



Scoliosis, Superior Mesenteric Artery Syndrome, and Adolescents

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Adolescent idiopathic scoliosis is defined as a lateral curvature of the spine that can occur in any region of the spinal column. For curves that require surgical correction, spinal fusion is the surgical treatment, and superior mesenteric artery syndrome is a possible complication. Risk factors for superior mesenteric artery syndrome include a small aorta-superior mesenteric artery angle, spinal lengthening, and an asthenic habitus. Asthenic habitus may be due to natural build, peptic ulcer disease, or anorexia, especially among adolescent females. Research regarding adolescent idiopathic scoliosis and superior mesenteric artery syndrome is warranted to identify if some adolescents are more likely to develop superior mesenteric artery syndrome. The advanced practice nurse can identify which adolescents may develop superior mesenteric artery syndrome and provide safe care to avoid this complication.

What is Scoliosis?

Scoliosis is a condition in which the spine curves laterally instead of maintaining a straight alignment in the coronal plane. A scoliotic spine departs from midline in the frontal plane and does not have the normal spinal curves in the thoracic and lumbar regions (Morrissy & Weinstein, 2001). The apex of a scoliotic curve can be found in any region of the back including cervical, cervicothoracic, thoracic, thoracolumbar, lumbar, and lumbosacral (Morrissy & Weinstein). Depending on the degree of curve and the location of the apex, the thorax and surrounding structures can be misaligned and their function may be hampered (i.e., respiratory or gastroesophageal function).

Scoliosis often develops in children without any past medical/neurological history. The scoliotic process may be congenital or neuromuscular, but most diagnosed cases of scoliosis have no known cause and are termed idiopathic (Behrman & Kliegman, 2002; Morrissy & Weinstein, 2001; Tsirikos & Jeans, 2005). Idiopathic scoliosis can develop at any age, and adolescent idiopathic scoliosis comprises approximately 80% of scoliosis cases (Morrissy & Weinstein). Children diagnosed with adolescent idiopathic scoliosis are often of the age 11 years or older (Behrman & Kliegman). Incidence of idiopathic scoliosis is similar among males and females, with females diagnosed slightly more often. Curves in females are more likely to progress and require treatment than in males (Behrman & Kliegman).

When an adolescent is diagnosed with idiopathic scoliosis, their curve is measured and a treatment plan is developed. Treatment for scoliosis depends on the child's age, the skeletal maturation, and the location, degree, classification, and progression of the curve. Younger age and female gender are risk factors for having a progressive curve that requires treatment (Behrman & Kliegman, 2002). Treatment options are variable and range from monitoring/observation every few months to wearing a hard brace such as a Thoraco-Lumbo-Sacral Orthosis (TLSO) or a soft brace such as SpineCor (Morrissy & Weinstein, 2001; Weiss & Weiss, 2005). The most invasive treatment option is spinal fusion surgery.

Several techniques for spinal fusion exist, including the posterior method, the anterior thoracotomy or thoracoscopic approach, or the combined anterior and posterior approach (Al-Sayyad, Crawford, & Wolf, 2005; Morrissy & Weinstein, 2001). These techniques involve placement of rods, hooks, bone fusion, or pedicle screws on the spinal column to decrease the lateral curvature of the scoliotic spine (Lenke et al., 2003; Storer et al., 2005). The goal of spinal fusion surgery is to realign the curve so that the spine is as straight as possible and to prevent further curve progression (Morrissy & Weinstein). Preserving mobility and balance and preventing discomfort are also important. Spinal fusion surgery has been performed for many years, but it is not without possible complications. Superior mesenteric artery syndrome has been repeatedly documented as a complication of scoliosis surgery (Amy, Priebe, & King, 1985; Avinash, 2003; Crowther, Webb, & Eyre-Brook, 2002; Evarts, 1971; Evarts, Winter, & Hall, 1971; Griffiths, & Whitehouse, 1978; Hutchinson & Bassett, 1990; Sapkas & O'Brien, 1981; Shah, Albright, Vogt, & Moreland, 2003; Shapiro, Green, Fatica, & Boachie-Adjei, 2001; Vitale, Higgs, Liebling, Roth, & Roye, 1999; Zhu & Qiu, 2005).

