Implicit learning in dyslexics… *the cerebellum?*

Synthesizing nonsense words for the Language, Learning and the Cerebellum study

Saskia Haitjema

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Saskia Haitjema BSc

3163164

Supervisors:
Nadya Goldberg
Casper van Oers
prof.dr.F.N.K. Wijnen
prof.dr.L.J. Kappelle

Neuroscience & Cognition
Utrecht institute of Linguistics OTS
Rudolph Magnus Institute Utrecht
University Medical Center Utrecht
Utrecht University

Een jongen uit Houten Castellum
die kon niet zo heel erg goed spellum
tot de dokter die
zei: " 't is dyslexie"
zei hij: "neejoh tis mun zere bellum"
Saskia Haitjema

Taal is wat water was toen H_2O nog niet was uitgevonden,
iedereen heeft het erover maar niemand weet wat het is.
Riny Huijbregts

Waarom was ik eigenlijk geneeskunde gaan studeren?
Omdat ik achter de waarheid wilde komen;
de waarheid die nog niet in begrippen was vastgelegd
en zodoende verwaterd en uiteengerafeld was.
Alfred Döblin

I would rather have a free bottle in front of me than a prefrontal lobotomy.
Dean Martin
Beide hebben hetzelfde resultaat.
Bon Verweij

Summary

Part I

Because of the cognitive symptoms in cerebellar patients, the cerebellum is currently thought to be involved in cognitive functioning. Cerebellar loops running to and from the cerebral cortex let researchers to the hypothesis that the cerebellum could, along with the basal ganglia, be part of a subcortical pathway involved in language.

There is evidence pointing towards cerebellar dysfunction in developmental dyslexics. They exhibit deficits in balance, speed in motor tasks and procedural learning. The procedural learning deficit hypothesis states that in dyslexia, procedural learning mechanisms (via the cerebellum) fail in the process of implicitly learning phonological rules in language acquisition, leading to reduced phonological awareness and poor word recognition. These mechanisms then give way to problems in reading and spelling. However, not much research has been done into this hypothesis with the cerebellum as the key to the influence of implicit learning on developmental dyslexia.

The Language, Learning and the Cerebellum study aims to prove that there is a network in the brain including the cerebellum that is involved in language acquisition through implicit learning mechanisms. The study therefore contains two participant groups, dyslexics and patients with known cerebellar disease. Both groups will be compared to matched control groups on a neuropsychological test battery, implicit learning tasks and (for dyslexics and their controls only) a structural MRI scan. The hypotheses are that a (relative) cerebellar deficit is related to impaired implicit learning (e.g. dyslexics and cerebellar patients perform worse on both the implicit learning tasks than their controls) and that the cerebellum of dyslexics varies significantly from the cerebellum of healthy controls in volume.

Part II

The Language, Learning and the Cerebellum study consists of a variety of different tasks. Each of the tasks has to be piloted first. The implicit learning tasks that will be used in the Language, Learning and the Cerebellum study are a serial reaction time task (measuring implicit learning of motor skills) and an artificial grammar learning task (AGL). In this AGL task, participants have to listen to center-embedded sentences consisting of pseudowords and created by a phrase-structure grammar to approach human grammar as closely as possible. After listening to these 'grammatical' sentences, they will listen to grammatical as well as 'ungrammatical' sentences (that cannot be made according to the grammar) and judge whether each sentence is grammatical or

ungrammatical. Hypothesis is that they will judge right above chance, indicating they learned the grammar implicitly. For this AGL task, the sound files for the lexicon of pseudowords have to be made which will then be put into sentences according to a grammar that accounts for center-embedding. The pseudowords contain phonological and prosodic cues to address the center-embedding within the grammar and increase its learnability. In this case, phonological cues that are added are the first consonant and the vowel of each word. These cues are necessary as there are no other ways of finding out center-embedding with words that contain no meaning. Prosodic cues such as modulated pitch are added to the four-word sentences to approach prosody in center-embedded sentences in natural human language and increase learnability.

The aim of this pilot study is to create 32 sound files of nonsense words for a previously made artificial grammar and test the perceivability of their phonological and prosodic cues. Three requirements have to be met regarding the perceivability of the cues. (1) All 32 words have to be equally well intelligible. (2) All 4 variants (one for each place in the sentence) of each word have to be equally intelligible. (3) Words have to be intelligible at first as well as at non-first occurrence. 10 participants were made to listen to the 4 variants of the 32 randomly presented stimuli. They had to type the words they heard. Typed responses and accuracy (right/wrong) were obtained. Wrong responses that violated the cues of the artificial grammar language were marked as 'critical mistakes'. Analysis was performed by descriptive statistics and repeated measures ANOVA of these critical mistakes only. (1) Of the 32 words, three were perceived significantly worse than average, generating more critical mistakes (*blong, blum, trul*). (2) The number of critical mistakes of the four variants did not differ significantly. (3) Three words were perceived significantly worse than average at first occurrence (*blong, plis, trul*) and three words were perceived significantly worse than average at non-first occurrence (*blim, blong, blum*). Due to the number of critical mistakes generated by them, all four stimuli variants of *blim*, *blong*, *blum*, *plis* and *trul* will be resynthesized and tested again.

Most of the synthesized sound files of the nonsense words have proven to be highly applicable for the Artificial Grammar Learning task within the Language, Learning and the Cerebellum study.

Acknowledgements

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I also want to thank Jesse Cornelissen, who provided me with 3840 generated sequences I had already decided to write myself by hand.

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Last but not least I want to thank my parents, who were pushing me to finish this thesis (it would probably have taken me the rest of my life if not for them).

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Preface

As a student of both medicine and linguistics looking for a research internship, I got involved in the Language, Learning and the Cerebellum study of the recently established university focus area 'Neuroscience & Cognition Utrecht' in June 2010. In July 2010 I studied the topic of language, learning and the cerebellum in literature and put together a neuropsychological test battery as well as the research protocol for the study.

The topic intrigued me and I never left the study. When I was looking for a topic for my last research internship in medicine, I knew exactly what I wanted: to be part of this research group again.

Of the extensive study for which I made the research protocol, the group decided to start with a small part, regarding dyslexics and their performance on implicit learning tasks as well as a magnetic resonance imaging (MRI) study into the cerebellar volumes of dyslexics, compared to controls.

For this study I conducted a few pilot studies. I report on one of them in this thesis. For the sake of clarity, I divided the report into two sections. I sketch the background of the cerebellum, developmental dyslexia and implicit learning, as well as how everything fits together in the Language, Learning and the Cerebellum study, in part I. Part II contains the report on the pilot study.

I hope my efforts will, as a small subpart of a large project, add knowledge to the scientific corpus and eventually lead to a better understanding of the world around us.

Part I

Language, Learning and the Cerebellum,

current research and new developments

1. The Cerebellum

The human cerebellum (hindbrain) is located caudally of the cerebrum in the posterior cranial fossa (at the back of the head). It consists, much alike the cerebrum, of a cortex and cerebellar nuclei lying underneath. From the cerebellar nuclei the cerebellum connects with the rest of the brain via output to for example the red nucleus, thalamic nuclei and the reticular formation. In turn thalamic nuclei send fibers to the cerebral frontal, parietal and temporal cortices [Fabbro 2000].

Cerebellar syndromes

Early 20th century neurologists were the first to describe cerebellar dysfunction syndromes. Neurological symptoms of these syndromes were e.g. nystagmus, intentional tremor and dysdochokinesia [Fabbro 2000]. Language impairment as a result of articulation problems has also been reported, but articulation is considered a motor skill. However, late 20th century case reports of patients with cerebellar stroke report agrammatism [Silveri 1994] and anomia [Zettin 1997], symptomes that cannot be explained by loss of motor skills.

The 'dysmetria of thought' theory was the first attempt to explain these case reports by posing that not only movement but also cognitive and emotional processes are being handled in the cerebellum [Schmahmann 1991]. As a clinical counterpart of this theory, the Cerebellar Cognitive Affective Syndome (CCAS) was brought into life [Schmahmann 1998]. Dysmetria of thought or CCAS is clinically characterized by impaired executive function, spatial cognition, affective regulation and linguistic processing [Schmahmann 1998].

