high afinity to the virus (nearly 80 % compared with the leucocytes) is threatened with direct viral invasion, but also an indirect invasion upon the failure of selfdefence immunological mechanisms against other infactions.

6.4.3 Diseases with mixed or unknown etiology

The diseases that belong to this group are those diseases of medical interrest not marely because of the therapeutico – preventive point of view but also due to their scientific research value. Upon the manifestation of nervous system disease the main cause never is an individual isolated cause of that disease. There is always a multifactorial conditioned state. In many diseases the decisive factors remain unknown. Diseases that belong to this group can be futher subdevided into three subgroups: **CNS tumors, neuroimmunological diseases and neurodegenerative diseases** of the CNS.

Tumors of the CNS develop either directly in the nerve tissues (primary tumors), or they originate from other tissues (secondary, metastatic tumors).

Even in case of tumors we suppose the presence of an inborn predispositions, that are activated due to the effect of the outside enviroment and mainly the blastogenic effects. The question of the chemical or viral origin of these tumors remain still unsolved. During childhood as well as during adultdood the most common tumors are **gliomas** (about 88 % of the affected children and 50 % of adults). In children there is commonly **craniopharyngeoma** (4 %) and **teratoma** (3 %). In older people there is often **meningeomas** (20 %), **metastases** (10–20 %) and **tumors of the neural sheeths** (10 %).

The neuroimmunological diseases of the CNS form till now a nonhomogenous group of diseases, that have a common manifestation and immunological basis. **Multiple sclerosis** most probably belongs to this group of diseases (with a high content of gammaglobulins in the cerebrospinal fluid), and **myesthenia gravis** that is a defect in nerve impulse transmission at the neuromuscular junction. Antibodies against the brain tissue were described even in **Alzheimer disease** (a disturbed cholinergic neurotransmission).

The degenerative diseases of the CNS might have many etiopathogenic causes. There causes might be vascular changes, nutritional disturbances, neuroinfection, but demyelinisation might be a secondary change resulting from the destruction of the nervous system. Some **unclear etiology** is for e.g. that of **senile demencia**, degenerative changes of the cerebellum, or progressive subcortical encephalopathy. Compared to the previous diseases **Parkinson syndrome (paralysis agitans**) has got a well known etiology and mainly what concerns the neurotransmission and dopaminergic mechanism in the nigra strata. Neuroinfections as well as poisoning, arterosclerosis and other factors might be triggering factors yet the direct mechanism of the substancia nigra destruction remain unclear.

6.5 Some syndroms occuring upon the injury of the nervous system

To the extrinsic causes of nervous system injury we might commonly add **trauma, accidents, the effect of mechanical power**. Because of the fact that all the medullary nerves are mixed (they contain the afferent sensitive, and efferent motor fibers), upon their injury ther will be a resulted so called **peripheral nerve syndrom**, that commences disturbance of sensation as well as motor disturbances. Upon the destruction of the anterior horn (that contains motor fibers) there will be plegia and upon the destruction of the posterior horn (that contains only the sensitive nerve fibers) the result will be loss of sensation (hyposthesia or anaesthesia, yet if the lesion conserns the afferent fibers in the affected horns, there might be hyperesthesia).

Upon **the lesion of spinal cord or brain** the condition is more complicated by **bleeding** (haemorhage), an **increased pressure** in the closed cranial system or the closed spinal canal, **edema** and other lesions (degenerative changes) of the nerve tissue. The neurological finding is characterized according to the level and the localisation of the CNS injury: we devide them into **spinal syndroms** (for e.g. the transversal separation of the spinal cord syndrom – with motility and sensation loss acording to the level of lesion,

the period of spinal shock with the gradual development of the spinal automatism), **the partial separation of the spinal cord syndrom** also known as **the Brown – Sequard syndrom** is muscular plegia at the affected side with the loss of all kinds of sensation except temperature, cold, and pain that are loss on the same side of the lesion due to the decussation of tracts.

Upon **the longitudional spinal cord injury (bulbar syndroms) and pons (pontine syndroms)** the nuclei of the cranial nerves together with the pyramidal tract above its decusation are often affected (**crossed hemiparesis**). It is manifested for e.g. by unilateral paralysis of the facial nerve, and a contralateral paralysis of the body. Upon the injury of important regulatory centers (and mainly the respiratory center) sudden death might occur (usually due to respiratory center paralysis). This might occur up medullary compression and fracture of the atlas – the second cervical vertebrae.

Upon **midbrain injury** it is important to remind you that the midbrain is the outlet of the oculomotor and trochlear nerves. There are also the pyramidal tract and the sensory tracts here. That is why upon the lesions of midbrain we most of all deal with the oculomotor reflex mechanisms (accomodation, anisocoriosis, loss of light reflex, pathological nystagmus) and hemiparesis and hemianaesthesia of the contralateral side of the body. More extensive lesion might result in decerebration rigidity.

Cerebellar syndroms are manifested by cerebellar disfunction – i.e. loss of the coordination of **automatic movements**. Dysfunction of paleocerebellum (vermis) are manifested by static dysfunction and automatic synkinesis – the coordination between the muscles of true an the lower limbs during walking and standing (the patient stands and walks on wide base, then it looks as if under the effect of alcohol). Cerebellar disfunctions are manifested mainly by disturbance of limb movement and disturbance of language. **The movenments become hypermetric and dysmetric**, the patients finger either misses the point or hits it strongly.There is no tremor at rest, yet it appears upon movement (so called **intentional tremor**). The landmark is **the scanded language** and **a shaky handwriting**.

The thalamus is considered as a kind of conducting station of the sensory tracts that are directed toward the cerebral cortex. It is an important subcortical center for motor and vegetative automatisms. It envolves the mechanism of sleep, attention, and emotions.

