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Shoulder joint dysfunctions:

Where to look for the cause?

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Abstract: Shoulder joint dysfunction manifested in the form of pain, muscle weakness and limited range of motion is almost always associated with anatomical disorders both of degenerative or inflammatory origin. In many cases the causes of the disease remain unclear.

In this paper I consider three approaches for clinicians: the orthopaedic model, the fascial dysfunction model, and the neuromuscular (reflex) model. Each of these approaches is justified in terms of aetiology, treatment and has statistically reliable positive results.

Muscles, although they depend largely on ligaments, tendons, fascia, play a key role in stabilisation and movement. Being organised into subsystems of muscle-fascial chains (MFC) they ensure the functioning of a single system of biotensegrity.

The large number of approaches and proposed treatment methods highlights the complexity shoulder joint diseases problem. Each of the models presented here demonstrates significant advantages, and which of these models a specialist should choose depends on many factors

Indexing Terms: shoulder; adhesive capsulitis; frozen shoulder; Impingement syndrome; rotator cuff; fascia.

Introduction

The big number of different complaints on pain and dysfunction in shoulder joint and shoulder area can confuse any specialist.

We would not consider the trauma-provoked shoulder pain. Firstly, because either fracture or dislocation are sufficient grounds for pain and functional limitations at the level of shoulder joint. Secondly, because trauma-associated pain is much less common than pain that occurs spontaneously which is much more difficult to explain.

We will not also consider problems arising due to systemic pathological processes, such as oncology, osteoporosis etc. Here, the aetiology of shoulder joint dysfunction is also more or less evident.

More often, specialists are faced with the problem of diagnosing and curing of exactly those dysfunctions that arose suddenly (or gradually) without any visible reason, and despite the treatment continue to disturb the patient. Symptoms are increased, life quality and ability to work are going to be significantly impaired.

Like in other cases, it also important here which concept determines the approaches to diagnosis and subsequent treatment.

> *… success of the specialist in the prevailing majority of cases will depend primarily on the correct diagnosis because, according to medical proverb 'Qui bene diagnoscit, bene curat' (whoever diagnoses well, treats well) …*

We will consider three approaches:

- orthopaedic
- fascial dysfunction model and
- neuromuscular (reflex).

Each of these approaches is justified in terms of aetiology, treatment and has statistically reliable positive results.

Orthopaedic model

A peculiarity of orthopaedic approach is that the shoulder joint dysfunction manifested in the form of pain, muscle weakness and limited range of motion is almost always associated with anatomical disorders both of degenerative or inflammatory origin. In many cases the causes of the disease remain unclear. (Table 1)

Table 1: Nosology of shoulder joint dysfunction.

Nosological form	Frequency of detection	Key Features/Reasons
Symptomatic shoulder pain:	The 3rd place among muscloskeletal complaints (after the lower back and neck)	Symptom but not diagnosis. Causes: overload, microtrauma, degenerative changes, reflected pain (cervical spine, heart, gallbladder). Prevalence increases with age and with heavy physical work.
Shoulder impingement syndrome:	The most frequent cause of chronic shoulder pain, up to 44 - 65% of all visits.	Subacromial space narrowing → compression of supraspinatus tendon and bursa; predispos- ing — osteophytes, shape of the acromion and muscle imbalance. Often registered in people who perform overhead arm movements.
Biceps tendinopathy:	In 5-7% of patients with shoulder pain; often accompanies other shoulder pathologies.	Tendon overload inside the intertubercular groove, impingement, tendon instability, repetitive overhead movements. Often seen in athletes and people who perform repetitive arm movements. Often associated with other pathologies such as rotator cuff tears.
Supraspinatus tendinopathy:	The most often shoulder tendinopathy, registered up to 60% of rotator cuff tears.	*Supraspinatus* tendon both microtrauma and ischaemia during impingement, age-related degeneration.
Shoulder joint bursitis:	Up to 20% of patients with acute shoulder pain; often secondary to impingement or trauma	Often induced by repetitive motion or trauma. Prevalence increases with age. Overuse, impact, infection or systemic disease.
Shoulder joint instability:	Prevalence: up to 1.7% In young active people, up to 2%; after a traumatic dislocation, the risk of repeated dislocations is up to 70%. Incidence of dislocations: 23.9 per 100,000 persons/year	Most often in men aged 15–29. Often associated with physical activity and sports. Traumatic dislocation, capsule and ligaments stretching, connective tissue dysplasia, sports overload.
Frozen shoulder syndrome (adhesive capsulitis:	Prevalence: up to 2–5% of the population. In the structure of shoulder diseases is 10–15%	More often in women aged 40–60 years. Often associated with diabetes (up to 20%) and thyroid disease. The joint capsule inflammation and fibrosis; primary (idiopathic) or secondary (after injury/surgery, with endocrinopathies).

Rotator Cuff Injury (Tear/Tendinopathy)	→ Overall prevalence: 20.7–m23.5% → Symptomatic ruptures: about 10% Asympto- matic ruptures: about 16.5%. Up to 20% of people over 60 years old, in athletes up to 30–40%; in 80-year-olds, up to 50%. In shoulder diseases structure it has the 2nd place and makes up to 20–30%	Age-related degeneration, repetitive over-head stress, trauma; most commonly the m. supraspinatus is affected. Prevalence increases with age, especially after age 50. More often in men and in physical work
Osteophytes of the shoulder joint	In 30 - 50% of patients over 50 years; often found in acromioclavicular joint arthrosis	Bone growths in arthrosis, overload; aggravate impingement
Labral tear	SLAP injuries in throwing and weightlifting athletes account for up to 20 - 25% of sports shoulder injuries	Trauma (falling on outstretched arm), repetitive throwing motions, shoulder instability
Joints' labrum degeneration	MRI finding in people over 40 years, is asymptomatic in up to 30%	Age-related degenerative changes, chronic instability, overload

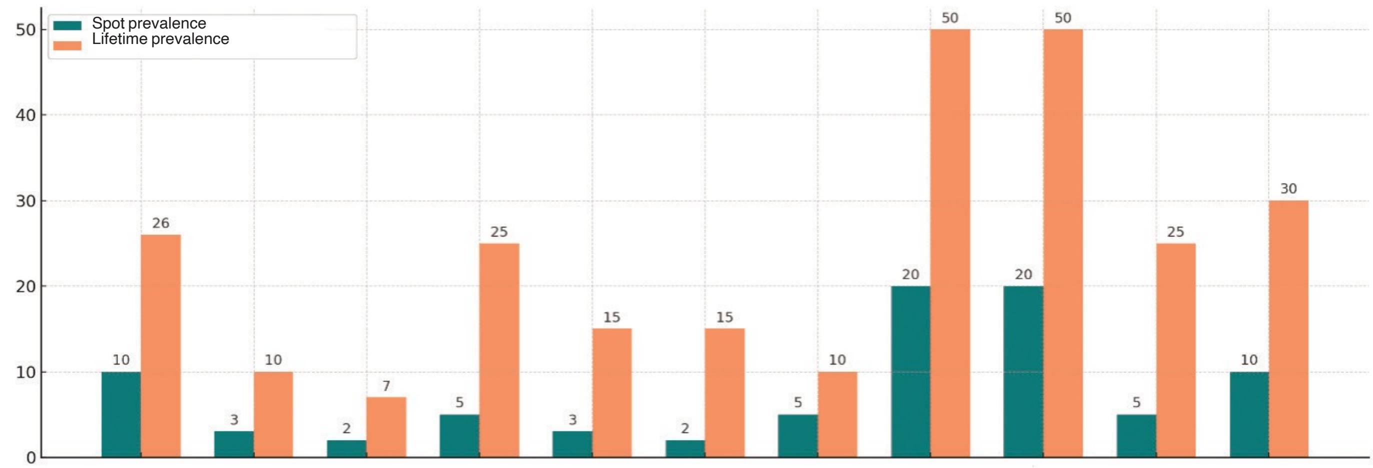
Thus, the aetiology of shoulder joint diseases and dysfunctions is quite unclear. There is no clear merge between individual conditions. Even in cases where anatomical abnormalities (torn labrum, rotator cuff injury) are recorded immediately after the injury it cannot be reliably stated that these changes arose precisely during the injury and were not present before it, i.e. are truly traumatic and not degenerative.

