The Role of Connected Speech in distinguishing types of Apraxia of Speech following Brain Damage

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Abstract

Apraxia of speech (AOS) is a common condition resulting from brain damage causing impairments to speech planning and programming that is distinct from aphasia and dysarthria. Following a stroke, AOS is most often seen as a part of nonfluent Broca's aphasia. Recovery and dissolution of speech in stroke induced apraxia of speech (sAOS) and progressive AOS (pAOS) appear on a symptomatic level to be reversals of each other, but the existing evidence shows that sAOS is distinct from pAOS. This suggests different causes that have relevance for management of pAOS.

In this article, we examine the similarities and differences between sAOS and pAOS and conclude that while the two conditions present similarly, certain features differ significantly, which may suggest different mechanisms underlying their manifestation. The role of connected speech as opposed to single word utterances is noted.

Keywords: apraxia of speech; stroke induced apraxia of speech; progressive apraxia of speech; language recovery; language dissolution.

1. Introduction

Apraxia is classically defined as an impairment in the ability to perform purposeful, voluntary movements in the absence of neuromuscular disorder. Traditionally it is characterised as an impairment in the planning, the programming, the initiation, and the co-ordination of an action or action sequence in the absence of neuromuscular impairment (for review see Code, 1998; Utianski & Josephs, 2023). Acquired apraxia is usually caused by damage to the left hemisphere (in right-handers) through stroke or a progressive neural degeneration. Apraxia can affect most aspects of human action control and a variety of forms have been described over the years. The major varieties are ideational, ideomotor, constructional, and buccofacial or oral (Liepmann, 1900), as well as apraxia of speech (AOS), which is of central concern in this article. Liepmann (1900) provided the first detailed descriptions, but the term 'apraxia' appears to have originated with Steinthal (1871). A review is provided by Goldenberg (2013).

Apraxia of speech (AOS) is a common condition, usually resulting from left brain damage, causing impairments to speech planning and programming. AOS is distinct from the articulation and voice problems of dysarthria, and from the language processing impairments of aphasia. Aphasia is the term most aphasiologists use to describe impairments in the expression and comprehension of language in any modality, whether through speech, writing or linguistic signing, and it is caused by some acquired form of damage to the brain. People with AOS are said to know what they want to say but are unable to say it. More formally, this describes further classic features in many definitions of AOS: problems with the initiation of articulatory action and articulatory groping or searching for articulatory positions.

Darley, Aronson and Brown (1975; p. 255) defined AOS as 'an articulatory disorder resulting from impairment due to brain damage of the capacity to program the positioning of speech musculature for the volitional production of phonemes and the sequencing of muscle movements'. Thus, 'programming the positioning of speech musculature for speech 'phones' (the 'produced' phonemic segments) is the core feature according to their definition.

Later, Rosenbek, Kent and LaPointe (1984) catalogue a range of main characteristics of AOS, mainly associated with articulatory complexity. Speech errors increase with the complexity of the motor task: consonants are harder to produce than vowels and single consonants are easier than clusters of consonants. Errors increase with word length and

occur more on imitation than in spontaneous speech. In addition, errors occur less often on automatic than on propositional speech. Articulatory complexity correlates with the frequency of occurrence of consonants and errors are more frequent on low than on higher frequency consonants.

Modern phonetic theory views the production of speech as the coordination of a number of components (often termed 'gestures', see for example Ball, Code, Tree, Dawe & Kay, 2004), each component utilizing different combinations of the organs of speech. For example, the glottal gesture (vocal fold activity in the larynx) needs to be coordinated with articulator contact to produce the required degree of voicing (vibrating vocal folds) and the timing of the onset of voicing (called 'voice onset time', or VOT) for the production of oral stop consonants for the specific language. The velic gesture (movement of the soft palate to enable or to block airflow through the nasal cavity) needs to be coordinated with the various articulatory gestures to distinguish oral, nasal, and nasalized sounds (e.g. oral stop 'b' [b] from nasal stop 'm' [m], from the nasalized vowel [õ], (as in French 'non'). The tongue body gesture also needs to be coordinated with the lip gesture to produce doubly articulated sounds such as English 'w' [w]. Gesture coordination requires not only accurate movement of speech articulators, but also accurate timing of these movements, and it is this complex coordinator activity that can be affected in AOS.

