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Original Article

Investigation of inspiratory intercostal muscle activity in patients with spinal cord injury: a pilot study using electromyography, ultrasonography, and respiratory inductance plethysmography

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Abstract. [Purpose] The respiratory function in patients with cervical spinal cord injury is influenced by inspiratory intercostal muscle function. However, inspiratory intercostal muscle activity has not been conclusively evaluated. We evaluated the inspiratory intercostal muscle activity in patients with cervical spinal cord injury by using inspiratory intercostal electromyography, respiratory inductance plethysmography, and ultrasonography. [Participants and Methods] Three patients with cervical spinal cord injury were assessed. The change in mean amplitude (rest vs. maximum inspiration) was calculated by using intercostal muscle electromyography. Changes in intercostal muscle thickness (resting expiration and maximum inspiration) were also evaluated on ultrasonography. The waveform was converted to spirometry ventilation with respiratory inductance plethysmography, and the waveform at the xiphoid was considered to determine the rib cage volume. Each index was compared with the inspiratory capacities in each case. [Results] Intercostal muscle electromyography failed to measure the notable myoelectric potential in all the patients. The rib cage volume was higher at higher inspiratory capacities. The changes in muscle thickness were not significantly different between the patients. [Conclusion] The rib cage volume (measured with inductance plethysmography) was greater in the patients with cervical spinal cord injury when inspiratory intercostal muscle activity was high. Respiratory inductance plethysmography can capture inspiratory intercostal muscle function in patients with cervical spinal cord injury.

Key words: Inspiratory intercostal muscle, Respiratory inductance plethysmography, Spinal cord injury

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INTRODUCTION

Cervical spinal cord injury (CSI) paralyzes the inspiratory intercostal muscles[1], which can further decrease the tidal volume[2]. Therefore, the role of inspiratory intercostal muscles is considered to be important for the inspiratory function of patients with CSI. Frostell et al.[3] demonstrated the technical feasibility of electromyography (EMG) in assessing the residual

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activity of the inspiratory intercostal muscles following CSI. However, needle EMG is invasive, and this limits its applicability in routine clinical practice. In contrast, Surface EMG (s-EMG) is a non-invasive option that has been used in other cases[4]. However, its inability to detect any intercostal activity in some individuals demands validation of its true usefulness.

Hodges et al.[5], with the help of ultrasound, evaluated muscle function in terms of how muscle thickness changes during contraction, and revealed that greater contraction strength was associated with a greater increase in the thickness of tibialis anterior, biceps brachii, and transversus abdominis. The feasibility of ultrasound evaluation in measuring the thickness of the inspiratory intercostal muscles in chronic obstructive pulmonary disease patients has already been demonstrated[6]. In our previous study, we hypothesized that inspiratory intercostal muscle thickness would increase with increasing inspiratory effort. We used ultrasound to measure thickness changes and confirmed this relationship in healthy adults[7]. However, the association between inspiratory intercostal muscle contraction and inspiratory effort has not been explored in individuals with CSI in a similar manner.

The inspiratory intercostal muscle function can also be evaluated based on the movement of a patient's chest during breathing. Typically, this is done using respiratory inductance plethysmography (RIP), in which a device measures changes in the circumference of the chest and abdomen with the help of transducer elastic bands wrapped around the chest and abdomen. RIP is also used to evaluate chest and abdominal movements in patients with CSI[8]. The ability of this technique to record inspiratory intercostal muscle activity has also been validated in an animal model of CSI, where the authors reported a positive association between rib cage displacement and EMG mean amplitude[9]. However, this has yet to be properly investigated in humans with CSI. Establishing a standard protocol to assess inspiratory intercostal muscle function remains an important challenge in the field, as related indicators could then be used to rate the efficacy of training interventions in patients with CSI.

Respiratory function can be measured in terms of inspiratory capacity (IC). Research on inspiratory muscle pacing[10] revealed that, in a CSI patient, about 50% of the IC originates from diaphragm contraction, while the remainder depends on the movement of the inspiratory intercostal muscles. This implies that, in the absence of diaphragm paralysis, IC reflects input from the residual descending motor pathways that project to inspiratory intercostal muscle, and should therefore decrease in patients with severe CSI in proportion to how much of this innervation is lost.

Accordingly, we hypothesized that a lower IC would be a predictive marker of reduction in contraction strength (i.e., lower root-mean-square [RMS] amplitude on EMG), contraction-related changes in muscle thickness (i.e., on ultrasound images), and rib cage mobility (i.e., RIP) during maximum inspiration for patients with CSI. Our objective in this study was to investigate the inspiratory intercostal muscle activity of CSI patients during maximum inspiration using EMG, ultrasound, and RIP, followed by the evaluation of the association between the corresponding indices and IC to explore how these indices could be used to evaluate the functioning of the inspiratory intercostal muscle.

