Gastrointestinal (GI) illnesses are common in athletes. Various causes include adverse physiologic adaptations of the gut during exercise; excess ingestion of carbohydrate drinks, alcohol, and anti-inflammatory medications; emotional stressors; exposure to pathogens in closed environments and during travel; trauma; and abdominal wall pressure overload. Commonly encountered conditions in this population include self-limited food poisoning, gastroesophageal reflux disease (GERD), hiatal hernia, irritable bowel syndrome (IBS), and viral gastroenteritis. More serious conditions such as Crohn’s disease (CD), ulcerative colitis (UC), appendicitis, mesenteric adenitis, and invasive diarrhea are far less common in this population and generally present dramatically with abdominal pain, nausea, vomiting, and bloody diarrhea. Vigilance for such conditions is essential; however, timely diagnosis and treatment can be delayed as most athletes have enhanced pain tolerance, often do not verbalize their symptoms in a timely manner, and frequently indulge in self-medication prophylactically and therapeutically.

Unfortunately, evidence-based management of GI illnesses in athletes is limited because most studies have compared various GI illnesses between different sports, rather than comparing athletes to nonathletes. This article reviews the evidence that is available specifically relating to etiology, pathophysiology, clinical presentation, relevant differential diagnoses, acute management, and recommendations for specialist consultation of various GI illnesses in the training room setting.
Regular, moderate physical activity promotes several health benefits for the GI tract. Physiologic digestive processes, such as gut emptying, are enhanced [1] and there is a reduced incidence of liver disease [2], cholelithiasis [3], colon cancer [2], and constipation [4,5]. Conversely, frequent, high-intensity exercise causes undesirable symptoms such as heartburn, chest pain, bloating, belching, nausea, vomiting, abdominal cramps, frequent urge to defecate, diarrhea, and constipation. These symptoms are reported by approximately 20% to 50% of high-performance athletes, are more commonly encountered in women than men, are more common in younger athletes, and are less frequent in sports that incorporate more gliding movements, such as cycling [1,5–8]. However, there is a lack of difference between the number of reflux episodes, gastric emptying (GE), transit time, and intestinal permeability and glucose absorption between runners and cyclists at rest [8]. Therefore, exercise-induced GI distress is not a result of a particular GI phenotype that makes certain individuals more susceptible to the plethora of undesirable symptoms. Although the precise mechanisms for the various GI symptoms are poorly understood and studied [9], several theories and a few studies have postulated the potential detrimental alterations in GI function commonly encountered in competitive athletes.

For example, gut ischemia secondary to attenuated GI blood flow [8,10] is a well-defined exercise adaptation that redirects blood flow to active muscles and the pulmonary vasculature [11]. Exercise typically reduces GI blood flow in excess of 50% [12], which makes the gut mucosa susceptible to ischemic injury [13]. Mucosal injury increases gut mucosal permeability, enhances occult blood loss, and induces translocation of protective bacterial flora and generation of endotoxins that can induce diarrhea [8,14]. Other suggested mechanisms for increased GI distress include increased vibration of the abdominal wall and bouncing of the organs, especially in runners [6,8] and various neuroimmunoendocrine adaptations to exercise. Increased catecholamines and several GI peptides, including gastrin, motilin, secretin, peptide histidine-methionine, and vasoactive intestinal peptide, result in increased gut transit time that negatively affects GI homeostasis [5,8,11,15].

Additionally, wide-ranging fluid intake and dietary patterns through largely hormonal mechanisms can further alter homeostasis of the GI tract. For example, GE is slowed by high-intensity exercise [2,16,17] following consumption of beverages in a dehydrated state [18], and is independent of either training status [19] or gut blood flow and plasma volume [20]. The excitement of competition elevates circulating catecholamines that are known to suppress thirst [21,22]. Furthermore, athletes are frequently averse to the unpleasant sensation of a full stomach during competition. Therefore, it is not uncommon to delay hydration during competition, which in turn may expedite fluid and electrolyte imbalance.

