

Whiplash-associated disorders and temporomandibular symptoms following motor-vehicle collisions

Joel B. Epstein, DMD, MSD¹/Gary D. Klasser, DMD²

Recent research has shown that temporomandibular symptoms may be associated with or occur independently of whiplash-associated disorders related to motor-vehicle collisions. A PubMed/Medline search was conducted using the terms “temporomandibular disorders,” “orofacial pain,” “temporomandibular joint,” “whiplash,” and “whiplash-associated disorders and motor-vehicle accidents and motor-vehicle collisions” for the years 1995 to 2009. Systematic reviews, meta-analyses, and clinical studies were included if they addressed temporomandibular disorders, whiplash epidemiology, diagnosis, and prognosis. References in the selected articles were also reviewed (including those prior to 1995) if the articles specifically addressed the topic. An evidence base was established for general outcomes using the Oxford Centre for Evidence-Based Medicine Levels of Evidence. Temporomandibular symptoms may develop following motor-vehicle collisions and be more complex, representing a component of a symptom cluster of potentially regional and widespread pain impacted by psychosocial factors. Oral health care providers must be aware of the relationship between temporomandibular symptoms, whiplash-associated disorders, and trauma and the more complex nature of the symptoms for appropriate diagnosis and management. (*Quintessence Int* 2011;42:e1–e14)

Key words: motor-vehicle collisions, temporomandibular symptoms and disorders, whiplash-associated disorders

The association between temporomandibular symptoms following motor-vehicle collisions and those specifically associated with whiplash injury has been controversial. Whiplash-associated disorders are commonly associated with motor-vehicle collisions, usually when a vehicle is rear-ended. Classification of whiplash-associated disorders is shown in Table 1. The Quebec Task Force on Whiplash-Associated Disorders defined whiplash as an acceleration-deceleration mechanism of energy transfer to the neck resulting from a rear-end or side-impact motor-vehicle collision but that may

also occur during diving or other mishaps. It also indicated the impact may result in bony or soft tissue injuries, which in turn may lead to a variety of clinical manifestations such as whiplash-associated disorders and include reduced or painful jaw movement.³ These symptoms are associated with temporomandibular disorders (TMDs) and may present with jaw pain and/or dysfunction in addition to headache, dizziness, hearing disturbances, and neck pain or dysfunction following motor-vehicle collisions.^{4–12} Furthermore, TMDs may present without whiplash-associated disorders as sole independent manifestations due to motor-vehicle collisions. The superimposition of these confounding conditions (whiplash-associated disorders and TMDs) adds to the difficulty in diagnosis and management. The purpose of this focused narrative review is to provide an update, based upon recent literature, on TMDs associated with or independent of whiplash-associated disorders

¹Professor, Department of Oral Medicine and Diagnostic Sciences, College of Dentistry; and Department of Otolaryngology and Head and Neck Surgery, College of Medicine, University of Illinois at Chicago, Chicago, Illinois, USA.

²Assistant Professor, Department of Oral Medicine and Diagnostic Sciences, College of Dentistry, University of Illinois at Chicago, Chicago, Illinois, USA.

Correspondence: Dr Joel B. Epstein, 801 South Paulina Street, Chicago, IL 60612. Email: jepstein@uic.edu

Table 1 Clinical classification of whiplash-associated disorders ^{1,2}		
Grade	Clinical presentation*	Subgrade
0	No complaint, no physical signs	
1	Neck pain, stiffness, or tenderness	
2	Neck complaint and musculoskeletal signs: decreased range of movement and point tenderness	2A: point tenderness, normal range of movement 2B: point tenderness, abnormal range of movement
3	Neck complaint and neurologic signs: decreased or absent deep tendon reflexes, weakness, or sensory deficits	
4	Neck complaint and fracture or dislocation	

*Deafness, dizziness, tinnitus, headache, memory loss, dysphagia, and temporomandibular pain can appear in all grades.

resulting from motor-vehicle collisions. Oral health care providers who have patients presenting with TMDs following motor-vehicle collision-based trauma should be aware of the etiology, prognosis, and general principles of patient evaluation and management, as many of these cases represent a regional and widespread pain condition. By understanding the complexity of issues associated with certain individuals following motor-vehicle collisions, the clinician should be better able to provide appropriate management.

STUDY SELECTION

A PubMed/Medline literature search was conducted using the terms “temporomandibular disorders,” “orofacial pain,” “temporomandibular joint,” “whiplash,” and “whiplash-associated disorders and motor-vehicle accidents and motor-vehicle collisions” (between 1995 and 2009) from English-language peer-reviewed journals. The review of articles was limited to the

time frame stated because 1995 is when the Quebec Task Force monograph was published, providing the first standardized definition of whiplash-associated disorders. Furthermore, it should be noted that the Quebec Task Force was unable to find any published article of methodological rigor on this topic published before 1993.³ Systematic reviews, meta-analyses, and clinical studies were included if they addressed TMDs, whiplash epidemiology, diagnosis, and prognosis. In addition, references in the articles selected according to our criteria were also reviewed (including those prior to 1995) if the articles specifically addressed the aforementioned four factors. An evidence base was established for general outcomes using the Oxford Centre for Evidence-Based Medicine Levels of Evidence. In this system, grade A is the highest level, based upon consistent evidence for Level 1 studies; grade B is evidence from Level 2 or 3 studies or extrapolation from Level 1 studies; and grade C is from Level 4 studies or extrapolation from Level 2 and 3 studies.



