European Spine Journal (2019) 28:967–975
https://doi.org/10.1007/s00586-019-05935-0

ORIGINAL ARTICLE

Back pain and sagittal spine alignment in obese patients eligible for bariatric surgery

Alexandre Peixoto de Mello[1,2] · Glaucus Cajaty dos Santos Martins[3,4] · André Raposo Heringer[4] · Raphael Barbosa Gamallo[4] · Luiz Felipe dos Santos Martins Filho[4] · Antônio Vítor de Abreu[3] · Antonio Carlos Pires Carvalho[3] · Maurício de Pinho Gama[3,5]

Received: 27 July 2018 / Revised: 14 February 2019 / Accepted: 26 February 2019 / Published online: 15 March 2019

Abstract

Objective The objective of this research was to evaluate the prevalence of cervical and lumbar pain in obese patients eligible for bariatric surgery and to investigate possible changes in sagittal spine alignment in these patients.

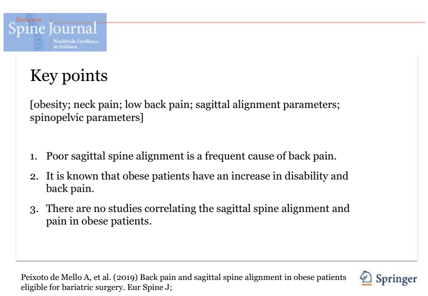
Methods The following parameters were compared in 30 obese patients and a control group of 25 non-obese volunteers: body mass index, prevalence of cervical and lumbar pain assessed by visual analog scale (VAS), Neck Disability Index [NDI] and Oswestry Disability Index [ODI], as well as radiographic parameters of the spine and pelvis measured with Surgimap software.

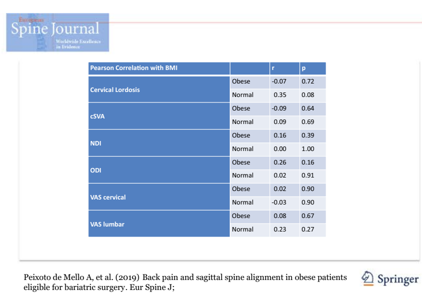
Results The cervical and lumbar VAS and the NDI and ODI were significantly worse in obese patients. Compared with the control group, the cervical sagittal vertical axis (cSVA) of the obese group had higher variance (p value = 0.0025) and the cervical lordosis was diminished (p value = 0.0023). Thoracic kyphosis, lumbar lordosis, and the pelvic parameters were not significantly different between the groups.

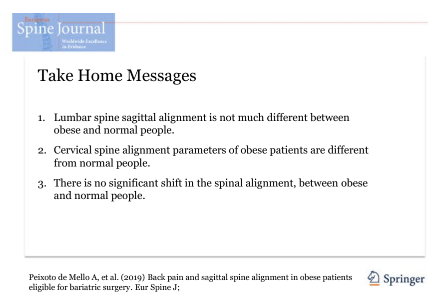
Conclusions Obese patients demonstrated lower functional performance compared with their non-obese counterparts, while cervical lordosis was diminished and the cSVA was increased in obese patients.

Graphical abstract

These slides can be retrieved under Electronic Supplementary Material.







Keywords Obesity · Neck pain · Low back pain · Sagittal alignment parameters · Spinopelvic parameters

Introduction

Obesity is one of the most important public health problems, currently responsible for approximately 7.1% of all global deaths [1]. In 2016, according to the World Health Organization (WHO) criteria, 13% of the adult population was obese, including 11% of men and 15%

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s00586-019-05935-0) contains supplementary material, which is available to authorized users.

✉ Alexandre Peixoto de Mello
apeixotodemello@hotmail.com

Extended author information available on the last page of the article

of women. The worldwide prevalence of obesity nearly tripled between 1975 and 2016 [2]. The global economic impact of obesity is about $2.0 trillion, or 2.8% of the global gross domestic product (GDP), roughly equivalent to the global impact of smoking or armed violence, war, and terrorism [3].

The incidence of musculoskeletal disorders [4, 5], including back pain [6, 7], is high in obese patients. However, still no definition in the literature exists about the cause and effect association between obesity and back pain. Classically, obesity is associated with degenerative joint disease due to the mechanical stress placed on the cartilage by increased local pressure, stress, tension, shear forces, and/or increased hydrostatic pressure within the cartilage [8], and it is natural to believe that the same would happen in the vertebral structures. A recent research showed an increased risk of vertebral fractures in obese individuals, directly related to BMI e body shape [9]. However, more recent studies involving visceral fat proteins point to adipokines as playing an important role in the development of joint injury [10]. Adipokines are a group of proteins produced by white adipose tissue that are believed to induce a state of systemic low-grade inflammation [11, 12]. Some recent researches connected them to intervertebral disk degeneration [13, 14]. A recent review of the literature that evaluated overweight and obesity alone revealed that there were substantial costs due to lost productivity among affected workers [15, 16].

