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Evaluation of a hypertonic saline-dextran solution for treatment of dogs with shock induced by gastric dilatation-volvulus

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Objective—To test the hypothesis that small volumes of hypertonic saline-dextran (HSD) solution can be used to effectively resuscitate dogs in shock induced by gastric dilatation-volvulus (GDV), and, compared with administration of large volumes of lactated Ringer's solution (LRS), can be used to limit the overall volume of fluid needed for resuscitation.

Design—Prospective, clinical study.

Animals—15 dogs with GDV-induced shock.

Procedure—Initially, HSD solution (5 ml/kg of body weight) or LRS (60 to 90 ml/kg) was administered. All dogs then received a maintenance administration (20 ml/kg/h) of LRS. Cardiorespiratory, blood gas, and serum biochemical analyses were performed over a 4-hour period after initiation of treatment.

Results—Systolic arterial and central venous pressures and plasma volume increased more rapidly in dogs in the HSD + LRS group. The cumulative dose of fluids administered to dogs in the HSD + LRS group was significantly less than that administered to dogs in the LRS group. Serum sodium and chloride concentrations and osmolality increased significantly in dogs in the HSD + LRS group, but not in dogs in the LRS group. Ventricular arrhythmias were detected in both groups of dogs, but did not appear to be induced by either form of fluid therapy.

Clinical Implications—Administration of HSD rapidly restored cardiorespiratory function and induced resuscitation equivalent to administration of large volumes of LRS. Use of HSD solutions to treat dogs in GDV-induced shock may be more efficient than use of isotonic fluids. Administration of HSD solution was not associated with noticeable complications. (*J Am Vet Med Assoc* 1997;210:226–230)

The principal goals of preoperative treatment of dogs in shock induced by **gastric dilatation-volvulus (GDV)** are to decompress the distended stomach to improve venous return and gastric perfusion and to expand blood volume to optimize cardiorespiratory function.[1,2] The latter goal is typically accomplished by IV administration of large volumes of isotonic 0.9% NaCl solution or **lactated Ringer's solution (LRS)**. The rationale for the experimental and clinical use of hypertonic saline solutions has been that these solutions require only a small volume of fluid to achieve rapid and effective resuscitation.[3,4] Given the predisposition for GDV to develop in large- and giant-breed dogs, a substantial advantage may be gained by the use of small volumes of hypertonic NaCl solutions for resuscitation of dogs with GDV-induced shock.[1] In an investigation comparing the use of small volumes of **hypertonic saline (7% NaCl solution) in 6% dextran 70 (HSD)** to large volumes of 0.9% NaCl solution for treatment of GDV-induced shock in dogs, initial resuscitation with HSD solution followed by a continuous maintenance infusion of isotonic fluid produced a more-sustained hemodynamic response and required less cumulative fluid volume than administration of large volumes of isotonic NaCl solution.[1] Untoward effects of the HSD-induced resuscitation were not observed. Furthermore, numerous studies on traumatic,[5,6] hemorrhagic,[7-10] and endotoxemic[11] shock in dogs support the use of hypertonic NaCl and NaCl-dextran solutions as methods of fluid resuscitation. Clinical trials in dogs[12,13] and human beings[14-16] further support the therapeutic efficacy of hypertonic NaCl solution for treatment of shock.

We performed a prospective clinical study to test the hypothesis that small volumes of HSD solution can be used to effectively resuscitate dogs in GDV-induced shock, and, when compared with administration of large volumes of LRS, can be used to effectively limit the overall volume of fluid necessary for resuscitation.

Materials and Methods

Experimental design—Fifteen dogs in which GDV-induced shock was diagnosed were entered into the study. Gastric dilatation-volvulus was diagnosed by a combination of signalment, clinical history, physical findings, and abdominal radiography. Shock was defined when dogs had 4 or more of the following 8 cardiorespiratory system and physical abnormalities: systolic arterial pressure < 120 mm of Hg (as measured by ultrasonic Doppler flow detector); heart rate > 120 beats/min; respiratory rate > 30 breaths/min; capillary refill time > 2 seconds; white, gray, or bright red mucous membranes; cold limbs or rectal temperature < 37.8 C; dog was recumbent or weak; and pulses were weak or thready. Dogs with severe dehydration (> 8%), coagulopathy, and that were pregnant or had a history of heart failure, renal disease, or any other major medical problem were excluded from the study.

