

**STATE OF MICHIGAN  
IN THE SUPREME COURT**

HAROLD HUNTER JR.,  
Plaintiff-Appellant,

Docket No. 147335  
Court of Appeals Docket no. 306018  
Genesee Circuit Court  
Hon. Joseph J. Farah  
LC No. No 10-094081-NI

-vs-

CITY OF FLINT TRANSPORTATION  
DEPARTMENT,

Defendant-Appellee.

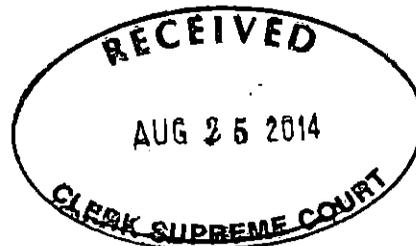
and

DAVID SISCO and AUTO CLUB  
INSURANCE ASSOCIATION,

Defendants.

**BRIEF AMICUS CURIAE OF THE  
MICHIGAN ASSOCIATION FOR JUSTICE**

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## STATEMENT OF QUESTIONS PRESENTED

### I.

#### **DOES "BODILY INJURY" INCLUDE "PAIN" AND "SUFFERING"?**

Plaintiff-Appellant Harold Hunter answers "YES."

Defendant-Appellee Flint Department of Transportation would answer "NO."

The trial court answered "NO."

The Court of Appeals answered "NO."

Amicus curiae Michigan Association for Justice answers "YES."

## **INTEREST OF AMICUS CURIAE**

The Michigan Association for Justice (MAJ) is an organization of Michigan lawyers engaged primarily in litigation and trial work. MAJ recognizes an obligation to assist this Court in important issues of law that would substantially affect the orderly administration of justice in the trial courts of this state.

## **STATEMENT OF FACTS**

Amicus curiae Michigan Association for Justice adopts the statement of facts in plaintiff-appellant's brief on appeal.

## ARGUMENT I

### “BODILY INJURY” INCLUDES “PAIN” AND “SUFFERING.”

(a)

**The Court of Appeals’ construction of “bodily injury” was unduly restrictive.**

As discussed fully in plaintiff-appellant’s brief on appeal, the Court of Appeals panel in this matter held that “bodily injury” “encompasses only ‘a physical or corporeal injury to the body [therefore] . . . the trial court erroneously ruled that plaintiff may recover damages for pain and suffering . . .” *Hunter v Sisco*, 300 Mich App 229, 240; 832 NW2d 753 (2013).

The Court of Appeals in *Hunter* professed to rely on *Wesche v Mecosta County Road Com’n*, 480 Mich 75; 746 NW2d 847 (2008), which stated that “[t]he word ‘bodily’ means ‘of or pertaining to the body’ or ‘corporeal or material, as contrasted with spiritual or mental’” [and] [t]he word ‘injury’ refers to ‘harm or damage done or sustained, [especially] bodily harm.’ 480 Mich 84-85 (citations omitted).

As plaintiff-appellant’s brief in this Court explains, the *Wesche* construction of the phrase “bodily injury” in MCL 691.1405 is unduly restrictive. Amicus curiae Michigan Association for Justice adopts plaintiff-appellant’s argument to this effect.

In the companion case to the present matter, *Hannay v MDOT*, docket no. 146763, this Court granted leave to determine “whether economic loss in the form of wage loss may qualify as a ‘bodily injury’” under MCL 691.1405, citing *Wesche, supra*. Amicus curiae concurs with plaintiff-appellee’s Argument I(A) in *Hannay*, as well as the position of the amicus curiae filed in support of plaintiff-appellee concerning the interrelationship of *Wesche, Hannay* and the present case.

As discussed *infra*, however, even under the restricted reading of the term found in *Wesche, supra*, “pain” and “suffering” should be recognized as “bodily injury.”

(b)

**“Pain” is an “injury” to the body.**

There is an extensive literature on the science of pain. Even a relatively brief review should establish that “pain” is both a result of physical injury and an “injury” in itself.

One authority explains, “pain is caused when peripheral nerves are activated by a stimulus to the skin or internal organs,” leading to the generation of an electrical signal along the axon of the nerve.<sup>1</sup> Pain “alerts us to danger and potential external or internal sources of harm.”<sup>2</sup> Pain “often has its source in the peripheral nervous system.”<sup>3</sup>

“Pain” has been defined as:

[a] primary condition of sensation or consciousness, the opposite of pleasure; the sensation which one feels when hurt (in body or mind); suffering, distress. Or: in specifically physical sense: Bodily suffering; a distressing sensation as of soreness (usually in a particular part of the body).<sup>4</sup>

Currently, however, “pain researchers and clinicians utilize the International Association for the Study of Pain definition of pain[,] ‘Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.’”<sup>5</sup>

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<sup>1</sup> Turkington, *The Encyclopedia of the Brain and Brain Disorders (2nd ed.)* (Facts on File, Inc.: New York, 2002), p 238.

