Basal ganglia functions in language –
the example of Huntington's Disease

Dissertation
zur Erlangung des Doktorgrades der Philosophie
an der Karl-Franzens Universität Graz

eingereicht von
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am
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Graz, 2010
Statement of originality

The work presented in this thesis is, to the best of the author's knowledge and belief, original and the author's own work, except as acknowledged in the text. The material contained in this thesis has not been submitted, either in whole or in part, for another degree at the Karl-Franzens University of Graz or any other academic institution worldwide.
Acknowledgements

Without a lot of people this work would not have ever been completed. You all have encouraged me over the past years to complete this work. Thank you!

First of all, I'd like to thank my PhD supervisors Ao. Univ. Prof. Dr. phil. Hanspeter Gadler and Ao. Univ. Prof. Dr. phil. Annemarie Peltzer-Karpf, for academic discussion, advice in the field of brain and language, encouragement and support and especially for their precious time answering all my mails and literally thousands of questions. Thank you very much to both of you!

Furthermore I'd like to thank Univ. Prof. Dr. med. univ. Dr. phil. Hanspeter Kapfhammer for his valuable time and patience having me as an external doctorate student.

Thank you Verena Thiemann, for joining this project in the first year. I appreciate very much your help concerning the construction of this study, the language screening and your help in data collection in the first period of this project.

Sincere thanks to my former boss O. Univ. Prof. Dr. phil. Bernhard Hurch from the linguistic department of the Karl-Franzes University Graz, as well as to my dear colleagues from this institute: Dina ElZarka, Ingeborg Fink, Andrea Lackner, Veronika Mattes, Thomas Schwaiger and Ursula Stangel. Thank you to all of you for helpful suggestions and mental support in all my years of study.

Outside of linguistic department of the Karl-Franzens University Graz, special thanks to every member of the former Chorea Huntington Study Group, of the University Clinic Graz. Thank you for providing me with precious data, contacts and important information about HD. Special thanks for the sincere inclusion into the study group and for answering all my questions and mails. Thank you: Karen Hecht, Brigitte Herranhof, Anna Hödl, Rottraut Ille, Hanspeter Kapfhammer, Michael Koppitz, Daniela Kejzar, Markus Magnet, Nicole Müller, Annamaria Painold, Monika Scheibl, Helmut Schöggl and Jasmin Ullah!
Further I would like to thank Claudia Vogrincic and Thomas Wernbacher from the psychologic department of the University of Graz, for being of so much help regarding inquiries about the statistical analysis of my data. Thank you for your patience answering all my questions, also via mail!

On the other side of the world I would like to sincerely thank Prof. Bruce Murdoch for heartly welcoming me at the University of Queensland, School of Health and Rehabilitation Sciences. Thank you so much for your time reading my manuscripts and for so much helpful suggestions and advice. I think my work would not be the same without meeting you!

Furthermore, thank you my dear colleagues at the School of Health and Rehabilitation Sciences! You made my stay unforgettable: Thank you Caroline Barwood, Jo Folker, Linda Goozee, Carly Meyer, Anne Walker and Mini Wong for all your good advice and positive thinking, for your encouragement to keep on going and for being my friends. Thank you girls for all the lovely moments we have been laughing and chatting together! I miss you heaps!

Sincere thanks also to my dear ex-roomy Holly Corbett from New Zealand and my great Swiss 'copy' friend Annina Schmid who made a lot of evenings after hard work so much more enjoyable. Thank you girls for a memorable time with good food and wine, for your encouragement and for all your positive thinking!

I cannot thank enough my dear family, respectively my mum and my dad who always believed in me and supported me wherever they could throughout my life. Thank you my loves!

Thank you my friends Christina Cisek, Marlene Cisek, Carolin Gosch, Nina Kohlbach, Margit Leoni, Barbara Schwarz, Verena Thiemann, Katharina Toth, Jasmin Url, Sandra Wagner, Kerstin Weber and Anna Zaff for listening to me, for cheering me up and for being always there for me whenever I needed you!
Last but not least, I would like to say thank you to my dear fiancé, Andreas Pichlbauer. I do not even know how to express my deep thanks in a few words. You have been there when I was close to giving up, you have encouraged me to go on, but you would not have blamed me for giving up either. You know maybe the best how hard everything was for me in the past years. Thank you for being my friend and sometimes even my psychologist, for following me to Australia and for all your energy! Without you my love, I could not have made this happen. I love you!

My deepest gratitude is expressed to all HD patients and healthy volunteers who have participated in my study. Thank you so much for your time and patience!
This work is dedicated to all patients suffering from HD, to demonstrate my deepest respect how they manage life and keep on going!
Alles Wissen geht aus einem Zweifel hervor
und endet in einem Glauben.

*Maria von Ebner-Eschenbach*
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Huntington's disease

Huntington's disease (HD), also called Huntington's Chorea or Chorea Major is named after the American doctor George Huntington, who provided its first detailed description in 1872.

"Chorea is essentially a disease of the nervous system. The name „chorea“ is given to the disease on account of the dancing propensities of those who are affected by it [...]" (Huntington, 1872: 317)

HD is an inherited, terminal, neurodegenerative disease that causes psychiatric abnormalities, cognitive dysfunctions and progressive motor disturbances. Although there are several treatment strategies, currently there is no effective method to delay or prevent the onset and progression of this devastating disorder (for a comprehensive overview of HD see for instance introductions by Harper, 2002; Kosinski and Landwehrmeyer, 2005; Durr, 2007 or Walker, 2007).

1.1 Epidemiology and prevalence

Huntington's disease is one of the most common genetic disorders occurring in adult life. Apart from Finland (0.5 per 100.000) there is a rather uniform high prevalence of HD in Europe (4-7 per 100.000) (cf. Harper, 1992: 366f). Despite the fact that Austria has a rather low incidence of HD with 6-12 per 100.000 affected people (cf. Hödl & Bonelli, 2005: 9), it is one of the most common neurodegenerative diseases in this country. This could also be shown within a long-term monitoring study of the mortality trend of HD (calculated by the number of people who died with the diagnosis of HD) in Austria between 1970-2001 by Ekestern and Lebhart (cf. 2005: 17f). Globally the highest concentration of patients with HD (700 per 100.000) can be found in the Zulia region of Venezuela near Lake Maracaibo (cf. Berlit, 2006: 904).

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1 As the main focus of this thesis is to discuss language changes in HD, treatment strategies will not be addressed within this work. For a comprehensive introduction into therapeutic trials in HD see for instance Kieburtz and Shoulson (2002), Bonelli, Wenning and Kapfhammer (2004) or Bonelli and Hofmann (2006).
1.2 The origin of HD – the mutation of a gene

The HD gene was initially identified in 1983 by Gusella et al., members of the Venezuela Collaborative Huntington's Disease Project. This research group conducted a 20 year study of the world's largest family with Huntington's disease in Venezuela at Lake Maracaibo, which led to the location of the Huntington's disease gene at the tip of the human chromosome 4. After the localization of the gene it took ten more years to identify the mutation in the HD gene (also called Interesting Transcript 15 = IT15) (cf. Harper & Jones, 2002: 118), that is responsible for causing the disease. The isolation of the gene and the identification of the mutation, represents a landmark in the understanding of the disorder.

Since that time, we know that HD is an inherited disease with autosomal dominant inheritance and complete penetrance. Autosomal dominant inheritance means that an abnormal gene from one parent is capable of causing a certain disease, even though the matching gene from the other parent is normal. In other words, the abnormal gene dominates the healthy gene pair. If one parent has a dominant gene defect, each child has a 50% chance of inheriting the disorder (cf. Pritchard & Korf, 2003: 40f).

HD is further known to be completely penetrant. Penetrance describes the proportion of carrying a particular variation of a gene, e.g., if 50% of individuals carrying the HD allele develop HD, the HD allele has a 50% penetrance (cf. Pschyrembel, 2007: 1457). An example of the HD inheritance is given in Figure 1 below.

---

2 The world's largest known family with HD descended from a woman who lived in Venezuela, near Lake Maracaibo, approximately 200 years ago. Intensive research on this family began in 1981 and led to the establishment of the Venezuela Cooperative Huntington's Disease Project (cf. Gusella et al., 1983: 235).

3 Prior to the discovery of the HD gene in 1993 by The Huntington's Disease Collaborative Research Group, the diagnosis of HD could only be made by the presence of characteristic neurological symptoms together with a positive family history. The discovery of the gene has led to the development of increasingly reliable and valid molecular diagnostic tests, which identify mutations of the DNA. These tests can be applied to symptomatic patients (diagnostic testing to confirm the disease), individuals at risk for HD (presymptomatic testing) and more recently even to asymptomatic, fetuses and embryos (prenatal testing). For further information about diagnosis and genetic testing for HD and its problems and risks see for instance Folstein et al. (1986), Maat-Kievit et al. (2000), Simpson et al. (2002), Tibben (2002), Creighton et al. (2003), Meincke et al. (2003), Margolis and Ross (2003) as well as Tibben (2007).
Figure 1 shows the autosomal dominant inheritance of HD (adopted from Harper & Jones, 2002: 115)

The HD gene contains three DNA bases, cytosin, adenine and guanine (= CAG), that repeat themselves multiple times and are therefore known as trinucleotide repeats. In unaffected, healthy adults the CAG triplet is repeated approximately 20 times, whereas a number of more than 40 CAG repeats is responsible for developing the disease (cf. The Huntington's Disease Collaborative Research Group, 1993: 976).

If the child inherits the gene from the father, a phenomenon called 'anticipation' occurs (cf. Young, 1998; Duyao, 1993; Snell et al., 1993; Ranen et al., 1995), where an augmentation of CAG repeats (up to 4) occurs in the child and causes the child's age of disease onset to be higher than that of the father's (cf. Berlit, 2006: 905). Studies have reported a range of disease onsets, including age of onset between 30-50 years (cf. Cattaneo, Rigamonti & Zuccato, 2004: 60), between 35 and 50 years (cf. Haake-Weber, Förster & Cursiefen, 2005: 132) and around 45 years of age (cf. Berlit, 2005: 567).

It has been assumed that there is an inverse correlation between the number of CAG repeats and the age of disease onset; the more CAG repeats an individual has, the earlier HD manifests. This correlation has been found in studies of juvenile HD onset.

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4 Due to the fact that there are patients who have a hypothetical number of 45 CAG repeats and evidence HD's specific symptoms since 6 years, whereas there are other patients who have more than 60 CAG repeats but experience no symptoms at all (Hödl, 2009: personal communication), within clinical studies the number of CAG repeats itself is not a reliable measure for determining HD onset/progression. In contrast to this, the measure 'days since disease onset' (disease progression measure) under control of the 'number of CAG repeats' (disease onset measure) is used instead. The measure 'days since disease onset' is usually assigned by a experienced neurologist based on the patient's first symptoms. Depending on when the patient is seeing the doctor determines the score 'days since disease onset'. Thus unfortunately this score is not fully objective. Given the fact, that a patient is not seeing the doctor at the time when experiencing first symptoms, but already had the symptoms since a couple of years, symptoms are still assigned at the time when meeting the neurologist for the first time.

5 This phenomenon could be the explanation for forms of juvenile HD with a large number of CAG repeats. Evidence exists that approximately 80% of juvenile onset HD cases are inherited through the father (cf. Nance & Mayers, 2001: 154).
For instance, Nance and Myers (cf. 2001: 154) reported that an onset of HD with 20 or before, is usually associated with a repeat size of more than 60. Andrew et al. (cf. 1993: 400) described one patient with disease onset at 5, that exhibited an expansion of 121 trinucleotide repeats. Various other studies have also provided evidence of a strong inverse correlation between the number of CAG repeats and the onset of HD (Craufurd & Dodge, 1993; Snell et al., 1993; Illarioshkin et al., 1994; Kieburtz et al., 1994; Brandt et al., 1996).

Unfortunately, things have shown to be not that easy. CAG repeats per se seem not to determine age of onset and disease progression, alone. Additionally, to the length of CAG repeats, other factors, such as genetic and environmental aspects, have been suggested to influence the onset and the progression of HD (cf. Duyao et al., 1993: 391; Simpson, Davidson & Barron, 1993: 1016).

In detail, paternal versus maternal transmissions (Trottier, Biancalana & Mandel, 1994; Ranen et al., 1995) as well as interactions between expanded CAG length and the length of the normal allele (Djoussé et al., 2003) have moreover been reported to be crucial for disease onset. Similarly, Stine et al. (cf. 1993: 1547f) concluded that repeat size alone explains less than half of the variance in onset age (42%). If the number of CAG repeats is < 50, the amount of variation in the age of onset is less than 10%. Thus the onset age of individuals with 39 repeats ranges from 30 to 65 years of age. That repeat size alone is perhaps a poor predictor of onset age could moreover be approved by Myers (cf. 2004: 257), as individuals with 44 CAG repeats exhibited onset of HD as young as 31 or as old as 66 years.

Langbehn and colleagues (2004) suspected that also environmental and additional unknown genetic factors contribute to the onset of HD. They pointed out that the contribution of genetic factors as well as environmental modifiers are less obvious in individuals with higher repeat size (such for instance 44), due to the major impact of ployglutamat length. Modifiers might play a greater role, if the CAG repeat size is less than for instance 40 (cf. Langbehn et al., 2004: 275).

Additionally to the correlation between CAG repeat length and the age of disease onset, the association of CAG repeat length with the progression of HD symptoms has been studied. While no significant relationship between the age of onset of HD and the rate of disease progression has been reported (p > 0.05) (cf. Kieburtz et al., 1994: 873), the length of the CAG repeats correlated highly with the age of disease onset (p < 0.001) in the study by Brandt et al. (1996). In detail, patients with long CAG repeats (47 or more),
showed more rapid decline in neurological and cognitive abilities over two years of study, compared to patients with short expansions of trinucleotide repeats (37-46 repeats) (cf. Brandt et al., 1996: 527). A significant positive correlation between the CAG repeat length and and the rate of progression of neurological (p < 0.001) and psychiatric features (p < 0.001) has further been demonstrated by the study of Illarioshkin et al. (cf. 1994: 631). Rosenblatt et al. (2006) revealed further that CAG repeat length is statistically significant (p < 0.01) associated with the rate of disease progression in HD patients and thus patients with the shortest expansions, appear to have the best prognosis in regards to disease progression.

In conclusion, we do not know for sure by now what determines the onset and the progression of HD. Unfortunately, we do not know either what is responsible for the specific mutation in the gene, noted in HD patients. This latter question will be addressed next.

1.3 What is responsible for HD's pathogenesis? – mouse models give insight

Currently it is not known which neurodegenerative mechanisms are causing the core symptoms seen in HD. A breakthrough in HD research was the development of transgenic mouse models (Mangiarini et al., 1996; Davies et al., 1997). Mangiarini et al. (1996) as well as Davies et al. (1997) found that in transgenic mice with a CAG repeat expansion ranging from 115-150 repeat units, the development of a progressive neurological phenotype occurred, similar to that seen in juvenile HD, in humans. In the brains of these transgenic mice, polyglutaminic aggregates, predominantly located in the nuclei of cells (neuronal nuclear inclusions), have been found. It could be revealed that these aggregates contained huntingtin⁶. After Davies et al. (1997) these aggregates have been widely accepted as a key feature of HD and other CAG triplet disorders.

Numerous additional new models have since been created (see for instance 'Modeling Huntington's Disease in cells, flies and mice' by Sipione and Cattaneo, 2001) and each has provided new information about the disease. These models allow the detection of

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⁶ The HD gene, which is defect in HD, contains three DNA bases: cytosin, adenine and guanine (=
CAG), as previously mentioned. These nucleotides form a chain of amino acid glutamat which further constitutes the protein huntingtin (cf. The Huntington's Disease Collaborative Research Group, 1993: 976).
the earliest manifestation of the disease, and further are able to show the behavioral, neuropathological and even electrophysiological factors associated with the disorder. Additionally, these models have made it possible to test different pharmacological approaches used to delay the onset or slow the progression of HD in mice (For further information about mouse models in HD see for example Kosinski et al., 1999; Menalled and Chesselet, 2002; Rubinsztein, 2002 as well as Levine et al., 2004). Mouse models can thus be seen as being crucial for the establishment of future therapeutic investigations.

Based on results of mouse models, as described above, huntingtin can be considered a crucial factor for the development of HD specific symptoms. Nevertheless, some questions must be addressed concerning huntingtin in HD: a.) where is huntingtin expressed in the human brain?, b) what is its function in normal brains?, and c) how does it contribute to the specific pathogenesis seen in Huntington's disease in humans?. These questions will be addressed in the following subchapters.

1.3.1 Where can huntingtin be found?

Di Figlia et al. (cf. 1995: 1076f) as well as Young (cf. 2003: 299) revealed that huntingtin is widely expressed in neurons throughout the brain. More specifically, huntingtin could be found in the cortex, the cerebellum as well as within cells of the caudate nucleus and the cytoplasm of cell bodies, in dendrites, axones, and even in vesicle membranes, but not in the cellular nuclei (cf. Sharp et al., 1995: 1069f). Bhide et al. (cf. 1996: 5529) reported that huntingtin is even expressed in the developing brains of human fetus. Immunocytochemistry analysis of normal human fetal brains has revealed an expression of huntingtin in the frontal cortex, the parietal cortex, the occipital cortex, the thalamus, the striatum, the pons and the cerebellum. Based on these findings White et al. (cf. 1997: 407) demonstrated that during embryogenesis in HD mice, a reduction of huntingtin to below 50% resulted in reduced neurogenesis and profound malformations of the cortex and the striatum.
1.3.2 What is the function of huntingtin?

The main function of huntingtin remains puzzling. Various competing theories have been developed, that will be discussed briefly now.

The association of huntingtin with vesicle membranes suggests that this protein may participate in intracellular transport mechanisms and enhance or support vesicular transport (cf. Di Figlia et al., 1995: 1079). An association of huntingtin with vesicle trafficking has also been stated by Velier et al. (1998). Furthermore, Smith, Brundin and Li (2005) emphasised, that huntingtin might be involved in the cell machinery that controls synaptic transmissions. The findings of White et al. (cf. 1997: 407) may indicate that huntingtin participates in the formation of the brain. Thus in general, huntingtin seems to be necessary for developing and sustaining normal brain functions (cf. Young, 2003: 301). Moreover, huntingtin is believed to have a neuroprotective function and prevents cells from dying by inhibiting caspases, which are the most important enzymes of apoptose\(^7\) (Rigamonti et al., 2001). Cattaneo, Zuccato and Tartari (cf. 2005: 923f) stated that huntingtin also seems to control the production of the BDNF\(^8\). Only normal huntingtin, but not the mutant protein has been reported to stimulate the BDNF production. Thus, the reduction or even the loss of the normal function of huntingtin might decrease BDNF-support of striatal targets and may therefore be crucial for the establishment of HD. This theory is in accordance to the assumption by Gauthier et al. (2004).

1.3.3 Theories about HD's pathogenesis

Controversy remains in the literature in regards to the ontogenesis of HD. Several competing theories exist, that will be summarised as follows.

One theory concerning the ontogenesis of HD suggests that an increased number of CAG repeats causes the protein huntingtin to mutate and to become toxic (cf. Sharp & Ross, 1996: 6; Cattaneo et al., 2001: 183; Bates, 2003: 1643; Cattaneo, 2003: 34).

\(^7\) For more information about 'Apoptosis and Caspases in Neurodegenerative Diseases', such as HD, see for instance Friedlander (2003).

\(^8\) BDNF is the abbreviation for brain derived neurotrophic factor, a protein, which supports the survival of neurons, especially striatal neurons and controls the activity of cortico-striatal synapses (see for instance Widmer & Hefti, 1994: 1669).
Thus, huntingtin's hypothetised normal function in for instance intracellular transport, as described above in chapter 1.3.2, is assumed to be altered. Consecutively toxic huntingtin is believed to form intracellular polyglutamine aggregates that manifest as nuclear inclusions in special areas of the brain which lead to neuronal death, as seen in the course of HD (Ross, 1997; Cattaneo, 2003; Allsopp, 2000).

There is also compelling evidence that in contrast to a 'toxic gain of function' (cf. Sharp & Ross, 1996: 6; Cattaneo et al., 2001: 183; Bates, 2003: 1643; Cattaneo, 2003: 34), a loss of the normal function of huntingtin could be a possible mechanism in the development of the disease (Cattaneo et al., 2001). Given, that huntingtin has a core role in neurogenesis (Bhide et al., 1996; White et al., 1997), a loss of this function may potentially cause a change in the morphology of the neurons and/or glia cells in HD brains, leading to dysfunction and death (cf. Nopoulos et al., 2007: 1432).

The main concern with these two theories is that healthy huntingtin as well as mutant huntingtin have been reported to be heterogenously distributed throughout different areas of the (HD) brain (for details see also chapter 1.3.1). As for the ontogenesis of HD a specific neuropathology in mainly striatal regions of the brain has been assumed, it is doubtful that huntingtin is the only factor responsible for the development of HD. Becher et al. (1998) reported that these neurons with the most intracellular inclusions were found in the neocortex, specifically in the frontal, temporal, parietal, occipital, primary motor and insular cortical neurons, as well as in the striatum within caudate and putaminal neurons. Inclusions could be identified to a lower degree in the amygdala, hippocampus, and cerebellum but could not be found in the thalamus, substantia nigra, olivary complex in the medulla oblongata or cerebellar Purkinje cells (cf. Becher et al., 1998: 391f). Gourfinkel-An et al. (cf. 1997: 717) showed a similar distribution of mutated and normal huntingtin throughout the brain. Moreover, Gutekunst et al. (cf. 1999: 2526) pointed out that aggregates were primarily observed in gray matter and were infrequent in white matter. Specifically, more aggregates could be found in the cortex than in the striatum, substantia nigra, hypothalamic nuclei, thalamus, or brainstem nuclei such as the nucleus cuneatus. Aggregates were also rare in the hippocampus and cerebellum.

By demonstrating that aggregates could only be found in 4% of the striatal neurons in a total of 12 HD brains (cf. Kuemmerle et al., 1999: 845), Kuemmerle and colleagues (1999) as well as Gutekunst et al. (1999) concluded that huntingtin aggregates may not
predict HD. Thus, it can be summarised that the formation of polyglutamine aggregates alone cannot explain – if at all – specific cell death mechanisms in the course of HD. Other mechanisms must be involved in the neurodegeneration of HD.

Several alternative cell death hypothesis will be presented now, as summarised from the literature. Ferrante, Kowall and Richardson (cf. 1991: 3884) for instance indicated that proliferative rather than degenerative mechanisms might contribute to cell death mechanisms in HD. Specifically, newly formed dendritic arbors and an increased number of dendritic spines have been reported, which may facilitate neuronal excitation and thus lead to HD's hyperkinetic symptoms. Beal et al. (1991), Bazzett et al. (1993) as well as Ferrante et al. (1993) also supported the assumption by Ferrante, Kowall and Richardson (1991), that excitotoxicity is the crucial mechanism of cell death in HD. Subsequently it has been suggested that impaired energy metabolism could underlie excitotoxic neuronal death in HD (Beal, 1992). Similarly, Shin et al. (2005) suggested that an expression of mutant huntingtin in glia cells may contribute to neuronal excitotoxicity, as these cells normally influence transmitter transport, such as the uptake of the excitatory neurotransmitter glutamate. Jakel and Maragos (2000) however indicated that under specific circumstances dopamine, a transmitter which occurs in high concentration in the basal ganglia, might become neurotoxic and is therefore responsible for neuronal cell death in HD. Sugars and Rubinsztein (2003) concluded that mutant huntingtin might impair gene transcription (transcription of DNA into messenger RNA is dysregulated), which could thus also lead to the specific HD pathogenesis. Landles and Bates (2004) summarised that impaired protein misfolding, impairment in intracellular transport, mitochondrial dysfunction and defective synaptic transmission are involved in the pathogenic mechanisms that contribute to HD. Ciammola et al. (2007) suggested that huntingtin mutation causes the BDNF production to decline and thus leading to neurodegeneration in HD patients.

In conclusion, it remains unclear what the crucial factors in the development of symptoms in HD are. Supposedly it is the sum of factors that contribute cumulatively to the origin of this devastating disorder. It is certain however, that in HD there is a specific change in brain morphology. How HD brains change, which areas of the brain are most affected, and how these changes contribute negatively to HD's symptomatology, will be discussed in the following chapter.

9 The excitotoxicity hypothesis of HD suggests that the selective striatal neuronal loss results from an excessive activation of excitatory neurotransmitters such as glutamate, that is expressed in huntingtin. Thus it is suggested that excitotoxicity is involved in HD (cf. Bazzett et al., 1993: 177).
1.4 Neuropathological changes in HD

The earliest and most striking neuropathological changes, associated with HD, have been reported to be in the striatum (see Picture 1 below) (Vonsattel et al., 1985).

Picture 1 shows the basal ganglia and associated nuclei (from Bear, Connors & Paradiso, 2007: 465)

On a macroscopic level, Vonsattel and DiFiglia (cf. 1998: 371) as well as Vonsattel (cf. 1999: 162) reported a bilateral symmetric atrophy of the striatum in 95% of 1000 tested HD patients. Based upon the neuropathological evaluation of 159 HD post mortem brains, Vonsattel et al. (1985) proposed a five point scale (0-4) for grading the degree of neuropathological involvement (macroscopically as well as microscopically) of the striatum in HD, which is still used today (Vonsattel, 2007). Within this scale the following five grades of striatal degeneration can be distinguished from each other:

1) At grade 0, no abnormal macro or microscopic neurological characteristic of HD can be identified.
2) At grade 1 no macroscopic abnormality can be observed, but microscopic examination reveals mild neuronal cell loss, particularly in the medial half of the head of the caudate nucleus and to a lesser extent in the dorsal region of the putamen.
3) Grade 2 indicates macroscopic evidence of an atrophy in the head of the caudate nucleus, but a convex outline at the lateral ventricle is retained. Microscopically a mild loss of neurons is present in the medial portion of the caudate nucleus and the dorsal region of the putamen.
4) In grade 3 macroscopically, the nucleus caudatus is shrunken to half of its normal thickness, thus the head of the caudate nucleus forms a straight line. The putamen is moderately shrunken. These macroscopical changes consecutively lead to an enlargement of the anterior horn of the lateral ventricle. Microscopically, neuronal loss is more diffuse throughout the medial sections of the caudate nucleus; but only moderately severe in the lateral half of the caudate nucleus. Severe neuronal loss is also found in the superior half of the putamen.

5) In grade 4 the caudate nucleus presents a medially concave, severely shrunken appearance. The putamen and the internal capsule are also atrophic. Nerve cell loss is very severe and diffuse throughout the caudate nucleus and the putamen.

Striatal degeneration appears to have an ordered and topographic distribution. Specifically, the tail of the caudate nucleus shows more degeneration than the body, which in turn seems to be more involved than the head. Similarly, the caudal portion of the putamen is more degenerated than the rostral portion. As the disease progresses, striatal degeneration moves in caudo-rostral as well as in dorso-ventral/medio-lateral direction (cf. Vonsattel & DiFiglia, 1998: 371ff; Vonsattel, 1999: 161ff).

The atrophy of neurons in the striatum leads to a widening of sulci as well as to a shrinkening of gyri and to an enlargement of the lateral ventricles. As the disease worsens an overall atrophy of the brain is evident (cf. Ross & Margolis, 2001: 144; Vonsattel, 1999: 162). Thus, it is not surprising that HD brains are smaller than normal brains and thus also weigh less. This assumpton was supported by De La Monte, Vonsattel and Richardson (cf. 1988: 522) who demonstrated a 60% reduction in brain weight in the cross sectional area of the nucleus caudatus, the putamen and the globus pallidus, in 30 HD brains studied.
Picture 2 shows a MRI scan of a healthy control, versus the diffuse neuropathological changes with widening of ventricles and massive atrophy in the striatum and the whole brain of a patient suffering from late stage HD (unpublished data from the University Clinic Graz, Department of Psychiatry).

In addition to an atrophy within the striatum, also significantly increased BOLD responses (blood oxygen level dependent) could be reported for HD patients (Clark, Lai & Deckel, 2002). Paulsen et al. (2004) measured significantly less activation in subcortical structures, including the nucleus caudatus, on fMRI, even in presymptomatic HD patients. Reduced striatal blood flow, measured by SPECT, has additionally been reported by Harris et al. (1999), again even for presymptomatic HD patients. A PET study by Ciarmiello et al. (cf. 2006: 219) further evidenced severe hypometabolism in basal ganglia areas in preclinical HD patients. From these studies it can be summarised that various pathologic alterations in HD brains occur sometimes even years before the first symptoms manifest. Specifically, Aylward et al. (cf. 2004: 70) reported that the putamen, as well as the caudate volumes are severely reduced in individuals with CAG repeat expansions, even 9 to 20 years prior to the onset of a diagnosable HD. Presymptomatic neuropathological changes in HD brains have also been reported in numerous studies including for instance Aylward et al. (1996), Harris et al., (1999), Aylward et al. (2000), Thieben et al. (2002), Aylward et al. (2003) as well as Paulsen et al. (2006). As the caudate volume has been reported to decrease with increasing clinical severity, Aylward et al. (cf. 2004: 71) concluded that the caudate volume could possibly be used as an outcome measure in clinics for HD patients. Furthermore, Paulsen et al. (2004) suggested fMRI as a useful tool to identify early caudal degenerations, before the onset of clinical symptoms\(^\text{10}\).

\(^{10}\) Further, as already briefly addressed within chapter 1.3.3, as well as for instance stated by Neal et al. (1994) and Penney et al. (1997), there is a strong inverse correlation between the clinical onset of HD
As shown above, HD's neuropathology is characterised mainly by striatal changes. Nevertheless, to a minor extent, neuropathological changes have been reported for several other brain regions, even outside the subcortical area. Specifically, widespread neuronal degeneration, including the cerebellum, thalamus, hippocampus, brainstem, cerebral white matter has been reported to occur even in early to mid-stages of HD (Rosas et al., 2003). Furthermore, the substantia nigra, hypothalamus, amygdala, insular cortex as well as premotor and sensorimotor cortices have been mentioned to degenerate in presymptomatic HD patients (Douaud et al., 2006). Even within stage I and stage II of HD significant thinning of brain tissue in the sensorimotor, parietal, posterior superior and middle frontal, entorhinal, precuneus, cuneus and occipital cortical areas could be found (cf. Rosas et al., 2008a: 1059; Rosas et al., 2008b: 200). Significant abnormalities outside the striatal area have further been detected within the thalamic region as well as in mesial temporal lobe structures (Jernigan et al., 1991). Distributed grey matter pathology and progressive white matter atrophy even before clinical onset of HD could additionally be reported in the study by Thieben et al. (2002), Henely et al. (2006), Rosas et al. (2006) as well as by Squitieri et al. (2009). As exemplified within this chapter, in regards to neuropathological changes in HD, much still remains unanswered. Nevertheless, there is evidence that neuropathological changes do arise even years before the manifestation of the first symptoms and occur in almost every part of the brain, including the cortex, the basal ganglia and other subcortical structures such as the thalamus.

Contrasting localism 11 (Gall & Spurzheim, 1809), it is hypothesized that all brain areas affected, are highly interconnected with each other in terms of networks (see for instance Mesulam, 1990), or circuits such as the cortico-basal ganglia-thalamo-cortical circuits stated for the first time by Alexander, DeLong and Strick, back in 1986. Based on these considerations the assumption arised that atrophies in various brain structures involved in these circuits lead to disruptions in the neuronal loops and thus contribute to the development of the neuropsychiatric, neuropsychological, and the severity of neuropathological involvement. An earlier clinical HD onset is thus assumed to be associated with a more severe neuropathological change (Myers et al., 1985; 1988). A positive relationship between the number of CAG repeats and brain atrophy has also been proved by Kassubek et al. (2004). This result has further been supported by Ruocco et al. (2008).

11 The hour of birth of localism was in 1809 by Franz Joseph Gall's phrenology; a theory which states that all human abilities are stored in certain areas of the brain. The concept of localism, especially in the domain of aphasiology, will be addressed in more depth in chapter 2.1.
characteristic neurological-motor symptoms, as evident in the example of HD. In order to follow subsequent descriptions of HD, a brief description about cortico-basal ganglia-thalamo-cortical circuits is provided below.

1.4.1 Cortico-basal ganglia-thalamo-cortical circuits – explaining motor functioning

“. . . [the basal ganglia] are of undoubted importance clinically, but what contribution they make to the motor system, and how their dysfunction contributes to such neurologic disorders as [...] Huntington's disease, has been a riddle” (Graybiel, 1995: 60)

The basal ganglia are the largest subcortical structure in the human brain. They are considered a complex and highly interconnected group of nuclei in the forebrain, midbrain and diencephalon and have been the object of intensive research over many decades. The basal ganglia classically include the following nuclei (cf. e.g., Mink, 1999; Mendoza & Foundas, 2008; Mink, 2008):

1) The striatum, which consists of the putamen and the nucleus caudatus.
   The nucleus caudatus itself consists of three parts: the head at the front (caput), the body in the middle (corpus) and the tail at the end (cauda).
2) The globus pallidus (GP)\(^{12}\) which can be subdivided into:
   the globus pallidus external (GPe) and the globus pallidus internal (GPi)
3) The subthalamic nucleus (STN)
4) The substantia nigra (SN) with its two main parts:
   the pars compacta (SNC) and the pars reticulata (SNR)

Briefly, within these nuclei a distinction can be made between a group of input and a group of output structures. The input structures (the caudate nucleus and the putamen = striatum) receive direct input from all regions of the neocortex. Contrastingly, the basal ganglia output structures (the GPi and the SNC; collectively termed as the pallidum) project back to the cerebral cortex (mostly to the frontal cortex) via the thalamus. Originally, one nucleus of the thalamus, the ventrolateral thalamus (the thalamus consists of numerous different nuclei with various functions; the main three thalamic nuclei

\(^{12}\) Sometimes globus pallidus and putamen are called nucleus lenticularis.
participating in language, are given in Appendix II), was thought to project to a single cortical area, the primary motor cortex. This assumption arose from the theory that the basal ganglia function mainly in the domain of motor control\textsuperscript{13} (Kemp & Powell, 1970). To simplify the highly complex interactions between the basal ganglia the thalamus, and the cortex, models have been developed. The original basal ganglia model by Albin, Young and Penney (1989) was important in regards to the first formulation of a unifying model of the functional organization of the basal ganglia circuits for both normal and abnormal functioning.

\textbf{1.4.1.1 \hspace{1cm} Direct and indirect basal ganglia pathways}

The original model by Albin, Young and Penney (1989) is based on the direct and indirect pathways of the flow of cortical information through the basal ganglia and back to the cortex. Cortico-striatal projections are processed and transmitted to the basal ganglia output nuclei (GPi and SNr) via a direct route, or indirectly via the GPe and the STN (cf. Smith et al., 1998: 354). These distinct pathways compete with each other functionally, to release movement (direct pathway) or to inhibit movement (indirect pathway).

More specifically, within this model, the striatum can be seen as the main input centre of the basal ganglia (cf. Chesselet & Delfs, 1996: 417), as it gets massive excitatory inputs from various cortical areas. The received information is integrated within the striatum primarily by spiny neurons. Within the striatum there are four major types of spiny neurons: medium spiny neurons, large aspiny neurons, the medium aspiny cell and the small aspiny cell. The largest subtype of spiny neurons which make up to 80-95\%, are the medium spiny neurons (cf. Mink, 1999: 952; Mink, 2008: 726f). These striatal medium spiny neurons are inhibitory projection neurons that carry the output of the striatum to the basal ganglia's output areas, the GPi and the SNr. The target areas of the basal ganglia's output nuclei are the thalamic nuclei. These nuclei project received (excitatory or inhibitory) information back to the cortex (frontal cortex) (cf. Smith et al., 1998: 356; Mink, 1999: 954).

\textsuperscript{13} In 1986, Alexander, DeLong and Strick reviewed the results of numerous anatomical studies and proposed that it cannot be that simple. They proposed a theory that the basal ganglia participate in five parallel loops with various regions of the cerebral cortex and thus cannot just contribute to motor functioning. Details of the basal ganglia and their participation in cognitive tasks and language functions will be given in chapter 2.3.5.
Table 1 shows various basal ganglia nuclei and their neurotransmitters

<table>
<thead>
<tr>
<th>neurons</th>
<th>transmitters</th>
<th>characteristics</th>
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</thead>
<tbody>
<tr>
<td>corticostriatal</td>
<td>glutamat</td>
<td>excitatory</td>
</tr>
<tr>
<td>striatum</td>
<td>GABA(^{14})</td>
<td>inhibitory</td>
</tr>
<tr>
<td>GPi</td>
<td>GABA</td>
<td>inhibitory</td>
</tr>
<tr>
<td>GPe</td>
<td>GABA</td>
<td>inhibitory</td>
</tr>
<tr>
<td>SNr</td>
<td>GABA</td>
<td>inhibitory</td>
</tr>
<tr>
<td>SNc</td>
<td>dopamine</td>
<td>excitatory/inhibitory</td>
</tr>
<tr>
<td>STN</td>
<td>glutamat</td>
<td>excitatory</td>
</tr>
</tbody>
</table>

As exemplified above, the entire cerebral cortex projects onto the striatum. After integration of this information in the striatum, it is modulated by two opposing but parallel pathways: The direct versus the indirect pathway. These two competing pathways are described in normal functioning (see Figure 2 in chapter 1.4.1.1), additionally in the example of HD (see Figure 3 in chapter 1.5.3.1), as well as in the example of PD (see Appendix I), adopted and simplified from Albin, Young and Penney, (1989), Smith et al. (1998), Alexander and Crutcher (1990), DeLong (1990), Mink (1999), Gutekunst, Norflus and Hersch (2002), Murdoch (2004), as well as Murdoch and Whelan (2009).

Both pathways end in the thalamus. In turn, the two returning thalamo-cortical pathways reach precisely various regions of the frontal cortex (in this example of the motor cortex) in healthy adults, when the direct and the indirect pathways are in balance. In contrast to this, imbalance between the activity in the direct and indirect pathways, are supposedly responsible for hyper- and hypokinetic features of basal ganglia disorders (cf. Murdoch, 2004: 143). The most important modulator for the balance between the activity of the direct and indirect pathways at the level of the striatum, is dopamine; via an dopaminergic input from the SNc. In detail, release of dopamine from nigrostriatal projections (from the SNc to the striatum) via dopamine subtype D\(_1\), results in a facilitation of movement within the direct pathway. Against that, release of dopamine from nigrostriatal projections via dopamine subtype D\(_2\) results in an inhibition of movement within the indirect pathway (cf. Smith et al., 1998: 356). In detail, an extraordinarily high concentration of dopamine (dopamine receptors D\(_1\) and D\(_2\), receiving dopamine projections from the SNc) has been found in the putamen and the nucleus caudatus, which compose the striatum (cf. Sano et al., 1959: 587).\(^{15}\)

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14 GABA is the abbreviation for γ-aminobutyric acid.
15 Even though it is well accepted that the loss of dopaminergic neurons in the substantia nigra, is the
1.4.1.1.1  Direct pathway

The direct pathway originates from the striatal neurons that contain GABA and conveys from there to the basal ganglia output nuclei (GPi and SNr).

The cerebral cortex (motor cortex) is connected to the striatum by excitatory neurons using glutamate as their transmitter. Within the striatum there is activation of GABAergic neurons. The neurons connecting the striatum to the GPi and SNr also use GABA as a neurotransmitter. Because the neurons of the basal ganglia output nuclei are themselves inhibitory, there are two inhibitory synapses between the striatum and the thalamus. The effect of this is an inhibition of the inhibition (disinhibition), which in turn acts as an excitation and thus facilitating the thalamic projection to the motor cortex and consecutively also facilitating the initiation of movement.

1.4.1.1.2  Indirect pathway

The indirect pathway has the opposite function to the direct pathway and therefore inhibits movement. The difference between the direct and the indirect pathway is that there is an indirect projection from the striatum to the basal ganglia output nuclei via the external segment of the globus pallidus (GPe) and the STN.

The cerebral cortex (motor cortex) is connected to the striatum by excitatory neurons that use glutamate as their neurotransmitter. The striatum itself uses GABA as a neurotransmitter. Further, the neurons connecting the striatum to the GPe use GABA as a neurotransmitter and so does GPe itself. Due to the fact that there is an activation of GABAergic neurons in the striatum, these neurons inhibit the GPe and secondarily facilitate the STN, which is glutaminergic by virtue. Consecutively there is a strong effect of the STN to the GPi and the SNr, which are inhibitory by virtue and thus inhibit the thalamus in turn. Consecutively this results into an inhibition of the thalamocortical projections and thus inhibits the initiation of movement.

core pathophysiological factor for the ontogenesis of PD (e.g., Bartels & Leenders, 2009), interestingly, dysfunctions in dopaminergic neurotransmissions have now also been shown to lead to the degeneration of striatal neurons and consecutively may be crucial for the development of HD symptoms (cf. Jakel & Maragos, 2000: 240). It is moreover hypothesized that dopamine may have a crucial role in motor functioning and thus a dysfunctional dopaminergic neurotransmission may lead to motor symptoms as shown in transgenic HD mice (Hickey, Reynolds & Morton, 2002).
According to this classical model, as demonstrated in Figure 2, the basal ganglia are regarded as the main motor structures of the brain, second to the cerebellum. Further research has challenged this limited view. From the original model by Albin, Young and Penney (1989) further anatomical and functional models of the basal ganglia have been developed (see for instance Alexander, DeLong & Strick, 1986; Alexander & Crutcher, 1990; Alexander, Crutcher & DeLong, 1990; Cummings, 1993; Gurney et al., 2004; McHaffie et al., 2005), sometimes also based on primate brains (see for instance DeLong, 1990; Wise, Murray & Gerfen, 1996; Middleton & Strick, 2002), providing insight into how these nuclei might work and what their function may be. It is now accepted that from a functional perspective the basal ganglia should not be viewed in isolation.

“[...The] basal ganglia, along with their connected cortical and thalamic areas, are viewed as components of a family of “basal-ganglia-thalamocortical” circuits, that are organized in a parallel manner and remain largely segregated from one another, both structurally and functionally” (Alexander, Crutcher & DeLong, 1990:119)

The basal ganglia receive topographically organised projections from multiple cortical areas. This topographical organization is maintained within the projections back to the cortex, mainly to the frontal cortex, through the thalamus (cf. Haber & Calzavara, 2009: 71). Each circuit is thought to engage separate regions of the basal ganglia and the thalamus with output that projects to different parts of the cortex. Given, the segregated
nature of the cortico-basal ganglia-thalamo-cortical circuits, collectively they may have a unified role in modulating the operations of the entire frontal lobe and thereby influence motor activities, cognitive-linguistic and limbic processes (cf. Murdoch, 2004: 143). In detail, five different cortico-basal ganglia-thalamo-cortical circuits have been reported (Alexander, DeLong & Strick, 1986; Alexander, Crutcher & DeLong, 1990; Cummings, 1993; Strick, Dum & Mushiake, 1995; Strick, Dum & Picard, 1995; Middleton & Strick, 2000): a motor circuit that projects to the primary motor cortex, an oculomotor circuit that projects into the frontal eye fields, two prefrontal circuits projecting to the dorsolateral prefrontal as well as to the lateral orbitofrontal cortex, and a limbic circuit projecting to the anterior cingulate.

Nevertheless, the purpose of most of the basal ganglia loops cannot as yet be specified in any detail. There is some concern about the fact that the motor circuit subserves motor functioning, as well as the oculomotor circuit controls saccadic eye movement, and the dorsolateral prefrontal circuit is supposedly linked to executive functioning and motor programming. Projections to the orbitofrontal cortex as well as to the anterior cingulate have been studied the least, but have been reported to be involved in limbic functions (cf. Cummings, 1993: 875).

Disruptions to one or the other area, as just mentioned, within these loops can for instance result in various frontal lobe syndromes. An example of a frontal lobe syndrome is the dysexecutive syndrome, due to disruptions of the dorsolateral prefrontal and the orbitofrontal loop. The so called dysexecutive syndrome is associated with marked changes in personality such as irritability, as described by Benton (1968), Strub (1989), Sandson et al. (1991), Chan (2001), Godefroy (2003) and Slachevsky et al. (2006).

Interestingly, numerous other patients have been added to the literature with classical features of a frontal lobe syndrome but with lesions outside the frontal areas.

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16 The frontal lobe syndrome was described for the first time with the famous patient Phineas Gage, who sustained a destructive lesion in the prefrontal portion of the frontal lobe (see for instance Damasio et al., 1994). Since then it is known that the syndromes of apathy, nonmotivation, poor memory, reduced activity, lack of drive, inability to plan ahead, lack of concern, uncoordinated behaviour, inappropriate euphoria on one hand but irritability and aggression on the other hand, distractability, poor attention, perseverative and stereotypes responsive behaviour, gait disturbances and forms of dynamic aphasia (Luria & Tsvetkova, 1968) can be summarised as the frontal lobe syndrome (Strub, 1989; Thimble, 1990).

17 The term executive functions refers to high-cognitive functions such as planning, decision making, response initiation and response suppression, problem-solving, rule deduction, cognitive flexibility, strategy, operating in non-routine situations such as novel, conflicting or complex tasks, stored in the frontal lobe of the brain (cf. Godefroy, 2003: 1). Damage to the frontal lobes may render to dysfunctions in executive functioning, collectively known as dysexecutive syndrome (cf. Chan, 2001: 551), a subsynrome of a more global frontal lobe syndrome.
Frontal lobe syndromes have also been described in patients with bilateral globus pallidus lesions (Strub, 1989), following left thalamic infarctions (Sandson et al., 1991), following bilateral basal ganglia lesions (Laplane et al., 1989) and in the course of thalamic atrophy (Kassubeck et al., 2005).

Due to HD's specific neuropathology, with a specific neuropathology in the basal ganglia and various cortical areas (Vonsattel et al., 1985; Vonsattel & DiFiglia, 1998; Vonsattel, 1999; Rosas et al., 2003; Douaud et al., 2006; Rosas et al., 2008a; Rosas et al., 2008b; Jernigan et al., 1991; Thieben et al., 2002; Henely et al., 2006; Rosas et al., 2006; Squitieri et al., 2009), disruptions within the cortico-basal ganglia-thalamo-cortical loop are the result, leading to HD's specific symptoms. The main symptoms in the course of HD will be described next.

1.5 Clinical findings – the three main symptoms of HD

Due to these neuropathological changes, as previously presented, specific symptoms arise in the course of HD. In detail, HD has traditionally been characterised by three core symptoms including neuropsychiatric, neuropsychological and neurological-motor abnormalities. These involve diverse aspects of abnormal behaviour and personality change that are accompanied by multiple mental disabilities such as subcortical dementia and abnormal body movements in the form of chorea and others.

In order to define HD stage and its progression, two main rating scales can be differentiated, measuring symptoms associated with HD: The Unified Huntington's Disease Rating Scale (UHDRS) and the The Total Functional Capacity Scale (TFC).

The Unified Huntington's Disease Rating Scale (UHDRS), developed by the Huntington Study Group in 1996, is a clinical rating scale that assesses clinical performance and capacity in HD patients, such as: behavioural abnormalities (e.g., anxiety, suicidal thoughts etc.), cognitive dysfunctions (e.g., verbal fluency) and motor dysfunctions (e.g., ocular pursuit, saccade initiation etc.). The UHDRS is also a useful tool for measuring changes in the clinical features of HD over time. As the motor subpart of this scale has been used within this study, detailed information regarding its scoring procedure will be given in chapter 4.1.
The Total Functional Capacity Scale (TFC) by Shoulson and Fahn (1979) (also used within this research), allows a general rating of HD patient's 'total functional capacity' in the following five domains:

1) Engagement in occupation  
2) Capacity to handle financial affairs  
3) Capacity to manage domestic responsibilities  
4) Capacity to perform activities of daily living  
5) Degree of care needed by the patient

In the course of HD these functions decrease, one after another. Following, the decrease allows the classification of the 5 HD stages, representing its progression.

At an early stage (1), right after diagnosis, the person suffering from HD can function fully both at work and at home. At the early intermediate stage (2) the person remains employable to a lower capacity and is still able to manage daily life. At the late intermediate stage (3) the person can no longer work or manage household responsibilities alone. To handle daily affairs supervision is needed. In the early advanced stage (4) the person is no longer independent in daily activities, but is still able to live at home with the support of the family. At the advanced stage (5) the person with HD requires complete support in daily activities from professionals such as nurses (Shoulson & Fahn, 1979)18.

As the UHDRS as well as the TFC rate HD's specific symptoms and their effects on daily life, HD's three main symptoms will be described next.

### 1.5.1 Neuropsychiatric symptoms

The majority of HD patients experience neuropsychiatric symptoms, with a prevalence of major psychiatric disorders ranging from 68 to 95% (cf. Woodcock, 1999: 156). As these symptoms have been reported to be the first ones to occur (observable even 10 years before diagnosis) (cf. Duff et al., 2007: 1345), Rosenblatt and Leroi (cf. 2000: 29) noted that basal ganglia disorders such as HD can even be considered a

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18 In addition to these five stages Shoulson et al. (1989) proposed an alternative scoring mechanism, using a scale from 0-13. A total functional capacity (TFC) score of 13 on this ordinal scale indicates full capacity in all five categories, whereas a TFC score of 0 indicates total incapacity (cf. Shoulson et al., 1989: 272).
neuropsychiatric disorder. Neuropsychiatric symptoms associated with HD resemble those described in a frontal lobe syndrome. Specifically, irritability and bad-tempered outbursts have been summarised as the most common and sometimes also the earliest behavioural manifestations in HD. Patients become irritable for no obvious reason, even the slightest provocation can provoke an outburst of angry and violent behaviour (cf. Craufurd & Snowden, 2002: 78f). Thus Rosenblatt et al. (2003) supported the notion that one of the biggest burdens on caregivers in the context of neuropsychiatric disturbances, is the management of aggressive behavior of HD patients as they react disproportionally to situations and emotions e.g., disappointment or frustration in interpersonal situations. Caine and Shoulson (cf. 1983: 732) clustered the psychiatric syndromes of 24 HD patients and identified furthermore disorders of 1) arousal, attention, and concentration, 2) mood and affect as well as 3) personality. Paulsen et al. (cf. 2001: 311) showed that in 98% of a total of 52 patients tested, the most prevalent neuropsychiatric symptoms were dysphoria (69.2%), agitation (67.3%), irritability (65.4%), apathy (55.8%) and anxiety (51.9%). Delusions and hallucinations were also prevalent, but to a lesser degree (11% for delusions and 1.9% for hallucinations). Similar findings have also been reported by Thompson, Snowden and Craufurd (2002) and Kingma et al. (2008).

Using the 'Problem Behavior Assessment Scale for Huntington's Disease' by Naarding and Janzing (cf. 2003: 338) the following neuropsychiatric symptoms were revealed: severe loss of energy, lack of initiative (apathy), impaired judgement, emotional blunting and self-neglect. According to Rosenblatt and Leroi (2000), major depression19, mania, psychotic disorders (such as schizophrenia, paranoia, diverse psychoses and hallucinations), obsessive-compulsive disorders (for details see for instance Cummings & Cunningham, 1992 or Beglinger et al., 2007), aggression and sexual disorders are also part of the neuropsychiatric abnormalities detected in HD. The most frequent sexual disorders reported for HD patients (both sexes) are hypoactive sexual desire and inhibited orgasm (Fedoroff et al., 1994).

19 In the clinical context the Hamilton Depression Rating Scale (HDRS) also known as Hamilton Rating Scale for Depression (HDRS) is most widely used to rate the severity of a patient's major depression. The original version contains 17 questionnaire items focusing on depressed mood, guilt, suicide, insomnia, work and interests, retardation, agitation, anxiety, somatic, genital, hypochondrias, insight, and loss of weight, which pertain symptoms of depression. A 21-items version also exists, which gives further information about the type of depression (e.g., obsessional and compulsive symptoms, paranoid symptoms). The items are measured either on a five-point or three-point scale. The higher the score, the more severe the depression is (cf. Hamilton, 1960; 1967). A more detailed description of the scoring procedure is given in chapter 4.1.
Similarly, Craufurd, Thompson and Snowden (2001) reported a loss of libido in 62% out of 134 HD patients tested and thus concluded that hypossexuality is far more common than other sexual disorders such as hypersexuality or paraphilias in HD patients (cf. Craufurd, Thompson & Snowden, 2001: 224).

In regards to depression, Paulsen et al. (cf. 2005b: 498) demonstrated high rates of depressive symptoms during the initial stage of the disease and a peak during stage 2. Depressive symptoms can disappear during middle and later stages of the disease perhaps due to an adaptation to the illness. Thus, not surprisingly, depressive stages are highly correlated with suicidal ideation. The examination of 831 HD patients revealed that 5.7% of HD patients committed suicide and 27.6% have attempted suicide at least once (cf. Farrer, 1986: 305). The prevalence of suicide has been reported to be four times higher among HD patients before diagnosis than among those who have been diagnosed positive with HD (Schoenfeld et al., 1984). This finding has further been supported by Robins (2000) and Paulsen et al. (2005a).

Although George Huntington (1872) noted changes in personality, less consideration has been given to cognitive impairments caused by HD. However, cognitive changes are an invariable feature of HD (cf. Craufurd & Snowden, 2002: 71) and thus will be discussed in the following chapter.

### 1.5.2 Neuropsychological symptoms

HD patients are affected by a specific neuropsychological deficit, resembling a dysexecutive syndrome. It is assumed, that changes in executive functioning, are due to a disruption within the cortico-basal ganglia-thalamo-cortical circuit (cf. Weinberger et al., 1988; Lawrence et al., 1996; Bäckman et al., 1997; Lawrence, Sahakian & Robbins, 1998). These cognitive changes are assumed to start even in presymptomatic HD patients (e.g., Butters, et al., 1978; Rosenberg, Soerensen & Christensen, 1995; Hahn-Barma et al., 1998; Brandt et al., 2002; Soliveri et al., 2002; Lemiere et al., 2004; Peinemann et al., 2005; Van Raamsdonk et al., 2005; Baudic et al., 2006; Ward et al., 2006; Van der Hiele et al., 2007; Wahlin, Lundin & Dear, 2007).

Due to their dysexecutive syndrome patients have been reported to fail problem solving, have particular difficulty with cognitive tasks that require organization, planning and sequencing of events (Brandt, Folstein & Folstein, 1988; Rosenberg, Soerensen &
Christensen, 1995; Lawrence et al., 1996; Lemiere et al., 2004; Peinemann et al., 2005),
have difficulties organizing their day-to-day activities and can appear behaviourally
disorganized and in general HD patients have difficulty to cope with multiple tasks
simultaneously. For instance walking and talking at the same time can be impossible
and thus HD patients are often noted to stop walking when answering a question
(cf. Crauford & Snowden, 2002: 64). In accordance to these findings, Sprengelmeyer,
Lange and Hömberg (1995) reported that divided attention (the ability to simultaneously
monitor information given to different modalities) is severely affected in HD patients.
Moreover, a poor performance in problem solving, as well as difficulty with shifting of
attention due to an inflexible, apathic like pattern of thinking (e.g., Baudic et al., 2006),
have been described. Executive functions have been tested in HD patients using a
number of neuropsychological tests20. Perseverative card sorting behaviour within the
Wisconsin Card Sorting Test has been reported by Josiassen et al.(1983) as well as by
Peinemann et al. (2005). Troubles solving the Tower of Hanoi Puzzle have been
mentioned by Wahlin, Lundin and Dear (2007) as well as an ineffectivity performing the
Stroop Colour Interference Test has been stated by Lemiere et. al. (2004) and
Peinemann et al. (2005). These failures in multiple domains of cognition seen in HD,
can be summarised under the broad term of dementia21. Dementia refers to symptoms of
intellectual dysfunction due to widely varying aetiologies and neurophysiological
mechanisms (cf. Albert, Feldman & Willis, 1974: 121). Dementia is not a unitary
disorder. A difference is stated between the dementia seen in Alzheimer's disease and
that seen in HD (cf. Paulsen et al., 1995: 48). The dementia of the Alzheimer type is
often named as cortical dementia with severe, predominant memory impairment that
affects both recall and recognition performance as well as language (Cummings &
Benson, 1992; Salmon & Butters, 1992). In contrast to this, the dementia associated
with HD is of subcortical nature and will be addressed in the next chapter.

20 Executive functions are tested in neuropsychology with the Wisconsin Card Sorting Test (Berg, 1948),
the Tower of London Test (Shallice, 1982), the Tower of Hanoi Puzzle (a comprehensive description
of the puzzle is given for instance in Cohen et al., 1985), the Stroop Test (Stroop, 1935) or with
fluency tasks (for details see chapter 1.5.2).

21 The mini-mental state examination (MMSE) is a brief 30-point questionnaire test that is used to assess
cognition and is thus further commonly used in medicine to screen for dementia. The MMSE is
devided into two sections. The first section covers orientation, memory and attention. The maximum
score for this part is 21. The second part tests the ability to name, follow verbal and written
commands, write a sentence spontaneously and copy a complex figure. The maximum score for that
part is nine. In total any score over 27 (out of 30) is effectively normal. Below this, 20-26 indicates
mild dementia, 10-19 moderate dementia, and below 10 severe dementia (cf. Folstein, Folstein &
McHugh, 1975).
1.5.2.1  **Subcortical dementia**

Due to the fact that HD is characterised by a prominence of lesions to the basal ganglia, HD is considered a subcortical dementia (Folstein, Folstein & Mc Hugh, 1975). The specific symptoms of memory dysfunctions in this type of dementia are: forgetfulness (but not amnesia), defects in concentration, slowing of thought processes, deficits in problem solving, difficulties in recall (tested for instance by letter/category fluency tasks as will be addressed in detail in chapter 3.5). Recognition is typically not affected (Butters et al., 1978; Caine et al., 1978; Butters, 1984; Butters et al., 1986, 1987, 1988). Furthermore, deficits in learning new skills have been reported (Martone et al., 1984; Butters et al., 1985; Heindel, Butters & Salmon, 1988; De Diego-Balaguer et al., 2008). Psychopathological symptoms such as emotional or personality changes (apathy and depression; as described in chapter 1.5.1) as well as impaired mental flexibility (as described in chapter 1.5.2), slow, dysarthric, but not aphasic speech and deficits in spatial orientation and visuomotor integration\(^{22}\) have also been associated with HD (Albert, Feldman & Willis, 1974; Folstein, Folstein & Mc Hugh, 1975; Butters et al., 1978; Cummings & Benson, 1984; Brandt & Butters, 1986; Brandt, Folstein & Folstein, 1988; Brandt, 1991; Brandt & Butters, 1996).

The only function reported to be fully intact in HD patients with subcortical dementia versus cortically demented patients, is the patients' language function, in terms of a lack of aphasic symptoms (cf. Butters et al., 1978: 585; Whitehouse, 1986: 1). Similarly, Cummings and Benson (1984) emphasised that clinically significant aphasia, agnosia and apraxia are not typically seen in the context of HD's specific symptomatology.

Additionally, Zakzanis (1998) reported that verbal skills are the least impaired functions in HD. If deficits are noted in language it is argued that these tasks go beyond language and require memory and cognitive abilities such as organization, sequencing, and linguistic strategies (cf. Cummings & Benson, 1992: 97).

Nevertheless, the legitimacy of the distinction between a cortical and a subcortical dementia and the assumption of a lack of language problems associated with subcortical dementia, can be questioned on several points:

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\(^{22}\) Spatial orientation and visuomotor integration is tested in clinical neuropsychology often with visuospatial tasks such as the Rey-Osterrieth Complex Figure. This test requires the patient to copy a complex figure (Rey, 1941; Osterrieth, 1944). In order to perform correctly on this task different abilities are needed, such as visuospatial abilities, memory, attention, planning and working memory.
1) Cummings (1986: 693) stated that “Sufficient information is available, however, to demonstrate that subcortical dementia is characterised by a clinical syndrome that is identifiable and can be distinguished from the syndrome of cortical dementia“. In contrast to this, Whitehouse (1986: 1) reviewing essentially the same literature, concluded that “[...] recent neuropathological, and neurochemical studies offer little support for this classification system“. Similarly, Butters et al. (1998) suggested that although the distinction between cortical and subcortical dementia may be useful in regards to pathophysiology (cortical versus subcortical affected brain areas), it may be ineffective in terms of clinical assumptions. This is because symptoms of both dementias can resemble each other (cf. Butters et al., 1998: 734). Thus, this argument would lead to the assumption that language changes, present in cortical dementias, could also be part of a subcortical dementia.

2) Furthermore, Brown and Marsden (cf. 1988: 368ff) concluded that the use of certain language assessments itself might be responsible for the presence of negative results in regards to language functions in subcortical dementias. As will be addressed in chapter 3, the use of aphasia batteries, designed for cortical injured patients, can be regarded as not sensitive enough to reveal language problems in subcortical dementias. This assumption could be proved, as Podoll et al. (1988), Wallesch and Fehrenbach (1988) as well as Chenery, Copland and Murdoch (2002) used cortical aphasia batteries (the Aacherner Aphasia Battery = AAT23 and the Western Aphasia Battery = WAB24), focusing on ‘primary language abilities’25 (cf. Chenery, Copland & Murdoch, 2002: 467) and did not evidence any language problems in HD patients. Contrastingly, tests comprising 'complex language functions', e.g., the Test of Language Competence-Expanded

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23 The Aachener Aphasia Test (AAT) is a standardized neurolinguistically conceived aphasia test developed for German speaking neurologically impaired patients. The AAT comprises the Token Test, tasks concerning spontaneous speech, repetition, written language, naming and comprehension (Huber et al., 1983).

24 The WAB is a standardized aphasia assessment, developed by Kertesz (1982), to probe clinical aspects of primary language functions in aphasic patients. The WAB comprises 8 subtests including content, fluency, auditory comprehension, repetition, naming, reading, writing and calculation.

25 Briefly, under the term 'primary language functions' basic cognitive-linguistic functions such as repetition and naming can be summarised. The term 'complex language functions', refers to pure linguistic functions, requiring pure linguistic abilities, as tested e.g., within sentence generation tasks (for further information see also chapter 4.3).
The TLC-E\textsuperscript{26}, the Test of Word Knowledge (TOWK)\textsuperscript{27} and the Word Test-Revised (TWT-R)\textsuperscript{28} (cf. Chenery, Copland & Murdoch, 2002: 467) did detect linguistic deficiencies in HD patients.

3) Moreover, Turner, Moran and Kopelman (cf. 2002: 150) stated that it is difficult to sustain the idea that only cortical dementia is characterised by the combination of aphasia, apraxia and agnosia, since apraxia is known to occur in disorders affecting the basal ganglia. Thus it could be assumed that aphasic syndromes may also occur in the course of subcortical dementia. Additionally, Turner Moran and Kopelman (cf. 2002: 150) supported the existence of subcortical-cortical connections. Damages to these projections may cause language changes in subcortical dementias such as HD.

4) The assumption by Cummings and Benson (cf. 1992: 97) that linguistic disturbances within subcortical dementias go beyond language and thus require memory and cognitive abilities such as organization, sequencing, and linguistic strategies, can be regarded as inappropriate. Regardless of the tasks testing language (e.g., naming, repetition, fluency, word formation etc.), they always interfere somehow with cognition. Thus it is difficult \textemdash\ or even impossible \textemdash\ to test language functions on their own, without also testing cognition to some extent.

In conclusion, various high-cognitive changes seem to occur in HD's neuropsychological symptomatology. Thus it could be assumed that also language changes arise within HD's subcortical dementia, due to disruptions within the cortico-basal ganglia-thalamo-cortical loop.

Nevertheless, so far we have addressed neuropsychiatric as well as neuropsychological changes in HD's specific symptomatology. The occurrence of neurological symptoms \textemdash\ as the third piece of HD's symptomatology puzzle \textemdash\ will be discussed in the following chapter.

\textsuperscript{26} The TLC-E, developed by Wiig and Secord (1989), measures metalinguistic higher-level language functions such as recognizing and interpreting the alternative meanings of ambiguous sentences, making inferences, creating a sentence out of three given words or interpreting metaphoric expressions.

\textsuperscript{27} The TOWK, developed by Wiig and Secord (1992), identifies semantic knowledge such as figurative language, synonyms, antonyms or word definitions after brain injury.

\textsuperscript{28} The TWT-R also developed by Huisingsh, Barrett and Zachman (1990), assesses a subject's capacity for verbal reasoning and the ability to recognize critical semantic features of the vocabulary. The subject is for example asked to change a senseless sentence to make sense.
1.5.3  Neurological symptoms

HD is characterised by significant neurologic symptoms, specifically by involuntary movements due to basal ganglia dysfunctioning and irregularities of the neurotransmitter dopamine (cf. Kishore & Calne, 1999: 1). As previously described, the basal ganglia are interconnected with the neocortex by parallel loops. Dysfunctions in one or the other structure within these parallel loops (damage to the striatum in the example of HD), result amongst other symptoms, into major movement disorders (cf. Graybiel et al., 1994: 1826).

1.5.3.1  Neurophysiological basis of HD motor disturbances

In the example of HD, there is a selective loss of GABAergic neurons in the striatum. This is thought to lead to a reduced inhibition of the GPe, which in turn has an even stronger inhibitory projection to the STN. This in turn leads to a reduced excitatory projection from the STN to the basal ganglia output nuclei. This reduced inhibitory outflow from the GPi and the SNr results in a massive disinhibition of the thalamus and thus enhances movement. Consecutively, an overactivation of the motor cortex results, which in turn causes uncontrolled motor activation such as chorea. Additionally, there is an even stronger excitatory projection of the basal ganglia output nuclei to the thalamic nuclei, within the direct pathway, and thus supports the enhancement of movement (adopted and simplified from Albin, Young & Penney, 1989; Smith et al., 1998; Alexander & Crutcher, 1990; DeLong, 1990; Mink, 1999; Gutekunst, Norflus & Hersch, 2002; Murdoch, 2004; Mink, 2008; Murdoch & Whelan, 2009).

The neurophysiological basis for movement disorders in Parkinson's Disease (PD) as the opposite of HD, is given in Appendix I.
1.5.3.2 Specific motor disturbances

As the disease worsens, psychiatric and psychological changes are accompanied by characteristic involuntary movements. Prior to the clear appearance of extrapyramidal signs such as chorea, minor motor abnormalities do occur. Oculomotor disturbances are among the earliest signs in affected people. These include for example, abnormal eye movements, delayed initiation of voluntary saccades (Ruiz et al., 2001) as well as an impaired optokinetic nystagmus (Lasker & Zee, 1997). As suggested by Folstein et al. (1983), disturbances in motor speed as well as fine motor control (e.g., severe slowness in finger tapping), general restlessness and hyperreflexia can be seen as further examples of early movement abnormalities in HD patients. As the disease worsens, pure hyperkinetic movement disorders occur (cf. Cudkowicz, Martin & Koroshetz, 1999: 159). The main hyperkinetic movement disorder of HD is chorea (cf. Cardoso et al.,

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30 The optokinetic nystagmus (OKN) is a subcortically-mediated function, which occurs when an optical stimulus moves across the retina. This reflex is a rhythmic to and from movement of the eyes and allows the eye to follow objects in motion when the head remains stationary. As the element moves out of sight, the eyes will 'snap back' to fixate and follow another one (cf. Pschyrembel, 2007: 1367).

31 Hyperkinesia refers to an abnormal increase in muscular activity as muscle tone decreases at the same time, which results in a wide variety of abnormal involuntary movements. The opposite of hyperkinesia is known as hypokinesia. Hypokinesia or akinesia is a disorder characterised by the lack of or the slowness of initiation and execution of voluntary and associated movements. Patients report reduced speed of activities of daily living, slowness and general fatigue. They even have difficulty in changing from one motor pattern to another (compare for instance Lakke, 1981: 314; Fahn, 2007: 289f.). Both hyperkinesia and hypokinesia can be explained on the basis of the involvement of the direct and indirect pathways of the basal ganglia circuits (Berardelli et al., 1999), as exemplified in chapter 1.4.1.1.1 (direct pathway) and chapter 1.4.1.1.2 (indirect pathway).
in up to 90% of affected patients (cf. Bhidaysari & Truong, 2004: 528), hence the old name 'Huntington's Chorea' arised (cf. Kremer, 2002: 32). This symptom is characterised by short, excessive, involuntary, irregularly timed, randomly distributed, muscular contractions. These abrupt movements can occur for any part of the body. It is possible that movements spread from one body part to another (cf. Vieregge, 2007: 54). Movement patterns differ from patient to patient, but in general various chorea patterns can be dedected in HD patients. Mostly, chorea starts with slight movements of the fingers and toes and progresses to facial grimacing and eyelid elevations (cf. Bhidayasiri & Truong, 2004: 527f). As the disease advances chorea of the face, neck, trunk, limbs and legs can be described. Chorea of the trunk moves the whole body in different directions and thus breathing and speech are negatively influenced, leading to dysarthria (cf. Huber, 2005: 126) Chorea of the trunk also leads to swallowing impairments (dysphagia), which are assumed to contribute to aspiration and and even death (cf. Kremer, 2002: 37). As chorea is involuntary, the movements cannot be stopped voluntarily and patients are sometimes not even aware of their symptoms. In general, chorea stops during sleep, but worsens by anxiety and stress (cf. Cudkowicz, Martin & Koroshetz, 1999: 159). Severity may vary from restlessness, intermittent gestures and expressions and an unstable gait to a continuous flow of disabling movements. Frequent flections and extensions of the fingers can be seen, as well legs may be alternatively crossed and uncrossed as well as toes flexed and extended. Thus the gait can become dance-like (cf. Lakke, 1981: 318; Kremer, 2002: 32) and the label 'chorea', from the Greek word 'dance' is appropriate (cf. Vieregge, 2007: 54). Thus Koller and Trimble (cf. 1985: 1451f) described that the gait in HD as a characteristic feature of the illness is unlike that in any other neurological disease, distinguished by a difficulty in initiation of locomotion, reduced associated movements of the arms in walking and small shuffling steps. Only rarely can patients walk tandem or stand on one foot for longer than 20 seconds. Their steps vary in length and feet are often placed too far laterally (cf. Cudkowicz, Martin & Koroshetz, 1999: 151f). As a consequence of gait disturbances and the loss of normal rhythm of walking, patients experience frequent falls (Grimbergen et al., 2008) which can cause serious injuries or even death (cf. Koller & Trimble, 1985: 1452). Due to serious injuries after frequent falls, patients are often unable to remain in their own home and are moved to a nursing home, sometimes even very early in their disease (Wheelock et al., 2003). Another factor accompanying HD's motor disturbances is a massive weight loss
The full spectrum of motor impairments in HD can be summarised to include eye movement abnormalities, chorea, tremors, myoclonus, tics, dysarthria, dysphagia, hyperreflexia, as well as a coexistence of Parkinsonian features such as bradykinesia and chorea (Lakke, 1981; Thompson et al., 1988; Cardoso et al., 2006). As the disease advances, chorea tends to disappear and is replaced by hypokinetic movement disorder such as bradykinesia as well as frequent arrests of movements known as 'freezings' (cf. Jankovic, 2007: 738).

