Neuro-orthopaedic gait disorders and activity-promoting measures

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Being able to walk upright is one of the essential skills that characterize people. Safe and efficient walking enables people to interact with their environment. To do this, they must be able to absorb and process all sensory information. Under healthy conditions, this happens automatically, i.e. largely without conscious control. In addition, efficient, economical walking requires a functional musculoskeletal system. The brain receives most of the information about the position of the joints and the state of tension in the muscles, fascia, tendons and capsule structures via mechanoreceptors, including the position of the feet. The stable base enables the entire body to straighten up against gravity (postural control). This enables unconscious control during walking so that the person can concentrate on their environment,
z. For example, you can watch out for traffic, hold conversations, avoid obstacles and react to bumps and sudden changes.

Strategies to ensure stability when walking

As people strive to avoid putting themselves in danger, they choose a strategy that enables them to remain stable based on their individual potential. If there is a loss of balance, humans fall back on phylogenetically older patterns to restore stability when walking, regardless of the cause. Before humans had straightened up and developed bipedal locomotion in their evolutionary history around 200,000 years ago, they also had to explore the environment in search of food and to flee from potential enemies. Our first ancestors came from the water. Without extremities, only the torso was available to move around in this medium. When the first creatures reached land, locomotion was still carried out with the trunk, although the rudimentary extremities were already in place. For the quadruped, the front and rear paws formed the stable fixed point and locomotion was initiated by the trunk. It was only when humans became erect that they were able to use their hands as gripping organs and make tools. For this purpose

The function of being able to oppose the thumb developed, which structurally led to the development of the thumb-saddle joint. The ability to run in order to hunt and run away from danger was made possible by the development of the big toe and its long flexor tendon. These abilities and the structural, anatomical developments necessary for them occurred at roughly the same time in evolutionary history (Leori-Gourhan 2006) [20, 21]. What came last goes first. This means that the ability to control the intrinsic hand and foot muscles is most impaired in the event of an illness and - depending on the extent of the pathology - is usually not fully recovered.

In the case of gait instability, two fundamentally different strategies can be observed to ensure stability of the stance leg: Depending on the individual's potential, either the inner arch buckles (eversion type) or the outer edge of the foot is subjected to increased load when standing and walking (inversion type). Both foot misalignments are accompanied by compensation strategies that move further upwards. The knee is either flexed more or extended more (Fig. 1) [22]. In the long term, this

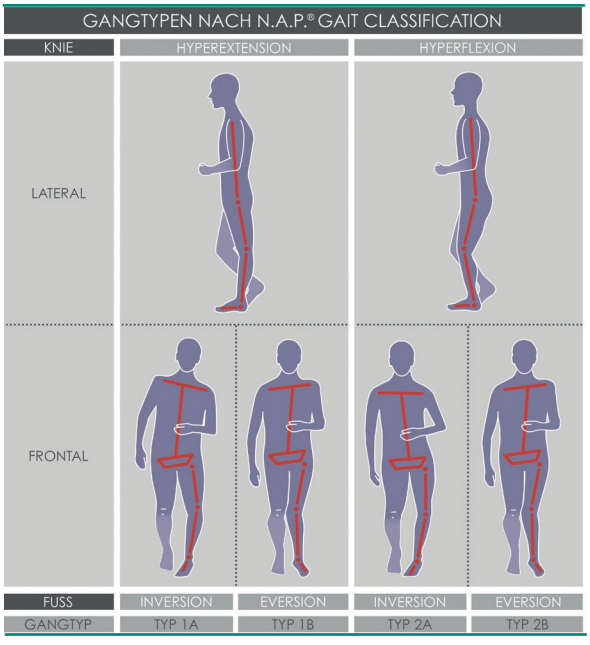

Fig. 1: Gait types according to N.A.P. Gait Classification (Sabbagh 2015)

joints are incorrectly loaded, resulting in secondary structural damage. In addition, this loss of postural control, which is associated with the inability to automatically adapt to changing environmental conditions, causes an increased risk of falling.

Biomechanical and neurophysiological prerequisites for stance stability

The stable forefoot is the prerequisite for both hip extension and dorsiflexion in the upper ankle joint (movement of the tibia over the forefoot) and the resulting controlled knee extension.

The stability of the ball of the big toe is ensured by the activity of the peroneal muscles and the intrinsic foot muscles. The posterior tibialis muscle straightens the medial arch of the foot. The stable forefoot enables the body's center of gravity to be shifted forwards and upwards, which is achieved through the primary activity of the hip extensors and pelvitrochanteric muscles. The double-jointed ischiocrural muscles pull the tibia backwards and upwards and cause the knee to extend.

