Dr.Sickesz publications

Correction of the anatomical changes of whiplash injury

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Abstract: The upper neck is often neglected during examination. However, pathology of the upper neck vertebrae may result in diverse and sometimes severe signs and symptoms. For the inexperienced, these signs are often difficult to relate to subluxation that has resulted from a minor accident. A whiplash lesion may have serious adverse effects on body function and quality of life. Surprisingly, most of the literature on whiplash deals with the treatment of pain and psychological discomfort rather than anatomical correction of vertebral luxation, which is almost always present in a whiplash lesion. It is our opinion that an actual whiplash lesion will not completely heal if the normal vertebral position is not restored. Immobilisation, traction, or even worse, incompetent manipulation, must be considered as an incomplete cure and will sometimes even aggravate the problems of the patient. This paper details the anatomical changes in whiplash injury and the possibility of adjustment. The work is based on more than 30 years experience with several thousand whiplash patients.
Keywords: whiplash, luxation, cervical anatomy, adjustment

Introduction

Whiplash injury does not necessarily have to be caused by a car accident. Any event in which the head snaps backwards may result in a number of anatomical changes. For not completely understood reasons the whiplash usually occurs on the left side. A whiplash injury primarily affects the anatomical position of C2, which subsequently may affect vertebrae below and above its level.

Krakeness et al (2003) conducted magnetic resonance imaging (MRI) studies in the atlas area with 92 whiplash- injured and 30 uninjured individuals. Their study strongly indicated that whiplash trauma could damage the tectorial and posterior atlanto-occipital membranes; this can be shown on high-resolution MRI. Better knowledge of normal anatomical variations and improved image quality should increase the reliability of lesion classification.

Tencer et al (2003) conducted a remarkable study comparing the sequels of a rear impact on 28 volunteers and 11 cervico-thoracic human cadaveric spines. Head acceleration, individual vertebral kinematics from high- speed video, local nerve root pressure and facet joint contact pressures were collected during impacts. Each specimen was tested first at an impact acceleration similar to that of volunteers, who reported minimal or no symptoms after

unprepared volunteers. Vertebral shear translations showed the largest (and only significant) increases with increased impact acceleration. This data implies that facet shearing was most sensitive to the increased acceleration in this experiment and may be a primary mechanism of cervical spine injury in rear impacts.

Luxation

Luxation is a very complicated process. A thorough understanding of the anatomical changes is essential for proper adjustment. Here, we simplify the *normal* movement of the vertebrae along three axes: anteroposterior (AP) axis, craniosacral or longitudinal (L) axis and the transverse (T) axis through the facet joints (Figure 1). Each movement along an axis includes some shifting translation over the adjoining vertebra.

Abnormal movement of vertebrae includes shifting, which is greater than normal movement along an axis, or shearing, which is not a consequence of normal rotation. This may lead to abnormal rotation or even *locked* luxation of a vertebra, due to interference of the facet joint's cartilage with that of an adjacent facet joint. These abnormal positions are usually *dorsal* to the original position, and translation

the test, then at double the acceleration. Head X (forward) and Z (upward) accelerations of cadaveric specimens were very similar in time sequence and magnitude to those of

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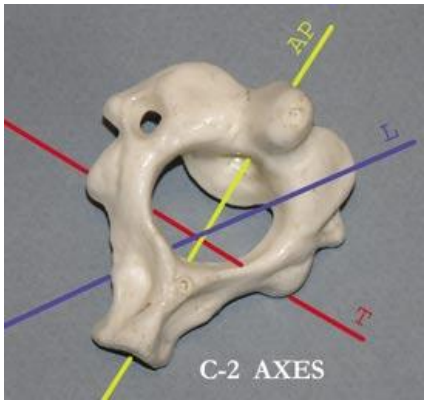

Figure 1 Anteroposterior (AP) axis, longitudinal (L) axis and the transverse (T) axis.

is no longer possible. An abnormal rotated dorsal position is called a rotated dorsal position (RDP). A luxated, locked dorsal position is called a luxated dorsal position (LDP). The typical left-sided whiplash injury has two problems:

1. The left side of C2 slides dorsally over C3, and C2 rotates to the left along the AP-axis. C2 is then in a transverse-dorsal locked position. The cartilage of its lower left facet joint locks onto that of the upper facet joint of C3. Also, anticlockwise rotation along the L-axis is needed to find room to luxate in a dorsal direction. The spinous process moves to the right (seen from dorsal observation), and C2 leans slightly 'backward'. The rotation of C2 along the L-axis may be detected on an anteroposterior, open-mouth cervical x- ray, when relating the position of the spinous processes to the median line. A displacement away from the median line indicates a rotation along the L-axis. An abnormal position of the T-axis may also be observed. The left transverse process can then be felt as a small protuberance, too far dorsal and too low. This can be verified by placing the index fingers on both transverse processes and comparing its positions. The patient will feel the transverse process as a sore bump.
2. C3 and C4 also rotate *anticlockwise* and are then in a RDP (Figure 2).

Late luxation, another lock-up

After approximately ten years, if uncorrected, the weight of the skull will cause C2 to gradually rotate further in an anticlockwise direction, even to the extent that its *right* facet joint becomes locked on or over C3's right facet joint. The body of C2 may be observed to have shifted to the right of the median line. Even for the inexperienced, this is usually well detectable on x-ray.

Figure 2 C3 and C4 in rotated dorsal postion.

Luxation of the atlas

Occasionally, the whiplash injury is accompanied by subluxation of C1, the atlas, relative to C2, a less frequent complication but sometimes with severe consequences. This luxation always occurs in a ventral direction, because the dentoid process of C2 prevents bilateral translation of C1 in a dorsal direction. The luxation usually involves rotation of the atlas along the AP-axis, causing substantial stretching of the transverse atlas ligament. There are two nervous structures in front of the transverse processes of the atlas: the superior cervical ganglion and the vagus nerve on its way from the skull base through the jugular foramen to the peripheral nervous system. A ventrally luxated position of the transverse process of the atlas may interfere with the normal functioning of these structures, which may lead to life-threatening symptoms. Repositioning the atlas often alleviates these symptoms in a matter of days. This extremely interesting phenomenon will be dealt with elsewhere.

Diagnostic procedures

Physical examination The luxated dorsal position of C2 can be verified standing behind the sitting patient. In normal circumstances, the tips of both index fingers, positioned on the transverse processes of C2 will be shown at equal height. With a whiplash lesion either the subluxated left transverse process of the axis cannot be felt anymore, or felt in too low and too far dorsal a position (Figure 3).

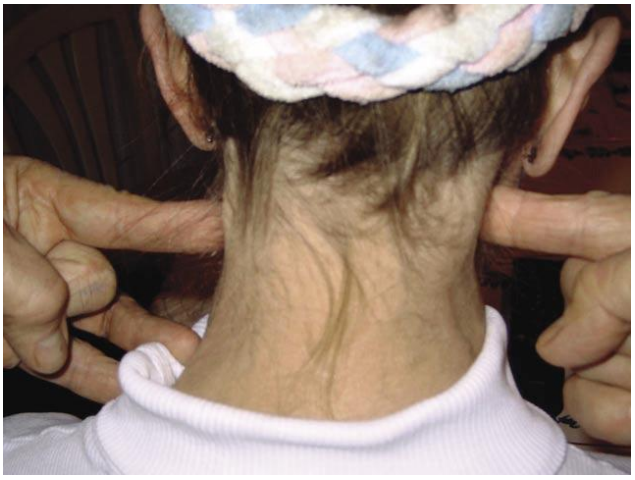

Figure 3 Subluxated left transverse process.

Visualisation: MRI scanning and x-rays

MRI and x-rays are only valuable if the projection is correct and there is sufficient experience to know what to look for.

The attached scan shows a typical uncomplicated whiplash lesion with rotation of the atlas and also rotation along the AP-axis, which can be concluded from the asymmetrical image of the anterior arch, because the left side is not in the same plane of this projection (Figure 4).

One should also note that this scan was made with the patient lying down, eliminating the weight of the head, which tends to accentuate the typical vertebral displacement in the upright position.

The x-ray, according to the Palmer basal-posterior projection will show the atlas rotation along the L-axis when comparing a line, drawn through the vertebroarterial foramen in each transverse process, with a line that

connects the most proximal points of the mandible. Also, any displacement of the dentoid process of C2, in relation to the atlas may be visualised, provided that the skull is projected in an exact symmetrical plane (Figure 5).

