KMANPUB | Health Nexus Vol. 4 No. 3 (2026): 1-13

Article history:
Received 24 February 2026
Revised 15 June 2026
Accepted 23 June 2026
Initial Publication 29 June 2026
Final Publication 01 July 2026

Comparison of Electromyographic Activity Patterns of Gluteus Maximus, Biceps Femoris, and Tensor Fasciae Latae During Treadmill Walking in Individuals with Piriformis Syndrome

Arsham Sarvi[1], Mohammad Ali Seyed Hoseini[2*]

1. Department of Corrective Exercises, Sr.C., Islamic Azad University, Tehran, Iran
2. Department of Physical Education, Parand C., Islamic Azad University, Parand, Iran

*** Corresponding author email address:** ahoseini78@iau.ac.ir

A r t i c l e I n f o

Article type:
Original Research

How to cite this article:
Sarvi, A. & Seyed Hoseini, M. A. (2026). Comparison of Electromyographic Activity Patterns of Gluteus Maximus, Biceps Femoris, and Tensor Fasciae Latae During Treadmill Walking in Individuals with Piriformis Syndrome. *Health Nexus, 4*(3), 1-13.
https://doi.org/10.61838/kman.hn.5701

A B S T R A C T

Piriformis syndrome is a common neuromuscular disorder characterized by gluteal pain and sciatic nerve irritation, which may alter gait patterns and lower-limb muscle activation. Understanding these alterations can help identify compensatory mechanisms and guide effective rehabilitation strategies. This study aimed to compare the electromyographic activity patterns of the gluteus maximus, biceps femoris, and tensor fasciae latae during treadmill walking in individuals with piriformis syndrome versus healthy controls. In this case-control study, 14 male participants (7 with piriformis syndrome and 7 healthy controls), aged 20–40 years with normal BMI, were recruited. Surface electromyography was recorded from the gluteus maximus, biceps femoris, and tensor fasciae latae of both lower limbs using the ME6000 system during treadmill walking at each participant's preferred speed. Signal processing included band-pass filtering (20–450 Hz), notch filtering at 50 Hz, full-wave rectification, and RMS envelope extraction. Activation onset, activation offset, minimum amplitude, and maximum amplitude were determined from three consecutive gait cycles. Independent t-tests were used for statistical analysis with a significance level of $p < 0.05$. The piriformis syndrome group showed delayed onset and earlier offset across all three muscles. The gluteus maximus demonstrated significantly higher minimum amplitude and lower maximum amplitude in both limbs ($p < 0.05$). The biceps femoris showed significant temporal alterations in both limbs, with reduced maximum amplitude only in the left limb ($p = 0.018$). The tensor fasciae latae demonstrated significant temporal alterations in both limbs, with increased minimum amplitude only in the right limb ($p = 0.019$). The findings suggest that individuals with piriformis syndrome demonstrate altered muscle activation timing and amplitude during gait. However, due to the cross-sectional case-control design, causal relationships and underlying neuromuscular mechanisms cannot be definitively established. Rehabilitation should incorporate neuromuscular control exercises to restore proper activation timing and coordination.

Keywords: *Piriformis syndrome, surface electromyography, muscle activation, gait*

1. Introduction

Piriformis syndrome (PS) is a neuromuscular disorder characterized by gluteal pain and sciatic nerve irritation resulting from compression of the sciatic nerve by the piriformis muscle (1). The piriformis muscle, a flat pyramid-shaped structure located deep within the gluteal region, originates from the anterior surface of the sacrum and inserts into the greater trochanter of the femur (2). Its primary functions include external rotation, abduction, and stabilization of the hip joint during weight-bearing activities (3). The sciatic nerve typically passes inferior to the piriformis; however, anatomical variations in which the nerve passes through or above the muscle may predispose individuals to nerve compression and the development of PS (4, 5).

Despite its clinical significance, PS remains a diagnostic challenge due to symptom overlap with other lumbosacral conditions, including lumbar disc herniation, spinal stenosis, and sacroiliac joint dysfunction (6). Prevalence estimates range from 0.3% to 6% among patients with sciatica, with women affected more frequently than men (7). The condition is commonly observed in middle-aged, physically active individuals, particularly athletes involved in sports requiring repetitive hip rotation (8, 9).

The etiology of PS is multifactorial, encompassing direct trauma, muscle spasm or hypertrophy, anatomical anomalies, biomechanical imbalances including gluteal muscle weakness, and repetitive activities that overload the hip joint (10, 11). Chronic spasm of the piriformis can compress the sciatic nerve, leading to pain, paresthesia, numbness, and motor weakness in the lower extremity (7). Additionally, inflammation and fibrosis within the muscle may perpetuate a cycle of pain and dysfunction (12). The condition significantly impacts quality of life, impairing daily activities and recreational participation (13).

Muscle activation patterns provide critical insights into neuromuscular control and compensatory strategies adopted in musculoskeletal disorders (14). Electromyography (EMG) is a well-established tool for assessing the timing and magnitude of muscle activity during dynamic tasks such as walking (15). In individuals with PS, altered activation patterns have been observed in surrounding hip and thigh muscles, including the gluteus maximus, biceps femoris, and tensor fasciae latae (TFL), reflecting compensatory mechanisms aimed at maintaining pelvic stability while minimizing pain (16, 17).

The gluteus maximus is the largest and most powerful hip extensor, playing a critical role in pelvic control and force generation during gait (18). It acts as a synergist of the piriformis in hip external rotation and extension; weakness or inhibition of the gluteus maximus may place excessive demands on the piriformis, predisposing it to spasm and hypertrophy (19). The biceps femoris, a key component of the hamstring group, contributes to hip extension and knee flexion, with its activity closely coordinated with the gluteus maximus during locomotion (19-21). The TFL assists in hip flexion, abduction, and internal rotation, providing lateral stability to the pelvis during single-leg stance (22). Dysfunction in any of these muscles may disrupt the kinetic chain, leading to altered loading patterns and increased stress on the lumbopelvic region (23).

Recent studies have emphasized the importance of evaluating muscle activation patterns during functional tasks to better understand the pathophysiology of PS and design effective rehabilitation protocols (24, 25).

The relationship between PS and altered muscle activation extends to the entire kinematic chain of the lower extremity. Pain and neural irritation from sciatic nerve compression may be associated with altered activation of gluteal muscles and synergistic muscles such as the hamstrings and TFL (26). These compensatory changes may manifest as delayed onset, shortened or prolonged activation duration, and altered amplitude of EMG signals during gait (16).

Given the complexity of PS and its significant impact on functional performance and quality of life, comprehensive biomechanical investigations are needed to elucidate the relationship between piriformis dysfunction and altered neuromuscular control of hip and thigh muscles. Therefore, the present study aims to compare the electromyographic activity patterns including onset timing, offset timing, minimum amplitude, and maximum amplitude of the gluteus maximus, biceps femoris, and tensor fasciae latae during treadmill walking in individuals with PS versus healthy matched controls.