GI distress secondary to slowed GE [20] has been reported by up to 40% of dehydrated athletes who consumed a 7% carbohydrate beverage, as compared to euhydrated counterparts [18]. GE can be further slowed by the consum-
tion of more palatable hypertonic carbohydrate beverages after 20 minutes of running [23]. Conversely, excessive fluid intake that produces shifts in osmotic pressure [11], and excessive consumption of food rich in fat, protein, and fiber in proximity to the onset of competition can also promote undesirable GI symptoms [24]—an effect that is magnified by increased postprandial motor activity [25] and enhanced gut transit time observed following physical activity [8,26].

In summary, various physiologic adaptations to exercise make competitive athletes more susceptible to GI distress.

**DYSPHAGIA**

The swallowing mechanism is a complex process involving several closely coordinated voluntary and reflexive actions within the oropharynx and esophagus. Dysphagia, a common swallowing disorder in the athletic population, is most commonly caused by acid reflux. However, it is important to recognize and expeditiously treat other potentially serious yet rarer causes of dysphagia.

A careful history is crucial to diagnosis and can determine the cause of dysphagia in 80% of cases [27]. Although athletes may present with a spectrum of complaints, a few pivotal elements of a detailed history allow anatomic classification of dysphagia into two subclasses: oropharyngeal or esophageal. First, does the dysphagia occur with liquids, solids, or both? Second, does the difficulty swallowing occur soon after the initiation of deglutition or is it delayed? Finally, is dysphagia painful or painless, and if painful, does the pain during deglutition localize to the neck or the chest?

Oropharyngeal dysphagia (OD) typically presents with greater difficulty swallowing liquids compared with solids; is characterized by repeated attempts to swallow secondary to a choking sensation or a sensation of incomplete swallowing; occurs within the first second of deglutition; and presents with odynophagia that localizes to the neck. Other characteristics may include either a change in dietary habits, dehydration, regurgitation, or unexplained weight loss.

Commonly encountered causes for painful OD include tonsillitis, pharyngitis, laryngitis, cervical adenitis, and thyromegaly. Other less common causes of OD include Ludwig’s angina, retropharyngeal abscess, or anaphylaxis resulting from insect sting, drug and food allergies, or exercise.

Unlike painful OD that is induced by inflammation or mechanical pressure, painless OD is caused by disordered neuromuscular functioning of the pharynx and upper esophageal sphincter. Causes in athletes are rare and include Guillain-Barre syndrome (which should be suspected in an athlete presenting with painless OD and respiratory distress following influenza vaccination), globus hystericus as a manifestation of GERD, and laryngeal cancer in athletes who have a history of chewing tobacco abuse.

In contrast to OD, esophageal dysphagia (ED) presents with difficulty swallowing solids and liquids, occurs several seconds after swallowing, and is characterized by odynophagia that localizes to the sixth thoracic dermatome.
Frequent complaints include an ill-defined substernal pressure and burning, and in severe cases, frank chest pain.

ED occurs with various mechanical and inflammatory disorders affecting the esophageal body or its peristaltic function. Common causes of painful ED in athletes include esophagitis induced by acid reflux [8,29] or, less commonly, infections such as herpes simplex virus, cytomegalovirus, and candida in immunocompromised individuals. Other causes include caustic injury to the lower esophagus from bilious vomiting, or pill esophagitis following treatment with tetracyclines, nonsteroidal anti-inflammatory drugs (NSAIDs), or alendronate. Acid reflux may also induce diffuse esophageal spasm (DES) that presents with symptoms similar to GERD and pill esophagitis, including noncardiac chest pain, globus, and regurgitation. Other less common, yet important, causes of esophageal dysphagia in the athletic population include aortic aneurysm in athletes who have Marfanoid features; mechanical obstruction of the esophagus from strictures, webs, or malignancy; or motility disorders, such as achalasia or scleroderma.

