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Solitary Epileptic Seizures in the Clinical Practice

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1. Introduction

An epileptic seizure may be conceptualized as a paroxysmal pathological process in the brain of a heterogeneous etiology with heteromorphic clinical and electrophysiological manifestation.The cases of epileptic seizures are classified according to The International Classification of Epileptic Seizures (ICES) published for the first time by The International League Against Epilepsy (ILAE) in 1970 and revised in 1981 (Commission on Classification and Terminology of the International League Against Epilepsy, 1981). This classification is a clinical one related to semiology of the seizures not to their etiology. Therefore it is necessary to exclude an acutely occurring cause responsible for occurrence of the seizure. In such cases we talk about the so-called acute symptomatic seizures. The underlying cause may be structural (e.g. head trauma), metabolic, toxic (e.g. alcohol), or an acute CNS infection, etc.. The most frequent acute symptomatic seizures are the febrile seizures. In fact, the acute symptomatic seizures occur more frequently than epilepsy ("unprovoked" seizures). The risk of occurrence during one's life is very high - approximately 5% in males and 2.5% in females. If the acutely occurring cause has been withdrawn or cured without a residuum in the form of a brain lesion, the seizures do not recur (Dasheiff, 1987; Fromm, 1987). The antiepileptic medication is necessary for suppressing the seizures in the acute stage but usually there is no need for treatment continuation after the complete cure of the underlying disease. If the acutely occurring cause was not responsible for epileptic seizure we talk about a so-called unprovoked seizure. If the unprovoked epileptic seizure occurs in relation to a preceding neurological insult, the disorders is regarded as secondary to this insult; we call it the late symptomatic epileptic seizure or late symptomatic epilepsy in case of seizure recurrence. A general principle of treatment for the symptomatic (secondary) epileptic seizures has been a primary effort for resolution of the underlying disease that is the etiological factor responsible for the seizures. Given that it is impossible, the antiepileptic treatment in accordance with the treatment guidelines for individual seizure types (together with adherence to right living, behavioural precautions and concomitant solutions of the social and psychological issues) is indicated (Hovorka et al. 2004a; Hovorka et al. 2004b; Ošlejšková, 2007). Approximately 5% of the population experiences one unprovoked epileptic seizure in the lifetime (Forsgren et al., 1996; Hauser et al. 1993). The febrile seizure before the age of 5 occurs in approximately 5% of population (Hauser et al. 1996). Only about 25% of people experiencing the first unprovoked seizure see the doctor and nearly always the seizure is a generalized tonic-clonic one. Most of the people have no

risk factors for the onset of epilepsy, normal neurological examination as well as normal initial EEG (Pedley et al., 1995). The occurrence of the first unprovoked epileptic seizure requires always a thorough evaluation. The risk of misdiagnosis is high as non-epileptic seizures make 20-33% of newly-diagnosed cases.

EEG is an important non-invasive examination method that informs about electrical activity of the brain. It plays an important role in differential diagnostics of seizures. The greatest diagnostic benefit of EEG belongs to diagnosis of epilepsy. Finding of interictal epileptiform graphoelements supports the diagnosis of epilepsy with specificity of 96% (Vojtěch, 2008). In patients who experienced the first unprovoked epileptic seizure 30-40% catchment of specific epileptiform EEG abnormalities after the first EEG examination was most often reported in the literature (King et. al., 1998; Shinnar et al., 1994). Higher catchment was reported in EEG realized within 24 hours from experienced seizure than after 24 hours (51% versus 34%) (King et. al., 1998). Abnormal EEG occurs more frequently in patients with partial seizures than in patients with generalized seizures and in patients with late symptomatic etiology of epilepsy than in patients with idiopathic epilepsy (Shinnar et al., 1994).

Imaging examinations represent one of the basic methods in diagnostics of patients with epileptic seizures. Their development has significantly contributed to accurate diagnostics and classification of epileptic syndromes. It is necessary to realize that these methods can help reveal etiology of seizures and determine etiopathogenetic diagnosis. The fact that each patient after first epileptic seizure must undertake these examination is common and generally accepted. MRI is an advantageous imaging method for CNS. At present this method is the first choice method. MRI can reveal structural lesions and brain anomalies which CT examination, that is less sensitive, cannot (heterotopias, demyelinizations, anomalies of gyrifications, vascular malformations, etc.) (Bořuta et al., 2007). MRI is markedly more advantageous in patients with temporal epilepsy where it is able to express even very tiny structural changes and mesial temporal sclerosis (Carrilho et al., 1994). In addition, in MRI examination patients are not exposed, in contrast to CT, to radiation load. CT advantages involve better availability, relatively low price, possibility to examine non-cooperating patients because the examination takes only several seconds and it is less sensitive to movable artifacts. Moreover, CT has less contraindications comparing to MRI. MRI cannot be realized in patients with metal implants and clips, pacemaker, uncontrollable claustrophobia. Result can be adulterated if the patient does not cooperate.

