Curr Gastroenterol Rep (2012) 14:367–372
DOI 10.1007/s11894-012-0271-7

NUTRITION AND OBESITY (S MCCLAVE, SECTION EDITOR)

Neurologic Complications of Bariatric Surgery: Involvement of Central, Peripheral, and Enteric Nervous Systems

David J. Frantz

Published online: 2 June 2012

Abstract Approximately one in three Americans is obese. Current society guidelines recommend bariatric surgery after conservative measures at weight loss have failed. The frequency of bariatric surgeries has increased significantly over the past decade. While considered both safe and effective, bariatric surgery presents a distinct set of risks. This review focuses on the neurological complications of bariatric surgery. Injuries have been reported at all levels of the nervous system, including the central, peripheral, and enteric nervous system. Injury can be classified according to time of presentation and location. The two main mechanisms of nerve injury are from mechanical injury or as a consequence of malnutrition. Encephalopathy, peripheral neuropathies, myelopathies, and radiculoneuropathies have all been reported. Mechanical injuries likely occur from mechanical compression. Malnutrition injuries result from multimicronutrient deficiencies. The most likely candidates are vitamin B12, folate, zinc, thiamin, copper, vitamin A, and vitamin E deficiencies.

Keywords Bariatric surgery · Roux-en-Y · Laparoscopic adjustable gastric band · Nervous system · Complication · Weight loss · Malnutrition · Micronutrient · Deficiency · Neuropathy · Mononeuropathy · Radiculopathy · Myelopathy · Radiculoneuropathy · Wernicke–Korsakoff · B12 · Folate · Zinc · Copper vitamin A · Vitamin E

D. J. Frantz (✉)
Division of Gastroenterology and Hepatology,
University of North Carolina School of Medicine,
130 Mason Farm Road, Bioinformatics Building CB# 7080,
Chapel Hill, NC 27599-7080, USA
e-mail: dfrantz@unch.unc.edu

Introduction

Epidemiology of Obesity

Approximately one in three adults in the United States is obese [1•]. The impact of obesity on fiscal resources may become one of the most significant health burdens that the United States will face. In 2001, the American Surgeon general declared that obesity had reached epidemic proportions and issued a call to action to prevent and decrease overweight and obesity [2]. Despite this call to action, the most recent analysis of the NHANES data reveals that there has been little change in the prevalence of obesity over the past 12 years [1•]. It is estimated that in 2008, $147 billion per year was spent on obesity-related health problems. As the population continues to age, this number will only continue to increase [3].

Treatment

Despite efforts to produce effective weight loss medications, current medical therapy for obesity is limited. The current practice guidelines recommend a concerted effort of diet and lifestyle modification, followed by trials of pharmacologic therapy. Bariatric surgery is recommended for patients who have a BMI$\geq$40 kg/m^2 with no obesity related comorbidities, or for patients with a BMI$\geq$35 kg/m^2 with comorbid conditions such as hypertension, obstructive sleep apnea, diabetes, or dyslipidemia, who have failed conservative treatment.

Early bariatric procedures included jejunoileal bypass, duodenal switch and biliopancreatic diversion with duodenal switch. These procedures induced significant weight loss, but fell out of favor because of severe malnutrition. Vertical banded gastroplasty was the surgery of choice for a

while, but rates of suture line failures and gastrogastric fistula formation were high. Laparoscopic and endoscopic vertical sleeve gastroplasty is gaining favor and may become the procedure of choice in the future. It is less invasive than a Roux-en-Y bypass and produces better weight loss than laparoscopic adjustable gastric banding (LAGB). Intragastric balloons have been tried with mixed success, and are only available outside the United States. Other endoscopically-placed devices and procedures are being studied.

Currently, laparoscopic Roux-en-Y gastric bypass and LAGB are the procedures most commonly performed in the United States. Laparoscopic Roux-en-Y surgery is a minimally invasive procedure in which the stomach is transected near the gastroesophageal junction, creating a small pouch with a volume of approximately 30 cc. The pouch is in continuity with the small bowel through a gastrojejunal anastomosis termed the 'alimentary' or 'roux' limb. The gastric body remnant drains through a biliopancreatic limb that then connects with the roux limb approximately 75–150 cm distal to the gastrojejunal anastomosis.