Eligible dogs were alternately assigned to receive sterile HSD solution or sterile LRS as initial fluid therapy. Veterinarians treating these dogs were advised to initially administer the HSD solution at a dosage of 5 ml/kg of body weight over a 5-minute period or to administer the LRS at a dosage of 60 to 90 ml/kg over a 60-minute period. It was recommended that maintenance fluids (ie, LRS) subsequently be administered at a dosage of 20 ml/kg/h. Veterinarians were

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restricted in terms of the type of crystalloid fluid administered, but were free to adjust the volume of fluid necessary to adequately resuscitate the dogs.

Evaluation of response—Gastric decompression by use of a trocar or stomach tube, catheterization of a jugular vein, electrocardiography, and collection of a sample of venous blood were performed prior to treatment. A pressure cuff was positioned over the median artery or dorsal pedal artery for measurement of systolic arterial pressure, using an ultrasonic Doppler flow detector.[a] The following variables were measured prior to treatment (baseline values) and at 15, 60, 120, and 240 minutes after initiation of fluid therapy: heart rate, central venous pressure, systolic arterial pressure, rectal temperature, and respiratory rate. Body weight was measured prior to treatment. Heparinized venous blood samples were collected prior to treatment and at 15, 60, and 240 minutes after initiation of fluid administration for determination of PCV, Pv_{O_2}, pH, and base deficit. Serum samples were obtained at the same times for measurement of albumin, total protein, sodium, chloride, and potassium concentrations, and osmolality.

The initial volume of HSD solution or LRS administered was recorded. After the initial treatment, the cumulative volumes of isotonic fluids administered were recorded at 60, 120, 180, and 240 minutes.

Statistical analysis—Variables involving repeated measures were analyzed with a multivariate repeated-measures ANOVA. When significant ($P < 0.05$) group or time interactions were observed, additional testing was performed, using Dunnett's test and Tukey's test for determining differences within groups and between groups, respectively. Variables measured once during the study were evaluated for between-group differences, using a *t*-test. All data were reported as the mean ± SEM.

Results

Animals—Mean age of dogs in the HSD + LRS and LRS groups was 6.9 ± 2.0 and 5.9 ± 0.9 years, respectively. Mean weight of dogs in the HSD + LRS and LRS groups was 36.9 ± 5 and 41 ± 6 kg, respectively. All dogs underwent surgery for evaluation of GDV, decompression, repositioning, and fixation of the stomach. In the LRS group, 2 dogs were euthanatized during surgery, and 1 dog died 6 hours after surgery. Two dogs in the HSD + LRS group were euthanatized during surgery. Gastric necrosis was judged to be extensive in the dogs that were euthanatized.

Baseline values—Most of the values obtained during baseline measurements did not differ significantly between the treatment groups in the study reported here (Tables 1 and 2). Heart rate was significantly higher in the HSD + LRS group during baseline measurements (190 ± 13 beats/min) than in the LRS group (147 ± 12 beats/min); however, heart and respiratory rates were increased over reported reference ranges[17,18] for both groups. Systolic arterial pressure was at the low end of the reference range in both groups. Mean central venous pressure was less than the reference range in both groups. The pH of venous blood was less than the reference range in the HSD + LRS group, but not in the LRS group; however, the values were not significantly different.

Response to treatment—Mean amount of fluid administered during the first hour to dogs in the HSD + LRS group (23 ± 4 ml/kg) was approximately equal to the dosage of HSD (5 ml/kg) and LRS (20 ml/kg/h) recommended in the study protocol, whereas the mean amount of fluid administered during the first hour to dogs in the LRS group (53 ± 6 ml/kg) was somewhat less than the dosage (60 to 90 ml/kg/h) suggested in the protocol (Fig 1). The cumulative volume of fluid received by dogs in the HSD + LRS group at each period of measurement was significantly less than that received by dogs in the LRS group. Mean total amount of fluid administered to dogs in the HSD + LRS group after 4 hours (54 ± 13 ml/kg) was significantly less than that administered to dogs in the LRS group (90 ± 11 ml/kg).

