The University of Manitoba

Effects of Stress Predictability and Dietary Physical Properties on Ulceration and Acidity in the Rats's Forestomach

by

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For my father

ABSTRACT

Shock predictability and dietary physical property (liquid or solid) were varied within an inescapable and unavoidable shock situation for 48, 72, or 96 hr in order to assess the effects of these variables on rumenal and glandular ulceration and pH in the stomachs of 168 rats. Subjects in the liquid diet condition had significantly greater ulceration and significantly lower pH (higher acidity) in the rumen than those subjects in the solid diet groups. It appears that dietary physical properties may be as important as chemical and nutritional properties with respect to gastric ulcerogenesis. Shock predictability did not markedly affect rumenal or glandular pH and did not produce glandular ulceration in any subject. Rumenal pH and ulceration increased following 72 hr of conditioning. The results also indicated a strong negative correlation between ulceration and pH in the rumenal portion of the stomach. It appears that the period required for acidity increase accounts for the three to five day latent period in the development of rumenal ulceration. Under all experimental conditions, the rumenal portion of the stomach was consistently more acidic than the glandular portion. Although increased acidity is common to ulceration development in both divisions of the stomach, the present results indicate that a uniform acidity increase may not result from the same treatment. The liquid diet produced increased rumenal acidity but did not change glandular pH. It appears that the presence or absence of increased

acidity is determined by the nature of the stressor employed. The finding that a single stressor increased acidity and produced ulceration in the rumen, but effected no glandular ulceration or pH changes provides support for this hypothesis.

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CHAPTER I

INTRODUCTION

1.1 Review of Psychological Variables in Experimental Ulceration

This section provides a review of psychological variables involved in the production of experimental gastric ulceration. The psychological variables are classified along two dimensions: helpless-control and predictable-unpredictable. The final discussion concerns the role of psychological and physical variables as they relate to the prevention of and the recovery from experimental ulceration.

Ulceration of the stomach and digestive tract occurs among most people in most parts of the world (Gallart-Mones, 1958; Kurokawa, 1958; Pulvertaft, 1958; Watkinson, 1958). Eusterman and Balfour (1935) noted that 10% to 12% of all people at some time in their lives suffer from a gastric or duodenal ulcer as revealed by autopsies and X-ray studies. Despite these facts, less is known about ulceration than many other human diseases (Robbins, 1967). The temporal development of ulcers and the relationship of psychological factors to gastric pathology remain unclear.

On the basis of extensive experimental research and clinical observation, many factors have been shown to contribute to the etiology of gastric ulceration. These factors can be divided into the categories of physical and psychological variables. That a psychological variable by itself can lead to gastric ulceration has not yet been conclusively demonstrated, however much research implicates psychological factors in gastric pathology. Most research has combined a physical parameter

(e.g., shock) with a psychological one (e.g., fear). It is research involving both psychological and physical variables which this review will consider.

The actual beginning of the systematic investigation of psychological factors as they relate to gastrointestinal pathology was provided by Wolf and Wolff (1947) in their classic study of Tom, who had an opening in the stomach (gastric fistula) for forty-seven years. W. B. Cannon, in introducing the study, noted that "perhaps most illuminating of all the observations are those on the intimate relations between emotional states and gastric activity (p. vi)". It is worthy of note that Wolf and Wolff examined the gastric concomitants of spontaneously occurring emotions in Tom. They did not artificially or experimentally attempt to induce emotional situations. Basically, this study correlated gastric reactions with ongoing emotions as labelled by Wolf and Wolff. In the case of aggressiveness and hostility, the authors noted that the gastric mucosa became dark red and engorged with blood (hyperemia), acid secretion increased to three times the normal (control) level and that gastric motility increased. Hostility and anger were associated with the highest acidity level of all emotions considered in the study. Chronic emotional tension or anxiety, was found to result in hyperemia and prolonged increases in gastric motility and acidity. Wolf and Wolff remarked that following chronic emotional tension, the mucosal membrane was more susceptible to trauma than at any other time. This may have been due to the apparently anxiety-induced reduction in the protective action of the mucous membrane. The Wolf and

Wolff study directed the attention of researchers to the relationship between emotional changes and physiological changes. It was evident from the study of Tom that some relationship existed, but its experimental investigation remained incomplete.

Mahl (1949) began this investigation. He noted the confusing nature of the research dealing with psychological processes leading to peptic ulcers. Mahl began his paper by stating "Emotional processes are now generally regarded as playing a primary role in the etiology of peptic ulcers (p. 30)". This statement was followed with a lengthy discussion concerning the lack of agreement on this issue. Most of the disagreement arose from different researchers reporting that widely different emotions all seemed to produce ulcers. This confusion may have been due to the fact that at this time, it was proposed that the term "emotion" be eliminated from the psychological literature (Duffy, 1934; 1941), reflecting disagreement over the meaning of emotion. Duffy felt that the term "emotion" was redundant with those of "activation" and "motivation". Mahl stated, however, that chronic anxiety or fear seemed to be the most substantiated emotion which led to gastrointestinal disorders. He also noted that prolonged increases in gastric acidity led to gastric and duodenal ulcers. He reasoned that if chronic fear and gastric acidity were involved in ulcer etiology, then a relationship between these two variables should be possible to demonstrate. Mahl exposed dogs to a light CS followed randomly by a strong shock. It is important to note here that the shock in this situation was unpredictable and uncontrollable. (The implications of this fact will be discussed later.) Mahl's results confirmed the hypothesis that exposure

to chronic anxiety brings about pathophysiological changes. After up to six months of chronic fear, six out of seven dogs showed increased gastric acidity. Removal of the animals from the experimental chamber or omission of the shock resulted in a return of acidity levels to the normal control level. At no time was ulceration found in any of the animals, but Mahl noted that this finding did not necessarily contradict the acidity theory of ulceration since dogs rarely developed peptic ulcers unless surgically altered.

Mahl's study demonstrated a rise in both free and total acidity in response to chronic fear. These gastric acid increases were elicited by the CS alone, without shock reinforcement, thereby providing support for an association between a psychological variable and gastric acid changes.

Mahl (1953) examined a human patient diagnosed as suffering from chronic anxiety. Intragastric samples were taken from this patient over the course of 32 psychoanalytic sessions, each of one hour duration. Prior to the session, the stomach was washed with physiological saline. The gastric acid samples were collected immediately after each analysis session. The psychological data was independently rated for anxiety and classified as high or low anxious. This data was then correlated with the acidity findings. The results showed that high acidity and low acidity correlated significantly with high anxiety and low anxiety respectively. These results are again inconclusive with respect to the relationship of psychological variable to gastric pathology since no ulceration data could be obtained, and since the patient had evidently been suffering from anxiety for a long time and as such, many uncontrolled non-psychological factors could have been involved.

Heller, Levine and Sohler (1953) investigated the relationship between gastric acidity and "normally produced" anxiety. Anxiety was induced by informing hospital patients of the need for a second test of the stomach contents, leading the patients to believe that their illness was more serious than first thought. The persistence of the elevated gastric acid levels which occur in response to anxiety was examined. Specifically, they studied whether the elevated acidity levels would remain if the anxiety remained for a period of 12 hours. The results showed that elevated gastric acid levels remained as long as did the anxiety. A complicating factor in this study, however, was that the patients used in the study were suffering from gastrointestinal pathology and as such, it is difficult to conclude whether the acidity levels reflected anxiety or organic disease.

Following 1953, the research on psychological factors relating to gastric ulceration can be classified according to a helpless-control dimension and a predictable-unpredictable dimension proposed by Seligman (1968).

Many studies utilizing the procedures of conflict, avoidance, conditioned fear and restraint to produce gastric ulcers, were carried out prior to Seligman's proposed classifications. These concepts are defined, supporting research cited, and then the relevant ulceration literature documented and discussed in relation to these dimensions in the following two sections.

(a) Helpless-Control Dimension

Seligman, Maier and Solomon (1971) offered a definition of controllability in terms of conditional probability. In an instrumental situation, uncontrollability is defined as that situation where a reinforcement

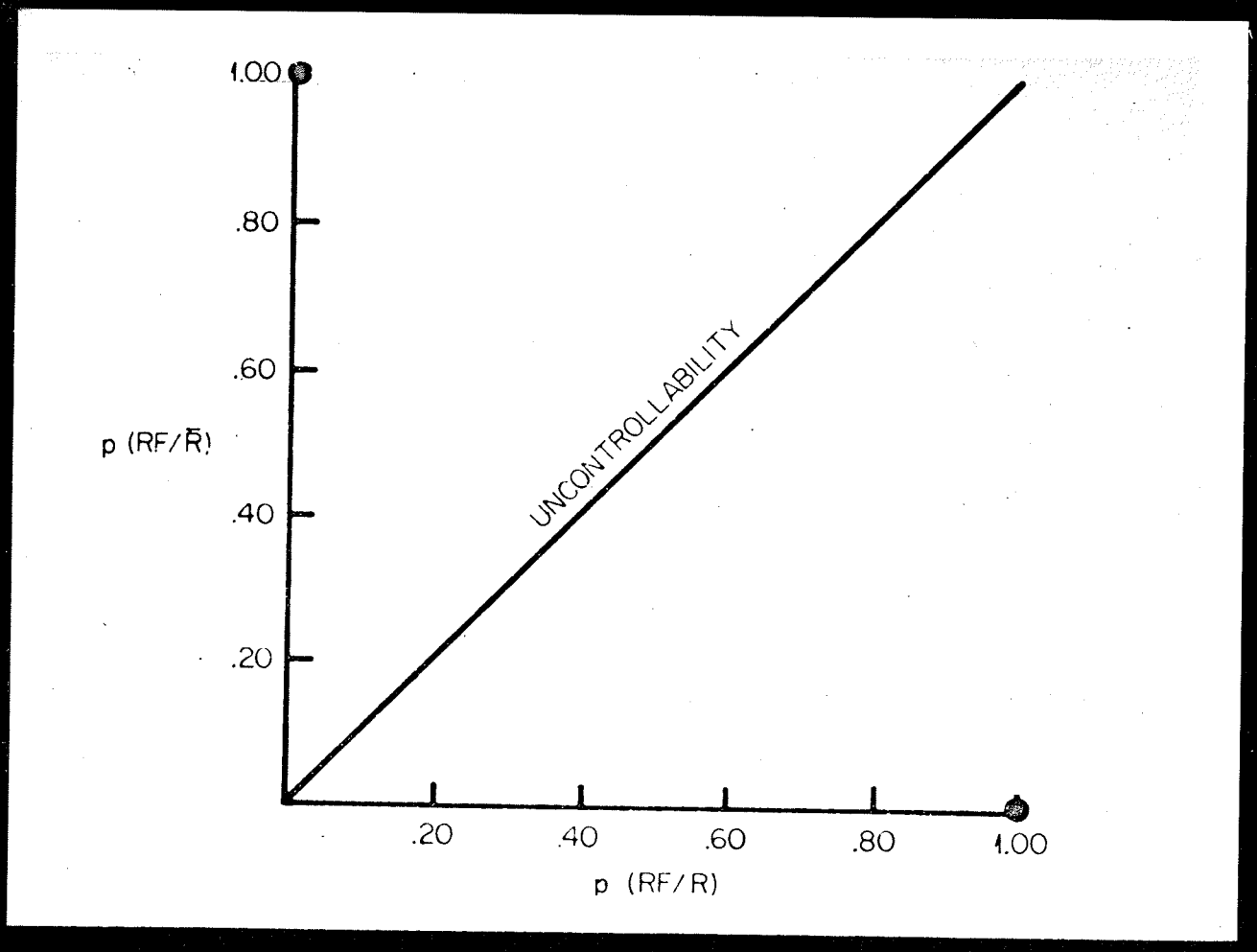
is completely independent of a response; that is, a response does not control or result in reinforcement. Within the classical conditioning paradigm, uncontrollability is defined as that situation where the CS and the US are neither produced, reduced, terminated or prevented by any response the organism emits. Figure 1.1 shows a probability view of control or helplessness within an instrumental framework. Helplessness (uncontrollability) is defined as the situation where the probability of reinforcement given that a response has occurred or p (RF/R), is equal to the probability of a reinforcement given that no response has occurred or p (RF/$\bar{R}$). In other words, the organism is helpless or "not in control" if reinforcement is independent of its responding.

(i) Helplessness

Mowrer and Viek (1948) began the study of helplessness. The authors noted that both clinical and experimental observations suggested that fear aroused by physical pain was a function of whether the pain was under the subject's control Mowrer et al. hypothesized that the fear aroused by physical pain increased over time, while the same stimulation, subject to termination by the organism, resulted in little or no such fear. One group of rats could terminate shock by jumping vertically so that all four feet were off the ground. A second group of animals could not terminate the shock. The results showed that animals which could escape the shock by jumping exhibited fewer anxiety or fear reactions (as measured by inhibition of eating) than those animals which could not terminate the shock. These early results provide support for the helpless end of Seligman's first dimension.

FIGURE 1.1

Seligman's et al. (1971) view of helplessness in terms of conditional probability. When $p(RF/R) = p(RF/\overline{R})$, the organism is helpless.


1.00
.80
.60
.40
.20
p (RF/R̄)
UNCONTROLLABILITY
.20
.40
.60
.80
1.00
p (RF/R)

Seligman and Maier (1967) provided experimental support for the hypothesis of "learned helplessness" resulting from an uncontrollable situation. One group of dogs was trained to press a panel to escape shock, thereby allowing the animals control over the shock. These dogs showed normal acquisition of a subsequent shuttlebox escape/avoidance task. A second group of dogs was first given inescapable shock (absence of control over shock) and then placed into the shuttlebox escape/avoidance situation. They showed a marked deficiency in their ability to acquire the new task, and in fact never did acquire it. These results were interpreted as supporting a learned helplessness explanation of interference with escape responding. Seligman, et al. argued that in the first phase of the study, the subjects had learned that shock termination was independent of responding. When transferred to the shuttlebox situation, the animals failed to respond because they had already learned that responding had no effect on shock administration. These results suggested that the controllability-uncontrollability aspect of an organism's first experience with an aversive situation can profoundly affect the subsequent behaviour of that organism.

Overmier and Seligman (1967) extended the previous findings and found that the use of higher shock intensities in the inescapable situation did not attenuate the interference effects on later avoidance behaviour. They concluded that simple adaptation to shock was not occurring. To eliminate the possibility that a competing instrumental response might be occurring during the period of inescapable shock, dogs were curarized for this phase of the study. The interference with subsequent avoidance behavior was still present after this treatment.

Finally, Seligman, Maier and Geer (1968) showed that the learned helplessness effect could be alleviated by repeatedly compelling the dog to make the instrumental response which terminated the shock. Dogs which had been given prior inescapable shock, were dragged through the opening in the shuttlebox, thus terminating the shock. Eventually the dogs acquired this response on their own. The authors concluded that instrumental control over aversive events is necessary to prevent the development of subsequent abnormal behaviour.

Weiss (1968) indicated that animals in a helpless or uncontrollable situation develop greater pathological damage than do animals in a situation over which they have control. In this experiment, one group of rats was trained to escape and avoid shocks by jumping onto a platform. A second group of rats was trained to escape and avoid shock by pressing a copper plate with their noses. Two other groups were yoked to the escape/avoidance groups; that is, they were shocked whenever their counterparts failed to avoid shock. The results indicated that yoked subjects, who were helpless since their responding was independent of shock administration, lost more weight, developed more and larger stomach ulcers, defecated more and showed greater inhibition of drinking in the shock situation than did subjects which could escape and avoid shock (the subjects which had control over the situation). Similar to Weiss's findings were those of Moot, Cebulla and Crabtree (1970) who varied the control-helpless dimension in their experiment by allowing rats instrumental control over shock. Rats were trained to lever press for food on a variable interval schedule during which each reinforcement was paired with electric shock. One group of

animals terminated the shock by responding while a second group was helpless and could not avoid the shock. The results showed that animals which could escape shock developed significantly fewer ulcers than animals receiving inescapable shock. This study provides support for Seligman's helpless-control dimension by illustrating that the degreee of instrumental control an organism exerts over a moxious stimulus, is an important variable in the psychological and physiological severity of a conflict or fear-producing situation.

It is evident that many conditioned fear situations represent helplessness on Seligman's helpless-control dimension. Much research on experimental ulceration has utilized the conditioned fear paradigm. As noted earlier, Mahl (1949) used an uncontrollable shock situation, since shock administration was independent of the subject's responses. Thus Mahl's findings of increased gastric acidity as a function of chronic fear may, in part, be due to the fact that the subjects were helpless with respect to control over shock.

Weisz (1957) used a conditioned fear paradigm, subjecting rats to 30 days of uncontrollable shock. These animals developed significantly more rumenal ulceration than did control subjects. Since the animals were on either a 46 hour or 47 hour food deprivation schedule, and since Mikhail (1966; 1972) has shown that food deprivation per se is an important ulcerogenic agent, it may be that Weisz's results were, in part, due to the food deprivation variable.

