Electrocardiographic changes associated with dehydration in rabbits (Oryctolagus cuniculus)

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ABSTRACT
A study was conducted on twelve healthy and clinically normal New Zealand White rabbits of either sex, weighing between 2-3 kg and aged between 1-2 years to observe the electrocardiographic changes during induced dehydration. Progressive dehydration till about 10% body weight loss was monitored that reflected in increased haematocrit and serum potassium levels. Increase in heart rate was seen with progressive water deprivation. Electrocardiographic changes observed were reflective of those associated with hyperkalaemia and include a significant increase in QRS complex and T wave amplitude along with an increase in the duration of all the waves. These characteristic changes were fairly indicative of the hydration status, more precisely on the electrolyte balance that can be assessed for an early recognition and clinical intervention.

Key words: Dehydration, Electrocardiography, Hyperkalaemia, Rabbit.

In Physiology and Medicine, dehydration is the excessive loss of body water. In the dehydrated state, it impacts unfavourably upon cardiovascular function, including cardiac output and peripheral blood flow (Gonzalez-Alonso et al., 1998). The most prominent manifestation of water deprivation is loss of body weight and decrease in both blood and plasma volumes (Aarseth and Klug, 1972). Since dehydration is invariably associated with electrolyte imbalance, it was envisaged that these disparities could possibly affect the conduction changes of the heart, and be reflected within the electrocardiogram (ECG). El-Sherif and Turitto (2011) opined that depending on the particular type of electrolyte disorder, alterations of cardiac current kinetics may promote proarrhythmic or antiarrhythmic effects, and expression of the same fundamental electrophysiological principles that underlie the normal electrical behaviour of the heart.

The present report records ECG changes associated with progressive dehydration in rabbits attempting to elucidate the plausible electrophysiological changes in the light of electrophysiological changes.

Twelve healthy and clinically normal New Zealand White rabbits of either sex, weighing between 2-3 kg and aged between 1-2 years were used to observe the electrocardiographic changes during induced dehydration. The experiment was approved by the Institutional Animal Ethics Committee (IAEC).

Prior to ECG recordings, the animals were properly restrained and were allowed to stand for 10-15 mins to familiarize it with the experimental ambience. The ECG recordings were made in sternal recumbency as per the method described by Tilley (1979) using a multichannel electrocardiograph (NASAN, NE-3I). Three bipolar standard limb leads (I, II and III) and three unipolar augmented limb leads (aVR, aVL, and aVF) were used to record the electrocardiogram. The ECG machine was calibrated to give 20mm deflection per mv of input and recordings were traced with a paper speed of 50mm/second. Site for attachment of electrodes were trimmed with scissors and cardiac gel was applied to increase conductivity. Electrodes were attached with small crocodile clips with flattened teeth as previously described by Ahmed (2002) and were attached directly to the animal’s skin proximal to the olecranon on the caudal aspect of the appropriate forelimb, and over the patellar ligament on the cranial aspect of appropriate hind limb. To compare the ECG changes for induced water deprivation, a normal electrocardiogram was recorded beforehand to ensure that the animal was healthy as per Ahmed et al. (2008).

Ear vein puncture of the rabbits was done for whole blood and serum collection. Packed cell volume (PCV) was calculated using an automated blood cell counter and serum potassium (K+) was estimated by the turbidometric method using commercially available diagnostic kit (Lifechem, Kamineni Life Sciences Pvt. Ltd., Hyderabad) following manufacturer’s instructions. Paired PCV and K+ samples were evaluated taken before and during dehydration.

Dehydration was induced in the normal rabbits by keeping them off water but not pelleted feed for 24 hours.

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An approximate 10% decrease in body weight was evaluated for each animal and an electrocardiographic recording was also traced immediately. The corresponding packed cell volume and serum potassium concentration was estimated from each rabbit.

Progressive dehydration was recorded in the rabbits monitored every two hours in a 24 hour period. As was expected, with progressive water deprivation there was a decrease in the body weight of animals apparently due to dehydration.

Haematocrit values were observed to proportionately increase with dehydration, together with increase in serum potassium values. For an average 10% (± 0.42) loss in body weight, haematocrit from baseline: 32.58 ± 0.51 (%) increased to 40.67 ± 0.98 (%); serum potassium concentration from baseline: 3.04 ± 0.19 (mmol/l) significantly increased (P<0.01) to 4.08 ± 0.26 (mmol/l) (Fig. 1).

