Evaluation of Respiratory Muscle Strength in the Acute Phase of Stroke: The Role of Aging and Anthropometric Variables

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Background: During hospitalization, stroke patients are bedridden due to neurologic impairment, leading to loss of muscle mass, weakness, and functional limitation. There have been few studies examining respiratory muscle strength (RMS) in the acute phase of stroke. *Objective:* This study aimed to evaluate the RMS of patients with acute stroke compared with predicted values and to relate this to anthropometric variables, risk factors, and neurologic severity. *Methods:* This is a cross-sectional study in the acute phase of stroke. After admission, RMS was evaluated by maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP); anthropometric data were collected; and neurologic severity was evaluated by the National Institutes of Health Stroke Scale. The analysis of MIP and MEP with predicted values was performed by chi-square test, and the relationship between anthropometric variables, risk factors, and neurologic severity was determined through multiple linear regression followed by residue analysis by the Shapiro-Wilk test; $P < .05$ was considered statistically significant. *Results:* In the 32 patients studied, MIP and MEP were reduced when compared with the predicted values. MIP declined significantly by 4.39 points for each 1 kg/m^2 increase in body mass index (BMI), and MEP declined significantly by an average of 3.89 points for each 1 kg/m^2 increase in BMI. There was no statistically significant relationship between MIP or MEP and risk factors, and between MIP or MIP and neurologic severity in acute phase of stroke. *Conclusion:* There is a reduction of RMS in the acute phase

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of stroke, and RMS was lower in individuals with increased age and BMI. **Key Words:** Stroke—respiratory muscle strength—MIP—MEP—obesity—aging.

Introduction

Stroke affects approximately 16.9 million people worldwide, generating about 100,000 people with functional disability per year.[1] Stroke is the main cause of mortality and chronic disability in adults in Latin America and Brazil, with around 200,000 cases annually.[2-4]

Stroke is defined as neuronal death due to prolonged ischemia, which is caused by obstruction to cerebral blood flow or intracranial hemorrhage.[5,6] About 90% of patients present hemiparesis, with decreased strength and motor control after stroke, compromising daily life activities, mobility, and locomotion.[7,8]

During hospitalization, stroke patients become bedridden due to neurologic impairment, leading to loss of muscle mass, weakness, and functional limitation.[9] The main complications during hospitalization due to immobility are reduction of chest expansion and respiratory complications, with pneumonia being the most common in this population.[10]

Respiratory complications may also occur due to changes in respiratory patterns, as well as from weakness of respiratory muscles. Respiratory muscle strength (RMS) is one of the most important factors in maintaining intact lung function.[11] Weakened muscles result in decreased diaphragmatic movement and chest expansion, thus increasing mechanical resistance to respiration, and decreased ventilation and cough effectiveness, thus leading to difficulty in eliminating secretions, which significantly increases the risk of lung infections.[12] In detailed physiological studies of respiratory muscle function, it was found that parameters of RMS (maximum inspiratory and expiratory mouth pressures, sniff pressure) in stroke subjects were reduced by one third to one half, and can be of importance in achieving physical capacity a posteriori and the ability to perform daily life activities in the long term.[13,14]

There have been few studies that examine RMS in the acute phase of stroke, and the present study has as an innovation the description and the relationship of sociodemographic factors and their impact on RMS in the acute phase of stroke, so that health professionals are attentive in the conduct of clinical care and to the patient with potential risk of respiratory complications. We hypothesized that low values of inspiratory and expiratory pressure decrease the efficiency of ventilatory mechanics, especially in individuals with aging and higher comorbidities in acute phase of stroke; therefore, the aim of this study was to evaluate the RMS of patients with acute stroke compared with the predicted values and to relate this to anthropometric variables and severity of the neurologic condition.

Methods

Study Design, Setting, and Participants

This is a cross-sectional study of patients admitted to the stroke unit of the Botucatu Medical School from July until December 2016. We evaluated individuals with a diagnosis of ischemic stroke confirmed by neuroimaging (computed tomography or magnetic resonance), aged over 18 years, and who scored 0 in item 1a of the National Institutes of Health Stroke Scale (NIHSS). Patients with hemorrhagic stroke, previous complaints of dysphagia, prior stroke with a score of more than 2 on the modified Rankin scale, pre-existing dementia, clinical instability, comatose state, acute and chronic pulmonary diseases, and other neurologic diseases were excluded. After signing the informed consent, RMS and both demographic and anthropometric data, in addition to the NIHSS scale, were evaluated in the stroke unit.