Health Nexus
E-ISSN: 2981-2569

2. Methods and Materials

2.1. Study Design

This study employed a case-control design to compare electromyographic activity patterns of selected lower extremity muscles between individuals with piriformis syndrome and healthy matched controls during treadmill walking. The research was conducted at the DM Sports Medicine Center in Tehran, Iran. Written informed consent was obtained from all participants prior to enrollment.

2.2. Participants

A total of 14 male participants were recruited for this study, comprising 7 individuals diagnosed with piriformis syndrome and 7 healthy controls. Participants were aged between 20 and 40 years, with a body mass index within the normal range (18.5–24.9 kg/m²). Recruitment was conducted through physiotherapy clinics and specialized health-related social media platforms in Tehran. All participants were right-leg dominant, determined by the preferred kicking leg. The sample size was calculated using G*Power software (version 3.1.9.4) based on an independent t-test comparison, with $\alpha = 0.05$, power = 0.80, and a medium effect size ($d = 0.50$), indicating a minimum of 15 participants per group. However, due to recruitment limitations and strict inclusion criteria, 14 participants meeting all criteria were ultimately enrolled. This final sample size was smaller than the a priori estimated sample, which may have reduced statistical power and increased the likelihood of Type II errors. Therefore, the findings should be interpreted with caution and considered preliminary.

2.2.1. Inclusion and Exclusion Criteria

For the piriformis syndrome group, inclusion criteria consisted of: male gender, age range 20–40 years, normal BMI, absence of recent direct trauma to the gluteal region, and clinical diagnosis of piriformis syndrome established by a specialist physician based on clinical examination and positive provocative tests (Freiberg, Pace, and FAIR tests). Exclusion criteria for this group included: history of surgery in the lumbar, pelvic, femoral, or lower extremity regions; presence of underlying conditions affecting muscle activation patterns including lumbar disc herniation, spinal stenosis, tumor, infection, advanced hip or knee osteoarthritis; use of muscle relaxants, anti-inflammatory drugs, or strong analgesics within 48 hours prior to assessment; presence of lumbar radiculopathy; acute severe pain preventing execution of motor tasks; and history of psychiatric or cognitive disorders impairing cooperation. No imaging or electrodiagnostic confirmation was used; therefore, diagnostic misclassification cannot be completely excluded.

For the healthy control group, inclusion criteria included: male gender, age 20–40 years, normal BMI, and absence of any gluteal or lower extremity pain or dysfunction. Exclusion criteria were identical to those applied to the piriformis syndrome group, with the additional exclusion of any history of piriformis syndrome or related symptoms.

2.3. Outcome Measures and Electromyographic Variables

The primary outcome measures were electromyographic activity parameters of three selected muscles: gluteus maximus, biceps femoris, and tensor fasciae latae. For each muscle and each lower limb (right and left), four specific variables were extracted from the EMG signals: activation onset time (percentage of gait cycle), activation offset time (percentage of gait cycle), minimum muscle activity amplitude (microvolts), and maximum muscle activity amplitude (microvolts). These parameters were derived from three consecutive gait cycles during treadmill walking at each participant's preferred speed (16).

Electromyography Equipment and Instrumentation

Surface electromyography signals were recorded using an ME6000 8-channel electromyography system (Mega Electronics Ltd., Finland) with a sampling rate of 1000 Hz. Disposable Ag/AgCl surface electrodes (Ambu A/S, Denmark) with a diameter of 10 mm were used. A reference electrode was placed on the bony prominence of the lateral epicondyle of the femur, an area with minimal electrical activity. The raw EMG signals were amplified with a gain of 1000 and common-mode rejection ratio > 100 dB. The system was calibrated prior to each testing session according to the manufacturer's specifications. All signals were band-pass filtered online between 10 and 500 Hz to reduce motion artifacts and high-frequency noise. Data acquisition and initial processing were performed using ME6000 software

Health Nexus
E-ISSN: 2981-2569

(Mega Electronics Ltd., Finland) and subsequently exported to MATLAB.

Skin Preparation and Electrode Placement

Prior to electrode placement, the skin over each target muscle was carefully prepared to minimize impedance and optimize signal quality. The procedure involved shaving any excess hair, cleansing the skin with 70% alcohol solution, and gently abrading the epidermis with a fine-grit abrasive pad to remove dead skin cells. Electrodes were positioned according to the SENIAM (Surface Electromyography for the Non-Invasive Assessment of Muscles) guidelines, with an inter-electrode distance of 20 mm center-to-center. Electrode placement for each muscle was as follows (19, 27):

Gluteus maximus: Electrodes were placed at 50% of the line from the sacral hiatus to the greater trochanter, on the muscle belly. The reference electrode was positioned on the sacrum. Electrode orientation was aligned with the muscle fiber direction (oblique downward and outward).

Biceps femoris: Electrodes were positioned at 50% of the line from the ischial tuberosity to the lateral epicondyle of the tibia, on the lateral aspect of the posterior thigh. The reference electrode was placed on the lateral femoral condyle. The electrodes were aligned vertically and parallel to the muscle fibers.

Tensor fasciae latae: Electrodes were placed approximately 10 cm distal to the anterior superior iliac spine, on the anterolateral aspect of the thigh, where the muscle belly is palpable during hip internal rotation. The reference electrode was positioned on the iliac crest. Electrode orientation was aligned with the short muscle fibers in a downward oblique direction.

All electrode placements were marked with semi-permanent ink to ensure consistent positioning for subsequent measurements. Skin-electrode impedance was confirmed to be below 5 kΩ prior to signal acquisition.

Experimental Protocol and Gait Assessment

Testing was conducted in a temperature-controlled laboratory environment (22–24°C) between 9:00 AM and 12:00 PM to minimize circadian variations in muscle activity. Following skin preparation and electrode placement, participants underwent a standardized warm-up consisting of 5 minutes of light stretching and 5 minutes of treadmill walking at a comfortable self-selected pace. The experimental walking protocol was performed on a motorized treadmill (Sportstech, Germany) set at 0% incline. Each participant's preferred walking speed was determined during a 2-minute familiarization period, during which participants were asked to identify the speed at which they felt most comfortable and maintained their natural gait pattern. The preferred speed range was established between 3.5 and 4.5 km/h. Once a stable gait pattern was achieved (typically after 2–3 minutes of walking at the selected speed), continuous EMG data were recorded for 30 seconds. This duration ensured capture of at least 20–25 complete gait cycles. Participants were instructed to maintain a forward gaze, avoid holding the treadmill handrails, and walk naturally without altering their gait pattern. A video camera (Logitech, USA) synchronized with the EMG system recorded the participants' gait cycle to identify heel-strike events.

