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Shoulder disorders: a state-of-the-art review

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This paper provides an up-to-date overview of the occurrence, diagnosis, risk factors, prognostic indicators and outcome of shoulder disorder (SD), and of the validity and reproducibility of diagnostic classifications and diagnostic imaging techniques for SD. Furthermore, the available evidence on the effectiveness of non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroid injections and physiotherapy for SD is summarized on the basis of randomized controlled trials with an acceptable quality of their methods. The annual incidence of SD is estimated at about 7%, its 1-year period prevalence at about 51% and its lifetime prevalence at about 10%. While approximately 50% of all patients with SD seek medical care, about 95% are treated in primary health care. Of all new episodes of SD presenting to primary care, approximately 50% seem to resolve within 6 months, while about 40% seem to persist for up to 12 months. Several prognostic indicators for a favourable or a poor outcome of SD have been identified, but a comprehensive prognostic model is not available. While evidence for the prognostic validity of popular diagnostic classifications of SD is lacking, their reproducibility has been shown to be poor. The accuracy and clinical usefulness of diagnostic imaging techniques appear to be sufficiently verified for SD in secondary care, while their clinical usefulness in primary care and prognostic validity are not. NSAIDs and steroid injections for SD have been shown to be effective within 6 weeks, but their effect on long-term outcome remains unclear. There is very limited evidence for the effectiveness in SD of physiotherapy, including exercise therapy, ultrasound, electrotherapy, laser, mobilization and manipulation.

Key words: shoulder; occurrence; risk factors; diagnosis; prognosis; treatment; outcome; clinical trials; effectiveness; diagnostic classification; magnetic resonance imaging; computed tomography; diagnostic ultrasound; non-steroidal anti-inflammatory drugs; corticosteroid injections; physiotherapy.

Shoulder disorders (SD) constitute a medical, social and economic challenge to society. SD are frequently seen in the general population and are a common reason for seeking medical care. Hence, they are a relevant health problem for clinicians, providers and policy-makers in health care. Pain in the deltoid region is a prominent complaint for most patients with SD.[1] When this pain is severe, lying on the impaired shoulder may cause problems with sleeping. Many patients report stiffness when their pain is elicited or aggravated by movement. Pain and stiffness usually restrict the use of the arm and hand, and thereby limit daily activities during work and leisure time.[2,3] Occupation, work and psychosocial and behavioural factors are considered to be important constituents of disability in a large proportion of patients with SD.[4,5]

The time-honoured method of teaching medicine—see one, do one and teach one—has led to innumerable opinion-based recommendations on the prognosis, diagnosis and treatment of SD. The purpose of this paper is to provide an up-to-date

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overview of the evidence base on the prognosis, diagnosis and conservative treatment of SD. To this end, data from sound clinical studies and systematic reviews are summarized. Given its scope, this paper is meant to be neither a systematic nor a comprehensive review.

INCIDENCE, PREVALENCE AND RISK FACTORS

There are relatively few sound prospective studies available conducted in a general population that consider the incidence, prevalence and potential risk factors of SD. The available prospective studies provide insufficient information about the selected population and its risk status at inclusion. In addition, their operational definition of SD is often ambiguous. So far, the available data on the incidence, prevalence and potential risk factors of SD exhibit considerable variation.

Incidence and prevalence

Based on his Swedish population survey, Allander estimated the annual incidence of SD to be about 1% and reported that the annual incidence of SD peaks at 2.5% in the fourth and fifth decades of life.[6,7] During a survey between 1971 and 1975 in the USA, about 7% of the adult population between 25 and 75 years of age reported an episode of SD lasting at least 1 month during the preceding year.[8]

The 1-year period prevalence of SD in the adult general population in various countries is reported to range between 20% and 51%.[9,10] In a Swedish population survey, prevalence estimates ranged from 7% for those between 30 and 35 years of age, to 25% for those between 56 and 60 years of age.[11] In the UK, the prevalence in the adult population is reported to be about 15%[9], while for those over 70 years of age, the prevalence is estimated at 20%.[12–14] The point prevalence of SD in a Finnish population survey was much lower: about 2% between 30 and 64 years of age and about 1% for those aged 65 or more.[2] In a Swedish population, the point prevalence of SD at 79 years of age (average) was estimated to be 16%.[15] The lifetime prevalence of SD in the adult general population is suggested to be approximately 10%.[1]

Risk factors

Trauma (including contusion, fracture, rupture, minor instability and joint displacement), surgical intervention and intravenous infusion, high age, related thoracic kyphosis, acromioclavicular and glenohumeral osteophytes, and the impairment of consciousness have all been reported to have a causal relationship with SD.[16–19] Although generalized conditions such as osteoarthrosis, stroke, polyneuropathy, multiple sclerosis, rheumatoid arthritis, polymyalgia, fibromyalgia, ankylosing spondylitis and diabetes mellitus are reported to be associated with SD, they must be considered to be concomitant rather than causal.[2,7,17,20,21] In a US survey, nearly 40% of the adults with SD reported concomitant neck complaints.[8] There are indications that reduced mobility of the cervicothoracic spine and adjacent ribs plays a central role in maintaining SD.[8,16] Reduced mobility of this region has been reported to have a positive predictive value for SD of 84%, while persons with such a reduced mobility have a threefold risk of developing SD.[16]

It has been suggested that a depressive personality is a risk factor for SD[22], and psychosocial, cognitive and behavioural traits are considered to contribute generally to

the perpetuation of musculoskeletal pain and associated disability behaviour, including those of SD.[4,5,23–25] It is assumed that patients with inadequate coping styles and catastrophic beliefs, i.e. wrongful beliefs and expectancies about the detrimental consequences of the condition, may develop kinesiophobia—an irrational fear of movement and (re)injury—and movement-avoiding behaviour as a consequence of the elicitation or aggravated of pain during daily activities. With positive reinforcement of this fearful behaviour, for example by spouse solicitousness, patients are more likely to express pain and disability, and become inactive during daily life. Subsequent to pain and inactivity, reactivity of the sympathetic nerve system, muscular reactivity and depression have been demonstrated to decrease pain tolerance and augment painful experiences.[4,25–28] Hence, psychosocial, cognitive and behavioural traits are considered to be risk factors for the perpetuation of SD.

