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# Relationship between some serum electrolytes and electrocardiographic indices of *Trypanosoma brucei* infected dogs

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**Abstract**

The effect of *Trypanosoma brucei* infection on changes in concentration of some serum electrolytes and the consequence of these changes on electrocardiographic (ECG) indices were investigated in dogs. The nature of association between each of the electrolytes and the various ECG indices were studied at different days during the infection. The serum concentration of sodium, potassium and calcium were assayed before and at 8th, 16th, and 24th days after infection in dogs. The ECG was also recorded concurrently with serum electrolyte assay in each dog. The infection, although it caused a significant change in serum sodium ($P \leq 0.001$) and potassium ion concentration ($P \leq 0.01$) of the dogs, it did not significantly affect their serum calcium ion concentration. ($P \geq 0.05$). There was no difference between the serum sodium and potassium levels of arrhythmic and non-arrhythmic dogs but serum calcium concentration of arrhythmic dogs was higher than that of non-arrhythmic ones. The QT, RR Variability, QT and $QT_C$ width correlated strongly and significantly with serum sodium, potassium and calcium at different times during the period of infection. There was a significant correlation between calcium and heart rate on the 16th and 24th day of infection. Potassium correlated with T/R and the R wave voltage at various times during the infection In conclusion, *T. brucei* infection affected the serum electrolytes concentration and may have caused ECG changes through mechanism that involves the modulatory responses of the autonomic nervous system and the ion channels.

*Keywords*: Calcium, Electrocardiogram, Potassium, Sodium, *Trypanosoma brucei*
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## Introduction

African canine trypanosomiasis is a disease of public health and social importance characterized by anemia, myocarditis, corneal opacity, lymphadenopathy, and convulsion (Morrison *et al.,* 1981; Matete, 2003). This disease in dogs is caused by *Trypanosoma brucei brucei, T. brucei rhodesiense, T. brucei gambiense, T. congolense and T .evansi* (Losos & Ikede, 1972).

This disease like canine babesiosis, is a septicemic condition involving both cardiac and renal pathology (Morrison *et al.,* 1981). Systemic and metabolic disturbances that induce ECG changes and arrhythmia often characterized canine babesiosis (Jacobson & Clarke, 1994; Jacobson *et al.*, 2000). Similar alterations may also be seen in *T. brucei* infection of dogs. ECG changes and arrhythmia have been reported in both experimental *T. brucei* and *T. cruzi* in dogs (Ndungu *et al.*, 1991; Lana *et al.*, 1992). Serum biochemical markers of hepatic and cardiac damage have been investigated in *T. brucei* infection in dogs. So far not much has been reported on the serum electrolyte correlates of the ECG changes in *T. brucei* infected dogs.

Knowing the role played by serum electrolytes like calcium, sodium, and potassium in the presence of a suitable myocardial substrate will aid our

understanding of the pathogenesis, diagnosis, treatment and prognosis of the diseases.

The modulatory role of serum electrolytes like calcium, sodium, and potassium in the pathogenesis of the arrhythmia and the relationship between the ECG waves and these serum electrolytes in African canine trypanosomiasis was investigated in this present study.

**Materials and methods**

Ten healthy dogs that weighed between 4 and 8Kg and aged between 3 and 6months were used for the study. The dogs received all routine vaccinations against common dog diseases and were all certified free from heartworms. They were acclimatized for two weeks and screened against trypanosome species and other hemoparasites. Dogs were kept in fly proof kennel, fed twice on commercial dog food and allowed access to water *ad libitum*. *T. brucei* (Federe strain) obtained from National Veterinary Research Institute, Vom, was used for this study. The parasites were preserved by sub-passaging in donor albino rats. This work is in conformity with the regulations of the ethical committee of the College of Veterinary Medicine, University of Agriculture, Abeokuta.

A six leads (I, II, III, AVR, AVL and AVF) body surface electrocardiogram was recorded before inoculation of each dog with 1ml of PBS-diluted blood containing $1\times10^6$ of the parasite intraperitoneally and their ECG subsequently monitored serially on days 8, 16, and 24 post infection. The ECG was recorded at a paper speed of 25mm/sec and pen sensitivity of 1mm=0.1mV with the dog on right lateral recumbency. Coupler Davis electrodes were placed on the appropriate limbs and electrode gel was used to maintain good contact. All recordings were made on one of the channels of a four channel universal Student Oscillograph (Harvard Apparatus, UK) by the same ECG technician.

