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## *Full Length Research Article*

# REVIEW ABOUT EEG

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**ABSTRACT**

Electroencephalography (EEG) is an emerging signal in biosignals. It can indicate the electrical activity of brain. Consequently, it can be applied to biosignals acquisition systems to reduce the data rate to realize risk factors and diagnosis performance. They are highly process in nature and it also contains useful information about the brain state. In this review, I have discussed about diseases such as dementia, brain tumor, stoke through EEG signal. Determination of EEG diseases causes, types and treatments. These review also perhaps the EEG diseases in detail.



## INTRODUCTION

### Biosignal

Biosignal may be a summarizing term for all types of signals that may be (continually) measured and monitored from biological beings. The term biosignal is commonly accustomed mean bio-electrical signal however in reality, biosignal refers to each electrical and non-electrical signals. Electrical biosignals ("bio-electrical" signals) area unit typically taken to be (changes in) electrical currents made by the total of electrical potential variations across a specialised tissue, organ or cell system just like the system.

### Types of Bio Signals

Electroencephalogram (EEG)
Electrocardiogram (ECG)
Electromyogram (EMG)
Mechanomyogram (MMG)
Electrooculography (EOG)
Galvanic skin response (GSR)
Magneto encephalogram (MEG)

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EEG, ECG, EOG and electromyogram are measured with differential electronic equipment that registers the distinction between 2 electrodes connected to the skin. However, the galvanic skin response measures resistivity and therefore the million measures the flux induced by electrical currents (Electroencephalogram) of the brain. Electrical currents and changes electrical resistance across tissues can also be measured from plants. Bio-signals may additionally see any non-electrical signal that's capable of being monitored from biological beings, like mechanical signals (e.g. the Mechanomyogram or MMG), acoustic signals (e.g. phonetic and non-phonetic utterances, breathing), chemical signals.

### Electoencephalogram (EEG) Signals

Electroencephalography (EEG) is that the recording of electrical activity on the scalp. Electroencephalogram measures voltage fluctuations ensuing from ionic current flows among the neurons of the brain [1]. In clinical contexts, electroencephalogram refers to the recording of the brain's spontaneous electrical activity over a brief amount of your time, typically 20–40 minutes, as recorded from multiple electrodes placed on the scalp.

**EEG Signal and Information Provide**

Several patients are said a medical specialist to own associate degree graph (EEG), that records electrical impulses from the nerves within the head. “Electro” refers to the electrical impulses sent from one vegetative cell to a different. These impulses are the method nerves confer with one another and acquire info from the brain to the remainder of the body. “Encephalo” refers to the top, and “gram” refers to the written record. Encephalogram exams are done by golf stroke electrodes (detectors of electricity) on the scalp and seeing what the electrical impulses appear as if once the patient is awake, asleep, in an exceedingly area with a flashing light-weight or generally once the patient is asked to breathe deeply over and over.

**EEG Techniques Include**

EEG is most frequently wont to diagnose encephalopathy, that causes obvious abnormalities in graph readings. It's additionally wont to diagnose sleep disorders, coma, encephalopathies, and death. Graph used to be a first-line methodology of diagnosing for tumors, stroke, and alternative focal brain disorders, however this use has weakened with the arrival of high-resolution anatomical imaging techniques like tomography and CT. Despite restricted spacial resolution, graph continues to be a valuable tool for analysis and diagnosing, particularly once millisecond-range temporal resolution (not potential with CT or MRI) is needed.

**Source of EEG Activity**

The brain's electrical charge is maintained by billions of neurons. Neurons area unit electrically charged (or "polarized") by membrance transport proteins that pump ions across their membranes. Neurons area unit perpetually exchanging ions with the animate thing environment, as an example to keep up resting potentials and to propagate action potentials. Ions of comparable charge repel one another, and once several ions area unit pushed out of the many neurons at constant time, they'll push their neighbours, United Nations agency push their neighbours, and so on, in a wave. Scalp EEG activity shows oscillations at a spread of frequencies. Many of those oscillations have characteristic frequency ranges, spatial distributions and area unit related to totally different states of brain functioning (e.g., waking and therefore the varied sleep stages). The neuronal networks underlying some of these oscillations are understood (e.g., the thalamocortical resonance underlying sleep spindles), whereas several others don't seem to be (e.g., the system that generates the posterior basic rhythm). analysis that measures each EEG and vegetative cell spiking finds the link between the 2 is complicated, with a mix of EEG power within the gamma band and innovate the delta band relating most powerfully to vegetative cell spike activity.

**Clinical Use**

A routine clinical EEG recording typically lasts 20–30 minutes (plus preparation time) and usually involves recording from scalp electrodes. Routine EEG is typically used in the following clinical circumstances:

To distinguish epileptic seizures from other types of spells, such as psychogenic non-epileptic seizures, syncope (fainting), sub-cortical movement disorders and migraine variants.

To serve as an adjunct test of brain death

To prognosticate, in certain instances, in patients with coma

To determine whether to wean anti-epileptic medications.

At times, a routine EEG is not sufficient, particularly when it is necessary to record a patient while he/she is having a seizure. In this case, the patient may be admitted to the hospital for days or even weeks, while EEG is constantly being recorded (along with time-synchronized video and audio recording). A recording of an actual seizure (i.e., an ictal recording, rather than an inter-ictal recording of a possibly epileptic patient at some period between seizures) can give significantly better information about whether or not a spell is an epileptic seizure and the focus in the brain from which the seizure activity emanates. Epilepsy monitoring is typically done:

To distinguish epileptic seizures from alternative sorts of spells, like mental non-epileptic seizures, syncope (fainting), sub-cortical movement disorders and migraine variants.

