Status epilepticus (pediatrics)

90% of uncomplicated generalized seizures last less than 2 minutes, rarely up to 5 minutes, i.e. it is necessary to treat each seizure lasting 5, maximum 10 minutes as status epilepticus (SE). SEs are also ≥ 2 discrete seizures between which there is no adjustment of consciousness. Refractory (non-responsive to treatment) SE is characterized by SE lasting > 60 minutes and resistant to treatment modalities I.–II. step (see therapy). The mortality of generalized tonic-clonic SE is 10–20%.

SE most commonly manifests as generalised tonic-clonic seizures, but there are other types of status epilepticus, including non-convulsive status epilepticus.

Classification

The classification of SE corresponds to the same classification as for epileptic seizures.

Classification in terms of convulsive vs. of non-convulsive SE

Convulsive status epilepticus

1. SE with generalised convulsions
2. SE with focal (partial) convulsions and secondary generalisation
3. SE with myoclonus.

SE with simple focal seizures, epilepsia partialis continua

Seizures can be permanent, especially if they are associated with a focal CNS lesion. Simple partial seizures can be tonic (= permanent muscle contraction of a body part) or clonic (= alternation of muscle contraction and relaxation) without major alteration of consciousness. Simple partial seizures can be accompanied by various subjective bodily or sensory sensations (e.g. visual hallucinations). Prolonged simple partial seizures (often motor and clonic) are often called epilepsia partialis continua. Simple partial seizures are not always associated with diffuse CNS damage unless they progress to complex partial SE or are associated with secondary generalisation.

Non-convulsive status epilepticus

Non-convulsive SE is characterised by continuous epileptogenic activity on the EEG without a motor correlate.

1. Status of absences
2. Status of focal seizures with complex symptomatology
3. Status of complex focal seizures and absences

Classification in terms of generalised vs. of focal SEs

SE classification

Generalised SE	Focal SE
Convulsions	Simple : Without alteration of consciousness
Absence (possibly with subsequent convulsions	Complex : With impaired conciousness

Pathophysiology

From a pathophysiological point of view, SE is classified into three groups:

- Acute symptomatic SE - Convulsions are caused by acute infection, CNS trauma, hypoxemiay, hypoglycemia, intoxication, ion imbalance, etc.;
- SE is a chronic progressive neurological impairment (neurodegenerative disease);
- SE is a non-progressive neurological impairment - convulsions in connection with CNS insult in the perinatal period, epileptic syndrome, tumour, hydrocephalus;
- Idiopathic SE - The cause of spasms is not revealed

In children < 2 years, convulsions with temperature and acute symptomatic SE are the most common, in children > 2 years, the most common cause is idiopathic or SE with non-progressive neurological impairment.

Cramps for which we are able to identify the cause are called symptomatic, in the case of an unclear cause we choose the term idiopathic or cryptogenic.

Occurrence of convulsions

It is caused by abnormal rapid electrical activity of brain neurons. The cause may be an increase in excitatory neurotransmitters, e.g. glutamate or, conversely, a failure of neuronal inhibition. γ-Aminobutyric acid (GABA) is the main inhibitory neurotransmitter in the CNS. Deficiency of this acid or alteration of GABA receptors can lead to excessive CNS excitation and seizure prolongation. We can theoretically assume these disorders in patients with a poor response to benzodiazepines (the site of their activity is precisely the GABA receptor).

Alteration of consciousness and/or motor activity occurs. Hypoxic-ischemic mechanisms are the principle of neuronal damage in SE. The consumption of oxygen, glucose and energy substrates (ATP, creatine phosphate) is significantly increased in the brain tissue during a seizure. After 20 minutes of stat duration, pO 2 and cytochrome aa3 activity decrease in the cortex. Toxic amounts of lactate, arachidonic acid, prostaglandins and leukotrienes accumulate in neurons. This leads to the development of cytotoxic brain edema and the death of neurons in some areas. In addition, there is also a breakdown of autoregulation of cerebral perfusion, which further contributes to the disparity between the demand and supply of energy substrates and oxygen.