Back pain is an important public health problem. It is the second leading cause of absenteeism at work [17, 18]. Additionally, it is responsible for a significant proportion of long-term absenteeism, with an estimated 32% of patients not returning to work within 1 month [19]. It is estimated that 54% to 80% of the adult population will have at least one episode of back pain during their lifetime [20]. There is an apparent relationship between obesity and back pain, but the details remain poorly understood [21–23].

Additionally, the change in physiological sagittal curves in the spine (cervical and lumbar lordosis and thoracic kyphosis) [24, 25] and decreased mobility of the spine [26] are related both to obesity and to back pain. Currently, a value is assigned to the sagittal spinal balance in the genesis of pain [24]. While there is no normal sagittal balance, it is important to maintain an appropriate balance between the curvatures of the spine and the pelvis [27].

The objective of this study was to evaluate the prevalence of cervical and lumbar pain in obese patients eligible for bariatric surgery, and to investigate possible changes of parameters of sagittal alignment in these patients and the role of these changes in the origination of the pain.

Materials and methods

A cross-sectional study registered and approved by the Committee on Ethics in Research (66439416.1.0000.5646) was performed with informed consent of participants.

A group of 30 patients in the Bariatric Surgery Outpatient Clinic of the Hospital of Ipanema was randomly selected for the study. The group was selected as the patients elected for bariatric surgery in the outpatient clinic between January and June of 2017. Another group of 25 volunteers from the health team was selected to serve as a control group, while those who were obese or who had back pain were excluded (Table 1). The control group was structured to be similar in gender composition and mean age to the study group. Individuals who had previously undergone surgical procedures for spine issues or fractures of the lower limbs were excluded.

Obesity was determined by the body mass index (BMI), as recommended by the WHO [28].

Participants were questioned about their cervical and lumbar pain and were presented with a ruler depicting a visual analog scale of pain. The answer was classified as

Table 1 Groups composition

Group	Obese	Control
Sex		
Male		
Total	5	5
Percentage	17%	20%
Female		
Total	25	20
Percentage	83%	80%
D'Agostino–Pearson		
p value	< 0.0001	< 0.0001
Mann–Whitney		
p value	0.833	
Age		
Mean	51	50
Standard deviation	7	13
D'Agostino–Pearson		
p value	0.451	0.281
Student's *t*		
p value	0.784	
BMI		
Mean	39.5	23.5
Standard deviation	4.6	3.3
D'Agostino–Pearson		
p value	0.009	0.004
Student's *t*		
p value	< 0.0001	

0 (no pain) to 10 (no more intense pain possible); with mild pain being 0–3, moderate pain being 4–6, and severe pain being 7–10.

Functional involvement and limitations associated with back pain have been assessed by the Neck Disability Index (NDI) [29] and the Oswestry Disability Index (ODI) [30]. These scales evaluate the impact of pain on the life of the patient by assessing main activities of daily living. Each question is given a value, resulting in a score for each activity (domain) and an overall score (sum of items).

X-rays of the spine were taken in the standing position.

Using the software Surgimap Spine (Nemaris Inc., New York-NY, USA) [31], the following parameters were measured on digital radiographs of the groups (Fig. 1):

- cervical lordosis was measured from the lower plateau of C2 to the C7 lower plateau;

