Chapter 7
Alzheimer’s Disease Recognition with Artificial Neural Networks

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ABSTRACT
Alzheimer’s Disease (AD) is the most common cause of dementia, and is well known for its affect on memory loss and other intellectual abilities. The Electroencephalogram (EEG) has been used as a diagnosis tool for dementia for several decades. The main objective of this work was to develop an Artificial Neural Network (ANN) to classify EEG signals between AD patients and control subjects. For this purpose, two different methodologies and variations were used. The Short Time Fourier Transform (STFT) was applied to one of the methodologies and the Wavelet Transform (WT) was applied to the other methodology. The studied features of the EEG signals were the Relative Power in conventional EEG bands and their associated Spectral Ratios ($r_1$, $r_2$, $r_3$, and $r_4$). The best classification was performed by the ANN using the WT Biorthogonal 3.5 with AROC of 0.97, Sensitivity of 92.1%, Specificity of 90.8%, and 91.5% of Accuracy.

INTRODUCTION
Alzheimer’s Disease (AD) is an incurable illness, a chronic progressive and irreversible neurodegenerative brain disorder and the most common cause of dementia in the elderly (Ballard, Gauthier, Corbett, Brayne, Aarsland, & Jones, 2011). In 2001, there were more than 24.3 millions of live people that have contracted AD and according to estimates of Delphi in 2040 there will be 81.1 million AD patients (Ballard, Gauthier, Corbett, Brayne, Aarsland, & Jones, 2011). The cause of AD is not yet known. So far, no one single factor has been identified as being responsible to cause AD. It seems that a combination of factors, such as: age, genetic inheritance, environment, lifestyle,
education, obesity, diabetes, hypertension, cholesterol, tobacco, alcohol, Down’s syndrome and head injury, may be responsible for this disease (Cummings, 2004; Mayeux, 2003; Román, 2002). The causes of AD are not well understood and the disease may develop silently for many years before symptoms appear. The life expectancy following diagnosis is approximately seven years (Mölsä, Marttila, & Rinne, 1986).

The diagnostic accuracy is relatively low and there is not a biomarker able to detect AD without invasive tests (Ballard, Gauthier, Corbett, Brayne, Aarsland, & Jones, 2011; Bird, 2001). This progressive disease of brain can affect several cerebral areas connected with memory, thinking, planning and attention (Bird, 2001). An autopsy or brain biopsy is the only way to make a definitive diagnosis of AD (Hatfield, Dudas, & Dening, 2009). The diagnosis of AD is difficult, and symptoms are often confounded with other normal symptoms of aging or manifestations of stress. Also, Alzheimer’s symptoms are often subtle at the beginning (Waldemar, 2007). Usually, the diagnosis is made based on history and findings on Mental State Examination. But diagnosis is also performed through blood tests, spinal fluid, brain scans, EEGs and imaging techniques, such as Magnetic Resonance Imaging (MRI), Positron Emission Tomography (PET) and Computerized Tomography (CT) (Weiner, 2009; Prichep, 2007).

The neuropathology of AD is characterized by the medial temporal lobe atrophy and the accumulation of neurofibrillary tangles and amyloid plaques. In fact, amyloid plaques and neurofibrillary tangles are clearly visible by microscopy in brains of patients with AD (Tiraboschi, Hansen, Thal, & Corey-Bloom, 2004). However, the ultimate cause of neurons death in Alzheimer’s still unknown, but some evidences suggest that the abnormal processing of beta-amyloid protein can probably be the responsible. The internal support structure for brain cells depends on the normal functioning of a tau protein. In patients with Alzheimer’s, threads of tau protein suffer alterations that cause them to become twisted. Some researchers believe this may seriously damage neurons, taking them to die (Tiraboschi, Hansen, Thal, & Corey-Bloom, 2004). This disease is an acquired disorder of cognitive and behavioral impairment that notoriously interferes with social or occupational functioning of patients. It is therefore a disease extremely debilitating, with significant social, psychological and economical loss for the patient, the family and society that has to lead to decreased quality of life and absence from work early (Bonin-Guillaume, Zekry, Giacobini, & Michel, 2005; Bonin-Guillaume & Rosenstein, 1998).

The progression of the disease can be classified in four different stages. The first stage is known as Mild Cognitive Impairment (MCI) and corresponds to a variety of symptoms: difficulty in remembering recent events (memory loss is the most common early symptom of AD and the hippocampus, associated with memory processing, is particularly injured in the early stages of this disease), subtle changes in behavior, confusion, discrete loss of autonomy in activities of daily life, disorientation in time and space, loss of spontaneity and initiative, judgment and personality changes. However, the first stage does not change substantially the daily life. As Alzheimer’s progresses memory problems persist and worsen and the next two stages of Alzheimer’s disease, Mild and Moderate AD, are characterized by increasing cognitive deficits and complete dependence on caregivers: difficulty in recognizing people, learning disabilities, inappropriate behavior, irritability, aggression, failure of judgment and thought obsessed. At the last stage, Severe AD, a complete deterioration of personality occurs: weight loss even with proper diet, total dependence, silence, strict bed rest, inability, extreme irritability, culminating in death (Maciel, 2007; Waldemar, 2007; Nygard, 2003; Landes, Sperry, Strauss, & DS, 2001).

While there is still no cure for AD, something must be done to keep the quality of life and help a person staying active. Current approaches in the