# Side-to-side range of movement variability in variants of the median and radial neurodynamic test sequences in asymptomatic people

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# Objective

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To provide a better understanding of the normal asymmetries between left and right elbow range of motion (ROM) during variants of the median and radial upper limb neurodynamic tests (ULNT) in an asymptomatic population.

# Methods

Within-subject comparisons of left and right elbow flexion ROM were conducted on 51 asymptomatic participants (26 females, 25 males; mean age 29.7, SD 5.9 years). Range of motion was measured using an elbow mounted electrogoniometer during median and radial ULNTs. The participants were positioned supine with the neck in contralateral lateral flexion and the scapular stabilised in neutral. The arm was then passively positioned in 90° shoulder abduction and external rotation and 90° elbow flexion. The median nerve biased position involved full forearm supination and full wrist/finger extension, while the radial biased position involved full pronation with wrist/finger flexion. The elbow was then passively extended to onset of resistance (R1) and onset of discomfort (P1) in separate tests.

### Results

There were no significant differences in mean ROM between sides, with the ROM of one side significantly correlated with the opposite side (median  $R^2 = 0.62$ , radial  $R^2 = 0.85$ ) for both P1 and R1. Lower bound scores accounting for measurement error and within-subject variability indicate that flexion ROM differences between sides of greater than 15° for the median ULNT and 11° for the radial ULNT could indicate asymmetry beyond normal variation in a similar age-matched population.

### Conclusion

The normal variability in ROM observed between sides highlights the need to combine ROM findings with those of symptom provocation and structural differentiation in determining the clinical significance of a median or radial neurodynamic test.

### Commentary

Upper limb neurodynamic tests have become a commonly used clinical tool in the assessment and diagnosis of peripheral neuropathic pain. In the absence of central pain mechanisms as the primary cause of symptoms, ULNTs are generally accepted as tests biased towards the mechanosensitivity of nerve roots, brachial plexus and peripheral nerve trunks (Nee et al 2012). A positive test is indicated by (i) reproduction of the patient's symptoms and (ii) structural differentiation via movement of more distal or proximal joints along the path of the nerve, that either aggravate or ease symptoms. A third plausible indicator is a reduction in ROM on the symptomatic side compared to the asymptomatic side (Butler 2000). While ROM asymmetries are commonly accepted as partial indicators in the normal asymmetries that

may exist between sides (Nee et al 2012). This is pertinent considering asymptomatic subjects can also report symptoms of aching, pain, burning and tingling in response to ULNTs (Nee et al 2012). Therefore, this recent study is worthy of review and may aid clinicians in their interpretation of ULNT ROM findings, helping to avoid false positives and prioritise management.

The clinical relevance of this study is strengthened by its use of therapistadministered variants of ULNTs, without mechanical stabilisers utilised in other similar studies (Van Hoff et al 2012). There are multiple variations for biasing the median, radial and ulnar nerves in ULNTs; however, all require skilled manual handling to ensure the intended neural structure is progressively loaded, thereby achieving an accurate test of the nerves' mechanosensitivity (Butler 2000). In the absence of precise movement and sound patient communication, false tests can easily occur.

If the findings of this study are to be used clinically as a cut-off for potentially normal asymmetry in ULNTs, then the pre-placement of the neck in contralateral lateral flexion and the use of R1 and P1 as end points are important aspects to consider. It is common clinical practice to elicit symptoms with the neck in neutral, then to utilise neck lateral flexion as a sensitising manoeuvre to aid in structural differentiation, especially when suspecting more distal pathology such as carpal tunnel syndrome or radial nerve entrapment (Butler 2000). Contralateral lateral flexion of the neck reduces the ROM available in an ULNT, effectively pre-loading the peripheral neural structures (Coppitiers et al 2001). A previous study of similar design found higher ROM variability when the neck remained in neutral and the end point was marked by 'firm resistance' - 27° and 20° of elbow flexion for the median and radial ULNTs, respectively (Covill and Petersen 2012), compared to 15° and 11°, respectively, in the present study. These findings are not representative of a symptomatic population, however they may suggest that pre-loading the neural structures and stopping at R1 or P1, rather than 'firm resistance' (Covill and Petersen 2012), provides a more accurate representation of ROM differences between sides.

An objective cut-off value indicating when differences in ROM between sides are likely to be a result of pathology would be an ideal clinical measure for interpreting the significance of an ULNT. In addition, this would provide a clear measure of outcome, guiding treatment and aiding in communication with other involved parties, such as employers and insurance providers. In this regard, the values reported in this study should be used cautiously. The use of an electrogoniometer and a relatively young population are the obvious limitations in the clinical value of these findings. In fact, the actual mean differences between sides were not found to be significant. However, consideration of measurement error and within-subject variability revealed potential differences of 15° and 11°. The clinical value of this study should instead be interpreted through the increased understanding that normal asymmetry can exist in upper limb neurodynamics. As a result, ROM findings need to be coupled with symptom provocation and structural differentiation for a ULNT to be interpreted as a positive sign of peripheral neuropathic dysfunction.

Nick Black (MPhtySt) Postgraduate student (MPhty OMT) School of Physiotherapy University of Otago

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# Do stingers affect scapular kinematics in rugby players?

