

Epidemiology of epilepsy

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This article is a brief review of the epidemiology of epilepsy. The article covers the incidence and prevalence of epilepsy, the frequency of epilepsy types, etiology and risk factors for epilepsy, prognosis and factors affecting remission, drug withdrawal, morbidity and mortality of epilepsy, sudden unexplained death in epilepsy (SUDEP), treatment gap and medically intractable epilepsy.

EPILEPSY is a disorder characterized by recurrent seizures of cerebral origin, presenting with episodes of sensory, motor or autonomic phenomenon with or without loss of consciousness. Epilepsy is the second most common chronic neurological condition seen by neurologists. It is estimated that there are 55,00,000 persons with epilepsy in India, 20,00,000 in USA and 3,00,000 in UK. Three to five per cent of the population have a seizure sometime in their life and half to one per cent of the population have 'active epilepsy'.

Incidence, prevalence and mortality studies provide crucial measures of the frequency and therefore the burden of the disease and allow the planning of services. The applications of epidemiological techniques in the field of epilepsy have extended beyond the usual concept of prevalence and incidence¹. The objectives of epidemiological studies also include (1) identification of risk factors for epilepsy and to estimate the effect of potential interventions; (2) to determine overall prognosis for seizure control and the identification of factors which may modify this prognosis; (3) to assess the risk for other conditions in both the patient as well as in relatives and (4) to evaluate interventions, including drug trials.

Difficulties in conducting an epidemiological study of epilepsy

There are immense difficulties in establishing precise epidemiological statistics for a heterogeneous condition like epilepsy². The diagnosis of epilepsy is essentially clinical, based on an eyewitness account of the seizure. Neurological examination and investigations may be normal between attacks. Sometimes patients may not be aware of the nature of attacks; seizures occurring at night may go unnoticed and hence may not be reported. Patients with infrequent or mild seizures may not re-

ceive ongoing medical care and so may be missed in epidemiological surveys. Patients may also tend to deny a history of epilepsy in view of the stigma attached to it or feign epilepsy³. Moreover, the lack of access to EEG or neuroimaging facilities in most community-based studies may lead to inaccurate diagnosis of epilepsy, its type or etiology.

The diagnostic criteria for epilepsy may not be uniform in different studies. Persons with acute symptomatic (provoked) seizures, single unprovoked seizure, febrile seizures and inactive epilepsy may not be excluded in some studies, resulting in higher incidence/prevalence⁴. It is recommended that the guidelines established by the ILAE Commission on Epidemiology and Prognosis be followed in epidemiologic studies of epilepsy⁵. According to these guidelines, a person with active epilepsy is one who has had at least one epileptic seizure in the previous five years, regardless of antiepileptic drug treatment. Epilepsy in remission (no seizures for five or more years with or without treatment) should also be distinguished. Moreover in community surveys, it may be difficult to exclude pseudoseizures and syncope. It is estimated that 10–20% of patients evaluated at centres for epilepsy surgery do not have epilepsy.

Incidence and prevalence rates may vary if age-specific rates are not calculated, since the occurrence of epilepsy differs in different age groups. Assessing the sensitivity and specificity of screening instruments and conducting a validation resurvey improve the accuracy of results⁶. Apart from these methodological factors, it is possible that there may be real differences in incidence or prevalence due to the occurrence of unusual types of epilepsy such as 'hot water epilepsy'⁷ or due to preponderance of central nervous system infections such as cysticercosis, tuberculosis, malaria, HIV, viral encephalitis, etc⁸.

