Whiplash Injuries

Diagnosis and Treatment

Second Edition

Dario C. Alpini · Guido Brugnoni
Antonio Cesarani *Editors*

Springer

Whiplash Injuries

Dario C. Alpini • Guido Brugnoni
Antonio Cesarani
Editors

Whiplash Injuries

Diagnosis and Treatment

Second Edition

Springer

Editors
Dario C. Alpini
ENT-Otoneurology Service
IRCCS "Don Carlo Gnocchi" Foundation
Milan
Italy

Guido Brugnoni
Italian Academy of Manual Medicine
Italian Institute for Auxology
Milan
Italy

Antonio Cesarani
Department of Clinical Sciences and
Community Health
University of Milan
Milan
Italy

Audiology Unit
IRCCS "Ca' Granda" Ospedale Maggiore
Policlinico
Milan
Italy

ISBN 978-88-470-5485-1 ISBN 978-88-470-5486-8 (eBook)
DOI 10.1007/978-88-470-5486-8
Springer Milan Heidelberg New York Dordrecht London

Library of Congress Control Number: 2014932997

Foreword

I have dedicated my early researches to the study of learning disturbances and behavioral disorders in childhood, especially with regard to autism. The focus of my study was on the correlation between posture, that is the language of the body, and mental health. During my fellowship at Stanford University in the 1970s, I developed the idea that measurement of postural control might be the best way to investigate cognitive functions. The basic posture of the human being is upright bipedalism; maintaining dynamic equilibrium of the body, which is like an inverted pendulum on a relatively restricted base area, is an evolutionary phenomenon directly linked to high-level cognitive development. In adulthood, maintaining stance is perceived to be easy, at least under normal conditions on steady surfaces, but it actually requires a very high level of sensorimotor control to allow a low-effort antigravitary alignment of the different parts of the body.

One of the major positive aspects of an upright position is the possibility of having an improved perception of the environment. In the horizontal plane, the atlanto-occipital joint enables a wide range of head movement, thus allowing the eyes to explore further. The relatively limited support base allows the body to turn rapidly in order to control the environment behind us. In the vertical plane, the lumbosacral spine allows the extension of the head and the trunk in order to explore the environment over our head. Thus, the human posture is an evolution from the quadrupedal motion, offering tremendous possibilities exploring the environment along the three space axes.

The posturographic equipment I developed in the 1970s I called tetra-ataxiometry because it records simultaneously the pressures exerted on the ground by the four supports: right and left toes and right and left heels. It also allows investigating how the free movement of the neck during rotation, extension, and flexion influences the control of the upright position. In other words, the equipment lets us to investigate how abilities that were specific to human beings correlated with motor programs specific to quadrupeds. In the phylogenesis, bipedal posture was relatively recently implemented on quadrupedal motor control, and this can be retraced by observing how a child fights against the force of gravity in the early years of life.

This concept explains the pathogenesis of whiplash associated disorders (WADs) that frequently cause impairments not proportionate to the intensity of the impact. The relatively low energy transferred to the body during an accident specifically affects the two most phylogenetically recent parts of the body – the atlanto-occipital

joint and the lumbosacral spine – thus limiting the ability of the body to rotate the head and to extend the head and the trunk. In other words, the whiplash effect makes the human body akin to that of primates. This probably reveals the biological and social importance of WADs.

This second edition is primarily based on the evolutionary concepts of the vestibular system, originating in quadrupeds and advanced in bipeds. The vestibular system supports, in an entirely unique way, perceptual orientation, dynamic body stabilization, retinal image stabilization, and autonomic nervous functions.

This book discusses the different aspects of whiplash, a particular kind of trauma that has a significant impact on the body as a whole. In WADs, pain is an important aspect, but it is only one of the effects, with functional ones being more prevalent. This book is therefore particularly interesting and complete as it focuses both on the painful and the functional aspects of WADs.

Prof. Reuven Kohen-Ratz,
Professor Emeritus of Special Education – Hebrew
University of Jerusalem
Jerusalem, Israel

Preface to Second Edition

The incidence of whiplash injuries has been estimated to be 1/1,000 people per year in the Western world. Chronic spine pain and associated symptoms following whiplash injury cause severe individual burdens of disease and high costs of healthcare systems at least in the Western countries (with the exception of Lithuania and Greece due to specific approach of health system and insurance companies to whiplash associated disorders in these countries).

The first edition of this book was prepared one year before that the Quebec Task Force (QTF) presented the first classification of whiplash associated disorders (WADs). QTF guidelines are still widely accepted both for diagnosis and treatment.

Furthermore, evidence based medicine (EBM) became more and more important in these last 10 years in leading clinicians toward the most effective approach to diseases and disorders, including WADs. However, whiplash mechanism is so complicated to lead to complex disturbances that very frequently they are very difficult to simplify into EBM tracks.

The term "whiplash" dates back to 1928, when the American physician H.E. Crowe used it in a symposium of traffic accidents held in San Francisco. Crowe did not refer to the injury as such, but as the motion of the head and neck that underwent in conjunction with a collision.

The interaction between the accident and the human body is not simply a mechanic interaction. In contrast to mechanical systems in which component parts interact linearly to produce a predictable output, the components of complex systems interact nonlinearly over multiple scales and produce unexpected results. The output of mechanical systems can be controlled by manipulating each one of its parts, while the output of complex systems is dynamic, behaving differently according to initial conditions and feedback. This is the case of whiplash and WADs.

In order to minimize the risk of long-term problems among people with acute whiplash injury, concurrently acute stress disorder (ASD) and/or posttraumatic stress disorder (PTSD) should be diagnosed and adequately treated.

For the above complex reasons, we thought to be necessary an updating of the first edition that takes into account the different challenges to whiplash, the most updated EBM diagnostic and therapeutic indications, and the evolution of QTF guidelines.