EVIDENCE FOR THE PREVALENCE AND INCIDENCE OF WHIPLASH-ASSOCIATED DISORDERS AND TMDs

Whiplash-associated disorders following motor-vehicle collisions are estimated to occur in one-third of all collisions and are associated primarily with rear-end collisions.^{13,14} Whiplash-associated disorders are the most common motor-vehicle injury treated in emergency rooms in the United States.^{15,16} An incidence of 328 visits per 100,000 people were reported in the United States, and in Canada, whiplash-associated disorders represented 83% of accident claims with an annual incidence of 67 visits per 100,000 people.^{17,18} In a Belgian study, 400 consecutive TMD patients were assessed for jaw injury, and 24.5% of these patients were found to have had an onset of pain and dysfunction linked directly to trauma of mainly whiplash-accident origin.¹⁹

The prevalence of TMD symptoms associated with whiplash-associated disorders was assessed in a population survey completed at 6 weeks and 4, 8, and 12 months following the collision.¹⁶ Overall, the results indicated that females had more complaints prior to and following collisions. A total of 8,109 claims were assessed, and 16.4% reported TMDs. Onset of new TMDs after motor-vehicle collisions was seen in 14.9% (11.6% male, 17.2% female), while 4.3% reported TMDs prior to the motor-vehicle collision. TMDs after collisions were more common in patients with whiplash-associated disorders (17.4% [13.2% male, 20.0% female]) and were reported in 4.7% without whiplash-associated disorders. The relative risk of TMDs after motor-vehicle collisions was 3.35 higher in those with whiplash-associated disorders. Females had a more than 50% increased risk of TMDs following motor-vehicle collisions. Those older than 50 years of age had a 35% increased risk of TMDs. Those who recalled hitting their head were 40% more likely to report new jaw pain following the collision. Patients who reported dysphagia after the injury had a 3.75 relative risk

of TMDs, a relative risk of 2.0 of ringing in the ears, and a two fold increased risk of TMDs associated with visual change, numb arms or hands, dizziness, nausea, increased headache, and neck pain. Reports of accident parameters (direction of impact, headrest involvement, seatbelt use, whether the car was drivable after the accident) were not associated with risk of TMDs. Assessment of recovery after the accident was hampered by 44% participation of the 1,128 patients with new symptoms of TMDs in whom the follow-up survey found recovery from TMDs in a median of 120 days; 70% reported no pain by 4 months, and 78% reported no pain by 1 year.¹⁶

Similar to the above study, 40 consecutive patients with whiplash-associated disorders were compared to 40 control subjects. The former patients experienced more frequent temporomandibular joint (TMJ) pain ($P < .001$), limited jaw opening ($P < .01$) and muscle tenderness ($P > .01$).²⁰ However, the presence of joint sounds, deviation during mouth opening, and the overall presence of a symptom were not significantly different between groups. Another study assessed 93 patients with whiplash-associated disorders and identified TMDs in 55 of these (59%); yet, 27 (29%) of those diagnosed with whiplash-associated disorders were not diagnosed with TMDs.²¹ In a recent narrative literature review of 32 articles that assessed the possible relationship between TMDs and whiplash-associated disorders, it was concluded that a low to moderate incidence and prevalence of TMDs were associated with whiplash.²² Based upon the data, the level of evidence assigned for the prevalence of whiplash-associated disorders and TMDs due to motor-vehicle collisions is grade A.

EVIDENCE FOR THE DELAYED DIAGNOSIS AND RECOGNITION OF TMDs

In a prospective study,²³ 60 consecutive whiplash-associated disorder patients following car accidents were compared to controls in a country in which there is a

lack of financial compensation from this type of injury. Patients were examined in the emergency room and completed a structured questionnaire. These patients had neck complaints with or without muscle findings, did not have cervical fracture (whiplash-associated disorders grades 1 to 3, see Table 1), and had no loss of consciousness. Differences in jaw-related symptoms or function were seen in patients versus controls at first examination following motor-vehicle collisions (12% vs < 5%; $P = .048$), and at 1 year follow-up, TMDs were identified in 33% vs 5% in controls ($P = .004$). New symptoms of TMDs were five times higher in subjects than controls and higher in females. Fifty-nine patients were assessed at 1 year (37 female, 22 male), and 19 patients had TMD symptoms compared to six controls ($P = .04$). TMDs were reported as the primary complaint in 5% at the first visit and in 19% ($P = .04$) at 1 year follow-up with no significant increase seen in controls at 1 year. These findings may reflect progression in TMDs and/or improvement in other symptoms (excluding TMDs) associated with whiplash-associated disorders.

TMJ pain began with trauma in 7%, which was significantly different from the controls ($P = .048$), and increased during follow-up ($P = .008$) and as compared to controls ($P = .04$). Delayed onset of new symptoms of TMDs was seen in approximately 33% of whiplash-associated disorder patients with TMDs vs 7% of controls ($P < .001$, odds ratio ~ 7). Twenty percent of whiplash-associated disorder patients reported TMDs as their primary complaint after 1 year ($P = .04$). Painful clicking developed during follow-up in 19% of whiplash-associated disorder patients, and painful locking developed in 14% with only one patient (2%) having TMD symptoms prior to the accident. Treatment for TMDs was not provided to study patients or controls during follow-up, although 12% of patients who had been involved in motor-vehicle collisions spontaneously requested treatment. This indicates a significant risk for delayed onset of TMDs following whiplash trauma, making this an important consideration for patient evaluation, diagnosis, prognosis, management, and medicolegal issues.²³

The evidence for delayed onset and/or delayed recognition of TMDs as a result of motor-vehicle collisions is grade B.

