Alcohol, affect, and physiology: Positive effects in the early stages of drinking.

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Running head: Alcohol, affect, and physiology

The author would like to acknowledge Linda Grossman, Patricia Meek, David Newlin, Joseph Newman, and Kenneth Sher, all of whom made significant contributions to this research program while they were graduate students at Indiana University. Of particular note are the contributions of Kenneth Sher to the work with high risk populations, and of Patricia Meek to the EEG work.

This research has been supported by NIAAA grant AA05004.

Over the past five years, our laboratory has been engaged in a program of psychophysiological research that has sought the answer to two questions: (a) What are the effects that alcohol has on nonalcoholic individuals? and (b) Do differences among these individuals in the magnitude and nature of alcohol's effects play a significant role in the etiology of alcoholism? In this chapter, I will be primarily focusing on the first question, which could be simply restated as asking: What does alcohol do? In presenting this research, I intend to go beyond just describing our findings, and to offer a careful consideration of which short-term effects of alcohol consumption can be construed as being positive, beneficial, and thereby reinforcing.

This seems to be an opportune time for taking such stock, for we have now run close to 400 subjects in four different experiments that have studied the effects of alcohol on a broad range of biological and behavioral variables. The major portion of this work has examined the effects of alcohol on the functioning of the autonomic nervous system (ANS), both in terms of how alcohol affects resting autonomic levels, and how it affects the responsivity of the ANS to stressful stimuli. Also in the physiological domain, we have studied the central nervous system effects of alcohol by evaluating how it alters brain reactivity. We have explored the effects alcohol has on processes of self-disclosure by having sober and intoxicated subjects compose and deliver brief speeches about their strengths and weaknesses. Using video recordings obtained in our most recent study, we have begun to examine in a new way the old question of

how alcohol affects emotional responding. Instead of relying on self-report data exclusively, we have utilized fine-grained measurement of facial expressive behavior to afford a better understanding of what alcohol does to subjects' internal emotional states. Taken in sum, the breadth of this data set should prove adequate to support a thorough examination of the reinforcing consequences of drinking.

Lest it seem overly strange to be talking about the positive and reinforcing consequences of drinking in a climate of opinion that clearly views alcohol consumption as highly pernicious, some mention should be made of the conceptual thrust of this work. Early in our thinking about this research area, we grappled with the issue of whether to focus on the negative or on the positive outcomes associated with alcohol use. We considered studying such negative outcomes as the disruptive effects of alcohol on cognitive performance, motor performance, and the production of socially adaptive behavior. Similarly, we considered focusing on its deleterious long term effects on physical health, mental health, job performance, economic welfare, and family life. These negative outcomes are of great consequence, but it seemed that studying them was not the optimal strategy for achieving an understanding of <u>why</u> people start drinking and of <u>why</u> they keep drinking. Surely people are not initially attracted to alcohol because they want to destroy their health, lose their jobs, and ruin their social and family lives. Rather, it is almost certain that these dire outcomes recede into a very distant corner of awareness during the early stages of drinking. Of course one

could posit the existence of some intrinsic self-destructive psychopathology that brings people to alcohol abuse, but we decided instead to look for outcomes in the early stages of drinking that could be viewed as being positive, beneficial, and reinforcing. From this viewpoint, people start drinking because they get something valuable, functional, helpful, and even pleasurable from it. Following this reasoning, it seemed that the place to start our study of alcohol and alcoholism was early in the process, when drinking was still pleasant, helpful, and rewarding, and long before the negative outcomes begin to emerge.

Psychophysiologists make lousy hedonists. In thinking about "pleasure", our thoughts usually drift down toward the viscera. In our laboratory we initially operationalized pleasure and positive outcome in two ways. First in terms of state, we thought that alcohol consumption might transport the drinker into a more enjoyable and pleasurable physiological state. Second in terms of reactivity, we thought that alcohol might serve to buffer the individual from the physiological chaos that results from the hostile insults served up in the typical laboratory experiment. As the work progressed, this focus broadened to include psychological and behavioral effects as well, and these too were considered in terms of the state and reactivity hypotheses. We will examine each of these hypotheses in turn, but before proceeding, one additional prefatory comment is in order. I will be adopting the strategy of trying to make the strongest statement possible about the positive and reinforcing consequences of alcohol, in hopes of articulating a point of view that has not been well-represented in the literature. I will

undoubtedly be overstating the case; clearly, alcohol use can be both highly reinforcing and extremely harmful.

The alcohol-induced state

The brain

We know from forty years of biological research that alcohol has a depressant effect on the central nervous system. A very reliable finding has been that alcohol slows the dominant alpha frequency in the resting brain (e.g., Davis, Gibbs, Davis, Jetter, & Trowbridge, 1941; Doctor, Naitoh, & Smith, 1966; Engel & Rosenbaum, 1945; Holmberg & Martens, 1955; Kotani, 1965; Newman, 1959; Varga & Nagy, 1960). Some researchers have interpreted this biological effect in terms of a weakening of the inhibitory functions of the neocortex, and by metaphor, to a loosening of behavioral inhibition. To continue with this analysis, being less inhibited and thereby more spontaneous could be considered to be positive outcomes, and thus these first characteristics of the physiological state produced by alcohol are potentially reinforcing ones.

Behavior

From a clinical point of view, behavioral inhibition requires that the person maintain a continuing state of vigilance and self-awareness. Behavior cannot be selectively inhibited unless the person is continually aware of how he or she is behaving or is about to behave. We have obtained experimental evidence that alcohol consumption reduces self-awareness. In four different studies, we have asked subjects to make a three minute speech on the topic of what they like and dislike about

their bodies and personal appearance. Applying a coding system developed by Exner (1973) to these speeches, we have consistently found (e.g., Hull, Levenson, Young, & Sher, 1983) that alcohol reduces the proportion of speech statements that are coded "self-focused"--that is, those statements that concern only the speaker. Further, alcohol increases the proportion of statements that are coded "external-focused"--that is, those that concern someone other than the speaker.

This finding is well-illustrated from data obtained in our most recent study. In this study (N=192), which I will refer to as the "dose-response study", a double-blind balanced placebo design (Rohsenow & Marlatt, 1981) was used to enable separation of effects attributable to alcohol's pharmacologic action from those attributable to expectancies about this action. Subjects were administered one of three doses of vodka in grapefruit juice--0 g ethanol/kg body weight, .5 g/kg, or 1 g/kg. Appropriate procedures were adopted to manipulate subjects expectations as to whether they were consuming alcohol or not (modeled after those used in Levenson, Sher, Grossman, Newman & Newlin, 1980).

In this dose-response study, alcohol had significant effects both in reducing self-focused statements, $F(2,179)=6.18$, $p=.003$, and in increasing external-focused statements, $F(2,179)=7.40$, $p=.001$. Examination of Figure 1 will reveal that both effects were incremental at increasing doses. These results indicate that alcohol enabled subjects to divert the focus of critical attention away from themselves and redirect it toward other people; thus alcohol reduced their compliance with the

experimenter's explicit request to be self-critical. It is uncertain whether this result reflected an empowering effect of alcohol (which would enable the normally compliant laboratory subjects to reject the unpleasant request) or a lowering of the level of cognitive functioning (which would cause subjects to drift "off-task"). In either case, the end result is easily construed as positive and reinforcing for the intoxicated subject, who managed at least a partial escape from the onerous self-disclosure.

Insert Figure 1 about here

Mood.

The next piece of this puzzle is the effect of alcohol on mood. The logic of the argument here is straightforward. If the state produced by alcohol is to be construed as being pleasurable and positive, then subjects should report feelings that are congruent with such a pleasurable state. In two studies (Levenson et. al, 1980; Sher & Levenson, 1982) using different mood questionnaires we found this to be true. Compared to subjects consuming tonic only, subjects consuming the 1 g/kg dose reported feeling more "cheerful" in the first study, $F(1,88)=12.48$, $p<.001$, and reported feeling more "pleasure" in the second, $F(1,79)=12.67$, $p<.001$.

There is additional evidence from yet another source. In all of our studies we have used a device that we call the "anxiety dial" to obtain a continuous self-report of tension. This device consists of a large war surplus radio knob with a long

translucent plastic cursor that traverses a 180° scale. The scale is anchored by the legends "extremely calm" at 0° and "extremely tense" at 180°. If we look at the average dial position during the period while subjects are waiting for the stressor part of the experiment to begin, we can obtain another indicator of the effects of alcohol on mood. In the two studies just cited, subjects who consumed the 1 g/kg dose reported lower levels of tension on the rating dial than those who consumed tonic only, but in only one of these studies was the difference statistically significant (Study 1: mean rating 2.0 vs 2.8, $t(88)=2.31$, $p<.05$; Study 2: mean rating 3.14 vs 3.70, $t(78)=1.33$. Figure 2 portrays the results from our recent dose-response study; the effect was statistically significant, $F(2,180)=5.10$, $p=.007$, and was incremental at higher doses. Taken together with the self-report data, the phenomenological state produced by alcohol, which can be described as being cheerful, pleasant, and with reduced tension, is certainly positive and undoubtedly reinforcing.

Insert Figure 2 about here

Autonomic nervous system.

The final, and probably most complex, component of the state produced by alcohol is the ANS. Historically, the entire issue of the physiological effects of alcohol has lost clarity when different terminologies have been used interchangeably. For example dimensions such as depressant-stimulant, or arousing-relaxing, or tension reducing-tension increasing have had different meanings when used by different investigators. These

semantic problems have increased when investigators have attempted to aggregate results across multiple response systems. In the case of the ANS, there has been a historical bias toward viewing it as responding in an "all or none" fashion, but this bias is contradicted by five decades of research showing the capacity of the ANS for specificity and differentiation in response when activated by pharmacological agents, cognitive states, and emotional states. With due awareness of this historical background, our research rejected the view of the ANS as a monolith, but rather tried to characterize the effects of alcohol separately for each autonomic response system that we were able to study. I will summarize our findings using the arousing-relaxing dimension and then evaluate the complete set of ANS effects in terms of the whether they might be viewed as being positive and reinforcing.

In terms of arousal effects, we have consistently found that consuming a 1 g/kg dose of alcohol increases heart rate (by about 6 bpm), and increases skin conductance (by about 4 umhos). These two changes both indicate higher levels of arousal. In terms of relaxant effects, we have consistently found that alcohol increases pulse transmission times by about 11 msec (indicating decreases in cardiac contractile force and/or decreases in mean arterial blood pressure). Other investigators, using more direct measures of cardiac contractile force, have found a similar reduction in myocardial performance (e.g., Child, Kovick, Levisman, & Pearce, 1979; Knott & Beard, 1972). We have also found that alcohol produces dilation in the arteries of the

finger. A similar dilation in the arteries of the skin (e.g., Wallgren & Barry, 1970) accounts for the flushing that often accompanies alcohol consumption. Our basis for characterizing vasodilation as a relaxant effect is that the opposite effect, peripheral vasoconstriction, is part of the ANS response to stress.

Thus it would appear that alcohol has an arousal effect on the heart's rate and on electrodermal activity, but has a relaxant effect on the heart's force of contraction and on the vasculature. There are many additional complexities involved. For example, decreases in the heart's force of contraction and increases in heart rate are compensatory changes that function to maintain a stable cardiac output. Thus it is entirely possible that in resting subjects, one of these observed changes is a response to alcohol, while the other is a compensatory response to avoid an inappropriate level of cardiac output.

This complex, but consistent, pattern of autonomic nervous system effects is depicted in Figure 3, which presents the findings from our dose-response study.

