Risk Level Classification Dementia or Determination of Dementia via Chaos Technique

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ABSTRACT

This paper aims to classify and determine Dementia, which is a collective term used to describe the problems that people with various underlying brain disorders or damage can have with their memory, language, and thinking. Alzheimer’s disease is the best known and most common disorder under the umbrella of dementia. It measures brain function in the hope of determining and differentiating certain functional conditions of the brain. It is used in patients with cognitive dysfunction involving either a general decline of overall brain function or a localized or lateralized deficit. The raw EEG signals are sampled and parameter like energy, variance, peaks, sharp and spikes waves, duration, events and covariance are detected. In this classification, it provides a better way to diagnose dementia patients. It also aims to safeguard the patient’s life during the critical situations. The aim of this work is to investigate possible metabolic impairment as dementia results from brain damage. The causes include the following: Alzheimer’s Disease, Stroke, Pick’s disease, Huntington’s, Down’s Syndrome, Creutzfeldt-Jacob, AIDS, alcoholism, Parkinson’s disease, and other neurodegenerations using Chaos-Voxel.

1. INTRODUCTION

Histories of dementia commence with Alzheimer's description in 1906, the first case of Alzheimer's disease. Yet the history of dementia before 1906 is quite rich, dating back to the ancient Greek and Roman philosophers and physicians. Over 2500 years since ancient times, the concept of dementia has evolved from a rather vague notion that mental decline occurred inevitably in old age, to become defined today by a distinct set of clinical and pathological features with the potential for treatment and prevention within grasp. Throughout history, many elderly individuals with unpredictable, and the line between mental disorders and dementia were hazy at best. The identification of Alzheimer's disease at the onset of the 20th century was a turning point for the understanding of dementia, and the concepts and Voxel.

Histological findings presented by the early researchers of Alzheimer's disease remain relevant still today. Indeed, these early findings are proving to be a continuing source of insight, as many of the issues debated at the turn of the century remain unresolved today. This paper thus traces the history of the evolution of our current conceptualization of dementia. Alzheimer's disease (AD), also known as Alzheimer disease, or just Alzheimer's, accounts for 60% to 70% of cases of dementia. A chronic neurodegenerative disease that usually starts slowly and gets worse over time. The most common early symptom is difficulty in remembering recent events.

Electroencephalogram (EEG) contains set of potential differences developed as a result of volume of currents from active tissue (neural) throughout the conductive media of the brain. The measurements can be either by placing special intracranial electrodes or using sensors. Due to large number of patients, it is impossibly lengthy task to treat every patient. So there is need of new technology which should be sensitive and generate false alarm so neurologist can easily discard them.

The purpose of the proposed system does not mean to replace neurologist but it is to lessen the burden and remove the time consumption. This can help us to lessen the percentage of population highly and rapidly. This false alarm feature can lessen the burden on neurologist and can handle the right seizure and can do treatment by either giving drug or through neuro-stimulation. The neuro-stimulation and drug delivery are better than antiepileptic drugs as in seizure patient cannot push the alarm by own.

The uncovering of dementia which includes visual scan of EEG recordings for the spikes and seizures. These seizures are generated due to some drugs side-effects. This sometimes happens due to ageing due to hereditary irregularities, development anomalies’ and febrile convulsion. In some patients these seizures happens more than 100 times a day. Autopsy and craniotomy are no longer to demonstrate the useful correlates of useful deficits. In 20th century, the neuroimaging’s advancement is helpful to get the more described structure of brain and its functionalities. Vascular dementia (VaD) is not a single disease but a group of cognitive impairment caused by different
mechanisms causing ischaemia or haemorrhage secondary to cerebrovascular disease (multiple infarcts, single strategic infarct or small vessel disease.) Increasingly the term vascular cognitive impairment (VCI) is used to encompass the spectrum of deficit, in which VaD is the most severe form of disease. VaD is the second most common form of dementia in the West after Alzheimer’s disease. It is the most common form in some parts of Asia. Incidence increases with age. VaD is thought to account for around 17% of dementia in UK. Prevalence of dementia following first stroke varies depending on location and size of the infarct, definition of dementia, interval after stroke and age among other variables. Overall, stroke doubles the risk of developing dementia.