As previously mentioned in chapter 1.5, HD's motor disturbances are usually tested with the motor subtest of the UHDRS. The UHDRS motor score quantifies motor symptoms in regards to HD, such as oculomotor function, dysarthria, chorea, dystonia, gait and postural stability. The severity of the motoric dysfunction is rated on a scale ranging from 0-4 (0 = no impairment; 4 = severely affected) by summing up the various questions of each motor subsection (ocular pursuit, saccade initiation, saccade velocity, dysarthria, tongue protrusion, maximal dystonia, maximal chorea, retropulsion pull test, finger taps, pronate/supinate hands, fist hand-palm test, rigidity arms, bradykinesia body, gait and tandem walking). Thus the worst one can get is a score of 4 in all 31 subsessions, that is a score of 124 in total (cf. Huntington Study Group, 1996: 136-142).

1.6 Summary and perspectives

As presented in chapter 1, HD is a progressive subcortical disease, that worsens over 5 stages and manifests in neuropsychiatric, neuropsychologic and neurologic symptoms. Traditionally, HD has not been associated with linguistic disturbances.

The aim of this thesis is thus to demonstrate that linguistic disturbances do indeed occur in HD. In order to do so, HD's specific symptomatology must be considered. Furthermore, in order to effectively measure language changes in HD patients, disease

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32 Tremors are rhythmic, regular, oscillating movements of limbs, trunk, head (cf. Lakke, 1981: 315).
33 Myoclonus is characterised by sudden shock-like contractions of a muscle or muscle group. These constraints may be joined by other movements, may occur at rest or during action, and may be repetitive, rhythmic or isolated (cf. Lakke, 1981: 317).
34 Tics are sudden, stereotyped, complex, repetitive, normally coordinated but inappropriate movements. Tics may be single such as sniffling, vocalization, finger movements or may occur in combination and may even be accompanied by impulsive and emotional behaviour (cf. Lakke, 1981: 316).
35 Bradykinesia, a hypokinetic movement disorder, refers to a slowness in the execution of movements, slowing of activities of daily life such as dressing, feeding, brushing etc. (cf. Jankovic, 2007: 738).
factors including 'days since disease onset' under control of the 'number of CAG repeats' as well as HD stages have to be considered.

A critical literature review addressing 'language function's in Huntington's disease', will be provided in chapter 3, discussing the incorporation of these factors.

Firstly, within chapter 2, a brief overview will be given on language and the brain, focusing on the basal ganglia in language, as the area specifically affected in HD. These considerations in sum, are in order to justify the assumption of this thesis, that language changes do occur in the course of HD, as a disease of the basal ganglia.
2 Language and the brain – subcortical functions in language

In the following chapter a brief overview will be given concerning the relationship between brain and language, starting with early theories about language and the brain, including the emergence of aphasiology.

It should be mentioned at this point, that the history of aphasiology is much more complex than will be shown within this work. Due to the fact that the main focus of this thesis is on language functions in Huntington's disease (HD), as a subcortical disorder, only a few milestones within the history of classical aphasiology will be presented here.

Special attention will be dedicated to the discussion and criticism of limitations of classical models of aphasia. More specifically, recent theories about language and the brain will be presented, with special attention to basal ganglia functions in language.

2.1 Early theories about language and the brain

Much of what is known about how we use language and how language is stored in our brain was for a long time solely based on studies of patients with language disturbances. While the Edwin Smith Surgical Papyrus of 3500 B. C. contains references of the loss of speech, the first source that clearly recognises the role of brain damage in speechlessness is given in the early Hippocratic Corpus (approx. 400 B. C.) (cf. Goodglass, 1988: 249). Benton and Joynt's (1960) review paper entitled 'Early descriptions of aphasia' provides a summary of the works that investigated the loss of speech and language prior, the 19th century. This review paper revealed that a number of clinical forms of aphasia, albeit under a different name, e.g., motor aphasia, jargon aphasia and amnestic aphasia existed in the literature even before the 18th century.
century. It is only sensory aphasia that had not been recognized as a specific entity until Carl Wernicke in 1874. Moreover, it was already known by then that aphasia occurs as a consequence of various diseases of the brain. However, at that time the localisation of aphasia could not be achieved (cf. Benton & Joynt, 1960: 124). The hour of birth of localism was in 1809 by Franz Joseph Gall's phrenology; a theory which states that all human abilities are stored in certain areas of the brain. In addition, the question of where language is stored in the brain also arose. Gall was the first to make a distinction between two aspects of language function and thus stated a lexical component of language 'Wortsinn' and additionally a motoric component of the faculty of spoken language 'Sprachsinn', both supposed to be localized in the inferior anterior frontal lobe (cf. Gall & Spurzheim, 1809 cited from Whitaker, 1998: 33).

2.2 The emergence of aphasiology

Gall's hypothesis that the frontal lobe is the seat of language, had been rejected for more than 40 years, prior to Broca's influential article 'Remarks on the seat of the faculty of articulate language, followed by an observation of aphemia', in 1861. In this paper, Broca has stated for the first time, that the faculty of language is localized in the left frontal convolution. This theory has been developed on the basis of the pathological history of a patient (Monsieur Leborgne) whose linguistic understanding was normal, but who was able to pronounce only the meaningless monosyllable 'tan' and was thus referred to as Monsieur 'Tan-Tan'.

"[T]his abolition of speech in individuals, who are neither paralyzed nor idiots, is a sufficiently important symptom so that it seems useful to designate it by a special name. I have given it the name Aphemia [...]" (Broca, 1861: 51)

Basically, the only thing Monsieur 'Tan-Tan' was missing, was the faculty to articulate words. After suffering from aphemia for 21 years, Broca's most famous patient died on 17th April 1861. Because Broca suspected that maybe his defect in articulate language could have been largely due to his cerebral lesion, autopsy of his brain was performed as soon as possible (cf. Broca, 1861: 51), revealing destruction of the following organs:

39 The German doctor Johann Gaspar Spurzheim, who studied medicine in Vienna, was hired by Franz Joseph Gall as his assistant and thus Gall added Spurzheim's name as a coauthor to his books and publications (cf. Zola-Morgan, 1995: 365).
"The small inferior convolution of the temporal lobe, the small convolutions of the insual and the underlying part of the striate body, finally, in the frontal lobe, the inferior part of the transverse frontal, and the posterior part of those two great convolutions designated as the second and third frontal convolutions. Of the four convolutions which form the superior part of the frontal lobe, only one, the superior and most medial one, has been conserved, not in its integrity, for it is softened and atrophied, but in its continuity, and if one puts back in one's thought all that has been lost, one will find that at least three quarters of the cavity has been transversed by the frontal lobe" (Broca, 1861: 68)

Facing this extensive destruction it is puzzling why Broca suspected the third frontal convolution of the inferior frontal gyrus to be the seat of aphemia? According to Schiller (1979) Broca may have assumed that the slowly progressing lesion (over 21 years in the case of Monsieur Leborgen) started in one specific center of defect (e.g., in the third frontal convolution that was destroyed the most) and was spreading from there to all other areas disrupted, including the superior temporal gyrus. According to this hypothesis, Broca may have conveyed that the third left convolution, as the area most affected and the origin of the defect, was responsible for Monsieur 'Tan-Tan's' deficit in articulate language (aphemia\textsuperscript{40}) (cf. Schiller, 1979: 186).

Nevertheless, Broca's theory that the faculty of language is localized in the left frontal convolution has been challenged by mainly two neurologists, Theodor Meynert (1866) and Carl Wernicke (1874) evidencing a sensory counterpart to motoric Broca's area. Meynert studied fibre tracts of the brain\textsuperscript{41} and exemplified, on the basis of a case study, a connection of the acoustic nerve by a fibre path which he called the acoustic tract (Acusticusstrang), to the fossa Sylvii deep in the temporal lobe of the cortex (Meynert, 1886).

\textsuperscript{40} Throughout the 1860s various debates followed Paul Broca's aphasiological descriptions, especially in terms of terminology. Competing terms existed to designate disorders of speech; alalia used by Jacques Lordat (1843), aphemia coined by Paul Broca (1861) and ultimately aphasia has been introduced by Armand Trousseau (1864). Trousseau complained that aphemia in Greek means infamy and therefore is not a suitable label for language disturbances. This fact could also convince Broca and thus he stated his preference for the term aphasia. The original aphemia of Broca was clearly a disorder affecting only speech production but aphasia on the other hand also serves a useful label for disorders of speech and language (cf. Henderson, 1990: 86).

\textsuperscript{41} Meynert distinguishes two major fibre systems, the projection and association pathways. Meynert points out that there are two types of projection paths, the centripetal tracts carrying sensory information to the cortex and the centrifugal tracts, conveying motor impulses to the periphery. Moreover, he mentions that there are three levels of the projection system: The first level includes fibres coursing between the cortex and subcortical ganglia, a second between the thalamus and the central gray matter of the brain stem and a third between the gray matter of the brain stem and the sensory or motor end-organs in the periphery. The association tracts on the other hand start and end in the cortex. This dichotomy of Meynert between sensory and motor regions was fundamental to Wernicke's theory of aphasia (cf. Eggert, 1977: 23).
In Meynert's patient an embolus had blocked an artery in the insula, leading to pathological changes in the most posterior gyrus of the insula and in the posterior portions of the temporal lobe above the insula, part of what we now call the planum temporale and part of what we would refer to as Wernicke's area (cf. Whitaker & Etlinger, 1993: 565). Even though Broca's area was intact in Meynert's patient, a failure of word retrieval mechanisms and defective morphology could be found. Meynert's patient failed to retrieve words necessary for communication (e.g., the word 'Hand'); semantically deviated words escaped instead ('gelb' instead of the word 'Hand'). The morphology of words was defective moreover (the word 'Husten' had been changed to a German non-word 'Hutzen') (cf. Meynert, 1866: 154f).

While concluding this article, Meynert received another brain in which almost the entire insula, as well as an impressive part of the operculum and the superior surface adjacent to the Sylvia fissure of the primary temporal gyrus had been destroyed, also leading to the same aphasic symptoms as described before (cf. Whitaker & Etlinger, 1993: 567). On the basis of these pathological findings Wernicke (1874) questioned for the first time the existence of only one language center.

Consecutively, Wernicke published his influential work 'Der aphatische Symptomenkomplex. Eine psychologische Studie auf anatomischer Basis' in 1874. In this paper Wernicke differentiated for the first time the motoric and the sensory language areas and respectively motor and sensory aphasia from each other.\(^42\)

\(^{42}\) Whereas Whitaker and Etlinger (1993) try to prove that already Meynert (1866) gives evidence for the anatomical basis of language comprehension in the superior temporal gyrus and thus Wernicke (1874) as his student mainly copied Meynert's ideas, Eling (2006) criticised this assumption and points out that Meynert did not address sensory aphasia and deficits in language comprehension, in this paper. Nevertheless, Meynert influenced Wernicke's knowledge of neuroanatomical brain structure such as the fibre structure of the brain, as mentioned before.
A decade later, in 1885 Wernicke published 'Recent works on aphasia', which is not primarily concerned with a critical review of the aphasia literature. It contains the clearest and most detailed exposition of his concept of Wernicke's aphasia. Based on Wernicke's assumptions about language and the brain, a classical model of aphasia started to develop. A brief description of this model of aphasia will be presented below.

2.2.1 The classical model of aphasia

Under Wernicke's (1874) assumption, language is a learned behavior. During language acquisition the child is always exposed to language by listening. Thus the child stores the sound image of words in the sensory language center of the brain. By producing words, the child stores the motor image of the word in the motor language center of the brain. When speaking, these two centers are interconnected with each other by conduction pathways in a way that each acoustic image arouses the corresponding combination of motor movements. This leads to the following schema of speech apparatus, as shown below. A center 'a' located in the central projection area of the acoustic nerve, around the fossa Sylvii (sensory language center), contains the memory images of the 'acoustic speech sounds'. A center 'b' is situated in the language motor zone of the cortex and is the deposit of motor speech representations to the corresponding sounds. Center 'a' and center 'b' are interconnected with each other by means of an association pathway (cf. Wernicke, 1874: 19).44
While the destruction of center 'a' leads to sensory aphasia (Wernicke's aphasia) that is loss of speech comprehension with preservation of motor speech production, the destruction of center 'b' causes motor aphasia (Broca's aphasia) with intact speech comprehension but loss of motor speech production. For the disruption of the pathway 'a-b', with preservation of center 'a' and center 'b', Wernicke (1885) has designated the name conduction aphasia (cf. Wernicke, 1885 cited from Eggert, 1977: 174f).

In 1885, Lichtheim elaborated Wernicke's (1874, 1885) basic model of aphasia, by adding new categories of his own. Because his model is an amplification of the original schema of Wernicke, it is often referred to as the Wernicke-Lichtheim model (cf. De Bleser, 2001: 7). During the 1880s under Lichtheim, the introduction of two new labels into language models, took place and thus word-concept and transcortical language function (and also the concept of transcortical aphasia) were defined.

In Lichtheim's model of spoken language, language is also understood as a learned behavior, which is dependent on the two main centers, sensory (A) and motor (M), as previously stated by Wernicke (1874) as center 'a' and center 'b'. Lichtheim (1885) supposed a third center (B), the 'concept center' or 'center of object representations', which gives meaning, 'intelligence' to the words produced. Within the circuit of spoken language, there also exist, (as already mentioned by Wernicke, 1885), two afferent components, 'a' and 'm', coming from A and M. These centers are responsible for transmitting acoustic impressions of the word (a) as well as motor impulses (m) to the ear, as well as to the organs of motor articulation.

These centers predicted, are interconnected with each other in 7 different ways, as exemplified in the diagram below (cf. Lichtheim, 1885: 435f).
Picture 4 shows Lichtheim's model of spoken language (from Lichtheim, 1885: 437)

Interruptions of these connections, caused for instance by brain lesions, result in seven possible aphasia types, depending on which connection is disrupted: cortical sensory aphasia (1), subcortical sensory aphasia (2), transcortical sensory aphasia (3), cortical motor aphasia (4), subcortical motor aphasia (5), transcortical motor aphasia (6) and conduction aphasia (7).

Lichtheim (1885) abandoned Wernicke's (1885) classification (as given above) and recommended the following confusing nomenclature instead: nuclear speech-deafness (1), peripheral conduction speech-deafness (2), central conduction speech deafness (3), nuclear aphasia (4), peripheral conduction aphasia (5), central conduction aphasia (6) and conduction aphasia (7) (cf. Eggert, 1977: 182f).

Additions to the old terms motor, sensory and conduction aphasia are cortical or subcortical. Cortical refers to centers a (A) and b (M) which are themselves destroyed. Against that, subcortical refers to the conduction pathways on either side of a (A) and b (M) (cf. Eggert, 1977: 183).
Table 2 shows aphasia classification and symptom descriptions adapted from Lichtheim (cf. 1885: 438-460) and Wernicke 1885 in Eggert (cf. 1997: 183ff)

<table>
<thead>
<tr>
<th>Aphasia classification</th>
<th>Cortical motor</th>
<th>Cortical sensory</th>
<th>Conduction</th>
<th>Transcortical motor</th>
<th>Subcortical motor</th>
<th>Transcortical sensory</th>
<th>Subcortical sensory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesion in M</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
</tr>
<tr>
<td>Lesion in A</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
</tr>
<tr>
<td>Connection of M-A</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
</tr>
<tr>
<td>Connection of B-M</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
</tr>
<tr>
<td>Connection of M-m</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
</tr>
<tr>
<td>Connection of A-B</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
<td>violation no</td>
</tr>
<tr>
<td>Connection of A-a</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
<td>violation yes</td>
</tr>
</tbody>
</table>

Violation: yes/no
2.2.2 Modifications of the classical model of aphasia

A number of modifications (especially as far as terminology is concerned) were introduced in the second half of the 19th century and the beginning of the 20th century. A new period of aphasiology started with the work of Norman Geschwind in 1970 (see also Geschwind, 1972), during which the original Wernicke-Lichtheim aphasia classification from 1885 was reconsidered and modified. Geschwind pointed out the relevance of the classical model of aphasia to modern neurology and noted the presence of the arcuate fasciculus, a fibre tract that connects the motor and sensory language areas with each other. Geschwind, moreover, supposed the interruption of this fibre tract leading to the syndrome of conduction aphasia. By revising Wernicke's concept of conduction aphasia (interruption of the connection M-A), Geschwind's contribution to Wernicke's original concept is thus called the 'Wernicke-Geschwind' model of language (cf. for instance Kolb & Whishaw, 2003: 496; Bear, Connors & Paradiso, 2007: 625f).

The neurologists Norman Geschwind, Harold Goodglass, Davis Benson and Edith Kaplan in the USA (= Boston school of aphasia) (e.g., Benson & Geschwind, 1971; Goodglass & Kaplan, 1972; Goodglass & Geschwind, 1976) as well as Walter Huber, Klaus Poeck, Dorothea Weniger and Klaus Willmes in Germany (= Aachen school of aphasia) (e.g., Huber, Poeck & Willmes, 1984) followed this syndrome based localistic approach of aphasia and considered the various aphasic symptoms to be a homogeneous syndrome complex that can be tested with the help of standardized language assessments (cf. De Bleser, 2001: 15f) such as the Boston Diagnostic Aphasia Examination (= BDAE) (Goodglass & Kaplan, 1972), the Western Aphasia Battery (= WAB) (Kertesz, 1982) or the Aachener Aphasia Battery (= AAT) (Huber, Poeck & Willmes, 1984).

Until then, attempts had been made to define a fixed number of clinically recognizable aphasic groups, according to numerical values as obtained by various tasks within

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48 Reil (1808, 1812) was the first who identified the group of fibres running deeply into the white matter of the temporal, parietal and frontal region of the brain, located around the Sylvian fissure of each hemisphere. In 1822 Burdach named this perisylvian fissure the Fasciculus arcuatus (= arcuate fasciculus). It was Constantin von Monakow, who first identified the arcuate fasciculus as the tract connecting Broca's and Wernicke's areas. This view has been accepted by Wernicke in 1908 and was introduced again by Geschwind in 1970 (cf. Catani & Mesulam, 2008: 956).
language batteries. Based on a numerical taxonomy (Kertesz & Phipps, 1977), objective test scores and clusters of aphasic syndromes could be provided, free from previously intuitive classification methods, such as fluency and non-fluency classification systems (cf. Kertesz, 1979: 14).

However, until now there has not been a complete consensus on how the various manifestations of aphasia should be classified and hence also no aphasia classification has gained universal acceptance yet. Numerous classification systems can be found in the literature, which indicate that none of them are completely satisfactory. Kertesz (1979: 1) argued that „Most clinicians will agree that although aphasic disability is complex, many patients are clinically similar and will fall into recurring identifiable groups. [...] Indeed there is a need for classification“.

Thus, most authors do agree that there are eight major types of aphasia: Broca's aphasia, Wernicke's aphasia, conduction aphasia, global aphasia, anomic aphasia, transcortical motor, transcortical sensory and mixed transcortical aphasia. An example for a recent classification of these aphasia syndromes, as established for instance by Benson and Geschwind (1971), Goodglass and Geschwind (1976), Benson (1988) and Goodglass (1993), is given in Table 3.

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49 Using fluency as a measure of classification, Goodglass and Kaplan, 1972 stated the following: „The major subdivision among the aphasic syndromes is based on the character of the speech output. [...] The [...] speech interrupted, awkwardly articulated with great effort is referred to as „nonfluent“. The contrasting „fluent“ forms of aphasia are marked by facility in articulation and many long runs or words in a variety of grammatical constructions, in conjunction with word-finding difficulty for substantives and picturable action words“ (Goodglass & Kaplan, 1972: 54).
Table 3 shows a recent aphasia syndrome classification summarised and adapted from Benson and Geschwind (1971), Goodglass and Geschwind (1976), Benson (1988) and Goodglass (1993).

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>violation</td>
<td>violation</td>
<td>violation</td>
<td>violation</td>
<td>violation</td>
<td>violation</td>
<td>violation</td>
<td>violation</td>
</tr>
<tr>
<td>fluent spontaneous speech</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>repetition</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>auditory comprehension</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>reading aloud</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>reading comprehension</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>writing</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>wordfinding &amp; naming</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

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50 In Broca's aphasia the Broca's area in the posterior inferior frontal lobe of the language dominant left hemisphere, is affected (cf. Benson, 1988: 270).
51 In Wernicke's aphasia the Wernicke's area in the posterior superior portion of the temporal lobe of the language dominant left hemisphere is affected (cf. Benson, 1988: 272).
52 From all aphasia types, Anomic aphasia, is the type with the least predictable lesion site (cf. Goodglass & Geschwind, 1976: 418) and results of diverse causes (cf. Goodglass, 1993: 214).
53 Extensive brain damage involving Broca's and Wernicke's area as well as the whole perisylvian area, leads to global aphasia (cf. Benson, 1988: 273).
55 Lesions, linking the Broca's area with the supplementary motor areas and limbic structures that are essential for the initiation of speech, result into transcortical motor aphasia (cf. Goodglass, 1993: 213).
56 Transcortical sensory aphasia results from deep going lesions in a zone inferior to the angular gyrus and between the posterior end of the Sylvian fissure and the temporal-occipital junction (cf. Goodglass, 1993: 214)
57 Lesions in the middle cerebral and the anterior and posterior cerebral artery territories as well as lesions in the subcortical tissues are responsible for causing mixed transcortical aphasias (cf. Benson, 1988: 274)
2.2.3 Challenging the classical model of aphasia

The increasing knowledge of language, by the foundation of linguistics and linguistic theory (Bloomfield, 1933; de Saussure, 1959 cited after de Saussure, 1974; Chomsky 1965; Trubetzkoy, 1967; Chomsky & Halle, 1968; Jakobson, 1968; Jakobson, 1971; Bloomfield, 1983) on the one hand and the establishment of psycho-neurolinguistic language models (e.g., Morton, 1970; Marshall & Newcombe, 1973; Butterworth, 1980; Garrett, 1980 & 2000; Patterson, 1988; Levelt, 1989 & 1999; Cholewa, 1993; De Bleser, 2004) on the other hand, have helped to understand that language is a very complex process that involves more than just 'reception' and 'production' mechanisms, as previously suggested by Broca, Wernicke and Lichtheim (cf. Dronkers & Larsen, 2001: 20).

Furthermore, with the help of neuroimaging techniques, it is now possible to combine results from language assessments with excellent localization information about brain lesions.

The sum of these facts makes it difficult to maintain the assumption that only a small number of fixed subtypes of aphasia (e.g., Broca's aphasia) exist and that these syndromes are associated only with a few isolated language centers (e.g., Broca's area for language production).

2.2.3.1 Why traditional aphasia classifications are useless

The classical aphasia batteries, like the BDAE or the WAB, based on the classical model of aphasia, have shown to be useless. Whitaker (1984) for instance reported that only 60% of all aphasia patients can be classified with this model. Similarly, Caramazza (cf. 1984: 18) argued that the defined aphasia symptoms are not specific enough. The term, 'comprehension deficits' is too general and must be specified as types of comprehension deficits (e.g., syntactic comprehension deficit), in order to be useful. Schwartz (cf. 1984: 7) strengthened on the other hand that there is no need for a classification into particular aphasia syndromes at all, as some aphasic characteristics are included in several aphasia syndromes. For instance, a failure in syntactic parsing is not just included in Broca's aphasia, but also in Wernicke's aphasia.
Further factors which cannot be explained by traditional aphasia models, include double
dissociations between the retrieval of grammatical categories, such as verbs and nouns
(Caramazza & Hillis, 1991; Damasio & Tranel, 1993; Perani et al., 1999; Bird, Howard
& Franklin, 2000; Humphreys & Forde, 2001; Neininger & Pulvermüller, 2003; Hauk,
Johnsrude & Pulvermüller, 2004; Hauk & Pulvermüller, 2004), double dissociations
concerning disturbances in naming and comprehension of selective semantic categories
of concrete nouns, such as animals, fruits, vegetables and so forth (Warrington &
McCarthy, 1983; Warrington & Shallice, 1984; Hillis & Caramazza, 1991) and the
evidence of progressive language impairment with the isolation of specific semantic
categories (Basso, Capitani & Laiacona, 1988).
Thus, syndrome based aphasia classification is disappearing more and more from
clinical praxis and is replaced by a linguistic founded single case approach, which tries
to explain linguistic changes by the implementation into language processing models,
such as LeMo (De Bleser, 2004) for German (for a brief introduction see for instance
Aichert & Kiermeier, 2005).

2.2.3.2 Why Broca's area and Broca's aphasia are not necessary 'direct proportional'

Marie (1906) was among the first to question Broca's assumption of the direct
correlation of Broca's area and Broca's aphasia. Marie criticized the classical model of
aphasia by evidencing that there are patients who can talk without any difficulty,
although the left frontal convolution is destroyed. Additionally, in a number of Broca's
aphasics no lesion of the third frontal convolution could be found. Thus, Marie stated
that “the third left convolution plays no special role in the function of
language” (Marie, 1906: 59). Moreover, Marie noted that the disorder described by
Broca was not just related to lesions in Broca's area, but rather to lesions involving more
medial brain structures, such as the insula and the basal ganglia. This assumption could
further be proved by Lhermitte et al. (1973). Lhermitte and colleagues reported the
coexistence of fluent and non-fluent speech in one patient, with an extensive lesion into
Broca's area in addition to a posterior lesion, involving Wernicke's area and the
fasciculus arcuatus, interconnecting Broca's and Wernicke's area. Mazzocchi and
Vignolo (1979) reported moreover a patient with destroyed Broca's area but
nevertheless suffering from Wernicke's aphasia. Another patient with slight Broca's aphasia has been noted to suffer from lesions extending far into the entire Wernicke's area. Patients suffering from global aphasia with preserved Wernicke's area have further been reported.

The statement, that only Broca's area is responsible for establishing Broca's aphasia symptoms has started to become further challenged, by using neuroimaging techniques such as CT and MRI (for an overview on functional neuroimaging of language see for instance Damasio, 1989; Small & Burton, 2001; Friederici, 2001; Thompson, 2005).

For instance, Signoret and colleagues (1984) pointed out that Paul Broca had made his descriptions of the neuroanatomical correlates of language solely on visual assessments of his patients’ brains. More specifically, after Broca's historic paper in 1861 he deposited the brains of Leborgne and Lelong in the Dupuytren Museum (Paris), without having sliced or drawn them.

Picture 5 shows the lateral view of Broca’s first patient, Monsieur Leborgne (from Dronkers et al., 2007: 1436)
As the brains have been preserved in the Paris School of Medicine, recent neuroimaging investigations have been started to rediscover the neuroanatomic correlation of Broca's aphasia (cf. Signoret et al., 1984: 304f). The research group around Signoret (1984) established an anatomical description of Leborgne's brain, using CT scans. Brain analysis revealed no lesion involving Wernicke's area, in Tan's brain, supporting Broca. However, widespread damage to frontal, temporal, insular and subcortical brain areas of the left hemisphere could be found, additionally to the lesion in the third left convolution, described by Broca (cf. Signoret et al., 1984: 305f).

In 2007 Dronkers et al. had the unique opportunity to scan both brains (Leborgne's and Lelong's), by using high-resolution MRI. Massive destruction of grey and white matter in the left hemisphere of both brains could be detected. Both patients' lesions extended significantly into medial regions of the brain, in addition to those surface lesions described by Broca in 1861. A comparison of the two brains revealed that both had lesions in the arcuate fasciculus, connecting posterior and anterior brain areas.

Moreover, complete destruction of the deep frontal and parietal areas was ascertained. Especially in Leborgne' brain there was extensive subcortical involvement, including the claustrum, putamen, globus pallidus, head of the caudate nucleus and the internal as well as the external capsula.

Thus, it is evident that lesions to Broca's area alone were not to cause the long lasting, severe speech arrests described by Broca.
It is more likely that the mutism of Broca's patients was influenced by lesions in the white matter tracts and subcortical areas (cf. Dronkers et al., 2007: 1436ff). Evidence of this unproportional relationship between Broca's are and Broca's aphasia could further be shown in various other studies, examining different patients. For instance, a CT study by Poeck, De Bleser and von Keyserlingk (1984) showed that the lesion of Broca aphasics was predominantly in or extended to Wernicke's area and vice versa. Moreover, lesions of Broca's aphasics rarely involved the foot of the third frontal convolution but included the rostral insula and reached into the frontal white matter, above Broca's area (cf. Poeck, De Bleser & von Keyserlingk, 1984: 75).

Basso et al. (1985) studied the anatomical correlations of 207 aphasic patients, as defined through CT. The study revealed that 36 out of 207 aphasic subjects tested, did not support the classical model of aphasia. There could be reported three cases without aphasia but with important lesions involving the classical speech area, two cases with aphasia with lesions outside of the classical language zone, ten patients suffering from global aphasia in the absence of injury to Wernicke's area, eight cases with Wernicke's aphasia due to large lesions extending beyond Wernicke's area, six cases with fluent aphasia after anterior lesions and six cases with nonfluent aphasia after posterior lesions (Basso et al., 1985: 214-224). Willmes and Poeck (cf. 1993: 1534) found an even weaker relationship between lesion sites and aphasic syndromes. For the fact that Wernicke's aphasia would result from damage to Wernicke's area a probability of only 0.48 % has been stated and a probability of only 0.35 % could be shown for Broca's aphasics, whose language disturbance resulted from damage to Broca's area.

Tying into these findings, Mohr et al. (cf. 1975: 349) suggested that the language disturbance resulting from infarctions limited to Broca's area differs from the language disorder called Broca's aphasia, which does – against the dated view – extend far outside Broca's area. Whereas a restricted Broca's area infarction does not cause Broca's aphasia, infarctions affecting Broca's area and its immediate surroundings cause long lasting language disturbances such as mutism, verbal stereotypes and agrammatism. Mohr (1978) moreover stated on the basis of precise anatomical correlations using CT imaging, that the area of brain damage causing Broca's aphasia is diffuse and not restricted to the left frontal convolution and thus concluded „since the term Broca aphasia specifies neither the topography nor the temporal course of the lesion in the individual case, a revised terminology seems appropriate“ (Mohr, 1978: 323).
To avoid further ambiguities, Mohr (2006: 391) proposed that „[... the term[s] Broca's aphasia [...] might preferably fall into disuse, in favor of other terms, [...] that better describe the spectrum observed“.

Even more critical is the assumption that Broca's area itself may not exist (Ullman, 2006). Whereas Broca's area is typically defined in terms of the pars opercularis and the pars triangularis of the third frontal convolution in the inferior frontal lobe, represented in Brodmann's (1909) cytoarchitectonic map as areas BA 44 (pars opercularis) and BA 45 (pars triangularis), Ullman (2006) stated that the so called Broca's area does not exist in isolation and is rather part of the cortico-basal ganglia-thalamo-cortical circuits.

Further challenging the concept of Broca's area, Hagoort (2005) as well as Keller's et al. (2009) pointed out that Broca's area is not a very well defined concept, addressing the big variance in gross anatomy of Broca's area between people and summarizing how definitions of this brain area vary between studies. In a similar way Lindenberg, Fangerau and Seitz (2007) analysed within a meta-analysis paper the term Broca's area and could show that not only the anatomical regions assigned to Broca's area, but also the understanding of the underlying concept, differs widely between studies. The scope of Brodmann areas associated with the classical Broca area range from Brodmann area 6 to 46, including Brodmann areas 44,45 and 47. Additionally, the functions assigned to Broca's area range from an expressive function only, to a motor speech center, to supposed fractions of the Broca's area for receptive and expressive language functions in the domains of semantics, syntax and phonology (cf. Lindenberg, Fangerau & Seitz, 2007: 24f).

In conclusion, the notion of circumscribed language centers, as stated at the beginning of the 19th century, is dated today. Modern concepts of the organization of language functions support the theory of multiple representations and cerebral networks (e.g., Mesulam, 1990; Knight, 2007). In the sense of multiple representations, in one circumscribed brain area more than one function may be presented. Single brain areas – so far if there exists something like that – are on the other hand supposed to be linked via cerebral networks. Thus it seems logical that a circumscribed brain lesion may lead to more than one functional impairment and vice versa, a certain symptom may be the result of a lesion in several brain regions. For that reason it also seems evident that cortical and subcortical structures are interconnected with each other. Thus it seems evident that not only cortical but also subcortical brain structures, such as the basal
ganglia, participate in language. Similarly, Broca's area is interconnected with other cortical brain structures, which supposedly fulfill the same functions (Ullman, 2006) and thus these areas together participate in linguistic functioning.

Before going into further detail concerning supposed network and circuit models dealing with linguistic functioning, the following chapter addresses the hypothesized role of the subcortical structures in language.

2.3 Subcortical participation in language – towards the concept of subcortical aphasia

"For the past 200 years, virtually all attempts to account for the natural bases and the evolution of human language have focused on the neocortex" (Lieberman, 2001: 32)

However, as exemplified above, the classical cortical model is not able to account for all language disturbances found. Thus Crosson (1985: 263) stated that "[...] the role of subcortical structures in language may explain phenomena which are unexplained by the classical model of aphasia".

Even back in 1906, Marie suggested that the lentiform nucleus has a core role in language (cf. Marie, 1906: 66f). The first systematic study of the effects of subcortical lesions upon language, was undertaken by Marie's student, Moutier, in his doctoral thesis, back in 1908. Within his work he described three patients whose brain lesions were found at autopsy not to extend into the cortex, but nevertheless were all non-fluent aphasics (cf. Moutier, 1908 cited in Wallesch & Papagno, 1988: 256).

Subsequently, the classical cortical language model was challenged by stereotactical surgery. Using stereotactical techniques, Penfield and Roberts (1959) proposed that the thalamic nuclei such as the pulvinar, connect the two classical language areas and thus have a core linguistic functioning (cf. Penfield & Roberts, 1959: 207). Furthermore, the effect of electrical stimulation of the ventrolateral thalamus on object naming was studied by Ojemann and Ward (1971), proving the results of Penfield and Roberts (1959), concerning thalamic language functioning. The existence of a definable syndrome of aphasia associated with thalamic hemorrhage has been suggested furthermore, beginning with Fisher in 1959, followed by several other authors, such as Luria (1977), Reynolds et al. (1979), Alexander and LoVerme (1980), Crosson et al.
As well as more recently by Crosson (1986), (1992) and (1999). An attempt to formulate a unique pattern of language deficit following thalamic aphasia has been made by Crosson (1984). Crosson assumed that thalamic aphasia is associated with fluent language output, accompanied with substitutional, perseverative as well as paraphasic errors; sometimes even deteriorating into a meaningless jargon. And yet, there is lesser impairment of language comprehension and repetition (cf. Crosson, 1984: 496).

Thus it can be concluded that the thalamus is associated with language. Given, that the thalamus is the main brain structure regulating impulses from the basal ganglia to the cortex, within cortico-basal ganglia-thalamo-cortical circuits (as discussed in detail in chapter 1.4.1), this structure has to be included within this work in order to comprehensively discuss basal ganglia functions in language. A brief description of the thalamic anatomy, as well as a literature review addressing the main three thalamic nuclei participating in language, are given in Appendix II.

In contrast to a thalamic association with language, theories concerning the participation of the dominant (left) basal ganglia in language, have not been profound enough. Nevertheless, the concept of subcortical aphasia with respect to lesions of subcortical areas, appeared in the literature as an equivalent to the concept of cortical aphasia (introduced by Broca, 1861 and Wernicke, 1874) in 1906, when Marie published his influential article, entitled 'Que faut-il penser des aphasies sous-corticales?'. Marie's suspicion of a subcortical participation in language could be proved by the introduction of methods for the in vivo imaging of brain lesions, such as computed tomography, in the 1970s. Consecutively the number of studies concerning the role of basal ganglia structures in language, started to increase. Numerous case reports of patients who developed aphasic like syndromes from lesions of subcortical structures, such as the basal ganglia, have been added to the literature. Indicating that aphasic syndromes could be observed also in patients with subcortical lesions, the term subcortical aphasia was mentioned to be suitable (cf. Cappa, 1997: 424).

“[...]This term is now used for those language disturbances resulting from a lesion of the deep nuclei of the cerebral hemispheres, the basal ganglia or the thalamus” (Wallesch & Papagno, 1988: 256)
Addressing the concept of subcortical aphasia, Vignolo (cf. 1984: 96) pointed out, that in addition to the classical language areas of Broca and Wernicke, subcortical regions of the left hemisphere, such as the basal ganglia, also play an important role in language processing. This assumption has for instance been supported by Naeser and Hayward (1978), Kertesz, Harlock and Coates (1979), Kornhuber, Brunner and Wallesch (1979) as well as by Mazzocchi and Vignolo (1979).

Given, that this thesis addresses language functions in HD as a basal ganglia disorder, focus hereinafter will exclusively be on theories concerning basal ganglia functions in language, neglecting other subcortical structures and their connection to language functions.

In detail, on the basis of the literature, as will be presented next, several mechanisms can be summarised, which enable us to explain how lesions in the basal ganglia can lead to language changes, such as subcortical aphasic syndromes:

1) Language changes in patients with subcortical lesions (e.g., basal ganglia lesions) account for a direct involvement of these structures in language.

2) More recently, it has been suggested that lesions in the connection (= disconnection) of subcortical areas (respectively focusing herein on the basal ganglia) with the classical language areas in the cortex (Broca's and Wernicke's area) result in a 'distance effect' of the cortex from the basal ganglia and hence contribute to language changes such as aphasic syndromes. Different patterns of disconnection thus lead to different profiles of aphasia.

3) The 'distance effect' of subcortical structures (respectively focusing herein on the basal ganglia) from the cortex, is supposed to produce depressed metabolic functioning in the traditional language zones of the cortex and consecutively leads to diaschisis effects and linguistic dysfunctioning.

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58 This terminology dates back to Norman Geschwind (1970), who noted for the first time the presence of the arcuate fasciculus, a fibre tract that connects the motor and sensory language areas. He proposed further that a disruption of this fibre tract leads to a disconnection of the motor from the sensory language area and vice versa. This terminology is still used nowadays, to give a possible explanation why language disturbances after basal ganglia lesions can occur (cf. Schmahman & Pandya, 2008: 1038).

59 Probably the best description of the diaschisis effect has been provided by Von Monakow (1969: 27-36) who defined this condition as „the loss of excitation, resulting in a cessation of function, in areas remote from yet connected to a primary site of brain damage“.
More evidence proving the role of the basal ganglia in language comes from linguistic disturbances – even though not always classifiable on traditional aphasia schemes – in the course of neurodegenerative basal ganglia disorders, such as PD and HD. These factors will be addressed in depth, within the next subchapters.

2.3.1 Direct involvement of subcortical structures in language

Language changes in patients suffering from subcortical lesions (e.g., basal ganglia lesions) account for a direct involvement of these structures in linguistic functioning. To prove this assumption, Damasio et al. (1982) showed that there is a powerful association between aphasia and a lesion in a well-circumscribed anatomic region such as the anterior limb of the internal capsule and both the head of the caudate nucleus and the anterior aspect of the putamen.

Caudate infarcts, extending into the internal capsule and the putamen, producing language abnormalities such as aphasia, have also been reported by Caplan et al. (1990) as well as by Kirk and Kertesz (1994). Specifically, a variety of aphasic like symptoms have been mentioned, that are caused by different subcortical lesions (Basso, Salla & Farabola, 1987). Even minimal differences in the localisation of the localisation (a few millimeters are enough) can cause different aphasic symptoms (cf. Crosson, 1985: 286).

Descriptions of the variability of subcortical aphasias, due to different lesion sites, are given for instance by Damasio et al. (1982), Naeser et al. (1982), Wallesch et al. (1985), Alexander, Naeser and Palumbo (1987), Naeser (1988), Kennedy and Murdoch (1989) as well as by D'Esposito and Alexander (1995). In detail, Naeser et al. (1982) as well as Naeser (1988) distinguished between three aphasic concepts, resulting from lesions involving the putamen and the internal capsule: capsular/putaminal lesions with anterior-superior, posterior as well as anterior-superior and posterior white matter extension. In detail, anterior-superior lesions led to a language profile specific for Broca's aphasia (Broca's aphasia symptomacity is exemplified in Table 3), but inconsistent with Broca's aphasia in terms of an almost normal phrase length, intact

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60 Anterior-superior white matter extension reached in detail from the periventricular white matter and the corona radiata lateral to the body of the lateral ventricular white matter and corona radiata lateral to the body of the lateral ventricle and deep to Broca's area (Naeser et al., 1982; Naeser, 1988).

61 Posterior white matter extension reached from the area inferior to the Sylvian fissure and superior to the temporal horn (Naeser et al., 1982; Naeser, 1988).
grammatical form and sentence production. Capsular/putaminal lesions with posterior white matter lesion extension resulted in a language profile seen in Wernicke's aphasia. Nevertheless, capsular/putaminal lesions with posterior white matter lesion extension were dissimilar to Wernicke's aphasics, regarding a right hemiplegia. Capsular/putaminal lesions with both anterior-superior and posterior lesion extension led to a language profile similar to global aphasia with mutism and the production of stereotypes, such as 'yes,yes' (cf. Naeser, 1988: 365). Peach and Tonkovich (1983) evidenced three cases suffering from aphasia after subcortical damage, matching the syndromes described by Naeser et al. (1982) and Naeser (1988). Support for a possible distinction between anterior and posterior subcortical lesions (Naeser et al., 1982; Naeser, 1988) has further been given by Cappa et al. (1983). Anterior subcortical lesions, including the putamen and the anterior limb of the internal capsule, could be associated with atypical non fluent aphasia, whereas posterior subcortical lesions, including the putamen and the posterior limb of the internal capsule could be associated with mild fluent aphasia (cf. Cappa et al., 1983: 240).

Various other aphasic like symptoms, due to basal ganglia lesions have been indicated by Wallesch et al. (1983). In detail, long lasting language deficits in articulatory, syntactic and lexical functions after left basal ganglia lesions have been reported. Selective deficits in syntactic comprehension associated with bilateral damage to the putamen and the head of the caudate nucleus have also been stated by Pickett et al. (1998). Similarly, persistent aphasic symptoms including an impairment in auditory and reading comprehension, naming, repetition, reading, writing and spontaneous speech have been noted, following a lesion within the striato-capsular region, by Murdoch et al. (1991). Fabbro, Clarici and Bava (1996) found moreover reduced spontaneous speech, agrammatic errors such as omissions of full verbs and substitutions of inflectional morphemes, semantic as well as verbal paraphasias, neologisms and echolalias in patients with basal ganglia lesions. In addition, Brunner et al. (1982) evidenced that aphasic syndromes are more severe when the cortical lesion extended into the basal ganglia. Interestingly, some aphasic symptoms, such as automatisms and recurring utterances only occured in patients tested, if there were combined cortical and basal ganglia lesions (cf. Brunner et al., 1982: 293; see also Kornhuber, Brunner and Wallesch, 1979 as well as Aram and Eisele, 1993).
Evidence for a direct involvement of subcortical structures, such as the basal ganglia in language, as just presented, have been developed further. As the basal ganglia (especially the striatum, as the main input area of the basal ganglia) receive input from literally all major sensory and multimodal regions of the cortex, it is assumed more recently, that they act as a kind of high level integrator of information, including language-related processes (cf. Robin & Schienberg, 1990: 98). Thus it seems evident that it is through the basal ganglia's role as an integrator of complex information, as well as a modulator of cortical information (cf. Mehler, 1988: 26f), that lesions in these areas produce aphasic like symptoms. The striatum's neuroregulatory and neuromodulatory function in language processes has also been addressed in Crosson's (1992) article 'Is the striatum involved in language?'.

Thus, it is assumed nowadays that not subcortical structures, such as the basal ganglia alone, but the disruption of their connection to the language areas in the cortex (= disconnection resulting in a 'distance effect'), might be crucial for developing language disturbances in subcortical patients. This 'distance effect' of the cortex from subcortical areas and vice versa, is addressed next.

2.3.2 The 'distance effect'

More recently, it has been suggested that lesions in the connection (= disconnection) of subcortical areas (respectively focusing herein on the basal ganglia) from the classical cortical language areas, result in a 'distance effect' of the basal ganglia from the cortex, and hence contribute to language changes such as aphasic syndromes. Different patterns of disconnection thus lead to different profiles of aphasia.

This theory is based on the assumption that the cortex and the subcortical structures (e.g., the basal ganglia) do not work in separation from each other. More likely, they are interconnected with each other by means of cortico-basal ganglia-thalamo-cortical circuits (Albin, Young & Penney, 1989), as suggested for motor functioning within chapter 1.4.1. In addition to the classical motor circuit, further circuits have been proposed, supposedly involved in higher cognitive functions (e.g., Alexander, DeLong & Strick, 1986; Alexander & Crutcher, 1990; Alexander, Crutcher & DeLong, 1990; Cummings, 1993; Gurney et al., 2004; McHaffie et al., 2005) and even in language (e.g., Crosson, 1985; 1992a; 1992b; Lieberman et al., 1992; Crosson et al., 1997; Pickett...
et al., 1998; Lieberman et al., 2001; Lieberman et al., 2002; Crosson & Haaland, 2003; Whelan et al., 2003a; Whelan et al., 2003b; Whelan et al., 2004; Lieberman, 2008) (for further details see chapter 2.3.5). These language related models suggest that not only cortical areas, such as the Broca's area, but also subcortical brain structures, like the basal ganglia, do participate in language functioning. This has for example also been attributed by Ullman (2006), suggesting that Broca's area is part of a cortico-basal ganglia-thalamo-cortical circuit. Interactions between the frontal cortex and the basal ganglia have further been outlined by Frank et al. (2001). A distributed network model of language organization has also been stated by Mesulam (cf. 2000: 37f). Within this model, distributed language networks, integrating association areas of the frontal, temporal and parietal lobes, as well as the thalamus and the basal ganglia, to classical cortical areas, such as Broca's and Wernicke's area, are assumed.

As already addressed previously in chapter 1.4.1, the striatum can be regarded as the main input area of the basal ganglia, receiving input from literally all major sensory and multimodal regions of the cortex (Alexander & Crutcher, 1990). Thus it can be concluded that the basal ganglia act as a kind of high level integrator of information, including also language-related processes. Thus it seems evident that lesions in the connection of the basal ganglia and the cortex (and vice versa), produce aphasic like symptoms (cf. Robin & Schienberg, 1990: 98). Different patterns of disconnection are hence supposed to lead to different profiles of aphasia.

Duffau and Capelle (2001) as Duffau et al. (2002) could further prove this theory by evidencing specific subcortical language pathways during intraoperative mapping, using direct electrical stimulations. Stimulation of one or the other pathway resulted into various language disturbances. In addition to cortico-basal ganglia connections as well as subcortical language pathways, also connected to the cortex, reciprocal cortico-cortical connections have been reported by Yeterian and Van Hosen (1978). Based on the theory that the striatum is the main input area of the basal ganglia, it is hypothesised that it receives projections not only from one cortical region, but from all cortical areas reciprocally interconnected to that area (cf. Yeterian & Van Hosen, 1978: 61; Chesselet & Delfs, 1996: 417).

Further evidence for the theory of a 'distance effect' could be given by Mega and Alexander in 1994. This study group was able to identify a core profile of aphasia, in 17 patients, due to damage of three out of five frontostriatal circuits, stated by Alexander, DeLong and Strick, back in 1986. Language disturbances mainly included executive,
generative language functions, such as word fluency, sentence generation and discourse, but spared responsive language such as language comprehension, repetition and sometimes even naming, ranging from fluent to non-fluent aphasic categories. These symptoms are in accordance to the concept of dynamic aphasia, described by Luria and Tsevtkova back in (1968). Based on these assumptions and evidence, specific language models started to develop, based on classical cortico-basal ganglia-thalamo-cortical circuit models (see for instance the models developed by Crosson, 1985; Wallesch & Papagno, 1988; Whelan et al., 2003a; 2003b; 2004 as exemplified in chapter 2.3.5).

As previously mentioned, the 'distance effect' is supposed to produce metabolic dysfunctions in the traditional language zones of the cortex. These hypometabolic changes, as the result of an interruption between the cortex and subcortical structures, consecutively result into neuronal loss and aphasic syndromes. The product of 'distance effects' are discussed in further detail in the next subchapter.

2.3.3 'Distance effects' produce conditions of diachisis

The 'distance effects' of subcortical structures (including the basal ganglia) from the cortex, are supposed to produce depressed metabolic functioning in the traditional language zones of the cortex, which consecutively result in diachisis effects and linguistic dysfunctioning.

Metter et al. (1983a) as well as Metter et al. (1984) were among the first to show that lesions of the basal ganglia result in diachisis effects in the traditional language zones of the cortex, connected to the damaged area in the subcortical region (caudate nucleus and thalamus). In sum, data of these studies provide physiological evidence to support the theory that subcortical lesions contribute to abnormal cortical functioning and hence create aphasic symptoms. This theory was corroborated by Weiller et al. (1993), reporting aphasia after striatoCapsular infarction and selective neuronal loss of the cerebral cortex, due to prolonged insufficient rCBF. Hojer-Pedersen and Petersen (1989) evidenced that changes of the rCBF occurred after subcortical ischemic infarctions and thus contributed to aphasia. The effect of hypoperfusion onto the cortex has also been described by Kang et al. (2000), indicating that neuronal changes, such as necrosis,

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62 As this aphasia concept will also be of importance in order to interpret linguistic disturbances of HD patients made within this study, a detailed description of this aphasic concept is given in Appendix V.
occur in the cortex after subcortical infarction and consequently lead to aphasic syndromes. This hypothesis has been supported in a similar way by Olsen, Bruhn and Öberg (1986), Perani et al. (1987) as well as by Hillis et al. (2002), indicating that aphasic symptoms in subcortical patients result from diachisis effects, including cortical hypoperfusion. Furthermore, Hillis et al. (2004) stressed an association between the kind of language impairment and regions of cortical hypoperfusion. This study showed that patients suffering from acute left caudate infarct, exhibited Broca's aphasic symptoms, due to hypoperfusion in Broca's area. Contrastingly, impaired auditory comprehension has been associated with hypoperfusion of the posterior superior temporal gyrus (cf. Hillis et al., 2004: 526). Okuda et al. (1994) supported furthermore the theory of variability in subcortical aphasia due to variable sites of cortical hypoperfusion. Their study showed that cortical hypoperfusion, mainly in the perisylvian areas, including Broca's and Wernicke's areas was crucial for the development of subcortical global aphasia. A relation between aphasia severity and the degree of cortical dysfunction in subcortical aphasia, could further be shown by Vallar et al. (1988) as well as by Demeurisse (1997).

Nevertheless, the role of subcortical structures, including the basal ganglia, in the development of pure linguistic disturbances in the course of neurodegenerative diseases, such as PD & HD has further been proven. Before addressing language changes in HD, a brief overview will be given next, dealing with linguistic changes in the course of PD.
2.3.4 Subcortical participation in language – the example of Parkinson's disease

An additional support for the hypothesis that language changes occur in neurodegenerative, subcortical diseases such as HD, the example of Parkinson's disease (PD)\(^{63}\) can be seen. Similar to HD (as will be discussed in chapter 3), in PD there exists no uniform picture yet, concerning linguistic deficits in this subcortical disorder. Again, there is an inconsistency in language tests used, a lack of the consideration of genetic data as well as of the linguistic interpretation and analysis of the data. Nevertheless, PD patients have been reported to exhibit pure linguistic difficulties, especially in the domain of syntactic production and syntactic comprehension (Illes, 1988; Illes, 1989; Lieberman, Friedman & Feldman, 1990; Lieberman et al., 1992; McNamara & Durso, 2000). Lieberman et al. (1992) concluded that the syntactic comprehension deficit noted for PD patients, is similar to that found in Broca's aphasics, with an inability to apply syntactic rules. Sentence comprehension deficits, as well as sentence judgement deficits in PD patients could also be reported by Grossman et al., (1992a), Grossman et al. (1992b), Grossman et al. (1993), Geyer and Grossman (1994), as well as by McNamara et al. (1996). PD patients moreover displayed difficulties in verbal fluency (Henry & Crawford, 2004), in verbal inflection (Ullman et al., 1997a; Longworth et al., 2005), in verb generation (Péran et al., 2003b; Crescentini et al., 2008a), in semantic priming (Copland, 2003; Castner et al., 2007; Chenery, Angwin & Copland, 2008), in the reception of prosody (Lloyd, 1999) as well as in the understanding of metaphors (Monetta & Pell, 2007).

Even though it is not known yet if there is something like a core linguistic profile in subcortical diseases, it is obvious from the literature (PD and HD as will follow), that the basal ganglia must play a certain role in linguistic functioning. Defects within these areas, or deficits in the connection of these areas with other brain structures, are thus likely to contribute to linguistic deficiencies.

\(^{63}\) An important pathophysiological feature of PD is the loss of dopaminergic neurons in the substantia nigra, as exemplified in Figure Appendix I within Appendix I and thus resulting into hypokinetic motor disturbances (cf. Bartels & Leenders, 2009: 1). Thus it can be referred to as being just the opposite of HD with a hypokinetic motor syndrome (cf. Mendoza & Foundas, 2008: 182).
In addition to the five cortico-basal ganglia-thalamo-cortical circuits (e.g., Alexander, DeLong & Strick, 1986) as described in chapter 1.4.1, Middleton and Strick (cf. 2000: 194) evidenced that at least 9 other cortical areas (except the primary motor cortex), are the target of the basal ganglia's output. Basal ganglia output projections are believed to include subdivisions of the premotor, oculomotor, prefrontal and even the inferotemporal cortex. On the basis of these loops it is assumed that additionally to motor functions, an involvement of the basal ganglia in various emotional and cognitive functions can be suggested (cf. Murdoch, 2004: 140). Specifically, a learning-related function (Graybiel, 2005; Packard & Knowlton, 2002) as well as a role in memory (Packard & Knowlton, 2002) and focused attention (Brown & Marsden, 1998) have been reported. Moreover, it is suspected that the basal ganglia are crucial for pure linguistic tasks (e.g., Crosson, 1985; 1992a; 1992b; Lieberman et al., 1992; Crosson et al., 1997; Pickett et al., 1998; Lieberman, 2001; Lieberman, 2002; Crosson & Haaland, 2003; Whelan et al., 2003a; 2003b; Whelan et al., 2004; Lieberman, 2008).

Concerning the main aim of this thesis, to describe language changes in HD, as a disease of the basal ganglia, the role of the cortico-basal ganglia-thalamo-cortical loops in language will be addressed in detail next.

Even though motor models, as for instance explained in chapter 1.4.1, can be used in order to explain hyper- and hypokinetc forms of speech disturbances (hyper- versus hypokinetc dysarthria) in the course of basal ganglia disturbances (e.g., Murdoch, 2004), these models cannot be adjusted as easy to the domain of language and its disturbances. Nevertheless, there have been some attempts.

In detail, one of the first suggestions of the participation of cortico-striato-pallido-thalamo-cortical loops in language, can be found in the work of Brunner et al. (1982), in which Wallesch participated. Wallesch continued his work and published in 1985 a study of several patients with subcortical aphasias. The most complete version of Wallesch's theory has been published in 1988, in cooperation with Papagno. Wallesch and Papagno (1988) supposed the cortico-striato-pallido-thalamo-cortical loop to take part in the selection of lexical alternatives within language production. Details of Wallesch and Papagno's model are given in chapter 2.3.5.1.
After Wallesch, Crosson generated in 1985 an additional model, to explain subcortical functions in language. Even though the main focus of his work was on thalamic functions in language, he further stressed the role of the basal ganglia in linguistics. In detail, Crosson (cf. 1985: 269) stressed the existence of neuroanatomical pathways between cortical and subcortical structures implicated in language and pointed out that:

„It is very unlikely that the various cortical and subcortical structures involved in language operate in isolation from one another to produce the phenomena we know as language. It is more probable that these structures operate in an organized and coordinated fashion to produce and comprehend language“ (Crosson, 1985: 269)

Briefly, within Crosson's (1985) model the basal ganglia are described as having a regulatory function upon the cortex. If certain structures within the basal ganglia are defective, the cortex gets overactive and the inflow of irrelevant language material is the result. Details of Crosson's model are given in chapter 2.3.5.2.

Nevertheless, based on Wallesch and Papagno's as well as Crosson's model, further paradigmas have been presented, focusing on the cortico-basal ganglia-thalamo-cortical circuity. For instance, Ullman (2006) predicted Broca's area (BA 44, 45), as part of the frontal cortex, to be involved within the cortico-basal ganglia-thalamo-cortical circuit. Within Broca's area one channel is predicted to encompass pars triangularis and BA 45, while the other channel encompasses pars opercularis and BA 44. This hypothesis is based on the assumption that all frontal regions have been found thus far to receive inputs from the cortico-basal ganglia-thalamo-cortical circuits, suggesting that also the Broca's area is very likely to receive such inputs64.

Given, that all models, as just mentioned, only refer to the direct pathway within the cortico-basal ganglia-thalamo-cortical loop, the importance of the subthalamic nucleus within the indirect pathway of the cortico-basal ganglia-thalamo-cortical loop in the mediation of linguistic processes has moreover been added to contemporary basal ganglia models, explaining language by Whelan et al. (2003a; 2003b; 2004).

In conclusion, all of these models are hypothetical, referring to cortico-basal ganglia-thalamo-cortical loops stated for the first time by Alexander, DeLong and Strick, back in 1986. None of them is perfect, nor complete and may even be incorrect in one way or another. For instance, the models developed by Crosson (1985) and Wallesch and

64 Frontal regions have further been shown to receive basal ganglia projections comprising portions of the ventral premotor cortex, including area F5, which has been claimed to be homologues to BA 44 in particular (cf. Rizzolatti & Arbib, 1998: 188).
Papagno (1988) pointing out the direct role of the basal ganglia in language, are imperfect in the following: The model by Crosson (1985) holds on to localistic perspectives, such as the existence of distinct anatomical areas for language production and language comprehension. The model by Wallesch and Papagno (1988) on the other hand, implies a major role of the basal ganglia in selecting the most appropriate lexical alternative in language production, via internal motivational factors and external constraints, without determining what these constraints are.

Nevertheless, theoretical assumptions about the striatum's function in language and its defect in HD patients, will be strengthened in this thesis by the implementation of errors found, in Wallesch and Papagno's as well as Crosson's cortico-basal ganglia-thalamo-cortical circuit model of language functioning. Thus, a comprehensive description of these models will be given next.

2.3.5.1 Wallesch and Papagno's model of subcortical participation in language

Wallesch and Papagno's model of subcortical participation in language has been influenced by various research, as described in Appendix III.

Briefly, Wallesch and Papagno's (1988) model of subcortical participation in language is based on the following assumptions:

The cortex is organised in parallel modules of homogeneous functions (e.g., Mountcastle, 1957; Goldman-Rakic, 1984; Philipps, Zeki & Barlow, 1984). Cortical areas are assumed to be reciprocally interconnected with each other (Yeterian & Van Hoesen, 1978; Van Hoesen, Yeterian & Lavizzo-Mourey, 1981). Furthermore, the cortex and subcortical structures are hypothesised to be interconnected with each other by means of a loop involving the cortex, the striatum, the globus pallidus, the ventral lateral/ventral anterior thalamus as well as the frontal cortex (presented in chapter 1.4.1, as well as stated for linguistic functioning previously by Brunner et al., 1982; Damasio et al., 1982 and Wallesch et al., 1985). Furthermore, there are specific thalamic nuclei which are supposed to be involved in language (presented in Appendix II).

In Wallesch & Papagno's model, within cortical processing, competing lexical alternatives are processed in parallel modules that are transmitted from posterior to
anterior language cortices. Posterior and anterior language cortices are reciprocally
connected with each other as well. This parallel, modular organization in the
transmission from the posterior to the anterior cortical language area is profitable for
the individual because it saves time and increases the flexibility of responses.
For lexical decision making various lexical options are valid at the same time. In order
to choose the proper lexical item, cortical areas are supported by subcortical structures
within the hypothesized 'cortex-striatum-globus pallidus-ventral lateral/ventral anterior
thalamus-frontal cortex' loop, stated by Brunner et al. (1982). It is supposed that inputs
from limbic (e.g., anterior cingulate cortex subserving motivational factors such as
personal constraints to choose a certain word), reciprocally connected anterior and
posterior regions of the cortex (subserving language) and non-language cortical areas
(e.g., supplementary motor area subserving external factors such as ease of production)
communicate with subcortical areas (including the thalamus and the striatum).
The collocation of information from these structures is assumed to ultimately lead to the
selection of the best lexical alternative, among multiple cortically generated choices.
The best alternative that is aligned with internal motivational factors and external
constraints, is selected. This happens via the striatum, which projects upon the globus
pallidus. The globus pallidus in turn controls and modulates the thalamic nuclei. The
thalamic nuclei transmit information back to the anterior language zone, which is
reciprocally connected with the posterior language zone.
Under normal conditions, thalamic nuclei prevent less optimal lexical alternatives from
getting into the anterior language zones. According to this model, a lesion to the
thalamic portion of the loop results in semantic paraphasias, because inhibition of
inappropriate lexical alternatives is lost and thus also inappropriate lexical alternatives
can get into language output channels. Against that, a lesion of the globus pallidus
would lead to symptoms seen in a transcortical motor aphasia. As the inhibition over
thalamic centers is lost, an overexcitation of the cortex may result, resulting in non-
fluent language output.
Interestingly however, Wallesch and Papagno's model does not stress the effect of
striatal lesions on language outcome at all. But it could be assumed that lesions in the
striatum could have similar effects on language as lesions within the globus pallidus,
resulting in an overexcitation of the cortex, due to a loss of the inhibitory projection to
the thalamus.
2.3.5.2 Crosson's model of subcortical participation in language

Crosson (1985) strengthened the following centers to be implicated in language, based on former studies on thalamic participation in language (as addressed in Appendix II):

1) The anterior language zone (language formulator; not referring to motor planning, includes the concepts of words used for language and the formulation of syntactic structures as well as the decoding of complex syntax)
2) The posterior language zones (language decoder; includes the phonological and semantic encoding of language symbols)
3) The ventral anterior nucleus of the thalamus
4) The ventral lateral nucleus of the thalamus
5) The pulvinar
6) The caudate nucleus
7) The putamen
8) The globus pallidus

65 Because all information, if not other displayed within this chapter, is based on Crosson (1985), no further references are given herein.
Crosson (1985) exemplified possible pathways between these structures, generating a working model of subcortical participation in language. Under Crosson's (1985) assumption, language is formulated by the anterior language zone. Each formulated segment is controlled/monitored for semantic accuracy by the posterior language cortex, which is responsible for language decoding. Language segments are conveyed from the anterior formulation center to the posterior decoding center and back via the pulvinar. If a semantic inaccuracy is found during monitoring, information about the needed correction is conveyed from the posterior language cortex via the pulvinar back to the anterior language cortex. If no semantic inaccuracies are found, language segments are released from the anterior language cortex for programming (articulation).

It can be summarized that no matter which information of the basal ganglia influences the cortex, information must always be mediated by the globus pallidus. From the globus pallidus information is transferred then to the thalamic nuclei. Thus it is the thalamus, under control of the globus pallidus, which influences the cortex.

These cortical and subcortical projections can be summarized as follows (cf. Carpenter & Sutin, 1983: 504f & 514ff):

1) The globus pallidus projects to the ventral lateral and ventral anterior nucleus of the thalamus as well as to the pulvinar.

2) Influences from the basal ganglia upon the anterior cortical language center is mediated through the ventral anterior nucleus, which receives input from the brainstem. Thus, the ventral anterior nucleus may also transmit arousal or activation impulses to the anterior cortical language zones.

3) Influences from the basal ganglia upon the posterior and even upon the anterior cortical language center is mediated through the pulvinar.

4) The ventral lateral thalamus sends fibres to the motor cortex and thus this nucleus is not primarily associated with language; rather with speech.

Based on these considerations as just presented, Crosson (1985) supposed three main functions of the structures within the hypothesized cortico-striato-pallido-thalamo-cortical loop:

1) Cortical functions: Language formulation and language decoding are both functions of the cortex, respectively of the anterior and posterior language zones.

2) Thalamic functions: The cortico-thalamo-cortical pathway provides the mechanism through which the posterior language area is able to check the
encoded language for semantic accuracy. Within the bidirectional loop from the anterior language cortex via the pulvinar to the posterior language cortex, the thalamus fulfills a function of semantic monitoring. This hypothesis could be proved by evidence of semantic paraphasias in thalamic aphasia with more disturbances in language output than in language comprehension (e.g., Alexander & LoVerme, 1980).

3) Basal ganglia functions: The basal ganglia itself have two functions within the cortico-basal ganglia-thalamo-cortical model of language. One function is the regulation of the flow of excitatory impulses from the ventral anterior thalamus, to the anterior cortical language areas. The other one is a motor release mechanism, which allows language segments to be released at the proper time, after semantic monitoring and the transmission of information to the anterior language segments have taken place. Evidence of this hypothesis is given for instance by Van Buren (1963), as he showed that the stimulation of the caudate nucleus in the language dominant hemisphere resulted in the interruption of ongoing language by the flow of irrelevant material into language. Nevertheless, the production of irrelevant language material due to caudate stimulation, is supposed to be mediated by the globus pallidus and the thalamic nuclei, because the basal ganglia itself have no direct output to the cortex.

2.3.5.2.1 Cortico-thalamo-cortical pathway

The cortico-thalamo-cortical pathway provides the mechanisms through which the posterior language zone checks the decoded language for semantic accuracy. In particular, there is a transmission of semantic information from the anterior language zone to the ventral anterior thalamus, from the ventral anterior thalamus information is then transmitted to the pulvinar and from there to the posterior language zone in the cortex. Since there, a bidirectional pathway exists between the anterior and the posterior language areas via the pulvinar. The language segment is then transmitted back from the pulvinar to the ventral anterior thalamus, which gets excitatory afferents from the reticular formulation. Excitatory impulses are then conveyed from the ventral anterior nucleus to the anterior language zone, which provides the proper level of arousal for the execution of the language formulator.
Transmissions of linguistic information from the anterior language zone to the posterior language zone and back are given in Figure 5 and 6 (adopted from Crosson, 1985), below.

Figure 5 shows the transmission of semantic information from the frontal to the posterior language zone.
2.3.5.2.2 Cortico-basal ganglia-thalamo-cortical pathway

The putamen and the caudate nucleus (= striatum) receive input (among others) from the two main language areas in the cortex. The output from the striatum is mediated through the globus pallidus. The globus pallidus maintains an inhibitory influence over the ventral anterior thalamus. As the ventral anterior thalamus gets excitatory afferents from the reticular formulation, as shown in Figure 6, the globus pallidus (being inhibitory by virtue) regulates the amount of excitation of the ventral anterior thalamus upon the language formulator. Lesions of the inhibitory globus pallidus would thus lead to a disinhibition of the ventral anterior thalamus, to an overactivation of the anterior cortical language zones, resulting into paraphasic language. Against that, too much stimulation of the globus pallidus would excite inhibitory mechanisms and thus result into the inhibition of the ventral anterior nucleus and the interruption of ongoing language.

The second function of this pathway is a motor release mechanism, allowing language segments to be released at the proper time. After semantic monitoring, the posterior language zone projects to the striatum. The striatum exerts an inhibitory influence upon the inhibitory globus pallidus and thus pallidal inhibitory mechanisms onto the thalamus are inhibited, resulting into an overactivation of the ventral anterior thalamic nucleus, which is excitatory by virtue. This in turn increases the excitation of anterior cortical mechanisms, allowing the release of semantically verified language for motor planning\textsuperscript{66}. Once motor planning has been completed, anterior cortical zones project to the striatum, restoring again the inhibitory mechanism of the caudate nucleus. This hypothesized mechanism for the release of formulated language is shown in Figure 7 below.

\textsuperscript{66} Normally, when a person is not speaking, the pallidal inhibitory mechanisms are not inhibited, and thus there is no increase in excitation of the anterior language mechanisms.
2.4 Summary and perspectives

The role of subcortical structures, including the basal ganglia, in language has now been debated for more than one century (Marie, 1906). However, even after such a long period of time, little consensus exists, regarding whether or how these structures contribute to language.