Cerebellum as part of the neural substrate for language

At least twenty million fibers run from the cerebral cortex to the cerebellum [Stein 1992]. Among them are fibers coming from Broca's area [Schmahmann 1991]. Apart from loops that are particularly involved in motor function (e.g. refining movement), there are two loops that are thought to be particularly involved in modulating cognitive function (and could thus be part of the neural substrate of cognitive syndromes), namely the cerebrocortico-ponto-cerebellocortico-dentato-thalamo-cerebrocortical loop (via the dentate nucleus of the cerebellum) and the cerebrocortico-rubro-olivo-neodentato-cerebrocortical loop (via the neodentate portion of the dentate nucleus) [Leiner 1993]. Both loops carry information from the cerebellar hemisphere to

the contralateral cerebral hemisphere and vice versa. They include, apart from the cerebellum and the cerebrum, the basal ganglia, which form a complex of brain nuclei at the base of the cerebrum.

In effective connectivity, one group of neurons influences the outcome (and thus the function) of another [Booth 2007]. For example one group of neurons can *refine* the information the other group is carrying. Anatomical substrates for effective connectivity in the cerebellum are found in the cerebellar loops. The cerebellum is, through those loops, thought to *regulate* information coming from the cerebrum rather than *generate* language and cognition. In other words: in the same way in which the cerebellum "regulates rate, force, rhythm and accuracy of movement, it may also control the speed, capacity, consistency and appropriateness of cognitive and linguistic processing" [Murdoch 2010]. This would account for the more severe linguistic changes after *cerebral,* rather than cerebellar, events.

Because of the lateralization of language in the brain and the fiber crossings, the right cerebellar hemisphere is thought to be involved in language processing (unlike the left cerebellar processing of visuo-spatial tasks, generated by the right cerebral hemisphere) [Fabbro 2000]. This hypothesis is confirmed by brain imaging of left-handed people whose language is lateralized to the right in the cerebrum: activation in brain imaging during language tasks in these subjects does indeed occur in the left cerebellar hemisphere [Hubrich-Ungureanu 2002]. A parallel has also been observed between improvement of right cerebellar blood flow and clinical recovery from aphasia [Marien 2007]. As a result of this compelling evidence, the crossed dominance for language is now considered a typical characteristic of brain organization [Jansen 2005].

fMRI (functional magnetic resonance imaging) and PET (positron emission tomography) studies have been used to detect activity of cerebellar areas in cognitive tasks.
The first study reporting cerebellar activity during language processing without articulation (which can easily be attributed to activation of the motor pathways within the cerebellum) was obtained using PET [Petersen 1988]. Subjects had to *think* of appropriate verbs while reading presented words (e.g. ‘to eat’ at the presentation of ‘cake’) rather than saying them aloud.

Other language tasks that, thanks to brain imaging and cerebellar stroke patients, are now known to engage the cerebellum are word finding [e.g. Baillieux 2008], prosodic segmentation [Strelnikov

2006], verb generation [Frings 2006], word association [Cook 2004], sentence construction [Cook 2004], synonym generation [Fabbro 2000], semantic discrimination [Xiang 2003], verb and noun substitution [Fabbro 2000], word completion [Silveri 1994] and rhyming [Booth 2007].

Besides that of the cerebellum, involvement of basal ganglia in linguistic tasks has also been confirmed by brain imaging methods [e.g. Booth 2007], providing evidence for a proposed IFLBC (Inferior Frontal Lobe Basal Ganglia Cerebellum) language network. Linguistic activation within these structures include left putamen (part of the basal ganglia) during phonological processing [Tettamanti 2005] and left caudate nucleus (part of the basal ganglia) in detection of syntactical and phonological anomalies [Moro 2001, Abdullaev 1997]. The cerebellum fits in by amplifying and refining (through effective connectivity) cortical activation through modulation of the basal ganglia in phonological processing [Houk 2005]. The fusiform gyrus (the bottom part of the temporal lobe) has the strongest output to the cerebellum (with no output to the putamen), demonstrating cerebellar involvement in orthographic representation of language, perhaps mapping the information into phonological output to the inferior frontal cortex or using it in the refinement process [Booth 2007].

The notion that the cerebellum, along with the basal ganglia, could be part of a subcortical pathway involved in language has led researchers to direct their attention to language disorders. One of the most intensely researched and yet incomprehensible language disorders is dyslexia.

2. Dyslexia

In 2012, *dyslexia* will celebrate its 125th birthday as a syndrome without a revealed pathophysiological mechanism. The German ophthalmologist Rudolf Berlin was the first to use the term dyslexia in 1887 [Wagner 1973]. Despite the efforts of more than a century of research the question of what dyslexia actually *is* thus remains unanswered. The 'disease' dyslexia has no 'symptoms', only a continuously changing set of 'characteristics', based on a group of non-homogeneous 'patients' (e.g. dyslexics usually do not feel ill and not all dyslexics exhibit the same characteristics of dyslexia).

Dyslexia[1] is classified as a learning disorder in the diagnostic manual of mental disorders DSM-IV [American Psychiatric Association 2000]. There are three criteria for the reading disorder that is considered 'dyslexia': (1) reading achievement is substantially below that expected given the person's chronological age, measured intelligence and age-appropriate education, (2) the disturbance significantly interferes with academic achievement or activities of daily living and (3) if a sensory deficit is present, the reading difficulties are in excess of those usually associated with it.

However, one might ask if dyslexia is purely a reading disorder, as many dyslexics also have problems with spelling (classified as a disorder of written expression in DSM-IV). One might equally take issue with the definition of dyslexia given by the World Federation of Neurology in 1968: "A disorder manifested by difficulty learning to read, despite conventional instruction, adequate intelligence and sociocultural opportunity. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin." [World Federation of Neurology in 1968]. However, this definition does give a possible *explanation* for dyslexia, namely 'constitutional origin'.

Another definition of dyslexia is provided by the International Dyslexia Association (and is adopted by the United States National Institute of Child Health and Human Development). The definition states that dyslexia is "a specific learning disability that is neurological in origin and characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding

[1] The term 'dyslexia' will be used for developmental dyslexia only.

abilities" [International Dyslexia Association 2002]. This definition does acknowledge both spelling and decoding abilities, but remains vague on the origin: 'neurological'.

The given definitions thus leave several questions unanswered. Is dyslexia a (partly) congenital condition? What neurological origin are we talking about? How severe do the difficulties have to be before one can be diagnosed? From what age can dyslexia be diagnosed? What is meant exactly by 'poor spelling' and 'decoding abilities'?

In the last decade, the prevalence of dyslexia in The Netherlands in primary school children aged 4-12 years old increased from 3.9% to 5% (5.0 to 5.4% in boys, 2.8 to 4.7% in girls). At age 11, 7.2 to 12% of children had been diagnosed with dyslexia (8.3 to 12.9% in boys, 6.1 to 11% in girls) [CBS 2008, 2011]. The data suggest that boys are diagnosed earlier (first diagnosis made at age 4) compared to girls (first diagnosis made at age 5) with a general trend between 2001 and 2010 to diagnose all children an average of one year earlier [CBS 2010]. The explanation for this rise is controversial, given the lack of unequivocal criteria for dyslexia.

From gene to syndrome

A clear pathophysiological mechanism would make definitions and diagnoses much less vague. A possible start of identifying such a pathophysiological mechanism would be finding a genetic defect. A defect was for example discovered for Down syndrome (namely the genotype trisomy 21) in 1959, long after the first doctor described the fenotype in 1838 [Wikipedia 2012]. It took scientists up to 2001 to detect a microscopic anomaly (deletion) on a chromosome (22q11) in children with velo-cardio-facial syndrome, a syndrome that had been described from 1965 on [McDonald nd]. A genetic basis for dyslexia could act as a starting point to end the definition problem and at the same time provide a clear diagnostic measure for the syndrome.

Recent Dutch research shows that 30% of children with at least one dyslexic parent develop dyslexia, while prevalence in the general population is on average 5% [Van Bergen 2012]. This finding suggests at least a familiar susceptibility for dyslexia, but does not necessarily imply a genetic basis. To confirm such a genetic basis, twin studies were performed, comparing monozygotic twins (with exactly the same genome) with dizygotic ones (that share genes like normal siblings). The twin studies confirmed a genetic basis of dyslexia; in a recent review [Scerri 2010] the concordance rate for monozygotic twins was always higher than for dizygotic twins

(1.00 vs 0.52 [Zerbin-Rüdin 1967], 0.91 vs 0.45 [Bakwin 1973] and 0.68 vs 0.38 [DeFries 1996]). However, these studies also emphasize the fact that genetics alone does not account for the presence of dyslexia, as the concordance rate for monozygotic twins was not 100% for all cases. In conclusion: genetic status can make people susceptible to dyslexia, but does not cause dyslexia.

