



# GASTROINTESTINAL INVOLVEMENT IN THE POST-POLIO SYNDROME (PPS)

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*This study has not been published by a peer-reviewed Journal -- or on the Internet until now, except for America Online. [Tom Walter](#) put together this article from talks the Dr. gave about it in the early '90's, plus feedback he gave to some local participants.*

## **BACKGROUND**

During the late 1980's to early 1990's, Dr. Anuras was Director, Division of Gastroenterology, Department of Internal Medicine at Texas Tech University in Lubbock. Along with Terri Bozeman, R.N., they surveyed over 750 post-polio patients regarding their gastrointestinal symptoms. Following report is from a paper they delivered at The Second Texas-Oklahoma Post-Polio Symposium, September 21-22, 1991, at the Ramada Inn of Wichita Falls, TX.

(To that point, they had included 500 patients in the results; [Table 3](#) below has been updated to reflect the symptoms of the first 754 participants in the study.)

## **ABSTRACT**

Gastrointestinal involvement is common in the post-polio syndrome, and it appears to affect the entire gastrointestinal tract. Unfortunately, there are only a few studies in this fascinating area. More extensive studies are needed to understand the pathologic and pathophysiologic processes in this problem, so that patients can be treated properly.

We report our survey of gastrointestinal symptoms that could affect up to 50 per cent of the post-polio syndrome patients in this review. We also propose the underlying pathophysiologic changes, outline the diagnosis and treatment for difficulties of various parts of the gastrointestinal tract.

## **INTRODUCTION**

Normal gastrointestinal motility is a function of the gastrointestinal smooth muscle and is regulated by both the intrinsic (myenteric plexus) and extrinsic nerves of the gastrointestinal tracts. Extrinsic nerves from the central nervous system (brain and spinal cord) connect the enteric nervous system (submucosal plexus and myenteric plexus) with the central nervous system. Therefore, any functional or structural abnormality of either the smooth muscles or the intrinsic or extrinsic nerves will result in gastrointestinal dysmotility causing gastrointestinal symptoms.

The symptoms produced depend on areas of involvement. This abnormality may either involve the entire gastrointestinal tract or be limited only to certain parts depending on the underlying disease. [Table 1](#) lists various causes of neuromuscular disease of the gastrointestinal tract that can cause gastrointestinal dysmotility by extrinsic denervation to certain parts of the gastrointestinal tract.

## **TABLE 1**

### **CAUSES OF NEUROMUSCULAR DISEASE OF THE GASTROINTESTINAL TRACT**

#### **A. SMOOTH MUSCLE DISEASE**

1. Familial Visceral Myopathies
  - a) Type I
  - b) Type II
  - c) Type III
2. Infantile and Childhood Visceral Myopathy
3. Connective Tissue Disease
  - a) Scleroderma
  - b) Dermatomyositis
  - c) Systemic lupus erythematosus
  - d) Mixed connective tissue disease
4. Muscular dystrophy
  - a) Myotonic dystrophy
  - b) Duchenne's muscular dystrophy
5. Amyloidosis
6. Metabolic Disease
  - a) Hyperthyroidism
  - b) Hypothyroidism
  - c) Hyperparathyroidism
  - d) Hypoparathyroidism

#### **B. ENTERIC NERVE DISEASE**

1. Familial Visceral Neuropathies
  - a) Autosomal dominant types
  - b) Autosomal recessive types
2. Congenital Disorders
  - a) Aganglionosis-Hirschsprung's disease
  - b) Hypoganglionosis
  - c) Hyperganglionosis-neurofibromatosis
3. Infectious Visceral Neuropathy
  - a) Parasitic-Chagas' disease
  - b) Viral-Poliomyelitis
4. Drug or Chemical Induced Visceral Neuropathy
5. Paraneoplastic Visceral Neuropathy
6. Parkinson's Disease

#### C. EXTRINSIC NERVE DISEASE

1. Spinal cord Injury
2. Multiple Sclerosis
3. Poliomyelitis
4. Diabetes Mellitus

Results from our survey suggest that PPS may also cause damage to the intrinsic nerve (myenteric plexus) of the gastrointestinal tract. Future physiologic and pathologic studies of the gastrointestinal tract in PPS will elucidate these abnormalities.

