References

1. Hans WH, Daphne VH: Review of the prevalence and incidence of eating disorders. Int J Eat Disord 2003; 34:383–396
2. Pfohman M, Criaddle LM: Epidemiology of intracranial aneurysm and subarachnoid hemorrhage. J Neurosci Nurs 2001; 33:39–41
3. Krahn DD, Mitchell JE: Case report of bulimia associated with increased intracranial pressure. Am J Psychiatry 1984; 141: 1099–100

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Facilitation in Inducing Folie a Deux Through Healthy Precipitator

TO THE EDITOR: Folie a deux is a psychotic disorder characterized by a shared delusion that is instigated by a psychotic inducer and a healthy recipient (1). We describe an unusual case in which the psychosis in the recipient appeared only after the most influential member of the family adapted the psychotic ideas of the inducer.

"Mr. G" was a 23-year-old man with no history of psychiatric disorder. He was hospitalized with a paranoid delusion that his brother-in-law was a Mafia leader who had plans to murder him. Mr. G also heard voices telling him to kill himself or he would be subjected to torture. Consequently, he tried to strangle himself in the hospital.

Additional history revealed that Mr. G's sister developed similar symptoms prior to his hospitalization and that she had been in close contact with him during that time. Their mother was aware of her daughter's delusional ideas. While the mother did not believe in the delusions, she provided her daughter with unconditional support and even checked her house for surveillance equipment without asking questions. The mother felt an obligation to maintain family cohesion, even if her actions contradicted her own comprehension of reality. Therefore, she avoided any confrontation with her daughter out of fear the daughter might commit suicide, which is what an uncle did several years previously.

Subsequent to his sister's delusions, Mr. G's first psychotic symptoms appeared when he realized that his mother was fully supportive of his sister's beliefs. Notably, his psychosis exacerbated only after his mother's visits on the psychiatric ward, and his attempt to strangle himself occurred after a visit from her. He received intensive treatment with doses of diazepam (30 mg/day) as well as individual and family psychotherapy that focused on reality testing of both the patient and the family.

It became apparent that the mother played a major role in the family and had indirectly supported Mr. G's psychosis. Following intervention to clarify the mother's influence on Mr. G, she changed her attitude and denied explicitly the existence of his delusions. Consequently, Mr. G began to improve rapidly. After six sessions of family psychotherapy, he was free of psychosis and could be discharged.

It is generally accepted that a dyad composed of a charismatic psychotic inducer and an induced person with dependent character traits is necessary for the development of shared psychosis (2). To our knowledge, the case presented here is the first documented case in which the pathogenic influence of a noncharismatic psychotic inducer was enhanced by a healthy charismatic family leader who was fully supportive of psychotic ideas without sharing those ideas. This case introduces the possibility that a psychotic inducer does not have to be a dominant person in a family. On the other hand, it does emphasize the role of a family leader in transmitting an induced psychosis (3).

References

1. Mickaud R: Translation of Lasègue and Farlet's paper of 1877. Le folie à ou folie communiquée. Am J Psychiatry 1964; 121(suppl 4)
2. Howard R: Induced psychosis. Br J Hosp Med 1994; 51:304–307
3. Latan B: The psychology of social impact. Am Psychol 1981; 36:343–365

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Immobilization Panic

TO THE EDITOR: Fear-associated freezing/immobilization is a well described, adaptive, defensive behavioral phenomenon that is common in many species of animals and occurs during conditions of natural threat or fear. Although freezing behavior has been assessed in humans utilizing stress/anxiety paradigms in the laboratory (1), no study, to our knowledge, has explicitly examined the prevalence of freezing or immobilizing behaviors in a clinical sample.

In our study, we used the NIMH Panic Questionnaire (2), a self-report instrument designed to elicit detailed, syndrome-specific information in patients with panic disorder, to obtain information regarding panic-related, freezing/immobilizing behavior. The frequency ("never," "rarely," "sometimes," "always") and severity ("mild," "moderate," "severe," "extreme") of 44 panic attack symptoms were obtained in a mixed treatment- and community-based sample of 1,118 people who met self-reported DSM criteria for panic disorder. In our analysis, we focused on a single item that determined whether the subjects were actually immobilized during a panic attack.

Among the participants, 198 (18%) reported "always" being immobilized during panic attacks. Fifty-three percent of the participants reported varying frequencies of immobilization panic ("sometimes" [N=405] and "rarely" [N=188]). Thus, 71% of the participants reported lifetime episodes of immobilization panic. Notably, subjects with positive lifetime histories of immobilization panic were 2.3 times (95% confidence interval [95% CI]=1.73–2.95, $p<0.001$) and 1.6 times (95% CI=1.21–2.09, $p<0.001$), respectively, more likely to suffer from disabling chronic anxiety and sleep panic attacks relative to panic disorder patients who had never experienced immobilization panic. Moreover, panic disorder patients who had experienced immobilization panic were 2.4 times (95% CI= 1.64–3.57, $p<0.001$) more likely to also experience work impairment relative to panic disorder patients who did not report immobilization panic. This latter finding is noteworthy because work absenteeism is increased in panic disorder patients relative to nonpanic disorders in primary care settings.

