

# Constipation: How does it exist?

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## Abstract

**Background:** Functional defecation disorders are common and affect approximately 50% of patients with chronic constipation. The etiology of functional defecation disorders is not well known, but several pathophysiologic mechanisms have been described, including failure of rectoanal coordination, paradoxical anal contraction or insufficient relaxation of anal sphincter during defecation and impairment of rectal sensation as well as secondary slowing of colonic transit. Aim of this manuscript is to review the pathogenesis and surgical treatment of constipation.

**Method:** An extensive Medline search, textbooks, scientific reports and scientific journals are the data sources. We also reviewed reference lists in all articles retrieved in the search as well as those of major texts regarding pathogenesis and surgical treatment of constipation.

**Results:** There are many studies about the pathogenesis and pathophysiology of constipation. Also there are many medical and surgical treatment strategies were developed.

**Conclusion:** The patients with constipation symptom must be evaluated carefully, and medical treatment must be tried after differential diagnosis is made. Only in patients that do not respond to the medical, surgical treatment can be considered.

## INTRODUCTION

Functional defecation disorders are common and affect approximately 50% of patients with chronic constipation. The etiology of functional defecation disorders is not well known, but several pathophysiologic mechanisms have been described, including failure of rectoanal coordination, paradoxical anal contraction or insufficient relaxation of anal sphincter during defecation and impairment of rectal sensation as well as secondary slowing of colonic transit. Symptoms alone are inadequate to distinguish patients with defecation disorders from those with other types of constipation. Detailed clinical evaluation and anorectal physiologic tests are required for definitive diagnosis.

Constipation is defined as infrequent defecation with hard consistency that can be expelled by force, and is the symptom of many diseases. Constipation is a symptom complex rather than a disease itself. According to the internationally accepted ROME III definition (Table.1), criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis is defined as "constipation" (1,2,3).

Table 1: Diagnostic criteria for functional constipation (Rome III)

1. A- Must include two or more of the following:
  - a. Straining during at least 25% of defecations,
  - b. Lumpy or hard stools in at least 25% of defecations,
  - c. Sensation of incomplete evacuation in at least 25% of defecations,
  - d. Sensation of anorectal obstruction / blockage in at least 2% of defecations,
  - e. Manual maneuvers to facilitate in at least 25% of defecations (e.g. digital evacuation, support of the pelvic floor),
  - f. Fewer than three defecations per week.
3. B- Loose stools are rarely present without the use of laxatives.

4. C- There are insufficient criteria for irritable bowel syndrome.

Among those parameters, number of defecation is the easiest one to detect, whereas others are relatively subjective. By this reason we can define constipation practically as "defecation 2 times or less in a week with extensive effort". In fact, we can define constipation in respect to the shape of feces.

This definition needs to be identified from Irritable Bowel Disease but this is not easy since both can be manifested as overlapped together. Nevertheless, there is little relation between Rome criteria for functional constipation and physiological measurements of slow colon passage.

Many patients present with other symptoms rather than constipation. Abdominal pain, swelling, distention, nausea vomiting after meals, digital manipulations for defecation are common symptoms (4).

#### Epidemiology

Constipation is seen in young and middle aged adults with a 3:1 ratio of women to men, whereas with same distribution older ages. In western societies 95% of adult population has bowel movements ranging between once/two days to 2 times / day (5).

Defecation frequency of 3 times/week or less in 4%, 2 times or less/week in 1-2% of population is observed. It is estimated that the constipation prevalence in U.S.A is 2-34% (6).

The differences in the prevalences expressed in various epidemiologic studies are thought to be based on the differences and discordances in the definition of constipation. Drossman et al. found the prevalence of functional constipation as 3.6%. Prevalence of pelvic outlet obstruction was found as 11% at another population based study. A female predominance was observed at that study. Irritable bowel disease was observed at 17% of the same population (7).

In a prospective study, it is showed that, 5% of 200 female patients developed constipation after hysterectomy. This shows that secondary constipation may develop after surgery. Prevalence of any form of constipation is 40% among the elder people in same population, most of whom are either hospitalized or nursed at home. Pelvic exit symptoms in the same population are %24. In a small cohort

study performed in elder constipated patients, pelvic exit delay is observed more frequently than slow passage constipation (8).

## PATHOPHYSIOLOGY INNERVATION

Motor and sensory functions of colon are regulated by nervous system of bowel and include extrinsic and intrinsic innervation. Intrinsic nervous system consists of myenteric, submucosal and mucosal nerve plexi. Myenteric plexus regulates the smooth muscle functions that are responsible for motility whereas, submucosal plexus is responsible of ion transport and absorption. Extrinsic nervous system is composed of parasympathetic and sympathetic autonomic innervations. While right colon innervation is carried out by parasympathetic nerves that are carried by vagus nerve, transverse colon, left colon and rectum innervation is accomplished by sacral parasympathetic nerves (S<sub>2</sub>-S<sub>4</sub>) that enter from distal colon. Sympathetic nerves reach the bowel along with superior and inferior mesenteric arteries and result in tonic sympathetic effects (9).

They usually express excitatory properties for sphincter muscles and inhibitory for non-sphincterics. Sympathic system affects the bowel by  $\alpha 2$  receptors. Clonidine –a  $\alpha 2$  receptor agonist- increases the compliance of the colon by decreasing its tonus. Yohimbine - a  $\alpha 2$  receptor antagonist- has a reverse effect to clonidine. The  $\alpha 1$  and  $\beta$  receptors have no effects to the colonic tonus (10). The functions of enteric and extrinsic neurons are regulated by some neurotransmitters that are released from between the neurons. There include noradrenalin, acetylcholine, opioids, serotonin, somatostatin, CCK, substance-P, VIP, and neuropeptide-Y (11). It has been shown that, while cholecystokinin stimulates pancreatic secretions maximally, it has no effect on colonic tonus or phasic activity (11).