After 60 minutes of a convulsive state, irreversible damage occurs especially to the hippocampus, amygdala, cerebellum, thalamus and middle neocortical layers. Repetitive convulsions in the newborn and infant age may contribute to a general reduction in the number and size of brain cells and to the formation of an organic epileptic focus. Other residual findings include hemiparesis or psychomotor retardation. On the other hand, even after an average of 90 minutes of stat, the patient may not have any residual neurological findings.

In addition to the above-mentioned direct neurotoxic mechanisms, hypoxia and hypercapnia from the stasis of secretions in the upper airways, obstruction by a sunken tongue, laryngospasm, aspiration of stomach contents, tonic spasms of the respiratory muscles, hyperthermia endanger the patient in a convulsive state. Hypoxemia and hypercapnia further adversely affect cerebral metabolism. Increased sympathetic activity during convulsions leads to systolic hypertension, arrhythmias. Arrhythmia is often one of the sudden causes of death during stat.

Lactic acidosis is the result of hypoxia and increased lactate production during muscle spasms. Prolonged convulsions can cause the breakdown of muscle cells (rhabdomyolysis) with subsequent myoglobinuria and the risk of acute renal failure.

The longer the status lasts, the more difficult it is to end it, because inhibitory neurotransmitters in the brain decrease proportionally to the duration of the status. Individuals with organic CNS involvement respond worse to treatment than patients with previous normal neurological findings.

Etiology

The most common cause of SE is etiological units from the group of **acute symptomatic SE** (infection, trauma, hypoglycemia, hypoxemia, intoxication), followed by **idiopathic SE** and **febrile SE**.

The most common intoxications leading to SE include: local anesthetics, overdose of anticonvulsants, ethanol, insulin, CO, cyanide, heavy metals, pesticides, cocaine and amphetamines, nicotine, tricyclic antidepressants.

Not only CNS infection is the cause of SE. The source of the infection may not always be clear (pneumonia, otitis media). Early and consistent treatment is essential, as infection lowers the threshold for convulsions in predisposed patients. SE in children often develops as a result of epileptic syndromes: Lennox-Gastaut syndrome, myoclonic-astatic epilepsy, absence, partial seizures.

Newbrons	**Children under 6 years of age**	**Children older than 6 years of age**
- Perinatal damage: Hypxia, bleeding, CNS malformations - Metabolic disorders: Hypoglycemia, hypocalcemia, hypomagnesia, hyponatremia -Hereditary disorders of metabolism : Lipidoses, aminoaciduria	Perinatal damage, trauma, infection, epilepsy, degenerative CNS disease, tumours, intoxication, metabolic disorders, neurocutaneous syndromes, febrile convulsions, idiopathic	Perinatal damage, trauma, infection, epilepsy, degenerative CNS disease, tumours, intoxication, iodiopathic

Clinical

Clinically, for the sake of simplicity, seizures can be divided into **focal** and **generalized**.

Generalized tonic-clonic seizures (in the old nomenclature *grand mal*)

Tonic-clonic seizures are the most commonly seen seizures in intensive care units. They arise during bilateral synchronous epileptic activity.

- The onset of generalized tonic-clonic convulsions is usually sudden. If the patients are standing or sitting, they suddenly fall to the ground, they are pale, the pupils are in mydriasis, the eyes are rolled up or to the side, the muscles are in contraction. Due to the increased muscle tone of the chest and abdominal muscles, we can hear sound phenomena similar to grunting when breathing. Urinary and/or faecal incontinence is common.
- After this brief tonic phase (10–30 seconds), clonic jerks occur. During a seizure, children are unresponsive, and areactivity persists nonconstantly even postictal.

