Response of Gait Output and Handgrip Strength to Changes in Body Fat Mass in Pre- and Postmenopausal Women

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ARTICLE INFO

ABSTRACT

Background: Available preliminary data on menopause does not relate changes in body fat mass (BFM) and handgrip strength (HGS) (an indicator of body/muscle strength) to gait parameters.

Objective: To determine the relationship between BFM, HGS and gait parameters, namely, stride length (SL) (an indicator of walking balance/postural stability), stride frequency (SF), and velocity (V) (gait output), to guide gait training.

Methods: Ninety consenting (45 postmenopausal and 45 premenopausal) female staff of the University of Nigeria Teaching Hospital, Enugu, were randomly selected and assessed for BFM and HGS with a hydration monitor and dynamometer, respectively, in an observational study. The mean of 2 trials of the number of steps and time taken to cover a 10-m distance at normal speed was used to calculate SF, SL, and V. Data were analyzed using an independent t test and a Pearson correlation coefficient at $P < 0.05$.

Results: Premenopausal (BFM = 42.93% [12.61%], HGS = 27.89 [7.52] kg, stride ratio = 1.43, and velocity = 1.04 [0.01] m/sec) and postmenopausal (BFM = 41.55% [12.71%], HGS = 30.91 [7.07] kg, stride ratio = 1.44, and velocity = 1.06 [0.01] m/sec) women showed no significant differences in gait output/velocity ($t = 0.138$; $P = 0.89$; $d = 0.029$). At postmenopause, BFM was significantly and negatively ($r = -0.369$; $r^2 = 0.1362$; $P = 0.013$) correlated with SL, whereas HGS was positively and significantly ($r = 0.323$; $r^2 = 0.104$; $P = 0.030$) correlated with gait output at premenopause.

Conclusions: BFM may adversely influence walking balance at postmenopause, whereas HGS may enhance gait output at premenopause but not postmenopause. Therefore, muscle strengthening alone may not enhance gait output in postmenopausal women without balance training.

Introduction

Women may experience a decline in physical function during menopause,[1] but there are inconsistent views on whether this decline is due to aging or changes in hormonal status.[1] The menopausal period in women has been associated with a decline in estrogen production,[2,3] which often causes marked physiologic changes in the body, including fatigue,[4] decreased strength,[5] and vascular calcification.[6] This will have some implications for body fat mass (BFM)[7] and hand grip strength (HGS)[4] and, consequently, the parameters of human walking because the skeleton and muscles are parts of the locomotor apparatus.

Early postmenopausal status is associated with a preferential increase in intra-abdominal fat and weight gain,[8] but whether or not these changes are caused by menopause or hormone therapy has attracted discordant views.[9] So far, several scientific studies have identified aging and lifestyle as the primary factors behind

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weight gain in women around the time of menopause.[10–;12] Aging is associated with a slowing of the metabolism,[13,14] wherein lean body mass decreases while body fat accumulates throughout adulthood.[15] Independent of weight variations, menopause has been shown to be associated with major changes in body composition and fat distribution[16] that may affect physical function,[17] including HGS.[4] Therefore, an essential part of physical examinations is the evaluation of hand muscle strength,[18,19] which is defined as the force applied by the hand to pull on or suspend from an object. HGS is a specific, sensitive, reproducible, simple, and noninvasive test of sympathetic function.[20] It is important as an index of general health and a screening test for the integrity of both upper and lower motor neurons, which innervate vital locomotor organs, and should have implications for human walking.[21] HGS has been considered among of the most reliable physical measures of human strength and is seen as the single item most reasonably representative of total body strength.[22] Many factors influence the strength of the grip, including muscle strength, hand dominance, fatigue, time of day, age, nutritional status, restricted motion, and pain.[23] HGS is related to, and predictive of, other health conditions, although the relationship is not thought to be causative. For instance, normal HGS is positively related to normal bone mineral density in postmenopausal women, with some researchers suggesting that it should be used as a screening tool for women at risk of osteoporosis.[20] So far, available preliminary data have not related BFM and HGS to basal gait parameters in women at menopause. This is relevant for understanding the contributions of muscle strength (indicated by HGS) and BFM to changes in physical function characterized by gait output at menopause and likewise informs the relevant interventions.

