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Role of EEG in Epilepsy

Manjari Tripathi and Man Mohan Mehendiratta

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1. Introduction

The human electroencephalogram (EEG) was discovered by the German psychiatrist, Hans Berger, in 1929. Its potential applications in epilepsy rapidly became clear, when Gibbs and colleagues in Boston demonstrated 3 per second spike wave discharge in what was then termed petit mal epilepsy. EEG continues to play a central role in diagnosis and management of patients with seizure disorders—in conjunction with the now remarkable variety of other diagnostic techniques developed over the last 30 or so years – because it is a convenient and relatively inexpensive way to demonstrate the physiological manifestations of abnormal cortical excitability that underlie epilepsy (Smith, 2005) [1].

The electroencephalograph records spontaneous electrical activity generated in cerebral cortex. This activity reflects the electrical currents that flow in the extracellular spaces of the brain, and these reflect the summated effects of innumerable excitatory and inhibitory synaptic potentials upon cortical neurons. This spontaneous activity of cortical neurons is much influenced and synchronized by subcortical structures, particularly the thalamus and high brainstem reticular formation. Afferent impulses from these deep structures are probably responsible for entraining cortical neurons to produce characteristic rhythmic brain wave patterns, such as alpha rhythm and sleep spindles.

The EEG provides confirmation of Hughlings Jackson's concept of epilepsy – that it represents a recurrent, sudden, excessive discharge of cortical neurons; but like other ancillary tests, it must be used in conjunction with clinical data.

2. How can EEG help in epilepsy?

• Diagnosis of epilepsy

- ◦ Differential diagnosis of paroxysmal neurological events

- ◦ Distinction between a focal and generalised seizure disorder

- ◦ Identification of syndrome specific changes

- ◦ Recognition of photosensitivity

- Management of epilepsy

  - ◦ Assessing risk of recurrence after an unprovoked seizure

  - ◦ Selection of antiepileptic treatment

  - ◦ Likelihood of seizure relapse if medication is withdrawn

  - ◦ Identification of epileptogenic region in epilepsy surgery candidates

  - ◦ Investigation of cognitive decline

  - ◦ Detection of non-convulsive status

  - ◦ Monitoring in convulsive status

Although the diagnosis of seizures and epileptic syndromes is primarily made from careful history and examination, the EEG remains an important investigative tool. The EEG often provides supportive evidence of seizure disorder and assists with classification of seizures and epilepsy. Moreover, EEG findings are important for determination of seizure focus and may also help with prognosis under certain circumstances. (Sundaram M et al, 1999) [2]

3. Technical considerations

3.1. Electrodes

The international ten-twenty system of electrode placement, originally proposed in 1958 (Jasper, 1958) [3], is now widely used and is the recommended standard method for recording scalp EEG. The American EEG Society has recently advocated slight modifications to the original alphanumeric nomenclature (American EEG Society) [4] The original T3, T4, T5 and T6 are now referred to as T7, T8, P7 and P8 respectively. This modification allows standardized extension of electrode placement in the sub-temporal region (e.g., F9, T9, P9, F10, T10, P10) and designates named electrode positions in the intermediate coronal lines between the standard coronal lines (e.g., AF7, AF3, FT9, FT7, FC5, FC3, FC1, TP9, TP7, CP5, CP3, CP1, PO7, PO3 and so on). Additional and more closely spaced scalp electrodes placed midway between the standard electrodes of the 10-20 system often provide further localization of epileptiform discharges in patients with partial seizures (Morris et al, 1987) [5]. Several electrodes are available for demonstrating temporal lobe activity. Sphenoidal electrodes are particularly useful for detecting mediobasal temporal discharges and are inserted under the mandibular notch (app. 2.5 to 3 cm anterior to the tragus) and directed posterosuperiorly towards the foramen ovale (Rovit et al, 1961). [6] Anterior "cheek" electrodes (placed on the maxilla