It is evident, that a total understanding of the neurophysiology of the basal ganglia is needed first – which has not been reached yet – in order to come up with a working theory of basal ganglia participation in language and the explanation of language disturbances in subcortical diseases (cf. Wallesch, 1997: 273). Nevertheless, in bits and pieces the core mechanisms supposedly leading to language changes within subcortical pathology, are studied. Thus, we know from the literature, as just presented, that there are mainly three mechanisms responsible for contributing to language changes in subcortical patients. Previously it has been suggested that subcortical structures themselves have a direct role in language functions. More recently, it has been stated that not lesions in subcortical structures, rather their disconnection from cortical areas and resulting metabolic changes, result in language disturbances.
Additionally, evidence from neurodegenerative diseases such as PD and HD prove the debated role of the basal ganglia in the development of linguistic disturbances. Moreover, models have been developed, explaining cortico-basal ganglia-thalamo-cortical circuits in language functioning. But unfortunately until now, no clear picture has emerged regarding language changes in subcortical patients. From the existing literature it is evident that many degrees of severity, as well as many different combinations of linguistic symptoms result from different lesions in the connection of the basal ganglia from the cortex. Thus, a distinct classification of aphasia, following subcortical disturbances is not possible at this time and thus also the concept of subcortical aphasia might not always be useful (cf. Robin & Schienberg, 1990: 97).

Tying in here, Wallesch and Papagno (1988) summarised that mainly these language tasks which involve the highest number of degrees of freedom, namely spontaneous utterances and free choice in the lexicon, seem to be most markedly affected in patients suffering from lesions deep in the subcortical nuclei (cf. Wallesch & Papagno, 1988: 267). Interestingly however, subcortical patients often do not show any deficits on classical aphasia batteries (cf. D'Esposito & Alexander, 1995: 40). This lack of evidence for linguistic deficits in subcortical patients, may be due to the use of inappropriate language material (classical aphasia batteries, designed for cortically injured patients). These tests seem not to be sensitive enough to reveal and classify language problems in subcortical disorders and are thus inappropriate for subcortical patients. This assumption could for instance be proved by Podoll et al. (1988), Wallesch and Fehrenbach (1988) as well as by Chenery, Copland and Murdoch (2002), as will be given in chapter 3.

A further deficit regarding the study of linguistic changes in subcortical patients is the mainly non-linguistic analysis and interpretation of data. Unfortunately almost all papers published in the past, addressing language changes in subcortical patients have been written by neurologists or neuropsychologists, testing language with classical aphasia batteries. Analysis of errors has been conducted on the basis of quantitative statistical measures and in regards to a classification into traditional aphasic syndromes. As could be shown within this chapter, this didn't work.
The quotation of the linguist, Roman Jakobson (1956) „If aphasia is a language disturbance, as the term itself suggests, then any description and classification of aphasic syndromes must begin with the question of what aspects of language are impaired in the various species of such a disorder“ (Jakobson, 1956: 69), has been ignored many years in the field of neurolinguistics. Nevertheless, this assumption may be even more 'en vogue' now, than any time before. Even though various neuroimaging techniques are available today for studying brain functions, a detailed description of how language changes, as a result of, for instance subcortical lesions, cannot be answered with these methodologies either. Against that, linguistics could contribute positively to the understanding of linguistic deficits in subcortical patients by analyzing the language output on a linguistic basis, implementing linguistic disturbances found into a well-grounded theory. This is what the present PhD thesis is meant to achieve. Before heading to the methodology and result section of this study, a critical discussion of 'language functions's in Huntington's disease' will be given in the next chapter.

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67 For further information on Jakobson's attempt towards a linguistic typology of aphasic impairments see for instance Jakobson (1964).
3 Language functions in HD

As described in chapter 1.5.2.1, a striking feature of the subcortical dementia associated with HD is the absence of aphasia (see for instance Folstein, Folstein & McHugh, 1975; Butters et al. 1978; Cummings & Benson, 1984; Brandt & Butters, 1986; Brandt, Folstein & Folstein, 1988).

However, there is still an ongoing debate as to whether language functioning is affected in HD as a subcortical disorder. Indeed, (Brandt & Butters, 1986) state that “Although HD patients do not typically have classical aphasic syndromes, they do develop specific abnormalities in the use of language” (Brandt & Butters, 1986: 316).

Several mechanisms, as just described in chapter 2, could explain how lesions in the basal ganglia can lead to language changes. It is possibly through a disruption in the connection of the basal ganglia to the cortex and resulting metabolic disturbances, that linguistic dysfunctions occur. Further evidence for a specific function of subcortical structures, such as the basal ganglia could be given by the example of PD and HD.

The present PhD thesis aims to contribute to this ongoing discussion, by examining if linguistic changes do occur during the course of HD, and if so, if they are part of the subcortical dementia seen in HD. Thus, a comprehensive review of the literature associated with 'language changes in HD' will be given. The literature search was performed using electronic databases, including Web of Science, Web of Knowledge, and Science Direct (1980-2009), as well as a systematic checking of reference lists published in review articles and other reports, up to now. Results of the literature research indicate that only a small number of papers have been published in the area of language changes in HD.

However, in the following section, this literature will be analyzed in regards to specific language changes during the course of HD. This analysis will take into consideration the patient's stage of HD, genetic variables such as CAG repeats, 'days since disease onset', and the three core symptoms associated with the disorder.
3.1 HD patient's performance in standardized language batteries

Kennedy et al. (1981) were among the first to research language impairments associated with HD. Using a comprehensive language battery including the Boston Naming Test (BNT)\(^{68}\), the Revised Token Test (RTT)\(^{69}\), and the Neurosensory Center Comprehensive Examination for Aphasia (NCCEA)\(^{70}\), Kennedy et al. (1981) investigated language function in 30 HD patients. The results of this study indicated that the HD patients were significantly impaired on the BNT, RTT, and on the word fluency subtest of the NCCEA. Patients in the early stages of HD also demonstrated impaired language in the areas of organization, sequencing, and elaboration. Interestingly however, the pattern of language impairment, did not resemble the aphasic symptoms seen in patients with cortical damage. Therefore, contrary to common belief, Kennedy et al. (cf. 1981: 81f) demonstrated that specific aspects of language function (e.g., picture naming, word fluency and language comprehension) can be disrupted in HD. Contrastingly, using the Aachener Aphasia Test, Caspary et al.'s (1990) and Podoll et al.'s (1988) research demonstrated that patients with HD (45) did not present any primary language changes. However, patients in the middle and late stages of HD produced more errors, compared to patients in the early stages of HD, specifically in the Token Test, repetition, writing, naming, and picture matching. Caspary et al. (1990) as well as Podoll et al. (1988) hypothesized that these language changes developed secondary to other neurological and neuropsychological changes, such as dementia (cf. Podoll et al., 1988: 1475 ff).

Additionally, Chenery, Copland and Murdoch (2002) investigated 'primary' and 'complex language functions' in 13 HD patients. Results showed that 'primary linguistic ability', as assessed by the Western Aphasia Battery (WAB) and the Boston Naming Test (BNT), was preserved. This finding was supported by Podoll et al. (1988) and Wallesch and Fehrenbach (1988). However, extended testing using tests assessing 'complex

\(^{68}\) The BNT developed by Kaplan, Goodglass and Weintraub (1983) represents an objective measure for the ability to name line drawings.

\(^{69}\) The Token Test has first been developed by DeRenzi and Vignolo (1962) and revised for German by Orgass in 1976, as well as for English in 1978 by McNeil and Prescott (Revised Token Test = RTT). The Token Test is a sensitive test to assess language comprehension, attention and general cognitive functioning.

\(^{70}\) The NCCEA first designed by Spreen and Benton in 1969, is a aphasia test battery consisting of 24 short subtests, involving different aspects of language, such as recognition of sounds, tactile recognition, forming letters into words etc.
language functions', revealed difficulties in various areas. Impairments in complex language tasks in HD patients, have further been supported more recently by Hartelius et al. (2008).

Nevertheless, in more detail, in the study by Chenery, Copland and Murdoch (2002), the Test of Language Competence-Expanded Edition (TLC-E), the Test of Word Knowledge (TOWK) and the Word Test-Revised (TWT-R) revealed deficits in complex language tasks involving lexico-semantic operations, the interpretation of ambiguous, figurative and inferential language as well as problems in set-shifting. Specifically, on the Oral Expression subtest of the TLC-E; the HD patients often generated sentences that contained only one of three given words (e.g., Providing the three target words 'without, difficult, wrong' resulted in sentence productions like: 'The race was too difficult') (cf. Chenery, Copland & Murdoch, 2002: 470). When the patients attempted to construct a sentence using more of the target words, the sentence frequently became ungrammatical (e.g., Using 'before, rather, after' in a sentence relating to a movie theatre resulted in a production like 'Before, which would you rather? After too.') (cf. Chenery, Copland & Murdoch, 2002: 470). On the Ambiguous Sentence Task of the TLC-E, the HD patients were often only able to provide one definition, not two. On some occasions, the patients gave two similar meanings or paraphrased the sentence without giving a precise interpretation (e.g., For the sentence 'Can you believe Mary wanted to run as well as me?', HD patients responded 'that's competition') (cf. Chenery, Copland & Murdoch, 2002: 470). On the Figurative Language Test of the TLC-E, the HD patients were unable to adequately explain idiomatic expressions; they often responded with an inadequate literal interpretation (e.g., When one HD patient was asked to provide an explanation for the metaphor 'It's hard to zero in on his ideas' in the context of two students talking about a teacher, the HD patient responded 'They don't like the teacher'.) (cf. Chenery, Copland & Murdoch, 2002: 471). On the Making Interference Subtest of the TLC-E, the HD patients experienced furthermore difficulty processing detailed or implied information (difficulty in integrating story information with contextual or prior knowledge).

Similarly, on the TOWK (Multiple Contexts Task), the HD patients found it difficult to provide two definitions of homophonic words. Even though the HD patients were often able to provide one definition, they were unable to shift attention and provide an alternative meaning (e.g., The meaning of 'mole' as a small animal could be given, but it couldn't be thought of a second meaning. It happened also that the same meaning was
given in two different contexts; e.g., For 'manual' was given 'manual car' and 'drive manually') (cf. Chenery, Copland & Murdoch, 2002: 469). This specific type of response could be interpreted as cognitive inflexibility, a common symptom noted in patients with HD (Hanes, Andrewes & Pantelis, 1995), due to their dysexecutive syndrome (Brandt, Folstein & Folstein, 1988; Rosenberg, Soerensen & Christensen, 1995; Lawrence et al., 1996; Lemiere et al., 2004; Peinemann et al., 2005). Thus, it can be concluded “that a specific language profile exists in HD” (Chenery, Copland & Murdoch, 2002: 471).

3.2 HD patient's performance in narrative and descriptive language tasks

In 1987, Gordon and Illes examined spontaneous speech (based upon an account of travel) and descriptive language (based upon a description of the Cookie Theft Picture71) in 12 patients with HD. The results of this study indicated a disturbance in language production in HD, similar to aphasic syndromes. The HD patients evidenced a significant reduction in the number of words produced, a diminished level of syntactic complexity, reductions of melodic line (prosody), in phrase length, articulatory agility and grammatical form, an increase in paraphasic errors and wordfinding difficulties, frequent silent intervals and a loss of conversational initiative (cf. Gordon & Illes, 1987: 7). In a follow up study involving 10 HD patients, Illes (cf. 1989: 629ff) demonstrated that HD patients require more prompts and questions to elicit an equivalent amount of speech, compared to healthy controls. In planning sections temporal interruptions, and word repetitions were frequent. The HD patients also evidenced a reduction in the syntactic complexity as well as a predominance in closed class phrases in contrast to Alzheimer's disease patients.

Productive syntactic abilities have also been examined by Murray and Lenz (2001) in nine patients with HD. The study was designed to explore possible relationships

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71 The Cookie Theft Picture as a standardized sample for eliciting descriptive speech is part of the Boston Diagnostic Aphasia Examination (BDAE) by Goodglass and Kaplan (1983). The BDAE is a comprehensive language test battery designed to evaluate a broad range of language impairments that often arise as a consequence of brain injury. This examination is designed to describe symptoms of language dysfunction. Thus it evaluates perceptual modalities (auditory, visual and gestural), processing functions (comprehension, analysis and problem solving) and verbal responses (writing, articulation and manipulation).
between syntactic changes and cognitive or motor symptoms associated with HD. Within a semi-standardised 5 to 10 minute conversational discourse, patients were asked to talk about themselves, their life, hobbies and profession. HD patients displayed pure linguistic changes in productive syntax. Specifically, they produced shorter utterances and a smaller proportion of grammatical utterances, compared to their healthy peers. Interestingly, patients with greater cognitive deficits and more severe dysarthria performed more poorly on syntactic production. Thus, these results support the hypothesis that changes in language production in HD, at least to some degree, are due to cognitive decline (Podoll et al., 1988; Wallesch & Fehrenbach, 1988) and motor speech disturbances, such as dysarthria (e.g., Yorkston et al., 1999).

Murray (2000) examined the spoken language abilities in 10 HD patients. Spoken language samples were elicited from the 'Cookie Theft Picture' (Goodglass & Kaplan, 1983) and the 'Grocery Store Scene' (Helm-Estabrooks, 1992). Results indicated that the HD group produced shorter utterances, a smaller proportion of grammatical utterances, a larger proportion of simple sentences, and fewer embeddings per utterance, compared to the control group. Moreover, a smaller proportion of correct information units and informative utterances were produced. No morphological errors were observed in the verbal output of any HD participant in the study. In sum, the HD patients appeared to produce as much as their controls, but less of what they said was judged to be informative. The linguistic deficits in HD were identified in the absence of dementia and dysarthria. To conclude, for patients with HD, impairments in quantitative, syntactic and informative aspects of spoken language ability could be observed. Wallesch and Fehrenbach (1988) examined spontaneous language production, using the AAT, in 18 patients with HD. No semantic or phonemic paraphasias were revealed and the phonological aberrations that occurred were due to dysarthria. The information density as measured by words per unit also remained unaffected. But the number of subordinate clauses (e.g., within relative sentences) was found to be markedly lower in advanced HD patients. A significant decrease in syntactical complexity (less frequent use of constructions using subordinate clauses) was also observed as the disease progressed. These changes were not described as being linked to aphasia, but rather to a pragmatic deficit resulting from dementia.

72 The Grocery Store Scene is a subtest of the Aphasia Diagnostic Profiles by Helm and Estabrooks (1992) and evokes picture description in patients.
Thus Wallesch and Fehrenbach stated that “Huntington's chorea undisputedly leads to dementia, which may cause language abnormalities” (Wallesch & Fehrenbach, 1988: 371).

Jensen, Chenery and Copland (2006) assessed oral descriptions in terms of lexical-semantic and syntactic abilities in six individuals with HD, using the 'Cookie Theft Picture' (Goodglass & Kaplan, 1983). In regards to syntax, Jensen, Chenery and Copland (2006) identified disturbed sentence formulation abilities in HD patients. In particular, significantly more grammatical errors (as the most common grammatical error made by HD subjects, omissions of 'a' or 'the' at the beginning of a sentence were found) were recorded in HD patients, compared to controls. In terms of information content, HD patients produced significantly fewer action information units than controls. For example, the proposition 'there is a boy with a kite' received credit for including subject and object information but was missing action information units, such as 'boy is flying a kite' (cf. Jensen, Chenery & Copland, 2006: 74). Jensen, Chenery and Copland (2006) inferred that the HD subjects' semantic memory was intact, despite subcortical damage, as semantic and phonemic paraphasias were absent, and the HD patients did not display significantly more word finding difficulties, compared to the controls.

3.3 Naming theories try to explain naming difficulties in HD

Deficits in naming could also be found in the language symptomatology of HD patients. Until now the source of naming deficits associated with dementias such as HD has not been established.

One theory exists (Coslett and Saffran, 1989) that names four different stages involved in naming. In a first stage the analysis of the visual features of a given stimulus is addressed. In a second, semantic stage, visual features are pooled with semantic knowledge. In a third lexical stage the phonological representation is retrieved and pooled with the semantic knowledge. In a production stage, the phonetic representation is given. Deficits in those stages are thought to lead to naming disturbances. Two other theories, addressing the naming difficulty seen in HD, have been developed, based on this theory.
A perception theory by Rochford (1971) exists as well as a semantic feature theory by Schwartz, Marin and Saffran (1979). According to a so called perception theory demented patients exhibit misrecognitions, in terms of giving the name of an object similar in appearance to the stimulus item (cf. Rochford, 1971: 439). Thus naming errors are perceptually based according to this theory.

On the other hand a semantic feature theory is proposed to give an alternative explanation for the naming deficits in HD. Based on this hypothesis naming deficits are due to deficits in the access to the semantic lexicon (linguistically based theory). Schwartz, Marin and Saffran (1979) tried to prove this theory in a patient with a progressive dementing disease of unknown origin. In naming a total number of 140 common household objects the patient made a total of 51 errors. Forty six out of 51 errors were selections of the semantic distractor (e.g., shown a fork and chosen a spoon). Furthermore the patient consistently overextended verbal labels (e.g., the semantic category dogs) to closely associated distractors (e.g., cats). Thus Schwartz and colleagues (1979) concluded that these naming errors are due to a breakdown in the access to the semantic store.

3.3.1 Naming performance in HD

Hodges, Salmon and Butters (1991) studied the nature of naming deficits seen in HD, by administering the Boston Naming Test (BNT) (Kaplan, Goodglass & Weintraub, 1983) to 16 patients suffering from HD. The authors revealed that the deficit in confrontation naming is primarily perceptual. The great proportion of visually based errors implies the defect at the first stage of the naming process in the content of the naming theory by Coslett and Saffran (1989) and thus also supports the perception theory by Rochford (1971).

In contrast to this finding, Frank, McDade and Scott (1996) administered three standardized naming tests to six HD patients with mild dementia, and six HD patients with moderate dementia. In detail, the Peabody Picture Vocabulary Test73 (Dunn, 1981),

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73 Actually used as individual intelligence test, it also assesses the vocabulary. Four numbered pictures are shown to the patient. The examiner is describing one of the pictures and asks the subject to point to or to say the number of the picture which fits the description.
the Visual Recognition Test as a subtest of the Illinois Test of Psycholinguistic Abilities (Kirk, McCarthy & Kirk, 1968) and the Confrontation Naming Test as part of the Boston Naming Test (BNT) (Kaplan, Goodglass & Weintraub, 1983) were used to study naming ability. In contrast to the study by Hodges, Salmon and Butters (1991), Frank, McDade and Scott (1996) did not report any visual misperceptions in HD patients. However, a restricted access to semantic features for concept identification was found in HD, instead. In more detail, the matching of the perception with semantic knowledge was problematic in HD patients. Thus the semantic feature theory by Schwartz, Marin and Saffran (1979) could be supported, evidencing the HD patients experience significantly deficient lexical access.

Within the study by Chenery, Copland and Murdoch (2002) a detailed analysis of the Boston Naming Test (BNT) (Kaplan, Goodglass & Weintraub, 1983) as well as of the Word Definitions Subtest of the Test of Word Knowledge (TOWK) (Wiig and Secord, 1992) showed a higher proportion of semantically related errors (swordfish for seahorse) and to a lesser extent visually related errors (didgeridoo for asparagus). These results provide further support for the semantic feature theory by Schwartz, Marin and Saffran (1979).

### 3.4 HD patient's performance in discourse comprehension and comprehension of prosody

Murray and Stout (1999) noted that patients with HD, even those with mild or no dementia, are at risk of discourse comprehension difficulties. By administering the Discourse Comprehension Test (DCT) to nine patients (before dementia diagnosis) with HD, it could be shown that questions pertaining to details and implied information lead to difficulties in comprehension. This is due to the fact, that for questions relying on implied information, heuristic strategies such as world knowledge, are ineffective.

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74 The Illinois Test of Psycholinguistic Abilities has been designed to measure acquisition and use of language in 4 to 8 year olds. It is used in assessing children's language in educational, research and clinical settings.

75 The DCT, designed by Brookshire and Nicholas (1993), is a standardized test battery to assess comprehension ability in brain damaged adults. This test goes beyond the single word and sentence stage and tries to test comprehension in discourse situation (reading comprehension and listening comprehension). Patients are asked to listen to/read the discourses and answer yes/no questions. The comprehension and retention of stated and implied main ideas and details concerning the stories, is addressed by this test.
Contrastingly, the HD patients performed better on questions that focused on main ideas and stated information. In sum, it could be noted that there is a strong relationship between general cognitive decline and discourse comprehension difficulties in HD. Not simply discourse comprehension but also comprehension of prosody has been proven to be affected in HD patients, as examined by Speedie et al. (1990). The study, split up in two parts, involved a total of 12 non-demented HD patients (6 in study one and 6 in study two) in early stages of their disease. In the first part of the study affective and propositional prosody were evaluated. Subjects were presented tape-recorded sentences (30 sentences in 3-6 seconds in duration) in random order and were asked to denote the prosody of the 'sentence-speaking-voice' as happy, sad or angry (affective prosody) to denote it as a statement, question or command (propositional prosody). The study revealed that HD patients were impaired in the comprehension of both types of prosody. In the second part of the study prosodic discrimination (affective versus propositional), rhythm discrimination (subjects are asked to listen to 5, 6 or 7 beat rhythmic patterns and were to indicate if the rhythms are the same or different) and tonal memory (it requires the subject to indicate which of of a pair of 4, 5 or 6 tones is different) were studied. Patients were able to discriminate rhythmic patterns quite well, however more difficulties were found on tonal aspects and prosodic discrimination. As tonal and prosodic parts of language are important for the understanding of subtle aspects of communication, those impairments are thought to contribute negatively to social interaction even early in the course of the disease.

### 3.5 Fluency performance

Fluency performance has further been proven to become affected in the course of HD. Fluency performance indicates cognitive flexibility to name as many exemplars of a given category, usually within one minute in so called letter or category fluency tasks. In order to understand subsequent descriptions of impairments in HD patients's fluency performance, a brief introduction will be given into verbal fluency itself, as well as into the two main domains verbal fluency can be tested in, such as clustering and switching.
3.5.1 Verbal fluency

Based on the notation that language disorders associated with HD, mainly concern frontal aspects of language such as e.g., verbal fluency (cf. Péran et al. 2003a: 565) those aspects have also been studied in detail in HD patients. In 1938 Thurstone proposed, that intelligence is made up of a small set of independent factors, referred to as primary mental abilities, and suggested that verbal fluency was one of these (cf. Thurstone, 1938: 29). Thurstone (1938) developed subsequently a written pre-version of the now known Oral Letter Fluency Association Test (mostly tested with the letters A, F and S) designed by Borkowski, Benton and Spreen (1967).

Verbal fluency is typically tested in letter and category fluency domains. Within a letter fluency task words must be produced according to phonemic constraints (e.g., produce words beginning with the letters A, F, and S (Borkowski, Benton & Spreen, 1967) within a 60 seconds time frame. This test, referred to as the AFS test, is still used in neuropsychology (e.g., within the cognitive subtest of the UHDRS). More recently, semantically based verbal fluency tasks (category fluency tasks) have been used in cognitive-linguistic assessments to study brain damaged patients (e.g., Goodglass & Kaplan, 1972). In contrast to letter fluency tasks, category fluency tasks require subjects to produce as many exemplars of a given semantic category (e.g., animals) as they can within 60 seconds. Research has shown that letter and category fluency are mediated by distinct neurological structures; where letter fluency appears to be sensitive to frontostriatal pathology (e.g., Rosser & Hodges, 1994), and category fluency to temporal lobe deficits (e.g., Hodges et al., 1992). Unfortunately it is not that easy. More recent research found out that prefrontal areas subserve semantic fluency as well (Frith et al., 1991; Gurd et al., 2002). In detail, the bilateral cingulate cortex, the bilateral left inferior frontal gyrus, the bilateral frontal operculum as well as the cerebellar vermis, could be proven to be significantly activated in normal volunteers while switching between semantic subcategories within a semantic fluency task (Gurd et al., 2002). Left dorsolateral cortical activation (e.g., in BA 46) could moreover be found in a PET study, researching normal volunteers performing a semantic fluency task (Frith et al., 1991).
3.5.2 Clustering and switching

Two underlying, distinct cognitive strategies have been found to be associated with fluency tasks: clustering and switching (cf. Troyer, Moscovitch & Winocur, 1997: 139). Clustering is defined as the successive access of a sequence of words (a minimum of two words is considered to be a cluster) within the same phonemic or semantic subcategories (e.g., fox, fire, flu, fine = letter fluency; dog and cat belong to the same semantic superset 'domestic animals' = category fluency) (cf. Troyer, Moscovitch & Winocur, 1997: 140). Clustering can also include items that are regarded to be errors such as repetitions, supercategory-items, neologism errors, non-category items and synonymic items (cf. Troyer, Moscovitch & Winocur, 1997: 140), as these productions can provide additional interesting information about underlying cognitive processes. Interestingly research has found that the production of words within clusters tends to be produced in temporal clusters (Gruenewald & Lockhead, 1980), with a short time interval between words produced within a cluster and longer pauses between clusters (cf. Bousfield & Sedgewick, 1944: 153). The fact that semantically related items are recalled together in time, has further been supported by Wixted and Rohrer (1994). When pauses between clusters are prolonged switchings to another category occur. Thus semantic switching can be referred to as the total number of single word transitions between clusters (cf. Troyer & Moscovitch, & Wincour, 1997: 145).

Using these criteria as just presented, the total number of clusterings per supercategory, as well as the number of items a cluster is made of, has been computed within the category fluency task of this study, as shown in chapter 4.4.1 as well as within chapter 6.1. Thus optimal switching might suggest the production of clusters of semantically related words and once a subcategory is exhausted, switching to another category would be suspected to occur. After retrieval of typical and familiar items of a subcategory, switching would abandon that 'old' category and the movement to a 'new' category is supposed to be most effective within verbal fluency (cf. Troyer, Moscovitch & Winocur, 1997: 139; Ho et al., 2002: 1277; Reverberi, Laiacona & Capitani, 2006: 469).
3.5.3 Fluency performance in HD

As just described, clustering accesses semantic memory, switching on the other hand is supposed to involve working search processes within these stores (cf. Troyer, Moscovitch & Wincour, 1997: 139).

Nevertheless, until now it is not clear where fluency deficits in HD patients arise from. There is an ongoing debate as to whether reductions in verbal fluency are due to linguistic (in detail semantic) storage deficits, or if they are the result of ineffective retrieval mechanisms from these stores. More evidence could be found that the later assumption is true. For instance, a specific lack in recalling verbal information within a fluency task could be confirmed in various studies by Butters et al. (1986; 1987 and 1988). Accordingly, Butters (1984: 19) stated that "retrieval deficits are perhaps the most important factors in HD patients's memory deficits". Similarly, category fluency tasks in an uncued as well as in a cued\(^\text{76}\) version were administered by Randolph et al. (1993), to 49 subjects suffering from degenerative brain diseases. It could be shown that HD patients improved significantly within cued tasks. On the basis of this evidence it could be inferred that this specific fluency problem in HD patients, might be due to initiation and retrieval problems, rather than the result of a general semantic breakdown (cf. Randolph et al., 1993: 86; Rosser & Hodges, 1994: 1393).

Further support for the theory that HD patient's fluency deficits are due to retrieval problems, arise from study outcomes by Tröster et al. (1989) as well as by Monsch et al. (1994). Monsch et al. (1994) indicated, that HD patients' performance on category and letter fluency tasks is significantly impaired, compared to controls. Similarly, reduced category fluency of HD patients, compared to patients suffering from Alzheimer's disease, has been stated by Tröster et al. (1989). Because it is well known that HD patients have a severe bradyphrenia\(^\text{77}\), this deficiency may result in difficulties retrieving information on episodic and semantic memory tasks (Butters, 1984).

Thus, Monsch et al. (cf. 1994: 28) concluded that HD patients do not suffer from a loss in semantic structure, rather from a general impairment in retrieval mechanisms. This was also shown by Tröster et al. (1998), who noted reduced average cluster sizes on letter (AFS) as well as on semantic fluency tasks, within HD patients. As the average

\(^{76}\) The cued version of the category fluency task uses 'within category cues' as a retrieval aid (e.g., animals that people keep in their home as pets; animals that are found on a farm) (cf. Randolph et al., 1993: 84).

\(^{77}\) Is referred to as a deceleration of cognitive functioning due to cognitive decline and dementia.
Cluster size can be regarded as an index of the ease of access to the semantic memory (cf. Troyer, Moscovitch & Winocur, 1997: 139), reduced cluster sizes on fluency tests can be best interpreted as changes in the efficiency of access to semantic memory stores (cf. Tröster et al., 1998: 302).

Thus, it can be inferred from the study results as just given, in order to perform effectively within this task, a founded strategy, such as organised search processes through the semantic memory are needed (cf. Reverberi, Laiacna & Capitani, 2006: 470). But, as already mentioned in chapter 1.5.1 and chapter 1.5.2, frontal patients and also patients suffering from HD, with interruptions within the cortico (fronto)-basal ganglia-thalamo-cortical (frontal) circuits, suffer from a dysexecutive syndrome (cf. Caine et al., 1978: 379; Lawrence, Sahakian & Robbins, 1998: 380) and thus fail to initiate problem solving strategies and further have particular difficulty with cognitive tasks that require executive functions such as organization, planning and sequencing of events (Brandt & Butters, 1986; Brandt, Folstein & Folstein 1988; Brandt, 1991; Burgess & Shallice, 1996b; Craufurd & Snowden, 2002). Because frontal patients also suffer from response initiation deficits (cf. Burgess & Shallice, 1996b: 270), they are in general simply slower while retrieving items. In sum all these factors could lead to reductions of the total number of words produced within fluency tasks (cf. Reverberi, Laiacna & Capitani, 2006: 470).

In addition to problems understanding the origin of fluency deficits, more and more criticism is arising, concerning the methodology of fluency tasks, respectively the evaluation of clusterings and switchings. In detail, there is no standardized evaluation criterion regarding the evaluation of clusterings. Further, there is strong doubt on the methodology used by Troyer, Moscovitch and Winocur (1997), concerning the evaluation of switchings. Following the switching methodology by Troyer, Moscovitch and Winocur (1997) as given in chapter 4.4.1 as well as in chapter 6.1, study results show that frontal as well as HD patients, produce a lower number of switchings between subcategories in contrast to controls (Tröster et al., 1998; Troyer et al., 1998; Rich et al., 1999; Ho et al., 2002). This report of a reduced number of switchings is in contrast to more recent research, showing the opposite. For instance, Reverberi, Laiacna and Capitani (cf. 2006: 477) pointed out that there is an increase in the number of relative switchings78 for left frontal patients. In detail, the disorganised search through the

78 The relative number of switchings is defined as the ratio of the raw number of observed switches divided by the total number of words generated minus 1, including repetitions (cf. Reverberi, Laiacna & Capitani, 2006: 472f).
semantic memory in frontal lobe patients, is supposed to lead to the production of a disorganised sequence of words, such as 'apple – orange – cherry – blackberry – pear – tangerine – banana etc.', due to an increase in switchings between clusters (cf. Reverberi, Laiacona & Capitani, 2006: 470). These results can possibly also be adopted into the field of HD, as patients suffering from HD have been reported to suffer from a dysexecutive syndrome, similar to frontal lobe patients.

Criticisms regarding the methodology used by Troyer, Moscovitch and Winocur (1997) is further expressed by Reverberi, Capitani and Laiacona (2004) as well as by Mayr (2002). Whereas Mayr (2002) criticised the numerical implications of the proposal by Troyer, Moscovitch and Winocur (cf. 1997: 140), referring to the absolute number of transitions between clusters, Reverberi, Capitani and Laiacona (2004) stated a wrong or too loose definition of subcategories and consequently also that of switchings. Because of a lack of consideration of these factors in the study by Troyer, Moscovitch and Winocur (1997) and the use of this study as reference material for further research on fluency by Tröster et al. (1998), Troyer et al. (1998), Rich et al. (1999) as well as by Ho et al. (2002), caution is warranted in the interpretation of study results addressing fluency performance.

On the basis of classical fluency tasks (letter versus category), a verb and noun generation task has been assessed to 26 HD patients in the study by Péran et al. (2003a). The word generation task was based on 40 concrete nouns and 40 action verbs. Subjects were instructed to produce a semantically related noun of a given noun (e.g., 'If I tell you fork, you may answer spoon'), a semantically related verb of a given verb (e.g., 'If I tell you drink, you may answer eat'), a semantically related noun of a given verb (e.g., 'If I tell you wine, you may answer drink') and a semantically related verb of a given noun (e.g., 'If I tell you eat, you may answer meat'). The number of correct responses was calculated, as well as the errors produced, classified. No difference was noted between patients and controls in noun production tasks. But more difficulties could be revealed in tasks implying switching between categories (VN and NV).

Deficits in the switching between semantic categories might reflect a form of cognitive inflexibility, a common symptom noted in patients with HD (Hanes, Andrewes & Pantelis, 1995), due to their dysexecutive syndrome (Brandt, Folstein & Folstein, 1988; Rosenberg, Soerensen & Christensen, 1995; Lawrence et al., 1996; Lemiere et al., 2004; Peinemann et al., 2005)
3.6 Morphological performance in HD

In HD, changes in the morphology could also be observed. In detail, inflectional morphology\textsuperscript{79} has been noted to be impaired. Ullman et al. (1997a) reported HD patients to be more impaired in the computation of regular than in irregular past tense forms of English verbs. Ullman, Hickok and Pinker (1995) demonstrated the same pattern in an agrammatic aphasic patient with stroke induced damage to Broca's area. In order to account for the features found, a mental model of morphology has been stated by Ullman et al. (1997a). This model will be presented first, in order to follow subsequent descriptions about morphological changes in HD patients.

3.6.1 Ullman's model of lexicon and grammar

Ullman's (1993) PhD thesis, concerning 'The computation of inflectional morphology' continued the ongoing debate as to whether linguistic forms are stored and retrieved as a whole (full listing hypothesis) (Manelis and Tharp, 1977 as well as Butterworth, 1983), or if they are decomposed in their minimal entities (decomposition hypothesis) (Taft & Forster, 1975). This lexicon/rule controversy is based on models, stating that the human 'language faculty' is organized along a dual processing architecture, comprising both, a mental lexicon and a computational grammar (Chomsky, 1965; Pinker, 1999 cited from Pinker, 2000).

Two capacities of long term memory have been stated, the declarative, explicit memory in contrast to the procedural, implicit memory (Tulving, 1972; Cohen & Squire, 1980) (for further details about the procedural/declarative system see Appendix IV).

According to Ullman, this hypothesized division of the brain into two major kinds of memory systems (procedural vs. declarative memory)\textsuperscript{80} also contributes to language.

\textsuperscript{79} Changes in inflectional morphology, in the course of HD have been shown in English and French verbs. For these studies the old dichotomy in regards to past tense formations between regular and irregular English verb forms has been used. Regular forms are transferred into past tense by the application of the productive ed-suffixation to the stem of the verb (walk-walk-ed). The past tense forms of irregular verbs on the other hand, are supposed to be memorized (dig-dug). For French, on the other hand, a distinction between even three conjugation groups has been used instead. Conjugation group 1 refers to regular verbs with infinitives ending in -er. This is the most productive class. There are also subregular verbs ending in -ir which refer to conjugation type 2; those follow the same suffixation pattern as regular verbs. Together with verbs ending in -oir, which are highly unpredictable in their suffixation patterns, irregular verbs, belong to conjugation group 3.

\textsuperscript{80} A brief introduction into the two main capacities of long term memory: the declarative and the
More specifically, our use of language depends on two capacities: a mental lexicon of memorized words (declarative memory) and a mental grammar (procedural memory) of grammatical rules (e.g., Ullman et al., 1997a; Ullman, 2001a; 2001b; 2001c; 2004). It has been hypothesized that the mental lexicon also includes idiosyncratic information, like how many arguments a word can take, the irregular past tense forms of e.g., English verbs, information about bound morphemes and idiosyncratic phrases like (kick the bucket) (cf. Ullman, 2004: 233). Ullman argued further that this declarative memory system is also responsible for storing word-specific knowledge, such as word sounds and word category (cf. Ullman, 2004: 244f). On the other hand, linguistic forms that are entirely predictable do not need to be memorized in this mental lexicon; they can be derived by grammatical rules (regular past tense with -ed in English). The knowledge of these rules may be defined as mental grammar and is part of the procedural rule based memory system. On the basis of this dissociation, Ullman et al. (cf. 1997a: 267) argued that irregular verb forms are unpredictable and constitute a fixed list. Ullman further suggested that these forms have to be memorized, whereas regular forms can be generated by rules (copying the stem of the verb and adding the past tense suffix -ed; for English). Under normal conditions, the retrieval of an irregular form, generally blocks the past tense rule (dig-*digged). But under certain circumstances (brain damage), irregular forms cannot be retrieved successfully from the memory any more. Instead, the past tense rule is applied to irregular verbs, resulting in so called 'overregularization errors' such as (dig-*digged).

It has moreover been revealed that these two parts of memory can be associated with different neurological structures. Studies of human amnesia and animal models have identified the medial temporal lobe memory structures, in detail the hippocampus, together with the entorhinal, perirhinal and parahippocampal cortex to be crucial for establishing declarative memory (Squire & Zola-Morgan, 1991). Zola-Morgan and Squire (1993) supposed also diencephalic structures to be part of the declarative system. The procedural memory system on the other hand has been stated to be composed of cortico-basal ganglia-thalamo-cortical circuits and the cerebellum (e.g., Squire & Zola, 1998).

Ullman suggested a similar neuronal dissociation for language. According to Ullman et al. (1997a) as well as Ullman (2001a, 2001b, 2001c, 2004), the temporal-parietal/medial-temporal declarative system underlies the mental lexicon, rule governed procedural memory, is given in Appendix IV.
products on the other hand, underly the frontal/basal ganglia procedural system. Thus it has been suggested that patients with damage to the temporal or parietal cortex should be affected worse at producing irregular past tense forms (dig-dug), retrieved from the memory, than regular, rule governed verbs (walk-walked). Because of a lack in retrieving irregular forms, the past tense rule, which should only be applied to regular verbs, is also applied to irregular forms. Therefore, overregularizations of irregular forms (dig-*digg) are supposed to occur. But, as these patients do not lack the regular past tense rule, they should be able to apply the past tense rule to novel regular verbs nevertheless (plag-plagged) (cf. Ullman et al., 1997a: 267).

In contrast to this, patients with impairments in the procedural memory should be worse at producing regular than irregular forms. This is due to disturbances in the mental grammar, stored in frontal-basal ganglia circuits. As these patients lack the past tense rule, they are supposedly not able to apply the rule to novel regular forms. Because these patients lack the regular past tense rule, overregularizations shouldn't occur in this patient group either (cf. Ullman et al., 1997a: 268).

Ullman however also strengthened that “In practice, these differences might be rather relative rather than absolute” (Ullman et al., 1997a: 268). Thus, in consecutive studies not all hypotheses addressed within this model, could be proved.

### 3.6.2 The model of lexicon and grammar and HD

The hypothesized model of lexicon and grammar has been tested in various psycholinguistic experiments, clinical contexts as well as in neuroimaging studies (Rubenstein, Garfield & Milliken, 1970; Stanners et al., 1979; Ullman, 1993; Clashen, Eisenbeiss & Sonnenstuhl-Henning, 1997; Marslen-Wilson & Tyler, 1997; Ullman, Bergida & O’Craven, 1997b; Sonnenstuhl, Eisenbeiss & Clashen, 1999; Ullman, 1999; Ullman & Gopnik, 1999; Beretta et al, 2003; Ullman et al., 2005; Vannest, Polk & Lewis, 2005b; Newman et al., 2007).

Prove of this theory has further been given from dissociations between the retrieval and the production of regular and irregular verbs, in HD patients. As HD affects mainly the striatum, which is part of the so called cortico-basal ganglia-thalamo-cortical loop and is thus associated with the procedural memory, rule application is assumed to be affected the most in this patient group. Evidence for this assumption is given from HD patients,
in morphological (presented next), as well as in syntactic rule application (presented in chapter 3.6 as well as in 3.7). Nevertheless, as stated previously, the strict dichotomy as stated by Ullman et al. (1997a) could not always be approved in clinical contexts.

For instance, in the study by Ullman et al. (1997a) the memory/rule dissociation has been tested on regular and irregular real and non verbs of English. In regular verbs and non verbs the ed-suffixation has been applied superfluously, which leads to oversuffixation errors such as (*walked-ed). Overregularization errors occurred as well, in terms of overregularizing irregular verbs (dig-*digged).

As HD is associated with excessive motor activity, such as chorea, it has been suggested that the specific kind of lesion leading to chorea, also lead to overactive rule use. This assumption could be proved, as chorea measures correlated significantly with the rate of overregularizations. Thus, it can be assumed that it is the specific kind of lesion that leads to chorea that also leads to overactive rule use. The default rule was overactive and hence resulted in overregularization as well as oversuffixation errors in HD patients. This gives evidence that such errors cannot be simply attributed to motor perseverations; this rather suggests a major role for the basal ganglia in rule use (cf. Ullman, 1997a: 274).

Longworth et al. (2005) replicated the study by Ullman et al. (1997a) with HD patients, but could not prove the role of the basal ganglia in language. In sum, mild HD patients made either no regularization errors, or a single regularization error. Thus, in whole it cannot be said that this patient group evidenced a specific impairment in linguistic rule application. Similarly, the moderate HD patients made few to no regularization errors and no multiple suffixed errors at all. In sum, no evidence could be shown of an association between a basal ganglia disorder and a deficient rule application. Nevertheless, Teichmann et al. (2005) replicated Ullman et al.’s (1997a) study as well, by using French verbs. However, in this study five types of verbs have been used, to test the theory of lexicon and grammar: regular verbs (e.g., arriver), subregular verbs (e.g., finir), irregular verbs (e.g., boir), regular nonverbs (e.g., froucher) and subregular nonverbs (e.g., pichir). In this study, there couldn't be found a difference between regular and irregular verbs, as predicted originally in the model stated by Ullman and colleagues (1997a). Even though, controls performed better than HD patients, there was no significant difference between HD group I and II. The only difference between the
two HD groups was with irregular verbs, with better performance in HD stage I. In general, HD patients performed better with irregular verbs than with non-verbs. Nevertheless patients showed worse performance in subregular non-verbs, compared to regular non-verbs. Impairment with subregular non-verbs in HD patients corresponded mainly with overregularizations, suggesting that the subrule impairment was compensated by the over-use of the default rule. Again, defective basal ganglia functions have been attributed to the overapplication of linguistic rules. Teichmann et al. (2006) investigated moreover the rule of the striatum in morphological processing. HD patients at an early stage (stage I and stage II) were assessed in grammatical judgement and lexical decision tasks, on conjugated verbs and non-verbs. In the grammatical judgement task patients made right/wrong judgements on auditorily presented verb (regular verbs and irregular verbs) and non-verb forms (regular non-verbs and subregular non-verbs). Error forms were constructed by adding incorrect suffixes to the verb and non-verb roots, resulting in the following error types: overregularizations, subregularizations, double suffixations and nonexistent French suffixes. Again, HD patients were mostly impaired on non-verbs; especially on subregular non-verbs. Errors mainly resulted from the overapplication of the productive default rule.

In the lexical decision task, Teichmann et al. (2006) used a speeded lexical decision task with verb versus non-verb decisions. In this task, HD patients showed a similar impairment in rule application, with spared lexical abilities, again in the domain of subregular non-verbs. In conclusion, it could be stated that the striatum has a core function in linguistic rule application, as problems in linguistic rule application do occur in HD patients with deficient procedural memory. However, there couldn't be found a difference between regular and irregular verbs, as predicted originally in strong dissociation model stated by Ullman and colleagues (1997a).
3.7 Syntactic performance in HD

Only marginal descriptions of syntactic changes in HD have been given by Gordon and Illes, back in 1987. Patients described, evidenced a diminished level of syntactic complexity as well as reductions in phrase length. Illes (1989) also reported HD patients to reduce syntactic complexity in conversations as well as to use predominantly closed class phrases. Evidence of syntactical difficulties have further been described by Murry (2000), Murray and Lenz (2001) as well as by Jenery, Copland and Murdoch (2006). More recently, grammatical aspects of HD spoken language have been identified as ungrammatical and fragmentic in the study by Heemskerk (2008). Specifically, 48% of all uttered sentences in the test, were ungrammatical. In detail, ungrammatical sentences were incorrect in word order, incorrect because of ommissions of subjects, verbs, objects, articles, prepositions as well as because of incorrect verb positioning and other not specified grammatical failures (cf. Heemskerk, 2008: A10). The hypothesis of the declarative/procedural model (Ullman et al., 1997a) has been stated by Teichmann et al. (2005), (2007) and (2008) also for syntactic processes. The function of the striatum, mainly affected in HD, in syntax, has further been proved by Friederici, Yves von Cramon & Kotz (1998), Moro et al. (2001), Friederici and Kotz (2003) and Kotz et al. (2003). Regulative functions of the basal ganglia (respectively the striatum) in the application of syntactic rules has additionally been mentioned within leading research literature, concerning language functions in Parkinson's disease (e.g., Lieberman et al., 1990; Grossman et al., 1992a & 1992b; Lieberman et al., 1992; Grossman et al., 1993). On the basis of this evidence, it seems evident that syntactic changes are also to occur in the course of HD, as a basal ganglia disorder. In detail, Teichmann et al. (2005) tried to examine the rule versus lexicon hypothesis by contrasting rule application and lexical processes in syntactic tasks. Teichmann et al. (2005) argued for a crucial role of the striatum in rule application not just in morphological domains, as stated by Ullman et al. (1997a), but also in syntactic and non-linguistic domains such as mathematics81. 15 HD patients in early stage of the disease (stage I and stage II) as well as 20 healthy volunteers were tested in a sentence picture matching task. Passive-active and subject-object relative clauses, paired with one picture at a time, were used within a sentence picture matching task. The sentences

81 In terms of brevity constraints within this chapter the author of this thesis will only focus on the role of the striatum in syntactic rule application.
were presented in four conditions: active, passive, plausible and non-plausible. Therefore, active-plausible, active-non-plausible, passive-plausible as well as passive-non-plausible sentences could be formed. In this paper three different comprehension strategies were assumed for subjects to get to the correct meaning of the various sentences. Depending on the sentence presented, different strategies have to be used. Semantic information is needed to get to the meaning of the sentence (girl, flower, watering; the girl waters the flower). Secondly the word order of each specific language is crucial for understanding a sentence (the girl = S, waters = V, the flower = O). And finally a syntactic analysis is assumed which delivers the thematic roles\(^\text{82}\) of the sentence, based on semantic information and word order of the specific language (the girl waters the flower and not the flower waters the girl). In sentences that are passive and non-plausible, only syntactic rules can allow comprehension of the sentence. By guessing, or just by using semantic information and word order it is not possible to get to the proper meaning of the sentence. You have to understand the thematic roles of the sentence in order to understand who is doing what to whom. Thus passive, non-plausible sentences were predicted to be the most impaired in HD patients. This could be confirmed within this study. In detail, HD patients in stage II, were more impaired in this task than HD patients in stage I. It is not surprising that active and plausible sentences could be performed easier than passive and non-plausible sentences, thus the most problems could be shown, as assumed above, in passive, non-plausible sentences. Because passive, non-plausible sentences can just be understood by the use of syntactic rules, those rules are supposed to be impaired in HD patients. 

Teichmann et al. (2005) argued that this confirms again the dual route model of the procedural/declarative model stated by Ullman et al. (1997a), that rule application (not just in morphological but also in syntactic conditions) is accomplished by procedural memory, within parts of the so called cortico-basal ganglia-thalamo-cortical circuit. For further support of those results, a PET correlational study (using the same language tasks as presented in Teichmann et al., 2005) has been administered to 31 HD patients in early stages (I and II) as well as to 20 healthy controls, by Teichmann et al. (2008). As the striatum is most affected in HD patients in early stages (I and II), these results

\(^{82}\) The term thematic/semantic role/relation (Wilkins, 1988) is synonymous to the label theta role stated within generative grammar (Chomsky, 1981) as well as to Fillmore's (1968) deep case in his case grammar. It describes the underlying relationship within a sentence that a participant has with the main verb in a clause. If within a sentence someone named John hits someone who is called Bill, then the semantic role of John is the agent of this event and the semantic role of Bill can be seen as the patient (cf. Payne, 1997a: 47).
moreover give evidence for the role of the striatum in rule application. In detail, results show bilateral activation in the ventral striatum, the left putamen and left pallidum, for passivated sentences (derived by syntactic rules), as well as activation within dorsally localized regions of the left caudate head for active sentences (derived by the lexicon)\textsuperscript{83}.

Results of this study couldn't in detail resolve the lexicon/rule conflict, by showing involvement of only subparts of the striatum (ventral/dorsal dissociation) in both lexicon and rule governed tasks. But as mentioned in chapter 1.4, as HD's neuropathology spreads progressively from the striatum to various cortical regions (Jernigan et al., 1991; Thieben et al., 2002; Rosas et al., 2003; Douaud et al., 2006; Henley et al., 2006; Rosas et al., 2006; Rosas et al., 2008a; Rosas et al., 2008b; Squitieri et al., 2009) thus the involvement of other brain areas, due to the hypothesized cortico-basal ganglia-thalamo-cortical circuit is not impossible (cf. Teichmann et al., 2008: 1053). Further proof of the assumed role of the striatum in syntax, could be given in another study. Teichmann et al. (2007) analyzed the function of the striatum in sentence processing. Within a semantic priming task, 24 patients suffering from HD at early stage (stage I), were present with idiomatic prime sentences (kick the bucket), manipulated idiomatic sentences (Peter kicked the bucket), idiom-derived passive sentences (Peter was kicked by the bucket), idiomatic sentences with word changes (Peter crushed the bucket) as well as idiom-derived passive sentences with word changes (Peter was crushed by the bucket). Not surprisingly, HD patients performed normally with idioms and idiom-derived sentences, because they are supposed to be stored in the mental lexicon as separate language chunks (Ullman et al., 1997a). On the other hand, HD patients displayed selective abnormalities with all passivated, rule-derived sentences. Thus, it can be concluded that HD patients, even at an early stage of their disease, have problems with syntactical rule use. These deficits could be explained by a deficient striatum, under normal conditions associated with linguistic rule application. However, the problem processing syntactical rules could alternatively be explained by Grodzinsky's (1990) hypothesis of 'Trace Deletion' (for details see chapter 7.4.1).

\textsuperscript{83} Similar results could also be found for results in inflectional morphology as presented in Teichmann et al. (2005). In detail, PET results showed activation of dorsal portions of the caudate nucleus for lexical operations as well as more ventral proportions of the caudate nucleus and the putamen for rule application.
3.8 Summary

As exemplified above, HD literature concerning language disturbances, is very controversial. Whereas primary linguistic disturbances were not evident in the use of traditional aphasia batteries, such as the WAB and the ATT (Podoll et al., 1988, Wallesch & Fehrenbach, 1988 as well as Chenery, Copland & Murdoch, 2002) linguistic disturbances were profound by using language tests such as the TLC-E; TOWK, TWT-R (Chenery, Copland & Murdoch, 2002), as within other more complex language tasks, such as tested by Hartelius et al. (2008).

Narrative as well as descriptive language performances have further been described to be impaired in HD patients within the studies by Gordon and Illes (1987), Wallesch and Fehrenbach (1988), Illes (1989), Murray (2000), Murray and Lenz (2001) as well as by Jensen, Chenery and Copland (2006).

Uncertainty exists concerning naming performace in HD. Whereas Kennedy et al. (1981), Podoll et al. (1988), Hodges, Salmon and Butters (1991) as well as Frank, McDade and Scott (1996) did show deficiencies in naming, using the BNT, Chenery, Copland and Murdoch (2002) couldn't find naming problems in HD patients, using the same assessment. Furthermore, there exists uncertainty in terms of where these deficits in naming – if there are some – are due to. Hodges, Salmon and Butters (1991) are in strong belief that naming deficits in HD patients result from visual problems, whereas Frank, McDade and Scott (1996) argue that naming deficits are semantically originated.

Discourse comprehension difficulties could be found within the study by Murray and Stout (1999) as well as difficulties comprehending prosodic markers have been stated by Speedie et al. (1990).

Whether, various disturbances in fluency performance were found by all authors referred to in this thesis (Butters et al., 1986; 1987; 1988; Tröster et al., 1989; Randolph et al., 1993; Monsch et al., 1994; Ross and Hodges, 1994; Tröster et al., 1998; Rich et al., 1999; Ho et al., 2002; Péran et al., 2003a), it is unsure where these deficits arise from, as well as if the methodology used within these tasks has been appropriate.

Morphology is moreover assumed to be affected in the course of HD (Ullman et al., 1997a; Teichmann et al., 2005, 2006). This assumption has been rejected however by Longworth et al. (2005). Morphological deficits have been shwon to be primarily due to a problem in the application of morphological rules.
Moreover, syntactic errors could be found by the following authors: Gordon and Illes (1987), Illes (1989), Murray (2000), Murray and Lenz (2001), Teichmann et al. (2005), Jensen, Chenery and Copland (2006), Teichmann et al. (2007), Heemskerk et al. (2008), as well as by Teichmann (2008). Similarly as in the case of morphology, also rule use, in detail syntactic rule use, seemed to be defective in HD patients.

A general overview about studies dealing with language functions in HD is given in Table 4, below. In detail, within this table, studies exclusively dealing with language changes in HD are referred to; presented in the order how they have been addressed to in the literature review. Studies cited only on the basis of abstracts from scientific meetings as well as based on review papers, have not been included in this summary.
Table 4 shows a comprehensive summary on studies dealing with language functions in HD

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>N</th>
<th>Language Assessments</th>
<th>HD_Confirmed By</th>
<th>HD_Stages</th>
<th>Depression-Dementia-Chorea</th>
<th>Language Disturbance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Kennedy et al., 1981</strong></td>
<td>30</td>
<td>BNT, RTT, NCCEA</td>
<td>XXX</td>
<td>mild, moderate,</td>
<td>XXX</td>
<td>yes: BNT, RTT, NCCEA</td>
</tr>
<tr>
<td><strong>Podolli et al., 1988</strong></td>
<td>45</td>
<td>AAT</td>
<td>positive family history &amp; involuntary movements</td>
<td>early, middle, late</td>
<td>SKAUB<strong>84</strong></td>
<td>no: no primary linguistic errors</td>
</tr>
<tr>
<td><strong>Chenery, Copland &amp; Murdoch, 2002</strong></td>
<td>13</td>
<td>WAB, BNT, TLC-E, TOWK, TWT-R</td>
<td>positive family history &amp; involuntary movements</td>
<td>Shoulson &amp; Fahn, 1979; stage mean 2.85</td>
<td>MDRS<strong>85</strong></td>
<td>yes: TLC-E, TOWK, TWT-R no: BNT, WAB</td>
</tr>
<tr>
<td><strong>Wallesch &amp; Fehrenbach, 1988</strong></td>
<td>18</td>
<td>AAT</td>
<td>positive family history &amp; involuntary movements</td>
<td>Shoulson &amp; Fahn, 1979; stage I-II</td>
<td>XXX</td>
<td>no: no primary linguistic errors</td>
</tr>
<tr>
<td><strong>Gordon &amp; Illes; 1987</strong></td>
<td>12</td>
<td>spontaneous language &amp; Cookie Theft Picture</td>
<td>XXX</td>
<td>XXX</td>
<td>XXX</td>
<td>yes: mainly syntax</td>
</tr>
<tr>
<td><strong>Illes, 1989</strong></td>
<td>10</td>
<td>open end autobiographical questions</td>
<td>positive family history, involuntary movements, dementia &amp; neuroimaging (CT)</td>
<td>early &amp; middle</td>
<td>XXX</td>
<td>yes: mainly syntax</td>
</tr>
<tr>
<td><strong>Murray &amp; Lenz, 2001</strong></td>
<td>9</td>
<td>conducted spontaneous language about themselves, hobbies, profession</td>
<td>XXX</td>
<td>XXX</td>
<td>Hamilton &amp; MDRS</td>
<td>yes: syntax</td>
</tr>
<tr>
<td><strong>Murray 2000</strong></td>
<td>10</td>
<td>Cookie Theft Picture &amp; Grocery Scene</td>
<td>genetic testing, involuntary movements &amp; positive family history</td>
<td>XXX</td>
<td>Hamilton &amp; MDRS</td>
<td>yes: syntax, informativeness</td>
</tr>
<tr>
<td><strong>Jensen, Chenery &amp; Copland, 2006</strong></td>
<td>6</td>
<td>Cookie Theft Picture</td>
<td>positive family history &amp; involuntary movements</td>
<td>XXX</td>
<td>MDRS</td>
<td>yes: syntax</td>
</tr>
<tr>
<td><strong>Hodges, Salmon &amp; Butters, 1991</strong></td>
<td>16</td>
<td>Boston Naming Test</td>
<td>positive family history, involuntary movements &amp; dementia</td>
<td>Shoulson &amp; Fahn, 1979; stage I-IV</td>
<td>MMSE</td>
<td>yes: naming (visually originated)</td>
</tr>
<tr>
<td><strong>Frank, McDade &amp; Scott, 1996</strong></td>
<td>12</td>
<td>Peabody Picture Vocabulary Test, Visual Recognition Test &amp; Confrontation Naming Test as part of the BNT</td>
<td>positive family history &amp; involuntary movements</td>
<td>mild to moderately demented</td>
<td>HIRS**, ZRS**, GDS<strong>88</strong></td>
<td>yes: naming (semantically originated)</td>
</tr>
<tr>
<td><strong>Murray &amp; Stoot, 1999</strong></td>
<td>9</td>
<td>Discourse Comprehension Test</td>
<td>genetic testing, involuntary movements &amp; positive family history</td>
<td>Shoulson &amp; Fahn, 1989; mean TFC score (9.11)</td>
<td>Hamilton &amp; MDRS</td>
<td>yes: discourse comprehension</td>
</tr>
</tbody>
</table>

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84 SKAUB=Skala für abnorme unwillkürliche Bewegungen (Kulhanek, 1980)
85 MDRS=Mattis Dementia Rating Scale (Mattis, 1988)
86 HIRS=Hachinski Ischemia Rating Scale (Hachinski et al., 1975)
87 ZRS=Zung Rating Scale for depression (Zung, 1965)
88 GDS=Global Deterioration Scale for dementia severity grouping (Reisberg et al., 1982)
<table>
<thead>
<tr>
<th>Year</th>
<th>N</th>
<th>Test/Task Description</th>
<th>Family History/Involuntary Movements/Dementia</th>
<th>Other Measurements</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Speedie et al., 1990</td>
<td>6</td>
<td>denote prosody/discriminate prosody</td>
<td>dementia (&gt; 24 on MMSE)</td>
<td>MMSE early</td>
<td>yes: comprehension of prosody</td>
</tr>
<tr>
<td>Butters et al., 1986</td>
<td>20</td>
<td>letter fluency</td>
<td>positive family history &amp; presence of choreatic movements</td>
<td>MDRS early &amp; advanced</td>
<td>yes: letter fluency</td>
</tr>
<tr>
<td>Butters et al., 1987; 1988</td>
<td>12</td>
<td>letter &amp; category fluency (animals)</td>
<td>positive family history, involuntary movements &amp; dementia</td>
<td>MDRS Shoulson &amp; Fahn, 1979, stage II-IV</td>
<td>yes: letter and category fluency</td>
</tr>
<tr>
<td>Randolph et al., 1993</td>
<td>24</td>
<td>category fluency (supermarket)</td>
<td>positive family history &amp; involuntary movements</td>
<td>MDRS Shoulson, 1981; stage I &amp; II</td>
<td>yes: category fluency</td>
</tr>
<tr>
<td>Rosser &amp; Hodges, 1994</td>
<td>10</td>
<td>letter &amp; category fluency (3 living &amp; 3 man made categories)</td>
<td>positive family history, involuntary movements, dementia &amp; (MRI/CT)</td>
<td>MDRS XXX</td>
<td>yes: letter and category fluency; but more on letter fluency</td>
</tr>
<tr>
<td>Monsch et al., 1994</td>
<td>42</td>
<td>letter &amp; category fluency</td>
<td>positive family history, involuntary movements &amp; dementia</td>
<td>MDRS XXX</td>
<td>yes: letter and category fluency</td>
</tr>
<tr>
<td>Tröster et al., 1989</td>
<td>20</td>
<td>category fluency (supermarket)</td>
<td>positive family history, involuntary movements</td>
<td>MDRS XXX</td>
<td>yes: category fluency</td>
</tr>
<tr>
<td>Rosser &amp; Hodges, 1994</td>
<td>10</td>
<td>letter &amp; category fluency (3 living &amp; 3 man made categories)</td>
<td>positive family history, involuntary movements, dementia &amp; (MRI/CT)</td>
<td>MDRS XXX</td>
<td>yes: letter and category fluency; but more on letter fluency</td>
</tr>
<tr>
<td>Monsch et al., 1994</td>
<td>42</td>
<td>letter &amp; category fluency</td>
<td>positive family history, involuntary movements &amp; dementia</td>
<td>MDRS XXX</td>
<td>yes: letter and category fluency</td>
</tr>
<tr>
<td>Tröster et al., 1998</td>
<td>24</td>
<td>letter &amp; category fluency (animals)</td>
<td>positive family history, involuntary movements &amp; dementia</td>
<td>MDRS XXX</td>
<td>yes: letter &amp; category fluency; reduced switching and smaller clusters</td>
</tr>
<tr>
<td>Rich et al., 1999</td>
<td>72</td>
<td>letter fluency</td>
<td>genetic testing, positive family history &amp; involuntary movements</td>
<td>MDRS XXX</td>
<td>yes: reduced switching</td>
</tr>
<tr>
<td>Ho et al., 2002</td>
<td>21</td>
<td>letter &amp; category fluency (animals)</td>
<td>genetic testing, positive family history, involuntary movements &amp; dementia</td>
<td>MDRS XXX</td>
<td>yes: progressive impairment in switching</td>
</tr>
<tr>
<td>Péran et al., 2003</td>
<td>26</td>
<td>verb and noun generating task</td>
<td>genetic testing, positive family history, involuntary movements, dementia &amp; depression</td>
<td>MDRS XXX</td>
<td>yes: switching, problems with verbs</td>
</tr>
<tr>
<td>Ullman et al., 1997a</td>
<td>17</td>
<td>past tense formation (regular/irregular engl. verbs)</td>
<td>XXX</td>
<td>MMSE XXX</td>
<td>yes: ling. rule application</td>
</tr>
<tr>
<td>Teichmann et al., 2005</td>
<td>30</td>
<td>conjugation task/ sentence-picture match</td>
<td>genetic testing, involuntary movements, dementia &amp; (MRI)</td>
<td>MDRS &amp; UHDRS motor Shoulson, 1981; stage I &amp; II</td>
<td>yes: ling. rule application</td>
</tr>
<tr>
<td>Longworth et al., 2005</td>
<td>10</td>
<td>past tense production/comprehension</td>
<td>genetic testing, involuntary movements &amp; dementia</td>
<td>MDRS &amp; UHDRS motor mild to moderately demented</td>
<td>no: ling. rule application</td>
</tr>
<tr>
<td>Teichmann et al., 2006</td>
<td>15</td>
<td>acceptability judgements &amp; lexical decision task of conjugated verbs</td>
<td>genetic testing, involuntary movements, dementia &amp; (MRI)</td>
<td>MDRS &amp; UHDRS motor Shoulson, 1981; stage I</td>
<td>yes: ling. rule application</td>
</tr>
<tr>
<td>Teichmann et al., 2008</td>
<td>31</td>
<td>PET study based on Teichmann et al., 2005</td>
<td>genetic testing, involuntary movements &amp; dementia</td>
<td>MDRS &amp; UHDRS motor Shoulson, 1981; stage I &amp; II</td>
<td>yes: striatum is involved in rule application</td>
</tr>
<tr>
<td>Teichmann et al., 2007</td>
<td>24</td>
<td>priming task</td>
<td>genetic testing, involuntary movements &amp; dementia</td>
<td>MDRS &amp; UHDRS motor Shoulson, 1981; stage I</td>
<td>yes: linguistic rule application</td>
</tr>
</tbody>
</table>
3.9 Critical discussion of the literature

More and more literature is arising, trying to test language functioning in HD. So far the results of these studies are controversial. Whereas some do show linguistic abnormalities in the course of HD, others try to prove the opposite. This inconsistency in results may be due to the following criteria:

1) Lack of genetic testing
2) Lack of information about HD stage
3) Lack of information about motor, psychiatric and neuropsychological disturbances
4) Other factors influencing language outcome
5) Doubtful model of lexicon and grammar – contrasting 'naturalness'
6) Lack of embedding into linguistics

3.9.1 Lack of genetic testing

One reason for contradicting results concerning language functions in HD, may be the lack of genetic testing, prior the inclusion in the study. Whereas in some studies no explanation has been given regarding the confirmation of HD, in terms of genetic testing (Kennedy et al., 1981; Gordon & Illes, 1987; Ullman et al., 1997a; Murray & Lenz, 2001), inclusion was based in other studies solely on the basis of a positive family history and the presence of involuntary movements such as e.g., chorea (Butters et al., 1986; Podoll et al., 1988; Wallesch & Fehrenbach, 1988; Tröster et al., 1989; Randolph et al., 1993; Frank, McDade & Scott, 1996; Chenery, Copland & Murdoch, 2002; as well as Jensen, Chenery & Copland, 2006). Thus, it can be assumed that maybe also misdiagnosis (e.g., Tourette's syndrome due to the existence of involuntary movements), were likely to occur.

In other studies, HD diagnosis was based on positive family history, involuntary movements such as chorea and the presence of dementia (Butters et al., 1987 & 1988; Hodges, Salmon & Butters, 1991; Monsch et al., 1994; Tröster et al., 1998).

Moreover, dementia solely, as rated by the MMSE (Folstein, Folstein & McHugh, 1975) has been used as a criteria for inclusion, by Speedie et al. (1990). Nevertheless, dementia itself is not one of the first symptoms to occur in HD, thus this is not a very
sensitive criteria to use for an inclusion in the study. Additionally to a positive family history of HD, the presence of involuntary movements such as chorea and the presence of neuropsychologic factors such as dementia, more recently neuroimaging techniques have been used, showing striatal degeneration and thus justifying the inclusion of study subjects. In more detail, CT scans have been used in the study by Illes (1989), MR and CT scans on the other hand by Rosser and Hodges (1994). Only in the latest publications regarding language changes in HD, a genetic test based on the number of CAG repeats has been used additionally to factors such as positive family history, involuntary movements, dementia and/or neuroimaging results, representing the most sensitive criteria for inclusion in the study (Murray & Stout, 1999; Rich et al., 1999; Murray, 2000; Ho et al., 2002; Péran et al., 2003; Longworth et al., 2005; Teichmann et al., 2005; 2006; 2007 & 2008).

3.9.2 Lack of information about HD stage

Inconsistent results regarding language functions in HD, are moreover due to a lack of information about HD stage. In studies by Gordon and Illes (1987), Tröster et al. (1989), Monsch et al. (1994), Rosser and Hodges (1994), Ullman et al. (1997a), Tröster et al. (1998), Rich et al. (1999), Murray (2000), Murray and Lenz (2001), Ho et al. (2002), Péran et al. (2003a) as well as by Jensen, Chenery and Copland (2006), no information has been given about HD stage as rated by Shoulson and Fahn (1979) or Shoulson (1981). In other studies, a non-standardized description has been provided in terms of (mild-moderate-severe or early-middle-late) HD stage, with a lack of explanation how these stages have been defined (Kennedy et al., 1981; Butters et al., 1986; Podoll et al., 1988; Illes, 1989; Speedie et al., 1990; Frank, McDade & Scott, 1996).

In the study by Longworth et al. (2005) dementia has been used for establishing HD stages, dividing patients included, into mildly and moderately demented group. Against that, in studies considering HD stage, as rated by Shoulson and Fahn (1979) or Shoulson (1981), HD patients at different stages have been included in the following studies: Butters et al. (1987), Butters et al. (1988), Wallesch and Fehrenbach (1988), Hodges, Salmon and Butters (1991), Randolph et al. (1993), Murray and Stout (1999), Chenery, Copland and Murdoch (2002), Teichmann et al. (2005; 2006; 2007 and 2008).
Due to the fact that HD patients' performance not only in the cognitive, but also in the motor and the psychiatric domain, does change from stage to stage, no consideration at all, or the inclusion of patients from different HD stages, may therefore have been crucial for contributing to contradictory study results.

### 3.9.3 Lack of information about motor, psychiatric and neuropsychological deficits

Furthermore, the consideration of psychiatric, neuropsychological as well as motor disturbances (in terms of standardized testing), have to be taken into consideration, in order to look objectively at the outcomes of the language screening. Unfortunately, no consideration of depression, dementia or motor disturbances such as chorea, has been given in the studies by Kennedy et al. (1981), Gordon and Illes (1987), Wallesch and Fehrenbach (1988), Illes (1989), Randolph et al. (1993), as well as Ullman et al. (1997a).

Only in the study by Péran et al. (2003a) dementia, depression as well as motor disturbances, rated by the MMSE, the MDRS and the UHDRS motor score, have been taken into account. In all other studies referred to within this thesis, either one or even two out of the three core symptoms of HD, haven't been rated by standardized testing procedures. Frank, McDade and Scott (1996) considered dementia and psychiatric aspects, but using rather uncommon and uncomparable testing methods, such as the Hachinski Ischemia Rating Scale (= HIRS) (Hachinski et al., 1975) for the exclusion of vascular dementia, the Zung Rating Scale (= ZRS) (Zung, 1965) for the determination of depression, as well as the Global Deterioration Scale (= GDS) (Reisberg et al., 1982) for dementia severity grouping.

