

Inpatient Constipation



Joshua Allen-Dicker, MD, MPH^{a,b,*}, Jonathan Goldman, MD^{a,c},
Brijen Shah, MD^{d,e}

KEYWORDS

• Inpatient constipation • Prevention • Behavioral therapy • Pharmacologic therapy

HOSPITAL MEDICINE CLINICS CHECKLIST

1. Any of the following signs should raise concern for inpatient constipation:
 - a. A decrease in the frequency of a patient's bowel movements compared with their ambulatory baseline.
 - b. The absence of sensation of complete evacuation.
 - c. Reported need for increased straining with defecation.
 - d. The need for digitalization or per rectum therapy to evacuate.
2. Thorough medication reconciliation should be performed at the time of admission for all patients to identify common medications that could predispose them to constipation.
3. Strategies for preventing inpatient constipation should take into account a patient's risk factors for developing constipation (age >60 years, history of outpatient constipation, need for intensive care unit stay or intubation, intra-abdominal surgery within the last week, or planned use of a known constipation-causing medication for longer than 24 hours).
4. For patients deemed at high risk for developing constipation, a scheduled osmotic laxative such as polyethylene glycol should be initiated.
5. New-onset inpatient constipation should be viewed with a critical eye for evidence of serious underlying pathophysiology (eg, obstruction, motility disorder, systemic illness).

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^a Harvard Medical School, Boston, MA 02115, USA; ^b Division of General Medicine and Primary Care, Department of Medicine, Beth Israel Deaconess Medical Center, 330 Brookline Avenue, W/Space-2, Boston, MA 02215, USA; ^c Department of Medicine, Beth Israel Deaconess Hospital Needham, Needham, MA 02492, USA; ^d Division of Gastroenterology, Department of Medicine, Icahn School of Medicine at Mount Sinai, New York, NY, USA; ^e Department of Geriatrics and Palliative Medicine, Icahn School of Medicine at Mount Sinai, New York, NY, USA

* Corresponding author.

E-mail address: DrJoshuaAD@gmail.com

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6. Treatment of constipation can begin with an osmotic laxative (and rectal stimulant laxative in certain populations).
7. Treatment-resistant constipation should prompt a thorough reevaluation of the patient, including a physical examination and potential abdominal imaging to assess for ileus or obstruction. If reevaluation is unrevealing, an osmotic laxative bowel preparation should be considered.

DEFINITIONS*How is constipation defined?*

Colloquially, constipation is thought of as a problem with stool frequency. Normal frequency is reported to be between 3 bowel movements per day and 3 per week.¹ Qualitative features of defecation (eg, straining, stool character, sensation of incomplete evacuation) are an equally important component of constipation. Assessment of both of these elements can help in determining the cause and treatment plan for this common problem.

The Rome III criteria offer a consensus definition of constipation that accounts for both qualitative and quantitative functioning.² Per these guidelines, a patient must:

1. Have rare loose stool without use of laxatives
2. Fail to meet criteria for irritable bowel syndrome
3. Demonstrate 2 of the following: straining during at least 25% of defecations, lumpy or hard stools in at least 25% of defecations, sensation of incomplete evacuation for at least 25% of defecations, sensation of anorectal obstruction/blockage for at least 25% of defecations, manual maneuvers required to facilitate at least 25% of defecations (eg, digital evacuation, support of the pelvic floor), or fewer than 3 defecations per week.

How is inpatient constipation defined?

Existing diagnostic criteria for constipation such as the Rome III focus on chronic bowel disorders in the ambulatory patient and not the acute inpatient disorders focused on herein. For this purpose, the authors propose a new mechanism for defining inpatient constipation. A hospitalized patient meeting any of the following criteria should be considered to have inpatient constipation:

1. A decrease in the frequency of bowel movements compared with their ambulatory baseline
2. The absence of sensation of complete evacuation
3. Reported need for increased straining with defecation
4. The need for digitalization or per rectum therapy to evacuate.

EPIDEMIOLOGY*How common is constipation in the general population?*

Prior estimates of constipation prevalence in community-dwelling adults have ranged from 2.5% to 79%.³ Precision with this estimate is impaired by several factors, including poor reporting and heterogeneous definitions of disease.

Prior studies have identified specific subpopulations that may be at increased risk for constipation. Constipation has consistently been demonstrated to increase with age, with the highest prevalence in those older than 60 years.³ Gender discrepancies in constipation have also been identified, with an increased prevalence in females.^{3,4} Prior studies have associated frequency of constipation with lower income levels and lower levels of parental education, suggesting a role for socioeconomic factors.

Constipation prevalence may also vary geographically. One review identified a low median prevalence of 10.8% in Asian populations. By contrast, constipation was more common in North American, European, and Oceanic populations, with a median prevalence of 16%, 19.2%, and 19.7% respectively.³ Although diet and other modifiable factors likely play a role in this variation, these results suggest that cultural and genetic factors may also affect the pathophysiology of constipation.

