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ERROR PROCESSING IN NORMAL AGING AND IN BASAL GANGLIA DISORDERS

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Abstract—Recently it has been shown that effects of aging and pathologically induced changes of basal ganglia structures may have quite similar effects on cognitive functions mediated by the medial prefrontal cortex. The question appears, if this pattern may be assignable to other cognitive functions that are mediated via the basal ganglia and medial prefrontal brain areas. Error processing is a component of executive functions that also depends on these areas and especially on the anterior cingulate cortex (ACC). Hence we ask, if error processing functions are differentially modulated by normal aging and basal ganglia diseases. Error processing mechanisms in these groups were investigated using a cognitive event-related potential (ERP), the error negativity. Enrolling an extended sample of young and elderly controls, as well as patients with Parkinson's and Huntington's disease, we show that modulations of error processing differ between aging, different basal ganglia diseases. Despite that the examined basal ganglia disorder groups (Parkinson's and Huntington's disease) differ in their age they show similar modulations in error processing, suggesting that aging effects are overridden by pathogenic effects. The study shows that it may be valuable to compare aging not only to different forms of basal ganglia disorders in order to gain knowledge about age- and disease-related mechanisms and the effects of these on cognitive functions. Diseases of the basal ganglia may impact error processing above and beyond the effects of normal aging. Although many aging, Parkinson's disease and Huntington's disease studies on error processing functions have already been published, this study ties together several related observations across all of these groups in one experiment.

Key words: aging, Parkinson's disease, Huntington's disease, error processing.

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Abbreviations: ACC, anterior cingulate cortex; DA, dopaminergic; ERN (Ne), error related negativity; ERP, event-related potential; HD, Huntington's disease; M, mean; PD, Parkinson's disease; PD-de novo, de novo (drug naive) Parkinson's disease patients; PD-off, Parkinson's disease patients, off-medicated; pHD, pressymptiomatic gene mutations carries Huntington's disease; RT, response time.

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The processing of errors is a basic executive function, which induces corrective and adaptive actions after a (response) error has occurred, e.g. the attempt to inhibit the error on-line, the immediate correction of the error and the slowing in response times (RTs) after an error (e.g. Rabbitt, 1990). Error processing is reflected in event-related potential (ERP) after an incorrect response as a negative component, the error (related) negativity (Ne or ERN) (Falkenstein et al., 1990; Gehring et al., 1993). It is assumed that the mid-brain dopaminergic (DA) system and medial prefrontal areas, like the anterior cingulate cortex (ACC) interact in producing the Ne (e.g. Hester et al., 2004; Rushworth et al., 2004). If an event is worse than expected (i.e. an error), the DA system sends a signal to the ACC, which in turn elicits the Ne (Holroyd and Coles, 2002).

In a recent study (Wild-Wall et al., 2008) it was shown that age and basal ganglia diseases (i.e. Parkinson's disease (PD) and Huntington's disease (HD)) have similar effects on functions depending on medial prefrontal brain areas (i.e. interval timing functions). The study by Wild-Wall et al. (2008) shows that effects of aging and pathologically induced changes of basal ganglia structures may be quite similar. The question appears, if this pattern is specific for this examined function, or may be assignable to other cognitive functions that are mediated via the basal ganglia and medial prefrontal brain areas, i.e. error processing functions. Even though it is known from different studies that PD, symptomatic HD patients, as well as healthy elderly subjects, show a decline in error processing (e.g. Falkenstein et al., 2001; Beste et al., 2006, 2008; Willemssen et al., 2008), a direct comparison allowing an examination of this question is lacking. A lack of difference between the groups may suggest similar effects of basal ganglia function and effects in age and disease on medial prefrontal brain functions. On the contrary, a more graduated pattern in the modulation of error processing would suggest function dependent effects of aging on processes, depending on the basal ganglia and the medial frontal cortex. If HDs and all elderly groups do not differ from each other, this may suggest that even faint changes are sufficient to alter error processing functions (see also: Wild-Wall et al., 2008).

