Plasma Insulin Levels During Imaginary Food Ingestion Under Hypnosis[1] (34454)

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The role of the vagus nerve in the release of insulin is not well understood. Studies in the cat revealed that there is significant parasympathetic innervation of the islets (1). Over 40 years ago, electrical stimulation of the vagus was shown to lower blood sugar levels (2). More recently insulin release was evoked in anesthetized dogs by stimulation of the vagus (3, 4). Also, recent *in vitro* studies demonstrated release of insulin via cholinergic agents (5). These findings led us to investigate the possible physiologic role for this vagal mechanism.

Although Kun and Horvath (6) postulated a reflex arc involving the taste end plates in the mouth and the vagus nerve, recent studies demonstrated that the ingestion of sweet noncaloric beverages do not provoke a rise in insulin levels (7). The studies of Sakata *et al.* (8) and Shimazu *et al.* (9) suggest that centers in the brain are involved in glucose regulation. We therefore, have used hypnosis to establish a psychological stimulus for simulating a "physiological" release of plasma insulin.

Methods. Seven healthy subjects, four men and three women, between the ages of 20 and 30 years, were studied. Before the experiments, these subjects were hypnotized on several occasions to facilitate their response to the stimulus. A list of foods which the subjects preferred was also obtained. On the day of the study, the subjects were fasted for 4 hr and an indwelling needle was placed into an arm vein. After base line samples were obtained, the subjects were hypnotized by one of us (D.G.) using a standard technique of relation and eye closure. Following the induction of hypnosis but before giving suggestions specific to the experiment, a resting blood sample was drawn after 4 min. Seven additional blood samples were then taken at 4-min intervals in each stage of the experiment.

Six min after hypnosis was induced, "hunger" was introduced followed 4 min later by the suggestion that the subjects were eating their favorite foods at a restaurant. After the "meal," the subjects remained hypnotized an additional 4 min before being awakened. Plasma insulin was measured by a charcoal method (10), NEFA by the method of Dole (11), and glucose by the glucose oxidase method (12).

Results. All subjects achieved an adequate depth of hypnosis in 10–15 min. All reported they had experienced the tasting and swallowing of food.

Induction of hypnosis alone did not affect the levels of insulin. While four subjects failed to show any significant change, three subjects responded with elevations in plasma insulin (Fig. 1). In one subject this occurred

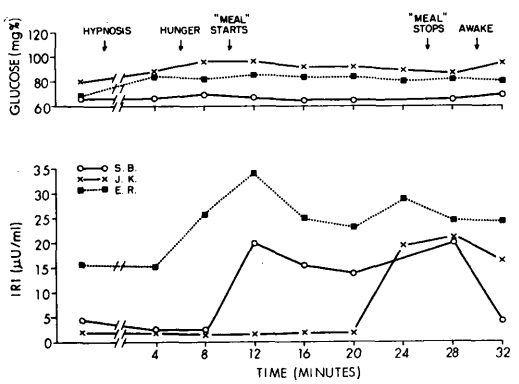

FIG. 1. Blood glucose and plasma insulin levels during hypnotically induced imaginary meal.

[1] Supported by Grant AM-00396 from the National Institutes of Health.

TABLE I. Changes in Nonesterified Fatty Acids (μeq/liter) during Imaginary Food Ingestion under Hypnosis.

Time (min)	Condition	Subjects S.B.	J.K.	M.N.	M.G.
	Control	1271	1175	1024	1143
0	Hypnosis starts				
+4		1363	871	1228	1269
+6	"Hunger" is induced				
+8		1452	1090	1101	1213
+10	"Meal" starts				
+12		1325	1051	1013	1215
+16		1372	1009	1034	1187
+20		1198	934	1052	1098
+24		1146	890	1062	1101
+26	"Meal" stops				
+28		1068	815	801	895
+29	Awakens				
+32		1054	799	987	821

after the "hunger" stimulus. In another, this occurred just after the "meal" stimulus and in a third, a rise was noted at the end of the "meal."

The NEFA were observed to fall in four of the seven subjects (Table I); the two subjects with the greatest fall (S.B. and J.K.) showed also a plasma insulin rise and the NEFA fall was noted to begin during the induction of the "meal." The other two NEFA "responders" (M. N. and M. G.) showed a fall immediately after the "meal" stopped and their insulin levels were constant throughout the experiment. There appeared to be no correlation between the depth of trance and the response to the stimulus of food suggestion.

Discussion. The data in this study suggest that the "thought" of a meal can evoke a rise in plasma insulin in some subjects. It may well be that this occurs via cortical centers and the vagus nerve. If so, this may be analogous to the vagal phase of gastric acid secretion. However, it can not be ruled out that these rises in insulin were due to changes in sympathetic discharge or to unknown factors.

The reasons for the variation in response remain speculative. The depth of trance is a multidimensional variable and was not uniform across subjects or within each experiment. Although the reality of the imaginary situation varied as a function of trance depth, the physiologic response most likely was also a function of other sensorimotor and cognitive-affective alterations.

The fall in NEFA was not unexpected. NEFA levels have been demonstrated to change with levels of hormones and with fear and anxiety (13–15). Moreover, Penick *et al.* (16) demonstrated a fall in NEFA with the mere sight of food. It is possible that the fall in NEFA after the "meal" stimulus was associated with a rise in insulin secretion. We believe this observation suggests an approach to the evaluation in humans of higher neurological regulation of humoral adaptations.

Summary. Seven healthy subjects were exposed to imaginary food ingestion under hypnotic stimulus and plasma glucose, immunoreactive insulin and NEFA were measured during the experiment. Three subjects showed elevation in plasma insulin either after the induction of "hunger" or after the beginning of the "meal." Fall in NEFA was seen in four subjects. No changes in blood glucose were detected.

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Received Sept. 11, 1969. P.S.E.B.M., 1970, Vol. 133.