

EFFECTS ON HUMAN GASTRIC ACID SECRETION OF
BIOFEEDBACK TRAINING AND RELAXATION TRAINING:
TWO STUDIES USING TELEMETRIC MEASUREMENTS

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ABSTRACT

Effects on Human Gastric Acid Secretion of Biofeedback Training and Relaxation Training: Two Studies Using Telemetric Measurements

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This thesis was designed to study the efficacy of two common treatment procedures on gastric acid pH. In study I, four male subjects were given five to nine biofeedback training sessions in order to see if they could learn to voluntarily raise and lower their gastric acid pH. Subjects' pH was determined telemetrically. Only a few appropriate pH changes occurred after training. These were not due to the biofeedback per se since no development of learning was seen over the course of the training sessions. Explanations relating to the adequacy of the feedback and to the cognitions and expectations of subjects and experimenter were offered to account for the results. In study II, two of the subjects underwent progressive relaxation training to see the effect of muscle relaxation on gastric acidity. Results showed less acid pre to post in both control (reading) and relaxation conditions but this decrease in acid output over a period of time appeared to be somewhat larger after the control period. It was speculated that the parasympathetic vagus nerve may have been less inhibited when

sympathetic arousal decreased following the relaxation exercises. Consistently higher pH peaks (less acid) were noted only within 45 seconds following the exercises possibly due to more rapid gastric emptying as a result of contracting and relaxing stomach muscles.

It is suggested that the telemetric method used in the present study may not be useful for biofeedback training but could be used in other psychological studies to explore cognitive and affective influences on acidity since this technique is much less aversive than other forms of acid measuring equipment.

ACKNOWLEDGEMENTS

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TABLE OF CONTENTS

	Page
GENERAL INTRODUCTION	1
Secretion of Acid by the Stomach	1
The Dependent Measure--pH	2
Methods of Assessing Acidity	3
Cephalic Influences on Gastric Acid Secretion	5
INTRODUCTION TO STUDY I - BIOFEEDBACK TRAINING	8
Research on Biofeedback Training of Gastric Acidity	9
Evaluation of Heidelberg Technique	14
Hypotheses Underlying this Study	16
Method--Subjects	16
Apparatus	17
Procedure and Design	18
Results	22
Discussion	39
INTRODUCTION TO STUDY II - RELAXATION TRAINING	49
Method--Subjects	54
Apparatus and Procedure	54
Results	55
Discussion	63
SUMMARY	69
REFERENCES	71
REFERENCE NOTES	76
APPENDICES	77

GENERAL INTRODUCTION

The purpose of this study is to examine some behavioral approaches to changing levels of gastric acidity using healthy subjects who have no history of peptic ulcer disease. The first phase of the study was designed to ask whether awareness of one's level of gastric acidity, through a technique of bio-feedback training over several sessions, may enable a subject to voluntarily effect changes in acidity after the training is completed. The second phase of the research deals with the effects of progressive muscle relaxation training on gastric acidity.

Secretion of Acid by the Stomach

Secretion of acid by the stomach is stimulated in two major ways. The first, called the cephalic phase, refers to the effect of psychological influences or of the sight, smell and taste of food on gastric secretion (Walsh, 1973). The stimulus acting on the brain is mediated through the autonomic nervous system, in particular the vagus nerve, which has a direct effect on the acid-producing parietal cell, and an indirect effect by stimulating release of the hormone gastrin which in turn stimulates the parietal cell to release acid.

The second major phase of acid secretion is the gastric phase. This phase is initiated by stimuli arising in the stomach such as food, alkalization of stomach contents, or gastric distension. These stimulate locally the release of the hormone gastrin which enters the circulation and returns to stimulate the acid cells of the stomach to release acid (Wolf & Welsh, 1972).

The present study is concerned with influences that stimulate or inhibit the cephalic phase of acid secretion.

The Dependent Measure--pH

The degree of acidity in the stomach can be measured by various techniques and reported in several ways (Davenport, 1977). For the purpose of this experiment, degree of acidity will be measured in pH units and changes will be assessed relative to the basal acid level at the beginning of each experimental session since a subject's pH is expected to vary from day to day. pH is the logarithm of the reciprocal of the hydrogen ion concentration in a solution and is expressed by the formula $pH = \log \frac{1}{(H^+)}$. Thus an increase in pH reflects a decrease in acidity and a decrease in pH represents an increase in acidity (Hills, 1973).

At the present time little is known about methods of inhibition of gastric acidity other than through pharmacological

or surgical means. This is of clinical importance since much evidence (e.g. Menguy, 1966; Isenberg, 1973; Eisenberg, 1977) suggests that gastric acid plays an important role in the development of peptic ulcer disease. Variations in volume and concentration of hydrogen ion appear to be key factors in characterizing gastric juice as a corrosive medium (Eisenberg, 1977). Therefore it would seem important to explore methods of influencing the pH of gastric acidity other than through medication or surgery. The digestive enzyme pepsin is active at pH 1.5-3.5 and all treatment approaches are directed at raising the pH of gastric acid above these levels (Hardy, 1958; Silen & Skillman, 1976). It is recognized that the effects of the manipulations carried out in this study may be different in subjects who suffer from ulcer disease. However, it is important to first ask some preliminary theoretical questions of healthy subjects.

Methods of Assessing Acidity

Early methods of studying the cephalic phase of acid secretion as an isolated phenomenon involved direct observation using human subjects or dogs with esophageal and gastric fistulae. When the effects of the sight, smell, chewing of food or sham feeding were studied in this manner, gastric acid secretion was directly observed to have increased (Walsh, 1973).

Presently several methods are available to assess the degree of acidity in the stomach. Samples of gastric juice may be aspirated through a swallowed gastric tube. The retrieved acid is titrated with an alkaline substance to a specific pH end point. This allows for measurement of both the volume and concentration of gastric juice over a specified period of time. A modification of this technique is to have the subject swallow a glass pH electrode and a tube, and to titrate within the stomach with an alkali, again to a specific pH end point (Isenberg, 1973).

Telemetric methods have been devised whereby a capsule can be swallowed, instead of a glass electrode, in order to measure pH within the stomach. The telemetering capsule is less aversive to swallow compared to the glass electrode (Noller, 1960). Since psychological or cephalic effects could result simply from the aversive nature of the first two methods, the elimination of the discomfort of intubation is an important advantage of the telemetric method (Connell & Waters, 1964). This is especially important in a research setting where several trials may be required of the same subject. Furthermore, the telemetric technique involves less manipulation of the stomach. This minimizes gastric phase effects thus allowing for better study of the cephalic phase of acid secretion.

Cephalic Influences on Gastric Acid Secretion

The literature relevant to behavioral manipulations intending to study cephalic effects will be discussed in a section specifically devoted to research on biofeedback training of gastric acidity.

The effects of emotions on gastric acid secretion were studied by Beaumont in 1833 (Mahl, 1950). Mahl cites Beaumont to report that the acid of his patient was inhibited during fear. Wolf and Wolff (1942) reported that their patient inhibited gastric acid secretion during periods of fear and sadness and increased secretion during periods of resentment, hostility and anxiety. Cannon (1909), on the other hand, reported that "vexation . . . is accompanied by a failure of secretion." Using the method of direct observation because these patients had gastric fistulae, results of these early studies suggested that emotions or feelings had some influence on gastric secretion, motility and vascularity. Chappell and Stevenson (1932) reported that six weeks of daily group psychological training given to subjects with diagnosed peptic ulcer disease led to a significant reduction of symptoms on a three year follow up. Experimental subjects were taught much of the "psychology of learning and forgetting" and methods of thought control. Control subjects were not given this training.

Somewhat later studies tried to determine the type of stress that would provoke ulceration. Wolf (1956) reported that British civilians during World War II had an increased incidence of ulcers, but during the same period of time, the ulcer incidence of prisoners of war decreased. This led to the suggestion that variables such as uncertainty and threats of bombing were more stressful than the more predictable life in a prisoner of war camp. Several studies have been carried out on rats and monkeys attempting to show the influence of predictability, uncertainty or conflict on gastric acidity (Brady, 1958; Glavin & Mikhail, 1976; Gliner, 1972; Moot, Cebulla & Crabtree, 1970; Paré & Livingston, 1973; Seligman & Meyer, 1970; Weiss, 1970). The results of these studies have been conflicting. Rats are reported to demonstrate many strain, sex and social setting differences with regard to ulceration. There has been much disagreement in the literature particularly over the concept of 'executive capacity' or the influence on acidity of the ability to control a response or avoid its consequences (Mikhail, Kamaya & Glavin, 1978).

Mahf (1950) studied the gastric acidity of undergraduate students during exam and control periods and reported a significantly higher degree of acidity during the exam period. He furthermore felt, on the basis of his studies with dogs, that chronic fear or anxiety leads to an increase in secretion, while during acute fear situations, secretion

does not increase (Mahl, 1949).

Although many studies have been done which suggest that the psychological status of the individual plays a role in the production and inhibition of gastric acidity, the nature of this role is not yet understood (Wolf, Note 1). It is the purpose of the present study to explore the effect of some behavioral manipulations on the pH of gastric juice and in so doing to attempt to further clarify some of the factors that may affect stomach acidity.

INTRODUCTION TO STUDY I - BIOFEEDBACK TRAINING

The first phase of this study was planned to examine the effect of biofeedback training on a subject's ability to control the pH of his gastric acidity.

Biofeedback training is a form of operant conditioning; it is also called instrumental learning. The traditional view about the difference between operant conditioning and classical conditioning is that operant conditioning is mediated by the central nervous system acting on skeletal responses and that classical Pavlovian conditioning is mediated by the autonomic nervous system acting on smooth muscle (Kling, 1971). Miller and Carmona (1967), however, showed that autonomic responses in dogs, such as salivation, could be modified through instrumental learning.

Traditionally, individuals are not viewed as normally able to control their autonomic responses. Biofeedback attempts to teach subjects to control specific visceral or muscular activities by providing them with feedback of small changes in these activities. This feedback is viewed as the reward or reinforcing stimulus of the operant conditioning paradigm (Shapiro & Schwartz, 1972). Operant conditioning theory stipulates that behavior followed by reward increases, and

behavior followed by punishment decreases (Skinner, 1969).

If pH of gastric acidity is used as feedback, this theory would predict that when a subject receives pH feedback that is appropriate to his instruction, this would be rewarding to him.

This response should then increase. Similarly, pH feedback not appropriate to instruction would be non-rewarding and should therefore decrease in frequency.

Research on Biofeedback Training of Gastric Acidity

Attempts have been made in recent years to teach voluntary control of gastric acidity with biofeedback training (Hubel, 1974). Moore and Shenkenberg (1974) reported that one healthy subject, during four training sessions, was able, with feedback, to increase or decrease gastric acid in correct response to instruction. The feedback was given at 10-minute intervals on samples of acid drawn through a nasogastric tube. This method has been criticized since it may require too much time in order to give subjects adequate information on the state of their acidity (Welgan, 1974). Furthermore, the effects of anticipated feeding had previously been studied with this same subject. It is not clear whether the correct responses were due to his biofeedback training or to the earlier classical conditioning. The subject was not able to make the correct response when biofeedback was withdrawn.

Welgan (1974) studied 10 patients with duodenal ulcers, using a nasogastric tube and measuring pH in vitro. His subjects were given biofeedback training in one 30-minute session. They were instructed only to raise their pH (lower acidity)-- Welgan did not attempt to train his subjects to lower pH (raise acidity). He reported that the subjects were able to significantly increase pH and decrease the volume of acid secreted. Unfortunately, both the experimental design and data analysis of this study are questionable. Welgan did not test the subjects' ability to increase pH following the training when biofeedback was withdrawn.

Whitehead, Renault and Goldiamond (1975) reported that three out of four healthy subjects, when given visual gastric pH feedback and financial reward, increased gastric acidity in correct response to instruction. As well, the subjects were reported to have reduced high rates of secretion but not below basal levels. The authors recognized that the high level of acid produced by their subjects may have been influenced by the repeated infusion of titrant directly into the stomach. They used an intragastric electrode to measure the pH and then measured the amount of titrant required to neutralize pH to 7. This amount was converted to milliequivalents and divided by the time elapsed before pH decreased to 2 to give a rate of acid secretion over time. This method has been criticized

because the continual repeated manipulation throughout the experiment of pH from 2 to 7 could trigger the gastric phase of acid secretion. Thus efforts to measure only cephalic phase effects would be confounded (Gorman, Note 2).

Gorman (Note 2) carried out a similar study and trained three healthy subjects using biofeedback of the rate of acid secretion and financial reward based on performance. The subjects all showed some ability to alter gastric secretion in the appropriate direction. His method involved maintaining intragastric pH at a steady state (3.5). He measured the amount of titrant required to maintain that state. Because stabilizing this end point required adequate mixing of stomach contents, Gorman's technique involved infusion of substantial amounts of water into the stomach. It is known that gastric distension stimulates the gastric phase of acid secretion (Wolf & Welsh, 1972). Indeed, Gorman's subjects' rates of secretion were higher than those reported using aspiration techniques. Thus this method as well produced a mixture of gastric and cephalic effects.

Another method which did not involve infusion of either titrant or large amounts of water into the stomach was used in a pilot biofeedback study (Sigman, Nowlis & Borzone, Note 3). This study attempted to train one healthy subject to increase and decrease the pH of his gastric acidity through continuous

pH feedback. An intragastric glass electrode continuously measured stomach pH. This information was immediately relayed to the subject in the form of auditory tone feedback. In addition, stomach contents were withdrawn frequently in order that the pH feedback would reflect the current acid pool. This was done because it is difficult to assess the rate at which acid drains out of the stomach. This seemed to best control for the variable of gastric emptying. In vitro analyses provided confirmation of the intragastric pH readings.

