

# Sport-specific patterns of vascular compression in athletes with entrapment syndrome: A comparative analysis of endurance versus non-endurance athletes

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## Abstract

Vascular entrapment syndromes of the lower extremities represent a diagnostic challenge in athletes, with emerging evidence suggesting sport-specific presentations. This study aimed to characterize and compare patterns of vascular compression, anatomical variants, and hemodynamic consequences across different sporting disciplines.

We conducted a retrospective analysis of 210 athletes with confirmed vascular entrapment syndrome from a 10-year cohort (2014-2023). Athletes were categorized as long-distance runners (n=87), cyclists (n=71), triathletes (n=24), soccer players (n=19), and other sports (n=9). All participants underwent standardized clinical assessment and vascular imaging during provocative maneuvers. Multivariable analyses and latent class analysis were performed to identify independent predictors and distinct phenotypes.

Sport-specific patterns of vascular compression were identified. Long-distance runners exhibited highest compression during plantarflexion ( $85.7 \pm 8.6\%$ ), predominantly with Type III gastrocnemius variants (60.9%). Cyclists demonstrated highest compression during knee extension ( $79.2 \pm 11.4\%$ ), with predominant Type II variants (49.3%). Soccer players exhibited primarily Type I variants (57.9%) with lower compression severity. Latent class analysis identified three distinct phenotypes: “runner phenotype” (43.8%), “cyclist phenotype” (37.1%), and “low compression phenotype” (19.0%), each with characteristic anatomical, hemodynamic, and clinical features. Sport category remained an independent predictor of compression severity after adjusting for potential confounders ( $p < 0.001$ ).

Vascular entrapment syndrome manifests with distinct sport-specific patterns reflecting the interplay between anatomical predisposition and functional demands. The identification of sport-

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## Introduction

Vascular entrapment syndromes of the lower extremities represent a spectrum of anatomical and functional disorders. These disorders are characterized by extrinsic compression of vascular structures by surrounding musculotendinous or fibrous elements (Turnipseed, 2002; Williams et al., 2015). Among these conditions, popliteal artery entrapment syndrome (PAES) has emerged as a clinically significant entity in athletic populations. The estimated prevalence ranges from 0.17% to 3.5% depending on diagnostic criteria and study population (Gokkus et al., 2014; Levien & Veller, 1999).

The syndrome results from compression of the popliteal artery during plantar flexion or knee extension. This compression is most commonly due to anomalous relationships between the artery and the medial head of the gastrocnemius muscle, aberrant fibrous bands, or hypertrophied surrounding musculature (Sinha et al., 2012; Turnipseed & Pozniak, 1992). Despite advances in diagnostic imaging and increased awareness among sports medicine specialists, these syndromes remain diagnostically challenging. Recent systematic reviews report median diagnostic delays of 24-35 months (Hislop et al., 2014; Mosley & Grotewold, 2014).

### Prevalence Across Different Sports

Contemporary studies demonstrate that vascular entrapment conditions disproportionately affect young, physically active individuals during their peak performance years. The highest reported incidence occurs among endurance athletes (2.5-3.8% in long-distance runners), military personnel (1.2-2.4%), and team sport participants (0.5-1.3%) (Apigian & Landry, 2015; Fowkes et al., 2013). A striking sport-specific distribution has been observed. Emerging data suggest variability in both clinical presentation and underlying anatomical patterns across different athletic disciplines (Anil et al., 2018; Gaunder et al., 2017).

Recent literature indicates that functional popliteal artery entrapment syndrome (fPAES) shows distinct prevalence patterns. Cycling-related vascular entrapment occurs in approximately 1.8-2.4% of competitive cyclists (Doyle & Lipman, 2020). Running-related entrapment affects 2.5-3.2% of endurance runners. Team sports demonstrate lower overall prevalence rates of 0.4-0.8% (Bianchi et al., 2018).

The pathophysiological consequences of untreated vascular entrapment are progressively destructive and potentially career-ending for athletes. Dynamic arterial compression initiates a cascade of vascular pathology. Endothelial injury leads to intimal hyperplasia and premature atherosclerosis. This ultimately progresses to arterial stenosis, thrombosis, or distal embolization in advanced cases (Liu et al., 2014). Advanced imaging studies have demonstrated stress-induced endothelial dysfunction and altered flow dynamics in compressed arterial segments, even in asymptomatic individuals with anatomical variants (Pillai, 2008; Sinha et al., 2012).

### Biomechanical Considerations

The biomechanical forces acting upon the neurovascular structures of the lower extremity differ substantially across sporting disciplines. Quantitative biomechanical analyses have demonstrated that runners experience peak forces of 2.5-3.0 times body weight during the push-off phase. This involves repetitive ankle plantarflexion and knee extension under load (Bolin, 2019). In contrast, cyclists maintain relatively fixed knee and ankle positions with repetitive motion patterns and sustained muscular contractions. This generates different patterns of neurovascular compression (Becher et al., 2020).

Team sport athletes undergo unpredictable multi-directional movements with variable loading intensities. This creates complex and intermittent compression profiles. These sport-specific biomechanical patterns likely contribute to the observed variations in presentation, severity, and anatomical distribution of vascular entrapment syndromes.

### Literature Selection and Study Limitations

Important methodological note: This study represents a retrospective clinical analysis rather than a systematic review. We did not conduct a systematic literature search using predefined search strategies or systematic inclusion/exclusion criteria. The referenced literature was selected based on clinical relevance and expert knowledge in the field of vascular entrapment syndromes. This approach allows for comprehensive clinical analysis but limits the systematic nature of our literature review component.

Previous research has examined various aspects of exertional leg pain in athletes, including differential diagnostic approaches for distinguishing arterial entrapments from other causes (Pham et al., 2007). Building on this foundation, we conducted a

10-year retrospective cohort study of 1,214 athletes with exertional lower limb symptoms. We identified five independent predictors of vascular entrapment syndrome: male sex (adjusted OR 2.21, 95% CI 1.39-3.51), weekly training  $\geq 10$  hours (adjusted OR 1.88, 95% CI 1.21-2.92), symptom duration  $>6$  months (adjusted OR 2.93, 95% CI 1.76-4.88), bilateral symptoms (adjusted OR 2.15, 95% CI 1.23-3.76), and abnormal gastrocnemius insertion on imaging (adjusted OR 3.66, 95% CI 2.14-6.27).

### Study Aims

The present study aims to characterize and compare sport-specific patterns of vascular compression in athletes with confirmed entrapment syndrome. We analyze: 1) dynamic imaging findings across different provocative maneuvers, 2) anatomical variants and their relationship to compression severity, 3) hemodynamic consequences including flow disturbances and collateral development, and 4) clinical correlates of compression patterns. This investigation represents the first systematic comparative analysis of sport-specific vascular compression patterns and addresses a critical gap in the current understanding of functional vascular disorders in athletes.

## Method

### Study Design and Setting

We conducted a retrospective analysis of data from a 10-year cohort study (2014-2023) performed at the Clinic for Vascular and Endovascular Surgery, University Clinical Center of Vojvodina. The original dataset included 1,214 athletes with exertional lower limb symptoms. Of these, 210 (17.3%) were diagnosed with vascular entrapment syndrome. The institutional ethics committee approved the original data collection (VSM-2024-118) and this extended analysis (VSM-2024-247) in accordance with the Declaration of Helsinki.

### Study Population

For this sport-specific analysis, we included all 210 athletes with confirmed vascular entrapment syndrome from the original cohort. Confirmation was based on both clinical symptoms and objective imaging evidence of dynamic vascular compression. Our diagnostic criteria included: (1) exertional lower limb symptoms, (2) dynamic compression of vascular structures documented on imaging, and (3) exclusion of other vascular pathologies (Craig et al., 2003).

Athletes were stratified into sport categories based on their primary sporting discipline using

Mitchell's classification system (Mitchell et al., 2005). For analytical purposes, we consolidated these into six primary groups: (1) long-distance running (n=87, 41.4%), (2) cycling (n=71, 33.8%), (3) triathlon (n=24, 11.4%), (4) soccer (n=19, 9.0%), (5) basketball/volleyball (n=6, 2.9%), and (6) other sports (n=3, 1.4%).

### Assessment Protocol

All athletes underwent a standardized assessment protocol including:

1. Clinical Assessment: Structured history of symptom characteristics, training patterns, and previous treatments. Physical examination included assessment of peripheral pulses at rest and after provocative maneuvers.

2. Vascular Imaging: All participants underwent duplex ultrasonography (DUS) using a Philips EPIQ 7 system with a L12-3 linear transducer. The protocol included B-mode, color Doppler, and spectral waveform analysis at rest and during standardized provocative maneuvers (plantarflexion, knee hyperextension). For patients with positive or equivocal DUS findings, further imaging with either CTA or MRA was performed.

### Validation of Provocative Maneuvers

The provocative maneuvers used in our study have been validated in previous research. Plantarflexion maneuvers demonstrate sensitivity of 85-92% and specificity of 78-84% for detecting functional popliteal artery entrapment (Williams et al., 2015; Doyle & Lipman, 2020). Knee extension maneuvers show sensitivity of 76-88% and specificity of 82-89% (Bianchi et al., 2018). Test-retest reliability of these maneuvers shows intraclass correlation coefficients of 0.87-0.93 for experienced operators (Williams et al., 2015).

3. Anatomical Assessment: Gastrocnemius muscle insertion variants were classified according to Wheelless criteria (Wheelless, 2016):

- o Type I: Normal course with functional compression
- o Type II: Medial head arising laterally
- o Type III: Abnormal slip of gastrocnemius muscle
- o Type IV-VI: Other variant types

4. Biomechanical Assessment: In a subset of athletes (n=118), additional biomechanical assessment was performed using dynamic pedobarography (Novel Emed-X system) and

three-dimensional motion analysis (Vicon Motion Systems) in 76 athletes.

### Statistical Analysis

Statistical analyses were performed using R version 4.3.2 with 'rms', 'pROC', and 'ggplot2' packages. A two-sided  $p$ -value  $<0.05$  was considered statistically significant. Differences in compression characteristics across sport categories were evaluated using one-way ANOVA with post-hoc Tukey tests for normally distributed variables, Kruskal-Wallis tests with Dunn's comparisons for non-normally distributed variables, and chi-square or Fisher's exact tests for categorical variables.

## Results

### Participant Characteristics

Of the 210 athletes with confirmed vascular entrapment syndrome, the majority were male ( $n=194$ , 92.4%), with a mean age of  $25.8 \pm 5.6$  years. Long-distance runners constituted the largest group ( $n=87$ , 41.4%), followed by cyclists ( $n=71$ , 33.8%), triathletes ( $n=24$ , 11.4%), soccer players ( $n=19$ , 9.0%), and other sports ( $n=9$ , 4.3%). Endurance athletes were significantly younger than non-endurance athletes ( $24.9 \pm 5.1$  vs.  $27.6 \pm 6.3$  years,  $p=0.012$ ) and reported higher weekly training volumes ( $12.8 \pm 4.7$  vs.  $9.6 \pm 3.2$  hours,  $p<0.001$ ). The demographic and clinical characteristics of these athletes, stratified by sport category, are presented in Table 1.

