# Study on Electroencephalography (EEG) Past, Present & Future

A Thesis submitted in partial fulfillment of the requirements for the Award of Degree of Bachelor of Science in Electrical and Electronic Engineering

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## **DEPARTMENT OF ELECTRICAL AND ELECTRONIC ENGINEERING**

## FACULTY OF ENGINEERING

## **DAFFODIL INTERNATIONAL UNIVERSITY**

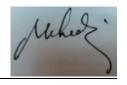
## August-2018

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## Certification

This is to certify that this thesis entitled "**Electroencephalography (EEG) Past, Present & Future**" is done by the following student under my direct supervision and this work has been carried out by him in the laboratories of the Department of Electrical and Electronic Engineering under the Faculty of Engineering of Daffodil International University in partial fulfillment of the requirements for the degree of Bachelor of Science in Electrical and Electronic Engineering. The presentation of the work was held on.

### Signature of the Candidate



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### Countersigned



Md. Mahmudur Rahman Assistant Professor Department of Electrical and Electronic Engineering Faculty of Engineering Daffodil International University **Dedicated To...** 

## My beloved Parents And

## **All of My Teachers**

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### ABSTRACT

The field of electroencephalography (EEG) has witnessed a dramatic development during the last decade. Electroencephalography (EEG) has been in continuous development over at least 70 years and is firmly established as a tool in the management of epilepsy. The electroencephalogram that had been principally used as a 'post-hoc' diagnostic procedure is now fully used as an 'on-line' monitor of neural function with its excellent temporal resolution.

For a while, the technique fell into disregard because of difficulties with interpretation, specificity and sensitivity. Whilst clinicians have to be aware of these problems, they have been largely addressed by recent computer digitization of signals, which permits longer standard recordings and monitoring linked to a simultaneous video. Neurophysiological monitoring in the operating room, neurological intensive care unit (ICU) and during endovascular procedures allows early identification of impending neurological deficits before irreversible neurological impairment.

These techniques are not only an essential component of a specialist epilepsy service, where inpatient video-EEG telemetry is vital both for diagnosis and assessment before neurosurgical treatment, but also in general and acute medical settings, particularly for the management of status epilepticus. Further developments in computing will extend the use of EEG in all of these roles and long-term monitoring for diagnosis and management of coma will become more widely available. The advent of digital EEG with digital storage and the ability to manipulate data with digital reformatting, filter and sensitivity changes has allowed us to maximize the information and reduce artifacts. These changes have revolutionized the way in which EEG is performed and interpreted.

Keywords: EEG; Epilepsy; Technology; Telemetry; Clinical applications; Monitoring.

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## **Chapter 1** Introduction to Brain Signal

Take a minute and put your hand onto your head and think about what you are touching. Within a centimeter of your fingers, there is a piece of the most mysterious and rarely understood matter in the universe. And that is our brain.

Inside each of our brains, there are roughly 100 billion highly specialized cells called neurons. They make about 500 trillion connections called synapsis. These unique cells transmit important information alarming us the sense and interacts with the world around us. If you want to take a closer look, you will see that this information is transmitted between neurons using chemicals called neurotransmitters where tiny structures called synaptic vesicles fuse with the membrane of one neuron and release chemicals signals into the gap. The second neuron can receive them.

Scientists already knew some about how this neurotransmission process works. But now after over 10 years of collaborated research of Stanford University and SLAC national accelerator laboratory along with the ultra-bright X-rays, scientists now have a better idea of exactly how these tiny vesicles might fuse with the membrane of one neuron to transmit their signals. The key to this fusion is the collaboration between special proteins called snares and synaptotagmin-1. They are then triggered by calcium to cause the vesicle of fuse with the membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state. Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neurotransmitters to leave the neuron. This experiment represents the first time when scientists have seen how synaptotagmin-1 interacts with the snares of the atomic scale and scientists are more confident that this protein resembles before calcium arrives allowing the fusion process and resulting neurotransmission to happen very quickly getting information from point A to point B in less than a millisecond. The end result is that our nervous system can work at an incredible speeds enabling us to sense, react to and interact with the world around us.

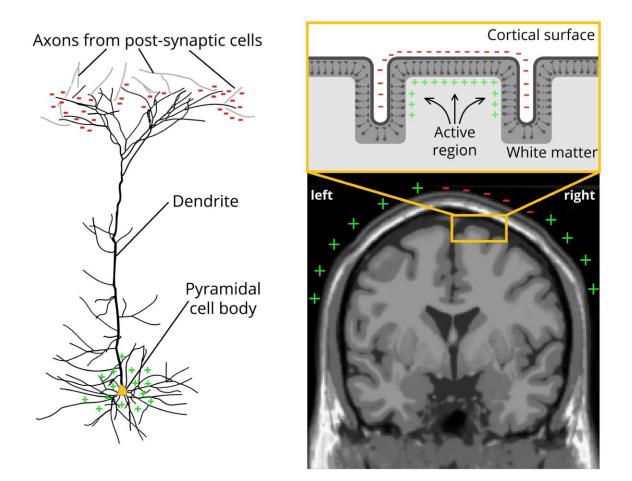


Fig: 1.1 Activities inside human brain

And now the scientists have been able to use the bright X-ray SSRL, the LCLS and the ANL light source to see how this particular process works. It opens the door to better understand our nervous system and ways that even our brains can think of.

## **Chapter 2** Introduction to Electroencephalography (EEG)

## 2.1 What is Electroencephalography (EEG)

- Electroencephalography (EEG) is the recording of electrical activity along the scalp.
- > EEG measures voltage fluctuations resulting from activation of neurons of the brain.
- > We can use EEG techniques to detect brainwaves.
- > During the EEG test, small electrodes like cup or disc type are placed on the scalp.
- They pick the brain's electrical signal and send them to a machine called Electroencephalogram.



Fig: 2.1 Epileptic spike and wave discharges monitored with EEG

## 2.2 EEE generation

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Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neurotransmitters to leave the neuron.

This experiment represents the first time when scientists have seen how synaptotagmin-1 interacts with the snares of the atomic scale and scientists are more confident that this protein resembles before calcium arrives allowing the fusion process and resulting neurotransmission to happen very quickly getting information from point A to point B in less than a millisecond.

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### 2.3 Types of EEG

There are several different types of EEG and can be listed as the following:

#### 2.3.1 Normal EEG

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#### 2.3.2 Sleep EEG

If you want to take a closer look, you will see that this information is transmitted between neurons using chemicals called neurotransmitters where tiny structures called synaptic vesicles fuse with the membrane of one neuron and release chemicals signals into the gap. The second neuron can receive them.

Scientists already knew some about how this neurotransmission process works. But now after over 10 years of collaborated research of Stanford University and SLAC national accelerator laboratory along with the ultra-bright X-rays, scientists now have a better idea of exactly how these tiny vesicles might fuse with the membrane of one neuron to transmit their signals.