Studies in occupational settings[4,7,29–37] report that the occurrence of SD depends on the type of work and the population of workers included. The occurrence of SD seems to be related to workplace design, work with vibrating hand-tools, overuse, high workload, stressful work, monotonous work, poor job satisfaction, lack of autonomy and job control, perceived high demands, isolation and hostility, and little social support in the workplace, as well as a long time between breaks. However, in such studies, anthropometric variables do not seem to be related to SD, while female workers and workers of high age have been reported to be more vulnerable to SD.

COST-OF-ILLNESS AND MEDICAL CONSULTATION

Approximately 18% of all sick leave benefit claims for musculoskeletal disorders in Scandinavian countries in 1994 were reported to concern neck and shoulder problems.[38] In the Netherlands, musculoskeletal disorders account for about 6% of the total health care costs.[39] This means that they are the second most costly diagnostic group. Patients with SD are considered to be the third largest group of those with musculoskeletal disorders in primary health care, after patients with lower back and neck disorders.[40,41] These insurance data about cost-of-illness can be used to illustrate the socio-economic consequences of SD. However, such data have been reported to depend on the nature of the population included and the operational definition of SD.[38] Furthermore, the occurrence of SD and associated medical and work-related costs also depend on the local insurance policy.[38] The available studies on medical consultation for SD report from different countries and provide insufficient background information on the patients included, while their operational definition of SD often is ambiguous. As a consequence, their results show considerable variation.

It has been reported that nearly 50% of all patients with SD consult a physician.[42] It appears that patients seem to cope with their SD without (re)consulting their GP. In the UK, fewer than 40% of patients of 70 years of age and older with SD sought treatment[12–14], while two-thirds of patients with persistent or recurrent complaints included in a Dutch observational study did not seek treatment for their SD.[43] Approximately 5% of all consultations in general practice are reported to concern SD[1,40], and each year about 1 per 100 adults in the UK consult their GP with a new episode of SD.[9] Estimations of annual consultation rates of SD in general practice in the Netherlands range between 15 and 25 per 1000 patients.[44,45] In the Netherlands, about 5% of all patients with SD are referred to a physician in secondary health care[41,45], and in the US, about 95% of all patients with SD are treated in primary health care.[46] SD are reported to comprise about 10% of all referrals to physiotherapists in

the UK and the Netherlands.[40,17–50] An observational study in Dutch general practice showed that about 40% of patients consulted their GP at least once more in the year following initial consultation.[45]

DIAGNOSIS

The diagnosis of SD and its classification appears to be a controversial subject.[51] Fundamental to this controversy is the arbitrary nature of the diagnostic terminology and the lack of generally accepted diagnostic criteria. Pain on movement, a reduced passive range of motion and a loss of muscle strength, all during abduction and anteflexion, are the principal components of the diagnosis of SD. In general, pain on movement is associated with an entrapment of subacromial soft tissue under the coraco-acromial complex, a reduced range of motion with fibrosis and adhesions of the glenohumeral joint capsule and surrounding soft tissue structures, and a loss of muscle strength with tears of the rotator cuff muscles or biceps tendon. Initial inflammation and oedema resulting from entrapment are supposed to mature into chronicity with associated fibrosis and adhesions of the glenohumeral joint capsular and surrounding soft tissue structures. However, repeated entrapment is also considered to result in wear and tear, and eventually in partial or full-thickness tears of either the rotator cuff muscles or the biceps tendon. As yet, it remains to be shown whether these different conditions represent different entities, i.e. whether their course and outcome are different, or whether they represent different stages in a clinical spectrum, which evolve into each other.

It is assumed that the structure from which problems in the shoulders arise can be localized using selective soft tissue tension techniques, i.e. specific active, passive and resisted movements and manoeuvres. Accordingly, conditions that are supposed to be mutually exclusive have been labelled and arranged by Cyriax[52] and others[53–57] in quite detailed diagnostic classifications. However, the arbitrary nature of the popular diagnostic classification of Cyriax[52] has been shown in several studies. In three studies involving 101, 100 and 201 patients respectively[44,58,59], practitioners were unable to arrive at one particular diagnosis in many of the cases, indicating that the diagnostic criteria underlying the different diagnoses are not mutually exclusive. Furthermore, several studies have shown the poor reproducibility of Cyriax's diagnostic classification. After an independent and blinded assessment of 26 patients with SD from secondary care seen in random order, three rheumatologists achieved a 46% agreement on their diagnosis.[60] In another study with a similar design, two skilled physiotherapists achieved an agreement of 91% and a kappa coefficient of 0.88 after the diagnostic assessment of 21 shoulders in 19 patients.[61] However, little additional information is given about the patients included in this study. General practitioners and physiotherapists have reached an agreement of 63% and a kappa coefficient of 0.31 on the diagnosis of 120 patients with SD who were referred for physiotherapy.[62] In this latter study, agreement was possibly reduced by the considerable time between the subsequent assessments. Finally, two trained physiotherapists achieved a kappa value of 0.45 after the diagnostic assessment of 201 patients with SD, poor agreement being associated with severe pain, bilateral and persistent complaints.[59]

The purpose of any diagnostic process is to collect information that can be used to guide decisions about prognosis and treatment rather than to arrive at a specific diagnostic label. Given the available data on its poor clinical performance, Cyriax's diagnostic classification is not very useful when decisions about prognosis and

treatment have to be made. Similar limitations have been reported for diagnostic classifications of soft tissue disorders of the neck and upper extremity.[63] In addition, recent studies illustrate that the same is true for selective soft tissue tension principles and the related diagnostic classification of Cyriax when applied to musculoskeletal conditions in the lower extremity.[64–66]

