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Dementia and Language

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Introduction

Dementia is a generalized loss of functions that results from cerebral disease. Dementia occurs in absence of acute confusion (i.e., the deficits do not occur exclusively during the course of a delirium). According to the American Psychiatric Association's *Diagnostic and statistical manual of mental disorders* (DSM-IV), a dementia syndrome is characterized by multiple deficits in cognition, including memory impairment, which are the direct consequence of physiological changes. The DSM-IV criteria require that these deficits must be of a sufficient magnitude to impair social or occupational function. Historically, diagnostic classifications for dementia have included subtypes based on characteristics such as typical symptoms presentation, the progression and course of the disease, and psychiatric and behavioral features, as well as presumed causes (e.g., a general medical condition, persisting effects of a substance, multiple etiologies). The concept of dementia has evolved over the past hundred years. It has long been associated with a progressive decline of cognitive functions and with an irreversible course. Nowadays, definitions of dementia are descriptive and rely on typical symptoms presentation, and thus do not necessarily imply a progressive degeneration. However, the primary progressive dementias are most common and often present with language and communication disorders.

As the population in Western countries ages, the prevalence of progressive dementias resulting from

brain diseases increases. Recent epidemiologic studies have suggested that the prevalence of dementia in industrialized countries is approximately 1.5% at age 65 years, rising to approximately 25% by age 80 years (Lobo *et al.*, 2000). For example, according to the Canadian Study of Health and Aging (1994), more than 364 000 Canadians over 65 suffer from dementia. Among these individuals, 65% presented with dementia of the Alzheimer's type (DAT) whereas the remaining suffer from vascular dementia (VaD) and other forms of dementia. Moreover, it is predicted that over three-quarters of a million Canadians will have Alzheimer's disease and related dementias by the year 2031. Thus, progressive dementia is becoming an increasingly important public health concern across the world.

Diagnostic Criteria and Differential Diagnosis

In recent years, with the growing concern for early diagnosis of progressive dementia (i.e., dementia resulting from a gradual degeneration of the brain), several researches have been conducted on prodromal forms of dementia. Mild cognitive impairment (MCI) refers to the clinical condition between normal aging and DAT in which persons experience memory loss to a greater extent than one would expect for age, yet they do not meet currently accepted criteria for clinically probable Alzheimer's disease. Work from Petersen and colleagues on MCI provided for the diagnostic criteria that are commonly accepted (Petersen *et al.*, 2001; Petersen, 2004). Recent studies suggest that a percentage of individuals presenting with mild cognitive impairment later developed typical symptoms of progressive dementia, especially DAT. According to Petersen *et al.* (2001), a clinical diagnosis of MCI requires a memory complaint, preferably corroborated by an informant, and an impaired memory function. Although a subtype of MCI may present with cognitive deficits other than memory, the amnesic type presents with salient memory failures in conjunction with a preserved general cognitive functioning and intact performance in activities of daily living. Individuals diagnosed with MCI are, however, not demented.

Because of the absence of biological markers or simple diagnostic methods, the early detection of dementia relies on various assessments, performed to rule out other possible causes and to identify specific forms of the disease (i.e., differential diagnosis). Neuropsychological testing plays an important role in the assessment of individuals with cognitive impairment. A large number of studies have sought to identify the neuropsychological features that distinguish the different forms of dementia. What emerges from these studies are descriptions of cognitive functioning among which some distinctions are useful for the differential diagnosis of dementia. For example, there are remarkable differences between patients with DAT and VaD with respect to verbal long-term memory and executive functions. However, these patients also presented with similar language, constructional ability, attention, and memory deficits (Looi and Sachdev, 1999).

This inconsistency and overlapping of cognitive deficits is certainly due to the important heterogeneity of neuropsychological manifestations in the early stages of the disease (for a review, see Rosenstein, 1998) as well as to methodological problems, including the application of diagnostic criteria, selection of neuropsychological tests (aiming at large cognitive domains instead of more precise components of cognition) and lack of adequate matching of patients groups. As we mentioned above, the definition of dementia continues to evolve. Current studies have refined clinical criteria of dementia and, for example, have made possible the distinction between the different presentations of frontal lobe disease.