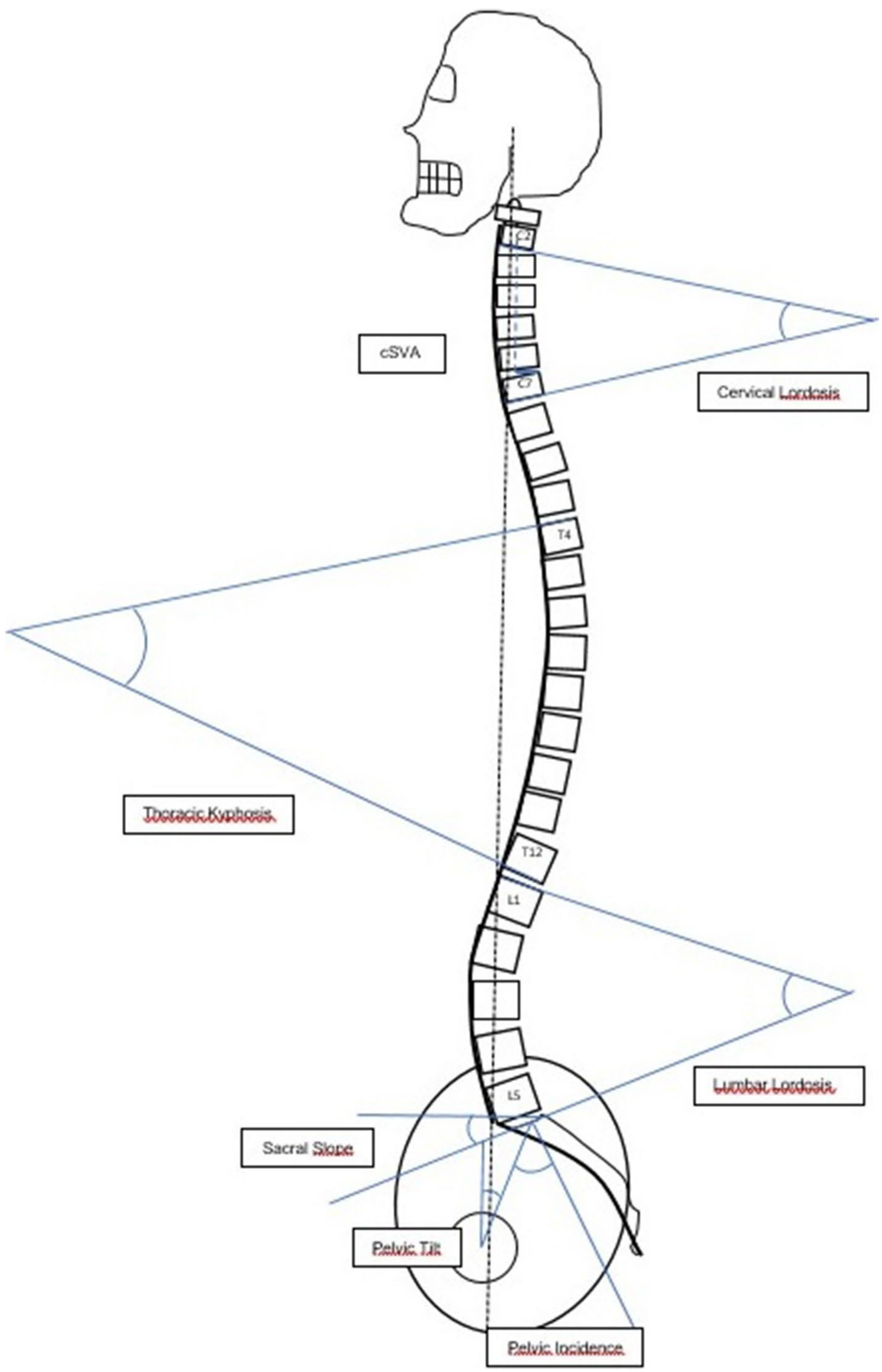


Fig. 1 Radiographic sagittal alignment parameters

- thoracic kyphosis was measured from the upper plateau of T5 to the T12 lower plateau;
- lumbar lordosis was measured from the upper plateau of L1 to the upper plateau of S1;
- pelvic incidence (PI) was measured as the angle between a line perpendicular to the ground at the midpoint of the sacral plateau, and a line connecting this point to the central axis of the femoral head, on a lateral radiograph of the pelvis;
- pelvic tilt (PT) was measured as the angle between a line perpendicular to the ground and a vertical line connecting the midpoint of the top plateau of S1, to the midpoint of the line that connects the center of the femoral heads, on a lateral radiograph of the pelvis;
- sacral slope (SS) was measured as the angle between the upper plateau of S1 and a line parallel to the ground, on a lateral radiograph of the pelvis;
- cervical sagittal vertical axis (cSVA) was measured as the distance between the line perpendicular to the ground passing through the center of C2 and the posterior superior border of C7, on a lateral radiograph of the cervical spine.

The Roussouly classification [32] was used to better evaluate the sagittal postural pattern. It is based on sacral slope and then on the number of vertebrae in the lordosis, and it initially described four types:

- Type 1 Lordosis. The sacral slope is less than 35°, which is usually associated with a low pelvic incidence. The apex of the lumbar lordosis is in the center of L5 vertebral body. The lower arc of lordosis is minimal, decreasing toward zero as the sacral slope approaches the horizontal. The inflection point is low and posterior, creating a short lordosis with a negative lordosis tilt angle. The upper spine has a significant kyphosis of the thoracolumbar junction and thorax.
- Type 2 Lordosis. The sacral slope is less than 35°. The apex of the lumbar lordosis is located at base of the L4 vertebral body. The lower arc of lordosis is relatively flat. The inflection point is higher and more anterior, decreasing the lordosis tilt angle but increasing the number of vertebral bodies included in the lordosis. The entire spine is relatively hypolordotic and hypokyphotic.
- Type 3 Lordosis. The sacral slope is between 35° and 45°.
- Type 4 Lordosis. The sacral slope is greater than 45°.

We used the D'Agostino–Pearson test to access the normality of the groups. We then used the Student's *t*-test or the Mann–Whitney *U* test to evaluate the difference between the groups, using the first for normally distributed groups and the second for the others. *p* values were considered significant when they were lower than 0.05. The correlation between the two groups was assessed with Pearson's correlation for normally distributed and Spearman's correlation test for non-normally distributed variables. We used BioEstat 5.3 software for statistical analyses.