Insert Figure 3 about here

Finally, in all four studies we have looked at the effects of alcohol on a global measure of somatic nervous system activity (i.e., gross motor movement). Alcohol has had no effect on this measure in any of the experiments.

It is very difficult to say whether a given set of ANS changes are positive and reinforcing or not. But the thrust of our argument compels us to conclude that the visceral state in

which we find our intoxicated subjects is--taking considerable metaphorical liberties--quite positive. After all, their hearts are not pounding very hard, their arteries are quite relaxed, and the flow of blood to the periphery slightly warms their skin. What could make this state even more idyllic? Perhaps the slight surge of energy that goes along with the modest speeding of the pulse, with the pleasant glistening that comes from having a bit of sweat gland activity thrown in for good measure.

Alcohol and reactivity

The next group of effects we have studied pertain to reactivity--the influence of alcohol on physiological and emotional responses to external stimuli. The question here has been whether alcohol dampens the physiological perturbations caused by stressful environmental events. We have looked at three groups of effects: First, the brain response to tones; second, the ANS response to two different stressors (a moderately painful electric shock and having to deliver the self-disclosing speech); and third, the facial expressive responses to electric shock. I will present each of these in turn, briefly describing the experimental procedures that were utilized.

The brain.

We have studied alcohol's effects on brain reactivity in two experiments. In both we used standard procedures to obtain cortical evoked potentials, which provide a reasonable index of brain reactivity. For each subject the cortical electroencephalogram (EEG) measured from the vertex (C_z) was recorded during 100 one-second trials. Each 1-second trial

consisted of 300 msec of silence and then a 80 db, 400 Hz tone of 700 msec duration. Trials were separated by a random interval. Eyeblink activity was monitored on line by a digital computer and trials on which the subject blinked were discarded and replaced. The computer stored the EEG's and averaged the 100 blink-free trials. The averaging technique results in the emergence of the specific cortical response to the tone from the background of noncontingent EEG activity.

Figure 4 presents the averaged evoked potentials from subjects at three different doses of alcohol (N=64 at each dose). The progressive reduction of the overall amplitude of the evoked potential at higher doses can be seen. In Figure 5, the significant reductions can be seen in the P1-N1 component, $F(2,138)=4.77$, $p=.01$, the N1-P2 component, $F(2,176)=26.88$, $p<.001$, and the P2-N2 component, $F(2,145)=21.67$, $p<.001$. In an earlier study (N=39) that used only the 1 g/kg and 0g/kg doses, we had found the same pattern of results. In both studies, alcohol only diminished the amplitude of the evoked response; the latencies of the various response components were not affected.

Insert Figure 4 about here

Insert Figure 5 about here

We consider diminished cortical reactivity to be a potentially reinforcing consequence of alcohol consumption, especially if the subject is desirous of achieving a state of in which the impact of jarring external stimulation is lessened.

Although our EEG data do not directly bear on this point, we believe alcohol also reduces the impact of unpleasant and unwanted "internal" stimuli such as those associated with anxiety, with reliving unpleasant events that occurred during the day, with intrusive thoughts, and with worries about the future.

Autonomic nervous system.

We have examined the effects of alcohol on ANS responses to stress in three studies. The paradigm used in all three studies was quite similar. Subjects consumed their beverages over a 45 minute period and then sat quietly for 30 minutes to allow for absorption. The stressor portion of the experiment lasted for 23 minutes, consisting of a seven minute baseline, a six minute countdown period in which a timer ticked off the seconds remaining until the stressor was administered, the stressor (the electric shock or giving the three minute self-disclosing speech), and then 10 more minutes of recording.

Across studies, the most consistent finding was that alcohol reduced the cardiovascular responses to these stressors. In Figure 6, the heart rate response of sober subjects and subjects who have consumed a 1 g/kg dose of alcohol are shown. The dampening of the response is shown at two points--at the beginning of the countdown to the stressor, and at the stressor itself. The same dampening effect was found for both the shock stressor and for the self-disclosing speech. In Figure 7, the pulse transmission time response is shown with similar dampening of the two peaks.

Insert Figure 6 about here

Insert Figure 7 about here

The results from our dose-response study provide some further data on the nature of this stress-response dampening effect of alcohol. Figure 8 portrays the results at several points of maximal reactivity, showing that in the case of the heart rate response to the speech, the heart rate response to the shock, and the pulse transmission time response to the shock, the major stress response dampening effect occurred at the 1g/kg dose, with relatively little evidence of the effect at the .5g/kg dose. Prior to this study we had speculated (e.g., Sher & Levenson, 1982) that the higher dose might be necessary before the cardiovascular response was significantly dampened. This dose dependence is supported by a study by Wilson, Abrams & Lipscomb (1980), in which dampening of the heart rate response to a stressful social interaction was found at a 1 g/kg dose but was not found at the lower .5 g/kg dose.

Insert Figure 8 about here

It is not at all certain whether the stress-response dampening effects of alcohol extend to other ANS systems besides the cardiovascular system. Dampening of the cardiovascular response to stimulation at moderately high dosages of alcohol has been reported in several other laboratories (e.g., loud tones: Lehrer & Taylor, 1974). Results with electrodermal response, on the other hand, are less consistent. Skin conductance responses

to simple stimuli such as tones (Carpenter, 1957; Greenberg & Carpenter, 1957) and words (Coopersmith, 1964; Lienert & Traxel, 1959) have been found to be dampened by alcohol, but in our laboratory and others, alcohol has not significantly affected skin conductance responses to more complex stressors. It is likely that the skin conductance responses to simple sensory stimuli have a different psychophysiological meaning than the responses to complex social stimuli. With simple stimuli, we may be seeing the well-documented sweat gland responses that accompany signal detection; these responses may be dampened in much the same way as are cortical responses to sensory stimuli (e.g., our findings with tones). Extending this line of reasoning, the skin conductance responses to complex social stressors could be more indicative of the role of the sweat glands in emotional sweating, and these latter responses may be less susceptible to the stress response dampening effects of alcohol.

Even if the stress response dampening effect of alcohol is limited to the cardiovascular system, this lowering of reactivity in a major ANS system must be viewed as highly positive and reinforcing. It is one of the great ironies of alcohol that despite the devastating effects of alcoholism on the heart and liver, moderate drinking affords several potentially valuable benefits for cardiovascular health. Since heightened beta-adrenergic reactivity (e.g., heart rate increases in response to acute stressors, increases in cardiac contractility) is a factor in several etiological models of hypertension (e.g. Obrist, 1981)

and coronary heart disease (e.g., Gorlin, 1976), alcohol's dampening of heart rate and cardiac contractile responses could be quite beneficial. Possibly related to this are the findings from several epidemiological studies (e.g., Hennekens, Rosner, & Cole, 1978; Kannel & Gordon, 1970) that show lower rates of coronary heart disease in moderate drinkers, as compared to abstainers and to heavy drinkers[1].

Emotional responding.

In our most recent work, we have started to examine the effects of alcohol on emotional responding in a new way. We undertook this work to try to better understand the basis for our consistent findings of diminished cardiovascular responses to stress. These findings have begged the question of how these effects are mediated. Does alcohol act peripherally to directly dampen the reactivity of the heart and blood vessels? Or does it act centrally to raise hypothalamic triggering points for setting off autonomic activity? Or does it act to change subjects' appraisal processes, making them see the threat inherent in our shock and speech stressors as being much lower than they would if sober.

Given the fact that our subjects were consuming different doses of alcohol, using self-report measures of emotional state to attempt to determine mediating mechanisms would be even more suspect than usual. Thus, we decided to utilize fine-grained measurement of facial expressive behavior using Ekman and Friesen's Facial Action Coding System (FACS; 1978). FACS is an anatomically-based coding system that allows decomposition of any

facial expression into the underlying muscular actions by repeated viewing of slow-motion video recordings. Unfortunately, FACS is very time-consuming, taking about one hour to score one minute of facial behavior. For this reason, we decided to focus initially only on the facial responses to the shock stimulus, since these occurred in a brief time period. We have now completed the scoring of 21 female subjects, all with a positive expectancy for alcohol, and distributed equally into our three dosage conditions. With all due caution owing to the small sample size, the results are extremely interesting. But before presenting these findings, an introduction to the facial reactions to electric shock is in order.

Facial reactions to shock. Basen on viewing the facial responses of hundreds of subjects to shock, there seem to be three "windows" of expression that are distinguishable and theoretically important: (a) Anticipation. The first window is the final five seconds of the countdown to the shock. In this window some subjects show an expression in anticipation of the shock. There is great variability in the nature of this expression. Some subjects show the prototypical expression of fear, some show attempts at emotional control, some show contempt. (b) Shock. The second window is the shock itself. Virtually all subjects show some facial reaction to the shock. There is much less variability here, since almost all show some variant of the prototypical expression of fear. Figure 9 shows a full-face prototype of fear. In the 21 subjects scored so far, almost all of the expressions included contraction of the risorius muscle which pulls the lip corners straight back toward

the ears. Contraction of this muscle is part of the prototypical expression of fear and may have an evolutionary function in terms of causing the mouth to assume the proper shape for screaming. (c) Reaction. The third window occurs between three and five seconds after the shock at which time some subjects show a new expression. This is often a "reaction to their reaction" to the shock. Again there is much variability. Some subjects smile; some subjects show contempt.

Insert Figure 9 about here

Effects of alcohol on facial responses to shock. We will look at the effects of alcohol in each of the three windows in turn. In the anticipation window, alcohol sharply reduced the occurrence of several kinds of preparatory facial behaviors. Figure 10 shows the reduction in overall expressiveness, and in two specific categories of facial behavior: (a) reduction in the number of fear expressions and indicators of attempted emotional control (i.e., lip biting, lip pressing, lip tightening); and (b) reduction in the number of "unfelt" or "false" smiles (citing evidence tracing back to Darwin, Ekman & Friesen, 1982, describe these as smiles that include the action of zygomatic major, which pulls the lip corners up, but do not include contraction of obicularis oculi, which raises the cheeks and tightens the lower eyelid). What might this mean? Under the influence of alcohol, subjects may appraise the stressor as being less threatening and thus not react in anticipation of it. They may be less threatened by their imminent display of affect and thus less

likely to try to control it. Or they may be less concerned with trying to put up a brave front and thus less likely to smile falsely for their own benefit or for the benefit of the experimenter.

Insert Figure 10 about here

As previously indicated, in the shock window, most all subjects show a variant of the fear expression, which includes contraction of the risorius muscle. Using the strength of the risorius contraction as a simple indicator of the intensity of the fear expression, alcohol reduces the intensity of the fear response (Figure 11). It is important to note that alcohol does not produce facial responses in the shock window that differ in kind from those produced by sober subjects; they only differ in intensity. This finding can be interpreted as meaning that alcohol lessens, but does not eliminate, the impact of the shock.

Insert Figure 11 about here

In the reaction window, alcohol reduces the occurrence of reactive facial behaviors. Figure 12 shows the reduction in the occurrence of "felt" or genuine smiling (Ekman & Friesen, 1982, describe this as smiling that includes contraction of both zygomatic major and obicularis oculi). What might this mean? At higher doses subjects may have built up less tension and dread in anticipation of the stressor, and thus they would have less need to reduce tension by smiling after the stressor was over. Alternatively, at higher doses subjects could be less amused by

their own reactions to the shock. This may be because they had shown less of a fear response, because they were less aware of their response, or because they were more accepting and less amused by their response.