PARTICIPANTS AND METHODS

We included patients with CSI hospitalized at the Ibaraki Prefectural University of Health Sciences Hospital between May and August 2018, who consented to participate in the research. Candidates were excluded if they had respiratory illness or trouble following directions. This study was conducted with the approval of the ethics committee of the Ibaraki Prefectural University of Health Sciences (2018, accession no. 830) and complied with the ethical standards of the Declaration of Helsinki 1964. Participants gave written informed consent after a thorough study orientation. Raw data were anonymized to obscure personally identifiable information.

Three patients were enrolled (Table 1). American Spinal Injury Association Impairment Scale (AIS) is used to evaluate neurological symptoms and diagnose the severity of spinal cord injury. The degree of injury and severity are evaluated based on the sensory of each medullary segment and the motor function of the key muscles[11]. American Spinal Injury Association (ASIA) motor score defines the key muscles of the 10 medullary segments of the upper limbs C5-Th1 and lower limbs L2-S1 and is the total of the scores of the manual muscle testing of each muscle on both sides (out of 100 points)[11].

Inspiratory intercostal muscle thickness was measured using a diagnostic ultrasound system (LOGIQ iM, GE Healthcare Japan Corp, Tokyo: B-mode, PRF=12.0 MHz, linear probe). The participants were evaluated in the supine position. A linear probe was placed over the 2nd intercostal space, 2–3 cm right of the right edge of the sternum, and the 2nd parasternal intercostal muscle was identified in the sagittal plane. The participants were verbally instructed to exhale normally and to inhale as deeply as possible. Static ultrasound images of the region were captured after resting expiration and maximum inspiration.

Intercostal muscle activity was measured using an s-EMG system (P-EMG plus; Oisaka Electronic Equipment, Hiroshima, Japan). The participants were evaluated in the supine position. Two surface electrodes were placed on the skin, 1 cm apart, directly above the 2nd parasternal intercostal muscle. The area was first scrubbed with alcohol-soaked cotton to minimize skin-electrode impedance. The ground electrode was placed on the right ulnar styloid process. Raw EMG signals were filtered through a 20–450 kHz bandpass filter (P-EMG plus Filter Box, Oisaka Electronic Equipment, Hiroshima, Japan) and digitized using an A/D converter (PowerLab 16/35; ADInstruments, Australia).

Chest movements were measured using a RIP system (Respitrace, AMI, USA). Participants were evaluated in the supine position, with two sensor bands (Respibands, AMI, USA) wrapped around the chest and abdomen, one at the level of the xiphoid process and the other directly above the navel. Raw RIP signals were digitized using the aforementioned A/D

Table 1. Clinical characteristics of the participants

		Case 1	Case 2	Case 3
Age (years)		50s	60s	50s
Gender		Male	Male	Male
Height (cm)		161	162	168
Weight (kg)		70	68	57
BMI (kg/m^2)		27.0	25.9	20.2
Neurological damage level	Motor	C4	C4	C2
	Sensory	C4	C6	C4
AIS		D	D	D
ASIA Motor score	Upper limb	36	44	26
	Lower limb	32	46	50
Vital capacity (%)		100.6	88.7	82.6
Time after injury (months)		21	58	48

BMI: body mass index; AIS: American Spinal Injury Association Impairment Scale; ASIA Motor score: American Spinal Injury Association Motor score.

converter. Inspiratory volume was measured by a spirometry test (pneumotachograph for volume: V_{PN}) at the same time as RIP. The participants wore a facemask attached to a respiratory flow head (MLT300L, ADInstruments, Australia), and their breathing patterns were amplified and recorded by the system (ML141 Spirometer MK2, ADInstruments, Australia). EMG and flow rate signals were synchronized at a sampling frequency of 1 kHz using software for real-time data analysis (LabChart 8.0, ADInstruments, Australia). Chest movements were recorded simultaneously with inspiratory intercostal s-EMG and RIP. After setting up the s-EMG and RIP equipment, a researcher asked the participant to breathe normally for 30 s, and then verbally instructed them to take a maximum of three inhalations ("Please breathe deeply to fully expand your chest.") at an interval of 5–10 s, exhaling normally after each cycle.