More specifically, in this edition, the Editors tried to include all the pathological effects of whiplash and not just the traditional neck and spine effects. The leading idea of this book is the conception of whiplash as a trauma of the body as a whole, obviously with different impact on the different parts of the body. The second leading idea is that the effects of whiplash are substantially unpredictable per se and thus a brain damage has to be expected just like a temporo-mandibular joint disorder or low back pain.

Some problems have been encountered in updating the chapter dedicated to medico-legal aspects. Various studies on how possible insurance compensation may influence the course of whiplash injuries have produced diverging results. Generally, however, there is no evidence indicating any significant difference in the results between those who have applied for such compensation and those who have not.

Furthermore, a common legal procedure about compensation of whiplash injuries in the different countries of the Western world is lacking. For these reasons we focused on management and treatment of patients disorders, but we avoided to include a chapter about medico-legal aspects of WADs.

Finally, we can state that the spirit of this second edition is paying our attention to collect in this book *What's new – What else besides pain – What to do* for patients affected with WADs.

Dario C. Alpini
Guido Brugnoni
Antonio Cesarani

Milan
Milan, Italy
November 2013

Preface to First Edition

This book is based on the proceedings and discussions of a closed workshop held in Santa Margherita Ligure in January 1995.

It was an original scientific experience: no public was admitted. For 3 days the main contributors of this book remained closed in a wonderful hotel.

At the same hotel in the 1930s, Guglielmo Marconi performed his first experiments with radio waves. The hotel was therefore an ideal place, although the problems we discussed were not so "revolutionary" like Marconi's experiments.

On these days round tables, we performed highly restricted meetings on specific topics and presentation of selected papers, only for very few persons, were performed.

Discussions were on definition, ethiopathogenesis, physiopathology, clinical and instrumental evaluations, and medicolegal and therapeutic aspects of whiplash injuries.

All the attendants tried to report and discuss personal experiences and ideas in order to compare them.

All the discussions were especially aimed to prepare the chapters you will read in this book.

We returned to our homes very tired, but very rich in our minds. We really hope that after reading this book you will be as tired as rich.

We are very grateful to Pharmacia, who supported the closed workshop and the preparation of this publication.

Antonio Cesarani
Milan, Italy Dario C. Alpini

Contents

Whiplash: An Interdisciplinary Challenge

1

A. Cesarani, C.F. Claussen, and D.C. Alpini

Definitions of whiplash syndrome are controversial. Generally speaking, the syndrome comprehends symptoms following a traffic accident, usually a rear-end collision. These symptoms are varied and variably combined:

- Orthopedic, such as neck pain and functional limitation of cervical movements
- Neurological, such as paresthesias
- Audiological, such as tinnitus and hypoacousia
- Otorhinolaryngological, such as dysphagia and dysphonias
- Equilibriometric, such as vertigo and dizziness
- Odontoiatric, such as disturbances of occlusions and temporomandibular joint pain
- Neuropsychological, such as anxiety and attentional disturbances

The term whiplash was used for the first time in 1928 and included several mechanisms. For example, the kinematics of the head-neck movements are different in rear-end collisions than in side collisions, and it is different for the driver rather than the passenger. It is also different if the subjects wore safety belts or not.

Thus, the first challenge is definition. In this book, whiplash injury can be defined as a noncontact quick (« 50 ms) acceleration-deceleration head-neck trauma [1–3].

A. Cesarani
Department of Clinical Sciences and Community Health,
University of Milan, Milan, Italy

Audiology Unit, IRCCS "Ca' Granda" Ospedale Maggiore Policlinico, Milan, Italy
e-mail: antonio.cesarani@unimi.it

C.F. Claussen
Neurootologic and Equilibrium Society (NES), Bad Kissingen, Germany
e-mail: claussensology@gmx.de

D.C. Alpini (✉)
ENT-Otoneurology Service, IRCCS "Don Carlo Gnocchi" Foundation, Milan, Italy
e-mail: dalpini@dongnocchi.it

D.C. Alpini et al. (eds.), *Whiplash Injuries*,
DOI 10.1007/978-88-470-5486-8_1, © Springer-Verlag Italia 2014

Table 1.1 Abbreviated injury score (AIS)

AIS-Code	Injury
0	No injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical
6	Maximum
9	Not further specified (NFS)

The different combinations of symptoms lead to different syndromes so described in the literature: cervical syndrome, traumatic cervical syndrome, cervico-cephalic syndrome, and cervicobrachial syndrome.

Generally, all syndromes are characterized by a plethora of functional symptoms and lack of sufficient objective morphological findings. Either for diagnosis or for therapy, different specialists and examinations are usually necessary; however, visit to different specialists usually leads to different "specialized" diagnosis and treatments.

The second challenge is gathering documentation of the functional and morphological basis for the patient's complaints. Documentation is indispensable for medicolegal expertise and/or for treatment planning.

Due to the complexity and the wide differences of the legal system regarding insurances' approach to WAD, this reedition explains how to document WAD in patients but not how to use this documentation for medicolegal expertise. In this way, medicolegals of the different countries can adapt our information in their specific field of application.

Since a patient's complaint is caused by the trauma that lasts for months and can persist for years, the third challenge is an interdisciplinary approach to the treatment in order to avoid chronic impairment and over specialized therapy.