Atropine decreases the colonic tonus (12). There are five subtypes of serotonin receptors. These have important effects on the regulation of motor activity and sensory innervation of colon. 5-HT<sub>3</sub> antagonists (e.g., ondansetron) delay the oro-caecal and colonic passage along with inhibiting colonic tonus and compliance. 5-HT<sub>4</sub> receptor agonists stimulate acetylcholine secretion, thus display prokinetic activity (13). A somatostatin analogue octreotide decreases the postprandial colonic tonus, particularly decreasing motility in descending colon and rectum in a fashion that is paradoxical to the gastrocolic reflex (14). Several neuropeptides show abnormal segmental distribution

in the colons that are resected in constipated patients (15).

### EXTRINSIC NEUROPATHY

Injury of autonomic nerves after trauma may progress to infections, degeneration of neurons and neuropathies, disturbances in secretory motor and sensory functions and usually results in constipation. In the patients that have spinal cord injury above the sacral segment, proximal and distal colonic transport time increases due to loss of control. Colonic motility rate and tonus are normal and the response to feeding is little or none (16).

Spinal cord lesions that include sacral segments or injury in the efferent nerves that emerge from these segments result in the loss of rectosigmoidal neural innervation and anal sphincteric control. In the patients with such lesions, left colon loses its constrictor ability, rectal tonus and sensitivity decreases and those results in colonic dilatation and fecal impaction (16). Parkinson's disease and multiple sclerosis are often associated with constipation. There is a decrease in the number of dopamine containing neurons in Parkinson's disease. Lewy bodies have been found in the neurons of myenteric plexus (17). In addition to that, there is a relaxation defect in the skeletal muscles of pelvic floor. As shown in a study, constipation which is the most common symptom of the disease is seen in 43% of patients of multiple sclerosis (18). It has been proposed that, the slow colonic transit time and lack of postprandial motor response seen in the colon of multiple sclerosis patients result from the disturbance in parasympathetic control of colon in spinal cord injuries. There is constipation secondary to autonomic dysfunction in diabetes mellitus (DM). In a large group that consists of diabetic patients, constipation rate in patients with diabetic neuropathy is 22% whereas; the rate is 9.2% in the group of diabetic patients without neuropathy. However, comparison with the control group fails to show a big difference (19). In a study carried out by Feldman et al, constipation is seen more frequently in insulin dependent DM when compared to insulin independent DM. Altomare et al performed a study in 23 patients that have low transit rate without obstruction and irritable bowel syndrome and it shows sympathetic disorders in postganglionic tests in 70% of constipated patients and 8% of control group (20).

### ENTERIC NEUROPATHY

Hirschsprung's disease is a disorder characterized as the lack of ganglions in myenteric and submucosal plexi. First symptoms are identified on birth and rarely on newborn or early childhood. Agangliosis start from anal verge and

expand proximally. Hirschsprung's disease is documented in deficiency of nerve growth factor in colon muscle layer. Idiopathic megacolon and megarectum can be congenital or acquired. However the role of the defect in enteric nervous system is insignificant. In megacolon, dilated segment and normal phasic contractions are present but colonic tonus is decreased (21). In a study Gattuso et al conducted on 30 patients with megacolon or megarectum; longitudinal, circular and muscularis mucosa fibrosis and significant dilatation in megarectum histological specimens due to smooth muscle hypertrophy is documented. Even a decrease in neural tissue density in megarectum specimens is present but these are not observed in idiopathic megacolon (22).

Chagas disease is caused by *Trypanozoma cruzi*, an agent that destructs the enteric plexus with infection. The disease is characteristically presents with constipation. Krishnonurty et al showed a decrease in number, and a disorder in structure of argiophilic neurons, found in the resected colon material of constipated patients (23).

Schouteen et al, who study the neuropathogenic changes in patients with idiopathic low passage constipation, documented the decrease or complete absence of neurofilaments in myenteric plexus. Destructive effects of laxatives on enteric neurons are not clearly understood. In a study of Milner et al, it has been shown that, no changes occur in colonic structure and neuropeptide levels with chronic use of laxatives (24). Another study conducted documented that, no increase in the argiophilic dye absorption occurs, although argiophilic neurons decrease (25). Recent studies focus on Cajal cells, an interstitial cell type. It's been assumed that the Cajal cells play an important role in regulation of intrinsic electrical activity and electromechanic connections in bowel. The studies show that the distribution of Cajal cells in different colonic localizations and the volume in the myenteric plexus of resected colon decrease in idiopathic slow transit constipation (26).

### COLONIC MOTILITY

Normal passage time from mouth to caecum is approximately 6 hours. Regional passage time with radioopaque substance is approximately 12 hours each for right colon, left colon and sigmoid colon. In general, colonic transit time is 36 hours (13). Bowel transit time is affected by several factors. Previously it has been told that, ileocolic junction regulates the bowel transit time. However, the studies conducted on patients with right hemicolectomy

showed that, despite the loss of ileocolic sphincter, there is no significant change in bowel passage time. Diet, calorie intake, water influx in colon, distal obstruction affects colonic transit time. As the fiber content of diet increases, colonic transit time decreases, defecation frequency increases and the feces gains a softer nature. When calorie intake is decreased, colonic transit slows down, but is back to normal once normal calorie intake is established (27).

There are three types of contraction pattern were described. These are segmentation, driving movements and mass movements. Segmental activities are formed by the contraction of isolated circular smooth muscle fragments. This kind of activity is not driving. The result of segmentation is an obstacle for the luminal content trying to move to distal segments. Increase in segmentation results in constipation. Colonic segmentation increases as a response to meals and decreases in sleep. Segmental activities are decreased by atropine, 5-HT<sub>3</sub>, PGE<sub>2</sub> and bradikinin. Drugs like Prostaglandin and cholecystokinin increase the segmental activity (10). There is no response to the intravenous infusion of *in vivo* PGF<sub>2</sub>, but PGE<sub>2</sub> inhibits the segmental activity (28).

Documentation of driving activity in colon is harder than in esophagus and small intestine. Such activities are formed by the systematic contractions of circular muscles. This occurs in small intervals in colon. On the contrary, mass movements are high amplitude contractions that are not frequent. These movements push the luminal content to the distal and are related to defecation. Recent studies show a decrease in the frequency and number of high amplitude waves in patients with slow passage constipation, idiopathic constipation and normal passage constipation (29). However, absence of postprandial early motor response is documented in patients with slow passage (30).

