



Original Article

# Primary and secondary gait deviations of stroke survivors and their association with gait performance

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**Abstract.** [Purpose] Stroke survivors exhibit abnormal pelvic motion and significantly deteriorated gait performance. Although the gait of stroke survivors has been evaluated at the primary level pertaining to ankle, knee, and hip motions, secondary deviations involving the pelvic motions are strongly related to the primary level. Therefore, the aim of this study was to identify the kinematic differences of the primary and secondary joints and to identify mechanism differences that alter the gait performance of stroke survivors. [Subjects and Methods] Five healthy subjects and five stroke survivors were recruited. All the subjects were instructed to walk at a self-selected speed. The joint kinematics and gait parameters were calculated. [Results] For the stroke survivors, the range of motion of the primary-joint motions were significantly reduced, and the secondary-joint motions were significantly increased. Additionally, for the healthy subjects, the primary joint kinematics were the main factors ensuring gait performance, whereas for the stroke survivors, the secondary-joint motions were the main factors. [Conclusion] The results indicate that while increasing the range of motion of primary-joint movements is the main target to achieve, there is a strong need to constrain and support pelvic motions in order to improve the outcome of gait rehabilitation.

**Key words:** Stroke gait, Primary and secondary gait deviation, Pelvic motion

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## INTRODUCTION

After stroke, damaged descending neural pathways can cause abnormal movements, including an abnormal gait pattern<sup>1)</sup>. Consequently, stroke alters the kinematic, kinetic, and muscle activation patterns of survivors and can frequently cause spasticity at a certain joint. Subjects with antagonist groups of weakened muscles and spasticity tend to suffer more from passive stiffness and joint contracture than those without<sup>2-4)</sup>. Although the specific figures vary among studies, it is reported that 38–60% of stroke survivors suffer from muscle weakness and spasticity, and that stroke survivors with spasticity are more prone to be functionally impaired than those without spasticity<sup>5)</sup>.

In particular, weakened and spastic muscles at the knee joint interfere with voluntary knee movements. Various symptoms can arise because of this pathology, such as crouch gait, stiff-knee gait (SKG), and genu recurvatum with altered excursion and a reduced range of motion (RoM) of the lower limb joints<sup>6-10)</sup>. Crouch gait is defined as excessive knee flexion in the

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initial phase of gait due to weak hip extensors, knee extensors, or ankle plantar flexors<sup>8, 11, 12</sup>). SKG is characterized by significantly diminished and delayed knee flexion during the swing phase and is mainly caused by the spasticity of the rectus femoris muscle, weakness of the hip-flexor muscle, and over-activity of the ankle plantar flexor, which lead to an inefficient gait pattern<sup>10, 13, 14</sup>). Genu recurvatum, which is defined as knee hyperextension during the stance phase, is caused by quadriceps malfunction<sup>7, 9</sup>). All of these symptoms substantially increase the energy expenditure during walking and can lead to excessive pain and chronic joint degeneration if not corrected.

With the dysfunction of voluntary knee movements, compensatory pelvic motions are employed by stroke survivors to avoid foot drop and to compensate for the effects of gravity on the body during the swing and stance phase<sup>15</sup>). The pelvic movements during gait have received attention with regard to knee and foot mechanisms. Saunders<sup>16</sup>) proposed the concept of six determinants of gait, the six factors responsible for minimizing the vertical and lateral displacements of the center of mass of the body<sup>17-19</sup>). Many studies have shown that stroke survivors not only have an increased anterior pelvic tilt but also a contralateral pelvic drop in the coronal plane. Furthermore, the affected side of the pelvis is retracted in the transversal plane<sup>20-23</sup>). Karen and colleagues reported that pelvic lateral displacement was significantly increased to keep the body balanced during walking in patients with acute hemiparesis after stroke<sup>19</sup>).

