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Chronic hypothermia and energy expenditure in a neurodevelopmentally disabled patient: a case study

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Abstract

Hypothermia is defined as a core body temperature of <35°C and results in a decrease in measured resting energy expenditure. A 51-year-old mentally disabled patient experienced chronic hypothermia from neurologic sequelae. Because of her continued weight gain and increased body fat in the presence of presumed hypocaloric nutrition, indirect calorimetry measurements were performed twice in a 3-month period. The resting energy expenditure measurements prompted a reduction of her daily caloric intake to prevent further overfeeding. Hypothermia reduces oxygen consumption and, as a consequence, decreases resting energy expenditure. In patients for whom chronic hypothermia is a problem, nutritional intake must be adjusted to prevent overfeeding, excessive weight gain, and the long-term complications of an excess of total calories.

Assessing energy needs in patients with neurodevelopmental disabilities is challenging. The severity of their disease and the extent of ambulation are important factors affecting the energy requirements of these patients.[1-4] Body temperature is also a critical modulator that influences energy requirements. Hyperthermia and hypothermia divergently affect the resting energy expenditure (REE).[5-7] The resulting hypermetabolism observed in patients with hyperthermia is well established.[8] However, the influence of hypothermia upon energy expenditure may be overlooked.

Hypothermja results in a decrease in oxygen consumption (V_{O_2}), carbon dioxide production (V_{CO_2}) and ultimately in a decrease in measured REE.[8] Patients with severe neurodevelopmental disabilities may often experience episodes of chronic hypothermia.Warming techniques are used to prevent heat loss; however, when the patient has prolonged states of hypothermia, nutrition adjustments should be made.

Case Study

A 51-year-old female resident of a state developmental rehabilitation and long-term care facility was assessed by the pharmacy nutrition support consultants. The resident had congenital hydrocephalus secondary to toxoplasmosis and generalized tonic clonic seizures. She was confined to a wheelchair and was immobile secondary to spastic quadriplegia. The patient was 165 cm tall (determined by segmented height determinations) and weighed 52 kg. She experienced frequent episodes of hypothermia presumably as a result of neurologic sequelae from her chronic seizure disorder. An electric blanket, warm clothes, and a ski hat were used to prevent heat loss, but the patient still routinely remained hypothermic. Over several months, the

Table 1. Monthly weights and nutritional intake

Month/Year	Weight	% Body fat	Nutrition product	Regimen	Caloric intake (kcal/d)
May 1995	52.3		Osmolite*	80 mL/hr × 18 hr	1525
June 1995	52.1			60 mL/hr × 18 hr	1145
July 1995	51.8		Jevity*	40 mL/hr × 18 hr	765
August 1995	53.4			60 mL/hr × 18 hr	1145
September 1995	52.7				
October 1995	53.6	30			
November 1995	54				
December 1995	55			60 mL/hr × 15 hr	950
January 1996	54.6	32			
February 1996	56.1			55 mL/hr × 15 hr	875
March 1996	57.6				
April 1996	58.2	33			
May 1996	56.9			60 mL/hr × 12 hr	750
June 1996	57.1				

*Ross Products Division, Abbott Laboratories, Columbus, OH

patient had continued to gain weight even with presumed hypocaloric nutrition support. Two separate evaluations of thyroid function within several months ruled out hypothyroidism as a potential etiology for her hypometabolism and hypothermia. She did not ingest any nutrition by mouth. Enteral nutrition, with a 1.06 kcal/mL fiber- containing isotonic feeding formulation, was given via gastrostomy at 60 mL/h for 18 hours to provide a total of 1145 kcal and 48 g protein per day. A nutrition assessment revealed that the patient had abundant fat stores with normal serum proteins, which prompted a reduction in her caloric intake. However, she continued to gain weight without evidence of edema. To assess body composition and weight, measurements of tricep and calf fat skin folds were performed periodically with a skin folds caliper (Lange Skinfolds Caliper; Cambridge Scientific Industries, Cambridge, MD) and total body fat estimated using the mean of three measurements from two different body sites according to the method of Slaughter et al.[10] Body weight was measured by use of a sling every 2 to 4 weeks. These data verified our clinical observations of abundant fat stores in this patient. Table 1 depicts body weight, body fat, and changes in her nutritional regimen.

