EWA WOLAŃSKA
Warsaw University, Institute of Applied Polish Studies,
Department of Speech and Language Therapy and Voice Production
ORCID ID: https://orcid.org/0000-0003-1069-6534

Disorders of Language Functions and Other Cognitive Functions in Semantic Dementia

SUMMARY

The aim of this article is to describe the singularities of linguistic and cognitive disorders as well as changes in behaviour and personality in semantic dementia (SD). SD, also known as the semantic variant of Primary Progressive Aphasia (svPPA), is a progressive neurodegenerative disorder characterised by loss of semantic memory. SD patients usually have difficulty updating previously known words and recognising familiar objects and faces. Clinical symptoms include anomia, multimodal misunderstanding of word meanings, aphasia with preserved speech fluency and associative visual agnosia. The speech of SD patients is characterised by pauses needed to find the missing words, reduced frequency of occurrence of autosemantic words, the presence of semantic paraphasia, increased verb-to-noun ratio and multiple repetitions. As the disease progresses, changes in behaviour and personality are often seen as similar to those observed in frontotemporal dementia.

Key words: semantic dementia, semantic memory, aphasia, aphasia with preserved speech fluency, anomia, associative visual agnosia, degeneration of frontal and temporal lobes

INTRODUCTION

Semantic dementia is a neurodegenerative disease, with the axial symptom involving the gradually increasing loss of multimodal semantic knowledge with relatively preserved efficiency of the phonological, morphological and syntactic subsystems (Adlam et al. 2006; Rohrer et al. 2008). As the diseases develops, other cognitive disorders, subject to generalisation, emerge in addition to the isolated lexical-semantic deficits. Moreover, most patients experience behavioural and personality disorders of varying severity. These deficits are caused by bilateral (often asymmetrical) atrophies in the anterior temporal lobes of the brain. Histopathological examination usually reveals neuronal atrophy and tau-positive,

ubiquitin-positive gliosis (Davies et al. 2005; Kertesz et al. 2005; Sikorska, Liberski and Wszołek 2005). This disease usually commences between the ages of 55 and 70. The average duration of the disease from the onset of symptoms to the patient's death is usually 7–8 years (Hodges et al. 2003; Roberson et al. 2005; Kertesz et al. 2007). Epidemiological studies do not find any significant disproportion in the number of cases among women and men (Kertesz et al. 2007).

HISTORY OF RESEARCH

The term *semantic dementia* was introduced and popularised at the turn of the 1990s (see, e.g., Snowden et al. 1989; Hodges et al. 1992). However, cases of people with symptoms identical to those of SD were observed and described already in late 19th and early 20th century. At that time, they were referred to as *verbal deafness* or *verbal amnesia* (see, e.g., Pick 1892, 1904; Déjerine and Sérieux 1897; Rosenfeld 1909). For nearly seven consecutive decades, cases of people with progressing aphasia-like features were of little interest to researchers. In clinical practice, they were usually classified as the Alzheimer's disease (AD).

In the first half of the 1970s, Elizabeth K. Warrington examined and then described three cases of her patients with clinical symptoms largely similar to those described by Arnold Pick (1892, 1904, 1906). Warrington diagnosed the patients as having progressive difficulties in naming the objects shown (anomia) and loss of understanding of word meanings. The linguistic disorders were accompanied by a associative visual agnosia, where observations are created, but do not evoke the memory traces necessary to give meaning to those observations. In addition, the author of the research stated that the patients did not suffer from episodic memory deficits or spatial and temporal orientation disorders. Thus, she captured the most important difference between the symptoms experienced by her patients and the disorders occurring in Alzheimer's patients (Warrington 1975). As a result, semantic dementia started to be perceived as a separate clinical entity.

