Title
Similar Disorders Viewed With Different Perspectives: A Challenge for Neurology and Psychiatry

Journal
JAMA Neurology, 51(10)

ISSN
2168-6149

Authors
Starr, Arnold
Sporty, Larry D

Publication Date
1994-10-01

DOI
10.1001/archneur.1994.00540220023010

Similar Disorders Viewed With Different Perspectives

A Challenge for Neurology and Psychiatry

DURING THE past 10 years, we have explored clinical and theoretical relationships of psychiatry and neurology, using the venue of a didactic teaching conference. The patients come from the neurology or psychiatry inpatient or consultation services. Their history and examination are presented by the student or resident, and then the patient is interviewed and examined by the authors, one, a neurologist, and the other, a psychiatrist. We will present some of the recurring themes that unite neurology and psychiatry in the clinical management of patients and how certain issues separate the disciplines.

Arnold Starr, MD

NEUROLOGY AND PSYCHIATRY EMPLOY DIFFERENT 'LANGUAGES' TO DESCRIBE SIMILAR PROCESSES: DENIAL AND ANOSOGNOSIA

Certain patients do not "know" why they are in the hospital. When the patients originate from the neurological service, they can have a hemiparesis or be demented and yet have little insight into their problem. The patient with hemiparesis "neglects" the personal and extrapersonal space on the side of the affected limb and will suggest that the affected limb could not be his/her own. The demented patient, when unable to perform mental arithmetic or remember a list of words, may attribute the performance deficits to transparent excuses such as "never" having been able to perform such tasks. The neurologist refers to the loss of insight or knowledge about one's medical condition as an *anosognosia*. There is an assumption that regions of the brain regulating self-knowledge have been damaged. It is relevant that patients with anosognosia are more likely to appear depressed than are patients whose insight into their disordered neurological state is preserved.

The patient from the psychiatric service will have been admitted because of inappropriate and potentially dangerous behavior. The patient, when questioned about the events, may consider the behavior as appropriate to the situation or deny that the behavior occurred. The psychiatrist refers to the patient's lack of insight as a *denial*.

The common theme in these neurological and psychiatric patients is their relatively poor knowledge about their conditions. The neurological patient usually has a structural lesion of the brain. The psychiatric patient usually has a "normal neurological examination" and normal imaging studies of the brain. When these expected associations are not present (eg, normal study results in the neurological patient or abnormal study results in the psychiatric patient), assistance is sought from the physicians on the "other" service, often leading to transfer to the "appropriate" service, or worse territorial disputes between the physicians on the two services.

We have come to consider patients with anosognosia or with denial as having a common disorder of brain structures and functions controlling insight and knowledge. While lesions of the frontal and right hemisphere are frequently accompanied by a lack of awareness of deficits, there are sufficient reports of anosognosia accompanying lesions in widely separate areas of the brain to implicate many brain regions in self-awareness. The failure of psychiatric patients to recognize their own behaviors could also be due to altered function in some of these same systems.

The mechanisms of anosognosia are as yet unclear.[1] Proposals that anosognosia accompanying unilateral neglect is due to attentional or sensory deficits cannot account for the failure of such patients to describe mental images of *prior* experiences that have occurred contralateral to the hemispheric lesion.[2] This finding suggests that anosognosia is due to disordered cognitive processing. Psychiatrists have traditionally considered denial as a cognitive process providing resolution of psychological distress. Psychiatrists seem comfortable with the lack of identifiable brain changes with the occurrence of denial, whereas neurologists have tried to asso-

ciate anosognosia with particular brain lesions. This difference may reflect how neurologists and psychiatrists view mechanisms of behavior rather than the difference between the processes of anosognosia and of denial.

Clinical observation attests to the capacity of the systems underlying self-knowledge to be dynamic. Clinical recovery for both the neurological and psychiatric patients accelerates as they become "aware" of their deficits. For the neurological patient with an acute lesion, insight gradually reappears. Insight may not improve in patients with degenerative disorders. For the psychiatric patients, medications such as phenothiazine may be of help in allowing the reestablishment of insight, so that cognitive therapy directed to the problem can begin.

We postulate that disorders of the neurological systems underlying self-knowledge contribute to psychiatric and neurological illness, but that the designation of the accompanying symptoms of the disorder, ie, anosognosia or denial, is different for the two disciplines. The Greek word *gnosis* may be translated as either "knowledge" (the neurologists' choice) or as "insight" (the psychiatrists' choice), reflecting the similar origin of the two descriptors.