approximately 2 cm anterior to the site of entry of the sphenoidal electrode) and anterior temporal electrodes (placed 1 cm above one third the distance from the external auditory meatus to the external canthus) are also useful for demonstrating epileptiform discharges (ED) from the temporal lobe and the yield appears comparable to that from sphenoidal electrodes (Krauss et al, 1992) [7].
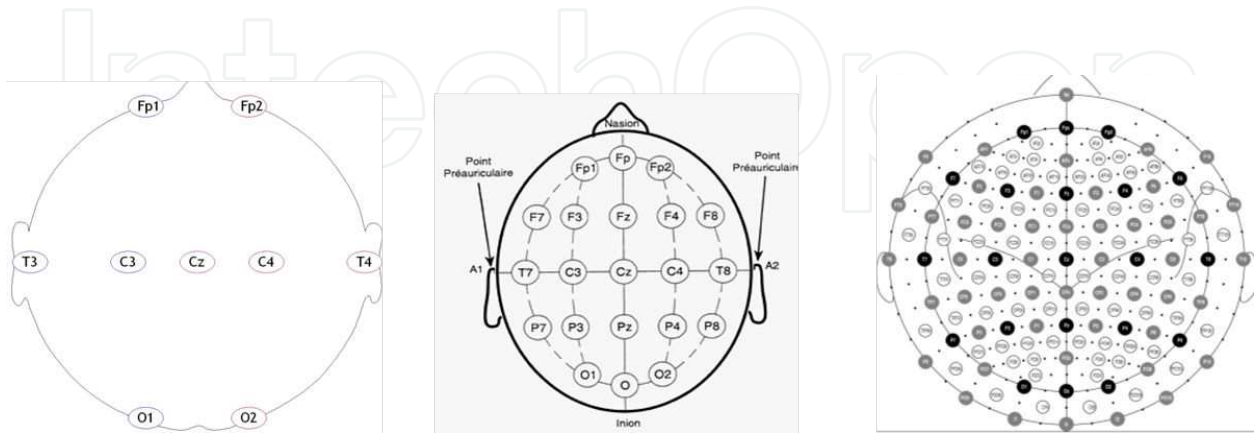

Figure 1. Figure of 10x20 system, 10x10, 10x5; neonatal-10-20, 10-5 placement
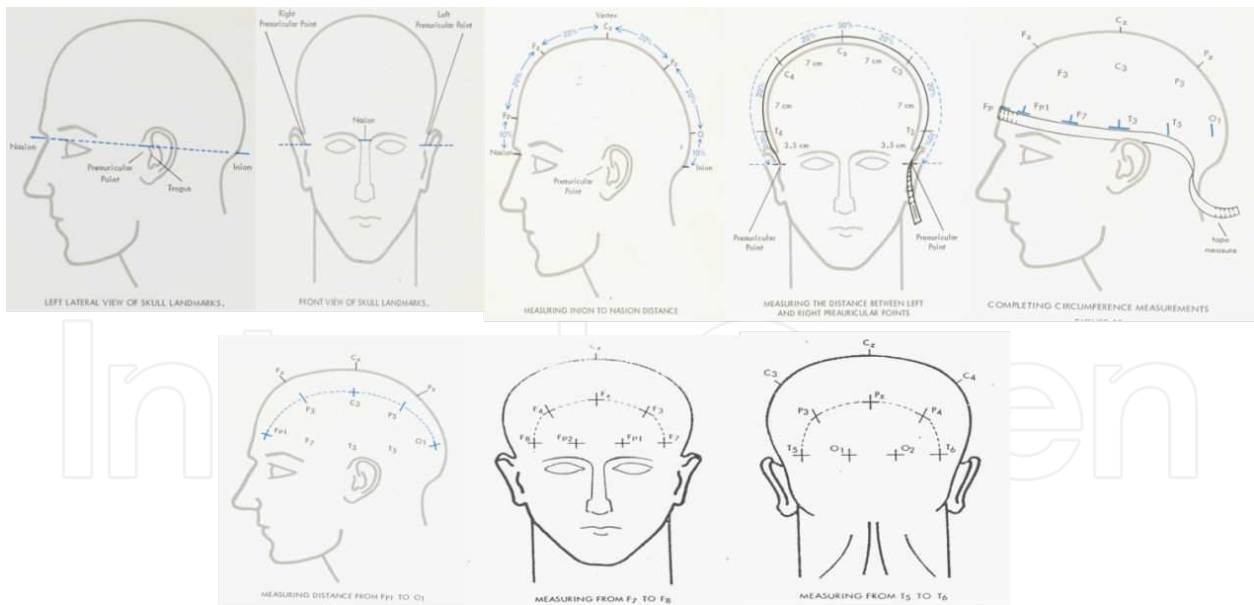

