Journal of Communication Disorders 51 (2014) 64–77

Contents lists available at ScienceDirect

Journal of Communication Disorders

# Auditory feedback perturbation in children with developmental speech sound disorders

Hayo Terband [a,b,*], Frits van Brenk [a], Anniek van Doornik-van der Zee [a,c]

[a] Utrecht Institute of Linguistics-OTS, Utrecht University, Utrecht, The Netherlands
[b] Centre for Language and Cognition (CLCG), University of Groningen, Groningen, The Netherlands
[c] Division of Speech and Language Therapy, HU University of Applied Sciences Utrecht, Utrecht, The Netherlands

ARTICLE INFO

*Article history:*
Received 19 April 2014
Received in revised form 7 June 2014
Accepted 30 June 2014
Available online 1 August 2014

*Keywords:*
Speech sound disorders
Childhood Apraxia of Speech
Auditory feedback control
Auditory feedback perturbation
Internal models

ABSTRACT

*Background/purpose:* Several studies indicate a close relation between auditory and speech motor functions in children with speech sound disorders (SSD). The aim of this study was to investigate the ability to compensate and adapt for perturbed auditory feedback in children with SSD compared to age-matched normally developing children.
*Method:* 17 normally developing children aged 4.1–8.7 years (mean = 5.5, SD = 1.4), and 11 children with SSD aged 3.9–7.5 years (mean = 5.1, SD = 1.0) participated in the study. Auditory feedback was perturbed by real-time shifting the first and second formant of the vowel /e/ during the production of CVC words in a five-step paradigm (practice/familiarization; start/baseline; ramp; hold; end/release).
*Results:* At the group level, the normally developing children were better able to compensate and adapt, adjusting their formant frequencies in the direction opposite to the perturbation, while the group of children with SSD followed (amplifying) the perturbation. However, large individual differences lie underneath. Furthermore, strong correlations were found between the amount of compensation and performance on oral motor movement non-word repetition tasks.
*Conclusions:* Results suggested that while most children with SSD can detect incongruencies in auditory feedback and can adapt their target representations, they are unable to compensate for perturbed auditory feedback. These findings suggest that impaired auditory–motor integration may play a key role in SSD.

**Learning outcomes**: The reader will be able to: (1) describe the potential role of auditory feedback control in developmental speech disorders (SSD); (2) identify the neural control subsystems involved in feedback based speech motor control; (3) describe the differences between compensation and adaptation for perturbed auditory feedback; (4) explain why auditory–motor integration may play a key role in SSD.



* Corresponding author at: Utrecht Institute of Linguistics OTS, Utrecht University, Trans 10, kmr. 0.25, Utrecht, 3512 JK, The Netherlands. Tel.: +31 30 253 7546.
*E-mail addresses:* h.r.terband@uu.nl (H. Terband), f.j.vanbrenk@uu.nl (F. van Brenk), anniek.vandoornik@hu.nl (A. van Doornik-van der Zee).

http://dx.doi.org/10.1016/j.jcomdis.2014.06.009
0021-9924/

## 1. Introduction

### 1.1. Developmental speech sound disorder

Pediatric or developmental speech sound disorder (SSD) is an umbrella term that encompasses a variety of diagnostic labels, such as Speech Delay, Phonetic Articulation Disorder, Phonological Disorder with subtypes consistent or inconsistent, and pediatric Motor Speech Disorders with subtypes Childhood Apraxia of Speech and developmental dysarthria (Dodd, 2005; Lewis et al., 2006; Strand, McCauley, Weigand, Stoeckel, & Baas, 2013; Terband, Maassen, Guenther, & Brumberg, 2014). Although these categories are meant to represent core impairment at different levels of speech development and different parts of the speech production chain, speech and language processes are interactive and influence each other during development (Kent, 2004; Smith & Goffman, 2004; Strand et al., 2013; Terband & Maassen, 2010; Terband et al., 2014). Consequently, there is a large overlap in clinical symptoms which, moreover, change during successive stages of development: often starting with no or little babbling and subsequently less variegated babbling during the first year, followed by a slowed expansion of vocabulary during the second year, and the presence of many speech sound substitution errors in kindergarten (Maassen, 2002; Maassen, Nijland, & Terband, 2010; Terband & Maassen, 2010). From then on, the speech is characterized in general as unintelligible and often effortful, with a high rate of mispronunciations or apparently 'normal' slips of the tongue (ASHA, 2007; Dodd, 2005; Maassen et al., 2010; Shriberg, 2010).

Prevalence of SSD reported in the literature is around 6% (Broomfield & Dodd, 2004; Campbell et al., 2003; Law, Boyle, Harris, Harkness, & Nye, 2000; McKinnon, McLeod, & Reilly, 2007; Shriberg, Tomblin, & McSweeny, 1999) and SSD are estimated to form about 75% of all communication disorders in children (Van Borsel, 2009). The majority of these children visit speech pathologists for examination and treatment. However, little is known about the mechanisms behind SSD. More specifically: how do the phonological and sensorimotor impairments relate to the core deficit, derived or consequential deficits and adaptive strategies, and how do they express themselves in symptomatology? This knowledge could be crucial for effective diagnosis and treatment of children that suffer from SSD.

Findings from both behavioral and neurocomputational modeling studies indicate that the interaction between auditory and speech motor functions may play a critical role in SSD. Empirical data indicate that there is a close relation between production symptoms and perceptual acuity in children with speech sound disorders (SSD): poor perceptual performance is related to poor production performance (Edwards, Fox, & Rogers, 2002; Groenen, Maassen, Crul, & Thoonen, 1996; Maassen, Groenen, & Crul, 2003; Nijland, 2009; Raaymakers & Crul, 1988). Computer simulations with the DIVA model suggest that the ability for auditory self-monitoring might influence the nature of speech-motor deficits during development (Terband et al., 2014). These findings indicate that the quality of auditory feedback and the capacity to use it may be key factors in SSD.

### 1.2. Auditory feedback control in adults and children

The role of auditory feedback in speech production is twofold. First, it is important for acquiring sensory goals for producing speech sounds and serves as a teaching signal for the acquisition and adaptation of speech motor programs (Guenther, Hampson, & Johnson, 1998; Guenther & Perkell, 2004; Perkell, 2012; Perkell et al., 1997). Second, auditory feedback serves as a guiding signal in the online control and correction of speech movements. The coordination of the timing and magnitude of the movements in the different systems involved (i.e., the muscular structures controlling respiration, resonance, phonation, and the articulators) has been found to be largely dependent on auditory feedback (Perkell, 2012; Perkell et al., 2007, 1997). Furthermore, several studies demonstrated that perturbation of auditory feedback during speech production elicits a compensatory response in the opposite direction to maintain the intended auditory outcome (Cai, Ghosh, Guenther, & Perkell, 2010; Houde & Jordan, 2002; Villacorta, Perkell, & Guenther, 2007). In this experimental paradigm, an acoustic cue is manipulated in real time during speech production and presented to the speaker through headphones. This creates an apparent mismatch between the speech sound the speaker intended to produce and what he/she hears. Using the auditory feedback control subsystem, the speaker is able to generate a compensatory response to the acoustic shifts in order to match the intended target. However, it has been found that some speakers show production changes that follow the direction of the perturbations and not all speakers show a consistent response. Typically, around 70% of the speakers show (partial) compensation (see Cai et al., 2010 for an overview). A possible explanation is that in the speakers who do not show a response, the perturbations might be undetected by the speech motor control system (Purcell & Munhall, 2006). It has also been suggested that there is a trade-off with somatosensory feedback and that individual speakers weight the different types of feedback differently (Katseff, Houde, & Johnson, 2012; Lametti, Nasir, & Ostry, 2012; Purcell & Munhall, 2006).

Only a few studies have investigated auditory feedback in speech development. Ménard and colleagues used a lip-tube to alter F1 and F2 of vowels produced by 4-year-old French speakers (Ménard, Perrier, & Aubin, 2013; Ménard, Perrier, Aubin, Savariaux, & Thibeault, 2008). Results showed that while the children were able to reach compensation in some vowels, a consistent compensatory effect was absent. This suggests that they were able to use auditory feedback to develop a compensatory strategy, but were unable to (learn to) update and store their representations. In an auditory perturbation study involving 9–11-year-old children, spectral properties of /s/ were manipulated toward /?/ in a series of monosyllabic words (Shiller, Gracco, & Rvachew, 2010). Results showed that the children were able to compensate for the altered auditory feedback to a comparable degree as adults, albeit with a larger token-to-token variability. These findings were replicated for

vowel perturbations in 4-year-olds by MacDonald and colleagues (MacDonald, Johnson, Forsythe, Plante, & Munhall, 2012) in a study that investigated the ability to compensate for perturbation of F1 and F2 of the vowel /ɛ/ in toddlers (2-year-olds) and young children (4-year-olds) as compared to adults. While the 2-year-olds did not show any response to the perturbation, the results for the 4-year-olds showed similar compensation with a larger token-to-token variability compared to the adults (MacDonald et al., 2012). In a recent study in our own lab (van Brenk, Terband, & Cai, 2014), we investigated auditory perturbation of F1 and F2 of the vowel /e/ in CVC-words in 4–9 year-old children compared to young adults (aged 19–29 years). The results showed that the children were able to compensate and adapt in a similar or larger degree compared to the young adults, even though the proportion of speakers displaying a consistent compensatory response was higher in the group of adults (van Brenk et al., 2014). Furthermore, results did not show differences in token-to-token variability. The increased ability to successfully use auditory feedback for compensation and adaption suggests that crucial steps are made in the development of auditory–motor integration around the age of 4.

