A NEW METHOD OF STUDY
OF UPPER GASTROINTESTINAL
TRANSIT TIME AND SECRETION
IN ILEOSTOMY PATIENTS

by

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A Thesis submitted in partial fulfilment of
the requirements of the degree of
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in the Department of Surgery

We accept this thesis as conforming
to the required standard

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ABSTRACT

There is a need for a simple, safe, reproducible and non-invasive method for studying upper gastrointestinal motility in humans. Existing methods, measuring electrical contractions and intraluminal pressure changes have limitations in their correlation with the physiology of what is actually happening to ingested food.

Transit time has been suggested as a more physiologic means of studying gut motility; therefore a method was developed to measure transit time and secretory changes in response to ingested liquids, using ileostomy patients.

2.5 gm of polyethylene glycol (PEG) was added to 500 ml of normal saline, and given orally to volunteers with ileostomies. The ileostomy effluent was collected for 2 hours in 10 minute aliquots. PEG assay was performed by the turbidimetric method of Hyden, using Malawer's modification with an emulsifier.

The following were measured: most rapid, mode, median, mean and total transit times.

A study was then performed to determine if different foodstuffs - carbohydrate, fat, and protein - produce measurable changes in transit time. 2.5 gm of PEG was added to 500 ml of (a) 90 ml Lipomul in 410 ml normal saline (b) 5% dextrose (c) 100 ml of Travasol 10% in 400 ml distilled water. The volumes were chosen to produce isoosmolar test feeds.

Validating studies showed satisfactory reproducibility and individual variation (r = 0.68 for volume recovery, r = 0.69 for PEG recovery, p = < 0.5)
The recovery pattern of a test feed of 500 ml normal saline was found to follow a skew distribution, with mode, median and mean transit times all different. The most reproducible and easily measured, was mode, or peak, transit time (average 40 minutes for volume and PEG recovery).

Significant delays in all transit times were found (p = < 0.01) using each of the test feeds: (a) for Lipomul a peak volume recovery of 60 minutes and PEG recovery of 70 minutes; (b) for 5% dextrose a peak volume recovery of 90 minutes and PEG recovery of 90 minutes; (c) with Travasol, negligible amounts of ileostomy output were obtained over 2 hours.

The most rapid transit time was consistently less than 10 minutes, as measured by PEG appearance from the ileostomy. This is far less than previously described by standard methods, but is in accordance with transit times measured to the ileocaecal valve in intact gastrointestinal tracts using the recently-introduced breath hydrogen method following lactulose ingestion.

Comparison of total volume recovery with total PEG recovery over 2 hours indicates whether net absorption or secretion has occurred: (a) with normal saline a volume recovery of 62% and PEG recovery of 48% indicates net secretion; (b) with Lipomul a volume recovery of 66% and PEG recovery of 58% also indicates net secretion, with no significant difference from normal saline (p = < 0.05); (c) with 5% dextrose a volume recovery of 4% and PEG recovery of 13% indicates net absorption, significantly different from normal saline (p = < 0.01); (d) for Travasol a volume recovery of 1% and PEG recovery of 1% indicates no net absorption or secretion, but confirms the above finding of a very large delay in transit time.
These studies have shown that isotonic solutions of normal saline, glucose, fat and protein result in widely different peak transit times in ileostomy patients. They also result in widely different fluid outputs from the ileostomy due to net absorption or secretion. These differences have not been described before.
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Because of the relative inaccessibility of the small bowel for investigation, and also perhaps because of the relative rarity of diseases affecting it, little is known about the physiology of small bowel function. The main areas of study have been motility and transit time.

**Intestinal Motility**

"Motility" has been intensively investigated by methods measuring electrical activity and mechanical contractions (1). The electrophysiology of small bowel activity has been characterized into a Basic Electrical Rhythm (B.E.R. or "slow waves" Figure 1), not directly initiating contraction, but determining its distribution and frequency, as a pacemaker, and Action Potentials ("Spikes" Figure 2), associated with smooth muscle contraction (2,3).

Intestinal contractions have been separated into 4 patterns - Segmental, Sequential, Peristaltic Rushes, and Retrograde (1). Measurement of intraluminal pressure using water filled balloons, open-tipped fluid-filled catheters connected to external transducers (4), and radiotelemetering devices, has shown 4 basic types of contraction, based on pressure and duration. Each contributes to the overall pattern of contraction (5).

Attempts have been made to relate these observations to the progress of chyme (1,6). None of these studies however, have been very rewarding in the basic understanding of the physiology and pathophysiology of intestinal function (7). The modifying influences on motility have been studied in detail, namely the autonomic nervous system, chemicals, and hormones (1). Cholinergic parasympathetic nerves stimulate smooth muscle and facilitate contraction, whilst adrenergic and noradrenergic activity inhibits contraction. There is also a definite pathway, via the thalamus and hypothalamus, whereby external stimuli that evoke an emotional response can
**FIGURE 1** Diagram of Slow Waves in the small intestine (from Bockus HL, ed. Gastroenterology, 3rd ed.)

- **Type Waves**
  - Slow waves (HER)
  - Slow waves
  - Action potential
  - Muscle contraction

- **Locus**
  - Longitudinal muscle layer
  - Circular muscle
  - Diffuse

**Electrodes**

**Longitudinal:**

Electrical activity and pressure recorded from the duodenum of an unanesthetized dog. At an earlier operation, the duodenum was brought out through the abdominal wall and covered by a skin flap so that it formed a permanent handle-like loop. Electrical recording was made from needle electrodes inserted through the muscular layer of the skin; pressure was recorded by means of an open-tip catheter inserted through a thin-walled hypodermic needle.

**FIGURE 2** Showing how Slow Waves and Action Potentials contribute to intestinal contractions (from Bockus HL, ed. Gastroenterology, 3rd ed.)
influence the autonomic nervous system, and hence intestinal activity (8,9,10). Serotonin (5HT) may have a physiologic role in peristalsis, possibly by stimulating ganglia, sensitizing smooth muscle to the actions of acetylcholine, and sensitizing stretch receptors in the mucosa, lowering their threshold (11,12).

