

chronic schizophrenia. The electro-oculogram was used to monitor eye movements while each patient observed a luminous spot on an oscilloscope screen, which moved with a sinusoidal motion at a frequency of .4 Hz for 20 cycles. Tracking accuracy was analyzed by a computer which computed the root mean-square error (the difference between the target and the electro-oculographic signal expressed in standard deviation units). The clinical status of patients was assessed with scales that assess severity of symptoms (BPRS, SCL-90) and global functioning (GAS, AXIS-V). In addition, chronic schizophrenics were assessed in terms of duration of illness and number and length of hospitalizations.

No significant differences in tracking proficiency were found between chronic and first-episode patients. In addition, none of the clinical indexes correlated significantly with eye tracking dysfunction. These findings suggest that pursuit integrity is not associated with chronicity. In addition, the present data support the conclusion that poor tracking is a trait measure and is not affected by the subject's clinical condition.

## SLEEP AND SUICIDALITY IN SCHIZOPHRENIA

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The biological basis of suicidal behavior in schizophrenia remains unclear. There is evidence for a relationship between sleep EEG abnormalities and suicidal behavior in depressive patients. Schizophrenia is associated with: 1) reduced rapid eye movement (REM) latency; 2) reduced slow wave sleep (SWS); 3) impaired sleep maintenance and efficiency; and 4) the variable increase in amount of REM sleep. We examined the relation between suicidality and EEG sleep in medication-free (>2wks) schizophrenic and schizoaffective patients with (Sch-S) (n=20) and without (Sch-N) (n=21) suicidal behavior (ideation, attempts).

The lifetime history of suicidal behavior was scrutinized by review of hospital charts by a research assistant blind to the sleep data. Ten patients had ideations alone; 10 patients had ideations and attempts. Hand scored EEG measures were compared between the two groups using ANOVA. Sch-S had significantly higher Hamilton scores than Sch-N patients. REM time (minutes) and REM activity in the first REM period were significantly increased in the Sch-S group. There was a trend for the total REM time and REM activity to be increased in the Sch-S group as well. The Hamilton Depression scores did not correlate significantly with any of the sleep measures. The association between suicidality in schizophrenia and increases in REM sleep is consistent with: 1) data suggesting the inhibitory effect of serotonin for REM sleep, and 2) the association between reduced serotonergic function and suicidality in psychiatric disorders.

## SENSORY GATING ASSESSED BY P50 SUPPRESSION IN NORMAL CONTROLS: EFFECTS OF CHANGING STIMULUS INTENSITY

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Schizophrenics are reported to be deficient in auditory-evoked P50 suppression compared to controls. Substantial disagreement exists, however, regarding the amount of suppression reported in controls. To investigate methodological factors which may explain this inconsistency, we studied controls using different stimulus intensities.

10 volunteers with no history of mental illness were assessed (6 males/4 females, mean age 31.6 years) using a conditioning-testing paradigm. EEG was recorded from CZ, bandpass filtered for 0.3-300 Hz, amplified  $\times$  100,000, digitized and recorded for 250 msec following each click. Epochs reading  $\pm$  100  $\mu$ V were rejected. 120 click pairs (.04 msec click duration, 500 msec inter-click interval) of 90 dB or 110 dB peak intensity were randomly delivered at 10 second intervals. P50 suppression scores were calculated for each click intensity.

Significant suppression was noted at each click intensity. Mean suppressions were 66.4% and 67.6% at 90 and 110 dB respectively (repeated measures ANOVA  $P < .008$ ). Although no significant difference was noted between P50 suppression at 90 or 110 dB, conditioning P50 amplitude was significantly reduced at 90 dB compared to 110 dB (repeated measures ANOVA,  $P < .002$ ).

These early findings suggest that P50 suppression is unaffected by changes in stimulus intensity in this range. Further work is essential to identify factors which account for the current divergence in the literature.

