Electrocardiographic Studies During Endotoxic Shock and after Flunixin Meglumine, ketanov Infusion Singly and in Combination in Male Buffalo Calves

Corresponding Author:
Dr. Digvijay Singh,
Professor, Dept of Veterinary Physiology and Biochemistry, 141004 - India

Submitting Author:
Dr. Digvijay Singh,
Professor, Dept of Veterinary Physiology and Biochemistry, C.O.V.S., GADVASU, 141004 - India

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Author(s): Singh D, Bansal SK

Abstract

15 apparently healthy buffalo calves were divided into 3 groups of 5 animal each. Each group was infused E.coli endotoxin @ 5 µg/kg BW/hour for 3 hours and subsequently group 1 was intravenously Fusixin meglumine @ 1.1 mg/kg BW while group 2 was given intravenous infusion of Ketanov @ 1.12mg/kg BW and in group 3 a combination of two NSAIDs was given to investigate effect of the NSAIDs alone or in combination. A decrease in 'T'wave, its reversal and 2nd degree A-V conduction block was observed during infusion of endotoxin which could not be removed after I/V infusion of Flunixin meglumine or Ketanov alone or in combination.

Introduction

Shock is a progressive deterioration in the microcirculation due to inability of the cardiovascular system to maintain blood pressure and flow. Septic shock is an acute circulatory failure occurring in the presence of severe infection. Shock is a state of underperfusion of the tissues that are most vital to life such as brain, myocardium, kidney, liver and intestine. Mc Donnel (1974) defined shock as a clinical syndrome in which the cardiovascular system is unable to maintain pressure and flow above the minimum physiological requirements.

Constable et al (1991a) observed significant decrease in cardiac index, aortic pressure, pulmonary arterial hypertension and increased pulmonary vascular resistance at the end of endotoxin infusion. Reece and Whalstrom (1973) reported tachycardia in endotoxic cow calves while Singh (1979) observed atrial fibrillation in buffalo calves suffering from endotoxia due to E. coli endotoxin along with those of Proteus and Staphylococcus.

During shock, there is substantial morbidity and mortality in cattle, especially neonates (Gerros et al 1993). Septic shock is still an unresolved problem in regard to management of the therapy (Gellin et al 1980, Singh et al 2007).

Singh et al (1999) believed that no single drug or therapy can treat endotoxemia. A combination of specific therapies which include fluid replacement, antibiotics, endotoxin mediator blockers and repair of acid-base balance can counter the specific disturbances during shock. Three possible approaches in treating endotoxia are:

i) The interaction of endotoxin with target cells can be blocked by inducing tolerance,
ii) decreasing plasma endotoxin concentrations,
iii) interfering with endotoxin binding (Hardie and Krusse-Elliott 1990).

The present study was conducted to monitor the effects of infusion of Flunixin meglumine and Ketanov alone and in combination with another NSAID on the electrocardiogram.

Materials And Methods

Normal healthy buffalo calves (15), aged between 4 months to 1 year were dewormed with fenbendazole @ 5 mg/kg BW and divided into 3 groups of 5 animals each. All groups were infused with E. coli endotoxin (lyophilized, phenol extracted 0111: B4 lipopolysaccharide) @ 5µg/kg BW/hr for 3 hours. Group I animals were infused with Flunixin meglumine (FM) @ 1.1 mg/kg BW. Group II animals were infused with Ketanor@ 1.1 kg/kg BW while group-III endotoxic buffalo calves were infused with Flunixin Meglumine and Ketanov.

