Published in final edited form as:

PM R. 2014 October ; 6(10): 908–913. doi:10.1016/j.pmrj.2014.03.012.

Effect of Body Mass Index on Hemiparetic Gait

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Abstract

Objective—To evaluate the relationship between body mass index and spatiotemporal, kinematic, and kinetic gait parameters in chronic hemiparetic stroke survivors.

Design—Secondary analysis of data collected in a randomized controlled trial comparing two 12-week ambulation training treatments.

Setting—Academic medical center

Participants—Chronic hemiparetic stroke survivors (n=108, > 3 months post-stroke)

Methods—Linear regression analyses were performed of body mass index (BMI) and selected pretreatment gait parameters recorded using quantitative gait analysis

Main Outcome Measures—Spatiotemporal, kinematic, and kinetic gait parameters

Results—A series of linear regression models which controlled for age, gender, stroke type (ischemic versus hemorrhagic), interval post-stroke, level of motor impairment (Fugl-Meyer score), and walking speed found BMI to be positively associated with step width (m) ($\beta=.364$, $p<.001$), positively associated with peak hip abduction angle of the nonparetic limb during stance (deg) ($\beta=.177$, $p=.040$), negatively associated with ankle dorsiflexion angle at initial contact of the paretic limb (deg) ($\beta=-.222$, $p=.023$), and negatively associated with peak ankle power at push-off (W/kg) of the paretic limb (W/kg) ($\beta=-.142$, $p=.026$).

Conclusions—When walking at a similar speed, chronic hemiparetic stroke subjects with a higher BMI demonstrated greater step width, greater hip hiking of the paretic lower limb, less

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Disclosures: Financial disclosure statements have been obtained, and no conflicts of interest have been reported by the authors or by any individuals in control of the content of this article.

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paretic limb dorsiflexion at initial contact, and less paretic ankle power at push-off as compared to stroke subjects with a lower BMI and similar level of motor impairment. Further studies are necessary to determine the clinical relevance of these findings with respect to rehabilitation strategies for gait dysfunction in hemiparetic patients with higher BMIs.

Keywords

Body Mass Index; Stroke; Rehabilitation

Introduction

Obesity is a primary risk factor for stroke and a major public health concern associated with both increasing healthcare costs and disability.¹ The prevalence of obesity among stroke survivors in the United States is high. In a recent stroke mortality study², 64.3% of stroke survivors were found to be overweight or obese based on body mass index (BMI). Of those individuals who sustain a stroke each year, 75% will experience walking limitations.³ However, despite the high prevalence of both obesity and walking limitation within the post-stroke patient population, there is limited understanding of the effect of obesity on post-stroke gait patterns. The primary objective of this study was to evaluate the relationship between BMI and spatiotemporal, kinematic, and kinetic gait parameters among persons with chronic hemiparesis using quantitative gait analysis. Our primary hypothesis was that stroke survivors with a higher BMI, but similar level of motor impairment, would ambulate with decreased cadence, increased double support time, decreased stride length, and decreased walking speed based on an established association between obesity and functional disability in the nonstroke population.⁴⁻¹³ However, prior studies have demonstrated that spatiotemporal, kinematic and kinetic parameters of hemiparetic gait may vary depending on level of not only level of motor impairment¹⁴⁻¹⁶ but also walking speed^{14, 17-19}. Thus, our second hypothesis was that a higher BMI may not be associated with specific spatiotemporal, kinematic or kinetic gait differences when our analyses controlled for both level of motor impairment and walking speed. At present, PM&R physicians have no clear scientific basis for understanding how obesity might affect the kinematics and kinetics of gait following a stroke, whether our short or long-term therapy goals should be modified because of obesity, or whether our therapy prescriptions need to be modified because of obesity. If in this chronic hemiparetic patient population a higher BMI is associated with characteristic spatiotemporal deficits and/or kinematic and kinetic gait deficits, independent of the level of motor impairment or walking speed, then BMI should be considered when formulating post-stroke therapeutic interventions intended to enhance post-stroke gait patterns and functional mobility.

Methods

Study Design

This study is a secondary analysis of data collected in a randomized controlled clinical stroke trial (RCT) which compared the lower limb motor relearning effect of 12 weeks of ambulation with a peroneal nerve stimulator versus usual care.²⁰ This present study analyzed BMI and pretreatment quantitative gait analysis (QGA) data collected in the larger

RCT. For purposes of this secondary analysis, the BMI was calculated based on pretreatment weight and height measurements, which were available for 108 subjects.

