

Basic Electrophysiology of the Electroencephalography

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Although neuroimaging techniques and other diagnostic procedures has been developed, electroencephalography (EEG) is still very important for the evaluation of various brain diseases and functional studies of human brain. EEG is formed mainly by spatial and temporal summations of postsynaptic potentials generated from a large population of pyramidal cells that can be considered as a collection of oscillating dipoles. EEG shows continuous rhythmic oscillation depending on sleep-waking state. Alpha rhythms are generated in cortical areas acting as epicenters with local spread, although the precise cellular mechanism is still unknown. It's been known that neurons in the nucleus reticular thalami are the pacemakers of sleep spindle. Alterations in the circuit of the reticular nuclei-thalamocortical relay neuron-cortical neuron are responsible for generalized spike and wave complexes. At the intracellular level, large paroxysmal depolarizing shifts produce focal epileptic spikes. Slow waves of EEG appear to be related to thalamocortical and/or corticothalamic deafferentation. The interpretation of routine EEG requires a well training from a qualified EEG teacher and reading adequate amount of EEG under supervision. Frequent misinterpretations of routine EEG have been observed in both local clinics and general hospitals. The most common findings of normal routine EEG misinterpreted as abnormal are normal variants and artifacts of various sources. There are considerable variations of normal EEG rhythms and pseudoepileptiform discharges. Eyeball movements produce prominent or subtle EEG changes over the frontal regions that are sometimes hard to be differentiated from abnormal slow waves over that region. Systematic approach was described for a good interpretation of routine EEG.

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20~30%

(current sink)

millisecond

field potential

가

가

21

(temporal and spatial summation) 가

가 field potential

가⁵⁻⁷

가

가

6 cm²

가

(<2 msec)

(phase cancellation)

1. (Source of EEG potentials)

Ebersole

20 cm²

가

⁵

가

10¹⁰⁻¹²

(neuron)

(source)

(glial cell)가

1000

field potential

100~300

가

(Voltage - dependent intrinsic oscillation),

2

gap junction

neuron - glia communication

4

⁴, ⁵

⁶

(pacemaker cell)

2.

가

(apical dendrite)

(basal dendrite)

가

(dipole)가

(voltage field)

가

가

(current source)

가 (equivalent dipole source) tangential source가

3 가

가 radial source 가 (Fig. 1A). (tangential source) 가 (Fig. 1C). (Fig. 1B).⁵ (volume conductor) (voltage generator), (basal fore - brain) (rhythm generator).⁹ (solid angle principle). 가 가 10 가 가

