In schizophrenia, blink rates are frequently elevated and the peak of the electroencephalographic alpha rhythm is often absent or of a lower frequency. Emerging evidence suggests that both blinks and the alpha rhythm may be controlled by a linked neuroanatomical circuit that begins in rostral pons and involves several subcortical structures as well as the occipital cortex. Blink-alpha abnormalities in schizophrenia further suggest that this blink-alpha neurocircuit may be a locus of the pathophysiological process of this disorder.

Recent studies of brain metabolism and neuropathology have implicated structures within the frontal and temporal cortex in the pathophysiology of schizophrenia (Buchsbaum et al. 1982; Kovelman and Scheibel 1984; Jakob and Beckmann 1986; Weinberger et al. 1986; Falkai et al. 1988). The implication that multiple cortical sites are involved in schizophrenia, however, confuses the issue of localization and might be most parsimoniously accounted for by postulating the involvement of one or more subcortical loci.

Patterson (1987) reviewed studies supporting the notion of subcortical involvement in schizophrenia. The present article presents evidence that a well-delineated group of subcortical structures, along with, perhaps, the occipital cortex, regulate both the spontaneous eyeblink rate and the alpha rhythm, important correlates of schizophrenia. Two lines of evidence are reviewed: (1) data supporting the existence of a neurocircuit involved in the modulation of the spontaneous eye blink rate and the alpha rhythm, and (2) studies concerning alterations in the blink rate and the alpha rhythm in schizophrenia.

The Blink-Alpha Neurocircuit (BANC)

This putative neurocircuit includes the pontine tegmentum rostral to the level of the seventh cranial nerve, cerebellum, substantia nigra, midbrain tectum, lateral geniculate bodies (LGB), and the occipital cortex.

The Pons. Blinks are probably initiated in the pons. The evidence for this comes from depth electrode studies in subhuman primates in which triphasic discharges occurred in the pontine reticular formation (PPRF) before each spontaneous blink (Cohen and Feldman 1968). The facial musculature responsible for blinking is controlled mainly through cranial nerve 7 as exemplified by the unilateral paralysis of blinking in facial nerve lesions. The oculomotor nucleus and its accessory nucleus, however, maintain the tone of the levator muscle necessary for the eye to remain open (i.e., ptosis accompanies lesions affecting the third nerve). Hence, blinks may involve intermittent ablations of the tonic input to the levator as well as active contraction of the facial musculature.

Lateral Geniculate Bodies (LGB). Discharges in the LGB soon after a blink, suggest that blink-related signals ascend through rostral pons and midbrain to the LGB (Cohen and Feldman 1968). As noted below,
the alpha rhythm is also prominent and coherent in the LGB of dogs (Lopes da Silva et al. 1973a, 1973b).

Substantia Nigra. Blink rate is reduced in Parkinson's disease (Hall 1945; Adams and Victor 1981; Karson et al. 1984). This reduction occurs even when the signs and symptoms of the illness are mild and is correlated with severity (Karson 1983; Karson et al. 1984). For example, the mean blink rate ± standard deviation (SD) of a group of medication-naïve patients with relatively mild Parkinson's disease was only 12 ± 10/minute compared with 24 ± 15/minute in normal controls (p < 0.01) (Karson et al. 1984). Cell loss in the substantia nigra is a pathological index of Parkinson's disease (Adams and Victor 1981; Forno 1981). It is therefore likely that this pathological degradation plays a role in the decreased blink rate, suggesting that the substantia nigra facilitates blinking in normal individuals.

Midbrain Tectum. The blink rate in progressive supranuclear palsy (PSP) is even lower than in Parkinson's disease, with mean rates of 4 ± 3/minute (Karson et al. 1984; Golbe et al. 1987). Gliosis in PSP is concentrated in tectal structures such as the periaqueductal gray, the superior colliculus, and the pretectal area (Adams and Victor 1981). Of these structures, the superior colliculus is heavily implicated by its well-documented role in visual and visuomotor functions (Denny-Brown and Chambers 1976). More direct evidence for the involvement of the superior colliculus is a report that altered blink rate is associated with mass lesions of the sylvian aqueduct (nystagmus retractorius). Such lesions usually impinge on the superior colliculus (Smith et al. 1959). Ablation of the superior colliculus in subhuman primates results in spells of staring lasting over 30 seconds (Denny-Brown and Chambers 1976). Together, these data suggest that the destruction of tectal structures, particularly the superior colliculus, decreases blink rate. As with the substantia nigra, therefore, structures in and around the midbrain tectum may facilitate blinking.