How common is constipation as a complication of inpatient hospital stays?

Hospitalized patients are at an increased risk for constipation in comparison with their community-dwelling counterparts. One study comparing the prevalence of constipation across locations found that patients on a geriatric ward had a prevalence of 79%, compared with those in a nursing home (59%) and elderly community dwellers (38%).⁵

Within the inpatient population, there are several subpopulations that have been shown to be at increased risk for constipation. Critically ill patients are routinely reported to be at increased risk of constipation.⁶ One such report found the rate of constipation in a medical and surgical intensive care unit population to be as high as 69.9%.⁷ The postoperative period also poses an increased risk for the development of constipation.⁸ In one study of postoperative thoracic surgery patients, 50% reported having dry or hard stools or difficulty with evacuation in the initial postoperative period; on average it took 17 days to reestablish a baseline defecatory pattern after surgery.⁹

In each of these populations, the pathophysiology is likely a result of multiple underlying factors, including immobility, systemic inflammation, administration of opioid medications, and decreased enteral fiber and fluid intake.

What is the cost of inpatient constipation?

The direct economic burden of inpatient constipation is considerable, but understudied. The 2001 National Ambulatory Medical Care Survey, National Hospital Ambulatory Medical Care Survey, and National Hospital Discharge Survey identified 2.7 million provider visits that year whereby constipation was the reason for seeking care or the primary diagnosis, and found the total cost of constipation to be US\$235 million.¹⁰ Although only 1.4% of the cases were inpatient visits, they were responsible for 55% of the cost (\$129 million). It is likely that this is a significant underestimation of the direct cost of inpatient constipation, as the study was designed to examine only cases for which constipation was the primary diagnosis, and did not include patients who developed constipation as a secondary complication of their hospitalization. A subsequent study found that inpatients who were prescribed treatment for constipation were hospitalized 0.69 days longer at an additional cost of \$1668 per patient, compared with patients who did not receive treatment for constipation. Taken together, these studies suggest that inpatient constipation carries a significant financial cost.¹¹

In addition to this direct financial burden of inpatient constipation, there is also the indirect cost of constipation on patient care. Constipation in the critical care setting is

associated with prolonged ventilation, intravenous vasopressor requirements, length of stay, and mortality.^{6,12,13} Through its effects on the aforementioned outcomes, constipation likely results in greater indirect downstream costs in the form of diagnostic tests, treatment, and prolonged hospital stays. One could also hypothesize similar associations for inpatients outside of the critical care setting.

Although research has yet to fully examine the full financial burden of inpatient constipation and its relationship to length of stay, readmission, and patient satisfaction, available data to date suggest a substantial effect.

What are the risk factors for developing constipation in the hospital?

Among inpatients, factors intrinsic to the patient, in addition to extrinsic factors related to the hospital environment itself, may compound the risk of developing constipation.

A somewhat obvious, but nevertheless major risk factor for developing constipation is personal history.¹⁴ Those patients with constipation in the ambulatory setting and/or a history of laxative use are particularly prone to developing constipation in the hospital.

Elderly inpatients are at increased risk of developing constipation in comparison with their outpatient counterparts, which may be related to decreased appetite resulting in a poor gastrocolic reflex, weakening of the abdominal muscles that normally initiate defecation, and reductions in anal squeeze pressure and anal sensation.¹⁵

Underlying comorbidities may also place a patient at risk: disease processes such as hypercalcemia, scleroderma, muscular dystrophy, and pregnancy can alter bowel motor function; disorders such as hypothyroidism, diabetes, Parkinson disease, and spinal cord lesions can impair the neurologic inputs to the bowel.

As discussed earlier, critical illnesses and postoperative status can both predispose patients to constipation.

Extrinsic factors native to the hospital setting may also contribute to the increased risk of constipation. Treatment of acute illnesses often necessitates the initiation of new medications, many of which (opiates, nonsteroidal anti-inflammatory drugs, antiemetics, diuretics, calcium-channel blockers, iron supplements, adrenergic vasopressors, and any medication with anticholinergic effects) can result in decreased bowel motility (**Table 1**).¹⁶ Dehydration and dietary changes associated with illness, in addition to decreased mobility secondary to acute illness or medically indicated hospital tethers (eg, telemetry, catheters, intravenous pumps) may also result in slowed bowel motility.

What are the different classes of constipation and how do they relate to the inpatient setting?

Normal transit of stool through the bowel depends on intact motor function of smooth muscle and appropriate neurologic inputs via the brain, spinal cord, and enteric plexus. Constipation can therefore be classified by the speed at which the stool moves through the colon.¹⁵ Conventional classes include slow-transit, normal-transit, and no-transit constipation, anorectal dysfunction, and obstruction.

Slow-transit constipation refers to constipation whereby the stool passes through the colon at a decreased rate, and includes any process that results in disordered colonic movement.