Signal Processing and Analysis

Raw EMG signals were exported from the acquisition system and processed using MATLAB (version R2021a, MathWorks Inc., USA). The signal processing protocol followed established guidelines for surface EMG analysis. The raw signals were first filtered using a fourth-order zero-lag Butterworth band-pass filter with cutoff frequencies of 20 Hz and 450 Hz to remove movement artifacts and high-frequency noise. A 50 Hz notch filter was applied to eliminate power line interference. The filtered signals were then full-wave rectified to convert all negative components to positive values. A linear envelope was obtained by smoothing the rectified signals using a root mean square (RMS) algorithm with a moving window of 50 milliseconds. The RMS calculation was performed according to the following equation:

$$RMS = \sqrt{(1/N \sum_{i=1}^{N} x_i^2)}$$

Where x_i represents the signal amplitude at sample i and N represents the number of samples within each window. The resultant RMS envelope represented the muscle activity profile throughout the gait cycle (28).

Gait Cycle Segmentation and Parameter Extraction

Gait cycles were segmented based on video-recorded heel-strike events. Each gait cycle was defined from initial heel-strike of one foot (0% of cycle) to the subsequent heel-strike of the same foot (100% of cycle). For each participant, three consecutive and stable gait cycles were selected for

Health Nexus
E-ISSN: 2981-2569

analysis to minimize variability. Heel-strike events were identified by synchronizing the video recordings with the EMG signals. The following parameters were extracted for each muscle and each limb (right and left):

Amplitude-related variables: From the smoothed RMS envelope of each gait cycle, the maximum amplitude (peak) was identified as the highest point of the envelope, representing the highest electrical activity of the muscle. The minimum amplitude was identified as the lowest point of the envelope, representing the baseline activity. Both values were expressed in microvolts (μV). These parameters were averaged across the three selected gait cycles for each participant. Because maximum voluntary isometric contraction (MVIC) normalization was not performed, amplitude variables represent raw EMG values and should be interpreted as relative between-group observations rather than definitive indicators of muscle activation capacity.

Temporal variables: Activation onset and offset times were determined using a threshold-based algorithm. Baseline muscle activity was calculated as the mean RMS amplitude during a quiet standing period (30 seconds) prior to walking. The activation threshold was defined as the mean baseline activity plus three standard deviations. Muscle activation onset was identified as the first time point at which the RMS envelope exceeded this threshold for at least 25 milliseconds continuously. Conversely, activation offset was identified as the time point at which the RMS envelope fell below the threshold for at least 25 milliseconds continuously. Both onset and offset times were normalized and expressed as a percentage of the gait cycle duration. These temporal variables were also averaged across three consecutive gait cycles.

2.4. Statistical Analysis

Statistical analyses were performed using SPSS software (version 26.0, IBM Corp., USA). Descriptive statistics (mean ± standard deviation) were calculated for all demographic and dependent variables. The normality of data distribution was assessed using the Shapiro-Wilk test. Levene's test was employed to evaluate homogeneity of variances between groups. To compare muscle activation parameters between the piriformis syndrome group and healthy controls, independent samples t-tests were conducted for each dependent variable (onset time, offset time, minimum amplitude, and maximum amplitude) for each muscle and each limb. The significance level was set at $\alpha = 0.05$ for all analyses. All statistical assumptions were checked prior to analysis, and no violations were detected. Graphical presentations of results were created using Microsoft Excel (Microsoft Corp., USA) for visual representation of group differences. Given the large number of comparisons performed across muscles, limbs, and EMG variables, the possibility of inflated Type I error should be considered when interpreting statistically significant findings, particularly those with p-values close to 0.05.

3. Findings and Results

3.1. Descriptive Statistics of Participant Characteristics

Descriptive statistics for demographic characteristics of both groups are presented in Table 1. The piriformis syndrome group and healthy control group were comparable in terms of age, height, weight, and body mass index. Independent t-tests confirmed no significant differences between groups for any demographic variable ($p > 0.05$ for all), indicating successful group matching.

Table 1

Demographic Characteristics of Participants (Mean ± SD)

Variable	Piriformis Syndrome Group (n=7)	Healthy Control Group (n=7)	p-value
Age (years)	37.40 ± 6.25	40.06 ± 4.28	0.70
Height (cm)	175.26 ± 8.00	178.33 ± 6.30	0.49
Weight (kg)	72.66 ± 3.16	71.00 ± 6.00	0.44
BMI (kg/m²)	25.01 ± 2.49	25.15 ± 2.98	0.45

BMI: Body Mass Index; SD: Standard Deviation

Health Nexus
E-ISSN: 2981-2569

3.2. *Descriptive Statistics of Electromyographic Variables*

Descriptive statistics for all electromyographic variables (activation onset, activation offset, minimum amplitude, and maximum amplitude) for the three selected muscles gluteus maximus, biceps femoris, and tensor fasciae latae are presented in Table 2. Values are reported as mean ± standard deviation for both right and left limbs in each group. The Shapiro-Wilk test was employed to assess the normality of data distribution for all dependent variables across both groups. Results indicated that all variables were normally distributed ($p > 0.05$ for all), satisfying the assumption for parametric statistical analyses. However, these inferential results should be interpreted in light of the small sample size and the absence of correction for multiple comparisons.

Table 2

Descriptive Statistics of EMG Variables for All Three Muscles (Mean ± SD)

Muscle	Variable	Side	Healthy Group	Piriformis Syndrome Group
Gluteus Maximus	Onset Time (% gait cycle)	Right	4.71 ± 4.28	9.85 ± 4.05
	Onset Time (% gait cycle)	Left	4.28 ± 2.75	9.85 ± 3.38
	Offset Time (% gait cycle)	Right	45.00 ± 2.76	39.85 ± 4.41
	Offset Time (% gait cycle)	Left	44.57 ± 2.63	40.14 ± 4.25
	Minimum Amplitude (μV)	Right	14.28 ± 2.62	18.85 ± 3.76
	Minimum Amplitude (μV)	Left	14.71 ± 3.94	19.00 ± 3.05
	Maximum Amplitude (μV)	Right	204.71 ± 29.20	149.57 ± 27.82
	Maximum Amplitude (μV)	Left	203.71 ± 34.40	150.00 ± 28.09
Biceps Femoris	Onset Time (% gait cycle)	Right	85.42 ± 3.35	91.28 ± 3.14
	Onset Time (% gait cycle)	Left	85.71 ± 4.07	92.28 ± 3.35
	Offset Time (% gait cycle)	Right	13.57 ± 2.82	9.14 ± 2.11
	Offset Time (% gait cycle)	Left	13.28 ± 2.69	8.85 ± 2.19
	Minimum Amplitude (μV)	Right	18.00 ± 1.91	18.14 ± 2.99
	Minimum Amplitude (μV)	Left	18.28 ± 2.81	19.00 ± 4.08
	Maximum Amplitude (μV)	Right	191.14 ± 28.96	153.00 ± 40.80
	Maximum Amplitude (μV)	Left	194.85 ± 31.50	145.57 ± 35.42
Tensor Fasciae Latae	Onset Time (% gait cycle)	Right	1.57 ± 1.39	7.85 ± 2.19
	Onset Time (% gait cycle)	Left	4.00 ± 1.63	8.00 ± 2.00
	Offset Time (% gait cycle)	Right	17.85 ± 1.77	11.28 ± 2.21
	Offset Time (% gait cycle)	Left	18.00 ± 2.16	11.85 ± 2.43
	Minimum Amplitude (μV)	Right	14.42 ± 2.99	20.00 ± 4.54
	Minimum Amplitude (μV)	Left	14.28 ± 4.46	18.27 ± 4.75
	Maximum Amplitude (μV)	Right	307.85 ± 27.35	290.71 ± 42.37
	Maximum Amplitude (μV)	Left	310.14 ± 31.56	287.00 ± 43.61