EVIDENCE FOR REGIONAL AND WIDESPREAD SYMPTOMS ASSOCIATED WITH WHIPLASH-ASSOCIATED DISORDERS AND TMDS

The literature reports regional and widespread symptoms associated with whiplash-associated disorders, including hearing and vestibular complaints. A recent study assessed 20 whiplash-associated disorder patients, 20 controls and 20 patients with acoustic neuroma. Differences in eye-movement control, postural stability, smooth pursuit neck torsion test, and dizziness were seen in whiplash-associated disorder patients, acoustic neuroma patients, and controls. A dizziness handicap was similar in whiplash-associated disorder and acoustic neuroma patients.²⁴ Mild traumatic brain injury has also been documented in patients experiencing direct impact in addition to a possible or documented loss of consciousness.¹⁵

Grushka et al²⁵ compared 54 post-motor-vehicle collision patients to 82 nontrauma TMD patients (control group). Post-motor-vehicle collision patients reported more orofacial pain complaints than nontrauma TMD patients who more commonly reported jaw joint sounds at the first clinical visit. Statistically significant differences were seen between post-motor-vehicle collision patients and controls in earache (62% vs 44%), ear stuffiness (48% vs 27%), neck and shoulder complaints (94% vs 62%), backache (77% vs 42%), numbness or pain in extremities (68% vs 23%), headache (91% vs 69%), jaw pain on waking (84% vs 66%), facial pain (93% vs 69%), poor sleep (86% vs 56%), dizziness (73% vs 25%), and stress (85% vs 50%) (all $P < .05$). Therefore, TMDs post-motor-vehicle collisions were more commonly associated with regional pain in the head, neck, and shoulders, and ear complaints, sleep disorders, and increased stress were more common, thereby confirming a more widespread syndrome consistent with regional and central mechanisms. Widespread pain may nega-

tively impact prognosis and complicate management approaches.

In a study¹⁰ involving 7,462 patients who met criteria for whiplash-associated disorders, of whom 45% completed follow-up surveys, it was found that symptoms commonly reported following motor-vehicle collisions included neck and back pain, fatigue, dizziness, extremity tingling, tinnitus, cognitive problems, paresthesia, headache, memory and cognition complaints, spinal pain, nausea, and jaw pain. These common widespread and diverse symptoms present a cluster of physical symptoms, suggesting a regional and widespread systemic disorder and/or possible central changes that negatively impact prognosis.¹⁰ A supportive study assessed experimental pain in 12 whiplash-associated disorder patients and controls using intramuscular electrical stimulation. The repeated electrical stimulation resulted in muscle pain in both groups with increased sensitivity to stimulation and larger areas of referred pain in whiplash-associated disorder patients, suggesting altered nociceptive input and central processing in whiplash-associated disorders.²⁶ In contrast, a prospective but uncontrolled study of 155 patients assessed by telephone interview reported only one case of TMD symptoms at 1 year.¹²

From these reported clinical and experimental studies, grade A evidence was assigned for regional and widespread symptoms in relation to symptoms associated to whiplash-associated disorders and TMDs following motor-vehicle collisions.

EVIDENCE FOR PSYCHOLOGIC SYMPTOMS ASSOCIATED WITH WHIPLASH-ASSOCIATED DISORDERS AND TMDs

Assessment of depressive symptoms following whiplash-associated disorders was conducted in 5,211 subjects.²⁷ New symptoms of depression were reported in 42% of subjects after 6 weeks and in an additional 18% at 1 year. Preinjury mental health problems increased the risk of postinjury depression. Emotional functioning following mild trau-

matic brain injury was found to be associated with significantly increased symptom complaints and global distress compared to controls.²⁸ Fifty consecutive patients with whiplash were assessed within 1 week of injury, at 3 months, and at 2 years and showed that somatization, insomnia, anxiety, and depression became abnormal in 81% of patients after 3 months, remained abnormal in 69% at 2 years, and were elevated in patients with more severe continuing complaints.²⁹ The clinical status of patients at 2 years was predicted by psychologic scores and neck symptoms at 3 months, and psychologic changes become established by 3 months. These findings suggest psychologic changes (including depression) may be part of a whiplash-associated disorder symptom cluster.

Regional and central mechanisms, including sleep disorder, and psychologic changes may occur as a result of persisting whiplash-associated disorder and is associated with orofacial pain and symptoms of TMD. Therefore, a grade A level of evidence was attributed to the presentation of psychologic symptoms associated with whiplash-associated disorders and TMDs.

EVIDENCE FOR THE PRINCIPLES OF MANAGEMENT OF WHIPLASH-ASSOCIATED DISORDERS AND TMDs

The approach to management of TMDs following motor-vehicle collisions must be consistent with management of the aforementioned broader symptoms. Therefore, literature that discusses management of whiplash-associated disorders and the more limited studies on TMD management after motor-vehicle collisions were reviewed.

The general principles of physical medicine, physical therapy, and directed medications for musculoskeletal pain and chronic pain were discussed.⁴ A population-based survey of a random sample of 2,000 people from the general adult population without whiplash-associated disorder was conducted by mail.³⁰ Pain associated with whiplash-associated disorders was more

negative than other noncollision-caused pain ($P < .017$) with 55% of whiplash-associated disorder patients reporting active coping (activity and exercise) strategies as being important for recovery. Whiplash-associated disorder patients reported greater pessimism regarding return to usual activities. Approximately 40% felt symptoms would not improve quickly, with fewer whiplash-associated disorder patients (18%) vs non-whiplash-associated disorder patients (32%) expecting symptom resolution ($P = .006$). The belief of greater difficulty and less probability of improvement with whiplash-associated disorders may affect management outcome.³⁰

A review of randomized clinical trials of treatment of adults with whiplash-associated disorder was published.³¹ Thirty-six trials were identified reviewing oral nonsteroidal anti-inflammatory drugs (NSAIDs), centrally acting psychotropic agents, steroids, and anesthetic agents. For acute whiplash-associated disorders, prednisone administered within hours of injury reduced pain at 1 week, but did not affect pain at 6 months vs placebo. For chronic symptoms, intramuscular lidocaine was superior to placebo and dry needling and similar to ultrasound. Muscle relaxants and analgesics had limited evidence of effect. Myofascial trigger point injection was found effective, but no difference using saline or botulinum toxin as the active agent was reported. A prospective, controlled study of 37 whiplash-associated disorders patients suggested that botulinum toxin led to improved functional quality of life.³²

In a study³³ of 29 whiplash-associated disorder subjects assessed at baseline and then randomized to treatment for vestibular rehabilitation or nontreatment, it was reported that the intervention group had statistically significant improvement in physical and questionnaire assessments. A study by Klobas et al²¹ involved 94 consecutive patients with whiplash-related conditions, of which 55 had TMDs. These patients were randomly assigned to a jaw exercise group ($n = 25$) or no treatment ($n = 30$), and no differences in signs and symptoms of TMDs were noted at 3- and 6-month follow-up.