No-transit constipation is an extreme case of slow-transit constipation whereby the stool does not transit at all. Often referred to as ileus, this class of constipation is the end result of a variety of neurologic, inflammatory, and pharmacologic pathways.

Table 1
Common medications associated with an increased risk of chronic constipation

Medication Class	Odds Ratio ^a	95% CI
Aluminum antacids	1.72	1.48–2.01
Antipsychotics (eg, chlorpromazine)	1.90	1.54–2.34
Antispasmodics (eg, dicyclomine)	3.34	2.94–3.80
Antidepressants (eg, amitriptyline)	1.91	1.71–2.13
Antihistamines (eg, diphenhydramine)	1.76	1.59–1.94
Anticonvulsants (eg, carbamazepine, phenytoin)	2.82	2.19–3.63
Calcium supplements	2.49	1.54–4.03
Diuretics (eg, furosemide)	1.65	1.47–1.86
Iron supplements (eg, ferrous sulfate)	1.48	1.29–1.71
Opioid analgesics (eg, morphine)	1.63	1.40–1.91
5-HT ₃ receptor antagonists (eg, ondansetron)	Not available	—
Antineoplastic agents (eg, vinca alkaloid agents)	Not available	—
β-Blockers (eg, atenolol)	Not available	—
Nonsteroidal anti-inflammatory agents (eg, ibuprofen)	Not available	—
Calcium-channel blockers (eg, verapamil, nifedipine)	Not available	—

Abbreviations: CI, confidence interval; 5-HT, 5-hydroxytryptamine (serotonin).

^a Refers to risk of development of chronic constipation.

Data from Talley NJ, Jones M, Nuyts G, et al. Risk factors for chronic constipation based on a general practice sample. *Am J Gastroenterol* 2003;98(5):1107–11.

Normal-transit constipation refers to constipation whereby the stool passes through the colon at a normal rate, but with decreased frequency of evacuation; this may be the result of disorders of anorectal sensation or motor function, or inadequate stool bulk (low fiber).

Anorectal dysfunction refers to disorders that impair the normal functioning of the anorectal anatomy, including disorders of the pelvic floor and internal and external anal sphincters.

Obstruction refers to a mechanical obstacle to bowel transit, which may result from barriers extrinsic or intrinsic to the bowel wall. Extrinsic factors, such as postsurgical or inflammatory intra-abdominal adhesions, are the most common cause.¹⁷ Intrinsic factors include strictures, malignancy, and inspissated stool.

HISTORY, EXAMINATION, AND DIAGNOSIS

What aspects of the medical history are useful in determining the type of constipation?

Treatment modalities for constipation depend on the underlying pathophysiology. Therefore, to help develop a more effective treatment plan, the authors recommend that clinicians consider the presence of slow-transit, no-transit, and normal-transit constipation, anorectal dysfunction, and obstruction. When approaching an inpatient who reports ongoing constipation, a detailed history will help identify the potential etiology.

No-transit constipation (ileus) is associated with a history of intra-abdominal surgery (either remote or recent), ongoing intra-abdominal infection, exposure to inhalational

anesthetics, and acute systemic illness. It typically presents with new symptoms of abdominal distention, poorly localized abdominal pain, and nausea. In addition to constipation, patients may also report absence of flatus (obstipation).

Patients with normal-transit constipation may report a history of chronic constipation symptoms such as a sense of incomplete evacuation, with or without abdominal pain and distension relieved by moving their bowels. Bowel frequency is usually normal.

Slow-transit constipation is a decrease in bowel frequency from what is accepted as normal physiology.

Anorectal dysfunction is typically a chronic process seen in elderly patients and women who report difficulty with defecation, excessive straining, and a sensation of anorectal obstruction. On history, patients may report intermittent leakage of liquid stool or frequent need for manual disimpaction. Loose liquid-like stool may represent overflow diarrhea in the patient with constipation who has anorectal dysfunction, caused by movement of loose fecal material around more solid inspissated stool.

Acute obstruction may present as new-onset vomiting associated with midabdominal pain and obstipation. Symptoms may be reported as paroxysmal and cramping. Emesis may be feculent. Malignancy is a less common cause of obstruction, but may result as chronic onset of the aforesaid symptoms associated with a change in stool caliber, blood in the stool, iron-deficiency anemia, or systemic signs of chronic illness.¹⁷

Are there warning signs for inpatient constipation that should prompt further workup?

Any patient who develops constipation as an inpatient should be examined with a critical eye. On history, the physician should elicit any prior malignancies and differentiate between constipation and obstipation. Of note, what may appear to an inpatient physician as acute constipation may actually be an exacerbation of a more chronic process. The physician is obligated to ask about what each patient's "normal" status is and the time course of any recent changes. In addition, a thorough review of systems may reveal evidence of a chronic neoplastic, gastroenterologic, endocrinologic, or neurologic disorder.

