

# WHIPLASH INJURIES: TREATMENT WITH PROLOTHERAPY AND A NEW HYPOTHESIS

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## Introduction

Whiplash injuries have constituted a minority of sprains in most practices dealing with musculoskeletal problems. Severe cases have been intractable clinically and have been somewhat enigmatic to the health care professionals charged with managing them. In many of these cases the severity of claimed pain and dysfunction has seemed disproportionately large. This paper describes the management of these injuries by a technique called prolotherapy based on a protocol developed by M.J. Ongley about 20 years ago. The author encountered this style of treatment from the originator and has incorporated it into his practice since 1984. The impression that the technique is effective has been subjected to three retrospective surveys based on this practice. This paper describes the findings of these surveys cumulatively. Based on the success of treatment of ligaments in the posterior aspect of the neck, a new hypothesis is advanced to explain the enigma of whiplash injuries in the discussion section of the paper.

## Materials and Method

### Patients

The practice on which this survey was based is a single handed internal medicine practice, in California between 1977 and 1996, and in Washington State between June 1996 and September 1998, the time of completion of the last portion of the survey. The practitioner developed an interest in musculoskeletal conditions and has contributed research in this area, accordingly he receives referrals of complex cases considered to have ligament injuries. Amongst these cases there are many with whiplash injuries. The practice has been subject to retrospective surveys on three occasions, i.e., in August 1987 by a nurse, in April of 1995 by two college students conducting a senior project at a California State University, Cal Poly,<sup>1</sup> and in August of 1998 by a nurse at the present practice location, Tahoma Clinic, in Kent Washington State. Surveys were conducted by mail with questionnaires on all consecutive cases treated during the time frames of the surveys, but not the whole period of the use of this method was surveyed. No survey was conducted from mid 1995 to mid 1996. The first two surveys focused on low back conditions treated with prolotherapy, but information was gleaned on neck conditions at the same time. The first survey has been published.<sup>2</sup> The third portion of the data, i.e., that collected from the practice in Washington State, since its relocation to this site, surveyed neck conditions only, in preparation for this report. The data are presented

cumulatively, however. The number of patients involved are: 17 until August 1987; 14 until May 1995 and 17 until August 1998 making a total of 48 patients. The demographic data are presented in table 1. Table 2 indicates the modalities of treatment which had been attempted unsuccessfully prior to treatment with Ongley's technique. The duration of chronic pain prior to treatment is summarized in Table 3. A clear account of a whiplash situation in a rear-end automobile traffic accident was elicited from almost all the cases. Unfortunately, a detailed tally of the mechanism of injury was not recorded in the initial surveys.

Table 1. Demographics

Men	17
Women	31
Age 20-29	9
Age 30-39	13
Age 40-49	17
Age 50-59	4
Age 60-69	4
Age >70 years	1

Table 2. Modalities of treatment which had failed before prolotherapy was undertaken.

Modality of Treatment	Number of Cases
Chiropractic	32
Traction	15
Acupuncture	13
Collar	11
Physiotherapy	30
Surgery	4
Other	14

Table 3. Duration of symptoms prior to starting treatment with prolotherapy

Duration	Number of patients
>5 Years	20
2 - 5	13
1 - 2	7
<1 Year	8

### Clinical Routine

Prolotherapy by Ongley's technique was offered to patients who had severe and persistent pain and dysfunction in the neck following ligamentous injuries in whose cases objective findings of ligament sprains could be identified and who had suffered for more than six months and in whose cases less invasive modalities had been tried adequately and the resultant benefit was absent or slight. For details of the diagnostic approach readers are referred to the relevant text.<sup>3</sup>

### Treatment Routine

There follows a generic description of the routine used in these cases. This routine was followed, by and large, but minor modifications were applied as considered appropriate clinically in individual cases. The practitioner did, however, make a major and conscious attempt not to vary the routine over this fifteen year span for the specific purpose of conducting this evaluation.

### Analgesia.

Intravenous light conscious sedation is used and the patient monitored for heart rate, blood pressure and oxygen saturation with a finger oximeter. Sedation is tailored to individual needs, the modal doses are 3mg. of Medazolam and 25mg. of Ketamine.

### Injection technique - Local anesthesia.

Ten cc disposable syringes and a 19 gage 3 inch disposable hypodermic needle are used. The initial step is one of infiltrating 0.5% lidocaine into the ligamentum nuchae, intertransverse ligaments, zygoapophyseal joint areas and over the laminae of the cervical vertebrae so that some infiltration is directed towards the ligamenta flava, but no attempt is made to pass the needle anterior to the laminae in order to avoid damaging essential structures. The injection technique calls for skill and care for the risk of penetrating the spinal canal is a hazard to be meticulously avoided. The cervical nerve roots enveloped in their dural sleeves are occasionally inadvertently touched or impinged upon by the needle. The patient will report a sharp pain with radiation. When this occurs, the needle should be withdrawn without injection, with experience and care this misadventure is

infrequent. The infiltration is performed by droplet injection at each needle tip placement site. The volume placed at each site should be no more than 0.2cc, so that multiple repositioning of the needle tip is called for. The volume of local anaesthetic used varies up to a total of 50cc.