Data presented in this report indicate that immobilizing/freezing behaviors are common, yet clinically underappreciated, events during panic attacks. The fact that patients with

immobilization panic report a more severe course of illness underscores the importance of soliciting such data as part of standard clinical assessment.

Our laboratory, as well as that of others (3), observes a phenomenological overlap and possible comorbid association between panic attacks and sleep paralysis, a rapid eye movement-related event characterized by muscle atonia and frightening immobilizations that can either emerge from or intrude upon sleep/wake states (4). Future studies are needed to determine the nature of freezing behaviors and related phenomena (e.g., muscle atonia/paralysis) in panic and other anxiety disorders during sleep/wake states.

References

1. Azevedo TM, Volchan E, Imbiriba LA, Rodrigues EC, Oliveira JM, Oliveira LF, Lutterbach LG, Vargas CD: A freezing-like posture to pictures of mutilation. Psychophysiology 2005; 42:255–260
2. Scupi BS, Maser JD, Uhde TW: The National Institute of Mental Health Panic Questionnaire: an instrument for assessing clinical characteristics of panic disorder. J Nerv Ment Dis 1992; 180: 566–572
3. Bell CC, Dixie-Bell DD, Thompson B: Further studies on the prevalence of isolated sleep paralysis in black subjects. J Natl Med Assoc 1986; 78:649–659
4. Uhde TW: The anxiety disorders, in Principles and Practice in Sleep Medicine (3rd edition). Edited by Kryger MH, Roth T, Dement W. Philadelphia, WB Saunders, 2000, pp 1123–1139

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Author Retraction

In July 2002, an article entitled "Expression of Oct-6, a POU III Domain Transcription Factor, in Schizophrenia" was published in the *Journal* (159:1174–1182). We wish to retract one of the conclusions.

Since publishing this study, our laboratory at the Institute of Psychiatry, King's College London has had difficulty reproducing some of the findings. In particular, the primary observation that Oct-6 is ectopically expressed in schizophrenic brain tissue could not be reproduced in the Stanley series of postmortem samples. These negative findings were recently published (1).

This failure to reproduce led us to re-examine the data underlying the original publication. We now conclude as follows:

Figure 1 in the original paper presented the characterization of an anti-Oct-6 antibody by electromobility shift assay. These data were contributed by one of the co-authors (D.M.) from his laboratory and are completely correct as far as we are aware.

Figure 2, the principal component of the study, illustrated the immunohistochemical analysis of schizophrenic and control tissue and was conducted in the King's College Laboratory. These data have proved irreproducible in other schizophrenia samples (1), but we have no specific evidence that they are incorrect. Material from the original samples is no longer available, so unfortunately a direct rerun of these precise experiments is not possible. Nonetheless, we would say that these data should be regarded as unreliable.

Figure 3 presented an immunoblot analysis of Oct-6 expression in schizophrenic and control tissue. We have prima facie evidence that these data are fraudulent. There are two reasons for reaching this conclusion. First, close examination of the lanes on this figure indicate that they have been manipulated and cannot be what they purport. Second, we have been able to track the derivation of this figure from the primary data, and that analysis reveals that the data have been manipulated. Needless to say, these data should not be considered reliable.

In light of these revelations, we retract the finding that Oct-6 is dysregulated in schizophrenic brain tissue.

The appropriate authorities at King's College were informed of the suspicion of fraud, and an investigation was carried out under the College's "Regulations for Investigating and Resolving Allegations of Research Misconduct." That investigation, assisted by the production of a report from an independent expert, concluded unequivocally that some primary data produced in the King's College Laboratory by Dr. Maria Ilia had either been falsified or had been wrongfully manipulated to produce a misleading analysis. As a result of this, the signatories to this letter now formally retract the paper.

We would like to make clear that the source of the data was a single researcher in the King's College Laboratory. The investigation by the College has attached no suspicion at all to the other authors of the paper. That includes those currently and previously at the Institute of Psychiatry or in Rotterdam.

Finally, we wish to apologize sincerely to *The American Journal of Psychiatry* and its readership. You have a right to expect the highest standards of academic practice from authors whose work is submitted to you. Clearly, there has been a substantial failure in this regard, and we are embarrassed and distressed that this has occurred.

Reference

1. Uhbi K, Price J: Expression of POU-domain transcription factor, Oct-6, in schizophrenia, bipolar disorder and major depression. BMC Psychiatry 2005; 5(Oct 24):38

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The Journal *sought comment from Dr. Ilia on this matter, but none was received.*