Rotator cuff tendon tear

Labral or rotator cuff tendon tear registration immediately after injury does not prove that injury occurred at that time: such changes often precede the event and are discovered incidentally. Causal inference usually requires pre-injury imaging or documentation, clear acute signs (bone/muscle swelling, fresh haematoma, atrophy absence in case of a large tear), consistency with the injury mechanism and clinical picture. This is directly indicated in reviews devoted to complex differentiation and high frequency of asymptomatic findings. (1 – 10)

Despite the fact that various shoulder joint pathologies are very common, in most cases they are not vitally constant but have a recurring or episodic nature. A person may suffer from them throughout the certain period of life but have no symptoms at the time of examination. This is evidenced by the data of the point and lifelong prevalence of shoulder joint diseases*. (Figure 1)

Figure 1: Comparison of spots' and lifetimes' prevalence of shoulder joint diseases



The comparison of shoulder joint diseases point and lifetime prevalence (data are averaged from different sources (AAOS, UpToDate, JSES, Arthroscopy, epidemiological studies). Exact data may vary depending on population and the method of calculation.

* Point prevalence is the proportion of people who have a disease at exact point in time (e.g., "on the date of the survey"). Lifetime prevalence is the proportion of people who ever had a disease during their lifetime.

By analysing the diagram we can stress that rotator cuff tears and labral degeneration are cumulative pathologies. Almost half of people encounter them at least once in their lives but not everyone experiences permanent symptoms. This indicates a high proportion of subclinical, i.e. asymptomatic changes recorded on MRI in older people.

Adhesive capsulitis, impingement syndrome, rotator cuff injury and bursitis are transient conditions. Their lifetime prevalence is approximately 2 times higher than the point prevalence. This means that many patients experience a limited episode of the disease which then passes.

Exact shoulder pain (10% local, 26% lifelong) is one of the most often complaints of the musculoskeletal system. It is comparable in prevalence with pain in the lower back or neck.

The orthopaedic model orientation on anatomical factor affecting the deep structures of examined area (the shoulder joint capsule, the joints' labrum, the tendons of the rotator cuff muscles) creates the basics for a rather narrow corridor of actions of specialist providing assistance to the patient. This is clearly seen on example of certain nosological forms diagnostics and treatment.

Impingement syndrome

Thus, for example, impingement syndrome (subacromial pain syndrome) is a pinching of *supraspinatus* tendons, the long head of *biceps brachii* and subacromial bursa. Patients complain of pain along the frontal, lateral or upper surface of the shoulder radiating along its lateral surface. The pain is dull, aching, acute when exerting weight (lifting objects with an outstretched arm), lying on the painful side, night pain, and limitation of activity.

Table 2: Main provocative tests for impingement syndrome.

Test	What does it evaluate?	Technique	Positive result
1. Hawkins-Kennedy test	*supraspinatus* or subacromial bursa compression	The shoulder is flexed at 90°, the elbow is flexed, the doctor performs the shoulder internal rotation	Pain in the anterolateral shoulder
2. Neer test	Rotator cuff tendon compression	The doctor passively raises the straightened arm forward and up to the end (maximal flexion)	Pain in the upper shoulder
3. Painful Arc Test	General compression in subacromial space	The patient actively moves arm to the side (abduction)	Pain between 60° and 120°
4. Jobe Test (Empty Can Test)	*supraspinatus* function	Arm in 90° abduction and 30° horizontal adduction, shoulder internally rotated (fingers down), the doctor presses from above	Pain or weakness
5. Drop Arm Test	Rotator cuff tear (often supraspinatus)	The patient's arm is raised to 90°, then we ask him to lower his arm slowly.	Unable to hold the hand, it falls quickly
6. External Rotation Resistance Test	Internal rotators, possible impingement	Arms are pressed to the body, elbows are bent at 90°, the patient resists external rotation	Pain or weakness in the arm

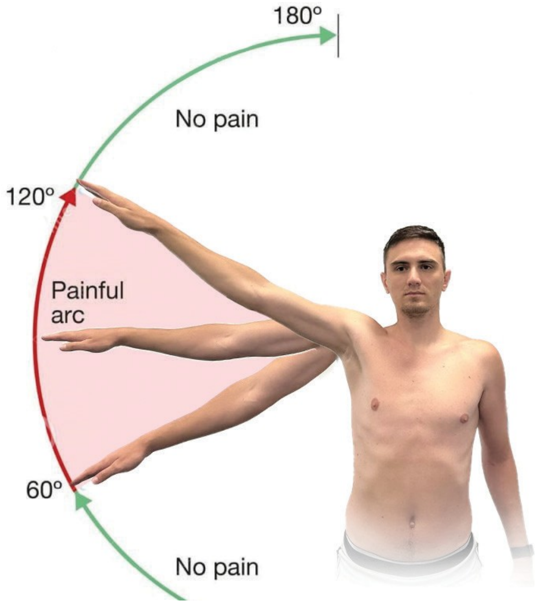


Figure 2: The range of motion in the shoulder joint (abduction) at which the patient experiences pain in impingement syndrome (painful arc of Dawborn).

Figure 3: Neer test, pain in the shoulder area during passive lifting of the patient's straightened arm forward (in the sagittal plane) at an angle of more than 90°, with internal shoulder rotation (sensitivity is 72–79%, specificity equals to 53–60%).

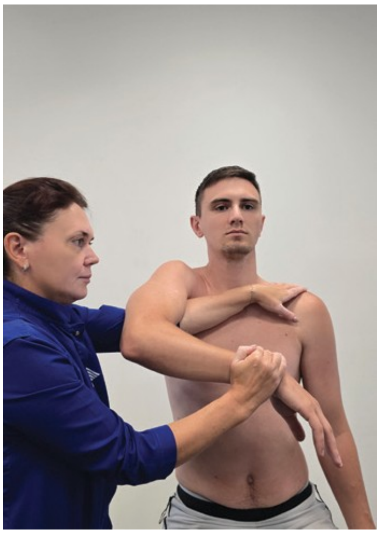


Figure 4: Hawkins–Kennedy Test, pain in the shoulder area with shoulder passive flexion to 90°, forearm flexion to 90° and quick internal shoulder rotation (sensitivity 79%, specificity 59%

A series of tests are provided to verify the diagnosis (Table 2, Figure 2–4).

Usually the diagnosis is made in case of 2–3 positive tests combination. The Neer test suggests lidocaine 1% (10 ml) injection into the subacromial space (diagnostic test), pain relief confirms impingement.

