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Comparative study on the effects of acetated Ringer's solution, lactated Ringer's solution, Ringer's solution, and 5% glucose-acetated Ringer's solution on canine hemorrhagic shock

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Abstract: The abilities of acetated Ringer's solution (AR), lactated Ringer's solution (LR), Ringer's solution (R), and 5% glucose-acetated Ringer's solution (AR-D) to improve canine hemorrhagic shock were investigated. All solutions studied were infused at 1 $ml \cdot kg^{-1} \cdot min^{-1}$ for 90 min after base excess (BE) reached about -13 $mEq \cdot l^{-1}$ by maintaining the mean blood pressure (MBP) at 40 mmHg. MBP, renal blood flow (RBF), vertebral blood flow (VBF), and urinary output significantly increased after the start of infusion of AR, LR, R, and AR-D. The VBF and urinary output were particularly improved with AR-D. The arterial blood level of HCO_3^- and BE were also increased after the start of infusion of AR, LR, and AR-D but not of R. AR infusion improved BE more effectively than LR. Although AR-D, AR, and LR increased HCO_3^-, the blood pH did not increase in AR-D. The value of plasma acetate increased after the start of infusion of AR and AR-D but not of LR, and R. On the other hand, plasma lactate and pyruvate levels were higher with LR than with AR. The increase in the lactate/pyruvate ratio induced by LR was larger than that by AR. The plasma norepinephrine and epinephrine levels decreased after the start of all infusions. Plasma insulin and glucose levels were markedly increased after the start of AR-D infusion but were not affected by AR, LR, and R. These results indicate that the effectiveness of various infusion solutions such as AR, LR, R, and AR-D during canine hemorrhagic shock varies. AR-D may be useful for increasing both peripheral blood flow and urine output. AR may also be useful for improvement in metabolic acidosis and surgical diabetes induced by hemorrhagic shock.

Key words: Acetated Ringer's solution, Lactated Ringer's solution, Ringer's solution, 5% glucose-acetated Ringer's solution, Canine hemorrhagic shock, Metabolic acidosis, Surgical diabetes

Introduction

A shift towards anaerobic metabolism during shock causes increased lactate production and acidosis [1]. There is considerable evidence to suggest that shock in general produces cardiac dysfunction and liver abnormalities [2–4]. The administration of lactate, coupled with increased endogenous lactate production, may place further strain on the liver to clear lactate. A threefold or more increase in blood lactate results from anaerobic glycolysis and decreased utilization by the liver as a result of hepatic dysfunction. In contrast with lactated Ringer's solution (LR), acetated Ringer's solution (AR) has been reported to be beneficial during hemorrhagic shock and to increase cardiac contractility without increasing metabolic acidosis [5]. Furthermore, the clinical usefulness of AR in normovolemia is well documented [6,7].

It has been reported that elevation of circulating glucose levels can occur during various stressful situations, including major surgery, trauma, burn injury, hypothermia, and sepsis [8]. The hyperglycemic response to stressful states may be maladaptive due to the potentially harmful effects of hyperosmolality on solute and water conservation, as well as brain function and other vital organs. From this point of view, the infusion of 5% glucose-acetated Ringer's solution (AR-D) to patients with hyperglycemia induced by stressors such as hemorrhagic shock may increase the circulating glucose level.

Therefore, in the present study, the influence of AR, LR, Ringer's solution (R), and AR-D on hemodynamic parameters, metabolic acidosis, and hyperglycemia during hemorrhagic shock in a canine model were investigated.

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Methods

General procedures

Twenty male beagle dogs (Nihon CLEA, Tokyo, Japan and OBC, Shizuoka, Japan) weighing between 9 and 12 kg (10 to 12 months in age), were anesthetized with sodium pentobarbital (25 mg·kg^{-1}, i.v.). Additional doses of sodium pentobarbital were given when necessary to maintain anesthesia. The trachea was intubated, and the animals were mechanically ventilated with room air delivered from a large-animal respirator (SN-480-3, Shinano, Tokyo, Japan). The ventilation rate (12–18 cycles·min^{-1}) and tidal volume (15–20 ml·kg^{-1}) were adjusted to maintain arterial blood pH and $Paco_2$ within physiological ranges. Body temperature was measured from rectum and was maintained at 36° ± 1°C with a heated pad. A polyethylene cannula was placed in the lower abdominal aorta through the left femoral artery and connected to a pressure transducer (Gould, Oxnard, Calif., USA) to measure systemic arterial blood pressure (SBP). The mean blood pressure (MBP) was obtained by electrical averaging. The heart rate (HR) was measured by a cardiotachometer (1321, NEC Sanei, Tokyo, Japan) triggering an R wave at lead II on the electrocardiogram.