In the past we also evaluated the findings of various modification of EEG examination and imaging methods in our patients who experienced solitary unprovoked epileptic seizure (Kollar et. al., 2009). We found that catchment of epileptiform manifestations in native EEG in patients who experienced solitary unprovoked epileptic seizure (14.29%) is lower than reported in literature (*King et al., 1998*; Shinnar et al., 1994; *Vojtěch, 2008*). It might be explained by accepted fact of transient incidence of abnormalities in EEG records. That is the reason that transient incidence of epileptiform EEG abnormalities in patients with epilepsy is considered the factor participating on different results of particular studies. High percentage of non-specific (non-epileptiform) abnormal EEG records in our cohort of patients who experienced solitary epileptic seizure was in agreement with literature data (Kollár et al., 2009). The results of our study – see Table 1.

It is very important to realize the limits of EEG examination. Firstly - normal EEG finding does not rule out clinical diagnosis of epilepsy and presence of epileptiform EEG abnormality does not confirm that the patient has epilepsy. Recurrent occurrence of abnormal interictal EEG findings in the group of non-epileptic seizures is also known (Kuba

et al., 2001). That´s why EEG must be recognized as the method that plays a very important role in diagnostics of epilepsy or paroxysmal disorders, however, as the adjuvant examination method its role is limited. In clinically clear epileptic manifestations EEG can confirm, or in specific cases support, clinically clear diagnosis of epilepsy. In clinically absent typical epileptic manifestations high cautiousness is needed in evaluation of the diagnosis (incorrect evaluation or over-evaluation of EEG finding).

	Normal	NFA	NGA	EFA	EGA
EEG (n=84)	41 (48.81 %)	23 (27.38 %)	8 (9.52 %)	8 (9.52 %)	4 (4.77 %)
EEG after SD (n_1=59)	35 (59.33 %)	9 (15.25 %)	6 (10.17 %)	6 (10.17 %)	3 (5.08 %)
LTM-EEG after SD (n_2=46)	35 (76.09 %)	6 (13.04 %)	2 (4.35 %)	2 (4.35 %)	1 (2.17 %)

Table 1. Interictal EEG findings, EEG findings after SD and LTM-EEG after SD in patients who experienced solitary unprovoked epileptic seizure (Kollar et al., 2009). (Abbreviations see in part 5).

Statistical comparing of diagnostic benefits of CT and MRI examinations of the brain in our group of patients confirmed, as in the other works, that MRI examination of the brain in patients who experienced solitary unprovoked epileptic seizure is definitely the first choice method. The results of our evaluation – see Table 2,3.

	UNPROVOKED SOLITARY EPILEPTIC SEIZURES	
	Number of patients	Number/ whole (%)
CT brain	21	
Normal	5	23.81 %
Pathology	16	76.19 %
MRI brain	6	
Normal	2	0.33 %
Pathology	4	0.67 %
Realized CT and MRI	57	
CT normal MRI normal	28	49.12 %
CT normal MRI pathol.	12	21.05 %
CT pathol. MRI pathol./ closer specification CT by MRI examination	17/12	29.83 %
CT pathol. MRI normal	0	0 %

Table 2. Findings of CT and MRI examinations of the brain in patients after solitary unprovoked epileptic seizure (Kollar et al., 2009).

	Benefit
CT of brain (n=57)	5 (8.77 %)
MRI of brain (n=57)	29 (50.88 %)

Table 3. Patients after solitary unprovoked epileptic seizure in which both imaging methods were realized (Kollar et. al., 2009).

Statistical evaluation of benefits of MRI examination comparing to CT examination by binomic test of proportions: p<0.0001 – high significant difference between proportions (Kollar et. al., 2009).