Sawrey (1961) attempted to isolate a psychological component from

the physical variables involved in gastric ulcerogenesis. Specifically, Sawrey attempted to separate fear from shock in a conditioned fear paradigm. One group of rats received a buzzer CS followed 100% of the time by shock. The second group of rats received a buzzer CS followed 50% of the time by shock. The animals were maintained on a 20 hour food and water deprivation schedule while remaining in the conditioning apparatus for 14 days. The results showed that the regular group in which the buzzer CS was always followed by shock developed significantly fewer ulcers than the irregular group in which there was no 100% predictable CS. It should be noted that one group of rats received "predictable" shock while the second group received "unpredictable" shock. Subsequent research, to be discussed later, has shown this to be a potent variable in the production of experimental ulceration (Weiss, 1968; Caul, Buchanan and Hays, 1972). This fact, coupled with the physical parameters of food deprivation and shock (since the rats did get 50% of the shocks) may have contributed to Sawrey's results.

Sawrey and Sawrey (1964) administered varying amounts of fear conditioning to rats prior to placing them into restraint for 48 hours. While in restraint, the CS for fear was periodically presented to the rats but in no instance was the US ever presented. The results indicated that ulceration rate increased as a direct function of the amount of prior fear conditioning. This study, however, only demonstrates the effects of a psychological variable when used in conjunction with a powerful physical variable since restraint has been shown to induce a very high ulcer incidence by itself (Ader, 1964; Brodie, 1962; Brodie and Hanson, 1960; Hanson, 1963; Senay and Levine, 1967; Caul and Buchanan, 1972).

Pare (1968) exposed rats to unavoidable and uncontrollable tone-shock pairings for either one hour, 24 hours, five days or 24 days. No ulceration was found in any animal at any time period. These results are readily explained by the fact that the rats in this study, were never food or water deprived. Food deprivation must be used in conjunction with other manipulations if experimental ulceration is to be produced (Weisz, 1957; Sawrey and Weisz, 1956; Mikhail, 1966).

Sawrey and Sawrey (1968) examined the ulcerogenic properties of a shock US in an uncontrollable fear conditioning paradigm. Restrained rats were given various US intensities and durations over a 48 hour period. Sawrey and Sawrey concluded that ulceration increased as a function of US intensity, but not of US duration. These results, however, must be qualified in light of the previously cited literature demonstrating the ulcerogenic properties of restraint by itself.

An opposite finding was reported by Mikhail (1969). Using an uncontrollable fear conditioning paradigm as the other researchers had done, Mikhail found that ulceration did not develop even when conditioned fear was superimposed upon ulcerated rats following 24 hours of restraint. In a second experiment, 20 hours of fear conditioning and 48 hours of food deprivation preceeded 18 hours of pylorous ligation stress. Measures of free and total gastric acidity revealed no differences between rats given the fear CS and those not given the CS during the period of pylorous ligation. Mikhail accounted for these results by stating that the conditioned fear treatment was not ulcerogenic even when mild shock was used. As in other studies using conditioned fear, this experiment did not demonstrate the

ulcerogenic properties of a psychological variable alone since the pylorous ligation procedure was used in conjunction with the fear treatment. Some support, however, was demonstrated for pathogenic changes to the non-reinforced CS's.

It can be seen that from the helpless end of Seligman's continuum, some confusion exists. Nevertheless, Seligman (1971) concluded:

> "Therefore the dimension of controllability affects the resultant emotional upset and physiological stress, as well as the subsequent acquisition of instrumental responses. Shocks which S cannot modify are more distressing and stressful than shocks with which S can cope, even though the physical stimuli are the same (p. 362)."

(ii) Control

The other end of Seligman's continuum is the control end, meaning than an animal's responses affect the occurrence of some subsequent event. In other words, the animal's behavior affords it control over the situation. The best and most extensively studied paradigm illustrative of control is the free-operant (Sidman) avoidance situation (Sidman, 1953). In a typical Sidman avoidance paradigm, the animal must emit a response, usually a lever press, at least once every 20 seconds in order to postpone a shock for a further 20 seconds.

The classic experiment using this paradigm was the "executive" monkey study of Brady (1958). In this study, monkeys were restrained and tested in pairs. Both members of a pair received shocks but only one monkey could prevent them. The "executive" monkey's lever was functional and its responses prevented or allowed shocks for both members of a pair. The other monkey was a yoked control meaning that its lever was disconnected so that

shock was independent of its responding. This monkey received the same number of shocks at the same time as did the "executive" monkey but only the "executive" monkey had the additional psychological stress of responding imposed upon it. Brady's results showed that "executive" monkeys developed severe ulceration and often died while control monkeys showed no signs of gastrointestinal pathology. These results are opposed to those of Weiss (1968) and to Seligman's hypothesis. The "executive" monkey had control over the situation and should have suffered less, if any damage, while the helpless yoked control monkey should have developed ulcers. In addition to the small number of subjects used, there is a confounding variable in Brady's study which may explain the discrepant results. Subjects were not randomly assigned to groups; that is, to the executive or control group. They were assigned to groups on the basis of a pre-test for the rapidity of acquisition of the avoidance task. Those monkeys which acquired the task rapidly were assigned to the "executive" group while slower monkeys were designated as controls. Sines, Clelland and Adkins (1963) and Lepanto, Moroney and Zenhausern (1965) have shown that rats which are susceptible to ulcers acquire an avoidance response faster than controls. Therefore, it may be that the "executive" monkeys in Brady's study were constitutionally more emotional and prone to ulcers than were the yoked control monkeys.

Foltz and Millett (1964) replicated Brady's "executive" monkey experiment with 20 subjects and failed to find ulceration in any of them. This experiment was similar to Brady's in that avoidance sessions alternated with rest sessions and each of these sessions was of six hours duration. The only difference between this study and Brady's was that Foltz and Millett

eliminated the pre-test selection task for the rapidity of acquisition of avoidance responding. The avoidance sessions were signalled by the presence of a red light while rest sessions were indicated by the absence of the light. According to Seligman, this situation is both controllable and predictable for the "executive" monkey. As such, it should have developed less physiological damage than the yoked control monkeys whose situation was helpless. The data followed this pattern. There was evidence of ulceration in control monkeys but none was found in the "executives". It thus appears that helplessness results in more physiological damage than when the subject has control over the situation. That these helpless monkeys were more emotional and anxious than the "executives" was verified by the authors who described the behaviour of one control monkey.

> "There was evidence of striking psychomotor responses each time the red light came on, in that this monkey would stand up in its chair and vocalize and gesticulate towards the "executive", apparently showing an emotional response to the "on" epoch and the "executive's" inability to learn fast enough to suit the control (p. 449)."

The human analog of the "executive" monkey experiment was investigated by Davis and Berry (1963). Using a noise avoidance task, subjects were run in pairs consisting of one executive who could respond to avoid the noise and one control whose responses were ineffectual. External abdominal electrodes revealed greater gastrointestinal activity in the executive subjects, thus supporting Brady's (1958) research since increased gastrointestinal motility has been correlated with gastric pathology (Ganong, 1971).

Brady (1963) commented on the Davis and Berry experiment noting that in his research, the typical finding was that of decreased acidity and gastric motility during the "on" sessions while both of these measures rose sharply during the "off" or rest periods. He explained Davis and Berry's conflicting results by stating that in the "normal" physiological condition, gastric acidity and motility covary in the same direction but while under the stress of a behavioural avoidance task, this relationship might not hold.

Rice (1963) examined the apparently crucial variable of the "on-off" schedule of responding. The responding-rest ratio was increased from 1:1 to 12:12 in increments of one hour. The schedules used were even hour ratios of responding and non-responding time; that is, 1:1, 2:2, 3:3, ..., 11:11, and 12:12. The results showed that a ratio of 6:6 (as used by Brady) resulted in the greatest ulceration development in rats. Brady had found that with monkeys, ulceration developed only when a responding-rest ratio of 6:6 was used while Rice found that all schedules produced some ulceration with the peak at a 6:6 schedule. Rice concluded that his results generally supported Brady, attributing discrepancies to species differences.

Rice's research, however, was not supported by Pare (1971). Pare used a six hour "on" - six hour "off" schedule of avoidance responding in rats. Very few ulcers were found in either "executive" or yoked-control subjects and no differences were evident between these two groups. Hickey (1962) had found similar results. In both of these studies, free food and water intake was monitored and found to be relatively stable. It is possible that these results may have been influenced by the availability of food. It was noted previously that food deprivation appears to be a necessary

condition for the experimental production of ulcers (Mikhail, 1966).

Continuing with research on the gastrointestinal effects of avoidance responding, White (1964) examined human gastric activity during a shock avoidance task and during a condition of passive acceptance of a mild light or tone stimulus. Results indicated that gastrointestinal activity decreased during avoidance behaviour but rose during rest or passive stimulation periods. The results were seen as supporting Brady (1958) but not Davis and Berry (1963).

Pare (1972) examined gastric acidity in rats at each end of Seligman's helpless-control dimension. In one experiment, gastric acidity was measured in an unavoidable or helpless situation. Rats, fitted with a chronic gastric fistula, were subjected to 23 hours of continuous conditioned fear. The results indicated that both free and total stomach acidity increased over the entire session, while total volume of gastric juice declined. No ulceration data was reported. In a second experiment, rats with chronic gastric fistulae were subjected to an avoidable shock situation representing the other extreme of Seligman's continuum. Pare used a one hour "on" - one hour "off" schedule for three hours per day for six days. The schedule on a given day was: one hour rest, one hour avoidance, then one hour rest. Gastric acid samples were collected each hour as in the previous study. The results revealed an acid secretion pattern very similar to that found in the helpless situation. Free and total stomach acidity increased and total gastric volume decreased over sessions and days. Pare's results on gastric activity cannot be extended to gastric pathology since the stomachs were not examined for ulceration. His finding of no differences in gastric activity

(as indicated by acid output) between the helpless and control ends of Seligman's continuum may have been influenced by the fact that three days separated each acid collection session and that during these days, ad lib food and water was available to the rats. The presence of food in the stomach tends to buffer gastric acidity (Levine and Senay, 1970) and this fact may have masked any differences between Pares groups.

The inconsistent literature on the relationship of avoidance responding to ulceration reflects the fact that several confounding factors are involved. Rice (1963) indicated that the stress-rest ratio was important especially with respect to species differences. Brady (1963) noted that different autonomic control mechanisms may be operating to alter gastric acidity and motility in the same or different directions. It is well known that wide individual differences exist in autonomic reactivity (Sternbach, 1966). Hodges and Spielberger (1966) considered the problem of individual differences with human subjects and stated: "Despite the growing concensus concerning the importance of cognitive factors in mediating physiological responses to stress, many investigators continue to ignore individual differences in the subject's interpretation of the stressor situation (p. 292)." This hypothesis may well explain the confusing results on human avoidance behaviour and gastric function. Finally, the control-helpless dimension is common to all the avoidance studies, and should be taken into consideration when evaluating them. The main conclusion arising from Brady's research and other studies which support it is that the variable of having to respond to avoid shock was responsible for the ulceration. Seligman's main conclusion is that the psychological variable of helplessness is an important

ulcerogenic agent. In both cases, the evidence suggests that psychological and emotional factors are important contributors to gastric pathology but they have not been shown to be sufficient ulcerogenic agents by themselves.

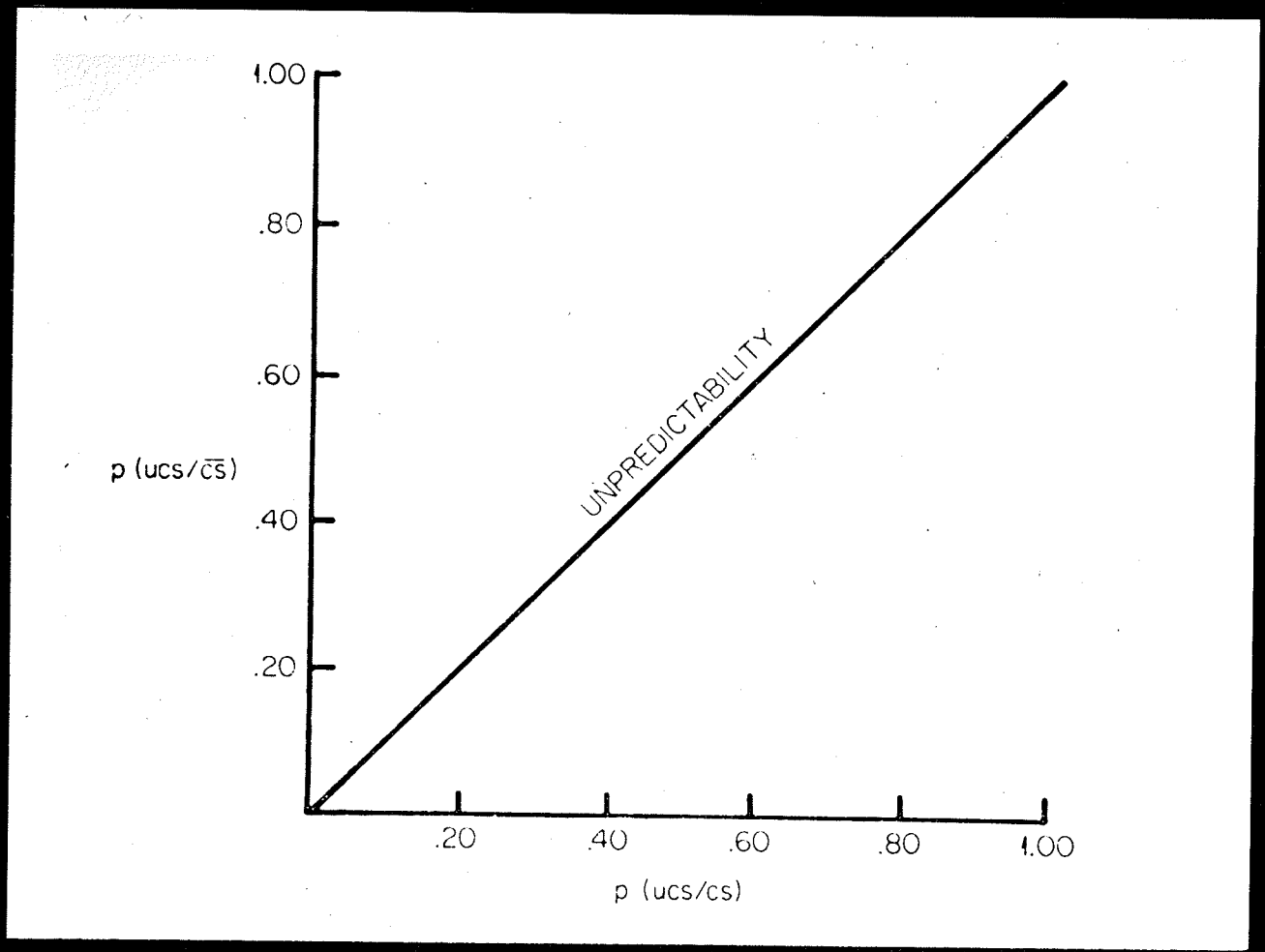
(b) Predictable-Unpredictable Dimension

Seligman et al. (1971) have proposed a second dimension along which studies using psychological variables to produce gastric ulceration may be classified. Predictability, in a classical conditioning paradigm, is defined as that situation wherein the occurrence of a CS changes the probability of a US occurring; that is, a CS stands in a predictive relationship to a US if and only if the probability of a US given that a CS has occurred or p (UCS/CS) is not equal to the probability of a US given that a CS has not occurred or p (UCS/$\overline{\text{CS}}$). If these probabilities are equal, so that whether a CS occurs does not affect the probability of occurrence of US then the situation is defined as being unpredictable. Figure 1.2 shows a diagram of the predictable-unpredictable dimension in terms of conditional probability. Seligman et al. stated that shocks which occurred independently of CS's, so that the shocks were unpredictable, were more aversive and stressful than were predictable shocks. A primary measurement of aversiveness is choice and the following review of research indicates that both animals and humans prefer predictable to unpredictable aversive situations.

Lockard (1963) used a shuttlebox paradigm to study rats' preference for signalled or unsignalled shock. One group of rats was given a light CS five seconds prior to being shocked in one compartment only. A

FIGURE 1.2

Seligman's *et al.* (1971) view of unpredictability in terms of conditional probability. When a p (UCS/CS) = p (UCS/$\overline{CS}$), the situation is unpredictable.


1.00
.80
.60
.40
.20
p (ucs/$\overline{cs}$)
UNPREDICTABILITY
.20
.40
.60
.80
1.00
p (ucs/cs)

second group received random CS presentations between shocks. The dependent measure was simply the number of trials spent in each compartment by both groups of rats. The results indicated that the experimental subjects spent 90% of the trials in the compartment with the CS while the control animals spent about 50% of the trials in each of the two compartments. Lockard concluded that a warning stimulus such as a CS prior to shock, may not be aversive to an animal but may even be reinforcing for its informational or predictive value.

Preference for predictable shock was studied in a different paradigm by Perkins, Levis and Seymann (1963). These researchers placed rats in a tilt box apparatus for eleven hours on each of three consecutive days. In one side of the tilt box, rats received a three second light CS followed by a brief shock (signal-shock) while conditions were reversed on the other side of the box (shock-signal). Following the first three days, the conditions in each side of the box were reversed, and the rats were tested for three more days. Preference was measured by the number of trials spent in each compartment of the tilt box. The results showed that rats reliably preferred the signal-shock condition on the first three days and showed a non-significant trend to prefer the signal-shock side on the following three days when conditions were reversed.