3. (Rhythmicity of EEG) (rhythm) 가

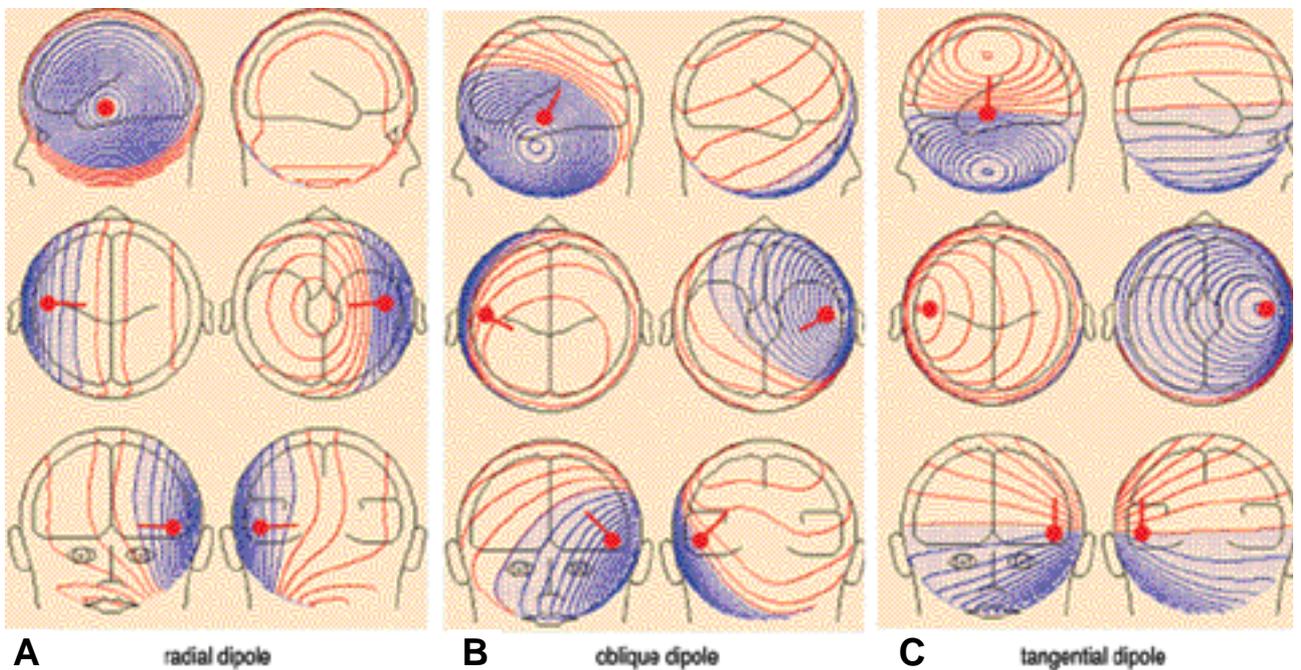


Figure 1. Simulation model of dipole source representing relationship between orientation of dipole source and scalp potential field. Suppose there is a fixed dipole source in left middle temporal gyrus, depicted as a dot indicating negative polarity (blue) and a rod indicating positive polarity (red). In case of radial orientation of a dipole source (A), the maximal negativity of electrical potential is recorded directly above the source. In case of tangential orientation (C), the electrode picking up maximal negative voltage potentials should be inferobasal electrode, such as sphenoidal or T9 electrode rather than midtemporal one. Note maximal positive potential can record in vertex area at a time. As the orientation of dipole source is oblique to anterior aspect (B), inferior frontal and anterior temporal area show maximal negativity.

RE GABA
가
가 가 RE
(thalamocortical neuron, TC)
TC (rebound spike burst)가
가
가
12
1) (synchronization) (desynchro- TC
nization) (whole
1~20 Hz (neuron
brain) (neuronal ensemble) 4) (slow waves of deep sleep)
(neuronal networks) (driving force) (non - REM sleep) 3 - 4
가
(distributed system), RE 가 , TC
(pacemaker) 10 12
forebrain) , (basal
가 (epileptiform
abnormality) (non - epileptiform abnor-
2) (alpha rhythm) (regional), (generalized)
8~13 Hz 가
1. (regional epileptiform discharges)
(cortico -)가 1% ()
cortical pathway) (visual cortex) 가 가
(equivalent dipole) 13
4 - 5 (epicenter) ,
(coherence) 50~200 msec (20~40 mV)
가
(slow action potential) , 1~2
11
3) (sleep spindles) (PDS) paroxysmal depolarization shift
7~14 Hz 가
1.5~2 0.1~0.2 Hz PDS 14
(reticular
thalamic nucleus, RE)

2. (generalized epileptiform discharges) 3~5 Hz(가 200~300 msec 가)

(absence epilepsy) 3 Hz (spike and wave complex, SWC) 3. (regional slow waves)

가

(aberrant thalamocortical rhythm)

TC T-type calcium channel 가
TC RE TC

voltage generator

(cortical activity) 가

RE - TC - corti

cal neuron

3 Hz SWC가

EPSP

가

가

가 cortical deaf -

200~300 msec

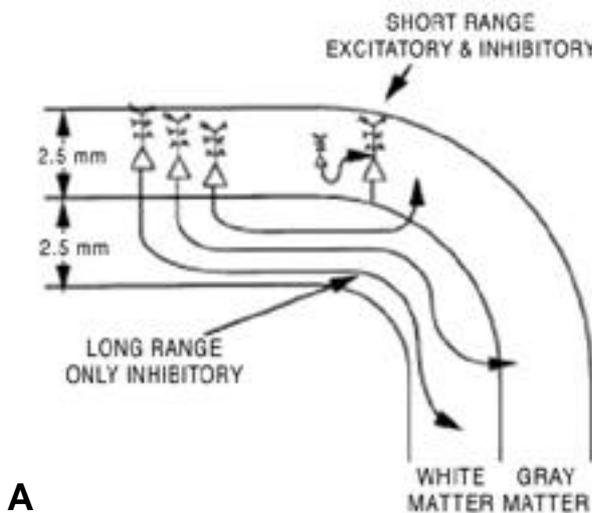
ferentation

SWC

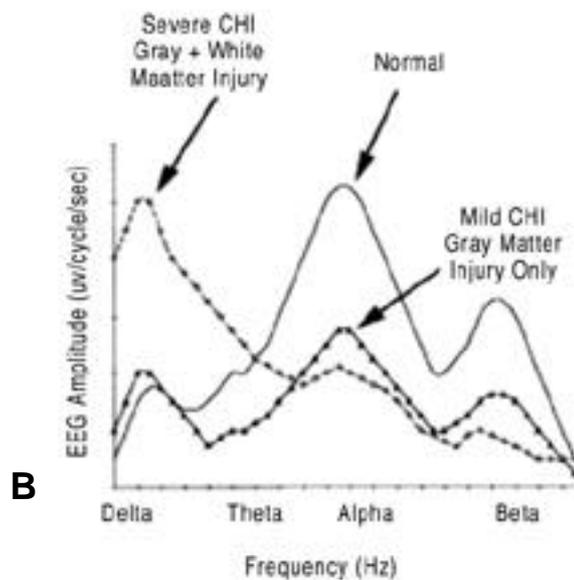
(TC)