There are two laparoscopic adjustable gastric bands approved for use in the United States. Both consist of a silicon tube that is positioned around the gastric body approximately 1–3 cm from the gastroesophageal sphincter, measured on the lesser curvature. A path is dissected around the stomach, generally through the pars flaccida of the hepatogastric ligament. The tube encircles the stomach and is locked together to form a band that compartmentalizes the stomach. It can be anchored in place with gastrogastric sutures to help prevent slippage. A subcutaneous port is then brought up and inserted on top of the abdominal rectus muscles for easy access. Saline is injected or removed through the port to control the diameter of the band. Frequent physician visits are required to adjust the band to maintain weight loss. The FDA recently expanded approval for LAGB to patients with a BMI of 30–34 kg/m^2 with comorbid conditions [4].

Physiology of Weight Loss

The exact physiology of bariatric surgery is incompletely understood. Most authors agree that weight loss results from a combination of caloric restriction, malabsorption and modulation of the entero-encephalic endocrine axis [5•]. Each bariatric surgery option creates differing degrees of these effects. For example, laparoscopic Roux-en-Y surgery restricts the amount of calories that can be eaten per meal. The gastrojejunostomy creates a dumping syndrome that contributes to malabsorption, and the length of the roux limb, which is responsible for the majority of the malabsorption associated with the procedure, delays mixing of chyme and biliopancreatic fluids. Finally, the altered anatomy created during surgery modulates the entero-encephalic endocrine axis.

The role of the entero-encephalic endocrine axis deserves important mention. Within a week of Roux-en-Y surgery, most patients have a significant reduction in their insulin resistance that precedes weight loss. Anecdotally, some patients also report an almost immediate loss of interest in food. The adjustable laparoscopic gastric band does not impart the same improvement in insulin resistance as bypass surgery, but it also induces sensations of satiety and decreases hunger that is attributed to a neurohormonal response. The complex interplay between the signaling peptides from gut, brain, and endocrine system (which now includes adipokines), is a burgeoning field that holds promise for future weight loss therapies.

Epidemiology of Bariatric Surgery

The lack of effective medical therapy for obesity and the relative safety and efficacy of bariatric surgery has prompted many professional societies to promote bariatric surgery after conservative measures of weight loss have failed [6]. The number of bariatric procedures has increased dramatically from 20,000 procedures in 1999 to approximately 120,000 in 2008, with evidence suggesting that this trend is slowing or plateauing [7, 8]. Roux-en-Y procedures are the predominant bariatric procedure done in the United States, followed by the adjustable gastric band [7]. The 30-day mortality for Roux-en-Y surgery is estimated to be less than 1 % [7, 9, 10]. Laparoscopic adjustable gastric banding has even less short term mortality and morbidity [11]. Long term outcomes from prospective data are currently being collected [9, 12].

Neurologic Complications of Bariatric Surgery

There are a variety of neurologic complications that can occur, and these can be classified by time to presentation, by mechanism of injury, and by physical location. This review will focus on the potential neurological complications organized by time to presentation and location.

Immediate Complications

Most immediate complications involve the enteric and peripheral nervous system and are a direct result of surgery. These include peripheral neuropathies, meralgia paresthetica, gastroparesis, excessive vagal stimulation and possibly achalasia [13–17].

Immediate Peripheral Nerve Injuries

Morbidly obese patient are inherently at higher risk for peripheral neuropathies because of their larger body habitus.

There are a variety of peripheral neuropathies reported after surgery. In particular, stretch injury to the brachial plexus and ulnar neuropathy are commonly reported [15]. Careful attention to positioning, padding, and repositioning during prolonged surgeries are all-important to help prevent injury [15]. Patients undergoing bariatric surgery are also at risk for rhabdomyolysis and myopathies associated with compression injury. There are case reports of gluteal compartment syndrome after bariatric surgery, which has resulted in sciatic nerve damage [18, 19].