The electrocardiogram of all animals were recorded using ECG machine. The recordings were made in the frontal plane for time voltage fluctuations. The cardiac rate, timings and potentials were calculated from the second standard lead of ECG. The data thus collected were analysed using completely randomized design ANOVA and 't' test.
Results and Discussion

A non significant change in the p-wave potential and timings during endotoxin infusion was observed (Table 1). Administration of various treatment combinations did not induce any significant change in the potential and timings in all groups (Table 1). Similar trends have been reported in bovines (Singh et al 2007). The normal mean QRS potential varied between -0.26±0.05 to 0.64±0.17 ml. Non-significant changes in the QRS potential and timings during endotoxin infusion and after respective treatment were observed (Table 1). The normal mean T-wave potential ranged between 0.02±0.14 to -0.30±0.01 m V (table 2) which is lower than 0.08±0.02 m V (Grewal et al 1998) 0.37±0.05 m V (Singh et al 1982) and 0.29±0.00 m V (Singh et al 2007). T-wave reversal was observed in all the animals of group I and III during endotoxin infusion and even after treatment throughout the period of observation (Fig 1 & 3). The reversal of T-wave potentials indicate ischaemic ventricular myocardium (Singh et al 2007).

The timings of P-wave QRS complex and T Wave did not significantly change during endotoxin infusion and after treatment (Table 1 and 2). No significant change was observed upon endotoxin infusion and after treatment in P-R and Q-T interval. The changes in S-T interval were non-significant during and after endotoxin infusion and after treatment. The mean heart rate/min ranged between 44.12±3.43 to 50.91±7.68 beats/min (Table 5) which is close to 48.67±4.84 to 50.50±15.50 beats/min (Sobti et al 1981) but slightly less than 52.91±2.92 to 61.50±1.19 beats/min (Singh 2000), and 52.60±2.92 to 61.50 beat/min (Singh 2007). A significant change in heart rate was observed at the end of observation period i.e., 7 hrs of start of endotoxin infusion in groups II and III which were treated with ketanov and a combination of Flunixin meglumine and ketanov. The A-V block in one animal of group III observed at 5, 6 and 7 hours i.e. end of observation period (Fig 2) is suggestive of conduction blockade. This A-V conduction block could be due to harmful effects of endotoxin directly on conduction system of heart. The damage caused by endotoxin in the initial stages of endotoxin infusion may be of permanent nature and it cannot be reversed by flunixin meglumin alone or in combination with ketanov. The decrease in T wave potentials although non-significant is suggestive of toxic depression of heart which affects pumping efficiency and thereby, reduces blood flow, tissue perforation and pressures. The results of present investigation makes us conclude that there is no effect of Flunixin Meglumine or ketanov alone or in combination with other NSAID on electrocardiogram of endotoxemic buffalo calves.

References

7. Singh D.V. and Bansal, S.K 2007 Electrocardiographic studies during endotoxic shock and after Flunixin meglumine, ketanov infusion singly and in combination in male buffalo calves
Illustrations

Illustration 1

Figure-1

ECG RECORDING AT VARIOUS STAGES OF ENDOTOXIC SHOCK AND AFTER TREATMENT WITH FLUNIXIN MEGLUMINE IN BUFFALO CALVES (GROUP-I)

NORMAL

1 HOUR

2 HOUR

3 HOUR (END OF ENDOTOXIN INFUSION)

4 HOUR (AFTER FLUNIXIN MEGLUMINE INFUSION)

5 HOUR

6 HOUR

7 HOUR
Illustration 2

Figure-2

ECG RECORDING AT VARIOUS STAGES OF ENDOTOXIC SHOCK AND AFTER TREATMENT WITH FLENIXIN MEGLUMINE & KETONV IN BUFFALO CALVES (GROUP-III)

NORMAL 1 HOUR

NORMAL 1 HOUR

NORMAL 3 HOUR 4 HOUR

3 HOUR (END OF ENDOTOXIN INFUSION) 4 HOUR

5 HOUR A-V BLOCK

6 HOUR A-V BLOCK

7 HOUR A-V BLOCK
Illustration 3

Figure-3

ECG RECORDING AT VARIOUS STAGES OF ENDOTOXIC SHOCK AND AFTER TREATMENT WITH FLUNIXIN MEGLUMINE & KETONOV IN BUFFALO CALVES (GROUP-III)

NORMAL

1 HOUR

2 HOUR

3HOUR (END OF ENDOTOXIN INFUSION)

4 HOUR

5 HOUR

6 HOUR

7 HOUR

REVERSE T WAVE

REVERSE T WAVE

REVERSE T WAVE

REVERSE T WAVE

REVERSE T WAVE
Illustration 4

Table 1: Pwave and QRS complex potentials and timing at different time intervals during endotoxic shock and after treatments.