Some learning appeared to develop over the three training sessions and was demonstrated in a session shortly following training when feedback was not available. The subject, however, was unable to control his pH in both directions in a non-feedback session held one month following training. Although the method seemed technically adequate for purposes of biofeedback training, the subject found that swallowing the tube and retaining it in place for 90 minutes was very uncomfortable. It was felt that the aversive nature of the method could affect the training or gastric secretion.

The above studies lend tentative support to the concept that biofeedback training may enable subjects to achieve control of their gastric acidity. In addition, support for the transfer of visceral learning to a non-feedback situation is partially suggested by the single subject pilot study (Sigman

et al., Note 3) and by Weiss and Engel (1971) who showed that patients were able to transfer control of cardiac rhythm, learned with feedback, to non-feedback conditions.

The mechanism that mediates the subjects' responses to biofeedback is not understood (Kimmel, 1967; Katkin & Murray, 1968; Crider, Schwartz & Shnidman, 1969; Shapiro & Schwartz, 1972). Is the trainer's goal to teach an association between a particular 'feeling-state' in the stomach and pH? Are certain thoughts and feelings related to the pH of high and low acidity? Debriefing of the subject used in the pilot study suggested that cognitive and emotional mediation play an important role in effecting changes in gastric acidity. It also suggested that the subject needs a stockpile of situations or thoughts that he can use when he is instructed to increase or decrease acidity in the non-feedback situations. The subject reported that situations at times lost their potency for effecting changes and that he had to change his focus from one experience or situation to another at different times in order to maintain control. It would seem then that the more training a subject is given the more opportunities there would be to learn either the association of particular acid-related feelings with increased and decreased pH respectively, or the association of cognitive or emotional factors with the pH. Which of these associations the subject makes will not be

tested by this experiment.

It was the purpose of Study I to allow subjects several biofeedback training periods in order to optimize the chance that they would develop the ability to increase and decrease their pH upon instruction. More training would also increase the chances that any learning would transfer to the non-feedback condition.

Each subject underwent 10 to 14 experimental sessions which included baseline control, pre-training, training, and post-training. It was thus important to choose the least aversive method of monitoring gastric acidity that would still allow biofeedback information to the subject. A radio telemetric technique employing the Heidelberg Capsule appeared to fulfill these criteria and was used in this study.

Evaluation of Heidelberg Technique

The Heidelberg Capsule is a small pH telemetry transmitter enclosed in a plastic casing which can be swallowed without difficulty. It continuously transmits pH values from the stomach to a receiver where they can be read off the panel meter.

Evaluations of the Heidelberg system have been carried out (Yarbrough, McAlhany, Cooper & Weidner, 1969; Andres & Bingham, 1970; Johannesson, Magnusson, Sjoberg & Skov-Jensen, 1972) and the capsule method is reported to give results

comparable to those obtained by the standard aspiration technique causing very little discomfort to the patients. The pH response to injection of dilute acid or bicarbonate is immediate and appropriate (Watson & Paton, 1965). Connell and Waters (1964) reported that readings established by the pH telemetering capsule did not vary from the pH recorded by a glass electrode by more than 0.5 pH unit. It is recognized that this disadvantage of the technique may prevent adequate feedback if subjects have a low baseline pH range. Because of the logarithmic nature of a pH measurement, a large change in acidity will show only a small change in pH when the pH is in the 1 to 2 range. Therefore, a modification of Noller's (Note 4) method is used in some of the subjects' biofeedback training and in the relaxation training phase of this research. This involves giving the subjects a constant amount of alkali (5 ml 0.1N sodium bicarbonate) in order to bring the pH to a higher range (4-6) where subsequent changes of acidity can be more readily measured. Thus, in addition to recording, for example, a subject's pH before and after a behavioral manipulation, a neutralization curve indicates the concentration of acid in the stomach at the moment of swallowing the alkaline substance and the rate of secretion during the following minutes. The single doses of sodium bicarbonate are felt to be only a minimal gastric intervention that does not confound

the cephalic phase of acid secretion. Furthermore, the questions that are being asked, particularly with regard to the effects of relaxation training can be more satisfactorily answered using this measure of acid output in addition to pH.

Hypotheses Underlying This Study

For the purpose of this study it is hypothesized that subjects who are not made aware of the pH of their gastric secretions will be unable to control their pH upon instruction, i.e. appropriate changes will not be made in the pre-training sessions. It is further hypothesized that, with adequate feedback, bidirectional control of gastric acid pH can be learned. Finally, it is expected that if a subject learns to control acidity through biofeedback training, he will be able to make appropriate changes in the post-training sessions in the absence of pH feedback information.

Method

Subjects

The subjects were 4 healthy male volunteers ranging in age from 23 to 46 years. G.G. and D.S. were naive to a biofeedback training experience. D.N. was familiar with several modes of feedback other than pH of gastric acidity. H.S. had undergone biofeedback training of gastric pH 14 months previous to this experiment. Bidirectional control had not been

maintained, however, in a non-feedback session held one month following his training although unidirectional control was maintained.

None of the subjects had ulcer disease although G.G. reported a five year history of hyperacidity but no demonstrated ulcer. The somewhat lack of uniformity in the previous experience of subjects emanates from the difficulty in securing subjects for such experiments.

Subjects fasted for eight hours prior to each session.

Apparatus

The pH was studied by means of a Heidelberg telemetering pH sensor, Model No. HK-26 630231. The battery transmitter is encapsulated in an indigestible acrylic container, 20 x 7 mm, and weighs 1.55 g. The pH measuring cell consists of an external annular antimony electrode and an internal silver chloride electrode. The two are separated by a dialysis membrane permeable to ions but not to larger molecules. The battery was activated by immersion in .9% saline solution just before the test. The capsule was then calibrated in buffer solutions of pH 1 and 7. Buffers and rinse waters were heated to body temperature 37° C. by means of a Fisher heater Model No. H 2025-1. Silk thread (3.0) was used to tether the capsule so that it would not leave the stomach during the course of

the session. The thread was fixed to the subject's cheek with adhesive tape.

The signals emitted by the capsule were received by a special belt antenna worn around the subject's waist and were then amplified onto the attached pH meter where they could be read off the panel. pH values were continuously charted by a pen recorder incorporated in the pH meter. (Figure 1).

Procedure and Design

Prior to the first session it was explained to each subject that he would swallow an inert and non-absorbed capsule attached to a thread; that the capsule measures the degree of acidity in the stomach; and that after the experimental session, the thread would be cut and the remainder swallowed with water. Subjects were told that after the experiment the capsule could either be withdrawn by its string or the string and capsule could leave the body in the natural way within 48 hours.

Each subject came to the laboratory in the morning having fasted overnight. After the capsule was swallowed and tethered, the subject sat in a recliner chair. The room was bare except for the equipment. It had no windows. Only the experimenter was in the room with the subject. The interaction was limited to the experimental instructions except when subjects

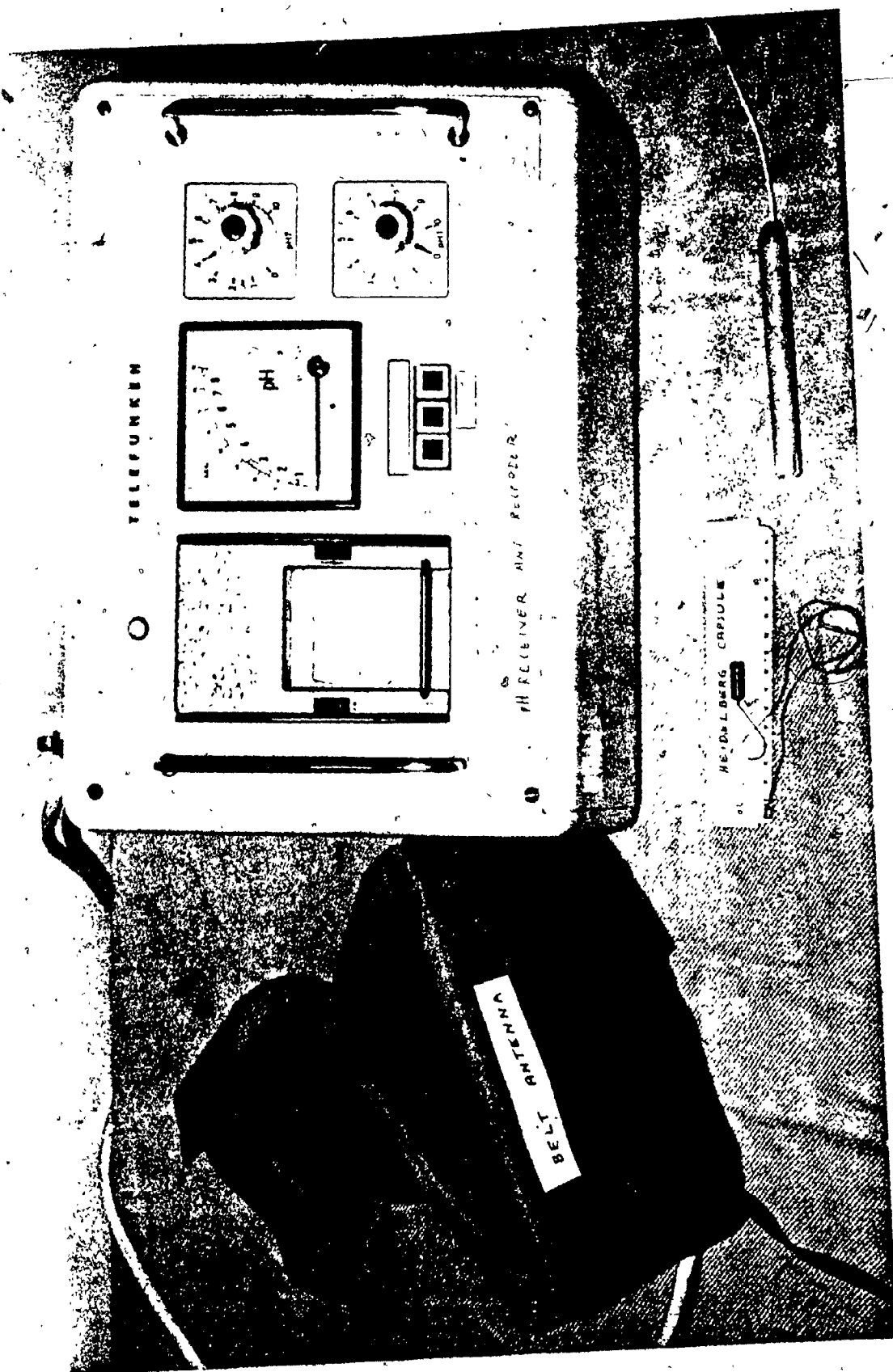


Figure 1. Belt antenna, pH receiver and recorder, and Heidelberg Capsule with attached thread.

initiated conversation or volunteered subjective reports.

Baseline control: Each of the four subjects had from two to four pure baseline sessions in order to assess temporal effects over the 75 minute interval that would be used for the experimental conditions. In addition, two subjects, D.S. and G.G., spent varying lengths of time resting in the laboratory (up to 60 minutes) before swallowing the capsule. The purpose of this was to allow assessment of any effects on pH of time spent in the laboratory prior to the sessions.

Pre-training: Each subject had a pre-training session (H.S. had two) to examine his ability to voluntarily raise or lower pH on instruction prior to receiving any feedback. At this time subjects were given the following instruction: "During these sessions you will be asked to increase or decrease acidity. What works for one person (thoughts of anger, relaxation, etc.) may not necessarily work for another person and what helps you one day may not work in the next session. Use whatever method you wish in order to follow the instructions without moving out of the chair or falling asleep." Instructions to raise pH consisted of the following: "Try to lower your acidity now by using any thoughts or feelings that you think may help you." Instructions to lower pH were: "Try to raise your acidity now by using any thoughts or feelings that you think may help you."

All pre-training, training and post-training sessions started with a 15 minute reading or resting period until the basal pH for the day had been reached. The baseline sessions demonstrated that the pH reached basal levels within this period of time. At that point, if a subject's pH was below 3.5 he was instructed to raise it. If it was above 3.5 he was asked to lower it. Instructional periods lasted 15 minutes and were followed by a 10 minute rest period.

Biofeedback training: Subjects had from five to nine training sessions with biofeedback. They received verbal feedback approximately every 30 seconds during their training, e.g. "that's good . . . moving in the right direction; acid going down (or up)"; or "there's been an increase (or decrease) in acidity . . . try to decrease (or increase) it"; or "maintaining the same level," depending upon whether pH was moving in the right direction, the wrong direction or not moving at all. Verbal instructions and feedback referred to direction of acidity rather than pH since it was more readily understood by the subjects. As well, D.S. and G.G. received some of their feedback in the form of ticks on a chart that they were able to see. The ticks showed them the level of their acidity every 30 seconds. This method was introduced after they had had three training sessions with verbal feedback. H.S. and D.N. received a form of visual feedback during the latter part of their

training also. The equipment was positioned so that they could see the pH meter themselves. In addition, they were given 5 ml 0.1N sodium bicarbonate (NaHCO_3) to neutralize their acid thus raising their pH. They were able to see the effects of the alkaline substance on their pH. In these instances, when instructed to decrease acidity, subjects were asked to keep the pH up high as long as possible after swallowing the 5 ml 0.1N NaHCO_3 . When they were to increase acidity, they were instructed to try to keep the pH low down and to counter the effects of the alkaline substance.

Post-training: Sessions without feedback were held three and seven days following training for three of the four subjects. H.S. had one post-training sessions three days after training. The instructions in these post-training sessions took the same form as those given prior to and during training but the subjects were unaware of the level of their acidity.