To examine the above questions, we enrolled several groups of healthy young and old people, as well as basal ganglia disease groups into the study.

EXPERIMENTAL PROCEDURES

Participants

Six groups were enrolled into study. A group of 17 medicated patients with PD measured after overnight withdrawal off their

Table 1. Antiparkinsonian medication for medicated PD per day in milligram (mg)

Patient	Medication (dose/day in mg)	Patient	Medication (dose/day in mg)
1	L 125	9	L 50
2	L 250, Rop 2	10	L 600, *Pr* 0.54, E 800, S 5, A 300
3	L 437.5	11	L 375, C 4.5
4	*Pr* 0.804, A 200	12	L 187.5, Rop 6, A 300
5	L 447.5, C 2	13	L 187.5, *Pr* 1.05, E 600
6	L 500, C 6	14	L 125
7	L 600, C 4, E 1000, A 200	15	L 325, C 4, S 10
8	L 700, C 5.5, E 1000, A 400	16	L 500, C 2

Abbreviations: A, amantadine; C, cabergoline; E, entacapone; L, L-DOPA; *Pr*, pramipexol; Rop, ropinirol; Rot, rotigotin; S, selegiline.

medication (PD-off) from 50 to 79 years of age (mean (M)=66.8; SD=8.5) was recruited. The M daily dose of antiparkinsonian medication is displayed in Table 1.

This group was complemented by a de novo (drug-naive) PD-group (PD-de novo) from 41 to 75 years of age (M=59.6; SD=10.4), serving to control for possible long term medication effects. The PD-groups were complemented with two HD groups. Here, a group of 15 right-handed, unmedicated HD-patients defined by a positive gene test and manifest clinical symptoms from 29 to 57 years of age (M=37.1; SD=7.4) was recruited. Additionally 15 right-handed presymptomatic gene mutation carriers from 22 to 50 years of age (M=32.9; SD=7.9) were recruited.

These clinical groups were complemented by two healthy control groups, one young (n=15) group from 27 to 49 years of age (M=34.5; SD=5.5), which is comparable to the HD-groups, and an old group from 52 to 75 years of age (M=65.2; SD=7.2), which also served as control group for the PD patients. More detailed demographical data are given in Table 2. All groups had a similar educational background. All participants gave written informed consent. The study was approved by the ethics committee of the University of Bochum.

Stimuli and procedure

To measure error-processing we used a "Flanker Task" (Kopp et al., 1996). The stimuli consisted of vertical arrays of arrowheads or circles. The central part of the stimulus was defined as target. When the target was an arrowhead the subjects had to press a button on the side the target pointed to; when the target was a circle, no response had to be given (Nogo targets). Nogo targets had a probability of 20%, arrowhead targets of 80%. Above and below each target a flanker was presented pointing either to the same side (congruent trials) or to the opposite side (incongruent trials) of the target. Congruent trials had a (total) probability of 60%, incongruent trials of 20% and 20% Nogo-trials. The Nogo-trials were not analyzed here. Right and left pointing flankers were equiprobable. The flankers preceded the targets by 100 ms (stimulus onset asynchrony, SOA 100) to further strengthen their influence and consequently further increase the error rate in incongruent trials (Willemssen et al., 2004). Flankers and targets were presented for 100 ms. The next flanker was presented 800–1200 ms (interval randomized) after the response of the subjects, or 1900–2300 ms after a Nogo target. Altogether 420 stimuli were presented in four blocks of 105 stimuli each, which were interrupted by short breaks. The subjects were asked to react as fast as possible to the arrowhead targets.

