A Primary Care Provider's Guide to Diet and Nutrition After Spinal Cord Injury

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Abstract: Physiological changes that occur after spinal cord injury (SCI) are profound and affect almost every organ system in the human body. Energy balance is significantly altered due to motor paralysis, spasticity or flaccidity, neurogenic sarcopenia, neurogenic osteopenia, sympathetic nervous system disruption, and blunted anabolism. Energy expenditure is markedly reduced, whereas hypothalamic control of appetite and satiety is diminished, resulting in discordant energy intake. Ultimately, neurogenic obesity ensues as the result of a positive energy balance. Even though nutritional guidelines for persons with SCI have been available since 2009, the necessity for body composition assessment and total daily energy expenditure was insufficiently addressed such that most individuals with SCI continued in positive energy balance despite "adherence" to the guidelines. Macronutrients must be carefully assessed to optimize caloric intake, while micronutrient consumption may need to be supplemented in order to meet recommended daily allowances. Such a diet would emphasize foods with low caloric yet high nutrient density. This article reviews current literature regarding nutritional requirements for SCI and provides a straightforward plan for implementing more rigorous dietary interventions meant to address the obesity crisis in this especially vulnerable population. **Key words:** *body composition, diet, energy expenditure, energy intake, metabolism, obesity, paraplegia, spinal cord injury, tetraplegia*

Health Maintenance Checklist

1. Encourage the patient to reduce the number of cardiometabolic risk factors to <3, such as recommending a goal to:
   a. Reduce body fat to achieve body mass index ≤ 22 kg/m^2;
   b. Reduce triglycerides to ≤ 150 mg/dL and increase HDL-C to ≥ 40 mg/dL;
   c. Reduce fasting blood glucose to ≤ 100 mg/dL and/or HbA1c to < 7%.
2. Encourage exercise ≥ 150 minutes per week to increase energy expenditure sufficiently to achieve neutral of negative (fat loss) energy balance.
3. Encourage adoption of a heart-healthy diet with focus on fruits, vegetables, whole grains, low-fat dairy, poultry, fish, legumes, and nuts to achieve neutral or negative (fat loss) energy balance.
4. Recommend limiting saturated fat to 5% to 6% of total caloric intake.

Episodic Care Key Points

1. Determine resting energy expenditure (REE) every 1 to 3 years by indirect calorimetry to ensure accurate assessment of energy balance, and use REE to predict total daily energy expenditure (TDEE) to assist with nutritional counseling.
2. Annually assess body fat with dual-energy x-ray absorptiometry (DXA) or with obesity surrogate of body mass index ≥ 22 kg/m^2.
3. Review negative energy balance, targeting diet (intake) and exercise (expenditure) as needed for fat loss, lipid management, and target HbA1c.
4. Annually access fasting lipid profile or at minimum HDL-C and triglycerides and consider a prescription of statin or extended-release niacin to achieve target triglycerides ≤ 150 mg/dL and HDL-C ≥ 40 mg/dL.
5. Assess fasting blood glucose and HbA1c every 3 years, and consider prescription of metformin (first-line agent) to achieve target HbA1c <7%; second- and third-line agents may be required.

Case Report

A 54-year-old man with C5 American Spinal Injury Association Impairment Scale (AIS) A tetraplegia for 35 years presents to you for establishment of primary care associated with a recent move from across the country. He has no family history of heart disease, diabetes, hypertension, or dyslipidemia, but he includes each of these diagnoses in his past medical history, which is also notable for autonomic dysreflexia, neurogenic bladder, neurogenic bowel, osteoporosis, spasticity,