In the fasting state, the human small intestine shows only minimal motor activity. After a meal is eaten and food enters the duodenum, the motor activity of the intestine increases markedly (12). The function of this activity is to mix the food thoroughly with the digestive juices (effected mainly by segmenting contractions), and to transport the mixing and digesting contents of the intestine onward from the duodenum (effected by both segmenting and peristaltic contractions).

In general (13), the motor and electrical activities of small intestinal muscle are increased by cholecystokinin-like peptides (cholecystokinin - pancreozymin, and gastrin) and also prostaglandin-E, and decreased by secretin-like peptides, with the possible exception (14) of vasoactive intestinal peptide (VIP). The following table summarizes current thinking:

<table>
<thead>
<tr>
<th>Stimulate small bowel contractions</th>
<th>Inhibit contractions</th>
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<td>Cholecystokinin</td>
<td>Secretin</td>
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None of these actions have been demonstrated physiological. What is known is: (1) cyclic bursts of gastroduodenal contractions that occur during fasting are accompanied by increased plasma motilin levels. (2) contractions characteristic of the fed state are copied by exogenous administration of gastrin and CCK. The effect of GIP on small bowel motility has not been investigated. The effect of intravenous cholecystokinin is both rapid and striking, and considerably more marked than the effect of gastrin. All regions of the small intestine appear to behave in the same way, with the exception of the second part of the duodenum, adjacent to the sphincter of Oddi, which relaxes in response to cholecystokinin. The increased motor activity produced by cholecystokinin is accompanied by a corresponding decrease in the time taken by the contents of the small intestine to reach the colon (12).

**Transit Time**

Transit time has been examined less than other aspects of gastrointestinal motility, but may represent a more physiological assessment of what is actually happening to ingested foods.

Borgstrom in 1958 described a method for analyzing the digestion and absorption process, by sampling chyme at different levels of the intestinal tract through holes in polyvinyl tubing, under conditions very closely resembling the physiologic state (15). Longstreth has since confirmed that the presence of a transpyloric tube does not alter gastric motility, secretory or hormonal responses (16). A reference substance, polyethylene glycol, was incorporated as a marker into test meals, and the appearance time, volume and composition measured by aspiration of samples at intervals along the intestine. Developments of this original technique have led to the dye dilution method, marker washout technique, and the use of markers for perfusion studies to measure absorption from or secretion into the lumen (17). Small bowel transit, flow rates and peak velocities can be measured.
Markers

A variety of markers is available, the criteria being that they should be inert, non-absorbable, mix uniformly, readily assayed, and completely recoverable (18).

In the radiological method, barium sulphate is the usual marker, and similarly vegetable dyes such as carmine red are easy to detect (19), but measure only the head and tail of a stream. Barium may also give a falsely short time, and its transit time related to that of normally ingested substances remains to be established.

Colored beads, non-digested seeds, such as sesame, and radiopaque pellets are useful, mainly for mouth-to-anus transit time (19).

Chemical markers, such as polyethylene glycol and chromium sesquioxide, give consistent recoveries (20) and, depending to some extent on the homogenicity of the intestinal contents (21), distribute evenly under normal conditions and accurately measure transit time (22,23). Isotopic variants are also available, with the advantage of easy assay by scintiscanning (24). Markers can be given as a bolus (dye dilution) or intermittently (marker washout) or continuous infusion (perfusion studies). The measurement of breath hydrogen following lactulose ingestion was introduced recently by Levitt (25) and is being increasingly used.

Polyethylene glycol (PEG) is commonly used as a non-absorbable volume marker in intestinal perfusion and flow studies. It has been assumed that PEG does not affect water and electrolyte movement, but recent studies have shown (27) a progressive reduction in water and electrolyte absorption in the jejunum and ileum with increasing concentrations of PEG in the perfusing fluid. It seems that this most likely results from an osmotic effect rather than an inhibition of active absorption or stimulation of secretion, as the effect is mainly seen at PEG concentrations above 5 gm/litre.
Known Transit Times

Most available information has been derived from barium studies. These measure the most rapid transit time, from the moment gastric emptying starts, to the arrival of the head of the column at the ileo-cecal valve. These show a large variation in normal subjects, with a range 15 min - 5 hours in 315 subjects, mean 84 min., reported by Kim (28). Texter found a range of 2-3 hours (29). Marine-Fiol reported a range of 2 1/2 - 6 hours in 49 normals (30). In a classic study, Lonnerblad found a mean transit time of 178 minutes in 108 subjects (31).

These studies also showed poor reproducibility in a given subject: Lonnerblad found an average 66 minute difference from the 1st measurement in 35 repeat studies. This mean standard deviation for a single determination could be 1 hour.

Burkett has stated it is the total time ingested material in contact with the bowel wall that is important (32), and therefore records total transit time - usually measured as the time for all ingested pellets to be passed. Other workers measure mean transit time (33), expressed as $\sum \frac{tm}{m}$

where $t$ = ingestion to recovery time of samples

$m$ = amount of marker in sample

Bond and Levitt have shown a transit time average of 72 minutes (range 25-118) for 10 gm. of Lactulose, using polyethylene glycol as a marker (25). Individual reproducibility they found +14%. They were, however, using a collecting tube extending down the entire length of the small bowel to collect ileal samples, and this may not represent a physiological situation.

It is known that the volume and composition of ileostomy fluid differs from that passing the normal ileo-cecal valve (33). Soergel and Hogan however,
have studied transit time in ileostomy patients using polyethylene glycol as a marker and found a mean transit time of 1 1/2 hours, similar to that obtained for small bowel in intact gastrointestinal tracts (22).

Factors Affecting Transit Time

Transit time is the product of a variety of conditions of motor activity of the gut and physical state of the food.

Using barium, Golden has stated that most rapid transit time is not influenced by the rate of emptying of the stomach (34). Weigen confirmed this in dogs (35), and Lonnerblad's studies in humans also showed no appreciable change in stomach emptying time with respect to transit time. He also found that emotional factors (fright, apprehension) could not be shown to markedly influence transit time.

