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PII: S0966-6362(19)31792-8

DOI: https://doi.org/10.1016/j.gaitpost.2019.12.022

Reference: GAIPOS 7418

To appear in: *Gait & Posture*

Received Date: 17 July 2019

Revised Date: 9 October 2019

Accepted Date: 16 December 2019

Please cite this article as: Bonaventura RE, Giustino V, Chiaramonte G, Giustiniani A, Smirni D, Battaglia G, Messina G, Oliveri M, Investigating Prismatic Adaptation effects in handgrip strength and in plantar pressure in healthy subjects, *Gait and amp; Posture* (2019), doi: https://doi.org/10.1016/j.gaitpost.2019.12.022

Manuscript number GAIPOS_2019_752_R1_Revised Manuscript

Investigating Prismatic Adaptation effects in handgrip strength and in plantar pressure in healthy subjects

Running title: Prismatic Adaptation effects in strength and posture

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Highlights

- Prismatic Adaptation induces different effects on hand grip and plantar pressure.
- Prismatic Adaptation weakens strength in the hand contralateral to deviation side.
- Prismatic Adaptation induces a forward displacement in plantar pressure.

Abstract

Background

Prismatic Adaptation (PA) is a visuomotor procedure inducing a shift of the visual field that has been shown to modulate activation of a number of brain areas, in posterior (i.e parietal cortex) and anterior regions (i.e frontal cortex). This neuromodulation could be useful to study neural mechanisms associated with either postural measures such as the distribution of plantar pressure or to the generation of muscle strength. Indeed, plantar pressure distribution is associated to activation of high-level cognitive mechanisms taking place within the posterior regions of the brain dorsal stream, especially of the right hemisphere. Conversely, hand force mostly rely on sensorimotor mechanisms, fulfilled by anterior regions of the brain and involving both hemispheres.

Research question

Since PA effects have been reported to affect both sensorimotor and higher level cognitive processes, is it possible to hypothesize a modulation of both hands strenght and plantar pressure after PA?

Methods

Forty-six healthy subjects (male=23; mean age=25±3 years) were randomly divided into two groups: a leftward prismatic adaptation group (l-PA) and a rightward prismatic adaptation group (r-PA). Hand strength and plantar pressure were assessed, immediately before and after PA, using the handgrip task and baropodometric measurement, respectively.

Results

Both l-PA and r-PA induced a significant decrease of strength in the hand contralateral to the lenses deviation side. Only r-PA was associated with an increase of the forefoot plantar pressure in both feet. Modulation of interhemispheric inhibitory processes at sensorimotor and higher cognitive level may account for the present results.

Significance

PA exerts effects on body posture and hand strength relying on different mechanisms. The PA effects on hand strength are probably related to the modulation of interhemispheric inhibition of sensorimotor

processes, involving both hemispheres. The PA effects on body posture are probably related to modulation of body representation, involving mainly the right hemisphere.

Key words: Prismatic Adaptation; Body Posture; Baropodometry; Handgrip task; Strength.

1. Introduction

In the last decades, prismatic adaptation (PA) effects have been widely investigated either in visuomotor processes [1,2] and higher level cognitive domains [3]. PA induces a lateral displacement of the visual field, enhancing cortical activity in the hemisphere ipsilateral to the lenses deviation side [4], an activation involving both posterior brain regions of the dorsal stream (i.e. occipito-parietal cortex) and anterior regions, mainly in the frontal cortex.

This pattern of neuromodulation explains why PA has also been used to study the physiological mechanisms of postural control, in both neurological patients [5,6] and healthy subjects [7]. An aspect of body posture particularly suitable to be studied with PA is the weight distribution among feet, that allows a symmetrical distribution of plantar pressure [8] in terms of reaction force to the ground [9]. Plantar pressure is controlled by both subcortical [10,11] and higher level brain mechanisms, such as the elaboration of an internal model of the body [12] and attention processes [13].

PA could also be useful to study and modulate processes associated to regulation of muscle strength. Remarkably, hand strength relies on sensorimotor processes that regulate corrections and adjustments depending on the executed force task [14,15]. Attentional resources, directionally shifted by PA, are also involved in regulating feet and hand movements. Indeed, a reduction of muscle force has been found when attention is shared between hand and leg [16].