Stroke survivors affected by voluntary knee movement dysfunction with abnormal pelvic motion exhibit remarkably deteriorated gait performance, which is characterized by significantly reduced gait velocity, and step and stride lengths<sup>14</sup>). The differences in the gait velocity, and step and stride lengths identify stroke-survivor gait performance, which is the major goal of gait rehabilitation<sup>24, 25</sup>), and therapists use these variables to plan and provide proper guidance for gait rehabilitation. Therefore, identifying the mechanisms involved in gait performance, such as the gait velocity and step or stride length, is crucial for providing better and quantitative clinical interventions. Nevertheless, to our knowledge, little work has been done on identifying the underlying mechanisms that affect the gait performance of stroke survivors with voluntary knee movement dysfunction in the context of their primary (ankle, knee, and hip joint) and secondary (pelvis) motions. Therefore, the aim of this study was to identify the kinematic differences of the primary and secondary joints of stroke survivors. Once these differences are ascertained, further efforts can be made to identify their associations with the gait performance of stroke survivors. The hypothesis of this study was that there is increased pelvic motion with decreased ankle, knee, and hip-joint RoMs in stroke survivors compared with controls, and that the increased pelvic motion is associated with the gait performance of stroke survivors. We hope that our findings will serve as a guideline for designing improved clinical interventions that aim to rectify the dysfunction of voluntary knee movements and provide guidance for rehabilitation-oriented gait-pattern improvements.

## SUBJECTS AND METHODS

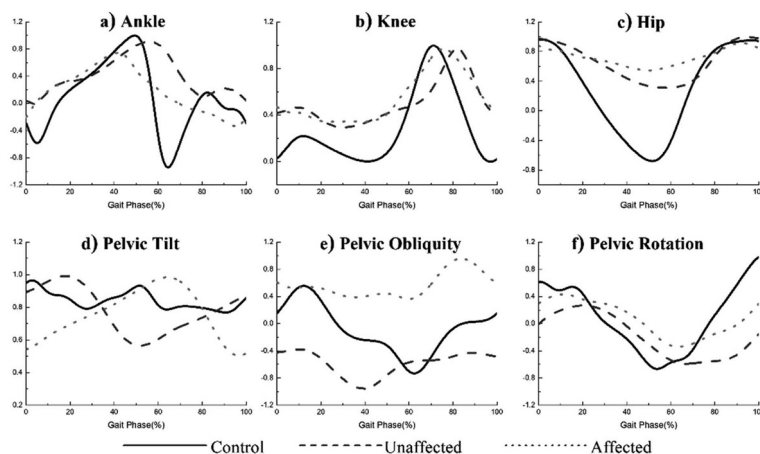
The subjects of this study were five healthy young males (age:  $29 \pm 2.9$  years; height:  $171.4 \pm 5.7$  cm; and weight:  $66.6 \pm 5.8$  kg) and five stroke patients (age:  $61.2 \pm 10.0$  years; height:  $161.6 \pm 5.0$  cm; and weight:  $64.9 \pm 7.3$  kg). The stroke patients were community-dwelling stroke survivors who had ambulatory hemiparesis due to either right or left supratentorial ischemic stroke or intracerebral hemorrhage more than two years earlier. All the survivors exhibited impaired gait. Participants with movement disorders or orthopedic diseases that might have influenced their gait, such as arthrosis or total hip-joint replacement, were excluded from the study. This study was conducted with the prior consent of all the subjects, and written informed consent was obtained from each subject. No human rights were violated during the experiment. This study was approved by the Institutional Review Board.

With 16 reflective optical markers attached to the body, all the subjects were instructed to walk at a self-selected speed along a 10-m walkway in a gait laboratory. The markers were attached to anatomic landmarks located on the pelvis and lower extremities in accordance with the Plug-in-Gait marker set. During the trials, the subjects did not wear an orthosis, and were not provided with any weight support. Eight high-speed optical cameras (Vicon, Oxford, UK) captured the three-dimensional (3D) positions of the reflective markers at a sampling rate of 100 Hz, and the gait kinematics were calculated according to the positions of each marker. The subjects were asked to repeat the trial, if they were not tired, until five successful trials had been completed.

A customized software package of the Vicon motion capture system (Nexus, Oxford, UK) was used for pre-processing the raw kinematic data. The marker data were low-pass filtered using a zero-lag fourth order Butterworth filter with a cutoff frequency of 6 Hz. All gait-related parameters were grouped into three categories: the control of the healthy subjects, and the unaffected and affected limbs of the stroke survivors. Gait phases such as heel-strike and toe-off, as well as the kinematic profiles of the ankle, knee, and hip-flexion/extension angles and the pelvic tilt, obliquity, and rotation, were determined using the software. For further analysis, the gait phase, gait kinematic parameters, and 3D marker data were analyzed using Matlab (MathWorks, Natick, MA). The RoMs of the kinematic profiles, as well as the step length, stride length, gait velocity, and lateral displacement of the pelvis, were computed using a customized Matlab program.