CLASSIFICATION AND TERMINOLOGY

A disease superior to semantic dementia is *frontotemporal lobar degeneration* (FTLD). Although nowadays researchers agree that FTLD is a heterogeneous group of symptoms, the classification of specific variants is still a contentious issue. One of the most popular classifications distinguishes three main forms of clinical syndromes within FTLD: *frontotemporal dementia* (FTD), *semantic dementia* (SD) and *progressive non-fluent aphasia* (PNFA)[1] (see, e.g., Galariotis

[1] In the original paper, the authors use the simplified term *progressive aphasia* (PA).

et al. 2005; Neary, Snowden and Mann 2005). In other classifications, the degeneration of the frontal and temporal lobes is only divided into the *frontal variant of FTD* (fvFTD) / *behavioural variant of FTD* (bvFTD) and *temporal variant of FTD* (tvFTD) / *language variant of FTD (lvFTD*), the first variant being identified with FTD and the second variant with SD and PNFA (see, e.g., Bozeat et al. 2000; Perry and Hodges 2000; McKhann 2001; Bidzan 2012).

In the first decade of the 21st century, a team of researchers led by Maria Luiza Gorno-Tempini proposed a new classification of diseases related to the degeneration of frontal and/or temporal lobes, which manifest themselves through selective, progressive and dominant linguistic disorders. Ultimately, three main clinical forms of *primary progressive aphasia* (PPA) were distinguished: *nonfluent variant of PPA* (nfvPPA), *semantic variant of PPA* (svPPA) and *logopenic variant of PPA* (lvPPA) (see Gorno-Tempini et al. 2004, 2008, 2011).

Clinical symptoms, which mainly include a gradually increasing multimodal loss of semantic knowledge, are most commonly referred to as semantic dementia. However, other names and descriptive terms of the disease can be found in literature, for instance "primary progressive sensory transcortical aphasia with visual agnosia", "primary progressive semantic aphasia", "fluent primary progressive aphasia", "semantic variant of primary progressive aphasia" (svPPA), "temporal variant of frontotemporal dementia" (tvFTD), while Japanese literature uses the term "aphasia gogi" (Japanese *gogi*; literally: word meaning) (see, e.g., Kertesz, Polk and Kirk 1992, 1998; Tanabe et al. 1992; Bozeat et al. 2000; Perry and Hodges 2000; McKhann 2001; Gorno-Tempini 2004, 2011; Adlam et al. 2006).

Regardless of the aforementioned differences in classifying and naming the disease which manifests itself as progressive aphasia-type language deficiencies, researchers have no doubt that it is a completely separate category of disorders, constituting an independent clinical entity (see Mesulam 1982, 2001; Kertesz, Davidson and McCabe 1998, 2003; Jodzio 1999; Adlam et al. 2006; Harciarek and Kertesz 2009; Olszewski 2008; Sitek et al. 2008; Harciarek 2012; Pąchalska 2012).

CHARACTERISTICS

The axial symptom of semantic dementia is a gradual anomia connected with difficulties in understanding the meanings of previously known words. The impoverished lexis and loss of semantic knowledge affects both active and passive vocabulary (in speech and writing) (Kertesz, Davidson and McCabe 1998; Grossman and Ash 2004; Hodges and Patterson 2007; Harciarek, Jesso and Kertesz 2008). Initially, comprehension deficits mainly concern personal and geographical names and chrematonyms (names of some individual or serial industrial prod-

ucts) that are rarely used in a particular language (Kipps, Knibb and Hodges 2007; Caine, Breen and Patterson 2009). As the disease progresses, lexical-semantic problems also include common words. Initially, problems with actualisation may concern only selected semantic categories (*category-specific deficits*) (Lambon Ralph et al. 2003). Over time, they also include names of everyday objects. Patients ask their caregivers about the meaning of some words, usually nouns, occurring in the utterances addressed to them (see, e.g., CAREGIVER: *Give me a grater*. PATIENT: *Grater? What is a grater?*) (see Kertesz et al. 2010).

