FOREWORD

The main motivation for me to begin work on this textbook was an effort to refine knowledge of the qualified public about rehabilitation and provide a framework regarding the true objectives of this field. Our profession is sometimes misconceived as massage therapy, exercising after orthopedic procedures, rehabilitation and sometimes it is reduced to only the use of therapeutic agents (modalities). I have also encountered the opinion that it is linked to or even directly considered some kind of an alternative treatment. Another important motivation for me was the lack of current study materials for physicians undergoing residencies, for graduate and post-graduate physical therapy students, as well as for physicians of other clinical specialties who want to be introduced to the methods of treatment rehabilitation used in their specialization.

In my view, I consider it essential that the foundation for rehabilitation treatment approaches be neither a trend nor a school of thought (chiropractics, osteopathy, musculoskeletal medicine), but rather a wide, general foundation in the fields of clinical physiology and neurophysiology. It also needs to be appreciated that rehabilitation is not only limited to diagnostic and treatment methods, but it also attempts to limit the extent of psychological, behavioral and social changes related to the consequences of an injury or illness. Therefore, rehabilitation should not be perceived as strictly a medical field but a field that over-reaches these boundaries and extends into the social, academic and work arenas. Comprehensive (integrated) rehabilitation applies to individuals whose health was compromised to a varied extent as a result of an illness, injury or a congenital defect and who require special assistance to achieve the highest possible level of independence. A person with a disability perceives limitations that they are unable to overcome while performing certain activities but they feel able and healthy in a number of other activities. Removing and solving these limiting problems is one of the particularly important tasks of rehabilitation. Therefore, the concept of rehabilitation must complement not only the treatment process but also the subsequent rehabilitation process.

From this point of view, rehabilitation is a very broad field which cannot be covered in detail in one book. Similarly, it is not possible to cover this extensive subject by one specialist. Success is based on a coordinated effort of various specialists.

In this book, I focused on the treatment component of rehabilitation and devoted more space to it than the educational, social and occupational areas. Given the fact that the diagnostic and treatment approaches of rehabilitation are focused primarily on the movement system, this field reaches into practically all clinical fields (neurology, orthopedics, internal medicine, oncology, immunology, psychiatry, etc.). Movement function plays an important role in all of these clinical fields. This is because physical activity and its repeated action manifest themselves by a change in function in a number of systems (cardiorespiratory, immune, central nervous system and metabolic changes), which allows for influencing these systems through modulation of its intensity, frequency and form. Another reason why rehabilitation reaches into several medical fields is the fact that the sensory afferent inputs from the entire body are always processed not only within its own sensory modality (visual, acoustic, proprioceptive, integumentary, etc.), but also within an integrated motor function. Our eyes, respiratory muscles, tongue, etc. serve not only the function they are dominantly selected for, but they also participate in postural and locomotive functions. This is well observed in athletic performances in which maximal force or a precisely accurate movement needs to be accomplished. For example, to strike a ball with required force, a tennis player makes a movement with their extremity, which is linked to a face expression, movement of the tongue in the direction of the stroke, eye movement in the direction of the stroke, modification of breathing by diaphragm activity (a grunt, Valsalva) to facilitate trunk stabilization, position of the contralateral extremity into the opposite (reciprocal) position etc. It is an overall involuntary movement pattern that interlinks individual sensory modalities and, thus, it is related to the majority of medical fields. This principle of modality integration within postural locomotor functions is a component of CNS development and it was established based on this principle. The fact that the described integration occurs at higher levels of control than the spinal cord and the brain stem is significant. This can also provide hypotheses regarding the effects of a number of alternative approaches whose justification of spinal cord and brain reflexology is not sufficient and is therefore substituted in clinical practice by alternative explanations. These central programs are organized above the brain stem level and can explain why functional pathologies become
chained in predetermined sequences; why needle application in a single point has functional consequences in a completely distant area of the body including the visceral region; why an internal dysfunction does not only show reflexive response in the corresponding segment but in quite distant areas and in various different modalities (skin hyperalgesic zones, changes in dermographism, muscle trigger points, joint restrictions, etc.); why respiratory function can be influenced through eye movement (eye movement automatically causes change in the breathing pattern); why breathing pattern changes with a change in hand position, and a number of other phenomena. The control system of the postural locomotor functions then provides us with a program that offers a completely new approach in the understanding of rehabilitation approaches.

Clinical diagnosis focused on symptomatology organized within postural locomotor functions should not be considered an exclusive component of treatment rehabilitation but also a component of the remaining clinical specialties.

I based the structuring of the General and Special Sections of the textbook on the function of the movement system in relation to individual clinical specialties. Therefore, I did not base them on diagnoses but rather on the functional manifestations of the disease. The General section of the textbook includes functional symptomatology and syndromology in dysfunctions of the nervous, musculoskeletal and internal systems and their clinical and laboratory examinations. The majority of treatment approaches are also presented in this context meaning that the treatment based on symptomatology and syndromology dominates. In the Special Section of the textbook, treatment rehabilitation is presented in individual clinical specialties – neurology, orthopedics, internal medicine, oncology, gynecology and psychiatry.

I purposely devoted less attention to occupational therapy, balneology and therapeutic agents (modalities) than these treatment approaches deserve. The reason is not to underestimate their value, but rather them already being reasonably available and sufficiently described elsewhere.

In clinical approaches of the General Section of the textbook, I have extensively drawn from and expanded on a trend known worldwide as the “Prague School.” In rehabilitation, the roots of this school of rehabilitation can be found in the Neurology Clinic of Professor Henner whose concept of neurology was very broad and therefore included even vascular diseases and movement system diseases within neurological symptomatology. Treatment rehabilitation was promoted by K. Obrda who, together with J. Karpisek, wrote the first rehabilitation textbook for neurological diseases and organized an international congress in 1965. On the theoretical level, F. Vele and O. Stary played an important role. Stary and K. Lewit demonstrated the significance of painful functional deficits of the movement system. In this aspect, the large contribution of Professor J. Jirout who was the founder of functional radiology of the spine, needs to be remembered. Thanks to the work of Professor V. Janda, the basic significance of movement patterns was gradually underwood and the term “functional pathology of the movement system” was established. This presentation was even further strengthened by the influence of scientific studies and personal contact with D.G. Simons and J.G. Travell to whom we are grateful for providing detailed knowledge of muscle trigger points that also cause a limitation in joint mobility, so called joint restrictions. To understand the function of the movement system, individual dysfunctions, such as trigger points and joint restrictions, need to be understood in the context of the entire movement system, i.e. the laws of chaining of functional dysfunctions. The key to this understanding was a better knowledge of the control function of motor skills. The new approach of treatment rehabilitation during movement re-education is based on utilization of knowledge about human motor development. This new trend enriches the current empirical and physical approaches by findings originating from the control processes of the CNS that mature during motor development. Dr. Vaclav Vojta, whose work we are currently trying to continue, has a significant role in this approach. Professor Vojta also came from Henner’s department and his conceptualization is an inherent component of contemporary clinical rehabilitation practice. Unfortunately, the neurophysiological principle of the entire approach to developmental kinesiology is still not fully appreciated due to disagreements about indication and the type of application of the Vojta method. However, not many critics understand the true basis of the Vojta approach. What is essential and substantial is not only the way that rehabilitation of movement dysfunction is utilized, but also the fact that the concept of developmental kinesiology is combined with the neurophysiological view relying on the findings of neurosciences associated with the currently predominant physical mechanical views.

In this book, I was also trying to respect and emphasize more certain general principles that condition the treatment effect, however, to convey the information in a written form is significantly limited for some of them. The respect for a comprehensive patient perspective is one such principle. The fact that human life occurs under specific biological, psychological, social-psychological, materialistically economic and ecological conditions needs to be implanted within the diagnostic, treatment and preventative approaches. Diseases and injuries cannot be viewed in isolation but rather need to be integrated within the context.
of such relationships because the treatment processes and rehabilitation are significantly affected by them.

I also aspired to prevent this textbook from becoming a proponent of only one method but rather support a variety of rehabilitation approaches based on a person’s individual needs. The problem is that this does not allow for providing a clear-cut treatment approach for movement dysfunctions because these approaches need to also be modified to the patient’s, and sometimes even the therapist’s, personality. In this context, protocols based on one uniform foundation outlining what and how much needs to be done cannot be implemented. These approaches are a method of choice, offering the option of finding individual solutions to how to effectively proceed and how to best modify the approach for a specific individual.

It is almost impossible to express in written form some principles that significantly affect the results of a rehabilitation treatment. This, for example, includes the mutual trust between the patient and the rehabilitation specialist, which cannot be substituted by a client-expert relationship or by a work performance contract. The importance of effective communication, charismatic approach, suggestive appeal and one’s own experience developed by sensory perceptions are additional examples.

Despite these limited options, I believe that this book will assist in better orientation in the broad field that rehabilitation truly is and thus will help fulfill the purpose for which it was written.

_Pavel Kolar_
body” exercise sessions, in which the entire body does not exercise all in one day, but rather the training of individual muscle groups is spread over several days.