Fluid balance affects GIS transit. Approximately 9 liters of fluid reaches the bowel with oral intake and endogenous secretions. Small intestines transport approximately 1.5 liters of fluid to colon, most of it is reabsorbed there and maximally 200 ml of water is expelled with feces. Preston et al documented the increase in motilin, pancreatic polypeptide, and gastrin secretions in response to water injection in constipated patients. The motility increase associated with this forms the basis of antegrade enema technique (31). Sjolund et al showed the decrease in basal and postprandial motilin levels (32).

In addition this, van der Sijp et al documented that, there is no change in the levels of neurotensin, motilin, gastrin,

pancreatic polypeptide secreted from upper GIS to circulation and fluctuations in somatostatin levels (33). Immunoreactive serotonin receptor levels are found in lower concentrations in circular muscles and muscularis mucosa of patients with colonic inertia. There is positive relationship between serotonin receptor levels in circular muscle and colonic transit time (12).

### ETIOLOGY

Various factors cause constipation in adults and in children. There may be pain due pelvic floor tension during defecation. Usually this condition is thought to be the predisposing factor of childhood. The reason of this is the common belief of patients that is, "Defecation must be done every day". Some of these patients consider feces as toxic and try to defecate whenever possible. This is such a fear that, the patients think something will rupture, constipation will develop or other dangerous effects will emerge if they fail to eliminate the daily fecal content. Due to this reason, most of the patients start to use laxatives and enemas and become addicted to them. Again according to the explanations, one or two of the family of such patients engage in arguments about constipation and such arguments usually take place very severely. Again some of these patients believe that if they don't use laxatives they will never be able to defecate. Despite those false beliefs about bowel functions, most patients adapt to this reduced defecation habit. Those patients have rigid programs and they use a laxative or enema every morning. They are free in their programs and they don't want to deal with bowel functions to spend a comfortable day.

Elder people usually get constipated easily since they don't have a defecation habits. Some feel disgusted to talk about bowel functions. Sexual or emotional trauma history may accompany this condition. Frequent stress and general tension can reach to pelvic floor tension and the patients can suffer from this stress. It's quite important that the patients act as if there is no cure for their disease. If the constipation of the patient last long, they start to exert force to defecate. Although this helps to empty the rectum at first, the event contributes to the development of paradoxical pelvic contraction. If the exertion of force is increased, pelvic floor muscles are involved too (34). The causes of constipation are given in Table.2.

**Figure 1**

**Table 2: Causes of Constipation**

<b>ENDOCRINOLOGICAL</b>	<b>NEUROLOGICAL</b>
Diabetes Mellitus	Parkinson's Disease
Hypopituitarism	Brain Tumor
Hypothyroidism	Multiple Sclerosis
Pseudohypoparathyroidism	Scleroderma
Hypercalcemia	Spinal Cord Injuries
Pheochromocytoma	Spinal Cord Tumors
Glucagonoma	
Pregnancy	
<b>METABOLIC</b>	<b>PSYCHIATRIC</b>
Uremia	Depression
Hypokalemia	Psychosis
Porphyria	Anorexia Nervosa
Amloidosis	Obsessive Compulsive Disorders
Dehydration	
<b>OPERATIONS</b>	<b>ORGANIC DISEASES</b>
Pelvic Operations	Organic Obstructive Bowel Diseases
(Hysterectomy, Retropexy, Cystectomy)	(Tumors, Brid, Strangulation of Hernia
Anal Operations	Volvulus, Endometriosis, Invagination etc.)
(Anal stenosis due to hemorrhoidectomy etc.)	
Narrowing due to Anastomose	
<b>DIET</b>	<b>CHANGES IN LIFE STYLE</b>
Inadequate intake of fiber	Vacation, immobility,
Inadequate consumption of fluid	Events that affect daily life
<b>FUNCTIONAL DISEASES</b>	<b>PELVIC EXIT OBSTRUCTION</b>
Functional Obstructive Bowel Diseases	Rectal Prolapsus
Congenital Aganglionosis	Rectocele
Acquired Aganglionosis (Chagas Disease)	Anal Stenosis
Pseudoobstruction (Ogilvie Syndrome)	Rectal Intusseption
Megacolon	Paradoxical Contraction of Puborectal
	Muscle
Delay in Colon Transit Time	Hypertonus of Internal Sphincter
Irritable Bowel Disease	Perineal Looseness
	Megarectum

It has been found that bacterial flora and intraluminal pH show variance in constipated patients. The increase in colonic flora and ability to digest fibers results in smaller feces in some patients. On the other hand, these bacteria ferment the polysaccharides, fatty acids and bile acids that are not absorbed and some of the resulting metabolites act as intraluminal mediators and cause strong peristaltic

contractions and rapid movement of intraluminal content (35).

Constipation is seen in women, especially in reproductive period, on time of puberty. Davies et al documented that transit time decreases and fecal content hardens in luteal period (36). Other studies, however, showed otherwise (37,38).

### CLASSIFICATION OF CONSTIPATION

- **Simple Constipation:** Average Frequency or hard passage of feces. Symptoms are intermittent and minimally disturbing. Response to conservative treatment (intake of fiber) is good (39).
- **Secondary Constipation:** It occurs secondarily to local or systemic diseases. For example, during the course of mechanical obstruction, adverse drug effects (narcotic analgesics, NSAIDs, some chemotherapeutics, calcium channel blockers, antiarrhythmics), hypothyroidism, hypercalcemia, some neurological diseases (multiple sclerosis, Parkinson's Disease), some psychiatric disorders (depression, anorexia nervosa)
- **Chronic Idiopathic Constipation (CIC):** It consists of colonic inertia and pelvic exit obstructions that are not related with any organic cause (paradoxical contraction of puborectal muscle). 4 main pathologies are observed in CIC:
  - Colonic inertia (retardation of colon passage)
  - Pelvic exit obstructions
  - Colonic inertia + pelvic exit obstructions
  - Irritable bowel disease

### COLONIC INERTIA

Colon contents leave the colon in at most 72 hours in normal population (40). Prolongation of this period shows retardation in colon passage. This condition is defined as colonic inertia and actually occurs as a result of autonomic nerve pathologies (degenerations of Auerbach and Meissner plexi appear in gastrin, motilin, pancreatic polypeptide, glucagons and other peptide deficiencies). Colonic inertia can be segmental or diffuse. Involvement of other hollow organs is defined so, any surgical treatment will be useless in those patients (41). It has been proposed that genetic factors (Ret oncogene) may be involved in development of colonic inertia (42). Megacolon (diameter of colon exceeds 8 cm in

ascending colon; 6 cm in descending colon and haustrations are lost) causes CIC.

