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The Recovery and Dissolution of Apraxia of Speech Following Brain Damage .

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Introduction

The recovery and dissolution, or continuing worsening, of speech in **stroke induced apraxia of speech (sAoS)** and **progressive AoS (pAoS)** when considering the symptoms of AoS, appear to be reversals of each other.

I will compare speech breakdown following stroke and progressive brain damage. The current evidence suggests that sAoS is distinct from pAoS. This suggests different causes that have relevance for management of pAoS. I plan to sketch…

- the recovery of AoS following **aphasia** from **stroke**
- And contrast it to what is known about the gradual **dissolution** of speech in **progressive AoS**.
- These two opposing patterns - **gradual recovery vs. gradual dissolution** - arise from separate forms of brain damage & are significantly different, although they share some major features.
- The neural representation of speech is not unitary, but a complex arrangement of abilities processed in distinct neural networks in separate anatomical locations. The role of accompanying impaired **cognitive functions**, have a significant impact on the emergence of apraxic symptoms following stroke and emerging in progressive AoS.

Apraxia of speech more rarely occurs on its own, but is most often accompanied by nonfluent aphasia. In fact, 'nonfluency' is defined in terms of impairments of articulatory agility and prosody (Goodglass & Kaplan, Poeck et al (in Code, etc)

In fact, *Leborgne*, the 'original' Broca's aphasic person , had significant apraxia of speech, and relatively little aphasia according to Broca (1861). Broca called the condition *aphemia.*

And a number of researchers have suggested, that nonfluent Broca's aphasia should be reformulated as a combination of aphasia and AoS (Bernt & Caramazza, 1980; Mohr *et al.,* 1978).

Bernt & Caramazza (1980) *state:*
'The symptoms that characterize Broca's aphasia are explained as predictable behavioral manifestations of **a central disruption of the syntactic parsing component of the language system**, coupled with a (theoretically independent) **articulatory deficit that affects only the speech output system**.'

Mohr et al. (1978), following a large survey of cases, concluded that Broca's aphasia did not result from a lesion limited to Broca's area, but resulted from:

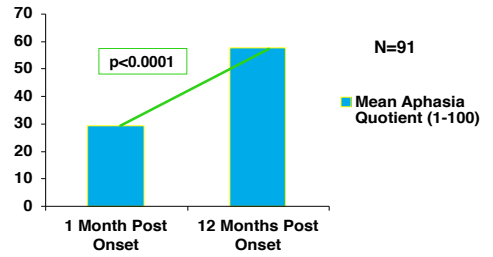
- a large lesion involving the area of supply of the upper division of the left middle-cerebral artery which produces a **global aphasia**.
- The damage includes the operculum, the 3rd frontal convolution, the anterior parietal region, the insula, and both sides of the central Rolandic fissure, extending deep into the underlying white matter.
- This produces what they defined as '**Big Broca's aphasia**' or *the **operculum syndrome**,* with:
- a severe and persisting apraxia of speech with either mutism or a nonlexical speech automatism (eg, *ti, ti; tan, tan)* with the later emergence of agrammatism and severe reading and writing problems if there is any recovery.
- Therefore, we shall consider recovery and progression in nonfluent aphasia as well as AoS.

- **Apraxia in stroke** typically occurs following a 'horizontal' lesion affecting large areas of neighbouring brain, whereas…
- Evidence suggests that **progressive apraxia** develops 'vertically', transmited along neuronal pathways rather than by anatomical proximity.
- The possibility arises that progressive damage reveals a gradual unfolding of neural phylogeny that mirrors the evolution of the nervous system.
- While the gradual recovery of speech in *nonfluent* aphasia and AoS following anterior stroke are relatively well known…
- the longitudinal patterns of deterioration in gradually progressive AoS and nonfuent aphasia are less well known.

Recovery of AoS with Nonfluent Aphasia From Stroke and

Dissolution of Language & Speech Production in Progressive AoS & Nonfluent Aphasia

Post-stroke aphasia improves significantly on aphasia batteries like the *Western Aphasia Battery (WAB)*

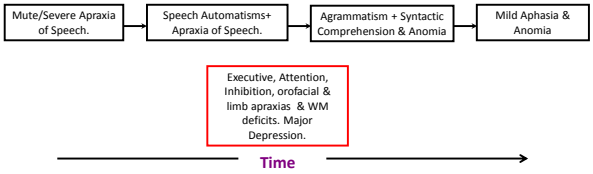


Code,C., Rowley, D. & Kertesz, A. (1994) Predicting recovery from aphasia with connectionist networks: preliminary comparisons with multiple regression. *Cortex*, 30, 527-532