Results

The pre-training, training and post-training results were evaluated by two independent observers for changes appropriate to instruction. If learning appeared to have developed during the biofeedback training, the pre- and post-training results would have been compared. If minimal effects occurred during training, a liberal method of analyzing

training and post-training results would have been applied.

Baseline Sessions

Varying the length of time from 15 to 60 minutes during which D.S. and G.G. were allowed to rest in the laboratory prior to swallowing the capsule did not appear to have any effect on the ensuing 75 minutes of pH measurement. The first of their three baseline sessions each shows marked fluctuations in pH that occurred mainly during periods of tugging and subsequent discomfort with the string and conversation with the experimenter. Generally, pH during the baseline sessions for G.G., D.S. and D.N. was steady and low indicating a low basal pH for these three subjects. The basal pH of subject H.S. varied from session to session and within one of the four sessions as well. The last baseline session for each subject is shown in Figure 2.

None of the subjects showed a consistent trend over sessions for the pH either to rise or fall during the 75-minute baseline periods. The pH fluctuations that were observed in the baseline sessions occurred when there was tugging and discomfort with the string, excessive swallowing, or when the subject was reading or having conversation with the experimenter.

Pre-Training Sessions

When D.S. and G.G. were each twice instructed to raise

Smallmouth
bream
V. strabus

D.S. Aug. 10/77

Smallmouth
bream
V. strabus



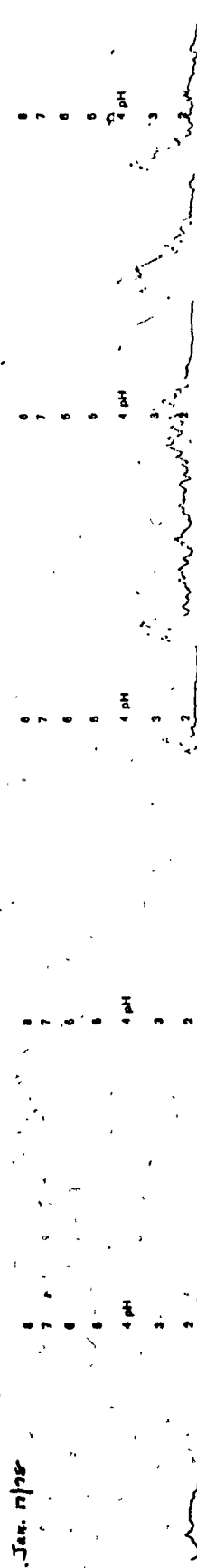
G.G. Aug. 10/77

Smallmouth
bream
V. strabus



D.N. Jan. 11/78

Smallmouth
bream
V. strabus



H.S. Mar. 21/78

Smallmouth
bream
V. strabus



Figure 2. The last baseline session for subjects D.S., G.G., D.N. and H.S. respectively.

their pH in the non-feedback session carried out prior to their biofeedback training they were unable to do so. (The pH of D.S. went from a 15 minute basal level of .5 to .25 at the end of the session and that of G.G. from 1 to .25 reflecting a trend to become more acidic [Figure 3].)

The pH of subject D.N. remained steady throughout his two instructional periods to raise pH as it did during the entire session (Figure 3).

H.S. had two non-feedback pre-training sessions because at the first instruction a highly appropriate pH response occurred. However, it was interpreted to be a result of its association with the string pulling reported by the subject at the start of the period since pH changes were not appropriate to the other instructions he received during the rest of this and during his second pre-training session (Figure 4).

Training Sessions

The pH of subject D.S. remained steady throughout each of his five biofeedback training sessions showing no appropriate response to instructions. Figure 5 shows his fifth training session.

The pH of subject G.G. remained relatively steady during his six training sessions showing only 3 appropriate pH changes out of the 13 instructions given to him. No development

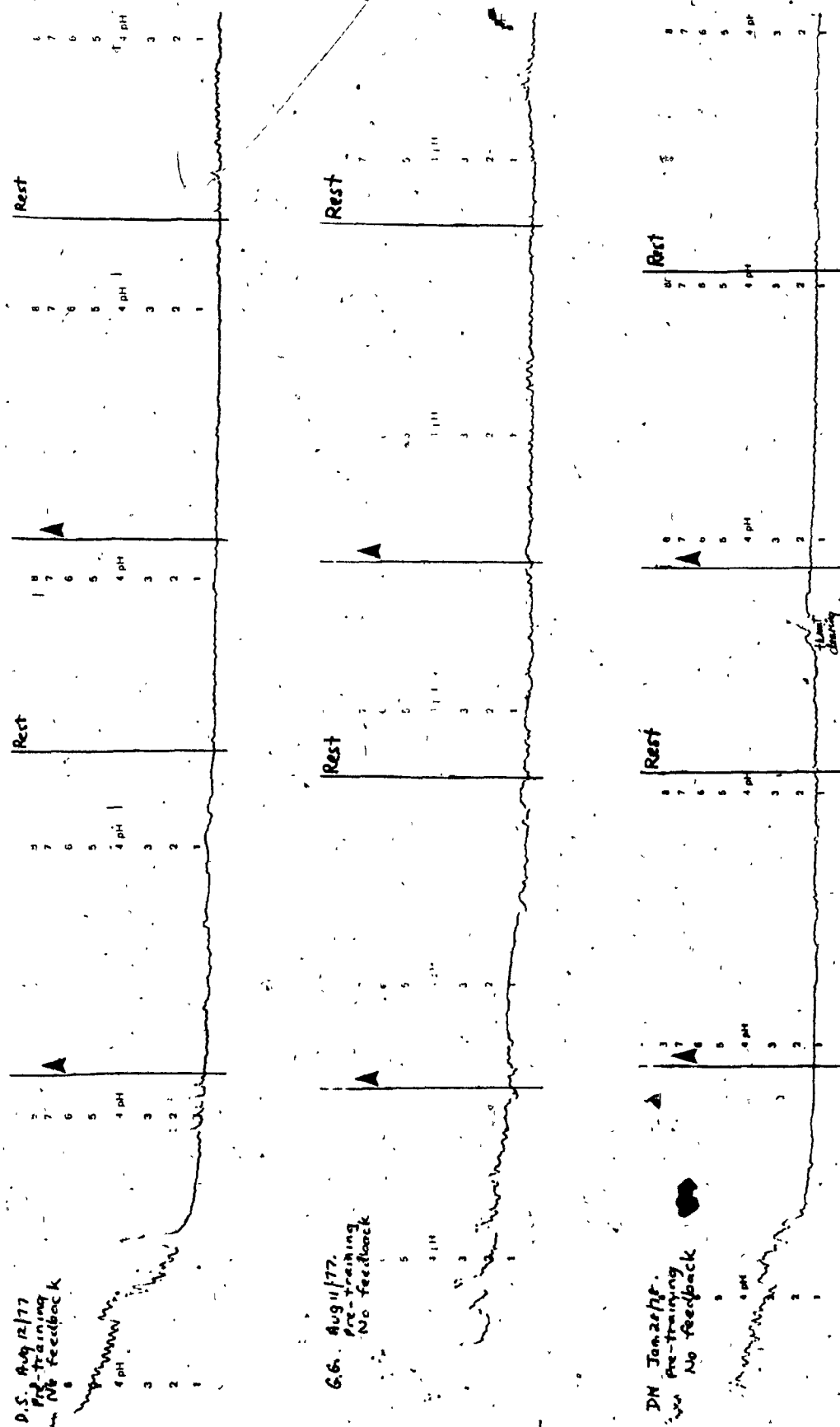


Figure 3. Pre-training sessions without feedback for subjects D.S., G.G. and D.N. Instructions were given at the vertical lines to increase pH (Δ), decrease pH (∇), or rest.

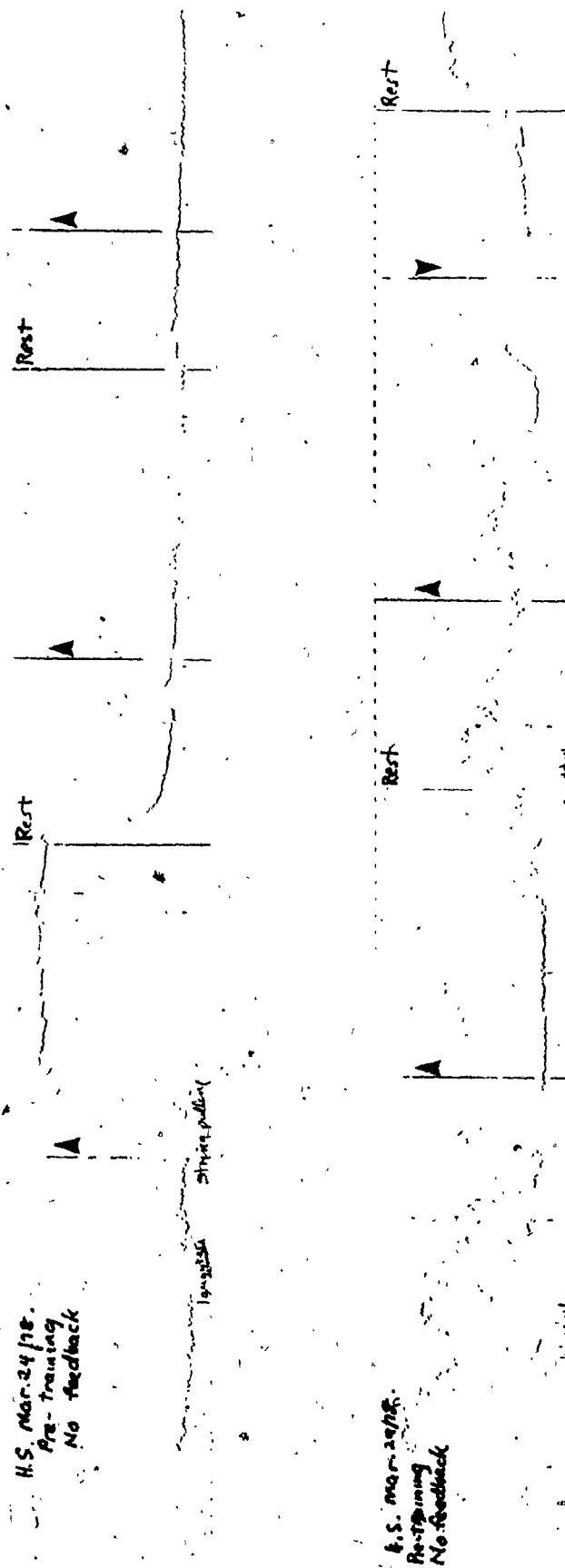


Figure 4. Pre-training sessions without feedback for subject H.S. Instructions were given at the vertical lines to increase pH (▲), decrease pH (▼), or rest.

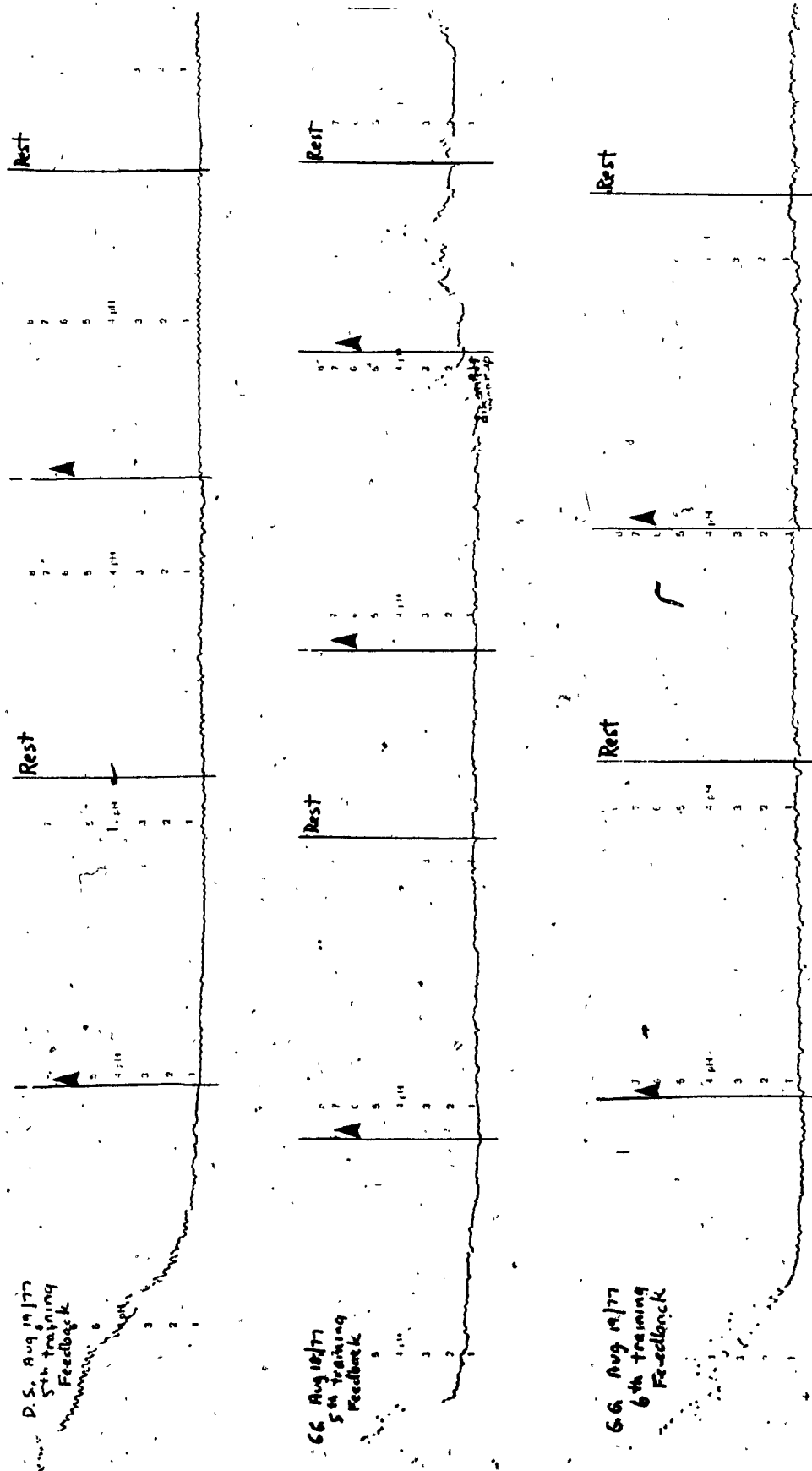


Figure 5. Fifth (subject D.S.) and fifth & sixth (subject G.G.) training sessions with feedback. Instructions were given at the vertical lines to increase pH (▲), decrease pH (▼), or rest.

of learning was seen over the training period. The fifth and sixth sessions of subject G.G. can be seen in Figure 5.