To assess the patient's REE, indirect calorimetry measurements were made twice in a 3-month period. Bedside measurements of V_{O_2}, V_{CO_2}, and respiratory quotient (RQ) were performed using a portable metabolic cart (MetaScope II; Colorado Medtech, Boulder, CO), according to the techniques and procedures of Feurer and Mullen.[11] Enteral nutrition was disconnected 2 hours before indirect calorimetry was done. Metabolic measurements were performed using a canopy system. The patient lay quietly awake in her bed during the entire procedure. Steady-state equilibration was determined when the patient maintained at least five consecutive 1-minute sampling intervals with a coefficient variation of < 5% for V_{O_2}, V_{CO_2}, and RQ. Results are given in Table 2. On the basis of the first indirect calorimetry measurement, measured REE was 612 kcal/d. The patient was receiving 1.7 times her measured energy expenditure, resulting in weight gain. This prompted the reduction of her enteral caloric intake to 950 kcal/d. A second measurement conducted 3 months later indicated that the patient's measured REE was 815 kcal/d

Table 2. Energy expenditure assessment

	Measurement 1 (October 1995)	Measurement 2 (January 1996)
Temperature (°C)	35.2	36.4
V_{O_2} (mL/min)	88	117
V_{CO_2} (mL/min)	75	101
RQ	0.86	0.86
REE (kcal/kg/d)	11.4	14.9
REE (kcal/kg$^{0.75}$/d)	30.9	40.6
REE (kcal/kg FFM/d)	16.3	22
% BEE	50	67
Weight (kg)	53.6	54.6

RQ, respiratory quotient; REE, resting energy expenditure; FFM, fat-free mass; BEE, basal energy expenditure as predicted by the Harris-Benedict equation for women[11]

at a slightly higher body temperature (Table 2). A caloric intake of 1.1 × REE is appropriate for weight maintenance in a nonambulatory patient[12] but because of her continued hypothermic state and abundant fat stores, her caloric intake was reduced to 750 kcal/d. Protein intake was empirically decreased proportionately to maintain a calorie to nitrogen ratio of 150:1; however, optimal protein requirements of this population are unknown. A vitamin and mineral supplement was initiated because this amount of enteral nutrition does not provide 100% of the recommended dietary allowance (RDA) for vitamins and minerals in adults. Although it is possible that vitamin and mineral requirements parallel energy requirements, we opted to supplement her vitamin and mineral intake to meet 100% of the RDA because she is approaching menopause and the vitamin and mineral needs of this unique population are unknown.

Discussion

Hypothermia is defined by Reuler[13] as a core temperature < 35°C and is categorized into the following three classes: acute, subacute, and chronic.[9] Acute hypothermia is a rapid decrease in body temperature that results in minimal metabolic and physiological changes. Subacute hypothermia is a slower decrease in body temperature from prolonged cold exposure. Chronic hypothermia occurs in conjunction with an underlying disorder such as a neurological disorder, seizures, diabetes, alcoholism, myxedema, or atherosclerosis. Medications also have been implicated in causing hypothermia.[9] In cases of chronic and subacute hypothermia, metabolic disorders are frequently severe and such persons may subsequently become hypothermic even at room temperature.[9]

The temperature of the body is regulated almost entirely by central nervous system feedback mechanisms, and almost all of these operate through temperature-regulating centers located in the hypothalamus. The hypothalamic thermostat responds to cold and warm signals from mainly peripheral receptors and stimulates heat controlling mechanisms (eg, shivering or sweating), thereby maintaining the core body temperature at 36.5 to 37.5°C.[14] A disruption in the temperature-regulating centers from anatomic (stroke, seizure, or head trauma) or

pathophysiological (hypothyroidism or hypoadrenalism) causes may lead to chronic hypothermia.[13,14]

During hypothermia, there is a decrease in oxygen consumption and a subsequent decrease in metabolic demand. This principle has been applied in cardiopulmonary bypass procedures and treatment of patients with closed head injuries (CHI).[15] Hypothermia results in a depression of cerebral electrical activity and metabolism, thereby reducing the accumulation of toxic metabolites, preserving high-energy phosphates, and improving ischemic outcome in CHI.[15] The resulting hypothermic effect from induction, as well as from cold exposure, is usually short term and rarely has nutritional implications. Modifications in nutritional regimens are applicable to those patients in whom prolonged hypothermia exists.

