Disability and Rehabilitation, 2008; 1–7, i*First* article

informa
healthcare

Obesity across the lifespan among persons with spina bifida

NIENKE P. DOSA[1], JOHN T. FOLEY[2], MICHAEL ECKRICH[3], DENISE WOODALL-RUFF[4] & GREGORY S. LIPTAK[1]

[1]*Center for Development, Behavior, and Genetics, SUNY Upstate Medical University, Syracuse, NY, USA,* [2]*SUNY Cortland, Cortland, NY, USA, and* [3]*Pediatric Hematology and Oncology, Vanderbilt University Medical Center, Nashville, TN, USA, and* [4]*Fit Families, SUNY Upstate Medical University, Syracuse, NY, USA*

Accepted 26 March 2008

Abstract

Purpose. Identify risk factors for obesity across the lifespan for individuals with spina bifida.

Methods. Cross sectional chart review study of 203 patients aged 6–58 years. Obesity was based on body mass index. Rates were calculated for children aged 6–11 years; adolescents aged 12–19 years and adults aged > 20 years. Chi-square analyses were used to determine differences in obesity rates among subgroups. An ordered logistic regression model was developed for the three age groups to estimate the probability of a change in BMI classification from normal weight to overweight or overweight to obese, controlling for sex, functional motor level, shunt status and insurance status.

Results. Obesity rates for children, adolescents and adults were 18, 8 and 37%, respectively. Obesity rates were higher among adults ($\chi^2 = 27$, $p < 0.01$) and for individuals who were publicly insured ($\chi^2 = 7.2$, $p < 0.03$). The ordered regression model for children demonstrated no independent association between sex, shunt status, functional motor level or insurance status and change in BMI category. For adolescents, lower functional motor level (i.e. sacral) increased the risk of becoming obese (Odds Ratio: 2.13; 95% CI: 1.12–4.06; $p < 0.02$). Among adults, female sex increased risk (OR = 2.28; 95% CI: 1.03–5.04; $p < 0.04$).

Conclusions. Obesity rates for children and adolescents with spina bifida are similar to the general population; however, obesity rates are higher among adults, particularly women. Risk factors are similar to those observed in the general population.

Keywords: *Obesity, physical activity, spina bifida, disability, lifespan*

Introduction

Obesity is a complex, multi-factorial chronic disease that develops from an interaction of social, behavioural, cultural, physiological, metabolic and genetic factors [1]. The Centres for Disease Control defines obesity as a body mass index (BMI) greater than 30 [2].Obesity rates for the general U.S. population have increased dramatically over the past 40 years. Among children aged 6–11 years, obesity rates increased from 4.2% to 18.8% between 1963 and 2004 [3]. Among adolescents aged 12–19 years, obesity rates increased from 4.6% to 17.4% between 1966 and 2004 [3]. Among adults, obesity rates increased from 15% to 32.9% between 1976 and 2004 [4]. Obesity rates are higher for women than for men. Other risk factors for the development of obesity include poverty, lower education level and sedentary lifestyle [4]. More than one-third of U.S. adults were obese in 2005–2006. This includes 33.3% of men and 35.3% of women. Although there has been a dramatic increase in US obesity rates over the past quarter century, recent data suggest that US obesity rates may have stabilised since 1999, particularly for women [5].

The obesity epidemic is related in part to declining rates of physical activity in the United States [6]. *Healthy People 2010* defines regular physical activity as exercise that promotes cardiorespiratory fitness 3 or more days per week for 20 or more minutes per occasion [7]. Recent data indicate that only 65% of adolescents engage in the recommended amount of

Correspondence: Nienke P. Dosa, Center for Development, Behavior, and Genetics, SUNY Upstate Medical University, 750 East Adams Street, Syracuse, NY 13210, USA. Tel: +315-464-7603. Fax: +315-464-7630. E-mail: dosan@upstate.edu