The loss of semantic knowledge is not "purely" linguistic. Over time, the semantic deficit covers all modalities that enable people to recognise objects of extra-linguistic reality with the senses of sight, hearing, touch, smell or taste. Patients lose not only the ability to name individual objects, but also to recognise their specific features. Not only are the patients unable to call a parrot *a parrot*, but they also lose knowledge of the fact that these birds have colourful feathers and a strong hook-like beak, and that some parrot species have the ability to imitate human speech. The patients are unable to actualise the word *horse* when seeing a photograph of a horse or hearing the sound of neighing.

The loss of multimodal conceptual knowledge distinguishes patients with AD or the logopenic variant of PPA from those with SD. The former may have problems with correctly naming food products, for example, but will be able to choose items needed to prepare a particular dish and process them properly (cut, fry, cook them, etc.). Meanwhile, SD patients will not be able to either distinguish between the different products or use them properly. SD patients sometimes "cook" soup with groats and kefir, or hollow out the tomato pulp like a seed nest of bell peppers when making a salad (Sitek et al. 2008).

In the course of the disease, the utterances made by SD patients become progressively impoverished in terms of content (the so-called "empty speech"). They are dominated by verbs while nouns are increasingly rare, replaced by demonstrative pronouns, for example, *this one, that one, such, here, there*. Unable to actualise a word, patients frequently use co-hyponyms that are more typical within a particular semantic category (e.g. *pigeons* instead of *canaries*, *apples* instead of *tangerines*) or more common hyperonyms (e.g. *birds* instead of *canaries*, *fruits* instead of *tangerines*). Much less frequently than in other neurodegenerative diseases with anomia, SD patients use periphrasis or support their utterances with gestures. Answers to questions asked by caregivers, for example, often sound like incomprehensible sequences of unrelated words (violated lexical collocations and phraseology).

Nearly all patients with SD experience deep disorders of linguistic pragmatics at an early stage of the disease. These include, e.g., excessive and unrestrained propensity to converse, inappropriate content, thematic perseverations and stereotypies (see Kertesz et al. 2007, 2010).

Moreover, in the early stages of the disease, SD patients already reveal specific problems in reading and writing, referred to, respectively, as surface dyslexia and surface dysgraphia respectively (Jefferies et al. 2004; Caine, Breen and Patterson 2009; Fushimi et al. 2009). Generally speaking, these disorders are characterised by incorrect reading and writing of words where the grapheme-phoneme relation while reading and the phoneme-grapheme relation while writing is not a one-to-one relation, i.e. there is no strict grapheme-phoneme correspondence. Problems of this kind manifest themselves in particular in the case of languages with the so-called deep orthography, e.g. in French or English spelling (see Frost, Katz and Bentin 1987; Katz and Frost 1992).

Patients with SD essentially retain phonological and grammatical (morphological and syntactic) competence. Their statements relatively rarely include phonemic paraphasias or agrammatisms (Adlam et al. 2006; Rohrer et al. 2008). The patient's lack of response to phonemic hints (e.g. providing the initial syllable of a word) when solving crosswords or playing scrabble also proves that the disorder concerns the semantic rather than the phonological level (Sitek et al. 2008). Patients with SD have no difficulty repeating words or entire sentences, but demonstrate problems with understanding during such attempts (see e.g., INVESTIGATOR: *Please repeat the following words: apple, window, shoe*. PATIENT: *Apple, window, shoe. What do you want me to do? What is an apple?*) (Harciarek 2012). When defining certain names or concepts, SD people usually provide very general or completely wrong definitions (Kipps, Knibb and Hodges 2007). The degradation of semantic memory ultimately results in mutism (Kertesz et al. 2008).

As the disease progresses, other cognitive disorders are added to the isolated lexical-semantic deficits. Some of the first symptoms include associative visual and sensory disorders (Kertesz, Davidson and McCabe 1998; Jodzio, 1999). Patients display better visual or tactile recognition of objects they use more frequently (e.g. a fork is identified more easily than a can opener) (Hodges and Patterson 2007). Prosopagnosia (impaired ability to recognise familiar faces) is observed in some SD patients, combined with loss of knowledge about specific individuals. Initially, the problem affects mainly distant relatives or rarely seen acquaintances, but over time it also covers the patients' loved ones (Thompson, Patterson and Hodges 2003; Hodges and Patterson 2007).

