The role of pragmatics in the diagnosis of dementia[†]

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Abstract: Dementia is a large and growing public health problem that poses considerable economic and social challenges to many countries around the world. The emphasis of clinical intervention is to delay the onset of severe functional limitations that are associated with poor outcomes and large health and social care costs. For this to be possible, however, clinicians must achieve earlier diagnosis of the condition than is currently the case. I argue that pragmatic language abilities hold promise as early behavioural markers of cognitive impairment. This paper describes some empirical work on the search for pragmatic markers of early cognitive impairment in 27 English-speaking participants with neurodegenerative disorders. With one exception, none of the participants had received a diagnosis of mild cognitive impairment or dementia. Yet, their performance in discourse production tasks suggested they were experiencing early disruption of pragmatic language skills that had been masked in some cases by compensatory strategies.

Appears in: Cummings, L. (2020) 'The role of pragmatics in the diagnosis of dementia', *East Asian Pragmatics*, 5 (2): 147-168. https://doi.org/10.1558/eap.39716

1. Introduction

The study of pragmatics has offered many interesting insights into human verbal and non-verbal communication. From its unique vantage point at the intersection of language and cognition, pragmatics offers us a window onto mental processes without which some of our most sophisticated forms of language use would not be possible. But what if pragmatics were to become more than merely a window onto these processes? What if we could exploit the concepts and principles of pragmatics to tell us when cognitive processes are not as integrated and nimble as they once were in adults without dementia or even mild cognitive impairment? This question has motivated me to consider if pragmatics could play a diagnostic role in adults with neurodegeneration long before conditions like Alzheimer's disease, progressive supranuclear palsy, and Parkinson's disease produce their first devastating symptoms and the patient is already in an advanced state of disease progression. I want to use this paper to explain why I believe pragmatics is a hitherto unexplored resource in the diagnosis of a group of conditions which remain to this day some of the most challenging in the neurologist's caseload.

The discussion begins by examining the extent of the problem that dementia poses for different countries around the world. Dementia presents a uniquely difficult challenge for societies to address. The diseases that cause many forms of the condition are an unwelcome consequence of the success that improvements in medicine and living conditions have had in extending the lives of people worldwide. In 2017, the populations of 71% of countries around the world could expect to live into the eighth decade of life and beyond (The World Bank, 2019), an achievement that was barely imaginable 100 years ago. But a comparison of life expectancy and healthy life expectancy[1] shows that we can expect to spend a significant number of years towards the end of our lives in poor health. In 2016, while average life expectancy at birth of the global population was 72.0 years, healthy life expectancy at birth was 63.3 years (World Health Organization, 2019a). If 8.7 years on average at the end of our lives are compromised by dementia and cognitive impairment, then the economic and social consequences of this situation are very grave indeed.

The paper continues with an examination of the current protocols for dementia diagnosis. A combination of clinical symptoms and results of biomarker tests is used to establish a diagnosis of dementia. It is unfortunately the case, however, that many diagnostic protocols fall far short of an acceptable level of accuracy. Rates of misdiagnosis are alarmingly high across many dementias. It is against this backdrop that behavioural markers have emerged as a serious contender in dementia diagnosis. It is the contention of this paper that clinicians and researchers will be well served by considering pragmatic language skills as a potential behavioural marker in a diagnosis of dementia. The grounds for this view are examined and revolve around the complex interaction that exists between pragmatics on the one hand and a set of cognitive skills known to disintegrate early in the course of many neurodegenerative diseases on the other hand. The paper concludes with discussion of some data and findings from a small-scale, qualitative study of 27 English-speaking participants with different neurodegenerative conditions that can lead to cognitive impairment and dementia.

2. Dementia: a global public health challenge

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In recent years, we have become accustomed to hearing about the growing problem of dementia and the healthcare challenges that age-related cognitive impairment poses for societies. But repeated exposure to media stories about dementia must not have the effect of lessening our concern about the scale of this health problem. Figures for diagnosed cases of dementia are truly staggering and amount to the single biggest public health challenge for many countries around the world. In a systematic review and meta-analysis, Prince *et al.* (2013) estimated that in 2010 there were 35.6 million people worldwide living with dementia. This number is expected to double every 20 years, resulting in some 115 million cases by 2050. Low- or middle-income countries, where the challenge of funding care is likely to be most acute, are expected to account for 71% of cases in 2050. In the United States, Alzheimer's disease is the only top 10 cause of death that did not experience a decrease between 2000 and 2015. While impressive gains were made in the treatment of HIV infection, heart disease, and strokes between 2000 and 2015, with deaths related to these diseases down by 55%, 11% and 16%, respectively, and some more modest decreases occurred in deaths related to breast cancer (–1%) and prostate cancer (–7%), deaths from Alzheimer's disease relentlessly increased (+123%) in the same 15-year period (Alzheimer's Association, 2018).