The skin of the back of the neck is prepped (sterilized) and the neck fully flexed by an assistant. The operator needs to place himself in a comfortable position, and it is usual to ask the assistant to place her hand across the top of T1. By grasping the assistant's index finger with the operator's left hand and using the 5th digit for palpation of the spinous processes in the midline, the operator has a good three-dimensional orientation and control. It is helpful to visualize the cervical vertebrae as if 'with x-ray eyes' and in particular to form a careful assessment of the depth of the cervical structures. After anesthetizing the skin over the spinous process of C2, the needle is introduced and contact made with the tip of the spinous process in order to gauge its depth. The needle is now 'walked' down across the side of the spinous process and across the lamina of C2 to the end of the transverse process on one side and then the other. By this 'walking' process, the depth of penetration is gauged from step to step so that if, inadvertently, the needle slips off the edge of the bony structure, the operator avoids passing it deeper than intended. By slight repositioning, the operator can re-identify the bony landmark. A similar process can be repeated along the transverse arch of the atlas. It should be remembered, however, that the atlas does not have a well-formed spinous process, and the risk of inadvertently passing beyond it towards the dura is greater at this level, at about 4.5cm depth. The needle is directed cephalad and anteriorly almost parallel to the skin of the flexed neck, this allows the operator to sense the depth more accurately without significant risk. It should be remembered that the vertebral artery courses posteriorly to the lateral aspect of the transverse process of the atlas and is at risk at this site. Next, the needle is angled inferiorly towards the spinous process of C3 and the 'walking down' process repeated bilaterally at this level. A separate needle insertion point at C5 is used for the C4 through C7 vertebrae. When aiming to insert a needle vertically towards the lamina in a parasagittal plane one might be tempted to withdraw the needle and reinsert it through a separate skin puncture. This can be avoided by the 'needle slide' technique, which consists of withdrawing the needle tip to the level of the sub-dermis, then sliding the skin with the needle tip sideways to the desired parasagittal plane, and reinserting it through the deeper structures more or less vertically. In addition to limiting the number of skin punctures, this maneuver increases accuracy and speed. While aiming the needle caudad towards the lamina and transverse processes of C7, which are broader structures, it should be remembered that the apex of the process is situated just in front of these bony landmarks. It is therefore advisable to sense the bony landmark and not insert the needle into soft tissue anterior to it, and inject only when in contact with bone. With this precaution, the complication of pneumothorax can be avoided reliably.

After having sensed the ligament consistency with the needle and noting the degree of pain provoked the clinician makes an assessment of the degree of inflammation present. When the

is thought to be significant the area is 'dis-inflamed' with triamcinolone mixed in with another 10cc of 0.5% lidocaine also by droplet injections into the area involved. The dose of triamcinolone may vary from 20-40mg. as judged appropriate.

The patient is asked to lie supine and the manipulation performed after the infiltration.

**Manipulation with Traction - the Lower Segment.**

The manipulative treatment aimed at the lower cervical segment is applied with longitudinal traction and is modelled on Cyriax' teaching. The patient lies supine, an assistant holding the lower limbs. The manipulator takes a firm hold of the head with one hand behind the occiput and the other cradling the chin without impinging on the larynx. Body weight is used for traction while the assistant applies a holding counter traction to the lower limbs. When the slack is taken up, and while maintaining traction and with the head in slight extension, rotation and over pressure are applied in both directions. The manipulator needs to sense that a full range of movement is achieved in both directions.

**Manipulation - the Upper Segment.**

The manipulation is aimed at C1 to C3. It is intended to mobilize the zygoapophyseal joints into a full range, overcoming any 'hang ups' which might restrict their full range of motion. The maneuver is performed in both directions. Assume a rotation to the left first with the patient lying supine. The manipulator stands on the patient's right facing the neck and head. He places his left forearm under the head. The head is slightly flexed in the saggital plane, rotated fully to the left and then side bent to the right. This brings it a little forward. The head rests on the operator's left forearm and his hand is cupped over the chin. The right hand is placed over the right masoid process, the forearm aligned in the saggital plane. The thrust is given with speed and accuracy to achieve a movement of less than 2mm at the zygo-apophyseal joints of the two upper cervical segments.

**Post Treatment Observation**

The patient is observed to become normally alert after the light conscious sedation (this usually takes ten to twenty minutes) and instructed to report for the first proliferant injection on the subsequent day.

**Proliferant Injections.**

The next day proliferant droplet injections, a total of up to 20cc, is infiltrated in the ligament structures of the cervical spine by the technique outlined for the local anaesthetic injection earlier in this section. The solution used consists of 1.25% phenol, 12.5% glucose, 12.5% glycerine and 0.5% Lidocaine. The operator will have acquired a sense of the ligament structures, the depth of the tissues, the sensitivity and any difficulties on the preceding day with the injection of

the local anesthetic. His familiarity with the anatomy in this particular case will help him in accurate needle placement. This routine is repeated thrice more at weekly intervals, making a total of four proliferant sessions.