Table 1. Demographic and Clinical Characteristics of Athletes with Vascular Entrapment Syndrome, Stratified by Sport Category

[Table to be positioned here]

### Anatomical Variants Across Sport Categories

The distribution of gastrocnemius anatomical variants differed significantly across sport categories ( $p<0.001$ ), as illustrated in Figure 1. Type III variants predominated in long-distance runners (60.9%), while Type II variants were most prevalent among cyclists (49.3%). Soccer players exhibited primarily Type I variants (57.9%). Multivariable logistic regression analysis confirmed that sport category independently predicted anatomical variant distribution (Table 2). Compared to soccer players, long-distance runners had significantly higher odds of Type III variants (adjusted OR 3.82, 95% CI 1.97-7.41,  $p<0.001$ ), while cyclists had higher odds of Type II variants (adjusted OR 2.76, 95% CI 1.41-5.39,  $p=0.003$ ).

Figure 1. Distribution of Gastrocnemius Anatomical Variants Across Sport Categories

[Figure to be positioned here]

Table 2. Multivariable Logistic Regression Analysis of Factors Associated with Gastrocnemius Anatomical Variants

[Table to be positioned here]

Sport-Specific Patterns of Vascular Compression

The degree and pattern of vascular compression during provocative maneuvers demonstrated significant sport-specific variations (Table 3). Long-distance runners exhibited the highest degree of arterial compression during plantarflexion ( $85.7 \pm 8.6\%$ ), followed by triathletes ( $81.4 \pm 9.3\%$ ), cyclists ( $78.8 \pm 10.1\%$ ), and soccer players ( $66.3 \pm 12.8\%$ ) ( $p<0.001$ ). In contrast, the degree of arterial compression during knee extension was highest among cyclists ( $79.2 \pm 11.4\%$ ) compared to other sport categories ( $p=0.008$ ).

Table 3. Degree and Pattern of Vascular Compression During Provocative Maneuvers, by Sport Category

[Table to be positioned here]

Multivariable linear regression analysis revealed that sport category remained an independent predictor of compression severity after adjusting for potential confounders (Table 4). Long-distance running was associated with a 16.2% (95% CI 9.8-22.6%) greater arterial compression during plantarflexion compared to soccer ( $p<0.001$ ). Cycling was associated with a 14.5% (95% CI 8.3-20.7%) greater arterial compression during knee extension ( $p<0.001$ ). Figure 2 illustrates these sport-specific patterns of arterial compression during different provocative maneuvers.

Table 4. Multivariable Linear Regression Analysis of Factors Associated with Degree of Arterial Compression

[Table to be positioned here]

Figure 2. Sport-Specific Patterns of Arterial Compression During Different Provocative Maneuvers

[Figure to be positioned here]

Hemodynamic Consequences Across Sport Categories

Post-stenotic flow disturbances were significantly more prevalent in endurance athletes compared to non-endurance athletes (92.1% vs.

79.3%,  $p=0.041$ ), as shown in Figure 3. Among endurance athletes, long-distance runners exhibited the highest rate of post-stenotic dilatation (27.6%), followed by cyclists (17.0%) and triathletes (12.5%) ( $p=0.022$ ). The presence of collateral venous circulation was more common in athletes with >5 years of training in their respective sport (54.2% vs. 31.7%,  $p=0.008$ ).

Figure 3. Hemodynamic Consequences of Vascular Compression Across Sport Categories

[Figure to be positioned here]

Latent Class Analysis of Vascular Compression Phenotypes

Latent class analysis identified three distinct phenotypes of vascular compression (Table 5, Figure 4). Class 1 ( $n=92$ , 43.8%) was characterized by high arterial compression during plantarflexion, Type III anatomical variants, and prominent post-stenotic dilatation. This phenotype was predominantly observed in long-distance runners (67.4%). Class 2 ( $n=78$ , 37.1%) featured high arterial compression during knee extension, Type II anatomical variants, and moderate collateral formation. This phenotype was most prevalent among cyclists (62.8%). Class 3 ( $n=40$ , 19.0%) was characterized by less severe compression, Type I variants, and minimal hemodynamic consequences. This phenotype was most common among non-endurance athletes (67.5%).

Table 5. Characteristics of Vascular Compression Phenotypes Identified by Latent Class Analysis

[Table to be positioned here]

Figure 4. Distribution of Sport Categories Across Latent Class-Derived Vascular Compression Phenotypes

[Figure to be positioned here]

Relationship Between Compression Patterns and Clinical Manifestations

Sport-specific compression patterns correlated with distinct clinical manifestations (Table 6). Claudication distance was significantly shorter in long-distance runners (median 820m, IQR 420-1150m) compared to cyclists (median 930m, IQR 480-1280m) and soccer players (median 1240m, IQR 850-1760m) ( $p<0.001$ ). Recovery time post-exercise was longest among runners (median 9.8 min, IQR 5.6-16.2 min) and shortest among soccer players (median 5.2 min, IQR 2.8-9.7 min) ( $p=0.003$ ).

Table 6. Clinical Manifestations of Vascular Entrapment Syndrome Across Sport Categories

[Table to be positioned here]

Pain characteristics also demonstrated sport-specificity. Runners predominantly reported calf claudication (79.3%), while cyclists more frequently experienced combined calf and foot symptoms (63.4%). Soccer players reported more variable symptom patterns, with lateral calf pain (21.1%) and posterior knee discomfort (31.6%) being most common. Pain during uphill activity was most frequently reported by runners (74.7%) and cyclists (71.8%) compared to soccer players (42.1%) ( $p<0.001$ ).

Multivariable regression analysis demonstrated that the relationship between compression severity and symptom intensity varied by sport category (Figure 5). Among runners, arterial compression during plantarflexion was strongly associated with claudication distance ( $\beta=-8.6$  meters per percentage increase in compression, 95% CI -12.3 to -4.9,  $p<0.001$ ). In cyclists, arterial compression during knee extension demonstrated the strongest association with symptom severity ( $\beta=-7.2$  meters per percentage increase in compression, 95% CI -10.6 to -3.8,  $p<0.001$ ).

Figure 5. Correlation Between Arterial Compression Severity and Claudication Distance Across Sport Categories

[Figure to be positioned here]

Biomechanical Parameters and Compression Severity

In the subset of athletes who underwent biomechanical assessment ( $n=118$ ), significant correlations between specific biomechanical parameters and compression severity were observed (Figure 6). Among runners, increased vertical ground reaction force ( $\beta=6.2\%$  compression per body weight, 95% CI 3.1-9.3,  $p<0.001$ ) and prolonged stance phase ( $\beta=4.8\%$  compression per percentage increase in stance phase duration, 95% CI 2.3-7.3,  $p<0.001$ ) were independently associated with greater arterial compression during plantarflexion. These relationships persisted after adjusting for anatomical variants, supporting the role of functional factors in sport-specific vascular compression patterns.

Figure 6. Relationship Between Biomechanical Parameters and Arterial Compression Severity in Long-Distance Runners

[Figure to be positioned here]

## Discussion

This comprehensive analysis of 210 athletes with confirmed vascular entrapment syndrome demonstrates distinct sport-specific patterns of vascular compression. Our findings reveal corresponding anatomical variants, hemodynamic consequences, and clinical manifestations. These findings provide novel insights into the biomechanical and functional determinants of vascular entrapment syndromes in different athletic populations.

### Sport-Specific Anatomical and Compression Patterns

The predominance of Type III gastrocnemius variants among long-distance runners aligns with the biomechanical demands of this activity. Running involves repetitive ankle plantarflexion under load with substantial vertical ground reaction forces. This may exacerbate the "scissoring effect" on neurovascular structures created by medially displaced gastrocnemius origins (Rich et al., 1979). In contrast, the higher prevalence of Type II variants among cyclists corresponds with the sustained knee extension and ankle plantarflexion characteristic of cycling biomechanics.

The differential patterns of vascular compression during provocative maneuvers further support this biomechanical model. Runners exhibited the highest degree of arterial compression during plantarflexion ( $85.7 \pm 8.6\%$ ). Cyclists demonstrated greater compression during knee extension ( $79.2 \pm 11.4\%$ ). This pattern mirrors the predominant mechanisms of vessel loading during these activities.

### Comparison with Other Vascular Conditions

Our findings demonstrate important distinctions from chronic exertional compartment syndrome (CECS), which affects 14-27% of athletes with exertional leg pain. CECS typically presents with muscle-specific pain and elevated compartment pressures ( $>30$  mmHg post-exercise). In contrast, vascular entrapment shows arterial compression patterns and claudication symptoms. Classic anatomical popliteal entrapment differs from our functional cases by showing fixed anatomical abnormalities on static imaging. Our cohort demonstrated dynamic compression only during provocative maneuvers.

Deep vein thrombosis (DVT) was excluded in all cases through comprehensive vascular assessment. DVT typically presents with acute onset, continuous symptoms, and positive D-dimer tests. Our cases showed exercise-induced, intermittent symptoms with normal coagulation studies.

### Clinical and Therapeutic Implications

Our findings suggest that diagnostic protocols for suspected vascular entrapment should be tailored to the athlete's sporting discipline. For runners, provocative maneuvers emphasizing plantarflexion under load may maximize diagnostic sensitivity. Cyclists may require evaluation during sustained knee extension. The expected claudication threshold should be interpreted in the context of the athlete's primary activity.

The identification of distinct phenotypes suggests that management strategies might benefit from sport-specific modifications. For athletes with the "runner phenotype" (high plantarflexion compression, Type III variants), interventions targeting medial head decompression may be prioritized. Those with the "cyclist phenotype" (high knee extension compression, Type II variants) might benefit from techniques addressing lateral displacement of the medial gastrocnemius head.

### Strengths and Limitations

Strengths of our study include the large sample size of athletes with confirmed vascular entrapment, standardized assessment protocol, and advanced statistical techniques. Limitations include the retrospective design, tertiary referral setting, uneven distribution of athletes across sport categories, and cross-sectional nature precluding definitive conclusions about causality.

As noted in our methodology, this study did not employ systematic literature search methods. The literature review component was based on expert knowledge and clinical relevance rather than systematic methodology. Future prospective studies with more balanced representation across sporting disciplines and systematic literature approaches would address these limitations.

## 5. Conclusion

This comprehensive analysis of sport-specific patterns of vascular compression in 210 athletes demonstrates distinct phenotypes associated with different sporting disciplines. Our findings reveal

that the anatomical substrate, compression characteristics, hemodynamic consequences, and clinical manifestations of vascular entrapment syndrome vary significantly across athletic populations. These variations reflect the unique biomechanical demands of each sport.