Impingement tests (Neer, Hawkins–Kennedy) have moderate sensitivity but low specificity. They are useful for diagnosis excluding but cannot con- firm it. (11, 12)

Shoulder joint X-ray, ultrasound investigation and MRI are used for visualisation. X-ray can show acromial osteophytes, acromion type (hook-shaped) and calcifications. Shoulder joint ultrasound investigation clearly shows tendons thickening, bursitis and subacromial space. MRI is considered to be the gold standard, it determines oedema, tendinopathy, cuff tears and subacromial space narrowing. (13 – 16)

Impingement syndrome treatment involves both conservative (the main approach in the early stages) and surgical (if conservative treatment is ineffective for >6 months). Conservative treatment involves rest and avoidance of provoking movements, NSAIDs for pain relieve and inflammation cessation and physiotherapy (ultrasound, laser, magnetic therapy, exercise therapy to strengthen both the rotator cuff and scapular stabilisers, back capsule stretching). The average duration of conservative treatment equals to 6 -m12 weeks, the first improvements often occur after 3 - 4 weeks and an expressive effect might be registered by 2 - 3 months.

Conservative treatment (3 - 6 months) is effective in 70–90% of patients; a longer course may be required in elder patients. (17) Conservative therapy gives a satisfactory result in 60% of cases after 2 years; from 3 till 6 months of multimodal treatment might be required in case of structural damage absence. (18) From 6 till 12 weeks of conservative treatment is used for small rotator cuff tears before referral to a surgeon. (19) Physical therapy was shown to be effective in pain elimination, both mobility and shoulder function improvement during the 6 [12 weeks. (20) Certain authors recommend physical therapy use for 6 - 12 weeks with rotator cuff strengthening, scapula stabilisation and stretching. (21) Other author indicates that shoulder joint recovery can take from several weeks till 6 months and in severe cases, up to a year. (22)

If pain persists for more than 3 - 6 months with full conservative therapy, injections (corticosteroids, PRP therapy*) or surgical intervention are considered.

Impingement syndrome surgical treatment involves arthroscopic subacromial decompression (osteophytes removal and the acromion correction), reconstruction of tendons in case of ruptures. Immobilisation occurs 1 - 2 weeks after surgery, exercise therapy and restoration of function after 2 -3 months, full restoration of load and sports after 4 - 6 months (sometimes up to 9 months).

Thus, as can be seen from the data presented, in case of shoulder impingement syndrome it is impossible to recommend this disease reliable diagnostic criteria and both the treatment and recovery periods are more prolonged.

Rotator cuff injury

The rotator cuff damage occupies the second place in shoulder joint diseases structure (20–30%). (23) This is trauma or degenerative damage of one or more tendons of the muscles that surround the shoulder joint, which ensure its stability and rotational movements**.

The causes of the disease are considered to be acute injuries (falling on an outstretched arm or elbow, sudden lifting of a heavy object, shoulder dislocation) and chronic degenerative changes

* PRP-therapy (Platelet-Rich Plasma therapy, autologous platelet rich plasma therapy) is a method of treatment in which the patient's own plasma with an increased platelets concentration is injected into the damaged area. Platelets secrete growth factors that stimulate angiogenesis, collagen synthesis, cartilage and tendon regeneration, reduced inflammation and stem cell activation. The idea is to 'initiate' the body's natural regeneration processes.

** The rotator cuff is a complex of four muscles (*supraspinatus*, *infraspinatus*, *teres minor* and *subscapularis*) and their tendons that encircle the head of the humerus like a 'cuff' and merge with the joint capsule.

(age-related tendons thinning, especially *supraspinatus*), repetitive overhead loads (sports and/or work), impingement syndrome. (24, 25) Metabolic diseases, especially diabetes mellitus, increase the risk of disease; rotator cuff damage is often associated with hyperlipidemia and hypertension. (26) Familial cases of rotator cuff damage are observed, and body mass index increase, smoking and other modifiable factors correlate with the risk of rupture. (27)

Symptoms of the disease, i.e. pain (especially when raising the arm to the side and above the head, at night when lying on the shoulder), weakness when shoulder abducting and rotating, clicks, crunching when moving, motion range limitation, arm function acute decrease with a complete rupture, cannot be considered as strictly specific for shoulder cuff damage. (28 – 31) However, many injuries can be asymptomatic, especially partial ruptures, up to 2/3 of cases without clinic manifestations. Symptoms are important, but not very specific, they cannot be trusted alone.

Rotator cuff damage diagnosis is based on a comprehensive analysis of clinical picture (symptoms), special tests (32, 33) and clinical visualisation (ultrasound, MRI) (Table 3).

Table 3: Clinical tests to evaluate the rotator cuff.

Test	What does it evaluate?	Technique	Positive result
Painful arc test	Muscle *supraspinatus*	The patient actively moves the arm to the side (abduction)	Pain between 60° and 120° angles
Empty Can (Jobe's test)	Muscle *supraspinatus*	The patient raises his arms forward at 90° and to the sides at 30°, thumbs down ("pouring out the can"). The examiner presses from the top	Pain and/or weakness with resistance
Full Can test	Muscle *supraspinatus*	Same as in Empty Can test but thumbs up	Less pain than in Empty Can test; weakness indicates damage
External Rotation Lag Sign	Muscle *infraspinatus* and muscle *teres minor*	The arm is in external rotation and 20° abduction, elbow is flexed. The examiner releases , the patient must hold	Hand returns inward (inability to hold)
Lift-off test	Muscle *subscapularis*	The patient places his hand behind his back, his hand back on his lower back, and tries to move his hand back	Inability to move the hand away from the back or pain
Belly-press test	Muscle *subscapularis*	The patient presses his palm to his stomach, trying to keep his elbow forward	Elbow goes back or weakness occurs
Drop Arm test	Muscle supraspinatus	The patient slowly lowers the arm from 90° abduction	The hand suddenly 'drops' or pain appears

However, special tests are also not a reliable source for diagnosis as they have different degrees of sensitivity and specificity* (Table 4).

Table 4: Diagnostic tests sensitivity and specificity in case of rotator cuff injury.

Test	Sensitivity	Technique	Positive result
Painful arc	~97% (very high)	<10% (very low)	If the test is negative → there is almost no rupture (useful for exclusion)
Empty Can (Jobe's test)	80–90% (high)	60–80% (moderate)	Balance: helps both to exclude and to confirm, but is not absolute
Full Can test	70–80%	70–80%	Alternative to Empty Can test; less pain due to subacromial space less compression
External Rotation Lag Sign	~30% (low)	~98% (very high)	If the test is positive → a rupture is almost certain (useful for confirmation)
Lift-off test	Low	>90% (very high)	Positive → specific for *subscapularis* rupture
Belly-press test	40–60%	85–95%	More convenient comparing with Lift-off test; positive → *subscapularis* lesion
Drop Arm test	20–35% (low)	80–98% (very high)	Positive → almost certain *supraspinatus* rupture; if negative, of little importance

Therefore, if the sensitive test is negative, then the disease is almost certainly absent ('rule out'), and if the specific test is positive, then the disease is almost certainly present ('rule in').

According to meta-analyses data, MR arthrography is the most sensitive and specific in diagnosing both complete and partial rotator cuff tears, while MRI (the 'gold standard' in clinic) and ultrasound demonstrate comparable accuracy (complete tears of m. supraspinatus: ultrasound ≈ 94/94%, MRI ≈ 94/89%; partial - ultrasound ≈ 89/89%, MRI ≈ 85/87%). For primary care and dynamic tests, ultrasound remains a justified choice, and for preoperative planning and broad differential diagnostics, MRI/MRA is preferable. (34, 38)

Arthroscopy is the most accurate method for rotator cuff injuries diagnosing, it allows both diagnosis and treatment (suturing the rupture, re- moving osteophytes, etc.). It provides direct visualisation of the tendons, labrum, capsule, and is considered to be the 'gold standard' with which ultrasound, MRI, and MR arthrography are compared. However, arthroscopy is not used for diagnostic purposes only as it is too invasive. Ultrasound and/or MRI are used for primary diagnostics because they are safe, noninvasive, highly sensitive and specific (for full-thickness ruptures ≈ 90–95%). Arthroscopy is used as a stage of surgical treat- ment but not just diagnostics.