Whiplash injuries vary from minor to severe. They can be classified according to the Abbreviated Injury Score (Table 1.1). Generally, the evolution of whiplash is divided into three phases:

1. The onset phase, involving local reactions with release of neuromediators such as serotonin, histamine, bradykinin, and classical inflammation [4]
2. The recovery phase, locally characterized by synthesis of new collagen fibers
3. The remodelling phase, in which the neck and the body modify their positions and movement strategies in order to restore normal daily life activities

During whiplash, the kinematics of the cervical spine is completely disrupted (Fig. 1.1). During the impact, the vertebrae do not reciprocally move harmonically such as during physiological antero- and retroflexion of the neck. Whiplash is characterized by transient, but not always temporary, reciprocal inversions of the different cervical segments [5–7]. For example, in the last phase of anteroflexion, inversions in segments CI–C2 and CO–Cl have been observed, while in the second phase of retroflexion, an inversion of the segment CO–Cl happens [8, 9]. The reciprocal inversions lead to ligament and soft tissue lesions, with a segmental

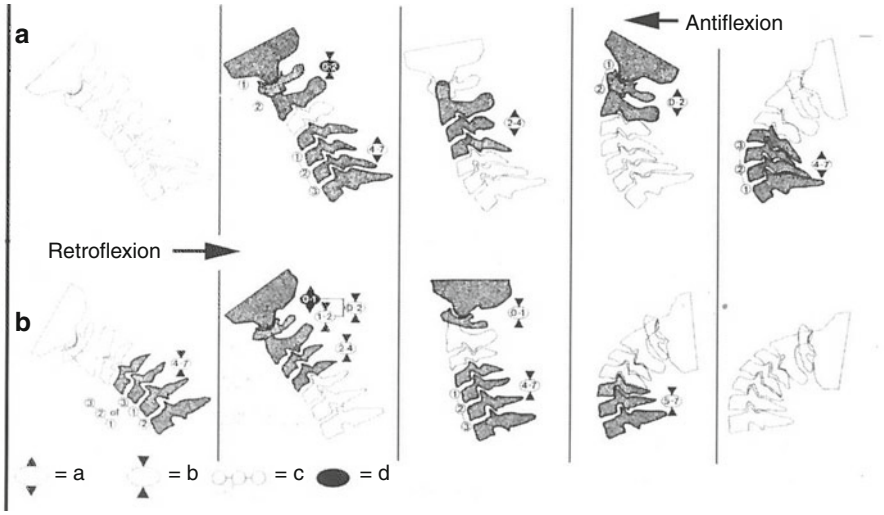

Fig. 1.1 Modification of reciprocal positions of cervical spine segments during antero-(**a**) and retroflexion (**b**) in whiplash mechanisms. In the initial phase of anteroflexion Cl–C2 and CO–C2 invert their position (**a**). In the second phase of retroflexion, inversion of CO–Cl is observed. a: flexion; b: extension; c: sequence; d: inversion symptoms

dysregulation causing a specific activation of nociceptive inputs and consequent somatomotor and sympathetic-motor dysfunctions [1]. The nociceptive inputs, via interneurons and alpha-motoneurons, provoke a dysregulation of motoneurons for the flexors and extensors leading to an asymmetrical hypertonus. The latter [10, 11] is generally observed in the trapezius and sternocleidomastoid with consequent compression of the accessorius nerve, and in scaleni with compression of the brachial plexus, responsible for paresthesias. The dysregulation of the orthosympathetic cervical system causes activation of vasoconstrictive subsystems, not always localized to the involved segments. Vasoconstriction induces dystrophic impairment contributing to the cervicofacial and cervicobrachial symptoms.

The ganglion cervicalis superius is localized at the CI–C4 segment and innervates the neck and upper respiratory and digestive tracts [12].

The cervicobrachial sympathetic complex from C5 to Th1, along with the postganglionate synapses in the cervical medius ganglion (C5–C6) and inferius (C7–C8), innervates the medium parts of the respiratory and digestive tracts.

Cervico- and craniospinal injuries during acceleration-deceleration lead to head/neck proprioception disruption causing transient, sometimes permanent, abnormal proprioceptive information regarding the reciprocal position of the neck, head, and trunk. In Fig. 1.2, the so-called spinocerebello-vestibulospinal circuitry is shown. General proprioception information (from muscles, ligaments, and joints) is integrated and elaborated in the cerebellum together with special proprioception information from maculae and cristae. From the cerebellum, efferent pathways return to spinal motoneurons through another elaboration in the vestibular nuclei (with special regard to the lateral Deiter's nucleus) [13–16].

