Insulin-Induced Hypoglycemia Stimulates Gastric Vagal Activity and Motor Function without Increasing Cardiac Vagal Activity
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Digestion 2005;72:43-48 (DOI: 10.1159/000087600)

Key Words
- Drinking capacity
- Gastric emptying
- Glucose clamp
- Intragastric volumes
- Vagal tone

Abstract

Background/Aims: We investigated whether increasing the efferent vagal activity by insulin-induced hypoglycemia would enhance gastric emptying and volumes in healthy subjects. Methods: Twenty healthy volunteers (10 males) were examined with and without vagal stimulation by insulin-induced hypoglycemia using a glucose clamp technique. Stomach function was tested by drinking meat soup (0.04 kcal ml\(^{-1}\)) at a rate of 100 ml min\(^{-1}\) until maximal capacity. Intragastric volume at maximal drinking capacity was determined by three-dimensional ultrasound. Respiratory sinus arrhythmia (RSA) was used as an index of cardiac vagal activity and plasma pancreatic polypeptide (PP) as a measure of gastric vagal activity, and skin conductance (SC) as a measure of sympathetic tone. Results: Insulin-induced hypoglycaemia increased drinking capacity (\(p = 0.002\)), gastric emptying (\(p = 0.02\)), PP (\(p = 0.004\)) and SC (\(p = 0.004\)), while intragastric volume was unchanged (\(p = 0.7\)) and RSA decreased (\(p = 0.03\)). Conclusion: Enhancement of gastric vagal activity by insulin-induced hypoglycemia increased drinking capacity and gastric emptying similarly, resulting in an unchanged intragastric volume. Enhanced efferent vagal activity to the stomach (as measured by PP) was not associated by enhanced cardiac vagal activity (as measured by RSA), possibly a consequence of stress-induced sympathetic activation during the procedure.