Two Main Contrasting Types of Primary Progressive Aphasia (PPA)

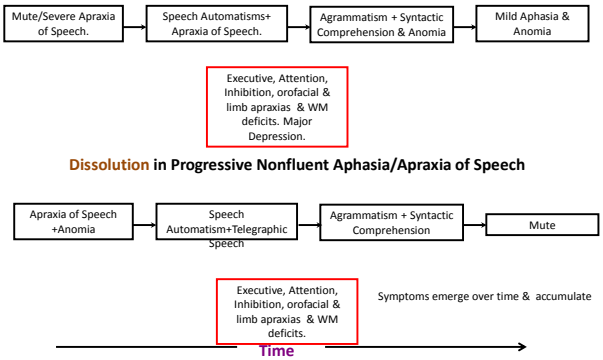
- **Primary Nonfluent Aphasia (PNFA)**
  - the progressive 'equivalent' of 'Big' Broca's aphasia **with apraxia of speech (pAoS)** from fronto-temporal-parietal damage?
- **Semantic Dementia (SD)**
  - the progressive 'equivalent' of Wernicke's/Jargonaphasia from temporo-parietal damage?

Recovery from Nonfluent 'Big' Broca's Aphasia with AoS) From Stroke



Up to 23.1% of people diagnosed as *global* or *Big Broca's* make good recoveries on the WAB (Kertesz, 1979) as symptoms diminish.

Recovery from Nonfluent 'Big' Broca's Aphasia From Stroke



Where does this leave us…?

- **Taking a symptomatic approach** the **recovery** of aphasia in stroke and **dissolution** in progression appear to be reversals of each other**.**
- But symptoms & syndromes and their relationship to linguistic domains take us only so far. Identical **symptoms can result from different causes.**
- Aphasia, like language, is not a unitary entity **and symptoms are multidimensional**. **There are significant differences in underlying causes of aphasic and apraxic symptoms from stroke & pAoS/PNFA in the nature of nonfluency.**
- Variable deficits in underlying **cognitive processes** essential for language processing – working memory, praxis, executive functions and mood disorders, all contribute to the observed syndrome.
- How much of the observed **recovery** or **dissolution** is a function of **restoration** (in stroke) **or of compensation/adaptation** and the impact of emotional state?

Significant Differences

Recent investigations suggest that there are significant differences between progressive and stroke aphasia with AoS at the 'sub-symptom' level. Particularly in:

- Single word vs. connected speech production
- Speech automatisms

Single Word vs. Connected Speech Production

- While single word production is relatively fluent in PNFA and pAoS…
- **connected speech is slow**, distorted with
  - syntactic errors,
  - shorter utterances,
  - lack of spontaneity
  - reduced complexity (Wilson et al, 2010; Patterson et al., 2006; Sajjadi et al., 2012; Code et al, 2013).
- This is **the opposite pattern** to nonfluent aphasia from stroke.
- Connected speech production in particular can have **significant diagnostic and theoretical relevance**.
- Connected speech has been compared in groups with
  - Semantic dementia (Wilson et al., 2010 ; Sajjadi, Patterson, Tomek, & Nestor, 2012a),
  - PNFA (Wilson et al., 2010 ; Sajjadi, Patterson, Tomek, & Nestor, 2012b),
  - stroke aphasia, Alzheimer' s disease (Knibb,Woollams, Hodges, & Patterson., 2009 ; Patterson et al., 2006).
  - pAoS & PNFA single case (Code et al., 2013)

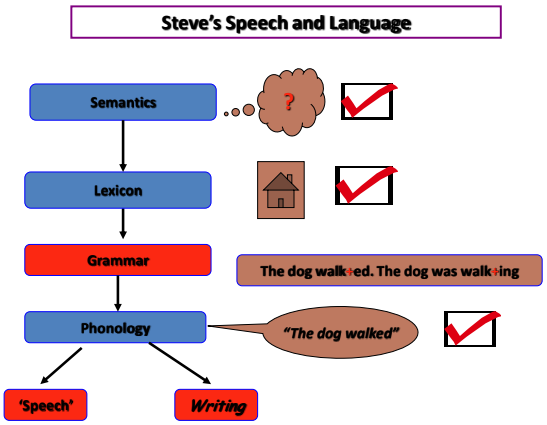
- **Steve was 65-68-yrs (dob: 23/08/40)**
- **He first noticed speech problems in November 1994.**
- **Summer of 1994, a bang on the head from a caravan door. Unconscious for about 1 minute.**
- **A 'First' in Chemistry from University College, London.**
- **School Master in a well-known Scottish public school for 21 years. Head of the Chemistry Department**
- **Retired early because of speech problems**
- **Became depressed and frustrated but outgoing & sociable.**
- **Steve died following a severe chest infection in December, 2007, and a post-mortem was carried out.**