* Sensitivity is the ability of a test to detect a disease when it is actually present. Specificity is the ability of a test to show a negative result when the disease is not present

What about the treatment? It can be conservative (more often with partial ruptures and tendinopathy) and surgical (in cases of complete rupture, severe weakness, conservative treatment inefficacy for 3 - 6 months).

Rest is recommended in acute phase (without complete immobilisation), NSAIDs (ibuprofen, naproxen), physiotherapy (ultrasound, laser, magnetic therapy), exercise therapy to strengthen the scapula and shoulder stabilisers, injections (corticosteroids, PRP, prolotherapy*) are used. Surgical treatment, arthroscopic or open tendon reconstruction, in severe cases installation of an augment or shoulder endoprosthesis.

The recovery occurs averagely after 3 - 6 months in case of the rotator cuff partial damage and proper rehabilitation, in case of a complete rupture and surgery, 9 - 12 months after. The longer treatment is delayed in case of a complete rupture, the higher the risk of muscle atrophy and irreversible loss of function. Thus, the diagnosis of this disease presents certain difficulties either due to the non-strict specificity of the tests performed, or due to the avail- ability of the instrumental methods used. Treatment and recovery periods are extended in time.

Adhesive capsulitis

A similar situation occurs with adhesive capsulitis (frozen shoulder syndrome) which is less common than impingement and rotator cuff pathologies but also creates a problem for both patients and specialists. According to large series of observations (23) adhesive capsulitis accounts for approximately 10 - 15% of all cases of pain and limitations in the shoulder, i.e., it accounts for approximately 1 in 10 cases in shoulder diseases structure. It is especially important in patients with endocrinopathies and diabetes (it occurs in these patients 2 to 4 times more often).

Adhesive capsulitis is an inflammation, fibrosis (scarring) and adhesion (fusion) of the shoulder joint capsule with the development of movement limitations (especially abduction and rotation) accompanied by severe pain, especially at night, The exact causes of adhesive capsulitis are not yet established. Risk factors include age 40 - 60 years, female gender, endocrine disorders (diabetes, thyroid disease), previous injuries and surgeries.

This syndrome is characterised by staging (Table 5) and the identification of primary (idiopathic) and secondary variants, after trauma, surgery and prolonged immobilisation. (42, 43, 44)

Table 5: Stages of adhesive capsulitis.

Stages of adhesive capsulitis development	Characteristic details
Initial phase - 'freezing' (inflammatory)	Progressive shoulder pain increases with movement and at night. As the pain increases, so does the mobility goes to limited. This phase usually lasts from 2 to 9 months.
Frozen' phase - fibrosis	Joint mobility is acutely limited, pain decreases, the arm is poorly abducted, it is difficult to dress and wash one's hair. Lasts for 4 - 12 months.
Thawing - 'defrosting' phase (recovery)	Gradual improvement of mobility, pain reduction. Lasts for 12–24 months.

* Prolotherapy (from Engl.- proliferation therapy, also called as regenerative injection therapy) is a method of chronic pain treating and instability of joints, ligaments and tendons using irritating solutions repeated injections into their attachment sites (most often a hypertonic glucose 10–25% solution, less often phenol, glycerin, morphine, etc.). This causes local controlled inflammation which stimulates fibroblasts proliferation (growth), increased collagen synthesis and ligaments/tendons 'remodelling'. As a result, the tissues are strengthened and joint instability and pain are reduced. (39 - 41)

Ross A. Hauser MD, and Daniel R Steilen-Matthias MD, University of Pennsylvania, in their article note '.... *frozen shoulder is an interesting problem. We're not entirely sure what causes it. It seems to be more common in people with diabetes. There also appears to be some kind of autoimmune reaction (inflammation) in the shoulder*'. (76)

Frozen shoulder diagnosis is based on clinical findings (limitation of range of motion, especially external rotation, diagnostic tests similar to those in cases of other shoulder diseases). Imaging (ultrasound, MRI) is not obvious but helps to exclude other causes, although '... *the need for MRI and its diagnostic value are questionable*'. (45)

Numerous methods were proposed for adhesive capsulitis treatment which are used at different stages of the disease and follow different goals (Table 6).

Table 6: Comparative characteristics of adhesive capsulitis methods of treatment.

Treatment	Aim	Results
Physiotherapy (the first line of treat- ment): • myofascial release • massage • kinesitherapy • laser therapy • shortwave diathermy • ultrasound therapy • hot compresses	Physiotherapy is divided into 'pain-relieving', 'mobilisation' and 'strengthening'	Pain reduction by 60%, increase in range of motion by 2 times
Hydrodilation - intra-articular injec- tions of saline (or distilled water) + anesthetic + corticosteroid	Adhesions dissections and increasing range of motion	Contradictory
PRP-therapy is the platelet-rich plasma injection into the joint. It is prescribed in the second phase of the disease.	Chronic inflammation reduce Stimulation of connective tissue remodelling	Pain reduction by 60%, increase in range of motion by 2 times
Prolotherapy, administration of hypertonic glucose solution (15 - 25%) with lidocaine, vitamin B12. Provided 2 - 3 times a week for 4 - 6 weeks	Local inflammation activation, collagen production increase, fibroblasts attraction, strengthening of damaged tissue	On average, the increase in range of motion over 3 - 6 months is 15 - 35%. Reduction in pain at rest
Shoulder joint capsule ultrasonic hydrodistension (Hydrodilatation, Distension Arthrography). A mixture is injected into the shoul- der joint cavity (usually through a posterior approach) under ultra- sound control: • physiological solution (basic volume 10–40 ml), • local anesthetic (lidocaine, bupiv- acaine), • glucocorticosteroid (triamcinolone, methylprednisolone)	Mechanical stretching of capsule - fibrous adhesions rupture; joint cavity volume increase - improvement of mobility; steroid component - inflammation and pain reduction; anaesthetic - immediately reduces pain, facilitating kinesitherapy procedures	Quick Range of Motion increase and pain reduction, especially in early stages of adhesive capsulitis. The effect lasts up to 2 years in most patients. Patients with diabetes mellitus experience more frequent relapses and less expressed recovery.
Shock wave therapy. Performed 2 times a week for 6 weeks	Elimination of adhesion and fibrosis	Pain reduction by 40–60%. Range of motion restoration by 50–70%
NSAIDs (non-steroidal anti-inflamma- tory drugs)	Inflammation reduction	Temporary relief. Long-term loss of function, chronicity of the process, cartilage degeneration acceleration

Intra-articular injection of corticosteroids (cortisone), lidocaine. Performed 3 times a week for 4 weeks.	Inflammation reduction	Temporary relief. The effect does not last more than 6 weeks
Arthroscopic capsular release	Elimination of adhesion	' ... had no positive effect on late mid-term clinical and functional outcomes'.Persistent limitation of motion for 2 years after surgery in patients with diabetes. No results after 12 months
Oral steroids. Catastrophic complication formation in the form of femoral head avascular necrosis should be feared even with a short course of oral steroids.	Inflammation reduction	Pain reduction, increased range of motion with short-term use (6 weeks) in the first stage

Most researchers agree that condition improves within 12 - 18 months with adhesive capsulitis conservative treatment but the duration of the disease can be up to 3 years even with treatment. (23, 46, 47) In case of surgical treatment active rehabilitation begins after 1 - 2 weeks, and the full range of active movements is usually restored within 6 - 12 weeks.

Analysing the data presented devoted to most common (according to the diagnoses) shoulder joint diseases it can be noted that for the orthopaedic approach, which is based on the anatomical model, shoulder joint dysfunctions are a rather complex problem in diagnosis and treatment.