Independently of each other, Winters et al[67] and De Jongh[58] have proposed very similar classifications for SD. On the basis of a cluster analysis and a factor analysis respectively, each concerning a group of about 100 patients with SD in primary health care, these authors have illustrated that patients can be divided consistently into three subgroups on the basis of pain and restriction of range of motion during the clinical evaluation of glenohumeral and scapulothoracic movement. First, they identified a large group of patients with clinically confirmed pain but without a reduced passive range of glenohumeral movement. De Jongh[58] further divided this subgroup into those with and those without a painful arc. Second, the authors identified a group of patients with both clinically confirmed pain and a severely reduced passive range of glenohumeral movement. Winters et al[67] divided this group into two further groups: patients with and without a severe or acute onset. Finally, a group of patients with a mild reduced passive range of glenohumeral movement was identified. This classification seems to be more comprehensive and less complex than the existing ones, while each of the identified subgroups are considered to be related to a particular treatment which is available in primary health care practice. However, the within and between observer reproducibility and prognostic validity of these systems have yet to be shown in prospective studies.

Diagnostic imaging techniques

Diagnostic imaging techniques such as magnetic resonance imaging (MRI), computed tomography (CT) and diagnostic ultrasound are assumed to be important in arriving at a diagnosis in patients with SD. In addition, they are considered to provide information in secondary health care practice for decisions about the treatment of displacement of the glenohumeral or acromioclavicular joint, rotator cuff tears and rupture of the glenohumeral joint capsule.[68] When such conditions are suspected in primary health care practice, patients are commonly referred for further diagnosis and treatment in secondary level health care. Most of the available diagnostic studies have compared different diagnostic imaging techniques with each other in cross-sectional designs. Their results have to be interpreted with caution as many are reported to be flawed or biased.[69–71] Since most have been conducted in secondary health care practice and use arthroscopy as the reference method, their relevance for primary care practice is debatable.

Diagnostic ultrasound is commonly used to detect soft tissue disorders in the shoulder; the visualization of movement during diagnostic ultrasound is considered to be a major advantage.[69] It is reported to be a simple and cheap technique, with little associated risk. However, diagnostic ultrasound appears to be difficult to master. On the basis of a pooled analysis of 63 studies on the diagnostic performance of ultrasound in SD, its sensitivity has been reported to range between 84% and 87%, and its specificity between 94% and 97%.[69] Its accuracy is described as depending on both the type of disorder and the skills of the radiologist.[69] CT and MRI commonly are used to detect joint and cartilage disorders in the shoulder. Based on a review of 11 out of 63 studies with sufficient information to allow assessment (MEDLINE 1983–1993), both CT and MRI have been reported to yield relatively many diagnostic errors[70], and they

appear to be of little help in detecting the site of the lesion and arriving at a diagnosis.[72] The sensitivity of CT has been shown to range between 17% and 100%, and its specificity between 55% and 100%.[70] The sensitivity of MRI ranges between 39% and 100%, and its specificity between 8% and 100%.[70] The combination of MRI with arthroscopy has been reported to improve its accuracy.[68]

There are very few well-designed studies comparing diagnostic imaging techniques for SD with the clinical signs and symptoms established during standardized clinical assessment.[69,70] Ure et al[74] reported that a clinical diagnostic assessment could detect only 53% of the cases with joint instability confirmed by arthroscopy. So far, the accuracy and clinical usefulness of MRI, CT and diagnostic ultrasound appear to be sufficiently established for SD in secondary care, but their clinical usefulness in primary care and their prognostic validity are not. This is because there are virtually no studies with a prospective design comparing different diagnostic imaging techniques with the outcome of SD. In future research, priority should be given to such evaluations.

OUTCOME AND PROGNOSTIC INDICATORS

There are several studies available describing the outcome and putative prognostic indicators of SD. Most studies have been conducted among patients seeking medical care in either primary or secondary health care practice. Hence, little is known about the natural history of SD, i.e. the recovery rate and remission rate in the general population. The studies that are available vary considerably with respect to their inclusion criteria, the risk status of patients at baseline, the operational definition of outcome and the procedure and duration of follow-up. Therefore, there is considerable variation in the available data on the outcome and prognosis of SD.

Outcome

Based on studies from secondary health care practice, it has been suggested that the long-term outcome of SD does not seem to be very favourable. Persistent pain and a limited range of movement have been reported after several years of follow-up.[7,17,74,75] A less pessimistic picture emerges from other, relatively well-conducted prospective studies in the community and primary health care practice.[43,45,76–78] However, there is a considerable amount of variation between the results of such studies, most probably because of differences in the operational definition of SD.

In a study in the UK with 166 patients consulting their GP with a new episode of SD, 21% reported recovery at a 6-month follow-up, and 49% at 12 months.[78] In a Dutch study with 349 patients seeing their GP with a new episode of SD, 23% reported to be recovered at a 1-month follow-up, and 59% at a 12-month follow-up.[43,45] About 55% of 137 patients with rotator cuff tendinitis were reported to suffer from residual complaints 19 months after their first medical consultation.[79] Thus, after 12 months, some 50% of all episodes of SD presenting in primary care appear to persist. Sobel et al report that about 50% of the 100 patients who presented themselves as being recovered 12 months after their initial visit to the general practitioner actually had residual complaints.[44]

Prognostic indicators

A favourable outcome within 3 months has been associated with mild trauma preceding symptoms[43,80], early presentation[43,79,81,82], preceding overuse and heavy and unusual

activities of the upper extremity[44,79], an acute onset[79], a high erythrocyte sedimentation rate[83], and the restricted prescription and use of medication.[84] A poor outcome of SD at approximately 3 months appears to be associated with severe pain on first presentation[43,78], a prior episode[78], a severe restriction of the passive abduction range[78,85], diabetes mellitus[20,21], concomitant neck pain[43], cervical spondylosis and radicular symptoms[21] increasing age[80,85], involvement of the dominant side[79,86] and sick leave from work.[84] Depressive complaints have been reported in association with chronic musculoskeletal pain.[26] Preliminary data suggest that psychosocial factors and personality traits, such as cognitions and behaviours, are likely to contribute to the persistence and recurrence of painful musculoskeletal conditions[4,5,22,23], including SD.[4]