## ATTENTIONAL DEFICIT ASSESSED WITH AERPs AND NEUROPSYCHOLOGICAL MEASURES IN SCHIZOPHRENIA: A FIVE YEARS STUDY

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Auditory event-related potentials (AERPs) were analyzed in a cross-montage at (Fz, Cz, Pz, C3, C4) in a signal detection task. Two groups of 15 schizophrenics with or without major formal thought disorders (+FTD, -FTD) were tested in a lexical decision task and were administered the Luria-Nebraska (LN) Neuropsychological Battery. Ss were tested in a longitudinal follow-up design with retest after 5 years following identical experimental and counterbalancing method to pre-test. Clinical evaluation was assessed according Andreasen's checklist and to psychiatric

rating of functional improvement. Results indicate that +FTD group is characterized by more psychotic positive and negative signs, by general electrophysiological "flatness", by deficient attentional modulations of frontal negativities, by pervasive attentional deficits of response-set type, by severe higher cortical function deficits and by absence of semantic priming effect, all of which get worse over time regardless of clinical development. -FTD group shows less severe psychotic symptoms, ERP indices of intrusion (large frontal N100 and P300 to ignored stimuli), cognitive perseveration (large late post response negativity FzN400–N700), distraction related to input dysfunction and hyperarousal, almost normally performance with LN, priming effect at slow speed of presentation, which may/or may not diminish over time with clinical improvement or discharge from hospital. Discriminant analysis correctly classified 85% of the Ss in each group. In conclusion, these results support the differentiation between 2 subgroups with structural versus functional pathology. Moreover the dissociation between clinical and neurophysiological variables in their temporal course indicates that the first maybe more reflective of clinical states, while the latter, more sensitive to trait aspects of the schizophrenia.

#### IMPAIRED EYE-TRACKING IN UNDERGRADUATES WITH SCHIZOTYPAL PERSONALITY DISORDER

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This study tests the hypothesis that individuals with schizotypal personality disorder (SPD) display deficits in smooth pursuit eye movements (SPEM). Such deficits constitute a putative genetic marker for schizophrenia, and therefore would be expected in SPD, which is considered to be genetically related to schizophrenia. It has been suggested that previous studies may be confounded by faulty diagnosis or by severe cognitive impairments in the clinical samples used. The present study seeks to address the issues of diagnosis and cognitive deficits by utilizing an unimpaired, undergraduate sample, and applying DSM-III-R diagnoses. The present study compares qualitative ratings of SPEM in 14 undergraduates diagnosed with SPD according to DSM-III-R criteria with 18 controls. These undergraduates were initially selected from a total pool of 822 Introductory Psychology students screened using a self-report questionnaire for schizotypy. T-tests indicated that the schizotypals had significantly worse SPEM ratings compared to controls. The two groups did not significantly differ on verbal and performance IQ subscales, indicating that SPEM deficits in SPD are not a function of cognitive deficits.

#### STABILITY OF VISUAL FIXATION IN SCHIZOPHRENICS AND NORMAL CONTROLS

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Until recently visual fixation of a stationary target was thought to be smooth pursuit of zero velocity. These two components of ocular motor behavior are now known to be at least partially independent. Nystagmus, square-wave jerks, slow drift and involuntary saccades can impair steady fixation of a stationary target. Unstable fixation has been reported in schizophrenic patients, as has failure of fixation to suppress vestibular nystagmus, but findings have not been consistent. We examined fixation stability in first-episode schizophrenics, chronic schizophrenics and normal controls using infrared reflectometry. Our results indicate that a disturbance in fixation is not characteristic of schizophrenia and support the independence of impaired pursuit from defective fixation mechanisms. The implications of these results for other features of ocular motor behavior in relation to schizophrenia are discussed.

#### REGIONAL CEREBRAL BLOOD FLOW DURING SINUSOIDAL PURSUIT, FIXATION AND SACCADIC EYE MOVEMENTS IN PATIENTS WITH SCHIZOPHRENIA AND NORMAL CONTROLS

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Saccadic eye movement abnormalities have been reported during performance of fixation and smooth pursuit eye movement tasks in patients with schizophrenia. To investigate the pathophysiologic mechanisms underlying saccadic dysfunction in schizophrenia, we used the oxygen-15 water method for measuring regional cerebral blood flow (rCBF) with positron emission tomography in 5 male patients with schizophrenia and 5 age matched male normal controls. rCBF was measured during performance of sinusoidal smooth pursuit, fixation, and visually-guided saccadic eye movement tasks. Data were collected on the Scanditronix PC2048-15B brain tomograph which produces 15 slices with reconstructed resolution of 6–6.5 mm in three planes. rCBF data were normalized (i.e. each pixel of rCBF expressed as a percentage of the whole mean brain metabolism) for data analysis. Group differences in mean rCBF for the frontal eye fields, as well as other cortical and subcortical structures implicated in the control of saccadic eye movements will be presented for all eye movement tasks. Implications of these findings for the pathophysiology of eye movement dysfunction in schizophrenia will be discussed.