Figure 2. Measurement landmarks10-20 landmarks, measurements, deformity adjustment

3.2. Digital EEG

Digital recording machines are rapidly replacing the traditional "paper" systems.

Advantages:

- Digital EEG is particularly useful for detecting and analyzing ED as the waveforms in question can be reformatted in various montages after the recording is completed.

- Very little storage space requirement, elimination of paper costs, automatic event detection and the ability to network different recording stations.

- Filter and paper speed settings with digital recordings are accurate and automatic, thereby avoiding technician oversight.

- Problems due to pen alignment and curvilinear effect are not seen with digital systems.

Disadvantages:

- The incompatibility of systems made by different vendors, often forcing one to resort to paper printouts for transmission of EEG data between two centers.

- Comparing two separate epochs is somewhat cumbersome, as only limited data can be observed simultaneously on the monitor (Gorney, 1992) [8].

3.3. Activation procedures

3.3.1. Hyperventilation

Forster [9], in 1924, first demonstrated that hyperventilation (HV) may precipitate absence seizures in children and this method of activation has since become routine during EEG recordings. Although HV is particularly useful for demonstrating generalized epileptiform discharges, it may also activate focal epileptiform discharges in up to 10% of patients with partial epilepsies (Miley and Forster, 1977) [10]. The neuronal irritability during HV is considered to be due to brainstem mediated cerebral vasoconstriction induced by hypocapnia.

Hyperventilation should be avoided in patients with potential for brain damage from further vasoconstriction, e.g. malignant hypertension, subarachnoid hemorrhage, sickle cell disease or trait.

3.3.2. Photic stimulation

Photic stimulation (PS) is useful for activation of generalized epileptiform discharges. Testing is generally done with stepwise increase of frequencies up to 30 Hz with a strobe light at a distance of 20 to 30 cm from the eyes. At low frequencies, PS is recommended with eyes open and then closed. At medium and higher frequencies, stimulation should start with the eyes open, and the patient is asked to close the eyes during PS, thereby continuing with PS for a few more seconds with the eyes remaining closed. Eye closure during PS is particularly useful for augmenting ED and should routinely be used. ED outlasting PS strongly suggest general-ized seizure disorder, whereas those confined to the train of PS may be an incidental finding in nonepileptic subjects, especially in the setting of drug withdrawal or toxic metabolic encephalopathy, or simply represent a genetic trait (Newmark and Penry, 1979) [11].

Photic stimulation is particularly useful in primary generalized epilepsy and ED may occur during PS in up to 40% of these patients (Gastaut et al, 1958) [12]. Recent evidence indicates that approximately a quarter to a third of EEGs with photic related ED also contain spontaneous focal or generalized ED elsewhere in the records (Gilliam and Chiappa, 1995) [13].

3.3.3. Sleep Deprivation (SD)

When the first EEG fails to show ED in patients with epilepsy, sleep deprived recording often helps. Several studies have convincingly documented that the chances of finding ED increase with sleep deprived recordings in both partial and generalized seizure patients of all ages (Degan, 1980) [14]. Epileptiform discharges following sleep deprivation occur both in the awake and sleep portions of the EEG. Moreover, Rowan and co-workers (1982) [15] have shown that EEGs following sleep deprivation are more likely to contain ED than the recordings of similar length done following sedation.