Relevant differences occur between the parameters of hand strength and plantar pressure distribution in terms of hemispheric asymmetries and activated regions in each hemisphere. Indeed, stronger involvement of anterior regions (i.e., motor cortex) is required to control hand strength [17]. On the other hand, posterior brain regions (i.e., parietal cortex) might be more involved in regulating pressure distribution among feet, allowing the access to an internal model of the body and the control of its position in the space [18]. Another point concerns hemispheric asymmetries in postural control and muscle strength. Previous studies have shown a right hemisphere pivotal role on balance control and body posture in stroke patients [19,20] as well as in healthy subjects [21,22]. Conversely, motor control of each hand symmetrically depends on the activation of the contralateral brain hemisphere [23].

In this line, previously reported rightward-PA effects (i.e., right hemisphere activation) on body posture have been explained in terms of modulation of higher level cognitive processes [24], subserved by posterior regions of the right dorsal stream [25]. For instance, in a previous study, rightward-PA has been shown to rebalance the abnormal body weight distribution and therefore, the posture bias of patients after cerebrovascular accident [6]. The authors suggested that the rebalance in body weight distribution occurred through a PA-induced modulation of higher order processes of spatial orientation related to parietal lobe [6].

A previous study investigating changes in body sway in two groups of healthy subjects reported a forward displacement of the center of pressure (CoP) after both leftward and rightward PA [7]. Authors suggested that the observed changes in CoP reflected a displacement in the projection of body pressure, reflecting a change in body scheme. Unluckily, whether these effects extended directly to weight distribution among feet was not investigated. Additionally, asymmetries between plantar pressure and hands were not explored. Overall, to date, no studies have investigated whether PA might affect hand strength through modulation of anterior regions of the dorsal stream [26,27].

Indirect evidence might be found in neurophysiological and electrophysiological studies showing that PA modulates oscillatory activity over motor cortex (M1) as well as motor evoked potentials' amplitude [4,28].

The present study aimed at exploring interhemispheric asymmetries in the PA effects on hand strength and plantar pressure distribution. To this end, we evaluated hand strength and baropodometric functions immediately before and after leftward vs. rightward PA in two groups of healthy subjects. Since PA affects either sensorimotor and higher level attentive processes modulating the dorsal stream activity [29,30], wearing prisms could affect both hand and feet functions. Specifically, since body posture depends more on posterior dorsal stream regions of the right hemisphere [21,22,31] while hands strength is controlled by left and right motor cortices [32], we expected 1) changes in body posture occurring only after rightward PA; 2) changes in hand strength following either leftward and rightward PA.

2.Material and methods

2.1 Participants

Forty-six (male= 23; mean age= 25 ± 3 years) right-handed healthy participants were randomly assigned to a leftward Prismatic Adaptation group (l-PA; n= 23; mean age= 26 ± 3.92 years) or a rightward Prismatic Adaptation group (r-PA; n= 23; mean age= 25 ± 1.87 years). The l-PA group wore a 20° left shifting prismatic lenses and the r-PA group wore a 20° right shifting prismatic lenses. Participants handedness was assessed using the Edinburgh Handedness Inventory [33].

Exclusion criteria were prior diagnosis of psychiatric disease, brain injury, acute orthopaedic injury, pregnancy, depression, not corrected vision impairment or other neurologic diseases. Four subjects were excluded from the experiment: one subject due to pregnancy and three subjects due to knees' injuries. The study was in compliance with the Helsinki declaration. Participants were informed about the experimental procedures and provided their written informed consent to voluntarily participate in

the experiment. Experimenter and participants were both naïve to the experimental hypothesis tested. Table 1 shows participants demographic characteristics.

2.2 Experimental design

Baropodometric and handgrip measurements were collected twice: the first time before PA (Pre-PA) and the second after PA (Post-PA). The delay between the first and the second measurement was ~15 min, that is in the frame time of the PA effects [34,35].