<table>
<thead>
<tr>
<th>Time</th>
<th>P-wave Potentials (mV)</th>
<th>P-wave Timings (Sec.)</th>
<th>QRS Complex pot (mV)</th>
<th>QRS Timing in Sec.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group I</td>
<td>Group II</td>
<td>Group III</td>
<td>Group I</td>
</tr>
<tr>
<td>3 hrs</td>
<td>0.09±0.01</td>
<td>0.05±0.00</td>
<td>0.05±0.00</td>
<td>0.07±0.02</td>
</tr>
<tr>
<td>1 hr</td>
<td>0.08±0.01</td>
<td>0.05±0.00</td>
<td>0.07±0.01</td>
<td>0.06±0.01</td>
</tr>
<tr>
<td>2 hr</td>
<td>0.09±0.01</td>
<td>0.05±0.00</td>
<td>0.05±0.00</td>
<td>0.05±0.01</td>
</tr>
<tr>
<td>3 hr</td>
<td>0.08±0.12</td>
<td>0.05±0.00</td>
<td>0.05±0.01</td>
<td>0.05±0.01</td>
</tr>
<tr>
<td>4 hr</td>
<td>0.07±0.01</td>
<td>0.05±0.00</td>
<td>0.05±0.00</td>
<td>0.06±0.01</td>
</tr>
<tr>
<td>5 hr</td>
<td>0.07±0.01</td>
<td>0.05±0.00</td>
<td>0.05±0.00</td>
<td>0.06±0.01</td>
</tr>
<tr>
<td>6 hr</td>
<td>0.07±0.01</td>
<td>0.05±0.00</td>
<td>0.05±0.00</td>
<td>0.06±0.01</td>
</tr>
<tr>
<td>7 hr</td>
<td>0.07±0.01</td>
<td>0.05±0.00</td>
<td>0.05±0.00</td>
<td>0.07±0.01</td>
</tr>
</tbody>
</table>

Group-I Flunixin Meglumine
Group II Ketonov
Group III Flunixin Meglumine+ Ketonov
## Illustration 5

### Table 2

T Wave potentials & timings and heart rate at different time intervals during endotoxic shock and after different treatments.

<table>
<thead>
<tr>
<th>Time</th>
<th>T wave Potentials (mV)</th>
<th>T wave Timings (Sec.)</th>
<th>Heart Rate/min.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group I</td>
<td>Group II</td>
<td>Group III</td>
</tr>
<tr>
<td>3 hrs</td>
<td>-0.30±0.01</td>
<td>-0.10±0.08</td>
<td>0.02±0.14</td>
</tr>
<tr>
<td>1 hr</td>
<td>0.27±0.14</td>
<td>0.06±0.1</td>
<td>0.07±0.05</td>
</tr>
<tr>
<td>2 hr</td>
<td>0.17±0.10</td>
<td>0.10±0.08</td>
<td>0.01±0.04</td>
</tr>
<tr>
<td>3 hr</td>
<td>0.15±0.08</td>
<td>-0.02±0.08</td>
<td>-0.02±0.07</td>
</tr>
<tr>
<td>4 hr</td>
<td>0.10±0.08</td>
<td>-0.10±0.09</td>
<td>-0.01±0.04</td>
</tr>
<tr>
<td>5 hr</td>
<td>0.11±0.08</td>
<td>-0.03±0.07</td>
<td>0.02±0.06</td>
</tr>
<tr>
<td>6 hr</td>
<td>0.14±0.09</td>
<td>-0.01±0.09</td>
<td>0.11±0.07</td>
</tr>
<tr>
<td>7 hr</td>
<td>0.17±0.12</td>
<td>-0.01±0.09</td>
<td>0.15±0.06</td>
</tr>
</tbody>
</table>

Group-I Flunixin Meglumine
Group II Ketonov
Group III Flunixin Meglumine + Ketonov
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