ACTIVATION TASKS AND MOTOR FUNCTION IN PATIENTS WITH NEUROLEPTIC-INDUCED AKATHISIA

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The aim of this study was to evaluate the effect of activation tasks on neuroleptic-induced akathisia (NIA). Previous studies by Fleischhacker et al. (1993) revealed a decrease of symptoms during motor activation, whereas there was no consistent effect of mental activation on NIA.

In our study we investigated 11 patients suffering from acute NIA according to the criteria by Braude et al. (1983). Different activation procedures (motor activation, mental activation by simple and by difficult serial calculations) were carried out and akathisia scores (Barnes Akathisia Scale, Hillside Akathisia Scale, Analogue Scale) were determined using video rating technique. Motor activation led to a distinct decrease of symptoms (Barnes Akathisia Scale, objective part: $p < 0.005$). Likewise mental activation by simple serial calculations produced a significant reduction of NIA ($p < 0.05$), while mental activation under stressful conditions (difficult serial calculations) had no consistent effect. The diverse mental activation procedures differed significantly in their effects on NIA ($p < 0.05$).

We concluded that motor activation as well as mental activation reduces the symptoms of NIA, whereas stressful events increase them. Activation procedures might be helpful in differentiating NIA from other antipsychotic-induced movement disorders.

DYSFUNCTION OF SUPPLEMENTARY MOTOR AREA (SMA) IN CATATONIA: MOTOR ACTIVATION STUDIES WITH PET AND F-MRI

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Introduction: Catatonic patients do often show akinesia, which may be reversed by external stimulation. Such a deficit of the internal initiation of movements may be related with dysfunction of the Supplementary motor Area (SMA), which functionally is responsible for initiation of movements. We therefore investigated catatonic patients during activation with self-paced and self-initiated sequential finger opposition (SFO) in PET and F-MRI.

Methods: We investigated 10 catatonic patients (mean age: 33.2 −/− 5.4 years; 6 females, 4 males) according to diagnostic criteria by Lohr (1987), Rosebush (1990) and DSMIII/R. in a postacute state and compared them with healthy controls. PET studies were performed on a SIEMENS ECAT 47 PET tomograph. Before and during injection until 10 minutes after infection of Fluor-18 Desoxyglucose (FDG), patients continously performed sequential finger opposition (SFO) as a motor activation task. Similar motor task were performed with functional MRI. Functional MRI was performed using a gradient echo-EPI pulse sequence with TR = 1.8 ms, TE = 66 ms, FOV = 23 cm, matrix = 64 × 64 and voxel size = 3.13 × 3.16 × 4 mm. Series of 60 sequential multislice images parallel to AC-PC line were obtained.

Results: Controls showed consistent activation of motor cortex and supplementary motor area (SMA) in PET and fMRI. Catatonic patients showed no significant difference in activation of the motor cortex in either PET and fMRI. In contrast SMA activation in catatonic patients was significantly decreased when compared to controls. Furthermore 4 from 10 catatonic patients did not show any activation of SMA at all in either PET and fMRI.

Conclusion: Catatonic patients showed "normal" activation in motor cortex whereas activation of SMA was significantly reduced or even absent in either PET and fMRT when compared with healthy controls. Subsequently if may be concluded that SMA is hypofunctional in catatonia which could account for catatonic akinesia.

CATATONIA AND EXTRAPYRAMIDAL DISORDER

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There is a popular impression that the prevalence of catatonia had decreased compared with 100 years ago when it was first described. There is no real evidence of such a decrease in presentation. Catatonia remains a rare severe neuropsychiatric disorder and catatonic motor disorder a common part of severe psychiatric illnesses which can improve with successful treatment of such illnesses. What has changed over the last 100 years is the conceptual approach to catatonia. Catatonia was first described shortly before the syndrome of dementia praecox or schizophrenia was introduced, and was soon accepted as a integral part of this syndrome but has constantly been described in other disorders, both psychiatric and neurological. Extrapyramidal disorder was first described about the same time as catatonia but was restricted to neurological illnesses. Attempts by a few authors in the 1920s to apply this new neurological concept to psychiatric illnesses was unsuccessful. A conceptual divide between neurological and psychiatric disorder started at the same time that catatonia and schizophrenia were first described and has continued until very recently. Catatonia was psychiatric and extrapyramidal disorder neurological. However, the psychiatric aspects of extrapyramidal disorder, and the neurological basis of catatonia have gradually become established. For those who wanted to maintain a non-cerebral approach to psychiatric disorder, it became convenient to regard catatonia as belonging to a previous era and to be completely neurological. A more modern approach would be to accept catatonia as an extrapyramidal motor disorder which can occur as part of neurological or psychiatric illnesses.

CONCEPTUAL ISSUES IN CATATONIA RESEARCH

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In this review, the psychopathological aspects of catatonia will be examined in three overlapping areas:

1. The lack of psychopathological definition of catatonia: The fundamental problem, namely, by what psychopathological principle can a particular motor symptom be categorized as catatonic, has not been addressed by modern writers. As a result, the delineation of the clinical concept of catatonia remains to be arbitrary. Of all the authors, only Jaspers has provided a psychopathological definition of catatonia.

2. Deficiencies in the definition of catatonic symptoms: There seems to be no consensus regarding the description of particular catatonic signs and symptoms. Catatonia research cannot advance any further if it continues to operate with simplified descriptive terms.

3. Catatonia: syndrome and/or a group of disease entities? The currently prevailing "syndromal" view of catatonia fails to take into account the progress descriptive psychopathology has made in the past 150 years. The nosological standpoint of Wernicke-Kleist-Leonhard school implies a hierarchical model of catatonia with (1) motor symptoms and syndromes due to well-defined psychiatric and medical conditions and, (2) permanent aggregations of certain motor and other psychiatric symptoms without detectable gross organic pathology forming the putative disease entities of periodic and systematic catatonias.