Genetic research in dyslexics found aberrant alleles (variations of genes) on eight chromosomes: 1, 2, 3, 6, 11, 15, 18 and X [Scerri 2010]. On these chromosomes, nine different dyslexia regions have been named DYX1-9 successively. Genes located on these chromosome regions (loci) that are identified as possible causes of susceptibility for dyslexia are KIAA0319L on DYX 8 (chromosome 1), MRPL19 and C2ORF3 on DYX 3 (chromosome 2), ROBO1 on DYX 5 (chromosome 3), DCDC2 and KIAA0319 on DYX 2 (chromosome 6), DYX1C1 on DYX 1 (chromosome 15) and FMR1 and FLNA on DYX 9 (chromosome X) [Scerri 2010].

All these genes are thought to participate in brain development: they are expressed (make their proteins) in the human brain (cortex and/or basal ganglia and the cerebellum) [Scerri 2010]. KIAA0319L proteins influence the way brain cells migrate during brain development (neuronal migration). ROBO1 proteins are known to be involved in brain development and neuronal migration by the means of axonal guidance receptors, which regulate the direction and rate of neurite outgrowth (the way connections between neurons develop). DCDC2 proteins are shown to help in neuronal migration by bundling and stabilizing neuronal microtubules (small tubes that form the ever changing skeleton of a neuron). The proteins produced by KIAA039 have been associated with adhesion processes between neurons and glial cells. DYX1C1 proteins interact with estrogen receptors that have been proven to be important in synaptic plasticity (the way communication between neurons changes in response to experience) which plays an important role in learning and memory. FMR1 proteins interact with molecules that are responsible for both synaptic plasticity and neurite outgrowth and FLNA encodes a protein that modulates actin filaments (another sort of skeleton fibers) in the neuronal cytoskeleton. The exact functions of C2ORF3 and MRPL19 proteins are unknown, but both genes are expressed in the fetal and human brain and correlated with the expression of DYX1X1, DCDC2, ROBO1 and KIAA0319 [Scerri 2010].

Together with the genes PCNT (effect not known), DIP2A (neurite outgrowth, synaptic plasticity), S100B (hormonal effects on neurons and glial cells, neurite outgrowth, synaptic plasticity), PRMT2, DOCK4 (dendritic growth, branching of hippocampal neurons and cell migration) and GTF2I (brain

development, impairment leads to visuospatial construction deficits and specific language impairment) the genes discussed above appear to form a molecular signalling network in which their products interact to form signals (e.g. 'make microtubules') within and between neurons [Poelmans 2011]. Recently, three more candidate genes (VAPA, SLIT2, HMGB1) for dyslexia were suggested on the basis of this network [Poelmans 2011]. This network supports the hypothesis that dyslexia is a multigenetic neuronal migration disorder.

It turns out there is no single gene that is responsible for the susceptibility for dyslexia, all these genes most likely contribute individually to the disorder. Moreover, the observed abnormalities in the genome of dyslexics have not provided us with a clear pathophysiological mechanism for dyslexia, they rather pointed out that dyslexia is a brain disorder, which is something that scientists knew already.

From symptoms to brain

Another way to determine a pathophysiological mechanism for dyslexia is starting with the symptoms and trying to fit them into a brain 'area' that is responsible for all of them. To narrow down the exact 'location' in the brain, researchers have now collected dozens of neurological and cognitive characteristics of dyslexics.

Dyslexics have problems with reading, spelling and writing. Particularly the reading and spelling problems are assumed to result from a phonological processing problem [e.g. De Bree 2007]. Phonological processing is the way in which the brain breaks down words into the smallest building blocks; sound bits called phonemes (the smallest sound that can alter the meaning of a word). In order to do so, the processor needs to detect the phonemes in spoken language, and convert them from letters in written language (silent articulation). Dyslexics experience problems in detecting this phonemic structure of language. The phonological processor is part of the working memory [Baddely 1998]. Deficits in working memory (either the cause or the effect of failing phonological processing) [e.g. Rispens 2010] are another well known feature of dyslexia. However, observing symptoms of dyslexics more closely has recently led to a new domain of research in dyslexia: the cerebellum [Nicolson 2001].

A review article from 2009 gives the following summary of the cerebellar abberations in dyslexics [Stoodley 2009a]. Nystagmus, an example of poor eye movement (control), can be related to

cerebellar disease. Studying eye movement in dyslexics revealed a higher frequency of word fixations, longer time fixating and a greater number of regressions in English readers [Rayner 1998] although the latter was not the case for German and Italian readers [e.g. De Luca 2002]. Dyslexics also have poor binocular control [e.g. Eden 1994] and poor control of saccadic eye movements [e.g. Biscaldi 1998]. The cerebellum is known to be of great importance in balance. Impairment in balancing tasks has been reported in dyslexics [e.g. Fawcett 1999], most overt in balancing tasks with eyes open [e.g. Moe-Nilssen 2003]. The cerebellum acts as a fine tuning device in the motor system. Dyslexics are slower on peg moving [Stoodley 2006] and pointing [e.g. Velay 2002] tasks. Implicit motor learning (see also page 18) is both impaired [Howard 2006, Molinari 1997], and activates different cerebellar areas [Menghini 2006] in dyslexics as compared to control groups. To read successfully, appropriate eye movement, combined with linguistic processing, is crucial. Even Braille reading involves cerebellar areas, different from those that are active during tactile 'reading' of nonsense dots [Gizewski 2004].

Evidence of cerebellar involvement in dyslexia comes from studying cerebellar anatomy, mostly using brain imaging. Dyslexics seem to have reduced grey matter volume bilaterally for example in the anterior cerebellum [Eckert 2004] and cerebellar nuclei [Brown 2001]. Compared to controls, the cerebella of dyslexics are also more symmetric [Rae 2002], another study, however, showed a particularly small right anterior cerebellum in dyslexics [Eckert 2003]. Functional imaging reveals differences in metabolite distribution in the right cerebellum [Rae 1998] and less cerebellar activation during phonological word and pseudoword tasks [Brunswick 1999]. Also functional connectivity is severely diminished, indicating functional disruption of cerebellar loops [Stoodley 2009b].

There is thus evidence pointing towards cerebellar dysfunction in dyslexics. The only question is: how does the cerebellum influence the language disorder? The deficits in automaticity have led researchers to direct their attention to language acquisition and implicit learning.

3. Implicit Learning

One might think 'learning' solely occurs at school. Most of the knowledge gained, however, is learned completely unconsciously. The learning of unconscious knowledge is called acquisition [Carnie 2007]. Part of acquisition is even more obscure: acquisition without awareness of what has been learned afterwards. Description of the latter appeared in literature in 1967 and is called implicit learning [Reber 1967]. Deliberate, conscious learning, resulting in consciously accessible and often verbally statable knowledge is called 'explicit' (declarative) learning and is considered the opposite of this implicit (undeclarative) learning. Explicit learning and implicit learning are thought to involve different brain areas and result in different types of memory (the process by which knowledge is encoded, stored and later retrieved [Kandel 2000]). For example, implicit learning is intact in amnesia patients, while explicit learning is not [Knowlton 1996] so amnesiacs can learn things implicitly, resulting in implicit memory, without being able to recall the learning task (i.e. no explicit memory). Implicit learning can be measured by several implicit learning tasks, such as dynamic system control, probability learning and visual search in complex stimulus environments [Cleeremans 2002]. Discussed here are the serial reaction time task and the artificial grammar learning task.

Serial Reaction Time task

Since the introduction of the Serial Reaction Time (SRT) task in 1987 [Nissen 1987], the design remained largely the same. Subjects are presented with a series of visuo-spatial stimuli to which they have to respond by pressing corresponding buttons. Unbeknown to the subjects, the stimuli are presented as a repeated fixed sequence. After a few blocks of fixed sequences, reaction time to the stimuli decreases significantly. Learning of the sequence can be demonstrated by presenting a random instead of a fixed sequence; reaction time then increases again. The subjects typically remain unaware of the presence of a fixed sequence in the task, thereby indicating they have learned it *implicitly*.

