Epilepsy

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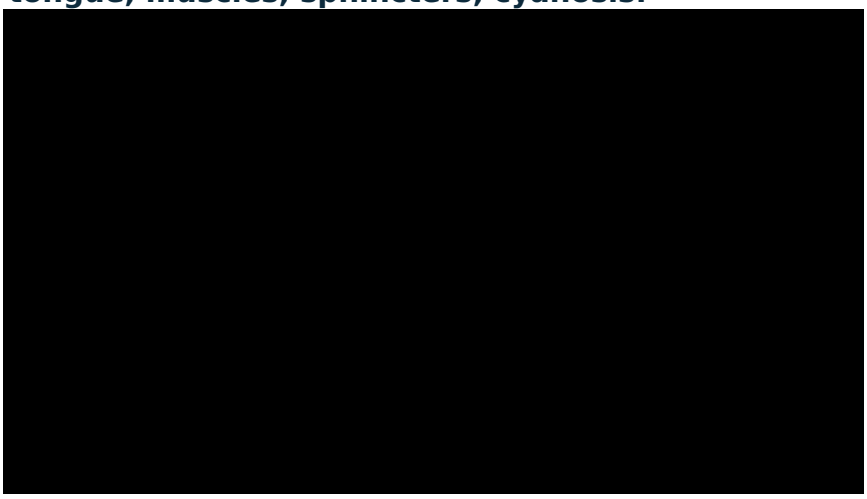
Epileptic seizure, epilepsy, epileptogenesis, status epilepticus:

Ischemia, hypoglycemia, hyponatremia, concussion:

Transient loss of consciousness = TLOC, vasovagal, cardiac:

Post-ictal period, tongue, muscles, sphincters, cyanosis:



Simple & complex partial seizure, generalized, grand mal=tonic-clonic:

Epileptogenesis, penicillin, paroxysmal depolarizing shift, febrile seizures:

By **epilepsy** (EP) we mean recurrent attacks of transient brain dysfunction caused by excessive discharges of brain neurons. Multiple neurons are depolarized simultaneously (partial seizures / generalized), where we can observe loss of consciousness, confusion, convulsions, EEG changes, vegetative symptoms, paraesthesia, psychiatric symptoms[1] and tearing of the corners of the mouth. During all seizures, the perception of the surroundings is lost. The incidence in the population is around 3-5% in adults and 0.5-1% in children.[2]

A recurrence of epileptic seizures is typical, which is sudden and uncontrollable at will. There are episodic changes in brain activity. It manifests itself with a change in behavior, behavior, impaired consciousness and changes in sensorimotor + autonomic functions. The cause of the seizure is a disturbance of the balance between the excitatory and inhibitory mechanisms of a certain group of neurons → abnormal discharges in the CNS. An EP attack can also occur subclinically, with changes in the EEG. A seizure can also be triggered by a certain stimulus[1].

Characteristics of seizures

We recognize several types of seizures. **Partial** (focal, local) **seizures** begin stereotypically in part 1 of the hemisphere, mostly in the cortex. Seizures are simple (without a disorder of consciousness) and complex, where the disorder of consciousness already occurs. If the seizures subsequently generalize, they are called **secondary generalized seizures**. We also have **primarily generalized seizures** that are bilateral from the beginning (when they affect a large area of the brain). Some seizures have a typical EEG image (eg petit mal: tip - wave 3 / s).[1] Before losing consciousness, the affected person acquires an immediate feeling of aura, which can have a variety of characters depending on the location of the epileptic lesion. After an attack, the epileptic may have post-seizure attacks - such as post-seizure hemiparesis or aphasia. These symptoms usually last for several hours, sometimes only a few minutes.