Pervin (1963) investigated human preference for predictable or unpredictable shock. He studied two situations of certainty or uncertainty under conditions of threat of shock. The relative desirability of the threat conditions and their relationship to anxiety was also examined. One group of university students was assigned to a condition whereby the experimenter

controlled unavoidable shock administration to them. A second group of students was allowed to control the unavoidable shock themselves. Questionnaire data revealed that subjects preferred to control the shock themselves, thereby making the shock predictable, and that they found this situation less anxiety-arousing than when the experimenter controlled the shock. The authors concluded that this data supported the hypothesis that predictability is preferable to and less anxiety-arousing than unpredictability. One other interesting result of this study deserves mention. It was found that over time, predictability became more anxiety-arousing and undesirable while unpredictability became more desirable and less anxiety-arousing. The author hypothesized that complete predictability is more desirable in a novel situation where there is more threat while some degree of uncertainty is desirable in repetitive and less threatening situations. Epstein (1973) has adequately summarized the situation of predictability-unpredictability (expectancy) with humans.

> "Expectancy is a double-edged sword. As an individual expands his awareness of potential danger, he is made more anxious, but, by doing so, he becomes less susceptible to being taken by surprise and overwhelmed by anxiety (p. 1)."

Lockard (1965) studied preference for signalled or unsignalled shock in an unavoidable (helpless) shock situation. Again using a two-compartment shuttlebox, Lockard gave rats the choice of a five second, one-half second, zero second or random warning signal prior to inescapable shock. The results indicated that rats preferred a five second or one-half second situation to a random or no CS situation. This study demonstrates further support for the hypothesis that rats prefer predictable aversive

stimulation in situations where such stimulation is unavoidable.

One final human study lends further credence to this hypothesis. Badia, Suter and Lewis (1967) examined whether organisms "prefer information about an aversive event that is uncertain, inescapable and unavoidable (p. 271)." This, and the other studies concerned with preference for predictable aversive situation, are based on the theory that warning signals (CS's) make the environment more predictable and allow the organism to make some prepartory response to minimize the impending aversive stimulation (Perkins, 1955). Badia's et al. results were consistent with the other research in this area (Glass, Singer and Friedman, 1969; Glass, Riem and Singer, 1971) in that most subjects preferred signalled to unsignalled shock.

The hypothesis that organisms reliably prefer signalled shock has been well documented. The reasons for this preference involve investigations of the behavioural and physiological effects of unpredictable shocks. The research indicates that this may well be the basis for the strong preference effect.

Azrin (1956) examined the effects of predictable or unpredictable shock on consummatory behaviour in the pigeon. Pigeons that had been trained to peck a key for food were shocked either in a predictable sequence (shock delivered on an FI schedule) or in an unpredictable sequence (shock delivered on a VI schedule). The dependent measure was suppression of key-pecking for food within each of these shock schedules. The results showed that VI shocks resulted in greater behavioural suppression than did FI shocks. These results demonstrate that unpredictable shocks results in more fear than did predictable shocks. That suppression of appetitive responding is an index of fear has

been well supported (Brady and Hunt, 1955; Sterritt, 1962; Sterritt and Shemberg, 1963). Brimer and Kamin (1963) reported similar results using rats as subjects and suppression of bar-pressing for food as an index of fear.

Measures of gastric ulceration indicate that unpredictable shock results in greater pathological damage than predictable shock. As mentioned earlier, Sawrey (1961) administered buzzer and light CS's to two groups of rats. For one group, the light was always followed by shock. For the second group, half the light presentations and half the shock presentations were followed by shock. These groups can be seen as receiving predictable and unpredictable shock respectively. The results confirmed the hypothesis, showing that the unpredictable group developed significantly more ulcers than the predictable group. Sawrey concluded:

> "It is possible that 'predictability' versus 'non-predictability' is the important variable (p. 348)."

More recently, Weiss (1968) administered signalled or unsignalled shock to rats through fixed tail electrodes (Weiss, 1967), for a period of 19 hours. The results provided a striking demonstration of the potency of the predictable-unpredictable variable. The unpredictable shock group developed a mean of 6.6 ulcers with a group incidence of 100%; the predictable shock group developed a mean of 1.2 ulcers with an incidence of 67% in this group; while a no shock control group showed a mean of .4 ulcers with a 25% ulcer incidence. The lesions in the no shock group were presumably due to the 19 hours of restraint. Although Weiss's results may have been influenced by the variable of restraint (as indicated by some ulcer

development in the control group) or by the variable of shock (3.5 milliamperes for 2 seconds), he concluded that:

> "the results of these experiments indicate that conceptions of stress must not underestimate the importance of psychological variables, since such variables can be even more important than the presence or absence of the physical stressor (p. 264)."

The present writer feels that this conclusion should be qualified to state that psychological variables do contribute to stress disorders but to date, this has only been tentatively demonstrated. Weiss's conclusion seems premature especially in that his data was probably influenced by restraint, certainly an ulcerogenic variable by itself (Caul and Buchanan, 1972) and by the very high shock intensity used, again having been shown to contribute significantly to ulceration (Sawrey and Sawrey, 1968; Sawrey, 1961).

Similar results were obtained by Seligman (1968) using response suppression as well as ulceration as indices of the effects of unpredictable shock. Seligman's data revealed that the unpredictable group of rats developed a mean of 9.1 ulcers with an incidence of 75% while no rats in the predictable shock group developed ulcers. The behavioural data revealed that response suppression was greater in the unpredictably shocked animals in all phases of the study. Seligman proposed a "safety-signal" hypothesis to account for the adverse effects of unpredictable shock. He stated that when a given stimulus reliably signals or predicts shock, then the absence of this stimulus predicts with the same reliability, the absence of shock, resulting in a safe situation. This hypothesis assumes that the organism learns which stimuli come to elicit fear - not just the whole experimental chamber but

the specific CS for shock. In the absence of this stimulus, fear is inhibited and normal appetitive responding will occur, while in its presence, fear will be present and response suppression occurs. When, however, there is no reliable signal for shock, there can be no reliable signal for safety. Therefore, the animal has no predictable "safe" period and thus remains in chronic fear. If it is assumed, as Seligman did, that suppression of appetitive responding and the presence of stomach ulcers are indices of fear, then the safety-signal hypothesis appears tenable at the present time.

Seligman and Meyer (1970) provided further support for the safety-signal hypothesis. In this study the persistence of the response suppression effect was examined. In addition, the variable of shock intensity was investigated in order to test for the existence of a correlation between degree of fear produced and amount of stomach pathology. Rats were divided into the following groups: High shock-predictable; low shock-predictable, high shock-unpredictable and low shock-unpredictable. All subjects were tested for 50 minutes per day for 70 consecutive days. With respect to the first question, the results showed that groups of rats receiving unpredictable shock suppressed their response rates and never recovered to more than 30% of their pre-shock rate. Seligman et al. concluded that "chronic fear is not transitory when safety is truly unpredictable (p. 302)." The relationship between the behavioural measure of fear (response suppression) and the physiological measure of fear (gastric pathology) revealed a high degree of association. Gastric pathology was divided into four categories: stomach ulcers, rough mucosal erosions without visible clotting, intestinal ulcers, and total gastric pathology (resulting from the sum of the first three categories). Significant

correlations were found for all four classes of physiological damage. The correlations between total gastric damage and response suppression was .74, thereby accounting for over 50% of the variance in this dependent measure. This can be considered a highly significant result (Vaughan and Corballis, 1969). Seligman et al. concluded that "a hypothesis which holds that an organism's fear in a dangerous situation can be determined by predictability of safety was confirmed by both behavioural and physiological indexes of fear (p. 207)."

Weiss (1970) presented a lengthy series of studies concerned with the effects of predictable and unpredictable shock, a preliminary report of which was previously discussed (Weiss, 1968). The critical point of this series of studies is that Weiss held the physical conditions constant (shock, restraint etc.) but varied the psychological conditions (predictability) and in this way, produced differences in physiological stress reactions. Weiss noted that the conflicting results in the area of predictable or unpredictable shock may have been due to the fact that the studies used a grid floor to deliver shock to the animals. He noted that with a grid floor, the rat can respond to various ways to reduce the intensity of the shock and can even avoid the shock by jumping off the grid. Thus a fixed tail electrode (Weiss, 1967) was used to deliver shock in these studies. With this method, any differences in the amount of shock received by each rat were eliminated. As noted in Weiss's 1968 study, rats receiving unpredictable and inescapable shock developed much more ulceration than rats receiving either predictable shock or no shock. In this study, Weiss also measured body temperature changes and plasma corticosteroid levels, an indicant of adrenal activity

and a primary stress response (Selye, 1950). The results again indicated that the unpredictably shocked rats had significantly elevated body temperatures and plasma corticosterone levels, both indicating that the stress of these animals was greater than that of the predictable group. Two further experiments in this series by Weiss revealed that unpredictably shocked rats lost significantly more body weight than rats receiving either predictable shock or no shock. This result was found in both restrained and free-moving rats, negating the role of restraint in contributing to the weight loss. Weiss concluded that the effects of the same physical stressor can be altered by psychological variables such as predictability. He also stated that certain stress responses, for example stomach ulceration, may be more affected by psychological than by physical parameters since in his studies, the predictably shocked rats differed very little from the non-shocked subjects.

A recent study by Caul, Buchanan and Hays (1972) confirmed Weiss's results. Ulceration was most severe in rats receiving unpredictable shock. A further physiological measure used in this study, that of heart-rate, failed to differentiate between predictably and unpredictably shocked rats. Based on their ulceration data, however, the authors concluded that their results demonstrated the "potency of unpredictability as a stressor (p. 671)."

In a series of three studies, Weiss (1971a; 1971b; 1971c) combined the helpless-control and the predictable-unpredictable dimensions and examined their effects on gastric pathology in the rat. Out of these experiments arose a theory to account for the data which indicates that

animals which have control over noxious stimulation develop less ulceration than do helpless animals. The outline of Weiss's first study (Weiss, 1971a) is shown in Table 1.1. Predictability was varied by presenting rats with either a beep (signal group), a series of tones increasing in intensity followed by a beep (progressive signal group), or no tone or beep (no signal group) prior to shock. Control was varied as in a previous study (Weiss, 1968) by having an avoidance-escape group which could respond to avoid shock, a helpless yoked group which received every shock that the avoidance-escape group received but which could not respond to avoid it, and a non-shocked control group. The results showed that within all three signal conditions, the avoidance-escape rats (control over the situation) developed less ulceration than the yoked rats (helpless), thus providing further support for Seligman's helpless-control hypothesis. Within warning signal conditions, it was found that avoidance-escape subjects developed more ulceration in the no signal (unpredictable) condition than in either the signal or progressive signal (predictable) conditions. A similar pattern of results occurred for the yoked (helpless) animals as well. Thus, support for the predictable-unpredictable dimension was also demonstrated. Weiss concluded his discussion of these results by stating that:

> "the psychological characteristics of the stressful situation - the predictability, avoidability, and escapability of shock - primarily determined how pathological the stress situation was, not whether the animal was exposed to the stressor (Weiss, 1968, p. 8)."

This conclusion is not inconsistent with that of Lazarus (1966) who, after

TABLE 1.1

OUTLINE OF EXPERIMENTAL TREATMENTS AND THE NUMBER OF SUBJECTS PER GROUP IN WEISS'S 1971a STUDY

Condition/Group	Avoidance-Escape	Yoked	Non-Shock
Signal	20	20	20
Progressive Signal	20	20	20
No Signal	20	20	20

reviewing the literature dealing with the control-helpless dimension in human subjects, stated that "predictable punishment is less threatening than predictable punishment. In the former the individual knows when to expect the harm even if he can do little about it. Such expectation affords a modicum of psychological control over the situation (p. 100)." Weiss (1971a) then proposed a theory to explain how control over a situation (coping responses) affect ulcer development. He began by noting that stress-induced ulceration was a function of (a) the number of coping responses an organism makes and (b) the amount of relevant feedback resulting from these responses. By coping responses, Weiss means any response which an organism emits which serves to reduce or "cope" with the aversive stimulation. In a controllable situation (e.g., a Sidman avoidance paradigm), lever presses would be the coping responses. In a helpless situation, coping responses would include jumping, crouching, freezing and any overt movements which might serve to reduce the shock stimulation. By relevant feedback, Weiss means stimuli which are not associated with the stressor. The feedback produced by a response can be considered as the consequence of that response. If a response decreases or eliminates aversive stimulation, it is termed relevant feedback. If a response does not alter the aversive stimulation, then the feedback resulting from such a response is termed irrelevant since little information is gained by the organism with respect to the effect of the response on the subsequent aversive stimulation. For example, if a response terminates the beep just prior to shocks, it results in the absence of any noise. Once the stimulus of silence is not associated with shock (the stressor), the feedback from the response which produced silence

and terminated the beep would be termed relevant feedback. Weiss's two postulate theory rests on these assumptions. The first postulate states that ulceration increases directly as the number of coping responses increases. Since the number of responses is intimately connected with their consequences in terms of feedback, the second postulate states that ulceration decreases directly as the amount of appropriate feedback from coping responses increases. A schematic view of this theory is shown in Figure 1.3. It can be seen from the diagram that given the number of coping responses emitted and the amount of feedback that these responses produce, the amount of ulceration can be predicted. Weiss (1971a) applied his data to this scheme, and the predictions obtained agreed with the observed data.

Weiss's data provides an excellent integration of Seligman's two dimensions along which psychological variables involved in ulceration can be classified and helps explain

> "why animals which have control over a stressor generally ulcerate less than do helpless animals: Animals which have control generally receive a considerably greater amount of relevant feedback for their coping attempts than do helpless animals. Thus the value of control for ameliorating ulcerogenic stress is said to lie essentially in the ability to produce relevant feedback from responses (p. 13)."

The next study in this series (Weiss, 1971b) demonstrated that decreasing the amount of feedback in a normally relevant-feedback-producing situation, could produce severe ulceration in animals which have control over a situation. It should be noted that this situation is similar to the results obtained by Brady (1958) with the "executive" monkeys. These

FIGURE 1.3

Weiss's (1971a) theory in schematic form. Given the number of responses (A) and the amount of relevant feedback from these responses (B), the amount of ulceration (C) can be predicted.

C
ULCERATION
None Mild Moderate Severe
A Many
RESPONSES
Few
Zero
B
High
Low Zero Negative
RELEVANT FEEDBACK
a

seemingly discrepant results can now be incorporated into the theories of Seligman (1971) and Weiss (1971a). One part of Weiss's theory holds that ulceration increases as the amount of relevant feedback decreases. Relevant feedback in yoked "helpless" animals is zero and thus they ulcerate more than do animals with control over shock. If a situation could be created in which relevant feedback from responding could be decreased below zero, into the negative range, then Weiss's theory would predict that despite being able to respond, animals in an aversive feedback situation would exhibit more ulceration than animals in a helpless but zero feedback condition. Weiss (1971b) created this situation by punishing coping responses with shock. That is, instead of responses producing stimuli not associated with shock, coping responses now produced shock and hence an aversive feedback situation. Three groups of animals; an avoidance-escape group (control over the situation), a yoked control group (helpless), and a non-shocked control group, were tested for a period of 48 hours. The first 24 hours of the session followed the usual procedure - responses of the avoidance-escape group postponed or terminated shock while responses of the yoked group were ineffectual. However, during the second 24 hours of the session, a brief shock was delivered to the avoidance-escape subjects each time they emitted a response, thereby resulting in an abrupt diminution of feedback into the negative range. Gastric ulceration data was consistent with the predictions of the theory. Avoidance-escape rats, although in control of the situation, developed significantly more ulcers than did yoked "helpless" rats. Thus feedback and its relevance were shown to be important variables involved in the production of experimental ulcers.

Weiss (1971c) varied feedback in the opposite direction. Since the previous study had shown that decreasing the amount of feedback resulted in increased ulceration, increasing the amount of feedback should decrease or even eliminate ulceration, as would be predicted from Weiss's (1971a) theory. In this study, Weiss used an unsignalled (unpredictable) shock situation since it had been shown to produce a reliably high degree of ulceration (Weiss, 1971a) and it would therefore be a good index against which to measure the effects of increased feedback from responding. Since ulceration in the unpredictable shock situation presumably resulted from the animals' emitting a large number of low feedback responses, Weiss deduced that one way to decrease ulceration would be to increase the amount of feedback. According to the theory, if feedback from responses is high, then regardless of how many responses are emitted, ulceration will not develop. Weiss therefore introduced a feedback stimulus into the unpredictable shock situation. This stimulus was a five second tone which followed each escape or avoidance response emitted by the animal. This stimulus was a source of feedback because the response-shock interval used was 200 seconds, and therefore, the occurrence of a five second tone following each response signalled a "safe" period of at least 195 seconds. In other words, following a tone, a shock could not occur for a minimum of 195 seconds. For one group of rats, the feedback stimulus was present, (high feedback group) while for a second group of rats it was ommitted (low feedback group). Yoked and non-shocked control animals were also included in the study which lasted 48 hours. The results provided confirmation of the predictions arising from Weiss's theory. Ulceration data revealed that the animals receiving the

feedback stimulus in the otherwise unsignalled shock situation, ulcerated little more than non-shocked controls and significantly less than the animals receiving the same unsignalled shock but without the feedback stimulus. Weiss noted that "it is apparent that the feedback stimulus produced a large reduction in stomach ulceration (p. 25)." Interpreting these results in light of his theory, Weiss stated that when relevant feedback is high (feedback signal present) ulceration will be low since increased responding will not affect ulceration. Conversely, when the amount of relevant feedback is low (feedback signal absent) ulceration will increase as does responding.