Exercises commonly used in treatment rehabilitation usually do not put significant strength demands on the exercising individual. Their goal is mainly inter-muscular coordination. The low intensity of such exercises generally does not cause significant exhaustion of metabolic muscle supplies and, therefore, can be repeated several times per day.

### 1.1.4 Dynamic Neuromuscular Stabilization

According to techniques of dynamic neuromuscular stabilization (DNS) by Kolar, muscle function is influenced within its postural locomotor function. This concept contains general principles and, therefore, it is included among the general physiotherapeutic methods.

Common muscle strengthening is based on anatomical function. Strengthening exercises are derived from the muscle's origin and insertion. This principle is also utilized for the majority of strength training equipment found in fitness gyms. In treatment rehabilitation, this principle is utilized for exercises based on muscle testing.

However, when developing muscle strength, not just the muscle’s origins and insertions need to be considered, but also its inclusion within the biomechanical chains. However, these cannot be deduced only from anatomical connections (how they are presented by the majority of authors), but also from the aspect of CNS control processes (central programs).

If, for example, the pectoral muscles are exercised, the muscles that stabilize their insertions are also always activated, i.e. the back muscles, diaphragm, abdominal muscles, etc. This function is automatic and, in many people, under very limited volitional control. In addition, the deep muscles that are especially important for postural functions (stabilization, tightening).

Under static conditions (standing, sitting) and during movement (locomotion), individual movement segments are reinforced by the coordinated activity of the agonists and antagonist – co-activation activity (coactivation synergy) (Fig. 1.1.4-1a).

Postural activity accompanies movement like a shadow (Magnus, 1924). Perhaps this statement could be corrected to say postural activity precedes and accompanies every purposeful movement. Even though the muscle in its anatomical function (derived from its origin and insertion) can reach maximum potential (when assessed by a muscle test), its integration into a specific postural (stabilization) function (biomechanical chain) can be quite insufficient and the muscle fails in this function.

If the muscle is weak during the stabilization of a segment(s), postural instability occurs (Fig. 1.1.4-1b). Incorrect muscle recruitment during stabilization is automatically and unconsciously incorporated by the individual into all performed movements and exercises. This results in stereotypical overloading, which is an important etiopathogenetic factor in a number of movement deficits.

It has already been described in Chapter 1.1.1 The Assessment of Postural Function in Section A of the textbook, that postural instability cannot be assessed by a muscle test, but rather by specific postural tests and several of these tests were mentioned. An example of such postural instability is the lumbosacral deviation from its neutral alignment which can be observed in a patient in prone when slowly lifting their head and the upper part of the trunk (extension test) (Fig. 1.1.4-2a,b).

Postural instability of the lumbosacral area is manifested by an anterior tilt of the pelvis. Similarly, this postural instability is manifested during hip flexion assessment in supine and sitting, in which insufficient stabilization at the insertional region of hip flexors (inserting to the pelvis and the thoracolumbar region) leads to an anterior pelvic tilt, lateral shift of the pelvis and an extension or lateral co-movement at the thoracolumbar junction. This implies that the muscles (in some scenarios together with ligamentous structures)
that should be stabilizing this segment are weak or the muscle pattern is incorrectly learned. This postural instability is not limited only to the lumbosacral region, but also significantly influences the muscle coordination of the extremities.

To prevent overloading of soft tissues and the skeleton, the muscle activity, or rather the CNS and ligamentous structures, must ensure that stabilization of the segment(s) occurs in a centrated (neutral) joint position (see section A. Diagnostic Approaches, Chapter 1.2 Kinesiology and Clinical Assessment of the Joint System, Centrated Joint Position). For this to occur, a balance among the muscles in the entire biomechanical chain, as well as, between the exerted stabilization muscle force and the external force being overcome and, thus, form the basic presumptions.

A deficit in segmental joint stabilization is often caused by the following:

1. **Improper neuromuscular control.** Its main causes include:

   a) **Disturbance in postural ontogenesis.** A deficit in muscle interplay occurs in relation to abnormal postural development. It is an incorrectly established posturally locomotor pattern (Fig. 1.1.4-3).

   b) **Habituation of incorrect dynamic stereotypes.** A deficit in muscle interplay develops by an incorrectly learned and fixated activity (incorrect practice, profession with a unilateral postural loading, cultural and esthetic factors, inability to relax and the associated deficit in selective movement and so on).

   c) **Defensive (protective) CNS or muscle functions.** CNS adaptation to a pathological situation leads to characteristic changes in muscle tone, as well as, the entire posture.

2. **Insufficiency of muscles that ensure segmental joint stabilization.** Motor programs ensuring stabilization of muscle interplay have a “strength dimension”. This means that, under normal conditions, the stabilization function works physiologically and it can be used in this fashion only to a certain extent under loading from external forces. This is often used in diagnosis by adding resistance to a defined position or movement or challenging the external conditions, which assist in accentuating the movement, or rather the postural pathology. The example can be a leaning test in which the individual in kneeling with hands on the floor shifts weight onto the upper extremities. The quality of the muscle synergy can determine the insufficiency of the stabilization, especially in the shoulder blade and shoulder girdle area. Correctly performed exercises strengthen movement and muscle synergies ensuring an adequate postural stabilization for this movement. With every strengthening exercise, body posture is strengthened, including its dynamics. This is the reason why, during strength training, the principles of a functionally centrated position and the movement in it needs to be respected. Only in such a case do physiological stabilization muscle synergies strengthen together with the primary movement and, thus, the exercise will have a positive effect on the entire movement system.

3. **Ligamentous insufficiency and deficits in local, regional and global anatomical parameters.** The properties of mesenchymal tissues and the anatomical parameters (torsional and colodiaphysal angles of the hip joint, shape of the patella, alignment of the glenoid fossa, etc.) are important factors that influence joint stabilization during the activity of external forces. In contrast to muscle function, this situation cannot be significantly influenced by exercise but, can at least, compensate for it. In certain cases, corrective surgeries are an option.

**General Principles of Practice Techniques**

1. With a goal-oriented effect on the stabilization function, general principles are used, which are based on the programs maturing during postural ontogenesis (global patterns – ipsilateral and contralateral locomotion patterns, joint centration and its reflexive influence on stabilization function, facilitation by trigger zones, support functions, resistance against planned motion, etc.).

2. Exercises begin by influencing trunk stabilization, or the deep stabilization system of the spine, which is the basic prerequisite for a specific function of the extremities.

3. The muscles are trained in developmental, posturally locomotor lines. Inclusion of these muscles into chains, or the central biomechanical programs, allows for the modulation of automatic muscle activation in its postural function.

4. When selecting an exercise to influence (segmental) stabilization, it needs to be considered that segmental stabilization is never linked only to the muscles of the corresponding segment, but it always functions within the global muscle synergy arising from support.
5. Postural (reinforcing) force must always correspond to the muscle strength that executes the movement (phasic motion), which means that the force executing the movement cannot be larger than the strength of the stabilizing muscles, otherwise the movement emerges from an alternate source (they are performed by compensatory, stronger muscles).

Note: The choice of an exercise is determined by the desired goal to be accomplished. Volitional control of an automatic postural muscle function is one of the main goals. A learned synergy of the stabilization muscles can gradually be included in common everyday activities.

Practice of Postural Stabilization of the Spine, Chest and the Pelvis

In patients with postural instability, trunk stabilization needs to be addressed at first. The influence of the deep spinal stabilization system must be preceded by exercises in developmental lines. These exercises are based on the basic postural pattern (trunk stabilization) for a specific movement of the upper and lower extremities. No movement of the extremities (locomotion) exists without stabilization (tightening) of the trunk as a whole. Every phasic movement requires trunk stabilization whose control is usually deficient in the majority of patients demonstrating deficits in the movement apparatus and it needs to be corrected. In reflex locomotion, the stabilization activity of the trunk (linked to the breathing pattern) is the first activity that emerges during reflex stimulation regardless of starting position (supine, prone, in initial position, etc.).

The chest wall, spine and pelvis form a common base for all movement activities. The stabilization function is integrated into all movements automatically and non-volitionally. The muscle synergy that ensures it needs to be viewed as the foundation for all exercises. The main prerequisite is, again, the knowledge of the physiological synergy, or the so called ideal pattern of deep spinal stabilization (see Special Section of the textbook, Chapter 2 Treatment Rehabilitation in Orthopedics and Traumatology, 2.4.1 Spine, Vertebrogenic Pain Syndrome, Rehabilitation). Within this context, we originate from a body posture that emerges as a program during postural ontogenesis and we can elicit this program (synergy) reflexively.

In the majority of therapeutic concepts and preventative approaches, an erect spinal position is preferred as a starting posture. We all have heard the command “straighten up”. The individual concepts are not different. The difference is in the view of the chest, shoulder blades and pelvic alignment and, thus, the muscle synergy that ensures their stabilization. An erect spinal posture is recommended from an ergonomic perspective during common movement activities (housework, lifting objects), as well as, during specific stabilization exercises and exercises against resistance. From this perspective, for example, Brügger’s concept is well known. The so called back school is based on this concept. The initial model is Brügger’s sitting position which is called for as the basic working position (Fig. 1.1.4-4).