The limited studies regarding the management of TMD symptoms following motor-

vehicle collisions resulted in a grade B level of evidence. The approach to management must be consistent with management of the broader symptoms. Prospective, randomized controlled trials of adequate subject number using appropriate measurement methodology are needed to enhance knowledge regarding management approaches.

EVIDENCE FOR THE PROGNOSIS OF WHIPLASH-ASSOCIATED DISORDERS AND ASSOCIATED TMD SYMPTOMS

An understanding of the prognosis of whiplash-associated disorders is complex because TMD symptoms may be associated with whiplash-associated disorders or present as an independent manifestation as a result of motor-vehicle collisions. Regardless, both whiplash-associated disorders and TMDs share a number of common physical and psychologic features (Table 2) that greatly influence, either alone or in combination, the prognosis of these disorders.

Approximately 15% to 40% of patients with acute whiplash-associated disorders that may include TMDs develop chronic symptoms.³⁴⁻³⁶ A recent meta-analysis utilizing 14 studies of 11 patient cohorts that assessed persisting complaints following whiplash injury identified several risk factors with strong evidence of predicting pain after 6 months.³⁷ The risk factors identified included high baseline neck pain intensity, presence of headache, high neck disability (whiplash-associated disorders grade 2 or 3; see Table 1), and no postsecondary education. Moderate evidence of risk was identified with catastrophizing, presence of neck pain, no seatbelt used, neck pain prior to accident, and being female.³⁷ Another systematic review found that pain and disability due to whiplash-associated disorders decreased rapidly in the first 3 months following motor-vehicle collisions, followed by little further improvement.¹⁸ The prognosis was affected by high initial pain, high whiplash-associated disorder score, anxiety and

Table 2 Physical and psychologic features of whiplash-associated disorders and TMDs

Regional	Physical		Psychologic	
		Widespread	Affective-motivational	Cognitive-behavioral
Musculoskeletal disorders		Central sensitization	Anxiety	Memory
Neuropathy		Traumatic brain injury	Depression	Behavior
Inflammation: proinflammatory cytokines		Spinal cord and brain stem injury	Worry	Sleep disturbances or dysfunction
Regional pain syndrome		Proinflammatory cytokines, pain sensitizers, or pain mediators	Catastrophic thinking	Deconditioning or dysfunction
Temporal and spatial summation		Temporal and spatial summation	Low self-expectations	—
Pain sensitizers or pain mediators		Pain sensitizers or pain mediators	Fear	—
—		Sleep disturbances or dysfunction	Frustration	—
—		Fatigue	Fatigue	—

depression, and being female. An earlier systematic review found age, sex, baseline neck pain and headache intensity, and the insurance compensation system to be predictive of recovery.¹ A Delphi (consensus by a panel of experts) survey to identify factors that may predict chronic pain and disability due to whiplash-associated disorder reported that risk factors for chronic pain included a history of chronic pain, physical factors (such as severe injury), and psychologic factors (pain-causing fear, avoidance of exercise, tendency to somatize, catastrophic thinking, low self-expectations, and symptoms of post-traumatic stress disorder).³⁸ In another study, patients who reported higher initial pain following motor-vehicle collisions also reported an increase in health care utilization.³⁹

It has been hypothesized that females may be at increased risk of whiplash-associated disorders and TMDs due to relatively less neck mass than males⁴⁰; however, after reviewing information from a large database, no relationship with body mass index was identified.⁴⁰

A prospective study of 76 patients with acute whiplash-associated disorders investigated features that predicted pain and disability at 6 months after motor-vehicle collisions.⁴¹ Greater physical symptoms

(including loss of neck movement), old age, hyperalgesia as determined by quantitative sensory testing, and posttraumatic stress predicted persistent symptoms. These findings show that both physical and psychologic factors play a role in recovery from acute whiplash-associated disorders.⁴¹ A prospective study⁴² designed to identify prognostic factors for whiplash-associated disorders up to 12 months after motor-vehicle collisions enrolled 125 patients who had mild to moderate whiplash-associated disorders persisting 2 weeks post-motor-vehicle collision. Interestingly, 64% of the patients recovered after 1 year. Neck pain intensity and work disability were the most consistent predictors for prognosis. Poor recovery was more common in females; those with low education levels; and patients with high initial pain reports, severe disability, high somatization, and sleep disorders.⁴² A systematic review of the literature that involved 50 articles reporting on 29 cohorts to assess recovery from whiplash-associated disorders reported that high initial pain intensity predicted prognosis, while older females with increased acute psychologic response involved in rear-end collisions who were compensated were not associated with adverse prognosis. Limited impact upon recovery was seen in those



with restricted range of motion, a high number of complaints, and prior psychological problems.⁴³

Chronic posttraumatic headache is reported in 30% to 90% of people following mild head injury with approximately one-third of those who experienced head injuries reporting headaches after 6 months and one-quarter after 4 years.⁴⁴ Many of these head injuries may have been the result of motor-vehicle collisions; however, the incidence of head injury is difficult to assess, as the majority of head injuries are mild and not reported; also, oftentimes, those that are reported are from heterogeneous populations. Regardless of the etiology, it has been reported that chronic posttraumatic headache and neck pain are influenced and perpetuated by physical, social, cultural, and psychological/emotional and cognitive factors. Furthermore, high levels of initial pain were found to predict delayed recovery.⁴⁴

Other prognostic indicators of whiplash-associated disorders that have been identified are related to neck pain 1 year after injury (reported by 50% of patients) with greater initial pain, increased number of symptoms, and greater initial disability predictive of slower recovery. Additionally, the direction of collision and headrest type may be prognostic, and coping style, depressed mood, and fear of movement were also associated with slow or limited recovery.⁴⁵ At a 1-year follow-up, it was reported that baseline stress response and initial pain severity were associated with increased risk of persistent pain, neck disability, and poorer self-report of general health, and acute stress reaction was associated with persisting whiplash-associated disorders.⁴⁶