4. Clinical Significance of Interictal Epileptiform Discharges (ED)

4.1. ED in nonepileptic subjects

Although the presence of interictal ED generally supports the diagnosis of seizure disorder, caution is necessary in interpreting the clinical significance as ED may occur in subjects without seizures. Among healthy adults without seizure history, the frequency of ED is approximately 0.5% (Robin et al, 1978) [16]. Practically none of these healthy subjects subsequently develops seizures. "Incidental" ED occur slightly more often (app. 2%) in subjects with a history of previous neurological insults such as trauma, stroke, craniotomy, infections, cerebral palsy or during migraine (Zivin et al, 1968) [17]. Up to 14% of these patients subsequently develop seizures. In children without prior seizures, ED may occur in up to 5% and this may be as high as 8% if adequate sleep is recorded (Okubo et al, 1994) [18]; these tend to be benign rolandic or occipital spikes or generalized 3 Hz spike-wave discharges and likely represent incidental genetic trait. Risk of subsequent seizures in these children is around 6% (Cavazzuti et al, 1980) [19]. Certain EEG patterns, however, almost always indicate associated clinical seizures and these include hypsarrhythmia and 1 or 2 Hz generalized slow spike-wave complexes.

4.2. ED in the first and serial EEGs

First standard EEGs in patients with a reasonably certain diagnosis of seizure disorder contain ED in approximately 50% (Ajmone-Marsan et al, 1970) [20]. Yield from the first EEG in children with absence seizures, however, is higher, around 75% (Goodin and Aminoff, 1984) [21]. Apart from sleep, several other factors have been shown to increase the likelihood of ED and these include i) recording within 48 hours of a seizure and ii) ongoing seizure frequency of at least one attack per month (Sundaram et al, 1990) [22]. The yield, however, is not significantly altered by neurological status, etiology of seizures, age of the patient and anti-epileptic drug therapy (Sundaram et al, 1990) [22].

Serial EEGs are often necessary for demonstrating ED. Most patients who eventually show ED do so by the fourth EEG. Recordings are persistently negative in only 8% of epileptics although there is evidence that a higher proportion of patients with partial seizures may have persistently negative serial EEGs (Sundaram et al, 1990) [22].

The above observations suggest that –

i.   the ideal time for obtaining an EEG is the first day or two after a seizure,

ii.   one should consider long-term monitoring if four routine recordings have remained negative in patients with ongoing "seizures".

5. Ictal EEG

While interictal ED generally provides support for the diagnosis of seizure disorder, electrographic or clinical seizures during EEG confirm seizures. The scalp EEG may not reflect all of the ictal activity as this depends on –

i.   the frequency-filtering properties of the skull and scalp,

ii.   the distance and orientation of the focus from the recording electrode and,

iii.   the surface area of the focus with respect to the recording electrode. In spite of these limitations, scalp recorded seizures provide valuable information regarding the seizure type and focus.

5.1. Partial seizures

Partial seizures, in scalp EEGs, are metamorphic, i.e., they show two or more distinct phases (Sharbrough, 1993) [23]. The most common patterns consist of a series of rhythmic waves, sequential spikes/sharp waves, a mixture of spikes and rhythmic waves or regional voltage attenuation. Most often the initial frequency of temporal lobe seizures is in the alpha or theta range with slower frequencies occurring in a lesser proportion (Geiger and Harner, 1978) [24]. Extra temporal seizures, however, often start in the beta frequencies rather than slower frequencies. With scalp EEG, the frequency may diminish or augment, but as the seizure ends, rhythmic waves or sequential spikes change to a slow spike-wave pattern that gradually decreases in frequency. Focal electrodecremental events are of excellent localizing value, reflecting intense neuronal depolarization or high frequency firing (Sharbrough, 1993) [23]. Following metamorphic seizures, there is often postictal delta slowing, suppression or activation of focal spikes. These postictal changes also have good localizing value for seizure origin and should be carefully sought (Kaibara and Blume, 1988) [25].

