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Compensation for balance disorders: analysis of this multifactorial process

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Abstract

The European Society for Clinical Evaluation of Balance Disorders (ESCEBD), based in Nancy, France, has been meeting yearly since 2005 to discuss equilibrium-related themes that are not yet clearly defined or standardized. One of our latest discussions was with regard to outlining strategies of internal and external compensation that may be used to cope with balance disorders. A Committee was elected to discuss the mechanisms of compensation that may be involved in coping with balance system disorders. Compensation, referring to the immediate or short-term adaptive mechanisms that are used to counterbalance the effects of deficiencies that disrupt balance maintenance, can include alternative strategies, resources, or environmental supports to overcome deficits or challenges associated with a deficiency. The strategies can be internal (*i.e*. utilizing the individual's own multi-sensory neural integration, motor, and cognitive resources) or external (*i.e*. modifying the environment, or using assistive or adaptive devices) to reduce fall hazard and enhance safety. This report focuses principally on internal compensation, generated by sensorimotor processes of the central nervous system (CNS) in response to impairment of either sensory information (*e.g.* vestibular pathologies), the musculoskeletal system (*e.g.* lower limb amputation and myopathies) or the CNS itself (*e.g.* upper motor neuron syndrome). The multifactorial process of compensation may explain the limitations encountered by the CNS in compensating for complex bodily impairments and may also limit our understanding of how the CNS adapts to balance disorders. Newly developed devices, such as wearable sensory substitution devices, are on the horizon as possible tools.

Key Words: balance disorders, compensation, adaptation, sensorimotor processes, sensory information reweighting.

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At the annual meeting of the European Society for Clinical Evaluation of Balance Disorders (ESCEBD), a committee was selected to discuss the mechanisms of compensation that may be used to address balance disorders. This topic is of clinical importance since the therapeutic approach towards balance disorders is based on the knowledge of pathophysiology.

Animals share common functions such as respiration and movement. In the bipedal human, movement and postural control are gravity-referenced and require a highly developed system for orientation and postural equilibrium.[1]

Balance control and postural maintenance is crucial to prevent falls when standing, but also during dynamic movement. It is a complex motor skill involving many systems and interaction of multiple sensorimotor processes.[1] This skill requires central processing of vestibular, visual, proprioceptive (musculo-tendino-articular) and exteroceptive (plantar cutaneous) information. These sensory afferents converge on the vestibular nuclei of the brainstem, which integrate them and enable the organization of reflex responses. Compensatory movements of the eyes, head and body are then generated in order to stabilize gaze and posture (Figure 1). These responses must be adapted to environmental conditions and the task to be carried out. Posture maintenance in three-dimensional space is necessary to carry out integrated motor activity.

The weight and hierarchy of sensory inputs (*i.e.* balance strategies) will be different depending on a subject's age, learning and experience (*e.g.* sports practised), previous pathologies, and also on specific situations, which can include environment, and sharing of attentional resources in multiple tasks.[2]

In a normal environment (adequate lighting, predictable base of support), healthy subjects rely on somatosensory (70%), vision (10%) and vestibular (20%) information. Sensory information is also reweighted depending on sensory context (*e.g.* unstable surface).[3,4]

The Central Nervous System (CNS) controls balance by continuously integrating sensory information, and initiating appropriate motor commands.[5] This is carried out by various corrective motor mechanisms based on anticipatory and reactive signals, regardless of whether disturbances to equilibrium are expected.[1] If balance control is affected by an acute or chronic deficiency of a system, the body will compensate for the loss as effectively as possible. The process will differ depending on the part of the body that has been damaged.[6]

