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CASE REPORT

Effective management of spasticity and impacts on weight change and resting energy expenditure in a female with spinal cord injury: a case report

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INTRODUCTION: The impact of muscle spasticity on weight change and energy expenditure after spinal cord injury (SCI) is not well understood.

CASE PRESENTATION: This case study reports changes to body weight and resting energy expenditure (REE) in a 36-year-old female (T3 AIS A SCI; 80 kg; body mass index = 28 kg m^{-2} at injury) requiring escalating therapies to manage severe spasticity. Body weight, spasticity medications and fasted REE (measured using indirect calorimetry, canopy hood) were recorded at 4, 16, 17, 20 and 44 months post injury. Spasticity was assessed at each time point using the Modified Ashworth Scale (MAS). At 4 months post injury, REE was high (1710 kcal per day) corresponding with severe spasticity in the lower limbs (4 on the MAS). Over the following 12 months, the patient experienced an 8 kg weight loss, visible lower limb muscle wasting and a 30% reduction in REE while requiring increasing drug therapies for spasticity. With insertion of an intrathecal Baclofen pump at 17 months and cessation of other medications, spasticity improved markedly and weight increased by 6 kg in 27 months without any significant change to REE (mean = 1260 kcal ± 2%).

DISCUSSION: Effective management of spasticity with intrathecal Baclofen appears to be associated with weight gain but not REE. Without body composition and activity energy expenditure data, this observation is difficult to explain. Regardless, routine weight monitoring with appropriate dietary counselling should be considered in this patient group to help prevent unintentional weight gain.

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INTRODUCTION

Spasticity is a common secondary complication of spinal cord injury (SCI). It is generally characterised by an increase in muscle tone with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex.[1] Spasticity is experienced to some degree by up to 80% of people with chronic SCI.[2,3] The negative consequences of spasticity are varied and may include pain, reduced ability to perform activities of daily living, increased risk of developing pressure injuries and reduced quality of life.[4–6]

Some individuals report that spasticity can have a positive impact and may in fact assist some activities of daily living such as dressing,[5] improve stability in sitting and standing, and increase venous return.[7] Further, spasticity has been associated with preservation of skeletal muscle size and fat-free mass.[8] While fat-free mass is known to be a strong predictor of resting energy expenditure (REE) in people with SCI,[9] small cross-sectional studies have failed to find an association between spasticity and REE.[10,11]

Resting energy expenditure (also known as basal metabolic rate) is the energy required to sustain basic processes vital for life and comprises approximately 70% of daily energy needs in a healthy person.[12] Activity energy expenditure and the thermic effect of food contribute to the remainder of total daily energy needs, comprising 20% and 10% of total daily energy needs in healthy people, respectively.[9,12] Indirect calorimetry is the gold standard for assessing REE following SCI as predictive equations have been found to overestimate energy requirements in this group.[13] Previous research has indicated good repeatability of indirect calorimetry following SCI, making it an appropriate tool to track changes to resting energy requirements over time.[14]

The presence or severity of spasticity is not generally considered when assessing nutritional needs in standard practice. Despite findings from previous studies, there are no data demonstrating whether problematic spasticity and its successful treatment over time has any influence on weight change or REE in people with SCI. This case report describes changes to body weight in a female with SCI requiring escalating drug therapies to manage severe spasticity and presents longitudinal REE data collected over the course of treatment.

CASE PRESENTATION

A 36-year-old female was admitted to the state-wide spinal injury rehabilitation unit following a motor vehicle accident that had occurred 4 weeks prior. Her injuries included an unstable C2 fracture, T3 and T6 column chance fractures and a traumatic thoracic aortic transection at T4 resulting in a T3 AIS A SCI. Medical history prior to the motor vehicle accident included depression and previous smoker. At the time of injury, the patient reported a usual body weight of 80 kg and a height of 170 cm (body mass index = 27.7 kg m^{-2}).