### PELVIC EXIT OBSTRUCTION (PEO)

PEO is encountered under different names in literature: Puborectal syndrome, spastic pelvic floor syndrome, anismus, nonrelaxing puborectal muscle syndrome, rectoanal dysnergy, immotile perineum, paradoxical puborectal contraction, pelvic floor dysnergy. In normal defecation, pelvic floor muscles, puborectal muscle and sphincters relax simultaneously. However in PEO, puborectal muscle and external sphincter exhibit disorganized contractions instead of relaxation by some reason and prevent defecation (43).

Due to this contraction of puborectal muscle, the anorectal angle (the angle that is between the line drawn through the center of anal canal and lines drawn from the posterior wall of rectum or according to another definition; the angle that is between the line drawn through the center of anal canal and lines drawn from the middle of inferior rectum) that should have widened cannot widen (44). Since anorectal line is not straightened enough and anorectal ring cannot relax, contents of rectum cannot be expelled to the anal canal. Addition to this, the conditions like internal sphincter being hypertonic, rectum wall contraction disorder and reduction in rectal awareness may prevent defecation (45).

Perineal Looseness can cause PEO too. Some claim that, exerting too much force or vaginal labor traumas may cause pudendal nerve injury that may result in perineal looseness (46).

Sigmoidocele is the descent of sigmoid colon to Douglas pouch in patient with deep pouches. It's a defecographic finding rather than a clinical finding.

Rectal Intusseption is another defecographic finding that can cause PEO. It's the overlapping of entire rectum wall layers from proximal to distal (43). Some researchers accept the overlapping of rectum mucosa for more than 3 mm as rectal intusseption (47). In a study conducted in 209 patients that have PEO, defecography showed 23% Rectocele, 10% rectal intusseption, 8% Sigmoidocele, 63% perineal looseness (48). In addition to this, diseases like rectal prolepses, rectocele, and anal stenosis may cause PEO too.

### CLINICAL FINDINGS

Defecation reduction, rectal bleeding, tenesmus, abdominal distension and pain, episodic diarrhea, nausea-vomiting, and sometimes false palpation of mass due to solid feces in

sigmoid colon can be encountered in clinic. Digital examination reveals an empty rectum most of the time. Sometimes, increased tone of anal canal and puborectal muscle can be felt.

### DIAGNOSIS

It's essential to discern whether the constipation is related to a systemic disease or not. If there are symptoms that suggest a systemic disease in the history physical examination, these must be consulted with the related departments.

After that step, any organic cause related to colon and rectum must be eliminated (e.g. tumors). Appearance of constipation that is not present before in middle aged or elder patients, suggest malignancy. It should be kept in mind that, in a patient who has chronic idiopathic constipation since his youth can develop malignancy. Namely, malignancy must be eliminated in a patient with constipation. The gold standard is the endoscopic examination. In general rectosigmoidoscopy is sufficient in patients who are under 40. However, if the patient is over 40, colonoscopy must be performed first. Another benefit of Endoscopy is that it also documents the benign formations related with chronic idiopathic constipation (megacolon, rectal ulcer).

Colon examination using double contrast enema, provides valuable information about the morphological structure of colon (colon length, presence of segmental or total megacolon, conditions of haustrations). However, colon X-rays in constipated patients are usually normal.

Studies are carried out to see if chronic idiopathic constipation is caused by colonic inertia or by pelvic exit obstruction or by combination of both. For this purpose, colonic passage time, defecography, EMG, balloon extraction test, and anorectal manometric studies are used.

### COLONIC PASSAGE TIME

It shows the passage time of colonic content. The patient ingest a gelatin capsule that is filled with a definite number of radioopaque rings and opens in terminal ileum. It is based on the fact of whether it is eliminated from the body within 72 hours or not. Another method used practically is the consumption of sitz-marks (a capsule that contains 24 radioopaque substances) in one time and evaluation with a direct abdominal X-ray after 3 days. In patients whose constipation is a result of colonic inertia, at least 8 or more opaque substances must be visualized. If the 3<sup>rd</sup> day X-ray couldn't be taken, it is taken on 5<sup>th</sup> day, and in normal population, 80% of radioopaque substance must be

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eliminated (49). Another approach is the observation of the journey of radioopaque rings with X-rays taken on the same hour for 5 days consecutively and it is thought to be more sensitive despite the fact that it has more x-ray shots. In the X-rays, the radioopaque substance may be seen as diffused completely or collected in a fashion that suggests segmental obstruction. On the other way, in the patients with pelvic exit obstruction, opaque substances are not encountered in colon but gathered in the pelvis, in rectum area (40). For the patients who have prolonged colonic passage time and who are candidates of a surgical treatment, examination of upper GIS motor function prior to the surgery (by upper GIS manometry, nuclear gastric emptying test, hydrogen breath test) is essential (41).

### DEFECOGRAPHY

It is used to document pelvis exit obstruction. Since it will show the defecation procedure step by step, the causes of pelvic exit pathologies can also be detected. Anismus, rectocele, rectal intusseption, Sigmoidocele, megarectum can be shown with this test. In the defecographic findings for anismus, anorectal angle does not increase. Normally, the angle should increase more than 5% (44,48).

### ELECTROMYOGRAPHY (EMG)

It's very valuable in the diagnosis of anismus. Bipolar needle or surface electrodes are used. Electrodes are placed on posterior middle line 12 mm distal to anal verge for puborectal muscle and to laterals for external sphincters (40). After the electrodes are applied to puborectal muscle, electrical activity of the muscle is recorded during the contraction, relaxation and exertion of force periods of the patient. Normally the activity increases in contraction phase and completely decreased during exertion of force. In anismus patients, activity increases in exertion of force period. Even though EMG and defecography are not superior to each other for the diagnosis of anismus, the diagnostic value increases two folds when they are combined (44).

