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Management of whiplash and whiplash associated disorders

Introduction

Whiplash-associated disorders (WAD) is the term given to the spectrum of effects frequently experienced by persons, most often via a road traffic collision, after acceleration/deceleration damage to the neck. The cardinal symptom is neck pain, but there are also frequent records of neck stiffness, dizziness, upper quadrant paraesthesia/anaesthesia, headache, and arm pain. Disability, reduced quality of life, and psychiatric depression are correlated with neck-related discomfort. It is a contentious disorder because WAD is always a compensable disability, with others even dismissing it as a legitimate condition.**1**
This is despite the plethora of research that indicates both clinical and psychological manifestations that have management consequences. The pressure of WAD, the treatment process following damage, and factors indicative of both successful and bad recovery will be outlined in this narrative analysis. It will discuss the diagnosis and evaluation of WAD. This will be accompanied by an overview of the latest findings for disease control. Whiplash Associated Disorders (WAD) have rapidly developed into an outsized problem for health care providers and in terms of patient suffering. Reviewing the literature Barnsley and colleagues" found that the share of persons having had a harm and who develop chronic neck pain varies between 14 and 42%. However, there's still considerable controversy over organic vs non-organic reasons for the chronic pain after whiplash.**2,3** Chronic pain may be a multi-component phenomenon with associations between injury, pain experience, impairment, physical maintaining factors, emotional and cognitive factors, further as environmental and socio- economic influences.**4,5** Individual's reaction to perceived disability may include changes in behaviour and physical functioning. These changes can maintain the matter itself or perhaps increase the degree of disability.**6** it's been suggested that chronic pain management programmes should address and handle behaviours, like avoiding activities, instead of the diagnosis and its cause.**7** General principles of learning may be applied to the physical, psychological and practical habits related to chronic conditions like pain and their accompanying fears.**8** Harding**8** also claims that rehabilitation has three main aims with regards to behavioural change i.e. decreasing undesired behaviours, initiating and increasing, moreover as maintaining, desired behaviours. The

individual's lifestyle, available training time, and occupation are possible targets in making the foremost of the rehabilitation programme. Thus, rehabilitation of the chronic pain patients requires a wider approach to achieve success, and any programme must include active patient participation**9** and measures to extend patient's self-efficacy beliefs.**10** Typically, physiotherapy approaches to chronic WAD are concerned with movement and performance. Reports of treatments like manipulation and mobilization,**11, 12** cervical traction,**13** acupuncture,**14, 15** transcutaneous nervous stimulation**16** and myofascial trigger points treatment,**17** are examples in line with this tradition. However, an outsized proportion of studies suffer from methodological deficiencies. Follow up assessment varies between `none '**15** immediate '**12** 5 minutes after treatment,**11** 1 week period,**14** 1 month **13** and 6 months.**16, 17** Thus, the clinical significance in a number of the results is also questioned because of a brief follow up period. Fattori and colleagues **14** did not randomly allocate patients to physiotherapy and acupuncture groups. Another study was retrospectively using telephone interviews for data collection.**12** Interviewers expected the patient to recall symptoms they would had both before and after the treatment. These examples raise serious questions of the inner validity within the studies. The failure to report compliance with exercises **16,17** used as home assignments is additionally a threat to internal validity. To conclude, it is important that new treatment approaches to problems like WAD are developed and evaluated in reasonably controlled studies. Integration of psychological techniques and physiatrics provides a multimodal approach within the treatment of patients with chronic pain. Combining these aspects is a vital strategy towards maximizing treatment effects.**8,18** Pain definitions that involve physiological, psychological and environmental components also need evaluation methods which are suited to reflect all three of those factors and taking under consideration pain characteristics, for instance if it's acute or chronic. As a consequence three measurement domains - the physiological, the behavioural and also the cognitive-affective components are distinguished besides the measurement of pain intensity.**4**

Pathophysiology of WAD

There is debate over the pathological mechanisms that underlie the WAD signs. The broad range of symptoms is one explanation for this. A spectrum of magnitude, from moderate irritation to long-term debilitating suffering, has to be provided for by pathological reasons. Neck discomfort, headache, memory disturbances, jaw pain, and various other signs are signs of whiplash, and it is unknown if they are manifestations of one incident, describe several accidents that occur at the same time, or are due to the initial crash. The anatomy of WAD was not explicitly discussed in the QTF (Quebec Task Force) consensus statement on whiplash,**19** and only a few suitable reports on the diagnosis of WAD were found. Research has continued on the pathophysiology of WAD in the last 10 years, but our understanding remains restricted. Any of the drawbacks are linked to analysis methodology. Pathology conclusions are frequently drawn from postmortem specimens, although such tests contain only critically wounded patients who have suffered massive injuries and are thus not indicative of mild injury patients. The research can also be useful because the nonlethal injuries found in this category may be similar, even though more serious, to injuries in patients with severe incidents that did not lead to death.**20** It is also difficult to understand studies in which cadavers are exposed to damage necessary to induce whiplash injury due to the different properties of living tissue relative to stiffer, cadaver in

animal experiments have interpretive drawbacks. Findings after surgery are beneficial, but surgery is only performed for a small group of WAD, and the surgery may occur after a lengthy period of chronic complications and at a period where initial pathological changes may no longer be visible and degenerative changes prevail. Imaging experiments are beneficial, but it may be important to neglect such pathological mechanisms. Diagnostic injections can help to determine the source of pain, but they have technological disadvantages.

Patient history and interview

It is mandatory that, due to their consistent prognostic potential, pain and injury be assessed as the first phase in clinical evaluation. The 11-point visual analog scale or numerical assessment scale is prescribed by the guideline-recommended pain scales, and the recommended indicator of impairment is the Neck Disability Index due to its clinimetric properties.**21** Other measures are also appropriate, though and some include the Whiplash Disability Questionnaire and the Patient Specific Functional Scale.**21** It is also important to consider There are several psychological questionnaires online, so it is often difficult for clinicians to pick the most fitting questionnaire/s to use. One recommendation is to choose specific questionnaires in the subjective evaluation based on the recorded symptoms of the patient. For example, in patients who experience trouble sleeping because of thinking about the crash, nightmares, or avoidance of driving due to anxiety, early signs of post-traumatic stress may be assumed. Using standardized questionnaires, these effects can be further measured, with the Impact of Events Scale prescribed for use by physiotherapists.**21** A score of 25 or 26 on the Impact of Events Scale suggests a mild level of post-traumatic stress symptoms.**22** Similarly, where other psychiatric causes tend to be present from the patient history and interview, these can also be further. Some questionnaires that could be helpful for physiotherapists, the analysis of scores, and their availability can be useful in the management and decisions taken on the basis of answers to these questionnaires depend on the level of the illness, whether acute or chronic, and this will be discussed below.