Figure 5 Basal posterior view.

The Palmer anteroposterior, open-mouth projection shows axis rotation along the AP-axis and rotation along the L-axis, including C3, by comparing the position of the spinous process relative to the median line (Figure 6).

It is important, in both scanning as well as x-ray, that the skull is *symmetrically* projected and immobilised because only then references to structures such as the mandibles and cheekbones can be made. This is sometimes difficult, because due to the discomfort of the lesion patients tend

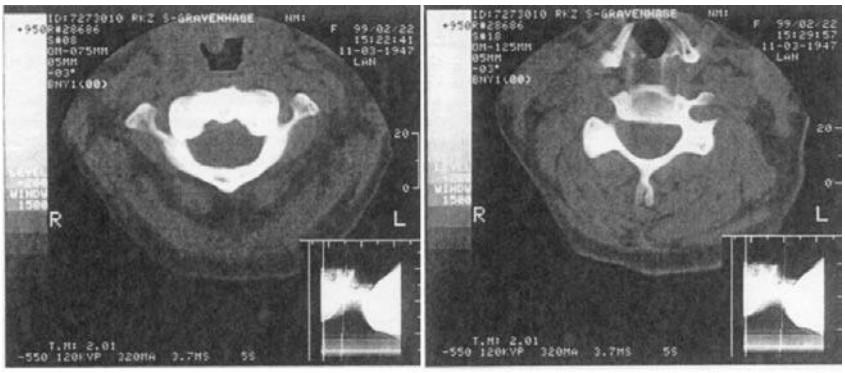

Figure 4 MRI scan of whiplash lesion.

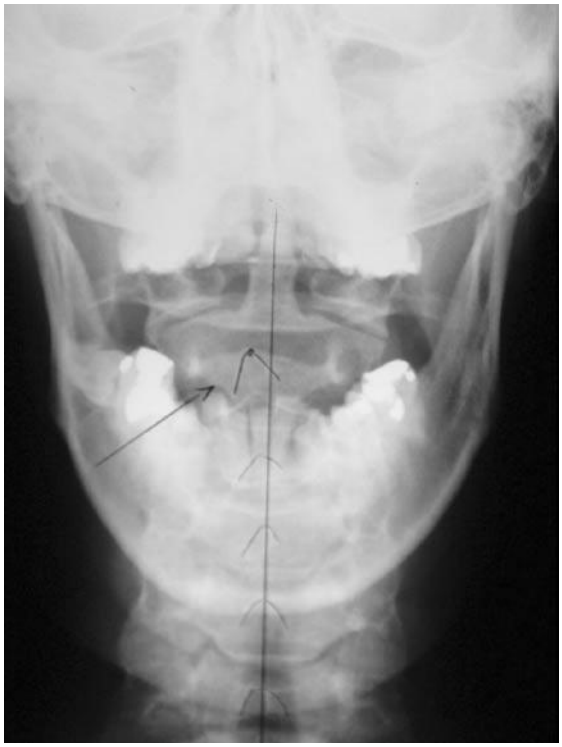

Figure 6 Anteroposterior, open mouth view.

to rotate their head, although to them it feels as though their head is in the 'straight' position. The interpretation of these x-rays requires some familiarisation, even from radiologists.

The adjustment

The following applies to a typical left-sided abnormality. Usually, if the anatomical situation is well understood only light pressure in the *right direction* is needed to correct the abnormal position. This type of treatment is called *ortho*manual therapy.

The adjustment of whiplash luxation includes two sessions, with at least 48 hours in-between, due to the formation of local oedema, which may impede further adjustments.

The objectives of adjustment are:

- unlocking of the LDP of C2, ie changing the LDP into a RDP;
- correction of the RDP of C4 and C3 into neutral position;
- correction of the C2 RDP into the neutral position. The sequence of the adjustments has been empirically determined. It is not always possible to adjust a vertebra in one step, and if adjacent vertebrae are affected, the usual procedure is to start away from the main lesion and work towards it, once the locking-up has been disengaged.

