



# Effects of decompressive operation on cardiac autonomic regulation in patients with cervical spondylotic myelopathy: analysis of blood pressure, heart rate, and heart rate variability

Peng Li<sup>1</sup> · Zihan Wei<sup>2</sup> · Haoping Zhang<sup>1</sup> · Kainan Zhang<sup>1</sup> · Junwei Li<sup>1</sup>

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## Abstract

**Purpose** To investigate the effects of cervical decompression operation on cardiac autonomic regulation and its relationship to recovery of somatic neurological function in cervical spondylotic myelopathy (CSM) patients.

**Methods** One hundred and thirty-two consecutive patients were enrolled in this study, in which 73 patients received decompression operation and the remaining 59 were treated non-operatively. The follow-up period was 6 months. Baseline and follow-up evaluation included Japanese Orthopaedic Association (JOA) score, office-based blood pressure (BP) measurement, heart rate (HR), and 24-h heart rate variability (HRV) assessment. Relationship between achieved JOA score (final JOA score–baseline score) and changes of BP, HR, and HRV parameters in both operative and non-operative groups was analyzed.

**Results** In operative group, patients' JOA score and markers of parasympathetic activity in HRV assessment were significantly higher than baseline level 6 months later. Blood pressure, especially systolic blood pressure (SBP), was significantly downregulated in both hypertension and non-hypertension patients. Mean heart rate was also significantly decreased. Furthermore, achieved JOA score was significantly negatively correlated with changes of SBP, minimal HR, mean HR, maximum HR, but significantly positively correlated with changes of HRV parameters reflecting parasympathetic activity. However, changes of JOA score, BP, HR, and HRV parameters in non-operative group were not significant.

**Conclusions** Cervical decompression operation could improve both somatic neurological function and cardiac autonomic regulation in CSM patients, and achieved JOA score was significantly positively correlated with improvement in HRV and cardiac parasympathetic activity.

## Graphical abstract

These slides can be retrieved under Electronic Supplementary Material.

**Key points**

1. Cervical decompression operation
2. Cervical spondylotic myelopathy
3. Cardiac autonomic regulation
4. Heart rate variability
5. Blood pressure
6. Heart rate

**Fig. 1**

**Fig. 2**

**Take Home Messages**

1. Cervical decompression operation improved both somatic neurological function and cardiac autonomic regulation.
2. Enhanced cardiac parasympathetic activity were found after operative treatment, which was characterized as down regulated blood pressure and heart rate and up regulated heart rate variability.
3. Achieved JOA score was significantly correlated with changes of cardiac autonomic parameters.

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Extended author information available on the last page of the article

**Keywords** Cervical decompression operation · Cervical spondylotic myelopathy · Cardiac autonomic regulation · Heart rate variability · Blood pressure · Heart rate

## Introduction

Cervical spondylotic myelopathy (CSM) defined as age-related degeneration of tissues of cervical spinal column and results in compression of spinal cord, which impairs both somatic and autonomic nervous system [1]. Somatic nervous dysfunction such as motor and sensory deficits and the recovery process after decompression surgery were massive documented [2], while reports on recovery of autonomic nervous dysfunction in CSM patients were still limited.

Cardiac autonomic nervous system was involved in regulation of heart rate and blood pressure. Recent studies revealed that cervical decompression surgery improved both systolic (SBP) and diastolic blood pressure (DBP) in patients concomitant hypertension [3], which indicated that compression of cervical spinal cord could be associated with hypertension, and decompression surgery could improve this type of hypertension. While not every CSM patient's blood pressure was higher than normal, how is the change of blood pressure in CSM patients without hypertension? Furthermore, how is the relationship between recovery of somatic neurological deficits and cardiac autonomic regulation after decompression operation? To answer these questions, we designed this prospective cohort study by investigating the changes of neurological function score, blood pressure, heart rate, and heart rate variability in CSM patients under different treatment method.

Heart rate variability (HRV) measurement is one of the best noninvasive techniques in evaluation of the cardiac autonomic function by quantifying beat-to-beat changes of heart rate [4]. While HRV parameters could be affected by various factors [5–7], including age, gender, some metabolic indexes, body mass index, drugs, and some disease such as diabetes, coronary artery disease, which indicated that patients involved in HRV analysis should be strictly controlled. In this study, these factors were considered in eligibility evaluation, detailed data and history information of each patient was recorded and difference between groups was compared separately.

