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You are better off running than walking revisited: Does an acute vestibular imbalance affect muscle synergies?

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ABSTRACT

It has been suggested that vestibular cues are inhibited for the benefit of spinal locomotor centres in parallel with the increase in locomotion speed. This study aimed at quantifying the influence of a transient vestibular tone imbalance (TVTI) on gait kinematics, muscle activity and muscle synergies during walking and running. Twelve participants walk or run at a self-selected speed with or without TVTI, which was generated by 10 body rotations just prior the locomotion task. Three-dimensional lower-limb kinematic was recorded simultaneously with the surface electromyographic (EMG) activity of 8 muscles to extract muscle synergies via non-negative matrix factorization. Under TVTI, there was an increased gait deviation in walking compared to running ($22.8 \pm 8.4^{\circ}$ and $8.5 \pm 3.6^{\circ}$, respectively; $p < 0.01$), while the number ($n = 4$) and the composition of the muscle synergies did not differ across conditions ($p = 0.78$). A higher increase ($p < 0.05$) in EMG activity due to TVTI was found during walking compared to running, especially during stance. These findings confirmed that the central nervous system inhibited misleading vestibular signals according to the increase in locomotion speed for the benefit of spinal mechanisms, expressed by the muscle synergies.

1. Introduction

Originating from the lateral vestibular nucleus (or Deiters nucleus) located within the brainstem, the vestibulospinal tract acts on extensor muscles during locomotion (Orlovsky, 1972a). Indeed, the functional recruitment of the plantar flexor muscles during the stance phase of the locomotion was related to the phasic activity of the vestibulospinal tract itself regulated by signals coming through the spinocerebellar tract and cerebellum (Kanaya, Unno, Kawahara, & Mori, 1985; Matsuyama & Drew, 2000; Orlovsky, 1972a). When electrically stimulated, the vestibulospinal tract increased the plantar flexor muscle tone, but the timing of the stimulation had no effect on the muscle coordination (Orlovsky, 1972b). It has been suggested that the regulation of muscle activity to occur in the appropriate phases of the locomotor cycle might be performed by a different, probably spinal, mechanism (Grillner, 1985). In addition, the activity of the Deiters' neurons evoked by a vestibular stimulation has been found to be substantially decreased during locomotion or inhibited by a concurrent stimulation of the mesencephalic locomotor region during a static task (Orlovsky & Pavlova, 1972).

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The inhibition of vestibular cues with the increase in movement velocity (from posture to walking to running) has been found also in humans (Brandt, 2000; Brandt, Strupp, & Benson, 1999; Dakin, Inglis, Chua, & Blouin, 2013; Jahn, Strupp, Schneider, Dieterich, & Brandt, 2000). For instance, Brandt et al. (1999) found a decrease in lateral deviation of gait during running compared with walking in patients with unilateral vestibulopathy and in healthy participants subjected to a transient vestibular tone imbalance (TVTI). More recently, Dakin et al. (2013) showed a decrease in the vestibulo-muscle coupling due to the increase in walking cadence, especially during the stance phase of the gait cycle. Overall, these results likely suggested a change in the neural networks implicated in the control of locomotion, with a higher dependence on vestibular inputs for posture and slow gait than for fast paced locomotor behaviours which relied more on automatic spinal networks. Very early in the literature (Orlovsky & Pavlova, 1972), the functional origin of this vestibular inhibition was hypothesized to avoid interferences from irregular head movements on the activity of spinal and supraspinal networks implied in the control of fast paced locomotor behaviours. Recent functional neuroimaging findings corroborated these assumptions (Deutschländer et al., 2009; Jahn et al., 2004, 2008; la Fougère et al., 2010). A low activity of supraspinal structures (cerebellum, thalamus and basal ganglia) acting on spinal neural networks has been found for balance control in quiet standing. However, as the movement pace increased (walking and running), the activity of these supraspinal networks increased while vestibular and somatosensory cortices were progressively inhibited (Jahn et al., 2004).

Recently, it has been suggested that low-dimensional modules formed by muscles activated in synchrony, named muscle synergies, may serve as building blocks simplifying the construction of motor behaviours (Bizzi & Cheung, 2013; d'Avella & Bizzi, 2005; Ivanenko et al., 2003; Ting & McKay, 2007). It is supposed that these muscles synergies constitute the bottom of a hierarchical neural control structure implying descending commands from spinal, supraspinal and cortical structures (Ting et al., 2015; Ting & McKay, 2007). Each muscle synergy is composed by a spatial component (also called motor module or muscle module) which represent the relative weighting of each muscle within the synergy and a time-varying component (also called motor primitive) which represent the recruitment of the muscle synergy over time (Hug, Turpin, Guével, & Dorel, 2010). While the neural hypothesis of the muscle synergies is still a matter of debate within the current literature (Kutch & Valero-Cuevas, 2012), a recurrent point in favour of this neural origin is the consistency of the motor modules across motor tasks and mechanical constraints. Such a consistency is interpreted as a depiction of hard-wired spinal neural networks and modulation in their temporal activation might be related to the integration of sensory inflows (Hagio, Fukuda, & Kouzaki, 2015; Hug, Turpin, Couturier, & Dorel, 2011; Oliveira, Silva, Lund, Kersting, & Farina, 2013; Safavynia & Ting, 2012, 2013; van den Hoorn, Hodges, van Dieën, & Hug, 2015). For instance, Martino et al.(2015) found four motor modules with consistent composition across unstable conditions of walking while they observed a widening of the motor primitives to cope with the dynamic instability.

