

1

Symptomatic Care Pending Diagnosis

CHEST PAIN

Method of

William E Cayley Jr., MD, MDiv



CURRENT DIAGNOSIS

- Initial evaluation of chest pain should include evaluation of clinical stability, a concise history and physical, and a chest x-ray and electrocardiogram (ECG) unless the cause is clearly not life-threatening.
- Chest pain described as exertional, radiating to one or both arms, similar to or worse than prior cardiac chest pain, or associated with nausea, vomiting, or diaphoresis indicates high risk for acute coronary syndrome. ECG identifies ST elevation myocardial infarction (STEMI), and cardiac biomarkers are essential for further evaluation of suspected chest pain in the absence of STEMI.
- The Wells, Geneva, and Pisa clinical prediction rules can help stratify a patient's risk of pulmonary embolism.
- Aortic dissection is an uncommon cause of chest pain, but patients with abrupt or instantaneous chest pain that is ripping, tearing, or stabbing should have evaluation for dissection with chest x-ray, computed tomography (CT), or magnetic resonance imaging.
- Esophageal rupture may be suspected in patients with pain, dyspnea, and shock following a forceful emesis, and prompt imaging with CT or esophagram is essential.
- Patients who have suspected tension pneumothorax and who are clinically stable should have a chest x-ray for confirmation before needle decompression is attempted.



CURRENT THERAPY

- Patients with STEMI require urgent reperfusion, and those with unstable angina or non-ST elevation myocardial infarction require admission for further monitoring and evaluation.
- Most patients with pulmonary embolism require admission for monitoring and anticoagulation, although outpatient treatment may be possible for low-risk patients after initial evaluation and anticoagulation.
- Prompt surgical consultation is required for patients with confirmed or suspected aortic dissection or esophageal rupture.
- Clinically unstable patients with suspected tension pneumothorax need immediate needle decompression.

Epidemiology

Chest pain is the chief complaint in 1% to 2% of all outpatient primary care visits. More than 50% of emergency department (ED) visits for chest pain are due to serious cardiovascular conditions such as acute coronary syndrome (ACS) or pulmonary embolism (PE), but these account for less than 15% of outpatient primary care encounters for chest pain, and up to 15% of chest

pain episodes never reach a definitive diagnosis. Other potentially life-threatening etiologies for chest pain include PE, dissecting aortic aneurysm (AA), esophageal rupture, and tension pneumothorax. In outpatient primary care the most common causes of chest pain are musculoskeletal, gastrointestinal, angina due to stable coronary artery disease (CAD), anxiety or other psychiatric conditions, and pulmonary disease.

Initial Assessment

In initial evaluation of chest pain, it is important to obtain a clear history of the onset and evolution of chest pain, especially details such as location, quality, duration, and aggravating or alleviating factors. Initial physical examination should include vital signs, assessment of the patient's overall general condition, and examination of the heart and lungs. If there are any clinical signs of instability (altered mental status, hypotension, marked dyspnea, or other signs of shock) then initial stabilization and diagnosis must both be addressed simultaneously, consistent with current guidelines for emergency cardiovascular care. Unless the history and physical examination suggest an obviously nonthreatening cause of chest discomfort, most adults with chest pain should at least have basic diagnostic testing with an ECG and a chest x-ray. Several clinical prediction rules are available to help confirm or exclude some common causes of chest pain (Box 1).