1~2 Hz

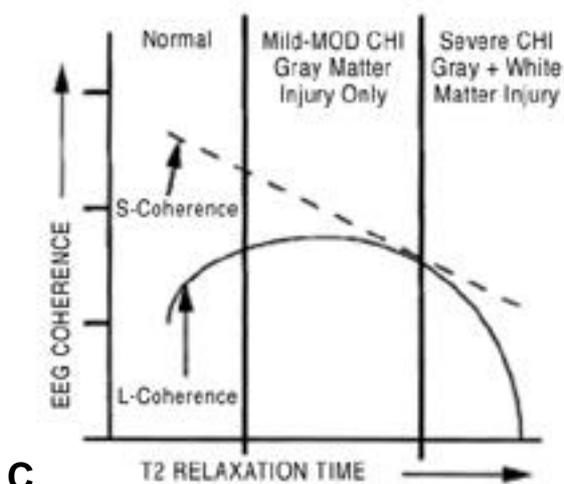
가



A



B



C

Figure 2. Two compartment model of functional connectivity of brain. (A) Two compartment model of cortico-cortical organization are consisting of 1) gray matter or alpha system demonstrating diffusive synaptic dynamic described by the negative exponential and of 2) white matter or beta system representing feedback loop spatial dynamic. The two compartments appear to be dynamically linked and exhibit competitive relationships in which changes in EEG coherence in the two compartments are inversely related. (B) Injury to the gray matter reduces both excitatory and inhibitory synaptic inputs to cortical pyramidal cells, which results in decreased EEG amplitude, esp. in the higher frequencies. Injury to the white matter only reduces the excitatory inputs to the neocortex, resulting in increased delta activity. (C) Increased T2 relaxation time in both the cortical gray and white matter are related to decreased EEG coherence between short interelectrode distance (e.g. 7 cm) and increased EEG coherence between long interelectrode distances (e.g. 28 cm).

가 () - (polymorphic delta activity) -

가 (generalized monomorphic activity)

가 thalamic deafferentation

Thatcher 17-19 (mm cm) (interpretation of routine EEG)

two compartment model (Fig. 2).

(coherence) MRI T2 relax -

ation time (7 (variation) 가

cm) 가 (28 cm) 가

가

4. (generalized slow waves)

1,000

3 가

. 2002 8 가

가 1~2

(left parieto - temporal region) sharp waves (Fig. 3)가

가 (Orfil, Tegretol, Topamax) 가, 가

가 (background rhythm)

가 (psychogenic seizure) 가

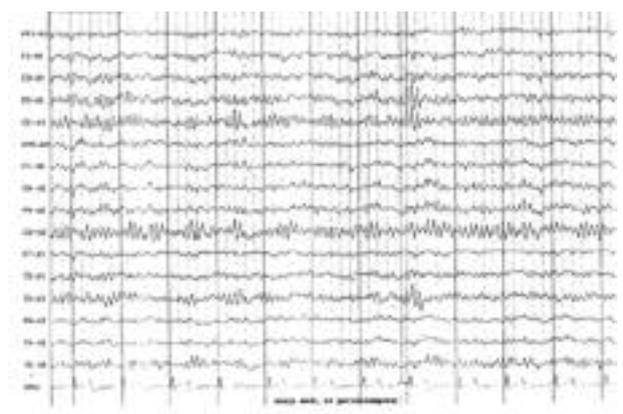


Figure 3. Incorrect interpretation of routine EEG. This EEG is recorded and interpreted as sharp waves (arrow) at left parieto-temporal region in a local neurology clinic. However, when you look into sharp-looking waves indicated by arrow, the rhythmicity, shape and continuity of the underlying background activities are not interrupted. There are no after-going slow waves that occur typically after sharp waves. Thus, these waves are accentuated underlying background rhythms during a little drowsy state, not sharp waves.

가 (Saline induction) 가

가

47 (ictal EEG discharges) 가 (variation) 가

(Fig. 4) 가 (movement artifact) 가 (benign pseudoepileptiform discharges) 가

가 (pseudoseizure) 가 P, QRS, T 가

가 (epileptic seizures) (non - epileptic seizures)

(abnormal EEG) 가 (spikes, sharp waves, slow waves, epileptiform abnormal discharges) (non - epileptiform abnormal discharge)



Figure 4. Incorrect interpretation of routine EEG. This EEG is interpreted as seizure discharges () during routine EEG recording in Neurology department of general hospital. “ A ” of left EEG tracing shows rhythmic EEG discharges with phase reversal at T5, but there were no electrical fields of “ A ” at adjacent electrodes (F7-T5, P3-O1). “ B ” of right EEG tracing shows rhythmic EEG discharges with phase reversal at P3 and C3 (double phase reversal), but there are no electrical field of “ B ” at adjacent electrodes (T5-O1). Paroxysmal EEG discharges recorded at only one scalp electrode and double phase reversal are usually artifacts. The rhythmic EEG discharges in this patient are produced by rhythmic head movement with hyperventilation during routine EEG recording.

가
(capsule)

(internal

1) (waveform)

가 (wave)가

EEG activity

regular rhythmic waves, sinusoidal waves, spindles, irregular arrhythmic waves, complexes, sharp waves, spikes, spike - and - waves, polyspikes, polyspike - and - wave

가

(epileptiform discharges)

(epileptiform discharges)
(tangential dipole)

(spikes) (sharp waves)

가

2.