#### 2.3.3 Common Physiological Artifacts

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#### **5.3.4 The Posterior Dominant Rhythm**

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#### **5.3.5 Provocation Techniques**

When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state. Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neurotransmitters to leave the neuron. This experiment represents the first time when scientists have seen how synaptotagmin-1 interacts with the snares of the atomic scale and scientists are more confident that this protein resembles before calcium arrives allowing the fusion process and resulting neurotransmission to happen very quickly getting information from point A to point B in less than a millisecond. The key to this fusion is the collaboration between special proteins called snares and synaptotagmin-1. They are then triggered by calcium to cause the vesicle of fuse with the membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state. Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neuronsmitters to leave the neuron.

## 2.4 The Developmental EEG: Premature, Neonatal and Children

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Figure 2.2 REM sleep is characterized by a more typically wake-appearing, desynchronized, mixed-frequency background, which may contain alpha frequencies, characteristic centrally dominant sharply contoured sawtooth waves, and rapid eye movement artifacts in lateral frontal electrode sites. Copyright 2013. Mayo Foundation for Medical Education and Research. All rights reserved. Figure courtesy of Erik K. St. Louis, MD.

If you want to take a closer look, you will see that this information is transmitted between neurons using chemicals called neurotransmitters where tiny structures called synaptic vesicles fuse with the membrane of one neuron and release chemicals signals into the gap. The second neuron can receive them.

## 2.5 Benefits of using Electroencephalography (EEG)

Electroencephalography has several benefits. The main benefit of Electroencephalography is its high time conductivity. It can take hundreds to thousands of shots of electrical activity across within a second. It is very useful but it has some disadvantages too.

## **Chapter 3** History of Electroencephalography (EEG)

## 3.1 Invention of Electroencephalography

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## 3.2. Progress of Electroencephalography

#### 3.2.1 Richard Caton's work

Richard Caton (1842–1926) invented it in 1875.

#### 3.2.2 Carlo & Emil Du's work

Carlo Matteucci 1811 and Emil Du Bois-Reymond 1818 invented it first.

### 3.2.3 First Electroencephalographic

Hans Berger 1873 discover EEG signals first.

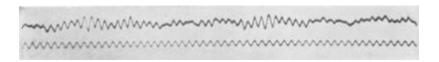


Fig: 3.1 The first human EEG recording obtained by Hans Berger in 1924.



Fig: 3.2 Hans Berger(1873-1941)

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#### **3.2.4 Limitations**

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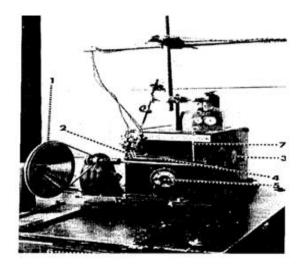


Fig: 3.3 Frist String Galvanometer with recording apparatus

#### 3.2.5 Works by other scientists

Take a minute and put your hand onto your head and think about what you are touching. Within a centimeter of your fingers, there is a piece of the most mysterious and rarely understood matter in the universe. And that is our brain.

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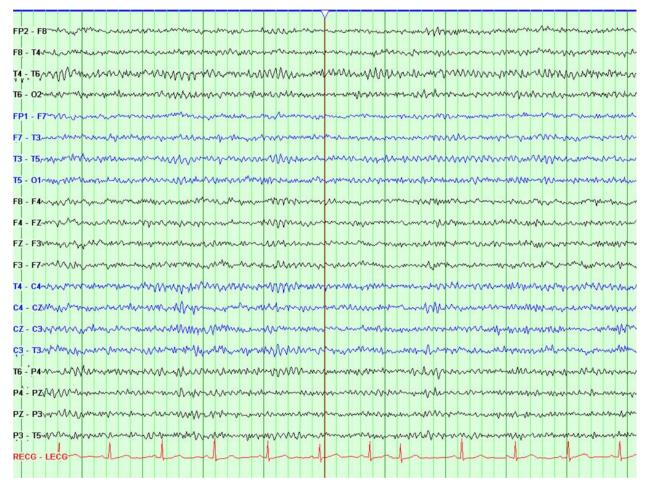


Fig: 3.4 Normal EEG recording from an adult using a longitudinal temporal and transverse bipolar montage.

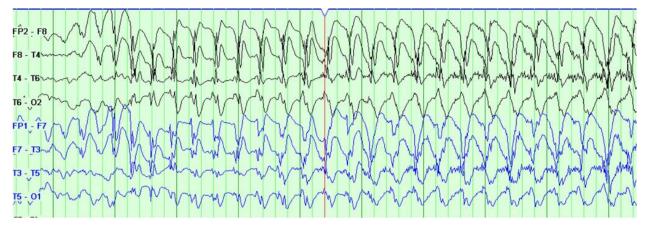


Fig: 3.5 Scalp EEG recording of ictal onset in a patient with absence epilepsy using a longitudinal bipolar montage.

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The end result is that our nervous system can work at an incredible speeds enabling us to sense, react to and interact with the world around us.



Fig: 3.6 Recording of ictal onset in a patient with right mesial temporal epilepsy due to hippocampal sclerosis using subdural electrodes inserted to cover the inferior surface of the temporal lobe cortex.

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## **Chapter 4** Electroencephalography (EEG) Signal Processing

## 4.1. Fundamentals of Electroencephalography (EEG) Signal Processing

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#### 4.1.1 EEE signal modeling

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## **4.2 Various Bands**

Inside each of our brains, there are roughly 100 billion highly specialized cells called neurons. They make about 500 trillion connections called synapsis. These unique cells transmit important information alarming us the sense and interacts with the world around us. If you want to take a closer look, you will see that this information is transmitted between neurons using chemicals called neurotransmitters where tiny structures called synaptic vesicles fuse with the membrane of one neuron and release chemicals signals into the gap. The second neuron can receive them. Scientists already knew some about how this neurotransmission process works. But now after over 10 years of collaborated research of Stanford University and SLAC national accelerator laboratory along with the ultra-bright X-rays, scientists now have a better idea of exactly how these tiny vesicles might fuse with the membrane of one neuron to transmit their signals

#### 4.2.1 Delta band (1-4 Hz)

Delta waves lie within the range of 0.5-4 Hz. These waves are primarily associated with deep sleep and may be present in the waking state. It is very easy to confuse artefact signals caused by the large muscles of the neck and jaw with the genuine delta response.

#### 4.2.2. Theta band (4-8 Hz)

Theta waves lie within the range of 4– 7.5 Hz. The term theta might be chosen to allude to its presumed thalamic origin. Theta waves appear as consciousness slips towards drowsiness. Theta waves have been associated with access to unconscious material, creative inspiration and deep meditation.