NEUROLOGY AND PSYCHIATRY MAY ENCOUNTER DIFFERENT ASPECTS OF DISORDERS OF THE SAME SYSTEMS

Temporal Lobe Epilepsy and Schizophrenia

Complex visual hallucinations are common in temporal lobe seizures and have also been reported at high incidence in patients with schizophrenia.[3] The hallucinations in temporal lobe epilepsy are ictal phenomena, often amenable to treatment with anticonvulsant drugs or surgery. The visual hallucinations in schizophrenia are considered a manifestation of the underlying disease, sometimes reflecting underlying psychodynamic conflicts, and are often amenable to treatment with neuroleptic drugs.

We have been struck by similarities of descriptions of the visual hallucinations experienced by patients with temporal lobe epilepsy and those with schizophrenia. The visual hallucinations in both disorders are recurrent, brief in duration, involve people or objects of unusual shape or form, and are accompanied by emotional experiences.

Some of the descriptions of the visual hallucinations of which we have heard are as follows.

A patient with the diagnosis of schizophrenia described repeated visions of childlike figures floating in the air at unusual angles. The figures wore white gowns and were silent. The patient was fearful when these visions appeared. Another patient with the diagnosis of schizophrenia described a pair of black patent leather shoes floating in the air. The shoes were disembodied. The patient found the visions disturbing. A third patient with schizophrenia described a cloaked figure of gigantic proportions that would appear suddenly. The figure was threatening.

A patient with temporal lobe epilepsy described a recurrent scene involving a short middle-aged man standing behind a counter with brightly colored objects to sell. The man was silent. The patient felt severe discomfort as the scene appeared. A second patient with temporal lobe epilepsy described a translucent floating rainbow-colored sphere before losing consciousness. There was pleasant sensation accompanying the visions.

The similarities of the hallucinogenic experiences in these patients with such diverse diagnoses as schizophrenia and temporal lobe epilepsy invite the proposition that similar brain regions are involved in these disorders. In one group of patients, there are associated cognitive and psychological factors that lead to the diagnosis of "schizophrenia." In the other patients, there are abnormalities on magnetic resonance imaging or electroencephalograms and clinical seizures leading to the diagnosis of a temporal lobe lesion. Recent studies[4] reveal statistical evidence of volume changes in parahippocampal and other brain areas in schizophrenia, suggesting that the temporal lobe is abnormal in schizophrenia.

Patients with temporal lobe lesions frequently have interictal personality disorders. The personality disorder may be secondary to subclinical seizure activity and be responsive to treatment with carbamazepine. Carbamazepine is also now commonly used in the treatment of personality disorders accompanying some psychiatric illnesses. Dostoevsky, a favored Russian novelist among neurologists and psychiatrists,[5-11] had temporal lobe epilepsy. In his novels, he recognized the relationship of temporal lobe disorders to altered behavior and emotions. His protagonists had temporal lobe seizures and singular, and even unlawful, behaviors. In *Crime and Punishment*, Raskolnikov embarks on an ill-conceived murder of an elderly woman. Psychiatrists and neurologists by nature of their separate training and theoretical assumptions emphasize different clinical aspects of this same novel. Traditional psychiatric interpretation of *Crime and Punishment* focuses on the role of Raskolnikov's guilt as the driving force that eventually leads to his arrest, whereas the neurologists' emphasis is on the role of the temporal lobe abnormality in contributing to Raskolnikov's altered behaviors.

Prosopagnosia and Capgras' Syndrome

Bilateral lesions of the occipital lobe with an accompanying altitudinal field cut can be associated with a persistent loss of facial recognition of familiar faces while preserving the ability to learn and recognize unfamiliar faces.[12] These patients can identify the familiar person by voice and may attribute their inability to recognize the face to some cosmetic change.

Psychiatric patients may identify a family member as an imposter. This visual misinterpretation is seen in schizo-

phrenia and dementing illnesses. The clinical syndrome is referred to as *Capgras' syndrome*. Patients will say that the person's face, while closely resembling that of a family member, has some ill-defined difference that makes the patient sure that the individual is an imposter.