The neuroanatomy of AOS has been examined in a number of studies. Dronkers (1996) conducted a lesion-overlap study comparing the CT or MRI scans of 25 AOS speakers at least 12 months post-onset of stroke, with scans of 19 stroke survivors without AOS. AOS speakers had lesions that included the precentral gyrus of the anterior insula of the left hemisphere, but the precentral insula was spared in all 19 speakers without AOS. She concluded that the cause of AOS is a left anterior insula damage, which impairs articulatory planning. However, Hillis, Work, Barker, Jacobs, Breese and Maurer (2004) pointed out the limitations of the lesion-overlap methodology with chronic stroke survivors with large lesions. Hillis et al. examined areas of damage and dysfunction indicated by low blood flow (hypofusion) in 40 participants with insular damage and 40 without within 24 hours of stroke, to minimize the potential of neuroplasticity resulting in neural reorganisation. Using this strategy, they were able to identify the probability of the lesion causing AOS and the probability of AOS simply being associated with the lesion. They found no association between AOS and lesions of the left insula, the anterior insula or the precentral region of the insula. But they did find an association with structural

damage or low blood flow in Broca's area for those patients with AOS. Further research is obviously required to determine if the differences in location of damage, or hypofusion, are similar or different in sAOS and pAOS. Joseph, Duffy, Strand, Whitwell et al. (2006) undertook a magnetic resonance imaging (MRI) analysis using voxel-based morphometry of 17 persons with progressive aphasia or AOS (n=11). This showed that the premotor and supplemental motor areas are the main cortical regions associated with AOS, and the anterior perisylvian region was associated with non-fluent aphasia.

In this article, we present a discussion of apraxia of speech from stroke (sAOS) and from progressive neurological disease (pAOS). Our focus is on the significant differences that have been identified by a range of studies between the production of single words compared to connected speech. We review the literature and present data from longitudinal studies of an English-speaking man (CS) with a primary progressive AOS and primary nonfluent aphasia (pNFA). Fuller details on demographic variables, case history, clinical presentation and autopsy can be found in a range of studies (Code, Müller, Tree & Ball, 2006**;** Code, Tree & Dawe, 2009; Code, Tree, & Ball, 2011; Code, Ball & Tree, 2013).

Later in the article we compare speech performance in connected speech with performance on single words. A variety of speech and language tasks are usually employed in assessing acquired disorders, and with CS these included picture description and reading a passage (connected speech) as well as single word repetition, and picture naming (single words). Many of CS's speech results are derived from the *Motor Speech Examination* (Wertz, 1984) and the *Dabul Apraxia Battery* (DAB) (Dabul, 2000). Fuller details of all the assessments undertaken are in Code et al (2013).

2. Speech Planning and Programming

It is hypothesized that between the abstract phonology and actual execution of speech, there is a component to plan and program the encoded string handed down from phonology and dispatch the co-ordinated result for execution. On Levelt's (1989) influential model of word production and comprehension, planning and programming appear to be indivisible. Levelt's terminology is looser, and he does not distinguish between planning and programming, as others do. He states, 'some buffering will be required to keep the phonetic plan (i.e., the motor program) available for execution' (p.414). AOS, in a component model, arises at the level of the Phonetic Plan. At this level an utterance is either planned (speech production) or retrieved (speech perception). The

phonetic plan is the result of phonological encoding and is a representation of 'how the planned utterance should be articulated - a program for articulation' (p.12).

On van der Merwe's (1997) model, planning entails the retrieval of the core motor plan, which stipulates the voice, place and manner of phones. The specific pattern of muscle tone, resistance, and force of movement is the responsibility of motor programming. Impairments of programming result in muscle tone or reflex deficits (van der Merwe, 1997; see also van der Merwe, 2021). Invoking van der Merwe's (1997) model of AOS, McNeil et al. (1997) restrict the term AOS to impairments at the planning stage and not to speech programming. For them, impairments arising from speech programming are either dysarthric or paraphasic (McNeil, Robin & Schmidt, 1997; McNeil, Doyle & Wambaugh, 2000).

