biopsied in patients with TBP are biopsied based on the fact that they are biologically dynamic (ie, new or changing compared to baseline images). It is plausible that albeit histologically “benign,” a higher percentage of these lesions, matched with their histologically equivalent, clinically static counterparts, are biologically destined towards melanoma (ie, the true melanoma precursor lesion). If this theory were valid, we should anticipate encountering fewer melanomas in patients followed with TBP. Although the number of melanomas in Risser et al’s study is too small to draw any firm conclusions, it is intriguing to note that three melanomas were diagnosed in patients that did not have TBP and no melanomas were diagnosed in patients with TBP.

In conclusion, based on the information provided above, we are of the opinion that despite there being no difference in the biopsy rate in the two arms of the study, TBP may indeed change provider behavior.

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REFERENCES

Fibroadenoma?

To the Editor: A “Letter to the Editor” in the November 2007 issue of the Journal is accompanied by two pictures of a lesion said to be a “congenital trichoadenoma.” The authors state that clinically the lesions were “diagnosed as a nevus sebaceous [sic].” May I suggest that the lesion depicted clinically in Figure 1 is, in fact, a nevus sebaceus and that the changes in the photomicrograph of Figure 2 represent components of that same hamartoma. It is not a trichoadenoma. If the photomicrograph had been shown at scanning magnification (which still could be done for purposes of elucidating the matter for readers of the Journal), it would become apparent that the findings histopathologic, just as those clinical, are all of nevus sebaceus.

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Iatrogenic xanthoderma

To the Editor: I read with interest the clinical review by Haught, Patel, and English on xanthoderma in the December 2007 issue of the Journal. I would like to add from my experience one additional cause of yellow skin.

A 46-year-old female presented with a recent onset of increasingly pronounced yellow skin. This had first been noticed and commented upon by her colleagues. The skin was an evenly toned yellow color with sparing of the conjunctiva. Bilirubin and beta-carotene levels were normal. The patient had been diagnosed with new onset systemic mastocytosis 5 years earlier and was a hypersecretor of gastric acid. To control the gastric symptoms of pain and diarrhea, she had been placed, 2 years earlier, on 300-mg tablets of ranitidine hydrochloride (Zantac; GlaxoSmithKline, New York, NY), 3 at a time, 4 times daily. The 300-mg Zantac tablet is bright yellow and contains D&C Yellow No. 10 Aluminum Lake.

The cause for the yellow skin in this case was absorption of dye from 12 large yellow tablets daily for 2 years. The color was probably made more evident by a coincident pallor from severe iron deficiency anemia (hematocrit, 25).

The Zantac was discontinued and replaced with a non-yellow H2 blocker. The yellow color faded
gradually and was no longer detectable 10 months later.

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