*Current Neurobiology 2013; 4 (1&2): 31-34* ISSN 0975-9042
http://currentneurobiology.com

# Behavioural and Psychological Symptoms of Dementia: An Overview.

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## Abstract

**The issue of mental and behavioural manifestations in patients diagnosed with Alzheimer's disease has seen an evolution of enormous proportions. This is why a new acronym has been introduced into contemporary literature and everyday language: BPSD (*Behavioural and Psychological Symptoms of Dementia*). BPSD is classified as "non-cognitive" symptoms of dementia. The symptoms usually appear heterogeneous and individual compared to the cognitive deficit itself. BPSD shows a high interindividual variability in the various types mental and behavioural symptoms, in their severity and the stage at which they appear. The pathogenic mechanism of mental and behavioural disorders is complex: the combination of strictly individual biological and mental factors (genetic predisposition, pre-morbid personality) with interpersonal and environmental factors (reduced social relations, hospitalisation, institutionalisation) can in part explain the triggering of such symptomatology. BPSD is conventionally divided into clusters, which include *apathy, depression, psychosis, agitation and aggression*. They are not a diagnosis in the categorical sense, but have an operative significance as they allow for the identification of targets for drug therapy. The need to define the characteristics of BPSD accurately has motivated the creation of numerous psychometric instruments: global scales that examine a wide behavioural and psychotic range and more specific scales aimed at particular symptomatologies.**

**Keywords:** Alzheimer's disease, Behavioural and Psychological Symptoms of Dementia

*Accepted 17th October 2013*

## Introduction

Dementias, of which Alzheimer's is the most common, are part of the chronic-degenerative illnesses typical of ageing and are a welfare priority for our country. Their significance, above all in terms of social costs, is bound to increase in the future because of the progressive ageing of the population, together with an increase in life expectancy. Dementia, which will become the sixth cause of death in first world countries over the next thirty years, today concerns approximately 6.4% of the population over 65 years of age with a slightly higher percentage in women [1]. Behavioural and psychological symptoms often occur in patients with Alzheimer's disease and related dementias leading to a complex clinical course and a more difficult management of these subjects. Such disorders are the most common cause of institutionalisation of the patient, drug prescription and medical intervention and contribute significantly to a reduced quality of life of the patient and increased stress for the caregivers [2-3]. Furthermore, these disorders are responsible for a considerable increase in costs for the illness linked to the need for hospitalisation and qualified care-staff [4]. Therefore, their effect on the prognosis and the course of the dementia is considerable, at times superior, to that of cognitive decay.

## Definition and classification

The issue of mental and behavioural manifestations in patients diagnosed with Alzheimer's disease has seen an evolution of enormous proportions. This is why a new acronym has been introduced into contemporary literature and everyday language: BPSD (*Behavioural and Psychological Symptoms of Dementia*). The term "*Behavioural and Psychological Symptoms of Dementia"* (BPSD) has been proposed to describe the spectrum of non cognitive manifestations of dementia that include verbal and physical aggression, agitation, psychotic symptoms (hallucinations and delusions), sleep disturbances and wandering. In 1996, the International Psychogeriatric Association de-

fined BPSD as "alterations of the perception, thought, humour or behaviour" which are often seen in patients with dementia. The presence of "behavioural" symptoms in dementia is certainly no recent discovery: Alois Alzheimer himself reported paranoia, sexually inappropriate behaviour, hallucinations and agitation in his initial description of the illness [5]. However, it is only over recent decades that the international scientific community has made a precise identification and codification of BPSD [2]. BPSD is classified as "non-cognitive" symptoms of dementia and the symptoms are considered to be relatively independent manifestations of cognitive deficit by some authors. According to others, however, some of the mental and behavioural manifestations of dementia are only the consequence of modifications of the cognitive functions, given the close correlation between the severity of the behavioural disorder and the degree of cognitive deficit. Unlike cognitive decline, the course of the BPSD is not straightforward: BPSD shows a characteristic fluctuating trend and can occur both in the early stages of the illness and in the later ones and usually appear heterogeneous and individual compared to the cognitive deficit itself. They show a high interindividual variability in the various types of dementia, in their severity and the stage at which they appear [6]. In the initial stages of Alzheimer's disease, the non-cognitive symptoms can be the cause of diagnostic errors because of their similarity to those of other psychiatric illnesses. The clinical assessment of BPSD is made quite difficult by the contemporary presence of cognitive symptoms. The characterisation of each psycho-behavioural symptom can be masked by an overall decline with the result that suitable therapeutic measures are not often taken into consideration [7].