Diagnosis and Treatment

Acute Coronary Syndrome

ACS includes acute myocardial infarction (MI) (ST-segment elevation and depression, Q wave and non-Q wave) or unstable angina. A review of prospective and retrospective studies correlating specific chest pain characteristics with the likelihood of ACS found that components of the patient history that increase the likelihood of ACS include radiation of pain to the right arm or shoulder, to both arms or shoulders, or to the left arm; or pain associated with exertion, diaphoresis, nausea, or vomiting; or pain described as pressure or as "worse than previous angina or similar to a previous MI." Similarly, components of the history that predicted decreased likelihood of ACS were pleuritic, positional, or sharp chest pain; pain in an inframammary location; or pain not associated with exertion. A physical examination finding of chest pain reproducibility with palpation also decreased the likelihood of ACS. Additional physical examination findings that may be helpful are the presence of hypotension or an S₃ on cardiac auscultation, both of which suggest increased likelihood of ACS. The Marburg Heart Score (MHS) can help exclude CAD in primary care patients with chest pain (most patients with a score of 2 or less will not have CAD), and use of the MHS when evaluating primary care patients with chest pain has been shown to improve clinical diagnostic accuracy. Recent studies have demonstrated that the presence or absence of typical cardiac risk factors (eg, diabetes, hypertension, smoking, high cholesterol, or family history) have little diagnostic value for determining the likelihood of ACS in patients over age 40.

An ECG should be obtained promptly for any patient with suspected ACS. ST-segment elevation in two or more contiguous leads, or presumed new left bundle branch block, is diagnostic of ST segment elevation Type I MI (STEMI) and requires urgent revascularization with thrombolysis or angioplasty at an appropriate facility. Ischemic ST-segment

BOX 1 Clinical Prediction Rules for Evaluating Patients with Chest Pain*

Acute Coronary Syndrome (ACS)

- Diagnosing cardiac ischemia with Exercise Treadmill Testing: Duke treadmill score: <https://www.mdcalc.com/duke-treadmill-score>
- What is the risk of a major cardiac event in the next 6 weeks? HEART Score: <https://www.mdcalc.com/heart-score-major-cardiac-events>.
- What is the risk of ACS in someone with chest pain? Marburg Heart Score: <https://www.mdcalc.com/marburg-heart-score-mhs>
- Assesses long-term risk in those with ACS: GRACE ACS risk calculator: <http://www.outcomes-umassmed.org/grace/>
- Estimates mortality for patients with unstable angina and non-ST elevation MI: TIMI risk score: <http://www.timi.org/>

Pneumonia

- Diehr diagnostic rule for pneumonia in adults with acute cough: <http://www.soapnote.org/infectious/diehr-rule/>

Pulmonary Embolism

- Geneva score for predicting risk of pulmonary embolism: <https://www.mdcalc.com/geneva-score-revised-pulmonary-embolism>
- Pisa clinical model for predicting the probability of PE: <https://ebmcalc.com/PulmonaryEmbRiskPisaCXR.htm>
- Wells scoring system for risk of pulmonary embolism: <https://www.mdcalc.com/wells-criteria-pulmonary-embolism>

Thoracic Aortic Dissection

- Aortic Dissection Detection Risk Score (ADD-RS): <https://www.mdcalc.com/aortic-dissection-detection-risk-score-add-rs>

TABLE 1 Marburg Heart Score

FINDING	POINTS
Woman > 64 years, man > 54 years	1
Known CAD, cerebrovascular disease, or peripheral vascular disease	1
Pain worse with exercise	1
Pain not reproducible with palpation	1
Patient assumes pain is cardiac	1

Abbreviation: CAD = coronary artery disease.

Approximately 97% of patients with a MHS score of 2 or less will not have CAD. Approximately 23% of patients with a MHS score of 3 or more will have CAD.

Adapted from: Haasenritter J, Bösner S, Vaucher P, Herzig L, Heinzl-Gutenbrunner M, Baum E, Donner-Banzhoff N. Ruling out coronary heart disease in primary care: external validation of a clinical prediction rule. *Br J Gen Pract*. 2012 Jun;62(599):e415-21. doi: 10.3399/bjgp12X649106. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3361121/>.

depression more than 0.5 mm, dynamic T-wave inversion with chest discomfort, or transient ST-segment elevation of 0.5 mm or more are classified as unstable angina or non-ST segment elevation Type I MI (NSTEMI). However, none of these findings is sensitive enough that its absence can exclude MI, and patients whose chest pain is not low risk still require further assessment for ACS.