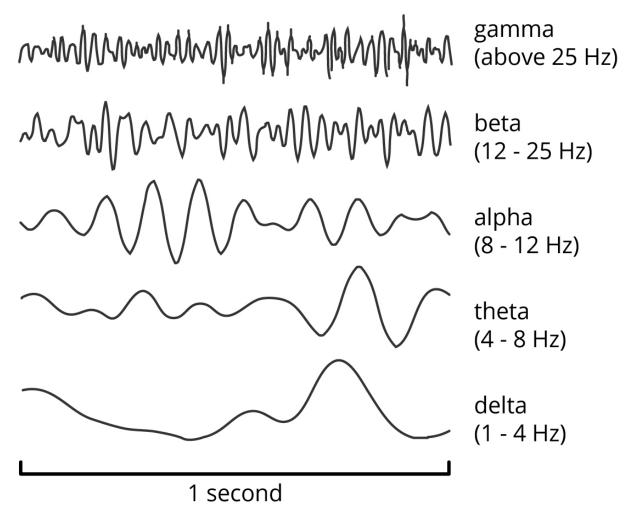


Fig: 4.1 Frequency variance per second

#### 4.2.3. Alpha band (8 - 12 Hz)

These unique cells transmit important information alarming us the sense and interacts with the world around us. If you want to take a closer look, you will see that this information is transmitted between neurons using chemicals called neurotransmitters where tiny structures called synaptic vesicles fuse with the membrane of one neuron and release chemicals signals into the gap.

#### 4.2.4. Beta band (12- 25 Hz)

Scientists already knew some about how this neurotransmission process works. But now after over 10 years of collaborated research of Stanford University and SLAC national accelerator laboratory along with the ultra-bright X-rays, scientists now have a better idea of exactly how these tiny vesicles might fuse with the membrane of one neuron to transmit their signals

#### 4.2.5 Gamma band (above 25 Hz)

These unique cells transmit important information alarming us the sense and interacts with the world around us. If you want to take a closer look, you will see that this information is transmitted between neurons using chemicals called neurotransmitters where tiny structures called synaptic vesicles fuse with the membrane of one neuron and release chemicals signals into the gap.

#### 4.2.6 Other Waves

Within a centimeter of your fingers, there is a piece of the most mysterious and rarely understood matter in the universe. And that is our brain.

Inside each of our brains, there are roughly 100 billion highly specialized cells called neurons. They make about 500 trillion connections called synapsis.

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membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state.

Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neurotransmitters to leave the neuron.

This experiment represents the first time when scientists have seen how synaptotagmin-1 interacts with the snares of the atomic scale and scientists are more confident that this protein resembles before calcium arrives allowing the fusion process and resulting neurotransmission to happen very quickly getting information from point A to point B in less than a millisecond. The end result is that our nervous system can work at an incredible speeds enabling us to sense, react to and interact with the world around us.

### **4.3 EEG Recording and Measurement**

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## 4.4 Conventional Electrode Positioning

The key to this fusion is the collaboration between special proteins called snares and synaptotagmin-1. They are then triggered by calcium to cause the vesicle of fuse with the membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state. Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neurotransmitters to leave the neuron. This experiment represents the first time when scientists have seen how synaptotagmin-1 interacts with the snares of the atomic scale and scientists are more confident that this protein resembles before calcium arrives allowing the fusion process and resulting neurotransmission to happen very quickly getting information from point A to point B in less than a millisecond. The end result is that our nervous system can work at an incredible speeds enabling us to sense, react to and interact with the world around us.

Points to note in the 10-20 system:

#### Nasion (Nz)

The noise between the eyes at the top of the nose.

#### Inion (Iz)

The bump at the back of the head.

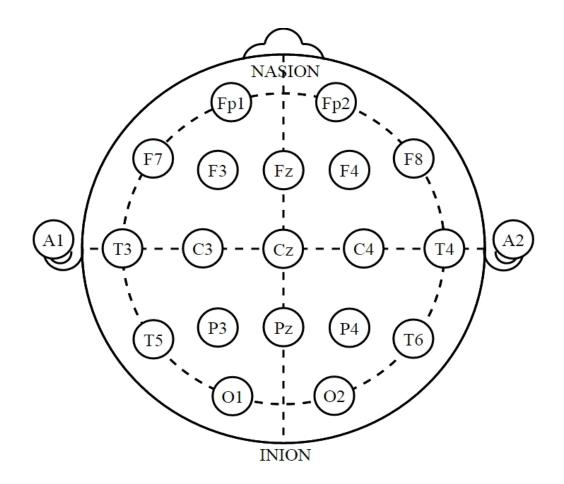


Fig: 4.2 Placement of Electrodes

The second neuron can receive them. Scientists already knew some about how this neurotransmission process works. But now after over 10 years of collaborated research of Stanford University and SLAC national accelerator laboratory along with the ultra-bright X-rays, scientists now have a better idea of exactly how these tiny vesicles might fuse with the membrane of one neuron to transmit their signals. The key to this fusion is the collaboration between special proteins called snares and synaptotagmin-1. They are then triggered by calcium to cause the vesicle of fuse with the membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state. Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neurotransmitters to leave the

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### 4.5 Number and distribution of electrodes

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## 4.6 Clean Electroencephalography data and artefacts

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#### 4.6.1 Physiological artefacts

The second neuron can receive them. Scientists already knew some about how this neurotransmission process works. But now after over 10 years of collaborated research of Stanford University and SLAC national accelerator laboratory along with the ultra-bright X-rays, scientists now have a better idea of exactly how these tiny vesicles might fuse with the membrane of one neuron to transmit their signals. The key to this fusion is the collaboration between special proteins

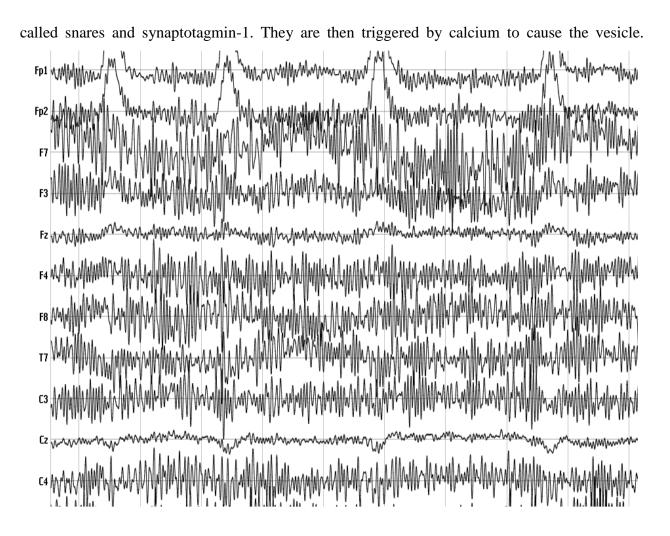


Fig: 4.3 Muscle activity of electric current

#### **Eye movements**

They are then triggered by calcium to cause the vesicle of fuse with the membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state. Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together. This finally triggers fusion allowing the neurotransmitters to leave the neuron. This experiment represents the first time when scientists have seen how synaptotagmin-1 interacts with the snares of the atomic scale and scientists are more confident that this protein resembles before

