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Gastric Emptying in Male Neurologic Trauma

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Prolonged gastric emptying half-time (GET1/2) has been observed in several neurological disorders. Most patients with moderate to severe neurologic trauma (NT) initially do not tolerate enteral or nasogastric feedings. However, previous findings of altered gastric emptying (GE) in patients with NT have been questionable. Quantitative measurements of GE, to determine a possible mechanism for intolerance to enteral feeding, are lacking. In this study, we measured GET1/2 sec of solid and liquid meals by radionuclide imaging in men who were neurologic trauma patients. **Methods:** A prospective study was conducted to assess GET1/2 in 30 men who were patients with spinal cord injuries (SCIs) and 20 men who were patients with head injuries (HIs) using radionuclide-labeled solid and liquid meals, respectively. Meanwhile, 18 and 14 male control subjects underwent the same imaging techniques for solid and liquid meals, respectively, to evaluate the normal ranges of solid and liquid GET1/2 sec (84.5 ± 16.7 and 29.2 ± 3.7 min). **Results:** In the 30 SCI patients, GET1/2 of solid meals was significantly prolonged (138.3 ± 49.2 min, $p < 0.05$), and 53% (16/30) of patients had abnormal GET1/2. A more prolonged GET1/2 and a higher incidence of abnormal GET1/2 were observed in patients with high-level injury, when compared with patients with low-level injury ($p < 0.05$). In the 20 HI patients, GET1/2 of liquid meals was prolonged significantly (51.7 ± 24.8 min, $p < 0.05$), and 65% (13/20) of patients had abnormal GET1/2. Coma, as indicated by the Glasgow Coma Scale score, was not a statistically significant factor influencing GET1/2 ($p > 0.05$). **Conclusion:** NT can cause significantly prolonged GE, especially in patients with high-level SCI.

Key Words: gastric emptying; neurologic trauma; head injury; spinal cord injury

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In humans, vomiting, abdominal distention and increased gastric residuals after neurologic trauma (NT) suggest the presence of abnormal gastric motility. Despite the clinical significance of abnormal motility, little is known about the gastric effects of NT (*1–3*). Motility abnormalities might

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predispose gastric reflux, associated with increased gastric acid secretion. The presence of these abnormalities may also explain the gastrointestinal (GI) complications of ulceration and hemorrhage that sometimes occur after NT (*3,4*). Delayed gastric emptying (GE) has been described in a variety of neurological disorders including bulbar poliomyelitis, brain tumors, tabes dorsalis and diabetic gastroparesis (*5*). The rate and completeness of GE are major determinants of the bioavailability of oral medication, and the efficiency of GE is highly dependent on an intact central nervous system. Hence, in NT, an impairment in GE could significantly diminish drug absorption and increase the risk of inhalation of gastric contents (*6,7*). In addition, there are important implications for the management of NT patients who do not tolerate enteral feedings given orally or by nasogastric tube (*7*). From a review of the literature (*1,6,8–13*), we found that GE was studied in only a small number of NT patients. Discrepancies among previous studies are due to differences in concepts and methodologies and have created some controversy with regard to the effect of NT on GE. Thus, in this study, gastric emptying half-times (GET1/2s) of solid and liquid meals in NT [including spinal cord injury (SCI) and head injury (HI)] patients were evaluated by practical radionuclide imaging techniques.

MATERIALS AND METHODS

Patients

A prospective study was conducted to assess the GET1/2 of radionuclide-labeled meals in 50 male NT patients. The GET1/2 of solid meals was measured in 30 male SCI patients and the GET1/2 of liquid meals was measured in 20 male HI patients. Among 30 SCI patients (Table 1), 12 patients had a high-level injury (quadriplegic, above the fifth thoracic spine, T5), and 18 patients had a low-level injury (paraplegic, below the twelfth thoracic spine, T12). Among 20 HI patients (Table 2), 10 patients had a low Glasgow Coma Scale (GCS) (*14*) score, and 10 patients had a high GCS score.

Before evaluation, none of the 50 NT patients had a history of intra-abdominal trauma, abdominal surgery or other systemic diseases such as diabetes mellitus, uremia or connective tissue disease or were being treated with drugs known to alter GE.