The duration of the QT, QRS and the amplitude of the R and T waves were recorded manually using the ruler and the calipers. The QT intervals were measured and averaged in five consecutive P-QRS-T complexes in each lead and the preceding RR interval were also measured and averaged. The intervals were measured manually to the nearest 0.5mm using calipers and ruler.

The QT interval was measured from the beginning of Q to the end of T which has been defined as the return of T to the isoelectric line (Brooksby *et al.*, 1999). On each electrocardiogram, the lengths of the longest and shortest RR interval were measured together with the corresponding following QT interval. The individual variability for the RR and QT were calculated for each ECG as the difference between the longest and the shortest RR interval and their corresponding QT interval length. The corrected QT (QTc) was derived with the Fridericia formula: $QTc=QT/RR^{1/3}$ (Fridericia, 1920). T amplitude was also evaluated as T/R ratio (Dvir *et al.*, 2004). The heart rate was determined by counting the number of cycles (RR interval) in six seconds and multiplying by ten.

Serum sodium, potassium, and calcium were assayed before infection and on days 8, 16, and 24 post infection. The blood samples for this assay were collected just before the ECG studies. Serum sodium concentration was determined spectrophotometrically according to the modified method as described in the Teco diagnostic test manual. Serum potassium concentration was also determined spectrophotometrically according to the method of Teri and Sesin (1958) as described in the Teco diagnostic test manual. The serum calcium concentration was determined colorimetrically according to the method of Ray Sarkar & Chauhan (1967) as described in the Randox test kit manual. The concentration of the individual electrolyte was correlated with some ECG variables before infection and on days 8, 16, and 24 post infection.

*Statistical analysis*

All data were expressed as mean ± standard deviation. Differences within parameters during the course of the disease were evaluated by ANOVA for repeated measures. Statistical significance between the pre- infection control and a value at a particular time point after the infection was determined by paired t- test with Bonferoni correction.

The arrhythmia group was also compared with the non-arrhythmia group using the t-test for independent sample. When $P \leq 0.05$, the infection was adjudged to have affected the parameter. The relationship between the serum potassium, sodium and calcium concentration and the repolarisation indices was evaluated by simple correlation. The strength of association, i.e. correlation coefficient (R) was considered significant when $P \leq 0.05$. All statistical tests were done using SPSS version 16.

**Results**

A total of 240 electrocardiograms were obtained from ten dogs. Fifty eight, representing approximately 25% were discarded due to the poor resolutions of the traces resulting from various forms of artifact. Only 36 out of 40 lead II electrocardiograms were analyzed for

traces. A total of 36 ECG were analyzed for arrhythmia. Twenty ECGs representing 55.5% showed various forms of arrhythmias starting from the 8th day of infection. Each of the affected electrocardiograms showed at least one form of arrhythmia. Ventricular premature contractions (VPC), ventricular tachycardia (VT), polymorphic ventricular tachycardia with left bundle branch block (LBBB) morphology, atrioventricular blocks (AVB), notched R wave, ST wave slurring (STS), ST wave depression (STD) and sinoventricular rhythm were displayed by the dogs at different times during the study.

Table 1 shows the weekly variation in the heart rate, R wave voltage, T/R voltage, QRS width, QT width, $QT_C$ width, RR and QT variability indices of the infected control and the *T. brucei* infected dogs. The heart rate of the infected dogs was significantly higher ($P<0.05$) than that of the control on days 8, 16, and 24 post infection. The T/R voltage was also significantly higher ($P<0.05$) in the infected dogs than in the control on days 8 and 24 post infection. However, the QRS width, QTc width, QT variability and RR variability did not differ significantly from the uninfected control dogs on days 8, 16 and 24 post infection.