The acceleration of the contralateral leg for the swing leg phase causes the tibia of the supporting leg to be transported over the forefoot. This is stabilized by the eccentric activation of the plantar flexors. The lower ankle joint moves in inversion (talus rotates outwards on the stable calcaneus). The activity of the peroneal muscles is also important to counteract the ongoing movement towards forefoot supination. The two-jointed The gastrocnemius muscle pulls the femur backwards and downwards. This passive restraining mechanism of the ischiocrural muscles and the gastrocnemius muscle creates an extension in the knee. Gravity exerts a flexor torque on the slightly flexed knee, which ultimately has to be stabilized by eccentric activation of the quadriceps muscle [23].

Biomechanical and neurophysiological prerequisites for swing leg initiation

The plantar flexors are not only important for stance stability and forward propulsion of the body in the push-off phase, but also for the initiation of the playing leg [20]. The activity of the long toe flexors, in particular the flexor hallucis longus and the peroneal muscles in conjunction with the knee flexors, enable foot and toe separation in the initial swing phase. This pre-stretches both the hip flexors and the tibialis anterior muscle so that the swing leg is automatically accelerated forwards and the back of the foot is lifted dorsally without much effort. If the extension synergy of the supporting leg is too weak the subsequent swing leg phase of the same leg is also impaired. In this case, the leg must be actively lifted forwards. This requires attention and hinders the automation of walking, which in turn increases the risk of falling.

Possible causes of gait instability

In order to be able to walk safely, a person must take in different information from the periphery, which is passed on centrally, where it is evaluated, selected and integrated with previous experiences. During this process, the brain stem in turn transmits nerve impulses to the periphery, to the eye, head, neck, trunk and limb muscles, with the aim of ensuring balance and the focused vision required for this. The cortex also plays a role here. Based on past experiences, e.g. having fallen on an uneven or slippery floor, we choose an appropriate movement pattern in order to be able to walk as safely as possible. Almost everyone has had the experience of walking on a slippery floor. You stiffen up to avoid a fall. The experience that this motor strategy is successful means that it is consolidated and can be used in repeated dangerous situations. Both peripheral and central injuries or illnesses cause balance disorders or result in the loss of postural control. Peripheral changes include loss of sensory and positional awareness, weakness of the erector muscles and muscle shortening that cause joint misalignment as well as visual and/or vestibular disturbances. Central injuries or diseases (Upper Motor Neuron Syndrome = UMNS) cause an impairment of the movement planning and control processes, as well as adaptive processes of peripheral structures. Dietz and Berger described the concept of "stiffness", which is caused by a decrease in sarcomeres, as early as 1983. The loss of activating signals from the central motor brain centers and the resulting muscle weaknesses (paresis) also lead to structural changes in the muscle fibers [6]. In this context, Hufschmidt and Mauritz (1985) described that a transformation from phasic to tonic muscle fibers can occur.

Protection mechanisms

If a person suffers an injury or illness, or if a degenerative process takes place, the primary location is

Whether the event is central or peripheral, automatically controlled mechanisms are generated that serve to protect the organism. Central pathologies are accompanied by changes in the periphery, and peripheral injuries cause central changes. This means that all disorders are ultimately part of a neuro-orthopaedic feedback loop!

First of all, it is important for the healing process that acutely endangered body structures are immobilized. To this end, biochemical processes are automatically set in motion via the direct connection of the limbic system with the autonomic nervous system and the hormone system (e.g. via the interplay of stress and growth hormones). Interestingly, there are descending nerve pathways from the limbic system that have direct access to the interneurons at spinal cord level. This means that if, for example, immobilization or stability is necessary - either to avoid pain or to prevent falls - muscles (agonists and antagonists) receive the appropriate information to prevent movement. This happens more or less unconsciously, so that muscles with a high tonic component are activated and muscles with a primarily phasic component are inhibited. Central disorders such as stroke or craniocerebral trauma often cause symptoms that look like weak foot dorsiflexion. The reason for this is not, as is often assumed, hyperactivity of the plantar flexors. In addition, the foot lifters often have sufficient strength. Since the work of Dietz and Benecke in the 1990s, it has been clear that the cause of central paralysis is predominantly reduced efferent central control [6]. This also leads to structural changes in the periphyseal muscles. The plantar flexors are compensatorily stiff due to their reduced activity, which is accompanied by a decrease in sarcomeres. This symptom (minus symptom) is often confused with a neurophysiologically induced hypertonus (plus symptom) [5, 6, 17]. Central lesions are not primarily associated with pain. However, the changes in the contractile and non-contractile structures described above often lead to secondary muscle pain and incorrect loading of the bones and joints. The result is an imbalance between strain and resilience, which perpetuates the pain symptomatology. The neurological patient then develops secondary orthopaedic complications. This often leads to a desire for passive means, be it medication and/or therapy, to relieve muscular tension or reduce tone. This usually results in a vicious circle: the patient has to stiffen up even more to compensate for the decrease in muscle activity.