During his 9 training sessions the pH response of subject D.N. was in the appropriate direction in 4 of the 25 instructional periods to raise pH and in 2 of the 6 periods when he was asked to lower pH. The eighth and ninth sessions are seen in Figure 6.

During his eight training sessions the pH response of subject H.S. was in the appropriate direction in 3 of the 14 periods of instruction to raise pH and in 2 of the 3 periods when the subject was asked to lower pH. The number of appropriate responses did not increase over the eight sessions. Sessions five and six can be seen in Figure 7.

Post-Training

D.S. was unable to raise his pH when he was first instructed to in his non-feedback sessions 3 days following his training. However, during the second instruction of this session, his pH rose considerably from 0.25 to 5 and remained above this level during the ensuing rest period. The subject received no feedback on this highly appropriate response but he nevertheless told the experimenter that he had experienced a decrease in acidity. He was unable to lower pH from the level of 5 when he was instructed to in the next period.

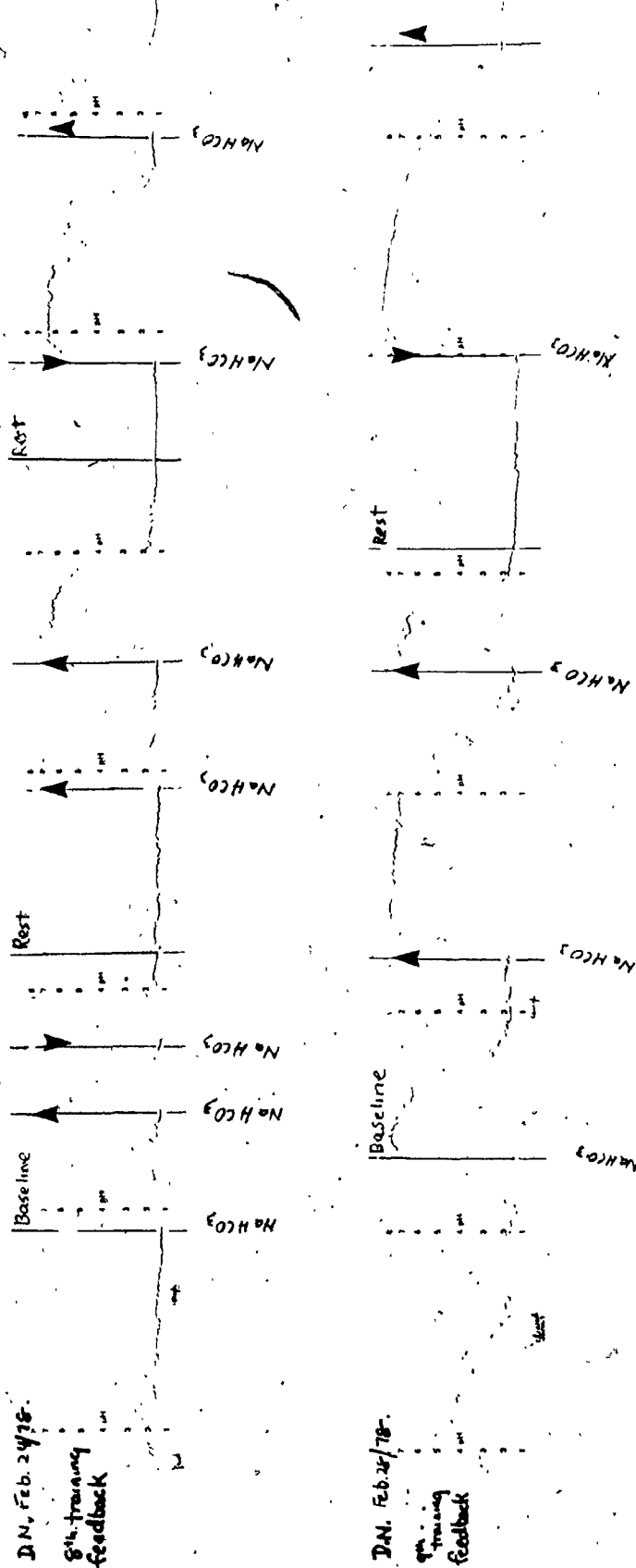


Figure 6. Eighth and ninth biofeedback training sessions for subject D.N. 5 ml 0.1N sodium bicarbonate (NaHCO_3) was given where indicated. Instructions were given at the vertical lines to increase pH (▲), decrease pH (▼), or rest. Instructional periods may be compared with the baseline neutralization curve in each session.

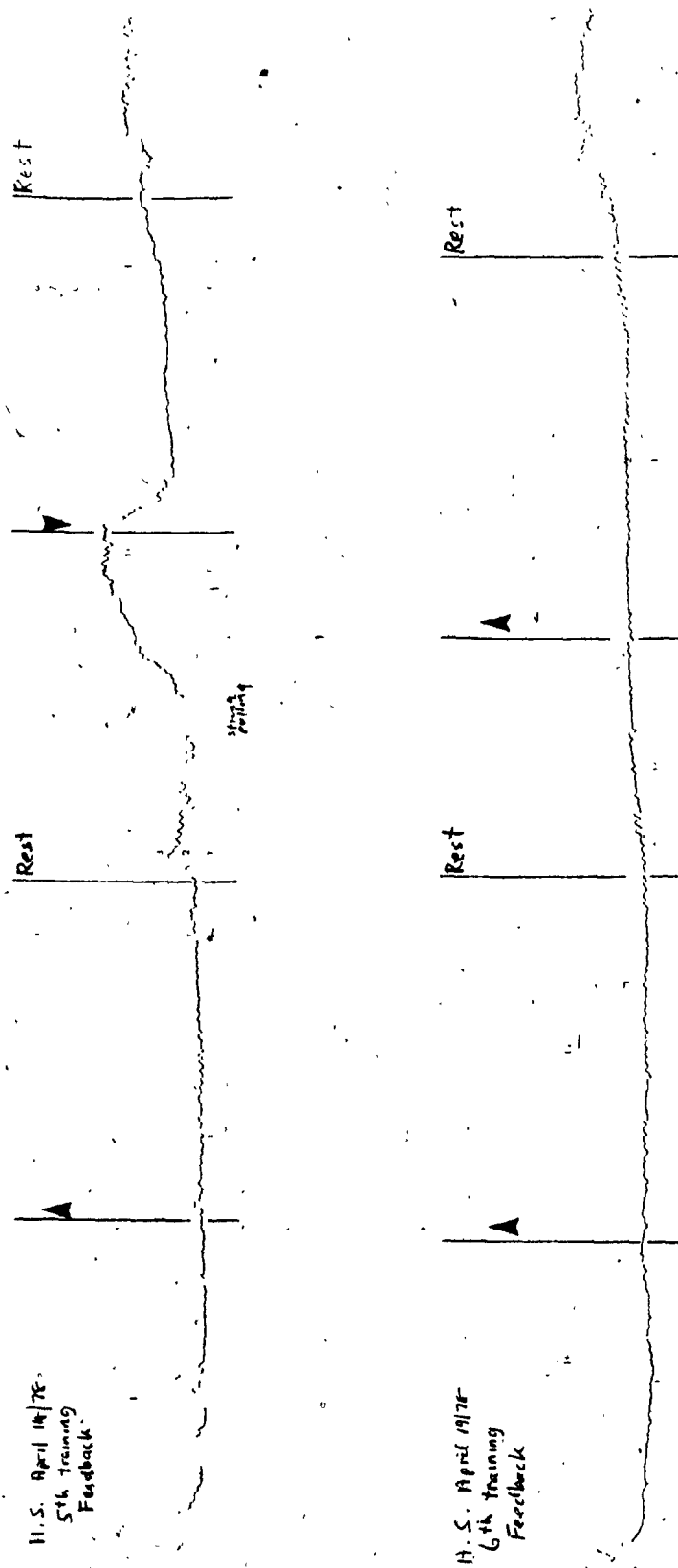



Figure 7. Fifth and sixth training sessions with feedback for subject H.S. Instructions were given at the vertical lines to increase pH (Δ), decrease pH (∇), or rest.

(This subject had never had the opportunity to be given an instruction to lower pH during his training since his pH had been consistently low throughout.) A fall in pH during the subsequent rest period of subject D.S. occurred after the experimenter's request, unexpected by the subject, to return for another session. During this other non-feedback session held 4 days later, D.S. was twice unable to raise his pH upon instruction and he reported this inability to the experimenter (Figure 8).

In the non-feedback session of subject G.G. held 3 days following his training, the pH rose appropriately from 1.25 to 4 almost immediately after he received his first instruction to raise it. It remained at that level during that period and the ensuing rest period. When he was asked to lower pH, the response was appropriate after a 5 minute latency and pH remained at 0.5 during the following rest period. In the second post-training session there were no appropriate pH changes. There was marked discomfort with the string during this session (Figure 9).

In his first post-training session without feedback, the pH of subject D.N. did not change appropriately during either of the 2 instructional periods to raise pH. When he was asked to increase acidity, however, D.N. effectively



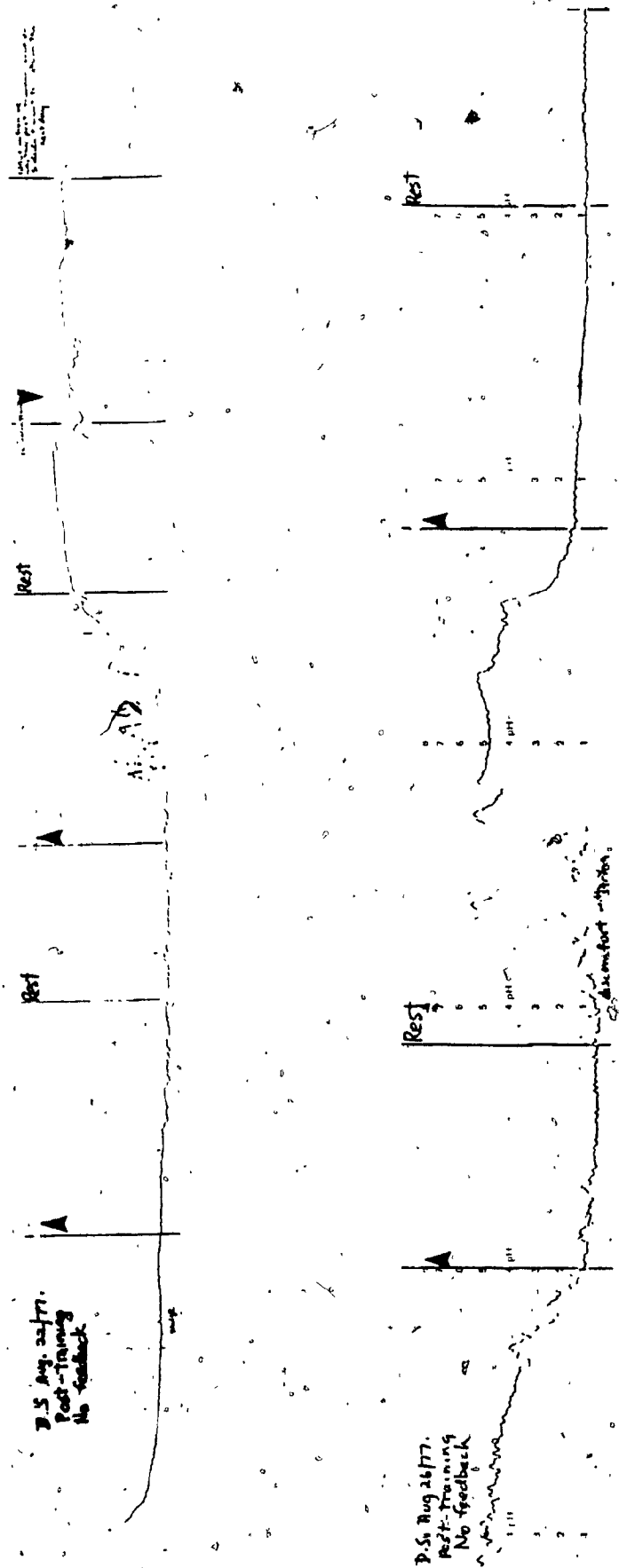


Figure 8. Post-training sessions without feedback for subject D.S. Instructions were given at the vertical lines to increase pH (Δ), decrease pH (∇), or rest.

