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RESEARCH ARTICLE

Sensory function in cerebral palsy: an overview and considerations for consequences

Reinald Brunner[1+], Walter Strobl[2+]

[1]University Children's Hospital Basel, Spitalstrasse 33, 4056 Basel, Switzerland
[2]University for Continuing Education Krems, Dr.-Karl-Dorrek-Straße 30, 3500 Krems an der Donau, Austria

+ shared first authorship

*reinald-g-h.brunner@unibas.ch

ABSTRACT

Alterations of sensory function are poorly recognizable but adequate sensory information and perception is a precondition for adequate motor function. It is of little surprise that in patients with cerebral palsy, the motor disorder is seen as the predominant lesion. This has led to the definition with motor overactivity and weakness in the forefront. However, sensory dysfunction was recognised already in the middle of the last century. It has been described more consistently in the modern literature and proven by fMRI and tractography studies. The sensory disorder can explain a wide part of the symptoms of cerebral palsy at least to some part. It comprises inappropriate and poorly controlled muscle activity, spasticity, impeded motor learning, developmental retardation, and pain. Treatment hence should focus on conveying the perception of stability and experiencing a wider range of different motions by more varying activities and functions. Early onset of treatment may help to reduce the developmental gap between patients and healthy children. Orthopaedic treatment, conservative and surgical, in the first place corrects biomechanically impeding deformities and provides mechanical stability.

Keywords: cerebral palsy; sensory function; motor disorder; perception; review paper

Introduction

Cerebral palsy is defined as a primarily motor disorder even in the most recent definition[1-4]. This complex of symptoms is the eye-catcher, which also found in the first description by Sir John Little and Sigmund Freud[5-7]. Movements are slow and poorly controlled. Short and stiff muscles as well as spasticity are further consequences. Functional and later structural deformities develop, leading to physical impairment and the need for compensations. The result is impeded participation in daily activities, schooling, and profession. A large spectrum of treatment approaches intends to correct these motor problems best possible but can hardly ever reach normality. Defining CP over motor symptoms, however, does not mean that mainly or even only motor problems are the primary problem. During the last decades, additional affections of neurological functions have been added as possibilities[1-3]. This includes deficits of the sensory system, eyesight, hearing, and cognition amongst others described already in the 50ties[8-10]. At the beginning of the 20th century, little knowledge existed about brain pathology and especially the functional consequences, and the state of brain examinations was poor. Hence It is not a surprise that visible motor dysfunction became the leading problem. However, in recent decades major progress has been made to understand brain function and to improve brain diagnostics by functional tests and imaging. New awareness and its impact on the understanding of cerebral palsy motivated us to write this article, which aims to merge new information from various sides and describe the consequences. However, some may appear philosophical and speculative.

How to evaluate sensory function in cerebral palsy?

Sensory function is difficult to assess in clinical settings, especially in children and patients who are not cooperative and unable to communicate. In 1958 Tachdjian found sensory lesions in hand examinations in 42%[9]. Regarding the difficulties in perceiving and communicating a deficit, this number is already impressive. The techniques to assess sensory function are still limited, and objective data can hardly be gained. As sensory function is essential for appropriate motor control, Poitras et al. carried out a systematic review on the impact of clinically assessed sensory function on motor deficits at the upper extremities. They found a moderate to high association for tactile function but were unable to draw a definitive conclusion, as the results were too heterogenic[11]. McLaughlin et al. described significant sensory deficits at the lower extremities in CP patients[12], and Sanger and Kukke found abnormal sensory function in dystonic and diplegic CP patients[13]. Prashad Mishra described disturbed sensory profile scores in all examined patients and the severity of disturbance correlated with functional mobility[14].

