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Effects of Ethyl Alcohol on Central Neurons[1]

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WAYNER, M. J., T. ONO AND D. NOLLEY. *Effects of ethyl alcohol on central neurons.* PHARMAC. BIOCHEM. BEHAV. 2(1) 499–506, 1975. – A method was developed for the electrophoretic application of ethyl alcohol through one capillary in a multibarrel microelectrode array in the vicinity of the recording electrode. Effects of ethyl alcohol and angiotensin II applied by means of electrophoretic ejection and ethanol administered intravenously on the frequency of extracellularly recorded action potentials of brain cells were determined. A total of 87 neurons in four different parts of the brain in female hooded rats anesthetized with a mixture of urethan and chloralose were tested. Results reveal that a most sensitive neurons appear to be those of the lateral hypothalamus within the medial forebrain bundle. Cells of the zona incerta and thalamus were also sensitive to ethanol. Cells of the cerebral cortex appear to be relatively less sensitive. Many of the ethanol sensitive cells also responded to angiotensin II and when tested the effects were potentiated by Na.

Ethyl alcohol Angiotensin II Drinking Alcoholism Electrophoresis
Hypothalamus Lateral hypothalamus Zona incerta Thalamus Cerebral cortex

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THE EFFECTS of ethyl alcohol on excitable nerve membrane have been studied in detail and the results have been summarized recently [5,10]. In essence, data on squid giant axon indicate that membrane excitability is depressed by ethyl alcohol through suppression of the increase in Na conductance which normally accompanies adequate stimulation [1, 6, 7]. Ethyl alcohol is more effective when applied externally and can reduce the efflux of Na^{22} by about 30% without any noticeable effect on the amplitude of the action potential [5]. Evidence has also accumulated which indicates that ethyl alcohol usually inhibits brain (Na+K)ATPase but that active ion transport and (Na+K)ATPase activity increases after chronic administration of ethanol [5]. Apparently, ethyl alcohol has a profound effect on excitable nerve membrane which involves molecular processes that precede the action potential and on the subsequent active transport of Na and K. Therefore, as ethyl alcohol permeates the blood brain barrier readily it can be expected to produce various effects within the central nervous system. In a study of ethyl alcohol on cat spinal reflexes [3], dorsal horn interneuron spontaneous discharges were depressed whereas spinal moto neurons were more resistant and larger doses produced a decrease of membrane conductance to both inward and outward constant current. In the cerebellum, ethyl alcohol tended to accelerate the discharge of interneurons and depress what appear to be Purkinje cells. Single cell activity recorded from the lateral vestibular nuclei was also depressed [4]. In the lateral hypothalamus (LH), 85% of the neurons examined were sensitive to sodium and ethyl alcohol; 53% increased in discharge rate and 32% decreased, and the remainder were not affected [8]. Additional evidence indicated that ethyl alcohol might selectively depress activity in neurons with high spontaneous discharge rates. These cells might be interneurons which normally inhibit, as part of a functional aggregate of cells, other neurons and produce in them relatively low discharge frequencies. Therefore, ethyl alcohol in the brain might have a selective effect on interneurons just as it does in the spinal cord, except that these interneurons in the LH are very sensitive to Na which makes them particularly vulnerable to ethanol because of its effect on sodium conductance and the excitability of the cell membrane. Consequently, the discharge rate of these Na sensitive interneurons is reduced by ethanol and other cells in the aggregate are released from inhibition and increase in frequency. As these data were obtained by means of intravenous administration of ethyl alcohol and are confounded by complex long latencies and prolonged changes which might be attributed to secondary effects or the production of some active metabolite, a more direct approach utilizing some other method such as microelectrophoresis seemed to be required. The purpose of the present investigation was to develop such a method for ethyl alcohol and to utilize it to determine neuronal sensitivity directly in terms of the

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modulation of spontaneous unit activity recorded in several different parts of the brain. Results reveal that the most sensitive neurons appear to be those of the lateral hypothalamus within the medial forebrain bundle (MFB). Cells of the zona incerta and thalamus are relatively less sensitive to ethyl alcohol. Many of the ethanol sensitive cells also responded to angiotensin II and when tested the effects were potentiated by the electrophoretic ejection of Na.

METHOD

Animals

Experiments were performed on female hooded rats, 250–290 g in weight, selected from our colony.

Procedure

Animals were anesthetized with a mixture of chloralose, 50 mg/kg, and urethan, 0.5 g/kg, administered intraperitoneally. Subsequent maintenance doses of chloralose, 10–20 mg/kg, and urethan, 0.1–0.2 g/kg, were injected when necessary. In general the remainder of the procedures were as described previously [10]. Briefly, the animal was fixed rigidly to a stereotaxic instrument, the skull was exposed, and removed over predetermined subcortical structures for the insertion of the electrodes. The exposed cortex was covered with a 1% solution of warm agar made with isotonic ringers solution. The lateral tail vein was a cannulated and was continuously infused with lactated ringers solution, pH of 6.0–7.0 and warmed to body temperature, at a rate of 0.00175 ml/min. Test solutions of NaCl and ethanol were infused at the rate of 0.54 ml/min.