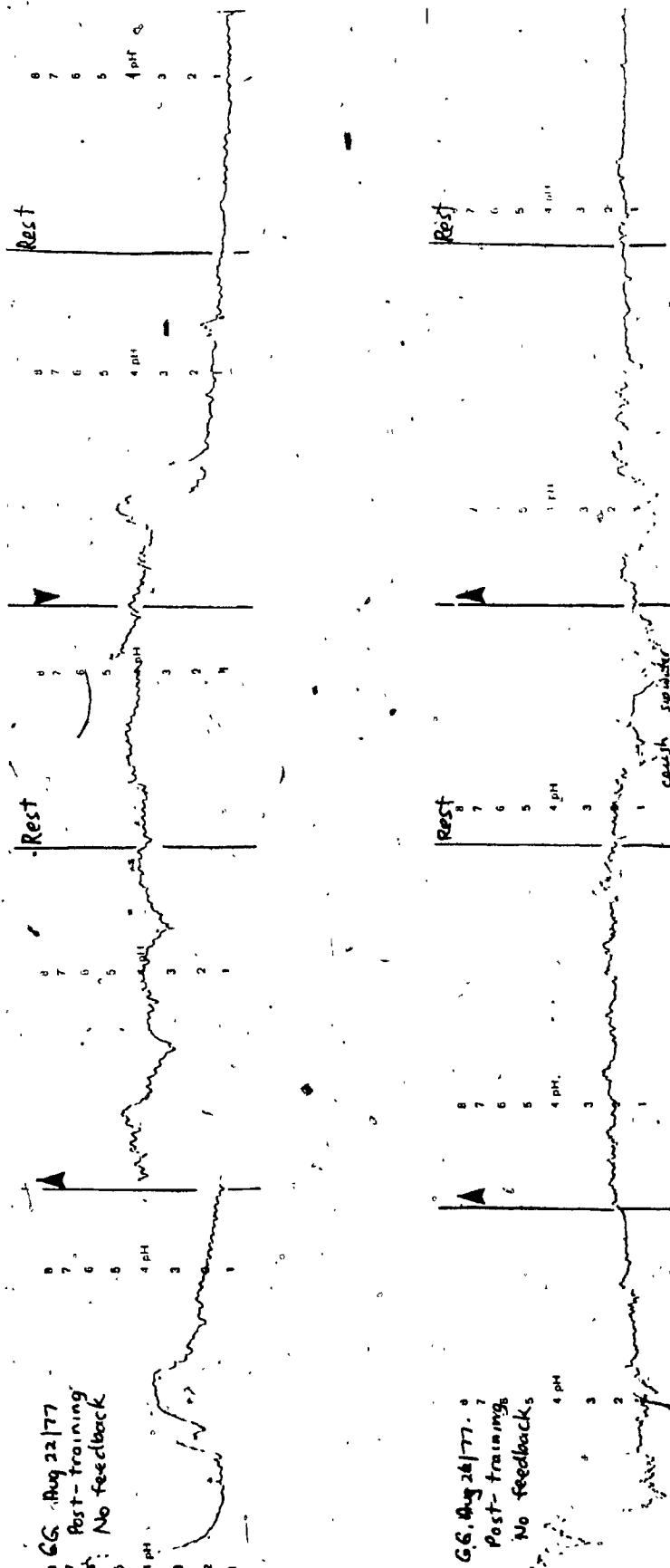


Figure 9. Post-training sessions without feedback for subject G.G. Instructions were given at the vertical lines to increase pH (\blacktriangle), decrease pH (\blacktriangledown), or rest.

countered the alkali dose of 5 ml 0.1N NaHCO_3 which had been administered to him. His pH did not rise and no neutralization curve was observed as they had been both 10 minutes prior to and 10 minutes following this instructional period. In his second post-training session without feedback no appropriate changes were observed (Figure 10).

In the post-training session without feedback of subject H.S., pH response was appropriate to instruction on 1 of the 2 occasions when he was asked to decrease acidity. The pH response was not appropriate when he was asked to increase acidity. The subject reported on this occasion that he had tried to get angry by magnifying angering situations in his mind but that he had been unable to experience them as angering (Figure 10).

Visual inspection of the data during both the training and post-training sessions revealed only a small number of changes appropriate to instruction. Therefore the most liberal criteria are used to examine and analyze the data in order to allow for the possibility that some factors were operating during the training to mask what may have been a greater number of changes. These most liberal criteria and statistical tests are biased in favor of showing a link between the training and post-training data to attempt to discover any possible relationship between the training and post-training effects.

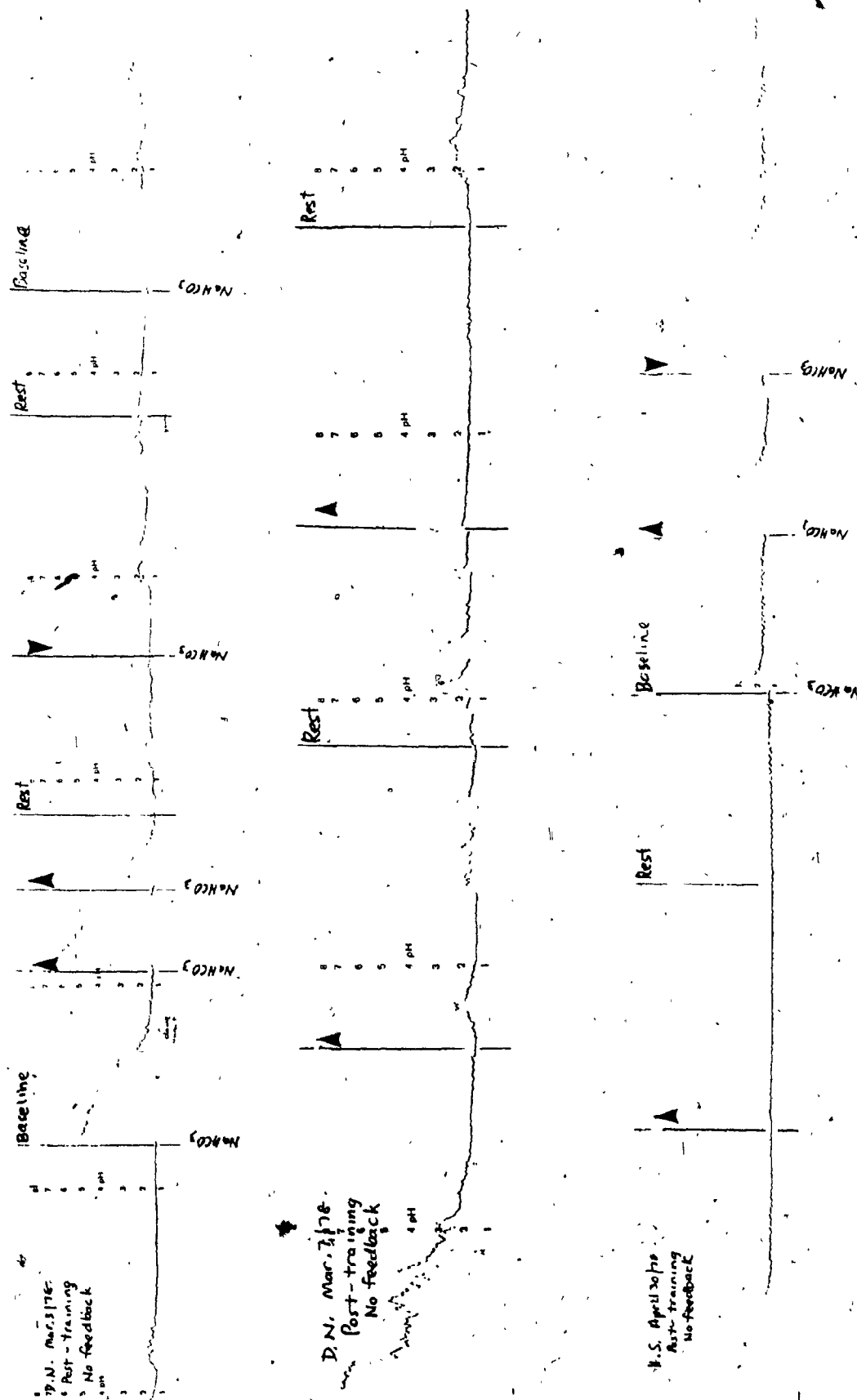


Figure 10. Post-training sessions without feedback for subjects D.N. & H.S. 5 ml 0.1N sodium bicarbonate (NaHCO₃) was given where indicated. Instructions were given at the vertical lines to increase pH (▲), decrease pH (▼), or rest. Instructional periods with NaHCO₃ may be compared with baseline neutralization curves.

Two independent observers inspected the data from the baseline, pre-training, training and post-training sessions. They were asked to examine the results of each instructional period for changes appropriate to the instruction. The number of appropriate responses to instructions to increase and decrease pH were added together because the numbers were so small, particularly in the post-training sessions. The percentages of pH changes appropriate to instructions regardless of direction were calculated for each subject in the training sessions and then in the post-training sessions. These percentages were then ranked. A Spearman correlation was carried out on the tied ranks. The assessment of the data by the two observers differed in the results of the training and not the post-training sessions with one observer assessing more responses as appropriate than the other observer. However, this did not affect the ordering and the same rank correlation was obtained by each observer: $r_s = .75$. Table 1 shows the results of the less liberal observer.

If the results of the training sessions of all four subjects are pooled, only 14 of the 71 or 19.7% of the instructions were correctly responded to. The pooled results of the post-training sessions show 5 out of 17 appropriate responses (29.4%).

Table 1

Appropriate responses before,
during and after biofeedback training

Subject	Pre- Training	Training			Post- Training		
		Proport ⁿ	%	Rank	Proport ⁿ	%	Rank
D.S.	0	0/10	0	1	1/5	20	1.5
G.G.	0	3/13	23	3	2/4	50	4
D.N.	0	6/31	19	2	1/5	20	1.5
H.S.	noise	5/17	29	4	1/3	33	3

Discussion

The first hypothesis of this experiment was that subjects who are unaware of their gastric pH will be unable to raise and/or lower their pH upon instruction prior to biofeedback training. This hypothesis was supported by the results of the pre-training sessions. The three subjects who had previously had no gastric pH feedback experience made no appropriate changes prior to training. The changes in the fourth subject's pH were interpreted as random or artifactual fluctuation.

The second hypothesis of this experiment was that, with adequate feedback, bidirectional control of gastric pH could be learned. The results do not demonstrate that learning developed during the training sessions.

Hypothesis three was that subjects would make appropriate pH changes following biofeedback training. Although some appropriate changes did occur in the non-feedback situation following training, it is suggested that the changes are not a result of learning achieved from the training per se since no development of learning was observed over the course of the training. The .75 correlation between training and post-training responses only suggests a relationship between these changes, i.e. that there is a tendency for whatever was happening in the training sessions to have been happening as

well following training.

Several explanations are offered to account for the inability of the biofeedback training to influence pH changes. These explanations will be discussed as they relate to the feedback, to the cognitive and affective state of the subject, and to the expectations of the subject and of the experimenter.

The feedback received by the subjects may not have been adequate for various reasons. This seems particularly probable in the case of the first two subjects whose pH in most sessions remained below 1.5 demonstrating very little variability within sessions. It is necessary for subjects to have some spontaneous pH changes with which they could associate either thoughts, emotions, acid-related feelings, or simply the biofeedback 'reward' in order for discriminative learning to occur. Without variability there is no opportunity for instrumental learning (Hulse, Deese & Egeth, 1975).

Another important feedback issue relates to the accuracy of the Heidelberg equipment which is within 0.5 pH units (Connell & Waters, 1964). At higher pH ranges, inaccuracies of 0.5 pH units are not significant. However, at a low pH range an unrecognized pH change of 0.5 unit could represent considerable change in acidity due to the logarithmic nature of the relationship of pH to acidity. It is possible that the subjects had been employing strategies which, in fact,

led to some changes in degree of acidity at the low pH range, but, because of the inaccuracy of the equipment, these changes could have been masked and not fed back to the subjects.

Therefore they would not have been reinforced for them, nor would they have learned about what may have been appropriate strategies.

A third factor which may have prevented adequate feedback relates to the pool of acid in the stomach. While the Heidelberg system has the advantage of replicating more closely than the intubation methods the 'real-life' stomach environment, this positive feature may present a disadvantage as well. pH measurement with the capsule does not involve withdrawing acid out of the stomach. This means that the feedback that is given to the subject reflects the total acid pool in the stomach. This pool is a composite of what has not yet drained from the last rest or instructional period and what is being produced during the current period. The pH feedback, therefore, may not actually reflect the most recent effort and change that a subject may have effected as well, or as convincingly, as if acid from the previous period had been withdrawn and the subject were acting on an empty stomach.

(This is a problem not only in biofeedback but in any research on gastric acidity. That is why in animal experiments fistulae or pouches are often used to secure exact collections of acid

(Eimas, Swan & Jacobson, 1967).) It was suspected that this problem might make biofeedback training with the Heidelberg unit difficult but it was felt that its many other advantages merited researching its use.

When it was seen that the pH of D.S. and G.G. did not vary and that they did not show any evidence of developing control of pH during their training, the above-mentioned feedback factors were considered. The decision was then taken to administer alkali to the second two subjects to supplement their feedback. It was hoped that this would better reflect to the subject the current acid state in the stomach. It clearly allowed for better feedback to these subjects on their ability to increase acidity although this did not lead to increased frequency of this response.