In patients with high-risk chest pain who do not have STEMI, elevated cardiac biomarkers distinguish NSTEMI from unstable angina. Cardiac troponins T and I are more sensitive for detecting NSTEMI than creatine kinase (CK) or the MB isoform (CK-MB). High-sensitive troponins will be coming into use with an even greater sensitivity (negative predictive values = 99.5%).

Initial management of NSTEMI includes hospital admission for antiplatelet, antithrombin, and antianginal therapy. Patients with unstable angina should also be hospitalized for observation, and patients with either NSTEMI or unstable angina require risk stratification using the TIMI or GRACE risk scores (see **Box 1**). Patients with chest pain suspicious for CAD, but no definite initial diagnosis of STEMI or NSTEMI on initial presentation, are often admitted to hospital for overnight observation and serial cardiac biomarker measurements at 6 and 12 hours after symptom onset to “rule out MI.” However, use of the HEART, TIMI, or GRACE scores may help determine who is at sufficiently low risk of major adverse cardiac events to allow discharge for close follow-up, and recent evidence suggests that the HEART score may outperform TIMI and GRACE in determining which patients are low risk. (see **Box 1**).

Patients at low risk for ACS or MI can usually defer further testing unless there are other risk factors in their family or past medical history markedly increasing the likelihood of CAD. Current recommendations are that all other patients with chest pain suggesting CAD should have further noninvasive testing within 7 days; however, a recent study of 4181 patients in an ED chest pain unit found that a reasonable alternative may be to test troponin levels twice over a 6-hour interval, with no stress testing done if both values are normal. Patients who can exercise and who have no left-bundle branch block, preexcitation, or significant resting ST depression on a resting ECG can be evaluated with an exercise stress ECG, and the Duke treadmill score can then be used to further quantify cardiac risk (see **Box 1**). Patients with baseline ECG abnormalities should have perfusion imaging performed, along with a stress ECG, and patients who cannot exercise may be evaluated with a pharmacologic stress or vasodilator test (e.g., dobutamine [Dobutrex]¹ or adenosine [Adenocard]). Patients at high risk for CAD or those with NSTEMI should generally proceed directly to angiography, which allows definitive assessment of coronary artery anatomy.

Pulmonary Embolism

There are no individual symptoms or physical examination findings that reliably diagnose or exclude pulmonary embolism (PE), but three clinical prediction rules have all been validated for use in determining likelihood of PE and therefore whether further testing is needed (**Box 1**). While the Pisa rule may be the most accurate, it is also the most mathematically complicated and depends on clinical, ECG, and x-ray findings. The Geneva rule requires blood gas and chest x-ray findings. The Wells rule (**Table 2**) is based on the simplest

TABLE 2 Simplified Wells Scoring System for Pulmonary Embolism

CLINICAL FINDING	SCORE
Symptoms of deep vein thrombosis (DVT)	3.0
No alternative diagnosis more likely than PE	3.0
Heart rate > 100 bpm	1.5
Immobilization greater than 3 days or surgery in past 4 weeks	1.5
Previous objectively diagnosed DVT or PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Probability of PE: < 2 points = low, 2–6 points = moderate, > 6 points = high.	

Abbreviations: DVT = deep vein thrombosis; PE = pulmonary embolism.

Adapted from: Miniati M, Bottai M, Monti S. Comparison of 3 clinical models for predicting the probability of pulmonary embolism. *Medicine (Baltimore)* 2005;84:107–114; Torbicki A, Perrier A, Konstantinides S, et al, ESC Committee for Practice Guidelines (CPG): Guidelines on the diagnosis and management of acute pulmonary embolism. *Eur Heart J* 2008;29:2276–315. <http://eurheartj.oxfordjournals.org/content/29/18/2276.long>.