**Exercises.**

The patient lies supine with the head on a soft pillow. Pendulum type movements of the head with rotation, side bending and lateral flexion to one side, so that the chin approximates the shoulder, are followed by reversal of the direction to the other side. Fifty of these movements in both directions are undertaken thrice a day.

**Results**

The levels of pain recounted retrospectively by the patients at the time of the surveys, before and after treatment is recorded in Table 4. Table 5 summarizes the patients' assessment of their pain on a five scale verbal score. Table 6 summarizes their reporting of daily living activities. Table 7 summarizes the influence of the treatment on dizziness and tinnitus. Table 8 reports on sleep disturbances.

**Statistical Analysis**

The statistical analysis on the following table (4) was performed by Leslie Bowker, Ph.D. of Cal Poly University, San Luis Obispo, California on the basis of review of the raw data on 45 of the cases. (Unfortunately the original forms of three of the subjects were incomplete in one detail or another.)

At the time of the surveys, patients were requested to recount their perceived level of pain before and after treatment. Subjective values of pain intensities were based upon a ten-point scale with 10 indicating extremely severe and 1 indicating no pain. The results are tabulated in Table 4.

Table 4: Perceived Levels of Pain Reported by Patients Retrospectively

	Perceived level of Pain After Treatment										TOTAL	
	Severe ←————→ None											
	10	9	8	7	6	5	4	3	2	1		
Perceived Level of Pain Before Treatment	10	4				3	5	1		8	3	24
	9	1						4	3			8
	8					1	2	1	2			6
	7			1					2	1		4
	6				1				1			2
	5								1			1
<b>TOTAL</b>	<b>5</b>			<b>1</b>	<b>4</b>	<b>6</b>	<b>3</b>	<b>5</b>	<b>17</b>	<b>4</b>		<b>45</b>

The average reduction in subjective pain was analyzed by subtracting the pain score after treatment from the pain score before treatment. The resulting values indicate the level of improvement. A paired t-test on the resultant data indicated a significant level of improvement at the .05 level of confidence.

#### Paired t test for level of improvement

Sample size	Mean Improvement Score	Standard Deviation	Standard Error of the Mean	T value	p
45	5.089	2.778	0.414	12.29	<0.0001

Table 5. Patients' assessment of level of pain using a verbal score.

Severity of pain	Before	After
Slight	0	17
Limited	0	6
Moderate	6	16
Severe	19	3
Very Severe	23	6

Table 6. Patients' retrospective assessment of daily living activities.

Daily Living Activities	Before	After
Normal	1	9
Slightly Limited	0	19
Limited	14	10
Restricted	21	8
Debilitated	12	2

Table 7. All patients were asked about the following symptoms. The positive reports are summarized.

Symptom	Before	After
Dizziness	15	7
Ringings in the Ears	11	5

Table 8. Some patients reported a sleep disturbance:

Severity	Before	After
None	3	6
Slight	7	14
Extensive	18	7

#### Complications

A single complication was recorded during this time of treatment and it occurred in 1986 when the practitioner was, presumably, less skilled than subsequently. The (female) patient reported severe pain in the distribution of C7 on one side during and immediately after an injection. Severe dysesthesia in this distribution developed and subsided gradually over the course of six weeks. There was no neurological defect on follow up over that time. It is thought that the injecting needle inadvertently impinged on the dural sleeve of that nerve root. Information from this patient is included in the initial survey.

#### Follow up

The period of follow-up varied. The first two surveys reviewed cases in whom treatment had been finished at least six months before the survey time. In the last survey this period was reduced to three months. The longest follow up is five years and the median is estimated at two years.

#### Discussion

##### Origins of the Term Whiplash and its Present Importance

Discussion of the term whiplash occurs in the literature from 1945<sup>4</sup>. Though it is said to have been used first by a Los Angeles orthopedist, Dr. Harold E Crow.<sup>5,6</sup> Like a slogan, the term is intuitively meaningful. It has, however, been subject to pedantic argumentation almost from the time it was coined.<sup>7</sup> There does not exist in the United States a uniform recording method which would allow the assessment of the frequency and expense from whiplash injuries, apart from sprains and strains in general. In Britain an increasing incidence of these injuries has been documented since the advent of compulsory use of seat belts.<sup>8,9</sup> It is estimated that 15-30% of car occupants examined soon after their accidents have sprains in the neck<sup>10</sup> and when evaluated later the incidence rises to 60%.<sup>11</sup> It is apparent from the 1988 census data from 14 states accumulated by the Bureau of Labor Statistics of the U.S. Department of Labor that neck injuries constitute approximately 3.5% of the total, and it is known that sprains and strains constitute the largest group of injuries responsible for the escalating insurance costs for American employers. It has been estimated informally that work-related cervical sprains constitute no more than a quarter of the whiplash injuries for which insurance claims are made in this country.

## Clinical Features

*Whiplash* being an imprecise term, for the purpose of this paper it was applied to patients in whose cases the important clinical findings are:

An account of neck and head pain coming on hours and up to two weeks after being subject to an extension-followed-by-flexion injury of the neck as occurs when a stationary car is struck from behind in traffic.

