

be signs of intracranial hypertension and different neurological symptoms that bring the patient to the neurologist.

- (b) **Subdural haematoma.** Can occur in any location, is extensive because dura mater and the arachnoidea are not tightly adherent. It occurs most commonly due to the rupture of small bridging veins.
- (c) **Intracerebral haemorrhage** usually occurs together with the cortical contusion – most commonly in the temporal or the frontal region due to the effect of the injurious mechanism. Yet the haemorrhage during the trauma might be deep in the tissues. The cause is vascular injury that is usually not very extensive.

2. Brain edema (is described elsewhere 3.7)

3. **Leak of the cerebrospinal fluid (CSF)** and blood from the nose and ear can be a complication of cranial base fracture. It can be prolonged and it creates an entry for infection.

4. **Infection.** Might complicate fracture healing and it can affect other structures e.g. meninges.

6.6.1.3 Late complications

1. **Scar formation and epilepsy.** During the process of scarring by the resorption of necrotic tissue degradation products, there will be formation of pigment scars. These can potentiate the occurrence of epileptic firing, mainly when the meninges adhere to the brain tissue.

2. **Chronic subdural haematoma.** Occurs mainly in the elderly patients and alcoholic patients. A thick layer of fluid and a partially coagulated blood gradually cumulates between the thickened dura mater and the arachnoidea. Signs of brain atrophy and compression appear in the adherent area. The pathomechanism is not clearly understood. We assume that, there will be damage of the small connecting bridging veins, which together with the lower blood coagulability can be the causative factor of this type of bleeding. The clinical manifestation usually includes a marked beginning with the progression of neurological symptomatology. The history doesn't include any serious head injury in most of the cases,

it is not uncommon to find only a benign head injury (for e.g. getting into or out of a car).

6.7 Brain edema

Edema (a swelling) of the brain can be characterized as **an increasing fluid content** in the brain tissue. Brain edema results in an increased extracellular (more in the white matter) or an intracellular (more in the grey matter) volume, increases the intracranial pressure, worsens the course of the primary disease. It occurs in many brain diseases. This process might be localized or generalized according to the causative agent. An example of the **localized edema** can be **brain infarction, local brain ischemia, haematoma or tumor**. An example of a **generalized brain oedema** can be that caused by **intoxication, metabolic disorders, hypoglycemia, generalized hypoxia** and a severe **head injury or malignant hypertension**. Brain edema can occur in association with the disequilibrium syndrome that occurs with **dialysis, diabetic ketoacidosis, hyposmolarity**, in different forms of **obstructive hydrocephalus** and upon **hepatic dysfunction**.

The outcome of brain edema, either the localized or the generalized type, is, worsening of brain perfusion, a disturbance of cerebral haemodynamics, and metabolism disturbances with the consequent loss of consciousness. The pathomechanism is given by the characteristics of the initial insult. In the initial stages the most common mechanism applied is the so called **cytotoxic component**, that leads to cellular membrane injury, disturbance of ion balance of the membrane (Na^+ , K^+) and the shift of water from the plasma to the interstitium. The major components of this phase is hence the shift of water into the interstitium based on the ionic imbalance that results from cellular membrane injury. In the later stages there will be the so known vasogenic component, that is manifested by capillary wall damage, and the shift of plasma proteins to the interstitium with the consequent oncotic water binding (shift of water to the interstitium). The edematous fluid has the tendency to spread in the white matter. From the

pathogenetic point of view we can differentiate two types of brain edema:

1. **The cytotoxic (metabolic) edema**
2. **The vasogenic edema**

A unique position is that of **the interstitial edema**, where some authors even describe brain edema as a result of ischemic causes – **ischemic edema** – as an independent unit.

In case of **the cytotoxic (metabolic) edema** the toxic factors cause a direct disturbance of the active transport of brain cells (neurones, glia, endothelial cells). The cells lose K^+ whereas Na^+ enters the cells and the cells enlarge. This is how the volume of the brain cell is increased on the account of the extracellular space. The cytotoxic edema is usually localized in **the gray mater** and can be combined with **the vasogenic edema**.