The purpose of this review is to determine if certain adolescents with adolescent idiopathic scoliosis, after spinal fusion surgery, are more at risk to develop superior mesenteric artery syndrome. In addition, this article will describe how the advanced practice nurse can identify these patients and offer both preventative and supportive care to avoid this complication. A literature search was conducted using databases including Medline, CINAHL, and ISI Web of Science. One Web site (eMedicine) was

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utilized on the Internet. All research was dated from 1971 through 2005. Key words included superior mesenteric artery, superior mesenteric artery syndrome, scoliosis, and disordered eating.

What is Superior Mesenteric Artery Syndrome?

The superior mesenteric artery branches off from the anterior abdominal portion of the aorta and descends from the aorta at an angle that ranges between 38° and 56° with an average of 45° . The superior mesenteric artery supplies blood flow to the small bowel, the cecum, and the ascending and transverse colons. The third portion of the duodenum lies horizontally between the vertical abdominal aorta and the superior mesenteric artery. This portion of the duodenum is also supported by the ligament of Treitz and cushioned by retroperitoneal fat and lymphatic tissue in the area of the aorta and superior mesenteric artery. If the angle between the superior mesenteric artery and the aorta is altered in any way, or if the ligament of Treitz pulls the duodenum higher into the aorta-superior mesenteric artery angle, the duodenum may become compressed and an obstruction can develop (see Figure 1).

A patient with superior mesenteric artery syndrome presents with signs and symptoms including nausea, emesis (which may be bilious), abdominal pain, and abdominal distension. The diagnostic study of choice for superior mesenteric artery syndrome is an upper gastrointestinal radiograph with oral contrast. This study may reveal dilation of the stomach or the first and the second portion of the duodenum (Evarts et al., 1971; Griffiths & Whitehouse, 1978; Sapkas & O'Brien, 1981). A vertical cutoff of oral contrast through the third portion of the duodenum due to superior mesenteric artery compression is diagnostic (Griffiths & Whitehouse). The goal of treatment is to decrease duodenal edema and relieve the obstruction.

How Are Superior Mesenteric Artery Syndrome and Scoliosis Related?

It is not surprising that superior mesenteric artery syndrome is linked to scoliosis. The procedure manipulates and lengthens the spine, causes a rapid increase in height, and alters the original aorta-superior mesenteric artery angle (Crowther et al., 2002; Evarts et al., 1971). The ligament of Treitz may also be affected by scoliosis surgery and subsequently change the position of the duodenum in relation to the superior mesenteric artery and aorta. Adolescent idiopathic scoliosis also occurs during the child's growth spurt, a time in which bodily changes may also alter the relationship between the superior mesenteric artery, spine, aorta, and duodenum (Hutchinson & Bassett, 1990; Shapiro et al., 2001). Despite lengthening of the spine and rapid increases in height, not every patient with spinal fusion develops superior mesenteric artery syndrome.

Currently, little data are available that clearly identify risk factors for adolescents who develop superior mesen-