Additional characteristics included:

1. Cases in which neurological (hard signs) and bony injuries have been excluded. (The presence of a neurological defects or fracture does not protect an individual from associated ligament injuries, but in these cases other considerations take precedence early in management and may obscure a pure diagnosis of *cervical soft tissue whiplash injury*.)
2. Many of these injured people report pain from other portions of the axial skeleton later.
3. They often report *posain* and *nulliness*.<sup>12</sup>
4. They frequently improve with manual therapy.
5. Those who fail to improve with manual therapy usually report temporary intermittent improvement with manual therapy.
6. Clinical examination shows a partial articular pattern of cervical movement (Cyriax).<sup>13</sup>
7. Firm palpation discloses tenderness over ligamentous attachments along the spinous processes and transverse processes (and lateral mass of the atlas) and zygoapophyseal joints, usually asymmetrically, in keeping with the recognized
8. Somatic dysfunction which they exhibit when examined by osteopathic techniques.<sup>14</sup>

## Severe Injury

The main factors have been reviewed.<sup>15,16,17</sup> That injury to the soft tissues occurs is not in dispute in cases of *severe collisions*. Many questions remain, however, in lesser impacts.

## A Paradox

Front-end collisions are more frequent than rear-end collisions. The clinical problem with whiplash injuries is much more common in rear-end impacts.<sup>18,19</sup> The paradox has been attributed, cynically, to guilt and litigation. In the rear-end collision, extension of the cervical spine occurs first. The flexion which follows, *the whip*, is the secondary motion attributed to the combined elasticity of the soft tissues of the neck and response of vehicular components, particularly the upright of the seat. Nonetheless, it is the posterior cervical

elements which are the source of the posain and nulliness and perchance those injured Why?

## Another Paradox

Sprains and strains, elsewhere in the body, usually recover. The posterior cervical *whiplash* injuries often persist.<sup>20,21,22,23,24,25,26,27</sup> The duration and severity of the symptoms exceed expectations by a large margin.<sup>28,29</sup> It has been noticed that associated ankle, knee, shoulder and other sprains from the same accidents recover more than the cervical whiplash.<sup>30</sup> In traffic courts in the United States rear-end collisions are charged to the responsibility of the driver of the rear vehicle. This might tend to increase litigation and claims by the "innocent" victim of the front vehicle. Nonetheless, in jurisdictions where no fault-insurance pertains, the *whiplash paradox* is observed as well. Accordingly, it is the *working assumption* of this paper that research should be directed to elucidating causes for the *whiplash syndrome* which are peculiar to this circumstance and differ from other sprains and strains.

## Clinical Considerations

The persistent clinical observation that drivers of vehicles who have been struck from the rear at low velocity become *whiplash patients* in whose cases injuries would not have been expected based on an initial analysis of the forces concerned is a clinical observation. It is proposed that we should look first at the known and probable factors responsible for the presumed injuries by way of a global analysis that is to say, regarding the forces and vectors affecting the head, neck and trunk in toto, and next consider a reductionist analysis of possible mechanisms which might be *specific* to the dysfunction of the human axial skeleton and its motion segments. It is proposed to review the possible contributions of factors controlling cervical movement in the light of mechanisms which have been recognized elsewhere in the body. These include: asymlocation,<sup>31</sup> self-bracing,<sup>32</sup> sacral entrapment<sup>33</sup> and persistent somatic dysfunction.<sup>34</sup> The not infrequent association of low back injury in whiplash cases has been noticed before<sup>35,36</sup> and also commented on in a recent textbook.<sup>37</sup> This association is thought to represent an *essential feature* of the *tensegrity model*.

## Physical Considerations

1. The acceleration of the front driver's head is greater when the mass of the rear vehicle is larger, and when the shock-absorbing characteristic of the front vehicle is deficient, both in chassis and seat-upright.
2. It is thought that the energy dissipated in the front driver's neck might be proportionate also to the duration of impact that is to say, the duration during which acceleration is imparted from the rear to the front vehicle (usually measured in msec).

### Muscles as Shock Absorbers

Large muscles serve as decelerators. This is also called eccentric contraction. From electromyographic studies we know that in a balanced position, including sitting upright, when not in motion, muscle activity is minimal. The human frame is balanced vertically through tension in ligaments and compression of the bones. It is likely that cervical muscles decelerate the head in whipping injuries when the subject has warning. In the case of front-end collisions the individual about to be injured is usually warned visually. In rear-end collisions the forces are thought to be dissipated in the noncontractile soft tissues of the neck (ligaments and fasciae) before reflex muscle action comes into play. It might be, therefore, that a force dissipated in the neck in this *unguarded* circumstance is more injurious overall. This supposition raises the need for electromyographic (EMG) studies during simulated rear-end impacts.

