Alcohol, reactivity, and the heart: Implications for coronary health and disease

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The relationships between alcohol and cardiovascular function and disease are inherently complex. A full accounting of these relationships would require making at least four critical distinctions: (a) acute effects of alcohol consumption vs effects associated with long term moderate use vs effects associated with long term chronic abuse; (b) effects in alcoholics vs effects in nonalcoholics; (c) effects on individuals with healthy cardiovascular systems vs effects on individuals with cardiovascular disease; and (d) effects of alcohol on resting, basal cardiovascular levels vs effects on cardiovascular reactivity. The existing literature on alcohol and the heart has not always taken account of all of these distinctions. Nonetheless, we do know that alcohol has a major impact on the heart and on processes of cardiovascular health and disease. In this chapter I will attempt to describe these effects and explore their bases.

Alcohol metabolism

At the outset, some basics principles of alcohol metabolism will be reviewed to facilitate an understanding of the cardiovascular effects that will be described later. Alcohol (ethanol) is absorbed rapidly into the blood stream from the stomach and the small intestine. The rate of absorption is slowed by the presence of food in the stomach. Maximally, only about 10% of the alcohol that is consumed is excreted or otherwise eliminated. Alcohol cannot be effectively stored in body tissues, thus the remaining 90% that is not eliminated directly in urine, breath, or perspiration is metabolized. Ethanol metabolism takes

place primarily in the liver. There the ethanol is oxidized by enzymes (mainly alcohol dehydrogenase; ADH) into its first metabolite, acetaldehyde, which is a toxic substance that has its own set of damaging cardiovascular effects. Normally, the acetaldehyde is quickly metabolized to form acetate. However in the presence of drugs used to treat alcoholism, such as Antabuse (disulfiram), the metabolism of acetaldehyde is inhibited and its levels increase. Acetate can be oxidized to CO_2 and H_2O or utilized for lipid synthesis.

Alcohol metabolism proceeds at a fairly steady rate of 100 mg ethanol/kg body weight/h. This is not an overly efficient system given typical alcohol doses. For example, a 150 lb individual drinking one ounce shots of 80 proof vodka would not completely metabolize even one drink in an hour (approximately 4/5 ounce would be metabolized). Extrapolating from this rate, one can see that, following a heavy bout of drinking in which a number of drinks were consumed, the removal of alcohol from the blood stream would take many hours. Experienced drinkers can take advantage of this constant metabolic rate to maintain a fairly constant blood alcohol level by spreading out their consumption of alcohol. Just as the rate of ethanol metabolism cannot be accelerated to accommodate an episode of rapid, high quantity drinking, it also cannot be accelerated to meet short term needs for energy. Thus, alcohol is not a particularly good food source to select when extra energy is required in a short period of time. Interestingly, alcohol cannot be metabolized by the heart, as there is no ADH present within the myocardium. However, the presence of acetaldehyde dehydrogenase in the heart enables the

metabolism of acetaldehyde.

Alcohol: links with disease

Chronic alcohol use can have deleterious effects on a strikingly wide range of organ systems. In a recent report to Congress on alcohol and health (1), evidence was reviewed that indicates alcohol is associated with disease processes in the gastrointestinal system, the liver, the cardiovascular system, the skeletal musculature, and the endocrine system. For present purposes I will focus on the harmful effects of alcohol on the heart.

Coronary heart disease.

Alcohol can damage the heart both indirectly through dietary factors associated with alcoholism (e.g., malnutrition) or directly. I will review some of the major disorders that result from each of these paths.

Dietary-mediated disorders. In the realm of dietary-mediated disorders, beriberi heart disease and cobalt beer cardiomyopathy are among the most prominent. Beriberi heart disease is caused by a deficiency in thiamine that persists for several months. Implicated in this disorder are diets that emphasize consumption of high carbohydrate foods such as polished rice and alcohol, both of which are deficient in thiamine. In this disorder there is a marked dysfunction in circulation primarily related to a reduction in peripheral vascular resistance. In response, both heart rate and cardiac output are increased with a resultant decrease in circulation time. Symptoms include warm skin, widened pulse pressure, anemia, apical

systolic murmur, enlargement of the heart, dilation of the pulmonary arteries, and neuritis of the peripheral nerves. A second dietary-mediated disorder, cobalt beer cardiomyopathy, was responsible for a high mortality rate among beer drinking alcoholics in the 1960's. During that period, cobalt was added to beer to improve its foaming qualities. The disorder that resulted was characterized by right-sided congestive cardiac failure (2) due in part to cobalt toxicity and due in part to malnutrition. With the banning of the practice of adding cobalt ions to beer, cobalt beer cardiomyopathy has been virtually eliminated.

Alcoholic cardiomyopathy. Alcoholic cardiomyopathy is the term applied to primary heart muscle disease in alcoholics that is not related to coronary, hypertensive, valvular, congenital or pulmonary heart disease, but rather to an intrinsic defect in the heart muscle itself (3). It should be added that there is increasing evidence from studies that have controlled for malnutrition that this disorder results from a direct toxic effect of acetaldehyde on heart muscle (4-6). The damage, which consists of noninflammatory lesions of the myocardium, appears to be centered in the mitochondria (7,8). As these structures are involved in the oxidation of energy sources, the availability of energy to the heart muscle is greatly compromised, and over time, this can lead to congestive heart failure. Increased myocardial accumulation of lipids is another feature of the disorder. Symptoms consist of shortness of breath and signs of congestive failure (e.g., edema, chest pain, fatigue, palpitation, blood-stained sputum). Alcoholic cardiomyopathy is slow to develop (10

years of heavy drinking is typical) and recovery is also slow (typically taking up to six years after the cessation of drinking).

Other harmful effects of alcohol on heart function. Alcohol has pernicious effects on the efficiency of the heart's contraction. This has been shown most clearly in terms of an impairment of left ventricular functioning at moderate doses in man (9,10) and in animals (11-13). Alcohol has been shown to negatively affect biochemical processes in the heart that are necessary for muscle contraction, including calcium uptake and binding (14), and sodium/potassium activation of adenosine triphosphatase (ATPase) activity of cardiac plasma membranes (15). These effects of alcohol on the heart muscle no doubt contribute to findings that alcohol intoxication and alcoholism lead to an increase in cardiac arrhythmias (16,17). These arrhythmias are thought to be involved in some cases of sudden death, particularly among young alcohol users (18). Among patients with existing heart disease, the depressant effects of alcohol on myocardial performance (19) are potentially quite dangerous.