## Pathogenesis

The pathogenic mechanism of mental and behavioural disorders is quite complex and certainly multifactoral: the combination of strictly individual biological and mental factors with interpersonal and environmental factors can in part explain the triggering of such symptomatology in the insane patient [2]. Of the abovementioned, a genetic predisposition plays a crucial role, as well as the dementing process itself: in fact, it can be established that the behavioural modifications are as severe as the cognitive impairment. Furthermore, even a modified pre-morbid personality correlates to a higher incidence of mental disorders over the course of the illness. Among the external elements important in the development of BPSD are the reduced interpersonal and social relations of the patient, as well as the stress of the caregiver. Hospitalisation and institutionalisation still contribute decisively to the pathogenesis of BPSD: there is often a close relationship between the high dependency of the patient for basic needs and their physical aggression, as just as frequently it is the nursing itself that triggers agitation attacks. It should be pointed out that possible causes of mental and behavioural perturbations can be attributed to the concurrent assumption of particular categories of drugs (antipsychotics, benzodiazepines, antidepressants), as well as to concomitant illnesses (urinary infections, respiratory infections, high temperatures) or to acute painful symptomatologies (fractures, arthrosis, etc.) [2].

The importance of these factors at the outset and during the course of the BPSD is not yet clear; however, from a clinical point of view, a clear, independent evaluation of the BPSD to understand the outcome of pharmacological or non-pharmacological treatments of such disorders seems to be of fundamental importance.

### Neurobiology

The neuropathological lesions in Alzheimer's disease are senile plaques and neurofibrillary tangles, associated with neuronal loss on both the hippocampus and the neocortex. There is also serious neuronal depletion in the subcortical nuclei that project to the cerebral cortex: the basal nucleus of Meynert, the locus coeruleus, the raphe nuclei. Although there is a pronounced loss of cholinergic neurons, some cerebral cholinergic areas are completely untouched. The complexity and extent of the brain damage are accountable for the multiplicity of the symptoms: cognitive disorders correlated to the loss of cholinergic neurons combined with behavioural and emotional anomalies.

The suppositions of BPSD neurobiology are based on neuropathological, neurochemical and neuroimaging studies. The impairment of the neurotransmitter systems (serotoninergic, dopaminergic, noradrenergic, cholinergic) in Alzheimer's disease as a result of the neurodegenerative process may contribute to the onset of BPSD, with the development of different case histories according to the cerebral areas and circuits involved in each case [8].

*A summary of available data suggests that:*
reduced levels of serotonin play an important role in the pathogenesis of depression, anxiety, agitation, aggression and impulsiveness, as well as psychotic symptoms,
an increase in the levels of noradrenaline and dopamine causes agitation and aggression and is associated with hallucinations and delusions,
reduced levels of acetylcholine are related not only to the cognitive symptoms, but also to the behavioural aspects.

## Clusters

Although several authors have used slightly different classifications, BPSD is conventionally divided into clusters, which include apathy, depression, psychosis, agitation and aggression [2].

If on the one hand these clusters are made based on the similarities with case histories of other illnesses, on the affinity of the symptoms, on a possible community of etiopathogenetic mechanisms and on a possible answer to some pharmacological intervention, on the other hand it is important to point out that the clusters have areas of superimposition. They are not a diagnosis in the categorical sense, but have an operative significance as they allow for the identification of targets for drug therapy. The clinical-diagnostic procedure of clustering of the various symptoms could help physicians in the choice of therapy considering that the groups of different symptoms can respond to the same drug treatment. The agitation cluster includes repetitive actions, wandering, walking aimlessly, sleep disturbance, noisiness and dressing/undressing. According to some authors, agitation is not a distinct diagnostic entity. However, the fact that many of the listed symptoms can appear as a consequence of depression, anxiety, pain and psychosis must be taken into consideration. Furthermore, there is comorbidity between agitation and psychotic symptoms, even if agitation is common in non-psychotic demented patients. Agitation is common in patients with Alzheimer's disease with cognitive functions both moderately and severely compromised [8-9]. Although agitation is clinically less well defined than psychosis, it is a significant problem from a management point of view as it often necessitates hospitalisation. The psychotic cluster includes delusions, hallucinations and misidentifications. The persecutory delusions are characterised by jealousy, theft, and the idea that a stranger lives in the house. The visual hallucinations, unlike in schizophrenia, are more common than the auditory ones. Olfactory hallucinations are very rare. Misidentifications include the "phantom phenomenon", misidentification of one's self, of other people and of what happens on TV. The overall impact of psychoses in dementias varies, according to several studies, from 20% to more that half of the patients: the wide variability of data can be traced to the differences between the instruments used for evaluation, as well as the lack of homogeneity of the study sample. The psychotic symptoms can appear at any stage of the illness, although they are more common in the intermediate or advanced stages [10].