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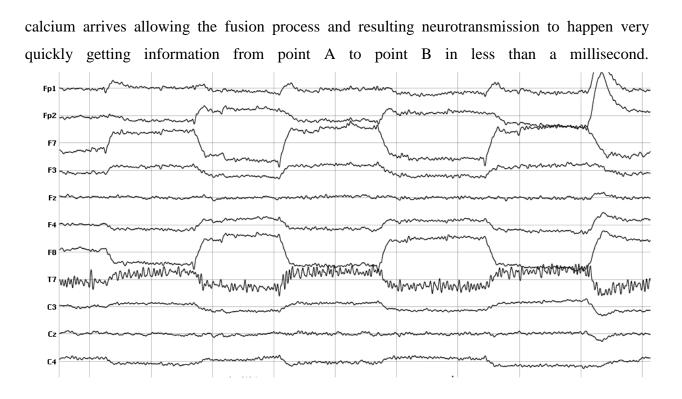


Fig: 4.4 Eye movement

## **Blinking:**

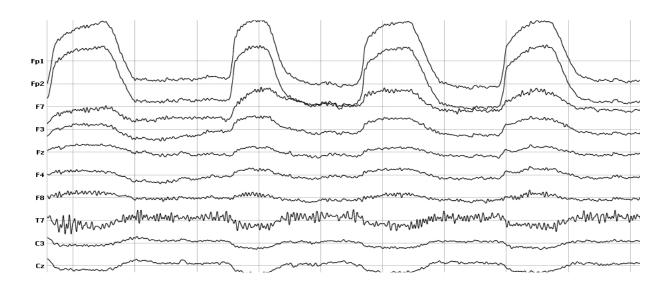


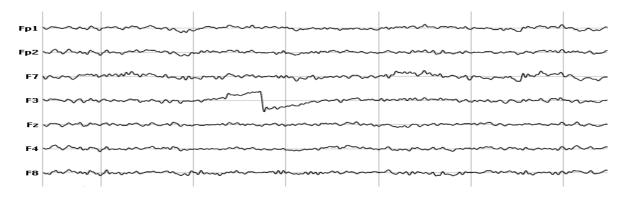
Fig: 4.5 Blinking effect

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#### 4.6.2 External sources of artefacts



Movement of an electrode can cause many artefacts (Fig 4.6).

Fig: 4.6 Movement of electrodes

#### Line noise

Line noise (50 Hz in the United States, 60 Hz in the Europe) probably have strong artefacts on the electrode listing - it is quite common in the raw Electroencephalography data.

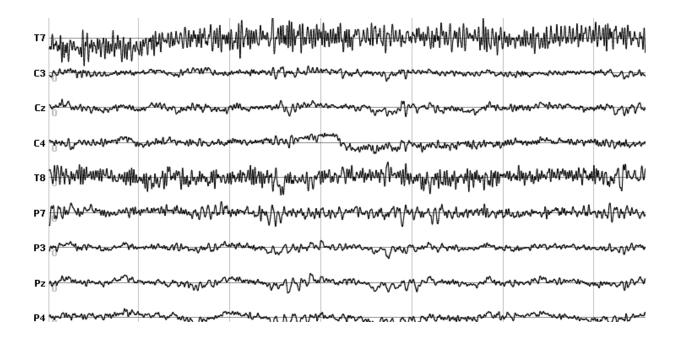


Fig: 4.7 Noise variation over frequency

# **Chapter 5** Application of Electroencephalography (EEG)

## **5.1 Medical or Clinical Applications**

It has severe medical applications.

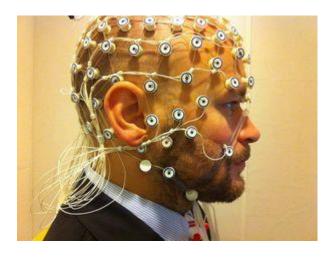


Fig: 5.1 An EEG recording setup

The key to this fusion is the collaboration between special proteins called snares and synaptotagmin-1. They are then triggered by calcium to cause the vesicle of fuse with the membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the proteins connect with the two and enter a pre-fusion state.

### 5.1.1 Dementia

Dementia is a syndrome that consists of a decline in intellectual and cognitive abilities. This consequently affects the normal social activities, mode, and the relationship and interaction with other people. EEG is often used to study the effect of dementia. In most cases, such as in primary degenerative dementia, e.g. Alzheimer's, and psychiatric disorder, e.g. depression with cognitive impairment, the EEG can be used to detect the abnormality.

Dementia is classified into cortical and subcortical forms. The most important cortical dementia is Alzheimer's disease (AD), which accounts for approx- imately 50 % of the cases. Other known cortical abnormalities are Pick's disease and Creutzfeldt– Jakob diseases (CJD). They are characterized clinically by findings such as aphasia, apraxia, and agnosia. CJD can often be diagnosed using the EEG signals. Figure shows a set of EEG signals from a CJD patient. On the other hand, the most common subcortical diseases are Parkinson's disease, Huntington's disease, lacunar state, normal pressure hydrocephalus, and progressive supranuclear palsy. These diseases are characterized by forgetfulness, slowing of thought processes, apathy, and depression. Generally, subcortical dementias introduce less abnormality to the EEG patterns than the cortical ones.

In AD the EEG posterior rhythm (alpha rhythm) slows down and the delta and theta wave activities increase. On the other hand, beta wave activity may decrease. In severe cases epileptiform discharges and triphasic waves can appear. In such cases, cognitive impairment often results. The spectral power also changes; the power increases in delta and theta bands and decreases in beta and alpha bands and also in mean frequency.

The EEG wave morphology is almost the same for AD and Pick's disease. Pick's disease involves the frontal and temporal lobes. An accurate analysis followed by an efficient classification of the cases may discriminate these two diseases. CJD is a mixed cortical and subcortical dementia. This causes slowing of the delta and theta wave activities and, after approximately three months of the onset of the disease, periodic sharp wave complexes are generated that occur almost every second, together with a decrease in the background activity [54]. Parkinson's disease is a subcortical dementia, which causes slowing down of the background activity and an increase of the theta and delta wave activities. Some works have been undertaken using spectral analysis to confirm the above changes [55]. Some other disorders such as depression have a lesser effect on the EEGs and more accurate analysis of the EEGs has to be performed to detect the signal abnormalities for these brain disorders.

The key to this fusion is the collaboration between special proteins called snares and synaptotagmin-1. They are then triggered by calcium to cause the vesicle of fuse with the membrane of the neuron. When a synaptic vesicle comes close enough to the membrane, the © Daffodil International University

proteins connect with the two and enter a pre-fusion state. Next when the neuron fires, calcium arrives and triggers the proteins which bend the neural membrane towards the vesicle membrane and draw the two together.