Five barrel micropipette arrays were employed in this study. Extracellular single unit discharges were recorded through one barrel filled with 4 M/1 NaCl, resistance varied from 5 to 25MΩ. Action potentials were amplified, monitored visually, stored on magnetic tape, and analyzed by conventional means. The other four barrels were filled with angiotensin II, CIBA 83% valine^5-angiotensin II and 17% ammonium acetate, 1.0–5.0 mM/1 in distilled water with a final pH of 5.0; 10% (0.9% NaCl in distilled water) plus 90% (95% ethanol) with a pH of 6.5; monosodium-l-glutamate, 2 M/l, pH of 8.0 (NaOH); and 0.9% NaCl m distilled water. Substances were ejected in the vicinity of the tip of a constant current source [9]. The resistances of these four barrels varied from 5–100 MΩ. The overall tip diameter of the five barrel array varied from 3–6 MΩ. Possible direct electrical effects of the microelectrophoretic currents were evaluated on the basis of previously published criteria [2] and unreliable data were discarded. Tests performed with NaCl and ammonium acetate alone in distilled water with similar ejection currents were negative and indicate that the effects reported here can be attributed to the angiotensin and ethyl alcohol. Both anodal and cathodal currents were employed in these control tests. Ethanol appears to be ejected more easily when the tip is negative and relatively little Na would be released. The ejection of Cl ions, even by relatively large currents, usually had no effect or produced a slight increase of spontaneous unit activity under these conditions. The final electrode tip position within the brain for each experiment was determine by perfusing the animal with the electrode shaft in place. After fixation the electrode was removed and the brain was sectioned at 40μ, stained with cresyl violet, and examined by means of a dissecting microscope.

RESULTS

The data on 87 neurons in four brain regions are summarized in Table 1. The number of cells, N, studied in each site is also included. Results clearly indicate an increase in discharge frequency in many cells during the electrophoretic ejection of ethyl alcohol. Only one cell decreased in frequency.

LH-MFB

Within the medial forebrain bundle of the lateral hypothalamus, LH–MFB, 12 cells were tested and 5 increased, none decreased, and 7 were not affected. Five of the 7 which were not affected were tested with angiotensin and 2 increased, none decreased, and 3 were not affected. A dose related increase in an LH–MFB neuron due to ethyl

TABLE 1

A SUMMARY OF THE EFFECTS OF ETHANOL AND ANGIOTENSIN II ON THE NEURONS OF FOUR DIFFERENT PARTS OF THE BRAIN

	Ethanol				Angiotensin			
Site	N	I	D	NE	N	I	D	NE
LH-MFB	12	5	0	7	5/7	2	0	3
Zona Incerta	17	7	1	9	5/7	5	0	0
					9/9	2	1	6
Thalamus	45	13	0	32	11/13	8	0	3
					24/32	1	1	23
Cortex	13	4	0	9	2/4	0	0	2
					8/9	0	0	8