Estimating national figures from local and regional data

A single study may not be representative of a large country such as India. A meta-analysis of studies from various regions may give a more realistic estimate of the national figures and help in planning services for treatment and allocation of resources. We⁶ identified twenty published and unpublished studies^{9–20}, on the prevalence of epilepsy from various parts of India

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Table 1. Studies of prevalence of epilepsy (per 1000) in India

Author, year of study, (ref.)	Population	Persons with epilepsy	Rate	Remarks
<i>Urban/Semiurban</i>				
Surya 1964 (9)	2731	6	2.2	Unpublished
Dube 1970 (9)	20468	46	2.24	Unpublished
Varghese 1973 (9)	1887	8	4.2	Unpublished
Mathai 1969 (10)	16135	121	7.5	DD, SI – Elaborate, AE
Gourie-Devi 1984 (11)	17734	44	2.48	DD, SI – WHO, AE
Bharucha 1985 (12)	14010	50	3.57	DD, SI – WHO, AE
Borah 1992 (17)	8010	80	9.99	DD, < 6 years age excluded
Sohi 1993 (18)	13968	121	8.66	DD, AE
Satishchandra 1995 (20)	51502	296	5.75	RS, SI – WHO, AE
Total (urban)	146445	772	5.27	
<i>Rural studies</i>				
Gopinath 1968 (9)	423	1	2.36	Unpublished
Elnagar 1971 (9)	1383	6	4.2	Unpublished
Sethi 1972 (9)	2691	6	2.2	Unpublished
Nandi 1975 (9)	1060	11	10.4	Unpublished
Carstairs 1976 (9)	1233	13	10.4	Unpublished
Isaac 1980 (9)	4209	37	8.8	Unpublished
ICMR 1983-A – (9)	35548	278	7.82	RS, AE not defined
ICMR-1983-B – (9)	39655	51	1.28	RS, AE not defined
ICMR-1983-C – (9)	34582	59	1.71	RS, AE not defined
ICMR-1983-D – (9)	36595	116	3.17	RS, AE not defined
Mathai 1969 (10)	29643	290	9.78	DD, SI – Elaborate, AE
Gourie-Devi 1984 (11)	39926	223	5.59	RS, SI – WHO, AE
Koul <i>et al.</i> 1986 (13)	63645	157	2.47	DD, SI – WHO, AE
Das <i>et al.</i> 1989 (15)	37286	114	3.06	DD, SI – WHO, AE
Mani 1991 (16)	64936	416	6.41	DD, SI – Elaborate, AE
Kokkat 1993 (19)	8595	48	5.58	DD, AE
Satishchandra 1995 (20)	51055	609	11.93	RS, SI – WHO, AE
Total (rural)	452465	2435	5.38	
Total (rural + urban)	598910	3207	5.35	

DD, door-to-door survey; RS, random survey; SI, screening instrument; AE, active epilepsy; WHO, World Health Organization protocol.

(Table 1). The studies were assessed regarding the methodology and definitions. The studies, which provided details on the age structure, age-specific rates and pattern of epilepsy, were chosen for meta-analysis. Both the crude values, as well as age-standardized prevalence rates after accounting for heterogeneity, were calculated for urban and rural men and women separately, besides overall rates. Age-standardized rates, after correction for heterogeneity due to inter study variation, revealed that the prevalence rates per 1000 (95% confidence interval) were as follows: overall 5.59 (4.15 to 7.03); males 6.05 (3.79 to 8.31); females 5.18 (3.04 to 7.32); urban population: 6.34 (3.43 to 9.25); rural population: 4.94 (3.12 to 6.76).

The differences in prevalence between males and females or urban and rural dwellers were not statistically significant. A subsequent large study from Kerala covering 2,38,102 people has also reported similar prevalence rates²¹.

Prevalence

Prevalence is an estimate of the number of people with epilepsy in a given population at a specified time (point prevalence) or during a defined time interval (period prevalence). Prevalence represents a complex interaction between several factors such as incidence, death or remission of illness. Prevalence studies do not give much information regarding aetiology or prognosis, and are primarily helpful for health planning purposes. In most countries worldwide, the prevalence of active epilepsy ranges from 4 to 10 per thousand population^{6,21-32}. Higher prevalence rates ranging from 14 to 57 per thousand have been reported from some African and South American countries¹. The variations may be partly due to local factors but largely due to differences in methodological approach, which have been highlighted earlier. The prevalence rates from some of the studies from

