Original Research Article

Acute Effect of Intravenous Ethanol on Electrocardiogram of New Zealand White Rabbits

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ABSTRACT

The present study was undertaken to study the effects of intra-venous (i.v.) ethanol (25%, 50% and 100%) on Electrocardiogram of New Zealand White rabbits. Twelve healthy and clinically normal New Zealand White rabbits of either sex, weighing between 2-3 kg and aged between 1-2 years were selected for the present study. 1 ml of ethanol (25%, 50% and 100%) was injected i.v. in the ear vein of the rabbit and ECG recordings were taken for 50 minutes. Rabbits exhibited a significant decrease in heart rate following the injection of 25% and 50% ethanol and the heart rate remained significantly (P<0.01) depressed for a period of 30 minutes. Low P-wave amplitude was observed. QRS amplitudes have increased significantly in 25% and 50% ethanol treated group. Increased QRS voltage, and prolonged ventricular activation time, likely represents left ventricular hypertrophy. But QRS amplitude has decreased significantly in 100% ethanol treatment. T wave amplitudes significantly increase in 25% and 50% treatment group and decrease in 100% treatment group. QT interval indicating the total duration of electrical activity in the ventricles was significantly increased compared with that of control group (50% treated group).

Keywords
Electrocardiogram, Ethanol, New Zealand White rabbit

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Introduction

Ethanol is known to exert significant effects on the electrical characteristics of the myocardium (Takeda et al., 1984). Ingestion of ethanol causes disturbances of cardiac rhythm and conduction (Marriott and Myerburg, 1974; Ettinger et al., 1976). Bollinger, who introduced the term ‘Munich beer heart’ in describing cardiac dilatation and hypertrophy secondary to chronic alcoholism (Bollinger, 1884), since then in vivo and in vitro effects of ethanol on heart have extensively been investigated (Altura and Atura, 1989). The study of electrocardiogram (ECG) pattern is very useful in the detection of abnormal heart conditions (Venkateshwarlu et al., 1977). Alcohol consumption causes ECG changes which include cardiac conduction...
abnormalities, prolongation of the QT interval, prolongation of ventricular repolarization and sympathetic stimulation (Rossinen et al., 1999). Sinus tachycardia or a supraventricular arrhythmia, commonly atrial fibrillation and non-specific ST-T changes are also observed in alcoholics (Klatsky, 1996). Alcohol alters the endocrinal function like increase in adrenocorticotrophic hormone, oxytocin and electrolytes, which may indirectly causes myocardial damage (Langer, 1992). Therefore, the present study was undertaken to study the acute effects of i.v. ethanol (25%, 50% and 100%) on electrocardiogram of rabbit.

Materials and Methods

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 before and after treatment. The experiment was approved by the Institutional Animal Ethics Committee (IAEC). Prior to ECG recording, the animals were properly restrained and were allowed to stand for 10-15 minutes 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. Then 1 ml of ethanol (25%, 50% and 100%) was injected i.v. in the ear vein of the rabbit and ECG recordings were taken for 50 minutes. To compare the ECG changes after injection of 25%, 50% and 100% ethanol, normal electrocardiogram was recorded beforehand to ensure that the animal was healthy as per Ahmed et al., (2008).

Results and Discussion

Heart rate

Rabbits exhibited a significant decrease in heart rate (Table 1) following the addition of 25% and 50% ethanol, and the heart rate remained significantly (P<0.01) depressed for 30 mins period. Similarly, ethanol has been previously shown to slow the heart rate of the invertebrate Ciona intestinalis (Pope and Rowley, 2002). Ethanol is known to elicit a biphasic response, with stimulatory effects produced at lower doses and inhibitory effects at higher doses (Earleywine and Martin, 1993). In the present study addition of 25% and 50% ethanol may influence vagal centres of the central nervous system and resulted bradycardia (Samonia and Hakumaki, 1982). Again injection of 100% ethanol significantly (P<0.01) increases heart rate, ethanol may inhibit ACh release and decreases cholinergic transmission in these inhibitory neurons, causing increase in the heart rate of rabbits (Carmichael and Israel, 1975; Erickson and Graham, 1973).

Cardiac Axis

QRS frontal axis in degrees were 90.00±0.258, 89.00±0.258 and 87.50±0.342 before ethanol injection and 87.66±0.494, 86.00±0.142 and 86.00±0.258 after injection of 25%, 50% and 100%
ethanol, respectively (Table 2). Similar findings were also observed by Swathi and Nasar Ahamed (2014) in alcoholic people. Ramanna et al., (2014) found mean (±SD) QRS frontal axis 63.10° ± 28.70° during exercise in control and 58.60° ± 31.00° in alcoholic persons.