To achieve the desired erect position of the spine, a slanted support surface under the buttocks is recommended to tilt the pelvis more forward (anteriorly). By tilting the pelvis anteriorly, the elicited lumbar spine curvature leads to the straightening of the spine. The shoulders are pulled backward, the lower extremities are shoulder width apart and the feet are supported with its entire surface on the mat. The lower extremity joints are at 90 degrees. The described position of the spine, pelvis, chest and shoulders is included into common movement activities and it is used when exercising against variable resistance. Sitting and exercising in this position on an unstable surface, most commonly on a ball, is popular today. In contrast to the developmental concept (Fig. 1.1.4-5a,b), in this positional model, the role of the chest during the formation and control of intra-abdominal pressure is not adequately understood (Fig. 1.1.4-5b).
From a biomechanical perspective, the recommended alignment of the chest or a deficit in its dynamics do not allow for the required activity of the diaphragm and the corresponding control of intra-abdominal pressure via the lateral group of abdominal muscles, which predisposes the anterior stabilizers of the spine to weakness. A similar situation involves the pelvis, which is positioned in excessive antversion in patients with an inflexible thoracic kyphosis during straightening of the spine. Also, this concept insufficiently takes into consideration the level and distribution of muscle tone in starting positions, when performing a specific exercise and during common daily activities.

In patients with a disturbance in anatomical proportions (i.e., an increased pelvic tilt) accompanied by a disturbance in the control of intra-abdominal pressure, these facts are considered especially important because during stabilization, the patient uses excessive force in the superficial spinal extensors, which leads to an imbalance in the internal forces and overloading of the lumbar spinal region.

During trunk stabilization, the focus is on the following:
- The influence on tightness and improvement of chest wall dynamics
- The influence on spinal straightening
- Postural breathing pattern training and the stabilization function of the diaphragm (intra-abdominal pressure control)
- Postural spinal stabilization training using reflex locomotion
- Deep spinal postural stabilization training in modified positions
- Exercising postural functions in developmental lines

**Influence on Tightness and Improvement of Chest Wall Dynamics**

Alignment and the dynamics of the chest wall are important requirements for physiological spinal stabilization. With an erect thoracic spine, the thorax should be in an inspiratory position and should exhibit an isolated movement, meaning that the thorax should move independently of the thoracic spine. A co-movement of the thorax with the spine is pathological because an insufficient movement occurs in the costovertebral articulations and during expiratory and inspiratory movements of the chest, a flexion and extension co-movement of the spine occurs and originates mainly in the thoracolumbar junction (Fig. 1.1.4-6a, b, c).

This deficit is most often linked to the shortening of the accessory respiratory muscles (mainly pectorales and scalenes) and upper scapular stabilizers of the shoulder blades. While working on the inspiratory alignment, a release of the chest wall stiffness is performed (this is considered especially significant) primarily in the lower rib area. Only in an unrestricted thorax can the chest wall expand when the diaphragm expands, leading to widening of the intercostal spaces (mostly between the lower ribs).

**Practice Example**

Patient lies on their back, lower extremities flexed and in slight abduction (shoulder width), feet supported

![Fig. 1.1.4-7 Soft tissue release](image-url)
on the floor. The thoracic spine is straight. In this position, the soft tissues of the lateral chest wall are being released (Fig. 1.1.4-7).

In the same position, the thorax is passively positioned in the most caudal position. The pectoral and abdominal muscles must be relaxed. In this alignment, a slight pressure against the lower ribs is generated and the patient breathes in against the therapist's resistance (Fig. 1.1.4-8a, b).

Full excursion of the lower part of the thorax is pursued (including posterior direction) without the thorax moving cranially and without activating the superficial spinal extensors. The abdominal muscles, as well as, the accessory breathing muscles must stay relaxed. This exercise can also be performed using resistive exercise bands (Fig. 1.1.4-9).

**Influence on Spinal Straightening**
Training of spinal straightening is another requirement for physiological stabilization of the spine. Most often in patients with deficits in stabilization in the thoracic spine move as a rigid unit; lacking isolated movement at individual segments. For treatment, traction mobilization techniques are used and straightening of the thoracic spine is practiced. To achieve this, correct stabilization of the shoulder blades is important. Stabilization of the shoulder blades by a muscle pull toward the spine (into adduction) does not allow for its straightening and blocks the straightening activity of the deep paravertebral muscles. For these reasons, extension training, or ejection of the thoracic spine, is performed. This is performed at first with the upper extremity supported and, therefore, in a closed kinetic chain.

**Practice Example**
The patient lies on their stomach, forearms are supported on the mat, palms are placed on the mat and the head is straight. The patient leans on their medial epicondyles and by pressing them into the mat, the head is lifted with the intent of forward movement along the longitudinal body axis (Fig. 1.1.4-10a-f).

The head lift originates in the mid-thoracic spine. During head lifting, the cervical spine straightens without sagging in its caudal portion. The shoulder blades adhere to the chest and have a tendency to move toward the points of support. For thoracic spine straightening, the activity of the serratus anterior is very important with respect to scapular stabilization. Its stabilization activity is only possible with activation of the lateral abdominal muscles that, together with the diaphragm, form the punctum fixum. The proper function of the shoulder blades and shoulder adductors is important. Their direction of pull should not be toward the spine, but rather toward the support on the medial humeral epicondyles.

**Training of the Postural Breathing Pattern and the Stabilization Function of the Diaphragm**
A correct breathing pattern is another requirement for physiological stabilization of the spine. However,
the opposite is true as well: posture very sensitively influences breathing, which is known as postural respiratory function of the diaphragm. The goal is to include the diaphragm into breathing and thus into stabilization functions without participation of the accessory breathing muscles. Straightening of the spine and caudal chest alignment are required for this function. During inspiration, the ribs move laterally (wing movement), the lower chest aperture expands, the sternum moves ventrally and does not elevate with breathing. The abdominal muscles serve as a support for the diaphragm. It is important for the abdominal wall to expand not just in an inferior direction, but rather in all directions (i.e., laterally and posteriorly). The umbilicus should not move cranially (its movement reflects an undesirable muscle pull in the cranial direction).

Diaphragm activation has a fundamental role not only for breathing, but also for physiological stabilization of the trunk (see Fig. 1.2.3-5). The training is performed in various positions. During this exercise, the patient is taught to recruit the diaphragm, whose function during stabilization is not fully understood. After some practice time, with awareness and correction, we can indirectly distinguish its position without knowing its anatomical location.

Practice Example
The patient lies on their back, legs slightly apart, knees bent and feet resting on the mat. The patient moves their knees several times together and apart and then maintains them in a position that does not require a conscious effort. Another suitable position includes the lower extremities in shoulder-width abduction with the hips and knees at 90 degrees and the calves resting on a foot bench. In this position, the patient exhales, holds their breath and, without breathing in, moves the thorax and the abdominal cavity similarly as if they were breathing. The abdominal content behaves more or less as a liquid. Through this exercise, the pressure in the abdominal cavity changes. During instruction, the pressure from the abdominal cavity needs to be distributed equally in all directions, including in the backward direction against the mat and, in particular, the lower abdominal area should expand (the increased pressure should be directed down in the direction below the umbilicus into the groin and the pelvis). The same exercise is performed during inspiration.

Another version of this exercise in the above described position includes breathing training during increased intra-abdominal pressure. In this exercise, the therapist gently presses in a dorsal direction into

![Fig. 1.1.4-10a-f](image)

Physiological (a) and pathological (b) pattern in a 4-month-old child. Spinal straightening training with upper extremity support in a physiological (c, d) and pathological (e, f) position.
the patient’s groin area above the femoral heads (Fig. 1.1.4-11).

The patient must feel that the area of the abdominal wall above the hip joints presses against the therapist’s fingers. It is important for the force the patient exerts against the palpat ing fingers to not cause any cranial movement of the umbilical region or narrowing of the lower aspect of the thorax. In contrast, it must expand in all directions. Then, the patient practices breathing without relaxation activity of the lower part of the abdominal wall during expiration. The patient can also perform the exercise in sitting and in other modified positions.

In the next exercise, the patient is in the supine position. At the completion of the expiratory phase, the caudal position of the thorax is held and passively – by pressure from the therapist’s hands – pressed toward the center (proximally) at its lower aspect. The patient attempts to expand the thorax laterally against the therapist’s resistance and without inspiration, that is, similarly as if they were breathing in. The movement cannot be accompanied by accessory breathing muscle activity or a co-movement of the thoracic spine into flexion.

Arching of the lower thoracic and abdominal cavity without breathing is another version of this exercise. For accentuation, resistance is required and can be administered manually by a physical therapist (which allows for control of the executed movement’s quality). In the next phase, a resistive band can be used.

**Practice Example**

In the supine position with the lower extremity in a tri-flexion position (with supported lower extremities) and slight abduction (shoulder width), the 6th and 7th intercostal spaces are stimulated in the mammillary line by a slight pressure. The stimulation can be expanded by activating the nuchal line region on the contralateral side and the anterior superior iliac spine on the ipsilateral side of the stimulated thoracic zone.