Recent epidemiological studies also suggest that dementia is a growing public health problem in many Asian countries. In 1990, the number of people with dementia in China was 3.68 million. This increased to 5.62 million in 2000 and 9.19 million in 2010 (Chan *et al.*, 2013). The total number of people with dementia in China is projected to reach 14.1 million by 2020 and 23.3 million by 2030 (Xu *et al.*, 2017). Japan is the most aged society in the world, with an estimated national prevalence of late-onset dementia of 15% (Asada, 2017; Sado *et al.*, 2018). This compares with a national dementia prevalence of 13.9% in individuals aged 71 years and older in the USA in 2002 (Plassman *et al.*, 2007) and a pooled prevalence of 4.03% in individuals 55 years and older in China (Zhu *et al.*, 2019). The prevalence of dementia in Singapore and Korea is also well documented. On one diagnostic protocol, the prevalence of dementia in adults aged 60 years and above in Singapore is 10% (Subramaniam *et al.*, 2015). In 2008, dementia affected 528 per 100,000 population in Korea, a figure that was 4.5% of the total burden of disease in the country that year (Park *et al.*, 2013).

If the prevalence figures for dementia are staggering, then so too are the economic costs associated with the care of individuals with dementia. In 2018, the direct costs to American society of healthcare and long-term care for people with dementia is estimated to have been USD 277 billion. This is reflected in a three to fourfold increase of payments to Medicare beneficiaries with dementia aged 65 years and older (USD 48,028 per person with dementia versus USD 13,705 per person without dementia). By 2050, total healthcare costs are projected to have increased to more than USD 1.1 trillion (Alzheimer's Association, 2018). The estimated total annual costs of dementia in China increased from USD 0.9 billion in 1990 to USD 47.2 billion in 2010. Costs are predicted to reach USD 69.0 billion in 2020 and USD 114.2 billion in 2030 (Xu *et al.*, 2017). Sado *et al.* (2018) estimated that the societal cost due to dementia in Japan in 2014 is USD 112 billion. Even the best developed and funded healthcare systems in the world cannot sustain these costs. One way of reducing the costs of dementia care is to initiate much earlier in the course of neurodegenerative diseases the pharmacological and other interventions that have some prospect of extending an

individual's functional independence. But for this to occur, we need a more proactive approach to dementia diagnosis and management. Simply waiting for people to manifest the first signs of disease before initiating these interventions is not working.

The way forward in dementia care must involve much earlier diagnosis. It is estimated that substantial reductions in health and long-term care costs in the US, totalling around USD 7.9 trillion, could be achieved if 100% of people with Alzheimer's disease were diagnosed at the point when they are experiencing mild cognitive impairment (Alzheimer's Association, 2018). But even a diagnosis at the stage of mild cognitive impairment is still a 'late' diagnosis when one considers that the neurofibrillary changes associated with Alzheimer's disease can be traced back about 50 years, possibly even extending into adolescence (Ohm *et al.*, 1995). If these early pathological processes could be modified or halted by pharmacological interventions, then this would represent a step change in the treatment of dementia. However, a more immediately worrying scenario is that the ideal world of early diagnosis bears little resemblance to current clinical practice. Today, most people are still diagnosed with Alzheimer's disease without the use of biomarker confirmation and by physicians who have no specific expertise in neurodegenerative disease.[2] We will see later that even when biomarker confirmation is used, there is a large rate of misdiagnosis. Many dementias are diagnosed as Alzheimer's disease, only to be discovered to be some other form of neuropathology at the point of post-mortem examination. A way through these diagnostic challenges must be found if we are to deliver an acceptable level of care to patients with dementia and their families.

3. The need for better dementia diagnosis

The urgent need to improve differential diagnosis of dementia begins with a better understanding of the non-Alzheimer dementias (see Table 1). These conditions are often poorly understood and recognised by primary care physicians, with the result that they are incorrectly diagnosed as Alzheimer's disease (van Hout *et al.*, 2000). This has led to an over-diagnosis of Alzheimer's disease and under-diagnosis of other neurodegenerative dementias. An important first step in improving dementia diagnosis requires that clinicians become more aware of neurodegenerative diseases other than Alzheimer's disease that might better explain patients' symptoms. In my own work, I have encountered patients with corticobasal degeneration, Parkinson's disease, and progressive supranuclear palsy for whom a definitive diagnosis of their condition has taken several years.[3] The extent of this problem was acknowledged in an editorial in *The Lancet* in 2015 in which it was remarked:

> "Underdiagnosis and misdiagnosis of non-Alzheimer's dementias is a key issue. More research is needed, not only for effective pharmacological interventions, but also for differential diagnostic techniques and effective care, to ensure the proper management of patients and to optimise their quality of life."

Improving the diagnosis of non-Alzheimer dementias is a truly formidable task. Individuals with these conditions form a clinically heterogeneous population, with aetiologies including infectious diseases such as HIV, genetic disorders like Huntington's disease, and autoimmune conditions such as multiple sclerosis. These conditions are often managed by different medical specialists including neurologists, psychiatrists, and geriatricians. Some of these conditions are slowly progressive such as Parkinson's disease, while others like

Creutzfeldt-Jakob disease and motor neurone disease have a much shorter clinical course. The conditions marked (#) in Table 1 have been the focus of my work to date. I have either collected data from clients with these conditions or, in the case of vascular dementia and the language variants of frontotemporal dementia, I have used data from DementiaBank, part of the TalkBank system (https://dementia.talkbank.org/). I will later report on the first episode of data collection that took place in the UK in August 2018.

