Reference

1. Widom CS: The cycle of violence. Science 1989; 244:160-166

ANGELA E. McHOLM, PH.D. HARRIET L. MACMILLAN, M.D. ELLEN JAMIESON, M.ED. Hamilton, Ont., Canada

The Nosology of Juvenile Mania

To the Editor: Ellen Leibenluft, M.D., et al. (1) presented an informative and useful realignment of the nosology for juvenile mania. The authors considered an array of important "methodological and conceptual issues" in their analysis, but they did not clearly distinguish between the methodological and the conceptual. That is, to what extent do the authors put forth the new categorization on the basis of the difficulties in assessment of DSM criteria in the context of the juvenile population? Or do they believe that there is a fundamental distinction among the categories they propose? If the latter, to what extent is the conceptual distinction limited to the juvenile population, or should it be applied or adapted for adults as well?

Reference

 Leibenluft E, Charney DS, Towbin KE, Bhangoo RK, Pine DS: Defining clinical phenotypes of juvenile mania. Am J Psychiatry 2003; 160:430–437

HAROLD ALAN PINCUS, M.D. Pittsburgh, Pa.

Dr. Leibenluft and Colleagues Reply

To the Editor: We appreciate Dr. Pincus's comments on our article. We suggested these clinical phenotypes for juvenile mania because of the difficulties that arise when clinicians and researchers try to apply the DSM-IV criteria to children. The question of whether there is a fundamental distinction between these categories is an empirical one, and in the article, we suggested research strategies for addressing it (see our Table 1). For example, it is important to ascertain whether there are consistent differences between the phenotypes in neuropsychological and physiological function, longitudinal course, familial variables, etc. Should such differences exist, subsequent studies in adults would be warranted.

ELLEN LEIBENLUFT, M.D.
DENNIS S. CHARNEY, M.D.
KENNETH E. TOWBIN, M.D.
ROBINDER K. BHANGOO, M.D.
DANIEL S. PINE, M.D.
Bethesda, Md.

Delusional Thoughts in Alzheimer's Disease

To the Editor: The article by David Sultzer, M.D., et al. (1) provides strong additional support, by way of correlation analyses of the observer-rated severity of delusions, for the contribution of right frontal brain dysfunction to the appearance of abnormal beliefs in Alzheimer's disease. This form of analysis has the merit of accounting for the contribution of other variables, such as age, age at onset, and severity of dementia, as well as the behavioral factor of agitation, to variations in regional brain metabolism. There are, however, some comments to be made about the interpretation of the results and,

perhaps more important, about the method of study adopted by the authors.

The findings were seen as evidence for a linear relationship between delusional "severity" and the degree of impairment of metabolism in areas of the right frontal cortex. There are challenges to this interpretation. It is equally possible that the content and personal significance of the delusions described (about half of those outlined could reasonably be considered elements of a misidentification syndrome) might have had some variable influence on the behavioral assessment of delusion severity on the Neurobehavioral Rating Scale. In other words, an association of the nature, as much as neuropsychiatric severity, of abnormal beliefs with quantitative variation in regional brain metabolism has not been fully examined. Equally, there is evidence from case studies that delusions that have a substantial impact on behavior (and would have been highly rated on the Neurobehavioral Rating Scale) may appear at the minimal stage of Alzheimer's disease in association with subtle and confined cortical dysfunction and that they impair a specific set of cognitive abilities (2, 3).

The results of the study extend previous evidence from cross-sectional studies of similar populations. Reliance on a dimensional approach in a group showing diverse delusional phenomena, however, may continue to divert attention from methods more likely to foster an analytic understanding of delusional states. These methods will rely on the study of multiple single cases, as has been so fruitful in the analysis of Capgras syndrome (4), and will likely combine detailed clinical phenomenology, functional imaging, and cognitive neuropsychology (5). The discrimination of delusions with a factual content satisfying traditional clinical criteria from affectively laden persecutory beliefs may well be of heuristic value but will not sufficiently inform etiological studies in both organic and functional delusional disorders. Firmly held factual delusional beliefs can arise from specific memory failures and be affectively laden when the disorders of memory or other aspects of cognition involve issues of autobiographical knowledge and personal identity.

References

- Sultzer DL, Brown CV, Mandelkern MA, Mahler ME, Mendez MF, Chen ST, Cummings JL: Delusional thoughts and regional frontal/temporal cortex metabolism in Alzheimer's disease. Am J Psychiatry 2003; 160:341–349
- Venneri A, Shanks MF, Staff RT, Della Sala S: Nurturing syndrome: a form of pathological bereavement with delusions in Alzheimer disease. Neuropsychologia 2000; 38:213–224
- Shanks MF, Venneri A: The emergence of delusional companions in Alzheimer's disease: an unusual misidentification syndrome. Cogn Neuropsychiatry 2002; 7:317–328
- Ellis HD, Lewis MB: Capgras delusion: a window on face recognition. Trends Cogn Sci 2001; 5:149–156
- 5. Frith C: Commentary on Garety and Freeman II: cognitive approaches to delusions—a critical review of theories and evidence. Br J Clin Psychol 1999; 38:319–321

MICHAEL F. SHANKS, F.R.C.PSYCH, D.PHIL. ANNALENA VENNERI, PH.D. Auckland, New Zealand

Drs. Sultzer and Mendez Reply

To the Editor: We appreciate the comments by Drs. Shanks and Venneri that address the interpretation of results from