Patients with prosopagnosia and with Capgras' syndrome represent clinical manifestations of disordered facial processing. The neurological patient cannot match the visual image of the face with prior experience. The psychiatric patient, in contrast, recognizes the face but cannot connect it to the individual under regard. Both clinical syndromes are manifestations of the neurological systems involved in the automatic yet complex process of processing facial attributes. This perceptual system also extends to the identification of facial affect that can be impaired in patients with brain lesions.[13] The ability to correctly identify affect is essential for responding appropriately in social settings. Psychiatric patients frequently misinterpret facial expressions or body postures. The neurological systems involved in facial recognition and identification may be impaired in a variety of different ways, leading to characteristic clinical syndromes in neurology and psychiatry.

NEUROLOGY AND PSYCHIATRY ARE UNCOMFORTABLE WITH THE SAME PATIENT: CONVERSION DISORDERS VS ORGANIC DISORDERS

The neurological patient with unusual seizures is often referred to the psychiatrist to define an "emotional" cause for the behavior. The converse, a patient on the psychiatric service manifesting seizures, hemiparesis, or blindness is referred to the neurologist to ensure that "an organic lesion is not present." The need to ascribe the disorder to either psychiatric or neurological origin is often the compelling force in the assessment of these patients. This need for such a distinction may reflect current psychiatric diagnostic criteria that insist that the diagnosis of a conversion disorder cannot be applied if the patient has a neurological disorder that could account, in part, for the symptoms or signs (*Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition*[14]). In the course of our conferences, we have been struck by the frequent interaction of psychiatric and neurological issues in the description and analysis of patients' disorders.

A recurrent example is the patient with "pseudoseizures" who manifests seizures in response to emotional conflicts. Some of the seizures are "true" seizures, as manifested by electroencephalographic changes, and some are pseudoseizures with normal electroencephalograms and atypical clinical features. Effective treatment for these patients includes both psychiatric and neurological interventions. Either treatment alone is insufficient.

Patients meeting the criteria of conversion disorder frequently go on to have neurological lesions on long-term follow-up.[15] We must remember that many of the behaviors, once considered a conversion disorder, are now classified as organic (eg, dystonia, movement disorder, dyslexia). Patients, even when classified as having an "organic" disorder, still have psychological factors that influence their illness. In some of these patients, addressing the psychological issues is of great therapeutic benefit.

Finally, many of the classical criteria employed to distinguish conversion from organic disorders are unreliable.[16] For instance, "la belle indifference," the characteristic affect of patients with conversion disorder, can also be seen in seriously medically ill patients. Urinary and fecal incontinence, a classic occurrence in seizures, appears in patients with psychiatric disturbances in whom the incontinence may be an expression of an underlying psychological conflict or need. We saw one patient being evaluated for "seizures" who had a delusion that urination blessed the recipient site. The localization of the boundary of impaired cutaneous sensation exactly at the midline was considered a classic sign of conversion disorder. Many patients with lesions of central sensory pathways have also been known to split the midline in an attempt to convince the examiner of their illness.

We have come to appreciate two axioms that have facilitated our care of the neurological/psychiatric patient. First, almost all patients have emotional factors that impinge on the expression of neurological symptoms; second, neurological deficits contribute to psychological symptoms.

THERE CAN BE COEXISTENCE OF PSYCHIATRIC AND NEUROLOGICAL DISORDERS

We have cared for a young man with temporal lobe epilepsy in whom effective treatment of the seizures with various anticonvulsant drugs was accompanied by the development of an unremitting psychosis. Cessation of the anticonvulsant drug led to a return of the seizures, but a disappearance of the psychosis. There are other cases of this same sequence of events referred to as "forced normalization."[17] In our patient, the family found the seizures to be more socially acceptable than the psychiatric disorder. A combination of neuroleptic drugs with incomplete anticonvulsant control of the seizures has proved to be an effective approach for this patient.

Obsessive-compulsive disorders occur with high frequency in patients with basal ganglia disorders.[18,19] We have used a combination of therapies directed to the movement disorder and to the cognitive disorder to best care for such patients.