¹Not FDA approved for this indication.

combination of history and examination findings, and a comparison between the Wells and Geneva rules found the Wells rule has a lower failure rate. With the Wells and Geneva prediction rules, the likelihood of PE is approximately 10% in the low-probability category, 30% in the moderate-probability category, and 65% in the high-probability category. Routine tests done for patients with chest pain are not particularly helpful in diagnosing or excluding PE. The chest x-ray may be abnormal, but findings that typically occur with PE (atelectasis, effusion, or elevation of a hemidiaphragm) are nonspecific. ECG signs of right ventricular strain (S wave in lead I, Q wave and inverted T wave in lead III) may be helpful if present, but their absence does not exclude PE. Hypoxia may be present, but up to 20% of patients with PE have a normal alveolar-arterial oxygen gradient.

Additional tests recommended for evaluating patients with suspected PE include D-dimer testing by enzyme-linked immunosorbent assay (ELISA; sensitivity > 95%; specificity, approximately 40%) or latex agglutination (sensitivity 85%–90%), compression ultrasonography, ventilation–perfusion scintigraphy, or computed tomography (CT) angiography. Patients with suspected high-risk PE (i.e., those with shock or hypotension) should have immediate CT angiography and treatment for PE if the CT is positive, although an echocardiographic finding of right ventricular overload may be used to justify treatment for PE in the unstable patient with high clinical suspicion for PE when CT angiography is not available. Patients who have suspected PE and who are not at high risk (i.e., no shock or hypotension) and who have a high clinical probability (based on Wells, Geneva, or Pisa scoring) should also proceed directly to CT, with appropriate treatment if the scan is positive. Patients with low clinical probability of PE (based on one of the validated clinical prediction rules) do not need further D-dimer testing or imaging. Patients with intermediate clinical probability should initially have D-dimer testing; further testing or treatment for PE is unnecessary if the D-dimer is negative, and CT angiography should be performed if the D-dimer is positive.

Anticoagulation, with thrombolysis in high-risk patients, is the foundation of treatment for PE. Patients with shock or hypotension are at high risk and require hemodynamic and respiratory support, thrombolysis, or embolectomy, and then appropriate attention to anticoagulation. Normotensive patients with echocardiographic evidence of right ventricular dysfunction or serologic evidence of myocardial injury (elevated troponins or CK-MB) have intermediate risk and should be admitted for anticoagulation. Normotensive patients who have normal results on echocardiography and testing for myocardial injury are at low risk and often may be discharged early for management at home after initiation of anticoagulation. Anticoagulation for PE should be started at the time of diagnosis with unfractionated heparin, low-molecular-weight heparin, or fondaparinux (Arixtra) and continued for at least 5 days, with simultaneous initiation of oral anticoagulation with warfarin (Coumadin), titrated to maintain an international normalized ratio (INR) between 2.0 and 3.0. The newer anticoagulants dabigatran (Pradaxa), rivaroxaban (Xarelto), or apixaban (Eliquis) may be an option for patients who have difficulty maintaining steady anticoagulation with warfarin.

Thoracic Aortic Dissection

Thoracic aortic dissection is a much less common cause of chest pain; prevalence estimates are 2 to 3.5 cases per 100,000 person-years. Up to 40% of patients die immediately, and 5% to 20% die during or shortly after surgery. Risk factors for acute thoracic aortic dissection include hypertension, presence of a pheochromocytoma, cocaine use, weight lifting, trauma or a rapid deceleration event, coarctation of the aorta, and certain genetic abnormalities. Pain due to acute aortic dissection is perceived as abrupt and severe in 84% to 90% of cases, and more than 50% of patients describe the pain as sharp or stabbing. No physical findings are sensitive or specific for detecting aortic dissection, because approximately equal percentages of patients are hypertensive, normotensive, or have hypotension or shock, and the

most common physical findings (a murmur of aortic insufficiency or a pulse deficit) occur in less than half of patients.