Evaluation of renal blood flow, vertebral blood flow, and urine output

The left flank was opened between the iliac crest and the costovertebral angle. The left renal artery was exposed retroperitoneally, then isolated from the surrounding connective tissues. An electromagnetic flow probe was placed around the artery for continuous blood flow monitoring. Through a lower median cervical incision, the left vertebral artery as exposed and an electromagnetic flow probe was placed to monitor the vertebral blood flow as described above. The left ureter was cannulated to monitor urinary output which was represented as the pooled volume over 5 min.

Experimental protocols

All variables as described above were stabilized for at least 45 min before the study was begun. HR, systolic blood pressure (SBP), MBP, renal blood flow (RBF) and vertebral blood flow (VBF) were recorded simultaneously (control phase, C phase). Heparin (500 unit/kg) was administered intravenously to prevent blood coagulation. All dogs bled rapidly through the right femoral artery until the MBP reached about 40 mmHg by controlled the height of blood reservoir (hemorrhagic early phase, HE phase). Thereafter, the height of blood reservoir was fixed. Additional amounts of blood were either withdrawn from the femoral artery or transfused from the blood reservoir to achieve a MBP of 40 mmHg until the base excess (BE) decreased to about −13 mEq·l^{-1} (hemorrhagic late phase, HL phase). Thereafter, each Ringer's solution was infused at a constant flow rate of 1 ml·kg^{-1}·min^{-1} for 90 min through the left femoral vein. Arterial blood samples were drawn from the subclavian artery at the C phase, HE phase, HL phase, then at 15, 30 60, 90, and 120 min after the start of infusion to evaluate the hematocrit value, blood osmotic pressure, plasma levels of acetate, lactate, pyruvate, norepinephrine, epinephrine, insulin, glucose, and blood gases (pH, $Paco_2$, Pao_2, HCO_3^-, and BE). Animals were divided into the following infusion groups of 5 each: AR, LR, R, and AR-D. The composition of the infusions studied are listed in Table 1.

Table 1. Composition of infusion solutions

Item	AR	LR	R	AR-D
Na^+ (mEq·l^{-1})	130	130	146	130
K^+ (mEq·l^{-1})	4	4	4	4
Ca^{2+} (mEq·l^{-1})	3	3	3	3
Cl^- (mEq·l^{-1})	109	109	155	109
Acetate$^-$ (mEq·l^{-1})	28	–	–	28
Lactate$^-$ (mEq·l^{-1})	–	28	–	–
Glucose (w/v%)	–	–	–	5

AR, acetated Ringer's solution; LR, lactated Ringer's solution; R, Ringer's solution; AR-D, 5% glucose-acetated Ringer's solution.

Statistical analysis

All data are represented as means ± standard error. Statistical analysis was performed by one-way analysis of variance and the unpaired Student's *t*-test between AR and other solutions. *P* values less than 0.05 were considered significant.

Results

Figure 1 shows the time course of changes in MBP, HR, RBF, and VBF in the AR, LR, R, and AR-D groups. The MBP was lowered to about 40 mmHg by removing 411 ± 16 ml of blood via the femoral artery (bleeding rate: 4.5 ± 0.3 ml·kg^{-1}·min^{-1}, bleeding period: 7.8 ± 0.7 min). In the HE and HL phases, the RBF and VBF progressively decreased.

During fluid infusions, improvements in MBP, RBF, and VBF were seen before completion of the infusion in all groups. Especially, AR-D was significantly more effective ($P < 0.05$) in improving VBF than AR at 90 min after the start of infusion (10 ± 2, 14 ± 5, 5 ± 3, and 24 ± 6 ml·min^{-1} in the AR, LR, R, and AR-D groups, respectively). In the HR response, there was no significant change during the experiment.