### 3.9.4 Other factors influencing language outcome

There are two further factors (language material used and dysarthria) which could also influence HD patient's linguistic abilities. Primarily, the language material used, has a strong impact on the language outcome (Brown & Marsden, 1988). Specifically, aphasia batteries, designed for cortically
injured patients, are not sensitive enough to reveal language problems in subcortical
dementias. But these tests have also been used in HD patients and may be thus another
reason for negative results concerning the evidence of language problems in HD
patients. This assumption could be proved, by showing that Podoll et al. (1988),
Wollesch and Fehrenbach (1988) as well as Chenery, Copland and Murdoch (2002),
using cortical aphasia batteries (AAT and WAB), focusing on 'primary language
abilities' (cf. Chenery, Copland & Murdoch, 2002: 467) could not prove any language
problems in HD patients. Contrastingly, tests addressing 'complex language
functions' (cf. Chenery, Copland & Murdoch, 2002: 467), as tested e.g., by the TLC-E,
TOWK, TWT-R, showed linguistic deficiencies in the same patient group.
Mega and Alexander (1994) identified a core profile of aphasia due to subcortical
lesions, including impaired executive language functions such as word fluency, sentence
generation and discourse, but spared responsive language such as comprehension,
repetition and sometimes also naming. Based on these findings, Jensen, Chenery and
Copland (cf. 2006: 63) inferred that tasks which are associated with a 'high number of
degrees of freedom' such as propositional aspects of language, are more frequently
affected in patients with subcortical lesions, than others involving a 'lower degree of
freedom', such as responsive speech. Thus it seems evident that the test material itself
influences the language outcome.
Various other 'complex language functions' such as propositional language (e.g., picture
descriptions) as well as morphological and syntactical rule application could be shown
to be deficient in HD patients (Gordon & Illes, 1987; Illes, 1989; Speedie, 1990;
Murray & Lenz, 2001; Murray 2000; Teichmann et al., 2005, Jensen, Chenery &
Copland, 2006; Teichmann et al., 2007; 2008; Heemskerk, 2008).
In HD patients, language disturbances could also be found in the domain of narrative
language (Gordon & Illes, 1987; Illes, 1989; Murray, 2000; Murray & Lenz, 2001;
Discourse Comprehension Test, that especially questions pertaining to details and
implied information, as 'complex language functions', lead to difficulties in
comprehension, in HD patients. Nevertheless, speech characteristics associated with HD
are described as a hyperkinetic dysarthria with a number of variable dysarthric speech
features (see for instance Yorkston et al., 1999). Thus it seems evident that these factors
also contribute negatively to the language outcome. Hence, caution is warranted in the
interpretation of results concerning propositional language.
3.9.5 Doubtful model of lexicon and grammar – contrasting 'naturalness'

As reported in chapter 3.6.1 Michael Ullman from the Massachusetts Institute of Technology, stated the theory of a dual route model of lexicon and grammar, which is supposed to account for processes of inflectional morphology. This theory can be traced back to generative linguistics founded by Chomsky in 1965. In terms of morphology, Ullman states that there are default forms, which are regular and can thus be derived by morphological rules, in contrast to irregular forms which have to be derived from the mental lexicon. In Ullman's original model, the memory system responsible for rule derived forms is the procedural memory, in contrast to the declarative system which is responsible for the stored forms. Ullman tries to show that HD patients with deficits in the procedural memory, associated with the basal ganglia, have especially troubles with rule derived forms. Even though Ullman's generative concept has been tried to be proved in various contexts (Rubenstein, Garfield & Milliken, 1970; Ullman, 1993; Clashen, Eisenbeiss & Sonnenstuhl-Henning, 1997; Marslen-Wilson & Tyler, 1997; Stanners et al., 1997; Ullman, Bergida & O'Craven, 1997b; Sonnenstuhl, Eisenbeiss & Clashen, 1999; Ullman, 1999; Ullman & Gopnik, 1999; Beretta et al, 2003; Ullman, 2005; Vannest, Polk & Lewis, 2005b; Newman et al., 2007), the author of this thesis regards the assumption of this theory as doubtful and will explain in the next section of this chapter, why this is so. Ullman's original theory is doubtful mainly because it acts against the concept of 'naturalness' stated within the concept of 'Natural Morphology'. In all studies (also in various other contexts, not just based on HD patients) Ullman stresses, on the example of English verbal inflection (only!), that because of a defective procedural memory, responsible for rule governed forms, only regular forms lack in contrast to lexicon derived irregular forms.

But 'Natural Morphology' (cf. Wurzel, 1989: chapter 3) gives evidence that only the opposite can be possible in terms of a linguistic concept of 'naturalness' and thus regular forms are expected to be intact in contrast to defective irregular forms. Interestingly, Ullman's theory is based only on evidence given by inflectional morphology, where a distinction between regular and irregular verbs, respectively between weak (walk-walked) versus strong verbs (go-went), could still be plausible. But this approach is already

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89 In order to follow the subsequent discussion on 'Natural Morphology' versus the dual-route model stated by Ullman (1997a) and prior by Chomsky (1965), see chapter 7.3.1.
inadequate for strong inflecting languages such as Slavic and Baltic languages (Dressler, 2003) and even more for word formation processes such for instance German denominal adjective formation rules with competing productive suffixes such as -ig, -isch,-lìch, where none of them presents the default (Pounder, 2000). Evidence for the concept of 'naturalness' and thus contrasting the dual-route model stated by Ullman, is given in various contexts in a similar way, starting with Stampe (1979) and Jakobson (1941) for phonology and Dressler (1977a), Mayerthaler (1981) and Wurzel (1989) for the domain of morphology. Mainly Wurzel (cf. 1989: 64f) pointed out in detail why deficits in inflectional morphology should be the other way round as presented by Ullman back in 1997a; such as there is more likely a deficit for irregular forms in contrast to intact regular forms. In detail, there is strong evidence from language change, neologisms, nonsense words, linguistic errors, aphasic disorders and language acquisition (first and second) that there is a uniform direction that always strong verbs become weak, weak verbs are acquired before strong verbs and that strong verbs are getting lost the first (cf. Wurzel, 1989: 64f). In a more general sense, addressing 'naturalness' as stated first by Jakobson back in 1968, more marked forms (strong verbs) are those which are acquired the last and which on the other hand are getting lost at first in the course of language disturbances. This is just logical as weak forms are inflected additive segmental (fragen – frag-te; walk – walk-ed) and are thus transparent and uniform as well as maximal iconic and thus maximal natural and also minimal marked (cf. Wurzel, 1989: 67). In sum, the inflectional class with the more natural additive forms is also the more natural class. Thus, within Mayerthaler's (1981) concept of 'naturalness' there is a case of morphological naturalness whenever a formal 'more' corresponds to a semantic 'more. Because of all these reasons as just presented, Ullman's (1997a) theory is doubtful and so are results of his study, as they are in contrast to 'naturalness' concepts. Moreover, Ullman's (1997a) concept is bizzare as it could never be applied appropriately to the domain of word formation, including derivation, as there is nothing like one default form and one irregular form e.g., in German's word formation suffixes, as already stated before. As Ullman stated that “In practice, these differences might be rather relative than absolute” (Ullman et al., 1997a: 268) and as could be shown by various studies referring to Ullman's concept (Teichmann et al., 2005; Longworth et al., 2005; Teichmann et al., 2006) as the strict dissociation of lexicon and grammar could not be proved caution is warranted in the interpretation of results in terms of morphological deficits in HD patients.
3.9.6 Lack of embedding into linguistics

It can be criticised moreover that in the whole literature presented above, so far no detailed analysis of errors made, has been given. Error analysis was always based only on statistical analysis and/or superficial qualitative accomplishments, such as 'syntax is reduced in HD' (e.g., Illes, 1989). But in order to account for errors made, so far no linguistic theory has been used. In order to counteract the inconsistency in results, as just mentioned, 7 study aims were formulated for this thesis, as named in chapter 3.9.6.1. Based on these 7 study aims, 10 study hypotheses have been derived, as presented in chapter 3.9.6.2.

3.9.6.1 Study aims

1) A comprehensive analysis of the so far published literature dealing with language functioning in HD is given, as presented in Table 4, to reveal the underlying mechanisms of the controversial study outcomes of the past.

2) Within this project a language screening has been constructed that served to assess linguistic functioning in the domains of semantics, morphology and syntax, reported to be disordered in HD. The various tasks provided, differed in terms of their 'degree of freedom' (cf. Jensen, Chenery & Copland, 2006: 63) and thus also in their 'degree of complexity in language functioning' (cf. Chenery, Copland & Murdoch, 2002: 467). A detailed description concerning the division of tasks into high/low freedom tasks, as well as into tasks with primary/complex linguistic functioning, will be given in chapter 4.3. Moreover, language tasks referred to as being 'complex language functions', will be addressed not only within a quantitative analysis, but also within a qualitative-linguistic analysis, addressing errors made.

3) Moreover a detailed quantitative (statistical) as well as qualitative (linguistic classification of errors made) analysis of the performance on the language screening, of the 20 genetically approved HD patients (in contrast to the 20 healthy controls), will be given.

4) Regarding the fact that HD is associated with three core symptoms (psychiatric,
neuropsychological and neurological), the outcome of the patient's language screening will be correlated with their depression, dementia, as well as motor disturbance rate. Due to the fact that dementia, depression as well as motor disturbances have an impact on the language performance, results of the language screening will be correlated with rates on the HDRS, the MMSE and the UHDRS motor score.

5) Additionally, language results will be correlated with 'days since disease onset' (disease progression measure) under control of the 'number of CAG repeats' (disease onset measure). These measures should always be considered under the control of each other, as already discussed previously in chapter 1.2. Nevertheless, it is very likely that the score 'days since disease onset', under control of the 'number of CAG repeats' will correlate statistically significant with the language outcome measures. The further progressed HD is (the more days since disease onset and the higher the number of CAG repeats) the worse language performance is supposed to become.

6) Moreover, it will be examined if there is a difference in HD patients' performance on the language screening, regarding the stage of HD administered by the Shoulson Total Functional Capacity Scale (= TFCS) (1979). This aim has been formulated on basis of the hypothesis, that language disturbances are already to occur in early stages of the disease (I, II), when mainly the striatum is to degenerate (Vonsattel et al., 1985; Aylward et al., 1996; Vonsattel & DiFiglia, 1998; Vonsattel, 1999; Aylward et al., 2000; Clark, Lai & Deckel, 2002; Aylward et al., 2003 & 2004; Paulsen et al., 2004 as well as Ciarmiello et al., 2006). Nevertheless, this hypothesis is based on the assumption that there is a core function of subcortical areas in language, as dysfunctions of the striatum are assumed to lead to interruptions within the cortico-basal ganglia-thalamo-cortical circuits and thus contributing negatively onto the language outcome.

7) Theoretical assumptions about the striatum's function in language will be strengthened by the implementation of errors found within cortico-basal ganglia-thalamo-cortical circuit models. This is in order to justify basal ganglia functions in language, on the example of Huntington's disease. Further errors made will be tried to be explained by linguistic theories, such as the concept of 'Natural Morphology' and the 'Trace Deletion Hypothesis'. 
3.9.6.2  **Study hypotheses**

Based on these 7 study aims, 10 hypotheses have been derived for this study.

1) There is a statistically significant difference between patients and controls in regards to their performance in the language screening (language production and language comprehension).

2) Within subtasks of the language production subtest, there is a statistically significant difference between the performance of controls and patients.

3) Within subtasks of the language comprehension subtest, there is a statistically significant difference between the performance of controls and patients.

4) Within patients, there is a statistically significant effect of the HD stage on the performance in the language screening (language production and language comprehension).

5) Within patients, there is a statistically significant effect of the HD stage on the various subtasks of the language production subtest.

6) Within patients, there is a statistically significant effect of the HD stage on the various subtasks of the language comprehension subtest.

7) There is a statistically significant negative correlation between the disease duration score 'days since disease onset' (disease progression measure) under control of the 'number of CAG repeats' (disease onset measure) and the language screening (language production and language comprehension) in HD patients. This theory is based on the assumption that the more advanced HD is, the more language disturbances are supposed to occur as well.

8) Because only patients with mild to moderate depression were included in the study, depression should not have a statistically significant negative influence on the outcome of the language screening.

9) Because only patients with mild to moderate motor disturbances were included in the study, motor disturbances should not have a statistically significant negative influence on the outcome of the language screening.

10) Because only patients with mild to moderate dementia were included in the study, dementia should not have a statistically significant negative influence on the outcome of the language screening.
4 Method

After addressing the seven study aims (chapter 3.9.6.1) as well as the ten study hypotheses (chapter 3.9.6.2) the procedure of this study will be described within this chapter, focusing on patients tested and the kind of test items used.

4.1 Subjects

Twenty patients with genetically diagnosed HD, participated in this study. Demographic data for each clinical subject are presented in Table 5.

Table 5 demonstrates the demographic information of 20 HD patients included in this study.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Education_Years</th>
<th>Disease_Days</th>
<th>Stage</th>
<th>CAG</th>
<th>Handedness&lt;sup&gt;90&lt;/sup&gt;</th>
</tr>
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<tbody>
<tr>
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<td>52</td>
<td>M</td>
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<td>7</td>
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<td>53</td>
<td>M</td>
<td>9</td>
<td>3970</td>
<td>3</td>
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<td>R</td>
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</tbody>
</table>

<sup>90</sup> Hand edness has been tested by using an adopted German version of the EHI (=Edinburgh Handedness Inventory) by Salmaso and Longoni (1985).
As evidenced in Table 5, HD patients tested, were ± 47.6 years old, 12 were male and 8 female. Years of education were ± 11. Estimated average 'days since disease onset' measure for all 20 HD patients was ± 1693. In terms of HD stage (rated by the TFC, Shoulson & Fahn, 1979), five patients were at stage I, eight at stage II and seven at stage III. The number of CAG repeats was ± 46. All patients tested were native German speakers, and with the exception of two, all were right-handed.

Patients studied, were recruited from the 'Chorea Huntington Outpatient Clinic' of the Psychiatric Department of the LKH Graz. The study has been approved by the ethics committee of the Medical University Graz.

Nevertheless, to be included in this study, HD patients had to meet the following selection criteria:

1) Positive testing of HD, including the determination of the number of CAG repeats, as well as the measure 'days since disease onset'.

2) Determination of HD stage, rated by the TFCS (Shoulson & Fahn, 1979), as described in chapter 1.5.

3) No previous history of a neurological disease such as stroke, progressive dementia or psychiatric disorder.

4) Only mild to moderate cognitive impairment, rated by the MMSE (Folstein, Folstein & McHugh, 1975), as described in chapter 1.5.2.

5) Only mild to moderate psychiatric impairment, rated by the HDRS (Hamilton, 1960), as described in chapter 1.5.1.

6) Only mild to moderate motor disturbance, rated by the UHDRS motor score (Huntington Study Group, 1996), as described in chapter 1.5.3.2

7) Intact ability to follow a structured test paradigm.
Table 6 shows scores of HD patients tested on the HDRS, MMSE and the UHDRS motor score.

<table>
<thead>
<tr>
<th>Case</th>
<th>HDRS</th>
<th>MMSE</th>
<th>UHDRS motor</th>
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<tbody>
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<tr>
<td>20</td>
<td>X</td>
<td>24</td>
<td>68</td>
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</table>

The control group comprised 20 age, sex and education matched healthy volunteers. Exclusion criteria for the control subjects included:

1) Previous history of a neurological disease such as stroke or progressive dementia
2) A current psychiatric disorder
3) Learning or language disability
4) Hearing impairment

In order to explore whether language abilities are related to certain cognitive, psychiatric or motor factors, HD patients completed various batteries addressing their cognitive, psychiatric and motor abilities. Testing these functions, patients were evaluated by the neuropsychological, neurological as well as the psychiatric staff of the University Clinic Graz, using the MMSE (Folstein,
Folstein & Mc Hugh, 1975), the Hamilton Depression Scale (Hamilton, 1960)\(^91\) and the UHDRS motor score (Huntington Study Group, 1996).

### 4.2 Procedure

Regarding the primary aim of this study to evaluate basal ganglia functions in language, HD patients were studied in terms of specific language abnormalities associated with their disease. A specific language screening has been constructed for this thesis that served to assess a wide range of language functions, including morphology, syntax and semantics. The screening has been divided into two sections, addressing language production and language comprehension. The procedure for the language screening was explained to each participant prior to the testing session, and written consent was obtained from each study subject (20 patients and the 20 healthy volunteers). A single qualified linguist assessed all study participants in a single session, under standard testing conditions. Data collection took place in a semiprivate atmosphere (separate room at the outpatient clinic) with the same instructions being given to each subject in each testing session. For the whole language screening an assessment session that lasted approximately one hour (split over two sessions) was scheduled. During testing, the subject's responses were noted paper based on a special checklist form (see Appendix XX). Additionally, the testing session was audio recorded (MP3 player) and later analyzed by the same linguist who conducted the assessment. To ensure the accuracy of the scoring, a second qualified linguist (main supervisor of this thesis) reviewed the scores.

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\(^91\) The 21 item version of the Hamilton Depression Scale, designed by Max Hamilton in 1960, has been used in this study to rate depression on 21 variables on a 0-4 point scale (0 = no impairment; 4 = severely affected): depressed mood (0-4), guilt (0-4), suicide (0-4), insomnia early (0-2), insomnia middle (0-2), insomnia late (0-2), work and activities (0-4), psychomotor retardation (0-4), agitation (0-4), anxiety (0-4) psychological symptoms (0-3), somatic symptoms (0-4), genital symptoms (0-4), hypochondrias (0-4), loss of weight (0-3), insight (0-2), diurnal variation of symptoms (0-4), depersonalization and derealization (0-4), paranoid symptoms (0-3) and obsessional and compulsive symptoms (0-2). By summing up the total scores of each variable, the maximal score is 66, indicating a severe depression.
4.3 Language screening

Due to a lack of appropriate standardized language batteries for German speaking adults, a specific language screening has been constructed for this study that served to assess the linguistic domains morphology, syntax and semantics, reported to be disordered in HD, as discussed in chapter 3. Participants could perform the assessment by answering questions verbatim, by pointing to pictures or word cards or by sequencing word cards in their proper order. No written tasks were included due to present motor disturbances in HD patients. Morphology, syntax and semantics were tested in production and comprehension tasks, as described below:

Production tasks included:
1) Semantic fluency
2) Article assignment
3) Preposition assignment (local, temporal, causal and modal)
4) Plural formation
5) Verbal inflection
6) Providing deverbal and denominal adjectives
7) Providing deverbal and deadjectival nouns
8) Sequencing of sentence parts; syntax I
9) Guided syntactic production; syntax II

Comprehension tasks included:
1) Picture naming and recognition of semantic categories
2) Providing synonyms
3) Providing antonyms
4) Providing two semantic concepts of polysemes
5) Providing two meanings of homophonic words
6) Providing labels for subjects, objects and actions, based on definitions
7) Understanding metaphors
8) Answering a questionnaire based on common sense and general knowledge

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92 There are only a few language batteries, standardized for German speaking adults, e.g., The Aacherner Aphasie Test (Huber et al., 1983), the Aphasia Check List by Kalbe et al. (2002) as well as LeMo, which is only single word based by De Bleser et al. (2004). All of these are aphasia based and thus wouldn't have been suitable for testing HD patients with subcortical pathology.
9) Decoding thematic roles (agent, patient, recipient); syntax III
10) Reading comprehension

Based on the differentiation stated by Mega and Alexander (1994), Jensen, Chenery and Copland (2006) as well as by Chenery, Copland and Murdoch (2002), the various tasks used in this study, differed in regards to their 'degree of freedom' and thus also in terms of their 'degree of complexity in language functioning'.

In detail, this terminology (high/low, primary/complex) origined in the work of Mega and Alexander (1994), by identifying a core profile of aphasia due a disruption in three out of the five hypothesized frontostriatal circuits, stated by Alexander, DeLong and Strick, back in 1986 (for details see chapter 1.4.1). Language disturbances mainly included executive, generative language functions, such as word fluency, sentence generation and discourse, but spared responsive language such as language comprehension, repetition and sometimes even naming. These symptoms are in accordance to the concept of dynamic aphasia, described by Luria and Tsvetkova back in (1968). Based on this evidence Chenery, Copland and Murdoch (2002) stated a distinction between 'primary language functions', such as basic cognitive-linguistic abilities, e.g., repetition and naming and 'complex language functions' referring to pure linguistic functions, as tested e.g., within sentence generation tasks, propositional language and the understanding of homophones. Chenery, Copland and Murdoch (2002) further inferred that cortical aphasia batteries (AAT and WAB) test 'primary language abilities' in contrast to pure linguistic screenings such as the TLC-E, TOWK, TWT-R, addressing 'complex language functions' (cf. Chenery, Copland & Murdoch, 2002: 467) and thus suggested HD patients to be especially impaired in 'complex language functions', in contrast to 'primary linguistic abilities'.

Moreover a distinction has been made between tasks comprising a 'high/low degree of freedom' (cf. Jensen, Chenery and Copland, 2006: 63). Consecutively these authors inferred that tasks associated with a 'high degree of freedom' such as propositional aspects of language, are more frequently affected in patients with subcortical lesions, than those involving a 'lower degree of freedom', such as responsive language, which to some degree also included other capacities than pure linguistic domains, such as

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93 As this aphasia concept will also be of importance in order to interpret linguistic disturbances of HD patients made within this study, a detailed description of this aphasic concept is given in Appendix V.
cognitive (e.g., needed in repetition) and visual aspects (e.g., needed in naming). Even though altered from the original considerations, 'primary language functions' were understood within this screening as tasks where choosing from a given number of provided options was not possible. Thus, these tasks were considered as being 'high in their degree of freedom'. Against that, tasks in which the right answer could be achieved by choosing one out of various given linguistic options, were regarded to be 'low in their degree of freedom', and hence were referred to as 'primary in language functioning'.

Language tasks provided, have been chosen in regards to HD's specific symptomatology. As reported for instance by Brandt, Folstein and Folstein (1988), Rosenberg, Soerensen and Christensen (1995), Lawrence et al. (1996), Lemiere et al. (2004) as well as Peinemann et al. (2005), HD patients have specific disturbances in executive functions, responsible for rule deduction, foresight, organization, planning and sequencing of events and tasks. Thus language tasks have been chosen where selection mechanisms were required ('low degree of freedom tasks'; e.g., verbal inflection) as well as where linguistic answers had to be planned ahead and where linguistic rule deduction and application was needed ('high degree of freedom tasks'; e.g., guided syntactic production).

The assumption, that HD patients especially experience problems performing these tasks, is further based on the evidence that patients with subcortical lesions experience specific problems with language tasks that require working executive functions (Mega & Alexander, 1994; D'Esposito & Alexander, 1995; Chenery, Copland & Murdoch, 2002; Jensen, Chenery & Copland, 2006). Aphasic like syndromes due to dysfunctions in executive abilities have been described by Luria and Tsvetkova back in 1968 within their concept of dynamic aphasia, as a subtype of transcortical motor aphasia⁹⁴. As HD is considered to be a disorder with primary subcortical pathology, as described in chapter 1.4, it seems evident that this type of language disturbance could also account for HD patients.

⁹⁴ A detailed description of the concept of dynamic aphasia is given in Appendix V.
Nevertheless, within this study the following tasks within the language production subtest of the screening, do fit the criteria of being 'primary language functions' and thus can be regarded as being 'low in their degree of freedom':

1) Article assignment  
2) Preposition assignment  
3) Verbal inflection  

Contrastingly, the following tasks within the language production subtest fit the criteria of being 'complex language functions' and thus can be regarded as being 'high in their degree of freedom':

1) Semantic fluency  
2) Plural formation  
3) Providing deverbal and denominal adjectives  
4) Providing deverbal and deadjectival nouns  
5) Sequencing of sentence parts; syntax I  
6) Guided syntactic production; syntax II  

Based on the same considerations, tasks for the language comprehension subtest have been chosen, resulting in the following distribution for tasks with a 'low degree of freedom' and being 'primary in regards to their language functioning':

1) Picture naming and recognition of semantic categories  
2) Providing synonyms  
3) Understanding metaphors  
4) Answering a questionnaire based on common sense and general knowledge  
5) Providing labels for subjects, objects and actions, based on definitions  
6) Reading comprehension  

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95 Even though this task could also be regarded to be 'high in its degree of freedom', as there are no choices given to choose from this is not a primarily linguistic task, but focuses mainly on cognition and general knowledge and thus could be regarded as a task comprising 'primary linguistic function' regarding the definition given by Chenery, Copland and Murdoch (2002). Hence this task will not be analyzed in further detail within chapter 6 and thus will also not be included within 'high degree of freedom tasks' comprising 'complex linguistic functions'.

96 Even though this task could also be regarded to be 'high in its degree of freedom', as there are no choices given to choose from this is not a primarily linguistic task, but focuses mainly on cognition and general knowledge and thus could be regarded as a task comprising 'primary linguistic function' regarding the definition given by Chenery, Copland and Murdoch (2002). Hence this task will not be analyzed in further detail within chapter 6 and thus will also not be included within 'high degree of freedom tasks' comprising 'complex linguistic functions'.
Similarly, the following tasks with a 'high degree of freedom' and being 'complex in regards to their language functioning' have been defined within the comprehension unit:

1) Providing antonyms
2) Providing two semantic concepts of polysemes
3) Providing two meanings of homophonic words
4) Decoding of thematic roles; syntax III

Within a quantitative, statistical analysis, a quantitative measure of the total number of correct responses has been used for all tasks. Except for semantic fluency, syntax I, syntax II, for providing two semantic concepts of polysemes as well as for providing two meanings of homophonic words, a total score of 2 could be achieved per task. A score of 1 was given for correct answers with help or one autocorrection. For linguistically wrong answers, for non responses or more than one autocorrection a score of 0 was given. A score of 2 was only given if performed linguistically correct, without help and without autocorrection.

In regards to this rating scale, the total score for various tasks provided was as follows:

1) 30 for article assignment
2) each 20 for local, temporal, causal and modal prepositions
3) 20 for plural formation
4) 40 for verbal inflection
5) 18 for deverbal and denominal adjectives
6) 20 for deverbal and deadjectival nouns
7) each 14 for each subtask within picture naming and recognition of semantic categories
8) 20 for providing synonyms
9) 20 for providing antonyms
10) 20 for providing labels for subjects, objects and actions, based on definitions
11) 10 for understanding metaphors
12) 30 for answering a questionnaire based on common sense and general knowledge
13) each 8 for each subpart of syntax III (a-h)
14) 20 for reading comprehension

Examples and detailed descriptions of the kind of linguistically wrong answers are given for each subtype of the test separately in the chapters stated.
Because of major differences in the methodology used, an individual rating scale has been developed for semantic fluency, syntax I, syntax II, for providing two semantic concepts for polysemes as well as for providing two meanings of homophonic words and syntax III. Details of these individual scoring methods are given below, in chapter 4.4.1 for semantic fluency, in chapter 4.4.8 for syntax I, in chapter 4.4.9 for syntax II, in chapter 4.5.4 for providing two semantic concepts of polysemes, in chapter 4.5.5 for providing two semantic concepts of homophonic words and in chapter 4.5.9 for syntax III.

Additionally, in regards to linguistic importance and interest of errors made, qualitative analysis was accomplished for tasks with a 'high degree of freedom'; for those being 'complex in their language functioning', including the following:

1) Semantic fluency
2) Plural formation
3) Providing deverbal and denominal adjectives
4) Providing deverbal and deadjectival nouns
5) Sequencing of sentence parts (= syntax I)
6) Guided syntactic production (= syntax II)
7) Providing antonyms
8) Providing two semantic concepts of polysemes
9) Providing two meanings of homophonic words
10) Decoding of thematic roles (agent, patient, recipient) (= syntax III)

Details of the qualitative analysis concerning these tasks, are given in chapter 6.
4.4 Language production

Within this section the method of each subtask of the language production subtest is explained and some examples are given. The complete checklist of the language production subtest of the language screening is given in Appendix XX.

4.4.1 Semantic fluency

Semantic fluency performance involved the naming of as many subcategory items as possible, of the five given supercategory items animals, vegetables, fruits, clothing and birds, within one minute. As a typical quantitative measure the total number of correct responses, excluding namings regarded to be errors\footnote{Within semantic fluency the following error types occurred: repetitions (e.g., 2 or even more often 'Apfel' for the semantic supercategory 'fruits'), the naming of supercategory items (e.g., 'Salat' as the supercategory item for the subcategory items 'Eisbergsalat', 'Häuplsalat', etc.), the naming of synonymic items (e.g., Tomate and Paradeisa), neologisms (e.g., 'Nazarus' as a nonexistent word for an animal) as well as the naming of non-category members (e.g., Gewürze for category vegetables).} for each semantic supercategory, within one minute was recorded.

In order to perform a qualitative analysis, semantic subcategories have been worked out on the basis of zoological and semantic categories, influenced from the criteria stated by Troyer, Moscovitch and Wincour (cf. 1997: 145) as well as by Heiss, Kalbe and Kessler (cf. 2001: 8). Again, namings regarded to be errors, such as repetitions and neologisms, have been excluded.

The total list of items given within one minute per supercategory, from all participants of this study in sum (patients and controls), excluding neologisms and repetitions, categorized in semantic subcategories, is given in Appendix VII. On the basis of this list, further qualitative analysis has been performed, evaluating the number of clusterings, switchings, as well as the number of items a cluster is made of, adopting the criteria stated by Troyer, Moscovitch and Wincour, (cf. 1997: 145), by Heiss, Kalbe and Kessler (cf. 2001: 5f), by Ho et al. (cf. 2002: 1279), by Mayr (2002), for clusterings as well as from the criteria stated by Reverberi, Capitani and Laiacona (2004) as well as by Reverberi, Laiacona and Capitani (2006) for the domain of switchings. A more detailed description of the methodology of this task is given in chapter 6.1.
4.4.2 Article assignment

Each participant was presented with 15 written sentences without article. Three direct-article choices were given in random order to select the proper form.  
*Example:* Er knöpft ___ schwarze Hemd zu. (*der, dem, das*)  
Within a quantitative analysis the total number of correct choices was recorded.  
In detail, 2 points were given per sentence if choosing the right article, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. 0 points were given if choosing the wrong article, for more than one autocorrection and non responses. Thus, a total score of 30 could be reached within this task (15 x 2 = 30).

4.4.3 Preposition assignment

This task was subdivided into four parts, according to the four German types of prepositions (local, temporal, modal and causal).  
Per test part each subject was presented with 10 written sentences, without preposition. Three preposition choices were given in random order, to select the proper form.  
*Example:* Local: Der Topf steht ___ dem Herd. (*hinter, auf, inmitten*)  
Temporal: Der Kuchen muss noch ___ dreizehn Uhr im Rohr bleiben. (*um, bis, gegen*)  
Modal: Das Kochbuch ist ___ Deutsch geschrieben. (*über, aus, auf*)  
Causal: ___ ihrer gesunden Ernährung hat sie keinerlei Beschwerden. (*Dank, Durch, Aus*)  
The number of correct choices was recorded within a quantitative analysis. In detail, 2 points were given per sentence if choosing the right preposition, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. 0 points were given if choosing the wrong preposition, for more than one autocorrection and non responses. Thus, a total score of 20 could be reached within each subtask of the preposition assignment task (10 x 2 = 20 for local, temporal, causal and modal prepositions).
4.4.4 Plural formation

Each study subject was presented with 10 German singular words plus indirect article. The subject was asked to give the proper plural of the given word.

*e.g.,: ein Hase – viele Hasen*

As a typical quantitative measure, the total number of correct choices was noted. In detail, 2 points were given per plural formation, if providing the correct plural of the word, without help and without autocorrection. Against that, 1 point was given if performing correctly but with one autocorrection and help. 0 points were given if providing linguistically incorrect plural formations of a given word (one example for all various types of linguistically incorrect plural formations will be given in chapter 6.2. The full range of all errors made, are presented in Appendix IX), for more than one autocorrection and non responses. Thus, a total score of 20 could be reached within this task (10 x 2 = 20). Furthermore, the percentage of various error types made has been considered within a qualitative analysis, as will be presented in chapter 6.2.

4.4.5 Verbal inflection

20 sentences were presented to each study subject, without verb. Three possible inflected verb forms (including abberations, chosen in the sense of being e.g., non-existing German inflected verbs e.g., schaffen – *schief, stehen – *stunden, or abberations in the sense of being existing but not appropriate verbal inflections of the given sentence) were presented in random order to select the proper form.

*e.g.,: Du__den Marathon in Bestzeit. (läuft, lauft, gelaufen)*

Within a quantitative analysis the number of correct choices was recorded. In detail, 2 points were given per sentence if choosing the right verb, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. 0 points were given if choosing the wrong verb, for more than one autocorrection and non responses. Thus, a total score of 40 could be reached for this task (20 x 2 = 40).
4.4.6 Providing deverbal and denominal adjectives

Different nouns and verbs (total amount of nine test items; 6 nouns, 3 verbs) were presented to each study subject. Subjects were asked to generate a proper German adjective out of each given word.

*e.g.:* lesen – leserlich; Buckel – buckelig

Within a quantitative analysis the number of correct derivations/conversions was recorded. In detail, 2 points were given if providing the correct derivation/conversion of the word, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. Moreover, 1 point was given for the following productions, being German participles per se:

1) derived verb_participle (lesen – verlesen)
2) derived denominal verb_participle (Mutter – bemutternd)
3) denominal verb_participle (Furcht – gefürchtet)
4) composition_participle (Furcht – furchterregend)
5) lexical intrusion_verb_participle (lachen – lächelnd)
6) derived verb_adjective (achten – verächtlich)

The problem regarding the linguistic categorization of German participles needs to be addressed in this context, in order to understand the further scoring procedure. However, there is an ongoing debate whether the two German participles (present and past) can be categorized as adjectives or not. The author of this thesis is a defender of the theory that participles can be categorized as adjectives, supporting assumptions of the following authors. Römer (2006) pointed out that the two German participles (present and past) can be categorized as adjectives, as they are also used as such. In more detail, the two German participles (present and past), as derived from verbs with derivational morphemes, such as -end/-nd for present participles (e.g., führend,

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99 Grammarians distinguish between two main types of morphological operations: inflection and derivation. In regards to inflection the differentiation can be made between declination (e.g., of nouns; Tag-Tage) and conjugation (e.g., of verbs; ich gehe-du gehst). The main difference between inflection and derivation is that derivation causes a change in the syntactic category of words, whereas inflection does not. Thus derivation can be seen as the main word formation process (by derivation the verb 'institute' can be changed to a noun by suffixation of -ion > 'institution'. From the noun institution the adjective 'institutional' can be formed by applying the suffix -al and so on). Whereas in derivation always affixation has to occur in order to change the word class, in conversion the word class is changed by the application of a so called null or zero affix (essen; verb-dass Essen; noun). For a comprehensive overview about inflection, derivation and conversion see for instance Matthews (1991: chapter 4), Spencer (1991: 8-21), Haspelmath (2002: chapter 4) or Donalies (2007: 68ff).
überzeugend) and with derivational morphemes such ge-, -t and -en for past participles (e.g., gespielt, bekannt, erfahren), are used as adjectives (eine führende Marke, ein überzeugendes Gespräch, die gespielte Höflichkeit, die bekannten Lieder, eine erfahrene Dame) and can thus be categorized as such (cf. Römer, 2006: 143). Fleischer (1983b) moreover strengthened that adjective participles can be treated like adjectives due to the fact that there are adjectives which have the same derivational morphemes as participles (ge-, -t) (e.g., geblümt, gestreift, etc.) (cf. Fleischer, 1983b: 281). Erben (cf. 2000: 105) pointed out furthermore the existence of German participles such as (bekannt, erfahren) that are 'als Adjektive geläufig' ['known as adjectives'] and thus language users do not even connect these words with their verbal basis.

A further evidence for the theory that participles can be treated as adjectives, is given from print media, science or finance. From these contexts evidence exists that adjective participles are able to form adjective compoundings such as (e.g., wärmeisolierend, schlaffördernd, altersbedingt, textbezogen etc.), derived from noun and adjective participles (cf. Fleischer, 1983b: 240; Eichinger, 2000: 134). For further information about adjective compoundings, see for instance also Pümpel-Mader, Gassner-Koch and Wellmann (1992).

In sum, even though these linguistic forms looking like a participle, they function as adjectives. As the naive speaker of German (such as participants within this study) is prototypically not a linguist, and thus may not know the distinction between participles and adjectives, participle productions functioning as adjectives, have been regarded to be correct productions of this task, within this study.

However, going back to the scoring procedure of this subtask within the screening, 0 points were given, if providing linguistically incorrect derivations/conversions for a given word (one example for all various types errors made will be given in chapter 6.3; The full range of all linguistically incorrect adjectives is presented in Appendix X), for more than one autocorrection and non responses. Thus, a total score of 18 could be reached within this task (9 x 2 = 18). Furthermore, the percentage of various error types has been considered within a qualitative analysis, as will be presented in chapter 6.3.
4.4.7 Providing deverbal and deadjectival nouns

Different adjectives and verbs (total amount of 10 test items; 3 verbs, 7 adjectives) were presented to all subjects tested. All study subjects were asked to generate a proper German noun out of each given form.

*e.g.:* heiter – die Heiterkeit; werben – die Werbung

The number of correct derivations has been recorded within a quantitative analysis. In detail, 2 points were given if providing the correct derivation/conversion of the word, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. 0 points were given if providing linguistically incorrect derivations for a given word (one example for all various types errors made will be given in chapter 6.4. The full range of all linguistically incorrect derivations/conversions, are presented in Appendix XI), for more than one autocorrection and non responses. Thus, a total score of 20 could be reached within this task (10 x 2 = 20). Furthermore, the percentage of various error types have been considered within a qualitative analysis. A detailed description of the methodology of the qualitative analysis will be given in chapter 6.4.

4.4.8 Sequencing of sentence parts (= syntax I)

Each sentence (in total 20 sentences) was divided into its sentence phrases, respectively into parts of its sentence phrases, to make the task more tricky (e.g.: /in den Ferien/ > /in/den Ferien/) and presented to the subject in random order. Subjects were asked to arrange the sentence phrases/parts of sentence phrases in a grammatical proper way, to generate a meaningful sentence.

*e.g.:* Die Kinder/in/gehen/den Ferien/ klettern./gerne/ >

/Die Kinder/ gehen/ in/ den Ferien/ gerne/ klettern./

Quantitative analysis was based on the number of correct sequencings, for all 20 sentences together. A maximum score of 110 could be reached within this task, by summarizing all sentence phrases/parts of sentence phrases for all 20 test sentences.

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100 A (sentence) phrase is a term used in grammatical analysis to refer to a structure containing more than one word, and lacking the subject-predicate structure to be called a clause. Several types of sentence phrases can be distinguished such as adverbal phrases (e.g., yesterday morning,...) or adjectival phrases (e.g., the house, old and derelict,...) (cf. Crystal, 1980: 270).
Thus for instance a number of 6 sequencing errors within syntax I resulted in a total score of 104 (110 – 6 = 104) (one example for all various types errors made will be given in chapter 6.5. The full range of all sequencing errors are given in Appendix XII). Concerning a qualitative analysis the percentage of sequencing errors in respect to the total number of sentence phrases/parts of sentence phrases per sentence, has been recorded. Details will be given in chapter 6.5.

4.4.9 Guided syntactic production (= syntax II)

Three words (each word on a separate card) (in total 30 words for 10 sentences) were given to all study subjects in random order. Study subjects were asked to provide a meaningful sentence, using at least the three given words, regardless in which sequence. e.g., /WEIL/ /ZERBROCHEN/ /JUNGE/ > Weil der Junge den Krug zerbrochen hat, schimpft die Mutter.

Quantitative analysis was based on the number of correct sentences produced. In detail, a score of 10 could be reached per sentence (max score of 10 per sentence x 10 sentences of the task = total score of 100 for syntax II). A score of 2 was given for each correctly included word (2 x 3 = 6), additionally in regards to completion and correctness\(^{101}\) of the sentence provided, 2 more points were given each (2 + 2 = 4), which resulted in a maximum score of 10 per sentence. A score of 1 was given for one autocorrection or help. A score of 0 was given for more than one autocorrection, for non responses, for each word missing out of the three given items, for morphological changes of the words provided, for semantic incorrect sentences as well as for incomplete sentences (one example for all various types errors made will be given in chapter 6.6. The full range of all linguistic errors, are presented in Appendix XIV). Regarding qualitative analysis the percentage of various error types, have been considered. Details will be given in chapter 6.6.

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\(^{101}\)Correctness is defined in this context as the degree to which a sentence is linguistically correct; that there are no semantic, syntactic or morphological errors. Within a sentence various types of errors have been classified separately in terms of the defective linguistic domain (Details of the various error types displayed in this subtest of the screening, are given in chapter 6.6 as well as the full list is presented in Appendix XIV).
4.5 Language comprehension

In this section the method of each subtask of the language production subtest is explained and some examples given. The complete checklist of the language production subtest of the language screening is given in Appendix XX.

4.5.1 Picture naming and recognition of semantic categories

Ten pictures of the Snodgrass and Vanderwart Picture Inventory (1980) were presented to every study subject. Pictures were selected according to visual complexity. The total set of 10 pictures comprised 5 high visual complex (rating over 4) and 5 low visual complex (rating below 1.9) pictures. The ratings concerning the visual complexity of the Snodgrass and Vanderwart Picture Inventory were adapted for German, based on the scores evaluated by Genzel, Kerkohoff and Scheffter (1995), as exemplified below.

**Visual high complex stimuli:**
- Adler: 4.30
- Ananas: 4.13
- Herd: 4.21
- Heuschrecke: 4.21
- Ziehharmonika: 4.39

**Visual low complex stimuli:**
- Banane: 1.37
- Briefumschlag: 1.66
- Herz: 1.10
- Mund: 1.54
- Nadel: 1.79

After picture naming, study subjects were confronted with a questionnaire consisting of 6 questions per picture, according to the semantic features of the given picture. The questionnaires have been adopted from the Semantic Verbal Questionnaire by Laiacona, Barbarotto and Capitani (1993), as shown below.

1) General superior category:
   - z.B. Is it a vegetable, an animal or a fruit?
2) Subordinate within the same category:
   - z.B. Is it a skin fruit, a berry or a stone fruit?
3) Subordinate to perceptual attributes:
z.B. Does it have a round, curved or oval form?

4) Subordinate: comparison of perceptual attributes:
   z.B. Does it tast more bitter than a grapefruit?

5) Subordinate: associative functional attributes:
   z.B. Are you eating it with or without paring or are you pressing it out?

6) Subordinate: associative contextual attributes:
   z.B. Is it growing only in warm climate, in cold or in both?

e.g.,: Banane

1) Ist es ein Gemüse, ein Tier oder ein Obst?

2) Ist es eine Schalenfrucht, eine Beere oder ein Steinobst?

3) Hat es eine runde, eine gebogene oder eine kegelige Form?

4) Schmeckt es bitterer als eine Grapefruit? nein

5) Isst man es ohne Schale, mit Schale oder presst man es aus?

6) Wächst es bei kaltem Klima, warmem Klima oder gedeiht es bei beidem?

Quantitative analysis was based on correct picture naming, as well as on the total number of questions concerning the pictures, answered properly. Thus a total score of 14 could be reached per picture (a score of 2 for naming the picture correctly and a score of 2 each, for answering all of the 6 questions per picture properly (2 x 6 = 12 + 2 points for picture naming = 14). 1 point was given if performing correctly but with autocorrection and help. 0 points were given if naming incorrectly, if incorrectly answering a question, for more than one autocorrection and for non responses.

4.5.2 Providing synonyms

10 words (4 nouns, 4 verbs, 1 adjective and 1 adverb) were presented to each study subject. Each subject was given three options (each word on a separate card) to select the synonym of the given word. Abberations were chosen in the sense of being an antonym of the given word (e.g., hinab – hinauf) or sharing some semantic markers with the given item (e.g., Stuhl – Hocker, both are used to sit on) and being so called total abberations; being semantically farer away from the given item than the semantic abberation (e.g., Erdapfel – Adamsapfel). The subject was asked to point to the word
that has the same meaning as the given word.

\textit{e.g.,: hinab} \underline{abwärts}, \textit{hinauf}, \textit{hinter}

Within a quantitative analysis the number of correct synonym – choices was recorded. In detail, 2 points were given if choosing the correct synonym of the given word, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. 0 points were given if choosing the antonym, the semantic or total aberration, for more than one autocorrection and non responses. Thus a total score of 20 could be reached within this task (10 x 2 = 20).

\section*{4.5.3 \quad Providing antonyms}

10 words (3 nouns, 4 verbs, 3 adjectives) were presented to each study subject. Subjects were asked to give a word which is opposite in meaning to the given word.

\textit{e.g.,: gut} \textit{–} \textit{schlecht}

Within a quantitative analysis the number of correct antonym namings was recorded. In detail, 2 points were given if providing the correct antonym of the given word, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. 0 points were given if providing a linguistically incorrect answer (one example for all various types errors made, will be given in chapter 6.7. The full range of all linguistically incorrect antonyms, is presented in Appendix XV), for more than one autocorrection as well as for non responses. Thus, a total score of 20 could be reached within this task (10 x 2 = 20). Regarding qualitative analysis the percentage of various error types was calculated, as will be presented in chapter 6.7.

\section*{4.5.4 \quad Providing two semantic concepts of polysemes}

All study subjects were confronted visually with 9 polysemic words in total and were asked to give at least two semantic concepts of the given word.

\textit{e.g.,: Bank} \textit{– die Bank auf der man sitzt}

\hspace{1cm} \textit{– die Bank, in die man geht, um Geld zu holen}
Thus quantitative analysis was based on the provision of correct semantic concepts per given item. In detail, 4 points were given if providing two semantic concepts for the given polyseme, without help and without autocorrection. Against that, 2 points were given if providing only one out of two semantic concepts without help and without autocorrection. 1 point was given each, if naming both semantic concepts correctly, but with autocorrection and help. 0 points were given if providing linguistically inappropriate answers (all types of linguistically inappropriate answers are given in Appendix XVI), for more than one autocorrection and non responses. Thus, a total score of 36 could be reached within this task (9 x 4 = 36). The percentage of various error types are discussed in the context of a qualitative analysis, as will be given in chapter 6.8.

### 4.5.5 Providing two meanings of homophonic words

Each study subject was confronted auditively with a total of 10 homophonic words and was asked to give at least two different meanings of the given word.

*e.g.,: /sage/ (confronted auditively) – die Sage, die man erzählt*

– *ich sage im Sinne von sprechen*

Thus, quantitative analysis was based on the number of correct meanings provided. In detail, 4 points were given if providing two correct meanings of the given homophone, without help and without autocorrection. Against that, 2 points were given if providing only one out of two meanings without help and without autocorrection. 1 point was given each, if giving both meanings, but with autocorrection and help. 0 points were given if providing linguistically inappropriate answers (all types of linguistically inappropriate answers are given in Appendix XVII), for more than one autocorrection and non responses. Thus, a total score of 40 could be reached within this task (10 x 4 = 40). The percentage of various error types will be discussed in detail in chapter 6.9.
4.5.6 Providing labels for subjects, objects and actions, based on definitions

Objects (7), subjects (2) or an action (1) have been described to each study subject (10 tasks). Each subject was asked to give the appropriate label for the described subjects, objects and the action.

*Example:* *Man trocknet sich nach dem Waschen damit ab.* **Handtuch**

Based on the total number of correct labels provided, quantitative analysis could be revealed. In detail, 2 points were given if naming the correct subject/object/action, as described, without help and without autocorrection. Against that, 1 point was given if providing labels correctly, but with autocorrection and help. 0 points were given if providing a semantically inappropriate answer, for more than one autocorrection as well as for non responses. Thus, a total score of 20 could be reached within this task (10 x 2 = 20).

4.5.7 Understanding metaphors

5 German metaphors were presented to every study subject. All subjects were given three options to select the proper meaning of the given metaphor.

*Example:* **Wie Hund und Katz**

a) *Zwei Menschen, die sich nicht vertragen.*

b) *Bestimmtes Verhalten, dass Hunde zeigen, wenn sie auf Katzen treffen.*

c) *Bezeichnet das Phänomen, dass Hunde- bzw. Katzenbesitzer ihrem Haustier immer ähnlicher werden.*

Based on the total number of correct choices, quantitative analysis was performed. In detail, 2 points were given if choosing the right explanation for the metaphor, without help and without autocorrection. Against that, 1 point was given if choosing the right explanation, but with autocorrection and help. 0 points were given if choosing the wrong explanation, for more than one autocorrection and for non responses. Thus, a total score of 10 could be reached within this task (5 x 2 = 10).
4.5.8  Answering a questionnaire based on common sense and general knowledge

Every study subject was asked to answer (a total number of 15) open and closed questions, based on common sense and general knowledge.

*e.g.*:  
*Gibt es fünf Jahreszeiten?*  *Ja/Nein*

*Wie heißt unser Bundespräsident?*  *Heinz Fischer*

Quantitative analysis could be conducted on the basis of the total number of correct answers. In detail, 2 points were given if providing the correct answer, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help, as well as if giving an answer that is in semantic relationship to the right answer. 0 points were given if providing a semantically incorrect answer, for more than one autocorrection and for non responses. Thus, a total score of 20 could be reached within this task (15 x 2 = 30).

4.5.9  Decoding of thematic roles (= syntax III)

A sentence picture matching task (4 pictures per sentence) consisting of 32 sentences, separated for 8 different syntactic complexity levels ranging from a-h, regarding the decoding of thematic roles, such as agent, patient and recipient, was constructed, adopted from Gadler (1988). Each syntactic complexity level comprised 4 sentences (8 x 4 = 32 test sentences in total).

In detail, syntactic complexity ranged from:

(a) active sentences with monovalent verbs

(a. Das Auto fährt.)

(b) active sentences with obligatory bivalent verbs

(b. Der Hund beißt die Katze.)

(c) passive constructions with obligatory bivalent verbs

(c. Die Katze wird vom Hund gebissen.)

(d) active sentences with obligatory trivalent verbs

(d. Der Bub gibt dem Mädchen die Katze.)

(e) passive constructions with obligatory trivalent verbs

(e. Die Katze wird dem Mädchen vom Buben gegeben.)
(f) coordinated sentences

(f. Die Katze beißt den Hund und kratzt den Affen.)

(g) embedded relative clauses

(g. Die Katze, die den Hund beißt, kratzt den Affen.)

(h) attached relative clauses

(h. Die Katze beißt den Hund, der den Affen kratzt.)

Each study subject was asked to read the given sentence and to point to the appropriate picture (In this example picture 4).

**e.g.:** Das Krokodil, das den Affen beißt, küsst den Fuchs.

Der Affe, der das Krokodil beißt, küsst den Fuchs.

Das Krokodil, das den Fuchs beißt, küsst den Affen.

Der Fuchs, der den Affen beißt, küsst das Krokodil.

Within a quantitative analysis the number of correct sentence picture matchings was recorded. In detail, 2 points were given, if choosing the correct picture, representing the correct thematic roles, out of four the possible choices, without help and without autocorrection. Against that, 1 point was given if choosing correctly but with autocorrection and help. 0 points were given if choosing an inappropriate picture (permutation of thematic roles), for more than one autocorrection and for non responses. (one example for all various types errors made, will be given in chapter 6.10. The full range of all incorrect sentence picture matchings is presented in Appendix XVIII). Thus, a total score of 8 could be reached for each subpart (a-h) of syntax III (2 x 4 = 8).

Additionally, in regards to a qualitative analysis, the percentage of various thematic role permutations will be given in chapter 6.10.
4.5.10  Reading comprehension

All study subjects were asked to read two short texts (printnews and instruction manual) and to answer a specific questionnaire concerning each text (5 questions per text) on the basis of yes/no answers. Within a quantitative analysis the total number of correct answers per text was recorded. In detail, 2 points were given if providing the correct answer, without help and without autocorrection. Against that, 1 point was given if performing correctly but with autocorrection and help. 0 points were given if providing the incorrect answer, for more than one autocorrection as well as for non responses. Thus, a total score of 10 could be reached within this task (5 x 2 = 10).
5 Quantitative analysis – statistics

Statistical analysis of the raw data has been performed using SPSS Version 15.0. The statistical level of significance has been set on p < 0.05.

In detail, for each subtest of the language screening (language production versus language comprehension) for HD patients versus healthy controls, multivariate analysis of variance (MANOVA) and correlational analysis was performed.

For simplifying the process of statistical analysis, some subtasks of the two subtests of the language screening have been merged in respect to their linguistic content.

1) Within semantic fluency the five supercategory items (animals, vegetables, fruits, clothing and birds) have been pooled to semantic fluency in general.
2) The four types of German prepositions (local, temporal, modal and causal) have been merged to preposition assignment.
3) Plural formation and verbal inflection have been summarised to inflection.
4) Providing deverbal and denominal adjectives as well as providing deverbal and deadjectival nouns have been summarised to derivation/conversion.
5) Syntax I and syntax II have been simplified to syntax_production.

This approach has also been held for the various subtasks of the language comprehension subtest.

1) The ten subtasks within picture naming and recognition of semantic categories, have also been merged to single word understanding_visual.
2) Providing synonyms, antonyms, two semantic concepts of polysemes and two meanings of homophonic words, have been summed up to single word understanding_general.
3) In the same manner, the eight subtasks of syntax III have been pooled to syntax_comprehension.
4) The two subtasks of reading comprehension have been summarised to reading comprehension in general.
Multivariate analysis of variance and correlational analysis have been performed to prove the following 10 hypotheses, as previously presented in chapter 3.9.6.2:

1) There is a statistically significant difference between patients and controls in regards to their performance in the language screening (language production and language comprehension).

2) Within subtasks of the language production subtest, there is a statistically significant difference between the performance of controls and patients.

3) Within subtasks of the language comprehension subtest, there is a statistically significant difference between the performance of controls and patients.

4) Within patients, there is a statistically significant effect of the HD stage on the performance in the language screening (language production and language comprehension).

5) Within patients, there is a statistically significant effect of the HD stage on the various subtasks of the language production subtest.

6) Within patients, there is a statistically significant effect of the HD stage on the various subtasks of the language comprehension subtest.

7) There is a statistically significant negative correlation between the disease duration score 'days since disease onset' (disease progression measure) under control of the 'number of CAG repeats' (disease onset measure) and the language screening (language production and language comprehension) in HD patients. This theory is based on the assumption that the more advanced HD is, the more language disturbances are supposed to occur as well.

8) Because only patients with mild to moderate depression were included in the study, depression should not have a statistically significant negative influence on the outcome of the language screening.

9) Because only patients with mild to moderate motor disturbances were included in the study, motor disturbances should not have a statistically significant negative influence on the outcome of the language screening.

10) Because only patients with no to mild dementia were included in the study, dementia should not have a statistically significant negative influence on the outcome of the language screening.
5.1 Examination of conditions

Within statistical analysis the following definition of variables has been used:

1) DV1: language production
2) DV2: language comprehension
3) iDV1: group (patients/control group)
4) iDV2: HD stage (I, II, III)

In regards to significance any p value ≤ 0.01 is regarded to be high significant, in contrast any p value ≤ 0.05 is regarded to be significant. And thus any p value ≥ 0.05 is noted to be not statistically significant.

In order to justify the statistical analysis as given below, conditions of the raw data have been examined in advance. In detail, the correlation of the language production and the language comprehension subtest have been examined, using Pearson correlation. Also normal distribution and homogeneity of variance have been examined, by using the Kolmogorov-Smirnov test of goodness of fit to justify the application of an analysis of variance.

Pearson shows a strong correlation between language production and language comprehension \((r = 0.892; p < 0.01)\) therefore a MANOVA with language production and language comprehension as the two DV's, is justified.

Table 7 shows the Pearson correlation between language production and language comprehension

<table>
<thead>
<tr>
<th>language production</th>
<th>language comprehension</th>
<th>Pearson correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>(r)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.892</td>
</tr>
</tbody>
</table>

Even though normal distribution and homogeneity of variance could not be approved for all raw data of this thesis (for details of the Kolmogorov-Smirnov test of goodness of fit, which accounts for a normal distribution and of the Levene-Test, which accounts for homogeneity of variance, see AppendixVI) MANOVA can still be calculated.
This is because the F-test of an analysis of variance is known to be robust against departures from both normality and homogeneity of variance, particularly when the number of observations in each group is equal (cf. Stevens, 2007: 57). This has also been stated in a similar way by Bortz (1999) assuming that the consequence of departures from both normality and homogeneity of variance „[...] will not [...] lead to greatly distorted P-values or, consecutively, inappropriate conclusions [of the results]“ (Everitt, 1996: 55f). In a similar way Stevens (1999) suggested that

„[...] eine Normalverteilung durch die Wirkung des Zentralen Grenzwerttheorems [ist] auf alle Fälle gewährleistet (völlig egal wie die Verteilung der Rohwerte ist!). Bei geringeren Abweichungen der Rohwertverteilung von der Normalität reichen bereits 10 bis 20 Meßwerte pro Zelle für eine ausreichende Wirkung des Zentralen Grenzwerttheorems; bei sehr starken Abweichungen gilt das ab etwa 50 Meßwerten pro Zelle“ (Stevens, 1999: 75f).
5.2 Hypothesis 1

There is a statistically significant difference between patients and controls in regards to their performance in the language screening (language production and language comprehension).

MANOVA proves Hypothesis 1, by showing a statistically significant effect of the iDV1 group on the language screening (language production and language comprehension) ($F_{2,37} = 11.915; p < 0.01$)\textsuperscript{102}.

Table 8 shows the group effect on the language screening in general

<table>
<thead>
<tr>
<th>language screening</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th>Pillai</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>23.286</td>
<td>3.817</td>
<td>26.613</td>
<td>0.647</td>
<td>0.392</td>
</tr>
</tbody>
</table>

Univariate analysis of variance shows the statistically significant effect of the iDV1 group on language production and language comprehension separately. In detail, results indicate a statistically significant effect of the iDV1 group on language production ($F_{1,38} = 12.698; p < 0.01$) as well as on language comprehension ($F_{1,38} = 23.282; p < 0.01$).

Table 9 shows the group effect on language production and language comprehension separately

<table>
<thead>
<tr>
<th>language screening</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th></th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
<td></td>
</tr>
<tr>
<td>language production</td>
<td>31.381</td>
<td>5.936</td>
<td>36.184</td>
<td>1.049</td>
<td>$F_{1,38} = 12.698$</td>
</tr>
<tr>
<td>language comprehension</td>
<td>15.191</td>
<td>1.697</td>
<td>17.041</td>
<td>0.245</td>
<td>$F_{1,38} = 23.282$</td>
</tr>
</tbody>
</table>

\textsuperscript{102}All results of multivariate analysis given in this thesis, always refer to Pillai.
5.3 Hypothesis 2

Within subtasks of the language production subtest, there is a statistically significant difference between the performance of controls and patients.

MANOVA proves Hypothesis 2, by evidencing a statistically significant effect of the iDV1 group on the subtasks of the language production subtest in total ($F_{6,33} = 6.315; p < 0.01$).

Table 10 shows the group effect on the language production subtest in general

<table>
<thead>
<tr>
<th>language production</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th>Pillai</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
<td></td>
</tr>
<tr>
<td>HD patients (n = 20)</td>
<td>31.381</td>
<td>5.936</td>
<td>36.184</td>
<td>1.049</td>
<td>0.534</td>
</tr>
</tbody>
</table>

Univariate analysis of variance shows the statistically significant effect of the iDV1 group on the subtasks of the DV language production separately. In detail, results indicate a statistically significant effect of the iDV1 group on semantic fluency ($F_{1,38} = 22.703; p < 0.01$), article assignment ($F_{1,38} = 4.248; p < 0.05$), preposition assignment ($F_{1,38} = 12.145; p < 0.01$), inflection ($F_{1,38} = 13.830; p < 0.01$), derivation/conversion ($F_{1,38} = 21.524; p < 0.01$) as well as on syntax_production ($F_{1,38} = 4.866; p < 0.05$).

Table 11 shows the group effect on the various language production subtasks separately

<table>
<thead>
<tr>
<th>language production</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>semantic fluency</td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>article assignment</td>
<td>10.850</td>
<td>3.662</td>
<td>16.330</td>
<td>3.612</td>
</tr>
<tr>
<td>preposition assignment</td>
<td>27.450</td>
<td>4.261</td>
<td>29.500</td>
<td>1.277</td>
</tr>
<tr>
<td>inflection</td>
<td>18.263</td>
<td>1.795</td>
<td>19.725</td>
<td>0.550</td>
</tr>
<tr>
<td>derivation/conversion</td>
<td>27.525</td>
<td>2.392</td>
<td>29.625</td>
<td>0.809</td>
</tr>
<tr>
<td>syntax_production</td>
<td>11.275</td>
<td>5.796</td>
<td>17.450</td>
<td>1.356</td>
</tr>
<tr>
<td></td>
<td>92.925</td>
<td>23.400</td>
<td>104.475</td>
<td>0.850</td>
</tr>
</tbody>
</table>
5.4 Hypothesis 3

Within subtasks of the language comprehension subtest, there is a statistically significant difference between the performance of controls and patients.

MANOVA proves Hypothesis 3, by showing a statistically significant effect of the iDV1 group on the subtasks of the language comprehension subtest ($F_{7,32} = 6.963; p < 0.01$).

Table 12 shows the group effect on the language comprehension subtest in general.

<table>
<thead>
<tr>
<th>language comprehension</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th>Pillai</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>15.191</td>
<td>1.697</td>
<td>17.041</td>
<td>0.245</td>
<td>0.604</td>
</tr>
</tbody>
</table>

Univariate analysis of variance shows the statistically significant effect of the iDV1 group on the subtasks of the DV language comprehension separately. In detail, results indicate a statistically significant effect of the iDV1 group on single word understanding_visual ($F_{1,38} = 7.390; p < 0.05$), single word understanding_general ($F_{1,38} = 15.576; p < 0.01$), definitions ($F_{1,38} = 8.941; p < 0.01$), metaphors ($F_{1,38} = 10.498; p < 0.01$), questionnaires ($F_{1,38} = 11.472; p < 0.01$), syntax_comprehension ($F_{1,38} = 33.157; p < 0.01$) as well as on reading comprehension ($F_{1,38} = 17.857; p < 0.01$).
Table 13 shows the group effect on the various language comprehension subtasks separately.

<table>
<thead>
<tr>
<th>language comprehension</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>single word underst._visual</td>
<td>M 13.720</td>
<td>SD 0.399</td>
<td>M 13.970</td>
<td>SD 0.098</td>
</tr>
<tr>
<td>single word underst._general</td>
<td>M 22.838</td>
<td>SD 6.348</td>
<td>M 28.463</td>
<td>SD 0.575</td>
</tr>
<tr>
<td>definitions</td>
<td>M 19.200</td>
<td>SD 1.196</td>
<td>M 20.000</td>
<td>SD 0.000</td>
</tr>
<tr>
<td>metaphors</td>
<td>M 7.800</td>
<td>SD 3.037</td>
<td>M 10.000</td>
<td>SD 0.000</td>
</tr>
<tr>
<td>questionnaires</td>
<td>M 29.200</td>
<td>SD 1.056</td>
<td>M 30.000</td>
<td>SD 0.000</td>
</tr>
<tr>
<td>syntax_comprehension</td>
<td>M 6.231</td>
<td>SD 1.202</td>
<td>M 7.806</td>
<td>SD 0.228</td>
</tr>
<tr>
<td>reading comprehension</td>
<td>M 7.350</td>
<td>SD 1.348</td>
<td>M 9.050</td>
<td>SD 1.191</td>
</tr>
</tbody>
</table>

5.5 Hypothesis 4

Within patients, there is a statistically significant effect of the HD stage on the performance in the language screening (language production and language comprehension).

MANOVA could not show a statistically significant effect of the iDV2 HD stage on the language screening in general (F₄,₃₄ = 2.222; p > 0.05) and thus Hypothesis 4 couldn't be confirmed.

Table 14 shows the effect stage on the language screening.

<table>
<thead>
<tr>
<th>language screening</th>
<th>HD patients (n = 20)</th>
<th>Pillai</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M 23.286</td>
<td>0.414</td>
<td>F₄,₃₄ = 2.222</td>
<td>0.087</td>
</tr>
</tbody>
</table>
5.6 Hypotheses 5 and 6

Hypothesis 5: Within patients, there is a statistically significant effect of the HD stage on the various subtasks of the language production subtest.

Hypothesis 6: Within patients, there is a statistically significant effect of the HD stage on the various subtasks of the language comprehension subtest.

Because MANOVA could not show a statistically significant effect of the iDV2 HD stage on the language screening in general ($F_{4,34} = 2.222; p > 0.05$), as evidenced in chapter 5.5, the effect of the iDV2 HD stage on both DV's language production and language comprehension, will not be displayed herein.

5.7 Hypothesis 7

There is a statistically significant negative correlation between 'days since disease onset' under control of the 'number of CAG repeats' and the language screening (language production and language comprehension), in HD patients (n = 20). Thus the higher the score 'days since disease onset' under control of the 'number of CAG repeats' is, the more advanced HD is and thus also the more language disturbances are supposed to occur.

By performing correlational analysis, Hypothesis 7 could be confirmed, showing a still statistically significant negative correlation between 'days since disease onset' under control of the 'number of CAG repeats' and language production ($r^{103} = - 0.468; p < 0.05$) in HD patients (n = 20). Moreover there could also be detected a still statistically significant negative correlation between 'days since disease onset' under control of the 'number of CAG repeats' and language comprehension ($r = - 0.464; p < 0.05$) in HD patients (n = 20). Thus it can be concluded that the further away the onset of the disease is (the more advanced HD is) the worse language performance gets.

$103$The Spearman-Rho correlational coefficient ($r$) for non normal distributed variables has been used in the context of this study. ($r$) measures the strength of a supposed linear association between two variables and can vary between -1 to + 1. Thus ($r$) can account for a negative and a positive correlation between two variables.
Table 15 shows the effect 'days since disease onset' under control of the 'number of CAG repeats' on language production and language comprehension separately.

<table>
<thead>
<tr>
<th>language screening</th>
<th>'days disease onset' under control of the 'number of CAG repeats'</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>r</strong></td>
</tr>
<tr>
<td>language production</td>
<td>r = -0.468</td>
</tr>
<tr>
<td>language comprehension</td>
<td>r = -0.464</td>
</tr>
</tbody>
</table>

5.8 Hypothesis 8

Because only patients with mild to moderate depression (HDRS) were included in the study, depression should not have a statistically significant negative influence on the outcome of the language screening in HD patients (n = 20).

Hypothesis 8 could be proved by showing that there is no statistically significant negative correlation between the HDRS and the language production subtest (r = 0.215; p > 0.05) in HD patients (n = 20) as well as between the HDRS and the language comprehension subtest (r = 0.172; p > 0.05) in HD patients (n = 20).\(^{104}\)

Table 16 shows the effect of the HDRS on language production and language comprehension separately.

<table>
<thead>
<tr>
<th>language screening</th>
<th>HDRS (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>r</strong></td>
</tr>
<tr>
<td>language production</td>
<td>r = 0.215</td>
</tr>
<tr>
<td>language comprehension</td>
<td>r = 0.172</td>
</tr>
</tbody>
</table>

\(^{104}\)For testing the null hypothesis that depression has no influence on the outcome measures of the language screening, the statistical level of significance might be raised from \(p \leq 0.05\) to \(p \leq 0.2\), consecutively a certain relevance of this control factor cannot be ignored. However do notice that there is no evidence of a statistical significant correlation between these variables.
5.9 Hypothesis 9

Because only patients with mild to moderate motor disturbances (UHDRS_motor score) were included in the study, motor disturbances should not have a statistically significant negative influence on the outcome of the language screening in HD patients (n = 20).

Even though patients have been only mild to moderately severe motorically affected (UHDRS motor score from 1-68 out of 124) there could be shown a statistically significant correlation between language performance (including language production and language comprehension) and the UHDRS motor score. Thus Hypothesis 9 could not be approved.

In detail, a statistically significant negative correlation between the UHDRS motor score and language production \( (r = -0.496; p < 0.05) \) in HD patients \( (n = 20) \) as well as a statistically significant negative correlation between the UHDRS motor score and language comprehension \( (r = -0.555; p < 0.05) \) in HD patients \( (n = 20) \), could be reported.

Table 17 shows the effect of the UHDRS motor score on language production and language comprehension separately.

<table>
<thead>
<tr>
<th>language screening</th>
<th>UHDRS motor score (n = 20)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( r )</td>
<td>( p )</td>
<td></td>
</tr>
<tr>
<td>language production</td>
<td>( r = -0.496 )</td>
<td>0.026</td>
<td></td>
</tr>
<tr>
<td>language comprehension</td>
<td>( r = -0.555 )</td>
<td>0.011</td>
<td></td>
</tr>
</tbody>
</table>
5.10 Hypothesis 10

Because only patients with mild to moderate dementia (MMSE) were included in the study, dementia should not have a statistically significant negative influence on the outcome of the language screening in HD patients (n = 20).

Hypothesis 10 could be proved by evidencing no statistically significant negative correlation between the MMSE and language production \( r = -0.319; p > 0.05 \) in HD patients \( (n = 20) \). Neither could there be detected a statistically significant negative correlation between the MMSE and language comprehension \( r = -0.357; p > 0.05 \) in HD patients \( (n = 20) \).

Table 18 shows the effect of the MMSE on language production and language comprehension separately.