(systematic interpretation of EEG)

(Fig. 5).

(amplitude), (waveform), (distribution), (frequency), (phase relation), (rhythmicity), (persistence or amount), (timing), (reactivity) 20
9가

가

sharp tran -

sients (Fig. 6).

가 sharp transients

가

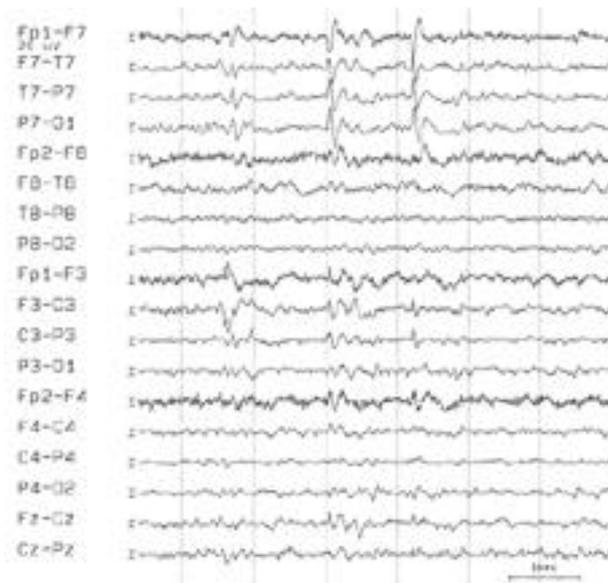


Figure 5. Sharp waves are recorded at left frontotemporal region with a phase reversal at F7 electrode. These sharp waves break down the underlying baseline EEG rhythms and are followed by slow waves.

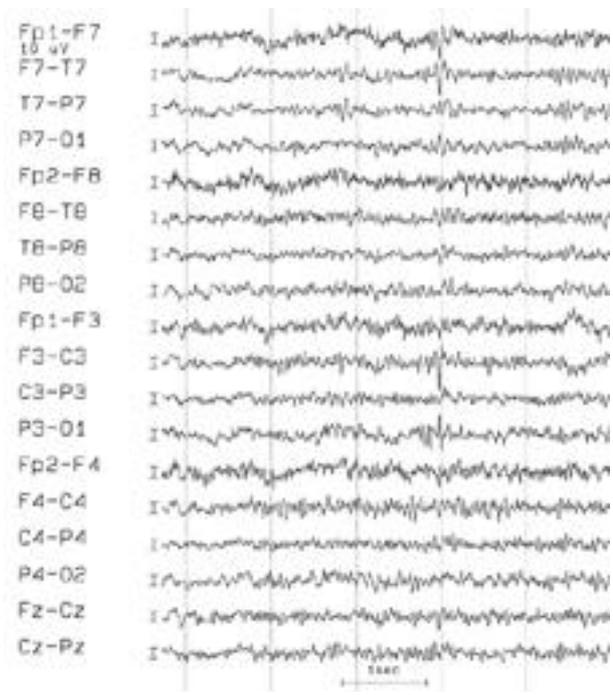


Figure 6. Sharp transient is recorded at left hemisphere (arrow). Although the indicated wave has relatively high amplitude with a sharp wave appearance, it does not break down the underlying baseline rhythm and is not followed by a slow wave.

(epileptic seizures) . 가 . (paroxysmal discharges) 가 (drowsy response) 가 (benign pseudoepileptiform patterns) (Fig. 7).

2) (frequency) 1 . 1 3 cycles 가 3 Hz . 1 (wavelength) 가 250 ms (0.25 sec) 4 Hz가 . “ (alpha rhythm) ” 가 alpha band (8~13 Hz) 가 가 . 가 band 가 (rhythms of alpha frequency) . “ ” posterior

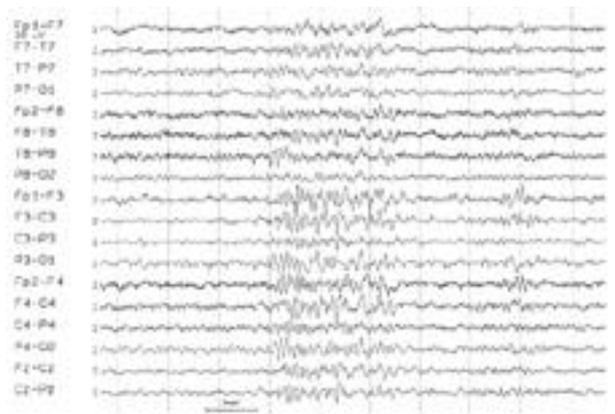


Figure 7. Drowsy response. There are slow eyeball movements and alpha rhythms are slow and poorly formed, and run on and off. This EEG features suggest that the patient is in drowsy state. Paroxysmal medium to high amplitude waves with mixed frequencies recorded diffusely with a parasagittal predominance. Although the shapes and patterns of these paroxysmal discharges are totally different from the underlying baseline rhythms and conspicuous, these waves are normal drowsy response, so-called benign pseudoepileptiform patterns. The paroxysmal drowsy responses should not be interpreted as abnormal discharges.

dominant rhythm occipital rhythm . background slow . 가 . 1 가 5~6 Hz가 , 3 가 8Hz .²¹ 1 가 5 Hz , 3 6 Hz , 5 7 Hz , 8 8 Hz . 1358()~5678() 가 (eye blinking, rapid eye movement, muscle artifact) . (eye blinking) (muscle artifact) 가 가 . (waking, drowsy, light sleep, deep sleep, REM sleep) 가 가 1 Hz 가 가 (Fig. 8).