Fascial model

Since the end of the last century, orthopaedics and physiotherapy started to to notice that shoulder mobility limitations are associated not only with the capsule and tendons but also with fascial layers. Since the 2000s, fascia started to be considered as biomechanically active structures that affect kinematics and actively participate in a biotensegrity system where forces are transmitted not only through bones and muscles but also through fascial tension. Fascia is a kind of biological spring that saves metabolic energy, providing movement, stability and compensation. Due to collagen and elastin fibres presence that stretch and then return to their original position fascia accumulate and release the energy of elastic deformation*.

The energy of elastic deformation works in the fascial structures of the shoulder girdle which ensures the movements adequacy, stability and compensation (Table 7).

While raising the arm, throwing a ball or pulling yourself up, the fascia of the shoulder stretches (storing energy) and then returns to its original position helping the muscles to return the arm or stabilise the joint. For example, when you quickly pull your arm back (before throwing), the fascia of the chest and shoulder area stretch, storing energy. When you throw, some of this energy is released, facilitating the movement. This is how the cycle of movement and recovery is ensured.

The fascia act as tension cables, stabilising the joint without expending metabolic energy (stabilisation without active muscle tension). Of particular importance are the clavicular-sternal connection through both the fascia and ligaments (stabilises when arm is rotating), the connection with the spine through the *trapezius* and *rhomboid* fascia (involved in loads transfer when lifting weights, carrying a bag, etc.).

The shoulder girdle fasciae form single myofascial chains. Sliding limitation in one zone can cause the entire limb dysfunction. It is an active organ of sense and regulation of movements involved in pain and limitation of mobility which is fundamentally important, for example, in case of adhesive

* he energy of elastic deformation is the potential energy that accumulates in a body during its elastic (i.e. reversible) deformation. When external forces stop to act this energy is released and the body returns to its original state.

capsulitis. (48, 49) The shoulder fasciae (i.e. deltoid, trapezius, thoracic) can participate in the pathogenesis of both impingement and adhesive capsulitis as the result of impaired sliding. (51)

Table 7: Involved fascia

Fascia	Location	Function
Fascia m. trapezius	Covers the back, scapulae	Connects the skull, neck, spine and scapula. When raising the arm or turning the head, it is stretched, stabilizes the scapula, participates in force transfer to the spine
Fascia m. pectoral	Covers the *pectoralis major* muscle	Transfers force from the chest to the shoulder joint and further to the arm. When doing bench presses, pull-ups, push-ups, it accumulates and returns elastic energy
Fascia m. deltoid fascia	Covers the *deltoid* muscle	Participates in shoulder joint stabiliza- tion and control of movement. Works as an elastic "bridge" between the thoracic, trapezius and shoulder regions
Axillary fascia	Connects the chest and shoulder area	Provides mobility and stability when moving the arm up, back, forward. Vascular-nerve bundles pass through it, so its tension can cause functional disorders

Fascia is considered now as a key element in pathogenesis of adhesive capsulitis (both capsule and fascia fibrosis), impingement (the subacromial-subdeltoid fascia thickening), myofascial pain syndromes, postoperative and post-traumatic contractures, (48 – 51)

The concept of fascial dysfunctions forms the basis of methods used to treat the shoulder joint diseases.

The FM method (Fascial Manipulation by Luigi Stecco) is a method of manual therapy where the are- as of fascial thickening ('Centres of Coordination') are targeted improving the sliding of fascial layers and the balance of myofascial chains.

It is widely used for chronic shoulder pain. The technique is aimed at fascial tissue restructuring through manual action. (53, 54)

With the US-FHR method (Ultrasound-Guided Fascia HydroRelease): saline is injected into fascial seals (for example, coracohumeral ligament) under the ultrasound control, adhesions are thus mechanically 'broken', tissue sliding is improved, pain is reduced, and mobility is increased. (52)

Considered a minimally invasive procedure it provides a quick effect but requires ultrasound skills. Luigi Stecco's method, Fascial Manipulation (FM), is based on anatomy and biomechanics, requires training and precise search for fascial points.

Unlike these methods, the FDM (fascial distortion model) proposed by Stephen Typaldos has a number of advantages. Typaldos considered fascial distortions to be the main source of pain impulses, distortions (damage to connective tissue structures), which elimination leads to immediate pain relief. This is an important link in fascial medicine, especially, for shoulder pain and dysfunction (adhesive capsulitis, impingement, tendinopathies) explanation. According to the model he proposed, fascial distortions can be diagnosed according to patient complaints, gestures and clinical examination. (55 – 58) Thus, this model offers a relatively simple system of diagnosis, correction and pain and dysfunction rapid elimination. There are 6 types of connective tissue distortions (Table 8).

Table 8: Types of fascial distortions, their diagnosis and treatment.

Distortions	The essence of connective tissue distortion	Diagnostic manipulation	Treatment
TB Triggerband distortion (trigger cord)	Structural damage of longitudinal fascial tissues (fascia, ligaments, tendons) which fibres are subject of deformation, twisted, torn, detached, crumpled (feeling of 'tethers' or 'tight string'); partial rupture of fascial fibres separation under the influence of excessive angular forces against the background of longitudinal fascia stretching. Occurs acutely or gradually.	• Sliding fingers move- ments from the middle of the back towards the mastoid process; • Sliding fingers move- ments along the shoulder frontal surface; • Sliding fingers move- ments along the back of the shoulder; • Sliding fingers move- ments from the shoulder towards the mastoid process	The intense deep pressure and rubbing along the painful cord. The aim is to 'tear' the pathologically stretched fascial fibrse and to restore sliding.
CD Continuum distortion	A disturbance in the transition between different types of tissue (e.g. bone-ligament, bone-tendon, tendon-muscle, capsule-bone, etc.) that results in pain, tenderness or decreased function. It is a disturbance in the continuum between hard and soft tissues where the 'connection' of tissues lost its coherence (enthesopathy, point pain). Is the main cause of calcific tendinitis	Finger pointing on the pain location	Point compression pressure directly into the enthesis area. The aim is to 'restructure' the transition zone.
HTP Herniated trigger point	Soft tissue prolapse through the overlying fascia (perceived as a 'ball' or 'knot'). A common cause of acute shoulder pain and limited movement (especially abduction) in the shoulder joint. Mechanism of formation: excessive pressure in the 'inside out' direction as a result of muscle tension when lifting weights, coughing, sneezing.	Pressure with fingers in the supraclavicular fossa	Firm pressure with fingers with or elbow directly on the bulge. The aim is to 'reset' the fascial hernia and restore tissue integrity.
FD Folding distortion	Damage, incorrect folding/ bending of fascial sheets (often with injuries with hyperflexion/ extension) occurring in folded fascia (joint capsule, intermuscular septa: FD, refolding distortion - the effect of damaging forces during the folded fascia compression, FD, unfolding distortion the effect of angular forces during the folded fascia stretching. Occurs acutely. Causes deep pain in the joint.	• Rubbing with a finger across the humeral neck lateral surface; • hand placed on shoulder	Joint mobilisation and traction, manual techniques of flexion and extension with pressure on the folds. The aim is to 'straighten' and to restore the fascial sheets normal direction.

Table 8: concluded.

