Recommendations of the Swiss Neurological Society (Schweizerische Neurologische Gesellschaft)

Diagnosis and therapy of sport-related concussion

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Introduction

The diagnosis and treatment of sport-related concussions has changed and improved considerably in recent years. Nevertheless, the affected athletes are often insufficiently diagnosed and not treated – or only recommended physical rest [1].

Sport-related concussion is caused by a direct or indirect force (blow) against the head [2]. The brain, its surrounding bony structures, the vestibular labyrinth, the eye (including eye muscles) and the cervical spinal cord (including surrounding structures) can be affected by the impacting force. Therefore, in addition to the distinction to moderate and severe trauma brain injury (TBI), the identification of the affected systems is critical for the therapeutic procedure.

This article provides an overview of diagnostic and therapeutic recommendations for neurologists. The recommendations are based on the latest scientific knowledge of head injuries [3], sport-related concussions [2, 4] and mild TBI (mTBI) [5, 6]. The overview considers the international and national guidelines of the British *National Institute for Health and Care* (NICE) [3], the *American Academy of Neurology* (AAN) [4], the *American Medical Society for Sports Medicine* (AMSSM), [7] and the *Concussion in Sport Group* (CISG) [2, 8–10] as well as various sports federations (e.g. *World Rugby* [11], *National Football League* [12], *National Hockey League* [13], *English Ice Hockey Federation* [14], *Parachute Canada* [15], *Water Polo Canada* [16]).

Definition and classification

The definition and classification of sport-related concussion are inconsistent [17]. In the International Classification of Diseases (ICD-10), sport-related concussion is defined as a form of brain injury associated with a temporary loss of normal brain function in response to head injury. According to the AMSSM, a concussion is defined as "a traumatically induced transient disturbance of brain functions that involves a complex pathophysiological process" [7]. The underlying pathophysiological process is assumed to be changes at the cellular (neuronal/glial) level as well as functional disorders of neuronal networks [7, 17, 18].

In the context of TBI, sport-related concussion is classified as mTBI [7, 19]. Sport-related concussion is differentiated from moderate or severe TBIs using conventional computed tomography (CT) or magnetic resonance imaging (MRI) of the brain, which show no structural neuronal damage after a concussion [2, 17]. Improved imaging and post-processing techniques now make it possible to detect axonal shear injuries in the first days after trauma – even in the case of mTBI. However, these procedures are not yet standardized and are still the subject of studies [5, 20].

It should be noted that in the Anglo-American region, an abnormal MRI with mild clinical abnormalities is assigned to mTBI and described as "complicated mild traumatic brain injury" [21].

Symptoms

A concussion can lead to a variety of signs and symptoms, which can appear after a few minutes or only after hours or even days. Common symptoms include concentration problems (40–90%) [22], head-

aches (70–80%) [23], vertigo, dizziness and balance problems (37–81%) [24, 25], neck pain (20–50%) [24], visual problems (20%) [26], fatigue (20–50%) [24] and autonomic dysfunctions (20–60%) [27, 28]. Sport-related concussion usually does not lead to unconsciousness [1, 7].

Most signs and symptoms of a concussion are non-specific and can also be caused by associated injuries to adjacent structures or systems (Table 1 in the appendix) [29]. A clear assignment of all relevant post-traumatic symptoms to a single structure or system is not always possible. However, especially in the case of persistent or increasing signs and symptoms or findings, a classification should be attempted within the framework of careful interdisciplinary diagnostics because the results can lead to different therapeutic measures, and some symptoms (e.g. headaches and vertigo) are considered predictors of their persistence [30–32].

Diagnostics

The *initial diagnosis* after a sports-related head injury is usually made by the team physician or the competition physician, who assumes the role of the "case manager". As part of the initial diagnosis, the physician must decide whether: (a) immediate emergency management and transport to hospital is required, (b) the athlete must be removed from the current training or competition ("when in doubt, take them out") [11], or (c) the athlete can continue to train or compete. Clinical expertise in the differential diagnoses of head injuries and their primary care is essential to fulfilling this role [33].