Meralgia Paresthetica is caused by compression damage to the lateral cutaneous nerve where it passes through the inguinal ligament. Patients develop pain, paresthesias, or hypersensitivity in the anterior and lateral aspects of the thigh [13]. It varies in degree from mild pruritus, which can be confused for factitious dermatitis, to frank anesthesia in the region. Contributing factors include traction forces caused by the pannus on the inguinal ligament, increased intra-abdominal pressure from laparoscopic insufflation, and normal anatomical variants that predispose the nerve to damage during the supine and the lithotomy positions required for surgery. The condition is treated conservatively and usually resolves spontaneously. The incidence of meralgia paresthetica is unknown. It does not affect ambulation and likely goes underdiagnosed. In refractory cases, local nerve blocks have been helpful to reduce the symptoms of debilitating pain.

Vagal nerve injury can occur during bariatric surgery. Some of the nausea and vomiting that accompanies bariatric surgery can be attributed to vagal injury resulting in decreased motility [14]. After a few months, reinnervation, which improves function and reduces symptoms, may occur. In contrast, there are also case reports of excessive vagal nerve stimulation resulting in a vasovagal response that, in rare cases, can lead to arrhythmias and death. One case occurred after placement of an intragastric balloon that triggered a bradyarrhythmia, resulting in cardiac arrest [17]. Another patient suffered cardiac arrest within minutes of having the band adjusted in the office. The authors concluded that gastric distension from a barium meal ingested prior to the adjustment, in conjunction with tightening of the band, precipitated a vagal response from which the patient was unable to compensate [20].

Immediate Enteric Nerve Injury

Enteric nerve injury after bariatric surgery is rarely, if ever, reported as an adverse event. One study evaluated patients who had undergone surgical treatment of achalasia. The authors attributed the cause of achalasia in approximately one quarter of the cases to lower esophageal trauma from surgery or from automobile accidents. Two of the patients in their series had undergone bariatric surgery [16]. Possible enteric nerve damage may have played a role in these cases, but retrospective observational studies are insufficient to define causality. Rather, as described above, immediate alterations in the enteric nervous system by bariatric surgeries is one of the reasons given for the surgery's success.

Late Complications

The exact prevalence and incidence rate of long term neurologic injury following bariatric surgery is unclear. Most data describing neurologic injuries comes from retrospective and observational studies that include a variety of bariatric procedures. In 1987, Abarbanel et al. reported that 4.6 % (23 of 500) bariatric surgery patients experienced some form of neurological complication that became apparent between 3 and 20 months after surgery [21]. In 2004, Thaisetthawatkul et al. published a retrospective case–control series of patients from the Mayo Clinic, and found that 16 % (71 of 435) of patients developed some form of peripheral neuropathy [22]. Dr. Singh, in a brief review of the literature, conservatively estimated the neurological complication rate to be somewhere between 5 and10 % [23].

The mechanism of long term neurological complications is thought to be predominantly from malnutrition, but compression-type injuries from the loss of protective fat pads or structural changes are also to blame. In addition, some authors have noted finding inflammatory changes in nerve biopsies from patients thought to have bariatric-related complications. Though causality cannot be established, the authors have postulated that a cachexia-like state may cause an inflammatory state that is responsible for damaging the nerves [22].

Malnutrition can become a significant problem in patients who have undergone bariatric surgery. Surprisingly, obesity is a preexisting risk factor for malnutrition, and it is estimated that 20 %-30 % of obese patients have micronutrient deficiency prior to surgery [24]. After surgery, their nutritional status can rapidly worsen. Structural changes, loss of absorptive surface, prolonged vomiting, altered dietary patterns, bacterial overgrowth, loss of gastric acid and loss of intrinsic factor may all contribute to malnutrition after bariatric surgery.

