EFFECTS OF ETHANOL AND METHANOL ON SPONTANEOUS ELECTROMYOGRAPHIC SINGALS AND NEUROMUSCULAR LATENCY

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Summary : The effect of alchols (ethanol and methanol) on rat electromyogram (E.M.G) and neuromuscular latency were studied in thiopentone anesthetized albino rats. Both alcohols were given intraperitoneally (100 *mg*/100 *g* of body weight) to the respective groups and the controls received saline. Electromyographic signals were recorded from gastrocnemius muscle. For latency studies both the alcohols were given intravenously (iv) at a dose of 20 *mg*/100 *g* of body weight, and response to *in situ* sciatic nerve stimulation was studied before and after alcohol administration.

Our results show that both ethanol and methanol induce spontaneous electromyographic signals and in addition produce changes in the latent period and the amplitude of the response.

Key words : rat electromyogram, spontaneous signals, latency, ethanol, methanol

INTRODUCTION

The primary objective of this study is to elucidate the action of ethanol and methanol on the spontaneous electrical discharges in the muscle of the rat, since these alcohols are known to produce changes in the fluidity of the cell membrane (2, 5, 12). Chin and Goldstein (2) have shown that low concentration of ethanol increases the fluidity of cellular and subcellular membranes *in vitro*. They suggested a similar action *in vivo* also. Later work of the same authors have shown that in chronic alcohol administration, an adaptive change develops and the membranes become more resistant to the fluidizing effect of alcohol (3).

These effects are probably due to the action of alcohol on the lipid layer of the cell membrane. The expansion and the separation of the lipid molecules by alcohol is believed to increase the fluidity of the cell membranes (11).

It has been shown by Peter *et al.* (9) that alcohol increases the amplitude and duration of the miniature end plate potential (m.e.e.p.) of toad neuromuscular junction which is due to the prolongation of the decay phase of m.e.e.p.

Sachedev *et al.* (10) have shown that ethanol increases twitch tension in indirectly stimulated muscles, which may be related to enhanced release of acetylcholine. Ethanol suppressed isometric twitch tension without affecting the tetanus amplitude in frog muscle. This suppression was attributed to the inhibition of calcium release from the endoplasmic reticulum (6).

Ballantyne *et al* (1) demonstrated predominant axonal dysfunction in chronic alcoholics. This and other similar studies have shown the involvement of distal peripheral nerves in chronic alcoholics.

Since our perusal of literature has not shown the specific action of ethanol on E.M.G. and neuromuscular latency and further similar work on methanol action is virtually not available in literature, this preliminary study was undertaken to elucidate the acute effect of ethanol and methanol on E.M.G. and neuromuscular latency in rats.

MATERIAL AND METHODS

Fifty rats of either sex anaesthetised with thiopentone sodium (40 *mg/kg*) were used in this study. The animals were divided into two major groups. One group was used for monitoring spontaneous E.M.G. signals with alcohols and the other group was used for latency studies. The sub groups are shown below.

Group - A (n=30) :

A—1 : (n=10) - Controls : Spontaneous E.M.G. signals were recorded from gastrocnemius muscle by concentric needle electrodes using MS-4 E.M.G. machine for first ten minutes. One *ml* of saline was injected intraperitoneally and its effects on E.M.G. noted for the next ten min.

A—2 : (n=10)- E. M.G. signals were recorded as above for ten minutes, one *ml* of ethanol was given ip (100 *mg*/100 *g* of bw). E.M.G. signals were recorded for the next ten min.

A—3 : *(n=10)* - Similar procedure was adopted for this group with methanol (100 *mg*/100 *g* of bw).

For latency studies sciatic nerve was exposed in the thigh and placed on a bipolar stimulating electrode which was connected to the stimulator output of the E.M.G. machine. Supramaximal shocks were used to stimulate the nerve. The latency was measured using "strobe" facility available with MS-4.

Group B (n=20) :

B—1 : (n=10) — The latency measurements were done before and one minute after ethanol injection, which was given intravenously through jugular vein, Twenty milligram (0.2 *ml*) per 100 *g* of body weight was the dose of ethanol used.

B—2 : (n=10) — Similar procedure was adopted to study the effect of methanol, using the same dose as ethanol.