If any of the following signs and symptoms or findings (i.e. *red flags)* are present, the team or competition physician must make an emergency referral to the nearest hospital: any type of unconsciousness, Glasgow Coma Scale (GCS) < 15, seizure, persistent (anterograde or retrograde) amnesia, persistent disorientation/confusion, imbalance with a tendency to fall, persistent and/or high-intensity headache, vomiting, vertical double vision, spontaneous nystagmus, hearing loss, pupil difference, focal neurological deficit, or evidence of soft tissue or bony injury above the clavicle level (e.g. facial/skull fracture or cervical spine injury), anticoagulation [5, 6, 34].

Emergency management after head injuries must follow clear principles and standardized measures such as Advanced Trauma Life Support (ATLS) or as described in the European Trauma Courses [3]. Likewise, the indication for further emergency diagnostics, in particular CT imaging, should be based on standardized decision rules (illustrated by e.g. the NICE[3]/CHIP prediction rule[35]/LTHV documentation sheet for primary care[1] [35–37]).

Unconsciousness of any duration or GCS < 15 can indicate a more severe concussion or a moderate or severe TBI. This means that the affected athlete must stop training or competition and should not return to sport on the same day [2].

Because symptoms or findings can also occur with some delay, all athletes should be closely *monitored* by a reliable person (e.g. family member or acquaintance) 24 hours after a head injury with respect to clinical worsening or the recurrence of signs and symptoms or findings [2].

Within three days, the team or competition physician should *re-examine* the affected athlete in order to assess the course of signs and symptoms and take any necessary further action. If any of the signs and symptoms or findings listed in Table 1 is present, a *clinical triage examination* should be performed either by a neurological specialist experienced in the diagnosis of concussion or a physician experienced in neurological assessment. If none of these signs and symptoms or findings is present, a controlled and gradual return to sport, school and/or work should be initiated. This structured procedure applies also to athletes who have suffered an unobserved head trauma and present to their team physician, family doctor, or medical officer with latency.

With the help of a thorough *neurological clinical examination*, the neurologist can make a clear differential diagnosis with respect to the head injury and arrange for individually focused additional examinations to clarify the causes and anatomical localization of deficits. This method of clinically-directed examination is not only cost-efficient but also reduces the probability of false-positive results [33].

If combined signs and symptoms such as headache, memory problems, vertigo and impaired hearing/vision occur, *a diagnosis by a multidisciplinary medical team specialized in concussion* is recommended [33]. During this examination it is determined whether: (a) it can be "waited", (b) additional examinations are indicated, or (c) early rehabilitation measures should be initiated. In the decision-making process, it should be noted that "waiting" involves active monitoring. As already mentioned, a concussion is a dynamic process, and its signs and symptoms can newly develop or change over time.

Additional examinations by specialists serve to adequately classify headaches [38] and fatigue [25] as well as vestibular, visual, auditory, cognitive, emotional, or cervicogenic signs, symptoms and findings (Table 2 in the appendix) [1, 7, 24, 32].

1 These decision rules or documentation forms assume that the physician is able to recognise focal neurological deficits (e.g. vertigo syndromes and central oculomotor disorders).

Based on the cardinal symptom *vertigo*, which is after headache the most frequent symptom after a head trauma [24, 25], the diagnostic procedure will be described *by way of example*. Vertigo, dizziness or daze feeling after head trauma can be of both vestibular and other origin. This is because the control of body and gaze stability is based on a multi-sensory integration of vestibular, visual, proprioceptive and other sensory signals. Identifying the exact pathomechanism is complex but nevertheless essential for correct therapy planning.

In athletes with persistent vertigo or daze feeling after a head injury, the peripheral vestibular deficit often combines with a central dysfunction. As a result, central mechanisms for compensating peripheral-vestibular deficits may be impaired [39]. Common findings in athletes with persistent symptoms after a concussion include visually induced vertigo and visual dominance of balance control. Both conditions develop as a result of an unconscious vestibular avoidance strategy.

