



Shoulder Pain — Where Are We Now?

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Abstract

Purpose of Review Shoulder pain is common and costly. For the past century, diagnosis and management has been based upon presumed patho-anatomical abnormalities. With the evolution of imaging techniques and new insight about the causes of musculoskeletal pain, this review evaluates the evidence that a patho-anatomical approach remains justified.

Recent Findings Imaging modalities have developed considerably but, so far, have only proven value in evaluating full thickness rotator cuff tears prior to surgery. Correlation between imaging findings and symptoms is otherwise poor, with limited evidence of the value and impact of imaging for decision-making. Much of shoulder pain is chronic and few people have single-site musculoskeletal pain. Pain studies suggest that chronic shoulder pain is associated with both central and peripheral pain sensitisation. Moreover, functional MRI points to an effect of cognitive affective pain processing rather than nociception. Few of the established therapies, medical or surgical, that treat the presumed patho-anatomical cause have been shown to have lasting benefit.

Summary Much of the evidence suggests that shoulder pain is more similar than different from mechanical low back pain. For most people with shoulder pain, the best approach might well be de-medicalisation, support to (self)manage pain, emphasis on retaining movement and identifying adverse beliefs and risk factors for disability and chronicity. Approaches like this are currently being evaluated and more research is desperately required.

Introduction

Shoulder pain is common, with an estimated annual incidence of 0.9 to 2.5% [1] and point prevalence of 18–26% [2–5]. It is reported to be the third most common musculoskeletal symptom presenting for health care, making up an estimated 4% of annual consultations by adults in UK primary care [6,7]. Unfortunately, shoulder pain is also difficult to diagnose and to treat effectively [8, 9•]. Although several interventions appear to offer short-term relief, up to 50% of people remain symptomatic 18 months after presentation [10] and the course and prognosis may follow a relapsing-remitting pattern, making its assessment at a single time point potentially misleading. Symptoms can be disabling, affecting sleep and performance of daily activities both at home and in the workplace [11,12]. Shoulder pain results in a substantial economic burden including healthcare costs [13], and impacts on work participation

including reduced productivity, increased sickness absence, and higher risks of early retirement or permanent work disability [14–17]. The relationship with work disability is set to become increasingly important as governments take measures to encourage people to work to older ages because shoulder pain is strongly correlated with age (prevalence rate amongst adults aged > 70 years has been estimated as 21% [18]). This review will explore the known risk factors for shoulder pain and its differential diagnosis, consider the evidence surrounding the effectiveness of separation of shoulder pathologies by patho-anatomical diagnosis, and review the evidence underpinning current therapeutic modalities. Ultimately, we question whether a new approach is needed to the diagnosis and management of shoulder pain.

Risk factors for shoulder pain

Risk factors for shoulder pain have mostly been identified from epidemiological studies in which self-reported “shoulder pain” is investigated in connection with a range of socio-demographic, clinical, occupational, and other factors (Table 1). A range of physical occupational exposures (lifting, pushing/pulling, repetitive movements, vibration, long periods of static posture (desk time)) have been implicated, but the most compelling evidence points to the highest risk being associated with the cumulative effects of more than one of these exposures [11,19,20,21] particularly amongst female workers [19]. The combination of exposures does not have to be simultaneous, and it appears that it is the total number of different exposures involved that is important. Almost all studies that include psychosocial work factors find at least one to be associated [22] but what is evaluated, and how, varies considerably, including for example perceived stress; demands at work; autonomy; availability of support at work (from peers and/or supervisors); job satisfaction; stimulating work; and career prospects. Shoulder pain may, in turn, influence psychosocial work factors such as job satisfaction and the view of the individual about their level of support at work.