AIDS dementia complex
Binswanger disease
Chronic kidney disease
Corticobasal degeneration #
Creutzfeldt-Jakob disease
Frontotemporal dementia (behavioural variant)
Frontotemporal dementia (language variants) #
Huntington's disease #
Hydrocephalus
Lewy body disease
Motor neurone disease #
Mixed dementia (e.g. Alzheimer's disease and vascular dementia)
Multiple sclerosis #
Multiple system atrophy – cerebellar subtype
Parkinson's disease #
Progressive supranuclear palsy #
Syphilis
Vascular dementia (vascular cognitive impairment) #
Wernicke-Korsakoff syndrome #

Table 1: Non-Alzheimer dementias and associated medical conditions

Clearly, we cannot make progress in the diagnosis of dementia without addressing the neglect of non-Alzheimer dementias. But we also cannot make progress if our current diagnostic methods are not fit for purpose. Clinical evidence suggests that our best available diagnostic techniques are not serving us particularly well. The diagnosis of dementia is typically arrived at through biomarker tests and clinical symptoms. A descriptive classification scheme proposed by Jack *et al.* (2016), known as the A/T/N system, organises

seven major biomarkers of Alzheimer's disease according to three binary categories (see Figure 1). The 'A' and 'T' biomarkers stand for amyloid and tau, the abnormally shaped proteins that cannot be broken down by cell enzymes, the accumulation of which causes nerve cell death and injury. These abnormal proteins may be found in the cerebrospinal fluid (CSF) as well as the cortex of the brain. The 'N' biomarkers indicate neurodegeneration or neuronal injury and dysfunction and include measures of brain atrophy (e.g. hippocampal and cortical volume) on structural magnetic resonance imaging (MRI). The National Institute on Aging and Alzheimer's Association Research Framework for Alzheimer's disease uses this classification scheme to provide the research field with a common language for diagnostic purposes (Knopman *et al.*, 2018).

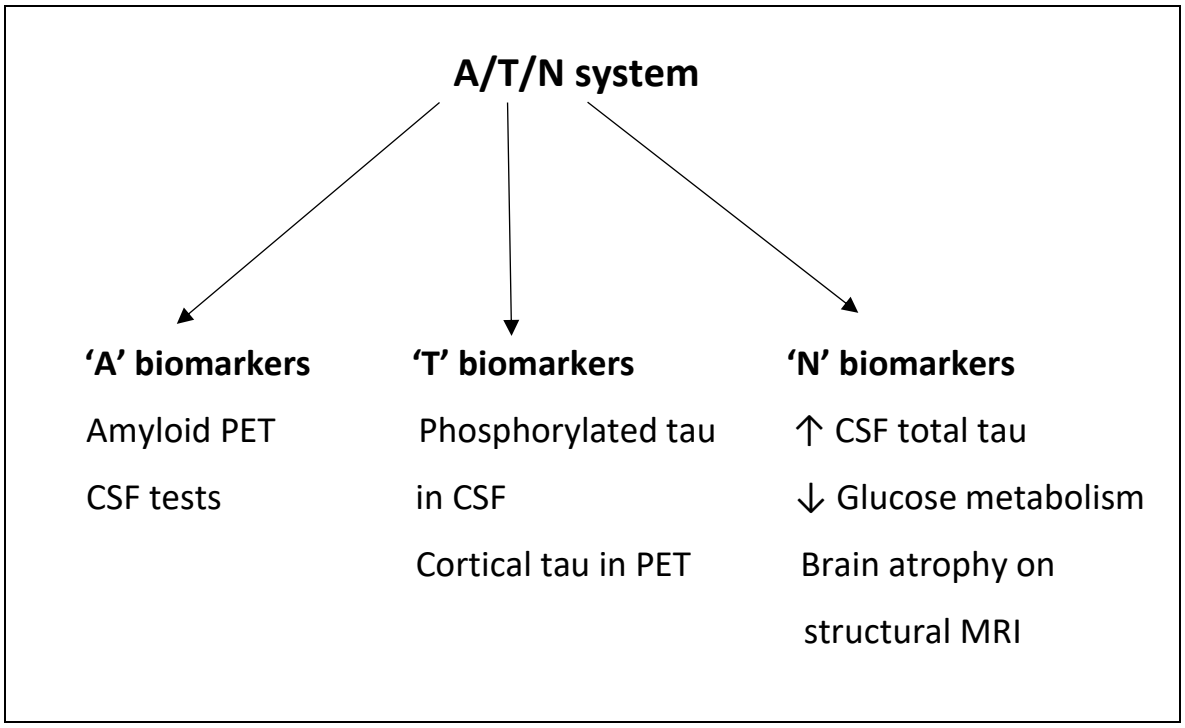

Figure 1: The A/T/N system for the classification of AD biomarkers

Biomarkers are widely heralded as the future of dementia diagnosis. But their diagnostic potential is somewhat overstated. Biomarkers fail to reliably distinguish one form of dementia from other forms of dementia. For example, tau pathology is a histopathological feature of several neurodegenerative diseases including Alzheimer's disease and progressive supranuclear palsy. Also, there are many patients with normal cognition who have amyloid deposits,[4] and many patients with Alzheimer's disease who have very few amyloid deposits (Morris *et al.*, 2010). That biomarkers are not a 'silver bullet' in dementia diagnosis was amply demonstrated in a study by Beach *et al.* (2012) of patients from more than 30 Alzheimer's Disease Centers located throughout the United States. Autopsy was used to confirm a clinical diagnosis of Alzheimer's disease in 919 patients who met the study's selection criteria. Not only did 16.7% of patients with a clinical diagnosis of probable Alzheimer's disease not meet neuropathological criteria on autopsy, but 39.4% of patients with no clinical diagnosis of Alzheimer's disease satisfied the histopathological threshold for Alzheimer's disease. Clearly, there is more to the clinical diagnosis of Alzheimer's disease than biomarkers alone can contribute.