The reflexive response is demonstrated by a change in the breathing pattern. Lower, or diaphragmatic, breathing occurs without participation of the accessory breathing muscles. The diaphragm flattens, meaning the punctum fixum is on the ribs, not on the centrum tendineum of the diaphragm. The thorax is positioned in a caudal alignment. At the same time, the spine straightens. The abdominal muscles are activated and act against the flattened diaphragm and pelvic floor, thus leading to an increased intra-abdominal pressure. The stabilization function of the abdominal muscles together with the flattening of the diaphragm (punctum fixum in the rib region) manifest themselves by a caudal shift of the umbilicus.

The patient is aware of the activation and the therapist strives for the given pattern to come under volitional control. This can be achieved by the involvement of the abdominal brace during diaphragmatic breathing and by gradual unweighting of the lower extremities. Then, the upper extremities movement against resistance is added into the exercise. The exercise can be performed in other positions.
Training of Deep Postural Stabilization of the Spine in Modified Positions

As soon as the patient at least partially masters control of the stabilization function and the physiological postural breathing pattern, the exercises can be performed in modified and more challenging positions or can include resistance (Fig. 1.1.4-13a-h). During treatment, it is important to select adequate exercises to avoid muscle substitution and compensatory patterns which the patient may have been using. The exercises can be modified to place greater emphasis on various muscle groups, for example, the deep neck flexors.

Exercising Postural Functions in Developmental Lines (Sequences)

The starting position for exercises is derived from the basic locomotor positions of postural development (development of body posture and verticalization process): supine, sidelying, side sitting, on all fours with support on the knees or the feet, tall kneeling, stepping forward in tall kneeling, etc., as well as from positions derived from locomotor transitional phases allowing the transition from one position to the next, i.e. a transition from side sitting to the all fours position, from all fours to bipedal standing, from supine to side sitting and later to standing, or locomotion. Thus, the posturally locomotor development that occurs as part of CNS maturation serves as the base for the starting position (Fig. 1.1.4-14 through 17).

During selected locomotion (i.e., the transition from side sitting to quadruped), individual parts of muscles, or individual muscles from muscle groups, gradually participate. Positioning in individual moments of locomotor movement also allows to selectively influence the postural function of parts of individual muscles, or muscle groups. It is exercising in a somewhat “frozen” position of a locomotor phase. Later, the entire transitional phase of the locomotor movement can be practiced from the starting side sitting position to a quadruped position (Fig. 1.1.4-18 and 19).

The selection of the original position is based on the patient's individual predisposing factors. The rule is to advance from positions with lower postural demands (for example, supine position with triflexion of the lower extremities) to positions that are posturally more challenging, which can also include unstable support surfaces and resistances. The initial education requires a therapist’s assistance.

The established starting locomotor position reflexively activates the deep stabilization system of the spine, ensuring trunk and spine tightening (see above). The upper and lower extremities become involved in the support and stepping forward functions. The stepping forward and support functions are part of two basic developmental patterns – ipsilateral and contralateral. Based on the position selected, the extremities on one side are stepping forward and the contralateral extremities are supportive – the ipsilateral model – or their function is reversed, i.e. if
toxication or metabolic dysfunctions. In children, the cerebellum can be affected by cerebral palsy. A deficit of the cerebellum results in an inhibitory function, which is manifested as disproportional, uncoordinated movements, apraxia, clumsiness, tremor and deficits in stability, gait, gaze and language.

1.13.2 Basic Clinical Manifestations of a Cerebellar Lesion

**ATAxia**

Ataxia is a basic movement deficit that occurs in the presence of a cerebellar lesion. It is a deficit in the coordination of volitional movements, their decomposition, clumsiness and inaccuracy. The movement is incorrectly timed or targeted, not smooth or efficient and shows many deviations into directions other than these intended. Based on the area or function affected by ataxia, it can be gait ataxia, postural (trunk) ataxia, extremity ataxia or language ataxia (cerebellar dysarthria). Standing ataxia is characterized by unsteady standing with a wide base of support with a tendency to fall in various directions. Gait ataxia is manifested by insecure wobbly gait with a wide base of support and a tendency toward falling (drunk walk). Step length is asymmetrical; the patient is unable to ambulate in a straight line. Patients often interpret their gait insecurity as dizziness; however, it is not a true vertigo.

Hypermetria, adiadochokinesia and asynergy are also components of ataxia.

**HYPERMETRIA**

Hypermetria denotes incorrect estimation of movement in a sense of overshooting. Movement begins and occurs too quickly, it is rushed, without a target and is inaccurately completed as a result of delayed and insufficient activation of the antagonists. Clinically, this deficit is most often assessed as an upper extremity dysmetria via the finger-to-nose test (Fig. 1.13.2-1).

A patient with their arm stretched in front of them attempts to touch the index finger to the tip of their nose as accurately as possible at first with eyes open and, if accomplished successfully, with the eyes closed. The assessment can be even more accurate by having the patient touch alternately the examiner’s index finger and their nose. The movement becomes gradually faster and the examiner changes the position of their index finger. Dysmetria denotes missing the target, overshooting is called hypermetria (see Fig. 1.13.2-1). Volitional effort to compensate for hypermetria is manifested as bradyteleokinesia, which is slowing of the movement prior to reaching the target followed by several compensatory movements to the side and only after that does the patient’s finger reach the target (nose). On the lower extremity,
ADIADOCHOKINESIA
Adiadochokinesia is a deficit in quick, alternating or repetitive movements. The alternating activation of agonists and antagonists is affected. The patient performs, for example, quick pronation-supination of both forearms. Movement typically lacks regular rhythm and individual phases are asymmetrical. Sometimes the movement may overshoot or undershoot and the physiological movement deceleration prior to reaching a target is absent. A certain degree of adiadochokinesia is physiological during preschool years when the cerebellum is still “maturing” and is frustratingly persistent in the non-dominant upper extremity. Adiadochokinesia can also be assessed in the tongue. The patient is asked to slightly protrude their tongue and quickly move it from side to side. The movement is non-rhythmic, uncoordinated and the tongue does not reach the corners of the mouth.

ASYNERGY
Asynergy is a deficit in the coordination of a muscle or a group of muscles during movement. The timing of activation of individual muscles into movement is missing. The muscles work without mutual functional continuity and more complex movements lack smoothness. Movements are separated into individual phases and the movement lacks timely activation of the antagonists to appropriately decelerate the movement. This lack of coordination is typically manifested during a test in which the therapist stands behind the patient and pulls them by the shoulders. With a cerebellar dysfunction, a physiological synkinesis of knee flexion is absent as the knees remain extended and, thus, the patient has a tendency to fall backward – manifestation of asynergy (Fig. 1.13.2-4). At the same time, excessive defensive synergies of the upper extremities can be observed and are manifestations of hypermetria. Asynergy can be also tested by the supine-to-sit test or sit-to-stand test, in which uncoordinated movement can be observed. A patient places their crossed arms across their chest and attempts to sit up from supine position. If cerebellar dysfunction is present, the patient excessively lifts their lower extremities above the mat. In the case of ipsilateral cerebellar hemisphere
involvement, the deficit is observed on the ipsilateral lower extremity (Fig. 1.13.2-5).

**FLACCIDITY**

Flaccidity denotes decreased muscle tone and, therefore, it is also known as cerebellar hypotonia. Range of motion is increased as a result of decreased resistance from the antagonists which lack timely deceleration of movement. This is demonstrated by the rebound test (Fig. 1.13.2-6). Increased synkinesis of the upper extremities during ambulation is a sign of flaccidity. Deep tendon reflexes are pendular. Joint play is increased and many signs typical for hypermobility are seen. Elementary postural reflexes are decreased or absent. Hypotonia manifests itself in endurance tests, i.e., during the Mingazzini test, a slow lowering of the raised extremity occurs.

![Fig. 1.13.2-6 Test to assess flaccidity: the examiner pulls on and quickly releases the patient’s raised forearm; a patient with cerebellar syndrome cannot stop forearm movement in time and it hits their chest](image)

**CEREBELLAR TREMOR**

A cerebellar tremor is an intention tremor which means that it is manifested during a specific movement. It is most prominent at the end of a movement and is best observed in the finger-to-nose coordination test (see Fig. 1.13.2-1) Titubation is a rhythmic, slow head or upper trunk tremor occurring mainly in the anterior-posterior direction.

**EYE MOVEMENT DISTURBANCES**

In a cerebellar dysfunction, eye movement disturbances are manifested by non-continuous movements consisting of a series of twitches. In principle, it is an uncoordinated movement of the eyeballs with signs of asynergy, hypermetria and an intention tremor. Cerebellar nystagmus is gross, occurs toward the side of the lesion and becomes more prominent when looking toward the affected side.

**PALEOCEREBELLAR AND NEOCEREBELLAR SYNDROME**

To a certain extent, the phylogenetic-anatomical division of the cerebellum also possesses functional significance. The verminal (medial) part mainly coordinates movements of the eyes and the body in relation to gravity and movement of the head in space. It especially affects the trunk musculature, balance and erect body posture. With a simultaneous deficit in the archicerebellum, eye movement disturbances and gaze paretic nystagmus are present. These medial (verminal) lesion manifestations correspond to the paleocerebellar syndrome, which can be combined with archicerebellar involvement.

