differences were comparable for all groups under these varying conditions.

Blood lactate and pyruvate levels were normal immediately after anesthesia and did not change appreciably during the hour of preparation before bleeding. At the end of the shock period, lactate, pyruvate and excess lactate were considerably elevated. These decreased after fluid and blood infusion, but after 3 hours there was still some excess lactate in all groups along with compensated metabolic acidosis. No differences were noted in these measurements in the three groups (Table 6, Fig. 6).

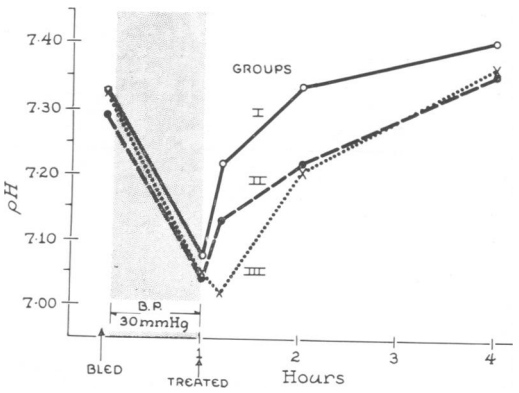

FIG. 5. pH change.

TABLE 5. *Acid-Base Data—Arterial Blood (P_{CO_2} in mm./Hg, CO_2 Content in Vol. %)*

Condition	Group I			Group II			Group III		
	pH	P_{CO_2}	CO_2 Content	pH	P_{CO_2}	CO_2 Content	pH	P_{CO_2}	CO_2 Content
Before bleeding	7.33	44	50	7.29	45	48	7.33	39	43
60′ at 30 mm./Hg	7.07	26	18	7.04	25	17	7.04	19	15
10′ after fluids	7.22	34	35	7.13	38	31	7.02	48	35
60′ after fluids	7.34	35	39	7.22	39	35	7.21	38	32
180′ after fluids	7.41	26	34	7.36	29	36	7.37	30	33

All animals in Groups I and II survived beyond 72 hours and until sacrificed. One animal in Group III died. This animal had a satisfactory blood pressure and cardiac output at the end of the period of study but developed the highest excess lactate during the shock period and the lowest arterial pH and CO_2 content. Excess lactate did not return toward normal after treatment as it did in other animals. This was also the only animal to take blood back spontaneously from the reservoir during the hour at 30 mm. Hg.

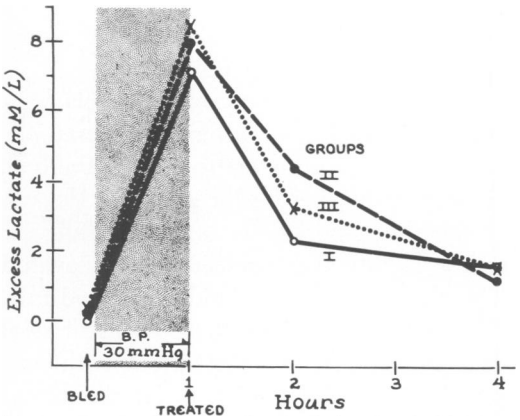

FIG. 6. Excess lactate in mM./L.

Discussion

The shock preparation used in the present study was designed to produce severe and prolonged shock, but treatment was begun before the time of predicted irreversibility. By this means, it was hoped that the problems of hepatosplanchnic pooling of blood and gastrointestinal bleeding which are specific to the dog in irreversible shock might be avoided. All animals except one survived and were normal when sacrificed after 72 hours. The single death occurred in an animal which received only its shed blood, but it took blood from the bleeding reservoir prior to treatment, indi-

TABLE 6. *Blood Lactate, Pyruvate and Excess Lactate. In mMol./L. ± 1 S.D. (mMol./L. × 9 = mg. % approximately)*

Condition	Group I			Group II			Group III		
	Lactate	Pyruvate	Excess Lactate	Lactate	Pyruvate	Excess Lactate	Lactate	Pyruvate	Excess actate
After anesthesia	0.85 (±0.25)	0.049 (±0.01)	0.0	0.8 (±0.23)	0.055 (±0.01)	0.0	0.72 (±0.18)	0.042 (±0.01)	0.0
Before bleeding	0.69 (±0.86)	0.033 (±0.01)	0.002	0.73 (±0.13)	0.728 (±0.01)	0.15	1.39 (±0.8)	0.134 (±0.17)	0.306
60′ at 30 mm./Hg	9.8 (±2.5)	0.135 (±0.003)	7.24 (±0.76)	10.6 (±1.1)	0.134 (±0.12)	8.7 (±1.3)	10.8 (±3.5)	0.122 (±0.01)	8.56 (±2.4)
60′ after fluids	3.8 (±1.2)	0.059 (±0.03)	2.4 (±1.15)	4.18 (±1.35)	0.058 (±0.05)	4.42 (±1.8)	4.66 (±1.7)	0.05 (±0.03)	3.31 (±1.1)
180′ after fluids	2.48 (±1.1)	0.045 (±0.01)	1.65 (±1.10)	1.52 (±0.84)	0.015 (±0.04)	1.29 (±0.6)	2.1 (±1.25)	0.024 (±0.01)	1.67 (±1.07)

cating circulatory decompensation prior to treatment.