The vasogenic edema is the most important type clinically. It is caused by **osmotic disturbances**, changes in **the membrane permeability, disturbance of the Na pump**, the toxic effect of **free O^- radicals** (oxygen) that cause the Ca^{2+} influx to the cells to be increased. There might be a co-association of prostaglandins with leucotrienes. The generation of the so called permeability disturbance of the blood brain barrier as a consequence of a certain pathogenic effect of (trauma, inflammation, tumor or vascular injury). The blood-brain barrier dysfunction can lead to the diffusion of **plasma proteins extravasally to the extracellular spaces**. **Water follows the shift of plasma proteins**, which increase the content of water in the brain parenchyma. The vasogenic edema appears in the area of injury and it spreads to the peripheries, where it affects **the white mater** prominently, whereas the gray mater is relatively resistant. The first to be affected are the basal ganglia. Edema is worsened by the coaction of the resulting ischemia (tissue compression, worsening of the perfusion).

Some authors consider some other type of brain edema:

Ischemic edema is most commonly associated with brain infarction. Brain ischemia is accompanied by vasogenic or even cytotoxic component. Ischemia of the brain tissues will gradually result in **an intracellular edema** of the subcellular organelles (mitochondria). In case of a continuous ischemia and

tissue hypoxia there will be an irreversible damage and necrosis where lysosomes are released from the cells. Lysosomes initiate an autodigesting process, during which the blood brain-barrier is prominently further destroyed and its permeability is increased. **Diffuse edema of the gray and the white mater** appears in status epilepticus, uremia and eclampsia.

Interstitial edema is most commonly seen in case of obstructive hydrocephalus. Edema is caused by **a transependymal shift of the cerebrospinal fluid** from the ventricles to the extracellular spaces of the brain tissue. By this way the volume of fluid is increased mainly around the ventricles. The hydrostatic pressure in the white mater rises and there will be destruction of the myelin sheaths. The involvement of **white mater** mainly is typical for edema that occurs with inflammations or allergic situations.

Brain edema is potentiated by **the so called stress post-burden reaction**. There will be the activation of hypothalamo-hypophyso-adrenal system with the consequent release of catecholamines. There will be peripheral vasoconstriction and dilatation of the cerebral arterial system. This will become more prominent by cerebral hypercapnia. The resulting perfusion disturbance, will worsen the ischemia even more and there will be ganglion cell membrane injury with the disturbance of the active and passive transport. The neurones undergo **Alzheimers degeneration** and they necrotize. By this the brain edema reaches its peak.

The progressive progression of the brain oedema has apart of the primary outcomes even secondary outcomes due to increasing **the intracranial pressure**. In more advanced stages these two mechanisms complete each other and prominently worsen the patient's general condition.

A precisely localized brain edema is very rarely seen. The most common form is the combined one. The primary ischemic origin of the brain edema can occur in blood diseases (anaemia, haemoglobinopathy, etc.), in cardiovascular disorders (shock, MI, arrhythmia, bleeding, vasospasms, decapitated hypertension, hypotensive states, etc.), in breathing disorders, in cases of high cerebrovascular resistance, in cases of intracranial expansive processes, intracranial hypertension, and spasm states (epilepsy, eclampsia). On the other hand when **the brain edema is rather due to toxicity** it is usually a case of malignant brain tumors, upon contusions and brain malacia, in

cases of haematoma, and in case of using neurotoxic poisons. Brain edema might also occur in cases of hypoproteinaemia, or in ionic spectrum disturbances. Brain edema very commonly occurs 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 doesn't have to be linked to the brain edema. It is of a great value to monitor the intracranial liquor pressure, X-ray of the skull, 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 treatment 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, electrolytes 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 H₂O). 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 liquor and blood might change only slightly. Despite of this fact there are some compensatory possibilities, and mainly those of regulation of liquor formation and resorption 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 arises 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 thoroughly discussed elsewhere.

In children with still opened cranial sutures the intracranial hypertension (ICH) is associated with cranial expansion of the fontanelles 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