Balance maintenance is a complex function involving the

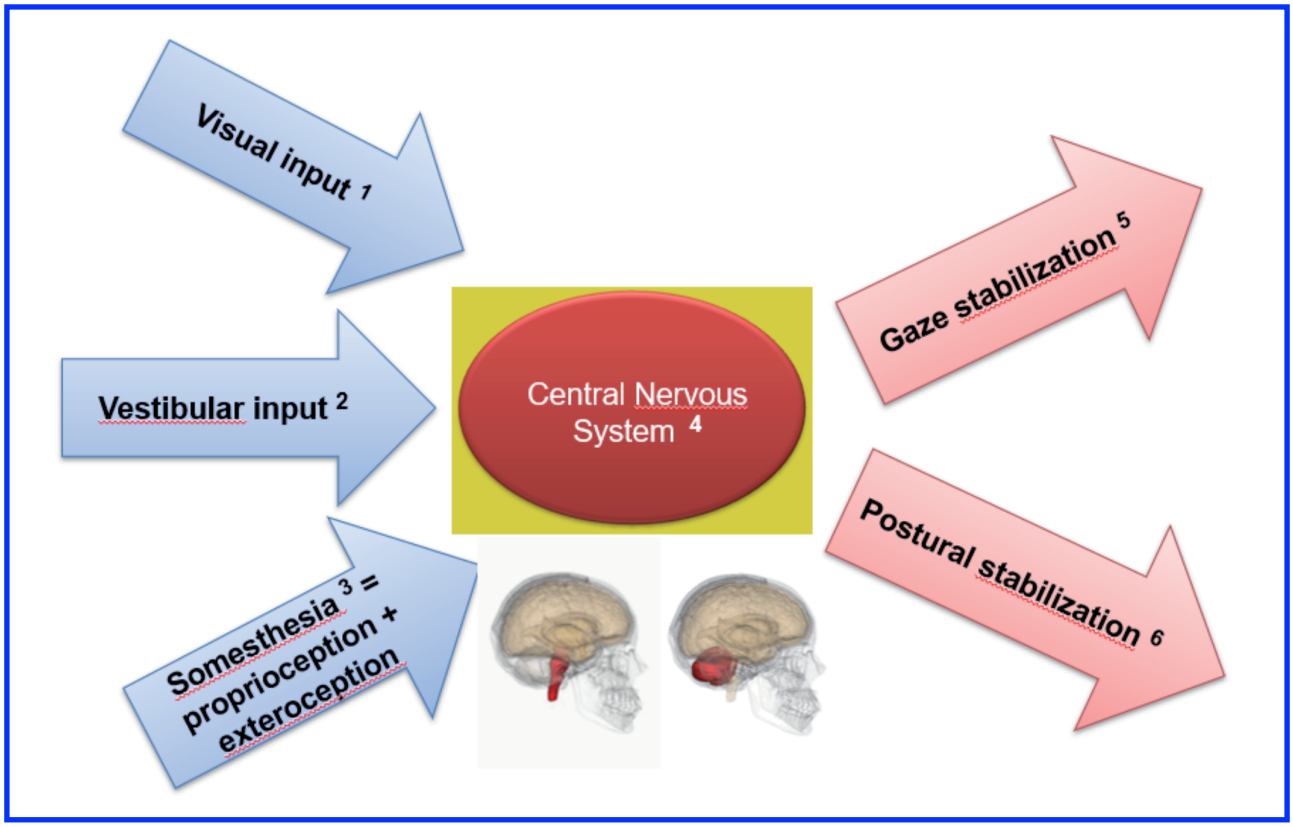

Figure 1. *Balance control, a multimodal function. The Vestibulo-Ocular Reflex (VOR) (2-4-5) stabilises a visual image on the retina during head movements, using compensatory eye movements of the same amplitude but in the opposite direction. The Optokinetic Response (OKR) (1-4-5), also referred to as the optokinetic nystagmus (OKN), stabilizes visual image during large field visual motion. Efferent pathways from the vestibular nuclei also synapse with vestibulospinal pathways and they play an essential role in posture regulation (2-3-4-6).*

interaction of multiple sensorimotor processes, and as a result the pathophysiology of balance disorders and also related compensation strategies are difficult to understand.

Compensation (from the Latin *compensatio*, "to counterbalance") refers to the immediate or short-term adaptive mechanisms (that may become long-term if necessary) that are used to counterbalance the effects of deficiencies that disrupt balance maintenance. This can include alternative strategies, resources, or environmental supports to overcome deficits or challenges associated with a deficiency. The strategies can be internal (*i.e.* utilizing the individual's own resources) or external (*i.e.* modifying the environment, or using external aids). If compensation is prolonged, neural adaptation develops as a long-term adjustment and a modification of physiological, neural, and behavioural processes. This adaptation occurs gradually over time to accommodate changes for balance control.[7]

This article will outline strategies of internal and external compensation that may be used to cope with balance disorders. Compensation can be complex, and may depend on the location of the impairment. We will focus principally on internal compensation, which is generated by sensorimotor processes of the CNS in response to impairment of either sensory information (illustrated by vestibular pathology), the musculoskeletal system (illustrated by lower limb amputation and myopathies) or the CNS itself (illustrated by Upper Motor Neuron Syndrome, UMNS).

Principal compensation strategies for balance disorders

Internal and external compensatory strategies both play significant roles in managing balance disorders. These strategies aim to ensure safety and stability.

Internal compensation

Internal compensation involves utilising a patient's sensory, motor, and cognitive resources to adapt to balance impairments. These strategies include: i) sensory integration: information from intact sensory systems (Figure 1) is processed and integrated to compensate for one or more deficits ("sensory information reweighting"); ii) motor adaptation: movement patterns and muscle activation can be adjusted to compensate for weaknesses, asymmetries, or impairments in motor function. This may involve recruiting alternative muscle groups, modifying gait patterns, or using compensatory movements to maintain stability; iii) cognitive strategies: cognitive processes such as attention, concentration, and anticipation can be engaged to enhance balance control. Cognitive strategies may include focusing on maintaining balance, anticipating environmental hazards, or conscious planning of movements.