Meeroff states that gastric emptying appears to be regulated by osmolality of duodenal contents. Ostick has studied the effect of consistency of food on gastric emptying (36), and Bennett has shown stimulation of intestinal propulsion by lowered intraluminal pH (37).

The possibility of "streaming" in the gastrointestinal tract has been raised by several authors. Streaming occurs if there is a differential flow rate between the solid and liquid phases of the gut contents. Findlay concluded that streaming does not occur in normal subjects (21).

Waller has suggested colonic transit, rather than gastric emptying or small bowel transit time, determines overall transit time, at least in patients with gross abnormalities of total transit time - constipation and diarrhea (26). Harvey however, states that the orderly assimilation of a meal is in large part dependent upon optimal rates of gastric emptying and of small bowel transit, and that changes in motor activity after food, seen at all levels of the gastroentestinal tract, are markedly influenced by gastrointestinal hormones (10).
Gastrointestinal Hormones and Secretion

In 1938 Nasset reported that certain extracts of intestinal mucosa stimulate intestinal secretion (38). He named the active principle "enterocrinin".

In addition to the three recognized gastrointestinal hormones, secretin, gastrin and cholecystokinin, there are a large number of what might be called candidate hormones. Of the three well known hormones, only gastrin (and pentagastrin) stimulates intestinal secretion. Glucagon, a pancreatic islet hormone, also stimulates (39). Two other peptides, vasoactive intestinal peptide (VIP) and gastric inhibitory peptide (GIP) have also been shown to stimulate small gut secretion (39,40,41,42,43,44).

The effect of these peptides on gut secretion has been shown to be similar to that of cyclic AMP, stimulation of the adenyl cyclase system being the likely final common path for all known stimulants, including cholera toxin and prostaglandins (45). Excessive secretion from the gut is a well known pathological phenomenon, best illustrated by cholera. Rates of fluid secretion similar to those produced by cholera toxin have been produced in response to several gut peptides (39,46,47).

As in cholera, the protein content of these secretions is low, indicating that mucosal integrity has been maintained, unlike the secretion resulting from increased interstitial hydrostatic pressure. The possibility has been raised that cholera toxin acts at least in part by releasing one of the gut peptides (53). Some pancreatic islet-cell tumors are known to produce VIP, causing depressed gastric secretion and watery diarrhea.

A different explanation, based on the same observations, would be to regard the small bowel not as an organ which receives some 2 litres of gastric and
digestive secretions into its proximal end each day and passes 1 litre
distally through the ileocaecal valve, but as an organ in active flux,
analagous to the kidney. If, say, 50 litres of intestinal secretions were
produced daily, and 49 litres actively resorbed, through a cyclic AMP-mediated
mechamism, the net result would be the same. Profound changes could then be
brought about by any interference with cyclic AMP. It is possible that
cholera toxin, and also the peptides increasing net secretion, may act by
blocking the cyclic-AMP mechanism. Conversely, potentiation of the resorptive
mechanism would produce a decrease in net secretion.

GIP was isolated from a side fraction obtained during the purification of
cholecystokinin - pancreozymin by Brown in Vancouver (48,49). It is a
candidate for the theoretical hormone "enterogastrone", suggested originally
by Kosaka and Lim, which is liberated by fat or its digestion products and
which inhibits gastric secretion and motility. In addition to an inhibitory
effect on acid secretion, GIP has been shown to be in insulinotropic,
potentiating insulin release to an intravenous infusion of glucose, and
improving glucose tolerance in man (44,50).

Thus GIP has been shown to have at least 3 diverse effects: the inhibition of
gastric acid secretion and motility (enterogastrone effect), release of
insulin (incretin effect), and stimulation of intestinal secretion
(enterocrinin effect). The physiologic significance of the enterocrinin
effect of GIP is still uncertain. Helman and Barbezat have shown (40,41),
using a triple lumen tube jejunal perfusion technique that during GIP infusion
there is a net reduction in sodium, potassium and bicarbonate absorption, and
that chloride flux is switched from absorption to secretion. They conclude
that GIP induces secretion from the small bowel.
Recent work has shown that physiological levels of GIP probably do not cause significant inhibition of gastric acid or pepsin secretion in humans (52), and that its principle effect would seem to be insulinotropic. Bloom has in fact suggested that the alternative title of "Glucose-dependent Insulinotropic Peptide" (still GIP) is preferable (53).

The effect of G-I hormones can be investigated by administration, or by provoking their physiological release. In humans, the latter is obviously preferable. Cleator and Gourlay in Vancouver have shown that oral ingestion of food substances, in particular glucose and fat, provoke the release of GIP (54). This has recently been confirmed by Williams (55), who has shown that only glucose of the common dietary sugars produces a strong stimulation, and that only triglyceride in the form of corn oil, rather than individual fatty acids, causes GIP release. Thomas has demonstrated weak stimulation of GIP secretion by intraduodenal amino acids (56). This is not confirmed in Williams' recent study.
PURPOSE OF STUDY

There is a need for a simple technique which reliably reflects the transit time of ingested foodstuffs in the small bowel, and which is practical, safe, reproducible and, therefore suitable as an experimental model for investigation of the control of gut motility.

The objectives in this work are to develop and validate such a method in ileostomy patients, and then to use it in patients with established ileostomies to determine the influence of various fluids and primary foodstuffs on upper gastrointestinal transit and secretion, and their possible relationship to GIP.