The two most commonly reported deficiencies include vitamin B12 and thiamine. Some estimate that up to 70 % of bariatric surgery patients will develop vitamin B12 deficiency [25]. Thiamin (Vitamin B1) deficiency is also commonly reported, though the prevalence is unknown. Other deficiencies associated with neurologic injury include folate, vitamin D, riboflavin, niacin, pyridoxine, vitamin C, and vitamin E. The mechanisms of injury are beyond the scope of this review, and the reader is referred to articles by Dr. Kumar and Dr. Kazemi; both have published excellent reviews on the relationship between micronutrient deficiencies and their association to neurologic disorders [26••, 27••].

Late Central Nervous System Complications

Central nervous system complications from bariatric surgery include Wernicke–Korsakoff syndrome, Acute Post-Gastric Reduction Surgery neuropathy (APGARS), and posterolateral myelopathy. Wernicke syndrome is caused by thiamin (vitamin B1) deficiency. Thiamin is essential for energy production and myelin formation in nerve cells. Body stores of thiamin are measured in weeks to months. Once depleted, patients develop a subacute encephalopathy, called Wernicke's encephalopathy, that is classically associated with oculomotor abnormalities, gait disturbances, and mental status changes. A recent systematic review of Wernicke's encephalopathy after bariatric surgery, found that only 30 % of patients presented with all three of the classic features of Wernicke's encephalopathy. Patients presented almost exclusively within six months of surgery, and over 90 % of cases reported protracted emesis of approximately three weeks duration prior to symptom development [28]. If recognized early, administration of thiamin, 500 mg intravenously, three times daily for three days, can potentially reverse symptoms. In the systematic review, 51 % of patients had a full recovery. Untreated, Wernicke's encephalopathy often leads to Korsakoff syndrome, and sometimes death. Patients suffer permanent antegrade and retrograde memory impairment that presents as confabulation on exam. The two syndromes are felt to represent a spectrum of disease, and the disorder is now often referred to as Wernicke–Korsakoff syndrome [26••, 29].

Acute Post-Gastric Reduction Surgery neuropathy, APGARS, is a relatively new term used to describe the polyneuropathy that can occur after weight loss surgery [30]. The definition of the APGARS syndrome is not well defined and controversial. The authors define it broadly as a syndrome of vomiting, hyporeflexia, and weakness associated with bariatric surgery. Hyperreflexia, confusion, psychosis, ophthalmoplegia, and gait disturbances can also be present. APGARS most likely represents multiple nutrient deficiencies, and appears to be an umbrella term used to describe a host of neurologic disorders, including Wernicke–Korsakoff syndrome, dry beriberi, and posterolateral myopathy.

Another cause of central nervous system dysfunction is posterolateral myelopathy, which involves degeneration of the spinal cord. A recent case series from the University of Arkansas, found that 12 of their 26 cases of patients with neurologic conditions after weight reduction surgery suffered from posterolateral myelopathy. Patients reported an insidious onset of gait ataxia, leg spasticity, and hyperreflexia [31]. All patients had lost proprioception and vibratory sensation. A majority of patients had T2 abnormalities seen on imaging of the spinal cord. Deficiencies in vitamin B12, copper, and vitamin E have all been found in these patients to varying degrees. Treatment with nutritional supplements resulted in ten of 12 patients showing improvement. In a subsequent literature review, the same authors identified 139 patients with neurologic complications following bariatric surgery. They only found five cases of myelopathy, whereas the majority of reports were of encephalopathy. The authors concluded that the disparity in distribution between their case series and the literature likely resulted from reporting bias. Encephalopathy occurs much sooner after surgery, whereas myelopathy presented late, with a mean time to presentation of 9.9 years.