External compensation

External compensation involves modifying the environment or using external aids to provide additional support and enhance stability, including: i) compensation devices: assistive devices such as canes, walkers, or walking sticks will provide physical support and improve balance reducing fall risk; ii) adaptive equipment: specialized equipment or adaptive technologies can be designed to assist with balance impairments. This includes balance boards, stability balls, and wearable devices which provide feedback or assistance in maintaining equilibrium. Wearable devices offer sensory substitution to cope with balance disorders by providing real-time feedback about body position, movement, and orientation. These devices use various sensors which detect changes in balance, and translate this information into tactile, auditory, or visual cues that are detected and interpreted, helping to stabilize the body (Table 1). Using these devices requires significant cognitive effort (thought) to maintain balance whereas in the healthy individual, this is an automatic task; iii) environmental modifications: making changes to the physical environment will reduce fall hazards and enhance safety. This may include installing handrails, grab bars, non-slip surfaces, and adequate lighting in areas where balance impairment is a concern.

By combining internal and external compensation strategies, individuals with a balanced compromise can learn to compensate effectively. Rehabilitation programs must be tailored to an individual's specific needs in order to optimize internal compensation mechanisms and provide guidance on the use of external aids and environmental modifications that can enhance safety and quality of life. Table 1 gives examples of internal and external compensation mechanisms according to the type of impairment.

Compensation for sensory disorders: the case of vestibular impairment

The vestibular system is a highly integrated sensory network that allows us to maintain balance, orient in space, stabilize vision, and perceive motion. Vestibular pathology can result in balance disorders, which the CNS deals with by initiating motor and cognitive adaptation, and also by using sensory information reweighting.

Both acute and chronic vestibular deficits can affect the vestibular system, and can compromise the two reflex pathways it drives (vestibulo-ocular reflex and vestibulo-spinal reflex). Compensation is essential so that normal everyday activities can be safely resumed. It involves adaptive mechanisms that allow individuals to maintain balance despite their deficit. The brain, visual system, and musculoskeletal system all play roles in compensating for the lack of accurate vestibular information, but the nature of these mechanisms varies depending on the vestibular deficit (*e.g.* unilateral vs bilateral).

Acute deficit

An acute deficit, such as an acute vestibular loss, often produces intense symptoms: vertigo (an intense spinning sensation, worsened by head movement), nystagmus, postural imbalance with falls toward the affected ear, and autonomic symptoms such as nausea and vomiting.[8] In the acute phase, the body experiences a sudden loss of vestibular input and a patient may be essentially immobile. As they gradually mobilize, patients tend to adopt a wide-based stance to increase stability. Their centre of gravity will deviate toward

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Table 1. *Physiological strategies (internal compensation) and medical/environmental interventions (external compensation) to compensate for balance impairments.*

Affected structure	Body strategies used for compensation	Medical and environmental interventions
Unilateral vestibular impairment	Sensory adaptation: • Contralateral inner ear • Visual • Proprioceptive Motor adaptation for balance	Sensorimotor rehabilitation
Bilateral vestibular impairment	Sensory adaptation: • Visual • Proprioceptive Motor adaptation for balance	Sensorimotor rehabilitation Vestibular implants Vibrotactile stimulation belt
Moderate to severe hearing loss	Visual adaptation: *e.g.* lip reading	Behind-the-ear or in-the-ear-hearing aids Unilateral: Bone Anchored Hearing Aid (BAHA) / Contralateral Routing Of Signals (CROS)
Severe to profound hearing loss	Visual adaptation: *e.g.* lip reading, sign language	Bilateral: cochlear implants
Decrease in visual acuity	Sensory adaptation: • Auditory, tactile, olfactory	Glasses, contact lenses, cataract surgery
Blindness	Sensory adaptation: • Auditory, tactile, olfactory Motor adaptation for balance	Sensorimotor rehabilitation Cane for the blind, podotactile strips and nails, stair nosing, audible pedaestrian signals (APS) Braille system, seeing eye dog Visual to electrical signal transduction on the tongue, visual to voice transduction by AI devices in eyeglass
Proprioception *e.g.* Ehlers-Danlos syndrome	Sensory adaptation: • Vision, proprioception Motor adaptation for balance	Sensorimotor rehabilitation Compression garment, taping band
Proprioception + pain *e.g.* lower limb osteoarthritis	Sensory adaptation: vision Motor adaptation for balance	Sensory rehabilitation (physiotherapy) Surgery (hip, knee prosthesis) Cane, shoehorn
Multisensory deficit: • Parkinson disease (proprioceptive + vestibular deficits)	Sensory adaptation (vision) Motor adaptation for balance	Sensorimotor rehabilitation (dry and aquatic physiotherapy), dopamine Subthalamic nucleus stimulation,
• Diabetic neuropathy (proprioceptive, visual, inner ear deficit)	Sensory adaptation (linked to the types of deficit) Motor adaptation for balance	Sensorimotor rehabilitation
Neurological impairment: *e.g.* upper motor neuron syndrome	Motor adaptation for balance: • automatic (↑base of support, muscle co-activation, toe-walking, etc.) • cognitive (↑attention, etc.)	Walking aids Sensorimotor rehabilitation

the affected ear. Walking is slower, with short and wide steps, and a longer stance phase.[9] These gait patterns are developed if balance is poor[10] or if a patient has perceptive dizziness[11] and the aim of this is to counterbalance the associated gait instability.[12] Patients will often gaze at stationary objects to anchor their sense of position and orientation, or hold on to nearby objects (*e.g.*, walls, furniture) for extra stability, and often keep their head as still as possible.[8] Following the initial phase of intense symptoms, a patient will be able to re-activate and gradually regain balance.