3.3. *Comparison of EMG Variables Between Groups*

Because multiple independent comparisons were performed, findings with p-values close to 0.05 should be interpreted cautiously and regarded as exploratory.

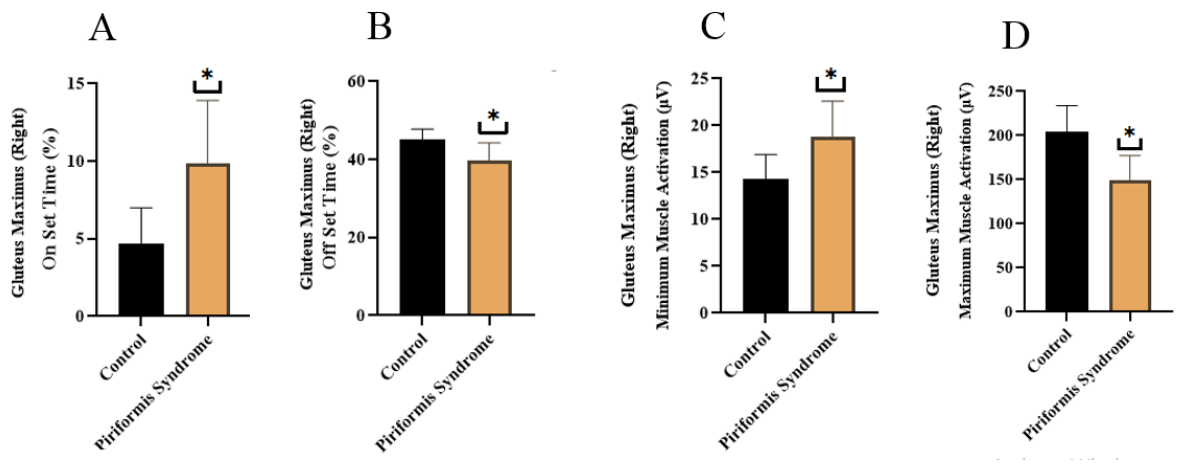
Gluteus Maximus

For the right limb, independent t-tests revealed significant differences between the piriformis syndrome and healthy groups across all four EMG parameters. The onset time was significantly delayed in the piriformis syndrome group (9.85 ± 4.05%) compared to healthy controls (4.71 ± 4.28%; $p = 0.013$) (Figure 1A), while the offset time occurred significantly earlier (39.85 ± 4.41% vs. 45.00 ± 2.76%; $p = 0.023$) (Figure 1B). Additionally, the minimum amplitude was significantly higher in the piriformis syndrome group (18.85 ± 3.76 μV vs. 14.28 ± 2.62 μV; $p = 0.022$) (Figure 1C), whereas the maximum amplitude was significantly lower (149.57 ± 27.82 μV vs. 204.71 ± 29.20 μV; $p = 0.004$) (Figure 1D).

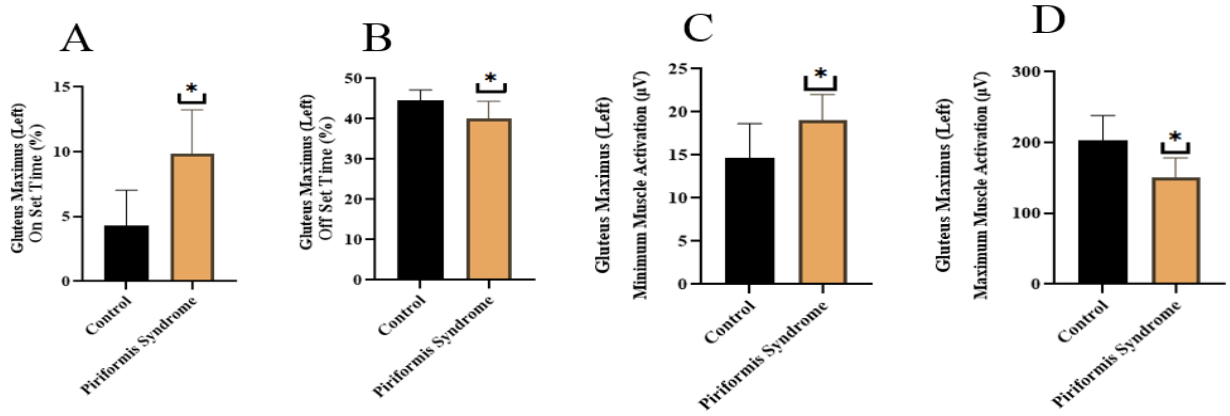
Health Nexus
E-ISSN: 2981-2569

Figure 1

Comparison of EMG parameters for gluteus maximus - right limb



Comparison of EMG parameters for gluteus maximus - left limb



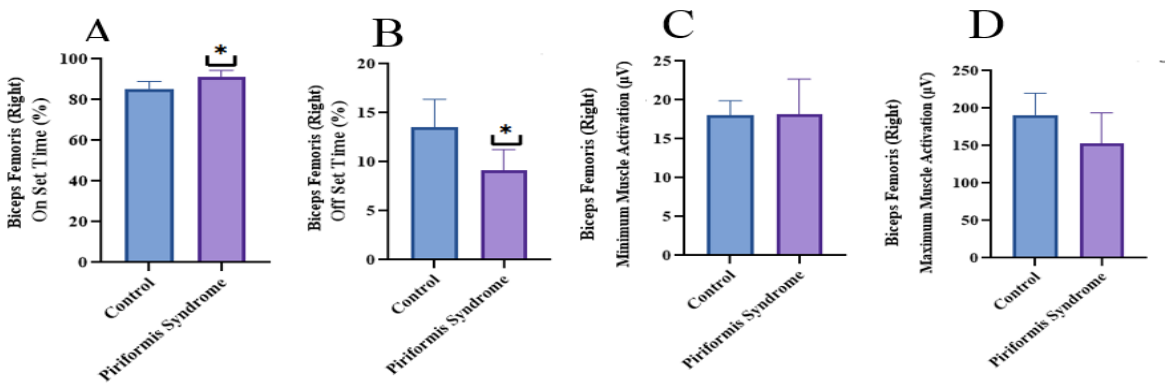
Biceps Femoris

For the right limb, the onset time was significantly delayed in the piriformis syndrome group (91.28 ± 3.14%) compared to healthy controls (85.42 ± 3.35%; p = 0.007) (Figure 3A), and the offset time occurred significantly earlier (9.14 ± 2.11% vs. 13.57 ± 2.82%; p = 0.006) (Figure 3B). However, the minimum amplitude showed no significant difference between groups (18.14 ± 2.99 μV vs. 18.00 ± 1.91 μV; p = 0.941) (Figure 3C), and the maximum amplitude demonstrated only a trend toward lower values in the piriformis syndrome group (153.00 ± 40.80 μV vs. 191.14 ± 28.96 μV; p = 0.067) (Figure 3D).