McNeil and colleagues (1997) restrict the diagnosis of *pure* AOS to four kernel characteristics. These are: lengthened segment durations, lengthened inter-segment durations, distorted movement transitions and distorted sound substitutions. The standard searching and groping behaviour, as well as speech initiation difficulties, are not features of AOS on their model. In contrast, Buckingham (1991) suggests that articulatory groping and searching implies that the individual has generated an underlying phonological form, and it is the articulatory program the individual is searching for.

Ballard et al (2016) conducted a study of 72 persons with AOS who also had aphasia, using statistical modelling techniques with the outcome measure of expert judgement on presence of AOS. Just two measures were sufficient to distinguish between participants with aphasia alone and those with AOS and aphasia. These were a measure of speech errors on words of increasing length (e.g., *thick, thicken, thickener*[1]) and a measure of relative vowel duration in three-syllable words with weak–strong stress pattern (e.g., *banana, potato*).

3. Aphasia and Apraxia of Speech

The classic forms of aphasia from stroke are relatively well known, although there is still controversy about the usefulness of classifying aphasia into types. 'Fluency' is a major division between two main patterns of language impairment: speech is usually fluent in those who have aphasia from mainly posterior left hemisphere damage, which variably results in comprehension impairments, paraphasias, and sometimes jargonaphasia. Wernicke's aphasia is the term widely used to describe this form of fluent aphasia. This contrasts with nonfluent aphasia from inferior frontal damage, resulting in

speech automatisms (recurrent utterances), agrammatism, repetition problems and AOS. Broca's aphasia is the term most often used to describe nonfluent aphasia.

Extensive study has established that language and speech can be compromised in various ways by progressive neurological disease in the absence of significant impairments to other aspects of cognition (Croot, Patterson, & Hodges, 1998; Garrard & Hodges, 1999; Mesulam, 1982; Patterson, Graham, Lambon-Ralph, & Hodges, 2006; Scholten, Kneebone, Denson, Field, & Blumbergs, 1995; Snowden, Neary, & Mann, 2002; Thompson, Ballard, Tait, Weintraub, & Mesulam, 1997). Different forms of primary progressive speech and language deterioration have been identified (e.g., Gorno-Tempini et al., 2011; Patterson et al., 2006; Snowden et al., 2002; Tree & Kay, 2008) and three broad forms of primary progressive aphasia (PPA) have been described. Semantic dementia (SD) (Garrard & Hodges, 1999; Snowden. Goulding, & Neary, 1989) arises from gradual posterior parieto-temporal damage with fluent speech and significant semantic processing disturbance. Progressive nonfluent aphasia (pNFA) (Gorno-Tempini et al., 2011; Graham, Patterson, & Hodges, 2004; Patterson et al., 2006; Tree, Kay, & Perfect, 2005), is caused by progressive frontotemporal and frontobasal damage. The third form is logopenic progressive aphasia (LPA) (Gorno-Tempini et al., 2011), with the two necessary core symptoms of impaired single-word retrieval in spontaneous speech and naming, and impaired repetition of sentences and phrases. Other possible symptoms include slow speech, simplified grammar, phonemic paraphasias in spontaneous speech, but good semantic abilities. With incremental neural deterioration, symptoms emerge that change patterns of impairment across progressive aphasias, and LPA may be a variant of pNFA. Knibb, Woollams, Hodges, and Patterson (2009) suggest that pNFA includes varieties like LPA and pAOS which may be seen as points on a continuum within progressive nonfluent aphasia (Knibb et al., 2009). This 'change of type' as patterns of symptomschange with deterioration, is an apparent reversal of the same process seen with recovery from stroke aphasia (Kertesz, 1979).

Pure AOS is rare, whether from stroke or progressive deterioration. It occurs most often combined with aphasia or other cognitive impairment. It is AOS that accounts for much of the nonfluency of Broca's aphasia, thus Broca's aphasia (and pNFA) includes AOS (Berndt & Caramazza, 1980; Goodglass & Kaplan, 1983; Marquardt & Sussman, 1984; Mohr *et al.,* 1978). Researchers in the second half of the 20th century suggested that nonfluent Broca's aphasia should be reformulated as a combination of aphasia and AOS (Bernt & Caramazza, 1980; Mohr *et al.,* 1978). *Leborgne*, Broca's famous case, had

significant AOS and relatively little aphasia, according to Broca (1861), and Broca called the condition he first described in *Leborgne, aphemia*, a term still used in some professional and national groups.