Finally Weiss extended his theory to account for Brady's (1958) "executive" monkey results which previously had been inexplicable. Citing the common criticism of the Brady study, Weiss noted that those monkeys assigned to be "executives" were chosen on the basis of a pre-test for Sidman avoidance response rate. Those monkeys with the fastest acquisition and highest response rate became the "executives". According to Weiss's theory, ulceration is a function of the number of responses emitted and thus, the "executive" monkeys were more likely to ulcerate than the yoked controls even before the experiment began. In addition, the "executive" monkeys were placed into an unsignalled (Sidman) avoidance situation - a situation which in itself is ulcerogenic due to the low relevant feedback resulting from coping responses made. Weiss concluded that:

> "The executive monkeys, therefore, were high-rate responders placed into a low feedback condition, which is just the combination of circumstances that the theory states will produce severe ulceration (p. 29)."

Although only tentatively demonstrated, it is evident that the role of psychological variables involved in experimental ulceration cannot be ignored. It is also evident that more research is being directed toward their study. The fact that theories concerning the role of psychological factors involved in specific psychosomatic disease states (ulceration) are being advanced indicates, to the present author, that a position of importance has finally been given to such factors.

1.2 Prevention of and Recovery from Experimental Ulceration

As can be seen from the previous review, most research has concentrated on parameters relevant to the production of ulceration. Little research has focused on the recovery from or prevention of experimental ulceration.

Mahl (1949) first mentioned one aspect of recovery but did not recognize or study it as such. He noted that when his subjects (dogs) were removed from the chronic fear situation, their gastric acidity levels returned to the "normal" pre-treatment baseline level. Although this observation was noted as early as 1949 by Mahl, the recovery factor per se was not studied until 1960 when Brodie and Hanson investigated the time course of recovery from restraint-induced ulceration. These authors noted that 24 hours of restraint stress produced ulceration in nearly all animals studied. Following this procedure, animals were sacrificed at 12 hour intervals and ulcer incidence recorded. It was found that 72 hours were required for complete recovery. Brodie and Hanson also stated that food deprivation prolonged healing time while access to food during recovery

advanced the healing process. It should be noted that restraint ulcers appear only in the glandular portion of the rat's stomach while food-deprivation ulcers usually appear in the rumen. This, in itself suggests a different ulcer etiology in the two portions of the rat stomach, and that perhaps the critical variable is not food deprivation but some other parameter. This point will be discussed later.

McFee, Stone, Goodale, Bernstein and Wangensteen (1963) used gastric freezing to prevent food deprivation and cold stress ulcers in the rat. Their results showed that the freezing procedure afforded the rumen good protection against these ordinarily ulcerogenic agents, although the freezing technique itself produced a high mortality rate. The researchers concluded that freezing was a good method by which to prevent rumenal ulceration.

Mikhail (1972) investigated the effects of the psychological variable of conditoned fear on the recovery from experimental ulceration. Ulceration was first produced by the restraint method, then the rats were exposed to the conditioned fear during recovery from these lesions. Rats were sacrificed at various time periods during 87 hours of recovery to assess the effects of fear on the healing process - a technique similar to that of Brodie and Hanson (1960). It was found that continuous or intermittent fear did not retard the healing of restraint-induced glandular ulcers but appeared to initiate rumenal ulcers. Thus, the study of recovery from ulceration led to the serendipitous finding that a single treatment could be both ulcerogenic and non-ulcerogenic. This led Mikhail to suggest that "rumen and corpus ulceration have different etiologies (p. 119)."

Mikhail noted that the variable of food deprivation seemed to be the one involved in rumenal ulceration. He cited several studies which used the conflict procedure to induce ulcers (Sawrey, Conger, and Turrell, 1958; Ader, Tatum and Beels, 1960; Weisz, 1957) and noted that the rumenal ulceration resulting from these "psychological" variables was probably due to the effects of food deprivation since it has been shown that exposure to shock decreases food intake (Sterritt, 1962; Sterritt and Shemberg, 1963) and since Ader et al. (1957) reported that the conflict-exposed rats ate significantly less than did controls. Mikhail concluded:

> "it appears that the procedures of conflict and conditioned anxiety exert their ulcerogenic effects partly through reduction of the animal's food intake and partly through direct physical damage by shock (p. 120)."

The study by Mikhail (1972) supports the hypothesis of different ulcer etiologies in the two portions of the rat's stomach. This study can also be seen as demonstrating that food deprivation per se is an important ulcerogenic variable, a finding which had been noted by other researchers (Robert and Nezamis, 1958a, b). This aspect of gastric ulcerogenesis was further qualified by Mikhail and Hirschberg (1972). This study found that starvation-induced rumenal ulceration could be significantly reduced by the addition of <u>non-nutritive</u> bulk during the food-deprivation period. This suggested that not food-deprivation per se but the absence of bulk may be the important variable in the production of rumenal ulceration. One group of rats was fed a diet of liquid sucrose for six days, while a second group was fed solid granular silica and methylcellulose (non-nutritive bulk) for the same period. Two other kinds of non-nutritive solid diets were also

used to test the generality of the preventative effects of solid bulk. It was found that both ulcer incidence and severity were significantly lower in the groups receiving solid diets than in the liquid diet group. Thus, solid bulk appeared to reduce food deprivation ulcers. The authors concluded that the physical properties of food (liquid or solid) were as significant as chemical and nutritional factors involved in gastric ulcerogenesis.

Glavin (1972) extended the Mikhail and Hirschberg findings to a different stressor condition. Restraint and cold (Levine and Senay, 1967) preceeded by a three day diet of liquid sucrose were used for the simultaneous production of both rumenal and glandular ulceration in rats. The effects of liquid or solid diets as they affected recovery from this ulceration were assessed. It was found that the presence of the non-nutritive bulk significantly enhanced recovery while the liquid diet delayed healing. Thus the presence of non-nutritive bulk appears to be critical in the initiation and prevention of experimental ulceration. This hypothesis received support from the clinical observations of Tournet (1969) who noted that rumenal ulceration in domestic animals appeared to be a function of lack of bulk in the diet (cited by Mikhail and Hirschberg, 1972).

Mikhail and Glavin (1972) tested the preventative effects of bulk on pylorous-ligated rats (Sun and Chen, 1963). It should be noted that this technique is extremely stressful, producing a 100% severe rumenal ulcer incidence after only 16 hours of ligation. As such, it provided a demanding test of the preventative action of non-nutritive bulk. One groups of rats was food deprived for 48 hours prior to ligation. A second group was food

deprived but allowed access to non-nutritive bulk for the same time period. All animals were then pylorous ligated for a period of 16 hours, after which they were sacrificed and assessed for stomach pathology. It was found that the non-nutritive bulk significantly reduced the effects of this severe ulcerogenic procedure. Thus the preventative action of bulk seems to be well substantiated across several paradigms ranging from mildly to severely stressful.

1.3 Overview of the Anatomy and Physiology of the Rat Stomach

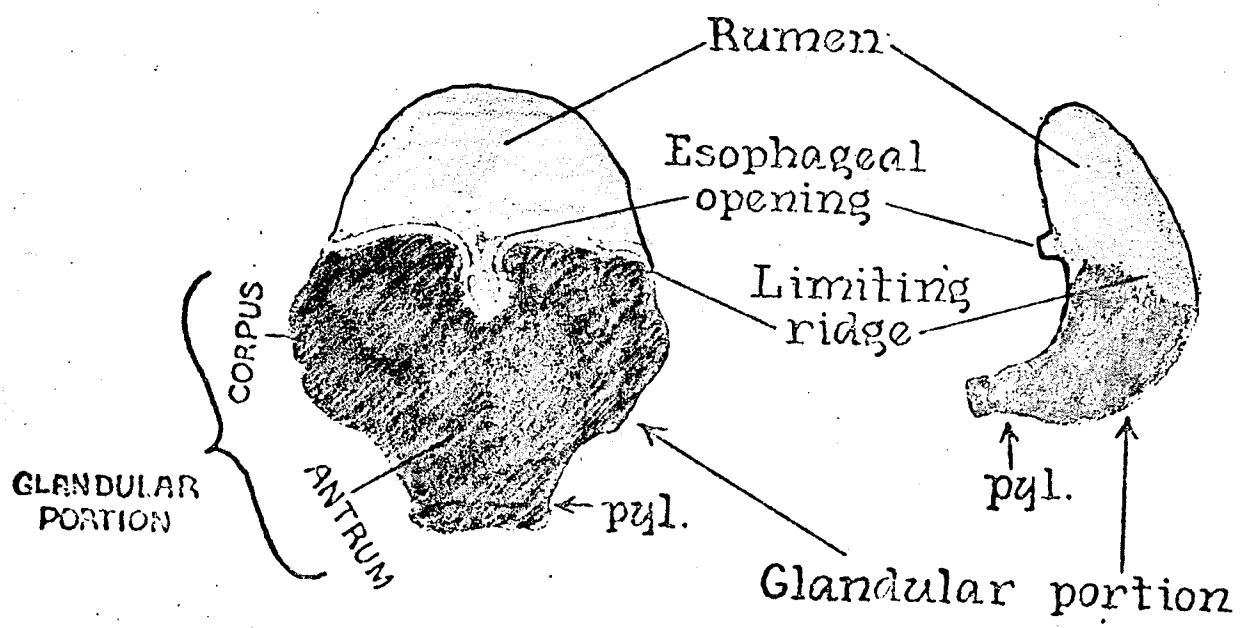
Before discussing the method used in the present study and in order to clarify subsequent discussion of the results, a brief overview of the structure and function of the rat stomach and upper gastrointestinal tract is given here. The discussion will center around Figure 1.4 which illustrates the internal and external views of the rat stomach in the normal or non-pathological state.

The two major divisions of the rat stomach are the upper two-fifths called the rumen and lower three-fifths called the body or glandular portion. The rumen is the non-secretory portion of the stomach which is lighter in color and thinner than the body. It is covered with epithelial or structural cells and its function is thought to be that of food storage prior to the actual digestive process (Berg, 1942). The ulcers which appear in the rumen are those usually resulting from starvation, maintenance on a diet of liquid sucrose and pylorous ligation.

Separated from the rumen by the raised white transverse ridge is the body of the stomach. This is the glandular or secretory portion of the stomach which Shay, Komarov, Fels, Meranze, Gruenstein and Siplet (1945)

FIGURE 1.4

Internal and external views of the rat stomach showing the major anatomical subdivisions. (From Shay et al., 1945).


Rumen
Esophageal opening
Limiting ridge
CORPUS
GLANDULAR PORTION
ANTRUM
pyl.
pyl.
Glandular portion

believe to be anatomically and functionally analogous to the body of the human stomach. It is darker in color and covered in large folds which contain parietal, chief and mucin-producing cells which secrete hydrochloric acid, pepsin and mucous respectively. Some researchers (Jackson and Thompson, 1971) divide the body into the corpus and antrum on the basis of the type of lining and glandular activity; however, for the purposes of this discussion, the body of the stomach will be considered as a whole. The ulcers which usually develop in the body are those resulting from restraint, cold stress, shock and various pharmacological agents (e.g., phenylbutazone).

Boyd (1970) has clarified the terms used to describe ulcers which form in the gastrointestinal tract of both animals and humans. According to Boyd, ulcers which appear in the stomach itself are called gastric ulcers, and those which appear in the duodenum are called duodenal ulcers. Both these types of ulcers have been loosely referred to as peptic ulcers. This may be due to the fact that the stomach and duodenum are above the more alkaline bile and pancreatic juice so that both are exposed to the more acidic gastric juice, and thus gastric and duodenal ulcers would seem to have a common causative agent. Boyd states, however, that gastric and duodenal ulcers differ in their hereditary characteristics, symptoms, and amount of acid secretion involved and as such, should be considered separately.

The process of digestion involves the action of acid in the stomach. Stomach acidity has been implicated in many studies as an important ulcerogenic agent. Therefore, a brief discussion of acid secretion

and its possible role in gastric ulcerogenesis follows.

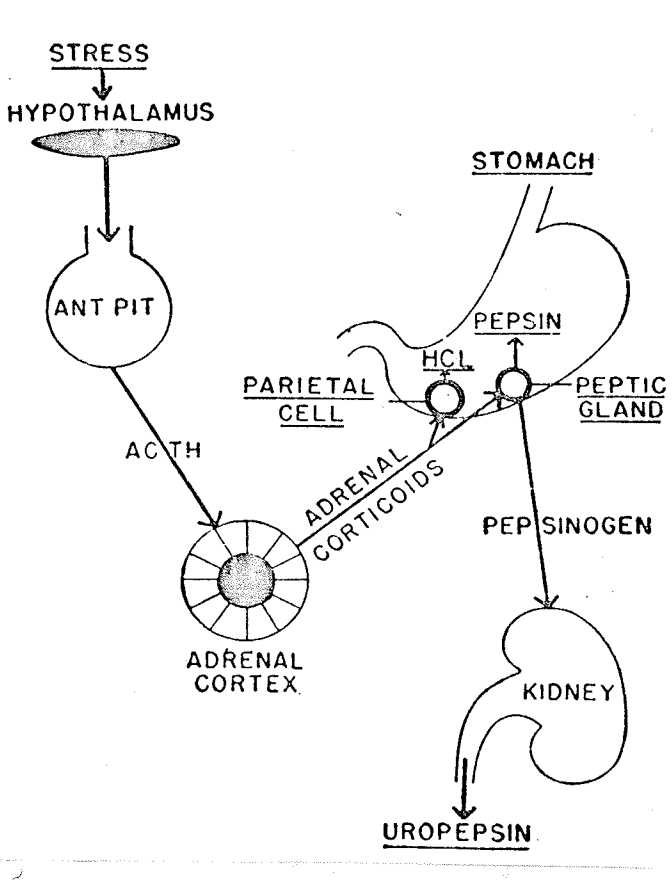
Acid secretion in the stomach is both neurally and hormonally controlled (Boyd, 1970). Neural control of acid secretion is achieved via the parasympathetic vagus nerve which liberates acetylcholine in the stomach. The acetylcholine stimulates histamine release which, in turn, results in increased hydrochloric acid secretion. The hormonal control of stomach acidity can be affected by emotional disorders. Gellhorn and Loofbourrow (1963) noted that emotional stress can act via the hypothalamus which stimulates the anterior pituitary by means of a corticotropin releasing factor. This causes the anterior pituitary to release adrenocorticotrophic horome (ACTH) which stimulates the adrenal cortex. The corticoids of the adrenal glands act on parietal and chief cells in the stomach and cause their respective secretions of hydrochloric acid and pepsin to increase.

It is thought (Boyd, 1970) that gastric lesions are formed due to the increased acid and pepsin secretions which destroy the protective mucous lining of the stomach. The protective influence of the mucosa, often called mucosal competence or resistance is thought to be related to blood circulation (Boyd, 1970). The resistance of the mucosa is increased or decreased as a function of the action of the gastric acidity. When mucosal resistance is low, the stomach surface is exposed and becomes vulnerable to the action of the acids present. Figure 1.5 illustrates the hormonal pathway through which emotional stress can affect the gastro-intestinal system.

It is evident that physiological function in general and gastro-intestinal function in particular cannot be considered apart from

FIGURE 1.5

Hormonal pathway by which emotional stress influences gastric secretory activity. (From Harrison *et al.*, 1966).


STRESS
HYPOTHALAMUS
STOMACH
ANT PIT
PEPSIN
HCL
PARIETAL CELL
PEPTIC GLAND
ACTH
ADRENAL CORTICOIDS
PEPSINOGEN
ADRENAL CORTEX
KIDNEY
UROPEPSIN

psychological function. Boyd (1970) concluded:

> "The stomach then, is a veritable sounding board of the emotions, and when one considers the bombardment it suffers from neurogenic, secretory, and hormonal stimuli, not to mention exogenous irritants of every sort and description, the wonder is that anyone has a healthy digestion. (p. 798)."

1.4 Statement of the Purpose of the Study

The main purpose of the present study was to investigate acidity changes in the rumen over the temporal course of the development of rumenal ulceration. As a result, it was felt that some information could be gained as to why rumenal ulceration takes from three to five days to develop. The effects of a predictable or unpredictable but inescapable shock situation on rumenal acidity were assessed in order to examine the role of acid in the development of rumenal ulceration.

In addition to the main purpose, the study will attempt to deal with the following problems:

(a) To investigate the existence of a correlation between acidity and ulceration in the rumenal portion of the stomach.

(b) To study ulcer development in both portions of the rat stomach by means of separate acidity measures, and thereby investigate the suggestion of Mikhail (1972), that ulcers in the two portions of the rat stomach may have different etiologies.

(c) To assess the effects of the physical property of food (liquid or solid) on ulceration and to confirm the apparently protective effect of solid bulk in a situation which has been shown to be highly ulcerogenic (Mikhail and Hirschberg, 1972).

