Mapping alexithymia: how the brain identifies, processes and talks about emotions

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Introduction: Alexithymia is a personality construct characterized by a continuum spectrum of difficulties in identifying, interpreting and communicating feelings. Aim of this study is to provide a brain model of physiological activation, processing and communication of emotions, and to investigate the association between alexithymia and brain functionality in these three conditions.

Methods: Nineteen healthy young subjects underwent the TAS-20 scale and a functional MRI (fMRI) scan. The 2x3 fMRI design included 2 conditions (neutral-N and negative emotional-NE) and 3 tasks: physiological activation (film watching), processing (judgment of felt emotion pleasantness/intensity), and preparation for emotion communication (felt emotion report). In each task, one-sample t-tests and multiple regressions were performed to investigate differences between N and NE conditions and the relationships between the NE fMRI signal and TAS-20 scores.

Results: Three subjects were alexithymic according to TAS-20. Compared to N, NE condition showed: a greater recruitment of the anterior and middle cingulum, superior frontal cortex, and supplementary motor area during the activation task; an additional activity of the precuneus and middle frontal cortex during processing; an additional involvement of the occipital associative cortices for communication. In the three tasks, all the regions were less activated with increasing values of TAS-20.

Conclusions: The brain emotional system includes cognitive and sensorimotor regions responsible for emotional awareness and body sensations, parietal and frontal regions for recalling and processing emotional memories, and cortical associative regions to organize emotional material for communication. Alexithymia is associated with a reduced recruitment of each part of this complex system.

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EP3115

Discriminative ability of the “Parkinson’s disease – cognitive functional rating scale” in cognitive impairment profiles different from Parkinson’s disease

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Introduction: The “Parkinson’s Disease-Cognitive Functional Rating Scale” (PD-CFRS) was recently validated as a useful instrument to explore the impact that cognitive decline in Parkinson’s disease (PD) exerts on functional aspects of daily living. It is presently unknown whether the instrument captures similar construct in other conditions associated to cognitive impairment (CI).

Methods: In addition to the PD-CFRS, 200 patients received a comprehensive cognitive assessment (MMSE, MoCA, PD-CRS and MDRS-2). Thirty-one mild cognitive impairment (MCI)-amnestic patients (32%), 33 MCI-multidomain (34%) and 33 PD-MCI (34%) completed the non-demented group. Dementia group consists on 35 Alzheimer (AD) patients (34%), 34 with vascular dementia (VD) (33%) and 34 PD with dementia (PDD) (33%). The Blessed Dementia Scale (BDS) and the Global Deterioration Scale (GDS) were used as functional “gold standards”. Coefficients of variation, logistic regression, effect-size analysis and ROC curves measured the PD-CFRS discriminative ability between conditions.

Results: The PD-CFRS presented high concurrent validity with the BDS (ICC=0.828) and elevated correlation levels with all functional and cognitive scores (for all p<0.001). ROC curve analysis [AUC=0.992; 95%CI: 0.984-0.999] showing a PD-CFRS cut-off score of ≥9 [(SEN=0.942; SPE=0.948)] for detecting functional impairment in dementia. A similar cut-off (≥9) resulted when excluding PD patients (AUC=0.997; SEN=0.986; SPE=0.969). Cohen’s d >2.60 (≤10% overlap) between MCI and dementia subgroups indicate an irrelevant interference between PD-CFRS scores.

Conclusions: The discriminative properties in diagnostics other than PD suggested that the PD-CFRS is an excellent tool to evaluate functional aspects in the transition from MCI to dementia.

Disclosure: Nothing to disclose

EP3116

Cortical blindness with residual face perception: a case of facial blindsight

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Introduction: Blindsight refers to unconscious residual visual abilities despite destruction of visual cortex. Such capabilities have been described in several patients for color and shape discrimination, facial emotion recognition or navigation skills.

Methods: Here we present A.M., a patient suffering from partial cortical blindness (he presents only small degrees of brightness perception) with residual abilities in face processing. We designed forced choice tasks to test form perception (Figure 1A-B), color perception, face perception (Figure 1C, E, G, H), and emotion perception (Figure 1D,F).

Results: A.M. presents the remarkable capacity to distinguish between jumbled/normal faces, known/unknown faces and famous people’s categories even if he isn’t able to recognize or even describe them. In contrast, he performs at chance for form and color discrimination.

Conclusion: This case confirms that face processing involves distinct neural system from object recognition and suggests that it could occur without complete awareness through a subcortical pathway independent of the primary visual cortex.