Physical examination of WAD

The physical assessment of the WAD patient meets the same general examination protocols commonly followed for the examination of any disease of the cervical spine, but involves certain additional procedures based on WAD testing results. One purpose of the physical test is to use the QTF grading system to assess the grade of the disorder. **23** A Grade II condition would have physical symptoms of reduced neck mobility range and palpable 'tenderness' relative to Grade I, where neck pain is recorded by the patient but without physical signs. Grade III is defined by the existence of clinical neurological symptoms in the dermatomal or myotomal distribution of

reduced muscle power, deep tendon reflexes, and feeling. It should be remembered that many WAD patients will experience diffuse sensory loss or/and generalized muscle fatigue signs, both of which may be bilateral, but these results do not generally suggest peripheral nerve compromise and may signify altered central nociceptive processes. The investigation of nociceptive processes in WAD has been the subject of much research. Systematic reviews suggest that there is good evidence for the involvement of chronic WAD **24**,**35** of augmented central nervous system nociception production and modest evidence that cold hyperalgesia (a possible predictor of these processes) is correlated with weak injury recovery.**26** Clinically, central hyperexcitability can be suspected from patient subjective accounts, including: A standardized questionnaire such as the self-reported Leeds Evaluation of Neuropathic Symptoms and Signs to determine for a neuropathic pain factor can be used to further evaluate these symptoms.**27** Functional assessments can include the use of pressure algometers, discomfort with ice application,**28** or with observed enhanced bilateral responses to the brachial plexus irritation test.**29** Study findings include poorer performance relative to asymptomatic control participants on motor control measures affecting the cervical flexor, extensor and scapular muscle groups; improvements in muscle morphology of the cervical flexor and extensor muscles; lack of strength and stamina of the cervical and scapular muscle groups; and sensorimotor changes manifested by increased re-position of the joint.

Diagnosis and assessment of WAD

The classification of whiplash accidents by the Quebec Task Force (QTF) was put forward in 1953 and remains the classification form now used worldwide. While the QTF system is very simplified and focused only on signs and symptoms, it makes it easier to provide a shared vocabulary regarding the disease for physicians and all people interested in the treatment of patients with WAD. Although health outcomes for this community can be diverse, most patients fall into the WAD II grouping, and this has been defined as one challenge with the QTF system.**30** Changes to the QTF system have been suggested, but have usually been more complicated,**30** and not readily taken up by all parties interested with WAD management for this purpose. In recent years, the WAD diagnosis has improved little. Specific tissue injury or peripheral lesion cannot be detected in the vast majority of cases.**31** While earlier studies identified lesions in the cervical spine at autopsy in people who died as a consequence of a road traffic accident,**32** this work did not translate into the clinical setting, possibly due to the insensitivity of available imaging techniques. The best scientific evidence available is for zygapophysial joint pathology found in highly selective patients with chronic WAD using radiofrequency neurotomy techniques,**33** but its occurrence is not established in the total WAD community. Injury to other tissues, including spinal discs, ligaments, and nerve tissue, is likely to be found in some patients to varying degrees.**31** Existing clinical guidelines for the treatment of acute WAD suggest that radiological imaging be done only to detect WAD grade IV (i.e. fracture or dislocation) and that clinicians adhere with the Canadian C-Spine or Nexus law when producing it in contrast to the lack of advancement achieved in the diagnosis of peripheral pathology, there has been a great deal of reason to characterize the

disorder in terms of its physical and psychological appearance, and some of the main developments in this field have implications for and will be outlined in the clinical examination of WAD.

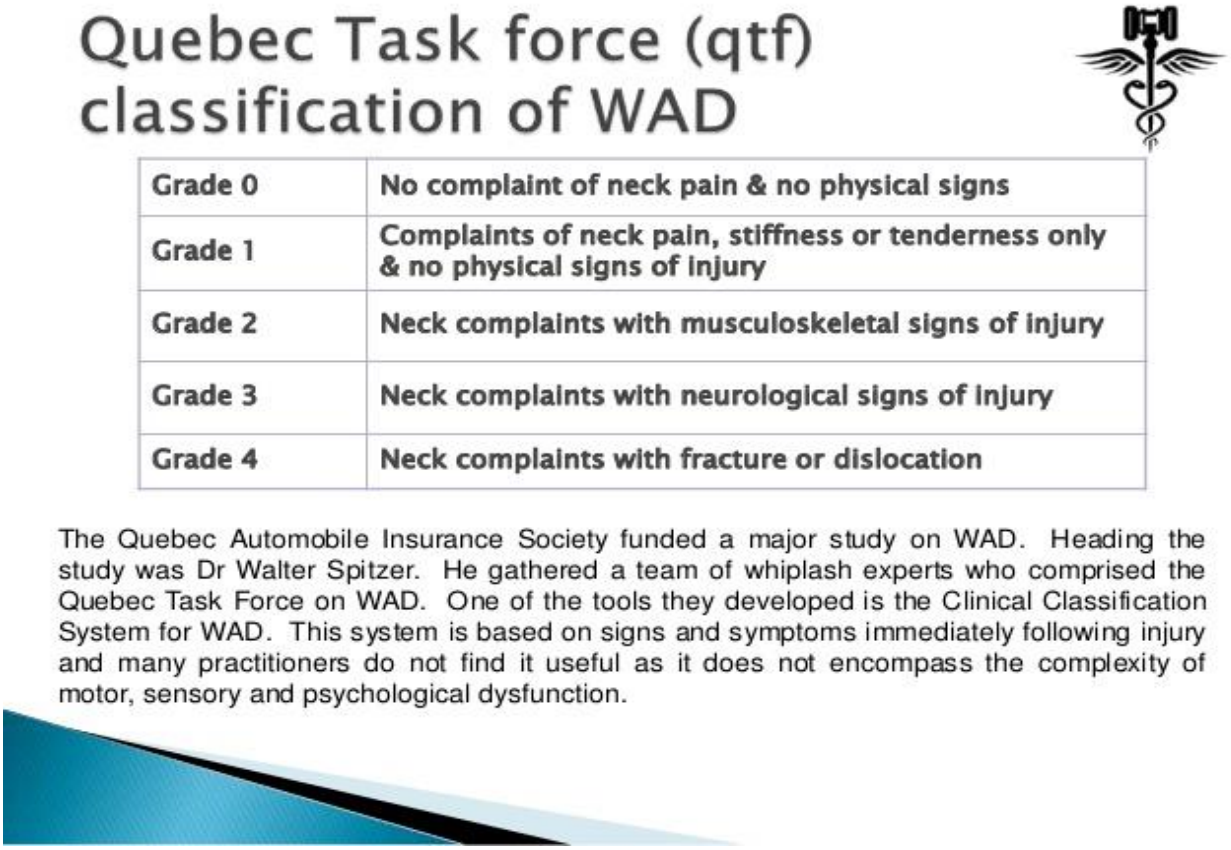


Image available at https://www.slideshare.net/Cain105/just-a-simple-whiplash-16432885

Management of WAD

WAD treatment depends to a certain degree depending on whether the disease is in the early acute stages (usually described as 0-12 weeks) or has already developed a chronic condition (> 12 weeks post-injury). These time limits are unpredictable, but are chosen because they are compliant with existing WAD treatment guidelines.**21**,**34** The therapeutic course of WAD, where much rehabilitation happens in the first 2-3 months, is important because this time period allows the ability to avoid a persistent disease from progressing theoretically. As both physical and psychological influences are involved in both acute and chronic WAD, and there is evidence of strong associations between these factors,**35** approaches to management should be compatible with the existing biopsychosocial paradigm. Surprisingly, there have been comparatively few clinical studies compared to certain other musculoskeletal pain disorders for a disease that involves a major personal and economic strain. Exercise and operation The essence of acute WAD treatment is the provision of advice encouraging the transition to daily activity and