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There is also little evidence that the use of experts results in substantially improved diagnostic accuracy. Sabbagh *et al.* (2017) reported accuracy of 77% for a diagnosis of Alzheimer's disease, even among experts. They added: "Because of limited diagnostic capabilities, physicians do not feel comfortable or skilled in rendering a clinical diagnosis of AD" (p. S83). The physicians (Dutch general practitioners) in the study conducted by Van Hout *et al.* (2000) expressed diagnostic confidence in 59% of all cases they assessed. However, even this relatively low figure may have been an over-estimation of their diagnostic capabilities when only 53% of their diagnoses of dementia type were subsequently confirmed by a multidisciplinary team in an outpatient memory clinic. The diagnostic situation is more difficult still for non-Alzheimer dementias. Clinical diagnostic accuracy for dementia with Lewy bodies is as low as 50% in some studies (Hohl *et al.*, 2000). And while the sensitivity of diagnostic criteria for the behavioural variant of frontotemporal dementia (FTD) has been found to be relatively high at 85% – sensitivity is the ability of criteria to identify those patients with frontotemporal dementia – the specificity (the ability of criteria to identify those without FTD) is low at just 27% (Vijverberg *et al.*, 2016). In view of these figures, it is hardly surprising that there is growing interest in the contribution that *behavioural* markers can make to dementia diagnosis. This is where I believe pragmatic language criteria could have a significant diagnostic role to play.

4. Pragmatics as a behavioural marker of dementia

There are, I believe, strong grounds for my optimism that pragmatics can serve a role in the diagnosis of dementia. Unlike rule-based aspects of language, pragmatics is uniquely sensitive to, and intertwined with, a range of cognitive functions. These functions include mental state attribution or theory of mind, without which no hearer could attribute a communicative intention to the mind of a speaker (Cummings, 2015). Theory of mind and other aspects of social cognition deteriorate insidiously in neurodegenerative disease (Cummings, 2013, 2014a). It is the disintegration of these skills, even in adults with normal cognitive aging, that explains the insensitivity to other people's feelings and borderline impolite remarks that elderly people are often credited with exhibiting. It is also the disintegration of these skills that explains the comments of spouses of people with neurodegenerative disease that their partners seem to miss the implications of remarks in conversation or fail to detect when a speaker is being ironic or telling a joke, often long before the appearance of other symptoms. As these high-level pragmatic skills begin to 'pull back', phonology, syntax, and semantics remain relatively unaffected. This can give the speaker the appearance of possessing greater communicative competence than is, in fact, the case.

Aside from theory of mind, executive functions such as planning, inhibition and cognitive flexibility are early to deteriorate in neurodegenerative disease (Cummings, 2009, 2014b). Clinical impairment of these skills is evident at the stage of mild cognitive impairment and often before the emergence of motor and other symptoms (Cummings, 2017, 2020). Executive functions are interwoven with pragmatic language skills in conversation and narrative discourse. These cognitive abilities are necessary to plan the events in a story, to inhibit inappropriate responses in conversation and to adapt our utterances to different hearers and contexts. When executive functions begin to deteriorate in adults with neurodegenerative disorders, it is often high-level conversation skills and performance in

narrative, expository and procedural discourse that serve as an early indicator of executive dysfunction (Cummings, 2019a, 2019b). It is executive function deficits that lead adults with neurodegenerative disorders to repeat information in a conversation and to relate events in a story in the wrong order so that temporal and causal relations are misrepresented. Moreover, these discourse anomalies occur even as speakers can produce well-formed utterances and use words in a meaningful way. In section 6, we will examine data from English-speaking adults with neurodegenerative disorders that confirm some of these conversational and discourse difficulties.

There is a further ground for my optimism that pragmatics can play a role in the diagnosis of neurodegenerative dementias. There is a clear precedent for the view that pragmatics can contribute to medical diagnosis (Cummings, 2012). For many years, medical and health professionals have been using pragmatic language criteria, albeit unknowingly, to diagnose a range of neuropsychiatric and neurobehavioural disorders. The diagnosis of schizophrenia is based on positive and negative symptoms contained in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013). Positive symptoms are the presence of abnormal beliefs such as delusions (the holding of false and bizarre beliefs) and hallucinations (the perception of things that do not exist). A third positive symptom is disorganized speech also known as formal thought disorder. The language of a speaker with formal thought disorder is tangential, illogical, disorganized, and incoherent (Black and Andreasen, 2011). Gricean maxims of quality, quantity, relation and manner frame many mutual expectations between speakers and hearers in conversation but have little traction in the language of an adult with formal thought disorder. This is a point well-recognised by psychiatrists who, as part of their diagnostic training, must become skilled in assessing the nature and extent of these pragmatic anomalies.