Participants

The protocols of both the RCT and this secondary analysis were approved by the Institutional Review Board(s) of the involved academic medical center; each subject gave written consent prior to participation. All subjects enrolled in the parent study were a minimum of 18 years of age and medically stable. Subjects demonstrated unilateral hemiparesis with ankle dorsiflexion strength of no greater than 4/5 on the Medical Research Council (MRC) scale. Each subject demonstrated dorsiflexion weakness during ambulation such that inefficient gait patterns with the need for compensatory strategies were exhibited when ambulating a minimum of 30 feet. Each subject was trialed with the PNS device to confirm that ankle dorsiflexion to $> \text{ or } = 0$ degrees could be elicited in response to surface stimulation of the common peroneal nerve while ambulating. Exclusion criteria included lower extremity edema, concomitant neurological diagnoses, uncompensated hemineglect, Mini Mental Status Exam score $< 4^{\text{th}}$ quartile, fixed ankle plantarflexion contracture (passive dorsiflexion $> \text{ or } = 0$ degrees), genu recurvatum (knee extension past neutral in stance when trialed with the PNS device, or history of Botulinum toxin injection to the affected lower extremity in the preceding 3 months. No inclusion or exclusion selection criteria of the RCT were contingent on subject BMI.

Variables

Body Mass Index (BMI) (Independent Variable)—The body mass index (BMI)²¹ is defined as the body weight divided by the square of the height (kg/m^2) and is a widely used screening tool to identify weight problems within a population. A BMI below 18.5 is defined as underweight condition; a BMI of 18.5 to 25 is defined as optimal weight; a BMI > 25 is defined as an overweight condition; a BMI > 30 is defined as obese (Class I, BMI 30-35; Class II, BMI 35-40); a BMI > 40 is defined as morbid obesity (Class III).

Lower Extremity Spatiotemporal, Kinematic, and Kinetic Gait Parameters (Dependent Variables)—All spatiotemporal, kinematic, and kinetic gait parameters were measured using a Vicon system (Vicon Motion Systems Limited, Oxford, United Kingdom), a motion measurement and analysis system which tracked the trajectories of retro-reflective markers in the field of view of multiple cameras mounted around the periphery of the laboratory. Infra-red strobe lights mounted on each camera illuminated the measurement space at 60 Hz during the data collection for the first 48 subjects. After a system upgrade, the camera sampling rate was increased to 100 Hz for the remaining 62 subjects in the parent study. AMTI Biomechanics Platforms (Advanced Mechanical Technology, Inc., Watertown, MA) were embedded in the walkway of the laboratory. Illumination, motion capture data, and analog to digital conversion of transducer input were synchronized and controlled by the Vicon system, which in turn was controlled by a Pentium based PC. Data were processed using the Vicon Plug-In-Gait biomechanical model in Vicon supplied software using the 15 markers of the Lower Extremity Plug-In Gait marker set to generate joint angles, moments, powers, and spatio-temporal parameters of gait. Non-filtered data were normalized to 51 points per stride (increments of 2% of stride) for analysis. All QGA

sessions were performed under the direction of the same gait laboratory engineer using standardized procedures. The specific spatiotemporal, kinematic, and kinetic parameters analyzed were identified a priori for analysis and are listed in Table 1.

Statistical Analysis

Participant characteristics including age, gender, interval post-stroke, stroke type, BMI, pretreatment Fugl-Meyer score (lower extremity), and the selected gait parameters were evaluated for mean, standard deviation and/or frequency. Prior to performing linear regression, we checked that our data met the necessary assumptions in order to apply a valid linear regression model. A series of scatterplot graphs exhibited that each of the parameters was approximately linear. We plotted the residuals versus predicted values. The plots showed the residuals formed horizontal bands around zero and were random and patternless. Therefore, our error terms had an approximate mean of zero for each parameter, and did not violate the assumptions of homoscedasticity and independence of error terms. Also, normal probability plots showed that the residuals for each parameter did not depart much from the straight line angled at 45 degrees representing the normal distribution. A correlation analysis was performed to evaluate the association between BMI and walking speed. Linear regression analyses adjusted for subject characteristics were then performed using BMI as an independent variable and each of the gait parameters as dependent variables. Bonferroni correction for multiple comparisons was not performed due to the exploratory nature of this analysis and the desire to avoid a Type II error.²² A post-hoc power analysis²³ was performed to evaluate whether non-significant results were due to a lack of statistical power; $\alpha=0.05$, two-tailed. The necessary sample size to achieve 80% power was calculated if the post-hoc power for a particular linear regression model was found to be less than 80%.