2.3 Postural assessment

Baropodometric evaluation was conducted using the freeMed® posturographic system (Sensor Medica®; Guidonia Montecelio, Roma, Italia), consisting of the freeMed®Maxi platform and the freeStep®software. Signal was digitalized at a sampling frequency of 50 Hz. The baropodometric test, lasting 5 sec, was performed in a sound-isolated room. Each participant was required to stand barefoot in orthostatic stance on the platform with the head in neutral position, gazing forward, arms along the trunk and feet placed side-by-side with both heels in line. The following parameters were considered: rearfoot/forefoot and total plantar pressure (%); rearfoot/forefoot and total surface area (cm^2).

2.4 Handgrip test

Each participant performed 3 trials of 3 sec of maximal isometric handgrip on a mechanical dynamometer (KernMap model 80K1 - Kern®, Kern & Sohn GmbH, Balingen, Germany), alternatively with the dominant and the non-dominant hand, with 3 min rest between each trial. The subjects performed the handgrip test while seating in a chair, back at 90º angle with sacrum, shoulder blades immobilized to the backrest, head in neutral position, gazing forward, and elbow joint positioned at a 90º angle, as recommended by the American Society of Hand Therapists [36]. The best performance out of the 3 trials (kg) was included in the statistical analyses.

2.5 PA procedure

We followed the same PA procedure as in previous studies [4,28]. Subject sat in basic position (right index finger at the sternum) in front of the concave side of a curved Plexiglas panel at a distance of 57-cm. The panel was graded with vertical lines corresponding to the degrees of the visual angle (covering a total visual angle of 120°). Three vertical lines of the panel were marked to indicate central position (0°), left position (21° to the left), right position (21° to the right). During PA, the experimenter, facing the opposite side of the panel, randomly pointed in one of the three marked positions of the panel.

The task required to point with the right index finger the panel point indicated by the experimenter and then return to the basic position. Pointing accuracy was collected in five experimental conditions: pre-exposure, blind pre-exposure, early exposure (first 9 trials while wearing prisms), late exposure (last 9 trials while wearing prisms), blind post-exposure (after prisms removal). In the blind exposure conditions the pointing task was performed with hidden arm. Prismatic lenses were worn only during the exposure condition.

Exposure condition included 90 trials, while the other conditions included 30 trials. All the trials were equally and randomly distributed in the three marked positions of the panel.

2.6 Data analysis

Analyses were conducted on the mean accuracy of the 5 experimental conditions: pre-exposure, blind pre-exposure, early exposure, late exposure, blind post-exposure. Prismatic adaptation was analysed using a 5×2 repeated measures ANOVA, with Condition (all 5 experimental conditions) as within-subjects factor and Group (l-PA vs. r-PA) as between-subjects factor.

Handgrip

Handgrip performances were analysed using a 2×2×2 repeated measures ANOVA with Time (pre-PA vs. post-PA) and Hand (left vs. right) as within-subjects factors and Group (l-PA vs. r-PA) as between-subjects factor.

Plantar Surface Area

Total plantar surface data were analysed using a 2×2×2 repeated measures ANOVA with Time (pre-PA vs. post-PA) and Feet (left vs. right) as within-subjects factors and Group (l-PA vs. r-PA) as between-subjects factor.

Forefoot/rearfoot plantar surface data were analysed using a 2×2×2×2 repeated measures ANOVA with Feet (left vs. right), Time (pre-PA vs. post-PA) and Area (forefoot vs. rearfoot) as within-subjects factors and Group (l-PA vs. r-PA) as between-subjects factor.

Plantar Pressure

Total plantar pressure data were analysed using a 2×2 ANOVA, with Time (pre-PA vs. post-PA) as within-subjects factor and Group (l-PA vs. r-PA) as between-subjects factor. Since changes in pressure distribution in one foot are accompanied by proportional changes in the other one (i.e., the two variables negatively correlate), analyses have been conducted only on pressure distribution in the right foot. Namely, whether pressure on the left foot increases, it proportionally decreases on the right foot. Similarly, forefoot plantar data were analysed using a 2×2×2 ANOVA with Time (pre-PA vs. post-PA) and Feet (left vs. right) as within-subjects and Group (l-PA vs. r-PA) as between-subjects factors. As for total pressure distribution among feet, whether pressure on the forefoot increases,

pressure distribution in the rearfoot proportionally decreases, therefore analyses were conducted only in the forefoot data.