In any patient with severe chest pain that is abrupt or instantaneous in onset or has a ripping, tearing, or stabbing quality, acute thoracic aortic dissection should be suspected. Physical examination should assess for a pulse deficit, a systolic pressure differential between limbs of greater than 20 mm Hg, a focal neurologic deficit, or a new aortic regurgitation murmur. It is also important to ask about a family history of connective tissue disease (including Marfan syndrome), about any family or personal history of aortic dissection or thoracic aneurysm, and about any known aortic valve disease or recent aortic interventions. D-dimer testing has been proposed as a way to screen for aortic dissection, but it is more important to obtain prompt imaging and surgical intervention for those in whom dissection is confirmed. A low-risk Aortic Dissection Detection Risk Score (ADD-RS) can help exclude the diagnosis of aortic dissection, while patients with a high-risk score require further evaluation.

An ECG should be obtained in all patients with suspected aortic dissection to exclude STEMI (which can manifest with similar symptoms). In all low- and intermediate-risk patients, a prompt chest x-ray can help by either confirming an alternative diagnosis or confirming the presence of thoracic aortic disease. High-risk patients should have prompt imaging with CT or magnetic resonance imaging (MRI), and those who are in shock or clinically unstable may be evaluated by bedside transesophageal echocardiography. If thoracic aortic dissection is confirmed on imaging, urgent surgical consultation is required. Medical therapy should be started with intravenous β -blockers. Patients with dissection of the ascending aorta require urgent surgery, whereas those with descending thoracic aortic dissection may be managed medically unless hypotension or other complications develop.

Esophageal Rupture

Esophageal rupture, or Boerhaave's syndrome, has a high mortality rate. Esophageal rupture is rare, and the most common cause is endoscopically induced injury, but it can happen in other settings as well. Common misdiagnoses include perforated ulcer, MI, PE, dissecting aneurysm, and pancreatitis. The "classic" presentation has been described as pain, dyspnea, and shock followed by forceful emesis, but history and physical are commonly nonspecific. Diagnosis most commonly is made by contrast esophagram or CT scan of the chest.

Patients whose rupture is diagnosed less than 48 hours after symptom onset should be treated surgically (especially if sepsis is present) or endoscopically. Those who present more than 48 hours after symptom onset may be considered for conservative treatment with hyperalimentation, antibiotics, and nasogastric suction.

Tension Pneumothorax

Tension pneumothorax is relatively rare among patients presenting with chest pain, but it is potentially life-threatening if not treated properly. Common symptoms and physical findings include chest pain, respiratory distress, decreased ipsilateral air entry, and tachycardia; hypoxia, tracheal deviation, and hypotension are less common.

Emergency needle decompression is usually recommended if tension pneumothorax is suspected, but this is ineffective in some cases and is associated with risks to the patient of pain, bleeding, infection, and cardiac tamponade. However, waiting for radiographic confirmation of the diagnosis is associated with up to a four fold increase in mortality due to delay in decompression of the pneumothorax. Patients most likely to benefit from an immediate attempt at needle decompression are those with an oxygen saturation < 92% while on oxygen, a decreased level of consciousness while on oxygen, a systolic blood pressure less than 90 mm Hg, or a respiratory rate < 10. Patients without these signs of instability may be better managed by waiting for chest imaging to confirm or exclude the presence of a pneumothorax. Although plain chest x-rays have typically been used for diagnosing a pneumothorax, one study has found that pleural ultrasound may have higher sensitivity.

TABLE 3 Diehr Diagnostic Rule for Pneumonia in Adults with Acute Cough

SIGNS AND SYMPTOMS	
FINDING	POINTS
Rhinorrhea	-2
Sore throat	-1
Night sweats	1
Myalgia	1
Sputum all day	1
Respiratory rate > 25	2
Temperature > 100°F	2
Interpretation	
Score	Probability of Pneumonia
-3	5
-1	12
0	21
1	30
3	37

Adapted from Cayley Jr WE. Diagnosing the cause of chest pain. *Am Fam Physician* 2005;72:2012–2021. <http://www.aafp.org/afp/2005/1115/p2012.html>.