Figure 2

Comparison of EMG parameters for biceps femoris - right limb

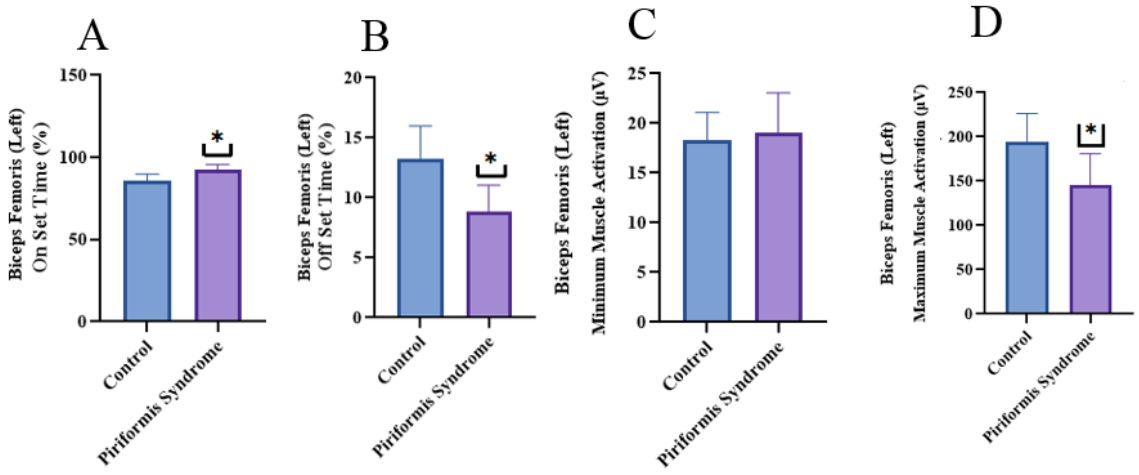
Health Nexus
E-ISSN: 2981-2569



For the left limb, the onset time was significantly delayed in the piriformis syndrome group (92.28 ± 3.35% vs. 85.71 ± 4.07%; p = 0.006) (Figure 4A), and the offset time was significantly earlier (8.85 ± 2.19% vs. 13.28 ± 2.69%; p = 0.006) (Figure 4B). The minimum amplitude showed no significant difference (19.00 ± 4.08 µV vs. 18.28 ± 2.81 µV; p = 0.710) (Figure 4C), while the maximum amplitude was significantly lower in the piriformis syndrome group (145.57 ± 35.42 µV vs. 194.85 ± 31.50 µV; p = 0.018) (Figure 4D).

Figure 3

Comparison of EMG parameters for biceps femoris - left limb



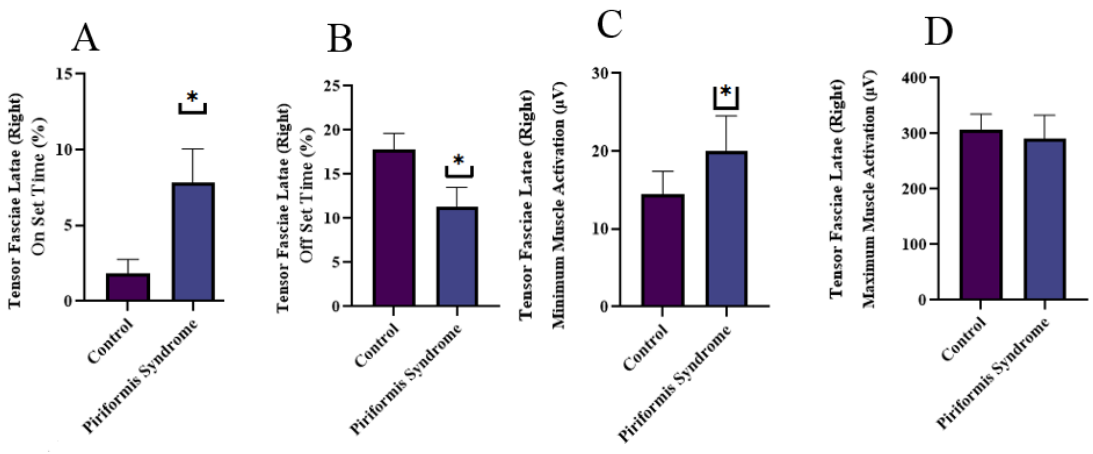
Tensor Fasciae Latae

For the right limb, the onset time was significantly delayed in the piriformis syndrome group (7.85 ± 2.19%) compared to healthy controls (1.57 ± 1.39%; p = 0.001) (Figure 5A), and the offset time occurred significantly earlier (11.28 ± 2.21% vs. 17.85 ± 1.77%; p = 0.001) (Figure 5B). The minimum amplitude was significantly higher in the piriformis syndrome group (20.00 ± 4.54 µV vs. 14.42 ± 2.99 µV; p = 0.019) (Figure 5C), while the maximum amplitude showed no significant difference (290.71 ± 42.37 µV vs. 307.85 ± 27.35 µV; p = 0.386) (Figure 5D).

