

According to the type of cerebral oxygen supply disorder there will be some localized changes that usually occur upon short lasting and mild decrease of pO_2 , or generalized (diffuse) injury, that might be the result of a total disorder of oxygen supply to the brain. In this relation, it is worth mentioning that similar neuronal injuries can also occur in acute hypoglycemia, and barbiturate or CO intoxication.

6.13.3 Hyperoxic syndrome

One of the disturbances of O_2 supply to the brain in which brain damage occurs due to an increased pO_2 in the brain. It follows inhalation of pure O_2 under high pressure (e.g. upon treating CO poisoning). There might be syncope, nausea, blurred vision, epileptic attack. The mechanism is unknown. We suppose that a high pO_2 results in vasoconstriction and lowering of glucose supply and hence a slower metabolite wash out that leads to the injury of nerve cells.

6.14 Cerebral infarction

The basic cause of **brain (cerebral) infarction** is **failure of oxygen supply to the brain** (as well as failure of glucose supply). The infarction is in the area supplied by a terminal vessel. The most common cause is a local disturbance of the blood flow via vessels (occlusion), yet it is very common that a central cardiovascular failure contributes to these disturbances of perfusion. The result will be necrosis of all tissues supplied by the terminal vessel.

The pathophysiology of the occurrence of infarction can be summarized as follows:

risk factors – disturbance of perfusion – infarction (ischemia, necrosis).

As we already mentioned, there is commonly a local arterial occlusion or stenosis.

1. **Arterial occlusion** is usually caused by thrombosis on the atheromatous plaque or embolization (mostly cardiac – atrial fibrillation – endocarditis – intramural thrombus that follows myocardial infarction). Small infarcts can result from small

emboli from the sloughed atheromatous plaques in the region of the internal carotid artery.

2. **Arterial stenosis.** Commonly occurs in cases of atheromatosis, because **atheroma** causes a turbulent blood flow in the area of narrowing and there will be some disturbance of the vascular anastomosis among the neighbouring areas and blood vessels. Despite of all that it is not necessary that an atheromatous narrowing itself should always result in infarction. The infarction usually occurs when a central cardiovascular insufficiency contributes to the narrowing. The cerebral infarction usually occurs upon lowering the blood pressure during sleep, or when the blood pressure decreases in cases of shock or myocardial infarction. One of the less common causes of the brain infarction is dissecting aneurysm, arteritis and very rarely it could be caused by vascular spasm.

Hypertensive patients have a higher risk to develop cerebral infarction, that is indirectly caused by speeding up the development of arteriosclerotic changes and formation of **atheromas**. In these cases there will be formation of large and small cerebral infarctions, as well as dementia that is conditioned by arteriopathy. In cases of malignant hypertension there might occur the so called **hypertensive encephalopathy**. The pathomechanism of its occurrence is a very fast raise of the blood pressure that consequently leads to disturbed cerebral circulatory autoregulation. There will be a focal dilatation of the small vessels, an injured endothelial lining and a disturbance of the integrity of vascular wall that leads to protein leak into the interstitial space (together with the erythrocytes), that might end up with **brain edema**. This condition is clinically manifested with some neurological signs, spasms, stupor till coma. Papillary edema of the optic nerve is always a sign of an increased intracranial pressure.

The clinical picture differs according to the affected site. The injury might affect any area of the brain yet some areas and localities are more commonly involved. The most vulnerable area is the area of the so called end arterioles in those areas that lack collaterals. The cerebral cortex is relatively protected by a rich network of vascular collaterals. The injury is common to occur in the area of the medial cerebral artery and mainly the end arterioles

that supply the area of basal ganglia and capsula interna. The cerebral tissue changes that occur due to age are multiple such as microemboli that result in **microinfarction** with the following reduction of cerebral function. This type of cerebral atrophy is a very common cause of senile dementia in our population.

The greatest loss of function occur directly following the occurrence of infarction. During this period the situation is worsened by brain edema and disturbance of the cerebral vascular supply (ischemia, hypoxia, hypoglycaemia, etc.) A large infarction is associated with loss of consciousness. Within few days, when the infarct is organized, macrophages will appear in the area, there will be the formation of new capillaries, the edema ceases, and the area of functional dissability decreases. The compensation of the lost functions is one of the characteristics of the brain tissue. However, this compensation might require few weeks and commonly maximal rehabilitation. A complete clinical renewal of the lost functions is possible in cases of small infarct only.

6.15 Intracranial haemorrhage

A spontaneous **intracranial haemorrhage** (IH) is very rare to occur. Usually there is a **local vascular abnormality**, that is manifested in cases of **hypertension**. A number of IH cases occur in **cerebral tumors**, systemic **diathesis** and **arterio-venous malformations**.

Basically, the intracranial haemorrhages are divided into two groups:

1. Intra cerebral haemorrhage
2. Subarachnoid haemorrhage.

6.15.1 Intracerebral haemorrhage

Intracerebral haemorrhage occurs in middle aged patients suffering from **hypertension**. They often have **microaneurisms** on the small of the cerebral arteries. A direct cause of haemorrhage is **rupture** of one of these aneurisms. The haemorrhage commonly occurs in the region of medial cerebral artery –

and mainly its bifurcation to supply the basal ganglia and the internal capsule, the haemorrhage might also occur in pons and cerebellum. The course is usually sudden (acute), from complete health, the patient develops headache, dizziness and hypertension commonly accompanies these conditions, the haemorrhage will proceed very fast. There will be an intracranial hypertension CNS functional disability, unconsciousness and death might occur. If the haemorrhage is localized, the manifestations will be less dramatic. There might be variable degrees of dysfunction (paralysis according to the locality of the haemorrhage). Intracerebral haemorrhage always runs the typical course and usually spills into the ventricular space or the subarachnoid space.

In cases of untreated hypertension, the pressure in the brain capillaries might increase resulting in altered vascular wall permeability. This is how proteins, erythrocytes and other intravascular components reach the brain tissues and brain edema might occur. We are talking about **microhaemorrhages** into the brain tissue. The common end result of this process is the gradual necrosis of brain tissue and the formation of **the hypertensive encephalopathic symptomatology**.

The risk of intracranial haemorrhage is higher in patients suffering from hypertension by about 7-folds. The pathomechanism arises from the fact that in hypertensive patients the blood pressure is markedly raised in small arterioles with the resulting degeneration of the vascular wall. This might directly lead to wall rupture and hence the formation of aneurism and its following rupture. The prominent pulsation of these arterioles leads to the compression of the surrounding tissues (excavation), and a small perivascular space will be formed (lacunes). The vascular wall loses its back up that also leads to its weakening.

6.15.2 Subarachnoid haemorrhage

Subarachnoid haemorrhage is the most common manifestation of saccular aneurisms that are situated in the area of circle of Willis. The basic cause is a congenital malformation of the elastic constituent of the vascular wall. The aneurism is rare to be manifested during birth. The size of the aneurism can vary from 1–2 mm till 1–2 cm. However not all aneurisms cause haemorrhage. The incidence increases with age. The aneurism can rupture directly into the brain tissue