Insert Figure 12 about here

This microanalytic dissection of emotional responding fits nicely with our other findings that have been presented on the effects of alcohol on reactivity. Looking at the facial data, alcohol lessens the overall impact of a stressful event. The shock or the speech comes and goes, and the intoxicated subject responds with the expected, albeit somewhat diminished, facial expression associated with fear. But that is the extent of the damage. In contrast, the duration of the stressor is extended for sober subjects; it begins with a period of anticipatory arousal, continues during the stressor proper, and then spills over into the period after the stressor has been terminated. Again the effects of alcohol are positive and reinforcing. The world is made less disruptive, more manageable, and less a matter for concern.

Conclusions and implications

We have now reviewed all of the findings we have obtained to date concerning the effects of alcohol on state and reactivity variables drawn from response systems that span the brain, self-disclosing behavior, mood, the ANS, and emotional facial expression. At each juncture we have argued the case that could be made that these effects are positive and reinforcing.

Admittedly, in some instances the argument has been more speculative than we would have liked, but taken together, the bulk of the evidence clearly indicates that nonalcoholic drinkers can obtain a number of positive, beneficial, and reinforcing consequences of moderate drinking. This should come as no surprise given the high incidence of drinking in our society.

Because our work is done in the laboratory and not in the field, we are essentially limited to studying the acute, short-term consequences of alcohol consumption. Thus, many of the negative, chronic, and long-term effects escape our inquiry. Nonetheless, there are several negative acute consequences of alcohol consumption that we could have studied but have not (e.g., negative effects on cognitive and psychomotor performance). In addition, the timing of our experiments is such that we are more likely to detect effects associated with the ascending limb and plateau phases of alcohol absorption. Some of the descending limb effects on mood are undoubtedly far less positive. Still, given our concern with better understanding why people drink, the short-term, immediate, positive consequences are probably of greatest importance. Again risking stating the obvious, it is no great great mystery why people continue to drink once addictive processes of dependence and tolerance begin to act; the challenge for the behavioral sciences is to understand what it is about alcohol consumption in the short-term that makes it worth running the risk of the negative outcomes that are associated with long-term alcohol consumption. To adopt a single metaphor, such as saying that alcohol consumption is "tension-reducing" greatly oversimplifies and unfairly minimizes

the richness of positive outcomes that can be associated with the early stages of drinking.

There are many questions that remain to be answered. At a very basic level we need to better understand the relations between alcohol's effects on the ANS and on emotional expressive response. We know from our ANS data that the cardiovascular responses of intoxicated subjects are diminished compared to those of sober subjects, but the grain of measurement for these data has been too coarse (20 or 30 second averages) to allow precise determination of exactly where the cardiovascular dampening effect begins and ends. The ANS undeniably has inherently longer "time-constants" than the facial expressive system, but we would predict that a precise second-by-second analysis of these data would reveal that the effects of alcohol on cardiovascular reactivity parallel the effects on facial responding in the three windows of reactivity that have been described. Such fine-grained analysis of physiological data would greatly enhance our understanding of the effects of alcohol on physiological responses to stress. Another issue for which we will soon be able to provide an answer is whether there are consistent gender differences in the effects of alcohol. Our recently completed dose-response study was the first of our studies to include female in addition to male subjects. And finally, we have speculated (e.g., Levenson et al., 1980) that the effects of psychological expectancy may be more pronounced at the moderate .5 g/kg dose than at the higher 1 g/kg dose, where we have consistently failed to find any effects attributable to

expectancy. We will find out whether this speculation is true when the data analyses from the dose-response study are completed.

Individual differences: Do some drinkers get "more bang for the buck?"

As noted at the start of this chapter, in addition to studying what alcohol does, we have also been studying how individuals differ in these effects. Inherent in our thinking has been the notion that if the short-term effects of alcohol are overwhelmingly positive and reinforcing, then any individual who was predisposed by nature or by nurture to experience an extra increment of these reinforcing effects would be much more likely to engage in higher rates of drinking behavior. But, who are these people?

Our studies of individual differences in the effects of alcohol are currently in midstream. We have been studying two groups that we believe may be predisposed to obtain more of the reinforcing effects of alcohol than do other people: (a) the children of alcoholics; and (b) individuals who fit a personality profile that includes traits of outgoingness, impulsiveness, aggressiveness, and antisociality. There is evidence that both groups are at heightened risk for alcoholism. A genetic factor in the incidence of alcoholism among children of alcoholics has been extensively studied (e.g., Goodwin, 1979; Goodwin & Guze, 1974). The personality profile we have described is one that has been shown to be at high risk for alcoholism in several prospective studies (e.g., Jones, 1968; McCord & McCord, 1960; Robins, Bates, & O'Neal, 1962).

When this research is completed, we will have studied both of these risk groups in terms of the full range of state and reactivity variables described in this chapter. Thus far we have completed studies of the personality risk factor that compared subjects who met this profile with those who did not in terms of alcohol's effects on resting ANS levels, and on ANS reactivity to the shock and speech stressors. The results have supported our hypothesis concerning individual differences, in that subjects who matched the personality profile had the most pronounced cardiovascular stress response dampening at the 1 g/kg dose (Sher & Levenson, 1982). Thus, these subjects are seen as deriving a larger portion of this potentially positive and reinforcing consequence of alcohol consumption. We believe that this may be an important factor in mediating their heightened risk for alcoholism, especially if it turns out that they are receiving similarly greater amounts of the other reinforcing consequences of alcohol as well. We should know in the next year or so whether this is true, and also whether the children of alcoholics show a similar pattern.

References

Carpenter, J.A. (1957). Effects of alcoholic beverages on skin conductance: An exploratory study. Quarterly Journal of Studies on Alcohol, 18, 1-18.

Child, J.S., Kovick, R.B., Levisman, J.A. & Pearce, M.L. (1979). Cardiac effects of acute ethanol ingestion unmasked by autonomic blockade. Circulation, 59, 120-125.

Coopersmith, S. (1964). Adaptive reactions of alcoholics and nonalcoholics. Quarterly Journal of Studies on Alcohol, 25, 262-278.

Davis, P.A., Gibbs, F.A., Davis, H., Jetter, W.W., & Trowbridge, L.S. (1941). The effects of alcohol upon the electro-encephalogram (brain waves). Quarterly Journal of Studies on Alcohol, 1, 626.

Devenyi, P., Robinson, G.M., & Roncari, D. A. (1980). Alcohol and high-density lipoproteins. Canadian Medical Association Journal, 123, 981-984.

Doctor, R.F., Naitoh, P., & Smith, J.C. (1966). Electroencephalographic changes and vigilance behavior during experimentally induced intoxication with alcoholic subjects. Psychosomatic Medicine, 28, 605.

Ekman, P., & Friesen, W.V. (1982). Felt, false and miserable smiles. Journal of Nonverbal Behavior. 6(4), 238-252.

Engel, G.L., & Rosenbaum, M. (1945). Delirium III: Electroencephalographic changes associated with acute alcoholic intoxication. Archives of Neurology and Psychiatry, 53, 44.

Exner, J.E. (1973). The self-focus sentence completion: A study of egocentricity. Journal of Personality Assessment, 37, 437-455.

Goodwin, D.W., & Guze, S.B. (1974). Heredity and alcoholism. In B. Kissin & H. Begleiter (Eds.). The Biology of Alcoholism, Volume 3: Clinical Pathology. New York: Plenum Publishing Corporation.

Goodwin, D.W. (1979). Genetic determinants of alcoholism. In J. H. Mendelson & N.K. Mello (Eds.), The diagnosis and treatment of alcoholism. New York: McGraw-Hill.

Gorlin, R. (1976). Coronary artery disease. Philadelphia: W. B. Saunders.

Greenberg, L.A., & Carpenter, J.A. The effect of alcoholic beverages on skin conductance and emotional tension: I. Wine, whisky, and alcohol. Quarterly Journal of Studies on Alcohol, 1957, 18, 190-204.

Hennekens, C.H., Rosner, B., & Cole, D.S. (1978). Daily alcohol consumption and fatal coronary heart disease. American Journal of Epidemiology, 107, 196-200.

Holmberg, G., & Martens, S. (1955). Electroencephalographic changes in man correlated with blood alcohol concentration and some other conditions following standardized ingestion of alcohol. Quarterly Journal of Studies on Alcohol, 16, 411.

Hull, J.G., Levenson, R.W., Young, R.D., & Sher, K.J. (1983). The self-awareness reducing effects of alcohol consumption. Journal of Personality and Social Psychology 44, 461-473.

Jones, J.C. (1968). Personality correlates and antecedents of drinking patterns in adult males. Journal of Consulting and Clinical Psychology, 32, 2-12.

Kannel, W.B., & Gordon, T. (1970). Some characteristics of the incidence of cardiovascular disease and death: Framingham study. 16-year follow-up. Washington, D.C.: U.S. Government Printing Office.

Knott, D.H. & Beard, J. D. (1972). in The Biology of Alcoholism, Vol.2: Physiology and Behavior, Kissin, B. and Begleiter, H. (ed.), New York: Plenum Press.

Kotani, K. (1965). EEG studies on endogenous psychoses and alcohol intoxication. Bulletin of the Osaka Medical School, Supplement 12, 11.

Lehrer, P.M., & Taylor, H.A. (1974). Effects of alcohol on cardiac reactivity in alcoholics and nonalcoholics. Quarterly Journal of Studies on Alcohol, 35, 1044-1052.

Levenson, R.W., Sher, K.J., Grossman, L., Newman, J., & Newlin, D.B. (1980). Alcohol and stress response dampening: Pharmacological effects, expectancy, and tension reduction. Journal of Abnormal Psychology, 89, 528-538.

Lienert, G.A, & Traxel, W. (1959). The effects of meprobamate and alcohol on galvanic skin response. Journal of Psychology, 48, 329-334.

McCord, W., & McCord, J. (1960). Origins of Alcoholism. Stanford, California: Stanford.

Newman, H.W. (1959). The effect of alcohol on the electroencephalogram. Stanford Medical Bulletin, 17, 55.

Robins, L.N., Bates, W., & O'Neal, P. (1962). Adult drinking patterns of former problem children. In D.J. Pittman & C.R. Snyder (Eds.), Society, culture and drinking patterns. New York: Wiley.

Rohsenow, D.J., & Marlatt, G.A. (1981). The balanced placebo design: Methodological consideration. Addictive Behaviors, 6, 107-122.

Sher, K.J. & Levenson, R.W. (1982). Risk for alcoholism and individual differences in the stress response dampening effect of alcohol. Journal of Abnormal Psychology, 91, 350-367. Reprinted in (1983). Digest of Alcoholism Theory and Application, 2, 40-47.

Thornton, J., Symes, C. & Heaton, K. Moderate alcohol intake reduces bile cholesterol saturation and raises HDL cholesterol. (1983). The Lancet. 2, 819-822.

Varga, B., & Nagy, T. (1960). Analysis of alpha rhythm in the electroencephalogram of alcoholics. Electroencephalography and Clinical Neurophysiology, 92, 933.

Wallgren, H., & Barry, H. Actions of alcohol, volumes I and II. New York: Elsevier, 1970.