The typical timeline for recovery from an acute vestibular deficit, for example, a vestibular neuritis, often follows a predictable pattern, generally spanning several days to weeks.[13] Patients often prefer to stay in bed for 1 to 3 days. After 5 to 7 days, nystagmus is largely suppressed by fixation in the primary position but is still present for 2 to 3 weeks during lateral gaze away from the lesion.[8] Gradual resolution of vertigo occurs, allowing for slow, controlled head movements without severe symptoms, and regaining the ability to walk with visual guidance, but with persistent imbalance. Gait patterns that have been adopted such as reduced walking speed and a wider base often improve over a month or two.[9] Most individuals achieve full recovery within a few months, but a small number may develop chronic symptoms such as subtle imbalance when performing complex movements or in low-light environments, or dizziness provoked by rapid jerky head movements, especially under physical or mental stress.[13]

Chronic deficit

Chronic deficits that can arise from this ineffective recovery process can also result from conditions such as bilateral vestibulopathy.[14] They generally result in milder but persistent symptoms as the brain continues the compensation process. These symptoms can include unsteadiness, visual disturbances, and cognitive fatigue. At this stage, the body has had time to develop compensation strategies for the vestibular loss. CNS adaptation, proprioceptive feedback, and visual cues play significant roles in maintaining balance over time. The brain increasingly relies on visual and proprioceptive information for balance maintenance. However, in low-visibility situations, standing balance may be impaired, especially in a patient who relies more on visual input.[15,16] Patients may weight shift to try and stabilize and reduce postural sway. even when standing still, and they also learn to use muscle feedback and controlled movements to minimize sway. They may also reduce unnecessary head movement, particularly quick movements, to avoid triggering imbalance or disorientation. During walking, they may take shorter strides to compensate for gait instability.[17] They now need to think about the previously automatic task of balance maintenance, especially when changing direction[17] or navigating uneven surfaces. They will rely on visual information, such as watching the ground carefully to avoid missteps.[18] When vestibular loss is associated with a somatosensory deficit, *i.e.* in Parkinson's disease, visual dependence may further develop.[19]

Vestibular rehabilitation exercises can help improve compensation. Cawthorne and Cooksey observed that soldiers with post concussion injuries recovered more quickly if they were mobilized in the early stage after injury, rather than being bedridden.[20] Their exercise protocol focused on strengthening visual and proprioceptive reliance, recalibrating gaze stabilization, and gradually exposing patients to movements that built tolerance for head and body movement. Over time, these compensation processes and exercises improve balance and reduce the risk of falls, allowing individuals to regain confidence in standing and walking.

Compensatory processes during vestibular deficit may be difficult to understand

Compensation is carried out by using the remaining vestibular function, and also other sensory information (even if those signals are not optimal). As an example, gaze stabilization during head movement is provided by the vestibulo-ocular reflex in healthy subjects but if there is a deficit, substitute movements of the head and body are used to stabilize gaze.

It must be emphasized that patients (and all individuals) do not start off with "everything perfect," and will have differing levels of redundancy, which may compromise any compensation processes that may become necessary. The aging process also results in a loss of redundancy. Patients who were previously categorized as clinically "normal" because they were functioning within normally accepted population parameters may be unaware of such a loss of redundancy in a sensory pathway until a critical threshold is reached. They accept the definition of "normal life" as "how I have always been". In a patient where an unrecognized sensory compromise already exists (longstanding or even congenital), the process of compensation may be different from that of a patient who was initially intact. This may be a major reason why compensation and relative recovery from similar pathology can vary so widely between patients, and is so poorly understood in the clinical setting.

The mechanism itself can also complicate the process of compensation. For example, a patient with a vestibular deficit may utilize visual information to compensate for the loss, but this increased reliance on visual stimuli can itself elicit symptoms, especially in low-visibility situations.[21] Sometimes, utilizing the visual system to compensate can create autonomic symptoms,[22] not because the visual signal is erroneous, but because the vestibular system can no longer suppress an orientationally inaccurate signal. In short, although visual input is often helpful from a balance maintenance point of view, the new reliance on it may produce symptoms, as any motion sick individual will describe.

The process of "sequential" compensation (*i.e.* recovery from two events separated in time) is also more of a challenge, and it is not well understood why the "decompensation" and "recompensation" processes required in such a patient are much more challenging (and often less effective).