Methods

Population and study design

Ninety consenting female staff members of the University of Nigeria Teaching Hospital, Enugu, including 45 women at premenopause (mean age = 33.98 (16.99) years; mean height =1.607 m) and 45 women at postmenopause (mean age = 49.22 (24.5) years; mean height =1.611 m), were examined in a cross-sectional observational study for responses of gait output to HGS and BFM at postmenopausal stages. Enugu was selected because many studies have been done on menopause in Enugu,[24,25] most of which have investigated functional decline associated with menopause,[26] and therefore the results of this study would be easily situated in the current literature for easy comparison. The sample size for the study was calculated using the Fisher equation based on an estimated national menopause prevalence of 3.6%, giving a sample size of 58 participants. This is reasonable, because 0 to 3 patients per month seen by gynecologists across Nigeria presented with symptoms of menopause,[27] and a 1% prevalence rate of premature menopause has been previously reported.[28]

At the onset, a mailing was sent to all female workers in the hospital explaining the purpose of the study and soliciting their participation. Using the hospital telephone directory, telephone text messages were also sent to each female staff member by 6 trained research assistants, and 248 female workers who indicated a willingness to participate in the study were enlisted. This population was further categorized into 2 broad groups comprising 128 clinical and 120 nonclinical staff. Thereafter, the target population was separated into 4 categories based on information provided by them on a self-reported questionnaire. Menopausal status was classified into 3 groups, namely premenopausal stage: regular menstrual cycles; perimenopausal stage: ≥3 months without menses, large changes in cycle length, and hot flashes; or postmenopausal stage: ≥12 months without menses or had undergone a hysterectomy. Based on this classification, 93 pre- and 72 post-

menopausal women who had not undergone hysterectomy were identified and asked to provide further information on whether or not they had used estrogen. Based on the available data, they were further categorized into 4 groups, namely never used estrogen, used estrogen for ≤5 years, used estrogen for ≥5 years, and not sure of ever using estrogen. A list of women who never used estrogen was drawn up comprising 86 (38 clinical and 48 nonclinical) premenopausal and 64 (31 clinical and 33 nonclinical) postmenopausal women, from which the required number was selected by a simple random sampling method while providing for almost equal representation for clinical and nonclinical staff. The study process involved 3 stages: obtaining informed consent, physical assessment, and gait analysis. The test instrument was a fat analyzer/hydration monitor (Jamar Hand Dynamometer Model 78010; Misfit, Helsinki, Finland), a stadiometer, measuring tape, and a stopwatch. These instruments were used to measure the BFM, HGS, height, and walk time, respectively.

Ethical approval

Participants gave their written informed consent before participation and after the purpose of the study was explained to them. This study followed the principles of the Declaration of Helsinki and informed participants of their right to withdraw from the study at any time of their choice, which was strictly respected. The study protocol was also approved by the University of Nigeria Health Research Ethics Committee on certificate number NHREC/05/01/2008B. Participants' confidentiality was maintained by using code numbers instead of names and ensuring that records were destroyed at the end of the study.

Data collection

The investigators had the approval of the hospital administration, including heads of clinical and nonclinical departments of the hospital, to approach and recruit consenting female staff members for the study. The participants who met the eligibility criteria for the study were subjected to various physical assessments and gait analysis. Five inclusion criteria were applied as follows: at least 1 year of a regular menstrual cycle before the study for women at premenopause; nonmenstruation for at least 1 year for women at postmenopause; no history of other diseases of a metabolic, neurologic, or orthopedic nature; age not <25 years but not >64 years; and natural menopause (ie, no hysterectomy or bilateral oophorectomy before final menstrual period).