Footnotes

1. Another possible mediator of this relationship is the increase in high density lipoprotein (HDL) that is associated with alcohol consumption (e.g., Thornton, Symes, & Heaton, 1983). HDL is thought to be an anti-atherogenic factor. High HDL levels in the blood have been shown to be associated with low levels of coronary heart disease, but the entire issue is controversial (e.g., Devenyi, Robinson, & Roncari, 1980)

Figure Captions

1. Effects of alcohol dose on self-disclosure. Alcohol reduces percentage of self-focus statements and increases percentage of external-focus statements.
2. Effects of alcohol dose on self-report of tension. Alcohol reduces self-reported tension (ANX).
3. Effects of alcohol dose on prestressor autonomic nervous system levels. Alcohol increases heart rate (HR), increases skin conductance (SCL), increases pulse transmission time (PTT), and increases dilation of blood vessels in finger (FPA).
4. Effects of alcohol dose on the cortical average evoked potential. Alcohol diminishes the amplitude of the evoked potential.
5. Effects of alcohol dose on the individual components of the average evoked potential. Alcohol reduces P1-N1, N1-P2, and P2-N2 components
6. Effects of alcohol on heart rate response. Data are plotted so that higher arousal (i.e., faster heart rate) is in the upward direction.
7. Effects of alcohol on pulse transmission time response. Data are plotted so that higher arousal (i.e., shorter pulse transmission time) is in the upward direction.
8. Effects of alcohol dose on cardiovascular responses at points of maximal reactivity. Alcohol reduces heart rate response to the countdown (HR-COUNTDOWN), heart rate response to the speech (HR-SPEECH), heart rate response to

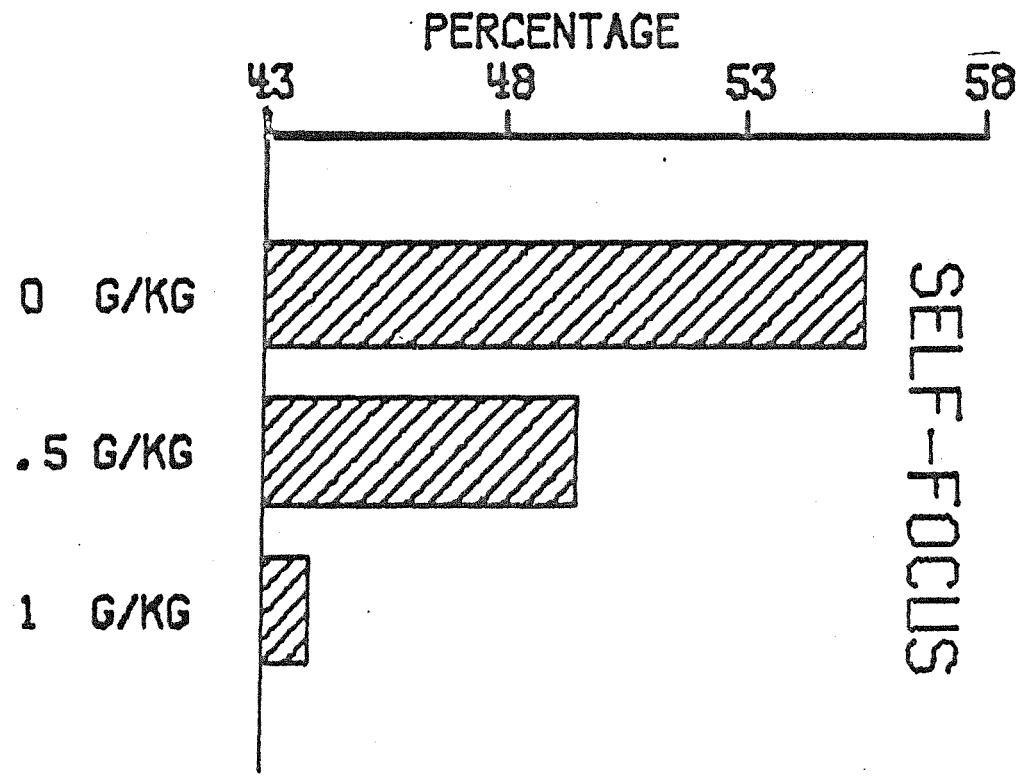
the shock (HR-SHOCK), and pulse transmission time response to the shock (PTT-SHOCK).

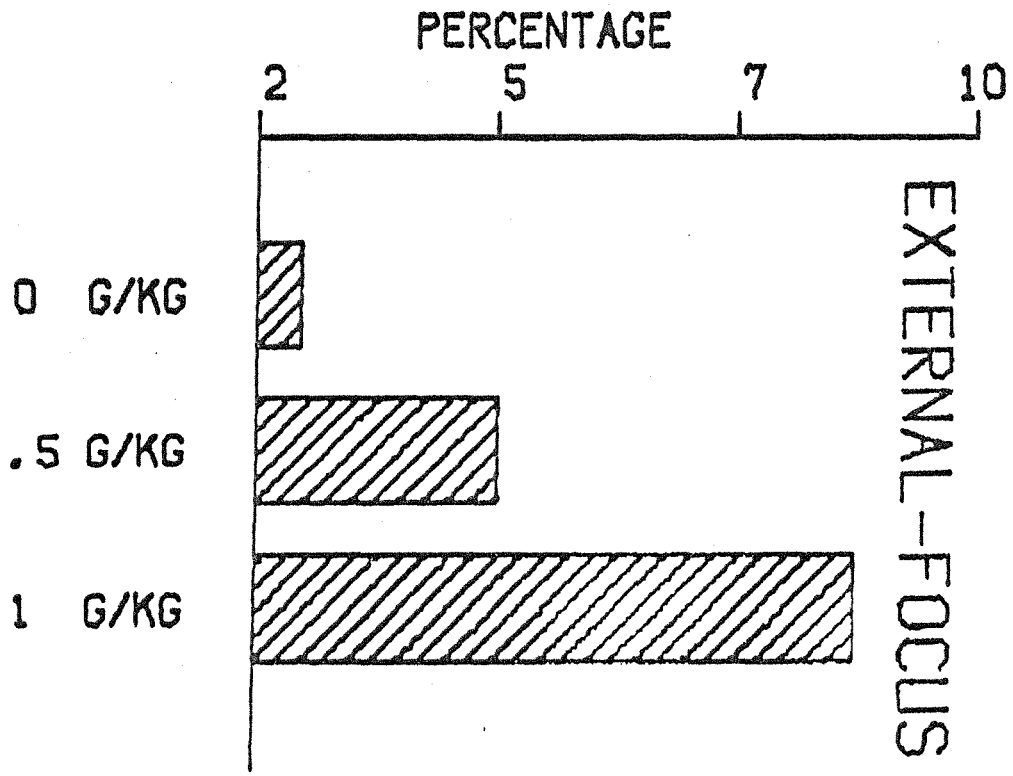
9. The prototypical facial expression of Fear. Note that brows are drawn up and together, upper eyelid is raised, lower eyelid is tensed, lip corners are pulled back horizontally. (Photograph copyright by Paul Ekman)

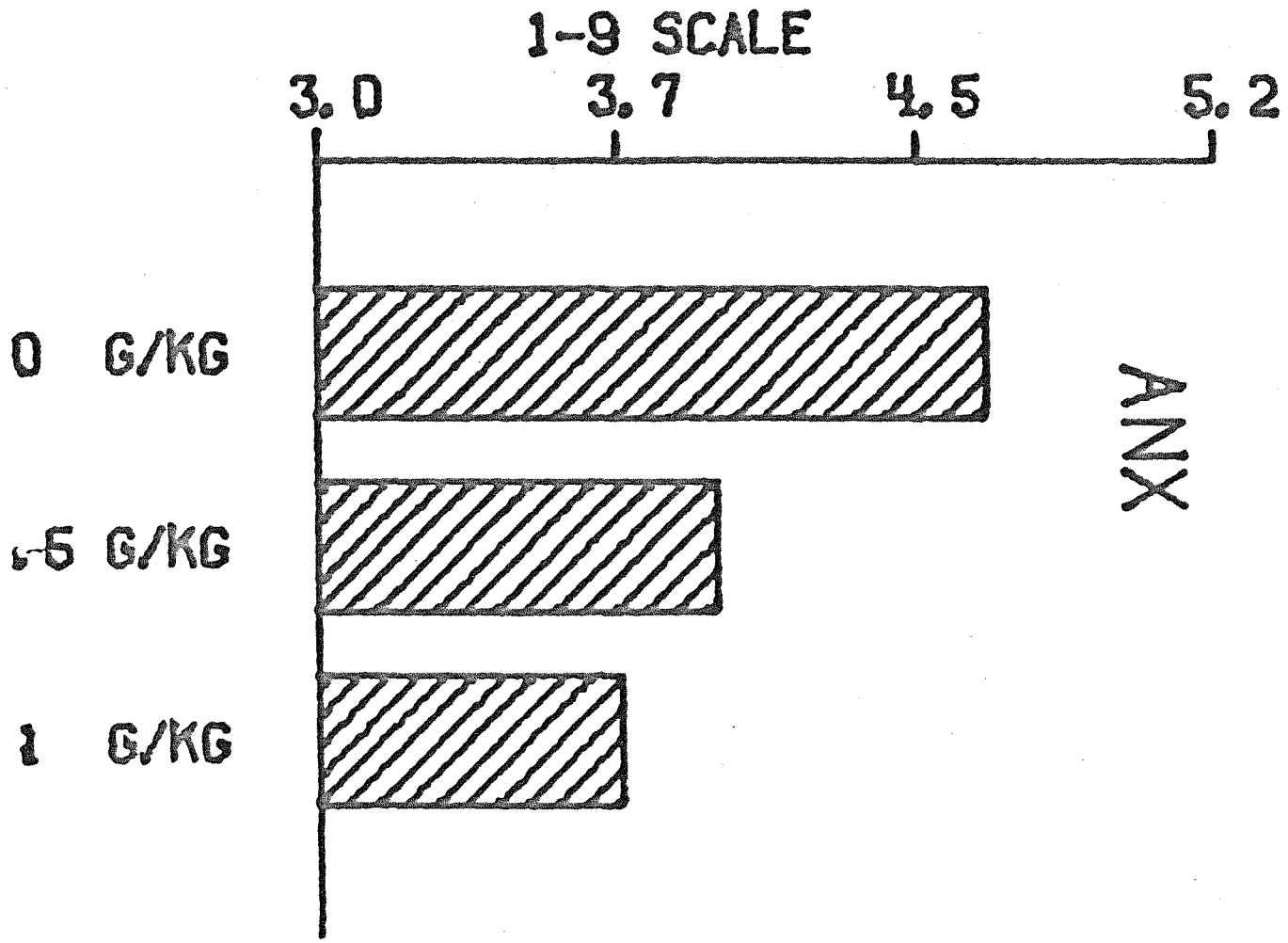
10. Effects of alcohol dose on facial expressions in anticipatory window preceding shock stressor. Alcohol reduces overall facial expressiveness, and in particular, signs of fear and attempts at emotional control.