Alcohol: Links with coronary health

Thus far I have presented an overwhelmingly negative picture of the long term effects of alcohol abuse on the heart. Clearly, like all other drugs, there is a dose at which the substance becomes toxic. For alcohol, the toxic effects are particularly insidious for two reasons. First, they seem to be cumulative over time, with a number of alcohol-related cardiac disorders taking years to develop (e.g., 10 years for alcoholic cardiomyopathy).

And second, in the short term, moderate alcohol consumption may have a number of beneficial effects on the heart.

Epidemiological studies

There is now a substantial amount of evidence from a large number of epidemiological studies indicating that moderate consumption of alcohol (approximately two-three drinks per day) is associated with a lowered risk for coronary heart disease. The nature of the relationship seems to be "J-shaped", with heavy drinkers being at the highest risk, followed by nondrinkers (i.e., abstainers), followed by moderate drinkers. Whether a given study finds this "J-shaped" relation or more of a "U-shaped" relation (i.e., heavy drinkers and nondrinkers being at equally elevated risk compared to moderate drinkers) may depend on a number of factors, including the range of consumption levels that are sampled.

In the best of these studies, controls have been included for other risk factors such as smoking, age, race, and gender. A number of different methodologies have been used. Hennekens, Rosner, and Cole (20) compared drinking patterns in patients with heart disease and in matched controls. They found more abstainers and heavy drinkers among the patients. Using data from participants in Oakland's Kaiser-Permanente Health Plan, Klatsky, Friedman, and Siegelaub (21) found that moderate drinkers were less often found among cardiac patients than among controls, while abstainers were more often found among patients than among controls. In the prospective Tecumseh Community Health Study (22) the "J-shaped" relation was also found, with even higher risk for

heart disease among former drinkers who had stopped drinking. In the prospective Honolulu Heart Study (23) moderate drinkers were found to be at the lowest risk for myocardial infarctions. In a prospective study of Puerto Rican men (24), the "J-shaped" relation was found for angina pectoris, nonfatal myocardial infarctions, and nonsudden coronary heart disease death. In both this study and the prospective Yugoslavia Cardiovascular Disease Study (25), no association was found between alcohol consumption and sudden cardiac death.

As is often the case with these kinds of epidemiological data, there are a number of problems. Found relationships between alcohol consumption and cardiac health may well be mediated by other factors, and the direction of causality cannot be determined. Measurement problems also abound. There are a number of ways of determining how much alcohol a person consumes on average, most of which involve retrospective recall. Regardless of how accurate such recall is over the short term, it is unlikely that most people can maintain this kind of accuracy over the long term. If it is the cumulative effects of chronic alcohol use that are thought to be of etiologic significance in some forms of heart disease, then a measure of the pattern of alcohol consumption over a 5-10 year period is called for. Of course, such a measure would be very difficult to obtain. There are other problems as well. LaPorte, Cresanta, & Kuller (26), in a very thorough examination of the implications of the alcohol-heart disease relation, point to the importance of separating life-long nondrinkers from those who have stopped drinking after a history of heavy consumption. It is unlikely that the pathways

that mediate the purported higher risk for heart disease among heavy drinkers are the same as those that mediate the risk for abstainers. As indicated earlier, whether a curvilinear relationship appears to be "U-shaped" or "J-shaped" may be a function of where the lines between categories of drinking are drawn and how wide a range of consumption levels are included.

What seems to be most consistent in all of these studies is the lowered risk for coronary disease among moderate social drinkers. I will turn now to an examination of some potential mediators of this lowered risk.

Alcohol and high density lipoproteins

One potential mediator of the lowered risk for coronary heart disease among moderate drinkers that has received a great deal of attention is the relationship between alcohol consumption and the production of high density lipoprotein cholesterol. Cholesterol is a lipid that is nonsoluble in water; to be transported in the blood stream, it must be attached to a soluble substance. To accomplish this, cholesterol binds with various lipoproteins, which are termed low density lipoprotein (LDL) or high density lipoprotein (HDL) depending on their density and molecular structure. At one time it was thought that the overall level of serum cholesterol was correlated positively with risk for coronary heart disease (27); however, it is now believed that the correlation is positive for LDL, but negative for HDL (28,29). The role of HDL in coronary health is not fully understood. HDL appears to transport cholesterol from the tissues to the liver for excretion. It is also thought to be less likely

to be incorporated in atherogenic plaques than LDL (30), and may even block the incorpation of LDL in these plaques.

Alcohol consumption has been shown to be associated with elevated serum HDL levels in a number of studies (31-34). This has led to speculation that the association between moderate alcohol consumption and lowered risk for coronary heart disease may be mediated in part by the increase in HDL associated with alcohol consumption. This speculation is strengthened by the observation that the amount of alcohol associated with significantly elevated HDL levels (35) and the amount of alcohol associated with lowered risk of coronary heart disease are similar (i.e., about three drinks per day). Elevated HDL levels seem to reside quickly once alcohol consumption is reduced; thus sustaining these levels would require a long term commitment to maintaining a level of moderate drinking (with the incumbent risks of increased tolerance leading to increasingly higher levels of drinking). Furthermore, the clinical significance of the HDL increase associated with alcohol is uncertain, especially since the subclass of HDL that is increased by alcohol consumption is not the subclass (i.e., HDL_2) that is thought to have anti-atherogenic properties (36).

Alcohol and diminished neuroendocrine reactivity

Perhaps the most elegant and convincing work here are the animal studies carried out by Brick and Pohorecky (37-39). In a series of studies with rats, they have shown that alcohol diminishes the stress-induced increases in the blood of: (a) free fatty acids; and (b) corticosterone. There is an intimate

intertwining of functions in stress between the autonomically-mediated changes in cardiac function, the release of free fatty acids from adipose tissue (mediated by direct sympathetic innervation of these tissues or by action of catecholamines released from the adrenal medulla), and the release of corticosterone from the adrenal cortex. Lowered neuroendocrine activation could have beneficial cardiovascular effects. For example, Carruthers (40) has pointed to the potentially atherogenic effects of the free fatty acids released under the influence of catecholamine action in response to stress. In addition, both the catecholamines and corticosteroids may have direct toxic effects on the coronary arteries (41). If Brick and Pohorecky's findings in rats hold for humans and thus, alcohol consumption in humans also reduces the amount of catecholamines, free fatty acids, and corticosterone released in response to stress, then the potential benefits for cardiovascular health are considerable.