Other Causes of Chest Pain

A patient with chest pain and cough, fever, egophony, or dullness to percussion might have pneumonia, but none of these individual findings are specific enough to confirm the diagnosis. A large study in 1984 developed a decision rule (Table 3) using seven clinical findings to predict the likelihood of pneumonia. Although there is ongoing debate over the reliability of pneumonia diagnosis based solely on history and physical examination and chest x-ray is usually considered the reference standard, a recent Cochrane review found two trials suggesting routine chest radiography does not affect the clinical outcomes in adults and children presenting suggestive of a lower respiratory tract infection. Thus, at least for clinically stable outpatients, treatment for pneumonia based on appropriate clinical findings alone may be reasonable.

Heart failure alone is an uncommon cause of chest pain, but it may accompany ACS or cardiac valve disease. A displaced apical impulse and a prior history of CAD support this diagnosis, and because virtually all patients with heart failure have exertional dyspnea, its absence is very helpful in excluding this diagnosis. An abnormal ECG and cardiomegaly on chest x-ray can increase the likelihood of heart failure among patients with chest pain, and B-natriuretic peptide (BNP) is now commonly used for detecting heart failure in patients presenting with acute dyspnea. One recent study also found that point-of-care ultrasound (POCUS) may have high sensitivity and specificity for diagnosing acute cardiogenic pulmonary edema.

A 3-item questionnaire has been developed specifically to assess for panic disorder among patients with chest pain referred for cardiac evaluation (Table 4), and there is good evidence that psychological interventions such as cognitive behavioral therapy (CBT) and breathing exercises can improve chest pain symptoms for patients with nonspecific chest pain who do not have CAD. However, even in patients with possible panic disorder, further cardiac testing should be done if there are significant cardiac risk factors.

Gastrointestinal disease can cause chest pain, but the history and physical examination are relatively inaccurate for diagnosing or excluding serious gastrointestinal pathology. However, if life-threatening cardiovascular or pulmonary causes of chest pain have been excluded, it is appropriate to try a short course of high dose PPI (omeprazole

TABLE 4 Evaluation for Chest Pain from Panic Disorder

ITEM	0	1	2	3	4	5
When you are nervous, how often do you think “I am going to pass out?”	Never	Rarely	Half the time	Usually	Always	
During the last 7 days, including today, how much have you been bothered by pains in the chest?	Not at all	A little bit	Moderately	Quite a bit	Extremely	
To what degree is your chest pain tiring or exhausting	None	Mild		Moderate	Severe	

Adapted from: Dammen T, Ekeberg O, Arnesen H, Friis S. The detection of panic disorder in chest pain patients. *Gen Hosp Psychiatry*. 1999 Sep-Oct;21(5):323–332. PubMed PMID: 10572773. <http://www.ncbi.nlm.nih.gov/pubmed/10572773>.

Approximately 76% of patients with a score of 4 or less will not have panic disorder (PD).

Approximately 71% of patients with a score of 5 or more will have PD.

[Prilosec] 40 mg twice daily³, lansoprazole [Prevacid] 30 mg daily, or esomeprazole [Nexium] 40 mg twice daily³) to evaluate for undiagnosed gastroesophageal reflux disease (GERD) as the cause of chest pain (even for patients without typical GERD symptoms.)

Chest wall pain can usually be diagnosed by history and examination if other etiologies have been excluded, and chest wall pain is more likely if the patient’s pain is reproducible by palpation. Measurement of the sedimentation rate is not generally helpful in making the diagnosis, although in unusual situations radiography may be helpful.

