ELECTROCARDIOGRAPHIC CHANGES AFTER ETHANOL AND METHANOL ADMINISTRATION IN ALBINO RATS

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Summary : As an acute effect, both ethanol and methanol prolong P-R, Q-T and ST intervals in rat electrocardiogram. The R wave amplitute showed a marked increase with methanol. Other ECG abnormalities like S-T segment changes, appearance of Iso-electric S-T segment, inverted 'T' wave, ventricular ectopic and wandering pace maker were more common in methanol treated animals.

Key words : rat ECG methanol ethanol

INTRODUCTION

Information pertaining to the electrocardiographic (ECG) changes due to acute effects of ethanol in rat is scanty and moreover practically no literature is available on the effects of methanol on rat ECG. Hence a preliminary investigation was undertaken to study the acute effects of these alcohols on rat ECG.

MATERIAL AND METHODS

Healthy wistar strain albino rats of either sex (110-230 *g* body weight) were used for this study. The overnight starved animals were anaesthetized with chloralose (8 *mg*/100 *g*) by intraperitoneal route. Ethanol and methanol (200 *mg*/100 *g*) were given orally in two groups of animals (n-10) and saline treated animals served as controls. ECG was recorded every 15 min in lead II in supine position at a paper speed of 50 *mm*/sec upto 75 min.

ECG Recordings were analysed for heart rate, P-R, Q-T, S-T, and QRS intervals, R wave amplitude and various abnormal waves with special emphasis on electrolyte induced abnormalities. Students 'T' test was used for the statistical analysis of the data.

RESULTS

Ethanol administration significantly decreases heart rate (Table I) after 70 min where as methanol effect was noticed at 60 *min* ($P<0.005$). The P-R interval was prolonged at all periods of observation in both the groups of animals (Table II to VI).

TABLE I : Heart rate changes after ethanol and methanol.

	15 min	*30 min*	*45 min*	*60 min*	*75 min*
Controls (n=20)	343 ±10.6	340 ±8.4	345 ±12	344 ±8.4	344 ±10.6
Ethanol treated group n=10	368 ± 9.6	354 ±17.5	358 ±12.0	358 ±12.6	291 ±13.7
Methanol treated group n=10	331 ±18.7	336 ±10.8	304 ±18.0	296* ±5.8	250* ±10.4

Values are mean ± SE
*$P<0.005$.

Q-T and S-T intervals as well as the QRS duration were prolonged in both the experimental groups. The R wave amplitude showed a fall at 15 and 30 min in ethanol group, whereas in methanol treated animals R wave amplitude was increased at all periods of observation ($P<0.05$) except at 15 min (Table II to VI).

TABLE II : Changes in rat ECG intervals after ethanol and methanol at 15 min.

	P-R Interval (sec)	*Q-T (sec)*	*S-T (sec)*	*QRS (sec)*	*Amplitude of 'R' wave (mv)*
Controls n=20	0.048 ±0.0012	0.077 ±0.0029	0.051 ±0.002	0.015 ±0.001	0.06 ±0.025
Ethanol group n=10	0.052 ±0.0017 P<0.05	0.077 ±0.0017 P<0.05	0 06 ±0.002 P<0.05	0 0157 ±0.0018	0.478 ±0 019 (P<0.05)
Methanol group n=10	0.052 ±0.003 P<0.05	0.082 ±0 0082 P<0.05	0.064 ±0.0075 P<0.05	0.0157 ±0.0017	0.6 ±0.048

Values are mean ± SE.

TABLE III : Changes in rat ECG intervals after ethanol and methanol at 30 min.

	P-R (*sec*)	*QT* (*sec*)	*ST* (*sec*)	*QRS* (*sec*)	*R wave amplitude* (*mv*)
Controls n=20	0.046 ±0.001	0.065 ±0.001	0.05 ±0.0019	0.015 ±0.0019	0.6 ±0.025
Ethanol treated group n=10	0.052 ±0.0017 P<0.05	0.087 ±0.0026 P<0.05	0.0671 ±0.0026 P<0.05	0.0185 ±0.0013 P<0.05	0.485 ±0.0401 P<0.05
Methanol treated group n=10	0.055 ±0.00187 P<0.05	0.090 ±0.007 P<0.05	0.068 ±0.005 P<0.05	0.0185 0.0013 P<0.05	0.671 ±0.051

Values are mean ± SE.

