# Functional reactivation for neck pain patients

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## Introduction

The natural history of neck pain is poorly understood, and little research about its causes or treatments has been performed (Borghouts et al. 1998). A number of factors handicap both scientists and clinicians in pursuit of answers for this common problem. First, the severity of symptoms is not directly related to the severity of trauma. Second, objective findings are poorly correlated with the symptoms reported in the head, neck, or upper quarter regions. The biomedical model depends upon accurate identification of the cause of pain and unfortunately this has proven elusive.

The pursuit of the structural cause of neck pain has led to excessive diagnostic testing with imaging modalities which rarely succeed in finding the pain's cause. In the neck, the false positive rate for imaging has been reported to be as high as 75% with an asymptomatic population (Boden et al. 1990, Teresi et al. 1987). Such poor specificity marks imaging as an inappropriate screening method. Bush found that most cervical disc herniations regress with time without resorting to surgery (Bush et al. 1997). Additionally, he found that the larger the disc herniation the more likely it is to spontaneously reabsorb. Therefore, it is important to avoid 'labeling' patients as being damaged since this may have disabling effects in terms of promoting the 'sick role' and interfering with functional reactivation (Main & Watson 1999, Bogduk 2000).

While exact diagnosis of the cause of pain is difficult it is fortunately quite easy to 'rule out' sinister factors such as tumor, infection, fracture or serious medical disease (Spitzer et al. 1995). Additionally, management directed at patient reassurance and early functional reactivation are the most promising (Spitzer et al. 1995). Medicalization of the problem by excessive diagnostic testing or overly aggressive treatment is not warranted and is likely iatrogenic.

According to a recent meta-analysis review of manual therapy (mobilization, manipulation, massage) it was concluded that there was insufficient data to recommend it (Harms-Ringdahl & Nachemson 2000). But, when manual therapy was used in combination with other active treatments there is moderate evidence of benefit. A recent study found that chronic neck pain patients receive more benefit from a combination of low-technology exercise and manipulation than with either high-technology exercise or manipulation alone (Bronfort et al. 2001). Most outcomes were similar for the two exercise groups, except that patient satisfaction was higher for the combined exercise and manipulation group.

## Functional issues

Since structural pathology does not correlate well with pain the focus of care is on restoring function. For this reason functional disturbances in activities of daily life (ADLs) such as sitting, carrying, posture, etc have become the focus of health care professionals looking for treatment targets. In addition, specific performance deficits involving endurance, flexibility or coordination are all potentially clinically relevant.

An important concept is that of instability. Andersson has defined a functional instability as, ‘loss of the ability of the spine under physiologic loads to maintain relationships between vertebrae in such a way that there is neither initial nor subsequent damage to the spinal cord or nerve roots, and in addition, there is neither development of incapacitating deformity or severe pain. (Andersson & Ortengren 1994). Agonist-antagonist muscle co-activation is a central aspect of joint stability. Loss of normal function and balance of agonist and antagonist muscles will compromise joint stability.

Panjabi has concluded that most whiplash patients experience mild soft-tissue injury which does not cause tissue failure and thus are undetected by static imaging procedures (Panjabi et al. 1998). In these sub-failure injuries the soft tissues are not torn, but are stretched beyond their elastic limit resulting in instability and poor healing. Instability will render the osteoligamentous structures vulnerable to repeated strain with normal ADLs and be compensated for in the muscle system.

One model which can explain these compensations is called the pain-adaptation model of Lund (Lund et al. 1991). Lund theorized that when pain is present there is a decreased activation of muscles during movements in which they act as agonists and increased activation during movements where they are antagonists (Lund et al. 1991). This model is in stark contrast to the pain-spasm-pain model which suggests that muscle tension is necessarily increased when painful stimuli are present. Rather, it appears the rule is **muscle imbalance**, with certain muscles tending towards hyperactivity and others towards inhibition. It is well known that certain muscles respond to inflammation or injury by becoming inhibited (Barton & Hayes 1996, Jull et al. 1999, Jull 2000, Silverman et al. 1991, Watson & Trott 1993, Treleaven et al. 1994) and atrophying (Hallgren et al. 1994, McPartland et al. 1997). It is also commonly accepted that other muscles such as the upper trapezius (Balster & Jull 1997, Bansevicius & Sjaastad 1996, Hall & Quintner 1996, Madeline et al. 1999, Nederhand et al. 2000) and sternocleidomastoid (SCM) (Jull 2000) respond to injury or overload by tensing or becoming overactive.