### 1.3. Auditory feedback in children with SSD

Only a few studies have investigated auditory feedback in children with SSD, and these have mainly focused on subtype CAS. In terms of psychomotor function, the deficit CAS can be summarized as a deficit of generating robust motor speech commands. Terband and colleagues (Terband & Maassen, 2010; Terband, Maassen, Guenther, & Brumberg, 2009) theorized that children with CAS suffer from a disordered development of the functional synergies/coordinative structures that underlie speech motor coordination causing impairment of the forward model leading to poor feedforward control. Subsequently, the introduction of errors due to poor feedforward control would cause the speech production system to rely more heavily on the sensory feedback control subsystems, as a derived deficit or as an adaptive strategy.

This idea was investigated in a series of computer simulations with the DIVA model in which the reliance on feedback control was systematically increased during production attempts in the acquisition of forward commands. The simulations accounted for four key speech-motor symptoms of speech production in CAS: stronger coarticulation, distorted productions of speech sounds, searching articulatory behavior, and high variability across productions (Terband et al., 2009). Subsequent computer simulations suggested that facilitating the use of auditory feedback by slowing down articulation rate could enhance the learning of motor commands in the context of a speech-motor deficit (Terband & Maassen, 2010).

Very recently, Iuzzini and colleagues investigated the effects of auditory feedback masking on vowel space and voice onset time (VOT) for voiceless plosives in the speech of school-aged children with CAS and SD compared to age-matched normally speaking children (Iuzzini, Green, & Hogan, 2014; Iuzzini, Hanen, Green, & Hogan, 2013). The results showed a smaller vowel space area in the children with CAS relative to the normally developing children when auditory feedback was masked while they performed similar to their peers in the unmasked condition (Iuzzini et al., 2014). In terms of VOT, results revealed a masking effect for the group of children with CAS while the children with SD and normally speaking children did not show an effect of masking (Iuzzini et al., 2013). The children with CAS produced shorter VOT's when auditory feedback was masked, which would cause a deterioration of the voicing contrast. Consistent with earlier findings (Lewis, Freebairn, Hansen, Iyengar, & Taylor, 2004), the children with CAS also produced shorter VOT's compared to their normally speaking peers in general. Together, these findings are in support of increased reliance on auditory feedback in CAS and suggest that auditory feedback may facilitate speech production in school-aged children with CAS.

### 1.4. Aim of the present study

To unravel the role of auditory feedback control in SSD, the aim of the present study was to investigate the ability to compensate and adapt for perturbed auditory feedback in children with complex SSD (age 4–8) compared to age-matched normally developing children and correlate these abilities with speech-motor and phonological symptoms based on standardized speech/language production and perception tests. Auditory feedback was perturbed by shifting the first and second formant of the vowel /e/ in real time during the production of CVC words in a five-step paradigm comprising practice/familiarization, start/baseline (no perturbation), ramp (amount of perturbation is slowly increased), hold (full perturbation) and end/release (no perturbation). The difference in formant frequencies between start and hold denotes the amount of compensation and forms a measure of motor control, i.e. whether speakers are able to notice and act on the mismatch between the motor command and the corresponding auditory result. The difference in formant frequencies between start and end denotes the amount of adaptation and is a measure of motor learning, i.e. whether speakers are able to update their speech motor representations.

Based on previous research suggesting that children with SSD demonstrate an increased reliance on auditory feedback, this particular group might be expected to show stronger compensatory effects as compared to their healthy peers. On the other hand, the process to detect the incongruencies in auditory feedback and create an online compensatory response relies heavily on reliable internal (forward and inverse) models, which have been hypothesized to be weak or underspecified in children with SSD and particularly in children with subtype CAS (Terband & Maassen, 2010; Terband et al., 2014). Such an impairment of internal models would present itself as a reduced compensatory effect in children with SSD as compared to normally developing children. Furthermore, we hypothesize that the strength of adaptation will be weaker in the group of children with SSD, compared to the age-matched control group. Previous studies have associated children with SSD with an implicit learning deficit (Iuzzini et al., 2013; Shriberg, Lohmeier, Strand, & Jakielski, 2012), which would present itself as

an impaired ability to use auditory feedback to update feedforward representations. According to this hypothesis, motor learning as measured by the strength of adaptation will be weaker in the children with SSD as compared to their age-matched control group.

## 2. Methodology

### 2.1. Participants

A total of 28 children participated in the study. All participants were native speakers of Dutch. Eleven children with SSD (6 males and 5 females aged 3.9–7.5 [mean = 5.1 years, SD = 1.0 years]) were referred by speech pathologists. The diagnosis of the children with SSD was established using standardized speech perception and production tests, and a short case history. No participants suffered from hearing problems (pure-tone thresholds not exceeding 25 dB HL), language comprehension problems (a score less than 1 SD below population average), subnormal intelligence (IQ less than 1 SD below population average), organic disorders in the orofacial area, gross motor disturbances, or dysarthria. The normally developing children (8 males and 9 females, aged 4.1–8.7 [mean = 5.5 years, SD = 1.4 years]) were recruited through local schools and acquaintances. Detailed background- and diagnostic data are presented in Table 1. Groups were equivalent on age [$t(26) = .898$, $p = .377$], gender, [$\chi^2(1) = .150$, $p = .699$], receptive vocabulary [WCQ: $t(26) = 1.111$, $p = .277$], and auditory discrimination [words: $t(25) = 1.510$, $p = .143$; nonwords: $t(26) = .976$, $p = .338$] but the group of children with SSD scored significantly lower on intelligibility [ICS: $t(26) = 4.945$, $p < .001$].

The study protocols were approved by the Medical Ethical Screening Committee of the University of Applied Sciences Utrecht. Written consent was obtained from all parents or caretakers prior to starting the study.

### 2.2. Experimental procedures

The data were collected at the children's school, speech clinic, or at a familiar local community center. For each child, two 60-min sessions were planned in two consecutive weeks to collect all data. Regarding the standardized speech/language production and perception tasks, the PVVT, PALPA, and CAI were administered by the third author, while the oral motor movement assessment was administered by an independent speech and language therapist. The PALPA and the CAI were administered by computer and presented over headphones (type Philips SBC HP800) and in the case of CAI, audio were recorded by an omnidirectional externally powered table microphone (type Shure 2XU). Finally, the ICS questionnaire was filled in by one of the children's parents/caretakers.

For the auditory perturbation protocol, the experimental procedure was as follows. The participants were seated in front of a PC-monitor showing pictures of the target words, to be read or named aloud. The stimuli were three Dutch CVC words all containing a long unrounded close-mid front vowel: /be?r/ (bear), /ve?r/ (feather), /pe?r/ (pear). An animated bird flying over one of the pictures cued the participant to speak the intended word. The Audapter software module (Cai et al., 2010), a custom MEX-based software module written in Microsoft Visual C++ and executed under MATLAB, was used to track and shift vowel formants, and played back the target word over headphones (type Sennheiser HD 380 pro) in real-time. The first formant was raised 25% and the second lowered 12.5%, yielding a more open and more central vowel. With these manipulation parameters, it was ensured perturbation effects were not overtly audible, and the vowel quality was unchanged. Recordings were made by an externally powered lapel microphone (type Audio-Technica AT803b). The perturbation paradigm consisted of five phases. See Fig. 1 for a schematic overview. In the practice phase, participants were made familiar with the paradigm, and trained to produce the words within certain loudness and duration boundaries (74–84 dB SPL at 10 cm microphone distance, 300–500 ms duration), to optimize formant tracking and shifting. The start phase served as a baseline for unperturbed vowels. In the ramp phase, the perturbation was introduced and linearly ramped to maximum perturbation. In the hold phase, the maximum perturbation was applied, followed by the end phase where perturbation was suspended. The total number of tokens was 111. Due to demonstrated fatigue and attention loss, children aged below 7.0 years participated in a shorter version of 84 tokens.