Differential diagnosis of shoulder pain

Shoulder pain does not always arise directly from the shoulder joint complex and can also be referred, or be the presenting feature of systemic disease (Figure 1) (reprinted with permission from Rees J and Carr A). Despite the plethora of differential diagnoses however, the vast majority of shoulder pain is regional pain which has hitherto widely been attributed to localised pathology

Table 1. Risk factors for shoulder pain

Type of risk factor	Specific factor	Reference(s)
Socio-demographic	Older age	Sansone et al. [73]
	Female gender	Miranda et al. [20]
Personal	Obesity	Luime et al. [74]
	Cigarette smoking	Ryall et al. [75]
Clinical	Diabetes mellitus	Shah et al. [76]
	Stroke	Anwer et al. [77]
Psychological	Emotional distress	Nahit et al. [78], Bovenzi et al. [79]
	Somatisation	Sarquis et al. [80]
Occupational	Heavy physical load (heavy lifting, pushing, pulling, carrying, holding)	Beach et al. [81], Andersen et al. [82], Bernard [20]
	Working with arms raised above shoulder height	Harkness et al. [83]
	Repetitive work	Leclerc et al. [84], Descatha et al. [85]
	Exposure to vibration	Bernard [20]
	Working in awkward postures (trunk flexed, twisted postures)	Miranda et al. [19]
	Psychosocial work environment	Van der Windt et al. [22]

affecting the peri-articular soft tissues (ligaments, muscles, capsule, tendons, labrum, bursae). A detailed discussion of the referred pain syndromes, systemic diseases, and articular pathologies is outside the scope of this review, which will focus instead on the so-called soft tissue conditions as the cause of the majority of shoulder pain in the population.

For the last 100 years, clinicians and researchers have attempted to better understand the causes and burden of localised shoulder pain using patho-anatomical classification systems which separated different types of shoulder pathology [23–25]. Patho-anatomical entities (otherwise known as “specific shoulder conditions”) which were described include the following: impingement; rotator cuff tears; adhesive capsulitis; bicipital tendinopathy; bursitis; calcific tendinopathy; instability; and labral tears. In clinical medicine, separation of different patho-anatomical diseases is important when it defines conditions with a different cause, prognosis, or response to treatment (e.g. “pulmonary tuberculosis” versus “chest infection”). As this review considers the development of imaging for shoulder pain, our developing understanding of the importance of multi-site musculoskeletal pain, and increasing knowledge of the brain changes which accompany acute and chronic pain, the reader is encouraged to consider to what extent the patho-anatomical separation of shoulder pain stands up.

Imaging and shoulder pain

As imaging techniques have developed, they have been employed to a growing extent amongst people with shoulder pain. In particular, X-ray, magnetic

Diagnosis of Shoulder problems in Primary Care:

Guidelines on treatment and referral

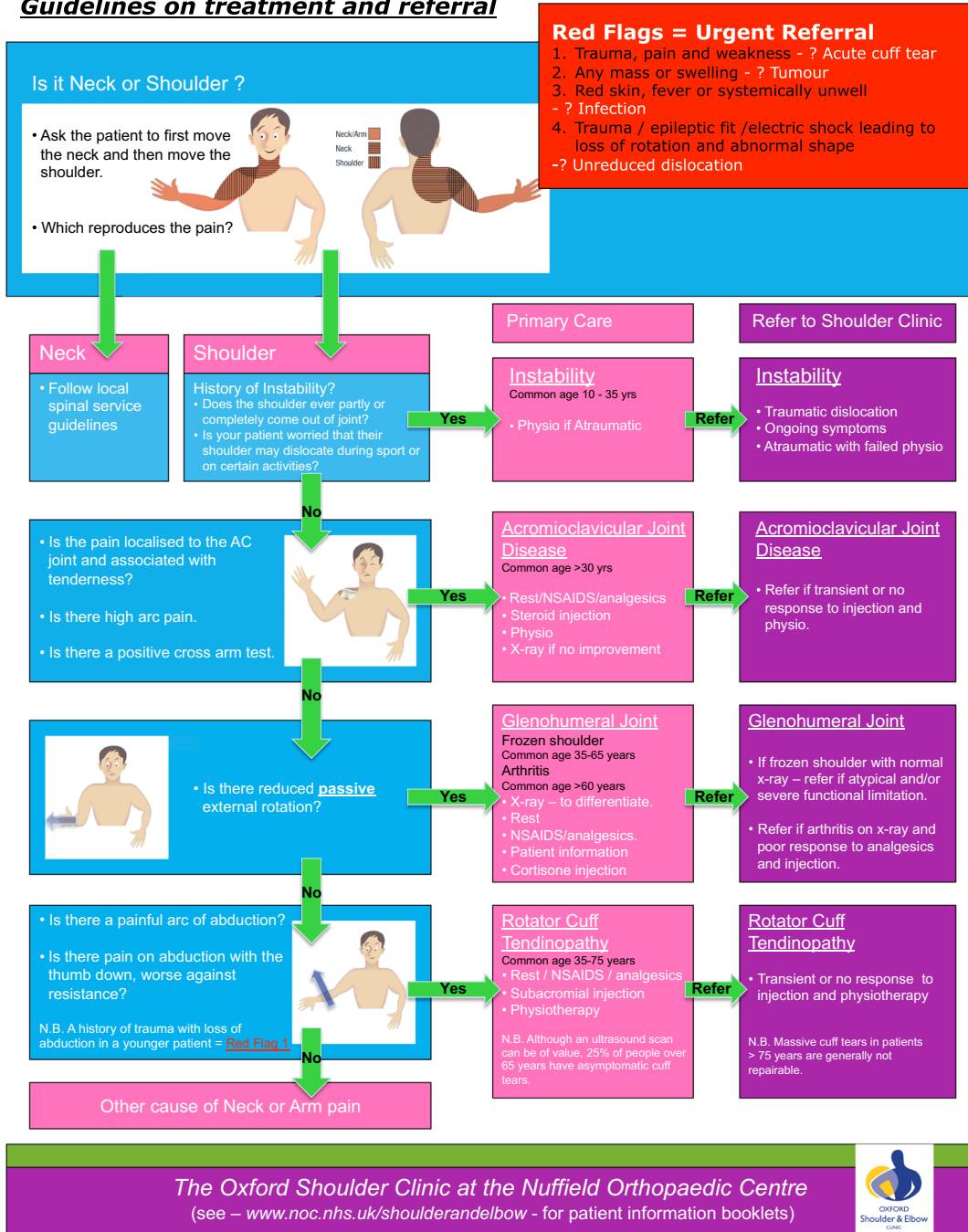


Figure 1. Schematic for the diagnosis of shoulder problems in primary care. (Reproduced with kind permission of Rees JL, and Carr AJ).

resonance imaging (MRI) scanning, magnetic resonance arthrography (MRA), and ultrasound scanning are commonly used modalities whilst bone scintigraphy, PET scanning, and CT arthrography are less commonly used. MRI, MRA,

and ultrasound have been shown to accurately detect some patho-anatomical abnormalities such as rotator cuff (RC) tears, tendinopathies, and subacromial bursitis [26,27], and to do so more accurately than clinical examination. In the planning of surgery for rotator cuff tears, a Cochrane review found evidence suggesting that any of MRI, MRA, or ultrasound could equally be used for detection of full thickness tears amongst people with shoulder pain for whom surgery is being considered [28]. However, the authors found that neither MRI nor ultrasound had good sensitivity for detecting partial thickness tears, with ultrasound sensitivity possibly considerably poorer than that of MRI. The authors were clear however that their findings related only to pre-operative imaging for acute or severe shoulder pain. Importantly, a US study which investigated patients with rotator cuff tears undergoing operative and non-operative treatment found that pain and functional status were not associated with any MRI features including tear size and thickness; fatty infiltration; and muscle atrophy [29]. It is important to realise that rotator cuff tears are also common in those without shoulder pain (prevalence 4 to 51% increasing with age) [30,31], and do not always correlate with symptom severity [32–34].

For the majority of shoulder pain for which surgery is not considered, the role of imaging is far from well-established and surveys of practice in Australia [8] and the UK [9] showed marked heterogeneity of what primary care doctors respond they would request in response to clinical vignettes and a heavy reliance upon imaging “in support of their clinical diagnosis”. It is interesting to note that use of ultrasound for shoulder pain has increased 10-fold in Australia even without any clear indication of its role [8]. In one recent trial in which patients were randomised to have ultrasound-tailored treatment of their shoulder pain versus usual care, no statistically significant difference was seen between rates of recovery in either arm [35]. Indeed, a systematic review of the relationship between shoulder symptoms and all imaging modalities concluded that, although this was a conflicting literature, there was no significant association between most imaging features and symptoms in the high-quality, cross-sectional studies [36••]. They did however find low-quality evidence that enhancement of the joint capsule on MRI and increased uptake on PET were associated with symptoms in adhesive capsulitis. Additionally, they found convincing evidence from high-quality longitudinal studies that enlarging rotator cuff tears were associated with an increased incidence of symptoms [36••].