References

- Cao AM, Choy JP, Mohanakrishnan LN, Bain RF, van Driel ML: Chest radiographs for acute lower respiratory tract infections, *Cochrane Database Syst Rev* 12, 2013 Dec 26. CD009119.
- Cayley Jr WE: Chest pain—tools to improve your in-office evaluation, *J Fam Pract* 63(5):246–251, 2014 May.
- Cayley Jr WE: Diagnosing the cause of chest pain, *Am Fam Physician* 72:2012–2021, 2005.
- de Schipper JP, Pull ter Gunne AF, Oostvogel HJ, van Laarhoven CJ: Spontaneous rupture of the oesophagus: Boerhaave’s syndrome in 2008. Literature review and treatment algorithm, *Dig Surg* 26:1–6, 2009.
- Harskamp RE, Laeven SC, Himmelreich JC, et al: Chest pain in general practice: a systematic review of prediction rules, *BMJ Open* 27;9(2):e27081, 2019.
- Hendriks JM, Geersing GJ, Lucassen WA, et al: Diagnostic prediction models for suspected pulmonary embolism: systematic review and independent external validation in primary care, *BMJ* 8;351:h4438, 2015.
- Hiratzka LF, Bakris GL, Beckman JA, et al: 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, *Circulation* 121:e266–e369, 2010. Erratum in *Circulation* 2010;122(4):e410.
- Kisely SR, Campbell LA, Yelland MJ, Paydar A: Psychological interventions for symptomatic management of nonspecific chest pain in patients with normal coronary anatomy, *Cochrane Database Syst Rev* 30(6):CD004101, 2005.
- Leigh-Smith S, Harris T: Tension pneumothorax—time for a re-think? *Emerg Med J* 22:8–16, 2005.
- McConaghy JR, Oza RS: Outpatient diagnosis of acute chest pain in adults, *Am Fam Physician* 87(3):177–182, 2013 Feb 1.
- Nienaber CA, Clough RE: Management of acute aortic dissection, *Lancet* 385(9970):800–811, 2015.

³Exceeds dosage recommended by the manufacturer.

O'Connor RE, Al Ali AS, Brady WJ, Ghaemmaghami CA, Menon V, Welsford M, et al: Part 9: acute coronary syndromes: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care, *Circulation* 132(18 Suppl. 2):S483–S500, 2015.

Raja AS, Greenberg JO, Qaseem A, Denberg TD, Fitterman N, Schuur JD: Clinical Guidelines Committee of the American College of Physicians. Evaluation of patients with suspected acute pulmonary embolism: best practice advice from the clinical guidelines committee of the American College of Physicians, *Ann Intern Med* 163(9):701–711, 2015.

Reinhardt SW, Lin CJ, Novak E, Brown DL: Noninvasive Cardiac Testing vs Clinical Evaluation Alone in Acute Chest Pain: A Secondary Analysis of the ROMICAT-II Randomized Clinical Trial, *JAMA Intern Med.* 178(2):212–219, 2018 Feb 1.

Swap CJ, Nagurny JT: Value and limitations of chest pain history in the evaluation of patients with suspected acute coronary syndromes, *JAMA* 294:2623–2629, 2005.

Wong BC: Is proton pump inhibitor testing an effective approach to diagnose gastroesophageal reflux disease in patients with noncardiac chest pain?: a meta-analysis, *Arch Intern Med.* 2005 Jun 13;165(11):1222–8.

CONSTIPATION

Method of

Melissa Gaines, MD



CURRENT DIAGNOSIS

- While constipation is a benign process, it is important to recognize signs of a serious condition.
- Classification of normal transit constipation, slow transit constipation, or pelvic floor dysfunction guides therapy.
- Clinical testing has low benefit, but colonoscopy or imaging can assess for organic causes.



CURRENT THERAPY

- Initial therapy includes soluble dietary fiber to improve symptoms in chronic constipation.
- Osmotic laxatives are preferred while using stimulant laxatives as rescue agents.
- Surgery is reserved for pelvic floor dysfunction after optimal therapies have failed.