HRV could be measured in both time and frequency domain [8]. HRV could be determined by software from 24-h dynamic electrocardiogram recording. The time domain of HRV was measured as beat-to-beat intervals or as variables derived from differences in adjacent normal-to-normal (NN) intervals. Frequency domain of HRV was measured with power spectral analysis (PAS). PSA provided information about heart rate distribution as function of frequency. Frequency domain analysis could be classified as short-term and long-term recordings, 24-h long-term recording was used in

this study. Analysis of changes of parameters in both time and frequency domain could discriminate between the sympathetic and parasympathetic contributions of HRV. According to previous study [9], SDNN was used for general assessment of HRV, HF value along with RMSSD and pNN50 were considered as markers of parasympathetic activity, and LF reflected sympathetic (and parasympathetic) activity.

## Materials and methods

### Study design

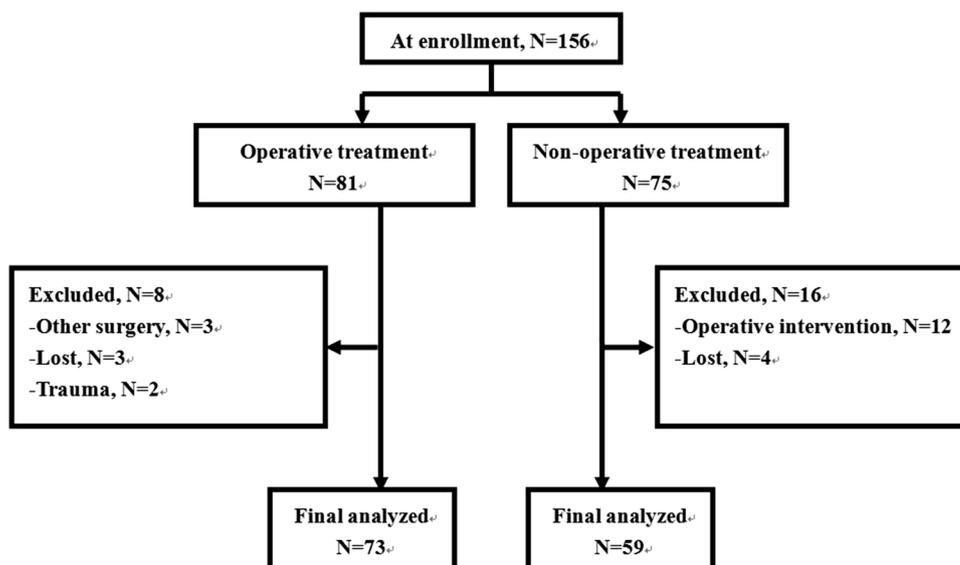
The ethics committee of our university approved this prospective observational cohort study. After confirmation of eligibility, consecutive patients with CSM, who received cervical decompression operation or non-operative treatment between June 2014 and September 2017, were included in this study, and the follow-up term for each patient was 6 months. All patients received diagnosis of CSM secondary to disk herniation, facet degeneration, and calcification or hypertrophy of ligamentum flavum and posterior longitudinal ligament. Before assessment, all patients should stop medication like antihypertensive drugs, drinking alcohol, and coffee for at least 24 h. Baseline evaluation included history taking, physical examination, body mass index (BMI), blood examination of plasma glucose, total cholesterol and triglyceride, office-based blood pressure measurement, scoring system for cervical myelopathy by the Japanese Orthopedic Association (JOA) [10], and 24-h heart rate variability recording based on dynamic electrocardiogram. Patients with hypertension were required not to change their medication during this study.

### Study population

The inclusion criteria were as follows: CSM patients with different degrees of myelopathy symptom; age between 40 and 70 years; spinal cord compression on radiological image; willing to participate in this study; and signed informed consent and 0–10 visual pain scores  $\leq 3$  points. Patients with a history of any of the following were excluded: secondary hypertension; prior cervical spine surgery; pain scores  $> 3$  points; tumor; trauma; obesity with body mass index (BMI)  $\geq 28$ ; concomitant diabetes; coronary heart disease; cardiac dysrhythmia needing medication; and chronic pulmonary, kidney and liver disease.

Altogether 156 patients were initially enrolled (Fig. 1), in which 81 received cervical decompression surgery and 75

**Fig. 1** Flowchart illustrating patients recruitment and group information



were treated with non-operative method. During the follow-up, 5 patients were excluded from the operative group due to other cervical surgery (3) and trauma (2), and 12 patients were excluded from the non-operative group due to transmit to operative intervention. Another 7 patients (operation 3; non-operation 4) were lost to follow-up. As a result, a total of 132 patients (operation 73; non-operation 59) with detailed data were included in the final analysis.