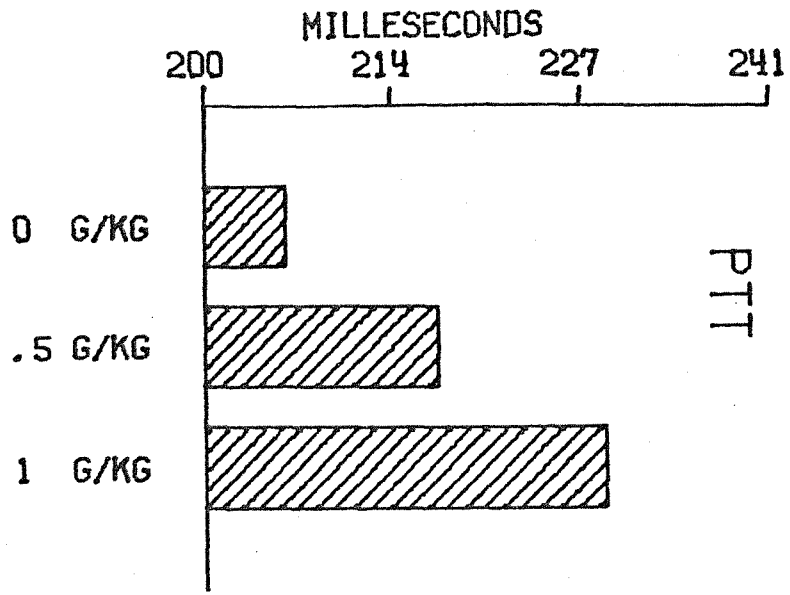
11. Effects of alcohol dose on facial expressions to shock stressor. Alcohol reduces number of subjects showing high intensity contraction of risorius muscle (pulls lip corners back horizontally).

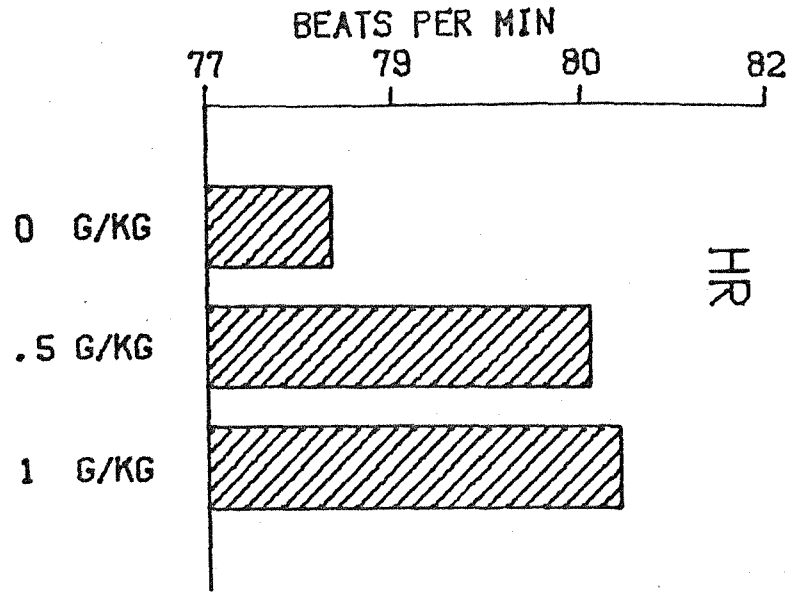
12. Effects of alcohol dose on facial expression in reaction window following shock stressor. Alcohol reduces the number of felt smiles.