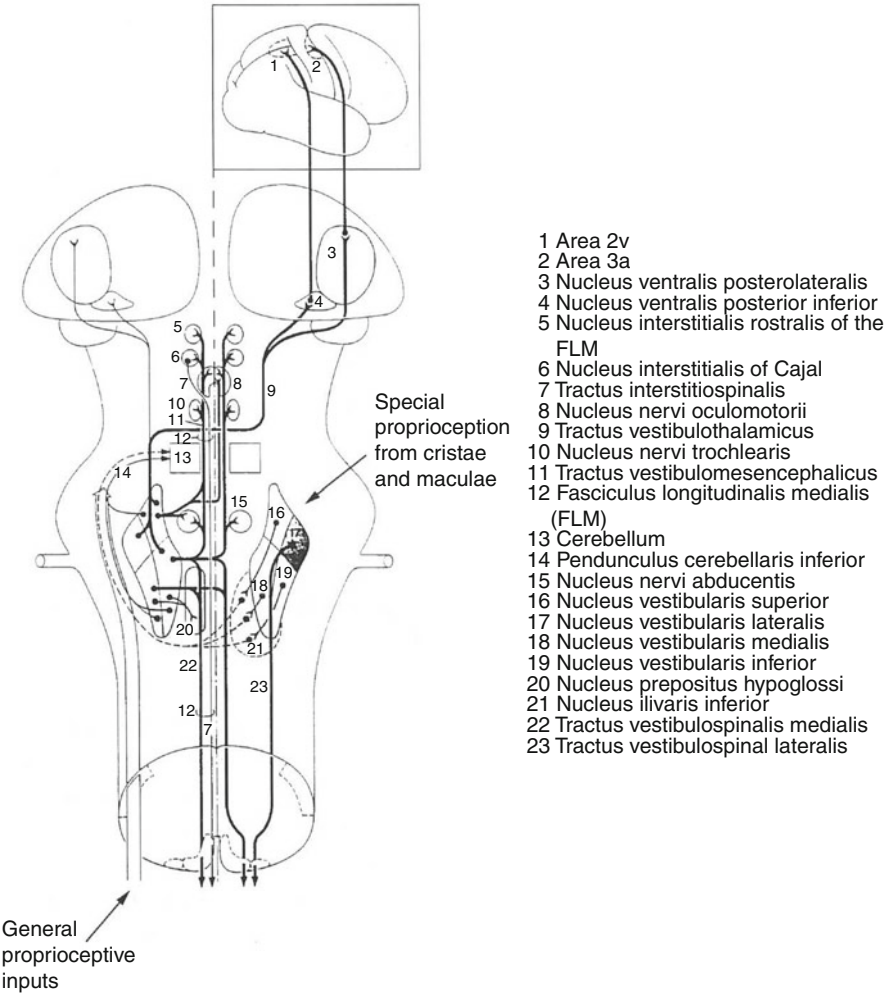

Fig. 1.2 The connections of the vestibular system: efferent connections of the vestibular nuclei and the spinocerebello-vestibulospinal circuitry

This circuitry is the anatomo-neurophysiological basis needed to understand why an apparent segmental injury often becomes an injury of the whole individual. Equilibrium disorders can be directly caused by perturbation of the integration of general and special proprioception due to abnormal peripheral inputs from the neck or for central dysfunction, particularly of the brainstem [17–20].

Whiplash is a true head trauma even if there is no contact of the head with an object. Cerebral contusions or intracranial bleeding are extremely rare, but abducens mono and bilateral palsy [21] and laryngeal palsy have been described [22]. Neurovegetative symptoms and affective-cognitive symptoms are as frequent as in post-commotional syndromes and are characterized by hyperesthetic emotional and

neuroasthenic symptoms including tinnitus, dysphasia, nausea, unsteadiness, and vertigo [23–29].

True neuropsychological disorders are frequent after whiplash. Typically, especially 6–8 months after the trauma, psychological symptoms appear: restlessness, nervousness, anxiety, emotional instability, difficulties in concentration, and depression. In 15–25 %, this neuropsychological syndrome can evolve chronically and can be misdiagnosed as an "indemnity syndrome." Experimental postmortem studies showed focal contusions in frontal and temporal cortex, corpus callosum, subcortical structures, diencephalon, and subdural and subarachnoid microbleeding as consequences of sudden angular accelerations. Rarely, alterations are macroscopic, while generally, microscopic lesions have been revealed [30–33].

One of the central networks involved in post-whiplash disorders is the ascending reticular activating system (ARAS). Experimental studies showed involvement of the ARAS especially for transitory accelerations. In Ommaya's hypothesis [34, 35], the centripetal forces during acceleration-deceleration lead to an abnormal stretching of the cerebrum with, furthermore, a sudden transient increase of the pressure of the cerebrospinal fluid [36]. In this hypothesis, whiplash provokes a commotio cerebri with potential injuries of temporal cortex, amygdala, hippocampus, medial temporal cortex, corpora mamillaria, medial thalamus, basal nuclei, prefrontal cortex, and retrosplenial cingulate cortex. All these structures may be involved in the genesis of neuropsychological symptoms [2, 37, 38].

The cerebral effects of whiplash have been studied also by electroencephalography (EEG). After a period ranging from 1 to 9 years in patients with a chronic whiplash syndrome, EEG alterations have been observed with percentages ranging from 30 to 49 %.

Glucose cerebral metabolism has been investigated by positron-emission tomography (PET) [39], and alterations have been shown in the frontal and temporal cortex and in nucleus caudatus. Lesions of frontal cortex are responsible for attentional disorders.

Pathogenesis of otoneurological disorders is still debated and includes the following explanations:

- Mechanical compression with dynamic stenosis of the vertebral artery
- Sympathetic abnormal stimulation
- Proprioceptive disorders especially from the cervical and lumbar regions [40, 41]
- Central vestibular system disorder

Probably, equilibrium disorders derive from different combinations of different mechanisms. The kinematics of the acceleration-deceleration are different, for example, for the driver, who usually opposes the trauma by means of his/her arms on the wheel, and the passenger, who usually receives, completely and passively, the impact forces. Differences can be observed if patients did or did not wear safety belts during the impact: with safety belts the movement of the head and the neck is not sagittal but torsional with the first dorsal vertebra as fulcrum. The sense of the torsional component is different: clockwise for the passenger, counterclockwise for the driver. Furthermore, the impact is rarely caused by a perfect sagittal rear-end

collision, while very often, the impact causes a rotational acceleration of the car. Torsional and angular accelerations cause microlesions in different parts of the brain, and this can lead to different combinations of signs and symptoms in the so-called whiplash syndrome [42].

Torsional and rotational acceleration-deceleration can lead to cranio-mandibular dysfunctions, too. Computer simulations [43] showed an initial derangement of condylus position with a myogenic secondary reaction during hyperextension and consequent modification of the condylus position. During hyperflexion, the retropositioning of the condylus leads to a compression of the posterior ligament rich in vessels and proprioceptors [44–47].

