

Akinesia, Tardive Dysmetria, and Frontal Lobe Disorder in Schizophrenia

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Abstract

Two iatrogenic effects of antipsychotic medications other than tardive dyskinesia have been recently described in schizophrenic populations: akinesia and tardive dysmetria. These effects involve activational, cognitive, and affective rather than motor changes, and closely resemble two most common prefrontal syndromes: dorsolateral and fronto-orbital/mediobasal. It is possible that the widely reported "frontal lobe dysfunction" in some chronic schizophrenic patients at least in part reflects iatrogenic changes in the mesolimbic/mesocortical dopamine system, which projects extensively into prefrontal areas. The degree of iatrogenic versus genuine contribution to the frontal lobe dysfunction in schizophrenia needs to be ascertained further, and the heterogeneity of known frontal lobe syndromes must be taken into account in describing schizophrenic populations. The mechanisms of noniatrogenic contributions to the frontal lobe dysfunction in schizophrenia may reflect a variety of anatomical sources and require further examination.

Tardive dyskinesia has long been recognized as an iatrogenic effect of antipsychotic drugs. Recently, two types of iatrogenic neuroleptic-induced effects of an activational, cognitive, and affective rather than motor nature have been described in schizophrenic patients.

The first set of reports (Rifkin, Quitkin, and Klein 1975; Rifkin et al. 1978) introduced the phenomenon of "akinesia" and described it as "a behavioral state of diminished spontaneity characterized by few gestures, unspontaneous speech, and, particularly, apathy and difficulty

with initiating usual activities" (Rifkin, Quitkin, and Klein 1975, p. 672). "Less extreme instances may be seen only as diminished social activity or interest in work" (Rifkin et al. 1978, p. 488). The authors indicate that manifestations of akinesia are different from those of depression.

The second report (Wilson et al. 1983) introduced the phenomenon of "tardive dysmetria." The authors point out that the phenomenon involves "changes in affect, activational level, and interpersonal interaction" and resembles hypomania. They describe it as follows:

The patients tended to be loquacious and to speak in loud voices. They showed thought disconnection and were generally circumstantial and aimless in conversation. Disassociated, inappropriate statements were common. The prevailing mood was generally one of euphoria, but unheralded, inexplicable, explosive changes in affect occurred as good humor changed rapidly to explosive hostility or sullen petulance. Social withdrawal and autistic preoccupation were punctuated by episodes of overreactive behavior when the subject talked loudly, often close to the observer's face, and was quite intrusive and invasive of the privacy of others. [p. 188]

Although the two described phenomena are quite different, in fact opposite in certain respects (the first characterized by predominant aspon-taneity, the second by predominant behavioral disinhibition), and their clinical descriptions are scant, they are conspicuously similar to the two most commonly specified syndromes

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that follow focal damage to the prefrontal areas of the brain and are frequently observed in neurological and neurosurgical clinics.

The clinical and neuroanatomical heterogeneity of "frontal" syndromes has been pointed out by numerous authors (Kleist 1934; Blumer and Benson 1975; Hecaen and Albert 1978; Damasio 1979; Luria 1980). Hecaen and Albert (1978) provide these descriptions of the two main ones:

Lack of initiative or spontaneity is a characteristic feature of frontal lobe pathology. This is linked to a general diminution of motor activity. The patient no longer voluntarily carries out the necessary daily activities of life, such as getting out of bed in the morning, washing or dressing himself, feeding himself, or even urinating or defecating in the toilet. . . . The actual ability to do the various activities of daily living is not impaired—the patient is not paralyzed, apraxic, or confused. When he is vigorously urged to do something, he can do it. What is impaired is the ability to initiate spontaneously a desired or an automatic motor task.

This rupture between the patient and the external world, this diminution of activity in manipulating real objects, and this reduction of interpersonal exchange are what appear to be a loss of interest on the part of the patient. Whether there is a true loss of interest or an apparent loss of interest due to an impairment of spontaneous initiation of activity, the effect on the examination remains the same. [p. 368]

Frequently one sees an apparent heightening of affective tone, with euphoria and lack of concern for the present or the future. This behavioral change does not, however, necessarily reflect a true alteration of mood. Such patients, in the midst of apparent euphoria, often state that they are not at all happy. A puerile or silly attitude may be maintained, often with an

inappropriate use of pretentious language. Erotic behavior, sexual exhibitionism, or lewd remarks are not rare. Euphoric excitation may take on an atypical hypomanic aspect, "moria": the patient overexcited, manifesting erotic behavior, bothers the examiner with inappropriate jokes and caustic or facetious remarks (*Witzelsucht*).