Symptoms produced by gastrointestinal dysmotility are variable depending on the part of the gastrointestinal tract involved. [Table 2](#) lists symptoms produced by dysmotility of certain parts of the gastrointestinal tract. We can see that there are a variety of symptoms that can be caused by gastrointestinal dysmotility. Thus, patients with the same disease may have different complaints that may appear to be unrelated. Patients with PPS may have difficulty with swallowing and constipation, and both symptoms can be caused by PPS.

TABLE 2

#### **SYMPTOMS PRODUCED BY DYSMOTILITY OF VARIOUS PARTS OF THE GASTROINTESTINAL TRACT**

	ORGANS	SYMPTOMS
1.	Oropharynx	Difficulty initiating swallows, food pooling in the pharynx, choking when swallowing, aspiration and aspirated pneumonia in severe cases.

2.	Esophagus	Difficulty swallowing (dysphagia), food sticking in the mid-sternum area, occasional substernal pain with swallows (odynophagia).
3.	Stomach	Nausea, vomiting, abdominal fullness long after meal, recurrent symptoms of stomach outlet obstruction (gastroparesis).
4.	Small intestine	Abdominal pain and bloating after meal, nausea, vomiting, diarrhea, recurrent symptoms of small bowel obstruction in severe cases (intestinal pseudoobstruction).
5.	Colon	Constipation, abdominal pain and bloating, recurrent symptoms of colonic obstruction in severe cases (colonic pseudoobstruction).
6.	Anus	Constipation.

## GASTROINTESTINAL INVOLVEMENT IN PPS

There are only a few studies of gastrointestinal involvement in polio survivors, and all are limited to oropharyngeal dysphagia. Gastrointestinal involvement has been virtually neglected by physicians who take care of PPS patients. We surveyed the nature and incidence of gastrointestinal symptoms in PPS patients by sending out questionnaires to 3,000 PPS patients. [Table 3](#) lists the symptoms that we analyzed from the first 754 PPS patients who responded to our survey.

TABLE 3

## GASTROINTESTINAL SYMPTOMS OF 754 PATIENTS

INCIDENCE PER CENT	SYMPTOMS	ORGANS INVOLVED
24	Difficulty Initiating Swallow	Oropharynx
32	Choking with Swallowing	Oropharynx
32	Difficulty Swallowing (dysphagia)	Esophagus
51	Heartburn	Esophagus
28	Nausea	Stomach, Small Intestine
12	Vomiting	Stomach, Small Intestine
53	Abdominal Bloating	Stomach, Small Intestine, Colon
40	Abdominal Pain	Small Intestine, Colon
32	Diarrhea	Small Intestine, Colon
48	Constipation	Colon, Anorectum
0.7	Intestinal pseudoobstruction	Small Intestine

The results from our survey suggested that gastrointestinal symptoms are common and extensive in PPS patients. These gastrointestinal symptoms may be caused by extensive gastrointestinal

dysmotility from the oropharynx to the colon. To understand the pathophysiology and pathology, we need to study the motility and histology of the esophagus, stomach, small intestine and the colon of PPS patients. Full thickness biopsies of bowel wall from surgical or autopsy specimens will enable us to examine the neuromuscular apparatus of the gastrointestinal tract in these patients.

## DIAGNOSTIC WORK-UPS FOR GASTROINTESTINAL SYMPTOMS IN PPS PATIENTS

Since our survey showed extensive involvement of the gastrointestinal symptoms, the diagnostic work-ups must be directed to the patients' complaints (see [Table 3](#)). [Table 4](#) lists the work-ups for various parts of the gastrointestinal tract.

TABLE 4

## DIAGNOSTIC WORK-UPS FOR GASTROINTESTINAL ORGANS IN PPS PATIENTS

ORGANS	DIAGNOSTIC TESTS			
Oropharynx	(1) Video esophagram,	(2) Upper esophageal sphincter manometry.		
Esophagus	(1) Barium esophagram,	(2) Esophageal manometry.		
Stomach	(1) Upper gastrointestinal x-rays,	(2) Radionuclide gastric emptying study.		
Small intestine	(1) Plain abdominal x-rays,	(2) Small bowel x-rays,	(3) Small intestinal manometry.	
Colon	(1) Plain abdominal x-rays,	(2) Barium enema,	(3) Radio-opaque markers study,	(4) Colonic manometry.
Anus	(1) Anorectal manometric study.			

If the complaints are oropharyngeal symptoms, video or cine-esophagram is useful to evaluate the swallowing mechanism of the patients. The abnormalities include unilateral bolus transport through the pharynx, pooling in the valleculae or pyriform sinuses, delayed pharyngeal contraction, and impaired tongue movements. Upper esophageal sphincter manometry may show incoordination between upper esophageal sphincter relaxation and pharyngeal contraction during swallowing.