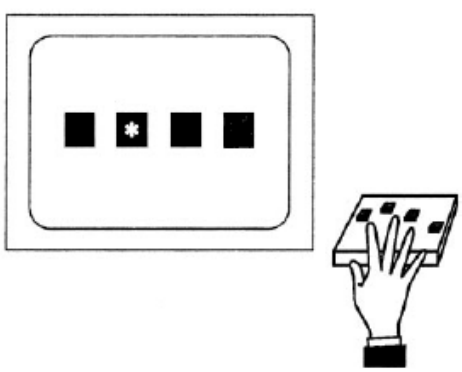

Figure 5 SRT task

(figure adapted from Doyon 2002)

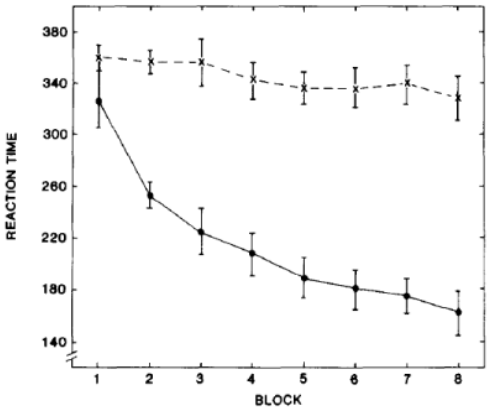

Figure 6 Example of an SRT graph
Mean of median reaction time in milliseconds
filled circles: repeating sequence, x's: random sequence
bars represent standard errors
(figure adapted from Nissen 1987)

The anatomical brain architecture that is needed to perform the SRT task, can be investigated through patients with various diseases that cause brain damage. Diseases that have been tested with SRT include Korsakoff's syndrome (KS) [e.g. Nissen 1987], Huntington's disease (HD) [e.g. Knopman 1991, Kim 2004] Parkinsons disease (PD) [e.g. Doyon 1997] and cerebellar degeneration (CD) [e.g. Molinari 1997]. KS affects the whole brain, but mainly the mammillary bodies, HD is located in the basal ganglia, PD in the substantia nigra of the midbrain and CD in the cerebellum. In all cases a decreased performance, as can be measured by reaction time, was found in the patients compared to matched controls. In one cerebellar stroke study, this only applied to the hand ipsilateral to the lesion [GomezBeldarrain 1998]. In another study cerebellar patients did show signs of learning, but only after they were declaratively primed [Molinari 1997].

Another way to show involvement of brain areas in the SRT task is brain imaging. Cerebellar involvement in the SRT task has been confirmed by PET [Matsumura 2004] and fMRI [Doyon 2002]. The cerebellum seems to be part of a network that is involved in the SRT, also including basal ganglia [Rauch1995, 1997]. An interesting fact is the seemingly early involvement of the cerebellum, suggesting that if the SRT motor skill is learned, the cerebellum is no longer needed to perform the skill [Doyon 2003]. Dyslexics have been shown to be impaired in the SRT task [Vicari 2003] and they also show diminished cerebellar activation on fMRI during the acquisition of a new sequence [Russeler 2006].

Artificial Grammar Learning task

Introduced in 1967, Artificial Grammar Learning (AGL) was the first proposed measure of implicit learning [Reber 1967]. Required for the task is some sort of artificial grammar 'machine' that can generate meaningless strings of letters for example (figure 7).

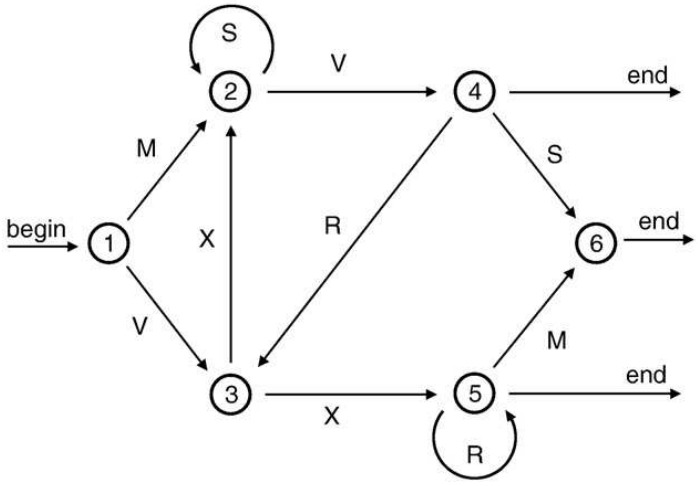

Figure 7 AGL finite artificial grammar machine (figure adapted from Floël 2009)

In the artificial grammar learning task, first these 'grammatical' strings are presented to the subjects in a so called acquisition phase, with the subjects being unaware of the set of rules that underlies the strings. In the classification phase the subjects are presented with new strings, grammatical as well as non-grammatical ones (strings that cannot be formed by the finite grammar machine) ones. They have to decide whether the new strings are grammatical or not, based on their intuition or gut feeling. Subjects are generally able to pick the grammatical strings out of the presented strings correctly above chance, but they are not able to explain their choices. This indicates they learned the rules by which the strings were formed implicitly. Implicit learning can also be demonstrated without telling the subjects about the grammaticality rules in the classification phase, simply instructing them to select the strings they 'like more'. This method is called preference classification and yields the same results as grammaticality classification [Folia 2008].

The AGL task is mostly made by a grammar that produces strings of letters (e.g. figure 7). It can however also be made in a more 'natural' way, generating strings of phrase structures consisting of words rather than letters (figure 8) [Opitz 2003].

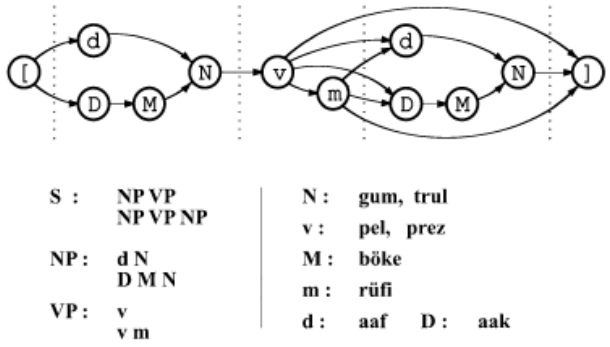

Figure 8 Language-like artificial grammar BROCANTO

S: sentence, NP: noun phrase, VP: verb phrase, N: noun, V: verb, M: adjective, m: adverb, d/D: determiner

(figure adapted from Optiz 2003)

However, BROCANTO does not account for recursion. Grammar of human languages is thought to be too complex to be represented by a finite-state grammar as it contains recursion. Recursion in grammar of human languages can be seen in center-embedded sentences. Center-embedded sentences consist of a sentence within another sentence (figure 9).

The dog that licks the child is a golden retriever.

Figure 9 Center-embedded sentence

The red sentence is embedded within the blue sentence

In a center-embedded artificial grammar, center-embedded dependencies exist within the strings. Apart from humans, some animals can also learn finite-state grammars [Fitch 2004]. However, they have not been proven to be able to learn a center-embedded artificial grammar. Current knowledge suggests the implicit learning of center-embedded structures is a distinguishing feature between humans and other species, making the appearance of it a crucial step in human evolution.

AGL was found to be impaired in children with dyslexia [Pavlidou 2010] yet another study found no impairment in adult dyslexics [Russeler 2006]. AGL seems to be intact in Parkinson's disease [Witt 2002, Lieberman 2004] and Huntington's disease [Lieberman 2004]. Performance on AGL is correlated with language acquisition [Kaufman 2010]. Not much research has been done on cerebellar activation during artificial grammar learning. Researchers focus on different parts of the brain most of the time (e.g. the occipital lobe [Thiel 2003]) or do not use the appropriate artificial

grammar learning task (e.g. asking subjects to extract the rules beforehand [Opitz 2003, Opitz 2004]). The role of the cerebellum in language acquisition, particularly artificial grammar learning, has never been investigated before with brain imaging techniques. The impact of cerebellar damage on AGL has been investigated only once [Witt 2002], but without fMRI. This study did not find evidence for impairment of implicit learning in those patients, letting the authors conclude that AGL is a cortical mediated function [Witt 2002]. However the study contained cerebellar atrophies in the vermal region rather than focal cerebellar hemispheric lesions, accounting for different damaged areas [Leggio 1995 in Marien 2001].