3) (amplitude) μV . low (under 20 μV), medium (20~70 μV), high (over 70 μV) . low medium

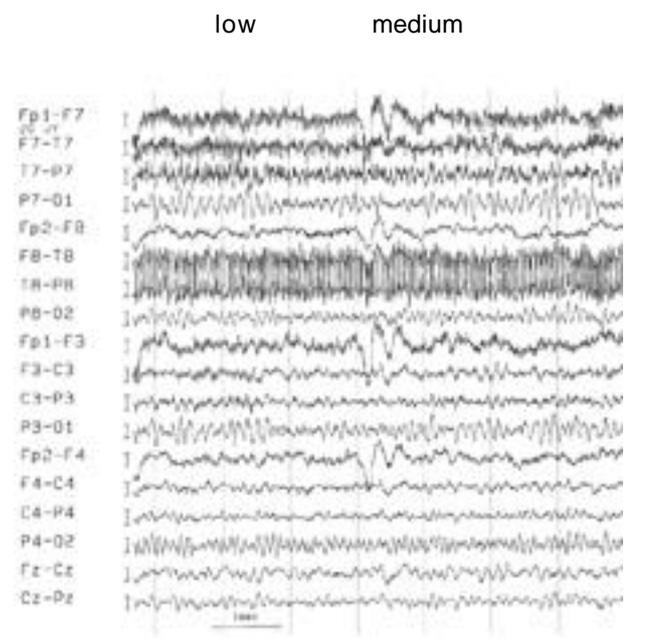


Figure 8. Asymmetry of alpha rhythm frequency. The alpha rhythm frequency of left occiput (7~7.5 Hz) is slower than that of right occiput (9~9.5 Hz). This EEG finding suggests abnormality of left cerebral hemisphere.