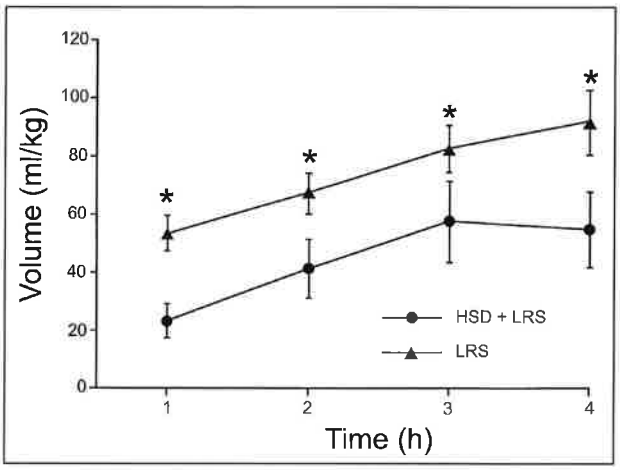

Figure 1—Cumulative volume of fluids administered to 2 groups of dogs for treatment of shock resulting from gastric dilatation-volvulus. Dogs initially received 7% NaCl in 6% dextran 70 and lactated Ringer's solutions (HSD + LRS) or lactated Ringer's solution (LRS) alone. *Volume administered differs significantly ($P < 0.05$) between groups.

Cardiorespiratory responses of the 2 groups to fluid administration were similar (Table 1). Heart rate decreased from the baseline values in dogs in both groups, but mean heart rate was significantly lower only in the HSD + LRS group. Respiratory rate decreased similarly in both groups, though the change was only significantly different at 15 minutes in the HSD + LRS group. Systolic arterial pressure in dogs in the HSD + LRS group was increased significantly from baseline at 15 and 240 minutes after onset of treatment. Systolic arterial pressure did not increase significantly from baseline values in dogs in the LRS group until 240 minutes after initiation of treatment. Central venous pressure in dogs in the HSD + LRS group increased significantly from baseline values 15 minutes after the onset of treatment and stayed that way for the remainder of the study. Central venous pressure in dogs in the LRS group did not increase significantly from baseline values until 60 minutes after onset of treatment (Table 2).

Total plasma protein and albumin concentrations in dogs in the HSD + LRS group were decreased significantly from baseline values at 15, 60, and 240 minutes after onset of treatment (Table 2). Total plasma protein concentration, but not albumin concentration, decreased significantly in the LRS group.

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Table 1—Cardiovascular and respiratory function (mean ± SEM) in dogs in shock resulting from gastric dilatation-volvulus that were initially treated with 7% NaCl in solution 6% dextran 70 and lactated Ringer's solution (HSD + LRS) or that initially received only lactated Ringer's solution (LRS)

Variable	Reference range	Group	Before treatment	Time after initiation of treatment (min) 15	60	120	240
Systemic arterial pressure (mm of Hg)	100–150	HSD + LRS	124 ± 21	169 ± 14*	119 ± 15	115 ± 18	199 ± 21*
		LRS	134 ± 15	138 ± 15	136 ± 17	152 ± 22	181 ± 19
Central venous pressure (cm of H_2O)	0.0–3.0	HSD + LRS	−0.7 ± 1.2	3.5 ± 1.3*	4.2 ± 1.6*	4.3 ± 1.4*	6.1 ± 2.0*
		LRS	−1.1 ± 1.4	0.0 ± 2.2	4.0 ± 1.8*	5.6 ± 1.6*	4.8 ± 1.1*
Heart rate (beats/min)	70–120	HSD + LRS	190 ± 13	144 ± 6*	138 ± 7*	157 ± 9	166 ± 18
		LRS	147 ± 12	117 ± 11	149 ± 6	147 ± 5	123 ± 9
Respiratory rate (breaths/min)	10–30	HSD + LRS	43 ± 6	25 ± 3*	30 ± 8	27 ± 7	29 ± 7
		LRS	38 ± 9	32 ± 6	31 ± 5	30 ± 4	28 ± 7
Temperature (C)	37.5–39.2	HSD + LRS	38.6 ± 0.2	38.3 ± 0.3	37.3 ± 0.6	37.1 ± 0.3*	37.4 ± 0.3*
		LRS	37.8 ± 0.6	37.9 ± 0.6	37.1 ± 0.6	37.0 ± 0.6*	37.4 ± 0.6