In a separate study, Tran and colleagues analysed the abnormalities found on ultrasound scans from 3000 people referred because of shoulder pain from primary care [37•]. Instead of classifying on the basis of any pre-existing systems of classification, they used statistical latent class analysis to develop clusters from the ultrasound findings. The analysis yielded four distinct clusters, which had only some resemblance to traditional patho-anatomical ones: (a) bursitis with minimal inflammation elsewhere; (b) bursitis with extensive inflammation elsewhere; (c) rotator cuff tears; and (d) limited pathology [37•]. Amongst the 777 of these 3000 individuals who completed questionnaires, they found that people in groups (a) and (b) were most likely to have received steroid injections, those in group (c) most likely to have been treated surgically and that those in group (d) who underwent surgery reported poor outcomes. Notably, the shoulder pain and disability index scores were lowest in group (d) and highest in group (c).

Overall therefore, imaging has facilitated comparison of shoulder symptoms with presumed patho-anatomical abnormalities but imaging-detected structural changes increase with age and are common in asymptomatic subjects [38]. Therefore, incidental pathology identified on scans may lead to over-diagnosis, increase concerns for patients, and potentially lead to unnecessary referrals for surgical opinion. Moreover, not all imaging modalities available allow visualisation of all of the shoulder complex. In particular, the most widely used imaging modalities: x-ray and ultrasound offer rather limited visualisation. X-rays offer excellent views of the bones and joints, and the position of the humeral head and can help to exclude calcific tendonitis but fail to image normal soft tissues. Ultrasound can effectively identify full-thickness rotator cuff tears and be useful in planning surgical intervention and can identify inflammation but otherwise there is currently limited evidence for their utility in diagnosis and management of shoulder pain. Altogether currently therefore, imaging has not done much to build confidence in the presumption that our pre-defined patho-anatomical abnormalities represent separate diseases with different causes, responses to treatment, or prognoses.

Multisite musculoskeletal pain

Over a decade or more now, there has been growing recognition that musculoskeletal pain rarely occurs only at a single anatomical site. One study, for example, showed that only 16.8% of responders reported single-site pain whilst 53% reported pain at >1 site [39]. In fact, a number of population-based studies have found that more than half of their population reported multi-site pain [40–42]. Even amongst people at work in 24 countries across 5 continents, multi-site pain was considerably more common than single-site pain [43]. It is noteworthy that most of the early epidemiological literature which described the risk factors for incidence and prevalence of shoulder pain, focused on people defined by saying “yes” to pain in one or both shoulders, and did not include any consideration of pain at other anatomical sites. However, it is becoming clear that presence of pain at other anatomical sites is of relevance to the risk of development of musculoskeletal pain [44••], the risk of disability from the pain [39,45•,46], the response of the pain to treatment and its prognosis [45•,47•]. Several cohort studies of people presenting with shoulder pain in primary care have also reported poorer outcomes of pain and function, and lower recovery rates after 3–12 months in those with musculoskeletal pain at other sites [48–51]. Therefore, extrapolating from this evidence, the relatively poor prognosis reported for shoulder pain might usefully be at least partly explained by the presence/absence of pain at other musculoskeletal sites. More research is required which takes multisite pain into account in exploring response to treatment and/or prognosis.

Shoulder pain and the brain

Some further insight can be gleaned from investigations of pain sensitivity amongst people with shoulder pain. In a group of adults aged 18–85 years awaiting arthroscopic shoulder surgery with unilateral shoulder pain who did not report pain > 3 months at any other anatomical site, and a group of healthy

controls without shoulder pain, features of both peripheral and central pain sensitisation in relation to painful stimuli including pressure pain and thermal sensitivity were demonstrated [52]. Interestingly, some shoulder pain subjects demonstrated peripheral and central pain sensitisation whilst in others, the pattern was more central or more peripheral and the authors could not “explain” these variable patterns with any data that they had collected. Their findings were consistent with those seen in other unilateral musculoskeletal conditions, and the authors suggested that the standard treatment approaches for shoulder pain that focus on the peripheral stimulus (steroid injections, physiotherapy) would have limited effectiveness for the management of people with central sensitisation patterns.