Although the metabolic effects of hypothermia have not been formulated directly, the metabolic effects of hyperthermia have been addressed by Du Bois.[16] These data demonstrate that there is a 13% increase in measured REE for every degree Celsius rise in body temperature. However, Du Bois suggested that the relationship between body temperature and energy metabolism may be more variable as described by van't Hoff's Law. This law states that for every rise in temperature of 10°C, the velocity of chemical reactions increases between twofold and threefold. Therefore, a 10% to 20% increase in measured REE can be expected with a 1°C rise in body temperature. This principle is referred to as the Q_{10} effect.[17] Studies have corroborated the existence of this effect, although there is a wide variation in clinical practice and extenuating circumstances (eg, burn, cancer, or infection) that will account for variable metabolic demands.[17,18]

Although the Q_{10} effect or van't Hoff's Law is often described in hyperthermia, the opposite is presumed to be applicable for hypothermia. The Q_{10} effect is a logarithmic linear equation; therefore a degree Celsius below normal core body temperature would be expected to result in a 10% reduction in V_{O2} and a decrease in REE.[17] Hypothermic patients exhibit a decrease in V_{O2}, V_{CO2}, and REE, and basal metabolic rate may fall as much as 50% of normal at 28°C.[13]

A substantial decrease in measured REE with hypothermia was observed in this case study. At a body temperature of 35.2°C, the measured REE was 612 kcal/d, which was 50% of her predicted energy expenditure (1221 kcal); at a body temperature of 36.4°C, her measured REE was 815 kcal, which was 67% of her predicted energy expenditure (1218 kcal; Table 2). Therefore, a decrease in body temperature of 1.2°C resulted in a 25% decrease in measured REE. Similar decreases were observed when energy expenditure was normalized to body weight (23%), metabolic body sir (24%), or fat-free mass (26%; Table 2). Because the patient's measured REE was lowered more than what was anticipated, it may be presumed that hypothermia does not entirely account for her decrease in energy expenditure. Lowering of her energy expenditure may also be attributed to inactivity and presumed diminished muscle capacity secondary to spastic quadriplegia.[1,19] Lack of ambulation significantly reduced total energy expenditure and muscle wasting ensues. Patients with cerebral palsy and myelodysplasia who cannot ambulate have a significantly lower energy expenditure than those who are mobile.[1] Normothermic resting energy expenditure was lower than predicted in this nonambulatory patient. Presumably, this can be

attributed to the combined effect of decreased physical activity and muscle mass atrophy because skeletal muscle metabolism is a major determinant of resting energy expenditure.[20,21] Unfortunately, without direct measurement of muscle mass by more sophisticated techniques, decreased skeletal muscle mass cannot solely account for the decrease in resting energy expenditure.

This case report depicts an individual with chronic nonshivering hypothermia; she was incapable of regulating her body temperature, similar to what is seen in poikilothermic animals. Poikilothermia is defined as a fluctuation in core temperature of > 2°C as a result of changes in ambient temperature.[22] Poikilothermia is common in other animal species such as reptiles and fish. Some animals such as the marmot and black bear behave as both homeotherms and poikiliotherms; they are homeothermic when it is warm and hibernate during the winter.[17] However, it is rare in humans.[22] The etiology of this rare disorder in humans is not entirely clear and is often multifaceted. Altered body temperature homeostasis with non shivering hypothermia can occur in patients with severe brain disease, epilepsy, stroke, seizure disorders, or in patients with a transection above the first thoracic segment of the spinal cord.[13,14,17,22]

The propensity to gain weight is apparent in our patient as a result of her chronic hypothermic state (Table 1). The goal of therapy in this patient is to prevent overfeeding, which has been associated with fatty liver,[23] increased V_{CO_2},[24] and in longterm patients, an increase in body fat leading to obesity. Obesity contributes to the impairment of cardiac, respiratory, and immunologic functions and increases the incidence of diabetes, hypertension, and osteoarthritis.[25,26] Additionally, excessive weight gain in the nonambulatory patient may increase the risk of decubitus ulcers.[27] Decubitus ulcer are associated with an increase in local infections, sepsis, and osteoarthritis and are associated with a 50% morbidity in chronically institutionalized patients.[28] Fortunately, this patient maintained good skin integrity despite this significant weight gain as a result of the efforts of nursing and physical therapy staff to rotate frequently the patient's position in the bed and out of bed (eg, wheelchair, bean bag chair, or sling).