Alcohol and diminished cardiovascular reactivity

In this section, I will be presenting data obtained from my own laboratory relevant to understanding the acute effects of alcohol consumption on cardiovascular reactivity. This work has only studied nonalcoholic experienced social drinkers (they drink approximately two drinks per day) and will be used to develop a second potential bridge between moderate alcohol use and cardiovascular health--diminished cardiovascular reactivity. The notion that a reduction in cardiovascular reactivity is health-promoting is based on models of cardiovascular disease that posit

a causal role for an overreactive heart and vasculature. This hyperreactivity model is implicit in many psychological theories of the etiology of coronary heart disease. For example, one hypothesized mediational pathway between the Type A personality profile and cardiovascular disease is cardiac hyperreactivity (e.g., recent reviews 42,43). It is unknown whether this hyperreactivity reflects the presence of an ongoing disease process or whether it is a causative element in disease etiology (or both). Models of the role of cardiac hyperreactivity in the etiology of cardiac disease range from simple heuristic models (e.g., "a heart that is always working harder wears out faster") to the elaborate. An example of the latter can be found in Obrist's model (44), in which cardiac hyperreactivity is linked with atherosclerotic processes in the etiology of essential hypertension. Clearly if these models are true, or even partly true, anything that reduces cardiovascular reactivity has the potential for interrupting or slowing these disease processes.

In our work (45-47) subjects have been administered a weight-corrected dose of alcohol, typically utilizing a double-blind balanced placebo design (48). A wide range of central nervous system, autonomic nervous system, and behavioral variables have been studied, both in terms of how alcohol affects basal levels and how it affects reactivity when the subject is exposed to laboratory stressors (e.g., loud tones, electric shock, self-disclosure). We have studied approximately 400 subjects in these studies and the pattern of cardiovascular findings that has emerged has been remarkably consistent.

Subjects start the experimental procedure in a completely sober state (verified by Breathalyzer) and then consume one of three doses of alcohol (either 0 g ethanol/kg body weight, .5 g/kg, or 1.0 g/kg) in 45 minutes. For example, a 145 lb subject would consume 7 oz of vodka in the high dose (1 g/kg) condition, which would produce a blood alcohol concentration of approximately .10%. This is the legal intoxication limit in most states. We wait another 30-45 minutes to allow for absorption and then obtain "basal" physiological measures while subjects wait for the experiment to start. Using these procedures, we have found in three different studies that alcohol causes an increase in resting heart rate and a lengthening of pulse transmission time (i.e., the time interval between the R-wave of the electrocardiogram to the arrrival of the pulse pressure wave at the finger). Longer pulse transmission times are indicative of decreases in cardiac contractile force, to the exent that part of the lengthening is accounted for by longer pre-ejection periods (49). Obviously our procedures are not ideal for obtaining estimates of baselines, since subjects know they will be subjected to the stressor later in the experiment. Nonetheless, these cardiovascular findings are quite consistent with those reported by others who have utilized other baseline procedures. This is true both for heart rate (50-53) and for measures of contractile force (9,10,54,55).

In Figure 1 these effects can be seen across three dosage conditions. In all of our studies, we have found heart rate increases of about six beats per minute and pulse transmission time increases of about 11 msec in the 1 g/kg high dose, compared

to the 0 g/kg no alcohol condition. We have also measured finger pulse amplitude in one study, and found alcohol to increase it; this would be consistent with alcohol's known vasodilative properties.

Insert Figure 1 about here

It is likely that changes in heart rate and in contractility are compensatory adjustments that function to maintain a stable cardiac output (probably the heart rate increase compensates for a direct depressant effect on the myocardium produced by alcohol). While these are statistically significant changes, they are not clinically significant. Nonetheless, from the point of view of the experimenter hoping to assess cardiac reactivity, they must be taken into account. While these changes are not so large as to run the risk of subjects reaching cardiac ceiling and floor limits, they are sufficiently large to play havoc with the interpretation of reactivity differences across conditions.

There are many mathematical approaches to the problem of adjusting for differing baselines across conditions (as would occur in a study of cardiovascular reactivity that compared intoxicated and sober subjects). These approaches range from the computation of simple change scores (which we have used in our research), to more complicated techniques based on regression and covariance. Any of the standard correction techniques is better than no correction. Unfortunately the literature includes many studies that have failed to distinguish the effects of alcohol on cardiovascular baselines from the effects on cardiovascular

reactivity, and also with studies that have found basal effects, but then have failed to correct for them in their analyses of reactivity.

After correcting for basal effects, we have consistently found alcohol to diminish cardiovascular reactivity. This has occurred in multiple studies, using different stressors, and in measures of both heart rate and pulse transmission time. In Figures 2 and 3, these effects, which we have termed "stress response dampening" effects of alcohol, can be seen for heart rate and pulse transmission times. In contrast to the effects of alcohol on basal levels for these two measures (which were in different "directions"--an arousal effect in speeding heart rate, and a relaxant effect in lengthening pulse transmission time), the effects on reactivity consisted of the same diminished reactivity for both. It is also interesting to note that the stress response dampening effects of alcohol are not global autonomic effects. Skin conductance responding, for example, has not been attenuated by alcohol in any of our studies.

Insert Figures 2 and 3 about here

Our confidence in the robustness of these effects on cardiovascular reactivity has been increased by similar findings from other laboratories (56,57). However, it should be noted that we have found that pronounced dampening of cardiovascular reactivity occurs only with the high dose (1 g/kg). This can be readily seen in Figure 4, in which changes in heart rate and pulse transmission time from prestressor levels are plotted for

three different doses during times of maximum cardiovascular reactivity (i.e., while subjects watch a timer count down the last few seconds before the stressor, when they receive a painful electric shock, while they give a self-disclosing speech).

Insert Figure 4 about here

The overall picture from this research to date is that of diminished cardiac reactivity produced by a fairly high dose of alcohol. Assuming models of cardiovascular disease etiology that posit a etiological role for heightened cardiac reactivity are true, these findings that alcohol reduces cardiovascular reactivity provide another mediational pathway for understanding the relation between moderate alcohol use and lowered incidence of coronary heart disease.