<sup>2</sup> Zimmerman, *Basic Physiology of Pain Perception* in Lautenbacher & Fillingim (Eds), *Pathophysiology of Pain Perception* (Kluwer Academic: New York, 2004), p 1.

<sup>3</sup> Zimmerman, *supra* at 4 (citation omitted).

<sup>4</sup> Loeser, *Pain as a disease*, in 81 Aminoff, Boller & Swab, *Handbook of Clinical Neurology*, 3rd Series (Edinburgh: Elsevier, 2006), ch 2, at 13, quoting Oxford English Dictionary.

<sup>5</sup> Loeser, *supra* at 13.

The study of “pain” overlaps multiple areas in science and medicine.<sup>6</sup> Pain “is the subject of biology, medicine, psychology and the social sciences.”<sup>7</sup> For example, the International Association for the Study of Pain, was organized over 40 years ago and “brings together scientists, clinicians, health-care providers, and policymakers to stimulate and support the study of pain and to translate that knowledge into improved pain relief worldwide.”<sup>8</sup>

The basics of “pain” as a physiological phenomenon are well-established. The perception of “pain” lies in the activation of specialized cells in the human body broadly referred to as “nociceptors.” The term comes from the Latin root “noci-” meaning “hurt, pain or injury.”<sup>9</sup> Nociceptors “respond preferentially to noxious stimuli.”<sup>10</sup> Noxious means “injurious or potentially injurious.”<sup>11</sup> As one authority explains:

Pain, like all perceptions, results from a specific spatial-temporal pattern of neurological activity in the cerebral cortex. In most cases, however, pain is initiated by events that occur on the skin, or in deep tissues such as viscera, bone, or muscle. Specialized primary afferent neurons, called nociceptors, constantly survey the environment surrounding their terminals. *These neurons are selectively tuned to respond rapidly to mechanical, thermal, or chemical stimuli that are of a sufficient intensity to cause tissue damage or of a quality that indicates existing tissue damage.*<sup>12</sup> [Emphasis supplied.]

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<sup>6</sup> [www.iasp-pain.org](http://www.iasp-pain.org).

<sup>7</sup> Zimmerman, *supra*, at 1.

<sup>8</sup> [www.iasp-pain.org](http://www.iasp-pain.org).

<sup>9</sup> Stedman’s, p 1323.

<sup>10</sup> Meyer, Ringkamp, Campbell & Raja, *Peripheral mechanisms of cutaneous nocieption* in McMahon & Koltzenburg (Eds), *Wall and Melzack's Textbook of Pain (5th ed)*, (Elsevier, Edinburgh, 2006), p 3.

<sup>11</sup> Meyer et al., *supra*, at 3.

<sup>12</sup> Caterina, Gold & Meyer, *Molecular biology of nociceptors* in Hunt & Koltzenberg (eds). *The Neurobiology of Pain* (Oxford: Oxford U Press, 2005), ch 1, p 1.

There are “millions” of nociceptors in the skin, joints, muscles and membranes around internal organs.<sup>13</sup> Nociceptors are concentrated in injury-prone areas, such as finger and toes.<sup>14</sup> Neurologic injuries to peripheral somatic nerves almost always produce significant pain.<sup>15</sup>

“The ultimate symptom of pain” is a result of “mechanical, chemical, or thermal irritation of tissue containing nociceptive receptors.”<sup>16</sup>

*Chemical mediators are released or synthesized from the damaged tissue. When these mediators accumulate in sufficient quantity they activate [nociceptors].*<sup>17</sup>  
[Emphasis supplied.]

Perceived pain “stems from nerve impulses that reach the central nervous system” by way of nociceptive afferent fibers that “are specialized in conveying *signals concerning potential damage* or health risks originating from outside or inside the body. . .”<sup>18</sup> (emphasis supplied). Nociceptors “have been found in every internal organ of the body.”<sup>19</sup>

Multiple types of nociceptors have been identified, which respond in different ways to different noxious stimuli.<sup>20</sup>

Unlike the innocuous receptors, nociceptors often respond to multiple stimulus modalities and are therefore polymodal. Any stimulus that can produce pain can activate nociceptors including intense heat, intense mechanical stimuli, and various chemicals.<sup>21</sup>

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<sup>13</sup> Swanson, *Mayo Clinic on Chronic Pain*, (Mayo Clinic, Rochester, Minnesota, 1999), p 2.

