Cardiac output during endotoxic shock in goats

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Received: 30 September 1993

The haemodynamic aspects of septic shock have been fairly studied in calves (Suhay and Kohly 1983) and buffalo calves (Singh and Kohli 1980) but not in goats. This paper reports the cardiac output response at different stages of experimentally induced endotoxic shock in goats.

Six clinically healthy Black Bengal male goats, 8-10 months old and weighing 10-15 kg, were kept off feed overnight prior to induction of shock. The animals were secured comfortably in right lateral recumbency, and carotid artery and jugular vein were exteriorised through left jugular furrow under local anaesthesia. Silastic catheter was placed in the artery and a 3-way stop-cock was attached to the catheter for injection of dye and collection of blood sample for cardiac output (Q) measurement. Endotoxic shock was induced by injecting intravenously endotoxin of Escherichia coli serotype 055B5 (Sigma Chemical Co., USA) @ 100 µg/kg body weight. Cardiac output was measured using Evans Blue dye (T-1824) by the technique described by Stowe and Good (1960). It was calculated according to the formula as below:

\[ Q = \frac{60 \times A}{T \times C \cdot (1-H)} \]

Where
- Q is cardiac output in litre/min;
- A is the amount of dye injected in mg;
- T is time in second between the first appearance and its reappearance following recirculation;
- C is the average concentration of dye in mg/litre of plasma, and
- H is the haematocrit value measured by micro-haematocrit method.

Q was also calculated on body weight basis, i.e. ml/kg/min. Cardiac index (Q/body surface area m²) was calculated on the basis of Q/min/m² of the body surface area (BSA) according to Meech's formula as BSA = K/W²/3, where K, is 0.121 for goats and W is weight of animals.

\[ CI = \frac{QL/min}{BSA (m^2)} \]

Stroke volume (SV) was measured by dividing Q, ml/min by heart rate/min. Stroke index in turn was calculated by dividing the stroke volume (in ml) with body weight (in kg). Heart rate was determined by auscultation with stethoscope.

The stabilized pre-shock value of each parameter served as normal control (stage 0). Since the survival time of each animal following shock varied widely, the time elapsing between induction of shock and death was divided into stages 1-5 of equal duration. Early shock indicated stage 2 and terminal shock stage indicated stage 5.

Statistical analysis of data was done with
one-way analysis of variance. Critical difference (CD) test was applied to study the difference between two means (Snedecor and Cochran 1967).

The average survival time was 6.5±1.21 hr (range 4-12 hr). The mean values of heart rate showed significant (P<0.01) and consistent and progressive increase from 96.5±1.39 to 157.3±1.90/min at the terminal stage. This should be due to the reflex response of the baroreceptor and chemoreceptor in the face of hypotension and circulation endotoxin.

Cardiac output decreased significantly (P<0.01) from the base value of 4.14±0.12 to 3.20±0.12 and 2.86±0.09 litre/min in the early and terminal stages of shock respectively. Decreased cardiac output could be correlated directly with the changes in MAP, CVP and inversely to the heart rate. The decrease in cardiac output is recorded if heart rate is increased significantly in the presence of decreasing arterial pressure (Sugimoto et al. 1960). The increase in the heart rate beyond physiological limit resulted into impaired ventricular filling and diminished systolic ejection. Furthermore, there was a linear relationship between pressure and flow rate in the systemic circulation (Read et al. 1957). Therefore in the present study marked increase in heart rate along with greater hypotension and significant decline in CVP were the chief reasons of decreased Q in endotoxic shock.

Cardiac index decreased (P<0.01) in the early and terminal stages from the base value of 7.96±0.11 to 5.96±0.16 and 4.40±0.09 litre/m²/min respectively. In the present study cardiac index exhibited higher value than Q (litre/min) at the control as well as at the early and terminal stages of shock. Cardiac index being the expression of cardiac output in relationship to body surface area, the lower body surface in the goats would have exhibited lower values of cardiac index.

There was significant (P<0.01) fall in stroke volume from the base value of 43.01±1.97 to 25.93±0.92 ml/beat in the early stages of shock and 15.05±0.84 ml/beat in the terminal stage. The changes in the stroke index followed almost similar pattern as observed in the context of stroke volume. The greater increase in the heart rate coupled with fall in cardiac output was the cause of higher decrease in stroke volume in the terminal shock stage than early stages. This constituted the inadequacy of perfusion leading to hypoxia and death at the late stages of endotoxic shock and substantiates the findings of Sahay and Kohli (1983) in calves.

ACKNOWLEDGEMENT

The first author thanks the ICAR, New Delhi, for the financial assistance in the form of Senior Fellowship to carry out this experiment.

REFERENCES