Laboratory (electrophysiological) examinations can significantly improve the differential diagnostic process with respect to the selection of therapeutic options. In cases of persistent vertigo or blurred/foggy vision, additional vestibular tests are essential in addition to the known basic clinical examinations (observation of spontaneous and gaze-evoked nystagmus nystagmus, horizontal head impulse test, test of vertical divergence (test of skew), Romberg test and provocation maneuvers to identify positional vertigo/nystagmus). The aim of these examinations is to identify or rule out peripheral-vestibular disorders. The laboratory vestibular examination typically consists of: video head impulse test along all semicircular canals (V-HIT), dynamic visual acuity (DVA) or functional head impulse test (f-HIT), cervical and ocular vestibular-evoked myogenic potentials (cVEMP, oVEMP), subjective visual vertical (SVV), fundus photography, caloric tests, ocular motor tests (saccades, smooth pursuit, optokinetic nystagmus and recording of spontaneous, gaze-evoked, positional, head shaking and vibration nystagmus).

Differential diagnosis based on signs and symptoms

Some post-traumatic signs and symptoms are specific to an impairment of the central nervous system (e.g. memory problems, confusion, or vertical nystagmus) and may indicate a concussion or a more severe form of TBI [2, 17, 24].

The most important differential diagnoses within the different systems are described below:

Pain

The causes of headache vary – for example cerebral (e.g. migraine or tension type), cervical (e.g. biomechanical, myofascial, or neural/nerval [24, 40]), or temporo-mandibular [24] – and should be diagnosed according to international criteria [38]. A post-traumatic headache is a headache attributed directly to the trauma.

Retro-orbital pain can be caused by an ocular injury or a dysfunction of the upper cervical spine (referred pain) or the temporo-mandibular joint. A neuro-musculoskeletal examination should be carried out to determine whether there is a dysfunction of the upper cervical spine [24].

Migraine headaches are often accompanied by nausea. However, nausea can independently be caused by vestibular dysfunction [41]. Head trauma can activate a pre-existing migraine. It is essential to identify a chronic headache caused by the overuse of analgesics [38].

Vestibular symptoms

Vertigo, dizziness or balance disorders can be caused by a peripheral vestibular dysfunction (labyrinth, vestibular nerve), a central vestibular lesion in the context of the concussion [24, 41–43], or other impairments (such as autonomic dysregulation, dehydration, or cervical dysfunctions [44] etc.). If vestibular disorders prevail, additional examinations of postural control, walking and the lower extremities are indicated in order to not overlook injury involving the vestibulospinal or somatosensory system as well as concomitant injury of the legs [24].

Short-term (less than one minute) head position dependent rotational vertigo is specific to benign paroxysmal positioning vertigo (BPPV), which can be caused post-traumatically by detached otoliths in the vestibular semicircular canals (canalo- or cupulolithiasis). BPPV can be diagnosed with provocation maneuvers (Hallpike maneuver for the posterior or anterior semicircular canals, supine-roll maneuvers for the horizontal semicircular canals) under observation of the positional nystagmus, preferably using Frenzel goggles or video-oculography. The affected semicircular canals can easily be freed from the concrements by appropriate liberation maneuvers (e.g. Epley maneuver for posterior canalolithiasis, Gufoni manoeuver for horizontal canalolithiasis or cupulolithiasis, Yacovino maneuver for anterior canalolithiasis).

A “feeling of being in a lift” can be an indication of a post-traumatic dysfunction of the otolith organs [45].

Visual symptoms

If an athlete describes symptoms such as “my eyes cannot follow/are slowed down” or “blurred vision” during

rapid head movements, it should be assumed that gaze stabilization is impaired due to a deficient vestibulo-ocular reflex or that the processing of visual (optokinetic) information is dysfunctional [24]. Vestibular causes can be identified using the video head impulse test [46] and the dynamic visual acuity test [47].

Photophobia or sensitivity to light can be caused by disorders within the eye (i.e. pupil, retina) or along the neuro-visual pathways or as part of post-traumatic migraine headaches [48]. Mouches volantes-like symptoms can be caused by ocular pathologies, increased perception of physiological vitreous opacity, or increased sensitivity within the visual pathways and the visual cortex [49].