As yet, no attempts have been made to construct a comprehensive prognostic model in properly designed prospective studies. However, in a cohort of patients referred to a physiotherapist, 40% of the 35 patients who fulfilled three or four criteria—namely (a) only dominant side impairment, (b) this being the first episode of SD, (c) a lack of pain radiating below the elbow, and (d) a lack of concomitant cervical or elbow disorder—were completely free of symptoms at a 6-week follow-up. In contrast, those patients who fulfilled none, one or two of these criteria achieved a similar recovery rate of 40% (72 out of 180) only after 12 weeks of follow-up.[87]

OUTCOME MEASURES

There is no 'gold standard' that provides a valid and reliable estimate for clinically relevant change in any subgroup of patients with SD. In many studies, clinically relevant change depends on the judgement of either clinicians or patients. Frequently, a standardized clinical assessment of signs and symptoms, such as pain, range of motion and muscle power, is used to evaluate the outcome of SD. These variables are considered to be process or surrogate measures for the outcome of SD, and little is known about their responsiveness. The reliability of the measurement of range of motion is described as being satisfactory.[88,89] Interobserver reproducibility with goniometers of shoulder abduction, flexion and lateral rotation appears to be sufficient (an interclass correlation coefficient (ICC) of around 0.87), while for extension, adduction and medial rotation, interobserver reproducibility appears to be poor (having an ICC in the region of 0.45).[90] Intraobserver reproducibility in all directions of movement was high in this same study (the ICC being approximately 0.95) and appears to be independent of the size of the goniometer.[90] The reproducibility of the measurement of pain and functional limitations with 7-point scales and visual analogue scales has been reported to be satisfactory.[91–96] In addition, some active motor tests have been reported to possess sufficient reliability for the assessment of the limitation of functional activities in patients with SD.[97,98]

Patient-perceived recovery, evaluated with disease-specific functional status measures, is considered to be an important outcome variable for SD in many clinical studies. Recently, almost simultaneously, four questionnaires were developed to assess the limitation of functional activities and movements of the arm in patients with SD. The Croft Disability Questionnaire (CDQ) includes 22 items with a yes, no answer scale and a 24-hour recall frame. The items of the CDQ concern functional activities and movements using the arm.[99] The Shoulder Pain and Disability Index (SPADI) consists of a separate 5-item pain scale and an 8-item disability scale, with a 7-day recall frame.[100] In order to make the SPADI suitable for telephone administration, the original visual analogue answer scales have been converted into 0–10 numerical scales.[101] The

Shoulder Rating Questionnaire (SRQ) consists of 19 items with a 5-point ordinal answer scale: 4 items relate to pain, 6 to daily activities, 3 to recreational and athletic activities, 5 to work and 1 to satisfaction. The SRQ also includes one visual analogue scale for global assessment, as well as one item to indicate the domain of most important improvement. The SRQ has a recall frame of 1 month.[102] The Shoulder Disability Questionnaire (SDQ) is a patient-completed 16-item questionnaire. The items have a 24-hour recall frame with 'yes', 'no' and 'not applicable' as response options.[103] All the four measures described above include items that refer to problems with sleeping and dressing. The overlap with respect to item content is greatest between the CDQ and the SDQ.

The responsiveness for the SDQ was compared with that for other measures in primary care physiotherapy at a 6-week follow-up. The area under the receiver operator characteristic curve (AUC) for the SDQ was 0.72, for severity of shoulder pain 0.80 and for functional mobility 0.67.[103] In general practice, the AUC for the SDQ at 3 and 6 months of follow-up was reported to be 0.84 and 0.90 respectively.[104] An AUC of 0.91 at 3 months follow-up[105] and a standardized response mean of 1.38 were reported[105] for the responsiveness of the SPADI in primary care in the USA. During all these evaluations of responsiveness, patient-reported improvement was used as the external criterion. As yet, it is unclear which of these functional status measures is the most responsive, and there is little information about their performance compared with that of more conventional outcome measures such as severity of mobility restriction, pain and symptoms.

EFFECTIVENESS OF CONSERVATIVE TREATMENT

The purpose of treatment is to influence the course and prognosis of SD by increasing the extent and speed of recovery. Patients with SD are believed to benefit from advice, analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), steroid injections, manipulation, mobilization and physiotherapy, including exercise therapy and the application of physical modalities. A substantial number of randomized controlled trials on these conservative treatments have been published. Here, only the results of the randomized controlled trials with an adequate design are outlined, in so far as they concern the success of treatment or pain relief according to the patients.

Non-steroidal anti-inflammatory drugs

The treatment of SD frequently is initiated with the prescription of NSAIDs. These are assumed to act by inhibiting prostaglandin synthesis, resulting in the relief of pain and the suppression of inflammatory processes in articular or peri-articular structures. Adverse reactions include mild gastrointestinal symptoms, which occur frequently, as well as the less frequent, but more serious, complications such as gastrointestinal bleeding, renal insuffiency, hepatitis and bronchospasm.[106]

The effectiveness of NSAIDs in SD was evaluated in a systematic review[106] including 19 randomized controlled trials (RCTs) harvested from Medline and Embase (before 1994). According to the systematic assessments of methods by two blinded reviewers, many RCTs provided insufficient information about the randomization procedure, the co-interventions and the control of compliance. The five RCTs[107–111] that yielded internally valid results are summarized in Table I. NSAIDs are considered useful in the short-term, i.e. within 4 weeks, management of SD on the basis of placebo-controlled

Table 1. Summary of randomized clinical trials with sound methods on the effectiveness of various forms of non-steroidal anti-inflammatory drug (NSAIDs).