## Treatment

Treatment selection was decided by senior author's careful assessment of the patients and also with the agreement of the patients. Patients with mild CSM who refused surgical intervention could be treated non-operatively under strict supervision, if the neurological dysfunction deteriorated progressively, operative intervention would be recommended. Non-operative treatment included gradual rehabilitation training, physical therapy, intermittent cervical immobilization using soft collar, and medication such as nonsteroidal anti-inflammatory drugs (NSAIDs). Cervical decompression surgery could be done with the following techniques: anterior cervical discectomy and fusion; anterior cervical corpectomy and fusion; posterior one-side open-door decompression.

## Data collection

All data were collected and analyzed by trained staff using a blind method, which means these staff had no grouping and treatment information of each patient. After baseline evaluation, patients were treated operatively or non-operatively, and the follow-up assessment, including office-based blood pressure measurement, JOA score evaluation

and 24-h heart rate variability analysis, was performed 6 months after treatment.

Office-based blood pressure of each patient was measured in the morning between 8 and 10 am, and each measurement would be repeated twice using electronic blood pressure monitor (HEM-1000, Omron, Japan) after patients remained at rest in seated position for at least 10 min in quiet room with the room temperature of 24 °C; the mean of these two values was recorded. If the difference between these two measurements was bigger than 10 mmHg, one more measurement would be required.

Dynamic electrocardiogram was attached to each patient, and continuous heart rate data were recorded for 24 h, and patients were required to stay in similar environment under similar conditions with the baseline recording. Heart rate variability of each patient was analyzed using dynamic electrocardiogram analyzer (MarS, GE, USA). Both time domain and frequency domain were used in this study. Parameters used in time domain analysis in this study included: standard deviation of all NN intervals (SDNN); standard deviation of the averages of NN intervals in all 5-min segments of the entire recording (SDANN); the square root of the mean of the sum of the squares of differences between adjacent NN intervals (RMSSD); and NN50 count divided by the number of all NN intervals (pNN50). The frequency domain parameters included: variance of all NN intervals (total power); power in the ultra-low frequency range ( $\leq 0.003$  Hz, ULF); power in the very low frequency range (0.003–0.04 Hz, VLF); power in the low frequency (0.04–0.15 Hz, LF); and power in high frequency (0.15–0.4 Hz, HF).

Changes in somatic neurological function were evaluated with the achieved JOA score, which was calculated as final JOA score–baseline score.

## Statistical analysis

Data of frequency domain were presented as mean  $\pm$  standard error, and all the other data were presented as mean  $\pm$  standard deviation. Paired t test was used to determine significant changes between two groups; nonparametric Wilcoxon signed-rank test was used when required condition was not satisfied. Spearman correlation coefficients were used to analyze the relationships between changes in JOA scores (final JOA score–baseline score) and changes in blood pressure (final value–baseline value), heart rate (final value–baseline value), heart rate variability time domain (final value–baseline value), and frequency domain (final value/baseline value). All statistical analyses were performed using SPSS 16.0 (SPSS Inc, USA), and  $p < 0.05$  was considered significant.

## Results

Baseline of age, gender, BMI, blood glucose, total cholesterol, and triglyceride were comparable between non-operative group and operative group (Table 1). The number of patients with concomitant hypertension was 24 (40.68%)

in non-operative group and 31 (42.47%) in operative group, and the baseline blood pressure and antihypertensive medication plan between groups were also comparable. The JOA score in operative group was significantly lower than non-operative group. After 6 months, achieved JOA score of operative group was significantly higher than that of non-operative group. In operative group, achieved JOA score of patients without hypertension was little greater than that of patient with hypertension, but not significant.