On physical examination a clinician should look for absent bowel sounds, intra-abdominal masses, ascites, pain, abdominal rebound or guarding, a rectal mass, a full rectal vault, changes in rectal tone, or new lower extremity neurologic deficits. A review of laboratory values can discover electrolyte imbalances such as hypercalcemia.

MANAGEMENT

What nonpharmacologic interventions can be used to prevent or treat inpatient constipation?

Although diet is frequently thought to contribute to constipation, literature on the use of dietary interventions for constipation remains conflicting.^{18,19} Foods rich in dietary fiber, including legumes, whole grains, nuts, fruits, and vegetables, may improve bowel transit. Some inpatient settings allow for the ordering of high-fiber diets, which may benefit patients with histories of chronic constipation. Before such an approach, one must be aware of the side effects commonly reported with these dietary interventions, which include flatulence, bloating, and poor taste.

Behavioral interventions may also provide benefit to acutely ill inpatients. Allowing for scheduled time on a commode or toilet, regardless of urge to stool, may improve stooling frequency. Similarly, encouraging physical activity may also promote bowel activity. Unfortunately, quality research in these 2 areas remains absent.

What pharmacologic interventions can be used to prevent or treat inpatient constipation?

In discussing pharmacologic agents, the authors use the term laxative to refer to any compound or drug that can facilitate bowel movements. There are multiple available pharmacologic laxatives (Table 2). However, there are significant limitations to the body of evidence supporting the use of these interventions: (1) a dearth of high-quality and head-to-head trials; and (2) study populations consisting mostly of chronically constipated and not acutely constipated persons, or persons undergoing bowel preparation before endoscopy.

Surfactant Laxatives

Surfactant laxatives act as detergents, decreasing surface tension and allowing increased water entry into stool. A well-known surfactant laxative is docusate sodium. Prior studies of this drug class have not consistently demonstrated a meaningful effect and have been limited by poor study quality.^{20,21} However, possibly as a result of their low cost (\$0.05 per dose) (see Table 2), this drug class is frequently used in both the inpatient and outpatient settings. Onset of action for this class of medications is thought to be between 12 and 72 hours.

Bulk-Forming Laxatives

Bulk-forming laxatives act by absorbing water, thus adding bulk and softening the stool. Examples in this class include psyllium, methylcellulose, and wheat dextran. Within the class, psyllium has the best overall supporting evidence. Prior research has demonstrated improvement in stool frequency and ease of defecation with use of psyllium versus placebo in chronic constipation.²² In addition, a head-to-head trial demonstrated superiority of psyllium over docusate sodium in improving stool

Laxative Class	Drug	Onset of Action	Average Cost per Dose (US\$)
Surfactant	Docusate sodium	12–72 h	0.05
Bulk-forming	Psyllium	12–72 h	0.06
Osmotic	Polyethylene glycol 3350	24–48 h	0.50
	Lactulose	24–48 h	0.81
	Magnesium citrate	<6 h	1.18
Stimulant	Senna	6–10 h	0.30
	Bisacodyl (oral)	6–10 h	0.33
	Bisacodyl (rectal)	<60 min	0.19
Enema	Normal saline	<60 min	—
	Sterile water	<60 min	—
	Sodium phosphate	<60 min	—
Opioid antagonist	Methylnaltrexone	<30 min	72.50

Cost analysis was completed using Medi-Span. When unavailable, online reviews of national drug-store chain pricing were performed. Of note, these data on costs are based on estimated outpatient pricing and may not reflect inpatient costs.

frequency in chronically constipated patients.²³ The research quality of both of these trials is considered to be poor, but remains the best evidence in support of this class of drugs. Of note, bulk-forming laxatives are generally fermented by colonic flora, which can lead to symptoms of bloating and cramping. Psyllium is generally priced \$0.06 per dose. Onset of action of this class of medications is thought to be 12 to 72 hours.

Osmotic Laxatives

Osmotic laxatives contain poorly absorbed molecules that create an osmotic gradient to pull water into the intestinal lumen, thus softening the stool. Examples of osmotic laxatives include polyethylene glycol 3350, lactulose, and magnesium citrate. Of these medications, polyethylene glycol is thought to have the strongest supporting evidence.²¹ Multiple trials have demonstrated its superiority over placebo.^{24,25} Several head-to-head trials have demonstrated superiority of polyethylene over other common laxatives, although the design quality of these studies (eg, open-label, homogeneous populations) remains suboptimal. In a trial comparison with lactulose, polyethylene glycol demonstrated superiority with respect to stool frequency and straining.²⁶ Similarly, in a trial versus psyllium, polyethylene glycol demonstrated superior outcomes.²⁷ Whereas lactulose is fermented by colonic bacteria, leading to gas production and bloating, polyethylene glycol is not.

The literature examining magnesium citrate is focused on its use as a preparatory agent before colonoscopy. In this setting, it has been shown to be as effective as polyethylene glycol.^{28,29} However, there are concerns regarding its potential for causing hypermagnesemia, either as a result of one-time usage in those with reduced kidney function, or with chronic usage in those with even normal kidney function.