Following whiplash either for mechanical lesions of cervical dynamic or for central dysfunctions, some modifications of patient posture are usually observed [48]: the head modifies its position according to an antalgic flexion; the head reduces its rotational and lateroflexion movements. During subject movements, the rotation of the body is along the trunk and not the neck, the trunk is ipsilaterally rotated with respect to the side of the lesion, the pelvis is rotated according to head antalgic position, and the position of center of gravity (CoG) is modified. In the months following the trauma, erector paravertebral muscles become hypotonic, and the prevalence of flexors induces a forward displacement of the CoG facilitating forward falling. Rotation of the trunk increases unsteadiness, and forward CoG displacement provokes a relative flexion of the legs to oppose falling and derangement of normal ankle and hip strategies. In fact, Equitest shows a prevalence of ankle strategies and delayed motor control test latencies either in backward or in forward translations.

From a cybemetic point of view, equilibrium disorders are provoked by distortion and desynchronization of the proprioceptive chain:

- Modification of the proprioceptive cervical inputs to the vestibular nuclei and reticular formation
- Desynchronization between special vestibular inputs and general cervical inputs regarding head position and movement
- Modification of the cervico-spinal reflexes

If desynchronizations of proprioceptive signals “force” the limits of the patient’s equilibrium system calibration (see Chap. 29), the system is not able to adapt and it becomes dysfunctional: vertigo and dizziness appear.

Thus, the fourth challenge is correct treatment of an apparently localized injury that causes a systemic dysfunction. The treatment must be planned on the basis of an accurate functional local and systemic diagnosis. In the acute phase, treatment is aimed at reducing the impairment that is principally caused by local dysfunction. In the immediate post-acute phase, impairment is due to the dysregulation of somatic and vegetative reflexes at the involved spinal segment level. This dysregulation, if not correctly treated, can lead to the so-called chronic phase of segmental impairment. Further evolution of the syndrome includes non-segmental dysregulation: primary damage induces secondary damage. The chronic syndrome can lead to disability with motor dysfunction of the patient, usually combined with vertigo and dizziness. Rarely, motor dysfunction leads to social handicap with working and/or attending to normal daily life activity.

Rehabilitation is generally the treatment of choice either in acute or in chronic syndromes, and it is usually combined with drugs [49] and with both physical [50–54] and instrumental therapies. Rehabilitation is aimed either at the treatment of the spine or at ataxia and motor uncoordination. In the chronic phase, rehabilitation must also include instruction for ergonomic daily life activities [55, 56].

References

1. Foreman SM, Croft AC (1988) Whiplash injuries. The cervical acceleration/deceleration syndrome. Williams & Wilkins, Baltimore
2. Jenkins A et al (1986) Brain lesions detected by magnetic resonance imaging in mild and severe head injuries. Lancet 23:445–446
3. McKenzie JA, Williams JF (1975) The effect of collision severity on the motion of the head and neck during "whiplash". J Biomech 8:257–259
4. Dooms GC et al (1985) Mr imaging of intramuscular hemorrhage. J Comput Assist Tomogr 9:908913
5. White AA III, Panjabi M (1978) Clinical biomechanics of the spine. Lippincott, Philadelphia
6. Wilmink JT, Penning L (1987) Rotation of the cervical spine. A CT study in normal subjects. Spine 12:732–738
7. Wismans JSHM et al (1986) Omni-directional human head-neck response. In: 30th Stapp Car Crash conference proceedings, San Diego, SAE paper 861893. Society of Automotive Engineers, Warrendale
8. Wike BD (1967) The neurology of joints. Ann R Coll Surg Engl 41:2550
9. Wyke BD (1979) Neurology of cervical joints. Physiotherapy 65:72–76
10. Hinoki M, Kurosawa T (1964) Note on vertigo of cervical origin. Pract Otol (Kyoto) 57:10–20
11. Hinoki M (1985) Vertigo due to whiplash injury: a neurotological approach. Acta Otolaryngol Suppl 419:9–29
12. Miura Y, Tanaka M (1970) Disturbances of the venous system in the head and neck regions in rabbits with whiplash injury. Brain Nerve Inj (Tokyo) 2:217–223
13. Drukker J, van der Wal JC (1990) Centrai verbindingen van het vestibulaire apparaat. In: Fischer AJEM, Oosterveld WJ (eds) Duizeligheid en Evenwichtsstoomissen. Data Medica, Utrecht, pp 29–45
14. Drukker J, Jansen JC (1975) Compendium anatomie. De Tijdstroom, Lochem
15. Igarashi M et al (1972) Nystagmus after experimental cervical lesions. Laryngoscope 2:1609–1621
16. Ikeda K, Kobayashi T (1967) Mechanisms and origin of so-called whiplash injury. Clin Surg 22:1655–1660
17. Dichgans J et al (1974) The role of vestibular and neck afferents during eye-head coordination in the monkey. Brain Res 71:225–232
18. Doerr M et al (1984) Eye movements during active head turning with different vestibular and cervical input. Acta Otolaryngol 98:14–20
19. Doerr M et al (1991) Tonic cervical stimulation: does it influence eye position and eye movements in man? Acta Otolaryngol 111:2–9
20. Gray LP (1956) Extralabyrinthine vertigo due to cervical muscle lesions. J Laryngol Otol I70:352–361
21. Shifrin LZ (1991) Bilateral abducens nerve palsy after cervical spine extension injury. Spine 16:374–375
22. Helliwell M et al (1984) Bilateral vocal cord paralysis due to whiplash injury. Br Med J 6:1876–1877
23. Dikmen S et al (1986) Neuropsychological and psychological consequences of minor head injury. J Neurol Neurosurg Psychiatry 49:1227–1232