Thirty-two healthy, ambulatory, unmedicated and age- and weight-matched men were enrolled as control subjects. Eighteen control subjects underwent the same imaging technique as that used for the 30 SCI patients to evaluate the normal range of the solid meal GET1/2 (84.5 ± 16.7 min, mean ± s.d.) (Table 3). Fourteen control subjects underwent the same imaging technique as that used for the 20 HI patients to evaluate the normal range of the liquid meal GET1/2 of (29.2 ± 3.7 min, mean ± s.d.) (Table 4).

Independent Student's t-tests were used to evaluate the statistical differences in the mean of the GET1/2 between the following two subgroups: high- versus low-level injury in SCI patients and low versus high GCS score in HI patients. If the p value was < 0.05, the difference in the mean of the GET1/2 sec between the two subgroups was considered significant.

If the GET1/2 of a solid meal was >117.9 min (longer than mean + 2 s.d. of the GET1/2 of the 18 control subjects), it was considered to be prolonged and abnormal. If the GET1/2 of a liquid meal was >36.6 min (longer than mean + 2 s.d. of the GET1/2 of the 14 control subjects), it was considered to be prolonged and abnormal. Chi-square tests were used to evaluate the statistical differences in the incidences of abnormal GET1/2 between the following two subgroups: high- versus low-level injury in SCI patients and low versus high GCS score in HI patients. If the p value was <0.05, the difference in the incidence of abnormal GET1/2 between the two subgroups was considered significant.

TABLE 1
Detailed Data of 30 Spinal Cord Injury Patients

Patient no.	Age (yr)	Injury level	Injury duration (yr)	GET1/2 (min)
1	21	Low	Short	186.9*
2	21	High	Long	102.5
3	24	Low	Long	251*
4	25	Low	Short	135.4*
5	25	Low	Long	129.4*
6	26	High	Short	84.2
7	27	Low	Long	163.5*
8	28	High	Long	185.4*
9	29	High	Long	94.7
10	30	Low	Short	105.6
11	33	Low	Long	112.3
12	34	High	Short	176.3*
13	34	Low	Short	105.1
14	36	High	Short	187.2*
15	38	Low	Long	99.3
16	38	Low	Long	82.1
17	40	Low	Short	103.5
18	42	High	Long	253.1*
19	42	Low	Long	89.6
20	42	Low	Long	95.6
21	43	High	Short	186.4*
22	44	High	Short	194.2*
23	44	High	Short	152.7*
24	45	Low	Short	105.6
25	46	Low	Short	121.3*
26	46	Low	Short	87.6
27	47	Low	Short	94.7
28	47	High	Long	135.7*
29	48	High	Long	204.5*
30	48	Low	Long	123.6*

*Abnormal GET1/2.
GET1/2 = gastric emptying half-time.

Gastric Emptying of Solid Meals

The standard solid meal consisted of two fried eggs, mixed with 500 μCi ^{99m}Tc-phytate, placed between two pieces of toast to form a sandwich. The total meal weighed 300 g, and it contained 312 Kcal: 28% protein, 15% lipid and 57% carbohydrate (*15*). SCI patients and control subjects fasted for at least 8 hr before the test. Each subject was asked to eat the meal within 5 min. These studies were performed with the subjects in the supine position and a gamma camera (Apex 609R, Elscint, Haifa, Israel) in a left anterior oblique position. The period of data collection began as soon as the subjects had finished the meal. The data from the solid-phase studies were acquired for 30 sec per frame, at 10-min intervals, for a total of 90 min. A region of interest (ROI) corresponding to the stomach was outlined to determine the gastric counts for each image. Correction was made for radioactivity decay. A power exponential fit was used to analyze the time-activity curve over the stomach and to calculate the solid meal GET1/2 (*15–17*).