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Table 1. Modified Ashworth scale[15]

Rating	*Definition*
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of a range of motion when the affected part(s) are moved in flexion or extension
1+	Slight increase in muscle tone, manifested by a catch, followed my minimal resistance throughout the remainder (less than half) of the range of motion
2	More marked increase in muscle tone through most of the range of motion, but affected part(s) easily moved
3	Considerable increase in muscle tone, passive movement difficult
4	Affected part(s) rigid in flexion or extension

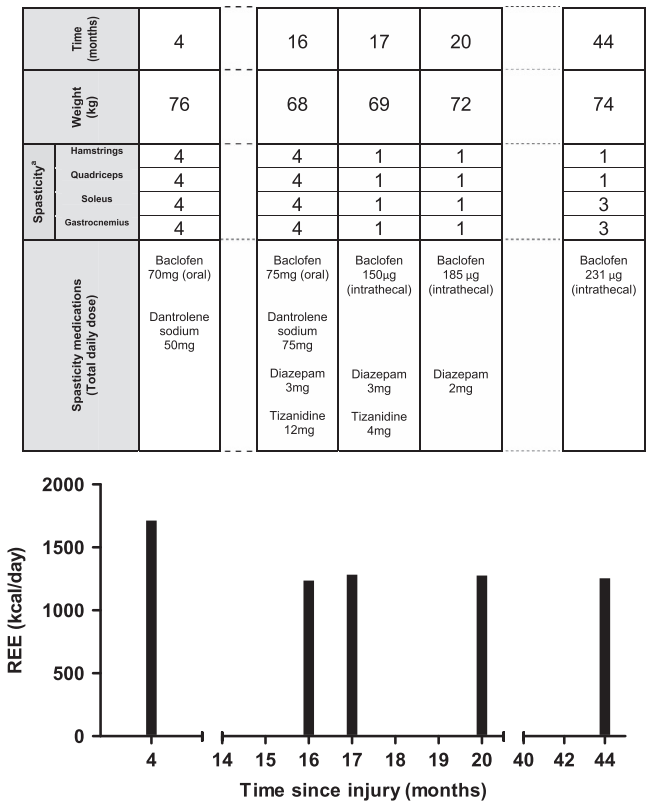

Figure 1. Changes to body weight, spasticity, medications and resting energy expenditure over 44 months following spinal cord injury.

Information was collected at multiple time points, spanning from the initial inpatient rehabilitation until nearly 4 years post injury. Body weight was measured using wheelchair scales with the wheelchair weight deducted (Wedderburn WM501, NSW), anti-spasticity medications and doses (including baclofen, dantrolene sodium, diazepam and tizanidine) were recorded and spasticity was assessed by the treating physiotherapist using the Modified Ashworth Scale (Table 1).[15] Measurement of REE was performed at the bedside using indirect calorimetry (ventilated canopy hood; TrueOne 2400 Metabolic Measurement System; Parvo Medics, Salt Lake City, UT, USA). Gas and volume calibration occurred before each measurement. Tests were undertaken after an overnight fast (⩾8 h), following morning medications but prior to bowel therapy. The patient was asked to rest quietly, lying supine for approximately 20 min before the test commenced. Inspired oxygen and expired carbon dioxide was measured for a minimum of 20 min, with the first 5 min of data discarded as per best practice recommendations[16] and steady-state REE calculated using the Weir equation with $< 10\%$ coefficient of variation in VO_2 and VCO_2. Values for REE are reported in kcal per day to remain consistent with existing literature on this topic.

At the time of the first REE measurement (4 months post injury), weight had decreased to 76 kg (−4 kg) and had been stable for 4 weeks. Despite 70 mg of oral baclofen and 50 mg of dantrolene sodium daily, spasticity was assessed as a rating of 4 on the modified Ashworth scale in the hamstrings, quadriceps, gastrocnemius and soleus bilaterally. The patient's REE was considerably higher than that predicted (measured REE 1712 kcal per day vs Harris–Benedict predicted 1532 kcal per day). At the time of testing, there was no evidence of infections, no febrile episodes in the 7 days prior and no recent surgeries. No further measures of REE were undertaken during the initial rehabilitation admission, and the patient was discharged 7 months after her date of injury on a combination of 75 mg oral baclofen, 100 mg of dantrolene sodium and 3 mg of diazapam for ongoing spasticity.

After discharge, the patient was followed up at the SCI outpatient clinic. Her spasticity remained severe, preventing self-catherisation (requiring an indwelling catheter) and contributing to an erratic bowel routine. The patient reported significant anxiety and an inability to participate in social events due to her spasticity. Botulinium toxin was deemed an inappropriate treatment given the generalised nature of the spasms and the decision was made for a trial of tizanidine, a drug not approved for use in

Australia at the time. Following appropriate approvals, a trial of tizandine was commenced in addition to existing spasticity medications and the dose escalated up the maximum of 8 mg three times per day over a 2-month period. While the patient reported a slight decrease in spasm frequency and decreased 'bounce' in her lower limbs, her spasticity continued to be severe and the decision was made to proceed with a trial of intrathecal baclofen, which proved successful.