**PALEOCEREBELLAR SYNDROME**

Clinically, the paleocerebellar syndrome is manifested mainly as deficits in standing and walking with staggering and a tendency of falling in various directions (trunk or axial ataxia). Paleocerebellar (previously known as large) asynergy is manifested by a coordination deficit mainly in the trunk and the proximal lower extremity muscles, incorrect judgment of movement to maintain standing, transition from sitting or from supine, backward bending or straightening. Spontaneous falls in various directions, especially backward, are common when the central part of the cerebellum (vermis) is injured.

**NEOCEREBELLAR SYNDROME**

The neocerebellar syndrome develops when the cerebellar hemispheres are injured and it is manifested mainly as movement deficits in the ipsilateral extremities. In contrast to pyramidal pathways, cerebellar pathways cross twice and, thus, each hemisphere always affects the ipsilateral extremities. The neocerebellar syndrome displays hypermetria, dyscoordination and adiadochokinesia of movement in ipsilateral extremities. An extremity asynergy (small asynergy) is demonstrated mainly in fine motor skills of the upper extremities. Purposeful movements are disturbed by an intention tremor and increased passivity; neocerebellar ataxia occurs. Lower extremity involvement leads to deficits in standing and walking. Pathological lesions usually overreach the anatomical boundaries of cerebellar regions and, thus, clinical symptomatology is mixed.

**PSEUDOCEREBELLAR SYNDROME**

Pseudocerebellar syndrome develops with a frontal lobe injury. The clinical picture is similar to paleocerebellar syndrome with gait dysfunction known as frontal ataxia being the most obvious. Ambulation is wide-based, very unsteady and the patient feels like they are being pulled backward and shows a tendency
to their home environment and continuity of care needs to be ensured for such patients (rehabilitation institutes, skilled nursing facility). When the progression is favorable and the patient has an appropriate home environment, the patient can be discharged to home care. Some patients after a CVA need to utilize caretaking services to a various extent following their discharge to a home setting.

Mobility deficits, limited independence and a symbolic function deficit greatly affect the patient's life. To accept this new reality is one of the hardest tasks that the patient and their caretakers need to deal with. Here, an important role can be played by non-profit and public organizations. In the Czech Republic, the Association for Rehabilitation of Persons after Cerebrovascular Accidents was formed from an initiative of patients after CVA and healthcare workers. Integration back into the society of patients after CVA is the main goal of this organization. The public organization "Afazie" offers programs and counseling for patients after CVA with a speech deficit.

In conclusion, it needs to be emphasized that patient care during hospitalization after a CVA is provided by an entire team of collaborating specialists. No less important is the assurance of appropriate care when the patient is being transferred to a home setting. In the entire system of care for patients after CVA, the family physician has an irreplaceable role and often is the first person in contact with the patient during their transition to their home setting and ensures further follow up care.

Comprehensive balneologic treatment in patients after a CVA is indicated when the acute stage subsides, especially in cases when it is presumed that the affected functions are recovering. It is mainly beneficial in assisting in the renewal of mobility, improving independence and the patient's quality of life. A statement from an internist regarding the patient's ability to handle physical demands from the cardiovascular perspective also needs to be provided. Comprehensive balneologic treatment is administered based on the recommendations from a neurologist or a rehabilitation physician. It is contraindicated in patients who suffered a CVA more than twice and also in patients with a severe phatic deficit or cardiac insufficiency. In the Czech Republic, balneologic treatment for patients after a CVA is available in Dubí, Karvina, Msene, Velké Losiny, Vraze and Janské Lazne.

1.21 CEREBRAL PALSY

Cerebral palsy (CP), also known as infantile cerebral palsy (ICP), was originally called Little’s disease after London physician, John Little, who was the first to describe it in 1859. It is defined as the neurodevelopmental and non-progressive involvement of the child's motor development due to sustained (and completed) prenatal, perinatal or early postnatal brain damage. The damage, which occurs during the perinatal or postnatal period, is not stable and continues to progress. Postnatal scarring, progressive atrophy, gliosis with retraction or cavitation develop. Brain imaging methods may or may not show clear signs of disturbances – microcephaly, macrocephaly, hydrocephalus, porencephaly, agenesis of gyri, lissencephaly, etc. In patients with CP, motor deficits are regularly seen; however, in many cases other systems can be involved as well.

Epidemiology

Cerebral palsy affects 1.5–2.5 out of 1,000 newborn children. Its incidence has not been linked to a decrease in newborn mortality in recent years. Long-term incidence is being monitored in Australia, Denmark, the USA and, especially, in Sweden.

Data about the incidence of children with cerebral palsy in the Czech Republic is quite different. In the Czech Republic, there are 16,000–20,000 children with cerebral palsy and about half of them require continuous rehabilitation.

Causes of Onset

The causes of brain damage can be diverse. They can be divided into three groups:

1. Prenatal – intrauterine infections are prenatal factors that most often cause brain damage. This category often includes TORCH (toxoplasmosis, rubella, cytomegaly, herpes infection). Other causes include developmental malformation, drugs used by the mother, etc. A number of these factors can lead to various degrees of premature birth. Premature birth is one of the etiological factors of CP for two reasons – it involves delivery of a very fragile head of the baby through a firm birth canal and, at the same time, the child is being born without completely matured biological functions. Genetic factors are being continuously discussed, but so far they are not a proven etiological factor of CP.

2. Perinatal – abnormal childbirths are perinatal factors that most often cause the onset of CP. They result in a brain trauma, especially ischemia and hypoxia. Ischemia and hypoxia selectively damage individual brain structures based on their level of maturity and vulnerability. The key role in brain damage by hypoxia or ischemia is played by excitatory amino acids (aspartate, glutamate) and by the activation of N-methyl-D-aspartate receptors with subsequent influx of calcium into the cells, which leads to cell death if cells do not show sufficient activity. In pre-term babies, hypoxia and ischemia...
are a result of periventricular leukomalacia. In term babies, a selective neuronal necrosis occurs in predilected areas, such as the hippocampus, cerebellum and the basal ganglia. Periventricular leukomalacia (PVL), especially its cystic form (cPVL) is considered to be the main predisposing factor for the onset of CP. Bilateral occipital PVL, identified by an ultrasound during the neonatal period, predicts occurrence of CMO with an almost 99% certainty.

3. Postnatal – these include mainly early newborn infections, most often bronchopneumonia or gastroenteritis.

**Etiology and Pathogenesis**

Etiology and pathogenesis of CP are a multifactorial interrelation of individually predicted factors of development and are a subject of constant clinical-epidemiological reassessment. It is primarily a severe prematurity (under 32 weeks of gestational age or under 1,500 grams) that predisposes the immature newborns to cerebral morbidity and severe deficits in neuromotor and mental development due to structural and functional immaturity of the CNS, circulatory instability with a tendency of pressure passive cerebral circulation, insufficient activity of the anti-oxidative defense system or increased sensitivity in regards to toxic activity of excitatory amino acids. Occurrence of CP and its manifestations vary depending on the level of prematurity or birth weight. Jessen at al. found that over 40% of children with early cerebral mortality, without taking into consideration gestational age or birth weight, later demonstrated a severe deficit in movement functions. They also pointed out the fact that the frequency of involvement in physical abilities does not vary among individual birth categories, but the incidence of involvement of cognitive abilities increases with the infant's decreased birth weight.

**1.21.1 Screening for Risk of CP**

Cerebral palsy occurs in 8–10% of children born prematurely. Approximately 40% of all children with CP are born prematurely. Screening aimed at neuromotor development is an essential step in timely recognition of children with CNS involvement. Children who show abnormal models during spontaneous motor behavior and with positional reactions are included in a clinical unit known as central coordination disturbance (CCD). It is important to realize that CCD does not mean that the patient will develop CP. Cerebral palsy develops only in a very small percentage of children in whom CCD has been identified.

Based on the grade of CCD (grade 3 and 4), repeated neurological assessments are performed and assis-tive methods are indicated (specific metabolic screening, neurophysiological examination, ultrasound or other imaging methods – CT, MRI, genetic and other testing). A differential diagnosis of motor function deficits (elimination of etiology other than CP) must be performed no later than the child's adjusted age of nine months. However, the actual identification of CNS involvement needs to be established much earlier, no later than at two months of age.

Timely identification of children with CP allows for the indication of early care and means earlier initiation of therapy. This approach can significantly decrease the functional consequences and prevent motor and cognitive complications of a late diagnosis. During the assessment of postural functions, the question remains how to define the criteria outlining the severity of a deficit that requires the child to be included in therapy. It is important to identify the children with a non-significant central deficit in muscle function in whom spontaneous correction occurs.

Vojta's screening of postural development, as well as, other methods are used in the assessment of newborns and infants at risk who are suspected of delayed psychomotor development (see General Section of the textbook, A. Diagnostic Approaches, Chapter III Neuromotor Development and its Assessment).

**1.21.2 Types of Cerebral Palsy and their Clinical Presentation**

Despite the overall diversity of the clinical picture of CP, usually a movement deficit is the dominant manifestation, which is usually visible in the earliest stages of the disease and poses the greatest problems to the patient. The character of the motor deficit is given by the involved CNS area. Based on the clinical picture (dominating signs), several forms of CP can be distinguished. They develop gradually during brain maturation and have different prognoses, different predispositions for development of contractures and joint deformities and also respond differently to the same therapeutic approaches.