ISSN 0963-8288 print/ISSN 1464-5165 online
DOI: 10.1080/09638280802356476

physical activity and only 15% of adults perform the recommended amount of physical activity [6]. In 2005, nearly 13% of U.S., adults engaged in no regular physical activity. The proportion of U.S. adults with disabilities who were physically inactive was 25.6%, nearly double that of the general population [8]. *Healthy People 2010* goals include the recommendation that the proportion of Americans who engage in regular physical activity be increased from 65 to 85% for adolescents and from 15–30% for adults [7]. The CDC has identified several subpopulations at risk for lower rates of regular physical activity, including women (of all ages); people with low income; African-Americans and Hispanics; adults in the northeast and southeast; and people with disabilities [7].

Obesity rates among people with spina bifida have been assessed by a variety of methods [9–25] and range from 28 to 50% among children to 34–64% among adolescents and adults [9,16,17,23,24]. The causes of obesity in this population are multifactorial. Non-ambulatory status and sedentary lifestyle [23,24] have been implicated, as have neuroendocrine disturbances related to hydrocephalus and Chiari malformation [9,16]. Metabolic studies have documented that people with spina bifida have less lean body mass and lower basal metabolic rates than peers [23]. Lower levels of everyday activity, possibly related to executive dysfunction [23,24] and metabolic mal-adaptation to stress [13] have also been reported. In addition, short stature is common, particularly among individuals with spina bifida who have higher functional motor levels (e.g. thoracic level) [9,11,14]. Among non-ambulatory children with spina bifida, obesity rates begin to become greater than rates observed in the general population during the preschool years [13]. Early and aggressive dietary intervention [25] and regular physical activity are recommended for the prevention of obesity in children with spina bifida [26,27]. The incidence of obesity increases with age across the lifespan in the general population [4]. This has been observed in cross-sectional studies of persons with spina bifida as well [14]. Adverse health consequences of obesity among persons with spina bifida include preventable secondary medical conditions such as decubitus ulcers, gastro-esophageal reflux disease, metabolic syndrome, social isolation, depression, and reduced mobility and independence [28,29].

There are very few published studies on physical activity and fitness among persons with spina bifida. Preliminary studies suggest that physical activity and fitness rates are 20–30% lower than in the general population, particularly among those who have shunted hydrocephalus [23,24]. Cost of adaptive equipment, transportation difficulties and suboptimal adaptive physical education in the schools have been identified as barriers to regular physical activity and sports participation for people with disabilities [27,30–35]. Additional barriers specific to people with spina bifida include perceived and/or actual medical contraindications to sports participation such as concerns regarding continence, shunted hydrocephalus and decubitus ulcers.

Recent studies have documented that youth with spina bifida, though generally able to perform activities of daily living independently, are not engaging in the full range of adolescent behaviours such as friendship activities, team sports and regular physical activity [36]. Lack of participation in peer-appropriate activities such as sports, has been linked to suboptimal social outcomes in adulthood [27]. The implications of obesity and limited physical activity on long-term health and social outcomes for persons with spina bifida are significant [27,28,37]. Therefore, this study was conducted for the following purposes:

1. Describe obesity rates across the lifespan for persons with spina bifida at a regional referral centre.
2. Provide preliminary data on physical activity in this population, based on chart review.
3. Compare rates of overweight, obesity and physical activity among children, adolescents and adults with spina bifida.
4. Compare obesity and physical activity trends among persons with spina bifida with historical trends observed in the general population.

Methods

We conducted a year long cross-sectional study by chart review. Study protocol was approved by the Research Subjects Review Board at SUNY Upstate Medical University.