In their seminal computerised tomography study involving a large survey of cases, Mohr et al. (1978) concluded that Broca's aphasia did not result from a lesion limited to the classical Broca's area, but rather resulted from a larger lesion involving the area of supply of the upper division of the left middle-cerebral artery, which produces a *global* aphasia affecting all or most modalities. The damage includes the operculum, the 3rd frontal gyrus, the anterior parietal region, the insula, and both sides of the central Rolandic fissure, extending deep into the underlying white matter. This extensive damage produces what Mohr et al. (1978) called 'Big Broca's aphasia' or *the operculum syndrome,* with a severe and persisting AOS with either mutism or a nonlexical speech automatism (e.g., *ti, ti; tan, tan)* with the later emergence of agrammatism and severe reading and writing problems, if there is any recovery.

A variety of terms have been used to describe this non-dysarthric and non-aphasic progressive speech production impairment: These include pure progressive aphemia (Cohen et al., 1993), slowly progressive anarthria (Broussolle et al., 1996), primary progressive anarthria (Silveri, Cappa, & Salvigni, 2003), and (primary) progressive apraxia of speech (Duffy, 2006 ; Josephs, Duffy, Strand, Machulda, et al., 2012 ; Ricci et al., 2008). It occurs either in combination with some progressive language or or are reported as relatively pure speech production impairments, but nonetheless are not primarily dysarthric nor aphasic conditions (Broussolle et al.,1996 ; Chapman, Rosenberg, Weiner, & Shobe, 1997; Cohen, Benoit, Van Eeckhout, Ducarne & Brunet, 1993; Duffy, 2006 ; Hart, Beach, & Taylor, 1997; Josephs et al., 2012; Ricci et al., 2008; Silveri et al., 2003; Tyrrell, Kartsounis, Frackowiak, Findley, & Rossor, 1991). Josephs et al (2021) provide an in-depth examination of progressive AOS.

4. Illustrative case

C.S. was a 62-year-old (DOB: 23/08/40), right-handed man at the time of testing. He was Head of Chemistry in a well-known British public (i.e. 'private') school for 21 years before early retirement because of increasing speech problems, which were first noticed by his family in 1992. At this time too he experienced reduced sensation in parts of his upper and lower limbs and his family noted on a number of occasions some clouding of consciousness and verbal nonfluency on waking from a nap.

By August, 2002, a wide range of longitudinal testing had been completed, including assessment of intelligence, perception, memory, language and action/gesture. Longitudinal performance over the 4 year period 2001-2004 is described in Code et al. (2006). The investigations of speech production reported here took place over a 12-month period between December, 2001 to November, 2002 that we organised into a Time 1 (T1) (December 2001 – March 2002) and Time 2 (T2), (April, 2002 - November, 2002.) It was not possible to repeat all tests at Time 2, due to rapid deterioration in C.S.'s speech, and in his general health. C.S. had very severely impaired speech by the end of the current investigation and meaningful testing of speech was no longer possible.

Problems with naming objects are a classic feature of both progressive language and speech conditions as well as following stroke, but problems naming objects can have a variety of possible causes, which can arise at semantic, lexical, phonological and/or phonetic levels. However, deciding at which level a naming error arises in any individual is not always straight forward. A range of phonetic and psycholinguistic variables are found to impact on object naming (Morrison, Chappell & Ellis, 1997). To test the hypothesis that CS's articulatory errors in naming were associated with lexical access, we analysed his errors in confrontation naming of 210 words which varied in word frequency, imageability, age of acquisition (AoA), phonemic length and syllabic length, as well as examining omissions, additions and substitutions of segments, response delays and overall errors (Code, Tree & Ball, 2011). No significant naming errors were found that were due to lexical access (see Ball et al 2013 for description of assessments of naming). However, CS produced additions, omissions, and substitutions of speech segments during his responses. Regression analysis provided the only significant predictor of articulation errors, which was the phonemic length of the word, with none of the other lexical variables influencing error production. Additionally, the only error type predicted was omissions. CS did not have a problem accessing words from his lexicon, but one articulating the words.