Consequent upon increased haemo-concentration, there was a resultant increase in the heart rate of the animals as evident in the ECG from a mean 208 ± 4.797 bpm to a mean of 214 ± 5.995 bpm. The changes in the electrocardiogram during the dehydrated state can be mostly explained by the serum ionic changes, mostly potassium (K⁺). Reportedly however, the serum K⁺ concentration at which electrocardiographic changes develop is somewhat variable (Wrenn et al., 1991; Aslam et al., 2002).

During dehydration a significant increase in QRS complex and T wave amplitude (Fig. 2, Table 1) was observed along with an increase in the duration of all the waves (Table 2). It is reported that the classical tall, peaked T waves, reduced amplitude and eventually loss of the P wave and marked widening of the QRS complex are seen in one of five hypokalemic human patients with serum potassium levels usually above 5.5-6.5 mmol/l (Slovis and Jenkins, 2002). In a study by Peoples et al. (2009) no alterations in cardiac electrophysiology was observed in men with significantly increased serum sodium and potassium levels at 7% water deficiency. Although the factors influencing the effect of serum potassium levels on cardiac electrophysiology are not entirely understood, the concentrations of other electrolytes,
Table 1: Amplitude (mv) of various electrocardiographic waves in rabbits under induced dehydration (Mean ± SE).

<table>
<thead>
<tr>
<th>Leads</th>
<th>P(B)</th>
<th>P(D)</th>
<th>QRS(B)</th>
<th>QRS(D)</th>
<th>T(B)</th>
<th>T(D)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0.040 ± 0.005</td>
<td>0.045 ± 0.003</td>
<td>0.097 ± 0.011</td>
<td>0.168 ± 0.034*</td>
<td>0.148 ± 0.019</td>
<td>0.271 ± 0.017**</td>
</tr>
<tr>
<td>II</td>
<td>0.042 ± 0.004</td>
<td>0.053 ± 0.002</td>
<td>0.114 ± 0.014</td>
<td>0.167 ± 0.021**</td>
<td>0.125 ± 0.010</td>
<td>0.176 ± 0.025**</td>
</tr>
<tr>
<td>III</td>
<td>0.032 ± 0.005</td>
<td>0.050 ± 0.000</td>
<td>0.117 ± 0.015</td>
<td>0.168 ± 0.019**</td>
<td>0.100 ± 0.000</td>
<td>NT</td>
</tr>
<tr>
<td>aVR</td>
<td>0.043 ± 0.004</td>
<td>0.048 ± 0.005</td>
<td>0.122 ± 0.012</td>
<td>0.168 ± 0.024</td>
<td>0.132 ± 0.007</td>
<td>0.199 ± 0.018*</td>
</tr>
<tr>
<td>aVL</td>
<td>0.022 ± 0.002</td>
<td>0.070 ± 0.000</td>
<td>0.057 ± 0.017</td>
<td>0.157 ± 0.017**</td>
<td>0.166 ± 0.030</td>
<td>0.149 ± 0.030</td>
</tr>
<tr>
<td>aVF</td>
<td>0.044 ± 0.004</td>
<td>NT</td>
<td>0.075 ± 0.011</td>
<td>0.150 ± 0.000**</td>
<td>0.050 ± 0.000</td>
<td>NT</td>
</tr>
</tbody>
</table>

*P<0.05; **P<0.01; NT-Not traceable; B-Base; D-Dehydration

Table 2: Duration (sec) of different electrocardiographic waves in rabbits under induced dehydration (Mean ± SE).

