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Modulation of Gastric Acid Secretion by Hypnosis

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The ability of hypnosis to both stimulate and inhibit gastric acid secretion in highly hypnotizable healthy volunteers was examined in two studies. In the first, after basal acid secretion was measured, subjects were hypnotized and instructed to imagine all aspects of eating a series of delicious meals. Acid output rose from a basal mean of 3.60 ± 0.48 to a mean of 6.80 ± 0.02 mmol H^+/h with hypnosis, an increase of 89% ($p = 0.0007$). In a second study, subjects underwent two sessions of gastric analysis in random order, once with no hypnosis and once under a hypnotic instruction to experience deep relaxation and remove their thoughts from hunger. When compared to the no-hypnosis session, with hypnosis there was a 39% reduction in basal acid output (4.29 ± 0.93 vs. 2.60 ± 0.44 mmol H^+/h, $p < 0.05$) and an 11% reduction in pentagastrin-stimulated peak acid output (28.69 ± 2.34 vs. 25.43 ± 2.98 mmol H^+/h, $p < 0.05$). We have shown that different cognitive states induced by hypnosis can promote or inhibit gastric acid production, processes clearly controlled by the central nervous system. Hypnosis offers promise as a safe and simple method for studying the mechanisms of such central control.

Hypnosis is a state of intensified and focused concentration (1). The ability to experience hypnosis, "hypnotizability," is associated with the occurrence of profoundly absorbing experiences (2,3). Thus, hypnosis provides an opportunity for especially clear control of cognitive processes such as imagery, making it of potential interest in the study of psychophysiologic phenomena. Measures of hypnotizability are available that provide a means of identifying individuals highly capable of such focused attention to cognitive tasks (1,4), allowing for the selection of subjects suitable for study.

It has long been claimed that hypnosis can affect aspects of human physiology. Despite intriguing reports suggesting that hypnosis can alter skin test reactivity (5), finger temperature (6), and cortical visual evoked potentials (7), most studies in this area have a variety of methodologic deficiencies that call their conclusions into question (8). Moreover, many studies are flawed because they seek to establish a characteristic physiologic profile of the hypnotic state. It would be more appropriate to use hypnotically induced imagery to attempt to intensify control over physiologic processes (9).

Gastric acid production is an attractive parameter to attempt to influence by hypnotically induced imagery; it is easily measured and for many years gastric secretion has been observed to change with various emotional states (10,11). Indeed, several older studies have measured the gastric secretory response to hypnotic suggestions of hunger or eating (12–14), or relaxation and sleep (15,16). However, the findings are variable and the studies involved very few subjects, questionable experimental techniques, or outdated analytical methods. Thus, using a substantial number of subjects and applying rigorous methodology we sought to test the hypothesis that appropriate hypnotic suggestions could either augment or inhibit gastric acid secretion. Not only would such a demonstration provide additional evidence that hypnosis can enhance cognitive control over an aspect of human physiology, but hypnotically induced alteration of gastric acid secretion might be a valuable model for investigating the role of the central nervous system (CNS) in the acid secretory process.

Materials and Methods

Subjects

Studies were done at Stanford and at the University of North Carolina (UNC). Eligible subjects were healthy

Abbreviations used in this paper: **PAO, peak acid output; UNC, University of North Carolina.**

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men and women 18–60 yr of age with no history of gastrointestinal disease who were taking no medications. Potential participants, having responded to posted advertisements, were screened for high levels of hypnotizability by either the Hypnotic Induction Profile (1) at Stanford, or the Harvard Group Scale (17) at UNC. Scores of ≥7 out of a possible 10 or ≥10 out of a possible 12, respectively, were required for a volunteer to be invited to participate. Volunteers were paid $50 per completed session.

Methods

For all studies, after an overnight fast subjects were intubated between 7:30 and 9 AM with a 14- or 16F vented nasogastric tube (Salem Sump, Sherwood Medical, St. Louis, Mo.). In UNC subjects, the tip of the tube was placed in the most dependent portion of the stomach under fluoroscopic guidance. To ensure optimal tube position, a modification of the water recovery test (18) was used in all subjects, both before and after completion of the gastric fluid collections. Only when ≥40 ml of the 50 ml of water introduced into the stomach was recovered on both occasions were the data included in the analysis.