<table>
<thead>
<tr>
<th>language screening</th>
<th>MMSE (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( r )</td>
</tr>
<tr>
<td>language production</td>
<td>-0.319</td>
</tr>
<tr>
<td>language comprehension</td>
<td>-0.357</td>
</tr>
</tbody>
</table>

\(^{105}\)For testing the null hypothesis that motor disturbances have no influence on the outcome measures of the language screening, the statistical level of significance might be raised from \( p \leq 0.05 \) to \( p \leq 0.2 \), consecutively a certain relevance of this control factor cannot be ignored. However do notice that there is no evidence of a statistical significant correlation between these variables.
6 Qualitative analysis regarding errors made

Additionally, in regards to linguistic importance and interest of errors made, qualitative analysis has been accomplished for tasks with a 'high degree of freedom' and being 'complex in regards to linguistic functioning'. As already described in chapter 4.3, in these tasks, no choosing from a given number of provided options was possible. In order to perform correctly fully intact primary linguistic functions, such as retrieval of morphological as well as syntactic rules and semantic retrieval strategies, were needed. In detail, qualitative analysis has been made for the following tasks, as already presented in chapter 4.3:

1) Semantic fluency
2) Plural formation
3) Providing deverbal and denominal adjectives
4) Providing deverbal and deadjectival nouns
5) Sequencing of sentence parts (= syntax I)
6) Guided syntactic production (= syntax II)
7) Providing antonyms
8) Providing two semantic concepts of polysemes
9) Providing two meanings of homophonic words
10) Providing labels for subjects, objects and actions, based on definitions
11) Decoding of thematic roles (agent, patient and recipient) (= syntax III)

Details of the methodology and results of the qualitative analysis concerning these tasks, are given below.
6.1 Semantic fluency

Semantic fluency involved, as already mentioned in chapter 3.5, the naming of as many subcategory items as possible, of the five given supercategory items (animals, vegetables, fruits, clothing and birds), within one minute.

As a typical quantitative measure the total number of correct words (excluding namings regarded to be errors such as repetitions, e.g., 2 or even more often 'Apfel' for the semantic category 'fruits', neologisms, e.g., 'Nazarus' as a nonexisting word for an animal, synonymic items, e.g., Tomate and Paradeisa, supercategory items, e.g., 'Salat' as the supercategory item for the subcategory items 'Eisbergsalat', 'Häupsalat') and non-category members (e.g., 'Weintrauben' as a member of the category 'fruits' for the category 'vegetables) for each semantic supercategory within one minute, has been recorded.

Based on all namings of patients and controls together for animals, vegetables, fruits, clothing and birds, excluding repetitions and neologisms, semantic subcategory boundaries were endeavoured to be worked out on the basis of zoological and semantic categories. On the basis of that list, as given in Appendix VII, qualitative analysis has been performed, evaluating clusterings, switchings as well as the number of items a cluster is made of, influenced by but adopted from Troyer, Moscovitch and Wincour (cf. 1997: 145), Tröster et al., (1998), Troyer et al. (1998), Rich et al. (1999), Heiss, Kalbe and Kessler (cf. 2001: 5f) as well as by Ho et al. (cf. 2002: 1279) for clusterings as well as from Reverberi, Laiacona and Capitani (2006: 470f) for the domain of switchings.

But as already briefly mentioned in chapter 3.5 as well as in chapter 4.4.1, criticism is arising concerning this methodology regarding the evaluation of clusterings and switchings. The main problem is that there is no standardized evaluation criterion in terms of the evaluation of clusterings and thus also of switchings.

In more detail, the standard methodology used by Troyer, Moscovitch and Wincour (1997), concerning the evaluation of clusterings and switchings has started to be questioned, for instance by Mayr (2002), Reverberi, Capitani and Laiacona (2004) as well as by Reverberi, Laiacona and Capitani (2006). Whereas Mayr (2002) criticized the numerical implications of the proposal by Troyer, Moscovitch and Wincour (1997), referring to the absolute number of transitions between clusters as switchings (cf. 1997:...
Reverberi, Capitani and Laiacona (2004) strengthened a supposedly wrong or too loose definition of subcategories and consequently also that of switchings. Because of a lack of consideration of these factors in the study by Troyer, Moscovitch and Wincour (1997) and consecutively the use of this study as reference material for further research on semantic fluency by Tröster et al. (1998), Troyer et al. (1998), Rich et al. (1999), Heiss, Kalbe and Kessler (2001) as well as by Ho et al. (2002) caution is warranted in the interpretation of the results of these studies. In detail, following the switching methodology by Troyer, Moscovitch and Wincour (1997), study results show that frontal as well as HD patients produce a lower number of switchings between subcategories (Tröster et al. 1998; Troyer et al., 1998; Rich et al., 1999; Ho et al., 2002). This reduced number of switchings is in contrast to more recent research, showing the opposite. For instance Reverberi, Laiacona and Capitani, (cf. 2006: 471) pointed out that there is an increase in the number of relative switchings106 for left frontal patients, in contrast to controls. Further the disorganised search through the semantic memory in frontal lobe as well as in HD patients, as mentioned previously in chapter 3.5.3, is supposed to lead to the production of a disorganised sequence of words, such as ‘apple – orange – cherry – blackberry – pear – tangerine – banana’ etc. due to an increase in switchings between clusters (cf. Reverberi, Laiacona & Capitani, 2006: 470). Within this study the determination of clusterings (clusters made of 2, 3, and 4-10107 items) has been adopted from the methodology used by Troyer, Moscovitch and Wincour (1997), in terms of defining subcategories based on semantic and zoological criteria and consecutively organizing the items given in those semantic clusters. In terms of the number of clusterings, switchings, as well as of the number of items a clusters is made of the criteria stated by Reverberi, Laiacona and Capitani (2006) seemed to be appropriate for this study. In detail, the number of clusterings, switchings, 2_clusters, 3_clusters, 4-10_clusters for the semantic fluency task, has been evaluated by dividing the number of clusterings, switchings, 2_clusters, 3_clusters, 4-10_clusters for each supercategory (separately for animals, vegetables, fruits, clothing and birds) through the total number of correct responses given for each supercategory108, including

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106The relative number of switchings is defined as the ratio of the raw number of observed switches divided by the total number of words, including repetitions (cf. Reverberi, Laiacona & Capitani, (cf. 2006: 472f). The relative number of switchings is in contrast to the methodology used by Troyer, Moscovitch and Wincour (1997), focusing on the total number of transitions between clusters as a value for determining the number of switchings.

107For the reason of statistical simplification of the raw data, the infrequent clusters made up of 4,5,6,7,8,9 or 10 items have been pooled to 4-10_clusters.

108If the total number of items given for the supercategory 'animals' is 15 and the number of clusterings
repetitions; thus the number of clusterings, switchings, 2_clusters, 3_clusters and 4-10_clusters could be controlled for the total number of items given. Thus clusterings, switchings, 2, 3, 4-10 clusters controlled for the number of total items have been referred to as clusterings_corr; switchings_corr, 2_clusters_corr, 3_clusters_corr as well as 4-10_clusters_corr. This procedure has been accomplished, because it seems evident that the total number of items given has a strong impact on the number of clusterings, switchings, 2_clusters, 3_clusters, 4-10_clusters; it makes a difference if a person names only 8 items and gives 3 clusters within those items, or names 30 items and also gives only 3 clusters within those items. Consecutively the results of each division for clusterings, switchings, 2_clusters, 3_clusters and 4-10_clusters for all five supercategories have been summed up in order to get the total number of clusterings_corr, switchings_corr, 2_clusters_corr, 3_clusters_corr, as well as 4-10_clusters_corr for the semantic fluency task in total.

6.1.1 Statistical analysis – examination of conditions

In order to justify the statistical analysis as given below, conditions of the raw data have been examined in advance. Normal distribution and homogeneity of variance have been examined by using the Kolmogorov-Smirnov test of goodness of fit to account for the application of an analysis of variance. Results show that normal distribution and homogeneity of variance could be approved for most, but not for all aspects of the semantic fluency task. Even though normal distribution and homogeneity of variance could not be approved for all raw data of this task, MANOVA can still be calculated because the F-test of an analysis of variance is known as being robust against departures from both normality and homogeneity of variance, particularly when the numbers of observations in each group are equal (cf. Stevens, 2007: 57). Further references to justify a MANOVA in this case, are given in chapter 5.1. In detail, MANOVA has been calculated to account for the number of clusterings and switchings, the number of 2_clusters, 3_clusters and 4-10_clusters, between HD patients and healthy controls for the semantic fluency task in general.

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is 3, one has to divide 3/15 = 0.2, which gives you the proportion of clusterings under control of the total number of items given, as presented in chapter 6.1.1.1.

109Details of the Kolmogorov-Smirnov test of goodness of fit, which accounts for normal distribution as well of the Levene-Test, which accounts for homogeneity of variance, are presented in Appendix VIII.
6.1.1.1  **Hypothesis**

There is a statistically significant difference between patients and controls for the number of clusterings_corr, switchings_corr, and clusters_2, 3, 4-10_corr.

MANOVA proves the study hypothesis, by showing that there is a significant effect of the iDV1 group on semantic fluency in general ($F_{5,34} = 3.536; p < 0.05$)\(^{110}\).

Table 19 shows the group effect on semantic fluency in general

<table>
<thead>
<tr>
<th>semantic fluency</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th>Pillai</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.907</td>
<td>0.242</td>
<td>0.863</td>
<td>0.220</td>
<td>0.342</td>
</tr>
</tbody>
</table>

Univariate analysis of variance shows the effect group on clusterings_corr, switchings_corr, 2_clusters_corr, 3_clusters_corr, 4-11_clusters_corr separately. Results indicate a statistically significant effect of the iDV group on **clusterings_corr** ($F_{1,38} = 5.091; p < 0.05$), **switchings_corr**\(^{111}\) ($F_{1,38} = 10.642; p < 0.01$) and on **3_clusters_corr** ($F_{1,38} = 4.273; p < 0.05$). But there couldn't be detected a statistically significant effect of the iDV group on **2_clusters_corr** ($F_{1,38} = 1.978; p > 0.05$) and on **4-10_clusters_corr** ($F_{1,38} = 0.365; p > 0.05$).

Table 20 shows the effect group on clusterings_corr, switchings_corr, 2, 3, 4-10_clusters_corr separately

<table>
<thead>
<tr>
<th>semantic fluency</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
<th></th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>clusterings_corr</td>
<td>1.220</td>
<td>0.244</td>
<td>1.366</td>
<td>0.156</td>
</tr>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>switchings_corr</td>
<td>2.077</td>
<td>0.436</td>
<td>1.568</td>
<td>0.545</td>
</tr>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>2_clusters_corr</td>
<td>0.674</td>
<td>0.256</td>
<td>0.771</td>
<td>0.174</td>
</tr>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>3_clusters_corr</td>
<td>0.247</td>
<td>0.112</td>
<td>0.319</td>
<td>0.106</td>
</tr>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>4-10_clusters_corr</td>
<td>0.316</td>
<td>0.161</td>
<td>0.289</td>
<td>0.121</td>
</tr>
</tbody>
</table>

\(^{110}\)All results of multivariate analysis given in this thesis always refer to Pillai.

\(^{111}\)In detail, an increase in the number of switchings as presented for HD patients within this study, is in accordance with study results by Reverberi, Laiacona and Capitani (2006), using the relative number of switches as an alternative to the questionable methodology by Troyer, Moscovitch and Wincour (1997).
6.1.2 An alternative method for the analysis of fluency tasks

The main problem of fluency tasks is, the problem in defining criteria which allow an objective definition of clusters and switches. This problem is not resolved yet. But an alternative, in using prototypicality values, has been presented by Reverberi, Capitani and Laiacona (2004).

Addressing the problem that semantic categories are 'fuzzy' and can't thus be that easily categorized (cf. Aitchison, 1994: chapter 4) as administered within the methodology by Troyer, Moscovitch and Wincour (1997), Reverberi, Capitani and Laiacona (2004) focused on Rosch's (1975) concept of prototypicality instead. Within their paper and later within the paper by Reverberi, Laiacona and Capitani (2006) this study group points out that only the way how categories are defined changes the number of switchings. If clusters are defined too tight, too many switchings are to occur, against that if categories are defined too loose, an unappropriate number of switchings is defined either. Against that Reverberi, Capitani and Laiacona (2004) used the total number of fruits provided within the category fluency task and created a matrix consisting of all possible fruit pairs based on the total list of fruits given. All pairs have been consecutively rated by alternative raters to define e.g., the grade of prototypicality and the degree of similarity between fruit pairs on a scale raging from 0 to 10. On the basis of these scores, cluster analysis has been performed, defining clusters of items, belonging to the same semantic category. This method can thus be seen as a future directive of this study in order to define more effectively the number of relative clusters and switchings.
6.2 Plural formation

As previously described in chapter 4.4.4, each study subject was presented with 10 German singular words plus indirect article. The subject was asked to give the proper plural form of the given word. As a typical quantitative measure the total number of correct choices was noted. Furthermore the frequencies of various errors types, as well as the percentage of incorrect plural formations, has been considered within a qualitative analysis. Within plural formation the following error subcategories could be found:

1) 0-plural (eine Katze – viele Katze)
2) e-plural (ein Bär – viele Bäre)
3) double plural; -ss and e-plural (ein Känguru – viele Kängurusse)
4) umlaut and e-plural (ein Hund – viele Hunde)
5) lexicalised item and corresponding correct plural (ein Känguru – viele Kakadus)
6) deletion of the final vowel and e-plural (ein Känguru – viele Kängure)
7) phonological proximity and application of inflectional morpheme instead of plural morpheme (ein Huhn – viele Huhndes)
8) autocorrection_right
9) non response

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 21 and 22.
Table 21 shows the frequency of errors made for HD patients versus controls separately, for all error types, within plural formation.

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>frequencies</td>
<td>frequencies</td>
<td></td>
</tr>
<tr>
<td>0-plural</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>e-plural</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>double plural -ss and e-plural</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>umlaut and e-plural</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>lexicalised item and correct plural</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>deletion_final vowel and e-plural</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>phonol. prox. and flex. morpheme_plural morpheme</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>non response</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>11</td>
<td>3</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within plural formation 10 different test items have been presented to each of the 20 HD patients as well as to each of the 20 control subjects of the study (10 x 20 = 200 tested items in total for HD patients vs. 10 x 20 = 200 test items for healthy controls). To assume further, that answering all 200 test items correct, would represents a total of 100%. Thus one has to divide those 100% through the total number of test items presented, in order to get 1 % (100 : 200 = 0.5). The value 0.5 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 11 errors within this task, in comparison to healthy controls, who only made a total of 3 errors, hence it can be inferred that the number of correct responses for HD patients was (200 – 11 = 189) versus (200 – 3 =197) for healthy controls. In Table 22, the percentage of correct responses/errors, for HD patients and controls, is given.

Table 22 shows the percentage of correct responses/errors for HD patients versus healthy controls for plural formation.

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 200 = 0.5 x 189 = 94.5%</td>
<td>100 : 200 = 0.5 x 197 = 98.5 %</td>
</tr>
<tr>
<td>percentage of errors</td>
<td>100 : 200 = 0.5 x 11 = 5.5%</td>
<td>100 : 200 = 0.5 x 3 = 1.5 %</td>
</tr>
</tbody>
</table>
6.3 Providing deverbal and denominal adjectives

As already described in chapter 4.4.6, different nouns and verbs (total amount of 9 items; 6 nouns, 3 verbs) have been presented to each study subject. The subject was asked to generate a proper German adjective out of each given word form. As a typical quantitative measure the total number of correct choices was noted. Quantitative analysis has been accomplished additionally, regarding the frequencies of various error types, as well as the percentage of incorrect derivations. Within providing deverbal and denominal adjectives, the following error subcategories could be found:

1) neologism (Furcht – furchtig)
2) lexical intrusion_noun (Furcht – Früchte)
3) lexical intrusion_adjective (lesen – durchlässig)
4) lexical intrusion_verb (lachen – kichern)
5) deverbal noun (achten – Achtung)
6) denominal verb (Furcht – fürchten)
7) compound_noun (Wind – Windrad)
8) saying (Glück – Glück auf!)
9) derived verb (lesen – verlesen)
10) derived denominal verb_participle (Mutter – bemutternd)
11) denominal verb_participle (Furcht – gefürchtet)
12) compound_participle (Furcht – furchterregend)
13) lexical intrusion_verb_participle (lachen – lächelnd)
14) derived verb_adjective (achten – verächtlich)
15) diminuitive (Holz – Hölzchen)
16) autocorrection_right
17) help_right
18) non response

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 23 and 24.
Table 23 shows the frequency of errors made for HD patients versus controls separately, for all error types, within providing deverbal and denominal adjectives.

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>neologism</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>lexical intrusion_noun</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>lexical intrusion_adjective</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>lexical intrusion_verb</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>deverbal noun</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>denominal verb</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>compound_noun</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>saying</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>derived verb</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>derived denominal verb_participle</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>denominal verb_participle</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>compound_participle</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>lexical intrusion_verb_participle</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>derived verb_adjective</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>diminuitiv</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>help_right</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>non_response</td>
<td>25</td>
<td>5</td>
</tr>
<tr>
<td>total</td>
<td>75</td>
<td>15</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 9 different test items have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study. (9 x 20 = 180 tested items in total for HD patients vs. 9 x 20 = 180 test items for healthy controls). To assume further, that answering all 180 test items correct, would represents a total of 100%. Thus one has to divide those 100% through the total number of test items presented, in order to get 1 % (100 : 180 = 0.555555555). The value 0.555555555 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 75 errors within this task, in comparison to healthy controls, who only made a total of 14 errors, hence it can be inferred that the number of correct responses for HD patients was (180 – 75 = 105) versus (180 – 15 = 165) for healthy controls. Below, in Table 24, the percentage of correct responses/errors, separately for
HD patients and controls, is given. Within this qualitative analysis the percentage of right/wrong answers is given additionally.

Table 24 shows the percentage of correct responses/errors for HD patients versus healthy controls for providing denominal and deverbal adjectives

<table>
<thead>
<tr>
<th>Percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of correct responses</td>
<td>100 : 180 = 0.55555 x 105 = 58.3%</td>
<td>100 : 180 = 0.5555 x 165 = 91.7%</td>
</tr>
<tr>
<td>Percentage of errors</td>
<td>100 : 180 = 0.5555 x 75 = 41.7%</td>
<td>100 : 180 = 0.5555 x 17 = 8.3%</td>
</tr>
</tbody>
</table>

6.4 Providing deverbal and deadjectival nouns

As already described in chapter 4.4.7, different adjectives and verbs (total amount of 10 test items; 3 verbs, 7 adjectives) have been presented to each study subject. The subject was asked to generate a proper German noun out of each given word form. As a typical quantitative measure the total number of correct choices was noted. Quantitative analysis has been accomplished additionally, regarding the frequencies of various error types, as well as the percentage of incorrect derivations. Within providing deverbal and deadjectival nouns, the following error subcategories could be found:

1) comparison (schnell – schneller – am schnellsten)
2) neologism (gut – die Gutheit)
3) nonstandard nominalisation (groß – die Große)
4) adjective phrase (heiter – die heitene Person)
5) lexical intrusion_noun (groß – der Gruß)
6) lexical intrusion_adjective (heiter – lustig)
7) lexical intrusion_verb (streben – bemühen)
8) autocorrection_right
9) help_right
10) non response
Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 25 and 26.

Table 25 shows frequency of errors made for HD patients versus controls separately, for all error types within providing deverbal and deadjectival nouns

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>comparison</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>neologism</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>nonstandard_nominalisation</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>adjective phrase</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>lexical intrusion_noun</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>lexical intrusion_adjective</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>lexical intrusion_verb</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>help_right</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>non response</td>
<td>55</td>
<td>10</td>
</tr>
<tr>
<td><strong>total</strong></td>
<td><strong>92</strong></td>
<td><strong>27</strong></td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 10 different test items have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study. (10 x 20 = 200 tested items in total for HD patients vs. 10 x 20 = 200 test items for healthy controls). To assume further, that answering all 200 test items correct, would represents a total of 100%. Thus one has to divide those 100% through the total number of test items presented, in order to get 1 % (100 : 200 = 0.5). The value 0.5 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 92 errors within this task, in comparison to healthy controls, who only made a total of 25 errors, hence it can be inferred that the number of correct responses for HD patients was (200 – 92 = 108) versus (200 – 27 = 173) for healthy controls.
Below, in Table 26, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 26 shows the percentage of correct responses/errors for HD patients versus healthy controls for providing deverbal and deadjectival nouns.

<table>
<thead>
<tr>
<th>Percentages</th>
<th>HD Patients (n=20)</th>
<th>Controls (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of correct responses</td>
<td>100 : 200 = 0.5 x 108 = 54%</td>
<td>100 : 200 = 0.5 x 173 = 86.5%</td>
</tr>
<tr>
<td>Percentage of errors</td>
<td>100 : 200 = 0.5 x 92 = 46%</td>
<td>100 : 200 = 0.5 x 27 = 13.5%</td>
</tr>
</tbody>
</table>

6.5 Sequencing of sentence parts (= syntax I)

As previously described in chapter 4.4.8, a total of 20 sentences has been presented to HD patients and healthy controls respectively. Each sentence has been divided into its sentence phrases or to make it even more complicate, sometimes even into parts of its sentence phrases (e.g., /in den Ferien/ > /in/ den Ferien/) and presented to the subject in random order. All study participants were asked to arrange the given sentence phrases/parts of sentence phrases in a grammatical proper way to generate a meaningful sentence. Quantitative analysis was based on the number of correct sequencings, in sum for all 20 sentences together. A maximum score of 110 could be reached within this task, by summarising all sentence phrases/parts of sentence phrases for all 20 test sentences. Concerning a qualitative analysis the percentage of sequencing errors in respect to the total number of sentence phrases/parts of sentence phrases, per sentence has been recorded within syntax I (as provided below in Table 27). In detail, sentence 1, is made up of 6 sentence phrases/parts of sentence phrases, thus assuming that sequencing all 6 sentence phrases/parts of sentence phrases right, would represent a total of 100%. Thus one has to divide those 100% through the total number of sentence phrases/parts of sentence phrases, that is 6 for sentence 1, in order to get 1% (100 : 6 = 16.66666667). The value 16.66666667 has further to be multiplied by the number of sequencing errors for sentence 1 (for details see Appendix XII) (in total 15 sequencing errors : 20 HD patients/healthy controls = 0.75), in order to get the percentage of sequencing errors for sentence one, corrected for the number of sentence phrases/parts of sentence phrases (16.66666667 x 0.75 = 12.5%).
The same procedure has been applied to all 20 sentences, resulting in the following percentages, as presented in Table 27.

Table 27 shows the percentage of sequencing errors for all 20 test sentences separately, always corrected for the number of sentence phrases/parts of sentence phrases.

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>errors_sequencing_sentence parts_sentence_1</td>
<td>12.5%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_2</td>
<td>10%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_3</td>
<td>11%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_4</td>
<td>11.4%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_5</td>
<td>12.5%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_6</td>
<td>11%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_7</td>
<td>10%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_8</td>
<td>10%</td>
<td>0.80%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_9</td>
<td>11.4%</td>
<td>0.70%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_10</td>
<td>10%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_11</td>
<td>11%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_12</td>
<td>16%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_13</td>
<td>13.3%</td>
<td>2.5%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_14</td>
<td>20.5%</td>
<td>0.5%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_15</td>
<td>11%</td>
<td>1%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_16</td>
<td>10%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_17</td>
<td>11%</td>
<td>2%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_18</td>
<td>13.3%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_19</td>
<td>14.3%</td>
<td>0%</td>
</tr>
<tr>
<td>errors_sequencing_sentence parts_sentence_20</td>
<td>10%</td>
<td>0%</td>
</tr>
</tbody>
</table>

Regarding the type of errors made, all mis-sequenced sentence parts have been identified and labeled in terms of linguistics (e.g., mis-sequencing of the modal verb /Wann/ihr/wieder/möchtet/zum Training/kommen/).

The full list of mis-sequenced sentences, marked linguistically for the type of mis-sequencings, separated for patients and controls, is given in Appendix XIII. Within this chapter, Table 28 is given, presenting each the total number of errors made within each linguistically defined type of sequencing errors.
Table 28 shows each the number of errors made within each type of sequencing errors, for HD patients and controls separately.

<table>
<thead>
<tr>
<th>linguistic types of sequencing errors</th>
<th>Errors_HD</th>
<th>Errors_controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>verbal phrase (predicate)</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>auxiliray</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>modal verb</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>O&lt;sub&gt;gen&lt;/sub&gt;</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>O&lt;sub&gt;Dat&lt;/sub&gt;</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>O&lt;sub&gt;Akk&lt;/sub&gt;</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>nominal phrase (subject)</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>adverb</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>adjective</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>preposition</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>prepositional phrase</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>possessive pronoun</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>(possessive) pronoun_phrase</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>personal pronoun</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>reflexive pronoun</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>conjunctional phrase</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>non response</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

6.6 Guided syntactic production (= syntax II)

As already presented in chapter 4.4.9, three words (each word on a separate card) (total amount of 30 words for 10 sentences) were given to the subject. The subject was asked to provide a meaningful sentence using at least the three given words, but no matter in which sequence. Quantitative analysis was based on the number of correct sentences produced.

Regarding qualitative analysis the frequencies of various error types, as well as the percentage of errors made, have been calculated. Within guided syntactic production, the following error subcategories could be found:

1) nominal inflection (Mutter – Mütter)
2) verbal inflection (schreibst – schreibt)
3) indefinite pronoun inflection (keine – kein)
4) composition (anders – anderswo)
5) comparison adverb (spät – später)
6) phonological error (wichtig – richtig)
7) change in word class (wichtig; adverb > wichtigen; adjective)
8) morphological agreement ('Der Junge hat das Glas zerbrochen, weil ihnen fad war')
9) change of modal verb (möchte – will)
10) scipping of element(s) ('Ich will nicht mehr'; 'keine' has been scopped)
11) semantic error_change in saying ('Lieber macht man's anders als man denkt')
12) semantic error_change of lexeme (Herrchen – Nachbar)
13) semantic agreement_causal conjunction ('Der Junge hat ein Fenster zerbrochen, weil er schlimm war')
14) delusion by homophones (spät – späht)
15) semantic incorrect sentence ('Weil der Junge zerbrochen ist...')
16) incomplete sentence
17) autocorrection_right
18) help_right
19) non response
Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 29 and 30.

Table 29 shows frequency of errors made for HD patients versus controls separately, for all error types within guided syntactic production.

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>nominal inflection</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>verbal inflection</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>indefinite pronoun inflection</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>composition</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>comparison_adverb</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>phonological error</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>change in word class</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>morphological agreement</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>change of modal verb</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>scipping of element(s)</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>semantic error_change in saying</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>semantic error_change of lexeme</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>semantic agreement_causal conjugation</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>delusion by homophones</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>semantic incorrect sentence</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>incomplete sentence</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>help_right</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>non response</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td><strong>total</strong></td>
<td><strong>78</strong></td>
<td><strong>6</strong></td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. As already presented in chapter 4.4.9, 3 test items, plus the instruction to make a correct (1) as well as complete sentence (1) out of the three given words (in total 5 tasks) were given to each study subject. 10 times, 5 different tasks in terms of various words presented (3 test items, plus the instruction to make a correct (1) as well as complete sentence (1) out of the three given words), have been given to 20 HD patients as well as to each of the 20 control subjects of the study (5 x 10 x 20 = 1000 tested items in total). To assume further, that answering all 1000 test items correct, would represent a total of 100%. Thus one has to divide those 100% through the total number of test items presented, in order to get 1 % (100 : 1000 = 0.1). The value 0.1 has further
to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 79 errors within this task, in comparison to healthy controls, who only made a total of 6 errors, hence it can be inferred that the number of correct responses for HD patients is \((1000 – 78 = 922)\) versus \((1000 – 6 = 994)\) for healthy controls. Below, in Table 30, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 30 shows the percentage of correct responses/errors for HD patients versus healthy controls for guided syntactic production.

<table>
<thead>
<tr>
<th></th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>(100 : 1000 = 0.1 \times 922 = 92.2%)</td>
<td>(100 : 1000 = 0.1 \times 994 = 99.4%)</td>
</tr>
<tr>
<td>percentage of errors</td>
<td>(100 : 1000 = 0.1 \times 78 = 7.8%)</td>
<td>(100 : 1000 = 0.1 \times 6 = 0.6%)</td>
</tr>
</tbody>
</table>

6.7 Providing antonyms

As previously described in chapter 4.5.3, 10 words (3 nouns, 4 verbs, 3 adjectives) were presented to the subject. The subject was asked to give a word which is opposite in meaning to the given word.

Within quantitative analysis the number of correct antonyms has been recorded. Regarding qualitative analysis the frequencies of various error types, as well as the percentage of incorrect antonyms, have been calculated. Within providing antonyms, the following error subcategories could be found:

1) not-negation (e.g., zugeben – nicht zugeben)
2) lexical abberation (e.g., gut – sauer)
3) synonym (e.g., aufmachen – öffnen)
4) help_right
5) non response
Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 31 and 32.

Table 31 shows frequency of errors made for HD patients versus controls separately, for all error types within providing antonyms

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20) frequencies</th>
<th>controls (n = 20) frequencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>not-negation</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>lexical intrusion</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>synonym</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>help_right</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>non response</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>20</td>
<td>2</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 10 test items have been presented to each of the 20 HD patients as well as to each of the 20 control subjects of the study. (10 x 20 = 200 tested items in total for HD patients vs. 10 x 20 = 200 test items for healthy controls). To assume further, that answering all 200 test items correct, would represent a total of 100%. Thus one has to divide those 100% through the total number of test items presented, in order to get 1 % (100 : 200 = 0.5). The value 0.5 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 20 errors within this task, in comparison to healthy controls, who only made a total of 2 errors, hence it can be inferred that the number of correct responses for HD patients was (200 – 20 = 180) versus (200 – 2 = 198) for healthy controls. Below, in Table 32, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 32 shows the percentage of correct responses/errors for HD patients versus healthy controls for providing antonyms

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 200 = 0.5 x 180 = 90 %</td>
<td>100 : 200 = 0.5 x 198 = 99 %</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 200 = 0.5 x 20 = 10 %</td>
<td>100 : 200 = 0.5 x 2 = 1 %</td>
</tr>
</tbody>
</table>
6.8 Providing two semantic concepts of polysemes

As previously presented in chapter 4.5.4, all study subjects were confronted visually with 9 polysemic words and were asked to give at least two semantic concepts of the given word. Thus quantitative analysis was based on the number of correct semantic concepts given, per polysemic word.

Furthermore the frequencies of various error types, as well as the percentage of errors, has been considered within a qualitative analysis.

Within providing two definitions of polysemes the following error subcategories could be found:

1) naming of only one item (Ball: den Ball zum Spielen)
2) 2nd naming is referring to the same lexeme as 1st (Ball: den Ball zum Tennisspielen, den Ball zum Fußballspielen)
3) compounding (Leiter: die Himmelsleiter)
4) lexicalised element (Stimme: die Stimmung)
5) help_right
6) non response

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 33 and 34.

Table 33 shows frequency of errors made for HD patients versus controls separately, for all error types within providing two definitions of polysemes

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>naming of only one item</td>
<td>14</td>
<td>7</td>
</tr>
<tr>
<td>2nd naming referring to the same lexeme as 1st</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>compounding</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>lexicalised item</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>help_right</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>non response</td>
<td>38</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>75</td>
<td>8</td>
</tr>
</tbody>
</table>
Within qualitative analysis the percentage of correct/wrong answers is given additionally. As already described in chapter 4.5.4, 9 test items, plus the instruction to give at least two semantic concepts of the given word (2) (in total 9 x 2 = 18 tasks) have been presented to each of the 20 HD patients as well as to each of the 20 control subjects of the study. (9 x 2 x 20 = 360 tested items in total for HD patients vs. 9 x 2 x 20 = 360 test items for healthy controls). To assume further, that answering all 360 test items correct, would represents a total of 100%. Thus one has to divide those 100% through the total number of test items presented, in order to get 1 % (100 : 360 = 0.277777777). The value 0.277777777 has further to be multiplicated by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 75 errors within this task, in comparison to healthy controls, who only made a total of 8 errors, hence it can be inferred that the number of correct responses for HD patients was (360 – 75 = 285) versus (360 – 8 = 352) for healthy controls. Below, in Table 34, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 34 shows the percentage of correct responses/errors for HD patients versus healthy controls for providing two definitions of polysemes

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 360 = 0.2777777 x 285 = 79.2 %</td>
<td>100 : 360 = 0.2777777 x 352 = 97.8 %</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 360 = 0.2777777 x 75 = 20.8 %</td>
<td>100 : 360 = 0.2777777 x 8 = 2.2 %</td>
</tr>
</tbody>
</table>
6.9 Providing two meanings of homophonic words

As previously described in chapter 4.5.5, the subject was confronted auditively with a total of 10 homophonic words and was asked to provide at least two meanings of the given word.

Quantitative analysis was based on the number of correct meanings provided per word. Regarding qualitative analysis the frequencies of various error types, as well as the percentage of errors made, have been considered.

Within providing two meanings of homophonic words the following error subcategories could be found:

1) naming of only one item (Rad/Rat: das Rad zum Fahren)
2) 2nd naming referring to the same lexeme as 1st (Waise/weise: wenn man viel weiß, ist man weise & eine Weise erzählen)
3) 2 namings referring to the same lexeme_semantic relation (küsste/Küste: die Küste am Meer, die Klippen)
4) new lexeme (bete/Beete: bitten)
5) compounding (Saite/Seite: Seitengasse)
6) compounding_neologism (Seidenseite)
7) saying (arm/Arm: arm, ich bin arm, wie eine Kirchenmaus)
8) help_right
9) non response
Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 35 and 36.

Table 35 shows frequency of errors made for HD patients versus controls separately, for all error types within providing two meanings of homophonic words

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>naming of only one item</td>
<td>34</td>
<td>7</td>
</tr>
<tr>
<td>2nd naming referring to the same lexeme as 1st</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>2 namings referring to the same lexeme_semantic relation</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>new lexeme</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>composition</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>composition_neologism</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>saying</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>help_right</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>non response</td>
<td>74</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>131</td>
<td>10</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. As already given in chapter 4.5.5, 10 test items, plus the instruction to give at least two meanings of the homophonic word (2) (in total 10 x 2 = 20 tasks) have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study. (10 x 2 x 20 = 400 tested items in total for HD patients vs. 10 x 2 x 20 = 400 test items for healthy controls). To assume further, that answering all 400 test items correct, would represent a total of 100%. Thus one has to divide those 100% through the total number of test items presented, in order to get 1 % (100 : 400 = 0.25). The value 0.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 131 errors within this task, in comparison to healthy controls, who only made a total of 10 errors, hence it can be inferred that the number of correct responses for HD patients was (400 – 131 = 269) versus (400 – 10 = 390) for healthy controls. Below, in Table 36, the percentage of correct responses/errors, separately for HD patients and controls, is given.
Table 36 shows the percentage of correct responses/errors for HD patients versus healthy controls for providing two meanings of homophonic words

<table>
<thead>
<tr>
<th></th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 400 = 0.25 x 269 = 67.25%</td>
<td>100 : 400 = 0.25 x 390 = 97.5%</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 400 = 0.25 x 131 = 32.75%</td>
<td>100 : 400 = 0.25 x 10 = 2.5%</td>
</tr>
</tbody>
</table>

6.10 Decoding of thematic roles (agent, patient, recipient) (= syntax III)

As already described in chapter 4.5.9, a sentence-picture matching task (4 pictures per sentence) consisting of 32 sentences, separated for 8 different syntactic complexity levels ranging from a-h, regarding the decoding of thematic roles, such as agent, patient and recipient, has been constructed, adopted from Gadler (1988). Each syntactic complexity level comprised 4 sentences (8 x 4 = 32 test sentences in total). In detail, syntactic complexity ranged from: (a) active sentences with monovalent verbs (a. Das Auto fährt.); (b) active sentences with obligatory bivalent verbs (b. Der Hund beißt die Katze.); (c) passive constructions with obligatory bivalent verbs (c. Die Katze wird vom Hund gebissen.); (d) active sentences with obligatory trivalent verbs (d. Der Bub gibt dem Mädchen die Katze.); (e) passive constructions with obligatory trivalent verbs (e. Die Katze wird dem Mädchen vom Buben gegeben.); (f) coordinated sentences112 (f. Die Katze beißt den Hund und kratzt den Affen.); (g) embedded relative clauses (g. Die Katze, die den Hund beißt, kratzt den Affen.) and (h) attached relative clauses (h. Die Katze beißt den Hund, der den Affen kratzt.)

Each subject was asked to read the given sentence and to point to the appropriate picture, representing the correct thematic role assignment. A detailed description concerning thematic role assignment of all test sentences provided, is given in Appendix XIX. Different error patterns did occur in this task, regarding various thematic role permutations. All types of permutational errors will be presented separately for syntax III_a-h, in the subsequent section of this work.

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112A coordinated clause is a clause belonging to a series of two or more clauses which are not syntactically dependent from one another and are joined by means of for instance a coordinating conjunction (e.g., and) (cf. Crystal, 1980: 92).
6.10.1 Task a

Within task (a) active sentences with monovalent verbs the following error types could be found:

1) Choices based on different predicates (PD) (*Das Auto fährt. – Das Auto steht.*)
2) Choices based on different subjects (SD) (*Das Auto fährt. – Der Traktor fährt*)

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 37 and 38.

Table 37 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles a

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20) frequencies</th>
<th>controls (n = 20) frequencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>SD</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>9</td>
<td>0</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for a) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences, would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to get 1 % (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 9 errors within this task, in comparison to healthy controls, who only made a total of 0 errors, hence it can be inferred that the number of correct responses for HD patients was (80 – 9 = 71) versus (80 – 0 = 80) for healthy controls. Below, in Table 38, the percentage of correct responses/errors, separately for HD patients and controls, is given.
Table 38 shows the percentage of correct responses/errors for HD patients versus healthy controls for decoding of thematic roles_a

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>$100 : 80 = 1.25 \times 71 = 88.8%$</td>
<td>$100 : 80 = 1.25 \times 80 = 100 %$</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>$100 : 80 = 1.25 \times 9 = 11.2 %$</td>
<td>$100 : 80 = 1.25 \times 0 = 0%$</td>
</tr>
</tbody>
</table>

### 6.10.2 Task b

Within task (b) active sentences with obligatory bivalent verbs, the following errors could be found:

1) Choices based on different objects (OD)
   
   *(Der Hund beißt die Katze. – Der Hund beißt das Schwein.)*

2) Permutations between subject and predicate (S↔O)
   
   *(Der Hund beißt die Katze. – Die Katze beißt den Hund.)*

3) autocorrection_right

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 39 and 40.

Table 39 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles_b

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>OD</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>S↔O</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>12</td>
<td>1</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for b) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to get 1
% (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of
correct/wrong answers within this task, separately for HD patients and healthy controls,
in order to get the percentage of correct/wrong answers, respectively the percentage of
correct responses/errors. As given above, HD patients made 12 errors within this task, in
comparison to healthy controls, who only made a total of 1 errors, hence it can be
inferred that the number of correct responses for HD patients was (80 – 12 = 68) versus
(80 – 1 = 79) for healthy controls. Below, in Table 40, the percentage of correct
responses/errors, separately for HD patients and controls, is given.

Table 40 shows the percentage of correct responses/errors for HD patients versus healthy controls for
decoding of thematic roles

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 80 = 1.25 x 68 = 85%</td>
<td>100 : 80 = 1.25 x 79 = 98.8 %</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 80 = 1.25 x 12 = 15%</td>
<td>100 : 80 = 1.25 x 1 = 1.2 %</td>
</tr>
</tbody>
</table>

**6.10.3 Task c**

Within task (c) passive constructions of simple sentences with obligatory bivalent
verbs, the following errors could be found:

1) Choices based on a different subjects (SD)
   
   (Die Katze wird vom Hund gebissen. – Das Schwein wird vom Hund gebissen.)

2) Permutations of subject and object (S↔O)
   
   (Die Katze wird vom Hund gebissen. – Der Hund wird von der Katze gebissen.)

3) autocorrection_right

Frequencies of various error types and the percentage of correct responses versus errors
made, are provided in Table 41 and 42.
Table 41 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles.

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>SD</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>S→O</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>10</td>
<td>0</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for c) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences, would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to get 1% (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 12 errors within this task, in comparison to healthy controls, who only made a total of 1 errors, hence it can be inferred that the number of correct responses for HD patients was (80 – 10 = 70) versus (80 – 0 = 80) for healthy controls. Below, in Table 42, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 42 shows the percentage of correct responses/errors for HD patients versus healthy controls for decoding of thematic roles.

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 80 = 1.25 x 70 = 87.5%</td>
<td>100 : 80 = 1.25 x 80 = 100 %</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 80 = 1.25 x 10 = 12.5 %</td>
<td>100 : 80 = 1.25 x 0 = 0%</td>
</tr>
</tbody>
</table>
6.10.4 Task d

Within task (d) active sentences with obligatory trivalent verbs the following errors could be found:

1) Choices based on a different direct object (dirOD)
   
   (Der Bub gibt dem Mädchen die Katze. – Der Bub gibt dem Mädchen den Hasen.)

2) Permutations of the subject and the indirect object (S↔indirO)
   
   (Der Bub gibt dem Mädchen die Katze. – Das Mädchen gibt dem Buben die Katze.)

3) Permutations of the subject and the noun of the prepositional phrase
   
   (S↔noun of PP) (Der Mann setzt das Baby zum Mädchen. – Das Mädchen setzt das Baby zum Mann.)

4) autocorrection_right

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 43 and 44.

Table 43 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles_d

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>frequencies</td>
<td>frequencies</td>
<td></td>
</tr>
<tr>
<td>dirOD</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>S↔indirO</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>S↔noun of PP</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>19</td>
<td>1</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for d) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences, would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to
get 1 % (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 19 errors within this task, in comparison to healthy controls, who only made a total of 1 errors, hence it can be inferred that the number of correct responses for HD patients was (80 – 19 = 61) versus (80 – 1 = 79) for healthy controls. Below, in Table 44, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 44 shows the percentage of correct responses/errors for HD patients versus healthy controls for decoding of thematic roles

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 80 = 1.25 x 61 = 76.3%</td>
<td>100 : 80 = 1.25 x 79 = 98.8 %</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 80 = 1.25 x 19 = 23.7%</td>
<td>100 : 80 = 1.25 x 1 = 1.2 %</td>
</tr>
</tbody>
</table>

6.10.5 Task e

Within task (e) passive constructions of simple sentences with obligatory trivalent verbs, the following error types could be found:

1) Permutations of the indirect object and the noun of the prepositional phrase

   (indirO→noun of PP)

   (Die Katze wird dem Mädchen vom Buben gegeben. –
   Die Katze wird dem Buben vom Mädchen gegeben.)

2) Permutations of the noun of the first PP and the noun of the second PP

   (noun of PP1→noun of PP2)

   (Die Blumen werden vom Großvater zur Großmutter gestellt.
   – Die Blumen werden von der Großmutter zum Großvater gestellt.)

3) Choices based on a different subjects (SD)

   (Die Katze wird dem Mädchen vom Buben gegeben. – Der Hase wird dem Mädchen vom Buben gegeben.).
Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 45 and 46.

Table 45 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles._e

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>indir.O→noun of PP</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>noun of PP1→noun of PP2</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>SD</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>13</td>
<td>0</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for e) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences, would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to get 1 % (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 13 errors within this task, in comparison to healthy controls, who only made a total of 0 errors, hence it can be inferred that the number of correct responses for HD patients was (80 – 13 = 67) versus (80 – 0 = 80) for healthy controls. Below, in Table 46, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 46 shows the percentage of correct responses/errors for HD patients versus healthy controls for decoding of thematic roles._e

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 80 = 1.25 x 67 = 83.8 %</td>
<td>100 : 80 = 1.25 x 80 = 100 %</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 80 = 1.25 x 13 = 16.2 %</td>
<td>100 : 80 = 1.25 x 0 = 0 %</td>
</tr>
</tbody>
</table>
6.10.6 Task f

Within task (f) coordinate sentences, the following error types could be found:

1) Permutations of the direct object of the first clause with the direct object of the second clause (dirO1↔dirO2)
   
   (Die Katze beißt den Hund und kratzt den Affen. – Die Katze beißt den Affen und kratzt den Hund.)

2) Permutations of the subject of the sentence with the direct object of the second clause (S↔dirO2) (Die Katze beißt den Hund und kratzt den Affen – Der Affe beißt den Hund und kratzt die Katze)

3) Permutations of the subject of the sentence with the object of the first clause (S↔dirO1) (Die Katze beißt den Hund und kratzt den Affen. – Der Hund beißt die Katze und kratzt den Affen.)

4) autcorrections_right

5) help_right

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 47 and 48.

Table 47 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles_f

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>dirO1↔dirO2</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>S↔dirO2</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>S↔dirO1</td>
<td>18</td>
<td>3</td>
</tr>
<tr>
<td>autcorrection_right</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>help_right</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>total</td>
<td>33</td>
<td>7</td>
</tr>
</tbody>
</table>
Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patient as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for f) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences, would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to get 1% (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 33 errors within this task, in comparison to healthy controls, who only made a total of 7 errors, hence it can be inferred that the number of correct responses for HD patients was (80 – 33 = 47) versus (80 – 7 = 73) for healthy controls. Below, in Table 48, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 48 shows the percentage of correct responses/errors for HD patients versus healthy controls for decoding of thematic roles_f

<table>
<thead>
<tr>
<th>Percentages</th>
<th>HD Patients (n = 20)</th>
<th>Controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of correct responses</td>
<td>100 : 80 = 1.25 x 33 = 41.3%</td>
<td>100 : 80 = 1.25 x 73 = 91.3%</td>
</tr>
<tr>
<td>Percentage of errors made</td>
<td>100 : 80 = 1.25 x 47 = 58.7%</td>
<td>100 : 80 = 1.25 x 7 = 8.7%</td>
</tr>
</tbody>
</table>

6.10.7 Task g

Within task (g) embedded relative clauses, the following error types could be found:

1) Permutations of the object of the matrix (O1) clause with the object of the relative clause (O2) (O1 ↔ O2)

   *(Der Lastwagen, der den Traktor zieht, schiebt das Auto. – Der Lastwagen, der das Auto zieht, schiebt den Traktor.)*

2) Permutations of the subject of the matrix clause (S1) with the object of the relative clause (O2) (S1 ↔ O2_Sfocus)

   *(Der Lastwagen, der den Traktor zieht, schiebt das Auto. – (Der Traktor, der den Lastwagen zieht, schiebt das Auto.)*
3) Permutations of the subject of the matrix clause (S1) with the object of the matrix clause

\[ (O1) \text{(S1} \leftrightarrow \text{O1}_S\text{focus}) \]

\((\text{Der Lastwagen, der den Traktor zieht, schiebt das Auto. – (Das Auto, das den Traktor zieht, schiebt den Lastwagen.)})\)

4) Permutations of the object of the matrix clause (O1) with the subject of the relative clause (S2) \( (O1 \leftrightarrow S2_O\text{focus}) \)

\((\text{Der Mann, den das Mädchen küsst, stößt den Buben. – Der Mann, den der Bub küsst, stößt das Mädchen.)})\)

5) autocorrection_right

Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 49 and 50.

Table 49 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles _g_

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>O1\rightarrow O2_S\text{focus}</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>S1\rightarrow O2_S\text{focus}</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>S1\rightarrow O1_S\text{focus}</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>O1\rightarrow S2_O\text{focus}</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>autocorrection_right</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>26</td>
<td>6</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patients as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for _g_) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences, would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to get 1 % (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of
correct responses/errors. As given above, HD patients made 26 errors within this task, in comparison to healthy controls, who only made a total of 6 errors, hence it can be inferred that the number of correct responses for HD patients was 
(80 – 26 = 54) versus (80 – 6 = 74) for healthy controls. Below, in Table 50, the percentage of correct responses/errors, separately for HD patients and controls, is given.

Table 50 shows the percentage of correct responses/errors for HD patients versus healthy controls for decoding of thematic roles_g

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 80 = 1.25 x 54 = 67.5%</td>
<td>100 : 80 = 1.25 x 74 = 92.5%</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 80 = 1.25 x 26 = 32.5%</td>
<td>100 : 80 = 1.25 x 6 = 7.5%</td>
</tr>
</tbody>
</table>

6.10.8 Task h

Within task (h) attached relative clauses, the following error types could be found:

1) Permutations of the subject of the main clause (S1) with the object of the relative clause (O2) (S1↔O2_Sfocus)
   
   (Die Katze beißt den Hund, der den Affen kratzt. – Der Affe beißt den Hund, der die Katze kratzt.)

2) Permutations of the subject of the main (S1) clause with the object of the main clause (O1) (S1↔O1_Sfocus) (Die Katze beißt den Hund, der den Affen kratzt. – Der Hund beißt die Katze, die den Affen kratzt.)

3) Permutations of the object of the main clause (O1) with the object of the relative clause (O2) (O1↔O2_Sfocus)
   
   (Die Katze beißt den Hund, der den Affen kratzt. – Die Katze beißt den Affen, der den Hund kratzt.)

4) Permutations of the subject of the main (S1) with the subject of the relative clause (S2) (S1↔S2_Ofocus) (Der Traktor schiebt das Auto, das der Lastwagen zieht. – Der Lastwagen schiebt das Auto, das der Traktor zieht.)

5) Permutations of the object of the main clause (O1) with the subject of the relative clause (= relative pronoun) (S2) (O1↔S2_Ofocus) (Der Traktor schiebt das Auto, das der Lastwagen zieht. – Der Traktor schiebt den Lastwagen, den das Auto zieht.)
Frequencies of various error types and the percentage of correct responses versus errors made, are provided in Table 51 and 52.

Table 51 shows frequency of errors made for HD patients versus controls separately, for all error types within decoding of thematic roles_h

<table>
<thead>
<tr>
<th>error types</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>frequencies</td>
<td>frequencies</td>
</tr>
<tr>
<td>S1↔O2_Sfocus</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>S1↔O1_Sfocus</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>O1↔O2_Sfocus</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>S1↔S2_Ofocus</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>O1↔S2_Ofocus</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>autocorrections_right</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>help_right</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>24</td>
<td>4</td>
</tr>
</tbody>
</table>

Within qualitative analysis the percentage of correct/wrong answers is given additionally. Within this task 4 sentences have been presented to each of the 20 HD patients as well as to each of the 20 control subjects of the study, per syntactic complexity level (4 for h) (4 x 20 = 80 tested sentences in total for HD patients vs. 4 x 20 = 80 test sentences for healthy controls). To assume further, decoding of thematic roles correctly for all 80 test sentences, would represent a total of 100%. Thus one has to divide those 100% through the total number of test sentences presented, in order to get 1 % (100 : 80 = 1.25). The value 1.25 has further to be multiplied by the number of correct/wrong answers within this task, separately for HD patients and healthy controls, in order to get the percentage of correct/wrong answers, respectively the percentage of correct responses/errors. As given above, HD patients made 26 errors within this task, in comparison to healthy controls, who only made a total of 6 errors, hence it can be inferred that the number of correct responses for HD patients was (80 – 24 = 56) versus (80 – 4 = 76) for healthy controls.
Table 52, shows the percentage of correct responses/errors, separately for HD patients and controls.

Table 52 shows the percentage of correct responses/errors for HD patients versus healthy controls for decoding of thematic roles.

<table>
<thead>
<tr>
<th>percentages</th>
<th>HD patients (n = 20)</th>
<th>controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>percentage of correct responses</td>
<td>100 : 80 = 1.25 x 56 = 70%</td>
<td>100 : 80 = 1.25 x 76 = 95%</td>
</tr>
<tr>
<td>percentage of errors made</td>
<td>100 : 80 = 1.25 x 24 = 30%</td>
<td>100 : 80 = 1.25 x 4 = 5%</td>
</tr>
</tbody>
</table>

6.10.9 Types of errors made within syntax III

As this task was about testing syntactic comprehension, in detail about decoding of thematic roles within (reversible) syntax, the type of theta role mis-decodings for all erroneous sentences will be given within this chapter. A description of theta role assignments for all sentences used, is given in Appendix XIX. On the basis of this description, theta role mis-decodings have been worked out and are presented in Table 53 separately for patients and controls.
Table 53 shows theta role mis-decodings in decoding of thematic roles a-h

<table>
<thead>
<tr>
<th></th>
<th>syntax III</th>
<th>theta roles</th>
<th>errors HD</th>
<th>errors controls</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>a</strong></td>
<td>PD</td>
<td>different predicate</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>different subject</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td><strong>b</strong></td>
<td>OD</td>
<td>different object</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>S→O</td>
<td>agent→patient</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>autocorrection_right</td>
<td></td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td><strong>c</strong></td>
<td>SD</td>
<td>different subject</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>S→O</td>
<td>agent→patient</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>autocorrection_right</td>
<td></td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td><strong>d</strong></td>
<td>dirOD</td>
<td>different dir. object</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>S→indirO</td>
<td>agent→recipient</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>S→noun of PP</td>
<td>agent→locativ</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>autocorrection_right</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>e</strong></td>
<td>indirO→noun of PP</td>
<td>agent→recipient</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>noun of PP1→noun of PP2</td>
<td>agent→locativ</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>different subject</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td><strong>f</strong></td>
<td>dirO1→dirO2</td>
<td>patient→patient</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>S→dirO2</td>
<td>agent→patient</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>S→dirO1</td>
<td>agent→patient</td>
<td>18</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>autocorrection_right</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>help_right</td>
<td></td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td><strong>g</strong></td>
<td>O1→O2_Sfocus</td>
<td>patient→patient</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>S1→O2_Sfocus</td>
<td>agent→patient</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>S1→O1_Sfocus</td>
<td>agent→patient</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>O1→S2_Ofocus</td>
<td>agent→patient</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>autocorrection_right</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>h</strong></td>
<td>S1→O2_Sfocus</td>
<td>agent→patient</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>S1→O1_Sfocus</td>
<td>agent→patient</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>O1→O2_Sfocus</td>
<td>patient→patient</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>S1→S2_Ofocus</td>
<td>agent→agent</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>O1→S2_Ofocus</td>
<td>agent→patient</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>autocorrection_right</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>help_right</td>
<td></td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>
7 Discussion

The purpose of this study was to investigate language functions in HD and thus to provide further evidence for a role of the basal ganglia in language.
In detail, within this study the performance of 20 HD patients (stage I, II, III) has been compared to age, sex and education matched healthy controls, in three linguistic domains including semantics, morphology and syntax. Tasks provided, differed in terms of their linguistic complexity, and hence 'complex language tasks' with a 'high degree of freedom' have been contrasted to 'primary linguistic abilities' with a 'low degree of freedom'.
Within this chapter a discussion of results will be given, including a specific discussion concerning the contribution of clinical values onto language outcomes, as well as specific linguistic changes in the course of HD. Linguistic errors are explained on the basis of linguistic considerations and linguistically based cortico-basal ganglia-thalamo-cortical circuit models.

7.1 Discussion of results – general

Main results of this study demonstrate HD patients to perform statistically significantly worse in all subtasks of the language screening, compared to matched healthy controls, and thus proving study hypothesis 1, 2 and 3, as demonstrated in chapter 3.9.6.2. These study results are in accordance with study outcomes e.g., by Kennedy et al. (1981), Gordon and Illes (1987), Speedie et al. (1990), Hodges, Salmon and Butters (1991), Frank, Mc Dade and Scott (1996), Ullman (1997a), Murray and Stout (1999), Murray (2000), Murray and Lenz (2001), Chenery, Copland and Murdoch (2002), Jensen, Chenery and Copland (2006), Teichmann et al. (2005, 2006, 2007, 2008), as these authors also reported statistically significantly poorer performances for HD patients in various language tasks, compared to matched controls.
Study hypothesis 4, 5 and 6 as described in chapter 3.9.6.2., assuming a statistically significant effect of HD stage on the performance of the language screening (language production and language comprehension), couldn't be proved within this study. Interestingly, there couldn't be shown a statistically significant effect of HD stage on the
language screening and thus neither on language production and language comprehension subtests. No matter in which stage of the disease HD patients were, all performed statistically significantly worse on the language screening, than did their healthy counterparts. These results are in accordance with the study outcomes by Saldert et al. (2009: personal communication), as this study group didn't find either any differences in results in regards to HD stage. This lack of effect of HD stage on the language output, might be due to a too loose or inappropriate definition of HD stages per se. As HD stages are rated for instance on the ability to manage daily life, including household responsibilities and financial affairs (for more information see a description of the Shoulson and Fahn, 1979 TFC scale, in chapter 1.5), hence it is not surprising that the inability in one or several of these domains does not necessarily contribute negatively onto language. Even though not yet addressed explicitly within the current HD literature dealing with language changes, a significant negative correlation could be found between the disease progression score 'days since disease onset' under control of the disease onset score, 'number of CAG repeats' and results of the language screening. Thus, study hypothesis 7 could be proved, as exemplified in chapter 3.9.6.2. In detail, results indicate, that the more days a patient evidences core symptoms of HD, the more affected language performance gets. This result seems obvious, as with further progression of HD, patients are getting more and more demented, evidence more and more motor and also depressive disturbances, which in sum are likely to contribute negatively onto language. This assumption could be proved by demonstrating a statistically significant negative correlation between the performance in the language screening and the UHDRS motor score. Even though a non statistically significant negative correlation between the language screening and other clinical values such as depression and dementia rates could be found for this study, a certain relevance of these control factors cannot be ignored. This is because linguistic disturbances seem to augment with an increase of the disease progression score 'days since disease onset' under control of the disease onset score 'number of CAG repeats', rated on the basis of the augmentation of symptoms associated with HD, as described above. Thus, in sum, study hypothesis 8, 9 and 10, as described in chapter 3.9.6.2, could only be approved partly by showing a statistically significant negative correlation between the performance on the language screening and the UHDRS motor score. Additionally, only a possible influence of cognitive as well as of depressive factors on the language output could be pointed out.
Nevertheless, in sum, results of this study suppose basal ganglia structures to contribute negatively to language changes, as language changes do occur in HD as a disease of the basal ganglia, even in early stages.

Due to the fact that in former HD literature, clinical values such as HD stage, motor, psychiatric and dementia scores, as well as 'days since disease onset' under control of the 'number of CAG repeats' haven't been considered properly, it is hard to compare results of this study with former study outcomes (for details see Table 4 in chapter 3). Only in more recent publications these factors have been considered, supporting results of this study that language changes arise even in early stages of the disease (presymptomatic stage and stage I), when there is mainly striatal degeneration\(^{113}\), (Vonsattel et al., 1985; Aylward et al., 1996; Vonsattel & DiFiglia, 1998; Vonsattel, 1999; Clark, Lai & Deckel, 2002; Aylward et al. 2000; 2003; 2004; Paulsen et al., 2004 as well as Ciarmiello et al., 2006). Thus it seems evident that the basal ganglia must be crucial somehow for language functioning (as evidenced for HD e.g., by Teichmann et al., 2005; 2006; 2007; 2008).

In detail, within this study, linguistic deficits could be found in all domains tested, specifically within semantics, morphology and syntax, in abilities concerning production and comprehension of language. Similar results have also been found in studies by Kennedy et al. (1981), Frank, McDade and Scott (1996), Chenery, Copland and Murdoch (2002) for the domain of semantics, in studies by Gordon and Illes (1987), Illes (1989), Murray (2000), Murray and Lenz (2001), Jensen, Chenery and Copland (2006), Teichmann et al. (2005, 2007, 2008) for the domain of syntax as well as in studies by Ullman et al. (1997a), Teichmann et al. (2005, 2006, 2007, 2008) for the domain of morphology.

However surprisingly, within this study there was no statistically significant difference between language tasks comprising a 'high' (no choosing from given options) versus a 'low' (choosing from given options) 'degree of freedom' and thus being 'primary/complex language functions' (Chenery, Copland & Murdoch, 2002; Jensen, Chenery and Murdoch, 2006). As shown by quantitative and qualitative analysis (for details see chapters 5 and 6) patients have been found to be impaired basically to the same degree in both types of language tasks (primary and complex). These results are in

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\(^{113}\)HD's neuropathology is characterised mainly by striatal changes, as exemplified in chapter 1.4. Nevertheless, to a minor extent, neuropathological changes have been reported for several other brain regions, even outside the subcortical region (Rosas et al., 2003; Douaud et al., 2006; Rosas et al., 2008b; Rosas et al., 2008a; Jernigan et al., 1991; Thieben et al., 2002; Henely et al., 2006; Rosas et al., 2006; Squitieri et al., 2009).
contrast to the study outcomes by Chenery, Copland and Murdoch (cf. 2002: 271), also using the distinction 'high/complex versus low/primary' and stated linguistic disturbances for HD patients mainly in 'complex language tasks' with a 'high degree of freedom' and to a minor degree in 'primary language tasks' with a 'low degree of freedom'.

Hence, it remains puzzling why in this study disturbances also arose within 'low degree of freedom' tasks? Probably, differences in the methodology used by Chenery, Copland and Murdoch (2002) and this study, were crucial for contrasting results. In more detail, within the study by Chenery, Copland and Murdoch (2002), tasks being 'primary linguistic functioning' and being 'low in terms of their degrees of freedom', were defined as tasks focusing on receptive language, including for instance naming and repetition. Against that, within this study tasks being 'primary in linguistic functioning' and being 'low in terms of their degrees of freedom' were defined as tasks with various answer options, provided in terms of competing lexical alternatives, including receptive language (e.g., naming) as well as higher language (e.g., understanding of metaphors). Hence it might be that especially the choosing of the right answer out of various given answer alternatives was problematic for the HD patients tested. Mischoosings due to various answer alternatives could be due to disruptive basal ganglia functions and the consecutive development of a dysexecutive syndrome (Brandt, Folstein & Folstein, 1988; Rosenberg, Soerensen & Christensen, 1995; Lawrence et al., 1996; Lemiere et al., 2004; Peinemann et al., 2005). More specifically, it could be assumed that the disruption of the basal ganglia from the cortex and from various other structures within the cortico-basal ganglia-thalamo-cortical loop (Alexander, DeLong & Strick, 1986; Middleton & Strick, 2000) is responsible for the language disturbances found, also in 'low degree of freedom' tasks. This hypothesis is based on the evidence that the basal ganglia are not only involved in motor functions. Against that they have been reported to participate in various emotional and cognitive functions (cf. Murdoch, 2004: 140), to have a learning-related function (Packard & Knowlton, 2002; Graybiel, 2005) as well as to have a certain role in memory (Packard & Knowlton, 2002), focused attention (Brown & Marsden, 1998) and in pure linguistic tasks (e.g., Crosson, 1985, 1992a; 1992b; Lieberman et al., 1992; Crosson et al., 1997; Pickett et al., 1998; Lieberman, 2001; 2002; Crosson & Haaland, 2003; Whelan et al., 2003a; 2003b; 2004 as well as Lieberman, 2008).

Within this thesis three models have been presented regarding cortico-basal ganglia-
thalamo-cortical circuits, as presented in chapter 1.4.1, as well as in chapter 2.3.5.1 and furthermore within chapter 2.3.5.2. A model, focusing on HD motor disturbances adopted and simplified from (Albin, Young & Penney, 1989; Alexander & Crutcher, 1990; DeLong, 1990; Smith et al., 1998; Mink, 1999; Gutekunst, Norflus & Hersch, 2002), as well as two models specifically referring to language functions involving subcortical pathology, relying on assumptions of this basic model, established by Crosson (1985) as well as by Wallesch and Papagno (1988)\textsuperscript{114}, have been addressed in detail in chapters 2.3.5.1 (Wallesch and Papagno, 1988) as well as in chapter 2.3.5.2 (Crosson, 1985). Regardless the notion that all of these models are hypothetical and are not always addressing language functions per se (in the case of the basic motor model), some hints out of every model may be useful in helping to explain language changes in HD patients tested.

More specifically, the original cortico-basal ganglia-thalamo-cortical model adopted and simplified by (Albin, Young & Penney, 1989; Alexander & Crutcher, 1990; DeLong, 1990; Smith et al., 1998; Mink, 1999; Gutekunst, Norflus & Hersch, 2002; Murdoch 2004; Mink, 2008), even though actually established for explaining motor disturbances in HD patients, describes what happens if the striatum is defective within the cortico-basal ganglia-thalamo-cortical network. This deficiency might also be of importance in order to understand how language changes in HD patients could arise. However, this model shows, that in the example of HD, there is a selective loss of GABAergic neurons in the striatum. This loss is thought to lead to a reduced inhibition of the GPe which would in turn have an even stronger inhibitory projection onto the STN. This might lead consecutively to a reduced excitatory projection from the STN to the basal ganglia.

\textsuperscript{114}As assumptions of the models stated by Wallesch and Papagno (1988) as well as by Crosson (1985) have been made upon patients with acute subcortical lesions it is hard to make a 'one to one’ conclusion for language changes of patients suffering from HD as a neurodegenerative disease. Within the previous chapters the author of this thesis has outlined to be in strong belief that there are no language areas per se, more likely are networks, interconnecting areas within the cortex with each other as well as cortical areas with subcortical structures such as the basal ganglia. As certain pathways within those networks are getting deficient (slowly deficient in neurodegenerative diseases versus abruptly defective in acute lesions), it is very likely that the adaption to those neurological conditions are different for patients suffering from acute lesions against others suffering from slowly progressing degenerative diseases such as HD. The adaptation to those changing neurological conditions is meant in the sense that neuronal pathways look for different ways in order to overcome those deficiencies in subcortical patients versus in patients suffering from acute lesions (for more information on neuroplasticity, see for instance Thompson, 2000). And thus language deficiencies may also be different in patients suffering from acute lesions versus in patients suffering from neurodegenerative diseases. Hence it will unfortunately not be possible at this point to draw any ultimative conclusion. But what we can say for sure is that language is changing in HD patients and that the basal ganglia, as the areas mainly affected in those patients, are somehow crucial for those linguistic changes.
output nuclei. This reduced inhibitory outflow from the GPi and the SNr results in turn in a massive disinhibition of the thalamus and thus consecutively leads to an overactivation of the motor cortex (explained in detail in chapter 1.4.1). Based on these original, motor based assumptions, Wallesch and Papagno (1988) suggested the cortico-striato-pallido-thalamo-cortical loop to take part in the selection of lexical alternatives within language production. As presented before, due to dysfunctions within the striatum (in the case of HD), a loss of the regulatory effect upon the basal ganglia output nuclei is the result which leads then to disinhibitions of the thalamus. As the thalamus gets overactive in turn, this consecutively ends up in an overexcitation of the cortex. Due to this cortical overactivation, various lexical alternatives are simultaneously active and thus linguistically mis-choosings are the result. As such mis-choosings obviously occurred in 'low degree of freedom' tasks, in the sense of choosing the wrong option out of three given linguistic answers, presented on word cards, maybe this model can be seen in a broader sense and thus might be helpful to explain errors found.

Further, as described in chapter 1.4.1.1, patients suffering from HD, experience a dysexecutive syndrome and thus have difficulties with cognitive tasks that require organization, planning and sequencing of events (Brandt, Folstein & Folstein, 1988; Rosenberg, Soerensen & Christensen, 1995; Lawrence et al., 1996; Lemiere et al., 2004; Peinemann et al., 2005). These deficits could further be a possible explanation for problems in 'low degree of freedom' tasks, where sorting and searching for the right answer option, is required. Within the concept of dynamic aphasia (Luria & Tsvetkova, 1968) an inability to select between competing verbal responses (Robinson, Blair & Cipolotti, 1998; Robinson, Shallice & Cipolotti, 2005) has also been reported. Thus, this concept could further account for HD patients' selection deficits in 'low degree of freedom' tasks. However, selection mechanisms have also been ascribed to the basal ganglia (Redgrave, Prescott & Gurney, 1999) as well as to the frontal lobe (Zhang et al., 2004). As interactions between the frontal cortex and the basal ganglia have been reported for working memory (e.g., Frank, Loughry & O'Reilly, 2001) and the basal ganglia have been proven to be the major areas defective in HD, it is likely that deficits in either the basal ganglia themselves or their disruption from the frontal lobe might be crucial for leading to selection deficits as seen in HD patient's 'low degree of freedom' selection-tasks. Against that, language disturbances found in the domain of 'high language functions' could be linked to the basal ganglia model, focusing on language,
stated by Crosson (1985). As the striatum has been reported to have a regulatory function upon the flow of excitatory impulses from the thalamus to motor (Albin, Young & Penney, 1989; Alexander & Crutcher, 1990; DeLong, 1990; Smith et al., 1998; Mink, 1999; Gutekunst, Norflus & Hersch, 2002; Murdoch 2004, Mink, 2008; Murdoch & Whelan, 2009) as well as to cortical language areas (Crosson, 1985), dysfunctions within this subcortical area, have been reported to lead to hyperkinesias in the field of motor disturbances as well as to the inflow of irrelevant language material, in the domain of language. In the model by Wallesch and Papagno (1988) these language intrusions have been described to be similar to a transcortical motor aphasia, respectively to dynamic aphasia (Luria & Tsvetkova, 1968). On the basis of the evidence given in a recent study (Crescentini et al., 2008b), that the concept of dynamic aphasia with a particular impairment in generating sentences and phrases when cued by a stimulus allowing many response options (thus being 'high in degrees of freedom), has also been reported for cases suffering from basal ganglia disturbances, it seems evident that this concept might also be of use in order to interpret HD patient's errors in 'high degree of freedom' tasks. In a similar way Alexander (2006) stated, that impairments in 'complex language' (discourse, open ended utterances, narratives) with a 'high degree of freedom' result in dynamic aphasia-like syndromes (Luria & Tsvetkova, 1968), possibly due to a disruption of the frontal attentional processes. As HD is a basal ganglia disorder characterised by an attentional deficit (see for instance Sprengelmeyer, Lange & Hömberg, 1995; Josiassen et al. 1983; Peinemann et al., 2005; Wahlin, Lundin & Dear, 2007; Lemiere et. al., 2004; as well as Baudic et al., 2006), possibly due to the disruption of the basal ganglia from the frontal lobe, it seems evident that this type of language deficit could also account for deficits in HD patients tested.