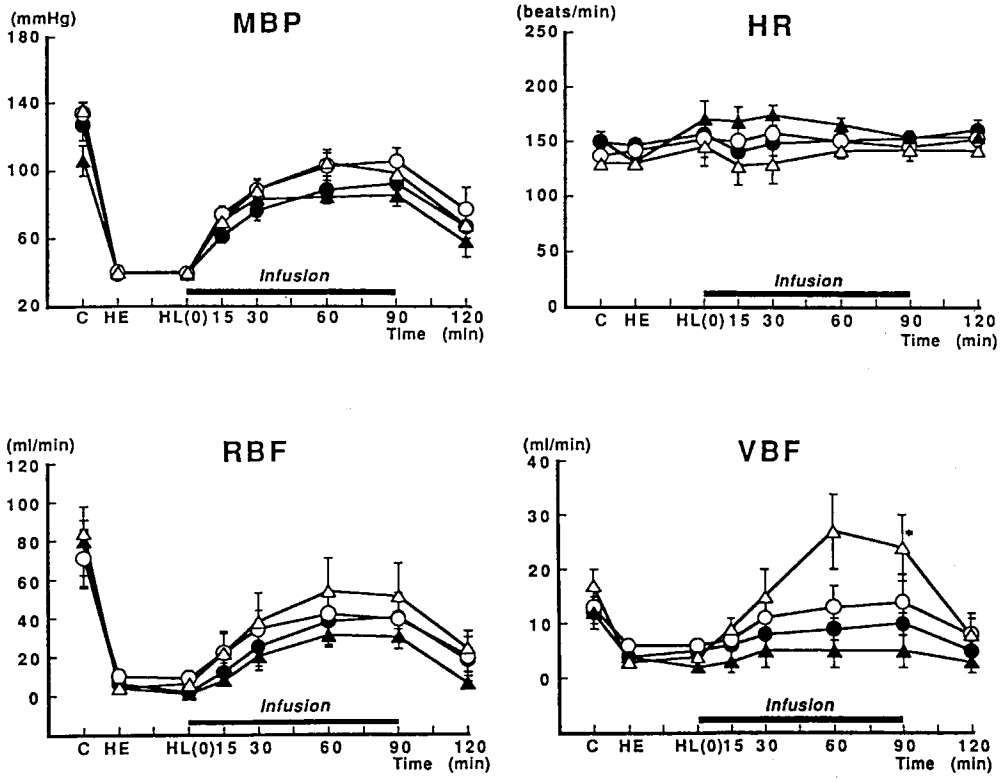

Fig. 1. Effects of acetated Ringer's solution (*AR, closed circles*), lactated Ringer's solution (*LR, open circles*), Ringer's solution (*R, closed triangles*), and 5% glucose-acetated Ringer's solution (*AR-D, open triangles*) on mean blood pressure (MBP), heart rate (*HR*), renal blood flow (*RBF*), and vertebral blood flow (*VBF*). *C*, control phase; *HE*, hemorrhagic early phase; *HL*, hemorrhagic late phase. $^*P < 0.05$ from AR group

Figure 2 shows the time course of changes in hematocrit value, blood osmotic pressure and urinary output in the AR, LR, R, and AR-D groups. The hematocrit value and blood osmotic pressure were slightly increased at the HL phase. In the HE and HL phases, urine output decreased to 0 ± 0 ml from 1.0 ± 0.2 ml in the AR group, 0.7 ± 0.1 ml in the LR group, 0.8 ± 0.2 ml in the R group, and 0.9 ± 0.2 ml in the AR-D group.

The hematocrit value fell during infusion of AR, LR, R, and AR-D and the largest decrease was seen with AR-D ($P < 0.05$) 90 min after the start of infusion ($28 \pm 1, 26 \pm 2, 26 \pm 1$, and $22 \pm 1\%$ in AR, LR, R, and AR-D, respectively). The blood osmotic pressure fell during the infusion of AR, LR, and R. However, the blood osmotic pressure in the AR-D group significantly increased ($P < 0.05$) from 15 to 90 min after the start of the infusion (325 ± 2, 328 ± 1, 335 ± 2, and 341 ± 1 $mOsm \cdot l^{-1}$ at 15, 30, 60, and 90 min after the start of infusion, respectively). Urinary output was also increased after the start of infusion in all groups ($P < 0.05$); the most being in the AR-D which was significantly different from the AR group 90 min after the start of infusion (1.9 ± 1.6, 0.8 ± 0.4, 1.2 ± 0.4, and 10.3 ± 3.1 ml, in AR, LR, R, and AR-D, respectively).