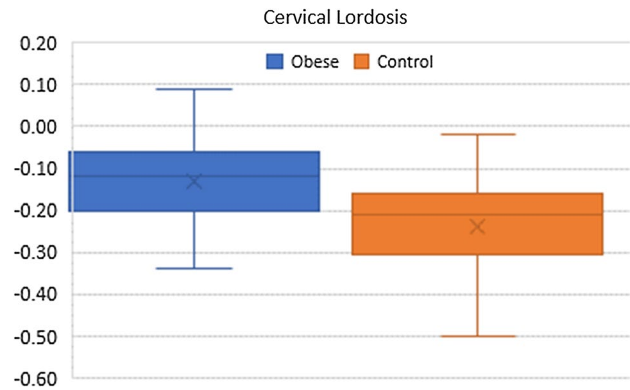


Fig. 2 Cervical lordosis results

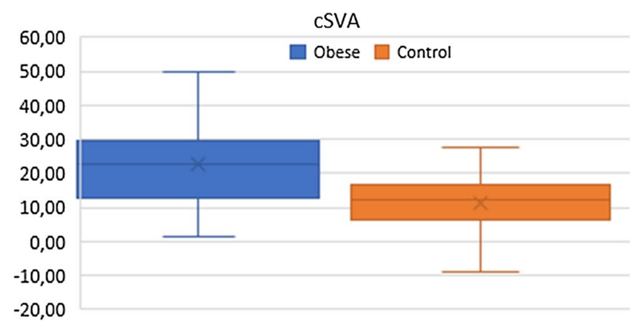


Fig. 3 cSVA results

Results

The groups were not significantly different in composition regarding sex, with five (17%) men in the group of obese patients and five (20%) in the control group. Although they were not normally distributed, their distribution was equivalent, as shown by Student's *t*-test. The groups were also equivalent with respect to age, with the mean age being 51 years in the obese group and 50 years in the control group. Only BMI showed a statistically significant difference, as shown in Table 1.

In relation to measures of spinal curvature, the only significant differences between the groups were in cervical lordosis and in cervical sagittal vertical axis (cSVA), as shown in Figs. 2 and 3.

Although they were significantly different, the Pearson correlation coefficients could not establish a definite relationship between BMI and the cervical lordosis (CL) and

Table 2 Radiographic and questionnaires results

Group	Obese			Control			Difference
	Mean	SD	Normality	Mean	SD	Normality	*p* value
CL	−13.30	12.02	0.10	−24.08	12.25	0.49	0.0023
cSVA	22.85	12.47	0.43	11.50	9.88	0.46	0.0025
TK	30.63	9.62	< 0.0001	31.92	9.80	0.08	0.6274
LL	−46.47	11.94	0.18	−47.48	12.90	0.22	0.7628
PT	15.13	6.25	0.67	12.10	8.38	0.47	0.1305
PI	49.97	8.38	0.16	50.00	11.03	0.57	0.9911
SS	35.00	9.32	0.26	37.85	11.35	0.38	0.3106
NDI	0.50	0.11	0.42	0.16	0.07	0.49	< 0.0001
ODI	0.48	0.11	0.52	0.08	0.06	0.35	< 0.0001
VAS cervical	4.20	1.69	0.65	0.40	0.82	< 0.0001	< 0.0001
VAS lumbar	4.40	1.92	0.94	0.40	0.82	< 0.0001	< 0.0001

Table 3 Main correlations with BMI

Pearson correlation with BMI	*r*	*p*
Cervical lordosis		
Obese	−0.0687	0.7181
Control	0.0851	0.6859
cSVA		
Obese	−0.0897	0.6375
Control	0.0163	0.9383
NDI		
Obese	0.1631	0.3890
Control	−0.0989	0.6381
ODI		
Obese	0.2627	0.1607
Control	0.0796	0.7051
VAS cervical		
Obese	0.0237	0.9012
Control	0.1423	0.4973
VAS lumbar		
Obese	0.0810	0.6704
Control	0.4270	0.0332

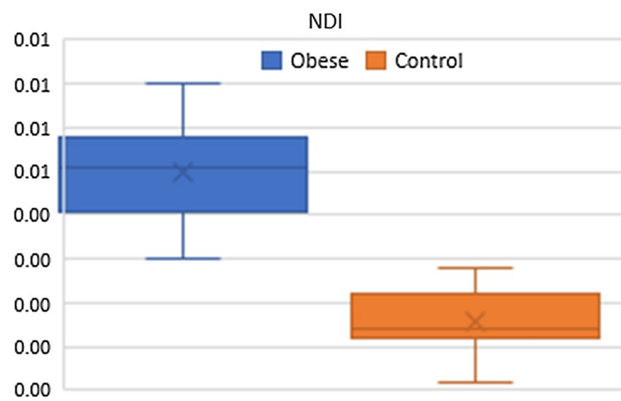


Fig. 4 NDI results

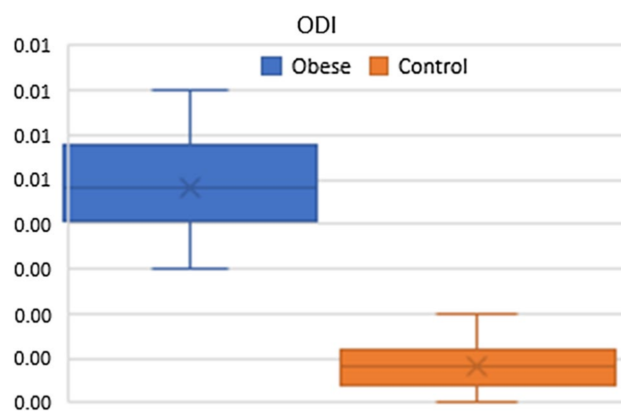


Fig. 5 ODI results

cSVA. Thoracic kyphosis (TK), lumbar lordosis (LL), and pelvic parameters showed no statistically significant differences between the groups. The results are shown in Table 2.