Participants were 221 consecutive individual patients aged 6–58 years who were evaluated for routine comprehensive care in 2003 at the Spina Bifida Centre of Central New York. This centre is a state-funded, hospital-based clinic located in an urban setting at SUNY Upstate Medical University in Syracuse, New York. It serves a 24-county catchment area that includes several small cities and an extensive rural population. The centre has provided comprehensive care to patients with spina bifida for more than 30 years. It is staffed by a neurodevelopmental pediatrician, two nurse specialists, a physical therapist, an occupational therapist and a social worker. In addition, the centre provides on-site consultation with two urologists, a physiatrist and an orthopedic surgeon.

Measures

Overweight and obesity were based on body mass index (BMI, weight in kilograms divided by the square of height in meters). Ambulatory status was defined as walking with or without braces or aids for activities in the community, with wheelchair use, if any, limited to long distances [38]. For ambulators, height (crown to heel) was measured by two nurses using a standing stadiometer. For non-ambulators we used arm span length, which was measured by two nurses using a metal rod from middle finger-tip to finger-tip across the area of the Adams apple. Among children and adolescents, overweight was defined as at or above the 95th percentile of the sex- and age-specific BMI charts maintained by the Centres for Disease Control. Among adults, obesity was defined as a BMI over 30 and extreme obesity was defined as a BMI over 40 [39]. Functional motor levels were determined by manual muscle strength testing performed by a physical therapist or physician and categorised according to criteria established by Ryan et al. [38].

We used patient report of regular physical activity other than adaptive physical education during school hours as primary outcome data. Although all patients were queried about physical activity at comprehensive annual visits, we did not use a standardised instrument or a structured interview template to determine physical activity rates.

Demographic data were extracted from billing sheets. Additional information regarding past medical and surgical history were extracted from the medical record.

Analyses

STATA software (version 9.2 for Windows) was used for all analyses. Descriptive statistics were used to determine physical activity and obesity rates for children aged 6–11 years; adolescents aged 12–19 years and adults aged >20 years. We also examined obesity rates for subgroups defined by general demographic factors and by spina bifida-specific characteristics such as shunt status, ambulatory status and functional motor level. Chi-square analyses were used to determine differences in obesity rates among subgroups. An ordered logistic regression model [40] was employed to estimate the probability of observing a change in BMI classification from normal weight to overweight /or overweight to obese for each age group, while controlling for sex, insurance status, shunt status and functional motor level. Physical activity was not included in the regression models because of the limited data on self-reported physical activity.

Results

Analyses were conducted on 203 patients aged 6–58 years for whom complete data were available (Table I). Age range followed a normal distribution pattern. Median age was 19 years. Seventeen percent ($n=34$) were children aged 6–11 years; 36% ($n=75$) were adolescents aged 12–19 years; and 47% ($n=94$) were adults aged 20–58 years. Fifty-five percent ($n=112$) were female. Race was predominantly Caucasian (93%). Two patients reported Hispanic ethnicity and two patients were African-American. Forty-six percent of patients ($n=93$) were publicly insured.

Functional motor levels in our patient population included: sacral 12% ($n=25$); low lumbar 36% ($n=74$); mid lumbar 36% ($n=74$) and thoracic/high lumbar 15% ($n=30$). Fifty-eight percent ($n=118$) were community ambulators, with or without crutches. Sixty-six percent ($n=134$) had shunted hydrocephalus.

The overall prevalence of obesity (BMI for age at or greater than 95th percentile) was 23%. Obesity rates were 18% among children, 8% among adolescents and 37% among adults. Extreme obesity (BMI > 40) occurred in 11% of adult women and 4% of adult men. Chi-square analyses documented

Table I. Spina Bifida characteristics and obesity prevalence based on 203 observations.