Language Disorders in the Major Forms of Dementia

With respect to language impairments, neuro-epidemiological and neuropsychological studies propose clinical linguistic profiles usually associated with common forms of dementias. Recent neuropsycholinguistic studies also largely contribute to a better characterization of language deficits in dementia by specifically identifying functional localization of impaired and preserved processing components and subcomponents of the linguistic processing system. In the following sections we will review current data on language and communication disorders in the most frequent primary progressive syndromes of dementia, namely DAT, VaD and dementia with Lewy Bodies, as well as in specific forms of frontotemporal lobe disease, namely frontotemporal dementia (FTD), progressive nonfluent aphasia (PNA) and semantic dementia (SD).

Language Disorders in Dementia of the Alzheimer Type

DAT presents with an association of cardinal features that allow for differential diagnosis. It also refers to probable Alzheimer's disease because pathological verification is necessary for a definitive diagnosis, that is, a primary degenerative dementia with a distinctive pathology (i.e., neurofibrillary tangles and

senile plaques). According to McKhann and colleagues, a diagnosis of probable Alzheimer's disease requires that an individual presents with a dementia syndrome established by clinical examination and documented by screening tests and procedures such as the Mini-Mental State Examination and confirmed by neuropsychological tests (McKhann *et al.*, 1984). Furthermore, cognitive deficits in two or more areas of cognition are required. A progressive worsening of memory and other cognitive functions characterizes the progression of the disease. These symptoms occur in clear consciousness and usually onset between age 40 and 90 years. There must be an absence of systemic disorders or other brain diseases that in and of themselves could account for the progressive deficits in memory and cognition (For a review of concepts and diagnostic criteria of DAT, see Kennedy *et al.*, 2001).

As for other types of dementia, DAT does not begin with global impairment in cognitive functions, but usually progresses through different stages. The most pervasive characteristic of DAT is undoubtedly a progressive episodic memory loss associated with decline in other cognitive areas. During the prodromal stage, which generally lasts about 2 years, cognitive problems are usually too mild to clearly distinguish from age-related changes and are not always identified through extensive testing. With respect to language, DAT patients often complain about word retrieval difficulties at this stage of the disease. They also sometimes report having difficulties in initiating conversation or understanding inferences or humor. Language deficits are usually more prominent after approximately 2 or 3 years from onset. Symptoms of anomia are more marked, especially for less used vocabulary. In conversations, patients mainly produce circumlocutions as well as generic and imprecise terms while semantic errors are still scarce. At this stage of illness, phonetic, phonemic, and syntactic aspects of language are preserved. The patients perform relatively normally in tests exploring reading aloud, repetition, and auditory comprehension. However, they may show signs of impairment in writing, confrontation naming and fluency tasks. Language is much more affected in the middle or intermediate stage of DAT, a stage that generally occurs between the third and the fifth year from onset. Phonetic and phonological abilities are still preserved but spontaneous speech is characterized by severe reduction, stereotyped utterances, and important anomia manifested in the production of verbal and semantic paraphasias as well as by occasional neologisms. Repetition and reading aloud are still often preserved while there is an important worsening of comprehension and spelling. In the final stage of the disease (after the fifth to six year post onset), all linguistic abilities are impaired. There is a severe deficit of comprehension and oral expression is impossible or limited to automatisms, verbal perseverations or stereotyped expressions.

