develop, some signs and symptoms due to local dysfunction of some brain centers or pathways might appear. Apart from this there will be some general signs (headache, vomiting, blurred vision). When the frontal lobe is involved by a tumor process there will be some psychological changes, if the parietal lobe is affected there will be epileptic seizures together with the psychological changes. Attacks of spasm and paralysis on the contralateral side occur when there is a tumor in the temporal lobe. When the occipital lobe is affected, some visual disturbances will appear (scotomes in the visual field.) Expansive processes in the posterior cranial cavity is manifested with cerebellar and cranial nerves dysfunction. Some characteristic signs and symptoms accompany hypophysial tumors, craniopharyngeomas, and vestibular nerve neurinomes that could be diagnosed by the charecteristic bony changes. Apart from the histological classification of tumors into benign and malignant we have to think about some **malignant tumors due to their position** when dealing with brain tumors.

Tumors of the spinal cord can arise directly from the spinal tissue. They either grow inside the spinal cord (**intra medullary tumors**) or they grow from the spinal nerves or spinal cord coverings (**extra medullary tumors**), or they might arise from the vertebral column (**extra dural tumors**). The extra medullary and extradural tumors compress the spine from outside. **Metastatic tumors** (most commonly in ca lung, ca prostate, renal carcinoma, tumors of the thyroid gland, and the ca brest) usually grow from the outside into the spinal coverings (extradurally) . Tumors of the spine are manifested by backache that commonly radiate along the spinal nerves and their enervation sites. Spinal compression will reveal some motor disturbance of the limbs (paraparesis paraplegia, quadriparesis, or quadriplegia), disturbances of sensation and disturbances of sphincter function. Benign tumors of the spine appear on x-ray due to their pressure changes, while the malignant tumors show distructive changes.

6.20 Injury of the spine

The primary pathological process plays role in the anatomical and functional outcomes of the spinal injury. There is no space to compensate the pressure or volume changes and this has got a very serious outcomes. An example of this might be fructure or dislocation of spinal vertebra, a tumor (usually secondary) that expands into the the spinal canal, disc prolapse, or meningeal tumor that causes compression of the nervous tissue.

The spinal cord is very similar to the brain tissue, the anterior and posterior roots as well as the vesseles are freely situated in the watery enviroment of the cerebrospinal fluid. Upon compression there will be some injury to the nervous and neuronal pathways and the extension of this injury depends on the seriousness and time length of the compression. The injury might be in the mild cases only focal, yet in the complicated cases there might be **a transverse lesion of the spinal cord** – a complete discontinuation of the spinal cord. The vascular compression leads to tissue infarction, and there might be an extensive injury to the nervous tracts and pathways with the consequent loss of function. This is commonly a complication of mechanical injury. Upon compression there will be some traction of tissue and coverings that can cause a secondary injury to the spinal roots for example. This might result in **radicutilis and spondylosis**. At the level of the lesion connections between the sensory and the motor fibers will be discontinued, those connections form the spinal reflex arch. According to the extent of injury there might even be a complete discontinuation of the longitudinal spinal tracts and hence disturbance in the regulatory effect of the brain on the regions below the lesion. There will be mascular paralysis and a loos of sensation below the lesion. Spinal reflexes below the lesion are not affected.

6.20.1 Ascending and descending degeneration

Long axons that are disconnected with their bodies will undergo progressive degeneration. Upon a

transversal lesion of the spine all the long tracts degenerate upwards (sensory tracts) and dawnwards (motor tracts). The most common example of **a descending degeneration** is the condition following brain infarction in the area of internal capsule. The degeneration spreads from the lesion along the corticospinal axons till their terminals in the anterior horns of the spine. After a period of time we might register this degeneration in the form of demyelination along the whole corticospinal tract. Those changes actually involve the whole pyramidal system. After a long time there will be gliosis and scarring of the tissue.

6.20.2 Injury of the motorpathways

The division of motor neurons and pathways into **the upper motor neuron** (UMN) and **the lower motor neuron** (LMN) based on anatomical and functional characteristics has got a great clinical and diagnostic value.

The upper motor neuron is formed of the cortical motor neurones (the precentral gyrus), the motor pathways in the brain that (pass through the internal capsule), motor pathways in the brain stem (where decussation of tracts on to the contralateral side take place) to meet the cranial nerves of the contralateral side. This crossing takes place in the pyramidal decussation, where the anterior corticospinal tract (non crossed) is separated from the pyramidal tract (being the crossed part of the motor pathway).

The lower motor neuron is composed of the carnial nerve nuclei (the motor nuclei) and their axons in the carnial nerve fibers, as well as the anterior horn cells with their axons in the spinal nerves. It is important to realize that during the long course of the motor neuron from the cerebral cortex till the anterior horn of the spine represent the upper motor neuron and this could be injured by multiple disease processes. The lower motor neuron can be injured in the spine, as well as in the peripheral nerves.

The most common cause of injury to the UMN is hemorhage in the area of internal capsule, different levels of this pathway could be involved in cases of multiple sclerosis or in case of other diseases that cause demyelination of the motor from the cortex till the spinal cord and its segments.

A typical example for LMN injury is poliomyelitis that affects the motor neurons in the anterior horn of the spine, or a peripheral neuropathy that destroys axons and their coverings and hence results in disturbances of transmission.

In both cases i.e. upper motor neuron and lower motor neuron lesion (UMNL and LMNL) the clinical picture will reveal paralysis, yet there are some important changes that differ in their quality and spectrum of the present reflexes in both the mentioned cases. When the lesion is in the upper motor neuron (central paralysis) the lower motor neurons will escape the control of the higher centers so the muscle tonus will increase, the tendon and other spinal reflexes are increased, and the extensory (Barbinsky) reflex will appear. This is why we call this type of paralysis the spastic type. Following a lower motor neuron lesion there is actually an error in the connection to the effector, so the reflexes are absent, and there will be muscular atrophy. This type is known as **the hypotonic paralysis**.

6.21 Diseases of the motor neuron

Are usually of unknown etiology. They appear more frequently in adult patients and mainly in men. The basic lesion is a progressive degeneration of the cortical and the spinal motor neuron, that will be manifested by their dysfunction. The variability of symptoms depends on the ratio of affected upper motor neuron to the affection of the lower motor neuron, and on the site of the clinicial lesion. The evaluation of this ratio is possible in the initial stages of the disease when the changes are bound to a certain localization, but later on there will be a diffused affection of tissue and here the differentation becomes much harder.

A progressive muscular atrophy is the main sign of neuronal atrophy (mostly due to lower motor neuron injury) due to the degeneration of neurons in the anterior horn. This degeneration worsens within many years, and the cells will become necrotic. Signs and symptoms of the initial stages appear within (1–4) years and are manifested as the affection of fine movement of the fingers, their fasciculation, later on the muscles of the hand will be affected, and at last