	Overall		Female		Male	
	n	(%) Obese	*n*	(%) Obese	*n*	(%) Obese
Age[1]						
All ages	203	23	112	28	91	18
6–11 years	34	18	23	22	11	9
12–19 years	75	8	43	9	32	6
>20 years	94	37*	46	48*	48	27*
Shunt status (ns)						
Hydrocephalus	134	26	68	32	66	20
No hydrocephalus	69	17	44	20	25	12
Ambulatory status(ns)						
Ambulatory	118	19	71	23	47	15
Non-ambulatory	85	28	41	37	44	20
Motor level (ns)						
Sacral	25	36	15	40	10	30
Low lumbar	74	20	45	24	29	14
Mid lumbar	74	24	35	31	39	18
Thoracic	30	17	17	18	13	15
Insurance status[2]						
Public insurance	93	30	49	37	44	23
Private insurance	110	17	63	21	47	13

[1] $\chi^2=27$, $p<0.01$.
[2] $\chi^2=7.2$, $p<0.03$.
*Includes extreme obesity (BMI > 40): Adults overall: $n=7$ (7%); five women (11%); two men (4%).

significantly higher obesity rates among adults ($\chi^2 = 27$, $p < 0.01$) and for individuals of all ages who were publicly insured ($\chi^2 = 7.2$, $p < 0.03$). Although not statistically significant, it is interesting to note that obesity rates were highest for those with sacral functional motor levels. However, obesity rates were lower, as expected, among those who were ambulatory versus non-ambulatory. We conducted additional chi-square analyses to examine the relationship between functional motor level, ambulatory status and BMI classification. These documented a significantly higher proportion of ambulatory individuals among normal weight children, adolescents and adults, and a significantly lower proportion of ambulators among adults whose BMI classification was obese (Table II).

The ordered regression model for children aged 6–11 years demonstrated no independent association between sex, shunt status, functional motor level, or insurance status and the outcome of interest i.e. change in BMI category from normal weight to overweight and/or overweight to obese.

Among adolescents aged 12–19 years, the ordered regression model documented an inverse relationship between obesity and functional motor levels. Those with higher functional motor levels (e.g. thoracic level) were less likely to be categorised as obese during adolescence than those with sacral and low lumbar functional motor levels (odds ratio: 0.47; 95% CI: 0.25–0.89; $p < 0.02$). To confirm this unexpected finding we used ANOVA to compare mean functional motor scores (Sacral 1; Low Lumbar 2; Mid-Lumbar 3; High-Lumbar/Thoracic 4) for each of the three BMI categories (normal weight, overweight and obese). This documented a significant difference between mean functional motor scores and BMI categories ($p = 0.037$). The mean functional motor score for the normal weight subgroup was 2.73 (95% CI: 2.48–2.98) while mean functional motor scores for the overweight and obese subgroups were 2.31 (95% CI: 1.79–2.82) and 1.83 (95% CI: 1.04–2.62), respectively. We next developed an ordered regression model that substituted ambulatory status for functional motor level. This

Table II. Relationship of functional motor level and ambulatory status to BMI classification in persons with spina bifida.

Age group	BMI category	Ambulatory status		Functional motor level: Sacral	Low lumbar	Mid lumbar	Thoracic	Total (n)
6–11 years	Normal wt[1]	Walker	Non–walker	0	2	2	3	7
			Walker	2	9	5	0	16
		Total		2	11	7	3	23
	Overweight ns)	Walker	Non–walker		0	1	1	2
			Walker		3	0	0	3
		Total			3	1	1	5
	Obese (ns)	Walker	Non–walker	0	0	1		1
			Walker	1	3	1		5
		Total		1	3	2		6
12–19 years	Normal wt[2]	Walker	Non–walker	0	3	7	12	22
			Walker	6	12	16	0	34
		Total		6	15	23	12	56
	Overweight (ns)	Walker	Non–walker	0	0	2	0	2
			Walker	2	6	2	1	11
		Total		2	6	4	1	13
	Obese (ns)	Walker	Non–walker	0	1	1		2
			Walker	2	2	0		4
		Total		2	3	1		6
> 20 years	Normal wt[3]	Walker	Non–walker	1	4	11	2	18
			Walker	4	10	5	0	19
		Total		5	14	16	2	37
	Overweight (ns)	Walker	Non–walker	0	2	4	4	10
			Walker	1	8	1	2	12
		Total		1	10	5	6	22
	Obese[4]	Walker	Non–walker	1	5	10	5	21
			Walker	5	4	5	0	14
		Total		6	9	15	5	35