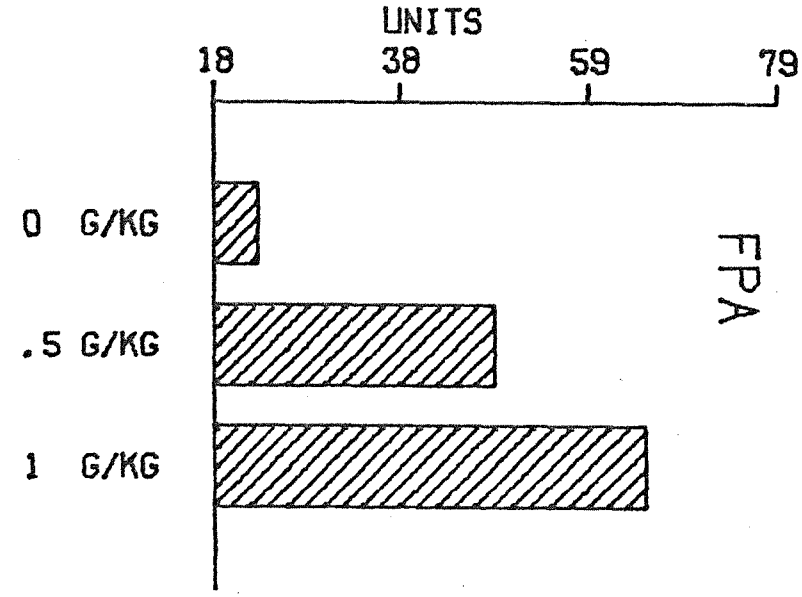

PERCENTAGE
43
48
53
58
0 G/KG
.5 G/KG
1 G/KG
SELF-FOCUS

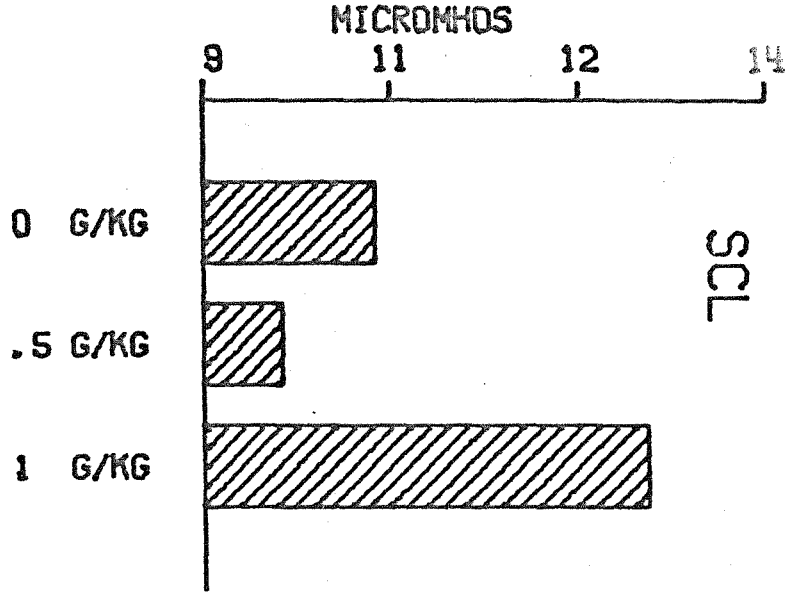

PERCENTAGE
2
5
7
10
0 G/KG
.5 G/KG
1 G/KG
EXTERNAL-FOCUS

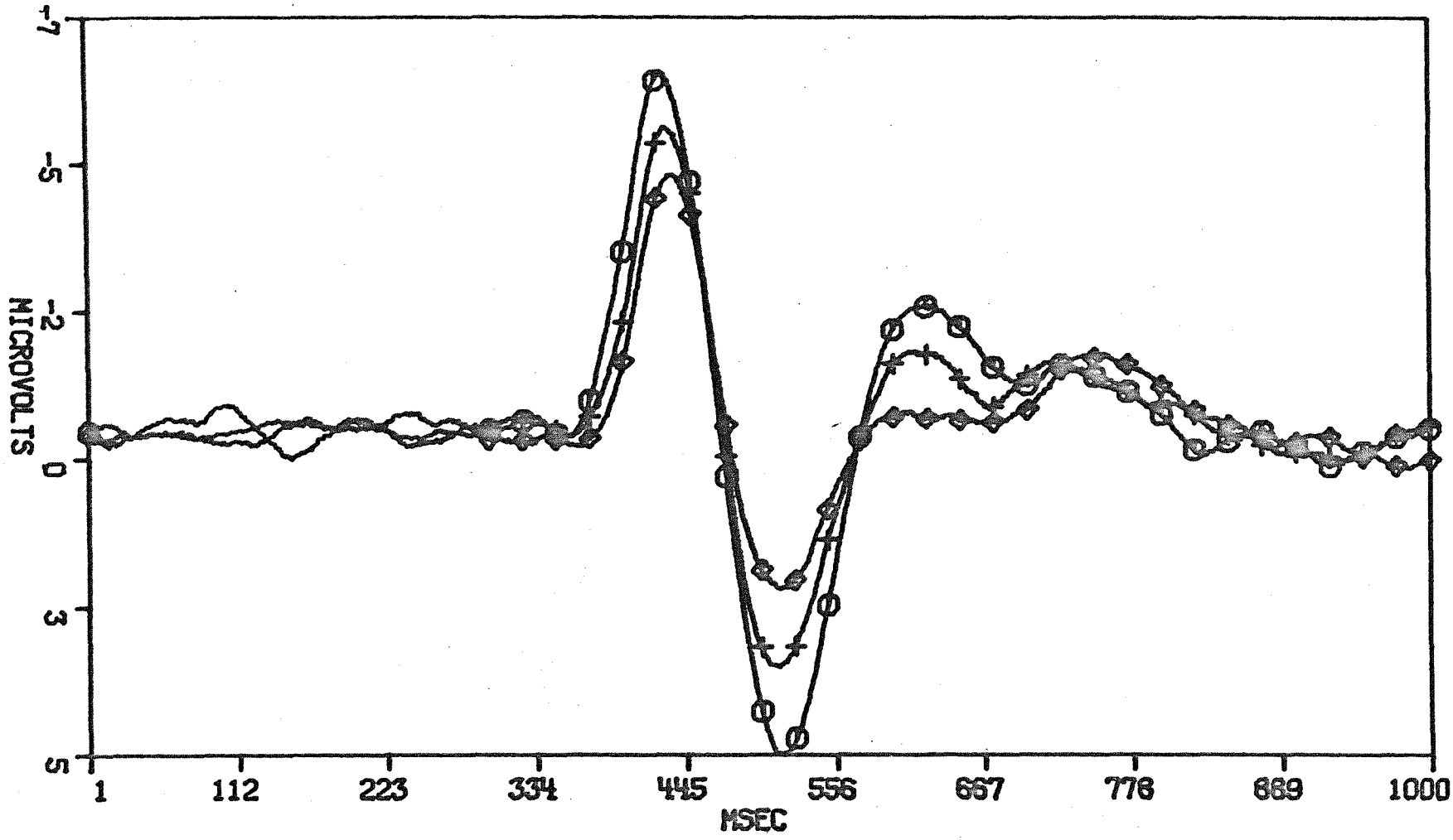

1-9 SCALE
3.0
3.7
4.5
5.2
0 G/KG
.5 G/KG
1 G/KG
ANX


MILLESECONDS
200
214
227
241
0 G/KG
.5 G/KG
1 G/KG
PTT


BEATS PER MIN
77
79
80
82
0 G/KG
.5 G/KG
1 G/KG
HR


UNITS
18
38
59
79
0 G/KG
.5 G/KG
1 G/KG
FPA


MICROMHOS
9
11
12
14
0 G/KG
.5 G/KG
1 G/KG
SCL

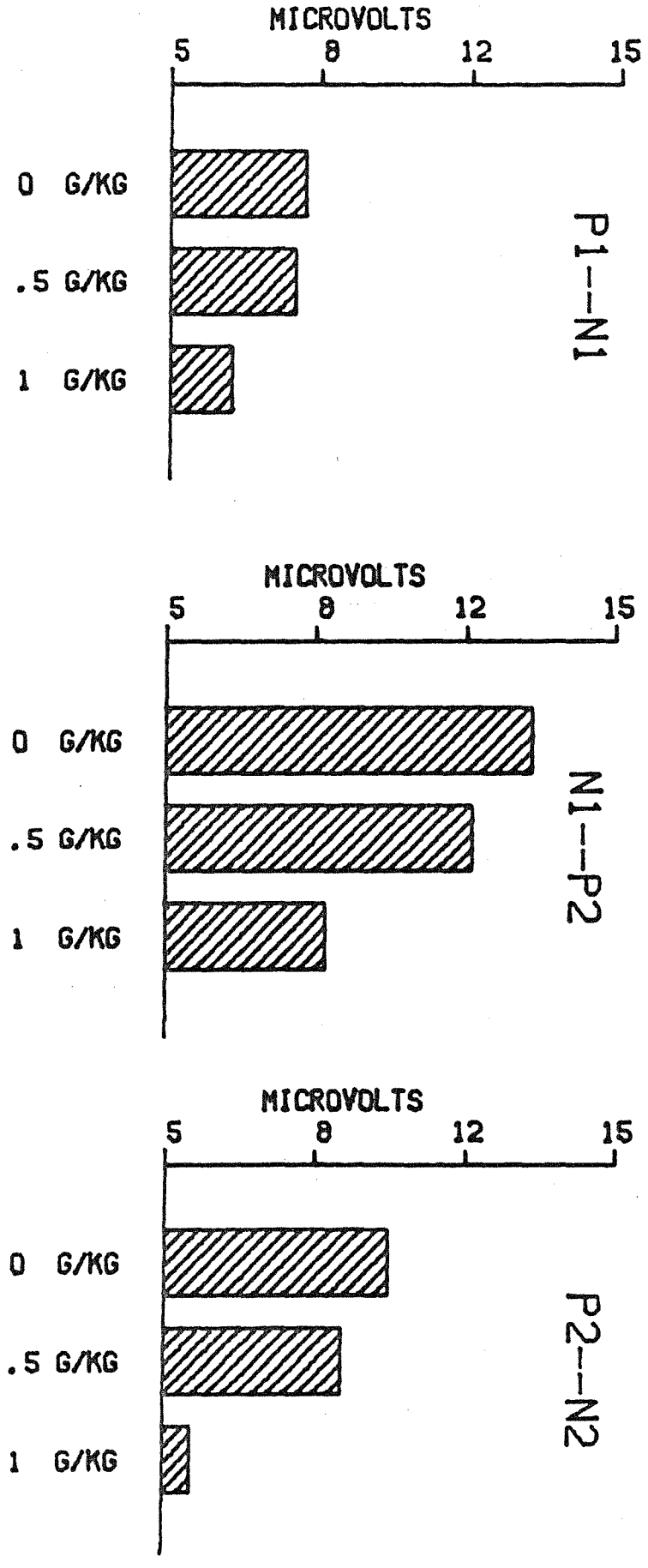


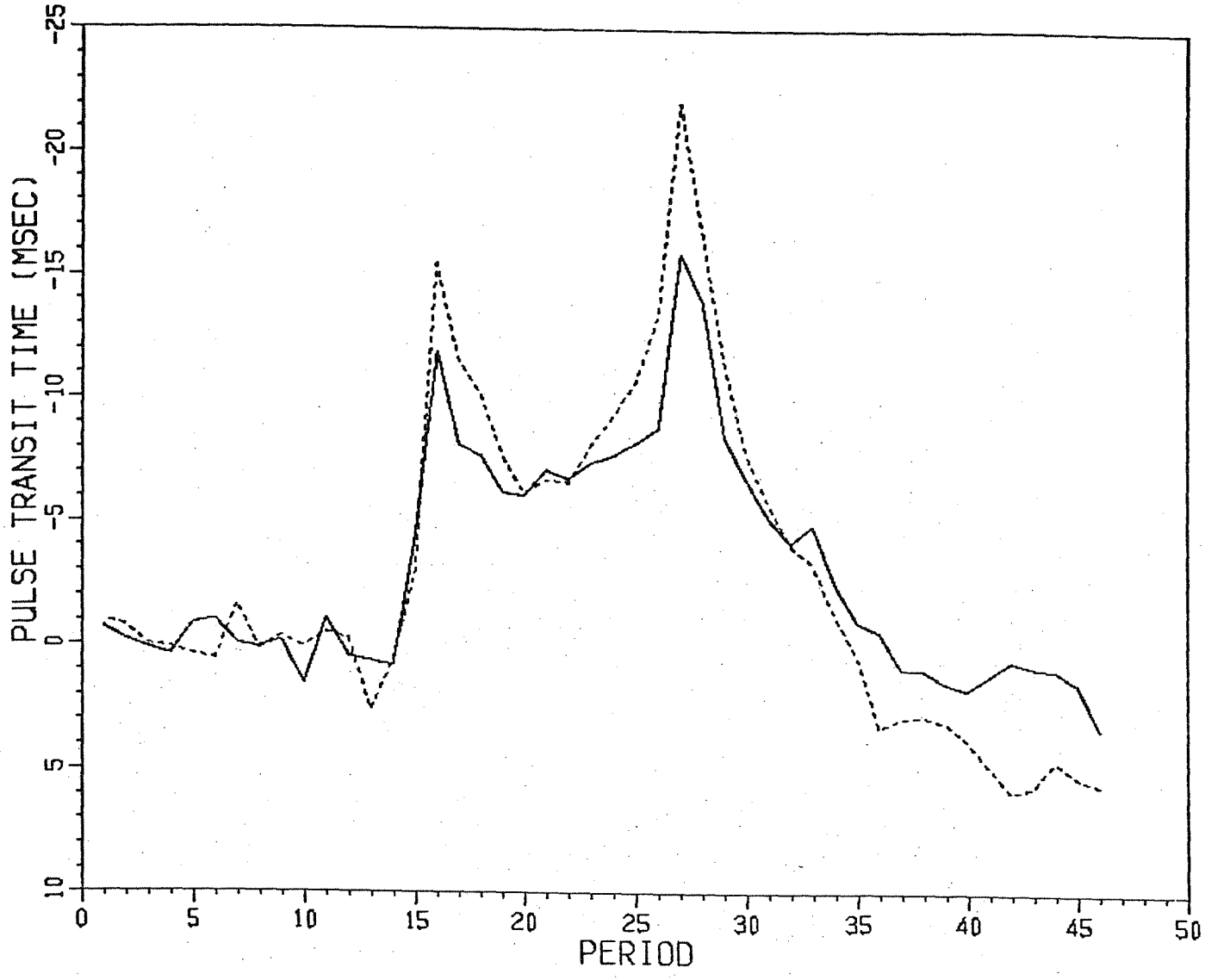
LEGEND

0 G/KG
.5 G/KG
1 G/KG

COMMENTS

TONE ONSET AT 300 MSEC


MICROVOLTS
5
8
12
15
0 G/KG
.5 G/KG
1 G/KG
P1--N1
MICROVOLTS
5
8
12
15
0 G/KG
.5 G/KG
1 G/KG
N1--P2
MICROVOLTS
5
8
12
15
0 G/KG
.5 G/KG
1 G/KG
P2--N2