Cerebellum. The blink rate in cerebellectomized rats is quadrupled relative to that in control animals (Freed et al. 1981). Although the applicability of these data to humans may be questioned because of the relatively low frequency of blinking in rats, we also found a similarly increased rate in three children with cerebellar tumors (Karson, unpublished observations). Because the destruction of the cerebellum seems to increase blink rate, it appears that the cerebellum normally inhibits blinking.

Occipital Cortex. The blink rate is decreased by visual fixation and visual tasks such as reading (Hall 1945; Karson 1983). In contrast, positron emission tomography (PET) indicates that the amount of visual cortex activation correlates with the complexity of the visual task (Phelps and Kuhl 1981). Moreover, the targeted saccades that occur during visual tasks such as reading are the saccades most prominently associated with activation of the occipital cortex (Fox et al. 1985). Hence, the activation of the occipital cortex may inhibit blinking. But the opposite also occurs, since visual activity and electrical activity in the visual cortex are suppressed during blinks (Volkmann et al. 1979; Bisseret and Maffei 1982).

Alpha “Squeak” and the Relationship of the Occipital Cortex to Alpha Rhythm. The alpha frequency band is usually defined as electroencephalographic (EEG) frequencies between 8 and 12 or 13 Hz (Hertz). Increased peak alpha frequency during blinking, called “alpha squeak,” has been previously described (Neidermaeyer and Lopes da Silva 1982). In a preliminary study of nine normal controls who were selected because of their low blink rate and ability to still horizontal eye movement permitting the analysis of EEG around discrete blinks, Karson (unpublished manuscript, 1989) found an increase of peak alpha frequency from 9.77 to 10.78 Hz preceding blinks (p < 0.001). The alpha rhythm is most prominent and most coherent over the occipital cortex of humans. In carefully done studies in dogs (Lopes da Silva et al. 1973a, 1973b), the alpha rhythm was most prominent not only in the striate cortex, which is analogous to occipital cortex in humans, but in the LGB as well. An important and unresolved controversy is whether alpha in humans is generated by nonspecific nuclei of the thalamus or is a “stochastic” electrical event generated mainly in the cortex (Morison and Dempsey 1943; Andersen et al. 1966; Lopes da Silva et al. 1973a, 1973b). A recent computerized model fitted the alpha rhythm with a "local neuronal circuit" model, supporting the notion of thalamic generation (Lagerlund and Sharbrough 1989). Nevertheless, because of the anatomical diversity of the thalamus, the controversy concerning alpha genera-
tion, and the lack of evidence linking the thalamus to blink rate, the thalamus is included with reticence in the BANC.

Increased Blink Rate in Schizophrenia

Reliability of Blink Rate. The usual methods for measuring blinks are electrophysiological measurement, direct observation, and observation from video. Direct observations of blink rates (counts) correlate highly with electrophysiological measures of blink rates (Karson 1983). The blink rate is reliably determined between observers and over time (the intraclass correlation for measurements made every other week for 6 weeks was 0.92 [p < 0.001], in 10 normal adults [Karson, unpublished data]).

Positive Studies. In the only study from the preneuroleptic era, a mean rate of 27 ± 18/minute was found in “psychosis” compared with rates ranging from 18 ± 8/minute to 22 ± 12/minute in other psychiatric disorders (Ostow and Ostow 1945). Twenty-one percent (8/38) of the psychotic patients and 6.5 percent (4/62) of the nonpsychotic patients had blink rates above 40/minute, the cutoff for the most rapid blinkers. Specific correlations between increased blinking and schizophrenia (cf. Cancro and Van Gelder 1982) and between blink rate and brain dopamine systems (Stevens 1978) have been suggested. A recent study also found increased blinking during visual fixation in schizophrenia (Helms and Godwin 1985).

