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. vitamin B) and so on. The resulting effects of these factors on the nervous system mainly depends on the degree of injury of the trophic 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 presynaptic 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 dentate gyrus of the hippocampus.

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. Vicariating 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.