Additionally, a recent study amongst patients aged 45–65 years with unilateral chronic shoulder pain (lasting > 6 weeks but < 24 months) used functional MRI of the brain to assess pain responses [53•]. The authors found that, compared with age- and sex-matched controls, individuals with chronic shoulder pain had changes on functional MRI that they interpreted as showing evidence that the experience of chronic shoulder pain amongst these patients may be mainly associated with cognitive affective pain processing, and perception modulation, rather than nociception [53•]. This study too provides evidence to suggest that chronic shoulder pain is perpetuated by central pain pathways even if the original cause of the shoulder problem was peripheral injury.

Further insight comes from a study by Jain and colleagues who explored predictors of pain and functional outcomes after surgery for rotator cuff tears [54•]. The authors found that none of socio-demographic characteristics; MRI findings (including tear size and muscle quality); shoulder strength; or variations in surgical techniques/performance of adjuvant surgical procedures predicted post-operative pain or function. However, they showed that pre-operative beliefs expressing less fear-avoidance for physical activity and mild weekly alcohol consumption (1–2 units), as compared with < 3 units/monthly, were the only factors which predicted better functional outcomes.

Taken together therefore, there is growing evidence for a role of central pain processing and perhaps other factors including beliefs, expectations, perceptions, somatisation, mood and affect both in the development of shoulder pain, and in its persistence to chronicity. What however is currently less clear is to what extent, if any, there is interaction between patho-anatomical entities and these factors.

Treatment of shoulder pain

Table 2 summarises a synthesis of the results from systematic reviews exploring the effectiveness of different treatment modalities for shoulder pain. Although more than 400 trials have been published, most studies have included a small number of participants, many are of poor quality, most have not measured outcomes beyond 6 months, and few have directly compared different active treatment options so that altogether, they offer little evidence to inform clinical decision making. Whilst there is short-term evidence for the effectiveness of some modalities, there is no conclusive evidence of benefit for any of the treatments in the longer term. Although the trials in Table 2 featured treatments

Table 2. Summary of the evidence from systematic reviews for the effectiveness of treatments for shoulder pain

Modality of treatment	Case definition	Review date, first author (ref)	No of trials included	No. of participants	Risk of bias (included trials)	Summary of results
NSAIDs vs placebo	Rotator cuff		tendinopathy	2014 Boudreault et al. [86]	12	n=10 to 108 per arm
Mean quality considered to be low to moderate	Oral		non-steroidal		anti-inflammatory drugs were found to provide short-term (2–4 weeks) pain relief (mean difference: -2.69, 95% CI -1.96 to -3.41), but not difference in function.	
Corticosteroid injection vs placebo/no injection	Rotator cuff		tendinopathy	2019 Lin et al. [87●●]	23 RCTs, 18 included in network meta-analysis	n=12 to 55 per arm
6/23 RCTs had high risk of bias in at least one domain	For patients with rotator cuff		tendinopathy, corticosteroid injection is significantly more effective than placebo pain reduction and functional improvement in the short term (3–6 weeks: SMD 0.51, 95% CI, 0.01–1.01 for pain and 0.33, 95% CI, 0.001 to 0.67 for function) but			

Table 2. (Continued)

Modality of treatment	Case definition	Review date, first author (ref)	No of trials included	No. of participants	Risk of bias (included trials)	Summary of results
	Adhesive capsulitis	2017 Wang et al. [88••]	not in the long term (>6–12 weeks) 4 RCTs + 1		non-randomised trial	$n=8$ to 40 per arm
All RCTs had high risk of bias in at least one domain, but overall quality considered moderate-high			Intra-articular corticosteroid injections were more effective in reducing pain at 0 to 8 weeks (MD -16.30; 95% CI -23.65 to -8.94), but there was no difference at 9 to 24 weeks.			
NSAIDs vs					corticosteroid injection	Shoulder pain
2014 Zheng et al. [89]	6 trials	267	participants, $n=15$ to 31 per arm	Variable (Jadad score 1–5)	1. For “remission”, NSAIDs were less effective than corticosteroids at 4 and 6 weeks (RR 0.64, 95% CI 0.45 to 0.92) 2. For pain relief, there was no difference for NSAIDs as compared with corticosteroids 3. For increasing the range of active abduction of the shoulder, there was no	