In the largest series of studies on this topic conducted thus far, blink rate during casual speech was examined in a large group (n = 77) of inpatients who met both DSM-III criteria (American Psychiatric Association 1980) and Research Diagnostic Criteria (RDC; Spitzer et al. 1978) for schizophrenia and was compared with that in a group of normal control subjects. The patients were predominantly male and had been withdrawn from neuroleptic medications for a modal period of 6 weeks (Karson 1983). Measurement was by direct observation for 3-minute periods. In this state, the mean blink rate for the schizophrenic patients of 28 ± 15/minute exceeded that of 24 ± 15/minute recorded for the normal control subjects (p < 0.025, two-tailed t test). A rate equal to or exceeding 40/minute (± 1 SD) occurred in 22 percent (14/77) of the patients but in only 8 percent (7/96) of controls ($\chi^2 = 3.71, p < 0.03$, one-tailed test).

In a similar but smaller and separate group of inpatients with schizophrenia, blinks were measured during EEG recordings after a period of medication withdrawal of at least 4 weeks. During this recording, the patients were requested to fixate on a red dot projected by a laser and not to speak. The blink rate for the 23 patients under these conditions of visual fixation was 25 ± 19/minute, which quadrupled the 6 ± 4/minute blink rate of 20 normal controls recorded under the same conditions (p < 0.001) (Karson, unpublished manuscript, 1989). Fifteen patients (65%) had a rate greater than 15/minute, the maximum rate recorded for any controls.

Medication-Naive Subjects. To examine the extent to which the blink rate may be related to the effects of chronic neuroleptic treatment, a group of medication-naive adolescent inpatients with schizophrenia or schizophreniform psychoses was compared with a group of age- and sex-matched nonpsychotic inpatient controls (Karson et al. 1986). These subjects were not housed in special inpatient research units. Blinks were measured during casual conversation for 3-minute periods and were found to be increased in the never-medicated patients with schizophrenia or schizophreniform disorder (16 ± 9/minute vs. 10 ± 6/minute). The low rates in both groups reflect the youth of the subjects, because blink rates do not reach maximal levels until early adulthood (Zametkin et al. 1979).

Negative Studies. Not all investigators have been able to substantiate an increased blink rate in schizophrenia. For instance, Tecce et al. (1978) found that a subgroup of schizophrenic patients with a negative emotional (“hedonic”) state had an increased blink rate but that the rate was not increased in the total group of patients. These results are difficult to interpret because the methods are inadequately described, and the medication status of the patients is unclear. Mueser et al. (1984) conducted a smaller but also negative study. Finally, Ferrier et al. (1984) reported rates of only 12-15/minute in acute and chronic schizophrenic patients, many of whom had never received neuroleptics. The interpretation of this study is limited by the lack of a control group and the fact that blinks were counted from video recordings (a method of untested validity).

Other Psychiatric Disorders. Eyelid flutter (Schwarz and Stern 1968) and increased blinking (Mackintosh et al. 1983) have been reported in depres-
in increased blink rate, presuming that TD is a neuroleptic effect.

Perturbed Alpha Rhythm in Schizophrenia

The alpha rhythm becomes most prominent when the eyes are closed and, when expressed as the percentage of total EEG power, most often presents as a large peak between 10 and 12 Hz. A poorly developed alpha rhythm in schizophrenia was noted in the earliest EEG studies (Lemere 1956; Hughes et al. 1938; Jasper 1938). A poorly developed alpha rhythm also is seen in some normal subjects (Davis 1939-40). A quantitative EEG study was performed in the early 1950's in Canada, presumably before the introduction of neuroleptics on this continent (Kennard and Schwartzman 1957). Part of this study involved 15 subjects diagnosed as having schizophrenia contrasted with a like number of “psychopathic” and normal control subjects. Only 2 of the 15 patients with schizophrenia (diagnostic criteria not specified with the exception that the subjects had IQ scores > 80) versus 9 of 15 “psychopaths,” and 9 of 15 normal subjects, had a well-developed single alpha peak. Although neuroleptic treatment may not be an issue in these findings, other medications including sedative hypnotics and reserpine were used in the treatment of schizophrenic patients at this time and could have accounted for some of the diminished alpha activity in the patients with schizophrenia. In a later landmark study (Itil et al. 1972), EEGs were recorded in 100 patients after treatment with placebo medications for an average period of 8 weeks. These investigators reported a decrement in this alpha activity band from 10 to 13 Hz. This finding has been replicated in one recent study (Jutai et al. 1984). An overall decrease in alpha activity was also found using telemetry (Stevens and Livermore 1982). In this later study, 9 of the 18 schizophrenic subjects had never received antipsychotic medications and the remaining patients had been withdrawn from medication from 1 to 6 months. A recent study of only 10 schizophrenic subjects, withdrawn from medications for at least 1 month, reports a relative decrease in alpha activity predominantly in the right hemisphere (Merrin et al. 1986).