TABLE IV : Changes in rat ECG intervals after ethanol at 45 min.

	P–R (*sec*)	*QT* (*sec*)	*S–T* (*seg sec*)	*QRS* (*sec*)	*R wave amplitude* (*mn*)
Controls n=20	0.044 ±0.04	0.062 ±0.002	0.05 ±0.001	0.015 ±0.001	0.6 ±0.02
Ethanol group n=10	0.051 ±0.0024 P<0.05	0.085 ±0.006 P<0.05	0.0685 ±0.0055 P<0.05	0.0185 ±0.0013 <P0 05	0.564 ±0.041
Methanol group n=10	0.055 ±0.0027 P<0.05	0.082 ±0.0026 P<0.05	0.062 ±0.0017 P<0.05	0 02 ±0.002 P<0.05	0.714 ±0.041 P<0.05

Values are mean ± SE.

TABLE V : Changes in raf ECG intervals after ethanol and methanol at 60 min.

	P—R (*sec*)	*QT* (*sec*)	*S—T* (*sec*)	*QRS* (*sec*)	*R wave amplitude* (*mv*)
Controls n=10	0.048 ±0.001	0.06 ±0.0012	0.058 ±0.002	0.016 ±0.0018	0.59 ±0.025
Ethanol group n=10	0.054 ±0.0027 P<0.05	0.0857 ±0.006 P<0.05	0.067 ±0.005 P<0.05	0.0185 ±0.0013 P<0.05	0.578 ±0.0265
Methanol group n=10	0.052 ±0.0026 P<0.05	0.087 ± 0039 P<0.05	0.067 ±0.039 P<0.05	0 02 ±0 002 P<0.05	0 671 ±0 05 P<0 05

Values are mean ± SE.

TABLE VI : Changes in rat ECG intervals after ethanol and methanol at 75 min.

	P—R (*sec*	*QT* (*sec*)	*S—T* (*sec*)	*QRS* (*sec*)	*R wave amplitude* (*mv*)
Controls n=10	0 047 ±0 0019	0.056 ±0.001	0 057 ±0.002	0.016 ±0.002	0 52 ±0 025
Ethanol group n=10	0 0542 ±0 0018 P<0 05	0 0928 ±0 0048 P<0 05	0 0742 ±0.0053 P<0.05	0 0185 ±0.0013 P<0 05	0 557 ±0 0213 P<0.05
Methanol group n—10	0 052 ±0.0076 P<0 05	0.097 ±0 0043 P<0.05	0 078 ±0 0037 P<0.05	0 0185 ±0 0013 P<0.05	0.692 ±0 0468 P<0.05

Values are mean ± SE.

Though changes in 'P' wave configuration, S-T segment, appearance of isoelectric S-T segment, inverted T wave, J-Point depression, ventricular ectopic and wandering pace maker were noticed occasionally in both the experimental groups, they were relatively more common in methanol treated animals (Fig. 1).