CHAPTER II

METHOD

2.1 Subjects

The subjects were 168 male albino rats of the Wistar strain obtained from Canadian Breeding Farms, Montreal, Canada. The animals were 65-70 days of age at the start of the experiment. The mean weight of the animals was 180 gr (S.D. = 11 gr). Each animal was randomly assigned to a treatment condition.

Table 2.1 presents an outline of the number of subjects in each of the experimental and control conditions. The four experimental groups consisted of: liquid diet + predictable shock, liquid diet + unpredictable shock, solid diet + predictable shock, and solid diet + unpredictable shock. The control groups consisted of: liquid diet + no shock and solid diet + no shock. In addition, a group of 24 no shock + ad lib diet control subjects was included. Each experimental and control group consisted of eight subjects and each group was replicated after 48, 72, and 96 hours of treatment with diet and shock.

2.2 Apparatus

Conditioning cages of three sides of stainless steel and a plexiglass front were employed. Each of the 20 cages measured 22.5 x 15.5 x 22.5 cm. The floor of each cage consisted of an electrified grid of metal bars .25 cm in diameter and placed 1.5 cm apart, through which a shock of 2.5 ma. and .5 sec. was administered (Fig. 2.1 and 2.2). Each cage was covered with a sheet of milk glass through which a light signal (CS) was

TABLE 2.1

OUTLINE OF EXPERIMENTAL GROUPS AND TREATMENTS SHOWING THE NUMBER OF SUBJECTS IN EACH CONDITION*

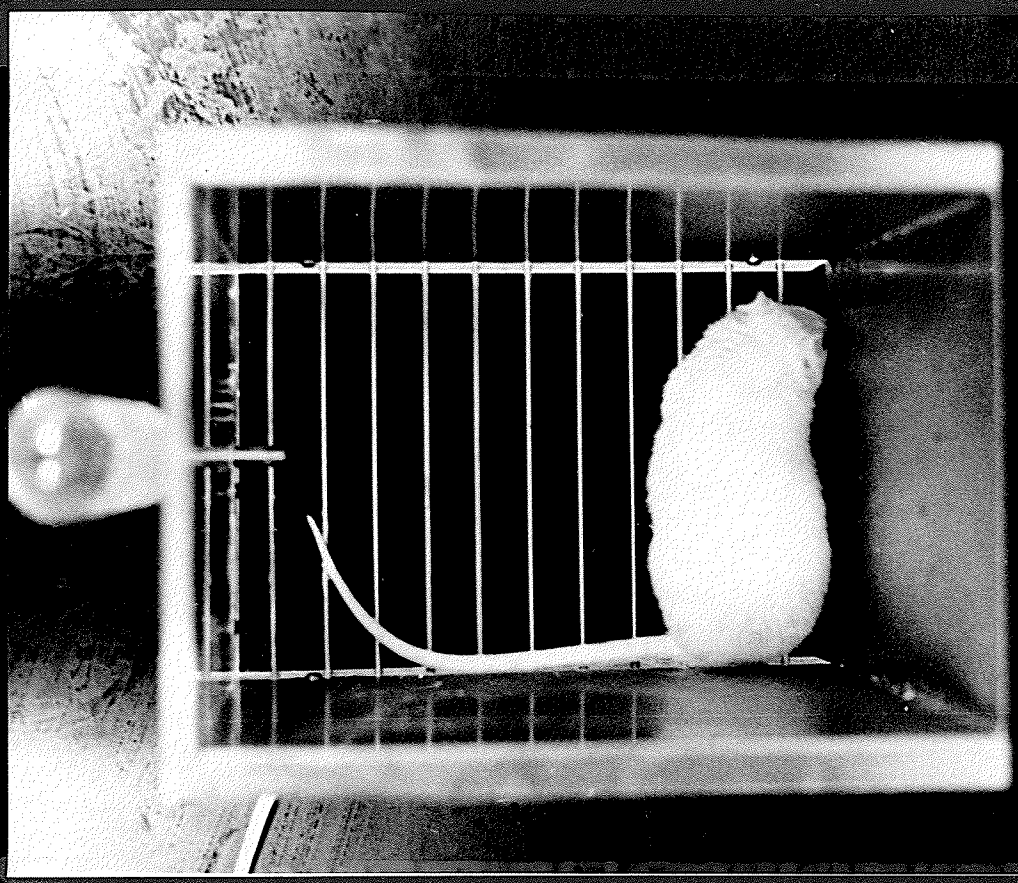
	LIQUID	SOLID
PREDICTABLE SHOCK	8	8
UNPREDICTABLE SHOCK	8	8
NO SHOCK	8	8

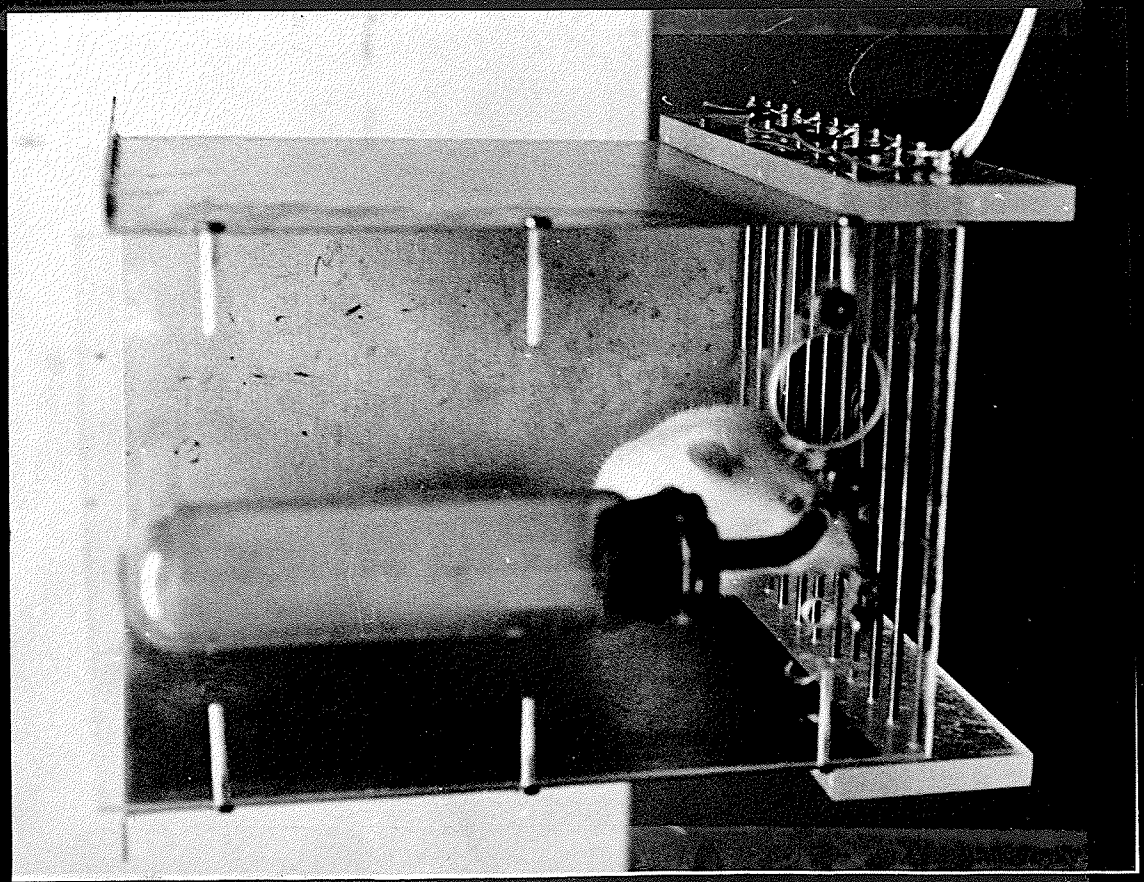
*Each diet and shock group was replicated at 48, 72, and 96 hr. of treatment.

FIGURES 2.1 AND 2.2

Figure 2.1 Top view of a conditioning cage used for the conditioned fear treatment. On the floor of the cage can be seen the grids through which the shock was administered. The top of a graduated cylinder is shown at the front of the cage.

Figure 2.2 Front view of a conditioning cage used for the conditioned fear treatment. The graduated cylinder at the front of the cage was used to administer the liquid diet. The solid diet was inserted through the round hole and secured by the two screws on either side of the hold.





presented. The light originated from five 60 w. electric bulbs which transmitted a light stimulus of 27 foot candles measured from the reflectance of a Kodak standard 18% grey card held at a 45° angle to the front of the cage at the level of the grid floor.

The duration of the CS was 12 sec. It should be noted that CS duration per se has been shown to have no effect on the acquisition of a conditioned emotional response (Brogden, 1954; Kamin, 1965). Brogden (1954) noted that:

> "there is no evidence that the duration of the CS affects either the acquisition or the characteristics of CR's independently of the interval between the onset of the CR and onset of the CS (p. 579)."

An electronic programmer was used in presenting the lights and shocks to the animals according to their respective schedules of predictable and unpredictable shock. The shock procedure of the present study is similar to that used by Freidman and Ader (1965) who varied periodic and aperiodic CS's and US's in their study. Periodic schedules (analogous to the predictable shock in the present study) were created by having the light CS occurring at a fixed time interval of 15 sec. and terminating simultaneously with a shock US. Aperiodic schedules (analogous to the predictable shock in the present study) were created by having the CS and US occur at random time intervals, ranging from 5.5 to 24.5 min.

The subjects were fed sucrose in a liquid or solid form. The liquid diet consisted of a 20% sucrose solution (20 gr sucrose/100 ml). The solid diet consisted of a mixture of granular silica, methylcellulose, and sucrose. A viscous solution of methylcellulose and water (1 gr methylcellulose per 100 ml) was prepared and mixed with the silica. The

concentration of sucrose was 20% by weight. Methylcellulose was used as a binding agent to form a mushy edible mixture out of the granular silica and sucrose. Bauer and Lehman (1951) noted that in rats, methylcellulose was not absorbed from the alimentary tract nor hydrolyzed to cellulose and methyl alcohol. It should also be noted that the chemical composition of sucrose cannot directly influence the pH of the gastric juice believed to be involved in ulcer formation (Senay and Levine, 1970).

All animals were killed by ether overdose (Fisher Scientific Co.). Stomach pH measurements were determined by Fisher Scientific pH paper (Cat. No. 14-837-1) having a range of 0.0 to 11.0 in increments of .5.

2.3 Procedure

(a) Experimental groups

Beginning at 1000 hr. on the first day, the experimental subjects were placed individually into the conditioning cages and administered the liquid or solid diet. The liquid sucrose was presented in 100 ml graduated cylinders and the solid diet was presented in small metal containers which were inserted through the front of the conditioning cages. All subjects in the solid diet condition also had access to liquid sucrose throughout the experiment. The conditioning treatment also began at 1000 hr. on the first day and consisted of a 12 sec. light CS which terminated with the onset of a .5 sec. 2.5 ma. shock US. The predictable shock condition was created by reinforcing 100% of the CS's with shock, with the time interval between CS and US pairings fixed at five min. The unpredictable shock condition was created by randomly reinforcing 50% of

the CS's with shock. The other 50% of the shocks and CS's occurred randomly with the restriction that a shock could not occur within one min. of a CS. The time intervals between shocks were: 144, 192, 96, 600, and 198 sec. with an average intershock interval of 300 sec. or five min. The total period of conditioning was 22 hr. out of each 24 hr. of the experiment, thereby allowing the subjects two hr. rest for each 24 hr. of conditioning. During the rest period, which occurred at the same time each day, (0800 hr. to 1000 hr.) all subjects remained in the conditioning cages with the CS and US turned off. Liquid and solid intake was measured and the containers refilled as necessary. Following the end of the conditioning periods, all subjects were killed by ether overdose, and their stomachs examined for ulceration according to the method of Mikhail and Holland (1966). This procedure involved making an incision in the abdomen to expose the stomach. The esophagus was ligated close to the esophagocardiac junction of the stomach and was then severed. The stomach was freed from attached tissue, and the duodenum was severed approximately three cm from the stomach. Twenty cubic centimetres of air were injected through the duodenum into the stomach. The duodenum was then ligated and the stomach remained inflated for approximately ten min. The inflation procedure served to expand the stomach so that when opened, it remained stretched. The detecting and photographing of ulcers were facilitated by the stretching procedure. The stomach was opened by an incision along the greater curvature and was laid on a clear plexiglass slide. A separate strip of pH paper was touched to the rumen and to the body of the stomach in order to determine the pH of these two portions. Two readings were

taken from each portion of the stomach. All stomachs were then washed of their residual contents and illuminated above a sheet of milkglass for ulceration rating and photographing. Ulceration was rated on a relative scale of 0 to 4 points from least to worst respectively (Lambert, 1968). A rating of 0 indicates the absence of pathology. A rating of 1 indicates a non-normal but not severely ulcerated stomach. Only one or two small ulcers are present in a stomach which receives a 1 rating. A 2 rating indicates the clear presence of a small number of ulcers while a 3 rating is indicative of a greater number of and often bleeding ulcers. A 3 rating also indicates the absence of perforation. A rating of 4 is reserved for the case of perforation in the stomach caused by a large number of severe, bleeding ulcers.

(b) Control groups

All control subjects remained in individual cages in the colony room of the Department of Psychology at the University of Manitoba. They were administered the liquid and solid diets in containers identical to those of the experimental subjects and in their home cages for the three time periods used in the study. Following expiration of a given time period, the control animals were killed and their stomachs examined in the same manner as were the experimental subjects.

(c) Statistical analyses

All analyses of variance, unless otherwise stated, were 2 x 3 x 3 factorial analyses of variance with two levels of diet (liquid and solid), three levels of shock (predictable, unpredictable, and no shock), and three levels of time (48, 72, and 96 hr.). The analyses of

variance were used to analyze the ulceration and acidity data.

Individual comparisons among pH means were performed with a two independent sample t-test. Comparisons among ulceration rating means were calculated with the non-parametric Mann-Whitney U test since ratings represent ordinal data. In both cases, the error rate was controlled by Dunn's (1961) technique. Essentially, this technique splits up the significance level of the individual comparisons to compensate for the number of comparisons performed. Dunn's procedure places an upper limit on the familywise error rate for the total number of comparisons calculated whether orthogonal or not. The original α level is split up among the comparisons such that the sum of the per comparison error rates is less than or equal to the original α level (Games, 1971).

All correlational analyses between ulceration and acidity, employed Spearman's coefficient of rank correlation.

CHAPTER III

RESULTS

3.1 Rumenal pH

Mean rumenal pH over time, as a function of diet (Figure 3.1) showed that the liquid diet groups were more acidic than were the solid diet groups at all three testing periods (48, 72, and 96 hours). This pattern was consistent across all experimental and control groups given the liquid diet (Appendix 1).

Analysis of variance (Appendix 2) of rumenal pH confirmed that the liquid diet condition resulted in significantly ($F=54.5$; df: 1,126, $p<.001$) greater acidity than the solid diet condition. The main effect of time showed a non-significant linear trend in the direction of increased acidity over time.

Mean rumenal pH over time, as a function of shock condition (Figure 3.2) revealed that the unpredictable shock groups increased in acidity over time, while the predictable shock and no shock groups did not start to increase in acidity until after 72 hours of treatment. Individual comparisons between means revealed no significant differences within shock conditions.

3.2 Glandular pH

Glandular pH measurements (Appendix 3) revealed a relatively constant mean pH of 3.5 (S.D.=6) in all groups tested at all time periods.

Under normal, liquid, and solid diet conditions, and in all shock conditions, glandular pH was more basic than rumenal pH (Appendix 3).