- After conception, some patients may experience weakness or paralysis of one or more parts of the body (Todd's palsy). It is important not to confuse secondary generalizing focal seizures with primary generalized seizures.

Akinetic-atonic seizures

We observe a sudden loss of muscle tone and consciousness. Akinetic seizures are characterized by sudden, short-lasting stiffness, while an atonic seizure is characterized by a fall with loss of extensor tone.

Myoclonic jerks

They can be small-segmental or massive generalized with flexion of the head, shoulders (sign of a closing knife). Myoclonic, akinetic and atonic seizures are also collectively called "minor motor seizures".

Absence (in old nomenclature ***petit mal***)

They are generalized seizures characterized by a sudden and brief loss of consciousness, usually lasting 5–30 seconds, i.e. glancing and areactivity. In typical absences, loss of posture or muscle tone is absent, and postictal confusion is absent. Small motor symptoms such as eyelid blinking may be expressed here. It is precisely these patients, where consciousness can only be impaired intermittently, that can cause diagnostic difficulties even with developed SE, because the clinical symptomatology is subtle and the correct diagnosis can only be established by EEG. Absences with a pronounced atonic, tonic or clonic component or accompanied by a pathological neurological finding are defined as atypical.

Simple focal seizures

Children with simple focal seizures have preserved consciousness. These are seizures that originate in a limited, anatomically or functionally definable area of the brain. As a rule, they appear on the opposite side of the body in relation to the bearing. A sensory or motor aura is often described (aura = subjective sensations experienced immediately at the beginning of a focal seizure). In childhood, motor forms are the most common, but sensory, autonomic and psychological forms can also appear. Motor activity usually affects the hands or face and leads to fixed forms of motor manifestations in a certain anatomical area.

Complex focal seizures

Complex focal seizures are called **psychomotor** or **temporal**. The clinic includes a disorder of perception and thinking. In the foreground are repetitive and complex movement patterns – blinking, clapping, aimless limb movements. There is usually a partial disturbance of consciousness and postictal mental depression (sleepiness to lethargy). Both focal simple and complex seizures can secondarily generalize.

Types of Epileptic Seizures

<table>
<tr><th>Generalized</th><th>Focal (partial)</th></tr>
<tr><td>Absence (petit mal)
• Typical
• Atypical</td><td>Simple (no impaired consciousness
• Motor
• Sensory
• Autonomic
• Psychic</td></tr>
<tr><td>Tonic-clonic (grand mal)</td><td>Complex (partial impairment of consciousness)</td></tr>
<tr><td>Clonic</td><td rowspan="4">Focal (Simple and Complex) with secondary generalisation</td></tr>
<tr><td>Tonic</td></tr>
<tr><td>Minor motor seizures
• Myoclinic
• Akinetic
• Atonic</td></tr>
</table>

As mentioned above, SE can manifest as classically generalized bilateral and synchronized tonic-clonic limb jerks or myoclonic rhythmic movements involving the limbs, face or eyes. Clonic activity may begin focally (rhythmic contractions of facial muscles or limbs), progress to hemiconvulsions, or become generalized.

Asynchronous alternating limb movements are often considered pseudoseizures, but a similar course can be seen in frontal lobar epilepsy. **Epilepsia partialis continua** is manifested by unilateral and occasionally focal (one hand or even one finger) clonic jerks.

Patients with SE with absences present with impaired consciousness, sometimes with the presence of clonic movements of the eyelids or upper limbs and automatisms affecting the hands and face. In some cases, the patient can answer simple questions, but a careful examination of the mental status will reveal a mild quantitative

disorder. Episodes of SE with absences often last longer than 12 hours.

If the cause of convulsions is sepsis or meningitis, we detect fever, respiratory distress, cyanosis, reduced peripheral perfusion, bulging fontanel in infants, meningeal signs (in children > 18 months), finding of petechiae or herpes blisters.