The replacement of vascular volume with buffered saline (Ringer's lactate solution) alone, buffered saline plus blood or blood alone produced much the same levels of cardiac output and oxygen consumption after three hours and the same progressive correction of metabolic acidosis and excess lactate. This confirms the fact that vascular volume restoration by a non-specific fluid without red cells which satisfactorily increases cardiac output will improve oxidative metabolism and correct the metabolic defects of shock prior to irreversibility. This can be accomplished with an electrolyte solution when sufficient volume is given to maintain a large quantity of the solution in the vascular compartment. Colloid is not necessary for this purpose, although Drucker et al.[5] have shown that dextran was as effective as blood in restoring vascular volume and correcting the altered metabolism of shock. This indicates that Ringer's lactate solution alone and in sufficient quantity may be satisfactory for moderate blood loss and is being used clinically by a number of groups.[1, 17, 20] Obviously, progressive hemodilution with reduced oxygen carrying capacity of arterial blood would become detrimental. The limits to which this can be extended have not been definitely established. In the present study, 60 cc. of blood/Kg. of body weight (estimated 73% of blood volume) was replaced with Ringer's lactate solution without known deleterious effects over the next three hours, and all animals survived. However, arterial O_2 content was reduced and O_2 consumption was maintained by greater O_2 extraction from the blood with very low mixed venous O_2 contents. This was definitely an abnormal state not present in animals receiving some or all of the shed blood. This allows the animal no reserve and makes it extremely vulnerable to any further insult. Decreased arterial O_2 saturation, decreased cardiac output or preexisting pulmonary or cardiac disease would produce tissue hypoxia with such a low venous O_2. Thus, with this extent of hemorrhage, red cells seem necessary not to correct the altered metabolism, because this can occur without red cells, but to restore the normal oxygenation capabilities of the organism. Drucker[5] found that replacement of 48 cc. of blood/Kg. of body weight with dextran (hematocrit 14–15) did not produce excess lactate in the next 9 hours. Wise *et al.*[21] found that in hemorrhagic shock in dogs treated with dextran or plasma alone, the compensatory mechanisms were rapidly exhausted when the hemoglobin fell to less than 50% of control. Tissue hypoxia then became evident.

The use of such a solution can decrease the volume of blood replacement necessary without sacrificing homeostatic capabilities. This is of interest in civilian defense planning for major disasters when blood in adequate amounts may not be available. The preparation, stockpiling and storage of materials for major disasters could be simplified with electrolyte solutions as compared to colloids.

Reynolds[13] demonstrated in 1949 that saline was effective in the treatment of hemorrhagic shock, and Parkins *et al.*[11] found that moderate hemorrhages could be replaced adequately with isotonic saline in volumes of two or more times the volume of blood lost. These animal studies have now been confirmed in human volunteers from whom 10% or 20% of blood volume was removed and replaced by Ringer's lactate solution in volumes four times the red cells and 1.3 times the plasma removed.[12] Vascular volume was maintained for at least 24 hours. Other studies have shown that survival in critical hemorrhagic shock in dogs can be improved by giving buffered saline or dextran in addition to part or all of the shed blood. Wolfman *et al.*[22] and Shires *et al.*[18] treated dogs in hemorrhagic shock with buffered saline in a volume of 5% of body weight and then returned all