FIGURE 3.1

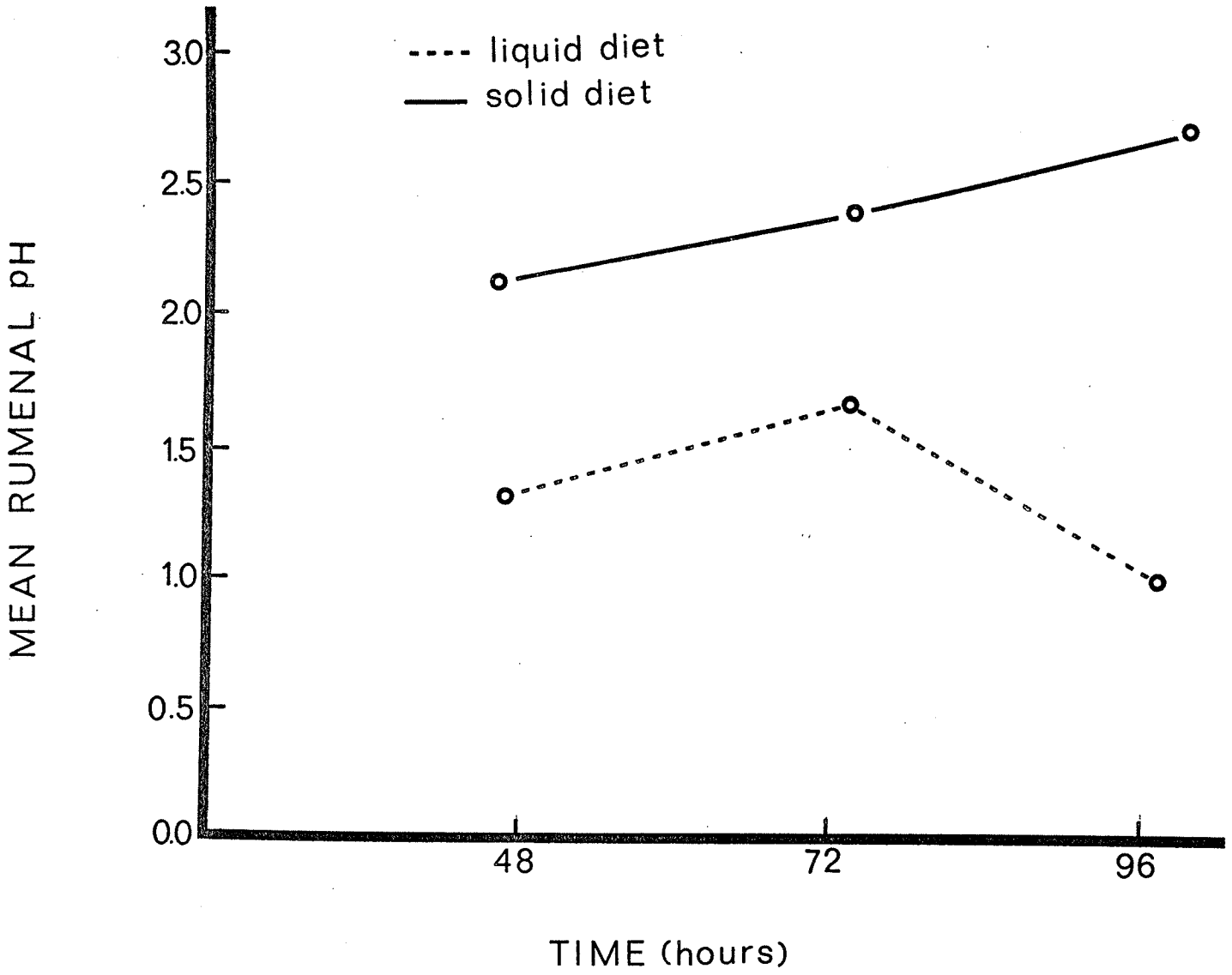
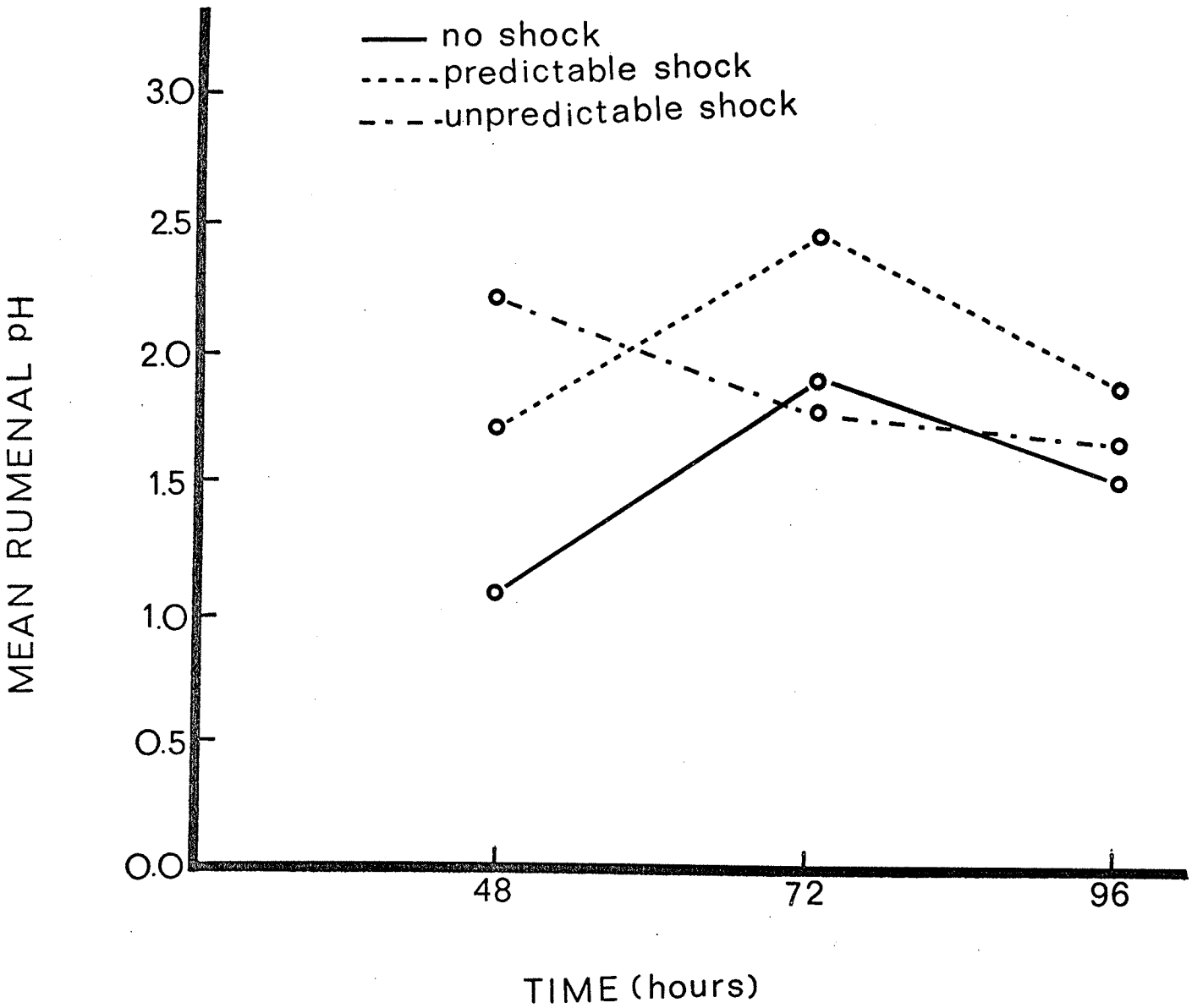

---- liquid diet
—— solid diet
MEAN RUMENAL pH
3.0
2.5
2.0
1.5
1.0
0.5
0.0
48
72
96
TIME (hours)

FIGURE 3.2


no shock
predictable shock
unpredictable shock
MEAN RUMENAL pH
3.0
2.5
2.0
1.5
1.0
0.5
0.0
48
72
96
TIME (hours)

3.3 Rumenal ulceration

Mean rumenal ulceration over time as a function of diet (Figure 3.3) revealed that little ulceration developed in the solid diet groups, while ulceration in the liquid diet groups increased over time. Analysis of variance (Appendix 4) revealed that the main effect of diet was significant ($F=107.52$; df: 1,126, $p<.001$) indicating that the liquid diet groups developed more ulceration at all testing periods. Ulceration incidence data (Appendix 5) indicated that the pattern of increased ulceration in the liquid diet condition was consistent across all experimental and control groups.

No significant differences in ulceration over time were found within the solid diet groups. Comparisons of mean rumenal ulceration over time within the liquid diet groups (Table 3.1) revealed that ulceration increased from 48 to 72 hours ($p<.003$) and from 48 to 96 hours ($p<.003$).

Rumenal ulceration as a function of shock (Figure 3.4) revealed that the unpredictable shock condition resulted in greater ulceration than either the predictable shock or no shock conditions. Analysis of variance (Appendix 4) confirmed that the shock effect was significant ($F= 12.09$; df: 2,126, $p<.001$). Table 3.2 presents individual comparisons of rumenal ulceration between levels of shock. Ulceration was greater in the unpredictable shock group than in both the no shock group ($p<.016$) and the predictable shock group ($p<.016$).

Analysis of variance (Appendix 4) revealed that ulceration in the rumen increased significantly over time ($F= 19.93$; df: 2,126, $p<.001$).

FIGURE 3.3

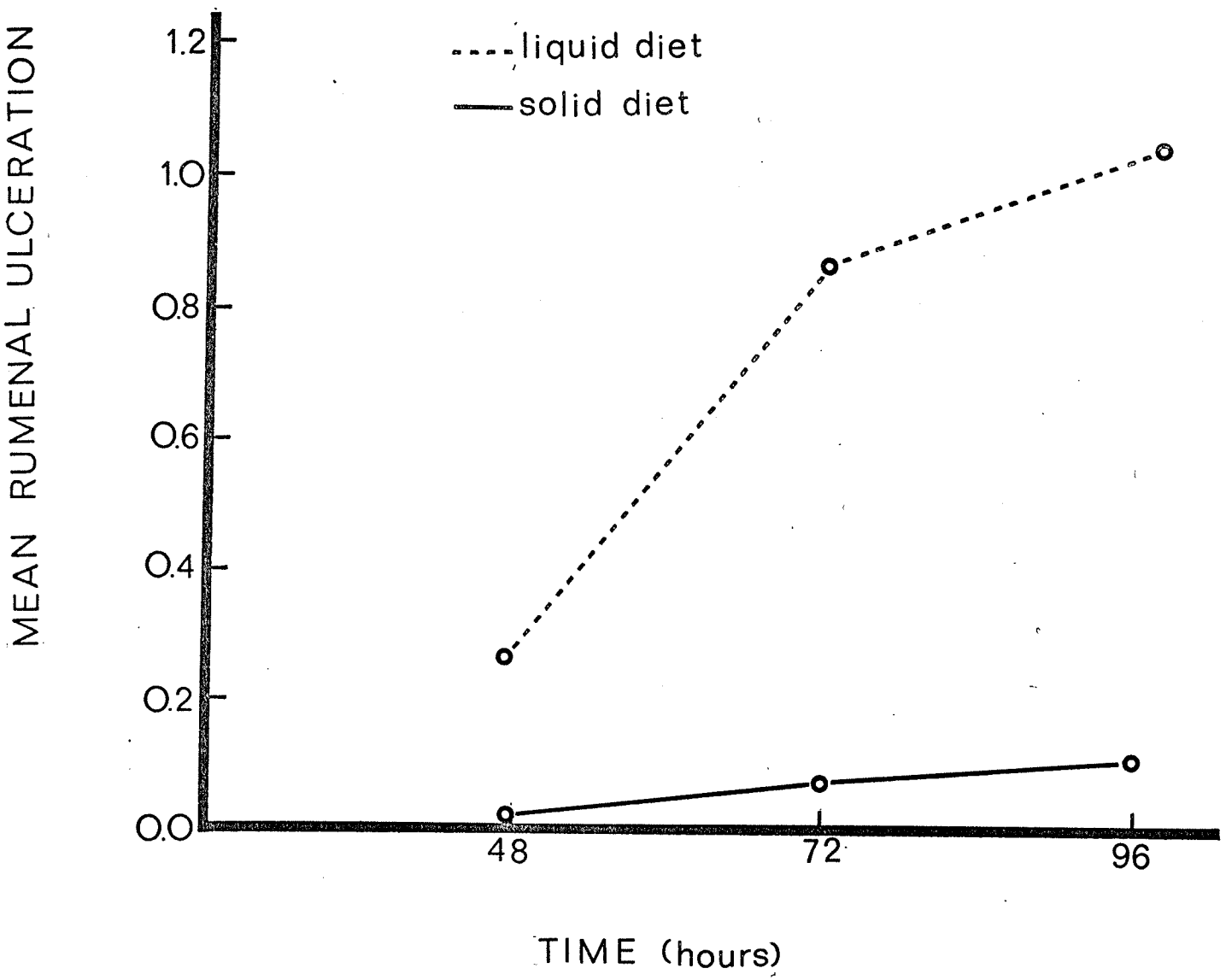

MEAN RUMENAL ULCERATION
1.2
1.0
0.8
0.6
0.4
0.2
0.0
liquid diet
solid diet
48
72
96
TIME (hours)

TABLE 3.1

MANN-WHITNEY U TEST COMPARISONS OF RUMENAL ULCERATION IN THE LIQUID DIET GROUPS BETWEEN LEVELS OF TIME

	72 hours	96 hours
48 hours	z= -2.92**	z= -5.23**
72 hours		z= -1.24

** $p < .003$

FIGURE 3.4

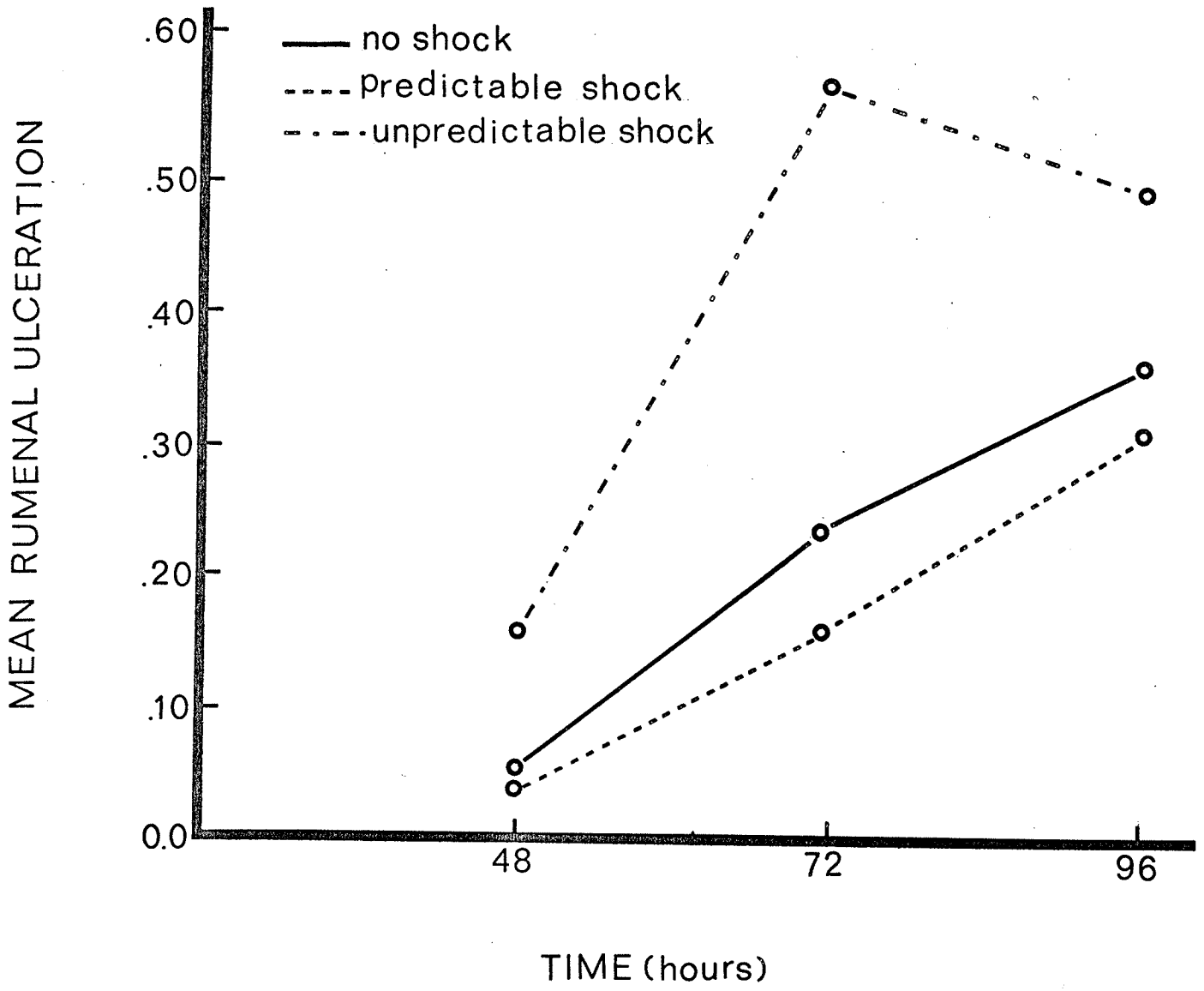

no shock
Predictable shock
unpredictable shock
MEAN RUMENAL ULCERATION
.60
.50
.40
.30
.20
.10
0.0
48
72
96
TIME (hours)

TABLE 3.2

MANN-WHITNEY U TEST COMPARISONS OF RUMENAL ULCERATION BETWEEN LEVELS OF SHOCK

	Predictable Shock	Unpredictable Shock
No Shock	z= -0.04	z= -2.59*
Predictable Shock		z= -2.73*

* $p < .016$

The time x diet interaction was also significant ($F = 11.06$; df: 2,126, $p < .001$) indicating that increasing the amount of time maintained on a liquid diet resulted in greater ulceration. Individual comparisons between levels of time (Table 3.3) showed that differences in rumenal ulceration between 48 and 72 hours and between 48 and 96 hours were significant ($p < .003$).

3.4 Glandular ulceration

No instance of glandular ulceration was observed in any subject throughout the entire experiment.

3.5 Correlations between ulceration and pH

Significant correlations between rumenal ulceration and pH (Table 3.4) were found for all groups except the liquid diet + shock groups tested at 96 hours. Figure 3.5 illustrates the increase in acidity and concomitant increase in ulceration over time.

These correlations can be expressed as a measure of association between the independent and dependent variables; that is, the proportion of variance in the dependent measure accounted for by the independent variable (Vaughan and Corballis, 1967). These results are shown in Table 3.5. Within the significant correlations, the proportion of variance accounted for by the independent variable ranged from 50% to 98%.