Because each subject had different RoMs yielding different kinematic profiles, the kinematic profiles were normalized with the corresponding RoMs of each profile for clearer comparison of the kinematic profiles and to eliminate the effects of different RoMs due to the severity of the pathology.

The SPSS program (SPSS Inc., Chicago, IL, USA) was used to analyze the data. The differences in the kinematic profiles



**Fig. 1.** Profiles of the ankle, knee, and hip-joint angles of the normal individuals and stroke survivors

**Table 1.** RoMs of the primary and secondary joints and important gait parameters

	Controls	Stroke patients	
		Unaffected	Affected
RoM of ankle (°)	30.41 ± 6.5	26.08 ± 12.3*	20.51 ± 4.1**
RoM of knee (°)	55.56 ± 5.6	41.25 ± 16.1*	21.27 ± 11.6**
RoM of hip (°)	41.78 ± 1.9	35.09 ± 14.4	19.80 ± 13.0**
RoM of pelvic tilt (°)	2.54 ± 0.6	12.12 ± 7.7*	14.32 ± 8.8**
RoM of pelvic obliquity (°)	7.56 ± 2.6	7.65 ± 1.9	8.66 ± 2.8
RoM of pelvic rotation (°)	11.71 ± 2.3	14.44 ± 11.9*	17.42 ± 14.0**
Lateral displacement (LD, mm)	67.30 ± 19.3	107.42 ± 27.2*	123.17 ± 27.1***
Normalized step length	0.71 ± 0.04	0.32 ± 0.23	0.39 ± 0.15
Normalized stride length	1.35 ± 0.07	0.67 ± 0.33*	0.71 ± 0.35**
Velocity (m/s)	1.03 ± 0.15	0.34 ± 0.22*	0.33 ± 0.20**
Stance phase duration (%)	60.00 ± 1.3	75.26 ± 5.9*	65.51 ± 11.2

\*Statistical difference between the control and unaffected side,  $p < 0.05$

\*\*Statistical difference between the control and affected side,  $p < 0.05$

\*\*\*Statistical difference between the affected and unaffected sides,  $p < 0.05$

among the groups were compared using the Pearson product-moment correlation coefficient. One-way ANOVA was used to investigate the differences in the RoMs of the kinematic profiles and the gait performances among the groups. When one-way ANOVA produced statistically significant results, Tukey's post hoc test was performed to examine the differences. All the significance levels were chosen as  $p = 0.05$ . To identify the association between the gait performance and other gait kinematic parameters, the Pearson product-moment correlation coefficient was used; i.e., the step length, stride length, and gait velocity were compared with the RoMs of the kinematic profiles. The correlation criterion  $r$  value indicates the following degrees of correlation: 0.9 to 1, very high; 0.7 to 0.9, high; 0.5 to 0.7, moderate; 0.3 to 0.5, low; and 0.0 to 0.3, little if any.

## RESULTS

All the stroke survivors who participated in the study had crouch gait, SKG, genu recurvatum, or a combination of these symptoms.

The kinematic profiles are shown in Fig. 1, and the RoMs of the kinematic parameters and gait performance are presented in Table 1. Figures 1a, b, and c show the normalized ankle, knee, and hip (AKH) profiles, respectively, which represent the primary-joint motions. The AKH profiles of the stroke survivors exhibited similar patterns to those of the controls, but the RoMs of the AKH joints were significantly lower. The correlation coefficients of the kinematic profiles between the controls and the unaffected-limb group were 0.402 ( $p < 0.001$ ) for the ankle, 0.650 ( $p < 0.001$ ) for the knee, and 0.885 ( $p < 0.001$ ) for the hip. The correlation coefficients between the controls and the affected-limb group were slightly higher: 0.678 ( $p < 0.001$ ) for the ankle, 0.895 ( $p < 0.001$ ) for the knee, and 0.966 ( $p < 0.001$ ) for the hip. However, the RoMs of the AKH profiles of the

