EFFECTS OF SMOKING ON GASTRIC
SECRETION AND GASTRIC
MOTILITY IN MAN

THESIS

Presented to the Graduate Council of the
North Texas State College in Partial
Fulfillment of the Requirements

For the Degree of

MASTER OF ARTS

By

Jess Mack McKenzie, B. A.

Denton, Texas

June, 1956
ACKNOWLEDGMENT

All experiments reported in this thesis were carried out in the Laboratories for Clinical Investigation and Research of the Scott, Sherwood, and Brindley Foundation and the Scott and White Clinic, Temple, Texas.

The advice and aid of Dr. N. G. Hightower, Jr. in development of the problem is gratefully acknowledged.

Appreciation is also due to members of the staff of the Scott and White Clinic who kindly referred patients for study and to those students in the School of Nursing who served as control subjects.
# TABLE OF CONTENTS

**LIST OF TABLES** .......................................................... Page v
**LIST OF ILLUSTRATIONS** ................................................. vi

Chapter

I. INTRODUCTION .............................................................. 1
II. REVIEW OF THE LITERATURE ............................................. 4
III. SUBJECTS, MATERIALS AND METHODS .................................. 14
IV. RESULTS ........................................................................... 22
V. DISCUSSION ........................................................................ 34

APPENDIX .............................................................................. 38

BIBLIOGRAPHY ....................................................................... 47
LIST OF TABLES

Table                                                                 Page

I. Subjects Employed ......................... 38
II. Effects of Smoking on Gastric Secretion ...... 39
III. Effects of Smoking on Gastric Motility in Eight Normal Subjects ............... 40
IV. Effects of Smoking on Gastric Motility in Seven Patients with Duodenal Ulcer .... 41
V. Summary of Effects of Smoking on Gastric Motility .................... 42
## LIST OF ILLUSTRATIONS

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Balloon System Used in Recording of Motility</td>
<td>43</td>
</tr>
<tr>
<td>2.</td>
<td>Schematic Diagram of Balloon-Photokymograph Recording System</td>
<td>44</td>
</tr>
<tr>
<td>3.</td>
<td>Pyrex Glass-spoon Manometer</td>
<td>45</td>
</tr>
<tr>
<td>4.</td>
<td>Composite Tracing Showing the Three Types of Antral Gastric Motility</td>
<td>46</td>
</tr>
<tr>
<td>5.</td>
<td>Continuous Tracing Illustrating Effects of Smoking on Antral Gastric Motility in a Patient with Duodenal Ulcer</td>
<td>46</td>
</tr>
</tbody>
</table>
CHAPTER I

INTRODUCTION

As early as 1805, certain members of the medical profession began to suspect that the use of tobacco contributed to a number of the disorders of man. In a letter (4) to Benjamin Waterhouse of Harvard University, President John Adams commended Dr. Waterhouse for a pamphlet the latter had written concerning the harmful effects of tobacco.

Since that time, the number of tobacco smokers has increased steadily. In a survey of 2,031 medical patients, Short and Johnson (6) in 1939 found that 64 per cent of them used tobacco. They noted that such symptoms as heartburn, excessive gas, and miscellaneous disorders of the gastrointestinal tract, occurred in 38 per cent of those who smoked, whereas only 21 per cent of the non-smokers were affected.

More recently, with increased cigarette smoking among both men and women (5), some physicians have come to regard this particular habit as a factor in carcinoma of the lung, and even in reducing the life span of man.

One of the more firmly established beliefs today is that smoking produces undesirable effects in patients with peptic ulcer. Batterman and Ehrenfeld (1) in 1948 studied
108 ambulatory patients with peptic ulcer in terms of their ability to respond to antacid therapy. They reported that, of 56 patients who continued to smoke after therapy was initiated, only 47 per cent showed a satisfactory response, while 85 per cent of 39 patients who never smoked showed satisfactory improvement. Acute exacerbations of symptoms occurred in 53 per cent of the smokers and in only 18 per cent of the non-smokers.

Although clinical evidence for these effects is convincing, there is little agreement as to the physiological effects of smoking on the stomach of man. The development of new techniques in gastrointestinal physiology (3) and recent concepts of the pathogenesis of peptic ulcer related to hypersecretion and hypermotility (2) warrant a reevaluation of this problem.

This thesis is concerned with the use of some of the newer techniques in a study of some of the effects of cigarette smoking on gastric secretion and gastric motility in normal subjects and in patients with active duodenal ulcer.
CHAPTER BIBLIOGRAPHY


CHAPTER II

REVIEW OF THE LITERATURE

An examination of available literature has revealed little agreement as to the effects of smoking on gastric secretion in man. Some reports have been made that gastric acidity and secreted volume are increased by smoking, whereas others suggest that volume and acidity are decreased or not affected.

It has been stated that smoking produces more of an increase in volume and acidity in patients with duodenal ulcer than in normal controls. Most investigations, however, have shown little difference.