Even if a large proportion of DAT patients presented with this standard description of language impairments, recent group studies have shown that there is an important lack of homogeneity across patients, with respect to (1) the evolution of the disease, (2) the relative preservation or damage of the different cognitive functions, and (3) the relative preservation or damage of specific components in each cognitive function, including language (e.g., Schwartz, 1990; Price *et al.*, 1993). By resorting to cognitive models of language processing, neuropsycholinguistic studies have largely contributed to the identification of functional origins of language deficits in DAT. What emerges from these studies is that the main impact of the disease falls on semantic memory, a component of explicit long-term memory which contains the permanent information related to objects, concepts, words, and their meanings. Because of its central role to the processing of language, the deficit affecting semantic memory leads to important difficulties in word comprehension and production. Therefore, when tested with comprehensive batteries of language, DAT patients usually show poor performance in picture naming, word–picture matching, picture or word sorting, and semantic questionnaires, even at the first stage of the disease. Moreover, the consistency for individual items across tests (i.e., the same items are preserved or damaged across tests), as well as the relative preservation of superordinate knowledge as compared to specific and detailed knowledge (e.g., knowing that a zebra is an animal but not that it has stripes or lives in Africa), is suggestive of a deterioration of semantic representations instead of access damage to preserved semantic representations. This question is, however, controversial, other researchers having reported experimental data that rather support a normal organization of semantic memory and an access deficit in DAT (For an extensive discussion, see Luzzatti, 1999). Furthermore, with respect to semantic memory, recent studies have also investigated whether the semantic loss in DAT affects some categories more than others. Most of these studies conclude that DAT patients perform worse on biological concepts (i.e., animals, fruits, vegetables, etc.) than on artifacts (i.e., vehicles, furniture, tools, etc.) (e.g., Chertkow and Bub, 1990). However, the opposite pattern was also found in other studies, while yet others failed to demonstrate any category effects. (For a review, see Whatmough and Chertkow, 2002.)

Articulatory, phonological, and syntactic abilities are usually considered unscathed until the final stage

of DAT. Recent studies, however, showed that, with the progression of the disease, these abilities may be damaged. For example, Croot and her colleagues (Croot *et al.*, 2000) have shown that, in less typical cases of DAT, phonological and articulatory abilities may be impaired, sometimes as a selective deficit. Syntactic processes required for sentence production (e.g. Bates *et al.*, 1995) and sentence comprehension (e.g., Waters, *et al.*, 1995) may also be compromised in some cases of DAT, even at the early stage of the disease. Whether this deficit is really syntactic in nature or is more explicable in terms of working memory disorder or in terms of semantic interpretative processing remains unresolved.

Studies on reading and writing impairments in DAT also reflect a relative lack of consistency. As mentioned earlier, according to the standard clinical portrait of DAT, reading and spelling abilities are considered to be largely preserved in patients even at rather advanced stages of the disease. In contrast to this classical conception, several recent single-case and group studies, resorting to cognitive neuropsychological or to paralleled-distributed-processing models have shown that the ability to read aloud and to spell words is frequently affected in DAT. These patients often demonstrate characteristics of surface alexia and surface agraphia: they are better at reading/spelling orthographically regular than irregular words (i.e., words with exceptional or unpredictable sound-to-spelling and spelling-to-sound correspondences), they show a preserved ability to read/write nonwords, and they tend to produce regularization errors in reading (e.g., bread → /brid/) and phonologically plausible errors in spelling (e.g., crane → CRAIN). Recent group studies, however, have shown that different patterns of written language impairment may be observed in DAT, a result that contradicts the hypothesis that the disease selectively impairs lexical-semantic routes of reading and writing (for a discussion and a review, see Luzzatti, 1999; Graham, 2000; Noble *et al.*, 2000).

Language Disorders in Vascular Dementia

The concept of VaD significantly changed in the 1990s with the publication of international diagnostic criteria (e.g., Roman *et al.*, 1993). VaD may result from an array of causes. It includes all dementias following ischemic or hemorrhagic cerebrovascular accidents, i.e., single vascular insult (most typically to a critical area of the brain), repeated cerebral infarction (also referred to as multi-infarct dementia), and chronic ischemia without discrete infarction. According to Roman *et al.* (1993), a diagnosis of probable VaD requires a loss of cognitive ability (i.e., a decline in memory and intellectual abilities that impairs functioning in daily living). The decline should be demonstrated by a loss of memory and deficits in at least two other cognitive domains. Additionally, cerebrovascular disease (CVD) must be defined by the presence of focal neurological signs consistent with stroke (with or without a history of stroke) and relevant evidence of CVD on computerized tomography or other cerebral imagery techniques. A temporal relation between dementia and CVD as shown by onset of dementia within 3 months following a documented stroke, abrupt deterioration in cognitive functions, or fluctuating, stepwise progression of cognitive deficits is also required (for a review, see Bowler and Hachinski, 2003).