A study was designed to show if the oral ingestion of foodstuffs calculated to provoke hormone release, and specifically GIP, produce measurable changes in small bowel motility, measured by transit time, and in secretion.
MATERIALS AND METHODS

Patients were selected from those attending the Stoma Clinic at St. Paul's Hospital, Vancouver, who had long standing ileostomies as a result of ulcerative colitis, and were in good health, with normally functioning ileostomies. This was done with the help of the United Ostomy Association, and informed consent was obtained from the patient and family physician. The study was approved by the University of B.C. Medical Screening Committee for research involving human subjects. There were a total of 9 patients, 5 female and 4 male, with an age range of from 23-63 years. All had had satisfactory stomas for at least 5 years (range 5-17 years). This provided patients with normal small bowel, easily accessible for study.
Initially, attempts were made to collect the ileostomy effluent directly from the ileostomy. The stoma appliance was removed, and a red rubber catheter inserted into the stoma. After a number of accidents and spillages, and also finding that a catheter placed in an ileostomy which is functioning is rapidly extruded anyway, it was found that simply collecting directly from the stoma bag was satisfactory.

The volunteer emptied the stoma bag immediately before each experiment, then left it open and emptying into a 250 ml. Pyrex glass beaker. Fluid specimens were collected by dependent drainage, poured into a 100 ml measuring cylinder, the volume measured and then pooled after a 5 ml aliquot had been taken for subsequent PEG analysis.
PILOT STUDIES

Study 1
An initial series of tests using various volumes of normal saline (200 cc, 300 cc, 500 cc, 750 cc and 1000 cc) in 4 volunteers, fasted overnight, were performed to ascertain the volume of fluid required to ensure a continuously measurable ileostomy output for at least 2 hours, the period chosen during which the hormonal responses to the feeds would occur. Not all volunteers were studied at all volumes. The test volume was drunk continuously, in less than 2 minutes. 500 ml was selected as suitable, measurable aliquots being obtainable at 10 minute intervals, and persisting for 2 hours. Volumes less than 500 ml did not produce predictable responses, in some cases the ileostomy not functioning at all during the test period, and volumes over 500 ml were difficult for some volunteers to drink continuously.

Study 2
Various amounts of Polyethylene Glycol (PEG) were added to the test feeds in the same 4 volunteers (2.5 gm, 5 gm, 10 gm, and 15 gm) to produce concentrations of from 500 mg/dl to 1500 mg/dl, to determine the amount to be added to produce a PEG level above 200 mg/dl in the ileostomy output, the threshold for accurate analysis (see under PEG analysis). Not all test volumes were studied at all PEG concentrations. 2.5 gm PEG per 500 ml was selected, a concentration of 500 mg/dl.
Carmine Red (300 mg) was initially added to all feeds, to act as a visual marker of the most rapid and total transit times so that some indication could be gathered of when PEG might be appearing, for subsequent analysis, and also as a solid phase marker, to assess possible "streaming".

Study 3
Individual variation under standard conditions, that is reproducibility, was then assessed in a total of 4 patients, measuring volume and PEG recoveries of 500 ml normal saline on at least 2 occasions, on different days.
TEST FEEDS

Six ileostomy patients were given test meals of:

1. 500 cc 5% dextrose
2. 500 cc normal saline
3. 90 cc Lipomul + 410 cc normal saline

All feeds were isoosmolar and constant pH (7.4) by measurement in St. Paul's Hospital clinical laboratory. Test meals were labelled with 2.5 gm polyethylene glycol 4000 (500 mg/dl). An IV drip with normal saline was established in the forearm, and 5 ml blood samples were taken at F, 5', 10', 20', 30', 45', 60', 90' and 120' and aliquoted for GIP, glucose and insulin. Benzamidine (50 u moles/ml) was added to GIP aliquots as a preservative.

Ileostomy effluent following the test meal was collected every 10 minutes, the volume recorded, and the sample pooled after an aliquot was taken for later PEG analysis. An aliquot of the pooled effluent collected over the test period was sent to the St. Paul's clinical laboratory for electrolyte measurement.

By comparison of the optical densities of the treated PEG samples to a set of standards, a measurement of the concentration of PEG in mg/dl was made. This value multiplied by the volume of that particular sample gives the actual amount of PEG in that sample. It is therefore possible to know how much of the 2.5 gm initially given is recovered over the test period. This recovery of PEG, expressed as a percentage of the amount given in the test feed, can then be compared to the volume recovery of the same feed, and an indication is then obtained as to whether net absorption or secretion has occurred.

Further studies were later added in 3 patients using:

4. 500 cc 31/3% dextrose/saline 0.3%
5. 500 cc distilled water
6. 500 cc normal saline + 2 tablespoons bran
7. 100 cc 10% Travasol + 400 cc distilled water.

The study design was the same as above.
PEG ASSAY

PEG analysis is performed according to the method of Sidney J Malawer (52). This method involves the development of an oil-in-water emulsion of PEG when exposed to trichloroacetic acid in the presence of barium ions. The oil-in-water emulsion is stabilized by the addition of an emulsifying agent, gum arabic. This produces a stable and prolonged peak of maximum turbidity. An initial Ba (OH)_2 - ZnSO_4 - BaCl_2 precipitation/filtration step serves to remove protein and sulfates (interfering substances) from the reaction mixture. A standard curve of PEG concentrations ranging from 200 - 1100 mg/100 ml gives a linear relationship with the optical density at 650 μm and therefore affords determination of the unknown PEG concentrations to the nearest mg per 100 ml (Figure 13 and Table 15).

Duplicate determinations were performed on all standards and samples. Standard solutions of PEG (Polyethylene Glycol 4000, Union Carbine Corporation, New York. Average molecular weight 3000-3700) were prepared freshly each morning for a single day's experiments. A single lot number of PEG was used throughout this study.
To 50 ml. Erlenmeyer flasks containing 1 ml of distilled water (for the blank), a 1 ml PEG standard solution, or gut juice sample with PEG concentrations designed to be between 300 and 1100 mg per 100 ml, the following were added:

- 10 ml of water
- 1 ml of 10% (w/v) BaCl$_2$ anhydrous
- 2 ml of 0.3 N Ba (OH)$_2$

These were mixed by swirling after each addition

- 2 ml of 5% 2nSO$_4$ . 7H$_2$O

The flasks were then capped with parafilm and shaken vigorously.

The flasks are then left to stand at room temperature for 10 minutes, and then the contents filtered through double thickness Whatman no. 42 filter paper.