The procedures for the study were explained to the participants, and only those who gave their written informed consent were involved in the study. Participants' anthropometric data were measured, including weight and height. The data generated were imputed into the bi-impedance electronic fat analyzer for each individual, who was requested to stand barefoot on the equipment until readings of their body compositions were displayed on the liquid-crystal display screen. HGS was subsequently measured using a dynamometer as prescribed by the American Society for Surgery of the Hand and the American Society of Hand Therapists, which is described in detail elsewhere.[29] Subsequently, participants were required to demonstrate their normal gait at a self-selected speed over a distance of 10 m marked out on the floor as previously described.[30,31] The number of steps and time required to complete the distance were noted, and the mean of 2 trials was determined and used to calculate the basal gait parameters (ie, stride length, stride frequency, and velocity).

Data/statistical analysis

With the number of steps and time taken to complete the 10-m distance, it was possible to calculate the basal gait parameters—

Table I
Classification of participants based on menopausal status (N=90).

Menstrual status	n	N%	MA*	MH
Premenopausal women	45	50	33.98 (16.99)	1.607
Postmenopausal women	45	50	49.22 (24.5)	1.611
Total	90	100		

MA = mean age; MH = mean height; n = frequency; N% = number of participants in percentage.
* Values for mean height are presented as mean (SD).

stride length, stride frequency, and gait output (ie, velocity), using the following mathematical relationships:

1. Strides = $\frac{No.\ of\ steps}{2}$
2. Stride frequency = $\frac{No.\ of\ strides}{time}$
3. Velocity = $\frac{distance}{time}$, OR
4. Velocity = stride length × stride frequency
   Additionally, the stride ratio was determined as the quotient of stride frequency / stride length.
5. Stride ratio = stride length/stride frequency
   The phases of stride studied were the stance duration, swing duration, and double-support duration. The phases of stride for participants were calculated using the mean ordinary stride duration for normal walking as the input in the regression equation (Phase duration = A + B × S), where S is the stride duration or the inverse of stride frequency. Thus, the following equations were used:
6. Stance duration = −0.17 + 0.72 sec
7. Swing duration = 0.18 + 0.27 sec
8. Double-support duration = −0.18 + 0.24 sec

Data were analyzed using the independent t test to compare the mean values of the parameters between the groups, whereas the Pearson correlation coefficient was used to determine the relationship between the parameters, at $P < 0.05$.

Results

Altogether, 90 women comprising 45 women at premenopause (mean age = 33.98 (16.99) years, mean height, 1.607 m) and 45 women at postmenopause (mean age = 49.22 (24.5) years, mean height = 1.611 m), were studied (**Table I**). There was no significant decrease ($t = 1.964$; $P = 0.053$; (95% CI, −0.03570–6.08015) in HGS (**Table II**) of postmenopausal women (27.89 [7.52] kg) compared with premenopausal women (30.91 [7.07] kg). The effect size was moderate ($d = 0.414$). Similarly, there was no significant increase in the BFM of postmenopausal compared with premenopausal women ($t = −0.519$; $P = 0.053$; 95% CI, −6.69133–3.91800; $d = −0.109$). In addition, there was no significant decrease in the stride length ($t = 0.367$; $P = 0.714$; 95% CI, −0.03899–0.05666) of postmenopausal (1.22 [0.12] m) compared with premenopausal women (1.23 [0.11] m), and the effect size was small ($d = 0.087$). Stride frequency was not significantly decreased ($t = 0.229$; $P = 0.819$; 95% CI, −0.03003–0.03785; $d = 0.133$) when postmenopausal women (0.85 [0.07] strides/sec) were compared with

premenopausal women (0.86 [0.08] strides/sec). Similarly, stride velocity was not significantly decreased ($t = −0.138$; $P = 0.890$; 95% CI, −6.29466–7.23598; $d = 0.029$), and the effect size was small ($d = 0.029$), when postmenopausal women were compared with premenopausal women.