There are also several examples in which a treatment directed to a neurological or to a psychiatric problem will create new problems of the "other sort" as unwanted side effects of the treatment. We have exploited these side effects as examples of the proximity between certain psychiatric and neurological diseases. Some examples to consider are the use of phenothiazine in psy-

chotic patients that may render them "parkinsonian" with bradykinesia, rigidity, or tremor. Treatment with levodopa may convert a patient with Parkinson's disease to one with hallucinations and paranoia. Electroconvulsive shock therapy, a treatment for depression, has unexpectedly affected a marked improvement of neurological symptoms in patients with Parkinson's disease.

CONCLUSIONS

We have come to appreciate that the fields of psychiatry and neurology are often separated artificially by language and by different assumptions and emphasis. The patients whom we see often benefit by reconsidering their disorder in terms of both disciplines allowing the application of appropriate therapies. We have presented examples of psychiatric syndromes that may have a neurological basis as well as the importance of psychiatric issues in many neurological illnesses. We recommend that efforts be made to bridge the gaps separating psychiatry and neurology, even to the point where the two specialties might become one. Advances in both specialties will be encouraged if residents in training and medical students were exposed to patient-based interactions between neurologists and psychiatrists. We prefer not to dwell on the distinctions between the disciplines but rather consider the mechanisms and treatment of brain disorders that reflect the common origins of psychiatry and neurology.

Arnold Starr, MD
Larry D. Sporty, MD
Department of Neurology
University of California–Irvine
La Jolla, CA 92717

REFERENCES

1. Heilman KM, Bowers D, Valenstein E, Watson RT. Hemispace and hemispatial neglect. In: Jeannerod M, ed. *Neurophysiological and Neuropsychological Aspects of Spatial Neglect*. Oxford, England: North Holland Press; 1987:115-150.
2. Bisiach E, Luzzatti C. Unilateral neglect of representational space. *Cortex*. 1978; 14:129-133.
3. Bracha HS, Wolkowitz OM, Lohr JB, Karson CN, Bigelow LB. High prevalence of visual hallucinations in research subjects with chronic schizophrenia. *Am J Psychiatry*. 1989;146:526-528.
4. Bogerts B, Meertz E, Schoenfeldt-Barsch R. Basal ganglia and limbic system pathology in schizophrenia: a morphometric study of brain volume and shrinkage. *Arch Gen Psychiatry*. 1985;42:784-791.
5. Kiloh LG. The epilepsy of Dostoevsky. *Psychiatr Develop*. 1986;4:31-44.
6. Alajouanine T. Dostoiewski's epilepsy. *Brain*. 1963;86:209-218.
7. Gastaut H. New comments on the epilepsy of Fyodor Dostoevsky. *Epilepsia*. 1984;25:408-411.
8. Voskuil PH. The epilepsy of Fyodor Mikhailovitch Dostoevsky (1821-1881). *Epilepsia*. 1983;24:658-667.
9. Miksanek T. Dostoevsky's *The Idiot* and psychic disorders in epilepsy. *Perspect Biol Med*. 1982;25:231-237.
10. Cirignotta F, Todesco CV, Lugaresi E. Temporal lobe epilepsy with ecstatic seizures (so-called Dostoevsky epilepsy). *Epilepsia*. 1980;21:705-710.
11. Morgan H. Dostoevsky's epilepsy: a case report and comparison. *Surg Neurol*. 1990;33:413-416.
12. McNeil JE, Warrington EK. Prosopagnosia: reclassification. *Q J Exp Psychol*. 1990;43A:267-287.
13. Young AW, Newcombe F, de Haan EFH, Small M, Hay DC. Face perception after brain injury. *Brain*. 1993;116:941-959.
14. American Psychiatric Association, Committee on Nomenclature and Statistics. *Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition*. Washington, DC: American Psychiatric Association; 1987.
15. Marsden CM. Hysteria: a neurologist's view. *Psychol Med*. 1986;16:277-288.
16. Lazare A. Current concepts in psychiatry: conversion symptoms. *N Engl J Med*. 1981;305:745-748.
17. Wolf P. Acute behavioral symptomatology at disappearance of epileptiform EEG abnormality: paradoxical or 'forced normalization.' *Adv Neurol*. 1991;55:127-142.
18. Schilder P. The organic background of obsessions and compulsions. *Am J Psychiatry*. 1938;94:1397-1414.
19. LaPlane D, Levasseur M, Pillon B, et al. Obsessive-compulsive and other behavioural changes with bilateral basal ganglia lesions. *Brain*. 1989;112:699-725.