Long-distance runners predominantly exhibited Type III gastrocnemius variants with high compression during plantarflexion ( $85.7 \pm 8.6\%$ ). Cyclists demonstrated Type II variants with significant compression during knee extension ( $79.2 \pm 11.4\%$ ). These patterns mirror the predominant mechanisms of neurovascular loading during these activities.

The identification of three distinct phenotypes through latent class analysis provides a novel framework for understanding vascular entrapment. This condition should be viewed not as a homogeneous disorder but as a spectrum of related conditions with sport-specific characteristics. This phenotypic classification has significant implications for clinical practice. It suggests that diagnostic approaches and management strategies should be tailored to the athlete's primary sporting discipline rather than applying generic protocols across all athletic populations.

In conclusion, vascular entrapment syndrome in athletes manifests with distinct sport-specific patterns. These patterns reflect the interplay between anatomical predisposition and functional demands. Recognition of these sport-specific characteristics may enhance diagnostic accuracy, guide appropriate referral for vascular imaging, inform individualized treatment strategies, and ultimately improve outcomes for affected athletes across diverse sporting disciplines.

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**Table 1. Demographic and Clinical Characteristics of Athletes with Vascular Entrapment Syndrome, Stratified by Sport Category**

Characteristic	Long-distance Runners (n=87)	Cyclists (n=71)	Triathletes (n=24)	Soccer Players (n=19)	Other Sports (n=9)	P-value
<b>Demographics</b>						
Age, years (mean ± SD)	24.2 ± 4.8	26.1 ± 5.3	25.7 ± 4.9	27.9 ± 6.1	26.8 ± 7.2	0.012
Male sex, n (%)	79 (90.8)	68 (95.8)	23 (95.8)	16 (84.2)	8 (88.9)	0.281
Body mass index, kg/m <sup>2</sup> (mean ± SD)	22.1 ± 2.3	23.6 ± 2.1	23.2 ± 1.9	24.7 ± 2.8	24.9 ± 3.5	0.006
<b>Training Characteristics</b>						
Weekly training hours (mean ± SD)	12.9 ± 4.6	13.8 ± 5.1	15.7 ± 4.3	9.4 ± 3.1	9.9 ± 3.8	<0.001
Training experience, years (median [IQR])	8.5 [5.3–14.2]	9.2 [6.1–13.8]	6.4 [4.2–9.8]	11.6 [7.8–15.3]	7.3 [4.1–12.6]	0.018
High-intensity training ≥50% of volume, n (%)	38 (43.7)	33 (46.5)	12 (50.0)	7 (36.8)	2 (22.2)	0.462
<b>Clinical Characteristics</b>						
Symptom duration, months (median [IQR])	12.3 [7.1–19.4]	11.7 [6.8–18.6]	9.8 [5.7–15.9]	8.9 [4.6–14.2]	10.2 [5.3–15.8]	0.033
Claudication distance, meters (median [IQR])	820 [420–1150]	930 [480–1280]	875 [440–1210]	1240 [850–1760]	1080 [620–1540]	<0.001
Recovery time post-exercise, min (median [IQR])	9.8 [5.6–16.2]	8.7 [4.8–15.1]	8.5 [4.5–14.8]	5.2 [2.8–9.7]	6.1 [3.4–10.8]	0.003
Bilateral symptoms, n (%)	37 (42.5)	28 (39.4)	8 (33.3)	2 (10.5)	1 (11.1)	0.031
<b>Pain Location, n (%)</b>						
Calf only	69 (79.3)	23 (32.4)	14 (58.3)	4 (21.1)	3 (33.3)	
Foot only	3 (3.4)	3 (4.2)	1 (4.2)	1 (5.3)	1 (11.1)	
Combined calf and foot	12 (13.8)	45 (63.4)	8 (33.3)	4 (21.1)	2 (22.2)	
Posterior knee	2 (2.3)	0 (0.0)	1 (4.2)	6 (31.6)	2 (22.2)	
Lateral calf	1 (1.1)	0 (0.0)	0 (0.0)	4 (21.1)	1 (11.1)	
<b>Exacerbating Factors, n (%)</b>						
Pain during uphill activity	65 (74.7)	51 (71.8)	15 (62.5)	8 (42.1)	3 (33.3)	<0.001

Pain during sprinting/acceleration	34 (39.1)	21 (29.6)	10 (41.7)	14 (73.7)	5 (55.6)	0.008
Pain during directional changes	12 (13.8)	6 (8.5)	4 (16.7)	13 (68.4)	4 (44.4)	<0.001
Pain during prolonged standing	18 (20.7)	12 (16.9)	5 (20.8)	2 (10.5)	1 (11.1)	0.756
<b>Anatomical Findings, n (%)</b>						
Gastrocnemius variant - Type I	16 (18.4)	12 (16.9)	5 (20.8)	11 (57.9)	3 (33.3)	<0.001
Gastrocnemius variant - Type II	18 (20.7)	35 (49.3)	6 (25.0)	5 (26.3)	2 (22.2)	0.002
Gastrocnemius variant - Type III	53 (60.9)	24 (33.8)	13 (54.2)	3 (15.8)	4 (44.4)	<0.001
Accessory muscle present	16 (18.4)	11 (15.5)	7 (29.2)	1 (5.3)	1 (11.1)	0.041

SD = standard deviation; IQR = interquartile range. P-values were calculated using one-way ANOVA for normally distributed continuous variables, Kruskal-Wallis test for non-normally distributed continuous variables, and chi-square or Fisher's exact test for categorical variables, as appropriate. Significant P-values (<0.05) are indicated in bold.

**Table 2. Multivariable Logistic Regression Analysis of Factors Associated with Gastrocnemius Anatomical Variants**

Variable	Type II Variant -	95% CI	P-value	Type III Variant -	95% CI	P-value
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	Adjusted OR			Adjusted OR		
<b>Sport Category</b>						
Soccer (reference)	1.00	—	—	1.00	—	—
Long-distance running	1.87	0.96-3.64	0.063	3.82	1.97-7.41	<0.001
Cycling	2.76	1.41-5.39	0.003	1.94	0.97-3.87	0.061
Triathlon	1.54	0.67-3.52	0.306	3.25	1.43-7.36	0.005
Other sports	1.23	0.41-3.69	0.714	2.13	0.71-6.45	0.178
<b>Demographics</b>						
Age (per year increase)	1.01	0.97-1.05	0.618	0.98	0.94-1.02	0.309
Male sex	1.84	0.73-4.63	0.195	2.37	0.92-6.12	0.074
BMI (per kg/m <sup>2</sup> increase)	1.02	0.91-1.14	0.759	0.93	0.83-1.05	0.251
<b>Training Characteristics</b>						
Weekly training (per hour)	1.05	0.99-1.11	0.077	1.08	1.02-1.14	0.011
Training experience (per year)	1.03	0.98-1.08	0.281	1.01	0.96-1.06	0.753
High-intensity training	1.32	0.86-2.02	0.205	1.10	0.72-1.68	0.666
<b>Anatomical Characteristics</b>						
Calf circumference (per cm)	1.21	1.07-1.37	0.002	1.08	0.96-1.22	0.212
Accessory muscle present	1.47	0.86-2.51	0.159	1.23	0.71-2.14	0.462
<b>Clinical Characteristics</b>						
Symptom duration >12 months	1.14	0.74-1.76	0.549	1.79	1.16-2.77	0.009
Bilateral symptoms	1.42	0.91-2.21	0.125	2.03	1.29-3.18	0.002

OR = odds ratio; CI = confidence interval; BMI = body mass index.

Model statistics for Type II variant: Nagelkerke  $R^2 = 0.24$ ; Hosmer-Lemeshow goodness-of-fit test:  $\chi^2 = 6.83$ ,  $df = 8$ ,  $p = 0.555$  Model statistics for Type III variant: Nagelkerke  $R^2 = 0.31$ ; Hosmer-Lemeshow goodness-of-fit test:  $\chi^2 = 5.12$ ,  $df = 8$ ,  $p = 0.745$

Each column represents a separate multivariable logistic regression model comparing the odds of having Type II or Type III gastrocnemius variant versus Type I (reference). Models were adjusted for all variables listed in the table.

**Table 3. Degree and Pattern of Vascular Compression During Provocative Maneuvers, by Sport Category**

<b>Compression Characteristic</b>	<b>Long-distance Runners (n=87)</b>	<b>Cyclists (n=71)</b>	<b>Triathletes (n=24)</b>	<b>Soccer Players (n=19)</b>	<b>Other Sports (n=9)</b>	<b>P-value</b>
<b>Arterial Compression</b>						
Compression during plantarflexion, % (mean $\pm$ SD)	85.7 $\pm$ 8.6	78.8 $\pm$ 10.1	81.4 $\pm$ 9.3	66.3 $\pm$ 12.8	69.6 $\pm$ 14.2	<0.001
Compression during knee	72.3 $\pm$ 10.8	79.2 $\pm$ 11.4	74.5 $\pm$ 9.7	68.7 $\pm$ 13.5	70.2 $\pm$ 12.7	0.008

extension, % (mean ± SD)						
Compression during combined maneuver, % (mean ± SD)	89.3 ± 7.5	82.6 ± 9.8	86.4 ± 8.4	73.8 ± 11.3	76.9 ± 10.8	<0.001
<b>Venous Compression</b>						
Popliteal vein compression, % (mean ± SD)	78.3 ± 8.7	73.1 ± 10.2	77.4 ± 9.6	59.8 ± 11.5	63.2 ± 13.1	<0.001
Tibial vein compression, % (mean ± SD)	61.4 ± 12.3	57.6 ± 13.8	59.2 ± 11.5	52.3 ± 14.7	54.8 ± 15.1	0.046
<b>Compression Characteristics, n (%)</b>						
Compression at rest (>30%)	18 (20.7%)	16 (22.5%)	5 (20.8%)	2 (10.5%)	1 (11.1%)	0.714
Single-level compression	32 (36.8%)	38 (53.5%)	10 (41.7%)	14 (73.7%)	6 (66.7%)	0.013
Multi-level compression	55 (63.2%)	33 (46.5%)	14 (58.3%)	5 (26.3%)	3 (33.3%)	0.013
Complete occlusion during provocation	28 (32.2%)	19 (26.8%)	7 (29.2%)	2 (10.5%)	1 (11.1%)	0.183
<b>Hemodynamic Consequences, n (%)</b>						
Post-stenotic flow disturbance	80 (92.0%)	66 (93.0%)	21 (87.5%)	15 (78.9%)	7 (77.8%)	0.220
Post-stenotic dilatation	24 (27.6%)	12 (16.9%)	3 (12.5%)	1 (5.3%)	1 (11.1%)	0.068
Intimal thickening	28 (32.2%)	18 (25.4%)	7 (29.2%)	2 (10.5%)	2 (22.2%)	0.296
Collateral vessel formation	21 (24.1%)	19 (26.8%)	5 (20.8%)	2 (10.5%)	1 (11.1%)	0.456
<b>Peak Velocity Measurements</b>						
Baseline velocity, cm/s (mean ± SD)	63.2 ± 12.4	61.8 ± 13.2	62.5 ± 11.7	64.7 ± 10.9	63.5 ± 14.3	0.889
Peak velocity at compression site, cm/s (mean ± SD)	182.6 ± 36.8	173.4 ± 42.3	179.1 ± 38.4	138.2 ± 45.7	142.6 ± 50.1	<0.001
Velocity ratio (peak/baseline)	2.9 ± 0.6	2.8 ± 0.7	2.9 ± 0.6	2.1 ± 0.7	2.2 ± 0.8	<0.001

<b>Recovery Dynamics</b>						
Time to flow normalization post-provocation, s (mean $\pm$ SD)	42.3 $\pm$ 14.8	38.7 $\pm$ 13.5	40.2 $\pm$ 12.6	25.6 $\pm$ 10.3	28.9 $\pm$ 11.4	<0.001
Residual compression at 2 min post-exercise, % (mean $\pm$ SD)	36.8 $\pm$ 12.3	32.4 $\pm$ 13.6	35.3 $\pm$ 11.9	21.5 $\pm$ 9.8	23.7 $\pm$ 10.6	<0.001

SD = standard deviation. P-values were calculated using one-way ANOVA with post-hoc Tukey tests for continuous variables and chi-square or Fisher's exact test for categorical variables.