exercise, and this approach is recommended in existing therapeutic guidelines.**21** Different forms of exercise have been investigated, including range-of-movement activities, McKenzie exercises, postural exercises, and exercise reinforcement and motor function. The findings of the study found that six physiotherapy sessions (a multimodal approach to exercise and manual therapy) were only marginally more successful than a single physiotherapist counseling session.**36** However only 45-50 percent of participants in either treatment group reported their condition at short (4 months) and long-term follow-up (12 m) as 'much improved' or 'better' Only 22 clinical experiments that meet the inclusion criteria were reported in a new systematic analysis and only 12 were of reasonable quality.**37** The authors found that exercise prescriptions are successful in relieving discomfort, but these benefits do not seem to be sustained over the long term.**37** Similar to the acute WAD case, it is not clear if one form of exercise is more effective than another form of exercise. For example, when the exercise studied was a specific motor and sensorimotor retraining program for the cervical spine combined with manual therapy, a graded functional exercise strategy and advice demonstrated greater changes in pain severity, pain bothersomeness and functional capacity, compared to advice alone.**38** In another study, similar results were seen when the exercise investigated was a specific motor and sensorimotor retraining program for the cervical spine combined with manual therapy. Exercise and manual therapy should be used in the treatment of both acute and chronic WAD, from a therapeutic standpoint. There is no evidence, however to prove that one method of exercise is preferable to another, and this is a field awaiting more study. The generally limited impact with 'exercise only', mean that a sub-group of patients who demonstrate a stronger response will require either more therapies that it usually would. However, owing to a lack of data, it is not clear which additional treatments can be used or how responders and non-responders should be specifically defined. The advice to physicians, however is that patient outcomes should be tracked and rehabilitation resumed only when there is significant change. The clinician will need to search at any causes that could be involved, such as neurological, environmental, or nociceptive processing factors, among others, in patients whose health is not improving.

Is it possible to do too much too early?

The Task Force on Neck Pain cautioned that high use of health insurance claims might intentionally prolong the healing of WAD during the first few weeks after the injury. A growing body of proof shows that time to recovery is closely and separately correlated with the form, severity, and timing of health care delivery. Two population-based cohorts from Saskatchewan, Canada were analyzed by Côté et al**120** and found an inverse association between the amount of visits to health care made during the first 30 days following injuries and the time it took patients to recover from their WAD. In fact, acute WAD patients who made more than two visits to general practitioners (in the first 30 days following injury), more than six visits to chiropractors, were seen by general practitioners and chiropractors, and those who consulted both general practitioners and specialists took longer to heal, on average, than patients who attended general practitioners only once or twice. Cassidy et al tested the feasibility of a province-wide rehabilitation scheme in another Saskatchewan cohort and observed that patients who enrolled in fitness therapy or a multidisciplinary outpatient

rehabilitation service within 120 days of their injury had slower recovery than those who accessed normal community treatment.**39** These results are confirmed by a recent Norwegian study which indicates that the likelihood of persistent neck pain after a whiplash injury is improved by early multidisciplinary assessment and advice.**40** Ultimately, the Dutch randomized study compared "education and counseling" by general practitioners (mean number of treatments = 3.9; mean length of treatment = 18.8 weeks) with "education and exercise" by physiotherapists (mean number of treatments = 12.7; mean period of treatment = 19.9 weeks) in patients with more than 4 weeks of WAD.**41** One year after the incident, patients reported lower levels of neck pain and headache severity in the general practitioner community than those seen more often by physiotherapists.**41** In view of an average of nine fewer visits per patient, the beneficial result of physician involvement resulted in summary, the epidemiological literature shows that the prognosis of whiplash accidents is correlated with the type and severity of clinical treatment. The latest body of research further shows that with education, exercises, mobilization, reassurance, pain management and motivation to regain their regular everyday life activities, acute whiplash injuries should be treated. In the early stages of WAD, health care professionals must be mindful of the risks of clinical iatrogenesis; the data tends to support the theory that "too much too early after the injury can delay recovery." Overtreating WAD patients is likely to encourage the production of chronic disease habits by emphasizing the use of passive coping behaviours.

Forces Acting on the Head and Neck during an MVA.

The literature includes two types of studies: those using anatomical preparations of the entire cadaver or cervical spine and those using live specimens exposed to rear-end impacts. The greater focus has been paid to rear-end crashes. Rear-end impacts cause the trunk and shoulders affected by the impact to accelerate forward. As the head is pushed backward in relation to T1, there is forced extension of the lower cervical spine.**42** With the upper spinal levels in flexion and the lower spinal levels in extension, the spine follows an S-curve. Before the stimulation of the paraspinal musculature, the sternocleidomastoid lengthens and becomes activated electromyographically.**43** The head then stretches, but less than maximally. Upon extension, the head is also accelerated forward, causing the entire neck to flex.**20** With the velocity of the impacting vehicle, the forces involved intensify. The result produces peak horizontal accelerations of about 4-5 g at speeds of 6-8 km/h, equivalent to plopping backward into a chair.1 This has been known as the baseline for moderate cervical strain.**19** However, 38 percent of participants subjected to controlled rear-end impacts experienced WAD effects at 8 km/h.**44** The head achieves a peak acceleration at speeds of 32 km/h (20 mph). The neck is subjected to shear forces parallel to the direction of impact during the rear-ended impact,**45,46** as well as friction, stress, flexion, and contraction at various cervical levels and at various stages of the event.**46** Cadaver arrangements exposed to rear-ended impacts reveal histological alterations on parts of cryomicrotome. These are lower components of the cervical spine which include ligament flavum stretch and break, anulus disruption, anterior longitudinal ligament rupture, and zygoapophysial joint fusion with capsular ligament tear.**47** Positioning the head and neck in forward bending or

contraction raises measured capsular facet strains.**48** Fractures of the facet joints' articular base, fractures of the articular pillar, avulsion/fractures of the endplate, and fractures of the vertebral body have been identified.**20**

Facet joints.

As a cause of neck pain following whiplash injuries, a lot of research points to the zygoapophyseal joints. The facet joints are heavily innervated and are a cause of pain alluded to. Given the whiplash process, the joints are at risk of overloading and injury. Fractures of the articular pillars after MVA have been seen in a multitude of tests. On regular radiographs, the fractures are not noticeable. There are also tears of the joint capsule and hemarthrosis of the joints.**20** Recent studies to determine the function of the zygoapophyseal joint in WAD has concentrated on diagnostic and therapeutic injections. Two separate local anesthetics and saline control were applied in random order under double-blind conditions at the level that appeared most symptomatic based on pain diagrams, and if this was unsuccessful, at another stage, in a randomized controlled sample using blocks of the medial branch of the cervical dorsal rami that supplied only the zygoapophysial joint.**49** Overall, 60% of the pain diagrams seemed to be most symptomatic. The prevalence of C2-C3 zygoapophyseal joint pain was 50 percent in patients with neck pain and headache of which headache was the main symptom. In an earlier report, the same researchers compared the relief obtained with a short-acting and longer-acting anesthetic. The study found that 54% of patients with chronic neck pain after MVA had injection pain relief lasting for the predicted time of operation of the anesthetic used. Interestingly, an additional 13 of 47 patients received pain relief that lasted significantly longer than the estimated length of the anesthetic.**50** This group of studies indicates that in a significant percentage of patients with chronic neck pain after MVA, the facet joints are a cause of pain. Referred pain from the joints, usually defined in WAD, has often been known as a cause of headache. It was hypothesized that the referred discomfort from the third occipital nerve alone which supplies the C2-C3 zygoapophyseal joint and the back of the brain, triggers headaches. Double-blind, managed blocks of the third occipital nerve were conducted by Lord et al.**51**; 53 percent of WAD patients whose main symptom was headache received satisfactory block relief depending on whether a short- or longer-acting anesthetic was used. A additional 7.5 percent of patients responded to the blocks, but the longer-acting agent was unable to differentiate.