In contrast with the extensive literature on language deficits in DAT, the patterns of cognitive deterioration in other forms of dementia are much less known. Consequently, the identification of clear neuropsychological differences between the major progressive dementias remains difficult to establish. In VaD, for example, most of the reported descriptions related to cognitive deficits concern behavioral manifestations and large cognitive domains. The most commonly reported cognitive impairments in VaD are bradyphrenia (i.e., slowed cognition), dysexecutive symptoms (e.g., difficulty in problem solving and planning), and decreased initiation and spontaneity. There are very few studies that have systematically described language disorders in VaD. Most of them were conducted in an attempt to differentiate VaD from DAT. The majority of these studies suggested that there was no significant difference in language function between the two dementia syndromes. However, in a few recent studies, VaD patients performed better than DAT patients in initial-letter-based verbal fluency tests (e.g., Lafosse *et al.*, 1997), an assessment task that is usually thought to reflect executive dysfunction. As compared to DAT too, anomia is less severe in VaD patients. On a qualitative point of view, they also show a very similar pattern of general naming errors (i.e., visuoperceptual, semantic, and phonemic) when compared to DAT (e.g., Lukatela *et al.*, 1998).

As of now, the nature and the prevalence of reading and writing deficits in VaD remain almost completely unknown. A few recent studies that have included reading and writing tasks in the neuropsychological assessment found that both abilities appeared to be more impaired in VaD. It is, however, impossible to clearly determine the functional origins of written language deficits. For written spelling, different investigations are suggestive of peripheral impairments (e.g., difficulty in writing letters and copying of sentences). Others found that, as compared to DAT,

VaD patients produced more spelling errors and produced grammatically less complex sentences, therefore suggesting a central origin of the deficit (see Graham, 2000).

Finally, as compared to DAT, the speech of individuals with VaD is less empty and conveys more information but they tend to produce shorter and less grammatically complex phrases (Powell *et al.*, 1988). The same authors also have shown that VaD patients often present with abnormal prosody and articulation.

Language Disorders in Dementia with Lewy Bodies

Together with DAT and VaD, DLB is another common form of progressive dementia. However, this entity is a relatively recent one, and as such, there is debate on its classification. DLB is commonly regarded as a Parkinson-plus syndrome that bears the clinical features of both Alzheimer's disease and Parkinson's disease. Pathologically, the cortical neurons of individuals presenting with DLB contain, as the name implies, Lewy bodies or intracytoplasmic inclusion bodies. Clinically, the disease presents with a grouping of distinctive symptoms. According to McKeith *et al.* (1996), the clinical presentation must include a dementia plus two of the following: a fluctuating cognition with pronounced variations in attention and alertness, recurrent visual hallucinations, and spontaneous motor features of parkinsonism (i.e., parkinsonian extrapyramidal symptoms) (for a review see Salmon *et al.*, 2001).

In comparison with DAT, relatively little is known about the cognitive deficits in DLB. A few retrospective studies have compared the performance of DAT and neuropathologically confirmed DLB on a range of neuropsychological assessment, including language. As with DAT, DLB patients show significant impairments in all areas of cognition. Both groups display similar deficits in episodic memory and language (confrontation naming, semantically based verbal fluency) while attention, initial-letter-based verbal fluency, visuoperceptual/spatial abilities, and psychomotor speed are usually more affected in DLB. There is almost no study that specifically addresses the question of the deterioration of language abilities in DLB. As an exception, Lambon Ralph and his colleagues (Lambon Ralph *et al.*, 2001) have recently tested the hypothesis that semantic processing is affected in DLB. Through a group study of 10 DLB and 10 DAT patients, they have shown that semantic impairment is not limited to DAT. Both demented groups exhibited impaired performance in a semantic assessment battery. However, while DAT patients showed equivalent deficit in every modality, DLB patients demonstrated more severe deficits when they were presented with pictures than with words, a performance interpreted by the authors as the result of a combination of semantic and visuoperceptual impairment.