In sum, the concept of dynamic aphasia can probably be seen as the linguistic consequence of a dysexecutive syndrome with an inability to plan ahead, resulting into a defective 'inner speech' (cf. Vygotsky, 1962: 44) (e.g., within syntax). Thus, in accordance with the concept of dynamic aphasia, patients within this study, experienced statistically significant difficulties in choosing the right answer out of three given answer choices, as well as within all generative language tasks (e.g., semantic fluency, sentence generation, word formation) where they had to apply pure linguistic rules, requiring functioning 'high linguistic capacities', the ability to plan ahead and an efficient 'inner speech'.

On these deficits we will focus on in detail next.
7.2 Discussion of results – semantic fluency

In terms of semantic fluency, patients performed statistically significantly worse compared to healthy controls, in terms of the total number of items produced within one minute (p < 0.01).

In detail, there was a statistically significant difference between patients and controls in regards to the number of clusterings_corr (in total) (p < 0.05) produced, in favour of the controls. Against that, in terms of switchings_corr, patients switched uncoordinatedly from one category to another, resulting in double as much switchings_corr for patients, in contrast to healthy controls (mean_patients = 2.077 versus mean_controls = 1.568).

Results of the semantic fluency task could be explained, by means of a dysexecutive syndrome (explained in detail in chapter 1.4.1.1). Deficits found are comparable to that seen in frontal lobe patients with problems in their executive functions, having particular difficulty with cognitive tasks that require organization, planning and sequencing of events (Caine et al., 1978; Brandt, Folsetin & Folstein, 1988; Rosenberg, Soerensen & Christensen, 1995; Lawrence et al., 1996; Lawrence, Sahakian & Robbins, 1998; Lemiere et al., 2004; Peinemann et al., 2005).

Clustering has been reported to involve working retrieval mechanisms from the semantic memory, whereas switching has been reported to require intact searching processes through the semantic stores (cf. Randolph et al., 1993: 86; Monsch et al., 1994: 28; Rosser & Hodges, 1994: 1393; Troyer, Moscovitch & Wincour, 1997: 139; Tröster et al., 1998: 302).

Furthermore, switching has been ascribed to underlie the executive functions (e.g., Gurd et al., 2002; Reverberi, Laiacona & Capitani, 2006), being seated in the frontal and basal ganglia areas of the brain. As these areas have been reported to be defective in HD and supposedly lead to the development of a dysexecutive syndrome in HD patients (for details see chapters 1.5.1 and 1.5.2), this syndrome could help to explain the switching particularities of HD patients tested.

In detail, HD patients tested supposedly lacked profound strategies to search the semantic memory and thus ended up switching desperately from one semantic category to another, leading to an augmentation in the number of switchings for HD patients, in contrast to healthy controls. Additionally, it seems evident that this ineffective switching procedure from one semantic category to another, also negatively affected the number
of clusterings_corr and thus resulted in a decreased number of clusterings_corr, compared to controls. These findings are in accordance to study outcomes by Reverberi, Laiacona and Capitani, (2006), who also reported diminished cluster sizes, but an increased proportion of switchings to different subcategories, in patients with frontal lobe pathology.

However, in conclusion, HD patient's fluency particularity (in sum for clusterings_corr and switchings_corr) seems to be mainly due to a general retrieval deficit from the semantic memory (Butters, 1984; Butters et al., 1986; 1987; 1988; Randolph et al., 1993; Rosser & Hodges, 1994), as the result of their dysexecutive syndrome.

7.3 Discussion of results – morphology

Continuing with morphology, patients tested, performed statistically significantly poorer in all tasks provided than their healthy counterparts. In detail, a statistically significant difference between patients and controls could be reported for inflection, including plural formation (p < 0.01). HD patients made a total of 11 errors, compared to healthy controls, who only made 3 errors. Moreover, a statistically significant difference between patients and controls could be shown for derivation/conversion, including providing deverbal and denominal adjectives (p < 0.01), with a total of 75 errors for patients compared to healthy controls, who only made 15 errors. A statistically significant difference could also be shown for HD patients in contrast to healthy controls, for derivation/conversion, including providing deverbal and deadjectival nouns (p < 0.01). More specifically, HD patients made a total of 92 errors, healthy controls contrastingly only 27 errors.

However, it is to question which types of errors did arise and due to which disturbed linguistic mechanisms they occurred? Further it remains puzzling which linguistic theory could account for the errors made? These questions are hard to answer and may remain unanswered at least for the time being. As language functioning is one of the most complex mechanisms occurring within the human body, it is to admit that it is often not possible to give an ultimative explanation why certain forms of errors occur and others don't. Because nobody yet knows exactly what is really going on in our brains when we use language, thus all our explanations are worse or better descriptions of possible
language mechanisms, but will always remain only assumptions or sometimes even guesses.

Unfortunately until now research in the field of language changes in HD did not yet test these morphological areas as profound as done within this study. Further, no linguistic theory has been used yet to explain the errors found. Thus it is not possible to refer to any publications or explaining theories at this point in order to support results of this study.

However, there has been an attempt within this thesis. A trend could be noted that some errors found fit into the linguistic concept of 'Natural Morphology'. In order to follow subsequent explanations in regards to morphological errors made, a brief introduction to this linguistic concept will be given next.

7.3.1 The concept of 'Natural Morphology'

The concept of 'Natural Morphology' arised in the 70ies of the last century, as the continuation of the theory of 'Natural Phonology', stated by Stampe back in 1979. The precusors of Stampe's theory of 'Natural Phonology' can mainly be found within the structuralistic 'Prague Circle', including Nikolai Troubetzkoy (1967) and Roman Jakobson (1941). Within Jakobson's (1941) work, several theses have been strengthened, later refined within the concept of 'Natural Phonology' and consecutively also within the concept of 'Natural Morphology'. In detail, this concept assumes that the acquisition of the phonological system by kids, as well as the loss of the same, due to e.g., aphasic disturbances, follows a chronological order, hold for any speaker of any language of the world. The fact that a fixed order is somehow inherited in language acquisition as well as in language loss, is explained by the 'principle of least effort' (cf. Jakobson, 1972: 20ff). This principle states that those speech sounds which require the least physiological effort for production (the acquisition of wide vowels is accompanied by the acquisition of forward consonants as for instance in words like 'Mama' and 'Papa'), are acquired the first by children, no matter which language of the world they learn and are lost the last in the course of language pathology. This order of acquisition is governed by the 'law of irreversible solidarity' or also known as 'implicational law' (see Jakobson, 1972: 51ff). Briefly, this law states that the acquisition of front sounds presupposes the acquisition of back sounds, but not vice versa. As a
prove of this theory the fact can be seen that within the languages of the world, there is no language which has back consonants without containing front consonants. Furthermore, consonantal as well as vowel oppositions which occur very rarely in the languages of the world, are thus among the latest phonological acquisitions of the child in first language acquisition and are thus the first ones to get lost in aphasia. Even though Jakobson did not make use explicitly of the terms 'markedness'\textsuperscript{115} and 'naturalness' in his 1941 work, it is clear nevertheless, that his concept implies universal preference for certain phonological oppositions\textsuperscript{116} over others, and in this sense, refers to more or less marked phonological oppositions (more marked oppositions are acquired the last in the process of language acquisition and are getting lost the first in the course of language disturbances) (for a more detailed information regarding the concept of markedness, within the concept of structuralism, see for instance Battistella, 1996: chapter 2). In the same sense, these values which are acquired first by kids and are getting lost the last in the course of language disorders, can thus be regarded as natural and consecutively also as unmarked.

Stampe (1979) as well as Donegan and Stampe (1979) refined the considerations by Troubetzkoy (1976) and Jakobson (1941) and stated the theory of 'Natural Phonology'. Within this theory, a differentiation has been made between innate, natural phonological processes, in contrast to learned phonological rules.

\textsuperscript{115}However, Jakobson already used this term in a previous work. In detail, the first published application of the marked/unmarked relation goes back to Jakobson's 1932 article 'The Grammatical Concepts of the Russian Verb' (reprinted in 'Roman Jakobson Selected Writings II. Word and Language' (1971) (cf. Jakobson, 1971: 136ff).

\textsuperscript{116}The idea of phoneme oppositions, has already been stated previously in Troubetzkoy's 'Grundzüge der Phonologie' (1976). Within this work, Troubetzkoy regarded the neutralizations of oppositions between phonemes as the unmarked status. These considerations about markedness have been continued within the concept of 'Natural Phonology' by Stampe (1979) and have been expanded moreover for the label 'naturalness'.
Table 54 shows the main differences between phonological processes and phonetic rules, summarised from Donegan and Stampe (1979: chapter 6)

<table>
<thead>
<tr>
<th>phonological processes</th>
<th>phonological rules</th>
</tr>
</thead>
<tbody>
<tr>
<td>Innate, natural, universal phonological trend</td>
<td>learned</td>
</tr>
<tr>
<td>phonetically motivated (enhance ease in articulation)</td>
<td>not phonetically motivated (do not enhance ease in articulation)</td>
</tr>
<tr>
<td>present limitations of speakers production</td>
<td>do not present limitations of speakers production</td>
</tr>
<tr>
<td>no semantic/grammatic function (not morphologically motivated)</td>
<td>semantic/grammatic function (morphologically motivated)</td>
</tr>
<tr>
<td>involuntarily, unconscious</td>
<td>conscious</td>
</tr>
<tr>
<td>apply to slips of the tongue and loanwords</td>
<td>do not apply to slips of the tongue and loanwords</td>
</tr>
<tr>
<td>optional or obligatory</td>
<td>only obligatory</td>
</tr>
</tbody>
</table>

It is well known by now that within language acquisition the child passes certain stages. In the 'babbling' stage the child is capable of producing all conceivable sounds of the world's languages. Within a pre-linguistic stage this ability disappears, as the child is exposed to (mostly) one native language. The acquisition of sounds of the native language follows a regular order, as previously mentioned by Jakobson (1941), no matter which language is acquired. Thus, every child beginning to acquire his or her native language is running also somehow through the entire corpus of phonological processes of the world's languages. These phonological processes are innate as they occur in all languages of the world and have the purpose to minimize articulatory agility (cf. Stampe, 1979: 9). An example of an innate phonological process is for instance the nasalization of vowels preceding nasal consonants, the devoicing of obstruents, or the unrounding of frontal vowels (see also Donegan & Stampe; 1979: chapter 6 or for a brief summary also Wurzel, 1989: 1-8). But as the child acquires his/her native language, specific phonological processes are filtered out, based on the child's specific linguistic environment. This adaptation of phonological processes to the phonological structure of the individual language is performed by means of language specific phonological rules, mostly motivated by morphology and thus referred to as morphonological rules such as the German umlaut in 'Haus – Häuser'. As these rules are of conventional character and mostly do not ease articulation, they have to be learned by the child. Another example for a learned phonological rule is the devoicing of final obstruents in German (/hunt/ in Hund).
That this rule is language specific and has to be learned can be seen when German
speakers try to learn English and struggle pronouncing final voiced obstruents
(/dog/ in dog) (cf. Donegan & Stampe, 1979: chapter 6; or for a brief summary also

Since the mid seventies there have been some attempts to extend the considerations of
the theory of 'Natural Phonology' also into the field of morphology, mainly driven by
Dressler's (1977a), Mayerthaler's (1981) and Wurzel's (1989) 'Natural Morphology'.
With the same considerations, as previously strengthened mainly by Jakobson (1941)
and Stampe (1979), Mayerthaler (1981) pointed out within his monograph that

"Nicht alle morphologischen Strukturen sind in natürlichen Sprachen gleichermaßen
verbreitet, nicht alle morphologischen Prozesse und Strukturen werden von Kindern
zur selben Zeit erworben, nicht alle morphologischen Strukturen werden vom
Sprachwandel gleich affiziert, nicht alle morphologischen Prozesse und Strukturen
werden von Sprachstörungen gleichermaßen in Mitleidenschaft gezogen, nicht alle
morphologischen Strukturen sind gleichermaßen leicht dekodierbar [...]“ (Mayerthaler, 1981: 2)

Form these assumptions it can be summarised that a morphological process or a
morphological structure is natural if "a) er/sie ist weit verbreitet ist und/oder b) relativ

In the theory of 'Natural Phonology', natural phonological processes were regarded to
make sounds easier to pronounce. Similarly, within 'Natural Morphology',
morphological simple is understood to be easily accessible (easily perceived and
retrieved). This is for instance stated by Dressler (cf. 2005: 267), as he defined natural
to be synonymous to cognitively simple, easily accessible, elementary and therefore
universally preferrable. Moreover, naturalness within the concept of 'Natural
Morphology' is considered to be the opposite to markedness. A morphological
phenomenon is thus less marked the more natural it is and vice versa (cf. Mayerthaler,
1981: 2).

It is important to note however, that the concept of markedness values, as stated within
the theory of 'Natural Morphology', is not part of the language system itself.
Within the concept of 'Natural Morphology', Mayerthaler (1981) distinguished three types of markedness, governed by Peirce's semiotic trichotomy of signs\(^\text{117}\) (summarised and simplified from Wurzel, 1989: 8ff):

1) semantic-categorial markedness (= sem values)
   This kind of markedness reflects how far morphological categories reflect prototypical speaker properties; the less semantically marked a category is, the more prototypical are speaker properties (singular is less marked than plural, first person is less marked than other persons etc.)

2) symbolization markedness (= sym values)
   This kind of markedness reflects how optimal symbolized a category is; the symbolization of a category is optimal or maximally natural, if the category is: 2a.) constructionally iconic, 2b.) constructionally uniform and 2c.) constructionally transparent

3) derived markedness (= m values)
   On the basis of sem- and sym-values, the m-values are determined. Hence, if a semantically unmarked category is symbolized unmarked, the derived form is also unmarked. But if the unmarked category is symbolized marked, then the resulting form is also marked.

Going back to 2a.) the concept of constructional iconicity can be explained as follows. Based on Peirce's definition of symbol, icon and index, icons can be referred to as the most natural signs. This is because there is a direct, causal connection between the sign and the object. Contrastingly, symbols can be regarded to be the least natural, as there is no logical connection between the sign and the object it refers to. Within the concept of 'Natural Morphology' those morphological forms which can be regarded as constructionally iconic, are those where there is a causal, logical and

\(^{117}\)Peirce divided signs according to their relation to objects and thus pointed out a tridivision of signs into icon, index and symbol. In detail, a symbol is defined as standing for a particular object or kind of object only in virtue of a convention (cf. Fitzgerald, 1966: 40: 45) This means that a symbol has no logical connection to the object it refers to (e.g., a white dove stands for peace). When a „sign signifies its object solely by virtue of being really connected with it“ (Peirce, 3.361 in Fitzgerald 1966: 45), we call such signs an index as there is a direct link, a causal conjunction as well as a logical, informative connection between the sign and the object it refers to. An example of an index is smoke as a sign of fire or all traffic signs have indexical function. „The third case is where the dual relation between the sign and its object is degenerate and consists in a mere resemblance between them. I call a sign which stands for something merely because it resembles it, an icon“ (Peirce, 3.362 in Fitzgerald, 1966: 40-45). An icon sign is thus a sign that resembles something, such as photographs of people. For a further reading on Peirce's concepts of semiotics see for instance also Greenlee (1973: 70-98).
transparent connection between the grammatical form and its function. Furthermore, constructional iconicity can have a strong and a weak characteristic. Within the strong characteristic there is a very broad differentiation between featureless (der Junge) and feature bearing (die Junge -n) coding. Within feature bearing coding a differentiation can be made between additive coding (adding of segments as in der Junge – die Junge-n) and modulatory coding (Mutter – Mütter; umlaut; gehen – ging; ablaut). It is also possible that additive and modulatory codings are realized together within one word (Haus – Häuser). Within additive coding a further differentiation can be made between segmental additive coding (within segmental additive coding a further differentiation can be made between more segmental (-issimus) and less segmental (-ior) coding) and modulatory additive coding (lengthening of segments; domus – domūs). (cf. Wurzel, 1989: 11).

In general, segmental additive processes dominate in languages of the world, with the consideration behind „[w]as semantisch „mehr“ ist, sollte auch konstruktionell „mehr“ sein“ (Mayerthaler, 1981: 25). This assumption can further be approved as languages of the world rather have a feature bearing plural coding than a feature bearing singular coding, as well as they rather have a feature bearing nonpresent coding than a feature bearing present coding (cf. Wurzel, 1989: 11). Mayerthaler summarized that the basic categories singular, positiv, indicative, present tense, nominative, masculine, tend towards featureless coding (cf. Mayerthaler, 1981: 28).

Based on these considerations the following hierarchy of iconicity can be assumed. A morphological process/category is maximal iconic, if it is constructionally iconic and it is segmental additive coded (boy – boys). A morphological process/category is less than maximally iconic, if it is constructionally iconic and it is modulatory additive coded (domus – domūs). A morphological process/category is minimal iconic, if it is constructionally iconic and modulatory coded (goose – geese). A morphological process/category is non iconic, if it is non constructionally iconic (sheep – sheep). A morphological process/category is contraiconic if the assymmetry of semantic markedness values is mapped onto an inverse assymmetry of symbolization (more form for singular than for plural; Elternteil = singular versus Eltern = plural) (cf. Wurzel, 1989: 11).
Going back to 2b.) the concept of constructionally uniformity can be explained as follows.

„Die Symbolisierung/Endkodierung eines Paradigmas Pi ist uniform, wenn Pi gemäß 'one function – one form' organisiert ist, andernfalls mehr oder minder nicht-uniform“ (Mayerthaler, 1981: 34)

Going back to 2c.) the concept of constructionally transparency can be explained as follows.


Nevertheless, according to Mayerthaler (1981: 103) the „Hauptfaktor, der über die Natürlichkeit morphologischer Einheiten entscheidet“ is perception, respectively the ease of perception. In this sense external inflection is regarded to be more natural than internal inflection and prefixes as well as suffixes are more natural than infixes.

In regards to morphological operations, the following differentiations can be made between (summarised and adapted from Mayerthaler, 1981: 109ff) category-constant processes, which do not change the basis of lexical categories (flections; ich kaufe – ich kaufte), category-changing processes, which change the basis of lexical categories (derivations; nation – national) and conversions or so called 0-processes, which change morphological categories to a phonological identical output (to bridge – the bridge).

Furthermore, additive processes can be differentiated from non-additive processes. Additive processes include all morphological operations in which segments are added also in the sense of adding length (in gemination). Thus again the distinction can be made between segmental additive (affixation and reduplication) and modulatory additive processes (elongation of segments of the basis, gemination).

Inverse to additive processes are so called non-additive processes, including subtractive, modulatory, 0-processes as well as contaminations. Examples of subtractive processes are cuttings (Omnibus to Bus) as well as acronyms and abbreviations (United States of America = USA). Examples of modulatory processes are ablaut (singe – sang) or umlaut (‘Haus’ – ’Häuser’). Examples of 0-processes are such as (sheep – sheep).

Examples for contaminations also called blendings are (smoke & fog = smog; German & English = Genglish; Europa & Asien = Eurasien) (cf. Mayerthaler, 1981: 109ff).
Because „[e]ine eindeutige Zuordnung zwischen Form und Funktion [...] perzeptiv leichter erfaßbar [ist] als eine einmehr- bzw. mehreindeutige Zuordnung [...]“ (Mayerthaler, 1981: 35), it is evident that all non additive processes have very low iconic functional potentials (cf. Mayerthaler, 1981: 112ff). In simple words, there is a case of morphological naturalness whenever a formal 'more' corresponds to a semantic 'more' (cf. Wurzel, 1989: 67).

In terms of markedness, a morphological process with respect to its symbolization is unmarked if the process is additive, maximally unmarked if the process is segmental additive, marked if the process is nonadditive as well as maximally marked if the process is subtractive. Based on these basic assumptions, Mayerthaler (1981) developed the concept of productivity. Under the perspective of 'Natural Morphology', a process is the more productive, the more transparent, iconic and uniform it is (cf. Mayerthaler, 1981: 124ff). Consecutively it is also clear that all forms which are transparent, uniform, iconic, unmarked and natural are those, which freely apply to new forms and are thus called productive (cf. Dressler, 2008: 457).

The important thing to keep in mind, which differentiates the concept of 'Natural Morphology' from e.g., generativisms is, that productivity is seen as a gradual concept. The dichotomous view of 'all or nothing' is abandoned within this concept (evidence from e.g., German plural morphemes such as Blum-en, Beet -e, Gärt-en, Gläs-er; it would be hard to answer with only generativistic assumptions, that one of these forms presents the default) (cf. Dressler, 2008: 459).

Within the theory of 'Natural Morphology' a specific concept – the concept of 'language specific normalcy' – is important to consider moreover in order to follow susequent explanations of morphological errors made within this study.

Due to the fact, that morphological processes are expressed from language to language differently, by language specific morphological forms (the plural is expressed in different ways from language to language, e.g., Blume – Blumen versus flower – flowers), morphological markers are of strictly language specific nature and thus do not have a universal basis.
However, there are various degrees of 'language specific normalcy' (cf. Wurzel, 1989: 66).

"The notion of normalcy introduced by us is related to those morphological phenomena that are felt to be 'more normal' than others by speakers, and according to the patterns of which, the 'less normal' morphological phenomena are unconsciously changed. In this general sense, the morphological normalcy of individual languages must be considered as a form of morphological naturalness if this term is not to lose its theoretical and empirical justification" (Wurzel, 1989: 69)

Studies have shown that some inflectional forms are regarded to be 'better' (more natural) forms, than others. Thus for instance in German, weakly inflected strong verbs (e.g., er *fechtete) are better accepted by speakers (even though ungrammatical), than strongly inflected weak verbs (e.g., er *tramm). The same holds for some plural forms of German. For strong masculine nouns (e.g., those with e- or 0-plural), ungrammatical plurals with umlaut (die *Hünde) are more acceptable than ungrammatical plural forms without umlaut (die *Flusse). For the neuters the opposite holds; in this case ungrammatical plurals without umlaut are more acceptable (die *Floße), than ungrammatical plurals with umlaut (die *Böte) (cf. Wurzel, 1989: 65f). This different status of competing inflectional classes ('better' and 'worse' examples), as just presented, can be characterised by the notion of 'language specific normalcy'.

As stated above, universal morphological naturalness favours the development and also the maintenance of inflectional systems, which follow the principles of iconicity, uniformity and transparency. In comparison, morphological normalcy, always favours the language specific characteristics of inflectional systems, irrespectively of the universal properties of such systems (e.g., *Hünde; introduction of the umlaut). There seems to be no general reason why sometimes the umlaut is more acceptable (respectively also more normal) than in other circumstances (cf. Wurzel, 1989: 69). Thus Wurzel (1989) concluded for these forms that "an explanation based on the notion of universal properties of inflectional systems seems to be out of place [herein]. It might seem that mere accident is at work here" (Wurzel, 1989: 69).
7.3.2 Discussion of morphological errors – in detail

However, in regards to results shown in chapter 6, it could be concluded that HD patients are not primarily morphologically impaired, as they are aware of morphological rules per se but experience various uncertainties in regards to the application of these rules. Due to these uncertainties the wrong morphological rules, even though being productive\textsuperscript{118} morphological rules for German per se, have been applied to the wrong words (ein Hund – viele *Hünde, ein Känguru – viele *Kängurusse, gut – die *Gutheit, Furcht – *furchtig, streben – die *Strebhaftigkeit). An impairment in morphological rule application, even though described for the domain of inflectional morphology, has also been mentioned for HD patients in the study by Ullman et al. (1997a) as well as by Teichmann et al. (2005 & 2006)\textsuperscript{119}. Theories of an impaired rule application within this study, are based on errors found in plural formation, providing deverbal and denominal adjectives as well as in providing deadjectival and deverbal nouns, as will be exemplified in detail now.

Within plural formation, all plural morphemes attached were productive plural morphemes of German, but the wrong plural morphemes have been attached to the wrong words (ein Bär – viele *Bär-e, ein Känguru – viele *Känguru-ss-e) (A list of all wrong plurals, separated for patients and controls, is given in Appendix IX).

Errors within providing deverbal and denominal adjectives seemed to be derived by similar mechanisms, as erroneous plural formations. Again, patients made the impression to know the productive adjectival morphemes of German but the morphological rules which give us the information to which words these morphemes have to be attached in order to make an existing German adjective out of a given word, seemed to be defective. This resulted into the production of neologisms, such as Buckel – *bückelig, Furcht – *furchtig, Mutter – *muttrig, Glück – *glückig (the total list of erroneous productions of providing deverbal and denominal adjectives is given in Appendix X). Even though neologisms have been produced, interestingly however,

\textsuperscript{118}The productivity of a morphological process (whether inflectional or derivational) has to do with how much (or, in the limiting case, whether) it is used in the creation of forms which are not listed in the lexicon. For example, the affix -en in oxen is not used very often by anyone in the formation of new words, and is correspondingly felt to be of very low productivity. Contrastingly the suffix -s is frequently used in the formation of many plurals by many people, and is thus felt to be of high productivity (cf. Bauer, 2005: 315). Thus productive morphological patterns are considered as those that freely apply to new words (cf. Dressler, 2008: 457).

\textsuperscript{119}Nevertheless it has to be stressed that these results are in contrast to the doubtful dual route model of lexicon and grammar, as stated by Ullman back in 1997a (for details see chapter 3.1.9.5).
the suffixes applied were always existing German adjectival suffixes (in most contexts the overapplication of the productive -ig adjectival suffix), which have nevertheless just been applied to the wrong words.

These results are similar to errors described by Dressler (cf. 1977a: 45) as well as by Dressler (cf. 1977b: 62) for German speaking aphasics who also evidenced the overapplication of the productive -ig suffix.

Furthermore, HD patients provided German correct, but not required derivations (e.g., achten – Achtung) as well as correct but not required nominal compoundings (e.g., Wind – Windrad, Holz – Holzmaserung, Mutter – Muttertag) within providing deverb al and denominal adjectives. In order to make these productions, morphology itself has to be intact. What seems to be defective is again the use of morphological rules.

Neologistic errors within providing deadjectival and deverb al nouns could also be explained by a defective morphological rule application. Again, the morphological rules per se, which give us the information to which words certain morphemes have to be attached, in order to make an existing German noun out of a given word, seemed to be defective in HD patients. Consecutively e.g., neologisms were the result, such as gut – die *Gutheit, nass – die *Nassheit, heiter – die *Heiterung (the total list of erroneous productions is given in Appendix XI).

However, it still needs to be questioned how these errors could be explained and which theory could account for the errors found?

For morphology in general, there is a trend that errors made could be fit into the theory of 'Natural Morphology' (Dressler, 1977a; 1997b; Mayerthaler, 1981; Wurzel, 1989). In accordance to evidences for this theory from synchrony, diachrony, first language acquisition, second language acquisition, slips of the tongue, acceptability ratings and aphasia, (Dressler, 1977a; 1977b; Mayerthaler, 1981; Dressler et al., 1987; Wurzel, 1989; Dressler & Ladányi, 2000; Dressler, 2003; 2005; 2008), HD patients tended to reduce marked forms (segmental modulatory, e.g. umlaut; nass – die Nässe) towards unmarked, more transparent and more natural forms (segmental additive forms, e.g., addition of the nominal suffix -heit; nass – *die Nassheit). Marked within this concept is understood as equal to unnatural, defined upon the semiotic criteria of iconicity. Thus the more iconic a form is, the more natural and the less marked it is.

However, following the concept of 'Natural Morphology' HD patients tended to avoid unnatural plural forms and thus produced natural forms instead, by adding segments
(ein Bär – viele *Bären, ein Känguru – viele *Kängurerusse). Also formations such as (ein Hund – viele *Hünde) appeared, which could be explained by language specific 'morphological normalcy' (Wurzel, 1989), as described previously in chapter 7.3.1.

In accordance to the assumptions of the theory of 'Natural Morphology', within

providing deverbal and denominal adjectives, the overapplication of the productive -ig suffix occurred and resulted in the production of neologisms (e.g., Buckel – *bückelig, Furcht – *furchtig, Mutter – *muttrig, Glück – *glückig; the total list of erroneous productions is given in Appendix X), in some cases again supporting the assumption that more marked forms (e.g., with umlaut; Mutter – mütterlich) have been changed to less marked, even though erroneous forms (e.g., segmental additiv forms; Mutter – muttrig). Errors found within providing deadjectival and deverbal nouns furthermore seemed to follow the pattern of 'Natural Morphology'. More marked forms (e.g., segmental modulatory forms with umlaut; gut – die Güte) have generally been changed to more transparant, more natural, less marked forms, resulting in neologisms such as gut – die *Gutheit, nass – die *Nassheit (the total list of erroneous productions is given in Appendix XI). Interestingly however, for providing deverbal and denominal adjectives as well as for providing deadjectival and deverbal nouns, the most frequently occurring error category were non responses. What could be inferred from this result? Supposedly, HD patients in general, had a lot of troubles performing this task. But why? Maybe within providing deverbal and denominal adjectives some of the words presented have been unappropriate test materials per se, as there were non responses for only certain words in contrast to others (the total list of words resulting in non responses is provided in Appendix X). As patients also needed a lot of help (the total list of words requiring help, is provided in Appendix X), thus it is possible that the task itself comprised a too high cognitive load and thus couldn't be performed correctly.

In contrast to this, within providing deadjectival and deverbal nouns, non responses were almost to occur to the same degree for all 10 words provided. Prompts in terms of help didn't have a positive impact on the patient's performance either. Due to the fact that also for healthy controls the number of non responses for this task was quite high, it

120However, unfortunately not all errors found, fit into the explanations of the concept of 'Natural Morphology'. Contrastingly, some even contradict this model (eine Katze – viele *Katze, ein Känguru – viele *Känguru) (no change to segmental additiv form). Nevertheless, as mentioned before, sometimes it is simply impossible to find evidence for the plausibility of every erroneous production.

As language functioning is one of the most complex mechanisms occurring within the human body and nobody yet knows exactly what is really going on in our brains when we use and receive language, all explanations we give are worse or better descriptions of possible language mechanisms. However, they always remain assumptions or sometimes even guesses.
seems evident that this task may not have been best to test this language function and thus can be considered as a limitation of this study. Even though it is not certain if the HD patients had difficulties with providing deverbal and denominal adjectives as well as with providing deadjectival and deverbal nouns per se or not, deficiencies with performing derivational/conversional morphology could be reported nevertheless as evidenced by various error patterns presented above. Thus, for morphology in sum it can be concluded that in accordance with previous findings (Ullman et al., 1997a; Teichmann et al., 2005; 2006), morphological rule application seems to be defective in HD patients. Against that, the morphological system itself remains intact. This dissociation has been evidenced by a high number of neologism errors due to the application of the wrong suffixes, but there remained the ability to produce complex compoundings and German correct, but not required conversions/derivations. As the basal ganglia have been reported to be the areas mainly affected in HD (see for instance Vonsattel et al., 1985; Aylward et al., 1996; Vonsattel & DiFilgia, 1998; Vonsattel, 1999; Aylward et al., 2000; Clark, Lai & Deckel, 2002; Aylward et al., 2003; 2004; Paulsen et al., 2004 as well as Ciarmiello et al., 2006) as well as to participate in linguistic rule application (Ullman et al., 1997a; 1999; 2001a; 2001b; 2001c; 2004; 2005; Teichmann et al., 2005; 2006; 2007; 2008), morphological deficits in terms of a defective linguistic rule application may thus be due to basal ganglia dysfunctions within the cortico-basal ganglia-thalamo-cortical circuit. As the striatum has also been reported to have a regulatory function upon the flow of excitatory impulses from the thalamus to cortical language areas (Crosson, 1985), dysfunctions within this subcortical area, have been reported to lead to the inflow of irrelevant language material in the field of linguistics. The inflow of irrelevant language material could thus possibly account for the wrong application of morphological rules, as reported for HD patients within this study. Similarly, Wallesch and Papagno (1988) suggested the cortico-striato-pallido-thalamo-cortical loop to take part in the selection of lexical alternatives in language production. As dysfunctions within the striatum (which is the case in HD) cause a loss of regulatory effect upon the basal ganglia output nuclei, disinhibitions of the thalamus are the results which consecutively end up in an overexcitation of the cortex. Due to this cortical overactivation, various lexical alternatives are simultaneously active and thus linguistic mis-choosings, in terms of choosing the wrong affixes for the wrong words, in morphological rule application, have been the result.
7.4 Discussion of results – syntax

In terms of syntax, patients performed statistically significantly poorer compared to their healthy counterparts, in all tasks provided. This result conforms with previous study outcomes regarding the syntactic performance in HD patients (see for instance Gordon & Illes, 1987; Illes, 1989; Murray & Stout, 2000; Jensen, Chenery and Copland, 2006 as well as Teichmann et al., 2007; 2008) also reported syntactic disturbances in HD patients.

In detail, a statistically significant difference between patients and controls could be reported for syntax_production, including sequencing of sentence parts (= syntax I) ($p < 0.05$) and guided syntactic production (= syntax II) ($p < 0.05$) as well as for syntax_comprehension ($p < 0.01$), including the eight subtasks of decoding thematic roles (= syntax III).

Again it is to question which types of errors did arise and due to which disturbed mechanisms they occurred? Further it remains puzzling which theory could account for the errors made? Similarly as previously discussed for morphology, former research did not test any of the mentioned syntactic aspects as profound as administered within this study. Further, no linguistic theory has been used yet to explain the errors found. Thus it is not possible to refer to any publications or explaining theories at this point in order to support results of this study.

However, there has been an attempt within this thesis. In linguistics a hypothesis exists that tries to explain the deficits in comprehending semantically reversible sentences. This hypothesis is referred to as 'Trace Deletion Hypothesis', formulated by Grodzinsky back in 1990. As comprehension of semantically reversible sentences has been tested in depth also in HD patients within this study, and as various errors occurred within this task, it seems important to briefly refer to this hypothesis at that point, as some errors could be explained by suggestions of this linguistic concept.
7.4.1 The hypothesis of 'Trace Deletion'

The 'Trace Deletion Hypothesis' by Grodzinsky (1990) can be seen as one of the first well grounded attempts to explain the core comprehension deficits in Broca's aphasics as first described by Caramazza and Zurif back in 1976. Within their study Caramazza and Zurif (1976) could show that Broca's aphasics have troubles comprehending semantically reversible relative clauses, in which both nouns can either be the agent or the patient. In other words, Broca's aphasics did not know who was doing what to whom. Thus in passive as well as in object-relative constructions Broca's aphasics tested performed at chance. Contrastingly these patients knew who was doing what to whom in active as well as in subject-relative constructions, leading to a performance above chance. At this time this was a surprising finding, because until then comprehension has been regarded to be spared in Broca's aphasia. However, Caramazza and Zurif (1976) attributed this deficit in comprehending semantically reversible sentences to the non-canonical\textsuperscript{121} nature of these syntactical constructions.

Grodzinsky (1986) tried to explain the agrammatic syntactic comprehension deficits in Broca's aphasics via a difficulty assigning theta-roles to moved syntactic elements. This explanation is grounded on Grodzinsky's (1990) hypothesis of 'Trace Deletion', which grounds theoretically on Chomsky's (1981) theory of 'Government and Binding', which in turn is influenced by Chomsky's 'Transformational Grammar' (Chomsky, 1965).

\textsuperscript{121} A major distinction can be made between canonical and non-canonical sentences. The canonical form is in each language the most typical word order (e.g., in English = SV0). From this canonical word order non-canonical forms (e.g., passive voice) are derived. Canonical sentences are active sentences (Mum = subject & agent; eats = verb & action; an apple = object & patient) and sentences with subject relatives (The apple = subject & agent; which = object & patient; is lying = verb & action; on the table = object & locative) with the default SVO word order for e.g., English. Non-canonical sentences on the other hand include passive sentences (The apple = subject & patient; is eaten = verb & action; by mum = object & agent) and sentences with object relatives (The apple = object & patient; which = object & patient; mum = subject & agent; lay = verb & action; on the table = object & locative), in which the default subject-verb-object word order is inverted. In non-canonical sentences the subject can be both, the agent and the patient. In contrast to this, canonical sentences have the default SVO structure, where the subject is always the agent and the object always the patient. In order to understand non-canonical sentences, syntactic analysis is needed. Within syntactic analysis the decoding of the thematic roles of sentences is proceeded and consecutively syntactic movements can be understood. Contrastingly, canonical sentences can be processed by using word-order information (heuristic default strategies that the subject is always the agent in a sentence) to reconstruct the thematic roles of the sentence. Thus it is not surprising that in general the processing of non-canonical sentences with a word order contradicting the default, is harder than the processing of canonical sentences.
Very briefly, transformations, as they were also in 'Transformational Grammar', are still the most important part within Chomsky's (1981) theory of 'Government and Binding'. In this theory, syntax is organised in several levels of representation, that are related to one another by rules (specifically called transformational rules/transformations/movements). These transformations, take place when the rules of syntax are applied. The theory of 'Government and Binding' distinguishes a surface structure (= S-structure) (generated by the rules of syntax), the sentence as we see it, from an underlying deep structure (= D-structure). Further a phonetic form (= PF), including the sound structure, derived from the S-structure, as well as a logical form (= LF), including the semantic information, also derived from the S-structure, are hypothesized. The rules of syntax generate a transformation from the D- to the S-structure and all the moved elements leave so called traces (= t) behind, indicating their places of origin within the D-structure (The boy is kissing the girl. > The girl is kissed by the boy). According to the theory of 'Government and Binding' the NP 'The girl' in the D-structure is represented after the verb 'is kissing' (The boys is kissing the girl). In the derived passive S-structure the NP 'The girl' is transformed into the position of the subject. In turn a trace of the argument of the verb (= in this case the NP 'The girl) is left behind, to indicate its previous position in the D-structure (cf. Chomsky, 1981: chapter 2). These traces are especially important in the case of passive and reversible sentences where the decoding of thematic roles/theta roles is necessary in order to correctly comprehend the sentence. As mentioned earlier within this work, theta roles describe the underlying relationship within a sentence that a participant has with the main verb in a clause. If within a sentence a participant 'the boy' kisses another participant 'the girl', then the semantic role of 'the boy' is that of the agent of this event and the semantic role of 'the girl' on the other hand is that of the patient (cf. Payne, 1997a: 47). In a passive clause, the theta roles are changed on the S-structure (The boy kisses the girl. > The girl is kissed by the boy). In order to correctly comprehend this sentence the trace is needed which has marked the transformation of the NP 'the girl' (object, patient) from the end to the beginning of the sentence, into the position of the subject. Only with the help of this trace (indicating the place of the transformed element in the D-structure; the girl = patient), according to the theory of 'Government and Binding' (Chomsky, 1981), we are capable to correctly interpret that 'the girl' always holds the thematic role of the patient, no matter in which position of the sentence it is placed.
According to Grodzinsky's (1990) 'Trace Deletion Hypothesis' these traces are missing in agrammatic patients, due to brain dysfunctions in the course of Broca's aphasia. In more detail, Grodzinsky argues that Broca's area is essential for the indexation of traces. Thus damage to Broca's area in the course of Broca's aphasia, results in the inability to process traces and is thus responsible for the sentence comprehension deficits observed in agrammatic Broca's aphasics. Consecutively the decoding of thematic roles, especially in passive and semantically reversible sentences, where transformations of elements take place, is not possible anymore (The boy is kissing the girl. > The girl is kissed by the boy) in this patient group, resulting in errors or performances at chance. Against that, canonical sentences in active voice, without transformations of elements can still be comprehended above chance. The 'Trace Deletion Hypothesis' tries moreover to explain how errors and chance performances in agrammatic patients arise. Given, that for instance the first NP of a sentence is lacking the trace due to the effect of syntactic transformation (The boy = agent; is kissing = action; the girl = patient > The girl = is transformed in the passive clause to the front of the sentence; is kissed = action by the boy = agent), the assignment of the thematic role to this NP in agrammatic patients happens by ways of a heuristic word order principle (= default strategy) that in canonical sentences (= presents the default) the first NP always is the agent of the sentence (The girl = *agent; is kissed = action; by the boy = agent), or by hints of passive morphology (by- phrase in English). This leads to the wrong representation of two NP's within one sentence, each assigned the thematic role of an agent. Thus agrammatic patients have to guess (chance performance) which of the two NP's is the agent of the sentence (summarised from Grodzinsky, 1986; 1990; 1995a; 1995b).

However, Grodzinsky's (e.g., 1986; 1990) strict dissociation that Broca's aphasics display better than chance performance in active (semantically reversible) sentences, but below chance, or worse than chance performance in passive (semantically reversible) sentences, has been criticised by several scientists. Schwartz, Saffran and Marin (1980) were among the first to show that the core hypothesis of the theory of 'Trace Deletion', that Broca's aphasics only show deficits in the comprehension of passive and object- relative clauses but above chance performance with active and subject- relative clauses, cannot always be confirmed.
Only two out of five agrammatic subjects tested, performed according to the theory of 'Trace Deletion', with above chance performance on active sentences and at chance/below change performance on passives. This divergent finding has been confirmed in various other studies as well.

A meta-analysis by Berndt, Mitchum and Haendiges (1996), using studies of aphasic sentence comprehension between 1980 and 1993, revealed that both active and passive sentences could be comprehended better than chance, that both, active and passive sentences could be comprehended no better than chance as well as that active sentences could be comprehended better than chance, whereas passive sentences could be comprehended no better than chance. In sum, the hypothesis of 'Trace Deletion' could not be confirmed with the outcomes of this study, as the predicted dissociation for problems with passive but spared performance for active, semantically reversible sentences, couldn't be found.

Similarly, within a meta-analysis testing 40 published data of Broca's aphasics, Caramazza et al. (2001) could show that Broca's aphasia is not associated with a consistent pattern of sentence comprehension performance. Contrasting they concluded that there are individual differences from patient to patient.

Interestingly, Grodzinsky et al. (1999) tested the same data within a meta-analysis and could show that Broca's aphasics as a group, comprehend active reversible sentences normally (100% correct), but passive relative clauses at a chance level, proving the hypothesis of 'Trace Deletion'.

As a reason for different results analyzing the same data, Caramazza et al. (2001) pointed out differences in the methodology used and criticised especially the statistical reasoning adopted by Grodzinsky et al. (1999).

Caramazza et al. (2005) further tested the core prediction of the 'Trace Deletion Hypothesis' by analyzing the sentence comprehension performance in 38 Italian agrammatic patients. Results of this study failed to confirm the predictions made by the 'Trace Deletion Hypothesis', as only 15% of patients tested performed at chance level on passive semantically reversible sentences and better than chance on active sentences. From these results it could be concluded moreover that agrammatic Broca's aphasics do not present with a homogeneous profile in their sentence comprehension performance. Even though all patients tested had lesions within Broca's area, nevertheless their comprehension performances were heterogeneous.
Similarly, study outcomes by Burchert and De Bleser (2004) could further not support the main idea of the 'Trace Deletion Hypothesis' by Grodzinsky (1990). In detail, as a group, six German agrammatic patients tested did not encounter problems in comprehending semantically reversible sentences. At an individual level, only two out of six patients could meet the agrammatic comprehension pattern as predicted by this hypothesis, with chance performance on passives and normal results for actives.

This subsequent growth in data addressing sentence comprehension abilities in Broca's aphasics pointed out in sum a certain degree of individual variation in the comprehension profiles of agrammatic patients. However, a quantitative, statistical analysis of a large data set (69 carefully selected Broca's aphasics), tested on nearly 6000 stimulus sentences, has indicated a highly robust selective impairment for the analysis of syntactic movement and thus confirming the hypothesis of 'Trace Deletion' (Drai & Grodzinsky, 2006). However, it couldn't be shown with this study either that inter-patient variability does not exist in this syndrome at all. Patient's performance on non-movement sentences exhibited just less variation than on sentences containing syntactic movement.

Furthermore, a variability in syntactic comprehension has been reported across languages. While English passive sentences seemed to be comprehended at chance level (Grodzinsky, 1986; 1990; 1995a; 1995b; 1999; Drai and Grodzinsky, 2006), this was not always the case for German, Dutch, Hebrew and Italian passives, also derived by syntactic movement (e.g., Friederici & Graetz, 1987; Burchert & de Bleser, 2004). These results lead to an important question as to whether the general assumption of the 'Trace Deletion Hypothesis' holds also for languages that have richer morphology than English (e.g., German). More specifically, the generalisation of the 'Trace Deletion Hypothesis' to morphologically rich languages cannot be taken for granted as number, case or gender morphology could provide explicit cues for the detection of the thematic roles in a sentence and thus might help the patient to comprehend who is doing what to whom in a sentence, without analyzing the sentence syntactically per se.

However, a recent study by Burchert, Friedmann and De Bleser (2003) could show that morphologically manipulated non-canonical sentences (manipulation of case, gender, number) did not improve the comprehension of non-canonical sentences in morphological rich languages (German and Hebrew).
Furthermore, study outcomes by Burchert and De Bleser (2004) revealed that the processing of semantically reversible sentences is not guided by semantics, nor by the variable of length or morphology (by-phrase).

Thus it seems that in general the hypothesis of 'Trace Deletion' could be used to explain errors found in the comprehension of semantically reversible sentences. But on the other hand the strong dissociation of an above chance performance for active and subject-relative clauses, in contrast to a below chance performance for passive and object-relative clauses has to be rejected on the basis of study outcomes presented above. As shown above, in sum, studies could not evidence a unique pattern of agrammatic comprehension, as predicted by the theory of 'Trace Deletion'. Even though a trace deletion pattern could be found for some subjects on some sentence structures, this pattern does not apply for all structures, nor for all agrammatic patients together. However, it seems that the hypothesis of 'Trace Deletion' can only explain, if at all, the comprehension deficits of a subgroup of agrammatic patients. Thus alternative neurolinguistic theories are necessary to explain additional comprehension deficits in agrammatic patients (cf. Burchert & De Bleser, 2004: 44). Consecutively, it could be concluded that it makes little sense to ask what the cause of the sentence comprehension deficits in Broca's aphasics is, as Broca's aphasics seem all to be different from each other and thus have different sentence comprehension abilities. As could be shown from divergent study outcomes, there is no single type of sentence comprehension impairment in agrammatic Broca's aphasics, most likely reflecting different areas of damage of mechanisms involved in sentence processing (not just trace deletion).

Furthermore, the rejection of the 'Trace Deletion Hypothesis' does not imply the rejection of the possibility that some patterns of comprehension deficits are due to the damage of some components within the language processing system, involved in syntactic analysis (e.g., defective mechanism of the assignment of thematic roles). Simply speaking, even though Broca's aphasics agrammatic sentence comprehension performance is not associated with a single pattern, this does not mean that nothing in some of these subjects comprehension deficits relates to the damage of some components of syntactic processing, as described by the hypothesis of 'Trace Deletion' (cf. Caramazza et al., 2005: 51f).

Heterogeneous study outcomes may indicate furthermore the involvement of various brain structures highly interconnected with each other (cf. Caramazza et al., 2005: 51f).
Important however for the explanation of syntactic comprehension deficits (syntax III) of HD patients within this study, is the question if results from Broca's aphasics could also be used to describe syntactic comprehension deficits in HD patients? Given the hypothesis that Broca's area, located within the frontal cortex, does not exist in isolation (Ullman, 2006), but is contrastingly part of the cortico-basal ganglia-thalamo-cortical circuit as predicted by cortico-basal ganglia-thalamo-cortical models (Albin, Young and Penney, 1989; Alexander, DeLong & Strick, 1986; Alexander & Crutcher, 1990; Alexander, Crutcher & DeLong, 1990; Cummings, 1993; Gurney et al., 2004; McHaffie et al., 2005; DeLong, 1990; Wise, Murray & Gerfen, 1996; Middleton & Strick, 2002) and is furthermore interconnected within the cortex with other regions fulfilling the same functions (Ullman, 2006), it could be possible that results from Broca's aphasics could also be helpful, at least to some degree, to explain the deficits in comprehending semantically reversible sentences in HD patients.

Unfortunately however, HD patient's performance in syntax III contrasts study results by Grodzinsky, (1986; 1990; 1995a; 1995b; 1999) as well as by Drai and Grodzinsky (2006) testing agrammatic Broca's aphasics. Performances of HD patients tested are rather comparable with studies outcomes by Schwartz, Saffran and Marin (1980), Berndt, Mitchum and Haendings (1996), Caramazza et al. (2001), Caramazza et al. (2005) as well as Burchert and De Bleser (2004), also not able to prove all assumptions of the hypothesis of 'Trace Deletion'. Interestingly, HD patients sometimes even made slightly more errors in semantically reversible active voice sentences than in semantically reversible passive sentences (see syntax III_b versus syntax III_c; syntax III_d versus syntax III_e). Furthermore slightly more errors occurred in semantically reversible sentences with subject focus than in constructions with object focus (see syntax III_g; syntax III_h), again disproving the core hypothesis of the theory of 'Trace Deletion'.

The question remains where these errors come from and why they are so divergent from errors found in Broca's aphasics?

One explanation for the difference in errors between HD patients and Broca's aphasics could be the degree of brain-pathology. As HD is characterized by a gross neurodegeneration, including mainly subcortical areas, this syndrome may affect cognitive abilities such as language worse than the mainly cortical lesion associated
with Broca's aphasia. Furthermore, in HD additional deficits arise such as a subcortical dementia, which could additionally influence cognitive abilities, including language comprehension, negatively. Thus in sum errors in HD patients were worse than in Broca's aphasics, as errors occurred not only in passive and object relative clauses, but also in active and subject relative clauses.

However, the question still remains why HD patients in general made slightly more errors in active and subject-relative constructions than in passive and object-relative clauses, compared to agrammatic patients and also across all languages reported.

At that point, it seems we have to be content with the following conclusion: Given that language comprehension is a very complex mechanism so far no model has been developed yet which is able to account for all various linguistic errors arising. Thus we sometimes (and unfortunately also in this case) just don't know why some kinds of errors arise and others don't and thus we are left with our best guess.

At least for errors in passive and object-relative clauses some hints from the hypothesis of 'Trace Deletion' (Grodzinsky, 1990) could be useful in terms of the explanation of errors. As predicted by this theory, due to the brain damage associated with HD the traces are deleted, which under normal conditions, index the position of moved elements in their former D-structure. Thus HD patients experience deficits in the assignment of theta roles in semantically reversible sentences, due to the deletion of these traces.
7.4.2 Discussion of syntactical errors – in detail

Briefly, the dysexecutive syndrome (for details see chapter 1.4.1.1), the syndrome of dynamic aphasia (Luria & Tsvetkova, 1968), including Kleist's (1930) idea of a 'loss of drive in thinking' as well as Grodzinsky's (1990) hypothesis of 'Trace Deletion', may help to explain some of the HD patients erroneous syntactic productions.

As HD patients have been reported to suffer from a dysexecutive syndrome (see chapter 1.4.1.1) as well as to display dynamic aphasia like symptoms, as evidenced for other basal ganglia patients (e.g., Crescentini et al., 2008b), it seems evident that these considerations could also account for syntactic deficits in HD patients.

In detail, within sequencing of sentence parts, HD patient's errors were direct proportional to the number of parts a sentence was comprised of. Not surprisingly, performance decreased as the number of sentence parts increased; with the highest number of errors for sentence 14, comprising 10 sentence parts (20.5% sequencing errors).

Due to this task's sorting characteristic it can be regarded as being highly sensitive for testing executive functions. As HD patients have been described to suffer from a dysexecutive syndrome, having difficulties with cognitive tasks that require organization, planning and sequencing of events (Brandt, Folstein & Folstein, 1988; Rosenberg, Soerensen & Christensen, 1995; Lawrence et al., 1996; Lemiere et al., 2004; Peinemann et al., 2005) difficulties performing this task could thus interact with mnemonic factors, such as executive functions. Due to the fact that disturbances in sequencing of sentence phrases/parts of sentence phrases could be shown for various syntactical categories to a similar degree, it is hard to determine which sentence phrases/parts of sentence phrases were the most likely to cause these errors. However, outstanding was the high number of mis-sequencings in sentences with more than one object. In detail, the most sequencing errors occured for accusative objects, to a lesser degree for genitive objects and to an even lesser degree for dative objects. Additionally further sequencing errors could be found in various syntactical domains including subjects, predicates, full verbs, auxiliary verbs, modal verbs, adverbs, adjectives, prepositions, as well as whole prepositional phrases, possessive pronouns, and possessive

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122Interestingly, problems in morphosyntactical agreement occured as well within syntax III, as there were sentences with more than one object.
pronoun phrases, personal pronouns, reflexive pronouns as well as also conjunctional phrases. (A comprehensive overview of all sequencing errors made, in various linguistic domains, differentiated for patients and controls, is given in chapter 6.5 as well as in Appendix XIII).

Within guided syntactic production study subjects were supposed to construct a grammatically meaningful sentence, including a number of given words. In accordance to study results by Chenery, Copland and Murdoch (2002), HD patients experienced troubles producing sentences on certain constraints. Patients tested, produced grammatically correct sentences when containing only one out of three given words. When including all three words given, sentences became ungrammatical.

In order to make a grammatically correct sentence, including all words given, words have been changed by the HD patients in terms of their syntactic categories (adverb; wichtig > adjective wichtiges; Morgen schreibst du ein wichtiges Diktat ), in terms of their inflections (Mutter > Mütter; schreibst > schreibt), they have further been changed to phonological similar words (wichtig > richtig), or to other modal verbs (möchte > will) and other lexemes (Herrchen > Nachbar) etc. (for a total list of errors made within guided syntactic production, see Appendix XIV).

Thus the assumption could be made that HD patient's syntactic system is intact per se. What seems to be lacking is the ability to construct sentences on certain constraints. As mentioned in Appendix V, in order to do so, the sentences have to be planned by means of 'predicative functions of inner speech' (cf. Vygotsky, 1962: 44; Luria & Tsvetkova, 1968: 297). Thus it could be assumed that HD patient's language deficits are probably similar to those seen in patients suffering from a dynamic aphasia (Luria & Tsvetkova, 1968) with a particular impairment in generating sentences when cued by verbal stimuli (cf. Luria & Tsvetkova, 1968: 301). It could be assumed further that the syntactic deficits evidenced in HD patients tested, are somehow linked to Kleist's concept of 'alogische Denkstörung' with a 'a loss of drive in thinking' (cf. Kleist, 1930: 101f), which has been incorporated later into Luria's and Tsvetkova's (1968) concept of dynamic aphasia (for more details see Appendix V). Kleist (1930) further included this concept into the symptomatology of schizophrenic patients as well into patients with neurodegenerative diseases, such as HD (cf. Pillmann & Möller, 2004: 155).
In conclusion, HD patient's syntactic deficits may be due to Kleist's (1930) concept of a 'loss of drive in thinking', which has also been reported to be part of HD patient's dysexecutive syndrome (for details see chapter 1.4.1.1) in the course of their subcortical dementia (Folstein, Folstein & Mc Hugh, 1975) and could further be related to the syndrome of dynamic aphasia (Luria & Tsvetkova, 1968).

Noteworthy are moreover errors within syntax III. A significant difference between patients and controls could be reported for syntax_comprehension, including syntax III (p < 0.01). As shown previously in chapter 6.10, a relative high percentage of errors could be reported for all subtasks of syntax III. Percentages of errors increased, with an increase of complexity in tasks; with the highest percentage of errors for coordinate sentences. It could be further inferred that HD patients have particularly difficulties in the assignment of theta roles (Table 53, as given in chapter 6.10.9, summarizes theta role assignment violations for all subtasks within syntax_comprehension, separately for HD patients and healthy controls).

Study outcomes could be supported by Teichmann et al.'s (2005) theory of a defective syntactic rule use in HD patients, by evidencing HD patients to be most impaired in non-plausible passive sentences. This result further accounted for the hypothesis that the understanding of thematic roles is defective in HD patients. In order to understand non-plausible passive sentences the thematic roles have to be understood, in order to figure out who is doing what to whom. A defective syntactic rule application has further been shown by Teichmann et al. (2007; 2008).

Errors made within syntax III could moreover be explained somehow by the hypothesis of 'Trace Deletion' (Grodzinsky, 1990), as described in detail in chapter 7.4.1.

However, interestingly, HD patients sometimes even made slightly more errors in semantically reversible active voice sentences than in semantically reversible passive sentences (see syntax III_b versus syntax III_c; syntax III_d versus syntax III_e). Furthermore slightly more errors occurred in semantically reversible sentences with subject focus than in constructions with object focus (see syntax III_g; syntax III_h), disapproving the main idea of the hypothesis of 'Trace Deletion' (Grodzinsky, 1990).

Explanations for the difference in errors between HD patients and Broca's aphasics could be the different degree of brain dysfunction in HD patients versus Broca's aphasics, as well as further deficits associated with HD, such as subcortical dementia.
Thus in sum errors in HD patients were worse than in Broca's aphasics, as errors occurred not only in passive and object relative clauses, but also in active and subject relative clauses. At least for the understanding of problems in the comprehension of passive as well as object-relative constructions, some hints from the hypothesis of 'Trace Deletion' could be useful for the explanation of errors. As far as this is concerned HD patients are possibly comparable to agrammatic Broca's aphasics, lacking the traces which give us the information which thematic role has to be assigned to a certain argument, leading to comprehension deficits in semantically reversible sentences.

7.5 Discussion of results – semantics

Also in terms of semantics, patients performed statistically significantly worse, than healthy controls, in all tasks provided. A statistically significant difference between patients and controls could be reported for single word understanding_general, including providing antonyms \((p < 0.01)\), due to a total of 20 errors for HD patients, compared to 2 errors in healthy controls. Further, a statistically significant difference between patients and controls could be shown for single word understanding_general, including providing two semantic concepts of polysemes \((p < 0.01)\), with a total of 75 errors for HD patients, in contrast to healthy controls, who only made 8 errors. For single word understanding_general, including providing two semantic meanings of homophonic words a statistically significant difference between patients and controls could additionally be shown \((p < 0.01)\). Within this task HD patients made a total of 131 errors, compared to healthy controls, who only made 10 errors.

In sum, on the basis of the kinds of errors made it could be assumed that HD patients do not lack semantic information of words per se, rather the access to these stores seems to be inefficient. Thus, in accordance with study outcomes by Hodges, Salmon and Butters (1991), Frank, McDade and Scott (1996) as well as by Chenery, Copland and Murdoch (2002) in HD patients, there is a lack in lexical retrieval due to problems in executive functioning, rather than a deficit in the semantic system per se. These results could further be supported by semantic fluency outcomes, as given in chapter 6.1.

This assumption will be justified in regards to errors made within providing antonyms, providing two semantic concepts of polysemes and providing two semantic
meanings of homophonic words. In detail, the storage/retrievement dissociation could be proved within providing antonyms, as HD patients produced existing German synonyms (aufmachen – öffnen) as well as semantically related lexical intrusions (zugeben – abspielen; gut – sauer), instead of the appropriate antonyms of the word. A person who is severely affected in its semantic system per se, would not be able to make this kind of errors. HD patients further made the impression to distract from their deficiencies to access antonyms of the given word out of the semantic memory by producing the opposite of the given word in terms of negations (kaufen – nicht kaufen; zugeben – nicht zugeben).

Retrievement issues have further been supported by showing a high number of errors due to non responses and right answers based on help.

Within providing two semantic concepts of polysemes similar error types could be found. Errors made, were mainly due to non responses, to a lesser degree due to namings where the second naming referred to the same lexeme as the first, as well as due to the naming of only one item. These findings are in accordance with Chenery, Copland and Murdoch (cf. 2002: 469), even though described for the domain of homophones. It has been suggested by these researchers that the effect of giving non responses, providing one definition but being unable to shift attention and providing an alternative meaning and thus providing perseverations of the first meaning instead (e.g., Bank: 'Parkbank, Terrassenbank; Blatt: Papier, Zeitungsblatt') might be due to an inflexibility in thinking, in the course of HD patients dysexecutive syndrome (cf. Lawrence, Sahakian & Robbins, 1998: 380).

The same error patterns could also be found within providing two semantic meanings of homophonic words. Erroneous productions were again based on the naming of only one item, on perseverations as well as on non responses. Striking herein was moreover a high amount of lexical intrusions (e.g., Saite/Seite: abseits; Lied/Lid: ein Leid das man hat) which support as well the hypothesized dissociation between a deficit in lexical access rather than in the semantic memory per se.
8 Conclusion

In conclusion, the main hypothesis of this thesis could be confirmed that the basal ganglia have a certain role in language. This is because linguistic functioning seems to be statistically significantly affected in HD patients, regardless of HD stage, in comparison to healthy controls. As the disease progresses linguistic errors worsen as well. Due to the specific neurodegeneration within the basal ganglia, in detail within the striatum, it has been assumed that the cortico-basal ganglia-thalamo-cortical circuit becomes defective and thus leads to the development of certain linguistic errors, as found in HD patients tested.

Interestingly, linguistic disturbances were not only to occur within 'complex language tasks' with a 'high degree of freedom', but also in 'primary language tasks' with a 'low degree of freedom'. As described within the cortico-basal ganglia-thalamo-cortical model focusing on language, linguistic errors of HD patients may be due to a loss of the regulatory function of the basal ganglia on the cortex, consecutively leading to a cortical overactivation. Hence, various lexical alternatives are simultaneously active and thus linguistically mis-choosings in 'low degree of freedom tasks' as well as defective rule application in 'high degree of freedom tasks' have been the result.

More specifically, in terms of morphology, morphological rule application has shown to be defective, even though the morphological system itself seems to be intact. This dissociation could be evidenced by a high number of neologism errors, due to the application of wrong morphological affixes on the one hand and the production of complex compoundings and correct but not desired German derivations/conversions, on the other hand. A trend could be found that some errors can be explained by the linguistic theory of 'Natural Morphology', as morphological forms tended to be reduced from more marked to less marked forms.

In terms of syntax, supposedly the concept of a dysexecutive syndrome (for details see chapter 1.4.1.1), including Kleist's (1930) idea of a 'loss of drive in thinking', could be tied into language and thus being responsible for the arise of dynamic aphasia like symptoms in HD patient's syntactic abilities (e.g., deficits in planning sentences on certain constraints). This assumption is further based on the evidence that HD patients
have been reported to suffer from a dysexecutive syndrome (for details see chapter 1.4.1.1) and to experience syndroms similar to that seen in dynamic aphasia (Mega & Alexander, 1994; D'Esposito & Alexander, 1995; Chenery, Copland & Murdoch, 2002; Jensen, Chenery & Copland, 2006).

At least to some degree various errors within syntax III could be explained by the linguistic theory of 'Trace Deletion' (Grodzinsky, 1990). Specifically, problems with passive and object-relative constructions could be explained by a lack of traces which index where a transformed element was positioned in the D-structure. As these traces are lost in HD patients, due to their brain dysfunctions, the assignment of thematic roles becomes defective resulting in a chance performance when comprehending semantically reversible sentences.

For semantics, probably also the concept of a dysexecutive syndrome could account for the errors made. As HD patients were unable to shift their attention, maybe due to a lack in executive functioning, they ended up giving no answers at all, gave only one out of two answers or became perseverative in their answering. Thus it could be concluded that HD patient's semantic memory itself is intact. What is lacking, is supposedly an effective access to the semantic store and thus retrievalment issues are the result.

In sum, language problems in HD patients tested, could be linked to a malfunctioning within the cortico-basal ganglia-thalamo-cortical circuit, resulting into dysfunctions in the executive functions and consecutively negatively influencing language output. However, unfortunately language disturbances found, are not easily to fit into any kind of typical aphasia classification (maybe not even into the concept of dynamic aphasia, as this type of aphasia is typically not associated with comprehension deficits). Possibly the language profile found, could be best described in terms of Luria and Tsvetkova's (1968: 307) quotation as being a type of 'aphasia without aphasia'?

Nevertheless, as language changes have been proven to occur in the course of HD, these deficits cannot be ignored anymore within HD's specific symptomatology. Hence, the concept of subcortical dementia seems further to be challenged. The consideration that the only domain fully intact in subcortical dementia is language, as mentioned in chapter 1.5.2.1, can't be held anymore, regarding results of this study.
As language functions have been proven to become severely affected in HD patients, no matter in which HD stage patient's are, it seems evident that these cognitive-linguistic deficiencies negatively affect communicative abilities and thus also the ability to live an autonomous life. Unfortunately until now there is a lack of descriptions of treatment strategies for cognitive-linguistic difficulties of HD patients. Thus, these considerations can be seen as an important future perspective of this study.

However, there are also a few limitations that need to be acknowledged and addressed regarding the present study. In order to further confirm the assumption of this study, that language functions are disturbed in HD, these deficiencies may be addressed now and pointed out as well important future research directives.
9 Limitations and future directives

In detail, limitations of this study concern among other things, the use of a non-standardized language screening. Unfortunately, as already mentioned previously in chapter 4.3, there are only a few standardized language batteries for German speaking adults, e.g., the Aacherner Aphasie Test (Huber et al., 1983), the Aphasia Check List by Kalbe et al. (2002), LeMo, which is only single word based, by De Bleser et al. (2004). As all of these tools have been designed for aphasic patients, these tests wouldn't have been suitable for testing HD patients with a subcortical pathology. Thus, a main future directive would be the standardization of a language battery, for subcortically affected adults.

Furthermore, in terms of brevity constrictions, the author of this study is aware of the fact that not all possible e.g., plural formations for German could be tested. These constrictions can be seen as a further limitation of this study. Nevertheless, this screening can be regarded as being part of a pilot study, testing language functions in German speaking HD patients, using a non-aphasic language tool. Extension of this screening, in terms of a larger consideration of linguistic items tested, can thus be seen as a future directive of this study.

In order to find out more about language functions in HD, it would be necessary to test all domains shown to be affected (morphology, semantics and syntax) in more depth. Unfortunately, this hasn't been possible within this project in terms of ethical constrictions, but can be seen as an additional directive for the future.

As already presented in chapter 7.1, no matter in which stage of HD patients were, all performed statistically significantly worse on the language screening, than did their healthy counterparts. As this effect is maybe due to a too loose or inappropriate definition of HD stage (it seems not surprising that an inability to handle financial affairs' contributing to stage 3 within the Total Functional Capacity Scale by Shoulson and Fahn (1979), does not necessarily contribute to language disturbances) the use of Shoulson's stages in determining HD progression can thus be seen as a further limitation of this study (and maybe of all studies dealing with HD, using Shoulson stages, determining disease progression in terms of standardized stages). Against that the bicaudate ratio (measured by MRI) has shown to be a more reliable outcome measure in clinical trials, by evidencing a significant correlation between the bicaudate ratio and
syntactic rule application in the study by Teichmann et al. (2005). Aylward et al. (2003) further proposed the caudate volume as a valuable outcome measure for Huntington's disease. Similarly, Andrews et al. (1999) pointed out that PET is sensitive in measuring striatal D₁ and D₂ dopamine receptors and thus to be useful in determining HD progression. Hence, in future research MRI measuring the volume of the caudate nucleus could be more efficient in determining HD stages, than using too loose Shoulson stages.

Furthermore, in comparison with other studies dealing with language functions in Huntington's disease (as presented in chapter 3), a total of 20 patients recruited for this study can't be regarded as a non representative sample for testing the hypothesis that language functions are disturbed in HD. Nevertheless, a bigger sample would of course support even more the significant results found and could thus be seen as a further future directive of this study.

9.1 Future directives concerning quality of life in HD

Addressing life with HD – until death, it is of special importance to the author of this study to strengthen mainly two mechanisms contributing positively to the enhancement of quality of life in HD patients. Hence these two factors, as given below, can be seen as the main future directives for HD patient's quality of life, regardless of science and further research.

9.1.1 Enhancement of quality of life – with HD day care units and interdisciplinary home visiting teams

It is difficult to imagine an experience more demanding than that of dealing with HD. As explained in chapter 1, HD is a terminal disease without any options for cure. Because of its occurrence in midlife, HD can have devastating effects on the patients' life, including the loss of jobs, friends, mobility and independance (cf. Dawson et al., 2004: 124). These negative effects also cause major disruptions in family life, as shown in the study by Vamos et al. (2007). As HD progresses, it becomes more and more difficult for care to be provided at home. Unfortunately, patients even at an early age,
end up in nursing homes, where they and their needs are not known. At present, there are very few residential facilities capable of meeting the needs of people with HD. Frequently, care is provided in institutions such as aged care placements that are clearly inappropriate for midaged patients affected by HD (cf. Dawson et al., 2004: 124). This problem could also be reported by Julie Morrow (2009, personal communication), the junior welfare officer of the Australian Huntington's Disease Association – Brisbane, who the author of this work was glad to meet. But Mrs. Morrow reported that nevertheless a lot of effort has been put inside Australia into the establishment of an interdisciplinary team, including doctors, psychologists, social workers, physiotherpists, occupational therapists, speech-language pathologists, nutritionists, as well as welfare officers, travelling to patients' homes in regular intervals, in order to best possibly support families dealing with HD. This facility makes it possible for HD patients to stay at home as long as possible. Additionally, a phone hotline has been established for carers, to support them when personal meetings are not possible, due to long distances within Australia. Furthermore, day care centers have been established, meeting the demands of HD patients and supporting them to live their life as normal as possible. Within these centers people have the opportunity to take part in various therapeutic sessions, to perform manageable household activities in a structured atmosphere with the help – if needed – of a carer. These facilities make it possible for patients to share problems and thoughts, to structure daily routines. These opportunities in sum make life more purposeful and thus also more enjoyable and henceforth enhance the quality of life.

9.1.2 Enhancement of quality of life – with augmentative and alternative communication

As shown within this thesis, language functions are severely affected in HD, no matter in which stage patient's are. Thus, it seems evident that these cognitive-linguistic disturbances result further into disturbed communicative abilities. Unfortunately, until now there is a lack of information regarding treatment strategies for cognitive-linguistic-communicative difficulties in HD patients. Nevertheless, within this chapter it will be shown how HD's symptoms negatively contribute to patients' communicative abilities, how this affects their ability to live an
autonomous life and what can be done in order to support these patients best possibly. As disease progresses to late stages, patients become mentally incompetent and thus also communicatively impaired, due to progressive cognitive and emotional disturbances. The speech of HD patients has been described to become disturbed by a hyperkinetic dysarthria, due to massive motor disturbances, such as chorea (cf. Yorkston et al., 1999: 154ff). Additionally, disturbances within the striatum and its connections with the cortex via the cortico-basal ganglia-thalamo-cortical circuit, leads moreover to pure linguistic disturbances, as evidenced by the literature review given in chapter 3, as well as by the results of this study.