Figure 4

Comparison of EMG parameters for tensor fasciae latae - right limb

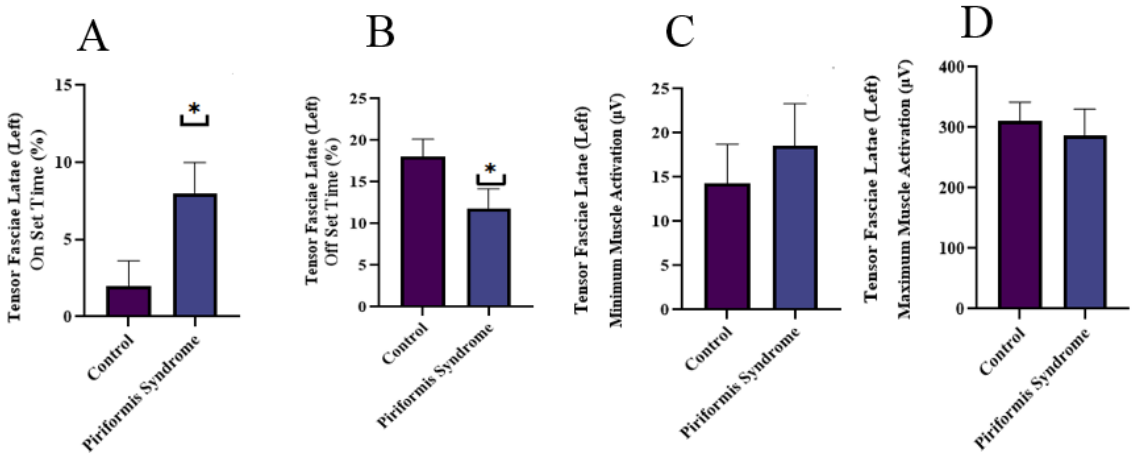
Health Nexus
E-ISSN: 2981-2569



For the left limb, the onset time was significantly delayed in the piriformis syndrome group (8.00 ± 2.00% vs. 4.00 ± 1.63%; p = 0.001) (Figure 6A), and the offset time was significantly earlier (11.85 ± 2.43% vs. 18.00 ± 2.16%; p = 0.001) (Figure 6B). However, the minimum amplitude showed no significant difference (18.27 ± 4.75 µV vs. 14.28 ± 4.46 µV; p = 0.108) (Figure 6C), and the maximum amplitude also showed no significant difference (287.00 ± 43.61 µV vs. 310.14 ± 31.56 µV; p = 0.278) (Figure 6D).

Figure 5

Comparison of EMG parameters for tensor fasciae latae - left limb



4. Discussion

The present study compared electromyographic activity patterns of the gluteus maximus, biceps femoris, and tensor fasciae latae during treadmill walking in individuals with piriformis syndrome versus healthy controls. The findings revealed significant alterations in both temporal and amplitude-related EMG parameters across all three muscles, with the most pronounced changes observed in the gluteus maximus. These findings indicate the presence of altered EMG characteristics during gait in individuals with piriformis syndrome. However, the present design does not permit direct inference regarding specific compensatory neuromuscular mechanisms.

The most consistent finding across all three muscles was delayed onset and earlier offset of muscle activation in the piriformis syndrome group, indicating shortened muscle activity duration during the gait cycle. For the gluteus maximus, onset was delayed by approximately 5% and offset occurred 5% earlier in both limbs. The biceps femoris

Health Nexus
E-ISSN: 2981-2569

demonstrated delayed onset ranging from 4% to 6% and earlier offset ranging from 4% to 5%. Similarly, the tensor fasciae latae showed delayed onset of approximately 4% to 6% and earlier offset of 6% to 7%. These temporal alterations may reflect modified gait-related muscle recruitment; however, pain intensity, symptom severity, and mechanical stress on the piriformis muscle and sciatic nerve were not directly measured (26, 29). The delayed activation of the gluteus maximus may be associated with pain or sciatic nerve irritation, although reflex inhibition was not directly assessed (16, 30), while the earlier offset may indicate altered timing of hip extensor activity; however, forward propulsion and pelvic stability were not directly evaluated using kinetic or kinematic measures (31).

Regarding amplitude-related parameters, the gluteus maximus exhibited significantly higher minimum amplitude and significantly lower maximum amplitude in the piriformis syndrome group compared to healthy controls in both limbs. The increased minimum amplitude may reflect altered baseline muscle activity; however, co-contraction and muscle tone were not directly measured in the present study (32). This finding aligns with studies on chronic low back pain, where patients exhibit elevated paraspinal muscle activity during gait as a protective mechanism (26, 33). The reduced maximum amplitude in the gluteus maximus may be associated with altered motor unit recruitment, although force-generating capacity, neural drive, and pain-related inhibition were not directly assessed (14, 29).

The biceps femoris demonstrated less consistent amplitude changes. Minimum amplitude showed no significant differences between groups in either limb, while maximum amplitude was significantly reduced only in the left limb, with a trend toward reduction in the right limb. This asymmetry should be interpreted cautiously because symptom laterality and severity were not quantified in the current study (1, 7). The reduction in maximum amplitude may indicate altered activation magnitude, but reduced force-generating capacity, neural inhibition, and disuse atrophy were not directly measured (20, 21). However, the preservation of minimum amplitude suggests that baseline EMG amplitude was relatively maintained, possibly due to the need for ongoing knee stabilization (34).

The tensor fasciae latae exhibited minimal amplitude changes, with increased minimum amplitude observed only in the right limb and no significant differences in maximum amplitude in either limb. This pattern suggests that while temporal alterations were observed in the tensor fasciae latae, its amplitude changes were less consistent; explanations related to muscle size or force generation remain speculative because force output was not measured (19, 23). Alternatively, the preserved maximum amplitude may indicate that the tensor fasciae latae was less affected in this sample, but inhibitory mechanisms cannot be inferred from the present data (27).

The findings align with previous research on altered muscle activation in musculoskeletal disorders. Studies on chronic low back pain have reported delayed activation of trunk muscles, increased co-contraction, and reduced force output, similar to the patterns observed in the gluteus maximus in our study (26, 32). Individuals with femoroacetabular impingement syndrome have also exhibited altered coordination of deep hip muscles, including delayed activation of the gluteus maximus and increased activity of the tensor fasciae latae (16, 30). Leung et al. (35) reported altered hamstring activation patterns in elite football players with low back pain, supporting the concept that proximal dysfunction can influence distal muscle recruitment. These similarities suggest that pain and joint dysfunction may be associated with comparable neuromuscular adaptations, although the present findings should not be interpreted as evidence of a direct causal pathway (14, 29).

However, some studies have reported different findings. Fattahi et al. (36) found that general fatigue led to increased muscle activity as a compensatory response to maintain balance, contrasting with the reduced maximum amplitude observed in our study. This difference may reflect distinct underlying mechanisms, such as fatigue-related compensation versus pain-related alteration; however, this interpretation remains speculative because neither fatigue nor pain intensity was experimentally manipulated or quantified in the present study. Shayesteh et al. (37) observed decreased EMG activity following resistance training, interpreted as improved neuromuscular efficiency. In contrast, reductions observed in the present study should be interpreted as altered EMG behavior associated with piriformis syndrome rather than definitive evidence of dysfunction.

Health Nexus
E-ISSN: 2981-2569

From a clinical perspective, these findings have important implications. Traditional rehabilitation programs often focus on stretching the piriformis and strengthening the gluteal muscles (1, 7). However, our findings may indicate the presence of altered neuromuscular control during gait; direct measurements of muscle strength and motor control were not obtained. Therefore, rehabilitation may consider motor control exercises aimed at improving the timing and coordination of muscle activation, rather than solely focusing on strengthening or stretching (14, 17). Exercises emphasizing early activation of the gluteus maximus during stance phase, such as single-leg squats and step-ups, may be considered to address delayed onset (27). Additionally, activities that promote pelvic stability and proper hip-kinetic chain alignment, such as balance training and perturbation exercises, may help improve activation patterns, although this requires confirmation in interventional studies (19, 23).