Language Disorders in Frontotemporal Lobe Disease

FLTD lobe disease refers to a heterogeneous group of neurodegenerative disorders and has been known under several different names, including frontal lobe dementia, Pick complex, and, more recently, frontotemporal lobar degeneration. FTLD is often used as an umbrella term to cover the different, but related, clinical presentations that involve a degeneration of the frontal and temporal lobes of the brain (McKhann *et al.*, 2001). According to recent updating of diagnostic criteria (Neary *et al.*, 1998; McKann *et al.*, 2001), three main types of FTLD may be described: (1) frontotemporal dementia or frontal lobe dementia (FTD), (2) progressive fluent aphasia or semantic dementia (SD), and (3) progressive nonfluent aphasia (PNA) . However, there is still a lack of consensus on the clinical classification of the FTLDs (for a discussion and a review, see Hodges and Miller, 2001). In the following subsections, we will briefly review current data on language and communication disorders in the three main types of frontotemporal lobe disease.

Language Disorders in Frontotemporal Dementia

According to Neary *et al.* (1998), FTD is marked by character changes and a disordered social conduct. These are the dominant features initially and throughout the disease's course. A diagnosis of FTD requires an insidious onset and gradual progression, an early decline in social interpersonal conduct, an early impairment in regulation of personal conduct, and an early emotional blunting and loss of insight (see Hodges and Miller, 2001). The detection of cognitive deficits at the early stage of the disease often remains problematic. Some patients show evident cognitive deficits at presentation while others may perform almost perfectly on a general neuropsychological battery. With respect to specific cognitive areas, memory is considered to be relatively spared in FTD but recent studies have shown that patients may present with impaired episodic and working memory, with a pattern different to what was encountered in DAT. In comparison to DAT too, instrumental functions of perception, spatial skills, and praxis are usually intact or relatively well preserved. Language also appears to be largely spared in FTD patients, who often show a reduction in spontaneous speech but perform

normally on tests exploring lexical-semantic abilities (e.g., naming, word–picture matching).

Language Disorders in Semantic Dementia

SD is a clinical syndrome that results from a degenerative disease of the temporal lobes. The core features of SD include: (1) the selective impairment of semantic memory, causing important difficulties in word production and comprehension, (2) the relative sparing of the grammatical and phonological structure of language, (3) normal perceptual skills and nonverbal problem-solving ability, and (4) relatively spared autobiographical and episodic memory (for a review, see Hodges *et al.*, 1998). In other words, semantic dementia is marked by a selective impairment of semantic memory with a relative sparing of nonsemantic aspects of language.

As already mentioned, semantic memory occupies a central place in cognition and language processing. Therefore, SD patients experience important deficits in every cognitive task requiring the activation of semantic representations. Spontaneous speech is usually fluent, well articulated and grammatically correct but present many signs of word-finding difficulties: miscarried sentences, latencies, and occasional semantic paraphasias. Repetition is typically well preserved for both words and nonwords while the ability to repeat short sentences or strings of unrelated words depends on the deterioration/preservation of their meaning. SD patients usually complain about loss of memory for words. This deficit is obvious in tests of naming to definition and confrontation naming in which patients mainly produce semantic errors consisting in the production of a superordinate (lion → animal) or a category co-ordinate (lion → tiger) concept. This pattern of errors reflects a loss of attribute knowledge along with preservation of general superordinate information. SD patients are also impaired on tests of verbal and nonverbal semantic memory. Spoken and written single-word comprehension is affected in tasks of word–picture matching, synonymy judging, word sorting, etc. The centrality of the semantic deficit is also confirmed by tests exploring nonverbal semantic knowledge, such as tests of semantic relatedness judgment on pictures, picture sorting, etc. Whether the semantic deficit in SD is more severe for one category of items than another (i.e., category specific) is also discussed in the literature (for a review see Garrard *et al.*, 2002).

Reading and spelling impairments have also been reported in many cases of SD. These patients often demonstrate surface alexia and surface agraphia characteristics. A relationship between semantic impairment on the one hand and impaired word reading (e.g., Funnel, 1996) and impaired word spelling (e.g., Macoir and Bernier, 2002) on the other has been proposed. This hypothesis is questioned, however, since there have been cases reported in which irregular words are read or spelled correctly without any evidence of comprehension.

Language Disorders in Progressive Nonfluent Aphasia

In 1982, Mesulam reported six patients who showed a syndrome of slowly progressive language impairment without associated cognitive or behavioral disorders. This progressive nonfluent aphasia (PNA) shares the features of FTD but a disorder of expressive language is the dominant feature initially and throughout the course of the disease (Neary *et al.*, 1998). Other aspects of cognition are intact or relatively well preserved. A diagnosis of PNA also requires an insidious onset and gradual progression with primarily nonfluent spontaneous speech and at least one of the following: agrammatism, phonemic paraphasias, and anomia. Other clinical features as the presence of stuttering or oral apraxia and the preservation of social skills also support the diagnostic.