24. Merskey K (1984) Psychiatry and the cervical sprain syndrome. Can Med Assoc J 1: 1119–1121
25. Miller Fisher C (1982) Whiplash amnesia. Neurology 32:667–668
26. Olsnes BT (1989) Neurobehavioral findings in whiplash patients with long-lasting symptoms. Acta Neurol Scand 80:584–588
27. Rowe MC, Carlson C (1980) Brainstem auditory evoked potentials in post-concussion dizziness. Arch Neurol I37:670–683
28. Rubin W (1973) Whiplash with vestibular involvement. Arch Otolaryngol 97:85–87
29. Winston KR (1987) Whiplash and its relationship to migraine. Headache 27:452–457
30. Sa H et al (1988) Whiplash syndrome: fact or fiction? Orthop Clin North Am 19:791–795
31. Hodgson SP, Grundy M (1989) Whiplash injuries: their long-term prognosis and its relationship to compensation. Neuro Orthop 7:88–91
32. Miller H (1961) Accident neurosis. Br Med J 1:919–925
33. Zomeren AH, Brouwer WH (1990) Attentional deficits after closed head injury. In: Deelman BG et al (eds) Traumatic brain injury: clinical, social and rehabilitational aspects. Swets & Zeintlinger, Amsterdam/Lisse, pp 33–48
34. Ommaya AK et al (1968) Whiplash and brain damage. JAMA 204:75–79
35. Ommaya AK (1988) Mechanisms and preventive management of head injuries: a paradigm for injury control. In: 32nd annual proceedings of Association of the Advancement of Automobile Medicine, Seattle, WA, Sept 12–14 1988
36. Swenson MY et al (1990) Transient pressure changes in the spinal canal under whiplash motion. Report R-008. Department of Injury Prevention, Chalmers University of Technology, Gothenburg
37. Hofstad H, Gjerde IO (1985) Transient global amnesia after whiplash trauma. J Neurol Neurosurg Psychiatry 48:956–957
38. Kischka U et al (1991) Cerebral symptoms following whiplash injury. Eur Neurol 31:136–140
39. Humayun MS et al (1989) Local cerebral glucose abnormalities in mild closed head injured patients with cognitive impairments. Nucl Med Commun 10:335–344
40. Thodem U, Mergner T (1984) Effects of proprioceptive inputs on vestibulo-ocular an vestibulospinal mechanisms. Prog Brain Res 76:109–119
41. Ushio N et al (1973) Studies on ataxia of lumbar origin in cases of vertigo due to whiplash injury. Agressologie 14:73–82
42. Campana BC (1988) Cervical hyperextension injuries and low back pain. In: Rosen P (ed) Emergency medicine, vol I. CV Mosby, Philadelphia, pp 799–817
43. Kronn E (1990) A study on the incidence of temporomandibular dysfunction due to internal derangement in patients who have suffered from a whiplash injury. Dublin School of Physiotherapy, Dublin
44. Eckerdahl O (1991) The petrotympanic fissure: a link connecting the tympanic cavity and the temporomandibular joint. Cranio 9:15–21
45. Lader E (1983) Cervical trauma as a factor in the development of TMJ dysfunction and facial pain. Cranio 1:86–90
46. McGlone R et al (1988) Trigeminal pain due to whiplash injury. Injury 19:366
47. Roydhouse RH (1973) Whiplash and temporomandibular dysfunction. Lancet 1394–1395
48. McElhaney JH et al (1976) Handbook of human tolerance. Report Japan Automobile Research Institute Inc, Yataba-Chuo, Tsukuba
49. Vink R et al (1988) Treatment with the thyrotropin-releasing hormone analog CG 3703 restores magnesium homeostasis following traumatic brain injury in rats. Brain Res 460:184–188
50. McKinney LA (1989) Early mobilisation and outcome in acute sprains of the neck. Br Med J 299:1006–1008
51. Mealy K et al (1986) Early mobilisation of acute whiplash injuries. Br Med J 292:656–657
52. Odent M (1975) La réflexotherapie lombaire. Nouv Presse Med 4:26

53. Rydevik B, Szpalaski M, Acbi M, Gunzburg R (2008) Whiplash injuries and associated disorders: new insights into an old problem. Eur Spine J 17:359–416
54. Seletz E (1958) Whiplash injuries: neurophysiological basis for pain and methods used for rehabilitation. JAMA 168:1750–1755
55. Fitz-Ritson D (1990) The chiropractic management and rehabilitation of cervical trauma. J Manipulative Physiol Ther 13:17–25
56. Foley-Nolan D et al (1990) Post whiplash dystonia well controlled by TENS: case report. J Trauma 30:909–910

Part I

General Aspects

Epidemiology of Whiplash-Associated Disorders

2

F. Ioppolo and R.S. Rizzo

Until recently, there was no consensus on the definition of whiplash. According to the Quebec Task Force (QTF) on whiplash-associated disorders (WAD), "whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear-end or side-impact motor vehicle collisions, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries (whiplash-injury), which in turn may lead to a variety of clinical manifestations called Whiplash-Associated Disorders."[1] Patients with whiplash can be classified by the severity of signs and symptoms: Grade 0 means no complaints or physical signs; Grade 1 indicates neck complaints (such as pain, tenderness, and stiffness) but no physical signs; Grade 2 indicates neck complaints and musculoskeletal signs (such as a decreased range of motion or muscle weakness); and Grade 3 and Grade 4 indicate neck complaints and, respectively, neurological signs (such as sensory deficit) or fracture or dislocation.

The incidence of whiplash injury varies between different parts of the world, with rates as high as 70 per 100,000 inhabitants in Quebec [1], 106 per 100,000 in Australia [2], and 188–325 per 100,000 inhabitants in the Netherlands [3]. Versteegen also reported a sharp increase in whiplash injuries from 1989 to 1995 in the Netherlands, in conjunction with a more or less stable pattern of seat belt use [4].