Results

Participant characteristics and gait parameters are presented in Table 1. Distribution, mean (standard deviation), and range of participants by BMI classification are presented in Table 2. The association between BMI and walking speed was not significant ($r_s = .09$, $p = .34$). Using linear regression models which controlled for age, gender, stroke type, interval post-stroke, level of motor impairment (Fugl-Meyer score), and walking speed, we found BMI to be significantly positively associated with step width(m) ($\beta=0.364$, $p<.001$), significantly positively associated with peak hip abduction angle of the nonparetic limb during stance (deg) ($\beta=.177$, $p=.040$), significantly negatively associated with ankle dorsiflexion angle at initial contact of the paretic limb (deg)($\beta=-.222$, $p=.023$), and significantly negatively associated with peak ankle power at push-off (W/kg) of the paretic limb (W/kg)($\beta=-.142$, $p=.026$) (Table 3). The associations between BMI and all other selected spatiotemporal, kinematic, and kinetic gait parameters were not significant. Post-hoc power analyses demonstrated that statistical power was adequate for the linear regression models of most of the spatiotemporal, kinematic, and kinetic parameters; sample size calculations for the three parameters with less than 80% power are presented (Table 3).

Discussion

A primary finding in this analysis was that an increase in BMI was not associated with a decrease in walking speed, decrease in cadence, increase in double support time, or decrease in stride length in chronic hemiparetic stroke survivors. Stenholm et al²⁴ noted that BMI was a predictor of walking limitation over time and that the coexistence of physical impairment(s) with high BMI further predisposed to later life walking limitation. Thus our hypothesis when designing this secondary analysis was that stroke survivors with an increase in BMI would exhibit a greater degree of walking limitation, for a given level of motor impairment, which would be evident in the spatiotemporal gait parameters. Our findings are at odds with one recent stroke study²⁵ and multiple prior studies of obesity in otherwise healthy adult populations, which suggest that obesity has a negative impact on walking speed^{11, 13}, functional ambulation,^{6-7, 11} balance,^{7-8,26} and/or physical disability.^{4-5, 9-10} The lack of an association between BMI and these specific spatiotemporal gait parameters is likely related to our study design. QGA may not be an optimal method for measuring steady state walking speed given the relatively short distances (10 meters) over which walking speed was recorded per trial. Negative effects of an increasing BMI on walking speed in hemiparetic gait may be more evident with greater distances or ambulation times. Cadence and double support time are both directly related to walking speed, thus the lack of association with BMI in this analysis would be anticipated. It is important to note that the relationship between the functional mobility of any given stroke survivor within a community and spatiotemporal, kinematic, and kinetic parameters measured in a gait laboratory is not clearly defined. A change in spatiotemporal parameters which we may characterize as detrimental (decreased walking speed, stride length, cadence, and/or increased double support time), may in fact be compensatory and ultimately result in enhanced functional mobility.

A secondary finding in this analysis was a positive association between BMI and step-width, the horizontal distance between the midpoint of the midline of each foot in weight-bearing. This finding is consistent with other nonstroke studies^{13,26-28} which have demonstrated, using gait analyses, that step-width is increased in obese subjects as compared to nonobese subjects. In a recent study of the effect of obesity on gait biomechanics, Westlake et al²⁹ simulated, in healthy subjects, the increase in thigh mass and circumference which would be expected with a 10 point increase in BMI. An increase in step-width was noted in two simulated “obese” conditions, an increased circumference condition and an increased mass and circumference condition. The simulated “obese” conditions did not affect kinematic or kinetic parameters measured at the knee. The authors concluded that the increase in step-width noted in the “obese” conditions was the result of physical constraints introduced by a wider thigh segment (circumference). Sarkar recently found an increase in both step-width and “toe-out” foot angle in nonstroke obese subjects²⁶ in a study of the effect of obesity on balance. Thus, an increase in step-width may additionally be a compensation to widen the base of support and stabilize static and/or dynamic balance.

A third finding is that an increase in BMI was associated with a decrease in paretic dorsiflexion angle at initial contact despite controlling for both walking speed and level of motor impairment (Fugl-Meyer score). Dorsiflexion weakness is a common post-stroke