Subjects sat in a comfortable armchair in a quiet room. Gastric contents were collected in 15-min aliquots using an intermittent suction pump supplemented by occasional manual syringe suction. A small amount of air was periodically injected into the main channel of the tube to ensure patency.

In the first study ("study 1: acid stimulation") subjects read or talked during the first hour. Then a hypnotic trance state was induced by either a rapid induction method (1) by D. Spiegel at Stanford or an arm levitation technique (19) by K. Klein at UNC. While under hypnosis subjects were asked to imagine eating a series of what for them would be the most delicious meals possible. The instructions involved visualizing the physical surroundings where the meal would be consumed, and attending to all sensory aspects of the eating process, including food appearance, aroma, texture, and taste. Subjects were periodically asked to describe what they were experiencing; questions and further instructions from the experimenter followed that were designed to enhance pleasurable sensations. This phase lasted an additional hour.

The second study ("study 2: acid suppression") consisted of two separate sessions, which were held in random order. In the "no-hypnosis" session a conventional gastric analysis was performed (20): after a basal collection during the first hour, 6 μg/kg of pentagastrin (Pentavlon; Ayerst, New York, N.Y.) was given subcutaneously; gastric fluid was then collected for a second hour. The procedure for the "hypnosis" session was similar. However, for this session the subject was hypnotized after intubation and before the basal collection began, and hypnosis continued through the second (postpentagastrin) hour. The hypnotic suggestion was to concentrate on an image or scene that would promote deep relaxation and divert the subject's attention from feelings of hunger. Images employed by different subjects included lying on a beach, watching a sunset, and meeting a friend in Paris. The experimenter, through periodic conversation with the subject, enhanced the desired feelings by additional instructions.

The hydrogen ion content of the gastric juice samples was determined by a standard titration technique (20). For study 1, the acid content of each 15-min aliquot was multiplied by 4 and expressed as millimoles of H^+ per hour. For study 2, basal acid output, expressed as millimoles of H^+ per hour, was calculated by summing the acid content of the first four 15-min samples, and peak acid output (PAO) was determined by summing the two adjacent 15-min second-hour samples with the greatest hydrogen ion content and multiplying by 2 (20).

Statistical Analysis

For study 1 (acid stimulation), the mean acid output for the first (no-hypnosis) hour was compared with that of the second (hypnosis-stimulated) hour using a paired *t*-test. Additionally, the first hour mean value was similarly compared with the values of each 15-min interval (expressed as millimoles of H^+ per hour) of the second hour. For study 2 (acid suppression), a paired *t*-test was used to compare basal acid output for the no-hypnosis and hypnosis sessions, and likewise for PAO. The Wilcoxon signed-rank test, which makes no assumptions about how the data are distributed, was used as a confirmatory test. Calculations were done using SAS/PC statistical software (SAS Institute, Inc., Cary, N.C.). A p-value of <0.05 (two sided) was considered significant. All values are expressed as mean ± SE except as indicated.

This study was approved by the Stanford and UNC institutional review boards. All subjects signed an informed consent statement.

Results

About 45% of the volunteers screened had high enough hypnotizability scores to be eligible; almost all those found eligible chose to participate. Fourteen women and 18 men took part in at least one of the two studies. Nine subjects were studied at Stanford, and 23 at UNC. The mean age (±SD) of all subjects was 28.6 ± 7.6 yr, and mean weight (±SD) was 134 ± 17 lb for the women and 163 ± 19 lb for the men.

The results of two of a total of 64 sessions were not included in the analyses because of failure of the post-session water recovery test. Not all subjects participated in both studies because of scheduling difficulties or because some subjects were reluctant to undergo the pentagastrin injection in study 2, or to experience the discomfort of nasogastric intubation on more than one occasion.