All the disability scores showed statistically significant differences ($p < 0.05$) between the two groups, but no definite correlation could be established between BMI and these scores by Pearson correlation coefficients ($p < 0.05$). These results are shown in Table 2, while in Table 3 we show the results of the Pearson correlation. Figures 4 and 5 also highlight these results.

In the obese group, the Pearson correlation showed a moderate inverse relationship between lumbar lordosis and BMI (− 0.3897). In the same group, a moderate relationship was also established between cSVA and thoracic kyphosis (0.3897) and cSVA and PI (0.380). The Pearson correlation coefficients established a strong relationship between NDI and ODI (0.848).

Discussion

Individuals in the obese group presented with painful symptoms in the cervical and lumbar spine more often than those in the control group. In addition, the group of obese individuals exhibited worse functional performance of the cervical and lumbar spine in the activities of daily living as evaluated by NDI and ODI, respectively, compared with the control group.

Although some studies [4, 33] have indicated that obese individuals have a higher incidence of musculoskeletal pain, including lumbago, it has not yet been defined if increased weight has a direct relationship with the prevalence and onset of back pain [23, 34].

Some studies have shown a correlation between obesity and back pain [6, 13, 20, 23], suggesting that obesity is a risk factor for causing back pain. Other studies have determined that this relationship is weak or non-existent, raising the possibility that other factors are causing the pain including environmental factors and genetics [13, 22, 35, 36].

Some studies have also noted a direct relationship between obesity and low back pain [22, 36]. The present study showed an increase in pain reported in the group of obese patients compared with the control patients. Similarly, the obese patients also had worse functional outcomes compared with the control patients, as measured by the NDI and ODI. This result does not agree with other studies [34, 37–39], suggesting that other factors may be related to the back pain.

A strong correlation was evidenced between ODI and NDI classifications. This result is in accordance to the higher rates of cervical and lumbar pain and disability evidenced in our study and others [6]. It is possible that the process of cervical and lumbar lordosis adaptation in the global compensation of sagittal balance could be related to this disability functional relationship.

There are few publications regarding alterations in sagittal spine balance associated with obesity. In a bibliographic survey, we identified few articles that describe the angular values in physiological curves in the cervical, dorsal, and lumbar spine in obese subjects, and even fewer in patients with morbid obesity. González-Sánchez et al. [34] found no difference in sagittal curvature of the spine between obese and non-obese individuals, but they used an electromagnetic apparatus and no radiographic examinations. To our knowledge, it has not been described in the literature how the spine curves behave and their compensatory mechanisms in severe obesity (BMI > 40).

An important finding of our study was the existence of significantly smaller values of cervical lordosis in obese subjects compared with the control group. We also found a significantly larger cSVA in obese subjects. The average value of the cSVA was 2.2 cm in the obese group, which is significantly greater than that of the control group (0.63 cm). However, this value did not indicate severe cervical sagittal imbalance (greater than 4 cm) [39].

In the present study, a mild inverse correlation was established between lumbar lordosis and BMI in the obese group. An increase on BMI was related to lower values of lumbar lordosis in the obese individuals. Boulay et al. [40] also found a correlation between BMI and lordosis.

A mild correlation was established between cSVA and thoracic kyphosis (TK) and cSVA and pelvic incidence (PI) in the obese group. These correlations may indicate an adaptation process in the spine to obtain sagittal balance. This process may take many years and could be related to higher BMI in the first years of life if we consider the PI values [21, 27, 41]. These results are original and deserve further studies.

The study of cervical spine alignment is gaining greater importance as it relates to the postural compensation mechanisms after large surgeries for thoracolumbar deformities and cervical myelopathy. The importance of the cervical spine in the global compensation of sagittal balance has also been increasingly valued [42, 43].

The difference in the cervical measures that we observed between the obese and control groups has not been shown in the literature. The importance of these findings requires further research with a larger group of patients, and merits re-evaluation and additional analyses in the future.

No differences were observed between the average values of the pelvic measures between the obese and control groups. Guigui et al. [44] also did not show any correlation between BMI and pelvic parameters. This conflicted with the results of Kulcheski et al. [45], who observed increased values of pelvic incidence and pelvic tilt in obese patients. However, these authors did not have a control group and compared their results with values in the literature, which may have created bias in the comparison and analysis of the data Roussouly and Pinheiro-Franco [46] and Romero-Vargas et al. [47] reinforced the importance of this topic, given the absence of definitive findings and the importance of the subject.