The subjects appeared able to increase acidity notably when they could experience feelings of anger according to their subjective reports suggesting cognitive and affective cephalic mediation of the acidity response (Appendix A). It was interesting to note that both D.N. and H.S. reported that they could not always replicate these situationally related feelings of anger when they were instructed to increase acidity (Appendix B). H.S. had reported this same difficulty during his training in the pilot study. It is possible that, for these subjects, experiencing the anger in their imagination can only

be accomplished a limited number of times after the original angering experience. One can speculate as to whether these first imaging experiences served as a type of psychological catharsis preventing further use of these images to produce a physiological change for these subjects at least in the acid response system. It has been suggested that emotionally stressful situations might give rise to either hyper- or hypo-function of the stomach depending upon the nature of the circumstances and the subject's interpretation of the event (Wolf & Welsh, 1972). On one particular occasion during his training D.N. reported having set himself to think of unpleasant situations and having savoured the experience. These attempts however were met with a response of less acidity rather than more as per the instruction, much to the subject's surprise (Appendix C). Perhaps had further questioning taken place with regard to these unpleasant feelings they would have been described as of a sad, frightening or disgusting nature which some report inhibit the stomach's secretory activity (Wolf & Welsh, 1972; Gorman, Note 2). Further research in this area would be enhanced by more extensive use of subjective reports.

Meichenbaum (1976) in discussing cognitive factors in biofeedback therapy proposed that subjects be trained to use imagery as a tool in changing physiological responses.

Shapiro and Schwartz (1972) reported that they carried out successful biofeedback training in which subjects acquired heart rate control and no consistent imagery pattern was apparent. Future research should assess specifically whether internal sources of stimulation can be used with a view to reliably controlling autonomic responses. Results of this research suggest links between cognitive and affective experiences and the acidity response but the ability to voluntarily evoke thoughts with their accompanying affect upon demand has not been demonstrated. The non-naive subjects appeared to have more imagery tactics in their repertoire than did the naive subjects as evidenced by the amount of subjective reporting. This, however, is confounded by the fact that the experimenter knew the non-naive subjects better than the first two subjects, perhaps rendering them more willing to discuss their tactics with the experimenter.

Other factors that may have contributed to the results of this research are the expectations held by the subjects and the experimenter or the atmosphere in which the training took place. The results of the pre-training sessions of subjects D.S. and G.G. in which they became more instead of less acidic suggest that the instructions may have been perceived as stressful to them. This idea is strengthened by the contrasting results of their first and second post-training sessions. The

experimenter had discussed with both D.S. and G.G. prior to their first post-training session that no learning had developed during their training and that this post-feedback session was simply a matter of completing the planned protocol for the research. They knew that the design of the experiment was to compare pH changes upon instruction before and after the biofeedback training sessions. Because they were aware that they had not developed control of pH over the training period, there was no pressure for them to perform in what they had expected to be their final session in the laboratory. This, and the thought that it was their final capsule-swallowing session, may have enhanced their ability to relax and 'let go' during this session when they were instructed to raise pH. Furthermore, the experimenter is aware of having felt less anticipation than at the other sessions. Certainly no appropriate pH changes were expected of the first two subjects since so little had been observed in earlier sessions and then only in the case of G.G. Perhaps additionally this non-stress feeling was conveyed to these subjects. Gorman (1976) noted that his subjects were able to inhibit secretion when in a relaxed attentive state. It may be that D.S. and G.G. had tried too hard and did not relax during their pre-training and training, but when, at the first post-training session, there was nothing to lose since nothing had been gained, they could

achieve a more appropriate mental state under which changes could occur. In this regard, Peper (1976) related an analogy between biofeedback training of a visceral response and urination, i.e. the urine starts to flow when one finally gives up and no longer cares. In contrast to D.S. and G.G., the second two subjects had reported feeling they had made some appropriate changes over the course of their training. They and the experimenter may have been feeling some stress in their post-training session when they were not as successful in the direction of decreasing acidity.

The results of this study suggest that the question of whether pH feedback given by the capsule unit can teach healthy subjects control of gastric pH can only be answered using those subjects who have a higher and more variable pH baseline. These are the subjects for whom the feedback would be adequate to ask the question. It should be noted that if such subjects could be found, one might expect individual differences in visceral learning just as there are individual differences in learning a motor skill. If subjects could learn to voluntarily control pH with this equipment, the following important questions could perhaps be answered. How long does learned control last when feedback is withdrawn? Does reward (feedback) withdrawal lead to extinction as is seen in the operant conditioning paradigm (Kling, 1971)? The possibility exists

that patients who are symptomatic because of hyperacidity (ulcer patients) could receive the reward of symptom improvement by learning acidity control. These patients are not likely to have a baseline pH high enough to enable them to get adequate feedback using the capsule alone. However, perhaps they could be trained while symptomatic using the alkaline titrant. This should relieve their discomfort and if they could then learn to maintain the pH high for a longer period of time with biofeedback, the associated relief from pain may serve as a stronger reward than would simply an appropriate response which was the reward used in the present experiment.

In conclusion, the absence of learning of acidity control using the Heidelberg Capsule System does not imply that biofeedback training cannot be effective in this area using other techniques. However, the Heidelberg System presents minimal discomfort to subjects (relative to other forms of acid measuring equipment) permitting a better measure of cephalic phase effects. It eliminates the risk of irritating gastric mucosa through the standard measuring techniques thus minimizing any gastric phase effects. It more closely parallels the natural environment of the stomach making generalizations from the laboratory to 'real-life' conditions more meaningful. For these reasons, future research using this equipment should attempt to answer other questions regarding the relation of

the mind and the gut. It appears that the issue of voluntary control of pH with biofeedback training can only be studied with the Heidelberg System if it were possible to secure subjects with a higher basal pH. However, the biofeedback component itself may prove useful in helping ulcer subjects explore various cephalic influences on their acidity pH and, more importantly, coping strategies which may alleviate symptoms..

INTRODUCTION TO STUDY II - RELAXATION TRAINING

Phase two of this experiment was designed to explore the effects of a modified form of Jacobsonian progressive relaxation training on gastric acidity (Behavior-Media-Relaxation Exercises, Note 5).

Jacobson (1970) reported that his clinical experience often led him to include peptic ulcer as a tension disorder along with disorders such as hypertension, certain coronary symptoms, irritable colon, etc. He reasoned that tension or anxiety and relaxation are mutually exclusive because what we visualize actually or in image determines our emotions. He maintained that if we visualize relaxation and carry out the relaxation exercises, our emotions will be thus directed. He reported that the reduction of muscle tension also led to the reduction of autonomic activity especially that of the sympathetic nervous system (Jacobson, 1967).

Chinnian, Nammalvar and Rao (1975) reported that pulse pressure and pulse rate (two sympathetically controlled activities) were significantly decreased following progressive relaxation as compared to control periods, thus supporting Jacobson's earlier reported results with regard to sympathetic activity (Jacobson, 1938). There were significant changes in respiratory rate in Chinnian's subjects but these followed the

control sessions as well. Furthermore, Reinking and Kohl (1975), in studying the effects of various forms of relaxation training on physiological measures, reported that the best single correlate of sympathetic arousal was skeletal muscle action potential. Their subjects, with practice in Jacobsonian progressive relaxation reduced muscle tension by 50%.

Tarler-Benlolo (1978) in her review of relaxation studies concluded that different strategies employed in relaxation techniques/ lead to different patterns of physiological response and she cited Davidson and Schwartz (1976) who have classified the focus of relaxation procedures as primarily inhibiting either somatic or cognitive activity in either an active or passive manner. They further stated that since different physiological measures reflect predominantly cognitive versus somatic processes, such measures should be changed predictably according to the form of relaxation used.

The progressive relaxation technique employed in this study appears to be an active procedure. It would seem that it should inhibit not only muscle tension but also extraneous cognitive activity. It requires both the tensing and relaxing of muscles progressively through the body and, as well, the concentration on instructions and on the discrimination of feelings of tension versus relaxation. Jacobson's theory implies that if peptic ulcer is a tension disorder, decrease

in muscle tension or somatic activity should relieve the disorder, since anxiety will be relieved. In contradistinction to Jacobson's thinking (e.g. Jacobson, 1967), the hypothesis of this experiment is that if relaxation training has any positive effect on the reduction of gastric acidity it will be rather because of the type of cognitive rather than somatic activity involved in the exercises. Gastric acid secretion is a visceral, not a somatic activity. It is mediated by the vagus nerve and the parasympathetic nervous system. The physiological correlates of relaxation training cited earlier are mediated by the sympathetic nervous system. There is a lack of empirical data suggesting parasympathetic correlates of muscle relaxation.

Benson, Beary and Carol (1974) wrote that "the relaxation response appears to be an integrated hypothalamic response which results in generalized decreased sympathetic nervous system activity, and perhaps also increased parasympathetic activity." This then suggests that if the relaxation response leads to an increase in parasympathetic activity, the vagus nerve would be activated with a concomitant increase in the production of gastric acidity. However, we also know of the influences of central or cognitive activity on gastric secretion. Wolf (1977) reported that a placebo given to the same eighteen subjects on two different occasions by two different experimenters produced opposite effects on acid secretion. Furthermore, the

same dose of an acid-inhibiting pharmacodynamic agent has been shown to produce varying effects on acidity at different times on a conscious subject contrasting with the uniform inhibiting effects seen on a subject rendered unconscious by an automobile accident (Wolf, Note 1).

It is difficult to predict what the effects of relaxation training will be on gastric acidity. Will there be a physiological effect of decreased sympathetic activity, increased parasympathetic activity, and increased acidity or will the cognitive activity involving the subjects' focus on instructions be associated with a decrease in acidity? It is expected that, contrary to what Jacobson's thesis might suggest, little if any change in acidity will result from practiced relaxation exercises, and that if a change is seen, it will be in the direction of more, rather than less, acidity since there is no evidence to suggest that a cognitive focus on instructions reduces gastric acid secretion. On the other hand, there is evidence that relaxation exercises lead to a decrease in sympathetic arousal and there is also some suggestion that the sympathetic and parasympathetic systems have paradoxical effects. We know that when Wolf's patient with a gastric fistula reported experiencing sudden fear this was associated with sympathetic hyperactivity and his gastric secretory response was a decrease in acid secretion (Wolf & Wolff, 1942).

A review of the literature has revealed little data in the area of relaxation training and the gastric response system. Beaty (Note 6) carried out feedback-assisted relaxation training as a treatment for patients with gastric ulcers. He reported that two of his five subjects dropped out of treatment during the baseline phase and continued to have symptoms. He concluded that, since the other three patients had no stomach pain and were taking no ulcer medication at the end of the program, his treatment was functionally related to the reduction in symptoms. However, it is not known whether the three patients who remained in the program would have improved without treatment. No acidity measures were taken. Jacobson (1970) reported on a patient who in 1944 suffered from disabling anxiety and gastric achlorhydria (anacidity) among other symptoms. Within a month of progressive relaxation training this patient was reported to have been able to discontinue his capsules of hydrochloric acid because his achlorhydria was replaced by more nearly normal gastric acidity. It is not possible to determine from the above clinical reports whether there was a functional relatedness between the relaxation training and either of the two opposing acidity responses.

Because of the physiological evidence cited earlier, it would seem more likely that if any gastric results of progressive relaxation training are seen, they would be in the

direction of increased rather than decreased gastric acidity.

Method

Subjects

The subjects were two of the male volunteers who participated in the biofeedback phase of the experiment--G.G. and H.S. Neither had experienced relaxation training prior to the study. They fasted for eight hours prior to each experimental session. Each session took place in the morning.

Apparatus and Procedure

The apparatus and procedure were the same as those used in Study I of this research.

Baseline Control: Each subject had two baseline sessions in order to assess the temporal effects of the 20 minute interval that the relaxation exercises would last in the experimental condition. After a 15 minute basal pH level was reached, subjects in these baseline control sessions were given 5 ml 0.1N NaHCO_3 to assess the degree of acidity at that time. Once the pH had returned to or close to the basal level, a 20 minute time interval was allowed to elapse. This was followed by another dose of 5 ml 0.1N NaHCO_3 to assess the degree of acidity at the end of the 20 minutes. Subjects read throughout these sessions. At the end of the second control session each subject was instructed in the principles of the

relaxation training that they were to learn and practice daily over the next eight days. The principles and the specific muscle tensing and relaxation instructions were given on tape and followed by the subjects at that time in order to assure the experimenter that the subjects were able to understand and accurately follow the instructions.

Training: Subjects practiced tensing and relaxing the required muscle groups as instructed on the tape on eight occasions in their own homes. Duration of practice was 20 minutes daily. The time of day was left to the subjects' preference.


Post-training: These sessions (two per subject) were carried out in the same time frame as the baseline control sessions. Each subject was given 5 ml 0.1N NaHCO_3 after a 15 minute basal pH level had been reached. When the pH had returned to the basal level or close to it, the relaxation tape was put on and subjects went through the progressive relaxation exercises that they had practiced at home. After the 20 minute procedure, subjects were again given 5 ml 0.1N NaHCO_3 to assess the degree of acidity at that time.

Results

Data Analysis

Several data analysis procedures were explored before

the nature of the distribution of the data became known. Due to the exploratory nature of this project, it was not clear prior to the experiment which measures would be most appropriate to analyze. It was decided that it would be important to evaluate the amount of acid in the stomach immediately upon conclusion of the 15 minute basal period and the 20-minute reading period in control sessions, and upon conclusion of the 15-minute basal period and the 20 minute relaxation exercise period in the experimental sessions. In addition to the measure of acid immediately following these periods, it was felt that it would be important to look at the acid output over the ensuing minutes. Therefore the results were evaluated in two ways. First, the amount of acid in the stomach at the conclusion of both the control period and of the relaxation exercises was compared to the amount of acid in the stomach at the end of the 15 minute basal period in control and experimental sessions respectively. This amount of acid was measured by the peak pH within 45 seconds of the administration of the 5 ml 0.1N NaHCO_3 . The basal pH of each session was subtracted from each of the peaks within the session so that results between sessions could be compared.