### BALLOON EXTRACTION TEST

The patient is put in left lateral supine position and a balloon filled with 50 -60 ml of warm water in front of a 2mm catheter is placed just above the anorectal junction by pulling. The patient is asked to extract the balloon by exerting force after a resting period of 3-4 minutes. Normal people can perform this without any difficulty while it's difficult for constipated patients (40,44). Fleshman et al found out that 9 patients out of 21 diagnosed with anismus failed to

extract the balloon. Thus, they reported that defecography has a false positive diagnostic value and balloon extraction test performed in defecation position is more specific (50).

### ANORECTAL MANOMETRY

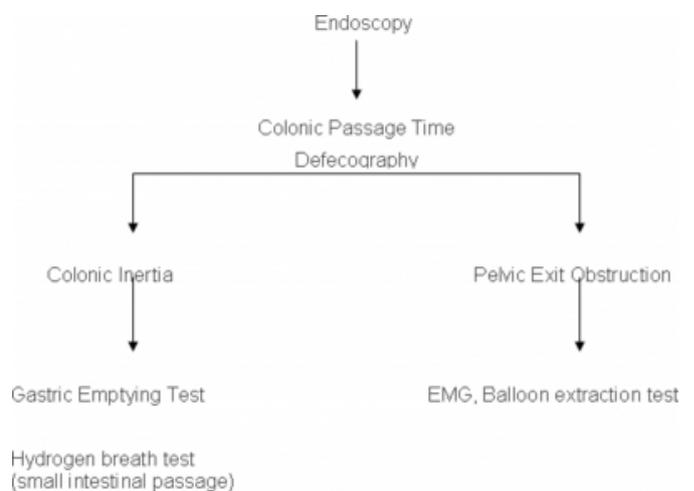
It is performed to measure the pressures formed by pelvic floor and anal sphincters. Some pressure abnormalities may be seen in CIC. Anal resting pressures may be elevated. Relaxation amplitude may be decreased or totally lost in rectoanal inhibitor reflex (RAIR). RAIR is especially lost in adult Hirschsprung's Disease and megarectum (51). Another advantage of anorectal manometry is that, it's important in the postoperative evaluation of total colectomy and iliorectal anastomoses.

Additionally there are tests like pudental nerve terminal motor permeability, rectal sensation and perinometer.

As a result, it's important to conduct these tests to expose the causes of CIC and to plan the treatment. All of the tests should be carried out as shown in the algorithm below (Figure 1).

Figure 2

Figure 1: Algorithm in the diagnosis of constipation



### TREATMENT

#### CONSERVATIVE TREATMENT

In constipation due to colonic inertia, the diets of patients are regulated. Simple laxatives and enema are used. Exercises conducted. Intake of substances like cellulose that increase the amount of fiber rich feces, fruits and vegetables, meals cooked with olive oil and large amounts of fluid is advised. The number and amplitude of colonic movements that are seen in adults in the morning and after the meals, are decreased CIC. Benzofuran group drugs are suggested to

increase such movements in these patients. (36). Additionally, osmotic agents that increase the inflow of fluid to the bowel lumen like magnesium hydroxide, polyethyleneglicol, laxatives and enemas are used (37,51).

### SURGICAL TREATMENT

In conditions where conservative treatment fails, surgery may be considered only if all the tests identify the source of CIC as colonic inertia. Careful and selective approach must be used before decision for surgery is made. If surgery is based on the anorectal physiological studies, satisfactory results can be obtained. Motility disorder in remaining parts of GIS should be clearly eliminated. Psychiatric conditions of the patients should be evaluated carefully. Patients must be informed about the surgical procedure, its complications and possible results (increase in the number of defecation, softening of fecal content, abdominal pain, incontinence). In general, defecation in every 15 days constitutes a surgical indication, although there is no definite agreement on the time period.

The most distinguished method for surgical treatment is Total colectomy and ileo-rectal anastomosis. Postoperative results show variation. Ghosh et al documented that, in 21 patients with CIC, who underwent colectomy and ileo-rectal anastomosis, presented with abdominal pain, swelling, sense of urgent defecation and forced defecation more frequently than the control group (15 patients who had total colectomy for other purposes). Additionally, 71% of the patients who had this operation experienced brid ileus in later period ( $p < 0.01$ ). However, different values are obtained in different studies. A first few cases that are operated laparoscopically in our clinic, are satisfactory.

Also, it's disturbing that; of these patients, 68% had esophageal motor dysfunction, 19% had delayed gastric emptying, 10% had increased small intestinal passage time, 54% had urodynamic abnormalities and 14% had abnormal results in autonomic function tests (52).

Piuta et al conducted a study to emphasize the importance of diagnostic tests and total colectomy. 71% of the patients had successful results, 21% had no improvement in life quality and 8% had no benefits from the treatment. Psychiatric disorders or afferent nervous system disorders have been identified in all of the patients who had no benefit from the treatment (53).

Beck et al performed abdominal total colectomy to 14 patients and reported that all the patients have satisfactory

results (54).

Rantis et al applied conservative medical treatment to 51 patients with CIC. 35% of patients had no successful results so, to a total of 12 suitable patients (24%), total colectomy (5 patients), anterior resection with rectopexy (3 patients), colostomy (2 patients), sigmoidectomy (1 patients with volvulus), abdominoperineal resection (1 patient) are performed. Among these patients 84% had successful results (55).

Gregory et al performed subtotal colectomy to 112 patients and were successful in 67%. Among these patients, 21% had incontinence, 46% had occasional diarrhea, and 4% had abdominal pain. Diarrhea and incontinence is seen more frequently in patients who had ileo-rectal anastomosis than the patients who ileo-sigmoid anastomosis. However this result is not statistically significant. Again there is no difference between anastomose level and the quality of life. The patients who have permanent ileostomies, have significantly lower scores of life quality (56).