### Injury Assessment

The characteristic scenario in these cases is of an attendance in an emergency room, the exclusion of bony fractures by x-ray, of hard neurologic deficit through examination, and dismissal, often with cervical immobilization, analgesics and muscle relaxants. The large (but incompletely documented) proportion of victims whose problems persist and to whom the appellation *whiplash injury* is subsequently applied are considered not to have *pathological lesions*, by which it is meant histologically provable ligament or muscle disruption, tearing, inflammatory reactions, let alone neurological disturbances or bony fractures. Apart from the clinical features outlined above, the presence of inflammation is usually blocked by the use of nonsteroidal anti-inflammatory agents. From cases which have been evaluated and in whose circumstances autopsies were performed and in whose cases by definition the injuries were very severe, there has been shown an extraordinary disparity between the clinical and imaging evaluation of the trauma and the observed autopsy findings.<sup>38</sup> It follows that in nonfatal, and hence lesser injuries, the standard clinical approach and imaging techniques, as available contemporaneously, are weak tools indeed. We are forced, therefore, into a situation where it is necessary to postulate mechanisms of injury based on our general information and necessarily dispensing with the reductionist requirement of demonstrated histological lesions. A recent study<sup>39</sup> has shown that in low speed rear impacts the range of movement of the head has been within the physiological range in flexion and extension. A compressive force was demonstrated, however, so the forces were most *unphysiological*. Perhaps this disorganized movement is disruptive? Perhaps one or more zygoapophyseal joint capsule passes the threshold from repairable damage to inevitably progressive disruption jeopardizing future *integrated function* of the cervical spine?

### Somatic Dysfunction

*Somatic dysfunction* is a term used in osteopathic circles,<sup>40</sup> recently replacing the time honored, but supposedly less precise term *osteopathic lesion*. Asymmetric palpatory findings in the axial skeleton, associated in recent years with recognition of asymmetric motion restrictions, is due clearly to *soft tissue dysfunction*. The most common clinical finding in patients who have the whiplash syndrome is of joint dysfunction.<sup>41</sup> This has also been documented with cineradiology.<sup>42</sup> The reductionist definition of this *dysfunction* is signally absent and a repertoire of explanatory theories and hypotheses has grown around this absence, most easily lumped together as the *trigger point cult*. It has been suggested that persistent altered tension in muscles following the *jolt syndrome* is responsible.<sup>43</sup> The notion of persistent reflexes was subject to laboratory research earlier.<sup>44</sup> The weakness in this hypothesis is, in this specific context, that the rear-end whiplash injury is so common. One might ask why this should this phenomenon not apply to the victims of front-end collisions?

### Subtle Ligament Injuries

Ligament relaxation is a term that was introduced by Hackett<sup>45</sup> and with the addition of the approach taught by Cyriax<sup>46</sup> has served as a framework for making a diagnosis in the treatment of axial human soft tissue injuries using traction, manipulation and prolotherapy with success.<sup>47</sup> These treatments are predicated on the assumption of ligament *relaxation* or injuries which have hitherto gained little credence in the broader medical milieu. Even when reviewers discuss the controlling effect of ligaments, the recognition of the need for treatment of this tissue is neglected.<sup>48</sup>

### A New Hypothesis

#### Reflex Muscle Contraction

Reflex contraction of muscle after a startle occurs within about a fifth of a second. This is also the time sequence of the whipping. Is it possible that early muscular contraction is paradoxically harmful? As the initial injury is that of cervical extension, it might be expected that the flexors would contract, due to the stretch reflex, first. It is not unlikely that this contraction coincides with, the elastically induced, forward whip of the neck, further enhancing the flexion. The secondary forward acceleration of the head was observed early,<sup>49</sup> confirmed,<sup>50</sup> and labelled *phase II* recently. There is an account of modelling that with the initial impact the head, about to be whipped, moves forward first.<sup>51</sup>

#### Reflex Inhibition

After a stretch reflex has occurred, at any spinal level, there is a latency period, of about 400msec, during which it cannot be elicited again. There have been some suggestions that the

latency can affect adjacent reflexes at the same level. It would be of interest to evaluate EMG tracings in experimental whiplash simulations with this question in mind. With the rotational and oblique injuries the same question applies, though the muscles involved vary. This raises a question regarding the characteristics of neck muscles. Neuromuscular units have been classified according to their speed of response and endurance of repetition. The frequency of these units may vary amongst peoples' necks and possibly affect the outcome of whiplash injuries. Young women, for instance, are thought to be more prone to whiplash injuries.<sup>52,53</sup>

### Seat Back Stiffness

Another consideration of the possible role of reflex inhibition arises from the suggestion that with certain stiffness of the seat-back the initial movement of the head is one of *flexion*.<sup>54</sup> If this is so, it might be that in certain conditions reflex contraction of the muscles of extension of the head coincides with the backward throw of the head! This would exacerbate the extending force. Would the phenomenon of latency after a stretch reflex abolish the muscular flexion of the neck on rebound? If so, what are the mechanical and postural factors which promote an initial *flexion* of the head in rear-end impacts? Are they advantageous through serendipity? All these considerations should be tested. Severy's work,<sup>55</sup> however, did not support the supposition of initial flexion. Contradictory observations may be due to different conditions. Perhaps distinction should be made between seat-back *elasticity* and *shock absorption*, rather than between *hard* and *soft* seat-backs. In any case the *elasticity* itself has, one would presume, parameters reflecting the rate of return of the imparted energy (fast and slow spring). One might presume that the *rhythm* of the return of the elastic energy is of great importance. This writer is not aware of an engineering report on this consideration.