The result of the study on some serum electrolytes of *T. brucei* infected dogs as shown in Table 2 revealed that the infection in dogs caused a significant change in their mean serum sodium ($P\leq0.001$) and potassium ion concentrations ($P\leq0.01$).The infection however, did not significantly affect mean serum calcium ion concentration of the dogs ($P\geq0.05$). Although the mean serum potassium concentration of the infected dogs on the 8th and 16th day of infection was significantly lower than the concentration of this electrolyte in uninfected dogs, the serum potassium concentration on the 24th day of infection was significantly higher than on the 8th day ($P\leq0.01$) and on the 16th day ($P\leq0.01$).Though the serum sodium level of the infected dogs on days 16 and 24 was significantly higher than the uninfected control, its concentration on the 16th day was significantly higher than that of the 24th day.

When mean serum sodium, calcium, and potassium concentrations of animals with arrhythmia were compared with those without arrhythmia as shown in Table 3, it was found that only the mean serum calcium concentration of arrhythmic animals were significantly higher than of the non-arrhythmic animals. The mean serum potassium and sodium concentration of the arrhythmic animals were not significantly different from non-arrhythmic animals. As shown in Table 4, heart rates of uninfected animals correlated significantly with potassium ($P\leq0.05$, $R=0.86$). A statistically significant correlation was found between potassium and QT width on the 16th and with $QT_C$ on the 8th day of infection. There was also a strong and significant correlation between potassium and R wave voltage on the 8th ($P\leq0.001$; $R= -0.97$) and 16th day of infection ($P\leq0.05$; $R= 0.80$). T/R voltage, $QT_V$, and $RR_V$ correlated significantly with potassium on day 24

Table 5 showed that both QT and $QT_C$ widths correlated strongly and significantly with serum sodium before and on the 16th day of infection. Although serum sodium correlated strongly with RR variability before and on the 16th day of infection, its correlation with heart rate though strong, was not significant ($P\geq0.05$; $R= 0.86$). There was a strong and significant correlation between sodium and $QT_V$ on the 24th day of infection.

In Table 6, it can be seen that there was a negative and insignificant association between either R wave amplitude or QT width and serum calcium level before and during the course of infection. Except for the association between QT width and the serum calcium which was significant only on the 8th day of infection, all other associations between these two ECG variables and serum calcium seen before and throughout the 24 days of infection were not significant ($P>0.05$). Both heart rate and RR variability correlated strongly and significantly with the serum calcium level on the 8th and 16th day of infection. While $QT_C$ correlated significantly with the serum calcium on the 16th and 24th day of infection, the QT variability only correlated significantly with serum calcium on the 24th day.

**Table 1**: Mean (±SD) weekly variation of ECG indices in lead I electrocardiogram of *T. brucei* infected dogs

| Parameter | Control | 8 days PI | 16 days PI | 24 days PI |
|---|---|---|---|---|
| Heart rate (bpm) | 140.00±30.23 (100-180) n=8 | 165.00±31.62* (120-200) n=8 | 191.50±17.78* (180-220) n=8 | 173.33±27.32* (140-200) n=6 |
| R wave voltage(mV) | 0.91±0.55 (0.32-1.50) n=8 | 0.87±0.90 (079-1.00) n=8 | 0.81±0.27 (0.42-1.12) n=8 | 0.90±0.74 (0.32-1.98) n=8 |
| T/R voltage (mV) | 0.22±0.17 (0.11-0.50) n=8 | 0.26±0.02* (0.24-0.28) n=8 | 0.29±0.12 (0.22-0.5) n=8 | 0.38±0.04* (0.33-0.43) n=6 |
| QRS width(sec) | 0.02±0.00 (0.02-0.02) n=8 | 0.03±0.01 (0.02-0.04) n=8 | 0.03±0.01 (0.02-0.04) n=8 | 0.03±0.01 (0.02-0.06) n=8 |
| QT width(sec) | 0.18±0.03 (0.16-0.24) n=8 | 0.16±0.01 (0.15-0.18) n=8 | 0.16±0.01 (0.15-0.18) n=8 | 0.16±0.04 (0.16-0.17) n=6 |
| QTc width(sec) | 0.24±0.02 (0.22-0.28) n=8 | 0.24±0.01 (0.23-0.25) n=8 | 0.25±0.02 (0.22-0.27) n=8 | 0.24±0.01 (0.23-0.25) n=6 |
| QT variability(sec) | 0.03±0.01 (0.02-0.04) n=8 | 0.03±0.01 (0.02-0.04) n=8 | 0.03±0.01 (0.02-0.06) n=8 | 0.03±0.02 (0.00-0.04) n=6 |
| RR variability(sec) | 0.09±0.07 (0.02-0.20) n=8 | 0.08±0.04 (0.04-0.14) n=8 | 0.03±0.01 (0.02-0.04) n=8 | 0.03±0.01 (0.02-0.04) n=6 |