First session

Unlocking C2

C2 is completely locked on C3 (no translation), whereby the left caudal facet joint is locked on the left cranial facet joint of C3; C2 is rotated around the AP-axis towards the left and anticlockwise in the L-axis. It is in the LDP.

1. Place the patient on their *right side* with the head supported by a pillow. Stand behind the patient and use the right thumb to press the spinous process of C2 downwards to the table. C2 will rotate further anticlockwise along the L-axis, which is needed to disengage the left facet joint of C2 from C3 (Figure 7). The luxation is unlocked; C2's position has been changed from a LDP into a RDP. The right facet joint may now become locked on C3.
2. Next, turn the patient on their *left side* and this time, press the right side of the spinous process downwards with the left thumb, which will rotate C2 in the opposite direction and unlock a possible fixation of the right facet joint (Figure 8).
3. Put the patient into the *prone* position with the chin pointing towards the chest and the head in supports attached to the table. Standing at the end of the table, facing the patient's skull, the right

transverse process of C2 is pressed upward (dorsally) with the left thumb, in a first attempt to neutralise the rotated dorsal position of C2. The complete correction is not always possible at this point in time (Figure 9).

Adjustment of C3 and C4

C3 and C4 still need correction. C4, together with C3, were forced by C2 in corrective anticlockwise RDP, which means that the right caudal facet joint of C4 engages the right cranial facet joint of C5 which still is in normal position. Experience has taught that the best sequence is to adjust C4 first, then C3 and eventually C2 again.

1. To adjust C4's position, keep the patient in the *prone* position. Standing at the patient's head, place the left thumb under the right transverse process of C4
   (ie against the ventral side) and press it up, in dorsal direction; then, using the right thumb, press the left transverse process of C4 towards the table, to rotate C4 towards the neutral position (Figure 10).
2. Position the patient on the *right side*, this time without the pillow, with the head hanging down over the shoulder. This will create more room for the facet joint of C3. Standing behind the patient the left thumb presses the left transverse process of C3 in ventral

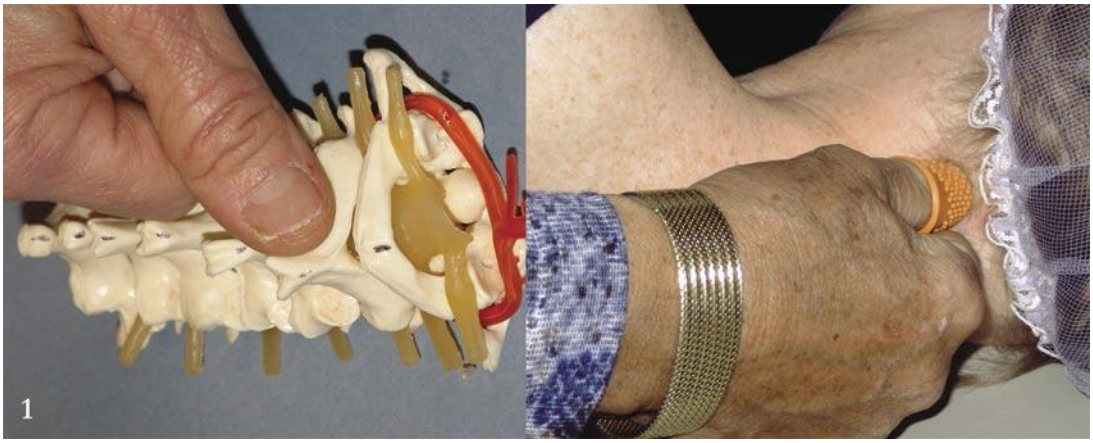

Figure 7 Anticlockwise rotation of C2 along the L-axis.