Polyethylene glycol is generally \$0.50 per dose, lactulose \$0.81 per dose, and magnesium citrate \$1.18 per dose. Onsets of action are less than 6 hours for magnesium citrate, 24 to 48 hours for lactulose, and 24 to 48 hours for polyethylene glycol.³⁰

Stimulant Laxatives

Stimulant laxatives act by enhancing colonic secretion and increasing intestinal motility via stimulation of the myenteric plexus. The most well-known members in this class are bisacodyl and senna. Bisacodyl has strong evidence for superiority over placebo, but no notable head-to-head trials with other drugs.³¹ Senna has been shown to be superior to lactulose in improving stool frequency and consistency, although the overall quality of the literature remains poor.³² Cramping may result from either medication. Senna is often priced \$0.30 per dose; bisacodyl \$0.33 per dose. Oral bisacodyl and senna are both thought to have an onset of action of 6 to 10 hours. Bisacodyl is unique in that it is also available in a rectal formulation with a markedly faster onset of action (15–60 minutes). Bisacodyl may thus be an option for those patients who are not able to tolerate anything by mouth, but may only be effective for patients in whom stool has reached the rectal vault or the left side of the colon. Prior literature suggested that long-term use of these medications damages the colonic nervous system; however, recent studies have refuted this claim.³³

Enema

Enemas are liquid suspensions that act by softening stool and stimulating the rectum when introduced into the rectum. Although specific properties of an enema largely depend on the chemical composition of the suspension (common solutions include normal saline, sterile water, and sodium phosphate), overall high-quality data on effectiveness are lacking for all forms of enemas. Enemas are most helpful in patients who have functional or anatomic reasons that impair their ability to empty the rectum. One

particular class of patients who may benefit from enema therapy are those who do not have rectal sensation (eg, those with spinal cord injury). In these patients, a rectal examination positive for impacted stool should prompt consideration of an enema.

From a safety standpoint, no enema should ever be administered to a patient who cannot subsequently evacuate the enema (eg, patients with altered mental status or neurologic deficits). If an enema is retained, damage to the colonic mucosa and fluid-electrolyte shifts can occur (eg, hyponatremia, hyperphosphatemia). Sodium phosphate enemas are contraindicated in any patient with impaired renal function, as severe hyperphosphatemia and hypocalcemia can result.

Given the heterogeneity of enema formulations, there are no consistent data on pricing. Onset of action is generally within 60 minutes.

Peripheral Opioid Antagonists

Peripheral opioid antagonists, such as methylnaltrexone, act to reverse the effects of opiate medications on bowel motility. This class of medication has restricted penetration of the central nervous system, and therefore does not reverse the analgesic effects of opiates. It has good-quality evidence and but no head-to-head comparisons with other interventions.³⁴ It has limited Food and Drug Administration approval (only for opioid-induced constipation in patients with advanced illness who are receiving palliative care), and is comparatively costly (\$72.50). Average onset of action is 30 minutes.

What are appropriate first-line interventions to prevent inpatient constipation?

Published research has focused on management of chronic constipation. No evidence-based protocols for prevention of inpatient constipation have been published or validated. The authors therefore base the following set of recommendations on existing literature on effectiveness, safety, pharmacology, and head-to-head comparisons, where available.

As a first step in preventing inpatient constipation, on admission each patient should be assessed for their risk of developing constipation during their hospitalization. Patients should be considered high risk if they have any of the following attributes: age greater than 60 years, a history of outpatient constipation, need for intensive care unit stay or intubation, intra-abdominal surgery within the last week, or planned use of a known constipation-causing medication for longer than 24 hours (see earlier discussion). Inpatients without any of these attributes can be considered as being at low risk (Fig. 1).

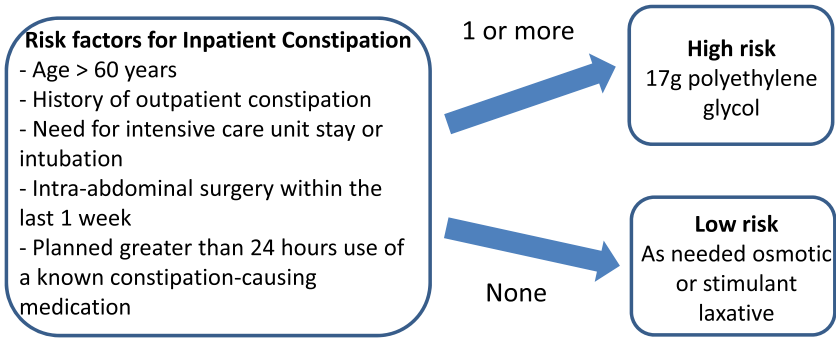


Fig. 1. Approach to prevention of constipation in inpatients.