Persistent dysphagia necessitates further workup and specialist evaluation. Persistent noncardiac chest pain warrants a definitive diagnosis. Concomitant anemia, unexplained weight loss, anorexia, and nontender cervical or supraclavicular lymphadenopathy [28] could suggest occult GI malignancy [30].

**GASTROESOPHAGEAL REFLUX DISEASE**

Symptomatic reflux of gastric contents into the esophagus is encountered in approximately 60% of athletes and occurs more frequently during exercise than at rest [31,32]. Exercise not only exacerbates GERD, as demonstrated in studies that used ambulatory pH monitoring [33–35], but is also a contributing factor to reflux in healthy volunteers [32]. And although proton pump inhibitor therapy reduced acid reflux in runners, symptoms of heartburn, chest pain, and regurgitation were omnipresent, suggesting a multifactorial cause of GERD during exercise [36].

Although the precise mechanisms are not well defined, either the isolated effects or combination of several physiologic alterations contribute to acid reflux during exercise. Suggested mechanisms include gastric dysmotility [29]; relaxation of the lower esophageal sphincter (LES) [8]; enhanced pressure gradient between the stomach and esophagus [32]; gastric distension [24]; delayed gastric emptying [8,20] (especially in a dehydrated state [18]); enhanced intra-abdominal pressure in sports such as football, weight lifting, and cycling [32]; and increased mechanical stress by bouncing of organs [8].

The duration, amplitude, and frequency of esophageal contractions decline with exercise intensity at or above 90% of VO\(_2\)max, and the number of gastroesophageal reflux episodes and the duration of esophageal acid exposure are correspondingly increased during exercise at 90% VO\(_2\)max. Additionally, increase in reflux episodes has been observed despite a lack of differences in esophageal peristaltic velocity and pressure in symptomatic and asymptomatic cyclists at 70% of VO\(_2\)max. These data suggest that an increased number of LES
relaxations may contribute to increased reflux during exercise [8]. Thus, exercise has profound effects on acid reflux that are intensity-dependent [29].

The degree of acid reflux is also sports-specific. Collings and colleagues [32] demonstrated that athletes involved in predominantly anaerobic sports, such as weight lifters, experience the most heartburn and reflux. In the same study, runners had mild symptoms and moderate reflux and cyclists had mild symptoms and mild reflux compared with weight lifters. In support, Peters and colleagues [10] showed that although distance runners are prone to acid reflux, lower GI symptoms, such as the urge to defecate and development of diarrhea, are more prevalent (71%) than upper GI symptoms (36%). Cyclists exhibited varying degrees of upper (67%) and lower (64%) GI symptoms, and triathletes conformed to the patterns of GI distress during running and cycling.

Athletes who have exercise-related GERD may present with several symptoms that include substernal chest pressure, pain or burning that may mimic angina, sour taste, intermittent water brash, globus hystericus, eructation, nausea, and vomiting. A subset of athletes may present with atypical cough, hoarseness, and wheezing—symptoms that mimic either exercise-induced bronchospasm (EIB) or vocal cord dysfunction.

There is good success in treating GERD with either lifestyle modifications, medications, or both. Effective lifestyle modifications are a potent first-line treatment in athletes who have GERD. These include avoidance of laying down to sleep within 4 hours of the evening meal, postprandial exercise, and excessive consumption of foods that relax the LES, including chocolate, peppermint, onions, high-fat foods, alcohol, tobacco, coffee, and citrus products. Furthermore, sleeping on two pillows to enhance gravity-associated esophageal clearance reduces the symptoms of GERD. And finally, athletes treated with calcium-channel blockers for either migraine prophylaxis or control of hypertension should be counseled on the propensity for these drugs to reduce LES pressure and worsen GERD.