### Head Rotation

The chances of the forces in a whiplash injury being dissipated strictly in the sagittal plane are small. Almost all instances are associated with a degree of rotation. Accordingly, the structures affected are multiple.

### Coupled Motion

Peripheral joints characteristically have a hinge or a single plane of movement. A few of the joints in the axial skeleton also have a single hinge or plane of movement such as the atlanto-occipital joint or the lumbar zygoapophyseal joints in a small portion of the range of flexion from 'neutral.' The osteopaths have coined the term *motion segment* to define the relationship between adjacent vertebrae in motion. This is a *misnomer*. The majority of axial skeletal motion is *integrated* to wit, motion is *coupled*. This term is used to mean that due to the tension of the soft tissues, namely the zygoapophyseal joint capsules, ligaments and fasciae, as well as the alignment

of the cartilaginous surfaces of the zygoapophyseal joints, side bending is always associated with (coupled with) rotation and vice versa. Gliding at these flat joints occurs as well, hence the term *facet*. Indeed, with the exception of *dysfunction*, all motions are associated with a degree of motion in adjacent levels. For instance, when the inferior segment of the cervical spine side-bends, rotary motions occur in all the vertebrae. Several studies have been conducted to define these *coupled motions*. The subject has been confounded by terminological disputes and inconsistencies in the findings of various researchers.<sup>56,57,58</sup> The cardinal observation is that the integrated motion is governed by the soft tissues. The contributions to the overall motion from each metamer (motion segment) is variable, hard to predict and subject to anomalies. For this reason generalizations made on the basis of biomechanical studies on particular specimens has yielded poor and conflicting results. When integrated dysfunction is severe it causes symptoms; and graduates to the title *somatic dysfunction*. It might be said that the usual movement of a normal, or optimal, spine is *integrated*. After disruption of the tension in the restraining tissues *asynchronous* motion can lead to *entrapment* of individual segments so that when the neck is subject to a full range of movement increased motion is called for at the remaining motion segments which now become *hypermobile* or loose. This is analogous to the rapid growth of a hole in front of the knee of a pair of trousers, once the thinned cloth begins to give. The gradual wear and tear on the cloth has to advance to a point when tearing begins. Once it begins the progression of failure is rapid. In mechanics the equivalent is called *fault propagation*. In the soft tissues we need to introduce the *concept of failure of the tensegrity*<sup>59</sup> model.

### Asymmetric Entrapment

Ligament *relaxation* has been shown to be responsible for pelvic dysfunction, the sacrum being entrapped asymmetrically between the ilia<sup>60</sup>. Can a similar phenomenon be ascribed to the cervical spine? It has been postulated that due to the three-joint complex of the axial skeleton in the upright or weight-bearing posture characteristic of humans this phenomenon can occur at any level. From a review of the anatomy, of the upper cervical segment this seems so. The zygoapophyseal joints of the atlas and axis are not matched, as would be expected in *form closure*<sup>61</sup>. The inferior facet surfaces of the atlas and the upper surfaces of the zygoapophyseal joints of the axis are convex to convex. This is responsible for side shifting or rotation of the atlas in weight bearing. One might anticipate that ligament relaxation will enhance this phenomenon and that through the integrated relationship with the other cervical segments the tendency to asymmetric alignment and function would be exacerbated. This relationship has been referred to as *the tensegrity model*.<sup>62,63</sup>

## Conclusion

If the mechanism of injury proposed here is correct, i.e., that the sterno-cleido-mastoid muscles translocates the head forward due to reflex contraction, in turn due to the forceful and unguarded extension of the head from the initial forces in whiplash situations one would expect a shear force to stress the end-plate attachments of the intervertebral discs at the inferior cervical segment. The capsules of the zygoapophyseal joints at these levels, as well as the other ligaments and fascia controlling the neck might be expected to be subject to shearing forces and tear at this level. These are the tissues which are subject to ligament hypertrophy with prolotherapy. It is concluded that the improvement in these patients confirms the above hypothesis regarding the mechanism of injury.

## Summary

Received opinion has it that minor whiplash injuries are trivial. They are not.

The success of treatment with prolotherapy, as described here confirms the hypothesis that the posterior ligamentous structures of the human cervical spine are subject to an unusual mechanism of injury in the unique circumstance summarized by the term whiplash. It behooves the medical profession to take the account of victims of this injury seriously and offer them appropriate recognition and treatment.