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EP3117
Neuropsychological assessment in SCA36: ‘Costa da Morte’ ataxia
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Introduction: SCA36 is a recently described spinocerebellar ataxia (SCA), caused by an intronic GGCCTG repeat expansion in NOP56, relatively frequent among Galician patients with ataxia. In addition to cerebellar manifestations, motor neuron symptoms and sensorineural hearing loss are additional features of SCA36. Although cognitive impairment in patients with different SCAs is variable, prefrontal dysfunction is common to all subtypes. Our aim was to explore cognitive and affective areas in SCA36.

Methods: We evaluated 15 SCA36 patients (9 women, 6 men, mean age 62.7±14.4 years), from ‘Costa da Morte’, a coastal region in Northwestern Spain. All study subjects had a genetically confirmed NOP56 expansion and variable severity of motor dysfunction measured through the SARA scale. The following tests were used for the study: Frontal Assessment Battery (FAB), to measured frontal and executive functions; Mini-Mental State Examination (MMSE) for general cognitive performance and Geriatric Depression Scale (GDS) to detect the presence of affective problems.

Results: The mean score obtained with the FAB was 12.1±3.9, while MMSE results were 24.8±4.4 and GDS score ranged 14±8.2

Conclusions: Although preliminary, these results indicate that in SCA36 there is mild cognitive impairment as disease progresses, however dementia is uncommon. In concordance to what is observed in other SCAs, SCA36 patients show a variable degree of fronto-executive deficit. Special attention should be paid to affective aspects and mood, that are also frequently altered. Sensorineural hearing impairment, characteristic of SCA36, hampers the application of some neuropsychological tests.

Disclosure: Nothing to disclose

EP3118
Cognitive impairment in multiple sclerosis patients: validity of a computerized cognitive screening battery
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Objective: To investigate the pattern of cognitive impairment in relapsing-remitting multiple sclerosis (RRMS) and secondary progressive multiple sclerosis (SPMS) patients, using a computerized battery.

Methods: RRMS patients (N=50), SPMS patients (N=30) and healthy controls (N=31) were assessed by a computerized neuropsychological battery (Central Nervous System Vital Signs, CNS VS), Trail Making Test (TMT) A, B, semantic and phonological verbal fluency tasks.

Results: The overall prevalence of cognitive dysfunction was 53.75%, while frequency of cognitive dysfunction was 38% for RRMS and 80% for SPMS patients. Comparison of performance between groups demonstrated that RRMS patients differed from controls with large effect size on reaction time, medium effect size on TMT A and small effect size on TMT B, phonological verbal fluency task, composite memory, psychomotor speed and cognitive flexibility. SPMS patients differed from controls in all neuropsychological measures (except complex attention) with large effect sizes on TMT A, B, phonological verbal fluency task, composite memory, psychomotor speed, reaction time and cognitive flexibility. Between patient groups (RRMS and SPMS), medium effect sizes were present on TMT B and psychomotor speed, while small effect sizes were present on composite memory and processing speed.

Conclusion: CNS VS appears to be sensitive in detecting cognitive impairment in RRMS and SPMS patients. Significant impairment in episodic memory, executive function and processing speed was detected, with gradual increment of the frequency as disease progresses.

Disclosure: Nothing to disclose
EP3119

Theory of mind in multiple sclerosis

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Introduction: Social behaviour and interaction is strongly linked to the ability to understand the minds of others and their feelings. Theory of Mind (ToM) is defined as the capability to make inferences about mental states of other individuals. Social cognition allows to understand the mind of others, acting according such information and process. Our aim is to evaluate and assess the ToM in patients of Multiple Sclerosis (MS).

Methods: We studied 38 patients of Relapsing Remitting Multiple Sclerosis (RRMS) with EDSS of 2 (or less) utilizing the Test of Eyes Expression (Baron Cohen, 2001), Facial Affect Recognition and the Faux Pas Test. Neuropsychiatric and Cognitive profiles were studied with standard batteries for MS.

A healthy control group of 38 individuals, was paired according age, gender, education and intellectual level.

Results: MS patients were significantly impaired in ToM Test, specially in the facial affect recognition (mainly decoding emotions of anger and fear) and in social behaviour related personal interaction (faux pas). These results had no correlation with cognitive performances or other variables linked to disease (EDSS, evolution time, treatment) in our group of patients.

Conclusions: Our findings suggest that Social Cognition and ToM are disrupted functions in RRMS patients, and are no related with neuropsychological deficits. This compromise (deficits in emotional decoding, inference of mental states of other individuals, ability to perceive other’s feelings) could contribute to increase the psychosocial burden of our patients, increasing disability in MS.

Disclosure: Nothing to disclose

EP3120

Behavioral dysexecutive disorders in Huntington’s disease

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Dysexecutive symptoms such as apathy, impulsivity and distractibility have been reported on clinical ground in HD. However previous studies have assessed dysexecutive behavioral disorders using clinical interview or nonspecific questionnaires incorporating other features resulting in highly variable evaluation with poorly defined performance indices. This study aimed to assess dysexecutive behavioral abnormalities in HD, using a validated instrument, the Behavioral Dysexecutive Syndrome Inventory (BDSI)[1].

