cases of haematoma, and in case of using neurotoxic poisons. Brain edema might also ocur in cases of hypoproteinaemia, or in ionic spectrum disturbances. Brain edema very commonly occur as a consequence of a neurosurgical performance.

According to the course of the disease the brain oedema might be either **acute or chronic, localized or generalized**. The localized brain edema is characterized by localized symptoms, headache, vertigo, vomiting, papillary edema, and slight disturbance of consciousness. A generalized brain edema is associated with loss of consciousness, signs of brain stem compression with threatening of vital functions, and there might be decerebration rigidity. The characteristic sign that accompanies this condition is **bradycardia and an increased blood pressure** in the systemic circulation. Tachycardia and a decreased blood pressure is usually a sign of deterioration.

The diagnosis of brain edema has to be based on a proper evaluation of all signs of intracranial hypertension. It is incorrect to talk about brain edema, as soon as the brain volume increases. It might be a case of increased blood volume (bleeding) or liquor volume (obstruction), that doesnďt have to be linked to the brain edema. It is of a great value to monitor the intracranial liquor pressure, X-ray of the scull, angiography and mainly CT.

The treatment has to be systematic in treating the cause, early, and complex. It has to provide an adequate exchange of gases, stabilize the circulation and remove the cause of brain edema. The symptomatic tratment leads to:

1. The decrease of the metabolism and energy requirement of the brain tissue,

2. Stabilization of the metabolic balance by the balanced fluids, electrolites and proteins,

3. Increase in the stability of haematoencephalic (blood-brain barrier),

4. Removal of the pathological overload of fluids from the brain tissues by changing the osmotic gradient (osmotherapy).

6.8 Intracranial hypertension

Intracranial hypertension or **a high intracranial pressure syndrom**. The intracranial tension is determined by the reciprocal relation between the intracranial space volume and the volume of the tissues (brain, blood, and the cerebrospinal fluid). That are situated within it. The intracranial pressure in the normal conditions ranges between 5–5 mm Hg (0.7–1.2 kPa or 60–180 mm H_2O). During the physiological conditions the intracranial volume can change only during childhood (until a complete adhesion of the intracranial sutures). The ratio of the brain tissue to the liqour and blood might change only slightly. Despite of this fact there are some compensatory possibilities, and mainly those of regulation of liquor formation and resorbtion and even a partial shift of liquor extracranially. Later on it is possible to achieve a partial reduction of the blood volume upon increasing the blood pressure.

The intracranial pressure increases due to two general causes:

- **an expanding process and**
- **flow obstruction of the cerebrospinal fluid (hydrocephalus**)

An intracranial expanding process arrises either from the brain tissue itself or from its covers (meninges). The most important examples are:

1. Intracerebral bleeding and haematomas (traumatic or spontaneous), brain infarcts and tumors (primary or secondary – metastasis).

2. Usually a traumatic bleeding to the area of brain meninges (extradural, subdural, and subarachnoidal).

A very rare condition is the appearance of **meningeoma** and **hydrocephalus** that will be thorowly discussed elsewhere.

In children with still opened cranial sutures the intracranial hypertension (ICH) is associated with cranial expansion of the fontaneles and their pulsations. Upon percussion of the head there will be what is known by cracked-pot sound. In older people the intracranial cavity can not be changed in size

and that is why upon exhaustion of all the compensatory mechanisms together with the intracranial pressure raise there will be the appearance of papillary n. edema on the eye background, and due to the pressure on the vital centers in the medula oblongata there might be some resulting respiratory disturbanes as well as disturbances of the heart rate and action and blood pressure with multiple vegetative signs.

In more advanced cases of intracranial pressure there will be formation of intracranial pressure cones – the compression of the brain into foramen magnum (**Arnold-Chiaris syndrom**) and hence below the tentorium cerebelli. Due to the brain tissue shift a disturbance of function and structure of the CNS will occur. Brain edema is a serious complication of the intracranal hypertension.

In general the seriosity of the case depends on he site of lesion, time length of the expansion and the character of the causative agent. According to this we distinguish **4 stages of intracranial hypertension**:

1. The stage of **total compensation**. Where the expansive process causes an increase of the intracranial pressure, that leads to an increase in the resorbtion of the CSF and its lower production. This is how the volume of CSF is decreased, so that the space that is formed can be filled with the expansive process. In this case the expansion doesnd't compress the brain tissue yet, and so the symptoms of compression are minimal.

2. The stage of **partial compensation**. In this stage the compensation reduces the intracranial volume of the circulating blood with no change of the tissue perfusion. This is possible by vasoconstriction, that increases the blood pressure. The systemic arterial blood increases, reminding you that this shoud provide the proper brain perfusion.

3. The stage of **decompensation**. In this stage there are no possibilities to further compensate the brain tissue expansion without an injury to the brain tissue. There will be a shift of the brain tissue in the cranial cavity, herniation and distorsion, that accompany many CNS functional abnormalities. The systemic arterial pressure will raise yet the heart rate is slower (Cushingd's effect).