<table>
<thead>
<tr>
<th>Leads</th>
<th>P (B)</th>
<th>P (D)</th>
<th>PR (B)</th>
<th>PR (D)</th>
<th>QRS (B)</th>
<th>QRS (D)</th>
<th>QT (B)</th>
<th>QT (D)</th>
<th>T (B)</th>
<th>T (D)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0.030 ± 0.003</td>
<td>0.029 ± 0.002</td>
<td>0.048 ± 0.002</td>
<td>0.056 ± 0.002*</td>
<td>0.044 ± 0.003</td>
<td>0.043 ± 0.002</td>
<td>0.125 ± 0.005</td>
<td>0.144 ± 0.003**</td>
<td>0.070 ± 0.004</td>
<td>0.086 ± 0.002**</td>
</tr>
<tr>
<td>II</td>
<td>0.020 ± 0.000</td>
<td>0.030 ± 0.002**</td>
<td>0.045 ± 0.006</td>
<td>0.050 ± 0.002</td>
<td>0.045 ± 0.002</td>
<td>0.047 ± 0.002</td>
<td>0.125 ± 0.007</td>
<td>0.144 ± 0.002</td>
<td>0.072 ± 0.001</td>
<td>0.092 ± 0.002**</td>
</tr>
<tr>
<td>III</td>
<td>0.020 ± 0.000</td>
<td>NT</td>
<td>0.051 ± 0.003</td>
<td>NT</td>
<td>0.035 ± 0.002</td>
<td>0.040 ± 0.000**</td>
<td>0.120 ± 0.005</td>
<td>NT</td>
<td>0.073 ± 0.008</td>
<td>NT</td>
</tr>
<tr>
<td>aVR</td>
<td>0.024 ± 0.002</td>
<td>0.031 ± 0.002</td>
<td>0.053 ± 0.003</td>
<td>0.051 ± 0.002</td>
<td>0.051 ± 0.002</td>
<td>0.052 ± 0.002</td>
<td>0.120 ± 0.000</td>
<td>0.144 ± 0.003**</td>
<td>0.0800 ± 0.0000</td>
<td>0.086 ± 0.003</td>
</tr>
<tr>
<td>aVL</td>
<td>0.020 ± 0.000</td>
<td>0.040 ± 0.000**</td>
<td>0.034 ± 0.002</td>
<td>0.060 ± 0.000**</td>
<td>0.051 ± 0.003</td>
<td>0.040 ± 0.000</td>
<td>0.134 ± 0.003</td>
<td>0.123 ± 0.006</td>
<td>0.065 ± 0.003</td>
<td>0.060 ± 0.003</td>
</tr>
<tr>
<td>aVF</td>
<td>0.040 ± 0.000</td>
<td>NT</td>
<td>0.053 ± 0.002</td>
<td>NT</td>
<td>0.040 ± 0.000</td>
<td>NT</td>
<td>0.120 ± 0.000</td>
<td>NT</td>
<td>0.080 ± 0.000</td>
<td>NT</td>
</tr>
</tbody>
</table>

*P<0.05; **P<0.01; NT-Not traceable; B-Base; D-Dehydration
as well as levels of catecholamines, are said to play a major role (Surawicz, 1967; Leitch and Patterson, 1994). Potassium is specifically needed for terminating action potentials and contractions and initiating repolarization. Plausibly, electrocardiographic changes due to elevated potassium levels could be mediated via specific voltage-gated potassium channels that cause increased membrane repolarization of the cardiac action potential resulting in T wave peaks.

Hyperkalemia thus could result in reduced electrical conduction and often leads to disrupted heart rhythm. They are also believed to cause an overall membrane depolarization, inactivating the sodium channels causing a sluggish conduction of the electrical wave around the heart, which leads to smaller P waves and widening of the QRS complex. In human patients, potassium concentration above 6.5-7.5 mmol/l causes the P wave to widen and flatten or may disappear and the PR segment lengthens. At concentration of 7.0-8.0 mmol/l, the QRS widens uniformly affecting all portions of the complex and begin to merge with the T wave creating a sine wave pattern, unlike right or left bundle branch blocks that act on the terminal forces (Slovis and Jenkins, 2002).

In the present study however, no change was observed in P wave amplitudes, which nevertheless are reported in some mild to moderate hyperkalemic instances. The amplitude of QRS waves increased significantly in all the leads in the dehydrated state as would be expected and similar to earlier reports observed in anterior chest leads as reported by Saltykova et al. (2007). It could be understood that during dehydration, increase in QRS voltage may arise due to lower chest tissue electroconductivity. The amplitude of T waves increased significantly and peaked T waves were also observed as a result of hyperkalemia. Additionally, significant increase in the duration of P wave, P-R segment and QRS complex because of bundle branch blocks, QT interval, and lastly the T wave resulted due to increased ventricular repolarization.

In the present study, no significant change in the cardiac axis was noticed before (90° ± 0.617) and after dehydration (88° ± 0.708).

CONCLUSION

The electrophysiological reflections with progressive dehydration in rabbits were persistent with those reported with hyperkalaemia changes. These changes were fairly indicative and with clinical support could be suggestive of the hydration status and more precisely the electrolyte balance in the subject and may provide useful diagnostic clues for prompt recognition and timely intervention.

REFERENCES