To characterize seizures for the needs of treatment

To localize the region of brain from that a seizure originates for work-up of potential seizure surgery

Additionally, encephalogram could also be accustomed monitor sure procedures*:*

To monitor the depth of physiological state as an indirect indicator of cerebral introduction in arterial blood vessel endarterectomy

To monitor amobarbital impact throughout the Wada take a look at

EEG may be employed in medical care units for brain perform monitoring:

To monitor the impact of sedative/anesthesia in patients in medically induced coma (for treatment of refractory seizures or increased intracranial pressure)

To monitor for secondary brain injury in conditions like subarachnoid hemorrhage (currently a research method)

If a patient with epilepsy is being considered for resective surgery, it's typically necessary to localize the main target (source) of the epileptic brain activity with a resolution bigger than what's provided by scalp encephalogram. This can be as a result of the cerebrospinal fluid, or and scalp smear the electrical potentials recorded by scalp encephalogram. Low voltage, high frequency parts that can't be seen simply (or at all) in scalp EEG can be seen clearly in cardiogram. Further, smaller electrodes (which cover a smaller parcel of brain surface) permit even lower voltage, quicker parts of brain activity to be seen. Electroencephalogram isn't indicated for

designation headache. Continual headache may be a common pain drawback, and this procedure is usually employed in a search for a diagnosing, however it's no advantage over routine clinical analysis.

**Research Use**

EEG, and also the connected study of ERPs are used extensively in neurobiology, science, psychological science, neurolinguistics and psychophysiological analysis. Several encephalogram techniques utilized in analysis don't seem to be standardized sufficiently for clinical use.

**Relative Advantages**

The first human electroencephalogram recording obtained by Hans Berger in 1924. The higher tracing is electroencephalogram, and therefore the lower may be a ten Hertz temporal order signal. Many different strategies to check brain perform exist, as well as useful magnetic resonance imaging (fMRI), antielectron emission imaging, magnetoencephalography (MEG), Nuclear resonance chemical analysis, Electrocorticography, Single-photon emission X-radiation, Near-infrared spectroscopy (NIRS), and Event-related optical signal (EROS). Despite the comparatively poor abstraction sensitivity of electroencephalogram, it possesses multiple benefits over a number of these techniques:

Hardware prices are considerably not up to those of most different techniques [2]. EEG sensors are often employed in a lot of places than functional magnetic resonance imaging, SPECT, PET, MRS, or MEG, as these techniques need large and immobile instrumentation. As an example, meg needs instrumentation consisting of liquid helium-cooled detectors which will be used solely in magnetically secure rooms, altogether cost accounting upwards of many million dollars [3] and functional magnetic resonance imaging needs the employment of a 1-ton magnet in, again, a protected area. EEG has terribly high temporal resolution, on the order of milliseconds instead of seconds. Electroencephalogram is often recorded at sampling rates between 250 and 2000 Hz in clinical and analysis settings. However fashionable electroencephalogram information assortment systems are capable of recording at sampling rates on top of 20,000 Hz if desired. MEG and eros are the only different noninvasive cognitive neuroscience techniques that acquire information at this level of temporal resolution [3].

EEG is comparatively tolerant of subject movement, not like most different neuroimaging techniques. There even exist ways for minimizing, and even eliminating movement artifacts in encephalogram data [4] EEG doesn't claustrophobia simple phobia, not like functional magnetic resonance imaging, PET, MRS, SPECT, and typically MEG [5] EEG doesn't involve exposure to high-intensity (>1 Tesla) magnetic fields, as in a number of the opposite techniques, particularly magnetic resonance imaging and MRS. These will cause a range of undesirable problems with the info, and additionally prohibit use of those techniques with participants that have metal implants in their body, like metal-containing pacemakers[6]
EEG doesn't involve exposure to radioligands, not like positron emission tomography [7]. ERP studies is conducted with comparatively easy paradigms, compared with that is block-design MRI studies

**Relative Disadvantages**

Low spatial resolution on the scalp functional magnetic resonance imaging, for instance, will directly show areas of the brain that are active, whereas encephalogram needs intense interpretation simply to hypothesise what areas are activated by a specific response [13]. EEG poorly determines neural activity that happens below the higher layers of the brain (the cortex). Unlike PET and MRS, cannot establish specific locations within the brain at that numerous neurotransmitters, drugs, etc. may be found [7]. Often takes an extended time to attach a topic to electroencephalogram, because it needs precise placement of dozens of electrodes round the head and also the use of various gels, saline solutions, and/or pastes to stay them in place. Signal-to-noise ratio is poor, thus refined information analysis and comparatively large numbers of subjects are required to extract helpful data from EEG[14].

