



The efficacy of conservative management of micro-traumatic posterior shoulder instability

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Background: Microtraumatic posterior shoulder instability (PSI) is characterized by symptomatic posterior translation of the glenohumeral joint. A common etiology is a gradual overload of glenohumeral joint structures. The recommend initial treatment for microtraumatic PSI is rehabilitation; however, the evidence to support this recommendation is limited. The aim of this study is to investigate the patient-reported outcome measures and return to sport success of participants with microtraumatic PSI who participate in a posterior instability rehabilitation program.

Methods: In the single-group study design, 24 shoulders in 22 sporting participants (17 male, 5 females; mean age, 21.1 years, standard deviation 10.1 years) diagnosed with microtraumatic PSI undertook the Watson Posterior Instability Program (WIP-p) over 24 weeks. Outcome measures included the Melbourne Instability Shoulder Score and Western Ontario Shoulder Instability Index at baseline, 6, 12, and 24 weeks. Failure of conservative management and time to return to sport was measured. Treatment effects were determined using linear mixed models, with 95% confidence intervals. Significance was set at 0.05.

Results: After 24 weeks of the WIP-p, participants had significant improvements at 12 (effect size or standardized mean difference (SMD): 1.1, $P < .001$) and 24 weeks (SMD: 1.8, $P < .001$) on the Western Ontario Shoulder Instability Index and significant improvements at 6 (SMD: 0.74, $P = .036$), 12 (SMD: 0.41, $P = .007$) and 24 weeks (SMD: 1.7, $P < .001$) on the Melbourne Instability Shoulder Score. For return to sport, 20 of the 22 (90.1%) participants returned to full activity at the 24-week time point, while two went on to have reconstructive surgery.

Discussion and Conclusion: The WIP-p resulted in a high level of return to sport and significantly improved functional outcomes in patients with microtraumatic PSI. A small proportion of sporting participations with microtraumatic PSI may fail conservative rehabilitation and require surgical consideration.

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Ramsay Health Research and Ethics Committee approved this study (Project 165).

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Posterior shoulder instability (PSI) is the dislocation, subluxation, or translation of the humeral head posteriorly from the center of the glenoid fossa, which interferes with joint function and produces symptoms.^{1,41,43} The prevalence of PSI is reported to be between 2 and 10% of all shoulder instabilities^{5,28,34,36}; however, this figure accounts primarily for acute posterior dislocations

presenting to emergency departments and fails to represent those with chronic posterior subluxations or small but symptomatic posterior translations.³⁴ The true prevalence of PSI is unknown. Depending on the severity, PSI may affect activities of daily living, occupational participation, and sporting performance.^{20,41,21,58}

A recent Delphi study came to a large consensus on the classification of PSI, which included traumatic, atraumatic, or microtraumatic subgroups.³² The initial mechanism of injury to the shoulder is what determines the PSI subgroup classification.³² Traumatic PSI is associated with significant shoulder trauma, (such as a fall on an outstretched flexed and internally rotated shoulder) a full dislocation and structural damage to the glenohumeral joint (ie, reverse Bankart lesions and reverse humeral avulsion of the glenohumeral ligament). Atraumatic PSI has no seemingly obvious mechanism of injury or cause of onset but is associated with poor motor control of the scapula and humeral head, a higher rate of shoulder congenital abnormalities (eg, glenoid retroversion and hyperplasia), and a lack of structural lesions. Microtraumatic PSI is caused by tasks with a gradual or repetitive overload of posterior joint structures, such as months of back-to-back tennis with a heavier than usual racquet. Patients with microtraumatic PSI are more varied with the contributing factors to their presentation; however, this subgroup can present with acquired structural lesions of the glenohumeral joint (eg, labral tears) due to the repetitive overload of glenohumeral joint structures.^{32,55}