Table 2. Prevalence of epilepsy – data from some countries

Author, year (ref.)	Country	Prevalence (per 1000)	Remarks
Pond 1960 (22)	UK	6.2	Active epilepsy
Krohn 1961 (23)	Norway	2.3	Active epilepsy
Bird 1962 (24)	South Africa	3.7	
Sato 1964 (25)	Japan	1.5	Active epilepsy
Rose 1973 (26)	USA	18.6	Lifetime prevalence, children only
Juul-Jensen 1983 (26)	Denmark	12.7	Lifetime prevalence
Granieri 1983 (27)	Italy	6.2	Active epilepsy
Li 1985 (28)	China	4.4	Age adjusted
Sridharan 1986 (3)	Libya	1.9	Age adjusted, age > 15 years only
Haerer 1986 (29)	USA	6.7	Active epilepsy
Haerer 1986 (29)	USA	10.4	Lifetime prevalence
Osuntokun 1987 (30)	Nigeria	5.3	Active epilepsy
Hauser 1991 (31)	USA	6.8	Active epilepsy
Hauser 1991 (31)	USA	8.2	Lifetime
Aziz 1997 (32)	Pakistan	9.98	Crude rates
Aziz 1997 (32)	Turkey	7.0	Crude rates
Sridharan 1999 (6)	India	5.59	Meta-analysis active epilepsy
Radhakrishnan 2000 (21)	India	4.7	Age adjusted, active epilepsy

different parts of the world are given in Table 2. Prevalence is fairly uniform in different ages, reaching 6 to 8 persons with epilepsy per 1000 individuals by adolescence, except for an increase in the elderly. The estimated number of persons with epilepsy in India is approximately 5.5 million, among whom approximately 4.1 million reside in rural areas⁶.

Prevalence of epilepsy syndromes

The prevalence of some of the epilepsy syndromes is given below¹: West Syndrome: 1.4–1.9 per 10,000 children, 1–9 per 100,000 population; Lennox–Gastaut Syndrome: 1.3–3 per 10,000 children; 2–6 per 100,000 population; juvenile myoclonic epilepsy: 3 per 10,000 (2.5% of prevalent persons with epilepsy); idiopathic localization-related epilepsy: 2.4–4 per 10,000 population (4–8% of prevalent persons with epilepsy).

Incidence

The incidence of epilepsy is a measure of the number of new persons with epilepsy per 100,000 population per year. Incidence cohorts provide more valuable information than prevalence cohorts, since they include mild and severe persons with epilepsy; persons with epilepsy are identified at an earlier time in the course of their illness and hence more likely to provide reliable information regarding potential antecedents. However, incidence studies require ongoing surveillance of a population of adequate size over a sufficiently long time and hence are very expensive.

The incidence of epilepsy ranges from 40 to 70 per 100,000 in most developed countries and from 100 to 190 per 100,000 in developing countries^{22,23,25–27,33–37}

Table 3. Incidence of epilepsy

Author, year of study (ref.)	Country	Rate (per 100,000)	Remarks
Pond 1960 (22)	UK	70	All seizures
Krohn 1961 (23)	Norway	11	All seizures
Sato 1964 (25)	Japan	17	All seizures
Zielinski 1974 (35)	Poland	26	Excluding SS, FS
Juul-Jensen 1983 (26)	Denmark	39 (men) 28 (women)	Excluding FS
Granieri 1983 (27)	Italy	31	Excluding SS, FS, PS
Placencia 1992 (36)	Ecuador	190	Excluding FS
Hauser 1993 (37)	USA	44	Excluding SS, FS, PS
Mani 1997 (33)	India	49.3	Excluding HWE

FS, febrile seizures; SS, single seizure; PS, provoked seizure; HWE, hot water epilepsy.

(see Table 3). Based on a solitary study³³ which reported an incidence of 49.3 per 100,000, the number of new persons with epilepsy in India each year would be close to half a million. The difference in incidence rates among various studies could largely be related to methodological differences. Incidence peaks in the first few years and in the later years of life, reflecting the multiple etiologies were found at the two extremes. In 50 to 60% of patients, epilepsy begins before the age of 16. In Western countries, the incidence in young children is declining, while incidence in elderly is increasing. The Minnesota study¹ shows stable rates of overall incidence from 1935 to 1979. Differences in incidence rates in males and females are not statistically significant. There is no evidence of racial predilection in incidence, though the incidence is significantly higher in the lower socioeconomic classes.