Table 2. Descriptive data for the different groups of ages, sex, general level of intelligence (MWT-B), depression (BDI), mental rotation performance assessed via the LPS (subtest 7) as measured for executive control function, unified Parkinson's Disease Rating Scale (UPDRS)/Unified Huntington's Disease Rating Scale (UHDRS motor scores), CAG-repeat size (CAG), estimated age of onset (eAO) and the age of onset[a]

	Young		Old		PD-off medication		PD-de novo		pHD		HD	
	M (SD)	Range	M (SD)	Range	M (SD)	Range	M (SD)	Range	M (SD)	Range	M (SD)	Range
Age	34.5 (5.5)	27–49	65.2 (7.2)	52–75	66.8 (8.5)	50–79	59.6 (10.4)	41–75	32.9 (7.9)	22–50	37.1 (7.4)	29–57
Sex	8 Females/7 males		8 Females/9 males		8 Females/9 males		8 Females/7 males		7 Females/8 males		7 Females/8 males	
MWT-B	119 (10.1)	101–130	126 (14.5)	104–143	129 (8.3)	118–136	113 (11.3)	92–124	109 (10.8)	90–130	109 (10.6)	95–130
Mental rotation	14 (3.6)	11–20	15 (5.8)	8–26	13.4 (4.7)	8–25	14.8 (7.5)	5–34	13.1 (4.5)	9–24	13.9 (5.7)	8–30
BDI	2.2 (3.1)	0–11	6.3 (5.4)	1–18	6.7 (4.1)	0–14	8.2 (4.5)	1–17	3.8 (3.6)	0–14	4.1 (3.5)	0–14
UPDRS/UHDRS	NA	NA	NA	NA	15.9 (5.3)	6–26	12.6 (5.4)	3–21	0	0	17.1 (7.9)	9–32
CAG	NA	NA	NA	NA	NA	NA	NA	NA	42.8 (1.7)	39–46	46.1 (5.5)	40–55
eAO	NA	NA	NA	NA	NA	NA	NA	NA	44.5 (4.1)	36.5–52.5	NA	NA
Ao	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	35.1 (10.9)	20–43

[a] The latter three are only given for the appropriate HD-group.

Data processing and analysis

During the task the EEG was recorded from 32 electrodes (Ag/AgCl) (Fpz, Fp1, Fp2, Fz, F3, F4, F7, F8, FCz, FC3, FC4, FC5, FC6, Cz, C3, C4, C7, C8, Pz, P3, P4, P7, P8, Oz, O1, O2, M1, M2), two lateral and four vertical EOG electrodes (sampling rate: 500 Hz). Cz was used as primary reference. The filter bandwidth was from DC to 80 Hz. Impedances were kept below 5 kΩ. The EEG was digitally filtered using a 0.10 Hz high-pass and 20 Hz low-pass filter. From the EEG response-locked ERPs were computed, beginning 400 ms before and ending 700 ms after the correct or incorrect response. After this, eye movement artifacts were corrected with the Gratton-Coles-algorithm using the EOG data (Gratton et al., 1983), followed by a baseline correction (−200–0 ms [i.e. response]). Remaining artifacts were rejected using an amplitude criterion of ±80 μV followed by re-referencing all data to linked mastoids. The Nogo trial data were not further evaluated within the present study, which focused on error processing and not on inhibition. The amplitude of the Ne in error trials and of the correct-related negativity (Nc/CRN) in the correct trials (Ford, 1999; Yordanova et al., 2004) was measured relative to the peak of the positivity, which precedes both components (Gehring and Knight, 2000) at the electrodes Fz, FCz and Cz. For the electrophysiological data the M and standard error of the M (±SEM) are given. For further statistical analyses a repeated measures ANOVA with the factor "electrode" (Fz, FCZ, Cz) and "correctness" (correct vs. false responses) as within-subject factor and "group" (young control, old control, PD off medication, PD-de novo, pressymptiomatic gene mutations carries Huntington's disease [pHD], HD) as between-subject factor was calculated. This design is comparable to one used by Wild-Wall et al. (2008).