MRI visualises sensory and motor pathways

Another approach to diagnose sensory affections is the brain pathology itself. Typically in patients with spastic cerebral palsy, the brain shows defects of the white matter around the lateral ventricles, a lesion of the internal capsule. While the tracts in the spinal cord are well organised, this is not the case in the internal capsule. A lesion hence affects tracts of various contents: motor, sensory, and others. In more severe affections or other types of cerebral palsy, other structures such as brain nuclei and the

grey matter can be involved. The progress in radiological cerebral imaging increased our knowledge on the sensory side. In the last decade, progress of the elaboration of data from MRI exams allowed to study intracerebral pathways by diffusion tensor imaging and diffusion tensor tractography[15]. The technique of tractography further allows to visualize pathways of interest, such as motor and sensory pathways. Staudt showed the situation in spastic unilateral cerebral palsy (hemiplegia)[16]. While the axons of the upper motor neurons on the hemiplegic side are destroyed and the innervation of the affected side by the ipsilateral motor cortex is maintained, the axons from the sensory nerve cells still grow in from the periphery and arrive on the contralateral side of the brain. The brain hence needs to build up new pathways to transport the sensory information to the opposite side. New synaptic pathways are created for this compensation. Although in a chain of synaptic transmissions, information may be hidden at certain waystations of a chain of synapses, the relevant information can be recovered. The network can compensate for the inaccuracy of individual neurons and synapses[17,18]. Despite all these possibilities to control synaptic communication and recovery of sensory information, the recovery in patients with hemiplegic cerebral palsy remains incomplete, and the final information is unprecise[19]. More modern studies using AI for the analysis of intracerebral pathways revealed various and prior not known alterations of pathways in unilateral cerebral palsy patients and their correlations with the AHA test[20].

The introduction of fMRI and tractography some twenty years ago opened the possibility to visualise the defects of the white matter pathways[15]. Various tracts, sensory, motor, or others can be identified and studied according to size[21-26]. Of great interest is the correlation of tractography data with clinical data. Several studies found a higher deficit for motor and sensory affections of the intracerebral pathways with increasing severity of CP indicated by the GMFCS levels,[21-23]. An exception is the contradictory study of Rha[13].

Do sensory deficits cause movement disorders?

In a recent review on somatosensory function studies using imaging techniques Knijenburg et al. described that tactile function was affected in almost all brain affections, even malformations, in various degrees and qualities[27]. In contrast to motor pathways, somatosensory pathways can sprout through the lesion but reach a broader area at the somatosensory cortex. Papadelis et al. found a correlation between the grade of the affection of the somatosensory pathways and the clinical sensory deficits[19]. We can state, that the modern literature strongly suggests that somatosensory connectivity in the brain of CP patients is altered and is reflected in a sensory deficit. The somatosensory abnormality and sensory deficit have been accused of affecting motor function at the upper extremities[28,29]. The affection of the motor and somatosensory pathways correlates with the severity of the CP affection expressed in the GMFCS values.

It was already striking how long it took to prove the somatosensory alteration in patients with cerebral palsy. The most probable explanation in our view is the lack of reliable clinical assessment methods for non-cooperative and non-responsive patients although sensory deficits in clinical examination of patients with

CP are increasingly described. We must accept that besides the motor affection, somatosensory deficits are most probably present as well. Dan describes cerebral palsy as a sensorimotor disorder in his editorial 2020, too[30]. Even more, it is surprising that the consequences of somatosensory alteration are hardly ever discussed. Only very few papers link somatosensory affections with functional problems. A correlation with motor dysfunction of the upper extremity was described[11,29,31,32].

Besides the lack of structural sensory pathways, altered perception of sensory information is another factor leading to the complex sensory disorder. An inadequate body awareness has been described although it is difficult to assess[33]. Marsico et al. interestingly describe in a recent paper the difficulties which patients with upper motor neuron lesion to appropriately localise tactile stimulation[34]. This difficulty increases the inability for adequate motor activity.