<sup>14</sup> Swanson, *supra*, at 2.

<sup>15</sup> Rauck, *Trauma*, in Raj, *Pain Medicine: A comprehensive review (2nd ed)*. (Mosby: St. Louis, 2003), ch 6, p 38.

<sup>16</sup> Cailliet, *Soft Tissue Pain & Disability (3rd ed)*, (Philadelphia: F. A. Davis Co. 1996), p 14.

<sup>17</sup> Cailliet, *supra* at 16.

<sup>18</sup> Loeser, *supra* at 4

<sup>19</sup> Loeser, *supra* at 6.

<sup>20</sup> Caterina, Gold & Meyer, *supra*, at 3-5.

<sup>21</sup> Caterina, Gold & Meyer, *supra*, at 3

Pain from injury can be caused by partial or complete transection [cutting] of peripheral nerves, including limb amputation; spinal cord trauma, including compression; fractures and dislocations; soft tissue injury, including tendonitis, fasciitis, ligament injury; and head injury.<sup>22</sup>

Traumatic damage to tissues causes “pain,” but it also causes other reactions. For example, “[a] large area of vasodilation [enlargement of blood vessels] or flare [reddening of the skin] often surrounds a cutaneous [skin] injury.”<sup>23</sup> It is “thought to be caused by a peripheral axon reflex in nociceptive fibers.”<sup>24</sup>

These authorities merely confirm what every sensate human being knows. “Pain” results from a “damage or wound” to a person’s “head, neck, trunk, and limbs,” i.e., “pain” is a “bodily injury.”

(c)

**“Suffering” is a natural consequence of the pain caused by “bodily injury.”**

The first definition in the dictionary of “suffer” is “to experience pain, illness, or injury.”<sup>25</sup> A full discussion of the effects of “pain” on behavior is beyond the scope of this brief and is, indeed, the subject of extensive investigation in medical fields.<sup>26</sup> It is, however, clear from even a limited review of the subject that “pain” causes consequences that can be described as “suffering.”

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<sup>22</sup> Warlow, *The Lancet Handbook of Treatment in Neurology*, (Elsevier: Edinburgh, 2000), pp 386-388.

<sup>23</sup> Caterina, Gold & Meyer, *supra*, at 5.

<sup>24</sup> Caterina, Gold & Meyer, *supra*, at 5.

<sup>25</sup> [www.merriam-webster.com](http://www.merriam-webster.com).

<sup>26</sup> See, e.g., Linton, *Understanding pain for better clinical practice: A psychological perspective*, (Elsevier: Edinburgh, 2005).

“The experience of pain involves almost every part of the brain, the spinal cord and the peripheral nervous system, the immune system, endocrine glands and the metabolic system.”<sup>27</sup>

“Information about pain reaches brain regions involved in emotion, sensory perception, body movement and hormonal release.”<sup>28</sup>

Pain is . . . the most common reason for patients to seek medical treatment. *Pain is always colored with emotional reactions that in some circumstances, or when the pain becomes chronic, can lead to profound alterations of behavior, ranging from anxiety to depression.* Pain is a sensory experience that can be evoked by many different kinds of stimuli . . . Pain accompanies inflammation and tissue repair, and when it goes away it tells us that the process has been completed, but pain can also last well beyond the point of tissue repair or appear without tissue damage or be felt by stimuli that do not normally evoke pain . . . <sup>29</sup> [Emphasis supplied.]

Loeser<sup>30</sup> explains:

*Suffering is a negative affective response generated in the brain by pain, and by psychological states such as fear, anxiety, stress or loss of loved objects. What we do not know is whether this negative affective response to pain originates within the brain when information saying “pain” is received, or whether nociceptive information reaching the dorsal horn leads to the activation of circuits leading to the production of both pain and suffering at the spinal and brainstem levels. . .*

*Suffering usually leads to pain behavior: the things a person does or does not do that are ascribed to tissue damage.* Examples include saying “ouch”, grimacing, limping lying down, consuming health care, and refusal to work. All pain behaviors are real. . .

*It is suffering that drives patients to seek health care, not nociception or pain.* How societies deal with the suffering of their members changes over time and may differ widely over time. To some degree this can be due to variations in the resources available to the society, but it is also related to ethical, religious and philosophical concepts. [Emphasis supplied.]