Disinhibition/Impulsivity

- An impulsive tendency was noted early on by his family.
- Testing indicated some executive impairment, attentional impairment and disinhibition.
- A questionnaire developed (Nyatsanza et al. 2003) to examine behavioural changes was completed by Steve's wife.
- This indicated that he had became more **rigid & fixed in his opinions** and **developed routines that were difficult to discourage** with a frequency of several times per week.

- Nyatsanza, S., et al. (2003). A study of stereotypic behaviours in Alzheimer's disease and frontal and temporal variant frontotemporal dementia. Journal of Neurology, Neurosurgery and Psychiatry, 74, 1398–1402.

Steve's Speech and Language



Speech

Motor speech examination:

- **no apparent motor weakness or sensory loss, but some spastic *dysarthriaphonia* (strained-strangled voice)**
- **Problems initiating laryngeal & oral movement (coughing & producing vowels on command almost impossible)**
- **Dabul Apraxia Screening Battery revealed a range of praxic problems**

Connected Speech in PNFA & Nonfluent Aphasia in Stroke

	Control (N5) Means (sd)	PNFA Means (sd)	Stroke NFA Means (sd)
Reading Passage			
Patterson et al 2006			
Wpm	175.4 (35.0)	50.1 (37.4)	22.5 (17.7)
Total Time	47.0 (9.8)	182.5 (111.2)	342.0 (175.1)
Wilson et al 2010 WPM		50.9 (25.3)	
Automatic Counting			
Wpm	105.9		
	Mean 17 secs		

From Patterson, K., Graham, N., Lambon-Ralph, M., & Hodges, J. (2006). Progressive non-fluent aphasia is not a progressive form of non-fluent (post-stroke) aphasia. Aphasiology, 20, 1018–1034.

Steve's Speech Production – Single Words

'Bread'

'No'

'Less'

'Bad'

'Cold'

Steve's connected speech & matched controls with data on PNFA (N10) and Stroke NFA (N10) for rates of connected speech (words per minute - wpm).

	Steve Time1	Steve Time2	Control (N5) Means (sd)	PNFA Means (sd)	Stroke NFA Means (sd)
Picture Description					
Wpm	12.7	6.3	137.4 (35.4)	27.8 (18.7)	25.2 (11)
Total Words	19	11			
Total Time	1.30 secs	1.44 secs			
Conversation					
Total Time	4 mins. 41 secs				
Steve Pause Time	1 min. 43 secs				
Longest Pause	21 secs				
Total Words	38				
Mean utterance length	1.72 words				
Longest utterance	5 words				

PNFA & CVA/NFA figures from Patterson, et al (2006).

Continued…

Steve's connected speech and matched controls and data on PNFA (N10) and Stroke NFA (N10) and for rate of **connected speech** (wpm).

	Steve T1	Steve T2	Control (N5) Means (sd)	PNFA Means (sd)	Stroke NFA Means (sd)
Reading Passage					
Wpm	43	14.3	175.4 (35.0)	50.1 (37.4)	22.5 (17.7)
Total Time	180	544	47.0 (9.8)	182.5 (111.2)	342.0 (175.1)
Word Omissions	8	9			
Automatic Counting					
Wpm	26.4	14.3	105.9 (Mean 17 secs)		
Time Duration					
1-30	70	34			
1-10	*15*	*39*			
11-20	*27*	*39*			
21-30	*28*	*52*			

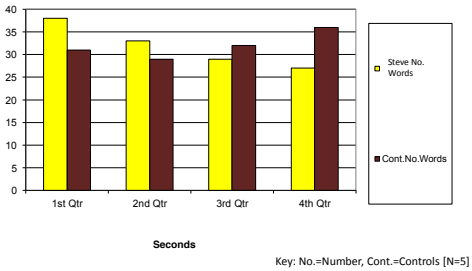
Steve's Connected Speech Production

Grandfather Passage
February 2002

Steve's 'Apraxia of Speech'

- significant initiation problems
- significant pausing *between* words in earlier continuous speech, though single words and short phrases are produced relatively quickly
- intrusive *schwa ('script' -> /scrip-a-ta/* & some between-word pausing, but less within words.
- Connected speech severely impaired
- Single word production significantly better than connected speech, with increasing fatigue with progress through - *a diagnostic sign?*

Connected Speech
Changes in speech rate in Steve & controls in **connected reading aloud**
(Grandfather Passage).

