

Epilepsy (pediatrics)

This article has been translated from WikiSkripta; ready for the **editor's review**.

This article has been translated from WikiSkripta; the **formatting** needs to be checked.

This article has been translated from WikiSkripta; the **translation** needs to be checked.

Epilepsy^{[1][2][3]} is a disease manifested by **recurrent paroxysmal attacks of transient cerebral dysfunction** with pathological electrical activity of nervous tissue caused by excessive discharges of cerebral neurons. The diagnosis of epilepsy is made on the basis of **repeated unprovoked seizures**.

See the Epilepsy page for more information.

The **overall prevalence of epilepsy in children** is around **4%**.

Distribution according to etiology

According to the etiology, epilepsy can be divided as follows:

- primary (idiopathic);
- secondary (symptomatic);
- epilepsy-like conditions.

Primary epilepsy

It is the primary pathology of brain tissue at the cellular level - idiopathic epilepsy. They typically have a positive family history, a negative personal history, normal psychomotor development, a normal neurological finding, a normal intellect, and normal EEG activity between seizures^[3].

Most epileptic seizures between the **ages of 3 and 15** fall into this category.

Secondary epilepsy

They are caused by a pathological formation in the brain tissue that changes its electrical properties and manifests itself as epilepsy only as a symptom. These are congenital malformations of the brain (*cortical dysplasia*), perinatal damage, mesiotemporal sclerosis, strokes, tumors, inflammation, injuries, postoperative scars, vascular anomalies, degenerative diseases and more. They have a rather negative family history, a positive personal history, delayed psychomotor development, decreased intellect, slow basic EEG activity, signs of a brain lesion in imaging methods^[3].

This category includes most seizures between the ages of **0 and 3 years and from the age of 15 throughout adulthood**.

Non-epileptic seizures

In addition to the first signs of epilepsy (triggered by fever, for example), the first seizures in a child may be caused by another disease (often accompanied or caused by fever). The **differential diagnoses** of epilepsy include:

- **simple febrile convulsions**;
- CNS infections: **meningitis, encephalitis**, brain abscess;
- other causes of convulsions (fever, migraine, psychogenic non-epileptic seizures, affective seizures, childhood masturbation, tics, iactatio capitis, syncope, cardiac arrhythmias, hyperventilation tetany, emotional tremor,...).

Simple febrile convulsions are the most common cause of convulsions in children aged 6 months to 6 years with a peak aged 1-2 years. This is a genetic predisposition to fever-induced convulsions, which affects 2-4% of children. The convulsions themselves probably occur in the *prodromal phase* of fever or in the *increment stage* as a result of poor regulation of body temperature increase during the child's development, when heat is no longer generated by brown fat, but the tremor mechanism of heat production is not fully mature and tremor is not sufficiently regulated. Febrile convulsions usually have an insignificant family and personal history. The duration of the seizure is **less than 10 minutes, they are not asymmetric** (= absence of focal neurological finding), they **do not recur during one infection**, the child is asymptomatic after the seizure. ⚠ **If any of these conditions are violated, then these are considered complicated febrile convulsions and the etiology must be searched for (neuroinfections, etc.)**

See the Febrile Convulsions page for more information.

See the Fever page for more information.

Seizure characteristics

If the pathological electrical activity occurs only in one place in the cortex, which is topically matched by the symptoms of the seizure, or at least in one place the pathological electrical activity begins and generalizes only secondarily, we call such seizures **focal** or **partial**. This is 40-60% of seizures in children, most often for genetic reasons, other causes include tumors, heart attack or dysgenesis.

If the pathological activity is based on the deep structures of the brain and the entire cortex is affected at once (the primary focus cannot be determined), we call the attack **generalized**.

Partial seizures

If the pathological activity affects only a small part of the cortex and there is no impairment of consciousness, the seizure is called a **simple partial**. If there is a loss of consciousness, the attack is called a **complex partial**. Partial seizures can spread and, when all areas of the cortex are affected, are then considered to be **secondarily generalized** (eg, *Jackson's* tonic-clonic seizures generalizing proximally from a focal attack on the periphery). Then a certain disorder of consciousness may occur. **Simple partial seizures** preceding complex or secondary generalized seizures are **perceived by the patient as a so-called "aura"**. The first choice in the treatment of partial seizures is carbamazepine or valproate.