There was no significant correlation between BFM and HGS (**Table III**) in premenopausal women ($r = 0.175$; $P = 0.250$, $r^2 = 0.031$) or postmenopausal women ($r = 0.142$; $P = 0.351$; $r^2 = 0.020$). Similarly, BFM was not significantly correlated with stride length ($r = −0.257$; $P = 0.089$; $r^2 = 0.066$), stride frequency ($r = −0.210$; $P = 0.167$; $r^2 = 0.044$), or stride velocity ($r = −0.18$; $P = 0.222$; $r^2 = 0.032$) in premenopausal women. Although BFM was not significantly correlated with stride frequency ($r = −0.108$; $P = 0.012$; $r^2 = 0.480$) or velocity ($r = −0.146$; $P = 0.021$; $r^2 = 0.338$) in postmenopausal women, it was, in contrast, significantly negatively correlated with stride length ($r = −0.369$; $P = 0.013$; $r^2 = 0.136$). Furthermore, HGS was not significantly correlated with stride length ($r = 0.175$; $P = 0.031$; $r^2 = 0.249$) or stride frequency ($r = 0.147$; $P = 0.336$; $r^2 = 0.022$) in postmenopausal women, unlike stride velocity ($r = 0.323$; $P = 0.030$; $r^2 = 0.104$), which showed a significant positive relationship.

Discussion

There was no significant difference in the total BFM between pre- and postmenopausal women, although the postmenopausal women had a relatively higher BFM than the former. However, this does not imply that the regional distribution of fat may not have varied significantly. For instance, an earlier study[32] revealed that subcutaneous abdominal adipose tissue areas were significantly greater in premenopausal women, whereas visceral abdominal adipose tissue areas and the subcutaneous to visceral abdominal adipose tissue area ratios were significantly greater in postmenopausal participants. After adjusting for body mass index, no significant differences emerged between the 2 groups in regard to total abdominal adipose tissue areas, waist circumference, hip circumference and waist-hip circumference ratio. The same trends may be applicable to this study, considering that women at postmenopause had greater BFM than those at premenopause.

Relevance of findings to the field

There could be a recompartmentalization of body composition at postmenopause as indicated by the findings of this study. This is consistent with the fact that menopause is a biological landmark that defines the transition from the reproductive to the nonreproductive phase of life in women.[33] The physiologic response to the decline in estradiol and related hormonal changes leads to climacteric symptoms[34] that are characteristic of this phase of life. Increased adiposity and consequent loss of muscle mass are significant physiologic developments associated with menopause,[35] which lead to significant loss of muscular strength and a decline in the functional capacity of women.[36] It was evident in this study that although the total BFM was marginally increased, HGS was also marginally reduced in postmenopausal women, unlike

Table II
Mean (SD) of body fat mass (BFM), hand grip strength (HGS), and basal gait parameters (stride length [SL], sctride frequency [SF], stance duration [SD], swing duration [SW], double-support duration [DS], and stride ratio [SR] in pre- and postmenopausal women (N=90).

Group	HGS	BFM	SL	SF	Velocity	ST	SW	DS	SR*
Pre	30.91 (7.07)	41.55 (12.71)	1.23 (0.11)	0.86 (0.08)	1.05 (0.17)	0.67 (8.83)	0.49 (3.56)	0.099 (2.82)	1.43
Post	27.89 (7.52)	42.93 (12.61)	1.22 (0.12)	0.85 (0.07)	1.04 (0.15)	0.68 (10.12)	0.50 (4.04)	0.102 (3.25)	1.44
t value	1.964	−0.519	0.367	0.229	0.465	2.713	2.373	∞	
P value	0.053	0.605	0.714	0.819	0.643	0.996	0.9901	1.000	

* Calculated as stride length / stride frequency.

Table III

Relationship between handgrip strength (HGS), body fat mass (BFM), and basal gait parameters (stride length [SL], stride frequency [SF], and stride velocity [SV]) among premenopausal and postmenopausal women (N = 90).