Psychological factors have been reviewed regarding the prognosis of late whiplash syndrome.⁴⁷ No association was found among personality traits, distress, well-being, social support, life control, and psychosocial work factors. Limited evidence was seen with post-traumatic distress and chronic symptoms. In a study involving 275 consecutive chronic whiplash-associated disorder patients assessed by valid questionnaires,⁴⁸ it was found that widespread pain in multiple body regions was associated with pain intensity and prevalence of complaints and dysfunction. Furthermore, widespread pain was also associated with depressive symptoms, coping skills, life sat-

isfaction, and general health complaints. In other studies,^{49,50} it was found that depressive symptoms impact pain. Passive coping was associated with slower recovery, and pain severity was related to depression, catastrophizing, and quality of life. Finally, claim closure as assessed in 5,398 subjects was seen earlier in those with less severe pain, better function, and absence of depressive symptoms.⁵¹

Recognition and management of regional and widespread symptoms and evaluation of depression is important in promoting rehabilitation of whiplash-associated disorders (level of evidence grade A), as well as cases of related orofacial symptoms. The studies of prognosis of whiplash-associated disorders and associated TMD symptoms have direct and important implications related to patient assessment and management following motor-vehicle collisions. Therefore, the studies reviewed have led the authors of this study to designate a grade A level of evidence for the prognosis of whiplash-associated disorders and associated TMD symptoms.

EVIDENCE FOR THE PROGNOSIS OF TMDs FOLLOWING MOTOR-VEHICLE COLLISIONS

While the literature does not clearly exclude neck complaints from TMDs, this section focuses on TMD patients following motor-vehicle collisions who may or may not have cervical disorders. As suggested in the previous section, patients with postinjury TMDs do not respond as well to treatment as those involved in nontrauma cases. Patients who do not recover and return to work prior to settlement of claims appear to continue to have symptoms.⁹

Fifty patients with TMDs after motor-vehicle collisions were compared to 50 matched nontrauma-induced TMD controls.⁵² Posttrauma TMD patients reported statistically significantly ($P < .001$) more severe facial pain, neck pain, earache, headache, and sleep disturbances. Examination findings confirmed the history with greater masticatory muscle, neck muscle, and TMJ tenderness in the trauma group. In addi-

Table 3 Potential factors affecting prognosis of chronic whiplash-associated disorders and TMDs

History/preexisting conditions	Physical factors	Psychologic factors
Prior chronic pain	Severity of injury/regional pain (headache, facial, neck, shoulder, back)	Psychosocial stressors, depression, or anxiety
Prior TMDs, headache, neck, and back complaints	Vehicle damage/direction of impact (rear)	Somatization
Preexisting systemic conditions (such as arthritis)	Direct facial trauma	Catastrophic thinking, low self-expectations
Preexisting psychologic factors	Loss of consciousness or traumatic brain injury	Fear, avoidance of exercise (passive coping)
Regional and widespread pain	Female	Posttraumatic stress disorder
—	Body mass index	Sleep disorder
—	Nature of prior work	Social support
—	—	Medicolegal factors (litigation)

tion, greater impact upon work and recreational activities was reported in trauma patients. In another study using the same sample, it was reported that posttrauma TMD patients received more types of treatment ($P < .0001$), took more medications (analgesics, muscle relaxants, and antidepressants; all $P < .001$), had more health care visits ($P = .07$), received treatment over longer periods ($P = .06$), and had poorer outcomes ($P < .001$).⁵³ Another study confirmed that TMD patients following motor-vehicle collisions show poor response to management and require more treatment as compared to nontrauma cases.⁵⁴ Sixty percent of motor-vehicle collision cases had symptoms consistent with a depressive disorder vs 14% of nontrauma cases. Other studies confirm the poorer prognosis of TMDs in motor-vehicle collisions.^{5,25} Mechanisms include sensory and central hypersensitivity that may result in both more severe and increased regional and widespread pain.^{55,56} One study reported posttrauma TMD patients to have similar responses to conservative therapy; however, the posttrauma group required continuing analgesics, which suggested persistence of pain.⁵⁷ Sixteen matched patients with TMDs associated with and without whiplash-associated disorders were assessed; trauma

cases were found to be associated with increased somatization and depression, and poorer outcomes were seen.⁵⁸ In an extensive review of the literature, Fernandez et al²² suggested a poorer prognosis for resolution of TMDs associated with motor-vehicle accidents as compared to idiopathic/nontraumatic TMDs. Contrarily, in a clinical report involving 400 TMD patients, no differences were found in outcome (pain and dysfunction) from a conservative treatment (counseling, occlusal appliance, physical therapy, and medication) between the trauma and nontrauma groups at a 1-year evaluation.¹⁹ Based upon the reviewed articles, it appears there is inconsistent but generally a poor prognosis for the resolution of TMDs (independent of whiplash-associated disorders) following motor-vehicle collisions. Therefore, a grade A level of evidence was assigned.

Many potential factors may be responsible for the progression of symptoms moving from an acute phase to one of chronicity. It is important for oral health care providers to understand what these may be so that appropriate management strategies may be utilized. Therefore, a review of these potential factors affecting prognosis of whiplash-associated disorders and TMDs has been provided in Table 3.



EVIDENCE FOR THE DIRECTION OF COLLISION AND PROGNOSIS OF WHIPLASH-ASSOCIATED DISORDERS AND TMDs

Front-end collisions lead to a higher risk of direct dental and facial injury, while rear-end collisions are associated with higher risk of whiplash-associated disorders and TMDs.⁵⁹ Poorer outcomes of treatment were impacted by complaints despite minimal vehicular damage, lack of use of headrest, driver position, and ongoing insurance claims.