It is important to recognize that simple partial seizures, especially those with sensory rather than motor symptoms, may not be associated with discernable changes in routine scalp EEG in up to 80% of seizures (Devinsky et al, 1988) [26]. However, the yield in these patients may be augmented by using additional closely spaced electrodes (Bare et al, 1994) [27].

5.2. Generalized seizures

Typical absence seizures are characterized by isomorphic and stereotyped patterns that do not evolve as partial seizures. However, the spike-wave discharges may change from 3.5 or 4 Hz at the onset to 2 or 3 Hz as the seizure progresses. Also, the spike amplitude may decrease during the later part of the seizure. Atypical absence attacks frequently show gradual onset and offset with spike-wave discharges occurring at frequencies less than 3 Hz.

Generalized tonic-clonic seizures may be preceded by diffuse polyspike-wave complexes. Ictal recordings during the tonic phase typically shows generalized attenuation with or without high frequency rhythmic waves that gradually increase in voltage ("epileptic recruiting rhythm") and evolve into polyspikes. The clonic phase is characterized by paroxysmal spike activity mixed with slow waves and the post-ictal period shows generalized attenuation followed by gradual recovery of rhythms (Gastaut et al, 1972) [28].

Myoclonic seizures are associated with 10 to 15 Hz polyspikes with or without slow waves, whereas tonic seizures show generalized paroxysmal fast activity or diffuse voltage attenuation preceded or followed by sharp and slow wave complexes. Generalized atonic seizures may show 2-3 Hz spike-wave discharges or may not be associated with any scalp EEG change.

5.3. PLEDs (Periodic lateralized epileptiform discharges)

Although PLEDs have traditionally been considered "interictal" (Young et al, 1988) [29], there is some evidence that this pattern in some patients may be "ictal" in nature, especially when seen following traditional ictal patterns (Handforth et al, 1994) [30]. Moreover, Reiher and co-workers (1991) [31] reported that the "PLEDs plus pattern", consisting of periodic epileptiform activity closely followed by brief, low amplitude, stereotyped rhythmic discharges, is often associated with clinical seizures and may indeed be a foreteller of imminent seizures. However, whether the PLEDs or PLEDs plus pattern requires aggressive treatment similar to status epilepticus remains unclear (Treiman, 1995) [32].

6. Prognosis of epilepsy

Routine EEG is useful for prognostic purposes in at least three situations:

i. assisting in epilepsy syndrome classification,

ii. predicting recurrence after the first seizure and

iii. providing information on seizure relapse after anticonvulsant withdrawal.

6.1. Classification of epilepsy

The EEG provides important information for classification of various epileptic syndromes and thereby assists in predicting the natural history of the syndrome. For example, a child with

normal neurological examination and rolandic spikes in EEG has a high probability of "outgrowing" seizures and may not even need treatment following isolated, infrequent seizures. Similarly, generalized 4-6 Hz spike-wave and polyspike discharges in an adolescent with seizures suggest juvenile myoclonic epilepsy of Janz: a condition with a high response rate to valproic acid.

6.2. First seizure

Prediction of recurrence after a single seizure is clinically important and many studies have addressed this question. However, differences in methodology make comparison of these studies difficult and the results still remain somewhat controversial. A meta analysis of sixteen published reports suggests that EEG abnormalities may increase the risk of recurrence after first seizure (Berg and Shinnar, 1991) [33].

A recent large prospective study of children with single unprovoked seizure (Shinnar et al, 1994) [34] showed that, in those without obvious etiology ("idiopathic"), the presence of epileptiform discharges in the EEG was associated with a recurrence rate of 54% whereas the rate was only 25% when the first EEG was normal. In the above study, the EEG was not of any predictive value in children with remote symptomatic seizures.

