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DISCUSSION

DEAN E. GODWIN, M.D. (820 Professional Building, Long Beach).—The Program Committee invited Doctor Houck to present this subject before our section because it was felt that the importance of this danger has not been sufficiently emphasized to the otolaryngologist.

Doctor Houck has presented his subject well, and we are indebted to him for stressing the dangers of this condition. When he states that the condition is terminal, as a rule, in the infant, and frequently fatal in the adult, we should each think how much we personally have contributed to this danger among our patients.

Many otolaryngologists routinely prescribe oily sprays; many pediatricians frequently prescribe plain or medicated liquid petrolatum for instillation in infants' noses by a medicine dropper. Too many laymen think that if a little medicine is good, more is better, and use these oils much more freely than the physician intends.

Doubtless there are many more cases of this condition than have been diagnosed or suspected. The carefully worked-out case presented by Doctor Houck is startling in its findings, but is only an example of what may be going on in some of our patients.

It behooves us, then, to carefully weigh the dangers of indiscriminate nasal or intratracheal instillations of oil or the careless prescribing of oily medicaments to our patients.

CIGARETTE SMOKING*

ITS EFFECT ON THE VOLUME AND ACIDITY OF THE GASTRIC JUICE, WITH PARTICULAR REFERENCE TO DUODENAL ULCER

By HAROLD ROSENBLUM, M.D.
San Francisco

DISCUSSION *by Arthur L. Bloomfield, M. D., San Francisco; Allan L. Cohn, M. D., San Francisco; Harold Lincoln Thompson, M. D., Los Angeles.*

THE medical literature contains frequent references to the proscription of tobacco smoking in the management of patients with peptic ulcer. This attitude has been derived largely from clinical observation, and has been handed down by authorities whose opinions have been considered worthy of respect and repetition. Many distinguished medical writers [1-4] have maintained the belief that tobacco smoking may aggravate the symptoms of peptic ulcer, or, further, that smoking may predispose to this disease.

Comment has been made, notably by Trowell [5] and by Johnson,[6] that such opinions have been held in spite of the absence of scientific proof that the smoking of tobacco is harmful to patients with ulcer.

It has been shown [4,7] that the inhalation of tobacco smoke by smokers has an appreciable effect on the peristaltic contractions of the stomach, as demonstrated by kymographic registration. This effect is evidenced by a period of hypercontractility which continues for about fifteen minutes after the beginning of the inhalation of cigarette smoke, and which is followed by a 45-minute period of atony.

The volume and acidity of the gastric juice of patients with functional and with organic gastrointestinal disease have been studied by Gray.[8] His findings indicated a variable response to tobacco in the patients with functional disease. In patients with duodenal ulcer, smoking was usually followed by an increase in the volume of gastric juice which was associated, although not constantly, with increase in its acidity.†

Skin-testing of patients with peptic ulcer for allergic sensitivity to tobacco or its extracts has shown no significant relationship.[8-12]

Search for a statistical correlation between the habit of smoking tobacco and the incidence of peptic ulcer has been productive of only negative results by investigators in both England [5] and America.[13] No etiological relationships could be discovered by this method of study.

PROBLEM AND METHOD

The present study was undertaken to determine the immediate effect of the smoking of a single cigarette upon the acidity and volume of the gastric juice of fasting patients with and without peptic ulcer. Conditions of the experiment were controlled so that comparison could be made between the "ulcer" and "non-ulcer" groups. The effect of the tobacco was compared with the response produced by the subsequent administration of a dose of alcohol adequate to produce definite increase in the acidity of the gastric juice.

The following technique was used:

The patients to be used as subjects for the experiment had been instructed to abstain from eating, drinking, and smoking for at least twelve hours preceding the test, which was started at 8:30 a. m. A Rehfuss tube was introduced into the patient's stomach, and all gastric contents were removed, mixed, measured for volume, and were then reintroduced through the tube into the patient's stomach, save for 10 cubic centimeters which were retained for chemical examination.‡ The subject was then asked to smoke a cigarette, using any popular American brand. Twenty minutes after smoking was begun, all of the gastric content was aspirated for examination. Fifty cubic centimeters of 7 per cent ethyl alcohol were then instilled through the tube, and at the end of twenty minutes 10 cubic centimeters of the gastric juice were removed for study. The total volume of the gastric content was not determined at this stage, the last specimen being used only for determination of acidity. The possibility that an effect might be produced by the passage of the tube and the reinjection of gastric secretion was controlled by study of a group of patients in whom these procedures were

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* From the Departments of Medicine of the Mount Zion Hospital and of the University of California Medical School, San Francisco.