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<sup>27</sup>Turkington, *The Encyclopedia of the Brain and Brain Disorders (2nd ed.)* (Facts on File, Inc.: New York, 2002), p 238

<sup>28</sup> Turkington, *supra*, at 238.

<sup>29</sup> Cervero & Jensen, *Preface*, in 81 Aminoff, Boller & Swab, *Handbook of Clinical Neurology*, 3rd Series (Edinburgh: Elsevier, 2006), p i.

<sup>30</sup> Loeser, *supra*, at 14.

“In the biomedical context, the word pain is used primarily for two different constructs:

(1) pain as a unit of perception and as a sensory system and (2) pain as suffering and disease.”<sup>31</sup>

*Such a complex experience involves many components of brain function - sensory, motor, autonomic, emotional and cognitive - and is therefore the expression of the integrative workings of the nervous system. Pain is the result of activity in the nervous system starting with a transduction mechanism at peripheral receptor sites to a highly modulated process generated in various brain regions. Pain is, above all, an elaborate expression of the workings of the brain.*<sup>32</sup> [Emphasis supplied.]

Pain perception is based in a chemical process that culminates in the psychological experience of “pain.” The biochemical mechanisms that create “pain,” however, have other consequences. As Cailliet<sup>33</sup> details, the process of pain perception in turn creates muscle spasms, which lead to more pain:

Among these chemical mediators [released in response to noxious stimuli] are phospholipids that break down from arachidonic acid to form prostaglandin E. There are also inflammatory mediators liberated from trauma called leukotriens that do not undergo the same breakdown sequence of arachidonic acid and so are not influenced by nonsteroidal anti-inflammatory agents. Trauma also causes a breakdown of blood platelets that release serotonin, which acts as a vasoconstrictor and causes local edema. The resultant muscle *spasm* that locally accompanies trauma is possibly mediated through a neural pattern . . . wherein the nociceptor impulses emanating through the dorsal root ganglion send impulses through neuronal connections to the anterior horn cell with resultant muscular contraction. The nociceptor stimuli can emanate from the skin, the blood vessels, joint capsules, ligaments and muscles.

\* \* \*

The muscle thus being involved as *recipient* of the nociceptive reaction becomes an *initiator* or nociception. *This is truly a vicious cycle of pain causing muscle spasm, which in turn produced a nociceptor site of pain.* [Additional emphasis supplied.]

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<sup>31</sup>Zimmerman, *Basic Physiology of Pain Perception* in Lautenbacher & Fillingim (Eds), *Pathophysiology of Pain Perception* (Kluwer Academic: New York, 2004), p 1.

<sup>32</sup>Cervero & Jensen, *supra*, p i.

<sup>33</sup>Cailliet, *Soft Tissue Pain & Disability (3rd ed.)*, (Philadelphia: F. A. Davis Co. 1996), p 16.

Pain signals connect with portions of the brain “directly related to the emotions of fear and anxiety.”<sup>34</sup>

As even this brief review illustrates, “pain” is inextricably linked to the experience of “suffering.” A “bodily injury” that creates “pain” will inevitably cause “suffering” as well.

(d)

**Injuries from motor vehicle accidents lead to “pain and suffering” as a result of “bodily injury.”**

In an automobile accident, “kinetic energy from deceleration injuries transmits tremendous force and ultimate disruption of the bony thorax and underlying tissues, in particular to the pulmonary parenchymal tissue.”<sup>35</sup> Soft tissue injuries occur with all penetrating lesions and most nonpenetrating or blunt trauma.<sup>36</sup>

The damage caused by a trauma, such as an auto accident, can have lasting consequences.

*Pain and the associated stress response* observed in trauma patients *should always be viewed as detrimental . . .* Circulating catecholamines and other neuroendocrine peptides have repeatedly been shown to be deleterious in animals and humans.<sup>37</sup> [Emphasis supplied.]

Long-term sequelae from peripheral somatic nerve injuries often result and can be particularly difficult to treat.<sup>38</sup> One study of 3,047 motor vehicle accident patients found that “[a]t 12 months after injury, 1818 patients (62.7%) reported pain related to their injury.”<sup>39</sup> Three-fourths of the patients with moderate to severe injuries to the neck or spine had injury-related pain at 12 months.<sup>40</sup>

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<sup>34</sup>Cailliet, *supra*, at 24.

<sup>35</sup>Rauck, *supra*, at 35.