Conflicting reports regarding the effects of smoking on gastric motility have also been noted. In normal individuals some investigations have demonstrated a decrease in motility whereas others have shown little change in activity. When the effects of smoking have been determined for patients with duodenal ulcer, no significant change in motility has been observed.

The literature will be reviewed first, as to the effects of smoking on gastric secretion and second, the effects of smoking on gastric motility.
Gastric Secretion

Gray (4) in 1929 studied the effects of smoking on gastric secretion in man, using two groups of fifty subjects each. The control group consisted of patients with heartburn, pylorospasm, "probable gastritis," and so-called "duodenal ulcer symptom complex" (negative roentgenographic diagnosis for duodenal ulcer). The second group consisted of fifty patients with a positive diagnosis for duodenal ulcer. All subjects were chronic smokers. Gastric secretion was evaluated for each subject by fractional analysis after a test meal. On separate occasions each subject smoked prior to the meal, one hour after the meal, and two hours after the meal. On one other occasion the subject was given a test meal, but did not smoke at any time during the test. In still another experiment the effects of smoking on fasting gastric secretion was determined.

Gray found that smoking while in the fasted state produced an increase in secreted volume in all patients studied. Patients with heartburn showed an increase from six cubic centimeters to eighteen cubic centimeters; increase was from nine to twenty-five cubic centimeters in patients with duodenal ulcer symptom complex, from eleven to twenty-four in those with pylorospasm, and from fifteen to twenty in patients with probable gastritis. Patients with duodenal ulcer exhibited an increase from twenty to thirty-five cubic centimeters.
When the effects of smoking were observed before and after a test meal the incidence of hyperacidity increased only in patients with heartburn and duodenal ulcer symptom complex. This occurred only when the patients smoked two hours after the test meal. In patients with duodenal ulcer the incidence of hyperacidity was unchanged when smoking occurred prior to or after the test meal. Gray defined hyperacidity as an increase in the values for acid and secreted volume as compared to the values obtained with a meal but without smoking.

Gray also reported a study of the effects of smoking de-nicotinized cigarettes. He found no difference in secretory response when patients with duodenal ulcer smoked standard and de-nicotinized cigarettes.

Schnedorf and Ivy (5) made a study of the effects of smoking on the fasting gastric secretion of twenty-five normal chronic smokers, fifteen non-smokers, and twenty chronic smokers with duodenal ulcer. Each subject smoked four to seven cigarettes over a period of one hour and fifty minutes after a control period of forty minutes. In the twenty-five normal subjects who were chronic smokers, no increase occurred. Only one of the fifteen non-smokers showed an increased secretory volume and acidity after smoking. Twenty-two of the forty normal subjects showed a decrease in volume and acidity. Of twenty ulcer patients, one showed an increase in volume and acidity, and eleven
demonstrated a decrease in volume and acidity. Smoking de-nicotine-ized cigarettes produced results which were not significantly different from those obtained when ordinary cigarettes were smoked.

There was no tendency toward conditioned reflex to smoking in ten smokers and ten non-smokers, and it was shown that learning to smoke had no effect on the average acidity of ten non-smokers after they had smoked six cigarettes per day for four to six weeks.

Schnedorf and Ivy also studied the effects of smoking on fasting secretion in four dogs with Pavlov pouches. The animals smoked through small trochars inserted into the trachea. A slight depression in volume and acidity was noted after smoking. Nicotine, in doses equivalent to one, two, and five cigarettes, was injected subcutaneously into fasting dogs with Pavlov pouches, and no change in acid production was noted. In dogs with pouches of the entire stomach, no change in acidity occurred when the stomach was perfused with water saturated with the smoke of two cigarettes. Acidity was depressed, however, when this perfusate was injected into the small intestine.

Three dogs with Pavlov pouches were examined for their response to a test meal with and without subcutaneous doses of nicotine. It was found that nicotine had no effect on their secretory response to the meal.
Ehrenfeld and Sturtevant (3) studied the effects of smoking on the gastric acidity of thirty-three patients without gastrointestinal lesions and twenty-three patients with duodenal ulcer. Each patient was tested on two consecutive mornings for his response to an ethanol test meal. On one of these occasions the subject smoked two cigarettes. A definite increase in acidity of about ten milliequivalents per liter in response to the test meal was shown in 76 per cent of the thirty-three controls when they smoked, 18 per cent showed no change, and 6 per cent exhibited a slight decrease. Of the twenty-three peptic ulcer patients, 87 per cent showed a definite increase in acidity after smoking and 13 per cent of the patients showed no change. In another experiment, ten patients were tested on three successive days: without smoking, after smoking two popular-brand cigarettes, and after smoking partially de-nicotinized cigarettes (0.74 per cent nicotine). Each patient showed a definite increase in acidity after smoking popular-brand cigarettes, whereas after smoking de-nicotinized cigarettes, only five showed a very slight increase in acidity.