As a result of agonist-antagonist muscle imbalance movement patterns are altered and synergist overactivity is frequently observed. This type of altered motor control easily escapes traditional functional testing which only assessed strength and flexibility. Edgerton et al. studied altered muscle activation ratios of synergist spinal muscles during a variety of motor tasks in whiplash patients (Edgerton 1996). They discovered that underactivity of agonists and overactivity of synergists was able to discriminate chronic neck pain patients from those who had recovered with 88% accuracy. They concluded that, ‘The nervous system apparently can detect a reduced capacity to generate force from a specific muscle or group of muscles and compensate by recruiting more motoneurons. This compensation can be made by recruiting motor units from an uninjured area of the muscle or from other muscles capable of performing the same task’. Lauren et al. demonstrated strong support for this element of compensation with their functional study of neck and shoulder pain incidence (Lauren 1997). Their study showed a higher incidence of neck and shoulder pain in those individuals who performed tasks either extremely fast or slow. While those performing them in a medium range had very low incidence of neck and shoulder pain. Nederhand showed that a decreased ability to relax the upper trapezius muscles during static tasks as well as following exercise in mild-moderate whiplash patients correlates with increased neck pain following whiplash type injury (Nederhand 2000).

Watson and Trott (1993) found that two examination findings could differentiate headache from non-headache patients:

- forward head posture
- decreased isometric strength and endurance of neck flexors.

Treleaven et al. (1994) found the following three factors could distinguish post-concussion headache patients from asymptomatics:

- upper cervical joint dysfunction
- weak neck flexors
- tight suboccipitals.

Reduced endurance of the deep neck flexors is also found in a number of other published studies (Barton et al. 1996, Jull et al. 1999, Jull 2000, Silverman et al. 1991). Consistent with these findings are the reports that sustained loading of the neck in static postures leads to muscle fatigue and pain. Hamilton estimated that the critical threshold for static loading is 10% of maximum voluntary contraction (MVC) ability (Hamilton 1996). A load of this intensity can be maintained comfortably for 10 min. Jensen recommended that static work should not be maintained at levels above 2% of MVC (Jensen et al. 1993). Veiersted and Westgaard (1992) evaluated functional work tasks and concluded that symptoms were triggered by static loads of only 1.6% MVC.

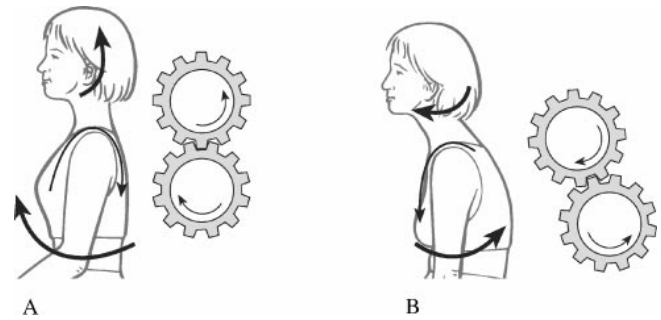


**Fig. 2** Head forward posture; (A) normal, (B) faulty.

## Functional assessment of neck pain patients

The ideal functions to assess are dynamic tasks such as lifting and carrying and static tasks such as maintenance of neck or shoulder posture. Unfortunately, accurate measurement of these functions is elusive. Two simple forms of assessment that can yield much clinically useful information are posture and movement pattern analysis.

Poor **posture** takes joints out of their aligned 'centrated' positions and alters muscle balance between antagonist muscles (Figs 1 & 2). A typical example is a person working on a computer with a head forward position. This will overstress both the upper (hyperextension) and lower (flattening) cervical spine.

**Fig. 1** Increased kyphosis from faulty sitting posture.

**Movement patterns** are important to assess because classic muscle tests evaluate strength, but not the quality of movement (Janda 1996, Liebenson 1996, Liebenson et al. 1998, Lewit 1999, Murphy 2000). Movement patterns involve coordinated movement involving many muscles. An example of a stable vs. unstable movement pattern is if cervical spine neck flexion is performed with or without hyperextension of C0-C1. The deep neck flexors (DNFs) maintain a 'neutral' alignment of the C0-C1 joint during head/neck flexion. But, if the sternocleidomastoid (SCM) predominate they can also raise the head, but only with the C0-C1 joint hyperextended (Jull et al. 1999, Treleaven et al. 1994). In both coordinated and incoordinated movement patterns the neck flexion movement can test as strong!