Implicit Learning in Language Acquisition

Clearly more research is needed into the cerebellum as the key to the influence of implicit learning on language deficits in developmental dyslexia. But what exactly is the causal pathway research needs to hypothesize on?

The human skill of language is acquired by both declarative and procedural learning. Declarative learning is responsible for the mental lexicon that is stored in the declarative memory and procedural learning is responsible for knowledge of grammatical rules that are stored in the non-declarative memory [Ullman 2004]. The procedural learning deficit hypothesis [Nicolson 2008] is a hypothesis of dyslexia that acknowledges neural networks in brain architecture, proclaiming that the procedural memory network fails. The procedural memory system in the brain comprises a cerebellar loop participating in effective connectivity including Broca's area (inferior prefrontal cortex), the basal ganglia and the cerebellum [Nadeau 1988, Ullman 2001]. The hypothesis states that in dyslexia, procedural learning mechanisms (via the cerebellum) fail in the process of implicitly learning phonological rules in language acquisition. The lack of these phonological rules leads to reduced phonological awareness and poor word recognition. These mechanisms then give way to problems in reading and spelling. It also impairs working memory because reduced phonological awareness leads to an overload of information in the phonological loop at the point that stores received information for a short period of time (phonological store) [Nicolson 2001]. However, declarative learning and memory are intact in dyslexics. The procedural learning deficit hypothesis claimes that many more developmental difficulties can be explained by failing learning systems [Nicolson 2007](figure 10).

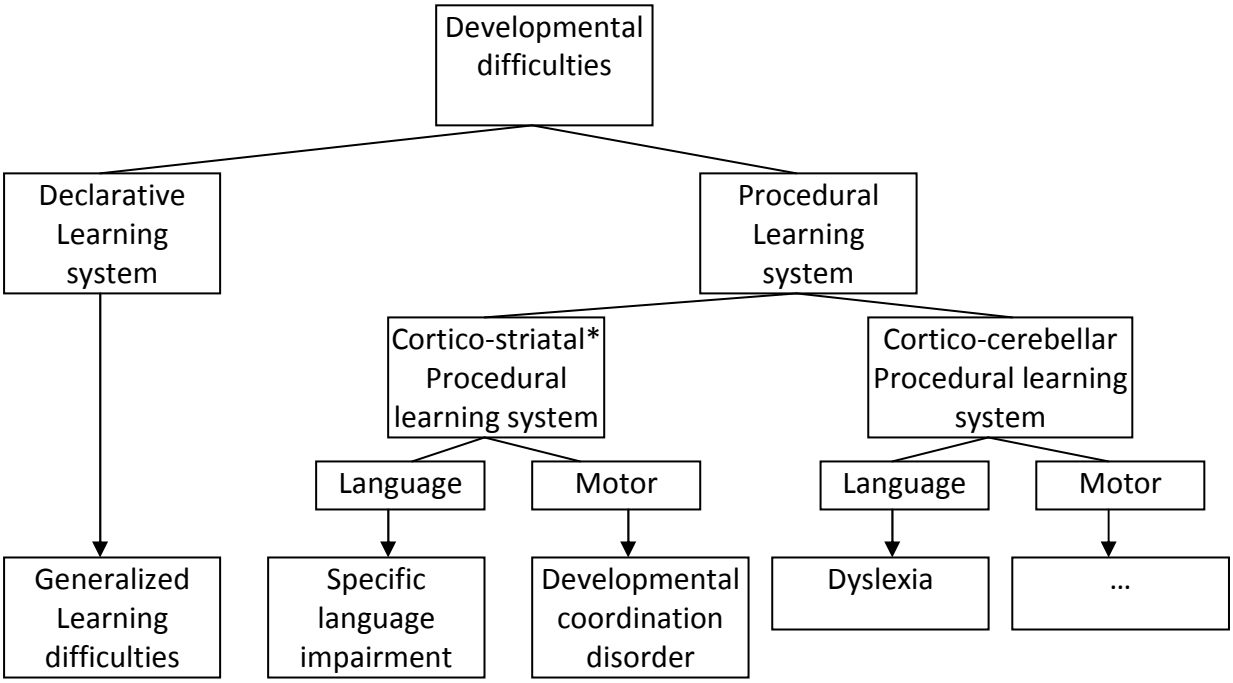

Figure 10 Neural system explanation for developmental difficulties

*** Patients suffering from specific language impairment have also shown impaired implicit learning. However, neuro-imaging revealed a different pattern from dyslexics (less striatal activation vs less cerebellar activation), giving rise to a hypothesized distinction between a dysfunctioning cortico-striatal vs a dysfunctioning cortico-cerebellar procedural learning system.**

(figure adapted from Nicolson 2007)

4. The Language, Learning and the Cerebellum study

The Language, Learning and the Cerebellum study aims to prove that there is a network in the brain including the cerebellum that is involved in language acquisition through implicit learning mechanisms.

The study therefore contains two participant groups, dyslexics and patients with known cerebellar disease (i.e. cerebellar infarction). Both groups will be compared to matched control groups on a neuropsychological test battery, implicit learning tasks and (for dyslexics and their controls only) a structural MRI scan. The hypotheses are that a (relative) cerebellar deficit is related to impaired implicit learning (e.g. dyslexics and cerebellar patients perform worse on both the implicit learning tasks than their controls) and that the cerebellum of dyslexics varies significantly from the cerebellum of healthy controls in volume. In a later stadium, fMRI will be added to the study, hopefully showing the neural network with involvement of the cerebellum in implicit learning tasks, as well as its absence in dyslexics, in 'real time'.

Part II

Synthesizing nonsense words

for the Language, Learning and the Cerebellum study

1. Place in the Language, Learning and the Cerebellum study

The Language, Learning and the Cerebellum study comprises a variety of different tasks, which have to be carefully composed. Each of the tasks has to be piloted first. The implicit learning tasks that will be used in the Language, Learning and the Cerebellum study are a serial reaction time task and an artificial grammar learning task. For the AGL, the sound files for a lexicon of words have to be made which will then be put into sequences according to a center-embedded grammar.

This pilot study functions as a preparatory study for the Language, Learning and the Cerebellum study by providing data for the AGL task that has been tested on perceivability on healthy participants.

Artificial Grammar Learning task

In the AGL task of the Language, Learning and the Cerebellum study, participants will firstly listen to grammatical strings drawn from the set of all possible strings of the artificial grammar (see below). In this phase they are asked to color in a mandala to avoid conscious rule extraction. This phase is followed by a classification phase in which the participants are presented with new grammatical as well as ungrammatical strings (strings that cannot be formed according to the grammar). They have to judge the grammaticality of the strings without receiving feedback about the correctness of the choice. Reaction time and accuracy are measured. After the classification phase participants are asked to give a confidence rating on a 7 point-scale about how sure they are about the correctness of their answers.

Question is whether the healthy participants will perform better than the dyslexics and cerebellar patients. The healthy participants are expected to judge the grammaticality of the strings right above chance and give themselves a low confidence rating, thereby indicating they learned the artificial grammar implicitly. The dyslexics and cerebellar patients are expected to judge the grammaticality at chance and give themselves a low confidence rating, thereby indicating they did not learn the artificial grammar.

Grammar

Sequences will be produced according to a grammar that generates sequences according to A^2B^2 and entails center-embedding dependencies between members of category A and category B to approach human grammar as closely as possible. The introduction of these dependencies results

into structures of type $A_1A_2B_2B_1$, $A_2A_1B_1B_2$, $A_1A_1B_1B_1$ and $A_2A_2B_2B_2$. Two specific violations, creating ungrammatical sequences within this lexicon, are category violation and dependency violation. Category can be violated by generating sequences of e.g. $A_1\mathbf{B}_2B_2B_1$. The dependencies between the categories can be violated by generating sequences of e.g. $A_1A_2B_\mathbf{1}B_1$. The sequences that are created this way will be used as 'ungrammatical' sentences in the classification phase of the AGL task.