### 2.3. Data analysis

Of the standardized speech/language production and perception tasks, the PVVT, DDK, and oral motor movement assessment were scored by the first and third authors while the PALPA was scored automatically by computer. The CAI was transcribed and scored in consensus by the third author and an independent transcriber based on broad phonetic transcription. For each production obtained in the auditory perturbation experiment, the mean first formant and mean second formant were measured from steady-state portions of the produced vowels using custom scripts for PRAAT (Boersma & Weenink, 2013). To remove speaker-specific and word-specific differences in formant values, the first formant and second formants were normalized using averaged formant values for each vowel in the start phase, separately for each speaker and each stimulus. The amount of compensation for auditory perturbation was quantified by calculating the difference in normalized formant frequencies between the hold and start phases, while the amount of adaptation to prolonged auditory perturbed vowels was quantified by calculating the differences in normalized formant frequencies between the end and start

**Table 1**
Overview of the children that participated in the study. PD = Phonological Disorder; DS = Developmental Stuttering; PAD = Phonetic Articulation Disorder; CAS = Childhood Apraxia of Speech. A detailed description of the diagnostic tasks can be found in Appendix A.

| ID | Diagnosis | Age (y) | Sex | WCQ (PPVT[1]) | Intelligibility (ICS[2]) | Auditory discrimination (PALPA[3]) | | Diadochokinesis (DDK; pataka CAI[4]) | | Oral-motor mov. assessment |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Words (% correct) | Nonwords (% correct) | Score[a] | Judgment[b] | Iso – seq – seq fast (% correct) |
| SSD1 | PD | 5.8 | m | 127 | 4.00 | 94 | 100 | 1 | 2 | 85 – 83 – 60 |
| SSD2 | PD/DS | 4.8 | m | 98 | 4.43 | 97 | 81 | 1 | 3 | 85 – 78 – 40 |
| SSD3 | PD+PAD | 7.5 | f | 106 | 4.00 | 94 | 86 | 1 | 3 | 92 – 94 – 50 |
| SSD4* | CAS | 4.2 | m | 110 | 3.86 | 47 | 78 | – | – | 35 – 50 –10 |
| SSD5 | PD | 6.6 | f | 84 | 3.42 | 64 | 44 | 1 | 3 | 77 – 67 – 40 |
| SSD6* | CAS/PD | 4.6 | m | 85 | 4.29 | 56 | 47 | 1 | 1 | 58 – 33 – 30 |
| SSD7* | CAS/PD | 3.9 | f | 129 | 3.57 | 61 | 58 | 0 | 0 | 65 – 83 – 30 |
| SSD8 | CAS/PD | 6.1 | m | 106 | 3.14 | 92 | 69 | 0 | 0 | 96 – 95 – 50 |
| SSD9* | PD+PAD | 4.3 | m | 126 | 3.57 | 39 | 42 | 0 | 2 | 85 – 67 – 70 |
| SSD10 | PD+PAD | 5.0 | f | 104 | 4.30 | 81 | 83 | 1 | 3 | 85 – 100 – 80 |
| SSD14 | CAS/PD | 5.0 | f | 115 | 3.86 | 94 | 86 | 0 | 0 | 77 – 78 – 60 |
| CON1 | NO | 7.9 | m | 120 | 5 | 94 | 94 | 1 | 4 | – |
| CON2 | NO | 7.1 | m | 117 | 5 | 92 | 89 | 1 | 3 | – |
| CON4 | NO | 4.1 | f | 104 | 5 | – | 31 | 0 | 2 | – |
| CON5 | NO | 4.4 | f | 123 | 5 | 94 | 83 | 1 | 3 | – |
| CON6 | NO | 4.9 | m | 125 | 5 | 86 | 69 | 1 | 3 | – |
| CON7* | NO | 5.7 | m | 107 | 4.43 | 72 | 78 | 1 | 4 | – |
| CON8 | NO | 4.1 | m | 90 | 4.28 | 81 | 72 | – | – | – |
| CON9 | NO | 7.3 | f | 110 | 5 | 78 | 78 | 1 | 3 | – |
| CON10 | NO | 4.3 | f | 104 | 4.14 | 72 | 61 | 1 | 3 | – |
| CON11 | NO | 5.2 | m | 111 | 4 | 94 | 78 | 1 | 4 | – |
| CON12 | NO | 6.5 | f | 115 | 4.43 | 97 | 97 | 1 | 4 | – |
| CON14 | NO | 7.1 | m | 130 | 5 | 97 | 94 | 1 | 3 | – |
| CON15 | NO | 4.1 | m | 139 | 5 | 92 | 94 | 1 | 2 | – |
| CON16* | NO | 4.2 | f | 109 | 5 | 50 | 58 | – | – | – |
| CON17 | NO | 8.7 | f | 126 | 5 | 97 | 100 | 1 | 4 | – |
| CON18 | NO | 7.2 | f | 106 | 4 | 97 | 94 | 1 | 4 | – |
| CON19 | NO | 4.8 | f | 102 | 5 | 64 | 50 | 0 | 2 | – |

| ID | Diagnosis | Picture naming (60 words CAI) | | | | Word repetition (WR; 10 words CAI) | | | | Non-word repetition (10 non-words similar to WR CAI) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | PCCI[c] | PCCCI[d] | PSSC[e] | atyp.[f] /typ. sub.proc.[g] | PCCI | PCCCI | PSSC | atyp./typ. sub.proc. | PCCI | PCCCI | PSSC | atyp./typ. sub.proc. |
| SSD1 | PD | .94 | .70 | .96 | 3/0 | .98 | .50 | 1.00 | 1/0 | .88 | .27 | .94 | 4/1 |
| SSD2 | PD/DS | 1.00 | 1.00 | .99 | 0/0 | 1.00 | .91 | .97 | 0/0 | .79 | .36 | .88 | 11/5 |
| SSD3 | PD+PAD | 1.00 | .96 | .97 | 0/0 | 1.00 | .95 | 1.00 | 0/0 | .88 | .95 | .95 | 13/5 |
| SSD4* | CAS | .94 | .61 | .87 | 2/5 | .56 | .23 | .62 | 17/14 | – | – | – | – |
| SSD5 | PD | .81 | .39 | .82 | 4/8 | .69 | .77 | .82 | 5/7 | .65 | .50 | .73 | 18/24 |
| SSD6* | CAS/PD | .88 | .65 | .92 | 5/4 | .91 | .77 | .95 | 0/5 | .73 | .09 | .67 | 4/3 |
| SSD7* | CAS/PD | .82 | .30 | .71 | 3/9 | .63 | .73 | .73 | 0/13 | .66 | .27 | .67 | 14/39 |
| SSD8 | CAS/PD | .69 | .26 | .81 | 22/14 | .59 | .41 | .59 | 15/9 | .71 | .09 | .72 | 18/24 |
| SSD9* | PD+PAD | .84 | .13 | .76 | 12/7 | .56 | .91 | .78 | 17/8 | .95 | 1.00 | .97 | 5/2 |
| SSD10 | PD+PAD | .91 | .83 | .94 | 7/3 | .89 | .77 | 1.00 | 6/0 | .91 | .95 | .97 | 9/1 |
| SSD14 | CAS/PD | .57 | .13 | .80 | 28/9 | .48 | .23 | .62 | 23/11 | .71 | .27 | .80 | 26/10 |
| CON1 | NO | .97 | .91 | | 1/2 | 1.00 | 1.00 | 1.00 | 0/0 | .98 | 1.00 | .94 | 2/2 |
| CON2 | NO | 1.00 | 1.00 | .99 | 0/0 | 1.00 | 1.00 | 1.00 | 0/0 | .96 | 1.00 | 1.00 | 0/1 |
| CON4 | NO | .99 | 1.00 | .99 | 2/0 | 1.00 | 1.00 | 1.00 | 0/0 | .84 | .77 | .91 | 8/8 |
| CON5 | NO | 1.00 | .83 | .99 | 0/0 | 1.00 | 1.00 | 1.00 | 0/0 | .86 | 1.00 | .97 | 9/11 |
| CON6 | NO | 1.00 | 1.00 | .96 | 0/0 | 1.00 | 1.00 | 1.00 | 0/0 | .96 | .91 | .95 | 2/0 |
| CON7* | NO | 1.00 | 1.00 | 1.00 | 0/0 | 1.00 | 1.00 | 1.00 | 0/0 | .89 | .82 | .97 | 7/2 |
| CON8 | NO | – | – | – | – | .87 | .95 | .89 | 0/0 | – | – | – | – |
| CON9 | NO | 1.00 | 1.00 | 1.00 | 0/0 | .98 | 1.00 | 1.00 | 1/0 | – | – | – | – |
| CON10 | NO | .97 | .96 | .98 | 1/0 | 1.00 | .91 | .99 | 0/0 | .83 | .59 | .88 | 11/6 |
| CON11 | NO | .99 | 1.00 | 1.00 | 1/0 | 1.00 | 1.00 | 1.00 | 0/0 | .98 | 1.00 | .98 | 2/2 |
| CON12 | NO | 1.00 | 1.00 | 1.00 | 0/0 | 1.00 | 1.00 | 1.00 | 0/0 | .94 | 1.00 | .96 | 5/3 |
| CON14 | NO | 1.00 | .96 | .99 | 0/0 | .98 | .95 | .90 | 0/2 | .85 | .95 | .98 | 14/4 |
| CON15 | NO | .94 | 1.00 | 1.00 | 2/1 | 1.00 | .95 | .99 | 0/0 | .92 | 1.00 | .99 | 7/4 |
| CON16* | NO | .97 | .87 | .98 | 2/1 | .96 | 1.00 | .97 | 0/0 | – | – | – | – |
| CON17 | NO | 1.00 | 1.00 | 1.00 | 0/0 | 1.00 | 1.00 | 1.00 | 0/0 | .99 | 1.00 | 1.00 | 0/0 |
| CON18 | NO | 1.00 | .96 | 1.00 | 0/0 | 1.00 | 1.00 | 1.00 | 0/0 | .99 | .95 | .98 | 1/0 |

**Table 1** (*Continued*)

| ID | Diagnosis | Picture naming (60 words CAI) | | | | Word repetition (WR; 10 words CAI) | | | | Non-word repetition (10 non-words similar to WR CAI) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | PCCI[c] | PCCCI[d] | PSSC[e] | atyp.[f] /typ. sub.proc.[g] | PCCI | PCCCI | PSSC | atyp./typ. sub.proc. | PCCI | PCCCI | PSSC | atyp./typ. sub.proc. |
| CON19 | NO | .94 | .83 | .96 | 3/3 | .89 | .86 | .97 | 6/6 | .86 | .73 | .95 | 12/12 |

*Data collection of the auditory perturbation task was unsuccessful, because the participant was unable to successfully produce the target words within required loudness and duration limits, or could not finish the experiment due to fatigue or attention loss.