#### 5.1.2 Additional work done by EEG

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#### 5.1.3 Epileptic Seizure and Nonepileptic Attacks

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#### 5.1.4 Psychiatric Disorders

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### **5.2 External effects**

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## **5.3 Applications of EEG monitoring**

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#### 5.3.1 Long-term Video-EEG Monitoring

Dementia is a syndrome that consists of a decline in intellectual and cognitive abilities. This consequently affects the normal social activities, mode, and the relationship and interaction with other people. EEG is often used to study the effect of dementia. In most cases, such as in primary degenerative dementia, e.g. Alzheimer's, and psychiatric disorder, e.g. depression with cognitive impairment, the EEG can be used to detect the abnormality.

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#### 5.3.2 Pitfalls in Video-EEG Monitoring

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### 5.3.3 Long-term Video-EEG Monitoring in a Preoperative Evaluation

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## **5.4 Various Applications**

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# **Chapter 6** Advantages & Disadvantages of Electroencephalography (EEG)

## 6.1 Advantages of Electroencephalography (EEG)

EEG has two clear advantages for brain research. Dementia is a syndrome that consists of a decline in intellectual and cognitive abilities. This consequently affects the normal social activities, mode, and the relationship and interaction with other people. EEG is often used to study the effect of dementia. In most cases, such as in primary degenerative dementia, e.g. Alzheimer's, and psychiatric disorder, e.g. depression with cognitive impairment, the EEG can be used to detect the abnormality.

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## 6.2 Disadvantages Electroencephalography (EEG)

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## 6.3 Limitations

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## **6.4 Abnormal activity**

Dementia is a syndrome that consists of a decline in intellectual and cognitive abilities. This consequently affects the normal social activities, mode, and the relationship and interaction with other people. EEG is often used to study the effect of dementia. In most cases, such as in primary degenerative dementia, e.g. Alzheimer's, and psychiatric disorder, e.g. depression with cognitive impairment, the EEG can be used to detect the abnormality.

Dementia is classified into cortical and subcortical forms. The most important cortical dementia is Alzheimer's disease (AD), which accounts for approx- imately 50 % of the cases. Other known cortical abnormalities are Pick's disease and Creutzfeldt– Jakob diseases (CJD). They are characterized clinically by findings such as aphasia, apraxia, and agnosia. CJD can often be diagnosed using the EEG signals. Figure shows a set of EEG signals from a CJD patient. On the other hand, the most common subcortical diseases are Parkinson's disease, Huntington's disease, lacunar state, normal pressure hydrocephalus, and progressive supranuclear palsy. These diseases are characterized by forgetfulness, slowing of thought processes, apathy, and depression.

Generally, subcortical dementias introduce less abnormality to the EEG patterns than the cortical ones.

In AD the EEG posterior rhythm (alpha rhythm) slows down and the delta and theta wave activities increase. On the other hand, beta wave activity may decrease. In severe cases epileptiform discharges and triphasic waves can appear. In such cases, cognitive impairment often results. The spectral power also changes; the power increases in delta and theta bands and decreases in beta and alpha bands and also in mean frequency.

# **Chapter 7 Discussion & Conclusion**

## 7.1 Discussion & Conclusion

Dementia is a syndrome that consists of a decline in intellectual and cognitive abilities. This consequently affects the normal social activities, mode, and the relationship and interaction with other people. EEG is often used to study the effect of dementia. In most cases, such as in primary degenerative dementia, e.g. Alzheimer's, and psychiatric disorder, e.g. depression with cognitive impairment, the EEG can be used to detect the abnormality.

## References

- American Electroencephalographic Society (1994). American Electro-encephalographic Society. Guideline thirteen: Guidelines for standard electrode positionnomenclature. Journal of Clinical Neurophysiology, 11, 111–113.
- Berger H (1929) Uber das Elektrenkephalogramm des Menschen. Arch Psychiat Nervenkr 87:527–570.
- de Vico Fallani., Astolfi, Cincotti et al. (2008). Structure of the cortical networks during successful memory encoding in TV commercials. Clinical Neurophysiology, 119(10), 2231–2237.
- Astolfi, De Vico Fallani, Cincotti, et al. (2008). Neural basis for brain responses to TV commercials: a high-resolution EEG study. IEEE Transactions in Neural Systems and Rehabilitation Engineering, 16(6), 522–531.
- Berka, Izzetoglu, Bunce et al. (2004). Real-time analysis of EEG indexes of alertness, cognition, and memory acquired with a wireless EEG headset. International Journal of Human-Computer Interaction, 17(2), 211–227.
- Berka, Levendowski, Lumicao et al. (2007). EEG correlates of task engagement and mental workload in vigilance, learning, and memory tasks. Aviation, Space, and Environmental Medicine, 78(5), B231–B244.
- Dawant B. M., Ozkan, M., Zijdenbos, A. and Margolin, R., 1991, A computer environment for 2D and 3D quantitation of MR images using neural networks. Mag. Reson. Imaging, 20: 64-65.
- Niedermeyer E.; da Silva F.L. (2004). Electroencephalography: Basic Principles, Clinical Applications, and Related Fields. Lippincot Williams & Wilkins.
- **9.** Chatrian, G.E.: The mu rhythm. In: Handbook of Electroencephalography and ClinicalNeurophysiology. The EEG of the Waking Adult, pp. 46–69. Elsevier, Amsterdam (1976).
- Ball, T., Kern, M., Mutschler, I., Aertsen, A., Schulze-Bonhage, A.: Signal quality of simultaneously recorded invasive and non-invasive EEG. NeuroImage 46(3), 708–716 (2009).