Distortions	The essence of connective tissue distortion	Diagnostic manipulation	Treatment
CyD Cylinder distortion	Fascial structures spatial orientation disruption (sensation of deep, 'drilling' pain); influence of angular forces causing spiral fascia twisting (superficial, proper, fibrous capsules of muscles) at the moment of its stretching or compression. Fascia loses the ability to straighten or compress, becomes less elastic. Sometimes this leads to a sharp loss of range of motion.	Upper part of the arm squeeze	Twisting and rotational manual techniques, often a combination of stretching and spiral compression. The aim is to 'realign' the fascia cylindrical structures and to reduce the chaotic tension.
TF Tectonic fixation	Fascial layers 'jamming' leading to limited mobility (very typical for adhesive capsulitis); loss of fascia sliding ability as a result of changes in synovial fluid amount and rheological properties. The most common cause is the joint's prolonged immobilisation. With shoulder TF, the entire joint capsule or part of it is fixed to the underlying bone). In severe cases, abduction, external and internal rotation are limited or almost completely lost. In less severe cases, patients complain of a feeling of /lack of lubrication' or 'movement of hard gears'.	• Inability to flex the shoulder without additional forward lean; • When lying on the stomach in a swimmer's position, the patient is unable to press the shoulder to the table.	Strong mobilisation techniques with a large volume of movement, 'jerking' or progressive manual impact on the joint to 'unlock' the fascial sheets. The aim is to restore mobility and sliding.

FDM therapy is one of the most intense and painful manual techniques. Patients often experience severe pain during treatment, but after-wards, significant relief. A distinctive peculiarity of this technique is diagnostics based on the patient's gestures and pain nature: each type of distortion has its own 'hints'. The clinical effect is often immediate but repeated sessions may be required to consolidate the result.

The fascial model used for patients with shoulder joint diseases has a number of advantages over the orthopaedic model (relatively simple and quick diagnostics, safe and relatively simple treatment methods, achieving the therapeutic goal in the shortest possible time), however, the long-term treatment results were not sufficiently studied.

There are no published randomised controlled trials (RCT) till now with a follow-up of ≥6–12 months on FDM for shoulder diseases. The best studies, Fink 2012 (FS) and Moradi 2023 (impingement), show a good short-term effect (rapid dynamics of ROM/pain) but the effect long-term sustainability was not confirmed. (59, 60)

According to FM (Stecco), the situation is similar: positive short-term dynamics (up to 1 month) but the authors themselves emphasise the lack of long-term observations (Follow-up) 12–24 months. (61)

As for US-FHR, pilot data, the authors explicitly request RCT with long-term follow-up. (62)

Muscular-neurological or reflex model

Assuming the connective tissue structures and fascia leading role in musculoskeletal system diseases and dysfunctions, Typaldos claimed, '*Muscles don't matter, think Fascia*'. However, it must be recognised that muscles, although they depend largely on ligaments, tendons, fascia, play a key role in stabilisation and movement. Being organised into subsystems of muscle-fascial chains (MFC) they ensure the functioning of a single system of biotensegrity. (63 – 68) The presence of four MFCs can be determined at the level of the shoulder girdle and upper limb (Table 9).

Table 9: Elements providing structural and reflex connections within the shoulder girdle and upper limb MFC.

Deep ventral MFC of the hand	Superficial ventral MFC of the hand	Deep dorsal MFC of the hand	Superficial dorsa MFC of the hand
• *Pectoralis minor* • Clavisternal fascia • *Biceps brachii* • Periosteum of m. radius • Radial collateral ligament • Thenar muscles • Acromioclavicular joint • Coracoid process of the scapula • Capsule of the shoulder joint, its frontal surface • Tendon of the long head of *biceps brachii* • Radial collateral ligaments of the elbow • Periosteum of the radius • Radial collateral ligament of the wrist	• *Pectoralis major* • *Latissimus dorsi* • Medial inter-muscular septum • Flexor muscles of the wrist & fingers • Retinaculum of the flexor muscles (carpal tunnel and Guyon's tunnel) • Palmar aponeurosis • Ligaments of the dome of the pleura on its side • Periosteum of the clavicle • Acromioclavicular joint • Frontal group of elbow joint medial collateral ligaments • Common retinaculum of the tendons of wrist and fingers flexor muscles	• *Rhomboid* muscles • *Levator* scapulae • Rotator muscles of the shoulder joint • *Triceps brachii* • Periosteum of the ulna • Ulnar collateral ligament of the wrist • Hypothenar muscles • Acromioclavicular joint • Spine (crest) of the scapula • Shoulder joint capsule, its back surface • Tendon of the shoulder triceps • Olecranon process	• *Trapezius* • *Deltoid* muscle • Lateral inter-muscular septum • Wrist and fingers extensor muscles • Ligaments of the dome of the pleura on its side • Acromioclavicular joint • Elbow joint lateral collateral ligaments • Wrist joint dorsal surface ligaments

According to the laws of MFC formation, any of the chain elements (muscular or connective tissue) dysfunction results in the entire chain dysfunction with the compensatory reactions development rom the muscles of other chains often accompanied by their shortening. This could leads to limited movement and an increasing pain reaction at the shoulder joint level.

The human body system is represented by a variety of structural and reflex functional connections with the help of which the resistance to changing environmental conditions is ensured. In case of shoulder joint dysfunctions, it is important to consider the structural connections with the chest, cervical and pelvic regions. For example, both fascia or tendon restriction leads to load redistribution throughout the system inducing the secondary dysfunctions such as impingement or compensatory ROM limitation (Figure 5). The presence of fascial conflict zones explains the muscles dysfunction at the level of the shoulder girdle and upper limb in cases where individual muscles dysfunction is not detected.

A load-resistant system implies stability which is ensured primarily by muscle tone. Considering muscle dysfunction as the main source of instability, it is necessary to take into account that at the level of the shoulder girdle there are 5 joints (3 true (*articulationes verae*) and 2 false (*articula- tiones spuriae*)) which stability depends on muscle structures (Table 10).

The functional or 'false' joints often include the subdeltoid (subacromial) joint, the functional space between the deltoid muscle and the shoulder joint capsule/subacromial bursa. Thus, the shoulder joint is considered in unity with other joints of the shoulder girdle which stability depends on normoreflexivity of muscles that provide them.

The above mentioned muscles numerous and different reflectory connections are of particular importance in case of dysfunction (Table 11).

Table 10: Shoulder girdle joints and muscles that stabilise them

The acromioclavicular joint:	*deltoideus*
The sternoclavicular joint:	*subclavius*
The scapulohumeral (shoulder) joint:	*supraspinatus* *infraspinatus* *subscapularis* *teres minor* *latissimus dorsi* *deltoideus* *pectoralis major* *biceps brachii* *triceps brachii*, long head *teres major* *coracobrachialis*
The claviculothoracic joint:	*subclavius* *trapezius* (upper part) *pectoralis major* (clavicular part) *sternocleidomastoideus*
The scapulothoracic joint:	*rhomboideus major* *rhomboideus minor* *serratus anterior* *pectoralis minor* *levator scapulae* *trapezius* (medial and lower parts)