Ligaments.

In post-mortem, animal, and cadaver tests, as well as in magnetic resonance imaging (MRI) and during surgery, ligamentous tears in the neck have been found. How much ligamentous trauma plays a role in moderate (less than grade 4) WAD, however is uncertain. Magnetic resonance

imaging should display severe ligament damage, but this has not been the case in groups of acute grades 1 to 3 WAD. The plain radiographs and MRI results of 100 patients without neurological damage examined within 3 weeks after acute whiplash injury were analyzed by Ronnen et al.**52**. Just one patient had a trauma-related pathological MRI, suggesting prevertebral edema. In the absence of MRI soft tissue damage, seventeen patients had kyphosis on a simple radiograph. The writers speculated that muscle spasms were responsible for kyphosis.**52** Other findings revealed that kyphosis is a common variant unrelated to trauma and muscle spasm.**53** One study of 40 WAD patients within 2 days of injury showed no patient with ligament, muscle, or other soft tissue injury,**54** and another study reached the same result of 39 patients imaged within 15 days of injury. Wilmink and Patijn**55** showed, though that the rating system commonly used to assess this injury is unable to accurately discriminate between controls and WAD patients. In WAD patients with recurrent discomfort and diminished ROM, Patijn et al.**56** used rotary computed tomography scans to test the alar ligament and concluded that no damage to this ligament was detected. With regard to the atlas, the authors observed decreased vertical translation of the skull and proposed that the WAD may rely on ligaments between the skull and C1, thereby understanding the symptom of decreased head extension seen in many WAD patients with no apparent structural injury.

TMJ and WAD

Clinicians have confirmed the coexistence of temporomandibular disorders (TMDs) and WAD and have suggested a mechanism. The postulated mechanism consists of sudden and unnecessary opening of the jaw as the head expands, resulting in the capsule of the temporomandibular joint (TMJ) widening and subsequent disk displacement. However, recent tests of human subjects in laboratory accidents do not indicate extreme jaw-opening forces in TMJs.**57,58** Thin, unregulated series show that patients presented with TMD symptoms following an MVA have a high frequency of TMJ-related MRI abnormalities.**59** However a controlled MRI analysis also found little distinction between 60 patients in TMJ disk displacement or effusion. There was no adequate analysis of the roles of anticipation, hypervigilance, and attribution of symptoms in linking typical symptoms to an MVA.**121**

Disc disease.

It is difficult to determine the occurrence of new disk damage in patients with WAD because of the high prevalence of disk disease in the asymptomatic population. There was not found any acceptable research on the role of disk injury. One research, however, sought to record this by evaluating 39 patients by MRI within 15 days and again at 2 years after injury. The survey was easy, and no control group existed. What disk abnormalities were associated with the injury could

not be determined, although only 3 of the 10 patients with disk disease in the original MRI showed signs that were consistent with the level of the disease. Original and follow-up imaging with greater sickness associated with a worse prognosis.**60**.

Nerve roots.

No publications that recorded the occurrence or prevalence of grade 3 WAD radiculopathy have been identified. These patients with paresthesia and symptoms of stiffness, heaviness or exhaustion in the upper limbs, unaccompanied by specific neurological results on clinical review, are the greatest diagnostic issue. Referred signs of segmental activation of connective tissue structures such as ligaments or facet joint capsules and reflex suppression of muscle contraction due to neck pain were postulated, in addition to nerve root, plexus, or peripheral nerve involvement.**20**

Muscles and Muscle Activation.

Many reports have recorded neck musculature injury after MVA, with the amount of damage being relative to the forces of the accident.**20** With whiplash grades 2 and 3, the exact prevalence of muscle injury is unknown. In general, patients that where studied with an MRI between 2 days and 3 weeks after injury did not exhibit signs of muscle damage.**52,54,60** It is also less clear whether muscle pathology leads to chronic symptoms. Two research to determine whether cervical muscle weakness occurs in chronic WAD were undertaken by Nederhand et al.**61**. In the first study, in patients with chronic WAD and normal controls, upper trapezius electromyographic (EMG) surface activity was compared. The WAD patients showed a typically reduced tendency to calm the muscle of the trapezius, prolonged muscle coactivation, and an almost double increase in activity following physical exercise provocation. In a follow-up analysis, 52 patients with chronic neck pain without a traumatic cause were associated with patients with WAD and there was little difference in muscle function between the classes. Another analysis of sternocleidomastoid activity in patients with chronic WAD showed relatively little activity, since unlike asymptomatic controls, most patients never achieved the stage of their ROM at which they could activate this muscle. It was proposed that apprehension of movement and reinjury contributes to protecting and hence to perpetuation of symptoms after the original injury has healed. Inside the area of low muscle function, participants tended to sustain their gestures. The writers speculated that this was due to avoidance of movement or injury, not muscle dysfunction.**62**

First 2 Weeks after Injury. Immobilization.

The Quebec study finds data showing that in non-injured subjects, soft collars do not limit the ROM. Delayed pain relief and ROM in WAD presenting within 4 days of injury were linked with soft collars in conjunction with prescription rest and analgesics. The clinical suggestion was that weak proof accepted restricting their use to brief periods of time. Gennis et al.**63** compared brief immobilization by a soft cervical collar with no immobilization during the initial whiplash injury management, following the QTF study. Subjects diagnosed with neck pain within 24 h of MVA were from a metropolitan level 1 trauma facility. The participants were randomized to a placebo group or soft-collar. For the first 2 weeks after injury, the soft-collar party was led to wear a Velcro fastened foam-rubber collar as much as they could handle. At the discretion of the prescribing practitioner, all classes were given analgesics and recommended to relax. Up to 6 weeks after injury, follow-up was done by telephone: 12 percent of the cohort either felt little different or felt worse at 6 weeks. With regard to recovery from symptoms, change, or worsening, there was no distinction between the classes. The study thus presented little evidence that when used acutely after whiplash injuries, wearing a soft cervical collar shortens or prolongs the length of neck pain.

Prescribed Rest versus Activity.

No research on the individual value of prescription rest in WAD was found in the QTF examination, but prescribed rest for 10-14 days was correlated with delayed recovery in conjunction with soft collars and analgesia in WAD.**64** The QTF analysis revealed that poor cumulative data indicated that extended rest periods was harmful to recovery. While no report on the independent effect of exercise was found, it was found that the prescription of home exercise coupled with guidance to indulge in exercises as accepted has short- and long-term benefit for WAD patients presenting within 4 days of injury. Borchgrevink et al.**65** analyzed the contrast between a 'act-as-usual' participant allowed to partake in regular pre-injury tasks and another group of participants granted time off from work and immobilized with a soft collar after the QTF review. Within 14 days of the MVA, patients were recruited from the emergency department and were assigned to one group. The same neck-training curriculum was offered to both classes and they were advised to execute the training from the first day. After 6 months, a blind observer compiled result measurements. A wide variety of conditions and other criteria is included in the tests, including the visual analog scale (VAS) for discomfort, pain during everyday life tasks, general change, and the amount of sick leave. Both groups progressed at 6 months; however, subjective signs and VAS of neck pain and headache showed a slightly better result for the act-as-usual group. Neck mobility and sick leave did not vary between the groups, indicating that the contrasting effects of the two groups may have played a part in psychological variables such as expectancy. 10 percent tended to have serious symptoms in the act-as-usual community. Rosenfeld et al.**66** contrasted early active mobilization with the normal therapy regimen and the impact of early initiation of treatment (within 96 h of MVA) with delayed onset (within 14 days). Primary treatment, emergency departments, and private hospitals were the subject of the report. The active group carried out soft, active, small-range rotational motions of the neck in two directions, repeated every waking hour 10 times in each direction. The typical procedure was an instructional leaflet,

instructions to rest the neck for a few weeks, and then to initiate vigorous neck and trunk movements two or three times a day. Cervical ROM and VAS at 6 months is included in the result variables. The active-treatment group gained more in pain relief, with the early-treatment group having a marginal benefit. Between the classes, the range of motion did not vary.