Occurrence, course and prognosis

About 5% of the population will have an EP seizure in their lifetime. Only 0.5% of the population then suffers from recurrent EP seizures. The first seizure occurs by the age of 20 in 75% of epileptics. Today's drugs are effective in 75-90% of epilepsy (see Surgical treatment of epilepsy).[1]

Etiology of epilepsy

In partial seizures, the epileptogenic stimulus causes recurrence of EP seizures. **Etiopathogenetic factors of partial (and secondarily generalized) seizures**:

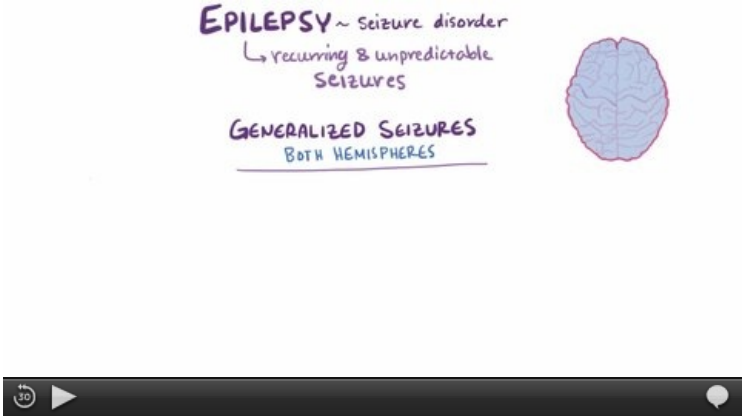
1. **prenatally** - maternal diseases during pregnancy, perinatal hypoxia, ischemia, birth trauma, congenital malformations, genetic disorders
2. **in newborns** - hypocalcemia, hypoglycemia, asphyxia, hyperhydration, congenital metabolic disorders, hyperbilirubinemia, etc.
3. **in infants** - febrile convulsions, CNS infections, congenital defects, etc.
4. **in childhood** - trauma, congenital defects, AV malformations, CNS infections
5. **in adolescence and adulthood** - trauma, CNS tumors, withdrawal symptoms, AV malformations, CNS infections
6. **in late adulthood and old age** - in addition vascular residues, degenerative diseases

An epileptic seizure may be a clinical manifestation of another pathological process, such as a **symptomatic (secondary) seizure** that occurs in cancer, CNS inflammation, trauma, and brain hemorrhage. This form can affect a person at any age, and is more common in old age. Epilepsy that manifests at an advanced age is called **tarda epilepsy**. Another species is **cryptogenic epilepsy**, which presupposes an organic etiology. **Posttraumatic epilepsy** occurs 6 months to 2 years after the injury as a result of traumatic intracranial hemorrhage or an impressive calf fracture. A seizure can also occur immediately after an injury. Amnesia is then

longer than 24 hours. The last mentioned species is **primarily generalized epilepsy**, which is also called idiopathic. It has no proven cause and a genetic predisposition is expected, which manifests itself by the age of 25 at the latest.[1]

Pathogenesis of epilepsy

It is a sudden and transient disorder of nerve cell function. Epilepsy is affected by **damaged neurons** (with uncontrollable activity and increased electrical activity), usually at least partially isolated, and with a reduction in axosomatic inhibitory synapses. (GABA and glycine mediators). The most common are changes in the temporal lobe (temporal lobe epilepsy)[2]. By propagating paroxysmal discharges into the stem structures (ARAS), impulses are projected into both cerebral hemispheres, resulting in a subsequent **loss of consciousness.** An EP attack (**electrophysiologically**) is an uncontrolled, synchronous discharge of a group of ganglion cells in the brain. Clinical manifestations reflect the area of the brain where the shock began. This place is called the epileptic foci or focus. The level of the seizure threshold fluctuates with age. It is lower in childhood, increases in adulthood and decreases again after the age of 60. **Seizure alertness** is individual, genetically determined and subject to the internal and external conditions of the organism[1].



Video in English, definition, pathogenesis, symptoms, complications, treatment.

Epileptic lesion

The basic pathological mechanism is the epileptic foci (focus); it is a different population of neurons with pathological electrical activity. In neurons, resp. in their membranes there is an action depolarization (paroxysmal depolarization shift), which causes hyperexcitability and in the deposit there are abnormal discharges, there are also manifestations of hyperuthythmicity and hypersynchrony. The lesion may be clinically silent for a long time, and when the seizure threshold is exceeded, a seizure with epileptic paroxysm will occur. The nature of the attack itself is always determined by the location of the shock and its spread.

Classification of epileptic seizures

An epileptic seizure is a symptom of epilepsy; can occur in a number of diseases.