Differential diagnosis

In the differential diagnosis, exclusion is the most important

- **Syncope**.
- **Affective seizures** (breath-holding spells).
- **Psychogenic seizures** – We observe the initial tight closing of the eyelids, hyperventilation, catatonic posture of the lower limbs and jerks of unchanging frequency. Atypical migraines and pseudoseizures must also be ruled out.
- **Sleep disorders** - somnambulism, night terrors (preschoolers) and narcolepsy (typically in adolescence) can be diagnosed already on the basis of anamnesis.
- **Benign myoclonia** - we observe as self-limited episodes of sudden jerky movements of the limbs, usually during falling asleep.
- **Torticollis or dystonia (Sandifer syndrome)** - can develop in children with gastroesophageal reflux. Acute dystonia can also be observed as an adverse effect of some drugs and can imitate tonic convulsions. The child has no disturbance of consciousness, no signs of postictal depression.

Syncope

Syncope represents a short-term disturbance of consciousness due to inadequate cerebral perfusion or a disturbance in the supply of energy substrates to the CNS. Short-lasting convulsive movements can be observed in a small percentage of patients. The most common cause is syncope mediated by the autonomic system. However, cardiac disease must always be ruled out (severe ventricular tachycardia - torsade de pointes or critical aortic stenosis can present as syncope).

Affective seizures

Affective seizures affect children between the ages of 6 and 18. month of life and their differentiation is possible on the basis of anamnesis. It comes as a cyanotic or "pallid" form.

Diagnostics

Anamnesis

Anamnesis is the basic guide to uncovering the cause of the development of SE. It is necessary to obtain a detailed history from the child, his parents or those who were present at the seizure. Of course, we obtain anamnestic data only after the patient has stabilized.

We ask about the nature and onset of convulsive activity, whether convulsions were observed only in the limbs or other parts of the body. The nature of the convulsions is important – eye movements, twitching of the eyelids, flexion, extension or stiffening of the limbs, other focal movements. We are interested in postictal neurological deficit and mental status. We find out if incontinence, cyanosis, amnesia was present.

We ask about the presence of fever or intercurrent illness, the presence of head trauma, CNS infection or disease (neurocutaneous syndromes), intoxication, other CNS abnormalities (VP shunt, perinatal damage), other diseases (immunodeficiency syndromes, SLE, diabetes mellitus).

Laboratory examination

While we provide the patient with adequate care and therapy, we perform basic laboratory tests – KO + differential budget of blood cells, electrolytes including calcium and magnesium, glycemia, urea, creatinine, liver tests, Astrup. It should be noted that the detected leukocytosis may be due to demargination of leukocytes during convulsions - in this case, the leukocyte value returns to normal within 12-24 hours. Other widespread tests include toxicological screening, ammonia, levels of antiepileptic drugs, carboxyhemoglobin. In urine chemistry, we look for myoglobinuria.

If a neuroinfection (febrile, meningism) is suspected, we perform a lumbar puncture, meningeal signs may be negative. Lumbar puncture is also often routinely indicated in patients with immunodeficiency, the indication may also be unclear etiology of SE.

Display methods

Imaging methods, if indicated, are also performed after the patient has been stabilized. Among the most important examinations is a CT scan of the head. Indications are patients with a history of previous neurological impairment, including impaired consciousness, patients with a persistent neurological deficit after convulsions subside. In the

case of reasonable suspicion of intracranial hypertension, a CT examination may be performed. performing a lumbar puncture. CT scan reveals CNS malformations, bleeding, focal lesions (tumor, abscess), displacement of structures across the midline.

Children with complex focal seizures that precede or lead to generalized tonic-clonic convulsions should have an MRI of the CNS (here we find changes due to transient vasogenic or cytotoxic brain edema). MRI is usually not readily available, moreover, it requires a thoroughly stabilized patient (length of the examination, the impossibility of therapeutic interventions during the examination), so it is usually indicated only in the following days.