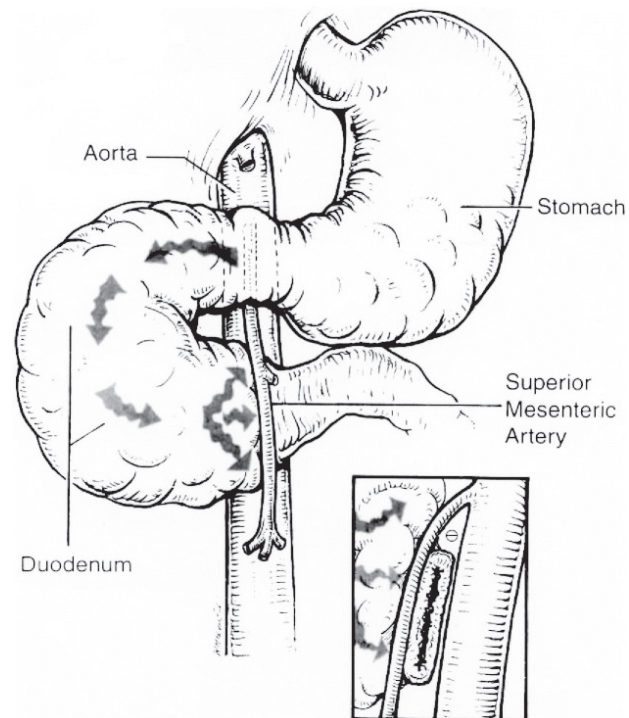


FIGURE 1. The duodenum crosses the abdominal cavity from right to left under the superior mesenteric artery and over the vertebral bodies. The superior mesenteric artery has an acute angle of takeoff from the aorta, referred to as the arterio-mesenteric angle. A number of variables can reduce the arterio-mesenteric angle, causing pathologic compression of the duodenum and intractable vomiting. In scoliosis surgery, these variables include the adolescent growth spurt, an acute increase in height after spinal instrumentation, and postoperative weight loss. Reproduced with permission from Lippincott, Williams, & Wilkins (Shah et al., 2003).

teric artery syndrome after surgical correction of idiopathic scoliosis. Case reports and retrospective studies identify spinal lengthening, a change in the aorta-superior mesenteric artery angle, and an asthenic habitus as causative factors in superior mesenteric artery syndrome (Amy et al., 1985; Avinash, 2003; Crowther et al., 2002; Evarts, 1971; Evarts et al., 1971; Hutchinson & Bassett, 1990; Sapkas & O'Brien, 1981; Shah et al., 2003; Shapiro et al., 2001; Vitale et al., 1999).

An asthenic habitus as a possible cause of superior mesenteric artery syndrome was a common topic of many research articles. Hutchinson and Bassett (1990) documented that all 8 of their patients who underwent spinal fusion surgery and developed superior mesenteric artery syndrome were "disproportionately thin in relation to their height" (p. 251). Avinash (2003) documented that approximately 80% of patients with superior mesenteric artery syndrome present with a thin habitus, and Shapiro et al. (2001) stated that the majority of patients with superior mesenteric artery syndrome are 20% or more under their ideal body weight. With an asthenic body build, three factors may play a role in the development of superior mesenteric artery syndrome after scoliosis surgery. First, because of a thin body build, the patient

may already have a small angle between the aorta and superior mesenteric artery that is further decreased after spinal fusion. Second, adolescent idiopathic scoliosis occurs during the growth spurt that may also contribute to the change in aorta-superior mesenteric artery angle as an already thin patient gains height. Third, a patient with asthenia may lack the retroperitoneal fat that protects the duodenum from the superior mesenteric artery when the spine is stretched and the superior mesenteric artery angle decreases (Amy et al., 1985; Avinash; Crowther et al., 2002; Hutchinson & Bassett; Shah et al., 2003; Shapiro et al.; Vitale et al., 1999). Patients may either lack the retroperitoneal fat to start with or may have insufficient retroperitoneal fat after surgery to protect the duodenum during postoperative convalescence.