Therefore, we aimed at reinvestigating the locomotion speed change paradigm to analyse the effect of an acute vestibular imbalance on the muscle synergies. As previously found and due to the vestibular imbalance, we hypothesized an increase in gait deviation (Brandt et al., 1999) and in muscle tone (Dakin et al., 2013) with the decrease in locomotion speed. According to the hypothesis of a neural origin of muscle synergies, we expected to find a consistency of motor modules across locomotion speeds and vestibular conditions (with or without an acute vestibular imbalance). Any changes in the motor modules could be interpreted as descriptive muscle synergies which mirror the altered pattern of locomotion due to the vestibular imbalance. On the contrary, the consistency of the motor modules would be in agreement with the lack of effect of the vestibulospinal tract on muscle coordination (Orlovsky, 1972b) and with the hypothesis of hard-wired motor modules into the spinal moto-neuronal network (Frère, 2017).

2. Experimental procedures

2.1. Participants

Twelve healthy participants (height: 166–196 cm, mass: 53.7–91.6 kg, 2 women and 10 men) between 20 and 29 years voluntarily participated in this study. They performed pendular rotary vestibular test with videonystagmography and video head impulse test to assess semicircular canals response. None of the participants showed any inner ear pathology, except one which has been excluded from this study because of a left vestibular hyporeflexia. Therefore, the analysis of the results was carried out on eleven participants. All the participants were informed of the purpose of the study and methods used before providing written consent. A local ethics committee approved the study and all the procedures conformed to the Declaration of Helsinki.

2.2. Protocol

Participants had to move through a motion capture volume of 10-m length, with their eyes closed and at a self-selected speed. The participant was asked to move forward as straight ahead as possible. Four conditions of displacement have been investigated: walk or run with or without (*i.e.*, control) vestibular imbalance. The conditions were performed in a randomized order, with two trials per condition with 5 min of rest in-between. During each trial, an experimenter was permanently beside the moving participant within touching distance to prevent any fall.

The acute vestibular imbalance was evoked by means of ten clockwise rotations at an angular velocity of around $360^{\circ}.s^{-1}$ of the participant seated and secured in a rotary chair (Framiral®, Grasse, France) with the eyes closed. After the ten rotations, the experimenter stopped the rotary chair according to the longitudinal axis of the room and helped the participant (still with the eyes closed) to stand up from the chair. According to Brandt et al. (1999), this procedure induced a transient vestibular tone imbalance (TVTI) which was performed just prior the locomotion task (walk or run).

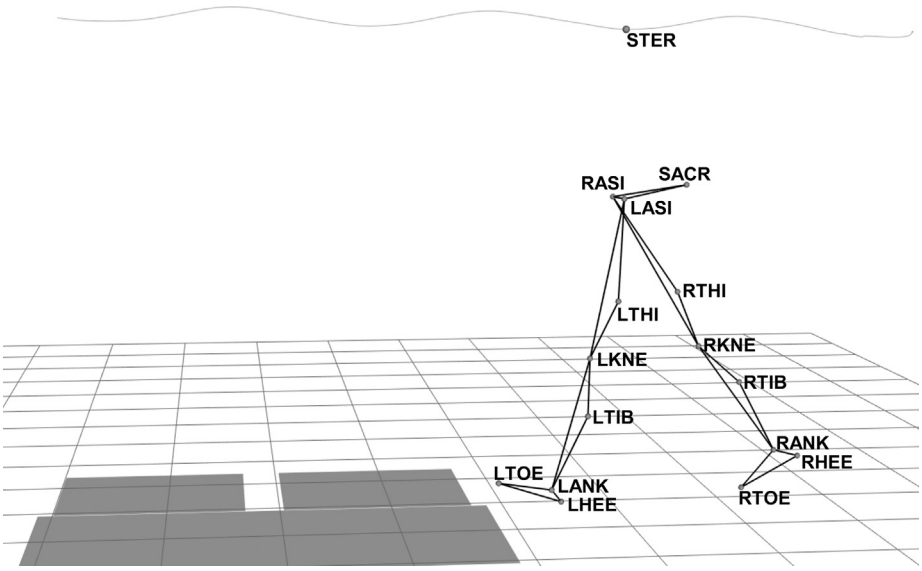

Fig. 1. Visualization of the marker set used to capture the lower-limb kinematics with an additional marker placed over the xiphoid process of the sternum (STER) to compute gait deviation and locomotion speed. R and L prefix: right and left side, respectively; ASI: anterior superior iliac spine; SACR: sacrum; THI: lower lateral 1/3 surface of the thigh; KNE: lateral femur epicondyle; TIB: lower lateral 1/3 of the shank; ANK: lateral malleolus; HEE: calcaneus; TOE: second metatarsal head.

2.3. Materials and data collection

Spatiotemporal and three-dimensional kinematic data of the locomotion were recorded bilaterally at 100 Hz using an optoelectronic motion analysis system (VICON Peak, Oxford Metrics, UK) consisting of nine infrared cameras spaced around the walkway. Infrared reflective markers were attached on each side of the participants to the skin following the marker set for Plug-In Gait lower body model in addition with one marker placed over the sternum dedicated to quantify the gait deviation and speed (Fig. 1). Marker labelling and trajectory reconstructions were performed using Nexus software (VICON Oxford Metrics, UK).

Surface electromyography (EMG) was collected unilaterally (right lower limb) from eight muscles: *tibialis anterior* (TA); *soleus* (SOL); *gastrocnemius medialis* (GM); *vastus medialis* (VM); *rectus femoris* (RF); *biceps femoris* (BF); *semitendinosus* (ST); *gluteus medius* (G_{med}). The surface EMG recordings were made using self-adhesive Ag/AgCl pairs of electrodes (Blue Sensor N, Ambu A/S, Ballerup, Denmark) with an inter-electrode distance of 20 mm (centre-to-centre). The electrodes were placed longitudinally with respect to the underlying muscle fibre arrangement (de Luca, 1997) and were located according to the recommendations of Surface EMG for Non-Invasive Assessment of Muscles (SENIAM) (Hermens, Freriks, Disselhorst-Klug, & Rau, 2000). Skin was shaved and cleaned with alcohol to minimize impedance before applying the electrodes and the wires connected to the electrodes were secured carefully with adhesive tape to avoid any movement-induced artefacts. Raw EMG signals were preamplified (with a gain of 10) and analog-to-digitally converted at a sampling rate of 1 kHz (myon 320, myon AG, Schwarzenberg, Switzerland).