A study of standard and filter-type cigarettes was made by Steigman, Dolehide, and Kaminski in 1954 (6) on forty-four hospital controls and fifty-four peptic ulcer patients. All
subjects were tested on two separate occasions for the effects on fasting secretion of smoking the two cigarette types. Of the forty-four controls, twenty-six showed a rise in acidity of ten to sixty milliequivalents per liter after smoking standard cigarettes, but only ten patients showed a rise of ten to sixty milliequivalents after smoking filter-type cigarettes. Of the fifty-four ulcer patients, fifty showed a rise of ten to eighty milliequivalents per liter after standard type cigarettes, whereas after smoking filter-type cigarettes, thirty-two showed a rise of ten to sixty milliequivalents.

Gastric Motility

Dickson and Wilson (2) in 1924 made fluoroscopic observations on gastric motility after a barium meal in thirteen normal men and one individual with a quiescent duodenal ulcer, and reported that gastric motility was slightly decreased after smoking several pipes or cigarettes immediately before or after the barium meal.

In 1925 Danielopolu, Simici, and Dimitriu (1), using a balloon-kymograph technique, studied the effects of cigar smoking on gastric motility in persons twenty to twenty-two years of age, a portion of whom were heavy smokers. After a control period, the subject smoked one cigar, and observations continued for another period. The authors reported a cessation of motility occurring ten to fifteen
minutes after the first few puffs. Durations of the control and test periods were not stated, however; nor was it stated whether the smoke was inhaled. A second cigar produced similar results. Cessation of motility was usually preceded by a short period of hypermotility, which the authors believed to indicate that small amounts of smoke tend to excite and large amounts to depress motility of the stomach.

Gray (4) in 1929 observed the effects of smoking on the peristaltic activity of fifty patients with duodenal ulcer and fifty patients with a variety of gastrointestinal symptoms. With the aid of fluoroscopy and barium meals, he observed the motility of each subject on four occasions. In the first test, the patient did not smoke. In the three remaining tests, the patient smoked prior to, fifteen minutes after, and one hour after a barium meal. Patients with probable gastritis and patients with duodenal ulcer symptom complex exhibited a slight increase in emptying time, but no effect on emptying time was noted in others of the first group when they smoked either before or after the meal. Duodenal ulcer patients exhibited a slight increase in emptying time following smoking.

Smoking caused a slight decrease of peristalsis in patients with heartburn but had no effect on motility in any of the other groups.
Schnedorf and Ivy (5) in 1939 confirmed the observations of Danielopolu et al. (1) that hunger contractions cease after the first few puffs of cigarette smoke and may not recur for fifteen to sixty minutes after smoking is stopped. Details of their methods for recording motility were not given. In another experiment they determined the effect of smoking on the emptying times of test meals. The stomach was first emptied of fasting contents. Then the subject ingested 250 cubic centimeters of beef tea. This procedure was followed in two separate tests. In one test the subject smoked, and in another smoking was not permitted. One hour after the meal was ingested, the stomach was emptied and the volume of the contents measured. The authors reported no significant change in the emptying times of seven normal chronic smokers and of twenty-two patients with duodenal ulcer who were, also, chronic smokers.

Steigman, Dolehide, and Kaminski (6), using a balloon-kymograph technique, studied antral and fundic motility in sixteen male subjects who were recovering from various illnesses, including gastric and duodenal ulcer. A control period of thirty minutes was first recorded. The subjects then smoked one cigarette. This was followed by a second period of thirty minutes, after which a second cigarette was given, and the effects were again recorded for another thirty-minute period. Both fundic and antral tracings were
interpreted according to the height of the contractions. On another occasion, filtered cigarettes were tested for their effects, using the same method.

No consistent change was noted in the fundic motility of six patients nor in the antral motility of ten patients after they each smoked two standard cigarettes. Filtered cigarettes were also found to produce no consistent change.
CHAPTER BIBLIOGRAPHY


CHAPTER III

SUBJECTS, MATERIALS AND METHODS

Subjects

Subjects employed in these studies were divided into a control group of seven young women and one man between the ages of nineteen and twenty-nine and a group of seven patients with duodenal ulcer between the ages of twenty-six and fifty-seven. Three of the ulcer group were women and four were men. All of the ulcer group showed a positive roentgenological diagnosis for duodenal ulcer. None of the patients with duodenal ulcer was obstructed or bleeding at the time of the experiment. All individuals in both groups were chronic smokers and had been smoking for at least six months prior to the tests. The average cigarette consumption of patients with duodenal ulcer was approximately one pack per day. Normal subjects consumed about one-half pack per day. None of the individuals in the normal group had a history of previous duodenal ulcer or symptoms of gastrointestinal disease. In the group of patients with duodenal ulcer all medication was withheld for twelve hours prior to the observations. The above information is summarized in Table I.
All subjects reported to the laboratory after a fasting period of at least eight hours, and all but a few had not smoked for at least seven hours. Tests were postponed for one hour or more when subjects admitted smoking against instruction.

