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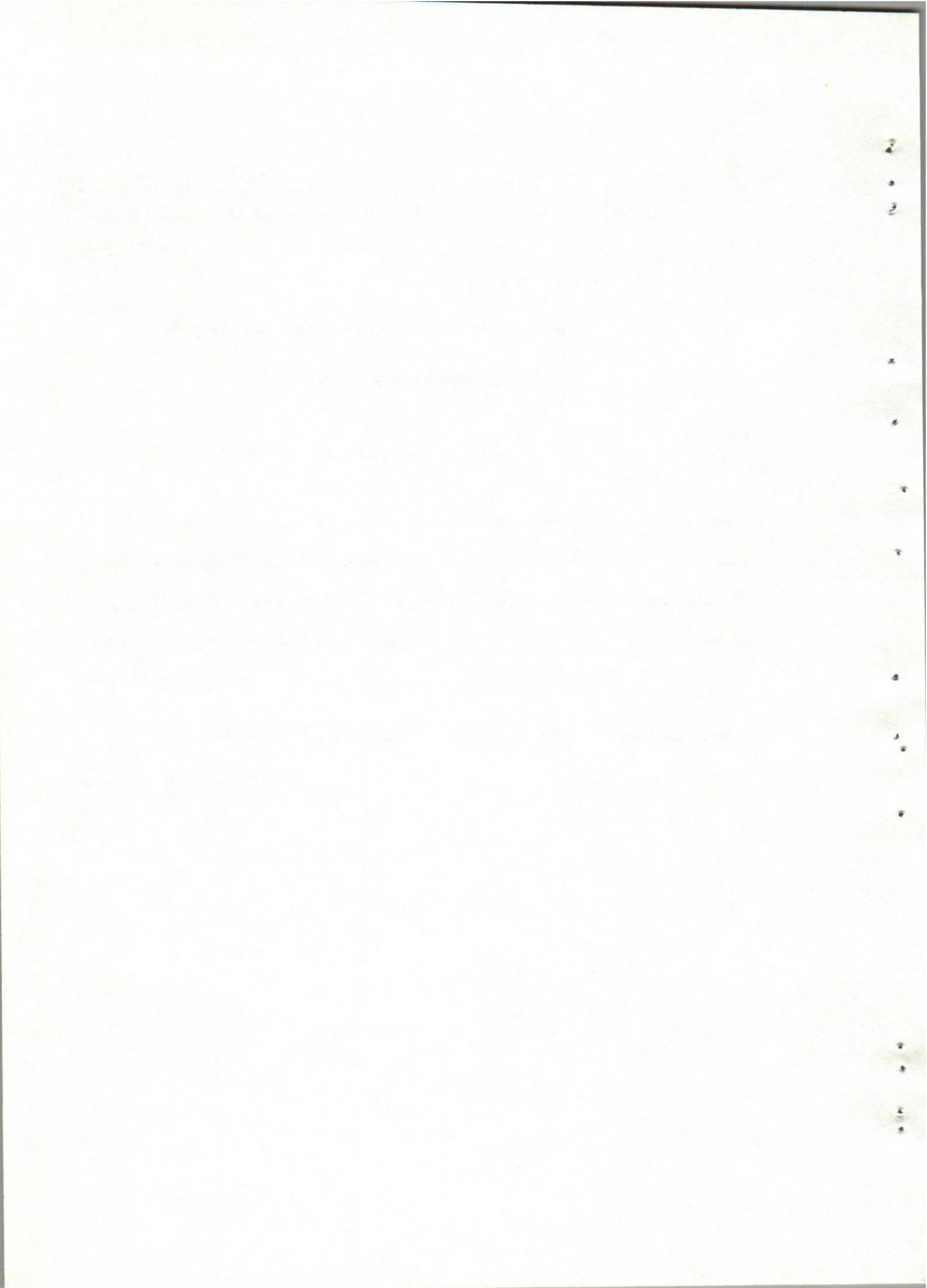
تغيرات حجم الدم والبلازما والهيماتوكريت

في الصدمة المعدية للعجول

ب . ساهاي ، ر . كوهلي

استهدفت الدراسة تقييم تأثير الصدمة المعدية على حجم الدم وبلازما الجسم وكذلك قيمة الهيماتوكريت في عجول الأبقار ولقد تبين أن حجم الدم والبلازما قد انخفض معنويًا في المراحل الأخيرة من الصدمة بمقدار ١٢ر٢٪ ، ١٥ر٨٪ بالترتيب عن الضوابط وهي الظاهرة التي لم تلاحظ خلال الفترة الأولى من الصدمة المعدية .