Several recent prospective studies suggest that the EEG is useful in adults with first seizure, especially among those with idiopathic seizures (Van Donselaar et al, 1992) [35]. The Dutch workers (Van Donselaar et al, 1992) [35] showed that when two EEGs (one baseline and one sleep deprived recording) are normal, the recurrence rate was 12% at two years, whereas in those with one or both EEGs containing ED, recurrence rate increased to 83%. The Italian first seizure trial group (1993) [36] also showed a 1.7 fold increase in seizure recurrence when the EEG contained ED. Some controversy still exists in this area as some authors maintain that the EEG findings are of no predictive value after first seizure (Hopkins et al, 1988) [37].

6.3. Anti-epileptic drug withdrawal

The role of EEG in predicting relapses after anti-epileptic drug withdrawal remains more controversial. A recent meta analysis discussing in depth various factors in predicting relapses after anti-epileptic drug withdrawal indicates that any EEG abnormality (epileptiform activity or slowing) is associated with a relative relapse risk of 1.45 (Berg and Shinnar, 1994) [38]. Other factors found to increase the relapse rate in the above meta analysis were adolescent or adult epilepsy onset (rather than childhood onset) and known remote etiology.

7. Video EEG monitoring

Although the EEG remains the gold standard for confirming seizures, an actual attack or event is rare during a standard 20 to 30 minute recording. Even serial EEGs may fail to reveal ED in up to 10% of epileptics (Ajmone-Marsan et al, 1970) [20]. When the nature of attacks or the

exact seizure focus cannot be ascertained with several routine EEGs, telemetry monitoring often provides necessary additional information. With current telemetry systems, EEG data may be collected continuously for several days or even weeks. This may be done as an inpatient procedure using VEEG or at home/work environment with ambulatory EEG. The equipment also has video capability and provides an opportunity to analyze physical changes during the ictus. Most of the equipment available today is highly sophisticated and digitised and portable.

VEEG monitoring is useful:

i.   for confirming the nature of epileptic attacks and nonepileptic events such as pseudoseizure, paroxysmal movement disorders, and sleep disorders

ii.   for determination of seizure focus in patients with atypical features (e.g. frontal lobe seizures, gelastic seizures), and for presurgical evaluation

iii.   for exact classification of seizures prior to appropriate therapy

iv.   for assessing the response to anticonvulsant therapy, and

v.   for research purposes, e.g. analysis of the relationship between the quantity of interictal spikes and clinical seizures, sleep etc.

Video telemetry is generally indicated when visual analysis of physical changes during the event is necessary as in pseudoseizure, frontal lobe seizures, and paroxysmal movement disorders. Ambulatory monitoring without video may be sufficient for confirming the nature of events such as syncope or absence attacks. VEEG is a labour intensive and costly method of investigating patients with difficult to control epilepsy. It involves continuous video and synchronised EEG recording done over many hours usually more than 24 hrs with documentation of at least 3 events or more (specially if discordant). The VEEG is also used in the differential diagnosis of the epilepsy specially when nonepileptic events are suspected. A short term VEEG (3-6 hrs) may be performed in patients where psychogenic non epileptiform events are suspected. It is also useful when the number of episodes are several in a day.

All procedures should be carried out ONLY by trained technicians and Neurologists trained in epilepsy and epilepsy monitoring. Since VEEG does carry a risk a standard operating procedure and manual should be available in all centres carrying this out.

Pearl

A normal EEG does not rule out the diagnosis of epilepsy.

Often the more the number of EEGs more the chance of picking up an abnormality.

The yield inceases with performing an EEG with both sleep and awake state.