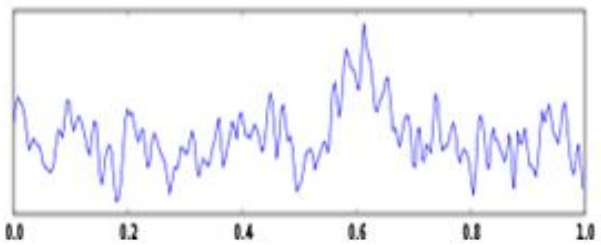
**Combining EEG with other Neuroimaging Techniques**

Simultaneous electroencephalogram recordings and functional magnetic resonance imaging scans are obtained successfully[15][16], although successful coincidental recording needs that many technical difficulties be overcome, like the presence of ballistocardiographic unit, magnetic resonance imaging pulse unit and also the induction of electrical currents in electroencephalogram wires that move inside the sturdy magnetic fields of the MRI. Whereas difficult, these are with success overcome during a variety of studies [17]. Similarly, simultaneous recordings with meg and electroencephalogram have additionally been conducted, that has many benefits over using either technique alone:

EEG needs correct data regarding sure aspects of the skull that may only be calculable, like skull radius, and conductivities of various skull locations. MEG doesn't have this issue, and a simultaneous analysis permits this to be corrected for. MEG and EEG each find activity below the surface of the cortex very poorly, and like EEG, the amount of error will increase with the depth below the surface of the cortex one makes an attempt to look at. However, the errors are very totally different between the techniques, and mixing them so permits for correction of a number of this noise. MEG has access to just about no sources of brain activity below a number of centimeters below the cortex. EEG, on the opposite hand, will receive signals from larger depth, albeit with a high degree of noise. Combining the two makes it easier to see what within the electroencephalogram signal comes from the surface (since meg is extremely correct in examining signals from the surface of the brain), and what comes from deeper within the brain, therefore allowing analysis of deeper brain signals than either electroencephalogram or meg on its own[18]. Electroencephalogram has additionally been combined with positron emission imaging. This provides the advantage of permitting researcers to visualize what electroencephalogram signals are related to completely different drug actions within the brain [19].

**Normal Activity**

The electroencephalogram is often represented in terms of (1) rhythmical activity and (2) transients. The rhythmical activity is split into bands by frequency.




**5.1 Fig: Normal activity EEG signal**

To some degree, these frequency bands are a matter of nomenclature (i.e., any rhythmic activity between 8–12 Hz can be described as "alpha"), but these designations arose because rhythmic activity within a certain frequency range was noted to have a certain distribution over the scalp or a certain biological significance. Frequency bands are usually extracted using spectral methods (for instance Welch) as implemented for instance in freely available EEG software such as EEGLAB or the the neurophysiological biomarker toolbox. Most of the cerebral signal observed in the scalp EEG falls in the range of 1–20 Hz

**Abnormal Activity**

Abnormal activity will loosely be separated into epileptiform and non-epileptiform activity. It may be separated into focal or diffuse. These will occur as interictal activity, between seizures, and represent a part of cortical irritability that will be predisposed to producing epileptic seizures. Interictal discharges aren't wholly reliable for determining neither whether a patient has epilepsy nor where his /her seizure might originate. Generalized epileptiform discharges often have an anterior maximum, but these are seen synchronously throughout the entire brain. They are strongly suggestive of a generalized epilepsy. Focal non-epileptiform abnormal activity may occur over areas of the brain where there is focal damage of the cortex or white matter. Intracortical Encephalogram electrodes and sub-dural electrodes can be used in tandem to discriminate and discretize artifact from epileptiform and other severe neurological events. More advanced measures of abnormal EEG signals have also recently received attention as possible biomarkers for different disorders such as Alzheimer's disease [20].

**Disorders**

**Dementia**

Dementia may be a broad class of brain diseases that cause future loss of the flexibility to assume and reason clearly that's severe enough to have an effect on an individual's daily functioning. For the identification to be present it should be amendment from however the person was previously [21].
The foremost common sort of dementia is Alzheimer's disease (75%) [21]. Different forms include Lewy body dementedness, vascular dementia, frontotemporal dementedness, progressive supranuclear palsy, corticobasal degeneration, ancient pressure abnormality and Creutzfeldt–Jakob ill health. Dementia becomes additional common with age. Whereas solely third of individuals between the ages of 65–74 have dementedness, forty seventh of individuals over the age of eighty five have some style of dementedness [21]. As additional individuals live longer, dementedness is changing into additional common.

**Signs and Symptoms**

Dementia affects the brain's ability to suppose reason and keep in mind clearly. The foremost common affected areas with memory, visual-spatial, language, attention, and executive perform (problem solving). Most styles of mental disease are slow and progressive. It's potential for a patient to possess two varieties of dementia at identical time. Regarding 100% of individuals with insanity have what's called mixed dementia, that is sometimes a mixture of {Alzheimer’s disease Alzheimer’s disease|Alzheimer’s|Alzheimers|presenile dementia} and another variety of dementia like frontotemporal dementia or vascular dementia [22][23]. Further psychological and behavioral issues that usually have an effect on those that have insanity include:

Disinhibition and impulsivity
Depression and/or anxiety
Agitation
Balance issues and Tremor
Speech and language issue
Trouble consumption or swallowing
Delusions (often basic cognitive process individuals are stealing from them) or hallucinations

Memory distortions (believing that a memory has already happened once it's not, thinking an recent memory may be a new one, combining two recollections, or confusing the individuals in an exceedingly memory)once people with dementia are place in circumstances on the far side their talents, there is also a explosive modification to tears or anger (a "catastrophic reaction")[24].Depression affects 20–30% of people who have dementia, and concerning two hundredth have anxiety[25].Psychosis (often delusions of persecution) and agitation/aggression additionally typically accompany dementia. Every of those should be assessed and treated severally of the underlying dementia [26].

**Mild Cognitive Impairment**

Within the 1st stages of dementia, the signs and symptoms of the illness is also delicate. Often, the first signs of dementia only become apparent once trying back in time. The earliest stage of dementia (actually, it's not even insanity, it may well be thought-about pre-dementia) is named gentle psychological feature impairment (MCI) (discussed in additional detail later). Seventieth of these diagnosed with MCI can attain dementia at some point [21]. In MCI, changes within the person's brain are happening for an extended time; however the symptoms of the illness are simply commencing to show. An individual with MCI can score between twenty seven and thirty on the Mini-Mental State Examination (MMSE) that may be a traditional score. They will have some memory hassle and hassle finding words however they solve everyday issues and handle their own life affairs well.