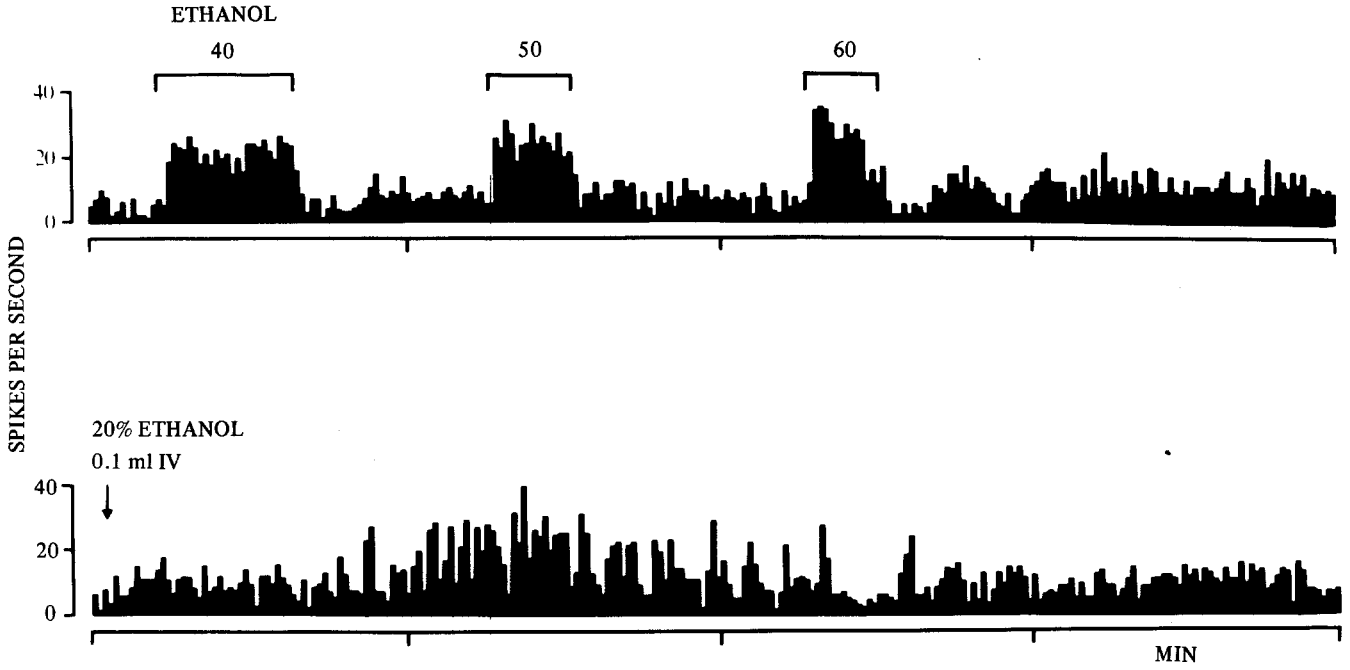

FIG. 1. An X-Y plot of the discharge frequency of an LH-MFB neuron before, during, and following the ejection of ethyl alcohol by currents of 40, 50, and 60 nA and the intravenous administration of 0.1 ml of 20% ethanol. Solid horizontal lines indicate the duration of the ejection current and the arrow indicates when the intravenous injection began.

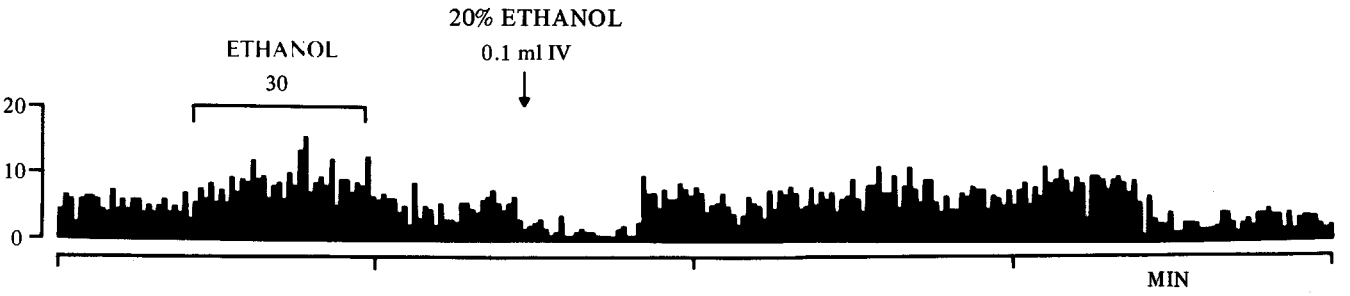

FIG. 2. Same as Fig. 1 except for a different LH–MFB neuron.

alcohol is illustrated by an X–Y plot of the frequency in Fig. 1. In the top part of Fig. 1 the frequency before, during, and following the administration of ethanol by ejection currents of 40, 50 and 60 nA are plotted as a function of time. In the lower part of Fig. 1 the discharge frequency of the same cell before, during, and following the intravenous administration of 0.1 ml of 20% ethanol is also presented. This cell clearly displays an increase in discharge frequency to both intravenous and electrophoretic ejection of ethanol. This cell was not noticeably affected by equal or larger ejection currents of Cl. Similar effects are illustrated by another LH–MFB neuron in Fig. 2. The effects of electrophoretic ejection of ethanol are quite similar; whereas, the effects of intravenous administration of ethyl alcohol appear to be more complex. The complex change in discharge frequency of hypothalamic neurons due to the intravenous administration of ethanol has been observed previously [8,10]. A small increase in frequency can usually be observed within 15 sec which then persists with a second more pronounced increase in frequency occurring with a latency of 50–250 sec. Such an effect is illustrated in the lower part of Fig. 1. The initial change in frequency in the second LH–MFB cell in Fig. 2 appears to be a decrease for about 20 sec which is then followed by a pronounced increase for approximately 90 sec. This initial brief inhibitory effect is difficult to interpret at present. The effects of intravenous administration of various active substances are always difficult to interpret because of long latencies, fluctuations in baseline, and uncontrolled secondary changes.

Two types of neurons were studied in the LH; those within the medial forebrain bundle, LH–MFB, which increases in discharge rate in response to angiotensin II and are inhibited by basolateral amygdala stimulation; and those more ventral in the lateral hypothalamus, LH, which decrease in frequency in response to angiotensin II and are excited by basolateral amygdala stimulation [11]. The effects of ethanol on an LH neuron are illustrated by the X–Y plots in Fig. 3. In the top part of the figure, dose related increases to ethanol by ejection currents of