Microtraumatic PSI is becoming more recognized as a pathology that affects sporting populations.^{34,11,46,55} The shoulder of throwing or throwing type athletes, such as tennis players, cricketers, and baseballers are subject to high, repetitive forces, particularly in the follow-through phase of their serve or throw as the posterior capsulolabral complex and rotator cuff work to decelerate the arm.³⁴ Alternatively, posteriorly directed axial loads to the shoulder applied repetitively may be the mechanism of injury in sports, such as swimming (hand entry through the pull phase), gymnastics, and overhead weight lifting.³⁴ These mechanisms can cause microtrauma to the posterior capsulolabral complex and the articular surface of the posterior rotator cuff muscles, leading to breakdown of passive and/or active stabilizers and subsequent posterior humeral head subluxation.³⁴ This attenuation of shoulder support may be structural (eg, posterior labral tears, articular surface rotator cuff tears, and acquired capsular laxity), nonstructural (eg, a loss of scapular and humeral head motor control, endurance, and strength), or a combination of both. Factors that may contribute to the development of microtraumatic PSI in the presence of microtrauma include scapular dyskinesia (typically a lack of scapular upward rotation) and reduced muscle strength of the posterior deltoid and rotator cuff.^{26,32,54}

Selection of the most appropriate management for PSI can be challenging if the classification and contributing factors are not defined. There is a growing body of evidence that shows patients with traumatic, structural PSI have better outcomes with surgical stabilization, especially if they participate in contact or overhead sports.^{6,57} Patients with atraumatic PSI typically respond well to a rehabilitation program focusing on normalizing scapular mechanics, posterior deltoid, and posterior rotator cuff muscles.²⁶ Due to the variability in presentation in patients with microtraumatic PSI, the evidence for best management is still evolving, though experts recommend 6 months of targeted shoulder rehabilitation, and if this fails, surgery may be considered.³⁵ The current guidance on rehabilitation content and clinical decision-making is limited,²⁶ with some evidence for initial motor control movement reeducation, with a focus on scapular, posterior deltoid, and rotator cuff training, and then progressing to more functional exercises.^{4,26} To date, no PSI studies (traumatic, microtraumatic, or atraumatic) evaluating the efficacy of a rehabilitation program have reported

enough detail (such as exercise examples, load, and dosage) for practitioners to replicate their rehabilitation programs in the clinical setting.

There is a need to understand the impact of rehabilitation on patients with microtraumatic PSI who participate in sport. The aim of this study was to investigate the effect of the Watson Instability Program for Posterior Shoulder Instability (WIP-p) on instability-specific outcomes and return to the sport of patients with microtraumatic PSI. The WIP-p⁵¹ is based on the WIP¹ for multidirectional instability (MDI),^{49,50} which has proven efficacy in this population.^{47,52} We hypothesize that the majority of patients with microtraumatic PSI will have significant improvements on patient-reported outcome measures (PROMs) and successful return to sport (at preinjury level) after an intervention with the WIP-p.

Materials and method

Study design and participant inclusion criteria

This single-group intervention study had ethical approval from Ramsay Health and Human Research Ethics Committee (Project No. 165) and was conducted at the Melbourne Shoulder Group and Melbourne Orthopaedic Group, Victoria, Australia.

Participants with clinically diagnosed microtraumatic PSI who competed in sports were recruited between February 2016 and February 2018. The diagnosis of microtraumatic PSI was confirmed by one of the two experienced shoulder surgeons (G.H. and S.Bawrood) or four experienced shoulder physiotherapists (L.W, S.Balster, R.L, and S.W). The preliminary diagnosis of microtraumatic PSI was made via the patient's reported history of the mechanism of onset,³² which included a period of repetitive overload in positions of (typically combined) glenohumeral joint flexion, horizontal adduction, and internal rotation.^{8,32,54} The diagnosis of microtraumatic PSI was confirmed by a battery of clinical tests that was selected based on a recent Delphi study,³² a systematic review of test diagnostic accuracy in PSI⁸ and instability tests in a previous shoulder instability randomized controlled trial.⁴⁷ Included patients must have a positive result for at least 2 of the 3 following tests; (i) posterior apprehension test, (ii) posterior draw test in 0–30° abduction, and (iii) posterior draw test in 80–120° abduction. A positive test included apprehension (including muscle guarding) and not just signs of laxity or pain alone.²⁵ All participants had a 3T magnetic resonance imaging (MRI) scan. Participants were excluded if they had traumatic or atraumatic PSI, anterior shoulder instability, or a significant rotator cuff tear (>1 cm). Traumatic shoulder instability was defined as contact with an external object (eg, a fall, impact with another body or surface) with conscious awareness by the patient of a subluxation or dislocation in association with a sudden onset of pain.⁴⁷ Atraumatic PSI was defined as symptom onset with no seemingly obvious mechanism of injury or cause for onset.³² Anterior instability was diagnosed with a positive anterior apprehension test or structural lesions on MRI indicative of previous anterior dislocation (ie, Bankart lesions and Hill Sachs lesion),⁴⁴ and MRI scans were used to detect significant rotator cuff tears.¹³ Patients with acquired structural lesions to the posterior capsule-labral complex only (the presence of a structural lesion in the absence of a reported glenohumeral joint trauma) could still be included as acquired posterior structural lesions may be a feature of microtraumatic PSI.^{32,54}