Subsequent longitudinal measures of weight, spasticity, medications and REE were undertaken prior to pump insertion (16 months post injury), 14 days post pump insertion (17 months post injury), once an effective stable dose of intrathecal baclofen was established and other spasticity medications were ceased (20 months post injury) and a final follow-up measurement (44 months post injury).

See Figure 1 for a summary of body weight, spasticity severity, spasticity medications and REE at 4, 16, 17, 20 and 44 months post SCI. There was a significant decrease in body weight and REE at 16 months post SCI (−477kcal per day and − 8 kg, respectively) despite ongoing severe spasticity prior to intrathecal baclofen pump insertion. Following pump insertion at 17 months, weight increased by 6 kg while REE stabilised (fluctuating between 1235 and 1282 kcal per day; ~200 kcal less than Harris–Benedict predicted REE). Spasticity improved significantly, with the patient reporting an increase in independence, ability to self-catheterise and successfully manage her own bowel therapy. While there was some return of spasticity in the gastrocnemius and soleus bilaterally at 44 months, this was not associated with any increases in REE nor did the patient report any impact on independence.

DISCUSSION

A reduction and stabilisation of spasticity with intrathecal baclofen was associated with substantial unintended weight gain in this female patient with T3 complete paraplegia. Similar to previous cross-sectional studies, there did not appear to be a relationship between spasticity severity and REE.

It is possible that spasticity may influence energy balance through effects on activity energy expenditure, which is difficult to measure in clinical practice and was not captured here. Previous studies have compared spasticity with low-tension resistance training.[11] Measurement of REE in a resting, supine position, in the absence of active spasms is unlikely to capture any potential increases in energy expenditure attributable to spasticity.

The initial REE measurement was higher than that predicted and conflicts with previous work that shows common energy prediction equations greatly overestimate REE in this patient group.[13] All subsequent REE values were ~200 kcal below Harris–Benedict-predicted REE (~15% overestimate of REE using the Harris–Benedict equation) and consistent with reported literature in patients with SCI.[13] The observed reduction in REE at 16 months is possibly due to the introduction of new oral medication and alterations to body composition. Tizanidine is a selective, short-acting α-agonist that reduces sympathetic nervous system activity to decrease muscle spasticity. In healthy volunteers, tizanidine has been shown to reduce REE by 9% at doses of 12 mg per day.[17] As this medication had been administered prior to REE testing, it may explain part of the reduction in REE. Although changes to body composition were not captured, a subjective loss of muscle bulk in the lower limbs was observed and body weight decreased by 8 kg over 12 months, which may have contributed to the remaining discrepancy in REE between 4 and 16 months.

Following baclofen pump insertion at 17 months, the increase in body weight did not correspond with an increase in REE. This increases the likelihood of gains in fat mass, which is less metabolically active compared with fat-free mass[18] and is associated with increased inflammation and risk of developing chronic disease.[19] Routine weight monitoring in individuals undergoing treatment for severe spasticity should be considered to aid prevention of undesirable weight gain, which is a recognised phenomenon following SCI. Dietary assessment and counselling may also be indicated to help individuals to achieve energy balance.

This case report is the first to monitor body weight changes and REE throughout the decision-making process to manage severe spasticity in a person with SCI. Indirect calorimetry measures were performed on the same machine as per the standardised protocol, by the same clinician, each time. High-quality data were obtained, with the coefficient of variation for VO_2 and VCO_2 less than 5% for all measurements. The main limitation of this case report is the absence of body composition data, which would help to further explain the changes to body weight and REE, and the absence of activity energy expenditure data, which is difficult to capture in clinical practice. The length of time between the first and second REE measurements also makes it difficult to explain other factors that may have contributed to the large weight loss and decrease in REE observed initially.

Further research examining the nutrition-related impacts of spasticity and its management after SCI is warranted. Individuals with spasticity who are resistant to first-line treatment may be suitable candidates to commence longitudinal monitoring of body weight, body composition and REE throughout trials of second- and third-line treatments. Provocation of spasticity while undergoing objective assessment of energy requirements may also be useful. Alternatively, objective measurement of activity energy expenditure should also be considered, if available.

CONCLUSION

Effective management of spasticity with intrathecal Baclofen appears to be associated with substantial weight gain. While improvements in spasticity did not correspond with a decrease in REE, spasticity may influence energy balance through impacts on activity energy expenditure. Routine weight monitoring with appropriate dietary counselling should be considered in this patient group to help prevent unintended weight gain.

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This work was granted an exemption from requiring formal ethical review by the Metro South Human Research Ethics committee. Patient consent was obtained for this information to be published.

COMPETING INTERESTS

The authors declare no conflict of interest.

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