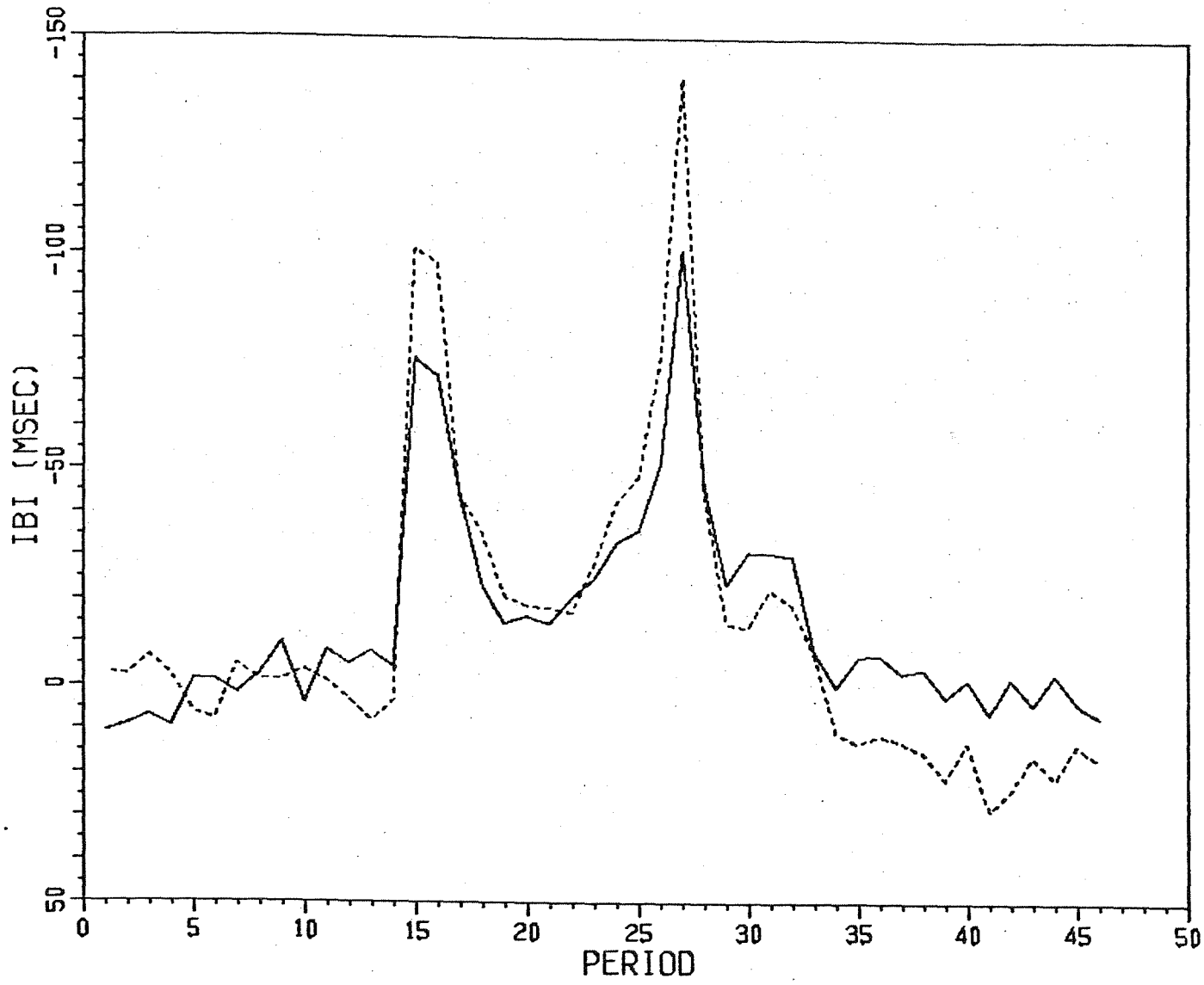


LEGEND

——— ALCOHOL
------- NO ALCOHOL

COMMENTS

PERIOD 15=START OF COUNTDOWN
PERIOD 27=STRESSOR

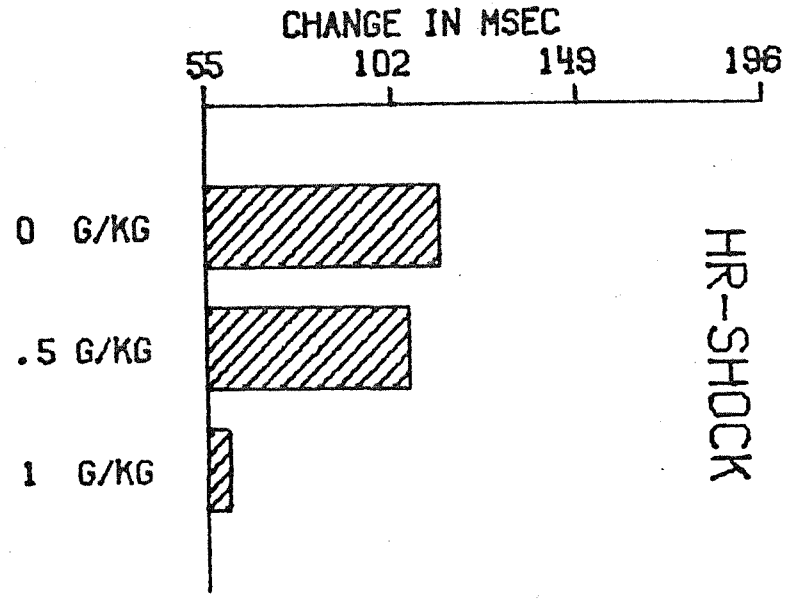


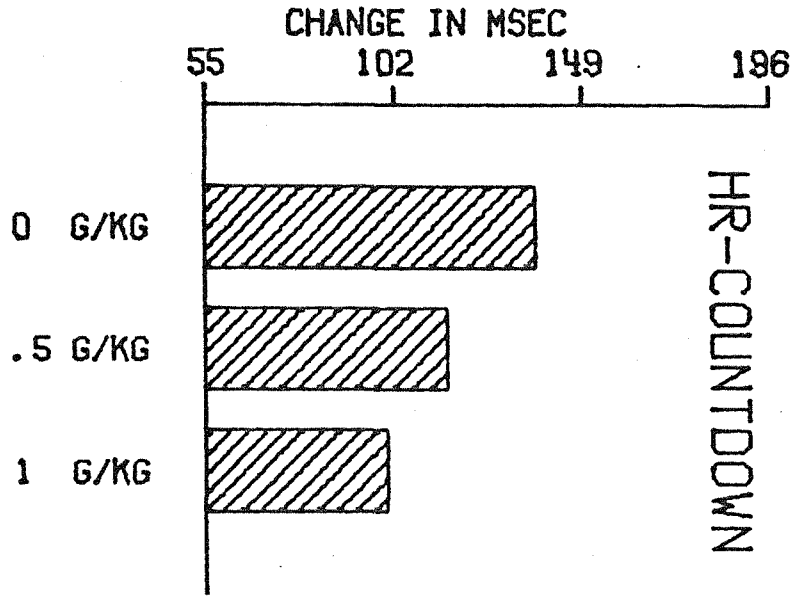
LEGEND

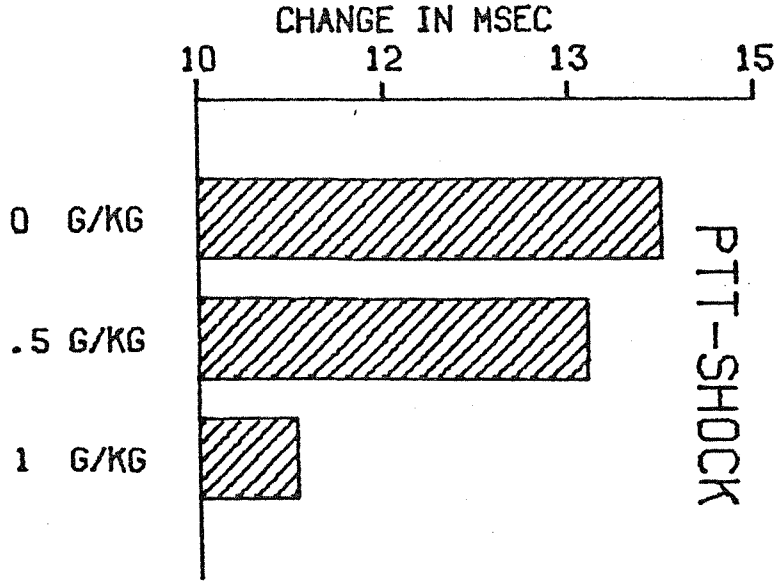
ALCOHOL
NO ALCOHOL

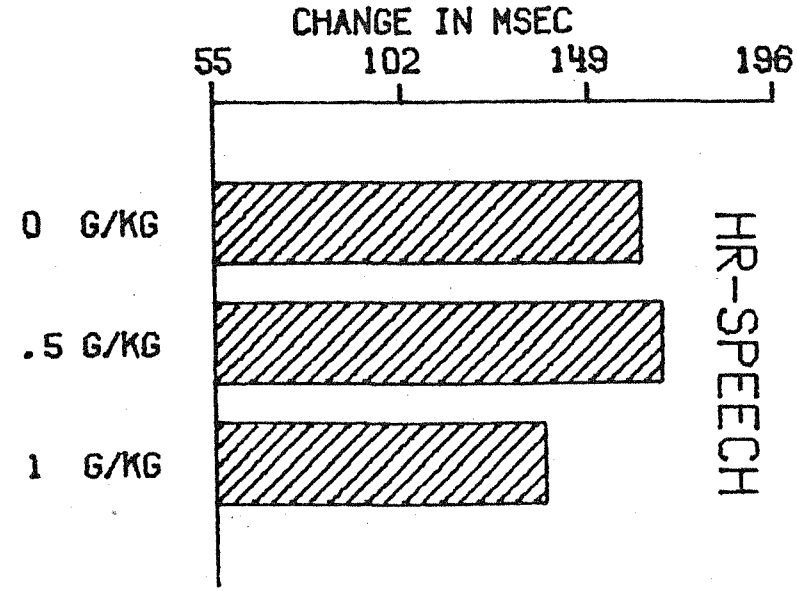
COMMENTS

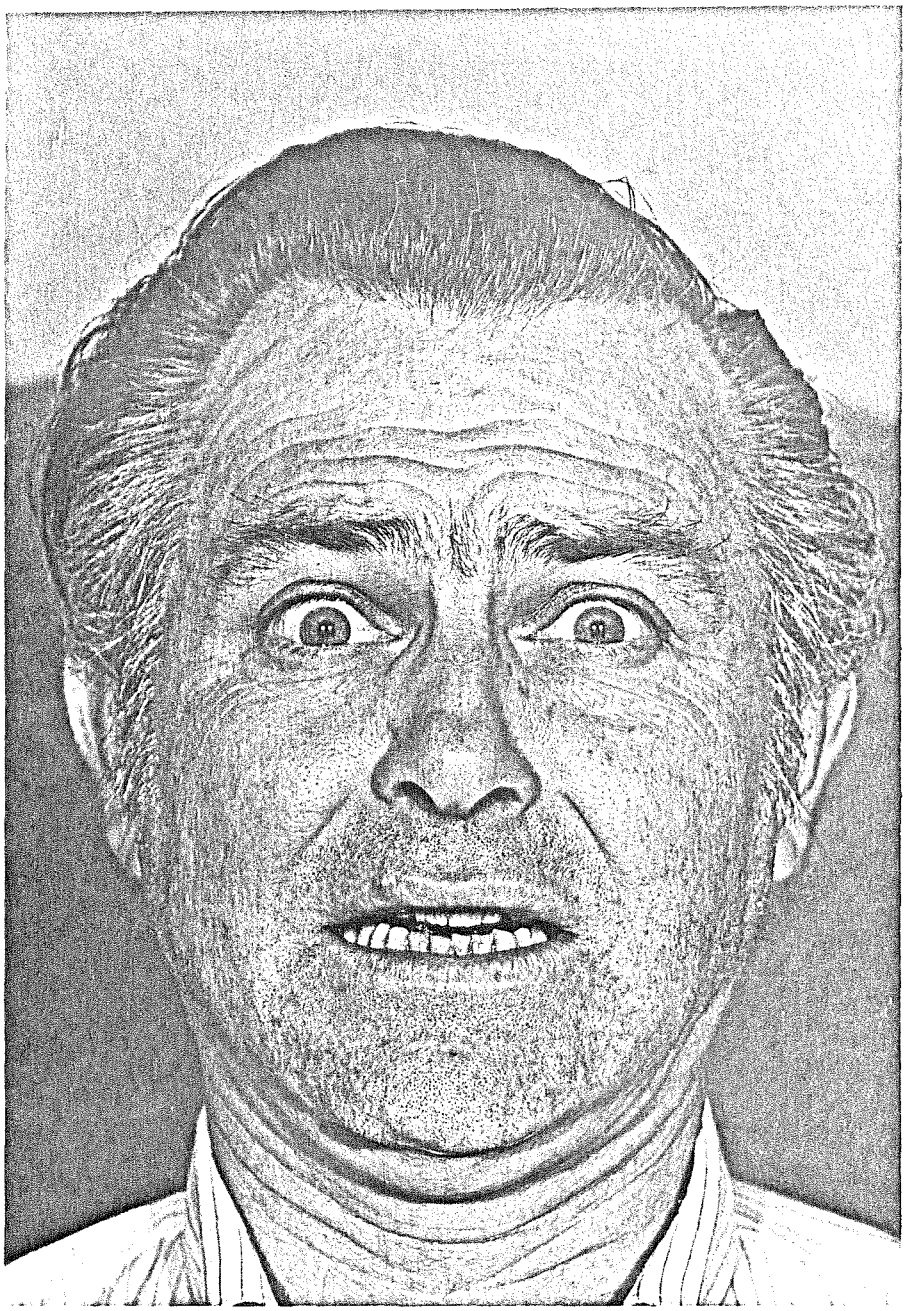
PERIOD 15=START OF COUNTDOWN
PERIOD 27=STRESSOR