The Roussouly classification helps in the understanding of the distribution of contact forces for different lumbar alignments [41]. Although there was a 50% prevalence of Type 2 in the obese group, it did not reach statistical significance. Chanplakorn et al. [27] has reported a similar distribution of postural patterns across BMI categories. Araújo et al. [21] described that (BMI > 30) would be related to Roussouly Type 1; otherwise, types 2 and 4 would be associated with overweight (BMI 25 to 29.9).

In addition, weight loss in patients with severe obesity (i.e., BMI between 35 and 40), and a reduction in the clinical complications related to obesity, such as diabetes, obstructive sleep apnea, and other cardiovascular risk factors, can be achieved with surgical treatment [48]. However, a clear benefit in relation to back pain has not yet been demonstrated.

This study, as with all cross-sectional studies, cannot unequivocally determine cause and effect, and the results cannot be extrapolated to the general population because of the small sample size. A larger study with a greater number of subjects is necessary to validate our results. However, the originality of the approach of evaluating radiographic patterns of the spine segments, as well as pain and functional parameters in severely obese individuals, makes this study a valuable contribution to the literature. This research sought to evaluate the prevalence of cervical and lumbar pain and analyze whether there would be abnormalities in sagittal spine alignment in obese patients. These results may be useful in guiding the treatment of obese patients with back pain.

Acknowledgments We thank Peter Mittwede, MD, PhD, from Edanz Group (www.edanzediting.com/ac) for editing a draft of this manuscript. We thank Isabela Peixoto de Mello for drawing the figure representing the measures.

Author contributions Alexandre Peixoto de Mello and Glaucus Cajaty dos Santos Martins designed the studies, analyzed data and wrote the manuscript. André Raposo Heringer and Raphael Barbosa Gamallo collected the data from the patients. Maurício de Pinho Gama revised the statistics. Luiz Felippe dos Santos Martins Filho, Antônio Vítor de Abreu and Antonio Carlos Pires Carvalho revised the analyzed data and the manuscript.

Funding The study was performed using personal resources from the authors and exams performed at Hospital Federal de Ipanema as part of the pre-operative evaluation.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest.

References

1. Kushner RF, Kahan S (2018) Introduction: the state of obesity in 2017. Med Clin North Am 102:1–11. https://doi.org/10.1016/j.mcna.2017.08.003
2. World Health Organization, WHO (2018) WHO Obesity and Overweight. In: World Health Organisation http://www.who.int/mediacentre/factsheets/fs311/en/. Accessed 14 Feb 2019
3. Dobbs R, Sawers C, Thompson F et al (2014) Overcoming obesity: an initial economic analysis. McKinsey Global Institute, New York City
4. Deere KC, Clinch J, Holliday K et al (2012) Obesity is a risk factor for musculoskeletal pain in adolescents: findings from a population-based cohort. Pain 153:1932–1938. https://doi.org/10.1016/j.pain.2012.06.006
5. Martins GC, Martins Filho LF, Raposo AH et al (2018) Avaliação radiográfica e de sintomatologia dolorosa do joelho em indivíduos com obesidade grave—estudo controlado transversal. Rev Bras Ortop 53:740–746. https://doi.org/10.1016/j.rbo.2017.06.024
6. Nilsen TIL, Holtermann A, Mork PJ (2011) Physical exercise, body mass index, and risk of chronic pain in the low back and neck/shoulders: longitudinal data from the Nord-Trøndelag health study. Am J Epidemiol 174:267–273. https://doi.org/10.1093/aje/kwr087
7. Heuch I, Heuch I, Hagen K, Zwart J-A (2013) Body mass index as a risk factor for developing chronic low back pain: a follow-up in the Nord-Trondelag health study. Spine (Phila Pa 1976) 38:133–139. https://doi.org/10.1097/BRS.0b013e3182647af2
8. Wearing SC, Hennig EM, Byrne NM et al (2006) Musculoskeletal disorders associated with obesity: a biomechanical perspective. Obes Rev 7:239–250. https://doi.org/10.1111/j.1467-789X.2006.00251.x
9. Ghezelbash F, Shirazi-Adl A, Plamondon A et al (2017) Obesity and obesity shape markedly influence spine biomechanics: a subject-specific risk assessment model. Ann Biomed Eng 45:2373–2382. https://doi.org/10.1007/s10439-017-1868-7
10. Gegout PP, Francin PJ, Mainard D, Presle N (2008) Adipokines in osteoarthritis: friends or foes of cartilage homeostasis? Jt Bone Spine 75:669–671. https://doi.org/10.1016/j.jbspin.2008.07.008
11. Conde J, Scotece M, Gómez R et al (2011) Adipokines: biofactors from white adipose tissue. A complex hub among inflammation, metabolism, and immunity. BioFactors 37:413–420. https://doi.org/10.1002/biof.185
12. Thijssen E, van Caam A, van der Kraan PM (2015) Obesity and osteoarthritis, more than just wear and tear: pivotal roles for inflamed adipose tissue and dyslipidaemia in obesity-induced osteoarthritis. Rheumatology 54:588–600. https://doi.org/10.1093/rheumatology/keu464
13. Segar AH, Fairbank JCT, Urban J (2018) Leptin and the intervertebral disc: a biochemical link exists between obesity, intervertebral disc degeneration and low back pain—an in vitro study in a bovine model. Eur Spine J. https://doi.org/10.1007/s00586-018-5778-7
14. Terashima Y, Kakutani K, Yurube T et al (2016) Expression of adiponectin receptors in human and rat intervertebral disc cells and changes in receptor expression during disc degeneration using a rat tail temporary static compression model. J Orthop Surg Res 11:147. https://doi.org/10.1186/s13018-016-0481-z
15. Goettler A, Grosse A, Sonntag D (2017) Productivity loss due to overweight and obesity: a systematic review of indirect costs. BMJ Open 7:e014632. https://doi.org/10.1136/bmjopen-2016-014632
16. Di Cesare M, Bentham J, Stevens GA et al (2016) Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19.2 million participants. Lancet 387:1377–1396. https://doi.org/10.1016/S0140-6736(16)30054-X
17. Buchbinder R, Blyth FM, March LM et al (2013) Placing the global burden of low back pain in context. Best Pract Res Clin Rheumatol 27:575–589. https://doi.org/10.1016/j.berh.2013.10.007
18. Hoy D, March L, Brooks P et al (2010) Measuring the global burden of low back pain. Best Pract Res Clin Rheumatol 24:155–165
19. Wynne-Jones G, Cowen J, Jordan JL et al (2014) Absence from work and return to work in people with back pain: a systematic review and meta-analysis. Occup Environ Med 71:448–456. https://doi.org/10.1136/oemed-2013-101571
20. Manchikanti L, Singh V, Datta S et al (2009) Comprehensive review of epidemiology, scope, and impact of spinal pain. Pain Physician 12:E35–E70
21. Araújo F, Lucas R, Alegrete N et al (2014) Individual and contextual characteristics as determinants of sagittal standing posture: a population-based study of adults. Spine J 14:2373–2383. https://doi.org/10.1016/j.spinee.2014.01.040

