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ACID-BASE STATUS IN LOW- AND HIGH-FLOW STATES IN SEPTIC SHOCK IN CALVES

P. N. SAHAY* and R. N. KOHLI

Department of Surgery and Radiology, Punjab Agricultural University, Ludhiana 141004, India

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Abstract

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Acid-base status was studied in 20 clinically healthy calves exhibiting either hypokinetic (low flow) or hyperkinetic (high flow) state or a combination of the two forms of cardiac dynamics during experimentally induced septic shock. There was uncompensated metabolic alkalosis in the majority of the animals irrespective of cardiac kinetics. But two animals of the low flow group exhibited uncompensated metabolic acidosis. These findings suggest that alterations in acid-base status are largely independent of the variations in the flow states.

Septic shock, acidosis, alkalosis, calves.

Early efforts in understanding the mechanisms of septic shock were focused on the cardiovascular system but recently metabolic events are being considered important (Wolfe and Burke 1978). The metabolic derangement with shock is suggested to determine the haemodynamics (Wright et al. 1971) and to be significant denominator in cardiovascular instability in man (Robertson et al. 1969). In the bovines, response of the cardiovascular system to septic shock has been in three distinct categories (Sahay 1982). These animals may exhibit hyperkinetic dynamics (high flow state) or hypokinetic dynamics (low flow state) or a combination of the two flow states. The present investigation was thus undertaken to evaluate changes in the acid-base status during such flow states in calves subjected to experimental septic shock.

Material and Methods

This study was conducted on twenty healthy male calves of one to one and half year of age and maintained under uniform managemental schedule. Animals were kept off feed and water, 24 and 12 hours, respectively, prior to induction of shock. Under local analgesia, siliconised catheters were placed in the carotid artery and jugular vein. A three-way stop-cock was attached to each catheter for facilitating the collection of blood samples as well as for monitoring of different parameters of cardiac kinetics. A stabilization period of one hour was allowed, after the catheterizations, for development of a physiologic steady state. Subsequent to this, analysis of various parameters was done to form the base value. For induction of shock, right flank laparotomy was performed under local infiltration analgesia and a 50–55 cm segment of jejunum was strangulated by ligating the venous channels of this segment. Intestines were repositioned in the abdominal cavity and wound closed routinely.

Arterial and venous blood was collected in heparinized precalibrated capillary tubes at intervals ranging from 1 to 2 hours and analysed immediately. The pH and P_{CO_2} (partial pressure of carbon dioxide) of arterial & venous blood were measured with blood gas analyzer (Radiometer, Copenhagen, The BME 33 microequipment) at 37 °C. Bicarbonate (HCO_3) and base excess- extracellular

* Present address: Ranchi Veterinary College, Ranchi, Birsa Agricultural University, Ranchi – 834007, India

fluid of arterial blood were derived from Siggard-Andersen Nomogram using measured pH and P_{CO_2}. Arterial carbonic acid (H_2CO_3) was calculated as the product of P_{CO_2} values and the solubility coefficient of CO_2 (0.03).

The time elapsing between creation of strangulation and death of each animal was divided into five stages of equal duration and values plotted accordingly. Thus, in an animal which took 30 hours to die, first stage was taken at 6 hours, second at 12 hours, third at 18 hours and so on. One-way analysis of variance and Student's t-test were applied for statistical analysis of data.

Results and Discussion

Success was achieved in creating septic shock in 20 calves. It has been convincingly demonstrated that in such a model of shock in calves, positive gram-negative haemoculture, associated inflammatory response and deteriorating haemodynamics indicate the shock to be septic in nature (Singh and Kohli 1981). The manifestations of cardiac output (Q) and related parameters were variable. Twelve animals exhibited hypokinetic dynamics (low flow state) in which Q decreased throughout the period of study whereas 3 calves manifested a hyperkinetic (high flow) feature where there was an increase in Q. In the remaining 5 animals mixed kinetics i. e. high flow state in early shock period followed by low flow state in late shock stages or viceversa, were observed. To preclude ambiguity in the results, the data for the acid-base status of these groups were analyzed separately and are presented in Table 1.