- **11.** Badcock, Preece, de Wit et al. (2015). Validation of the Emotiv EPOC EEG system for research quality auditory event-related potentials in children. PeerJ, 3, e907.
- **12.** Cohen (2011). It's about time. Frontiers in Human Neuroscience, 5(2), 1–15.
- Cohen (2014). Analyzing neural time series data. Theory and practice. Cambridge, MA: MIT Press.
- 14. Crone, N.E., Miglioretti, D.L., Gordon, B., Lesser, R.P.: Functional mapping of human sensorimotor cortex with electrocorticographic spectral analysis. ii. Event-related synchronization in the gamma band. Brain 121 (12), 2301–2315 (1998)
- 15. Babiloni, F., Cincotti, F., Lazzarini, L., Millan, J., Mourino, J., Varsta, M., Heikkonen, J., Bianchi, L., Marciani, M.G.: Linear classification of low-resolution EEG patterns produced by imagined hand movements. IEEE Trans. Rehabil. Eng. 8(2), 186–188 (2000)
- 16. Handy (2005). Event-related potentials: A methods handbook. Cambridge: MIT Press.
- **17.** Swartz, Barbara E. (1998). "The advantages of digital over analog recording techniques". Electroencephalography and Clinical Neurophysiology. **106** (2): 113–7.
- 18. Coenen, Anton, Edward Fine, and Oksana Zayachkivska. (2014). "Adolf Beck: A Forgotten Pioneer In Electroencephalography.". Journal of the History of the Neurosciences. 23 (3): 276–286.
- Pravdich-Neminsky, VV. (1913). "Ein Versuch der Registrierung der elektrischen Gehirnerscheinungen". Zbl Physiol. 27: 951–60.
- **20.** Haas, L F (2003). "Hans Berger (1873-1941), Richard Caton (1842-1926), and electroencephalography". Journal of Neurology, Neurosurgery & Psychiatry. **74** (1): 9.
- **21.** Millet, David (2002). "The Origins of EEG". International Society for the History of the Neurosciences (ISHN).
- 22. Williamson-Noble FA. Venous pulsations. Trans Ophthalmol Soc UK 1952;72:317–26.
- Coccius EA. Ueber die Anwendung des Augen-Spiegels, nebst Angabe eines neues Instrumentes. Leipzig: Muller, 1853:3–23.
- 24. Elliot RH. The retinal pulse. Br J Ophthalmol 1921;5:481–500
- 25. Baurmann M. Ueber die Entstehung und klinicke Bedeutung des Netzhautvenenpulses. Ber Zusammenkunft Dtsch Ophthalmol Ges 1925;45:53–9.
- **26.** Attariwala R, Giels CP, Glucksberg MR. The influence of elevated intra-ocular pressure on vascular pressures in the cat retina. Invest Ophthalmol Vis Sci 1994;35:1019–25.

- **27.** Levine DN. Spontaneous pulsations of the retinal veins. Microvasc Res 1998;56:154–65.
- **28.** Fry WE. Variations in the intraneural course of the central retinal vein. Arch Ophthalmol 1930;4:180–7.
- 29. Fry WE. The pathology of papilloedema: an examination of forty eyes with special reference to compression of the central vein of the retina. Am J Ophthalmol 1931;14:874–83.
- **30.** Dardenne G, Dereymaeker A, Lacheron JM. Cerebrospinal fluid pressure and pulsatility: an experimental study of circulatory and respiratory influences in normal and hydrocephalic dogs. Invest Neurol 1969;2:193–216.
- I Lorentzen SE. Incidence of spontaneous venous pulsation in the retina. Acta Ophthalmol 1970;48:765–76.
- **32.** Levin BE. The clinical significance of spontaneous pulsations of the retinal vein. Arch Neurol 1978;35:37–40.
- **33.** Gucer G, Viernstein L. Long-term intracranial pressure recording in the management of pseudotumour cerebri. J Neurosurg 1978;49:256–63.
- **34.** Hedges TR, Baron EM, Hedges TR, et al. The retinal venous pulse: its relation to optic disc characteristics and choroidal pulse. Ophthalmology 1994;101:542–7.
- 35. Rabbetts RB. Visual examination of the eye and ophthalmoscopy. In: Bennett AG, Rabbetts RB, eds. Bennett and Rabbett's clinical visual optics, 3rd edn. Oxford: Butterworth Heinemann, 1998:301–29.
- 36. Dreher AW, Tso PC, Weinrab RN. Reproducability of topographic measurements of the normal and glaucomatous optic nerve head with the laser tomographic scanner. Am J Ophthalmol 1991;111:221–9.
- **37.** Ubeyli ED, Combined neural network model employing wavelet coefficients for EEG signals classification, Digital Signal Processing, 19, 2009, 297–308.
- 38. Orhan U, Hekim M, Ozer M, EEG signals classification using the K-means clustering and a multilayer perceptron neural network model, Expert Systems with Applications, 38(10), 2011, 13475–13481.
- **39.** Berger H (1938) Uber das Elektrenkephalogramm des Menschen (14th report). Arch Psychiat Nervenkr 108:407–431

- 40. Towle VL, Bolaños J, Suarez D, Tan K, Grzeszczuk R, Levin DN, Cakmur R, Frank SA, Spire JP. (1993). "The spatial location of EEG electrodes: locating the best-fitting sphere relative to cortical anatomy". Electroencephalogr Clin Neurophysiol 86 (1): 1–6.
- 41. "Guideline seven: a proposal for standard montages to be used in clinical EEG. American Electroencephalographic Society.". Journal of Clinical Neurophysiology 11 (1): 30–6. 1994. PMID 8195424.
- **42.** Adrian ED, Matthews BHC (1934) The Berger rhythm, potential changes from the occipital lobe in man. Brain 57:345–359.
- **43.** Grey Walter W (1950) Epilepsy. In: Hill JDN, Parr G (eds) Electroencephalography. Macdonald, London, pp 228–272.
- **44.** Gibbs FA, Gibbs EL, Lennox WG (1937) Epilepsy, a paroxysmal cerebral dysrhythmia. Brain 60:377–388.
- **45.** Gibbs FA, Gibbs EL, Lennox WG (1943) Electroencephalographicclassification of epileptic patients and control subjects. Arch Neurol (Chicago) 50:111–128.
- 46. Benbardis SR, Tatum WO (2003) Overinterpretation of EEGs and misdiagnosis of epilepsy. J Clin Neurophysiol 20:42–44.
- 47. Sharbrough F, Chatrian G-E, Lesser RP, Lu¨ders H, Nuwer M,Picton TW (1991) American Electroencephalographic Society guidelines for standard electrode position nomenclature. J Clin Neurophysiol 8:200–202.
- **48.** Flink R, Pedersen B, Guekht AB et al (2002) (ILAE Commission Report). Guidelines for the use of EEG methodology in the diagnosis of epilepsy. Acta Neurol Scand 106:1–7.
- **49.** Kennett RP (2000) Neurophysiologic investigation of adults. In: Oxbury JM, Polkey CE, Duchowny M (eds) Focal Intractable Epilepsy. WB Saunders, London, pp 333–362.
- 50. Halasz P, Filakovszky J, Vargha A, Bagdy G (2002) Effect of sleep deprivation on spikewave discharges in idiopathic generalized epilepsy: a 4 x 24 hour continuous long term EEG monitoring study. Epilepsy Res 51:123–132.
- Luders HO, Comair YG (eds) (2001) Epilepsy Surgery, 2nd edn. Lippincott, Williams and Wilkins, Philadephia.
- **52.** Berg AT, Shinnar S (1991) The risk of seizure recurrence following a first unprovoked seizure: a quantitative review. Neurology 41:965–972.