Intubation Techniques

A cylindrical Sawyer balloon of thin latex rubber was attached with rubber cement to the distal end of a Miller-Abbot tube approximately one meter in length. The balloon was five centimeters in length, three centimeters in diameter, and had an undistended capacity of approximately thirty-five milliliters. One lumen of the Miller-Abbot tube opened into the balloon. The other lumen was opened proximal to the balloon by cutting several small holes, through which gastric juice could be aspirated. The tube is illustrated in Figure 1.

The subjects were intubated with the balloon undistended. When the gag reflex was excessive, the throat was sprayed with a 2% per cent solution of tetracaine hydrochloride before intubation. All subjects were requested to expectorate their saliva throughout the test. After intubation the balloon was weighted with thirty cubic centimeters of water and placed in the antrum with the aid of fluoroscopy. Subjects remained on the X-Ray table in the supine position throughout the remainder of the test.
Gastric secretion and antral motility were recorded for a control period of fifty minutes. The subjects then smoked one cigarette for a period of ten minutes or less. This was followed by another observation period lasting sixty minutes. Cigarettes employed in the tests were of standard size and were not filtered.

Recording of Motility

Motility was recorded by the method of Hightower (2) using a photokymograph recorder equipped with glass-spoon manometers previously described by Kubicek, Sedgewick, and Visscher (4).

After the balloon was located in the pyloric antrum, it was emptied of all water and air. The balloon was then filled with water under a pressure head of fifteen centimeters of water. The apparatus (Figure 2) used for filling the balloon consisted of a glass cylinder of two hundred milliliters capacity, which contained approximately seventy-five milliliters of water and was connected to a buret, a water reservoir, and a glass-spoon manometer. The buret was calibrated in centimeters and was used to determine the pressure head during filling of the balloon. The water reservoir was calibrated in milliliters, and readings were made before and after filling the balloon, in order to measure the volume of water which was placed into the balloon and tube. The upper portion of the cylinder, the
glass-spoon manometer, and the tubing connecting them contained air, which acted as a compressible cushion between the balloon and the manometer. Without this cushion of air the balloon is virtually incompressible.

The glass-spoon manometer (Figure 3) is linear in response to pressure changes, has great sensitivity, and exhibits little hysteresis (2,4). To the tips of the manometers were attached small planoconvex mirrors having a focal length of one meter. One manometer, which showed a deflection of eleven millimeters per ten centimeters of water pressure, was used to record gastric motility in all experiments.

Another glass-spoon manometer was attached with plastic tubing to a rubber pneumograph tube, which was placed around the subject's chest during each experiment. This was necessary, since pressure changes within the abdominal and thoracic cavities are known to affect intragastric pressure (1). The spoon manometers attached to the balloon and pneumograph tube were fixed in the photokymograph apparatus at a distance of one meter from a prismatic lens, behind which moved a strip of photostat paper. This paper was 11.5 inches (29.2 centimeters) wide and was moved at a uniform rate of 0.47 millimeters per second by an electric motor. The speed of the film was controlled by a gear box mounted on the side of the camera.
The spoon mirrors received light from two shielded light sources mounted on either side of the camera housing. Another mirror was attached to a small iron rod, and this was fixed at one meter from the lens in order to obtain a baseline on the photostat paper. A typical motility record is shown in Figure 4.

Time lines were obtained on the photostat paper by means of a flashing timer light, which was connected to an electric timing system capable of thirty-second, ten-second, and one-minute impulses. Most of the recordings were made using the one-minute time lines; a few were made with the thirty-second timer. All timing systems were pre-calibrated with a stop watch.

Analysis of Motility Records

A method, described by Hightower and Code (1), for the quantitative analysis of antral gastric motility records has been followed in the analyses of records obtained in the present study.

The durations of control periods and of periods following smoking were noted. All waves were counted and typed according to their amplitude, duration, and form. Amplitude was expressed in centimeters of water and duration in seconds. For each period of observation the mean values of amplitude and duration were noted. Total activity for each
period was obtained by dividing the sum of the durations of Type I and Type II waves by the total duration of the period.

Measurement of Gastric Secretion

Samples of gastric juice were drawn every ten or fifteen minutes during the experiment. Approximately eight samples were taken during the entire test.

Samples were then filtered through surgical gauze and set aside in the icebox. Not more than six hours after termination of the test, one milliliter aliquots were taken from all samples and titrated for free HCl and total acidity (3, p. 386) with N/10 NaOH. Titrations were made with a 2.0 milliliter microburette. The indicators used were Toepfer's reagent and Phenol phthalien, respectively.