## References:

- 1 Iseminger T, Palaikis R. A Retrospective study of prolotherapy. Biological Science Dept, College of Science and Mathematics, California Polytechnic State University, San Luis Obispo 1995.
- 2 Dorman T. Treatment for spinal pain arising in ligaments - using prolotherapy: A retrospective survey. *Journal of Orthopedic Medicine* 1991 Vol 13 No1. P13-19
- 3 *Diagnosis and Injection Techniques in Orthopedic Medicine.* Dorman T, Ravin T. Williams & Wilkins. Baltimore MD 1991.
- 4 Davis, AG. Injuries of the Cervical Spine. *J Am Med Assn* 1945;127:149-156.
- 5 Knepper WE. The genesis, growth and destruction of the "whiplash" myth in the continuing revolt against whiplash. The Defence Research Institute, Inc. Milwaukee, Wis, Feb 1964.
- 6 Janes JM. Severe extension-flexion injuries of the cervical spine. *Mayo Clinic Proc.* :40:353-368. 1965.
- 7 Seletz, E. Whiplash Injuries: A New Concept and the Medicolegal Implications. *Medical Trial Technique Quarterly Annual*, 1960.
- 8 Rutherford WH, Greenfield AA, Hayes HRM, Nelson JK. The medical effects of seat belt legislation in the United Kingdom. London:HMSO, 1985.
- 9 Porter KM. Neck sprains after car accidents (editorial). *Br Med J* 1989;298:973-974.
- 10 Larder DR, Twiss MK, Mackay GM. Neck injury to car occupants using seat belts. In 29th annual proceeding of the American Association for Automobile Medicine. Des Plains, Illinois: American Association of Automobile Medicine, 1985: 153-65.
- 11 Deans GT, Magalliard JN, Kerr M, Rutherford WH. Neck sprains - a major cause of disability following car accidents. *Injury* 1987; 18: 10-2.
- 12 Dorman T, Ravin T. *Diagnosis and Injection Techniques in Orthopedic Medicine.* Williams & Wilkins, Baltimore 1991.
- 13 Cyriax J. *Textbook of orthopaedic medicine*, vol. 1. 8th ed. London: Bailliere Tindall, 1982.
- 14 Greenman PE. *Principles of manual medicine.* Baltimore: Williams & Wilkins, 1989.
- 15 Jackson R. *The Cervical Syndrome.* American Lecture Series. Charles C Thomas. Springfield IL. 1978.
- 16 Foreman SM, Croft AC. *Whiplash Injuries.* Williams & Wilkins. Baltimore. 1988.
- 17 Teasell RW, Shapiro AP. *Cervical Flexion-Extension/Whiplash Injuries.* State of the Art Reviews. Hanley & Belfus. Philadelphia. 1993.
- 18 Larder DR, Twiss MK, Mackay GM. Neck injury to car occupants using seat belts. In 29th annual proceeding of the American Association for Automobile Medicine. Des Plains, Illinois: American Association of Automobile Medicine, 1985: 153-65.
- 19 Deans GT, Magalliard JN, Kerr M, Rutherford WH. Neck sprains - a major cause of disability following car accidents. *Injury* 1987; 18: 10-2.
- 20 Balla, JI. The late whiplash syndrome. *Aust NZ J Surg* 1980;50.
- 21 Jackson R. Crashes cause most neck pain. *Am Med News* Dec 1966.
- 22 Jackson R. The positive neck findings in alleged neck injuries. *Am J Orthop* 1964;6:178-181,184-187.
- 23 States JD, Korn MW, Masengill JB. The enigma of whiplash injuries. Thirteenth annual conference proceedings. Rochester: American Association of Automotive Medicine 1969:83-108.
- 24 Hirsch SA, Hirsch PJ, Hiramoto H., et. al. Whiplash syndrome. Fact or fiction? *Orthop Clin North Am* October 1988.
- 25 Norris SH, Watt I. The prognosis of neck injuries resulting from rear-end vehicle collisions. *J Bone Joint Surg [Br]* 1983.
- 26 Baur E. Whiplash injuries of the cervical spine and Swiss insurance against accidents (author's transl). *Radiol Clin* 1975;44.
- 27 Unterharnscheidt F. Pathological and neuropathological findings in rhesus monkeys subjected to -Gx and +Gx indirect impact acceleration. In: Sances A, Thomas DJ, Ewing CL., et. Al., eds. *Mechanism of head and spine trauma.* New York: Aloray, 1986.
- 28 Bring G, Westman G. Chronic Posttraumatic Syndrome after Whiplash Injuries. *Scan J Prim Health Care* 1991; 9: 135-141.
- 29 Gargan FM, Bannister GC. Long term prognosis of soft tissue injuries of the neck. *J Bone Joint Surg.* 72B:901-903, 1990.
- 30 McNabb, I. Whiplash Injuries of the Neck. *Manitoba Medical Review.* 46: March 1966 p172-174.
- 31 Dorman T, Ravin T. *Diagnosis and Injection Techniques in Orthopedic Medicine.* Williams & Wilkins, Baltimore 1991.