**Early Stages**

In the early stage of dementia, the person can begin to indicate symptoms noticeable to the people around them. Additionally, the symptoms begin to interfere with daily activities.

**Middle Stages**

As dementia progresses, the symptoms first experienced within the early stages of the dementia generally worsen. The speed of decline is different for every person. The person cannot sometimes operate outside of his or her own residence, and usually shouldn't be left alone. He or she is also able to do easy chores round the house however not abundant else and begins to need help for private care and hygiene aside from easy reminders [21].

**Late Stages**

In the late stages of dementia, symptoms worsen. People tend to want twenty four hour supervising. They will wander, have hallucinations, fall or become unable to manage their bladder or bowels (incontinent). He or she might have lost the flexibility to eat or swallow. The person can would like help with just about each side of lifestyle. Their appetence might decline to the purpose that the person doesn't wish to delapidate all [21]. He or she might not wish to or be able to get out of bed. If they need memory issues they will now not acknowledge acquainted individuals. The person is also agitated. He or she might have vital changes in sleeping habits or have bother sleeping at al l[21].

**Cause**

**Reversible Causes Of Dementia**

There are four main causes of easily reversible dementia: hypothyroidism, Vitamin B12 deficiency, Lyme disease, and neurosyphillis. All people with memory difficulty should be checked for hypothyroidism and B12 deficiency. For Lyme disease and neurosyphilis, testing should be done if there are risk factors for those diseases in the person [21].

**Mild Cognitive Impairmen**

Mild cognitive impairment basically means that the person is exhibiting memory or thinking difficulties, but it is not severe enough yet to be given a diagnosis. He or she should score between 25-30 on the MMSE. Around 70% of people with MCI will go on to develop some form of dementia [21]. MCI is mostly divided into two classes. the primary is one that's primarily memory loss (amnestic MCI). The second class is something that's not primarily memory difficulties (non-amnestic MCI).

**Fixed Cognitive Impairment**

Various sorts of brain injury could cause irreversible psychological feature impairment that may not get worse over time. Traumatic brain injury could cause generalized damage to the nervous tissue of the brain (diffuse axonal injury), or a lot of localized injury (as additionally could neurosurgery).
A temporary reduction within the brain's provide of blood or chemical element might cause hypoxic-ischemic injury. Strokes (ischemic stroke, or neural structure, subarachnoid, subdural or extradural hemorrhage) or infections (meningitis and/or encephalitis) moving the brain, prolonged epileptic seizures and acute hydrocephaly may additionally have long effects on knowledge. Excessive alcohol use might cause alcohol dementia, Wernicke's encephalopathy and/or dementia.

**Alzheimer's Disease**

Alzheimer's disease is that the most typical sort of dementia [27]. Its most typical symptoms are memory loss and word-finding difficulties. People with Alzheimer's even have trouble with visual-spatial areas (for example they will begin to urge lost often), reasoning, judgement, and insight. Insight refers as to whether or not the person realizes he/she has memory issues. Common early symptoms of Alzheimer's embody repetition, obtaining lost, difficulties keeping track of bills, issues with preparation especially new or sophisticated meals, forgetting to require medication, and word-finding issues.

Vascular Dementia

Vascular dementia could be a sort of dementia that's caused by disease or injury to blood vessels within the brain, largely strokes. The precise symptoms of this dementia rely upon wherever within the brain the strokes have occurred and whether or not the vessels are large or small [21].

**Diagnosis**

As seen higher as, there are several specific varieties and causes of dementia, usually showing slightly completely different symptoms. However, the symptoms are very similar and it's sometimes troublesome to diagnose the kind of dementia by symptoms alone. In several cases, the identification cannot be fully certain except with a brain diagnostic assay, however this is} very seldom suggested (though it can be performed at autopsy). In those that are becoming older, general screening for psychological feature impairment mistreatment psychological feature testing or early identification of dementia has not been shown to boost outcomes. However, it's been shown that screening exams are helpful in those people over the age of sixty five with memory complaints

**Brain Tumours**

A brain tumor or intracranial neoplasm occurs when abnormal cells form within the brain [28].There is two main types of tumors: malignant or cancerous tumors and benign tumors [28].

**Brain Tumours are Classed as Either Primary or Secondary**

Cancerous tumors can be divided into primary tumors that started within the brain and those that spread from somewhere else known as brain metastasis tumors [29]. All types of brain tumors may produce symptoms that vary depending on the part of the brain involved [29]. These may include headaches, seizures, problem with vision, vomiting, and mental changes [29]. The headache is classically worst in the morning and goes away with vomiting [28] .More specific problems may include difficulty in walking, speaking and with sensation [29][30]. As the disease progresses unconsciousness may occur[30]

**Types of Primary Brain Tumour**

Tumors are typically benign or malignant, will occur in several components of the brain, and may or may not be primary tumors. A primary growth is one that has started at intervals the brain, as essential a growth, that are some things that has unfold to the brain from another a region of the body[31]. The incidence of biological process tumors are scores of prevailing than primary tumors by 4:1[32]. Tumors might or might not be symptomatic: some tumors are discovered as a results of the patient has symptoms, others show up incidentally on associate imaging scan, or at associate autopsy.

The most common primary brain tumors are [33]
Gliomas (50.4%)
Meningiomas (20.8%)
Pituitary adenomas (15%)
Nerve sheath tumors (8%).