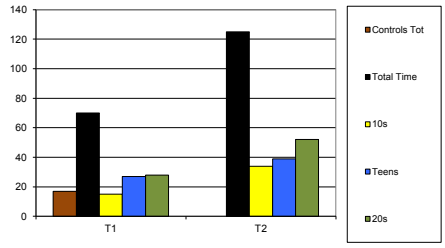


Key: No.=Number, Cont.=Controls [N=5]

Steve slows down as he progresses through the 130 word passage
Controls speed up

Connected Speech

Time taken for Steve to recite the 10s (1-10), the teens (11-20) and the 20s (21-30) at Time 1 and Time 2 and controls (N=5) total time in **automatic counting**.



T1=Time 1; T2=Time 2; Tot.=Total.

Steve's duration gradually increases as he recites 1-30

Utterance length & response latencies in Single Words

for Steve & age, sex, education matched controls in 35 repeated high frequency single syllable words.

	Utterance length	Response latencies
	Mean Length in msecs (sd; range)	
Steve	.478 (.122)	.868 (.442; .256-2.518)
Controls	.623 (.104)	.635 (116; .403-.928)
Mean Diff.	.143*	.233^

* t = 6.789 (df 34); p<.0001; ^t = 2.763 (df=34); p< .009.
Time 1

Note that Steve produces ***shorter* single words** than controls.

Aphasic Speech Automatisms in Nonfluent (Big Broca's) Aphasia from Stroke

Lexical:

Pronoun+Modal/ Aux Verb (most common):
- *I want…; I can/can't…; You can't…; I think…;*
- *{I can talk.,} -> {I can try…}*

Others: Expletives, Proper Names, Numbers, Yes/No

Nonlexical:

- **/tu tu/**
- **/di di/**
- **/tan tan/**

Both nonpropositional & typically occur every time, or nearly every time, **when attempting speech,** with no apparent relationship to context.

In severe form may be **unaware** due to **attention or inhibition deficit.**

Speech automatism occur in progressive nonfluent aphasia too, but they appear to be different.

Steve's Automatisms:

- *yes yes*
- *right yes* or
- *right yes.*

- They occurred **after** production of single words & **before** gestures in testing, and less in connected speech, and **not in place of more propositional speech**.
- Occurred when there was an **opportunity**, little opportunity in connected speech.
- In this case, appeared to reflect a **vocal overflow of inner speech due to some disinhibition** as an **affirmation of a correct response** & associated with occurrences of apraxia of speech.
- **Unaware -** the **underlying cause appeared to be disinhibition,** and weak attention/monitoring, **not aphasia.**

Code, C., Tree, J. & Dawe, K. (2009) Opportunities to say 'yes': rare speech automatisms in a case of progressive nonfluent aphasia and apraxia. **Neurocase,** 15, 445-458

Orofacial Movements to Command

Action	8/2/02	– 23/8/02
Stick out your tongue	5	4
Roll your tongue	5	2
Push cheeks out with tongue	5	4
Try and touch nose with tongue Lick	3	1
bottom lip	5	4
Whistle	2	1
Puff out cheeks	5	3
Kiss	3	3
Clear throat	4	2
Bite lower lip	5	3
Show me your teeth	5	3
Take a deep breath	4	3
Open your mouth wide	5	4
Smile	5	3
Pout with bottom lip	5	2
Cough	3	2
TOTALS (max=80)	**69**	**44**

Conclusions

Recovery and dissolution of speech and language impairment in stroke and progressive aphasia appear on a symptomatic level to be reversals of each other, but…

Examination of the underlying causes of symptoms (single word vs. connected speech production, speech automatisms) suggest different causes that have relevance for management of progressive AoS.

Hardly surprising perhaps, given that…

- **Apraxia and aphasia in stroke** typically occurs following an 'horizontal' lesion affecting large areas of neighbouring brain reflecting **impairments to blood supply**.
- Evidence suggests that **progressive apraxia and aphasia reflects neuronal damage** which develops 'vertically', transmitted along **neuronal pathways that may underlie core neural networks,** rather than by anatomical proximity.
- The possibility arises that progressive damage reveals a gradual unfolding of neural phylogeny that mirrors the evolution of the nervous system.
- Treatments would need to take account of progressive underlying impairments & their impact on everyday family life.