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† Since this paper has been in press, experiments have been reported (Bernay, P., and Faure, G.: Decaffeinized Coffee and Denicotinized Tobacco in Gastric Diet, Arch. d. Malad. de l'App. Digest, 27:865, 1937) which indicate that tobacco, including "denicotinized" tobacco, acts as a powerful stimulant to the gastric secretions as to both its acidity and volume.

‡ In these experiments the concentration of free and total hydrochloric acid in the gastric juice was expressed in terms of the number of cubic centimeters of tenth normal sodium hydroxid (stated in the text as degrees of acidity) necessary to neutralize 100 cubic centimeters of gastric juice, using dimethyl amido-azobenzol and phenolphthalein as indicators.

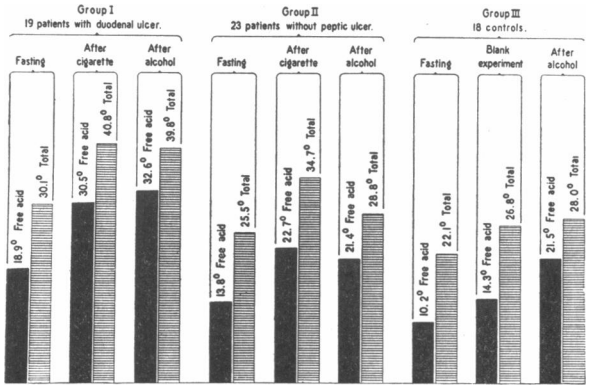

Table 1.—The response of gastric juice acidity to the smoking of a cigarette followed by 50 cubic centimeters of 7 per cent alcohol (groups 1 and 2), and to the same dose of alcohol alone (group 3). (Ewald degrees—gastric acidity.)

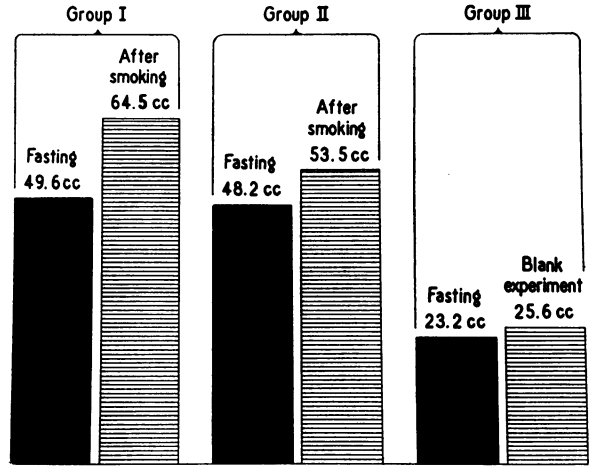

Table 2.—The fasting volume of the gastric juice compared with the volume after smoking.

carried out, but with omission of the smoking of the cigarette.

Thus, three groups were studied: (1) Nineteen patients (twelve males and seven females) with duodenal ulcer; (2) twenty-three patients (twelve males and eleven females) with miscellaneous gastrointestinal conditions, functional and organic, but without peptic ulcer or carcinoma; and (3) eighteen patients (four males and fourteen females), similar to those in group (2) (with the exception of two who had duodenal ulcer), who were used as a control group. All patients in groups (1) and (2) were chronic cigarette smokers who habitually inhaled the smoke. No patient with gastric ulcer, Addisonian anemia, or achlorhydria after the administration of alcohol, or who had had a gastroenterostomy, was included in any of the groups. The average age of the patients in each of the three groups was forty-three years. The total number of patients in the three groups was thirty-eight males and twenty-two females.

