

Effect of dysmorphic plantar arch on venous hemodynamics of the lower limb.

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Background: The role of the plantar venous pump (PVP) on venous return is evident but the effects of the foot morphology have never been characterized properly.

Method: 52 healthy volunteers – 26 with normal plantar arch (control) and 26 with dysmorphic plantar arch (in two subgroups: 13 flat feet, 13 hollow feet) – were included. Using Doppler ultrasound, we measured the diameter and the peak systolic velocity in the large veins of the lower limb after PVP stimulation by manual compression and bodyweight transfer.

Result: The mean diameter of the studied veins varied from 1.9mm to 2.8mm in the control group and from 1.9mm to 2.7mm in the dysmorphic plantar group. The foot arch morphology did not affect significantly the venous blood flows, except in the great saphenous vein during manual compression.

Conclusion: PVP stimulation results in an increase of venous blood velocity independently of the plantar morphology.

Keywords: foot venous pump, foot static disorders, flat foot, hollow foot, peak systolic velocity

INTRODUCTION

Venous return from the lower limbs to the heart is supported by three mechanisms called venous pumps: (i) the thigh pump, (ii) the calf pump, and (iii) the foot pump. The dysfunction of one of these pumps is related to Chronic Venous Insufficiency CVI of the lower limbs since it leads to venous hypertension, parietal distension, and excessive venous stasis in the legs [1]. Despite the negative impact of CVI on the quality of life and although venous stasis in the legs can result in serious long-term complications, such as skin damage [2] or deep venous thrombosis [3], data concerning the prevalence of CVI remain scarce.

The venous pump of the foot is the most distal contributor to venous return from the lower limbs to the heart. Regarding the anatomical and functional organization of the foot venous system, Uhl [4]–[6] and Horwood [7]) described two different groups. The medial part includes the medial marginal vein on the dorsal foot connected by 3 or 4 perforating veins to medial plantar veins (deep in the arch). The second lateral part is mainly composed of the lateral pedicle formed by long and large lateral plantar veins joining medial plantar ones at the level of the calcaneal confluent to form the posterior tibial veins. The deep lateral venous system has few or even no connection with the superficial marginal lateral system.

Although differences exist regarding the vessel dimensions, this anatomical-functional organization was confirmed by Corley *et al.*[8] and Ricci *et al.*[9]. Moreover, unlike what F. Lejars suggested in 1890, Uhl [3,4,5] demonstrated that, strictly speaking, the foot venous pump is essentially intermuscular and located deeply at the level of the foot arch and can be called Plantar Venous Pump PVP. Indeed, it is mainly composed of deep plantar lateral and medial veins which constitute a blood reservoir of about 15 to 25 mL [5]. Uhl, Corley *et al.*, Ricci *et al.*, and others studying PVP all showed that it empties at each gait cycle with weight-bearing and plantar muscle contractions following these 3 steps: (1) high compliance of large caliber plantar veins as well as relaxation of plantar muscles allow PVP filling during the swing phase of the gait. (2) Then, the contact of plantar sole with the ground during stance phase permits a first direct compression of the PVP which empties into tibial and saphenous veins. (3) At the end of the stance phase during propulsion, the support is on the tip of the foot. Toe flexion causes plantar muscle contraction allowing a second compression of the PVP which continues to fill. Therefore, the PVP is at the origin of venous return in lower limbs, before being relayed by the calf muscle pump.

If the function of the foot pump is now well described, its hemodynamic performance remains poorly understood. Indeed, in most studies, PVP was activated through different compression techniques such as muscular exercises, intermittent pneumatic compression IPC, or micro-mobile foot compression device. But hemodynamics was measured in popliteal and posterior tibial veins, or in the common femoral vein [10]–[14]. The venous blood flow at this level is strongly affected by the calf pump. It is crucial to measure venous flows directly at the outlet of the foot in the ankle to uncouple PVP from calf pump effects.