2.4. Data processing

Gait cycles were defined as the time between heel-strike to the next ipsilateral heel strike, while steps were defined as the time between heel strike of one foot to heel strike on the contralateral foot. Heel strikes were determined from the local vertical minima of the right heel marker position (van den Hoorn et al., 2015). Similar to the method proposed by Martino et al. (2015), steps related to gait initiation and termination were discarded for the analysis of gait patterns. The following general gait parameters were calculated for each subject in each condition: walking speed ($km.h^{-1}$), gait deviation (°), step width (mm), step length asymmetry (Clark, Ting, Zajac, Neptune, & Kautz, 2010), step duration (s) and step length (m). Step length asymmetry was obtained by first calculating the ratio of one step length to its respective overall cycle length. Then, the deviation from symmetry was quantified by the difference between 0.5 and this step ratio. Ranges of angular motion (RoM) were calculated from the flexion/extension joint angles of both lower the limbs (hip, knee and ankle). The kinematic data were time-normalized in order to obtain 100 data points for each gait cycle.

Raw EMG signals were band-pass filtered (20–450 Hz, Butterworth filter, 4th order), rectified, smoothed with a zero-lag low-pass filter (8 Hz, Butterworth filter, 4th order), and time-normalized in order to obtain 100 data points for each gait cycle. For each gait cycle and each muscle, the EMG envelope was normalized to the maximum level activity. Four to eight gait cycles were analysed for each participant and each condition. Non-negative matrix factorization (NMF) was performed from this dataset. For this purpose, the multiplicative update rules algorithm (Lee & Seung, 2001) was used to extract muscle synergies (Matlab *nnmf.m* function; option = 'mult'), as follows:

$$\boldsymbol{E} = \boldsymbol{WC} + \boldsymbol{e}$$

E is an *i*-by-*j* initial matrix (i = number of muscles and j = number of time points), **W** is a *i*-by-*n* matrix (n = number of synergies), **C** is a *n*-by-*j* matrix, and **e** is a *i*-by-*j* matrix. **W** represents the muscle synergy vector matrix (*i.e.*, motor modules), **C** is the synergy activation coefficients matrix (*i.e.*, motor primitives), and **e** is the residual error matrix. The algorithm is based on iterative updates of an initial random guess of W and C that converge to a local optimal matrix factorization [see Lee and Seung (2001) for more details]. To avoid local minima, the algorithm was repeated 50 times for each participant. The lowest cost solution was kept (*i.e.*, minimized squared error between original and reconstructed EMG patterns).

To determine the number of muscle synergies to extract, we used the proposed method by Cheung et al. [named knee point method herein; Cheung et al. (2009)]. Briefly, the variance accounted for [VAF, (Torres-Oviedo, Macpherson, & Ting, 2006)]-number of synergies curve was constructed from both the original EMG dataset and an unstructured EMG dataset generated by randomly shuffling the original dataset across time and muscles. The number of muscle synergies was then defined as the point beyond which the original-slope drops below 75% of the surrogate-slope. It corresponds to the number beyond which any further increase in the number of extracted synergies yields a VAF increase smaller than 75% of that expected from chance.

According to Martino et al. (2015), the full width at half maximum (FWHM, in % of cycle) has been calculated from each motor primitive in each condition. The FWHM was calculated as the sum of the durations of the intervals in which the motor primitives (after subtracting the minimum throughout the gait cycle) exceeded half of its maximum.

For each mode of locomotion (walking or running), a cross-validation procedure was performed to assess the similarity of the muscle synergies between the TVTI and the control condition. Briefly, the motor module matrix extracted during the TVTI condition was held fixed in the NMF algorithm while the motor primitive matrix of the control condition was free to vary (Allison et al., 2018; Frère & Hug, 2012; Hug et al., 2011; Torres-Oviedo et al., 2006). The VAF for each muscle (VAF_{muscle}) was used to quantify the success of the fixed motor modules and the newly motor primitives to reconstruct the EMG patterns of the control condition. A $VAF_{muscle} > 75\%$ was considered satisfying (Frère & Hug, 2012; Hug et al., 2011).

In addition with the muscle synergies extraction, we calculated the level of muscle activity during the stance and swing phase of all the studied gait cycles. In each phase (stance or swing), we computed the average root mean square (RMS) value of each myoelectric signal. For each mode of locomotion (walking or running), we normalized the level of activity of the TVTI condition according to the one of the control condition. The normalized activity of each muscle were grouped and averaged according to each motor module previously found with a similar set of muscles (Clark et al., 2010): **W**#1 composed of VM, RF and G_{med} muscles; **W**#2 composed of SOL and GM; **W**#3 composed of TA muscle; **W**#4 composed of BF and ST muscles. This provided a change in the level of activation in each muscle synergy according to the vestibular stimulation and mode of locomotion.

2.5. Statistical analysis

The statistics were performed with Statistica Software (STATISTICA software; StatSoft, Tulsa, OK). Spatiotemporal and kinematic data were found to be normally distributed (Shapiro–Wilk tests) with homogenous variances (two-sample *F*-tests). To assess the effect of the mode of locomotion (walking and running) and the effect of vestibular imbalance (TVTI and control) on gait deviation, speed, step width, step length asymmetry, step duration and joint angles ranges of motion, we used a two-way ANOVA for repeated measures.