Other supportive evidence linking adolescent idiopathic scoliosis, the asthenic habitus/retroperitoneal fat theory, and superior mesenteric artery syndrome comes from the fact that this complication has also been documented in patients with anorexia nervosa and severe weight loss in conditions such as cancer or burns (Avinash, 2003; Pentlow & Dent, 1981). There is no current research that clearly links superior mesenteric artery syndrome with the loss of retroperitoneal fat. The idea is assumed because many of the patients who have adolescent idiopathic scoliosis and superior mesenteric artery syndrome present with a thin habitus before surgery. Although many investigators support the theories of a thin habitus and changes in superior mesenteric artery angles, they also suggest that discordant data are available (Hutchinson & Bassett, 1990; Shapiro et al., 2001; Vitale et al., 1999). Other authors dispute the asthenic habitus/retroperitoneal fat hypothesis and contend that weight loss and the supposed loss of retroperitoneal fat occur as a result of superior mesenteric artery syndrome.

Are There Other Risk Factors for Superior Mesenteric Artery Syndrome After Spinal Fusion Surgery?

It is not yet clear which adolescents are more at risk to develop superior mesenteric artery syndrome after spinal fusion surgery. Asthenic habitus, spinal lengthening, changes in the aorta-superior mesenteric artery angle, and the retroperitoneal fat theory have been documented as the supposed reasons why some patients develop superior mesenteric artery syndrome after spinal fusion surgery. Alternate theories exist, such as associated gastroesophageal ailments or eating disorders, that warrant further investigation.

Avinash (2003) cited a cause of superior mesenteric artery syndrome that may correlate with adolescent idiopathic scoliosis and the development of postfusion superior mesenteric artery syndrome. The article documented that peptic ulcer disease was present in 25–45% of patients with superior mesenteric artery syndrome and hyperchlorhydria in 50% of patients. Although Avinash's article is not specific to adolescent idiopathic scoliosis, perhaps patients with adolescent idiopathic scoliosis have undiagnosed concomitant gastroesophageal ailments, such as peptic ulcer disease, due to their spinal curve.

Perhaps these gastroesophageal conditions are a trigger for some patients with adolescent idiopathic scoliosis to develop the asthenic habitus that may predispose them to superior mesenteric artery syndrome. Although Avinash did not present scenarios regarding gastroesophageal ailments, scoliosis, and superior mesenteric artery syndrome, can we not suppose that a large scoliotic curve could push on the stomach and cause regurgitation and reflux of stomach contents? The result may be less oral intake due to discomfort. A severe spinal curvature pushing on the stomach may also decrease stomach volume. Reduced stomach volume may also cause decreased oral intake if the patient suffers from early satiety, and if the stomach of a patient with an adolescent idiopathic scoliosis is often empty, perhaps he/she experiences hyperchlorhydria and prefers to eat even less. Further research is needed to explore the connection between adolescent idiopathic scoliosis, superior mesenteric artery syndrome, peptic ulcer disease, and hyperchlorhydria.

Aside from gastroesophageal conditions as a possible cause of superior mesenteric artery syndrome in adolescent idiopathic scoliosis, it is interesting to note that the majority of documented cases of adolescent idiopathic scoliosis and superior mesenteric artery syndrome in the literature were females. Several authors specifically describe the asthenic habitus of these females (Amy et al., 1985; Avinash, 2003; Evarts, 1971; Evarts et al., 1971; Griffiths & Whitehouse, 1978; Hutchinson & Bassett, 1990; Sapkas & O'Brien, 1981; Vitale et al., 1999). When considering that slightly more females are diagnosed with adolescent idiopathic scoliosis, and females tend to have more progressive curves that require treatment, further studies of the asthenic body build as an independent risk factor for superior mesenteric artery syndrome are warranted. Could the asthenia be due to a naturally thin body build, a growth spurt, peptic ulcer disease, or are there other factors, such as eating disorders, involved?

In general, adolescence is a time of self-consciousness and uncertainty related to body image, which may be further heightened by adolescent idiopathic scoliosis (Behrman & Kliegman, 2002; Reichel & Schanz, 2003). Although males can also have feelings of self-consciousness and body image disturbance, only the female perspective of adolescent idiopathic scoliosis and adolescence will currently be discussed.