The mechanism of compensation is one of adaptive learning. An example of this process is when there is a congenital void of an important part of the balance system followed by later lesions. In such a case there is a further challenge to the compensation process, involving having to "change" a compensation strategy. For example, Usher's syndrome

type 1 is characterized by sensorineural hearing loss followed by subsequent progressive blindness, but also by vestibular dysfunction. As a young child, a patient with this genetic disorder might have only subtle balance deficits due to effective visual compensation, but the subsequent loss of vision (often in teenage years) will require a new compensation strategy (*i.e.* "recompensation"), which may be much less effective, and appear to the clinician as a "new development" of vestibular pathology. [23] (Figure 2).

Compensation for vestibular loss requires the effective use of remaining motor and sensory systems to restore physical function as effectively as possible. The more severe the initial damage, the greater the challenge will be to recover to a level where no physical deficit is detected. However, there are caveats to this statement.

The process of vestibular compensation is also difficult to define. If a patient has no complaints, but has an objective abnormality on testing, we may regard this as being "fully

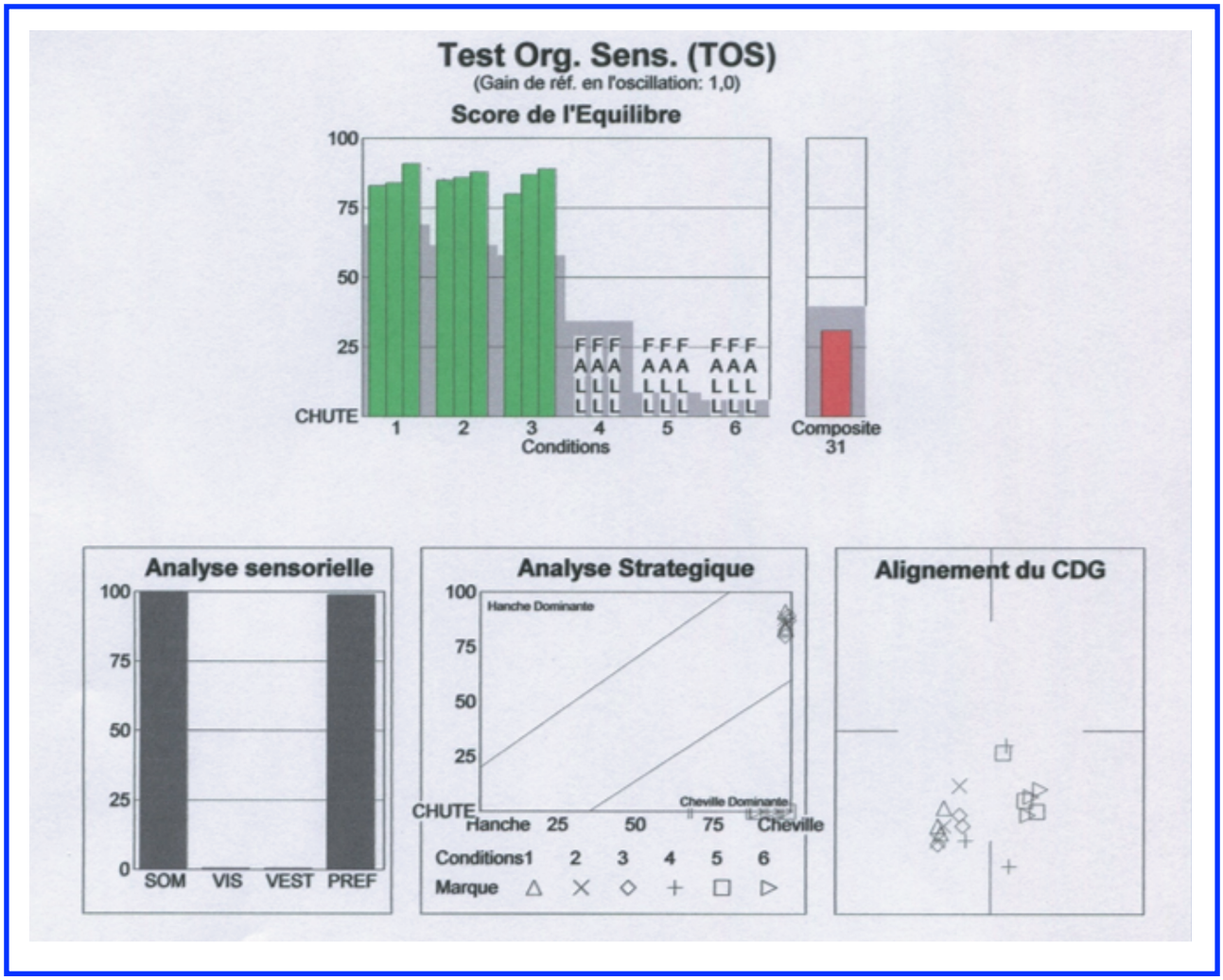

Figure 2. *Postural control in Usher's syndrome type 1.*
The six conditions of the Sensory Organisation Test (EquiTest, Neurocom, Clackamas): Conditions 1–3 performed on a fixed platform with eyes open (1), eyes closed (2), vision sway-referenced (3). Conditions 4–6 performed on a sway-referenced platform with eyes open (4), eyes closed (5) and vision sway-referenced (6). Sensory analysis scores: Condition 2/Condition 1: somatosensory contribution to balance control (SOM), Condition 4/Condition 1: visual contribution (VIS), Condition 5/Condition 1: vestibular contribution (VEST). Composite score: global balance performance. Low score: poor postural control.
In this teenager, Composite score, VIS and VEST scores very low, SOM 100%. Lower ratios: inability to compensate for disruptions in selected sensory inputs.
The strategic analysis of postural control shows in the context of these sensory deficits the use of a bottom-up (ankle strategy/"Cheville Dominante") strategy in the 6 conditions and not a top-down ("Hanche Dominante") strategy, confirming preference of somatosensory input.

compensated" for the incurred insult as found in some "normals",[24] perhaps caused by hereditary defect or unrecognized trauma. This was also discussed by NASA, where flight vomiting in astronauts was suggested to result from asymetrical well-compensated vestibular disease.[22] Compensation may also involve maximizing recovery of a partially damaged system by a combination of healing of the damaged structures and utilization of other sensory system information to optimize function.

The process of compensation for vestibular pathology (which by definition documents the presence of pathology) can sometimes be detected in a clinical exam, completed by videonystagmography (caloric tests, head shaking test, head impulse test, skull vibration-induced nystagmus test…), but this is not always the case, and it is important for the clinician to understand that a patient with symptoms can sometimes have a normal clinical examination (*i.e.* absence of signs).

Compensation for musculoskeletal disorders: the cases of lower limb amputation and myopathies

In musculoskeletal disorders, the unlesioned CNS generates internal compensations taking into advantage external devices if needed.

Lower limb amputation