RESULTS

Behavioral data

RTs on correct and false responses were analyzed using a repeated measures ANOVA with the within-subject factor "correctness" and the between subject factor "group." There was a main effect "correctness" ($F_{(1,87)}$=149.96; P<0.001), with RTs being generally shorter on false (338.8±7.1), than on correct responses (410.3±5.1). There was no interaction "correctness×group" ($F_{(5,87)}$=0.34; P>0.8), but a main effect group ($F_{(5,87)}$=21.62; P<0.001). The PD-off (433.8±13.3) and PD-de novo (441.7±13.7) responded much slower than the pHD (308.4±13.7), HD (375.3±13.7) and young control group (289.7±13.7) (P<0.015), but not than the old controls (398.5±12.9) (P>0.3). Old controls differed from young controls and pHDs (P<0.001). The young controls differed from all other groups (P<0.001), except the pHD-group (P>0.9). RTs of correct responses committed after an error (posterror-RTs) were generally prolonged, which reflects the behavioral adaptation after an error (Rabbitt, 1966). Therefore we subjected the M reaction time of correct responses in succession and those after an error as within-subject factor to a repeated measure ANOVA with "group" as between-subject factor. RTs on correct response after an error were significantly longer (419.09±6.9) ($F_{(1,87)}$=20.79; P<.001), than RTs on correct responses in succession (396.1±5.0), indicating a slowing effect. No interaction with the factor group was obtained ($F_{(5,87)}$=0.46; P>0.8), i.e. posterror slowing did not differ between groups.

Error rates were analyzed in a repeated measures ANOVA using "congruency" as within-subject factor and "group" as between-subject factor. There was a significant main effect congruency ($F_{(1,87)}$=190.74; P<0.001) showing that errors were more frequent in the incongruent (14.1±0.9) than in the congruent condition (3.6±0.4). This effect was not different for the groups (interaction "group×congruency" $F_{(5,87)}$=0.48; P>0.7). Also the main effect group was not significant ($F_{(5,87)}$=1.60; P>0.16). The error rates for the different conditions and groups are given in Table 3.

Neurophysiological data

The Ne is shown for all groups in Fig. 1. The repeated measures ANOVA revealed a significant main effect "electrode" ($F_{(2,174)}$=87.22; P<0.001). It is shown that potentials were maximal at FCz (−6.19±0.20), followed by Fz (−5.50±0.16) and Cz (−4.15±0.13). All electrodes differed from each other (P<0.001). Additionally, there was an interaction "electrode×group" ($F_{(10,174)}$=14.48; P<0.001).

More important, there was a main effect "correctness" ($F_{(1,87)}$=379.78; P<0.001), showing that potentials were stronger for incorrect (−7.81±0.23) (i.e. Ne), compared to correct trials (−2.75±0.12) (i.e. Nc). The factor "correctness" interacted with "electrode" ($F_{(2,174)}$=61.12; P<0.001). It is shown that the Ne amplitudes differed between electrodes ($F_{(2,28)}$=37.63; P<0.001) (Fz: −13.49±1.35; FCz: −15.21±1.47; Cz: −6.16±0.56). Fz and FCz did not differ from each other. For the Nc (correct trials) no difference between electrodes was obtained ($F_{(2,28)}$=0.04; P>0.9).

The main effect "correctness" was further modulated by the factor group, as the interaction "correctness×group" reveals ($F_{(5,87)}$=15.23; P<0.001). Subsequent Bonferroni-corrected univariate ANOVAs showed that the groups strongly differed regarding their Ne amplitudes ($F_{(5,87)}$=31.50; P<0.001) (see Fig. 2). There was no significant group difference for Nc amplitudes ($F_{(5,87)}$=1.29; P=0.274). It is revealed that pHDs (−11.62±0.62) and young controls (−11.35±0.58) showed the strongest Ne, not differing from each other (P>0.3). The pHD-group differed from all other groups (P<0.019). Young controls showed higher amplitudes than both PD-groups (PD-off: −4.70±0.61; PD-de novo: −4.43±0.62) and the symptomatic HD-group (−6.01±0.62) (P<0.002). The symptomatic HD-group and both PD-groups did not differ from each other (P>0.9). The old control group showed a stronger Ne (−8.75±0.58) than both PD-groups (P<0.001) and the