RESULTS

There were no spontaneous E.M.G. signals in the control rats (A-1) except some random spikes which occurred at sporadic intervals. Ethanol treated animals (A-2) showed spontaneous E.M.G. signals which started 158±14.3 seconds after the injection and lasted for 222±42.0 sec. Methanol treated animals also showed similar electrical activity which appeared within a short latent period of 5.6±1.3 seconds and lasted for 160.4±26.26 sec. (Table I).

TABLE I : Effect of alcohols on spontaneous electrical activity.

Alcohol	*Onset time (sec)*	*Net duration (sec)*
Ethanol	158±14.3	222±42.0
Methanol	5.6±1.34	160.4±26.26
P	<0.001	not significant

Values are mean of 10 observations ± S.E.

The total duration of spontaneous electrical activity with ethanol and methanol did not show any statistical difference whereas the time of onset of these spontaneous signals is significantly quicker with methanol.

The latency studies show that ethanol treatment increases the latency from 1.32±0.091 msec to 1.82±0.158 m sec ($P<0.05$). Methanol on the other hand decreases the latent period from 1.48±0.065 m sec to 1.24±0.066 msec ($P<0.05$) (Table II). In addition it was observed in latency studies that these alcohols decreases the amplitude of the E.M.G. signals significantly.

TABLE II : Latency changes after alcohols.

Alcohol	*Initial latency (msec)*	*Latency after alcohol (msec)*
Ethanol	1.32±0.091	1.82±0.158*
Methanol	1.48±0.065	1.24±0.066*

Values are mean of 10 observations ± S.E., *=$P<0.05$

DISCUSSION

The classical concept of ethanol depressing the central nervous system (CNS) is well documented. This depression is selective, occurring in certain areas of the CNS. Alcohol inhibits the inhibitory systems of the CNS resulting in elation and/or aggression. From the available evidences, it can be presumed that all the actions of ethanol is due to its action on the cell membrane, though the exact mechanism is still not clear.

In this series of experiments both ethanol and methanol produced spontaneous electrical activity in the muscle. Most of these potentials were in the form of fibrillations and positive waves. Occasional fasciculations were also noted. This indicates two possibilities : (i) The spontaneous electrical activity may be due to an direct action of these alcohol on the CNS, and (ii) It could also be due to a direct action on the muscle membrane.

Due to the preliminary nature of this investigation we have not attempted to elucidate the mechanism of action or the site of action. However in the case of frogs this spontaneous action potentials persisted even after sciatic nerve sectioning. This indicates that the most important site of action is on the muscle membrane itself, since alcohol is known to alter the fluidity of the phospholipid layer of the cellular and subcellular membranes (2).

Alcohol could have influenced the calcium release from the endoplasmic reticulum of the muscle, thereby altering the micro environment of the muscle, leading to spontaneous electricai activity. The work of Pachingear *et al.* (8) support this concept. They have reported a decreased calcium binding in the smooth endoplasmic reticulum of alcohol treated dogs Alcohol also increases the permeability of cell membrane to sodium, chloride, and potassium (13). This may lead to extracellular hyperkalemia resulting in a reduction of the endplate potential and hence the action potential of the muscle (7). Both ethanol and methanol are known to reduce the current flow through ionic channels in a concentration dependent manner, thereby markedly reducing the size and decay time constant of spontaneous presynaptic excitatory potentials in fish neuromuscular junction (4). Thus the spontaneous potentials recorded in our experiments has a multifaceted origin and some of these mechanisms may be responsible for the muscle tremor seen in chronic alcoholics.

Both ethanol and methanol induce spontaneous E.M.G. signals and this fact indicates that both alcohols have similar "physical" action on the muscle membrane though their action differs in other respects. The longer latency of onset of potential with ethanol indicates that its action on the cell membrane is relatively slow, compared to methanol. Why methanol is able to act quicker is not known at present and the review of literature has also not shown any possible clue. Probably the permeability of the cell membrane is more for methanol than for ethanol.

Alternatively the effect of delayed onset of spontaneous E.M.G, signals and the prolongation of latency in ethanol treated animals indicate the presence of reduced sensitivity of the receptor/effector mechanisms. Increased sensitivity of these end organs to methanol may also play a role in this respect.

In addition, the changes in the fluidity of the cell membrane in these alcohol treated animals may contribute to the alterations in the extracellular ionic balance which may also influence the receptor sensitivity. Further the observation that both these alcohols reduce the amplitude of the action potentials on nerve stimulation lend support to this concept of involvement of ionic imbalances in this process.

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