Key points on sport-related concussion

- Sport-related concussion is categorized as mild TBI. In contrast to moderate or severe TBI, structural changes are generally not detectable in conventional tomographic imaging of the brain (CT, MRI).
- The diagnosis of concussion is made clinically and should be based on the patient's medical history as well as neurological, neuropsychological, neuro-vestibular, auditory, ophthalmological and neuro-musculoskeletal examinations. The results of these examinations form the basis for further diagnostic and therapeutic procedures.
- The signs and symptoms after a concussion are heterogeneous and predominantly belong to the group of neurological diseases (including headaches, vertigo, vestibular disorders, sensitivity to light/noise, fatigue and memory problems). In most cases, a concussion does not result in unconsciousness.
- In the majority of cases, the initial assessment of a sports-related head injury is carried out by the team physician or competition physician, who assumes the role of the case manager. To perform this role, clinical expertise in differential diagnoses and their primary care is required.
- In the case of initially existing signs and symptoms such as unconsciousness, loss of consciousness, amnesia or disorientation, or in the case of persistent cerebral, cervicogenic, vestibular, cochlear, ophthalmological symptoms, an initial clinical examination should be carried out within three days by a neurological specialist experienced in TBI. During the triage examination it is determined whether: (a) the course can be monitored, (b) additional examinations are indicated, or (c) early rehabilitation measures should be initiated.
- Laboratory neuro-physiological and neuro-radiological examinations are necessary for the indication of multi-modal and coordinated therapies depending on the initial signs and symptoms, in the case of persistent symptoms, or in the presence of certain risk factors at the beginning of and during the course of treatment.
- Therapies after concussion are based on current guidelines of the professional societies. In the case of multiple symptoms, a multi-modal approach by an interdisciplinary team is required.

Hearing symptoms

Sensitivity to noise or tinnitus can be caused by a dysfunctions of the cochlea (caused by a cochlear commotion or contusion) or within neuroauditive pathways. In the case of tinnitus, the association with functional disorders of the cervical spine and the temporo-mandibular joint must be considered as a differential diagnosis [50–53].

Symptoms of the autonomic nervous system

Changes in heart rate [54] at rest or during cognitive [55] or physical [56] activity, palpitations, hyper-/hypohidrosis, or an increased feeling of heat may indicate autonomic cerebrovascular dysregulation. Other causes such as the presence of a bacterial or viral infection, electrolyte or hormonal disorders, or pre-existing mood disorders (e.g. as anxiety or depression) should be investigated as a differential diagnosis, especially if the symptoms persist.

Neurocognitive symptoms

Concentration deficits and mnestic problems can be caused by the concussion itself or can result from other disorders. Vertigo, for example, correlates with neurocognitive impairment and is considered a predictor of a longer recovery time after a concussion [57]. Sleep disorders also have a negative effect on cognition [58].

Affective/emotional symptoms

Post-traumatic affective symptoms must be distinguished from pre-existing mood disorders such as depression and anxiety [24, 59].

Motor symptoms

The initial inability to move the extremities as an expression of transient paralysis in two or all four extremities may be an indication that the spinal cord is involved (differential diagnosis concussion or spinal cord contusion). If a spinal cord injury is suspected, an MRI should be initiated [60].

Therapy

A sound diagnosis forms the basis for recommending adequate and coordinated therapeutic and rehabilitative measures as well as for planning the gradual return to sport, school, or work. It is recommended that therapy and the resumption of physical and cognitive activities (sport, school and work) be started in parallel.

The therapeutic and rehabilitative approach has changed considerably in recent years. A "rest until resolve of symptoms" approach was prescribed until

about 2016. In recent years, an initial cognitive and physical rest phase (24–48 hours) should be followed by a controlled increase in physical and cognitive activity [2, 61]. Here, symptom-based aerobic training is increasingly gaining a significant role [62].

The therapeutic approach is based on the main signs, symptoms and laboratory findings and their interpretation. Standardized methods and techniques should be used for the individual areas. These can be found in the guidelines of the respective professional societies. It is important to compare every prescribed medication with the anti-doping regulations (www.antidoping.ch).