Another study of collision-associated TMDs in 219 consecutive patients revealed that reduced maximum jaw opening, headache, and facial pain were associated with degree of vehicle damage (as represented by cost of repair), with the greatest limitation in those involved in impacts that resulted in write-off of the vehicle. Higher-speed impact (over 40 mph) was associated with greater pain (all $P < .05$).⁶⁰ Direction of impact was examined, and facial pain was more common with rear impact, followed by front and then side impact ($P < .02$); head position at impact (when recalled) showed that turning to the side was associated with increased pain.⁶⁰ A grade B level of evidence was ascribed to collision characteristics and their impact upon prognosis for whiplash-associated disorders and TMDs.

EVIDENCE REGARDING THE INFLUENCE OF LITIGATION ON THE PROGNOSIS OF WHIPLASH-ASSOCIATED DISORDERS AND TMDs

The impact of ongoing litigation was assessed in 35 post-motor-vehicle collision patients and compared to 19 cases not in litigation.²⁵ Statistically significant differences were seen in litigating vs nonlitigating patients: earache (72% vs 42%), TMJ noises (97% vs 65%) (both $P < .05$), headache (97% vs 79%), and dizziness (82% vs 58%) ($P < .10$). A greater number of symptomatic complaints were noted by

litigating patients (15 vs 7 for nonlitigating, $P = .004$).²⁵ Increased numbers of complaints have been reported by others in litigating patients, whose complaints included TMDs.⁶¹ However, a study⁶² in Lithuania assessed TMD symptoms by questionnaire in patients with whiplash (response rate 79%) an average of 27 (range 14 to 41) months after a rear-end motor-vehicle collision compared to controls and found TMDs were not common or comparable to controls. In addition, this study found that acute neck symptoms were higher following motor-vehicle collisions than in controls and that those symptoms typically resolved within 4 weeks. The lack of increased chronic complaints and TMDs may be attributed to limited compensation in Lithuania.

Another study interviewed 30 previously treated patients with TMDs following motor-vehicle collisions to assess status of prior symptoms.⁶³ Approximately 75% had persisting jaw pain, dysfunction, and headache, with more than 80% having neck pain. Jaw pain was moderate or severe in 56%, headache in 63%, and neck pain in 70%. Jaw pain was improved or resolved in 65%, headache in 60%, and neck pain in 50%. Persisting symptoms continued to have negative impact upon patient-reported quality of life. No differences were seen in this study between those who had settled or had ongoing claims, and jaw dysfunction and head and neck pain continued, suggesting ongoing litigation or settling the claim did not impact the common persistence of pain and dysfunction.⁶³ Due to the conflicting outcomes of the impact of litigation upon the prognosis of whiplash-associated disorders and TMDs, a grade B level of evidence was allocated to this subject.

LIMITATIONS OF STUDY

This article should be used judiciously and within the context of a narrative review with its inherent limitations. A systematic literature review or meta-analysis uses a strict methodology to answer specific research questions with predefined outcomes,

Table 4 Evidence-based statements of whiplash-associated disorders and TMDs following motor-vehicle collisions

Summary statement	Level of evidence*	References
TMDs are associated with whiplash-associated disorders	A	10,14,17,18,19,20
Delayed diagnosis of TMDs may occur	B	21,34
TMDs are associated with regional and widespread pain	A	8,13,22,24
TMDs and whiplash-associated disorders are associated with psychologic symptoms	A	25,26,27
Management follows principles of physical medicine and psychologic support	B	19,28,29,31
Prognosis of TMDs and whiplash-associated disorders	A	16,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48,49,50
Prognosis of TMDs following motor-vehicle collisions†	A	3,7,20,23,53,55,56,59
Motor-vehicle collision impact characteristics and TMDs	B	58,59
Conflicting outcomes of litigation upon outcome of whiplash-associated disorders and TMDs	B	23,60,61,62

*Oxford Centre for Evidence-Based Medicine Levels of Evidence (see text for explanations). †As reported independently of whiplash-associated disorders.

whereas a narrative review is employed to address a broader range of questions and provides a summary of findings. Although this article utilized rather general inclusion/exclusion criteria, formal blinded appraisal and assessment of studies reviewed was not administered. The nature of the current literature led to inclusion of studies as per inclusion criteria assigned that did not include requirements for specific research designs and specific and designated statistical approaches and numerical sample size calculations. Additionally, the literature review and articles were selected from only one of several available international scientific literature databases. Upon reviewing and analyzing the included articles, there were large variations in the results that may have been due to variables that included the heterogeneity of populations studied, clinical features, or outcomes targeted and type of data reported. Recognizing these limitations, a narrative review was conducted and a standardized grading system was utilized in producing levels of evidence for each topic discussed.

CONCLUSION

TMDs have been clearly documented to follow motor-vehicle collisions; however, TMDs are identified in only a subset of whiplash-associated disorder patients or as an independent finding. TMDs may not be diagnosed at the time of first assessment, due to the development of symptoms at a later date or later recognition of ongoing dysfunction. TMDs independent of or associated with whiplash-associated disorders appear to occur predominantly in females and may often be accompanied by other regional or widespread pain that may reflect central, systemic, and psychologic effects. These findings suggest that multidisciplinary management is necessary in many patients and that oral health care providers must consider all the factors involved using a biopsychosocial approach when managing individuals who are experiencing orofacial signs and symptoms related to motor-vehicle collisions. To aid oral health care providers in the management of these complex individuals, a summary of the features and findings of the material discussed of whiplash-associated disorders and TMDs is presented in Table 4.