**Signals and Symptoms of Brain Tumours**

The exact symptoms you have will depend upon several factors, as well as the scale of the neoplasm and its position in your brain. Some brain tumours don't cause any symptoms and should solely be discovered out of the blue. The most common symptoms of brain tumours are headaches and seizures (fits). If you have a headache, it may be worse at night and early in the morning, but wears off as the day goes on. You may also feel sick or vomit and have blurred vision. These symptoms can be caused by increased pressure in your skull from the tumour – this can happen even if your tumour is benign. These symptoms aren't always caused by a brain tumour but if you have them, see your GP. More common causes of symptoms like this may be migraine headaches or stroke.

**Causes of Brain Tumours**

Aside from exposure to vinyl chloride or radiation, there are not any familiar environmental factors related to brain tumors. Mutations and deletions of alleged tumour suppressor genes, like TP53, are thought to be the reason behind some varieties of brain tumors [34]. people with varied inherited diseases, like Von Hippel-Lindau syndrome, multiple endocrine pathological process, and autosomal dominant disease sort a pair of are at high risk of developing brain tumors. Although studies haven't shown any link between cellular phone radiation and brain tumors [35] the globe Health Organization has classified portable radiation on the IARC scale into cluster 2B – probably malignant neoplastic disease. Which means that there "could be some risk" of carcinogenicity, therefore further analysis into the long, serious use of mobile phones must be conducted [36].

**Diagnosis of Brain Tumours**

Your GP will ask you about your symptoms and examine you. He or she may carry out tests to assess your reflexes, co-ordination, muscle strength, memory and vision. You may have the following tests to confirm diagnosis and to find out what type of brain tumour you have.

**Blood tests –** these assess your general health and check for specific chemical markers in your blood.

**CT scan –** this uses X-rays to make a three-dimensional image of your brain.

**MRI scan –** this uses magnets and radio waves to produce images of the inside of your brain.

You may need to have a biopsy (where a small sample of tissue is removed) to find out the type and grade of your tumour. Your doctor will use your CT and MRI scans to accurately find the position of the tumour. The biopsy will then be sent to a laboratory for testing. Your treatment will be planned according to the type and grade of brain tumour you have.

**Treatment of Brain Tumours**

Because completely different brain tumours develop in several ways in which, your treatment can vary betting on which kind you have got, its size and grade, and position in your brain.
Betting on the kind of growth you have got, the doctors and nurses taking care of you'll discuss your treatment choices in additional detail and provides you extra recommendation and knowledge therefore you'll be able to build a choice regarding what treatment you would like to own.

**Watchful waiting**

If your tumour is slow-growing and not causing many symptoms, you will not need any treatment at once. Your condition will be monitored closely with routine check-ups and scans. This is often called active monitoring or watchful waiting.

**Surgery**

The aim of surgery is to induce rid of the maximum amount of the tumour as possible. Malignant brain tumours can be difficult to remove completely, but your surgeon is still likely to recommend taking out as much of it as possible.

**Non-Surgical Treatment**

**Radiotherapy**

Radiotherapy uses a targeted beam of radiation to destroy your tumour while minimising the damage to the surrounding healthy tissue. Radiotherapy is usually used either after surgery to kill any remaining tumour cells, or as an alternative to surgery.

**Chemotherapy**

Chemotherapy uses medicines to destroy cancer cells. You usually have it as tablets or by injection. Only a few chemotherapy medicines are effective at treating brain tumours – the most commonly used are temozolomide or a combination of three medicines called procarbazine, lomustine (also called CCNU) and vincristine. This treatment might also be used if your tumour comes back. It's possible that once your surgeon is removing your tumour, he or she is going to

place little implants (called wafers) that release therapy medicines into the affected space of your brain to kill any remaining tumour cells.

**Steroids**

Steroids are hormones (chemicals) that your body makes to assist reduce swelling. Synthetic (man-made) steroids will facilitate to reduce swelling that will be caused by your brain tumor, surgery or radiation therapy.

**New treatments**

New treatments for brain tumours are being tested in clinical trials all the time. Some of these acts like chemotherapy medicines and others affect brain tumours in different ways. You may be given these as tablets or injections. It's possible that you will be able to take part in a clinical trial to test one of these new treatments – speak to your doctor for more information.

**Stroke**

A stroke happens once the blood offer to a part of your brain is interrupted or severely reduced, depriving brain tissue of gas and food. Inside minutes, brain cells begin to die. A stroke may be a medical emergency. Prompt treatment is crucial. Early action will minimize brain injury and potential complications. The great news is that strokes are often treated and prevented, and lots of fewer Americans die of stroke currently than even fifteen years past.

**Classification**

Strokes is assessed into two major categories: anemia and hemorrhagic[37].Ischemic strokes square measure caused by interruption of the blood give, whereas hurt strokes result from the rupture of a vessel or associate abnormal anatomical structure. Relating to eighty seven of strokes square measure anemia, the rest square measure hurt. Some hemorrhages develop among areas of anemia ("hemorrhagic transformation"). Its unknown what variety hurt strokes very begin as anemia stroke [38].

**Signs Snd Symptoms**

Stroke symptoms usually begin suddenly, over seconds to minutes, and in most cases don't progress additional. The symptoms depend upon the realm of the brain affected. The additional in depth the realm of brain affected, the lot of functions that are probably to be lost. Some types of stroke will cause further symptoms.

