eas it enters into the cranial cavity and forms the falx cerebri, tentorium cerebelli and those being a rigid structures might be the cause of cerebral injury in case of its displacement during (accidents, intracranial hypertension, herniation). What is known as the extradural lesions are commonly localized between dura matter and the bone – dura mater is sloughed or separated from the bone. Subdural lesions are also common. They might spread very easily to the peripheries, because the arachnoid membrane and dura mater are attached to each other quite freely (only slightly attached). **The arachnoid membrane** is a very smooth cover from which tiny trabeculae arise via the subarachnoid space to **the pia mater**. The subarachnoidal space is composed of blood vessels and cerebrospinal fluid. Diseases that affect this space, do spread very quickly to the peripheries, above the whole brain and spinal cord surface. Pia mater prevents the penetration of infection to the brain tissues. Pia mater is closely attached to the cerebral surface and the surface of the spinal cord and it copies its curves and invaginations and it invaginates along with the cerebral blood vessels to the brain tissue. All the mentioned covers – dura mater, arachnoidea and pia mater play the role of a true barrier (**the blood-brain barrier**), that separates cerebrospinal fluid and blood from the brain tissue. It is important to remember that although the cerebrospinal fluid has a very similar composition like the extra cellular fluid of the brain, the changes of the cerebrospinal fluid cause only a minor indirect effect on the brain tissues in different CNS diseases.

The blood-brain barrier at the level of pia mater is formed of astrocyte dendrites. This barrier is made of three layers:

1. On the brain surface
2. Along the penetrating vessels to the depth of the brain tissue. The space remaining between the vascular wall and pia mater is called Virchowd's space and it is analogous with the subarachnoid space.
3. At the level of capillaries pia mater does not exist any more. And here the astrocyte dendrites together with the capillary endothelium and the basal membrane form the true specialized selective blood-brain **(hematoencephalic) barrier**.

Pia mater actually separates two CNS compartments – the external and the internal, that have a different embryonic origin. These are most of all tissues of neuroectodermal origin, i.e. **the CNS tissue** itself that constitutes of **neurons, and neuroglia** (ependymal cells, astroglia, oligodendroglia, microglia). These tissues are characterized by a highly specialized functions, they are hence vulnerable, sensitive, to many injuries and degenerative processes.

Another group of tissues is mesodermal in origin. Many kinds of tissues belong to this group for e.g. blood vessels, brain coverings (meninges) and macrophages. The macrophages reach the brain tissue during the embryonal development and they change to microglia. These tissues have some similar characteristics to any other tissue anywhere in the organism and they are affected by for e.g. inflammatory diseases.

6.2 Neuronal injury

Neurons are very sensitive to many external and internal unfavorable effects. We are mainly talking about **anoxia, hypoglycemia, viral infections, metabolic disorders, vitamin deficiency** (for e.g. vitamine B) and so on. The resulting effects of these factors on the nervous system mainly depends on the degree of injury of the throphic function of the neuron and the maintenance of its integrity. The decisive factor is the site of injury, type of the nerve cell, but other factors also have a great value (for e.g. the degree of cellular differentiation, the relation to glial cells etc.). During some physical or chemical effects there might be either **reversible short lasting injury** or **an irreversible neuronal injury**. Neuronal injury might be of many degrees:

1. Functional injury caused for e.g. by pressure (hypoxia, that is reversible with the following normalization.
2. Death of an axon without interruption to the endoneural tubes.
3. Axonal death with interruption of the endoneural tubes.

4. Interruption of the nerve fibers.

5. Interruption of the nerve cords.

The regeneration of peripheral nerves is possible, as long as the interruption is not very long lasting and as long as the endoneural sheath remains intact. Upon its interruption we might need a surgical repair to enable the regeneration of the nerve fibers to grow within the repaired endoneural sheath.**Regeneration is a long lasting process** (according to nerve length) it might last few months. Upon an interruption of motor fiber and its muscular ending, the skeletal muscle will atrophy and its sensitivity towards a certain neurotransmitter will increase. We are talking about a denervation hypersensitivity that is explained by the activation of a larger number of receptors compared with the number of activated receptors before denervation (acetylcholine), or the disturbance of the back trapping (uptake) of the transmitter to the pre synaptic area (noradrenalin).

When we talked about **the primary degeneration of neurons**, we have to mention, **that neurons might also be injured secondary**:

1. The retrograde degeneration – upon axonal injury there will be first of atrophy of the distal part of the axon and later on there will be degeneration of the neuron itself.

2. The transsynaptic degeneration process, is that process where the injured neuron is connected to a neuronal network via the help of synapses and hence it can spread to the other connected neurons and lead to their degeneration.

What concerns the nerve cell bodies themselves – neurons – there basically might be what is known as **fast necrosis** that is associated with acute functional disturbances of the neuron, or **slow atrophy**, that is associated with gradual loss of neuronal function. A specific type of this slow (cumulative) atrophy and reduction of neurons is represented by the effect of age on the CNS (mainly during senile age). The symptom of these might be for e.g. **dementia, disturbance of memory, disturbance of some other functions of the higher nervous function (senile dementia)**. If this process of slow atrophy is activated during early age, we are talking about **presenile dementia**.In these case we have to realize that there is no way for the atrophied neurons to regenerate. **Neurogenesis** in man was not yet proven, even though some signs of regeneration were noticed in the dentale gyrus of the hipocampus.

The ability of the nervous system to become accommodated to the variable effects – even the pathological – for e.g. **the compensation** of many dysfunction is related to the previously mentioned CNS plasticity. Many mechanisms take place in this process:

1. **Redundancy** (the ability of other centers to compensate the function of the injured neuron).

2. **Alternation** (the ability of other centers to take over the function of the injured area).

3. **Vicariing functions** (other physiological mechanisms that make up for the disturbed physiological function – for e.g. the loss of vision is partially compensated by a more adequate touching sensation or hearing sensation.

4. **Dischiasis** (the functional depression of a certain center as a result of its lower or on the contrary extraordinary higher stimulation by the injured part, the function is then normalized, if the stimulation activity is normalized).

In relation to the mentioned facts we might draw the attention to the fact that during certain life periods the compensatory abilities of the nervous system are markedly variable. As a rule, in the early postnatal period and **in the early childhood the plasticity is the highest** and it decreases with age. Some factors are very important such as the nutritional factors, the composition of diet, but even the qualitative factors such as the effect of environment, the social contact and the factors that enable the communication of the member with his environment (for e.g. an appropriate function of the sensory organs). These have very effective effects on the formation of the highest function of the nervous system in the areas of the highest neuronal function.

On the other hand it is necessary to point out to the fact that the largest group of psychiatric patients do not suffer any morphological changes of the CNS, brain cells, neurons, that might spot the light (and explain) the cause of the illness. Attention in this case is drawn to the subcellular level, eventually to the brain biochemistry.