As always there are caveats. All of the results I have described were obtained with nonalcoholic subjects with healthy cardiovascular systems. Alcoholic subjects may well present a different picture, with greater tolerance to the cardiovascular effects of alcohol combined in some fashion with greater vulnerability to the cumulative toxic effects of alcohol. In addition, subjects with existing cardiovascular disease may be much more sensitive to the myocardial depressant effects of alcohol. This could become life threatening at high doses. Finally, the effects of alcohol on cardiovascular reactivity may well be as sensitive to the drinker's position on the slope of the ethanol absorption curve as are the central nervous system effects. Many of these latter effects reverse in direction and

rebound at different times over the time course of absorption.

Alcohol and diminished emotional reactivity

The final pathway to be explored is that of alcohol's effects on emotional reactivity. Here what we know is based only on the most preliminary of data. A great deal of study has been done on the effects of alcohol on subjects' self reports of mood. However, I know of no previous work that has looked at the emotional effects of alcohol using a behavioral measure that affords the same precision of measurement for emotional reactivity that has been obtained for measurement of cardiac reactivity. My interest in this problem derived from dissatisfaction with using self-report measures of emotional state that have typically been used in this context.

In our laboratory, using standard mood inventories, we had consistently found that intoxicated subjects reported feeling more "cheerful" and more "pleasant" than sober subjects. The problems with the generic use of these kinds of self-report measures are well known and need not be repeated here. In the context of alcohol, however, with its profound effects on the central nervous system, the differences in cognitive and appraisal processes in intoxicated and sober subjects are so profound as to make the use of self-report measures even more suspect. In addition, mood inventories essentially provide only a "basal" or "tonic" retrospective measure.

In studies of reactivity, what is needed is a more continuous, and more fine-grained measurement. At first we tried to obtain this by having subjects rate their tension levels continuously on a joystick-type device. While the results in the

prestressor phase were consistent across studies (i.e., subjects reported feeling less tense at higher doses of alcohol), they were much less consistent during the stressor procedure. Intoxicated subjects often "forgot" to adjust the dial. When they did remember, and when differences were found as a function of alcohol dosage, it was uncertain whether it was their "tension" levels or their "appraisal" facilities that had been altered.

For all of these reasons, and because alterations in subjects' emotional reactivity must be considered as potential explanations for alterations in autonomic and in neuroendocrine reactivity, we began to explore the use of measures of momentary changes in facial expression as a method for understanding the changes in emotional reactivity brought about by alcohol. We chose the face for a number of reasons. Of all the nonverbal emotional systems, its response patterning in different emotional states is best established. It is a very fast response system; emotional facial expressions typically come and go from the face in a matter of seconds. The face is integrated with autonomic functioning in emotion in a number of different ways. Among these are the autonomically-mediated changes in the face such as blushing, blanching, crying, and sweating. The face can be studied unobtrusively from video recordings. And finally, anatomically-based methods now exist for decomposing complex facial expressions into their component muscle contractions, and for relating these facial expressions to discrete emotional states (e.g., Ekman & Friesen's Facial Action Coding System; FACS) (58).

Using FACS we have scored the facial responses to electric shock of 21 women, seven at each of three different doses of alcohol. The process of FACS scoring is very slow, taking about one hour to score one minute of facial behavior from repeated slow motion viewing of video tape. By year's end we hope to have completed scoring the facial behavior of 100 subjects of both genders and under two different kinds of stressful situations. In the meantime, what we have found to date has been quite interesting.

Based on observing the facial responses of many experimental subjects to shock over the years, we have identified three response "windows" that are differentiable. First, there is the anticipation to the shock. This occurs in the last five seconds prior to the shock, while subjects watch the seconds remaining before the shock count down on a digital display. Second, there is the shock itself. Most subjects show an expression which starts about 500 msec after the shock. Third, there is the secondary reaction, which occurs three to five seconds after the shock. In each of these windows we have obtained evidence that alcohol reduces the emotional impact of the stressor.

Anticipation. There is great variability in this window. Some subjects show expressions that signify fear; others show expressions that signify contempt. Some subjects show attempts at controlling emotion that focus around the mouth. These include lip biting, lip tightening (orbicularis oris muscle), pressing the lips together, raising the chin boss (mentalis muscle). Some show "false" smiles, which involve only the mouth and not the eyes and cheeks (59). The effects of alcohol in this window are

seen most clearly in these attempts at emotional control. In Figure 5, alcohol is shown to reduce the overall amount of expression in anticipation of the shock, and in particular, the occurrence of expressions of fear and emotional control. We have interpreted this as signifying that alcohol has made the stressor seem less stressful, and made subjects feel less of a need to try to control their emotional behavior or to put on a "brave front" (i.e., false smiles) for their benefit or that of the experimenter. In terms of the implications of these findings for understanding patterns of cardiovascular arousal, it is important to note that heightened cardiovascular reactivity may be associated both with the arousal of certain negative emotions (e.g., 60) and with attempts to reduce or repress emotional expression (e.g., 61-62).

Insert Figure 5 about here

Shock. In the shock window, most all of our subjects showed some variant of the prototypical facial expression of fear. The most commonly represented element of this expression was the contraction of the risorius muscle, which pulls the lip corners straight back toward the ears. This is a central feature of the facial expressions that signify both fear and pain. Since it was common to all subjects, the intensity of the risorius contraction provided a convenient index of fear intensity. FACS allows scoring the intensity of muscle contraction on a five-point scale. In Figure 6, it can be seen that alcohol reduced the intensity of the risorius contraction, providing some indication

that the shock was less impactful.

Insert Figure 6 about here

Reaction. The reaction window is perhaps the richest in terms of the emergence of individual variation, since there the subject often provides a secondary reaction. This is a kind of emotional "comment", which may reflect some appraisal of what has come before. Subjects are often "surprised" at their reactions and even amused by them. It is also a time to release tension, since, in our experimental procedure, there is no further stress or discomfort after the shock has been administered. Again, alcohol reduced the amount of facial expressive behavior. In Figure 7, alcohol is shown to reduce the occurrence of genuine or "felt" smiling (this is smiling that includes the eyes and cheeks in addition to the mouth; 59). As was the case in the anticipation and shock windows, alcohol reduced the amount of emotional responsivity. Intoxicated subjects accumulated less tension; and thus, they had less tension to release.