	BFM		HGS		SL		SF		SV	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
BFM (r)	1	1	0.175	0.142	−0.257	−0.369	−0.210	−0.108	−0.18	−0.146
r^2	1	1	0.031	0.020	0.066	0.136	0.044	0.012	0.032	0.021
P value			0.250	0.351	0.089	0.013*	0.167	0.480	0.222	0.338
HGS (r)	0.175	0.142	1	1	0.175	0.090	0.147	−0.139	0.323*	−0.110
r^2	0.031	0.020	1	1	0.031	0.008	0.022	0.019	0.104	0.012
P value	0.250	0.351			0.249	0.555	0.336	0.362	0.030*	0.471
SL (r)	−0.257	−0.369	0.175	0.090	1	1	0.498**	0.133	0.704**	0.534**
r^2	0.066	0.136	0.031	0.008	1	1	0.248	0.017	0.496	0.285
P value	0.089	0.013	0.249	0.555			0.385		0.0001***	0.0001***
SF (r)	−0.210	−0.108	0.147	−0.139	0.498**	0.133	1	1	0.747**	0.727**
r^2	0.044	0.012	0.022	0.019	0.248	0.018	1	1	0.558	0.529
P value	0.167	0.480	0.336	0.362	0.0001***	0.385			0.0001***	0.0001***
SV (r)	−0.186	−0.015	0.323*	−0.110	0.704**	0.534**	0.747**	0.727**	1	1
r^2	0.035	0.000	0.104	0.012	0.496	0.285	0.558	0.529	1	1
P value	0.222	0.338	0.030	0.47	0.0001***	0.0001***	0.0001***	0.0001***		

* Significant at P < 0.05.

** Significant at P < 0.001.

*** Significant at P < 0.0001.

premenopausal women. This suggests a possibility that changes in BFM may adversely influence total muscle strength in postmenopausal women. Because menopause induces muscle loss and consequently a loss of muscle strength, it is reasonable that the associated gain in BFM may play a role in distorting the muscular configuration that optimizes physical functioning, especially walking function. Invariably, physical function, which relies on muscle strength among other factors, may be more compromised in postmenopausal women than premenopausal women. This might explain why BFM was significantly and negatively correlated with stride length in postmenopausal women.

Stride length is a reliable measure of balance and an indicator of postural control in human walking.[30,31,37] Thus, a decline in stride length in postmenopausal women compared with premenopausal women suggested an increased tendency for imbalance and postural instability that may predispose postmenopausal women to a fall, which has been previously reported.[38] In essence, increased BFM seems to adversely influence stride length, which may negatively influence walking balance and postural control in postmenopausal women. The relationship between HGS and velocity (gait output) in premenopausal women supports the above conclusion. This is reasonable because it was revealed that as HGS (an index of muscle strength) increased, there was a possibility of a significant and proportionate increase in velocity (gait output) in premenopausal women, unlike in postmenopausal women. This implies that increased HGS (an index of muscle strength) may translate to improved gait function in premenopausal women, but not postmenopausal women. The other components of the neuromuscular system that support a safe upright stance in walking appeared to be optimized in premenopause. Therefore, there may be other intervening physiologic variables that influence walking speed in postmenopausal women apart from muscle strength. In essence, improving the muscle strength of postmenopausal women alone may not necessarily translate to improved gait speed or walking function. It is possible that other factors, such as balance coordination, information processing for motor performance, and reaction time, which all deteriorate with aging, may play mediatory roles.[39] In fact, the influence of aging on physical functioning could be a contributory factor that might explain variations in HGS (muscle strength) and its relationship with gait output in this study. Cooper et al[40] studied a birth cohort of women born in the same week who completed physical performance tests at the same age. Their findings revealed that natural menopause correlated with comparable physical functioning levels in premenopausal and perimenopausal women, giving credence to the view that variation in age may partly explain the observed relationship between HGS and gait velocity in postmenopausal women compared with premenopausal women in this study. However, this does not discount the significant contributions of menopause in recompartmentalizing the body, particularly in the redistribution of body fat between the visceral and subcutaneous adipose tissues, as already highlighted.