To assess the influence of the four conditions of locomotion on the number of muscle synergies, we used the Cochran's Q test. The Pearson's correlation coefficient (r) was used as a similarity criterion for the motor modules between TVTI and control condition within each mode of locomotion. As previously done (Frère & Hug, 2012; Safavynia & Ting, 2012), we considered a pair of motor modules to be similar if $r = 0.834$, which corresponds to the critical r for 9 degrees of freedom (*i.e.*, 11–2 muscles) at $p = 0.01$. In addition, for each extracted synergy we generated 1000 random permutations of the weightings obtained from the extraction of the motor module vectors. Then we calculated the r-value for each pair (two comparisons for each of the 11 subjects = 22 pairs). Therefore, 88,000 r-values (22 pairs × 1000 iterations × 4 synergies) were obtained, yielding a distribution of r-values expected by chance. An r-value of 0.834 corresponded to the 96.6th percentile of the distribution. Consequently, we considered a pair of motor modules with an $r = 0.834$ more similar than expected by chance, and thus motor modules with an $r < 0.834$ were considered different. A Fisher's exact test was used to assess the relationship between the number of similar motor modules and the mode of locomotion. Also, a one-way multivariate analysis of variance for repeated measures was used to determine whether VAF_{muscle} from the cross-validation differed between the modes of locomotion among the eight studied muscles.

The comparison of the shape (*i.e.*, waveform) of motor primitives between both conditions within a mode of locomotion was assessed using two criterions: the Pearson's correlation coefficient (r) and the circular cross-correlation coefficient (r_{max}). The index of similarity corresponded to both the averaged r- and r_{max}-value between each pair of participants.

Multivariate analyses of variance for repeated measures have been used to assess changes in FWHM and in the normalized EMG activity of the motor modules. The level of significance was $p = 0.05$.

3. Results

3.1. Spatiotemporal and kinematics data

Gait deviation was significantly lower during running in comparison with walking [$F(1,10) = 21.4$, $p < 0.01$] and the TVTI

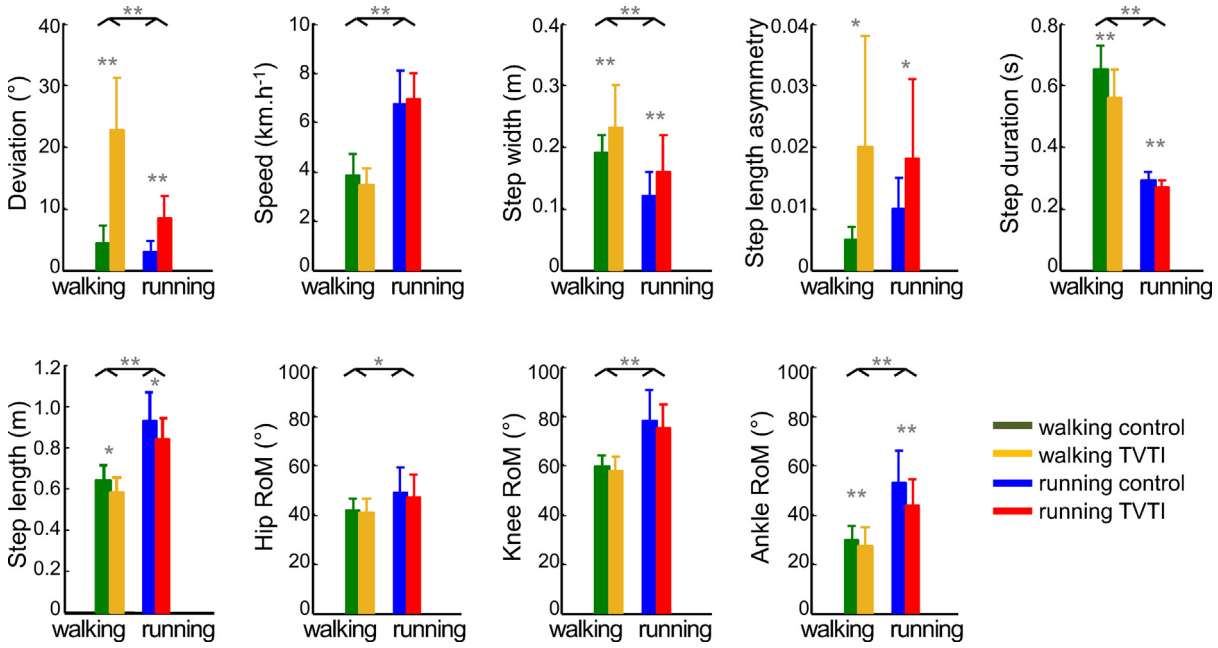

Fig. 2. Spatiotemporal and kinematic gait parameters among the four conditions of locomotion. Data are means (± SD). TVTI: transient vestibular tone imbalance; RoM: range of angular motion. *Difference between conditions with $p < 0.05$; **difference between conditions with $p < 0.01$.

induced a significant increase in gait deviation for both modes of locomotion [$F(1,10) = 91.2$, $p < 0.01$; Fig. 2]. A significant interaction effect has been found highlighting that the TVTI had a lesser effect during running than walking [$F(1,10) = 13.6$, $p < 0.01$]. Self-selected speed of locomotion was significantly higher during running in comparison with walking [$F(1,10) = 669.5$, $p < 0.01$], but it was not influenced by the TVTI for each mode of locomotion [$F(1,10) = 0.2$, $p = 0.66$; Fig. 2]. Running significantly decreased step width in comparison with walking [$F(1,10) = 138.1$, $p < 0.01$; Fig. 2], while TVTI significantly increased step width in comparison with control condition [$F(1,10) = 14.7$, $p < 0.01$]. However, no interaction effect has been found for step width [$F(1,10) = 0.4$, $p = 0.56$]. Regarding the step length asymmetry, there was no main effect of locomotion mode [$F(1,10) = 0.8$, $p = 0.40$] but a significant main effect of the TVTI [$F(1,10) = 5.7$, $p = 0.04$; Fig. 2]. A significant interaction effect has been found expressing that the TVTI had a lesser effect on step length asymmetry during running than walking [$F(1,10) = 7.9$, $p = 0.02$]. Both running and TVTI significantly decreased step duration in comparison with walking [$F(1,10) = 258.6$, $p < 0.01$; Fig. 2] and control condition [$F(1,10) = 13.9$, $p < 0.01$], respectively. Also, an interaction effect has been found revealing that TVTI had a lesser effect during running than walking on step duration [$F(1,10) = 8.4$, $p = 0.02$]. Running significantly increased step length in comparison with walking [$F(1,10) = 127.8$, $p < 0.01$; Fig. 2], while TVTI significantly decreased step length in comparison with control condition [$F(1,10) = 9.0$, $p = 0.01$]. However, no interaction effect has been found for step length [$F(1,10) = 0.5$, $p = 0.49$].