Finally, in a unique and difficult to reproduce study using a multilead array and “mapping” of EEG activity, a set of quadruplets with schizophrenia had much reduced alpha activity compared to 16 normal control subjects (Buchbaum et al. 1984).

Other recent EEG mapping studies with large multilead arrays, conducted on inpatients withdrawn from medication, have proved less supportive of alpha reduction in schizophrenia (Morhisa et al. 1983; Karson et al. 1987, 1988a). All these recent EEG-mapping studies suffer from having only a small number of subjects. In addition, these subjects were usually drawn from atypical samples of specially housed inpatient research volunteers who were “treatment refractory,” which also implies that each subject had received treatment with antipsychotic medications. The chronic effects of neuroleptic treatment on alpha activity, even in the face of prolonged medication withdrawal, are unknown. The earlier quantitative studies are also difficult to interpret because the diagnostic criteria for schizophrenia do not satisfy current

Dyskinesias. The effect of tardive dyskinesia (TD) on blink rate is confounded because patients with TD are often schizophrenic (Stevens 1978). No increase is evident in TD when the controls are patients with schizophrenia (Karson 1983). In noniatrogenic dyskinesias, the matter is also unsettled. Two studies of blinking in Tourette's syndrome are in conflict, despite agreement that increased frequency and force of blinks can be an early sign of the disorder (Bonnet 1982; Karson et al. 1985). A nonsignificant trend for an increased blink rate has been found in Huntington's chorea (Karson et al. 1984). The most compelling link between increased blink rates and dyskinesia is with the dyskinesia occurring during the course of dopamine agonist treatment of Parkinson's disease (Karson 1983). The problem with the interpretation of these data is that while the blink rate of parkinsonian patients with dopamine agonist-induced dyskinesia doubled that of other parkinsonian patients, it was not increased relative to normal control subjects. Moreover, data from the preneuroleptic era study and the medication-naive adolescent schizophrenic patients noted above weigh heavily against a primary role for TD

The effect of tardive dyskinesia (TD) on blink rate is confounded because patients with TD are often schizophrenic (Stevens 1978). No increase is evident in TD when the controls are patients with schizophrenia (Karson 1983). In noniatrogenic dyskinesias, the matter is also unsettled. Two studies of blinking in Tourette's syndrome are in conflict, despite agreement that increased frequency and force of blinks can be an early sign of the disorder (Bonnet 1982; Karson et al. 1985). A nonsignificant trend for an increased blink rate has been found in Huntington's chorea (Karson et al. 1984). The most compelling link between increased blink rates and dyskinesia is with the dyskinesia occurring during the course of dopamine agonist treatment of Parkinson's disease (Karson 1983). The problem with the interpretation of these data is that while the blink rate of parkinsonian patients with dopamine agonist-induced dyskinesia doubled that of other parkinsonian patients, it was not increased relative to normal control subjects. Moreover, data from the preneuroleptic era study and the medication-naive adolescent schizophrenic patients noted above weigh heavily against a primary role for TD