1 ml aliquots of the filtrates are transferred to 16 by 150 mm test tubes, 3 ml of gum arabic solution (Acacia Gum Arabic, U.S.P. powder. Fisher Scientific Co. Cat. No. 9-85) is added and mixed by gentle agitation. 4 ml of 30% trichloroacetic acid (TCA) containing 5% (w/v) BaCl$_2$ anhydrous, are added by pipette, the tubes capped with parafilm, and mixed by inverting 5 times.

This TCA reagent is added to the samples sequentially and each mixed immediately.

Sixty to ninety minutes later, the optical densities of the samples are read against the blank in standard 4 ml, 10 mm light path absorption cells, using a Pye Unicam Spectrophotometer set at 650 μm and a slit width of 0.04 mm.

A standard curve is constructed and the unknown samples are read from this graph to the nearest mg per 100 ml.

With this procedure, the final reaction mixture (8 ml) contains 0.19 to 0.69 mg of PEG if the original samples contained 300 to 1100 mg per 100 ml.
STATISTICS

Standard statistical methods are employed, with data collected suitable for analysis by paired t-test. Values are recorded as mean ± S.E.M. Regression analysis was used to plot correlation coefficients of control and reproducibility experiments using a logarithmic transformation. Detailed calculations were according to Bailey NTJ: Statistical methods in Biology, The English Universities Press, London, 1974.
RESULTS

Pilot Studies: Controls and Reproducibility

Study 1

Oral doses of 200 ml and 300 ml of normal saline produced negligible ileostomy outputs, varying between 0 and 5 ml per 10 minute interval. 500 ml produced a reliable and predictable response in all individuals tested, with a peak recovery at 40 minutes, returning to basal levels within 2 hours (Figure 3). Volumes greater than 500 ml produced the same recovery pattern, but the 10 minute aliquots became much larger (up to 390 ml) and rather unmanageable, and the volunteers had some difficulty drinking the volume continuously.

Study 2

It was found that addition of 2.5 gm of PEG to 500 ml of test feed (500 mg%) resulted in satisfactory levels for analysis, above 200 mg% in the ileostomy output.

PEG analysis of the specimens collected in Study 1 showed the same pattern for PEG recovery as for volume recovery (Figure 4), the striking finding being that measurable quantities of PEG appeared in all cases in the first aliquot, collected at 10 minutes. This is considerably faster than the most rapid transit times reported so far by other methods. Use of carmine red was discontinued after an initial 3 studies in which the volunteers were inconvenienced by its continuing appearance in ileostomy effluent for at least 24 hours. It initially appeared in less than 10 minutes in all 3 volunteers.

Study 3

Individual reproducibility proved satisfactory (Figures 3 and 4 showing readily identifiable recovery patterns with acceptable S.E.M. values).

Typical individual results are shown in Figures 5a and 5b.

The reproducibility of the method was tested with a regression analysis of pooled data, using logarithmic transformations of volume and PEG values (Figure 6). Correlation coefficients were 0.68 for volume recovery and 0.69 for PEG recovery, acceptable values for biological data of this sort, the statistical significance being $p = < .05$. 
EFFECTS OF TEST FEEDS

(a) Transit

A striking delay in the peak volume recovery (90 min) and PEG recovery (90 min) was shown in response to 500 ml of 5% glucose, and also in response to 500 ml saline containing 90 ml of Lipomul, which produced a peak volume recovery at 60 min. and PEG recovery at 70 min (Figure 7). Detailed results are presented in Tables 7 - 10. These are significant changes (P = < .01).
(b) Secretion

Large changes are seen when percentage recovery of volume and PEG over the 2 hours collecting period are calculated (Table 1). These are presented as Histograms in Figure 8.

There is no significant difference between the PEG recovery for saline and Lipomul - 62% and 58% (p = > .05), but a significant difference (p =< .05) between volume recoveries - 249 ml for saline (48%) and 323 ml for Lipomul (66%).

This indicates net fluid absorption with saline, and net fluid secretion with Lipomul.

The most dramatic, and unexpected, finding was with 500 ml of 5% glucose, where average volume recovery was just 21 ml at 2 hours (4%), and PEG recovery was 13%. (p = < .01). This indicates a very large net fluid absorption. In fact, in 2 of the 6 patients tested, there was no recordable output at all from the stoma for the 2 hour period.

The possibility of PEG absorption also has to be considered with these results. After this part of the study was completed and the results apparent, a further 3 tests were performed in the same patients using isoosmolar glucose in saline and 500 ml of distilled water.

Negligible amounts of ileostomy output were obtained over 2 hours (Tables 11 and 12), containing minimal PEG.

A further 3 studies were done using an isosmolar protein solution, 100 ml of 10% Travasol in 400 ml water, and minimal volume and PEG recoveries obtained over 2 hours (Table 13).
(c) **Electrolyte Composition**

Analysis of the total volumes collected over 2 hours for electrolytes is shown in Table 2.

This shows a net secretion of potassium with a saline test feed, and a larger net secretion with glucose or Lipomul.

There is net secretion of chloride in response to saline and no change with Lipomul.

There is net secretion of sodium in response to Lipomul when compared to saline.

Secretion of sodium and chloride in response to dextrose cannot be assessed because of the small volume of the samples, and could not be measured with dextrose in saline or protein because no samples could be obtained.
(d) Hormonal Responses

These are shown in Figure 9.

GIP, sugar and insulin levels were unchanged after normal saline ingestion, as expected.

Sugar and insulin levels were unchanged after Lipomul, but GIP increased to expected levels, although rather slower than expected. This may be related to the use for radioimmunoassay of an early antiserum, GP01.

GIP, sugar and insulin levels increased to expected values after oral glucose.
DISCUSSION

Validity of Method

This series of studies has shown that the method proposed for studying upper gastrointestinal transit time in humans, using ileostomy patients, is safe, simple, reproducible and subject to acceptable individual variation. The use of PEG 4000 as a Liquid-phase marker has been shown to consistently and reproducibly match the recovery of volume in control studies using oral normal saline.