Implications for care teams and policymakers

Janssen et al[41] revealed that there are menopause-related increases in visceral adipose tissues independent of age, and this has been supported by evidence from cross-sectional[42,43] and longitudinal studies.[44,45] The physiologic mechanisms responsible for this are not yet fully understood, but in vitro administration of follicle stimulating hormone to preadipocytes in murine studies caused a redistribution of visceral fat mass and an increase in adipocyte lipid droplets and adipocyte lipid synthesis.[46] In fact, there was an alteration of the serum concentration of adipokines that included leptin and adiponectin as well as subgroups of lipids, especially triglycerides. This therefore suggests that follicle stimulating hormone might trigger fat redistribution and add to a proinflammatory environment.[46] The same is applicable for cortisol, which enhances visceral fat expansion[47] but does not vary significantly in premenopausal compared with postmenopausal women. However, serum concentrations of estrogen may alter the expression of cortisol receptors in visceral fat and local levels of glucocorticoids,[47] and may have implications for physical functioning. Therefore, a decline in estrogen levels may alter the expression of cortisol receptors in visceral fat in postmenopausal women and may partly explain the increase in BFM recorded in this study for this population.

Available studies revealed that women with surgical menopause recorded slower chair-rise times compared with women with natural menopause. Additionally, women who experienced natural menopause at older ages recorded faster walking speeds and less self-reported functional limitation in later life than women who experienced natural menopause at younger ages. This points to the relevance of estrogen in modulating gait function through its influence on fat distribution, muscle bulk, and muscle strength, which are important variables that may determine the efficiency and ef-

fectiveness of the human locomotor apparatus. Early age at surgical menopause was also linked to increased levels of self-reported functional limitations. It was observed that differences in walking speed and chair-rise times persisted after adjusting for potential confounding variables or covariates,[40] and may be predictive of future health outcomes.[48] This will have implications for walking velocity because velocity is the quotient of distance and walk-time (ie, distance / time). Velocity is also the product of stride length and stride frequency and, in essence, reflects the proportional combination of stride length and stride frequency under a physiologic drive that may be influenced by menopausal changes in women. It was evident from the results of this study that there was a marginal decrease in walking velocity in postmenopausal compared with premenopausal women; the stride ratio (ie, stride frequency and stride length) was also marginally altered. Therefore, the neural drive for walking function in pre- and postmenopausal women may not be similar.

Although the stride length was marginally greater in premenopausal women than postmenopausal women, the stride ratio was greater in the latter compared with the former. Because stride ratio is a quotient of stride length and stride frequency (ie, stride length / stride frequency),[49] this implied that relative to stride frequency the stride length was increased in postmenopausal women compared with premenopausal women, if velocity was constant and will have implications for the interaction of stride length and stride frequency to produce velocity (ie, velocity = stride frequency × stride length). Because stride length is a reliable measure of balance control,[37] its increase relative to stride frequency in postmenopausal women could represent an attempt to improve balance control, probably due to an imbalance in the system. This could be an indication of a greater tendency toward instability in the system, which should elicit a compensatory drive to optimize stability and prevent a fall by increasing the double-support duration—an indicator of postural stability.[37,50] This was in fact observed, because double-support duration was increased in postmenopausal women compared with premenopausal women. The double-support duration is not only an indicator of postural stability but also of speed transition, especially a switch from walking to running. Invariably, increased double-support duration represented an attempt to minimize speed change and delay speed transition from walking to running in postmenopausal women compared with premenopausal women. Therefore, postmenopausal women should be slower walkers compared with premenopausal women, and this explains why the stride velocity or gait output was slower in postmenopausal women than premenopausal women in this study. Ordinarily, at a constant velocity, a decrease in stride length observed in postmenopausal women would trigger a compensatory response to increase stride frequency. However, this was not realized in postmenopausal women, because their stride length, stride frequency, and velocity did not equal the values recorded in premenopausal women. Therefore, the equation for the relation between velocity, stride length, and stride frequency, (ie, velocity = stride length × stride frequency) experienced a physiologic limitation in postmenopausal women.