- the type of seizures is determined by: anamnesis, observation, EEG
- determination of the type of seizure contributes to the localization of epilepsy in the CNS, we search for the etiology only by imaging methods
- **primary epilepsy** - strong genetic predisposition, no structural, metabolic or pathological abnormality is the basis, there are no deviations from the norm in the period between seizures
- **secondary (symptomatic) epilepsy** - accompanies other CNS diseases, prognosis worse than primary epilepsy[1]

Partial epileptic seizure (focal, focal)

- the location of the epileptic discharge, which may spread to the environment or even generalize; partial seizures are a manifestation of a localized (focal) brain lesion
- always secondary (tumor, inflammation)[1]

Partial simplex seizure

- consciousness preserved, with symptoms:
  - **motor** (tonic / clonic) - 1 segment of limb, Jackson's motor epilepsy, aversive seizures (from gecus praecentralis)
  - **sensory** - pseudohalucinations, illusions, paraesthesia, pain, Jackson's sensitive epilepsy (from the postcentral gyrus and sensory cortex)
  - **autonomic** - TF, DF, nausea, redness, pain

- **psychic** - dreamy states, deja vu, depersonalization (from the limbic system and cortex)[1]

Partial seizure with complex symptomatology

- consciousness impaired: (temporal lobe epilepsy) - aura (unciform crisis, depersonalization, hallucinations / illusions, abdominal aura), seizure (absent in appearance, stereotyped movements)
  - **loss of consciousness** follows a partial simplex attack
  - **loss of consciousness from the beginning**[1]

Partial seizure secondarily generalized

- parc. → to the trunk → loss of consciousness → thalamocortical circuit → to both hemispheres
- prodromes → aura → ictus → postparoxysmal period
  - **partial simplex seizure with secondary generalization**
  - **complex partial seizure with secondary generalization**
  - **partial simplex seizure in complex with secondary generalization**[1]

Generalized seizure

- bilaterally localized seizures, symmetrical without focal onset
- the beginnings of epileptic discharges are localized to the mesodiencephalic reticular formation and project diffusely throughout the brain
- with convulsions (convulsive) / without convulsions (non-convulsive)
- A disorder of consciousness seizure states, without aura, both primary and secondary
- a typical course has three phases: Pre-seizure period - *aura* (may be absent); the *seizure* itself, which lasts several minutes; then the patient falls *asleep*.
  - **absence** (petit mal): "bump" (peeking, twitching of eyelids), fading / redness, EEG: tip / slow wave
    - ***typical absence***
    - ***atypical absence***
  - **myoclonic seizure** - fast muscle. twitches without loss of consciousness, EEG: spike discharge
  - **tonic attack** (West's syndrome) - tonic spasms of the torso and flexion HKK, extension of HD, in children, mental retardation, falls
  - **clonic seizure** (childhood and Janz's juvenile myoclonic epilepsy) - minor freq. twitches than myoclonus, loss of consciousness, children
  - **atonic attack** (asthma attack, Lennox-Gastaut syndrome) - loss of postural muscle tone → sudden fall to the ground, there may not be a disorder of consciousness
  - **tonic-clonic seizure** (grand mal) - loss of consciousness, fall, cyanosis, salivation, pupil areflexia, (1) tonic phase (EEG: high symmetrical spikes) → (2) tonic-clonic phase (↑ HM, TF, tonic contractions and short relaxation, EEG: high slow waves / tip / wave complexes) (3) relaxation phase (muscle weakness and incontinence, EEG: isoelectric line) → awakening and confusion / sleep[1]

Unclassified seizures

Status epilepticus

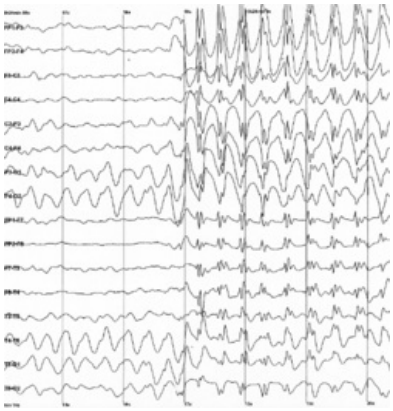
- 90% of uncomplicated generalized seizures last less than 2 minutes, rarely within 5 minutes, ie. as a status epilepticus (SE) it is necessary to treat every seizure lasting 5, maximum 10 minutes. The mortality of generalized tonic-clonic SE is 10-20%.
- impaired consciousness even between seizures → brain damage can occur
- urgent condition! (exhaustion, ↑ TK)[1]
- accompanied by fever, leukocytosis, acidosis and there is a risk of energy depletion and collapse of the body, cerebral hypoxia from respiratory hypoventilation and cerebral edema