Lower limb amputation refers to the surgical removal of a portion of the leg at various levels depending on the severity of injury or disease. Prosthetic (Προθεσις, «action of adding») limbs are customized to replace the lost limb and are designed to provide function, mobility, and support. Advanced technologies incorporate sensors and artificial intelligence to adjust the prosthetic's movement to the user's activity to provide better control, energy efficiency, and balance.[25,26] In addition to prosthetics, other assistive devices may be used during rehabilitation or for additional support (crutches, canes, walkers).

Amputees rely on their CNS to adapt for the biomechanical and sensory changes introduced by using a prosthesis. According to the prosthetic design and alignment adjustment, the CNS generates internal compensatory mechanisms to maintain stability, control gait, and optimize energy efficiency. These adaptations are the result of a combination of proprioceptive feedback, motor control strategies, and learned motor patterns. To compensate for the loss of sensory feedback from the amputated limb, amputees may rely more on visual and proprioceptive inputs from the intact limb and upper body that can influence postural stability.[27] Over time, the CNS integrates sensory feedback from the residual limb and the prosthesis to create a new «body map» that accounts for the altered biomechanics.[28] In addition, the CNS alters gait mechanics to maintain balance and progression according to prosthetic alignment or mechanical limits. For example, reducing the stance time on the prosthetic side and redistributing weight toward the intact limb is a common compensation.[29] The CNS may compensate for the insufficient dynamic contribution of the prosthetic limb by higher involvement of the intact limb for weight absorption at the beginning of the stance phase[30,31] and for push-off generation. On the other hand, prostheses are continually being developed to better respond to the biomechanics of walking under different conditions: passive prostheses offer basic functionality, semi-active prostheses are able to modify their behavior instantaneously using microprocessor technology, and active prostheses provide external power through motors with the ability to act.[25] In summary, the internal CNS compensations allow amputees to adapt dynamically to prosthetic mechanics, ensuring functional mobility and reducing the risk of falls.

Myopathies

Myopathies are a group of disorders characterized by primary dysfunction of muscle fibers. Duchenne muscular dystrophy, leading to progressive muscle degeneration,[32] will be presented to describe compensation for balance disorder.

Early signs of muscle weakness such as difficulty in running or rising from the floor (Gowers' maneuver) appear usually in toddlers (2-3 years).[32] Muscle weakness progressively increases affecting first the trunk and then both the lower and upper extremities, with loss of independent ambulation from the age of 8-12 years.[33] Before losing independent ambulation, typical patterns of the MSS are seen related to muscle weakness and its compensation.

Weakening of the hip extensor and abdominal muscles causes the pelvis to tilt forward,[34] which is compensated for by exaggerated lumbar, and then thoracolumbar lordosis to maintain sagittal upright posture.[35] Later, thoraco-lumbar scoliosis usually develops due to uneven muscle strength and support.[35] Maintaining balance with these deformities requires energy, leading to quick fatigue and reduced endurance. This may need external compensation such as spinal braces to support the spine and reduce the progression of scoliosis and lordosis or walkers or canes to improve balance and reduce the strain on the spine.