Participants attended physiotherapy once a week for 12 weeks then once a fortnight for an attentional 12 weeks (18 sessions in total), where they were prescribed and progressed a set of home exercises as per the WIP-p protocol. A 24-week protocol was chosen based on the results of a previous randomised controlled trial in



Figure 1 Scapular correction into upward rotation during active flexion.

MDI, revealing that the largest treatment effects and return to sport did not occur until 6 months.⁴⁷

Intervention

The WIP-p has an assessment and intervention component. The assessment component involves determining the effect of therapist-assisted scapular and humeral head correction, which has been previously described.^{49,51} To assess the effect of scapular correction, the therapist asks the patient to perform a clinical test (typically active flexion and abduction range of motion), observing the presence of any scapular dyskinesis though range (typically a lack of upward scapular rotation or elevation) in association with the patient's symptoms. The therapist then manually assists the scapula to correct the faulty motor pattern (typically facilitating scapular upward rotation or elevation) and reassess the test, noting any improvement in range of motion, through range subluxations, pain, or any other reported symptom (Fig. 1). To assess the effect of humeral head correction, the therapist typically chooses a clinical test that will stress the posterior joint capsule (typically active flexion or horizontal flexion) palpating and observing any symptomatic posterior humeral head translation. The therapist then provides a posterior-to-anterior support to the humeral head to prevent posterior humeral head translation and reassess the test,



Figure 2 Posterior humeral head correction during horizontal adduction.

again noting improvement in the range of motion, through range subluxations, pain, or any other reported symptom (Fig. 2). Often PSI patients will require a combination of scapular and humeral head correction. The effect of correction determines what patient-specific scapular position the patient will need to retrain. For example, if the therapist-assisted scapular position that improved symptoms was upward rotation, then scapular upward rotation is the position to retrain. The effect of humeral head control assists in confirming the diagnosis of PSI as well as confirming the positions where the patient loses posterior humeral head control. For example, if the patient loses posterior humeral head control when their shoulder is moved across into the sagittal (flexion) plane, then posterior humeral head control should be well-established in the coronal plane prior to attempting to rehabilitate in the sagittal plane.^{54,51}

The intervention component has 5 stages, which has been previously published, outlining in detail, exercise drills, dosages, and load progressions.⁵¹ Stage 1 (scapular phase) involves regaining motor control of the scapula, using the position found best in the effect of correction. This typically involves an upward rotation, elevation drill (Fig. 3), as these patients often lack sufficient scapular upward rotation.⁵¹ Once patient have regained control with the weight of the arm then a scapular resistance band is added, which is a TheraBand (TheraBand, Akron, OH, USA) looped around the scapula and anchored anywhere to resist the patient's drill (Fig. 4).^{53,49} The scapular resistance band enhances scapular motor recruitment,⁹ and its use is often continued with other glenohumeral joint exercises. Once the patient has regained



Figure 3 Scapular upward rotation drill in standing.