Solely a main effect of locomotion mode has been found for hip [$F(1,10) = 7.1$, $p = 0.02$] and knee RoM [$F(1,10) = 59.5$, $p < 0.01$; Fig. 2]. Ankle RoM was significantly higher during running in comparison with walking [$F(1,10) = 108.2$, $p < 0.01$] and the TVTI induced a significant decrease in ankle RoM for both modes of locomotion [$F(1,10) = 18.5$, $p < 0.01$; Fig. 2]. A significant interaction effect has been found highlighting that the TVTI had a lesser effect during walking than running [$F(1,10) = 6.2$, $p = 0.03$].

3.2. Number of muscle synergies

According to the knee point method, we extracted four muscles synergies for ten participants for the control walking and the TVTI running conditions and for nine participants for the control running and the TVTI walking conditions (Fig. 3A). From the Cochran's Q test (four muscle synergies = 1 and the other cases = 0), we did not find any relationship between the four conditions of locomotion and the number of extracted muscle synergies ($Q(3) = 0.8$, $p = 0.86$). Therefore, we systematically extracted four muscle synergies for each condition of locomotion for the subsequent analyses. When walking, four muscle synergies accounted for a mean VAF of 95.3 ± 1.7% and of 94.3 ± 1.2%, for control and TVTI conditions, respectively. When running, four muscle synergies accounted for a mean VAF of 96.5 ± 1.0% and of 95.3 ± 1.1%, for control and TVTI conditions, respectively (Fig. 3B). TVTI induced a significant decrease in VAF values in both walking and running conditions [$F(1,10) = 24.8$, $p < 0.01$)], while running VAF values were significantly higher than walking [$F(1,10) = 14.3$, $p < 0.05$)].

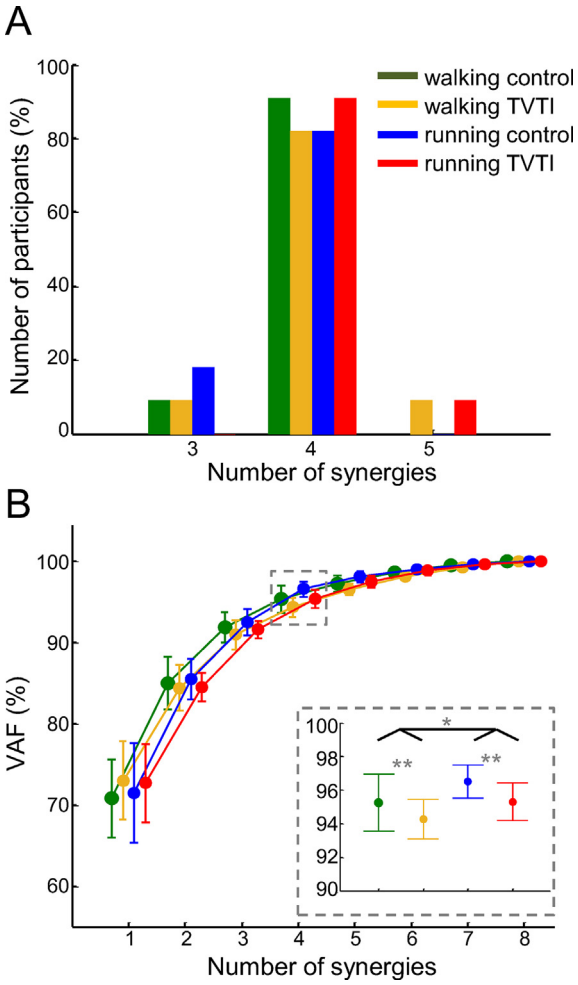

Fig. 3. (A) Number of muscle synergies retained per condition based on the knee point method (Cheung et al., 2009). (B) Variance accounted for (VAF) as function of the number of muscle synergies for each condition of locomotion. Data are means (± SD). (Inset) Differences in VAF among the conditions of locomotion at four muscle synergies. TVTI: transient vestibular tone imbalance. *Difference between conditions with $p < 0.05$; **difference between conditions with $p < 0.01$.

3.3. Similarity of muscle synergies

The extracted muscle synergies are depicted in Fig. 4, which showed the commonalities in both motor modules and primitives between the control and TVTI conditions when walking as well as running. For all the conditions, the four muscle synergies were found to be functionally relevant. The first muscle synergy implied mainly the knee extensor muscles and hip abductor muscles (VM, RF and G_{med}), which was activated during the load acceptance at the beginning of the stance phase. The second muscle synergy implied the plantar flexor muscles (SOL and MG) during the propulsion at the end of the stance phase (around 40% and 20% of the gait cycle during walking and running, respectively). The third muscle synergy mainly involved the TA muscle at the initiation of the swing phase, while the fourth muscle synergy implied the hamstring muscle (BF and ST) at the end of the swing phase. On considering the threshold *r*-value of 0.834, we found that 6 comparisons (out of 11 possibilities, *i.e.*, 54.6%) were considered as similar between control and TVTI during walking for **W**#1, as well as 11 (100%) for **W**#2, 7 (63.6%) for **W**#3 and 11 (100%) for **W**#4. Similarly, 8 comparisons (72.7%) were found similar between control and TVTI during running for **W**#1, as well as 9 (81.8%) for **W**#2, 10 (90.9%) for **W**#3 and 10 (90.9%) for **W**#4. Overall, the Fisher's exact test found no relationship between the number of similar motor modules and the mode of locomotion ($p = 0.78$). Moreover, the cross-validation procedure led to high values of $VAF_{muscles}$ (Table 1) for walking and running, with no main effect of mode of locomotion [Wilk's $\lambda = 0.1$, $F(8,3) = 2.9$, $p = 0.21$].