Primary generalized seizures

They arise when primarily both hemispheres are affected by pathological electrical activity and typically lead to impaired consciousness^[2]. If there is only a disturbance of consciousness (the patient freezes, does not respond), the seizure is called a **typical absence** (*petit mal*). In a similar manifestation with the inclusion of a motor component (automatic opening and closing of the eyes, etc.), the seizure is referred to as **atypical absence**. In addition, seizures may be **tonic**, **clonic**, **tonic-clonic** (*grand mal*), **atonic** (*astatic*), **myoclonic**, and seizures of **infantile spasms**^[3]. The first choice in the treatment of generalized seizures is valproate.

Epilepsy syndromes

Epileptic syndromes in children include, but are not limited to^[1]:

- **benign neonatal convulsions** - idiopathic epilepsy^[3] with manifestation in the first week after birth; in relation to AD disorder on chromosome 20; good prognosis;
- **West's syndrome** (*infantile spasms*) - symptomatic epilepsy^[3] with manifestation mostly at the age of 3 months to 1 year; inconspicuous several-second contractions of the neck, trunk and upper limb muscles appearing in the *clusters* during the day, on the EEG a typical picture of hypsarrhythmia (large disorder), the etiology can be diverse, often unexplained, most often identified as tuberous sclerosis; the prognosis is poor, the response to treatment is low, most children have mental retardation (the degree depends on the timeliness and success of treatment);
- atonic-akinetic and atonic convulsions - symptomatic epilepsy based on brain abnormalities (often tuberous sclerosis^[1]) with manifestation at the age of 1-3 years; seizures for several seconds with loss of muscle tone, during which the body falls to the ground; the head must be protected with a helmet to prevent injury; seizures are usually on waking or falling asleep, 50 or more per day;
- **Lennox-Gastaut syndrome** - symptomatic epilepsy^[3] based on brain injury or malformation with manifestation within 5 years; various types of seizures including atonic-astatic, partial, atypical absences and generalized tonic-clonic seizures; slowly progressing mental retardation and personality changes; mostly poor response to treatment;
- acquired epileptic aphasia (*Landau-Kleffner syndrome*) - epilepsy with manifestation in children aged 3-7 years and with pathological activity in the speech areas of the cortex; acquired auditor agnosia develops; a child who has already talks loses this ability; there is still much uncertainty about the mechanism of aphasia;
- **benign focal epilepsy** (*Rolandic epilepsy, epilepsy with centrotemporal spikes*) - manifestation in 5-10 years; **very common** (21:100,000, 16% afebrile convulsions in children under 15 years of age^[1]), focal motor spasms of the face and arms, response to treatment good, prognosis good, disappears around 15 years of age;
- **childhood and juvenile absences** - idiopathic epilepsy^[3] with manifestation of childhood absences in children aged 4-12 years and juvenile absences in individuals aged 10-17 years with a possible combination of tonic-clonic seizures; usually children with normal psychomotor development;
- Rasmussen's encephalitis - a rare chronic encephalitis in children aged 6-10 years of unknown etiology with persistent motor spasms; surgical treatment;
- **juvenile myoclonic epilepsy** (*impulsive petit mal, Janz syndrome*) - idiopathic epilepsy^[3] with manifestation at 12-19 years, AD disease on chromosome 6^[1]; upper limb myoclonia of several seconds within 90 minutes of awakening^[1] (eg *brushing teeth*, etc.), occasional absences, later rather generalized tonic-clonic seizures in the morning or evening provoked by photostimulation, lack of sleep and alcohol consumption^[2]; response to valproate treatment good but lifelong treatment^[1];

A special form of epilepsy also occurs, for example, in Angelman syndrome.

Status epilepticus

See the *Status epilepticus (pediatrics)* page for more information.

Status epilepticus is a condition of **ongoing clinical or electrical seizures lasting more than 30 minutes** (regardless of the state of consciousness) or **recurrent seizures without recurrence of consciousness for more than 30 minutes**.

