Chapter 2
Alzheimer’s Electroencephalogram Event Scalp and Source Localization

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ABSTRACT
Alzheimer’s disease is the most common cause of dementia which causes a progressive and irreversible impairment of several cognitive functions. The aging population has been increasing significantly in recent decades and this disease affects mainly the elderly. Its diagnostic accuracy is relatively low and there is not a biomarker able to detect AD without invasive tests. Despite the progress in better understanding the disease there remains no prospect of cure at least in the near future. The electroencephalogram (EEG) test is a widely available technology in clinical settings. It may help diagnosis of brain disorders, once it can be used in patients who have cognitive impairment involving a general decrease in overall brain function or in patients with a located deficit. This study is a new approach to improve the scalp localization and the detection of brain anomalies (EEG temporal events) sources associated with AD by using the EEG.

INTRODUCTION
In the last decades there have been deep changes in the age structure of world population, characterized by a progressive decrease in young population and inversely, by an increasing proportion of elderly people (Ballard, et al., 2011; Machado, 2002). The decrease of mortality and the simultaneous decrease of fertility levels have contributed to the phenomenon called overall aging of population. The aging population has become a fact of scientific interest around the world because the
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elderly are most vulnerable to the onset of certain degenerative diseases (Jonker, Launer, Hooijer, & Lindeboom, 1996). Alzheimer’s disease (AD) is one of the most debilitating diseases of developed societies. AD is an incurable illness, a chronic progressive and irreversible neurodegenerative brain disorder and the most common cause of dementia in the elderly (Ballard, et al., 2011). AD represents 60% of elderly diseases. Gradually, over time, most parts of the brain will suffer damage and symptoms will severely increase (Blennow, Leon, & Zetterberg, 2006). In 2001, there were more than 24.3 million of live people that have AD and according to estimates of Delphi in 2040 there will be 81.1 million AD patients (Ballard, et al., 2011). Experts do not know yet why the brain cells deteriorate. 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, stress, genetic inheritance, environment, lifestyle, lower educational qualifications, obesity, diabetes, hypertension, cholesterol, tobacco, alcohol, Down’s syndrome and head injury, may be responsible for this disease (Cummings, 2004; Mayeux, 2003; Román, 2002). Ageing is considered the main risk factor to acquire AD; after the age of 65, the risk of developing the disease doubles every five years. Another factor is family history; people who have a close relative who developed Alzheimer’s disease have a slightly higher risk of eventually developing the disease themselves (Blennow, 2005). Gender is considered another factor; a higher percentage of women develop AD than men. Heart disease, like high cholesterol, hypertension or poorly controlled diabetes are considered risk factors to develop AD (Blennow, Leon, & Zetterberg, 2006; Lahiri, Farlow, Greig, & Sambamurti, 2002).

The diagnostic accuracy is relatively low and there is not a biomarker able to detect effectively AD without invasive tests (Ballard, et al., 2011; Bird, 2001). The first symptoms of AD are memory loss, particularly difficulty remembering newly learned information, and concentration problems (Gwyther, 2001). As the disease progresses patients manifest general cognitive problems, confusion, personality / behavioral changes and also disorientation (Bird, 2001). Finally, there is a global brain atrophy and patients acquire a complete inability (Cummings, 2004). Therefore, permanent aids of family members or caregivers are increasingly requested (Jeong, 2004). An autopsy or brain biopsy is the more accurately way to make a definitive diagnosis of AD (Hatfield, Dudas, & Dening, 2009). The AD diagnosis 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). Senile plaques and neurofibrillary tangles in the medial temporal lobe and cortical areas are two pathological hallmarks of brains ravaged by AD (Blennow, 2005; Mattson, 2004). These two abnormal structures are responsible for damaging and killing nerve cells (Cummings, 2004). However, the ultimate cause of neurons death in AD is 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