*Within each group, values differ significantly ($P < 0.05$) from baseline values.

Table 2—Results (mean ± SEM) of serum biochemical analyses for dogs in shock resulting from gastric dilatation-volvulus that were treated initially with HSD ± LRS or LRS alone

Variable	Reference range	Group	Before treatment	Time after initiation of treatment (min) 15	60	240
Total protein (g/dl)	5.4–7.8	HSD + LRS	6.8 ± 0.2	5.0 ± 0.3*	4.6 ± 4.0*	4.8 ± 0.6*
		LRS	6.1 ± 0.6	4.9 ± 0.7*	4.6 ± 0.6*	4.6 ± 0.7*
Albumin (g/dl)	2.2–3.4	HSD + LRS	2.9 ± 0.1	2.0 ± 0.1*	1.9 ± 0.1*	1.7 ± 0.2*
		LRS	2.4 ± 0.3	2.0 ± 0.2	1.8 ± 0.2	1.8 ± 0.2
PCV (%)	39–56	HSD + LRS	47 ± 2	32 ± 2*	33 ± 1*	34 ± 4
		LRS	37 ± 4	30 ± 4	30 ± 4	34 ± 6
Sodium m (mEq/L)	144–154	HSD + LRS	151 ± 2	164 ± 3*,†	158 ± 2*,†	158 ± 3†
		LRS	149 ± 1	150 ± 2	149 ± 1	149 ± 2
Potassium h (mEq/L)	3.8–5.8	HSD + LRS	3.7 ± 0.2	3.4 ± 0.2	3.7 ± 0.2	3.4 ± 0.2
		LRS	3.9 ± 0.4	3.9 ± 0.3	3.9 ± 0.2	4.0 ± 0.3
Chloride h (mEq/L)	93–121	HSD + LRS	110 ± 2	133 ± 2*,†	127 ± 3*,†	128 ± 4*,†
		LRS	110 ± 1	114 ± 1	112 ± 1	115 ± 1
Osmolality (mOsm/kg)	280–310	HSD + LRS	308 ± 3	331 ± 5*,†	317 ± 5*,†	317 ± 6
		LRS	303 ± 3	305 ± 3	301 ± 4	301 ± 4
Pv_{O_2} (Torr)	> 28	HSD + LRS	30 ± 2	41 ± 4	60 ± 19*	53 ± 13*
		LRS	32 ± 4	38 ± 6	78 ± 25*	42 ± 5*
pH	7.31–7.42	HSD + LRS	7.28 ± 0.8	7.29 ± 0.03	7.21 ± 0.04	7.35 ± 0.02
		LRS	7.33 ± 0.03	7.32 ± 0.02	7.26 ± 0.03	7.31 ± 0.07
Base deficit (mEq/L)	0–5	HSD + LRS	3.5 ± 2.7	5.0 ± 1.5	4.2 ± 1.4	2.0 ± 1.5
		LRS	5.8 ± 2.4	4.0 ± 1.4	6.9 ± 2.1	2.8 ± 2.0

*Within each group, values differ significantly ($P < 0.05$) from baseline values. †For each variable, values differ significantly ($P < 0.05$) between groups.