The mean values of arterial blood pH showed a tendency to rise at all stages in all the three forms of shock but this rise was greater in high flow and combination of high and low flow states. Exceptions to this feature were two calves in the low flow group (hypokinetic) where the arterial pH from the base values of 7.538 and 7.498, decreased to 7.316 and 7.307, respectively, in the terminal stage. The arteriovenous pH difference (A-V pH) increased consistently, this being statistically significant ($P < 0.01$) with low flow group. Though the mean values of arterial P_{CO_2} did not manifest striking variations as the shock progressed, there were wide variations in the individual animals in low flow group. In six animals of this group the P_{CO_2} decreased at the terminal stage as compared to the preshock value, whereas in the other six animals P_{CO_2} increased at the same stage.

Arterial bicarbonate exhibited a tendency to increase with progression of shock in all forms. However, in combination shock this increase was discernible only at third and fifth stages (Table 1). In low flow state, HCO_3 showed appreciable individual variations. In two calves HCO_3 dropped to 22.5 and 23.6 mmol/l at the terminal stages from the preshock value of 32.0 and 29.7 mmol/l, respectively, whereas in four calves there was no appreciable alteration. The mean values of base excess showed a pattern of rise at different shock stages in a variable magnitude and this rise was significant in the high flow group. The most interesting feature was, however, observed in low flow shock where two animals exhibited base deficit, five animals base excess and in remaining five animals base excess values remained almost unaltered.

The correlation of pH, P_{CO_2}, HCO_3, H_2CO_3 and base excess in this study elucidates uncompensated metabolic alkalosis in four animals of low flow, two animals of combination and all animals of high flow group. This is in sharp contrast to the progressive uncompensated metabolic acidosis observed in man and dog during septic shock (Lillehei 1971 and Hinshaw et al. 1979). These marked species variations in the acid-base status of bovines following septic shock have been

Table 1

Mean Values + S. E. of various acid-base parameters during low flow (A), high flow (B) and combination of low and high flow (C) septic shock in calves

Parameters	Stages of shock					
	0	1	2	3	4	5
Arterial pH						
A	7.455 ±0.014	7.468 ±0.017	7.488 ±0.012	7.490 +0.010	7.504 ±0.015	7.482 ±0.030
B	7.453 ±0.023	7.469 ±0.012	7.505 ±0.009	7.526 ±0.004	7.530 ±0.008	7.547 ±0.018
C	7.467 ±0.003	7.460 ±0.023	7.469 ±0.017	7.542 ±0.042	7.495 ±0.023	7.544 ±0.038
A−V pH						
A	0.041 ±0.008	0.055 ±0.011	0.075 ±0.009	0.090 ±0.010	0.106 ±0.013	0.135 ±0.015
B	0.055 ±0.005	0.030 ±0.017	0.060 ±0.033	0.108 ±0.031	0.109 ±0.031	0.147 ±0.034
C	0.052 ±0.024	0.024 ±0.012	0.074 ±0.031	0.104 ±0.034	0.107 ±0.037	0.145 ±0.029
Arterial P_{CO_2} mm Hg.						
A	39.0 ±1.06	39.0 ±1.7	38.7 ±2.0	39.5 ±1.6	39.1 ±1.4	39.6 ±1.7
B	39.2 ±1.3	39.1 ±1.6	41.8 ±1.2	42.8 ±1.1	42.0 ±1.1	39.7 ±1.5
C	37.2 ±2.6	39.3 ±2.7	36.5 ±4.5	38.6 ±2.2	35.4 ±2.3	37.4 ±1.6
Arterial HCO_3 mmol/l						
A	27.4 ±1.1	28.0 ±0.8	29.2 ±1.4	29.8 ±1.3	30.8 ±1.0	29.7 ±1.4
B	27.1 ±1.2	28.2 ±0.5	32.9 ±1.5	35.9 ±0.6	35.5 ±0.3	34.9 ±1.5
C	28.5 ±2.8	27.9 ±1.8	27.3 ±4.7	34.2 ±2.4	27.5 ±2.9	32.6 ±2.1
Arterial H_2CO_3 mmol/l						
A	1.13 ±0.04	1.14 ±0.06	1.16 ±0.6	1.18 ±0.05	1.17 ±0.04	1.18 ±0.05
B	1.17 ±0.04	1.17 ±0.04	1.25 ±0.03	1.28 ±0.02	1.25 ±0.03	1.19 ±0.04
C	1.11 ±0.08	1.18 ±0.08	1.09 ±0.14	1.31 ±0.10	1.06 ±0.07	1.12 ±0.05
Base Excess mmol/l						
A	3.5 ±0.9	4.1 ±0.8	5.3 ±1.3	5.8 ±1.2	7.0 ±1.1	5.6 ±1.6
B	3.1 ±1.4	4.6 ±0.7	8.8 ±1.3	11.3 ±0.6	11.3 ±0.2	10.9 ±1.5
C	4.6 ±2.8	3.8 ±1.7	3.1 ±1.3	10.1 ±2.4	4.6 ±2.4	8.8 ±2.3