The diverse aspects of excitation and euphoria are rarely permanent. In general, they appear episodically, superimposed on an underlying background of abulia and apathy. The euphoric periods may alternate with periods of apparent depression. True depression, however, is rarely seen; rather, a picture of asthenia and akinesia imitates a true depression. A disorder of activity, rather than of affectivity, is the hallmark.

Loss of impulse control, manifested by outbursts of irritability, is common. These outbursts may be the first behavioral change noted; often they reach such a degree of violence that they force a family to institutionalize the patient. [pp. 367-368]

Various authors (Kleist 1934; Blumer and Benson 1975; Damasio 1979; Luria 1980) attempted to specify critical lesion loci within the frontal lobes differentially responsible for the two aforescribed syndromes.

According to these authors, the first one, consisting of affective flatness and reduced motor and cognitive activity, follows lesions of the dorsolateral prefrontal convexity; the second one, associated with behavioral disinhibition, loosened social controls and judgment, and wide oscillations of affect with predominant euphoria, follows lesions of the fronto-orbital/medio-basal surfaces.

Blumer and Benson (1975) suggest:

The two types of personality changes occurring after frontal

lobe lesions may be described as: (1) toward apathy and indifference ("pseudodepressed"), and (2) toward puerility and euphoria ("pseudopsychopathic"). . . . Patients who suffer the "pseudodepressed" type of frontal lobe personality alteration appear to have lost all initiative. They respond in an automaton-like fashion . . . slowness, indifference and apathy . . . characterize the personality alteration produced by prefrontal convexity lesions, the condition we term "pseudodepressed."

. . . The "pseudopsychopathic" type of frontal lobe personality is best characterized by the lack of adult tact and restraints. Such patients may be coarse, irritable, facetious, hyperkinetic or promiscuous; they often lack social graces and may, on impulse, commit antisocial acts. Paranoid or grandiose thinking may be present. While they may flare with anger, they do not bear a grudge. Outwardly, at least, their behavior resembles that of the sociopathic personality . . . the "pseudopsychopathic" personality traits apparently follow injury to the orbital frontal lobe or pathways traversing this region. [pp. 157-158]

Blumer and Benson (1975) attribute the early descriptions of the two syndromes to Kleist (1934):

[Kleist] confirmed Welt's thesis of the significance of lesions to the orbital brain, and related changes toward immoral, unfaithful, deceitful, thievish, and defiant behavior to orbital lesions. Kleist also noted puerile and facetious behavior in patients with orbital lesions. Euphoria was observed as a frequent early mental change in patients with frontal-orbital injuries; the euphoria was usually transient and at times changed to a dysphoric mood. Kleist presumed that the unity of personality and man's self-determination were related to the orbital brain and its connections. In contrast, lesions of the upper portions of the frontal lobes (convexity) were associated with lack of psychic and motor initiative. Lack of thought

formation (impoverished and stereotyped modes of thinking) characterized these patients. [p. 153]

Thus, there is a high degree of consensus in the neurological and neuropsychological literature regarding the existence, clinical features, and underlying neuroanatomy of two major frontal syndromes. The first (dorsolateral convexital) bears a remarkable clinical similarity to the iatrogenic akinesia described by Rifkin and his associates (Rifkin, Quitkin, and Klein 1975; Rifkin et al. 1978), and the second one (orbitofrontal) is as remarkably similar in its clinical features to the tardive dysmetria described by Wilson et al. (1983).

This similarity between known neurological syndromes and the iatrogenic effects of antipsychotic medications can be approached in light of the pathway distribution of the mesolimbic/mesocortical dopamine system.

Whereas the nigrostriatal dopamine system projects predominantly into the basal ganglia, the mesolimbic/mesocortical dopamine system projects substantially into the basal forebrain and the prefrontal cortex. Both orbitofrontal/mesofrontal and dorsolateral cortices receive dopamine projections, the former being more abundant (Bannon and Roth 1983; Brown, Crane, and Goldman 1979). In these prefrontal regions higher concentrations of dopamine are found than in any other cortical area, both in absolute terms and relative to other transmitter systems (Lindvall et al. 1974; MacBrown and Goldman 1977; Brown, Crane, and Goldman 1979).