Figure 5: Structural connections of arm MFC with trunk and lower limb MFC

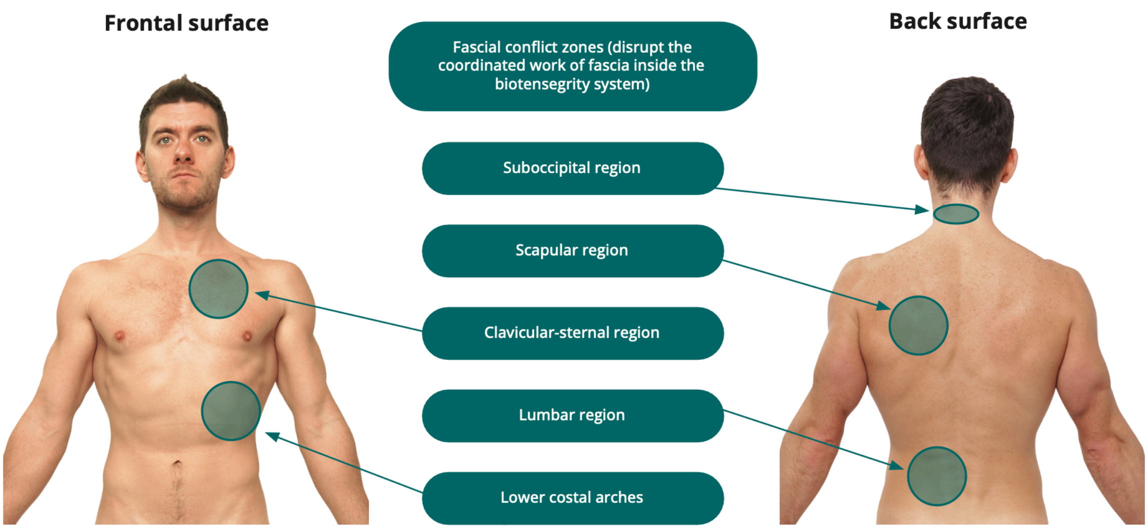


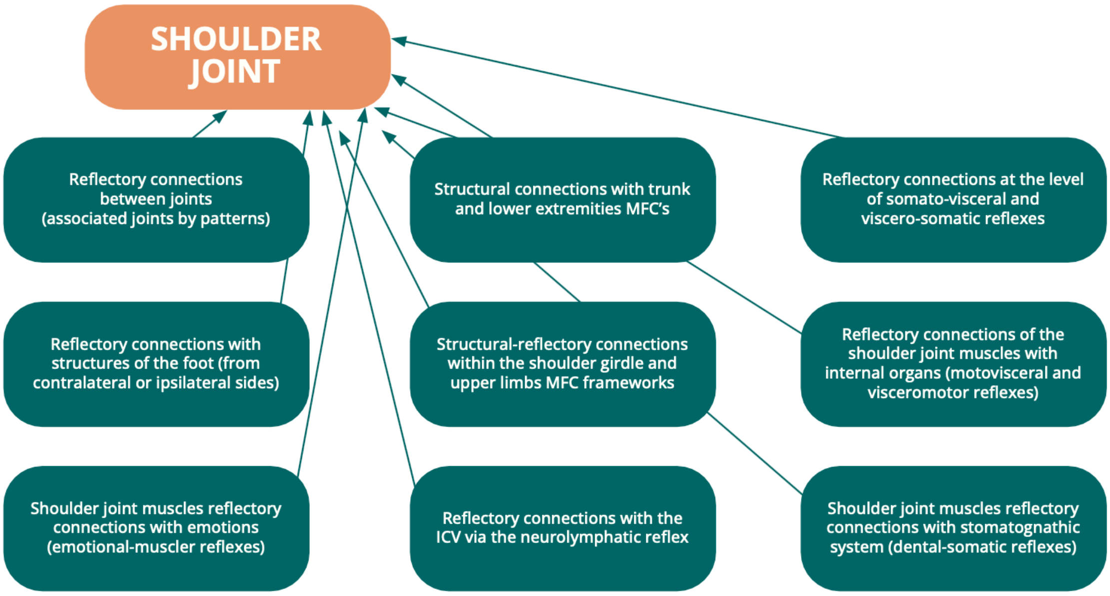
Table 11: Reflectory connections of the shoulder girdle muscles

Muscle	Organ	Meridian	Tooth	Emotion
Supraspinatus	Brain	Anterior median VC		Uncertainty, fear of new steps
Infraspinatus	Thymus	Triple Heater TR		Joy
Subscapularis	Heart	Heart C	12, 22	Joy ↔ sadness, emotional exhaustion
Teres minor	Thyroid gland	Triple Heater TR		Condemnation, criticism, joy
Latissimus dorsi	Pancreas	Spleen and pancreas RP	16, 26	Overwork, exhaustion, care
Deltoid	Lungs / Large Intestine	Lungs P, Large Intestine GI	13, 23	Insecurity, loss of support, sadness, sorrow
pectoralis major (clavicular portion)	Stomach	Stomach E	15, 25	Rejection, resentment, denial
Pectoralis major (sternal portion)	Liver	Liver F	15, 25	Pride, contempt, anger, discontent
Teres major	Pineal body	Back median VG		Restrained emotions, melancholy
Coracobrachialis	Lungs	Lungs P		Sadness, melancholy, sorrow
Pectoralis minor	Spleen	Spleen and pancreas RP		Feeling depressed, lack of energy, difficulty expressing yourself, worrying
Trapezius (upper portion)	Eye, ear, kidneys	Kidneys R	11, 21	Fear, survival
Trapezius (middle and lower portions)	Spleen and pancreas	Spleen and pancreas RP	18, 28	Caring, excessive responsibility
Levator scapulae	Parathyroid glands	Triple Heater TR		Irritability, persistence, joy
Rhomboid muscles	Liver	Liver F		Anxiety, worry, joy
Serratus anterior	Lungs	Lungs P	13, 23	Sadness, melancholy, sorrow, inability to 'take a deep breath'
Biceps brachii	Stomach	Stomach E		Rejection, anxiety, resentment, fear of not coping, denial
Triceps brachii	Spleen and pancreas	Spleen and pancreas RP		Mistrust, isolation, care

Muscles' reflectory connections make them very susceptible to influence of various environmental factors which can be a source of instability and subsequent the shoulder girdle joints dysfunction. This is also the basis for the statistically identified and repeatedly mentioned in the orthopaedic model pattern between the pancreatic and thyroid glands diseases and pain and shoulder joint function limitation.

Reflectory connections are formed not only at the level of muscles but also at the level of connective tissue formations due to which the shoulder joint itself is subject of influence from other bodies structures (Figure 6).

Figure 6: Shoulder joint functional connections (structural and reflectory)



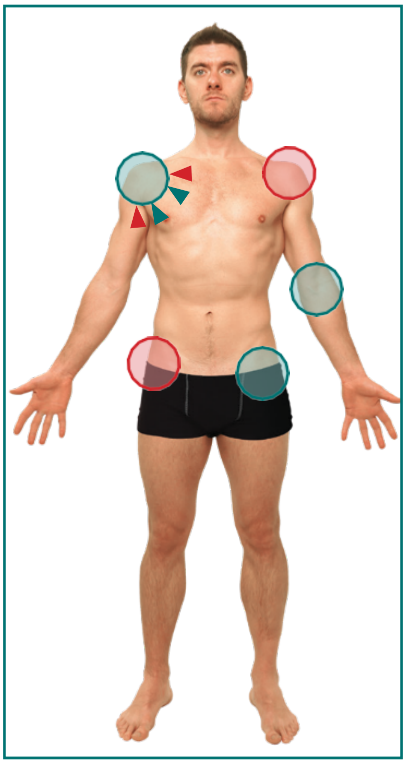
In applied kinesiology and neurophysiology, movements are performed along the crossed reflectory arches, for example, the left hip joint - the right shoulder joint, the elbow joint - the contralateral shoulder joint. This is due to the their reflectory paths crossing at the level of the spinal cord and because of gait reflexes (stepping pattern) patterns. That is, with the hip joint dysfunction, secondary reflectory dysfunction of the shoulder joint on the opposite side may manifest. (69 - 73)

The shoulder joints are associated in a mirror-like manner. This is a classic mirror pattern: the right and left limbs work in a synchronous, mirror-like balance. When mobility is limited or there is a fascial block in one shoulder, the other shoulder joint often performs a compensatory function which is confirmed by clinical observations and EMG studies. (74, 75)

The shoulder and hip joints are also associated on the same side. Here we can see the concept of breathing pattern associations: the position of the diaphragm, chest and pelvis changes during the period of inspiration and expiration; the shoulder and hip joint located on one side of the body work in concert to maintain postural balance. This is reflected in the ipsilateral respiratory pattern models (analogous joints are involved synchronously). This is used in applied kinesiology, to correct respiratory patterns and postural dysfunctions.