A further characteristic of delusions and hallucinations in demented patients is the spontaneous remission of the symptoms, often in the space of days or weeks. This is very important from the point of view of the correct assessment of clinical studies [9]. Another behavioural aspect within the BPSD is aggression. It can reveal itself in various forms such as physical aggression, verbal aggression, aggressive resistance, destruction of objects and threats [10]. The depression cluster includes mood swings, emotional lability, sadness, crying, hopelessness, low self-esteem and guilt. In the majority of cases the intensity of depression is fluctuating. Spontaneous remissions and improvements are more common than in usual depressive episodes, even though they are shorter. A comorbidity between dementia and depression has always been acknowledged. Depression that appears after the beginning of the cognitive symptoms of Alzheimer's disease often appears in patients who have had other depressive episodes in their lives. Depression is considered a risk factor for Alzheimer's disease [9]. The characterisation of depression over the course of Alzheimer's disease is an important aspect. Some studies have tried to identify a particular profile of depression in Alzheimer's but the overall picture that emerges is quite controversial and does not allow for the definition of a characteristic symptomatogical pattern [2-11]. There are numerous problems with the semiologic and diagnostic definition of depression linked to dementia. There is considerable overlapping of the symptomatology of the two conditions. For example, apathy and emotional lability are common both in depression and dementia. Even vegetative symptoms, such as those related to appetite and sleep, are to be found in both disorders and, therefore, are not practical for diagnosis. The most practical symptoms for diagnosis, in the initial stages of dementia, are the nuclear ones of depression (sadness, anhedonia, lower self-esteem, etc), while more attention should be paid to behavioural signs (agitation, aggression, refusal) towards the later stages of the illness [7].

## Assessment Scales

For years literature has tried to identify a means of detecting only the behavioural symptoms of Alzheimer's disease, excluding and separating them from the cognitive and functional symptoms.

The need to define the characteristics of BPSD accurately has motivated the creation of numerous psychometric instruments [12]. There are many methods that can be grouped into two types: global scales that examine a wide behavioural and psychotic range, and more specific scales aimed at particular symptomatologies or functions. Among the main global scales are the Neuropsychiatric Inventory (NPI) or the Behavioural Pathology in Alzheimer's Disease Rating Scale (BEHAVE-AD). The NPI scale assesses the frequency and severity of hallucinations, aggression or agitation, depression, anxiety, euphoria, apathy, disinhibition, irritability/emotional lability, hyperactivity and sleep disturbance [13]. The Behave-AD is a test to assess the severity of 25 potentially treatable behavioural disorders that appear most frequently in patients with Alzheimer's disease. Created by B. Reisberg in 1987, it measures many symptoms particular to Alzheimer's disease and their independence from the progression of the cognitive symptomatology. Among the various symptomatological categories analysed are delusions and paranoia, hallucinations, activity disturbance, aggression, daily routine disorders, emotional disorders, anxiety and phobias [14].

Among the "specific symptom" scales are the Clinical Insight Rating Scale (CIR) for disorder awareness, the Geriatric Depression Scale and the Hamilton Scale for Depression for depression and the Cohen-Mansfield Agitation Inventory and Ryden Aggression Scale for agitation and aggression. In particular, Cohen-Mansfield defines the various manifestations of agitation and aggression in patients and qualifies them on a scale called CMAI where there is a distinction between physical aggressive behaviour, physical non-aggressive behaviour and verbal agitation. The scale has very little use in the initial stages of the disease when aggression and agitation are expressions of other mental disorders such as psychosis, depression and anxiety [15].

## Conclusion

Clinical observation and psychometric investigation have highlighted the frequent presence of mental disorders and behavioural modifications in Alzheimer's disease and other dementias. Although there has been remarkable progress in recent years, there is a need for further investigation into the clinical definition and the neurobiological aspects of BPSD.

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