Differential Diagnosis and Physical Therapy Management of a Patient With Radial Wrist Pain of 6 Months’ Duration: A Case Report

When evaluating a patient with radial wrist pain, clinicians need to consider a number of possible etiologies, including systemic disease, referred pain to the radial aspect of the wrist, and local tissue dysfunction. A commonly considered local tissue dysfunction in patients with radial wrist pain is De Quervain syndrome or stenosing tenosynovitis of the first dorsal compartment. Although implying an inflammatory etiology, the histopathology of stenosing tenosynovitis is generally characterized by fibrosis and fibrocartilaginous metaplasia leading to thickening of the extensor retinaculum in the absence of inflammatory findings. The sliding impairment of the entrapped but normal abductor pollicis longus and extensor pollicis brevis tendons within the stenosed extensor compartment is thought to be responsible for the symptoms. Epidemiological studies in industrial settings have shown a point prevalence of 8% for De Quervain syndrome, when wrist pain and a positive Finkelstein test, with or without tenderness to palpation of the radial wrist, were used as diagnostic criteria. More relevant to nonindustrial settings, in 485 patients with upper extremity musculoskeletal disorders consisting mostly of computer users and musicians, 17% were diagnosed with De Quervain syndrome on the right and 5% on the left, based solely on a positive Finkelstein test.

Relevant to a patient with radial wrist pain is the differential diagnosis between stenosing tenosynovitis and an entrapment neuropathy of the superficial radial nerve. Moore described the superficial radial nerve as a critical neural structure in this area. After crossing the elbow, the radial nerve splits and forms the posterior interosseous nerve and the superficial sensory branch of the radial nerve, which supplies the sensation to the radial aspect of the forearm and wrist. This superficial sensory branch courses along the radial wrist and suggests neuropathy of the superficial radial nerve as a differential diagnostic option.

Case Description: The patient was a 57-year-old man with right radial wrist pain of 6 months’ duration. The referral diagnosis was De Quervain syndrome, but a previous course of electrophysical agents-based physical therapy management had been unsuccessful. The physical examination ruled out the cervical, shoulder, elbow, and wrist joints as possible sources of pain. In this case, the diagnosis of entrapment neuropathy of the superficial radial nerve, rather than De Quervain syndrome, was primarily based on the symptom provocation resulting from a modified radial bias upper limb nerve tension test. Based on this diagnosis, treatment consisted of active and passive exercises using neurodynamic techniques.

Outcomes: After 1 treatment session, the patient noted changes with regard to current pain intensity and function that exceeded the minimal clinically important difference and the minimal detectable change, respectively. After only 2 treatment sessions, the patient reported a complete resolution of symptoms and a full return to work.

Discussion: This case report critically evaluates the diagnostic process for patients with radial wrist pain and suggests neuropathy of the superficial sensory branch of the radial nerve as a differential diagnostic option.


Key Words: De Quervain syndrome, neuropathy, superficial sensory branch radial nerve, thumb

Study Design: Case report.

Background: Differential diagnosis for patients with radial wrist pain requires consideration of systemic disease, referred pain to the radial aspect of the wrist, and local tissue dysfunction. The list of possible local dysfunctions should include De Quervain syndrome, as well as entrapment neuropathy of the superficial radial nerve.

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distally into the forearm along the brachioradialis muscle and tendon. At the proximal third of the forearm, the nerve runs between the tendons of the extensor carpi radialis brevis and brachioradialis muscles, and during pronation these tendons may mechanically compromise the radial nerve. At the beginning of the lower third of the forearm, it traverses over the abductor pollicis longus and extensor pollicis brevis muscles and continues toward the hand between the first and third dorsal compartments, dividing into its terminal branches. One of these branches in particular, called the anterior terminal branch, passes almost directly over the first dorsal compartment and provides sensation to the dorsum of the thumb. In addition to possible compromise proximally in the forearm between the extensor carpi radialis brevis and brachioradialis tendons, tissue trauma at the wrist might affect local neural structures. Butler hypothesized that small nerve branches might become embedded in organizing scar tissue, leaving them ischemic and overly sensitive to chemical and mechanical stimulation.