22. Dario AB, Loureiro Ferreira M, Refshauge K et al (2017) Obesity does not increase the risk of chronic low back pain when genetics are considered. A prospective study of Spanish adult twins. Spine J 17:282–290. https://doi.org/10.1016/j.spinee.2016.10.006
23. Lake JK, Power C, Cole TJ (2000) Back pain and obesity in the 1958 British birth cohort: cause or effect? J Clin Epidemiol 53:245–250. https://doi.org/10.1016/S0895-4356(99)00155-9
24. Chaléat-Valayer E, Mac-Thiong J-M, Paquet J et al (2011) Sagittal spino-pelvic alignment in chronic low back pain. Eur Spine J 20:634–640. https://doi.org/10.1007/s00586-011-1931-2
25. Chun S-W, Lim C-Y, Kim K et al (2017) The relationships between low back pain and lumbar lordosis: a systematic review and meta-analysis. Spine J 17:1180–1191. https://doi.org/10.1016/j.spinee.2017.04.034
26. Vismara L, Menegoni F, Zaina F et al (2010) Effect of obesity and low back pain on spinal mobility: a cross sectional study in women. J Neuroeng Rehabil 7:3. https://doi.org/10.1186/1743-0003-7-3
27. Chanplakorn P, Sa-ngasoongsong P, Wongsak S et al (2012) The correlation between the sagittal lumbopelvic alignments in standing position and the risk factors influencing low back pain. Orthop Rev (Pavia) 3:11. https://doi.org/10.4081/or.2012.e11
28. World Health Organization (WHO) (2000) Obesity: preventing and managing the global epidemic. Report of a WHO consultation, Switzerland
29. Vernon H (2008) The neck disability index: state-of-the-art, 1991–2008. J Manip Physiol Ther 31:491–502. https://doi.org/10.1016/j.jmpt.2008.08.006
30. Fairbank JC, Pynsent PB (2000) The Oswestry disability index. Spine (Phila Pa 1976) 25:2940–2952. https://doi.org/10.1097/00007632-200011150-00017 **discussion 2952**
31. Akbar M, Terran J, Ames CP et al (2013) Use of surgimap spine in sagittal plane analysis, osteotomy planning, and correction calculation. Neurosurg Clin N Am 24:163–172
32. Roussouly P, Gollogly S, Berthonnaud E, Dimnet J (2005) Classification of the normal variation in the sagittal alignment of the human lumbar spine and pelvis in the standing position. Spine (Phila Pa 1976) 30:346–353. https://doi.org/10.1097/01.brs.0000152379.54463.65
33. Tsuritani I, Honda R, Noborisaka Y et al (2002) Impact of obesity on musculoskeletal pain and difficulty of daily movements in Japanese middle-aged women. Maturitas 42:23–30. https://doi.org/10.1016/S0378-5122(02)00025-7
34. González-Sánchez M, Luo J, Lee R, Cuesta-Vargas AI (2014) Spine curvature analysis between participants with obesity and normal weight participants: a biplanar electromagnetic device measurement. Biomed Res Int. https://doi.org/10.1155/2014/935151
35. Leboeuf-Yde C (2000) Body weight and low back pain. A systematic literature review of 56 journal articles reporting on 65 epidemiologic studies. Spine (Phila Pa 1976) 25:226–237. https://doi.org/10.1097/00007632-200001150-00015
36. Segar AH, Urban JPG, Fairbank JCT, Judge A (2016) The association between body mass index (BMI) and back or leg pain in patients with spinal conditions. Spine (Phila Pa 1976) 41:E1237–E1243. https://doi.org/10.1097/BRS.0000000000001606