[1] $\chi^2 = 8.52$ ($p = 0.036$).
[2] $\chi^2 = 25.2$ ($p < 0.000$).
[3] $\chi^2 = 8.60$ ($p = 0.035$).
[4] $\chi^2 = 8.38$ ($p = 0.039$).

documented no independent association between ambulatory status and BMI classification among adolescents with spina bifida. Finally, because arm span may not directly correlate with length in persons with spina bifida, particularly for individuals with high lumbar or thoracic functional motor levels (many of whom have short torsos) [38,41], we re-calculated BMI using arm span length adjusted $\times 0.95$ for mid-lumbar, and $\times 0.90$ for high lumbar/thoracic functional motor levels. The ordered regression model based on adjusted BMI values demonstrated no independent association between sex, shunt status or functional motor level and change in BMI category from normal weight to over weight and/or overweight to obese. The only significant variable in the adjusted model was diminished risk of being in a higher weight category for adolescents who were privately insured (OR: 0.23; 95% CI: 0.08–0.70; $p < 0.01$).

Among adults, being female was the only variable significantly to predict being in a higher BMI category (OR = 2.28; 95% CI: 1.03–5.04; $p < 0.04$).

A test of the proportional odds assumption for each age group (likelihood ratio test model) showed no significant difference in the coefficients among BMI categories. This indicates that our model was consistent across the three BMI categories for all three age groups.

Eight adults (11%) and five adolescents (15%) reported regular physical activity. No regular physical activity was reported for children aged 6–11 years. The types of exercise programs reported in this chart review study are listed on Table III.

Limitations

The study is based on a convenience sample of patients with spina bifida who were evaluated at a regional referral centre. Because this was not a population based study, our rates may not apply to all people with spina bifida. In particular, our obesity rates may not be applicable to individuals with spina bifida who are African-American or of Hispanic ethnicity, who were under-represented in our sample. Physical activity rates were based on chart review and were not established using a standardised measure. We also did not collect any systematic data on quality or quantity of adaptive physical education that is received by children and adolescents. We, therefore, did not include physical activity among the independent variables in the ordered regression models and cannot comment on the association between physical activity and obesity in persons with spina bifida.

Weights, heights and armspans for our BMI calculations were obtained from a single measurement, rather than an average of three separate measurements. Use of BMI as an obesity measure is problematic in a population that consists of both ambulators and non-ambulators. BMI calculations using armspan measurements for individuals with higher functional motor levels (e.g., thoracic level) may underestimate obesity rates because many of these individuals have short torsos [41]. Arm span and supine length may not be interchangeable measures of length in persons with spina bifida. We, therefore, ran two sets of regression models using BMI values that were based on adjusted and unadjusted arm span lengths. Different risk factors for obesity were identified in the adjusted versus unadjusted models. This suggests that the effect of insurance status and/or functional motor level on obesity in either model is modest. In both the adjusted and unadjusted models, women were more likely to be in a higher BMI category. Thus, being female can be considered a major risk factor for obesity for adults with spina bifida. In summary, risk factors for obesity in this cross-sectional study of individuals with spina bifida across the lifespan are very similar to known risk factors for obesity in the general population: female sex, adult age and low socioeconomic status.

Table III. Regular physical activity other than school-based adaptive physical education reported by adolescents and adults with spina bifida.