Steroids.

The QTF did not find any suitable trials of nasal, intrathecal, or epidural steroids. The effect of high-dose intravenous methylprednisolone compared with placebo was subsequently studied by Pettersson and Toolanen**67** in a total of 40 patients with grade 2 or 3 WAD within 8 h of MVA. Before administration of the drug or placebo, both classes were similar with respect to VAS for neck and radiating pain. The findings at 6 months were sufficiently serious for the occurrence of complications to preclude a return to work, the number of sick days before and after injury, and the sick-leave profile after injury. There was a substantial disparity between the groups that preferred methylprednisolone-treated subjects in terms of debilitating effects, overall number of sick days, and sick-leave profile. Research drawbacks include the limited number of participants (n 20) in each category and the absence of other outcome variables to be included. The number of sick days will not be specifically linked to a course in pathophysiology. In order to better test this intervention, larger confirmatory trials are required. Given the possible side effects of high-dose steroids, the potential cost-benefit balance is not evident for this treatment.

Treatment between 2 Weeks and 6 Months.

The workout. No appropriate experiments assessing the effect of exercise in the sub-acute period were found in the QTF review. Two research later indicated that WAD patients had head repositioning (kinesthetic sensitivity) defects.**68,69** Soderlund et al.**70** investigated whether kinesthetic sensitivity training increases the outcome of WAD and shows little effect of head position training. The participants were WAD patients referred within 20 days of an MVA from an emergency room. They have been randomized into one of two sections. Education and guidance on ROM movements, walking, postural sensitivity, and pacing were given to the first group and were recommended for the first few weeks to refrain from lifting or heavy carrying. The second category was applied to this regimen for kinesthetic awareness and synchronization exercises. Pressure ranking, injury, self-efficacy, coping, cervical ROM, and cervico-cephalic kinesthetic sensitivity were included as outcome factors. Self-efficacy is the self-reported confidence in through suffering, the tasks of everyday life. Before the start of therapy, results were assessed by these means, at 3 months and at 6 months. For kinesthetic awareness and synchronization tasks, no advantage over the regular curriculum was found. Subjects that remained asymptomatic after 6 months have greater initial self-sufficiency and self-reporting of injury attributable to pain (confidence in the ability to effectively complete everyday life tasks despite pain). In the sub-acute phase, active (as opposed to passive) care can result in better outcomes. A multimodal regimen of

supine relaxation preparation, counselling to relieve anxiety, manual massage and activation of the cervical spine and active exercises to reduce cervical lordosis were compared with passive ultrasound and transcutaneous electrical stimulation in 60 consecutive patients recruited on average 30 days after the MVA. Pressure ranking, ROM, self-rating of medication effectiveness, and return-to-work delay were the result indicators. At the 30- and 180-day follow-up, the range of motion did not vary, but the multimodal community returned to work faster and had less extreme discomfort and better control perception. While small groups were included in the research, it indicates that findings can be enhanced even if ROMs are not changed.

Treatment in the Chronic State.

Percutaneous Neurotomy with Radio Frequency. The QTF study has not found any research involving facet capsule deafferentation. There is now strong evidence that in patients with chronic WAD, facet innervation neurotomy can relieve pain. Lord et al.**71** associated several radio-frequency percutaneous lesions to the innervation of the facet joint with a placebo experiment in which the temperature of the probe was not elevated in 24 participants at an average of 34 months after MVA. With double-blind, placebo-controlled local anesthesia, the required facet joint was identified. In the active-treatment group, the median time for pain to return to 50% of the preoperative value was 263 days, compared with 8 days in the control group. At 27 weeks, 7 patients were pain-free in the active-treatment group and 1 patient in the monitoring group. Thus in some patients, the facet capsule is potentially a cause of chronic pain. When suffering is alleviated, psychiatric depression is more likely to be overcome in people with chronic WAD. Wallis et al.**72** found that after effective change in pain after percutaneous radiofrequency neurotomy, psychological discomfort in patients with WAD increased at 3-month follow-up relative to subjects receiving placebo blocks in which no lesion was generated. Using the SCL-90-R, psychological distress was assessed. Subjects endured symptoms for at least 3 months, with an average period of suffering of more than 24 months.

Exercise.

No research was found on the independent value of exercise in chronic WAD, as with the QTF study. Experience in people with chronic neck pain, not specifically associated with MVA, usually favours aggressive therapy rather than passive treatment.**73,74,75,76,77** Reduced symptoms are associated with reinforcing the cervical spine. The effect of a regimen requiring physical fitness, systematic encouragement of graded exercise, and therapy on patients with WAD (grade 1 or 2) for at least 6 months was studied in one unregulated clinical sequence of 6-month follow-up.**78** Change in somatic symptoms, psychiatric depression, and severity of pain was shown. None of the patients worked full-time at first; 65 percent of the subjects returned to work full-time at follow-up, and 92 percent returned to work at least part-time. Reasonable clinical trials with particular activities that evaluate the influence of exercise on pain as well as physical, psychological, and social activity would be needed to elucidate the impact of exercise in managing WAD.