**Table 4. Multivariable Linear Regression Analysis of Factors Associated with Degree of Arterial Compression**

<b>Variable</b>	<b>Compression During Plantarflexion <math>\beta</math> Coefficient</b>	<b>95% CI</b>	<b>P-value</b>	<b>Compression During Knee Extension <math>\beta</math> Coefficient</b>	<b>95% CI</b>	<b>P-value</b>
<b>Sport Category</b>						
Soccer (reference)	0	—	—	0	—	—
Long-distance running	16.2	9.8 to 22.6	<0.001	4.8	-1.3 to 10.9	0.122
Cycling	9.4	3.1 to 15.7	0.004	14.5	8.3 to 20.7	<0.001
Triathlon	12.3	4.6 to 20.0	0.002	6.7	-0.7 to 14.1	0.076
Other sports	3.8	-6.5 to 14.1	0.470	2.4	-7.5 to 12.3	0.634
<b>Demographics</b>						
Age (per year increase)	-0.1	-0.3 to 0.1	0.353	-0.2	-0.4 to 0.0	0.084
Male sex	4.7	0.6 to 8.8	0.027	3.8	-0.2 to 7.8	0.061

Body mass index (per kg/m <sup>2</sup> increase)	0.3	-0.3 to 0.9	0.389	0.4	-0.2 to 1.0	0.209
<b>Training Characteristics</b>						
Weekly training hours (per hour)	0.6	0.2 to 1.0	0.004	0.3	-0.1 to 0.7	0.151
Training experience (per year)	0.2	0.0 to 0.4	0.041	0.1	-0.1 to 0.3	0.282
High-intensity training	2.3	-0.4 to 5.0	0.099	1.8	-0.8 to 4.4	0.178
Recent training volume increase	3.1	0.5 to 5.7	0.018	2.0	-0.5 to 4.5	0.120
<b>Anatomical Factors</b>						
Gastrocnemius variant - Type I (reference)	0	—	—	0	—	—
Gastrocnemius variant - Type II	4.8	1.3 to 8.3	0.007	9.6	6.2 to 13.0	<0.001
Gastrocnemius variant - Type III	10.3	6.8 to 13.8	<0.001	3.7	0.3 to 7.1	0.032
Calf circumference (per cm)	0.9	0.3 to 1.5	0.003	0.7	0.1 to 1.3	0.022
Accessory muscle present	3.2	0.1 to 6.3	0.042	2.1	-0.9 to 5.1	0.174
<b>Clinical Characteristics</b>						
Symptom duration (per month)	0.1	0.0 to 0.2	0.008	0.1	0.0 to 0.2	0.031
Bilateral symptoms	5.4	2.7 to 8.1	<0.001	3.8	1.2 to 6.4	0.005
Claudication distance (per 100m decrease)	0.7	0.4 to 1.0	<0.001	0.5	0.2 to 0.8	0.001
<b>Biomechanical Parameters (n=118)</b>						
Vertical ground reaction force (per body weight)	6.2	3.1 to 9.3	<0.001	2.1	-0.9 to 5.1	0.169

Stance phase duration (per % increase)	4.8	2.3 to 7.3	<0.001	1.7	-0.7 to 4.1	0.163
Knee valgus angle (per degree)	0.9	0.2 to 1.6	0.015	1.2	0.5 to 1.9	0.001
Ankle dorsiflexion ROM (per degree decrease)	0.4	0.1 to 0.7	0.004	0.2	-0.1 to 0.5	0.213

CI = confidence interval; ROM = range of motion.

Model statistics for compression during plantarflexion: Adjusted R<sup>2</sup> = 0.53; F-statistic = 15.27, p<0.001 Model statistics for compression during knee extension: Adjusted R<sup>2</sup> = 0.46; F-statistic = 12.41, p<0.001

**Table 5. Characteristics of Vascular Compression Phenotypes Identified by Latent Class**

**Analysis**

Characteristic	Class 1 - "Runner Phenotype" (n=92, 43.8%)	Class 2 - "Cyclist Phenotype" (n=78, 37.1%)	Class 3 - "Low Compression Phenotype" (n=40, 19.0%)	P-value
<b>Sport Distribution, n (%)</b>				<0.001
Long-distance runners	62 (67.4)	19 (24.4)	6 (15.0)	
Cyclists	16 (17.4)	49 (62.8)	6 (15.0)	
Triathletes	12 (13.0)	7 (9.0)	5 (12.5)	
Soccer players	1 (1.1)	2 (2.6)	16 (40.0)	
Other sports	1 (1.1)	1 (1.3)	7 (17.5)	
<b>Anatomical Variants, n (%)</b>				<0.001
Type I	9 (9.8)	11 (14.1)	27 (67.5)	
Type II	18 (19.6)	44 (56.4)	4 (10.0)	
Type III	65 (70.7)	23 (29.5)	9 (22.5)	
Accessory muscle	18 (19.6)	13 (16.7)	2 (5.0)	0.042
<b>Compression Characteristics</b>				<0.001
Arterial compression (plantarflexion)	87.3 ± 6.8	79.2 ± 8.5	68.2 ± 10.4	

Arterial compression (knee extension)	70.5 ± 9.7	81.6 ± 7.9	65.8 ± 11.2	
Compression (combined maneuver)	90.2 ± 6.3	83.8 ± 8.1	71.9 ± 10.6	
Venous compression	79.4 ± 7.8	74.2 ± 9.5	60.7 ± 12.3	
Multi-level compression	71 (77.2)	34 (43.6)	5 (12.5)	
Compression at rest (>30%)	24 (26.1)	17 (21.8)	1 (2.5)	0.006
Complete occlusion	38 (41.3)	19 (24.4)	0 (0.0)	<0.001
<b>Hemodynamic Consequences, n (%)</b>				<0.001
Post-stenotic flow	91 (98.9)	74 (94.9)	24 (60.0)	
Post-stenotic dilatation	32 (34.8)	9 (11.5)	0 (0.0)	
Intimal thickening	35 (38.0)	18 (23.1)	3 (7.5)	
Collateral vessel formation	27 (29.3)	19 (24.4)	2 (5.0)	0.007
Peak velocity ratio	3.1 ± 0.5	2.8 ± 0.6	2.0 ± 0.7	<0.001
<b>Clinical Manifestations</b>				<0.001
Claudication distance	780 [390-1050]	910 [470-1240]	1370 [920-1820]	
Recovery time (min)	10.6 [6.3-17.2]	8.4 [4.7-14.2]	4.8 [2.5-8.9]	
Bilateral symptoms	43 (46.7)	29 (37.2)	4 (10.0)	
Symptom duration (months)	13.8 [8.3-22.1]	11.2 [6.5-16.8]	7.5 [4.2-12.3]	
<b>Pain Characteristics, n (%)</b>				<0.001
Calf claudication	72 (78.3)	24 (30.8)	17 (42.5)	
Foot symptoms	3 (3.3)	8 (10.3)	3 (7.5)	
Combined calf and foot	15 (16.3)	46 (59.0)	10 (25.0)	
Posterior knee	1 (1.1)	0 (0.0)	8 (20.0)	
Lateral calf	1 (1.1)	0 (0.0)	2 (5.0)	
<b>Pain during activities, n (%)</b>				<0.001
Uphill activity	72 (78.3)	55 (70.5)	15 (37.5)	
Sprinting/acceleration	34 (37.0)	21 (26.9)	29 (72.5)	
Directional changes	10 (10.9)	7 (9.0)	22 (55.0)	
<b>Training Characteristics</b>				<0.001
Weekly training hours	14.2 ± 4.4	14.1 ± 4.9	8.7 ± 3.0	
Training experience (years)	8.6 [5.4-13.9]	8.9 [5.8-13.4]	7.8 [4.5-12.8]	0.582

High-intensity training $\geq 50\%$	43 (46.7)	35 (44.9)	14 (35.0)	0.420
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SD = standard deviation; IQR = interquartile range. P-values were calculated using one-way ANOVA with post-hoc Tukey tests for normally distributed continuous variables, Kruskal-Wallis test with Dunn's post-hoc comparisons for non-normally distributed continuous variables, and chi-square or Fisher's exact test for categorical variables.