Late Peripheral Nervous System Complications

Intermediate and late peripheral nervous system complications present in a variety of peripheral neuropathies. These include mononeuropathies, polyneuropathies, and acute polyradiculoneuropathies. Peripheral neuropathies are the most common neurologic complaint. The best study of peripheral neuropathy after bariatric surgery is the case series published by the Mayo Clinic. They evaluated 435 patients and found that 71 (16 %) had developed some form of peripheral neuropathy. This included 39 (9 %) with mononeuropathies, 27 (6 %) with polyneuropathies, and 5 (1 %) with polyradiculopathy [22]. These findings are similar to the case series from Arkansas that documented five cases of polyneuropathy, five cases of polyradiculoneuropathy and two cases of optic neuropathy [31]. Their subsequent literature review identified 46 cases of polyneuropathy followed by 23 cases of polyradiculoneuropathies, and five cases of optic neuropathy. Koffman et al. reported a case series of 96 patients with neurological complications, and found that 62 % had some form of peripheral neuropathy. Twenty-seven patients had a generalized polyneuropathy, eight had cranial nerve mononeuropathy, and two had radiculopathy [29].

Polyneuropathy is generally described as burning feet syndrome. It usually starts in the lower extremities and progresses centrally, and in some instances also involves the upper extremities. Patients suffer from symmetric sensorimotor changes including anesthesia, tingling paresthesias, and pain [29]. Some patients report associated weakness and gait ataxia, and autonomic dysfunction including incontinence [14, 32]. The etiology of polyneuropathy is poorly understood. Most authors agree that it is likely a combination of multiple nutrient deficiencies. The most likely candidates include thiamin, B12, copper, vitamin E, and possibly vitamin D.

Mononeuropathies present more acutely and are usually asymmetric, involving only one side of the body. The most well-described mononeuropathy is median nerve neuropathy followed by optic nerve neuropathy. Other affected nerves include the sciatic, superficial radial, lateral femoral cutaneous, peroneal, and ulnar nerves. Meralgia Paresthetica, which was discussed earlier as an acute neurological injury, can also be a late finding. The exact etiology of mononeuropathies is

unclear, but laxity in supporting structures resulting in injury, as well as subacute malnutrition, may be the cause. No specific micronutrient deficiency has been definitively linked to mononeuropathies.

Polyradiculoneuropathy is a rare occurrence after bariatric surgery. It generally presents acutely, starting with pain in the lower extremities. Soon after the onset of pain, weakness rapidly sets in and patients often become bedbound. The presentation closely resembles Guillain–Barré syndrome. Lumbar puncture testing did not reveal evidence of elevated protein in the spinal fluid, as is found in Guillain–Barré syndrome. Nerve conduction studies confirmed the diagnosis of polyradiculoneuropathy, but no specific micronutrient deficiency has been identified in these patients. Nutritional support resulted in improvement of symptoms, but despite treatment, most continued to have some gait disturbances [14, 32].

In the case–control series published by Mayo clinic, the authors reported the results of five sural biopsies taken from patients with polyneuropathy or radiculoneuropathy. All five biopsies showed varying degrees of inflammatory infiltrates. In observational studies, causality cannot be confirmed, but this is an intriguing finding. The authors concluded that malnutrition and cachexia induced by bariatric surgery may contribute to, or cause, an inflammatory process that damages the nerves. However, most cases respond to nutritional therapy, and patients who reported taking vitamin supplements, and those receiving nutritional counseling, reported fewer neurologic complications; thus, the role of these findings is unclear.

Intermediate and Late Enteric Nervous System Complications

No long-term complications involving the enteric nervous system, have been reported in the literature. However, bariatric surgery alters the enteric nervous system, and this is felt to be one of the reasons why surgery is effective. In some instances, patients loose excessive weight, become significantly malnourished, or develop refractory vomiting. The contributions of the enteric nervous system to these symptoms are simply unknown. Surgical revision can be beneficial in refractory cases.

Conclusion

Neurological complications following bariatric surgery are estimated to occur in 5 %–10 % of patients. The two most likely mechanisms of injury are from compression injury or from malnutrition. Approximately 20 %–30 % of obese patients have some degree of micronutrient deficiency prior to bariatric surgery. After surgery, prolonged vomiting, changes in dietary patterns, bacterial overgrowth, altered flow of the biliopancreatic fluids, and bypass of the duodenal absorptive surface area, all contribute to the risk of complications from malnutrition. Because of this lifelong risk, bariatric surgery patients must continue to take vitamin supplements, undergo routine physician visits, and have scheduled lab tests. Patients should be followed by multidisciplinary teams when possible [33••, 34].

