Gastroparesis is a functional disorder of gastric emptying which is characterized by gastric stasis, atony, and dilatation with postprandial nausea and vomiting in the absence of mechanical obstruction of the pylorus or duodenum. It was first described and mainly observed in diabetics and has been attributed to autonomic neuropathy (1). Gastroparesis may lead to cachexia which is difficult to treat.

We observed a 16-year-old male patient who, after abdominal radiation for a testicular tumor, developed gastroparesis with gastric atony, marked antral hypomotility, severe postprandial vomiting, and extreme weight loss. During treatment with carbachol, but not with metoclopramide, antral motility and gastric emptying were restored. The patient became asymptomatic and quickly recovered. Remission continued even after termination of medication.

**CASE REPORT**

A 16-year-old boy underwent a right orchidectomy for a testicular seminoma; this was followed by radiation of the iliac and inguinal (4500 rad) and the paraaortic (4500 rad) region over six weeks. Three weeks after starting the radiation treatment, the patient developed progressive nausea and vomiting which persisted after the end of treatment and became increasingly worse during the following weeks. After four months, he suffered from regular postprandial vomiting even after intake of small quantities of fluid, had lost more than 20 kg of body weight, and his general condition was deteriorating. Six months after the onset of radiation he was referred to our institution.

On admission, the patient was cachectic (body weight 49 kg, height 191 cm), pale, and dehydrated. Temperature, pulse, and blood pressure were normal as was the examination of chest and abdomen. There were no signs of autonomic dysfunction (normal blood pressure responses to position, normal response to Valsalva maneuver), and no other abnormal physical findings were made. In particular, there was no evidence for persistence or recurrence of the testicular tumor.

Abnormal laboratory findings were: hemoglobin of 9.5 g/dl, potassium of 2.4 mmol/liter; chloride of 87 mmol/liter; and base excess of 8.0 mmol/liter. All other laboratory data including serum calcium and thyroid hormone levels were normal, as were x-ray of the chest and abdomen, and ultrasound and computed tomography of the abdomen. A gastroduodenoscopy revealed a dilated stomach with considerable amounts of retained secretion; the mucosa was normal, and no mechanical obstruction of pylorus or duodenum was noted.

A barium meal showed marked gastric dilation (Figure 1). Radiologically, fundus and corpus showed vigorous peristaltic contractions. In contrast, antrum and prepyloric region remained amotile and distended throughout the examination. The barium was retained in the stomach for several hours and was propelled into the duodenum only in a right-sided prone position. During emptying of the stomach in this position as well as in late films, no mechanical obstruction was present in the stomach or small intestine. Barium meals and motility studies (as described below) were performed after several days of gastric decompression and of total parenteral nutrition and restoration of normal potassium, chloride, and base excess levels.

**Motility Studies.** Gastric motility was estimated manometrically using a double-lumen gastric tube. The catheters were slowly perfused with saline (0.1 ml/min) using a Harvard infusion syringe and connected to strain gauges attached to a Gould recorder (Gould Inc., Cleveland, Ohio). The position of the tube was checked by fluoroscopy. Antral activities were recorded after an overnight fast. The effects of 10 mg of intravenous metoclopramide and of 0.25 mg of intramuscular carbachol on antral motility were tested on different days.

Basal antral activities were recorded for 8 hr; in the antrum no pressure activity was registrated. The catheters were slowly perfused with saline (0.1 ml/min) using a Harvard infusion syringe and connected to strain gauges attached to a Gould recorder (Gould Inc., Cleveland, Ohio). The position of the tube was checked by fluoroscopy. Antral activities were recorded after an overnight fast. The effects of 10 mg of intravenous metoclopramide and of 0.25 mg of intramuscular carbachol on antral motility were tested on different days.
Fig 1. Barium meal examination before carbachol treatment: marked gastric atony and dilation without antral peristalsis. No gastric emptying 85 min after the barium meal.

indicated that metoclopramide (10 mg, intravenous) had no effect on antral activity (Figure 2A). In contrast, intramuscular administration of 0.25 mg of carbachol evoked a vigorous increase in antral motor activity starting 18 min after injection (Figure 2B). To test whether antral motor activity induced by carbachol was effective in propulsing gastric contents, another barium meal study was carried out. Gastric dilatation and antral paresis similar to those observed in the initial examination were noted before administration of carbachol; however, 15-20 min after the intramuscular injection, intense peristaltic movements started and caused complete emptying of the stomach within 40 min.

Further Course. In view of these findings, we started a therapeutic trial with carbachol and administered 2 mg of the drug orally before each meal. From the day of starting treatment, no further postprandial vomiting occurred. The general condition of the patient improved quickly and completely, and no side effects were noted; parenteral nutrition was discontinued with no recurrence of clinical symptoms or abnormal laboratory data. Within two weeks, he had gained 8 kg of weight and was discharged home. During follow-up as an outpatient, the dose was gradually reduced, with no impairment of his well-being. Four months after discharge, drug therapy was discontinued, and no symptoms recurred. The patient remained in excellent health, with a body weight of 71 kg, without any complaints and without evidence for recurrence of the testicular tumor. All laboratory data had remained in the normal range.

Three months after termination of carbachol treatment, gastric anatomy and emptying were normal on barium meal and antral motor activity was now demonstrated manometrically. During follow-up for a further nine months, the patient has remained symptom-free.

DISCUSSION

This patient had the typical symptoms of gastroparesis with a failure of gastric emptying of both solid and liquid meals without endoscopic or radiographic evidence of mechanical obstruction. Extreme antral hypomotility in the presence of undisturbed fundic activity was demonstrated radiologically and manometrically.

The absence of antral motility during the height of this patient's illness might be attributed to inadequate sensitivity of our manometric recording device in a capacious viscus. This explanation seems unlikely in view of the ease with which the restoration of antral activity by intramuscular carbachol was recorded using the same recording system, and at a time when the stomach was just as capacious and hypomotile. Another possible interpretation is that during our observation, the antrum was continually in a phase of quiescence. The duration of our observation period suggests that this explanation is not applicable. Rather, it appears that the gastric pacemaker was preserved but motor activity was not propagated to the antrum. This concept is further supported by the fluoroscopic observation of a dilated, amotile antrum in the presence of vigorous contractions in the proximal stomach.

Since there was no history of diabetes mellitus or gastric operation, the disturbance was probably due to the radiation of the paraaortic region which involved large areas of the distal, but not of the proximal stomach. The alternative hypothesis of a paraneoplastic pseudoobstruction syndrome caused by the tumor rather than by its treatment appears less likely because the patient had no symptoms prior to treatment of the tumor, developed his disturbance only during abdominal irradiation, and had no evidence of persistent or recurrent malignancy in spite of severe gastrointestinal symptoms during hospitalization as well as during a further 16 month follow-up as an outpatient.

While gastroparesis is a well-recognized complication of diabetes mellitus (1-7) and of vagotomy (8, 9), the syndrome has not, to our knowledge, been