In case of any of the listed joints primary dysfunction (trauma in the anamnesis, stability disturbance of muscular or ligamentous genesis, etc.), reflectory relationships underlie the clinical manifestation at the level of the shoulder joint (Figure 7). In this case, primary dysfunction can proceed latently.

Figure 7: Reflectory pain in the shoulder area

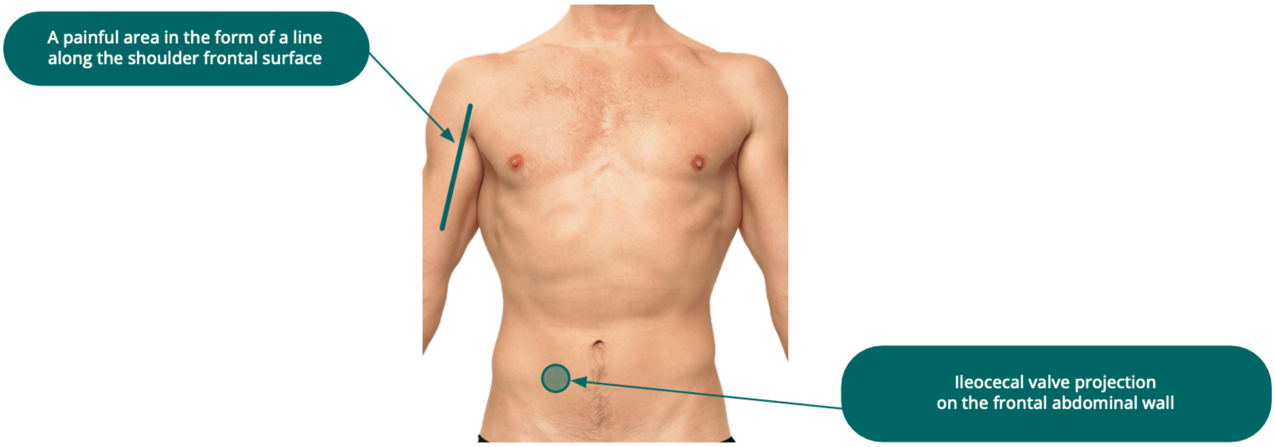


We found that during MMT (manual muscle testing) of the shoulder flexor muscles (*pectoralis major*, *coracobrachialis*, *deltoideus*) in some cases their functional weakness or hyper-reflexivity can be eliminated by provoking the contralateral or, less frequently, ipsilateral foot. In these cases, the patient complains on the shoulder joint dysfunction (pain, limited movement) but, in fact, the source of the problem is the primary dysfunction at the level of the foot or ankle. We identified talus subluxation (38%), plantar aponeurosis dysfunction (46%) and calf muscles functional weakness (16%) among such dysfunctions. The disruption of muscle group reflexivity, i.e. shoulder flexors, we believe to be a compensatory sanogenetic reaction of the body aimed to the system static and dynamic balance restore and may not manifest itself clinically for a long time but to be a source of the shoulder joint structural disorders (Figure 8).

Figure 8: S Reflectory connection 'foot - shoulder' demonstrating the shoulder joint diseases causality originating from the foot latent dysfunctions

The source of reflectory pain in the shoulder joint area may be the neurolymphatic reflex of the open ileocecal valve (ICV). The area of this reflex is located along the inner edge of the right *biceps brachii*. Patients often indicate a line in the projection of the inter-tubercular groove, along the tendon of the long head of the right *biceps brachii* (Figure 9).

Figure 9: Neurolymphatic reflex of the ileocecal valve



Reflectory pain from ICV is often interpreted as biceps tendinopathy (according to the orthopaedic concept) or TC, trigger cord, according to the FDM model. The ICV correction allows to eliminate the patient's complaints during some minutes.

The shoulder joint might be a 'target' of a large amount of referred pain. The mechanism of reflected pain from internal organs is realised through somatovisceral and viscerosomatic reflexes (excitation of sensory neurons, incorrect pain localisation by brain) due to general innervation (through C3-C7, T1-T3 spinal cord segments) (Table 12).

Table 12: Reflectory connections of the shoulder girdle muscles

The source	Example	Nature of pain	Innervation
Heart:	Myocardial infarction, angina	Pain in the left shoulder, dull, squeezing, sometimes radiating to the neck, jaw and arm	C4 - C5 (n. supraclavicularis)
Lungs, pleura:	Pneumonia, apex tumors, pleurisy	Pain in the shoulder/ supraclavicular region on the affected side of the organ, intensifies with respiration, coughing	C4, upper thoracic ganglia
Cervical spine:	Osteochondrosis, disc herniation	Aching/shooting pain, one-sided, provoked by neck movement	C5 - C7 (n. radialis, musculocutaneus)
Hepatobiliary tract, liver, gallbladder:	Diaphragmatic abscess, cholecystitis	Pain in the right shoulder	C3 - C5 (through n. phrenicus)
Spinal cord and plexuses:	Plexopathy, tumors, syringomyelia	Different pain + sensory/ motor disturbances	Brachial plexus, cervical nerves

Therefore, according to the muscle-neurological or reflectory model the causes of pain and the shoulder joint area dysfunction are quite diverse:

- reflected pain
- muscle dysfunction
- dysfunction of shoulder girdle and upper limb MFC
- misalignment of the ligaments receptors and joint capsules
- neuromotor reflex pathological activity, neurolymphatic ICV reflex, and
- dermatomotor reflex, dental-somatic reflex, emotional-muscular reflex, viscero-motor reflex and viscero-somatic reflex.

Such a variety of etiological factors makes the search of the shoulder joint disease source quite difficult. However, understanding the source of its dysfunction and its correction in many cases allows you to help the patient quickly and reliably.

Conclusion

The large number of approaches and proposed treatment methods highlights the complexity shoulder joint diseases problem. Each of the models presented here demonstrates significant advantages, and which of these models a specialist should choose depends on many factors, accessibility for the patient, the balance between epy efficacy and side-effects, the degree of the technique mastery by the specialist, the reliability of diagnostic techniques, etc.

But the success of the specialist in the prevailing majority of cases will depend primarily on the correct diagnosis because, according to medical proverb '*Qui bene diagnoscit, bene curat*' (whoever diagnoses well, treats well).

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About

A new generation and synthesis of manual muscle testing (MMT) protocols have appeared in the Ukraine, Russia and Central Europe thanks to the work of Gleb Kirdoglo, MD, PhD. At the invitation of medical centres and specialised associations, the UAAK organises training cycles in Germany, Estonia, Latvia, Kazakhstan, Moldova, and Israel. In 2012, the *Ukrainian Association of Applied Kinesiology and Medical Rehabilitation*- UAAK- was created and registered. The number of students who have attended the training now exceeds 5,000. The President of the Ukrainian Association, Dr Gleb Kirdoglo, initiated the creation of professional AK communities in Kazakhstan and Moldova. These MMT research results have been presented at major scientific conferences in Kyiv and Moscow. Courses to learn such techniques directly from Dr Kirdoglo and his colleagues are available via http://eanw.info/uapk.html. Over 700 specialists from 9 countries have become members of the UAAK.

Also by this author

Kirdoglo GK. Etiopathogenetic significance of hidden compression syndromes in chronic low back pain syndrome development. URL Asia-Pac Chiropr J. 2023;3.3. apcj.net/Papers-Issue-3-3/#KirdogloLBP