Lexicon

The sequences contain words: the lexicon. Words were in this case monosyllabic words with a C-C-V-C (C = consonant, V = vowel) order of phonemes (e.g. *brup (*brœp)). The words were constructed according to the grammar: categories A and B were characterized by voiceless and voiced consonant in onset respectively (e.g. *b* vs *p*). Phonological cues that pointed towards the dependencies were front (1) or back (2) vowel nucleus (e.g. *o (*ɔ) vs *e (*ɛ)). Second consonants were always liquids or glides, the last consonants ones that produced a phonotactically legal coda. The first consonant in the onset and the vowel quality thus formed the 'critical' cues. These phonological cues are necessary for the participants because the grammar produces an artificial language without any other feature within the words (e.g. meaning) to distinguish the categories and subcategories of the words. The critical cues are thus necessary to be able to implicitly learn the center-embedded artificial grammar (figure 1).

A1	A2	B1	B2
plong (plɔŋ)	plem (plɛm)	blong (blɔŋ)	blen (blɛn)
plun (plœn)	plis (plɪs)	blum (blœm)	blim (blɪm)
prot (prɔt)	prel (prɛl)	brong (brɔŋ)	breg (brɛɣ)
prus (prœs)	prin (prɪn)	brup (brœp)	brig (brɪɣ)
tron (trɔn)	treng (trɛŋ)	dron (drɔn)	dweng (dwɛŋ)
trul (trœl)	trig (trɪɣ)	drus (drœs)	drit (drɪt)
twok (twɔk)	twel (twɛl)	dwot (dwɔt)	dres (drɛs)
twuk (twœk)	twik (twɪk)	dwul (dwœl)	dwis (dwɪs)

Figure 1 Words of the lexicon with their phonological representation between brackets, sorted by word subcategory

A1: voiceless onset with back vowel, A2: voiceless onset with front vowel

B1: voiced onset with back vowel, B2: voiced onset with front vowel

Prosody

An auditory presentation of the words was chosen because dyslexics have profound reading problems. To add another cue to the underlying structure, mimicked prosody of natural center-embedded sequences (fall, flat, rise, fall) was added to the sequences by manipulating the pitch of the words. Varying the duration of the last phoneme (consonant) of each word according to its place in the sequence (long, short, long, long) added a phonological cue to indicate phrase boundaries. This way, four different versions of each word were created (e.g. plong$^{\text{first place in the sentence}}$ (fall in prosody, long duration of last phoneme), plong$^{\text{second place in the sentence}}$ (flat prosody, short duration of last phoneme) etc.).

Stimuli

All of the sound files for the nonsense words were generated by the Dutch text-to-speech software 'Fluency 5.0' [Dirksen 2008]. As a starting point the duration of each stimulus part (C-C-V-C) was for each phoneme determined according to a duration time study for Dutch phonemes [Waals 1999]. The stimuli were converted into audio .wav-files by the speech synthesis algorithm MBROLA [Dutoit 1996]. Finally, pitch was manipulated by using the Dutch phonetics software PRAAT [Boersma 1992]. Pitch was determined in accordance with the place of the word in the (artificial) sentence (1st position 115-125 Hz, 2nd position 95-85 Hz, 3rd position 95-115 Hz, 4th position 136-76 Hz) based on the prosody of natural center-embedded senctences. All sounds were converted to a sample frequency of 48000 Hz.

2. Hypothesis and Aim

The aim of this pilot study is to create the sound files of nonsense words for a previously made artificial grammar and test the perceivability of their phonological and prosodic cues.
All sound files will be auditorily presented to the participants one by one. After each stimulus, the participant has to type in the word he or she heard. Reaction time and accuracy are measured.

For the stimuli to be applicable to the Language Learning and the Cerebellum study, three requirements have to be met.
(1) All 32 words have to be equally well intelligible.
The grammar cannot be detected if for example all words beginning with /d/ are perceived as beginning with /t/. Therefore all 32 words have to be equally well intelligible.
(2) All 4 variants of each word have to be equally intelligible.
The grammar cannot be detected if only the first words of each sentence are perceivable. Therefore all 4 variants of each word have to be equally intelligible.
(3) Words have to be intelligible at first as well as at non-first occurrence.
Because these words do not exist in normal Dutch language, participants have to get used to them. However, the words have to be as intelligible as possible at their first occurrence to avoid wasting time on decoding the word and missing the next stimulus and its cue.

In this case, only violations of the grammaticality cues (i.e. wrong perception of the first consonant or wrong perception of the vowel) are critical mistakes. The task does not call for a perfect score (0% mistakes) but it is important that there are no outliers. Stimuli that generate significantly more critical mistakes than average others will therefore not be used in the Language, Learning and the Cerebellum study but have to be synthesized and tested again.

The hypothesis is that the all occurrences of all words will be well intelligible and the cues highly perceivable. This makes the stimulus set highly applicable for the Language, Learning and the Cerebellum study.

3. Methodology

Participants

12 participants (7 female) were invited to the phonetics lab of UiL-OTS. Two of them were excluded from further analysis because one had already participated in a previous experiment with the same nonsense words and a second one was a student of linguistics (i.e. trained in listening to phonemes). The participants' mean age was 23.3 years (range 21-27 years). All participants were university students or had a university degree in language and culture studies (2); Dutch language (1); English language (1); medicine (2); physics (1); mathematics (1); business economy (1); or food research (1). No hearing problems were reported. All participants listened to all nonsense words. No monetary reward was given.

Procedure

All four versions of the stimuli were presented in random order to the participants one by one, using headphones and a computer with zep software (http://www.hum.uu.nl/uilots/lab/zep/) in a soundproof cabin. After each individual stimulus, the participant had to type in the word he or she had perceived and press enter. If the answer was right, a new stimulus was presented to the participant. An answer was wrong if the participant mistyped the nonsense word. If the answer was wrong, the same stimulus was presented up to three times. If the stimulus was mistyped three times, the right nonsense word was presented on the computer screen in writing, and a new stimulus was presented to the participant. Each participant thus listened and responded to a minimum of 128 (4 x 32) different stimuli.

The typed response of every trial of each participant was obtained, as well as the response score (right or wrong). Wrong responses that violated the cues that address the artificial grammar language were marked as 'critical' (i.e. critical: plong (plɔŋ) typed 'plung', non-critical: plong (plɔŋ) typed 'plon').

After the test participants were asked to point out specific difficulties in the test to the experimenter.

Analysis

Statistical analysis was carried out with SPSS 15 for Windows. First of all descriptive statistics were used to determine participant scores and word scores. The means of total participant scores were compared to look for outliers. Critical mistakes were compared between words to look for outliers. To study the difference between the four variants of each word and the difference between first and non-first occurrence, the scores on these items were compared by repeated measures ANOVA.

4. Results

Participant scores

Participants made an average of 36.3 mistakes per minimum of 128 responses (range 8 - 88; SD 21.6; figure 2), of which 18.2 were critical (range 5 - 55; SD 14.7; data not displayed). One participant (m09) made, unexpectedly, a significantly larger number of mistakes than the others and was excluded from further analysis.

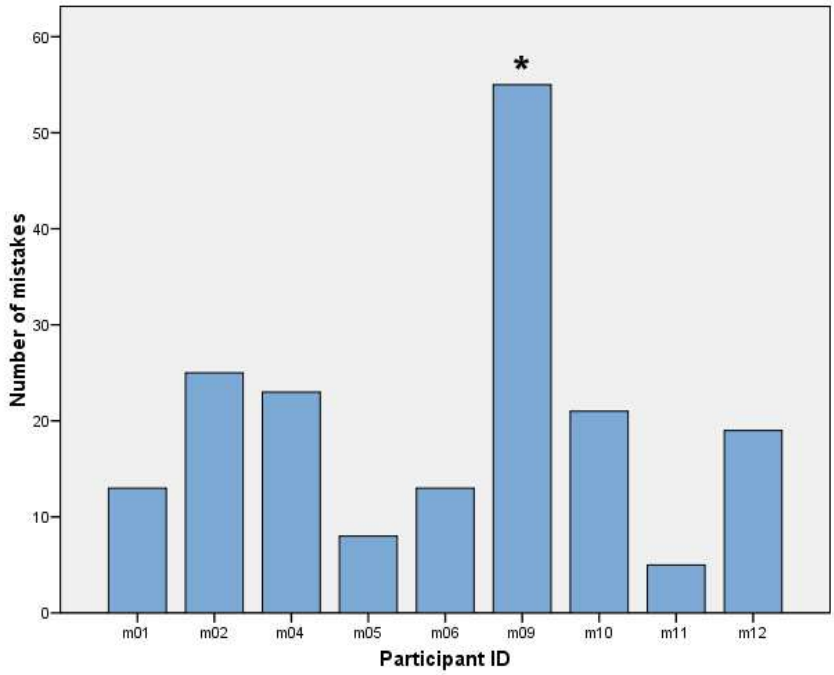

Figure 2 Number of overall mistakes per participant (* = significant)

Word scores

The words were generally typed correctly (figure 3). 6 words generated no mistake at all (*breg*, *dres*, *drus*, *prin*, *prus* and *twok*).