- 32 Vleeming A, Stoeckart R, Volkers AC, Snijders CJ. Relation between form and function in the sacroiliac joint. Part I: Clinical anatomical aspects. *Spine* 1990 Feb; 15(2):130-2
- 33 Dorman T. Pelvic Mechanics and Dysfunction. *J Orthop Med.* 2 1994. p45-48.
- 34 Greenman PE. Principles of manual medicine. Baltimore: Williams & Wilkins, 1989.
- 35 Gay JR, Abbott KH. Common whiplash injuries of the neck. *J Am Med Assn.* 152, 1698-1704. 1953.
- 36 Hamel HA, Otis EJ. Acute traumatic cervical syndrome (whiplash injury). *Southern med J* 55, 1171-1177. 1962.
- 37 Dorman, TA. Diagnosis and Injection Techniques in Orthopedic Medicine. Baltimore: Williams and Wilkins, 1981.
- 38 Twomey LT, Taylor JR. The Whiplash Syndrome: Pathology and Physical Treatment. *J Manual & Manipulative Therapy.* 1:1 1993. 26-29.
- 39 McConnell WE, Howard RP, Guzman HM, Bomar JB, Raddin JH, Benedict JV, Smith HL, Hatsell CP. Analysis of human test subject kinematic responses to low velocity rear end impacts. SAE SP-975. 930889.
- 40 Greenman PE. Principles of manual medicine. Baltimore: Williams & Wilkins, 1989.
- 41 Mennell JM. Assessment of residual symptoms from a "whiplash injury". Proceedings of the IVth international congress of physical medicine. Paris 6-11, September 1964.
- 42 Buonocore E, Hartman JT, Nelson CL. Cineradiograms of cervical spine diagnosis in soft tissue injuries. *JAMA* 198: 143-147. 1966.
- 43 Elson LM. The Jolt Syndrome: Muscle dysfunction following low velocity impact. *Pain Management.* November/December 1990 p317-326.
- 44 Korr I. The collected papers of Irvin M Korr. American Academy of Osteopathy, 2630 Airport Road, Colorado Springs, CO 80910. 1979.
- 45 Hackett GS. Ligament and tendon relaxation treated by prolotherapy. 3rd ed. Springfield: Charles Thomas Publisher, 1958.
- 46 Cyriax J. Textbook of orthopaedic medicine, vol. 1. 8th ed. London: Bailliere Tindall, 1982.
- 47 Kayfetz DO, Blumental LS, Hackett GS et al. Whiplash injury and other ligamentous headache - its management with prolotherapy. *Headache* 1963;1.
- 48 Havsy SL. Whiplash injuries of the cervical spine and their clinical sequence, part II. *Am J Pain Management:* 4, 2 73-82. 1994.
- 49 Severy DM, Mathewson JH, Bechtol CO. Controlled automobile rear-end collisions, an investigation of related engineering and medical phenomena. *Canadian Services Med J* 1955;11: 727-259.
- 50 Szabo TJ, Welcher JB, Anderson RD, Rice MM, Ward JA, Paulo LR, Carpenter NJ. Human occupant kinematic response to low speed rear-end impacts. SAE Technical Paper Series 940532 SAE February/March 1994.
- 51 McKenzie JA, Williams JF. The Dynamic behavior of the head and cervical spine during 'whiplash'. *J Biomech* 4:477-490, 1971.
- 52 Deans GT, Magalliard JN, Kerr M, Rutherford WH. Neck sprains - a major cause of disability following car accidents. *Injury* 1987; 18: 10-2.
- 53 Larder DR, Twiss MK, Mackay GM. Neck injury to car occupants using seat belts. In 29th annual proceeding of the American Association for Automobile Medicine. Des Plaines, Illinois: American Association of Automobile Medicine, 1985: 153-65.
- 54 Martinez JL, Garcia DJ. A model for whiplash. *1968 J Biomechanics* 1, 23-32.
- 55 Severy DM, Mathewson JH, Bechtol CO. Controlled automobile rear-end collisions, an investigation of related engineering and medical phenomena. *Canadian Services Med J* 1955;11: 727-259.
- 56 Fryette HH. Principles of osteopathic technic. Carmel: The Academy of Applied Osteopathy, 1954
- 57 Dvorak J, Dvorak V. Manual medicine; Diagnostics. Stuttgart: Georg Thieme Verlag, 1984:6-12.
- 58 Panjabi M, Dvorak J, Duranceo J., et. al. Three dimensional movements of the upper cervical spine. *Spine*;13:726-730.
- 59 Fuller RB. World game lecture series. Philadelphia: Univ Pennsylvania Museum, 1975.
- 60 Dorman, TA. Diagnosis and Injection Techniques in Orthopedic Medicine. Baltimore: Williams and Wilkins, 1981.
- 61 Snijders C J, Vleeming A, Stoeckart R. Transfer of lumbosacral load to iliac bones and legs. Part 1 - Biomechanics of self-bracing of the sacroiliac joints and its significance for treatment and exercise. In Low back pain and its relation to the sacroiliac joint. First interdisciplinary world congress. San Diego, Nov 1992. p233-254.
- 62 Levin SM. The icosahedron as the three dimensional finite element in biomechanical support. A natural hierarchical system. Philadelphia: Presented at the Annual Meeting NAAM, 1986.
- 63 Levin, SM. The space truss: A systems approach to cervical spine mechanics. Presented to the 6th Annual Am Assn Orthop Med. meeting San Diego. Feb 1989.

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