Authors (Condition included)	Treatments compared	Follow-up and success rate per group, and difference between group [95% confidence interval]
Petri et al[107]	① 1000 mg naproxen 4 weeks daily ($N = 50$) ② Placebo tablets 4 weeks daily ($N = 50$)	4 weeks ① 12/50 ② 8/50 No statistically significant difference
(painful shoulders)	① and ② Either triamcinolone or lidocaine injection	① minus ② 0.06 [–0.1↔0.2]
Adebajo et al[108]	① 150 mg diclofenac 4 weeks daily ($N = 20$) ② Placebo tablets 4 weeks daily ($N = 20$)	4 weeks ① 6/20 ② 0/20 Statistically significant difference
(rotator cuff tendinitis)	① and ② One subacromial lignocaine injection	0.3 [0.1↔0.5]
Mena et al[111]	① Flurbiprofen, decreasing dosage from 300 mg to 200 mg 14 days daily ($N = 35$) ② Placebo tablets 14 days daily ($N = 34$)	2 weeks ① 30/35 ② 19/34 Statistically significant difference
(acute tendinitis or bursitis)		0.3 [0.1↔0.5]
Berry et al[109]	① 1200 mg tolmetin sodium 4 weeks daily ($N = 12$) ② Placebo tablets 4 weeks daily ($N = 12$)	4 weeks ① 6/12 ② 5/12 No statistically significant difference
(painful shoulders)	① and ② One subacromial prednisolone/lignocaine injection	0.08 [–0.32↔0.48]
Zuinen[111]	① 100–150 mg diclofenac plus 400–600 mg misoprostol both 2 weeks daily ($N = 187$) ② 100–150 mg diclofenac daily ($N = 185$)	4 weeks ① 93/185 2 78/187 No statistically significant difference 0.01 [–0.09↔0.11]

RCTs with reasonably sound methods.[108,110] The available RCTs do not allow conclusions to be made about the risk-to-benefit ratio of NSAIDs. As yet, it remains unclear whether NSAIDs modify the long-term outcome or reduce the recurrence rate of SD. Despite the higher risk of adverse reactions with NSAIDs there are no RCTs comparing the effectiveness of NSAIDs with simple analgesics or with a wait-and-see policy. Priority should be given to properly designed RCTs addressing these questions.

Steroid injection

The injection of a local anaesthetic can be helpful in discerning whether pain on movement in a stiff shoulder originates from the impingement of subacromial soft tissue under the coraco-acromial complex. Disappearance of the pain (or painful arc) after the subacromial injection of a local anaesthetic is assumed to confirm such an impingement.[112] Adding a corticosteroid, such as triamcinolone, to this injection is believed to suppress the inflammatory reaction of soft tissue arising secondary to the impingement[112,113] and thereby speed up recovery. When the pain does not disappear after a subacromial injection with a local anaesthetic, it is assumed to originate from adhesions of the glenohumeral joint capsule and its surrounding soft tissue structures. Patients with severely reduced mobility of the shoulder are considered to benefit from an intra-articular injection of steroid.[113]

Dermal atrophy, bacterial arthritis, haemarthrosis and thrombophlebitis have been attributed to problems with injection technique, while systemic post-injection flare, urticaria and facial flushing have been ascribed to suspension preservatives. Ligamentous laxity, joint instability and the calcification or rupture of tendons and joint capsules have been associated with injections into tendons and repeated depot injections of the same joint.[113]

The effectiveness of steroid injections for SD has been evaluated in a systematic review[113] including 16 RCTs found in Medline and Embase (up until December 1995). The systematic assessments of the methods by two blinded reviewers revealed frequent poor blinding of the therapist and incomparability of the co-treatments. Furthermore, the assessment of methods was often hampered by incomplete information on the randomization procedure, prognostic comparability, treatment compliance, outcome variables and blinding of patients and outcome measurement. According to the systematic assessments of study methods, only three RCTs[107,108,114] were considered to yield internally valid results (Table 2). After this systematic review, two other systematic reviews[115,116] were published. None of these three reviews included the RCTs of Van der Windt et al[117], Winters et al[118] and Blair et al.[119] Because the methods of these RCTs seem sound, they are summarized in Table 2.

On the basis of the RCTs in Table 2, subacromial and intra-articular triamcinolone injections are effective within about 6 weeks for shoulders with soft tissue impairment or severely restricted mobility. Subacromial prednisolone injection does not appear to be effective for shoulders with soft tissue impairment.[114] There is as yet no indication of which patients, and at what time in the course of their SD, benefit most from which steroid injection. To date, it is unclear whether injecting only a local anesthetic is more effective than a wait-and-see policy, and whether multiple injections are more effective than single ones. Moreover, there is no evidence for a beneficial effect of steroid injections in the long term or in reducing the recurrence of SD. Thus, priority should be given to properly designed RCTs comparing the effectiveness of injecting only a local anaesthetic with its steroid combination, and contrasting each of these with a wait-and-see policy.

Table 2. Summary of randomized clinical trials with sound methods on the effectiveness of various forms of steroid injection