Imaging methods are not indicated in patients in whom MRI has already been performed as part of the diagnosis of an epileptic syndrome, in patients where the cause of SE is obvious (low levels of anticonvulsants, acute infection)

EEG

- All patients with the development of SE need an EEG examination, but treatment cannot be delayed while waiting for the EEG results. 30–60 min. a persistent seizure requires an EEG as soon as possible.
- EEG helps to differentiate between convulsive and nonconvulsive states, differentiates pseudoconvulsions (nonepileptic or psychogenic). EEG helps to differentiate the status of absences from the status of complex focal seizures (generalized spike-wave discharges are evidence for the former, focal spikes or sharp waves with generalization for the latter)
- EEG also helps in differentiating conditions unresponsive to treatment (renal and liver failure, hypoxic encephalopathy). It is indicated in patients requiring continuous administration of barbiturates (thiopental) or benzodiazepines. Patients in whom the disorder of consciousness persists after a seizure require an EEG to rule out subclinical SE. EEG can also diagnose the type of seizure and help indicate the most appropriate ongoing therapy. So, for example the clinically manifest tonic-clonic seizure, which resulted from secondary generalization, could have been preceded by a subclinical partial seizure that required specific treatment (phenytoin, carbamazepine).
- Epileptiform abnormalities on the EEG include **sharp waves, spikes, spike-slow wave combinations, etc.**
- In the case of generalized SE, the mentioned changes are seen over both hemispheres, in the case of focal SE, the changes originate from one region of the cortex. The so-called burst-suppression are characterized by short "bursts" = mixtures of sharp waves and spikes, followed by a long period of relative flattening of the EEG curve and periodically repeating. The EEG isoelectric line represents a low-voltage recording with minimal cortical activity. Both burst-suppressions and isoelectric recording may be present in comatose patients and are usually a poor prognostic sign. On the other hand, the achievement of the burst-suppression formula is the goal when managing a thiopental coma.

Therapy

Pre-hospital care

In the first place, it is necessary to ensure vital functions, i.e. open the airways, apply 100% oxygen. To reduce the risk of aspiration, we place the patient on his side. The introduction of an oral or nasopharyngeal airway will make it easier to maintain the required airway patency. If the patient breathes spontaneously, then we use a half-mask, in case of apnea, a mask with ambuvac. If it is not possible to ensure IV entry, we administer Diazepam 0.5 mg/kg pr (maximum 10 mg) or Midazolam 0.15–0.2 mg/kg im (maximum 5 mg) into the area of the deltoid muscle (fastest absorption is proven from this area). Midazolam has very good absorption even after intramuscular administration, on the contrary, the application of diazepam is now obsolete due to unpredictable resorption from the muscle. Therapy must be timely and aggressive to prevent irreversible damage to the CNS and the development of secondary complications (aspiration, cytotoxic brain edema, insufficient supply of energy substrates to the CNS).

Hospital care

At the beginning, we check the securing of the airways, if necessary we endotracheally intubate. We will ensure monitoring of vital functions - heart and respiratory rate, SaO 2 and blood pressure, we will measure rectal body temperature. During the first 5 minutes, we also provide access and take blood for laboratory tests, at the bedside we determine blood glucose with a glucometer, parameters of acid-base balance and an ionogram. In the case of ongoing convulsions, the impossibility of securing peripheral IV access and the ineffectiveness of the above-mentioned treatment in the pre-hospital area, we introduce intraosseous infusion (in children < 6 years), then CVK in the elderly.

We'll start anticonvulsants right away. Benzodiazepines are the drug of choice in the beginning. At the same time, we start an infusion with an isotonic solution containing glucose (hypoglycemic damage to CNS cells occurs soon during SE from any cause). It is therefore advantageous to provide two peripheral iv lines.

As part of a convulsive state, it is always necessary to prevent hypoxia, prevent hypoglycemia and at the same time adequately deal with convulsions!

