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Effect of Metoclopramide on Gastric Fluid Volumes in Diabetic Patients Who Have Fasted before Elective Surgery

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Background: **Diabetes-induced gastroparesis is believed to increase fasting gastric fluid volume before elective surgery. Metoclopramide is routinely administered preoperatively to reduce gastric fluid volume in these patients. This study compared nondiabetic controls to non–insulin-dependent and insulin-dependent diabetics to determine the effect of metoclopramide, administered before surgery, on gastric volumes in patients who fasted before surgery.**

Methods: **Control and diabetic patients fasted preoperatively before receiving either placebo or 10 mg intravenous metoclopramide 20 min before induction of anesthesia. After intubation, a gastric tube was placed, and stomach contents were aspirated with volumes compared among the groups.**

Results: **Both groups of diabetic patients were older than the control group, and insulin-dependent patients had a higher incidence of comorbidities compared with the non–insulin-dependent group. Fasting blood sugar and hemoglobin A_1C values were higher in both insulin-dependent and non–insulin-dependent patients. Gastric fluid volumes were similar in control, non–insulin-dependent, and insulin-dependent patients (8.0 ± 2.6 *vs.* 9.6 ± 4.1 *vs.* 17.7 ± 2.5 ml, respectively). In insulin-dependent diabetic patients, metoclopramide decreased gastric volume compared with placebo treatment (17.7 ± 2.5 *vs.* 7.8 ± 2.9 ml; $P = 0.027$). After stratification, a subpopulation of patients with poorly controlled diabetes, regardless of type, were identified to have increased gastric residual volumes.**

Conclusion: **In elective surgical patients who have fasted before surgery, gastric volumes are minimal, even in diabetics with severe neuropathic symptoms. Metoclopramide prophylaxis to reduce gastric volumes seems to be unnecessary unless the patient has a prolonged history of poor blood glucose control.**

NEUROPATHIC gastroparesis is among the many complications associated with both insulin-dependent (IDDM) and non–insulin-dependent diabetes mellitus (NIDDM). Reported incidence of gastroparesis ranges from 9.9% to 76%.[1–3] The relation between the duration of diabetes mellitus, glycemic control, and the prevalence of gastroparesis is poorly defined.[4] There is little correlation between gastrointestinal symptoms and objective data for gastric emptying in patients with IDDM or NIDDM.[2,5–7] Although no determinable duration of chronicity of diabetes can clearly predict the presence of gastroparesis in these patients, physicians have presumed a relation between the onset of gastroparesis and the development of other diabetic complications.[4]

The syndrome seems similar to gastric dysmotility secondary to vagal neuropathy. Diabetics with gastroparesis seem to have the same clinical symptoms and similar radiographic findings seen in postvagotomy patients.[8,9] The observation that atropine abolishes all antral activity supports the neural mechanism for gastroparesis.[9] However, delayed gastric emptying of liquids in diabetics with gastroparesis is unlike the increased rate of liquid emptying resulting in the "dumping syndrome" seen in postvagotomy patients.[10] It remains unclear whether vagal neuropathy is the etiology of gastroparesis in diabetic patients. Other theories for diabetic gastroparesis have been proposed, including a hyperglycemia-induced delay in gastric emptying of meals containing fat and protein, and diabetic-induced alterations in secretion of various hormones such as motilin, pancreatic polypeptide, somatostatin, glucagon, and gastrin.[10,11]

