



Original Article

## Immediate effects of rhythmic auditory stimulation on gait in stroke patients in relation to the lesion site

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**Abstract.** [Purpose] Rhythmic auditory stimulation has been used in gait training for stroke patients. However, few studies have investigated its effects in relation to lesion sites. Therefore, this study examined the immediate effects of rhythmic auditory stimulation on gait in stroke patients with lesions in different regions. [Subjects and Methods] One hundred and five patients were recruited and divided into five groups according to the lesion site: cerebellum, pons and medulla, thalamus, putamen, and corona radiata. During training, participants walked to an auditory, continuous rhythmic beat, which was set to each individual's cadence. [Results] Pre- versus post-test measures revealed significant increases in velocity and stride length in the cerebellum, pons and medulla, and thalamus groups. Although the putamen and corona radiata groups demonstrated increases in velocity and stride length, the increases were not significant. [Conclusion] Rhythmic auditory stimulation was effective in facilitating the prediction of motor timing and gait rhythm in stroke patients with lesions in the cerebellum, pons and medulla, and thalamus, which are associated with impairment of the timing mechanism.

**Key words:** Rhythmic auditory stimulation, Gait training, Stroke

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### INTRODUCTION

Gait training with auditory cues using metronome clicks or music is known to improve gait in Parkinson's disease<sup>1-4)</sup>, in which striatal dopamine decreases due to degeneration and depletion of dopamine neurons in the substantia nigra. Dopamine depletion causes cognitive disturbances, sleep difficulties, and various movement problems including gait disorders such as freezing of gait, festination, and decrease in velocity and stride length<sup>5)</sup>. To improve gait of patients with Parkinson's disease, external information, such as horizontal stripe design on the floor and metronome beats, helps elicit motor responses by bypassing the basal ganglia<sup>6)</sup>. Thaut et al. reported that patients with Parkinson's disease who had gait training with rhythmic auditory stimulation (RAS) had significantly improved gait velocity, stride length, step cadence and timing of EMG patterns in the anterior tibialis and vastus lateralis muscles compared to those without RAS<sup>1)</sup>. Hayashi reported that some gait improvement in Parkinson's disease can be achieved by passively listening to music without actual gait training<sup>3)</sup>. The disturbed internal rhythm formation could be corrected by external auditory rhythm, which helps achieve a stable gait rhythm.

Recently, RAS has been found to be highly effective in gait training for stroke patients and for those with Parkinson's disease<sup>7)</sup>. A systematic review of interventions for gait coordination following stroke revealed that auditory cueing is one of the most promising approaches in gait training<sup>7)</sup>. According to Thaut et al., in stroke patients, gait training with RAS significantly increased velocity and stride length and reduced electromyographic amplitude variability of the gastrocnemius muscle<sup>8)</sup>. In a study comparing RAS with neurodevelopmental therapy, Bobath based training, for stroke rehabilitation,

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**Table 1.** Participant characteristics

Lesion site	Subject, n	Gender, n		Age (years)	Time post-stroke, day	Stroke type, n	
		Male	Female			CH	CI
Cerebellum	20	15	5	71.3 ± 9.5	40.8 ± 30.6	12	8
Pons and medulla	26	21	5	67.4 ± 10.9	38.4 ± 22.8	3	23
Thalamus	22	18	4	64.0 ± 9.2	61.0 ± 33.0	18	4
Putamen	18	11	7	64.3 ± 13.4	42.7 ± 19.5	15	3
Corona radiata	19	12	7	72.8 ± 9.4	39.2 ± 23.2		19
Total/Mean	105	77	28	67.9 ± 11.0	44.37 ± 27.5	48	57

CH: cerebellar hemorrhage; CI: cerebellar infarction

greater improvement was seen in velocity, stride length, cadence, and symmetry with RAS than with the other approach<sup>9</sup>. These findings show that RAS is an effective intervention in gait training following stroke.

However, the effect of RAS on specific regions of the brain and its influence on motor output is not fully understood<sup>10</sup>. Few studies have investigated the effects of RAS in relation to the lesion site in stroke patients<sup>11</sup>. Therefore, the purpose of this study was to investigate the neurological effects of RAS on gait in stroke patients with lesion in different regions (i.e., the cerebellum, pons and medulla, thalamus, putamen, and corona radiata).

## SUBJECTS AND METHODS

Between April 2006 and June 2011, we included 105 inpatients from the post-acute rehabilitation unit. Participant characteristics are summarized in [Table 1](#). All subjects were classified stage 3 or above on the Brunnstrom recovery scale of the lower limb (i.e., lower limb spasticity and voluntary control through basic limb synergy)<sup>12</sup>).

All patients understood the purpose of this study and provided written informed consent prior to participation in the study in accordance with the ethical standards set by the Declaration of Helsinki.

Before RAS, each participant completed a 10-meter walk without RAS (pre-test), after which baseline data for gait cadence, velocity, and stride length were recorded. Then each participant walked to the auditory rhythmic cues at a frequency that matched his/her baseline cadence, as described by Thaut et al<sup>8, 9</sup>. The auditory cues were provided by a music therapist who either used metronome beats or played steady beats using a musical rhythm instrument such as a drum or an autoharp. When the participant's gait rhythm became stable and matched the auditory cue, the frequency of the auditory rhythm was gradually increased to achieve the ideal cadence without decreasing velocity and stride length and without losing gait balance. After RAS, each participant completed another 10-meter walk without RAS (post-test), after which final gait parameters were recorded. An appropriate walking aid such as a cane or a walker was used during training and testing if necessary. The whole procedure took about 20 minutes.