Study 1: Acid Stimulation

Thirteen women and 15 men participated in study 1. Subjective experiences of eating were ex-

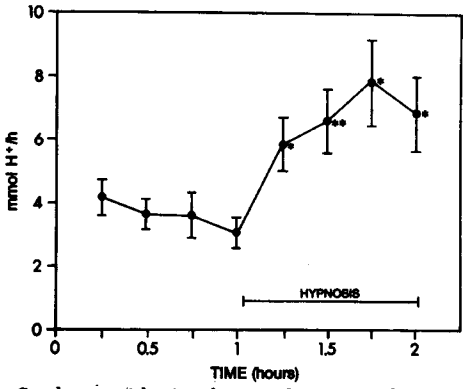

Figure 1. Study 1: acid stimulation. Gastric acid secretion in all subjects (n = 28) before and during hypnotic instruction to imagine eating pleasurable meals. *Vertical bars* represent ±standard error. Overall, the hypnosis mean hour value was significantly greater than the first hour (nonhypnosis) mean value ($p = 0.0007$). *$p < 0.005$, **$p < 0.001$ vs. first hour mean value.

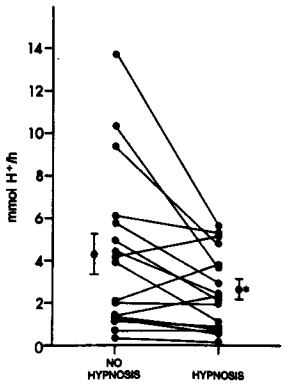

Figure 2. Study 2: acid suppression. Basal acid output in subjects without hypnosis and during hypnotic instruction to visualize an intensely relaxing setting. *Offset points with vertical bars* represent mean ± standard error. *p = 0.023 (paired *t*-test), p = 0.022 (signed-rank test) relative to no-hypnosis session.

tremely intense and vividly communicated in every case. The experimenters, who both have considerable experience in the use of hypnosis, were confident that all subjects rapidly entered a significant state of hypnosis. Figure 1 shows the gastric acid output for all 28 subjects for each 15-min period during the 2-h study. The mean acid output for each period of the first hour ranged from 3.04 to 4.20 mmol H^+/h, with a combined mean of 3.60 ± 0.48 mmol H^+/h. During the second (hypnosis) hour the mean acid output for each period ranged from 5.84 to 7.88 mmol H^+/h, with a combined mean of 6.80 ± 1.02 mmol H^+/h. Compared to the first hour, mean acid output during the hypnosis hour increased by 89% (p = 0.0007). Moreover, statistically significant increases occurred at each 15-min hypnosis interval (Figure 1). A similar pattern was seen when the women and men, the Stanford and UNC subjects, or only subjects participating in both studies were analyzed separately. For the 13 subjects who participated in both studies 1 and 2, the ratio of PAO during the hypnosis hour of study 1 to the pentagastrin-induced PAO in the study 2 session without hypnosis was 0.29.

Study 2: Acid Suppression

Seven women and 10 men participated in study 2. As with study 1, subjective experiences were quite intense in every case. Gastric acid values for comparable sessions were very similar regardless of the order in which the sessions were held. The basal acid output for each subject for the no-hypnosis and hypnosis session is shown in Figure 2. The mean basal acid output for the hypnosis session (2.60 ± 0.44 mmol H^+/h) was significantly less than that for the no-hypnosis session (4.29 ± 0.93 mmol H^+/h), [a 39% reduction, p = 0.023 (paired *t*-test), p = 0.022 (signed-rank test)]. Figure 3 shows the pentagastrin-stimulated PAO for each subject for the two sessions. The mean PAO for the hypnosis session (25.43 ± 2.98 mmol H^+/h) was significantly less than that for the no-hypnosis session (28.69 ± 2.34 mmol H^+/h) [an 11% reduction, p = 0.035 (paired *t*-test), p = 0.025 (signed-rank test)].