Parasternal intercostal muscle thickness was measured using static images taken after a maximum inspiration and resting expiration, on the monitor of the diagnostic ultrasound system. These were analyzed using the method applied when measuring the thickness of the parasternal intercostal muscles in healthy individuals[7], in which parasternal intercostal muscle thickness was measured perpendicular to the axis of contraction, as the distance between the lower edge of the deep fascia and the upper edge of the superficial fascia (precision: 0.1 mm) in the center of the area where the fibers of the deep and superficial fascia were observed to run parallel in the images. Intercostal muscle thickness changes (resting expiration and maximum inspiration) were calculated and compared with IC for each case to check the relationship. s-EMG data were analyzed in terms of the mean amplitude of the raw signals following RMS processing, i.e., of the waveforms of a resting exhalation and one of the three inhalations associated with the greatest IC. The change from rest to maximum inspiration was calculated (RMS amplitude at rest −RMS amplitude at maximum inhalation) and compared with IC to check for an association.

$$V_{RIP\text{-}RC}[\text{Liters}] = V_{RIP}(\text{IC}) \times \frac{RIP_{\text{-rib cage}}}{RIP_{\text{-rib cage}} + RIP_{\text{-abdomen}}}$$

As mentioned above, the IC was recorded using a pneumotachograph. Next, the RIP waveforms for the chest and abdomen were obtained using the Respitrace system from rest to maximum inspiration (mV). Then, the RIP waveforms of the chest and abdomen from rest to the maximum inspiration (mV) were converted into the inspiratory volume (L) measured by a spirometer. This new value was termed as V_{RIP}. The contribution of the chest to inspiratory volume was calculated by multiplying $V_{RIP\text{-}RC}$ with the ratio of the amplitude of the thoracic waveform to the combined amplitude of the thoracic and abdominal waveforms ($RIP_{\text{-rib cage}}$ and $RIP_{\text{-abdmen}}$, respectively). The calculation formula is as follows.

RESULTS

In all cases, inspiratory intercostal muscle activation (defined in terms of s-EMG RMS amplitude) and inspiratory intercostal muscle thickness was not notably different at maximum inspiration vs. at rest (Tables 2, 3).

Regarding the relationship between $V_{RIP\text{-}RC}$ and IC, $V_{RIP\text{-}RC}$ tended to be higher in patients with greater IC (Table 4).

DISCUSSION

High $V_{RIP\text{-}RC}$ (Inspiratory volume of the rib cage calculated by RIP) was associated with high IC among our patients. These results may indicate that inspiratory intercostal muscle activity is high in cases with high $V_{RIP\text{-}RC}$. The ability of RIP to record inspiratory intercostal muscle activity was validated in an animal model of CSI, with the authors reporting a positive associa-

Table 2. Individual changes in inspiratory intercostal EMG activity (RMS amplitude, s-EMG) from rest to inspiration

	Case 1	Case 2	Case 3
Rest (mV)	0.015	0.009	0.008
Maximum inspiration (mV)	0.015	0.009	0.009
Change (mV)	0.000	0.000	0.001

EMG: electromyography; RMS: Root Mean Square.

Table 3. Individual changes in inspiratory intercostal muscle thickness (ultrasound) from resting expiration to inspiration

	Case 1	Case 2	Case 3
Resting expiration (mm)	2.2	4.6	3.2
Maximum inspiration (mm)	2.2	4.6	3.0
Change (mm)	0.0	0.0	−0.2

Table 4. Relationship between individual $V_{RIP\text{-}RC}$ and IC (RIP)

	Case 1	Case 2	Case 3
$V_{RIP\text{-}RC}$ (L)	2.078	1.396	0.985
IC (L)	2.174	1.535	1.002

RIP: Respiratory Inductance plethysmograph; $V_{RIP\text{-}RC}$: Volume Respiratory Inductance Plethysmograph rib cage; IC: Inspiratory capacity.