Furthermore, it has been demonstrated that destruction of the ventral tegmental area, which is the point of origin of the mesolimbic/mesocortical dopamine system, can

effectively "simulate" a syndrome typically associated with a structural lesion in prefrontal areas, both in animals (Tassin et al. 1978; Simon, Scatten, and LeMoal 1980; Oades 1982) and in humans (Goldberg et al. 1982). Behavioral effects of interference with the mesolimbic/mesocortical dopamine projections, in other words, closely resemble the effects of lesions in the target areas of these projections. Brozoski et al. (1979) demonstrated that the behavioral effects of dopamine depletion in a circumscribed area of dorsolateral prefrontal convexity were quite similar to those of surgical ablation of the same area in rhesus monkeys.

It would therefore be almost predictable on *a priori* grounds that if neuroleptic-induced effects can occur in the mesolimbic/mesocortical system, these effects would have essential features of a "frontal syndrome."

The possibility that iatrogenic effects of neuroleptics can develop outside of the traditionally considered nigrostriatal system, notably in the mesolimbic/mesocortical system, has been raised by several investigators (Wilson et al. 1983). White and Wang (1983) demonstrated that prolonged treatment with antipsychotic drugs decreases the number of spontaneously active neurons both in substantia nigra and in the ventral tegmental area. The latter, of course, is the point of origin of the mesolimbic/mesocortical dopamine system with abundant projections into the prefrontal cortex and the basal forebrain (Simon et al. 1976; Cooper, Bloom, and Roth 1978; Simon et al. 1979). White and Wang (1983) further demonstrated that the decline of spontaneous activity of mesolimbic/mesocortical A10 DA neurons occurs faster and to a

greater extent than that of nigrostriatal A9 DA neurons, and identified a class of "atypical" neuroleptics that selectively affect the A10 dopamine neurons but not A9 dopamine neurons. In an extensive review, Joyce (1983) also argues that the atypical antipsychotics have both a greater affinity to and activity in the mesolimbic/mesocortical system than in the nigrostriatal system. Thus, at least theoretically, cases may exist in which iatrogenic "mesolimbic/mesocortical" effects of antipsychotic drugs are more pronounced than or totally unaccompanied by "nigrostriatal" effects. Indeed, whereas Wilson et al. (1983) observed a positive relationship between the severity of tardive dyskinesia and the features of tardive dysmetria, in some of the patients described by Rifkin and his associates (Rifkin, Quitkin, and Klein 1975; Rifkin et al. 1978) "akinesia" developed without tardive dyskinesia.

The observations by Rifkin et al. (1978) and Wilson et al. (1983) are clearly preliminary and the reported phenomena require further study. Whereas the existence of akinesia has been demonstrated in several double-blind studies, the empirical basis for accepting the existence of tardive dysmetria is less compelling, since the original study reporting it was conducted under open, nonblind conditions. Even if one assumes the existence of these syndromes, it is not clear whether they can be accounted for solely by exposure to neuroleptics or, as Mukherjee (1984) proposed with respect to tardive dysmetria, exposure to neuroleptics has to be combined with certain preexisting cerebral dysfunctions. Furthermore, available clinical descriptions of akinesia and tardive dysmetria are too scant to enable their definitive comparison with

known neurological syndromes. However, should their existence be confirmed, and in particular should their similarity to the known frontal syndromes be born out, these phenomena will be extremely important for two reasons.

The first reason is clinical. To the extent that there are iatrogenic effects of antipsychotic drugs other than extrapyramidal in nature, they have to be recognized and taken into account. Because the effects observed by Rifkin et al. and Wilson et al. involve higher order activational, cognitive, and affective changes rather than motor changes, their identification may be more difficult and more confounded by the features of the original disease than is the identification of tardive dyskinesia.

The second reason is more theoretical in nature and is related to our understanding of the schizophrenic disorder. Recently, numerous attempts have been made to describe schizophrenic populations both in terms of brain region-specific deficits and the neuroanatomy of involvement (Seidman 1983). Among various formulations that emerged from these attempts, the claims of the "frontal involvement" in schizophrenia are probably most prominent. Findings that led to these claims are based on various techniques: (1) neuropsychological studies that reveal pronounced cognitive deficit consistent with frontal damage (Flor-Henry 1976; Flor-Henry and Yeudall 1979; Kolb and Whishaw 1983); (2) metabolic studies using positron emission tomography (PET) (Farkas et al. 1980; Buchsbaum et al. 1982); (3) blood flow studies (Ingvar 1976; Mathew et al. 1981); and (4) electrophysiological studies (Heath 1977; Stevens 1977; Morihisa, Duffy, and Wyatt 1983) which reveal frontal dysfunction.