But we are not done. Psychiatrists must also identify negative symptoms in patients with schizophrenia. These symptoms are the absence of normal behaviours such as avolition (a lack of motivation) and diminished emotional expression or flattened affect. One negative symptom of interest for our current purposes is alogia or poverty of speech. Schizophrenic adults with poverty of speech produce under-informative utterances, typically a single word like ‘possibly’ and ‘maybe’, even when the conversational context requires a more extended response (Clegg *et al.*, 2007). It is a striking linguistic feature which psychiatrists must become skilled in identifying and distinguishing from co-morbid conditions such as depression that can also reduce a speaker’s verbal output (Cummings, 2014c). Psychiatrists who are making these linguistic judgements are doing more than merely describing pragmatic aspects of language. Rather, they are using pragmatic criteria to make a diagnosis of a significant psychopathology. The medical and linguistic judgements that guide the diagnostic process in psychiatry are shaped to a significant extent by knowledge of the pragmatics of language. It emerges that psychiatrists are applied pragmatists *par excellence*.

Pragmatic language features also play an important role in the diagnosis of attention deficit hyperactivity disorder (ADHD) in children and adults. ADHD is a behavioural disorder which has its onset in the developmental period. It has significant implications for the academic, social and occupational functioning of the individuals whom it affects. In DSM-5, a diagnosis of ADHD is based on six or more symptoms of inattention and six or more symptoms of hyperactivity-impulsivity. So central are pragmatic anomalies to these symptoms that

specific conversational difficulties are listed in DSM-5 to guide clinicians in their identification of inattentive and hyperactive-impulsive behaviours. The child with inattention "often does not seem to listen when spoken to directly". Meanwhile, the child with hyperactivity-impulsivity "often talks excessively", "often interrupts or intrudes on others", and "often blurts out an answer before a question has been completed". The explicit inclusion of these conversational behaviours in DSM-5 demonstrates the central significance afforded to pragmatic aspects of language in the diagnosis of this common mental disorder. Clearly, pragmatic language behaviours are already integral to the diagnostic criteria of DSM-5, a diagnostic system that is used by medical and health professionals worldwide. The argument of this paper is that we should not be afraid to add a few more pragmatic criteria to facilitate the diagnosis of neurodegenerative dementias.

5. A study of language in neurodegenerative disorders

The search for pragmatic aspects of language that can serve as early behavioural markers of cognitive impairment in neurodegenerative disease began in the UK in August 2018. A small-scale, qualitative study was undertaken of 27 individuals with neurodegenerative disorders other than Alzheimer's disease. Participants and spouses (or other relatives) were visited in their homes by the author who administered all tasks. The entire test session was audio-recorded. The aim of the study was to obtain a comprehensive language profile of all 27 participants, including detailed background information relating to family history, diagnosis, symptoms and medication. The study sought to replicate the approach taken by Dr Leo Kanner who produced the first clinical description of 'infantile autism' in 1943 (Kanner, 1943). Kanner's subjects were 11 children with autism aged 2 to 8 years. Kanner conducted a detailed case study of each child, including their behaviour and upbringing as well as the educational backgrounds of their parents. He described in considerable detail the unusual behavioural features of these children. His description of these 11 cases was the first step on the road to our present-day diagnostic criteria for autism. The details of the current investigation are presented below.

Participants: The 27 participants were 15 men and 12 women. Their average age was 60.2 years (range 35.5 years to 80.7 years). The diagnoses of the 27 participants were: Parkinson's disease (11 participants); Huntington's disease (7); progressive supranuclear palsy (3); multiple sclerosis (3); motor neurone disease (1); corticobasal degeneration (1); and alcohol-related brain damage (suspected Wernicke's encephalopathy) (1). Diagnoses were confirmed by medical specialists. None of the participants had a previous neurological disorder (e.g. stroke) or an earlier developmental or acquired language disorder. Several participants had motor speech disorder, although their dysarthria was mostly mild in nature and speech was sufficiently intelligible to be recorded. Participants lived at home and received varying degrees of support from spouses and other family members. Four participants were employed and 23 were either retired or unemployed. Only one participant, the individual with alcohol-related brain damage, was tested for and was found to have cognitive impairment.

Test protocol: There were a total of 12 language tasks used in the investigation. Tasks examined the following: spontaneous conversation about familiar topics; immediate and delayed story recall; picture description; sentence generation; phonemic (letter) fluency; semantic (category) fluency; narrative production; procedural discourse production; and

confrontation naming. These tasks are described further below. Test materials can be found in Cummings (2020).

• Spontaneous conversation – Each test session began with spontaneous conversation on topics such as employment, holidays, interests and hobbies, favourite television programmes, and the impact of having a neurodegenerative disorder on daily life. Some of these topics have neutral or positive emotional content (e.g. holidays), while others have negative emotional content (e.g. illness).
• Story recall – A 100-word story was read aloud to participants without any visual support in the form of pictures. The vocabulary in the story was appropriate to the cultural and educational background of the participants. The scenario portrayed in the story was also familiar to participants. Participants were asked to recall the story immediately and then again at the end of the test session in order to examine the effect of memory on recall.
• Picture description – Two picture description tasks were used. The Cookie Theft picture from the Boston Diagnostic Aphasia Examination (Goodglass *et al.*, 2001) was used to elicit extended discourse. The picture is a single, black-and-white line drawing depicting a domestic scene. Participants were also asked to tell a story based on a sequentially organized, six-frame, black-and-white cartoon called The Flowerpot Incident.
• Sentence generation – Participants were presented with single words, two words, and three words and were required to put these words into brief sentences. They were informed that the words could be used in any order in the sentences they produced.
• Verbal fluency tasks – Two verbal fluency tasks were presented to participants. They were a category (semantic) fluency task and a letter (phonemic) fluency task. During the semantic fluency task, participants were asked to produce the names of as many animals as possible within a timed period of 60 seconds. During the phonemic fluency task, participants were asked to name as many words as possible that begin with the letter 'F' in 60 seconds.
• Narrative production – Participants were asked to relate a well-known fictional narrative, the Cinderella story. They were first asked to refresh their memory of the story by viewing pictures in a wordless picture book. The book was closed during the telling of the story.
• Procedural discourse production – Participants were asked to explain how to undertake two simple, everyday tasks: preparing a ham and cheese sandwich and writing a letter to someone.
• Confrontation naming – Participants were shown black-and-white line drawings of objects and animals and were asked to name them. Target words varied in difficulty based on their frequency of usage. An equal number of low-, medium-, and high-difficulty words were used. All pictures were within the cultural experience of participants. If cues were required, their presence and type (phonemic and/or semantic) were recorded.