Authors (Condition included)	Treatments compared	Follow-up and success rate per group, and difference between group [95% confidence interval]
Petri et al[107] (painful shoulders)	① One subacromial injection of 40 mg intrabursal triamcinolone/lidocaine ($N = 50$) ② One subacromial injection of lidocaine ($N = 50$) ① and ② 1000 mg naproxen or placebo tablets 4 weeks daily	2 weeks ① 14/50 ② 10/50 No statistically significant difference 0.08 [–0.09↔0.25] 4 weeks 1 28/50 ② 14/50 Statistically significant difference 0.28 [–0.09↔0.47]
Adebajo et al[108] (rotator cuff tendinitis)	① One subacromial injection of 80 mg triamcinolone hexacetonide/lignocaine ($N = 20$) ② One subacromial injection lignocaine ($N = 20$) ① and ② Placebo diclofenac	4 weeks ① 14/20 ② 0/20 Statistically significant difference 0.7 [0.5↔0.7]
Vecchio et al[114] (rotator cuff tendinitis)	① One subacromial injection of 40 mg subacromial methylprednisolone/lignocaine ($N = 28$) ② One subacromial injection of lignocaine ($N = 28$)	12 weeks ① 9/28 ② 7/28 No statistically significant difference 0.06 [–0.18↔0.30]
Blair et al[119] (rotator cuff impingement)	① One subacromial injection of 2 ml 40 mg/ml triamcinolone plus 6 ml 1% lidocaine ($N = 19$) ② One subacromial injection of 6 ml 1% lidocaine ($N = 21$) ① and ② Exercise therapy	7 months ① 16/19 ② 6/21 Statistically significant difference 0.55 [0.30↔0.80]
Van der Windt et al[117] (painful shoulders with restricted mobility)	① Up to 3 subacromial injections of 40 mg triamcinolone acetonide in 6 weeks ($N = 52$) ② Physiotherapy, mainly exercise therapy and mobilization ($N = 56$)	7 weeks ① 40/52 ② 26/56 Statistically significant difference 0.31 [0.14↔0.48]
Winters et al[118] (either a painful shoulder or restricted glenohumeral mobility)	① Subacromial, or glenohumeral or acromioclavicular inta-articular multiple injection 1 ml 40 mg/ml triamcinolone acetonide and 9 ml with 10 mg/ml lignocaine ($N = 47$) ② Physiotherapy ($N = 35$) ③ Manipulation ($N = 32$)	5 weeks ① 35/47 ② 7/35 ③ 13/32 Statistically significant differences ① minus ② 0.55 [0.37↔0.73] ① minus ③ 0.35 [0.14↔0.56]

Physiotherapy

The restoration of functional capacity, pain relief and the improvement of mobility are the primary objectives of physiotherapy for SD. Exercise therapy is considered to be the cornerstone of physiotherapy for SD. It is frequently combined with manipulation, mobilization and physical modalities such as ultrasound, laser and transcutaneous electrotherapy.

The effectiveness of physiotherapy for patients with soft tissue SD was evaluated in a narrative review[120] and a systematic review[121], together including 20 RCTs found in Medline and Embase (up until January 1996). Systematic assessments of the methods by two blinded reviewers[121] revealed frequent poor blinding of the therapist and high proportions of treatment withdrawals and missing values. Furthermore, the assessment of methods was often hampered by incomplete information on the randomization procedure, prognostic comparability and co-treatments. In total, six RCTs[109,122–126] were considered to report internally valid results. After the release of this systematic review, other systematic reviews about transcutaneous electrotherapy[127], ultrasound[128] and laser therapy[129] reported that the design of four additional RCTs on SD were relatively sound.[130–133] Furthermore, three RCTs on SD including physiotherapy[87,117,118,124], have been published which were included in neither of these reviews. Since their methods are apparently sound, they have been included in Table 3.

From the 14 RCTS in Table 3, it can be concluded that there is some evidence for the effectiveness of exercise therapy when compared with no treatment[134], and for laser therapy compared with placebo.[125] Furthermore, sound RCTs show an apparent lack of effect of ultrasound therapy[87,109,122,130] and transcutaneous electrotherapy[87,132–133] in SD. Although it is not frequently used, there is some evidence for the effectiveness of (electro)magnetic field therapy in SD.[123,124] Finally, there are no internally valid RCTs available for other popular physiotherapy modalities used for SD, for example massage, heat and cold.

As yet, evidence for an effect of physiotherapy on the long-term outcome or recurrence of SD is lacking. From the available RCTs, it can be concluded that physical modalities do not contribute to the recovery of patients with SD. In addition, although exercise therapy is considered to be the cornerstone of physiotherapy, there is very limited evidence of its effectiveness. Furthermore, it remains unclear whether physiotherapy for SD is more effective than analgesics or a wait-and-see policy. Hence, priority should be given to properly designed RCTs comparing the effectiveness of exercise therapy against these two approaches. In addition, an evaluation of the effectiveness of physiotherapy as an alternative in the treatment of patients with SD with a high risk for the deleterious effects of NSAIDs or steroid injections should be considered.

Cognitive behavioural interventions

Some limited but promising results for the effectiveness of cognitive behavioural treatment in the prevention and treatment of SD have been reported.[4] Currently, a cognitive behavioural intervention package for the prevention of chronicity in subacute SD is being developed on the basis of similar management strategies that are available for other musculoskeletal conditions. Such treatment includes information, instruction and education in the areas of coping and self-care, misinterpretation and relabelling of the risk of (re)injury by pain and movement, and an unambigious and systematic stimulation of (gradual) resumption of usual activities. Before their clinical implementation, the effectiveness of cognitive behavioural interventions will have to be evaluated in well-designed RCTs.

Table 3. Summary of randomized clinical trials with sound methods on the effectiveness of various forms of physiotherapy

Authors (Condition included)	Treatments compared	Follow-up and success rate per group, and difference between group [95% confidence interval]
Brox et al[127] (stage II impingement syndrome)	① Exercise therapy twice a week 6 weeks ($N = 50$) ② Placebo laser therapy twice a week 6 weeks ($N = 30$) ③ Arthroscopic surgery, subacromial decompression ($N = 45$)	Median change in pain (100-point scale) 3 months ① 15 ② 15 ③ 25 6 months ① 25 ③ 25 No statistical (median) difference (3 and 6 months) ① minus ③ –5 [–10↔0]
Ginn et al[135] (local mechanical shoulder pain)	① Exercise therapy up to 10 sessions 1 month ($N = 38$) ② No treatment ($N = 28$)	1 months ① 32/38 ② 5/28 Statistically significant difference 0.66 [0.48↔0.84]
Winters et al[119] (shoulder pain with reduced mobility of the cervicothoracic spine and adjacent ribs)	① Physiotherapy, mainly exercises ($N = 29$) ② Mobilizations ($N = 29$)	5 weeks ① 3/29 ② 20/29 Statistically significant difference ① minus ② –0.6 [–0.8↔–0.4]
Winters et al[119] (either a painful shoulder or restricted glenohumeral mobility)	① Physiotherapy ($N = 35$) ② Manipulation ($N = 32$) ③ Multiple steroid injection ($N = 47$)	5 weeks ① 7/35 ② 13/32 ③ 35/47 Statistically significant differences ① minus ② –0.55 [–0.73↔–0.37] ① minus ③ –0.21 [–0.43↔0.01]
Van der Windt et al[118] (painful shoulders with restricted mobility)	① Physiotherapy, mainly exercise therapy and mobilization for 6 weeks ($N = 56$) ② Multiple steroid injections ($N = 52$)	7 weeks ① 40/52 ② 26/56 Statistically significant difference –0.31 [–0.48↔–0.14]
Høyer et al[133] (post-stroke arm pain)	① Microcurrent 6 sessions ($N = 8$) ② Placebo current 6 sessions ($N = 7$)	3 days mean (SD) change pain (0–100-point scale) ① 17 (24.2) ② –3 (29.7) No statistically significant (mean) difference 20 [–10↔50]