In the past we noticed, by the EEG finding evaluation in our group of outpatients with epilepsy, that only in a small amount of cases the EEG findings corresponded completely with the clinical image of epileptic seizure (Kollar et al., 2010). In relation to the imaging methods and their diagnostic agreement with clinical syptomatology of epileptic seizure and EEG findings, it is interesting that in the study of King et al. (1998), who evaluated the imaging of MRI abnormalities after the first epileptic seizure in 300 members of a group, consisting of both children and adults, they determined that, with patients having clinically diagnosed partial seizure, an epileptogenic lesion on the MRI was identified in 17% of cases. In 50 patients with clinical diagnosis of generalized seizure a structural lesion was identified in only one case and in the case of 49 patients with generalized epileptiform activity on EEG no structural lesion on MRI was identified. These facts covey to us the need to try and establish in our own group of patients the clinical typology of epileptic seizure, EEG findings and results of imaging methods. After this we determined the part of patients with complete diagnostic concordance between clinical image of epileptic seizure and results of auxiliary diagnostic methods (Kollar et al., 2010). The summary of all watched data in the group of patients after solitary epileptic seizure - see Table 4. The evaluation of clinical typology of epileptic seizures and results or realized examination - see Table 5.

The group of patients	n	The clinical type of epileptic seizure (ILAE,1981)										EEG																	CT/MRI		
		1			2					3	MT	EEG					EEG after SD					LTM-EEG after SD							N	P	0
		A	B	C	A	B	C	D	E			N	NNFA	NEFA	EEFA	0	N	NNFA	NEFA	EEFA	0	N	NNFA	NEFA	EEFA	0					
Solit. unprov. EPI	84	5	3	25	/	/	40	8	/	/	/	41	8	23	4	8	34	6	9	3	6	25	35	2	6	1	2	38	45/30	33/33	6/21

Table 4. The summary of all watched data in the group of patients after solitary unprovoked epileptic seizure (Kollar et al., 2010). (Abbreviations see in part 5).

The full diagnostic coincidence between the clinical picture of epileptic seizure, EEG examination (native interictal EEG, or EEG after SD or LTM-EEG after SD) and results of imaging methods (CT or MRI of the brain) we found only in 11 from 84 patients (13,1%)

after solitary epileptic seizure. The receiving diagnosis of unclear seizure status was determined in 57 out of 116 patients (49,14%) dismissed, as mentioned in 10 years' time period, with the diagnosis of solitary epileptic seizure. These percentages, together with a high part of unclear receiving diagnosis (the disturbance of consciousness of unclear etiology) in the patients, who were dismissed from our clinic with diagnosis of solitary epileptic seizure, suggests that the diagnosis of this group of patients is often problematic.

Coincidence	Solitary unprovoked EPI (n=84)
Clinical typology + EEG (EEG after SD, LTM-EEG afterSD)	16 (19,05%)
Clinical typology + CT, MRI	52 (61,90%)
Clinical typology + EEG + CT, MRI	11 (13,10%)

Table 5. The evaluation of clinical symptomatology of epileptic seizures with EEG, CT and MRI findings (Kollar et. al., 2010).

From unclear seizure status, which is accepted on the neurological departments, the more considerable part is made by unepileptic seizure status (Angus-Leppan, 2008; Perrig & Jallon, 2008). The correct diagnosis of seizure disorders require the strict observance of standard diagnostic proceeding (Martiniskova et al., 2009). The necessity are detailed anamnesis, adequate "erudition" of medical doctors working in this part of medicine, the right interpretation of auxiliary diagnostic methods results and in many cases the quality of cooperation between the doctors from other specializations (Bajaček et al., 2010; Hovorka et al., 2007; Kollar et al., 2010). Our results repeat the confirmation that diagnostic of seizure disorders with or without the disturbance of consciousness belong between the more difficult performances in the clinical praxis.

2. Solitary unprovoked epileptic seizure – the risk factors of probable seizure recurrence. To treat or not to treat the patient after the first unprovoked epileptic seizure?

2.1 Introduction

At least 5% of the general population experience one unprovoked epileptic seizure during their life (Forsgren et al., 1996; Hauser et al., 1982; Hauser et al., 1993). This is in contrast with an approximately 3-4% cumulative incidence of epilepsy (at least two unprovoked epileptic seizures) and with an approximately 4% incidence of the acute symptomatic seizures (Hauser et al., 1996). The risk of seizure recurrence after the first epileptic seizure has been shown to be most frequently 30-40% (range 23-71%) (Annegers et al., 1986; Engel & Starkman, 1994; Hauser et al., 1990; Kollar et al., 2006; Mann, 2005). This figure oscillates notably depending on certain risk factors. There are multiple risk factors mentioned in the literature. Berg & Shinnar (1991), referring to already published studies and meta-analyses, stated that multiple factors influence the recurrence risk of epileptic seizures - see Table 6.