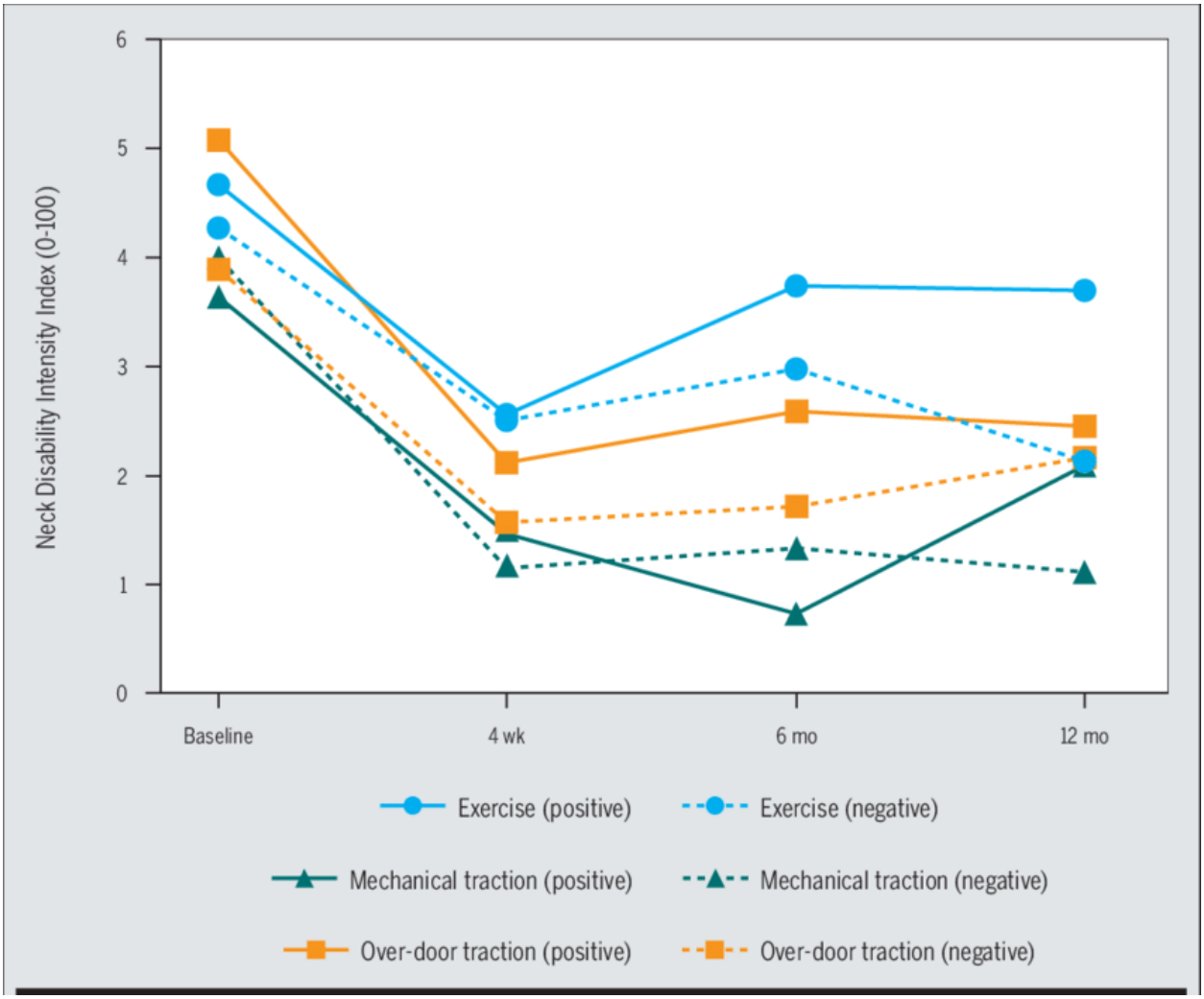
Prognosis.

A lack of high-quality epidemiological evidence on prognosis following whiplash injury was reported by the authors of the QTF report, and this inspired them to pursue a cohort analysis found in the report. The investigators were able to determine sociodemographic and collision-related prognostic variables for the length of benefits with the use of car insurance reports. A variety of subsequent studies have assessed prognostic factors and overall prognosis since the release of the QTF study. There has been a lack of consensus on various causes, and the general prognosis has been fiercely discussed in particular, with opponents arguing of virtually all research about methodological flaws. It should be noted that some reports conducted after the QTF report use cohorts of patients who are similar to those mentioned in the QTF analysis. Either a longer follow-up time or different findings than in the previous study are recorded in the follow-up studies. Therefore while in several trials, some prognostic variables can seem to be substantiated, this may reflect a continuation of previously published work. The QTF authors cited sources of subjects that may be ideal, acceptable, or of undesirable analytical consistency for evaluating prognosis in their analysis of prognostic factors. The investigators suggested that any people in a population who had sustained an acceleration-deceleration accident due to a motor vehicle crash would be the best source of subjects. No research followed the standards at the time of the study. Alternate sources of suitable topics include people obtaining medical attention or making claims for auto insurance and undertaking psychiatric review immediately after the MVA.

Clinical course of WAD and prognostic factors for recovery and non-recovery

Cohort reports have found that healing takes place within the first 2-3 months after the injury with a plateau in recovery after this point in time, if it does. **79,80** Even in people with poor overall recovery, there tends to be an initial reduction in symptoms to some degree in this early post-injury phase. Using trajectory simulation research, three different therapeutic recovery pathways after whiplash injuries were recently identified.**79** The first is a successful recovery route, where initial levels of pain-related impairment were mild to moderate and recovery was good, with 45% of individuals required to follow this pathway. The second pathway requires initial mild to extreme pain-related disabilities, with some rehabilitation but at 12 months with moderate stages of impairment. It is estimated that about 39% of wounded persons will follow this path. The third path includes initial injury combined with extreme pain and some rehabilitation to mild or severe impairment, with 16% of people expected that they will take this direction. They may provide valuable conceptualization of the alternative trajectories of healing for clinicians, distinguishing both people at risk of poor recovery and those who can recover better, with up to 50% of those suffering a whiplash injury experiencing residual pain and disability. This will continue to target people most in need of them with ever-shrinking health

services. Initially, higher levels of perceived pain and initially higher levels of impairment are the most consistent risk factors for poor recovery.**81,82** A new meta-analysis found that initial pain scores of >5.5 on a visual analog scale from 0 to 10 and scores of >29 percent on the Neck Disability Index are useful cut-off scores for therapeutic use. Other prognostic variables have been identified, including clinical variables with initial symptoms of mild post-traumatic stress, pain and symptoms of depressive mood.**81,83,84** In comparison, inadequate recovery has been found to predict poorer standards of recovery.**85,86** In other words, patients who do not plan to recover well do not eventually recover. At 12 months after damage, **86,87,35**, cold hyperalgesia has been shown to predict impairment and mental wellbeing effects and decreased cold pain resistance tested with the cold-pressor test projected ongoing disability.**88**



Predicted neck disability chart. NDI. Available at https://www.researchgate.net/figure/Adjusted-mean-Neck-Disability-Index-scores-AU.

A new systematic analysis found that moderate data is now available to endorse cold hyperalgesia as an adverse prognostic indicator.**26** Other sensory tests such as Walton et. al.**82** found that decreased pressure pain levels predicted neck pain-related impairment over a distal site in the leg at 3 months post-injury**89,** however other findings have indicated that this aspect is not an independent indicator of subsequent disability. The exact mechanisms influencing the hyperalgesic responses are not well known, but are widely accepted to indicate improved nocive responses. These considerations include motor and sensorimotor control tests such as the craniocervical flexion test, joint repositioning errors, and lack of balance.**90** Reduced range of neck motion is inconsistent with the fact that some studies have considered it to be predictive and others have not.**82** This is not to suggest that these factors should not be taken into consideration in the clinical assessment of WAD patients, but they should not be taken into account. Robust prognostic measures are not other variables generally known to determine outcome, such as those associated with settlement procedures and accident-related factors.**91** Similarly, demographic or social factors such as age, income and educational levels show unreliable prognostic capacity.**81,82** Step 1 or exploratory trials have been the majority of prognostic studies of WAD, with few confirmatory studies. In a recent study, a series of prognostic markers like initial disability, cold hyperalgesia, age and signs of post-traumatic stress were validated. The findings revealed that in discriminating patients with moderate/severe impairment from patients with complete rehabilitation or residual milder effects at 12 months post-injury, the collection showed strong precision (area under the curve 0.89, 95 percent CI 0.84 to 0.94).16 These results are clinically helpful, as physiotherapists typically strive to narrowly classify patients who are likely to report chronic moderate to severe symptoms in this field of science, such a confirmation analysis is unusual and goes some way towards having greater confidence in the use of these tests in the early assessment of whiplash injuries. A clinical prediction rule to classify both permanent moderate/severe impairment and complete recovery at 12 months post-injury was newly established on the basis of the findings of previous cohort studies. The findings revealed that an initial Neck Injury Index score of >40%, age >35 years, and a score of >6 on the hyperarousal subscale of the Post traumatic Stress Diagnostic Scale29 could predict reasonable sensitivity (43%, 95% CI 31 to 55), strong specificity (94%, 95% CI 89 to 96), and a positive predictive prediction in patients with moderate/severe disability at 12 months. A third medium-risk group will either rebound or develop chronic pain and impairment (>32% on the Neck Disability Index, score >3 on the hyperarousal subscale) at 12 months following injury, with a positive predictive benefit of 71%. A third medium-risk group will either recover or develop chronic pain and disability (>32% on the Neck Disability Index, score >3 on the hyperarousal subscale). The hyperarousal subscale contains five elements that measure the occurrence of symptoms, including: having trouble falling asleep, feelings of irritability, difficulties focusing, being excessively vigilant, and being quickly startled.**92**