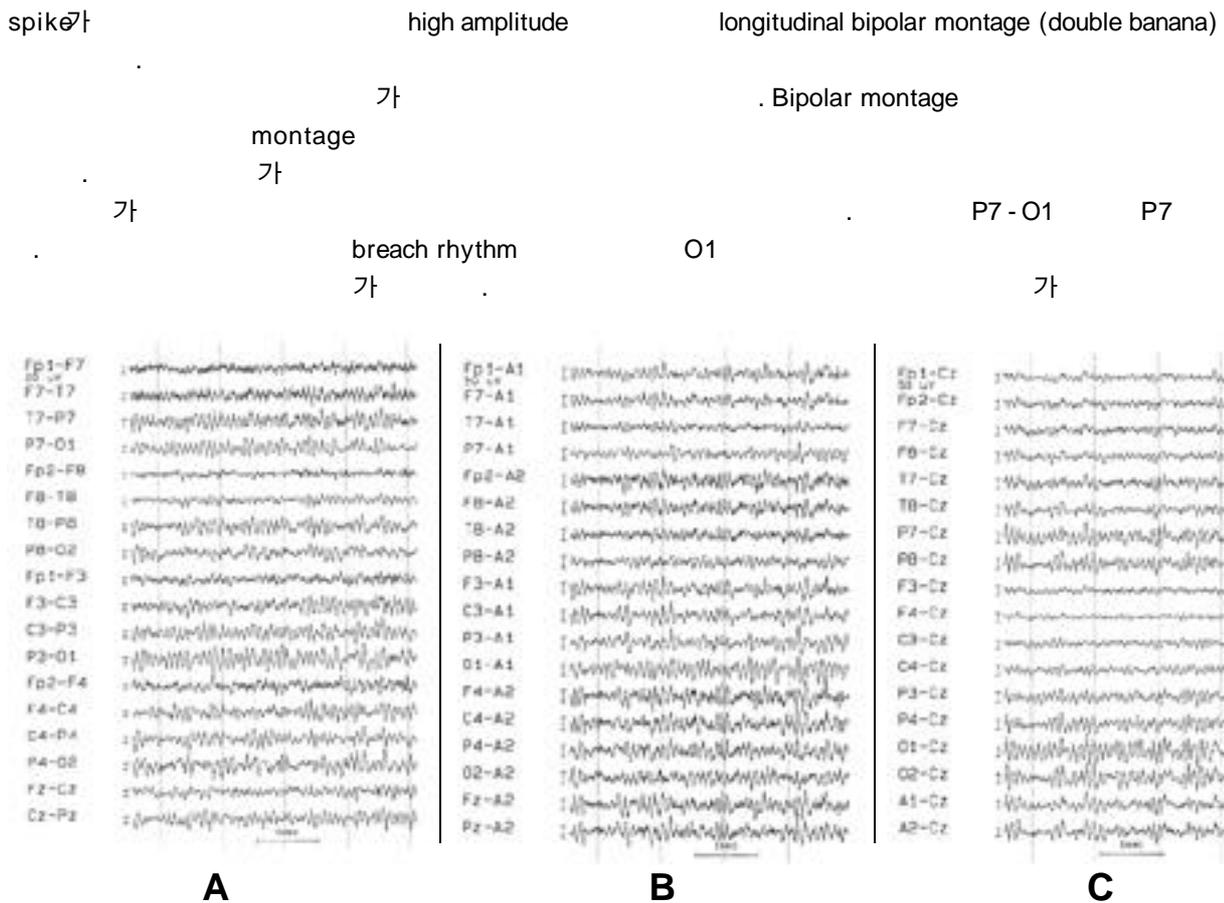


Figure 9. (A) Alpha rhythm is nicely recorded at both posterior head regions. However, bipolar montage shows only the amplitude difference of two electrodes (e.g., P7-O1, P8-O2), not the absolute amplitude of each electrode. Therefore, bipolar montage cannot compare the amplitudes of alpha rhythms between right and left hemispheres. (B) Ear reference (A1, A2) montage is contaminated by alpha rhythm because A1 and A2 electrodes are placed within the active field of alpha rhythm. Rhythmic activities recorded at both frontal electrodes are false rhythms produced by A1, A2 electrode contamination. (C) Cz reference montage shows alpha rhythm confined to both posterior head regions with little contamination at both frontal regions.

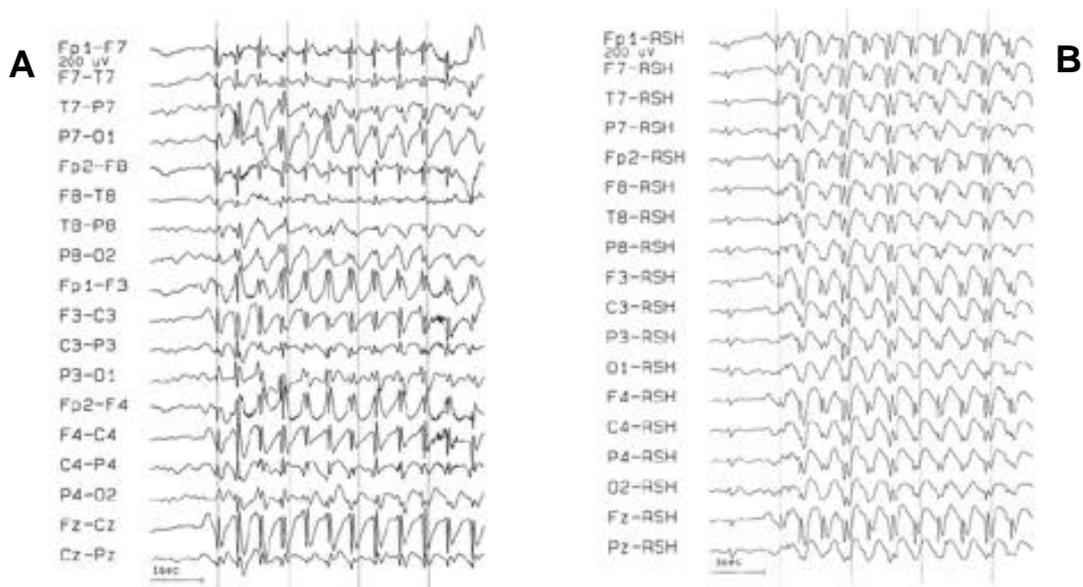


Figure 10. (A) Longitudinal bipolar montage shows generalized spike-wave complex. Because all electrodes produce epileptic discharges, the selection of reference electrode is almost impossible. (B) RSH (right shoulder electrode) reference montage is able to display a potential distribution of generalized spike-wave complex. Non-cephalic RSH electrode is far away from the brain, so relatively free from generalized epileptic discharges.

Cz reference montage
 A1, A2 reference montage (electrical field)
 (Fig. 9 - a, b, c).
 가,
 50%
 70%
 가
 vertex sharp transients,
 (sleep spindle) Cz
 reference
 vertex, frontocentral maximum
 A1, A2 reference transverse
 montage
 50%
 (asymmetry)
 paper EEG A1, A2 reference montage 2
 Cz reference montage 4
 A1, A2 reference 가
 , Cz reference 가
 가 , paper EEG 가
 Cz reference 2 A1, A2 refer -
 ence 4

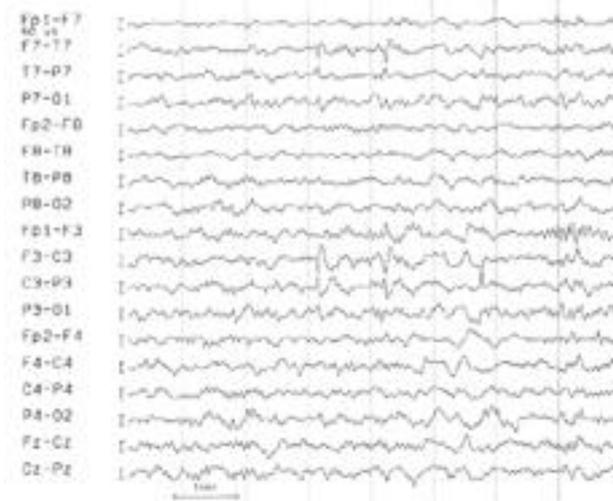


Figure 11. Phase reversals are seen at C3 and T7 electrode with a maximum deflection at C3. The location of maximum phase reversal indicates the epileptic focus at C3 (left central region).