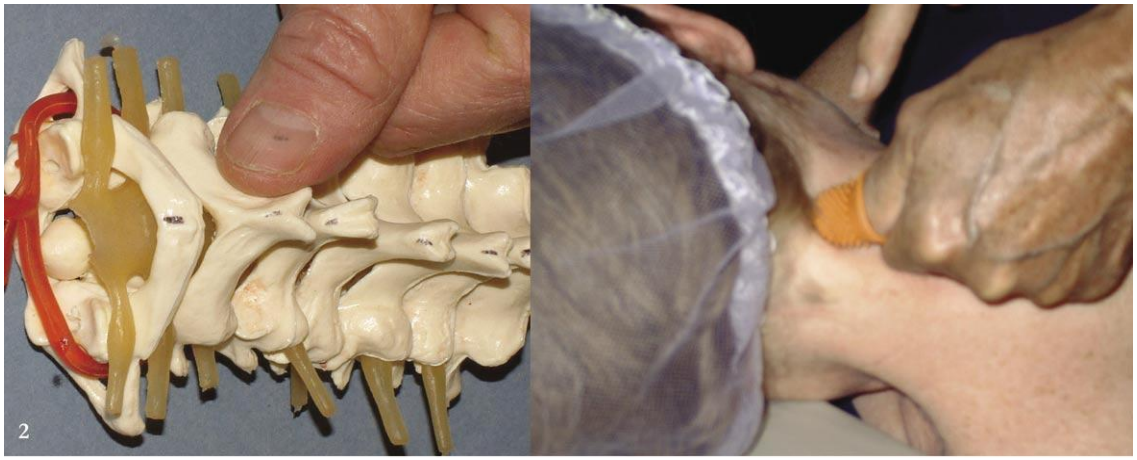

Figure 8 Clockwise rotation of C2.

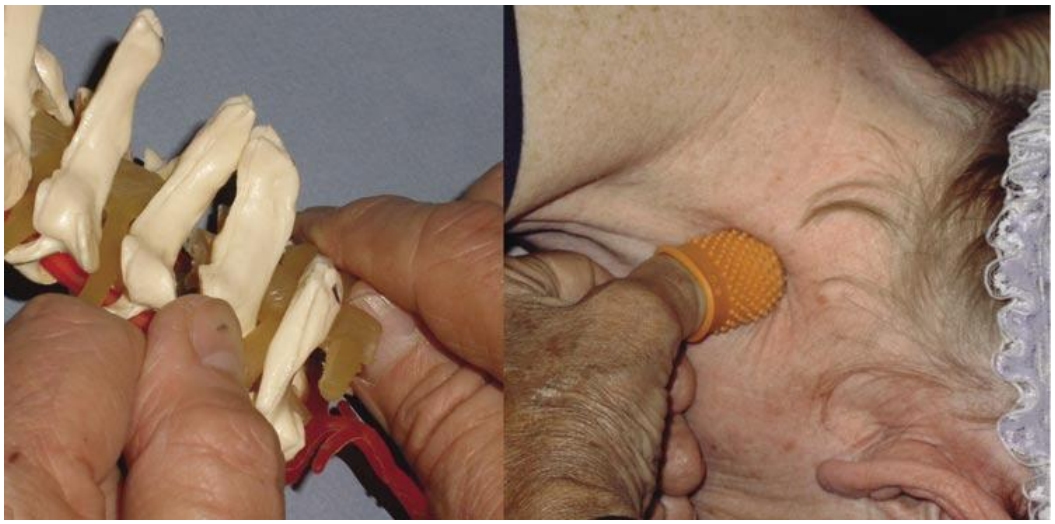

Figure 9 Upward movement of the right transverse process of C2.

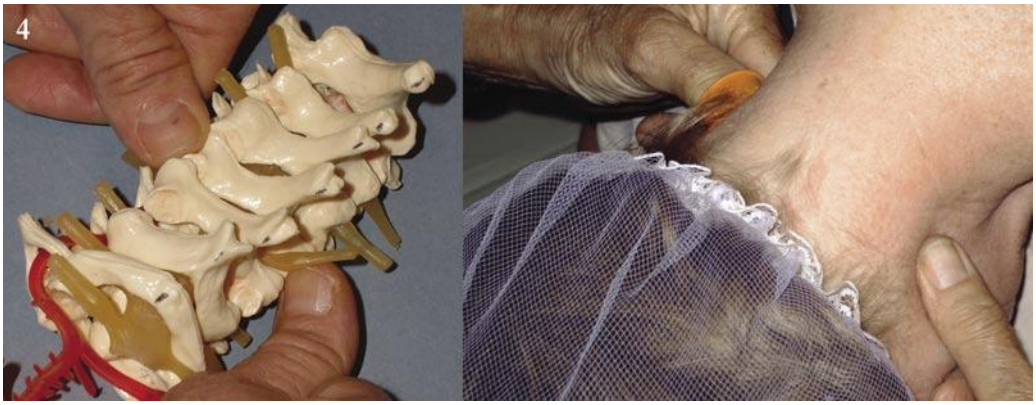

Figure 10 C4 adjustment.