the shed blood, increasing survival to 80–90% from the 20–30% survival with blood return alone. Dillon *et al.*[4] found the ideal replacement solution to be Ringer's lactate solution (buffered to a pH of 8.2 with NaOH) in a volume of four times the volume of red cells plus the volume of plasma lost in addition to half the shed blood. The reasons for this improvement in mortality have been ascribed to an increased extracellular fluid volume,[22] replacement of the functional extracellular fluid deficit produced by shock,[18] or replenishment of the functiontal sodium mass of the body.[4] Hemodilution with reduced blood viscosity and better flow characteristics of blood would also seem to play a part. In the present study there were differences in the three groups of animals at three hours after treatment in blood pressure, hematocrit and arterial and venous blood O_2 contents. In animals given only Ringer's lactate solution, the hematocrit was reduced to 40% of control immediately after infusion and was only 66% of control after 3 hours. In animals given buffered saline and blood, the hematocrit was normal after three hours, and animals given only blood were hemoconcentrated at that time. The importance of a normal hematocrit has been stressed by Crowell,[2, 3] who found that survival time and oxygen consumption were maximal in dogs with hemorrhagic shock when the hematocrit was in a normal range. Some immediate alkalinization of arterial blood was produced by the Ringer's lactate solution, and correction of excess lactate was not hampered by the lactate load. Blood pressure was lowest in animals given only buffered saline but calculated vascular resistance was the same in all three groups. Cardiac output increased well beyond the control value for a short time after Ringer's lactate solution infusion but just reached the control value after blood. This greatly increased cardiac output compensated for the hemodilution and reduced arterial O_2 content so that O_2 consumption was the same in all groups. Since the correction of altered metabolism proceeded on the same time scale with all three groups, any presumed beneficial effect of buffered saline plus blood in the treatment of shock would seem to be related to better flow characteristics of blood. This is evidenced by the larger cardiac output immediately after Ringer's lactate solution and the normal hematocrit when blood is also given. Volume for volume, a non-red cell containing solution which stays in the vascular space produces greater increases in blood flow than does whole blood, presumably because of the decrease in blood viscosity with hemodilution. It is possible that these alterations would be different at a critical period of irreversibility in shock, but this would also introduce problems peculiar to the dog. It is also possible that the beneficial effects of buffered saline and blood in previous studies are related to individual organ functions which were not measured in the present study. In Drucker's studies [5] comparing the metabolic effects of blood, dextran and saline, an irreversible preparation was used in which none of the animals survived. They found that the restoration of blood volume with a crystalloid solution (normal saline) in a volume equal to twice the volume of blood lost was less effective than blood or colloid (dextran) in improving oxidative metabolism or cardiovascular status. In the present study, Ringer's lactate solution was given in a volume approximately 2.5 times the volume of shed blood. This plus the non-irreversible stage of shock would seem to be responsible for the effectiveness of buffered saline in the present study.

The lag phase of correction of metabolic acidosis and decrease in excess lactate after the treatment of shock is again noted. Metabolic acidosis and excess lactate, although slight, were still present 3 hours after volume replacement. This may be related to the time interval required for the liver to handle a lactate load.

This study supports the clinical use of Ringer's lactate solution along with blood for the treatment of hypovolemic shock. It should be helpful not only in the initial emergency treatment of shock before blood is available but also along with blood to decrease the volume of blood necessary and for the other advantages which it may offer. The present report applies only to hypovolemic shock and not to other causes of hypotension. The study was carried out under controlled conditions with adequate ventilation and with the animal breathing 100% oxygen, in contrast to clinical situations with many uncontrollable variables. The potential danger of excessive hemodilution with reduced oxygen carrying capacity of the blood despite high blood flow or cardiac output must be recognized.

Summary

Hemorrhagic shock in three groups of dogs was treated prior to irreversibility by Ringer's lactate solution alone, Ringer's lactate solution plus half the shed blood, or blood alone. Measurements included cardiac output, oxygen consumption, blood gas tensions and contents, pH, hematocrit and lactate, pyruvate, and excess lactate levels. The total body metabolic deficit was adequately corrected by all three treatment programs, indicating that an electrolyte solution given in sufficient volume was satisfactory in this respect for the treatment of severe hypovolemic shock. However, when Ringer's lactate solution was used alone to replace a large blood loss, normal O_2 consumption was maintained only by increased cardiac output and increased O_2 extraction with a low venous O_2 content. This would make the animal vulnerable to any further insult. Ringer's lactate solution given with blood decreased the volume of blood required in hemorrhagic shock to provide adequate cardiac output, O_2 consumption and blood gas contents along with correction of altered metabolism. Ringer's lactate solution, in the volumes and rate given, transiently increased cardiac output beyond the control value, whereas blood did not. The buffered saline provided some immediate correction of metabolic acidosis, but 3 hours after treatment there were no significant differences in any of these measured parameters in the three groups. Animals given Ringer's lactate solution plus half the shed blood had a normal hematocrit at the end of the study. Previously described beneficial effects of electrolyte solutions in addition to blood as compared to blood alone in the treatment of hemorrhagic shock seem to be due in part to better flow characteristics of blood with hemodilution.