- 53. Kim LG, Johnson TL, Marson AG, Chadwick DW (2006) Prediction of risk of seizure recurrence after a single seizure and early epilepsy: further results from the MESS trial. Lancet Neurol 5:317–322.
- **54.** Harding GF, Herrick CE, Jeavons PM (1978) A controlled study of the effect of sodium valproate on photosensitive epilepsy and its prognosis. Epilepsia 19:555–565.
- 55. Binnie CD (2001) Cognitive performance, subtle seizures, and the EEG. Epilepsia 42(suppl 1):16–18.
- **56.** Berg AT, Shinnar S (1994) Relapse following discontinuation of antiepileptic drugs: a meta-analysis. Neurology 44:601–608.
- 57. Kaplan PW (2006) The EEG of status epilepticus. J Clin Neurophysiol 23:221–229.
- 58. Reuber M, Pukrop R, Bauer J, Helmstaedter C, Tessendorf N, Elger CE (2003) Outcome in psychogenic nonepileptic seizures: 1- to 10-year follow up in 164 patients. Ann Neurol 53:305–311.
- **59.** Boesebeck F, Freemann S, Kellinghaus C, Evers S (2009) Misdiagnosis of epileptic and non-epileptic seizures in a neurological intensive care unit. Acta Neurol Scand 122:189–195.
- **60.** Nei M, Lee JM, Shanker VL, Sperling MR (1999) The EEG and prognosis in status epilepticus. Epilepsia 40:157–163.
- **61.** De Lorenzo RJ, Waterhouse EJ, Towne AR et al (1998) Persistent nonconvulsive status epilepticus after the control of convulsive status epilepticus. Epilepsia 39:833–840.
- **62.** Rijsdijk M, Leijten FSS, Slooter AJC (2008) Continuous EEG monitoring in the intensive care unit. Neth J Crit Care 12:157–162.
- **63.** Sidhu KS, Balon R, Ajluni V, Boutros NN (2009) Standard EEG and the difficult-to-assess mental status. Ann Clin Psychiatry 21:103–108.
- **64.** Yamashita S, Morinaga T, Ohgo S, Sakamoto T, Kaku N, Sugimoto S, Matsukura S (1995) Prognostic value of electroencephalogram (EEG) in anoxic encephalopathy after cardiopulmonary resuscitation: relationship amoung anoxic period, EEG grading and outcome. Intern Med 34:71–76.
- **65.** Dr Mohammed Ashfaque Tinmaswala, Dr Valinjker S.K, Dr Shilpa Hegde, Dr Parmeshwar Taware Electroencephalographic Abnormalities in First Onset Afebrile and Complex Febrile Seizures and Its Association with Type of Seizures.

- 66. Gronseth, G. S.; Greenberg, M. K. (1995). "The utility of the electroencephalogram in the evaluation of patients presenting with headache: A review of the literature". Neurology. 45 (7): 1263–1267.
- **67.** Jordan, K.G. Nonconvulsive status epilepticus in the neuro-ICU detected by continuous EEG monitoring. Neurology, 1992, 42 (Suppl. 1): 194.
- **68.** Lesser, R.P., Luders, H., Dinner, D.S., Morris, H.H., Hahn, J.F. and Wyllie, E. Extraoperative cortical functional localization in patients with epilepsy. J. Clin. Neurophysiol., 1987, 4 (1): 27–53.
- **69.** Markand, O.N. Electroencephalography in diffuse encephalopathies. J. Clin. Neurophysiol., 1984, 1 (4): 357–407.
- **70.** Nuwer, M.R. Intraoperative electroencephalography. J. Clin. Neurophysiol.,1993, 10 (4): 437–444.
- Nuwer, M.R., Daube, J., Fischer, C., Schramu, J. and Yingling, C.D.Neuromonitoring during surgery. Report of an IFCN committee. Electroenceph.clin. Neurophysiol., 1993, 87: 263–276.
- 72. Ojemann, G.A. Intraoperative tailoring of temporal lobe resections. In: J.Engel, Jr. (Ed.), Surgical Treatment of the Epilepsies, Raven Press, New York. Chapter 39, 2nd edn. Vol. 2, 1993, pp. 481–487.
- **73.** Anderson, J. (22 October 2004) (Hardcover). *Cognitive Psychology and Its Implications* (6th ed.). New York, NY: Worth. p. 17. ISBN 0716701103.
- 74. Creutzfeldt OD, Watanabe S, Lux HD (1966). "Relations between EEG phenomena and potentials of single cortical cells. I. Evoked responses after thalamic and epicortical stimulation". Electroencephalogr Clin Neurophysiol 20 (1): 1–18.
- **75.** Nunez PL, Srinivasan R (1981). *Electric fields of the brain: The neurophysics of EEG* (http://books.google.com/books?id=gu5qAAAMAAJ). Oxford University Press.
- **76.** Hamalainen M, Hari R, Ilmoniemi RJ, Knuutila J, Lounasmaa OV (1993). "Magnetoencphalography - Theory, instrumentation, and applications to noninvasive studies of the working human brain". *Reviews of Modern Physics* **65** (2): 413–497.
- 77. Buzsaki G (2006). Rhythms of the brain. Oxford University Press. ISBN 0195301064.
- 78. Kirmizialsan, E.; Bayraktaroglu, Z.; Gurvit, H.; Keskin, Y.; Emre, M.; Demiralp, T. (2006).
  "Comparative analysis of event-related potentials during Go/NoGo and CPT:
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Decomposition of electrophysiological markers of response inhibition and sustained attention". Brain Research **1104** (1): 114–128.

- 79. Kisley, M. A.; Cornwell, Z. M. (2006). "Gamma and beta neural activity evoked during a sensory gating paradigm: Effects of auditory, somatosensory and cross-modal stimulation". Clinical Neurophysiology 117 (11): 2549–2563.
- Kanayama, N.; Sato, A.; Ohira, H. (2007). "Crossmodal effect with rubber hand illusion and gamma-band activity". Psychophysiology 44 (3): 392–402.
- 81. Gastaut, H. (1952). "Etude electrocorticographique de al reactivite des rhytmes rolandiques". *Rev. Neurol* 87 (2): 176–182. PMID 13014777.
- 82. Oberman, LM; Hubbard, EM; McCleery, JP; Altschuler, EL; Ramachandran, VS; Pineda, JA (2005). "EEG Evidence for mirror neuron dysfunction in autism spectrum disorders". *Cognitive Brain Research* 24 (2): 190–198.
- 83. Vespa, Paul M.; Nenov, Val; Nuwer, Marc R. (1999). "Continuous EEG Monitoring in the Intensive Care Unit: Early Findings and Clinical Efficacy". Journal of Clinical Neurophysiology. 16 (1): 1–13.
- 84. O'Regan, S; Faul, S; Marnane, W (2010). "2010 Annual International Conference of the IEEE Engineering in Medicine and Biology": 6353–6.
- 85. Hämäläinen, Matti; Hari, Riitta; Ilmoniemi, Risto J.; Knuutila, Jukka; Lounasmaa, Olli V. (1993). "Magnetoencephalography-theory, instrumentation, and applications to noninvasive studies of the working human brain". Reviews of Modern Physics. 65 (2): 413–97.
- 86. Schultz, Teal L. (2012). "Technical Tips: MRI Compatible EEG Electrodes: Advantages, Disadvantages, And Financial Feasibility In A Clinical Setting.". Neurodiagnostic Journal 52.1: 69–81.
- **87.** Murphy, Kieran J.; Brunberg, James A. (1997). "Adult claustrophobia, anxiety and sedation in MRI". Magnetic Resonance Imaging. **15** (1): 51–4.
- 88. Yasuno, Fumihiko; Brown, Amira K; Zoghbi, Sami S; Krushinski, Joseph H; Chernet, Eyassu; Tauscher, Johannes; Schaus, John M; Phebus, Lee A; Chesterfield, Amy K; Felder, Christian C; Gladding, Robert L; Hong, Jinsoo; Halldin, Christer; Pike, Victor W; Innis, Robert B (2007). "The PET Radioligand \11C]MePPEP Binds Reversibly and with

High Specific Signal to Cannabinoid CB1 Receptors in Nonhuman Primate Brain". Neuropsychopharmacology. **33** (2): 259–69.