Considering these 2 etiologies for radial wrist pain due to local tissue dysfunction and their different implications with regard to physical therapy management, the purpose of the current case report is to describe the physical therapy diagnosis and management of a patient with radial wrist pain of 6 months’ duration, previously unresponsive to conservative management, including medication, electrophysical agents, and splinting.

**CASE PRESENTATION**

In physical therapy there are 5 components to patient management: examination is followed by evaluation of the examination findings, establishing a diagnosis, producing a prognosis, and developing a plan of care. This process is followed by implementation of the interventions in the plan of care. This case presentation is modeled on this 5-component approach to patient management.

**History**

The patient was a 57-year-old male. He was 172 cm tall, weighed 98 kg, was right hand dominant, and employed as a bartender. Initial symptoms started insidiously 6 months prior to the initial physical therapy visit and consisted of pain and stiffness in the radial side of the right wrist. Three months after symptom onset, the patient first consulted with his general medical practitioner, who diagnosed De Quervain syndrome and started treatment with nonsteroidal anti-inflammatory medication. During a follow-up visit 1 week later, the general practitioner prescribed a short-arm thumb spica splint that immobilized the wrist in a neutral position and the thumb in abduction. The patient was instructed to stop working. An initial series of 6 weeks of physical therapy treatments was started approximately 1 month later, consisting of laser, ultrasound, transcutaneous electrical nerve stimulation, and diathermy. The patient reported that this management approach did not change his symptoms or improve his function.

The patient presented to the primary author’s clinic about 6 months after the initial onset of the symptoms. At the time of presentation, he was taking nonsteroidal anti-inflammatory medication and reported constant right radial wrist pain (Figure 2) during work activities. The intensity of his pain increased with repetitive movements at work and with carrying bottles from a storage to the bar area. Although the pain was generally burning in character, the patient also reported acute, electrical shock-like pain when trying to lift packs of multiple bottles and when attempting to turn the wrist to pour drinks. This acute pain caused the patient to have to stop working for several minutes and resulted in a constant burning pain lasting the rest of the day.

![Figure 1. Anatomy of the radial nerve (in red) in the forearm. At the beginning of the lower third of the forearm, the radial nerve traverses over the abductor pollicis longus and extensor pollicis brevis muscles, and continues toward the hand between the first and third dorsal compartments, dividing into its terminal branches. One of these branches, the anterior terminal branch, passes directly over the first dorsal compartment and provides sensation to the dorsum of the thumb.](image1)

![Figure 2. Patient reported distribution of pain (highlighted in red) over the lateral aspect of the wrist and dorsal aspect of the thumb.](image2)
Pain was reduced when not working and when using the thumb spica splint. Any activity without the spica splint resulted in an immediate increase in pain. The medical history of this patient included hypertension and a medical diagnosis of currently asymptomatic osteoarthritis in the neck and back.

Outcome Measures
Outcome measures included the 10-cm horizontal visual analogue scale (VAS) for current pain at rest and a 2-item patient-specific functional scale (PSFS), also using a VAS for scoring the individual items. The PSFS is a patient-specific outcome measure that investigates functional status by asking the patient to list activities that are difficult to perform, based on the patient’s condition, and to rate the level of limitation with each activity. The PSFS has been shown to identify changes in status and to be valid and responsive to change for patients with various clinical conditions but has not been studied in patients with wrist pain.

The patient rated his current resting pain at 5.4 on the VAS (0 representing no pain and 10 the worst pain imaginable). On the PSFS, where 0 indicates complete inability to perform a task and 10 represents the ability to perform the task at preinjury level, the patient rated his ability to lift to 2.9 and his ability to pour drinks at 3.1. The table provides data on these outcome measures, as reported at the beginning of each session, throughout and after the episode of care. The VAS is a reliable outcome measure for the measurement of pain. Kelly established its minimal clinically important difference as a change of 12 mm (95% confidence interval [CI]: 9, 15 mm). Westaway et al., though using a numeric rating scale in patients with neck and not wrist pain that yielded discrete rather than continuous scores, reported excellent test-retest reliability (ICC = 0.91) and a minimal detectable change (MDC) of 1.18 for individual items on the PSFS.