Figure 4 Scapular upward rotation drill with scapular resistance band (denoted by arrow) anchored to the patient's foot to resist upward rotation.

scapular motor control, they can then commence regaining motor control of posterior humeral head by activating the shoulder extensors (Fig. 5A and B) and external rotators (Fig. 6A and B), initially with TheraBand then with weights (0.5–1–2 kg+).⁵¹ Stage 2 involves establishing scapular control in higher ranges of elevation with extension rows at 45 degrees abduction (Fig. 7). Stage 3 involves establishing scapular control in 90 degrees of elevation (Fig. 8) before adding on external rotation exercises with TheraBands and then weights (1–2.5kg+) (Fig. 9A and B) in the coronal plane. Stage 4 slowly progresses patients into the scapular plane (Fig. 10) then sagittal plane (Fig. 11) and to gain control over once vulnerable positions. Stage 5 is the sports-specific and functional stage, where exercises are tailored to the needs and goals of the patient.⁵¹

Outcome measures

The primary outcomes were the Melbourne Instability Shoulder Score (MISS) and Western Ontario Shoulder Index (WOSI) at baseline, 6, 12, and 24 weeks.^{17,29,48} These PROMS are valid, reliable and responsive to measuring change in the shoulder instability population.^{15,17,29,48} Development of the MISS and WOSI has been reported, and both have good test-retest reliability.^{29,33,48} The MISS considers four subcategories of pain, instability, function, and occupational or sporting demands. The overall score ranges from 0 to 100 points, with a higher score indicating a higher functioning

shoulder. The WOSI covers four domains, including physical symptoms, sport or recreation, lifestyle, and emotional function. The overall score ranges from 0 to 2100 points, with a higher score indicating a higher level of shoulder disability.^{17,29,33} To facilitate interpretation, the total score of each questionnaire was converted into a percentage of a normal healthy shoulder (where 100% represents no shoulder deficit).^{17,33,48} The minimally clinically important difference for the converted total score of the MISS is 5 points⁴⁸ and for the WOSI, is 10.4 points.³³

Return to previous level of sport success (yes or no) was reported at the 24-week time point. Failure of conservative management (defined as requiring surgery and/or unable to return to sporting participation due to the shoulder) was recorded.

Data analysis

The MISS and WOSI were analyzed using linear mixed models (with 95% confidence intervals) due to their advantages in modeling repeated measures over time and adjusting for baseline scores.¹⁹ Missing data were accounted for by restricted maximum likelihood estimation within the linear mixed models.²² To provide a magnitude of the effect, the standardized mean difference (SMD), defined as the mean difference or standard deviation (SD) of the mean difference, was calculated for the MISS and the WOSI. The