Figure 3 shows the time course of changes in arterial blood pH, $Paco_2$, Pao_2, HCO_3^-, and BE in each group. At the HE and HL phase, arterial blood pH, HCO_3^-, and BE progressively decreased. $Paco_2$ was slightly decreased at the HE phase followed by an increased toward the control level at the HL phase. Moreover, Pao_2 was slightly increased during the HE phase followed by a slight decrease at the HL phase.

Marked improvement in the blood pH and BE were seen in the AR and LR groups. AR more significantly affected blood pH ($P < 0.05$) than LR, R or AR-D 15 min (7.154 ± 0.015, 7.068 ± 0.017, 7.075 ± 0.020, and 7.051 ± 0.011 in the AR, LR, R, and AR-D groups, respectively) and 30 min (7.166 ± 0.015, 7.099 ± 0.017, 7.076 ± 0.030, and 7.053 ± 0.018 in the AR, LR, R, and AR-D groups, respectively) after the start of the infusion. HCO_3^- progressively increased with AR, LR, and AR-D but not by R infusion, and there were no significant differences among AR, LR, and AR-D. Although HCO_3^- was increased after the start of AR-D infusion, the blood pH was not improved in the AR-D infused group. The improvement of BE induced by AR was significantly larger ($P < 0.05$) than those seen with LR, R, and AR-D 60 min after the start of infusion (-7.7 ± 0.9, -10.3 ± 0.6, -13.2 ± 1.3, and $-11.9 \pm$

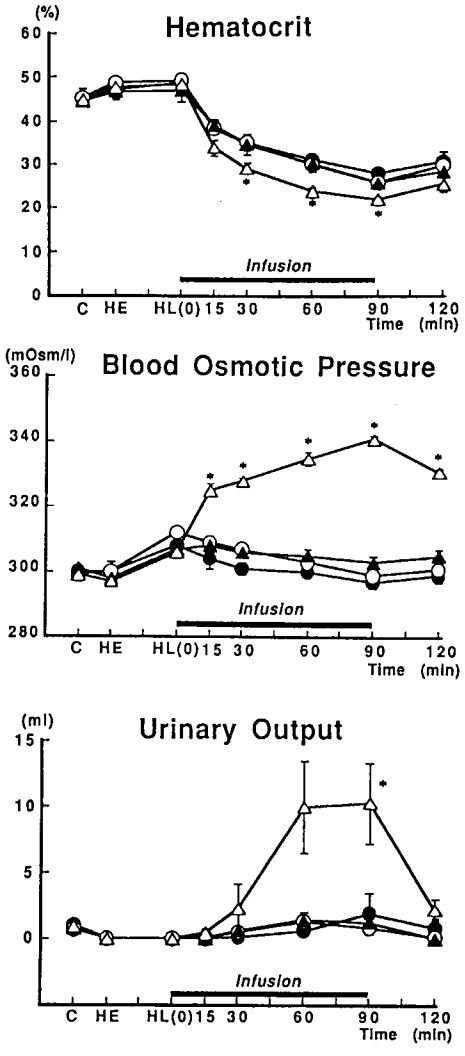

Fig. 2. Effects of AR (*closed circles*), LR (*open circles*), R (*closed triangles*), and AR-D (*open triangles*) on hematocrit value, blood osmotic pressure and urinary output. $*P < 0.05$ from AR group

0.7 $mEq \cdot l^{-1}$ in the AR, LR, R, and AR-D groups, respectively). The $Paco_2$ and Pao_2 did not significantly change during AR, LR, and R infusion. The $Paco_2$ level after the start of AR-D infusion was significantly increased ($P < 0.05$) compared with the AR group. However, the Pao_2 level was not significantly changed in the AR-D group.

Figure 4 shows the time course of changes in plasma acetate, lactate, and pyruvate levels, and in the lactate/pyruvate ratio in each group. Plasma levels of lactate and pyruvate, and the lactate/pyruvate ratio progressively increased during the HE and HL phases.

In the AR and AR-D groups, the plasma acetate level increased and reached a peak of 4.0 ± 0.3 and 2.5 ± 0.4 $mg \cdot dl^{-1}$, respectively, 15 min after the start of infusion. On the other hand, the plasma lactate level in the LR group was significantly higher ($P < 0.05$) than the AR group 15–60 min after the start of infusion. The plasma pyruvate level was also higher ($P < 0.05$) in the group than the AR group 30 and 60 min after the start of infusion. Moreover, the lactate/pyruvate ratio in LR was higher ($P < 0.05$) than that in AR 15 and 60 minutes after the start of infusion. In the AR-D group, plasma lactate and lactate/pyruvate ratio 60 minutes after the start of infusion were significant higher ($P < 0.05$) than that in the AR group.