Chloride concentration was determined for one milliliter aliquots of each sample using a modification of the Shales and Shales method for blood (3, p. 626). Titrations were made with a 2.0 milliliter microburette. Diphenylcarbazone was used as the indicator and mercuric nitrate (100 m.eq. per liter) employed as the reagent. It was later found that addition of one-tenth milligram of Bromphenol blue to each ten milliliters of diphenylcarbazone solution produced much sharper end-points than those obtained with diphenylcarbazone alone.
In statistical analyses (5,6) of motility and secretion data, was calculated for coefficients of correlation and differences between paired and unpaired means.
CHAPTER BIBLIOGRAPHY


A total of twelve normal subjects and nine patients with duodenal ulcer were tested in these experiments. Later, one patient with duodenal ulcer was also found to have a gastric carcinoma, and his record was deleted from the final data. Four of the normal subjects and one patient with duodenal ulcer became nauseated after they inhaled several puffs of cigarette smoke, and three of these regurgitated the tube. It was impossible to aspirate the gastric contents of two of the remaining eight normal subjects and one of the remaining seven patients with duodenal ulcer. Thus, motility data for eight normal subjects and seven patients with duodenal ulcer, and the secretion data for only six normal subjects and six patients with duodenal ulcer were available for final consideration.

Results obtained in secretion studies will be described first. This will be followed by an outline of the results obtained in studies of gastric motility.

Gastric Secretion

Approximately seven samples of gastric contents were drawn from each of the six normal subjects and six patients
with ulcers. Four samples were taken during the control period and three during the period after smoking.

Since samples could not be taken at exact times in relation to the smoking period, and since secretory changes over a fifteen minute period did not appear significant, values obtained for free HCl, total acidity, and chloride concentration were pooled and expressed as mean values for the control period and for the period after smoking in studies of both groups.

**Basal Secretion**

**Six normal subjects.**—A few samples of the basal secretions of these subjects contained bile, but its presence did not seem to appreciably affect the secretion data obtained.

During the control period, values for chloride concentration ranged from 74.2 to 133.2 milliequivalents per liter. Mean chloride concentration was 89.1. Free HCl varied from 12.5 to 32.8 milliequivalents per liter and the mean value was 22.6. Total acidity fluctuated between 23.3 and 45.3 milliequivalents per liter. Mean total acidity was 32.8. A summary of basal secretion values for normal subjects is given in Table II.

**Six patients with duodenal ulcer.**—All six patients in this group yielded copious amounts of gastric juice and much
more bile was found in samples for this group than in the gastric contents of any of the six normal subjects.

Chloride concentration varied between 94.7 and 123.5 m.eq. per liter, and the mean was 97.7. Free HCl values ranged from 4.4 to 68.0 m.eq. per liter. Mean free HCl was 36.5. The patient in which mean HCl was 4.4 exhibited consistently low values throughout the test. Values for total acidity fluctuated between 12.8 and 77.25 m.eq. per liter. Mean value was 44.4.

**Effects of Smoking on Gastric Secretion**

**Six normal subjects.**--Chloride concentration varied from 93.4 to 139.3 m.eq. per liter during the period after smoking with a mean of 104.7. This represented a mean increase of 15.6 m.eq. which was significant at the 2.0 per cent level. Free HCl varied between 22.7 and 37.3 m.eq. per liter, and the mean value for HCl was 31.6. The mean increase after smoking was 9.0 m.eq. which was significant at the 5.0 per cent level. Total acidity values ranged between 31.3 to 54.9 m.eq. per liter; mean total acidity was 44.4, representing a mean increase of 11.6 m.eq. which was significant at the 5.0 per cent level. Mean secretion values during both observation periods are given in Table II.

**Six patients with duodenal ulcer.**--Chloride concentration during the period after smoking varied between 70.2 and
122.7 m.eq. per liter. Mean chloride concentration was 106.0, representing a mean increase of 9.3 m.eq.

Free HCl values were between 10.6 and 61.0 m.eq. per liter. Mean value was 38.3 and represented an increase of only 1.8 m.eq.

Total acidity ranged between 22.0 and 78.0 m.eq. per liter during this period. Mean total acidity was 52.0. The mean increase was 8.4 m.eq.

None of the increases noted in the gastric secretion of patients with duodenal ulcer was statistically significant.

All values for gastric secretion are summarized in Table II.

Gastric Motility

Motility tracings satisfactory for analysis were obtained from eight normal subjects and seven patients with duodenal ulcer. In a few of these records it was necessary to delete small segments of tracings from analysis because of excessive artifacts due to paroxysms of coughing.

One of the eight normal subjects who was used only in motility studies smoked two cigarettes during the smoking period. Motility values obtained from the tracing of this subject matched closely those values obtained from the remaining seven normal subjects who smoked only one cigarette.
Data from all eight subjects were used in the analysis of the effects of smoking.

**Basal Motility**

The control periods for gastric motility studies ranged between fifty and sixty minutes for normal subjects and from forty-two to sixty-three minutes for patients with duodenal ulcer. Mean value for the control period was fifty-three and three tenths minutes for normal subjects and fifty-five and six tenths minutes for patients with duodenal ulcer.

Three types of waves were found in the motility tracings of both groups. A composite tracing, illustrating all three types may be found in Figure 4.

**Type I motility.**—Waves representing pressures below five centimeters of water were designated as Type I waves. These waves occurred in both rhythmic and non-rhythmic patterns. Rhythmicity was constant at three waves per minute.