FINDINGS

Group 1. *Cases of Duodenal Ulcer.*—In fifteen of the nineteen patients with duodenal ulcer, the smoking of a cigarette caused a substantial average increase in the free and total hydrochloric acid in the gastric juice. In twenty minutes following the beginning of smoking, the average free acidity rose from 18.9 to 30.5 degrees, an increment of 11.6 units. In the same period the average total acid increased from 30.1 to 40.8 degrees, a gain of 10.7 units. These changes (which can be seen in Table 1) represent increases of 61 per cent in the free, and 35 per cent in the total acid. Four patients showed little or no change. The subsequent administration of 50 cubic centimeters of 7 per cent alcohol produced practically no further increase in acidity. This stimulus, acting during the second twenty-minute period, resulted in an average rise of only 2.1 degrees of free, and a drop of 1.0 degree of total acidity. In fact, ten of the nineteen patients in this group showed less acidity, both free and total, twenty minutes after the administration of alcohol than they had had twenty minutes after beginning to smoke a cigarette. There were two instances in which lower concentrations of acid were found after both tobacco and alcohol had been used than the acidity which had been noted when the patients were in a fasting state.

In twelve cases there was an average increase in the total volume of gastric secretion from 44.5 to 72.1 cubic centimeters. In six other cases there was an average decrease from 62.2 to 54.4 cubic centimeters. In the one remaining case, in which the fasting content had measured 35 cubic centimeters, there was no change in volume. The average volume of the gastric juice in the entire group of nineteen patients with duodenal ulcer, was found to have increased after smoking from 49.6 to 64.5 cubic centimeters (Table 2). When it is remembered that this large volume of gastric juice also had a higher concentration of acid, it becomes apparent that the increment represented by greater volume and higher acidity is considerable.

Group 2. *"Non-ulcer" Cases.*—Twenty-three patients with miscellaneous gastro-intestinal conditions, but without peptic ulcer, were studied similarly. As before, the effect of smoking on the gastric juice was determined; and the response to subsequent instillation of alcohol was noted also. The average fasting acid was found to be 13.8 degrees of free and 25.5 degrees of total acid—which are values, of course, lower than those found in the patients with ulcer. After smoking, titration of the gastric juice showed 22.7 degrees and 34.7 degrees, respectively, of free and total acid, indicating increments of 8.9 degrees in the free and 12 degrees in the total acidity. As might be expected, the average increments were not as great as those found in the patients with duodenal ulcer, although the increases in percentage over the acidity of the fasting specimens were approximately the same as those found in Group 1. In Group 2, the rise in concentration of acid following smoking occurred in fifteen of the twenty-three patients. There were three cases in which the degree of acidity was not affected; and five others in which slightly lower values for acidity were found after smoking (Table 1).

The effect of smoking on the total volume of gastric juice was negligible, the average gastric content decreasing from 48.2 to 44.3 cubic centimeters.

As in Group 1, the administration of alcohol subsequent to the smoking period produced no further increase in concentration of acid (Table 2). The acidity twenty minutes after instillation of alcohol was, in fact, slightly less than it had been twenty minutes after the beginning of inhalation of cigarette smoke.

Group 3. *Controls.*—The eighteen patients in this group were used to study the possible effect on the gastric juice of the presence of the stomach tube and of the aspiration and reinjection of gastric contents. The smoking of the cigarette was omitted, but otherwise the experimental procedures were the same as those used with Groups 1 and 2. The administration of alcohol was included so that its effect on patients who had not smoked previously could be compared with its effect on the other groups. In Group 3, the average values for free and total acid in the fasting state were 10.2 and 22.1 degrees, respectively. After aspiration and reinjection, but without smoking, the values (Table 1) were 13.4 and 26.8, respectively. This lack of any material change was, however, in marked contrast to the brisk rise in acidity, following the instillation of alcohol, to levels of 21.5 of free and 28.0 degrees of total acid. Determinations of the volume of the gastric secretion (Table 2) showed no appreciable change following the control experiment.

CONCLUSIONS

1. Cigarette smoking by patients in a fasting state produced a significant average increase in the free and total acidity of the gastric juice in a group of nineteen patients with duodenal ulcer; and, to a somewhat less extent, in a group of twenty-three patients without duodenal ulcer.

2. The magnitude of the average increase in acidity after the smoking of tobacco was such that the subsequent administration to each patient of 50 cubic centimeters of 7 per cent ethyl alcohol produced no further rise.