Transcription, scoring and analysis: All audio-recordings were made using two digital voice recorders (Sony ICD-UX560F). Recordings were orthographically transcribed by an experienced transcriber who is familiar with the dialect and accent of Northern Irish English, the regional variety spoken by all participants in the study. The author scored and analysed all transcribed recordings. Quantitative results were obtained for the two verbal fluency tasks and for confrontation naming. Verbal fluency scores were compared with published normative data. All other tasks were analysed qualitatively.

6. Preliminary data and findings

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The data reported in this section is not intended to be exhaustive or comprehensive. Rather, it is presented with a view to illustrating the type of pragmatic language difficulties that arise in neurodegenerative disease and that may be considered significant as early behavioural markers of cognitive impairment. Limitations of space permit the examination of three aspects of pragmatics and discourse that were disrupted most often in the participants in the study. For a more in-depth analysis, the reader is referred to Cummings (2020).

Participants in this study exhibited marked impairments of the following aspects of pragmatics and discourse:

Information management – Participants often repeated, omitted and incorrectly sequenced information during the discourse production tasks in the study. When information was repeated and omitted, it reduced the informativeness of the speaker's discourse. A 42-year-old female participant with multiple sclerosis made considerable use of repetitive language during the Cinderella narrative, as the following extract illustrates:

"she (.) she had to leave by twelve o'clock, so she left twelve and (.) as she was leaving then to em twelve (.) twelve o'clock was the bells were ringing for twelve o'clock …"

Discourse that misrepresented the order of events in a story or the sequence in which stages in a task have to be performed contravened causal and temporal relations that confer coherence on discourse. A 57-year-old male participant with Huntington's disease had considerable difficulty ordering events during the Cinderella narrative. The narrative begins with Cinderella meeting a prince when this occurs quite late in the story. The fairy godmother is mentioned *after* Cinderella danced with the prince, when the fairy godmother's appearance in the story precedes Cinderella's attendance at the ball. Along with some incorrect information (e.g. the ugly sisters did something to the prince), this narrative is incoherent and difficult to follow:

"Cinderella met a prince eh, and eh, then she went away and danced him maybe or done whatever they done and then eh, the ugly sisters done something till him, and then the next thing was that, he got, she got locked up in castle no the, the fairy godmother appeared but she got locked up in the castle, and the fairy godmother sent somebody away with the key to get her out, and she married her princess or the, the king or whoever he is aye, so"

The failure to represent causal and temporal relations adequately within discourse suggests executive function deficits of the type described in section 4. This expressive deficit has a corresponding receptive deficit when participants were also observed to draw incorrect inferences. A 50-year-old female participant with multiple sclerosis drew two erroneous inferences during the Flowerpot Incident discourse production task. These inferences were that the gentleman in the scene entered his own home after he was struck on the head by a falling flowerpot, and that the woman he addressed was his wife.

Reference – The use of pronouns and other expressions to refer to people and objects was often disrupted in the language of participants in the study. Errors included use of pronouns that had an ambiguous referent. For example, the pronoun *she* failed to distinguish between the woman and the girl in the Cookie Theft picture or between Cinderella and her

stepmother in the Cinderella narrative. A participant in the study might first use a pronoun to refer to one character and then shift reference to another character without signalling a change of referent for the hearer. The result was that the narratives of participants were often difficult to follow, with the investigator having to undertake considerable interpretive work. Occasionally, a pronoun was introduced into discourse without any potential referent identifiable by the hearer. This occurred during conversation with a 69-year-old man (PAR) with Parkinson's disease. When asked about his former employment, the speaker introduced the pronoun *he* to refer to his employer without first establishing a referent. The investigator (INV) was forced to clarify the referent before the conversation could proceed:

INV: Can you tell me what type of work you were doing before you retired?
PAR: Van driving
INV: Okay, would you say a bit more about that?
PAR: It is it was carrying parcels eh, h, h, h into hospitals mostly
INV: Right
PAR: He'd a contract with the hospitals
INV: Right who, who had a contract?
PAR: Trimprint
INV: Trimprint

Impaired use of pronominal reference suggested that speakers were insufficiently sensitive to the knowledge of their hearers. This inability to read the mental states of hearers led speakers to assume that hearers would know the intended referents of the pronouns they used in discourse. Occasionally, this assumption was warranted – hearers were able to use aspects of context to establish the intended referent of a speaker's pronoun. But there were also occasions when context could not facilitate interpretation and no referent for a pronoun could be identified. While poor sequencing of events in a narrative could be attributed to executive dysfunction, impaired pronominal reference suggests a failure of theory of mind on the part of the participants in the study.