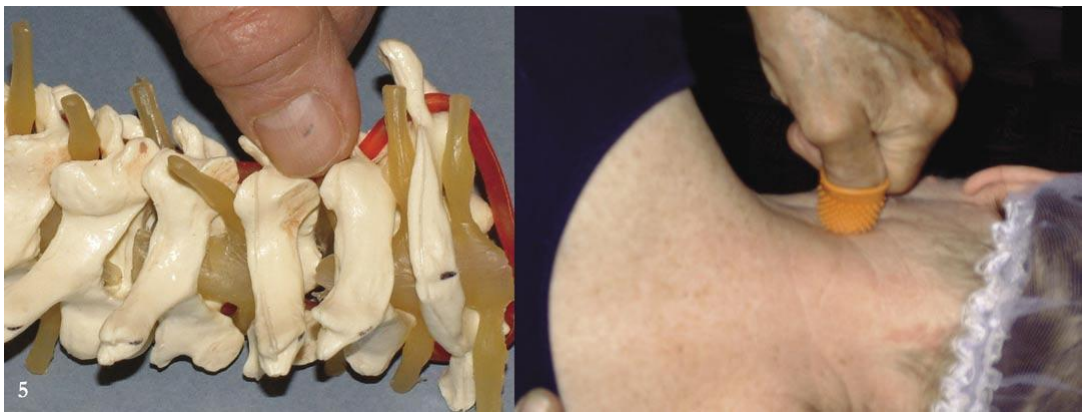

Figure 11 C3 adjustment.

(forward) direction. This is necessary in order to reduce the anticlockwise rotation around the L-axis. Remember, in its LDP, C2 has forced C3 and C4 along in anticlockwise rotation (RDP) (Figure 11).

Second session

The final adjustment of C3 and C4

1. C3 is still in RDP. Position the patient in the *prone position*. The right transverse process of C3 is

pressed upward (dorsally) and the left transverse process downward (ventrally) (Figure 12).

2. Then turn the patient on their *right side* and the transverse process of C4 is pressed in the ventral direction (Figure 13).

For the correction of a whiplash injury that is older than 10 years, the patient is adjusted in the *sitting* position. From behind, the left transverse process of C2 needs to be pressed in a ventral direction because of the more pronounced dislocation (Figure 14).

Adjustment of an atlas luxation

Place the patient in the *supine position*. If available, have an assistant apply light traction to the patient's head and initially press with a thumb the most ventrally dislocated transverse process of C1 in a dorsal direction. If needed, pressure also may be required to be applied on the other side. The transverse process of C1 is situated one centimetre in the dorsocranial direction relative to the mandibular angle, just under the point of the petrosal bone.

If needed, these corrections may be repeated after at least 6 weeks. Stretched ligaments may take a long time to heal and the patient should be instructed to avoid sudden head movements and abnormal forces against the head.

Materials and methods

A common pattern of symptoms may include the following: impaired mobility of the neck; cranial neuralgia (headache on top of the head; the dermatome of C2 is the scalp); occipital neuralgia (the dermatome