Ultimately, an improvement in body functions can only be achieved by training them and this is even accompanied by a decrease in muscle tone! [6]

Plasticity

Plasticity describes the adaptability of all body structures as a result of functional requirements.

Synaptic plasticity

Originally, the term "plasticity" was used to describe changes in synaptic transmission. Donald Hebb, a Canadian psychologist, hypothesized that a stimulus that leads to synaptic transmission (unconditioned stimulus) can be linked to a stimulus that does not excite the synapse (conditioned stimulus), so that the latter will gradually excite the synapse. He hypothesized that short-term functional changes in synaptic efficiency cause long-term structural changes (1949).

Today, scientists agree that plasticity exists at many levels of the nervous system and that long-term changes in morphological structure depend on experience.

Morphological changes that occur as a result of experience are also quite stable. Long-term changes, which are fundamental for motor learning, can only be achieved when meaningful actions are practiced in real-life situations [18, 19]. Briones et al [3] have found that plastic changes resulting from (sensorimotor) experiences persist even if no further practice takes place for some time after the original training. This is not the case when pure movements are practiced without learning experiences. This is presumably because the brain has to refer to past experiences in order to be able to anticipate new ones [15].

From today's perspective, plasticity describes the brain's ability to adapt to activities through structural reorganization. To this end, the brain decodes memory content and stores memories. Neuronal plasticity includes synaptogenesis, the formation of new synapses, long-term potentiation and inhibition (LTP and LTD), synaptic sprouting, the connectivity and reorganization of sprouts and dendrites as well as neurogenesis or nerve growth. In addition, non-neural components are also shaped by experience. These are angiogenesis, the development of new vascular connections, the increase in myelination (also in adulthood), astrocyte hypertrophy and envelopment of synapses by astrocytes, which lead to a specialized exchange of information [1, 2, 7, 11, 12, 13, 15].

Cortical plasticity

Not only the strength of synaptic transmission and the formation of new synapses are shaped by experience, but also the cortical representation of different body parts is, according to current knowledge, shaped by experience. Body parts are represented in the cortex in activity-dependent functional connections or networks. These allow the individual to access situation-dependent motor programs [8, 9].

Thus, one and the same movement requires the networking of different neurons, depending on the activity in which it occurs. Representations are thus activity-dependent and therefore dynamic [4]. Consequently, the body representation in the brain can be restored by practicing activities if it is lost through disuse and/or pain. This means that if you want to learn to walk, you have to practice walking.

Activity-dependent plasticity

The concept of "activity-dependent plasticity" was already described by Merzenich in 1984. In his studies, he showed that when certain body parts are used to solve a motor problem, they expand in the cortical representation - at the expense of those that are not used.

For the patient who cannot use the plantar flexors and knee flexors to stand stably (eccentric function), transport their weight forwards and initiate their swing leg (concentric function), this means that they have to use more proximal body structures to perform these tasks. In addition, he cannot use the stretch-shortening cycle of these muscles, which leads to slower and therefore less economical walking. The swing leg is initiated, for example, by lifting the pelvis (circumduction) or rotating dorsally and lifting the thigh forwards. The trunk is therefore not the problem, but the solution to the problem.