	Activity	Frequency reported
Adolescents	Weight training	2
	Bicycling	1
	Badminton	1
	Wheelchair racing	1
Adults	Weight training	2
	Aquatic therapy	1
	Bowling	1
	Bicycling (stationary)	1
	Stair climbing	1
	Wheelchair racing	1
	Wheelchair basketball	1

Discussion

This study documents obesity rates in children and adolescents with spina bifida that are comparable with the general population, and obesity rates among adults with spina bifida that are slightly higher than those found in the general population. Our findings also document nearly double the rate of extreme obesity among adult women with spina bifida, compared with the general population. Thus, obesity among individuals with spina bifida appears to increase with age, especially affecting adult women.

Obesity trends have been monitored in the general population for 30 years using BMI categories established by the Centres for Disease Control [4]. Within that timeframe, U.S. obesity rates have increased 4.2%–18.8% for children [3]; 4.6%–17.4% for adolescents [3] and 15–34% for adults [4]. Obesity rates in the general population appear to have reached a plateau in recent years [5].

An obesity epidemic parallel to that observed in the general population has not been documented and cannot be inferred for persons with spina bifida. This is because a variety of measures other than BMI have been used to assess obesity in this population, and because, to date, very few studies have been published on obesity in adults with spina bifida. Nevertheless, obesity has historically been considered a common secondary condition in persons with spina bifida. This study documents obesity rates that are lower than those reported in earlier studies. In addition, our study documents that obesity prevalence among persons with spina bifida is only slightly higher than the prevalence of obesity in the general population. This suggests that factors contributing to the U.S. obesity epidemic, such as limited physical activity and changes in diet, have had a limited impact on obesity trends in persons with spina bifida. Stated differently, obesity rates in the general population seem to be 'catching up' to obesity rates in persons with spina bifida.

In this study, the sacral motor level subgroup had a higher prevalence of obesity than subgroups with higher functional motor levels. In addition, the ordered regression model for adolescents documented that a lower functional motor level (e.g. sacral level) increased the risk for the development of obesity in that age group, whereas ambulatory status did not. Since many youths with sacral functional motor levels have a neurogenic bowel and/or bladder, it may be that bowel or bladder incontinence is a more significant barrier to regular physical activity (resulting in obesity) than is non-ambulatory status. Alternatively, adolescents with lower functional motor levels (e.g. sacral levels), many of whom have an 'invisible disability', may participate in more obesity-associated behaviours and/or social groups than those with higher functional motor levels (e.g. thoracic levels) who have a readily apparent physical disability [42]. Individuals with higher functional motor levels also are more likely to have hydrocephalus and other cerebral abnormalities. These may decrease the risk for obesity by altering appetite or satiety, by affecting metabolism, or by making swallowing more difficult. Finally, individuals with sacral levels may have an increased physiologic risk for obesity. Maternal obesity and maternal diabetes have been implicated recently as risk factors for neural tube defects, and are a well-established risk factor for sacral agenesis [43,44]. Three of the 75 adolescents in this study had documentation in the medical chart that they were born to diabetic mothers.

Among adolescents with spina bifida in this study, obesity and overweight were less common than among younger children or adults. A number of explanations are possible. First, this may be related to a 'cohort effect', i.e., teens born between 1984 and 1991 had different environments or interventions causing them to be more resistant to obesity, for example, different adaptive physical education programs in school. Our program had more nutritional support services during the mid 1980s than it did before or does now. It may simply be a random occurrence.

Obesity is a multi-factorial issue in persons with spina bifida. Further studies to examine causal mechanisms across the lifespan will provide insights that may also inform obesity reduction strategies in the general population. Prospective studies examining physical activity in persons with spina bifida, including a careful examination of barriers to regular physical activity, will provide practical strategies for preventing obesity and for improving quality of life in the emerging population of adults with spina bifida.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