Insert Figure 7 about here

These findings provide preliminary evidence that alcohol reduces the emotional reactivity of subjects and/or the emotional impact of stressful events. In the brief, but emotionally charged 10 second period that we have studied, sober subjects have ridden an emotional "roller coaster" from anticipatory fear and dread, to attempts to control their emotional response, to

fear and pain, and to joyful release. Intoxicated subjects have traversed a much "flatter" path with fewer perturbations. As was seen with their cardiovascular systems, alcohol has reduced their emotional reactivity. How much of the reduction in cardiovascular reactivity that we and others have associated with alcohol consumption is secondary to this reduction in emotional reactivity is unknown. It is unlikely that the two kinds of reactivity will ever be completely separable, since the evolutionary connections between the autonomic nervous system and emotion are so strong. But with these emotional data, as with the cardiovascular data, the potential mediational link between moderate alcohol consumption and reduced cardiovascular disease is clear. If alcohol makes the world seem less stressful, and if it makes the individual less emotionally labile, then two kinds of health benefits might ensue. First, if emotional arousal (e.g., anger, hostility) plays a role in the etiology of coronary heart disease, then a beneficial aspect of moderate alcohol consumption may derive from a diminution in these emotional/psychological factors. Second, if cardiovascular and/or endocrine hyperreactivity have etiological significance in the development of coronary heart disease, then a reduction in the emotional fuel for these arousal systems could be highly beneficial.

Concluding Comments

Alcohol use is clearly a significant and complex factor in any consideration of coronary disease and coronary health. Alcohol is a dualistic entity with both a harmful and a helpful side. In this chapter, I have tried to portray both sides of the

alcohol/cardiovascular relation. The injurious effects of alcohol, which contribute to cardiovascular, liver, and a wide range of other diseases, present a very negative case against heavy drinking. If to this evidence are added the other harmful effects of alcohol--the high potential for addiction; the damage to life and property associated with drunken driving; and the damage to mental health, to family functioning, and to society, then there can be little serious consideration given to endorsing the increased use of this drug. However, in looking at the other side of the issue, there is much that is potentially beneficial in terms of cardiovascular health that may be derived from the moderate use of alcohol. Added to this evidence are the other social "benefits" of drinking--the pervasive role that alcohol plays in our ritualized behaviors of courtship and mating, in celebration, in rites of passage, and in professional and business life.

My goal in writing this chapter has been to explore what is known about alcohol and the heart. Still, it is hard to ignore the practical implications of this research. Advocating moderate drinking as a means for increasing cardiovascular health would clearly be premature. We do not know enough about alcohol's effects on the heart, or about the boundaries of time and amount that delineate "health-promoting" drinking. Perhaps even more glaring than our lack of knowledge about drinking, is our lack of knowledge about drinkers. Even if we established conclusively that alcohol could promote cardiovascular health, and even if we knew precisely how much alcohol, consumed for how long, would

have this desirable effect (without producing injury to other bodily systems), we would still need to know a great deal more before encouraging the use of this addictive substance. In particular, we would need to discover methods for managing drinking that would keep today's moderate drinker from becoming tomorrow's alcoholic. Considering how little success the medical and behavioral sciences have had in developing methods for controlling addictive behaviors, it seems best to consider the use of alcohol as an adjunct to cardiovascular health to be an undeniably alluring Siren, but one best avoided.

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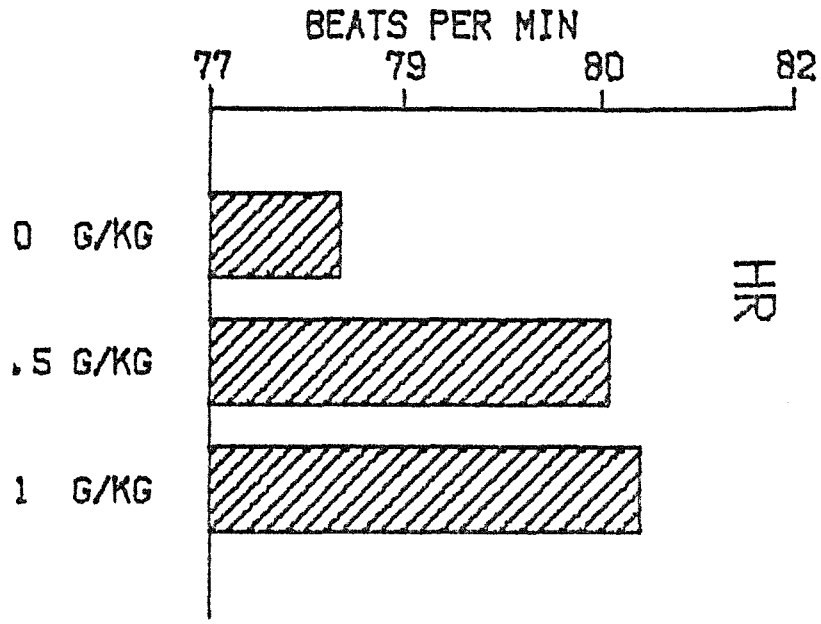
Figure Legends