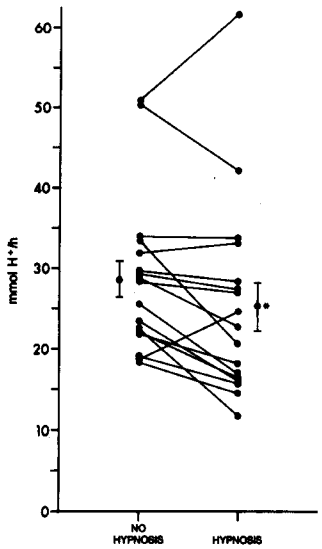

Figure 3. Study 2: acid suppression. Pentagastrin-stimulated PAO in subjects without hypnosis and during hypnotic instruction to visualize an intensely relaxing setting. *Offset points with vertical bars* represent mean ± standard error. *p = 0.035 (paired *t*-test), p = 0.025 (signed-rank test) relative to no-hypnosis session.

Discussion

We have shown that hypnotic suggestion can clearly stimulate the secretion of gastric acid. As our subjects were all in good health and highly hypnotizable, generalization to other groups would be speculative. These findings confirm older anecdotal observations of the effects of imagined eating (10,21, 22), and the results of a "food discussion" technique (23). That the findings are robust is suggested by the results being similar in a variety of subgroups, including subjects undergoing hypnosis by each of two different techniques. The goal of our study was to show that gastric acid secretion could be significantly influenced by a simple and reliable method of inducing intense mental imagery. It was not to show that hypnosis per se had a unique ability to modulate acid secretion; to do that would have required a control group of subjects of low hypnotizability.

Hypnosis-induced mental imagery appears to be capable of promoting gastric acid production by a "cephalic phase" stimulus initiated entirely within the CNS. This CNS event, by unknown mechanisms, initiates vagal efferent activity that leads to the stimulation of muscarinic cholinergic receptors on the parietal cell, and also may activate gastrin release (24). Existing cephalic-phase models of acid secretion have both practical and theoretical difficulties. Chemical vagal stimulation, for example by insulin hypoglycemia or 2-deoxy-*d*-glucose, is dangerous and may not be entirely centrally mediated. "Banquet settings" (25) are cumbersome, poorly validated, and may involve stimuli such as the odor of food, which initiate signals at lower levels within the CNS. The most widely used cephalic-phase model is "modified sham feeding" (26). Problems with this method include the need for preparation of a standard meal, which may not be optimally appetizing to all subjects, and thus may result in submaximal acid stimulation (27), and the risk that the subject will swallow bits of food, which would give invalid results. Peak acid output with modified sham feeding is ~45%–60% of maximally stimulated PAO (26,28,29), whereas with hypnosis in our study it was 29%. Rather than hypnosis being a less satisfactory cephalic phase model than modified sham feeding, a possible explanation for this difference is that modified sham feeding stimulates acid secretion by the activation of pathways outside the CNS or noncognitive pathways within the CNS, or both.

To our knowledge, this study is the first to adequately demonstrate significant inhibition of both basal and maximally stimulated gastric acid secretion by nonpharmacologic means, reductions of 39% and 11%, respectively. The observation that acid production may be truly suppressed by this technique is strengthened by its having been demonstrated both in the basal and the maximally stimulated states, and by statistical significance having been shown by both parametric and nonparametric analyses. These results suggest that in the human there are CNS mechanisms for the inhibition as well as promotion of gastric secretion. Central inhibition has been shown in animals by the intraventricular injection of various neurotransmitters and peptides (24), but the mechanism for the effect we observed is unknown. Imagery induced by hypnosis was able to significantly reduce not only basal acid output, but pentagastrin-stimulated PAO, a maximal inducer of acid secretion. Pentagastrin-stimulated PAO is quite constant over time in a given subject (30), making even the modest reduction with hypnosis notable. It is possible that even more marked suppression would have occurred if acid production had been submaximally rather than maximally stimulated.

In summary, we have shown that by using different hypnotic suggestions to induce intense mental imagery it is possible to either promote or inhibit gastric acid secretion. These effects must have been initiated solely by cognitive processes within the CNS. Of particular interest is that the content of the imagery determined the direction of change in acid production. Because of its simplicity and safety, hypnosis may serve as a useful model for further studying the mechanisms of CNS control of gastric secretion.

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