Benzodiazepines

- Benzodiazepines are first-line drugs in the treatment of SE, as they have a very rapid onset of action. They have presynaptic, postsynaptic and nonsynaptic effects, the most important being their effect on GABA receptors. Benzodiazepines increase GABA activity and thereby affect the main inhibitory neurotransmitter in the brain. Differences in bioavailability and pharmacokinetics may influence the choice of benzodiazepine preparation.

- Benzodiazepines are most effective in primary generalized epilepsy and partial hemiconvulsions in children without brain lesions, lower efficacy is reported in SE with partial symptomatology and lowest efficacy in SE with tonic convulsions or SE with absences, which may secondarily generalize and thus give the false impression of primary generalized tonic-clonic convulsions.

Glaucoma is a contraindication to their administration. The antidote for benzodiazepines is Flumazenil (Anexate), dose 10 µg/kg iv, ev. repeatedly.

Hydantoinates

Hydantoinates include phenytoin and fosphenytoin (used in the US). These drugs act on the motor cortex where they can inhibit the spread of seizure activity. Contraindications are bradyarrhythmias (SA block, AV block, Adams-Stokes syndrome).

Barbiturates

Barbiturates include thiopental, pentobarbital, and phenobarbital. They have sedative, hypnotic and anticonvulsant effects. Contraindications are acute intermittent porphyria and severe liver damage.

General anesthetics

In the treatment of refractory SE, the associative anesthetic propofol is used among general anesthetics. Its administration in this indication appears in the last 10 years.

Pyridoxine

Pyridoxine (vitamin B6) is a cofactor of glutamic acid decarboxylase and GABA transaminase. These are enzymes that synthesize GABA in the CNS. As already mentioned above, GABA is the main inhibitory neurotransmitter in the CNS and its deficiency predisposes to excitatory activity. Pyridoxine deficiency is very rare and is usually detected as early as neonatal convulsions, but may appear up to 3 years of age. Patients with this disease present with convulsions that are usually refractory to conventional therapy but respond promptly to 100 mg IV pyridoxine.

Causal treatment

If the cause of the convulsions is a metabolic disorder, infection, focal CNS lesion or malformation, then it is necessary to treat the cause as far as possible. As a rule, non-specific anticonvulsant therapy is not effective, convulsions are refractory or recurrent. If hypoglycemia is detected, we administer 2–4 ml/kg of 20% glucose. Some authors recommend that it is advantageous to deliver glucose to the second IV line together with pyridoxine. In the newborn age, we also consider vitamin deficiencies - we give Thiamin 100 mg iv and Pyridoxine 50-100 mg iv

Ionic imbalances can be the cause of recurrent spasms. The most common are hyponatremia and hypocalcemia. Hyponatremia is corrected with hypertonic NaCl solutions until the spasms subside. We must be careful not to induce a pontine myelinolysis syndrome by a sudden change in serum osmolality. Hypocalcemia is corrected with a slow bolus of 10% Calcium-gluconicum or 10% Calcium-chloratum during ECG monitoring, as administration of calcium can induce cardiac arrhythmias (bradycardia). Another cause of convulsions can be hypomagnesemia, most often in the neonatal period. In infants fed cow's milk, calcium and magnesium deficiency must always be ruled out during convulsions. *If convulsions are due to a deficiency of these ions (tetanic convulsions), we administer 10% Calcium Chloratum in 5 ml in children < 6 years and 10 ml in children > 6 years + 10% MgSO 4 in idem doses.

- If the cause of convulsions is hyperthermia, adequate cooling of the patient is essential.