Poor posture and/or faulty movement patterns are typical kinetic chain dysfunctions which cause functional instability by increasing biomechanical load to injurious levels. Such repetitive strain irritates pain sensitive structures and can be a key perpetuating factor of cervical pain. Common clinical relationships are shown in Table 1.

Treatment planning requires that the association between different painful tissues and functional deficits is uncovered. Otherwise treatment is purely empirical without even a working hypothesis to guide it. Fortunately, there is a predictable pattern for the relationship between painful joints and muscles (trigger points) and their associated muscle imbalances (short and inhibited muscle antagonists) (Table 2).

Pain referred from the SCM muscle(s) or upper cervical joints is related to muscle imbalance involving shortened suboccipitals combined with overactivity of the SCM and inhibition of the DNFs (Fig. 3). A simple screen is to perform the head/neck flexion test (Fig. 4).

**Table 1** Relationship between key sources of biomechanical overload and painful joints. (Taken from Liebenson C, Skaggs C. The role of chiropractic treatment in whiplash injury. In: Malanga G (ed), Whiplash, Sched pub 2002. Hanley Belfus, Philadelphia)

| Painful joint | Faulty posture | Faulty movement pattern |
|---|---|---|
| Cervico-cranial | Head forward | Neck flexion |
| Gleno-humeral | Rounded shoulder | Scapulo-humeral rhythm |
| Upper ribs | Slumped posture | Respiration |
| TMJ | Chin poke | Mouth opening |

**Table 2** Key myofascial or osteoligamentous pain syndromes and muscle imbalances associated with head and neck dysfunction (Taken from Liebenson C, Skaggs C. The role of chiropractic treatment in whiplash injury. In: Malanga G (ed), Whiplash. Sched pub 2002. Hanley Belfus, Philadelphia)

| Painful joint | Trigger points | Shortened muscle | Inhibited muscle |
|---|---|---|---|
| Cervico-cranial | SCM | Suboccipitals | DNFs |
| Gleno-humeral | Upper trapezius | Levator scapulae or subscapularis | Lower trapezius |
| Upper ribs | Scalenes | Pectorals | Diaphragm |
| TMJ | Lat. pterygoids | Masseter | Digastricus |

The neck flexion coordination test is positive if the chin pokes forward as the patient raises the head off the table. The test is negative if the head and neck curl in as the head is lifted towards the chest. An additional test can involve statically pre-positioning the head just 1 cm off the table with the chin tucked in. Then, ask the patient to hold this position for 10 seconds. If the chin pokes, the head lifts or drops the test is considered positive for poor endurance of the DNFs. Finally, a quantifiable test involves the use of a blood pressure cuff device preinflated to 20 mmHg under the head to support it without pushing the head up. The patient is instructed to perform chin tuck movements to increase the pressure by 2 mmHg increments up to a maximum of 30 mmHg (Jull et al. 1999, Jull 2000). Performance ability with this test is compromised in patients with cervicogenic headache compared to asymptomatic individuals.

Upper cervical flexion is important for maintenance of good spinal statics. The results of the head/neck flexion test can often be predicted on the basis of postural analysis of the head and neck. In standing analysis a head forward posture with a chin poke indicates agonist/antagonist/synergist muscle imbalance. In particular, the cervical extensors (the upper trapezius and suboccipitals) are not balanced by the co-activation of the DNFs – longus colli and capitus. As a result sternocleidomastoid substitution occurs.

The clinical relevance of this imbalance is that treatment of the myofascial or articular pain generators without subsequent neuromuscular reeducation will likely not correct the underlying problem. For instance, if trigger points in the SCM or painful cervical joints are present the inhibition of the DNFs must be corrected or else it is likely the trigger points or joint dysfunction will recur. Sometimes, it is the trigger point or joint dysfunction which is primary. The key is to see the chain reaction in the motor system and determine when the joint dysfunction, trigger points and movement patterns are all normalized.

Other functional tests of relevance in head/neck syndromes are listed in Table 3.