The cumulative incidence of epilepsy (chance of acquiring epilepsy at some time during life) is 2 to 4%. The chance of having at least one seizure, during a life-

time is approximately 8%. If one includes epilepsy, single seizures, acute symptomatic seizures and febrile seizures, the cumulative incidence is close to 10% by the age of 80 (ref. 34).

Type of epilepsy

In the new cases of epilepsy 50% have seizures of partial origin and 50% of generalized origin before the age of 40. After 40 years, the proportion of partial epilepsy rises to 75% by the age of 75. The figures vary in different studies due to the age of the population, investigations employed, etc. The proportion of incidence cases according to seizure type is shown in Figure 1. The incidence of some of the epilepsy syndromes is given in Table 4.

Etiology of epilepsy

Approximately 60% of all epilepsies are idiopathic or cryptogenic. Almost any type of brain pathology can cause seizures/epilepsy (see Figure 2). Cerebrovascular disease is the most commonly identified cause among adults, while perinatal insults seem to be most common among children. The etiology of seizures is multifactorial in any given individual and is best thought of as an interaction between genetically determined seizure thresholds, underlying predisposing pathologies or metabolic derangements and acute precipitating factors³⁴.

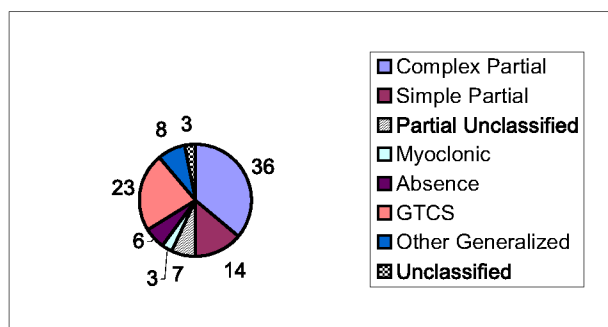


Figure 1. Proportion of incidence cases according to seizure type.

Table 4. Incidence of some of the epilepsy syndromes

Type	Rate (per 100,000)	Percentage of all cases
Idiopathic localization-related	1.7	7
Symptomatic localization-related	13.6	56
Juvenile myoclonic epilepsy	0.2-1.1	1-2.5
GTCS on awakening		1
West syndrome	0.9	1
Absence epilepsy	0.7-4.6	2-3
Lennox-Gastaut	0.9-4.0	1-3.2

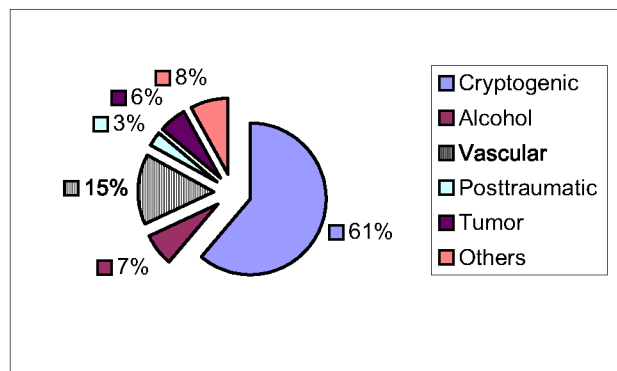


Figure 2. Etiology of newly diagnosed epilepsy.

Risk factors: There are factors such as head injury and infection for which a clear and substantial risk for epilepsy has been established and a direct causal relationship could be assumed¹. The relative altered risk with some of the cerebral insults are given in Figure 3. About 5% of persons with head trauma, cerebrovascular disease and CNS infections have acute symptomatic seizures. The occurrence of acute symptomatic seizures is associated with an additional increase in risk for epilepsy.