It remains unclear if the PVP function depends on the foot morphology. 77% to 85% of the population have a physiological foot [16]–[18]. This normal morphology is characterized by a footprint with support on digitigrade toe pad, on all the metatarsal pallet, on external edge of midfoot, and on heel (Figure 1.A). This physiological morphology ensures optimal support and locomotion. It serves as basis to specialists to define two types of Foot Static Disorders (FSD), which are flat and hollow foot, and assess their severity (three stages). Flat foot is characterized by several clinical signs including [19], [20]: (i) a calcaneal valgus, (ii) spread of the forefoot, and (iii) ligamentous hyperlaxity and excessive midfoot pronation leading to a flattening of plantar arch and concave external edge. The most important is flattening, especially at the midfoot (Figure 1.B). Hollow foot is the opposite (Figure 1.C): (i) excessive plantar concavity, (ii) reconciliation of anterior and posterior supports resulting in reduction of bearing surface and foot shortening, (iii) joint stiffness and muscular hypertonus. The prevalence of flat foot in adults is between 10% and 23% [17], [18], [21]–[23], whereas hollow arch concerns about 10% of the population [18], [21], [24]. These FSD were shown to reduce the PVP efficiency [25], [26]. However, these studies have been conducted on CVI patients where venous return is already impaired. It should be assessed whether flat and hollow foot disorders also affect PVP efficiency in healthy subjects.

Given this state of the art, the objectives of the study are: (1) to quantify PVP hemodynamic performances in the ankle with and without FSD (flat and hollow feet), and (2) to assess the potential effect of foot morphology on PVP efficiency in both groups.

For such quantifications, the study will focus on the weight bearing phase of the PVP and, hence, disregarding the dynamic effects due to foot impulsion. Therefore, measurements will be carried out in static position, which facilitates acquisition and repeatability of the measurements.

METHOD

Subjects

For this controlled, prospective, non-randomized, multicenter, open-label clinical study, 52 healthy volunteers were recruited. Each participant satisfied the inclusion criteria having between 18 and 60 years-old, no venous blood flow disorder, no lower limb dermatological problem, no orthostatic hypotension, no history of lower extremity fracture in the last 6 months, no regular use of orthopedic insoles in the last 2 years, no stage 3 of FSD, no medium or long-haul flight (more than 3h30) the week before the measurements and no pregnancy or breastfeeding situation. One limb per participant was studied. They were divided into two groups: the control group with 26 physiological feet and the “dysmorphic plantar” group with 13 hollow feet (subgroup 1) and 13 flat feet (subgroup 2). Ethical approval for the study was granted by the North-West III Research Ethics Committee of the University Hospital of Caen (Normandie, France) and all subjects signed informed consent.

Measurements

The Chippaux-Smirak’s index CSI was chosen to determine the morphology of the foot, which is defined as the ratio between the largest and smallest width of the foot as estimated from podographic measurements of foot-prints. CSI is non-invasive, easy to do from a static footprint and it correlates well with plantar arch parameters (Maes et al. [18]). The measurement of this podometric parameter was performed during a first visit by podiatrists using podograph (CAPRON podologie, France) to obtain footprints. The CSI ranges are reported in Table 1.

During the evaluation visit, the subjects were asked to stand barefoot on a phlebology stool. A color pulsed Doppler echography (Mylab™ 25Gold, Esaote SpA, Italy) with linear probes between 5 and 18 MHz, was performed by the phlebologist. Diameter (mm) of one foot vein (the first metatarsal interspace P1MI) and of four other veins at ankle level (posterior tibial vein PTV, anterior tibial vein ATV, great saphenous vein GSV, and small saphenous veins SSV) was collected. Then, peak systolic velocity V_{max} (cm/s) in each vein was measured during two different compressions of PVP activating venous return: (i) manual compression (MC) applied by phlebologist under the plantar arch, (ii) bodyweight transfer (BT) for which participants were asked to put all their weight on the opposite foot, then transfer all support on the studied foot and release.

Statistical analysis

Anthropometric data (gender, age, weight, and body mass index BMI) of the sample population are reported in Table 2. Mean and standard deviation (SD) were used for the descriptive analysis. A differential analysis between groups allowed assessing potential effect of FSD on PVP efficiency. In all the analyses, $p < 0,05$ (with a 90% confidence interval) was considered as statistically significant. First, Shapiro-Wilk test was performed to verify normality distribution for the outcome measurements. After checking whether the two groups were comparable in terms of age and weight, unpaired non-parametric tests were achieved (Welch’s t-test if normal distribution and Mann & Whitney’s U-test otherwise).