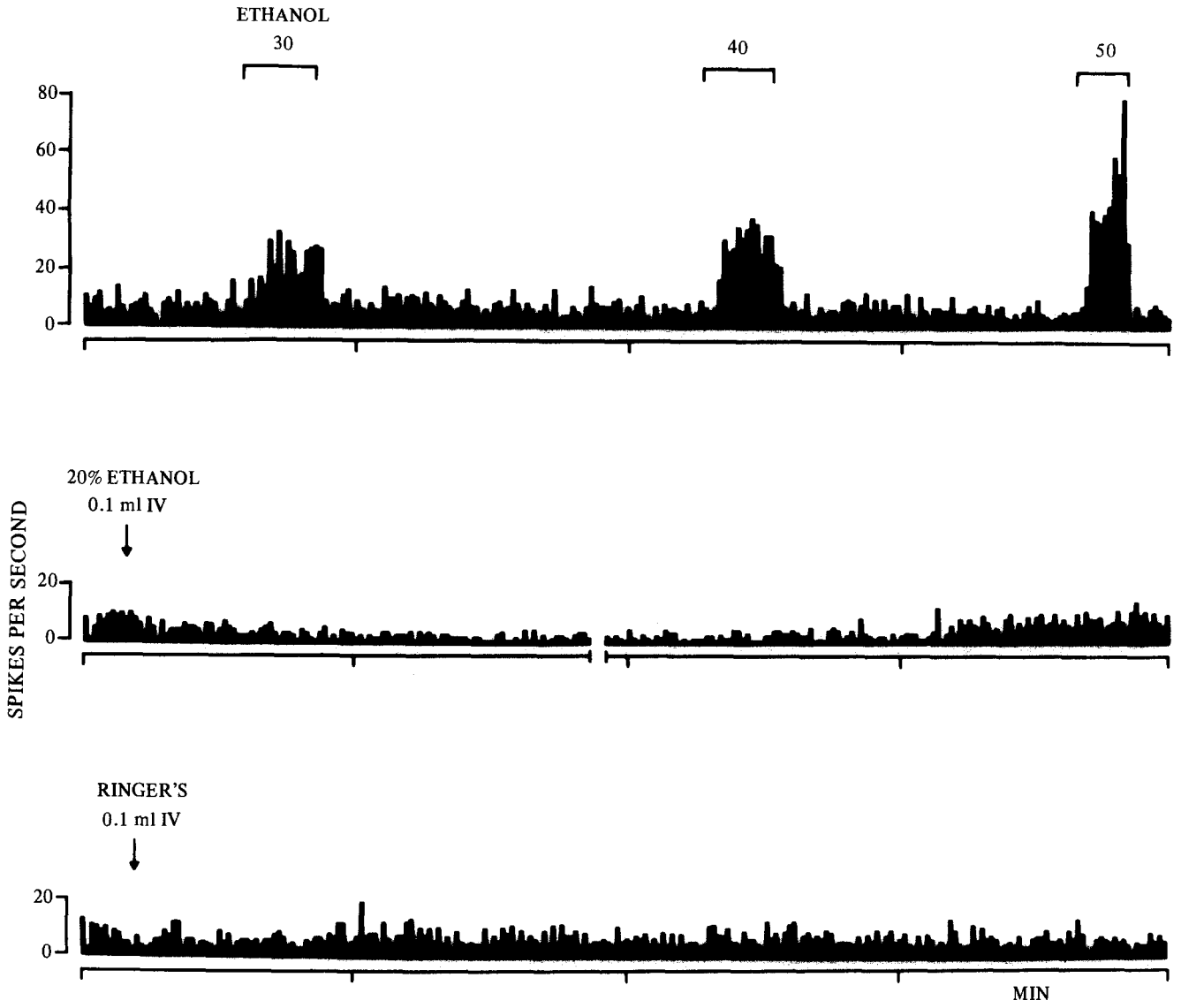

FIG. 3. An X-Y plot of the frequency of a lower more ventral LH neuron. Top: A dose related increase in frequency with ejection currents of 30, 40, and 50 nA. Middle: Same cell as in the top part of the figure. A prolonged decrease in frequency due to a 0.1 ml intravenous injection of 20% ethanol. Fifteen sec of the recording omitted in the middle of the X-Y plot. Bottom: No significant change in the discharge frequency of the same neuron following the intravenous injection of 0.1 ml of ringers solution.

30, 40, and 50 nA are presented. The effects are obvious. The same cell's response to intravenous administration of 0.1 ml of 20% ethanol is presented in the middle part of Fig. 3, approximately 15 sec of the recording were excluded from the middle of the X–Y plot. These data are more difficult to interpret because of the relatively long and obvious decrease in frequency which seems to follow the intravenous administration of ethanol. The same cell was not affected significantly by the intravenous application of 0.1 ml of ringers solution. The latency was about 45 sec and only a slight effect is just noticeable in the bottom part of Fig. 3.

Zona Incerta

Of the 17 cells within the zona incerta, ZI, which were tested with ethanol ejected electrophoretically, 7 increased, one decreased and 9 were not affected. Five of the 7 which increased were tested with angiotensin II and all 5 increased. Nine of the 9 cells which were not affected by ethanol were tested with angiotensin II and 2 increased, one decreased, and 6 were not affected. An example of Na sensitive ZI neuron dose related increase to ethanol ejected electrophoretically is illustrated by the X–Y plot in the top part of Fig. 4. These tests were followed by intravenous injections of 0.1 ml of ringers solution and 15% NaCl as presented in the remaining portion of the top X–Y plot and the lower portion of Fig. 4. The ringers solution produced a perceptible small increase in frequency. The increase in frequency due to the 15% NaCl was more pronounced and significant.