For patients identified as at high risk for constipation, evidence best supports starting them on 17 g oral polyethylene glycol daily. The latter can be held if the patient has more than 1 bowel movement per day. Where available, and when medically feasible, the authors recommend encouraging dietary fiber intake. Bowel movements should be documented in the medical record with vital signs, and should be part of a daily rounding routine.

Those patients stratified into the low-risk category for constipation should be started on a low-dose stimulant or osmotic laxative as needed, which have a low number needed to treat.³⁵ These medications should be given 30 minutes after eating to coincide with the morning gastrocolic reflex. Patient education is key to successful use of these medications in this population. The use of soluble fiber is not recommended because relevant data in the outpatient setting are of low quality, and it requires several days to weeks to see an improvement in bowel frequency.³⁶

Regardless of risk status, close coordination between physician, staff, and patient should occur to ensure: (1) encouragement for the patient to be out of bed and mobile (when medically appropriate), and (2) use of the toilet/commode at least twice a day regardless of patient-reported urge to move the bowels.

When possible, the authors recommend that the physician and the patient discuss the reasons for these interventions. The medical team and patient can therefore best ensure compliance, quick identification of new-onset constipation, and avoidance of any constipation-associated complications.

At discharge the medical team and patient should cooperatively assess the need for continuation of any of the aforementioned interventions. In patients who did not have constipation as a problem before admission, many will return to normal bowel function without the use of medications if they return to their baseline mobility status and outpatient diet. However, patients should be counseled about the risk of constipation from any new medications and the effects of new limitations in mobility or changes in diet, so that treatment can be considered accordingly.

What are appropriate first-line interventions to treat new-onset inpatient constipation?

The following discussion addresses treatment of slow-transit and normal-transit constipation. When approaching suspected no-transit constipation (ileus), anorectal dysfunction, and acute obstruction, management should begin with referral to the appropriate specialist (eg, gastroenterologist, surgeon).

Patients with new-onset constipation should undergo assessment regarding the underlying cause of their constipation. Understanding the etiology will help to focus appropriate treatment (eg, use of stimulant laxative for a condition promoting decreased bowel motility, use of a bulking laxative for a patient with decreased dietary intake) and identify relevant warning signs (as discussed next under treatment-resistant inpatient constipation).

As a general approach to treating inpatient constipation (**Fig. 2**), patients are considered as being either laxative-naïve (developing constipation in the absence of a constipation prevention regimen) or laxative-exposed (developing constipation in the presence of a constipation prevention regimen).

For those patients who have not been taking laxatives during their hospitalization, the authors recommend a trial of an osmotic laxative and stimulant laxative. Available evidence is most supportive of 17 g oral polyethylene glycol daily and either 17.2 mg oral senna daily or 10 mg oral bisacodyl daily.

For those patients who have been taking constipation-prevention medications during their hospitalization, the authors recommend a trial of an increased-dose osmotic

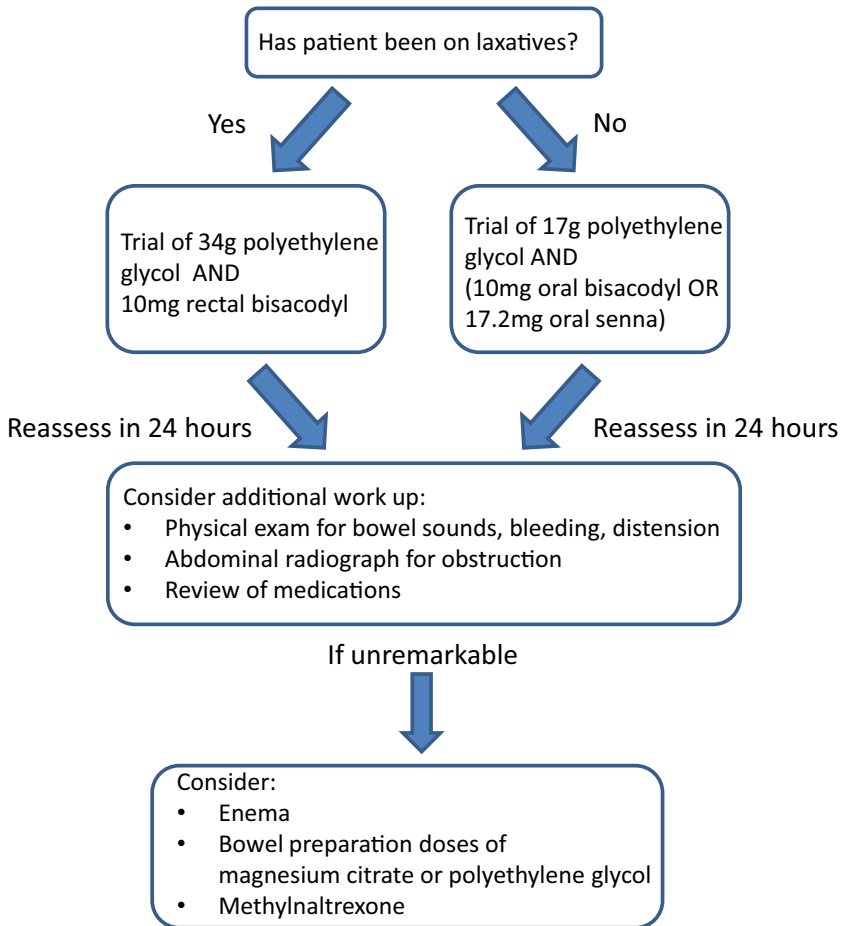


Fig. 2. Approach to treatment of constipation for inpatients.

laxative and stimulant laxative. The evidence is most supportive of 34 g oral polyethylene glycol and, because of its faster onset of action, 10 mg rectal bisacodyl.