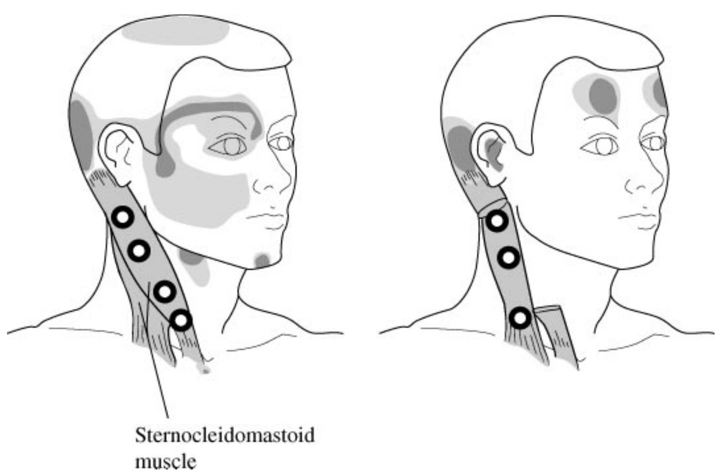


**Fig. 3** SCM trigger points. Reprinted from *Clinical Application of Neuromuscular Techniques*, Volume 1, Chaitow & Delany, by permission of the publisher Churchill Livingstone.)

## Treatment of neck pain patients

Most head/neck pain patients require a relatively straightforward evaluation and treatment approach since for the majority the prognosis is reasonably good. Unless there are 'red flags' of serious disease the patient should be reassured and reactivated. If needed pain-relief treatments should be offered. Avoiding unnecessary surgery, overmedication, and overexamination (especially with diagnostic imaging) is important in order to prevent 'medicalizing' the problem. In contrast, patients who are not satisfactorily recovering by the subacute phase require more aggressive management since it is easier to prevent than to treat chronic pain. The key time frame where aggressive management should be considered is between 4 and 12 weeks. Those with 'yellow flags' (psycho-social) risk factors of chronicity should be more aggressively managed even earlier. Such 'yellow flags' include past history of neck injury, low self-rated health, and high levels of psychological distress (Croft et al. 2001). This still does not mean MRIs on every patient, but it does mean a rehabilitation specialist

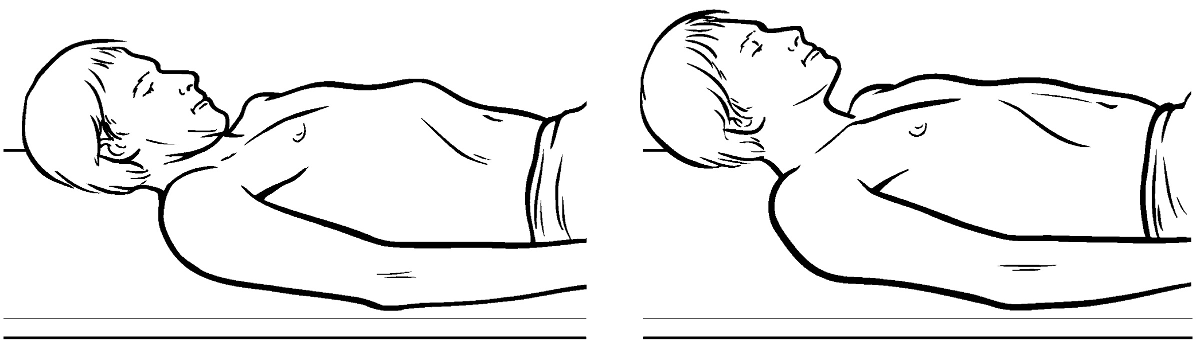


**Fig. 4** Head/neck flexion coordination test (after Janda); (A) normal, (B) faulty. Reprinted from *Muscle Energy Techniques*, Chaitow, by permission of the publisher Churchill Livingstone.

**Table 3** Functional tests for head/neck syndromes: see Liebenson C 2001. Self-treatment of mid-thoracic dysfunction: a key link in the body axis. Part 1: Overview and assessment. Journal of Bodywork and Movement Therapy 5: 90–98. (Taken from Liebenson C, Skaggs C. The role of chiropractic treatment in whiplash injury. In: Malanga G (ed) Whiplash. Sched pub 2002. Hanley Belfus, Philadelphia)

Postural analysis
- Head forward posture
- Shrugged or rounded shoulders
- Upper thoracic kyphosis

Movement patterns
- Head/neck flexion
- Scapulo-humeral rhythm
- Respiration
- T4-8 Extension test

should be involved. In particular, one with training in cognitive-behavioral approaches. The important point is that when a full diagnostic work-up is recommended it should not be limited to MRIs or other structural evaluations, but also include functional/physiological testing such as a functional capacity evaluation and a psycho-social evaluation. Table 4 presents an overview of the key steps to recovery.