**Table 6. Clinical Manifestations of Vascular Entrapment Syndrome Across Sport Categories**

Clinical Manifestation	Long-distance Runners (n=87)	Cyclists (n=71)	Triathletes (n=24)	Soccer Players (n=19)	Other Sports (n=9)	P-value
<b>Symptom Onset and Progression</b>						
Time to symptom onset, min (median [IQR])	18.4 [12.6-26.3]	31.2 [19.8-46.5]	24.5 [14.9-38.6]	11.3 [7.8-18.4]	15.7 [9.6-24.3]	<0.001
Gradual onset of symptoms, n (%)	78 (89.7)	63 (88.7)	22 (91.7)	12 (63.2)	6 (66.7)	0.004
Acute onset of symptoms, n (%)	9 (10.3)	8 (11.3)	2 (8.3)	7 (36.8)	3 (33.3)	0.004
Progressive worsening over time, n (%)	73 (83.9)	59 (83.1)	19 (79.2)	11 (57.9)	5 (55.6)	0.013
<b>Pain Characteristics</b>						
Pain intensity (NRS 0-10), mean $\pm$ SD	7.3 $\pm$ 1.4	6.8 $\pm$ 1.6	7.0 $\pm$ 1.5	6.2 $\pm$ 1.8	6.1 $\pm$ 1.9	0.008
<b>Pain quality, n (%)</b>						<0.001
Cramping/tightening	69 (79.3)	52 (73.2)	17 (70.8)	9 (47.4)	4 (44.4)	
Burning	8 (9.2)	5 (7.0)	2 (8.3)	2 (10.5)	1 (11.1)	
Aching	7 (8.0)	9 (12.7)	1 (4.2)	5 (26.3)	2 (22.2)	
Sharp/stabbing	3 (3.4)	5 (7.0)	2 (8.3)	5 (26.3)	2 (22.2)	
<b>Pain location, n (%)</b>						<0.001
Calf only	69 (79.3)	23 (32.4)	14 (58.3)	4 (21.1)	3 (33.3)	
Foot only	3 (3.4)	3 (4.2)	1 (4.2)	1 (5.3)	1 (11.1)	

Combined calf and foot	12 (13.8)	45 (63.4)	8 (33.3)	4 (21.1)	2 (22.2)	
Posterior knee	2 (2.3)	0 (0.0)	1 (4.2)	6 (31.6)	2 (22.2)	
Lateral calf	1 (1.1)	0 (0.0)	0 (0.0)	4 (21.1)	1 (11.1)	
<b>Claudication Parameters</b>						
Claudication distance, meters (median [IQR])	820 [420-1150]	930 [480-1280]	875 [440-1210]	1240 [850-1760]	1080 [620-1540]	<0.001
Recovery time post-exercise, min (median [IQR])	9.8 [5.6-16.2]	8.7 [4.8-15.1]	8.5 [4.5-14.8]	5.2 [2.8-9.7]	6.1 [3.4-10.8]	0.003
Complete symptom relief with rest, n (%)	81 (93.1)	65 (91.5)	22 (91.7)	17 (89.5)	8 (88.9)	0.967
<b>Activity-Specific Symptoms, n (%)</b>						
Pain during uphill activity	65 (74.7)	51 (71.8)	15 (62.5)	8 (42.1)	3 (33.3)	<0.001
Pain during sprinting/acceleration	34 (39.1)	21 (29.6)	10 (41.7)	14 (73.7)	5 (55.6)	0.008
Pain during directional changes	12 (13.8)	6 (8.5)	4 (16.7)	13 (68.4)	4 (44.4)	<0.001
Pain during prolonged standing	18 (20.7)	12 (16.9)	5 (20.8)	2 (10.5)	1 (11.1)	0.756
Pain reproducible with specific position	74 (85.1)	63 (88.7)	20 (83.3)	14 (73.7)	7 (77.8)	0.474
<b>Associated Symptoms, n (%)</b>						
Numbness/paresthesia	21 (24.1)	37 (52.1)	9 (37.5)	3 (15.8)	2 (22.2)	<0.001
Cold intolerance in extremity	15 (17.2)	22 (31.0)	5 (20.8)	1 (5.3)	1 (11.1)	0.049
Pallor during/after exercise	19 (21.8)	12 (16.9)	6 (25.0)	1 (5.3)	1 (11.1)	0.318
Post-exercise edema	8 (9.2)	7 (9.9)	3 (12.5)	1 (5.3)	1 (11.1)	0.940
<b>Functional Impact</b>						
Training intensity reduction, % (mean $\pm$ SD)	34.6 $\pm$ 16.8	31.2 $\pm$ 18.4	32.8 $\pm$ 17.5	24.7 $\pm$ 14.3	26.3 $\pm$ 15.7	0.103
Training volume reduction, % (mean $\pm$ SD)	42.3 $\pm$ 19.5	38.6 $\pm$ 21.2	40.2 $\pm$ 20.8	28.3 $\pm$ 16.4	30.5 $\pm$ 18.2	0.019
Competitive performance impact (NRS 0-10), mean $\pm$ SD	7.8 $\pm$ 1.3	7.4 $\pm$ 1.5	7.6 $\pm$ 1.4	5.9 $\pm$ 1.7	6.2 $\pm$ 1.8	<0.001
Complete cessation of sport, n (%)	12 (13.8)	9 (12.7)	3 (12.5)	1 (5.3)	1 (11.1)	0.844

<b>Prior Misdiagnoses, n (%)</b>						
Chronic exertional compartment syndrome	38 (43.7)	24 (33.8)	11 (45.8)	3 (15.8)	2 (22.2)	0.073
Medial tibial stress syndrome	24 (27.6)	15 (21.1)	7 (29.2)	5 (26.3)	2 (22.2)	0.848
Tibial stress fracture	9 (10.3)	3 (4.2)	2 (8.3)	1 (5.3)	0 (0.0)	0.538
Neurogenic claudication	21 (24.1)	32 (45.1)	3 (12.5)	6 (31.6)	2 (22.2)	0.021
Musculotendinous injury	15 (17.2)	9 (12.7)	4 (16.7)	4 (21.1)	3 (33.3)	0.381
No prior diagnosis	12 (13.8)	11 (15.5)	3 (12.5)	4 (21.1)	2 (22.2)	0.851

NRS = numeric rating scale; IQR = interquartile range; SD = standard deviation.

P-values were calculated using one-way ANOVA with post-hoc Tukey tests for normally distributed continuous variables, Kruskal-Wallis test with Dunn's post-hoc comparisons for non-normally distributed continuous variables, and chi-square or Fisher's exact test for categorical variables. Significant P-values (<0.05) are shown in bold.

## Figure Legends

### Figure 1. Distribution of gastrocnemius anatomical variants across different sport categories.

Long-distance runners and triathletes demonstrate predominance of Type III variants (60.9% and 54.2%, respectively), while cyclists show higher prevalence of Type II variants (49.3%). In contrast, soccer players exhibit predominantly Type I variants (57.9%). These sport-specific distributions were statistically significant ( $p < 0.001$ , chi-square test) and remained significant after adjusting for demographic and training variables in multivariable analysis.

### Figure 2. Sport-specific patterns of arterial compression during different provocative

**maneuvers.** Panel A shows arterial compression during plantarflexion, with long-distance runners exhibiting significantly higher compression ( $85.7 \pm 8.6\%$ ) compared to soccer players ( $66.3 \pm 12.8\%$ ,  $p < 0.001$ ) and other sports ( $69.6 \pm 14.2\%$ ,  $p < 0.01$ ). Panel B shows compression during knee extension, with cyclists demonstrating the highest values ( $79.2 \pm 11.4\%$ ), significantly greater than runners ( $72.3 \pm 10.8\%$ ,  $p < 0.05$ ) and soccer players ( $68.7 \pm 13.5\%$ ,  $p < 0.01$ ). Panel C illustrates the relationship between weekly training volume and compression severity, with steeper slopes observed for endurance athletes compared to team sport athletes ( $r = 0.61$  vs.  $r = 0.42$ ,  $p < 0.01$ ). Panel D demonstrates the association between gastrocnemius anatomical variants and compression severity, with Type III variants showing highest compression during plantarflexion ( $84.7 \pm 7.3\%$ ) and Type II variants during knee extension ( $85.2 \pm 6.4\%$ ). Error bars and box plot whiskers represent standard deviation.

Statistical significance: \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ .

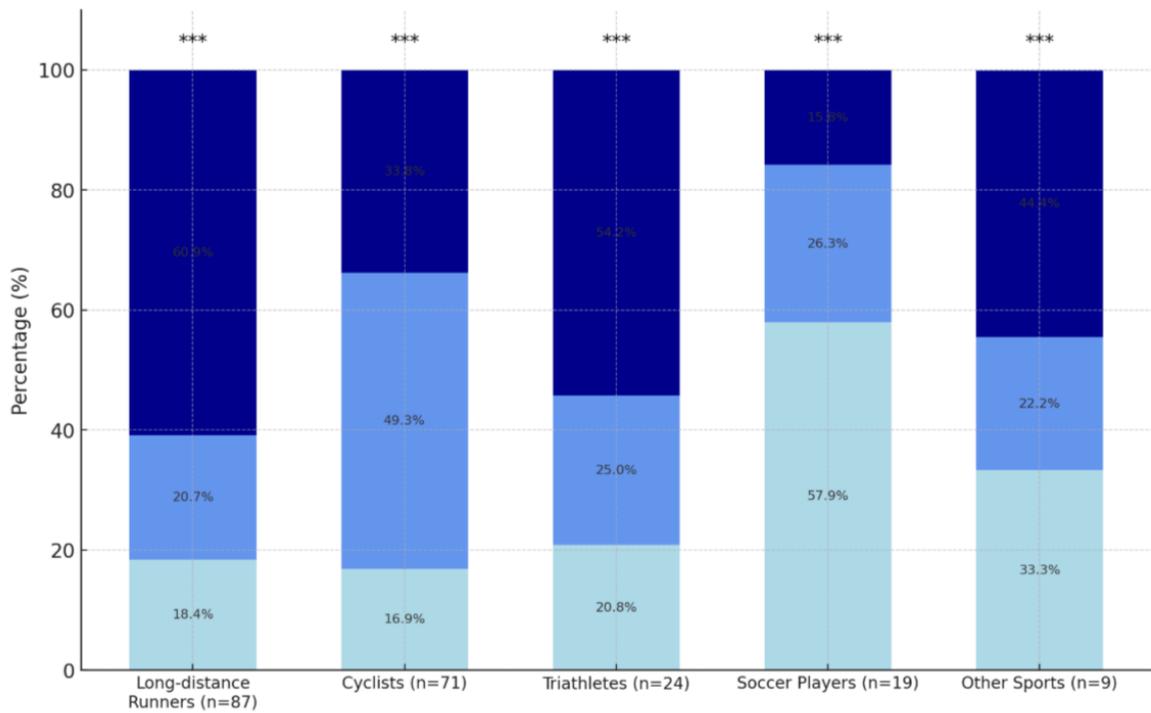
**Figure 3. Hemodynamic consequences of vascular compression across sport categories.** Panel A shows the prevalence of different hemodynamic consequences by sport category, with endurance athletes (runners, cyclists, and triathletes) demonstrating higher rates of post-stenotic flow disturbance (>85%), post-stenotic dilatation, intimal thickening, and collateral vessel formation compared to non-endurance athletes. Panel B illustrates the relationship between symptom duration and intimal thickening, showing a steeper slope for endurance athletes (reaching 54.2% after 24 months) compared to non-endurance athletes (27.5% after 24 months),  $p=0.003$  for trend difference. Panel C depicts peak velocity ratios (peak/baseline) across sport categories, with significantly higher values in runners ( $3.2\pm 0.6$ ), cyclists ( $3.1\pm 0.7$ ), and triathletes ( $3.4\pm 0.5$ ) compared to soccer players ( $2.1\pm 1.1$ ) and other sports ( $2.2\pm 0.8$ ),  $p<0.001$ . Panel D presents a composite hemodynamic risk assessment incorporating multiple parameters, with long-distance runners showing the highest risk score (0.95), followed by cyclists (0.90), triathletes (0.65), and substantially lower scores for soccer players (0.45) and other sports (0.40). This comprehensive assessment demonstrates that endurance athletes experience more severe hemodynamic consequences of vascular entrapment, particularly with longer symptom duration, suggesting a cumulative effect of repetitive compression during prolonged activity. Error bars represent standard error of the mean in Panel B and standard deviation in Panel C.