Table 3.—(*cont.*)

Authors (Condition included)	Treatments compared	Follow-up and success rate per group, and difference between group [95% confidence interval]
Van der Heijden et al[88]	① Transcutaneous bipolar interferential current 12 sessions 6 weeks (*N* = 73)	6 weeks ① 0.23 ② 0.22 ③ 0.20
	② Placebo electrotherapy 12 sessions 6 weeks (*N* = 72)	3 months ① 0.41 ② 0.39 ③ 0.40
(painful shoulders or limited range of motion)	③ No electrotherapy (*N* = 35)	No statistically significant differences
	①, ② and ③ Exercise therapy 12 sessions 6 weeks	① minus ② 0.01 [–0.13↔0.15] and 0.02 [–0.14↔0.18]
	① and ② Ultrasound or placebo ultrasound	① minus ③ 0.03 [–0.033↔0.39] and 0.01 [–0.19↔0.21]
Leandri et al[134]	① High-intensity TENS 3 times a week 4 weeks (*N* = 20)	1 month ① 15/20 2 5/20 3 5/20
(post-stroke arm pain	② Low-intensity TENS 3 times a week 4 weeks (*N* = 20)	2 months ① 18/20 ② 2/20 ③ 1/20
	③ Placebo TENS 3 times a week 4 weeks (*N* = 20)	Statistically significant differences
		① minus ② 0.5 ([0.23↔0.77] and 0.8 [0.61↔0.99])
		① minus ③ 0.5 [0.23↔0.77] and 0.85 [0.69↔1.00]
		No statistically significant differences
		② minus ③ 0 [–0.27↔0.02] and 0.05 [–0.11↔0.21]
Van der Heijden et al[88]	① Pulsed ultrasound 12 sessions 6 weeks (*N* = 73)	6 weeks ① 0.26 ② 0.19 ③ 0.20
	② Placebo ultrasound 12 sessions 6 weeks (*N* = 72)	3 months ① 0.42 ② 0.38 ③ 0.40
(painful shoulders or limited range of motion)	③ No ultrasound (*N* = 35)	No statistically significant differences
	①, ② and ③ Exercise therapy 12 sessions 6 weeks	① minus ② 0.07 [–0.07↔0.20] and 0.04 [–0.12↔0.20]
	① and ② Either electrotherapy or placebo electrotherapy	① minus ③ 0.06 [–0.11↔0.23] and 0.02 [–0.18↔0.22]
Downing and Downing[123]	① Continuous ultrasound 3 sessions per week 4 weeks (*N* = 11)	4 weeks ① 7/11 ② 4/9
	② Placebo ultrasound 3 sessions per week 4 weeks (*N* = 9)	No statistically significant difference
(supraspinatus tendinitis or subacromial bursitis or adhesive capsulitis)		0.2 [–0.23↔0.63)
Berry et al[110]	① Ultrasound (*N* = 12)	4 weeks ① 6/12 ② 9/12 ③ 5/12
	② Placebo ultrasound plus placebo tablets (*N* = 12)	No statistically significant differences
(rotator cuff disorder)	③ Acupuncture (*N* = 12)	① minus ② –0.25 [–0.62↔0.12]
		① minus ③ 0.08 [–0.32↔0.48]

Table 3.—(*cont.*)

Authors (Condition included)	Treatments compared	Follow-up and success rate per group, and difference between group [95% confidence interval]
Nykänen[131]	① Pulsed ultrasound (*N* = 35) ② Placebo ultrasound 12 sessions 4 weeks (*N* = 37)	4 weeks mean (SD) pain reduction (5-point scale) ① 2.5 (0.7) ② 2.4 (0.9)
(painful shoulders)	① and ② Exercise therapy	4 months (20-point scale) ① 13 (5) ② 13 (4) No statistically significant differences 5-point scale 0.1 [–0.3↔0.5] 20-point scale 0 [–2.1↔2.1]
Saunders[126]	① Laser 3 times per week 3 weeks (*N* = 12) ② Placebo laser 3 times per week 3 weeks (*N* = 12)	3 weeks statistically significant differences Graphical presentation of pain outcomes
(supraspinatus tendinitis)		
Vecchio et al[132]	① Laser 2 sessions per week 8 weeks (*N* = 19) ② Placebo laser 2 sessions per week 8 weeks (*N* = 16)	Mean (SD) change pain on movement (10-point scale) 4 weeks ① 2.7 (0.8) ② 1.2 (0.9) 8 weeks ① 3.6 (1.0) ② 1.8 (1.2) Statistically significant differences 1.5 [0.9↔2.1] and 1.8 [1↔2.6]
(rotator cuff tendinitis)		
Binder et al[124]	① Pulsed electromagnetic fields (PEMF) 5–9 times a day 1 hour 4 weeks (*N* = 15) ② Placebo PEMF 5–9 times a day 1 hour 4 weeks (*N* = 14)	4 weeks no statistically significant differences Graphical presentation of pain outcomes
(rotator cuff tendinitis)		
Leclaire and Bourgouin[125]	① Magnetotherapy 3 sessions per week 12 weeks (*N* = 22) ② No magnetotherapy 3 sessions per week 12 weeks (*N* = 25)	12 weeks mean (SD) change pain (4-point scale) ① 1.6 (0.6) ② 1.4 (0.7)
(painful restricted shoulders)	① and ② Thermotherapy, manual stretching and exercise therapy	No statistically significant differences 0.2 [–0.2↔0.6]