REFERENCES

- Cote P, Cassidy JD, Carroll L, Frank JW, Bombardier C. A systematic review of the prognosis of acute whiplash and a new conceptual framework to synthesize the literature. *Spine* 2001;26:e445–e458.
- Hartling L, Brison RJ, Arden C, Pickett W. Prognostic value of the Quebec Classification of Whiplash-Associated Disorders. *Spine* 2001;26:36–41.
- Spitzer WO, Skovron ML, Salmi LR, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: Redefining “whiplash” and its management. *Spine* 1995;20:15–73S.
- Epstein JB. Temporomandibular disorders, facial pain and headache following motor vehicle accidents. *J Can Dent Assoc* 1992;58:488–489, 493–495.
- Klobas L, Tegelberg A, Axelsson S. Symptoms and signs of temporomandibular disorders in individuals with chronic whiplash-associated disorders. *Swed Dent J* 2004;28:29–36.
- Seligman DA, Pullinger AG. A multiple stepwise logistic regression analysis of trauma history and 16 other history and dental cofactors in females with temporomandibular disorders. *J Orofac Pain* 1996;10:351–361.
- Visscher C, Hofman N, Mes C, Lousberg R, Naeije M. Is temporomandibular pain in chronic whiplash-associated disorders part of a more widespread pain syndrome? *Clin J Pain* 2005;21:353–357.
- Bergman H, Andersson F, Isberg A. Incidence of temporomandibular joint changes after whiplash trauma: A prospective study using MR imaging. *AJR Am J Roentgenology* 1998;171:1237–1243.
- Kolbinson DA, Epstein JB, Burgess JA. Temporomandibular disorders, headaches, and neck pain following motor vehicle accidents and the effect of litigation: Review of the literature. *J Orofac Pain* 1996;10:101–125.
- Ferrari R, Russell AS, Carroll LJ, Cassidy JD. A re-examination of the whiplash associated disorders (WAD) as a systemic illness. *Ann Rheum Dis* 2005;64:1337–1342.
- Kasch H, Hjorth T, Svensson P, Nyhuus L, Jensen TS. Temporomandibular disorders after whiplash injury: A controlled, prospective study. *J Orofac Pain* 2002;16:118–128.
- Heise AP, Laskin DM, Gervin AS. Incidence of temporomandibular joint symptoms following whiplash injury. *J Oral Maxillofac Surg* 1992;50:825–828.
- Quinlan KP, Annett JL, Myers B, Ryan G, Hill H. Neck strains and sprains among motor vehicle occupants—United States, 2000. *Accid Anal Prev* 2004;36:21–27.
- Probert TC, Wiesenfeld D, Reade PC. Temporomandibular pain dysfunction disorder resulting from road traffic accidents—An Australian study. *Int J Oral Maxillofac Surg* 1994;23:338–341.
- Cassidy JD, Carroll L, Cote P, Holm L, Nygren A. Mild traumatic brain injury after traffic collisions: A population-based inception cohort study. *J Rehabil Med* 2004;43(suppl):15–21.
- Carroll LJ, Ferrari R, Cassidy JD. Reduced or painful jaw movement after collision-related injuries: A population-based study. *J Am Dent Assoc* 2007;138:86–93.
- Cassidy JD, Carroll LJ, Cote P, Lemstra M, Berglund A, Nygren A. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *N Engl J Med* 2000;342:1179–1186.
- Kamper SJ, Rebeck TJ, Maher CG, McAuley JH, Sterling M. Course and prognostic factors of whiplash: A systematic review and meta-analysis. *Pain* 2008;138:617–629.
- De Boever JA, Keersmaekers K. Trauma in patients with temporomandibular disorders: Frequency and treatment outcome. *J Oral Rehabil* 1996;23:91–96.
- Kronn E. The incidence of TMJ dysfunction in patients who have suffered a cervical whiplash injury following a traffic accident. *J Orofac Pain* 1993;7:209–213.
- Klobas L, Axelsson S, Tegelberg A. Effect of therapeutic jaw exercise on temporomandibular disorders in individuals with chronic whiplash-associated disorders. *Acta Odontol Scand* 2006;64:341–347.
- Fernandez CE, Amiri A, Jaime J, Delaney P. The relationship of whiplash injury and temporomandibular disorders: A narrative literature review. *J Chiropr Med* 2009;8:171–186.
- Sale H, Isberg A. Delayed temporomandibular joint pain and dysfunction induced by whiplash trauma: A controlled prospective study. *J Am Dent Assoc* 2007;138:1084–1091.
- Treleaven J, LowChoy N, Darnell R, Panizza B, Brown-Rothwell D, Jull G. Comparison of sensorimotor disturbance between subjects with persistent whiplash-associated disorder and subjects with vestibular pathology associated with acoustic neuroma. *Arch Phys Med Rehabil* 2008;89:522–530.
- Grushka M, Ching VW, Epstein JB, Gorsky M. Radiographic and clinical features of temporomandibular dysfunction in patients following indirect trauma: A retrospective study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007;104:772–780.
- Kosek E, Januszewska A. Mechanisms of pain referral in patients with whiplash-associated disorder. *Eur J Pain* 2008;12:650–660.
- Carroll LJ, Cassidy JD, Cote P. Frequency, timing, and course of depressive symptomatology after whiplash. *Spine* 2006;31:e551–e556.
- Westcott MC, Alfano DP. The symptom checklist-90-revised and mild traumatic brain injury. *Brain Inj* 2005;19:1261–1267.
- Gargan M, Bannister G, Main C, Hollis S. The behavioural response to whiplash injury. *J Bone Joint Surg Br* 1997;79:523–526.