RESULTS

52 healthy volunteers, 20 male and 32 female, aged from 20 to 59 years-old (mean 39 years-old) were included. Over the entire sample population, the average BMI was between 18,5 and 24. Nevertheless, individuals included in the flat feet subgroup were slightly overweight (between 25 to 29.9). According to the Welch t-test results, control and dysmorphic plantar groups were similar in terms of age and weight (respectively $p=0,08$ and $p=0,32$).

The vein diameters (mm) are reported in Table 3. P1MI and ATV had the smallest caliber in both groups. Then came in crescent order: PTV and SSV, and finally GSV the larger one. Caliber could not be evaluated only in one subject for 3 of the 5 veins.

Mean, SD, p-value of differential analysis, and missing data regarding peak systolic velocity V_{max} (cm/s) during manual compression and compression by bodyweight transfer are respectively reported in Table 4 – Figure 2, and Table 5 – Figure 3. Mean manual compression V_{max} varied from 9.6cm/s in SSV and ATV to 26.9cm/s in P1MI in the control group. V_{max} were similar (from 8.2cm/s in SSV to 23.1cm/s in P1MI) in the dysmorphic plantar group. The mean $V_{max_{MC}}$ measured in GSV was significantly lower in the FSD group ($n=26$) than in the control group ($n=26$).

Bodyweight transfer compression generated higher V_{max} than manual PVP activation. Mean $V_{max_{BT}}$ varied from 12.2cm/s in ATV to 41.7cm/s in P1MI in control group. Mean $V_{max_{BT}}$ varied from 10.9cm/s in ATV to 39.1cm/s in P1MI in the dysmorphic plantar group. In this condition of compression, no significant difference was statistically observed.

Comparing the two groups ($n=26$ per group), the mean $V_{max_{MC}}$ measured in GSV was significantly lower in case of FSD.

DISCUSSION

Quantification of PVP performances

The veins ranked in decreasing order of V_{max} such as: P1MI, PTV, GSV, SSV, and ATV. This order was observed in both groups and regardless the compression method (manual or bodyweight transfer). These results are consistent with anatomical data since lateral and medial plantar veins are connected to P1MI in the forefoot and PTV in hindfoot [4], [6], [9], [16], [27]. Thus, compressed plantar veins results in an increase of blood flow especially in these two veins.

White *et al.* performed a 130mmHg IPC and a below knee Doppler ultrasound of PTV and ATV in healthy subjects. They measured a mean V_{max} of 123 ± 71 cm/s in PTV and 24 ± 14 cm/s in ATV [28]. In Dohm *et al.* [29], foot compression with IPC (130mmHg) and micro-mobile device (314mmHg) in healthy volunteers caused respectively a mean V_{max} of 57,9cm/s and 75,2cm/s in PTV, 6cm above the medial malleolus. Broderick *et al.* [10] measured, within a healthy population, the V_{max} in PTV during four difference exercises as bodyweight transfer from a foot to the other (57 ± 6 cm/s), and toe flexion (40 ± 14 cm/s). V_{max} of 22 cm/s at rest and of 123 cm/s during foot and calf IPC was measured in GSV at the level of the knee by Labropoulos *et al.* [30] in healthy subjects in bent knee sitting position. Their V_{max} values were 2 to 5 times superior to the ones measured in the present study. This could be explained by several reasons. First, in the present study, the volunteers were in standing position, whereas measurements were done in inversed Trendelenburg position in Dohm *et al.* or in bent knee sitting position in Labroupolos *et al.* In these conditions, gravity has less impact on venous return and there is little or no support on feet. As a result, the plantar venous reservoir was probably more dilated and filled at start, possibly allowing more venous emptying (velocity and quantity) during plantar compression. Secondly, in our study, levels of PVP compression may not have reached 130mmHg or 314mmHg. Despite the use of interface pressure sensor to control the applied force during manual

compression, this force could not be maintained homogenous for all the subjects. Likewise, volunteers performed bodyweight transfer from the opposite foot to the studied one more or less quickly and with more or less intensity. In the future, these maneuvers should be standardized as well as the duration and intensity of the compression using the pressure sensor to set a threshold. Finally, it should be noted that Labropoulos, Broderick et White, performed measurements at the knee level. Moreover, in the first two studies, the calf pump was directly activated: by IPC in Labropoulos, and because calf muscles are also activated during bodyweight transfer and toe flexion in Broderick. In the third, the calf pump was not directly stimulated. Nevertheless, it probably had a baseline activity. Thus, assessing only PVP activity requires measurements at the outlet of the foot. Collecting data higher in the leg with even unintended calf muscle stimulation could explain their higher results.