- 89. Mulholland, Thomas (2012). "Objective EEG Methods for Studying Covert Shifts of Visual Attention". In McGuigan, F. J.; Schoonover, R. A. The Psychophysiology of Thinking: Studies of Covert Processes. pp. 109–51. ISBN 978-0-323-14700-2.
- 90. Hinterberger, Thilo; Kübler, Andrea; Kaiser, Jochen; Neumann, Nicola; Birbaumer, Niels (2003). "A brain–computer interface (BCI) for the locked-in: Comparison of different EEG classifications for the thought translation device". Clinica Neurophysiology. 114 (3): 416–25.
- **91.** Schenck, John F. (1996). "The role of magnetic susceptibility in magnetic resonance imaging: MRI magnetic compatibility of the first and second kinds". Medical Physics. **23** (6): 815–50.
- 92. Feinberg, I.; Campbell, I. G. (2012). "Longitudinal sleep EEG trajectories indicate complex patterns of adolescent brain maturation". AJP: Regulatory, Integrative and Comparative Physiology. 304 (4): R296–303.
- **93.** Sereno, SC; Rayner, K; Posner, MI (1998). "Establishing a time-line of word recognition: Evidence from eye movements and event-related potentials". NeuroReport. **9** (10): 2195–200.
- 94. (http://search.japantimes.co.jp/cgi-bin/nb20090401a2.html) 1 Apr 2009, Japan Times.
- **95.** This brain test maps the truth (http:// timesofindia. indiatimes. com/ Cities/ This\_brain\_test\_maps\_the\_truth/ articleshow/ 3257032. cms) 21 Jul 2008, 0348 hrs IST, Nitasha Natu,TNN.
- 96. "Puranik, D.A., Joseph, S.K., Daundkar, B.B., Garad, M.V. (2009). Brain Signature profiling in India. Its status as an aid in investigation and as corroborative evidence as seen from judgments. Proceedings of XX All India Forensic Science Conference, 815 822, November 15–17, Jaipur."
- 97. Schlögl, Alois; Slater, Mel; Pfurtscheller, Gert (2002). "Presence research and EEG"
- **98.** Srinivasan, Ramesh (1999). "Methods to Improve the Spatial Resolution of EEG". International Journal. **1** (1): 102–11.

- 99. Kondylis, Efstathios D. (2014). "Detection Of High-Frequency Oscillations By Hybrid Depth Electrodes In Standard Clinical Intracranial EEG Recordings.". Frontiers in Neurology. 5: 1–10.
- Hämäläinen, Matti; Hari, Riitta; Ilmoniemi, Risto J.; Knuutila, Jukka; Lounasmaa, Olli V. (1993). "Magnetoencephalography—theory, instrumentation, and applications to noninvasive studies of the working human brain". Reviews of Modern Physics. 65 (2): 413–497.
- 101. Murakami, S.; Okada, Y. (13 April 2006). "Contributions of principal neocortical neurons to magnetoencephalography and electroencephalography signals". The Journal of Physiology. 575 (3): 925–936.
- 102. Jung, TP; Makeig, S; Humphries, C; Lee, TW; McKeown, MJ; Iragui, V; Sejnowski, TJ (2000). "Removing electroencephalographic artifacts by blind source separation". *Psychophysiology* 37 (2): 163–178.
- Jung, T.P.; Makeig, S.; Westerfield, M.; Townsend, J.; Courchesne, E.; Sejnowski, T.J. (2000b). "Removal of eye activity artifacts from visual event-related potentials in normal and clinical subjects". *Clinical Neurophysiology* 111 (10): 1745–1758.
- Joyce, Carrie A.; Gorodnitsky, Irina F.; Kutas, Marta (2004). "Automatic removal of eye movement and blink artifacts from EEG data using blind component separation". *Psychophysiology* 41 (2): 313–325.
- 105. Shackman, AJ; McMenamin, BW; Maxwell, JS; Greischar, LL; Davidson, RJ (2010). "Identifying robust and sensitive frequency bands for interrogating neural oscillations". NeuroImage 51 (4): 1319–1333.
- Nolan, H.; Whelan, R.; Reilly, R.B. (2010). "*FASTER*: Fully Automated Statistical Thresholding for EEG artifact Rejection". Journal of Neuroscience Methods 192 (1): 152–162.
- 107. Montez T, Poil S-S, Jones BF, Manshanden I, Verbunt JPA, van Dijk BW, Brussaard AB, van Ooyen A, Stam CJ, Scheltens P, Linkenkaer-Hansen K (2009). "Altered temporal correlations in parietal alpha and prefrontal theta oscillations in early-stage Alzheimer disease" (http://www.pnas.org/content/106/5/1614.abstract). PNAS 106 (5): 1614–1619.

- 108. MURI: Synthetic Telepathy (http://cnslab.ss.uci.edu/muri/index.html). Cnslab.ss.uci.edu. Retrieved 2011-07-19.
- **109.** "Mind Games" (http://www.economist.com/science/displaystory. cfm?story\_id=8847846). The Economist. 2007-03-23.
- **110.** Li, Shan (2010-08-08). "Mind reading is on the market" (http://www.latimes. com/business/la-fi-mind-reader-20100808,0,6235181,full.story). Los Angeles Times.
- 111. "Brains-on with NeuroSky and Square Enix's Judecca mind-control game" (http:// www. engadget. com/ 2008/ 10/ 09/brains-on-with-neurosky-and-squareenixs-judeccamind-control-ga/). Engadget. . Retrieved 2010-12-02.
- 112. "New games powered by brain waves" (http://www.physorg.com/ news150781868.html). Physorg.com. . Retrieved 2010-12-02.
- 113. Klimesch (1996). Memory processes, brain oscillations and EEG synchronization. International Journal of Psychophysiology, 24(1-2), 61-100.
- **114.** Mizuhara, Wang, Kobayashi, & Yamaguchi (2004). A long-range cortical network emerging with theta oscillation in a mental task. Neuroreport, 15(8), 1233.