Correspondence

We will try to publish authors' responses in the same edition with readers' comments. Time constraints may prevent this in some cases. The problem is compounded in the case of a quarterly journal where continuity of comment and redress is difficult to achieve. When the redress appears 3 months after the comment, 6 months will have passed since the original article was published. Therefore, we would suggest to our readers that their correspondence about published papers be submitted as soon as possible after the article appears.

Panic-Agoraphobia

To the Editor: Dr. Katerndahl's study[1] concluded that the characteristics of a panic attack are important in the development of phobic avoidance and its severity and scope. Fava, Grandi, and Canestrari,[2] in their article on prodromal symptoms and panic disorders with agoraphobia, suggest that phobic avoidance in panic disorders with agoraphobia *may not be secondary to the panic attack.* Generalized anxiety, hypochondriasis, and agoraphobia *preceded* the first panic attack in 18 of 20 patients in their study. Marks's[3] view is supported by these workers that agoraphobic symptoms are primary with respect to panic attacks, which is counter to current thinking and DSM-III-R classification.[4] Clearly, both studies challenge Klein's[5] view of the pre-panic patient as a confident and energetic individual.

The cluster of symptoms (phobias, anxiety, and hypochondriasis), which characterized the Fava, et al. study sample before panic attacks, suggests an anxious cognitive style that may lend itself to an *intensified interpretation* of a panic attack. Hence, the intensity of an attack may be the result of the rapidity of the onset of anticipatory anxiety and may be the result of a person's cognitive beliefs and attitudes about illness. It will be important to follow future studies to determine what will be the important prodromal symptoms in patients with panic disorders and whether phobic development precedes or follows a panic attack problem.

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The above letter was referred to the author of the article in question, who offers the following reply.

To the Editor: I wish to thank Dr. Gulledge for his thoughtful letter. His suggestion that cognitive and personality factors may be important to the development of panic attacks and agoraphobia is certainly well taken. Patients' interpretations of the significance of their symptomatology may be a critical factor. Path analysis is not designed to provide a definitive etiologic interpretation but, rather, to begin to develop causal models from nonprospective data. There may be important factors either preceding the panic attacks or intervening between panic attacks and agoraphobia that were not assessed but are indeed critical to the model. Path analysis cannot provide a definitive answer to etiologic questions.

Although I am aware of Marks's view that agoraphobic symptoms may be primary to panic attacks, certainly, the majority of evidence supports the panic-agoraphobia causal model.[1] Not only is it rare for agoraphobia to be found in the absence of preceding panic attacks,[2] but this relationship is also supported by genetic studies,[3,4] clinical studies,[5] treatment studies,[6] and the covariation of panic and agoraphobic fear.[7] In addition, relapse in treated agoraphobics is often preceded by resurgence of their panic attacks.[8,9] Marks himself acknowledges that " . . . it is undeniable that panic attacks can trigger avoidance."[10]

I am aware that the study by Fava, et al.[11] of 20 patients referred to the psychological medicine service of the University of Bologna in Bologna, Italy, found that 18 patients had phobic avoidance prior to their first panic attack. These findings may represent a very skewed population, producing skewed results. More likely, the authors have failed to recognize that, although agoraphobia develops over a period of time, panic disorder as well may not begin with a single, well-demarcated, full-blown panic attack. Limited symptom attacks tend to progress to full-blown panic attacks over time,[12] and there is considerable evidence that both infrequent and limited symptom attacks have similar rates of phobic avoidance to frequent and full-blown panic attacks, respectively.[13-15] Indeed, a study focusing on 144 agoraphobics without panic disorder found that 45 had infrequent panic attacks and that 11 had limited

symptom attacks.[16] Therefore, it may be that Fava, et al. failed to identify infrequent or limited symptom attacks that began prior to the onset of phobic avoidance. In my study, although there were several patients in whom the onset of panic attacks and phobic avoidance coincided, the remainder of patients had a significant lag time (mean = 3.18 years) between the onset of panic attacks and phobic anxiety.

The ultimate answer to the natural history of panic disorder and agoraphobia will have to wait for longitudinal studies in which patients at risk for the development of panic disorder/agoraphobia are followed over time for the development of each condition.

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Postpartum Pap Smear

To the Editor: In the article by Weiss, et al., "The Postpartum Papanicolaou Smear," in the January–March 1989 issue, cases of abnormal cervical cytology on the postpartum examination that were not predicted on the prenatal Pap results were presented. While I support the conclusion of the study, to perform Pap smears during prenatal care and again at the postpartum examination, even when the prenatal Pap is normal, I question the 4.9 percent "conversion" rate to abnormal cytology. When defining specimen collection techniques and interpretation of results, no mention was made of assessing adequacy of smears by the presence or absence of endocervical cells. The presence of endocervical cells is an important indicator of the adequacy of a Pap smear.[1] One can only be sure that the entire transformation zone has been accurately sampled if endocervical cells are present on the smear.[2] Furthermore, the rate of epithelial abnormalities has been reported to be lower in smears that contain no endocervical cells.[3] With pregnancy, changes occur in the anatomy of the cervix, and the endocervical canal is filled with a tenacious mucus that can block access to the columnar cells underneath, causing a lower yield of endocervical cells in pregnant women.[4]

Because the progression from normal endocervical cells to dysplasia to carcinoma-in-situ is more rapid than formerly thought, Dr. Weiss's study could hold important clinical implications. However, without the adequacy of sampling technique assessed, one would wonder about the degree of false-negative readings on the prenatal examination in the study group. This would potentially lessen the impact of the conclusion drawn in this paper.

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The above letter was referred to the authors of the article in question, who offer the following reply.

To the Editor: Dr. Baxley is correct. The apparent "conversion" of some normal Pap smears into abnormal Pap smears during the course of pregnancy may, in reality,