Figure 5 shows the time course of changes in plasma norepinephrine, epinephrine, insulin, and glucose levels in each group. Both plasma levels of norepinephrine and epinephrine were progressively increased at the HE and HL phases. The plasma insulin profile was biphasic: a decrease at the HE phase to 2.0 ± 0.5, 1.7 ± 0.1, 1.5 ± 0.0, and 1.6 ± 0.1 $mcu \cdot ml^{-1}$ from 3.2 ± 0.8, 4.5 ± 0.8, 2.3 ± 0.3, and 4.6 ± 1.2 $mcu \cdot ml^{-1}$, followed by an increase at the HL phase to 54.9 ± 35.6, 139.3 ± 51.2, 42.8 ± 18.9, and 61.4 ± 40.8 $mcu \cdot ml^{-1}$ in the AR, LR, R, and AR-D groups, respectively. Plasma glucose levels progressively increased at the HL phase to 176 ± 49, 321 ± 75, 172 ± 33, and 206 ± 75 $mg \cdot ml^{-1}$ in the AR, LR, R, and AR -D groups, respectively.

The plasma levels of norepinephrine and epinephrine decreased after the start of AR, LR, R, and AR-D infusion. These changes were not significantly different among the groups except at 15 min after the start of infusion in the LR group, and at 15 and 30 min in the R group. The plasma insulin levels decreased in the AR, LR, and R groups but that in the AR-D group was significantly higher ($P < 0.05$) than the AR-induced level. The plasma glucose levels in the AR, LR, and R groups decreased to 94 ± 18, 101 ± 10, and 73 ± 12 $mg \cdot dl^{-1}$ 90 min after the start of infusion, from 176 ± 49, 321 ± 75, and 172 ± 33 $mg \cdot dl^{-1}$ at the HL phase, respectively. In the AR-D group, the plasma glucose level progressively and significantly increased ($P < 0.05$) compared with the AR group from 15 to 120 min after the start of infusion, reaching a peak of 1112 ± 40 $mg \cdot dl^{-1}$ at 90 min after the start of AR-D infusion.

Discussion

The present study shows that AR improved metabolic acidosis more than LR at 60 min after the start of infusion. However, the plasma levels of insulin and glucose did not increase during AR infusion. Although AR-D increased the VBF and urinary output more than AR, the plasma glucose level in AR-D was much higher than that induced by infusion with other solutions. In the AR and AR-D groups, the plasma acetate level was increased significantly after the start of infusion. LR, however, caused a significant increase in plasma lactate