References

1. Boba, A.: Support of Blood Volume During Operation without Blood Transfusion. Surg. Forum, 17:61, 1966.
2. Crowell, J. R., Bounds, S. H. and Johnson, W. W.: Effect of Varying Hematocrit Ratio on the Susceptibijlity to Hemorrhagic Shock. Amer. J. Physiol., 192:171, 1958.
3. Crowell, J. R., Ford, R. G. and Lewis, V. M.: Oxygen Transport in Hemorrhagic Shock as a Function of the Hematocrit Ratio. Amer. J. Physiol., 196:1033, 1959.
4. Dillon, J., Lynch, L. J., Jr., Myers, R. and Butcher, H. R., Jr.: The Treatment of Hemorrhagic Shock. Surg. Gynec. Obstet., 122: 967, 1966.
5. Drucker, W. R., Holden, W. D., Kingsbury, B., Hofmann, N. and Graham, L.: Metabolic Aspects of Hemorrhagic Shock; II. Metabolic Studies on the Need for Erythrocytes in the Treatment of Hypovolemia Due to Hemorrhage. J. Trauma, 2:567, 1962.
6. Fogelman, M. J. and Wilson, B. J.: A Different Concept of Volume Replacement in Traumatic Hypovolemia. Amer. J. Surg., 99: 694, 1960.
7. Grayson, T. L., White, J. E. and Moyer, C. A.: Oxygen Consumptions; Concentrations of Inorganic Ions in Urine, Serum and Duodenal Fluid, Hematocrits, Urinary Excretions; Pulse Rates and Blood Pressure During Duodenal Depletions of Sodium Salts in Normal and Alcoholic Man. Ann. Surg., 158:840, 1963.
8. Horn, H. D. and Bruns, F. H.: Quantitative Determination of L-(+) Lactic Acid with Lactic Dehydrogenase. Biochim. Biophys. Acta, 21:378, 1956.
9. Huckabee, W. E.: Relationships of Pyruvate and Lactate During Anaerobic Metabolism. I. Effects of Infusion of Pyruvate or Glucose and of Hyperventilation. J. Clin. Invest., 37: 244, 1958.
10. McPherson, R. C. and Haller, J. A.: The Comparative Effects of Blood, Saline and Low

Molecular Dextran on Irreversible Hemorrhagic Shock. J. Trauma, **4**:415, 1964.
11. Parkins, W. M., Perlmutt, J. H. and Vars, H. M.: Evaluation of Crystalloidal Solutions in Hemorrhaged Dogs. Amer. J. Physiol., **170**:351, 1952.
12. Pruitt, B. A., Jr., Moncrief, J. A. and Mason, A. D., Jr.: Efficacy of Buffered Saline as the Sole Replacement Fluid Following Acute Measured Hemorrhage in Man. J. Trauma, (In Press.)
13. Reynolds, M.: Cardiovascular Effects of Large Volumes of Isotonic Saline Infused Intravenously into Dogs Following Severe Hemorrhage. Amer. J. Physiol., **158**:418, 1949.
14. Rosenthal, S. M. and Tabor, H.: Electrolyte Changes and Chemotherapy in Experimental Burn and Traumatic Shock and Hemorrhage. Arch. Surg., **51**:244, 1945.
15. Segal, S., Blair, A. E. and Wyngaarden, J. B.: An Enzymatic Spectrophotometric Method for the Determination of Pyruvic Acid in Blood. J. Lab. Clin. Med., **48**:137, 1956.
16. Shires, T., Brown, F. T., Canizaro, P. C. and Sommerville, N.: Distributional Changes in Extracellular Fluid During Acute Hemorrhagic Shock. Surg. Forum, **11**:115, 1960.
17. Shires, G. T., Carrico, C. J. and Coln, D.: The Role of the Extracellular Fluid in Shock. Int. Anesth. Clin., **2**:435, 1964.
18. Shires, T., Coln, D., Carrico, J. and Lightfoot, S.: Fluid Therapy in Hemorrhagic Shock. Arch. Surg., **88**:688, 1964.
19. Shires, T., Williams, J. and Brown, F.: Acute Change in Extracellular Fluids Associated with Major Surgical Procedures. Ann. Surg., **154**:803, 1961.
20. Trudnowski, R. J.: Hydration with Ringer's Lactate Solution. JAMA, **195**:545, 1966.
21. Wise, W., Head, R. L., Morse, M. and Allen, G. J.: The Physiological Effects of Acute Anemia Produced by the Replacement of Serial Hemorrhage with Dextran, Plasma, and Whole Blood. Surg. Forum, **8**:18, 1958.
22. Wolfman, E. F., Neill, S. A., Heaps, D. K. and Zuidema, G. D.: Donor Blood and Isotonic Salt Solution. Arch. Surg., **86**:869, 1963.