**Table 2.** Correlation of step length, stride length, and gait velocity with RoMs and other gait parameters

		Ankle	Knee	Hip	Pelvic tilt	Pelvic obliquity	Pelvic rotation	LD
Controls	Step length	0.692**	0.273	0.236	-0.132	-0.472*	0.434	-0.273
	Stride length	0.443	0.483*	0.222	-0.022	-0.170	0.341	-0.357
	Gait velocity	0.366	-0.187	0.593*	-0.240	-0.242	-0.095	-0.719**
Unaffected	Step length	0.836**	0.851**	0.976**	0.889**	0.196	0.747**	-0.303
	Stride length	0.813**	0.834**	0.978**	0.850**	0.215	0.716**	-0.318
	Gait velocity	0.553**	0.737**	0.889**	0.572**	0.342	0.449**	-0.479*
Affected	Step length	-0.249	0.855**	0.682**	0.753**	0.136	0.786**	-0.366*
	Stride length	-0.256	0.922**	0.851**	0.866**	-0.026	0.867**	-0.215
	Gait velocity	-0.148	0.735**	0.613**	0.629**	-0.106	0.646**	-0.395*

\*Statistically significant,  $p < 0.05$ \*\*Statistically significant,  $p < 0.01$ 

unaffected- and affected-limb groups were significantly lower than those of the controls (Table 1).

Figure 1d shows the normalized pelvic-tilt profiles. The unaffected-side pelvic motion (USPM) exhibited an excessively anterior-tilted pattern at the initial contact (IC), and the anterior tilt decreased during mid-stance (MST), whereas the affected-side pelvic motion (ASPM) showed a relatively reduced anterior tilt at the IC but reached the maximum anterior tilt in mid-swing (MS). There was no correlation between the control and USPM ( $r = -0.024$ ,  $p = 0.446$ ), or between the control and ASPM ( $r = -0.083$ ,  $p = 0.008$ ). The USPM was highly and negatively correlated with the ASPM ( $r = -0.766$ ,  $p < 0.01$ ). There was a significant difference in the RoMs of the pelvic tilts among the groups ( $p < 0.01$ ). The RoMs of the pelvic tilt of the unaffected and affected sides were larger than those of the controls by factors of 5.54 and 6.04, respectively.

The profile of the normalized pelvic obliquity is shown in Fig. 1e. The USPM decreased at the IC, with further reduction at the loading response and terminal stance, but was elevated during the swing phase. In contrast, the ASPM increased at the IC and further elevated during MS, indicating a hip-hiking pattern to counter the foot drop<sup>22</sup>). There was a mild correlation between the controls and the USPM ( $r = 0.450$ ,  $p < 0.01$ ), but no correlation between the controls and the ASPM ( $r = 0.274$ ,  $p < 0.01$ ). No significant differences were found between the RoMs of the pelvic obliquity of the control and stroke groups.

Figure 1f illustrates the normalized pelvic-rotation profile. The USPM was retracted at the IC, and the profiles are moderately or highly correlated with each other: the correlation between the controls and USPM was 0.426 ( $p < 0.01$ ), that between the controls and ASPM was 0.666 ( $p < 0.01$ ), and that between the USPM and ASPM was 0.946 ( $p < 0.01$ ). However, the RoM of pelvic rotation was significantly increased on both sides of the stroke survivors compared to the control.

Other important gait parameters are shown in Table 1. The lateral displacement of the pelvis, which indicates the postural performance during gait, was significantly higher for the stroke patients ( $p < 0.01$ ). Additionally, gait performances, such as the normalized step length, stride length, and gait velocity, were significantly reduced for both the unaffected and affected limbs of the stroke group ( $p < 0.01$ ).

To evaluate the relationship between the gait performances and other kinematic parameters, the correlations between the RoMs of the kinematic parameters and the normalized step length, stride length, and gait velocity were calculated (Table 2). For the control group, the RoM of the ankle was moderately correlated with the step length ( $r = 0.692$ ,  $p < 0.05$ ), and the RoM of the pelvic obliquity was negatively correlated with the step length ( $r = 0.483$ ,  $p < 0.05$ ). The RoM of the knee was slightly correlated with stride length, and the hip RoM was moderately correlated with the stride length ( $r = 0.593$ ,  $p < 0.01$ ). The lateral displacement of the pelvis was negatively correlated ( $r = -0.719$ ,  $p < 0.01$ ) with the gait velocity for the controls.