<sup>36</sup>Rauck, *supra* at 36.

<sup>37</sup> Rauck, *supra* at 35.

<sup>38</sup> Rauck, *supra* at 38.

<sup>39</sup> Rivara, MacKenzie, Jurkovich, Nathens, Wang & Scarfstein, *Prevalence of Pain in Patients 1 Year After Major Trauma*, 143(3) *Archives of Surgery* 282 (2008).

<sup>40</sup> Rivara et al., *supra*.

Consider some of the more common sequelae of “injury” to the “body” caused by motor vehicle accidents.

### Fractures.

*Several orthopedic injuries can require many months to heal. Many of these patients can be expected to have significant pain during the process. External fixators in place for prolonged periods can provide ongoing afferent nociception for some patients. These patients should be viewed as having subacute pain rather than chronic pain if the healing process is continuing. Many do not require further treatment after the offending device has been removed and the lesions have healed. Other patients develop chronic pain syndromes from these severe orthopedic injuries, in part as a result of the anatomic abnormality that alters gait and/or function. The compensatory mechanisms of the body can either be incomplete or lead to their own pain problems.<sup>41</sup>*

### Whiplash.

“Whiplash” is a movement of the neck that occurs in occupants of a stationary vehicle that is struck by another vehicle.<sup>42</sup> Symptoms “may not occur for 12 to 24 hours after a whiplash injury because muscular hemorrhage and edema may need to evolve prior to inciting a nociceptive response.”<sup>43</sup>

[A] variety of possible injuries [may] result[] from whiplash . . . These include strain or avulsion of the anterior annulus fibrosus, strain of the zygapophysial<sup>44</sup> joint capsules, and impaction injuries to the zygapophysial joints ranging from contusions to intra-articular meniscoids and resultant intra-articular hemorrhage, to subchondral and transarticular fractures. Whether or not these injuries occur depends on the magnitude of impact and the susceptibility or resistance to injury of the possible target structure.

\* \* \*

Various symptoms have been attributed to whiplash injury . . . Of these, neck pain and referred pain to the head or to the upper limb are the most prevalent and consistent. Indeed, *pain is the defining clinical feature of whiplash, and is the*

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<sup>41</sup> Rauck *supra* at 37.

<sup>42</sup> Bogduk, *Whiplash injury*, in 81 Aminoff, Boller & Swab, *Handbook of Clinical Neurology*, 3rd Series (Edinburgh: Elsevier, 2006), ch 53, p 79.

<sup>43</sup> Warfield & Bajwa, *Principles and Practice of Pain Medicine*, (McGraw-Hill: New York, 2004), p 263.

<sup>44</sup> Relating to “any of the articular processes of the neural arch of a vertebra of which there are usually two anterior and two posterior.” [www.merriam-webster.com](http://www.merriam-webster.com).

*only symptom readily attributed to an injury to the cervical spine.*<sup>45</sup> [Emphasis supplied.]

Neck pain, headache and shoulder pain, both acute and chronic, are commonly reported.<sup>46</sup>

As many as 74% of drivers involved in high-speed collisions suffer neck pain as a result of joint damage.<sup>47</sup>

In addition to neck pain, “whiplash” may cause other “bodily injury”:

Severe hyperextension injury may stretch the esophagus, with resulting edema or retropharyngeal hematoma, and may cause dysphagia. Bilateral vocal cord paralysis inducing hoarseness has been reported after severe flexion-extension injuries. Damage to the cervical sympathetic chain may occur, resulting in symptoms of nausea, dizziness, Horner’s syndrome, and tinnitus. . .

Injury to the brain itself from abrupt flexion and extension can cause concussion and cerebral contusion, with symptoms of loss of consciousness, dizziness, and headache. One study found that *up to one third of patients with cervical spine or spinal cord injury following trauma had significant head injuries.* These included skull fractures, subarachnoid hemorrhages, and intracranial hematomas.<sup>48</sup> [Emphasis supplied.]

#### Complex regional pain syndrome<sup>49</sup>

Crush injuries are particularly prone to developing reflex sympathetic dystrophy. Because this will not happen in most patients, when a patient feels pain that is out of proportion to the injury or does not respond well to standard opiates, the patient should be checked for [RSD].<sup>50</sup>

#### Hyperalgesia.

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<sup>45</sup> Bogduk, *supra* at 792-793.

<sup>46</sup> Bogduk, *supra* at 793.