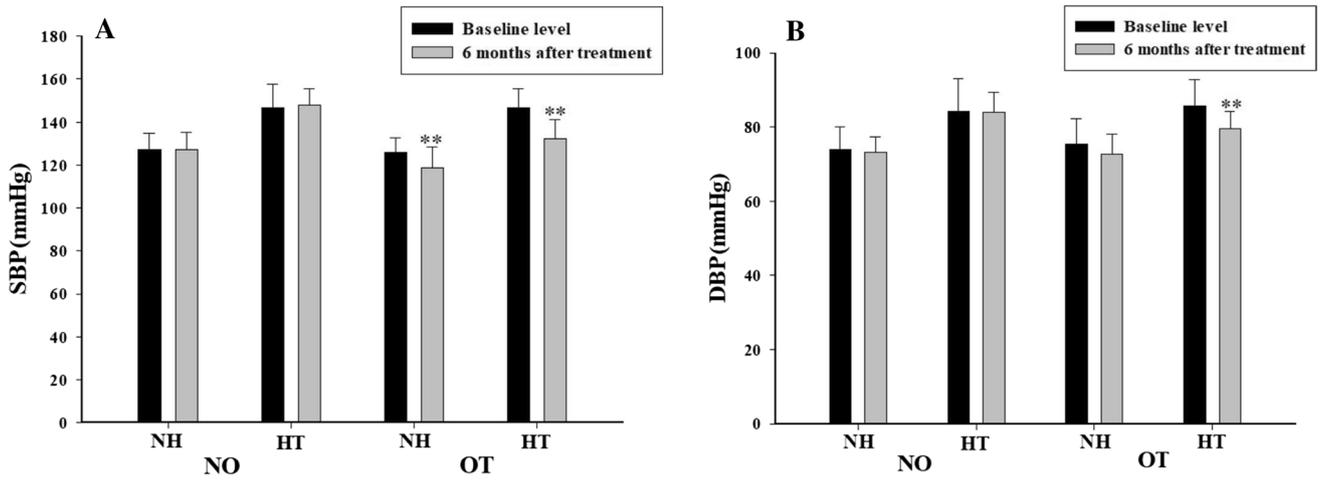
In operative group (Fig. 2), both systolic pressure (SBP) and diastolic blood pressure (DBP) were significantly downregulated 6 months after decompression surgery in hypertension patients from  $146.50 \pm 9.03$  to  $132.39 \pm 8.86$  mmHg and  $85.65 \pm 7.13$  to  $79.61 \pm 4.72$  mmHg, respectively. In non-hypertension patients, SBP was also significantly downregulated after surgery from  $125.80 \pm 7.05$  to  $118.90 \pm 9.47$  mmHg, but change of DBP was not significant. Furthermore, SBP of hypertension patients decreased by  $14.10 \pm 9.97$  mmHg, which was significantly ( $p = 0.01$ ) higher than that of non-hypertension patients ( $6.88 \pm 10.97$  mmHg). Changes of systolic pressure (SBP) and diastolic blood pressure (DBP) were not significant in patients treated non-operatively.

**Table 1** Demographic data and changes of JOA score of all the patients

	Non-operative treatment		Operative treatment	
	Non-hypertension	Hypertension	Non-hypertension	Hypertension
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Subjects ( <i>n</i> )	35	24	42	31
Age (years)	56.23 (7.60)	55.13 (7.71)	55.76 (8.66)	57.39 (8.76)
Male ( <i>n</i> )	20	15	23	20
BMI (Kg/m <sup>2</sup> )	23.29 (2.14)	23.24 (2.19)	23.34 (2.39)	23.37 (2.49)
Blood glucose (mmol/L)	5.08 (0.71)	5.07 (0.71)	4.97 (0.67)	5.04 (0.79)
Total cholesterol (mmol/L)	4.17 (1.15)	4.21 (0.80)	4.25 (0.80)	4.22 (0.83)
Triglyceride (mmol/L)	1.22 (0.41)	1.25 (0.41)	1.22 (0.37)	1.19 (0.38)
Systolic blood pressure (mmHg)	127.27 (7.44)	146.79 (10.72)	125.80 (7.05)	146.50 (9.03)
Diastolic blood pressure (mmHg)	73.89 (6.13)	84.21 (8.76)	75.52 (6.77)	85.65 (7.13)
Number of different antihypertensive drugs ( <i>n</i> )	–	1.42 (0.50)	–	1.32 (0.48)
<i>Antihypertensive drugs</i>				
ARB ( <i>n</i> )	–	7	–	8
ACEI ( <i>n</i> )	–	8	–	10
Diuretic ( <i>n</i> )	–	8	–	9
CCB ( <i>n</i> )	–	8	–	10
BB ( <i>n</i> )	–	3	–	4
Baseline JOA score, points	15.60 (0.50)	15.54 (0.51)	11.71 (1.47) n	11.48 (1.41) n
JOA score 6 months after treatment	15.80 (0.58)	15.67 (0.70)	14.00 (1.58) b	13.52 (1.24) b
Achieved JOA score, points	0.20 (0.46)	0.13 (0.51)	2.29 (0.86) n	2.03 (0.84) n

n:  $p < 0.001$  compared with non-operative group; b:  $p < 0.001$  compared with baseline data