3.4. Changes in FWHM of motor primitives

The Fig. 5 depicted the changes in FWHM of each of the four motor primitives due to the TVTI during both modes of locomotion. No main effect of locomotion mode [Wilk's $\lambda = 0.4$, $F(4,7) = 2.4$, $p = 0.15$], or vestibular stimulation [Wilk's $\lambda = 0.3$, $F(4,7) = 3.7$,

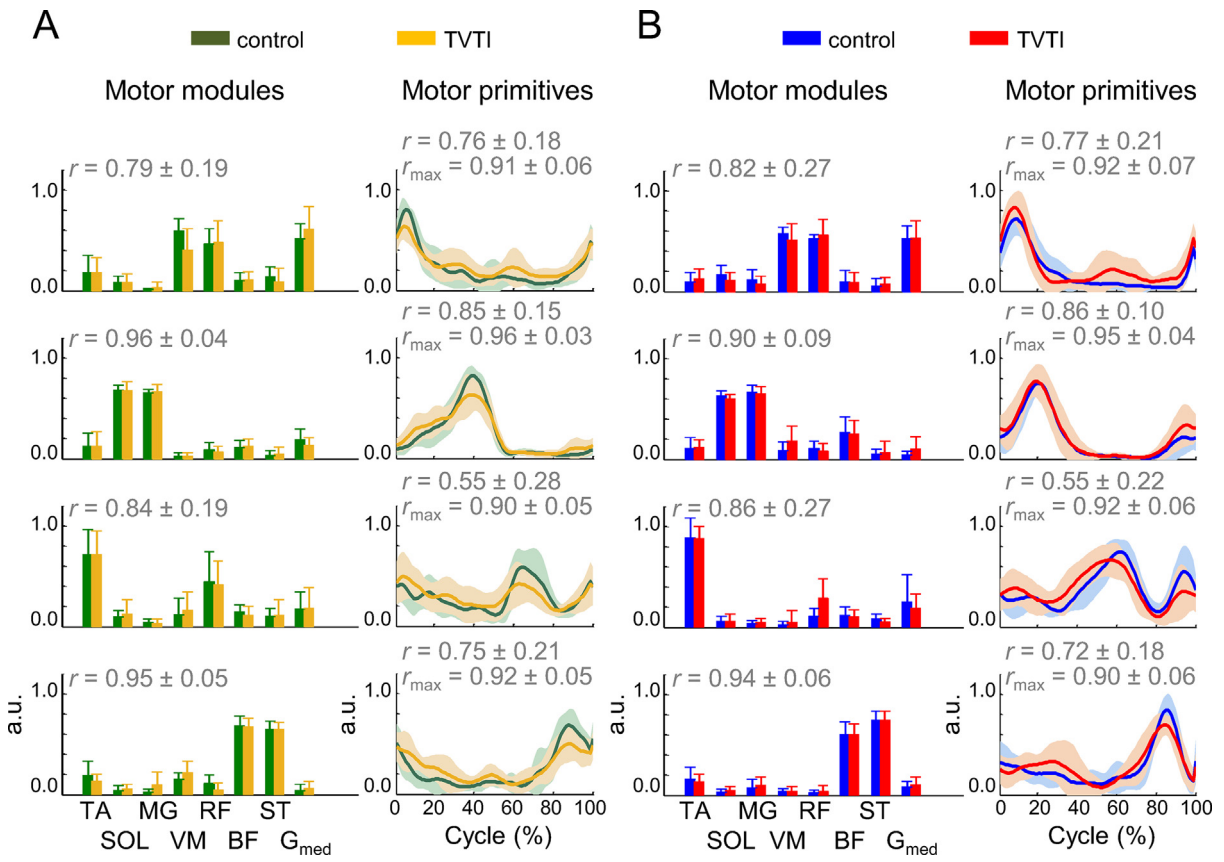

Fig. 4. Motor modules (means ± SD) and their corresponding ensemble-averaged (± SD) primitives during (A) walking and (B) running. The similarity of the motor modules between both conditions (control *vs.* TVTI) was assessed by the correlation coefficient (r) while the one of the motor primitives was assessed by the cross-correlation coefficient (r_{max}) in addition to r-value. TVTI: transient vestibular tone imbalance; TA: *tibialis anterior*; SOL: *soleus*; GM: *gastrocnemius medialis*; VM: *vastus medialis*; RF: *rectus femoris*; BF: *biceps femoris*; ST: *semitendinosus*; G_{med}: *gluteus medius*.

Table 1
Muscles variance accounted for (VAF_{muscle}, in %) from the cross-validation procedure. Data are means (range).

	TA	SOL	MG	VM	RF	BF	ST	G_{med}
Walking	97 (89–100)	98 (96–100)	98 (94–100)	91 (72–98)	96 (82–99)	96 (88–99)	96 (88–98)	95 (91–100)
Running	99 (97–100)	97 (92–100)	98 (93–100)	94 (84–99)	94 (88–99)	97 (94–99)	98 (96–99)	92 (72–100)

Note: TA: *tibialis anterior*; SOL: *soleus*; GM: *gastrocnemius medialis*; VM: *vastus medialis*; RF: *rectus femoris*; BF: *biceps femoris*; ST: *semitendinosus*; G_{med}: *gluteus medius*.