The musculoskeletal medicine approach to neck pain embraces the current evidence for **advice, manipulation** and **exercise**. Most importantly it recognizes the importance of reassurance and reactivation for promoting a quick recovery and minimizing the risk of chronicity.

## Advice

Advice for patients with head and neck pain is designed to reassure them about the positive prognosis for their condition and the safety of gradually resuming normal activities. Activity modification advice is more valuable than advice to avoid activities (Borchgrevink et al. 1998). Helpful biomechanical advice includes ergonomic suggestions for the workstation (chair, computer, phone, and desk) or when performing movement stereotypes such as carrying a briefcase, reaching overhead etc.

Reassurance that hurt does not necessarily equal harm is vital. If movements or postures cause symptoms to peripheralize from the neck down to the arm those activities should be reported to the clinician and modified (Rosenfeld et al. 2000). But, other activities which may only be locally uncomfortable are usually not harmful and patients should be reassured of this through a problem-solving approach (Vlaeyen et al. 2001, Shaw et al. 2001).

While improved biomechanics during sustained static tasks or repetitive dynamic tasks is important, patients should not be overly vigilant about posture in all activities (Indahl et al. 1995). Full range of motion will be lost if one is educated to stay in a 'neutral range' all the time. Full range activities that involve light load will beneficially stretch and mobilize tissues. Traditional back school should be replaced with a cognitive-behavorial approach emphasizing that hurt doesn't necessarily equal harm

**Table 4** Keys to Recovery --- the 5 Rs. (Taken from Liebenson C, Skaggs C. The role of chiropractic treatment in whiplash injury. In: Malanga G (ed), Whiplash. Sched pub 2002. Hanley Belfus, Philadelphia)

1. **Reassurance** that no serious disease is present and that improvement is likely to begin rapidly.
2. **Relieve** pain with medication or manipulation.
3. **Reactivation** advice that normal activities can be resumed (walk, swim, bike, etc.) and education about simple activity modifications to reduce biomechanical strain (i.e. Brugger relief position, chin tucks).
4. **Re-evaluation** of those entering the subacute phase for structural, functional or psycho-social factors.
5. **Rehabilitate/Recondition/Reeducate** muscles with McKenzie, stabilization, progressive strengthening, or cognitive-behavorial (indicated if high 'yellow flag' score) approaches.

(Indahl et al. 1995, Manniche et al. 1999).

Patients suffering neck pain do have injured tissues. They should be informed that those tissues will heal better with light activity than with rest (Indahl et al. 1995). Pain 'flare-ups' are normal and to be expected and are not a sign of further injury. Stress and emotional tension will tend to reduce an individual's pain threshold and intensify the symptoms associated with such 'flare-ups' (Indahl et al. 1995). Patients should be educated that stress plays a role, but that it does not cause injury and that the pain will run a course. Advice about physiologic coping strategies such as breathing techniques, light exercise, meditation etc is also important.

## Manipulation

Manual therapy and manipulation is important for facilitating recovery from neck pain. Normalization of afferent information from joints, muscles, skin, and fascia is important for promotion of healing and rehabilitation of function. If joints are 'locked' in the mid-thoracic or upper cervical regions movement will follow the 'path of least resistance' and overstrain vulnerable areas such as the lower cervical spine. Thus, joint mobilization/manipulation will improve load-sharing and thus functional stability. Gentle techniques include post-isometric relaxation, muscle energy, or other non-thrust techniques. Adjustments – by highly trained experts in high-velocity short amplitude thrust manipulation such as chiropractors – are appropriate for joints which are carefully examined and found to have restricted motion.

Shortened muscles and taut fascia should be relaxed and lengthened to improve functional stability. The suboccipitals, latissmus dorsi, pectorals, and hip flexors are prime examples. Adjunctive therapy to manual therapy can include physical agents such as heat/ice, electrical muscle stimulation, traction etc. Many of these methods are well suited for early care in the acute phase, but they can lead to patient dependency. These passive modalities should be seen as means to facilitate active rehabilitation and not as ends in themselves.

## Exercise

While advice can reduce the source of external repetitive strain, and manipulation can improve the performance of key muscles or joints, exercise is also frequently needed to improve the overall fitness of the entire kinetic chain as well as to facilitate 'deep' muscles important for spinal segmental stability.

