Case Report

Neonatal EEG- an overview

Rabindran[1], Shasidaran[2]

[1]Dr. Rabindran, Consultant, Neonatologist, Billroth Hospital, Chennai, India, [2]Dr. Shasidaran, Senior Resident, Department of Radiology, S.R.M. Medical College, Chennai, India.

Address for Correspondence: Dr. Rabindran, E- mail: rabindranindia@yahoo.co.in

Abstract

Electro-encephalogram (EEG) is the best non-invasive modality for brain monitoring. As brain continues to develop and mature in neonatal period, EEG of a normal newborn varies from time to time. Wave patterns may be normal at one developmental stage and abnormal at another stage. There are many types of EEG waves like alpha, beta, gamma & delta waves. Burst suppression occurs in very sick neonates following brain damage due to asphyxia & predicts poor prognosis. Isoelectric pattern occurs in severe asphyxia, circulatory collapse, massive intracerebral hemorrhage, severe inborn metabolic deficits, CNS bacterial or viral infections, drug-induced state, hypothermia, postictal recording and in malformations like hydranencephaly or massive hydrocephalus. Grading of severity of disease condition can be done based on EEG. They can be vital in arriving etiological diagnosis. EEG distinguishes between normal paroxysmal movements from epileptic seizures. Nearly 90% of abnormal movements mimiking seizures may be nonepileptic after EEG study. Early recordings, prolonged recordings at different activity states, serial short interval EEGs increase the prognostic value of EEGs. Long-time bedside monitoring of brain function can be done by amplitude-integrated EEG (aEEG). Some rare neonatal epilepsy syndromes have characteristic EEG features. Artifacts mimicking electrical seizures include environmental interference, electrode impedence abnormalities, motion artifacts and endogenous non-cerebral potentials which can be distinguished by Polygraphy. Drugs can alter background activity. EEG is a boon for non-invasive bedside continuous monitoring of the brain. Judicious application of this technique can help in prompt management of various pathologies like seizures , encephalopathies, epilepsy and asphyxia.

Keywords: EEG, neonatal epilepsy, Burst suppression.

Introduction

The best way to monitor brain function is to study the electrical signals produced by brain, the electro-encephalogram (EEG), a voltage signal measured using scalp electrodes [1]. Electrodes are usually placed based on the international 10-20 system. The head is divided into 10- 20% intervals with nasion as the front, inion as the back and preaurical points for side to side direction.

EEG signals are measured using differential amplifiers where the difference between two electrodes is amplified. The frequency content of normal newborn EEG signal is between 0.4-7.5Hz [2].

Because the brain is actively growing and developing, EEG of a normal newborn varies from time to time. Variations from normal age specific EEG waveforms are considered abnormal. Hence wave pattern may be normal at one developmental stage and abnormal at another stage.Unlike adult EEG, the importance of some EEG features in neonatal EEG remains uninterpretated. Moreover some abnormal findings may be transient following brain injury and may be missed unless serial EEG recordings are not done.

Types of EEG activity: There are many types of EEG waves. Delta waves are present widely with frequency between 0.5 – 3.5 Hz. They occur in infants and during deep sleep or anaesthesia. Theta waves occur in parietal and temporal lobes are 3.5 – 7.5 Hz waves occurring in small children and during drowsy state. Alpha waves are 7.5 – 13 Hz waves occurring posteriorly present at awake and resting state. Beta waves are above 13 Hz in frontal and central regions occurring at intense central nervous system (CNS) activation. Beta-delta complexes are slow waves with superim-posed fast frequency activity. They are most charac-teristic of prematurity

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with onset around 29 weeks chronological age and disappears by 38 weeks. They are initially central and later spreads posteriorly. Temporal sharp waves may be either normal or abnormal which can be differentiated by amplitude, duration, occurrence, complexity and polarity. Central positive sharp waves are postiverolandic sharp waves associated with intraventricular haemorrhage and periventricular leukomalacia.

Tracé discontinue is similar to burst suppression (BS), but is normal in premature babies [2]. Trace alternant is alternating active and less active periods seen in healthy neonates beyond 34-36 weeks of gestation during quiet sleep [2]. Burst suppression occurs in very sick neonates following brain damage due to asphyxia & is characterised by low signal (suppression) interrupted by outbursts of higher signals (bursts) [3]. Characteristics of BS pattern like length of burst and suppression intervals, percentage of suppression activity & spectral contents of bursts helps in prognostication [4]. Isoelectric pattern is seen following severe asphyxia, circulatory collapse, massive intracerebral hemorrhage, severe inborn metabolic deficits, CNS bacterial or viral infections, drug-induced state, hypothermia, postictal recording and in malformations like hydranencephaly or massive hydrocephalus.