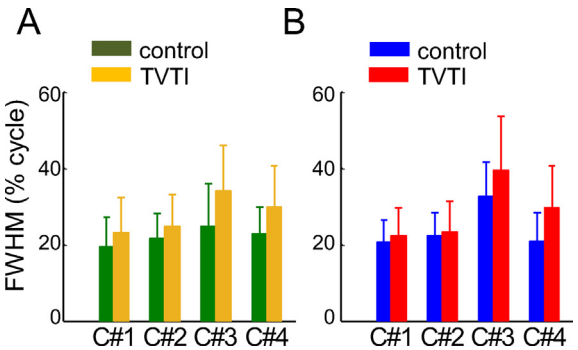

Fig. 5. Full width at half-maximum (FWHM) of the four motor primitives (C#1 to C#4) during (A) the walking conditions and (B) the running conditions. Data are means (± SD). TVTI: transient vestibular tone imbalance.

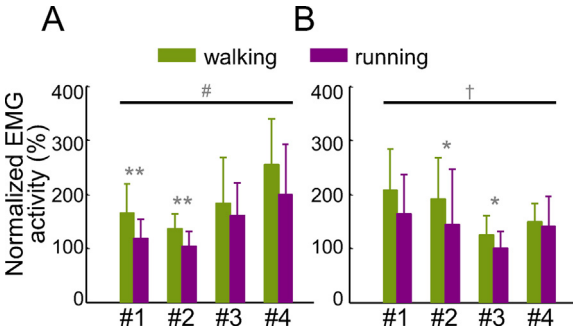

Fig. 6. Normalized myoelectric activity of each muscle groups (#1 to #4) during the transient vestibular tone imbalance condition relative to the control condition of locomotion (A) during the stance phase and (B) during the swing phase of the gait cycle. Data are means (± SD). #MANOVA main effect of the mode of locomotion with $p < 0.05$; †MANOVA main effect of the mode of locomotion with $p < 0.01$; *difference between conditions with $p < 0.05$; **difference between conditions with $p < 0.01$.

$p = 0.07$], nor interaction effect has been found [Wilk's $\lambda = 1$, $F(4,7) = 0.1$, $p = 0.98$].

3.5. Changes in muscle activity of muscles synergies

The normalized EMG activity of each muscle group differed significantly between running and walking during the stance phase of the gait cycle [Wilk's $\lambda = 0.3$, $F(4,7) = 4.7$, $p = 0.04$], with solely a significantly higher normalized activity in walking than in running for the first muscle group [Wilk's $\lambda = 0.5$, $F(1,10) = 11.3$, $p < 0.01$] and the second muscle group [Wilk's $\lambda = 0.5$, $F(1,10) = 12.0$, $p < 0.01$; Fig. 6A]. Similarly, during the swing phase, there was a significantly higher increase in normalized EMG activity due to TVTI in walking than in running [Wilk's $\lambda = 0.1$, $F(4,7) = 15.9$, $p < 0.01$]. In that case, the second muscle group had a higher normalized EMG activity in walking than in running [Wilk's $\lambda = 0.7$, $F(1,10) = 5.2$, $p = 0.046$], as well as for the third muscle group [Wilk's $\lambda = 0.6$, $F(1,10) = 7.7$, $p = 0.02$; Fig. 6B].

4. Discussion

The aim of this study was to determine the effect of transient vestibular tone imbalance (TVTI) during walking and running on gait kinematics, muscle synergies and their level of activity. The main findings were that gait deviation, step length asymmetry and step duration were more altered in walking than running due to the vestibular imbalance, while muscle synergies remained similar among the four conditions of locomotion. In addition, in response to the vestibular imbalance, there was larger increase of muscle synergies level of activity during the stance phase of the gait cycle while walking in comparison with running. Overall, these results confirmed our main hypothesis that the increase in pace induced an inhibition of vestibular cues for the benefit of spinal neural networks, expressed by muscle synergies, to control the locomotor behaviours.

We found here that the vestibular imbalance induced an increase in gait deviation, step width, step length asymmetry and a decrease in step duration, step length and ankle range of angular motion (Fig. 2). These findings corroborated with previous reports within the literature relative to the control of dynamic gait stability (Brandt et al., 1999; Jahn et al., 2000; Martino et al., 2015; Wuehr, Nusser, & Decker et al., 2016; Wuehr, Nusser, & Krafczyk et al., 2016). These results confirmed that the vestibular imbalance altered the locomotor behaviours, especially during walking. Indeed, the interaction effects (locomotion mode × vestibular imbalance) for gait deviation, step length asymmetry and step duration agreed with the speed dependence of the vestibular control of dynamic stability (Brandt, 2000; Brandt et al., 1999; Jahn et al., 2000; Schniepp et al., 2012; Schniepp, Möhwald, & Wuehr, 2017; Wuehr, Nusser, & Krafczyk et al., 2016). It is worth noting that solely the ankle joint kinematics have been affected by the vestibular imbalance in comparison with the two other lower limb joints (hip and knee). This reduced ankle range of angular motion might partly explain the reduced step length for both modes of locomotion and suggest an increase in step frequency to maintain the locomotion speed. These results agreed with those from Martino et al. (2015), while the gait instability differed in origin (cerebellar ataxia). Overall, the post-rotary transient vestibular tone imbalance applied herein might lead to a displacement of the internal representation of the straight ahead (Jahn et al., 2000) which could lead to an erroneous perception of the direction of locomotion and then provide the mechanical conditions of gait instability.

As previously found with this set of studied lower limb muscles, we extracted four muscles synergies for all the locomotion conditions with similar motor modules and primitives (Boccia, Zoppirolli, Bortolan, Schena, & Pellegrini, 2018; Chia Bejarano et al., 2017; Clark et al., 2010; Neptune, Clark, & Kautz, 2009; Routson, Clark, Bowden, Kautz, & Neptune, 2013; Ting et al., 2015). Our results agreed with previous studies which found that the low-dimensionality characterizing the multi-muscles activity was preserved between walking and running (Cappellini, Ivanenko, Poppele, & Lacquaniti, 2006; Hagio et al., 2015) or among unstable conditions of walking (Martino et al., 2015; Oliveira, Gizzi, Kersting, & Farina, 2012). We found that the reconstruction quality (*i.e.*, VAF) was significantly higher during running than walking and the vestibular imbalance systematically lowered VAF values (Fig. 3B). This could be interpreted as an increase in locomotor complexity (Clark et al., 2010) in walking, and even more during the TVTI condition, in comparison with the running conditions. However, these TVTI-related increases in motor complexity were not of enough