In another study conducted by Christian et al, subtotal colectomy was performed to 12 patients and success was achieved in 10 of them. One of the patients, who had unsuccessful results, later developed narcotic dependency. The other had normal preoperative defecography and high rectum compliance and because of this, he underwent proctectomy and ileo-anal pouch anastomosis later and the operation was satisfactory. 3 patients had abdominal pain, 2 patients had diarrhea and 1 patient had incontinence as complication (57).

Platell et al performed either total colectomy and ileo-rectal anastomosis or subtotal colectomy and caeco-rectal anastomosis to 96 patients. Postoperatively, 3.1% developed anastomose leak, 11.5% had pelvic abscess with 2.1% mortality rate. 81.6% of patients had symptomatic improvement, 51.2% had exertion difficulties, 50.65 had anal incontinence, 55.2% had abdominal pain, and 75.9% had abdominal swelling complaints. 35.6% of the patients later developed brid ileus and operated again. 9.2% of them had developed ileostomy requirement (58).

You et al performed different types of segmental colectomies to 10 patients. Improvement was observed in all the patients in the postoperative 3 month period and 3 patients developed the constipation symptoms again after 2 years. Subtotal colectomy + ileo-rectal anastomose is performed to those patients and they were improved (58).

This study had showed the sufficiency of segmental colectomy.

Rivera et al opened percutaneous caecostomy to 12 pediatric patients with anorectal abnormalities and spina bifida, and achieved successful palliation (59).

Pinho et al performed anorectal myectomy to 63 patients. Spontaneous rectal emptying is achieved only in 11 patients and 44 had failed. Incontinence was observed in 6 patients (60).

Eccersley et al performed irrigation to 11 patients from sigmoidostomy thru a canal. Also, irrigation is performed to 10 patients from transverse colon thru a canal. Abdominal swelling was observed in all the patients in transverse colon group but successful emptying was achieved. 3 patients from sigmoid group had benefit (61).

Additionally patients who are cured with complete laparoscopic total colectomy + ileo-rectal anastomosis are reported (62,63).

### BIOFEEDBACK THERAPY

It's a physiotherapeutic method used to regain the function of defecation and muscles of anorectal region in constipated patients with diagnosis of pelvic exit obstruction. After there patients are connected to the EMG or manometer with electrodes or perfusion catheters, they perform the contraction and relaxation maneuvers in a specific order and they relearn the working condition of the muscles via the images they obtain on the monitor. These exercises are conducted as 1-3 sessions per week for duration of 4-6 weeks (64). The patient is informed about regional anatomy, how the muscles work, and the mechanism of defecation before they start the therapy. The success rate of the treatment with such method is 31-100% (65).

### INJECTION OF BOTULINUM TOXIN

Inhibition of puborectal muscle contractions are based on paralyzing the muscle by injection of Botulinum-A toxin. It has no lasting efficiency. (66)

### SURGICAL MYOTOMY OF PUBORECTAL MUSCLE

It can be performed as partial or complete myotomy in anismus. The technique is abandoned due to postoperative failure and development of incontinence (51). Surgeons in Anatolia region may still perform this technique.

### SURGICAL MYOTOMY OF INTERNAL SPHINCTER

It was once performed in patients with pelvic exit obstruction but it is abandoned since it has no benefits.

### CONCLUSION

The patients with constipation symptom must be evaluated carefully, and medical treatment must be tried after differential diagnosis is made. Only in patients that do not respond to the medical, surgical treatment can be considered.

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### References

1. Glia A, Lindberg G, Nilsson LH, Mihocsa L, Akerlund JE. Clinical value of symptom assessment in patient with constipation Dis Colon Rectum 1999;42(11):1401-1410
2. Velio P, Bassotti G. Chronic idiopathic Constipation: Pathophysiology and Treatment. J Clin Gastroenterology 1996;22(3):190-196
3. Ozturk R, Rao SSC. Defecation disorders: An important subgroup of functional constipation, its pathophysiology, evaluation and treatment with biofeedback. Turk J Gastroenterol 2007; 18 (3): 139-149
4. Sandler RS, Drossman DA, Nathan HP, McKee DC. Symptom complaints and health are seeking behavior in subjects with bowel dysfunction. Gastroenterology 1984;87(2):314-318
5. Johanson JF, Sonnenberg A, Koch TR. Clinical Epidemiology of chronic constipation. J Clin Gastroenterol 1989;11(5):525-36
6. Kumar D, Bartolo DC, Devroede G, Kamm MA, Keighley MR, Kuipers JH, Lubowski DZ, Nicholls RJ, Pemberton JH, Read NW, et al. Symposium on constipation. Int J Colorectal Dis. 1992; 7(2):47-67.
7. Talley NJ, Weaver AL, Zinsmeister AR, Melton LJ 3rd. Functional constipation and outlet delay: a population-based study. Gastroenterology. 1993; 105(3):781-90.
8. Merkel IS, Locher J, Burgio K, Towers A, Wald A. Physiologic and psychologic characteristics of an elderly population with chronic constipation. Am J Gastroenterol. 1993; 88(11):1854-9.
9. Camilleri M, Ford MJ. Colonic sensorimotor physiology in health and its alteration in constipation and diarrhoeal disorders. Aliment Phar Therm 1998;12:287-302
10. Bharucha AE, Camilleri M, Zinsmeister AR, Hanson RB. Adrenergic modulation of human colonic motor and sensory function. Am J Physiol. 1997 Nov; 273(5 Pt 1):G997-1006.
11. El-Tawil AM. Persistence of abdominal symptoms after successful surgery for idiopathic slow transit constipation. Southern Medical Journal 2002;95;9:1042-6
12. Steadman CJ, Phillips SF, Camilleri M, Talley NJ, Haddad A, Hanson R. Control of muscle tone in the human colon. Gut. 1992 Apr; 33(4):541-6.
13. Zhao RH, Baig MK, Thaler KJ, Mack J, Abramson S, Woodhouse S, Tamir H, Wexner SD. Reduced expression of