For the unaffected-limb group, parameters including the RoMs of the ankle, knee, hip, pelvic tilt, and pelvic rotation were significantly or highly correlated with the step length and stride length, and moderately or highly correlated with gait velocity. For the affected side, the RoMs of the knee, hip, pelvic tilt, and pelvic rotation were the main contributors to the gait performance. Interestingly, the RoM of the ankle joint of the affected limb did not contribute to the gait performance of the stroke survivors. The lateral displacement of the pelvis was negatively correlated with the gait velocity of both sides.

In summary, the AKH flexion/extension RoMs of the controls were the main factors affecting their gait performance, and the pelvic kinematic RoMs, especially the pelvic tilt and rotation combined with the AKH joint motions, were the main factors behind the dysfunction of voluntary knee movement of the stroke patients.

## DISCUSSION

We examined the differences in kinematic profiles and investigated the relationship between the gait performance and kinematic parameters of stroke survivors with voluntary knee movement dysfunction. The results show that compared with the control group, the movements of the primary joints, such as the AKH, were significantly lower among the stroke survi-

vors, whereas the RoMs of the secondary joints were significantly higher. These results are consistent with previous studies, which concluded that stroke survivors have limited RoMs at the primary joints and increased RoMs at the secondary joints owing to compensatory movements. However, there was less agreement or no consensus on the relationship between the RoMs and gait performance. Therefore, the main emphasis of this study was to identify the relationship between the gait performance and the RoMs of the kinematic profiles of the primary and secondary joints to provide improved guidance for gait rehabilitation.

The normalized kinematic profiles of the primary deviation of the stroke patients were moderately or highly correlated with those of the control group, but the RoMs were significantly reduced, which were in agreement with previous studies<sup>6, 8–10, 26</sup>). The reduced RoMs at the primary joints can be explained by the fact that crouch gait, which is caused by weakness of the triceps surae muscle, makes the progression of the tibia during MST excessive, yielding excessive knee flexion from IC to MST. In addition, spasticity of the rectus femoris muscle, weakness of the hip flexor, and over-activity of the ankle plantar-flexor may have limited the peak knee angle during MS<sup>14, 27</sup>). These malfunctions of the lower-limb muscles resulted in limited RoMs of the ankle, knee, and hip flexion/extension movements in this study. With reduced primary joint RoMs, stroke survivors may actively modify their gait pattern by exerting compensatory strategies in order to facilitate locomotion<sup>15</sup>). This is important because primary abnormalities have been shown to develop into secondary deviations, and the two are spontaneously related to each other.

Increased pelvic tilt angles were observed in this study, which is consistent with a previous study that concluded that the majority of stroke patients increased their pelvic tilt angles by factors of 4.77 on the unaffected side and 5.63 on the affected side, with an excessively tilted anterior position<sup>22</sup>). As shown in Fig. 1e, the unaffected side of the pelvis slightly dropped at IC, whereas the affected side ascended during MS, exhibiting a hip-hiking pattern to avoid foot drop during MS<sup>23</sup>). There were no statistical differences in the RoMs of pelvic obliquity among the groups. In contrast, the pelvic-rotation profiles of the stroke survivors were highly correlated with those of the controls, and the RoMs of the pelvic rotation were higher in the stroke group (1.2 times on the unaffected side, 1.48 times on the affected side). The larger pelvic rotation of the stroke group is attributed to their performance of compensatory movements to increase the gait performance.

The gait performances of the stroke survivors were characterized by reduced gait velocity and step length. Restoring the gait velocity and step length is considered a main goal of gait rehabilitation. The results of this study show that the average gait velocity of the stroke survivors was lower (0.34 m/s for the unaffected limb and 0.33 m/s for the affected limb) than that of the controls (1.03 m/s). The normalized step length (0.32 m for the unaffected limb and 0.39 m for the affected limb) and stride length (0.67 m for the unaffected limb and 0.71 m for the affected limb) were also significantly lower than those of the control group (0.71 m for step length and 1.35 m for stride length).