*BMI* Body mass index, *ARB* angiotensin receptor blocker, *ACEI* angiotensin-converting enzyme inhibitor, *CCB* calcium receptor blocker, *BB* beta-receptor blocker

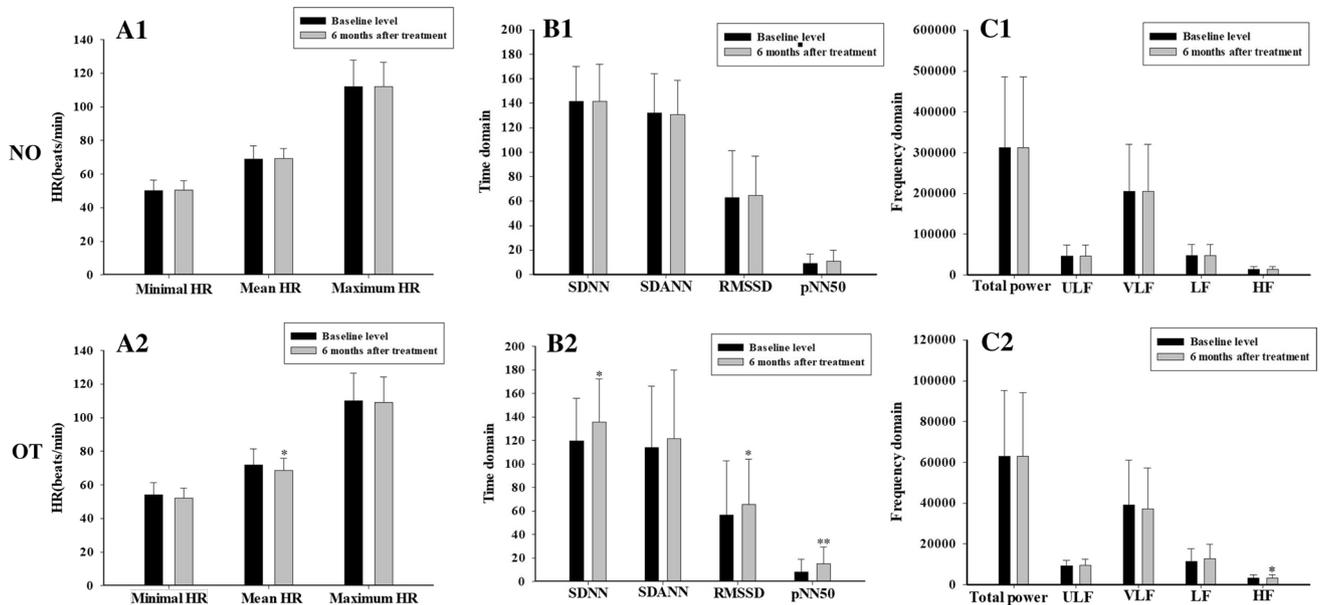


**Fig. 2** Changes of blood pressure after operative (OT) or non-operative (NO) treatment for CSM patients. **a** For systolic blood pressure (SBP) and **b** for diastolic blood pressure (DBP). *NH* Non-hypertension, *HT* hypertension

In operative group, mean heart rate (HR) ( $68.75 \pm 6.99$ ) was significantly ( $p = 0.029$ ) lower than that of baseline ( $72.07 \pm 9.31$ ) 6 months later (Fig. 3), while changes of minimal and maximum heart rate were not significant. In heart rate variability (HRV) analysis, SDNN ( $p = 0.008$ ), RMSSD ( $p = 0.009$ ), PNN50 ( $p < 0.001$ ), and HF ( $p = 0.002$ ) were significantly higher than those baseline levels 6 months after operative treatment. Changes of heart rate and heart rate variability in non-operative group were not significant.

changes in SBP ( $p = 0.002$ ), minimal HR ( $p = 0.005$ ), mean HR ( $p = 0.001$ ), maximum HR ( $p = 0.001$ ), and ULF in frequency domain of HRV ( $p = 0.013$ ). A significant positive correlation was found between achieved JOA score and changes in SDNN ( $p < 0.001$ ), SDANN ( $p < 0.001$ ), RMSSD ( $p = 0.006$ ), PNN50 ( $p = 0.001$ ), and HF ( $p = 0.005$ ). In non-operative group, correlation between achieved JOA score and changes in parameters of blood pressure, heart rate, and heart rate variability was not significant.