1. Effects of three doses of alcohol on prestressor cardiovascular levels.
2. Alcohol dampens the heart rate response to stress.
3. Alcohol dampens the pulse transmission time response to stress.
4. Effects of three doses of alcohol on cardiovascular responses to stress.
5. Alcohol reduces emotional reactivity in anticipation of electric shock.
6. Alcohol reduces emotional reactivity to shock.
7. Alcohol reduces emotion reactivity in reaction to shock.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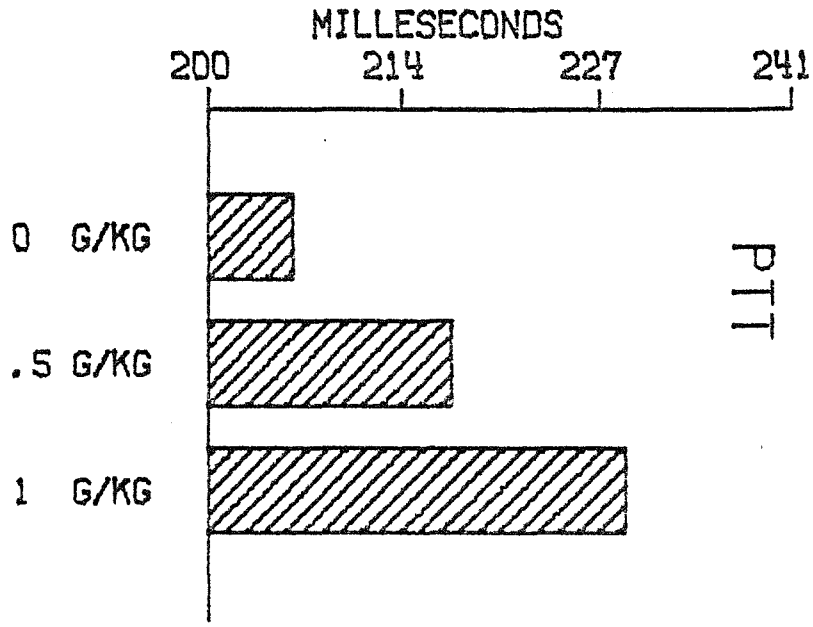
77
79
80
82
0 G/KG
.5 G/KG
1 G/KG
HR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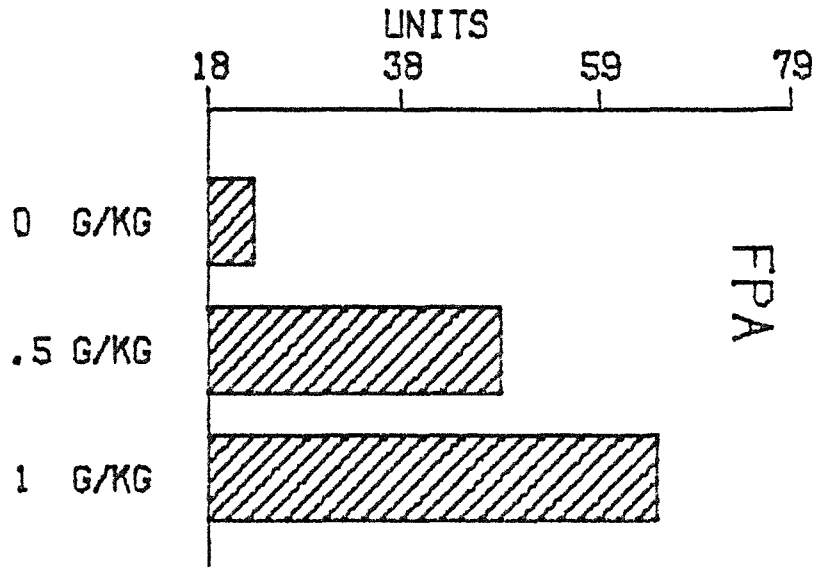
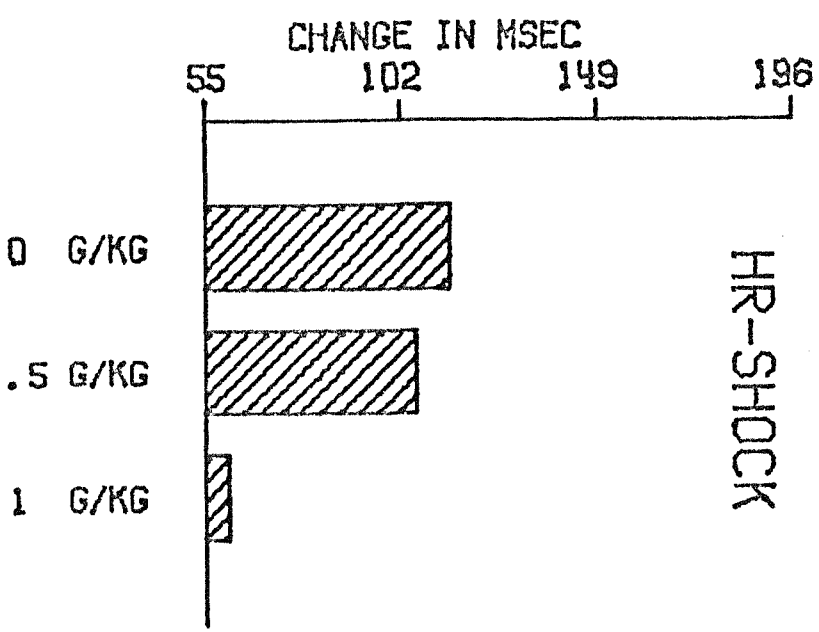
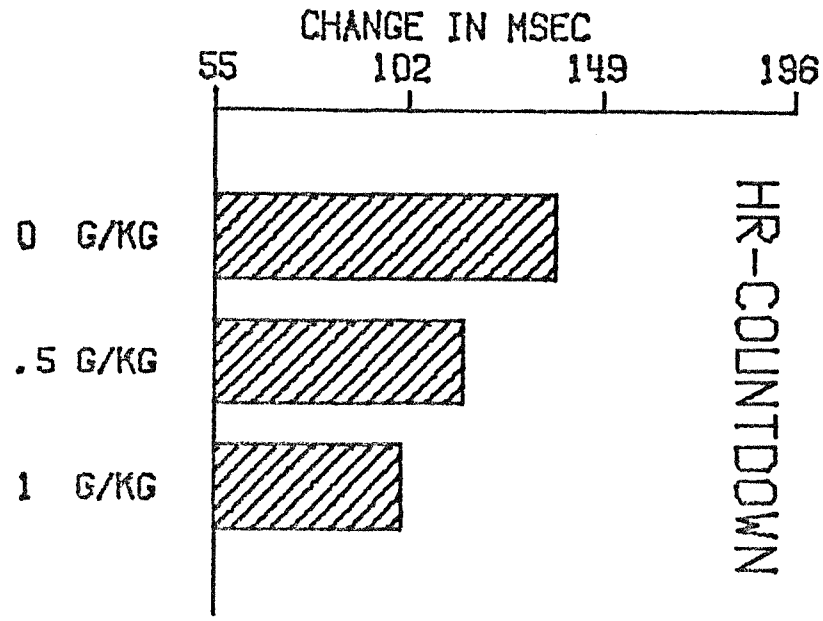
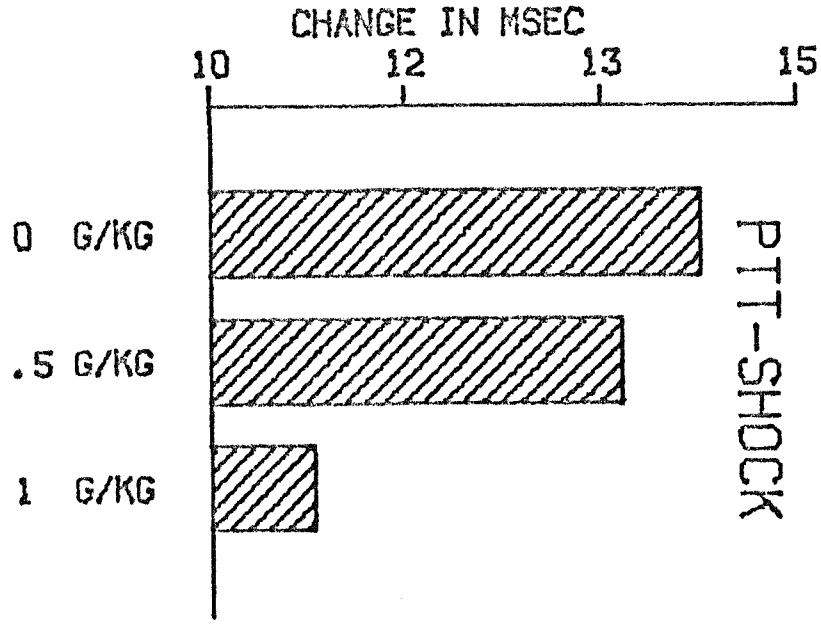
200
214
227
241
0 G/KG
.5 G/KG
1 G/KG
PTT