**Normal group:** Non-rhythmic type I waves in this group had a mean duration of 14.9 seconds. Their mean number per hour was 17.8, and they were present an average of 7.3 percent of the time. Rhythmic waves had a mean duration of 14.3 seconds, and their mean number per hour was 12.4. They were present an average of 5.5 percent of the time.
Ulcer group: Non-rhythmic Type I waves had a mean duration of 16.1 seconds, their mean number per hour was 12.2, and they were present an average of 5.7 per cent of the time. Rhythmic waves had a mean duration of 11.5 seconds, a mean number per hour of 28.4 and were present an average of 11.5 per cent of the time.

Type II motility.--Simple waves, representing pressures greater than five centimeters of water were designated as Type II waves. These waves occurred both in rhythmic and non-rhythmic patterns. When rhythmic, they usually occurred at a rate of three per minute, but a few patterns were seen in which the rate was one per minute.

Normal group: Mean duration of non-rhythmic Type II waves was 22.6 seconds. Their mean pressure was 19.1 centimeters of water, and their mean number per hour 20.7. They occurred an average of 4.6 per cent of the time. Rhythmic waves had a mean duration of 22.9 seconds, and a mean pressure of 21.2 centimeters of water. Their mean number per hour was 23.0. They occurred an average of 15.3 per cent of the time.

Ulcer group: Non-rhythmic Type II waves in this group had a mean duration of 29.5 seconds and a mean pressure of 20.9 centimeters of water. Their mean number per hour was 12.0, and they occurred an average of 9.0 per cent of the time. Rhythmic Type II waves had a mean duration of 23.5
seconds, a mean pressure of 23.6 centimeters of water, and their mean number per hour was 28.4. They occurred an average of 19.3 per cent of the time.

**Type III motility.**—Type III waves are thought to represent elevations in basal tonus of the gastric antrum. These waves are complex in that Type II waves are almost always superimposed upon them. These waves were present in the tracings of only a few subjects in each group, and because of their random occurrence they can only be considered as non-rhythmic. For these same reasons, these waves were not considered when the effects of smoking were evaluated.

**Normal group:** Type III waves were found in only a few tracings of this group. They had a mean duration of 144.9 seconds and a mean pressure of 5.9 centimeters of water. Their mean number per hour was 0.9, and they occurred an average of 2.5 per cent of the time.

**Ulcer group:** Type III waves occurring in the central tracings of this group had a mean duration of 87.3 seconds. Their mean pressure was 6.8 centimeters of water, and their mean number per hour was 1.5. They occurred an average of 6.3 per cent of the time.

**Total motility.**—Total motility for each control period was calculated by adding the total per cent of time present of all Type I waves to that of all Type II waves.
Total motility of the control periods was 42.7 per cent for normal subjects and 45.5 per cent for patients with duodenal ulcer. The difference between these mean values was not statistically significant.

Mean motility values for control period are given in Tables III and IV.

The motility values obtained in control studies of normal subjects corresponded closely to those found by Hightower (2) in twenty-five normal subjects.

**Effects of Smoking on Gastric Motility**

In studies of normal subjects the period following smoking ranged from 42 to 57 minutes. The mean period was 56 minutes. The period after smoking in studies of patients with duodenal ulcer varied between 35 and 61 minutes. The mean duration of this period was 57.2 minutes.

A sample motility tracing showing the effects of smoking on the gastric motility of one patient with duodenal ulcer is shown in Figure 5.

**Type I motility.** --Type I waves were present a total of 16.1 per cent of the time in normal subjects and 13.1 per cent in patients with duodenal ulcer.

**Normal group:** Non-rhythmic Type I waves exhibited a mean duration of 15 seconds; their mean number per hour was 17.1. They were present 7.2 per cent of the time. Rhythmic
Type I waves had a mean duration of 15.1 seconds. Their mean number per hour was 22.4, and they were present 9.6 per cent of the time.

Ulcer group: Non-rhythmic Type I waves had a mean duration of 16.3 seconds, their mean number per hour was 16.1, and they were present 7.2 per cent of the time. Rhythmic Type I waves had a mean duration of 15.5 seconds, and their mean number per hour was 13.1. They were present 5.9 per cent of the time.

Smoking had no effect on the rhythmicity of Type I waves in either group.

Type II motility.—Type II waves were present a total of 12.9 per cent of the time in normal subjects and 15.2 per cent in patients with duodenal ulcer. The changes in both groups were significant at the 5 per cent level.

Normal group: Non-rhythmic type II waves had a mean duration of 18.8 seconds, a mean pressure of 15.9 centimeters of water, and their mean number per hour was 13.5. They were present 8.2 per cent of the time. Rhythmic Type II waves had a mean duration of 22.5 seconds. Their mean pressure was 18.6 centimeters of water, and their mean number per hour was 7.8. They occurred 4.7 per cent of the time, showing a mean decrease after smoking of 10.6. This change was significant at the 2.0 per cent level.
Ulcer group: Non-rhythmic Type II waves had a mean duration of 25.3 seconds and a mean pressure of 16.2 centimeters of water. Their mean number per hour was 10.9. They were present 7.3 per cent of the time. Rhythmic Type II waves had a mean duration of 24.9 seconds, a mean pressure of 26.5 centimeters of water, and their mean number per hour was 7.9. They were present only 7.9 per cent of the time, a mean decrease of 11.4 which is significant at the 2.0 per cent level.