The PCV decreased significantly from baseline values only in dogs in the HSD + LRS group. Differences were not evident between groups for total plasma protein concentration, albumin concentration, or PCV. Serum sodium and chloride concentrations in dogs in the HSD + LRS group were increased significantly from baseline at 15, 60, and 240 minutes after onset of treatment, but not in dogs of the LRS group. Serum potassium concentration did not change significantly in either group. Mean sodium concentrations determined at 15, 60, and 240 minutes after the onset of treatment in dogs in the HSD + LRS group were significantly greater than the equivalent values in dogs in the LRS group. Osmolality in dogs in the HSD + LRS group was significantly increased from baseline values at 15 and 60 minutes after onset of treatment, but not at 240 minutes.

Mean pH values of dogs in both groups at all periods of measurement were below the reference range or at the low end of the reference range (Table 2). Mean Pv_{O_2} values in dogs in both groups were within the reference range for all time periods during the study. The increased Pv_{O_2} observed in dogs in both groups at 60 and 240 minutes after onset of treatment was likely associated with the administration of oxygen during general anesthesia and surgery.

Premature ventricular contractions (PVC) were detected in 3 of 8 dogs in the LRS group. The PVC were evident intermittently throughout the course of the study, but did not appear to be induced by treatment. Ventricular arrhythmias, including PVC and ventricular tachycardia, were observed in 1 of 7 dogs in the HSD + LRS group. The arrhythmias in that dog were first observed during infusion of HSD, prompting a decrease in the rate of HSD infusion and IV administration of a 60-mg bolus of a solution of 2% lidocaine hydrochloride. The PVC temporarily ceased after lidocaine administration, and the entire dose of HSD was administered. The electrocardiogram in that dog had not been monitored prior to the infusion period to determine whether the ventricular arrhythmias were evident prior to infusion or were induced by the HSD;

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however, the ventricular arrhythmias were detected intermittently throughout the 4-hour period of study.

Discussion

The small volume of HSD solution used in our study for treatment of dogs with GDV-induced shock was effective, compared with large volumes of LRS, in terms of improving cardiorespiratory function and increasing plasma volume. Systolic arterial and central venous pressures increased more rapidly in the HSD + LRS-treated dogs than in dogs in the LRS group, suggesting a more-rapid expansion of plasma volume. This proposed difference in rate of plasma volume expansion was reflected in the rapid decrease in heart and respiratory rates in dogs in the HSD + LRS group. Both forms of treatment were likely to have resulted in substantial plasma volume expansion, as evidenced by decreases in plasma protein concentrations and PCV. However, the more-rapid increase in central venous pressure and more-dramatic decreases in albumin concentration and PCV suggested a greater volume expansion in dogs in the HSD + LRS group, although this was not verified by plasma volume measurements. These cardiorespiratory benefits of treatment with HSD solution were achieved with administration of markedly less cumulative fluid volume than was evident in dogs that received only LRS. The volume of LRS needed to produce a similar degree of resuscitation at each period of measurement for dogs in the LRS group was approximately twice that needed in the HSD + LRS-treated dogs. The difference between groups in terms of cumulative volume of fluid administered at the end of the 4-hour period was approximately 40 ml/kg (1.6 L).

Limiting the cumulative volume of fluid administered to dogs with GDV-induced shock has several potentially important advantages. First, there is an advantage to a resuscitative regimen that requires only 5 minutes to administer. The efficacy, ease, and safety of HSD solution for resuscitation make it an attractive alternative to the use of large volumes of isotonic fluids. Similar to results of other studies, results of the study reported here emphasized the need for administration of additional isotonic fluids, but those may be administered at a moderate rate and volume. Second, HSD solutions restore hemodynamic function quickly.[7,19] This is beneficial in terms of limiting the period of shock and gastric ischemia in a condition such as GDV. The major drawback of rapid reestablishment of blood pressure and flow would be in conditions such as traumatic shock in which vessel injury may result in uncontrolled hemorrhage.[20,21] This is not of major concern when abdominal exploration immediately follows resuscitation and the vascular injury and hemorrhage that may result from GDV can be readily detected and controlled. Third, administration of a large volume of crystalloid fluids that are hypoosmotic (in terms of sodium) and hypooncotic leads to cellular and interstitial edema formation.[22,23] Cellular and interstitial edema can interfere with the mechanical and metabolic functions of various organs and can compromise distribution of blood flow and oxygen delivery.[22] These detrimental effects from administration of large volumes of isotonic fluids may be of particular importance in organs that have been injured and in which compromise of vascular integrity may promote edema. Hypertonic NaCl solution can limit edema in the brain and lungs of dogs with experimentally induced injuries, compared with isotonic NaCl solutions.[24-27] This effect of HSD solutions may provide an important therapeutic advantage for resuscitating dogs with GDV-induced shock or reperfusing ischemic gastric tissues.