**SPASTIC DIPLEGIA**

It belongs among the most frequent forms of CP. Numerous incidences of diplegia are reported by various authors. The data ranges from 41% to 65%. The severity of motor involvement differs. Spastic diplegia affects patients who achieve independent bipedal locomotion without support, but also patients who are completely apedal. Even if the patient is capable of independent bipedal locomotion, gait always shows pathological features. In a classic form of spastic diplegia, the lower extremities are always more affected than the upper extremities. Spastic diplegia
usually develops as quadriplegia in which neither extremity fulfills its basic function, such as support and grasping by the upper extremity and support and stepping forward by the lower extremity. By gradually including one upper extremity into a specific motor function (even if it is a pathological motor skill) spastic diplegia develops in a stage of triplegia and, by including both upper extremities, spastic diplegia occurs. Often, the plegia is asymmetrical and, in some patients, the dysfunction can result in monoplegia, which can be observed, for example, in unilateral leukomalacia. Spastic triplegia is being reported as an independent form of CP. The number of children with spastic triplegia has increased with the increased number of children who survive significant prematurity. A half of these children demonstrate epilepsy and only a third of them present with normal intellect. It is a very severe motor deficit that is very difficult to influence with therapy. Etiologically, intraventricular bleeding with asymmetrical infarction is common. Severely involved patients who demonstrate a more significant deficit in the upper extremities or in whom all four extremities are equally affected have a bilateral hemiplegia syndrome. It is reported that the incidence of this syndrome is 27% out of all patients affected by spastic diplegia. In most cases, the patients with bilateral hemiparesis also show mental impairment.

Approximately a third of children with spastic diplegia are born prior to 32 weeks of gestation, a third are born between 32–36 weeks and a third are born at term. Prematurity is linked to high incidence of perinatal factors.

The first manifestations of spastic diplegia can already be observed in the early stages of development through an assessment of the child’s motor behavior. During the first months after birth, especially in congenital motor deficits, a pathological motor pattern is visible, but it is not yet defined. It is non-specific or common to a number of later developing forms of CP or other syndromes. The clinical picture becomes more distinct only during the second or the third trimester after birth.

In any case, motor development lags behind the quality of motor patterns that are typically observed during physiological motor development (see General Section of the textbook, Chapter III Neuromotor Development and its Assessment).

All children with spastic diplegia present with a pathological postural foundation followed by pathological phasic mobility. The normal developmental pattern of eye-hand-mouth is not present; however, so called dystonic attacks are observed. They occur following acoustic or visual stimuli linked to an emotional component – when the child attempts to accomplish a specific task, for example, when grasping an object being passed to them. Dystonic attacks present mass generalized movements of the entire body in the patterns of tonic neck, tonic labyrinthine or other primitive postural reflexes or in their various combinations.

Dysmorphic features such as a gothic palate or pseudoharrison’s ridge below the ribcage (it is present in rickets) are typical in children with spastic diplegia. In more severe forms, approximately 20% of children with spastic diplegia show some signs of rigidity (more commonly in flexion types).

**SPASTIC HEMIPLEGIA**

Hemiplegia is a unilateral deficit in mobility most often spastic in nature. The entire half of the body is affected, including the facial and hypoglossal nerves. Spastic hemiparesis can be divided into congenital and acquired. If the acquired hemiparesis develops in infancy, it is difficult to distinguish it from congenital hemiparesis, especially if the episodes were present prior to the hemiparesis being identified. Pseudofacial stadium and central paresis of the facial nerve are more likely to indicate acquired hemiplegia. Acquired hemiparesis of vascular etiology requires a specific diagnostic and treatment approach. In contrast to congenital hemiparesis, aphasia also occurs in left-sided involvement. However, it is also possible that when the genotypically dominant hemisphere is affected, no developmental delay may be observed. Epilepsy poses a significant complication in children with spastic hemiplegia. It affects more than one third of patients. The seizures are focal or secondarily generalized. Most of them can be controlled by treatment. The presence of epilepsy is significantly linked to mental retardation. More than 50% of children with epilepsy suffer from mental retardation. In patients with hemiplegia, mental retardation can be identified in almost half of patients; in children without epileptic seizures, about one third presents with mental retardation. The severity of hemiplegic involvement, which is distinguishable mainly from the upper extremity involvement, is also closely linked to mental retardation. Hemiplegic form of CP affects more boys than girls and right-sided hemipareses occur somewhat more often. Divergent strabismus may also occur. Homonymous hemianopsia can also be present (it can nearly be eliminated by treatment).

The growth of the hemiplegic extremities is delayed in comparison to the healthy extremities. Hemihypogenesis is more pronounced on the upper extremity, which is almost always more affected than the lower extremity. It is measured based on the severity of involvement. The extent of the involvement can be assessed by the ability to perform an isolated movement and by the ability to assume a joint position that is developmentally typical of a younger age. In a mild form, the fingers can perform isolated movements.
In a moderate form, only the entire extremity can be moved and in a severe form, no isolated movement of the hand or any other upper extremity segment is possible. Isolated movements are also linked to the ability to attain a position in a movement segment. A child with spastic hemiparesis attains newborn positioning, which includes shoulder protraction, adduction and internal rotation, elbow flexion and protraction, wrist flexion and ulnar deviation, and finger flexion. Active shoulder flexion, abduction, and external rotation, and elbow extension and supination are assessed. In a hand, wrist extension, thumb opposition and abduction and flexion are being assessed. The more the child is able to attain a position close to physiological development, the lower the level of their involvement. In severe forms, the patterns typical for a newborn persist.

In children with spastic hemiplegia, not only the bones, but also the muscles are delayed in development and hemihypogenesis of the corresponding side develops (leg length discrepancy is on average 1.5 cm with 1–3 cm difference in circumference).

**CEREBELLAR FORM**

A cerebellar form as an individual occurrence is almost never present. Fetal factors contribute to a great extent on the onset of involvement. In the majority of children with a cerebellar syndrome, mental retardation is also observed but it usually is not severe. Sometimes, a cerebellar syndrome is accompanied by autism.

A cerebellar form of CP develops in relation to brain maturation and the specific signs of cerebellar involvement appear gradually based on brain structure maturity and their inclusion within motor function. Clinical signs, which include muscle hypotonia, trunk ataxia, hypermetria, intention tremor, and trunk ataxia all occur gradually during the process of CNS maturation.

**Clinical Picture**

**Hypotonia**

Central hypotonia, and with it, related delayed locomotor development dominate the clinical picture of a cerebellar syndrome. All muscles are flaccid and the joints can be bent to large angles. Joint excursion and muscle flexibility, as criteria of muscle tone, are manifested by *scarfs sign* in which the patient is able to cross their arms around their neck like a scarf. It is assessed whether the elbows are away from each other, above each other or are crossed over each other. Another sign is the *drawing compass* during which the lower extremity can be bent at the hip joint all the way to the trunk. In sitting, a child’s trunk can be bent to their lower extremities, which is known as the *arma-

**dillo sign*. Next, the flexibility of the knee and elbow flexors and extensors is being assessed followed by ankle range of motion, hand, thumb and finger flexibility, arm raising test, etc.

Hypotonia in childhood age is not the only manifestation of cerebellar involvement, but often, it is a mere transitional stage of CP. Quite different syndromes may arise from hypotonic forms. The onset of hypotonia is time-dependent on CNS maturity or rather its immaturity. Hypotonia in infancy always needs to be assessed for differential diagnoses. Some level of relative hypotonia can also be considered normal between 2–5 months of age.

Joint range of motion and muscle flexibility are not only dependent on the extent of the pathological process, but also on the physiological state of the nervous system and its developmental stage.

Reflexes are preserved or only slightly decreased. Thirty percent of children exhibit epilepsy.

**Dysmetria**

This is the incorrect targeting of a movement. The direction of the movement is correct, but the end of the movement shows hesitation. Thus, movements become inaccurate.

**Intention Tremor**

This is a tremor observed prior to reaching the target of movement. The movement is rough, irregular, rather slow and with large amplitudes. Typically, an intention tremor subsides prior to movement completion. It is seen in the upper and lower extremities.

**Trunk Ataxia**

It is a lack of coordination of the trunk caused by a disproportion between contraction intensity and the type of movement.

**Asynergy**

Asynergy is a deficit in cooperation of various muscle groups.

**Adiadochokinesis**

Adiadochokinesis describes the patient’s inability to perform alternating movements.

**Cerebellar Diplegia**

The cerebellar form as an individual entity is rare. Often, spasticity is found along with a cerebellar syndrome. Given the extent of motor involvement, it is classified as a separate form of CP.

This form demonstrates typical symptoms. The hypotonic picture begins to show spasticity in the second half of the first year of life. Slight flexion phenomena are observed and extension phenomena are usually not present. The increased tone begins especially distally, most often in the triceps surae, which later develop contractures. The picture of primitive reflexology lacks or shows a decreased tonic grasp of
the lower extremities. Trunk lateral flexion as a response to unilateral paravertebral skin stimulation is either absent or decreased, thus, the Galant reflex is decreased. Pseudoclonus emerges with the development of distal spasticity.