Patient education and advice

Different information and instructional interventions were examined for their efficacy in improving outcomes following whiplash injury, including guidance booklets, websites and videos.**93** In one case, an activation-focused educational video of advice was more successful in

decreasing WAD symptoms than no therapy at 24 weeks follow-up (result: no/mild symptoms vs moderate/severe symptoms). Actually, there appears to be significant variety in the quality of knowledge and advice offered to a patient, indicating that there is yet to be identified the right educational interventions as well as methods for behavioural change and system reform.**94** While patients understandably want guidance on the prognosis and effects of their injury,**95** it is not obvious that advice can improve longer per se No educational intervention tests for chronic WAD have been published, but interventions that inform patients about pain neurophysiology have had some impact on other chronic pain disorders and may also be effective in WAD treatment.

Behavioural analysis of WAD patients

Functional behaviour analysis is described by Haynes and O'Brien.**96** as The detection of meaningful, controllable, causal functional interactions applicable to a particular range of target activities for an individual client. Therefore a study of functional behaviour is concerned with figuring out which factors influence a phenomenon and how they do so. In order to alter certain chains of events, it attempts to detect which of the related variables are antecedents and which ones are the results of the actions. The aim is also to recognize multiple individual problem patterns, and the change in these behaviours is then set as therapy goals.**97** It is likely to improve the provision of meaningful outcomes for learning new behaviours in modifying behaviour. Factors that improve behaviour should be recognized at an early level. **7,8** Choosing individualized and appropriate daily tasks so the patient can perform their exercises is a guide to effective recovery plans. In-between sessions, such home assignments improve the morale of the patient**98** and are also critical in generalizing therapy results. In cognitive behavioural analysis, these variables are established for potential use in care.

Psychological analysis of chronic WAD patients

Variations in the human use of coping mechanisms also illustrate individual variations in responding to prolonged discomfort. **99** Some studies categorize coping efforts into active and passive dimensions. An instrumental activity such as exercise to relieve pain also involves descriptions of successful techniques. In the other hand, passive techniques involve removing or giving up control over an external force, e.g. relaxing or taking medication.**100** Pain habits, particularly patterns of maladaptive movement, can result from attempting to avoid such.**101** Philips**102** concluded that people with chronic pain spent a lot of time attempting to control the severity of pain by restricting their actions and preventing interaction with any circumstance. Asmundson and associates**103** concluded in their study that avoidance activity is often linked to lower success of therapy, leads to injury, and that the fear of pain will change the way pain is coped with. Avoidance is perceived by these scholars to be a detrimental coping action. It is also closely related to techniques for passive, maladaptive coping. Earlier findings**104** show that self-efficacy and successful coping mechanisms are essential variables for patients with chronic whiplash-associated conditions in predicting physical and psychosocial well-being. Altmaier and

colleagues**105** indicated that self-efficacy increases were correlated with increased functioning of patients. The findings have indicated that the long-term benefits of therapy may be improved by increased expectations in self-efficacy. Patients should be taught to 'self-reinforce' and to assign performance to themselves, according to Harding and Williams **7**. It increases their power and influence. Instead of focusing on others, patients can also learn to provide themselves with signs and reminders on when to apply new behavioural skills. Related components of recovery are also closely connected to the application of active coping mechanisms. Numerous studies**104,106,107** have demonstrated promising correlations between the use of social and physical functioning and successful coping mechanisms. Examination of individual coping mechanisms and recognition of responses in risk conditions with increased pain are important aspects of treatment planning.**108** Turk and Salovey**108** argue that for people with chronic pain, problem solving capacities are an integral aspect of treatment. The perceived burden of the recovery facility, practical workout conditions, problem management skills, active coping mechanisms and risk situations for relapse evaluation are factors that improve the incentive for participants to continue to partake in the recommended adaptive habits after they have been released from the treatment program. Thus after encountering challenges with multiple tasks, the patient is taught to build strategies for adaptive action and adjust actions accordingly.**108** Because patients do not always use acquired behaviour and activity habits in their daily activities, the outcome of therapy can be frustrating. The remedy would be to use daily tasks systematically, as with the case of fitness, in order to sustain new habits.

Clinical log book **Date: 22.11.20.** **Observed** □ **Treated** □ **Yes.** **Patient front sheet included**? □ **No**

Number:	Sex: Male Occupation: Driver	Age 38	Area treated L forearm, L wrist, mid. TSP

Complainings
Neck pain and stiffness, worsening of pain with neck movement, loss of range of motion in the neck headaches, most often starting at the base of the skull, tenderness or pain in the shoulder and upper back, dizziness, pain on deep inhalation.
L wrist pain.

Hypothesis
1. Facet lock T4-T8
2. Spasm of the intercostal muscles
3. Scaphoid non-union fracture
4. Whiplash

Red Flags □No
What and Why? Patient was sent for an X-Ray of the rib cage and lungs by his GP. The results ruled out any damage or ongoing condition with the lungs.

Examinations to support hypothesis:
CSP ROM restricted and painful in L+R rotation, flexion, L lateral flexion.
TSP: Compression test, irritation test, thoracic mobility testing, static joint play.
L wrist: Palpation, compression test, tuning fork on scaphoid, static joint play.

Diagnosis:
Whiplash
Facet lock T4-T6.
Wrist joint irritation due to thickening of healed fractured scaphoid L.

Treatment and patient advice
STW: LSP, TSP, Traps, CSP, L forearm, L arm, L Deltoid.
TENS: TSP, L forearm.
Dry needling: T3-T7 BL, L forearm, Traps BL, OA BL, DLJ BL, L5-S1 BL.
HVT: T4-T6 R, OA R, C3-C6 L.
Mobilizations: L shoulder, TSP, CSP.
MET: TSP, CSP, L wrist
Advice: Heat on TSP, ICE on L wrist. Assessment in 7 days time.

Ongoing treatment progression Continue with DTW on TSP and CSP. DTW on L wrist flexors/extensors for the next four treatments. Gradually increase myofascial work and cross friction on L wrist to brake down adhesions. By TTT 4, introduce stretching (flexion, extension, side bending BL) and light mobilization exercises for the wrist. Home advice: Rest from activities that aggravates the symptoms. Heat on TSP, NSAID's for the wrist (prescribed by his GP). Cat stretch, side lying rotations for the TSP.

Clinical log of patient presenting with pain in CSP, thoracic region and left wrist, secondary to MVA on his motorcycle in May 2020.