**Tests and Measures**

Visual observation of sitting posture revealed normal alignment without excessive forward head position or excessive shoulder protraction. Abnormalities of neck and shoulder posture would have justified increased focus on these areas as a possible cause or contributor for wrist pain due to referral from nonlocal dysfunction. Cleland et al. established less-than-chance intrarater agreement for visual assessment of forward head posture (κ = –0.1; 95% CI: –0.2, 0.0) but excellent agreement for the presence of shoulder protraction (κ = 0.83; 95% CI: 0.51, 1.0).

Examination of the cervical spine included active range of motion testing for flexion and bilateral sidebending. This was followed by overpressure in flexion and axial compression in sidebending (Spurling A test) in an effort to rule out myelopathic involvement and cervical radiculopathy, respectively. None of these tests reproduced patient symptoms. Cleland et al. reported poor to excellent intrarater agreement for symptom reproduction with cervical active range of motion into flexion (κ = 0.55; 95% CI: 0.23, 0.88), sidebending left (κ = 0.0; 95% CI: –0.22, 0.23), and sidebending right (κ = 0.81; 95% CI: 0.57, 1.0). Uchihara et al. reported sensitivity of only 3% for the use of the neck flexion test in patients with cervical myelopathy. Wainner et al. found substantial intrarater agreement (κ = 0.60; 95% CI: 0.32, 0.87) for the Spurling A test, but a sensitivity of only 50% (95% CI: 27%, 73%) for the diagnosis of cervical radiculopathy.

Examination of the right (symptomatic) shoulder included active range-of-motion testing, followed by overpressure for shoulder flexion, extension, abduction, and internal and external rotation at 90° of abduction. The intent of this portion of the examination was to rule out referred wrist pain from shoulder structures innervated by the C6 segment. Passive depression of the shoulder girdle was performed to assess for neural mechanosensitivity in the neck-shoulder region. Varus and valgus stress tests with the elbow extended/supinated were performed to rule out elbow involvement and, more specifically, to rule out an ulnohumeral lateral restriction causing radial nerve involvement at the elbow.

Active motions assessed at the right wrist followed by passive overpressure included flexion, extension, and radial and ulnar deviation. Accessory motion testing was done to assess mobility and pain reproduction for all carpal bones. None of these tests reproduced the patient’s symptoms. A literature search did not retrieve data on psychometric properties for these tests.

The patient had a positive right Finkelstein test. Symptoms of burning radial wrist pain were reproduced by passive thumb flexion, combined with ulnar deviation at the wrist. Butler described a variation of the upper limb nerve tension

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**TABLE**

<table>
<thead>
<tr>
<th>Scores on Visual Analogue Scale for Current Pain and Patient-Specific Functional Scale Items</th>
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<tbody>
<tr>
<td><strong>Current Pain</strong></td>
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<tr>
<td>Session 1 (day 1)</td>
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<tr>
<td>Session 2 (day 4)</td>
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<tr>
<td>Session 3 (day 7)</td>
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<tr>
<td>1 mo posttreatment</td>
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<td>3 mo posttreatment</td>
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<td>6 mo posttreatment</td>
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*Current pain was based on a 10-cm horizontal visual analogue scale, where 0 indicated no pain and 10 indicated maximum pain.*  
*Assessment of function was based on a 10-cm horizontal visual analogue scale, where 0 indicated unable to do the skill and 10 indicated ability to do the skill similar to prior to the injury.*
test with radial bias (ULNT2b) (FIGURE 3). To test the superficial sensory branch of the radial nerve, the patient was placed in a Finkelstein test position, with a clenched fist around his flexed thumb and the wrist in ulnar deviation. This resulted in an increase of the radial wrist pain, and the pain was described as burning and resembling an electrical shock. The proximal sensitizing movements of shoulder depression and contralateral cervical sidebending further increased symptoms. The same test sequence for the left upper extremity was negative. Wainner et al reported excellent interrater agreement for the ULNT2b test ($\kappa = 0.83; 95\% CI: 0.65, 1.00$); but it should be noted that these authors used a proximal-to-distal, rather than the distal-to-proximal sequence of movements that we used for this patient, and did not include wrist ulnar deviation and thumb flexion as a component of the test.