In operative group (Table 2), a significant negative correlation was found between achieved JOA score and



**Fig. 3** Changes of heart rate (**a**) and HRV parameters (time domain: **b**; frequency domain: **c**) after operative (OT) or non-operative (NO) treatment for CSM patients

**Table 2** Correlations between achieved JOA score and changes of blood pressure, heart rate and heart rate variability in non-operative and operative group

	Non-operative treatment	Operative treatment
<i>Blood pressure</i>		
Systolic blood pressure	0.025	– 0.364*
Diastolic blood pressure	0.044	– 0.139
<i>Heart rate</i>		
Minimal heart rate	0.125	– 0.367*
Mean heart rate	0.163	– 0.305**
Maximum heart rate	0.159	– 0.392**
<i>Heart rate variability</i>		
SDNN	– 0.087	0.623**
SDANN	– 0.030	0.391**
RMSSD	– 0.220	0.337*
PNN50	0.012	0.395**
Total power	0.092	– 0.080
ULF	0.172	– 0.275*
VLF	0.029	– 0.111
LF	– 0.017	0.095
HF	0.048	0.362*

Spearman's rho

\* Correlation is significant and  $p < 0.05$ ; \*\* Correlation is significant and  $p \leq 0.001$ 

## Discussion

In the present study, we demonstrated that decompression operation not only improved the somatic neurological function in CSM patients, but also improved the cardiac autonomic regulation by enhancing parasympathetic activity, which was characterized as downregulated blood pressure in both hypertension patients and non-hypertension patients, decreased mean heart rate, and upregulated heart rate variability, while changes of JOA score and cardiac autonomic regulation in patients treated non-operatively were not significant.

Autonomic dysfunction was well reported in tetraplegia patients with spinal cord injury [11], and clinically significant autonomic disorder in patients with CSM was mainly reported in form of bladder dysfunction [12], while studies reporting changes of cardiac autonomic regulation in CSM were very few. Cardiac autonomic regulation was dependent on the balance between sympathetic and parasympathetic nervous system. In patients with severe or complete cervical spinal cord injury, supraspinal control of sympathetic outflow was damaged, but the parasympathetic outflow was preserved, which resulted in cardiac dysrhythmias and disorder of blood pressure regulation [13]. In CSM patients, due to injury to spinal cord being less severe, both tonic inhibitory effect from intraspinal noradrenaline fibers

and reflex sympathetic control of arterial pressure would become abnormal, but not fully disrupted, and tended to show higher levels of blood pressure and heart rate. Srihari et al. [14] reported cardiac autonomic dysfunction in patients with cervical compressive myelopathy was limited, which indicated that degree of cardiac autonomic dysfunction correlated with the severity of somatic neurological deficit. In the present study, we found that HRV of patients was significantly upregulated with the higher level of SDNN after decompression operation, and predominant levels of RMSSD, PNN50, and HF, which reflected higher level of parasympathetic activity compared with baseline situation. Furthermore, improvement in somatic neurological function was significantly positive correlated with changes of markers of parasympathetic activity, while changes of marker of sympathetic activity were not significant, which indicated that regulation of sympathetic activity could be complicated in CSM patients after decompression operation.

Heart rate reflected interaction between parasympathetic and sympathetic nervous systems. Bradycardia was commonly reported in acute stage of cervical spinal injury [15]. In patients with severe spinal cord injury, bradycardia could be a severe problem which requires vasopressor therapy to maintain blood pressure [16], which was because of the increase in heart rate which was blunted due to sympathetic decentralization [17]. In contrast, patients with less severe spinal cord injury tended to show higher levels of heart rate, but there are few reports of heart rate regulation in CSM patients. Li et al. [18] reported that decompression operation could significantly decrease heart rate in CSM patients with hypertension, while this effect was not significant in non-hypertension patients. Considering that sympathetic activity could be more correlated with patients' overall active condition [19], in this study, postoperative heart rate parameters were evaluated under similar conditions with baseline recording. We found that mean heart rate of patients was slightly decreased after operative treatment, which was correlated with the increased level of cardiac parasympathetic activity after operation. Furthermore, achieved JOA score was negatively correlated with changes of heart rate parameters, which revealed that enhanced parasympathetic activity could play a positive role in rehabilitation period.