As documented in Smith, Latchford, Hall, Millner, and Dickson's (2002) research, several studies in the past have linked disturbed body image to eating disorders, but no research links adolescent idiopathic scoliosis to eating disorders. Smith et al. appear to be the first to recognize that adolescent idiopathic scoliosis occurs during a tumultuous time for a female, and the authors attempted to identify the prevalence of disordered eating behaviors in females with adolescent idiopathic scoliosis in England. Their study compared height, weight, and body mass index (BMI) in females with adolescent idiopathic scoliosis to normative data for same age girls. The study group had no other medical issue other than adolescent idiopathic scoliosis. There were 44 patients with adolescent idiopathic scoliosis, and the mean age was 16 years. Smith et al. used the International Classification of Diseases (ICD) to indicate disordered eating when the BMI is less than 17.5 kg/m². It should be noted that the authors

did not further delineate any specific type of eating disorder beyond this definition.

The results of the study identified no significant difference in height among the adolescent idiopathic scoliosis girls and the control data, but the adolescent idiopathic scoliosis group weighed significantly less and had significantly lower BMIs than the norm. Twenty-five percent of the adolescent idiopathic scoliosis study groups' BMIs were lower than 17.5 kg/m² and considered within the range of an eating disorder (Smith et al., 2002).

In this small study from England, Smith et al. (2002) illustrated that in some females with adolescent idiopathic scoliosis, a relationship may exist between adolescent idiopathic scoliosis, low weight, and BMIs within the anorexic/disordered eating behavior range. The study group did not have any other medical conditions that could cause weight loss, and there are no proven physiological causes for decreased weight in adolescent idiopathic scoliosis. There appears to be no other reason for the decreased weight and BMI, but the researchers did not investigate any other causes of weight loss in adolescent females with adolescent idiopathic scoliosis. Perhaps the patients had premorbid eating disorders or gastroesophageal ailments due to their spinal curve, such as those mentioned in Avinash's (2003) article. Despite Smith et al.'s correlation between adolescent idiopathic scoliosis and BMIs in the anorexic range, one cannot assume that the cause of the thin habitus was definitely an eating disorder. Another limitation exists within the study regarding the definition of disordered eating. The ICD classification for anorexia when the BMI is less than 17.5 kg/m² applies to adults and may not be an accurate measurement for adolescent females. One could argue that the altered height of patients with adolescent idiopathic scoliosis when corrected for scoliosis would further lower their BMIs and still correlate with definitions for anorexia.

Smith et al. (2002) are the first to identify a potential risk of eating disorders/anorexia in females with adolescent idiopathic scoliosis. From their research, one can infer that if a thin body build is correlated with the development of superior mesenteric artery syndrome after spinal fusion surgery, then patients with adolescent idiopathic scoliosis who have low weights and low BMIs should be screened for anorexia and identified as patients with the potential to develop superior mesenteric artery syndrome after spinal correction. They should also be screened for other causes of low weight and low BMI, such as peptic ulcer disease. Further investigation is warranted with larger study groups to truly identify if there are correlations between anorexia/eating disorders or peptic ulcer disease and adolescents with adolescent idiopathic scoliosis. If these correlations are found reliable and valid, future studies can identify the prevalence of peptic ulcer disease or anorexia in adolescents with adolescent idiopathic scoliosis who develop superior mesenteric artery syndrome after spinal fusion surgery.

Can We Identify Patients at Risk for Superior Mesenteric Artery Syndrome?

Currently, there are very few measurements to recognize exactly who will develop superior mesenteric artery syn-

drome after spinal fusion surgery for adolescent idiopathic scoliosis. In 2003, Shah et al. attempted to identify a weight/height parameter that could indicate which adolescent idiopathic scoliosis patients are at risk to develop superior mesenteric artery syndrome after corrective surgery. Shah et al. recognized that a thin body build is frequently documented in the literature pertaining to superior mesenteric artery syndrome. They believe that asthenia of a patient with scoliosis is somehow connected to other superior mesenteric artery syndrome risk factors such as changes in the aorta-superior mesenteric artery angle, the growth spurt, and the large increase in height after spinal fusion surgery. The goal of their retrospective chart review was to explore the connection between weight, height, and superior mesenteric artery syndrome before and after spinal fusion surgery.