This work shows that the recovery of volume and chemical markers following an oral saline load from an ileostomy follows a skew distribution. Therefore, mode, median and mean transit times will all be different (Figure 10). Previous studies of small bowel transit time have reported:

(a) most rapid transit time: for barium a range of 15 min - 6 hours, standard deviation 1 hour (31) and for PEG in lactose an average of 72 min with a range of 20 - 120 min and a reproducibility of + 14% (25).

(b) mean transit time: 90 min for PEG in saline (22)

(c) total transit time (18), which is probably best suited for measurement of mouth-to-anus transit.

This method conveniently provides a measurement of all these, and also allows mode and median values to be assessed.

Measureable amounts of PEG have always been present in the first 10 minute sample, thus the most rapid transit time by this method is consistently less than 10 minutes, and gives no useful comparative data. This in itself is considered an important finding in that most rapid transit times for small bowel this short have not generally been described before, although similar figures have been obtained by Levitt using the expired-breath $H_2$ measurement technique (25), an indirect method. The reasons for this variance from the literature may include: 1) most rapid transit times have not been measured
before in ileostomy patients 2) most rapid transit times have previously been measured by other methods - such as with barium, radiotelemetering capsules and with long transpyloric jejunal tubes.

The most reproducible and easily measured single value is mode or peak transit time. It is easily identified by inspection of the volume recovery data, is confirmed by subsequent analysis of PEG marker and is subject to little enough individual variation to make changes easy to detect. Peak transit time has not been used before in studies of intestinal motility.

Most work currently published using PEG as a marker employs Hyden's original method of Turbidimetric analysis, published in 1955 in a rather inaccessible journal (58). It took nearly a year to obtain a copy of this article through standard University of B.C. library procedures.

A modification, using an emulsifier, which simplifies the technique considerably by prolonging the stable phase of the emulsion, was published by Malawer in 1967 (57), but does not seem to have received attention.

This modification has provided extremely accurate estimation of PEG in our laboratory, correlation coefficients of standard plots being of the order of 0.997, and deserves wider use. The dose level of PEG used in this study was below the minimum level found to possibly exert an osmotic effect on absorption in the small bowel (27).
Advantages and Disadvantages

The advantages of a human model for studies of intestinal physiology are readily apparent: there is not the problem of whether the animal model reflects the human situation.

As already discussed, most information on small bowel function has been obtained either indirectly, or by rather unphysiologic means: long nasogastric tubes have been passed into the small bowel, often with balloons inflated, and the information obtained indirectly by perfusion methods. The model used in this study provides a direct method of studying the small bowel, using what is effectively a terminal fistula. In some ways this is reminiscent of the early studies on gastric secretion, using subjects with gastric fistulae. This method seems very suitable for the modalities under study, namely transit time and secretion.

A number of disadvantages of using a human model were also rapidly apparent during these investigations, despite the enthusiastic cooperation of the volunteers at all times. It was practicable to perform only one test per day, as the subjects fasted overnight, and thus only 3 or 4 per week, with inevitable cancellations due to illness, missed appointments, and problems with equipment and supplies. The work therefore proceeded much more slowly than other work in the department using animals. There were problems in the number of volunteers in collecting the required blood samples, due to poor peripheral veins which had been used repeatedly during hospital admissions, and 14 experiments had to be repeated or were incomplete because of this.
Possible Sources of Error

These can be considered under two categories; error inherent in the model, and errors in measurement.

The differences between the fluid emerging from an ileostomy and that passing a normal ileocecal valve have to be considered when interpreting data on secretion (33). With regard to motility, similar mean transit times have been observed from ileostomies as for small bowel in intact gastrointestinal tracts (21), but the absence of the ileocecal valve may possibly have an effect on transit.

Also, the absence of a colon in these patients has to be noted. Seal and Debas have shown (57) that the colon may well be the source of hormonal factors affecting at least gastric secretion. It is possible these hormones may also have an effect on small bowel.

None of the patients in this study had any difficulty taking the test feeds in a reproducible manner, in less than two minutes steady drinking.

The method of collecting samples was simple and provided volumes of ileostomy fluid of between 3 and 90 ml at 10 minute intervals, measured to the nearest 1 ml. The reproducibility figures already quoted support that methods of measurement were accurate for biological specimens of this sort.

The correlation coefficients quoted for our standard plots for PEG analysis (0.997) confirm that analytical error is extremely unlikely.

The main aim of this study has been to gather information on small bowel function: in the model used, mouth-to-ileostomy transit time has been measured. The test feeds were designed to all be equivalent, as far as known physical factors affecting gastric emptying are concerned, such as composition (Liquid), volume, osmolality, pH. In order to eliminate possible variations in gastric emptying rate and to obtain a pure measure of small bowel transit time, the original protocol called for a further series of tests to be done, instilling the test feeds directly into the duodenum via a nasogastric tube passed through the pylorus. This proved not possible within the time
available, due to lack of volunteers willing to comply. A subsequent investigator similarly was not able to perform these studies. A simpler method of measuring the effect of these test feeds on gastric emptying time has been devised (see further work).

A further consideration is that this method, measuring mouth-to-ileostomy transit time, may also be affected by other enteric secretions, including saliva, acid, mucons, bile, pancreatic juice, and succus entericus. The measurements obtained therefore will reflect the net effect of all these, less any absorption. The data obtained on secretion will also be the net effect of all these.
Effect of Test Feeds

This study has shown that oral ingestion of isotonic solutions of normal saline, fat, glucose and protein result in widely different transit times in ileostomy patients. They also result in widely different fluid outputs due to net absorption or secretion. These differences have not been described before. The test feeds were chosen to physiologically provoke the release of certain gastrointestinal hormones, G.I.P. in particular. The cause of these alterations in transit time and net fluid output may be due to the release of gastrointestinal hormones.
SUMMARY

This study was designed to validate a new method of studying upper gastrointestinal transit time and secretion in humans, using ileostomy patients, and Polyethylene Glycol (PEG) as a marker.

We have confirmed the accuracy of Malawer's modification, using an emulsifier, of Hyden's turbidimetric method of analysis of PEG.