motor impairment which may result in a functional leg length discrepancy most evident during the swing phase of gait. Dorsiflexion angle at initial contact (heelstrike) is a measure of ankle positioning as the paretic limb transitions from non-weightbearing (swing phase) to weightbearing (stance phase). In normal gait, the ankle is in a neutral position (approximately 0 degrees) at initial contact. In stroke survivors with dorsiflexion weakness, the ankle may be more plantarflexed (-5 degrees of dorsiflexion) at initial contact. Our findings suggest that as BMI increases, stroke survivors may be less likely to achieve optimal ankle positioning at initial contact and thus more likely to rely on compensatory maneuvers, such as hip hiking of the paretic limb, to address a functional leg length discrepancy. In this study, the lower extremity portion of the Fugl-Meyer score was used as a proxy for motor impairment. An obesity paradox³⁰ has been described which proposes that an overweight or obese patient is predisposed to specific stroke types which result in less severe motor deficits and thus a lesser degree of motor impairment. Thus, it is unlikely that a general relationship exists between BMI and post-stroke motor impairment to explain a negative relationship between BMI and paretic dorsiflexion angle at initial contact. An alternative explanation is that decreased dorsiflexion at initial contact may simply be due to mechanical obstruction of inter-segmental motion due to adipose tissue at the level of the joint.³¹ Lower extremity edema might also impede ankle joint motion, though this explanation is less likely given that subjects were excluded for lower extremity edema at study entry.

A fourth finding was that an increase in BMI was associated with an increase in nonparetic peak hip abduction angle during stance. A negative association between BMI and peak pelvic obliquity of the paretic side was approached but did not meet statistical significance (Table 3). Kerrigan et al³² defined hip hiking kinematically as an increase in nonparetic hip abduction during stance with a simultaneous elevation of the pelvis on the paretic side during swing. She further defined hip circumduction during gait as an increase in coronal angle of the paretic limb during swing, as compared to normal. Unfortunately, the QGA data available to us for this secondary analysis did not include change in coronal angle. However, our finding of increased paretic hip hiking does suggest that gait compensations associated with decreased dorsiflexion at initial contact may be more pronounced in stroke survivors who are overweight or obese.

Lastly, BMI was negatively associated with peak paretic ankle power at push-off in this analysis. This finding suggests that for any given walking speed, a stroke survivor with a higher BMI may generate lower paretic ankle power as compared to a stroke survivor with a lower BMI. This finding is consistent with prior studies^{12-13, 33} using various methodologies, which showed an association between obesity and decreased ankle plantarflexion, torque and/or power at push-off. The decrease in peak ankle power at push-off may be secondary to altered ankle and forefoot positioning during terminal stance associated with the increased step-width which, while not evaluated, may have been associated with an increase in external hip rotation, and increased external rotation and/or abduction of the ankle. Altered ankle positioning combined with constraints associated with increased segmental adipose tissue may interfere with the ability of the gastrocnemius muscles to generate maximal ankle power. As noted previously, BMI did not correlate with

Fugl-Meyer score, thus the possibility that our higher BMI subjects were more impaired as a group, is not likely.

Interpretation of this secondary analysis is primarily limited by the study design. As previously noted, spatiotemporal variables including walking speed were measured over short distances and may not accurately reflect performance over longer distances or times when fatigue associated with a higher BMI may be more likely to affect gait. Secondly, the BMI is a measurement of both muscle mass and body fat and varies depending on age and gender. In general, older subjects have more body fat for the same BMI than younger subjects and women tend to have greater body fat than men. Thus, the BMI may make overly simplistic assumptions about distribution of muscle and bone mass, particularly in the elderly patient population. Thirdly, the linear regression models predict a magnitude of change in specific gait parameter measurements associated with BMI level that while statistically significant, may not translate into clinically significant differences for any individual stroke survivor. Lastly, though the study was exploratory and a secondary analysis of a pre-existing data set, post-hoc statistical power analysis generally suggests that sample size was adequate and thus unlikely to explain negative findings.

Conclusion

In summary, this analysis provides important descriptive information about the effect of BMI on hemiparetic gait. While being overweight or obese might reasonably be anticipated to negatively affect post-stroke gait, no association was found between BMI and specific spatiotemporal parameters including hemiparetic walking speed, cadence, double support time, or stride length when measured over short distances. These findings suggest that therapy goals for household functional mobility should not necessarily be modified due to a higher BMI. However, this analysis also demonstrates that some of the classic kinematic changes seen in hemiparetic gait which may negatively affect energy expenditure and performance (footdrop and/or hip hiking of the paretic limb) are more pronounced as BMI increases. Lastly, similar to the nonstroke population, an increase in BMI was associated with characteristic spatiotemporal (step-width) and kinetic (peak ankle power at push-off) changes which may affect gait performance over longer distances. In addition to a general recommendation for weight loss, these findings suggests that therapies should focus on optimizing paretic ankle dorsiflexion positioning, ankle power generation at push-off, and compensatory strategies to enhance functional ambulation in hemiparetic patients with higher BMIs.

Acknowledgments

Supported by the National Institute of Child Health and Human Development (grant nos. R01HD44816, K23HD060689, and K24HD054600) and the National Institutes of Health Clinical and Translational Science Collaborative of Cleveland (grant no. UL1RR024989).