HCO_3 = bicarbonate, H_2CO_3 = carbonic acid. Stages 1−5 represent post-shock survival period divided into five stages of equal duration.

ascribed to the herbivorous diet of ruminants and possible variations in kidney function (Singh and Kohli 1980).

Though metabolic acidosis does not seem to be a feature of bovine septic shock, two animals in the low flow group evidenced moderate metabolic acidosis. This was accompanied by respiratory acidosis suggesting that metabolic acidosis was uncompensated. Interestingly, these two calves of low flow shock were those in which HCO_3 and pH had declined significantly and there was base deficit in the terminal stages. This elucidates that the feasibility of the development of metabolic acidosis cannot, altogether, be precluded when low flow septic shock supervenes in calves.

The progressive increase in the A-V pH difference with progression of all forms of shock in this study indicates metabolic derangement (Nelson and Swan 1974). This was due to an increase in the arterial pH and an almost concomitant decrease in the venous pH, except in very few cases. In man, metabolic derangements present during the different phases of shock have been suggested to primarily determine the haemodynamics (Wright et al. 1971). But it is evident from this study that the changes in the acid-base status are largely independent of the variations in the cardiac kinetics. Hence it appears that alterations in the acid-base status may not contribute much towards the variations in the cardiovascular dysfunction in the bovine septic shock.

Acidobazická rovnováha v septickém šoku u telat s hypokinetickou nebo hyperkinetickou krevní cirkulací

Acidobazická rovnováha byla sledována u 20 klinicky zdravých telat s hypokinetickou nebo hyperkinetickou krevní cirkulací, či s kombinací obou v průběhu experimentálně vyvolaného septického šoku. U většiny zvířat nastala nekompenzovaná metabolická alkalóza bez ohledu na cirkulační dynamiku. U dvou telat s hypokinetickou krevní cirkulací byla zjištěna nekompenzovaná metabolická acidóza. Tyto nálezy naznačují, že poruchy acidobazické rovnováhy jsou jen málo závislé na rozdílech v dynamice krevního oběhu.

Кислотно-основное равновесие в септическом шоке у телят с гипокинетической или гиперкинетической циркуляцией крови

Проводились исследования кислотно-основного равновесия у 20 клинически здоровых телят с гипокинетической или гиперкинетической циркуляцией крови, а также с комбинацией обеих в ходе экспериментально вызванного септического шока. У большинства животных наступил некомпенсированный метаболический алкалоз, несмотря на динамику циркуляции. У двух телят с гипокинетической циркуляцией крови был установлен некомпенсированный метаболический ацидоз. Установленные данные подсказывают, что нарушения кислотно-основного равновесия лишь незначительно зависимы от равностей динамики кровообращения.

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