**Evaluation and Diagnosis**

The clinical examination outlined above seemed to exclude a systemic but also a musculoskeletal etiology, with pain referral from the neck, shoulder, and elbow. Data on reliability and validity of the tests used are mostly nonexistent. The psychometric data available on the cervical spine screening tests indicated limited sensitivity. We therefore realize that ruling out these nonlocal musculoskeletal causes is based mainly on a pathophysiologic rationale. Negative findings on active range-of-motion, overpressure, and accessory testing for the wrist similarly seemed to rule out a causative or contributory joint dysfunction.

The examination for this patient indicated that the referral diagnosis of De Quervain syndrome was likely not accurate, and that a local neuropathy of the superficial sensory branch of the radial nerve was a more plausible diagnosis. Butler reported that the Finkelstein test should be considered a test for both De Quervain syndrome and an entrapment of the superficial sensory branch of the radial nerve. He suggested that structural differentiation be performed by investigating the effects of some sensitizing movements proximal to the wrist, such as elbow extension, shoulder abduction, and shoulder girdle depression. Decrease in symptoms associated with shoulder girdle elevation might also implicate the proposed neuropathic etiology. Therefore, in this patient, the increase in symptoms secondary to shoulder depression and cervical contralateral sidebending led us to a diagnosis of neuropathy of the superficial sensory branch of the radial nerve as opposed to a De Quervain syndrome, in which it was unlikely that these same maneuvers would have changed the nature and intensity of the symptoms.

**Prognosis and Plan of Care**

We were unable to locate information relative to the prognosis and natural history of this pathology. The plan of care included neurodynamic interventions, to be performed both in the clinic by the physical therapist as passive manual therapy techniques and at home by the patient as active exercises.

**OUTCOMES**

On the first visit following the examination the therapist educated the patient on the findings of the clinical examination and derived diagnosis and proposed the overall plan of care described above. Intervention at this first visit consisted of passive neural slider techniques for the radial nerve, specifically targeted to the superficial sensory nerve branch by adding ulnar deviation of the wrist and flexion of the thumb during the ULNT2b-derived technique (FIGURE 4). The slider intervention was completed over 15 to 20 minutes, in 4 sets of 5 minutes each, with a 1-minute rest between sets. Speed and amplitude of movement were adjusted such that no pain was produced. A nerve slider intervention is a neurodynamic maneuver intended to produce a sliding movement of neural structures in relation to their adjacent tissues. Nerve slider exercises involve application of movement/stress to the targeted nerve structure proximally, while releasing movement/stress distally, followed by the reverse combination.

Further, mainly because such exercises are less complicated to perform than the slider mobilization performed by the therapist during treatment sessions, the patient was also provided with a tensioner home exercise for self-mobilization of
the nerve (FIGURE 5). A tensioner exercise is a neurodynamic maneuver that produces an increase in tension (not stretch) in the targeted neural structures.6 This intervention is distinctly different from a slider exercise in that, with this technique, the movement/stress is simultaneously applied both proximally and distally to the targeted nervous structure and released.6 For the home exercise, the wrist was placed on the table, with the elbow in extension and pronation and the cervical spine rotated towards the side of pain (A). From this position, the patient turned the cervical-thoracic spine towards the opposite side increasing the pronation of the forearm, inducing tension of the radial nerve (B).


discussion

This case report describes the physical therapy diagnosis and management of a patient with radial wrist pain 6 months in duration. Examination findings—notably, a reproduction of symptoms with a variant of the ULNT2b and an increase in symptoms with sensitizing movements proximal to the wrist—and the ineffective course of previous physical therapy treatment put in doubt the referral diagnosis of De Quervain syndrome. Rather, the primary author entertained the working hypothesis of a local neuropathy of the superficial sensory branch of the radial nerve and started management with neurodynamic techniques. After the initial visit (at the beginning of visit 2), the patient noted improvement in current pain intensity exceeding the minimal clinically important difference of 12 mm and improvement on each of the 2 items of the PSFS exceeding the MDC90 of 1.18. After 2 visits, at the beginning of visit 3, there was a reported complete resolution of symptoms and full return to work. The rapid response to the single-intervention method provided to the patient, within the context of uncertainty of the cause-and-effect relationship that must be considered for any case report, provides support for the hypothesis of a local neuropathy of the superficial sensory branch of the radial nerve.