Amplitude of electrocardiographic waves

“P” waves of amplitude have decreased significantly in all the treated groups (Table 3). Swathi and Nasar Ahamed 2014) also found slight decrease in P wave amplitude in alcoholics though it was not statistically significant. Venkatesh (2011) also did not observe any significant difference in values of P wave duration and amplitudes. In our present study, low P-wave amplitude may be associated with low LA (Left atrial) voltage, displaced inter-atrial conduction, and slower conduction velocity, which contributes to short wave lengths and makes the tissue vulnerable to fibrillation (Park et al., 2016).

QRS amplitudes have increased significantly in ethanol treated group (25%, 50%). Increased QRS voltage, and prolonged ventricular activation time, likely represents left ventricular hypertrophy in a high percentage of cases (Manning and Smithley, 1964). But QRS amplitude has decreased significantly in 100% ethanol treatment. T wave amplitudes significantly increase in 25% and 50% treatment group and decrease in 100% treatment group. It may be due to alterations of sympathetic tone (Kitchin and Neilson, 1972). Robertson (1955) stated that low voltage of the T waves might be due to early myocardial degeneration or coronary sclerosis.

Duration of electrocardiographic waves

Duration of electrocardiographic waves has been presented in Table 4. Lorsheyd et al., (2005) showed prolongation of the PR interval and QRS complex after acute ingestion of alcohol.

### Table.1 Heart rates after treatment of ethanol (25%), ethanol (50%) and ethanol (100%) (Mean ± SE)

|                      | Control        | 10 min         | 20 min         | 30 min         | 40 min         | 50 min         |
|----------------------|----------------|----------------|----------------|----------------|----------------|----------------|
| Ethanol (25%) treated group | 213.00±0.683   | 200.00±0.291** | 202.500±1.708**| 200.667±0.882**| 200.000±2.582**| 214.333±1.308**|
| Ethanol (50%) treated group | 225.83±3.270   | 225.83±3.270   | 214.33±1.308   | 209.17±3.060** | 211.83±1.515*  | 215.17±0.980*  |
| Ethanol (100%) treated group | 213.00±1.308   | 213.00±0.719   | 225.00±3.416*  | 225.00±3.270*  | 214.00±1.167   | 212.00±2.716   |

### Table.2 Cardiac axes after treatment with ethanol (25%), ethanol (50%) and ethanol (100%) (Mean ± SE)

|                      | Control        | 10min           | 20 min         | 30 min         | 40 min         | 50 min         |
|----------------------|----------------|-----------------|----------------|----------------|----------------|----------------|
| Ethanol (25%) treated group | 90.000±0.258   | 83.167±0.401**  | 90.000±0.342   | 87.667±0.494** | 89.500±0.342   | 89.000±0.258   |
| Ethanol (50%) treated group | 89.000±0.258   | 89.000±0.258    | 88.000±0.101   | 86.000±0.142** | 85.000±0.258** | 85.000±0.258** |
| Ethanol (100%) treated group | 87.500±0.342   | 84.50±0.342**   | 84.50±0.342**  | 86.00±0.258*  | 88.50±0.342    | 86.50±0.342    |

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### Table 3: Amplitude of waves after treatment of ethanol (25%), ethanol (50%) and ethanol (100%) (Mean ± SE)

| Waves | Control | 10min | 20 min | 30 min | 40 min | 50 min |
|-------|---------|-------|--------|--------|--------|--------|
| P     | 0.050±0.00258 | 0.0250±0.003** | 0.050±0.00258 | 0.050±0.00258 | 0.0400±0.00258 | 0.0250±0.00342** |
| QRS   | 0.145±0.00342 | 0.300±0.00516** | 0.300±0.00516** | 0.287±0.00882** | 0.248±0.00654** | 0.200±0.00516** |
| T     | 0.650±0.00342 | 0.150±0.00258** | 0.165±0.00342** | 0.650±0.00342 | 0.108±0.00543** | 0.950±0.00342** |
| P     | 0.050±0.0003   | 0.050±0.003    | 0.050±0.003    | 0.025±0.003**  | 0.020±0.003**   | NT     |
| QRS   | 0.21±0.0007    | 0.29±0.007**   | 0.24±0.007**   | 0.29±0.007**   | 0.19±0.003   | 0.29±0.00558** |
| T     | 0.09±0.0007    | 0.05±0.003**   | 0.13±0.003**   | 0.15±0.003**   | 0.18±0.003** |
| P     | 0.051±0.00271  | 0.050±0.003    | 0.070±0.003**  | 0.222±0.007*   | 0.200±0.005** | 0.150±0.003** |
| QRS   | 0.242±0.00543  | 0.177±0.00494**| 0.290±0.003**  | 0.290±0.003**  | 0.150±0.003** |
| T     | 0.115±0.0003   | 0.100±0.003**  | 0.105±0.003**  | 0.105±0.003**  | 0.095±0.003** |