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and multiple sacral/ischial pressure injuries that are now healed. Medications include lisinopril, metoprolol, pravastatin, metformin, dulaglutide, tolteridine LA, and baclofen. He is frustrated that despite his adherence to his 1,800 kcal/d American Diabetes Association (ADA) diet, his hypertension, dyslipidemia, and diabetes appear to be getting worse. He does not exercise but had some perceived benefit to functional electrical stimulation leg cycle ergometry more than 10 years ago. On physical examination, blood pressure is 140/85 mm Hg, heart rate is 77 beats/min, height (by patient report) is 5 ft 9 in., and weight is 200 pounds. Heart and lung exam are normal, spasticity is minimal (Modified Ashworth 1.5), and neurological examination is otherwise consistent with C5 AIS A tetraplegia. Labs are notable for high-density lipoprotein cholesterol (HDL-C) of 33 mg/dL, triglycerides 150 mg/dL, hemoglobin A1c (HbA1c) is 8%, and fasting blood glucose (FBG) is 112 mg/dL.

Background

Spinal cord injury (SCI) affects almost every organ system in the body through its influence on somatic dermatomes and myotomes as well as the autonomic nervous system. Higher levels of SCI result in the ability to voluntarily activate fewer myotomes (muscle groups), with spastic paralysis present in myotomes below the level of injury (BLOI). Bone mass is also rapidly depleted with the loss of mechanical loading, neurogenic activation, and intermittent autonomic dysreflexia. Since the sympathetic nervous system arises from the thoracolumbar regions of the spinal cord, sympathetic blunting occurs in cervical and thoracic SCI, further diminishing resting metabolism. Unless dietary restriction is practiced, neurogenic obesity results due to an obligatory sarcopenia, neurogenic osteopenia, sympathetic dysfunction, and blunted anabolism. Obesity then mediates the metabolic syndrome that consists of central obesity, vascular inflammation, hypertension, dyslipidemia, and glucose intolerance.[1] A recent study indicated that more than 75% of 477 veterans with chronic SCI were obese, and more than 50% had metabolic syndrome.[2] To prevent these disease entities, energy balance must be initiated soon in the course of SCI; to reverse them requires negative energy balance.

Energy Balance

"The concept of energy balance reflects an ever-dynamic relationship between relative rates of change for energy intake and energy expenditure."[3(p336)] Energy intake reflects the number of calories consumed through the ingestion and digestion of macronutrients comprised of different caloric densities. Carbohydrates and proteins contain 4 kcal/g of tissue, whereas fats contain 9 kcal/g and alcohol has a caloric density of 7 kcal/g. Nutrition tables and software have been developed to quickly and accurately determine the total daily caloric intake based on the relative number of grams of carbohydrates, proteins, fats, and alcohol consumed as reported by a studied individual. Ideally, in research studies total caloric intake would be strictly measured and controlled by a clinical registered dietician (RD) who prepares and administers all fluids and foodstuffs consumed. Practically speaking, this is rarely done because of the rigor, limited choices, and social and vocational activities of the studied individual. Instead, self-reported 1-day, 3-day, or 7-day dietary recall logs have traditionally been utilized to estimate caloric intake, and they rely on the memory, accuracy, and ability of the studied individual to report consumption.

Energy expenditure is even more complex and difficult to quantify. Total daily energy expenditure (TDEE) is the sum of basal metabolic rate (BMR), the thermic effect of activity (TEA), and the thermic effect of food (TEF). BMR, often used interchangeably with resting energy expenditure (REE), is the minimal energy expenditure required to sustain life; it represents ~70% of the TDEE, and it is highly correlated with an individual's fat-free mass (FFM), which is comprised of active metabolic tissue, muscles, bones, and organs.[4] TEA is somewhat dependent upon REE, particularly muscle and bone mass, but it represents the additional calories expended during daily physical activity. For persons with SCI, ongoing spasticity may also contribute to REE and TEA.[5] TEF