Medications used to treat GERD include H₂-receptor antagonists, proton pump inhibitors (PPIs), and promotility agents. Although PPIs are effective in treating GERD and endoscopic negative reflux disease (ENRD) [37], and have been shown to be therapeutically superior to H₂-receptor antagonists [37–40], the literature on the efficacy of medications used to treat GERD in athletes is sparse. In a single-blind, crossover study, Kraus and colleagues [33] showed that markers of exercise-induced gastroesophageal reflux—percentage reflux time and number of reflux episodes—can be reduced by pretreatment with an H₂-receptor antagonist (300 mg ranitidine) 1 hour prior to running. Van Nieuwenhoven and colleagues [8] reported no significant changes in either percentage reflux time or number of reflux episodes between asymptomatic and symptomatic cyclists exercising at 70% of VO₂max. However, there was a higher number and increased duration of reflux episodes in symptomatic individuals. Therefore, a future double-blind, randomized control study to compare the effects of acid suppression on exercise-induced gastroesophageal reflux should include asymptomatic and symptomatic athletes.
There are good results managing GERD with PPIs as first-line treatment. For persistent symptoms, the addition of a motility-influencing agent such as dicyclomine may be effective. Athletes frequently eat shortly before bedtime, a deleterious habit that can enhance postprandial reflux. Dicyclomine, in turn, has been shown to decrease early postprandial reflux episodes [41]. Furthermore, success with the use of dicyclomine in a subset of athletes lends support to the theory [8] that symptomatic athletes may indeed have a higher number of LES relaxations during exercise. Finally, gastroesophageal reflux can provoke or exacerbate EIB by either silent aspiration into the tracheobronchial tree or by stimulating an acid-mediated esophagobronchial reflex [42,43]. The investigators have thus used treatment with PPIs in a subset of athletes afflicted with GERD and EIB and noticed that it reduces symptoms of EIB.

**DYSPESPIA**

Dyspepsia refers to a spectrum of vague GI symptoms, including gnawing or burning epigastric pain, nausea, vomiting, eructation, bloating, indigestion, generalized abdominal discomfort, and early satiety. The three most common causes for dyspepsia include peptic ulcer disease, GERD, and gastritis. Other less common causes in this population include diabetes, thyroid disease, and lactose intolerance.

Regardless of the cause of dyspepsia, the common clinical feature is mucosal damage of the upper GI tract. As discussed elsewhere, gut mucosal injury occurs commonly in athletes secondary to complex physiologic adaptations that deplete gut blood flow during exercise [8,10–13]. As a result, mucosal ischemia depletes cellular ATP, expedites cell death, and induces mucosal inflammation [44]. Frequent dehydration, repeated stress of competition, and excessive use of NSAIDs, medications, alcohol, caffeine products, and dietary supplements containing amino acids and creatine may further exacerbate mucosal injury and eventually lead to blood loss and anemia.

Dyspepsia in athletes is easily preventable. However, dyspeptic symptoms are vague, and athletes frequently delay assessment and treatment. Lifestyle modifications akin to those used to treat GERD and avoidance of NSAIDs, caffeine, tobacco, gas-producing foods (eg, broccoli, beans, eggs [30]), and dairy products in athletes who have lactose intolerance generally helps alleviate dyspeptic symptoms. Rehydration with carbohydrate drinks during exercise may lessen mucosal damage by enhancing blood flow to absorption sites in the gut, thereby reducing dyspepsia [45].

Failure of conservative therapy warrants testing and treatment for *Helicobacter pylori* infection, especially in athletes who are from underdeveloped countries. Although eradication of *H pylori* does not have significant effects on gastroesophageal acid reflux in patients who have established GERD, symptoms of regurgitation have been shown to improve [46]. Therefore, a trial of either PPI or H2-receptor antagonists may be useful in conjunction with appropriate antibiotic therapy if the patient is *H pylori*-positive. Specialist referral and esophagogastroduodenoscopy (EGD) are warranted in the presence of
“alarm” symptoms and signs, such as odynophagia, dysphagia, hematochezia, melena, anemia, positive fecal occult bleeding, loss of appetite, and unexplained weight loss.