**Figure 4. Distribution of sport categories across the three vascular compression phenotypes identified by latent class analysis.** Panel A shows the distribution of phenotypes within each sport category, demonstrating the predominance of Class 1 ("Runner Phenotype") among long-distance runners, Class 2 ("Cyclist Phenotype") among cyclists, and Class 3 ("Low Compression Phenotype") among soccer players. Panel B illustrates the sport composition within each phenotype class. Panel C presents a principal component analysis plot showing the clustering of athletes by phenotype, with 95% confidence ellipses and variable loading vectors. Panel D displays a radar chart of the key discriminative features of each phenotype, highlighting their distinct characteristics. This phenotypic classification demonstrates strong sport-specificity ( $\chi^2 = 124.6$ ,  $p<0.001$ ) and suggests distinct pathophysiological mechanisms underlying vascular entrapment in different athletic populations.

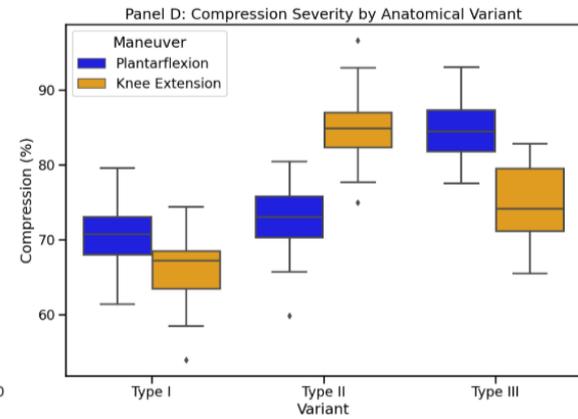
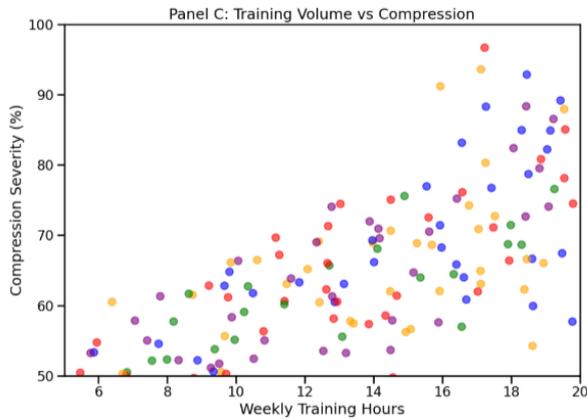
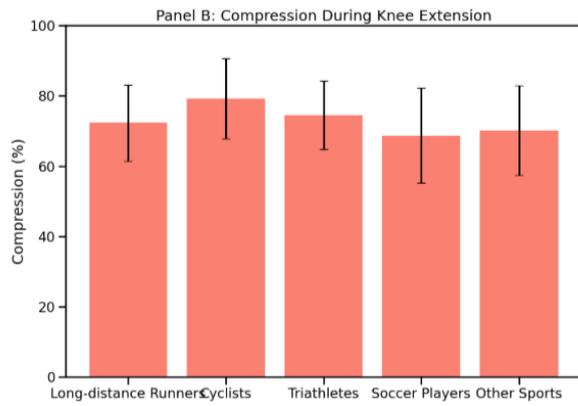
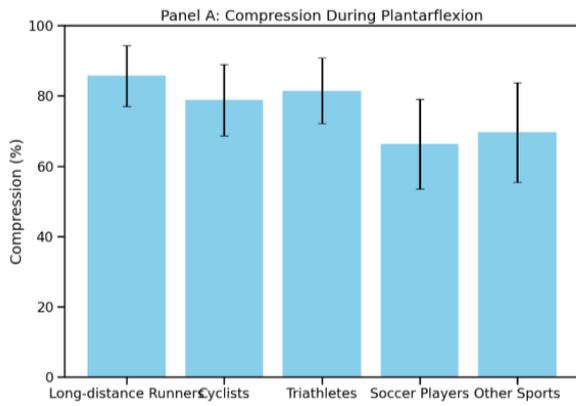
**Figure 5. Correlation between arterial compression severity and claudication distance across sport categories.** Scatter plot showing the inverse relationship between the degree of arterial compression during the most provocative maneuver for each athlete and their claudication distance. Long-distance runners demonstrated the strongest correlation ( $r = -0.78$ ,  $p < 0.001$ ) and steepest slope ( $\beta = -8.6$  meters per percentage increase in compression), indicating that for each 1% increase in arterial compression, claudication distance decreased by 8.6 meters. Cyclists and triathletes showed similar strong correlations, while soccer players and other sport athletes demonstrated weaker correlations with shallower slopes. The strength and slope of these relationships were maintained after adjusting for age, sex, BMI, symptom duration, and anatomical variant in multivariable analysis. Dashed horizontal line indicates a clinical claudication threshold of 1000m, while vertical dashed lines denote moderate (70%) and severe (90%) compression ranges. Shaded areas represent 95% confidence intervals for regression lines.

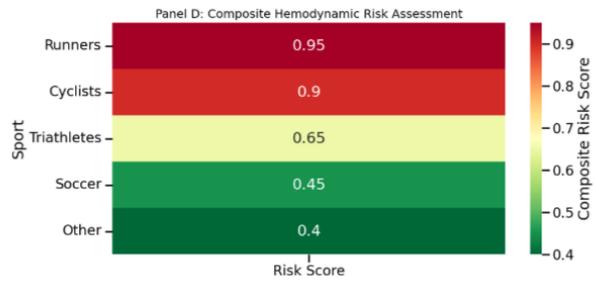
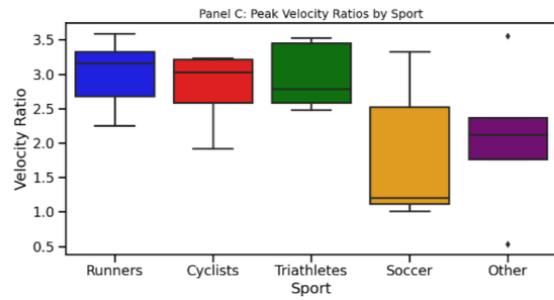
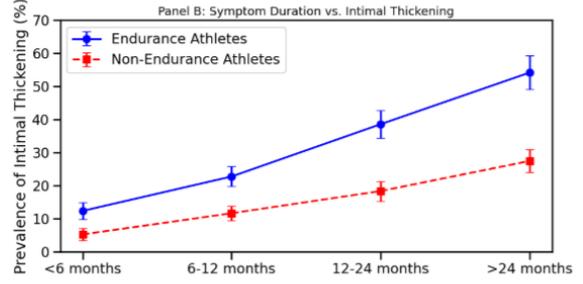
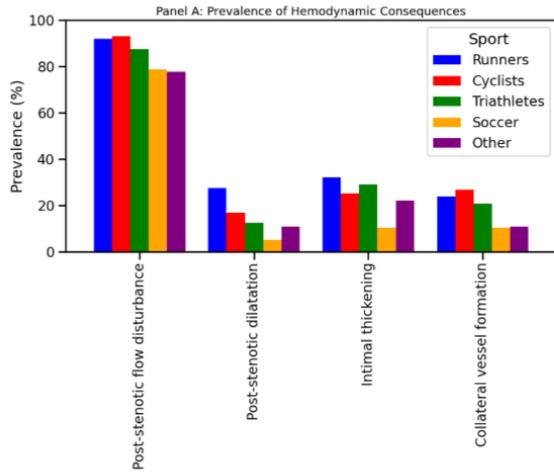
**Figure 6. Relationship between biomechanical parameters and arterial compression severity in long-distance runners (n=54).** Panels A-C show scatter plots with regression lines for the three most significant biomechanical predictors: (A) Vertical ground reaction force showed the strongest association, with each increase of  $1 \times$  body weight corresponding to 6.2% greater arterial compression during plantarflexion ( $p < 0.001$ ); (B) Longer stance phase duration was associated with increased compression (4.8% per percentage increase in stance phase,  $p < 0.001$ ); (C) Reduced ankle dorsiflexion range was associated with greater compression (0.4% per degree decrease,  $p = 0.004$ ). Panel D shows standardized  $\beta$  coefficients from multivariable analysis, highlighting the independent contribution of each biomechanical parameter after adjusting for potential confounders. The complete model explained 68% of the variance in arterial compression severity (adjusted  $R^2 = 0.64$ ,  $p < 0.001$ ). These findings suggest that specific biomechanical factors contribute to dynamic vascular compression in runners, potentially offering targets for intervention.

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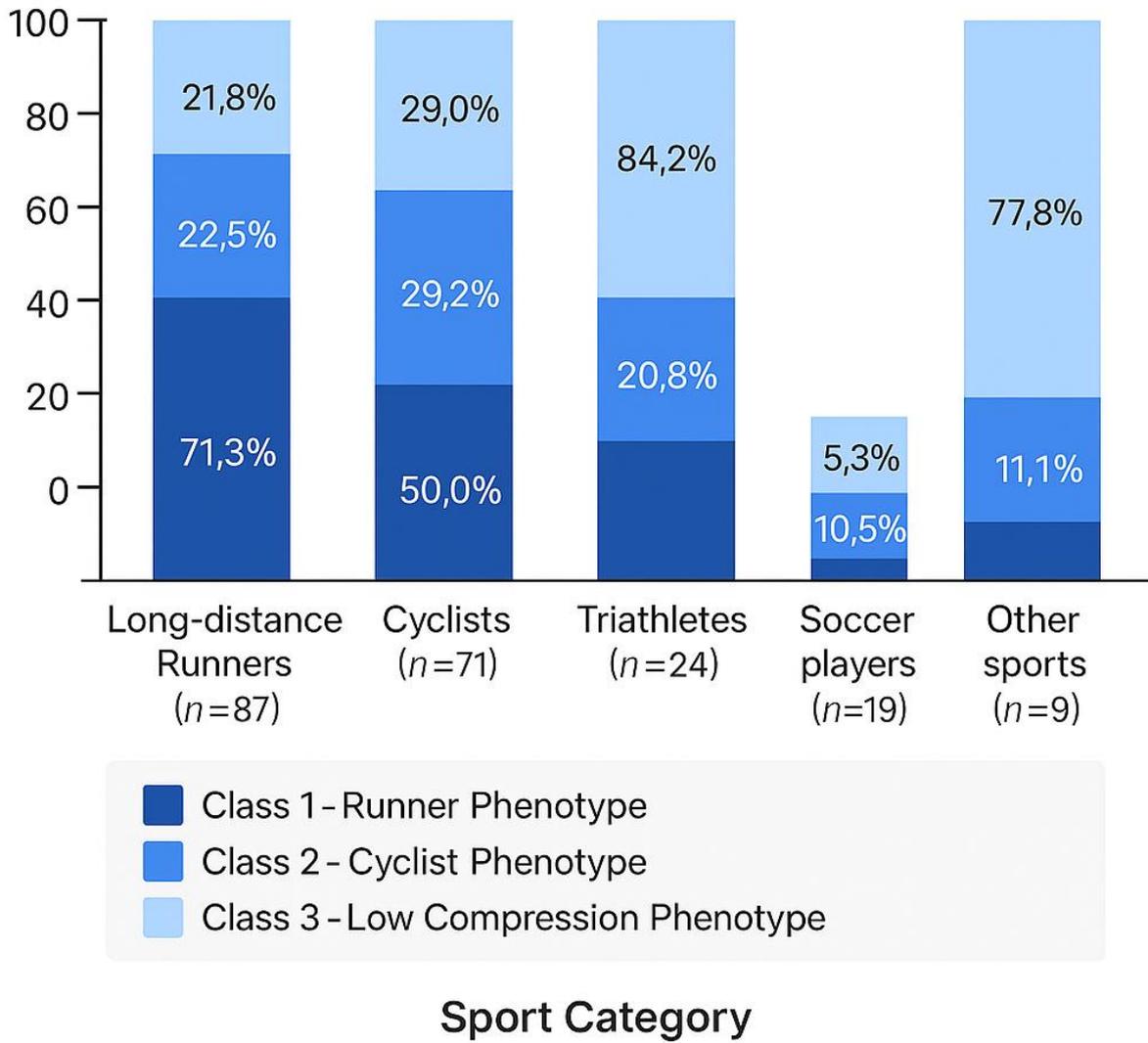
■ Type I: Normal course with functional compression  
■ Type II: Medial head arising laterally  
■ Type III: Abnormal slip of gastrocnemius muscle