magnitude to generate a change in the organization of the muscle synergies, such as previously found in case of neurologic disorders (Clark et al., 2010). Similar to the findings of Martino et al. (2015), the motor modules were not affected by the unstable conditions related to the vestibular imbalance. Our results related to the similarity criterion and the cross-validation procedure converged toward a consistency of the motor modules whatever the locomotion mode or vestibular state. This consistency agreed with the Orlovsky's pioneering outcomes (1972b) that stimulating the vestibulospinal tract did not change the timing of muscle activations and thus might not interfere with the muscle synergies organization. Moreover, the motor module consistency during walking (with or without TVTI) might be seen as additional evidence in favour of the neural hypothesis of the muscle synergies (Bizzi & Cheung, 2013; Frère, 2017; Hagio et al., 2015; Hug et al., 2011; Oliveira et al., 2013). Indeed, the most altered locomotor patterns were observed during the walking with TVTI condition, while we did not find any change in the motor modules during such condition in comparison with the control walking condition. This was also true for the running conditions, but the speed dependency of the vestibular control of locomotion (Brandt et al., 1999; Dakin et al., 2013; Jahn et al., 2000; Orlovsky & Pavlova, 1972; Schniepp et al., 2017) led us to expect such findings.

Motor primitives were also highly similar (Fig. 4) among the four locomotion conditions. In agreement with Cappellini et al. (2006) and Hagio et al. (2015), we found a shift toward the beginning of the gait cycle of the propulsion muscle synergy (#2) during running in comparison with walking (whatever the vestibular state), while the overall sequence of activation was preserved among the four locomotion conditions. Besides, we did not find any effect of the vestibular stimulation nor the locomotion mode on the widening of the motor primitives. This result disagreed with previous reports on various mechanical challenges or neural conditions of gait instability (Chia Bejarano et al., 2017; Martino et al., 2015; Oliveira et al., 2012). Therefore, we cannot claim that vestibular imbalance could lead to a central adaption that may change the temporal recruitment of muscle synergies to cope with poor balance. Without rejecting the effect of altered sensory cues on the muscle synergies' recruitment, one might suppose that the evoked vestibular imbalance did not generate a sufficient challenge, in terms of gait instability, to elicit this central strategy of widening the motor primitives.

As expected, the level of muscle synergy activity (*i.e.*, normalized EMG activity) increased with TVTI and this phenomenon was larger during walking than running. This agreed with the increase in muscle activity related to the electrical stimulation of the vestibulospinal tract (Orlovsky, 1972b) and with the speed dependent vestibular control of locomotion (Brandt et al., 1999; Schniepp et al., 2012). As the speed of locomotion increased, the coupling between the vestibular inflows and the muscle activity tended to decrease, especially for ankle plantar flexor muscles during stance (Dakin et al., 2013; Orlovsky & Pavlova, 1972). Moreover, our results revealed that this higher increase in muscle activity during walking than running followed a functional process, since the two first muscle synergies (load acceptance and propulsion) had larger level of activity during the stance of walking in comparison with running, while it was during the swing phase for the third muscle synergies (specific to the early swing). This highlighted the functional role of the vestibulospinal tract mainly dedicated to the activity of the plantar flexor muscles during the stance phase (Orlovsky, 1972a, 1972b).

The consistency of the motor modules and primitives despite kinematics changes of gait supports the neural hypothesis of the muscle synergies. It is worth noting that such hypothesis is still a matter of debate within the literature. For instance, previous reports (De Groote, Jonkers, & Duysens, 2014; Gritsenko, Hardesty, Boots, & Yakovenko, 2016) found that the activation of multiple muscles followed a similar low-dimensional organization due to the mechanical constraints of the musculoskeletal system without any assumption of synergistic commands. This may question any interpretation of the spinal origin of EMG-based muscle synergies without excluding a higher level of control (Rana, Yani, Asavasopon, Fisher, & Kutch, 2015). Therefore, an alternative view of our results could lie on the supraspinal integration of vestibular imbalance leading to an increase in muscle tone from vestibulocerebellum projections without affecting the cortical control of muscle synergies.

Some methodological considerations have to be disclosed. First, we extracted muscle synergies from a limited number of gait cycles (< 10). This was related to the curvilinear path of locomotion (especially during walking with TVTI) which led the participant to get out of the motion capture area. In addition, we consciously restricted the number of trials per condition to preserve participants from an early onset of dizziness or motion sickness. If this limited number of gait cycles decreased the opportunity to take into account the gait cycle variability, Oliveira, Gizzi, Farina, and Kersting (2014) showed that it had a little effect on the muscle synergies organization. Another limit was the unilateral design of this study, in terms of vestibular stimulation and EMG recordings. To generate the TVTI, the 10 rotations were systematically performed clockwise. This generally led to leftward lateral deviations but rightward deviations were also observed. But for the reason stated above (motion sickness), we decided to stimulate clockwise only to limit the number of TVTI trials. As the participants were free of unilateral or bilateral vestibular dysfunctions, one can reasonably think that an additional TVTI condition (due to counterclockwise rotations) would provide similar findings. Due to the curvilinear path of locomotion under TVTI (especially during walking), bilateral EMG recordings would allow to distinguish muscle synergies between the inner and outer lower-limbs and possibly reveal additional changes in the locomotor control under vestibular imbalance. However, recent findings from voluntary curvilinear walking showed very similar muscle synergies between both lower-limbs (Chia Bejarano et al., 2017). Therefore, one can suppose that our main findings would remain even with a bilateral study design.

5. Conclusion

Our results confirmed the hypothesis that the central nervous system inhibited the misleading vestibular signals in a speed dependent manner in favour of spinal mechanisms, expressed by the muscle synergies, to control the locomotor behaviours.

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