4) (distribution)
 wide-spread, diffuse or generalized
 (phase reversal)
 (maximum point)
 가
 (reference electrode)
 right shoulder,
 left shoulder 腦外 (extracerebral electrode)
 Right shoulder
 electrode (Fig.
 10 - a, b). (electrical activity)
 (lateralized)
 가
 vertex
 가
 가
 가

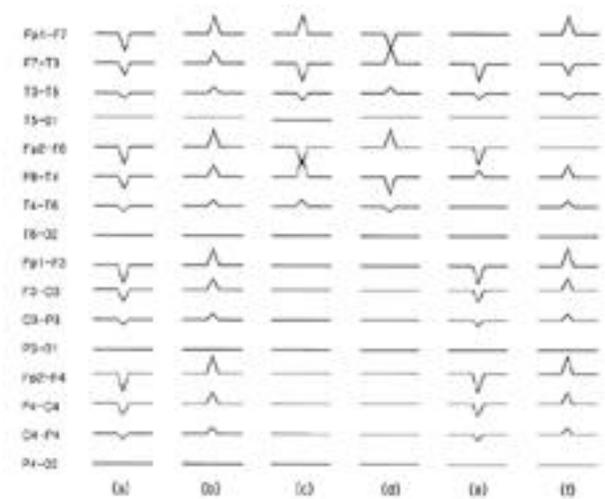


Figure 12. EEG changes according to eyeball movements. (a) upward eyeball movement, (b) downward eyeball movement, (c) left lateral eyeball movement, (d) right lateral eyeball movement, (e) left upward oblique eyeball movement, (f) left downward oblique eyeball movement.

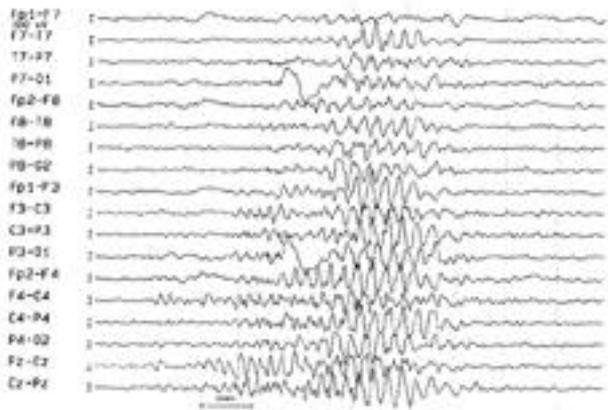


Figure 13. Hypnagogic hypersynchrony. Paroxysmal discharges of high amplitude slow waves intermixed with small sharp-looking waves are recorded during light sleep. This paroxysmal hypersynchrony is a normal drowsy response, not abnormal discharges.

가 (regional) (lobe) sharp waves (midline) bi - regional, (multifocal) 가 5) (phase) (timing) (polarity) (troughs) (peaks) in phase out of phase 가 180 (origin) (Fig. 11). 6) (reactivity) eye blocking 가 가 . 8~14 occipital slow waves “posterior slow waves of youth” 가

(alert) 1.5 가 가 (specificity) 가 (severity) 가 1~2 abnormality III가 가 abnormality I 가 (continuous slow) abnormality II - III(가 가 abnormality II Hans Lüders가 가²² - 1) 가 Abnormality III 1. Spikes, regional, left temporal lobe 2. Intermittent slow, regional, left temporal lobe The waking and sleep EEG suggest a partial seizure disorder arising from left temporal region and a mild regional cerebral dysfunction over that area. - 2) polyspike and wave mixture가 가 7 Hz Abnormality III 1. Polyspike - wave mixture, generalized 2. Background slow The waking and sleep EEG suggest a generalized seizure disorder and a mild diffuse cerebral dysfunction. background slow 24 가 8 Hz background slow

3.

(cornea) , (retina)
 (dipole) 가
 . , 가
 . 가
 2
 . 2 가
 가

Fig. 12 . 가 longitudinal bipolar montage 8 (temporal chain) 가
 , 8 parasagittal chain 가
 parasagittal chain 가
 parasagittal chain 가 (positive pole)
 Fp1, Fp2 가
 Fp1 - F3, Fp2 - F4 가 (Fig. 12 - a).
 가 가 Fp1 Fp2
 Fp1 - F3 Fp2 - F4 가
 가 (Fig. 12 - b). 가
 F7 가 F7
 Fp1 - F7 F7 - T3
 (positive phase reversal)
 , F8
 F8 Fp2 - F8 F8 - T4
 (negative phase reversal) (Fig. 12 - c).