Etiology	patients with tumours and inflammatory diseases of CNS have the highest risk of seizure recurrence; patients with focal (structural) lesions of CNS have higher risk of seizure recurrence than patient without focal neurological damage
Type of seizure	patients with partial seizures, particularly associated with Todd's post-ictal paresis, have higher percentage of seizure recurrence
EEG	appearance of the specific epileptiform abnormalities increases the risk
Duration of the follow-up	the risk decreases with time elapsed, the highest risk is in the first six months after the first seizure
Objective neurological examination	"positive finding" is an unfavourable factor (evidence of a structural lesion)
Febrile convulsion	history of febrile convulsions poses a higher risk of seizure recurrence
Family history	family history of epilepsy has been considered to be an unfavourable factor
Antiepileptic treatment	the lower risk of seizure recurrence with treatment initiation after the first seizure
Psychosocial environment	significant for the prognosis of the disease

Table 6. Factors influencing the risk of epileptic seizure recurrence (Berg & Shinnar, 1991).

2.2 Material and methods

We evaluated 116 patients (68 men, 48 women; age range 18-81 years) after a solitary epileptic seizure that had been hospitalized at our neurological department since January 1, 1997 to January 1, 2007. There were 84 patients having experienced an unprovoked seizure and 32 with an acute symptomatic epileptic seizure. The baseline information was obtained using a retrospective analysis of the medical records; eligible patients were contacted by telephone or by sending the questionnaire via the post. We evaluated the likelihood of seizure recurrence in 72 patients. Duration of the follow-up was 2 - 12 years. A certain portion of patients were followed prospectively at our outpatient department, others were monitored by their neurologists. We determined the number of patients in whom the seizure reoccurred, and time period between the first and the second epileptic seizures. We evaluated the following recurrence risk factors: incidence of the febrile convulsions; incidence of epilepsy in patients' relatives; period of the day, when the seizure appeared; objective neurological examination; the clinical type of seizure; EEG findings; aetiology of the seizure and the influence of antiepileptic treatment initiation after the first seizure. The logistic regression was used for the statistical assessment of the data obtained.

2.3 Results

The individual risk factors and their relation to the seizure recurrence after the first unprovoked epileptic seizure are shown in the Table 7.
The summary of the logistic regression:
i.   Patients with the partial epileptic seizure had 5 times higher recurrence risk (OR = 5.12, 95 % CI: 0.79 – 32.89).

		Patients with seizure recurrence after the first epileptic seizure	Patients without seizure recurrence	Patients with seizure recurrence/ patients with seizure recurrence + without recurrence	Statistically significant factor in terms of the seizure recurrence?
Etiology	idiopathic + cryptogenic	12	18	12/30 (40%)	No
	late symptomatic	14	28	14/42 (33.3%)	
Period of the day, when the seizure appears	day-wakeful condition	17	32	17/49 (34.7%)	No
	sleep-arousal	9	14	9/23 (34.7%)	
Clinical type of the seizure	generalized	14	29	14/43 (32.5%)	No
	partial	12	17	12/29 (41.4%)	
EEG findings	normal + non-epileptic abnormality	20	36	20/56 (35.7%)	No
	epileptic abnormality	6	10	6/16 (37.5%)	
Objective neurological examination	normal	19	34	19/53 (35.8%)	No
	pathological	7	12	7/19 (36.8%)	
History of the febrile convulsions	Yes	0	2	0/2 (0%)	
	No	26	34	26/60 (43.3%)	
Family history of epilepsy	Yes	3	2	3/5 (60%)	No
	No	23	64	23/67 (34.3%)	
Antiepileptic treatment initiation after the first unprovoked epileptic seizure	Yes	6	31	6/37 (16.2%)	Yes
	No	20	15	20/35 (66.5%)	
Seizure recurrence after the first epileptic seizure within …	3 months 6 months 1 year 3 years 5 years	17 18 24 25 26		17/72(23.6%) 18/72 (25%) 24/72(33.3%) 25 26	

Table 7. The individual risk factors and their relation to the seizure recurrence after the first unprovoked epileptic seizure in our group of 72 patients.

ii. Patients with epileptiform EEG findings had 5 times higher recurrence risk (OR = 5.84, 95 % CI: 0.98 -34.62).

iii. The antiepileptic treatment initiation after the first seizure seems to be the only statistically significant protective factor as the patients in our group had 7 times lower recurrence risk compared to the patients without medication (OR= 0.13; 95% CI: 0.03 – 0,6).