The remarkable similarity between

the neuroleptic-induced effects described by Rifkin et al. and Wilson et al., on the one hand, and the well-known frontal syndromes, on the other hand, directly raises the question as to the origins of such "frontal features" in schizophrenic populations. Although in the study by Rifkin, Quitkin, and Klein (1975), signs of akinesia were shortlived and disappeared rapidly following the changes of of medicational schedule, this in itself is no guarantee that other more subtle iatrogenic effects (other than tardive dyskinesia) are as easily reversible. In most if not all studies, samples in which frontal pathology was revealed consisted of chronic, institutionalized patients with long histories of treatment with antipsychotic medications. In these populations it is extremely difficult to distinguish between features of the underlying disease and persistent iatrogenic features, providing that such exist; and even taking the patients off medications for a limited period of time before the study may not be enough to disentangle the two factors. Just as a persistent nigrostriatal iatrogenic syndrome in the form of tardive dyskinesia is possible (Jeste et al. 1979; Smith and Baldesarini 1980; Jeste and Wyatt 1982; Kane and Smith 1982), so is a persistent mesolimbic/mesocortical iatrogenic syndrome which will predictably include frontal signs.

Among possible manifestations of frontal pathology in schizophrenia, those related to function can be accounted for either by genuine or by iatrogenic mechanisms. On the other hand, macroscopic structural changes in the frontal lobes could be less readily accounted for by iatrogenic mechanisms. It is of interest in this respect that whereas various assessments of function (neuropsychological, cognitive, electrophysiological, PET scan, blood flow)

frequently reveal patterns of selective "hypofrontality" in schizophrenic patients, assessments of structure, e.g., pneumoencephalography, computed tomography (CT), by and large do not. Although many studies revealed diffuse changes (Johnstone et al. 1978; Weinberger et al. 1979a, 1979b), these changes were not characterized by frontal preponderance. Among numerous CT studies of schizophrenic patients, only one revealed selective prefrontal hypodensity to the best of my knowledge (Golden et al. 1981).

Several post-mortem studies revealed microscopic changes in prefrontal cortex of schizophrenics (Tatetsu 1964; Miyakawa et al. 1972). These changes, however, are no more extensive than those in various other structures, which include hippocampi, nucleus accumbens, vast areas of diencephalon and brainstem, cerebellar vermis, and nonspecific cortical changes (Weinberger, Wagner, and Wyatt 1983).

One has to conclude that existing neuropathological studies do not provide sufficient basis for identifying prefrontal areas as the outstanding locus of deficit in schizophrenic patients.

Wilson et al. (1983) discuss the issue of the changing phenomenology of the schizophrenic disorder over the past several decades and the possible role of the advent and use of antipsychotic drugs in this change. Unfortunately, most neuropsychological, cognitive, and regional neuroanatomical studies of schizophrenia are relatively recent. We therefore cannot reliably judge whether the "frontal" signs in schizophrenia of the kind reported in these studies predated the advent of neuroleptics or whether they emerged together with the latter. Early descriptions of avolition and apathy in schizophrenic patients

(Kraepelin 1919; Bleuler 1950), in fact, resemble certain features of the dorsolateral frontal syndrome. Several colleagues with longer careers than mine in the field pointed out that, in retrospect, they are quite certain that they had observed frontal phenomenology in schizophrenic patients before the advent of neuroleptics. It is likely that the mechanisms of frontal phenomenology in schizophrenic patients are complex and may include both genuine and iatrogenic components.

Two types of possible genuine mechanisms of frontal dysfunction in schizophrenia can be hypothesized: those which entail an actual lesion in prefrontal areas (macroscopic structural, microscopic, or biochemical), and those, paradoxically, which do not.

The extent to which abnormalities of the dopamine system may play a significant role in schizophrenia (Horn and Snyder 1971; Stevens 1973; Snyder 1976) would in itself imply an intimate frontal involvement due to the distribution of the mesolimbic/mesocortical dopamine projections. This is an example of the first type of mechanism, and the search for other such mechanisms of direct frontal lesions in schizophrenia will undoubtedly continue.