4. (Pseudoepileptiform patterns) (artifacts)

(pseudoepileptiform patterns)

. Small sharp spikes (benign epileptiform transients of sleep), 6 Hz spike - and - slow - wave (phantom spike - and - wave), 14 and 6 Hz positive bursts, rhythmical mid - temporal discharge (psy -

chomotor variant), wicket spikes, occipital spikes and sharp waves of blind persons, SREDA (subclinical rhythmic EEG discharge of adults), midline theta rhythms (of Ciganek), paroxysmal hypnagogic hyper - synchrony

hypnagogic hypersynchrony 가 (Fig. 13).

가 (artifacts)
 . 가
 , muscle artifact, 가
 가
 movement artifact, ECG artifact, pulse wave artifact, perspiration artifact, tongue artifact, oropharyngeal artifact,
 가
 dental restoration artifact .
 , input cable, selector switch , 60 Hz artifact
 가

REFERENCES

- Schaal N. The fundamental neural mechanisms of electroencephalography. *Electroencephalogr Clin Neurophysiol* 1998;106(2):101-7.
- Duncan JS. Imaging and epilepsy. *Brain* 1997;120 (Pt 2): 339-77.
- Elger CE, Widman G, Andrzejak R, Arnhold J, David P, Lehnertz K. Nonlinear EEG analysis and its potential role in epileptology. *Epilepsia* 2000; 41 Suppl 3:S34-8.
- Speckmann E, Elger CE. Introduction to the neurophysiological basis of the EEG and DC potentials, In Niedermeyer E. *Electroencephalography. Basic principles, clinical applications and related fields*. 1999, Williams & Wilkins: Baltimore. 15-27.

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5. Ebersole JS. Noninvasive localization of epileptogenic foci by EEG source modeling. *Epilepsia* 2000;41 Suppl 3: S24-33.
 6. Buzsaki G, Traub RD, Pedley TA. The cellular basis of EEG activity. 3rd ed. In Ebersole JS, Pedley TA. *Current practice of clinical electroencephalography*. 2003; Philadelphia: Lippincott Williams & Wilkins. 1-11.
 7. Ebersole JS. Defining epileptogenic foci: past, present, future. *J Clin Neurophysiol*, 1997;14(6):470-83.
 8. Wong PK. Source modelling of the rolandic focus. *Brain Topogr* 1991;4(2):105-12.
 9. Bear MF, Connors BW, Paradiso MA. *Rhythms of the brain. Neuroscience Exploring the Brain* 2001; Baltimore: Lippincott Williams & Wilkins. 606-636.
 10. Steriade M, Gloor P, Llinas RR, Lopes de Silva FH, Mesulam MM. Report of IFCN Committee on Basic Mechanisms. Basic mechanisms of cerebral rhythmic activities. *Electroencephalogr Clin Neurophysiol* 1990; 76(6):481-508.
 11. Lopes da Silva FH, Vos JE, Mooibroek J, Van Rotterdam A. Relative contributions of intracortical and thalamo-cortical processes in the generation of alpha rhythms, revealed by partial coherence analysis. *Electroencephalogr Clin Neurophysiol* 1980; 50(5-6): 449-56.
 12. Steriade M, McCormick DA, Sejnowski TJ. Thalamocortical oscillations in the sleeping and aroused brain. *Science* 1993;262(5134):679-85.
 13. Binnie CD, Stefan H. Modern electroencephalography: its role in epilepsy management. *Clin Neurophysiol* 1999; 110(10):1671-97.
 14. de Curtis M, Avanzini G. Interictal spikes in focal epileptogenesis. *Prog Neurobiol* 2001;63(5):541-67.
 15. Snead OC, 3rd. Basic mechanisms of generalized absence seizures. *Ann Neurol* 1995;37(2):146-57.
 16. McCormick DA. Cortical and subcortical generators of normal and abnormal rhythmicity. *Int Rev Neurobiol* 2002;49:99-114.
 17. Thatcher RW, Biver C, McAlaster R, Salazar A. Biophysical linkage between MRI and EEG coherence in closed head injury. *Neuroimage* 1998;8(4):307-26.
 18. Thatcher RW, Biver C, McAlaster R, Camacho M, Salazar A. Biophysical linkage between MRI and EEG amplitude in closed head injury. *Neuroimage* 1998;7(4 Pt 1):352-67.
 19. Thatcher RW, Krause PJ, Hrybyk M. Cortico-cortical associations and EEG coherence: a two-compartmental model. *Electroencephalogr Clin Neurophysiol* 1986; 64(2):123-43.
 20. Fisch BJ. *EEG primer: basic principles of digital and analog EEG*. Amsterdam: Elsevier Science BV 1999;145-154.
 21. Ebersole JS, Pedley TA. *Current practice of clinical electroencephalography*. Philadelphia: Lippincott Williams & Wilkins 2003;102-108.
 22. Lüders H, Noachtar S. *Atlas and Classification of Electroencephalography*. Philadelphia, W.B. Saunders Company, 2000.