Presence of dementia-cognitive decline from higher level of functioning. This can be demonstrated as memory loss plus impairment in two or more different cognitive domains. It is established by clinical examination and neuropsychological testing using EEG signals. Deficits should be severe enough to interfere with activities of daily living-not secondary effects of the cerebrovascular event alone. Cerebrovascular disease, defined by the presence of signs on neurological examination and/or by brain imaging.

A. General Technique

During the final stages, the patient is completely dependent upon caregivers. Language is reduced to simple phrases or even single words, eventually leading to complete loss of speech. Despite the loss of verbal language abilities, people can often understand and return emotional signals. Although aggressiveness can still be present, extreme apathy and exhaustion are much more common symptoms. People with Alzheimer’s disease, Alzheimer disease or just Alzheimer’s, accounts for 60% to 70% of cases of dementia will ultimately not be able to perform even the simplest tasks independently; muscle mass and mobility deteriorate to the point where they are bedridden and unable to feed themselves. The cause of death is usually an external factor, such as infection of pressure or pneumonia, not the disease itself. Many algorithms are designed to detect these stages using EEG signals. McSharry et.al introduced an algorithm using multidimensional probability but it provides less false alarms compared to variance. Many time-frequency analysis techniques have been devising such as smoothed pseudo- Winger- Ville and reduced interference are used in conjunction with an artificial neural network. This method has great accuracy in finding the seizure. However, Chaos is tricky thing to define.

B. Development of EEG Biomarkers for Alzheimer’s disease

A growing body of evidence suggests that EEG analyses, including both resting state and event-related stimulation protocols, may be useful in early detection of neural signatures of dementia. Moreover, EEG-based analysis shows potential for discriminating across dementia sub-types, including Alzheimer’s (AD), Mild Cognitive Impairment (MCI), Vascular dementias, and the Lewy Body Dementias (LBD) – including Parkinson’s disease with Dementia (PDD) [1-6]. Although these approaches have been largely confined to university research investigations, if proven accurate, reliable, and scalable, the widespread use of EEG as a neuro imaging modality could provide an inexpensive, easy to implement alternative for early diagnosis and treatment outcome studies of the dementias.

Promising EEG biomarkers include: 1) increased power in the low frequency bands (i.e., theta, delta) with reductions in higher frequency bands (i.e., beta, gamma) [7 , 8]; 2) changes in the amplitude and latency of evoked potentials for both cognitive (i.e., attention, memory, learning) and sensory stimuli (i.e., visual, auditory, somatosensory) [9-13]; 3) reduction in the complexity of the EEG dynamics assessed with non-linear analyses (e.g., entropy, Grainger causality) [14]; and 4) abnormal functional connectivity as assessed by coherence, phase, and source localization (e.g., LORETA) analyses.

C. Single Voxel Technique

This technique uses fixed amount of tissues as unified signal to gain single spectrum. The volume of Voxel ranges between 2-8 ml in proton in proton MRS. For prolix process 8cm\(^3\) Voxel is used. Voxel must be positioned from basis of susceptibility, relics and lipids. The limitation of this technique is anatomic coverage and assessment of single area only during acquisition. So positioning of Voxel is must. The fundamental single Voxel localisation is to use three orthogonal slice selective pulses equally and enterprise the pulse sequence to collect only the echo signal from the Voxel in space where all three shares intersect. The two sequences generally used are called Stimulated Echo Acquisition Mode (STEAM) and Point Resolved Spectroscopy (PRESS). Three 90 degree pulsations are used and stirred echo is serene in STEAM. All other signals should be dephrased by the large implement gradient applied during the so called mixing time. While in PRESS, second and third are changed by 180 degree and crusher gradient is applied across the pulses to select the desired spin echo sign arising from all three frequencies.

1) Main Characteristics of STEAM

It is cooler to produce a 90\(^0\) pulse with a shrill slice profile than a 180\(^0\) pulse. There is incomplete recapture of signal.

A precise volume Voxel is formed. It can be formed with very short echo times

2) Main Characteristics of PRESS

Rather better signal to noise ratio because the motivated echo is formed from only half the available equilibrium magnetization.