Variables

Exposures

The independent variable RMS was evaluated using the Comercial Médica (São Paulo, Brazil) manovacuometer, with maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) being evaluated until 48 hours after admission.

Measurements

(A) Maximum inspiratory pressure (MIP): the patient was positioned at 45° on the bed and was requested to perform maximum expiration up to the residual volume level. Subsequently, maximal inspiratory effort was requested, and held for about 1 second. The reading of the measurement value was checked directly on the manovacuometer display. The maneuvers were repeated 3 times, at intervals of about 1 minute, with the highest MIP value used in the analysis.

(B) Maximum expiratory pressure (MEP): the patient was positioned at 45° on the bed and was requested to perform maximum inspiratory effort until total lung capacity was reached. A maximal expiratory effort was then requested, and maintained for about 1 second. The reading of the measurement value was checked directly on the manovacuometer display. The maneuvers were repeated 3 times, at intervals of about 1 minute, with the highest MEP value used in the analysis.

The test was performed by a trained physiotherapist with more than 5 years of clinical experience in RMS testing. During the maneuvers (MIP and MEP), the patient used a mouthpiece and a nose clip. During the force measurement maneuver, the manovacuometer was occluded manually. In the mouthpiece was a minimal orifice to allow inspiratory flow, in order to avoid closure of the glottis during the maneuver.[15,16]

Confounding Factors

The following clinical variables were evaluated as potential confounders:

(A) Hemodynamic variables: respiratory rate, systolic and diastolic blood pressure, heart rate, peripheral oxygen saturation, blood glucose monitoring, and temperature.

(B) Anthropometric variables: body weight (kg) was measured by means of a digital scale in patients who could perform an orthostatic posture or by an electronic balance fixed to a crane for bedridden patients. The height (m) was measured by means of a stadiometer fixed to the wall for standing patients. When it was not possible to gauge weight and height, weight and height were estimated by the nutritionist responsible, through formulas taking into consideration age, race, arm circumference, and knee height. After measuring weight and height, the body mass index (BMI) was calculated using the formula: weight (kg)/height $(m)^2$.

(C) Collection of risk factors through the patient admission procedure: hypertension, smoking, obesity, alcoholism, Chagas disease, congestive heart failure, coronary artery disease, diabetes, dyslipidemia, depression, prior stroke or transient ischemic attack, and prior acute myocardial infarction.

(D) Neurologic severity: evaluated through the NIHSS composed of 11 neurologic evaluation items; the higher the score, the worse the neurologic status.[17]

Sample Size

Because we are using a sample representative of the target population, our sampling is considered to be intentional and nonprobabilistic. We needed a minimum of 60 subjects to obtain a maximum sampling error of 7.5% and a confidence level of 95%.

Statistical Methods

The MIP and MEP values were described in the mean and standard deviation and the comparison with the predictive values was obtained using the Student's *t*-test. The predicted values of MIP and MEP for the study population were obtained through the equations proposed by Neder et al.[18] The analysis of the relationship between MIP and MEP according to the clinical variables was performed through multiple linear regression followed by residue analysis by the Shapiro–Wilk test. Data were analyzed using SPSS version 22 (IBM SPSS Statistics for Windows, IBM Corp., Armonk, NY), and considered statistically significant at $P < .05$.

Results

Thirty-two patients were studied: 43.7% were men, with a median age of 62.5 years, 87.5% were Caucasian. The most commonly observed risk factors were hypertension (81.2%), smoking (53.1%), and diabetes (37.5%). The TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification with the most frequency (34.3%) was lacunar stroke, and 46.9% of the patients presented strokes of unknown cause. The previous modified Rankin scale was 0 in 71.9% of patients, and the NIHSS median score for hospital admission was 3. Of the patients evaluated, 18.7% were thrombolytic and 53.1% underwent physiotherapy during hospitalization (Table 1). Regarding the hemodynamic variables, the values obtained were within the normal range, which made it possible to perform the manovacuometry safely and without intercurrences (Table 2).

The median of MIP obtained was 62.4 ± 35.3 cmH_20 in total (85 ± 36.2 cmH_20 in men and 46.9 ± 25.4 cmH_20 in women), whereas for MEP, it was 65.2 ± 35.9 cmH_20 in total (82.4 ± 28.9 cmH_20 in men and 51.2 ± 28.8 cmH_20 in women). When respiratory pressures were compared with the predicted value, a significant reduction in MIP was observed in the total sample ($P = .001$): $P = 005$ in men and $P = .001$ in women. When compared with the predicted values, a reduction in MEP was observed in the total sample ($P = .001$): $P = .001$ in men and $P = .001$ in women (Table 3).