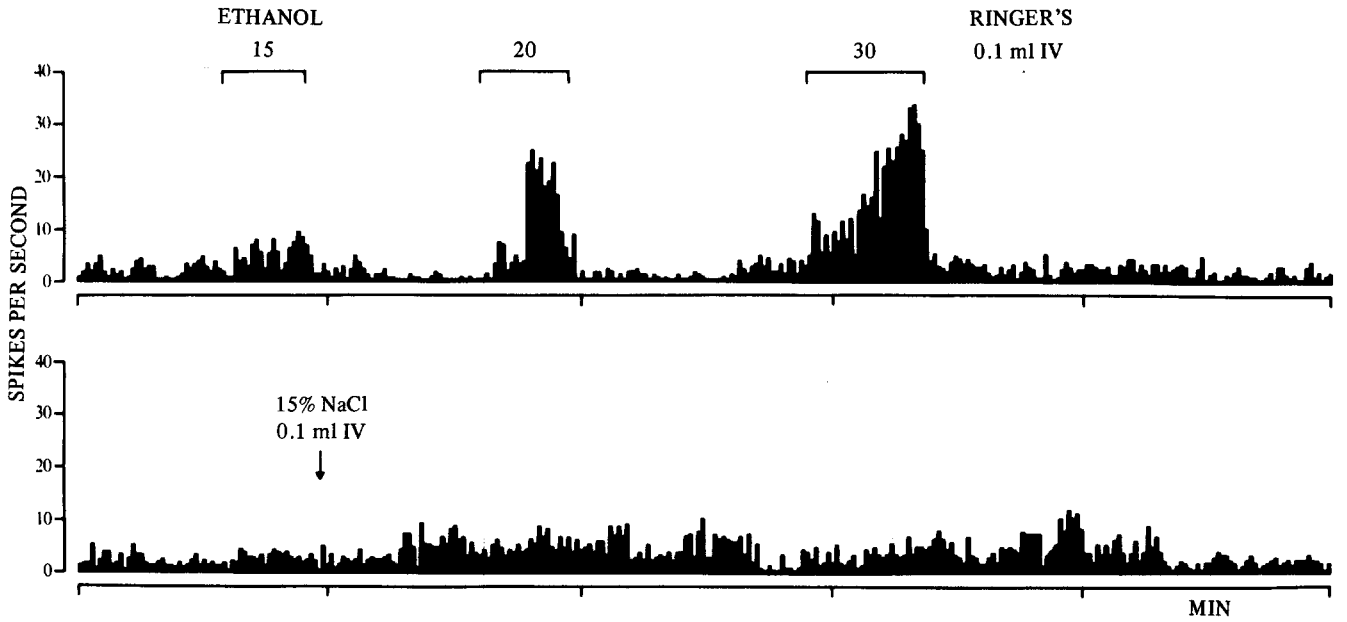

FIG. 4. An X-Y plot of the frequency in a zone incerta neuron illustrating a dose related increase to ejection currents of 15, 20, and 30 nA, a small increase to the intravenous injection of 0.1 ml of ringers solution (top), and a pronounced increase to 0.1 ml of 15% NaCl administered intravenously (bottom).

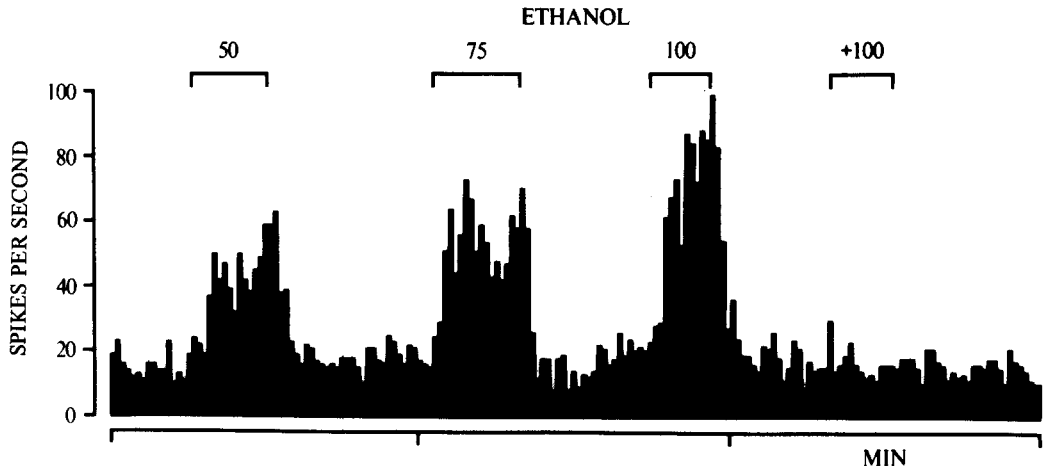

FIG. 5. An X-Y plot of the frequency of a typical thalamic neuron illustrating a dose related increase during ejection currents of 50, 75, and 100 nA. The immediate and rapid increase in frequency is obvious. Reversing the ejection current and making the tip positive at 100 nA had no effect.