The limitations of this study should be acknowledged. First, the sample size was relatively small (seven individuals per group), which may limit statistical power and generalizability. Second, the study only included male participants, limiting generalizability to female populations who are more commonly affected (7). Third, the study only assessed gait, and findings may not apply to other functional activities. Fourth, pain levels and symptom severity were not assessed, which could have provided valuable information about the relationship between clinical symptoms and EMG alterations. Fifth, piriformis syndrome diagnosis was established primarily through clinical examination and provocative tests without imaging or electrodiagnostic confirmation; therefore, the possibility of diagnostic misclassification cannot be completely excluded. Sixth, EMG amplitude values were not normalized to MVIC, limiting comparisons of muscle activation magnitude between participants. Seventh, the study did not assess gait kinematics, kinetic variables, muscle strength, or functional disability, restricting interpretation of the underlying biomechanical mechanisms. Finally, no correction for multiple statistical comparisons was applied, which may have increased the risk of Type I error.

Despite these limitations, the study has several strengths. The use of surface electromyography provided objective data on selected muscle activation patterns during treadmill walking. The inclusion of both limbs allowed preliminary comparison of bilateral EMG patterns. The focus on three key hip-related muscles provides useful preliminary information on EMG behavior in piriformis syndrome.

5. Conclusion

Individuals with piriformis syndrome demonstrated altered temporal and amplitude-related EMG characteristics during treadmill walking compared with healthy controls. These findings suggest the presence of modified neuromuscular behavior during gait. Nevertheless, the relatively small sample size, absence of EMG normalization, lack of biomechanical and pain-related measures, absence of correction for multiple comparisons, and case-control design warrant cautious interpretation. Future studies with larger samples, normalized EMG procedures, comprehensive biomechanical assessments, symptom severity measures, and longitudinal or interventional designs are needed to clarify the clinical significance of these findings.

Authors' Contributions

Arsham Sarvi contributed to the conceptualization of the study, data collection, data analysis, interpretation of findings, manuscript drafting, and manuscript revision. Mohammad Ali Seyed Hosseini contributed to study supervision, methodological design, interpretation of results, critical revision of the manuscript, and final approval of the submitted version. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Declaration

The authors declare that artificial intelligence tools were used only to assist with language editing, translation, and improvement of the manuscript's readability. All conceptualization, study design, data collection, data analysis, interpretation of findings, and final approval of the manuscript were performed by the authors. The authors take full responsibility for the accuracy, integrity, and originality of the content.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

Health Nexus
E-ISSN: 2981-2569

Acknowledgments

The authors thank the participating centers, clinicians, adolescents, and guardians who made the study possible.

Declaration of Interest

The authors report no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics Considerations

This study was conducted in accordance with the principles of the Declaration of Helsinki. All participants were informed about the study procedures, potential risks, and benefits before participation. Written informed consent was obtained from all participants prior to data collection. Participants were assured of the confidentiality and anonymity of their personal information and were free to withdraw from the study at any stage without any consequences.

References

1. Probst D, Stout A, Hunt D. Piriformis Syndrome: A Narrative Review Of The Anatomy, Diagnosis, And Treatment. PM&R. 2019;11:S54-S63. [PMID: 31102324] [DOI]
2. Jawish RM, Assoum HA, Khamis CF. Anatomical, Clinical And Electrical Observations In Piriformis Syndrome. Journal of Orthopaedic Surgery and Research. 2010;5(1):3. [PMID: 20180984] [PMCID: PMC2828977] [DOI]
3. Michel F, Decavel P, Toussirot E, Tatu L, Aleton E, Monnier G. The Piriformis Muscle Syndrome: An Exploration Of Anatomical Context, Pathophysiological Hypotheses And Diagnostic Criteria. Annals of Physical and Rehabilitation Medicine. 2013;56(4):300-11. [PMID: 23684469] [DOI]
4. Cassidy L, Walters A, Bubb K, Shoja MM, Shane Tubbs R, Loukas M. Piriformis Syndrome: Implications Of Anatomical Variations, Diagnostic Techniques, And Treatment Options. Surgical and Radiologic Anatomy. 2012;34(6):479-86. [PMID: 22327640] [DOI]
5. Lewis S, Jurak J, Lee C, Lewis R, Gest T. Anatomical Variations Of The Sciatic Nerve, In Relation To The Piriformis Muscle. Translational Research in Anatomy. 2016;5:15-9. [DOI]
6. Alrabai HM, Alrashidi Y, Aletebi RAA, Mufti TMA, Alali MYA, Alnazhan ASA. Piriformis Syndrome Is Often Overlooked As A Cause Of Gluteal Pain And Sciatica: Diagnostic Challenges And The Role Of Imaging—A Narrative Review. HSS Journal®. 2026;22(2):192-6. [PMID: 41328333] [PMCID: PMC12664778] [DOI]
7. Jankovic D, Peng P, Van Zundert A. Brief Review: Piriformis Syndrome: Etiology, Diagnosis, And Management. Canadian Journal of Anesthesia/Journal canadien d'anesthésie. 2013;60(10):1003-12. [PMID: 23893704] [DOI]
8. Morimoto Y, Oshikawa T, Imai A, Okubo Y, Kaneoka K. Piriformis Electromyography Activity During Prone And Side-Lying Hip Joint Movement. Journal of Physical Therapy Science. 2018;30(1):154-8. [PMID: 29410588] [PMCID: PMC5788797] [DOI]
9. Neal C, Gelatt T, Toma M. Prevalence And Clinical Patterns Of Piriformis Syndrome Among Actively Competing And Retired Elite Hockey Players. Sports. 2026;14(3):95. [PMID: 41893586] [PMCID: PMC13030342] [DOI]
10. Barzak B, Hankus K, Mielczarek A, Wozniak S. The Piriformis Muscle Syndrome–Anatomy, Diagnosis And The Role Of Physiotherapy. A Review. Medical Journal of Cell Biology. 2023;11(2):39-43. [DOI]
11. Chang C, Jeno SH, Varacallo MA. Anatomy, Bony Pelvis And Lower Limb: Piriformis Muscle: StatPearls Publishing; 2023.
12. Carro LP, Hernando MF, Cerezal L, Navarro IS, Fernandez AA, Castillo AO. Deep Gluteal Space Problems: Piriformis Syndrome, Ischiofemoral Impingement And Sciatic Nerve Release. Muscles, Ligaments and Tendons Journal. 2016;6(3):384. [PMID: 28066745] [PMCID: PMC5193530] [DOI]
13. Othman IK, Raj NB, Siew Kuan C, Sidek S, Wong LS, Djearamane S. Association Of Piriformis Thickness, Hip Muscle Strength, And Low Back Pain Patients With And Without Piriformis Syndrome In Malaysia. Life. 2023;13(5):1208. [PMID: 37240853] [PMCID: PMC10222669] [DOI]
14. Hodges PW, Mclean L, Hodder J. Insight Into The Function Of The Obturator Internus Muscle In Humans: Observations With Development And Validation Of An Electromyography Recording Technique. Journal of Electromyography and Kinesiology. 2014;24(4):489-96. [PMID: 24788026] [DOI]
15. Lencioni T, Carpinella I, Rabuffetti M, Marzegan A, Ferrarin M. Human Kinematic, Kinetic And Emg Data During Different Walking And Stair Ascending And Descending Tasks. Scientific Data. 2019;6(1):309. [PMID: 31811148] [PMCID: PMC6897988] [DOI]
16. Diamond LE, Van Den Hoorn W, Bennell KL, Wrigley TV, Hinman RS, O'Donnell J. Deep Hip Muscle Activation During Squatting In Femoroacetabular Impingement Syndrome. Clinical Biomechanics. 2019;69:141-7. [PMID: 31351343] [DOI]
17. Selkowitz DM, Beneck GJ, Powers CM. Comparison Of Electromyographic Activity Of The Superior And Inferior Portions Of The Gluteus Maximus Muscle During Common Therapeutic Exercises. Journal of Orthopaedic & Sports Physical Therapy. 2016;46(9):794-9. [PMID: 27494053] [DOI]
18. Elzanie A, Borger J. Anatomy, Bony Pelvis And Lower Limb, Gluteus Maximus Muscle: StatPearls Publishing; 2023.
19. Flack NAMS, Nicholson HD, Woodley SJ. A Review Of The Anatomy Of The Hip Abductor Muscles, Gluteus Medius, Gluteus Minimus, And Tensor Fascia Lata. Clinical Anatomy. 2012;25(6):697-708. [PMID: 22109658] [DOI]
20. Rodgers CD, Raja A. Anatomy, Bony Pelvis And Lower Limb, Hamstring Muscle2019.
21. Timmins R, Woodley S, Shield A, Opar D. Anatomy Of The Hamstrings. Prevention and Rehabilitation of Hamstring Injuries: Springer; 2020. p. 1-30. [PMID: 32599271] [DOI]
22. Agur AM, Dalley AF. Moore'S Essential Clinical Anatomy: Lippincott Williams & Wilkins; 2018.
23. Neumann DA. Kinesiology Of The Musculoskeletal System: Foundations For Rehabilitation: Mosby Elsevier; 2010.
24. Aali S, Rezazadeh F, Sheykhalizadeh H, Qasemi M, Akman O, Badicu G. Frequency Spectrum Analysis Of Ground Reaction Forces During Running In Individuals With Piriformis