In the case of post-traumatic *headache*, treatment is based on the guidelines for the phenotypically corresponding primary or secondary headache type [23, 32]. Headache attributed to medication overuse should be identified and treated adequately [63].

When treating *vertigo*, it should be taken into account that – especially if symptoms persist – the vestibular symptoms are often caused by a combination of peripheral and central dysfunctions [39]. In general, disturbances of the sensory signals always lead to an altered motor function, which must be considered in the therapeutic procedure. As a result, early rehabilitation after concussion regularly consists of parallel therapy programs. In the case of vertigo, dizziness and impaired balance, these include e.g. canalolith liberation maneuvers and neuro-rehabilitative measures to improve the control of the body's center of gravity, postural stability, and gaze stability during active head movements (vestibulo-ocular reflex), as well as desensitization in cases of increased visual (optokinetic) sensitivity or to improve vestibular processing in cases of visual dominance with vestibular avoidance behavior.

A *combination of active and passive measures* often makes sense. As soon as possible, components of the therapy should be implemented as a home program. Behavioral therapy measures can be helpful with respect to coping with symptoms, day structure, sleeping behavior, or for learning relaxation techniques.

Because several different, mutually influencing sensory and motor systems are usually affected by the concussion, a coordinated, *multi-modal therapy program*, put together by an experienced interdisciplinary team, is essential for a rapid return to sport, school and work. Only in this way can therapy interactions be adequately considered and existing standardized programs integrated into therapy planning.

Return to sport, school and work

After an initial rest period of 24 to 48 hours, a controlled, gradual and symptom-limited return to sport, work and school is recommended since around 2017 [2, 7, 19, 61]. A systematic procedure is presented in the Sport Concussion Assessment Tool (SCAT5©) [64] (for German see https://swissconcussion.com/downloads/).

Course and prognosis

The course of a sport-related concussion is described as generally favorable with a spontaneous reduction of symptoms within 10 to 14 days in 75% to 90% of cases [65, 66]. It should be noted, however, that this is based on studies conducted mainly with American football players and is therefore probably not applicable to all patient groups. In addition, most studies assessed symptom relief under reduced physical activity (Level 1–2 of the Return-to-Sports programme). Children, adolescents and athletes have a longer course [67].

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Appendix

Table 1: Anatomical regions potentially involved in signs and symptoms after head injury.

		Anatomical region involved				
Area	**Signs and symptoms**	Cere-bral	Cervi-cal	Vesti-bular	Coch-lear	Ophthalmo-logical
Alertness/ attention	Difficulty concentrating	✓	(✓)	(✓)	(✓)	(✓)
	Difficulty remembering	✓	–	–	–	–
	Feeling “slowed down/being slow”	✓	–	–	–	–
	Delayed, slow, or inadequate responses	✓	–	–	–	–
Awareness	Confusion	✓	–	–	–	–
	Disorientation	✓	–	–	–	–
	Feeling that “something is wrong”	✓	(✓)	✓	(✓)	(✓)
	Feeling dazed	✓	(✓)	✓	–	(✓)
Sleep	Fatigue, exhaustion, low energy	✓	(✓)	(✓)	–	(✓)
	Changes in sleep or sleep behaviour	✓	(✓)	(✓)	(✓)	–
Vertigo, dizziness/ balance	Spontaneous nystagmus, positional nystagmus	✓	–	✓	–	–
	Balance problems/standing or gait instability, unsteadiness, swaying	✓	–	✓	–	–
	Vertigo, dizziness	✓	(✓)	✓	–	(✓)
	Feeling of “being in a fog”	✓	✓	✓	–	(✓)
Emotions	Anxiety, irritability, nervousness	✓	(✓)	(✓)	(✓)	(✓)
	Depressive thoughts	✓	(✓)	(✓)	(✓)	(✓)
Headache	Exercise (physical exertion)-dependent	✓	✓	–	–	–
	Position-dependent	✓	✓	–	–	–
	Motion-dependent	✓	✓	–	–	–
	Cranio-cervical	✓	✓	–	–	–
	Retro-orbital	✓	✓	–	–	✓
Vision	Blurred vision	✓	(✓)	(✓)	–	✓
	Double images	✓	–	✓	–	✓
	Unilateral visual problems	✓	–	–	–	✓
	Sensitivity to light, photophobia	✓	–	–	–	✓
Hearing	Phonophobia, sensitivity to noise, tinnitus	✓	(✓)	–	✓	–

✓ = likely, (✓) = possible, – = not possible
Note: Many symptoms are non-specific and can have other causes.