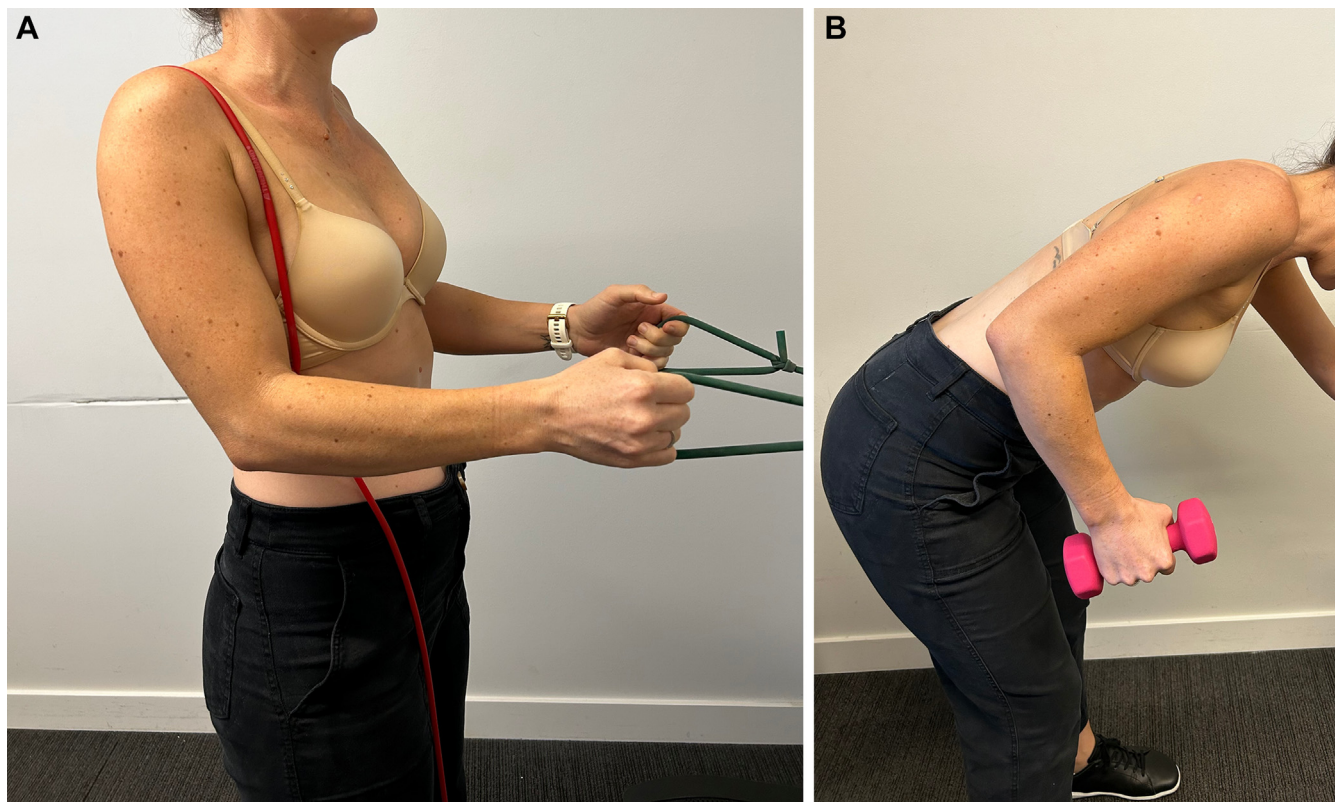


Figure 5 A. Standing extension row with Theraband in hands and scapular resistance band and B. Bent over row with a weight.