Therapy planning

For a long time, the paradigm prevailed that practicing proximal body parts, e.g. pelvic movements against therapeutic resistance, was an important prerequisite for controlling distal body parts. The idea of being able to use stronger body parts to generate nerve impulses that are synaptically transmitted to the weaker ones is still anchored in some neurophysiological concepts today. However, this lacks any logic. This approach

For example, the pelvis would be neuronally networked for the initiation of the free leg, thus pushing the representation of the already underrepresented foot even further into the background. This may indeed be a goal, for example in a patient with paraplegic symptoms, depending on the lesion level and potentials, who is fitted with splints in which the knee joint is locked. A patient who has undergone an amputation and is fitted with a prosthesis made of heavy material and an immobile knee joint also needs a strategy to be able to move their leg forward by lifting their pelvis. However, patients who have knee flexor potentials can learn to swing their leg forward without compensating via the pelvis (as described in the following clinical example). In this case, the foot does not remain attached to the ground, even if the toe flexors are too weak to push the toes off the ground during the initial swing phase. It can often be seen that the potential of the knee flexors i s present within other activities, e.g. being able to lift the buttocks when the legs are upright in the supine position (Fig. 2). Patients often say that the movement of the knee when walking "is not in their head". At the body structure and function level, they are able to control their ischio- crural muscles to a maximum approximation against gravity, but still evade with the pelvis and do not use their existing potential when climbing stairs (Fig. 3a+b+c). The aim of therapy is to reintegrate knee flexion in combination with hip extension into the neural network for free leg initiation. Patients must learn how their body structures must function during the activity (Fig. 3d).

Fig. 2: During the activity of lifting the buttocks, the ischiocrural muscles are trained in their maximum approximation. The stable forefoot must be ensured by the function of the Mm. peronaeii

Fig. 3a: Before therapy, Mr. S. is unable to flex his knee against gravity up to 90° and deflects with his hip; 3b: After three therapy sessions, he is able to flex his knee 90° against gravity with a stable hip; 3c: When climbing stairs, he does not use the potentials at body structure and function level

Clinical example:

A patient in his early 50s, Mr. S., suffered a medial infarction with right hemiparesis and global aphasia. After three months of inpatient and a further three months of outpatient rehabilitation, he presented at the practice. He can walk with a walking stick, but very unsteadily. Tests confirm the risk of falling described by his partner. Mr. S. would like to walk his dog and increase his mobility by cycling and driving. He has gait type 1A (see Fig. 1). He was fitted with a suitable orthosis that enabled him to shift his tibia over his forefoot after six months of targeted training. Once he had achieved a stable mid stance phase, he was fitted with a

Fig. 3d: During the stair climbing activity, Mr. S. receives proprioceptive feedback for his external rotators, which stabilize his pelvis. The knee flexors are trained by the therapist's traction and guiding resistance

He was prescribed a new orthosis with appropriate foot orthoses, which enabled him to train the push-off phase. He trained his knee flexors independently, initially in combination with hip flexion during the activity of rolling onto his side from a supine position (Fig. 4a). He needed the same synergy to pull his foot out of the loop of his bicycle pedal and bring his leg into the car, which he then managed to do independently after a few months (Fig. 4b). Bridging" enabled him to train his knee flexors in combination with hip extension (see Fig. 2). After just three therapy sessions of one hour each, he was able to hold his ischiocrural muscles in maximum approximation even against gravity (see Fig. 3a+b). Over the course of six years, Mr. S. not only succeeded in independently walking 10 km to ride a bicycle. He can drive a car on his own and mit seinem Hund einen zweistündigen Spaziergang im Wald unternehmen. Hierfür zieht er seine letzte Orthese gelegentlich an. Für kürzere Gehstrecken benötigt er diese nicht. Herr S. hat seine Orthesen als »aktivitätsfördernde Trainingsgeräte« akzeptiert und mit Erfolg genutzt. Es konnte dokumentiert werden, dass er heute – sechs Jahre nach Beginn der Therapie – nicht mehr sturzgefährdet ist. Sein Dynamic Gait Index war anfangs 9/24 Punkte. Heute erreicht er 22 Punkte. Die Einzeltherapie ist immer mehr in den Hintergrund gerückt, und heute ist das primäre Ziel, seine allgemeine Ausdauer zu erhalten, die er in der Kleingruppe an Trainingsgeräten absolvieren kann. Dies motiviert ihn sehr, zumal es ihm gelungen ist, 10 kg abzunehmen, wodurch sich seine allgemeine Kondition enorm verbessert hat. Einzeltherapie benötigt er nur noch einmal pro Monat, hauptsächlich to check and document that and how his mobility is maintained and that the risk of falling does not increase again.

Fig. 4a: During the activity of rolling on the less affected side, Mr. S trains his ischiocrural muscles in the knee flexion function in synergy with hip flexion

Fig. 4b: After a few months, he is able to pull his foot out of the foot loop independently. To do this, he also needs his ischiocrural muscles in the knee flexion function in synergy with hip flexion

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