Table 2: Symptom-related diagnostics after a concussion with respect to potentially involved neuro-anatomical regions.

		Common symptoms	Focus of the medical examination	Suspicious signs	Possible neuro-anatomical disorders or dysfunctions	Selection of indicated additional examinations
Somatic domains	Pain	• Headache: ○ Position-dependent ○ Effort-dependent ○ Pulsating vs pressing ○ Frontally emphasized ○ Retro-orbitally emphasized ○ Occipitally emphasized	• Medication history • Novel vs. pre-existing • Cranio-mandibular • Neuro-orthopedic (cervical spine/thoracic spine)	• Increased pericranial sensitivity to pain • Pressure-sensitive notches (supra-, infra-orbital, mental, occipital) • Tenderness on palpation /trigger points cervical (vertebrogenic, spondylogenic, neurogenic, myofascial)	• Post-traumatic: ○ Migraine like ○ Tension type ○ Cervicogenic • Overuse of pain medication: ○ Exercise-induced ○ Pre-existing exacerbated	• Sports physical therapy: ○ Cranio-mandibular function ○ Status of cervical/thoracic spine function • Standardised questionnaires (e.g. HIT-6, MIDAS) • Imaging (e.g. X-ray, MRI, CT)
		• Neck pain: ○ One-sided without side change ○ Radiating into the back of the head ○ Radiating into arm	• Cranio-mandibular • Neuro-orthopaedic (cervical spine /thoracic spine)	• Limited cervical/thoracic spine range of motion (active and passive) • Cervical spine /thoracic spine provocation maneuvers • Tenderness on palpation/trigger points cervical (vertebrogenic, spondylogenic, neurogenic, myofascial)	• Cervico-cephalic • Cervico-thoracic	• Sports physical therapy: ○ Cranio-mandibular function ○ Cervical spine/thoracic spine function • Standardised questionnaires (e.g. NDI) • Imaging (e.g. X-ray, MRI, CT)
	Vestibular	• Vertigo/dizziness: ○ Rotational vertigo ○ Elevator vertigo ○ Postural / perceptual dizziness • Nausea • Oscillopsia • Balance problems • “Feeling dazed” • Feeling of “not being present”	• Neuro-otological • Neuro-orthopedic (cervical spine /thoracic spine)	• Nystagmus: ○ Spontaneous nystagmus ○ Positional nystagmus ○ Positioning nystagmus ○ Head-shaking nystagmus ○ Gaze evoked nystagmus • Head impulse test with catch-up saccadees • Deficient optokinetic nystagmus decay • Lack of visual VOR suppression • Hypersensitivity to optokinetic stimuli • Balance problems	• Peripheral-vestibular ○ Labyrinth ○ Labyrinth/brain stem to N. VIII • Central-vestibular ○ Pontomedular/cerebellar (vestibulo-oculomotor pathways) ○ Multi-sensory integrative (vestibular, visual, somatosensory) ○ Thalamus/cortical (vestibulo-thalamo-cortical pathways)	• Sports physical therapy: ○ Balance ○ Vestibular/ocular motoric (e.g. VOMS) ○ Musculo-skeletal • Vestibular and oculomotor (laboratory tests: video HIT, oVEMP, cVEMP, SVV, fundus photography, calorics, DVA, VOG) • Laboratory dynamic posturography • Optokinetic-postural ○ Standardized questionnaires (e.g. SVQ, VVAS) ○ Laboratory tests (e.g. SVV)
	Visual	• Double vision • Focussing problems • Photophobia • Blurred vision	• Neuro-ophthalmological	• Eye muscle paresis • Accommodation insufficiency • Convergence insufficiency • Saccades ○ Slowing ○ Dysmetria ○ Increased Latency • Saccadic smooth pursuit	• Peripheral oculomotor ○ Eye muscle ○ Brain stem up to N. III, IV, VI • Central oculomotor ○ Brain stem ○ Cerebellar ○ Supranuclear core areas to cortical	• Vestibular/ocular motor (e.g. VOM) • Neuro-ophthalmological (laboratory tests: Hess screen, visual acuity test) • Visuo-postural ○ Standardized questionnaires (e.g. SVQ, VVAS) ○ Laboratory tests (e.g. SVV)