Table 3. Error rates on congruent and incongruent trials, separated for each group

	Congruent mean (SEM)	Incongruent mean (SEM)
Young control	3.6 (1.2)	15.4 (2.3)
Old control	2.0 (1.0)	11.8 (2.1)
PD-off	5.9 (1.1)	16.8 (2.2)
PD-de novo	1.7 (1.1)	10.8 (2.3)
pHD	4.8 (1.1)	14.3 (2.3)
HD	3.5 (1.1)	15.8 (2.3)

The M and standard error of the M is given.

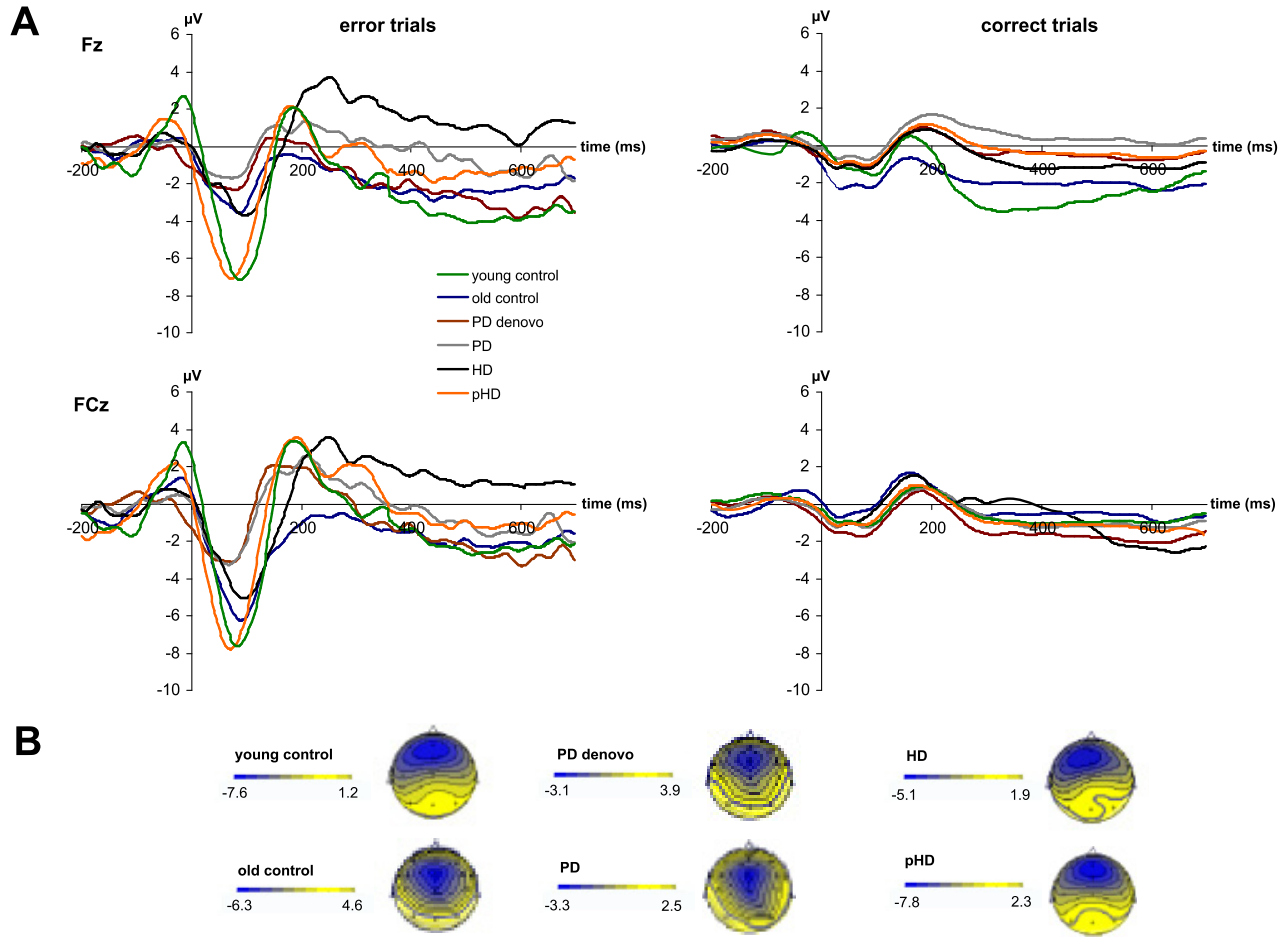

Fig. 1. Response-locked ERPs at electrode Fz and FCz for the error trials (right). Time point 0 denotes the erroneous response. As can be seen, a clear Ne is revealed in all groups, differing in their amplitude. A usual topography of the Ne is seen in all groups (left), with the Ne being maximal at frontal sites.

symptomatic HD-group (P=0.029) but a smaller Ne than the young controls (P<0.01).

Also the main effect "group" was significant ($F_{(5,87)}$= 19.18; P<0.001). Both PD-groups showed the lowest am-

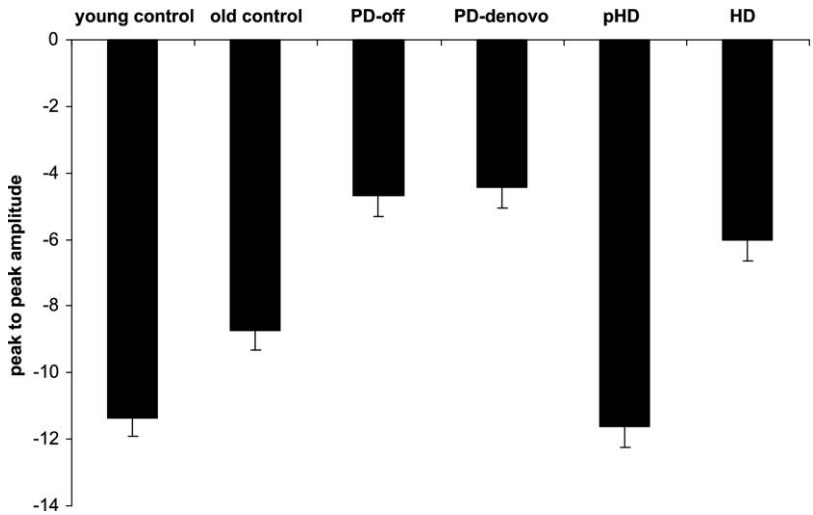

Fig. 2. Illustration of the main effect "group." Peak-to-peak amplitudes (in μV) of the Ne averages across electrodes Fz, FCz and Cz are given for each group separately.

Table 4. Significant differences between the groups (based on the main effect group)

	Young control	Old control	PD-off	PD-de novo	pHD	HD
Young control		ns	*	*	ns	*
Old control			***	***	*	ns
PD-off				ns	***	ns
PD-de novo					**	ns
pHD						*

plitudes not differing from each other (PD-off: −3.53±0.35; PD-de novo: −3.36±0.36) ($P>0.9$). The Ne was lower in the PD groups compared to all other groups ($P<0.001$), except the HD-group (−4.51±0.36) ($P>0.4$). The old control group (−5.75±0.34) showed lower amplitudes than the pHD-group (−7.36±0.37) ($P=0.030$). The young control (−6.29±0.36) group did not differ from old controls, and the pHD-group ($P>0.6$), but from all other groups ($P<0.014$). The HD-group did only differ from pHDs and young controls ($P<0.014$) (see also Table 4). For the ERP-latencies, the repeated measures ANOVA revealed no effects (all F's<1.4; $P>0.23$).