But why did word length predict sound omissions, but not sound substitutions and additions? It is easier for an impaired speech mechanism to omit a phone in a word, particularly a longer word, than to struggle to produce the whole word, but adding a phone should be harder for speaker with apraxia. CS tended to add sounds to words in naming, reading and repetition tasks, and schwa-like sound additions accounted for many. Our interpretation of the intrusive schwas agreed with Rosenbek *et al.* (1984), who

pointed out that such intrusions should not be considered 'phonemes': schwa-like intrusions for CS were compensatory and helped him produce speech. Research has shown that additions (e.g., Rosenbek, et al, 1984) and substitutions (e.g., Itoh & Sasanuma, 1984) are features of AOS. The classification of errors into substitutions is particularly problematic in AOS research (e.g., Itoh & Sasanuma, 1984), as a sound substitution can often be interpreted as an error arising from an impaired phonology, rather than an error arising from phonetic planning and/or programming (Code et al., 2011)

4.1 Recovery and Dissolution

It is a universal principle of neuropsychology that most individuals will make some recovery from aphasia, and many will make a significant recovery, following non-progressive brain damage recovery (e.g., Blomert, 1998; Code, 2001). For instance, Kertesz (1979) showed that 21% of 93 aphasic participants tested at 1-month post-onset (MPO) made full recoveries by 12 MPO. Patterns of recovery can be predictable, with speech and language profiles changing in certain ways.

Stroke typically occurs following a 'horizontal' lesion affecting large areas of neighbouring brain impairing the blood supply, whereas evidence suggests that pAOS develops 'vertically', transmitted along neuronal pathways rather than by anatomical proximity. Progressive damage may reveal a gradual unfolding of neural phylogeny that mirrors the evolution of the nervous system. While the pattern of gradual recovery of speech in sAOS is relatively well known, the longitudinal patterns of deterioration in gradually progressive pAOS is less so (see, for example, Tree et al, 2001). A potential distinction that has been examined between progressive and stroke conditions has been the relative ease of producing single words compared to connected speech for participants.

4.2 Single Word vs. Connected Speech Production

While single word production can be relatively fluent in pAOS, connected speech is slow when compared to that of neurologically healthy speakers and, significantly, to that of speakers with sAOS and pNFA. It is distorted with shorter utterances, a lack of spontaneity[2] and reduced complexity[3] (Wilson, Henry, Besbris, Ogar, Dronkers, Jarrold et al. 2010; Patterson et al., 2006; Sajjadi, Patterson, Tomek, & Nestor, 2012b; Code et al, 2013).

Connected speech production in particular can have significant diagnostic and theoretical relevance when differentiating between pAOS and sAOS. Connected speech has been compared in groups with progressive nonfluent aphasia (pNFA) (Wilson et al., 2010; Sajjadi et al., 2012b), Alzheimer' s disease, nonfluent aphasia from stroke (sNFA) (Knibb et al., 2009; Patterson et al., 2006) and semantic dementia (SD) (Wilson et al., 2010; Sajjadi, Patterson, Tomek, & Nestor, 2012a). However, there have been few detailed longitudinal studies. Wilson et al. (2010) analysed the speech of 50 participants with primary progressive aphasia, and other neurodegenerative as well as neurologically healthy control groups. A slow rate with segmental distortions, syntactic errors and reduced complexity was found in pNFA. In contrast, typical speech rate was found in participants with SA. Connected conversational speech and speech elicited by picture description in pNFA and a mixed PPA was analysed by Sajjadi et al (2012b). They showed that the connected speech of participants with pNFA during an interview is particularly useful for detecting different aspects of impaired speech.

We compared a variety of speech and language parameters longitudinally in CS. The data for CS are from Time 1 (T1) (November 2001– March 2002) and Time 2 (T2) (May 2002– November 2002). We examined phone omissions, substitutions and additions in repeated and read high frequency Consonant, Vowel Consonant (CVC) words (e.g., mat, hug, lab), single syllable words beginning with a CC(C) cluster (e.g., crew, sprang), single syllable words ending in CC clusters (/pt/, /kt/, /gd/). As can be seen in Table 1, at T1 there were few omissions, some substitutions, but predominantly additions, with no differences between word types.

