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).

6.6.1 Brain tissue injury

The brain is totally protected against the mechanical effects of the external enviroment because it is situated in the cranial cavity and covered by the brain meningi and the connective collgen stripes on which the brain lies and the cerebrospinal fluid by which it is enclosed. Despite all these facts the traumatic injuries of the brain and spine are quite common (e.g. box).

Generally those injuries are devided into **open and closed brain injuries**. The closed brain injury can be furtherly subclassified into many types such as **commotio cerebri, contusio cerebri, and compressio cerebri**. The injuries is most commonly caused by **a pressure wave**. Even an extensive shift of the brain in the cranial cavity (acceleration) can lead to brain tissue destruction as well as loss of function.

In case of **open injuries** there will be a communication between the extracranial and the intracranial space, the site of the injury will bleed (**haematoma, ischemia, and edema**, and the consequent – **infammation**).

When thinking about the mechanogenesis of brain injury we have to think about the traumatic destruction of the cervicocranial junction. **Injury of the cervical spine** is usually primary and it even determines the clinical symptomatology. The consequent brain damage (commotion or contusion) can be associated with the primary injury of the cervical spine – as action and reaction there will be a sudden and prominant hyperflexion with a consequent hyperextension (or on the contrary according to the direction of the pressure wave effect). It is expected that in some sport injuries this mechanism is of a marked importance.

When dealing with the characteristic changes on the cellular level in case of brain injuries we have to keep in mind that the brain cells can not devide, and hence their number is constant, and can not be renewed by substitution with new functional neurones. Brain injury can also be characterized as an interferance with the nervous system integrity, interferance with tissue organisation, and their subcellular share as a result of aplication of external power. The outcomes of the injury are determined by the type of injury, its localisation and extension. What is important for the patient are the associated complications such as (associated injuries, infections, inflammations, edema and etc.) that accompany the primary injury. The difference can be in the number of cells, that are damaged or reversibly damaged.

The faith of the reversibly damaged cells is determined by **the nutritional supply**, and mainly **oxygen supply**. The oxygen supply is the limiting factor of regeneration, that starts at the moment of injury occurence even with those injuries that lead to an acute disturbance or even stop the oxygen supply to the cells (for e.g. vasospasm), the changes occur gradually with increasing intensity with time. In tissues there is a certain oxygen reserve that is bound to the haemoglobin, and namely soluble Hb in the tissue fluid. The following changes are those of the slowing or stop ATP synthesis, the Ca^{2+} transport disturbances into the mitochondria and Na^{+} out from the mitochondria. This condition is accompanied with the drop of the ATP, and an increase of the AMP $+ PO_4^{3-}$ on the cytoplasm. Ischemia is characterised by the continuously increasing lactate concentration. The reversibility after cellular injury is associated with the fact whether the oxygen supply is renewed so that there will be renewal of the ATP production. If this doesnd't hapen, there will be worsening of the cytoplasmic pH as a a consequence of the mentioned changes and this will have its negative effect on glycolysis and homeostasis of the internal cellular enviroment. The permeability of the cellular membranes will change, and the passive movement of the ions and water across these membranes is increased. Mitochondria are the most commonly affected structures (the loss of Ca^{2+}, the condensation of matrix, and the drop or ceasing of ATP production, Na^{+} and water cummulation in the mitochondria, the drop of pH, and a partial protein denaturation), endoplasmatic reticulum (a higher permeability, dilatation and the block of proteosynthesis) and the cytoplasmic membrane (an increasing permeability). These changes are still reversible. The persistence of those conditions for a certain critical time that differs from tissue to another, can cause reversible changes or irreversible ones. The irreversibility is manifested by the inability to keep the homeostatic mechanisms, the loss of free energy production, and the dominance of the catabolic events, that are no longer connected to the structure, and further affection of the ion transport between the intra and extracellular spaces, till their complete similarly in concentration on both sides of the membrane. The irreversible changes continue to damage the cellular structures

and consequently cause its (necrosis). As an example to demonstrate some of the described changes we will talk about the cerebral commotion.

6.6.1.1 Closed-head trauma

Concussion is an acute, most commonly traumatic globally conditioned, yet short lasting and full reversible disturbance of the cerebral functions.

The basic symptom is **loss of consciousness**, that can last for few seconds to hours. In the clinical classification of the cerebral commotion we evaluate the time of lost consciousness and the length of **the posttraumatic amnesia** (a postconcussive syndrome). The changes of consciousness are in direct relation or proportional with brain structure injuries. The patientd̄s condition yet, is influenced by other accompanied symptoms of the trauma, such as shock, tiredness, pain and others.