The effect of tardive dyskinesia (TD) on blink rate is confounded because patients with TD are often schizophrenic (Stevens 1978). No increase is evident in TD when the controls are patients with schizophrenia (Karson 1983). In noniatrogenic dyskinesias, the matter is also unsettled. Two studies of blinking in Tourette's syndrome are in conflict, despite agreement that increased frequency and force of blinks can be an early sign of the disorder (Bonnet 1982; Karson et al. 1985). A nonsignificant trend for an increased blink rate has been found in Huntington's chorea (Karson et al. 1984). The most compelling link between increased blink rates and dyskinesia is with the dyskinesia occurring during the course of dopamine agonist treatment of Parkinson's disease (Karson 1983). The problem with the interpretation of these data is that while the blink rate of parkinsonian patients with dopamine agonist-induced dyskinesia doubled that of other parkinsonian patients, it was not increased relative to normal control subjects. Moreover, data from the preneuroleptic era study and the medication-naive adolescent schizophrenic patients noted above weigh heavily against a primary role for TD

The effect of tardive dyskinesia (TD) on blink rate is confounded because patients with TD are often schizophrenic (Stevens 1978). No increase is evident in TD when the controls are patients with schizophrenia (Karson 1983). In noniatrogenic dyskinesias, the matter is also unsettled. Two studies of blinking in Tourette's syndrome are in conflict, despite agreement that increased frequency and force of blinks can be an early sign of the disorder (Bonnet 1982; Karson et al. 1985). A nonsignificant trend for an increased blink rate has been found in Huntington's chorea (Karson et al. 1984). The most compelling link between increased blink rates and dyskinesia is with the dyskinesia occurring during the course of dopamine agonist treatment of Parkinson's disease (Karson 1983). The problem with the interpretation of these data is that while the blink rate of parkinsonian patients with dopamine agonist-induced dyskinesia doubled that of other parkinsonian patients, it was not increased relative to normal control subjects. Moreover, data from the preneuroleptic era study and the medication-naive adolescent schizophrenic patients noted above weigh heavily against a primary role for TD
compared with 5.0 ± 2.4 in other low peak alpha frequency (9.8 ± 1.9 ventricle-brain ratio, is nearly doubled in schizophrenic patients, which might be affected by disease processes that could then produce different alpha disturbances, it is possible to rationalize grouping all patients with irregular alpha under a single heading such as "perturbed alpha." When this unitary scheme is used, an overwhelming proportion of patients have perturbed alpha (Karson et al. 1988a, 1988b).

The decrement in "fast" alpha activity noted above (Itil et al. 1972) is consistent with another group of studies in which a reduced frequency of the alpha peak in schizophrenia has been observed (Rodin et al. 1966; Giannitrapani and Kayton 1974; Small et al. 1984; Karson et al. 1988a, 1988b). While the older studies tended to use 10 Hz as the dividing line between low and normal alpha frequency, a recent study of 16 normal subjects suggests that a slightly higher frequency of 10.2 Hz may be suitable as well (Coppena and Chassy 1986). Nevertheless, the proportion of schizophrenic subjects with either reduced alpha activity or a reduction in the peak alpha frequency may not be greater than seen in control subjects in some samples. If one presumes, however, that the alpha rhythm is generated by a system of neural structures, any of which might be affected by disease processes, it is possible to rationalize grouping all patients with irregular alpha under a single heading such as "perturbed alpha." When this unitary scheme is used, an overwhelming proportion of patients have perturbed alpha (Karson et al. 1988a, 1988b).

Cerebral Ventricular Size, Alpha Rhythm, and Blink Rate. Lateral ventricular size, as estimated by the ventricle-brain ratio, is nearly doubled in schizophrenic patients with low peak alpha frequency (9.8 ± 1.9 compared with 5.0 ± 2.4 in other schizophrenic patients, p < 0.001, Karson et al. 1988a, 1988b). In addition, neuroleptics reduce blinking in schizophrenic patients except those with lateral ventricular enlargement (Kleinman et al. 1984). Because much of the ventricular border is determined by the thalamus and the optic radiations, which connect the LGB with the occipital cortex, low-alpha frequency and the failure of neuroleptics to reduce blinks in patients with large ventricles may result from specific involvement of the thalamus and optic radiations. A recent magnetic resonance imaging (MRI) study demonstrated that the greatest degree of ventricular enlargement in schizophrenic patients overlies the anterior thalamus and superior colliculus, and therefore is consistent with this speculation (Kelsoe et al. 1988).