Table 1. Omissions, substitutions, additions in repeated CVC words (e.g., hug, lab), single syllable words beginning with a CC(C) cluster (e.g., crew, sprang) and single syllable words ending in a CC (/pt/, /kt/, /gd/) cluster.

	CVC Words (n=27)	Initial Clusters (N=41)	Final Clusters (N=17)	Total errors
Omissions	1	2	2	5
Substitutions	3	5	3	11
Additions (% all errors)	13 (76%)	16 (69%)	12 (70%)	41 (72%)
Total errors	17	23	17	57

Similar findings were observed in a comparison of reading aloud and repetition of high frequency CVC(C) words also at T1: more additions (16, 64%) were observed than

omissions (4) and substitution (5) with no significant differences found between reading and repetition (Table 2).

Table 2. Omissions, substitutions and additions of phones in high frequency CVC(C) words in reading aloud and repetition.

	Reading 1 syllable (n=35)	Repetition 1 syllable (n=35)	Total errors
Omissions	1	3	4
Substitutions	3	2	5
Additions (% errors)	7 (63%)	9 (64%)	16 (64%)
Total errors	11	14	25

CS's responses were particularly swift in the earlier stages of his speech degeneration and speech rate was slower than normal speech at T1. An informal comparison of his utterance duration in 35 repeated single syllable words with the examiner's production of the same words showed that CS's mean utterance duration (.471ms) compared to the examiner's utterance of the same words (.577 ms). This difference was significant (t= -5.36; p<.0001).

Table 3. Speech rate and response latencies for C.S. and age, sex and education-matched controls in 35 repeated high frequency single syllable words in ms at T1.

	CS	Controls	Mean Differences
Mean duration of utterance (SD)	.478 (.122)	.623 (.104)	.143*
Response latencies (SD, Range	.868 (.442; .256–2.518)	.635 (116; .403-.928)	.233^

*t = 6.789 (df=34); p < .0001; ^t = 2.763 (df = 34); p < .009.

CS had a tendency to rapidly jump in with his repetition despite being asked to wait until the examiner completed a stimulus before repetition. At T1 we analysed this feature of his behaviour with response latencies in 35 repeated single syllable high frequency words and compared CS with five age, sex and education-matched controls, and results are presented in Table 3. CS produced significantly shorter word lengths than controls (p < .0001), but response latencies were significantly longer (p < .009). However, CS's speed of response was far more variable than controls with the shorter measures reflecting his tendency to anticipate the repetition request.

We next compared words per minute (WPM) for CS to controls and also to data produced by groups of participants with pNFA (N10) and sNFA (N10), published by Patterson et al (2006) on a range of connected speech tasks. Results are illustrated in table 4.

On a 130-word paragraph read aloud at T1 CS's WPM was less than a quarter of the control mean. His WPM deteriorated markedly by T2, being about one twelfth of the control

mean. At T1 his speech contained widespread pause fillers (e.g., er, um), word and phrase repetitions and the speech automatisms, *yes* and *yes, yes.* Pauses[4] were fewer at T2 but the pauses were particularly long (e.g. one of 12 s, another of 16 s, and another of 18 s.). Also, unlike T1 where there were examples of syllable deletions, T2 was notable for final syllable repetitions (often several times). Examples of these competing strategies can be seen in: T1: 'wished' [wɪz]; 'dresses' [dɹɛs:]; 'cracked' [kɹæk]; T2: 'dresses' [dɛzɛzɛzɛzɛʊ]□□'ancient' [eɪnʃəɹəɹəɹəɹəɹə].

Patterson et al. (2006) remarked on the notable difference between the speech rates of their sNFA and pNFA participants and CS's data compares interestingly. Patterson et al showed that sNFA produced mean 22.5 wpm, pNFA 50.1 wpm and controls 171.4 wpm. C.S.**'** s rate of utterance on this task at T1 (43 wpm) is closest to the pNFA speakers reported by Patterson et al.