The concrete mechanism of primary hypertension was still unknown, and several risk factors of high blood pressure were reported including stress and some metabolic indexes [20]. In patients with CSM, damage to spinal cord was not so severe, and blood pressure regulation in these patients would still partially depend on sympathetic efferent nerves [21], while disruption of the descending pathways would lead to plastic changes within spinal cord [22] and peripheral neurovascular responsiveness [23] and finally cause blood pressure disorders. Previous study reported that the prevalence of hypertension in CSM patients was higher than that

of whole population [18], and cervical decompression surgery could significantly reduce concomitant high blood pressure [3], while no study was found to report the changes of blood pressure in CSM patients without hypertension after surgery. In this study, we found that decompression operation decreases both systolic pressure and diastolic blood pressure in CSM patients with concomitant hypertension, while in patients without hypertension, systolic blood pressure was also decreased after surgery, but change of diastolic blood pressure was not significant. Achieved JOA score was negatively correlated with change of systolic blood pressure, but positively correlated with changes of parasympathetic parameters in HRV measurement, which revealed that parasympathetic nervous system, not sympathetic nervous system, played an important role in blood pressure regulation in CSM patients during rehabilitation period.

In our present study, baseline JOA score of non-operative group was higher than that of operative group, and although we revealed that operative treatment could improve both somatic neurological function and cardiac autonomic regulation, it would be improper to conclude that operative treatment was superior to non-operative treatment in CSM patient. Another limitation of this study was that we cannot eliminate the effect of environment on HRV parameters, and patients were required to stay in similar condition during HRV measurement, which was clinically feasible and could minimize the affection of environment as much as possible. According to our clinical practice, the JOA score of most CSM patients after surgery could reach peak in 6 months, and therefore, we selected the time point of 6 months in this study, although it was relatively short period. HRV was reported as a strong and independent predictor of mortality after acute myocardial infarction [24], and heart rate was also reported to have significant negative effect on cardio- and cerebrovascular morbidity and mortality [25]. In this study, we demonstrated improved HRV and reduced mean heart rate in CSM patients after operative treatment; however, whether CSM promotes risk of cardiovascular accident in coronary heart disease patients still needs further investigation.

## Conclusions

We found that decompression operation could significantly improve both somatic neurological function and cardiac autonomic regulation by enhancing parasympathetic activity in CSM patients. Achieved JOA score was significantly correlated with changes of cardiac autonomic parameters, while changes of sympathetic parameters were not significant. Although changes of cardiac autonomic regulation described in this study were subclinical, the results could shed light on the recognition of recovery of autonomic nervous system

after operation in CSM patients and would be helpful for further investigation of cardiovascular risk in CSM patients.

## Compliance with ethical standards

**Conflict of interest** The authors declare that we have no conflict of interest.

## References

1. Brain WR, Northfield D, Wilkinson M (1952) The neurological manifestations of cervical spondylosis. *Brain* 75(2):187–225
2. Suzuki A, Misawa H, Simogata M, Tsutsumimoto T, Takaoka K, Nakamura H (2009) Recovery process following cervical laminoplasty in patients with cervical compression myelopathy: prospective cohort study. *Spine* 34(26):2874–2979
3. Yang L, Yang C, Pang X, Li D, Chen X, Shi J, Zhou X, Wu Y, Lu X, Yang H, Jia L, Peng B (2017) Cervical decompression surgery for cervical spondylotic myelopathy and concomitant hypertension: a multicenter prospective cohort study. *Spine* 42(12):903–908
4. Akselrod S, Gordon D, Ubel FA, Shannon DC, Berger AC, Cohen RJ (1981) Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. *Science* 213(4504):220–222
5. Vinik AI, Maser RE, Ziegler D (2011) Autonomic imbalance: prophet of doom or scope for hope? *Diabet Med* 28(6):643–651
6. Hillebrand S, Swenne CA, Gast KB, Maan AC, Cessie SL, Jukema JW, Rosendaal FR, Heijer Md, Mutsert Rd (2015) The role of insulin resistance in the association between body fat and autonomic function. *Nutr Metab Cardiovasc Dis* 25(1):93–99
7. Mølgaard H, Mickley H, Pless P, Bjerregaard P, Møller M (1993) Effects of metoprolol on heart rate variability in survivors of acute myocardial infarction. *Am J Cardiol* 71(15):1357–1359
8. Camm AJ, Malik M, Bigger JT, Breithardt G, Cerutti S, Cohen RJ, Coumel P, Fallen EL, Kennedy HL, Kleiger RE, Lombardi F (1996) Heart rate variability: standards of measurement, physiological interpretation and clinical use Task force of the European Society of Cardiology and the North American Society of pacing and electrophysiology. *Circulation* 93(5):1043–1065
9. Lakusic N, Mahovic D, Kruzliak P, Habek JC, Novak M, Cerovec D (2015) Changes in heart rate variability after coronary artery bypass grafting and clinical importance of these findings. *Biomed Res Int* 2015:680515
10. Yamauchi H, Hirabayashi K (1994) Scoring system (17-2) for cervical myelopathy (Japanese Orthopaedic Association). *J Jpn Orthop Assoc* 68:490–503
11. Thayer JF, Sollers JJ III, Clamor A, Koenig J, Hagglund KJ (2016) The association of resting state heart rate variability and 24-hour blood pressure variability in spinal cord injury. *J Neurol Sci* 361:52–59
12. Rajshekhhar V, Kumar GS (2005) Functional outcome after central corpectomy in poor-grade patients with cervical spondylotic myelopathy or ossified posterior longitudinal ligament. *Neurosurgery* 56(6):1279–1284
13. Grigorean VT, Sandu AM, Popescu M, Iacobini MA, Stoian R, Neascu C, Strambu V, Popa F (2009) Cardiac dysfunctions following spinal cord injury. *J Med Life* 2(2):133–145
14. Srihari G, Shukla D, Devi BI, Sathyaprabha TN (2011) Subclinical autonomic nervous system dysfunction in compressive cervical myelopathy. *Spine* 36(8):654–659