Due to the sum of these negative contributors, it is not surprising that the communicative abilities in HD patients are not fully intact.

Given, that the disease can span more than 10 years, individuals may be left without 'sufficient' communication for a very long period of time (cf. Klasner & Yorkston, 2001: 154). Consecutively, various treatment strategies are sometimes imposed on these patients, even against their will. But as a basic right of every human being, it must be assured that patients have as much autonomy as possible and can express their needs and demands (cf. Simpson, 2007: 181). In order to do so, they rely on functioning speech and language functions.

Due to the fact that this thesis is about language changes in HD, the main focus of this chapter will be on how to enhance linguistic-communicative abilities in HD patients, in order to express needs, even at the end stage of their disease.

Unfortunately, not a lot of literature is available on this topic by now. However, the author of this thesis is in strong belief that this issue is very important to address and also belongs to the field of speech-language pathology and linguistics as descriptions of language changes. Thus, a brief introduction will be given on a few advance directives, how to facilitate communication in HD patients.

In detail, the implementation of various so called 'augmentative and alternative communication' strategies is of importance to HD patients, in order to support and facilitate their communicative abilities. The Augmentative and Alternative Communication Special Interest Division 12, of the American Speech-Language-Hearing Association (ASHA) defined augmentative and alternative communication (= AAC) as follows:
“[...] AAC refers to an area of research, clinical and educational practice. AAC involves attempts to study and when compensate for temporary or permanent impairments, activity limitations, and participation restrictions of persons with severe disorders of speech – language production and/or comprehension, including spoken and written modes of communication” (ASHA, 2005: 1)

For a general introduction into the field of AAC, see for instance Beukelman and Mirenda (1998: chapter 1), or Beukelman and Mirenda (2005: chapter 1). In order to better understand the basic principle of the concept of AAC, a statement of a famous Austrian psychologist and philosopher Paul Watzlawick is that “Man kann nicht nicht kommunizieren [“one cannot not communicate”].

Hence, it seems evident that every form of communication is communication even though against our common understanding of communication in terms of vocalization. Thus, it is important to emphasize that AAC should be referred to as a system integrating any component that could be used for the purpose of communication, such as speech vocalizations, gestures, signs, symbols, individual strategies etc. (ASHA, 2004; 2005).

Various alternative AAC strategies have been reported for HD patients, in order to facilitate their communicative abilities and to remain autonome in decisions upon their lives. Nevertheless, AAC gives not back HD patients their normal cognitive-linguistic strategies and well formulated language capacities. Contrastingly, these strategies can offer a way to make themselves understood, even without speaking a word.

Unfortunately however, communication disorders associated with HD, are even very challenging for the field of AAC. This is because they reflect a complex interaction between impairments of motor functioning such as dysarthria and progressive cognitive-linguistic changes in combination with the three main HD symptoms, as mentioned in chapter 1. Thus, HD patient's AAC systems must meet these needs (speech disturbance, motor disturbance and cognitive impairment) in the present and must be adoptable in the course of HD's progression (cf. Klasner & Yorkston, 2001: 154).

Unfortunately however, there are very few publications, describing AAC interventions on HD patients (cf. Yorkston & Beukelman, 2007: 317). Nevertheless, a brief overview about various AAC strategies will be given now.

Due to the fact that communicative abilities in HD patients decrease over the 5 stages of HD, various AAC strategies can be used in every stage, in order to support communicative abilities. Klasner and Yorkston (2000), Klasner and Yorkston (2001),
Yorkston, Miller and Strand (2004) as well as Yorkston and Beukelman (2007) reported various strategies being able to be implemented in the early stages of the disease that are also useful in late and final stages of the disorder.

- As cognitive changes and depressive moods start to negatively affect communication, memory aids and organizational tools to break down tasks into manageable portions (cognitive supplementation strategy), may assist in maintaining daily activities. Using this concept, household activities can be broken down into small steps, such as for instance: put out canned cat food, fill up dry cat food once per day, check the water bowl....
- Key words (e.g., 'cat') can be used to trigger activities, such as to feed the cat.
- Alphabet boards containing letters, numbers and selected phrases, may be an important element for the communication of individuals with HD. For instance, visual cues can be used for spelling words, counting on the other hand can be used for cueing themselves within a conversation.
- Offering the person a choice among a small number of alternatives (what food to eat, which clothing to use for dressing etc.), can also be an effective strategy to break down communication into the most important pieces. This strategy can be adopted to patients in advanced stages of their disease, as yes-or-no answer systems (Do you want a drink? Yes – No).
- Because of the frequent occurrence of memory problems, compensatory techniques such as calendars and memory aids have also been reported to be effective. Memory aids can include personal data, daily information about what the patient did today, as well as forms to chart the patient's feelings about specific happenings etc.
- Mini-boards including conversation starters such as basic needs ('I have to go to the restroom'), have also been shown to be helpful.
- As also set shifting within conversations becomes problematic, cuing boards (including messages such as 'You have already talked about that' or 'You were talking about your trip last week') can be used to keep the person on the topic, to signal topic changes and to facilitate thinking processes.
- Furthermore, natural gestures or specific signs can be introduced, to supplement speech production.
- Communicative language abilities have been reported to be further facilitated and stimulated by participating in communication groups.
communication groups various communicative tasks can be administered, including for instance naming activities, gathering biographical information about each participant, to find common interests to talk about (Berarducci et al., 2003).

As HD advances and patients are no longer able to live at home, they mostly end up in nursing homes. Henceforth, carers in nursing homes must be made aware of AAC strategies already used by HD patients. Furthermore, the need to support the development of new AAC strategies for HD patients to use, has to be stressed. Even though verbal communication decreases over time in HD patients, or may diminish at all in later stages of the disorder, it is to keep in mind that this doesn’t mean that HD patients don’t have any communicative needs. However, communication or memory books can be an effortful tool as they provide information about the patient’s likes, dislikes, hobbies and interests (cf. Klasner & Yorkston, 2000: 245ff). As communicative memory booklets further create a vivid picture of HD patients lives before and after the onset of their disease they can thus be seen as alternative conversational pieces, to help unfamiliar carers to get to know their client (cf. Simpson, 2007: 180). An example of a memory book is given below in Picture 7.

Picture 7 shows a sample of a memory book (from Bourgeois et al., 2001: 210)

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123The implementation of memory books in nursing home residents with dementia, has been reported to even improve depressive moods and to result into general positive changes in quality of life (Bourgeois et al., 2001).
In conclusion, no matter which AAC techniques are administered, the most important factor to keep in mind when working with HD patients is 'to keep it simple'. Only simple, specific questions should be asked which can be answered in one word. Moreover, enough time should be given to answer questions. These indicators have especially been stressed by Julie Morrow (2009: personal communication) the junior welfare officer of the Australian Huntington's Disease Association, Brisbane. In a similar way Beukelman et al. (2007) as well as Small et al. (2003; 2005) summarised that in order to support the patient best possibly in its communication, distractions have to be reduced, complex sentences have to be shortened to simple phrases, questions have to be reduced to a yes/no format, as well as enough time must be allowed to respond. Within this study language abilities have been shown to be severely affected in HD patients, regardless of the stage of the disease. Consecutively, these deficits have been reported to contribute to deficiencies in linguistic-communicative abilities. Thus, it is to conclude that in addition to tests regarding psychiatric, cognitive and motor deficiencies, assessments of HD patient's speech-language as well as communicative capacities need further to be included in HD's specific testing procedures. An additional focus should be made on strategies to support language-communicative abilities (e.g., AAC) in order to allow HD patients to decide themselves as autonomously as possible, upon their lives. Even though some efforts have already been made in America and Australia to address AAC also in the domain of neurodegenerative diseases, such as HD, unfortunately a lot of work still needs to be done inside Europe and especially inside Austria.
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Appendix

I. Neurophysiological basis for PD movement disorders

In the example of PD\textsuperscript{124}, there is a selective loss of dopaminergic neurons in the substantia nigra, that normally projects to dopamine receptors in the striatum. Thus, within the indirect pathway, a loss of striatal dopamine leads to an excessive inhibition of the GPe. This leads in turn to a disinhibition of the STN, which in turn provides an excessive drive to the basal ganglia output nuclei. Consecutively the thalamic nuclei are inhibited. In the direct pathway, there is further a reduced inhibitory input to the basal ganglia output nuclei, thus also leading to an inhibition of thalamo-cortical neurons and thus resulting into an inhibition in the initiation of movements (adopted and simplified from Albin, Young & Penney, 1989; Alexander & Crutcher, 1990; DeLong, 1990; Smith et al., 1998; Mink, 1999; Gutekunst, Norflus & Hersch, 2002; Murdoch 2004; Mink, 2008; Murdoch & Whelan, 2009). According to DeLong (1990) these neurophysiological changes lead to symptoms such as akinesia and bradykinesia, seen in Parkinson's disease.

Figure Appendix I shows the basal ganglia pathways in PD (adopted from Murdoch, 2004: 152)

\textsuperscript{124}Since the increased activity of the direct pathway is associated with a facilitation of movement, the increased activity of the indirect pathway is associated with the inhibition of movement, it is suggested that Parkinson's disease (PD) is the result of an imbalance in the activity of the indirect pathway (over-activity of the indirect pathway). Contrastingly, HD is associated with an imbalance in favour of the direct pathway (over-activity of the direct pathway), as will be exemplified below.
II. The role of the dominant thalamus in language

Within the diencephalon, the thalamus is by far the largest component. The thalamus itself is made up of about 120 nuclei; about half of these nuclei send fibres to the cortex. Terminology related to thalamic nuclei is extremely complex and varies from author to author (cf. Augustine, 2008: 242ff). Nevertheless within this thesis, the role of the thalamus for language processes will be discussed and thus only thalamic nuclei stated to play a major role in language will be focused on herein.

The role of various thalamic nuclei for linguistic purposes has been tested via stereotactical surgery (= thalamotomy) (e.g., Bell, 1968; Riklan & Cooper, 1975; Vilki & Laitinen, 1976), via electrical stimulation (e.g., Ojemann, Fedio & Van Buren, 1968; Ojemann & Ward, 1971; Ojemann, 1975; Schaltenbrand, 1975), after thalamic hemorrhage (evidence of aphasia after thalamic hemorrhage) (e.g., Mohr, Watters & Duncan, 1975; Horenstein, Chung & Brenner, 1978; Reynolds et al., 1979; Alexander & Lo Verme, 1980; Glosser, Kaplan & Lo Verme, 1982; Crosson et al., 1986), after thalamic infarctions (evidence of aphasia after thalamic infarctions) (Fisher, 1959; Gorelick et al., 1984; Mori et al., 1986; Robin & Schienberg, 1990) as well as via the measurement of cerebral blood flow and metabolic changes in the thalamic nuclei (evidencing hypometabolism within those nuclei resulting into aphasia) (Metter et al., 1983a; Metter et al., 1984; Baron et al., 1986; Fasanaro et al., 1987; Metter et al., 1988). For a more comprehensive overview concerning thalamic language functions see for instance Botetz and Barbeau (1971), Brown (1975), Van Buren (1975), Crosson (1984) or Crosson (1992a: chapter 3).

These studies proved the following three thalamic nuclei, to play a major role in language:

1) The ventral lateral nucleus (speech functions) described by Ojemann and Ward (1971) and Crosson (1984).
2) The ventral anterior nucleus (language productive functions) described by Schaltenbrand (1965, 1975).
3) The pulvinar (language-receptive functions) described by Vilkki and Laitinen (1976); Ojemann, Fedio and Van Buren (1968).

It has been suggested moreover that these three nuclei project and receive input from the motor, premotor and temporoparietal cortex, the cortical areas of the dominant hemisphere to be associated with language (cf. Crosson, 1984: 493f). Details of the outputs and inputs from/to these nuclei are given in Table_Appendix I.

Table_Appendix I shows the inputs to and outputs from the three main thalamic nuclei associated with language (cf. Carpenter & Sutin, 1983: 514ff; Crosson, 1984: 493; Kiernan, 2005: 204f; Augustine, 2008: 250ff; Mendoza & Foundas, 2008: 195ff)

<table>
<thead>
<tr>
<th>nucleus</th>
<th>inputs</th>
<th>outputs</th>
</tr>
</thead>
<tbody>
<tr>
<td>ventral anterior nucleus</td>
<td>reticular formatio</td>
<td>frontal premotor cortex</td>
</tr>
<tr>
<td></td>
<td>frontal (premotor) cortex</td>
<td>insula</td>
</tr>
<tr>
<td>ventral lateral nucleus</td>
<td>primary motor cortex</td>
<td>primary motor cortex</td>
</tr>
<tr>
<td>pulvinar</td>
<td>temporoparietal cortex</td>
<td>temporoparietal cortex</td>
</tr>
</tbody>
</table>
III. Background regarding Wallesch and Papagno's model of the participation of subcortical structures in language

In 1982, Brunner et al. suggested a loop including the cortex, the striatum, the globus pallidus, the ventral lateral/ventral anterior thalamus as well as the frontal cortex, participating in language production. Wallesch et al. (1985) supported the assumption of this loop by measuring an increase in the rCBF within exactly these areas, in language production. The assumption of such a loop has further been supported by Damasio et al. (1982). More specifically, Damasio et al. (1982) assumed that all parts of the cortex send fibres to the striatum, in a topographically organized manner. Based on monkey studies it could be reported that rostral areas of the cerebral cortex project to the rostral striatum, whereas caudal areas project to the caudal striatum (Kemp & Powell, 1970). More specifically, associative areas of the prefrontal, temporal and parietal cortex are connected mainly with the caudate nucleus (= rostral part of the striatum) (Goldman & Nauta, 1977; Ragsdale & Graybiel, 1981; cf. Carpenter & Sutin, 1983: 579-611) and the primary motor cortex mainly with the putamen (= caudal part of the striatum) (Künzle, 1975; Carpenter & Sutin, 1983). In other words, all projections from discrete cortical areas terminate in restricted and well circumscribed striatal zones. Thus, the cortical input to the striatum can probably be seen as a 'point-to-point' transfer of information (cf. Parent & Hazrati, 1995: 94).

Wallesch and Papagno's (1988) model is moreover influenced by the assumption of cortico-cortically interconnected areas, projecting to circumscribed areas in the striatum (Yeterian & Van Hoesen, 1978; Van Hoesen, Yeterian & Lavizzo-Mourey, 1981). Cortical areas which are connected to another area in the cortex in ways of being functionally homogeneous, are also linked with overlapping cell groups in the striatum. Depending on the location of various cortical areas, a common projection can thus be found either to the head, to the body or to the tail of the caudate nucleus as well as to various parts of the putamen. This might imply that a given region of the striatum receives input not only from a particular area of the cortex, but also from other cortical areas reciprocally interconnected with that area (cf. Yeterian & Van Hoesen, 1978: 43).

Mountcastle (1957), Szentágothai (1975) as well as Popper and Eccles (1977) further indicated cellular modules within the cortex, sharing the same functions. All functionally common cortico-cortical projections have further been supposed to share a parallel modular organization (Goldman-Rakic, 1984; Philipps, Zeki & Barlow, 1984). These modules, originally only described for the primary sensory cortex (Mountcastle, 1957), have been assumed later on to be a general principle of cortical functional organization (Philipps, Zeki & Barlow, 1984).
IV. The two main capacities of 'long term memory'

Declarative, or explicit memory with data based information on the one hand and procedural or implicit memory with rule based information on the other hand (cf. Cohen & Squire, 1980: 209) are the two capacities of long term memory. Declarative memory is the aspect of human memory that stores facts. It is used in studying for school and storing facts as well as in recalling and retrieving past memories. Two types of declarative memory can be distinguished: Episodic and semantic memory (cf. Tulving, 1972: 385).

Episodic memory receives and stores information about happenings in particular places at particular times (the 'what', 'where' and 'when' is stored) (see for instance Nyberg et al.1996; Clayton & Dickinson, 1998; Tulving, 2002). More specifically, episodic memory refers to information that has been unique to an individual in a specific context, in the personal past ('I did such and such in such and such place at such and such time') (cf. Tulving, 1972: 389).

Semantic memory, on the other hand, consists of general knowledge about the world, acquired through media, education and other sources of information. It includes memory of faces, melodies, topographyies and of course also knowledge of language. Tulving (1972) supposed moreover that semantic memory contains organized knowledge about words and various verbal symbols, their meanings and referents, information about the relations among them and rules how these symbols and concepts interact with each other ('I think that the association between the words TABLE and CHAIR is stronger than the association between the words TABLE and NOSE') (cf. Tulving, 1972: 386).

Procedural memory on the other hand, subserves learning of new habits and skills, which are administered in daily life. Expressed in a more simple way, the 'how to do things' is stored in this memory system. Therefore, learning to ride a bike, learning to play a musical instrument can be referred to as typical exemplares of procedural memory. This memory subtype is also called implicit memory, because the process of learning happens unconsciously (implicit). Procedural memory is moreover shown in skill learning (musical instrument, priming tasks, simple classical conditioning and nonassociative learning such as habituation (Schacter, 1987; cf. Squire & Zola-Morgan, 1991: 1381).
V. Dynamic aphasia

The concept of dynamic aphasia, as first described by Luria and Tsvetkova, back in 1968, can basically be seen as a subtype of, or even the synonyme (cf. Goodglass, 1993: 212) of transcortical motor aphasia.

Lichtheim introduced the term transcortical motor aphasia in 1885, as a deficit of volitional language, with perseveration in all other domains. But as for all types of aphasic syndroms, there is also uncertainty in this case, regarding which domains are affected.

Agrammatism has been assumed to belong to this aphasia syndrome (cf. Goldstein, 1948: 294). In detail, Goldstein proposed that patients suffering from transcortical motor aphasia speak in telegram style and thus “[it] is hard for [them] to find the grammatical forms; [they] prefer the simplest ones, for instance, infinitives […]” (Goldstein, 1948: 81). (For a more recent overview on transcortical motor aphasia see for instance Alexander, 2003).

In a similar way, within dynamic aphasia, in addition to its major leitsymptom, in propositional language, also grammatical deficits, mainly in the syntactic domain, have been reported (Luria, 1970). For example, in 1970 Luria described a patient (case 8) who could still repeat single words and name objects but was unable to “[...] carry out any kind of involved narration...” and in addition “[...] his speech was limited to disconnected grammatically disordered word sequences [...]” (Luria, 1970: 207).

Thus it could be concluded that some grammatical disturbances are also part of dynamic aphasia.

More recently, (c.f. Mega & Alexander, 1994: 1827; D'Esposito & Alexander, 1995: 40) have pointed out that mainly generative language such as verbal fluency, sentence generation (to construct a sentence out of given words) and discourse are defective in this aphasia type. Contrastingly, spared responsive language such as repetition, comprehension and naming have been proven.

The concept of dynamic aphasia can possibly be linked back to the neurologist Karl Kleist, a pupil of Carl Wernicke. Kleist mentioned in his dissertation on “Weitere Untersuchungen an Geisteskranken mit psychomotorischen Störungen. Die hyperkinetischen Erscheinungen. Die Denkstörungen, hypochondrischen und affektiven Störungen bei akinetischen und hyperkinetischen Kranken” (Kleist 1909 cited from Neumärker & Bartsch, 2003: 420) the analogy of schizophrenia to neurological disorders marked by system degeneration, such as Huntington's Disease (cf. Pillmann & Möller, 2004: 155).

Kleist (1930) pointed out a specific type of language disturbance to arise in schizophrenia as well as in hyperkinetic disturbances. He labeled this type of language disturbance as 'Antriebsmangel der Sprache' (cf. Kleist, 1930: 101), which can basically be described as a type of transcortical motor aphasia, respectively a type of dynamic aphasia with a marked reduction of spontaneous speech output. Kleist included this type of language phenomena into his concept of 'alogische Denkstörunge' (cf. Kleist, 1930: 101f) in frontal lobe as well as subcortical patients. Consecutively he strengthened that this kind of 'disrupted thoughts' is characterized by a 'loss of drive in thinking'. Thus patients are not able to draw any conclusions of their thinking, start a thought, but cannot finish it and thus are globally unproductive in their thinking, which has hence also negative consequences onto language processes, such as grammar and syntax.

125This would again account for the concept of 'naturalness', as all complex forms are reduced to more simple forms.
Luria and Tsvetkova (1968) basically included Kleist's (1930) concept of a 'loss of drive in thinking' in their considerations on dynamic aphasia as they concluded that the basic deficit in this syndrome seems to be a disturbances in the 'predicative function of speech' (cf. Luria & Tsvetkova 1968: 297; Vygotsky, 1962).

In detail, Vygotsky (cf. 1962: 44) stated a link between the formulation of a thought and its expression in a phrase; this link he called 'inner speech'. This inner speech is supposed to be used by the individual to transform an idea to a verbal proposition. On the basis of this idea Luria and Tsvetkova (1968) concluded that “ [...] this inner speech with its predicative function, which takes part in forming the structure or scheme of a sentence, is disturbed in cases of dynamic aphasia” [...] “If the predicative function of inner speech is disturbed, a deterioration in 'propositioning' will follow” (Luria & Tsvetkova, 1968: 297).

These scientists tested their assumption on the basis of a patient with an hemorrhage in the posterior part of the frontal lobe, while performing various language tasks.

In a narrative task (talking about living and life in general) the patient was unable to construct a sentence, but when concrete questions were posed, the patient was able to finish the sentence. Thus it could be concluded that the patient's performance increased with a decrease in the degree of freedom (the more guided the production is, the better it gets).

In a syntactic task, the patient was given two words (house and hen) and was asked to construct a whole sentence, including both words. The patient was unable to do so and produced the following utterance instead ‘House...oh...house...I can't...and hen...house...oh, dear me...it is house...and nothing...’ (cf. Luria & Tsvetkova, 1968: 301).

But when this patient was provided with visual cues (e.g., pieces of paper; the number of cues in a row reflected the number of words in the sentence required), and could point to these cues while verbalizing the sentence, verbal output improved. On the basis of this evidence (cf. Luria & Tsvetkova, 1968: 375) suggested that this patient was unable to transform spontaneously a thought/concept into a linear sequence of lexical items (e.g. in terms of syntax), due to a problem of 'inner speech'.

VI. Examination of conditions_language screening

In order to run a multivariate analysis of variance for the raw data of this thesis, conditions of the raw data have to be examined in advance. In detail, within the examination of conditions, normal distribution and homogeneity of variance have also been examined by using the Kolmogorov-Smirnov test of goodness of fit and the Levene-Test, to account for the application of an analysis of variance.

Results show that normal distribution could not be approved for language production and language comprehension\(^\text{126}\) (\(p < 0.05\) for language production and \(p < 0.05\) for language comprehension).

Table_Appendix II shows the z-value and the p-value of the Kolmogorov-Smirnov Test, for language production and language comprehension in general

<table>
<thead>
<tr>
<th>language screening</th>
<th>Kolmogorov-Smirnov-Test</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>z</td>
<td>p</td>
<td></td>
</tr>
<tr>
<td>language production</td>
<td>1.616</td>
<td>0.011</td>
<td></td>
</tr>
<tr>
<td>language comprehension</td>
<td>1.397</td>
<td>0.040</td>
<td></td>
</tr>
</tbody>
</table>

Further, results indicate that normal distribution could not be approved for all aspects of the language production subtest (\(p > 0.05\) for semantic fluency, \(p < 0.01\) for article assignment, \(p < 0.01\) for preposition assignment, \(p < 0.05\) for inflection, \(p < 0.01\) for derivation/conversion as well as \(p < 0.01\) for syntax_production).

For the language comprehension subtest, normal distribution couldn't be approved for all subtasks either (\(p < 0.01\) for language comprehension_general, \(p < 0.01\) for language comprehension_visual, \(p < 0.01\) for definitions, \(p < 0.01\) for questionnaires, \(p < 0.01\) for metaphors, \(p < 0.05\) for syntax_comprehension as well as \(p > 0.05\) for reading comprehension).

Table_Appendix III shows the p-value and the z-value of the Kolmogorov-Smirnov Test, for all subtasks of the language production subtest separately

<table>
<thead>
<tr>
<th>language production</th>
<th>Kolmogorov – Smirnov – Test</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>z</td>
<td>p</td>
<td></td>
</tr>
<tr>
<td>semantic fluency</td>
<td>0.468</td>
<td>0.981</td>
<td></td>
</tr>
<tr>
<td>article assignment</td>
<td>2.028</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>preposition assignment</td>
<td>1.624</td>
<td>0.010</td>
<td></td>
</tr>
<tr>
<td>inflection</td>
<td>1.546</td>
<td>0.017</td>
<td></td>
</tr>
<tr>
<td>derivation/conversion</td>
<td>1.730</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>syntax_production</td>
<td>2.560</td>
<td>0.000</td>
<td></td>
</tr>
</tbody>
</table>

\(\text{126}\)There is violation of normal distribution if \(p \leq 0.05\).
Table Appendix IV shows the p-value and the z-value of the Kolmogorov-Smirnov Test, for all subtasks of the language comprehension subtest separately

<table>
<thead>
<tr>
<th>language comprehension</th>
<th>Kolmogorov – Smirnov - Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>z</td>
</tr>
<tr>
<td>single word understanding _general</td>
<td>1.663</td>
</tr>
<tr>
<td>single word understanding _visual</td>
<td>2.304</td>
</tr>
<tr>
<td>definitions</td>
<td>3.110</td>
</tr>
<tr>
<td>questionnaires</td>
<td>3.053</td>
</tr>
<tr>
<td>metaphors</td>
<td>2.859</td>
</tr>
<tr>
<td>syntax_comprehension</td>
<td>1.445</td>
</tr>
<tr>
<td>reading comprehension</td>
<td>1.267</td>
</tr>
</tbody>
</table>

Results show that homogeneity of variance\(^{127}\) could not be approved for language production and language comprehension (p < 0.05 for language production and p < 0.05 for language comprehension).

Table Appendix V shows the p-value of the Levene-Test, for language production and language comprehension in general

<table>
<thead>
<tr>
<th>language screening</th>
<th>Levene-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p</td>
</tr>
<tr>
<td>language production</td>
<td>0.004</td>
</tr>
<tr>
<td>language comprehension</td>
<td>0.000</td>
</tr>
</tbody>
</table>

Further, results indicate that homogeneity of variance could not be approved for all aspects of the language production subtest (p > 0.05 for semantic fluency, p < 0.05 for article assignment, p < 0.05 for preposition assignment, p < 0.05 for inflection, p < 0.01 for derivation/conversion as well as p < 0.01 for syntax_production).

For the language comprehension subtest, normal distribution couldn't be approved for all subtasks either (p < 0.01 for language comprehension_general, p < 0.01 for language comprehension_visual, p < 0.01 for definitions, p < 0.01 for questionnaires, p < 0.01 for metaphors, p < 0.01 for syntax_comprehension as well as p > 0.05 for reading comprehension).

\(^{127}\)There is violation of homogeneity of variance if p ≤ 0.05.
Table Appendix VI shows the p-value of the Levene-Test, for all subtasks of the language production subtest separately

<table>
<thead>
<tr>
<th>language production</th>
<th>Levene-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( p )</td>
</tr>
<tr>
<td>semantic fluency</td>
<td>0.927</td>
</tr>
<tr>
<td>article assignment</td>
<td>0.013</td>
</tr>
<tr>
<td>preposition assignment</td>
<td>0.008</td>
</tr>
<tr>
<td>inflection</td>
<td>0.005</td>
</tr>
<tr>
<td>derivation/conversion</td>
<td>0.000</td>
</tr>
<tr>
<td>syntax_production</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Table Appendix VII shows the p-value of the Levene-Test, for all subtasks of the language comprehension subtest separately

<table>
<thead>
<tr>
<th>language comprehension</th>
<th>Levene-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( p )</td>
</tr>
<tr>
<td>single word understanding_general</td>
<td>0.000</td>
</tr>
<tr>
<td>single word understanding_visual</td>
<td>0.000</td>
</tr>
<tr>
<td>definitions</td>
<td>0.000</td>
</tr>
<tr>
<td>questionnaires</td>
<td>0.000</td>
</tr>
<tr>
<td>metaphors</td>
<td>0.000</td>
</tr>
<tr>
<td>syntax_comprehension</td>
<td>0.000</td>
</tr>
<tr>
<td>reading comprehension</td>
<td>0.444</td>
</tr>
</tbody>
</table>
VII. Semantic fluency

Based on all namings, for the five given supercategories (animals, vegetables, fruits, clothing and birds), provided by patients and controls together, excluding repetitions and neologism errors, the following semantic subcategories were tried to be worked out on the basis of zoological and semantic categories:

For animals:
- living environment: domestic (house, barn, cage)
- living environment: wood, grassland or in mountane regions
- living environment: icy regions, steppe, desert of jungle
- living environment: air
- living environment: water

For vegetables:
- leavy vegetables and salads
- cabbages
- fruitive vegetables
- bulb vegetables
- onion-like vegetables, spices and herbs
- legumes
- sweet herbages

For fruits:
- stone fruits
- pomaceous fruits
- paring fruits
- citrus, exotic and souther fruits that would not naturally grow in Austria
- berries

For clothing:
- outerwear (shoulders until navel)
- underwear (navel until ankle)
- underwear (what you wear under general clothes) and sleeping wear
- outer wear (what you wear over general clothes)
- accessoires
- feastful clothing
- working clothing
- sports and casual clothing

For birds:
- not native Austrian birds and birds usually not living in the wild
- owl like birds
- birds of prey
- singing birds
- chickens and ducks
- ratites
- doves
- peckers
On the basis of that supercategories, the number of clusterings, switchings, 2_clusters, 3_clusters, 4-10_clusters has been constructed.

- **Tiere/animals:**
  Lebensraum: Domestiziert (Haus, Stall oder Käfig) – living environment: domestic (house, barn, cage)
  Esel – donkey
  Ferkel – piglet
  Frettchen – ferret
  Goldfisch – goldfish
  Hahn – rooster
  Henne/Huhn – hen
  Hamster – hamster
  Hund – dog
  Kalb – calf
  Katze – cat
  Kuh – cow
  Küken – poult
  Lamm – lamb
  Lippizaner – lippizaner (special type of a horse, famous for the region I am born)
  Meerschweinchen – guinea pig
  Ochse – bullock
  Pferd – horse
  Rind – cattle
  Schaf – sheep
  Schwein – pig
  Stier – bull
  Wellensittich – budgie
  Ziege – goat
  Zwerghuhn – bantam

Lebensraum: Wald, Wiese oder Gebirge – living environment: wood, grassland or in mountane regions:
  Ameise – ant
  Assel – wood louse
  Auerhahn – wood grouse
  Bär – bear
  Biene – bees
  Birkhuhn – black grouse
  Bergziege – montane goat
  Braunbär – brown bear
  Dachs – badger
  Dammwild – fallow deer
  Eichhörnchen – squirrel
  Elch – elk
  Fasan (Wildfasan)/Goldfasan – pheasant/gold pheasant
  Fliege – fly
  Floh – flea
  Fledermaus – bat
Frosch – frog
Fuchs – fox
Gams – chamois
Grille – cricket
Hase – bunny
Hirsch – deer
Hornisse – hornet
Hummel – bumble bee
Igel – hedgehog
Itis – polecat
Kaninchen – rabbit
Käfer – beetle
Kroete – toad
Laus – louse
Luchs – lynx
Marder – marten
Marienkäfer – ladybeetle
Maulwurf – mole
Maus – mouse
Murmeltier – marmot
Ratte – rat
Rebhuhn – partridge
Reh – roe deer
Rehbock – roe buck
Rentier – reindeer
Salamander – salamander
Schlange – snake
Schmetterling – butterfly
Schnecke – snail
Schwarzbär – black bear
Spinne – spider
Steinhörnchen – mosquito
Steinbock – ibex
Wespe – wasp
Wildsau/Wildschwein – wild sow
Wolf – wolf
Wurm – worm

Lebensraum: Eis, Steppe, Wüste oder Dschungel – living environment: icy regions, steppe, desert of jungle
Affe – monkey
Ameisenbär – anteater
Antilope – antelope
Bison – bison
Büffel – buffalo
Echse – saurian
Eisbär – polar bear
Elefanter – elephant
Faultier – sloth
Gepard – cheetah
Giraffe – giraffe
Gorilla – gorilla
Gürteltier – armadillo
Känguru – kangaroo
Kamel – camel
Kanarienvogel – canary
Koala – koala
Krokodil – crocodile
Leguan – iguana
Leopard – leopard
Löwe – lion
Nashorn/Rhinozeros – rhino
Nilpferd/Flusspferd – hippo
Orang Utan – orang utan
Panda – panda
Pavian – baboon
Papagei – parrot
Pinguin – penguin
Puma – puma
Reptil – reptile
Schimpanse – chimp
Springmaus – kangaroo mouse
Strauß – ostrich
Tarantel – tarantula
Tiger – tiger
Zebra – zebra

Lebensraum: Luft – living environment: air
Ara – macaw
Adler – eagle
Amsel – blackbird oder ouzel
Bussard – buzzard
Drossel – thrush
Eichelhäher – jaybird
Elster – magpie
Ente – duck
Falke – hawk
Fink – finch
Gans – goose
Geier – vulture
Habicht – goshawk
Kanarienvogel – canary
Kräh – crow
Meise – tit
Papagei – parrot
Rotkehlchen – robin
Schwalbe – swallow
Star – starling
Spatz oder Sperling – sparrow
Specht – woodpecker
Storch – stork
Taube – dove

**Lebensraum: Wasser – living environment: water**
Barsch – perch
Delphin – dolphin
Dorsch – codling
Fisch – fish
Forelle – trout
Hai – shark
Hecht – pike
Karpfen – carp
Krebs – crab
Lachs – salmon
Muschel – mussel
Otter – otter
Schildkröte – tortoise
Seestern – starfish
Wal – whale
Walross – walrus
Wasserschildkröte – snapping turtle
Zander – zander

- **Gemüse/vegetables:**
**Blatt- und Salatgemüse – leavy vegetables and salads:**
Artischocke – artichoke
Blattgemüse – leavy vegetables
Blaukraut – red cabbage
Endiviensalat – endive
Kopfsalat/Häuptlsalat – lettuce
Kraut – cabbage
Löwenzahnsalat – dandelion salad
Mangold – mangold
Radicchio – radicchio
Rotkraut – red cabbage
Ruccola – rocket
Sauerkraut – sauerkraut
Salat – salad
Spinat – spinach
Vogerlsalat/Rapunzel – lamb’s lettuce

**Kohlgemüse – cabbages:**
Blumenkohl/Karfiol – cauliflower
Brokkoli – broccoli
Chinakohl – chinese cabbage
Grünkohl – green cabbage
Kohl – cabbage or collard
Kohlrabi – kohlrabi
Kohlsprossen/Rosenkohl – brussels sprouts
Fruchtgemüse – fruitive vegetables:
Aubergine/Melanzani – aubergine
Gurke – cuke/cucumber
Kürbis – pumpkin
Paprika – pepper
Pfefferoni – pepperoni
Tomate/Paradeise – tomato
Zucchini – zucchini

Knollengemüse – bulb vegetables:
Babykarotten – baby carrots
Karotte/Möhre – carrot
Kartoffel/Erdapfel – potato
Kren/Merrettich – horseradish
Radieschen – radish
Rettich – radish
Ronen/rote Rüben – beetroot
Rübe – beet
Schwarzer Rettich – black radish
Spargel – asparagus
Zuckerrübe – sugar beet

Zwiebelgemüse, Gewürze und Kräuter – onion like vegetables, spices and herbs:
Chilli – chilli
Fenchel – fennel
Knoblauch – garlic
Kresse – cress
Lauch – leek
Lavendel – lavender
Majoran – marjoram
Nusskraut – nut cabbage
Petersilie – parsley
Porree – leek
Pfefferschote – black pepper
Salbei – sage
Schalotte – shallot
Schnittlauch – chive
Sellerie – celery
Thymian – thyme
Zwiebel – onion

Hülsenfrüchte – legumes:
Bohnen – beans
Buschbohnen – bush bean
Erbsen – peas
Fisolen – pole beans
Linsen – lenses
Stangenbohnen – runner beans
Süßgräser – sweet herbages
Mais – corn
Reis – rice

- Obst/fruits:
Steinobst – stone fruits:
Kirsche – cherry
Kriecherl – bullace
Marille – apricot
Nektarine – nectarine
Pfirsich – peach
Pflaume – plum
Ringlotte – greengage
Weichsel – morello/sour cherry
Zwetschke – plum

Kernobst – pomaceous fruits:
Apfel – apple
Birne – pear
Granny Smith Äpfel – granny smith apple
Jonathan Äpfel – jonathan apple

Schalenobst – paring fruits:
Erdnuss – peanut
Haselnuss – hazelnut
Kastanie – chestnut
Kokosnuss – coconut
Mandel – almond
Nuss – nut
Walnuss – walnut

Zitrus- Exotische und Süßfrüchte – citrus- exotic and southern fruits that would not usually grow in Austria:
Ananas – ananas
Avocado – avocado
Apfelsine – orange
Banane – banana
Feige – fig
Granatapfel – pomegranate
Grapefruit – grapefruit
Honigmelone – rock melon
Kaki – kaki
Kiwi – kiwi fruit
Klementine – satsuma
Litchi – leechee
Mandarine – tangerine
Mango – mango
Maracuja – passion fruit
Melone – melon
Orange – orange
Papaya – papaya
Pomelo – pommelo
Quitte – quince
Satsuma – satsuma
Wassermelone – water melon
Zitrone – lemon
Zuckermelone – sugar melon

Beeren – berries:
Brombeere – blackberry
Erdbeere – strawberry
Heidelbeere/Blaubeere – blueberry
Himbeere – raspberry
Holunder – elder
Johannisbeere – currant
Jostabeere – jostaberry
Physalis – cape gooseberry
Preiselbeere – cowberry
Rote Ribisel – red currant
Schilchertraube/Weintrauben/Trauben – grapes
Schwarzbeere – dewberry
Schwarze Ribisel – black currant
Stachelbeere – gooseberry
Waldbeeren – wild berries

• bekleidung/clothing:
Oberbekleidung (Schultern bis Nabel) – outerwear (shoulders until navel):
Bluse – blouse
Gilet – gilet
Hemd – shirt
Kapuzenpullover – hoody
Kurzärmeliges T-shirt – short sleeved T-shirt
Langärmeliges T-shirt – long sleeved T-shirt
Polo-Leibchen – polo shirt
Pullover – pullover
Pullunder – slipover
Rollkragenpullover – turtleneck
Sonnen- Top – sun top
Spaghettitop/Top/Trägerleiberl – top
Sport- Top – sport top
Sweater – sweater
Sweatshirt – sweatshirt
T-shirt /Leiberl – T-shirt
Weste – vest

Unterbekleidung (Nabel bis Knöchel) – underwear (navel until ankle):
Bluejeans – blue jeans
Cordhose – corduroys
Hose – pants
Jeans – jeans
Kleid – dress (ist eine Ausnahme, weil keine Unterbekleidung von Nabel bis Knöchel; is an exception as it is no underwear from the navel until the ankle)

Kurze Hose – short
Leggins – legging
Leinenhose – ducks
Minikleid – mini
Rock – skirt
Stretchhose – stretch pants

**Unterwäsche und Nachtbekleidung – underwear (what you wear under general clothes) and sleeping wear:**

Bademantel – bathrobe
BH – bra
Boxershorts/Unterhose – underpants
Dessous/Reizwäsche – lingerie
Kniestümpfe/Stutzen – stockings
lange Unterhose – long johns
Nachthemd – nightdress
Hot Pants/Pants – pants
Pyjama – pyjama
Pyjamahose – pyjama pants
Unterleiberl/Unterhemd – undershirt
Unterrock – underskirt
Skiunterwäsche – ski underwear
Socken – socks
String-Tanga – thong
Strumpfhose – tights

**Überbekleidung – outer wear (over general clothes):**

Anorak – anorak
Daunenjacke – down jacket
Jacke – jacket
Lederjacke – leather jacket
Pelzmantel – fur coat
Poncho – poncho
Regenmantel – rain coat
Regenjacke – rain jacket
Regenbekleidung – rainwear
Schladminger – there is no word in English, typical Styrian style of jacket
Sommermantel/Sommerjacke – summer jacket
Trachtenweste – livery vest
Trenchcoat/Mantel – coat
Wetterfleck/Cape/Umhang – cape
Windjacke – wind cheater
Wintermantel/Winterjacke – winter jacket

**Accessoires – accessoires:**

Badehaube – bathing cap
Fäustlinge – mittens
Fingerhandschuhe – gloves
Halstuch – bandanna
Handschuhe – gloves
Handwärmer – muff
Haube – cap
Helm – helmet
Hut – hat
Fliege – bow tie
Gürtel – belt
Kappe/Mütze – cap
Kopftuch – bandanna
Krawatte – tie
Pelzhandschuhe – furry gloves
Pelzkragen – furry collar
Regenschirm – umbrella
Schal – scarf
Schleier – bridal veil
Stirnband – bandeau
Stola – stola
Strumpfgürtel – garter belt
Schürze – apron
Tuch – shawl
Turban – turban
Verkleidung – costume
Wollhaube – wool cap

**Festliche Bekleidung – feastful clothing:**
Abendkleid – evening gown
Anzug – suit
Ballkleid – ball gown
Hosenanzug – trouser suit
Kostüm – suit for ladies
Sakko – jacket
Sonntagsanzug – Sunday's suit
Steireranzug – Styrian suit

**Arbeitsbekleidung – working clothing:**
Arbeitsbekleidung – work clothing
Berufsbekleidung – job clothing
Montur – uniform
Schürze – apron

**Sport- und Freizeitbekleidung – sports- and casual clothing:**
Badeanzug – bathing suit
Badehose – trunks
Bergjacke/Kletterjacke – hiking/climbing jacket
Bikini – bikini
Gymnastikhose – leggins
Jogginganzug/Trainingsanzug – track suit
Jogginghose – track suit trousers
Laufbekleidung – run clothes
Skianorak – ski anorak
Skigewand – ski clothes
Skihose – salopettes
Skioveral/Skianzug – skiing suit
Sporthemd – sport shirt
Sport-Top – sports top
Trainingshose – track pants
Trainingsjacke – tracksuit top
Turnhose – gym trousers
Wanderhose – hiking pants

**Schuhe – shoes:**
Baustiefel – boots to wear for construction works
Bergschuhe/Wanderschuhe – hiking shoes
Flip-Flops – thongs
Gummistiefel – rubber boots
Gymnastikschuhe – gymnastic shoes
Laufschuhe – jogging shoes
Patschen/Pantoffeln/Hausschuhe – slippers
Pelzstiefel – furry boots
Sandalen – sandals
Skischuhe – ski shoes
Sportschuhe – sports shoes
Stiefel – boots
Tourenskischuhe – tours ski shoes
Turnschuhe – pumps

- **Vögel/birds:**
  Nicht einheimische Vögel, oder Vögel die man in der Natur nicht findet – not native
  Austrian birds, or birds not living in the wild:
Aasgeier – carrion vulture
Ara – macaw
Eisvogel - kingfisher
Emu – emu
Flamingo – flamingo
Kakadu – cockatoo
Kanarienvogel – canary
Kiwi – kiwi
Kolibri – flower bird or hummingbird
Kondor – condor
Möwe – gull
Papagei – parrot
Pelikan – pelican
Pinguin – penguin
Sittich – parakeet
Strauß – ostrich
Wellensittich – budgie
**Eulenartige – owl like birds:**
Eule – owl
Kauz – fogy
Kuckuck – cuckoo
Uhu – eagle owl

**Greifvögel – birds of prey:**
Adler – eagle
Bussard – buzzard
Falke – falcon
Gänsegeier – griffon vulture
Geier – vulture
Habicht – goshawk
Hühnerhabicht – chicken hawk
Mäusebussard – common bussard
Bartgeier/Lämmergeier – lammergeier
Seeadler – sea eagle
Steinadler – golden eagle
Sperber – sparrow hawk

**Singvögel – singing birds:**
Amsel – ouzel
Bachstelze – wagtail
Baumläufer – treecreeper
Blaumeise – tomtit
Buchfink – chaffinch
Dohle – daw
Drossel – throstle
Eichelhähner – jaybird
Elster – magpie
Fink – finch
Gimpel – bullfinch
Grünling – greenfinch
Häher – jay
Kleiber – nuthatch
Kohlmeise – great tit
Krähe – crow
Lerche – lark
Mauersegler – hawk swallow
Mehlschwalbe – house martin
Meise – tit
Nachtigall – nightingale
Rabe – raven
Rauchschwalbe – barn swallow
Rotkehlchen – redbreast
Rotschwänzchen – redstart
Schwalbe – swallow
Spatz oder Sperling – sparrow
Star – starling
Stieglitz – goldfinch
Tannenhäher – nutcracker
Wiedehopf – hoopoe
Zaunkönig – wren
Zebrafink – zebra finch
Zeisig – siskin

**Hühner- und Entenvögel – chickens and ducks:**
Auerhuhn – capercailzie
Birkhuhn – black grouse
Ente – duck
Fasan – pheasant
Gans – goose
Goldfasan – gold pheasant
Huhn/Henne – hen
Perlhuhn – guinea fowl
Rebhuhn – partridge
Schneehuhn – snow grouse
Schwan – swan
Wachtel – quail
Zwerghuhn – bantam

**Lauf- und Schreitvögel – ratites:**
Fischreiher – heron
Kranich – crane
Reiher – egret
Schwarzstorch – black ostrich
Storch – stork
Trappe – bustard
Weißstorch – white ostrich

**Taubenartigen – doves:**
Brieftaube – carrier pigeon
Taube/Haustaube – dove

**Spechtvögel – peckers:**
Buntspecht – great spotted woodpecker
Schwarzspecht – black woodpecker
Specht – pecker
VIII. Examination of conditions_clustering

Normal distribution has been examined by using the Kolmogorov-Smirnov test of goodness of fit to account for the application of an analysis of variance. Results show that normal distribution could be approved for most aspects of semantic fluency tasks\(^{128}\), (\(p > 0.05\)) for 2_clusters_corr, corrected for the total number of correct items; for 3_clusters_corr, corrected for the total number of correct items (\(p > 0.05\)), as well as for 4-10_clusters_corr, again corrected for the total number of correct items (\(p > 0.05\)). Against that normal distribution could not be approved for clusterings_corr, corrected for the total number of correct items (\(p < 0.05\)), as well as for switchings_corr, corrected for the total number of correct items (\(p < 0.01\)).

Table Appendix VIII shows the p-value and the z-value of the Kolmogorov-Smirnov Test, for clusterings_corr, switchings_corr, 2_clusters_corr, 3_clusters_corr, 4-10_clusters_corr, separately

<table>
<thead>
<tr>
<th>semantic fluency</th>
<th>Kolmogorov-Smirnov-Test</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>z</td>
<td>p</td>
<td></td>
</tr>
<tr>
<td>clusterings_corr</td>
<td>1.739</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>switchings_corr</td>
<td>2.214</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>2_clusters_corr</td>
<td>1.107</td>
<td>0.172</td>
<td></td>
</tr>
<tr>
<td>3_clusters_corr</td>
<td>1.265</td>
<td>0.082</td>
<td></td>
</tr>
<tr>
<td>4-10_clusters_corr</td>
<td>0.791</td>
<td>0.560</td>
<td></td>
</tr>
</tbody>
</table>

Homogeneity of variance has been approved by using the Levene-Test. Results indicate that homogeneity of variance could be approved for all aspects of semantic fluency (\(p > 0.05\) for clusterings_corr; \(p > 0.05\) for switchings_corr; \(p > 0.05\) for 2_clusters_corr; \(p > 0.05\) for 3_clusters_corr; \(p > 0.05\) for 4-10_clusters_corr).

Table Appendix IX shows the p-value of the Levene-Test, for all aspects of semantic fluency separately

<table>
<thead>
<tr>
<th>semantic fluency</th>
<th>Levene-Test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>clusterings_corr</td>
<td></td>
<td>0.255</td>
</tr>
<tr>
<td>switchings_corr</td>
<td></td>
<td>0.745</td>
</tr>
<tr>
<td>2_clusters_corr</td>
<td></td>
<td>0.188</td>
</tr>
<tr>
<td>3_clusters_corr</td>
<td></td>
<td>0.910</td>
</tr>
<tr>
<td>4-10_clusters_corr</td>
<td></td>
<td>0.235</td>
</tr>
</tbody>
</table>

\(^{128}\)There is violation of normal distribution and homogeneity of variance if \(p \leq 0.05\).
IX. Plural formation_errors

Patients:

0-plural:
Eine Katze – viele Katze
Ein Känguru – viele Känguru

e-plural:
Ein Bär – viele Bäre 2x

double plural -ss and e-plural:
Ein Känguru – viele Kängurusse

umlaut and e-plural:
Ein Hund – viele Hunde 2x

lexicalised item and corresponding correct plural:
Ein Känguru – viele Kakadus

phonological proximity and application of inflectional morpheme instead of plural morpheme:
Ein Huhn – viele Huhndes

autocorrection_right:
Eine Katze – 1 viele Kätzchen, 2. viele Katzen1

non response:
Ein Kaninchen

Controls:

0-plural:
Eine Maus – viele Maus

e-plural:
Ein Bär – viele Bäre

deletion of final vowel and e-plural:
Ein Känguru – viele Kängure
X. Providing deverbal and denominal adjectives_errors

Patients:

neologism:
Buckel – bückelig
Furcht – furchtig 2x
lesen – lesig
achten – achterlich
Mutter – muttrig
Glück – glückig
lachen – lachbar

lexical intrusion_noun:
Furcht – Früchte
lachen – zum Freuen
Wind – Sturm

lexical intrusion_adjective:
lesen – durchlässig

deverbal noun:
lachen – zum Lachen
achten – Achtung 3x

denominal verb:
Holz – holzen
Buckel – bücken/buckeln
Mutter – bemuttern
Buckel – buckeln 2x
Furcht – fürchten 2x

compound_noun:
Wind – Windrad
Holz – Holzmaserung
Mutter – Muttertag
Mutter – Urmutter
Glück – Glückspilz

saying:
Glück – Glück auf

derived verb:
lesen – verlesen

derived denominal verb_participle:
Mutter – bemutternd
**denominal verb_participle:**
Buckel – gebückt
Furcht – gefürchtet

**compound_participle:**
Furcht – furchterregend 2x

**derived verb_adjective:**
achten – verächtlich

**diminuitiv:**
Holz – Hölzchen

**autocorrection_right:**
lesen – Buch, leserlich
Furcht – fürchten, fürchterlich

**help_right:**
lesen – lesbar 3x
Glück – glücklich 2x
achten – achtsam 2x
Holz – holzig
Furcht – furchtsam
Wind – windig

**non response:**
Wind 7x
Glück 4x
achten 5x
lachen 5x
Holz 2x
Buckel
Mutter

**Controls:**

**neologism:**
achten – ächtlich

**lexical intrusion_adjective:**
Furcht – ängstlich

**deverbal noun:**
achten – Achtung

**denominal verb:**
Mutter – bemuttern

**derived denominal verb_participle:**
Mutter – bemutternd 2x
**compound_participle:**
Furcht – furchterregend

**lexical intrusion_verb_participle:**
lachen – lächelnd 2x

**help_right:**
achten – achtsam

**non response:**
achten 2x
lachen
Buckel
Holz
XI. Providing deverbal and deadjectival nouns_errors

Patients:

comparison:
schnell – schneller laufer
schnell – schneller – am schnellsten

neologism:
gut – die Gutheit 2x
nass – die Nassheit 2x
vergelten – Vergeltbarkeit
streben – Strebhaftigkeit
heiter – Heiterschaft
groß – die Großig
streben – die Strebung
heiter – die Heiterung
heiter – heitrig
schnell – die Schnell
gut – Güte
heiter – das Heiter

nonstandard_nominalisation:
groß – die Große 2x
groß – die Großen
schnell – die Schnelle 2x
schnell – das Schnelle
gut – die Guten

adjective phrase:
heiter – heitere Person

lexical intrusion_noun:
groß – der Gruß
heiter – Heizung
vergelten – die Vergebung
werben – das Verb
gut – das Gut
gut – die Gutmütigkeit

lexical intrusion_adjective:
gut – gütig
heiter – lustig
nass – feucht

lexical intrusion_verb:
streben – bemühen

autocorrection_right:
groß – die Großig, die Größe
**help_right:**
groß – die Größe
schnell – die Schnelligkeit

**non response:**
gut 5x
essen 6x
groß 7x
werben 8x
streiben 7x
vergelten 9x
vergangen 4x
nass 4x
heiter 2x
schnell 3x

**Controls:**

**nonstandard_nominalisation:**
streben – die Streber
heiter – die Heiteren
gut – die Guten
nass – die Nassen
schnell – die Schnellen
vergelten – die Vergelter
streiben – der Streber 2x
gut – der Gute
gut – der Gute

**lexikal intrusion_noun:**
streben – die Strebe

**help_right:**
streben – die Strebsamkeit 4x
gut – die Güte
groß – die Größe

**non response:**
gut 2x
streiben 3x
vergelten 4x
schnell
XII. Sequencing of sentence parts errors

Patients:\[2^{129}\]:

Sentence 1 _6 sentence parts
/in den Ferien/gehen/Die Kinder/gerne/klettern./
/Die Kinder/in/den Ferien/gerne/klettern./gehen

Sentence 3 _5 sentence parts
/der neue Torwart/hat/nicht/gehalten./den Ball/

Sentence 4 _7 sentence parts

Sentence 5 _6 sentence parts
/Die Bestzeit/des Läufers/drei Minuten/unter/soll/liegen./
/Die Bestzeit/soll/unter/drei Minuten/liegen./des Läufers/

Sentence 6 _5 sentence parts
/Muss/immer/einen Gewinner/geben?/

Sentence 9 _7 sentence parts
/Ich/länger/mir/dieses langweilige Spiel/nicht/mehr/ansehen./\[mag fehlen\]

Sentence 11_5 sentence parts
/Der engagierte Teamchef/sein Team/informiert/über/eine neue Strategie./

Sentence 12 _5 sentence parts
/Der ehemalige Präsident/überreicht/den Pokal/dem überglücklichen Sieger/des Clubs/
/Der ehemalige Präsident/des Clubs/dem überglücklichen Sieger/überreicht/den Pokal./

Sentence 13 _6 sentence parts
/Der Trainer/dem traurigen Verlierer/die Hand/auf/die Schulter/legte/
/Der Trainer/dem traurigen Verlierer/legte/die Hand/auf/die Schulter./
/Der Trainer/legte/die Hand/auf die Schulter/dem traurigen Verlierer/
/Der Trainer/dem traurigen Verlierer/die Hand/auf/die Schulter./

Sentence 14 _10 sentence parts
/Nachdem/seine Teamkollegen/den Spieler/die rote Karte/erhalten hatte,/gab/er/den Ball/an/weiter./
/Nachdem/die rote Karte/den Spieler/erhalten hatte,/gab/er/den Ball/an/seine Teamkollegen/
Nachdem seine Teamkollegen die rote Karte erhalten hatte, gab er den Ball an den Spieler weiter.

**Sentence 15_5 sentence parts**
/Was bekommt als Preis? der Sieger des Rennens?

**Sentence 17_5 sentence parts**
Wo/im nächsten Winter/das Skispringen stattfinden?

**Sentence 18_6 sentence parts**
/Wann zum Training möchten ihr wieder kommen?/
/Wann möchte zum Training ihr wieder kommen?/
/Wann ihr wieder möchten zum Training kommen?/

**Sentence 19_7 sentence parts**
/Wie könnte es den Gegner gelingen, uns zu besiegen?/
/Wie könnte es gelingen, den Gegner uns zu besiegen?/
/Wie könnte es gelingen, den Gegner zu besiegen?/uns
/Wie könnte es gelingen, den Gegner zu uns besiegen?/
/Wie könnte es gelingen, den Gegner zu besiegen?/uns fehlt

**Controls:**

**Sentence 8_6 sentence parts**
/Man kann die Grundregeln leicht des Basketballs lernen./ (autocorrection_right)

**Sentence 9_7 sentence parts**
/Ich mag dieses langweilige Spiel nicht mehr/länger ansehen./ (mir fehlt)

**Sentence 13_6 sentence parts**
/Der Trainer legte auf die Schulter dem traurigen Verlierer die Hand/
/Der Trainer legte die Hand auf dem traurigen Verlierer die Schulter/

**Sentence 14_10 sentence parts**
/Nachdem die rote Karte der Spieler erhalten hatte, gab er den Ball an seine Teamkollegen weiter/
Sentence 15_5 sentence parts
/Was/der Sieger des Rennens/ bekommt/ als Preis?/

Sentence 17_5 sentence parts
/Wo/im/nächsten Winter/das Skirennen/ wird/ stattfinden?/
/Wo/im/nächsten Winter/ wird/ das Skirennen/ stattfinden?/
XIII. Types of sequencing errors

Patients:

verbal phrase (predicate):
/Die Kinder/in/den Ferien/gerne/klettern./
/Der ehemalige Präsident/dem überglücklichen Sieger/überreicht/den Pokal./
/Der Trainer/dem traurigen Verlierer/die Hand/auf/die Schulter./

auxiliary:
/Wo/im nächsten Winter/das Skispringen/wird/stattfinden?/

modal verb:
/zum/ersten Mal/darf/Die kleine Susi/alleine/ins Wasser/gehen./
/Die Bestzeit/des Läufers/drei Minuten/unter/soll/liegen./
/Ich/länger/mir/dieses langweilige Spiel/nicht mehr/ansehen./
/Wann/ihr/wieder/möchtet/zum Training/kommen?/

O Gen:
/Die Bestzeit/soll/unter/drei Minuten/liegen./
/Der ehemalige Präsident/überreicht/den Pokal/dem überglücklichen Sieger/des Clubs/
/Der Trainer/dem traurigen Verlierer/die Hand/auf/die Schulter./

O Dat:
/Der ehemalige Präsident/überreicht/den Pokal/dem überglücklichen Sieger/des Clubs/
/Der Trainer/dem traurigen Verlierer/die Hand/auf/die Schulter./

O Akk:
/Der neue Tormann/hat/nicht/gehalten./
/Der ehemalige Präsident/überreicht/den Pokal/dem überglücklichen Sieger/des Clubs/
/Nachdem/seine Teamkollegen/den Spieler/die rote Karte/erhalten hatte,/gab/er/den Ball/an/weiter./
/Nachdem/die rote Karte/den Spieler/erhalten hatte,/gab/er/den Ball/an/seine Teamkollegen/weiter./
/Nachdem/die rote Karte/den Spieler/erhalten hatte,/gab/er/den Ball/an/seine Teamkollegen/weiter./
/Nachdem/die rote Karte/den Spieler/erhalten hatte,/gab/er/den Ball/an/seine Teamkollegen/weiter./
/Nachdem/die rote Karte/den Spieler/erhalten hatte,/gab/er/den Ball/an/seine Teamkollegen/weiter./
/Wie/könnte/es/den Gegner/gelingen,/uns/zu/besiegen?/

Because of the fact that within one sentence sometimes more than one error category arose, all error types are always given within each sentence, for the reason of completion, even though sometimes not addressing a particular error type. The error type addressing has always been marked in bold and cursive font.
Für die Kinder geht es gerne an die Klettergeräte, und sie können in den Ferien oft alleine ins Wasser gehen. Die kleine Susi hat bislang nur selten diese Freiheit erlebt, aber diesmal darf sie zum ersten Mal alleine ins Wasser gehen.

Die Spielerin hat die rote Karte erhalten, nachdem sie die roten Karten werden, hielt sie am Ball weiter. Die Spielerin war nach einem längeren Training müde und nicht mehr in der Lage, sich auf die Karten zu konzentrieren.


Controls:

auxiliary:
/Wo/im/nächsten Winter/das Skirennen/wird/stattfinden?/
/Wo/im nächsten Winter/wird/das Skirennen/stattfinden?/

O_{Dat}:
/Der Trainer/legte/auf/die Schulter/dem traurigen Verlierer/die Hand/
/Der Trainer/legte/die Hand/auf/dem traurigen Verlierer/die Schulter./

O_{Akk}:
/Der Trainer/legte/auf/die Schulter/dem traurigen Verlierer/die Hand/

nominal phrase (subject):
/Nachdem/die rote Karte/der Spieler/erhalten hatte,/gab/er/den Ball/an/seine Teamkollegen/weiter./

possessive pronoun:
/Ich/mag/dieses langweilige Spiel/nicht mehr/länger/ansehen./

autocorrection_right:
/Man/kann/die Grundregeln/leicht/des Basketballs/lernen./
XIV. Guided syntactic production\textsuperscript{131}\_errors

Patients:

nominal inflection:
Alle Mütter kochen das Mittagessen. -4
  • noun inflection; Mutter > Mütter
  • verbal inflection; kocht > kochen

verbal inflection:
Gerne studierst du im Ausland. -2
  • verbal inflection; studiert > studierst

Alle Mütter kochen das Mittagessen. -4
  • noun inflection; Mutter > Mütter
  • verbal inflection; kocht > kochen

Morgen schreibt man wichtig. -4
  • verbal inflection; schreibt > schreibt
  • semantic incorrect sentence

Ich würde gerne im Ausland studieren. -2
  • verbal inflection; studiert > studieren

Morgen schreibt er einen wichtigen Test. -4
  • change in word class adverb; wichtig > adjective (wichtigen)
  • verbal inflection; schreibt > schreibt

Du schreibst mir morgen einen wichtigen Brief. -4
  • verbal inflection; schreibt > schreibt
  • change in word class adverb; wichtig > adjective (wichtigen)

Viele Schüler studieren gerne im Ausland. -2
  • verbal inflection; studiert > studieren

indefinite pronoun inflection:
Es gibt kein Wille mehr. -6
  • change in word class verb; will > noun (Wille)
  • inflection indefinite pronoun; keine – zu kein
  • semantic incorrect sentence

Keiner will mehr! -2
  • inflection indefinite pronoun; keine – keiner

\textsuperscript{131}Because of the fact that within one sentence sometimes more than one error category arised, all error types are always given within each sentence, for the reason of completion, even though sometimes not addressing a particular error type (e.g., Alle Mütter kochen das Mittagessen; focusing on error type verbal inflection; kocht > kochen, the error category (noun inflection); Mutter > Mütter is also mentioned.
**composition:**
Er möchte lieber woanders reden. -2
- composition; anders > woanders

Mein Sohn Ewald möchte lieber woanders wohnen. -2
- composition; anders > woanders

**comparison of adverb:**
Das Herrchen kann den Hund erst später füttern.
- comparison of adverbs; spät > später

Der Hund geht mit dem Herrchen später. -2
- comparison of adverbs; spät > später

**phonological error:**
Wenn du alles richtig schreibst, kannst du morgen früher aufhören. -2
- phonological error; wichtig > richtig

**change in word class:**
Ich möchte lieber etwas Anderes machen. -2
- change in word class_adverb; anders > noun (etwas Anderes)

Morgen schreibt er einen wichtigen Test. -4
- change in word class_adverb; wichtig > adjective (wichtigen)
- verbal_inflection; schreibt > schreibst

Es gibt kein Wille mehr. -6
- change in word class_verb; will > noun (Wille)
- inflection_indefinite pronoun; keine > kein
- semantic incorrect sentence

Viele wollen was Andres lieber. -4
- change of modal verb; möchte > wollen
- change in word class_adverb; anders > noun (was Andres = etwas Anderes)

Du schreibst mir morgen einen wichtigen Brief. -4
- change in word class_adverb; wichtig > adjective (wichtigen)
- verbal_inflection; schreibt > schreibst

Du schreibst morgen eine wichtige Nachricht. -2
- change in word class_adverb; wichtig > adjective (wichtige)

Bis morgen schreibst du die wichtige Hausaufgabe. -2
- change in word class_adverb; wichtig > adjektive (wichtige)

Lieber möchte ich etwas Anderes essen. -2
- change in word class_adverb; anders > noun (etwas Anderes).
Die morgige Arbeit, die du schreibst, ist wichtig. -2
● change in word class_adverb; morgen > adjective (morgige)

Ich möchte lieber etwas Anderes! -2
● change in word class_adverb; anders > noun (etwas Anderes)

Ich möchte lieber etwas Anderes essen. -2
● change in word class_adverb; anders > noun (etwas Anderes)

Morgen schreibst du den wichtigen Brief. -2
● change in word class_adverb; wichtig > adjective (wichtigen)

Du schreibst morgen für mich alles Wichtige auf. -2
● change in word class_adverb; wichtig > noun (alles Wichtige)

morphological agreement:
Wir studiert gerne im Ausland -2
● morphological agreement; wir studiert

Der Junge hat das Glas zerbrochen, weil ihnen fad war. -2
● morphological agreement; weil (der Junge) ihnen fad war

Meine Tochter und du schreibst morgen eine wichtige Schularbeit. -2
● morphological agreement; meine Tochter und du schreibst

change of modal verb:
Viele wollen was Andres lieber. -4
● change of modal verb; möchte > wollen
● change in word class_adverb; anders > noun (was Andres = etwas Anderes)

scipping of element(s):
Ich will nicht mehr! -2
● scipping of elements; keine

Lieber macht man’s anders als man denkt. -4
● semantic_error_change in saying; es ist oft anders als man denkt
● scipping of elements; möchte

Mein Nachbar muss noch spät mit dem Herrchen gehen. -4
● semantic_error_change of lexeme; Hund (Nachbar vs. Herrchen)
● scipping of elements; Hund

Der Junge hat das…-8
● incomplete sentence
● scipping of elements; zerbrochen & weil
● semantic incorrect sentence
Auf das Tonband gehen keine mehr...