Although the etiology of gastroparesis in diabetics remains unclear and symptoms may not be apparent, many physicians treat these patients empirically to prevent vomiting during anesthetic induction.[11,12] Metoclopramide, a medication used for diabetic gastroparesis, is a cholinergic agent that acts by increasing the release of acetylcholine at the neuromuscular junction within the gastric wall.[13] Further, metoclopramide has antidopaminergic properties that inhibit dopamine-induced gastric smooth muscle relaxation, and it also penetrates the blood–brain barrier and binds to medullary chemoreceptors.[14] This latter property may be important in the use of metoclopramide as an antiemetic. Metoclopramide, when given intravenously, has an onset of action between 1 and 3 min. Intramuscular and oral administrations have onset times of 10–15 min and up to 60 min, respectively.[15] The effect may persist for as long as 2 h after a single dose. The efficacy of metoclopramide in facilitating gastric emptying in diabetics has been well documented.[16,17] However, no study has examined the use of metoclopramide in diabetics who fasted for more than 6 h. Patients undergoing elective surgery are routinely asked to fast for several hours before surgery. Diabetic patients are often given metoclopramide prophylactically to reduce their risk of aspiration, because there is a theoretical possibility that they may have increased gastric residual volumes secondary to gastroparesis.[12]

This study was designed to answer two questions. Do diabetics have significantly higher gastric residual volumes after preoperative fasting compared with nondia-

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betics? If so, is the administration of metoclopramide effective in reducing gastric volumes to nondiabetic control levels?

Materials and Methods

After obtaining Institutional Review Board approval from Loyola University Medical Center, Maywood, Illinois, and informed written consent, 125 in-house or same-day-admission patients were enrolled in the study. Twenty-five patients were nondiabetic patients, and the other 100 patients were diagnosed with diabetes. Fifty of these diabetic patients were receiving long-term insulin therapy (IDDM), and 50 had NIDDM. Subjects who required elective surgery during general anesthesia and who had fasted for at least 8 h before surgery were selected for the study. Patients were excluded from the study if they were younger than 18 yr or older than 65 yr. Pregnant patients and those with steroid-induced hyperglycemia were excluded along with patients with a history of gastrointestinal surgery, hiatal hernia, or esophageal varices secondary to alcoholism. Extremely obese patients with a body mass index of 30% or greater over normal were not included in the study. Patients receiving medications known to improve gastric motility were also excluded.

On the morning of surgery, patients were allowed their usual medication with a small sip of water. The subjects were brought to the preoperative holding area, and upon placement of an intravenous catheter, 10 ml blood was obtained for determination of serum glucose and hemoglobin A_1C. The subjects in each of the three groups were then randomly assigned by lottery in a double-blind fashion to receive either saline or metoclopramide, 10 mg intravenous, in equal volume. Only the research nurse, who did not participate in clinical care or gastric aspiration, knew whether placebo or metoclopramide was administered. The time was noted for the administration of the drug, and approximately 20 min after administration, the patient was brought to the operating room. After placement of standard monitors, anesthesia was induced with 3–5 mg/kg thiopental and 2–3 μg/kg intravenous fentanyl. Tracheal intubation was facilitated using 1.5–2 mg/kg intravenous succinylcholine with cricoid pressure.

After the patient was intubated, a 14-French gastric sump tube was placed either orally or nasally. The correct position was confirmed by auscultation over the epigastrium while air was inserted through the sump using a 60-ml Toomey syringe. Gastric contents were aspirated through the sump tube while the epigastric region of the patient was manually massaged. To maximize the volume of aspirate obtained and to increase the likelihood that all gastric volume was collected, aspiration was performed with the patient in the supine, head-down, and right and left lateral decubitus positions. The time between administration of the study medication and aspiration of gastric contents was approximately 30 to not longer than 60 min.

Age, sex, height, weight, and a relevant history such as years of diabetes, blood sugar control with fasting blood sugar concentrations, and glycosylated hemoglobin concentrations were obtained on each patient. Also, all patients in each group were queried for symptoms suggestive of gastroparesis, including nausea, vomiting, epigastric fullness, bloating, and evidence of diabetic pathology. The time between administration of study drug and aspiration of gastric contents was recorded as well as volume of gastric content.