Hypothalamus is an important leading and coordination vegetative center. It is closery connected to the neuroendocrine diencephalo-hypophysal function, blood pressure, thermoregulation mechanisms are quite complex and hence their topographic diagnosis might be very difficult.

Between the thalamus and corpus striatum there is a strip of white mater, that is known as **the internal capsule**. Most of the ascending and descending tracts pass through it and they join the cerebral cortex with the spinal cord, the cerebellum, and the reticular formation. The intermal capsule is commonly affected with **cerebral haemorhage**. In case of a unilateral lesion there will be a prominent hemiparesis or hemiplegia (paralysis of half of the body) on the contralateral side of the lesion. The paralysis is so called **central type of paralysis**. Sometimes there are associated parasthesia, dysesthesia or even pain on the affected side (the ipsilateral side).

Lesion of the diencephalon is rarely localized in a small area. The cause is usually haemorhage. We can differentiate the hypotonic-hyperkinetic **neostriatal syndrom** (1) (inflamatory disease during childhood and the so called **minor chorea** – that is manifested with a prominant decrease of the muscle tonus and motor hypermetria), and hypertonic-hypokinetic **paleostriatal syndrom** (2) – the incidence together with the extrapyramidal system disturbance occurs with some encephalitis, and is manifested by disturbances of sleep (**somnolence**), **rigidity** (that is characterized by the fact that the patient stays in the given position). **The tremor** is resting tremor, and **the hypomimia** is typical.

Parkinson disease is an individual degenerative disease of the paleostriatum (see the degenerative diseases). Another one is **athetosis** (3), that mainly occurs as a result of the prenatal injury of the motor centers in the diencephalon. There will be a combined lesion of the paleostriatum and neostriatum, that is manifested by rigidity combined with hyperkinesis which is known as the athetotic type (a slow spasmatic tortuous involuntary movements that appear upon every intention to perform an active movement). Typical are the spasmatic expressions of the face. **The hypothalamic syndrom** (4) also belongs to this group. It is manifested by a prominant distur-

bance of the vegetative function. Many times we deal with an extensive inflamatory, vascular, or tumor injury, and that is why the symptoms are combined with other signs.

Syndroms that occur due to prosencephalon (forebrain) injury are usually manifested as cortical injury, that is very sensitive area,and that also contain the finest differentiated functions. **Injury to the occipital area** (1) is manifested at the beginning as disturbance of vision (**cortical blindeness**). Upon **parietal lobe injury** (2) there will be some localized sensory disturbance but also some gnostic changes i.e. inability to recognize objects upon touching and holding (apraxia – inability to recognize the shape of the subject upon touching it). During **frontal lobe lesion** (3) there will be some motor changes as well as behavioural and intelectual disturbance. Localized lesions of the pyramidal area are manifested by a precisely localized paralysis. More extensive damage might be manifested by hemiparesis. An epileptic locus in this area can result in the so called **Jackson epilepsy**. Disturbances of hearing and speech occur in case of **temporal lobe lesion** (4). When the white mater is affected by the lesion as well (tructs from the thalamus to the occipital lobe), there will also be visual disturbances. **Disturbances of speech** (5) belong to this group of injuries as well. The speech is a complex mechanism during which the integration mechanism of the whole extent of cortex takes place. The function of speech is devided into **sensory component** (hearing distinguishing the spoken word, vision – distinguishing the written word) and into **a motor or expressive component** that is concerned with thought arrangement into words and sentences and the preparation and realization of the motor plan during articulation or writing. In cases of **the posterior temporal lobe lesion (Wernick area**) there will be sensory aphasia – being the inability to understand the written or the spoken word. Whereas in lesions of the Brocad's area of the frontal lobe there will be motor aphasia, that means the inability to express with a speaking language. In cases of dysarthria the patient is able to create words and sentences, but cannot realize them due to the bad coordination of the articular muscles. These centers are located in the left hemisphere (the dominant hemisphere) in the right handed people.

6.6 Head injuries

Head injuries differ according to the mechanism by which the head injury occured and its extension. There are two main mechanisms that can be characterised as follows: **closed-head trauma (blunt trauma, e.g. box) and open-head trauma (penetrating trauma)**. In both categories neural tissues are damaged by compression that pushes the tissues together, tension that pulls or exerts traction on the tissues, shearing that slides tissues onto other tissues, or a combination of forces. With open-head trauma, tissues are directly damaged.

Direct strike or blow. That is usually causing injury to the soft tissue of the head, skull fructure with the consequent contusion and brain tissue laceration in the area of injury. Soft tissue injury is not dangerous despite the often occuring haemorhage. Skull fructure that is associated with the invagination of bone fructures into the brain with haemorhage and brain tissue injury is very serious condition. This condition requires a non postponed (immediate) neurosurgical approach.

Injury caused by accelaration, deceleration and rotation. Due to the fact that the head is relatively freely mobile and the brain is also relatively loosely situated in the cranial cavily, during some sudden movements and acording to the action-reaction low, acceleration-deceleration low, or possibly the rorational movement of the brain there might be some damage occuring by the bony cover and respectively by the hard covering layers, that separate the anterior and the posterior part of the brain (gyri are damaged, whereas the sulci are relatively protected).

The clinival manifestation of this condition is **concussion**, that is manifested by a transitional loss of consciousness that result due to the disfunction of the temporal neurones. The injury is not permanent and there will usually be a complete renewal of all the functions. In all types of head injury we have to know the mechanism of the injury and the possible brain destruction. For e.g. the so known **a contracoup injury** that occurs when the contusion of the brain tissue, is on the opposite site od the original strike (a coup injury – on the site of impact, where the skull hits the brain).