[a] DDK-score 1 = [pataka] could be produced; 0 = [pataka] could not be produced.

[b] DDK-judgment 4 = perfect; 3 = [pataka] in sequence in normal rate, but no acceleration; 2 = [pataka] in sequence incorrect ([t] or [k] could not be pronounced), but speeding up on two different consonants ([pata], [taka]) was possible; 1 = no fluent [pataka], not in sequence; 0 = no [pataka] production either in isolation or in a sequence of two.

[c] PCCI Proportion consonants correct in initial position.

[d] PCCCI Proportion consonant clusters correct in initial position.

[e] PSSC Proportion syllable structures correct (CV; VC; CVC; CCV; VCC; CVCC; CCVC; CCVCC).

[f] atyp. sub. proc. Number of atypical substitution processes (backing; abnormal stopping; denasalization; h-zation).

[g] typ. sub. proc. Number of typical substitution processes (fronting; stopping of fricatives or affricates; nasalization; gliding).

[1] Peabody Picture Vocabulary Test-III-NL (Dunn & Dunn, 2005).

[2] Intelligibility in Context Scale: Dutch (McLeod et al., 2013).

[3] Psycholinguistic Assessment of Language Processing in Aphasia (Bastiaanse et al., 1995).

[4] Computer Articulation Instrument (Maassen et al., in preparation).

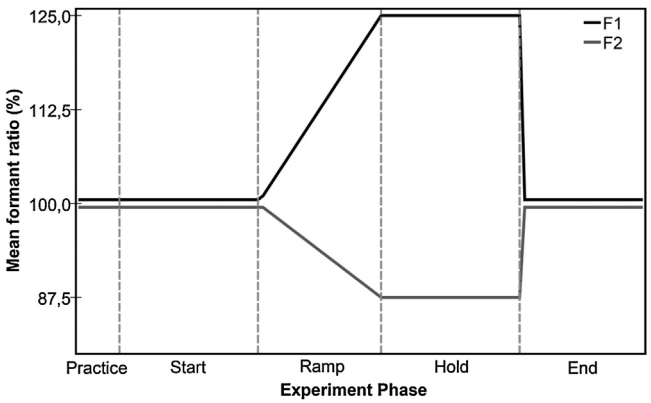


**Fig. 1.** Experimental paradigm auditory feedback perturbation. The number of trials for each phase was as follows (trial numbers of the short program are followed in parenthesis): Practice: 9 (9); Start: 27 (15); Ramp: 24 (18); Hold: 27 (18); End: 24 (15).

phases. To minimize the influence of de-adaptation during the end phase, the analysis of the end phase was limited to the first 12 trials after the perturbation was suspended.

The level of significance was set at $p < 0.05$, while $p$-values $< 0.1$ were qualified as statistical trends. Shapiro's test of normality and Levene's test of homoscedasticity were applied to the main outcome measures, prior to comparing the groups and phases by a series of statistical analyses. The results showed that both the requirements of normality and equality of variance were satisfied across all measures. On the group level, statistical analysis was done by means of Linear Mixed Model analyses separately for F1 and F2 data, with subject, phase, word, and repetition as correlated terms, and group and phase as fixed factors. Significant main and interaction effects were further explored by means of univariate tests where appropriate or a pairwise comparison using Fisher's Least Significant Difference Test. Correlations between speakers' mean normalized F1 and F2 were calculated using Pearsons's $r$. A correction to adjust for multiple statistical tests was not applied, as this creates an unacceptably high probability of making a Type II error in analyses with small group sizes (Nakagawa, 2004). Rather, multiple comparisons are accounted for in the interpretation of the results (conform e.g., Rothman, 1990; van Brenk, Terband, van Lieshout, Lowit, & Maassen, 2013).

Subsequently, individual analyses were done by means of Linear Mixed Model analyses separately for F1 and F2 data, with word, phase, and repetition as correlated terms, and phase as fixed factor. In three cases (two in the control- and one in the SSD group), the model could not reach convergence and was replaced by a simpler model with either normalized F1 or normalized F2 (two F1 and one F2) as dependent variable and phase as fixed factor. Correlations between speakers' mean F1 and F2 compensation and adaptation and their scores on selected speech/language production and perception test-scores (i.e., the particular tasks that are demanding for the auditory and/or motor systems; PCCI word and nonword repetition, word and nonword auditory discrimination and sequential fast oral motor movements) were calculated using Pearsons's $r$.

## 3. Results

### 3.1. Between- and within-group effects

Data collection of the auditory perturbation task was unsuccessful in six cases (2 normally developing children and 4 children with SSD), either because the participant was unable to successfully produce the target words within required loudness and duration limits, or because the participant was unable to finish the experiment due to fatigue or attention loss.

Fig. 2 presents normalized F1 and F2 values by group for the start, hold, and end phases. Statistical analyses revealed significant main effects of Group for F1 and F2 [F1: $F(1,542.571) = 34.820$, $p < .001$; F2: $F(1,688.428) = 6.392$, $p = .012$] and a significant Group × Phase interaction for F1 [$F(2,452.352) = 12.795$, $p < .001$]. For F2 the Group × Phase interaction failed to reach significance [$F(2,651.460) = 2.054$, $p = .129$]. No main effects of Phase were found. Furthermore, results showed a moderate correlation between speakers' mean F1 and F2 adjustments across phases [$r = -.307$, $p = .012$].

Between-group effects were further investigated for the Hold (compensation) and End (adaptation) separately, revealing significant effects of Group for F1 and F2 compensation (Hold) [F1: $F(1,333.763) = 28.614$, $p < .001$; F2: $F(1,367.032) = 6.664$, $p = .010$] and F1 adaptation (End) [$F(1,232.512) = 14.558$, $p < .001$]. For F2 adaptation however, the difference between groups failed to reach significance [$F(1,238.753) = 2.795$, $p = .117$].

Within groups, results showed respectively a significant and a trend effect of phase for the normally developing controls [F1: $F(2,346.308) = 5.197$, $p = .006$; F2: $F(2,458.877) = 2.892$, $p = .057$]. Pairwise comparisons showed that the control group successfully compensated for the perturbation (Start-Hold) for F1 [$p = .003$] and F2 [$p = .034$]. Furthermore, they showed trends of adaptation (Start-End) for F1 [$p = .064$] and F2 [$p = .075$]. The group of children with SSD on the other hand showed a significant effect of phase for F1 but not for F2 [F1: $F(2,383.640) = 7.841$, $p < .000$; F2: $F(2,358.001) = 0.396$, $p = .673$]. Pairwise comparisons indicated the differences in F1 to be significant both between Start-Hold (compensation) [$p = .001$] and Start-End (adaptation) [$p = .002$], but the results indicate that the children with SSD followed the perturbation for F1, thereby amplifying its effect.

### 3.2. Individual differences and correlations with standardized speech production and perception tests

To analyze the individual differences within groups, we tested for each participant separately whether they showed a significant effect of Phase. Within the group of normally developing children, results showed that for the perturbation of F1, 7 children compensated and adapted their formant frequencies in the direction opposite to the perturbation (of which 2 were trend effects) while 3 children followed the perturbation, 1 did not show a consistent response and 4 did not show a significant main effect. For F2, 8 of the normally developing children compensated/adapted successfully, 5 followed the perturbation, 1 was inconsistent and 1 did not show a significant main effect. Within the group of children with SSD none showed successful compensation/adaption for the perturbation of F1, 4 children followed the perturbation (1 trend) and 3 did not show a significant main effect. In the case of F2 perturbation, 3 compensated/adapted successfully, 3 followed the perturbation and 1 did not show a significant main effect. Detailed results of the statistical analyses per participant are provided in Appendix B.