Consequences of sensorimotor dysfunction

Somatosensory function provides information on the situation and change of the position of segments and joints, the tension of muscles and tendons, ligaments and joint capsules[35], and movement and acceleration. This information is essential for the nervous system to create an adequate motor answer on external forces as well as for planning movement[36,37]. If this information is lacking or the precision is poor, an adequate motor output is not possible. CP patients with somatosensory deficits have problems with controlling posture especially if they are challenged[38]. This means that in addition to the affection of the motor control side, the affection of the sensory side increases motor dysfunction. Muscle activities are wrong in time and inadequate in strength. Any movement pattern must be altered. Performing a specific task becomes more difficult and the movement will be abnormal. This includes walking. Let us consider in brief how walking is acquired. We copy the task we see in our parents and try to perform similarly. This learning process is a continuous chain of trial and error[39]. The brain shows adaptations even for connectivity[37]. We select a movement strategy to reach the goal. The muscle activation depends on the somatosensory information but also on the input of all other senses. We carry out the planned movement, and afterwards, we check whether we reached the goal and if there were difficulties. This check requires adequate sensory information again. Only on this basis the modification of the movement can be planned and a new trial is carried out. Once a satisfying strategy is developed, this is stored as a feedforward program and serves as predictor for future movements. Probably, this involves also sensory prediction[37]. Motor output hence depends on adequate sensory input, and sensory information is essential for motor learning and developing motor strategies. We used our cognitive function to learn this movement. But movement control has one more layer: the mental and psychological influence. If we do not feel safe on one leg, we will not dare to lift up and advance the opposite leg. If we succeeded with our new movement but on the cost of great fear, we will not choose this strategy again. Under stress, our muscle activity changes. Lack of sensory information and the inadequate motor control reduces the confidence in the supporting leg of the patient. The patient feels unstable,

insecure, and is more anxious about falling, which is compensated by an increase of tone and spasticity[40]. It results in less courage and motivation to explore new movements and activities, and it takes more time to acquire enough experience for building up motor patterns, which we see as developmental retardation. Indeed, Prasad Mishra found a lower activity level with increasing sensory deficits[14], and Dietz et al. described deficits of sensory organisation in children with developmental retardation[41]. The problem of lacking somatosensory information is complicated by the fact that other senses, especially vision, are affected in many patients with CP[42,43]. Visual information is another important sensorial input for developing behaviour and motor strategies, and is even more important as a compensation if other senses are defective.

Psychologic factors affect sensory function

Yet another level has major influence on our behaviour and motor function: the mental and psychological level. Human beings are not machines where sensory input combined with a movement goal produces motor activity. Psychological factors greatly influence our activities. Motivation is one of the positive factors which stimulates children to repeat movements again and again until the goal is reached. However, there are also negative factors such as fear and stress. Especially the fear of falling has gained a lot of interest in the past years reaching a number of more than 320 publications (PubMed #fear of falling#). This literature concentrates on adults and shows how this fear interferes with function and gait. However, we were unable to find similar literature for children with cerebral palsy although the sensory and motor deficits lead to similar problems. Especially adult patients able to express their feelings can articulate such psychological difficulties. If we are extremely shocked, we become petrified: stiff and unable to move. Patients with CP report that perceptual disorders that occur permanently or suddenly in everyday life cause psychological stress that affects the safety of their posture and movement. They have the feeling of being overwhelmed by too many sensory stimuli because the numerous environmental influences cannot be sufficiently processed. Stress and fear increase muscle tone and can be accused as one factor which leads to the high tone type of spasticity in patients with cerebral palsy[40]. Confidence in the legs produces a feeling of security and is essential for walking. In virtual reality, there is software which lifts you up to extreme heights such as a skyscraper, and lets you step out of a free plank. The height is frightening and locks you. It requires extreme voluntary strength to make even a little step forward although you are well aware that you are standing on an even and safe floor. This example shows the extreme power of psychological factors. The early development as a toddler comprises the management of upright posture. We learn to activate our muscles in a way that the external forces such as gravity and acceleration are controlled. It is, however, not only a motor task. It also requires the achievement of a feeling of security in the various positions which involve our senses. We will have difficulties developing this feeling of security if our senses do not provide adequate information. The consequence is that children with such affections like children with cerebral palsy have less motivation to be active[38,39,41], develop less movement variability in their motor strategies, and finally show a developmental retardation.