Appendix 6 presents photographs of the stomachs of representative subjects within each group at each testing period.

3.6 Liquid and solid sucrose intake

Daily liquid and solid sucrose intake (Appendix 7) revealed that within each testing period, both liquid and solid sucrose consumption remained homogeneous for all groups.

TABLE 3.3

MANN-WHITNEY U TEST COMPARISONS OF RUMENAL ULCERATION BETWEEN LEVELS OF TIME

	72 hours	96 hours
48 hours	z= -2.77**	z= -4.52**
72 hours		z= -1.45

** p<.003

TABLE 3.4

SPEARMAN'S COEFFICIENT OF RANK CORRELATION BETWEEN RUMENAL ULCERATION AND pH FOR GROUPS TESTED AT 48, 72, 96 HOURS[1]

	LIQUID DIET			SOLID DIET
Testing Period	control	predictable shock	unpredictable shock	unpredictable shock
48 Hours	-.72*	-.73*	-.92**	-.71*
72 Hours	-.91**	-.95**	-.82*	-.99**
96 Hours	-.88**	-.55	-.55	-.74*

* $p < .05$

** $p < .01$

[1] Groups not shown did not develop ulceration

FIGURE 3.5

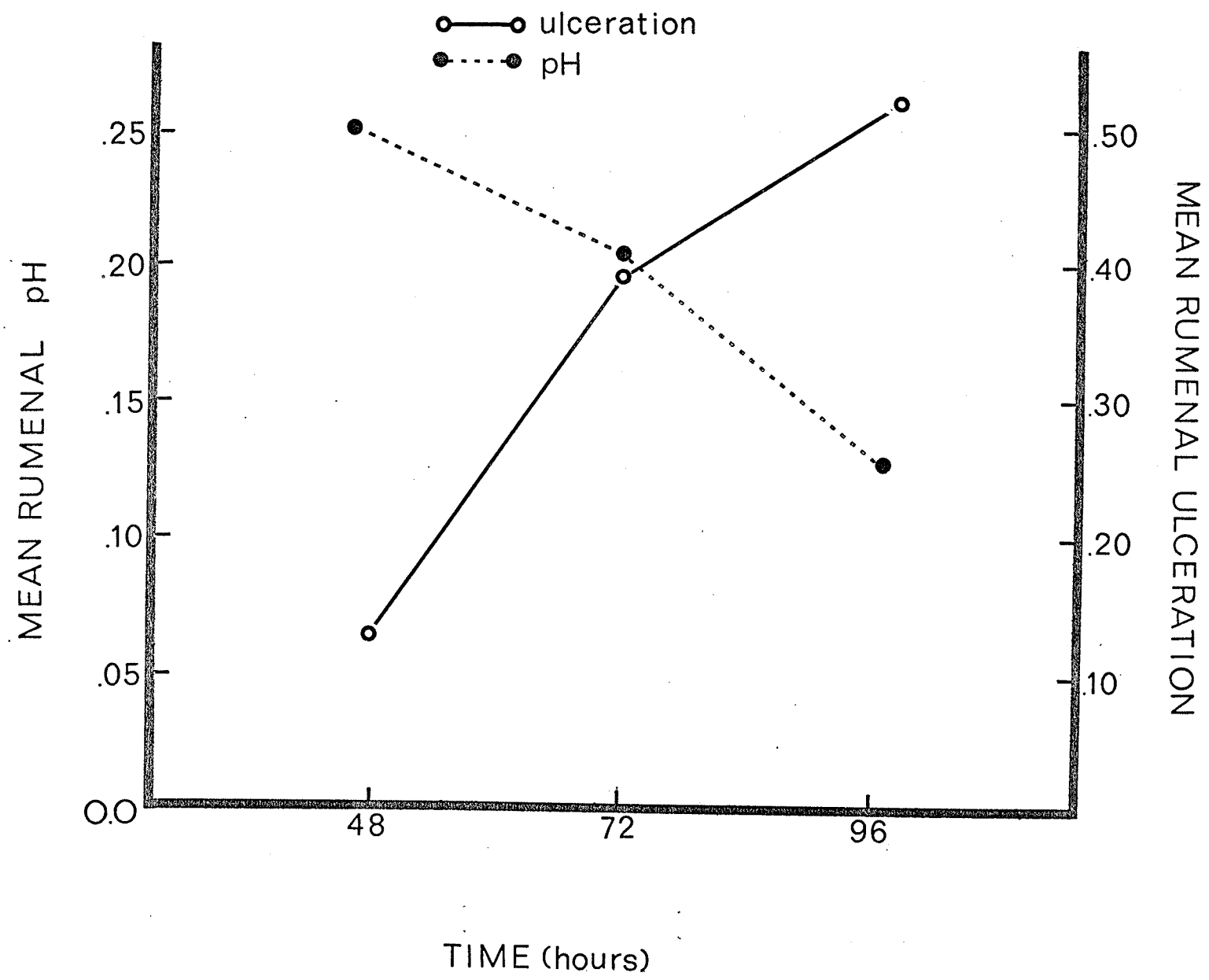

ulceration
pH
.25
.20
.15
.10
.05
0.0
48
72
96
.50
.40
.30
.20
.10
MEAN RUMENAL pH
MEAN RUMENAL ULCERATION
TIME (hours)

TABLE 3.5

LEAST PROPORTION OF VARIANCE ACCOUNTED FOR IN THE DEPENDENT VARIABLE BY THE INDEPENDENT VARIABLE (EXPRESSED AS A PERCENT) IN THE GROUPS WHICH DEVELOPED RUMENAL ULCERATION

		LIQUID DIET		SOLID DIET
Testing Period	control	predictable shock	unpredictable shock	unpredictable shock
48 Hours	51%	53%	84%	50%
72 Hours	82%	90%	67%	98%
96 Hours	77%	30%	30%	54%

CHAPTER IV

DISCUSSION

The findings of the present investigation are discussed below in the following order. First, issues related to stomach acidity; second, issues related to stomach ulceration.

(1) The lower acidity (higher pH) in the rumen of the solid diet rats relative to the liquid diet subjects. The pH data indicated that the liquid diet condition resulted in a consistently acidic state in the rumen. In contrast, the solid diet subjects displayed a consistently basic state in the rumen as well as very little ulceration in this portion of the stomach. The difference in rumenal pH as a function of diet was highly significant (Appendix 2). The reduction of rumenal ulceration in solid diet subjects was also observed in previous studies (Mikhail and Hirschberg, 1972; Glavin, 1972; Mikhail and Glavin, 1972). In these studies, however, the acidity of the rumen was not examined. The foregoing observations suggest that the anti-ulcerogenic effect of solid bulk is mediated by a reduction of acidity in the rumen of the stomach. The mechanism whereby solid bulk decreases stomach acidity may involve pressure on the hydrochloric acid-secreting (parietal) cells of the gastric mucosa. Only tentative evidence, however, indicates that parietal cells respond to pressure by decreasing their secretion rates (Babkin, 1950; Helander, 1964; Patt and Patt, 1969; Jackson and Thompson, 1971).

(2) The absence of stomach pH changes as a function of shock predictability. The present study revealed no significant differences in

rumenal or glandular pH as a function of shock predictability. Rumenal acidity in the unpredictable shock group increased steadily over time, while the predictable and no shock conditions did not produce an increase until after 72 hr of conditioning.(Figure 3.2). It appears that the variable of shock predictability does not markedly affect gastric acidity, although subjects in the unpredictable shock groups displayed a consistently but not significantly more acidic state in the rumen than did subjects in the other shock groups. These findings are contrary to those of Weiss (1970) and Caul, Buchanan, and Hays (1972) who found that severe glandular ulceration developed as a result of unpredictable shock. In the present study, glandular ulceration was not observed and glandular pH remained in a relatively constant basic state regardless of treatment. The present data do, however, provide some support for the view that exposure to fear-provoking situations reduces rather than increases stomach acidity (Mikhail, 1972). It should be noted in this regard that the rumenal pH data (Figure 3.2) indicated that the predictable shock subjects displayed a more basic state in the rumen than did the non-shocked rats.

(3) The increase of rumenal acidity following 72 hr of the treatment. The present results indicated that rumenal acidity began to increase after the third day of treatment in all but the unpredictable shock conditions (which increased steadily over time). A similar temporal pattern in rumenal acidity was observed by Shay et al.(1945) and by Sun and Chen (1963), although different stressors were used in these investigations. Brodie and Hanson (1960) found that rumenal ulceration developed after 72 hr of restraint stress. They explained the development

of rumenal ulceration in terms of the intermittent stress schedule used in their study, but no account was offered for the delay in ulcer formation. From the present results, it appears that the period of three days required for acidity increase accounts for the delay in the development of rumenal ulceration.

(4) The absence of treatment effects on the pH of the corpus. Glandular pH did not significantly change regardless of the treatment condition. This finding is inconsistent with that of Weiss (1970) but is in line with the observation (Mikhail, 1969) that conditioned fear does not increase gastric acid secretion. Weiss's data may have been a function of shock intensity per se rather than a function of shock predictability.

(5) The consistently greater acidity of the rumen relative to the corpus. The pH data revealed that in all groups, the rumenal portion of the stomach was consistently more acidic than the body. Glavin (1972) found similar results using restraint and cold stress. When the pH data from both portions of the stomach is considered together, these results tend to support the view that there is a dissimilar ulcer etiology in the two portions of the rat stomach (Shay et al., 1945; Robert and Nezamis, 1958; Mikhail, 1972). The rumen and the body, while receiving identical treatments, displayed very different acidity states at the times of testing. These findings suggest that acidity may be common to ulceration in both portions of the stomach, but that the pattern of acidity increase and the treatment necessary to produce it are different.

(6) The anti-ulcerogenic effects of solid diet. The liquid diet condition resulted in a steady increase in rumenal ulceration over time, while very little rumenal ulceration was found in any subjects administered the solid diet. This finding provides support for those of Mikhail and Hirschberg (1972) who found that solid non-nutritive bulk prevented starvation ulcers, and those of Mikhail and Glavin (1972) who found that solid bulk significantly reduced pylorous ligation-induced ulceration. It appears that the physical properties of food may be as important as the chemical and nutritional properties with respect to gastric ulcerogenesis.

(7) Shock predictability and ulceration. No instance of glandular ulceration was observed in the present study, although the unpredictable shock condition produced the most severe rumenal ulceration. These results are inconsistent with previous research investigating the variable of predictability (Seligman, 1968; Weiss, 1970; Caul, Buchanan, and Hays, 1972). The absence of glandular ulceration, however, may be explained by the findings of Senay and Levine (1971) who found a correlation between ulceration and acidity in the body of the stomach. The glandular portions of the stomachs in this study displayed a consistently basic state. Thus, ulceration would not be expected to develop since the Senay and Levine study implicated an acidic state as a necessary condition for glandular ulcerogenesis. It may also be argued that conditioned fear was not an ulcerogenic treatment. Wolf and Wolff (1947), Mahl (1949), and Brady (1958) found that fear inhibited gastric acidity. In fact, neither Wolf and Wolff nor Mahl found any evidence of ulceration in their subjects. Thus,

the present results support the position of Mikhail (1969) who argued that conditioned fear is not an ulcerogenic variable regardless of the shock intensity used. Given this research and the glandular pH data from the present study, it is not surprising that ulceration did not develop in the corpus of the stomachs in this investigation.

(8) Correlations between rumenal ulceration and pH. Rumenal ulceration and rumenal pH showed significant negative correlations in all but two groups. Previous research has demonstrated the existence of such a correlation only in the body of the stomach (Senay and Levine, 1971). Thus, the results of the present study extend those of Senay and Levine to include the rumenal portion of the stomach and indicate that rumenal ulceration is due to increased gastric acidity.

Considered together, the results of this study indicate that ulceration in the two portions of the rat stomach has a similar etiology, since increased acidity is common to both. Although acidity has been shown to be involved in ulceration in both portions of the stomach, the present results indicate that different treatments are necessary to produce the increased acidity- that is, a given treatment may increase acidity in one portion but not in the other. This means that a differential acidity response in the rumen and the body may result from the same stressor whether physical or psychological.

A common ulcerogenic factor- increased acidity- appears to be a necessary condition for ulceration development in both the rumen and the body. The results of the present study lead to the hypothesis that the presence or absence of the increased gastric acidity is determined by the nature of the stressor employed. The finding that a single

stressor increased acidity and produced ulceration in the rumen but effected no glandular ulceration or pH changes, provides support for this hypothesis.

CHAPTER V

SUMMARY

The main objective of the present study was to examine rumenal pH over the known temporal course of rumenal ulceration development in order to gain information into the three to five day time period necessary for rumenal ulcerogenesis. Glandular pH and ulceration measures were also recorded in order to test the hypothesis of different ulcer etiologies in the two portions of the rat stomach. Shock predictability and dietary physical property within an inescapable and unavoidable shock situation were varied in order to assess their effects on both rumenal and glandular ulceration and pH, and to test the hypothesis that solid bulk decreases stress-induced ulceration, while its absence enhances the development of such ulceration.

The results indicated that:

1) Rumenal acidity did not begin to increase until after the third day of treatment. Rumenal ulceration development coincided with the acidity pattern. It was concluded that gastric acidity was responsible for the three to five day delay in rumenal ulcerogenesis.

2) There were highly significant correlations between acidity and ulceration in the rumen, thereby extending the results of Senay and Levine (1971) who found that acidity and ulceration correlated significantly in the glandular portion of the stomach.

3) A diet of solid food resulted in significantly reduced acidity and ulceration levels when compared to a liquid diet. This finding supported those of Mikhail and Hirschberg (1972) who concluded that dietary physical properties were as important as the chemical ones in gastric ulcerogenesis.

4) The rumen and the body did not respond unidimensionally to the same stressor. Very dissimilar patterns of ulceration and acidity were found in the rumen and the body at all testing periods. On the basis of the data which indicated that the same stressor produced a differential acidity response in each portion of the stomach, it was concluded that the present study supported the hypothesis of different ulcer etiologies in the rumen and in the body of the rat stomach. It appears that although ulcerogenesis in both portions of the stomach has been shown to involve increased gastric acidity, a difference in ulcer etiology in the rumen and the body lies in the treatment which produces the increase. Support was thus demonstrated for the specific rather than the general effects of a stressor.

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APPENDIX 1

Rumenal pH Means in all Experimental and Control Groups at all Testing Periods

TABLE 1

MEAN RUMENAL pH IN ALL EXPERIMENTAL AND CONTROL GROUPS

TIME/GROUP	LIQUID			SOLID		
	predictable shock	unpredictable shock	no shock	predictable shock	unpredictable shock	no shock
48 hours	2.1	2.4	1.9	1.4	1.8	0.8
72 hours	2.8	2.4	2.3	2.0	1.4	1.5
96 hours	2.6	2.7	2.6	1.2	1.0	0.8

APPENDIX 2

Summary of analysis of variance

of rumenal pH.