As the disease progresses, the aforementioned cognitive impairments are accompanied by behavioural disturbances characteristic of the frontal variant of FTD. Excessive sociability and uninhibited behaviour are particularly common in this group of patients. Sometimes the so-called user behaviour is also observed, manifesting the patient's environmental dependency. The patients automatically reach for objects visible in their vicinity and use them as intended, e.g. when they see a comb, they start to comb their hair, when they see rain falling outdoors, they

open an umbrella indoors. They behave in this way even though they have neither the intention or the need to do so at the moment (Sitek et al. 2014). Some patients demonstrate compulsive behaviours, e.g. they might arrange jigsaw puzzles for hours or look at their watches constantly (Seeley et al. 2005). Like patients with the behavioural variant of FTD, SD patients may reveal changes in dietary preferences and obsessions with certain products (food fads), e.g. a constant desire to eat only bananas or sweets combined with drinking only milk (Kertesz 2006). These changes may also include attempts to consume inedible products (Snowden et al. 2001).

Despite increasing cognitive, linguistic and behavioural deficits, SD patients reveal surprisingly well-preserved episodic and autobiographical memory (Kertesz, Davidson and McCabe 1998; Graham et al. 2000; Hodges and Graham 2001; Scahill, Hodges and Graham 2005). They also do not display deficits in terms of temporal or spatial orientation (Hodges et al. 1992). Moreover, those patients usually have well-preserved perceptual and spatial functions and motor skills (Kertesz, Polk and Kirk 1992; Kertesz, Davidson and McCabe 1998).

DIAGNOSTIC CRITERIA

The diagnostic criteria for semantic dementia were first developed by John R. Hodges et al. in 1992 and further fine-tuned in 1998. In 2011, a team of researchers led by Marie Louise Gorno-Tempini proposed new three-level diagnostic criteria for neuropsychological, neuroimaging and histopathological research.

Clinical SD[2] diagnosis (level one) can be made when confrontational naming and understanding of single words is impaired. In addition, at least three of the following characteristics must be present: impaired knowledge of objects (particularly those that are rarely used or little known), dyslexia or surface dysgraphia,[3] preserved repetition and/or preserved verbal expression (grammar, articulation).

The results of neuroimaging examinations (level two) represent an additional aspect that enhances clinical diagnosis. MRI must show predominant atrophy of the anterior temporal lobe whereas PET and/or SPECT must show hypoperfusion in the aforementioned regions of the brain. The results of histopathological examination (level three) should indicate a specific neurodegenerative pathology, e.g. FTLD-tau (frontotemporal lobar degeneration tau), FTLD-TDP (frontotemporal

[2] In the nomenclature used by M.L. Gorno-Tempini et al., this is a semantic variant of progressive primary aphasia.

[3] According to E.J. Sitek et al. (2014), dyslexia and surface dysgraphia as a symptom in SD are of little diagnostic significance in the case of Polish language, because the grapheme-phoneme relationship (and the reverse relationship) in the Polish language is generally regular, and deviations only concern foreign borrowings with low or very low frequency (e.g. *pinceta*, *loggia*), which means that dyslexic and dysgraphic errors may depend on the patient's education.

lobar degeneration-transactive response DNA binding protein), or the presence of a known pathological mutation (Gorno-Tempini et al. 2011).

For SD to be diagnosed, full neuropsychological diagnostics must be conducted in order to exclude cognitive disorders specific to other neurodegenerative diseases. According to Emilia J. Sitek et al. (2008), basic neuropsychological differential diagnosis of SD should include the assessment of memory (semantic, episodic and operational), visual and spatial functions, visual gnosis, non-verbal problem solving and language functions. It would also be advisable to complement it with an assessment of executive functions, calculia and praxis.