ارتبط ارتفاع قراءة الهيماتوكريت - في المراحل الأخيرة للصدمة - بالسالب مع تغير حجم الدم .



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**CHANGES IN BLOOD AND PLASMA VOLUMES AND HAEMATOCRIT
FOLLOWING EXPERIMENTAL SEPTIC SHOCK IN CALVES**
(With One Table)

By
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SUMMARY

Septic shock was induced in seven clinically healthy calves to evaluate its effect on blood and plasma volumes and haematocrit at various stages of shock. Blood and plasma volume in later stages of shock, declined significantly ($P/ < 0.05$) to 12.2% and 15.8%, respectively from normal. However, there was no appreciable change in blood and plasma volumes and the haematocrit at initial stages of shock. The rise in the haematocrit in the terminal stage was negatively correlated with the plasma volume. These findings suggest that the septic shock in bovines is hypovolemic in its terminal stages. Moreover, the changes observed in this investigation were conspicuously different from those recorded in other species.

INTRODUCTION

In attempts to clarify the pathophysiological mechanisms in shock, much interest has been devoted to the flow properties of blood which is influenced by, apart from other factors, the volume changes and changes in haematocrit (BRANEMARK, 1968). As opposed to haemorrhagic shock, septic shock is considered to be normovolemic (SIMEONE, 1969) but the haemodynamic features associated with experimental endotoxin shock in animals suggest it to be contrary. Though limited studies on septic shock (SINGH and KOHLI, 1980, SAHAY and KOHLI, 1982) and shock induced by direct administration of endotoxin (TIKOFF *et al.*, 1965; ANDERSON *et al.*, 1973) in bovine have been conducted, there appears to be no study on the changes occurring in blood and plasma volumes in septic shock in this species. Endeavours of indirectly approximating the blood volume by some changes in other blood parameters are futile. Though the values of different constituents of blood alter as a result of changes in blood volume but these do not provide correct information on the blood and plasma volumes and may even be misleading if haemoconcentration or haemodilution is present (SWENSON, 1970). Therefore, it was imperative to study the changes in blood and plasma volumes directly, at different stages of septic shock in bovines.

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MATERIAL and METHODS

Experiments were conducted on seven healthy, cross bred male calves of 1 to 11/2 years of age and weighing between 72 to 106 kg. The animals were maintained under uniform feeding and managerial schedule prior to the experimentation. For producing septic shock a segment of jejunum (50 - 55 cm in length) was strangulated after right flank laparotomy. Venous drainage from this segment of jejunum was occluded while the arterial blood supply was kept intact.

Blood samples for normal values of blood and plasma volumes and haematocrit were collected just before the start of the experiment. Subsequent determination of these parameters was done at early shock and late/terminal shock states, which were tentatively established keeping in view the prevailing haemodynamic status and clinical manifestations of each animal (SAHAY and KOHLI, 1983).

The plasma volume was estimated spectrophotometrically by dye dilution method (GREGERSON, 1944) using Evans blue dye (T- 1824). The haematocrit was measured by the method of WINTROBE (1967). The true haematocrit was obtained by multiplying the haematocrit value with correction factor (0.87). The blood volume was calculated by following formula:

$$\text{Blood volume} = \text{Plasma volume} \times \frac{100}{100 - \text{true haematocrit}}$$

RESULTS

The mean values \pm S.E. of different parameters in normal and at early and late/terminal shock have been presented in Table (1).

Table (1)

Mean \pm S.E. of blood and plasma volumes and haematocrit in septic shock in calves.