How should one approach treatment-resistant inpatient constipation?

Certain patients may fail to respond to first-line therapy for constipation. Failure of response may be defined by absence of laxation after 24 hours of appropriate constipation-treating therapy. In these patients, the treating physician should consider additional workup and interventions.

Given that misdiagnosis of ileus or small bowel as constipation may be associated with worsening of symptoms and even bowel perforation, the authors recommend a low threshold for abdominal imaging with an abdominal radiograph. Distended loops of small bowel or colon should prompt divergence from the constipation treatment pathway. Additional workup of patients with a history of colonic malignancy or warning signs for malignancy (with a barium enema film or abdomen/pelvis computed tomography with contrast) to rule out obstructing lesions should be considered. Rectal examination to rule out inspissated stool (if not done earlier) should be completed at this time.

Following this consideration, the authors recommend initiation of an osmotic laxative bowel preparation agent (polyethylene glycol or magnesium citrate). In comparison with a stepwise augmentation of the patient's bowel regimen, this approach has the benefit of producing more timely laxation.

Enemas can also be used as needed for the treatment of acute constipation. Based on safety concerns, the authors recommend a trial of a saline enema over a phosphate enema. Initiating scheduled enemas without further workup and consultation from a gastroenterologist is not recommended.

Similarly, before escalating beyond this regimen, consultation with a gastroenterologist is recommended (if this has not already been done).

In patients with cancer who have suspected opiate-induced constipation and who are resistant to the aforesaid regimen, the authors recommend discussion with an oncologist or palliative care specialist before initiating methylnaltrexone.

PERFORMANCE IMPROVEMENT

At present there are no specific performance improvement measures related to prevention and treatment of inpatient constipation. However, the following practices may help improve the time to diagnosis and subsequent relevant clinical outcomes:

- Incorporation of constipation-related themes (eg, risk factors, warning signs) into the history and physical examination
- Continued vigilance for onset of constipation and changing risk factors throughout the hospital course
- Critical analysis of new-onset constipation
- Expedient use of appropriate therapy for prevention and treatment of constipation

GUIDELINES

No official guidelines exist regarding the diagnosis, prevention, and treatment of inpatient constipation.

REFERENCES

1. Martelli H, Devroede G, Arhan P, et al. Some parameters of large bowel motility in normal man. *Gastroenterology* 1978;75(4):612–8.
2. Longstreth GF, Thompson WG, Chey WD, et al. Functional bowel disorders. *Gastroenterology* 2006;130(5):1480–91.
3. Mugie SM, Benninga MA, Di Lorenzo C. Epidemiology of constipation in children and adults: a systematic review. *Best practice & research. Clin Gastroenterol* 2011;25(1):3–18.
4. Greenfield SM. The management of constipation in hospital inpatients. *Br J Hosp Med* 2007;68(3):145–7.
5. Kinnunen O. Study of constipation in a geriatric hospital, day hospital, old people's home and at home. *Aging* 1991;3(2):161–70.
6. Mostafa SM, Bhandari S, Ritchie G, et al. Constipation and its implications in the critically ill patient. *Br J Anaesth* 2003;91(6):815–9.
7. Nassar AP Jr, da Silva FM, de Cleva R. Constipation in intensive care unit: incidence and risk factors. *J Crit Care* 2009;24(4):630.e9–12.
8. Sendir M, Buyukilmaz F, Asti T, et al. Postoperative constipation risk assessment in Turkish orthopedic patients. *Gastroenterol Nurs* 2012;35(2):106–13.