Work-up for esophageal dysmotility includes barium esophagram and esophageal manometric study. Barium esophagram will rule out esophageal obstruction as a cause of dysphagia, and it will enable us to evaluate the diameter of the esophagus, gastroesophageal reflux and contractile activities of the esophagus. Esophageal manometric study will detect any motility disorder of the esophagus. The standard esophageal manometric technique uses low compliance water perfusion system through an esophageal manometric tube.

To evaluate gastric dysmotility, we obtain upper gastrointestinal (UGI) x-rays and radionuclide gastric emptying study. UGI x-rays will enable us to detect any ulcer or outlet obstruction that may

mimic gastric dysmotility. Radionuclide gastric emptying study using technetium-labelled scrambled egg will quantitate the emptying function of the stomach. In normal subjects, half of the solid contents will empty from the stomach in 45-90 minutes. If there is a delay in gastric emptying, it will take longer than 90 minutes for half of the solid contents to empty from the stomach. Radionuclide gastric emptying study is an excellent objective method to measure the stomach emptying function.

Abdominal bloating and distention may develop in patients with intestinal and colonic dysmotility. Plain abdominal x-rays are useful to detect gaseous distention of the dysfunctional organs. The patients may have ileus of the small intestine or colon due to severe dysmotility causing symptoms and signs of bowel obstruction without any evidence of mechanical obstruction. (It is called intestinal pseudoobstruction or colonic pseudoobstruction depending on the organ involved.)

Small intestinal dysmotility can be evaluated by small bowel x-rays and small intestinal manometric study. Small bowel x-rays enable us to rule out organic disease, to evaluate the size and contractility of the small bowel, and to measure the barium transit time of the small bowel. Small intestinal manometric study will detect any abnormal motility of the small intestine. During fasting, the small intestine has a unique pattern of motility called migrating motor complex (MMC).

MMC has three phases. Phase 1 is a quiescent period lasting for 10-15 minutes. It is followed by phase 2 which shows intermittent contractions that last for 60-90 minutes. The contractions will intensify with time and will reach phase 3 when the small intestine contracts intensely at approximately 11-13 times per minute. Phase 3 lasts for 5-8 minutes, and the small intestine becomes quiescent again, i.e., it returns to phase 1.

This cyclical motor activity will go on until the subjects are fed. After feeding, the subjects will have fed activity which resembles active phase 2. The fed activity does not have any cyclical motor activities. Fed activity usually lasts for 4-5 hours and the small intestine will return to fast pattern after that. Any small intestinal dysmotility will cause alteration of the above activities. Small intestinal manometry is very sensitive to detect abnormality in patients with small bowel dysmotility.

To evaluate colonic dysmotility, we use barium enema, radio-opaque markers study and colonic manometric study. Barium enema will enable us to rule out any organic lesion and to evaluate the size and length of the colon. Radio-opaque markers study will determine if colonic dysmotility is caused by colonic inertia or functional colonic outlet obstruction. Colonic manometric study will detect colonic dysmotility.

Function of the anorectal area can be evaluated by anorectal manometry. In normal subjects, the internal anal sphincter relaxes when the rectum is distended. If the patients lose this reflex, they will have constipation.

As we can see from the above discussion, there are specific diagnostic work-ups for certain parts of the gastrointestinal tract. A careful history will direct us to study the appropriate organs. From the

survey, it appears that gastrointestinal dysmotility in PPS patients can affect the entire gastrointestinal tract. Therefore, taking a good history from the patients is important to direct us to give appropriate diagnostic work-ups.

## **TREATMENT**

Treatment must be directed to the patients' problems. We will discuss the treatment of dysmotility of each organ separately.

### **(1) oropharyngeal dysmotility.**

The most serious complication of oropharyngeal dysmotility is aspiration during swallowing. The treatment must focus on the prevention of aspiration that may lead to aspiration pneumonia. Pharmacologic treatment has not been very effective. In patients who are less symptomatic, taking the time to eat or drink will reduce the incidence and severity of choking and aspiration. Dilatation of the cricopharyngeus muscle with bougies can be tried in more symptomatic patients. However, the effectiveness of the dilatation is not proven. It might be worthwhile to try before embarking on more invasive therapy.