MIP dropped significantly by 4.39 points for each 1 kg/m^2 increase in BMI. In this analysis, age was considered as confounding. There was no statistically significant relationship between MIP and other risk factors, severity of the neurologic condition in the acute phase of stroke, and treatment received (Table 4).

MEP declined significantly by an average of 3.89 points per 1 kg/m^2 increase in BMI. There was no statistically significant relationship between MEP with other risk factors, severity of the neurologic condition in the acute phase of stroke, and treatment received (Table 5).

Discussion

The respiratory pressures presented a reduction in the acute phase of stroke when compared with the predicted value for each sex and corrected for age. In the acute phase of stroke, inhibition of the cortical activity contributes to the reduction of motor control, hemiplegia, and weakness of the trunk muscles, mainly of the diaphragm and abdominal muscle.[19-22] The abdominal

Table 1. *Demographic and clinical variables of the patients studied*

Variable	n	%
Demographic		
Sex (male)	14	43.7
Age (y)*	62.5 (31-89)	
Race		
Caucasian	28	87.5
Non-Caucasian	4	12.5
Risk factors		
Hypertension	26	81.2
Smoking	17	53.1
Obesity	5	15.6
Diabetes	12	37.5
Alcoholism	10	31.2
Dyslipidemia	8	25
Prior stroke	8	25
CHF	0	0
Prior AMI	2	6.2
CAD	0	0
Depression	2	6.2
Bamford		
LACS	11	34.3
PACS	9	28.1
TACS	3	9.4
POCS	9	28.1
TOAST		
Large-artery atherosclerosis	3	9.4
Cardioembolism	6	18.7
Small-vessel occlusion	3	9.4
Other determined etiology	5	15.6
Undetermined etiology	15	46.9
Prior mRS		
0	23	71.9
1	5	15.6
2	4	12.5
NIHSS at admission*	3 (0-16)	
Thrombolysis	6	18.7
Physioterapy	17	53.1

Abbreviations: AMI, acute myocardial infarction; CAD, coronary artery disease; CHF, congestive heart failure; LACS, lacunar stroke; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; PACS, partial anterior circulation; POCS, posterior circulation; TACS, total anterior circulation; TOAST, Trial of Org 10172 in Acute Stroke Treatment.

*values in median.

muscles play an important role during inspiration, contributing directly to the action of the diaphragm for the maintenance of abdominal wall tonus. The increase in abdominal pressure developed during inspiration is transmitted to the lower rib cage by the apposing diaphragmatic fibers and may, therefore, improve the ability of the diaphragm to raise the lower rib cage. The weakness of the abdominal muscles may affect the synergism between the abdominal and diaphragm muscles, impairing the capacity of the diaphragm to generate force.[23]

Table 2. *Hemodynamic variables of the patients*

Variable	
Respiratory rate (bpm)	19 (12-36)
Blood pressure	
Systolic (mmHg)	144.5 (96-191)
Diastolic (mmHg)	83 (65-114)
Mean blood pressure (mmHg)	109.5 (65-141)
Heart rate (bpm)	72.5 (44-98)
Peripheral Oxygen Saturation (%)	96 (86-100)
Temperature (°C)	36.4 (36.2-36.8)

In the present study, there was a negative correlation between inspiratory muscle strength and both age and BMI: older patients and those with higher BMI had lower inspiratory muscle strength.

In relation to age, this may be related to the aging process that brings about changes in muscle functionality and loss of muscle fibers; this also occurs in the respiratory system. One of the main changes in the respiratory system with advancing age is reduction of the elastic recoil of the lungs and the compliance of the chest wall, leading to progressive stiffening due to the process of calcification of the ribs and vertebral joints.[24,25]

Watsford et al conducted a study with 72 elderly people divided into 2 groups of men and women and subdivided by age group (group A: 50-59, group B: 60-69, and group C: 70-79 years).[26] There was a difference in the rate of muscle strength reduction between men and women, but a reduction in RMS was observed in both sexes, and the higher the age group the greater the reduction in RMS. In a similar study with 100 men and women aged 40-89 years, divided into 5 subgroups by age group, there was a decrease in RMS with age.[27]

The mechanical effects of obesity on the respiratory system are well established. Obese subjects commonly have lower RMS, because the abdominal pressure influences the curvature and movement of the dia-

Table 3. *Comparison of the mean of the values obtained and predicted (in cmH₂0), of the respiratory pressures*

Sex	Pressure	Obtained	Predicted	P*
Man	MIP	85 ± 36.2	109.7 ± 7.7	.05
	MEP	82.4 ± 28.9	119.13 ± 7.8	.001
Woman	MIP	46.9 ± 25.4	78.2 ± 7.4	.001
	MEP	51.2 ± 28.8	75.5 ± 9.2	.001
Total	MIP	62.4 ± 35.3	89.4 ± 16.8	.001
	MEP	65.2 ± 35.9	91.1 ± 22.6	.001

Abbreviations: MEP, maximal expiratory pressure; MIP, maximal inspiratory pressure.