The second type of mechanisms, while genuine in the sense of not being iatrogenic, may act more indirectly. This is due to the unique position of prefrontal cortex at the top of neurocognitive hierarchy (Teuber 1964; Nauta 1971; Luria 1980). More than any other brain structure, prefrontal cortex serves the role of interrelating various morphologically and functionally diverse systems which include posterior cortices, the limbic lobe, striatum, amygdala, hypothalamus, dorso-medial thalamus, and mesencephalic

reticular nuclei, thus serving as a unique point of convergence of various influences within the brain (Nauta 1971; Carpenter 1976). Because of this complexity, Nauta (1971) suggested that inferring function from anatomy is more difficult with respect to the frontal lobe than other systems. It may be equally difficult to infer the anatomy of a deficit from its functional manifestations (metabolic, electrophysiological, or cognitive) when the frontal lobe is involved.

Frontal lobes are associated with the highest, phylogenetically and ontogenetically youngest levels of cognitive control (Luria 1980). On the other hand, a diffuse central nervous system (CNS) disease is likely to affect such levels first and foremost. It is possible, therefore, that a rather global cognitive dysfunction implicit in schizophrenia is likely to mimic a frontal lobe dysfunction more readily than any other area-specific kind of cognitive dysfunction. It may not be necessary, in other words, to postulate a selective frontal lobe disease to account for a behavioral pattern consistent with a frontal lobe dysfunction. Alternatively, given the uniquely rich system of projections connecting the frontal lobes with a wide range of diverse brain systems, the frontal lobes, more than any other system, are likely to be the point of summation of dysfunctions generated by many morphologically or chemically diverse systems due to a multifocal or diffuse CNS disease, or to reflect anatomically specific but remote CNS events or disturbances. The latter possibility is illustrated by the previously mentioned observations that lesions of the ventral mesencephalic tegmentum can simulate prefrontal damage (Simon, Scatten, and LeMoal 1980; Goldberg et al. 1982; Oades 1982).

This implies that frontal lobe disease can be "simulated" by a broad range of conditions at least in some respects. The above concerns apply to most brain structures, but particularly to prefrontal cortex.

According to Nauta (1971), a unique feature of the frontal cortex is in its "reciprocal relationship with . . . (1) parietal and temporal regions of cerebral cortex involved in the processing of visual, auditory and somatic sensory information, and (2) the telencephalic limbic system and its subcortical correspondents . . ." (p. 181). This makes the frontal cortex a "mediator of information exchange between the cerebral cortex and the limbic system" (p. 184) by virtue of its being "one and perhaps the only realm of neocortex where neural pathways representing the internal milieu converge with conduction systems representing the external environment as reported by *all* exteroceptive modalities" (pp. 182-183). Prefrontal cortex appears, therefore, to be central to that which can be termed "coordination between sensory/perceptual and homeostatic/motivational domains." The deficit of such interface being the central feature of schizophrenia is precisely what emerges as the common denominator across numerous formulations, both early and recent, attempting to capture the core of schizophrenic disorder. One would therefore expect that manifestations of schizophrenia will resemble those of frontal lobe damage in certain important respects. It may be most parsimonious to conclude that this is so precisely because frontal damage is present and plays a role in the mechanisms of schizophrenic disorder. Yet a parsimonious explanation is not always the correct one, and there may be more than one cause or chain of events leading to

destabilization of "sensory and perceptual vs. homeostatic and motivational" alignment.

This implies that schizophrenia is particularly suited to present itself as a frontal lobe disease, regardless of its actual mechanisms. Frontal lobe damage may in fact be central to schizophrenic disorder, but it does not *have to be* in order for the similarity between the two conditions to exist.

The interpretation of the findings of frontal dysfunction in schizophrenia is complicated by the fact that such findings are usually reported in global terms, without much regard for the heterogeneity of the frontal syndromes. As was pointed out before, the effects of dorsolateral and fronto-orbital/mediobasal prefrontal damage are quite different and in certain respects even opposite, in spite of the morphological proximity of the two areas. The mechanisms of these locus-dependent differences are not well understood at this time. Nevertheless, the descriptions of frontal dysfunction in schizophrenia will certainly benefit from being more specific in view of this heterogeneity. This may contribute to further subtyping of schizophrenia, providing that the descriptions reflect genuine rather than iatrogenic phenomena.

That the two frontal-like iatrogenic effects of neuroleptics described, respectively, by Rifkin et al. and Wilson et al. each resemble one of the two prefrontal syndromes is intriguing. The differences between the two aspects of the frontal lobes have been reasonably well studied in terms of their cytoarchitectonic characteristics, afferent and efferent projections, and behavioral pathology. These studies clearly indicate that the functional roles of the dorsolateral and fronto-

orbital/mediobasal aspects of the frontal lobes in the overall cerebral organization are quite different. In fact, it has been proposed that these two areas play reciprocal roles in the cortico-limbic-reticular mechanisms of arousal: activating and facilitatory, mediated by the dorsolateral frontal/amygdaloid/lateral hypothalamic/reticular system; and inhibitory, mediated by the fronto-orbital/mediobasal/amygdaloid/medial hypothalamic/reticular system (Pribram and McGuinness 1975).