Type III motility.--Results obtained for Type III motility were not considered in the final analysis of the effects of smoking.

Normal group: No Type III waves were found in the motility tracings of normal individuals during periods after smoking.

Ulcer group: Type III waves found in tracings of this group had a mean duration of 75.0 seconds, a mean pressure of 6.8 centimeters of water, and their mean number per hour was 1.0. They occurred 2.8 per cent of the time.

The effects of smoking on gastric motility are summarized in Tables III, IV, and V.

Total motility.--Smoking decreased total motility by decreasing the per cent of time present of rhythmic Type II waves. The decreases in total motility in both groups were significant at the 5.0 per cent level. Other differences in
mean values were analyzed statistically and found insignificant. A correlation of the decrease in motility with increased secretory values was not statistically significant.
CHAPTER BIBLIOGRAPHY


Generally, the results of these present experiments agree with most previous reports. Some disagreement exists, however, between these findings and those of Schnedorf and Ivy (4) concerning the effects of smoking on gastric acidity; nor do these results entirely support statements made by others that smoking produces greater secretory responses in patients with duodenal ulcer than in normal individuals. A comparison of such results is impractical, however, because of differences in experimental methods. Few investigators (3,4,5) have studied the effects of smoking on fasting gastric secretion, and most methods have been further complicated by the employment of test meals (3), secretagogues (4), and excessive smoking (4).

Most studies of the effects of smoking on gastric motility have employed fluoroscopic examination after ingestion of a barium meal. A few authors (1,5) have used balloon-kymograph methods, although none has interpreted the data quantitatively. One report (5) has more closely approached the quantitative method by interpreting motility tracings according to the heights of the waves.
When two variables such as gastric secretion and gastric motility are studied, the importance of simultaneous determination cannot be overemphasized. By using such simultaneous recording techniques, the present work has established that smoking produces a dissociation of gastric secretion and gastric motility. Thus, normal subjects experience a decrease in gastric motility after smoking, but their gastric acidity is increased. No explanations for this paradox are apparent at this time, but further research along these same lines may lead to more complete interpretations of these results.

Summary

An examination of available literature has revealed little agreement as to the effects of smoking on gastric secretion and gastric motility. It was thought that more concise results might be obtained by simultaneous measurements of secretion and motility, together with a more quantitative analysis of motility tracings.

The effects of smoking on gastric secretion and gastric motility were determined in eight normal subjects and seven patients with duodenal ulcer. All subjects were chronic smokers. None of the normal subjects gave a history of previous duodenal ulcer or had symptoms of gastrointestinal disease. Patients with duodenal ulcer had no other complications.
Motility was recorded by means of a water-filled balloon system and a photokymograph employing glass-spoon manometers. Samples of gastric contents were taken at fifteen minute intervals and titrated for free HCl, total acidity, and chloride concentration. Each experiment was divided into a control period of fifty minutes, followed by a ten minute period during which the subject smoked one cigarette, and a final period of sixty minutes.

Gastric acidity was significantly increased by smoking in normal subjects, but no significant change in acidity was noted in patients with duodenal ulcer.

Smoking produced a significant decrease in the gastric motility of each subject tested by reducing the number of rhythmic Type II waves.

Explanations for the dissociation of motility and secretion in normal subjects after smoking are not apparent at this time.
CHAPTER BIBLIOGRAPHY


2. Ehrenfeld, I. and Sturtevant, M., "The Effect of Smoking Tobacco on Gastric Acidity," American Journal of the Medical Sciences, CCI (January, 1941), 81-86.


<table>
<thead>
<tr>
<th>Subjects</th>
<th>Sex</th>
<th>Mean age (years)</th>
<th>Mean consumption (cig. 1 day)</th>
<th>Years smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 Normal subjects</td>
<td>7 F</td>
<td>20.4</td>
<td>11.4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>1 M</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 Patients with duodenal ulcer</td>
<td>3 F</td>
<td>45.6</td>
<td>20</td>
<td>26.2</td>
</tr>
<tr>
<td></td>
<td>4 M</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### TABLE II

EFFECTS OF SMOKING ON GASTRIC SECRETION

<table>
<thead>
<tr>
<th>Determination</th>
<th>Condition</th>
<th>6 Normal subjects</th>
<th>6 Duodenal ulcer patients</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chloride Conc.</strong>*</td>
<td>Before smoking</td>
<td>89.1</td>
<td>97.7</td>
</tr>
<tr>
<td></td>
<td>After smoking</td>
<td>104.7 **</td>
<td>106.0</td>
</tr>
<tr>
<td>**Free HCl ***</td>
<td>Before smoking</td>
<td>22.6</td>
<td>36.5</td>
</tr>
<tr>
<td></td>
<td>After smoking</td>
<td>31.6 *</td>
<td>38.3</td>
</tr>
<tr>
<td>**Total Acidity ***</td>
<td>Before smoking</td>
<td>32.8</td>
<td>44.4</td>
</tr>
<tr>
<td></td>
<td>After smoking</td>
<td>44.4 *</td>
<td>52.0</td>
</tr>
</tbody>
</table>