Intentions and speech acts – Participants in the study displayed several pragmatic behaviours that suggested they had failed to attribute correct intentions to the minds of story characters. As a result, participants misunderstood the speech acts that characters were performing. In the Flowerpot Incident task, the gentleman enters the building with the intention of remonstrating with the woman who owns the apartment from which the flowerpot fell. A 42-year-old male participant with Huntington's disease correctly established that the elderly woman who owned the apartment *apologised* to the man for the injury he had sustained. In return, the gentleman *expressed gratitude* by kissing the woman on the hand. However, this speech act was misinterpreted as the expression of a romantic intention on the part of the gentleman towards the woman:

"she apologised to the man for the bump on his head (.) and he kissed her hand and asked her for a date and the dog ran down the stairs with a bone as happy as Larry"

This mistaken attribution of an intention to the gentleman in the depicted scenario suggests some difficulty with theory of mind. Another error by this same participant confirms this suggestion. In the Cookie Theft picture description task, a woman is drying dishes at the sink.

She is daydreaming and, as a result, is unaware that water from the sink is overflowing onto the floor. This same male participant with Huntington's disease describes this woman's mental state in the following terms: "when she's drying the dishes seems to be looking out the window with a cross face on her". The mistaken attribution of anger to the woman in the scene suggests an impairment of affective theory of mind. It is also consistent with the finding of a specific deficit in negative emotion recognition in Huntington's disease (Bora *et al.*, 2016).

This speaker with Huntington's disease attributed mental states, albeit incorrectly, to the minds of story characters. Other participants in the study simply neglected the intentions and speech acts of characters in the scenarios they were asked to consider. The male character in the Flowerpot Incident is *angry* that he has been hit on the head by a falling flowerpot and he wants to *remonstrate* with the owner of the apartment from which the pot fell. For her part, the female character in the story is *unhappy* about the man's injury and wants to *make amends* by giving his dog a bone. The mental states and speech acts shown in italics are integral to an understanding of the actions of the two protagonists in this story. Yet, a 50-year-old female participant with multiple sclerosis neglected all mention of them in the narrative she constructed during this discourse production task:

"there's a gentleman right okay so this is a gentleman I'm going to call him Tom so Tom is out with his little dog he's got a walking stick (.) am (.) and a plant seems to have fallen on his head (.) em (.) whether it's outside his home em (4:99) his dog seems to be quite alarmed at this and, and sort of has sort of stepped back (.) em but Tom's going to go into the house must be his home and with the dog um following and he slams the door behind him (.) his wife's there and she welcomes this little puppy or his little dog in to em (2:10) and the dog's got a bone and he runs away with it which the wife has given him and we're not quite sure what the wife and husband are going to do now (.)"

7. The way ahead

It is clear from this brief examination that participants with neurodegenerative disorders can exhibit marked pragmatic and discourse deficits even as their structural language skills are relatively intact. It is argued in this paper that these deficits are potential behavioural markers of cognitive impairment in neurodegenerative disorders and that when sufficiently refined and validated, these markers can contribute to the early diagnosis of cognitive impairment in these disorders. I conclude this discussion with some initial thoughts on what these markers might look like. The empirical work that will be needed to identify them is at an early stage of development. Nevertheless, it is possible to discern key issues that must be addressed. These issues concern whether 'pragmatic' behavioural markers are likely to operate singly or in combination and whether these markers are best characterised in quantitative or qualitative terms.

From data examined to date, it seems clear that all pragmatic anomalies in the language of adults with neurodegenerative disease have the same consequence: they reduce the informational content that a speaker can convey to a hearer. If a speaker omits, repeats or incorrectly sequences events in a narrative, little information is conveyed to the hearer who must construct a rather scant mental representation of the narrative in consequence. The same is true of a speaker who fails to grasp the intentions of a story protagonist or partner in conversation and proceeds to misinterpret their speech acts. Once again, important

information is either missing or inaccurately captured, and a sub-optimal mental representation – one that is incomplete and/or contains false information – is the result.

Reduced informational content is the central communicative deficit of speakers with cognitive impairment in neurodegenerative disease. But what contributes to this reduced content varies across different neurodegenerative dementias. For speakers with one of the language variants of frontotemporal dementia, impairments of syntax and semantics might be the most significant factor contributing to reduced informational content. We saw in section 6 that structural language skills are relatively intact in speakers with multiple sclerosis, Parkinson's disease and Huntington's disease. Deficits in areas like reference, information management, intentions and speech acts are more likely to compromise the informational content that a speaker with one of these conditions can convey. To characterise the factors that contribute to reduced informational content in these speakers, our behavioural markers must reflect the pragmatic and discourse deficits of these disorders.

The model that I believe will ultimately best capture behavioural markers of different neurodegenerative diseases is one in which pragmatic language features come together to form a constellation of symptoms. The core deficit at the centre of the constellation is reduced informational content (see Figure 2 for the constellation in primary progressive aphasia, the language variant of frontotemporal dementia). The features that contribute to this reduction of informational content are arranged in concentric circles that move outwards from the centre. The pragmatic behaviours that contribute most to reduced content (e.g. poor use of reference) are arranged in the concentric circle immediately next to the centre of the constellation. Other features with a less significant contribution to make to reduced content are arranged in concentric circles towards the periphery of the constellation (e.g. emotion recognition problems in Huntington's disease). The respective significance of these different features can be quantified in terms of the percentage of variance they contribute to reduced informational content. The role of each pragmatic feature in the constellation can thus be precisely quantified, with those contributing the greatest variance occupying a concentric circle closest to the centre of the constellation.