We acknowledge that the rapid recovery of this patient may be inconsistent with nerve compression. An alternative mechanical explanation is that an adhesion developed somewhere along the sensory branch of the radial nerve. Neurodynamic exercises may have elongated such an adhesion or caused it to release from adjacent structures, thereby explaining the rapid improvement observed.

Based on a narrative review of the literature, Moore69 reported that patients with stenosing tenosynovitis usually report gradual, nontraumatic onset of frequently severe pain about the radial styloid that may refer distally to the thumb and proximally to the forearm and sometimes to the shoulder. Pain is generally constant but increases with grasping, abduction of the thumb, and ulnar deviation of the wrist, and may disturb sleep or even be sufficiently intense to render the hand useless. Some patients also may report stiffness or neuralgia-like complaints. Physical examination findings may include slight swelling about the radial styloid and normal but sometimes painful range of motion of the wrist and thumb. Passive stretching and contraction of the involved muscles and tendons generally
will increase pain. The Finkelstein test is considered pathognomonic.\textsuperscript{19} Electrodiagnostic studies, laboratory findings, and imaging tests are usually normal, but injection of a local anesthetic into the first dorsal compartment has been suggested as helpful in diagnosis.\textsuperscript{19}

Using a Delphi-based design, Palmer et al\textsuperscript{22} established consensus-based criteria for the diagnosis of upper limb disorders. De Quervain syndrome was defined as local pain over the radial styloid and tender swelling of the first extensor compartment, with pain symptoms reproduced by resisted thumb extension or a positive Finkelstein test. Walker-Bone et al\textsuperscript{33} reported good intrarater reliability ($\kappa = 0.66$) for diagnosis of De Quervain syndrome using these criteria in a primary care setting. However, these criteria have been criticized as circular reasoning, due to the clinically observed lack of specificity of these tests suggested as pathognomonic, and additionally for the absence of a gold standard to validate these tests.\textsuperscript{1}

Physical therapy management of De Quervain syndrome may entail patient education, splinting, ice, heat, transcutaneous electrical nerve stimulation, ultrasound, iontophoresis, friction massage, exercise, and joint mobilization. Solely based on a pathophysiologic rationale, research into the efficacy of these proposed interventions is yet lacking.\textsuperscript{14,21,30} Medical management includes glucocorticoid and/or anesthetic injection in the first dorsal compartment. If conservative management is unsuccessful, surgical interventions may be considered.\textsuperscript{1} Although misdiagnosis of radial wrist pain as De Quervain syndrome would in the worst-case scenario result in inappropriate and ineffective physical therapy treatment, the potential for medical-surgical iatrogenesis is far greater, emphasizing the need for correct diagnosis of patients with radial wrist pain.

With clinical presentations of De Quervain syndrome and local neuropathy of the superficial sensory branch of the radial nerve being very similar, the findings described on the variant of the radial bias ULNT test were, in our opinion, most relevant in the differential diagnostic process for this patient. Butler\textsuperscript{4} proposed 4 criteria to be, and in this scenario were, met to consider a neurodynamic test positive: (1) the test reproduces the patient’s symptoms, (2) structural differentiation supports a neurogenic source of these symptoms, (3) differences are present left to right and, if known, to normal responses, and (4) support exists from other data, including history, area of symptoms, and imaging tests. A case report on a patient with radial wrist pain, who did not respond to passive physical therapy but improved with joint mobilizations, has been previously published.\textsuperscript{32} Its main difference from the current case report is that in this earlier case report the radial bias ULNT test was considered negative because wrist symptoms did not increase by adding the sensitizing maneuvers of shoulder abduction or contralateral cervical sidebending. Also, in contrast to this earlier case report, accessory motion testing of the wrist was normal for the patient described in the current report. Our statement that these 4 criteria were indeed present can of course be challenged. Although the patient report of constant burning and intermittent shock-like pain has been suggested as indicative of the dyesthetic pain associated with peripheral nerve injury,\textsuperscript{20} the literature does not report identified common pathognomonic pain descriptors in peripheral or central neuropathic pain, thereby discounting this reported character of pain as supportive evidence.\textsuperscript{14} The International Association for the Study of Pain has defined neuropathic pain as pain initiated or caused by a primary lesion or dysfunction in the peripheral and/or central nervous system.\textsuperscript{18} Painful neuropathies are characterized by spontaneous and/or abnormal stimulus-evoked pain related to the presence of allodynia, whereby pain is caused by normally innocuous stimuli and/or hyperalgesia, in which case pain intensity evoked by normally painful stimuli is increased.