 Secondly, the amount of acid produced over the ensuing several minutes prior to and following control and exercise periods was assessed by estimating the areas under the curves

produced by the rise and subsequent fall of pH upon administration of the alkaline substance. These areas were computed by sampling the median pH of every 30 second period until pH returned to within .2 of the basal pH of that day, or for a maximum of 20 minutes if the pH did not return to the basal level. The basal pH was subtracted from each of these pH numbers to allow comparison of data between different sessions. The mean of every two numbers was multiplied by .5 minutes and these resulting numbers were added together to give a measure of the area under the curve.

A statistical analysis was not appropriate for the results of this study since there were only two subjects and there was considerable variability between them and within their replicated sessions. Because this was exploratory work, it was not desirable to enhance the likelihood of making a type II error by statistically analyzing data with so much variability and so few degrees of freedom. A summary of all the data is presented in Tables 2 and 3. An example of the variability is the case of G.G. where this same subject in the same condition (in the instances of both control and exercise conditions) showed variable responses on two different days (Table 3).

When the pH peaks were evaluated, that is, when the immediate effect on acidity was assessed, the results showed

Table 2

pH Peaks

(Maximum pH within 45 seconds of administration
of 5 ml 0.1N NaHCO₃ with basal pH subtracted from it)

Subject	Condition	Pre		Post	
G.G.	control	2.8	$\bar{x} = 3.02$	0.7	$\bar{x} = 2.47$
G.G.	control	3.25		4.24	
H.S.	control	5	$\bar{x} = 4.6$	2.8	$\bar{x} = 4.37$
H.S.	control	4.2		5.95	
G.G.	exercise	2.9	$\bar{x} = 3.75$	5.15	$\bar{x} = 4.97$
G.G.	exercise	4.6		4.8	
H.S.	exercise	3.15	$\bar{x} = 3.2$	5.4	$\bar{x} = 5.7$
H.S.	exercise	3.25		6	

Table 3

Area under Curve
(output over time)

Subject	Condition	Pre		Post	
G.G.	control	6.68	$\bar{x} = 21.36$.49	$\bar{x} = 40.61$
G.G.	control	36.04		80.73	
H.S.	control	5.46	$\bar{x} = 6.45$	13.86	$\bar{x} = 34.48$
H.S.	control	7.44		55.11	
G.G.	exercise	37.78	$\bar{x} = 40.64$	68.58	$\bar{x} = 51.69$
G.G.	exercise	43.5		34.8	
H.S.	exercise	1.58	$\bar{x} = 11.87$	8.74	$\bar{x} = 14.97$
H.S.	exercise	22.17		21.21	

that the pH peaks of each subject in each of their experimental (exercise) sessions became larger, whereas after the control (reading) period there was no systematic change in peak pH (Table 2). Figure 11 shows the pooled means and standard errors of peak pH in the experimental and control conditions. Each mean represents the pooling of two subjects over two sessions, i.e. four data points. There was a reduction of acid in the immediate post-exercise period compared to the pre-exercise period of these two subjects as measured by the pH peak within 45 seconds. A systematic change was not seen in the control condition in these subjects.

In contrast to the peak pH measures where an immediate decrease in acidity was seen following the exercises, when the area under the curve, that is the acid output over the ensuing period of time, was evaluated, the results pointed in the opposite direction. They are somewhat more equivocal, however, since as noted above variability is seen in both conditions (Table 3). The means of the areas under the curves of the replicated sessions of each subject increased (less acid) after both the control and exercise periods compared to before both conditions, but there was a greater relative increase after the control period. In other words, a greater reduction in mean acid output for each subject was noted after he read than after he did relaxation exercises (Table 3). Figure 12 shows

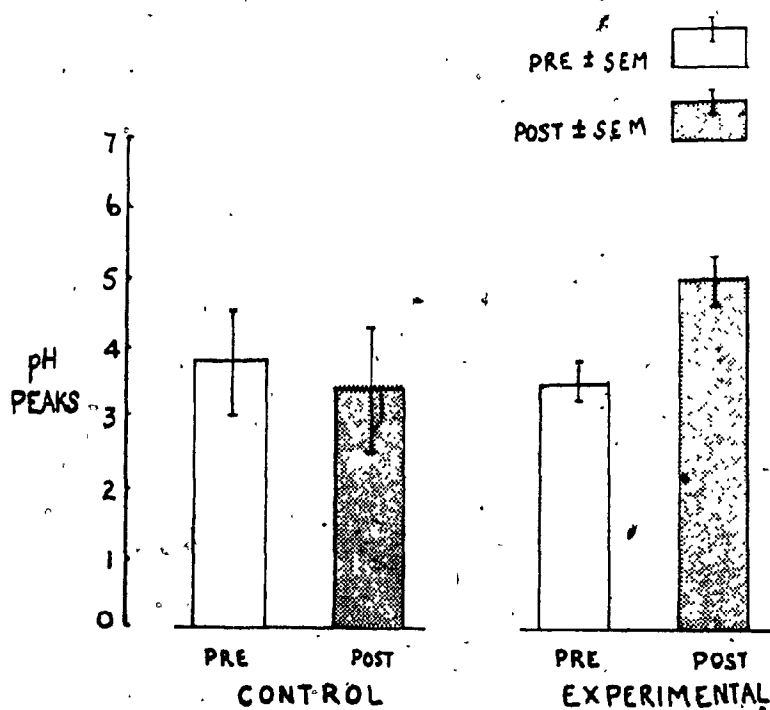


Figure 11. Pooled means and standard error of the mean \pm (SEM) of peak pH within 45 seconds of administration of 5 ml 0.1N NaHCO₃ pre- and post-control and exercise conditions.

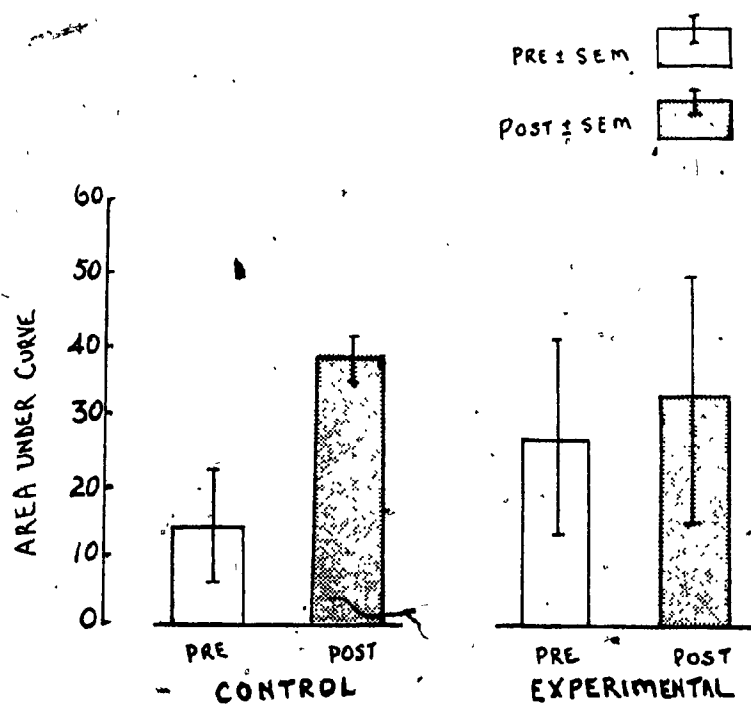


Figure 12. Pooled means and standard error of the mean (\pm SEM) of acid output over time (area under curve) pre- and post-control and exercise conditions.

the pooled means and standard errors of the pre and post areas under the curves in both conditions. Each mean here represents the pooling of two subjects over two sessions.

In summary, the data for these two subjects suggests that their acid output measured over a period of time decreased less following the relaxation exercises than it did following the control (reading) condition. This result is in contrast to the peak measures of acidity taken within 45 seconds where no systematic change was seen pre to post in the control condition, whereas in the exercise condition, a decrease in acidity was noted immediately after the exercises.

Discussion

The hypothesized effects of relaxation exercises on gastric acidity were that little change would be seen following the exercises, and that if an effect was seen, it would be in the direction of more, rather than less, acidity when compared to the control sessions. The results demonstrated by the two subjects in this study are not inconsistent with this hypothesis. However, because of the high degree of variability noted, particularly in the measure of acid output over a period of time, any conclusions to be drawn about these two subjects must be done very cautiously.

As reported in the Results section, two effects of

exercise and control conditions were evaluated--the immediate pH peak within 45 seconds and the amount of acid secreted over the ensuing period of time. Consistently higher pH peaks were noted in the 45 second period immediately following the relaxation exercises. pH peaks were not systematically higher in the control condition. If more subjects were studied, and if this immediate effect were again seen consistently only in the exercise condition, it may suggest that the contracting and relaxing of the stomach muscles promoted more rapid emptying of gastric acid. This gastric emptying would result in the immediate effect of a higher pH when stomach contents are neutralized with 5 ml 0.1N NaHCO_3 as was noted in the present study. If the exercises promoted the emptying of acid from the stomach, one would not expect any systematic increase in pH peak within 45 seconds in a control condition.

When the acid output over a period of time was evaluated, the results suggested that the mean output following the control (reading) period was considerably less than before it, whereas the mean output following the exercise period was only somewhat less than before the exercises. If more subjects were studied and these same differences were to recur, this would lend support to the hypothesis discussed earlier by suggesting a tendency for more acid to be produced following relaxation exercises than following a control period. This

would not be surprising in view of the effects of the relaxation training reported by Jacobson (1938), Chinnian et al. (1975) and Reinking and Kohl (1975). These effects included lowered sympathetic arousal as indicated by lowered heart rate and pulse pressure. It has been theorized that sympathetic arousal may have an anti-cholinergic effect, i.e. that the parasympathetic system may be inhibited when the sympathetic system is aroused (Moraes, Nyhus, Kalahanis, Bombéck & Das Gupta, 1978). It may be that the effect of the reverse, i.e. of a decrease in sympathetic arousal as is seen after progressive relaxation exercises, is that the parasympathetic system is disinhibited. Since the cholinergic vagus nerve is part of the parasympathetic system, this could leave it disinhibited to act on the parietal cells which produce acid. Further studies carried out on a larger number of subjects measuring both sympathetic and parasympathetic responses could test the above interpretation.

A possible confounding factor in the present study and a somewhat difficult one to overcome in future studies is what the subjects are doing or thinking in the control periods and prior to and following the exercises when the acidity measures are taken. Subjects were allowed to read material of their choice at all times in this study except while they were listening to the relaxation tape and carrying out the exercises.

The type of material they were reading and the interest they manifested in it may have had an effect on their gastric acid pH measures. Some evidence suggesting this will be presented recognizing that it was not systematically gathered but rather noted post hoc by the experimenter when notes of the sessions were examined.

In his first baseline session G.G. was reading and correcting a student's paper, a task he reported not enjoying because it was not the first time this student had rewritten this paper for him. G.G. became somewhat more acidic over the course of this session (Appendix D). In his second baseline session, he was reading a magazine in which he reported great interest and enjoyment. He became less acidic during that period of time (Appendix D). Similarly in an experimental session, when he was reading a novel in which he expressed great interest, his pH did not return to the low basal level in the usual amount of time prior to the exercise period (Appendix D). In his remaining session G.G. did not volunteer any comment about his reading material as he had in the above-mentioned sessions.

In the second baseline session of subject H.S., his pH was noted to be higher during the times he was reading material in which he reported interest (Appendix D). His interest in the content of his reading was not noted in his

other sessions.

In view of these serendipitous findings, it is suggested that any future studies that have the intent of measuring the effects of relaxation exercises on gastric acidity attempt to control for the cognitive influences on acidity throughout the control sessions and prior to and following the exercise periods when measures are being taken.

Furthermore, it is suggested that further research in this area make more extensive use of subjective reports than was done in these experiments. The reports that were volunteered suggest some links between cognitive and affective experiences and the acidity response. Future studies might assess whether particular internal stimuli systematically affect changes in acidity. Can these changes be interrupted? Can thoughts and images be used with a view to reliably controlling this autonomic response? It may be that individual differences in this area are so great that group studies would obscure clues about cognitive or affective influences on gastric acidity. It has been suggested that sensitive individual studies may be more fruitful in exploring the relationship between the mind and the gut (Wolf, Note 1).

In conclusion, the results of this exploratory experiment call for (1) more extensive study of the effects of a subject's involvement in reading material and other cognitive

influences on gastric acidity; (2) future relaxation training studies to measure sympathetic arousal concurrent with the measure of gastric acid secretion in order to attempt to answer the question of whether disinhibition of the parasympathetic vagus nerve occurs during times of inhibited sympathetic arousal; and (3) such relaxation studies to be carried out on a larger number of subjects to permit statistical analysis of the results.

SUMMARY

Study I was designed to ask whether awareness of one's level of gastric acidity, through a technique of biofeedback training, could enable a subject to voluntarily raise and lower his acidity upon completion of training. Although some appropriate changes did occur after training, it is suggested that the changes were not a result of the biofeedback per se since no development of learning was observed over the course of the training. Explanations relating to the adequacy of the feedback, to the cognitive and affective state of the subject, and to the expectations of the subject and experimenter were offered to account for the results. Verbal feedback from the subjects suggested that the Heidelberg Capsule telemetering system was a relatively non-aversive form of acid measuring equipment that could be fruitfully used in psychological studies.

Study II explored the effects of a modified form of Jacobson's progressive muscle relaxation training on the acidity response of two subjects. In contrast to what Jacobson's theory would have predicted, a decrease in acidity was not expected. Results showed that less acid was seen pre to post in both control (reading) and relaxation exercise conditions, but this decrease in acid output over a period

of time appeared to be somewhat larger after the control period. It was suggested that the decreased amount of acid that was seen only within 45 seconds after the exercise condition was due to the contracting and relaxing of stomach muscles which could have effected rapid gastric emptying leading to a higher pH right after the exercises when the alkaline substance was swallowed.

