develop, some signs and symptoms due to local dys-
function 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 to-
gether with the psychological changes. Attacks of
spasm and paralysis on the contralateral side oc-
cur when there is a tumor in the temporal lobe.
When the occipital lobe is affected, some visual distur-
bances will appear (scotomes in the visual field.)
Expansive processes in the posterior cranial cavity
is manifested with cerebellar and cranial nerves dys-
function. Some characteristic signs and symptoms
accompany hypophysial tumors, craniopharyngi-
omas, and vestibular nerve neurinomes that could be
diagnosed by the characteristic bony changes. Apart
from the histological classification of tumors into be-
nign and malignant we have to think about some
malignant tumors due to their position when deal-
ing 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
cancer lung, ca prostate, renal carcinoma, tumors of the
thyroid gland, and the ca breast) usually grow from
the outside into the spinal coverings (extradurally). Tumors of the spine are manifested by backache that
can cause severe pain and affect the paraspinal tissues and their
nerve roots. Spinal compression will reveal some
motor disturbance of the limbs (paraparesis paraple-
gia, quadriparalysis, or quadriplegia), disturbances of
sensation and disturbances of sphincter function. Be-
nign tumors of the spine appear on x-ray due to their
pressure changes, while the malignant tumors show
destructive changes.

### 6.20 Injuries of the Spine

The primary pathological process plays role in the
anatomical and functional outcomes of the spinal in-
jury. 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 fracture or
dislocation of spinal vertebra, a tumor (usually sec-
ondary) that expands into the the spinal canal, disc
prolapse, or meningeal tumor that causes compres-
sion of the nervous tissue.

The spinal cord is very similar to the brain tissue,
the anterior and posterior roots as well as the ves-
seals are freely situated in the watery environment of
the cerebrospinal fluid. Upon compression there will
be some injury to the nervous and neuronal path-
ways 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 radiculitis and spondylosis. At
the level of the lesion connections between the sen-
sory 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 com-
plete 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 muscular paralysis and a loss of sensation below
the lesion. Spinal reflexes below the lesion are not
affected.

#### 6.20.1 Ascending and descending de-
generation

Long axons that are disconnected with their bod-
ies will undergo progressive degeneration. Upon a
transversal lesion of the spine all the long tracts degenerate upwards (sensory tracts) and downwards (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 motor pathways

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 hemorrhage 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 clinical 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 differentiation 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