To control for possible confounders related to the degree of motor impairment (UHDRS or UPDRS scores) and of depressive mood disturbances (BDI score) additional ANCOVAs were calculated incorporating these scores. It is shown that the level of motor impairment (UHDRS or UPDRS scores) did not modulate group differences in the modulation of neurophysiological processes in erroneous and correct trials ($F_{(1,86)}=0.58$; $P>0.8$). A similar result is obtained when using BDI scores ($F_{(1,86)}=0.9$; $P>0.4$). Furthermore, an ANCOVA using the amplitudes at electrodes Fz, FCz and Cz as within-subject factor, group as between-subject factor and RT on error trials as covariate was calculated. It is shown that the covariate "RT" did not modulate amplitude variation at the different electrodes ($F_{(2,172)}=0.43$; $P>0.5$). A similar result is obtained when assessing a possible modulation of the average effect of the amplitude on error trials by the RTs ($F_{(1,86)}=1.25$; $P>0.3$).

DISCUSSION

In the current study we examined error processing, by means of ERPs. A clear Ne was seen being larger than the Nc on correct trials. The main finding of the study was that Ne-amplitudes were differentially modulated by group, i.e. age and type of basal ganglia disease. All groups were able to perform behavioral adaptation; despite some of them having a reduced Ne. This shows that a certain comparable degree of posterror slowing occurs in all groups irrespective of the overall size of the Ne/ERN in this group. Given the frequently-reported results of a within-subject relation of Ne amplitude with slowing (e.g. Debener et al., 2005) this result suggests that age- and disease-related changes in the overall Ne level do not change overall slowing. Slowing is an important strategy for avoiding further errors, and may depend on personality factors such as risk taking. Hence, slowing may be adjusted to a certain individual level regardless of the overall size of the Ne.

The behavioral data indicated that all groups committed a comparable amount of errors. Thus the group differences in the Ne amplitude are unbiased by the frequency of errors. Furthermore, the groups did not differ on correct trials suggesting specific effects related to error processing. Also possible influences of depressive symptoms (BDI) and the degree of motor impairment (UHDRS/UPDRS) are unlikely to influence the observed pattern. Similarly, the modulation of the Ne amplitudes seems to be unbiased by the different RTs, as revealed by the ANCOVA.

Old controls showed a smaller Ne than young controls. As the Ne critically depends upon the mesocorticolimbic DA-system (Holroyd and Coles, 2002), the decline of the Ne is likely due to a dysfunction of this system occurring during normal aging (for rev. Bäckman et al., 2006). Furthermore symptomatic HDs and both PD-groups showed a smaller Ne than old controls. This effect is unlikely to be biased by possible medication (after)effects, as similar reductions are obtained for the PD-de novo, the PD-off group (see also: Stemmer et al., 2007) and the unmedicated symptomatic HD-group. Even though this in line with recent studies (Willemssen et al., 2008), it is puzzling that a function, which is devoted to the dopamine system, is unaffected by DA treatment in a "DA disease." Yet, it is well known that PD is not a disease solely related to the DA-system (e.g. Hucho, 1995; Ahlskog, 2007). Hence, pathogenic mechanisms that contribute to the picture of PD and deterioration of cognitive functions are unlikely to be restricted to DA-system and also unlikely to be affected by DA treatment. Moreover, due to the "overdose" model (for rev. Cools, 2006) DA medication is accepted to improve the motor symptoms, but the effects on cognitive performance are more complex and not clear. Hence, DA treatment may leave error processing functions unaffected of DA medication.