1. American Obesity Association Fast Fact Sheet. Internet. http://obesity1.tempdomainname.com/subs/fastfacts/obesity_what2.shtml. Last accessed 26 March 2008.
2. North American Association for the Study of Obesity (NAASO) and the National Heart, Lung, and Blood Institute (NHLBI). The practical guide: identification, evaluation, and treatment of overweight and obesity in adults. Internet. http://www.nhlbi.nih.gov/guidelines/obesity/practgde.htm. Last accessed 26 March 2008.
3. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999–2000. JAMA 2002;288:1728–1732.
4. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. JAMA 2006;295:1549–1555.
5. Centers for Disease Control. Obesity among adults in the United States—no change since 2003–2004. Internet. Data Brief Number 1, November 2007. 8 pp. http://www.cdc.gov/nchs/pressroom/07newsreleases/obesity.htm. Last accessed 28 November 2007.
6. Centers for Disease Control and Prevention. Prevalence of physical activity, including lifestyle activities among adults in the United States, 2000–2001. MMWR 2003;52:764–769.
7. U.S. Department of Health and Human Services. Healthy People 2010, 2nd ed. With understanding and improving health and objectives for improving health, 2 Volumes, Washington, DC: U.S. Government Printing Office, November 2000.
8. Centers for Disease Control and Prevention. Physical activity among adults with a disability–United States, 2005. MMWR 2007;56:1021–1024.