Gastric Emptying of Liquid Meals

The standard liquid meal consisted of 500 ml 5% glucose water (25 g glucose, 100 Kcal) labeled with 500 μCi ^{99m}Tc-phytate (*15*). Each subject was asked to intake the liquid meal within 1 min, the HI patients through nasogastric tubes and the control subjects by the mouth. These studies were performed with the subjects in the supine position and with the gamma camera (Elscint Apex 609R) in the left anterior oblique position. The period of data collection began as soon as the subjects had finished the meal. The data were

TABLE 2
Detailed Data of 20 Head Injury Patients

Patient no.	Age (yr)	Injury duration	Glasgow Coma Scale	GET1/2 (min)
1	20	Short	High	102.3*
2	23	Long	High	23.6
3	24	Long	Low	24.1
4	27	Long	Low	30.2
5	29	Short	High	84.3*
6	29	Long	Low	36.9*
7	29	Long	Low	32.1
8	29	Long	Low	29.1
9	31	Long	High	18.6
10	31	Long	High	21.3
11	32	Long	Low	55.9*
12	34	Long	High	63.8*
13	36	Long	Low	56.1*
14	38	Long	Low	54.8*
15	41	Short	Low	87.3*
16	41	Short	High	50.9*
17	42	Short	Low	85.4*
18	48	Long	High	57.2*
19	52	Short	High	68.9*
20	52	Short	High	50.6*

*Abnormal GET1/2.
GET1/2 = gastric emptying half-time.

acquired for 20 sec per frame, at 20-sec intervals, for a total of 30 min. An ROI corresponding to the stomach was outlined to determine the gastric counts for each image. Correction was made for radioactivity decay. A power exponential fit was used to analyze the time-activity curve over the stomach and to calculate the liquid meal GET1/2 (*15–17*).

RESULTS

The standard solid meal consisted of two fried eggs mixed with 500 μCi ^{99m}Tc-phytate and placed between two pieces of toast to form a sandwich. To evaluate the stability of the phytate label for the solid meal, the sample of ^{99m}Tc-phytate-labeled fried eggs (5 cm^3) was suspended in a test tube with 10 ml gastric juice, then the tube was put into a shaking water bath (37°C) to incubate for 120 min. After the sample was centrifuged and washed, the radioactivity within the sample of fried eggs and within the supernatant liquid was counted, respectively, by a gamma counter. The radioactivity in the sample of fried eggs was over 97%. The results suggest that ^{99m}Tc-phyate-labeled eggs are stable enough in the stomach for at least 2 hr, and they should be used as a standard solid meal in GE studies.

TABLE 3
Gastric Emptying Half-Times of Solid Meals in 18 Healthy Chinese Men

Patient no.	Age (yr)	GET1/2 (min)
1	18	73.5
2	19	75.8
3	23	50.7
4	25	65.2
5	25	112.8
6	27	89.4
7	28	84.8
8	30	89.9
9	33	103.2
10	33	66.2
11	41	77.1
12	41	94.2
13	41	91.4
14	42	107.9
15	42	84.7
16	42	92.9
17	46	62.9
18	53	98.1

GET1/2 = gastric emptying half-time.

TABLE 4
Gastric Emptying Half-Times of Liquid Meals in 14 Healthy Chinese Men

Patient no.	Age (yr)	GET1/2 (min)
1	27	29.7
2	33	33.9
3	36	34.0
4	41	29.4
5	41	21.1
6	41	27.9
7	42	26.9
8	46	26.2
9	46	32.9
10	46	29.6
11	50	25.6
12	50	32.9
13	59	32.2
14	59	26.7

GET1/2 = gastric emptying half-time.

The detailed data of the 30 SCI and 20 HI patients in our study are shown in Tables 1 and 2, respectively.

In the 30 SCI patients, the GET1/2 of a solid meal was significantly prolonged (138.3 ± 49.2 versus 84.5 ± 16.7 min, mean ± s.d., $p < 0.05$), and 53% (16/30) of patients had an abnormal GET1/2. High-level injury (163.1 ± 50.5 min, 75%, 9/12) patients had statistically prolonged GET1/2s or higher incidences of abnormal GET1/2 than low-level injury (121.8 ± 41.9 min, 39%, 7/18) patients ($p < 0.05$).

In the 20 HI patients, the GET1/2 of a liquid meal was significantly prolonged (51.7 ± 24.8 versus 29.2 ± 3.7 min, mean ± s.d., $p < 0.05$), and 65% (13/20) of patients had an abnormal GET1/2. High GCS score (54.2 ± 27.6 min, 70%, 7/10) patients had prolonged GET1/2s or higher incidences of abnormal GET1/2 than low GCS score (49.2 ± 22.9 min, 60%, 6/10) patients. However, the difference was not significant ($p > 0.05$).