9. Rasmussen LS, Pedersen PU. Constipation and defecation pattern the first 30 days after thoracic surgery. *Scand J Caring Sci* 2010;24(2):244–50.
10. Martin BC, Barghout V, Cerulli A. Direct medical costs of constipation in the United States. *Manag Care Interface* 2006;19(12):43–9.
11. Suh DC, Kim MS, Chow W, et al. Use of medications and resources for treatment of nausea, vomiting, or constipation in hospitalized patients treated with analgesics. *Clin J Pain* 2011;27(6):508–17.
12. van der Spoel JI, Schultz MJ, van der Voort PH, et al. Influence of severity of illness, medication and selective decontamination on defecation. *Intensive Care Med* 2006;32(6):875–80.
13. Gacouin A, Camus C, Gros A, et al. Constipation in long-term ventilated patients: associated factors and impact on intensive care unit outcomes. *Crit Care Med* 2010;38(10):1933–8.
14. Cardin F, Minicuci N, Droghi AT, et al. Constipation in the acutely hospitalized older patients. *Arch Gerontol Geriatr* 2010;50(3):277–81.
15. Lembo A, Ullman S. Constipation. In: Feldman M, Friedman L, Brandt L, editors. *Sleisenger and Fordtran's gastrointestinal and liver disease*. 9th edition. Saunders; 2010. p. 259–84.
16. Talley NJ, Jones M, Nuyts G, et al. Risk factors for chronic constipation based on a general practice sample. *Am J Gastroenterol* 2003;98(5):1107–11.
17. Miller G, Boman J, Shrier I, et al. Etiology of small bowel obstruction. *Am J Surg* 2000;180(1):33–6.
18. Tomlin J, Read NW. Laxative properties of indigestible plastic particles. *BMJ* 1988;297(6657):1175–6.
19. Badiali D, Corazzari E, Habib FI, et al. Effect of wheat bran in treatment of chronic nonorganic constipation. A double-blind controlled trial. *Dig Dis Sci* 1995;40(2):349–56.
20. Hurdon V, Viola R, Schroder C. How useful is docusate in patients at risk for constipation? A systematic review of the evidence in the chronically ill. *J Pain Symptom Manage* 2000;19(2):130–6.
21. Gartlehner G, Jonas DE, Morgan LC, et al. Drug class review on constipation drugs: final report. Portland (OR): Oregon Health & Science University; 2007.
22. Fenn GC, Wilkinson PD, Lee CE, et al. A general practice study of the efficacy of Regulan in functional constipation. *Br J Clin Pract* 1986;40(5):192–7.
23. McRorie JW, Daggy BP, Morel JG, et al. Psyllium is superior to docusate sodium for treatment of chronic constipation. *Aliment Pharmacol Ther* 1998;12(5):491–7.
24. DiPalma JA, DeRidder PH, Orlando RC, et al. A randomized, placebo-controlled, multicenter study of the safety and efficacy of a new polyethylene glycol laxative. *Am J Gastroenterol* 2000;95(2):446–50.
25. Cleveland MV, Flavin DP, Ruben RA, et al. New polyethylene glycol laxative for treatment of constipation in adults: a randomized, double-blind, placebo-controlled study. *South Med J* 2001;94(5):478–81.
26. Attar A, Lemann M, Ferguson A, et al. Comparison of a low dose polyethylene glycol electrolyte solution with lactulose for treatment of chronic constipation. *Gut* 1999;44(2):226–30.
27. Wang HJ, Liang XM, Yu ZL, et al. A randomised, controlled comparison of low-dose polyethylene glycol 3350 plus electrolytes with ispaghula husk in the treatment of adults with chronic functional constipation. *Drugs R D* 2005;6(4):221–5.
28. Lawrance IC, Willert RP, Murray K. Bowel cleansing for colonoscopy: prospective randomized assessment of efficacy and of induced mucosal abnormality with three preparation agents. *Endoscopy* 2011;43(5):412–8.

29. Hassan C, Bretthauer M, Kaminski MF, et al. Bowel preparation for colonoscopy: European Society of Gastrointestinal Endoscopy (ESGE) guideline. *Endoscopy* 2013;45(2):142–50.
30. Wang HJ, Liang XM, Yu ZL, et al. A randomised, controlled comparison of low-dose polyethylene glycol 3350 plus electrolytes with ispaghula husk in the treatment of adults with chronic functional constipation. *Clin Drug Investig* 2004; 24(10):569–76.
31. Kienzie-Horn S, Vix JM, Schuijt C, et al. Efficacy and safety of bisacodyl in the acute treatment of constipation: a double-blind, randomized, placebo-controlled study. *Aliment Pharmacol Ther* 2006;23(10):1479–88.
32. Passmore AP, Wilson-Davies K, Stoker C, et al. Chronic constipation in long stay elderly patients: a comparison of lactulose and a senna-fibre combination. *BMJ* 1993;307(6907):769–71.
33. Kiernan JA, Heinicke EA. Sennosides do not kill myenteric neurons in the colon of the rat or mouse. *Neuroscience* 1989;30(3):837–42.
34. Thomas J, Karver S, Cooney GA, et al. Methylnaltrexone for opioid-induced constipation in advanced illness. *N Engl J Med* 2008;358(22):2332–43.
35. Ford AC, Suares NC. Effect of laxatives and pharmacological therapies in chronic idiopathic constipation: systematic review and meta-analysis. *Gut* 2011;60(2): 209–18.
36. Suares NC, Ford AC. Systematic review: the effects of fibre in the management of chronic idiopathic constipation. *Aliment Pharmacol Ther* 2011;33(8):895–901.