Typical motor patterns usually involve the lower limbs. To stabilize the knee and prevent it from buckling due to weak quadriceps, individuals may passively hyperextend their knees during the stance phase by a slight forward shift of the trunk. However, weakness of the trunk muscles considerably reduces the trunk's ability to limit its forward movement when walking.[35] To compensate for this weakness, reduction in walking speed and step length allows to decrease the body sagittal acceleration and its muscular control.[36–38] Toe-walking, *i.e.* with forefoot or flat foot contact on the ground from initial contact, typically occurs as muscles weaken in Duchenne muscular dystrophy.[39,40] This pattern allows the plantar flexors to support the body, decelerate the anterior tilt of the tibia and the forward motion of the trunk from initial contact, ensuring a more gradual forward displacement of the trunk and overall gait stability.[36,40] In addition, the usually associated increased step width and medial-lateral body acceleration[37,40] enhance balance[41] with passive contribution of the weak hip abductors to limit the pelvic drop on the swing leg due to their stiffness and contracture.[38] Later, trunk leaning towards the stance leg (waddling motion) helps to lift the pelvis on the swing leg side for foot clearance.

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Compensation for impairment of the CNS: the case of upper motor neuron syndrome

When the impaired CNS induces balance disorders, it will also generate compensation.

UMNS refers to a group of clinical signs and symptoms resulting from injury or dysfunction of the Upper Motor Neurons (UMNs).[42] UMNs originate in the motor regions of the cerebral cortex and transmit signals to the spinal cord or brainstem, where they synapse with lower motor neurons, which activate skeletal muscles. UMNS can result from a variety of etiologies, including stroke and Cerebral Palsy (CP).[42] Key features of UMNS include symptoms related to disinhibition of spinal stretch reflexes commonly related to the loss of inhibitory input from UMNs.[42,43] These symptoms include muscle spasticity related to velocity-dependant increase in stretch reflexes. Other symptoms of UMNS are commonly related to disrupted corticospinal projections: muscle weakness, impaired fine motor control, and the Babinski sign.[42,43] UMNS can lead to various motor disorders, including balance control deficits and gait disorders.

Non-specific gait disorders, such as reduced walking speed and stride/step length, are commonly found in patients with stroke or cerebral palsy.[44,45] In both patients with stroke or hemiparetic CP, gait is typically asymmetric with a shorter stance time and longer swing time of the affected limb, which are correlated to impaired balance control.[46–48]

Adaptive motor patterns will develop to cope with balance disorders during motor activities such as walking.

Balance control is mainly automatic, involving the brainstem, which influences spinal motor activity under the regulating control of the cerebellum. Interestingly, in more than 90% of brain strokes, the brainstem and cerebellar structures involved in automatic process of balance and gait control are intact.[49,50] In post-stroke subjects, these latter structures are particularly active while walking.[51,52]

In post-stroke subjects, gait deviations can be described compared to the gait of able-bodied subjects, but the underlying mechanisms still need to be further understood. Primary gait deviations would be mostly related to the initial disruption of descending neural pathways, while secondary deviations are neural adaptive processes.[53]

Adaptation processes can be both automatic (sub-cortical) and/or cognitive. Both of these processes involve the cerebellum, which is usually intact. These processes can be ones which have already been learned, ones which are newly developed, or both. Common compensatory mechanisms that appear to be independent from the underlying disease have been described across different patient groups.[53] For example, in post-stroke subjects, reduced foot clearance in swing phase could be compensated by common compensations such as either a pelvic hike, circumduction or vaulting that can also be observed in various neural pathologies (*e.g.* foot drop by peroneal nerve palsy or multiple sclerosis) and orthopedic pathologies (*e.g.* hip or knee osteoarthritis or leg-length discrepancy).[53] On the other hand, in post-stroke gait, the asymmetric pattern with higher kinetic involvement of the unaffected side may be a specific adaptation to the initial and ongoing paresis of the affected side.[54] This adaptation may involve both automatic and intentional cognitive processes, for support, balance, and progression purposes.

Muscle co-activation, referring to the simultaneous activation of agonist and antagonist muscles around a joint, is commonly present in UMNS and typically included in the exaggerated muscle contraction as a primary abnormality associated to spasticity.[42,43,55] However, muscle co-activation is a common compensatory mechanism that provides mechanical stability by stiffening joints and can improve balance such as during very slow walking speeds,[56] walking on a slippery surface[57,58] or in elderly.[59] In the functional condition of walking in adults with stroke or children with CP, muscle co-activation in lower limbs is particularly frequent in both the affected and unaffected limbs[60,61] and may represent an adaptation to compensate for impaired stability during step transition.[61,62]

Toe-walking (defined as an absence of the first heel rocker or as failure of the heel to make contact with the floor at the onset of stance) is a common motor disorder in adults after stroke[63] and hemiplegic and diplegic children with CP.[64–66] Thus, these persons typically first strike the ground with the foot flat or with the forefoot, usually in ankle plantar flexion (*i.e.* equinus).[65–67] Dynamic equinus is generally believed to be due to premature (before the foot initial contact on the floor) over-activity of the plantar flexors, caused by spastic hyper-excitable stretch reflexes in these muscles.[55,66] However, the existence and/or functional significance of exaggerated stretch reflexes during walking in spastic patients has been questioned for years[68,69] with no evidence of exaggerated sensory inputs to the ankle plantar flexors during the swing phase of walking.[70]