Values of the ECG parameters are expressed as mean ± SD. Values in parentheses are ranges. n=number of animals. PI=post infection, T/R=T voltage. *P<0.05 when compared to the control group

**Table 2:** Mean (±SD) weekly variation in serum electrolytes of *T. brucei* infected dogs

| Parameter | Control | 8 days PI | 16 days PI | 24 days PI |
|---|---|---|---|---|
| Ca (mMoles/L) | 2.91±1.27(8) | 3.76±0.49(8) | 3.89±1.23(8) | 4.66±2.58(8) |
| K (mMoles/L) | 8.00±2.71(8) | 3.54±0.23*(8) | 3.66±0.30*(8) | 8.99±2.65(8) |
| Na(mMoles/L) | 65.00±9.07(8) | ND | 172.29±36.42*(8) | 92.70±48.75*(8) |

Values are Mean ± SD of the number of animals in parenthesis. PI = post infection, ND = not done. *P<0.05 when compared to control group

**Table 3:** Mean (±SD) of serum electrolytes of dogs with or without arrhythmia

| Parameters | Arrhythmic Dogs | Non-Arrhythmic Dogs | P value |
|---|---|---|---|
| Na (mMol/L) | 105.41±50.66(10) | 113.62±64.01(14) | 0.740 |
| Ca (mMol/L) | 4.73±2.08(12) | 3.25±0.99(20) | 0.010 |
| K (mMol/L) | 7.19±3.40(12) | 5.37±2.76(20) | 0.109 |

Values in parentheses are the numbers of observation

**Table 4:** The weekly variation in the correlation indices of various lead I ECG parameters (table 1) and serum potassium concentration (table 2) in *T. brucei* infected dogs

| ECG Parameters | Control | | | 8 days P1 | | | 16 days P1 | | | 24 days PI | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | P | r | n | p | r | n | P | r | n | P | r | n |
| T/R voltage (mV) | 0.12 | -0.64 | 8 | 0.36 | 0.37 | 8 | 0.16 | -0.59 | 8 | 0.00 | 0.98 | 8 |
| R wave voltage (mV) | 0.98 | -0.00 | 8 | 0.00 | -0.97 | 8 | 0.02 | 0.80 | 8 | 0.84 | -0.09 | 8 |
| QT width (sec) | 0.28 | -0.72 | 8 | 0.15 | -0.56 | 8 | 0.00 | 0.90 | 8 | 0.58 | -0.28 | 8 |
| QTc width (sec) | 0.07 | -0.66 | 8 | 0.05 | -0.69 | 8 | 0.29 | 0.43 | 8 | 0.41 | 0.41 | 8 |
| QT variability (sec) | 0.54 | 0.26 | 8 | 0.16 | -0.55 | 8 | 0.11 | -0.61 | 8 | 0.01 | -0.90 | 6 |
| RR variability (sec) | 0.06 | -0.69 | 8 | 0.23 | 0.47 | 8 | 0.07 | 0.67 | 8 | 0.05 | -0.82 | 8 |
| Heart rate (bpm) | 0.01 | 0.86 | 8 | 0.88 | 0.12 | 8 | 0.16 | -0.54 | 8 | 0.71 | 0.16 | 8 |

T/R voltage= T wave voltage, OTc= corrected QT width; P=p value; r= correlation coefficient; n= number of animals

**Table 5:** The weekly variation in the correlation indices of the various lead I ECG parameters (table 1) and serum sodium concentration (table 2) in *T. brucei* infected dogs