3. A control study of eighteen patients was made, with the smoking omitted from the procedures; and in these patients the gastric acidity was not materially increased until after the instillation of alcohol, when definite increases in acidity were noted. This makes it clear that the response obtained in the first two groups was truly attributable to the use of tobacco.

4. A significant increase in the volume of gastric juice following smoking was found in the group of patients with duodenal ulcer. In these patients the average volume rose from 49.6 to 64.5 cubic centimeters, an increase of 30 per cent.

5. The clinical observation that the smoking of tobacco has an unfavorable influence on the condition of patients with peptic ulcer finds confirmation in the experiments reported above.

450 Sutter Street.

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DISCUSSION

ARTHUR L. BLOOMFIELD, M. D. (Stanford University Medical School, San Francisco).—Doctor Rosenblum's interesting observations seem quite definite, but there would appear to be some question as to his conclusion that, because acid increases after cigarette smoking, it is therefore necessarily harmful to patients with peptic ulcer. Furthermore, we have had some different results in studying gastric secretion in peptic ulcer cases. With duodenal ulcer, for example, the basal gastric acidity without any stimulus averaged in our series 95, and even when the powerful stimulus of histamin was added to this, there was relatively little further increase.

We are somewhat skeptical as to the adequacy of Doctor Rosenblum's method of attacking his problem, especially as regards returning the aspirated gastric juice to the stomach. Another procedure would seem to be to get a curve of the basal secretion over an hour or so, and then, on the following day, to repeat the test on the same patient while smoking.

ALLAN L. COHN, M.D. (450 Sutter Street, San Francisco).—Doctor Rosenblum's conclusions, based on accurate scientific observation, can bear the scrutiny of critical analysis. His work confirms, by scientific experimental proof, the beliefs and suspicions held for many years by clinicians that tobacco smoking is detrimental to the healing process of peptic ulcer.

Several points that he brings out merit analysis and discussion.

Proof has been presented in his series of ulcer cases that there is a definite increase in volume and acidity of the gastric secretion. But he has also shown that in his Group II there is approximately the same percentage of acid increase. Group I shows an increase of 60 per cent in free acid; Group II, a 64 per cent increase. Group I shows a 35 per cent increase in total acid, and Group II a 36 per cent increase.

From these figures it might be inferred that the use of tobacco has the same effect in nonulcer cases with gastric symptoms as in peptic ulcer cases. I wish to question this point. My criticism here is not directed so much to the conclusion as it is to the content of the premise. I desire to

raise the question: Does Group II consist only of nonulcer cases? If so, the conclusion would be logical.

From my recent observations with Schindler at the Billings Hospital, Chicago, and from my own experiences with the new flexible gastroscope, I believe we shall be compelled to revise, for purposes of classification as well as treatment, our ideas of what constitutes functional gastric disease. I am quite convinced that there exists a large group of so-called functional gastro-intestinal cases which can be shown to have mucosal ulcerations, and/or a type of gastritis which can be proved only by direct visualization of the gastric mucosa. These cases should be classified at present in Group I for purposes of this experiment, and not in Group II. If Group II, as amended, would still show the same percentage increase in acid, then only might one logically conclude that cigarette smoking causes the same percentage increase in gastric acidity, in gastric functional, and in peptic ulcer cases.

The importance of the work of Lickint, and of Danielopoln Simici and Dimitrin, referred to by Doctor Rosenblum, should be emphasized. They showed that inhalation of tobacco smoke causes, in smokers, gastric hypermotility. The use of a control group of smokers with normal stomachs, in Doctor Rosenblum's experiments, might have correlated these findings as regards acidity. This same group could also have shown the comparative effect of tobacco smoking by normals as against smoking by ulcer patients. The question would then arise, if there is a difference, is it due to the gastric pathology present? If no difference could be shown between the two groups, could we say that the increased acidity is an essential nicotine effect, or an effect due to some other factor present in the smoking process? The recent observations of Mahlo, as described in *Deutsche Medicine Wochenschrift, 62*: 1216, 1936, would lead us to the belief that the increase in acidity in Doctor Rosenblum's cases is not due to the effect of nicotin on the gastric pathology present. Mahlo contends that nicotin is not absorbed by either acid or alkali gastric mucosa, and has no effect on the lining epithelium of the stomach. It is absorbed, he says, in other organs and exerts its effects in the stomach through the splanchnic nervous system.