Bonferroni post-hoc tests were used to test main effects and interactions when appropriate. All the analyses were conducted using IBM SPSS Statistics software 23 (International Business Machines Corporation, Armonk, New York, United States).

3. Results

3.1 Prismatic adaptation

Figure 1 shows prismatic adaptation for l-PA and r-PA group across the five experimental conditions. ANOVA showed significant effects of the factors Group [$F(1,44)= 46.776$; $p< .001$; $\eta p^2= .325$] and Condition [$F(4,41)= 5.108$; $p= .029$; $\eta p^2= .104$] and a significant Group×Condition interaction [$F(4,41)= 662.583$; $p< .001$; $\eta p^2= .938$]. Lenses deviation was reflected by the difference between pre-exposure and early exposure trials, either in the l-PA ($p< .001$) and in the r-PA ($p< .001$) groups. Conversely, due to subjects' adaptation to prismatic deviation, no differences were found between pre-exposure and late exposure neither in the l-PA ($p= .085$) nor in the r-PA ($p= 1$) group. The presence of after effect was confirmed by a significant difference between blind pre-exposure and blind post-exposure either in the l-PA ($p< .001$) and in the r-PA ($p< .001$) group (Figure 1).

3.2 Handgrip

Figure 2 shows handgrip performance for the left and the right hand during the first and the second measurement across l-PA and r-PA groups.

ANOVA revealed significant main effects of the factors Hand [$F(1,44)= 37.730$, $p< .001$, $\eta p^2= .441$] and Time [$F(1,44)= 8.205$, $p= .006$, $\eta p^2= .157$] while the factor Group [$F(1,44)= .035$, $p= .853$, $\eta p^2= .001$] and the interaction Hand×Time [$F(1,44)= 3.345$, $p= .074$, $\eta p^2= .071$] were not significant. The interaction Hand×Time×Group was significant [$F(1,44)= 4.659$, $p= .036$, $\eta p^2= .096$]. Post-hoc tests revealed that l-PA reduced right hand strength (33.160 vs. 32.352, $p= .034$) and r-PA reduced left hand strength (31.506 vs. 30.389, $p= .006$) (Figure 2).

3.3 Plantar Surface Area

ANOVA on the total plantar surface area revealed a significant effect of the factor Feet [$F(1,44)= 12.576$, $p= .001$, $\eta p^2= .222$], while the factors Time [$F(1,44)= .137$, $p= .713$, $\eta p^2= .003$] and Group [$F(1,44)= 2.917$, $p= .095$, $\eta p^2= .062$] were not significant. The interaction Feet×Time was significant

[F(1,44)= 6.846, p= .012, ηp^2= .135], the interaction Feet×Time×Group was not significant [F(1,44)= .179, p= .674, ηp^2= .004].

ANOVA on the forefoot/rearfoot plantar surface area revealed a significant effect of the factors Feet [F(1,44)= 118.368, p< .001, ηp^2= .729] and Area [F(1,44)= 12.582, p< .001, ηp^2= .222], and no effect of the factors Time [F(1,44)= .102, p= .751, ηp^2= .002] and Group [F(1,44)= 2.895, p= .096, ηp^2= .062]. The interaction Feet×Area [F(1,44)= 4.305, p= .044, ηp^2= .089], Time×Area [F(1,44)= 6.417, p= .015, ηp^2= .127], Feet×Time×Group [F(1,44)= 5.806, p= .029, ηp^2= .104] were significant. None of the post hoc tests revealed significant differences (all *p values*> .05).

3.4 Plantar Pressure

ANOVA on the total plantar pressure revealed a significant effect of the factor Time [F(1,44)= 6.887, p= .012, ηp^2= .135] and not of the factor Group [F(1,44)= 1.028, p= .316, ηp^2= .023] neither of the interaction Time × Group [F(1,44)= .969, p= .330, ηp^2= .022]. Post-hoc tests on the main factor of Time revealed a decrease of plantar pressure on the right foot (49.537 vs. 48.006, p= .012) after PA, regardless of the lenses deviation side. There was no significant difference among the pre-PA measurements (all *p values*> .05).