Maturation of EEG: Maturation of EEG parallels anatomical and physiological brain development. There are developmental age specific waking and sleep patterns particularly during first 6 months of life. Persistence of immature patterns or reappearance of such patterns indicate cerebral dysfunction. Developmental EEG characteristics of premature and term baby are gestation specific. Temporal theta bursts, central beta-delta complexes &occipital slow activity is noted around 29-30 weeks. Temporo-occipital beta-delta complexes & temporal alpha bursts occur around 31-33weeks. Frontal sharp transients & high voltage beta activity occur during 34-35 weeks. Continuous bioccipital delta activity is characteristic for 36-37 weeks. Trace alternant pattern (NREM sleep) is specific for 38-40 weeks.

Grading of severity based on EEG: Mild abnormalities include dysmaturity & excessive sporadic sharp transients.Moderate abnormalities include abnormal or absent sleep–wake cycles, excessive discontinuity, persistent asymmetry and epileptiform abnormalities including seizures. Severe abnormalities include persistent low voltage, BS, and inactive/ isoelectric EEG [5]. Diffuse CNS Injury is characterised by Suppression-burst and Isoelectric wave patterns, internal dyschronism, multifocal sharp waves and absence of wake-sleep cycling. Focal CNS Injury is characterised by persistent focal abnormalities with focal slow waves, sharp waves along with voltage asymmetry.

Etiological Diagnosis: EEG can be vital in arriving etiological diagnosis. Positive rolandic sharp waves signifies underlying intraventricular haemorrhage (IVH) or periventricular leukomalacia, intraparenchymal or subarachnoid bleeding. Periodic laterized epileptiform discharges (PLEDs) is associated with herpes simplex encephalitis and seizure discharges of depressed brain. Early Infantile Epileptic Encephalopathy (EIEE) is characterised by pseudoperiodical suppression-bursts pattern.

West Syndrome (Infantile Spasms) is characterised by hypsarrhythmia along with disorganized and chaotic background activity. Early myoclonic encephalopathy and Ohtahara syndrome are characterized by presence of burst-suppression pattern [6]. Transient EEG burst-suppression is seen in barbiturate anesthesia and hypoxic-ischemic encephalopathy (HIE), while persistent burst-suppression is observed in deep brain tumors, severe congenital metabolic disorders such as non-ketotichyper glycinemia, or extensive brain malfor-mationsuch as hemimegalencephaly [7].

Periodic EEG pattern occur in methylmalonic aminoacidopathy. Comb-like rhythms are patho-gnomonicof maple syrup urine disease. Positive rolandic sharp waves is pathognomonic of IVH, periventricular leukomalacia without haemorrhage, intraparenchymal or subarachnoid bleeding. Positive temporal sharp waves (PTWs) occur in hypoxic-ischemia. Several ictal discharge patterns have been reported in HIE including focal spikes or sharp wave, multifocal spike and sharp wave discharges, prehypsarrhythmic or hypsarrhythmic patterns.

A seizure is an excessive synchronous discharge of neurons within the brain lasting from 10 seconds to 20 minutes. Background patterns correlate significantly with long-term outcome. EEG distinguishes between normal paroxysmal movements like nonconjugate eye movements, sucking movements without associated eye abnormalities and sleep-related myoclonus from epileptic seizures.Nearly 90% of abnormal movements mimiking seizures may be nonepileptic after EEG study

[8]. Early recordings (within first 48 hour of life), prolonged recordings at different activity states, serial short interval EEGs increase the prognostic value of EEGs [9]. Changes in continuity, frequency and amplitude indicate acute stage abnormalities whereas changes in maturity and waves forms indicate chronic stage abnormalities [10]. A normal or mildly abnormal EEG in first 24 hr of life has a positive predictive value of 94% in predicting a normal neurologic outcome following asphyxia [11].

aEEG: Long-time bedside monitoring of brain function can be done by amplitude-integrated EEG (aEEG), which is a filtered version of a two-channel EEG on a compressed time scale [12]. aEEG displays a trend of peak-to-peak amplitude derived from a single channel (P3-P4) of EEG. The signal is filtered and displayed after compression, with a time base of 6 cm/hour. Early aEEG accurately predicts severity of encephalopathy and long-term neurologic outcome [13]. aEEG has moderate sensitivity for detecting seizures which occur as abrupt voltage increase in upper and lower margins of trace, along with band narrowing[14]. Electrocardiogram activity, patient movement, high-frequency oscillator ventilation and electrode placement can lead to artifacts in aEEGs [15].