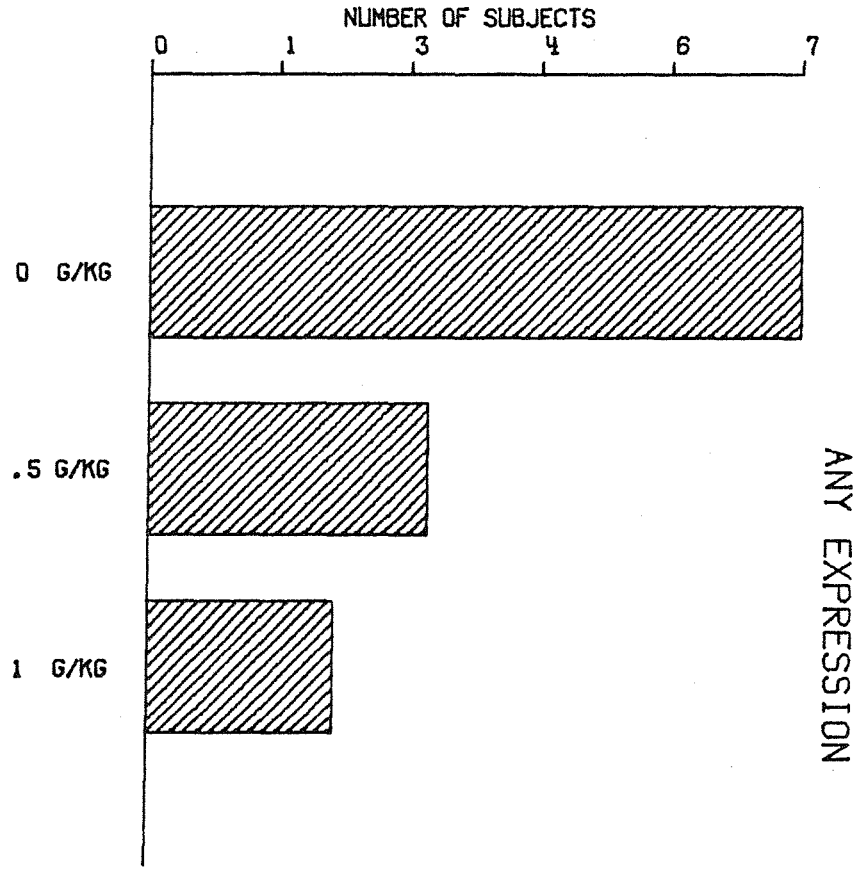

CHANGE IN MSEC
55
102
149
196
0 G/KG
.5 G/KG
1 G/KG
HR-SHOCK

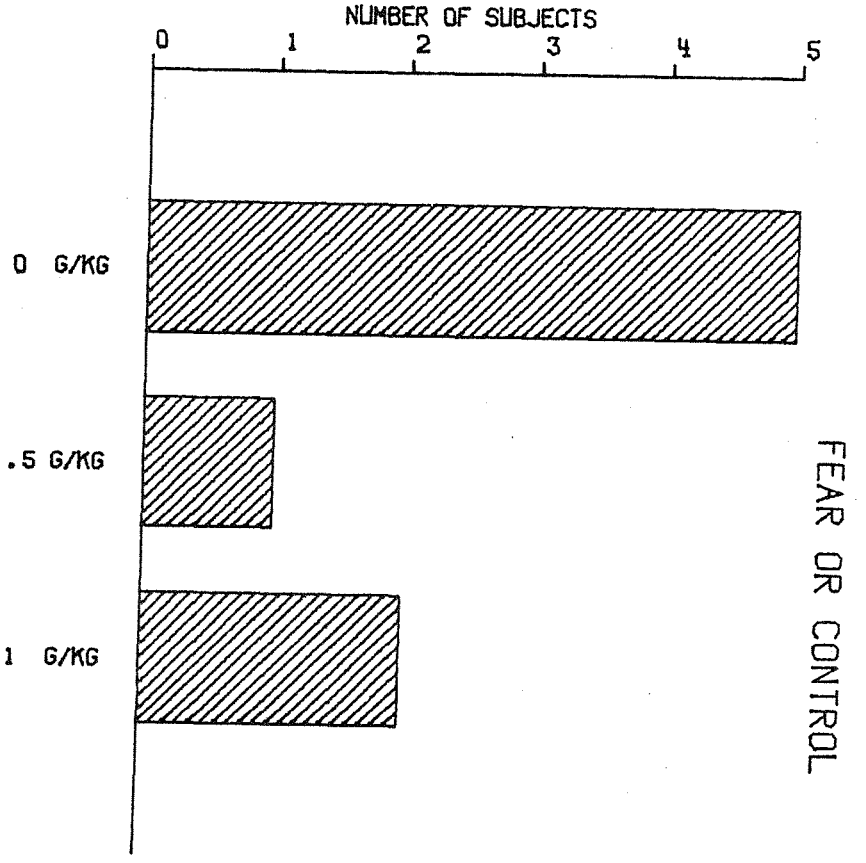

CHANGE IN MSEC
55
102
149
196
0 G/KG
.5 G/KG
1 G/KG
HR-COUNTDOWN

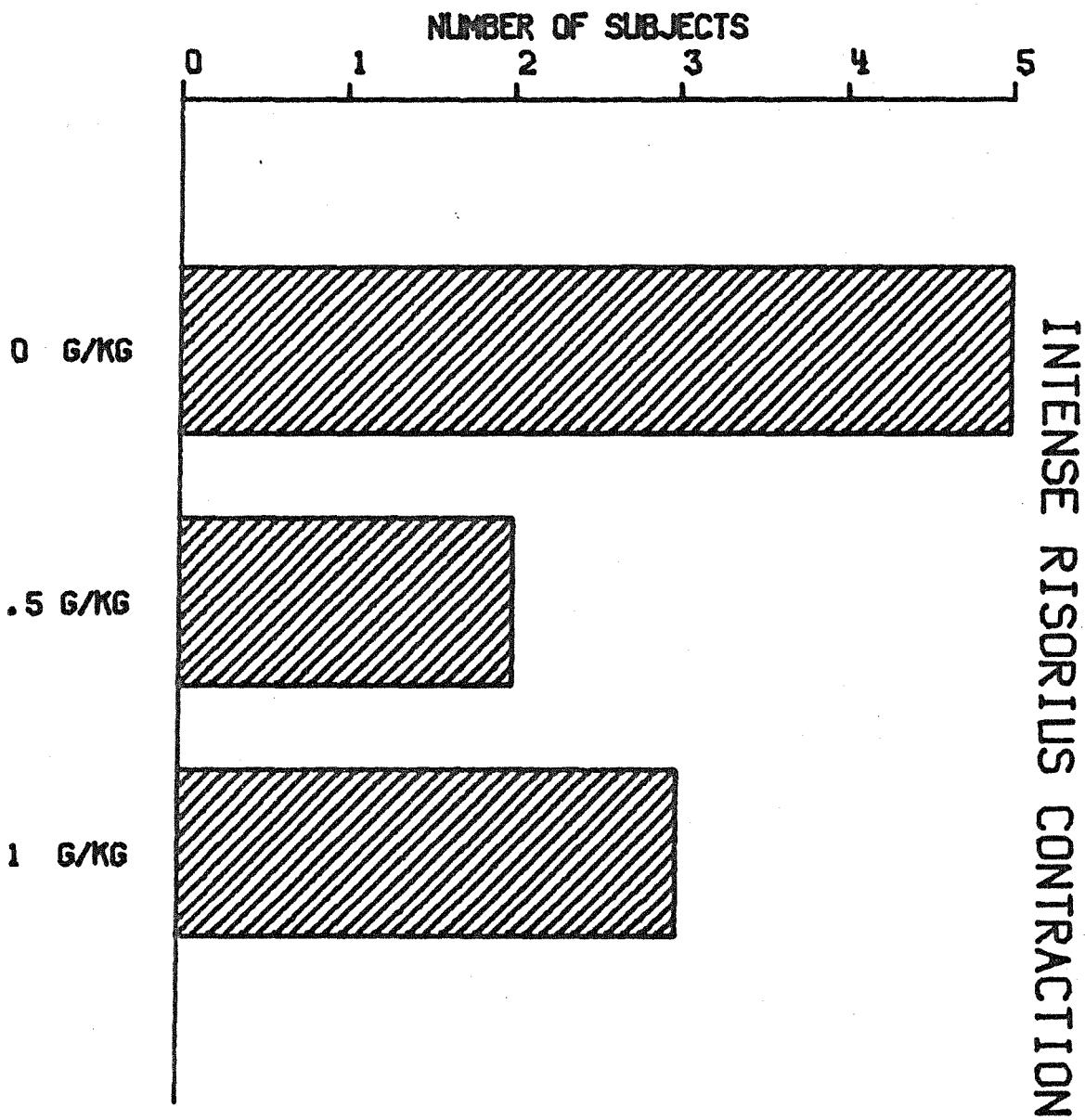

CHANGE IN MSEC
10
12
13
15
0 G/KG
.5 G/KG
1 G/KG
PTT-SHOCK

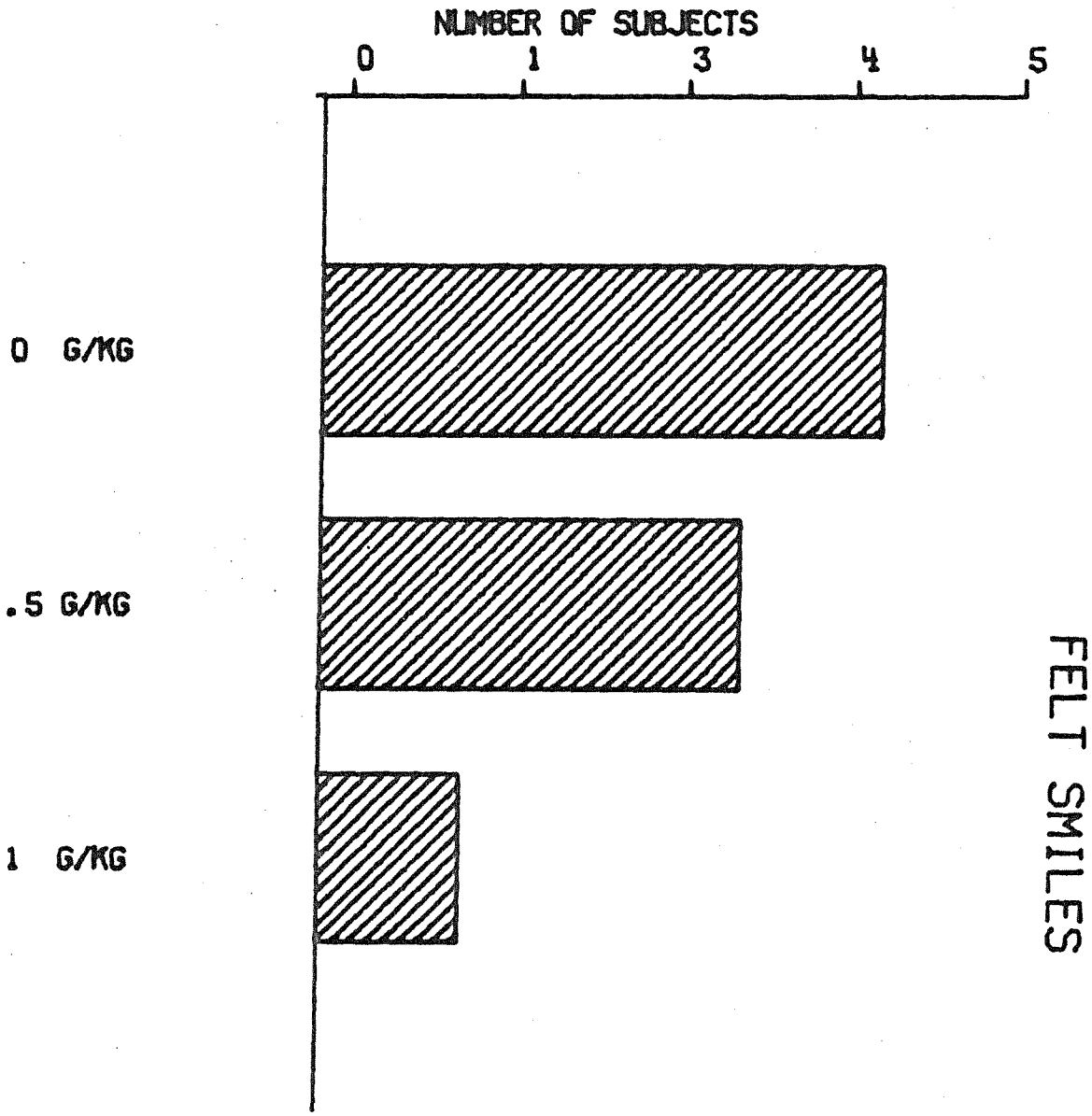

CHANGE IN MSEC
55
102
149
196
0 G/KG
.5 G/KG
1 G/KG
HR-SPEECH




NUMBER OF SUBJECTS
0
1
3
4
6
7
0 G/KG
.5 G/KG
1 G/KG
ANY EXPRESSION


NUMBER OF SUBJECTS
0
1
2
3
4
5
0 G/KG
.5 G/KG
1 G/KG
FEAR OR CONTROL


NUMBER OF SUBJECTS
0
1
2
3
4
5
0 G/KG
.5 G/KG
1 G/KG
INTENSE RISORIUS CONTRACTION


NUMBER OF SUBJECTS
0
1
3
4
5
0 G/KG
.5 G/KG
1 G/KG
FELT SMILES