Kawasaki T, Maki N, Shimizu K, Ota C, Urayama S, Moriya S, et al (2014) Do stingers affect scapular kinematics in rugby players? Journal of Shoulder and Elbow Surgery 23(12):e293– e299. doi 10.15619/NZJP/43.3.07 (Abstract prepared by Sharada Murty)

# Abstract

Aim: The rationale of this study was to investigate shoulder examination findings that are associated with scapular dyskinesis. This cross-sectional epidemiological study investigated the hypothesis that upper limb nerve traction injuries ('stingers') alter scapular kinematics in high-school rugby players.

# Methods

One hundred and sixty-five male Japanese high-school rugby players, without previous shoulder or elbow surgery or injury within the past month were recruited. Demographic data, including level of competition and injury history, were collected via a questionnaire. Three examiners (medical doctors) blinded to subjective data, completed physical examinations of both shoulders of each participant which included assessment of impingement, instability, shoulder girdle muscle strength, posterior capsular tightness and palpation of the acromioclavicular joint. Examiners evaluated scapular kinematics through video analysis and classified the type of scapular movement observed using the scapular dyskinesis test (4-type method).

### Results

Analysis of inter-rater reliability of assessment of scapular dyskinesis showed moderate reliability. Six subjects were excluded from further analysis as there was a lack of consensus on their classification. Of the 159 remaining players, 16 demonstrated scapular dyskinesis. Of these, 13 were classified as Type I dyskinesis and three exhibited Type III dyskinesis. Statistical analysis of the relationship between scapular dyskinesis and each of the examination findings demonstrated a significant association of Type I scapular dyskinesis with previous history of stingers.

### Conclusion

There is a significant association between Type I scapular dyskinesis and previous history of stingers in male high-school rugby players. The scapular dyskinesis test using the 4-type method is moderately reliable in measuring altered scapular motion.

#### Commentary

Abnormal scapular kinematics or scapular dyskinesis is associated with shoulder pathology including impingement, instability and acromioclavicular joint injury (Kibler et al 2013). Identification of factors that may influence scapular dyskinesis is important to establish appropriate injury prevention and rehabilitation protocols. However, evaluation of scapular dyskinesis necessitates a reliable method of assessment.

The authors of this paper have previously hypothesised that nerve traction injury to the shoulder results in scapular dyskinesis of the injured side (Kawasaki et al 2012). They suggest altered scapular dyskinesis may be due to neurological impairments and resultant disruption to neuromuscular coordination of scapular motion caused by traction to neural tissue. In this study, male high-school rugby players were assessed to identify findings related to the shoulder which may

be associated with altered scapular kinematics (which were observed, recorded by video and analysed by three examiners). Each examiner reported the presence or absence of scapular dyskinesis and categorised scapular movement patterns observed during shoulder flexion and abduction while the participant was holding a 3kg weight.

There are various recommendations for the assessment of scapular dyskinesis in the literature. Many of the assessment methods utilised clinically use a two-dimensional model to assess three-dimensional scapular motion. Despite this, there is consensus surrounding the use of the scapular dyskinesis test (referred to in this study as the 4-type method), which is reliable and easily adminstered clinically (Kibler et al 2013). Scapular movement is classified as: Type I, raising of the inferior angle of the scapula; Type II, raising of the length of the medial border; Type III, elevation of the superior border; Type IV, normal (Kibler et al 2013). Visual observation of scapular movement and determination of the presence or absence of dyskinesis is noted.

A multivariate analysis was used to evaluate the association between scapular dyskinesis and assessment findings. Only Type I scapular dyskinesis revealed a sample large enough to allow statistical analysis, with a significant association reported between Type I scapular dyskinesis and previous history of a stinger in high-school rugby players. This association has also been reported in adult rugby players (Kawasaki et al 2012, Vaccaro et al 2002). The authors of this study suggest that stingers cause scapular dyskinesis. As with other shoulder pathology there is strong evidence of the presence of altered scapular kinematics in players with a history of nerve traction injury but it is not evident whether this is a cause of pathology or the result of it (Kibler et al 2012). It is likely that many factors contribute.

Nerve traction injury affecting the upper limb is a common injury in collision sports, reported to occur in up to 50% of players (Vaccaro et al 2012). Symptoms are due to either traction of the brachial plexus or C5/6 nerve root causing shooting pain and/or paraesthesia down the arm to the hand (Kawasaki et al 2012). Weakness may or may not occur and symptoms usually last minutes or hours, and very rarely more than a day. Due to the usually transient nature of symptoms, players may not seek assessment or treatment before return to sport. The guidelines for return to play following a stinger injury include complete resolution of symptoms, return to baseline range of motion and strength (Vaccaro et al 2012). Despite following these guidelines, exacerbation of symptoms often occurs with relatively minor secondary trauma. The results of this study suggest that it is also important to assess scapular mechanics, and rehabilitation programmes should correct scapular dyskinesis before return to sport.

This study provides evidence for reliable methods of assessment of scapular dyskinesis. The authors report a clear association between nerve traction injury and altered scapular kinematics. Clinically, the findings of this study suggest assessment of scapular motion is important following stinger injury. Rehabilitation including strategies to restore normal mechanics before return to sport may minimise further injury.

Sharada Murty (BPhty, BSc, PGDip Sports Med) Balance Physiotherapy Ltd Christchurch

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