* denotes change significant at 5.0% after smoking
** denotes change significant below 5.0% level after smoking
*** milliequivalents per liter
### TABLE III

**EFFECTS OF SMOKING ON GASTRIC MOTILITY IN 8 NORMAL SUBJECTS**

<table>
<thead>
<tr>
<th>Condition of Observation</th>
<th>Non-Rhythmic</th>
<th>Rhythmic</th>
<th>Total Perzent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Observed time (Min.)</td>
<td>Time present</td>
<td>No. per.</td>
</tr>
<tr>
<td><strong>Type I Motility</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before smoking</td>
<td>53.3</td>
<td>7.3</td>
<td>17.8</td>
</tr>
<tr>
<td>After smoking</td>
<td>56.0</td>
<td>7.2</td>
<td>17.1</td>
</tr>
<tr>
<td><strong>Type II Motility</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before smoking</td>
<td>53.3</td>
<td>14.6</td>
<td>20.7</td>
</tr>
<tr>
<td>After smoking</td>
<td>56.0</td>
<td>8.2</td>
<td>13.5</td>
</tr>
</tbody>
</table>

* denotes change significant at 5% level
** denotes change significant below 5% level

Horizontal lines over "observation, pressure, and duration" indicate mean amounts.
TABLE IV
EFFECTS OF SMOKING ON GASTRIC MOTILITY IN 7 PATIENTS WITH DUODENAL ULCER

<table>
<thead>
<tr>
<th>Condition of Observation</th>
<th>Non-Rhythmic</th>
<th>Rhythmic</th>
<th>Total Percent of Time Present</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time (min.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before smoking</td>
<td>55.6</td>
<td>5.7</td>
<td>12.2</td>
</tr>
<tr>
<td>After smoking</td>
<td>57.2</td>
<td>7.2</td>
<td>16.1</td>
</tr>
</tbody>
</table>

Type I Motility

| Before smoking           | 55.6          | 9.0      | 12.0                        | 20.9 | 28.5 | 19.3 | 28.4 | 23.6 | 23.5 | 28.3 |
| After smoking            | 57.2          | 7.3      | 10.9                        | 16.2 | 25.3 | 7.9**| 11.1 | 26.5 | 24.9 | 15.2*|

Type II Motility

* denotes change significant at 5% level
** denotes change significant below 5% level

Horizontal lines over "observation, pressure, and duration" indicate mean amounts.
<table>
<thead>
<tr>
<th>Experimental groups</th>
<th>Condition</th>
<th>Type I (%T)*</th>
<th>Type II (%T)*</th>
<th>Type III (%T)*</th>
<th>Total (%T)* (Not including Type III)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 Normal subjects</td>
<td>Before smoking</td>
<td>12.8</td>
<td>29.9</td>
<td>2.5</td>
<td>42.7</td>
</tr>
<tr>
<td></td>
<td>After smoking</td>
<td>16.1</td>
<td>12.9***</td>
<td>---</td>
<td>29.0***</td>
</tr>
<tr>
<td>7 Patients with duodenal ulcer</td>
<td>Before smoking</td>
<td>17.2</td>
<td>28.3</td>
<td>6.3</td>
<td>45.5</td>
</tr>
<tr>
<td></td>
<td>After smoking</td>
<td>13.1</td>
<td>15.2***</td>
<td>2.8</td>
<td>28.3</td>
</tr>
</tbody>
</table>

* Total per cent of time present for both rhythmic and non-rhythmic waves.
** Denotes change significant at 5.0 % level
*** Denotes change significant at 2.0 % level
Fig. 1--Balloon system used in recording of motility. Note small holes in one side of tube for aspiration of gastric contents.
Fig. 2--Schematic diagram of balloon-photokymograph recording system. Clamps 1, 2, and 3 are closed when motility is being recorded.
Fig. 3—Pyrex glass-spoon manometer. Increased pressure within the spoon reduces its concavity. Note mirror attached to manometer tip. Two views are shown.
Fig. 4--Composite tracing showing the three types of antral gastric motility. Types I and II in these are rhythmic.

Fig. 5--Continuous tracing illustrating effects of smoking on antral gastric motility in a patient with duodenal ulcer.
BIBLIOGRAPHY

Books


Articles


Ehrenfeld, I., and Sturtevant, M., "The Effect of Smoking Tobacco on Gastric Acidity," *The American Journal of the Medical Sciences*, CCI (January, 1941), 81-86.
Gray, I., "Gastric Response to Tobacco Smoking," American Journal of Surgery, XII (October, 1929), 489-493.