Thalamus

A relatively large number of thalamic cells were tested because all of the neurons studied were located along essentially the same electrode tract which resulted from an attempt to place each electrode tip in the same predetermined site. Of the 45 neurons tested with ethanol, 13 increased in frequency, none decreased, and 32 were not affected. Eleven of the 13 which increased were tested with angiotensin II and 8 increased, none decreased, and 3 were not affected. Twenty-four cells of the 32 which were not affected by ethanol were tested with angiotensin II; one increased, none decreased, and 23 were not affected. The dose related effects of ethanol on a thalamic cell are demonstrated clearly by the X–Y plot in Fig. 5. The brief latency and rapid increase in frequency during the ejection of ethyl alcohol is obvious at each dose. Reversing the direction of the 100 nA ejection current to make the tip positive had no effect.

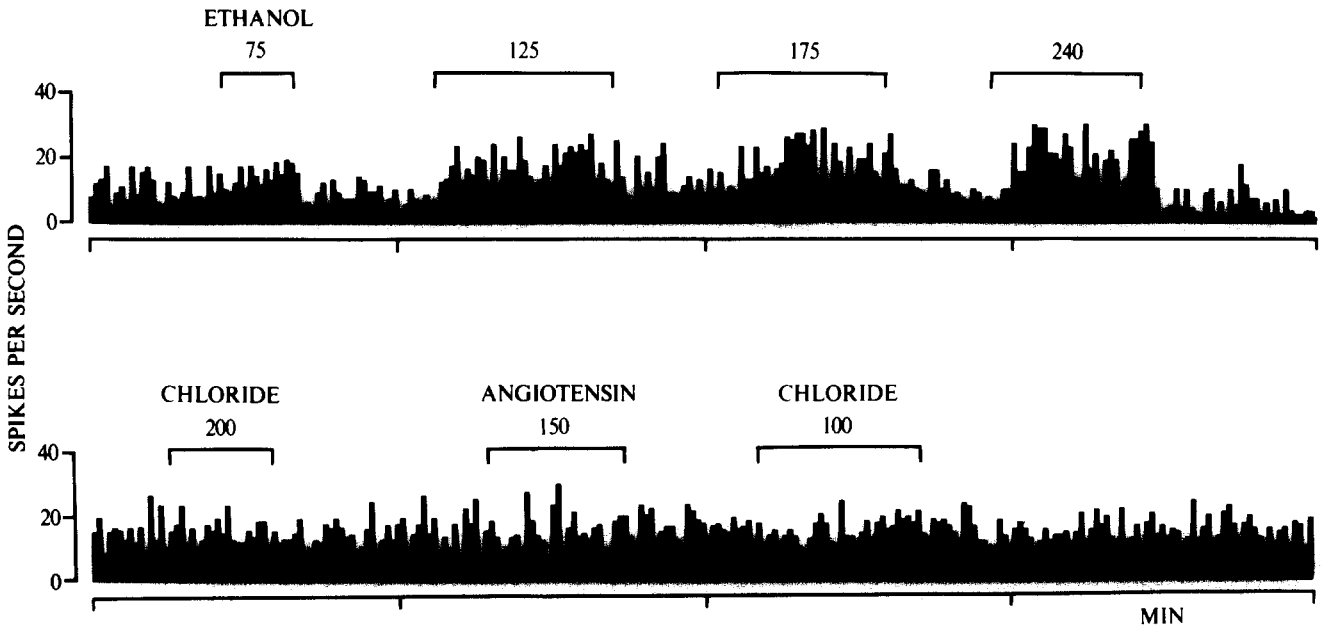

FIG. 6. An X-Y plot of the frequency in a cerebral cortex neuron illustrating a dose related increase beginning at the threshold ejection current of 75 nA. Ejection currents of 125, 175, and 240 nA produced pronounced increases; whereas, ejection currents of reversed polarity of 100 and 200 nA which released chloride ions had no effect. A current of 150 nA through a capillary filled with angiotensin II also had no noticeable effect on this cell.

Cerebral Cortex

Of the 13 cells in the cerebral cortex tested with ethanol, 4 increased in discharge frequency, none decreased, and 9 were not affected. Two of the 4 which increased were tested with angiotensin II and both were not affected. Eight of the 9 which were not affected by ethanol were tested with angiotensin II and none were affected. Reliable threshold data were obtained on only one cortical cell, which was 75 nA, and is illustrated by the changes in the X–Y plot of Fig. 6. Because of the high threshold of cortical cells, they were usually tested with ejection currents greater than 100 nA. The effects in thalamic and cortical cells were not as pronounced as they were in the LH–MFB, ZI, and thalamus. In Fig. 6 clear and obvious increases in response to electrophoretic ejection of ethanol occurred at 125, 175, and 240 nA but there were no significant changes during the application of positive currents of 100, 150, and 200 nA when chloride ions and angiotensin were ejected.