Table 2. (Continued)

Modality of treatment	Case definition	Review date, first author (ref)	No of trials included	No. of participants	Risk of bias (included trials)	Summary of results
Image-guided vs. blind					difference for NSAIDs as compared with corticosteroids	Subacromial shoulder pain
2015 Wu et al. [90]	7 trials	445	participants, n=20 to 46 per arm	Mean quality considered to be moderate	corticosteroid injection for shoulder pain Statistically significant difference in favour of ultrasound-guided injection at 6 weeks (mean difference 1.19, 95%CI 0.39 to 1.98 for pain; 5.01, 95% CI 1.82 to 8.19 for disability)	
Exercise and/or mobilisation	Rotator cuff disease	2016 Page et al. [91]	60 trials	3620	participants	Overall risk of bias was low in 3, unclear in 14, and high in 43 trials
Fifty-two trials investigated effects of manual therapy alone or exercise alone. There was little or no difference in					patient-important outcomes between manual therapy alone and control (placebo, no treatment, ultrasound therapy, taping). There was little or no difference in patient-important outcomes between exercise alone and	

Table 2. (Continued)

Modality of treatment	Case definition	Review date, first author (ref)	No of trials included	No. of participants	Risk of bias (included trials)	Summary of results
Exercise and/or mobilisation	Subacromial shoulder pain	2016 Steuri et al. [92••]	5 trials	189	placebo or other treatment options participants	Overall quality of evidence was assessed as very low
Exercise was superior to no treatment for pain (SMD -0.94 , 95% CI -1.69 to -0.19), with specific shoulder exercises found to be more effective than general exercise.						
Exercise and/or mobilisation	Frozen shoulder					
24/32 RCTs scored high risk of bias for at least 1 domain	No trial compared a					
				2014 Page et al. [93]	32 trials	1836 participants
					(capsulitis) combination of manual therapy and exercise versus placebo or no intervention. The best available data show that a combination of manual therapy and exercise may not be as effective as corticosteroid	

Table 2. (Continued)

Modality of treatment	Case definition	Review date, first author (ref)	No of trials included	No. of participants	Risk of bias (included trials)	Summary of results
Rotator cuff repair plus exercise vs exercise alone At 12 months, pooled mean difference was significantly better in those treated with rotator cuff repair (for pain: MD -0.87, 95 CI -1.30 to -0.43; for function: 5.98, 95% CI 2.43 to 9.54), which was not considered clinically important	Full thickness rotator cuff tear	2019 Karjalainen et al. [94●●]	injection in the short-term. Karjalainen et al. [94●●]	3 trials	335 participants	All trials were at high risk of bias for several domains
Decompression surgery vs placebo Decompression surgery was not more effective than placebo (MD	Subacromial pain	2019 Karjalainen et al. [95●●]	Karjalainen et al. [95●●]	2 trials	284 participants	The 2 trials had no domains with high risk of bias

Table 2. (Continued)

Modality of treatment	Case definition	Review date, first author (ref)	No of trials included	No. of participants	Risk of bias (included trials)	Summary of results
0.26, 95% CI -0.33 to 0.84)						
Decompression surgery + exercise vs exercise alone	Subacromial pain	2019	Karjalainen et al. [95••]	6 trials	556 participants	4 of the 6 trials had high risk of bias in at least one domain
Decompression surgery was significantly better compared to exercise alone (-0.56, 95% CI -1.09 to -0.02; 4 RCTs), but this was not considered clinically important.						
Manipulation under anaesthesia	Frozen shoulder		(capsulitis)	2012 Maund et al. [96]	4 trials	n=20 to 65 per arm
Trials had variable quality, majority at least one domain with high risk of bias	No significant differences were found between MUA and home exercises for pain or function.					
Distension vs placebo or	Frozen shoulder		(capsulitis)	2012 Maund et al. [96]	3 trials	n=9 to 39 per arm