We have found that, in a control situation, the recovery of PEG consistently and reproducibly matches the volume recovery of a test feed of normal saline from an ileostomy. It also matches the recovery of various foodstuffs given as test feeds, and provides an indication as to whether net absorption or secretion has occurred. In the situation of net fluid absorption in response to a particular foodstuff, such as glucose, the profound disappearance also of PEG from recovered samples must raise the possibility of absorption also of PEG: previously, PEG has been thought a non-absorbed marker.

We have found that the recovery pattern of a control test feed follows a skew distribution: therefore, the mode, median and average transit times will all be different. Previous studies of small bowel transit time have reported variously most rapid transit time, mean transit time, aor total transit time. This method conveniently provides a measure of all of them. We have found that the most reproducible and easily measured is mode, or peak transit time. This is readily identified by inspection of volume recovery data, and is confirmed by PEG analysis of samples, and is subject to little individual variation under standard or test conditions.

The most rapid transit time, by this method, measured by PEG recovery from the ileostomy, is consistently less than 10 minutes, far less than previously reported by other methods.

We have shown that isotonic solutions of normal saline, glucose, fat and protein result in widely different peak transit times in ileostomy patients. They also result in widely different fluid outputs from the ileostomy due to
net absorption or secretion. These differences have not been described before.

Further work includes investigation of whether these alterations in transit time and net fluid output are related to the release of gastrointestinal hormones.
Now that the method has been established and validated, and the observations made of the response to the various test feeds, a number of clinical applications have become apparent:

A patient with an ileostomy and short bowel syndrome due to small bowel resection, who was successfully treated with a reversed jejunal segment, has been studied and transit times measured in response to normal saline. The recovery pattern for volume and PEG is most interesting, and follows a biphasic pattern, with the second peak being strikingly larger (Figure 11). If this recovery pattern is compared to normal controls (Figure 12), one notes that the initial peaks coincide at 30 minutes, and that at 60 minutes, the time of the second peak in the study subject, there is a suggestion of a minor peak, or "hump" on the control curve. This could possibly be due to a reservoir effect in the normal small bowel, which acts to spread out volume transit by distending and effectively "ballooning", and this effect might be absent in a short bowel subject.

Patients with Koch-pouch "continent ileostomies" are being studied with respect to transit time and secretion.

Patients with ileostomies who catch influenza often become profoundly ill with uncontrollable diarrhea, dehydration and electrolyte imbalance. We have treated several such patients with oral doses of 50% glucose and have observed a dramatic diminution in ileostomy output to almost normal levels. A temporary diminution in output has been achieved in a patient with severe chronic ileostomy diarrhea, and also a diminution in the temporary diarrhea which follows refashioning of an ileostomy stoma. These empirical findings can now be studied further using this method.
Dietary fibre is known to exert a profound influence on gastrointestinal transit time, and its main effect has been presumed to be on the large bowel. The effect of bran on the small bowel is being assessed with this technique. So far, in three volunteers, no difference has been found in any of the types of transit time, measured by volume or PEG recovery, using bran in normal saline, when compared to controls with normal saline only.

The transit and secretory response to standard test feeds of normal saline is being studied during infusion of selected peptide hormones, commencing with GIP. This is to find out if any of the observed effect in response to lipomul and glucose can be ascribed to release of known hormones. Results are incomplete, due to problems with GIP supply and subsequently with GIP assay, but 3 tests have been performed so far (Table 14), giving a standard 500 ml of normal saline orally after an intravenous infusion of GIP has been established, designed to produce a physiological level of GIP in the systemic circulation. These have shown no significant change so far in peak or any other transit time by volume or PEG recovery ($p \geq 0.05$). Total volume recovery averages 55% (controls with oral saline alone 48%), and total PEG recovery averages 65% (controls 62%), no significant difference, and indicating a net secretion that is not significantly different from control experiments without GIP. Electrolyte levels in the total volume of ileostomy fluid recovered average exactly the same as control experiments with oral saline and no GIP infusion - Na 139, K 5.1, Cl 121 (Table 14). These preliminary findings would suggest that GIP has no effect on small bowel motility as measured by transit time, and has no effect on net secretion over a 2 hour period as measured by this direct method.
ACKNOWLEDGEMENTS

This work was carried out in a research year during residency training in General Surgery, under the direction of Dr. Iain Cleator, Director of the Gastrointestinal Clinic at St. Paul's Hospital, Vancouver, to whom I am very grateful for much help and encouragement.

Dr. Akira Nakayasu, an M.Sc. student from Kyoto, Japan, provided much stimulating discussion.

Radioimmunoassay of peptide hormones was done by Miss Nicola O'Connor, who also established our assay for polyethylene glycol.

Her successor in the G.I. Clinic, Mr. Douglas Burget gave constant help during the analysis and writing up of the data, and particularly with statistical interpretation.
FIGURE 3  Recovery of 500 ml oral normal saline from ileostomy
FIGURE 4

Recovery of 2.5 Gm dose polyethylene glycol (PEG) in 500 ml normal saline

PEG recovery mg

0 50 100 150 200 250 300 350

Time, minutes

0 5 10 15 20 25 30 35 40 45

n = 9
FIGURE 5a  Individual reproducibility. Recovery of 500 ml normal saline from ileostomy in one patient on two occasions.
FIGURE 5b  Individual reproducibility. Recovery of 500 ml normal saline from ileostomy in one patient on three occasions.
Reproducibility of method. A plot of volume recovery for one test against a second test on a different day, using logarithmic transformations.
FIGURE 7  The volume and PEG recoveries following 500 ml oral doses of normal saline, glucose and lipomul
FIGURE 8  The percentage recovery of volume and PEG from the ileostomy in response to test feeds of 500 ml normal saline, lipomul and glucose (5% dextrose), showing net fluid absorption with saline and glucose, and net secretion with lipomul.
FIGURE 9  Serum hormone responses to 500 ml oral saline, glucose and lipomul
FIGURE 10 Showing a Normal distribution (above), and a Skew distribution (below):
mode = most frequent value
median = arithmetic midpoint
mean = average value
(from Hays WL, Basic Statistics, 1967)
Figure 11