DISCUSSION

The use of radionuclide methods to study gastric emptying in humans began in 1966 when Griffith et al. (*18*) used an orally administered ^{51}Cr-labeled meal and a scintiscanner to quantify GE. The major advantages of this technique were that it was simple, noninvasive, avoided the need for sampling of intragastric contents and was highly acceptable to patients, unlike radiological or intubation methods. Radionuclide techniques are both reproducible and sensitive and, consequently, have widespread clinical and research applications. Therefore, in this study, we used radionuclide-labeled solid and liquid meals to evaluate GET1/2 in patients with NT, including SCI and HI patients.

Patients with SCI may have severe disturbances in autoregulatory and homeostatic mechanisms mediated by the autonomic nervous system. The motility of the stomach is controlled by parasympathetic and sympathetic nervous innervation. The function of the parasympathetic innervation (vagus nerve) is to increase the motility of the stomach, whereas the sympathetic innervation generally decreases the motility of the stomach (prolonged and abnormal GET1/2). The parasympathetic nervous innervation arises from the brain stem, and it is not influenced directly by SCI. However, sympathetic nervous innervation to the stomach arises from levels T5 through T12. Therefore, in high-level SCI injury (above T5), disconnection of the intact thoracolumbar sympathetic outflow from the brain stem results in the development of excessive splanchnic sympathetic activity of the isolated thoracic cord. The results are decreased gastric motility with a prolonged GET1/2. In low-level SCI injury (below T12), the connection of supraspinal autonomic centers with the segmental sympathetic outflow to the upper GI tract is intact, and autonomic hyperreflexia and prolonged GE do not develop (*10,19,20*). These theories support our results of prolonged GET1/2s and higher incidences of abnormal GET1/2 in SCI patients with high-level injuries than in SCI patients with low-level injuries.

Brain abnormalities can adversely affect the function of the upper GI tract (*21–24*). However, the possible causes of altered GE in HI patients are multiple and complicated. First, stress, Koo et al. (*25*) found that cold-restraint stress delayed GE in rats. That study suggested that stress affects the gut through the sympathetic system. Thompson et al. (*26*). found that cold and pain stresses significantly delayed GE in healthy adults. These studies emphasized the role of the central nervous system in the control of gut function. Second, increased intracranial pressure (ICP), Garrick et al. (*27*). found that increased ICP immediately suppressed the amplitude of gastric contractions by more than 80%. Lowering ICP to the normal range was the only method found to normalize gastric function. Rimmer (*28*) found that increased ICP significantly delayed GE in a patient with a brain tumor. Third, cytokines, Van Miert and De La Para (*29*) found that endotoxin and lipopolysaccharides are strong inhibitors of GE in rats. Nompleggi et al. (*30*) found that recombinant interleukin-l, when injected intraventricularly, significantly decreased GE in rats. Work from another study showed that patients who sustain HI have significantly elevated levels of interleukin-1 in the cerebrospinal fluid (*31*). Fourth, corticotropin-releasing factor, Tache et al. (*32*) reported that intracisternal injection of corticotropin-releasing factor dependently inhibited GE by 37%–80% in rats. Ott et al. (*12*) found that corticotropin-releasing factor was detectable in 2 of every 5 patients with severe HI. In this study, we found that HI patients often have GE abnormalities (13/20, 65%). From a review of the literature, we found that GE has been measured in only a small number of patients with HI, often by different nonisotope methods (*11–13*). In all previous studies, a trend toward slower GE in HI patients was observed (*11–13*). Inoue et al. (*33*) suggested that delayed GE was related to the comatose stage in patients with HI. However, in our study, when high GCS-score patients and low GCS-score patients were compared, there were no significant differences in the GET1/2 or in the incidence of abnormal GET1/2.

CONCLUSION

We conclude that a prolonged GET1/2 is likely to occur in NT patients. It is important to understand which factors will lead to a higher incidence of abnormal GET1/2s to increase the therapeutic efficacy of orally administered medications in NT patients.

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