- serotonin receptor(s) in the left colon of patients with colonic inertia. *Dis Colon Rectum*. 2003 Jan; 46(1):81-6.
14. von der Ohe MR, Camilleri M, Thomforde GM, Klee GG. Differential regional effects of octreotide on human gastrointestinal motor function. *Gut*. 1995 May; 36(5):743-8.
15. Sjolund K, Fath S, Ekman R, Hulten L, Jiborn H, Nordgren S, Sundler F. Neuropeptides in idiopathic chronic constipation (slow transit constipation). *Neurogastroenterol Motil*. 1997 Sep; 9(3):143-50.
16. Bruninga K, Camilleri M. Colonic motility and tone after spinal cord and cauda equina injury. *Am J Gastroenterol* 1997;92:891-4
17. Singaram C, Ashraf W, Gaumnitz EA, Torbey C, Sengupta A, Pfeiffer R, Quigley EM. Dopaminergic defect of enteric nervous system in Parkinson's disease patients with chronic constipation. *Lancet*. 1995 Sep 30; 346(8979):861-864.
18. Hinds JP, Eidelman BH, Wald A. Prevalance of bowel dysfunction in multiple sclerosis. A population survey. *Gastroenterology* 1990;98:1538-42
19. Battle WM, Snape WJ Jr, Alavi A, Cohen S, Braunstein S. Colonic dysfunction in diabetes mellitus. *Gastroenterology*. 1980 Dec; 79(6):1217-1221.
20. Altomare D, Pilot MA, Scott M, Williams N, Rubino M, Ilincic L, Waldron D. Detection of subclinical autonomic neuropathy in constipated patients using a sweat test. *Gut*. 1992 Nov; 33(11):1539-1543.
21. von der Ohe MR, Camilleri M, Carryer PW. A patient with localized megacolon and intractable constipation: evidence for impairment of colonic muscle tone. *Am J Gastroenterol*. 1994 Oct; 89(10):1867-70.
22. Gattuso JM, Kamm MA, Talbot JC. Pathology of idiopathic megarectum and megacolon. *Gut*. 1997 Aug; 41(2):252-257.
23. Krishnamurthy S, Schuffler MD, Rohrmann CA, Pope CE 2nd. Severe idiopathic constipation is associated with a distinctive abnormality of the colonic myenteric plexus. *Gastroenterology*. 1985 Jan; 88(1 Pt 1):26-34.
24. Milner P, Belai A, Tomlinson A, Hoyle CH, Sarner S, Burnstock G. Effects of long-term laxative treatment on neuropeptides in rat mesenteric vessels and caecum. *J Pharm Pharmacol*. 1992 Sep; 44(9):777-9.
25. Smith B. Pathologic changes in the colon produced by anthraquinone purgatives. *Dis Colon Rectum*. 1973 Nov-Dec; 16(6):455-8.
26. Sabri M, Barksdale E, Di Lorenzo C. Constipation and lack of colonic interstitial cells of Cajal. *Dig Dis Sci*. 2003 May; 48(5):849-53.
27. Davies GJ, Crowder M, Reid B, Dickerson JW. Bowel function measurements of individuals with different eating patterns. *Gut*. 1986 Feb; 27(2):164-9.
28. Tucker H, Schuster MM. Irritable bowel syndrome: newer pathophysiological concepts. *Adv Intern Med*. 1982; 27:183-204.
29. Bassotti G, Chiarioni G, Imbimbo BP, Betti C, Bonfante F, Vantini I, Morelli A, Whitehead WE. Impaired colonic motor response to cholinergic stimulation in patients with severe chronic idiopathic (slow transit type) constipation. *Dig Dis Sci*. 1993 Jun; 38(6):1040-5.
30. Kamm MA, Lennard-Jones JE, Thompson DG, Sobnack R, Garvie NW, Granowska M. Dynamic scanning defines a colonic defect in severe idiopathic constipation. *Gut*. 1988 Aug; 29(8):1085-92.
31. Ewe K. Intestinal transport in constipation and diarrhea. *Pharmacology*. 1988; 36 Suppl 1:73-84.
32. Preston DM, Adrian TE, Christofides ND, Lennard-Jones JE, Bloom SR. Positive correlation between symptoms and circulating motilin, pancreatic polypeptide and gastrin concentrations in functional bowel disorders. *Gut*. 1985 Oct; 26(10):1059-64.
33. van der Sijp JR, Kamm MA, Nightingale JM, Akkermans LM, Ghatei MA, Bloom SR, Jansen JB, Lennard-Jones JE. Circulating gastrointestinal hormone abnormalities in patients with severe idiopathic constipation. *Am J Gastroenterol*. 1998 Aug; 93(8):1351-6
34. Barnes PR, Hawley PR, Preston DM, Lennard-Jones JE. Experience of posterior division of the puborectalis muscle in the management of chronic constipation. *Br J Surg*. 1985 Jun; 72(6):475-7
35. El Oufir L, Flourie B, Bruley des Varannes S, Barry JL, Cloarec D, Bornet F, Galmiche JP. Relations between transit time, fermentation products, and hydrogen consuming flora in healthy humans. *Gut*. 1996 Jun; 38(6):870-7.
36. Davies GJ, Crowder M, Reid B, Dickerson JW. Bowel function measurements of individuals with different eating patterns. *Gut*. 1986 Feb; 27(2):164-9
37. Turnbull GK, Thompson DG, Day S, Martin J, Walker E, Lennard-Jones JE. Relationships between symptoms, menstrual cycle and oro-caecal transit in normal and constipated women. *Gut*. 1989 Jan; 30(1):30-4.
38. Hinds JP, Stoney B, Wald A. Does gender or the menstrual cycle affect colonic transit? *Am J Gastroenterol*. 1989 Feb; 84(2):123-6.
39. Voderholzer WA, Schatke W, Muhlendorfer BE, Klauser AG, Birkner B, Muller-Lissner SA. Clinical response to dietary fiber treatment of chronic constipation. *Am J Gastroenterol*. 1997 Jan; 92(1):95-8.
40. Pezim ME, Pemberton JH, Levin KE, Litchy WJ, Phillips SF. Parameters of anorectal and colonic motility in health and in severe constipation. *Dis Colon Rectum*. 1993 May; 36(5):484-91.
41. Altomare DF, Portincasa P, Rinaldi M, Di Ciaula A, Martinelli E, Amoroso A, Palasciano G, Memeo V. Slow-transit constipation: solitary symptom of a systemic gastrointestinal disease. *Dis Colon Rectum*. 1999 Feb; 42(2):231-40.
42. Knowles CH, Martin JE. Slow transit constipation: a model of human gut dysmotility. Review of possible aetiologies. *Neurogastroenterol Motil*. 2000 Apr; 12(2):181-96.
43. Lowry AC, Simmang CL, Boulos P, Farmer KC, Finan PJ, Hyman N, Killingback M, Lubowski DZ, Moore R, Penfold C, Savoca P, Stitz R, Tjandra JJ. Consensus statement of definitions for anorectal physiology and rectal cancer: report of the Tripartite Consensus Conference on Definitions for Anorectal Physiology and Rectal Cancer, Washington, D.C., May 1, 1999. *Dis Colon Rectum*. 2001 Jul; 44(7):915-9
44. Jorge JM, Wexner SD, Ger GC, Salanga VD, Nogueras JJ, Jagelman DG. Cine-defecography and electromyography in the diagnosis of nonrelaxing puborectalis syndrome. *Dis Colon Rectum*. 1993 Jul; 36(7):668-76.
45. Schouten WR, Briel JW, Auwerda JJ, van Dam JH, Gosselink MJ, Ginai AZ, Hop WC. Anismus: fact or fiction? *Dis Colon Rectum*. 1997 Sep; 40(9):1033-41.
46. Jorge JM, Wexner SD, Ehrenpreis ED, Nogueras JJ, Jagelman DG. Does perineal descent correlate with pudendal neuropathy? *Dis Colon Rectum*. 1993 May; 36(5):475-483.
47. van Dam JH, Ginai AZ, Gosselink MJ, Huisman WM, Bonjer HJ, Hop WC, Schouten WR. Role of defecography in predicting clinical outcome of rectocele repair. *Dis Colon Rectum*. 1997 Feb; 40(2):201-7.
48. Agachan F, Pfeifer J, Wexner SD. Defecography and proctography. Results of 744 patients. *Dis Colon Rectum*. 1996 Aug; 39(8):899-905
49. Bouchoucha M, Devroede G, Arhan P, Strom B, Weber