With the exception of Wernicke's encephalopathy, multiple micronutrient deficiencies have been proposed as the cause of most neurological complications of bariatric surgery. A combined expert medical guideline has been produced by the American Association of Clinical Endocrinologists, the Obesity Society, and the American Society for Metabolic & Bariatric Surgery. All bariatric surgery patients should take a multivitamin supplement twice daily and a calcium supplement once daily with further micronutrient supplementation as needed, particularly if a more malabsorptive procedure such as a biliopancreatic diversion has been performed. It is also recommend that all patients who present with neurologic complications, be tested for vitamin B12, folate, zinc, copper, vitamin A, and vitamin E deficiencies and treated accordingly.

When neurological complications from bariatric surgery do occur, all aspects of the nervous system are at risk. Disorders can be characterized by time and anatomical distribution. During the perioperative period, complications of the peripheral and enteric nervous system are most common. Thiamine deficiency causing Wernicke–Korsakoff syndrome is a rare perioperative complication which effects the central nervous system. Long term complications effect the central and peripheral nervous systems. Complications include myelopathies, polyneuropathies, mononeuropathies and polyradiculoneuropathies. In most cases of neurologic injury, patients respond to improved nutritional support.

Disclosure No potential conflicts of interest relevant to this article were reported.

Disclosures: Dr. Frantz has no relevant disclosures.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. • Flegal KM, Carroll MD, Kit BK, et al. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. JAMA. 2012;307(5):491–7. *Most current review of obesity trends in the United States*.
2. In *The Surgeon General's Call To Action To Prevent and Decrease Overweight and Obesity*. 2001: Rockville (MD).