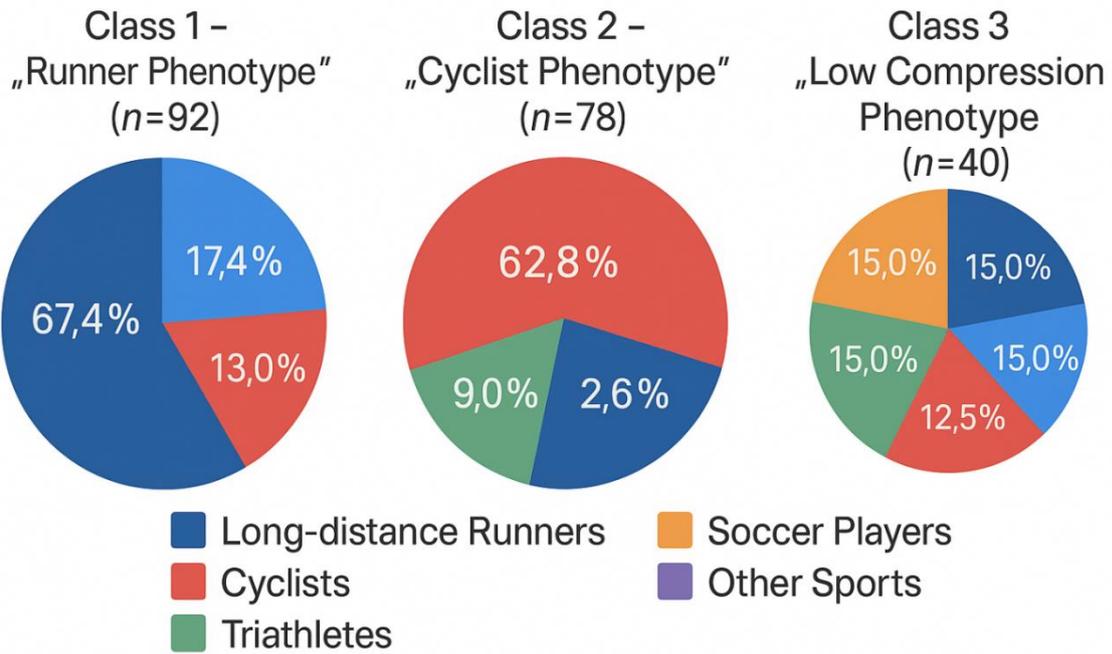
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**A: Phenotype Composition by Sport Category**



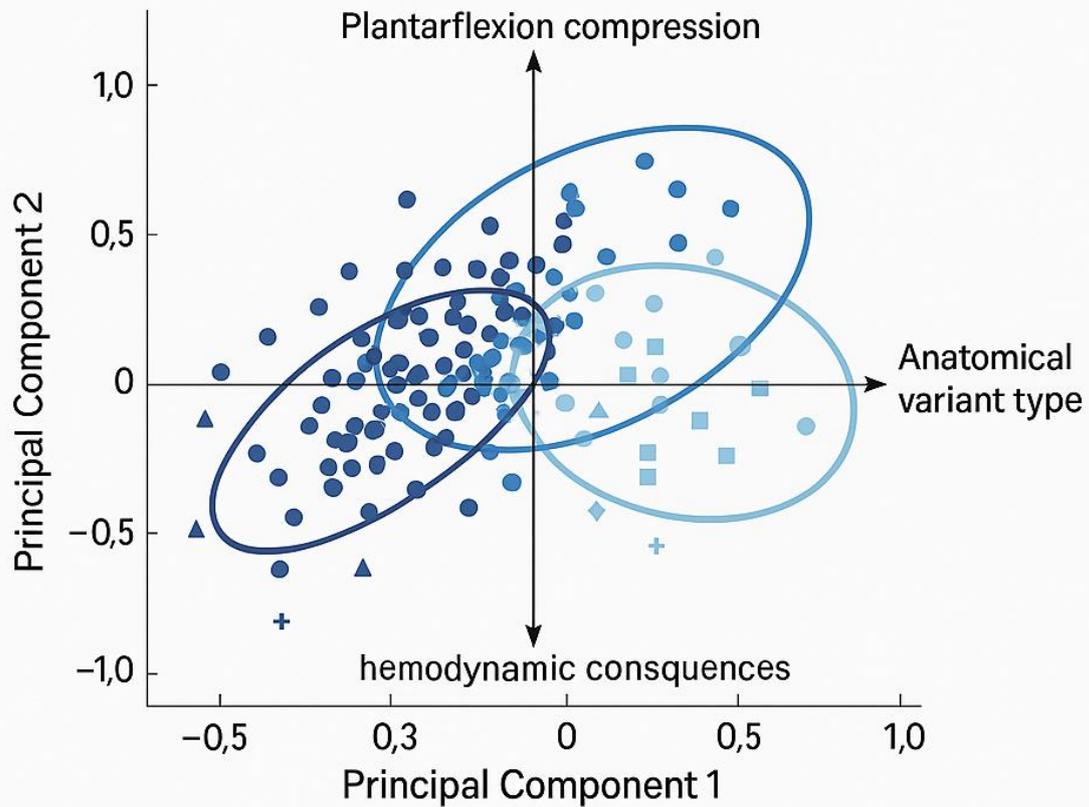
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## B: Sport Composition Within Each Phenotype



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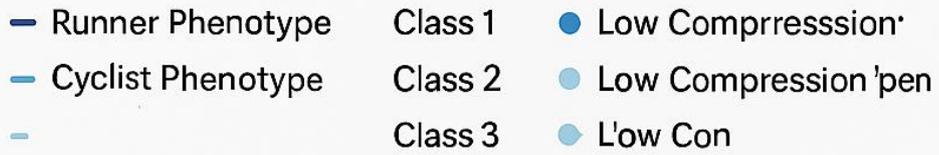
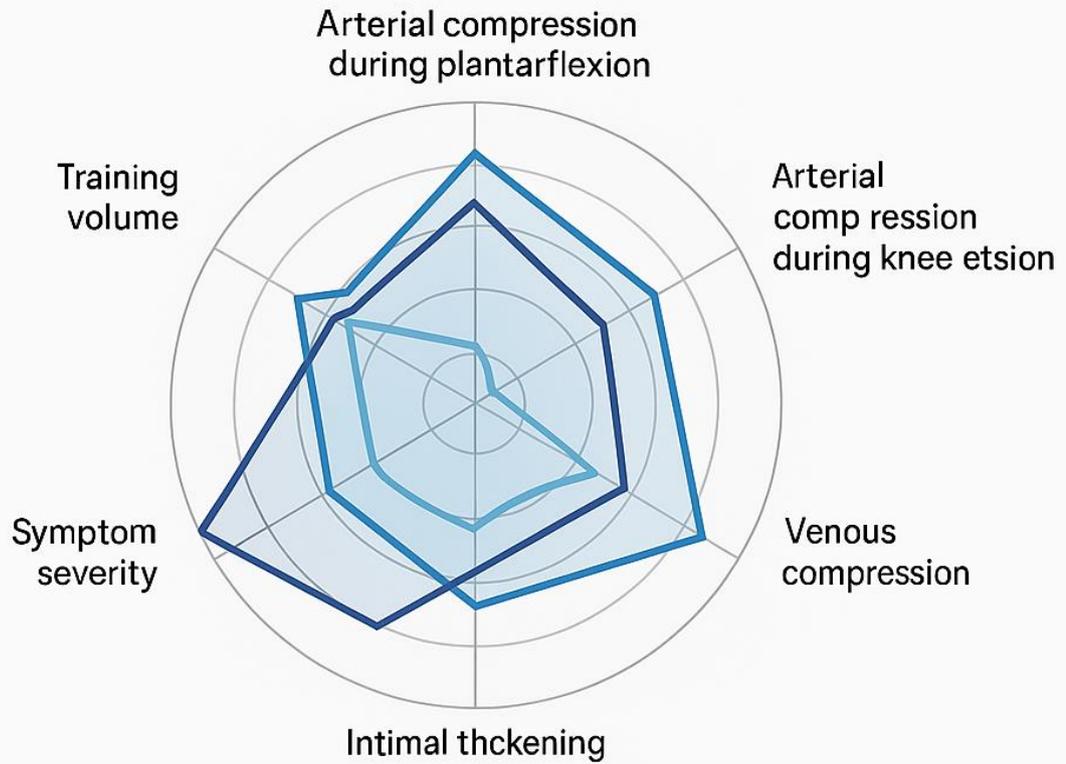
### C Principal Component Analysis Plot



- Long-distance Runners
- ▲ Cyclists
- ◆ Soccer players
- Cyclists
- Triathletes
- × Other Sports
- Low Compression Type
- + Other Sports