Statistical Analysis

Assuming that more than 50% of diabetic patients have gastric emptying abnormalities[1] and considering that metoclopramide treatment has been shown to improve gastric emptying by 20–25%, at least half of the untreated NIDDM and IDDM patients should have increased gastric volumes *versus* control. Further, metoclopramide should reduce increased gastric volumes to control levels in the treated patients. Based on this expectation, we computed a requisite sample size for our study using a smaller group of patients that had been collected at an earlier point in time. Gastric volumes were log transformed due to the extreme skewing that was observed in the data. The mean log gastric volumes for the IDDM and nondiabetic patients were 1.3 and 0.9, respectively (which correspond to 19.45 and 7.44 ml, respectively). The average of the SDs was 0.48. This yielded an effect size of 0.83. From this, we determined that a sample size of 25 per group would provide 82% power to detect an effect size of 0.83 with a two-tailed test using a 0.05 α level.

Before data analysis, all continuous variables (*e.g.*, gastric volume, age, height) were checked for normality. Transformed values for gastric volume were used for analysis, but the results are presented in the original (*i.e.*, reverse transformed) unit of milliliters.

Differences in continuous variables among patients in the control, NIDDM, and IDDM groups were assessed with one-way analysis of variance. Statistically significant analyses of variance were then followed with the Scheffé *post hoc* test to examine pairwise comparisons. Differences in frequency data were assessed with the chi-square test for association.

To examine the role of glucose control, patients were stratified by hemoglobin A_1C and glucose concentrations, and differences in gastric volumes among these strata were tested with the analytic methods mentioned above. All continuous variables are expressed as mean ± SD with significance determined at the $P < 0.05$ level.

Table 1. Intergroup Demographic Comparisons

	Control	NIDDM	IDDM
Patients, n	25	50	50
Age, yr	52.8 ± 16.7	63.6 ± 11.3*	59.8 ± 15.2
Height, cm	169.6 ± 9.3	170.0 ± 9.0	168.0 ± 11.4
Weight, kg	76.6 ± 12.7	83.5 ± 15.0	84.7 ± 20.6
Duration of diagnosis, yr	0	7.5 ± 6.6	17.8 ± 11.6†
M/F, %	56/44	46/54	59/41
FBS, mg/dl	94.7 ± 22.1	144.1 ± 51.0*	172.7 ± 62.5*†
HbA_1C, %	6.4 ± 1.0	9.2 ± 2.0*	11.2 ± 3.0*†
Diagnosis vascular, %	0	47	63
Diagnosis retinopathy, %	0	28	29
Diagnosis neuropathy, %	0	23	44†
Diagnosis skin ulcers, %	0	4	17†
Gastrointestinal history, %	0	2	6

Values are presented as mean ± SD.

* $P < 0.05$ compared with control group. † $P < 0.05$ compared with NIDDM group.

FBS = fasting blood sugar; Hb = hemoglobin; IDDM = insulin-dependent diabetic; NIDDM = non–insulin-dependent diabetic.

Results

The mean age of the NIDDM patients was significantly greater than the mean age of the controls, whereas mean ages of the IDDM patients did not differ from those of the NIDDM patients and controls. Groups were similar in height and weight. The IDDM patients had a higher incidence of neuropathy and ischemic skin problems compared with their NIDDM counterparts. In addition, fasting blood sugar was higher in the IDDM patients compared with the nondiabetic and NIDDM patients. Hemoglobin A_1C was also higher in the IDDM cohort compared with the NIDDM cohort (table 1).

Perioperative patient data were again analyzed and divided by study drug assignment. Fasting times for both liquid and solid were not appreciably different among these groups, nor were the times to aspiration of stomach contents after study (table 2). Gastric volumes collected *via* sump tube aspiration in all patients who were treated with placebo revealed little difference between nondiabetics or diabetics who were receiving insulin with severe complications (table 2). Gastric volumes after metoclopramide therapy were also similar in all three groups. Metoclopramide treatment had minimal impact on gastric volume in normal and NIDDM patients. A statistically significant reduction was noted in gastric volumes after metoclopramide therapy in IDDM patients (table 2). Although a 56% reduction in mean gastric volume was observed with metoclopramide treatment in IDDM patients, gastric volumes in nontreated IDDM patients were not importantly increased by clinical criteria after a fast of longer than 8 h.