**Early recognition**

Various systems are planned to extend recognition of stroke. Sudden-onset face weakness, arm drift (i.e., if an individual, once asked to boost each arms, involuntarily lets one arm drift downward) and abnormal speech square measure the findings presumably to guide to the right identification of a case of stroke increasing the probability by five, once a minimum of one amongst these is present). Similarly, once all 3 of those square measure absent, the probability of stroke is considerably shrunken (– probability magnitude relation of zero.39)[39] Proposed systems embody quick (face, arm, speech, and time),[40] as advocated by the Department of Health (United Kingdom) and also the Stroke Association, the American Stroke Association, the National Stroke Association (US), the los angeles Prehospital Stroke Screen (LAPSS)[41] and also the metropolis Prehospital Stroke Scale(CPSS)[42] Use of those scales is suggested by skilled guidelines[43].

**Causes**

A stroke happens once the blood provide to your brain is interrupted or reduced. This deprives your brain of chemical element and nutrients, which might cause your brain cells to die. A stroke could also be caused by a blocked artery (ischemic stroke) or a leaky or burst vessel (hemorrhagic stroke). Some individuals might expertise a short lived disruption of blood flow through their brain (transient anemia attack, or TIA).

**Ischemic Stroke**

Regarding eighty five percent of strokes are anemia strokes. Ischaemic strokes occur once the arteries to your brain become narrowed or blocked, inflicting severely reduced blood flow (ischemia). The foremost common ischaemic strokes include:

*Thrombotic stroke*: In thrombotic stroke a coagulum [44] (blood clot) typically forms around atherosclerotic plaques. Two forms of occlusion will cause stroke:

Large vessel illness involves the common and internal carotids, vertebral, and therefore the Circle of Willis [45]. Diseases that will type thrombiin the big vessels include (in descendent incidence): arteriosclerosis, constriction (tightening of the artery), aortic, carotid or arterial blood vessel dissection, varied inflammatory diseases of the vessel wall (Takayasu redness, giant cell redness, vasculitis), unexciting vasculopathy, Moyamoya malady and fibromuscular abnormality

Small vessel illness involves the smaller arteries within the brain: branches of the circle of Willis, middle arterial cerebral, stem, and arteries arising from the distal bone and basilary artery[46].Diseases that will type thrombi within the little vessels embody (in descendent incidence): lipohyalinosis (build-up of fatty hyaline matter within the vessel as a results of high blood pressurea} and aging) and fibrinoid degeneration (stroke involving these vessels are called lacunar infarcts) and microatheroma (small arterial sclerosis plaques)[47].

*clot stroke*. Viscus causes are often distinguished between high and low-risk [48]:

High risk: fibrillation and attack fibrillation, rheumatic illness of the mitral or aortal valve disease, artificial heart valves, famous cardiac thrombus of the atrium or ventricle, sick sinus syndrome, sustained chamber flutter, recent myocardial infarct, chronic myocardial infarct along with ejection fraction <28 %, symptomatic symptom heart disease with ejection fraction <30 %, expanded myocardiopathy, Libman-Sacks carditis, Marantic carditis, infective carditis, outgrowth fibroelastoma, left chamber nonmalignant neoplasm and artery bypass graft (CABG) surgery.

Low risk/potential: calcification of the annulus (ring) of the atrioventricular valve, patent gap ovale (PFO), chamber septate aneurism, chamber septate aneurism with patent opening ovale, left aneurysm while not coagulum, isolated left chamber "smoke" on diagnostic technique (no stenosis or chamber fibrillation), advanced fat within the aorta or proximal arch

**Silent stroke**

A silent stroke could be a stroke that doesn't have any outward symptoms, and therefore the patients are generally unaware they need suffered a stroke Despite not inflicting classifiable symptoms, a silent stroke still damages the brain, and places the patient at accumulated risk for every transient anemia attack and major stroke among the longer term. Conversely, people who have suffered a significant stroke are at risk of obtaining silent strokes [49]. During a broad study in 1998, quite eleven million individuals were calculable to possess experienced a stroke within the United States. More or less 770,000 of those strokes were symptomatic and eleven million were first-ever silent imaging infarcts or hemorrhages. Silent strokes usually cause lesions that are detected via the employment of neuroimaging like imaging. Silent strokes are calculable to occur at 5 times the speed of symptomatic strokes [50][51]. the danger of silent stroke will increase with age, however can also have an effect on younger adults and youngsters, particularly those with acute anemia[50][52].

**Risk Factors**

Several factors will increase your risk of a stroke. Some factors may also increase your probabilities of getting a attack. Stroke risk factors include:

**Lifestyle risk factors**

Being overweight or rotund
Physical inactivity
Heavy or binge drinking
Use of illicit medicine like cocain and methamphetamines

**Other Risk Factors**

Personal or case history of stroke, attack or transient ischaemic attack. Being age fifty five or older.

**Race —** African-Americans have higher risk of stroke than do individuals of alternative races.

**Gender —** Men have the next risk of stroke than ladies. Ladies square measure sometimes older after they have strokes and those they square measure additional probably to die of strokes than square measure men.

## DISCUSSION

EEG signals were captured from the scalp of the brain and measured in responds to various stimuli. Here, we investigate the use of brain signatures as a possible biometric authentication technique. In this review paper, brain EEG signal have been show that individuals exhibit unique brain patterns for similar tasks.