Classification of aEEG: In aEEG, background activity is classified into 3 groups based on different voltage cut-offs for the median upper margin (UM) and lower margin (LM): Normal (UM > 10 μV, LM > 5 μV), moderately abnormal (UM > 10 μV, LM < 5 μV) and suppressed (UM < 10 μV, LM < 5 μV) [16]. Based on pattern recognition, five major patterns are noted in aEEG: continuous normal voltage (CNV, band 25–10 μV), discontinuous normal voltage (DNV, UM > 10 μV, LM < 5 μV), continuous low voltage (CLV, band ≤ 5 μV), burst suppression (BS), and flat trace (FT,UM < 5 μV) [17]. In term neonates with HIE, CNV and DNV at six hours correlated with good outcome while CLV, BS and FT predicted poor outcome [18]. Abnormal background activity (persistent low voltage, inactive record, unvarying BS pattern) correlates with poor outcome [19]. Simultaneous raw EEG tracing alongside aEEG significantly increases sensitivity and specificity of aEEG in detection of background and seizure activity [20].

Neonatal epilepsy syndrome: Some rare neonatal epilepsy syndromes have characteristic EEG features. Pyridoxine dependency has generalized high amplitude background slowing, multifocal or generalized epileptiform abnormalities [21]. Herpes simplex encephalitis has generalized or asymmetric background slowing with PLEDS [22]. Nonketotic hyperglycinemia has BS pattern [23]. Ohtahara syndrome (Early infantile epileptic encephalopathy or EIEE) has BS in both wakefulness and sleep persisting unchanged for about two weeks. Early myoclonic encephalopathy (EME) has BS seen mainly in sleep [24].

Artifacts: Artifacts mimicking electrical seizures include environmental interference, electrode impedence abnormalities, motion artifacts and endogenous non-cerebral potentials. Polygraphy helps in differentiating awake and sleep states and in recognizing artifacts. Recently signal processing techniques like correlation, spectral analysis, wavelet transform, matching pursuits and time frequency distribution based singular value decomposition are used to detect neonatal seizures [25].

Effect of drugs on neonatal EEG: Drugs can alter background activity. Isoelectric or invariant discontinuous records, prolonged periods of inactivity occurs following a loading dose of phenobarbitone lasting upto an hour [26]. Plasma phenobarbitone levels above 6 mg/dL show significant background suppression [8]. Prolonged immobility due to sedation can cause scalp edema and subsequent artifactual EEG background attenuation [27]. Surfactant causes decrease in cerebral activity and thereby decreases burst rate on aEEG [28].

Conclusion

EEG is a boon for non-invasive bedside continuous monitoring of the brain. It needs expertise for interpreting the age specific changes, acute and chronic pathologies of the developing brain and to rule out artifacts. Judicious application of this technique at appropriate conditions can help in prompt management of various pathologies like seizures, encephalopathies, epilepsy and asphyxia. They are also helpful for prognostication and assessment of therapeutic outcome.

Keywords: EEG, neonatal epilepsy, Burst suppression.

Abbreviations

aEEG: Amplitude- Integrated EEG, **BS:** Burst Suppression **CLV**: Continuous Low Voltagecm-centimetre **CNS:** Central Nervous System **CNV**: Continuous Normal Voltage dL Decilitre **DNV**: Discontinuous Normal Voltage **EEG:** Electro-encephalogram **EIEE:** Early Infantile Epileptic

Encephalopathy. **EME:** Early Myoclonic Encephalopathy **FT:** Flat Trace **HIE:** Hypoxic- Ischemic Encephalopathy Hzhertz **IVH**: Intra-ventricular Haemorrhage **LM:** Lower Margin**mg:** milligram **NREM**: Non Rapid Eye Movement **PLED**: Periodic Laterized Epileptiform Discharge. **PTW**: Positive Temporal Sharp Waves **UM:** Upper Margin

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