In patients who have severe aspiration and recurrent aspiration pneumonia, cricopharyngeal myotomy should be considered. The technique involves sectioning the fibers of the cricopharyngeus down to the submucosa; and it can even be done under local anesthesia in poor-risk patients. Two-thirds of the patients operated upon have been relieved of dysphagia. There are a small number of patients who develop massive aspiration after cricopharyngeal myotomy. We strongly believe that any patients who undergo cricopharyngeal myotomy require esophageal manometric study and gastric emptying study.

Esophageal manometric study will allow us to evaluate the competency of the lower esophageal sphincter; the competency will help to prevent gastroesophageal reflux. Gastric emptying study will allow us to detect any delay in gastric emptying that may promote gastroesophageal reflux and aspiration. Abnormality of any of these two tests may exclude the patients from having cricopharyngeal myotomy.

### **(2) Esophageal dysmotility.**

The treatment for dysphagia due to abnormal motility has not been effective. Calcium channel blockers (such as nifedipine), nitroglycerine and balloon dilatation of the lower esophageal sphincter may be useful in some patients with diffuse esophageal spasm due to intrinsic nerve dysfunction of the esophagus. At least half of the patients with diffuse esophageal spasm do not respond to the above regimens.

In patients with gastroesophageal reflux and heartburn, antireflux regimen must be used to prevent complications of reflux esophagitis and stricture formation. We give a dose of H<sub>2</sub> receptor antagonists such as Tagamet, Zantac, or Pepcid before bedtime, and elevate the head of the bed

approximately 6 inches.

### **(3) Gastric dysmotility.**

Delayed gastric emptying usually occurs with gastric dysmotility. Metoclopramide has been used to treat this problem with variable results. A liquid and low fat meal empties faster from the stomach than a solid meal. Therefore, we advise the patients to take a low fat and liquid meal. Feeding formula such as Ensure, Vivonex, Isocal and so on can be used to supplement the calories.

A new prokinetic drug, cisapride, is being studied for delayed gastric emptying. Early results are encouraging. When this drug is available in the market, it will be useful for this group of patients.

### **(4) Small intestinal dysmotility.**

Small intestinal dysmotility may cause postprandial (after eating) abdominal pain and bloating. In severe cases, the patients may have intestinal obstructive symptoms and signs. There has been no effective prokinetic drug available yet for this problem. An ideal prokinetic drug should either enhance the contraction of a damaged muscle or normalize the coordination of a damaged myenteric plexus. No drug has, as yet, lived up to this expectation.

The symptoms in most patients occur intermittently, and only a small number of patients may have persistent symptoms. Since the symptoms are directly related to meals, manipulating the amount, the nature and the frequency of meals may help some patients. The patients should consume 25 calories/kg of ideal body weight per day divided into 3-4 equal amounts. Half of the calories may come from feeding formulas. The patients must avoid carbonated beverages to prevent adding excessive gas in the digestive tract.

Occasionally, nasogastric suction and intravenous fluid are needed when the patients have intestinal obstructive symptoms. When the obstructive symptoms are persistent or occur several times a week, a long term parenteral nutrition is the only treatment that will improve the patient's symptoms and nutritional status.

### **(5) Colonic dysmotility.**

The most common complaint is chronic constipation. The patients must avoid using stimulant laxatives such as Senokot, Dulcolax, etc., on a long term basis because most of the stimulant laxatives can damage the myenteric plexus when using them long term. This effect will further compound the difficulty that the patient already has.

We usually try the patients on high fiber diets or fiber compounds such as Fibercon or Metamucil. More than half of the patients will respond to these regimens. If the patients do not respond to fiber, we next try to use saline laxatives such as milk of magnesia. We titrate the dose of milk of magnesia to obtain one bowel movement a day. In early stages, it is not uncommon to use milk of magnesia up to two ounces per day. Later on, we are usually able to maintain the patients on one tablespoon daily. Tap water enema can be used if the patients have no bowel movement for three



days while on fiber or milk of magnesia.

In patients with obstipation despite adequate dose of fibers or milk of magnesia, an ileostomy or subtotal colectomy with ileoproctostomy may be required to take care of the problem. We encounter very few patients with colonic dysmotility with severe obstipation that require such a drastic approach.

**(6) Anorectal dysfunction.**

At the present time, we are not certain if PPS patients have any anorectal dysfunction. Anorectal manometric study in these patients will enable us to better understand the anorectal function in this group of patients. The medical treatment of anorectal dysfunction is disappointing. Surgical treatment such as rectal myectomy may be beneficial to some patients with anorectal dysfunction.

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