The authors reviewed charts for 17 patients who developed superior mesenteric artery syndrome after spinal fusion surgery. Six of the 17 patients had scoliosis (5 with adolescent idiopathic scoliosis). Weight before and after the diagnosis of scoliosis, weight percentile, height percentile, and weight percentile for height were identified. The weight and height information for the 17 patients who developed superior mesenteric artery syndrome were compared to that for 16 patients who underwent spinal fusion surgery at the same hospital but did not develop superior mesenteric artery syndrome. Comparisons were also made to age-matched controls in the general population via National Center for Health Statistics data (Shah et al., 2003).

Comparison of the scoliosis-superior mesenteric artery syndrome group and the nonsuperior mesenteric artery syndrome group reveals significant differences in the weight percentile for height at the time of diagnosis. The average weight percentile for height for the scoliosis-superior mesenteric artery syndrome group was 3% compared to 49% in the age-matched nonsuperior mesenteric artery syndrome group. Two of the six patients with scoliosis-superior mesenteric artery syndrome had further decreases in their weight percentile for height after surgery (25th percentile down to below 5th percentile). The authors state that the scoliosis-superior mesenteric artery syndrome group's weight percentile for height was also significantly lower than age-matched controls' in the general population. Looking at height alone or weight alone, no significant differences were identified between the scoliosis-superior mesenteric artery syndrome group, the nonsuperior mesenteric artery syndrome group or the general population (Shah et al., 2003).

According to Shah et al. (2003), the weight percentile for height can be used as an indicator for the risk of developing superior mesenteric artery syndrome after corrective surgery for adolescent idiopathic scoliosis. They believe that weight percentile for height quantifies the degree of asthenia and may reflect the relationship of the multiple risk factors of superior mesenteric artery syndrome after spinal fusion surgery (asthenia, adolescent growth spurt, changes in the aorta-superior mesenteric artery angle, height increase without compensatory weight gain, and any postoperative weight loss). Shah et al. suggested a parameter in which patients with weight percentile for height in the 5th percentile have a degree of asthenia that will alter the aorta-superior

mesenteric artery angle. Patients with adolescent idiopathic scoliosis who have weight percentile for height nearing 2 standard deviations below the mean may be prone to develop superior mesenteric artery syndrome after spinal fusion (Shah et al.). Similar to Smith et al. (2002), Shah et al.'s study is limited by inaccurate heights in the patients with adolescent idiopathic scoliosis, and their parameter might not be completely accurate because the heights of the patients with adolescent idiopathic scoliosis are not corrected.

Similar research was documented in an article by Zhu and Qiu (2005) from Drum Tower Hospital in Nanjing, China. Over a 6-year period (July 1997 to October 2003), 640 patients underwent surgical treatment for adolescent idiopathic scoliosis. Seven of the 640 patients developed superior mesenteric artery syndrome. Prior to surgery, all the patients were assigned a percent value for height and weight. Zhu and Qiu documented that the height percent of 7 patients with superior mesenteric artery syndrome was equal to or above the mean of same gender, and age matches the normal population. On the contrary, the weights of the 7 patients with superior mesenteric artery syndrome were in the 5–25% range and less than the mean of same gender, and age matches the normal population (Zhu and Qiu). The low weight percentiles in patients with superior mesenteric artery syndrome in China are very similar to Shah et al.'s (2003) documentation of weight percentile for height in their patients with scoliosis. In their conclusion, Zhu and Qiu echo Shah et al. and suggest that a weight percentile less than 25% is a potential risk factor for superior mesenteric artery syndrome in patients with adolescent idiopathic scoliosis who are undergoing spinal fusion.