9. Hayes-Allen MC. Obesity and short stature in children with myelomeningocele. Dev Med Child Neurol Suppl 1972;27: 59–64.
10. Hayes-Allen MC, Tring FC. Obesity: another hazard for spina bifida children. Br J Prev Soc Med 1973;27:192–196.
11. Rosenblum MF, Finegold DN, Charney EB. Assessment of stature of children with myelomeningocele, and usefulness of arm-span measurement. Dev Med Child Neurol 1983;25: 338–342.
12. Belt-Neidbala BJ, Ekvall S, Cook CM, Oppenheimer S, Wessel J. Linear growth measurement: a comparison of single arm-lengths and arm span. Dev Med Child Neurol 1986;28: 319–324.
13. Shepherd K, Roberts D, Golding S, Thomas BJ, Shepherd RW. Body composition in myelomeningocele. Am J Clin Nutr 1991;53:1–6.
14. Roberts D, Shepherd RW, Shepherd K. Anthropometry and obesity in myelomeningocele. J Paediatr Child Health 1991; 27:83–90.
15. Liptak GS, Shurtleff DB, Bloss JW, Baltus-Hebert E, Manitta P. Mobility aids for children with high-level myelomeningocele: parapodium versus wheelchair. Dev Med Child Neurol 1992;34:787–796.
16. Mita K, Akataki K, Itoh K, Ono Y, Ishida N, Oki T. Assessment of obesity of children with spina bifida. Dev Med Child Neurol 1993;35:305–311.
17. Jarzem PF, Gledhill RB. Predicting height from arm measurements. J Pediatr Orthop 1993;13:761–765.
18. Rotenstein D, Breen TJ. Growth hormone treatment of children with myelomeningocele. J Pediatr 1996;128:S28–S31.
19. Satin-Smith MS, Katz LL, Thornton P, Gruccio D, Moshang T Jr. Arm span as measurement of response to growth hormone (GH) treatment in a group of children with meningomyelocele and GH deficiency. J Clin Endocrinol Metab 1996;81:1654–1656.
20. Hochhaus F, Butenandt O, Ring-Mrozik E. One-year treatment with recombinant human growth hormone of children with meningomyelocele and growth hormone deficiency: a comparison of supine length and arm span. J Pediatr Endocrinol Metab 1999;12:153–159.
21. Trollmann R, Strehl E, Wenzel D, Dorr HG. Does growth hormone (GH) enhance growth in GH-deficient children with myelomeningocele? J Clin Endocrinol Metab 2000;85:2740–2743.
22. Littlewood RA, Trocki O, Shepherd RW, Shepherd K, Davies PS. Resting energy expenditure and body composition in children with myelomeningocele. Pediatr Rehabil 2003;6:31–37.
23. van den Berg-Emons HJ, Bussmann JB, Meyerink HJ, Roebroeck ME, Stam HJ. Body fat, fitness and level of everyday physical activity in adolescents and young adults with meningomyelocele. J Rehabil Med 2003;35:271–275.
24. Roebroeck ME, Hempenius L, VanBaalen B, Hendriksen JGM, Van Den Berg-Emons HJG, Stam HJ. Cognitive functioning of adolescents and young adults with meningomyelocele and level of everyday physical activity. Disabil Rehabil 2006;28:1237–1242.
25. Grogan CB, Ekvall SM. Body composition of children with myelomeningocele, determined by 40K, urinary creatinine and anthropometric measures. J Am Coll Nutr 1999;18:316–323.
26. Schultz AW, Liptak GS. Obesity, insights factsheet, spina bifida association of America. Internet. http://www.sbaa.org/site/c.liKWL7PLLrF/b.2700287/k.C25F/Obesity.htm. Last accessed 29 November 2007.
27. An J, Goodwin DL. Physical education for children with spina bifida: a Mother's perspective. Adapted Phys Activity Q 2007;24:38–58.
28. Simeonsson RJ, McMillen JS, Huntington GS. Secondary conditions in children with disabilities: spina bifida as a case example. Ment Retard Dev Disabil Res Rev 2002,8:198–205.
29. Nelson MD, Widman LM, Abresch RT, Stanhope K, Havel PJ, Styne DM, McDonald CM. Metabolic syndrome in adolescents with spinal cord dysfunction. J Spinal Cord Med 2007;30 (Suppl 1):S127–S139.
30. Institute of Medicine. The future of disability in America. Washington DC: National Academies Press; 2007.
31. Heath GW, Fentem PH. Physical activity among persons with disabilities—a public health perspective. Exerc Sport Sci Rev 1997;25:195–234.
32. Rimmer JH, Riley B, Wang E, Rauworth A, Jurkowski J. Physical activity participation among persons with disabilities: barriers and facilitators. Am J Prev Med 2004;26:419–425.
33. Rzimmer JH. The conspicuous absence of people with disabilities in public fitness and recreation facilities: lack of interest or lack of access? Am J Health Promot 2005;19:327–329, ii.
34. Rimmer JH, Rowland J, Yamaki K. Obesity and secondary conditions in adolescents with disabilities: addressing the needs of an underserved population. J Adolesc Health 2007; 41:224–229.
35. Murphy NA, PS Carbone, Council on Children with Disabilities. Promoting the participation of children with disabilities in sports, recreation, and physical activities. Pedaitrics 2008;121:1057–1061.
36. Buran CF, Sawin KJ, Brei TJ, Fastenau PS. Adolescents with myelomeningocele: activities, beliefs, expectations, and perceptions. Dev Med Child Neurol 2004;46:244–252.
37. Turk MA. Secondary conditions and disability, in workshop on disability in America: a new look – summary and background papers national academy of sciences. Internet. http://www.nap.edu/catalog/11579.html. Last accessed 29 November 2007.
38. Ryan KD, Ploski C, Emans JB. Myelodysplasia: the musculoskeletal problem: habilitation from infancy to adulthood. Phys Ther 1991;71:935–946.
39. Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. Am J Clin Nutr 2002;7597–7985.
40. Long JS, Freese, J. Regression models for categorical dependent variables using stata, 2nd ed. College Station, TX: Stata Press; 2006.
41. Rotenstein D, Adams M, Reigel D. Adult stature and anthropomorphic measurements of patients with neural tube defects. Eur J Pediatr 1995;154:398–402.
42. Christakis N, Fowler JH. The spread of obesity in a large social network over 32 years. NEJM 2007;357:370–379.
43. Hampton T. Maternal diabetes and obesity may have lifelong impact on health of offspring. JAMA 2004;292:789–790.
44. Botto LD, Moore CA, Khoury MJ, Erickson JD. Neural tube defects. NEJM 1999;341:1509–1519.