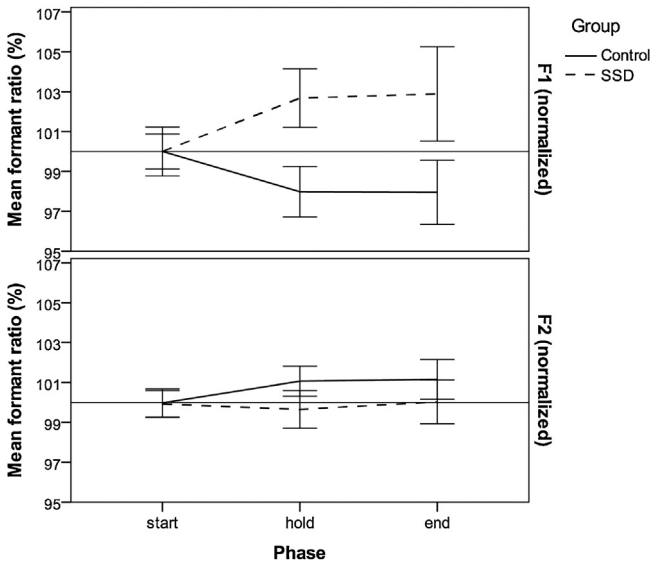


**Fig. 2.** Produced first and second formant frequencies, normalized to the mean formant values in the start phase.

To investigate how the ability to use auditory feedback to compensate for/adapt to a perturbation related to other speech motor and auditory skills, we calculated the correlations between the speakers' compensation and adaptation for the perturbation of F1 and F2 and their scores on the tasks of the speech/language production and perception tests that are especially demanding for the auditory and/or motor systems (i.e., PCCI word and nonword repetition, word and nonword auditory discrimination, and sequential fast oral motor movements). Results showed significant correlations between mean F1 and F2 compensation and the PCCI in the nonword repetition task [F1: $r = -.443$, $p = .050$; F2: $r = .508$, $p = .022$], indicating that better performance on the nonword repetition task is correlated with better compensation for the auditory feedback perturbation (Fig. 3). Correlations between mean F1 and F2 adaptation and the PCCI in the nonword repetition task were not significant [F1: $r = -.334$, $p = .149$; $r = .333$, $p = .151$]. Furthermore, no significant correlations with either PCCI in picture naming and word repetition or word and nonword auditory discrimination were found.

Oral motor movement skills were assessed in the children with SSD only; hence the correlation between compensation and adaptation for the perturbations and the score on the sequential fast task of this test was calculated over the data of this group only (Fig. 4). Results showed a significant strong correlation for compensation of F1 [$r = -.808$, $p = .028$] but the correlation with compensation of F2 failed to reach significance [$r = .655$, $p = .110$]. These correlations indicate that the children with worse sequential fast oral motor movement skills showed a stronger following/amplifying response.

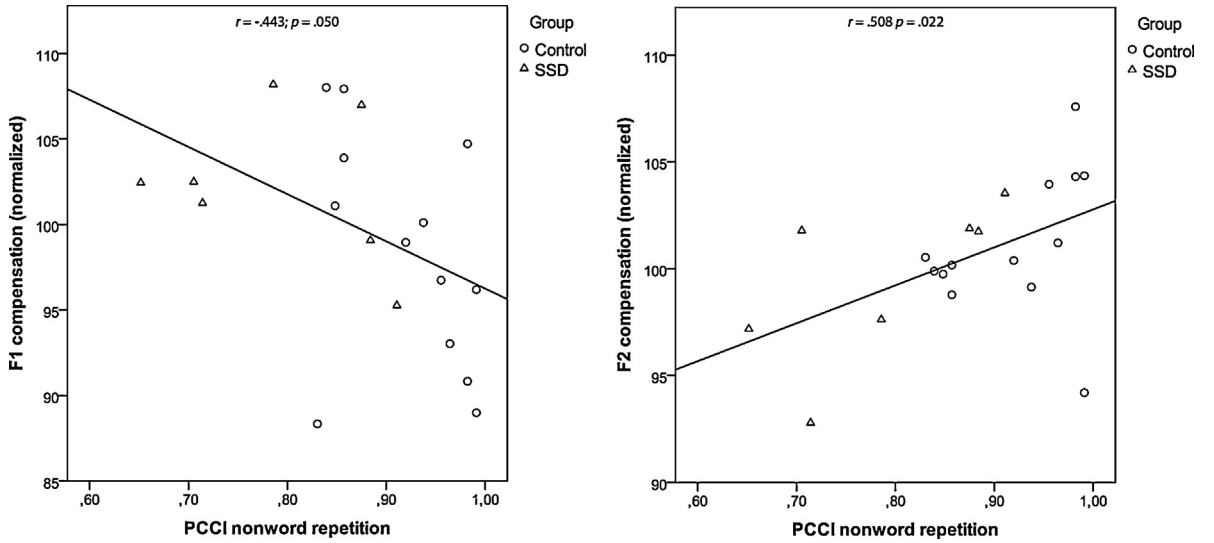


**Fig. 3.** Scatterplots of nonword repetition scores and respectively first formant (left) and second formant (right) compensation, with best linear fit.

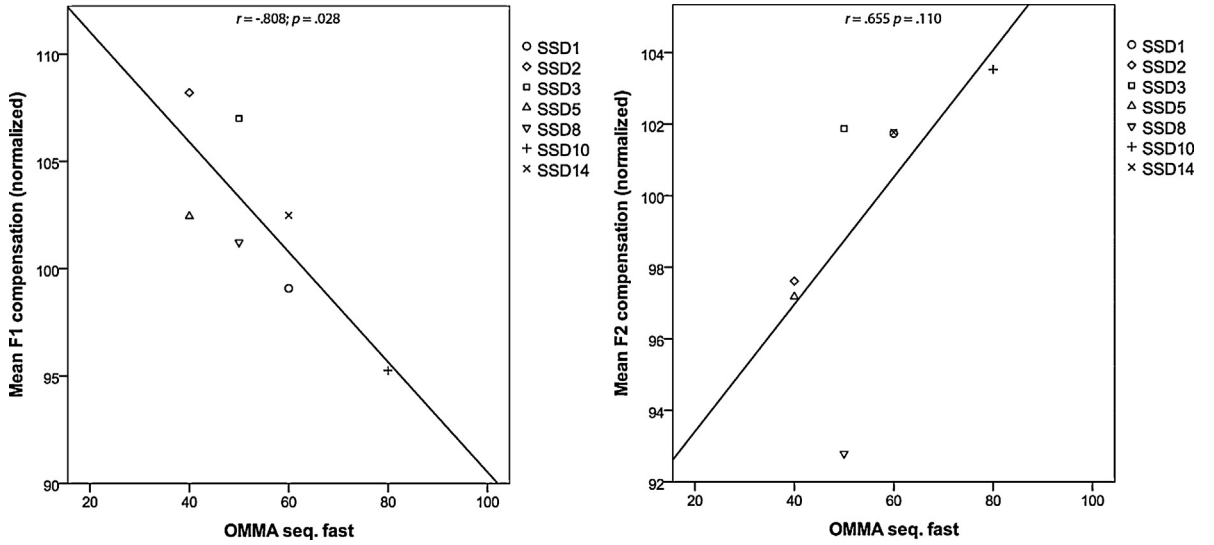


**Fig. 4.** Scatterplots of sequential fast oral motor movement scores and respectively first formant (left) and second formant (right) compensation, with best linear fit.

## 4. Discussion

### 4.1. Summary of findings

The present study investigated the ability to compensate and adapt for perturbed auditory feedback in children with SSD (age 4–8) compared to age-matched normally developing children. At the group level, the normally developing children were able to compensate and adapt but the children with SSD were not. In case of perturbation of the first formant, the normally developing children showed both compensation and adaptation, adjusting their formant frequencies in the direction opposite to the perturbation, while the group of children with SSD followed the perturbation, amplifying its effect. With respect to the second formant, the group of normally developing children successfully compensated for the perturbation and showed a trend toward adaptation, while the children with SSD failed to compensate or adapt.

Closer inspection of the individual data showed large differences between subjects. Not all participants behaved according to their group's means and results showed large differences in the amount of compensation and adaptation between individuals. Similar to what has been reported previously for healthy adults (MacDonald, Purcell, & Munhall, 2011), results showed a moderate correlation between speakers' mean F1 and F2 adjustments across phases. None of the children with SSD compensated or adapted successfully for the shifts of F1 and only three of the children with SSD compensated/adapted for the shifts in F2. For the children with SSD, results also showed strong correlations between the amount of compensation and fast sequential oral motor movement abilities. Furthermore, results showed moderate correlations between the amount of compensation and PCCI in non-word repetition, but no correlations were found with PCCI in word repetition or picture naming and not with auditory discrimination, neither for words nor for nonwords. Together, these results indicate that the speakers' ability to compensate and adapt for the perturbation of F1 and F2 is strongly correlated with their scores on the speech/language production and perception tasks that are especially strenuous on the auditory–motor system.

### 4.2. Impaired internal models

Results showed that most children with SSD could detect incongruencies in auditory feedback and could adjust their target representations, but were unable to compensate effectively. These findings suggest that impaired auditory–motor integration plays a key role in SSD and support the hypothesis of weak or underspecified internal models in children with SSD.