**MOTILITY DISORDERS**

**Diarrhea**

Distance runners and female athletes are most susceptible to lower intestinal motility disorders that include diarrhea [47,48] and irritable bowel syndrome (IBS) [8,10]. Although several theories have been proposed and a few studies have elucidated mechanisms, the literature is limited and the precise pathophysiology remains largely undefined in the athletic population. Possible causative factors include enteric fluid and electrolyte imbalance, mesenteric ischemia contributing to increased mucosal permeability [14], mechanical trauma [47], and altered colonic motility.

The data regarding the effects of exercise on colonic motility are varied. Although aberrant small intestine activity has been shown during exercise [4], physical activity does not have a well-defined effect on total transit time through the GI tract [25]. Some studies have shown that, by independent mechanisms, exercise can accelerate gut transit time of chyme and fecal residues [5] and intraluminal gas [26]. The transit of chyme and fecal residues is largely a passive process dependent on phasic motor activity and altered by exercise [49], whereas the transit of gas is influenced by visceral reflexes and involves contraction/relaxation of the abdominal wall [50,51]. Furthermore, running has been shown to delay small bowel transit time [1,8] and accelerate colonic transit time [8]. Other studies, however, have found that exercise does not alter small bowel or colonic motility [52], and transit time does not appear to be influenced by short bouts of high-intensity exercise and is independent of the mechanical bouncing of abdominal contents [53].

Fortunately, acute exercise-induced diarrhea, also known as “runner’s trots,” is considered physiologic diarrhea that does not result in dehydration or electrolyte imbalances and tends to improve with fitness level [1]. The diarrhea is defined as abnormally frequent, semisolid to watery bowel movements (>3 times per day) that can be acute (<4 weeks) or chronic (>4 weeks).

In healthy athletes, acute diarrhea is either induced by running, food poisoning, traveler’s diarrhea, or viral gastroenteritis [54]. Typically, the diarrhea is self-limited. In the absence of complications, such as fluid or electrolyte imbalance, protracted vomiting, melena, hematochezia, or frank bleeding per rectum, supportive management with intravenous fluids, rest, and bismuth is sufficient. Athletes who have viral gastroenteritis should temporarily be excluded from exposure to other teammates [54]. Avoidance of caffeine, NSAIDs, concentrated carbohydrate beverages, and foods with a high-glycemic index are also effective therapeutic interventions [30]. Antidiarrheal medications such as loperamide have adverse, depressive effects on the central nervous system, can affect heat dissipation and concentration [30], and should be used with caution.
Persistent diarrhea warrants a detailed workup to differentiate less serious causes from more severe infective and chronic causes. Although uncommonly encountered in athletes, infectious diarrhea must be ruled out if general supportive measures are inadequate. A history of a recent camping trip or well-water consumption could be suggestive of giardiasis. Athletes who have sickle cell trait or disease could have bloody diarrhea secondary to invasion with salmonella. Diarrhea associated with right lower quadrant abdominal pain that mimics appendicitis could be caused by *Yersinia enterocolitica*, and a history of bloody diarrhea following recent consumption of poorly cooked beef could suggest infection with hemorrhagic *Escherichia coli* (O157:H7). It is imperative to obtain stool cultures prior to instituting specific therapy because generic treatment with ciprofloxacin can prolong salmonella infection and increase the risk for hemolytic uremic syndrome in individuals who have hemorrhagic *E. coli* infection. Bloody diarrhea without an infectious cause, following the recent use of antibiotics, and associated with abdominal pain and fever is highly suggestive of *Clostridium difficile* colitis and should be aggressively treated with appropriate antibiotic therapy.

Chronic diarrhea in young athletes could be secondary to medications (e.g., laxative abuse, antibiotics), lactose intolerance, malabsorption, irritable bowel syndrome, hyperthyroidism, or inflammatory bowel disease (IBD). A stool osmolar gap readily distinguishes secretory diarrhea from osmotic diarrhea and is an especially pivotal component of the laboratory investigation for chronic diarrhea in a female athlete at risk for an eating disorder. Indications for specialist referral include chronic diarrhea secondary to malabsorptive syndromes, such as sprue, Whipple’s disease, and IBD.