<sup>47</sup> Bogduk, *supra* at 798. See also Warfield & Bajwa, *supra* at 263, reporting that “60% of patients who were injured in a car accident and presented to a hospital had neck pain[;] [a]fter 1 year, 26% of this group had persistent neck pain.”

<sup>48</sup> Warfield & Bajwa, *supra* at 263 (citations omitted).

<sup>49</sup> Previously referred to as “reflex sympathetic dystrophy;” the revised descriptor is thought to reflect the underlying mechanisms more accurately.

<sup>50</sup> Rauck *supra* at 38.

“Hyperalgesia” “is a common complaint when tissue is injured and/or inflamed.”<sup>51</sup>

Hyperalgesia is the enhanced pain sensitivity that often occurs after injury, inflammation or certain diseases. Hyperalgesia is characterized by a leftward shift of the stimulus-response function that relates magnitude of pain to stimulus intensity. *In other words, there is a lowering of the threshold for pain and an enhanced pain to normally painful stimuli. Nociceptor sensitization is the neurophysiological correlate of hyperalgesia.* Nociceptor sensitization is characterized by a lowered threshold for activation, an enhanced response to suprathreshold stimuli, and spontaneous discharge.<sup>52</sup>

Hyperalgesia “is a consistent feature of somatic and visceral tissue injury and inflammation. . .”<sup>53</sup> It “occurs not only at the site of the injury but also in the surrounding uninjured area. . .”<sup>54</sup>

Research confirms what every adult should know - motor vehicle accidents may have long-term consequences for their unfortunate victims. These results of physical impact are properly characterized as “pain and suffering” from “bodily injury.”

(e)

**“Pain and suffering” should be recognized as damages under the motor vehicle exception to governmental immunity.**

In Michigan in 2013, the State Police report that 71,031 people were injured in motor vehicle accidents.<sup>55</sup> The United States Department of Transportation estimates that 2,239,000 such injuries occurred in 2012.<sup>56</sup> The Michigan State Police do not track accidents by ownership

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<sup>51</sup> Johanek, Shim & Meyer, *Primary hyperalgesia & nociceptor sensitization* in 81 Aminoff, Boller & Swab, *Handbook of Clinical Neurology*, 3rd Series (Edinburgh: Elsevier, 2006), p 35.

<sup>52</sup> Johanek, Shim & Meyer, *supra* at 35.

<sup>53</sup> Meyer, Ringkamp, Campbell & Raja, *Peripheral mechanisms of cutaneous nociception*, in McMahon & Koltzenburg (Eds), *Wall and Melzack's Textbook of Pain (5th ed)*, (Elsevier, Edinburgh, 2006), p 13.

<sup>54</sup> Meyer et al, *supra* at 13.

<sup>55</sup> Criminal Justice Information Center Crash Statistics, [http://www.michigan.gov/documents/msp/2013\\_Year\\_End\\_for\\_WEB\\_459459\\_7.pdf](http://www.michigan.gov/documents/msp/2013_Year_End_for_WEB_459459_7.pdf)

<sup>56</sup> <http://www-nrd.nhtsa.dot.gov/Pubs/811552.pdf>, p 1. Estimates for 2011 and 2012 are 2,243,000 and 2,239,000, Appendix A.

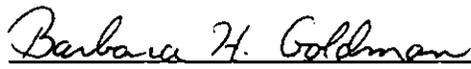
of the motor vehicle involved, but report 526 injuries from accidents with police cars, ambulances and firefighting equipment and 221 involving school buses.<sup>57</sup>

MCL 691.1405 provides that “[g]overnmental agencies shall be liable for bodily injury . . . resulting from the negligent operation by any officer, agent, or employee of the governmental agency, of a motor vehicle of which the governmental agency is owner . . .” Assuming that 2013 is a representative year, therefore, approximately ten percent of those injured in Michigan motor vehicle accidents are potential claimants under MCL 691.1405. It would be illogical to conclude that the Legislature, in enacting the statute, did not envision that damages could be awarded for “bodily injury” but excluded damages for “pain” and “suffering.”

There is no basis for holding that “pain” and “suffering” are not “bodily injury” within the meaning of the motor vehicle exception to governmental immunity.

#### **RELIEF REQUESTED**

Amicus curiae Michigan Association for Justice respectfully asks that this honorable court REVERSE the April 2, 2013 opinion of the Court of Appeals.

  
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<sup>57</sup> Criminal Justice Information Center Crash Statistics, *supra*.



Schulz, *Good Ol' Charlie Brown* (New York: Holt, Rinehart and Winston, Inc., 1957.)