After the first unprovoked epileptic seizure we recorded the seizure recurrence in 26/72 patients (36.1%); in 24/26 patients (92.3%) the seizure recurred within 12 months after the first unprovoked seizure.

16.22%

□ patients with recurrence after first unprovoked epileptic seizure

■ patients without recurrence

83.78%

a)

57,14%

□ patients with recurrence after first unprovoked epileptic seizure

■ patients without recurrence

42,86%

b)
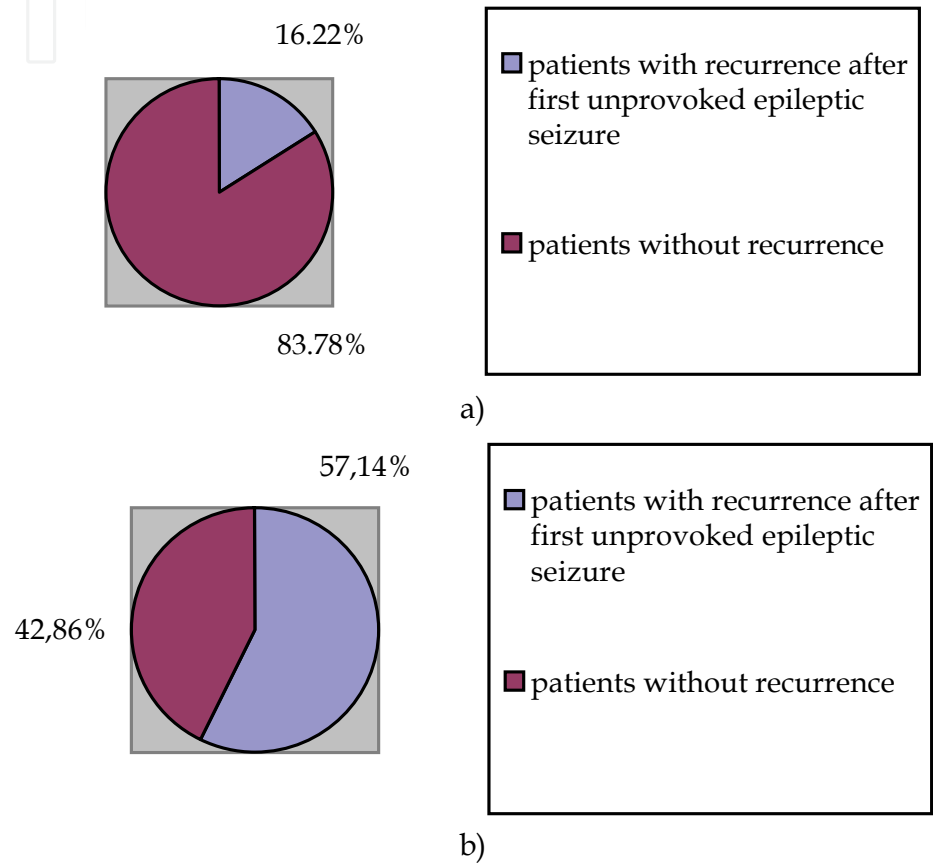
Fig. 1. Comparing the risk for recurrence in the group of patients , in which:
a) an antiepileptic treatment has been prescribed after first unprovoked epileptic seizure (n = 37)
b) an antiepileptic treatment has not been prescribed after first unprovoked epileptic seizure (n=35)