Concussion is not marely **a functional** or **a pathoanatomical unit**. One of the first trials to explain the functional changes during the cerebral commotion is **the Monakow's asynapse theory**, that explains the transitional changes due to reversible biochemical changes at the level of the synaptic junctions (their dysjunction). Despite the fact that many theories about **the synaptic junctional blocks** caused by an increased release of acetylcholine are overcommed, this theory is becoming important once again. It seems that the midbrain and its neurotransmitters play an important role in the pathogenesis of cerebral commotion. The disturbance of noradrenaline and acetylcholine in the reticular formation of this area causes some disorders of the postural activity and can be the cause of multiple neurovegetative signs that accompany cerebral commotion or are its result. It seems that some authors often talk about the physico-chemical changes in the brain cells that can be either directly associated with the effect of the pressure wave, that moves very fast via the brain tissue, or even with the accompanied neurovegetative changes. It's mainly dealing with vasospasms, that result in a transient ischemia of many parts of the brain. The mechanism of these changes causes the reversible type of neuron changes as mentioned above.

The mechanical pressure wave that passes via the cerebral tissues affects all its structures with either major or minor outcomes. That is why the actual mechanism of the injury is quite complex. Upon a hit on the head (a vertical force on the head) the main parts that are affected are the cortical areas of the cerebellum and the corpus callosum (a strike on the tentorium and falx cerebri), on the brain base the affected parts are the basal parts of the frontal and temporal lobes, and even the cranial nerves and vesseles. Upon an anterio-posterior blow there will be a horizontal shift of the intracranial masses, that leads to frontal lobes **contusion and haemorhage** can occur due to the injury of **the connecting vesseles**. In the posterio-anterior direction the frontal lobes, the temporal lobes, and the brain sterm are the endangered areas. The impact of the liquor wave damages the base of the 3rd ventricle. During these impacts the opposite sides of the brain might be injured due to oscillation (**contre coup mechanism**). Upon the rotational shifts of brain apart from the blasts on the bony cover there might be tissue injury due to torsion. Some tiny tears will appear on the borders between the grey and white mater with a multiple vascular damage and the possibility of intracerebral haemorhage. The notes about the structural basis of consciousness is incomplete up to date, and that is why even the explanation of loss of consciousness upon the cerebral contusion is not unified. We have to keep in mind, that the complete pathomechanism is yet unknown. We know that different brain structures have different sensitivity. Upon the cerebral contusion the mechanical tension affects the brain in the centripetal direction. With the increasing intensity of injury the damage will include only **the superficial brain structures (a mild degree**) affects even **the deeper structures of the brain (a moderate and a severe damage**). It is very likely that the most common cases of cerebral commotions (the typical) with loss of consciousness are upon involving the ascending reticular activation system the reticular formation (ARAS) of the brain stem. In cases of severe disturbances even the diencephalo-mezencephalic area is affected by the damage. On the contrary there are some known cases of the so called **concussion on feet**, that is characterized by only a mild alteration of consciousness, with no memory disorders or loss of motor control. Here we expect that the trauma didn't involve the ARAS.

In cases of cerebral commotion we are dealing with **a functional disorder**, without a clear pathologico-anatomical or histoloical changes. The injury of the membrane functions is **reversible**. Upon recur-

rent cerebral commotions (for e.g. boxers) there will be some qualitative changes in the pyramidal cells (chromatolysis, prolongation up to axonal tearing). Those mentioned changes are **irreversible** and their compensation is possible only thank to the plasticity of the nervous system as whole.

The extent and the localisation of the injury upon the cerebral commotion is found out with the help of the electroencephalography recording (EEG) and of the cerebral evoked potentials (EVP). Changes such as reduction and slowing of the bioelectrical activity in the cortical and subcortical structures, that were found with the help of implanted electrodes experimentally, prove the presence of a relationship between the pressure wave and the membrane structures of the brain. In the pathogenesis of cerebral commotion the changes of the vascular system and hence the oxygen supply of blood to the brain during the trauma play an important role. The angiography showed that within the first minutes after the injury there is a prominant **decline of blood flow** via the brain and oedema occurs (the increase of brain volume by about 3 %). The generation of brain oedema is related to the higher permeability of the cerebral vasculature and hence the disturbed blood flow regulation as a consequence of the injury of the brain worsens during the initial minutes. These changes usually return to normal spontaneously in the next stages.

Many authors pay an attention even to **the neuroendocrine changes** (dysregulation) upon the cerebral commotion. An overcommed traumatic injury together with cerebral commotion represent a stress for the organism. Due to the affection of hypothalamo-hypophysal system by the injurious mechanism (pressure wave, changes of vascularisation, liquor wave) there will be a disturbance of the adrenocortical balance, the concentration of biologically active substanes in the blood, the Na^+ proportion (hypernatriemia) and K^+ ions (hypokaliemia) due to the renal tubular changes, because the kidney is actually an excretory organ of the neurohumoral (hypothalamo-hypophyso-adrenal) system.