Figure 3 shows forefoot/rearfoot plantar pressure distribution in the l-PA and r-PA groups during the first and the second measurement. ANOVA on the forefoot/rearfoot plantar pressure revealed that neither the main factor Time [F(1,44)= .987, p= .326, ηp^2= .022] nor the factor Feet [F(1,44)= 1.978, p= .167, ηp^2= .043] were significant, whereas the interaction Time×Group was significant [F(1,44)= 5.847, p= .020, ηp^2= .117]. The post-hoc tests revealed an increase in forefoot plantar pressure (48.947 vs. 51.342, p= .020) in both feet after r-PA (Figure 3).

4. Discussion

The main result of the present study was that PA differently affected muscle strength and plantar pressure depending on the side of prismatic deviation. Namely, we found a significant decrease in muscle strength in the hand contralateral to the lenses deviation side after either leftward or rightward PA. A forward displacement of plantar pressure of both feet was found selectively after r-PA.

As a secondary result, we found a decrease of plantar pressure on the right foot after PA, regardless of the lenses deviation side. This effect could be explained by a compensatory postural adjustment activated by the visuomotor unbalance determined by PA, and leading to greater pressure on the non-preferred foot in order to obtain body stabilization. Further studies would better address asymmetries between dominant and non-dominant foot in body stabilization following visuomotor perturbation.

This is the first study investigating the effect of PA on handgrip strength and plantar pressure.

We suggest that the weakening of strength we observed in hands depends on inhibitory processes taking place both during the handgrip task and PA. Namely, it has been shown that PA induces an enhancement of excitability levels of M1 ipsilateral to the lenses deviation side [4,28], whereas, due to interhemispheric inhibitory processes, excitability levels in the contralateral M1 decrease [37]. On the other hand, during muscle contraction, activity in the M1 contralateral to the tested hand increases, while excitability levels in the M1 ipsilateral to the tested hand decrease [38,39]. It has been reported that the inhibition of M1 ipsilateral to the activated hand lasts until muscles contractions of medium-intensity are reached; when maximal voluntary contractions (MVC) are reached, the pattern of activation changes [40,41]. Studies using near-infrared spectroscopy and functional magnetic resonance imaging have shown that M1 contralateral to the tested hand is activated when muscle strength is exerted from 20% to 60% MVC and then cortical reactivity reaches a plateau. At this point, higher muscle contractions (i.e., above 60%) are obtained through activation of the M1 ipsilateral to the tested hand, that complements activation of the contralateral one [40,41]. Noteworthy, in our task 100% of the MVC was required. This implies that the contribution of the M1 ipsilateral to the tested hand was pivotal in order to execute the grip task. We suggest that PA disrupted the recruitment of the M1 ipsilateral to the tested hand since this was inhibited by interhemispheric inhibitory processes occurring during PA [37].

In other words, PA inhibited the hemisphere contralateral to the lenses deviation side, thus preventing M1 recruitment to exert 100% of MVC during the handgrip task. If so, one should expect that PA either increase or decrease handgrip depending on the level of muscle contraction. Further studies will better address this issue.

In sum, these results do not contradict studies reporting an enhanced cortical activation in M1 ipsilateral to the lenses deviation side [4]. In particular, our findings add evidence to previous studies investigating PA effects with TMS over M1 and reporting that PA induces changes in excitability levels of M1 ipsilateral to the lenses deviation side [4].

However, for hand strength weakening, we cannot exclude the occurrence of homeostatic plasticity phenomena, a natural neuron mechanism that reduce neuron's activity to prevent overstimulation and cells damage [42]. Indeed, the combination of M1 activation induced by motor grip and PA might have caused a suppressive effect and a consequent hand strength reduction [43]. In particular, a previous study has shown that PA excitatory effects may be reversed whether they are administered immediately after a conditioning excitatory paradigm of transcranial direct current stimulation [28]. Further studies combining behavioural with neurophysiological measures of M1 activation (i.e. analysis of motor evoked potentials) could better clarify this issue.