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


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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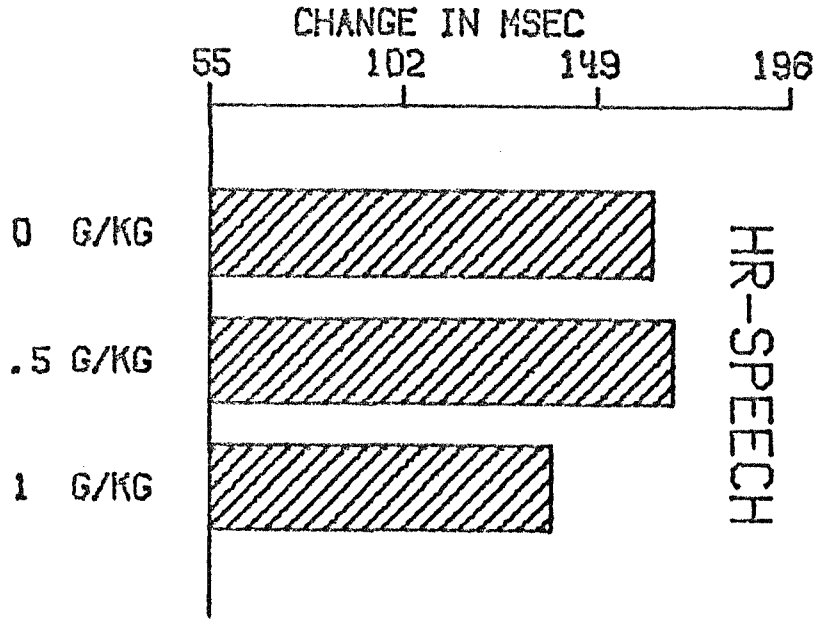
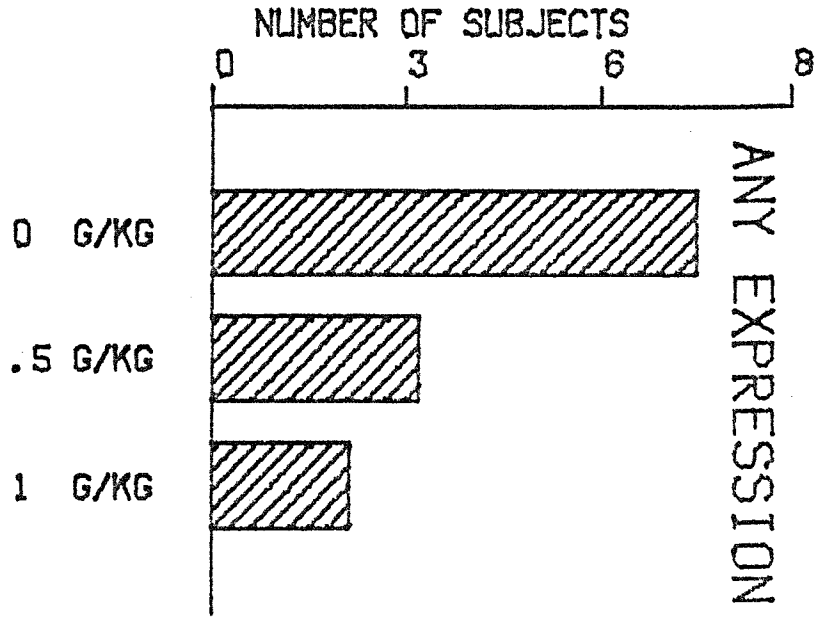
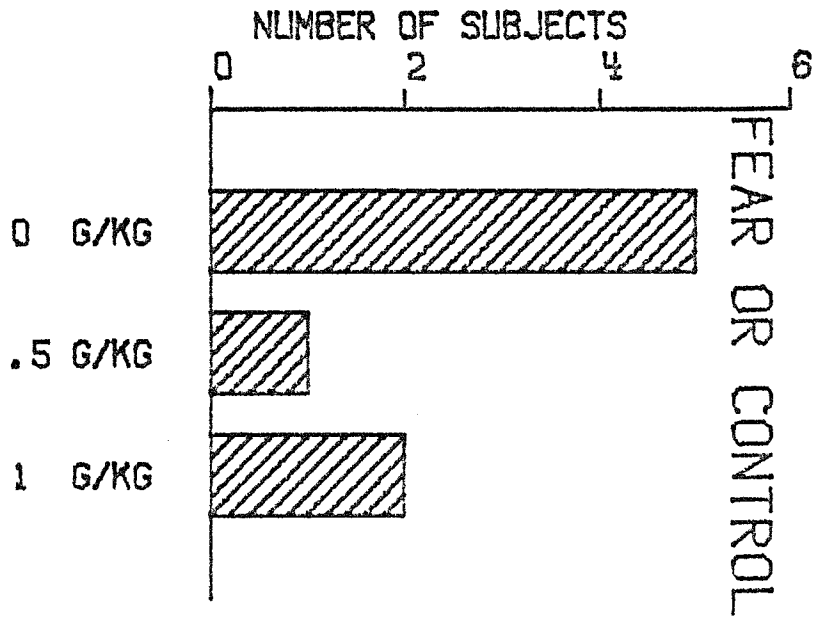
12
13
15
0 G/KG
.5 G/KG
1 G/KG
PTT-SHOCK