Neurophysiological mechanisms reflecting error processing are differentially modulated by age and different basal ganglia diseases, but are not differentially modulated by the different types of basal ganglia disorders. Hence the different basal ganglia disorders, regardless of their type and (in PD) their degree, induce a uniform and profound attenuation of the Ne mechanism beyond the normal attenuation observed in healthy aging. This pattern of results differs from the study by Wild-Wall et al. (2008). The current results suggest that functions, which are partly mediated via the basal ganglia and the ACC, as it is the case for error processing (Rushworth et al., 2004) are differentially modulated by age and type of basal ganglia changes. However, it should be noted that also changes in the orbitofrontal and lateral frontal cortex may also modulate this pattern, even though the literature focusing on these brain areas and the relation to the Ne/ERN is less consistent.

The results obtained in elderly people, compared to younger are in line with several other studies (e.g. Hogan

et al., 2005; Mathewson et al., 2005; Themanson et al., 2006). There are also several studies examining effects of PD on error processing (e.g. Falkenstein et al., 2001; Holroyd et al., 2002; Ito and Kitagawa, 2006; Stemmer et al., 2007; Willemssen et al., 2008). With one exception (Holroyd et al., 2002) all these studies found a reduction of the Ne in this disease, beyond the effects of normal aging, which is underlined by the current data. Similarly, the results underline findings of recent studies in HD that have shown that the Ne is reduced (Beste et al., 2006, 2008), when compared to healthy age-matched controls or presymptomatic gene mutation carriers. However, until now it was not clear how these conditions relate to each other.

Combining the results of Wild-Wall et al. (2008) and the current, it seems that MFC-networks mediating different cognitive functions differ in their vulnerability to age-related and basal ganglia dysfunctions. The network mediating error processing functions seems to be relatively robust against subtle changes of the DA-system or other subserving MFC-systems, as they are expressed in healthy older people. When DA-dysfunctions get more severe, as it is the case in the PD-groups and the symptomatic HD-group, error processing functions further deteriorate. It may be speculated that timing functions, compared to error processing functions, are organized in a "all-or-none" principle, i.e. if a system subserving timing functions is only faint dysfunctional, performance declines, whereas graduated dysfunctions in the DA-system lead to a similar graduated dysfunctions of neurophysiological processes of error monitoring. Future studies should focus on this topic. It may hence be speculated that age effects are not necessarily similar to effects of basal ganglia diseases, and may depend on characteristics of the network mediating the cognitive function.

Another interesting finding of the study was a lack of difference between the symptomatic HD-group and both PD-groups, while an age-dependent difference was observed between the healthy young and old control group, which were matched in age to the clinical groups. Thus, the results suggest that pathogenic effects may override aging effects on the Ne, which should normally appear due to the large age difference. It is likely that this depends on the degree of dysfunction. As revealed by the data, the pHD-group showed a much stronger Ne, despite that neuroanatomical and neurofunctional deteriorations are already evident in this stage (Kassubek et al., 2005; Bohanna et al., 2008), but may be counteracted by compensatory processes (Beste et al., 2007), which explains the lack of difference to healthy young controls. While HDs are much younger than healthy old controls, they display a weaker Ne than the controls. As HDs therefore display a pattern more extreme than observed in aging, but less expressed than in another basal ganglia disorders (i.e. PD) in a higher age, it may be hypothesized that HD may reflect an extreme form of aging, when referred to error processing functions.

CONCLUSIONS

In combination with findings from other studies, the results show that it may depend on the functional organization, underlying a cognitive function, if neurodegenerative basal ganglia disorders are comparable to healthy aging. Modulations of error processing differ between aging and type of basal ganglia disease. Despite that the examined basal ganglia disorder groups (PD and HD) differ in their age they show similar modulations in error processing, suggesting that aging effects are overridden by pathogenic effects. The study shows that it may be valuable to compare aging to different forms of basal ganglia disorders, in order to gain knowledge about age- and disease-related mechanisms and the effects of these on cognitive functions. Diseases of the basal ganglia may impact error processing above and beyond the effects of normal aging. Although many aging, PD and HD studies on error processing functions have already been published, this study ties together several related observations across all of these groups in one experiment.

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