2.4 Discussion

The antiepileptic treatment initiation after the first unprovoked seizure was the only significant factor decreasing the risk of seizure recurrence (7 times lower in our study) (OR = 0.13, 95% CI: 0.03-0.6) in our group of 72 patients. The influence of antiepileptic treatment initiation on the reduction of seizure recurrence has also been reported in earlier studies (Elwes et al., 1985; Kollar et al., 2006). On the contrary, the significance of this factor was not shown in other studies (Bora et al., 1995; Hopkins et al., 1988; Musicco et al., 1997). In our patients there were risk factors of the seizure recurrence showing a clinical, but not statistical, significance – the type of epileptic seizure (5 times higher recurrence risk in

patients after the first partial epileptic seizure: OR = 5.12; 95%, CI: 0.79 – 32.89) and EEG findings (5 times higher recurrence risk in patients with epileptiform EEG findings: OR = 5.84; 95% CI: 0.98 – 34.62). In consistence with the conclusions of Berg & Shinnar (1991) we observed a decrease of the recurrence risk of seizure with time that elapsed since the first seizure. The recurrence risk after the first unprovoked seizure was the highest within 12 months (24/26 patients, 92.3%). The differences in results of the studies evaluating the risk factors of seizure recurrence after the first unprovoked epileptic seizure may be attributed to unequal methods and baseline criteria, as well as to diverse durations of the follow-up. The meta-analyses performed help us orient ourselves in this area (Berg & Shinnar, 1991). In case of the first unprovoked epileptic seizure appearance there is a vital need for a thorough evaluation (Kollar et.al., 2009; Martiniskova et al., 2009).

3. Conclusion

The antiepileptic treatment initiation in patients after the solitary unprovoked epileptic seizure was the only statistically significant factor decreasing the risk of seizure recurrence in our group of patients. Based on the recent knowledge and despite of this finding we propose an individual, rather than automatic, antiepileptic treatment initiation, considering all risks, likelihood of seizure recurrence, social and psychological factors, employment and the potential side effects of the treatment.

4. Acknowledgment

5. Abbreviations

Abbreviations in Table 1.

n = whole number of patients who underwent interictal EEG examination
n_1 = number of patients who underwent EEG after SD
n_2 = number of patients who underwent LTM-EEG after SD
(The numbers of patients are not identical, in same cases of diagnosed epileptic disorder or epileptic focus the whole EEG diagnostic algorithm was not needed.)
NFA = non-epileptiform focal EEG abnormality
NGA = non-epileptiform generalized EEG abnormality
EFA = epileptiform focal EEG abnormality
EGA = epileptiform generalized EEG abnormality

Abbreviations in Table 4.

EEG = native EEG examination
EEG after SD = EEG examination after sleep deprivation with one- hour recording
LTM-EEG after SD = 24-hour eight-channel EEG examination after sleep deprivation
n = number of patients
NFA = non-epileptiform focal EEG abnormality
NGA = non-epileptiform generalized EEG abnormality
EFA = epileptiform focal EEG abnormality
EGA = epileptiform generalized EEG abnormality

N - norm
P - pathology
0 - wasn't realized.
The clinical type of epileptic seizure /ILAE, 1981, being short/:
1 - The partial (focal) seizures:
1A - the simplex partial seizures
1B - the complex partial seizures
1C - the partial seizures with the secondary generalization
2 - The generalized seizures without focal beginning (convulsive or nonconvulsive):
2A - the absence
2B - the myoclonic seizures
2C - the tonic-clonic seizures
2D - the tonic seizures
2E – the atonic seizures
3 - The unclassified epileptic seizures
MT = more types of epileptic seizures

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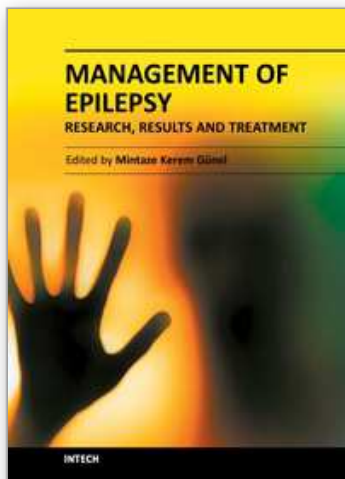
Management of Epilepsy - Research, Results and Treatment

Edited by Prof. Mintaze Kerem GÃ¼nel

Epilepsy is one of the most common neurological disorders, with a prevalence of 4-10/1000. The book contains the practical methods to approaching the classification and diagnosis of epilepsy, and provides information on management. Epilepsy is a comprehensive book which guides the reader through all aspects of epilepsy, both practical and academic, covering all aspects of diagnosis and management of children with epilepsy in a clear, concise, and practical fashion. The book is organized so that it can either be read cover to cover for a comprehensive tutorial or be kept desk side as a reference to the epilepsy. Each chapter introduces a number of related epilepsy and its diagnosis, treatment and co-morbidities supported by examples. Included chapters bring together valuable materials in the form of extended clinical knowledge from practice to clinic features.

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