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


NUMBER OF SUBJECTS
0
3
6
8
ANY EXPRESSION
0 G/KG
.5 G/KG
1 G/KG


NUMBER OF SUBJECTS
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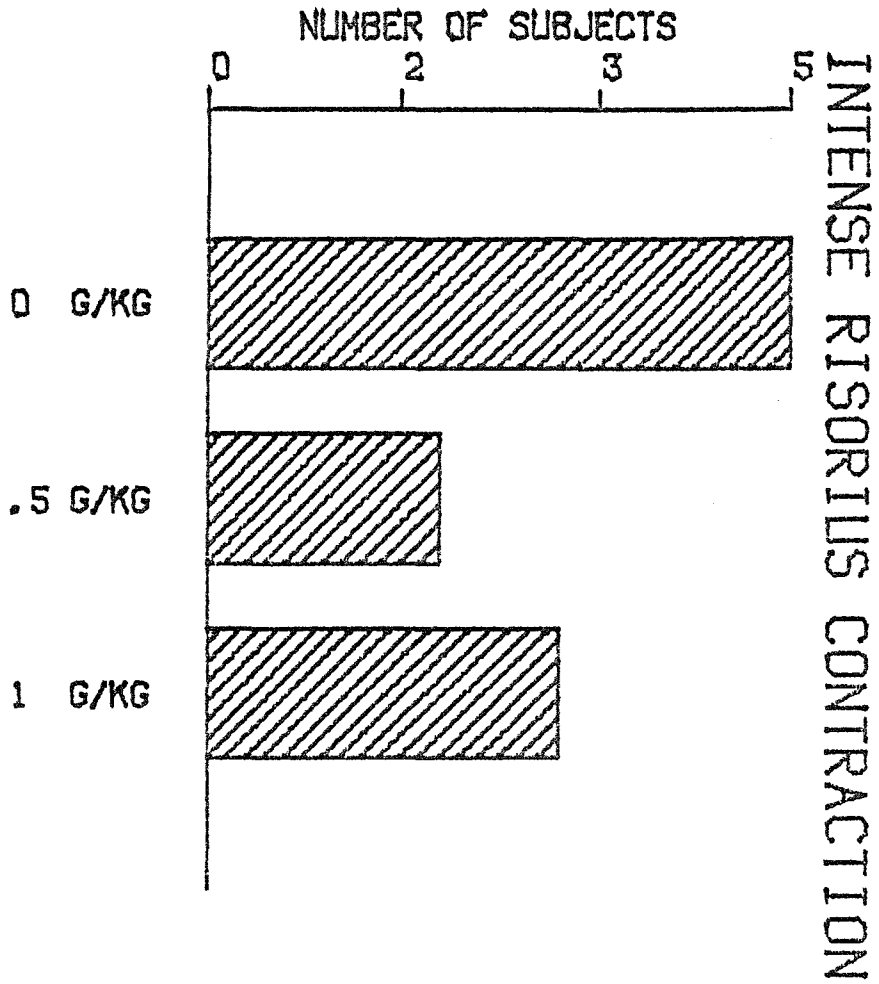
2
4
6
FEAR OR CONTROL
0 G/KG
.5 G/KG
1 G/KG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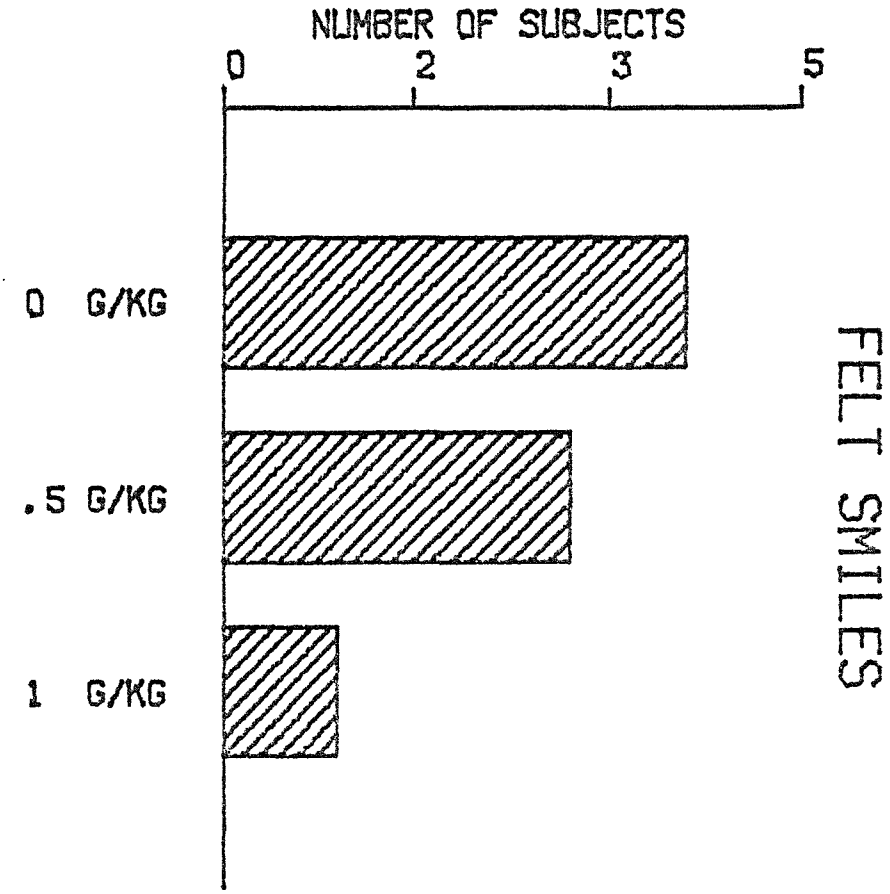
2
3
5
0 G/KG
.5 G/KG
1 G/KG
INTENSE RISORIUS CONTRACTION


NUMBER OF SUBJECTS
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2
3
5
0 G/KG
.5 G/KG
1 G/KG
FELT SMILES