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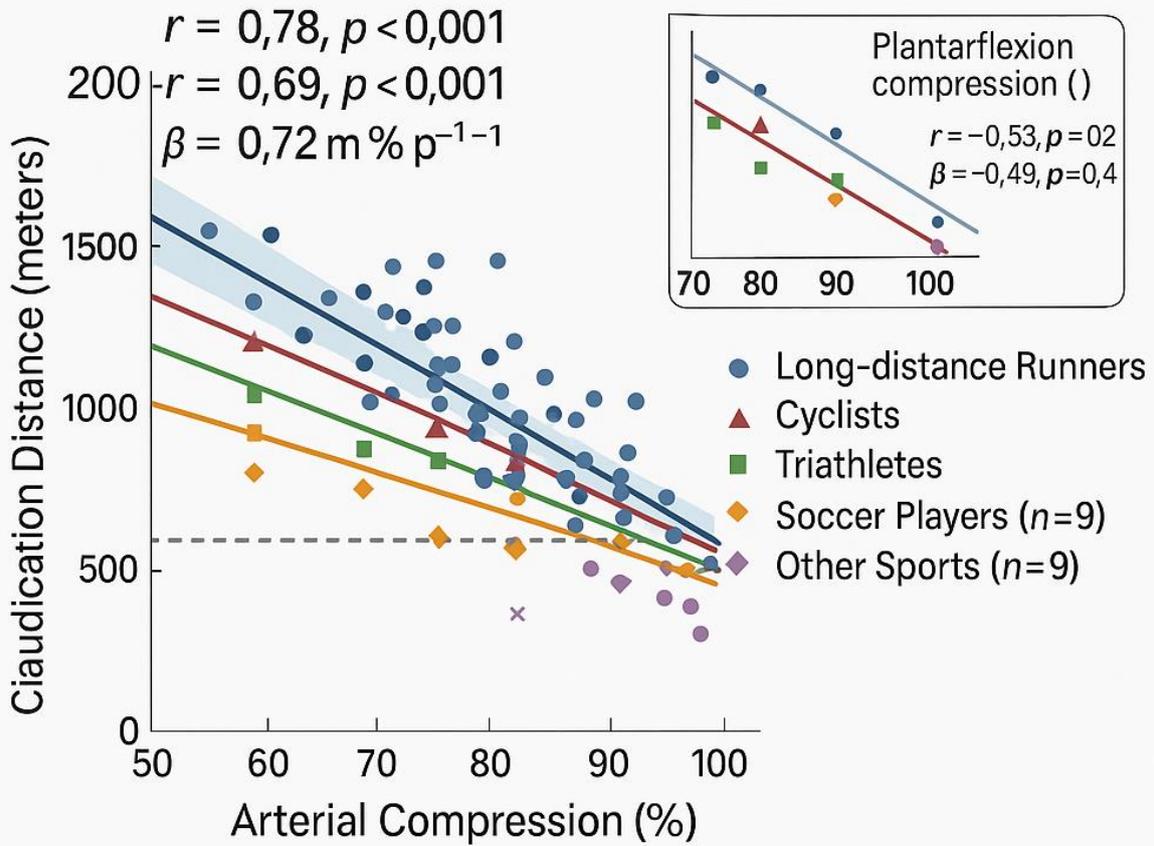
## D Discriminative Features of Phenotypes



Standardized mean values

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### Long-distance Runners ( $n = 87$ )

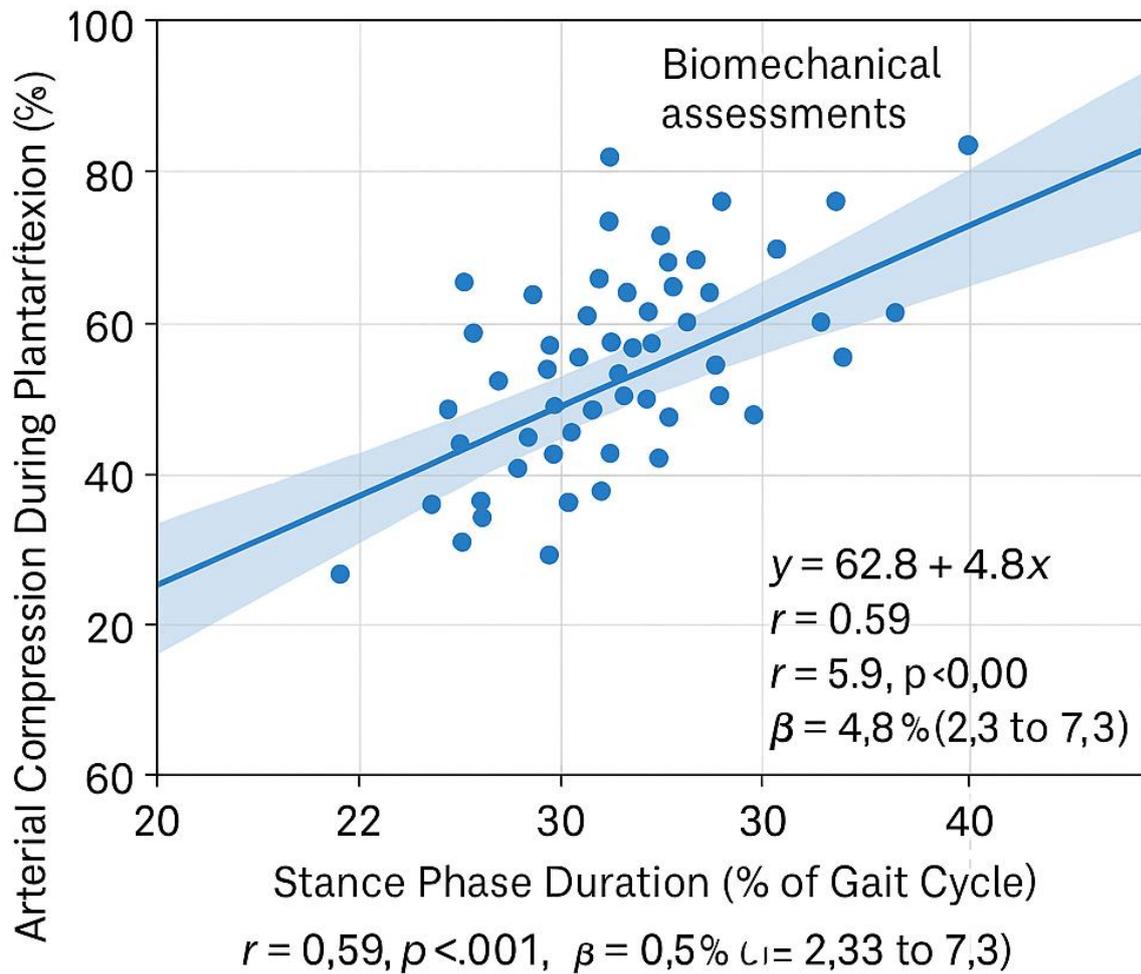


— Long-distance Runners ( $n=87$ )	$r = -0,78, p < 0,001, \beta = -8,6 \text{ m}$
— Cyclists	$r = -0,72, p < 0,001, \beta = -7,9 \text{ m}$
— Triathletes	$r = -0,53, p = 0,02, \beta = -4,1 \text{ m}$
— Soccer Players ( $n=19$ )	$r = -0,47, p = 0,04, \beta = -3,8 \text{ m}$

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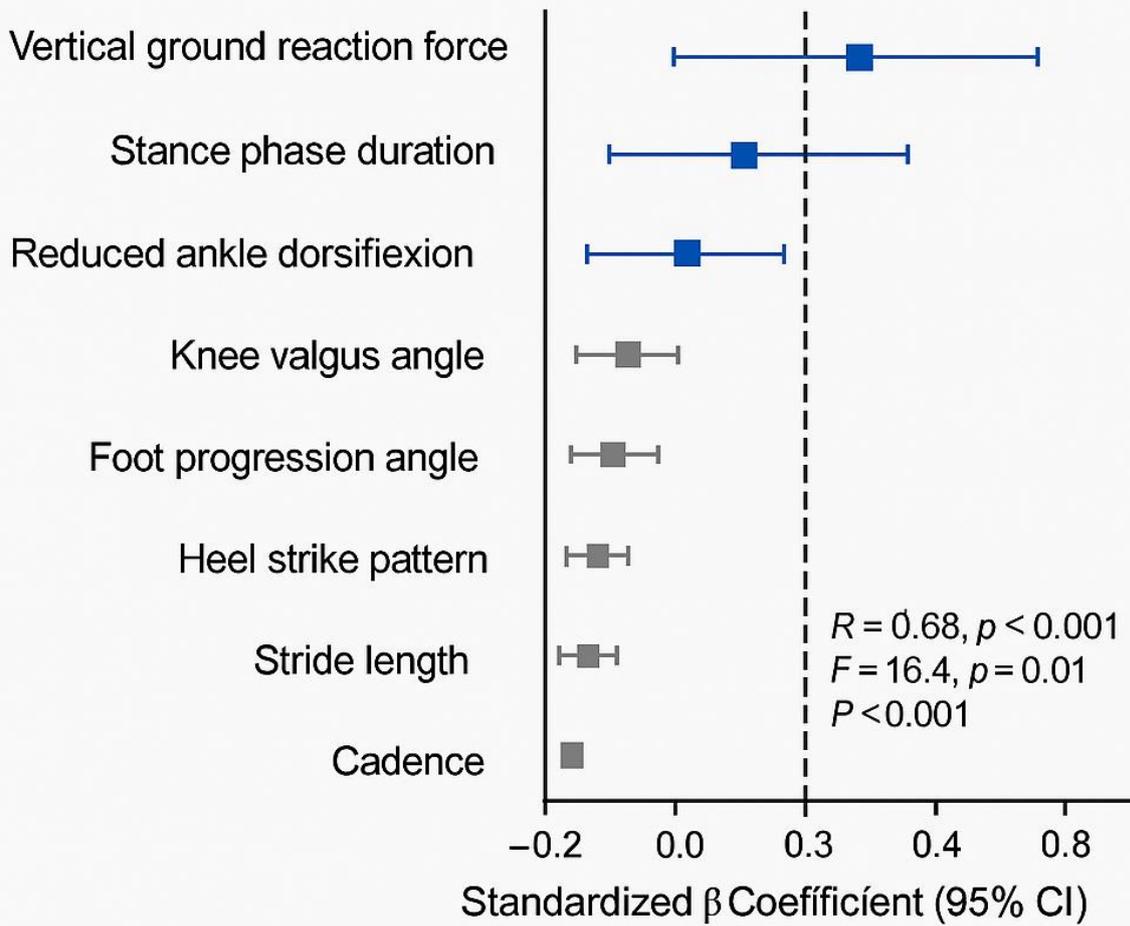


## B Relationship Between Biomechanical Parameters and Arterial Compression Severity in Long-Distance Runners





## D. Multiple Linear Regression Model



$R^2 = 0.68$  Adjusted  $R^2 = 0.64$ ,  $F = 16.4$ ,  $p = 0.001$

Model adjusted for age, BMI and training volume.

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