15. Hadley MN, Walters BC, Grabb PA, Oyesiku NM, Przybylski GJ, Resnick DK, Ryken TC (2002) Blood pressure management after acute spinal cord injury. *Neurosurgery* 50(3 Suppl):S58–S62
16. Furlan JC, Fehlings MG, Shannon P, Norenberg MD, Krassioukov AV (2003) Descending vasomotor pathways in humans: correlation between axonal preservation and cardiovascular dysfunction after spinal cord injury. *J Neurotrauma* 20(12):1351–1363
17. Takahashi M, Matsukawa K, Nakamoto T, Tsuchimochi H, Sakaguchi A, Kawaguchi K, Onari K (1985) Control of heart rate variability by cardiac parasympathetic nerve activity during voluntary static exercise in humans with tetraplegia. *J Appl Physiol* 103(5):1669–1677
18. Li ZQ, Zhao YP, Jia WY, Wang X, Chen B, Shahbaz M, Nie L, Cheng L (2015) Surgical treatment of cervical spondylotic myelopathy associated hypertension—a retrospective study of 309 patients. *PLoS ONE* 10(7):e0133828
19. Furlan R, Piazza S, Dell’Orto S, Gentile E, Cerutti S, Pagani M, Malliani A (1993) Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. *Cardiovasc Res* 27(3):482–488
20. Christofaro DG, Fernandes RA, Oliveira AR, Júnior IFF, Barros MV, Ritti-Dias RM (2014) The association between cardiovascular risk factors and high blood pressure in adolescents: a school-based study. *Am J Hum Biol* 26(4):518–522
21. Mathias CJ (1991) Role of sympathetic efferent nerves in blood pressure regulation and in hypertension. *Hypertension* 18(5 Suppl):22–30
22. Teasell RW, Arnold JM, Krassioukov A, Delaney GA (2000) Cardiovascular consequences of loss of supraspinal control of the sympathetic nervous system after spinal cord injury. *Arch Phys Med Rehabil* 81(4):506–516
23. Yeoh M, McLachlan EM, Brock JA (2004) Tail arteries from chronically spinalized rats have potentiated responses to nerve stimulation in vitro. *J Physiol* 556(Pt 2):545–555
24. Malik M, Farrell T, Cripps T, Camm AJ (1989) Heart rate variability in relation to prognosis after myocardial infarction: selection of optimal processing techniques. *Eur Heart J* 10(12):1060–1074
25. Tadic M, Cuspidi C, Grassi G (2018) Heart rate as a predictor of cardiovascular risk. *Eur J Clin Invest* 48(3):e12892. <https://doi.org/10.1111/eci.12892>

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## Affiliations

Peng Li<sup>1</sup> · Zihan Wei<sup>2</sup> · Haoping Zhang<sup>1</sup> · Kainan Zhang<sup>1</sup> · Junwei Li<sup>1</sup> 

✉ Junwei Li  
lijunweizz@126.com

<sup>1</sup> Department of Orthopaedics, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450000, Henan Province, People’s Republic of China

<sup>2</sup> Department of Geriatric Cardiology, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450000, Henan Province, People’s Republic of China