Considerably less is known about possible differences in the reliance of the two frontal regions on various biochemical (neurotransmitter and neuropeptide) systems. Two recent findings may be of particular relevance to these issues. The first finding is related to the reciprocity between the cortical (mesocortical) and subcortical (mesolimbic and nigrostriatal) dopamine systems, such that interference with the former enhances the latter (Pycocock, Kerwin, and Carter 1980; Bannon and Roth 1983). The second finding is that of the absence of terminal autoreceptors in mesocortical (but not mesolimbic or nigrostriatal) dopamine neurons, which leads to different patterns of response in the corresponding systems following acute and chronic treatment with neuroleptics (Bannon et al. 1982; Bannon and Roth 1983). It is possible that the aforementioned dopamine systems (mesocortical, mesolimbic, and nigrostriatal) have different degrees of contribution to the functions associated with various prefrontal areas. It is not inconceivable, for instance, that the mesocortical dopamine system is relatively more intimately involved in functions associated with the dorsolateral areas whereas the mesolimbic dopamine system is more involved in those associated with the orbitofrontal areas. If this is so, then

by having differential impacts on the dopamine autoreceptors, different medicational histories may have different effects on the balance between the cognitive controls associated with the two prefrontal areas. Careful examination of clinical and medicational histories leading, alternatively, to akinesia or tardive dysmetria may, in addition to helping understand the mechanisms and typology of frontal manifestations in schizophrenic populations, shed further light on the biochemical basis of difference between main frontal syndromes as they are seen following focal brain lesions, and on the biochemical basis of the complementary roles of the two aspects of prefrontal areas in normal organization.

The observations by Rifkin et al. and Wilson et al. certainly offer an additional challenge to our understanding of the widely reported frontal deficits in schizophrenia. It is not the purpose of this comment to imply that these deficits are solely iatrogenic in nature and have nothing to do with the underlying schizophrenic disorder itself. There is no basis for such an absolute conclusion, nor do I believe that this is the case. There is sufficient basis, however, for proposing that iatrogenic effects of neuroleptics may contribute to the frontal symptom picture in chronic schizophrenic patients and that the extent and nature of this contribution is not known at this time. Nor is it implied that possible genuine frontal deficits are necessarily caused by mechanisms in which actual frontal lobe damage plays no part. That would indeed be a twisted statement to make, and equally without foundation. There is sufficient basis, however, for proposing that anatomical interpretation of frontal dysfunction requires particular caution, greater

than that of most other dysfunctions which are presumed to have focal significance.

Intriguing as the claims of frontal lobe dysfunction being a major component of the schizophrenic disorder are, the evaluation of their validity and meaning will have to be critically dependent on further studies of frontal iatrogenic effects of neuroleptics. When this is done and genuine, noniatrogenic manifestations are identified beyond reasonable doubt, we will still be left perplexed, since not every frontal dysfunction is necessarily caused by a frontal lesion, structural or chemical; but then again, it may be. It is hoped that in the process we will find ourselves a step closer both to the understanding of schizophrenic disorder and to the unraveling of what Teuber (1964) termed "the riddle of the frontal lobe."

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Expressed Emotion in Families

Expressed Emotion in Families: Its Significance for Mental Illness, authored by Julian Leff and Christine Vaughn, has been recently published by The Guilford Press (200 Park Avenue South, New York, NY 10003). The discovery that discharged schizophrenic patients who returned home to parents or spouses often fared worse than those living alone led researchers to look for conditions within the family that might influence the schizophrenic patient's condition. At the vanguard of this endeavor was George Brown, whose recognition of the debilitating effect of high levels of expressed emotion—such as hostility, criticism,

and overinvolvement—has stimulated important insights as well as considerable controversy. In this volume, Leff and Vaughn, together with two other prominent investigators, address some of the confusions and misconceptions that have arisen regarding the measures of expressed emotion and the techniques for obtaining data, and present important new findings which significantly expand Brown's original insights. The book will be of great interest to psychiatric researchers as well as to all mental health professionals who work with schizophrenic patients and their families.