One approach to assessing nutritional status in the severely neurodevelopmentally disabled patient is to monitor weight, total body fat, serum protein levels, and to measure REE. Clinical parameters including the patient's ability to ambulate, the patient's appearance (eg, thin or obese) and any influencing disorders (eg, cancer, infection, or hypothermia) must also be addressed.[29] For example, some amount of weight loss may be acceptable and unpreventable during a rigorous period of stress such as during an active pneumonia. Additionally, if the frequency and duration of hypothermic episodes periodically increase or decrease, appropriate adjustments of nutrient intake may be necessary. Although an increase in lean body mass or body cell mass would be desirable, it may not be a realistic goal in this population that demonstrates little spontaneous movement. However, if serum proteins are decreased and the decrease is not due to chronic losses (eg, nephrotic syndrome), serial serum prealbumin levels could be used to monitor visceral protein repletion. On the basis of the patient's nutrition assessment, the nutritional goal for either weight gain, maintenance, or reduction is defined. Because growth charts for children and "ideal body weight" for adults are inaccurate in this population and serum proteins (eg,

Table 3. Percent fat by maturational groupings

Maturational level*	n	Sex†	% body fat
Prepubescent	66	Male (50)	19.0 ± 8.1
		Female (16)	23.2 ± 6.6
Pubescent	59	Male (30)	17.3 ± 7.3
		Female (29)	23.7 ± 6.8
Postpubescent	117	Male (58)	14.0 ± 6.2
		Female (59)	23.6 ± 6.0
Adult	68	Male (36)	16.4 ± 7.4
		Female (59)	26.2 ± 6.4

*Maturational staging was based on the Tanner Scale[33]
†Number of subjects in parentheses

albumin or prealbumin) are usually in the normal range, we use estimates of body fat to guide our therapy.[30] Table 3 depicts the mean values for percentage of body fat in normal subjects.[10] Our target range for percentage body fat was to fall within 1 SD of the mean normal values according to gender and maturational group. Because the combination of tricep (TSF) and calf (CSF) skinfolds measurements correlated best with body composition analysis (ie, with isotopic and bone mineral measurements), out of nine various skin folds sites ($r^2 = .80$), the following equations are used to estimate percentage of body fat and have a mean error of 3.8%:[10]

$$\text{Man: \% body fat} = 0.735\ (TSF + CSF) + 1.0$$
$$\text{Woman: \% body fat} = 0.610\ (TSF + CSF) + 5.1$$

Although these data were developed in normal individuals, recent data from Stallings and coworkers[31] have verified the accuracy of skinfolds techniques in assessing body fat in patients with cerebral palsy. They measured 136 patients, ages 2 to 12 years, with spastic quadriplegic cerebral pals to assess body composition and nutritional status. Results showed that calculations of body fat from two skinfolds correlated best with measures of fat mass from total body water.

Although anthropometric markers are crude and not very useful in hospitalized patients, they may be helpful for assessing nutritional recovery in longterm patients. Because standard nutrition assessment techniques are inappropriate for the neurodevelopmentally disabled population, normalization of fat stores may be a reasonable therapeutic goal. We are not the first to report using estimates of body fat as a means for assessing nutritional recovery in this population.[30] Isaacs et al[32] assessed 22 non ambulating, neurologically impaired children ranging in age from 1 to 12 years to determine appropriate outcome indicators of nutritional status that are measurable over time after gastrostomy placement. At least three nutrition assessments were performed ≥ 10 months after gastrostomy placement. The data indicated that tricep skinfolds fat mass was a more sensitive clinical indicator of change after placement than were weight and height measurements.

Hypothermia decreases measured REE by decreasing oxygen demand. When hypothermia becomes a chronic problem, nutrition adjustments should be made. Despite its presumed rarity, non shivering hypothermia or poikilothermia may be more common in patients with severe developmental disabilities who have suffered from years of modestly controlled seizure

disorders, who have severe mental retardation, spastic quadriplegia, and who are unable to eat a sufficient amount with assistance. We estimate that approximately 15% of the tube-fed patient population with severe neurodevelopmental disorders, a group we have previously described,[30] may suffer from this non shivering hypothermic disorder. An assessment of overall nutritional status that includes weight, body fat, and serum protein levels (eg, prealbumin) should be performed. Indirect calorimetry measurements are helpful to assess the patient's energy needs and should be performed. Nutrition adjustments based on goals derived from the nutrition assessment (serum proteins and body fat) and on measured REE can then be made. The prevention of underfeeding or overfeeding must be observed to avoid long-term complications and to provide the patient with optimal nutrition support. Further work regarding the incidence, etiology, management, and nutrition implications of this disorder in this population is warranted.

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