- J, Cugnenc PH, Denis P, Barbier JP. What is the meaning of colorectal transit time measurement? *Dis Colon Rectum*. 1992 Aug;35(8):773-82
50. Fleshman JW, Dreznik Z, Cohen E, Fry RD, Kodner IJ. Balloon expulsion test facilitates diagnosis of pelvic floor outlet obstruction due to nonrelaxing puborectalis muscle. *Dis Colon Rectum*. 1992 Nov; 35(11):1019-25.
51. Finco C, Luongo B, Savastano S, Polato F, Sarzo G, Caruso V, De Lazzari F, Merigliano S. Selection criteria for surgery in patients with obstructed defecation, rectocele and anorectal prolapse. *Chir Ital*. 2007 Jul-Aug;59(4):513-20
52. Rao SS. Constipation: Evaluation and Treatment of Colonic and Anorectal Motility Disorders. *Gastroenterol Clin North Am*. 2007 Sep; 36(3):687-711.
53. Platell C, Scache D, Mumme G, Stitz R. A long-term follow-up of patients undergoing colectomy for chronic idiopathic constipation. *Aust N Z J Surg*. 1996 Aug; 66(8):525-9.
54. Beck DE, Fazio VW, Jagelman DG, Lavery IC. Surgical management of colonic inertia. *South Med J*. 1989 Mar; 82(3):305-9.
55. Rantis PC Jr, Vernava AM 3rd, Daniel GL, Longo WE. Chronic constipation--is the work-up worth the cost? *Dis Colon Rectum*. 1997 Mar; 40(3):280-6.
56. Marchesi F, Sarli L, Percalli L, Sansebastiano GE, Veronesi L, Di Mauro D, Porrini C, Ferro M, Roncoroni L. Subtotal colectomy with antiperistaltic cecorectal anastomosis in the treatment of slow-transit constipation: long-term impact on quality of life. *World J Surg*. 2007 Aug;31(8):1658-64
57. Christiansen J, Rasmussen OO. Colectomy for severe slow-transit constipation in strictly selected patients. *Scand J Gastroenterol*. 1996 Aug;31(8):770-3
58. You YT, Wang JY, Changchien CR, Chen JS, Hsu KC, Tang R, Chiang JM, Chen HH. Segmental colectomy in the management of colonic inertia. *Am Surg*. 1998 Aug;64(8):775-7
59. Rivera MT, Kugathasan S, Berger W, Werlin SL. Percutaneous colonoscopic cecostomy for management of chronic constipation in children. *Gastrointest Endosc*. 2001 Feb; 53(2):225-8.
60. Pinho M, Yoshioka K, Keighley MR. Long-term results of anorectal myectomy for chronic constipation. *Dis Colon Rectum*. 1990 Sep; 33(9):795-7.
61. Eccersley AJ, Maw A, Williams NS. Comparative study of two sites of colonic conduit placement in the treatment of constipation due to rectal evacuatory disorders. *Br J Surg*. 1999 May; 86(5):647-50.
62. Inoue Y, Noro H, Komoda H, Kimura T, Mizushima T, Taniguchi E, Yumiba T, Itoh T, Ohashi S, Matsuda H. Completely laparoscopic total colectomy for chronic constipation: report of a case. *Surg Today*. 2002; 32(6):551-4.
63. Kessler H, Hohenberger W. Laparoscopic total colectomy for slow-transit constipation. *Dis Colon Rectum*. 2005 Apr; 48(4):860-1.
64. Glia A, Meta Gylin, Gullberg K, Lindberg G. Biofeedback retraining in patients with functional constipation and paradoxical puborectalis contraction *Dis Colon Rectum*.1997;40:889-96
65. Karlbom U, Hålliden M, Eeg-Olofsson KE, Pålman L, Graf W. Results of biofeedback in constipated patients *Dis Colon Rectum*.1997;40:1149-6
66. Hallan RI, Williams NS, Melling J, Waldron DJ, Womack NR, Morrison JF. Treatment of anismus in intractable constipation with botulinum-A toxin. *Lancet*. 1988 Sep 24; 2(8613):714-7.

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