Chauveau *et al.* [31] numerically evaluated the effect of compression stockings on virtual patients, healthy and with superficial varicose veins. After simulating a tiptoe maneuver, their model calculated a 1.8 cm/s Vmax in GSV at the level of the ankle in healthy virtual subject. Although it is much lower than values obtained in the present study, this difference may be explained as PVP was directly activated with plantar compression here, whereas tiptoe maneuver was used in their study.

As mentioned in the introduction, the study has focused on the weight bearing phase of the PVP and, hence, has disregarded the dynamic effects due to foot impulsion. The obtained results can be seen as a surrogate of the PVP activation during walk. Future studies should consider also the foot impulsion phase which will inevitably involve more complex measurements.

Impact of FSD on PVP efficiency

FSD does not seem to influence the caliber of the studied veins. We did not necessarily expect a difference between the two groups because all volunteers were healthy without any venous disease that could have altered venous wall. Moreover, the large inter-individual anatomical variability of the venous system does not allow to predict any difference in caliber of veins. The obtained values are consistent with literature. Van Bemmelen *et al.* [32] measured, for example, diameter going from 1,2mm to 4,3mm for some foot and low calf veins.

There is yet a trend showing modified PVP performances in case of FSD compared to control group. Indeed, Vmax measured by manual compression in GSV is significantly lower in « plantar dysmorphies » group ($p=0.04$). With equivalent diameter as explained earlier, and a lower Vmax, the venous blood flow may be reduced in GSV in case of FSD.

To go further in the analysis, we randomly selected 13 subjects of the control group to compare with the sub-groups “hollow feet” and “flat feet”. Again, Vmax by manual compression was significantly lower in GSV of the “flat feet” subgroup than in the 13 selected normal feet ($p=0,04$). This corroborates the study from Potério-Filho *et al.* [33]. They compared venous pressure in healthy normal foot and knee individuals with flat feet and genu valgum healthy subjects, during 2 exercises: walk and tiptoe maneuver, with and without high-heeled shoes. They found that venous pressure was significantly higher in flat feet with genu valgum, reflecting a less efficient venous return compared with control group. No significant difference was observed between the 13-subjects control group and “hollow feet” subgroup. Comparing the two subgroups with FSD, hollow arch morphology seemed to alter PVP performances because Vmax in GSV with both types of compression and in PTV during bodyweight transfer were significantly lower in hollow feet (respectively $p=0,04$, $p=0,02$ et $p=0,04$). An excessive concavity of the plantar arch could decrease the PVP efficiency more than its flattening. People with hollow feet could have more risk to develop venous insufficiency than people with flat feet. Moreover, in the study from Uhl *et al.* [21], in which the relationship between FSD and CVI was evaluated, hollow

feet represented the majority (16,6%) of the 31% of lower limbs having FSD. However, as this sub-analysis was achieved on small samples (n=13 for each group), these results should be confirmed on a larger sample size. It should also be noticed that FSD do not only affect the PVP, but also have an effect on the global venous return. Specifically, the soleus muscle contraction is affected by FSD. Such contraction, in turn, is the main responsible to peak systolic velocity in the PTV during weight transfer. Such cross effects are difficult to isolate but could be investigated separately in future studies.

It was observed that modifications of PVP hemodynamics in feet with FSD were remarkable at the GSV level. If these individuals develop varicose or CVI, it will more likely appear at the level of this vein. According to the foot venous system proposed by Uhl et al. [4], GSV drains the medial compartment, i.e. medial plantar veins, perforating veins (P1MI, medial ones), and medial marginal vein. Therefore, plantar FSD would first reduce hemodynamics performance of these veins.

Other differences between groups are not significant. This could be explained by the distribution of subjects into the two groups. Indeed, CSI was used to standardize it and define FSD stage when needed. Nevertheless, podiatrists observed that their experience-based diagnosis was not always in agreement with measured CSI, especially for some flat or hollow feet with a CSI closed to normal score. It would be interesting in a future study to consider podiatrist diagnosis for group distribution. Non-significant differences could also be explained by the physical activity of participants, as most of the volunteers had moderate to intense physical activity, which may improve venous return [34]–[36]. This study, it could have blunted the negative effects of plantar dysmorphism on PVP performances. It would be wise to consider physical activity (number of hours per week for example) as a variable in a future study.