One of the most interesting facts presented in this paper is that the administration of alcohol subsequent to the smoking period produced no further increase in concentration of acid. The knowledge of this fact by an ulcer patient who, having persisted in smoking, wishes also to return to alcoholic indulgence, could in itself lead to a very interesting discussion between patient and his medical adviser.

This paper presents many problems for further investigative work, and it is hoped that Doctor Rosenblum will continue his study.

HAROLD LINCOLN THOMPSON, M. D. (1930 Wilshire Boulevard, Los Angeles).—Doctor Rosenblum has ably presented a paper on a subject of importance to those interested in the physiology of the stomach and duodenum, and in peptic ulcer. I have been interested in problems of gastric secretion, with particular reference to the surgery of the stomach, for a number of years.

In an interpretation of the results of Doctor Rosenblum's study, one must recognize the three phases of gastric secretion, namely, cephalic (psychic), gastric, and intestinal. Doctor Rosenblum has stated that all patients in his test group were chronic cigarette smokers. Since gastric and intestinal phases of gastric secretion for all practical purposes were excluded by the conditions of this study, the observer has dealt only with the cephalic phase of secretion. The response which the author observed in his experiments was conditioned in all instances by an appetite for cigarette smoking. It would be interesting, therefore, to know the reaction in a similar group of nonsmokers as a further control on the author's observations.

I believe that in these experiments the other phases of gastric secretion are relatively unimportant, excepting in so far as the subject may have swallowed saliva or mucus, thereby slightly neutralizing the gastric content. I hope that the author will favor us with a subsequent report on the observations on similar groups of patients who are not chronic cigarette smokers.

PHYSICS: ITS APPLICATION TO ROENTGEN THERAPY*

By ROBERT E. PUGH, JR.
Pasadena

DISCUSSION *by Kenneth S. Davis, M. D., Los Angeles; John D. Lawson, M. D., Sacramento; George Stevenson Sharp, M. D., Pasadena.*

THE magnitude of roentgen therapy dosages is expressed in terms of the "r unit," which is merely a unit of quantity of radiation analogous to the "milligram" used for expressing the dosages of pharmaceuticals. It was originally defined in the interests of universal duplication by physicists rather than in terms of any physiological or biological reaction. Although its magnitude is arbitrary, its use in expressing relative quantities of radiation is not limited by this fact. Changing of the size of units expressing a certain quantity does not influence the magnitude of the quantity itself, if the number of units is changed to compensate for the change in size. For example, in the case of drugs; if the gram were the usual unit instead of the milligram, obviously the number of units required for a given quantity would be just 1/1000th the number required in terms of milligrams.

NEED OF A UNIVERSAL UNIT

It should also be pointed out that the magnitude of successful dosages, in terms of r units, were determined clinically by radiologists and clinicians, physicists having had no direct influence on the choice of such values. Roentgenologists have recognized the position of the physicist as advisor and aide in applying physical principles to these dosages, but other physicians need have no fear that the dosages themselves have been tampered with by nonmedically trained men. The same clinical or biological dosage could be expressed in any units without altering its biological effect; the important point is to choose the units so that they may be easily and universally duplicated. It appears to be desirable that the dosage be expressed in terms of a universally understood quantity, rather than in terms of some degree of biological reaction which is incapable of exact description and duplication.

DEFINITION OF THE R UNIT

The official definition of the r unit has been given in the literature many times, but for the purposes of this discussion such a formal definition may well be omitted, and attention rather directed to the fact that it is merely a unit of quantity of radiation, just as the milligram is a unit of weight of material. More universal understanding of this fact should lead to greater confidence in roentgen therapy, and less confusion in the concepts of dosage.

It so happens that the magnitude of the r unit which was chosen is such that, in the present state of the science of therapy, most dosages fall within the range of 50 to 5,000 r units. Because of the way in which it is defined, the r unit (or intensities

* Read before the Radiology Section of the California Medical Association at the sixty-sixth annual session, Del Monte, May 2–5, 1937.