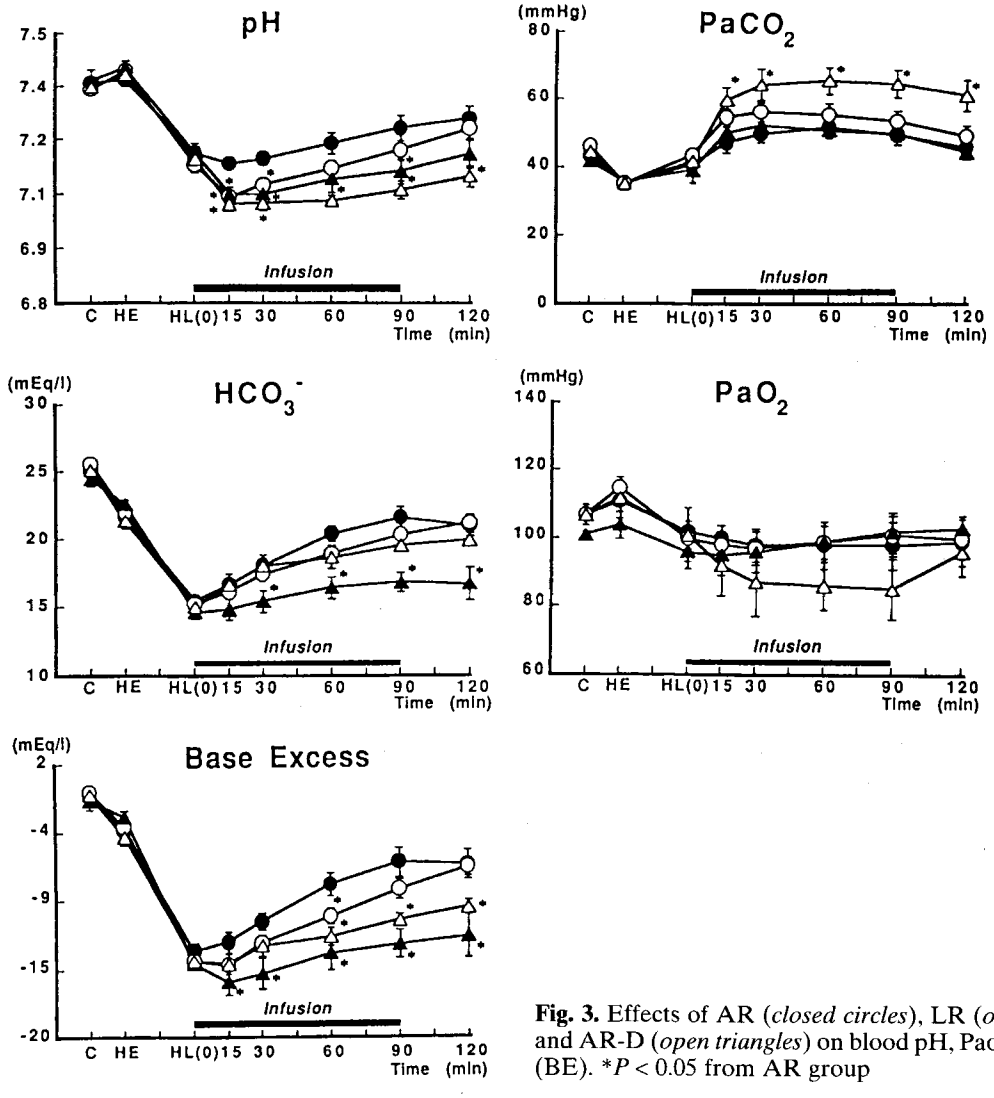

Fig. 3. Effects of AR (*closed circles*), LR (*open circles*), R (*closed triangles*), and AR-D (*open triangles*) on blood pH, $Paco_2$, Pao_2, HCO_3^-, and base excess (BE). $^*P < 0.05$ from AR group

15–60 min after the start of infusion. These results suggest that acetate utilization functioned well even during prolonged hemorrhagic shock. Moreover, the present study suggests that there are important differences in the effectiveness of infusion fluids during canine hemorrhagic shock.

Hemodynamic parameters were improved after infusion of all of the solutions studied. The greatest improvements in VBF and urinary output were seen during infusion of AR-D. RBF during infusion of AR-D also tended to show greater improvement than those in other groups. On the other hand, plasma insulin and glucose levels were markedly increased after AR-D infusion. AR-D also induced a higher blood osmotic pressure than those seen with AR, LR, and R. This hyperosmolality may be caused by hyperinsulinemia and hyperglycemia, and also fluid osmolality of AR-D, which is in the range of 500–600 $mOsm \cdot l^{-1}$. It has been reported that a rapid expansion of plasma volume occurred as osmotically induced fluid shifts from the cellular space into the circulation [9]. Therefore, the increase in RBF, VBF, and urinary output after AR-D infusion may be due to the increase of circulating volume by hyperosmolality (osmotic diuresis). Additionally, it is well known that the cerebral vessels are very sensitive to carbon dioxide tension. An increase in arterial blood CO_2 tension elicitis marked cerebral vasodilation; inhalation of 7% CO_2 results in a twofold increase in cerebral blood flow [10]. In the present study, $Paco_2$ level after the infusion of AR-D was higher than with AR, LR and R. Thus, this higher $Paco_2$ may have contributed to the increase of VBF seen in AR-D. On the other hand, it has also been demonstrated that cerebral vessel diameter and pH are inversely related, independent of the level of $Paco_2$ [11]. Moreover, lactate has also been proposed as a mediator of metabolic vasodilation [11]. In the present study, we also demonstrated that the plasma lactate level in the AR-D group was high and