Diagnostics

- the most important is the **anamnesis** and objective **description of the seizure**
- clinical findings (sometimes EEG) may be normal among seizures
- we carefully ask about the anamnesis in childhood and before childbirth
- injuries, febrile convulsions
- perceptions + feelings before the attack, circumstances + course of the attack
- the number of last seizures, whether preceded by alcohol abuse, sleep deprivation or another risk factor
- drug use
- **CT** and **MRI** → exclusion of secondaryity
- for monitoring the course of the disease, suitability and success of therapy hl. **EEG**
- **perfusion SPECT** helps to distinguish between primary and secondary epileptic foci
- **PET** is of research importance with the ability to closely monitor regional brain flow and metabolism
- EEG recording is sometimes normal in patients with epilepsy, with the use of activation methods (sleep deprivation) and long-term monitoring, the EEG finding is pathological in more than 90% of cases of epilepsy[1]

Morphological changes of the brain

- **primary epilepsy**: no specific morphological changes, kt. would reliably explain the EP, but in many cases there are changes (dysgenetic foci of the cerebral cortex during intrauterine development, focal scars of the cerebral cortex, loss of neurons monitored by glia proliferation)
- **secondary epilepsy**: various pathological conditions, 150 genetic syndromes are associated with epileptic manifestations[2]



EEG recording of an epileptic seizure in a child

Treatment of epilepsy

Diet

- we ban alcohol, driving, dangerous work, preventing long sleep
- we recommend a ketogenic diet, monophasic sleep, caution in sports
- prerequisite for successful treatment

First aid for epileptic seizures

During an ongoing epileptic seizure, we **prevent injuries** - we dangerously remove objects, support the head, and loosen clothing around the neck. We do not prevent motor manifestations of the attack and we wait for the attack to disappear, it should disappear spontaneously (motor manifestations within 5 minutes, other manifestations within 10 minutes). If the patient does not regain consciousness after the attack, we place him in a stabilized position.

In some cases, it is necessary to arrange transport to the hospital, in particular:

- if it is the first seizure or cumulation of seizures,
- if disorientation persists,
- if there is an injury that requires treatment,
- in the case of *Status epileticus*[3].

Status epilepticus

If the epileptic seizure does not subside within 5 minutes in the case of motor manifestations, or within 10 minutes in the case of a seizure without motor manifestations, or if another seizure occurs without the patient becoming aware, Status epilepticus occurs. He always requires the earliest possible medical care. The goals of treatment are to ensure vital functions, stop the seizure state, clarify its etiology and prevent recurrence - follow-up care[3].

For the treatment of Status epilepticus we administer drugs **intravenously:**

- benzodiazepines: diazepam or the more effective midazolam,
- also antiepileptics: phenytoin, valproate, levetiracetam, phenobarbital, lacosamide[4],
- ineffective thiopental, propofol[3].

Long-term oral treatment of epilepsy

The goal of treatment is to compensate for seizures without unacceptable side effects and ensure the patient's optimal quality of life. In general, most patients respond well to treatment.náhled|200px|Struktura diazepamu

Treatment is **initiated with monotherapy**, the dose is gradually increased until there is a significant reduction / disappearance of seizures, until the maximum doses or signs of drug toxicity are reached. If monotherapy is ineffective, the same procedure is repeated with another drug. Only after another treatment failure (less than 10%) is the combination of more antiepileptic drugs used[5].

The first seizure is usually not a reason to start treatment. Complete treatment of biochemistry and KO should be analyzed prior to treatment. The choice of antiepileptic drugs is determined by the type of attack, more precisely the epileptic syndrome[5].

Focal seizures

1. First choice monotherapy - levetiracetam, lamotrigine
2. Second-line monotherapy - carbamazepine, eslicacrbamazepine acetate, lacosamide, zonisamide, topiramate, valproate
3. Adjunctive therapy - brivaracetam, clobazam, gabapentin, pregabalin[3]

Generalized seizures with tonic-clinical convulsions

1. First choice monotherapy - levetiracetam, lamotrigine
2. Second-line monotherapy - topiramate, valproate
3. Add-on therapy - levetiracetam, pregabalin, zonisamide[3]

Absence

1. First choice monotherapy - etosuximide, lamotrigine, valproate
2. Second-line monotherapy - levetiracetam, topiramate
3. Add-on therapy - zonisamide[3]

Myoclonic seizures

1. First choice monotherapy - levetiracetam, valproate
2. Second choice monotherapy - lamotrigine
3. Add-on therapy - benzodiazepines, levetiracetam, topiramate, zonisamide[3]

The control of the use of the drug is its level in the serum, which also allows individual determination of the dose.[5].