**Irritable Bowel Syndrome**

A functional GI disorder that is devoid of either structural, biomechanical, radiologic or laboratory abnormalities, IBS affects up to 15% of the population [55]. Two forms have been described: “diarrhea-predominant” and “constipation-predominant.” IBS is twice as common in women and typically presents in the second or third decade of life. Approximately 50% of patients who have IBS have comorbid psychiatric illnesses, including depression and anxiety. Proposed mechanisms include increased motor reactivity to various stimuli, including stress, food, cholecystokinin, impaired transit of bowel gas [56,57], visceral hypersensitivity, impaired reflex control that delays gas transit [58], autonomic dysfunction, and altered immune activation [59].

The clinical characteristics are diverse, yet the most common symptoms include cramping abdominal pain relieved by defecation, altered stool frequency, altered stool form (mucus, watery, hard, or loose), altered stool passage (strain, urgency), a sense of incomplete evacuation, and abdominal distension especially following meals. Athletes who have IBS rarely have nocturnal symptoms and do not manifest systemic signs of illness or hematochezia. The differential diagnosis for IBS includes IBD, diarrhea secondary to sprue or lactose intolerance, thyroid dysfunction, laxative abuse, diabetes, and psychia-
tric illnesses. Current evidence does not support the routine use of laboratory studies and imaging to exclude organic GI disorders in patients who have typical IBS symptoms and are lacking “alarm” features [55].

Various lifestyle and dietary modifications are effective in treating IBS. Reassurance, stress reduction, consumption of small meals during the day, high-fiber diet, and avoidance of foods such as those containing lactose and candy that contains sorbitol are helpful first-line measures. The role of regular exercise in treating IBS has been studied in sedentary individuals who have IBS, and has been shown to be a fairly effective treatment modality [60].

Drug treatment of IBS is based on the athletes’ predominant symptoms. For diarrhea-predominant IBS, antidiarrheal (loperamide), antispasmodic (dicyclomine), and the 5-HT(3) receptor antagonist alosetron are efficacious. Loperamide does inhibit the absorption of various drugs, including warfarin, tetracyclines, NSAIDs, and thyroxine and should be used with extreme caution in athletes who have hepatic insufficiency secondary to mononucleosis. Alosetron is currently only approved for treatment of severe, chronic IBS. In a subset of patients who have diarrhea-predominant IBS and abdominal pain, tricyclic antidepressants such as amitriptyline are effective. For constipation-predominant IBS, fiber, bulk laxatives and stool softeners are used, but their efficacy is variable and may worsen symptoms. The 5-HT(4) receptor agonist tegaserod may be efficacious, but is approved only for women who have constipation-predominant IBS. Finally, the efficacy of psychologic counseling is, at best, doubtful [61].

**GASTROINTESTINAL BLEEDING**

The occurrence of exercise-induced GI bleeding has been well documented, particularly in distance runners [2,62–69]. Postexercise endoscopic colonic mucosal biopsy samples have revealed congestive and hemorrhagic vascular lesions [70,71]. Fortunately, GI bleeding following exercise is usually transient and occult; however, there are case reports documenting substantial upper and lower tract bleeds [5,63,66,72]. Ischemic colitis has been postulated to be a major cause of GI bleeding during and after intense exercise [73]. As blood is shunted from the viscera to the working muscles during maximal exercise, splanchnic blood flow is decreased by as much as 80% [74]. Postcompetition studies have supported this theory, as evidenced by the presence of endoscopic lesions consistent with transient ischemic damage caused by decreased blood flow with exercise [71]. Other proposed causes of exercise-induced GI bleeding include hemorrhagic colitis [72] or gastritis secondary to the frequent use of NSAIDs [62,75,76].