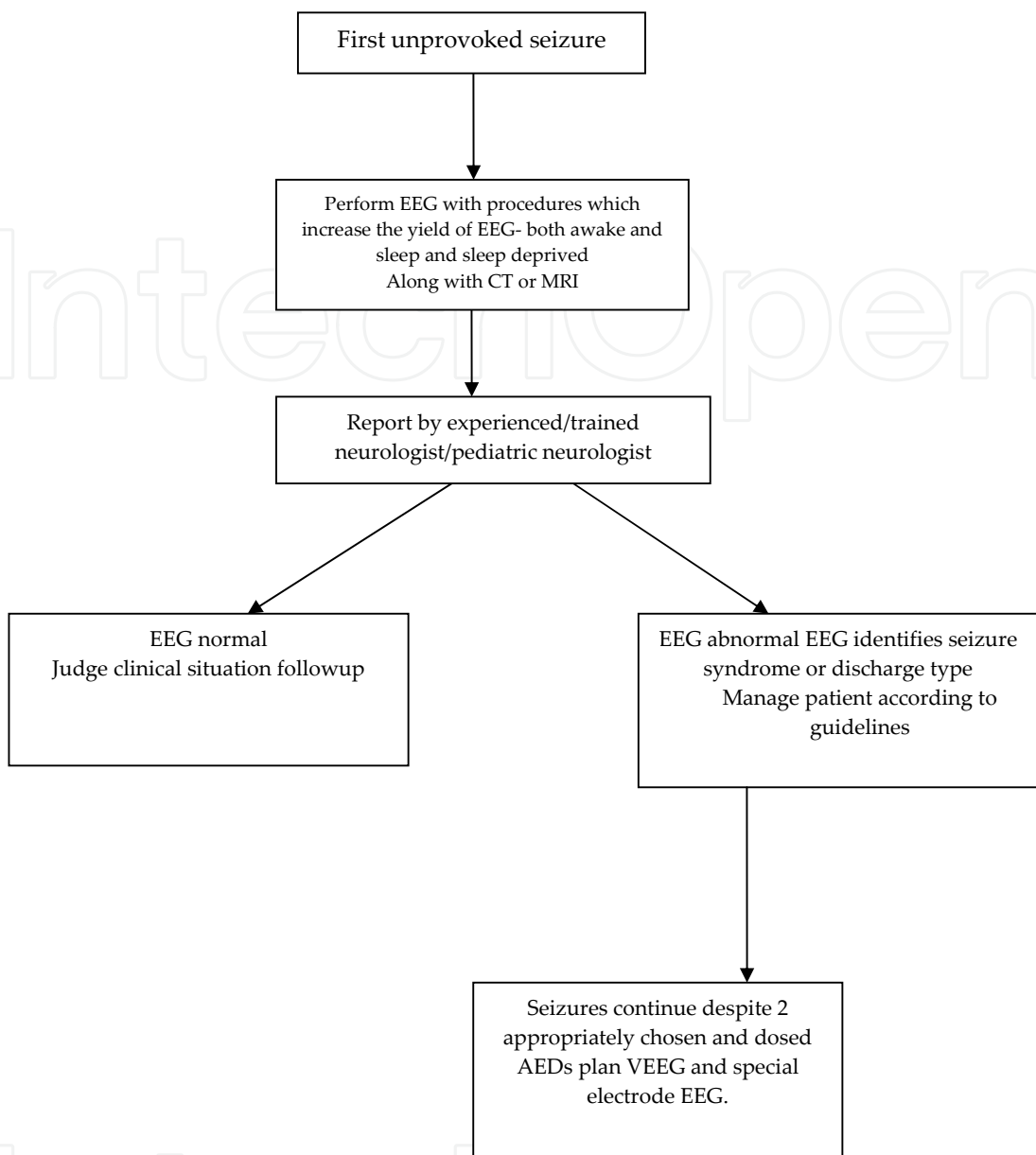An abnormal EEG should be interpreted according to the clinical situation.

Figure 3. Documentation before recording: Patient demographic details such as name, age, clinical diagnosis or indication for EEG, state of the patient, medication details, test number and comments have to be entered.

Author details

Manjari Tripathi[1*] and Man Mohan Mehendiratta[2]

*Address all correspondence to: manjari.tripathi@gmail.com

1 Neurology, Neurosciences Centre, AIIMS, New Delhi, India

2 Neurology, GB Pant Hospital, Delhi, India

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