## REFERENCES


1. Niedermeyer E. and da Silva F.L. (2004). *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*. Lippincot Williams & Wilkins. ISBN 0-7817-5126-8.[*page needed*]
2. 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. doi:10.1097/00004691-199901000-00001. PMID 10082088.
3. Hämäläinen, Matti; Hari, Riitta; Ilmoniemi, Risto J.; Knuutila, Jukka; Lounasmaa, Olli V. (1993). "Magneto encephalography-theory, instrumentation, and applications to noninvasive studies of the working human brain". *Reviews of Modern Physics* 65 (2): 413–97.Bibcode:1993RvMP...65..413H.doi:10.1103/RevModPhys.65.413.
4. O'Regan, S; Faul, S; Marnane, W (2010). "Automatic detection of EEG artifacts arising from head movements".*2010 Annual International Conference of the IEEE Engineering in Medicine and Biology*. pp. 6353–6.doi:10.1109/IEMBS.2010.5627282. ISBN 978-1-4244-4123-5.
5. Murphy, Kieran J.; Brunberg, James A. (1997). "Adult claustrophobia, anxiety and sedation in MRI". *Magnetic Resonance Imaging* 15 (1): 51–4. doi:10.1016/S0730-725X(96)00351-7. PMID 9084025.
6. 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. doi:10.1118/1.597854.PMID 8798169.
7. 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.doi:10.1038/sj.npp.1301402. PMID 17392732.
8. 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.
9. 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". *Clinical Neurophysiology* 114 (3): 416–25.doi:10.1016/S1388-2457(02)00411-X. PMID 12705422.
10. Sereno, SC; Rayner, K; Posner, MI (1998). "Establishing a time-line of word recognition: Evidence from eye movements and event-related potentials". *Neuro Report* 9(10): 2195–200. doi:10.1097/00001756-199807130-00009.PMID 9694199.
11. 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.doi:10.1152/

ajpregu.00422.2012. PMC 3567357.PMID 23193115. Lay summary – *Science Daily* (March 19, 2013).
12. http://www.ct.gov/ceq/cwp/view.asp?a=987&q=249438 [*full citation needed*]
13. Srinivasan, Ramesh (1999). "Methods to Improve the Spatial Resolution of EEG". *International Journal* 1 (1): 102–11.
14. Schlögl, Alois; Slater, Mel; Pfurtscheller, Gert (2002)."Presence research and EEG".
15. Horovitz, Silvina G.; Skudlarski, Pawel; Gore, John C. (2002). "Correlations and dissociations between BOLD signal and P300 amplitude in an auditory oddball task: A parametric approach to combining fMRI and ERP". *Magnetic Resonance Imaging* 20 (4): 319–25.doi: 10.1016/S0730-725X(02)00496-4. PMID 12165350.
16. Laufs, H; Kleinschmidt, A; Beyerle, A; Eger, E; Salek-Haddadi, A; Preibisch, C; Krakow, K (2003). "EEG-correlated fMRI of human alpha activity". *NeuroImage* 19(4): 1463–76. doi:10.1016/S1053-8119(03)00286-6.PMID 12948703.
17. Difrancesco, Mark W.; Holland, Scott K.; Szaflarski, Jerzy P. (2008). "Simultaneous EEG/Functional Magnetic Resonance Imaging at 4 Tesla: Correlates of Brain Activity to Spontaneous Alpha Rhythm During Relaxation". *Journal of Clinical Neurophysiology* 25 (5): 255–64.doi:10.1097/WNP.0b013e3181879d56. PMC 2662486.PMID 18791470.
18. Huizenga, HM; Van Zuijen, TL; Heslenfeld, DJ; Molenaar, PC (2001). "Simultaneous MEG and EEG source analysis". *Physics in medicine and biology* 46 (7): 1737–51.doi:10.1088/0031-9155/46/7/301. PMID 11474922.
19. Schreckenberger, Mathias; Lange-Asschenfeldt, Christian; Lochmann, Matthias; Mann, Klaus; Siessmeier, Thomas; Buchholz, Hans-Georg; Bartenstein, Peter; Gründer, Gerhard (2004). "The thalamus as the generator and modulator of EEG alpha rhythm: A combined PET/EEG study with lorazepam challenge in humans". *NeuroImage*22 (2): 637–44. doi:10.1016/ j.neuroimage.2004.01.047.PMID 15193592.
20. Montez, Teresa; Poil, S.-S.; Jones, B. F.; Manshanden, I.; Verbunt, J. P. A.; Van Dijk, B. W.; Brussaard, A. B.; Van Ooyen, A.; Stam, C. J.; Scheltens, P.; Linkenkaer-Hansen, K. (2009). "Altered temporal correlations in parietal alpha and prefrontal theta oscillations in early-stage Alzheimer disease". *Proceedings of the National Academy of Sciences* 106 (5): 165–70.Bibcode: 2009PNAS..106.1614M.doi:10.1073/pnas.0811699106. PMC 2635782.PMID 1916457
21. Solomon, Andrew E. Budson, Paul R. (2011). *Memory loss: a practical guide for clinicians*. [Edinburgh?]: Elsevier Saunders. ISBN 9781416035978.
22. What is vascular dementia? Alzheimer's Society.
23. Lee AY (2011). "Vascular dementia". *Chonnam Med J*47 (2): 66–71. doi:10.4068/cmj.2011.47.2.66.PMC 3214877. PMID 22111063.
24. Geddes, John; Gelder, Michael G.; Mayou, Richard (2005). *Psychiatry*. Oxford [Oxfordshire]: Oxford University Press. p. 141. ISBN 0-19-852863-9. OCLC 56348037
25. Calleo J, Stanley M (2008). "Anxiety Disorders in Later Life Differentiated Diagnosis and Treatment Strategies". *Psychiatric Times* 25 (8).
26. Shub, Denis; Kunik, Mark E (April 16, 2009). "Psychiatric Comorbidity in Persons With Dementia: Assessment and Treatment Strategies". *Psychiatric Times* 26 (4).
27. Thompson, S.B.N. "Dementia and memory: a handbook for students and professionals" Aldershot: Ashgate 2006.
28. "General Information About Adult Brain Tumors". *NCI*. 2014-04-14. Retrieved 8 June 2014.
29. "Adult Brain Tumors Treatment". *NCI*. 2014-02-28. Retrieved 8 June 2014.
30. *World Cancer Report 2014*. World Health Organization. 2014. pp. Chapter 5.16. ISBN 9283204298
31. "What you need to know about brain tumors". National Cancer Institute. Retrieved 25 February 2012.
32. Merrel RT (Dec 2012). *Dis Mon.* 58 (12): 678–89.doi:10.1016/j.disamonth.2012.08.009. PMID 23149521.
33. Park, Bong Jin; Kim, Han Kyu; Sade, Burak; Lee, Joung H. (2009). "Epidemiology". In Lee, Joung H. *Meningiomas: Diagnosis, Treatment, and Outcome*. Springer. p. 11.ISBN 978-1-84882-910-7.
34. Kleihues P, Ohgaki H, Eibl RH, Reichel MB, Mariani L, Gehring M, Petersen I, Höll T, von Deimling A, Wiestler OD, Schwab M (1994). "Type and frequency of p53 mutations in tumors of the nervous system and its coverings". *Molecular Neuro-oncology and Its Impact on the Clinical Management of Brain Tumors*. Recent results in cancer research 135. Springer. pp. 25–31. ISBN 3540573518.
35. Frei P, Poulsen AH, Johansen C, Olsen JH, Steding-Jessen M, Schüz J (19 October 2011). "Use of mobile phones and risk of brain tumours: update of Danish cohort study.". *BMJ (Clinical research ed.)*343: d6387. Doi:10.1136/bmj.d6387.PMC 3197791. PMID 22016439.
36. "IARC classifies radiofrequency electromagnetic fields as possibly carcinogenic to humans" (PDF).*World Health Organization press release N° 208*(Press release). International Agency for Research on Cancer. 31 May 2011. Retrieved 2 June 2011.
37. "Brain Basics: Preventing Stroke". National Institute of Neurological Disorders and Stroke. Retrieved 2009-10-24.
38. Donnan GA, Fisher M, Macleod M, Davis SM (May 2008). "Stroke". *Lancet* 371 (9624): 1612–23. doi:10.1016/S0140-6736(08)60694-7. PMID 18468545.
39. Goldstein LB, Simel DL (May 2005). "Is this patient having a stroke?". *JAMA* 293 (19): 2391–402.doi:10.1001/jama.293.19.2391. PMID 15900010.
40. Harbison J, Massey A, Barnett L, Hodge D, Ford GA (June 1999). "Rapid ambulance protocol for acute stroke". *Lancet* 353 (9168): 1935. doi:10.1016/S0140-6736(99)00966-6. PMID 10371574.
41. Kidwell CS, Saver JL, Schubert GB, Eckstein M, Starkman S (1998). "Design and retrospective analysis of the Los Angeles Prehospital Stroke Screen (LAPSS)". *Prehospital Emergency Care* 2 (4): 267–73.doi:10.1080/ 10903129808958878. PMID 9799012.
42. Kothari RU, Pancioli A, Liu T, Brott T, Broderick J (April 1999). "Cincinnati Prehospital Stroke Scale: reproducibility and validity". *Annals of Emergency Medicine* 33 (4): 373–8. doi:10.1016/S0196-0644(99) 70299-4.PMID 10092713.