Other proposed mechanisms include hematuria [63], traumatic hemolysis [77], impaired gut absorption [8], iron loss in sweat [78], decreased iron absorption [79], poor dietary iron intake [79], and mechanical trauma [8] to the gut during running. The lower incidence of occult bleeding in cyclists and the absence of occult blood in walkers [62] support the theory that mechanical trauma to the gut from frequent jostling in runners contributes to occult blood loss. As a group,
runners have been shown to have lower ferritin, serum iron, and haptoglobin. The frequency of iron deficiency is related to the distance and the intensity of running [65]. 85% of ultramarathoners (100 miles) and triathletes [65] have been found to have occult blood positivity. It is more prevalent in younger athletes who have quicker times, and is more common during competition.

The differential diagnosis for GI bleeding is extensive, yet in young, healthy athletes the most common causes should be excluded prior to an extensive clinical investigation. GI bleeding is frequently occult and athletes typically present with dyspnea on exertion, fatigue, and decreased endurance and strength—anemic symptoms that may be compounded in female athletes who have heavier than usual menstrual cycles. Although hematemesis is rare in this population, prolonged retching can produce an esophageal “Mallory-Weiss” tear in the esophagus. Diarrhea associated with bright red blood per rectum or hematochezia warrants further workup for potentially serious conditions such as ulcerative colitis and Crohn’s disease. Other common causes of lower GI bleeding in athletes include hemorrhoids and anal fissures caused by frequent straining during defecation, especially in a subset of athletes who have chronic constipation.

Early recognition and diagnosis of occult bleeding is critical to prevention of poor athletic performance. Recommendations have been made for endoscopy prior to instituting therapy for GI symptoms and anemia in runners [71]. However, the investigators have had good success in managing anemia using a graded approach prior to routine endoscopy. Athletes who have fatigue, poor performance, and decreased ferritin are treated with iron supplementation containing at least 180 mg of elemental iron [69]. The investigators also counsel athletes on prophylactic measures, such as optimizing dietary fiber intake, maintaining optimal hydration that may decrease exercise-induced GI ischemia [80], discontinuing the regular use of NSAIDs [75,76], and, in individuals who have constipation and hemorrhoids, using stool softeners. The investigators do not use H₂ receptor blockers on a routine basis. The data on the prophylactic use of H₂ blockers (cimetidine) are varied [66,81–83], and only a subset of athletes may have reduced occult blood loss following the use of cimetidine prior to competition [66,68]. Failure to respond to these initial measures, despite normalization of ferritin, necessitates further clinical investigation and specialist referral to rule out other causes of iron deficiency anemia.

Specialist referral and diligent monitoring of various medications are critical to optimal health and athletic performance in athletes who have IBD. Current literature contains conflicting data regarding the effect of intense athletic participation in patients who have IBD. The general consensus suggests that light and moderate exercise is well tolerated and can benefit patients who have IBD [2]. Moreover, physical activity can counteract some of the adverse effects of medications used to treat IBD, such as steroid-induced muscle weakness and osteoporosis. Unfortunately, gut ischemia may exacerbate GI bleeding in athletes who have IBD. Therefore, a safe guideline to follow is to ensure hemodynamic stability prior to practice and competition [30].
**ABDOMINAL PAIN**

Several self-limited GI illnesses in athletes present with abdominal pain, and although the cause is diverse, early differentiation of emergent, surgical causes from the more benign illnesses is of paramount importance. Mesenteric lymphadenitis, Meckel’s diverticulitis, strangulated inguinal hernia, ruptured ovarian cyst, ectopic pregnancy, acute salpingitis, acute cystitis, pelvic inflammatory disease, and endometriosis can all mimic acute appendicitis [84]. Abdominal pain, anorexia, nausea, vomiting, and fever are common symptoms, but a detailed history and clinical examination in the training room can determine if the athlete who has abdominal pain needs an emergent surgical consult. A history of recent respiratory tract or GI illness can cause mesenteric lymphadenitis. A tender palpable mass with overlying skin discoloration in the lower quadrant is suggestive of incarcerated inguinal hernia. Acute, sharp abdominal pain with nausea and vomiting during fixed times of the menstrual cycle can be either secondary to a ruptured ovarian cyst or endometriosis. Abdominal pain associated with hematuria and dysuria could be secondary to cystitis. Bilateral lower quadrant pain with vaginal discharge, immediately following menses, is typically caused by pelvic inflammatory disease. Finally, several medications can reduce the efficacy of oral contraceptives and therefore, acute, unilateral abdominal pain associated with hypotension and tachycardia could represent ectopic pregnancy.