There is complete retrieval of signal. It can be performed with little and long TE
II. CARE FOR PATIENTS WITH ADVANCED DEMENTIA AND EATING PROBLEM

Problem with eating, swallowing, and poor caloric intake are common in patients with advanced dementia and often develop during an acute medical event when the immediate prognosis is unclear. For care professionals, managing the patient with advanced dementia and swallowing problems and guiding caregivers through a process of decision making present enormous clinical challenges and require an interdisciplinary approach in order to provide an optimal care. Careful observation of patient at bedside is required to evaluate level of consciousness, position in bed, level of comfort, presence of muscle entropy and contractions, presence of an oxygen mask or nasal cannula, and presence of mouth breathing. Attention to the state of dentition and oral hygiene is paramount, as pain from dental carries, poor oral hygiene and xerostomia can contribute to problem with eating. Assessing the patient’s level of consciousness is critical for detecting delirium, a common and reversibly cause of eating problems. When delirium is present, a workup is aimed in diagnosing and treating modifiable factor is indicated.

III. CAUSES OF DEMENTIA

Dementia involves damage of nerve cells in the brain, which may occur in several areas of the brain. Dementia may affect people differently, depending on the area of the brain affected. Dementias can be classified in a variety of ways and are often grouped by what they have in common, such as what part of the brain is affected, or whether they worsen over time (progressive dementias). Some dementias, such as those caused by a reaction to medications or an infection, are reversible with treatment. Progressive dementias, Types of dementias that are not reversible and worsen over time include:

- Alzheimer's disease. In people age 65 and older, Alzheimer's disease is the most common cause of dementia. People generally may develop symptoms after age 60, but some people may have early-onset forms of the disease, often as the result of a defective gene. Although in most cases the exact cause of Alzheimer's disease isn't known, plaques and tangles are often found in the brains of people with Alzheimer's. Plaques are clumps of a protein called beta-amyloid, and tangles are fibrous tangles made up of tau protein. Certain genetic factors also may make it more likely that people will develop Alzheimer's. Alzheimer's disease usually progresses slowly over about eight to 10 years. Your cognitive abilities slowly decline. Eventually, the affected areas of your brain don't work properly, including parts of your brain that control memory, language, judgment and spatial abilities.

- Vascular dementia. Vascular dementia, the second most common type of dementia, occurs as a result of brain damage due to reduced or blocked blood flow in blood vessels leading to your brain. Blood vessel problems may be caused by stroke, infection of a heart valve (endocarditic) or other blood vessel (vascular) conditions. Symptoms usually start suddenly and often occur in people with high blood pressure or people who have had strokes or heart attacks in the past. Several different types of vascular dementia exist, and the types have different causes and symptoms. Alzheimer’s disease and other dementias also may be present at the same time as this dementia.

- Lewy body dementia. Lewy body dementia affects approximately 10 percent of people with dementia, making it one of the most common types of dementia. Lewy body dementia becomes more common with age. Lewy bodies are abnormal clumps of protein that have been found in the brains of people with Lewy body dementia, Alzheimer's disease and Parkinson's disease. Lewy body dementia symptoms are similar to symptoms of Alzheimer's disease. Its unique features include fluctuations between confusion and clear thinking (lucidity), visual hallucinations, and tremor and rigidity (parkinsonism). People with Lewy body dementia often have a condition called rapid eye movement (REM) sleep behavior disorder that involves acting out dreams.

- Front temporal dementia. This type of dementia tends to occur at a younger age than does Alzheimer's disease, generally between the ages of 50 and 70. This is a group of diseases characterized by the breakdown (degeneration) of nerve cells in the frontal and temporal lobes of the brain, the areas generally associated with personality, behaviour and language. Signs and symptoms of front temporal dementia can include inappropriate behaviours, language problems, difficulty with thinking and concentration, and movement problems. As with other dementias, the cause isn't known, although in some cases this dementia is related to certain genetic mutations.