Problems and future research:

- The effects of **compensation** on the emergence of symptoms
- The effects of **time since onset** (in stroke) vs. **time since diagnosis/referral/emergence** (?)
- These issues are seldom considered or controlled for in studies in progressive apraxia of speech or progressive aphasia.

Implications for Management of Steve's Multiple Progressive Conditions

Implications of Impairments for Communication

Speech? – significant problems, and worsening, but could we use Steve's recorded speech?

Gesture? – apraxia made gesture very difficult and unreliable. He was able to retain the' thumbs up' and, less so, 'thumbs down' signs until the end, but unreliably.

Writing? – becoming impossible.

Eye Pointing? – unable to reliably point with his eyes

Drawing? – became illegible

Understanding? – good

Alternatives to Speech

- We trained & practiced some basic-needs gestures/pantomimes regularly

(Eg, Thumbs up, Thumbs down, Drink, Eat, Cigarette, Toilet, Goodbye)

with variable success. In the end he retained only the 'thumbs up'.

- When he became effectively mute, we used writing.
- We used drawing also, but using a pencil became difficult for him

Management

Augmentative Aids to Communication?

- **We arranged an assessment at Frenchay Hospital Augmentative Aids Centre. A *Lightwriter* was loaned to him and was useful for a while.**
- **We set up a small phone training programme, to communicate with his sons, which was partly successful for a while.**

Lightwriter?

- Steve's writing and praxic problems impacted eventually on his ability to us the *Lightwriter*. We arranged single recorded words into files that might be used later when he was mute.
- This had limited success, because by this time his praxic problems limited his ability to access the words - it came too late.

However, for other clients this is a strategy worth considering - record speech while it is good for use when speech is not possible.

Assessment for Alternative Means of Communication – Regional Augmentative Aids Centre, Frenchay Hospital

- ***Lightwriter SL40*** - a keyboard-based communication aid for literate users featuring artificial voices, frequency-based word prediction, environmental control and SMS text messaging, a tactile keyboard and two screens - one facing the user, the other outward facing. The keyboard letters are all lower case, and has three changeable keyguards.
- Very useful at first, but as his writing and praxic difficulties increased it became increasingly useless.

Communication Boards

- To assist people who have little or no spoken language.
- This one is an A4 laminated card, featuring 15 photographs with matching phrases related to basic personal needs. It also includes the numbers one to ten, the letters of the alphabet, and icons for *Yes* and *No*.
- It goes some way towards returns control of conversation to the individual, carers, family and friends, who can be confident that they understand the wishes of the person who has lost their language skills.

Although basic, it provided some communication ability to Steve, and was not too difficult for him to use.

The Speakability Medical Passport

- Enables an aphasic people to discuss their medical needs with their doctor on an 'equal' basis.
- By being in possession of their own medical communication tool, a person regains a sense of control over their ability to communicate, and in a situation where it is most needed.
- The idea came from aphasic people who were instrumental in developing the contents to ensure that it fulfills all their requirements as a patient.

A Personalised Communication Resource

- In conjunction with Steve's son, who lived with Steve, we developed a *personalised communication folder*
- Used photographs and names of family, friends, involved professionals (Dr, SLT, CC) and places regularly visited (supermarket, university, park, hospital, pub) together with written names.
- It included photographs with words of objects in daily use – TV, radio, cigarettes, food, wine, beer, cloths.
- This was a particularly useful and well used resource.

Psychosocial

- Steve was in denial and ever hopeful that things would improve. Had a fear of Creutzfeldt-Jakob's Disease. We were able to convince him that he should have no fear of that, but the results of his last MRI scan would have been clearly devastating news for him.
- We did not show him his MRI scan
- We did tell him he had a serious brain condition that was causing his communication difficulties that he could be helped with.
- Steve's voluntary emotional facial expression was severely impaired.
- He rejected all suggestions that he might want counseling.
- Steve had a good social life early on – did accounts for his local bus company, Chair of his Speakability Self-Help Group, helped out in his local Post Office, drove his own car.

These activities reduced as his conditions deteriorated.

Family Liaison and Counseling

- We saw Steve's wife and two sons regularly, and they were fully informed of all our findings.
- He became separated and estranged from his wife, who went to live in France with a new partner.
- He had not got on well with sons or wife for many years.
- His younger son eventually moved into Steve's flat to care for him and was most actively engaged in his management and used his considerable IT skills to attempt ways to use Steve's recorded speech to aid communication
- Steve had a good SLT and GP, but a poor neurologist.

Thank You!

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