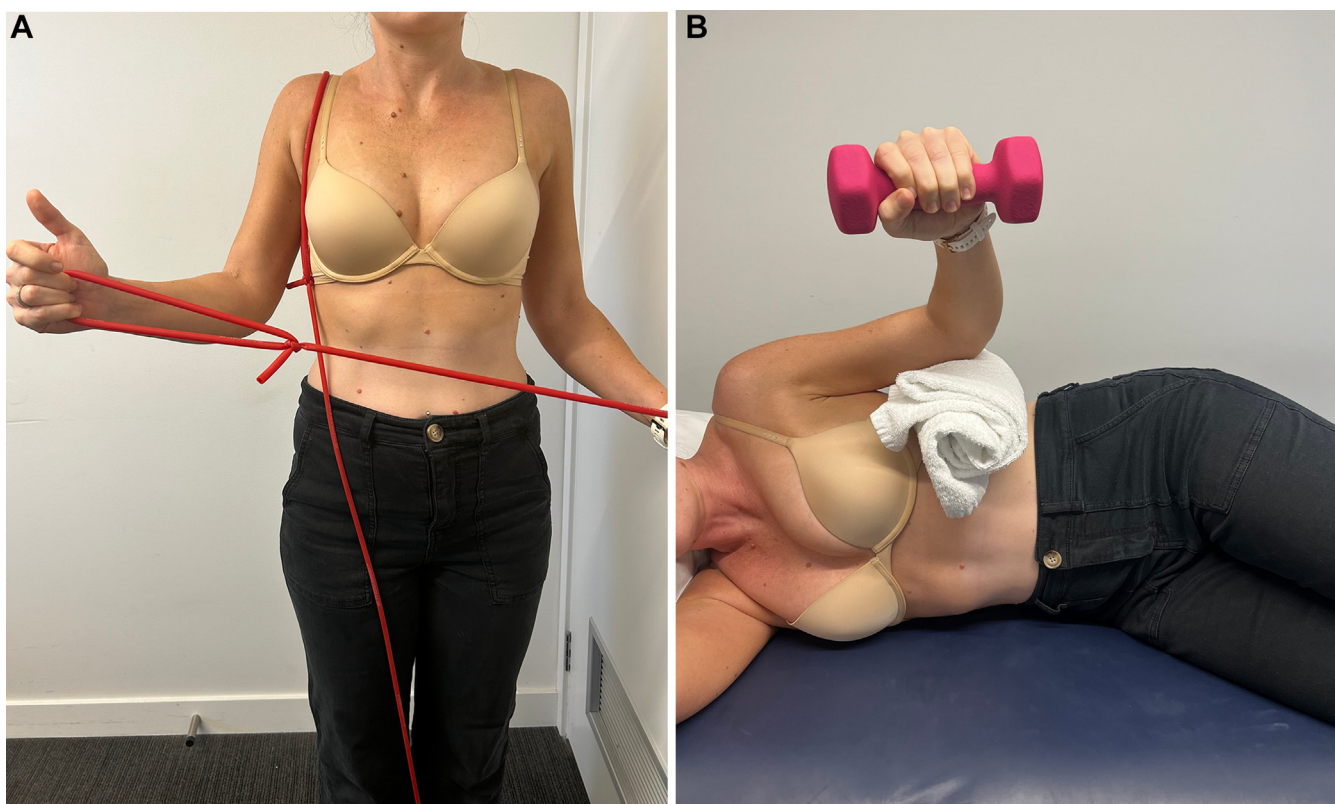


Figure 6 A. Standing external rotation with Theraband in the hand and scapular resistance band and B. Side lie external rotation with a weight.



Figure 7 Standing extension row at 45 degrees of abduction for scapular upward rotation control in higher ranges.



Figure 8 Standing extension row at 90 degrees of abduction for scapular upward rotation control in higher ranges.

magnitude of the SMD scores were interpreted using Cohen's guidelines, where a result of ≤ 0.20 – 0.49 is considered a small effect, 0.50 – 0.79 considered moderate, and a result of ≥ 0.80 considered a large effect.^{30,38} The frequency of success or failure of return to sport and rehabilitation overall is presented. Return to sport success and failure of conservative treatment was reported. Significance was set at 0.05.

Results

In this single-group study design, 24 shoulders in 22 amateur sporting participants (17 male, 5 females; mean age, 21.1 years, standard deviation 10.1 years) diagnosed with microtraumatic PSI undertook a 24-week exercise program specific for PSI. All participants had symptomatic posterior subluxation of the affected glenohumeral joint. Sporting categories of participants included 3 tennis players, 2 swimmers, 4 Australian football players, 2 cricketers, 3 netballers, and 8 weight lifters. Participants had their symptoms for a mean of 179.2 months (3.4 years) ranging from 4 to 520 months. Three of the 24 shoulders had a structural lesion, which included a small tear of the posterior band of the inferior glenohumeral ligament and two posterior labral tears.

After 24 weeks of the WIP-p (mean: 14.4 physiotherapy sessions, range 12–18) participants had significant improvements at 6 ($P = .036$, SMD: 0.74 = moderate effect), 12 ($P = .007$, SMD: 0.41 = small effect), and 24 weeks ($P < .001$, SMD: 1.7 = large effect) on the MISS (Table 1) and significant improvements at 12 ($P < .001$, SMD: 1.1 = large effect) and 24 weeks ($P < .001$, SMD: 1.8 = large effect) on the WOSI (Table 1).

For return to sport, 20 of the 22 (90.9%) participants returned to full activity at the 24-week time point. Two of the 22 (9.1%) participants were unable to return to their sport and were sent for surgical review. One of these participants was a tennis player and the other was a weight lifter; neither had an acquired structural lesion. Both the tennis player and the weightlifter, despite normalizing scapular mechanics and scapular and glenohumeral strength, had ongoing symptoms with their sporting activities; the tennis player with the follow through of their serve and preparation for their backhand volley, and the weightlifter with their overhead and bench press. In both cases, a specialist upper limb orthopedic surgeon and two senior shoulder physiotherapists agreed that conservative management had failed to return the patients to their respective high-level activities (competitive tennis and overhead weight lifting) due to deformation of the patient's capsulolabral structures, likely caused by their history of repetitive microtrauma. Due to a significant loss of passive integrity, these shoulders were unable to withstand the high force and repetitive nature of the patient's activities. The tennis player was offered a posterior capsular shift to restore the passive integrity of the glenohumeral joint^{23,24,31,56} though declined surgery as his shoulder function had improved enough for resolution of symptoms in activities of daily living. The weight lifter did not pursue surgical intervention and instead restricted their weightlifting activities.