F. Ioppolo (✉)
Department of Physical Medicine and Rehabilitation,
"Sapienza" University,
Piazzale Aldo Moro 5, 00185 Rome, Italy
e-mail: francescoioppolo@yahoo.it

R.S. Rizzo
Department of Physical Medicine and Rehabilitation,
"Sapienza" University,
Piazzale Aldo Moro 5, 00185 Rome, Italy

Physical Medicine and Rehabilitation Unit,
Nomentana Hospital, Largo Berloco 1, Fonte Nuova,
Rome 00010, Italy

D.C. Alpini et al. (eds.), *Whiplash Injuries*,
DOI 10.1007/978-88-470-5486-8_2, © Springer-Verlag Italia 2014

Moreover, Versteegen et al. identified patients who complained of neck pain after having been involved in a traffic accident and gone to an emergency room. Over a 20-year period, they found a tenfold increase in such visits, from an average annual incidence of 3.4 visits per 100,000 inhabitants (1970–1974) to 40.2 visits per 100,000 (1990–1994) [5].

Richter reported an increase of whiplash injuries in drivers injured in motor vehicle collisions in Hanover, Germany; these went from less than 10 % in 1985 to more than 30 % in 1997 [6].

The cumulative incidence of patients seeking healthcare for whiplash arising from a road traffic accident has increased during the last 30 years to recent estimates of >3/1,000 inhabitants in North America and Western Europe [7] and between 1.0 and 3.2/1,000 inhabitants in Sweden [8].

A 1983–1984 hospital-based study from the UK (which included persons going to the hospital for evaluation of WAD symptoms) reported an annual incidence of WAD of 27.8 (95%CI 23.6–32.6) per 100,000 inhabitants [9]. In the UK, insurance statistics indicate that 300,000 patients present per annum with whiplash-associated disorders [10].

With annual North American incidence rates estimated to be between 70 and 329 per 100,000 people [1, 11], whiplash injuries are the most common injury following a motor vehicle collision [4, 12]. Indeed, in 2000, whiplash was the most common emergency room-treated motor vehicle injury in the USA [4]. In the Canadian province of Saskatchewan, 83 % of traffic injury claims were for whiplash during 1994–1995, giving an annual incidence of 677 insurance claims per 100,000 adult population [13].

Incidence data for WAD are based mainly on study settings such as emergency room visits and insurance injury claims.

In literature, there are no published studies regarding the Italian epidemiology of WAD. Thus, data collection comes from the "Casellario Centrale Infortuni," which includes data on damage to the person with particular reference to those covered by "RC Auto" insurance [14].

Specifically, in 2009, there were 491,736 reports made to the CCI, 355,334 of which involved the cervical rachis. Of these latter, 218,754 qualify for definition as whiplash. Whiplash represented 44.5 % of all accidents.

In 2010, there was a notable decrease in the overall number of accidents and cases of whiplash. There were 130,433 cases of whiplash, which represents 42.8 % of all accidents.

2.1 Factors Associated with WAD

One potentially important factor for risk of WAD is the severity of impact, but no method exists to assess this in a standardized way. However, various preventive devices have a protective effect in passenger cars in rear-end collisions [7].

Although it seems that females are at slightly greater risk of WAD, the evidence of gender as a risk factor for seeking healthcare or making a claim for WAD is not consistent [15, 16].

Younger persons (aged 18–23) seem to be at greater risk of making insurance claims and/or being treated for WAD [13].

There is some evidence that neck pain before a collision might be a risk factor for acute neck pain after a rear-end collision [17]. Today, there are no scientifically admissible studies examining the effect of psychological, social, genetic, and cultural factors in the onset of WAD after traffic collision [7].

2.2 Prognosis

The Quebec Task Force states that whiplash injuries have favorable prognosis and their conclusion is that the 87 % and the 97 % of the patients recovered (recovery is defined by authors as cessation of time-loss compensation) from their injury at 6 and 12 months after the vehicle collision, respectively. Statement and conclusion are questionable. Whether these patients still had pain or discomfort and needed medical care, it was not reported. A review contradicted the QTF's conclusions that most whiplash injuries were short-lived [18]. These authors concluded that between 14 and 42 % of the whiplash patients developed chronic complaints (longer than 6 months) and that 10 % of those had constant severe pain. Internationally, the proportion of chronic complaints varies between 2 and 58 % [19, 20], but lies mainly between 20 and 40 %. Other studies observed that the proportion of patients who report pain and disability 6 months after the accident varies between 19 and 60 % [21, 22].

Various studies of how possible insurance compensation may influence the course of WAD have produced diverging results, but according to Jansen et al. [23], there is no evidence indicating any significant differences between those patients who have applied for such compensation and those who have not.

Regarding the economic cost due both to management of whiplash disorders and time off work, epidemiological results are limited, but it could be estimated as $3.9 billion in the USA and €10 billion in Europe in a year [24].

References

1. Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S et al (1995) Scientific monograph of the Quebec Task Force on Whiplash Associated Disorders: redefining 'whiplash' and its management. Spine 20(8 Suppl):8S–58S
2. Miles KA, Maimaris C, Finlay D, Barnes MR (1988) The incidence and prognostic significance of radiological abnormalities in soft tissue injuries to the cervical spine. Skeletal Radiol 17:493–496
3. Wismans KSHM, Huijkens CG (1994) Incidentie en prevalentie van het 'whiplash'-trauma. TNO report 94. R.B.V.041. TNO Road-Vehicle Research Institute, Delft
4. Versteegen GJ, Kingma J, Meijler WJ et al (2000) Neck sprain after motor vehicle accidents in drivers and passengers. Eur Spine J 9:547–552
5. Versteegen GJ, Kingma J, Meijler WJ et al (1998) Neck sprain in patients injured in car accidents: a retrospective study covering the period 1970–1994. Eur Spine J 7:195–200