Head injury with evidence of brain involvement is associated with a 3-fold increase in risk. The risk increases with severity of injury to 10% in survivors of severe head injury and 50% of survivors with penetrating head injury. The risk is elevated in the first 5 years for minor head injury and in the first 15 to 20 years for severe head injury. Prophylactic AEDs are effective in reducing early seizures, but not late seizures. AED treatment has no effect on death and neurological disability. There is insufficient evidence to establish the net benefit of prophylactic treatment at any time after injury³⁸. Individuals with cerebrovascular disease have a 20-fold increase in risk of developing epilepsy. The risk is elevated in the first 5 years following the first stroke. The risk is greater in persons with intracerebral or subarachnoid haemorrhage compared to cerebral infarction. Survivors of an infection of CNS have a 3-fold higher risk of epilepsy. The risk is 5-fold elevated for bacterial meningitis and 10-fold for viral encephalitis. The risk persists for 2 years after bacterial meningitis and 15 years following viral encephalitis. About 30% of patients with brain tumours present with seizures as an initial symptom.

However brain tumours account for only a small proportion of all epilepsy in epidemiological studies, even in the elderly. Alzheimer's disease is associated with a 10-fold increase in risk for epilepsy. Ten years following diagnosis of Alzheimer's, 10% of survivors will have seizures. Parkinson's disease is considered to be protective against seizures and may be associated with improvement in seizure control in those with epilepsy¹. The risk

of epilepsy is elevated 3.4 times in patients with multiple sclerosis. The risk of epilepsy induced by alcohol increases with increase in daily intake. For those who drink 300 g or more of alcohol daily, the risk is increased more than 20-fold.

With regard to family history, siblings of patients with epilepsy have 2.5 times increased risk for epilepsy, and the risk may be somewhat higher in offspring. There is higher concordance for epilepsy among monozygotic compared to dizygotic twin pairs. Factors such as immunization and perinatal insults have not been proven to have a causal association, despite adequate power. Neurological handicaps at birth such as cerebral palsy and mental retardation are associated with an increased risk for epilepsy, probably due to common antecedents. They should be considered as markers for underlying brain abnormalities, which are responsible for the neurological handicap and for epilepsy.

Two to five per cent of all children below the age of 5 years experience febrile seizures. Atypical febrile seizures are associated with a higher risk for epilepsy. The factors which predict subsequent development of epilepsy among children with febrile seizures include: low Apgar score; neurological abnormalities before first febrile seizure; focal, recurrent or prolonged seizures; postictal paralysis, and family history of epilepsy. Febrile seizures and epilepsy should be considered independent outcomes of a common antecedent. Simple febrile seizures do not carry an increased risk of subsequent epilepsy. Factors such as drug abuse, asthma, hypertension independent of cerebrovascular disease have been found to have an increased risk for epilepsy, the exact reasons for which are not known.

Prognosis of epilepsy

Predictors of remission

Spontaneous or treated: Thirty per cent of patients have mild epilepsy that does not require treatment and remits within a short period; 30% are easily controlled on AEDs; 20% have chronic epilepsy that responds only partially to AEDs and 20% have chronic unremitting epilepsy with little response to treatment³⁹. Epileptic syndromes such as West syndrome, early infantile epileptic encephalopathies, neonatal convulsions, severe myoclonic epilepsy of infancy, epilepsia partialis continua, Lennox–Gastaut, Landau–Kleffner syndrome (acquired epileptic aphasia), progressive myoclonic epilepsy of various types, complex partial seizures, nonconvulsive generalized status epilepticus, epilepsy due to cortical dysplasias have a poor prognosis, while childhood absence seizures, benign epilepsies of childhood (rolandic, benign occipital epilepsy, benign neonatal convulsions), epilepsy in the elderly, febrile

seizures, benign idiopathic neonatal convulsions have a good prognosis³⁴. The prognosis may require to be estimated at the time of first seizure, the time of first diagnosis of epilepsy, the time of achieving seizure control and on attempt at medication withdrawal after seizure control.