The diagnosis of language functions should be performed by a neurologopedist (or a speech therapist for the elderly) in collaboration with a neuropsychologist. The diagnosis should include assessment of spontaneous speech (in terms of pronunciation, grammatical and lexical correctness), verbal fluency, confrontational naming of visually presented objects, repetition and actualisation of automated sequences, understanding of words, expressions, phrases and sentences (simple and syntactically complex), semantic knowledge as well as reading and writing. The most important element of the diagnosis of language functions should be the assessment of the depth of anomia and the dominant type of naming errors (Roher et al. 2008). During the examination, the most important types of anomia should be considered, such as prioprial anomia (impaired actualisation of proper names), appellative anomia (impaired actualisation of common nouns), word selection anomia, e.g. an *apple* in the meaning of a *tangerine*, *John Paul II* in the sense of *pope Francis*, category-specific anomia, and modality-specific anomia (McKenna and Warrington 1980; Semenza 1997; Harley 2008; Kertesz 2010).

Language functions can be evaluated using diagnostic tools for poststroke aphasia and other language tests such as the Boston Diagnostic Aphasia Examination (BDAE), including selected tests from the BDAE battery prepared by Hanna K. Ulatowska, Maria Sadowska and Danuta Kądzielawa) (Goodglass and Kaplan 1983; Ulatowska, Sadowska and Kądzielawa 2004), the Boston Naming Test (BNT) (Kaplan, Goodglass and Weintraub 1983), the Western Aphasia Battery (WAB),[4] prepared by Maria Pąchalska and Bruce D. MacQueen) (Kertesz 1982; Pąchalska and MacQueen 1997), the set of tests to study cognitive processes in patients with brain damage, developed by Włodzimierz Łucki (1995), the dictionary from Wechsler Adult Intelligence Scale (WAIS-R; in the Polish re-normalised version) (Pearson Education 2008; Brzeziński et al. (2004), the Controlled Oral Word Association Test (COWAT) (Benton and Hamsher 1976), the California Verbal Learning Test (CVLT) (Delis et al. 1987) or the Verbal Concept Attainment Test (VCAT) (Rosen 1962). Unfortunately, some of these tools have not been adapted, standardised or normalised in Poland.

[4] A revised version of this instrument (Kertesz 2007) is now available.

PHARMACOLOGICAL TREATMENT AND NEUROLOGOPEDIC THERAPY

Attempts at pharmacological treatment of SD, including attempts to slow down the neurodegenerative process, have not yet produced satisfactory results. Psychotropic drugs are used to mitigate neuropsychiatric symptoms such as uninhibited behaviour, sugar cravings or compulsive behaviours (Hodges and Patterson 2007; Pąchalska 2008).

Neurologopedic therapy, with the main goal to improve the patient's communication skills, brings positive results at the initial stage of the condition. During rehabilitation, the therapist should focus on maintaining the language skills, the ability to build statements, and the use of verbal clues and hints. It should also place emphasis on creating compensatory communication strategies and indicate alternative methods of communication (using gestures, drawing, using a picture dictionary, etc.).

The meta-analysis conducted by Maya L. Henry, Pélagie M. Beeson and Steven Z. Rapcsak (2008) has proven that rehabilitation of semantic anomia brings positive results: patients re-learn forgotten words and meanings. The results of some of the aforementioned studies also prove that lexical-semantic exercises may slow down the progress of anomia. However, the authors emphasise that therapeutic efforts are beneficial in the early stages of the disease, when preserved semantic knowledge and relatively well-functioning episodic memory support learning.

SUMMARY

Semantic dementia remains a serious problem for modern science, both at the stage of diagnosis and during the therapeutic process. Neuropsychological and neurologopedic assessment is an essential element of the differential diagnosis of SD. It requires both standard methods of assessing cognitive functions (including the cognitive function of language) and dynamic experimental tests. Pharmacological therapy offers little therapeutic benefit. Neuroogopedic rehabilitation (mainly lexical-semantic exercises) is an important element of the therapeutic process, at the initial stage of the disease It is also important to provide psychological support to the caregivers of SD patients and appropriate psycho-education.

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