In addition to hand strength reduction, we found a selective plantar pressure forward displacement after r-PA but not l-PA. This result adds evidence to a previous study [7] reporting a forward displacement of the center of pressure (CoP) after both l-PA and r-PA, showing that r-PA may induce a shift either of the vertical projection of the center of pressure (as measured with stabilometry) and of the plantar pressure in terms of interaction between feet and ground reaction force (as measured with baropodometry) [9]. However, unlike Michael et al., we did not find an effect on plantar pressure after l-PA. Besides differences in the measurements (baropodometry vs. stabilometry), a methodological issue may account for this lack of result. Namely, in our study subjects performed an additional pointing task to measure PA after effect (blind post-exposure) [44]. Since subjects were prevented to watch their arm moving in order to adjust the pointing bias, an access to body postural representation was probably strongly needed [45]. We may speculate that the blind post-exposure caused a stronger modulation of the right hemisphere in order to retrieve internal and extra-personal body space representation [24] and to correct for the arm shift. Activation of the right hemisphere is potentiated after r-PA but not after l-PA (inducing left hemispheric activation). This could explain the plantar pressure displacement selectively observed following right PA. This hypothesis finds confirmation in previous studies showing that body sway and body weight distribution among feet are regulated by the internal body representation, linked to attentive process [7,24] taking place mostly in the right brain hemisphere [21,22,31].

These findings suggest that PA may induce the recalibration of representation of space [46] and of body space [7]. Further studies might explore the link between the direction of the PA induced shift in body posture and PA deviation side.

In conclusion, our results suggest that PA exerts effects on body posture and hand strength relying on different mechanisms. The PA effects on hand strength would be related to modulation of interhemispheric inhibition of sensorimotor processes, involving both hemispheres. On the other hand, the PA effects on body posture would be related to modulation of higher-level processes such as body representation, involving mainly the right hemisphere.

Conflict of Interest: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Disclosure: The work involves human subjects and it has been carried out in accordance with The Code of Ethics of the World Medical Association (https://www.wma.net/policies-post/wma-declaration-of-helsinki-ethicalprinciples-for-medical-research-involving-human-subjects/) (Declaration of Helsinki) for experiments involving humans.

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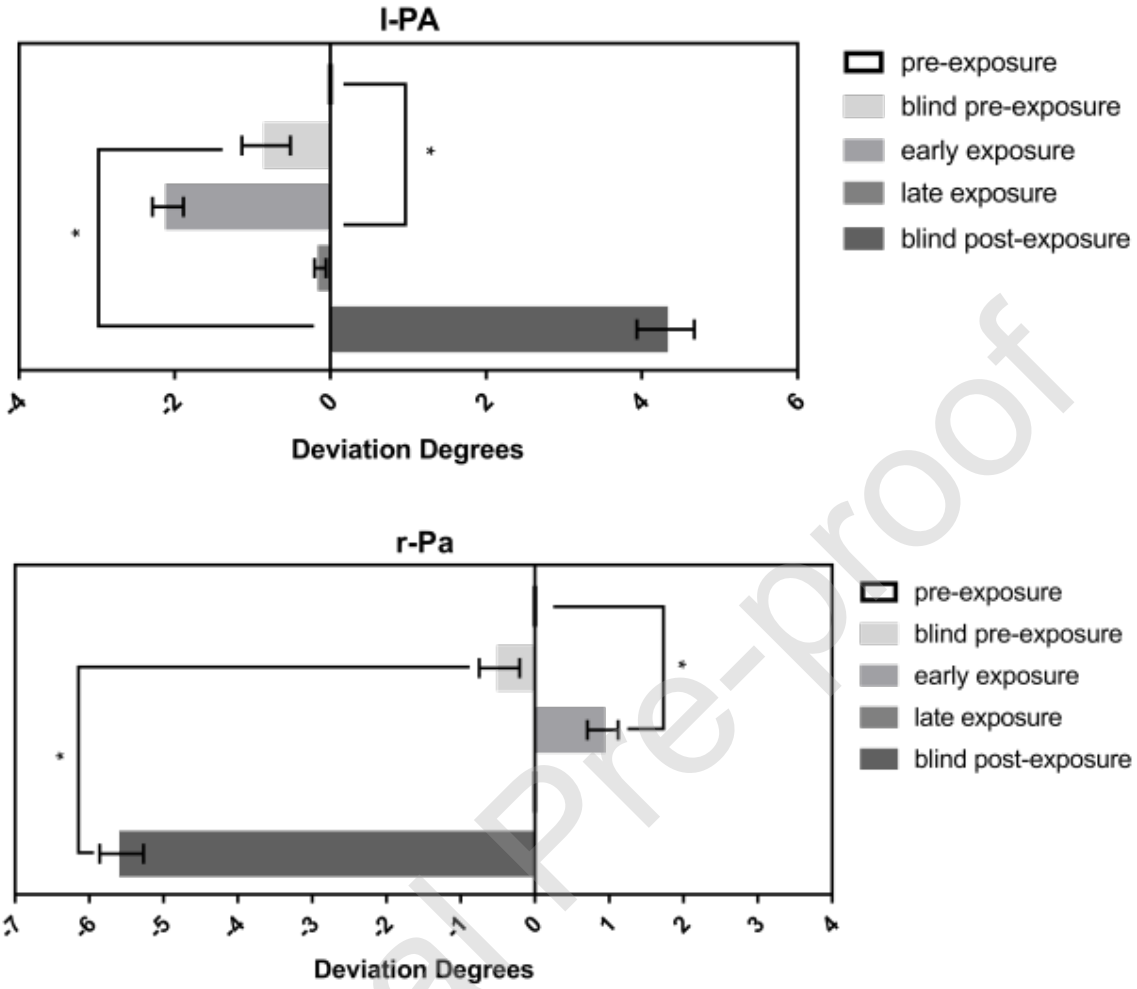
Table 1. Sample Demographic characteristics