37. Brooks C, Siegler JC, Cheema BS, Marshall PWM (2013) No relationship between body mass index and changes in pain and disability after exercise rehabilitation for patients with mild to moderate chronic low back pain. Spine (Phila Pa 1976) 38:2190–2195. https://doi.org/10.1097/BRS.0000000000000002
38. Heuch I, Heuch I, Hagen K, Zwart J-A (2015) A Comparison of anthropometric measures for assessing the association between body size and risk of chronic low back pain: the HUNT study. PLoS One 10:e0141268. https://doi.org/10.1371/journal.pone.0141268
39. Tang JA, Scheer JK, Smith JS et al (2012) The impact of standing regional cervical sagittal alignment on outcomes in posterior cervical fusion surgery. Neurosurgery 71:662–669. https://doi.org/10.1227/NEU.0b013e31826100c9
40. Boulay C, Tardieu C, Hecquet J et al (2006) Sagittal alignment of spine and pelvis regulated by pelvic incidence: standard values and prediction of lordosis. Eur Spine J 15:415–422. https://doi.org/10.1007/s00586-005-0984-5
41. Bao H, Lafage R, Liabaud B et al (2018) Three types of sagittal alignment regarding compensation in asymptomatic adults: the contribution of the spine and lower limbs. Eur Spine J 27:397–405. https://doi.org/10.1007/s00586-017-5159-7
42. Smith JS, Lafage V, Ryan DJ et al (2013) Association of myelopathy scores with cervical sagittal balance and normalized spinal cord volume. Spine (Phila Pa 1976) 38:S161–S170. https://doi.org/10.1097/BRS.0b013e3182a7eb9e
43. Lee S-H, Son E-S, Seo E-M et al (2015) Factors determining cervical spine sagittal balance in asymptomatic adults: correlation with spinopelvic balance and thoracic inlet alignment. Spine J 15:705–712. https://doi.org/10.1016/j.spinee.2013.06.059
44. Guigui P, Levassor N, Rillardon L et al (2003) Physiological value of pelvic and spinal parameters of sagital balance: analysis of 250 healthy volunteers. Rev Chir Orthop Reparatrice Appar Mot 89:496–506. RCO-10-2003-89-6-0035-1040-101019-ART3
45. Kulcheski AL, Soler X, Graells BML, Baretta G (2013) Avaliação angular do equilíbrio sagital em pacientes obesos. Coluna/Columna 12:224–227. https://doi.org/10.1590/S1808-18512013000300010
46. Roussouly P, Pinheiro-Franco JL (2011) Biomechanical analysis of the spino-pelvic organization and adaptation in pathology. Eur Spine J 20:609–618
47. Romero-Vargas S, Zárate-Kalfópulos B, Otero-Cámara E et al (2013) The impact of body mass index and central obesity on the spino-pelvic parameters: a correlation study. Eur Spine J 22:878–882
48. National Institutes of Health (1991) Gastrointestinal surgery for severe obesity. In: Consensus statement. National Institutes of Health consensus development conference, pp 1–20

Affiliations

Alexandre Peixoto de Mello[1,2] · **Glaucus Cajaty dos Santos Martins**[3,4] · **André Raposo Heringer**[4] · **Raphael Barbosa Gamallo**[4] · **Luiz Felippe dos Santos Martins Filho**[4] · **Antônio Vítor de Abreu**[3] · **Antonio Carlos Pires Carvalho**[3] · **Maurício de Pinho Gama**[3,5]

1 Universidade Federal do Estado do Rio de Janeiro (UNIRIO), Rua Silva Ramos 32, Rio de Janeiro, RJ, Brazil

2 Instituto Nacional de Traumatologia e Ortopedia (INTO), Rio de Janeiro, Brazil

3 Universidade Federal do Rio de Janeiro (UFRJ), Rio de Janeiro, Brazil

4 Hospital Federal de Ipanema (HFI), Rio de Janeiro, Brazil

5 Universidade de Brasília (UnB), Brasilia, Brazil