Parameters	Normal	Early shock	Late shock
Blood volume ml/kg	69.3 \pm 1.57	67.5 \pm 1.86	60.8* \pm 2.17
Plasma volume ml/kg	55.2 \pm 0.69	53.0 \pm 0.89	46.5* \pm 1.29
Haematocrit %	25.9 \pm 1.50	27.3 \pm 1.39	30.3 \pm 1.87

* Statistically significant ($p < 0.05$)

The decline in blood volume at early shock was marginal where the values diminished to 67.5 ml/kg from the preshock value of 69.3 ml/kg. However at terminal shock there was 12.2% decrease in blood volume and it was statistically significant ($P < 0.05$).

Plasma volume also evidenced statistically significant ($P < 0.05$) change where the decline was 15.8% at terminal shock. The values decreased to 46.5 ml/kg at this stage when

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compared to preshock value of 55.2 ml/kg. On the contrary, haematocrit exhibited a rise at both the stages of shock (Table 1).

DISCUSSION

The model used for producing septic shock in this study has earlier been reported from this laboratory (SINGH and KOHLI, 1980 and SAHAY and KOHLI, 1983). Administration of endotoxin intravenously has been reported to modify the properties of cell membrane in dogs resulting into increased permeability of the cells (SOLOMON and HINSHAW, 1968). Further, as a response of endotoxaemia there is excessive synthesis and release of histamine as evidenced by increased histidine decarboxylase activity (HINSHAW *et al.* 1961). The characteristic action of histamine explains a number of actions seen in endotoxaemic shock including hypotension, haemoconcentration, intravascular fluid loss (KUIDA *et al.* 1958 and GILBERT *et al.*, 1958). In this study, the ensuing shock was septic in nature which implied overwhelming sepsis leading to the release of endotoxins in the system. Apart from it the direct action of live bacteria is also contemplated contrary to the endotoxin administration. Bacteremia and endotoxins evoke primary cell damage and result into increased cell permeability (GELIN *et al.*, 1980).

Normally capillaries are permeable to smaller fractions of proteins and the intravascular albumin equilibrates with that of intravascular albumin at measurable rates (DAWIDSON *et al.*, 1980). But in endotoxin shock, plasma sequestration in intestines as a result of increased capillary permeability leading to the escape of larger protein macromolecules in intestines in low flow status (AUST *et al.*, 1957) and even when portal venous pressure is normal (CHIEN *et al.*, 1964), give sufficient evidence that decreased plasma volume in this study appears to be associated with the loss of intravascular colloids and proteins. The loss of plasma fluids and proteins to extravascular space results into lowering of plasma protein concentration and the total circulating plasma volume. Decreased total plasma proteins, albumin and globulin has been observed in a similar model of septic shock (SINGH and KOHLI, 1980). Therefore, it is postulated that the loss of plasma volume in septic shock in calves is probably due to the integrated actions of live bacteria, endotoxins and histamine at the cellular level. The magnitude of such loss (15.8%) in this study, however, was dramatically different from dogs (67%) under intestinal shock (DAWIDSON *et al.*, 1980) and this suggests a characteristic species variation.

The rise in haematocrit values at late shock indicated moderate haemoconcentration. This was probably due to combined effects of loss of plasma fluid from the circulation as well as splenic contraction in response to late effectiveness of sympathetic stimulation as observed in septic shock in calves. This rise in haematocrit at late shock stage (17%) was almost inversely comparable to plasma loss (15.8%) at the same stage. However, this observation was in contrast to the findings in rats where loss of plasma volume (upto 55% in terminal stage) in endotoxaemia in most cases had no correspondance with changes in haematocrit (SHAPIRO *et al.*, 1958).

Though the loss in blood volume was significant, it was lesser in magnitude (12.2%) as compared to that of plasma volume loss at late shock stage. These could well be attributed to the resulting increase in the haematocrit values at those stages. Further, the overall changes in blood volume also suggest that the plasma loss was not associated with concomitant loss of blood cells from the circulation in septic shock in calves of the present study. The moderate haemoconcentration observed in this study is different from the haemodilution observed in haemorrhagic shock in buffalo calves (SOBTI *et al.*, 1981). This probably was due to the

divergence of the models and nature of shock.

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