PRACTICAL GUIDELINES

The Dutch national guideline set for the diagnosis and treatment of SD seems to be the only one available. Its first edition was issued in 1990 by the Dutch College of General Practitioners[135] and primarily concerned SD with clinically established local mechanical pain and/or restricted mobility. This guideline has now been updated according to the evidence available in international literature.[136] For diagnostic triage, the second edition of this guideline uses the classification proposed by De Jongh[58] and Winters et al.[67] Other diagnostic procedures and imaging techniques are not included because they appear to provide little information for decisions about treatment in primary care practice. The principal recommendation for the management of SD in this guideline is watchful waiting for the beneficial natural course of newly presented episodes of SD in the majority of patients. Meanwhile, the patient is advised to stay active but avoid extremely painful movements. The first-choice medication for pain relief is paracetamol 500–1000 mg 3–4 times daily for 2 weeks. Thereafter, NSAIDs, i.e. ibuprofen 400 mg 3–4 times daily, diclofenac 50 mg 3–4 times daily or naproxen 250 mg 2–3 times daily, can be used for 2 weeks in patients with persistent SD. An intra-articular injection of triamcinolone is only endorsed for patients with persistent SD accompanied by clearly restricted passive glenohumeral external rotation. In addition, a subacromial injection of triamcinolone and lidocaine is recommended only for patients with pain on abduction and persistent SD. Physiotherapy is endorsed only for patients with 6 weeks' persisting SD accompanied by a severe limitation of daily activities.

SUMMARY

SD occur frequently, and the costs of medical treatment and invalidity and sick benefit claims are relatively high. Figures on the occurrence, risk factors, medical consultations and associated costs vary with the operational definition of SD. Estimations from population surveys for the annual incidence of SD range up to 7%, for its 1-year period prevalence up to 51%, and for its lifetime prevalence up to 10%. Many physical factors, as well as some psychosocial, personality and behavioural factors, appear to increase the risk of development of SD. The diagnosis and treatment of SD constitute a challenge for clinicians. About 50% of all patients with SD consult a physician, and approximately 95% of these are treated in primary care. The reproducibility of popular diagnostic classifications is poor, while information about their prognostic validity in lacking. A simple and comprehensive classification has been proposed, although its reproducibility and prognostic validity have yet to be shown. The accuracy and clinical usefulness of MRI, CT and diagnostic ultrasound have been established for SD in secondary care, but there is insufficient evidence for their prognostic validity and clinical usefulness in primary care.

About 23% of all new episodes of SD resolve within 6 months after presentation in primary care, but at least 50% are reported to persist at 12 months. Data on prognostic indicators for a favourable and a poor outcome of SD at 3 months appear to be consistent, but a comprehensive prognostic model is not yet available. RCTs with sound methods show that NSAIDs and steroid injections for SD are effective within 6 weeks, but there is no evidence on their effect on long term outcome. Furthermore, these RCTs provide very limited evidence for the effectiveness of various forms of physiotherapy for SD.

Practice points

- many patients with SD in the general population seem to cope without (re)consulting their GP for it
- of all new episodes of SD presented in primary care about 50% appear to persist for a year
- a poor outcome of SD at approximately 3 months is reported to be associated with severe pain and sick leave from work at first presentation, involvement of the dominant side, severe restriction of passive abduction range, a prior episode, concomitant neck pain, cervical spondylosis and radicular symptoms, high age, and diabetis mellitus
- the diagnostic classification of SD of Cyriax appears to be of little value for diagnostic and therapeutic decisions
- the accuracy and clinical usefulness of MRI, CT and diagnostic ultrasound appears to be sufficiently established for SD in secondary care populations, not for primary care populations
- patients with SD in primary care can be divided consistently in 3 subgroups: (1) pain without restricted movement; (2) pain plus severely reduced passive range of glenohumeral movement; (3) mildly reduced passive range of glenohumeral movement
- NSAIDs have been demonstrated to speed up overall recovery and pain reduction within 4 weeks
- subacromial triamcinolone injections for soft tissue shoulder impairment and intra-articular triamcinolone injections for severely restricted shoulder mobility have been demonstrated to speed up overall recovery and pain reduction within 6 weeks
- there is no evidence for an effect of physiotherapy on the outcome or recurrence of SD in terms of overall recovery and pain reduction
- there is sufficient evidence that physical modalities do not contribute to the overall recovery and pain reduction of patients with SD

The principal recommendations of the revised Dutch guideline on the management of SD are:

- watchful waiting for 2 weeks with paracetamol for pain relief and the advice to stay active and avoid extremely painful activities
- thereafter, ibuprofen or naproxen for persistent SD, 2–4 weeks initially
- intra-articular triamcinolone injection when glenohumeral exorotation is severely restricted (repeated up to 3 times, fortnightly)
- subacromial lidocaine/triamcinolone injection when there is pain during abduction (repeated up to 3 times, fortnightly)
- physiotherapy for persistent SD after 6 weeks only when daily activities are severely limited

Research agenda

- as yet, a comprehensive prognostic model for SD is lacking
- the prognostic validity of diagnostic imaging techniques remains unclear
- it is unclear which of the available functional status questionnaires is the most responsive, while there is little information on their performance compared to more conventional outcome measures such as severity of mobility restriction, pain, and symptoms
- there is insufficient evidence for the superiority of the effect of an injection with a local anaesthetic plus triamcinolone, compared to a local anaesthetic alone, and a wait-and-see policy
- it is unclear whether NSAIDs and triamcinolone injections modify the long term outcome or reduce the recurrence rate, and whether multiple triamcinolone injections are more effective than a single one
- evidence for the effectiveness of cognitive-behavioural treatment in prevention and treatment of SD is lacking

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