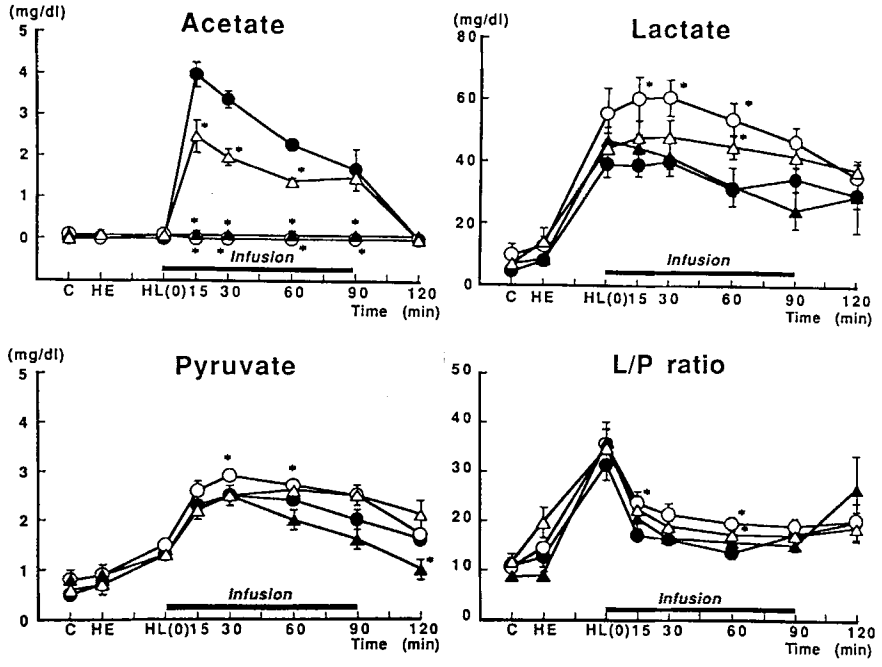

Fig. 4. Effects of AR (*closed circles*), LR (*open circles*), R (*closed triangles*), and AR-D (*open triangles*) on plasma acetate, lactate, and pyruvate, and on the lactate/pyruvate ratio (*L/P ratio*). $*P < 0.05$ from AR group

arterial blood pH was low compared with the AR group. From these results, it may be suggested that hypercapnia and metabolic acidosis during AR-D infusion may have increased the VBF.

The shift towards anaerobic metabolism during shock increases lactate production and stimulated metabolic acidosis [1]. It has also been reported that impaired hepatic perfusion during profound shock limits the capacity for lactate removal, and thereby aggravates lactic acidosis [12]. Kveim and Nesbakken [5] reported that acetate may be given as an alkalinizer during hemorrhagic shock without the risk of accumulation that occurs with an equivalent amount of lactate. In the present study, we demonstrated that the blood pH and BE were improved after the start of AR infusion more than with LR infusion. Moreover, the plasma acetate level increased during AR infusion but did not accumulate. These results are consistent with those of previous reports which showed that acetate utilization functioned well during prolonged hemorrhagic shock [5],

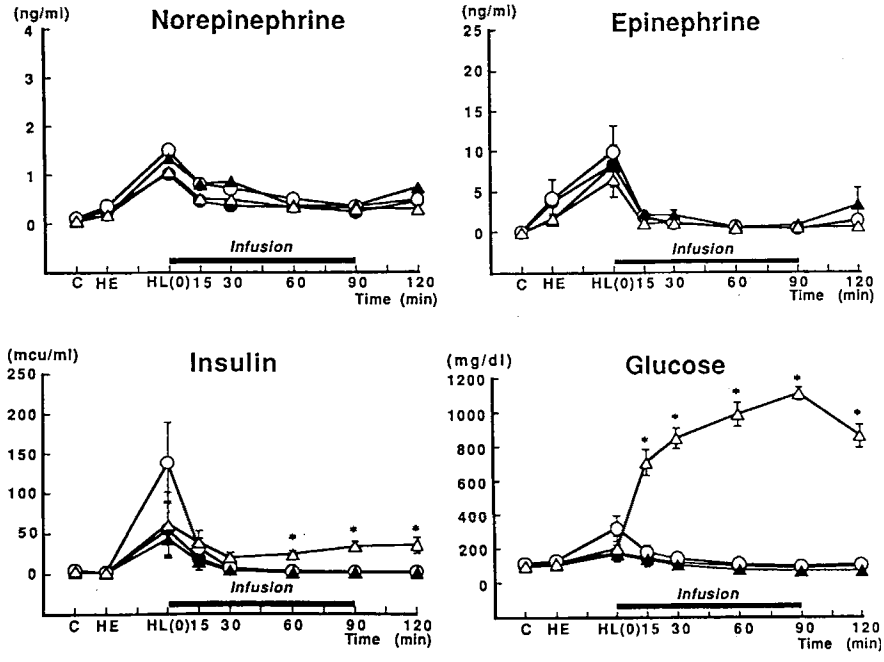

Fig. 5. Effects of AR (*closed circles*), LR (*open circles*), R (*closed triangles*), and AR-D (*open triangles*) on plasma norepinephrine, epinephrine, insulin, and glucose level. $*P < 0.05$ from AR group

suggesting that AR is better than LR in improving metabolic acidosis.