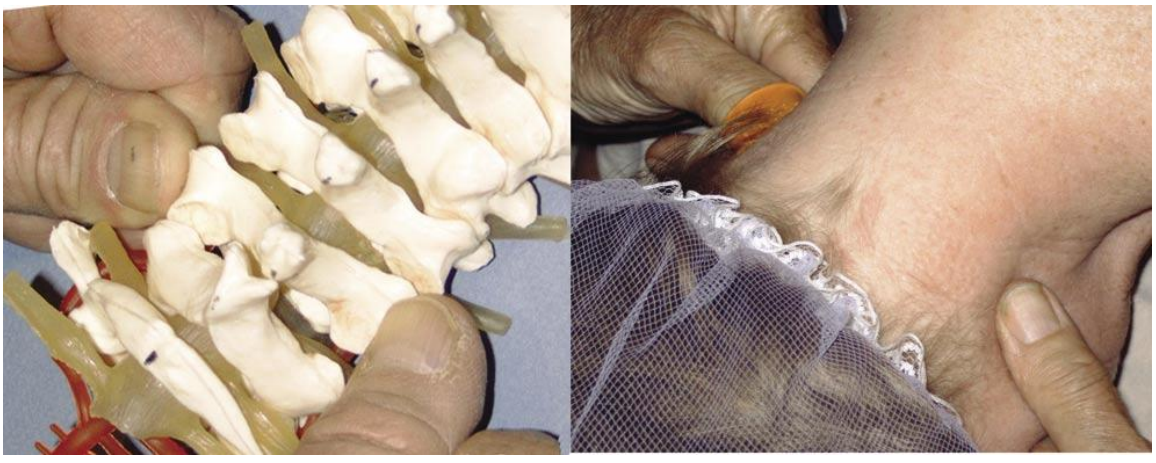

Figure 12 Dorsal and ventral movement to the right transverse process of C3.

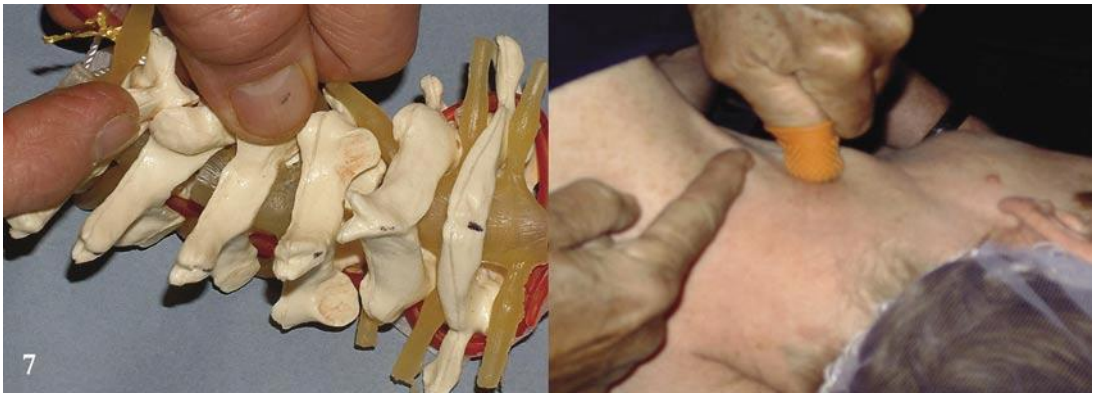

Figure 13 Ventral movement to the transverse process of C4.

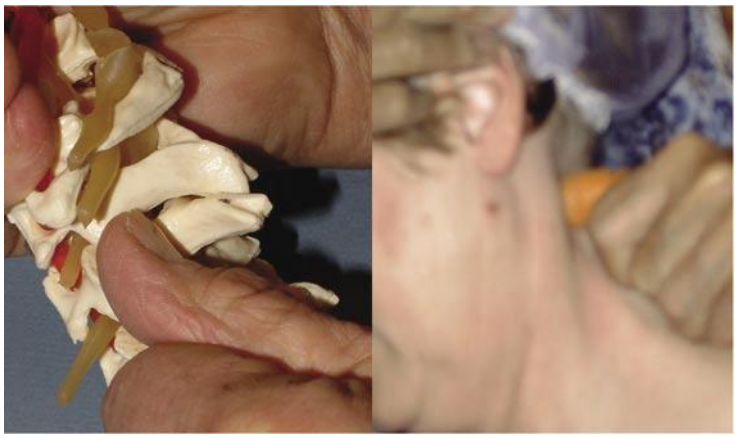

Figure 14 Correction of whiplash injury in the sitting position.