Pain and overloading sensory perception

However, there is not only a lack of sensory information. There is also aberrant and disturbing sensory perception such as pain. Pain causes fear and stress and may be another significant factor in influencing sensorimotor functions. Permanent malposition, biomechanical misalignment, and pathologic stresses on body parts cause chronic pain, the perception of which is also altered. Pain in CP occurs regardless of age and is underestimated. Studies in recent years have shown that all children with cerebral palsy, regardless of age, live with musculoskeletal pain, which is more severe the less mobile and the higher GMFCS levels of the children are[44,45]. This pain appears to be composed of two components: nociceptive pain and neurogenic neuroinflammation, which acts as a pain amplifier. Patients with CP are often unable to attribute pain but are nevertheless impaired in their drive for sufficient movement and upright posture. This pain avoidance strategy causes an additional "non-use" problem. Especially in less active, non-ambulatory patients, the inactivity of the skeletal muscles, the lack of muscle mass, and the lack of training lead to a lack of anti-inflammatory substances. Neuroinflammation is thus generated uninhibitedly when nociceptive nerve fibres are irritated, switches on the pain amplifier and weakens the body's pain defence[46]. This seems to be the cause for the development of underestimated chronic pain in children and adults with cerebral movement disorders. Pain is the strongest inhibitor of drive and motivation and the most important indicator of quality of life. Studies on quality of life show that children with CP do not have a reduced quality of life compared to their peers as long as their disability can be compensated and there is no pain[47]. As soon as musculoskeletal pain occurs, their quality of life is massively impaired[48]. This fact is not sufficiently recognised by practitioners and caregivers, and chronic pain and chronic fatigue are systematically underestimated (Jahnsen et al. 2003)[49]. Additionally, vascular ischemia of chronic spastic muscles may play a role in the development of a vicious circle enhancing chronic musculoskeletal pain in patients with cerebral disorders and needing more knowledge of the underlying mechanisms[50].

In addition to the locomotor system, pain primarily affects the gastrointestinal tract[51]. Severe gastrointestinal dysfunction associated with pain is observed in 23-33%[52], especially upper gastrointestinal disorders and dysphagia[53]. Regardless of its etiology, pain correlates with the occurrence of abnormalities of the locomotor system, such as postural and movement disorders. They are associated with an overload of afferent input and cause anxiety, stress, and insecurity in all complex movements, such as grasping and locomotion. This leads to avoidance behavior which, due to the non-use problem of the sensorimotor system, progressively reinforces postural and movement disorders.

Musculoskeletal and gastrointestinal pain and fatigue are common, they cannot be completely avoided but cause stress and fear, and lead to demotivation, avoidance, and non-use of the sensorimotor system. This fact may reinforce the sensory disorder.

Drawing consequences for the management?

In this context, recent studies interestingly conclude that afference-stimulating treatment procedures, such as physiotherapeutic

techniques, device-based procedures, and orthoses, may achieve positive effects on posture and movement. Suits applying compression and electric stimulation, as well as underwater treatments, warmth, reflex therapies, vibration therapy, and locomotion therapy, have been increasingly evaluated in recent years concerning their reported effects on the sensory system. Compression orthoses stimulate the neural mechanoreceptors, which are the primary pressure and stretch sensors responsible for exteroception and proprioception. Proprioceptive and exteroceptive input should thus enable training of the sensors with the improvement of depth perception and thus activation and strengthening of the tonic postural muscles. It is known from numerous studies in space exploration that proprioceptive and tactile stimuli must be permanently applied in the absence of gravity to maintain postural control and allow astronauts or cosmonauts to adapt more quickly to gravity after returning to Earth[54]. This basic principle of permanent sensory stimulus application takes up the complex method of "proprioceptive correction" and enables the treatment of people with severe brain damage and movement disorders in the context of neurorehabilitation[55]. Space-age corrective suits have been further developed in recent years as elastic, skin-fitting, full-body compression suits for the trunk or individual body parts, such as the pelvic belt[56-58]. A systematic review included 12 studies on dynamic compression orthoses such as Therasuit, Theratog, Adeli, and others. In children with cerebral palsy, significant changes in gait speed, cadence, step length, and symmetry were found in conjunction with training programs, so the authors recommend 18-60 sessions to achieve optimal results[59]. Suits with stimulation of mechanoreceptors by electrostimulation are also currently being tested[60]. Numerous observational studies and centre experiences report positive effects. Senso-orthoses with compression and/or electrostimulation can provide pain reduction, tone regulation, and improvement in patients' physiologically and psychologically important posture and movement. However, as in many other cases of multimodal conservative treatment measures, the evidence is not yet sufficient for a conclusive statement for most senso-orthoses in the current study situation. More knowledge about sensory functions is needed to understand their effectiveness, and to develop new tools that can support patients with cerebral palsy.