It was suggested that further psychological research in the area of gastric acidity make more extensive use of subjective reports than was done in these experiments. The few reports that were volunteered suggest some links between cognitive and affective experiences and the acidity response which merit further exploration.

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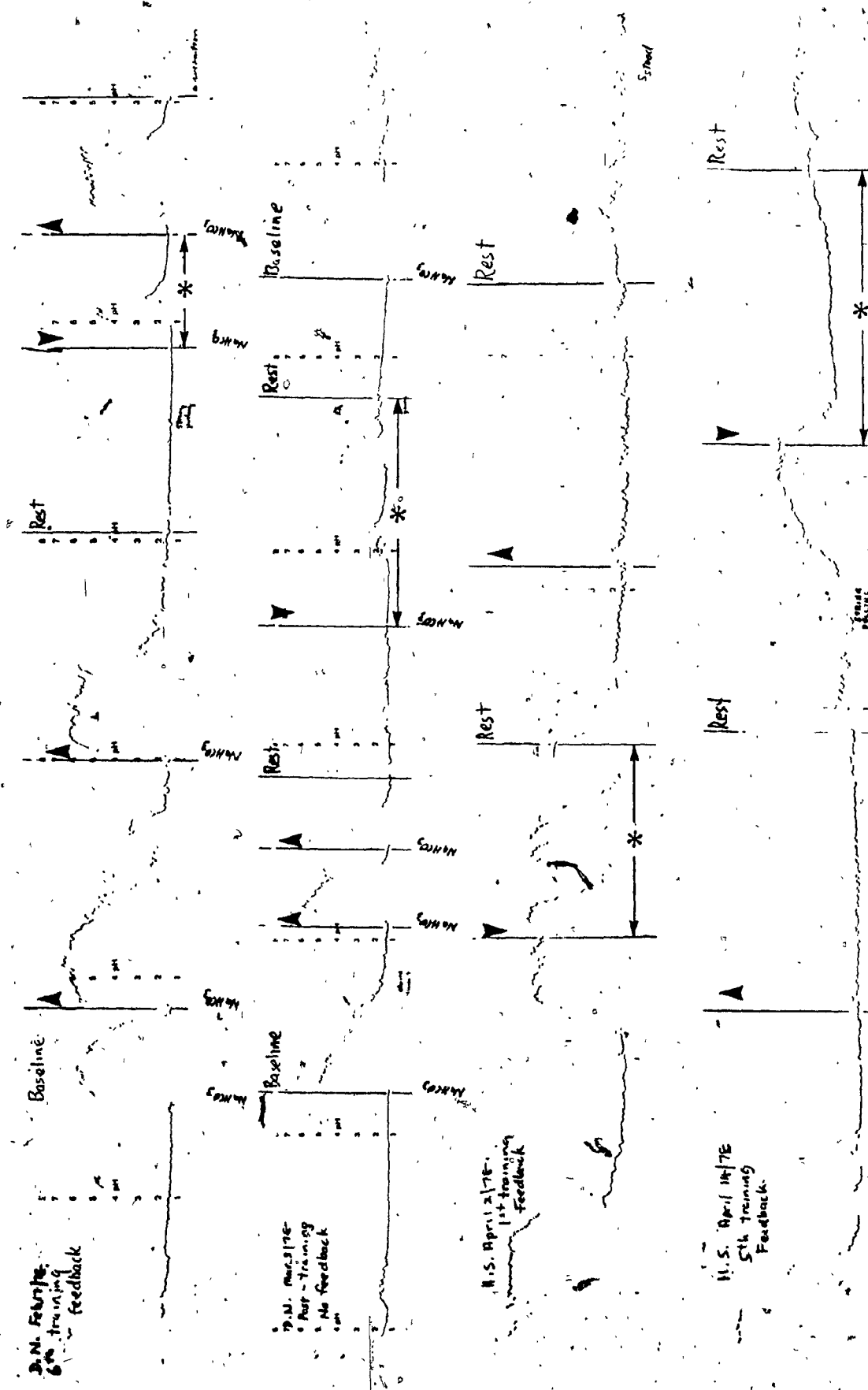
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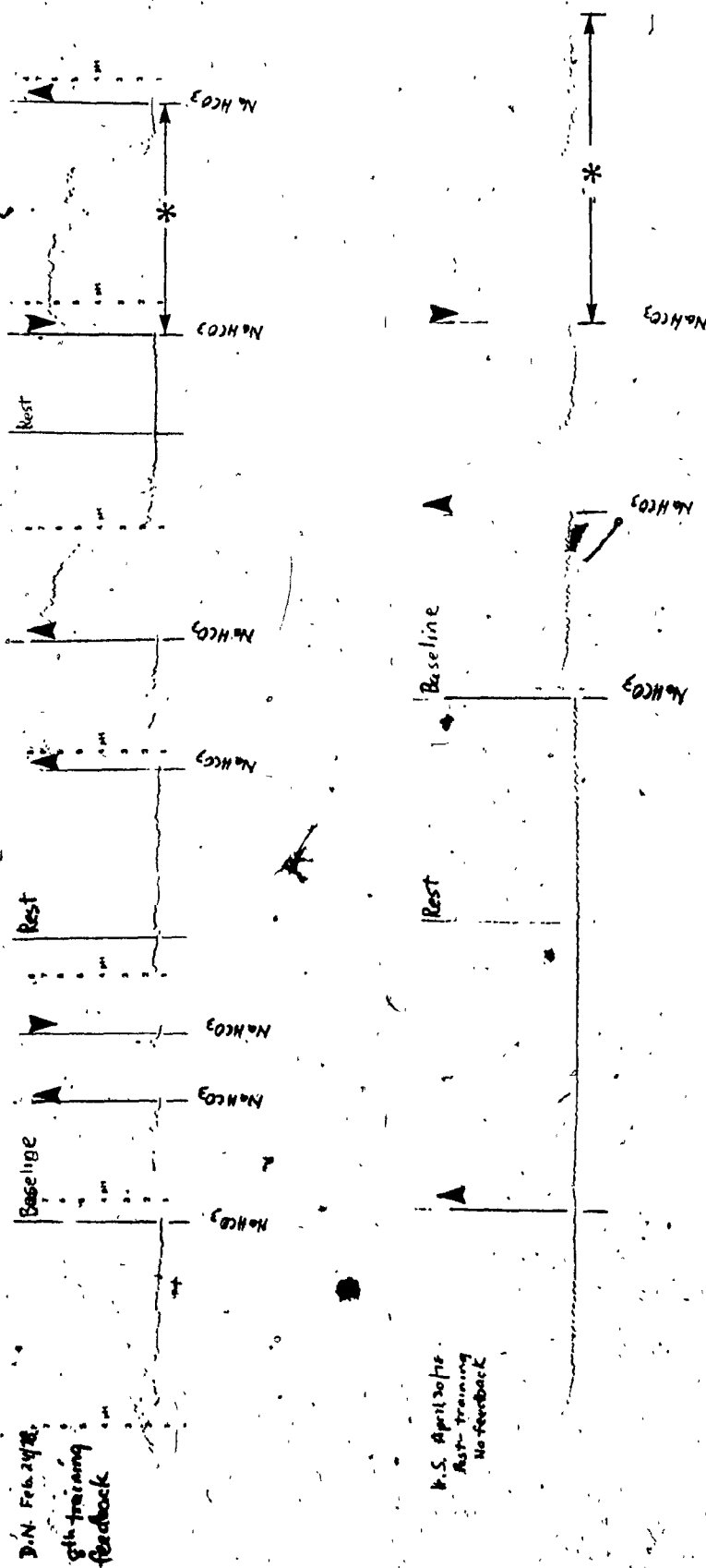
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APPENDIX A



Asterisks (*) refer to periods when subjects reported subjectively experiencing feelings of anger. Acidity increased during these periods.

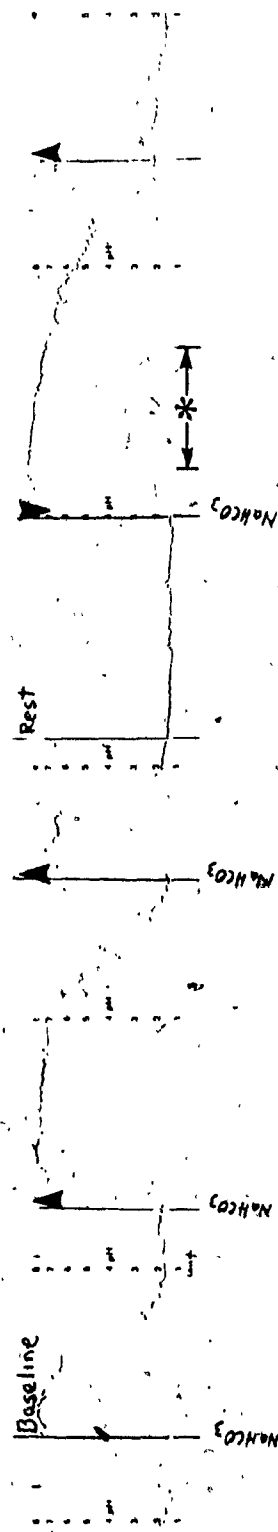
APPENDIX B



Asterisks (*) refer to periods when subjects reported an inability to feel anger over previously experienced angering situations. Acidity did not increase during these periods when compared with baseline neutralization curves.

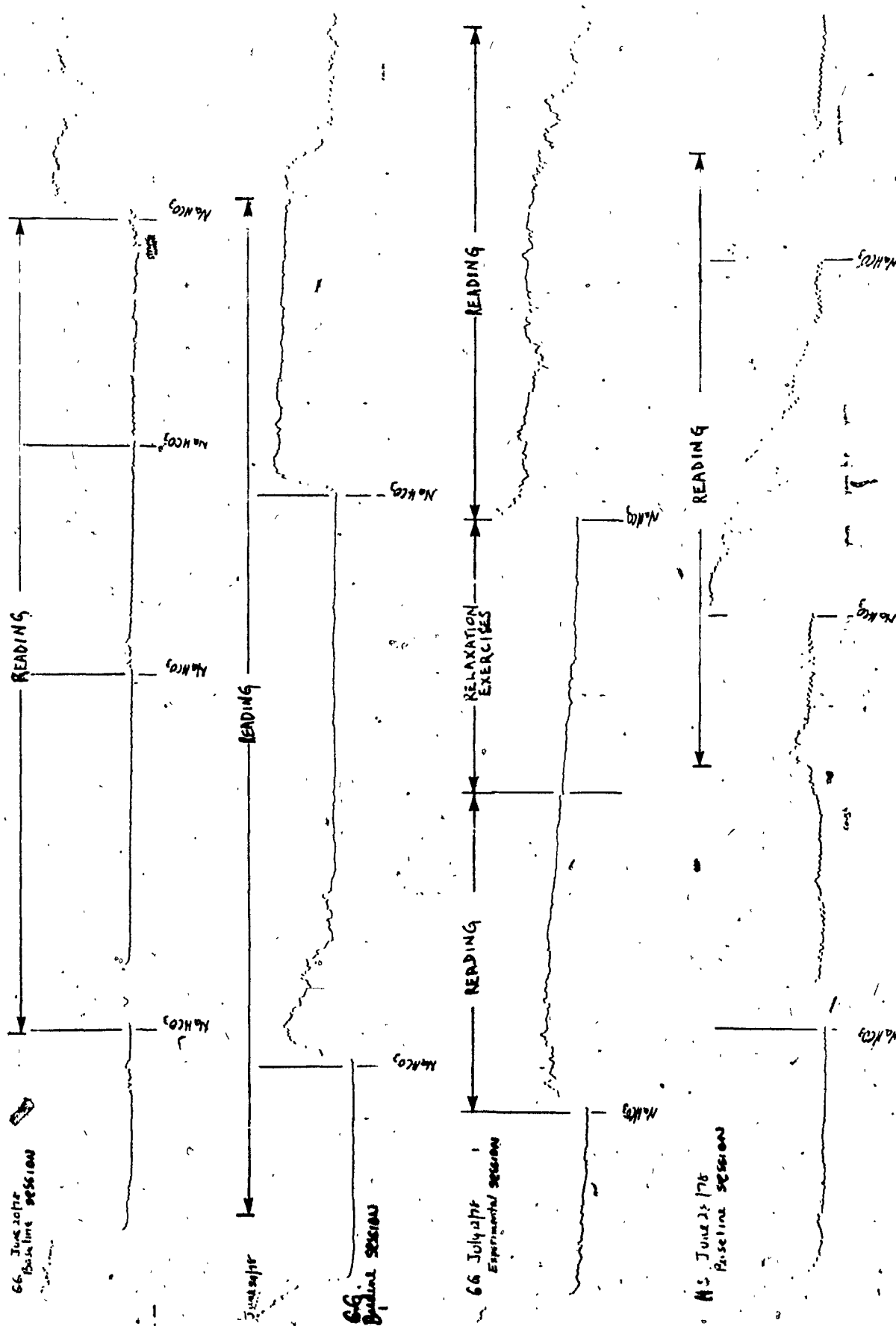
APPENDIX C

D.N.
Feb 24/78
1st training
feedback



Asterisk (*) refers to the period during which subject D.N. reported experiencing unpleasant feelings. Acidity decreased during this period.

APPENDIX D



Upper tracing shows trend towards increase in acidity while subject G.G. was reading material reported as 'unpleasant'. Other tracings show trend to decrease in acidity while subjects read material reported as interesting.