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the authors or upon any organization with which the authors are associated.

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Table 1

Participant characteristics (n=108) and spatiotemporal, kinematic, and kinetic gait parameters. Kinematic and kinetic parameters are of the paretic limb unless otherwise noted.

	Mean (SD) or frequency	Range
<i>Characteristic</i>		
Age (yrs)	53.3 (11.0)	27-84
Interval post-CVA (months)	45.4 (89.0)	3 - 677
Gender: Male vs female	66:42	
Type: Ischemic vs hemorrhagic	78:30	
Fugl-Meyer score (lower extremity)	20.2 (5.9)	6-33
BMI	28.5 (5.4)	19 - 48
<i>Spatiotemporal parameters</i>		
Cadence (steps/min)	65.9 (22.3)	(24.7, 154.1)
Double support (secs)	1.15 (.81)	(.32, 3.54)
Step width (m)	.26 (.06)	(.16, .41)
Stride length (m)	.65 (.26)	(.04, 1.16)
Walking speed (m/s)	.37 (.22)	(.05, .97)
<i>Kinematic parameters</i>		
Peak pelvic obliquity angle in swing (deg)	-1.7 (4.3)	(-19.9, 10.5)
Peak hip flexion in swing (deg)	33.9 (8.8)	(14.2, 53.2)
Peak hip abduction in stance, nonparetic (deg)	-3.4 (6.2)	(-18.1, 9.5)
Peak knee extension in stance (deg)	1.3 (9.7)	(-19.5, 23.3)
Peak knee flexion in swing (deg)	27.8 (14.3)	(-2.2, 60.7)
Ankle dorsiflexion angle at initial contact (deg)	-6.6 (8.8)	(-42.7, 14.0)
Peak ankle dorsiflexion in swing (deg)	5.9 (7.0)	(-19.0, 27.3)
<i>Kinetic parameters</i>		
Peak knee extension moment in stance (Nm/kg)	.32 (.27)	(-.03, 1.54)
Peak hip power at pre-swing (W/kg)	.34 (.26)	(.04, 1.32)
Peak ankle power at push-off (W/kg)	.47 (.50)	(.04, 3.18)
Peak ankle power at push-off, absolute (W)	36.2 (40.6)	(0, 178.6)

Table 2
Distribution, mean (SD), and range of participants by BMI classification

BMI	Classification	Number	Mean (SD)	Range
< 18.5	Underweight	0	na	na
18.5 – 25	Optimal	28	22.6 (1.9)	(18.6, 25)
> 25 – 30	Overweight	47	27.6 (1.5)	(25.1, 29.9)
> 30	Moderate obese			
> 30 – 35	Class I	21	32.3 (1.7)	(30.1, 35)
> 35 – 40	Class II	9	36.7 (1.1)	(35.1, 38.4)
> 40	Morbid obese			
	Class III	3	45.3 (4.5)	(40.2, 48)

Table 3

Linear regression analyses of BMI and selected gait parameters while controlling for level of motor impairment and walking speed; observed R^2 , post-hoc power, and sample size to achieve 80% power for nonsignificant findings. Kinematic and kinetic parameters are of the paretic limb unless otherwise noted.

	Standardized coefficient β	p-value	R^2	Post-hoc Power	Sample size to achieve 80% power
<i>Spatiotemporal parameters</i>					
Cadence (steps/min)	.032	.648	.560	>.99	
Double support (secs)	-.033	.618	.608	>.99	
Step width (m)	.364	<.001*	.261		
Stride length (m)	-.014	.727	.852	>.99	
<i>Kinematic parameters</i>					
Peak pelvic obliquity angle in swing (deg)	-.175	.078	.123	.80	
Peak hip flexion angle in swing (deg)	.108	.278	.095	.65	144
Peak hip abduction angle in stance, nonparetic (deg)	.177	.040*	.348		
Peak knee extension angle in stance (deg)	-.121	.229	.084	.58	163
Peak knee flexion angle in swing (deg)	-.060	.478	.343	>.99	
Ankle dorsiflexion angle at initial contact (deg)	-.222	.023*	.153		
Peak ankle dorsiflexion angle in swing (deg)	-.036	.718	.091	.62	150
<i>Kinetic parameters</i>					
Peak knee extension moment in stance (Nm/kg)	-.080	.404	.232	.99	
Peak hip power at pre-swing (W/kg)	.005	.940	.606	>.99	
Peak ankle power at push-off (W/kg)	-.142	.026*	.665		
Peak ankle power at push-off, absolute (W)	.048	.496	.552	>.99	

* Statistical significance, p .05.