IV. DIAGNOSIS

There is no one test to determine if someone has dementia. Doctors diagnose Alzheimer's and other types of dementia based on a careful medical history, a physical examination, laboratory tests, and the characteristic changes in thinking, day-to-day function and behaviour associated with each type. Doctors can determine that a person has dementia with a high level of certainty. But it's harder to determine the exact type of dementia because the symptoms and brain changes of
different dementias can overlap. In some cases, a doctor may diagnose "dementia" and not specify a type. If this occurs it may be necessary to see a specialist such as a neurologist or geropsychologist. Alzheimer's disease accounts for up to 80 percent of all dementia cases. Diagnosis may be complicated by other forms of dementia that have symptoms and pathologies similar to Alzheimer's disease. Knowing the key features and pathology of each type of dementia can help in the accurate diagnosis of patients, so they will receive the treatment and support services appropriate for their condition and maintain the highest possible quality of life. The following table identifies some of the clinical differences between the major dementias:

<table>
<thead>
<tr>
<th>Disease</th>
<th>First Symptom</th>
<th>Mental Status</th>
<th>Neuropsychiatry</th>
<th>Neurology</th>
<th>Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD</td>
<td>Memory loss</td>
<td>Episodic memory loss</td>
<td>Initially normal</td>
<td>Initially normal</td>
<td>Entorhinal cortex and hippocampus atrophy</td>
</tr>
<tr>
<td>FTD</td>
<td>Apathy; poor judgement/insight/speech/language; hyperorality</td>
<td>Frontal/executive,language;spares drawing</td>
<td>Apathy, disinhibition, hyperorality, euphoria, depression</td>
<td>May have vertical gaze palsy, axial rigidity, dystonia, alien hand, or MND</td>
<td>Frontal, insular, and/or temporal atrophy; spares posterior parietal lobe</td>
</tr>
<tr>
<td>DLB</td>
<td>Visual hallucinations, REM sleep disorder, delirium, Capgras' syndrome, parkinsonism</td>
<td>Drawing and frontal/executive; spares memory; delirium prone</td>
<td>Visual hallucinations, depression, sleep disorder, delusions</td>
<td>Parkinsonism</td>
<td>Posterior parietal atrophy; hippocampus larger than in AD</td>
</tr>
<tr>
<td>CJD</td>
<td>Dementia, mood, anxiety, movement disorders</td>
<td>Variable, frontal/executive, focal cortical, memory</td>
<td>Depression, anxiety</td>
<td>Myoclonus, rigidity, parkinsonism</td>
<td>Cortical ribboning and basal ganglia or thalamus hyper intensity on diffusion/FLAIR MRI</td>
</tr>
<tr>
<td>Vascular</td>
<td>Often but not always sudden; variable; apathy, falls, focal weakness</td>
<td>Frontal/executive, cognitive slowing; can spare memory</td>
<td>Apathy, delusions, anxiety</td>
<td>Usually motor slowing, spasticity; can be Normal</td>
<td>Cortical/or subcortical infarctions</td>
</tr>
</tbody>
</table>

V. TREATMENT AND CARE

Dementia is not a specific disease. It's an overall term that describes a wide range of symptoms associated with a decline in memory or other thinking skills severe enough to reduce a person's ability to perform everyday activities. Alzheimer's disease accounts for 60 to 80 percent of cases. Vascular dementia, which occurs after a stroke, is the second most common dementia type. But there are many other conditions that can cause symptoms of dementia, including some that are reversible, such as thyroid problems and vitamin deficiencies. Dementia is often incorrectly referred to as "senility" or "senile dementia," which reflects the formerly widespread but incorrect belief that serious mental decline is a normal part of aging.
Treatment of dementia depends on its cause. In the case of most progressive dementias, including Alzheimer's disease, there is no cure and no treatment that slows or stops its progression. But there are drug treatments that may temporarily improve symptoms. The same medications used to treat Alzheimer's are among the drugs sometimes prescribed to help with symptoms of other types of dementias. Non-drug therapies can also alleviate some symptoms of dementia. Ultimately, the path to effective new treatments for dementia is through increased research funding and increased participation in clinical studies. Right now, volunteers are urgently needed to participate in more than 180 actively enrolling clinical studies and trials about Alzheimer's and related dementias.