Although AR improved metabolic acidosis more than LR, AR-D was less effective compared with AR and LR. One possibile explanation for this may be that its pH because AR-D is more acidic than AR, LR, and R (4.1–4.9, 6.5–7.5, 6.5–8.0, and 4.5–7.0, respectively). Another possibility, which was reported by Alberti and Cuthbert [13], is that H^+ production by the glycolytic process enhanced metabolic acidosis. Actually, the increase in $Paco_2$ during AR-D infusion, which may result from glucose metabolism, was also observed in the present study. These results indicate that AR-D caused a slight improvement in metabolic acidosis, and it could supply energy in patients without insulin resistance and improve hemodynamics.

Hemorrhagic shock is associated with sympathetic nervous system activation [14], and catecholamines directly stimulate glucose production in the liver [15]. In the present study, the plasma norepinephrine and epinephrine levels were progressively increased during hemorrhagic shock. Additionally, catecholamines interfered with insulin-mediated glucose uptake [16]. Therefore, the release of catecholamines was closely related to the development of hyperglycemia during hemorrhagic shock. The use of spinal or epidural anesthesia to block afferent neural signals from the site of tissue injury prevents activation of the sympathetic nervous system during and after surgery [17]. At the same time, hyperglycemia is prevented, and there is no impairment of insulin secretion [16,18]. Similarly, impaired insulin secretion during surgical stress is reversed by the alpha adrenergic antagonist phentolamine [19]. Thus, the results in the present study also suggest that catecholamines may mediate hyperglycemia. On the other hand, increased plasma glucagon was reported during surgical stress [20]. However, there are conflicting reports by Dahn et al. [21] and Black et al. [22]. Dahn et al. [21] reported that increased insulin turnover under injury and sepsis might contribute to insulin resistance. Black et al. [22] reported that insulin resistance following injury appears to occur in the peripheral tissues, and is consistent with a postreceptor defect. These reports indicate that the mechanism of hyperglycemia is complex during hemorrhagic shock, and that it may be difficult to control the blood glucose level at that time. Thus, glucose-free infusion such as AR may be useful in avoiding hyperglycemia.

Okada et al. [23] evaluated the effects of LR, AR, and AR-D as perioperative infusions. They reported an increase in the plasma level of D-lactate with LR infusion, but not with other AR and AR-D solutions. The L-lactate was increased in the plasma in patients who received AR-D infusion, whereas there was no significant change in the other two groups. The plasma glucose in the AR-D group was significantly increased, and the level of insulin was also elevated simultaneously [23]. Arai et al. [24] also indicated the clinical usefulness of AR as an intraoperative fluid. Moreover, Kimura [25] reported rapid metabolization of AR and considered it to be a useful extracellular fluid replacement. Additionally, Kuze et al. [26] reported that when AR-D was infused in patients who were undergoing less invasive surgery for tympanoplasty under general anesthesia, the plasma concentrations of lactate, pyruvate, acetate, and glucose were higher than the cases with AR and LR. The levels of excretion of these metabolites into the urine after the infusion of AR-D were much higher than those after AR and LR infusions. AR-D should be administered in appropriate amounts that would not induce clinically significant metabolic alterations. Kuze et al. [26] also reported a significant increase in plasma lactate and pyruvate and in the lactate/pyruvate ratio after LR infusion, but not in patients who received AR infusion. However, a significant increase in plasma acetate was found after AR but not after LR infusion. The present results obtained from anesthetized dogs appear to confirm these clinical observation [23–26].

In conclusion, the effectiveness of various infusions such as AR, LR, R, and AR-D during hemorrhagic shock in a canine model were compared in the present study. AR-D may be useful for increasing of renal blood flow, vertebral blood flow, and urine output. AR may also be useful for improvement in metabolic acidosis and surgical diabetes induced by hemorrhagic shock.

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