*Student's *t*-test; MIP predicted: man = −.80 (idade) + 155.3; woman = −.49 (idade) + 110.4. MEP predicted: man = −.81 (idade) + 165.3; woman = −.61 (idade) + 115.6.

Table 4. *MIP according to the admission clinical condition, corrected for the effect of age and BMI*

Variables	β	SE	*P*	CI 95%	
Age	.51	.41	.227	−.36	1.39
BMI	−4.39	1.09	.001	−6.71	−2.07
Hypertension	9.29	20.02	.649	−33.38	51.95
Smoking	−15.36	11.23	.192	−39.29	8.58
Obesity	24.80	19.16	.215	−16.05	65.64
Diabetes	−.99	15.01	.948	−32.99	31.02
Alcoholism	−27.22	13.52	.062	−56.05	1.60
Dyslipidemia	7.75	12.52	.545	−18.93	34.43
Prior stroke	−.17	13.54	.990	−29.03	28.69
Prior mRS	2.02	8.87	.823	−16.89	20.92
NIHSS at admission	3.02	2.20	.190	−1.67	7.71

Abbreviations: β, beta estimate; BMI, body mass index; CI, confidence interval; MIP, maximal inspiratory pressure; mRS, modified Rankin scale; NIHSS, National Institutes of Health Stroke Scale; SE, standard error.
pSW = 0.565 R^2 = 88%.

phragm, changing the generation of inspiratory pressure.[28,29] Some authors suggest that RMS in obesity may be compromised by the increased load that the muscles are required to overcome, and by some reduction in their capacity.[30,31] Chlif et al demonstrated that MIP was considerably lower in obese patients when compared with the control group.[32] Excessive adipose tissue around the chest wall may alter normal lung function, reducing both the expiratory reserve volume and functional residual capacity, due to changes in the mechanics of the thoracic wall.[33]

In a systematic review with an aim to examine the effects of respiratory muscle training on respiratory function, RMS, and exercise tolerance in patients poststroke, the authors concluded that respiratory muscle training should be considered an effective method of improving respiratory function, inspiratory muscle strength, and exercise tolerance in patients poststroke.[34] The evaluation and training of RMS has become increasingly important in stroke units, being an important parameter for functional recovery and for a decrease in the number of complications during hospitalization.

The main limitations of our study were the sample size, heterogeneity of the topographies of the stroke, and that half of the patients underwent physical therapy through an internal management process. The level of physical activity and sedentary behavior of the study participants were not evaluated, and this factor may be associated with a reduction in the overall muscular strength of the individuals. However, this work makes an important contribution to international stroke research in showing that obese and older individuals tend to suffer decreased RMS and are more susceptible to complications in the acute phase of stroke; multiprofessional teams should constantly monitor these cases. Based on the results obtained, there is a reduction of RMS in the acute phase of stroke, and RMS was lower in individuals with increased age and BMI.

Table 5. *MEP according to admission clinical condition, corrected for BMI effect*

Variable	β	SE	*P*	CI 95%	
BMI	3.89	.62	.000	2.57	5.21
Hypertension	−14.45	23.32	.544	−63.88	34.99
Smoking	24.08	13.36	.090	−4.24	52.40
Obesity	−33.92	19.70	.104	−75.67	7.84
Diabetes	−11.69	16.18	.480	−46.00	22.61
Alcoholism	26.81	14.50	.083	−3.93	57.56
Dyslipidemia	−26.77	14.49	.083	−57.48	3.95
Prior stroke	13.29	15.95	.417	−20.53	47.11
Prior mRS	−16.75	9.32	.091	−36.52	3.01
NIHSS at admission	−4.58	2.50	.086	−9.89	.73

Abbreviations: β, beta estimate; BMI, body mass index; CI, confidence interval; MEP, maximal expiratory pressure; mRS, modified Rankin scale; NIHSS, National Institutes of Health Stroke Scale; SE, standard error.
pSW = 0.974 R^2 = 85%.

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