However, the story might be more complicated. Impairment of the internal models would be expected to prevent a consistent compensatory response, but does not unequivocally line up with the response of following/amplifying the perturbations that was found in the majority of the children with SSD. Previous studies have reported that a small minority of healthy adults followed the feedback perturbations, but have not provided an explanation why some speakers do so. The process of compensation and adaptation has been found to be relatively automatic and speakers have been found to show compensation even when explicitly instructed not to compensate for the perturbation (Munhall, MacDonald, Byrne, & Johnsrude, 2009). It is therefore highly unlikely that an amplifying response to the feedback perturbation is related to speaker-specific differences in task interpretation or differences in task execution strategies.

A possible explanation might be that the implemented formant shifts cause a "target drift", i.e., the motor system interprets the formant shifts as adjustments of the intended auditory outcome. The auditory targets are then updated accordingly and the shifted formant settings become the auditory target for the next trial. Such a process might be related to weak phonemic representations (that encode sensory targets and corresponding motor commands) and/or a large reliance on auditory feedback control. However, similar to calculating a compensatory response, the process of calculating an amplifying response relies heavily on the internal models. In the present study, we found significant correlations between the amount of compensation and PCCI in nonword repetition, a task that also requires the inverse mapping from auditory into articulatory space. A better performance on nonword repetition is correlated to a larger amount of compensation or in other words, a larger following response is correlated with worse performance on nonword repetition. Therefore, we interpret our current findings of responses following the perturbation as a problem in calculating the proper compensatory response indicating impairment of the internal models.

### 4.3. Limitations and further research

The results of this study are constrained in a number of ways. Firstly, the group of children with SSD was relatively small in numbers, and complex in symptomatology. To further test the strength of the results found in this study, future studies should include a larger number of children with clearly delineated subtypes of SSD, including a feasible number of children with Childhood Apraxia of Speech and Phonological Disorder. The presence of large within-group variations with respect to compensation and adaption both in children with SSD and normally developing children, and the fairly consistent observation of responses following the perturbation among the children with SSD, calls for future research to further investigate the detailed mechanisms of auditory–motor compensation and chart the processes that cause (or allow) the individual differences in compensatory response. One possibility would be to focus on the role of somatosensory feedback and investigate individual differences in trade-off between auditory and somatosensory feedback and whether these can be related to specific etiologies. To help pinpoint what causes the deviances in compensatory response, future

studies could use techniques of simultaneous electrophysiological recording and auditory–feedback perturbation during speech, similar to what has been proposed by Cai et al. (2012) in the context of stuttering.

### 4.4. Conclusions

The present results showed that the children with complex SSD that were included in this study could detect incongruencies in auditory feedback and could adapt their target representations, but were not able to compensate accurately. Furthermore, strong correlations were found between the amount of compensation and scores on the speech/language production and perception tasks that are especially strenuous on the auditory–motor system. These findings suggest that SSD does not involve an implicit learning deficit. Rather, they point in the direction of impaired internal models forming the core of SSD. As the internal (forward and inverse) models are prerequisites of goal directed motor learning, these findings call for research into the development of specifically targeted treatment methods. To further specify the role of auditory–motor integration in SSD, future studies that include a larger number of children with clearly delineated subtypes of SSD, including feasible numbers of children with Childhood Apraxia of Speech and Phonological Disorder, are warranted.

## Acknowledgements

This study was funded by the Netherlands Organization for Scientific Research (NWO-VENI grant 275-89-016 awarded to the first author). The authors gratefully thank the participants and their parents/caretakers for their time and effort, Margoke Nijssen for her help in data collection, and Shanqing Cai for his help with the Audapter software.

## Financial and Non-financial Disclosures

This study was funded by the Netherlands Organization for Scientific Research (NWO-VENI grant 275-89-016 awarded to the first author). The authors gratefully thank the participants and their parents/caretakers for their time and effort, Margoke Nijssen for her help in data collection, and Shanqing Cai for his help with the Audapter software. The authors have no nonfinancial relationships to disclose.

## Appendix A. Description of the diagnostic tasks.

| Task/assessment | Description |
|---|---|
| WCQ (PVVT-III-NL) | Dutch Version of The Peabody Picture Vocabulary Test of receptive vocabulary (Dunn & Dunn, 2005), with high correlations to verbal intelligence. This test is given verbally. The children were asked to point to one out of four pictures corresponding the stimulus word. Score is expressed by the Word Comprehension Quotient (WCQ; mean = 100, SD = 15). |
| Intelligibility (ICS) | The Intelligibility in Context Scale (McLeod, Harrison, & McCormack, 2013) is a quick parent report measure of children's intelligibility. The 7-item questionnaire rates the degree to which children's speech is understood by different communication partners (parents, immediate family, extended family, friends, acquaintances, teachers, and strangers) on a 5-point scale. A higher score denotes better intelligibility. |
| Auditory discrimination (PALPA) | Auditory discrimination task from the Dutch translation of the PALPA (Bastiaanse, Bosje, & Visch-Brink, 1995). Score is percentage correct. |
| Words | 36 pairs of CVC-words that were either the same (18 pairs), differed on one consonant (initial or final; 12 pairs), or were metatheses of each other (6 pairs, e.g. 'lor' vs 'rol'). |
| Non-words | 36 pairs of CVC-nonwords that were either the same (18 pairs), differed on one consonant (initial or final; 12 pairs), or were metatheses of each other (6 pairs, e.g. 'tus' vs 'sut'). |
| Diadochokinesis (CAI) | Maximum performance task using utterances of [pataka]. The children were first asked to produce 'pataka' once and when they succeeded they were asked to produce 'pataka' in a sequence of several repetitions of 'pataka'. After that the children were asked to speed up while producing a sequence of 'pataka'. This task is administered with the Computer Articulation Instrument (Maassen et al., in preparation). |

| | | |
|---|---|---|
| | PTK-score | 1 = [pataka] could be produced; 0 = [pataka] could not be produced. |
| | PTK-judgment | 4 = perfect; 3 = [pataka] in sequence in normal rate, but no acceleration; 2 = [pataka] in sequence incorrect ([t] or [k] could not be pronounced), but speeding up on two different consonants ([pata], [taka]) was possible; 1 = no fluent [pataka], not in sequence; 0 = no [pataka] production either in isolation or in a sequence of two. |
| Oral-motor movement assessment | Percentage correctly executed oral motor tasks. | |
| | Isolation | Positioning of the lips in rest; lip protrusion; lip spreading; stick out the tongue; move the tongue to corners of the mouth; move tongue up; move tongue down; click with the tongue. |
| | Sequential | Lips protrude and spread; move tongue left and right; move tongue up and down; open and close the jaw; check whether velum closes when blowing; check whether velum closes when sucking. |
| | Seq. fast | Same tasks as *sequential*, but in high tempo. |
| Picture naming (60 words CAI) | | This task consists of 60 images depicting 50 words with different consonants, consonant clusters and vowels at various positions (initial, medial, final) and ten words with complex consonant patterns. This task is administered with the Computer Articulation Instrument (Maassen et al., in preparation). |
| Word repetition (10 words CAI) | | Repetition task using the same ten words with complex consonant patterns as in *Picture naming*. The words were presented through headphones and the children were asked to repeat them. This task is administered with the Computer Articulation Instrument. |
| Non-word repetition (10 non-words CAI) | | Same task as *Word repetition*, using ten non-word stimuli with complex consonant patterns. The non-words had similar syllable structures as the words and consisted of syllables that did not exist as words in Dutch. This task is administered with the Computer Articulation Instrument. |