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represents the energy expended during digestion and is approximately 8% of TDEE for most individuals. Although TDEE tables for able-bodied (AB) individuals have been derived to estimate energy expenditure requirements,[6] the sarcopenia, osteoporosis, sympathetic nervous system, and anabolic blunting associated with SCI make it uniquely inappropriate to use those same tables for persons with SCI. Fortunately, Collins et al developed a similar compendium for persons with SCI including 27 activities across 170 individuals with SCI; REE averaged 2.7 mL O_2/kg/min compared to the standard 3.5 mL O_2/kg/min for AB individuals.[7] A recent systematic review comparing the accuracy of estimated and measured REE among persons with chronic SCI demonstrated large and significant differences, even when correcting for FFM.[8] Current recommendations indicate the need for indirect calorimetry to accurately determine REE.[9] REE should be assessed even during the acute trauma phase of SCI, as overfeeding early in the SCI course can contribute to inaccuracies and obesity even during the acute rehabilitation.[10-12] Once determined, REE can be multiplied by a correction factor of 1.15 to estimate TDEE.[13] When TDEE has been determined or estimated, negative energy balance can be achieved through increasing physical activity and/or decreasing calorie intake.

Diet Composition for Persons With SCI

Nutritional guidelines for persons with SCI were published in 2009, but they were fairly nonspecific and essentially mirrored the guidelines for AB individuals, with the caveat that metabolism was likely altered and protein reserves were depleted in the SCI population due to sarcopenia.[14] Several studies since then have reported macro- and micronutrient diet composition for persons with SCI with variable findings; not all included energy expenditure to determine energy balance.[15-20] The recent meta-analysis by Farkas et al[8] compared findings for persons with SCI to the current 2015-2020 Dietary Guidelines for Americans[21] with findings of positive energy balance despite diminished caloric intake, excess protein and carbohydrate intake compared to AB recommendations, low fiber intake relative to AB recommendations, and lower than recommended intake of vitamins A, B_5, B_7, C, D, and E.[22] Several mineral deficiencies were also reported for individuals with SCI, notably calcium, magnesium, and potassium.[22] Although fat intake for individuals with SCI appeared to fall within recommended daily allowance in the meta-analysis, several previous studies have demonstrated excess dietary fat and particularly saturated fat intake above the Dietary Guidelines.[17,20,23]

Several unique aspects of SCI must also be taken into account when considering nutrition counseling for persons with SCI. Although the lateral (appetite center) and ventromedial (satiety center) nuclei of the hypothalamus remain intact following SCI, several of the usual neurological and endocrine factors affecting these centers may be blunted due to the unique physiological disruption of afferent pathways and sympathetic nervous system.[3] Subsequently, physiological cues to suppress appetite that are present in AB are likely blunted or absent in persons with SCI. Body composition is markedly altered after SCI, with profound loss of metabolically active fat-free mass and commensurate increase in fat mass, such that body weight and body mass index (BMI) grossly underestimate obesity in SCI.[24] Persons with SCI whose BMI is 22 kg/m^2 or greater should be considered obese, and indirect calorimetry should be used to determine their actual REE.[9] Insensate skin and paralysis puts persons with SCI at significant risk for pressure injuries, and the low protein reservoir due to sarcopenia makes it difficult to provide adequate protein intake for wound healing without exceeding energy needs. Neurogenic bowel after SCI requires daily or every other day bowel care programs that typically include the need for fiber, chemical suppositories, and digital stimulation to prevent constipation or fecal incontinence. High-fat foods, spicy foods, caffeine, and other stimulants can alter the gut dynamics resulting in unexpected fecal incontinence for the person with SCI who has paralyzed sphincters. While fiber is advocated to facilitate gut motility, failure to drink adequate fluids in the attempt to reduce bladder complications may result in constipation

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and further autonomic compromise. Hence dietary counseling can be a tricky proposition for individuals with SCI.