The plantar flexors (gastrocnemius, soleus) i) decelerate the ankle dorsiflexion and the trunk's forward progression, and ii) accelerate the trunk upwards for body support, but only when the anterior part of the foot lever arm is touching the ground.[71,72] In toe-walking, plantar flexors exert an action from the initial foot contact onwards, contributing to increase the work of deceleration exerted by the leading leg joints during the weight acceptance phase of gait on the body's center of mass.[73] This early action of plantar flexors promoted by early flat-foot contact may be functional, *i.e.* a compensatory mechanism, and not due to primary muscular dysfunction. Interestingly, in children with CP wearing negative-heeled shoes compared to barefoot condition, a shift from ankle plantarflexion to dorsiflexion occurred quickly at initial contact under the effect of greater tibialis anterior activity, allowing flat contact between shoes and the ground and to maintain the same braking effect of the plantar flexors.[74] Thus, the footwear condition did not influence the early action of plantar flexors that contributes to the weight acceptance's role in controlling the body center of mass and balance during gait, suggesting a functional role of the plantar flexors.

Since the plantar flexors act indirectly on the trunk, they may be functionally linked to the trunk. From early childhood onwards, children with CP display trunk control disorders, while sitting[75–77] or walking,[78] strongly related to gross motor function deficits.[79] During walking, the body can be divided into two functional units; the locomotor unit and the passenger unit (upper body) and walking is efficient when stabilization demands of the passenger are minimized.[72] In children with CP, with poor control of

upper body acceleration, impaired control of axial segments during gait could account for gait disorders.[80] Recently, during the weight acceptance phase of gait in children with CP, a strong correlation has been demonstrated between the ankle dorsiflexion braking power due to plantar flexors and the upper and lower trunk deceleration.[81] In addition, a trunk-focused rehabilitation, that improved trunk postural control, decreased the excessive trunk deceleration and ankle dorsiflexion braking power during gait, strongly suggesting a functional role of the plantar flexors in dynamic balance control during gait.[81] Thus, this series of studies about the plantar flexors action in toe-walking in children with CP illustrates how difficult it can be to distinguish between the primary and secondary gait deviations in UMNS.

In summary, the control of balance is a priority motor function constantly ensured by automatic CNS processes that prevent the body from falling under the constant influence of gravity. Multisensory neural integration integrates the movements and dynamics of the body and its parts into the gravitational and visual environment, anticipates the effect of voluntary actions on the body's balance, and perceives unexpected balance disturbances. To compensate for balance disorders, internal compensation uses sensory, motor and cognitive resources and may be completed by external compensation based on assistive devices, adaptive equipments and environmental modifications. According to the type of body impairment, internal compensation may be more or less difficult to understand.
If there is a vestibular deficit, as an example of sensory impairment, compensation is undertaken using motor and cognitive adaptation and also reweighting of sensory information. Over time, a deficit may disappear due to total or partial vestibular recovery via this process. A "fully compensated" patient (*i.e.* with clinically normal gait pattern) should still be assessed diagnostically, as "decompensation" may occur (*i.e.* a new deficit can occur in modalities that were used in the compensation process).
If the MSS is impaired, it can induce balance disorders. Thus, the CNS will use the impaired MSS to compensate for balance disorders requiring at least modification in the motor output. Some motor modifications will specifically adapt to the impaired parts of the MSS whereas others will compensate for balance control. If an assistive device is needed, it would require a specific adaptation to functionaly incorporate it for balance control.
If the CNS is impaired, it can induce balance disorders but will also be involved in the compensation process. Depending on the location of neural impairment it can be difficult to distinguish between motor pattern directly related to the lesion, and motor compensation. For example, toe-walking was first thought to be a dysfunction related to central lesion in UMNS, but it actually contributes to compensate for balance disorders such as in myopathia or even when walking on a slippery walkway (using a flat foot strategy for maximum contact with the ground). In case of an impaired CNS, how a motor alteration contributes to balance control should be considered initially, as this can provide clues to help analyse complex motor disorders.

The highly adaptive capacity of the CNS to compensate for deficits is continuously utilized in newly developed devices such as external sensory substitution, *e.g.* vibrotactile belts for patients with severe vestibular loss.[82]

List of the abbreviations

CNS, central nervous system
CP, cerebral palsy
ESCEBD, European Society for Clinical Evaluation of Balance Disorders
VOR, vestibulo-ocular reflex
OKN, optokinetic nystagmus
OKR, optokinetic response
UMNs, upper motor neurons
UMNS, upper motor neuron syndrome

Conflict of interest

The authors declare no conflict of interest.

Ethics approval and consent to participate

Not applicable.

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