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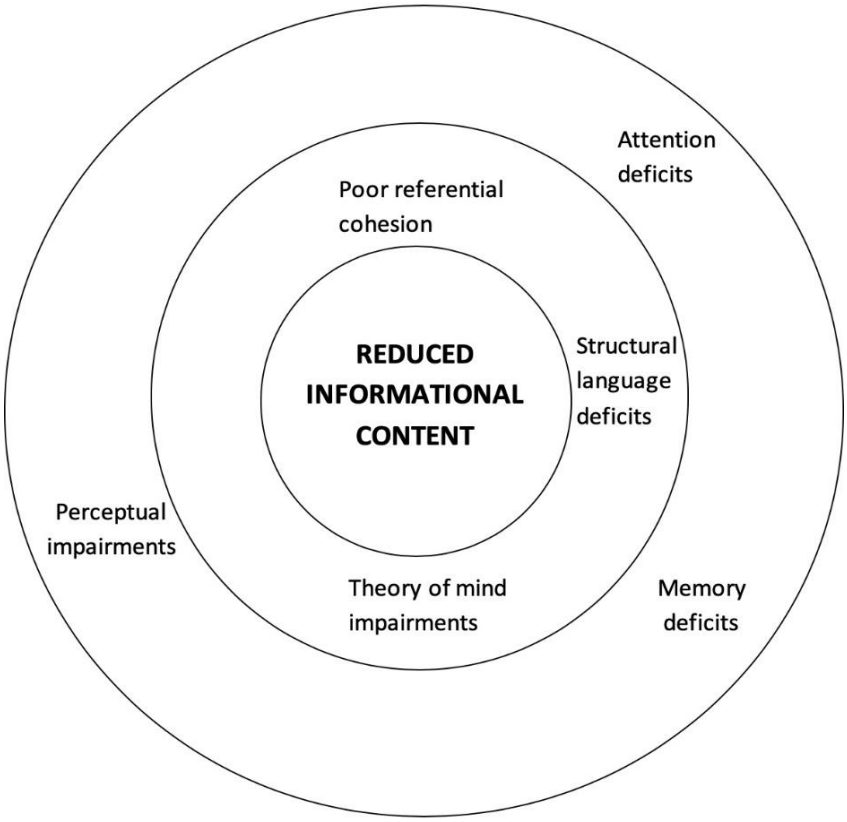

Figure 2: Constellation of behavioural markers in primary progressive aphasia

The empirical validation of this model of behavioural markers is a long-term project that will require the same investigative effort that has gone into understanding the behavioural phenotype of conditions like schizophrenia and autism spectrum disorder. It is, however, a feasible project that will deliver robust criteria that can contribute to an early diagnosis of cognitive impairment in the neurodegenerative dementias. Careful observation and detailed description of pragmatic aspects of language will deliver the criteria we need if we just look closely enough at conversation and other forms of discourse.

8. Summary

It has been argued in this paper that the pragmatics of language is a potentially rich source of behavioural markers that can be used in the early diagnosis of cognitive impairment in the neurodegenerative dementias. Dementia poses a global public health challenge, with even well-funded healthcare systems inadequately equipped to address it. This burgeoning health and social care problem could be substantially alleviated if earlier diagnosis were possible. It was argued, however, that our current diagnostic methods lead to high rates of misdiagnosis and that many affected individuals are already in a state of advanced disease progression before a diagnosis is made. Behavioural markers rooted in the pragmatics of language, it was contended, have the potential to improve diagnostic accuracy and to contribute to an earlier diagnosis of affected individuals. The paper examined some pragmatic aspects of language that could serve as behavioural markers of cognitive impairment in the neurodegenerative dementias and considered a model that may be used to characterise these markers.

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† This paper is based on a plenary lecture of the same title that was delivered at the 16th International Pragmatics Association Conference held at The Hong Kong Polytechnic University between 9-14 June 2019.

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NOTES

1. The World Health Organization (2019b) defines *healthy life expectancy* as "a form of health expectancy that applies disability weights to health states to compute the equivalent number of years of good health that a newborn can expect."

2. The National Institute on Aging (2018) remarks that "[u]se of biomarkers in clinical settings, such as a doctor's office, is limited at present. Some biomarkers may be used to identify or rule out causes of symptoms for some people. Researchers are studying many types of biomarkers that may one day be used more widely in doctors' offices and other clinical settings."

3. Remarks from spouses confirm the long periods of time that some diagnoses have taken. The wife of a 61-year-old man with Parkinson's disease reported that it took over 5 years to receive a diagnosis of his condition. A 72-year-old man with progressive supranuclear palsy (PSP) was diagnosed with Parkinson's disease in 2006. A diagnosis of PSP was only made 10 years later in 2016. His wife reported that she and other family members noticed subtle changes as far back as 1995 (e.g. altered posture, length of stride, loss of facial expression and withdrawal from social activities). The wife of a 66-year-old man with corticobasal degeneration reported that it took over 3 years to get a diagnosis of her husband's condition.

4. At least 20% and as much as a third of healthy older adults show significant amyloid deposition (Rodrigue *et al.*, 2009).

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