Treatment during pregnancy

Epilepsy is by no means a contraindication to pregnancy, but a serious side effect of some antiepileptic drugs is teratogenicity. Phenytoin, carbamazepine, valproate and phenobarbital are proven teratogens. We do not use these drugs in women of productive age unless necessary. If necessary, an appropriate method of contraception should be provided[6].

For women planning a pregnancy (ideally all of working age), **lamotrigine** or gabapentin is the ideal treatment option for **monotherapy**.[6].

Discontinuation of treatment

We consider the end of treatment at the earliest after 3 years without seizure in EEG without specific EP graphoelements, we slowly decrease with the dose (risk of rebound phenomenon!)[5].

Surgical treatment

Surgical treatment of epilepsy is considered in **drug-resistant epileptics**. According to the International League Against Epilepsy, these are patients who have more than one seizure per month for two years treated with a combination of at least three antiepileptic drugs at therapeutic doses, and the seizures also adversely affect the patient's quality of life. Currently, every epileptic should have an MRI scan to rule out an organic cause of epilepsy, such as low-grade glioma, arteriovenous malformations, cavernous or intertemporal sclerosis.

Examination

An epileptic considered for surgical treatment must undergo:

1. ictal and interictal EEG examination;
2. structural examination (MRI);
3. functional examination (Wada test, PET, SPECT, fMRI);
4. psychiatric and neuropsychological examination.

Operational Performance

The operation is indicated by a neurologist-epileptologist. Performed by:

- Anterior two-thirds **temporal lobectomy** with **amygdalohipocampectomy** (AHE) (70%) - the extent of lobectomy is determined by intraoperative EEG with respect to the functional cortex, it is the basic procedure for intertemporal sclerosis in patients with partially complex seizures.
- Extratemporal resection, most often **topectomy** (20%) - according to the detection of structural topical abnormality on MRI, after temporal resection in 70% of patients there is a complete regression of epilepsy, after extratemporal resection in 60% of patients.
- **Calosectomy, hemispherectomy, vagus nerve stimulation and multiple subpial transections** (10%) - indicated for non-focal seizures.

Differential diagnosis of epilepsy

- **conditions associated with impaired consciousness**: vasovagal syncope, cardiac arrhythmias (Adams-Stokes syndrome), migraine, hypocalcemia, ament conditions, narcolepsy, orthostatic hypotension
- **other paroxysmal diseases**: neuralgia n. V, Menier syndrome
- data on convulsions (especially clonic) increases the suspicion of an EP seizure
- sometimes it is difficult to distinguish a **hysterical (psychogenic) attack** - the patient usually does not injure himself in the fall and directs his fall, lacks mydriasis with pupillary areflexia, there are no positive irritating pyramidal phenomena, lack of blood pressure and heart rate, hysteria and EP often intertwine[1]

History

Early records of epilepsy are estimated from the Babylonian period, in 2080 BC. Since then, it has received many

names, and outside of epilepsy, a term such as epilepsy, morbid sacer, morbus divinus, divine or holy disease may be known to the public. It entered the Books and Dictionaries of Medical Records between 1067 and 1046 BC. However, at the time, it was called the magic of evil spirits, and ointments, fluids, and many other ineffective "drugs" were used to treat it. The first comprehensive view of this brain disease called epilepsy was established in the 4th century BC by Hippocrates. He named various types of seizures and even related symptoms that occurred in animals. Even so, epilepsy was still considered a punishment in the Middle Ages by higher influences, or even by God, it was also called God's disease. It was not until the second half of the 19th century that it was finally established that a brain disorder was responsible for the seizures, and bromides were used for treatment.

Sources

Related articles

- Epilepsie/PGS
- Chirurgická léčba epilepsie
- Klasifikace epileptických záchvatů

External links

- Epilepsie (česká wikipedie)
- Epilepsy (anglická wikipedie)

Source

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