14 patients (mean age: 55.5 years; SD 12.4; range: 34-73; mean duration: 8.7 years; SD=7; range: 2-30; mean MMSE score: 25; SD=3.4) with clinically diagnosed and genetically confirmed HD, were included. The assessment of depressive symptoms (Montgomery Asberg Depression Scale) showed a mild depression in 7 patients.

The BDSI is a highly structured caregiver based interview which rates frequency and severity of 12 dysexecutive disturbances (global hypoactivity with apathy, hyperactivity, irritability-impulsivity, euphoria, perseverative behavior; environmental dependency, social behavior disorders). The analysis of individual performance was performed using cutoff scores at the 5% level using normative data obtained in 96 controls[1].

The prevalence of behavioral dysexecutive syndrome was high (50%; 95%CI: 24-76). The behavioral profile was characterized by the prominence of irritability (50%), hyperactivity (43%), apathy (29%), disinterest (22%) and difficulties for anticipation (14%). This study based on the BDSI revealed in HD that behavioral dysexecutive disorders are: (1) frequent with a prevalence of 50%, (2) characterized by a specific profile with the prominence of irritability and hyperactivity (3) whereas the hypoactivity-apathy disorders were less frequent contrary to most other degenerative disorders.


Disclosure: Nothing to disclose
EP3121

A voxel based morphometric study to investigate volume differences in regional gray matter between patients associated with different subtypes of vascular cognitive impairment

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Introduction: Voxel-based morphometry (VBM) was used to investigate volume differences in regional gray matter between patients suffering from mild cognitive impairment associated with periventricular white matter hyperintensities (PWMH) and strategic single-infarct (SSI).

Methods: 14 patients with PWMH, 10 patients with SSI after 6-month poststroke time window and 16 healthy controls were included in this experiment. Participants were neuropsychologically tested to characterize cognitive function in 7 domains: orientation, attention, working memory, language, visuospatial ability, psychomotor speed, and memory. Magnetic Resonance Imaging scans were acquired and whole brain regional differences in grey matter volume between 3 groups were examined with VBM. Two-sample T-test models were used to assess the contribution of demographic variables, stroke-related variables, and voxel-based morphometry results to classification of cognitive impairment group membership.

Results: SSI and PWMH showed significant volume difference in regional gray matter. Significant grey matter volume reductions mostly in the bilateral temporal lobes were found in PWMH, compared with SSI. Decreased gray matter density in the left prefrontal areas could also be seen in PWMH.

Conclusions: These findings suggest that grey matter atrophy in temporal cortical and prefrontal regions are of particular relevance to PWMH-related cognitive decline, from a morphological point of view. Characteristics of grey matter atrophy in PWMH which is similar to degenerative disease prompt that merger or secondary neurodegenerative changes in the vascular cognitive impairment subtypes can not be ruled out.

Disclosure: Nothing to disclose

EP3122

Cerebral microbleeds and cognitive impairment in neurodegenerative and cerebrovascular diseases

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Introduction: We hypothesize that cerebral microbleeds (CMBs) influence in cognitive decline. Their presence and localization might be an additional criteria for diagnosis of dementia. Previous study demonstrated that patients with DLB (dementia with Lewy bodies) have more CMBs than patients with AD (Alzheimer’s disease).

Methods: We studied 120 outpatients with cognitive decline older than 65 years. MRI was performed on MR tomograph 1.5 Tesla. CMBs were analyzed using microbleeds anatomical rating scale (MARS). Neuropsychological battery included Montreal Cognitive Assessment scale (MoCA), Addenbrooke’s Cognitive Examination (ACE-R), Clock Drawing Test, fluency test and the visual memory test (SCT).

Results: We did not find cortical CMBs in patients with DLB in most cases (78%). Multiple (more than 3 cortical) CMBs were observed in two DLB cases only and might be considered as sporadic CAA (cerebral amyloid angiopathy). Most CMBs were observed in patients with AD+VaD (Vascular dementia) (73%) compared to AD+DLB (11%). CMBs were associated with worse memory and visuospatial functional domains and the total ACE-R score in AD+DLB groups in comparison with DLB negative CMBs group (p<0.05).

Conclusions: We hypothesize that a vascular pathological process is an universal factor which contributes to neurodegeneration irrespective of the type of dementia. CMBs might be a more sensitive indicator of the severity and clinical significance of cerebrovascular disease than the severity of leukoaraiosis. Multiple CMBs might represent an independent factor of cognitive decline. We guess that the vascular changes in LBD observed in Alzheimer’s involvement are a component that requires additional pathological studies.

Disclosure: Nothing to disclose