Serum sodium and chloride concentrations and osmolality increased significantly from baseline values immediately after administration of HSD solution, but, generally, returned to values not significantly different from those determined at baseline by 240 minutes. However, concentrations of these electrolytes and the osmolality remained higher than reference ranges, even at 240 minutes. These findings contrast slightly with results of our clinical study of the use of HSD solutions in dogs with traumatic shock.[12] In that study, serum sodium and chloride values were higher than reference range values, but typically did not increase as high as values for dogs of the study reported here. Results from the 2 studies varied, despite the administration of nearly equal volumes of HSD solution. Explanation may reside in the fact that larger volumes of isotonic fluid were administered after HSD treatment in the dogs in traumatic shock, which may have diluted the sodium and chloride loads. The highest serum sodium concentration measured in a dog in the study reported here (173 mEq/L) was reached 15 minutes after initiation of HSD treatment. The highest serum sodium concentration in a dog in our other clinical study was 161 mEq/L.[12] We have not observed untoward effects of hypernatremia in our studies.[12] Further, there have not been reported complications of hypertonic saline solution use in resuscitation of human beings in shock that could be directly linked to hypernatremia.[14-16,28] Autopsy of 7 human beings with extreme hypernatremia did not reveal evidence of central pontine myelinolysis,[28] a consequence of a rapid increase in serum sodium concentration.

Serum potassium concentration generally decreases after administration of hypertonic NaCl solutions in dogs[7,12] and human beings[15,16]; however, serum potassium concentration in the dogs of the study reported here did not change significantly after administration of either fluid regimen.

Results of the study reported here compare favorably with results of our previous study of the use of HSD solution for shock in dogs that resulted from experimentally induced GDV.[1] Fluid treatment protocols in that study were nearly identical to the protocol used in the study reported here in terms of volume of HSD solution and isotonic fluids administered to 2 groups of dogs, except that 0.9% NaCl solution was used in place of LRS in the other study. During the first hour of treatment, the resuscitative effects of large volumes of 0.9% NaCl solution were similar for dogs in the former study to those for dogs in the HSD + LRS group in the study reported here. However, cardiac output was significantly higher in the HSD + LRS group during the last 2 hours of the 3-hour resuscitative period, suggesting that blood flow and oxygen

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delivery were better sustained with small volumes of HSD solution. In 1 of our former studies,[1] PCV decreased more in the 0.9% NaCl solution-treated dogs, compared with the HSD + LRS-treated dogs of the study reported here. Reasons for this difference were not clear.

On the basis of the results of the study reported here as well as the results of previous studies,[1,7,11,12] we recommend that HSD solutions be used as the initial treatment for dogs with GDV-induced shock. Isotonic fluids then should be administered at a minimum rate of 20 ml/kg/h. However, whenever possible, the end point of resuscitation should be to optimize cardiorespiratory function, rather than to rely on administration of a fixed-volume of fluid.[4,7,11] Use of HSD solution limited the cumulative amount of isotonic fluids needed, but were more expensive to administer than LRS or 0.9% NaCl solutions, because the HSD solution contained dextran.[4] Use of HSD solutions to treat dogs in shock may be more timely and efficient than use of isotonic fluids, requiring less time during which fluid administration must be monitored and fluid bags have to be changed. Administration of HSD solution was not accompanied by noticeable complications, but the efficacy, safety, and potential advantages of treatment with HSD solutions have not been fully defined.

[a]Parks Medical Electronics Inc, Aloha, Ore.

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