

**CEREBRAL BLOOD FLOW IN POST-STROKE DEPRESSION**

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**Summary:**

Depression affects 50% of stroke patients. We hypothesized that severity of depression is related to the lesion's size and its proximity to the frontal pole. To test these hypotheses, we used  $^{99m}\text{Tc}$ -HM-PAO SPECT (single photon emission computer tomography) to define regional blood flow impairment in 15 patients (13 male; age  $65 \pm 10$ ) undergoing stroke rehabilitation.

We measured lesion volume by summing the areas with a 25% reduction in blood flow compared to the contralateral side. Impairment in frontal lobe blood flow was determined by the ratio of average counts per pixel in the damaged frontal lobe over the normal frontal lobe. Depression was rated blindly using the Hamilton Depression Scale.

We found that depression scores correlated with lesion volume ( $r=.49$ ,  $p=.07$ ,  $df=14$ ). Patients with SPECT lesions were significantly more depressed than patients without SPECT lesions fulfilling our 25% criteria ( $t=2.53$ ,  $p<.05$ ,  $df=13$ ). Depression scores tended to rise with blood flow deficits in either frontal lobe ( $r=.44$ ,  $p=.10$ ,  $df=14$ ). There was no difference in severity of depression between patients with right and left frontal deficits ( $t=.94$ ,  $p>.2$ ,  $df=13$ ). This pilot study suggests that regional blood flow imaging by SPECT is a valuable tool for investigating the pathophysiology of post-stroke depression.

**NADOLOL FOR CHRONIC IMPULSIVE AGGRESSIVE BEHAVIOR**

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**Summary:**

Eleven inpatients with chronic mental illness and chronic impulsive aggressive behavior were entered into a double blind, placebo controlled study. Criteria for entry into the study included a two-month history of four episodes per month of impulsive aggressive behavior as described in the Overt Aggression Scale. Subjects also met the first two criteria of the diagnosis of Intermittent Explosive Disorder as described in *DSM-III*. Subjects were maintained on concurrent psychotropic medication, with all dosages frozen at pre-study levels. One patient was dropped due to a hypotensive episode while taking placebo; one patient was dropped due to worsening aggressive behavior. Initial analysis shows consistent trends toward greater improvement in the treatment group as measured by BPRS, CGI, irritability subscale of the Nosie 30, and by modified OAS. CGI severity scale showed a pre-treatment mean of 4.6 for the placebo group, and 5.0 for treatment group with post treatment means being 4.6 and 3.67, respectively. CGI improvement score consistently favored the treatment group with post treatment means of 3.4 for placebo and 2.3 for treatment group.

**NEUROCHEMICAL CORRELATES OF DEFICIT SCHIZOPHRENIA**

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**Summary:**

Biological correlates of deficit characteristics in schizophrenia are being increasingly sought. In the literature, higher cerebrospinal fluid (CSF) 5-hydroxyindoleacetic acid (5-HIAA) concentrations, and lower CSF homovanillic acid (HVA) concentrations have been associated with slowed motor behavior and communication in schizophrenic patients. To derive a single, reliable measure of deficit characteristics in schizophrenic patients, we entered three items of the Brief Psychiatric Rating Scale (BPRS) reflecting negative symptoms, a work-history measure derived from the Strauss-Carpenter Scale, and three subscale scores of the Wechsler Adult Intelligence Scale-Revised (WAIS-R) into a principal components analysis to derive a single factor score. CSF HVA concentrations were not associated with this deficit factor score in schizophrenic patients. However, CSF 5-HIAA concentrations directly correlated with this factor score, and post-hoc bivariate correlation estimates between each of the measures comprising the factor and CSF 5-HIAA suggested that all measures contributed to the correlation between the factor score and CSF 5-HIAA concentrations. These findings add support to the hypothesis that brain serotonin function is linked to deficit schizophrenic characteristics.