Motor development depends on the severity of involvement, which varies in this form, similar to spastic diplegia. In an ideal scenario, the children can ambulate at two to three years of age. More severe forms achieve verticalization between the fifth and the tenth year at the latest. Verticalization and bipedal locomotion are not achieved in the most severe forms.

**DYSKINETIC FORM OF CEREBRAL PALSY**

A dyskinetic form of CP is characterized by abnormal movements or postures. The corresponding form of CP is classified based on whether hyperkinesis or dystonia predominate.

In the hyperkinetic form, irregular, repetitive and excessive movements dominate the clinical picture. In CP, these movements are most often divided into two forms:

- **Athetosis** – the most typical; described as writhing, inconsistent, fluctuating and involuntary movements that affect proximal aspect of extremities
- **Chorea** – it is distinguished from athetosis mainly by the speed of involuntary movement; distal aspects of the extremities are primarily affected

A dystonic form is characterized by abnormal changes in muscle tone resulting in typical changes in body posture. The dystonic subgroup also presents with non-volitional movements, but not nearly to such a great extent as the hyperkinetic subgroup. An isometric contraction disturbance is one of the main problems that are characteristic for a dyskinetic syndrome. Therefore, volitional movements recruit muscle groups from the entire body.

An athetoid syndrome can develop from a hypertonic, as well as, a hypotonic syndrome and it is observed in the first three months of life. Most future athetoses develop from neonatal hypotonia. Hypotonia is mainly axial (trunk) and proximal. Trunk hypotonia can be confirmed by the Landau reaction. In the third trimester, an infant’s head and pelvis almost flaccidly hang toward the mat with trunk instability, which is not seen as much in other syndromes. The trunk turns toward the jaw side. The extremities are flexed, however, a sudden opisthotonic posturing can occur with the lower extremities positioned in extension. In contrast to spastic diplegia and hemiplegia, athetoses show more diffuse involvement throughout the entire movement system.

Trunk instability is clearly manifested during axillary suspension. During a traction test, no neck flexion activity is observed during the entire first year. During this test, the lower extremities remain in flexion.

In the first three months of life, dyskineses have yet to manifest themselves. In this period, primitive reflexology and central acoustic deficits can be preliminarily used, which are often associated with dyskinesis. The inability to elicit the acoustic-facial reflex is a warning sign. Disrupted dynamics or the persisting presence of primitive reflexes and a differential diagnosis together predict the severity of the injury.

Persistence and an exaggerated response to the Galant reflex are typical signs of dysfunction. In contrast to spastic forms of involvement, the grasp reflex of the lower extremities is accentuated and persists after the first year of life or even for several years. In contrast, reflexive grasp of the upper extremities is diminished and ceases as early as in the second trimester. The cause is a predominant tendency toward finger extension, especially the thumb and the index. The Moro reflex persits into the third trimester and becomes a stereotypical response to various external stimuli. The heel reflex already cannot be elicited in the second trimester. The suprapubic and crossed extension reflexes become absent in the first trimester. Gait automatism persists for a long time, which is known as stepping (child moves as if to push off). The Babkin reflex persists longer than normal. Lower extremity stretch reflexes are normal or increased.

The first signs of future dyskinesia can already be recognized during the second trimester. Dystonic attacks begin to emerge. Based on external and sometimes also internal stimuli, the infant reacts with a hypertonic change in muscle tone, usually in the pattern of the tonic neck reflex (TNR), the tonic labyrinthine reflex (TLR) or opisthotonus. These are present mainly in athetoses developed from hypertonic stage. In children with hypotonia, dystonic attacks are less pronounced. They are provoked by a sudden change in position. Dystonic attacks also develop as a reaction to sounds. The children struggle with head control. In addition, significant changeability in muscle tone is observed. At rest, the infant is hypotonic and suddenly as a reaction to external stimuli hypertonia accompanied by opisthotonus may occur. When it subsides, hypotonia dominates, especially in the trunk. The severity of hypotonia shows a positive correlation with the severity of involvement. In addition to dystonic attacks, athetosis of the distal lower extremities emerges in the third trimester.

Swallowing poses significant problems. During swallowing, tongue support against the upper palate, which the child is unable to accomplish, is important. This results in malnutrition. Stimulation of the orofacial region elicits dystonic postures. Similarly, every attempt at movement results in grimacing.

Feeding dysfunctions are most pronounced in the third trimester. In the past, many children died during this period.
The children cannot chew and they constantly salivate. Their biting is homologous without lateral co-movement of the lower jaw. In some children, protruding of the tongue occurs long-term in a similar fashion as can be observed in a newborn as a reaction to unpleasant gustatory stimuli. This phenomenon is the so called “tapir’s mouth”. Food that is moved during feeding accentuates tongue protrusion.

The clinical picture also includes autonomic lability (increased perspiration) and emotional instability. Mental abilities are usually normal. Some children show above average intelligence. Scenarios in which mental involvement is present usually include athetosis combined with another type of involvement (cerebellar, spastic, etc.). Postural dysfunctions cause a vocalization deficit and a significant delay in speech development. The patient shows difficulty with pronunciation and articulation. The speech is throaty, explosive and less intelligible. When the child attempts to speak, they breathe in and the entire posture reflects co-movements. Given the normal mental development, the patient’s speech is significantly expressive, meaning that they can express a large content by using few words. The children show decreased facial expressions.

**MIXED TETRAPLEGIA**

Mixed forms of CP include patients in whom more forms of central involvement are combined. Often, for example, spastic diplegia, ataxia and dystonia or spasticity and dyskinetic syndrome are combined. It is usually a diffuse brain injury that also includes significant mental retardation – most often at the level of oligophrenia. This can also include children whose psychological development is not as severely involved. Most of such children can only live in an institution; none of them will be able to live independently in society in the future. The patients show a decreased life span. More than 50% of the children have epileptic seizures that are mostly severe in intensity and are difficult to control by medication. Central dystrophy is typical as a result of a swallowing deficit. The neurological finding corresponds to the type of involvement.

In some cases, amaurosis or mostly divergent alternating strabismus are present.

As far as kinesiologic development, upright posturing shows significant delay. For a long time, it remains at a newborn level.

**ATONIC DIPLEGIA**

Sometimes it is referred to as an atonic-astatic type of CP according to Forster. In this type of CP, the cerebellum is not affected, but the brain’s frontal lobes are involved. Some signs are similar to a cerebellar syndrome, especially significant hypotonia. The scarf, drawing compass and armadillo signs are present. Next to hypotonia, this type of diplegia presents with a more severe degree of mental involvement, usually at the level of oligophrenia. In the first months, the children are considerably apathetic, not interested in their surroundings, do not reach for objects and do not recognize the mother. They react to external stimuli by dystonic attacks. Typically, the postural pattern practically does not include the lower extremities until the third trimester.

In the first year, the child lies with the thighs abducted to 90 degrees. Plagiocephally occurs as a result of being in a supine position. Nystagmus is typically not part of the child’s clinical picture.

The dive reflex is associated with forward flexion of the upper extremities with clasped hands. During axillary suspension administered in the third trimester, the lower extremities remain in flexion and muscle tone increases.

### 1.21.3 Rehabilitation in Cerebral Palsy

#### DESIRED OUTCOME AND COPING PROCESS

**DESIRED OUTCOME**

Cerebral palsy includes patients with varied severity of involvement. Rehabilitation therapy depends on the extent of involvement and the “desired outcome”, which can be expressed by the term treatment expectation. Given the extent of motor and psychological involvement, the children can be divided into several groups and each group needs to be approached in an individual and specific way:

**Patients with a Severe Motor Deficit and Severe Mental Retardation**

Verticalization should not be expected in such patients and, in the majority of cases, sitting is also not accomplished. They are fully dependent on the care from others. The main goals for such patients include prevention of contractures and joint deformities, prevention of pressure ulcers and chest wall deformities that make breathing difficult. Treatment mostly consists of rehabilitation care and prophylactic methods.

**Patients with a Severe Motor Deficit and only a Moderate or Mild Degree of Mental Retardation**

In such patients, it is especially important to begin rehabilitation treatment as soon as possible. Contractures and neurogenically developed deformities pose the main problem. Even with intensive, correctly administered and timely initiated physical therapy, the effects of spastic or hypotonic manifestations cannot
2.5.5 Lower Extremity Orthoses

Considering the weightbearing function of the lower extremity, which fulfills static and dynamic functions, the selection of an appropriate functional orthosis is extremely important for the patient. The following criteria are taken into consideration when selecting an appropriate orthosis:
- Assessment of the extremity’s functional state
- Assessment of the extremity’s weightbearing status
- Range of motion and stability in individual segments
- Muscle strength
- Possible extremity shortening

BASIC CLASSIFICATION OF LOWER EXTREMITY ORTHOSES

FOOT ORTHOSIS – FO
Foot orthoses are applied either when correcting incorrect foot and toe alignment or to decrease the demands of weight bearing (orthopedic insert) (Fig. 2.5.5-1A,B), which can decrease the defect on the plantar surface. In severe deformities, toe correctors are applied (Fig. 2.5.5-2A,B).