		Common symptoms	Focus of the medical examination	Suspicious clinical findings	Possible neuro-anatomical/ functional disorders	Selection of indicated additional examinations
Somatic domains	Auditive	• Hearing loss • Phonophobia • Tinnitus	• Neuro-auditive (including auditory canal inspection)	• Hearing disorder ○ Hyperacusis ○ Hypoacusis	• Peripheral-cochlear Cochlea/brain stem to N. VIII • Central-cochlear • Brain stem/cortical	• Pure tone audiometry • Standardised questionnaires (e.g. THI)
	ANS	• Symptom provocation for ○ Physical strain ○ Change of position	• Internal	• Schellong test with orthostatic dysregulation, hypotension, vertigo/dizziness • Increased/reduced sweating	• Dysregulation of the autonomic nervous system	• Bicycle ergometer stress test • Schellong test • Heart rate variability testing
Cognitive domain		• Impairment of: ○ Concentration ○ Attention • Memory problems • "Slowed thinking" • Disorientation	• Neuropsychological	• Impairment of: ○ Orientation ○ Memory ○ Attention ○ Psychomotor processing speed	• Affected core domains: ○ Attention ○ Working memory ○ Executive functions	• Computer-assisted neurocognitive tests (e.g. CNS vital signs, ImPACT, CANTAB) • Paper-pen tests under neuropsychological instruction
Affective/emotional domain		• Lethargic, indifferent • Emotional, exhausted • Nervous, irritable, distractible • Sad, withdrawn, demotivated • Anxious, jumpy, worried	• Neuro-mental/ psychiatric	• Reduction of affect, reduction of drive • Apathy • Emotional blunting • General state of exhaustion • Anxiety symptoms (including nightmares) • Depressive mood • Jitteriness • Increased vigilance/self-observation		• Standardized questionnaires (e.g. GAD, HADS, CES-D, SF-36)
Sleep		• Problems falling asleep, sleeping though insomnia • Increased need for sleep • Decreased need for sleep • Daytime sleepiness	• ORL/internal medicine (including throat inspection)	• Drowsiness		• Standardized questionnaires (e.g. ESS, FSS)

Abbreviations: **ANS:** autonomic nervous system; **CES-D:** Center for Epidemiological Studies Depression Scale; **cVEMP**: Cervical vestibular evoked myogenic potentials; **CT**: Computed tomography**; DVA**: Dynamic visual acuity; **ESS**: Epworth Sleepiness Scale; **FSS**: Fatigue Severity Scale; **GAD**: Generalized Anxiety Disorder; **HADS**: Hospital Anxiety and Depression Scale**; HIT-6**: Head Impact Text-6; **MRI**: Magnetic resonance imaging**; NDI**: Neck Disability Index; **ORL**: Oto-rhino-laryngology; **oVEMP**: ocular vestibular evoked myogenic potentials; **SF-36**: Short Form-36 Health Survey; **SOT**: Sensory Organisation Test; **SVV**: Subjective Visual Vertical; **SVQ**: Situational Vertigo Questionnaire; **THI**: Tinnitus Handicap Inventory; **VOG**: Vestibulo-oculography; **VOM**: Vestibular/Ocular-Motor Screening; **VOR**: Vestibulo-ocular reflex; **VAS**: Visual Analogue Scale.

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