3. Finkelstein EA, Trogdon JG, Cohen JW, et al. Annual medical spending attributable to obesity: payer-and service-specific estimates. Health Aff (Millwood). 2009;28(5):w822–31.
4. Riley, K. *FDA expands use of banding system for weight loss*. 2011; Available from: http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm245617.htm.
5. • Park CW, Torquati A. Physiology of weight loss surgery. Surg Clin North Am. 2011;91(6):1149–61. vii, *Excellent review of the physiologic effects of bariatric surgery.*
6. Pontiroli AE, Morabito A. Long-term prevention of mortality in morbid obesity through bariatric surgery: a systematic review and meta-analysis of trials performed with gastric banding and gastric bypass. Ann Surg. 2011;253(3):484–7.
7. Nguyen NT, Masoomi H, Magno CP, et al. Trends in use of bariatric surgery, 2003–2008. J Am Coll Surg. 2011;213(2):261–6.
8. Livingston EH. The incidence of bariatric surgery has plateaued in the U.S. Am J Surg. 2010;200(3):378–85.
9. Longitudinal Assessment of Bariatric Surgery, Flum DR, Belle SH, et al. Perioperative safety in the longitudinal assessment of bariatric surgery. N Engl J Med. 2009;361(5):445–54.
10. Buchwald H, Estok R, Fahrbach K, et al. Trends in mortality in bariatric surgery: a systematic review and meta-analysis. Surgery. 2007;142(4):621–32. discussion 632–5.
11. Padwal R, Klarenbach S, Wiebe N, et al. Bariatric surgery: a systematic review and network meta-analysis of randomized trials. Obes Rev. 2011;12(8):602–21.
12. DeMaria EJ, Pate V, Warthen M, et al. Baseline data from American Society for Metabolic and Bariatric Surgery-designated Bariatric Surgery Centers of Excellence using the Bariatric Outcomes Longitudinal Database. Surg Obes Relat Dis. 2010;6(4):347–55.
13. Macgregor AM, Thoburn EK. Meralgia paresthetica following bariatric surgery. Obes Surg. 1999;9(4):364–8.
14. Shafi MA, Pasricha PJ. Post-surgical and obstructive gastroparesis. Curr Gastroenterol Rep. 2007;9(4):280–5.
15. McGlinch BP, Que FG, Nelson JL, et al. Perioperative care of patients undergoing bariatric surgery. Mayo Clin Proc. 2006;81(10 Suppl):S25–33.
16. Shah RN, Izanec JL, Friedel DM, et al. Achalasia presenting after operative and nonoperative trauma. Dig Dis Sci. 2004;49(11–12):1818–21.
17. Cubattoli L, Barneschi C, Mastrocinque E, et al. Cardiac arrest after intragastric balloon insertion in a super-obese patient. Obes Surg. 2009;19(2):253–6.
18. Benevides ML, Nochi RJ. Junior: [Rhabdomyolysis secondary to gluteal compartment syndrome after bariatric surgery: case report.]. Rev Bras Anestesiol. 2006;56(4):408–12.
19. Rodriguez Uranga JJ, Ucles Sanchez AJ, Perez Diaz JM. Neuropathy of common sciatic nerve secondary to compartment syndrome as a complication after bariatric surgery. Neurologia. 2005;20(2):94–7.
20. Oluigbo N, Fallouh B, Suresh V. Cardiac arrest following adjustment of a gastric band. Obes Surg. 2008;18(6):750–2.
21. Abarbanel JM, Berginer VM, Osimani A, et al. Neurologic complications after gastric restriction surgery for morbid obesity. Neurology. 1987;37(2):196–200.
22. Thaisetthawatkul P, Collazo-Clavell ML, Sarr MG, et al. A controlled study of peripheral neuropathy after bariatric surgery. Neurology. 2004;63(8):1462–70.
23. Singh S, Nautiyal A. Neurologic complications of bariatric surgery. Mayo Clin Proc. 2005;80(1):136–7. author reply 137.
24. Mason ME, Jalagani H, Vinik AI. Metabolic complications of bariatric surgery: diagnosis and management issues. Gastroenterol Clin North Am. 2005;34(1):25–33.
25. Thaisetthawatkul P. Neuromuscular complications of bariatric surgery. Phys Med Rehabil Clin N Am. 2008;19(1):111–24. vii.
26. •• Kumar N. Neurologic presentations of nutritional deficiencies. Neurol Clin. 2010;28(1):107–70. *Excellent review of the neurologic manifestations of micronutrient deficiencies. First part of the review contains tables that can be used as a pocket reference. Does an excellent job going reviewing outlining diagnostic workup for nutrient deficiencies*.
27. •• Kazemi A, Frazier T, Cave M. Micronutrient-related neurologic complications following bariatric surgery. Curr Gastroenterol Rep. 2010;12(4):288–95. *Excellent review of the current literature exploring specific micronutrient deficiencies associated with neurologic dysfunction after bariatric surgery.*
28. Aasheim ET. Wernicke encephalopathy after bariatric surgery: a systematic review. Ann Surg. 2008;248(5):714–20.
29. Koffman BM, Greenfield II LJ, et al. Neurologic complications after surgery for obesity. Muscle Nerve. 2006;33(2):166–76.
30. Chang CG, Adams-Huet B, Provost DA. Acute post-gastric reduction surgery (APGARS) neuropathy. Obes Surg. 2004;14(2):182–9.
31. Juhasz-Pocsine K, Rudnicki SA, Archer RL, et al. Neurologic complications of gastric bypass surgery for morbid obesity. Neurology. 2007;68(21):1843–50.
32. Colquitt JL, Picot J, Loveman E, et al. Surgery for obesity. Cochrane Database Syst Rev. 2009;2009(2):CD003641.
33. •• Mechanick JI, Kushner RF, Sugerman HJ, et al. American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery medical guidelines for clinical practice for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient. Obesity (Silver Spring). 2009;17 Suppl 1:S1–70. v, *Comprehensive professional tri-society guidelines for the care of bariatric surgery patients. Recommended reference for all providers caring for patients after bariatric surgery.*
34. Bal B, Koch TR, Finelli FC, et al. Managing medical and surgical disorders after divided Roux-en-Y gastric bypass surgery. Nat Rev Gastroenterol Hepatol. 2010;7(6):320–34.