Stratification of results by hemoglobin A_1C concentrations showed the highest fasting gastric volumes in patients with the worst long-term glucose control (hemoglobin $A_1C > 9\%$; table 3). Metoclopramide treatment significantly reduced this volume. No difference in gastric volumes were noted when patients were stratified by fasting blood glucose concentrations (table 3).

Discussion

Patients with diabetes are known to have disorders of gastrointestinal motility. A correlation has been found between slow gastric emptying and the presence of

Table 2. Intraoperative Data, Fasting Time, and Gastric Aspiration Volume

	Control		NIDDM		IDDM	
	Placebo (n = 10)	Metoclopramide (n = 15)	Placebo (n = 25)	Metoclopramide (n = 25)	Placebo (n = 25)	Metoclopramide (n = 25)
Aspiration time after treatment, min	33.6 ± 17.9	27.5 ± 13.8	41.4 ± 18.8	38.9 ± 18.0	36.9 ± 17.8	42.2 ± 25.0
NPO liquid, h	12.2 ± 2.7	10.6 ± 3.2	14.0 ± 5.1	12.6 ± 4.1	11.3 ± 3.4	11.0 ± 3.5
NPO solid, h	14.1 ± 3.9	15.2 ± 3.8	19.4 ± 12.9	14.5 ± 5.8	15.0 ± 5.6	14.4 ± 6.0
Gastric volume, ml	8.01 ± 2.6	6.5 ± 3.0	9.6 ± 4.1	4.6 ± 3.5	17.7 ± 2.5	7.8 ± 2.9*
Duration of diagnosis, yr			7.4 ± 1.1	7.5 ± 1.6	20.6 ± 2.4	15.3 ± 2.3
Fast blood glucose, mg/dl	85.2 ± 14.0	106.2 ± 26.0	154.5 ± 49.7	134.7 ± 51.6	168.5 ± 65.1	176.2 ± 61.2
Hemoglobin A_1C, %	6.7 ± 0.6	6.1 ± 1.2	9.4 ± 2.1	9.0 ± 2.0	10.8 ± 2.7	11.6 ± 3.3

Values are presented as mean ± SD.

* Compared with placebo insulin-dependent diabetic (IDDM) group, $P = 0.027$.

NIDDM = non–insulin-dependent diabetic; NPO = nothing *per os*.

Table 3. Gastric Residual Volume Stratified by Hemoglobin A_1C and Blood Glucose Concentrations

Hemoglobin A_1C Concentrations (%)	<7 (n = 25)*	7–9 (n = 41)	>9 (n = 59)
Gastric volumes			
All patients	4.8 ± 2.8 (0–50)	11.2 ± 2.7 (0–100)	8.6 ± 3.9 (0–100)
Placebo treatment	4.2 ± 1.2 (2–10)	10.8 ± 3.2 (0–50)	16.2 ± 3.6† (0–100)
Metoclopramide treatment	5.2 ± 3.7 (0–50)	11.6 ± 2.3 (3–100)	4.7 ± 3.4 (0–75)
Blood glucose Concentrations (mg/dl)	**<120 (n = 36)**	**120–160 (n = 44)**	**>160 (n = 45)**
Gastric volumes			
All patients	7.3 ± 3.1 (0–60)	7.9 ± 2.8 (0–100)	9.8 ± 3.9 (0–93)
Placebo treatment	10.3 ± 3.2 (0–60)	9.9 ± 3.5 (0–55)	14.5 ± 3.4 (0–93)
Metoclopramide treatment	5.3 ± 2.9 (0–30)	6.6 ± 2.2 (1–100)	6.6 ± 4.2 (0–75)

Values are presented as mean ± SD. Numbers in parentheses are ranges.