## Appendix B. Main effects of Phase and pairwise comparisons per participant.*

| | First formant (F1) | | | | Second formant (F2) | | | |
|---|---|---|---|---|---|---|---|---|
| Subject | Main effect ($p$) | Phase | Mean diff vs start | Post hoc ($p$) | Main effect ($p$) | Phase | Mean diff vs start | Post hoc ($p$) |
| SSD01 | 0.321 | End | 0.440 | 0.819 | 0.003 | End | −2.988 | 0.002 |
| | | Hold | 1.693 | 0.127 | | Hold | −2.405 | 0.007 |
| SSD02 | 0.000 | End | −12.468 | 0.000 | 0.001 | End | 3.617 | 0.001 |
| | | Hold | −8.297 | 0.000 | | Hold | 3.003 | 0.000 |
| SSD03 | 0.000 | End | −7.603 | 0.000 | 0.016 | End | 1.473 | 0.026 |
| | | Hold | −9.184 | 0.000 | | Hold | −0.280 | 0.536 |
| SSD05 | 0.003 | End | −1.609 | 0.506 | 0.330 | Hold | 1.568 | 0.453 |
| | | Hold | −7.849 | 0.001 | | End | 2.820 | 0.143 |
| SSD08 | 0.063 | End | −3.174 | 0.058 | 0.000 | End | 4.701 | 0.001 |
| | | Hold | −0.930 | 0.059 | | Hold | 8.644 | 0.000 |
| SSD10 | 0.215 | End | −2.099 | 0.337 | 0.000 | End | −7.342 | 0.000 |
| | | Hold | 2.476 | 0.151 | | Hold | −4.340 | 0.001 |
| SSD14 | 0.208 | End | 7.365 | 0.094 | 0.000 | End | −3.907 | 0.000 |
| | | Hold | 0.580 | 0.707 | | Hold | 0.489 | 0.578 |
| CON01 | 0.000 | End | −1.558 | 0.154 | 0.001 | End | −8.253 | 0.000 |
| | | Hold | −5.650 | 0.000 | | Hold | −5.168 | 0.008 |
| CON02 | 0.068 | End | 2.596 | 0.026 | 0.021 | End | 7.230 | 0.010 |
| | | Hold | 2.014 | 0.048 | | Hold | 1.740 | 0.078 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| CON04 | 0.005 | End | −13.903 | 0.001 | 0.001 | End | −3.675 | 0.001 |
| | | Hold | −4.506 | 0.042 | | Hold | −1.724 | 0.026 |
| CON05 | 0.001 | End | −13.083 | 0.005 | 0.329 | End | −3.156 | 0.235 |
| | | Hold | −13.163 | 0.001 | | Hold | 0.119 | 0.959 |
| CON06 | 0.040 | End | 4.843 | 0.018 | 0.000 | End | −5.928 | 0.000 |
| | | Hold | 0.565 | 0.732 | | Hold | −4.646 | 0.000 |
| CON08 | 0.001 | End | 9.919 | 0.015 | 0.001 | End | −2.788 | 0.050 |
| | | Hold | −3.903 | 0.090 | | Hold | 2.270 | 0.066 |
| CON09 | 0.001 | End | −0.385 | 0.886 | 0.000 | End | −1.164 | 0.284 |
| | | Hold | 7.736 | 0.009 | | Hold | −5.763 | 0.000 |
| CON10 | 0.000 | End | 17.573 | 0.001 | 0.001 | End | 0.186 | 0.830 |
| | | Hold | 19.853 | 0.000 | | Hold | −3.998 | 0.001 |
| CON11 | 0.010 | End | 12.275 | 0.004 | 0.000 | End | −4.370 | 0.027 |
| | | Hold | 9.167 | 0.016 | | Hold | −2.238 | 0.223 |
| CON12 | 0.791 | End | −1.218 | 0.544 | 0.001 | End | 3.396 | 0.002 |
| | | Hold | −0.105 | 0.954 | | Hold | 2.006 | 0.002 |
| CON14 | 0.649 | End | −1.074 | 0.364 | 0.005 | End | 1.623 | 0.323 |
| | | Hold | −0.282 | 0.756 | | Hold | 5.634 | 0.001 |
| CON15 | 0.108 | End | −1.693 | 0.489 | 0.000 | End | 3.035 | 0.000 |
| | | Hold | −3.589 | 0.036 | | Hold | 0.642 | 0.242 |
| CON17 | 0.000 | End | 9.551 | 0.000 | 0.000 | End | −3.685 | 0.000 |
| | | Hold | 10.347 | 0.000 | | Hold | −3.389 | 0.000 |
| CON18 | 0.090 | End | 2.087 | 0.160 | 0.000 | End | 8.988 | 0.000 |
| | | Hold | 1.387 | 0.056 | | Hold | 6.250 | 0.000 |
| CON19 | 0.119 | End | −0.297 | 0.837 | 0.000 | End | −0.250 | 0.736 |
| | | Hold | −2.773 | 0.042 | | Hold | −3.836 | 0.000 |

[*] Statistical analyses were done by a Linear Mixed Model analyses separately for F1 and F2 data, with word, phase, and repetition as correlated terms, and phase as fixed factor. In the cases CON11-F1, CON12-F1 and CLI5-F2, this model could not reach convergence and was replaced by a simpler model with phase as fixed factor.

## Appendix C. Continuing education questions

1. The role of auditory feedback in speech production is:
   a. To acquire sensory goals for producing speech sounds.
   b. To reduce cognitive demands while processing the speech signal.
   c. To serve as a guiding signal in the online control and correction of speech movements.
   d. (a) and (b).
   e. (a) and (c).
2. The general finding that a subgroup of speakers do respond to auditory feedback perturbations might be explained by the following:
   a. The experimental paradigm is too complex for some speakers.
   b. For individual speakers, perturbations remain undetected by the speech motor control system.
   c. Auditory feedback and somatosensory feedback cues are weighted differently by individual speakers.
   d. (a) and (c).
   e. (b) and (c).
3. In general, the findings with respect to compensation and adaptation for F1 in the group of children with SSD and the control group were the following:
   a. Both groups showed compensation and adaptation effects for F1.
   b. None of the groups showed compensation and adaptation effects for F1.
   c. The group of children with SSD showed compensation and adaptation effects for F1, but not the control group.
   d. The control group showed compensation and adaptation effects for F1, but not the group of children with SSD.
   e. The control group showed compensation and adaptation effects for F1, while the group of children with SSD only showed a compensation effect.

4. The relationship between both the PCCI in the nonword repetition task and mean F1 and F2 compensation can be described as followed
   a. Significant positive correlations with F1 and F2 compensation.
   b. Significant negative correlations with F1 and F2 compensation.
   c. A significant negative correlation with F1 compensation and a significant positive correlation with F2 compensation.
   d. A significant positive correlation with F1 compensation and a significant negative correlation with F2 compensation.
   e. There were no significant correlations.
5. The authors interpret the findings for the group of children with SSD with respect to auditory feedback perturbation as followed:
   a. They were unable to calculate a proper compensatory response, indicating an impairment of the feedback and feedforward internal models.
   b. They were unable to successfully produce the target words within required loudness and duration limits due to fatigue and reduced attention span.
   c. They were unable to detect incongruencies in auditory feedback and were unable to adjust their target representations.
   d. They lacked the cognitive demands to process the speech signal.
   e. The findings were related to speaker-specific differences in task interpretation or differences in task execution strategies.

## References


ASHA (2007). *Childhood Apraxia of Speech*. American Speech-Language-Hearing Association Available from: www.asha.org/policy

Bastiaanse, R., Bosje, M., & Visch-Brink, E. G. (1995). *PALPA: 1995. Dutch adaptation of Kay J, Lesser R Coltheart M. Psycholinguistic assessment of language processing in Aphasia*. Hove, UK: Lawrence Erlbaum Associates Ltd.

Boersma, P., & Weenink, D. (2013). *Praat: Doing phonetics by computer (version 5.3.42)*. Retrieved from: http://www.praat.org/

Broomfield, J., & Dodd, B. (2004). Children with speech and language disability: Caseload characteristics. *International Journal of Language and Communication Disorders, 39*, 1–22.

Cai, S., Beal, D. S., Ghosh, S. S., Tiede, M. K., Guenther, F. H., & Perkell, J. S. (2012). Weak responses to auditory feedback perturbation during articulation in persons who stutter: Evidence for abnormal auditory–motor transformation. *PLOS ONE, 7*(7), e41830.

Cai, S., Ghosh, S. S., Guenther, F. H., & Perkell, J. (2010). Adaptive auditory feedback control of the production of formant trajectories in the Mandarin triphthong /iau/ and its pattern of generalization. *Journal of the Acoustical Society of America, 128*, 2033–2048.

Campbell, T. F., Dollaghan, C. A., Rockette, H. E., Paradise, J. L., Feldman, H. M., Shriberg, L. D., et al. (2003). Risk factors for speech delay of unknown origin in 3-year-old children. *Child Development, 74*, 346–357.

Dodd, B. (Ed.). (2005). *Differental diagnosis and treatment of children with speech disorder* (2nd ed.). London: Whurr.

Dunn, L. H., & Dunn, L. M. (2005). *Peabody picture vocabulary test-III-NL [PPVT-III-NL]* (L. Schlichting, Trans.) Amsterdam, The Netherlands: Pearson.

Edwards, J., Fox, R. A., & Rogers, C. L. (2002). Final consonant discrimination in children: Effects of phonological disorder, vocabulary size, and articulatory accuracy. *Journal of Speech, Language, and Hearing Research, 45*(2), 231–242.

Groenen, P., Maassen, B., Crul, T., & Thoonen, G. (1996). The specific relation between perception and production errors for place of articulation in developmental apraxia of speech. *Journal of Speech and Hearing Research, 39*(3), 468–482.

Guenther, F. H., Hampson, M., & Johnson, D. (1998). A theoretical investigation of reference frames for the planning of speech movements. *Psychological Review, 105*(4), 611–633.

Guenther, F. H., & Perkell, J. S. (2004). A neural model of speech production and its application to studies of the role of auditory feedback in speech. In B. Maassen, R. Kent, H. F. M. Peters, P. H. H. M. van Lieshout, & W. Hulstijn (Eds.), *Speech motor control in normal and disordered speech* (pp. 29–50). Oxford, UK: Oxford University Press.

Houde, J. F., & Jordan, M. I. (2002). Sensorimotor adaptation of speech I: Compensation and adaptation. *Journal of Speech Language, and Hearing Research, 45*(2), 295–310.