What is the Role of the Advanced Practice Nurse in Scoliosis and Superior Mesenteric Artery Syndrome?

Advanced practice nurses caring for patients with adolescent idiopathic scoliosis need to recognize superior mesenteric artery syndrome as a possible complication following spinal fusion surgery, especially in thin females. Further research is needed to identify if male patients with adolescent idiopathic scoliosis and asthenic habitus are also at similar risk to develop superior mesenteric artery syndrome as female patients. Because advanced practice nurses typically follow these patients for several years before their spinal fusion surgery, a relationship will develop in which advanced practice nurses can identify those at risk years before surgery is proposed. In a trusting relationship over time between the patient with adolescent idiopathic scoliosis and the practitioner, advanced practice nurses can discuss eating disorders if appropriate, ask questions about gastroesophageal ailments, and explain the importance of good nutrition in general and in relation to future surgery. More specifically, under the watchful eye of an advanced practice nurse, a patient with adolescent idiopathic scoliosis with an asthenic habitus will either maintain or gain weight to avoid a poor outcome. Because of this relationship over time, patients with adolescent idiopathic scoliosis who could potentially develop superior mesenteric artery syn-

drome after spinal fusion surgery can decrease their risk due to the vigilant care of the advanced practice nurses.

Advanced practice nurses should document accurate height, weight, and weight percentile for height, keeping in mind that anthropometric measures are not exact for patients with adolescent idiopathic scoliosis. In conjunction with a nutritionist, advanced practice nurses can also screen for peptic ulcer disease by asking if the patient avoids any particular foods (i.e., chocolate, tomato sauce, orange juice). If advanced practice nurses are experienced in using screening tools for eating disorders, a psychologist can collaborate. Although there are no data on presurgery imaging studies, Avinash (2003) suggests that computed tomography (CT) scans and ultrasounds can reveal aorta-superior mesenteric artery distances and may also identify the amount of retroperitoneal fat. These imaging studies may also reveal the placement of the ligament of Treitz in relation to the duodenum. Prior to these radiological studies for patients with adolescent idiopathic scoliosis determined to be at risk for superior mesenteric artery syndrome, the medical team and the patient/family would have to discuss the benefits and risks. Shah et al. (2003) also suggested that preoperative weight gain has been used to offset the risk of an asthenic body build and the development of superior mesenteric artery syndrome in patients deemed at risk. A naso-jejunal tube can also be placed prior to surgery or intraoperatively in preparation of possible postoperative vomiting, weight loss, and development of superior mesenteric artery syndrome. The advanced practice nurse can also keep a watchful eye on patients with postoperative spinal fusion who lose weight or begin to vomit several days after surgery.

Advanced practice nurses working with the adolescent idiopathic scoliosis population can be the first line of defense in identifying which adolescents may be at risk for developing superior mesenteric artery syndrome after spinal fusion surgery. Multiple risk factors have already been recognized in the development of superior mesenteric artery syndrome, such as thin habitus, small aorta-superior mesenteric artery angle, changes in the aorta-superior mesenteric artery angle, and large height increase after surgery. More research is needed to truly identify if other factors, such as peptic ulcer disease, anorexia, and weight in comparison to height before treatment play a role in the development of superior mesenteric artery syndrome after corrective surgery for scoliosis. Research is also necessary to develop either a growth chart for adolescents with adolescent idiopathic scoliosis, develop calculations to obtain a corrected height and weight percentiles for height by knowing the angle/degree of curve, or methods to standardize documentation of height and weight percentiles for height in relation to the degree of curve. With more documentation and research of superior mesenteric artery syndrome, perhaps advanced practice nurses will be able to identify if there are some adolescents who are more at risk to develop superior mesenteric artery syndrome after spinal fusion than others and prevent this complicated setback.

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