Acute appendicitis typically presents with constant, progressive, shifting pain. The pain usually begins in the periumbilical area and subsequently moves to the right lower quadrant over several hours. Additionally, anorexia, nausea, vomiting, and fever are delayed symptoms that seldom precede the onset of pain. Although acute appendicitis is overwhelmingly more common, chronic appendicitis presenting as low back pain has been described in an athlete [85]. Appendicitis has also rarely been described following trauma to the abdominal wall [86]. And finally, a rare case of marked psoas hypertrophy in a bodybuilder has been shown to mimic appendicitis secondary to compression of the cecum [87].

Chronic abdominal pain in athletes in the absence of other constitutional GI symptoms is most commonly caused by hernias. Hiatal hernia [88,89] and umbilical and inguinal hernias have been described secondary to intra-abdominal pressure overload [88,90]. Weakness of the musculotendinous part of the posterior inguinal wall (“sportsmans’ hernia”) is also a common cause of groin pain in athletes [91–93]. Surgical correction is indicated for persistent symptoms [94–96] and laparoscopic hernioplasty is superior to conventional herniorrhaphy [97].

**MISCELLANEOUS GASTROINTESTINAL DISORDERS**

**Travel**

Although only a few GI illnesses are associated with travel, athletes are particularly susceptible to acute gastroenteritis from bacterial [98] and viral [54] causes.
Acute gastroenteritis typically presents with fever, nausea, vomiting, diarrhea, cramping, and myalgias. There have been reports of acute outbreaks of gastroenteritis in the athletic population including *Salmonella enteritidis* at the Junior World Rowing Championships at Poznan, Poland, in August 1995 [98], and Norwalk viral gastroenteritis in a football team in the United States [99]. Athletes who have acute gastroenteritis, especially those involved in contact sports, should be prevented from participating in practices and competition until they have been adequately treated, are afebrile, and are hemodynamically stable. And in order to prevent contamination and spread, the fundamental importance of good hygiene cannot be overemphasized.

**Immunizations**

Infectious hepatitis is common worldwide. Viral hepatitis, although rarely encountered in athletes, can be a significant source of morbidity. Outbreaks of hepatitis A have been documented in soccer and football players [100]. Athletes traveling to underdeveloped countries should receive the hepatitis A vaccination, with a booster at 6 months to ensure lifelong immunity. Unlike hepatitis A, which is transmitted by way of the fecal–oral route, hepatitis B is transmitted through blood-borne contamination. Transmission of hepatitis B between an infected and a healthy athlete generally requires sustained contact between an exposed skin or mucous membrane lesion—a situation that fortunately rarely arises [100]. Nevertheless, hepatitis B vaccination is recommended for all athletes, especially those who participate in contact sports [101]. A three-dose series of vaccinations over a 6-month period provides the most efficacious long-term immunity. However, an accelerated series of vaccines can be administered over 2 months for athletes in need of emergent protection [101].

**SUMMARY**

Various physiologic adaptations to high-intensity exercise predispose athletes to several common GI disorders. Although the potential mechanisms involved in this increased susceptibility have been identified, few studies have compared GI disorders between sedentary individuals and high-performance athletes. A clear understanding of these mechanisms is pivotal for medical professionals charged with the daily care of athletes to help develop effective diagnostic, preventive, and therapeutic algorithms.

**References**