TABLE 1

SUMMARY OF 3 x 2 x 3 ANALYSIS OF VARIANCE ON

RUMENAL pH: TIME, DIET, AND SHOCK

Source	df	MS	F
TIME	2	1.59	1.90
DIET	1	45.56	54.45**
SHOCK	2	1.44	1.73
TIME x DIET	2	2.25	2.69*
TIME x SHOCK	4	.92	1.09
DIET x SHOCK	2	.19	.22
TIME x DIET x SHOCK	4	.13	.16
WITHIN CELLS	126	.84	

** $p<.01$

* $p<.05$

APPENDIX 3

Experimental Data Showing Rumenal and Glandular pH Measurements and Ulceration Ratings in all Experimental and Control Groups

TABLE 1

RUMENAL ULCERATION RATINGS FOR THE SOLID DIET CONTROL GROUPS

Subject/Group	48 hours	72 hours	96 hours
1	0	0	0
2	0	0	0
3	0	0	0
4	0	0	0
5	0	0	0
6	0	0	0
7	0	0	0
8	0	0	0
Mean:	0	0	0

TABLE 2

RUMENAL AND GLANDULAR pH MEASUREMENTS FOR THE SOLID DIET CONTROL GROUPS

Subject/Group	48 hours		72 hours		96 hours	
	rumen	body	rumen	body	rumen	body
1	2.5	3.5	3.0	3.5	0.5	3.5
2	0.5	4.0	0.5	3.5	3.0	4.0
3	3.0	4.0	3.0	3.5	3.0	4.0
4	0.5	3.5	3.0	3.5	3.0	4.0
5	2.5	3.5	3.0	3.5	3.0	3.5
6	3.0	4.0	0.5	3.5	3.0	3.5
7	3.0	4.0	3.0	3.5	3.0	4.0
8	0.5	3.5	3.0	4.5	3.0	4.0
Mean:	1.9	3.7	2.3	3.6	2.6	3.8

TABLE 3

RUMENAL ULCERATION RATINGS FOR THE LIQUID DIET CONTROL GROUPS

Subject/Group	48 hours	72 hours	96 hours
1	0	0	1
2	0	0	1
3	0	2	2
4	0	0	1
5	0	2	1
6	0	1	1
7	1	0	1
8	0	0	1
Mean:	.13	.63	1.1

TABLE 4

RUMENAL AND GLANDULAR pH MEASUREMENTS FOR THE LIQUID DIET CONTROL GROUPS

Subject/Group	48 hours		72 hours		96 hours	
	rumen	body	rumen	body	rumen	body
1	0.5	0.5	3.0	4.0	3.0	4.5
2	3.0	3.0	0.5	3.5	0.5	3.5
3	0.5	0.5	3.0	4.5	0.5	3.5
4	0.5	0.5	0.5	3.5	0.5	3.5
5	0.5	3.5	3.0	4.0	1.0	3.5
6	0.5	3.5	1.0	3.5	0.5	3.5
7	0.5	4.0	0.5	3.5	0.5	3.5
8	0.5	3.5	0.5	3.5	0.5	3.5
Mean:	0.8	2.4	1.5	3.8	0.8	3.6

TABLE 5

RUMENAL ULCERATION RATINGS FOR THE AD LIB CONTROL GROUPS

Subject/Group	48 hours	72 hours	96 hours
1	0	0	0
2	0	0	0
3	0	0	0
4	0	0	0
5	0	0	0
6	0	0	0
7	0	0	0
8	0	0	0
Mean:	0	0	0

TABLE 6

RUMENAL AND GLANDULAR pH MEASUREMENTS FOR THE AD LIB CONTROL GROUPS

Subject/Group	48 hours		72 hours		96 hours	
	rumen	body	rumen	body	rumen	body
1	3.5	3.5	3.5	4.0	3.0	3.5
2	3.0	3.5	4.0	3.5	3.0	4.0
3	4.0	3.5	4.0	3.5	3.0	3.5
4	4.0	3.5	4.0	3.5	3.0	4.0
5	3.5	3.5	3.0	3.5	3.5	3.5
6	4.0	3.5	3.0	3.5	4.0	3.5
7	4.0	3.5	3.0	3.5	4.0	3.5
8	3.0	4.0	3.0	4.0	4.0	4.0
Mean:	3.6	3.6	3.4	3.8	3.4	3.6

TABLE 7

RUMENAL ULCERATION RATINGS FOR THE SOLID DIET PREDICTABLE SHOCK GROUPS

Subject/Group	48 hours	72 hours	96 hours
1	0	0	0
2	0	0	0
3	0	0	0
4	0	0	0
5	0	0	0
6	0	0	0
7	0	0	0
8	0	0	0
Mean:	0	0	0

TABLE 8

RUMENAL AND GLANDULAR pH MEASUREMENTS FOR THE SOLID DIET PREDICTABLE SHOCK GROUPS

Subject/Group	48 hours		72 hours		96 hours	
	rumen	body	rumen	body	rumen	body
1	0.5	3.0	3.0	4.0	3.0	4.0
2	1.0	3.5	3.0	4.0	3.0	4.0
3	3.0	4.0	3.0	4.0	3.0	4.0
4	1.0	4.5	3.0	4.0	3.0	3.5
5	3.0	4.0	3.0	3.5	3.0	4.0
6	3.0	4.0	1.5	4.0	3.0	3.5
7	3.0	4.0	3.0	4.0	1.0	3.5
8	3.0	4.0	3.0	4.0	3.0	4.0
Mean:	2.1	3.8	2.8	3.9	2.6	3.7

TABLE 9

RUMENAL ULCERATION RATINGS FOR THE SOLID DIET UNPREDICTABLE SHOCK GROUPS

Subject/Group	48 hours	72 hours	96 hours
1	0	0	0
2	0	0	0
3	0	0	1
4	0	0	0
5	0	0	1
6	0	0	0
7	0	1	1
8	0	1	0
Mean:	0.0	.25	.38

TABLE 10

RUMENAL AND GLANDULAR pH MEASUREMENTS FOR THE SOLID DIET UNPREDICTABLE SHOCK GROUPS

Subject/Group	48 hours		72 hours		96 hours	
	rumen	body	rumen	body	rumen	body
1	3.0	4.0	3.0	4.0	3.0	4.0
2	3.0	4.0	3.0	4.0	3.0	4.0
3	3.0	3.5	3.0	3.5	2.5	3.5
4	0.5	3.5	3.0	4.0	3.0	3.5
5	3.0	4.0	3.0	4.0	1.5	4.0
6	3.0	4.0	3.0	4.0	2.5	4.0
7	1.0	3.5	0.5	4.0	3.0	4.0
8	3.0	4.0	1.0	4.0	3.0	4.0
Mean:	2.4	3.8	2.4	3.9	2.7	3.9

TABLE 11

RUMENAL ULCERATION RATINGS FOR THE LIQUID DIET PREDICTABLE SHOCK GROUPS

Subject/Group	48 hours	72 hours	96 hours
1	0	0	1
2	0	1	1
3	0	1	1
4	0	0	1
5	0	1	1
6	1	0	1
7	0	1	1
8	0	0	1
Mean:	.13	.50	1.0

TABLE 12

RUMENAL AND GLANDULAR pH MEASUREMENTS FOR THE LIQUID DIET PREDICTABLE SHOCK GROUPS

Subject/Group	48 hours		72 hours		96 hours	
	rumen	body	rumen	body	rumen	body
1	2.5	3.5	3.0	3.5	1.5	3.5
2	1.5	3.5	1.5	3.5	2.0	3.5
3	1.0	0.5	1.0	4.5	1.5	4.0
4	1.5	0.5	3.0	4.0	1.0	3.5
5	1.0	3.5	1.0	3.5	0.5	3.5
6	0.5	4.0	3.0	3.5	1.5	4.0
7	1.5	3.5	0.5	3.5	1.0	3.5
8	1.5	4.0	3.0	4.0	1.0	4.0
Mean:	1.4	2.8	2.0	3.8	1.2	3.7

TABLE 13

RUMENAL ULCERATION RATINGS FOR THE LIQUID DIET UNPREDICTABLE SHOCK GROUPS

Subject/Group	48 hours	72 hours	96 hours
1	0	2	1
2	0	2	1
3	1	2	1
4	1	1	1
5	0	2	2
6	0	1	1
7	1	1	1
8	1	1	1
Mean:	.50	1.5	1.2

TABLE 14

RUMENAL AND GLANDULAR pH MEASUREMENTS FOR THE LIQUID DIET UNPREDICTABLE SHOCK GROUPS

Subject/Group	48 hours		72 hours		96 hours	
	rumen	body	rumen	body	rumen	body
1	1.0	4.0	1.5	3.5	1.0	4.0
2	3.0	4.0	1.5	4.0	0.5	3.5
3	0.5	4.0	0.5	4.0	1.0	4.0
4	2.0	3.5	3.0	4.0	1.5	4.0
5	3.0	3.5	1.0	4.0	0.5	3.5
6	3.0	4.0	0.5	3.5	1.0	3.5
7	1.0	4.0	1.5	3.5	1.5	4.0
8	0.5	4.0	2.0	3.5	1.0	3.5
Mean:	1.8	3.9	1.4	3.8	1.0	3.8

APPENDIX 4

Summary of Analysis of Variance of Rumenal Ulceration

TABLE 1

SUMMARY OF 3 x 2 x 3 ANALYSIS OF VARIANCE ON

RUMENAL ULCERATION: TIME, DIET, AND SHOCK

Source	df	MS	F
TIME	2	2.97	19.93*
DIET	1	15.99	107.52*
SHOCK	2	1.80	12.09*
TIME x DIET	2	1.64	11.06*
TIME x SHOCK	4	.28	1.87
DIET x SHOCK	2	.27	1.82
TIME x DIET x SHOCK	4	.35	2.38
WITHIN CELLS	126	.15	
TOTAL	143		

* $p < .001$

APPENDIX 5

Rumenal Ulceration Means and Percent

Rumenal Ulceration Incidence in all Experimental and Control Groups

TABLE 1

MEAN RUMENAL ULCERATION RATING IN ALL EXPERIMENTAL AND CONTROL GROUPS

TIME/GROUP	CONTROL			LIQUID		SOLID	
	ad lib	liquid	solid	predictable shock	unpredictable shock	predictable shock	unpredictable shock
48 hours	0.0	.13	0.0	.13	.50	0.0	0.0
72 hours	0.0	.63	0.0	.50	1.50	0.0	.25
96 hours	0.0	1.10	0.0	1.00	1.10	0.0	.38

TABLE 2

PERCENT RUMENAL ULCERATION INCIDENCE IN ALL EXPERIMENTAL AND CONTROL GROUPS

TIME/GROUP	CONTROL			LIQUID		SOLID	
	ad lib	liquid	solid	predictable shock	unpredictable shock	predictable shock	unpredictable shock
48 hours	0.0	12.5	0.0	12.5	50.0	0.0	0.0
72 hours	0.0	37.5	0.0	50.0	100.0	0.0	25.0
96 hours	0.0	100.0	0.0	100.0	100.0	0.0	37.5

APPENDIX 6

Photographs of Stomachs of Representative Subjects from those Groups Which Developed Ulceration

FIGURE 1

The stomach of a rat that had been exposed to the solid diet + unpredictable shock condition for 72 hr. Note the absence of rumenal ulceration relative to the stomach of a rat in the liquid diet condition (Figure 2).


SSh C5-72

FIGURE 2

The stomach of a rat that had been exposed to the liquid diet + unpredictable shock condition for 72 hr. Compare this stomach with that of Figure 1 and note the severe rumenal ulceration.


L SK65-72

FIGURE 3

The stomach of a rat that had been exposed to the solid diet + unpredictable shock condition for 96 hr. Note the absence of rumenal ulceration relative to the stomach of a rat in the liquid diet condition (Figure 4).

SShCS-96

FIGURE 4

The stomach of a rat that had been exposed to the liquid diet + unpredictable shock condition for 96 hr. Note the rumenal ulceration and compare this stomach with that shown in Figure 3.


LSACS-96

FIGURE 5

The stomach of a rat that had been exposed to the liquid diet control condition for 48 hr. Note the small number of rumenal ulcers relative to the liquid diet control stomachs shown in Figures 6 and 7.


LC-18

FIGURE 6

The stomach of a rat that had been exposed to the liquid diet control condition for 72 hr. Note the severe rumenal ulceration and compare this stomach to that shown in Figure 5.

FIGURE 7

The stomach of a rat that had been exposed to the liquid diet control condition for 96 hr. Note the severe rumenal ulceration relative to the liquid diet control stomachs at shorter time periods (Figures 5 and 6).

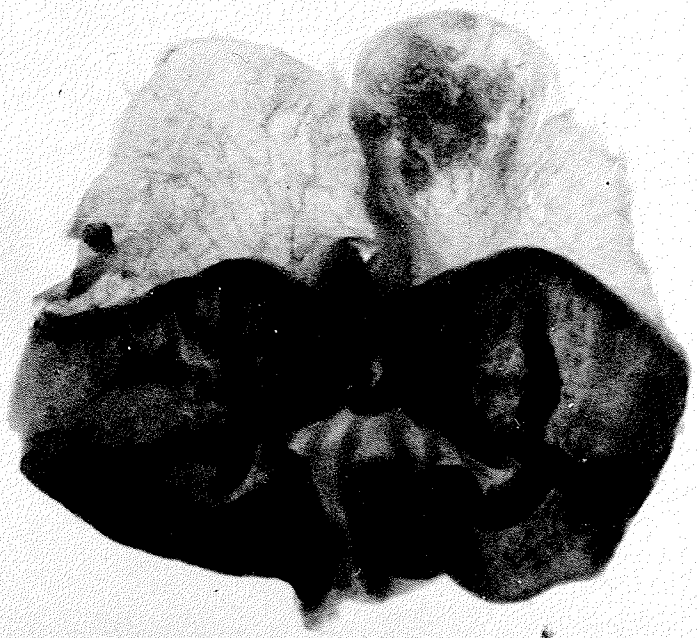
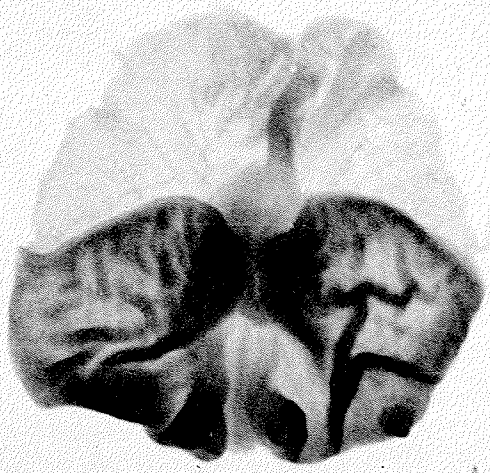

LC-96

FIGURE 8

The stomach of a rat that had been exposed to the liquid diet + predictable shock condition for 48 hr. Note the small number of rumenal ulcers and the similarity of this stomach to that shown in Figure 11.



L Sh CS^{+} - 9c

FIGURE 9

The stomach of a rat that had been exposed to the liquid diet + predictable shock condition for 72 hr. Note the similarity between this stomach and that shown in Figure 2.


ESHCS+-72

FIGURE 10

The stomach of a rat that had been exposed to the liquid diet + predictable shock condition for 96 hr. Note the similarity of this stomach to that shown in Figure 4.


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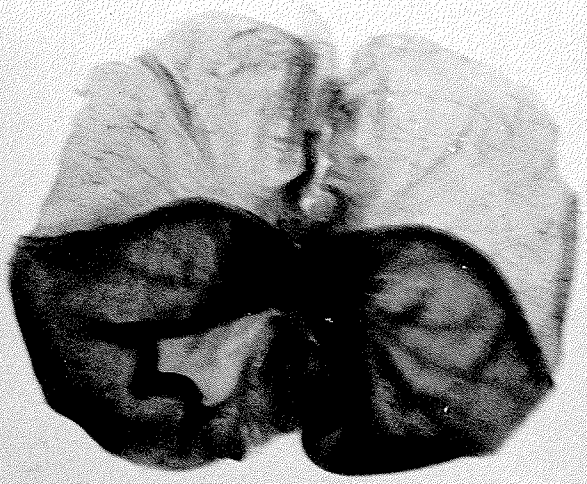
FIGURE 11

The stomach of a rat that had been exposed to the liquid diet + unpredictable shock condition for 48 hr. Note the similarity of this stomach to that shown in Figure 8.



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APPENDIX 7

Daily Liquid and Sold Sucrose Intake in all Experimental and Control Groups

TABLE 1

TOTAL LIQUID SUCROSE INTAKE IN THE 48 HOUR GROUPS (ml)

Subject/Group	LIQUID			SOLID		
	control	predictable shock	unpredictable shock	control	predictable shock	unpredictable shock
1	97	104	102	104	99	96
2	79	79	107	91	103	107
3	101	84	92	101	92	88
4	113	85	86	99	88	121
5	88	91	124	81	96	113
6	120	156	139	117	117	84
7	99	133	121	84	113	118
8	114	98	71	113	90	46
Mean:	101.38	101.38	105.38	98.75	99.75	96.63

TABLE 2

TOTAL LIQUID SUCROSE INTAKE IN THE 72 HOUR GROUPS (ml)

Subject/Group	LIQUID			SOLID		
	control	predictable shock	unpredictable shock	control	predictable shock	unpredictable shock
1	198	130	220	170	137	194
2	172	211	186	140	153	162
3	168	215	187	188	144	155
4	184	214	164	162	151	188
5	181	215	215	153	192	208
6	171	128	223	182	195	216
7	152	173	145	138	191	179
8	177	223	117	172	206	229
Mean:	175.38	188.63	182.13	163.13	171.13	191.38

TABLE 3

TOTAL LIQUID SUCROSE INTAKE IN THE 96 HOUR GROUPS (ml)

Subject/Group	LIQUID			SOLID		
	control	predictable shock	unpredictable shock	control	predictable shock	unpredictable shock
1	264	260	214	199	302	196
2	238	270	214	269	153	94
3	321	232	240	268	207	280
4	344	286	198	283	239	260
5	256	207	222	184	293	281
6	228	292	287	187	224	274
7	269	259	238	224	297	239
8	221	182	180	256	313	250
Mean:	267.63	248.50	224.13	233.75	253.50	234.25

TABLE 4

TOTAL SOLID SUCROSE INTAKE IN THE 48 HOUR GROUPS (gr)

Subject/Group	solid control	solid-predictable shock	solid-unpredictable shock
1	130	11	32
2	98	16	17
3	96	18	35
4	174	13	17
5	120	49	13
6	124	38	24
7	67	33	14
8	134	34	15
Mean:	117.88	26.50	20.88

TABLE 5

TOTAL SOLID SUCROSE INTAKE IN THE 72 HOUR GROUPS (gr)

Subject/Group	solid control	solid-predictable shock	solid-unpredictable shock
1	141	52	24
2	182	60	35
3	98	63	48
4	180	60	83
5	175	45	26
6	175	49	25
7	215	21	63
8	129	24	44
Mean:	161.88	46.75	43.50

TABLE 6

TOTAL SOLID SUCROSE INTAKE IN THE 96 HOUR GROUPS (gr)

Subject/Group	solid control	solid-predictable shock	solid-unpredictable shock
1	147	40	28
2	179	138	89
3	145	96	28
4	118	111	55
5	167	28	28
6	123	121	48
7	266	103	25
8	270	99	69
Mean:	176.88	92.00	46.25