**NT**-Not traceable

### Table 4: Duration of waves after treatment of ethanol (25%), ethanol (50%) and ethanol (100%) (Mean ± SE)

| Waves | Control | 10min | 20 min | 30 min | 40 min | 50 min |
|-------|---------|-------|--------|--------|--------|--------|
| P     | 0.030±0.00258 | 0.250±0.00342 | 0.0300±0.00258 | 0.0300±0.00258 | 0.0300±0.00516 | 0.0250±0.00342** |
| PR    | 0.040±0.00258 | 0.0450±0.00342 | 0.0500±0.00258 | 0.0500±0.00258 | 0.0500±0.00258 | 0.0500±0.00258 |
| QRS   | 0.0300±0.00258 | 0.0300±0.00258 | 0.0400±0.00258 | 0.0300±0.00258 | 0.0400±0.00258 | 0.0400±0.00258 |
| QT    | 0.140±0.00258 | 0.145±0.00342 | 0.150±0.00258 | 0.150±0.00258 | 0.150±0.00258 | 0.150±0.00258 |
| T     | 0.0550±0.00342 | 0.0550±0.00342 | 0.0917±0.00342** | 0.0500±0.00258 | 0.0800±0.00258** | 0.0600±0.00258 |
| P     | 0.04±0.0003 | 0.04±0.003 | 0.03±0.003 | 0.04±0.003 | 0.03±0.003 | NT |
| PR    | 0.06±0.0003 | 0.05±0.003 | 0.05±0.003 | 0.05±0.003 | 0.06±0.003 | NT |
| QRS   | 0.03±0.0003 | 0.03±0.003 | 0.03±0.003 | 0.03±0.003 | 0.03±0.003 | 0.03±0.003 |
| QT    | 0.12±0.0003 | 0.11±0.004 | 0.13±0.003 | 0.14±0.003** | 0.16±0.0038 | 0.15±0.003** |
| T     | 0.06±0.0003 | 0.06±0.003 | 0.05±0.003 | 0.08±0.003** | 0.10±0.003** | 0.08±0.003** |
| P     | 0.030±0.0003 | 0.025±0.0003 | 0.040±0.0003 | 0.050±0.003** | 0.040±0.003 | 0.030±0.003 |
| PR    | 0.060±0.0003 | 0.050±0.003 | 0.060±0.003 | 0.070±0.003 | 0.060±0.003 | 0.060±0.003 |
| QRS   | 0.030±0.0003 | 0.030±0.003 | 0.040±0.003 | 0.030±0.003 | 0.040±0.003 | 0.040±0.003 |
| QT    | 0.140±0.0003 | 0.120±0.003** | 0.140±0.003 | 0.150±0.003 | 0.150±0.003 | 0.150±0.003 |
| T     | 0.080±0.0003 | 0.040±0.003** | 0.070±0.003 | 0.080±0.003 | 0.080±0.003 | 0.090±0.003 |

**NT**-Not traceable
In contrast there is no change of P, P-R interval and QRS complex measurements in our study. The PR interval reflects the time needed to activate the atria, to conduct the impulse to the AV node and His bundle and start the ventricular depolarization. QRS complex is because of ventricular depolarization. QT interval in seconds was 0.12±0.003; 0.14±0.003 in normal rabbits and 0.15±0.003; 0.15±0.003 in (50% and 100%) ethanol treated animals.

Thus, QT interval was significantly increased, compared with that of control group (50% treated group). QTC interval in the electrocardiogram includes both ventricular depolarization and repolarization times and varies inversely with the heart rate (Lorsheyd et al., 2005). Alcohol consumption causes ECG changes which include cardiac conduction abnormalities, prolongation of the QT interval, prolongation of ventricular repolarization and sympathetic stimulation (Rossinen et al., 1999). T wave duration were (0.0550±0.00342, 0.06±0.003; 0.08±0.003) in normal rabbits and (0.0600±0.00258; 0.08±0.003 and 0.09±0.003) in 25%, 50% and 100% treated group respectively.

Therefore, it can be concluded from the present study that ethanol has effect on heart rate controlling and Electrocardiogram of New Zealand White rabbits.

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