Comparison of Effects in Different Parts of the Brain

Results on spontaneous discharge rates are summarized in Table 2. The number of cells are different from those reported in Table 1 because the several combinations of tests performed varied and depended upon the length of time a particular cell could be studied. The LH–MFB neurons have the lowest spontaneous discharge frequencies and seem to be less variable than cells of the zona incerta. Comparisons of the mean time course of the increase in discharge frequencies for each of the four parts of the brain examined are summarized in Table 3. Only data from neurons which displayed unequivocal increases were utilized in the analysis. Thalamic neurons responded

TABLE 2

A COMPARISON OF SPONTANEOUS DISCHARGE RATES IN SPIKES PER SEC BETWEEN FOUR PARTS OF THE BRAIN

Site	N	Mean Frequency	Standard Error	Range
Cortex	13	6.4	0.8	2–12
Thalamus	49	8.2	1.0	2–30
Zona Incerta	18	8.5	2.0	2–30
LH-MFB	12	5.2	1.0	1–13

TABLE 3

A COMPARISON OF THE TIME COURSE OF THE ETHANOL EFFECT IN SEC BETWEEN FOUR PARTS OF THE BRAIN DURING ELECTROPHORETIC EJECTION. MEANS AND STANDARD ERRORS.

Site	N	Latency	Time to Peak	After Effect
Cortex	4	3.5 ± 2.2	10.8 ± 3.9	1.5 ± 0.5
Thalamus	13	1.9 ± 0.3	7.4 ± 1.4	2.2 ± 0.8
Zona Incerta	8	6.1 ± 1.5	11.9 ± 2.2	5.0 ± 1.8
LH-MFB	5	4.6 ± 2.7	10.1 ± 2.7	3.2 ± 0.5

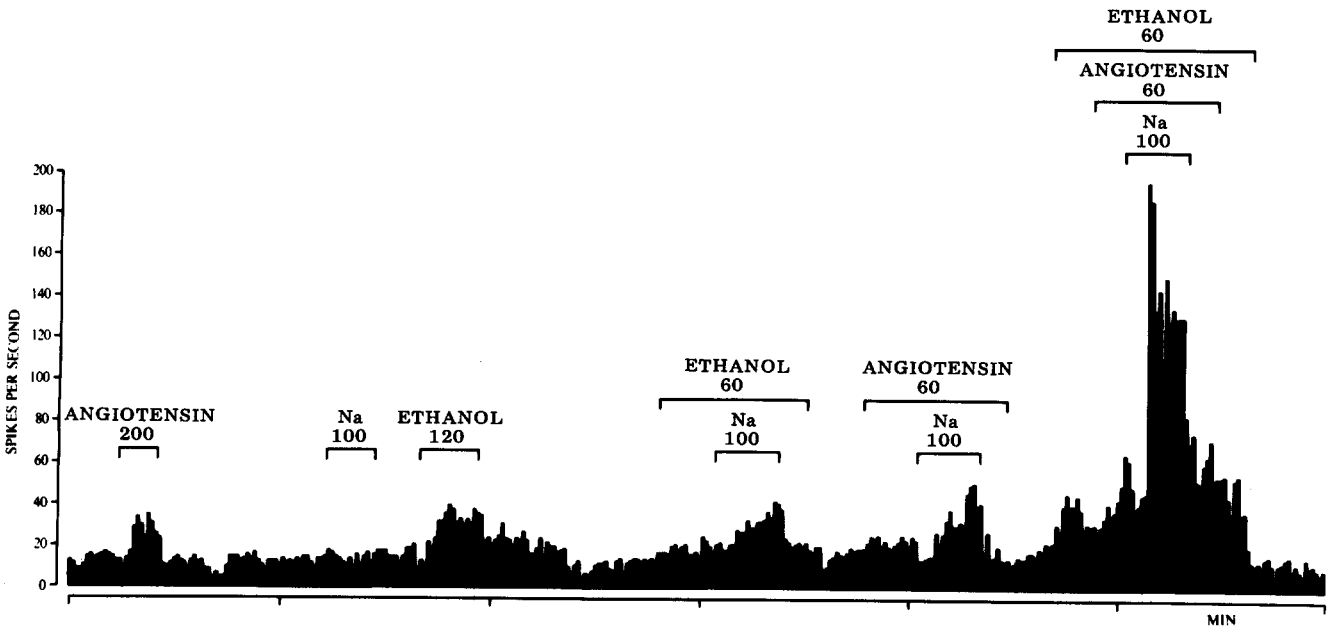

FIG. 7. An X-Y plot of the frequency in an LH-MFB neuron. Reading from left to right: the first three ejections illustrate responses to angiotensin (200 nA), Na (100 nA), and ethanol (120 nA): the fourth shows the enhancement of a small increase due to ethanol (60 nA) by the simultaneous ejection of Na (100 nA) which by itself had no noticeable effect; the fifth shows the enhancement of a small increase due to angiotensin II (60 nA) by the simultaneous ejection of Na (100 nA) which by itself had no noticeable effect; and the sixth which demonstrates the tremendous potentiation in frequency due to the combined application of ethanol, angiotensin, and Na.