Epidemiology

Constipation is common, with a prevalence of 1.9% to 27.2%, but the description of symptoms is variable. A thorough history and focused physical examination aids diagnosis. Treatment is directed toward relief of symptoms, alleviation of precipitating factors, and prevention of recurrence. While constipation is a benign process, it is important to recognize concerning signs for a more serious medical condition such as malignancy.

Risk Factors

Vulnerable populations include female, elderly, neurodegenerative disease, low-fiber diet, painful rectal disorders, hypothyroidism, and diabetes mellitus.

Pathophysiology

With aging, there is decreased rectal compliance, diminished rectal sensation, and decreased resting anal pressures, while colonic transit time is preserved. Normal transit constipation is a component of irritable bowel syndrome with normal transit time and stool frequency. Slow transit constipation is a condition with colonic dysmotility resulting from altered enteric nervous system. Defecatory disorders include structural disturbances of the pelvic floor. Pelvic floor dysfunction is the paradoxical contraction of the external anal sphincter and puborectalis muscles during defecation. Secondary causes of constipation are listed in [Table 1](#).

Prevention

Provide an environment of privacy and comfort to allow for natural defecation. Prescribe an adequate fluid and fiber intake with specific amounts that vary depending on the patient's condition. Encourage physical activity, with a low to moderate level of exercise depending on the patient's functional status. Develop a routine for defecation with a prompt response to a call to defecate urgently. Recurrent fecal impaction can be prevented with polyethylene glycol (PEG, MiraLax).

Clinical Manifestations

Patients will complain using qualitative terms of hard stools, a feeling of incomplete voiding, straining, prolonged time for laxation, the need for additional maneuvers, abdominal bloating, and abdominal pain ([Table 2](#)). A change in bowel habit differentiates the current complaint from a serious medical condition. Red flags include acute onset, weight loss, abdominal pain or cramping, rectal bleeding, nausea or vomiting, rectal pain, fever, or a change in stool caliber. Infants with abdominal distension and failure to pass meconium within 24 hours indicate Hirschsprung's disease. Patients should be asked if they have had loose stools or bowel incontinence to assess for fecal impaction. A medication review is required (see [Table 1](#)). Classification of patients with normal transit constipation, slow transit constipation, or pelvic floor dysfunction/defecatory disorders guides therapy.

Diagnosis

Diagnostic criteria have been established because the symptoms can vary (see [Table 2](#)). Conduct a physical examination that includes an assessment of vital signs, weight, volume status, auscultation of bowel sounds, abdominal percussion for tympani, and abdominal palpation for tenderness or mass. A rectal examination can detect resting rectal tone, fecal impaction, anorectal disorders, or rectal mass. Defecatory disorders show an increased resistance to the insertion of the examiner's finger with an impaired relaxation of the sphincter complex and reduced perineal descent during a Valsalva maneuver. Conduct laboratory testing on electrolytes, hemoglobin, thyroid-stimulating hormone, and fecal occult after initial measures fail. Red flag

TABLE 1 Causes of Constipation

Dietary	Low-fiber diet, dementia, depression, anorexia, dehydration
Metabolic	Diabetes mellitus, hypercalcemia, hypokalemia, hypothyroidism, systemic sclerosis
Neurologic	Parkinson's disease, spinal cord disorder, multiple sclerosis, cerebrovascular disease (stroke)
Iatrogenic	Antacids, iron, anticholinergics, antidepressants, antipsychotics, opiates, antiepileptics
Painful anorectal condition	Anal fissure, hemorrhoids, abscess, fistula, pelvic floor dysfunction, malignancy

TABLE 2 Rome III Criteria

More than two present: <ul style="list-style-type: none"> • Straining in more than 25% of defecations • Hard or lumpy stool in more than 25% of defecations • Sensation of incomplete evacuation • Sensation of anorectal blockage • Manual maneuvers • Fewer than three defecations per week
Loose stools are rare without laxative use
Insufficient criteria for irritable bowel syndrome
Symptoms for 3 months with onset 6 months before diagnosis