In the literature, a continuous seizure lasting only 20 minutes is reported as a criterion for status epilepticus.^[1]

This condition leads to brain hypoxemia, decreased cortical perfusion and thus irreversible brain damage. The convulsions themselves often resolve on their own, but the pathological electrical activity continues, so anti-epileptic treatment with EEG monitoring is necessary. ⚠️ **This is a condition that requires urgent care.**

Therapy

The treatment protocol can be, for example, the following^{[4][5]}:

- Time 0-10 minutes:
 - determining that this is a possible status epilepticus in development; exclusion of non-epileptic seizures (myoclonus, hysteria), ionic disruption, hypoglycemia;
 - emergency service dispatch report (= time 0 minutes);
 - securing the venous line, if possible;
 - blood pressure and ECG monitoring, airway management (suction, etc.);
 - **diazepam 0.3-0.5 mg/kg** i.v. or rectally (onset of action 7 min^[4]), max. 10 mg (adults 20 mg - according^[4] in two doses after 5 minutes, respiratory monitoring - cave: respiratory depression);
 - transport to the hospital.
- Time 10-20 minutes:
 - continuation of transport;
 - samples (blood count, ions, urea, creatinine, glucose, bilirubin, AST, ALT, GMT, lactate, ABR, antiepileptics);
 - **repeat diazepam** if the condition continues;
 - **phenytoin therapy 15-20 mg/kg i.v.** at a rate of 50 mg/min (synergism with diazepam; do not administer concurrently with glucose as it precipitates out of solution; slow application due to the risk of arrhythmia) until the full dose has been administered or until epileptic status has ended;
 - if the effect of the administered benzodiazepine does not appear, instead of phenytoin - especially in newborns - can be given **phenobarbital 10-20 mg/kg i.v.** (newborns 20 mg/kg, adults 10 mg/kg, max. 700 mg)^[5];
 - after admission to hospital monitoring of blood pressure, saturation, ECG, EEG, oxygen inhalation, consider intubation.
- Time 20-45 minutes:
 - if there is no effect of phenytoin, it is possible to administer **valproate bolus 15 mg/kg and continuously in an infusion of 1-2 mg/kg/hour** (especially in non-convulsive epileptic status)

⚠️ **If the status epilepticus persists in the 45th minute, this is an absolute indication for intubation and conduction of a barbiturate coma.**

- Time 45 minutes
 - barbiturate coma is guided by **thiopental**, **pentobarbital** or **propofol** in an initial bolus of 10 mg/kg and a continuous continuation of 3-5 mg/kg/hr with EEG monitoring;
 - attempts to reduce the dose after 6 hours according to EEG;
 - quality intensive care with the solution of internal complications, therapy of possible intracranial hypertension (dexamethasone, mannitol);
 - if status epilepticus continues after 36 hours, switch to alternative procedures (propofol, ketamine, topiramate by nasogastric tube);
 - transfer to target chronic antiepileptic therapy.

Reference

Related Articles

- Epilepsy
- Status Epilepticus (pediatrics)
- Febrile Convulsions

References

1. MARCDANTE, Karen J, Robert M KLEIGMAN a Richard E JENSON, et al. *Nelson essentials of pediatrics*. 6. vydání. Philadelphia : Saunders/Elsevier, 2011. 831 s. s. 678-683. ISBN 978-1-4377-0643-7.
2. MUNTAU, Ania Carolina. *Pediatric*. 4. vydání. Praha : Grada, 2009. 581 s. s. 497-512. ISBN 978-80-247-2525-3.
3. SÝKORA, Pavol. Epilepsia a epileptické syndrómy v detském věku – diagnostika a léčba. *Neurologie pro praxi* [online]. 2004, roč. 5, vol. 1, s. 30-35, dostupné také z <<http://www.neurologiepropraxi.cz/pdfs/neu/2004/01/08.pdf>>. ISSN 1803-5280.

4. KALINA, Miroslav. Status epilepticus. *Neurologie pro praxi* [online]. 2002, roč. 3, vol. 2, s. 87-93, dostupné také z <<http://www.neurologiepropraxi.cz/artkey/neu-200202-0008.php>>. ISSN 1803-5280.
5. RAMACHANDRANNAIR, Rajesh. *Pediatric Status Epilepticus* [online]. Medscape, [cit. 2013-10-30]. <<https://emedicine.medscape.com/article/908394-overview>>.