6. Richter M, Otte E, Pohlemann T et al (2000) Whiplash-type neck distortion in restrained car drivers; frequency, causes and long-term results. Eur Spine J 9:109–117
7. Holm LW, Carroll LJ, Cassidy JD et al (2008) The burden and determinants of neck pain in Whiplash associated disorders after traffic collisions, results of the Bone and Joint Decade 2000–2010 Task Force on Neck pain and its Associated Disorders. Spine 33(4 Suppl):S52–S59
8. Jansen GB, Edlund C, Grane P et al (2008) The Swedish Society of Medicine and the Whiplash Commission Medical Task Force. Whiplash injuries: diagnosis and early management. Eur Spine J 17(Suppl 3):S359–S418
9. Otremski I, Marsh JL, Wilde BR et al (1989) Soft tissue cervical spinal injuries in motor vehicle accidents. Injury 20:349–351
10. Burton K (2003) Treatment guidelines: is there a need? In: Proceedings of Whiplash Conference 2003, Bath, England, 6–8 May. Lyons Davidson Solicitors, Bristol
11. Quinlan KP, Annest JL, Myers B, Ryan G, Hill H (2004) Neck strains and sprains among motor vehicle occupants – United States, 2000. Accid Anal Prev 36:21–27
12. Berglund A, Alfredsson L, Jensen I, Bodin L, Nygren A (2003) Occupant-and crash-Related factors associated with risk of whiplash injury. Ann Epidemiol 13:66–72
13. Cassidy JD, Carroll LJ, Côté P et al (2000) Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. N Engl J Med 342: 1179–1186
14. Il Casellario Centrale Infortuni, Rapporto Statistico (2011) http://casellario.inail.it/repository/ContentManagement/information/N115396216/RapportoStatisticoCasellario2011.pdf
15. Bjornstig U, Hildingsson C, Toolanen G (1990) Soft-tissue injury of the neck in a hospital based material. Scand J Soc Med 18:263–267
16. Bring G, Bjornstig U, Westman G (1996) Gender patterns in minor head and neck injuries: an analysis of casualty register data. Accid Anal Prev 28:359–369
17. Obelieniene D, Schrader H, Bovim G, Miseviciene I, Sand T (1999) Pain after whiplash: a prospective controlled inception cohort study. J Neurol Neurosurg Psychiatry 66(3):279–283
18. Barnsley L, Lord S, Bogduk N (1994) Whiplash injury: clinical review. Pain 58(3):283–307
19. Coté P, Cassidy JD, Carroll L, Frank JW, Bombardier C (2001) A systematic review of the prognosis of acute whiplash and a new conceptual framework to synthesize the literature. Spine 26:E445–E458
20. Scholten-Peeters GGM, Verhagen AP, Bekkering GE, van der Windt DAWM, Barnsley L, Oostendorp RAB et al (2003) Prognostic factors of whiplash-associated disorders: a systematic review of prospective cohort studies. Pain 104:303–322
21. Freeman MD, Croft AC, Rossignol AM (1998) Whiplash associated disorders: refining whiplash and its management. The Quebec Task Force. Spine 23:1043–1049
22. Stovner LJ (1996) The nosologic status of the whiplash syndrome: a critical review based on a methodological approach. Spine 21:2735–2746
23. Jansen GB, Edlund C, Grane P, Hildingsson C, Karlberg M, Link H, Måwe U, Portala K, Rydevik B, Sterner Y, Swedish Society of Medicine; Whiplash Commission Medical Task Force (2008). Whiplash injuries: diagnosis and early management. The Swedish Society of Medicine and the Whiplash Commission Medical Task Force. Eur Spine J 17 (Suppl 3):S355–S417
24. Eck JC, Hodges SD, Humphreys SC (2001) Whiplash: a review of a commonly misunderstood injury. Am J Med 110:651–656

Functional Anatomy

3

C.L. Romanò, A. Mondini, S. Brambilla, and F. Ioppolo

The anterior faces of the cervical column is composed by a median portion made of the overlapping of the vertebral bodies which include the intervertebral disks on top of the bodies. This median portion is at first narrowed on the top (15 mm) and is progressively broadened toward the bottom, reaching 25–30 mm at the last cervical metamer.

The superior vertebral plate of the vertebral bodies that go from C3 to C7 ends laterally with two osseous raised portions directed toward the top, called unciform processes or uncus. These are included with the corresponding incisions on the lateral inferior side of the above vertebral body, functionally behaving like true articulations (Von Luschka articulations) that limit the lateral slant movement of the head.

Laterally, in correspondence of the top half of the vertebral bodies, the reliefs of the transverse processes can be noticed, and they end laterally with their anterior tuberculum. Of the latter, the C6 is called Chassaignac's tuberculum and represents an important surgical traceable point, since it is more neatly developed in respect to the others (Fig. 3.1) [1, 2].

The intertransversal foramina are found in the middle of the transverse processes and give way to the vertebral arteries which penetrate in most cases in C6, with its veins, the vegetative nerve, and the rachis nerve.

C.L. Romanò
Center of Reconstructive Surgery and Osteoarticular Infection,
IRCCS "Galeazzi" Orthopedic Institute, via R. Galeazzi, 4,
20161 Milan, Italy

A. Mondini • S. Brambilla
"G. Pini" Orthopedic Institute, Via Rugabella 4,
20122 Milan, Italy

F. Ioppolo (✉)
Department of Physical Medicine and Rehabilitation,
"Sapienza" University, Piazzale Aldo Moro 5,
00185 Rome, Italy
e-mail: francescoioppolo@yahoo.it

D.C. Alpini et al. (eds.), *Whiplash Injuries*,
DOI 10.1007/978-88-470-5486-8_3, © Springer-Verlag Italia 2014