Table 2. (Continued)

Modality of treatment	Case definition	Review date, first author (ref)	No of trials included	No. of participants	Risk of bias (included trials)	Summary of results
steroid injection Trials had variable quality, majority at least one domain with high risk of bias	No significant differences were found between distension and placebo or		corticosteroid injection for pain or function.			

for “shoulder pain”, most trials reported results for treatment of rotator cuff tendonitis, adhesive capsulitis (frozen shoulder), and, in one case, calcific tendonitis. This may suggest that patho-anatomical separation of shoulder pain allows separation of types of shoulder pain that will respond to different treatments. Unfortunately, the evidence here is far from convincing: firstly, the evidence suggests that clinicians can only poorly separate patho-anatomical abnormalities with clinical examination [55,56] even when examiners are taught together and using a pre-defined algorithm for diagnosis [55]. Interestingly, in the study by Bamji and colleagues, despite only 46% agreement about diagnoses, by the far the best agreement was that the optimal treatment would be an intra-articular steroid injection in 19/26 cases [56]. Qualitative research amongst physiotherapists (certified orthopaedic clinical specialists) also showed inconsistency in the labelling of shoulder conditions and little impact of diagnostic labels on treatment decisions [57].

Assuming that localised pathology would respond maximally if treatment is applied to the site, one interesting randomised controlled trial compared the benefit for intra-articular steroid injection injected into the shoulder with that for intra-muscular steroid injection into the gluteal muscle [58]. They were not able to show any significant benefit of the shoulder injection compared with the intra-muscular administration. In fact, a Cochrane review of placement of corticosteroid injections with imaging guidance, as compared to without, found no convincing evidence that intra-articular steroid injections placed into the shoulder with imaging guidance were any more effective than those placed using anatomical landmarks and also no more effective than the intra-muscular injection into the gluteal muscle [59]. These findings were borne out by a recently published large RCT which also found no additional benefit for ultrasound-placed as compared with anatomically placed corticosteroid injections [60]. One could hypothesise that these studies collectively suggest that the benefits of corticosteroids are systemic, but an alternative explanation is that the localised patho-anatomical abnormality is only part of the cause of the ongoing shoulder pain which may or may not be corticosteroid-sensitive.

Furthermore, two randomised controlled trials have recently compared outcomes after sub-acromial decompression surgery vs placebo surgery for sub-acromial shoulder conditions [61,62]. Both studies showed that there were no differences between outcomes amongst those who received “real” decompression as compared to placebo surgery, suggesting that a mechanical approach to treating the impingement does not result in significantly better patient outcomes. Likewise, a recent trial amongst patients with frozen shoulder compared two surgical interventions aimed to release the joint capsule (manipulation under anaesthesia and arthroscopic release surgery) with early structured physiotherapy plus steroid injection and found none of the interventions to be clinically superior [63••].

Taken together therefore, there is currently rather limited evidence to suggest that patho-anatomical separation is usefully differentiating diseases with different response to therapies, medical or surgical.

Shoulder pain prognosis

On the other hand, it could be that separation of shoulder conditions patho-anatomically tells us something about prognosis. In 2011, a Delphi exercise

took place to achieve consensus about the most important prognostic factors which would predict persistent shoulder pain (less than 50% improved) 3 months after presenting in primary care [64]. After a systematic review, 46 criteria were considered in the Delphi and the final consensus was that the following 10 factors were most important: duration of pain, catastrophizing of pain, the history of symptoms, fear-avoidance beliefs, presence of neck pain, severity of disability, comorbid mental health, current sickness absence, and presence of multisite pain were the factors which best predicted chronicity [64]. Contemporaneously, a model was derived statistically using the records of almost 600 primary care patients. The statistical model resulted in the following list of factors predicting persistent shoulder pain: sporting injury (yes); longer duration of symptoms; co-existent low back pain; bilateral shoulder pain; functional restriction; other upper limb pain. When tested, both models performed similarly (area under the curve (0.6–0.7)) in correctly assessing prognosis. Notably, neither model considers patho-anatomical factors other than pain at other anatomical sites.