Health Nexus
E-ISSN: 2981-2569

Syndrome Following An Eight-Week Neuromuscular Training Program. Journal of Men's Health. 2026;22(2). [DOI]
25. Jaiswal V, Sedhunivas R, Nikhilesh A. A Literature Review Of Effectiveness Of Dynamic Stretching Versus Met On Pain And Flexibility In Piriformis Syndrome. Scientific Hub of Applied Research in Emerging Medical science & technology. 2026;5(2):399-407. [DOI]
26. Van Dieën JH, Selen LP, Cholewicki J. Trunk Muscle Activation In Low-Back Pain Patients, An Analysis Of The Literature. Journal of Electromyography and Kinesiology. 2003;13(4):333-51. [PMID: 12832164] [DOI]
27. Bishop BN, Greenstein J, Etnoyer-Slaski JL, Sterling H, Topp R. Electromyographic Analysis Of Gluteus Maximus, Gluteus Medius, And Tensor Fascia Latae During Therapeutic Exercises With And Without Elastic Resistance. International Journal of Sports Physical Therapy. 2018;13(4):668. [PMID: 30140560] [PMCID: PMC6088126] [DOI]
28. Khushaba RN, Kodagoda S, Takruri M, Dissanayake G. Toward Improved Control Of Prosthetic Fingers Using Surface Electromyogram (Emg) Signals. Expert Systems with Applications. 2012;39(12):10731-8. [DOI]
29. Garland SJ, Gray VL, Knorr S. Muscle Activation Patterns And Postural Control Following Stroke. Motor Control. 2009;13(4):387-411. [PMID: 20014647] [DOI]
30. Diamond LE, Van Den Hoorn W, Bennell KL, Wrigley TV, Hinman RS, O'Donnell J. Coordination Of Deep Hip Muscle Activity Is Altered In Symptomatic Femoroacetabular Impingement. Journal of Orthopaedic Research. 2017;35(7):1494-504. [PMID: 27513847] [DOI]
31. Andriacchi T, Andersson G, Örtengren R, Mikosz R. A Study Of Factors Influencing Muscle Activity About The Knee Joint. Journal of Orthopaedic Research. 1983;1(3):266-75. [PMID: 6481510] [DOI]
32. Stokes IA, Gardner-Morse MG, Henry SM. Abdominal Muscle Activation Increases Lumbar Spinal Stability: Analysis Of Contributions Of Different Muscle Groups. Clinical Biomechanics. 2011;26(8):797-803. [PMID: 21571410] [PMCID: PMC3157598] [DOI]
33. Mannion AF, Taimela S, Müntener M, Dvorak J. Active Therapy For Chronic Low Back Pain: Part 1. Effects On Back Muscle Activation, Fatigability, And Strength. Spine. 2001;26(8):897-908. [PMID: 11317112] [DOI]
34. Van Der Made A, Wieldraaijer T, Kerkhoffs G, Kleipool R, Engebretsen L, Van Dijk C. The Hamstring Muscle Complex. Knee Surgery, Sports Traumatology, Arthroscopy. 2015;23(7):2115-22. [PMID: 24190369] [DOI]
35. Leung FT, Mendis MD, Stanton WR, Hides JA. The Relationship Between The Piriformis Muscle, Low Back Pain, Lower Limb Injuries And Motor Control Training Among Elite Football Players. Journal of Science and Medicine in Sport. 2015;18(4):407-11. [PMID: 25027772] [DOI]
36. Fatahi M, Minoonejad H, Alizadeh MH. The Relationship Between Proximal And Distal Lower Limb Muscle Activities With Time To Stabilization. Journal of Research in Rehabilitation Sciences. 2013;9(2):232-42.
37. Shayesteh M, Farahpour N, Jafarnezhadgero A. Comparisons Of The Effects Of Squat And Leg Press Exercises On The Emg Activity Of Quadriceps Femoris Muscles During Step Descending Activity. Journal of Applied Exercise Physiology. 2019;15(29):143-54.

Health Nexus
E-ISSN: 2981-2569