| ECG Parameters | Control | | | 16 days P1 | | | 24 days PI | | |
|---|---|---|---|---|---|---|---|---|---|
| | P | r | n | P | r | n | p | R | n |
| T/R voltage (mV) | 0.92 | 0.04 | 8 | 0.61 | 0.21 | 8 | 0.14 | 0.68 | 6 |
| Rwave voltage (mV) | 0.59 | 0.22 | 8 | 0.25 | 0.46 | 8 | 0.54 | 0.26 | 8 |
| QT width (sec) | 0.04 | -0.74 | 8 | 0.001 | 0.92 | 8 | 0.08 | -0.75 | 6 |
| QTc width (sec) | 0.04 | -0.72 | 8 | 0.00 | 0.90 | 8 | 0.13 | 0.69 | 6 |
| QT variability (sec) | 0.85 | -0.08 | 8 | 0.76 | -0.13 | 8 | 0.00 | -0.98 | 6 |
| RR variability (sec) | 0.00 | -0.92 | 8 | 0.00 | 0.92 | 8 | 0.52 | -0.33 | 6 |
| Heart rate (sec) | 0.40 | 0.35 | 8 | 0.31 | -0.41 | 8 | 0.34 | 0.86 | 6 |

T/R voltage=T wave voltage; QTc=heart corrected QT width; P=p value; r=correlation coefficient, n=number of animals

**Table 6:** The weekly variation in the correlation indices of the various lead I ECG parameters (table i) and serum calcium concentration (table 2) in *T. brucei* infected dogs

| ECG Parameters | Control | | | 8 days P1 | | | 16 days P1 | | | 24 days PI | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | P | r | n | p | r | n | P | r | n | p | r |
| T/R voltage (mV) | 0.00 | 0.97 | 8 | 0.35 | 0.38 | 8 | 0.07 | 0.66 | 8 | 0.12 | 0.71 |
| Rwave Voltage (mV) | 0.09 | -0.63 | 8 | 0.20 | -0.51 | 8 | 0.05 | -0.69 | 8 | 0.43 | -0.33 |
| QT width (sec) | 0.24 | -0.47 | 8 | 0.05 | -0.71 | 8 | 0.07 | -0.66 | 8 | 0.09 | -0.73 |
| QTc width (sec) | 0.16 | -0.55 | 8 | 0.20 | -0.51 | 8 | 0.01 | -0.82 | 8 | 0.04 | 0.83 |
| QT variability (sec) | 0.17 | 0.53 | 8 | 0.48 | -0.29 | 8 | 0.27 | 0.44 | 8 | 0.00 | -0.98 |
| RR variability (sec) | 0.30 | -0.42 | 8 | 0.02 | 0.78 | 8 | 0.00 | -0.98 | 8 | 0.85 | 0.09 |
| Heart rate(bpm) | 0.89 | 0.05 | 8 | 0.07 | 0.67 | 8 | 0.02 | 0.81 | 8 | 0.03 | 0.85 |

T/R voltage; T wave voltage, QTc=heart rate corrected QT width; P=p value; r=correlation coefficient; n=number of animals.

**Discussion**

*Trypanosoma brucei* infection as seen in this study caused a perturbation in the electrolyte homeostasis and consequently the electrocardiogram of the infected dogs. The elevated serum potassium level on the 24h day of infection may be due to the damaging effect of *T. brucei* on the kidney and the heart myocardium. Increased serum potassium which has been reported in myocardial ischemia (Kleber, 1987) and renal damage (Segey *et al.*, 2010) could cause a reduction in the resting membrane potential and quicken depolarization (Kline *et al.,* 1992).

Quite often, when pH decreases during myocardial ischemia, the action potential usually shortens due to the enhancement of potassium ion conductance as a result of increased adrenergic stimulation. This effect could be the reason for the tachycardia which some authors have reported in hyperkalemia (Ten-Eick *et al.,* 1992).

It was observed in this study that correlation between heart rate and serum potassium level of infected dogs on day 24 was both weak and insignificant. This may suggest that the mechanism for heart rate increase is independent of serum changes in potassium concentration and could be due to enhanced sympathetic activity which often characterized myocardial infarction and congestive heart failure (Esler & Kaye, 2000; Ten-Eick *et al.* 1992).

This assertion is reflected in the strong relationship between the RR and QT variability index on one side and the serum potassium concentration on the other. The elevated level of serum potassium on the 24th day which was above the reference range for dog appears to account for the reduction in the RR variability index. Reduction in RR variability has been associated with increased heart rate (Hanton & Rabemampianina, 2006).