The clinical picture of the cerebral commotion is manifested with **loss of consciousness** (a mild degree till 15 min, a moderate degree till 60 min, and a severe degree of more than 60 min lost consciousness). In case of the severe states we shoud keep in mind the possibility of **cerebral tissue contusion**. Loss of consciousness is associated with a retrograde amnesia (the patient doesnd't remember the situation shortly before the injury) or sometimes an anterograde amnesia (where the patient doesnd't remember situations shortly after or during the gain of consciousness). The accompaning signs of the cerebral commotion are some vegetative changes such as – nauzea, vomiting, dizziness, giddiness, and headache. In worse conditions there will be the so called hypothalamic signs: disorders of miction (oliguria, polyuria) salivation, sweating, disorders of blood glucose regulation (abnormal oral glucose tolerance test, sleep disturbances, leucocytosis and others). An important sign is **the blood pressure change, the orthostatic hypotension and orthostatic tachycardia**. In the period of reconvalescence there might usually be some sleep changes, inability to concentrate, headche, and dizziness the so called **postcommotion syndrom**). The cerebral commotion can be the cause of the manifestation of a latent brain disease, it can worsen the already present disease, it might accelerate the process of aging, the process of cerebral atherosclerosis, worsen Parkinson disease, worsen some psychological disease or even expose some latent brain tumor etc.

6.6.1.2 The following complication

Apart from the direct outcomes of head injury there might commonly be some complications that occur after few hours or days:

1. **haemorhage**
2. **brain edema**
3. **leak of the cerebrospinal fluid**
4. **infection**

1. Bleeding (haemorhage)

(a) **Extradural haematoma**. This type classically occurs as a complication of the linear skull fractures, when a meningeal artery is injured. In the typical case and as a result of haemorhage the dura mater is unsloughed from the bone and it compresses the brain tissue, so there will be a haematoma. According to the seriosity of bleeding will be the clinical manifestation. The lucid phase can last for few hours, gradually there will

be signs of intracranial hypertension and different neurological symptoms that bring the patient to the neurologist.

(b) **Subdural haematoma**. Can ocur in any location, is extensive because dura mater and the arachnoidea are not tightly adherent. It occurs most commonly due to the rupture of small bridging veins.

(c) **Intracerebral haemorhage** usually occurs together with the cortical contusion – most commonly in the temporal or the frontal region due to the effect of the injurious mechanism. Yet the haemorhage during the trauma might be deep in the tissues. The cause is vascular injury that is usually not very extensive.

2. **Brain edema** (*is described elsewhere 3.7*)

3. **Leak of the cerebrospinal fluid** (CSF) and blood from the nose and ear can be a complication of cranial base fructure. It can be prolonged and it creates an entery for infection.

4. **Infection**. Might complicate fructure healing and it can affect other structures e.g. meninges.

6.6.1.3 Late complications

1. **Scar formation and epilepsy**. During the process of scarring by the resorption of necrotic tissue degradation products, there will be formation of pigment scars. These can potentiate the occurance of epileptic firing, mainly when the meniges adhere to the brain tissue.

2. **Chronic subdural haematoma**. Occurs mainly in the elderly patients and alcoholic patients. A thick layer of fluid and a partially coagulated blood gradually cummulates between the thickened dura mater and the arachnoidea. Signs of brain atrophy and compression appear in the adherent area. The pathomechanism is not clearly understood. We assume that, there will be damage of the small connecting bridging veins, which together with the lower blood coagulability can be the causative factor of this type of bleeding. The clinical manifestation usually includes a marked beginning with the progression of neurological symptomatology. The history doesnďt include any serious head injury in most of the cases, it is not uncommon to find only a benign head injury (for e.g. getting into or out of a car).

6.7 Brain edema

Edema (a swelling) of the brain can be characterized as **an increasing fluid content** in the brain tissue. Brain edema results in an increased extracellular (more in the white mater) or an intracellular (more in the grey mater) volume, increases the intracranial pressure, worsens the course of the primary disease. It occurs in many brain diseases. This process might be localized or generalized according to the causative agent. An example of **the localized edema** can be **brain infarction**, local **brain ischemia, haematoma or tumor**. An example of **a generalized brain oedema** can be that caused by **intoxication, metabolic disorders, hypoglycemia, generalized hypoxia** and a severe **head injury or malignant hypertension**. Brain edema can occur in association with the disequilibration syndrom that occur with **dialysis, diabetic ketoacidosis, hypoosmolarity**, in different forms of **obstructive hydrocephalus** and upon **hepatic dysfunction**.

The outcome of brain edema, either the localized or the generalized type, is, worsening of brain perfusion, a disturbance of cerebral haemodynamics, and metabolism disturbances with the consequent loss of consciousness. The pathomechanism is given by the characteristics of the initial insult. In the initial stages the most common mechanism applied is the so called **cytotoxic component**, that leads to cellular membrane injury, disturbance of ion balance of the membrane (Na^+,K^+) and the shift of water from the plasma to the interstitium. The major components of this phase is hence the shift of water into the interstitium based on the ionic imbalance that results from cellular membrane injury. In the later stages there will be the so known vasogenic component, that is manifested by capillary wall damage, and the shift of plasma proteins to the interstitium with the consequent oncotic water binding (shift of water to the interstitium). The edematous fluid has the tendency to spread in the white mater. From the