Discussion

This single-group study revealed that amateur sporting patients with microtraumatic PSI had significant improvements on the MISS (6, 12, and 24 weeks) and the WOSI (12 and 24 weeks) after rehabilitation with the WIP-p. A large majority (91%) were able to return to their previous level of sport at 24 weeks. The magnitude of the treatment effect was large for the WOSI at 12 and 24 weeks and for the MISS at 24 weeks. The estimate of effect exceeded the MCID for the MISS at 6, 12, and 24 weeks and for the WOSI at 12 and 24 weeks, indicating not only statistical difference but a clinically meaningful improvement at these time points.¹⁴ Overall, these results demonstrate a high success rate of the WIP-p for return to sport and clinical outcomes measures for patients with microtraumatic PSI.

The findings of this study are consistent with the findings of a systemic review, reporting that patients with a nontraumatic history of PSI have improved pain and increased function after a shoulder rehabilitation program.²⁶ Blacknall et al⁴ reported a mean



Figure 9 A. External rotation at 90 degrees of abduction (coronal plane) with Theraband in the hand and B. Supported external rotation at 90 degrees of abduction (coronal plane) with a weight.



Figure 10 External rotation at 90 degrees of elevation (scapular plane) with Theraband in the hand.

change of 37.2 points on the WOSI at a mean of 172 days (approximately 24 weeks, range 1–160 days) in a group of 19 nontraumatic PSI patients after an exercise program. Similar to this study, the mean change between time points exceeded the MCID of the WOSI. When comparing the results of this study to other studies in MDI^{47,52} or atraumatic shoulder instability^{2,10} (not PSI specifically), the change scores of the MISS and the WOSI in the present study are smaller, but the effect sizes are similarly large at both the 12- and 24-week time points. This may again reflect the reduced sensitivity of clinical outcomes to measure change specifically in nontraumatic PSI populations.

The large proportion of patients returning to high-level sport after the WIP-p highlights the need to trial 6 months of rehabilitation before concluding failure of conservative treatment in most cases.^{4,10,47} Despite this success rate, a small proportion of participants (2 of 22) failed conservative management and required surgical referral. Interestingly, the presence of an acquired structural lesion did not determine the success of outcome; however, this finding must be interpreted with caution due to the range of sports included and the small sample size of this study. Repetitive overhead and/or throwing sporting participants were under-represented in this study. The demands required on a shoulder involved in sports, such as tennis or cricket bowling, (highly repetitive, rotation and unilateral forces) contrast with those imposed on the shoulder during netball or controlled overhead weight lifting.³⁴ Clinicians need to consider the patient's sporting participation when evaluating the success of conservative treatment. Future studies on subgroups of athletes in specific sports could be conducted to determine the effect of conservative management on a homogenous sample of populations with PSI.



Figure 11 Supported external rotation at 90 degrees of elevation (sagittal plane) with a weight.

Table 1
Results of patients-reported outcomes.

Outcome	Mean (SD)	Effect estimate (95% CI)	SMD (effect size)	P value
WOSI				
Baseline	47.2 (16.9)	-	-	-
6 weeks	53.9 (16.7)	4.3 (-3.8 to 12.4)	0.40	.294
12 weeks	64.7 (18.9)	13.9 (6.1-29.6)	1.1	<.001
24 weeks	67.8 (22.2)	21.7 (13.8-29.6)	1.8	<.001
MISS				
Baseline	59.3 (12.2)	-	-	-
6 weeks	68.1 (13.7)	6.2 (0.42-12.0)	0.7	.036
12 weeks	69.2 (12.4)	7.6 (2.2-13.0)	0.4	.007
24 weeks	71.6 (14.1)	15.4 (9.8-21.0)	1.7	<.001

Effect Estimate, mean change score from baseline; CI, confidence interval; SMD, standardized mean difference (effect size). MISS, Melbourne Instability Shoulder Score (0 points = worst possible shoulder, 100 points = best possible shoulder). WOSI, Western Ontario Shoulder Index (0 points = worst possible shoulder, 100 points = best possible shoulder); SD, standard deviation. Bold highlighted values reach statistical significance from baseline scores ($P < .05$). Effect size interpretation: ≤ 0.20 -0.49 = small effect, 0.50-0.79 = moderate effect, ≥ 0.80 = large effect.^{30,38}