Other Neurophysiological Studies Related to the BANC in Schizophrenia

Smooth Pursuit Eye Movements. Impairments in smooth pursuit eye movements may be a hallmark sign of schizophrenia. Many of the BANC structures are also critical to the generation of eye movements, including saccades. For instance, the triphasic discharge in the PPRF and the activation of the LGB that occur with a blink also occur with saccades (Cohen and Feldman 1968). The intimate relationship between blinks and saccades mediated by the cerebellum and pons is further elucidated by Zee et al. (1983). Neither of two subjects, one with a presumed lesion of the pontine tegmentum and another with cerebellar degeneration, was able to initiate eye movements without first blinking.

Holzman (1987) has argued that impairment in pursuit movements is related to frontal lobe dysfunction rather than to dysfunction of the BANC. A principal aspect of this argument centers around the finding of normal oculocerebellar reflexes and certain compensatory eye movements in schizophrenia (cf. Holzman 1987). The preservation of certain upper brainstem functions, however, does not necessarily imply that the entire upper brainstem is intact. Moreover, the tests used by Holzman's group are difficult to perform and interpret in relatively healthy subjects.

Differences between blinks and other eye movements are obvious, including mediating musculature and cranial innervation. In addition, antipsychotic drugs reduce blink rate (Karson 1983; Mueser et al. 1984) but do not appear to affect visual pursuit (Holzman 1987).

New Evidence About Evoked Potentials. Lindstrom et al. (1987) studied the early components of auditory evoked potentials (< 10 ms), which reflect brainstem function, in 20 schizophrenic subjects, 8 of whom had never received antipsychotic medications. Abnormalities occurred in 10 patients, including 5 of the drug-naive subjects. The authors relate these findings to the presence of auditory hallucinations.

Relevant PET Studies. Neuro-pathological change in the BANC can produce "hypofrontality" similar to that seen in schizophrenia. In a recent PET study, patients with progressive supranuclear palsy (PSP) were found to have reduced cortical metabolism, particularly in the superior frontal lobes, despite the
known absence of cortical pathology in this disorder (Foster et al. 1988).

Neuropathology of the BANC. Classical neuropathological studies of the BANC, while controversial, have at times suggested that there is gliosis in schizophrenia in such structures as the periaqueductal gray, pons, and vermis of the cerebellum (Nieto and Escobar 1972; Fisman 1975; Stevens 1982). Recent studies using newer more objective methods to determine gliosis have been applied to the BANC only with regard to the thalamus, and these are negative (Roberts et al. 1986; Pakkenburg and Gundersen 1988). In the latter study, Pakkenburg and Gundersen (1988) found a reduction in neuronal cell number in the mediodorsal nucleus of the thalamus. This indicates that structural changes in the BANC in schizophrenia could be due to processes other than neurodegeneration. With regard to the thalamus, such a possibility is further suggested by the finding of increased dopamine-to-norepinephrine ratios in schizophrenic thalami (Oke and Adams 1987).

Interest in another BANC structure, the cerebellar vermis, in schizophrenia has waned because of a negative report regarding Purkinje cell density in the vermis (Lohr and Jeste 1986) and a single negative MRI study of living patients (Mathew and Partain 1985). Studies of brain cell density, however, raise methodological issues, because these studies fail to account for the entire volume of the brain structure being studied (Braendgaard and Gundersen 1986).

Summary

The BANC. Blinks appear to be generated in the PPRF and related signals transmitted to the LGB. Structures near and within the tectum and the substantia nigra appear to facilitate blinking, whereas those in the cerebellum inhibit blinking. That the aforementioned structures may influence the occipital cortex is evidenced by reduced activity in the visual cortex during blinks and by alpha frequency induction preceding blinks. A bidirectional circuit with occipital cortex inhibiting other BANC structures is suggested by a reduction in blinking during visual fixation.