Although gait-performance deterioration varies with the severity of the pathological condition, the association of the gait kinematics with the gait performance (velocity, step length, and stride length) of stroke survivors remains unclear. Hsu investigated the most important parameter determining the gait velocity of patients who had survived mild to moderate strokes using regression analyses and determined that the hip flexors and knee extensors were the most important determinants of a comfortable gait and high gait velocity<sup>28</sup>). Kim examined the relationship of the magnitude and pattern of the kinematic and kinetic gait parameters to the preferred gait velocity, reporting that the RoMs of the hip and knee were moderately correlated with the gait velocity for the paretic limb and that the RoM of the ankle was correlated with the unaffected limb<sup>24</sup>). Cruz identified the paretic hip-extension strength as a contributor to the gait velocity<sup>25</sup>). However, these previous studies did not employ a control group of neurologically healthy individuals, nor did they investigate the contributions of the pelvic motion to the gait performance. As previously mentioned, secondary deviations can develop from primary deviations; thus, it is important to investigate the association between the pelvic motion and the gait performance. Our results show that the normalized step length, stride length, and gait velocity of the controls were moderately and significantly correlated with the AKH RoMs. The pelvic obliquity was negatively correlated with the step length, and the lateral displacement negatively contributed to the gait velocity. In contrast, for the unaffected-limb, all the gait parameters except for the pelvic obliquity and lateral displacement were contributors to the gait performance. For the affected limb, the RoMs of the knee, hip, pelvic tilt, and pelvic rotation were significant contributors to the gait performance.

Generally, the pelvic motion plays a central role in locomotion, contributing to the forward progression of the body and trunk vertical support. In addition, the vertical displacement of the human center of mass is partly reduced by the motion of the pelvis<sup>16</sup>). Thus, pelvic motions may not have contributed to the gait performance of the control group. The lateral displacement of the pelvis of the control subjects was highly and negatively correlated with the gait velocity because this parameter is related to stability and balance rather than the forward velocity of gait. In contrast to the results of the controls, the gait performance of the unaffected limb of the stroke group was bolstered by increasing the magnitude of the pelvic tilt and pelvic rotation with abnormal excursion of pelvic obliquity in order to compensate for the reduced RoMs of the AKH joints. Therefore, the pelvic movements were not only a compensatory motion aiming to clear the foot during the MS, necessitated by the lack of ankle dorsiflexion and knee and hip flexion, but also movements aimed at improving the step length, stride length, and gait velocity. This shows a deviation from the standard role of pelvic motion in healthy subjects. Our results show that pelvic obliquity does not contribute to gait performance and may only play a foot-clearance role through the adjustment of the hip-hiking pattern, as shown in Fig. 1e. The gait performance of the affected-limb was similar to that of the unaffected-limb, but the ankle RoM did not correlate with the gait performance of the affected side, a result

which is in agreement with previous studies<sup>24</sup>). Kim and Eng concluded that the ankle RoM is the key kinematic factor affecting the gait efficiency of adolescents with cerebral palsy. Our results show that ankle RoM did not contribute to any of the gait-performance parameters, demonstrating the necessity of ankle and knee muscle strength training for improving the lower-limb RoM and gait performance. In addition, gait rehabilitation should be conducted to encourage the use of AKH joint movements with constrained pelvic RoMs in order to reduce the influence of secondary deviations on the gait performance.

This study had several limitations. First, only five subjects participated; thus, the results need to be confirmed with a larger sample size. However, to increase the reliability, we reduced the variability by recruiting only stroke survivors with voluntary knee movement dysfunction and increasing the number of gait trials of each subject. Second, the control group and stroke group were not matched with respect to their ages. The main purpose of gait rehabilitation for stroke survivors is to increase their gait performance to make it as normal as possible. For this reason, healthy young subjects were selected as the control group in this study, as normal elderly subjects also have deteriorated gait patterns due to aging. Therefore, it is our opinion that a comparison between healthy young and stroke survivors provides the most useful guideline for effective gait rehabilitation. Third, only the kinematic differences and their associations to gait performances were evaluated. Considering that stroke survivors may use different mechanisms to achieve similar kinematic movement, both kinematic and joint-power information should be investigated in the future. Despite these acknowledged limitations, the study emphasizes the importance of pelvic-motion support and serves as a guide for gait rehabilitation.

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