	l-PA group (n=23)	r-PA group (n=23)
Age (years)	25±1.87	26±3.92
Years of education	17±1.27	17±1.85
Handedness	62%±0.23	66%±0.2
Weight (kg)	63.52±13.36	62.05±11.41
Height (cm)	167.65±10.55	170±9.85

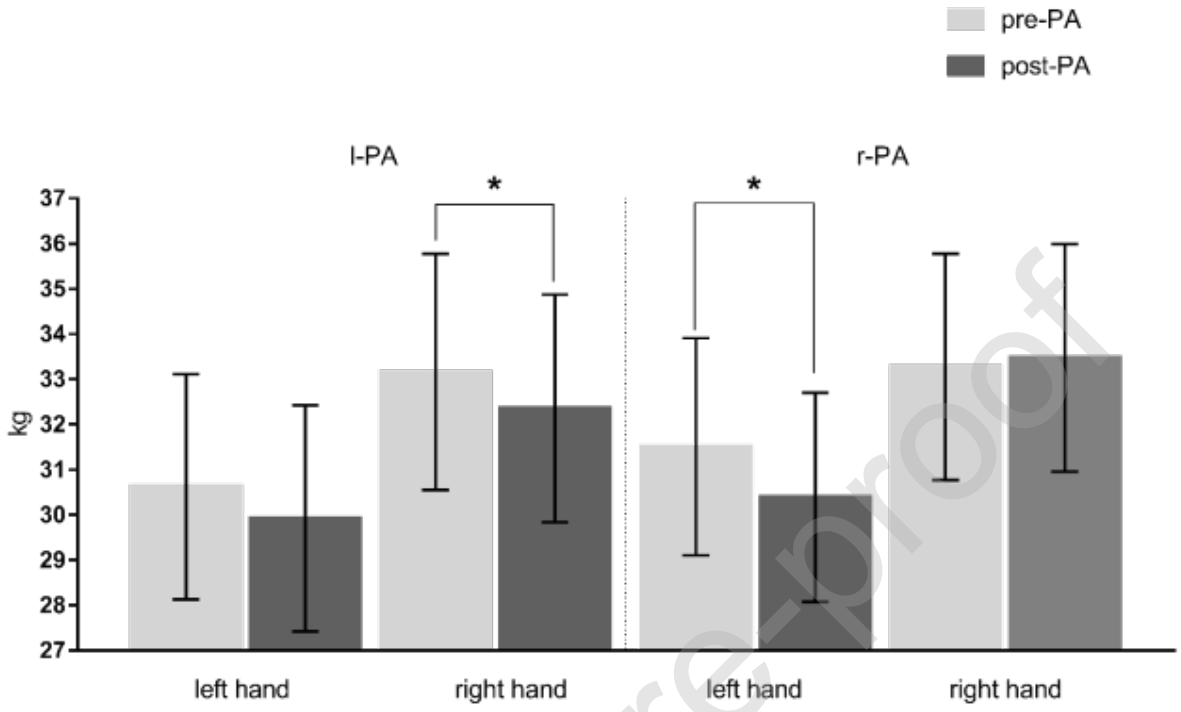
Legend: l-PA = leftward prismatic adaptation group. r-PA = rightward prismatic adaptation group