Time of first seizure: The risk of recurrence following a single seizure varies from 16 to 81% in various studies. The difference may be largely due to methodological factors. The only prospective study (National General Practice Study of Epilepsy) of this problem reveals that about 67% will experience a second episode by one year and 78% by three years⁴⁰. The risk may vary based on the etiology and the presence of risk factors. The risk is least in persons with acute symptomatic seizures and maximum in those with pre-existing cerebral pathology. The risk factors for seizure recurrence include: antecedent brain insult (most important), age < 16 or > 65 years, seizure type (partial), abnormal EEG (spike wave pattern), family history of epilepsy, prior acute seizures, including febrile seizures, occurrence of status epilepticus or Todd's paralysis³⁵. The chance of recurrence reduces as the duration of seizure freedom after the first seizure increases. Randomized controlled trials have found that treatment with antiepileptic drugs reduces the risk of further seizures by about half. However, there is no evidence that treatment alters long-term prognosis. Long-term antiepileptic drug treatment is potentially harmful⁴¹. The ongoing Multicentre Study of Early Epilepsy and Single Seizures (MESS Study) is likely to throw more light regarding recurrence after a single seizure and treatment of single seizure.

Time of diagnosis: About 54 to 73% of patients achieve remission in various studies (depends on definition of remission and the point at which determination is made). Known etiology, seizure type (partial) and abnormal EEG (spike and wave pattern) are associated with a reduced likelihood for remission¹. Age at diagnosis (extremes of age), number of seizures prior to diagnosis and duration of seizures prior to diagnosis are inversely proportional to likelihood of remission. Certain epilepsy syndromes such as West syndrome or Lennox–Gastaut syndrome have a poorer prognosis compared to others such as Rolandic epilepsy.

Factors during the course: Patients who come under control relatively late and have numerous seizures before being controlled as well as those who require multiple drugs to control their epilepsy, have a worse prognosis for eventual remission.

Predictors of successful/failed withdrawal after remission

Known etiology, abnormal neurological examination, seizure type (multiple or partial), age at onset (> 16

years), use of multiple drugs, number of seizures prior to control, seizures after initiation of AEDs, prolonged interval between onset and seizure control, persistently abnormal EEG, shorter duration of seizure freedom prior to attempted drug withdrawal and the occurrence of GTCS or myoclonic seizures are associated with increased risk of recurrence. The relative risk of seizure recurrence after AED withdrawal is given in Table 5.

MRC trial on AED withdrawal⁴² studied 1013 patients seizure-free for 2 years, who were randomized to continued treatment or slow withdrawal. Continued treatment group showed 22% recurrence after 2 years, while the slow withdrawal group had 41% recurrence. Risk of recurrence declined as the duration of seizure-free period increased, but in a complex manner.

Prognosis for psychosocial outcome

Persons with epilepsy have poor schooling, are unemployed or unmarried compared to matched controls³⁴. The stigma attached to the disease and discrimination in the society add to their misery. In a community survey from Kerala⁴³, 40% of individuals felt that persons with epilepsy cannot be properly educated or employed and 11% would object to their children having contact with children with epilepsy.

Morbidity and mortality

The standardized mortality rates (mortality rate compared with the general population on an age-adjusted basis) in epilepsy are 2 to 4 times higher than normal and are highest in the first 10 years after diagnosis, more so in the first year after diagnosis. Factors associated with a higher mortality include: male gender, extremes of age⁴⁴, marital status (single) and epilepsy symptomatic of diffuse or focal cerebral disease. Much of excess mortality is related to the underlying cause of the epilepsy rather than factors attributable to epilepsy per se. The mortality is higher even in patients with idiopathic epilepsy. The mortality is more with GTCS, but not in those with absence seizures. Mortality is also related to duration of disease and seizure frequency.