43. National Institute for Health and Clinical Excellence. *Clinical guideline 68: Stroke*. London, 2008.
44. "Thrombus". *Medline Plus*. U.S. National Library of Medicine
45. "Circle of Willis". The Internet Stroke Center.
46. "Brain anaurysm - Introduction". NHS Choices.
47. Fisher CM (1968-12-18). "The arterial lesions underlying lacunes". *Acta Neuropathologica* 12 (1): 1–15.doi: 10.1007/BF00685305. PMID 5708546.
48. Ay H, Furie KL, Singhal A, Smith WS, Sorensen AG, Koroshetz WJ (November 2005). "An evidence-based causative classification system for acute ischemic stroke". *Annals of Neurology* 58 (5): 68897.doi: 10.1002/ana.20617.
49. Miwa K, Hoshi T, Hougaku H, Tanaka M, Furukado S, Abe Y, Okazaki S, Sakaguchi M, Sakoda S, Kitagawa K (2010). "Silent cerebral infarction is associated with incident stroke and TIA independent of carotid intima-media thickness". *Intern. Med.* 49 (9):81722. doi:10.2169/internalmedicine.49.3211. PMID 20453400.
50. Herderscheê D, Hijdra A, Algra A, Koudstaal PJ, Kappelle LJ, van Gijn J (September 1992). "Silent stroke in patients with transient ischemic attack or minor ischemic stroke. The Dutch TIA Trial Study Group". *Stroke* 23 (9): 12204. doi:10.1161/01.STR.23.9.1220. PMID 1519274.
51. Leary MC, Saver JL (2003). "Annual incidence of first silent stroke in the United States: a preliminary estimate". *Cerebrovasc. Dis.* 16 (3):2805. doi:10.1159/000071128. PMID 12865617.
52. Vermeer SE, Koudstaal PJ, Oudkerk M, Hofman A, Breteler MM (January 2002). "Prevalence and risk factors of silent brain infarcts in the population-based Rotterdam Scan Study". *Stroke* 33 (1): 215.doi:10.1161/ hs0102.101629. PMID 11779883.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