Blink Rate. The weight of evidence suggests that blink rates are increased in schizophrenia. This increase may be highly state dependent. For instance, blink rates were only clearly increased in 20 percent of patients (> 40/minute) during casual conversation. But blink rates were increased beyond the range of normal in 65 percent of volunteers with schizophrenia when rates were measured during visual fixation. Possible explanations for this latter finding are (1) the failure of schizophrenic patients to fixate and invoke associated blink-reducing mechanisms or (2) the inability to suppress blinking in schizophrenia. The latter explanation is supported by the inability of patients to suppress blinks voluntarily. Bracha and Karson (unpublished data) found that the voluntary blink suppression time in 15 schizophrenic inpatients who had been withdrawn from medications for 4 weeks was only 5 seconds compared with 20 seconds for 52 normal subjects ($p < 0.003$). The latter explanation is also supported by the inability of schizophrenic patients to habituate their reflex blinking (Geyer and Braff 1982). Nevertheless, the increase of the blink rate outside the normal range in two-thirds of schizophrenic patients during visual fixation suggests that disruptions of the BANC may be common in schizophrenia. These disruptions are probably not an effect of treatment with antipsychotic medications, since an increased blink rate was also found in a typical group of medication-naive adolescents with schizophrenia (Karson et al. 1986). Results from a preneuroleptic-era study (Ostow and Ostow 1945), in which blink rates were increased in psychotic patients, also suggest that the increase in blinking in schizophrenia is not due to previous drug treatment. The major gap in knowledge about blinking rates in psychiatric disorders appears to be the paucity of information about blinking in psychotic disorders other than schizophrenia.

Perturbed Alpha Rhythm. The amount of alpha activity may be lower in schizophrenia and the frequency of the alpha peak is likely lower as well. Combining these differences along with other differences in alpha that occur in schizophrenia under the rubric of "perturbed alpha" seems to be a potentially useful categorization because a minority of patients with schizophrenia exhibit unperturbed alpha. As with blinking, however, the occurrence of poorly developed alpha and low alpha frequency has not been well studied for other psychiatric disorders.
Neuropathological Implications.

Increased blink rates in schizophrenia could be explained by an enhancement of the PPRF generator or nigrotectal facilitation or a decrement in the inhibitory function of the cerebellum and occipital cortex. A more complicated hypothesis might invoke decreased function of structures providing inhibitory feedback to PPRF or nigrotectal structures. The relevance of the direct interconnections between these brain structures (i.e., the superior colliculus, a putative blink facilitator, has neural connections with the substantia nigra, another putative facilitator [Graybiel 1978]) and more information concerning the anatomic control of alpha rhythm remain to be determined. Nevertheless, the plausibility of the BANC and the high percentage of schizophrenic patients with increased blink rates (during visual fixation) and perturbed alpha rhythms suggest that BANC structures may be important in the pathophysiology of schizophrenia.

References


Freed, W.J.; Karson, C.N.; Kleinman, J.E.; and Wyatt, R.J.


Volkman, F.C.; Riggs, L.A.; and Moore, R.K. A comparison of saccades and blinks in suppression of


The Authors
Craig N. Karson, M.D., is Professor of Psychiatry and Behavioral Sciences, University of Arkansas Medical Sciences, and Chief, Psychiatry Service, Veterans Administration Medical Center, Little Rock, AR. Roscoe A. Dykman, Ph.D., is Professor, and Stephen R. Paige, Ph.D., is Assistant Professor of Psychiatry and Behavioral Sciences, University of Arkansas Medical Sciences, Little Rock, AR.

---

**Announcement**

An international conference entitled *Schizophrenia 1990: Poised for Discovery* will be held July 15-17, 1990, at the Hyatt Regency Hotel, Vancouver, British Columbia, Canada. The conference will bring many professional people together to present their work, review progress, reappraise strategies, and to formulate new directions in the management of schizophrenia. Clinicians, researchers, and students will meet and work together in research, treatment, and rehabilitation in the fields of medicine, nursing, psychology, social work, and the basic sciences. This conference will also include special sessions aimed at providing information to the public about up-to-date research findings and their implications for treatment. These sessions will be of particular interest to the families, friends, and support groups for individuals who are coping with the challenges of life with schizophrenia.

For further information about the conference, please contact:

**International Conference Secretariat**
Schizophrenia 1990
c/o Venue West Ltd.
#801 - 750 Jervis St.
Vancouver, B.C.
Canada V6E 2A9