

A Review of the Literature Refuting the Concept of Minor Impact Soft Tissue Injury

(M.I.S.T.)

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Short Title: Refuting the M.I.S.T Concept

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Summary:

M.I.S.T. is a acronym that stands for Minor Impact Soft-Tissue. The concept refers to the claim that low damage impacts can not be associated with significant injuries. The

literature concerning late whiplash is reviewed. This review focuses on medical research which refutes the M.I.S.T. concept.

**Abstract:**

**Background:** Minor Impact Soft Tissue is a concept that seeks to identify late whiplash as a psychosocial phenomenon. However, the medical literature in this area has not been systematically reviewed since the Quebec Task Force in 1995.

**Objectives:** To review the medical literature which claims that late whiplash is an organic phenomenon causing significant disability.

**Methods:** The medical literature was reviewed in a narrative format.

**Results:** There are a significant number of studies which refute the M.I.S.T. concept.

**Conclusions:** A review of the literature does not support the validity of M.I.S.T.

In the mid nineteen nineties, the U.S. automobile insurance industry launched a new concept in claims handling called M.I.S.T., an acronym for Minor Impact Soft Tissue. The theory behind this claims stance was that it was virtually impossible to sustain a permanent or serious injury in a low damage car crash. As a result, these claims should be handled differently. This new concept has expanded to almost all major U.S. insurers, yet little has been published regarding its scientific validity. For many patients with objective physical exam findings but little automobile property damage, this policy has led to loss of insurance coverage for their injuries.

#### The Medical Literature in Support of M.I.S.T.

Early studies suggested that the g-forces involved in low damage crashes were comparable to those commonly seen with normal everyday activities of daily living.[1] This concept was driven home by the Quebec Task Force in 1995.[2] This report seemed to demonstrate that whiplash was a short lived and self-limited condition that didn't require more than supportive care. In addition, other similar reports suggested that late whiplash didn't exist in countries where there is no legal system to recover damages. [3] In addition, more recent studies performed in Saskatchewan suggested that when the ability to sue for pain and suffering was removed, the duration of the insurance claim for medical coverage was reduced.[4] Based on this information, it would then seem reasonable that insurers would adopt the M.I.S.T. policy. However, since multimillion

dollar decisions rely on this policy, the validity of the research in support of M.I.S.T. must be vetted.

Freeman was the first to point out that many of the studies refuting the existence of late whiplash had very poor methodology.[5] This author also published a research critique of the Quebec Task Force and the Saskatchewan study.[6] [7] which demonstrated that this research also suffered from flaws in methodology. As a result, the conclusions of these studies were called into question. In light of this information, the foundation for the M.I.S.T. needs further investigation.

#### The Literature Linking Low Damage High Energy Crashes to Serious Injury

A major building block of the foundation for M.I.S.T. relies on the concept that vehicle damage and occupant damage must be closely linked. Said another way, there must be a linear relationship between how hard a vehicle is struck (delta V or change in velocity) and serious injury rates. However, as a research question this phenomenon is very difficult to study. Up until recently, staged crashes were the only way any information regarding delta V and injury rates could be gleaned. However, staged crashes are specifically designed not to injure the participants. Therefore, staged crashes are a poor place to study injury rates.

The advancement in technology has provided an opportunity for these questions to be answered in a real world crash setting. Krafft has now studied the relationship between real world delta V as measured by “Black Boxes” installed inside many vehicles and chronic injuries.[8] While one would expect a linear relationship, none was found. For instance, chronic injury rates at delta V’s of 5-10 km/h were twice that of 10-15 km/h! In addition, again chronic injury rates at 15-20 km/h were twice the rates seen at 20-25 km/h delta V’s. These rates likely relate to the stiffness and elasticity of the vehicle and the complex interplay of seat design, occupant mass, occupant position, and vehicle dynamics. In addition, Krafft also discovered a much higher AIS 1 (WAD II or WAD III) chronic injury rate in the presence of a tow-hitch. This external factor hints at a list of complex kinematics that the M.I.S.T. program does not utilize to determine injury risk. Finally, in the same study, Krafft also concluded, “The two crashes which resulted in long-term disabling neck injuries had the highest peak acceleration (15 and 13 x g), but not the highest change of velocity.” This is again very concerning for the M.I.S.T. methodology, as it demonstrates serious neck injury resulting from high peak accelerations in high energy, but low damage and low delta V settings. Brault et al produced similar findings when investigating rear end collisions.[9] Their conclusions are also concerning for M.I.S.T.: “Objective clinical deficits consistent with whiplash associated disorders (WAD) were measured in both men and women subjects at both 4 km/h and 8 km/h. At 4 km/h, the duration of symptoms experienced by women was significantly longer when compared with that in men ( $p < .05$ ). There were no significant differences in the presence and severity of WAD between men and women at 4 km/h and

8 km/h or in the duration of WAD at 8 km/h. There was also no significant difference in the presence, severity, and duration of WAD between 4 km/h and 8 km/h. No preimpact measures were predictive of WAD.” In summary, Brault again concluded that trying to tie delta V to injury rates didn’t work. Siegmund again echoed the same findings while trying to create a model of rear end crash dynamics and long-term injury risk.[10] Again, there was no connection between delta V and injury risk. Finally, Davis in a meta-analysis of the medical literature on delta-V and long-term injury risk reached the same conclusion.[11]

Why is this uncoupling of crash damage and long-term injury rates occurring? Some clues can be found in studies presented at international congresses that show that vehicle stiffness has increased to reduce property damage in low speed crashes. However, the vehicle is only one parameter. Much more attention recently has been paid to seat back design in rear end crashes. Viano has concluded that one reason whiplash injuries are increasing is that seats have been made stiffer to avoid rearward occupant ejection in a seat back failure.[12] As seats are made stiffer, the shear forces (NIC) on the neck increase. In addition, newer studies by the same author suggest that for females, a lower relative mass as compared to seat back stiffness may play a role in serious neck injury at low speeds.[13] Head restraint characteristics are also likely involved.[14]

Clearly, the lack of a direct link between delta V and long-term AIS 1 neck injury rates calls into question the validity of a no damage, no injury policy.