The overall success of the WIP-p could be due to its focus on normalizing scapular function, focusing on posterior deltoid and rotator cuff function and eventually progressing participants into once vulnerable positions. Focusing on normalizing scapular and posterior deltoid and rotator cuff function adheres to recommendations for achieving optimal outcomes after conservative management in PSI.²⁶ The rotator cuff act to center the humeral head in the glenoid fossa and prevent uncontrolled translation during

various planes of shoulder motion.¹⁶ The posterior deltoid also acts as a mechanical buttress to prevent excessive posterior humeral head movement.²⁶ Adequate scapular function is imperative to maintain centering of the humeral head on the glenoid fossa,¹² and randomised controlled trials that have focused on normalizing scapular function prior to rotator cuff strengthening have yielded superior outcomes compared to a general shoulder strength program in multidirectional instability⁴⁷ and subacromial impingement syndrome.³⁷ Optimal scapular position also ensures optimal length-tension relationship, appropriate loading, and therefore function of the rotator cuff muscles.²⁷ Progressing exercises eventually into the flexion, internal rotation, and horizontal adduction planes, ensures that humeral head control is gained in positions that were once most vulnerable for the patient. Movement control is position-specific,^{45,39} so this progression is imperative for functional carry over into activities of daily living (eg, crossing arms over the body to take off a top, turning the steering wheel of a car) and sporting activities (eg, the follow through of a throw) for people with PSI.

Limitations of this study include a small sample of participants who compete in a variety of sports and no-control group. A control group would have increased confidence in the definitive effects of the intervention. However, given that patients had their symptoms on average for 3.4 years and displayed statistically and clinically significant improvements on the MISS and WOSI after the 6-month intervention, we can be confident that the treatment effects are not due to the passing of time. Glenoid retroversion and glenoid hyperplasia were not formally assessed in this study, which may be viewed as a limitation. However, the presence of congenital abnormalities alone does not define the subgroup of the participant; rather the mechanism of injury.³² While patients with atraumatic PSI typically have a higher rate of congenital abnormalities,³² they may also be present in some patients with microtraumatic PSI.⁵⁴ Future rehabilitation studies could correlate glenoid retroversion to clinical outcomes in microtraumatic PSI.

A strength of this study is that it is one of the first to investigate the effect of a rehabilitation program on microtraumatic PSI using instability- specific outcome measures.²⁶ To date, most studies investing the outcomes of rehabilitation programs in PSI fail to use PROMs^{3,40,59} or use PROMs that are not sensitive or specific to measuring changes in the instability population.^{7,18} A failure to use PROMs that are not valid, reliable, or responsive to measuring change in an instability population, means that important clinical change in response to an intervention may go undetected. This has implications for patients who are trialling rehabilitation with the prospect of surgical intervention if they are not improving. Another strength of this study is that it provides a level of efficacy for the WIP-p, and the WIP-p has been published with enough detail that can be replicated it in the clinical setting. Clinicians now have an evidenced-based resource to utilize with patients with PSI. Lastly, this study adhered to the subgrouping guidelines of a recent Delphi study³² and only included patients with microtraumatic PSI. In this study, all included patients had a history of microtrauma that was associated with symptom development rather than an insidious onset, which is characteristic of atraumatic PSI. While there may be overlap in the contributing factors of patients with atraumatic and microtraumatic PSI (ie, glenoid retroversion, scapular dyskinesia, and/or glenohumeral joint hypermobility), the primary distinction between these subgroups is the mechanism of symptom onset. Subgrouping instability patients by mechanism of injury is important for selection of the most appropriate treatment pathways.^{32,42,54} While the treatment may be similar for those with atraumatic and microtraumatic PSI groups in the majority of cases, surgery may be considered in the microtraumatic group who fail rehabilitation, owing to the possibility of acquired structural

lesions or acquired capsular deformation due to repetitive microtrauma.^{32,54}

Conclusions

The WIP-p resulted in a high level of return to sport and significantly improved functional outcomes in patients with microtraumatic PSI. A small proportion of sporting participations with microtraumatic PSI may fail conservative rehabilitation and require surgical consideration.

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