Revising current treatment concepts

Regarding the importance and influence of sensory functions, it is not surprising that focusing on mechanical factors does not completely correct the movement disorder in CP. Deformities of the locomotor system may force to take a pose or restrict movements which challenges motor control and balance. Correction of these deformities improves performance and function. However, correcting abnormal muscle activities may not necessarily have a similar effect as the reason may be inadequate sensory information or psychological reaction. In this context, it is interesting that recent reports cast doubt on the efficacy of spasticity treatment [61].

Therefore, we suggest examining the sensory pathways as best as possible. A profound and informative clinical exam of sensory function will remain difficult as the situation with small children and non-communicative patients will not change. We see a possibility, however, in

further evaluating MRI data in this respect in clinical routine. We need to develop a system where the whole treatment team of a patient has full information on sensory deficits, cognitive abilities, and psychological factors. We further need to find means and techniques to get information on altered sensation and perception including pain. All this information is essential for therapists who may better adapt their treatment plan to the sensory and cognitive capacities of the individual patient. Orthopaedic surgeons may be more alert for possible difficulties in the rehabilitation process. Orthopaedic technicians may find indications for specific devices easier enhancing the development of more helpful tools for daily life of patients with cerebral palsy.

Patients with sensory alterations react and produce movements, patterns, and activities, which differ from normal. Abnormal joint positions during function such as foot equinus and increase of muscle tension, which is regarded as part of spasticity, are mechanical expressions of their reaction[40]. Using these adaptations at the long term produces abnormalities of the locomotor system such as contractures and skeletal deformities. Especially orthopaedic treatment focusses on the mechanical correction of movement and posture. It is difficult, however, to imagine what would best correct the individual situation of an individual patient. The patient needs to learn to cope with his / her inadequacies and find the best compensation. For sure, additional musculoskeletal deformities lead to additional problems and difficulties for the patient. However, so called normal movement patterns may be illusory and even inadequate for a patient. We hence need to accept aberrant movement strategies as a consequence of the sensorimotor disorder but the development of deformities as additional obstacles needs to be prevented and corrected. Our treatment should focus more on the stimulation of senses given by challenging situations (sports or similar situations during the treatment sessions) and trust in the patients' possibilities to develop adequate compensations. Learning comes from errors, and thus we need to allow for errors. It will be a challenge for future treatment regimen to create challenging situations for our patients to guide their motor learning. Very early onset of treatment should aim at reducing the gap of movement experience and development between patients and healthy children. From the orthopaedic point of view, prevention and treatment of hindering deformities will remain the major task while muscle surgery, especially transfers, to improve function should be done with more caution. Normality as treatment aim will be difficult to reach if sensory problems persist.

Conclusion

We have good evidence that cerebral palsy is a sensorimotor disorder. The sensory defects as well as excessive and altered perception are poorly recognised and highly underestimated in respect of their importance. They affect motor function directly due to unprecise information and indirectly by their effects on psychological reactions and developmental retardation. These sensory alterations will affect the development of motor control with compensations for inadequate and disturbing perceptions. The best possible function should be the goal, not necessarily normality.

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