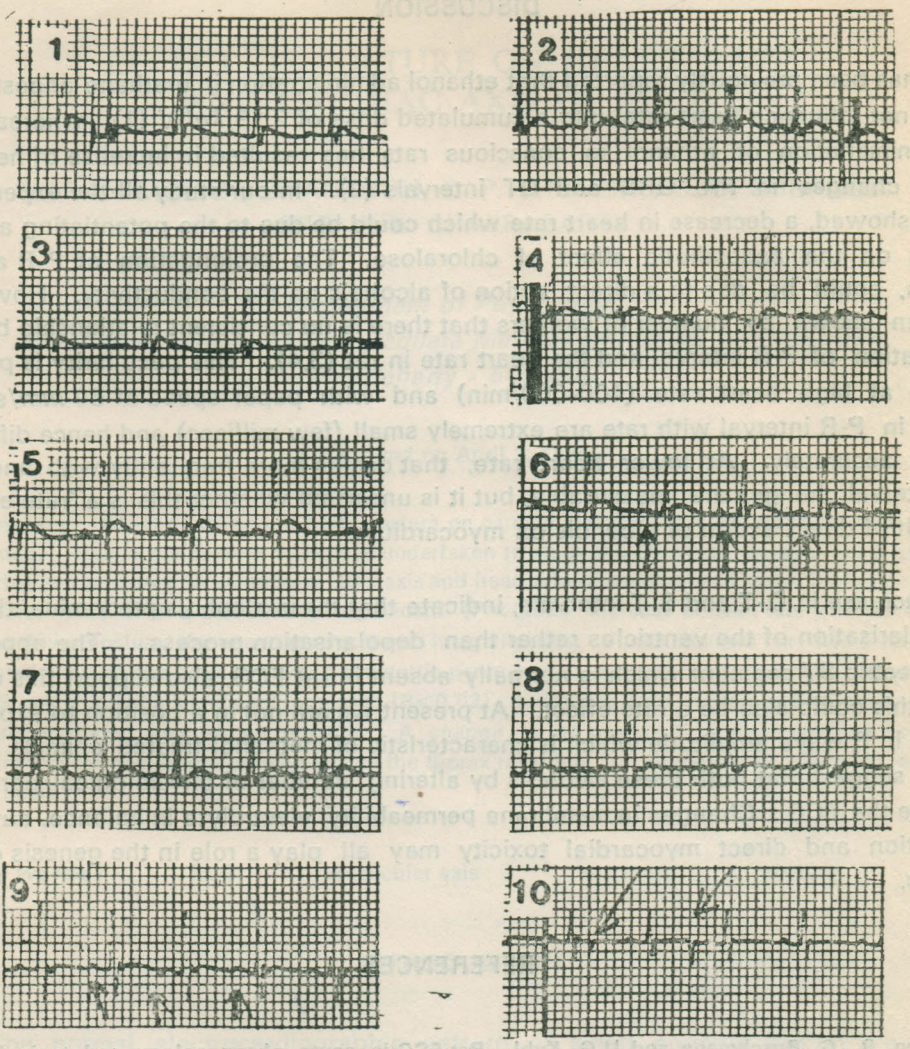

Fig. 1 : ECG of Control animals — Recor d taken at 30 minutes, (2) Effect of Ethanol — 30 minutes — Note the slight dacrease in R wave amplitude and slight increase in heart rate, (3) Effect of Ethanol — 75 minutes — Note the decrease in heart rate and prolongation of all the intervals, (4) Effect of Methanol — 30 minutes — Note the prolongation of P—R interval, (5) Effect of Methanol — 75 minutes — Note the increase of R wave amplitude and prolongation of all the intervals, (6) P wave abnormality, (7) Iso-electric S-T segment, (8) Auricular fibrillation, (9) Wandering pacemaker and (10) Ventricular ectopics.

Effects shown in 6, 7, 8, 9, and 10 are seen with both the alcohol but more common with Methanol.

DISCUSSION

It has been previously reported that ethanol administration in urethane anaesthetized rats did not influence heart rate upto a cumulated dose of 2.19/*kg*/IV (1), whereas acute oral administration of ethanol in conscious rats has resulted in increase of heart rate without changes in PR, QRS and QT intervals (2). In our study all the experimental animals showed, a decrease in heart rate which could be due to the potentiating action of alcohols on the anaesthetic effect of chloralose. The prolongation of P-R and Q-T intervals, could be due to a direct action of alcohols on the conductivity. However, it has been shown by number of workers that there is no consistent relationship between the duration of P-R interval and the heart rate in rat (3,4). This peculiarity is probably because at high heart rate (400-600/min) and with paper speed of 50 *mm*/sec, any change in P-R interval with rate are extremely small (few millisec) and hence difficult to measure accurately. At lower heart rate, that occurs more frequently with anesthesia these interval changes are measurable, but it is uncertain whether this is a 'rate effect' or the result of direct action of alcohols on myocardium.

Increase in Q-T and S-T intervals, indicate that these alcohols predominantly affect the repolarisation of the ventricles rather than depolarisation process. The appearance of iso-electric ST segment which is normally absent in rat ECG, also support this concept though this could also be a rate effect. At present we are not in a position to explain the increase in R wave amplitude which is characteristic of methanol treated animals. These findings suggest that both these alcohols by altering number of physiological parameters influence the ECG. Changes in membrane permeability, electrolyte imbalance, autonomic dysfunction and direct myocardial toxicity may all play a role in the genesis of these changes.

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