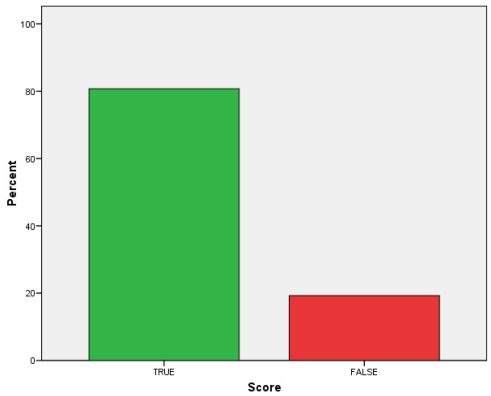

Figure 3 Overall perceivability scores for all stimuli

The average number of critical mistakes per stimulus was 4.0 (range 0 - 16; SD 4.4, figure 4). 10 words did not generate critical mistakes (*breg*, *dres*, *drit*, *drus*, *dweng*, *prin*, *prot*, *prus*, *twel*, *twok*).

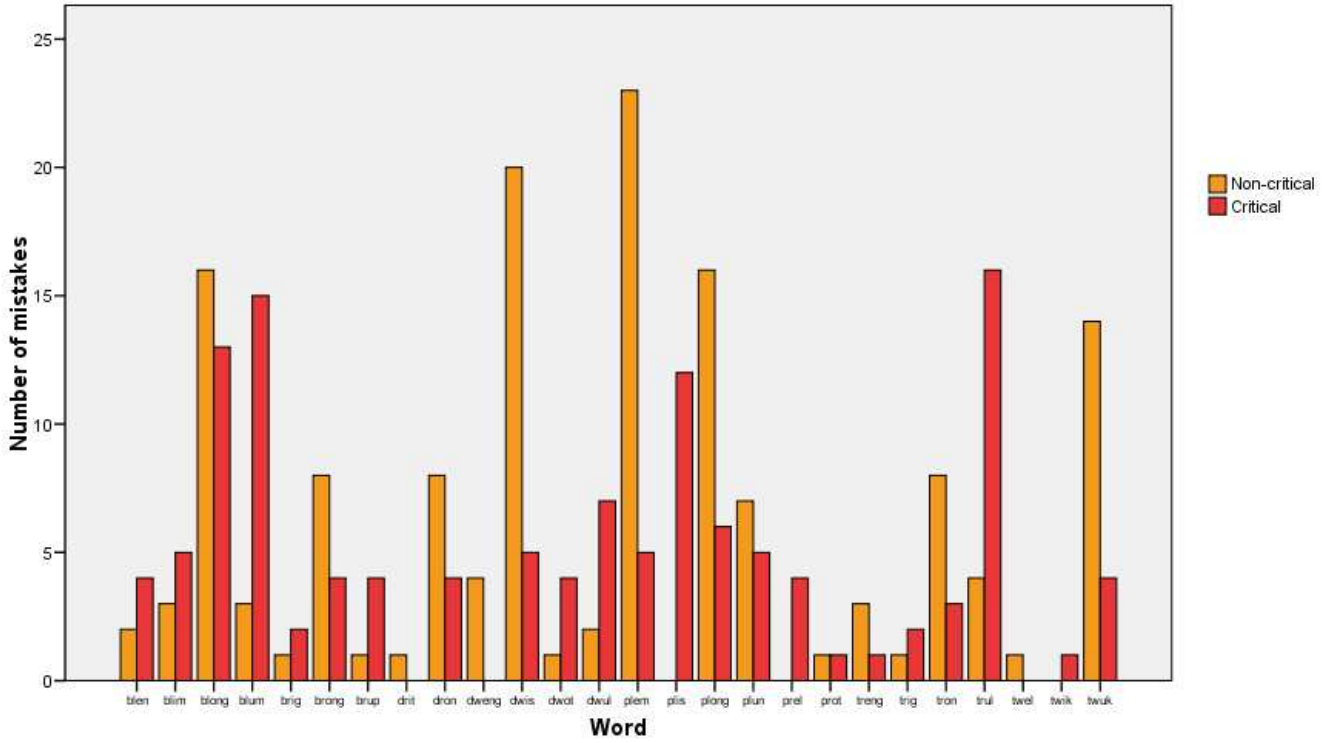

Figure 4 Number of mistakes per word

Three words were typed less correctly overall, namely *blong* (13 critical mistakes), *blum* (15 critical mistakes) and *trul* (16 critical mistakes). Mistakes in *blum* were all due to mistyping of the onset (e.g. *plum*). The majority of mistakes in *trul* were due to mistyping of the nucleus (e.g. *tril*), in *blong*, mistakes were equally distributed between onset (e.g. *plong*) and nucleus (e.g. *blan*). All critical mistakes are displayed in the Appendix.

Scores on the four variants

The mean number of critical mistakes for all four stimulus variants was 31.8 (range 21 - 39; SD 7.7). However, a repeated measures ANOVA with the independent variable 'variant' and dependent variable 'sum of critical mistakes' to compare the critical mistakes of the four variants revealed there was no significant effect of the four different variants on the number of mistakes (Wilks' Lambda = 0.459, F (3,6) = 2.355, p = 0.171, multivariate partial eta squared = 0.541, figure 5).

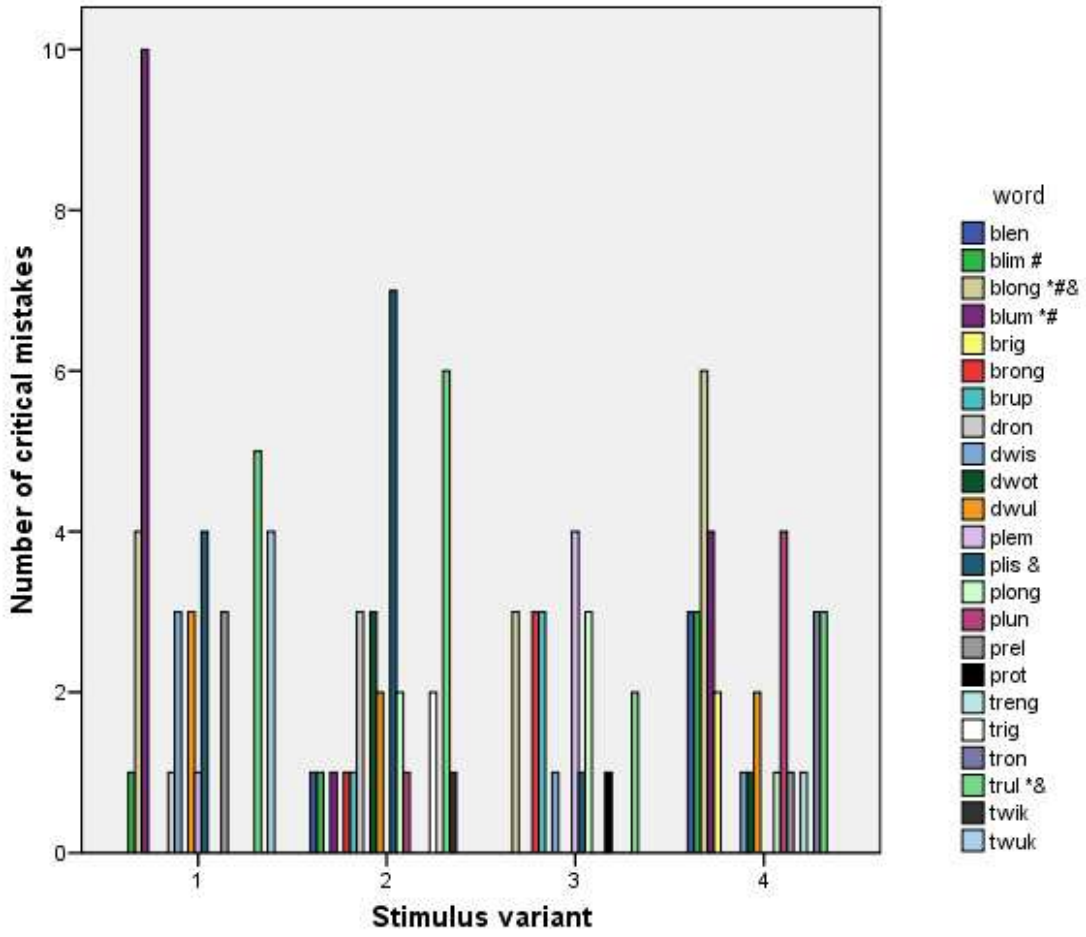

Figure 5 Number of overall critical mistakes for each stimulus, sorted by stimulus variant