Overview of Anticonvulsants used in the treatment of SE

Medicine	Dosage (initial dose vs. max. single dose)	Application	Onset of effect	Side Effects
Diazepam	0.2–0.4 mg/kg iv, max 5 mg in children < 5 years and 10 mg in children > 5 years	0.1 mg/kg/min	1–3 min.	Sedation, hypotension, respiratory depression, bradycardia, paradoxical hyperreactivity
Phenobarbital	15–20 mg/kg i.v., max. 1 g	1 mg/kg/min	5 min.	Sedation, hypotension, respiratory depression, paradoxical hyperreactivity, immunosuppression
Phenytoin	20 mg/kg i.v., max. 1 g	1 mg/kg/min	15 min.	Dysarthria, ataxia, sedation, hypotension, arrhythmia, thrombophlebitis, "purple glove" syndrome
Valproate	15–20 mg/kg i.v., max. 25 mg/kg	5 mg/kg/min.	? ? ?	Hypotension, arrhythmia, hepatopathy, pancreatitis
Midazolam	0,15–0,2 mg/kg i.v.,max. 5 mg	slow bolus	1–5 min.	Sedation, hypotension, bradycardia, respiratory depression, paradoxical hyperreactivity, apnea, laryngospasm
Propofol	2–5 mg/kg i.v., ev. another scheme	by titration	30–60 sec.	Sedation, hypotension, bradycardia, respiratory depression, bronchospasm, painful application, MAC, sudden cardiopulmonary arrest
Thiopental	2–4 mg/kg i.v.,ev. another scheme	by titration	30–60 sec.	Sedation, hypotension, respiratory depression, accumulation in adipose tissue, necrosis during extravasal or intraarterial penetration
Isofluran	0,5–1% by inhalation			Respiratory depression, hypotension, arrhythmia, malignant hyperthermia, laryngospasm, cough

Timing of anticonvulsant therapy

- Step I = benzodiazepines, 3-15 minutes.

After just 10 minutes of convulsions, the development of cytotoxic brain edema must be expected!

- Step II = phenytoin or phenobarbital, 15-45 minutes. In order, we prefer phenytoin (the advantage is less depression of the respiratory center). However, we change the order of phenytoin and phenobarbital in children younger than 18 months, except for patients with a head injury, where we again prefer phenytoin.
- Step III = general anesthesia, > 45 minutes.

Complications and prognosis

The percentage of patients with epilepsy who will develop SE sometime in their life is in a wide range of 1-10%. Long-term antiepileptic therapy is chosen based on the patient's seizure characteristics and EEG results. Patients with partial seizures respond better to phenytoin, carbamazepine, and phenobarbital (infants). Valproate and phenobarbital are the drugs of choice for patients with generalized tonic-clonic seizures, although carbamazepine and phenytoin are prescribed for patients with secondary generalization.

Complication

- Brain damage – loss of neurons in a prolonged or inadequately controlled seizure (persistent EEG activity);
- Hypoxia;
- Aspiration;
- Metabolic complications: lactate MAC, hypercalemia, hypoglycemia (in a prolonged attack), dehydration;;
- Hyperthermia;
- Cytotoxic rain edema;
- hypotension;
- myoglobinuria (damage to muscles from excessive motor activity during a seizure leads to the release of muscle enzymes and myoglobin, which can clog the renal tubules and result in renal failure);
- craniofacial injury during seizure;
- soft tissue injury;
- pulmonary edema nd cardiac arrhythmias during SE or as complications of therapy;
- DIC – in conjunction with significant leukocytosis and mild pleocytosis in the CSF, SE may resemble sepsis or CNS infection.

SE with generalized tonic-clonic convulsions lasting < 60 minutes has a better prognosis than SE lasting longer. Previously healthy children who have experienced prolonged SE with the necessity of applying general anesthetics do not have a favorable prognosis. Most develop epilepsy and their mental status does not return to normal.

Prolonged SE can lead de novo to the development of hippocampal sclerosis and this is apparently one of the reasons why these patients develop chronic and refractory epilepsy with complex partial seizures.

Links

Source

- HAVRÁNEK, Jiří: *Status epilepticus*.