The hyperkalemia of day 24 has a strong relationship with T wave voltage in this study. In fact, 98% of the variability in the T wave is accounted for by the serum potassium concentration. The presence of high amplitude T wave and sinoventricular rhythms in the course of the infection could be consistent with hyperkalemia (Matu *et al.,* 2000) which is a likely consequence of *T. brucei* myocarditis. This is likely because of the effect of serum potassium on various repolarising currents of the ventricular action potential (Gima & Rudy, 2002).

The elevated serum potassium seen in the infected dogs may also be the reason for some of the arrhythmias observed in this study. Atrioventricular conduction defects, ST segment changes and ventricular premature contractions all of which were seen in this study could be a consequence of hyperkalemia that may develop to ventricular tachycardia, fibrillation or asystole (Kein, 1995; Feldman & Ettinger, 1977).

Another plausible reason for the arrhythmia seen in the infected dogs could be the enhanced level of tissue catecholamines which often characterize acute myocardial ischemia. When tissue catecholamine increases, there is an up regulation of cardiac beta receptors and an associated increase in cyclic adenosine monophosphate (Maisel *et al.,* 1985). The beta receptor activation increases cardiomyocyte intracellular calcium influx (Bean *et al.,* 1984). Increasing intracellular calcium has been reported to cause arrhythmia via the mechanism of delayed after-depolarization (Undrovinas *et al.* 1992) Concurrent hyponatremia and hypocalcemia have often been seen in diseased dogs with hyperkalemia (Lee & Drobat, 2003; Tag & Day 2008). The changes in serum sodium and calcium after *T. brucei* infection may occur secondarily to changes in the serum potassium concentration of the infected dogs. The high serum sodium seen after two weeks in the infected dogs may be due to the renal compensatory response to hyperkalemia (Weiner & Wingo, 1998).The increase in sodium concentration in its self may not be responsible for any ECG change but rather its effect on the acid-base homeostasis may be the reason for the ECG alterations noticed.

The $QT_C$ and QT width correlate positively and significantly with serum sodium at this time. This may be because the increased serum sodium may trigger an acidosis through the $Na^+/H^+$ antiporter through which extracellular sodium is exchanged for intracellular hydrogen. The acidosis has been reported to reduce the sarcolemma sodium current and hence the rapid phase of depolarization of the ventricular action potential (Yatani *et al.*, 1984). This, thus, may increase the duration of the action potential and consequently increase the width of the QT and $QT_C$.

There is an increased intracellular sodium and calcium during myocardial infarction as a result of a decline in ATP production and malfunctioning of the Na/K ATPase pump (Janse & Wit, 1989; Kline *et al.,* 1992). This may explain the decline in serum sodium level on the 24th day of infection.

Serum calcium concentration of arrhythmic dogs in this study was found to be significantly higher than in the non-arrhythmic ones. Atrioventricular blocks, ventricular tachycardia, and sinus arrest have been seen in clinical hypercalcemia. Patel & Antzelevitch (2008) have found increased inhomogeneity in ventricular repolarisation in an experimental model of canine ventricular myocyte with short QT width. This finding could thus explain the relationship between hypercalcemia, shortened QT and arrhythmia

Serum calcium also correlated with the heart rate on the 16th and 24th days post infection. The significant positive correlation between the heart rate and serum calcium level of the *T. brucei* dogs could be because increased serum calcium enhanced the inward moving calcium current while promoting the outward potassium rectifier current, this would shorten the action potential and increase the heart rate.

The QT width has been reported to shorten with hypercalcemia (Carter & Andrus, 1922). The correlation studies on the serum calcium and the QT width in this study may have also confirmed this

earlier report. That is, there is a negative but insignificant correlation between serum calcium and QT width throughout the period of infection. Within the range of 5.5-8.5meq/L of serum calcium, in cattle, a proportional relationship has been found to exist between serum calcium and QT width (Littledike *et al.,* 1976). The positive and significant correlation between calcium and heart rate corrected QT on the 24th day of infection may be attributable to the removal of the heart rate effect.

The result of this study has also shown that 98% of the changes seen in RR and QT variability may have resulted from the dynamism of serum calcium changes. These two indices have been previously reported to be influenced either directly through the sinus node or indirectly through the ventricular myocardium.

In conclusion*, T. brucei* infection affected the serum electrolytes concentration and may have caused ECG changes through mechanism that involves the modulatory responses of the autonomic nervous system and the ion channels.

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