VI. DEMENTIA RISK AND PREVENTION

Some risk factors for dementia, such as age and genetics, cannot be changed. But researchers continue to explore the impact of other risk factors on brain health and prevention of dementia. Some of the most active areas of research in risk reduction and prevention include cardiovascular factors, physical fitness, and diet. Cardiovascular risk factors: Your brain is nourished by one of your body's richest networks of blood vessels. Anything that damages blood vessels anywhere in your body can damage blood vessels in your brain, depriving brain cells of vital food and oxygen. Blood vessel changes in the brain are linked to vascular dementia. They often are present along with changes caused by other types of dementia, including Alzheimer's disease and dementia with Lewy bodies. These changes may interact to cause faster decline or make impairments more severe. You can help protect your brain with some of the same strategies that protect your heart—don't smoke; take steps to keep your blood pressure, cholesterol and blood sugar within recommended limits; and maintain a healthy weight.

Physical exercise: Regular physical exercise may help lower the risk of some types of dementia. Evidence suggests exercise may directly benefit brain cells by increasing blood and oxygen flow to the brain.

Diet: What you eat may have its greatest impact on brain health through its effect on heart health. The best current evidence suggests that heart-healthy eating patterns, such as the Mediterranean diet, also may help protect the brain. A Mediterranean diet includes relatively little red meat and emphasizes whole grains, fruits and vegetables, fish and shellfish, and nuts, olive oil and other healthy fats.

VII. PROPOSED WORK

The EEG records of 347 patients data set has been taken from hospital Aligarh Muslim University, Aligarh, India who had been under treatment of Neurology.
C. Risk Level Estimation

The output of the chaos technique provides two risk levels i.e. risk level 1 and risk level 2. Since we are dealing with dementia patients so exact risk level cannot be found. So this can be done by automated system which can classify the risk level of patient being examined. The performance of this method is being given by: \( \text{PI}=\frac{p-m-f}{p} \times 100\% \). Where \( p \) is perfect classification and is given when physician and method both agrees; \( m \) is missed classification and is true negative of the machine to the physician. (I.e. complement values) and \( f \) is false alarm and is false positive of the machine in reference with the neurologist. Since the machine has to be sensitive. So its sensitivity can be calculated as \( S=\frac{p}{p+f} \times 100\% \).

VIII. RESULT AND DISCUSSION

The output of three epochs for each patient is optimized at single level. The study of the performance is given by performance index and quality value. These are calculated for each set of patients and are to be compared. The performance index obtained by Common Energy Level (determining and measuring accuracy) was 28% and for Chaos-Voxel it is 49%. EEG-based biomarkers show promise for utility in early detection of Alzheimer’s disease, notably:

- Frequency Bandwidths – significant decreases in parietal/temporal alpha, and global sigma, beta
- Frequency Bandwidth Ratios – excessive slowing in parietal and temporal regions
- Wavelet Analysis – significant increases in slow wave activity over the sensor motor region. These findings support potential for EEG as an inexpensive, easily-implementable biomarker for AD.

A. Quality Value

Goal of this research was to get perfect classification of dementia patients and get fewer false alarms. This can be done by the calculating following parameters as follows

- Classification rate
- Classification delay
- False alarm rate

Quality value can be computed as follows

\[ Q = \frac{C}{(fa+0.2) \times (td \times p_{pc} + 0.1 \times p_{ms})} \]

where \( C \) is the scaling constant.

\( Fa \) is the false alarm rate; \( td \) is the delayed time; \( p_{pc} \) is the percentage of perfect classification and \( p_{ms} \) is the percentage of missed classification. The value of constant \( C \) is set to be 10 for convenience. The higher the value of \( Qv \) better the method is.

The table below shows the value of quality standards.
As shown in table, The Risk Level is higher in this optimization technique also weighted delay is very less. Number or false alarm rate are also reduced by this technique. The quality value is highly increased. Hence it is more reliable than the previous technique.

### IX. CONCLUSION

This Chaos Voxel technique is very successful in determining and classifying the risk level. Almost negligible false alarm/set is calculated which leads neurologists’ burden much lower. This also has high performance index. The future research can be done on this chaos Voxel technique can rely this system accurately.

### REFERENCES