Figure 1. Mean pointing displacement during Prismatic Adaptation in the five experimental conditions across groups (leftward prismatic adaptation group and rightward prismatic adaptation group).



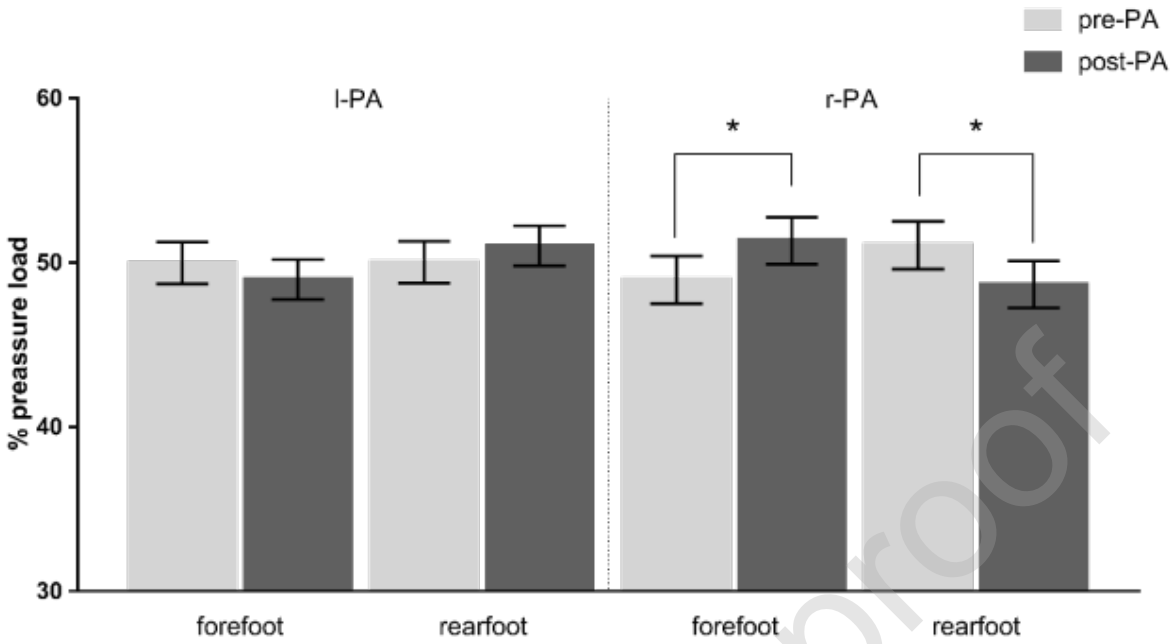
Legend: l-PA= leftward prismatic adaptation group; r-PA= rightward prismatic adaptation group;
*Error bars= Standard error of mean; *p<.001*
Negative values indicate leftward pointing displacement, positive values indicate rightward pointing displacement.

Figure 2. Differences in handgrip strength (left and right): mean values before and after prismatic adaptation (pre- PA, post-PA) across groups (leftward prismatic adaptation group and rightward prismatic adaptation group).



*Legend: l-PA = leftward prismatic adaptation group; r-PA = rightward prismatic adaptation group; pre-PA= before prismatic Adaptation; post-PA= after prismatic adaptation; Error bars = Standard error of mean; * p<.05.*

Figure 3 Plantar pressure distribution (forefoot/rearfoot) before and after prismatic adaptation (pre- PA, post-PA) across groups (leftward prismatic adaptation group and rightward prismatic adaptation group).



*Legend: l-PA = leftward prismatic adaptation group; r-PA = rightward prismatic adaptation group; pre-PA= before Prismatic Adaptation; post-PA= after Prismatic Adaptation; Error bars=Standard error of mean; * p<.05.*