(* overall significant difference, & significant difference at first occurrence, # significant difference at non-first occurrence)

Scores at first and non-first occurrence

Stimuli were typed correctly, even at first occurrence (figure 6) (i.e. the first time a participant heard and typed 'plong', 'plem', 'blong', etc.).

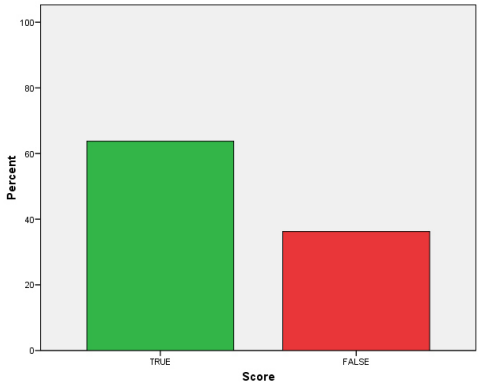

Figure 6 Scores at first occurrence of all stimuli

The mean number of critical mistakes at first occurrence per word was 2.6 (range 0-10; SD 2.7). The mean number of critical mistakes per word at non-first occurrence (either after a mistyped first occurrence or after hearning another variant of the word already) was 1.4 (range 0 - 10; SD 2.3). A repeated measures ANOVA with the independent variable 'occurrence type' and dependent variable 'sum of critical mistakes' to compare the critical mistakes made by the participants at first and non-first occurrence revealed there was a significant effect of the occurrence type on the number of mistakes (Wilks' Lambda = 0.578, F (1,8) = 5.878, p = 0.042, multivariate partial eta squared = 0.424), i.e. more critical mistakes were made at non-first occurrence.

Three words yielded significantly more transcription errors than average at first occurrence, namely *blong* (9 critical mistakes), *plis* (8 critical mistakes) and *trul* (10 critical mistakes) (figure 5). All mistakes in *plis* were due to mistyping of the onset (e.g. *blis*). The majority of mistakes in *trul* were due to mistyping of the nucleus (e.g. *trow*). Mistakes in *blong* were equally distributed between onset and nucleus (e.g. *plom* and *blan*).

Two stimuli generated a significantly different number of critical mistakes at non-first occurrence, namely *blum* (10 mistakes) and *trul* (6 mistakes) (figure 5). All critical mistakes in blum were due to mistyping of the onset (e.g. plum), in trul, mistakes in onset (e.g. drul) and nucleus (e.g. tril) were distributed evenly.

Debriefing information

When asked after completing the task, participants commented they had most trouble perceiving the coda of words ending in a nasal consonant (e.g. blu**m** was perceived as blu**ng**). However, the coda contained no grammaticality cue, and its exact spelling is therefore of minor importance. As only critical mistakes are displayed here, this mistyping is not shown in the displayed data.

5. Discussion

The aim of this pilot study was to synthesize 128 well perceivable sound files of 32 different nonsense words ('stimuli') for the Artificial Grammar Learning task that is part of the Language, Learning and the Cerebellum study. Most important features of the words were their grammaticality cues, which were the first consonant and the vowel. All 32 words had to be equally well intelligible, all four variants of each word (regarding the place in the artificial sentence) had to be equally well intelligible and words had to be equally intelligible at first as well as at non-first occurrence. The 128 sound files were tested on nine participants that had to type the pseudowords they heard.

Because the words were short and mimicked Dutch spelling, in this case mistyping will be considered as misperceiving. Critical mistakes were mistakes regarding the perceivability o the grammaticality cues. Of the 32 words, three (*blong*, *blum*, *trul*) were perceived significantly less well overall, generating significantly more critical mistakes than average. Differences between critical mistakes of the four variants were not significant. Significantly more critical mistakes were made at non-first occurrence than at first occurrence, presumably because there were at least three times as many non-first occurrences as first occurrences. Three words were perceived significantly less well than average at first occurrence (*blong*, *plis*, *trul*) and three words were perceived significantly less well than average at non-first occurrence (*blim*, *blong*, *blum*). Not all 32 words were equally intelligible regarding grammaticality cues. Due to the number of critical mistakes generated by them, all four stimuli variants of *blim*, *blong*, *blum*, *plis* and *trul* will be resynthesized and tested again.

Educational levels in this pilot study were high. However, educational levels in the Language, Learning and the Cerebellum study will also be high, because of the method of recruiting participants (through announcements in university buildings).

Most of the synthesized sound files of the nonsense words have proven to be highly applicable for the Artificial Grammar Learning task within the Language, Learning and the Cerebellum study.

6. Recommendations for the AGL task in the Language, Learning and the Cerebellum study

Recommendations for the AGL task in the Language, Learning and the Cerebellum study are firstly to take into account that the study deals with dyslexics that suffer from difficulty discriminating phonemes and slow phonological processing. This leads to ardousness in recognizing unfamiliar pseudowords. The speed of the presentation of the words and sentences in the acquisition phase of the AGL needs to be balanced. It cannot be to slow, since there is a risk of explicit learning of the grammar in non-dyslexic participants. On the other hand, it has to be slow enough for the dyslexics to be able to learn at all.

Secondly, it is higly recommended to resynthesize and test again the words that were significantly less well perceived than average in this study to prevent that the grammar cannot be learned because of unperceivable grammaticality cues.

Thirdly, the AGL task has not been validated as an implicit learning task. This calls for further piloting before integrating the AGL task within the Language, Learning and the Cerebellum study.

Lastly, in a later stadium functional MRI scans on dyslexics can be obtained to demonstrate cerebellar involvement in implicit learning. If the AGL task, as a measure of implicit learning in the language domain, will be part of this study, it could be helpful to create MRI compatible implicit learning tasks already to prevent different scores on the task on the sole basis of different task architecture.

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Appendix

Word	Number of critical mistakes	Mistakes
blen	4	plem, plen, plen, plem
blim	5	plim, plim, plim plim, din
blong	13	plom, blam, blang, blan, ban, blam, plong, lom, plong, plong, plong, plon, blan
blum	15	pnum, pnung, plum, plun, plung, plum, plum, plum, plung, plun, plun, plum, plun, plun, plum
brig	2	brieg, wrig
brong	4	plon, ron, dron, wron
brup	4	trinrup, tirnrup, rup, rup
dron	4	trong, dram, drang, dran
dwis	5	dries, drie, tris, dries, tris
dwot	4	twot, dwad, dwab, dwat
dwul	7	dweel, dwil, treel, dril, treel, dril, twil, dwil, twil
plem	5	blen, len, blem, blem, blen
plis	12	bwis, blis, blis, blis, lif, liz, blif, lis, liz, lis, lis, lis, blis
plong	6	lan, long, long, plan, blong, long
plun	5	plung, pnum, blum, blung, plung
prel	4	pril, preew, preel, pruil
treng	1	trrng
trig	2	trieg, triech
tron	3	tran, tram, tran
trul	16	ral, drul, drul, dwul, tril, tril, tro, tro, dro, tril, twril, twrel, twil, tro, trow, trou
twik	1	twiek
twuk	4	truuk, twuuk, truuk, fruk

Critical mistakes per word

(*breg*, *dres*, *drit*, *drus*, *dweng*, *prin*, *prot*, *prus*, *twel* and *twok* did not generate any critical mistakes and are therefore not displayed in the table)