30. Bostick GP, Ferrari R, Carroll LJ, et al. A population-based survey of beliefs about neck pain from whiplash injury, work-related neck pain, and work-related upper extremity pain. *Eur J Pain* 2009;13:300–304.
31. Peloso P, Gross A, Haines T, Trinh K, Goldsmith CH, Aker P. Medicinal and injection therapies for mechanical neck disorders. *Cochrane Database Syst Rev* 2005(2):CD000319.
32. Carroll A, Barnes M, Comiskey C. A prospective randomized controlled study of the role of botulinum toxin in whiplash-associated disorder. *Clin Rehabil* 2008;22:513–519.
33. Ekvall Hansson E, Mansson NO, Ringsberg KA, Hakansson A. Dizziness among patients with whiplash-associated disorder: A randomized controlled trial. *J Rehabil Med* 2006;38:387–390.
34. Poorbaugh K, Brismee JM, Phelps V, Sizer PS Jr. Late whiplash syndrome: A clinical science approach to evidence-based diagnosis and management. *Pain Pract* 2008;8:65–87.
35. Schofferman J, Bogduk N, Slosar P. Chronic whiplash and whiplash-associated disorders: An evidence-based approach. *J Am Acad Orthop Surg* 2007;15:596–606.
36. Williams M, Williamson E, Gates S, Lamb S, Cooke M. A systematic literature review of physical prognostic factors for the development of Late Whiplash Syndrome. *Spine* 2007;32:e764–e780.
37. Walton DM, Pretty J, MacDermid JC, Teasell RW. Risk factors for persistent problems following whiplash injury: Results of a systematic review and meta-analysis. *J Orthop Sports Phys Ther* 2009;39:334–50.
38. Miro J, Nieto R, Huguet A. Predictive factors of chronic pain and disability in whiplash: a Delphi poll. *Eur J Pain* 2008;12:30–47.
39. Crouch R, Whitewick R, Clancy M, Wright P, Thomas P. Whiplash associated disorder: Incidence and natural history over the first month for patients presenting to a UK emergency department. *Emerg Med J* 2006;23:114–118.
40. Yang X, Cote P, Cassidy JD, Carroll L. Association between body mass index and recovery from whiplash injuries: A cohort study. *Am J Epidemiol* 2007;165:1063–1069.
41. Sterling M, Jull G, Vicenzino B, Kenardy J, Darnell R. Physical and psychological factors predict outcome following whiplash injury. *Pain* 2005;114:141–148.
42. Hendriks EJ, Scholten-Peeters GG, van der Windt DA, Neeleman-van der Steen CW, Oostendorp RA, Verhagen AP. Prognostic factors for poor recovery in acute whiplash patients. *Pain* 2005;114:408–416.
43. Scholten-Peeters GG, Verhagen AP, Bekkering GE, et al. Prognostic factors of whiplash-associated disorders: A systematic review of prospective cohort studies. *Pain* 2003;104:303–322.
44. Solomon S. Chronic post-traumatic neck and head pain. *Headache* 2005;45:53–67.
45. Carroll LJ, Holm LW, Hogg-Johnson S, et al. Course and prognostic factors for neck pain in whiplash-associated disorders (WAD): Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33:S83–S92.
46. Kongsted A, Bendix T, Qerama E, et al. Acute stress response and recovery after whiplash injuries. A one-year prospective study. *Eur J Pain* 2008;12:455–463.
47. Williamson E, Williams M, Gates S, Lamb SE. A systematic literature review of psychological factors and the development of late whiplash syndrome. *Pain* 2008;135:20–30.
48. Peolsson M, Borsbo B, Gerdle B. Generalized pain is associated with more negative consequences than local or regional pain: A study of chronic whiplash-associated disorders. *J Rehabil Med* 2007;39:260–268.
49. Carroll LJ, Cassidy JD, Cote P. The role of pain coping strategies in prognosis after whiplash injury: Passive coping predicts slowed recovery. *Pain* 2006;124:18–26.
50. Borsbo B, Peolsson M, Gerdle B. Catastrophizing, depression, and pain: Correlation with and influence on quality of life and health—A study of chronic whiplash-associated disorders. *J Rehabil Med* 2008;40:562–569.
51. Cote P, Hogg-Johnson S, Cassidy JD, Carroll L, Frank JW. The association between neck pain intensity, physical functioning, depressive symptomatology and time-to-claim-closure after whiplash. *J Clin Epidemiol* 2001;54:275–286.
52. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. A comparison of TMD patients with or without prior motor vehicle accident involvement: Initial signs, symptoms, and diagnostic characteristics. *J Orofac Pain* 1997;11:206–214.
53. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. A comparison of TMD patients with or without prior motor vehicle accident involvement: Treatment and outcomes. *J Orofac Pain* 1997;11:337–345.
54. Romanelli GG, Mock D, Tenenbaum HC. Characteristics and response to treatment of post-traumatic temporomandibular disorder: A retrospective study. *Clin J Pain* 1992;8:6–17.
55. Scott D, Jull G, Sterling M. Widespread sensory hypersensitivity is a feature of chronic whiplash-associated disorder but not chronic idiopathic neck pain. *Clin J Pain* 2005;21:175–181.
56. Curatolo M, Petersen-Felix S, Arendt-Nielsen L, Giani C, Zbinden AM, Radanov BP. Central hypersensitivity in chronic pain after whiplash injury. *Clin J Pain* 2001;17:306–315.
57. Greco CM, Rudy TE, Turk DC, Herlich A, Zaki HH. Traumatic onset of temporomandibular disorders: Positive effects of a standardized conservative treatment program. *Clin J Pain* 1997;13:337–347.
58. Krogstad BS, Jokstad A, Dahl BL, Soboleva U. Somatic complaints, psychological distress, and treatment outcome in two groups of TMD patients, one previously subjected to whiplash injury. *J Orofac Pain* 1998;12:136–144.



Epstein/Klasser

59. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. Effect of impact and injury characteristics on post-motor vehicle accident temporomandibular disorders. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998;85:665–673.
60. Burgess JA, Kolbinson DA, Lee PT, Epstein JB. Motor vehicle accidents and TMDs: Assessing the relationship. *J Am Dent Assoc* 1996;127:1767–1772.
61. Burgess JA, Dworkin SF. Litigation and post-traumatic TMD: How patients report treatment outcome. *J Am Dent Assoc* 1993;124:105–110.
62. Ferrari R, Schrader H, Obelieniene D. Prevalence of temporomandibular disorders associated with whiplash injury in Lithuania. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;87:653–657.
63. Kolbinson DA, Epstein JB, Burgess JA, Senthilselvan A. Temporomandibular disorders, headaches, and neck pain after motor vehicle accidents: A pilot investigation of persistence and litigation effects. *J Prosthet Dent* 1997;77:46–53.