* All 25 patients with hemoglobin $A_1C < 7$ were nondiabetic control. † $P < 0.05$ vs. nondiabetic control.

autonomic neuropathy as assessed by standardized cardiovascular reflex tests.[2,5,7] This study was designed to determine whether diabetic patients, with varying disease severity, have higher gastric fluid volumes than nondiabetics after preoperative fasting. The work also was designed to determine whether metoclopramide is effective in reducing gastric fluid volume among diabetic patients to nondiabetic control levels. In the current study, NIDDM patients had fewer neuropathic symptoms compared with IDDM patients (table 1). If the correlation between diabetic symptoms and gastroparesis holds, IDDM patients should have a higher incidence of gastroparesis. In our study, we did not find a statistically significant correlation between diabetic type and the amount of residual volume after fasting.

The incidence of gastrointestinal symptoms may be as high as 76% in all diabetic patients.[2] There are patients in whom diabetic gastroparesis is the only diabetic complication. The severity of diabetes does not seem to be a predictor of the severity of gastroparesis. Patients with type 2 diabetes may have an incidence of delayed gastric emptying as high as 56%.[3] Although plasma glucose has been shown to be related to delayed gastric emptying, no significant relation between glycosylated hemoglobin (hemoglobin A_1C) and delayed gastric emptying has been observed.[4] This same finding has been noted with IDDM.[5] The current study did find a relation between poor blood sugar control, as evidenced by increased hemoglobin A_1C, and gastric residual volumes (table 3). Although not every patient in this subgroup had high fasting gastric volumes, a few had volumes large enough to present a genuine concern, should stomach contents reflux into the trachea after induction of anesthesia.

Another study evaluated diabetic patients presenting for outpatient procedures and found their mean gastric volumes to be 69 ± 17 ml.[18] The investigators compared these gastric volumes to those inpatients who fasted, whose mean volumes were 33 ± 4 ml, a value similar to that observed in the current study and in patients without diabetes. Our study did not find gastric volumes to be as high after preoperative fasting, although we did observe our diabetic subgroup with the worst glucose control to have gastric volumes similar to those reported by Ong *et al.*[18] They also opined that their measurement of gastric volumes may have been underestimated because they were not able to completely empty the stomach. This possibility also exists in our study. However, we were meticulous during our evacuation of stomach contents, rolling the patient from side to side, compressing the abdomen, and moving the Salem sump tube to help drain stomach contents. This technique is a valid and well-accepted method for assessment of gastric volume.[19] Another study compared blind gastric aspiration with direct aspiration using an endoscope and found the blind aspiration technique underestimated gastric volume by approximately 14 ml.[20] In that study, however, the patient was left supine and not moved from side to side to enhance recovery of gastric contents. Therefore, we believe we were successful in collecting comparable and probably a complete volume of stomach contents after intubation. Although this methodology may produce a systematic bias for undercollection, there is no reason to believe that collection bias would differ among groups. Further, the collector was blinded to therapy. Because this study only examined the risk of aspiration and not pneumonitis, gastric pH was not measured.

Newer evidence points to delayed gastric emptying being associated with fasting blood glucose concentrations.[6] In fact, rates of gastric emptying have been found to vary inversely with glucose concentration, an observation not confirmed by our data.[7] IDDM patients had the highest fasting blood glucose concentrations but

mean gastric residual volumes similar to NIDDM or control nondiabetic patients. After stratification to identify the patients with the poorest blood glucose control, however, a clinically significant relation was realized. One patient from the poor control group (hemoglobin $A_1C > 9\%$, blood glucose > 160 mg/dl) had gastric residual volumes of greater than 80 ml. This patient had the worst glucose control and the highest gastric volumes observed, although still relatively small in comparison with other studies.