We did not specifically test for allodynia or hyperalgesia. Neuropathic pain states are also often associated with nonpainful spontaneous and evoked sensory phenomena that may include paresthesia, dysesthesia, and hypesthesia in the affected area.\textsuperscript{16} We did not perform an extensive sensory examination specifically because of the sensory loss often noted as a key finding in neuropathic pain.\textsuperscript{14} With severity of injury dictating the presence or absence of deficits in impulse conduction, sensory deficits need not occur in neuropathy.\textsuperscript{20} Even if sensory deficits are present, routine clinical nerve conduction tests and even electromyography are often insensitive to minor or disseminated nerve damage.\textsuperscript{21} However, in artificially induced compression studies,\textsuperscript{27,28} sensory abnormalities are the first indication of mild nerve compression. Therefore, sensory evaluation by way of quantitative sensory testing would have been a valuable addition to the examination in the current case report to confirm the neuropathy differential diagnosis, and we recommend its inclusion into daily practice in future patients with a similar presentation. In our opinion, the key clinical supportive finding satisfying the fourth criterion was the rapid resolution of the patient’s complaints despite a 6-month duration and previously unresponsive condition coinciding with the start of what seemed to be an appropriate management strategy based on the working hypothesis of a diagnosis of local neuropathy of the superficial sensory branch of the radial nerve.

We acknowledge multiple limitations to this case report. First, a case report does not allow us to infer a cause-and-effect relationship. Second, it should be noted that the above examination did not exclude neuropathy involving the lateral antebrachial cutaneous nerve, which may innervate the area where the patient indicated pain,\textsuperscript{8} although treatment results described below seemed to support the assumption that probably solely the radial nerve branch was involved in this patient. Further, prior physical therapy interventions received by this patient were pas-
vative in nature. We do not know if other passive or active approaches different from neurodynamic interventions would have equally improved patient symptoms. Fourth, many of the tests employed in the examination of this patient lack data on reliability and validity, leading to a clinical-reasoning process mainly based on a pathophysiologic rationale. This applies to the tests suggested for De Quervain syndrome, and also to the radial bias ULNT test variant deemed relevant to our clinical diagnosis of neuropathy. Finally, the sensitivity for many of the screening tests is too low to allow us to confidently rule out the condition for which the tests were intended to screen. Specifically, this did not allow for a confident ruling out of a possible multiple-crush syndrome and even the rapid resolution with the management program of neural mobilization techniques instituted does not allow us to infer that such multiple-site pathology was not relevant, because a multiple-crush injury could also be hypothesized to be positively affected with this intervention. Future research should concentrate on establishing psychometric data for the tests in the diagnostic process described in this case report and should also attempt to establish clear diagnostic gold standard tests for the various conditions that can cause radial wrist pain.

CONCLUSION

The current case report describes the diagnosis and management of a patient with chronic and previously therapy-resistant radial wrist pain initially diagnosed with De Quervain syndrome. Clinical examination led to the working hypothesis of a diagnosis of neuropathy of the superficial sensory branch of the radial nerve. Physical therapy management consisted of neurodynamic techniques, and the patient experienced a complete resolution of symptoms and full return to work after only 2 treatment sessions over 7 days. Although no cause-and-effect relationship can be established, this case report seems to indicate that, in patients with radial wrist pain, neuropathy of the superficial sensory branch of the radial nerve should be considered during differential diagnosis. Future research investigating the psychometric properties for tests used in the clinical diagnosis of radial wrist pain should be investigated. Additionally, a clear diagnostic gold standard for the various conditions that can cause radial wrist pain is needed.

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