### The Literature that Defines Late Whiplash as a Serious Medical Condition

If late whiplash is a short-term mild muscle pull that should always resolve on its own with only supportive care, then the M.I.S.T. policy would again seem reasonable.

However, if data exists that this injury is more serious, then again M.I.S.T. would be called into question.

The early medical literature for late whiplash is clearly supportive of a M.I.S.T. policy.

The focus was on a muscle strain and possibly a ligament sprain. Hence the name sprain-strain was commonly used. Compared to other muscle strains such as a hamstrings injury, whiplash seemed to have an excessively long recovery time that could only be explained by psychological problems.[15-22] In addition, at that time, little was known about the central nervous system and pain and spinal ligament injuries that did not require surgery.

However, in the last decade, much has been learned about what is injured in late whiplash patients. As a result, the landscape has been significantly altered. What we would previously call a “soft-tissue” injury has now been redefined into numerous injury categories.

Seminal studies by Taylor and Twomey demonstrated that serious spinal injuries could be detected on cadaver dissection.[23-25] These patients had all died of other causes such as blunt abdominal trauma, yet many seemed to have very serious spinal injuries. These injuries included bleeding into the dorsal root ganglia, small fractures of the facet joints, bleeding into the facet joints, and other injuries. While these insults could be easily detected on dissection, they couldn't be detected on more advanced imaging.

In-vitro studies by Grauer and Panjabi were also telling. In simulated low speed rear end collisions, they demonstrated facet joint sparring in the cervical spine as well as significant ligament stretch injury to the anterior longitudinal ligament and facet joint capsules.[26-28] Other authors have now confirmed these findings and added to the database of significant joint and ligament injuries that occur at low speeds.[29, 30] In addition, these findings have also been confirmed in live volunteers in simulated low speed crash tests.[31] If the cervical facet joints were injured, then clinical studies would have to confirm that these joints were pain generators in a late whiplash population. Indeed, numerous studies have now confirmed that when these joints are anesthetized and treated, both short-term and long-term relief is the result.[32-35] In addition, when double blinded prevalence studies are reported, approximately 50% of patients with late whiplash have been found with injured neck joints.[36]

More recently, central sensitization has been the focus of late whiplash research. The early studies above demonstrating injury to the dorsal root ganglion as well as crash



research by Svensson showing injury to the same structure, has moved researchers to take a closer look at neurologic injury.[37-40] It has been noted by numerous researchers that late whiplash patients have different sensory thresholds than normal controls.[41-46] These patients show increased sensitivity to a variety of stimuli including pressure, light vibration, heat and cold, not only in the neck but also in body areas remote to the site of pain such as the front of the shin. This means that they feel things differently than someone with a normal sensory system. Importantly in those patients who fail to recover following injury, these sensory changes have been shown to be present from very soon after injury. As outlined above, the prevailing opinion in that this is due to sensitization of the the central nervous system. For instance, recent research has correlated elevated levels of a protein only released in CNS injury with more severely injured whiplash patients.[47] However, more surprising is that serum muscle injury markers are not elevated in whiplash patients, indicating that the muscle strain part of the whiplash theory espoused early on is likely not valid.[48]

Finally, as above, investigators over the last decade have reported that serious ligament injury is likely one cause of late whiplash injury. MRI indicators of upper cervical ligament injuries in the alar, transverse ligament, posterior atlanto-occipital membrane and tectorial membranes have been found in late whiplash patients but not in controls.[49-52] In addition, significant lower cervical ligament injury has also been reported by multiple authors both in vitro cadaver studies and in real world imaging studies.[29, 53-58]

From all of the above evidence it can be seen that, at least in some patients, whiplash is a complex, multifaceted condition that requires a suitable classification system to address these complexities.

#### Long-term Prognosis for Late Whiplash Injury

If late whiplash is more than a muscle pull or mild sprain, then are these problems minor “soft-tissue” injuries or do they have a major functional impact?

Berglund has looked at this issue in a large epidemiologic study where several hundred patients who sought specialist care for a rear end crash were compared to several thousand people not exposed to such a crash. Seven years after the crash, there was a 160%-370% increased risk for headache, thoracic and low back pain, as well as for fatigue, sleep disturbances and ill health.[59] The same type of investigation found a three fold increased risk for neck and shoulder pain seven years after a rear end crash exposure.[60] In addition, Squires reported on a group followed for 15.5 years.[61] 70% of these patients continued to report symptoms related to the original crash. Between years 10 and 15.5, 18% had improved, while 28% had worsened and 54% had stayed the same. Finally, Bunketorp conducted a similar investigation seventeen years after a crash.[62] She found that when patients who sought specialty care for injuries reported in an ER were compared to patients also seen in the ER but with no MVC related

complaints, that the disability rate in the injury group was 30-35% while the non-injury group reported an injury rate of only 6%.

### Is M.I.S.T. Still Scientifically Viable?

While many authors have published studies that would seem to support the M.I.S.T. hypothesis, the vast majority of work published in the last 10 years would not support M.I.S.T. Assuming an insurer must take the position that policyholder must at all times be given the benefit of the doubt; the M.I.S.T. program does not have overwhelming scientific support. We would argue that its time to retire M.I.S.T. in favor of a research based severity indexing approach that allows insurers to better allocate resources.

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