Because no true predictors exist to determine whether a diabetic patient will have increased gastric residual volumes after fasting, most anesthesia practitioners use prophylactic medications to reduce residual gastric volumes and the postulated risk of aspiration during induction of anesthesia. Several studies have demonstrated that metoclopramide therapy significantly improves gastric emptying, and its prokinetic and antiemetic properties are a valuable adjunct in the treatment of diabetes-related gastric stasis.[11,13]

Metoclopramide therapy may be beneficial for the treatment of diabetic gastroparesis, and this therapy did have a positive effect on reducing gastric volumes in our own diabetic population. However, is this therapy really indicated? The use of metoclopramide is not without adverse effects, which include sedation, dysphoria, agitation, dry mouth, periorbital edema, and extrapyramidal adverse effects, including leg restlessness and sedation. Although many of these symptoms are mild, they still may be severe enough to cause discomfort or exaggerated responses to phenothiazines, butyrophenones, or monoamine oxidase inhibitors. There are no data available to document what level of perioperative gastric volume, if any, actually increases the risk of aspiration.

A patient at risk for aspiration has been defined as having gastric volumes of 0.4 ml/kg. This volume criteria was obtained from experiments in which unilateral pulmonary instillation of gastric acid was performed on monkeys.[21] These criteria have been widely quoted and used to evaluate nearly all treatment regimens for prevention of pulmonary aspiration. Recent animal studies, however, have suggested that the critical volume may be 0.8 ml/kg.[22] Because aspiration remains an important cause of anesthesia morbidity and mortality, any reasonable approach to reducing potentially increased volumes is warranted. Our data demonstrated that even in diabetics with numerous comorbidities, residual volumes after preoperative fasting were small when determined immediately after anesthetic induction and intubation. Metoclopramide reduced these volumes, but these changes may be inconsequential because the volumes observed in nontreatment patients with blood glucose concentrations less than 160 mg/dl and hemoglobin A_1C less than 9% were less than the volumes traditionally considered necessary (25 ml) to produce pulmonary damage.[21] However, the range of volumes noted in these patients was small, demonstrating that diabetic patients, regardless of their comorbidities and levels of disease control, have no real need for prokinetic therapy to reduce residual volumes if they fast for more than 8 h.

A 1999 review of "Practice Guidelines for Preoperative Fasting and the Use of Pharmacologic Agents to Reduce the Risk of Pulmonary Aspiration" did not support the routine use of prokinetic agents as a method to reduce the risk of pulmonary aspiration.[12] However, these guidelines did recommend the use of prokinetic therapy with gastrointestinal reflux or metabolic disorders (*e.g.*, diabetes mellitus) that may increase the risk of aspiration. Our results do not totally support this recommendation. We agree with the authors that patients should be questioned for evidence of gastrointestinal reflux and, if present, treated preoperatively with antacids and prokinetic agents. However, our data do not support the routine use of prokinetic agents to reduce gastric volumes in diabetes. Even in diabetics with documented neuropathic conditions, residual gastric volumes after a preoperative fast were minimal and not strikingly different from normal nondiabetic patients. The current study, however, does support the use of prokinetic therapy in a subgroup of diabetic patients with extremely poor blood glucose control, because some of these patients were found to have gastric volumes after preoperative fasting that could be large enough to produce pulmonary complications should reflux and aspiration into the trachea occur after induction of anesthesia.

In conclusion, this study demonstrates that in diabetic patients undergoing elective surgery, after preoperative fasting, gastric volumes are usually small and clinically unimportant. Therefore, the routine use of metoclopramide for aspiration prophylaxis in NIDDM or IDDM patients who fast before surgery and do not have a history of reflux or dysphagia may be unwarranted and unnecessary. Metoclopramide may be indicated in patients with poorly controlled diabetes, high fasting blood glucose concentrations, and abnormal hemoglobin A_1C values of greater than 9%. Unless these conditions are present, its use in diabetic patients presenting for routine surgery who have fasted is unnecessary and should be abandoned as elective presurgical preparation in this patient population.

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