Low Energy, High Nutrient Density Foods

The 2015-2020 Dietary Guidelines for Americans recommend low energy, high nutrient dense foods to meet nutrient needs within calorie limits, with a variety of nutrient-dense foods across and within all food groups in recommended amounts.[21] More specific recommendations for persons with SCI include the adoption of a heart-healthy nutrition plan focusing on fruits, vegetables, whole grains, low-fat dairy, poultry, fish, legumes, and nontropical vegetable oils and nuts while limiting sweets, sugar-sweetened beverages, and red meats.[9] It is also recommended that saturated fats be limited to less than 6% of total caloric intake, sodium intake be limited to 2,400 mg or less for persons with hypertension, and the DASH (Dietary Approaches to Stop Hypertension) nutritional plan or Mediterranean nutritional plan be adopted if hypertension or other cardiometabolic risk factors are present.[9]

For fat loss that is likely to facilitate future weight maintenance, reduction of caloric intake sufficient to allow negative energy balance of 100-200 kcal/d is recommended to promote weight loss of 1 to 2 pounds/week.[3] More rapid weight loss is likely to incur undesirable loss of muscle and water, which will ultimately further reduce metabolic rate and require additional reductions in energy intake. Persons with SCI should consider a target BMI of 22 kg/m^2 or less or, when available, body composition assessment of less than 22% body fat for men and less than 35% body fat for women.[9]

Case Resolution

For the man in the case described at the beginning of the article, it was likely that his energy intake was exceeding his energy expenditure despite his compliance with his ADA diet. He was sent for additional testing to determine his resting metabolic rate and body composition. Indirect calorimetry was performed and demonstrated REE of 790 kcal/d. Using the equation from Farkas et al, TDEE was determined to be 790 kcal/d x 1.15 (i.e., 908.5 kcal/d). His 1,800 kcal/d ADA diet was providing twice the calories required to maintain energy balance; excess calories were being stored as adipose tissue, worsening his obesity and all components of the metabolic syndrome, including insulin resistance, dyslipidemia, and hypertension. Although BMI was calculated to be 29.5 kg/m^2, body composition determined by dual-energy X-ray absorptiometry (DXA) demonstrated 52% body fat, such that his actual fat mass was 104 pounds.

Our recommendations were to increase energy expenditure by 100 kcal/d using a functional electrical stimulation leg cycle ergometer (FESLCE) and to reduce his intake to 850 kcal/d. The exercise was also expected to increase lower extremity muscle and bone mass, with a subsequent increase in REE. He was highly motivated and fortunate to have resources to purchase an FESLCE and hire a fitness coach who was familiar with SCI and the FESLCE. He was referred to a dietician who had specialty experience with SCI, and she initiated a Mediterranean diet within the energy restrictions listed above but insisted that he also take a medical grade multivitamin with minerals and that he check in with her weekly. A sample meal plan for a day included one serving of poached eggs with black beans and spinach for breakfast (267 kcal), one serving of almond butter and celery for lunch (217 kcal), one serving of Italian vegetable soup and one serving of a spinach recipe for dinner (243 kcal), a single serving of bell pepper and hummus (101 kcal) as a snack. He was encouraged to drink 2 liters water daily and to avoid fluid consumption of alcohol and sweetened beverages. Based on an anticipated negative energy balance of 150 kcal/d, he would be expected to lose 1 pound of fat (3500 kcal) every 3 weeks. An additional option discussed was to reduce his antispasticity medication to allow increased spasms and spasticity throughout the day, which would increase his REE and TEA, however, this could result in more problematic spasticity. Finally, it was recommended that he follow up with his SCI physician monthly for 3 months, then at 3 months, 6 months, and 12 months to ensure safe weight loss and to wean medications as indicated with the anticipated loss of adipose tissue.

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Conclusion

The dietary and nutritional needs for persons with SCI have been understudied and likely understated due to profound changes in body composition associated with paralysis, sarcopenia, osteopenia, blunted anabolism, and blunted energy metabolism. Additionally, appetite and satiety signals enjoyed by the AB population are likely blunted or largely absent in high SCI. Neurogenic obesity almost always occurs in persons with chronic SCI, resulting in the cardiometabolic syndrome of insulin resistance, dyslipidemia, hypertension, and ultimately accelerated arteriosclerosis. Although pharmacological management is often indicated, the primary intervention required for long-term management includes behavior modification including diet and exercise. Initial evaluation of body composition and REE is warranted to ensure appropriate dietary and activity modifications, while close multidisciplinary follow up is recommended to ensure safe weight loss based on negative energy balance that is not excessive.

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