Exercises aimed at improving motor control are usually recommended. These movements focus on control rather than power. In fact, it is poor motor control not poor strength that has consistently been found to be of importance in functional instability. Patients learn to appreciate that the quality of the movement is more important than the resistance or repetitions. This is very different from how most people view exercise and so patients are re-educated about 'therapeutic' exercise. The first goal is for the patient to learn how to produce and control the movement in his or her functional training range. This is the painless range within which movements are performed in a coordinated way. Such training for coordination during arm abduction tasks has been demonstrated to be successful (Babyar 1996).

Training the 'deep' muscles which guide and control movement is difficult since these muscles are not ordinarily under voluntary control. Learning theory has been applied to this type of training (Shumway-Cook & Woollacott 1995). The first stage involves gaining conscious awareness of the poor postures and movement patterns. This is called the **cognitive-kinaesthetic** stage. The second stage is where awareness of the corrected postures and movements is achieved and thus corrective movements can be performed and practiced. This is called the **associative** stage. The third and final stage occurs when after daily practice of corrected movements and postures for many weeks a new motor program forms in the central nervous system such that improved motor control becomes more automatic. This is the **autonomous** stage. Many forms of exercise share these principles such as Pilates training and segmental spinal stabilization training.

Thus, while exercises initially require conscious control, the goal is to automatize coordination to lessen the consequences of poor motivation and compliance. Patients should first become consciously aware of the muscle or part of the body that is to be activated. Exercises and coordinated activities are prescribed which train the patient how to gain this volitional control. Finally the motor program becomes a subcortical engram and the patient achieves the desired effect without having to vigilantly concentrate on the function. Thus the patient is able to protect the vulnerable region during ADLs and when exposed to unexpected perturbations.

In the cognitive-kinaesthetic stage the clinician must initially find the patient's **functional range** – the position or movement which centralizes or decreases pain without unwanted superficial muscle activity (i.e. upper trapezius). The patient should then demonstrate that they have the kinaesthetic awareness to produce isolated movements of different joints and that they can find and maintain a 'neutral position' of certain key joints such as the cervico-cranial or scapulo-thoracic. This will show that they have learned to coordinate and co-activate antagonist muscles.

An example is teaching the patients to disassociate related movements such as scapulo-thoracic from scapulo-humeral or cervico-cranial from cervico-thoracic. The patient should be able to move their arm in abduction or flexion while fixing the scapulae inferiorly against the thorax. If excessive shrugging of the shoulder occurs this signifies poor scapulo-thoracic control. Another example is that the patient should be able to perform a chin tuck and use this skill dynamically so that when they rise from a chair or a bed they can avoid poking their chin. This type of postural correction is a key component in Alexander training methods.

In the associative stage the corrected postures and movements are trained repetitively to build *endurance* of the 'deep' stabilizers. The key here is to find two or three faulty or pain producing movements and focus on improving their function. The movements are not threatening since load is kept light (less than 50% of maximum voluntary contraction ability). But frequent repetitions (8–10) of very slow movements (up to 10 seconds/repetition) are required at least twice a day and sustained hold times (5–6 seconds). A minimum duration of 4–6 weeks is required.

In the autonomous stage improved motor control is integrated into ADLs on an automatic basis. This should begin to become 'habit' so that a low degree of attention is required and compliance and motivation issues recede into the background.

## Conclusion

Neck pain syndromes require an approach which focuses on restoring function. This necessitates a biopsychosocial approach not a biomedical one. Structural diagnosis is often overemphasized thus making patients fear movement and think of themselves as damaged. Such 'labeling' distracts patients from the real issues interfering with recovery which are functional/physiological and psycho-social.

Immediate reassurance coupled with early reactivation and pain relief advice is the standard of care for acute neck pain. Treatment should be based on functional assessment, and thus progress at regular intervals can be monitored with functional outcomes. Patient reactivation requires a studied approach which is both not too aggressive in the acute phase, nor too passive in the subacute phase.

Most patients recover uneventfully, but it is the minority of sufferers who challenge health care professionals and the health care, legal, and disability systems to come up with a better approach. Avoidance of the iatrogenic influence of over diagnosis and over treatment of acute patients is part of this. So is the reliance on purely structural (imaging and surgery) or symptomatic (medication, injections, physical therapy modalities, massage, and chiropractic adjustments) approaches for both acute and chronic syndromes. The modern approach is a 'problem-solving' one. Reassurance that there is nothing structurally wrong combined with reactivation advice or functional restoration treatments are the mainstays of this new paradigm.

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