Iuzzini, J., Green, J., & Hogan, T. (2014). *Effects of masking on vowel space area in Childhood Apraxia of Speech*. Paper presented at the Seventeenth Biennial Conference on Motor Speech.

Iuzzini, J., Hanen, A., Green, J., & Hogan, T. (2013). *Effect of auditory masking in children with CAS speech delay, and typical speech development*. Paper presented at the ASHA convention.

Katseff, S., Houde, J., & Johnson, K. (2012). Partial compensation for altered auditory feedback: A tradeoff with somatosensory feedback? *Language and Speech, 55*(2), 295–308.

Kent, R. D. (2004). Models of speech motor control: Implications from recent developments inneurophysiological and neurobehavioral science. In B. Maassen, R. Kent, H. F. M. Peters, P. H. H. M. van Lieshout, & W. Hulstijn (Eds.), *Speech motor control in normal and disordered speech* (pp. 1–28). Oxford: Oxford University Press.

Lametti, D. R., Nasir, S. M., & Ostry, D. J. (2012). Sensory preference in speech production revealed by simultaneous alteration of auditory and somatosensory feedback. *Journal of Neuroscience, 32*(27), 9351–9358.

Law, J., Boyle, J., Harris, F., Harkness, A., & Nye, C. (2000). Prevalence and natural history of primary speech and language delay: Findings from a systematic review of the literature. *International Journal of Language and Communication Disorders, 35*(2), 165–188.

Lewis, B. A., Freebairn, L. A., Hansen, A. J., Iyengar, S. K., & Taylor, H. G. (2004). School-age followup of children with Childhood Apraxia of Speech. *Language, Speech, and Hearing Services in Schools, 35*(2), 122–140.

Lewis, B. A., Freebairn, L. A., Hansen, A. J., Stein, C. M., Shriberg, L. D., Iyengar, S. K., et al. (2006). Dimensions of early speech sound disorders: A factor analytic study. *Journal of Communication Disorders, 39*(2), 139–157.

Maassen, B. (2002). Issues contrasting adult acquired versus developmental apraxia of speech. *Seminars in Speech and Language, 23*(4), 257–266.

Maassen, B., Groenen, P., & Crul, T. (2003). Auditory and phonetic perception of vowels in children with apraxic speech disorders. *Clinical Linguistics & Phonetics, 17*(6), 447–467.

Maassen, B., Nijland, L., & Terband, H. (2010). Developmental models of Childhood Apraxia of Speech. In B. Maassen & P. Van Lieshout (Eds.), *Speech motor control: New developments in basic and applied research* (pp. 243–258). Oxford: Oxford University Press.

Maassen, B., van Haaften, L., Diepeveen, S., de Swart, B., van der Meulen, S., Terband, H., et al. (in preparation). *Computer Articulatie-Instrument (CAI)*. Amsterdam: Boom test uitgevers..

MacDonald, E. N., Johnson, E., Forsythe, J., Plante, P., & Munhall, Kevin G.. (2012). Children's development of self-regulation in speech production. *Current Biology, 22*(2), 113–117.

MacDonald, E. N., Purcell, D. W., & Munhall, K. G. (2011). Probing the independence of formant control using altered auditory feedback. *Journal of the Acoustical Society of America, 129*(2), 955–965.
McKinnon, D. H., McLeod, S., & Reilly, S. (2007). The prevalence of stuttering, voice, and speech-sound disorders in primary school students in Australia. *Language, Speech, and Hearing Services in Schools, 38*(1), 5–15.
McLeod, S., Harrison, L. J., & McCormack, J. (2013). *Schaal voor Verstaanbaarheid in de Context*[Intelligibility in Context Scale: Dutch]. (J.C. van Doornik-van der Zee & H. Terband, Trans.). Charles Sturt University: Bathurst, NSW, Australia.
Ménard, L., Perrier, P., & Aubin, J. (2013). The role of auditory feedback in speech development: A study of compensation strategies for a lip-tube perturbation. *Proceedings of Meetings on Acoustics, 19*(1).
Ménard, L., Perrier, P., Aubin, J., Savariaux, C., & Thibeault, M. (2008). Compensation strategies for a lip-tube perturbation of French [u]: An acoustic and perceptual study of 4-year-old children. *Journal of the Acoustical Society of America, 124*(2), t1192–t1206.
Munhall, K. G., MacDonald, E. N., Byrne, S. K., & Johnsrude, I. (2009). Talkers alter vowel production in response to real-time formant perturbation even when instructed not to compensate. *Journal of the Acoustical Society of America, 125*(1), 384–390.
Nakagawa, S. (2004). A farewell to Bonferroni: The problems of low statistical power and publication bias. *Behavioral Ecology, 15*(6), 1044–1045.
Nijland, L. (2009). Speech perception in children with speech output disorders. *Clinical Linguistics & Phonetics, 23*(3), 222–239.
Perkell, J. (2012). Movement goals and feedback and feedforward control mechanisms in speech production. *Journal of Neurolinguistics, 25*(5), 382–407.
Perkell, J., Denny, M., Lane, H., Guenther, F., Matthies, M. L., Tiede, M., et al. (2007). Effects of masking noise on vowel and sibilant contrasts in normal-hearing speakers and postlingually deafened cochlear implant users. *Journal of the Acoustical Society of America, 121*(1), 505–518.
Perkell, J., Matthies, M., Lane, H., Guenther, F., Wilhelms-Tricarico, R., Wozniak, J., et al. (1997). Speech motor control: Acoustic goals, saturation effects, auditory feedback and internal models. *Speech Communication, 22*(2–3), 227–250.
Purcell, D. W., & Munhall, K. G. (2006). Adaptive control of vowel formant frequency: Evidence from real-time formant manipulation. *Journal of the Acoustical Society of America, 120*, 966–977.
Raaymakers, E. M., & Crul, T. A. (1988). Perception and production of the final /s-ts/ contrast in Dutch by misarticulating children. *Journal of Speech Language and Hearing Research, 53*(3), 262–270.
Rothman, K. J. (1990). No adjustments are needed for multiple comparisons. *Epidemiology, 1*, 43–46.
Shiller, D., Gracco, V., & Rvachew, S. (2010). Auditory–motor learning during speech production in 9–11-year-old children. *PLoS ONE, 5*(9), e12975.
Shriberg, L. D. (2010). A Neurodevelopmental framework for research in Childhood Apraxia of Speech. In B. Maassen & P. Van Lieshout (Eds.), *Speech motor control: New developments in basic and applied research* (pp. 259–270). Oxford: Oxford University Press.
Shriberg, L. D., Lohmeier, H. L., Strand, E. A., & Jakielski, K. J. (2012). Encoding, memory, and transcoding deficits in Childhood Apraxia of Speech. *Clinical Linguistics & Phonetics, 26*(5), 445–482.
Shriberg, L. D., Tomblin, J. B., & McSweeny, J. L. (1999). Prevalence of speech delay in 6-year-old children and comorbidity with language impairment. *Journal of Speech, Language, and Hearing Research, 42*(6), 1461–1481.
Smith, A., & Goffman, L. (2004). Interaction of motor and language factors in the development of speech production. In B. Maassen, R. Kent, H. F. M. Peters, P. H. H. M. van Lieshout, & W. Hulstijn (Eds.), *Speech motor control in normal and disordered speech* (pp. 225–252). Oxford, UK: Oxford University Press.
Strand, E. A., McCauley, R. J., Weigand, S. D., Stoeckel, R. E., & Baas, B. S. (2013). A motor speech assessment for children with severe speech disorders: Reliability and validity evidence. *Journal of Speech Language, and Hearing Research, 56*(2), 505–520.
Terband, H., & Maassen, B. (2010). Speech motor development in Childhood Apraxia of Speech (CAS): Generating testable hypotheses by neurocomputational modeling. *Folia Phoniatrica et Logopaedica, 62*, 134–142.
Terband, H., Maassen, B., Guenther, F. H., & Brumberg, J. (2009). Computational neural modeling of Childhood Apraxia of Speech (CAS). *Journal of Speech, Language and Hearing Research, 52*(6), 1595–1609.
Terband, H., Maassen, B., Guenther, F. H., & Brumberg, J. (2014). Auditory–motor interactions in pediatric motor speech disorders: Neurocomputational modeling of disordered development. *Journal of Communication Disorders, 47*, 17–33.
Van Borsel, J. (2009). *Basisbegrippen Logopedie. Deel 2: Communicatiestoornissen, Articulatiestoornissen*. Leuven: Uitgeverij Acco.
van Brenk, F., Terband, H., & Cai, S. (2014). *Auditory feedback perturbation in adults and children*. Paper presented at the seventeenth biennial conference on motor speech.
van Brenk, F., Terband, H., van Lieshout, P., Lowit, A., & Maassen, B. (2013). Rate-related kinematic changes in younger and older adults. *Folia Phoniatrica et Logopaedica, 65*(5), 69–77.
Villacorta, V. M., Perkell, J. S., & Guenther, F. H. (2007). Sensorimotor adaptation to feedback perturbations of vowel acoustics and its relation to perception. *Journal of the Acoustical Society of America, 122*(4), 2306–2319.