of C3 is the neck and the occiput); and dizziness (the superior cervical ganglion is located against the ventral aspect of the transverse processes of C1 and C2 and might get irritated by an abnormal position). This may

also give rise to vasoconstriction in the brain stem which may cause dizziness, concentration disorders, sleeping problems and tinnitus. Drottning (2003) noticed that in most whiplash studies, the whiplash headaches are not

defined. Post-whiplash cervicogenic headache (CEH) is typically a moderate headache with a benign, but often prolonged, course. It probably is unilateral and bilateral. Reduced neck mobility does not seem to be as common after whiplash as in chronic CEH in general. Post-whiplash CEH is accompanied by great disability and high use of medication. Although the natural course seems favourable for unilateral CEH during the first post-injury years, some patients will need specific treatment for their headaches. There is a lack of controlled studies to guide practitioners to choose investigations and treatment for chronic CEH. Further symptoms include facial neuralgia, one-sided tinnitus, loss of hearing and snoring (as the ventral aspects of vertebrae form the back of the throat). Since vertebral misalignments may cause oedema around the vertebra, the dorsal wall of the throat can also be involved. Oedema, which may even extend as far as the uvula, which may also cause sleep apnoea.

Haldorsen et al (2003) attempted to record the severity of symptoms in patients with late whiplash syndrome and to assess how the symptoms affected performance on a simple psychometric test. The Rivermead Post-Concussion Symptoms Questionnaire (RPQ) with an additional question on neck pain, modified RPQ (mRPQ), and the Short Test of Mental Status (STMS) were applied on 40 chronic whiplash patients. Symptoms reported by the whiplash group were compared with those of 50 normal controls. The patients scored significantly higher than the controls on all symptoms listed in the mRPQ.

Sterling et al (2003) concluded that those with persistent moderate/severe symptoms at 6 months after injury display generalised hypersensitivity suggestive of changes in central pain processing mechanisms. This phenomenon did not occur in those who recover or those with persistent mild symptoms.

Case studies

Forty randomly selected patients, 28 female and 12 male, were interviewed three months after completion of

their treatment.

Symptom studies

Table 1 shows the result of the treatment on the various symptoms.

Discussion

Improperly treated whiplash injury may have a profound effect on the quality of life, and sometimes leaves the patient with long-term disability. An optimal treatment of this condition is the anatomical correction of displaced vertebrae. However, it must be emphasised that all anatomical details need to be perfectly understood in order to conduct the proper correction. Normal function cannot be restored with abnormal anatomical relations. Even in chiropractic literature this is not always clear. Murphy (2000) comments that each individual case of whiplash is different, and it is not possible to generalise about treatment. He stipulates that appropriate treatment strategy is unique to each injury and is directed at the primary dysfunctions detected during the examination. However, chiropractors commonly employ the following treatments: gently moving the involved joint towards the direction in which it is restricted, which may involve the application of a short thrust in that direction. In many cases, instead of a thrust, a slow mobilising movement is used. Our opinion, derived from the adjustment of several thousands of whiplash patients in more than three decades, is that in almost every whiplash patient the typical anatomical lesions can be found, and that, with adequate adjustment, many signs and symptoms may significantly decrease, even several decades after injury and in older age groups. It is also evident that every additional treatment for pain and other discomfort will only have optimal results if the anatomical lesion has been corrected. It is therefore

Table 1 Response of symptoms to treatment

Symptom	*Number*	*Eliminated*	*Improved*	*Unchanged*
Cranial neuralgia	16	15 (94%)	1	–
Occipital neuralgia	28	23 (82%)	5	–
Dizziness	31	23 (74%)	5	3
Concentration disorders	28	14 (50%)	9	5
Sleeping disorders	22	13 (59%)	2	7
Restriction of mobility	32	28 (88%)	4	–
Snoring	25	8 (32%)	15	2
Sleep apnoea	18	8 (44%)	8	2
Facial neuralgia	7	5 (71%)	2	–
Tinnitus	11	6 (55%)	3	2

treatment. Their age distribution was:

10–20 years, 2 patients 20–30 years, 6 patients
30–40 years, 9 patients 40–50 years, 7 patients
50–60 years, 11 patients 60–70 years, 2 patients
70–80 years, 3 patients

The duration of symptoms was:

< 5 years, 22 patients 5–10 years, 8 patients
10–20 years, 6 patients 20–30 years, 4 patients

surprising to read that French et al (2003) conducted shoulder stabilisation and brachial plexus neurolysis in
800 patients with chronic whiplash syndrome, without mentioning if they tried to correct the typical anatomical whiplash lesions in the first place.

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