4. **The vasomotor paralysis**. In this stage there will be a viscious circle: due to the higher intracranial pressure there will be a low brain tissue perfusion, that leads to hypoxia of brain tissues (and the pCO_2 will increase). These changes already lead to a progressive neuronal injury and a vasomotor paralysis, that worsen the hypoxia even more. Hypoxia and hence the accumulation of CO_2 in the low perfused tissues disturbes vasoconstriction and leads to a prominant vasodilatation, that increases the intracranial pressure. Upon the increasing intracranial pressure in this stage even brain edema might occur. By this the pathological ring is closed.

The intracranial hypertension is manifested with many subjective and objective symptoms and signs. We are mainly talking about headache (e.g. the areas known as the ventilation areas in the IVth ventricle obstruction due to a localized carcinoma, that will worsen upon laying down, upon holding the breath, and other conditions leading to an increase of the intracranial pressure. The patients complain of nauzea, vomiting, respiratory disturbances, vertigo, blurred vision (papillary edema). The alterations of the brain are manifested with disturbances of the higher neurological activity (bad memory, concentration and etc.).

From the objective signs the higher liquor pressure is in the first place in the early stages the blood pressure raises (in the late stages it decreases), the heart rate and action is decreased (bradycardia), papillary edema might even lead to optic nerve atrophy and blindness. In case of brain tissue lesion some localized neurological signs might appear. We ought to mention that upon **a sudden diagnostic lumbar punction the so called medullary conning might occur** with its fatal drowbacks due to the injury to the vital centers of the medulla oblongata and the base of the brain. Apart of these so called primary results of the intracranial hypertension, there might also be some secondary complications. These might be vascular injuries (1), intracranial nerve injuries (2), a cerebrospinal fluid flow obstruction (3) and bony changes of the cranium (4).

1. **Papillary edema of the optic nerve** is a sign of intracranial hypertension that occurs due to the compression of the central retinal vein. Vascular compression and expansion can lead to their

rupture, haemorhage and infarction that might mask the original disease.

2. The oculomotor nerve (the IIIrd) and the abducent nerve (the VIth) are the anatomically most vulnerable nerves, and this is shown by **the paralysis of the eye bulbs**. The abducent nerve is vulnerable due to its long course in the subarachnoidal space. It is commoly injured on the contralateral side (the paradoxical sign) of the causative lesion.

3. An expansion that leads to intracranial hypertension might compress the midbrain, that can lead to the narrowing or even the obstruction of the aqueductus Sylvii. The cerebrospinal fluid circulation will be disturbed and the CSF will cumulate in the lateral ventricles and the IIIrd ventricle. An increased pressure leads to tissue damage in the area of the ventricular systems. The distension of the ventricular system worsen the intracranial hypertension.

4. A longlasting increase of the intracranial blood pressure leads to **the errosion of the brain bony cover**, which could be seen on the X ray. In children with intracranial hypertension the skull is deformed in shape, and especially if the fontanels are still open. Widening of the bony sutures of the skull, the pulsation of the fontanels and the prolonged time of fontanel ossification are other mechanisms, that can partially compensate for the process that leads to intracranial hypertension.

6.9 Hydrocephaly

In hydrocephaly the volume of the cerebrospinal fluid (CSF) is increased and the ventricles are dilated. In most of the cases the intracranial pressure is increased as well. Generally 3 possible mechanisms are accepted to be the cause of the development of hydrocephaly:

1. **An excessive production of CSF**. Where the choroid plexus produces more CSF, than normal whereas the resorbtion is relatively unchanged. Yet the overproduction of the CSF is not the cause of hydrocephaly. We have to look for the cause of the overproduction of the CSF.

2. **CSF flow obstruction** (the most common cause), that disturbes the circulation of the CSF in the intracerebral or the extracerebral space. According to the level of obstruction we devide hydrocephalus into the non communicating – closed (obstruction above the level of the IVth ventricle e.g. aqueductus Sylvii, foramina Megendie and Luschke – the CSF doesnd't reach the subarachnoidal space) or the communicating (the CSF circulation is obstructed in the subarachnoidal space.).

3. **Disturbance of the CSF absorbtion** (a rare abnormality). The absorbtion usually takes place in the venous sinuses of the arachnoid. This abnormality can result from an inflamatory process, bleeding, or the obliteration of the areas of resorbtion. The obstruction might be temporary upon taking some positions, or upon some head movements – this is known as **a ventil hydrocephaly**. It can develop with some tumor types, that grow from the stalk into the ventricular system of the brain.

The causes of hydrocephaly are devided into two groups:

1. congenital (developmental) abnormality,

2. acquired hydrocephaly

The congenital or the developmental abnormalities that might cause hydrocephalus can be devided into many groups:

1. **Arnold-Chiary malformation**, in which due to the increased intracranial pressure the cerebellum and medulla oblongata are pushed into the foramen magnum, upon which there will be CSF flow obstruction. The intracranial pressure increases and this might injure the respiratory centers in the medulla oblongata.

2. **Congential stenosis or atresia of aqueductus Sylvii**, that is manifested by an excessive dilitation of the IIIrd ventricle and the lateral ventricles with a following increased intracranial pressure.