Death in epileptic patients may be directly related to seizure/status epilepticus, due to accidents during a seizure

Table 5. Relative risk of seizure recurrence after AED withdrawal

Prognostic variable	RR (95% confidence interval)
Age < 16 years	1.75 (1.30 to 2.36)
Tonic clonic seizures	1.56 (1.09 to 2.22)
Myoclonus	1.84 (1.12 to 3.02)
Use of >1AED	1.85 (1.38 to 2.40)
Seizure after start of AED	1.56 (1.18 to 2.05)
Abnormal EEG	1.32 (1.00 to 1.75)

and drowning, suicide, side effects of antiepileptic drugs such as neoplasia, blood dyscrasia, hepatic failure, toxic epidermal necrolysis or due to Sudden Unexplained Death in Epilepsy (SUDEP)⁴⁵.

Sudden Unexplained Death in Epilepsy

SUDEP is a non-traumatic unwitnessed death occurring in a patient with epilepsy, who has been previously relatively healthy, for which no cause is found even after an autopsy². SUDEP is diagnosed by exclusion. Its frequency is estimated as between 1:200 and 1:1000 persons with epilepsy per year. SUDEP occurs predominantly in men with mean age of 30 to 32 years. One-third die in bed, and alcohol abuse increases the risk. Seizure duration is probably not a risk factor, with more deaths occurring in the first 10 years after onset. Most have secondarily generalized tonic clonic seizures, receive treatment with more than 2 AEDs and have IQ < 70 (ref. 46). The possible mechanisms include: central autonomic over-stimulation producing neurogenic pulmonary edema, cardiac arrhythmia, post-seizure cessation of cerebral electrical activity, suffocation during a seizure, deleterious action of AEDs and release of endogenous opioids.

Accidents during seizure: Persons with epilepsy have an increased risk of drowning (including bath-tub drowning), burns from cook tops, open flames, hot liquids, irons, curling irons, hot baths, showers, etc. Fractures include bilateral or unilateral posterior humeral fracture or dislocations, lumbar vertebral fracture, jaw fracture, facial and skull fracture. Accidents may occur during absence, complex partial, atonic, myoclonic and GTCS.

Treatment gap

This is defined as the percentage of persons with active epilepsy who are not receiving treatment. The treatment gap in underdeveloped countries ranges from 70 to 94%. It is estimated that about 3 million persons with epilepsy living in rural areas of India are not receiving any treatment⁶. The term treatment gap has further been refined. Absolute treatment gap may be used to describe the number of people who are not diagnosed at all and hence the question of therapy does not arise. Surgical treatment gap may be used to measure the proportion of people with intractable epilepsy, who might benefit from surgery, yet do not have it. The reasons for treatment gap include⁴ failure to identify persons with epilepsy; failure to deliver treatment to identified persons with epilepsy; knowledge, attitude and practices (cultural epidemiology) of the people, and the cost of antiepileptic drugs. Misconceptions about epilepsy form

the greatest barrier to treatment of persons with epilepsy. In a study from one of the most literate states of India⁴³, 27% thought that epilepsy is a form of insanity. In many societies, epilepsy is still considered a curse of God and people do not seek help from the doctor. The treatment gap may be narrowed by better identification of persons suffering from epilepsy, better delivery of treatment and education of public.

Pharmacoepidemiology deals with the study of patterns of drug use. It could also be helpful to prevent epilepsy. *Preventive epidemiology* assesses the impact of interventions such as good obstetric care, prevention of CNS infections such as cysticercosis by improving public health measures.

Medically intractable epilepsy

Approximately 20 to 30% of patients with epilepsy have intractable epilepsy and 10 to 50% of medically refractory patients may benefit from surgery. The surgically treatable disorders include mesial temporal sclerosis, benign neoplasms, developmental abnormalities and other focal lesions such as arteriovenous malformations⁴⁷.

Study of the epidemiology of epilepsy is of immense value in understanding the causes, outcome and prevention of epilepsy. It is also useful in planning proper services for persons with epilepsy and improving their quality of life. There is an urgent need for studies regarding incidence of epilepsy, prevalence of epilepsy syndromes, mortality, risk factors and pharmaco-economic data from developing countries such as India. Appropriate measures are also needed to reduce the incidence, ensure adequate treatment of all affected persons, prevent morbidity and mortality associated with epilepsy.

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