Patients with PNA present with complaints of difficulties in expressing themselves and finding words. Their spontaneous speech is markedly slow, hesitant, and sometimes agrammatic. Articulation as well as prosody is affected. Semantic errors and circumlocutions are rare but patients often produce phonological errors. Other patients sometimes present with stuttering or slow dysprosodic speech with verbal apraxia. Because of the spoken output deficit, PNA patients perform poorly on words, nonwords, sentence reading aloud, and repetition tests. Naming tasks are also affected, with word-finding difficulties and production of phonological errors. Their performance is, however, unaffected in every test of semantic memory. Except for complex syntactic structures, comprehension is normal in the early stages of the disease. With disease progression, the breakdown of phonological abilities gradually compromises speech comprehension and production. At later stages, patients also present with difficulties in other cognitive areas.

Conclusions

In this article, we have briefly described verbal communication disorders accompanying the major forms of primary progressive dementia as well as vascular dementia. These disorders are often prominent symptoms of the disease and may occur as early symptoms. We have shown that substantial information is available for language disturbances in DAT, SD, and PNA. In contrast with the extensive literature on these dementing illnesses, relatively little is known about

the patterns of language deterioration in other forms of dementia. For example, the few available descriptions of cognitive deficits in LBD, VaD and FTD mainly concern behavioral manifestations and large cognitive domains. More specific abilities are almost completely ignored and it is still difficult to identify functional deficits to language components.

The concept of dementia continues to evolve. Current studies have refined clinical criteria of dementia and, for example, have made possible the distinction between the different forms of FTD. An improvement in clinical diagnosis should result from the study of more specific subcomponents of the different cognitive domains. In this respect, cognitive neuropsychology is surely useful in that it allows the fractionation of many cognitive domains into specialized subcomponents that can be selectively impaired by a neurological affection. For example, such an approach has led to extensive descriptions of language deficits in DAT and semantic dementia. Moreover, further research is also needed to better characterize the effect of the progression of the disease on the various language abilities and components.

See also: Classical Tests for Speech and Language Disorders; Delirium and Language; Narrative and Discourse Impairments; Primary Progressive Aphasia in Nondementing Adults; Speech Impairments in Neurodegenerative Diseases/Psychiatric Illnesses.

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Demonstratives

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The Semantic Properties of Demonstratives

Demonstratives are deictic expressions; examples in English include *this* and *that* or *here* and *there*. These expressions indicate the relative distance of a referent in the speech situation *vis-à-vis* the deictic center (cf. Brugmann, 1904; Bühler, 1936; Lyons, 1977; Fillmore, 1997). The deictic center is defined by the speaker's location at the time of the utterance. For instance, in the following example, the referent of the proximal demonstrative *this* is closer to the deictic center (i.e., the speaker's location at the point of the utterance) than is the referent of the distal demonstrative *that*:

(1) **This one** (here) is mine, and **that one** (over there) is yours.

All languages have at least two demonstratives that indicate a deictic contrast (cf. Diessel, 1999a, 2005a), but the use of demonstratives is not generally contrastive. For instance, in example (2), the distal demonstrative *that* does not indicate a spatial contrast: the referent of the demonstrative may be an element in close proximity to the speaker's location or it may be a referent in great distance:

(2) Can you see **that** spot (on the back of my hand/on the moon)?

In some languages, certain types of demonstratives do not carry a specific distance feature (cf. Himmelmann, 1997; Diessel, 1999: chap 3). For instance, the German demonstrative *das* does not indicate the relative distance of the referent to the deictic center. In order to differentiate between a proximal and distal referent, the locational adverbs *hier*, *da*, and *dort* can be added to *das*, as in example (3), German (Germanic):

(3)

Das	**hier**	gefällt	mir	besser	als
this/that	*here*	*like*	*me*	*better*	*than*
das	**da**(drüber)				
this/that	*over there*				

'this one I like better than that one over there'