most readily. Latency was considerably shorter in the thalamus as compared to other parts of the brain. Time to peak discharge was the time which elapsed from when the ejection current artifact appeared in the record to when the discharge frequency reached its maximum value. After effect was calculated as the time which elapsed from when the artifact appeared due to termination of the ejection current and when the discharge frequency returned to its baseline value. In general thalamic neurons displayed the shortest latencies, time to peak discharge and a relatively short after effect. A comparison of ethanol thresholds was confounded by the fact that clear threshold data was obtained in only 7 thalamic cells, 6 cells of the zona incerta, and 5 LH–MFB neurons, and that relatively coarse increments of current intensity were utilized. The thresholds in the ZI were variable with a mean of 53.3 nA and a standard error of 19.0 nA. The data were more consistent for the thalamus, means of 51.4 ± 10.6 nA, and LH–MFB, mean of 30.0 ± 3.2 nA. A one-tailed t test of the difference in means between the thalamus and LH was 1.65 with 10 degrees of freedom with a probability of chance occurrence greater than 0.05 and less than 0.10. There is some indication then that-the cells of the LH–MFB constitute a population with a relatively low threshold to ethyl alcohol.

Combined Effects of Ethanol, Angiotensin II, and Na

In several cells the opportunity occurred to determine the modulation of unit activity by combined treatment with three substances. Results of one experiment in an LH–MFB neuron are summarized by the X–Y plot in Fig. 7. Increases in discharge frequency due to ejection of angiotensin II, Na, ethanol alone are illustrated. The Na ejection current of 100 nA did not produce a significant change in frequency. In the fourth test, reading from left to right, ethanol ejected by 60 nA of current produced a just noticeable increase in frequency which was then potentiated by the simultaneous ejection of Na. A similar pattern is displayed by the results of the fifth test where a small increase in discharge frequently produced by angiotensin ejected by 60 nA of current is potentiated by the ejection of Na with 100 nA. In the sixth test, a very pronounced potentiation of discharge frequency occurs in response to the simultaneous ejection of ethanol by 60 nA, angiotensin II by 60 nA, and Na by 100 nA. The multiplicative effects of the combined treatment are obvious.

DISCUSSION

Results indicate that ethyl alcohol is released from a glass capillary microelectrode which contains a relatively high concentration of ethanol mixed with a small quantity of NaCl by means of an electrophoretic effect which depends upon the ions in solution. Ethyl alcohol seems to be ejected when the electrode tip is negative and chloride ions are leaving and the sodium ions are being retained. These effects require further study to determine the quantity of ethyl alcohol ejected under these conditions.

Although thalamic neurons appear to be more responsive to ethyl alcohol applied by means of electrophoretic ejection currents in terms of latency and time to peak increase in discharge frequency, LH–MFB neurons were similar and were less variable with lower thresholds. These results support earlier data [8] obtained under similar

conditions but with intravenous administration of the ethyl alcohol which indicate that LH–MFB neurons are relatively more sensitive to ethyl alcohol than other cells of the brain. Of the 87 neurons tested in this study, 29 increased in response to the ejection of ethanol and only one decreased. Twelve of the cells tested were in the LH–MFB and 5 increased and the other 7 were not affected. Previous results [8] indicated that at least 32% of the LH–MFB neurons should have decreased, if the effects of electrophoretic ejection of ethyl alcohol are similar to intravenous administration of ethanol. As Na sensitive neurons are difficult to locate because of what appears to be a relatively small population and high threshold, it is difficult to determine if the same population of lateral hypothalamic neurons were being studied. When tested with Na as in Figs. 3, 4, and 5, several of the cells responded with increases in frequency. Obviously a larger sample of LH–MFB neurons are required before meaningful comparisons of this type can be made. However, because of the relatively low spontaneous discharge frequency of lateral hypothalamic neurons under the present experimental conditions, additional data will be difficult to obtain. Although the data on thresholds for ethyl alcohol are somewhat tenuous, they do indicate that the LH–MFB neurons are more homogeneous and appear to be more sensitive to ethyl alcohol than cells in other parts of the central nervous system. The after effects of ethanol ejection, persistence of an increase in discharge frequency after the ejection current was turned off, were variable and indicate that the results can be attributed to some active substance ejected rather than a nonspecific current effect. An interpretation which is also supported by the results obtained with cells tested with both electrophoretically and intravenously administered ethyl alcohol.

Results on the combined treatment of angiotensin, ethyl alcohol, and Na have been reported before [11,12] and the multiplicative increase in frequency suggests a possible synergistic action on common ionic membrane mechanisms. Large increases in discharge frequency which occur during the combined ejection of the three substances, as illustrated in Fig. 7, are difficult to attribute to some peculiar current effect because even the absolute amount of current flow is not much greater than that in the first test with angiotensin at 200 nA. Angiotensin II, ethyl alcohol, and variations in extracellular Na concentration might all influence drinking because of a low threshold effect on hypothalamic neurons.

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