Volume and PEG recovery with 500 ml normal saline + 2.5 gm PEG in patient with reversed ileal segment and short bowel syndrome.
Volume recovery from Figure 11 compared to controls (n = 6).
Figure 13

Polyethylene glycol standard curve at optical density 650 μm.
<table>
<thead>
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<th>TEST FEED</th>
<th>MEAN %</th>
<th>S.E.M.</th>
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Table 1  Percentage recovery from ileostomy of 500 ml of various test feeds.
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Table 2  
Electrolyte composition of ileostomy fluid following various test feeds, and following GIP infusion.
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<th>7</th>
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**TOTAL**

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</table>

| %  | 48.44 | 15.44 | 5.1  |

Table 3

Volume recovery of 500 ml oral normal saline (see also Figure 3).
## Table 4

Recovery of 2.5 gm PEG in 500 ml normal saline (see also Figure 4)

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<th>Test Minutes</th>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
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<th>9</th>
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<th>S.E.M.</th>
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<td>52</td>
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<td>224</td>
<td>7</td>
<td>39</td>
<td>109</td>
<td>321</td>
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<td>2002</td>
<td>1117</td>
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<td>88</td>
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<td>77</td>
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<td>53</td>
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### TABLE 5
Reproducibility of method
Volume recovery for one test using 500 ml normal saline against a second test on a different day, with logarithmic transformations
(See also Figure 6)
<table>
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<tr>
<th>Time</th>
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<th>PEG</th>
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<th>Log</th>
<th>PEG</th>
<th>Log</th>
<th>PEG</th>
<th>Log</th>
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<td>0</td>
<td>28</td>
<td>1.44</td>
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<td>455</td>
<td>2.658</td>
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</table>

\[ r = 0.5734 \]
\[ \text{MR/MC} = 0.6883 \]
\[ p = 0.05 \]

**TABLE 6**  
Reproducibility of method. PEG recovery for one test, using 500 ml. normal saline, against a second test on a different day, with logarithmic transformations.  
(See also Figure 6)
### TABLE 7

Volume recovery (ml) after 500 ml oral lipomul in saline.
(See also Figure 7)
<table>
<thead>
<tr>
<th>Time</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>MEAN</th>
<th>S.D.</th>
<th>S.E.M.</th>
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<td>0</td>
<td>19</td>
<td>0</td>
<td>25</td>
<td>0</td>
<td>8.80</td>
<td>12.24</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>0</td>
<td>3</td>
<td>15</td>
<td>133</td>
<td>0</td>
<td>30.20</td>
<td>57.80</td>
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</tr>
<tr>
<td>40</td>
<td>21</td>
<td>2</td>
<td>16</td>
<td>415</td>
<td>161</td>
<td>123.0</td>
<td>175.6</td>
<td></td>
</tr>
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<td>50</td>
<td>46</td>
<td>1</td>
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<td>244.0</td>
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<td>91</td>
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<td>76</td>
<td>105</td>
<td>217</td>
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<td>74.23</td>
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<td>92</td>
<td>118</td>
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<td>50</td>
<td>418</td>
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<td>182.3</td>
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<td>142</td>
<td>80</td>
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<td>74</td>
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<td>82</td>
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<td>200</td>
<td>104</td>
<td>151</td>
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</table>

TOTAL 627 537 1126 2338 2129 1351 839.0

% 25 21 50 100 94 58 37.36 15.2

**TABLE 8** PEG recovery (mg) after 500 ml oral lipomul in saline
(See also Figure 7)
<table>
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<th>MEAN</th>
<th>S.D.</th>
<th>S.E.M.</th>
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<td>0.6</td>
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<td>1.64</td>
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<td>2.17</td>
<td>0.97</td>
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<tr>
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<td>0 3 0 0 0</td>
<td>0.60</td>
<td>1.34</td>
<td>0.6</td>
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</table>

**TOTAL** 0 68 0 20 16 20.80 27.91

**%** 0 14 0 4 3 4.20 5.76 2.3

*TABLE 9* Volume recovery (ml) after 500 ml oral glucose. (See also Figure 7)
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<th>3</th>
<th>4</th>
<th>5</th>
<th>MEAN</th>
<th>S.D.</th>
<th>S.E.M.</th>
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<td>1.34</td>
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<td>28.86</td>
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| TOTAL| 0 | 1494|0 | 10| 6 | 302.0| 666.4 |
| %    | 0 | 60 | 0 | 1 | 3 | 12.80| 26.41 | 10.7  |

**TABLE 10**  
PEG recovery (mg) after 500 ml oral glucose.  
(See also Figure 7)
TABLE 11

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<th>VOL</th>
<th>PEG</th>
<th>VOL</th>
<th>PEG</th>
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Volume (ml) and PEG (mg) recovery of 500 ml 3 1/3% Dextrose: 0.3% saline and 2.5 gm PEG.
<table>
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**TABLE 12**

Volume recovery (ml) of 500 ml water + 2.5 gm PEG.
## TABLE 13

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Volume (ml) and PEG (mg) recovery after 100 ml 10% Travasol + 400 ml water + 2.5 gm PEG.
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<th>VOL (ml)</th>
<th>PEG</th>
<th>VOL (ml)</th>
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<td>20</td>
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</table>

**TOTAL** 217 1317 238 1341 364 2066

| % | 43 | 59 | 48 | 54 | 73 | 83 |

**Na** 140 139 139

**K** 5.4 5.4 4.6

**Cl** 111 122 130

**Averages:**
- Volume recovery: 55%
- PEG recovery: 65%
- Na: 139 : K: 5.1 : Cl: 121

**TABLE 14**
Recovery of 500 ml normal saline with 2.5 gm PEG and GIP infusion 0.8 u units/kg.
<table>
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<th>PEG mg/100 ml</th>
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<td>.756</td>
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</table>

**TABLE 15** Optical densities for PEG standard curve (see also Figure 13)
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