Paired t-tests were performed to determine if there was a significant difference between the pre- and post-test gait parameters (i.e., velocity and stride length) within the groups. The data were analyzed using SPSS, version 15, for Windows (SPSS, Chicago, IL, USA), and values of  $p < 0.05$  were considered statistically significant.

## RESULTS

As presented in [Table 1](#), participant characteristics across groups were relatively similar.

The velocities in the pre- and post-tests for each group are listed in [Table 2](#). Between the pre- and post-tests, the paired t-test revealed significant increases ( $p < 0.05$ ) in velocity in the cerebellum, pons and medulla, and thalamus groups. The putamen and corona radiata groups demonstrated increases in velocity. However, the increases were not significant.

The stride lengths in the pre- and post-tests for each group are listed in [Table 3](#). Compared to the pre-test, the paired t-test revealed significant increases ( $p < 0.05$ ) in stride length in the cerebellum, pons and medulla, and thalamus groups in the post-test. The putamen and corona radiata groups demonstrated increases in stride length. However, the increases were not significant.

## DISCUSSION

In this study, RAS was effective in increasing velocity and stride length in the cerebellum, pons and medulla, and thalamus groups. However, RAS did not influence gait parameters in the putamen and corona radiata groups. These findings suggest that the effects of RAS vary by the lesion site. Therefore, we opine that rehabilitation with RAS is effective in improving predictive motor timing in patients with lesions in specific regions.

The lesion sites that RAS affected were the cerebellum, pons and medulla, and thalamus. Lesions in these areas are associ-

**Table 2.** The velocities in the pre- and post-tests according to the lesion sites

Lesion site	Pre-test, m/min	Post-test, m/min	
Cerebellum	31.6 ± 14.6	37.7 ± 20.4	*
Pons and medulla	36.5 ± 18.6	39.4 ± 21.3	*
Thalamus	17.7 ± 10.3	21.0 ± 13.5	*
Putamen	21.1 ± 14.9	22.8 ± 16.6	
Corona radiata	22.9 ± 11.9	25.2 ± 15.7	

\*p&lt;0.05

**Table 3.** The stride lengths in the pre- and post-tests according to the lesion sites

Lesion site	Pre-test, m	Post-test, m	
Cerebellum	0.64 ± 0.23	0.73 ± 0.33	*
Pons and medulla	0.71 ± 0.24	0.75 ± 0.26	*
Thalamus	0.55 ± 0.15	0.61 ± 0.19	*
Putamen	0.54 ± 0.22	0.57 ± 0.24	
Corona radiata	0.57 ± 0.19	0.6 ± 0.20	

\*p&lt;0.05

ated with motor timing impairment; thus, RAS may compensate for the timing impairment. The cerebellum is known to play a critical role in anticipating and predicting timing<sup>13</sup>. Lesions in the cerebellum impair motor coordination and affect speed, rhythm, accuracy, and smoothness of motion, such as gait<sup>14</sup>. Along with the cerebellum, the pons and medulla play a role in the timing mechanism<sup>15</sup>. The pons receives signals from the cerebrum and relays information to the cerebellum. The pontine lesion can disrupt the corticopontocerebellar circuit and cause cerebral ataxia<sup>16</sup>. The medulla is also closely connected to the cerebellum. The inferior olivary nucleus, situated in the medulla, encodes the timing of sensory input and decodes it to the cerebellum by synchronizing the spikes of Purkinje cells with its rhythmic oscillatory activity. Therefore, the inferior olivary nucleus in the medulla has a role in the control of motor timing as a part of the olivo-cerebellar system<sup>17</sup>. Finally, the thalamus, which directly receives the output from the cerebellum, is another area where a lesion can cause motor timing impairment, cerebellar ataxia, and ataxia induced by the disturbance of the sense of depth<sup>18</sup>. Since RAS affected the velocity and stride length in these groups, RAS seems to compensate for the motor timing impairment, leading to an improvement in gait parameters.

Conversely, RAS did not influence the velocity and stride length in the putamen and corona radiata groups. Lesions in these sites may cause motor timing impairment; however, this is rare. In case of the putamen, impairment in motor timing occurs with bilateral lesions, not unilateral lesions<sup>19, 20</sup>. In case of unilateral putamen lesion, the intact hemisphere may compensate for motor timing in contrast to the unilateral lesions in the cerebellum, pons and medulla, and thalamus. Lesions in the corona radiata may cause an ataxic hemiparesis syndrome with accompanying sensory impairment by disrupting the cerebello-cortical pathway<sup>21, 22</sup>. However, the occurrence is infrequent and the influence on gait is minimal<sup>22, 23</sup>. As lesions in these regions do not significantly impair motor timing, expectedly, RAS does not significantly influence the gait parameters of patients with lesions in these regions.

Since patient characteristics across groups were relatively similar, it was unlikely that these would have influenced the effects of RAS. However, this study is limited to comparing the pre- and post-test gait parameters within the groups. Further studies, including a control group and performing between group comparisons are needed to clarify group differences.

In conclusion, RAS was effective in facilitating the prediction of motor timing and gait rhythm in stroke patients with lesion in the cerebellum, pons and medulla, and thalamus, which are associated with impairment of the timing mechanism.

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