Connected reading aloud is very different to the task of internally generating new language and speech during description of a scene. CS's picture description performance is the poorest of the connected speech tasks he completed, and WPM is substantially slower than the means for any of the other groups. He produced eight content words naming objects within the picture with no function words.

CS tended to slow down markedly with progress through a connected speech task. Automatic counting to 30 is the 'easiest' of the connected speech tasks requiring only the automatic production of rote learnt material and no generation of original speech. Controls took a mean of 17 secs to perform this task producing 106.9 WPM and table 4 shows that CSs WPMs were substantially fewer.

To examine this apparent slowing effect, we measured the overall time it took CS to produce 1-30 and also 1-10, 11-20, 21-30. This task also allows an examination of any syllable length effects. For 1-10 the mean and mode syllable length is one syllable when rounded, for 11-20 it is two syllables and for 21-30 it is three syllables.

It can be seen that production is fairly swift during 1-10, after which C.S. begins to slow down gradually. The controls perform more equally as they progress through the series. C.S. performs notably faster than controls on the first half of the task and considerably slower than controls on the final part.

CS took 180s on the reading passage and this was split into quarters of 45 s each and the number of words that C.S. read during each quarter was compared to controls. It took controls mean 47s to read the passage and this was quartered in the same way, and the words

produced in each 11.5 s were calculated. A similar pattern was found with a fairly swift production during the first quarter, followed by a gradual slowing. Controls performed more equally as they progress through the reading passage, and even sped up a little in the final quarter. C.S. performed considerably faster than controls on the first two quarters and considerably slower than controls on the final two quarters.

CS's notable slowing during connected speech tasks might be explained by fatigue particularly affecting his phono-articulatory apparatus and it may also indicate a syllable length effect in connected speech.

Table 4. Measures of connected speech in controls, pNFA and sNFA and CS at two testing times (pNFA & sNFA figures from Patterson, et al., 2006).

	CS time 1	CS time 2	Controls Means (SD)	pNFA Means (SD)	sNFA Means (SD)
Reading Passage					
WPM	43	14.3	175.4 (35.0)	50.1 (37.4)	22.5 (17.7)
Total time (sec)	180	544	47.0 (9.8)	182.5 (111.2)	342.0 (175.1)
Words per Quarter					
1st Quarter	39		31 (2.0)		
2nd Quarter	34		30 (1.48)		
3rd Quarter	29		33 (1.87)		
4th Quarter	27		36 (2.74)		
Picture Description					
WPM	12.7	6.3	137.4 (35.4)	27.8 (18.7)	25.2 (11)
Total words	19	11			
Total time(sec)	1.30	1.44			
Longest utterance	5 words				
Automatic counting					
WPM	26.4	14.3	106.9 (mean 17 seconds)		
Time 1-30	70	34			
Time 1-10	15	39			
Time 11-20	27	39			
Time 21-30	28	52			

4. Conclusions

In summary, there appears to be a comparative difference in performance on single word production compared to connected speech between progressive apraxia of speech and apraxia of speech from stroke. Connected speech production in particular can have significant diagnostic and theoretical relevance when differentiating between pAOS and sAOS.

How symptoms change during observed recovery following stroke or dissolution in progressive conditions in language and speech is a function of restoration (in stroke) or of compensation/adaptation in stroke and progressive disease, and of gradual deterioration in progressive disease. Individuals adapt to reduced neurocognitive resources and how that adaptation interacts with change in emerging patterns of deficit is, as yet, mostly unknown.

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Notes

[1] These three words belong to different word classes and so this could be a potential confound depending on the type of aphasia the speaker has.
[2] For example, in C.S.'s conversational speech sample, he produced no spontaneous or self-initiated speech and the conversation consisted entirely of questions (22) from the examiner and answers (22) from C.S.
[3] The features of AoS noted in previous literature include increasing errors in articulation as the complexity of the motor task increases, so that vowels are easier than consonants to produce and single consonants are easier than clusters of consonants (Rosenbek *et al.*, 1984; Duffy, 2005).
[4] By this we mean pauses that were judged not to be the normal pauses when reading aloud. All speech measures were undertaken by two trained independent judges see Code et al, 2013).