Medications

While not traditionally for physiotherapy, physiotherapists often prescribe over-the-counter drugs to patients or contact the General Practitioner of the patient concerning the need for medicine. It would seem sensible for acute WAD that the availability of pain relief in the early stages would be necessary, as with any acute injury or trauma,**109** particularly provided that initial higher pain thresholds are associated with poor recovery from whiplash injury and that features suggestive of central hyperexcitability are typical. Yet only few drug trials have been conducted in acute WAD. An early research found that intravenous methylprednisolone infusion administered for acute whiplash in a hospital emergency room resulted in less sick days over 6 months and less pain-related injury than those taking placebo medication.**110** While this is a fascinating discovery, it would not be possible in primary care environments and may have potentially adverse effects.**21** In a recent study, This compares with other disorders, such as low back pain and fibromyalgia, the latter of which is close to chronic WAD in sensory perception. Current clinical recommendations propose, by consensus, that general guidelines for pain management be adopted for the administration of treatment to patients with acute and chronic WAD**21** before additional data is available.

Interdisciplinary approaches

There are actually few suitable therapies for acute WAD available, as can be seen by the data discussed above. One explanation indicated for this is because a one size fits all' strategy has been used and this is sub-optimal because it lacks WAD's well-documented heterogeneity.**111,112,113,114** There are now several evidence suggesting that other variables found to be present in acute WAD and correlated with weak recovery may need to be taken into consideration in the early treatment of the disorder. These include, in particular, the sensory appearance of WAD, enabling some comprehension of the involved nociceptive mechanisms, and psychological influences that can hinder recovery. A new high-quality randomized study examined whether improved performance than normal treatment would be given by the early targeting of these causes. Measures of pain, disability, sensory control and

psychiatric causes, including general anxiety and post-traumatic stress symptoms, were used to examine participants with acute WAD (about 4 weeks duration). Care was adapted to the outcomes of this baseline assessment which may range from a multimodal approach to physiotherapy for guidance, exercise and physical therapy for those with few symptoms of central hyperexcitability and psychiatric trauma to an interdisciplinary operation comprising of drugs (if pain thresholds were greater than moderate and there were signs of central hyperexcitability if scores on psychological questionnaires were above threshold). This approach to pragmatic intervention was opposed to usual care in which the patient might get medication as they usually would. Research revealed no substantial variations in rehabilitation frequency (defined as validity of the Neck Injury Index, this strategy can be evaluated in future studies. In addition, 61 percent of study participants considered the medication (low-dose opioids and/or adjuvant agents) undesirable due to side effects such as dizziness and somnolence, and did not comply with the recommended dose, Compliance with conducting meetings with the clinical nurse was less than compliance with physiotherapy, possibly reflecting patient preference with physiotherapy. It can be expected that management requiring only physical therapy for chronic WAD would not be appropriate in conjunction with the biopsychosocial model of chronic pain. In a persistent WAD population, few studies with interdisciplinary therapies have been performed, and these approaches have been varied, from physiotherapists providing psychological-type treatments to psychological interventions only in addition to physiotherapy. Teasell et al**37** concluded in their systematic analysis that while most findings show that interdisciplinary approaches are successful, the complexity of the interventions makes it impossible to draw conclusions. Since that study, subsequent studies have explored persistent WAD psychological methods. Dunne and colleagues**115** found that cognitive behavioural trauma-focused treatment provided to people with chronic WAD and posttraumatic stress disorder resulted in reduced psychiatric effects of post-traumatic stress disorder, anxiety and depression, as well as decreased injury associated with pain. The findings of this research, while tentative, indicate that psychiatric therapies can be effective not only for improving psychological problems, but also for improving pain-related impairment. Some people with WAD will experience different psychological symptoms, especially those with an already chronic disorder, from a therapeutic viewpoint. For example, pain catastrophisation, pain-induced anxiety, pain coping mechanisms and other effects related to the stressful experience itself (road traffic crash), such as symptoms of post-traumatic stress or post-traumatic stress disorder, can be correlated with psychiatric symptoms. There is also emerging evidence that there might also be feelings of wrongdoing involved with the injury or settlement scheme**116**. In the clinical evaluation of patients with WAD, certain considerations will need to be assessed. If confident, as part of their recovery plan or to initiate effective referral, the physiotherapist may then decide to administer them. This may be for further assessment of the psychiatric effects by the general physician of the patient or a clinical psychologist. The decision to refer or not will be taken by means of appropriate questionnaires, with high scores suggesting that referral could be required and physiotherapy care that is psychologically aware with more mild scores, but with reassessment and referral if there is little change.

Future directions (Conclusion)

The detection of patients at risk of poor recovery and then preventing the progression of chronic pain and injury is an important goal for the treatment of acute WAD. There is still a lack of data available to direct the clinician to accomplish this purpose, and for physicians and scholars alike, this is challenging. Although the features of the disease and causes indicative of poor rehabilitation are now much better known, much less progress has been made in designing improved and successful interventions. The reasonable next step in the study process is to target, with more precise approaches, these variables, all of which are theoretically modifiable. Return to operation and exercise instruction and guidance will also be the cornerstones of early care for WAD, but additional research is required to evaluate the most appropriate type of exercise, dosage, and methods of providing these interventions. For patients at low risk of experiencing chronic pain, movement and exercise are likely to be appropriate, but this is yet to be formally checked. In the basic advice/activity/exercise strategy, those patients at low to high risk of poor rehabilitation will likely require additional therapies. This can include medications that target inflammation and nociceptive treatments, as well as approaches to help with early psychological damage reactions. This is not so easy to do as was seen in the aforementioned interdisciplinary analysis for acute WAD.**117** Not only did the subjects of this study find the side effects of treatment intolerable, but they were much less consistent with a clinical psychologist's participation (46 percent of participants attended less than 4 of 10 sessions) compared to the physiotherapist's attendance (12 percent attended fewer than four sessions over 10 weeks). Individuals with acute whiplash injuries may see themselves as experiencing a 'physical injury and are thus more likely to accept physiotherapy. The pressure of having multiple professionals to visit can often result in inadequate enforcement. The health care professionals better equipped to provide psychological treatments for acute WAD could be physiotherapists. In primarily chronic disorders such as arthritis,**118** this method has been studied, and recently in the treatment of acute low back pain,**119** with findings showing some early promise. This is not to suggest that physiotherapists can treat individuals with a diagnosed psychopathology, such as depression or post-traumatic stress disorder, because of course, certain patients will need referral to an adequately trained specialist. In the overall treatment plan of a patient with acute WAD, physiotherapists will also need to play a larger role. This would mean providing experience and an appreciation of what additional therapies such as medicine and psychiatric interventions are needed in the estimation of risk factors. While this has historically been the task of general practitioners, it is difficult to see how the busy primary care structure can allow patients to be adequately identified in order to first classify those at risk, establish a recovery strategy, monitor the progress of the patient, and adjust treatment if appropriate. More successful approaches require production and research in the case of chronic WAD. It is increasingly apparent that only limited impact sizes are reached through management methods that rely mainly on physical therapy. It is necessary, however to perform physical operation and exercise for patients' long-term general wellbeing, and it is a problem if chronic pain prohibits them from doing so. It includes randomized clinical trials that incorporate approaches to activity/exercise with other strategies such as therapeutic approaches, approaches to schooling, and medicine. It would be important to decide the optimum mixture and dosage of such approaches. A daunting and dynamic disorder is WAD, whether acute or chronic. With strong data arising of a multitude of physical and psychological causes that

exist to differing degrees in particular individuals, it is also clear that specialized expertise in this field are required by clinicians engaged in WAD management. Physiotherapists are the health care professionals who are expected to see the highest number of patients with WAD and expend the most time with these patients due to the set-up of the health system. Physiotherapists are well positioned to play a position of coordination or 'gatekeeper' in WAD management and study is therefore needed on health services models that involve physiotherapists in such a role.

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