The significant negative relationship between BFM and stride length in postmenopausal women suggested that increased BFM might adversely influence both walking balance and gait output in postmenopausal women. In essence, increased BFM, which is a common feature of menopause,[41–43] might distort the physiologic relationship between the basal gait parameters, especially stride length and stride frequency, with implications for gait velocity, balance, and postural stability. However, the difference in the mean of these parameters between pre- and postmenopausal women was not significant. Nevertheless, the results highlighted important trends that may have prognostic utility in clinical practice.

Strengths and weaknesses of the study

The findings of this study may be different from other studies, and may be explained by differences in the method of gait assessment, because other studies have used the slower walk-time of 2 attempts to calculate gait speed (in meters per second) in the usual steps, over a distance of 4 m.[51] However, it is not plausible that an individual's gait will be fully expressed over such a short distance, which informed the use of a 10-m distance for gait assessment in this study. In addition, this study used the average or mean value of stride and time required to cover the 10-m distance in 2 trials to determine the velocity and speed of walking, unlike several studies already described,[51] which used the slower time of 2 attempts to determine the speed of walking. The mean value should be more representative of the gait characteristics of the participants.

The lack of power of the statistical tests in view of the small number of the participants (n = 45 per group) could be a limitation of the study. In addition, the cross-sectional observational design of this study limited the exploration of gait parameters for changes that might occur over time with menopausal (premenopausal to postmenopausal) phases in relation to progressive variations in BFM and HGS. This would have fully elucidated the influence of menopause on gait characteristics in women and the likely functional implications that might arise, but was not done. Additionally, the whole spectrum of walking speeds[30,31] was not explored, because only the normal walking speed was measured to situate the findings in the contemporary literature, given that a previous study[51] also explored normal walking speed in this population. However, different activities of daily living require expression of various gait speeds other than normal walking speed. Moreover, the influence of certain physiologic limitations in human walking is amplified at high and slow speeds, but not at normal walking speed, and vice versa. Therefore, the findings of this study did not reflect events across the full spectrum of human walking speeds to elucidate how these events were influenced by different stages of menopause. Furthermore, the bioelectric impedance analysis that was used in this study is able to quantify BFM,[51] similar to a variety of measurement tools such as dual-energy x-ray absiorptiometry or computed tomography; however, no standard definitions are available to relate specific cutoff points to various levels of risks of mortality and physical functioning, particularly ambulatory function. This would have easily situated the findings of this study in the contemporary literature for easy comparison. Despite these limitations, this study was able to demonstrate that physiologic changes associated with menopause could have implications for gait, particularly walking balance and postural stability. It demonstrated the possibility that some physiologic limitations are imposed on the relationship between basal gait parameters such that they could no longer compensate for one another at postmenopause. Therefore, to a reasonable extent, the objective of the study was realized and may have clinical relevance in practice.

Conclusions

Unequal stride ratios were observed in pre- and postmenopausal women, with the latter being greater than the former. This suggests that at constant velocity (gait output), stride frequency could not compensate for stride length in postmenopausal women compared with control or premenopausal women. Thus, velocity was produced by an increase in stride length (index of stability) relative to stride frequency (index of speed change) in postmenopausal women compared with premenopausal women. This could be an indication that nonidentical neuromuscular strategies are employed to emphasize safety and balance control more than speed changes in postmenopausal women, and vice versa in premenopausal women. The study revealed that increases in

BFM may adversely influence stride length (a measure of stability) in postmenopausal women, whereas an increase in HGS (an index of body/muscle strength) may positively enhance gait output, and, invariably, physical function in premenopausal women. Because HGS has no positive relationship with velocity in postmenopausal women, unlike in premenopausal women, it could be that muscle strengthening programs alone may not be sufficient to enhance gait output in postmenopausal women without additional interventions to reduce BFM, improve coordination and balance training. The significant negative relationship between BFM and stride length (an indicator of balance and postural control) supports this conclusion. Therefore, postmenopausal changes in women may compromise walking balance, which may increase the tendency to fall. This highlights the need for a preventive intervention using exercise programs that not only improve muscle strength and balance coordination but also minimize BFM redistribution in postmenopausal women.

Acknowledgment

Conflicts of Interest

The authors have indicated that they have no conflicts of interest regarding the content of this article.

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