Subsequently, Kooijman and colleagues undertook a comprehensive systematic review of prognostic factors for shoulder pain in 2015 [65]. Between 2003 and 2014, they identified nine cohort studies which considered in total 60 prognostic factors for shoulder pain. Six studies were described by the authors as high-quality and three as low-quality according to their methodological quality assessment. They found convincing evidence that there was no prognostic association with shoulder physical examination findings, including whether or not range of motion was restricted; degree of restriction of motion; muscle strength; or baseline diagnosis of a large rotator cuff tear [65]. Likewise, treatments including physiotherapy and medication prescribed in primary care were also not shown to affect prognosis. In fact, the factors which best predicted poor prognosis were longer pain duration, higher pain intensity, co-existing neck pain, greater disability, and previous shoulder pain [65].

As we have already seen, there are few long-term follow-up studies after intervention but those we have all appear to suggest that most patients remain symptomatic in the medium to long-term, no matter what their starting diagnosis was and whether or not they received active treatment [24]. As argued by Croft et al. [66], the role of diagnosis in clinical practice is challenged by evidence that it does not always benefit patients and that factors other than pathoanatomical classification are important in determining patient outcome. In such cases, a prognostic framework that extends beyond diagnosis and incorporates a wide range of biological, psychological, and social information to predict future patient outcomes, could provide a basis for decision making and potentially more effective and efficient patient care.

Is shoulder pain therefore more like back pain?

In the early 1990s, based upon a wealth of evidence and primary research into the causes of and treatment for low back pain, the Clinical Standards Advisory Group produced new guidance for the diagnosis and management of low back pain [67]. The guidance suggested that most of low back pain was “simple mechanical back pain” and that our approaches were in danger of over-medicalising a common clinical state and causing or worsening disability, a point which was powerfully

reiterated by a recent review in the Lancet [68]. Since the 1990s, the approach to low back pain that has been recommended is a triage approach based upon red and yellow flags, minimal use of terminology, minimal investigation (no use of lumbar spine X-ray), and dispensing with the making of “anatomical” diagnoses for the majority [67,68]. Although the shoulder pain research field is nowhere near as established as the low back pain literature was in the 1980s, there do appear to be some important parallels developing in the evidence base for shoulder pain.

It is noteworthy therefore that initiatives both in the Netherlands [69••] and the UK [70•,71] involve approaching shoulder pain in a way that is more akin to that described above for low back pain. Importantly, both initiatives are focussed in primary care where the vast majority of shoulder pain presents and is managed and a different approach is possibly indicated for the small proportion of people with shoulder pain who reach secondary care. In the Netherlands, guidelines for the primary care management of shoulder pain propose a simple classification of shoulder pain into (i) subacromial shoulder pain (characterised by pain when lifting the arm); (ii) glenohumeral joint problems (characterised by pain and limitation of movement during abduction and rotation); (iii) other conditions, including shoulder pain related to problems of the cervicothoracic spine. Importantly, recommendations regarding treatment and referral are the same for subacromial and glenohumeral conditions, and are guided by the severity of pain, functional limitations, and impact on work and other activities, not by the presumed patho-anatomical diagnoses [69••]. In the UK, colleagues in Keele are investigating use of a single panel of questions for patients presenting with any one of five common musculoskeletal pain conditions (including shoulder pain) [70•,71]. After exclusion of any red flags, people with musculoskeletal pain are effectively “triaged” for the presence of widespread pain, and adverse psychological and behavioural traits that are likely to be associated with a poorer prognosis. The subsequent management of the pain is tailored accordingly. This approach has already shown promise for patients presenting with low back pain in primary care [72], and the trial of this approach for pain at other sites is underway [70•].

Conclusion

There is plenty of evidence that our current approach to shoulder pain is limited in its success. This is a common, costly problem which increases with age and, as longevity increases, is set to cause more disability. For the majority of cases of shoulder pain, we call for a new approach emphasising de-medicalisation, supporting people to manage their own symptoms and considering chronic shoulder pain as a chronic condition in its own right, much as is widely accepted for other chronic pain conditions such as low back pain.

Declarations

Conflict of Interest

Karen Walker-Bone declares that she has no conflict of interest. Danielle van der Windt declares that she has no conflict of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

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Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

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