CONCLUSION

We report in this article the first clinical study assessing the PVP function in volunteers with and without FSD. The main result is that the compression of plantar veins in the PVP has a significant effect on venous return. Blood velocities were increased especially in the perforating vein of the first metatarsal interspace and in the posterior tibial vein, which are the main outlets of medial and lateral plantar veins. Another very important results is the significant reduction of the blood velocity in the great saphenous vein for FSD subjects versus controls. These results should still be confirmed on a larger cohort, possibly including volunteers with more pronounced FSD.

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Table 1: Range of Chippaux-Smirak's index

	Stage 3	CSI < 15%
Hollow foot	Stage 2	15% ≤ CSI < 20%
	Stage 1	20% ≤ CSI < 30%
	Normal foot 30% ≤ CSI ≤ 40%	
	Stage 1	40% < CSI ≤ 50%
Flat foot	Stage 2	50% < CSI ≤ 60%
	Stage 3	CSI > 60%
		Stage 3
Hollow foot	Stage 2	15% ≤ CSI < 20%
	Stage 1	20% ≤ CSI < 30%
	Normal foot 30% ≤ CSI ≤ 40%	
	Stage 1	40% < CSI ≤ 50%
Flat foot	Stage 2	50% < CSI ≤ 60%
	Stage 3	CSI > 60%

Table 2: Anthropometric data of the sample population

GROUP	GENDER		Mean \pm SD (range)		
	FEMALE	MALE	AGE (years)	WEIGHT (kg)	BMI (kg/m ²)
TOTAL (n=52)	32 (62%)	20 (38%)	39.0 \pm 10.8 (20 – 59)	67.9 \pm 11.4 (42.1 – 96.2)	23.3 \pm 3.2 (16.0 – 30.7)
CONTROL (n=26)	16	10	36.4 \pm 10.5 (20 – 58)	66.4 \pm 10.3 (42.1 – 90.7)	22.7 \pm 2.8 (22.7 – 2.8)
PLANTAR DYSMORPHIES (n=26)	16	10	41.6 \pm 10.6 (21 – 59)	69.5 \pm 12.4 (44.6 – 96.2)	23.9 \pm 3.5 (16.0 – 30.7)
HOLLOW FEET (n=13)	10	3	42.3 \pm 11.1 (21 – 56)	64.2 \pm 13.4 (44.6 – 92.4)	22.4 \pm 0.8 (16.0 – 28.7)
FLAT FEET (n=13)	6	7	40.9 \pm 10.5 (29 – 59)	74.9 \pm 8.8 (63 – 96.2)	25.2 \pm 2.6 (22.5 – 30.7)

Table 3: Diameter (mm) of P1MI, PTV, GSV, SSV, and ATV in standing position

Veins	Control (n=26)		Plantar dysmorphies (n=26)		P-value	Missing data
	Diameter (mm)		Diameter (mm)			
	Mean	SD	Mean	SD		
P1MI	1.9	0.3	2.0	0.3	0.28	1
PTV	2.4	0.6	2.3	0.4	0.59	1
GSV	2.8	0.8	2.7	0.9	0.53	1
SSV	2.5	0.6	2.3	0.6	0.29	0
ATV	1.9	0.5	1.9	0.5	0.72	0

Table 4: Peak systolic velocity Vmax (cm/s) in healthy subjects with and without FSD during manual compression

Veins	Control (n=26)		Plantar dysmorphies (n=26)		P-value	Missing data
	Vmax _{MC} (cm/s)		Vmax _{MC} (cm/s)			
	Mean	SD	Mean	SD		
P1MI	26.9	16.5	23.1	13.4	0.35	7
PTV	19.7	12.1	22.3	13.7	0.53	3
GSV	15.6	6.4	12.0	4.6	0.04*	2
SSV	9.6	5.5	8.2	4.4	0.62	2
ATV	9.6	5.7	8.3	4.1	0.43	3

Table 5: Peak systolic velocity Vmax (cm/s) in healthy subjects with and without FSD during bodyweight transfer