tion between rib cage displacement and s-EMG mean amplitude[9]. The external and parasternal intercostal muscles are both inspiratory groups and act as the primary agonists of chest expansion during deep breathing[12]. It makes sense, therefore, that inspiratory intercostal muscle function would be better preserved in patients with CSI who have high $V_{RIP\text{-}RC}$, as we observed in this study. IC depends on the degree of residual descending motor pathway after CSI that projects to inspiratory intercostal muscle, with higher values reflecting greater preservation. We believe that this was the reason our cases with higher $V_{RIP\text{-}RC}$ also tended to have higher IC. However, the involvement of the diaphragm in chest motion cannot be ignored. Preceding research on breathing-related chest motion has reported that the upper thorax does not expand during inspiration in patients with complete CSI and intact diaphragm function[13–15]. Mortola et al.[8] separately investigated the motions of the rib cage and abdomen in healthy adults and tetraplegics (C5–C7) with intact diaphragm function. Their displacement profiles showed that chest diameter—as measured at the 3rd and 7th rib increased during inspiration in the former group, but not in the latter, due to the contraction of the diaphragm. This suggests that inhalation does not expand the chest if the inspiratory intercostal muscles are non-functional, as is the case in CSI. The normal function of the inspiratory intercostal muscle—an important inspiratory muscle group apart from the diaphragm, and a major agonist of rib cage expansion—needs to be restored in order for the chest to expand normally during breathing in patients with CSI. We believe that our use of $V_{RIP\text{-}RC}$ as an index of chest motion accurately captures the contribution of the inspiratory intercostal muscle to inhalation.

Our s-EMG assessment failed to reveal any obvious differences in inspiratory intercostal muscle activity between rest and maximum inspiratory effort. While s-EMG has been used to assess the same group in tetraplegics in previous work, its true usefulness requires further validation as it was unable to detect any myoelectric changes in some of the individuals tested[4]. We believe that the inspiratory intercostal muscles were activated, as evident from our data, which showed that chest expansion did occur. However, this activity was probably not detected because of the small magnitude of the EMG signal. Finally, the EMG signal may simply be incomplete since the EMG sensors were only placed at one site (over the 2nd parasternal intercostal muscle). This design was chosen with the intention of eliminating the effects of cross-talk[16]. However, this meant sacrificing insight into the external intercostal muscle, another inspiratory group widely distributed in the lateral and posterior rib cage.

Our ultrasound assessment revealed that inspiratory intercostal muscle thickness during maximum inspiratory effort was not different from that at rest, in individuals with CSI. In a pilot study conducted before, we confirmed that this group becomes significantly thicker at maximum inspiration than at rest in healthy adults[7]. However, similar assessments had not been performed for patients with CSI, so it was unclear whether inspiratory effort produces visible signs of contraction in individuals with compromised inspiratory intercostal muscle function. We did not observe changes in inspiratory intercostal muscle thickness during inhalation in our patients. However, possibly, our method was unable to detect the changes that

occurred; in normal adults, we recorded approximately 0.7 mm increase in thickness at maximum inspiration, so the corresponding changes in tetraplegics would be even less. Our apparent failure in detection might be partially attributable to the fact that measurements were only taken at one location.

In conclusion, this study aimed to determine if IC was associated with intercostal EMG activity, thickness changes, or $V_{RIP\text{-}RC}$ in three individuals with CSI. We hypothesized that patients with lower IC would have lower RMS amplitudes on s-EMG, smaller contraction-related changes in muscle thickness, and lower $V_{RIP\text{-}RC}$ on RIP. However, we did not observe any obvious differences in inspiratory intercostal muscle activity between resting state and at the maximum inspiratory effort, on s-EMG profiles. This negative finding could possibly be a consequence of mild potential changes during maximum inspiration in tetraplegics and our selection of only a single electrode site. Our ultrasound assessment revealed that inspiratory intercostal muscle thickness during maximum inspiratory effort was similar to that at rest in individuals with CSI. This negative finding was probably a consequence of the very slight thickness changes during maximum inspiration in tetraplegics and our choice of only a single measurement site. However, cases with higher $V_{RIP\text{-}RC}$ also tended to have higher IC. Higher $V_{RIP\text{-}RC}$ is indicative of better inspiratory intercostal muscle function; it makes sense that IC—which reflects residual descending input to this group—would be higher in such cases. Our findings suggest that $V_{RIP\text{-}RC}$ can capture inspiratory intercostal muscle function in CSI patients, making it a useful indicator for assessing inspiratory intercostal muscle in CSI patients. On the other hand, the number of subjects in this study was small, and therefore, the number of cases studied was insufficient. In our future studies, we plan to study a larger cohort of patients and examine the usefulness of $V_{RIP\text{-}RC}$.

In this study, in order to establish the evaluation of inspiratory intercostal muscle activity in patients with CSI, we evaluated inspiratory intercostal muscle activity in patients with CSI using inspiratory intercostal electromyography, RIP, and ultrasound. We conclude that $V_{RIP\text{-}RC}$ was high in the cases with high IC. These results may indicate that inspiratory intercostal muscle activity is high in cases with high $V_{RIP\text{-}RC}$ and that RIP can indicate inspiratory intercostal muscle function in patients with CSI.

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