- scipping of elements; will
- incomplete sentence
- semantic incorrect sentence

**semantic error_change in saying:**
Lieber macht man’s anders als man denkt. -4
- semantic error_change in saying; es ist oft anders als man denkt
- scipping of element; möchte

**semantic error_change of lexeme:**
Mein Nachbar muss noch spät mit dem Herrchen gehen. -4
- semantic error_change of lexeme; Hund (Nachbar vs. Herrchen)
- scipping of element; Hund

**semantic agreement_causal conjunction:**
Der Junge hat den Krug zerbrochen, weil er ihn umgestürzt hat. -2
- semantic agreement_causal conjunction

Der Junge hat ein Fenster zerbrochen, weil er schlimm war. -2
- semantic agreement_causal conjunction

**delusion by homophones:**
Das Herrchen späht zum Hund. -2
- delusion by homophones; spät > späht

Herrchen späht nach seinem Hund. -2
- delusion by homophones; spät > späht

**semantic incorrect sentence:**
Weil der Junge zerbrochen ist...-4
- incomplete sentence
- semantic incorrect sentence

Es gibt kein Wille mehr. -6
- change in word class; verb (will) > noun (Wille)
- change in inflection of indefinite pronoun (keine < kein)
- semantic incorrect sentence

Morgen schreibt man wichtig. -4
- verbal inflection (schreibst < schreibt)
- semantic incorrect sentence
Der Junge hat das…-8
- incomplete sentence
- scipping of elements; zerbrochen & weil
- semantic incorrect sentence

Auf das Tonband gehen keine mehr…-6
- scipping of element; will
- incomplete sentence
- semantic incorrect sentence

Weil der Junge Glas zerbrochen hat,…-4
- incomplete sentence
- semantic incorrect sentence

Weil ich keine Suppe mehr will…-4
- incomplete sentence
- semantic incorrect sentence

Weil der Junge zerbrochen…-4
- incomplete sentence
- semantic incorrect

**incomplete sentence:**
Auf das Tonband gehen keine mehr…-6
- scipping of element; will
- incomplete sentence
- semantic incorrect sentence

Der Junge hat das…-8
- incomplete sentence
- scipping of elements; zerbrochen & weil
- semantic incorrect sentence

Weil der Junge zerbrochen ist…-4
- incomplete sentence
- semantic incorrect sentence

Weil der Junge Glas zerbrochen hat,…-4
- incomplete sentence
- semantic incorrect sentence

Weil ich keine Suppe mehr will…-4
- incomplete sentence
- semantic incorrect sentence

Weil der Junge zerbrochen…-4
- incomplete sentence
- semantic incorrect
Ich möchte lieber …aso anders steht do…lange Pause…..Mein Sohn möchte lieber anders sein. -1
- autocorrection_right

- autocorrection_right

Er kann nicht essen gehen, weil er Überstunden heute macht [aso] wegen den Überstunden > Wegen den Überstunden kann er heute nichts essen. -1
- autocorrection_right

Morgen schreibst du eine wichtige Arbeit. Frau Lenz hat sich dann selbst ausgebessert: Es ist wichtig, dass du das morgen schreibst. -1
- change in word class; adverb (wichtig) > adjective (wichtige)
- autocorrection_right

Das Mittagessen wird von der Mutter gekocht. > Die Mutter kocht das Mittagessen. -1
- passive; kocht > gekocht
- autocorrection_right

Ich bin zu arm…um, weil…falsch…Weil ich arm bin, kann ich wenig einkaufen. -1
- semantic agreement_causal conjunction
- autocorrection_right

Cue: „ich will“ > „ich will keine mehr“-1
- help_right

Der Junge hat eine Fensterscheibe zerbrochen. (help: „und was ist mit weil?“) Aso weil… weil weil warum…weil er mit dem Fußball gegen eine Wand…fenster geworfen hat. -1
- semantic agreement_causal conjunction
- help_right

Cue: „Ich“ > Ich will keine mehr. -1
- help_right

Cue: möchte lieber > Ich möchte lieber anders heißen. -1
- help_right

Viele Kinder studieren…..ich…Wir…studiert Ausland…. (help: wir studiert, des passt aber nicht)…Sie…studiert gerne im Ausland. -1
- morphological agreement; sie studiert
- help_right
Das Einkaufen...(help: „Fangens mit weil an“..)Weil das Einkaufen schwer ist, tut mir der Arm weh. -1
  • semantic agreement_causal conjunction
  • help_right

Wegen Überstunden....(help: „und was ist mit Essen?“)...Wegen Überstunden....kann er sich das Essen leisten. -1
  • help_right

non response:
  • /WEGEN/ÜBERSTUNDEN/ESSEN/
  • /WEIL/ARM/EINKAUFEN/
  • /HUND/HERRCHEN/SPÄT/
  • /STUDIERT/AUSLND/GERNE/
  • /SCHREIBST/MORGEN/WICHTIG/
  • /MÖCHTE/LIEBER/ANDERS/2x
  • /WILL/KEINE/MEHR/

Controls:

verbal inflection:
  Studierst du gerne im Ausland? -2
  • verbal_inflection; studiert wird zu studiert

collection:
  Ich möchte lieber anderswo schlafen. -2
  • composition; anders > anderswo

phonological error:
  Die Arbeit, die du morgen schreibst ist richtig. -2
  • phonological error; wichtig > richtig

Andreas möchte lieber Torte essen. -2
  • phonological error; anders > Andreas

change in word class:
  Morgen schreibst du ein wichtiges Diktat. -2
  • change in word class_adverb; wichtig > adjective (wichtiges)

Du schreibst morgen eine wichtige Arbeit. -2
  • change in word class_adverb; wichtig > adjective (wichtige)
XV. Providing antonyms_errors

Patients:

not-negation:
kaufen – nicht kaufen 4x
zugeben – nicht zugeben 4x
gut – nicht gut

lexical intrusion:
zugeben – abspielen
gut – sauer

synonym:
aufmachen – öffnen

help_right:
kaufen 2x
einziehen 2x

non response:
kaufen 2x
zunehmen
Anfang

Controls:

not-negation:
kaufen – nicht kaufen

help_right:
kaufen – verkaufen
XVI. Providing two semantic concepts of polysemes

Patients:

naming of only one item:
Stimme: zum Reden 5x
Blatt: Papier 2x
Ball: zum Spielen 3x
Schloss: Zylinderschloss
Birne: im Sinne von Obst
Note: in der Schule
Leiter: zum Raufsteigen

2nd naming referring to the same lexeme as 1st:
Bank: Parkbank, Terrassenbank
Ball: Tennisball, Golfball
Blatt: Ahornblatt, Lindenblatt
Stimme: zum Sprechen; die Stimme, die in einem drinnen ist (innere Stimme)
Blatt: Papier, Zeitungsblatt
Stimme: Stimme von einem Vogel (Vogelstimme), Klavierstimme
Bank: Hausbank (in die man immer geht, um sein Geld zu deponieren, Volksbank
Leiter: Stehleiter, Holzleiter
Stimme: dumpfe Stimme, Tenorstimme
Stimme: meine Stimme, die Stimme von einem Glockenspiel
Leiter: die aus Holz zum Raufsteigen, die Himmelsleiter
Stimme: die die man hört beim Sprechen, die innere Stimme
Stimme: zum Sprechen, die Stimme beim Singen
Ball: Ball zum spielen, (Fußball, Federball)
Bank: Fußball, Federball

compounding:
Bank: Banküberfall
Note: Notenschlüssel
Blatt: Blattläuse

lexicalicalised item:
Stimme: Stimmung 2x

help_right:
Blatt:
Stimme
Flügel

non response:
Bank 3x
Blatt 2x
Note 3x

132 Within „Providing two semantic concepts of polysemes“ per given item, two semantic concepts had to be provided. Thus within the error subcategory „non response“, each non response for each items has to be multiplied by two (two semantic concepts of polysemes; in this example 19 x 2 = 38).
Obst 2x
Ball 1x
Schloss 2x
Flügel 2x
Leiter 2x
Stimme 2x

Controls:

naming of only one item:
Leiter: klettern
Stimme: sagen 2x
Bank: sitzen
Stimme: laut/leise
Ball: spielen
Leiter: Auf die Leiter raufsteigen

2nd naming referring to the same lexeme as 1st:
Stimme: eines Menschen, innere Stimme
XVII. Providing two meanings of homophonic words

Patients:

naming of only one item:
Küste vs. küsste: Felsküste, Küste im Meer (3x), Meeresküste, Küstenabschnitt, Küste (2x), leeren vs. lehren: ausleeren (2x)
Rute vs. Route: Rute mit der man angelt, Angelrute, die Rute zum Schlagen
Lied vs. Lid: das Lied singen (2x), Augenlid
Waise vs. weise: gescheit (2x), sehr gescheit sein, einweisen in die Klinik, irgendjemanden weisen, die weiße Farbe
Sage vs. sage: Sage die man Kindern erzählt, Unwahrheiten im Sinne von Sagen, die Sage, die Sage als Märchen
Arm vs. arm: arm sein (2x), der Arm an der Hand
Saite vs. Seite: die Seite im Buch
Beete vs. bete: die Beete mit Blumen, die Beete
Rat vs. Rad: das Rad, Fahrrad

2nd naming referring to the same lexeme as 1":
leeren vs. lehren: der Lehrer lehrt - die Lehre, die Lehre - jemanden etwas lehren
Küste vs. küsste: Sandküste – Bergküste
Rute vs. Route: Meeresroute – Fahrroute
Rat vs. Rad: Ich gebe einen Rat – ein alter Mann ist ein Ratgeber

2 namings referring to the same lexeme semantic relation:
Küste vs. küsste: Küste am Meer – Klippen

new lexeme:
Saite vs. Seite: abseits
Beete vs. bete: jdn. um etwas bitten, bitten
Waise vs. weise: ein Weißer, einweisen in die Klinik, irgendjemanden weisen, die weiße Farbe
Sage vs. sage: jdm. absagen
Lied vs. Lid: ein Leid das man hat
Rat vs. Rad: das Radar

compounding:
Rat vs. Rad: Kommerzialrat – guter Rat, jemandem einen Rat geben - Rathaus
Saite vs. Seite: auf der Seite – die Seitengasse

compounding_neologism:
Saite vs. Seite: Zeitungsseite – Seidenseite

saying:
Arm vs. arm: arm wie eine Kirchenmaus
Rat vs. Rad: guter Rat ist teuer

help right:
Rat vs. Rad: das Rad
non response\textsuperscript{133}:
Küste vs. küsste (2x)
Arm vs. arm (2x)
leeren vs. lehren (2x)
Saite vs. Seite (4x)
Rute vs. Route (3x)
Waise vs. weise (6x)
Sage vs. sage (4x)
Rat vs. Rad (4x)
Beete vs. bete (5x)
Lied vs. Lied (5x)

Controls:

naming of only one item:
Rat vs. Rad: das Rad, Autoteil
Waise vs. weise: gescheit (2x), ich bin weise
Küste vs. küsste: Ufer, Strand an der Küste

new lexeme:
Waise vs. weise: hinweisen (2x), weiße Farbe

\textsuperscript{133}Within „Providing two semantic concepts of homophonic words“ per given item, two semantic concepts had to
be provided. Thus within the error subcategory „non response“, each non response for each items has to be
multiplied by two (two semantic concepts of homophonic words; in this example 37 x 2 = 74).
XVIII. Decoding of thematic roles_errors

Patients:

a) active sentences with monovalent verbs
   - PD:
     Das Auto fährt. > Das Auto steht.
   - SD:
     Der Bär tanzt. > Der Löwe tanzt. (5x)
     Der Affe sitzt. > Der Löwe sitzt. (2x)
     Das Baby schläft. > Der Mann schläft.

b) active sentences with obligatory bivalent verbs
   - OD:
     Der Lastwagen schiebt den Traktor. > Der Lastwagen schiebt das Auto.
     Der Lastwagen zieht den Traktor. > Der Lastwagen zieht das Auto.
   - S↔O:
     Der Affe stößt den Bären. > Der Bär stößt den Affen. (2x)
     Der Lastwagen zieht den Traktor. > Der Traktor zieht den Lastwagen.
     Der Lastwagen schiebt den Traktor. > Der Traktor schiebt den Lastwagen. (2x)
     Der Hund beißt die Katze. > Die Katze beißt den Hund. (2x)
   - autocorrection_right:
     Der Lastwagen schiebt den Traktor. 1. Der Traktor schiebt den Lastwagen. (S↔O) 2. Der
     Lastwagen schiebt den Traktor.
     Der Lastwagen zieht den Traktor. > 1. Der Lastwagen zieht das Auto. (OD) 2. Der
     Lastwagen zieht den Traktor.
     Der Affe stößt den Bären. > 1. Der Affe stößt den Hasen. (OD) 2. Der Affe stößt den Bären.

  c) passive constructions with obligatory bivalent verbs
     - SD:
       Der Elefant wird vom Krokodil getragen. > Die Katze wird vom Krokodil getragen.
     - S↔O:
       Der Traktor wird vom Lastwagen gezogen. > Der Lastwagen wird vom Traktor gezogen. (3x)
       Der Bär wird vom Affen gestoßen. > Der Affe wird vom Bären gestoßen. (2x)
       Der Elefant wird vom Krokodil getragen. > Das Krokodil wird vom Elefanten getragen. (2x)
     - autocorrection_right:
       Der Traktor wird vom Lastwagen gezogen. > 1. Das Auto wird vom Lastwagen gezogen. (SD)
       2. Der Traktor wird vom Lastwagen gezogen.
       Der Elefant wird vom Krokodil getragen. > 1. Das Krokodil wird vom Elefanten getragen.
       (S↔O) 2. Der Elefant wird vom Krokodil getragen.
d) active sentences with obligatory trivalent verbs

- **dirOD:**
  Der Bub gibt dem Mädchen die Katze. > Der Bub gibt dem Mädchen den Hasen. (2x)
  Der Mann setzt das Baby zum Mädchen. > Der Mann setzt den Teddy zum Mädchen. (4x)

- **S↔indirO:**
  Der Hund bringt der Katze die Maus. > Die Katze bringt dem Hund die Maus. (5x)
  Der Bub gibt dem Mädchen die Katze. > Das Mädchen gibt dem Buben die Katze. (2x)

- **S↔noun of PP:**
  Der Mann setzt das Baby zum Mädchen. > Das Mädchen setzt das Baby zum Mann. (3x)
  Der Großvater stellt die Blumen zur Großmutter. > Die Großmutter stellt die Blumen zum
  Großvater. (2x)

- **autocorrection_right:**
  Der Großvater stellt die Blumen zur Großmutter. > 1. Der Großvater stellt die Gießkanne zur
  Großmutter (dirOD). 2. Der Großvater stellt die Blumen zur Großmutter.


e) passive constructions with obligatory trivalent verbs

- **indirO↔noun of PP:**
  Die Katze wird dem Mädchen vom Buben gegeben. > Die Katze wird dem Buben vom
  Mädchen gegeben. (4x)

- **noun of PP1↔noun of PP2:**
  Das Baby wird vom Mann zum Mädchen gesetzt. > Das Baby wird vom Mädchen zum Mann
  gesetzt. (6x)

- **SD:**
  Das Baby wird vom Mann zum Mädchen gesetzt. > Der Teddy wird vom Mädchen zum Mann
  gesetzt. (2x)
  Die Maus wird der Katze vom Hund gebracht. > Der Vogel wird der Katze vom Hund
  gebracht.

f) coordinated sentences

- **dirO1↔dirO2:**
  Der Traktor schiebt das Auto und zieht den Lastwagen. > Der Traktor schiebt den Lastwagen
  und zieht das Auto. (4x)
  Das Krokodil beißt den Affen und küsst den Fuchs. > Das Krokodil beißt den Fuchs und küsst
  den Affen. (2x)
  Der Lastwagen zieht den Traktor und schiebt das Auto. > Der Lastwagen zieht das Auto und
  schiebt den Traktor.
  Die Katze beißt den Hund und kratzt den Affen. > Die Katze beißt den Affen und kratzt den
  Hund.
• **S→dirO2:**
  Der Traktor schiebt das Auto und zieht den Lastwagen. > Der Lastwagen schiebt das Auto und zieht den Traktor. (2x)
  Der Lastwagen zieht den Traktor und schiebt das Auto. > Das Auto zieht den Traktor und schiebt den Lastwagen.
  Das Krokodil beißt den Affen und küsst den Fuchs. > Der Fuchs beißt den Affen und küsst das Krokodil. (3x)

• **S→dirO1:**
  Das Krokodil beißt den Affen und küsst den Fuchs. > Der Affe beißt das Krokodil und küsst den Fuchs. (4x)
  Der Lastwagen zieht den Traktor und schiebt das Auto. > Der Traktor zieht den Lastwagen und schiebt das Auto. (9x)
  Der Traktor schiebt das Auto und zieht den Lastwagen. > Das Auto schiebt den Traktor und zieht den Lastwagen. (4x)
  Die Katze beißt den Hund und kratzt den Affen. > Der Hund beißt die Katze und kratzt den Affen.

• **autocorrection_right:**
  Der Lastwagen zieht den Traktor und schiebt das Auto. > 1. Der Traktor zieht den Lastwagen und schiebt das Auto. (S↔dirO1) 2. Der Lastwagen zieht den Traktor und schiebt das Auto.

  g) **embedded relative clauses with subject vs. object focus:**
  • **O1↔O2_Sfocus:**
    Das Krokodil, das den Affen beißt, küsst den Fuchs. > Das Krokodil, das den Fuchs beißt, küsst den Affen. (4x)
    Der Lastwagen, der den Traktor zieht, schiebt das Auto. > Der Lastwagen, der das Auto zieht, schiebt den Traktor. (4x)

  • **S1↔O2_Sfocus:**
    Das Krokodil, das den Affen beißt, küsst den Fuchs. > Der Affe, der das Krokodil beißt, küsst den Fuchs. (4x)
    Der Lastwagen, der den Traktor zieht, schiebt das Auto. > Der Traktor, der den Lastwagen zieht, schiebt das Auto. (5x)
    Die Katze, die den Hund beißt, kratzt den Affen. > Der Hund, der die Katze beißt, kratzt den Affen.

  • **S1↔O1_Sfocus:**
    Das Krokodil, das den Affen beißt, küsst den Fuchs. > Der Fuchs, der den Affen beißt, küsst das Krokodil. (2x)
    Die Katze, die den Hund beißt, kratzt den Affen. > Der Affe, der den Hund beißt, kratzt die Katze.
    Der Lastwagen, der den Traktor zieht, schiebt das Auto. > Das Auto, das der Traktor zieht, schiebt den Lastwagen.
 Der Mann, den das Mädchen küsst, stößt den Buben. > Der Mann, den der Bub küsst, stößt das Mädchen. (2x)

**autocorrection_right:**
Das Krokodil, das den Affen beißt, küsst den Fuchs. > 1. Der Fuchs, der den Affen beißt, küsst das Krokodil. (S1↔O1) 2. Das Krokodil, das den Affen beißt, küsst den Fuchs.

h) attached relative clauses with subject vs. object focus

**S1↔O2_Sfocus:**
Das Krokodil beißt den Affen, der den Fuchs küsst. > Der Fuchs beißt den Affen, der das Krokodil küsst. (3x)
Der Lastwagen zieht den Traktor, der das Auto schiebt. > Das Auto zieht den Traktor, der den Lastwagen schiebt.

**S1↔O1_Sfocus:**
Das Krokodil beißt den Affen, der den Fuchs küsst. > Der Affe beißt das Krokodil, das den Fuchs küsst. (3x)
Der Traktor schiebt das Auto, das der Lastwagen zieht. > Das Auto schiebt den Traktor, den der Lastwagen zieht. (2x)
Der Lastwagen zieht den Traktor, der das Auto schiebt. > Der Traktor zieht den Lastwagen, der das Auto schiebt. (2x)

**O1↔O2_Sfocus:**
Das Krokodil beißt den Affen, der den Fuchs küsst. > Das Krokodil beißt den Fuchs, der den Affen küsst. (3x)
Der Lastwagen zieht den Traktor, der das Auto schiebt. > Der Lastwagen zieht das Auto, das den Traktor schiebt. (2x)

**S1↔S2_Ofocus:**
Der Traktor schiebt das Auto, das der Lastwagen zieht. > Der Lastwagen schiebt das Auto, das der Traktor zieht. (3x)

**O1↔S2_Ofocus:**
Der Traktor schiebt das Auto, das der Lastwagen zieht. > Der Traktor schiebt den Lastwagen, den das Auto zieht. (2x)

**autocorrection_right:**
Der Traktor schiebt das Auto, das der Lastwagen zieht. > 1. Der Lastwagen schiebt das Auto, das der Traktor zieht. (S1↔S2) 2. Der Traktor schiebt das Auto, das der Lastwagen zieht.
Controls:

b) active sentences with obligatory bivalent verbs
   ● OD:
   Der Lastwagen schiebt den Traktor. > Der Lastwagen schiebt das Auto.

d) active sentences with obligatory trivalent verbs
   ● autocorrection_right:
   Der Mann setzt das Baby zum Mädchen. > 1. Der Mann setzt den Teddy zum Mädchen. (dirOD) 2. Der Mann setzt das Baby zum Mädchen.

f) coordinated sentences
   ● dirO1↔dirO2:
   Der Traktor schiebt das Auto und zieht den Lastwagen. > Der Traktor schiebt den Lastwagen und zieht das Auto.
   
   ● S↔dirO1:
   
   ● autocorrection_right:
   Das Krokodil beißt den Affen und küsst den Fuchs. > 1. Der Fuchs beißt den Affen und küsst das Krokodil. (S↔dirO2) 2. Das Krokodil beißt den Affen und küsst den Fuchs.
   
   ● help_right:

g) embedded relative clauses with subject vs. object focus
   ● O1↔O2_Sfocus:
   Das Krokodil, das den Affen beißt, küsst den Fuchs. > Das Krokodil, das den Fuchs beißt, küsst den Affen.
   
   ● S1↔O2_Sfocus:
   
   ● S1↔O1_Sfocus:
   Der Mann, den das Mädchen küsst, stößt den Buben. > Der Bub, den das Mädchen küsst, stößt der Mann.
• **autocorrection_right:**
Der Lastwagen, der den Traktor zieht, schiebt das Auto. > 1. Der Lastwagen, der das Auto zieht, schiebt den Traktor. (O1↔O2) 2. Der Lastwagen, der den Traktor zieht, schiebt das Auto.

h) **attached relative clauses with subject vs. object focus**
• **O1↔O2_Sfocus:**
Der Lastwagen zieht den Traktor, der das Auto schiebt. > Der Lastwagen zieht das Auto, das den Traktor schiebt. (2x)

• **autocorrection_right:**
Das Krokodil beißt den Affen, der den Fuchs küsst. > 1. Der Fuchs beißt den Affen, der das Krokodil küsst. (S1↔O2) 2. Das Krokodil beißt den Affen, der den Fuchs küsst.

• **help_right:**
Der Lastwagen zieht den Traktor, der das Auto schiebt. > 1. Der Lastwagen zieht das Auto, das den Traktor schiebt. (O1↔O2) 2. Der Lastwagen zieht den Traktor, der das Auto schiebt.
XIX. **Definition_thematic roles_syntax III**

**a) active sentences with monovalent verbs**

a1) **correct**: Das Auto (S) fährt (P).
PD: Das Auto steht.
SD: Der Traktor fährt.
TA: Der Hubschrauber fliegt.

a2) **correct**: Der Bär (S) tanzt (P).
TA: Der Löwe sitzt.
SD: Der Löwe tanzt.
PD: Der Bär trinkt.

a3) **correct**: Das Baby (S) schläft (P).
PD: Das Baby isst.
TA: Der Bub steht.
SD: Der Mann schläft.

a4) **correct**: Der Affe (S) sitzt (P).
SD: Der Löwe sitzt.
TA: Das Krokodil frisst.
PD: Der Affe schwimmt.

**PD = different predicate**
**SD = different subject**
**TA = total aberration; different subject and different predicate**

**b) active sentences with obligatory bivalent verbs**

b1) **correct**: Der Hund (S) beißt die Katze (O).
OD: Der Hund beißt das Schwein.
TA: Der Elefant trägt das Krokodil.
S↔O: Die Katze beißt den Hund.

b2) **correct**: Der Lastwagen (S) zieht den Traktor (O).
TA: Das Fahrrad fährt gegen den Autobus.
S↔O: Der Traktor zieht den Lastwagen.
OD: Der Lastwagen zieht das Auto.

b3) **correct**: Der Lastwagen (S) schiebt den Traktor (O).
S↔O: Der Traktor schiebt den Lastwagen.
OD: Der Lastwagen schiebt das Auto.
TA: Der Lastwagen zieht das Auto.
b4) **correct: Der Affe (S) stößt den Bären (O).**
OD: Der Affe stößt den Hasen.
TA: Das Krokodil trägt die Katze.
S↔: O Der Bär stößt den Affen.

*S = subject of the sentence = agent
O = object of the sentence = patient
OD= different object (patient)
TA= total aberration; different subject (agent) & different object (patient)*

c) **passive constructions with obligatory bivalent verbs**

c1) **correct: Die Katze (S) wird vom Hund (O) gebissen.**
SD: Das Schwein wird vom Hund gebissen.
TA: Das Krokodil wird vom Elefanten getragen.
S↔O: Der Hund wird von der Katze gebissen.

c2) **correct: Der Traktor (S) wird vom Lastwagen (O) gezogen.**
TA: Der Radfahrer wird vom Bus gerammt.
S↔O: Der Lastwagen wird vom Traktor gezogen.
SD: Das Auto wird vom Lastwagen gezogen.

c3) **correct: Der Elefant (S) wird vom Krokodil (O) getragen.**
S↔O: Das Krokodil wird vom Elefanten getragen.
TA: Die Katze wird vom Hund gebissen.
SD: Die Katze wird vom Krokodil getragen.

c4) **correct: Der Bär (S) wird vom Affen (O) gestoßen.**
SD: Der Hase wird vom Affen gestoßen.
TA: Die Katze wird vom Krokodil getragen.
S↔O: Der Affe wird vom Bären gestoßen.

*S = subject of the sentence = patient
O = object of the sentence = agent
SD= different subject = patient
TA= total aberration; different subject (patient) & different object (agent)*

d) **active sentences with obligatory trivalent verbs**

d1) **correct: Der Bub (S) gibt dem Mädchen (indirO) die Katze (dirO).**
S↔indirO: Das Mädchen gibt dem Buben die Katze.
dirOD: Der Bub gibt dem Mädchen den Hasen.
TA: Der Bub wirft den Ball zur Frau.

d2) **correct: Der Hund (S) bringt der Katze (indirO) die Maus (dirO).**
dirOD: Der Hund bringt der Katze den Vogel.
TA: Der Löwe zeigt dem Bären das Schwein.
S↔indirO: Die Katze bringt dem Hund die Maus.
d3) **correct**: Der Mann (S) setzt das Baby (dirO) zum Mädchen (PP).
S↔noun of PP: Das Mädchen setzt das Baby zum Mann.
dirOD: Der Mann setzt den Teddy zum Mädchen.
TA: Die Frau wirft den Ball zum Buben.

d4) **correct**: Der Großvater (S) stellt die Blumen (dirO) zur Großmutter (PP).
S↔noun of PP: Die Großmutter stellt die Blumen zum Großvater.
dirOD: Der Großvater stellt die Gießkanne zur Großmutter.
TA: Der Bär zeigt dem Löwen das Schwein.

d1 & d2
S = subject of the sentence = agent
indirO = indirect object of the sentence = recipient
dirOD = different direct object = patient
TA = total aberration = different subject, different direct & indirect object

d3 & d4
S = subject of the sentence = agent
noun of PP = noun of the prepositional phrase (=PP) = locativ
dirOD = different direct object = patient
TA = total aberration: different subject, different direct object & different noun of PP

e) **passive constructions with obligatory trivalent verbs**

e1) **correct**: Die Katze (S) wird dem Mädchen (indirO) vom Buben (PP) gegeben.
indirO↔noun of PP: Die Katze wird dem Buben vom Mädchen gegeben.
SD: Der Hase wird dem Mädchen vom Buben gegeben.
TA: Der Ball wird vom Buben zur Frau geworfen.

e2) **correct**: Die Maus (S) wird der Katze (indirO) vom Hund (PP) gebracht.
SD: Der Vogel wird der Katze vom Hund gebracht.
TA: Das Schwein wird dem Bären vom Löwen gezeigt.

e3) **correct**: Das Baby (P) wird vom Mann (PP1) zum Mädchen (PP2) gesetzt.
noun of PP1↔noun of PP2 : Das Baby wird vom Mädchen zum Mann gesetzt.
SD: Der Teddy wird vom Mann zum Mädchen gesetzt.
TA: Der Ball wird von der Frau zum Buben geworfen.

e4) **correct**: Die Blumen (S) werden vom Großvater (PP1) zur Großmutter (PP2) gestellt.
noun of PP1↔noun of PP2: Die Blumen werden von der Großmutter zum Großvater gestellt.
SD: Die Gießkanne wird vom Großvater zur Großmutter gestellt.
TA: Das Schwein wird dem Löwen vom Bären gezeigt.
LVII

e1 & e2
indirO = indirect object = recipient
noun of PP = noun of the prepositional phrase (= PP) = agent
SD = different subject = patient
TA = total aberration; different subject, different indirect object & different noun of PP

e3 & e4
noun of PP1 = noun of the prepositional phrase 2 (= PP1) = agent
noun of the PP2 = noun of the prepositional phrase 1 (= PP2) = locativ
SD = different subject = patient
TA = different subject and different nouns of both PP’s

f) coordinated sentences

f1) correct: Die Katze (S) beißt den Hund (dirO1) und kratzt den Affen (dirO2).
dirO1↔dirO2: Die Katze beißt den Affen und kratzt den Hund.
S↔dirO1: Der Hund beißt die Katze und kratzt den Affen.
S↔dirO2: Der Affe beißt den Hund und kratzt die Katze.

f2) correct: Das Krokodil (S) beißt den Affen (dirO1) und küsst den Fuchs (dirO2).
S↔dirO1: Der Affe beißt das Krokodil und küsst den Fuchs.
dirO1↔dirO2: Das Krokodil beißt den Fuchs und küsst den Affen.
S↔dirO2: Der Fuchs beißt den Affen und küsst das Krokodil.

f3) correct: Der Lastwagen (S) zieht den Traktor (dirO1) und schiebt das Auto (dirO2).
S↔dirO2: Das Auto zieht den Lastwagen und schiebt das Auto.
dirO1↔dirO2: Der Lastwagen zieht den Traktor und schiebt den Lastwagen.

f4) correct: Der Traktor (S) schiebt das Auto (dirO1) und zieht den Lastwagen (dirO2).
S↔dirO2: Der Traktor schiebt das Auto und zieht den Lastwagen.
dirO1↔dirO2: Der Lastwagen schiebt das Auto und zieht den Traktor.

S = subject of the sentence = agent
dirO1 = direct object of the first clause = patient
dirO2 = direct object of the second clause = patient

g) embedded relative clauses with subject vs. object focus

g1) correct: Die Katze (S1), die (S2; RP) den Hund (O2) beißt, kratzt den Affen (O1).
(S-focus)
(O1↔O2): O1>O2 & O2>O1: Die Katze, die den Affen beißt, kratzt den Hund.
(S1↔O2): O2>S1 & S1>O2: Der Hund, der die Katze beißt, kratzt den Affen.
(S1↔O1): O1>S1 & S1>O1: Der Affe, der den Hund beißt, kratzt die Katze.
g2) **correct:** Das Krokodil (S1), das (S2; RP) den Affen (O2) beißt, küsst den Fuchs (O1).

**S-focus**

(S1↔O2): O2>O1 & O1>O2: Der Affe, der das Krokodil beißt, küsst den Fuchs.

(O1↔O2): O2>O1 & O1>O2: Das Krokodil, das den Fuchs beißt, küsst den Affen.

(S1↔O1): O1>O2 & O2>O1: Der Fuchs, der den Affen beißt, küsst das Krokodil.

---

g3) **correct:** Der Lastwagen (S1), der (S2; RP) den Traktor (O2) zieht, schiebt das Auto (O1).

**S-focus**

(S1↔O2): O2>S1 & S1>O2: Der Traktor, der den Lastwagen zieht, schiebt das Auto.

(S1↔O1): O1>S1 & S1>O1: Das Auto, das den Traktor zieht, schiebt den Lastwagen.

(O1↔O2): O1>O2 & O2>O1: Der Lastwagen, der das Auto zieht, schiebt den Traktor.

---

g4) **correct:** Der Mann (S1), den (O1; RP) das Mädchen (S2) küsst, stößt den Buben (O1).

**O-focus**

(S1↔O1): O1>S1 & S1>O1 Der Bub, den das Mädchen küsst, stößt den Mann.

(S1↔S2): S2>S1 & S1>S2 Das Mädchen, das der Mann küsst, stößt den Buben.

(O1↔S2): O1>S2 & S2>O1 Der Mann, den der Bub küsst, stößt das Mädchen.

---

h) **attached relative clauses with subject vs. object focus**

h1) **correct:** Die Katze (S1) beißt den Hund (O1), der (S2; RP) den Affen (O2) kratzt.

**S-focus**

(S1↔O2): O2>S1 & S1>O2: Der Affe beißt den Hund, der die Katze kratzt.

(S1↔O1): O1>S1 & S1>O1: Der Hund beißt die Katze, die den Affen kratzt.


---

h2) **correct:** Das Krokodil (S1) beißt den Affen (O1), der (S2; RP) den Fuchs (O2) küsst.

**S-focus**

(O1↔O2): O2>O1 & O1>O2: Das Krokodil beißt den Fuchs, der den Affen küsst.

(S1↔O2): O2>S1 & S1>O2: Der Fuchs beißt den Affen, der das Krokodil küsst.

(S1↔O1): O1>S1 & S1>O1: Der Affe beißt das Krokodil, das den Fuchs küsst.
h3) correct: Der Lastwagen (S1) zieht den Traktor (O1), der (S2; RP) das Auto (O2) schiebt.
(S-focus)
(S1↔O2): O2>S1 & S1>O2: Das Auto zieht den Traktor, der den Lastwagen schiebt.
(S1↔O1): O1>S1 & S1>O1: Der Traktor zieht den Lastwagen, der das Auto schiebt.
(O1↔O2): O2>O1 & O1>O2: Der Lastwagen zieht das Auto, das den Traktor schiebt.

h4) correct: Der Traktor (S1) schiebt das Auto (O1), das (O2; RP) der Lastwagen (S2) zieht.
(O-focus)
(S1↔S2): S2>S1 & S1>S2: Der Lastwagen schiebt das Auto, das der Traktor zieht.
(S1↔O1): O1>S1 & S1>O1: Das Auto schiebt den Traktor, den der Lastwagen zieht.
(O1↔S2): S2>O1 & O1>S2: Der Traktor schiebt den Lastwagen, den das Auto zieht.

S-focus:
S1 = subject 1 of the main clause = agent
O1 = object 1 of the main clause = patient
S2 = subject 2 as the relative pronoun (= RP) of the relative clause = agent
O2 = object 2 of the relative clause = patient

O-focus:
S1 = subject of the main clause = agent
O1 = object of the main clause = patient
O2 = object 2 as the relative pronoun (= RP) of the relative clause = patient
S2 = subject 2 of the main clause = agent
XX. Language screening_checklist

Anamnesebogen

Testdatum:

Name:

Geburtsdatum:

Händigkeit:

Ausbildung (Art/Dauer):

Beruf:

Krankheitsverlauf:
Sprachproduktion
A) Semantik
A1) Semantischer Speicher:
1. Tiere
2. Gemüse
3. Obst
4. Bekleidung
5. Vögel

B) Morphologie
B1) Bestimmte Artikel:
Beispiel: Der Pullover liegt im Kasten. (Die, Den, Der)
1. Das Kleid ist rot. (Die, Das, Dem) 012
2. Die blaue Hose wird gebügelt. (Das, Der, Die) 012
3. Die Frau zieht die Jacke an. (die, dem, der) 012
4. Das Mädchen trägt die Hüte auf dem Kopf. (die, den, dem) 012
5. Die Stiefel passen nicht zu den Röckchen. (dem, die, den) 012
6. Er knöpft das schwarze Hemd zu. (der, dem, das) 012
7. Der Ärmel des Sakkos ist schmutzig. (das, den, des) 012
8. Die Löcher in den Socken werden gestopft. (der, die, den) 012
9. Das Mädchen mit dem roten Haarband hat langes Haar. (der, die, dem) 012
10. Der Mann stopft das Unterhemd in die Waschmaschine. (das, der, den) 012
11. Sie zieht ein Taschentuch aus der Hosentasche. (den, der, dem) 012
12. Den Jungen gefallen die neuen Socken gut. (Die, Der, Den) 012
13. Der Mann legt den Handschuh in die Schublade. (der, den, die) 012
14. Die gestreifte Bluse steht der Dame gut. (der, die, den) 012
15. Das Schuhband des linken Schuhs fehlt. (dem, die, des) 012
Gesamt:
### B2) Präpositionen:

#### B2.1) Lokale Präpositionen

Bei dieser Aufgabe geht es darum die richtigen Lokalen Präpositionen zu finden. Eine lokale Präposition gibt uns an, wo sich etwas befindet (also unter, auf, etc.) Es werden Ihnen, so wie auch schon bei der letzten Aufgabe, pro Satz drei Kärtchen mit je drei unterschiedlichen lokalen Präpositionen vorgelegt. Dazu lege ich Ihnen ein Bild vor, der zum entsprechenden Satz passt. Bitte versuchen Sie aus den drei Kärtchen die Präposition auszuwählen, die zum Bild passt.

*Beispiel: Das Geschirrtuch liegt im Küchenschrank. (auf, im, unter)*

| 1. Der Topf steht auf dem Herd. (hinter, **auf**, inmitten) | 012 |
| 2. Die Tasse steht bei der Kaffeemaschine. (oberhalb, **bei**, auf) | 012 |
| 3. Inmitten der ganzen Gabeln liegt ein Kochlöffel. | 012 |
| (Außerhalb, **Inmitten**, Um) | |
| 4. Die Frau stellt das Häferl in das Regal über dem Herd. (auf, **über**, entlang) | 012 |
| 5. Vier Tassen stehen rund um die Teekanne. (um, außerhalb, entgegen) | 012 |
| 6. Die Zwiebeln liegen neben dem Knoblauch. (auf, **neben**, trotz) | 012 |
| 7. Die Milch steht im Kühl schrank. (in, längs, **im**) | 012 |
| 8. Oberhalb des Herds ist ein Dunstabzug angebracht. | 012 |
| (Unterhalb, Bei, Oberhalb) | |
| 9. Das Gewürzregal hängt an der Wand. (jenseits, unter, **an**) | 012 |
| 10. Vor dem Kühl schrank steht ein Müll eimer. (Neben, **Vor**, Zwischen) | 012 |

**Gesamt:**
B2.2) Temporale Präpositionen
Beispiel: *Nach* dem Mittagessen gehe ich spazieren. (Inmitten, Nach, Ab)
1. Ab zwölf Uhr wird in der Küche gekocht. (Aus, Außerhalb, Ab) 012
2. Der Kuchen muss noch bis dreizehn Uhr im Rohr bleiben. (um, bis, gegen) 012
4. An Freitagen gibt es zu Mittag immer Fisch. (An, Längs, Zwischen) 012
5. In den kalten Monaten wird es wieder öfter Suppe geben. (In, Bis, Um) 012
6. Um halb zwei muss ich wieder nach dem Braten sehen. (Hinter, Um, In) 012
7. Er hat in den letzten Wochen viel zu viel gegessen. (in, unweit, durch) 012
8. Seit Jahren hat Christina schon keine Bohnen mehr gegessen. (Seit, Hinter, Entlang) 012
9. Benjamin arbeitete *für* drei Jahre in einem Restaurant in Mexiko. (bis, nach, für) 012
10. Während der Kuchen im Rohr ist, wasche ich das Geschirr ab. (Während, Um, Mit) 012

Gesamt:

B2.3) Begründende Präpositionen
Bei dieser Aufgabe geht es darum die richtige Begründende Präposition zu finden. Es werden Ihnen, wie gerade eben pro Satz drei Kärtchen mit je drei unterschiedlichen Begründenden Präpositionen vorgelegt. Bitte versuchen Sie aus den drei Möglichkeiten die Präposition zu finden, von der Sie glauben, dass sie am besten zum vorgegebenen Satz passt.
Beispiel: *Durch* viel Zucker bekommt man schlechte Zähne. (Mittels, Vor, Durch)
1. Dank ihrer gesunden Ernährung hat sie keinerlei Beschwerden. (Dank, Durch, Aus) 012
2. Trotz ihrer Diät hat sie fünf Kilo zugenommen. (Dank, Trotz, Außer) 012
3. Anlässlich der bevorstehenden Geburtstagsfeier wird schon fleißig gebacken. (Diesseits, Anlässlich, Um) 012
4. Unter diesen Umständen kann ich nicht kochen! (Um, Unter, Durch) 012
5. Wegen des Stromausfalls funktioniert der Herd nicht. (Wegen, Durch, Trotz) 012
6. Zur Vollendung des Menüs benötigt man noch weitere Zutaten. (Hinter, Mit, Zur) 012
7. Durch den Brand kann die Küche bis auf weiteres nicht mehr betreten werden. (Angesichts, Außer, Durch) 012
8. Laut Angaben des Kochbüchls soll man die Spaghetti al dente kochen. (Laut, Aus, Durch) 012
9. Aufgrund des kalten Wetters trinkt Claudia jeden Abend Tee. (Aufgrund, In, Inmitten) 012
10. Gemäß der Angaben meiner Mutter nimmt man für die Zubereitung von Sachertorte fünf Eier. (Gemäß, Unter, Durch) 012

Gesamt:
B2.4) Modale Präpositionen
Bei dieser Aufgabe geht es darum die richtige Modale Präposition zu finden. Es werden
Ihnen, wie gerade eben pro Satz drei Kärtchen mit je drei unterschiedlichen Modalen
Präpositionen vorgelegt. Bitte versuchen Sie aus den drei Möglichkeiten die Präposition
to finden, von der Sie glauben, dass sie am besten zum vorgegebenen Satz passt.
Beispiel: **Ohne** Zucker schmeckt der Kaffee einfach nicht. (Ohne, Für, Aus)

1. Die Pfanne ist aus Gusseisen. *(aus, auf, bei)*
2. Das Kochbuch ist auf Deutsch geschrieben. *(über, aus, auf)*
3. Der Küchengehilfe ist in hohem Maße unordentlich. *(auf, in, seitens)*
   *(Mitsamt, Außerhalb, Hinter)*
5. Gegenüber dem letzten Mal schmeckt dein Essen heute grauenvoll.
   *(Aus, Ohne, Gegenüber)*
   *(Statt, Wegen, Gegenüber)*
   *(für, ohne, wegen)*
   *(außer, mit, neben)*
9. **Wider** Erwarten hat der Kleine alles aufgegessen.
   *(Wider, In, Längs)*
10. **Unter** Umständen musst du heute noch einmal einkaufen gehen.
   *(Zwischen, Gegenüber, Unter)*

Gesamt:

**B3) Flexion:**

B3.1) Nominalflexion – Numerus:
Die nächste Aufgabe ist etwas anders, als die vorherigen. Es werden Ihnen insgesamt 10
Hauptwörter vorgegeben. Bitte versuchen Sie diese in die Mehrzahl zu setzen.
Beispiel: ein Pferd – viele Pferde

<table>
<thead>
<tr>
<th>Artikel</th>
<th>Wortform</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>ein Kaninchen –</td>
</tr>
<tr>
<td>2.</td>
<td>ein Rind –</td>
</tr>
<tr>
<td>3.</td>
<td>eine Maus –</td>
</tr>
<tr>
<td>4.</td>
<td>ein Hund –</td>
</tr>
<tr>
<td>5.</td>
<td>eine Katze –</td>
</tr>
<tr>
<td>6.</td>
<td>ein Bär –</td>
</tr>
<tr>
<td>7.</td>
<td>ein Zebra –</td>
</tr>
<tr>
<td>8.</td>
<td>ein Huhn –</td>
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<tr>
<td>9.</td>
<td>ein Känguru –</td>
</tr>
<tr>
<td>10.</td>
<td>ein Vogel –</td>
</tr>
</tbody>
</table>
B3.2) Verbalflexion – Numerus, Tempus

Bei dieser Aufgabe geht es um das richtige Einsetzen von Zeit-also Tunwörtern. Sie bekommen pro Satz wieder 3 Kärtchen vorgelegt, auf denen sich drei mögliche Tunwörter befinden. Bitte versuchen Sie das Tunwort auszuwählen, von dem Sie glauben, dass es am besten zum entsprechenden Satz passt.

Beispiel: Ich **erledige** die Hausarbeit immer gleich am Vormittag. (**erledigst, erledigen, erledige**)  

1. Du…………den Marathon in Bestzeit.  
   läufst  
   lauft  
   laufen  

   springte  
   sprang  
   springe  

3. Die Aufsätze………..gestern von euch......  
   ist…….geschrieben worden.  
   wird…..geschrieben werden.  
   sind…..geschrieben worden.  

4. Als Sekretärin muss man stundenlang vor dem Computer......  
   saß.  
   sitzt.  
   sitzen.  

5. Die Maurer………..zufrieden vor der neuen Fassade des Hauses.  
   stehen  
   steht  

6. Die Salatbeete………..von der Bäuerin täglich......  
   wird….gegossen.  
   werden…..gegossen.  
   werden…..gegossen.  

7. Die neue Kellnerin………, als sie von ihren Kolleginnen beschimpft wurde.  
   weint  
   weintet  
   weinte  

8. In Zukunft………..die Teppiche nicht mehr händisch.........  
   wurden….gewebt werden.  
   wurden….gewoben werden.  
   werden…….gewoben werden.
9. Die Wahrsagerin……... dem Mädchen ein langes Leben......
    hat…….verheißt.
    hat…….verheißt.
    hat…….verheißt.

10. Der Verbrecher fängt zu………….an, als ihn die Polizei verhört.
    schwitzen
    schweißen
    schwitzten

11. Der Gast………., als er ein Haar in seiner Suppe fand.
    tobt
    tobet
    tobt

12. Der Bildhauer………ein Monument aus Marmor
    schaffte
    schuf
    schweif

    liegen
    lugen
    liegen

14. Der Angestellte………seinen Chef um eine Gehaltserhöhung.
    bat
    bittete
    gebeten

15. Ich………sofort mit dem Abwaschen....... 
    werde.......beginnen.
    werde.....begonnen.
    werde.....beginne.

    schläfte
    schlaffe
    schläft

17. Er………..gestern beim Fensterputzen von der Leiter....... 
    ist.....gefallt.
    wird......gefallen.
    ist......gefallen.

18. Drei Frisöre………gestern einer Fussballmannschaft die Haare.
    schniedeten
    schnitt
    schnitten
19. Der Hund…….., als er den Briefträger erblickt.
bellte
bellt
bellen

20. Als die Köchin den Truthahn aus dem Ofen nahm,……..ihr das heiße Fett auf die Finger.
tropfte
tropftet
tropft

Gesamt:
B4) Wortbildung
B4.1) Adjektivierung:

Beispiel: tasten – tastbar; Macht – mächtig
1. Holz –
2. Buckel –
3. Mutter –
4. lesen –
5. Furcht –
6. Glück –
7. achten –
8. lachen –
9. Wind

B4.2) Nominalisierung
Die nächste Aufgabe ist ganz gleich, nur dass jetzt Eigenschaftswörter (= Wiewörter) und Zeitwörter (= Tunwörter) vorgegeben sind. Bitte versuchen Sie nun aus diesen jeweils ein in der Deutschen Sprache existierendes Hauptwort (= Namenswort) zu bilden.

Beispiel: träge – die Trägheit; müde – die Müdigkeit
1. essen –
2. groß –
3. werben –
4. streben –
5. heiter –
6. gut –
7. vergangen –
8. nass –
9. schnell –
10. vergelten –

134Within a probe phase of the screening (including 5 healthy volunteers and 3 HD patients; test results are not included in the data analysis of this study) all healthy volunteers, as well as all HD patients gave non-responses or neologisms on the test item „Last“. Thus this item had to be excluded from the screening, resulting into 9 out of 10 former test items. Because of a lack of HD patients for an additional probe phase, the total number of 9 items for this part of the screening, was maintained and hence resulting into a total score of 18.
C) Syntax
C.1) Satzglieder ordnen
Die nächste Aufgabe ist einmal etwas ganz Anderes. Hier bekommen Sie einen Satz vorgelegt, der in seine einzelne Teile zerlegt wurde. Ich ersuche Sie nun diese Satzteile so zu ordnen, dass dabei ein sinnvoller Satz entsteht.


1. /Die Kinder/ gehen/ in/ den Ferien/ gerne/ klettern./
2. /Der Tennisprofi/ schlug/ den Ball/ ins Netz./
3. /Der neue Tormann/ hat/ den Ball/ nicht/ gehalten./
4. /Die kleine Susi/ darf/ zum/ ersten Mal/ alleine/ ins Wasser/ gehen./
5. /Die Bestzeit/ des Läufers/ soll/ unter/ drei Minuten/ liegen./
6. /Muss/ es/ immer/ einen Gewinner/ geben?/
7. /Die kanadische Abfahrtsläuferin/ will/ olympisches Gold/ gewinnen./
8. /Man/ kann/ die Grundregeln/ des Basketballs/ leicht/ lernen./
9. /Ich/ mag/ mir/ dieses langweilige Spiel/ nicht mehr/ länger/ ansehen./
10. /Gewinnt/ die erste Runde!/
11. /Der engagierte Teamchef/ informiert/ sein Team/ über/ eine neue Strategie.
14. /Nachdem/ der Spieler/ die rote Karte/ erhalten hatte,/ gab/ ihm/ die Hand/auf/ die Schulter./
15. /Was/ bekommt/ der Sieger/ des Rennens/ als Preis?/
16. /Wer/ von den Schülern/ möchte/ die nächste Mannschaft/ auswählen?/
17. /Wo/ wird/ im nächsten Winter/ das Skispringen/ stattfinden?/
18. /Wann/ möchten/ ihr/ wieder/ zum Training/ kommen?/
19. /Wie/ könnte/ es/ uns/ gelingen,/ den Gegner/ zu besiegen?/
20. /Warum/ wurde/ der Abfahrtslauf/ abgesagt?/

110 sentence parts
C.2) Satzproduktion mit vorgegeben Wörtern:
Beispiel: Mädchen läuft Buben > Das Mädchen läuft den Buben nach.
1. KINDER – SCHULE – GEHEN
2. KOCHT – MITTAGESSEN – MUTTER
3. WEIL – ZERBROCHEN – JUNGE
4. ÜBERSTUNDEN – WEGEN – ESSEN
5. ARM – EINKAUFEN – WEIL
6. HUND – SPÄT – HERRCHEN
7. STUDIERT – AUSLAND – GERN
8. SCHREIBST – MORGEN – WICHTIG
9. MÖCHTE – LIEBER – ANDERS
10. KEINE – MEHR – WILL
Sprachverständnis
A) Semantik
A.1) Semantische Kategorien von Bildkarten erkennen
Bei dieser Aufgabe geht es um das Benennen von Bildern. Sie bekommen von mir immer ein Bild vorgelegt, das Sie zuerst einmal benennen sollen. Danach werden Ihnen zu dem jeweiligen Bild Fragen gestellt, die Sie mit Ja oder Nein beantworten sollen. Kann es los gehen?

A1.1) Visuell komplexe Substantive
1) Adler:
   1. Ist es ein Tier, eine Pflanze oder ein Gegenstand? 012
   2. Ist es ein Vierbeiner, ein Insekt oder ein Vogel? 012
   3. Hat es Fell, Federn oder Stacheln? 012
   4. Ist es größer als ein Spatz? Ja 012
   5. Kann es fliegen, sprechen oder fahren? 012
   6. Wohnt es in einer Höhle, in einem Haus oder in einem Nest? 012

2) Ananas:
   1. Ist es ein Obst, ein Gemüse oder ein Tier? 012
   2. Ist es ein Steinobst, eine Südfrucht oder ein Wurzelgemüse? 012
   3. Hat es einen großen Kern, viele kleine Kerne oder eine Schale? 012
   4. Ist es süßer als eine Zitrone? Ja 012
   5. Schneidet man vor dem Verzehr gewöhnlich in Scheiben, paniert man es oder püriert man es? 012
   6. Man findet es gewöhnlich im Stall, auf der Straße oder in einem Obstkorb? 012

3) Herd
   1. Ist es ein Gegenstand, ein Obst oder ein Gemüse? 012
   2. Ist es eine Blume, ein Insekt oder ein Elektrogerät? 012
   3. Hat es Schalter, Beine oder Räder? 012
   4. Ist es schwerer als ein Mixer? Ja 012
   5. Kann man damit kochen, waschen, oder föhnen? 012

4) Heuschrecke
   1. Ist es ein Tier, eine Pflanze oder ein Gegenstand? 012
   2. Ist es ein Säugetier, ein Insekt oder ein Fisch? 012
   3. Hat es einen Rüssel, lange Ohren oder dünne Beine? 012
   4. Ist es größer als eine Mücke? Ja 012
   5. Kann es schwimmen, hüpfen oder kriechen? 012
   6. Findet man es gewöhnlich auf Wiesen, im Wasser oder unter der Erde? 012

Gesamt:
5) Ziehharmonika
1. Ist es eine Pflanze, ein Gegenstand oder ein Tier? 012
2. Ist es ein Musikinstrument, ein Fahrzeug oder Küchenutensil? 012
3. Hat es Tasten, Saiten oder ein Mundstück? 012
4. Ist es schwerer als ein Klavier? Nein 012
5. Kann man damit musizieren, schreiben oder kochen? 012

Gesamt:

A 1.2) Visuell einfache Substantive:
1) Banane 012
1. Ist es ein Gemüse, ein Tier oder ein Obst? 012
2. Ist es eine Schalenfrucht, eine Beere oder ein Steinobst? 012
3. Hat es eine runde, eine gebogene oder eine kegelige Form? 012
4. Schmeckt es bitterer als eine Grapefruit? Nein 012
5. Isst man es ohne Schale, mit Schale oder presst man es aus? 012
6. Wächst es bei kaltem Klima, warmem Klima oder gedeiht es bei beidem? 012

Gesamt:

2) Briefumschlag 012
1. Ist es eine Pflanze, ein Tier oder ein Gegenstand? 012
2. Handelt es sich um einen Büroartikel, ein Küchengerät oder Kleidungsstück? 012
3. Ist es aus Holz, aus Papier oder aus Metall? 012
4. Ist es schwerer als ein Briefkasten? Nein 012
5. Kann man etwas damit versenden, damit telefonieren oder braucht man es im Badezimmer? 012
6. Findet man es im Backrohr, in der Schreibtischlade oder im Bett? 012

Gesamt:

3) Herz 012
1. Ist es eine Form, ein Tier oder eine Pflanze? 012
2. Ist es eine Blume, eine Figur oder ein Baum? 012
3. Ist es eckig, hat es zwei Bögen oder hat es zwei Bögen und eine Ecke? 012
4. Ist es runder als ein Quadrat? Ja 012
5. Kann man es malen, danach singen oder damit putzen? 012
6. Kann man es in einem Liebesbrief, auf einem Notenblatt oder im Garten finden? 012

Gesamt:
4) Mund
1. Ist es ein Körperteil, eine Pflanze oder ein Tier? 012
2. Ist es eine Extremität, ein inneres Organ oder ein äußeres Organ? 012
3. Kann man damit riechen, essen oder hören? 012
4. Ist es größer als ein Bein? Nein 012
5. Dient es der Nahrungsaufnahme, braucht man es zum gehen oder zum hören? 012
6. Befindet es sich im Gesicht, am Oberkörper oder am Unterkörper? 012

Gesamt: 012

5) Nadel
1. Ist es ein Gegenstand, ein Tier oder ein Gebrauchsgegenstand? 012
2. Ist es ein Fortbewegungsmittel, ein Kleidungsstück oder Werkzeug? 012
3. Ist es spitz, gebogen oder rund? 012
4. Ist es härter als ein Strohhalm? Ja 012
5. Kann man damit nähen, schneiden oder zeichnen? 012
6. Findet man es in einem Werkzeugkoffer, einem Nähkästchen oder im Brillenetui? 012

Gesamt: 012
A2) Synonyme:
Bei der nächsten Aufgabe geht es darum, wieder aus drei Kärtchen das Wort auszuwählen, von dem Sie glauben, dass es die gleiche Bedeutung hat, wie das Wort auf Ihrem Zettel.
Beispiel: Karotte Möhre Tomate Schalotte
1. Sessel – (Kessel, Stuhl, Hocker)
2. Erdapfel – (Kartoffel, Adamsapfel, Zwiebel)
3. Sofa – (Bank, Mofa, Couch)
4. Handy – (Mobiltelefon, Fax, Handtasche)
5. sich vergnügen – (sich begnügen, sich amüsieren, sich langweilen)
6. hinab – (abwärts, hinauf, hinter)
7. verreisen – (verweilen, fortfahren, ausreißen)
8. entspannen – (erholen, anspannen, bespannen)
9. schaurig – (schrecklich, gruselig, staubig)
10. nachdenken – (verdenken, lernen, überlegen)
A3) Antonyme:
Bei dieser Aufgabe geht es um das Gegenteil von gerade vorher. Nun sollen Sie mir
Gegenteile, zu 10 vorgegebenen Wörtern nennen.
Beispiel: schwarz – weiß
1.Tag –
2.arm –
3.aufmachen –
4.hell –
5.Mann –
6.gut –
7.kauf en –
8.einziehen –
9.Anfang –
10. zunehmen –

A 4) Polyseme 135
Bei dieser Aufgabe geht es um das Finden unterschiedlicher Begriffe polysem er Wörter.
Polyseme sind Wörter, die für unterschiedliche Begriffe stehen können.
Beispiel: Feder: Feder eines Vogels, Feder aus Draht (zum Beispiel vom Fahrradsitz)
Ich ersuche Sie nun, mir bitte pro Wort mindestens zwei unterschiedliche Begriffe zu nennen,
die Ihnen zu dem entsprechenden Wort einfallen.
1. Birne
2. Bank
3. Ball
4. Blatt
5. Schloss
6. Flügel
7. Note
8. Leiter
9. Stimme

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included in the data analysis of this study) all healthy volunteers, as well as all HD patients gave non-
responses on the test item „Geist“. Thus this item had to be excluded from the screening, resulting into 9 out
of 10 former test items. Because of a lack of HD patients for an additional probe phase, the total number of 9
items for this part of the screening, was maintained and hence resulting into a total score of 18.
A5) Homophone:
Bei dieser Aufgabe geht es um Homophone. Homophone sind Wörter, die gleich klingen, aber unterschiedliche Bedeutungen haben.
Ich werde Ihnen jeweils ein Wort nennen und würde Sie dann bitten mir mindestens zwei Bedeutungen aufzuzählen, die Ihnen zu dem entsprechenden Wort einfallen; also was das Wort alles sein kann.

Beispiel: Waage vs. wage; das Wort kann einmal ein Hauptwort sein; also die Waage, die man zum Abwägen braucht und dann einmal ein Eigenschaftswort, dass einen nicht eindeutigen Zustand bezeichnet, im Sinne von wage sein.
1. Küste vs. küsste
2. Arm vs. arm
3. leeren vs. lehren
4. Saite vs. Seite
5. Rute vs. ruhte
6. Waise vs. weise
7. Sage vs. sage
8. Rat vs. Rad
9. Beete vs. bete
10. Lied vs. Lid

A6) Definitionen:

Beispiel: Man trocknet sich nach dem Waschen mit ihm ab. Handtuch
1. Man verwendet ihn, um Nägel ins Holz zu schlagen. Hammer 012
2. Man schläft in ihm: Bett 012
3. Wenn sie scheint, wird es draußen wärmer. Sonne 012
4. Man braucht ihn, um etwas auf Papier zu bringen. Stift 012
5. Man schaltet es im Zimmer ein, wenn es draußen dunkel wird. Licht 012
6. Wenn er da war, ist der Briefkasten voll. Briefträger 012
8. Ein italienisches Teiggericht, das man mit verschiedenen Zutaten belegen kann und anschließend im Ofen bäckt. Pizza 012
9. Die Zeit im Jahr, auf die man sich während des Arbeitens immer freut und in der man gerne verreist. Urlaub 012
10. Ein pelziger Geselle, der einmal pro Winter mit seiner Rute kommt und vor dem sich die Kinder fürcht. Krampus 012

Gesamt:
A7.) Metaphern:
Bei der nächsten Aufgabe geht es um das Verständnis von Sprichwörtern, oder Redewendungen. Sie sollen wiederum aus drei möglichen Erklärungen versuchen, die richtige zu finden.

Beispiel: Rabeneltern:

a) Vögel, die Brutpflege betreiben
b) Vogelkundige Eltern
c) Eltern, die ihre Kinder vernachlässigen

1. Wie Hund und Katz

a) Zwei Menschen, die sich nicht vertragen.
b) Bestimmtes Verhalten, dass Hunde zeigen, wenn sie auf Katzen treffen.
c) Bezeichnet das Phänomen, dass Hunde- bzw. Katzenbesitzer ihrem Haustier immer ähnlicher werden.

2. Jemandem nicht das Wasser reichen können.

a) Das Gegenüber ist so weit entfernt, dass man ihm den Wasserkrug zum Trinken nicht reichen kann.
b) An die Leistungen eines anderen Menschen nicht herankommen können.
c) Man ist zu geizig, um einem anderen Menschen ein Geschenk zu überreichen.

3. Jemandem das Herz brechen.

a) Jemandem bei einem Faustkampf schwere Verletzungen zufügen
b) Einer Person großen Liebeskummer bereiten
c) Eine Person so sehr erschrecken, dass sie dabei einen Herzenfarkt erleidet.

4. Warteschlange

a) Ein Reptil, das im Hinterhalt seiner Beute auflauert.
b) Volkstümliche Bezeichnung für eine Frau mit Brille, die sehr lange auf jemanden wartet.
c) Personen oder Fahrzeuge, die in einer Reihe stehen und auf etwas warten.

5. Die Nadel im Heuhaufen suchen

a) Nach einer schwer auffindbaren Sache suchen.
b) Altes bäuerliches Brauchtum, wo die Mägde nach erbrachter Heuernte im Heustadel Tischtücher besticken.
c) Wichtige landwirtschaftliche Pflicht, bei der der Bauer im getrockneten Gras nach spitzen Gegenständen sucht, die die Tiere beim Verzehr sonst verletzen würden.

Gesamt:
B1) Fragen:
Auch die nächste Aufgabe geht ganz schnell und ist ganz lustig. Ich stelle Ihnen ganz einfache Verständnisfragen und Sie antworten bitte mit ja/nein bzw. mit einer präzisen Antwort.

1. Kommt Dienstag vor Mittwoch? **Ja**
2. Hat das Jahr acht Monate? **Nein**
3. Wachsen Äpfel auf Bäumen? **Ja**
4. Ist es im Sommer kalt? **Nein**
5. Gibt eine Ziege Milch? **Ja**
6. Ist Kärnten in Österreich? **Ja**
7. Kann eine Katze bellen? **Nein**
8. Gibt es fünf Jahreszeiten? **Nein**
9. Gibt es roten Paprika? **Ja**
10. Wie heißt unser Bundespräsident? **Fischer**
11. Wo steht der Stephansdom? **In Wien**
13. Was ist ein PKW? **Ein Auto**
Warum wird das Ozonloch immer größer? **Wegen der Umweltverschmutzung**
15. Wer ist das Oberhaupt der katholischen Kirche? **Der Papst**

Gesamt:
B2) Reversible Satzgefüge:
Für diese Aufgabe wurden ganz lustige Zeichnungen angefertigt. Und zwar sind pro Blatt vier Zeichnungen oben. Sie bekommen immer einen Satz vorgelegt und lesen sich diesen in Ruhe durch. Dann schauen Sie auf das Blatt mit den Zeichnungen und versuchen jene Zeichnung auszusuchen, von der sie glauben, dass sie das darstellt, was der Satz aussagt.

a)
1) 1: Das Auto fährt. 012
2: Das Auto steht.
3: Der Traktor fährt.
4: Der Hubschrauber fliegt.

2) 4: Der Bär tanzt. 012
1: Der Löwe sitzt.
2: Der Löwe tanzt.
3: Der Bär trinkt.

3) 2: Das Baby schläft. 012
1: Das Baby isst.
3: Der Bub steht.
4: Der Mann schläft.

4) 4: Der Affe sitzt. 012
1: Der Löwe sitzt.
2: Das Krokodil frisst.
3: Der Affe schwimmt.

Gesamt:

b)
1) 3: Der Hund beißt die Katze. 012
1: Der Hund beißt das Schwein.
2: Der Elefant trägt das Krokodil.
4: Die Katze beißt den Hund.

2) 3: Der Lastwagen zieht den Traktor. 012
1: Das Fahrrad fährt gegen den Autobus.
2: Der Traktor zieht den Lastwagen.
4: Der Lastwagen zieht das Auto.

3) 1: Der Lastwagen schiebt den Traktor. 012
2: Der Traktor schiebt den Lastwagen.
3: Der Lastwagen schiebt das Auto.
4: Der Lastwagen zieht das Auto.

4) 2: Der Affe stößt den Bären. 012
1: Der Affe stößt den Hasen.
3: Das Krokodil trägt die Katze.
4: Der Bär stößt den Affen.

Gesamt:
c) 1) 3: Die Katze wird vom Hund gebissen.
   1: Das Schwein wird vom Hund gebissen.
   2: Das Krokodil wird vom Elefanten getragen.
   4: Der Hund wird von der Katze gebissen.

   2) 3: Der Traktor wird vom Lastwagen gezogen.
   1: Der Autobus wird vom Lastwagen gezogen.
   2: Der Lastwagen wird vom Traktor gezogen.
   4: Das Auto wird vom Lastwagen gezogen.

   3) 1: Der Elefant wird vom Krokodil getragen.
   2: Das Krokodil wird vom Elefanten getragen.
   3: Die Katze wird vom Hund gebissen.
   4: Die Katze wird vom Krokodil getragen.

   4) 2: Der Bär wird vom Affen gestoßen.
   1: Der Hase wird vom Affen gestoßen.
   3: Die Katze wird vom Krokodil getragen.
   4: Der Affe wird vom Bären gestoßen.

   Gesamt:
   d) 1) 4: Der Bub gibt dem Mädchen die Katze.
   1: Das Mädchen gibt dem Buben die Katze.
   2: Der Bub gibt dem Mädchen den Hasen.
   3: Der Bub wirft den Ball zur Frau.

   2) 1: Der Hund bringt der Katze die Maus.
   2: Der Hund bringt der Katze den Vogel.
   3: Der Löwe zeigt dem Bären das Schwein.
   4: Die Katze bringt dem Hund die Maus.

   3) 3: Der Mann setzt das Baby zum Mädchen.
   1: Das Mädchen setzt das Baby zum Mann.
   2: Der Mann setzt den Teddy zum Mädchen.
   4: Die Frau wirft den Ball zum Buben.

   4) 1: Der Großvater stellt die Blumen zur Großmutter.
   2: Die Großmutter stellt die Blumen zum Großvater
   3: Der Großvater stellt die Gießkanne zur Großmutter.
   4: Der Bär zeigt dem Löwen das Schwein.

   Gesamt:
   e) 1) 4: Die Katze wird dem Mädchen vom Buben gegeben.
   1: Die Katze wird dem Buben vom Mädchen gegeben.
   2: Der Hase wird dem Mädchen vom Buben gegeben.
   3: Der Ball wird vom Buben zur Frau geworfen.
2) 1: Die Maus wird der Katze vom Hund gebracht.  
2: Der Vogel wird der Katze vom Hund gebracht.  
3: Das Schwein wird dem Bären vom Löwen gezeigt.  
4: Die Maus wird dem Hund von der Katze gebracht.  

3) 3: Das Baby wird vom Mann zum Mädchen gesetzt.  
1: Das Baby wird vom Mädchen zum Mann gesetzt.  
2: Der Teddy wird vom Mann zum Mädchen gesetzt.  
4: Der Ball wird von der Frau zum Buben geworfen.  

4) 1: Die Blumen werden vom Großvater zur Großmutter gestellt.  
2: Die Blumen werden von der Großmutter zum Großvater gestellt.  
3: Die Gießkanne wird vom Großvater zur Großmutter gestellt.  
4: Das Schwein wird dem Löwen vom Bären gezeigt.  

Gesamt:

f)  
1) 1: Die Katze beißt den Hund und kratzt den Affen.  
3: Der Hund beißt die Katze und kratzt den Affen.  
4: Der Affe beißt den Hund und kratzt die Katze.  

2) 4: Das Krokodil beißt den Affen und küsst den Fuchs.  
1: Der Affe beißt das Krokodil und küsst den Fuchs.  
2: Das Krokodil beißt den Fuchs und küsst den Affen.  
3: Der Fuchs beißt den Affen und küsst das Krokodil.  

3) 2: Der Lastwagen zieht den Traktor und schiebt das Auto.  
1: Der Traktor zieht den Lastwagen und schiebt das Auto.  
3: Das Auto zieht den Traktor und schiebt den Lastwagen.  
4: Der Lastwagen zieht das Auto und schiebt den Traktor.  

4) 4: Der Traktor schiebt das Auto und zieht den Lastwagen.  
1: Der Lastwagen schiebt das Auto und zieht den Traktor.  
2: Der Traktor schiebt den Lastwagen und zieht das Auto.  
3: Das Auto schiebt den Traktor und zieht den Lastwagen.  

Gesamt:
g) 1) Die Katze, die den Hund beißt, kratzt den Affen.
2: Die Katze, die den Affen beißt, kratzt den Hund.
3: Der Hund, der die Katze beißt, kratzt den Affen.
4: Der Affe, der den Hund beißt, kratzt die Katze.

2) 4: Das Krokodil, das den Affen beißt, küsst den Fuchs.
1: Der Affe, der das Krokodil beißt, küsst den Fuchs.
2: Das Krokodil, das den Fuchs beißt, küsst den Affen.
3: Der Fuchs, der den Affen beißt, küsst das Krokodil.

3) 2: Der Lastwagen, der den Traktor zieht, schiebt das Auto.
1: Der Traktor, der den Lastwagen zieht, schiebt das Auto.
3: Das Auto, der den Traktor zieht, schiebt den Lastwagen.
4: Der Lastwagen, der das Auto zieht, schiebt den Traktor.

4) 1: Der Mann, den das Mädchen küsst, stößt den Buben.
2: Der Bub, den das Mädchen küsst, stößt den Mann.
3: Das Mädchen, das der Mann küsst, stößt den Buben.
4: Der Mann, den der Bub küsst, stößt das Mädchen.

Gesamt:

h) 1) Die Katze beißt den Hund, der den Affen kratzt.
1: Der Affe beißt den Hund, der die Katze kratzt.
3: Der Hund beißt die Katze, die den Affen kratzt.

2) 4: Das Krokodil beißt den Affen, der den Fuchs küsst.
1: Das Krokodil beißt den Fuchs, der den Affen küsst.
2: Der Fuchs beißt den Affen, der das Krokodil küsst.
3: Der Affe beißt das Krokodil, das den Fuchs küsst.

3) 1: Der Lastwagen zieht den Traktor, der das Auto schiebt.
2: Das Auto zieht den Traktor, der den Lastwagen schiebt.
3: Der Traktor zieht den Lastwagen, der das Auto schiebt.
4: Der Lastwagen zieht das Auto, das den Traktor schiebt.

4) 1: Der Traktor schiebt das Auto, das der Lastwagen zieht.
2: Der Lastwagen schiebt das Auto, das der Traktor zieht.
3: Das Auto schiebt den Traktor, der der Lastwagen zieht.
4: Der Traktor schiebt den Lastwagen, den das Auto zieht.

Gesamt:
C: Textverständnis:
Nun sind wir so gut wie am Ende. Nur noch diese Aufgabe....Sie bekommen jetzt zwei Texte vorgelegt, sie haben so lange Zeit, wie Sie benötigen, um den Text zu lesen. Lesen Sie sich alles in Ruhe durch und versuchen Sie sich den Inhalt des Textes zu merken. Anschließend werden Ihnen pro Text 5 Fragen vorgelegt, die Sie dann bitte zu beantworten versuchen. Los geht's!

C1) Gebrauchsanweisung:

*Fragen:*

1. Soll der Temperaturregler vor dem Einfrieren auf Stufe 6 gestellt werden? **Ja** 012
2. Kann man bis zu 2 kg Lebensmittel innerhalb von 24 Stunden einfrieren? **Ja** 012
3. Sind die neu einzufrierenden Lebensmittel nach einer Stunde vollständig durchgefroren? **Nein** 012
4. Bleiben Nährwert, Vitamine und Aussehen am besten erhalten, wenn die frischen Lebensmittel langsam gefroren werden? **Nein** 012
5. Muss der Temperaturregler 24 Stunden nach dem Einfrieren wieder auf die übliche Position zurückgestellt werden? **Ja** 012

**Gesamt:**

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C2.) Zeitungsartikel:

*Fragen:*

1. Ist die letzte Renovierung des Uhrturms zehn Jahre her? **Ja** 012
2. Lässt SP-Stadtrat Riedler Geld in die Renovierung fließen? **Ja** 012
3. Wird die Generalsanierung beginnen, bevor die Feuchtigkeitsschäden behoben wurden? **Nein** 012
4. Werden Experten von der KF-Universität Graz miteinbezogen? **Nein** 012
5. Soll der hölzerne Wehrgang unterm Dach des Uhrturms wieder begehbar werden? **Ja** 012

**Gesamt:**