ANKLE-FOOT ORTHOSIS – AFO
Ankle orthoses are applied to correct foot and ankle deformities, to stabilize talocrural articulation and to ensure decreased lower extremity weight bearing. The overview of orthoses includes rigid ankle orthoses, those with options to preset range of motion at the TC joint (Fig. 2.5.5-3), ankle elastic reinforcing braces (Fig. 2.5.5-4) and a dorsiflexion-assist orthosis (Fig. 2.5.5-5).

An orthosis with a firm ankle provides maximum immobilization of the ankle and foot complex in all planes. The anterior floor reaction AFO is another type of AFO. The principle of this orthosis lies in the slight ankle plantarflexion, which causes an extension force moment at the knee and increases its stability in the sagittal plane. An unweighting type AFO known as a patellar tendon bearing AFO (utilizing Sarmiento principles) is another modification to an AFO. Its main goal is to decrease the axial loading of the distal segment of the lower extremity during gait. It is used, for example, during functional treatment of fractures or to allow for complete healing of defects on the plantar aspect of the feet. This device, however, can only be used if the skin is intact in the areas the orthosis applies support and if the quadriceps femoris demonstrates sufficient strength to maintain knee stability.

Dynamic AFOs include plastic, metal and also composite fabrication materials. In contrast to stat-
in the actual ankle joint and its range is given by the thickness of the material used for the orthosis fabrication. Dorsiflexion assist AFO, in contrast to the PLS, uses a mechanical ankle joint with a built-in spring mechanism (Fig. 2.5.5-7). This orthosis assists with foot dorsiflexion during the swing phase and ensures smooth transition between the initial heel strike and mid-stance. The plastic AFO with a joint consists of a plastic ankle joint built-in between the foot and the shin portion of the orthosis made from thermoplast. For the orthosis to function effectively, the ankle needs to have at least 5° of dorsiflexion. The degree of plantarflexion can be limited by a stop.

An AFO made from a composite material uses the accumulated energy in an elastic skeleton of the plantar segment of the orthosis during heel strike. These orthoses are designed for active patients (Fig. 2.5.5-8 A,B).

The full effect of AFO wear also depends on the type and condition of the patient’s footwear.

Different heel heights of footwear significantly alter the orthosis’ biomechanical function.

**KNEE ORTHOSIS – KO**

The most simple knee orthoses include infrapatellar straps, knee elastic reinforcing braces and knee orthoses with joint bars. If more rigorous stabilization is needed, orthoses with constant rigid flexion or orthoses with restricted movement are selected. In severe combined instabilities and knee joint deformities, orthoses with firm construction are indicated and the TC joint is used as a secondary joint to stabilize the device. Corrective KO influences the knee alignment based on a three-point principle. Most often they are equipped, similarly to other KOs, by lateral or medial bars with a joint and adjustable straps. These KOs can correct knee varus or valgus, or even a genu recurvatum deformity (Fig. 2.5.5-9 A,B).

**KNEE-ANKLE-FOOT ORTHOSIS – KAFO**

A knee, ankle, and foot orthosis is selected mainly in patients who require stabilization and movement control in the knee and ankle joints.

A KAFO is an orthosis spanning from the patient’s thigh to the foot. This extent allows for function of the
actual AFO and, at the same time, allows for control of the knee joint in the sagittal and frontal planes.

A classic AFO is made from metal bars that connect the knee and ankle joints of the orthosis with leather or Velcro straps for attachment to the extremity. Firmness and durability are their advantage. The durability, however, is achieved in exchange for heavier weight. Another disadvantage of this type of KAFO includes a smaller range of contact surfaces, increased demand on the insole of the footwear and a less appealing cosmetic look.

A plastic KAFO is constructed from a cast of a patient's extremity. This ensures tighter contact with the larger surface area of the extremity thereby decreasing pressure points and increasing movement control of the entire extremity. The individual components of this orthosis are connected by joints. The advantages of this orthosis include low weight and better cosmetic appeal. However, a plastic KAFO is limited by skin condition and extremity size/volume (Fig. 2.5.5-10).

A special KAFO is fabricated from light construction materials (carbon, titanium) based on the patient's individual measurements. Their light weight is the main advantage. The disadvantages include relatively lower contact area in the area of the orthosis arches, which due to possible intolerable pressure points does not allow for correction and stabilization of significant knee joint deformities. Given its light weight, it is used in patients with lower extremity paraparesis (Fig. 2.5.5-11 A,B).

The function of a KAFO depends on the type of joints that are used in its construction. A single axis knee joint allows for unlimited flexion and extension in the sagittal plane. This type of joint is suitable for patients who demonstrate sufficient muscle strength to maintain stability in the stance phase, but, at the same time, demonstrate initial deformity of the knee joint: recurvatum, valgum or varum. Single axis knee joint with a lock will lock the joint in extension and thus, provides the knee with rigid stability in all planes. This type of joint is suitable for patients with a decreased ability to control the knee joint throughout the stance phase, leading especially to sudden knee flexion with gradual loading at the beginning
and during the stance phase. The orthosis’ knee joint can be utilized and locked at various degrees of knee flexion. This is utilized in patients who are unable to fully extend the knee, for example, in knee flexor contractures. Mechanical or microprocessor controlled knee joints in orthoses that affect the stance and swing phases of gait are among the more complex mechanisms used with a KAFO knee joint. This type of joint automatically locks when loaded during the initial contact of the heel with the floor and remains locked during the entire stance phase until it is unlocked at the moment when the heel comes off the floor at the end of stance phase. The patient’s cognitive functions need to be considered when selecting this type of knee joint as the patient needs to understand this mechanism to utilize it.

HIP-KNEE-ANKLE-FOOT ORTHOSIS – HKAFO

The indication for a HKAFO should always be preceded by careful consideration and consultation with the interdisciplinary team. This type of orthosis is a typical example of an uncomfortable device and donning and doffing can be very complicated for the patient and the treating assistant (Fig. 2.5.5-12A,B).

In contrast to the previous orthoses, these orthoses also consist of an elastic or rigid lumbar socket and hip bars with limited range of motion, which ensure stabilization of the hip joints. Some types of Reciprocal Gait Orthoses (RGO) (Fig. 2.5.5-13) are equipped with Bowden cables interconnecting the hip joints of the orthosis. When the center of mass shifts and one hip joint is in a swing phase, extension is elicited in the other hip joint, which is in stance phase. This leads to the elimination of simultaneous flexion in both hip joints and the decreased risk of the “clasp knife” phenomenon during gait. This orthosis is indicated in children with myelomeningocele, in patients with traumatic paraplegia and in patients with muscular dystrophy.

The indication for this orthosis must be preceded by careful examination and assessment of all aspects important for application of this device.

2.5.6 Trunk Orthoses

Application of trunk orthoses is an integral part of treatment of many problems linked to spinal instability following traumas or for the treatment of spinal deformities.

The recommendation for a trunk orthosis in patients with spinal conditions is based on an accurate assessment of spinal instability and the state of the myofascial system. Trunk orthoses are in such cases indicated to stabilize vertebral fractures or as a supplemental device after surgical stabilization.

In patients with spinal pain, soft lumbar belts are often routinely issued, which enhances the patient’s passivity as they use it as a “crutch”.

The treatment of spinal deformities in pediatrics is primarily with custom trunk orthoses. These devices should be indicated only by an experienced physician in clinical centers specializing in the treatment of scoliosis. Equally, the corset fabrication for scoliosis should be performed at a well-established orthotic and prosthetic center with sufficient experience, technical proficiency and trained personnel.

When applying trunk orthoses, a patient’s comorbidity, which can lead to worsening of the patient’s condition, needs to be taken into consideration if simultaneous cardiopulmonary involvement is present.

During orthosis selection, the functional demands need to be considered. It needs to be defined whether the orthosis should provide firm trunk stabilization following traumas, inflammatory spinal conditions, vertebral damage or if it is supposed to serve as a supportive device in painful spinal conditions caused by muscle weakness. The patient needs to be thoroughly educated in the use of a trunk orthosis.

BASIC CLASSIFICATION OF TRUNK ORTHOSES

The terminology involving trunk orthoses is not unified. The terms orthosis, corset, lumbar and abdominal belts are used. When selecting a trunk orthosis, it is recommended to use the international classification of trunk orthoses that includes the location on the trunk and simultaneously describes the construction design (fixation, reclination, flexion, extension, dis-
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### TREATMENT REHABILITATION – DIAGNOSTIC AND THERAPEUTIC APPROACHES

**REHABILITATIVE CARE**

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**Pavel Kolář**

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### A. DIAGNOSTIC PROCEDURES

#### 1 ASSESSMENT APPROACHES FOCUSED ON THE FUNCTION OF THE MOVEMENT SYSTEM

**Pavel Kolář, Karel Lewit, Olga Dyrhonová**

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#### 1.1 NEUROMUSCULAR FUNCTIONS AND THEIR CLINICAL EXAMINATION

**Pavel Kolář**

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### I FUNCTIONAL AND NEUROLOGIC SYMPTOMATOLOGY

#### 1.1.1 Examination of Postural Functions

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