Veins	Control (n=26)		Plantar dysmorphies (n=26)		P-value	Missing data
	Vmax _{BT} (cm/s)		Vmax _{BT} (cm/s)			
	Mean	SD	Mean	SD		
P1MI	41.7	30.3	39.1	27.7	0.30	3
PTV	22.7	18.7	21.4	14.6	0.71	1
GSV	19.1	10.1	20.8	10.4	0.59	1
SSV	16.8	11.1	13.5	8.9	0.35	2
ATV	12.2	6.8	10.9	9.2	0.20	2

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Figure 2: Peak systolic velocity during manual compression of PVP in P1MI, PTV, GSV, SSV, and ATV

Figure 3: Peak systolic velocity during bodyweight transfer compression of PVP in P1MI, PTV, GSV, SSV, and ATV

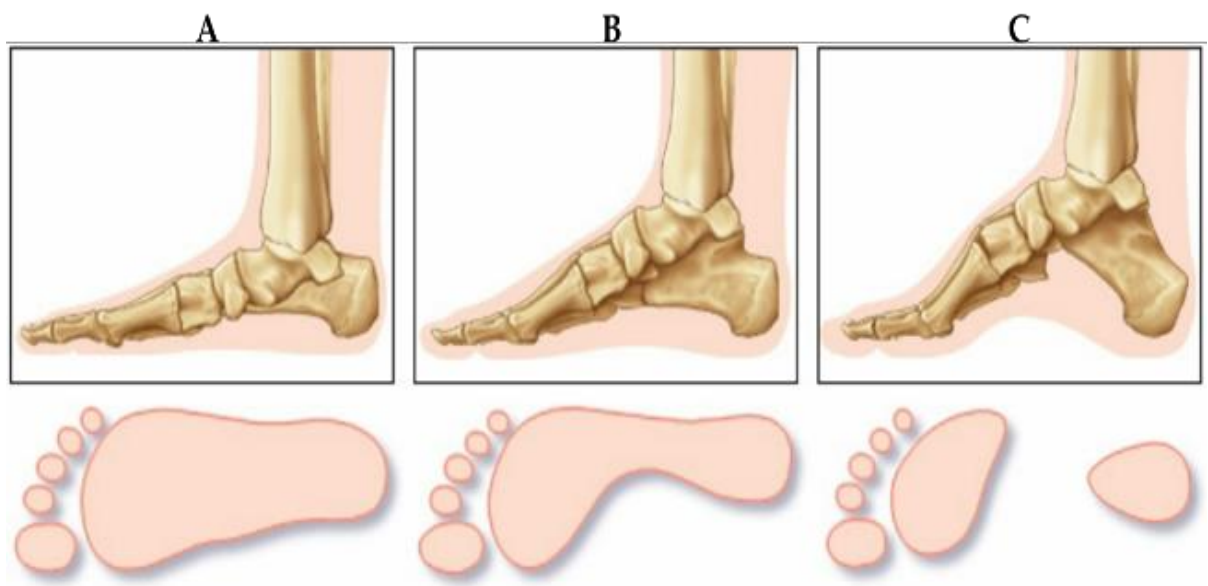


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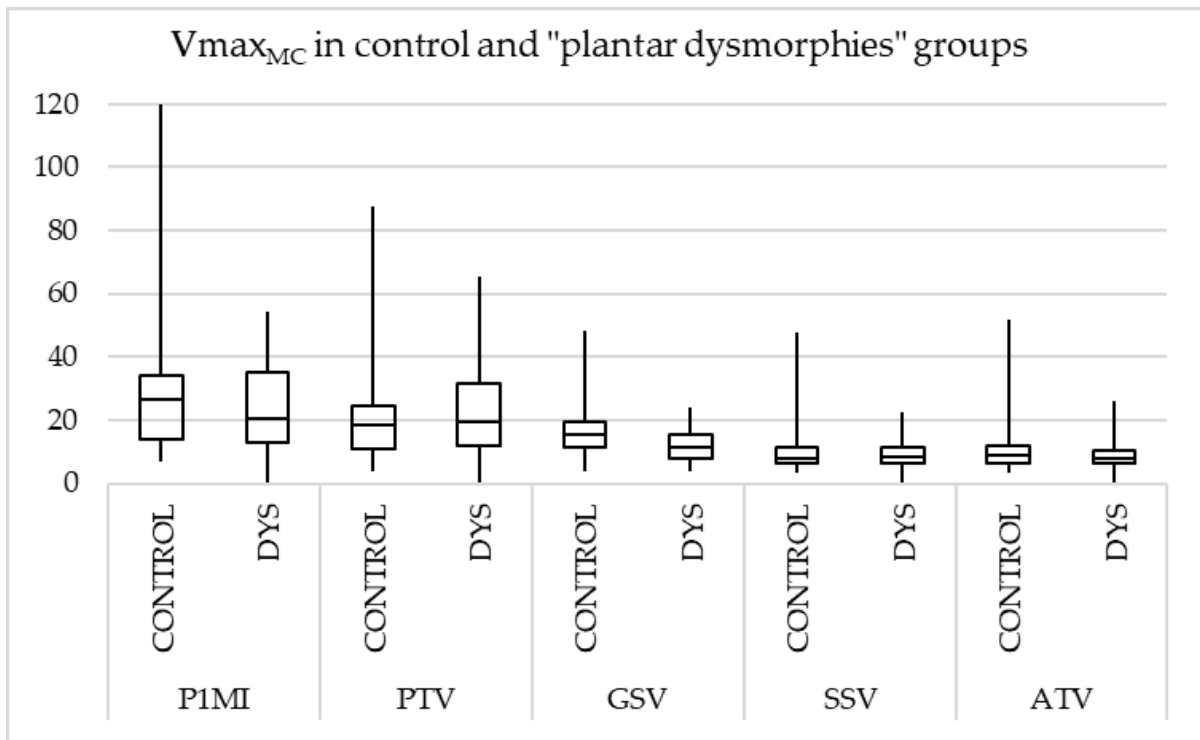


Figure 2: Peak systolic velocity during manual compression of PVP in P1MI, PTV, GSV, SSV, and ATV

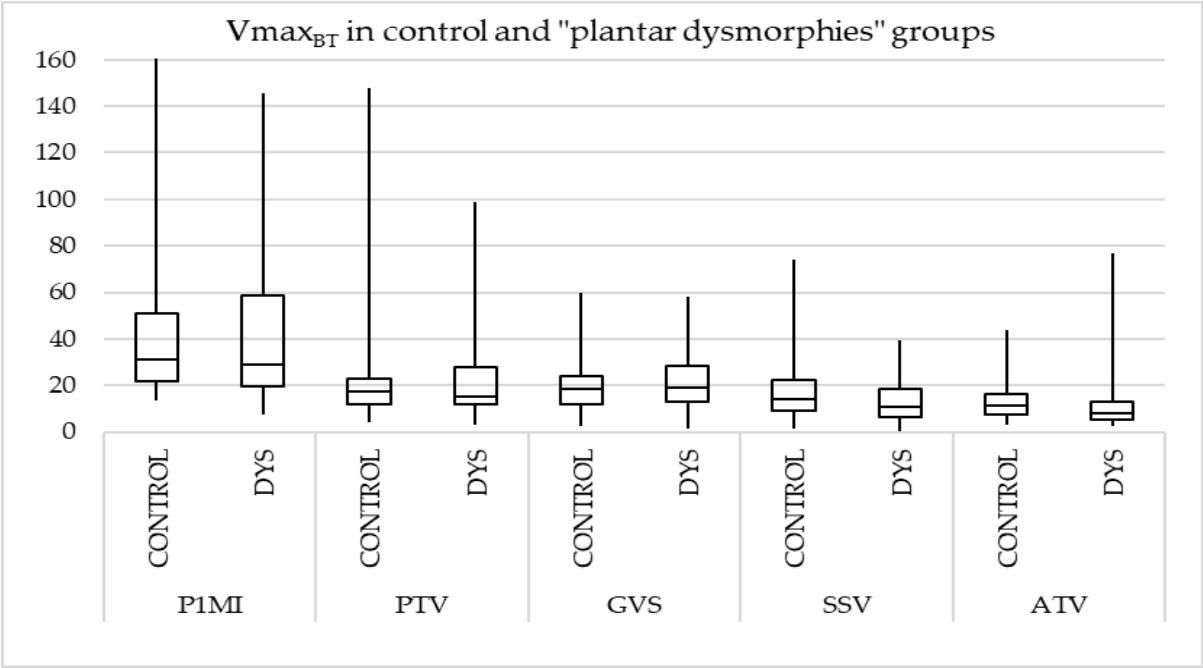


Figure 3: Peak systolic velocity during bodyweight transfer compression of PVP in P1MI, PTV, GSV, SSV, and ATV