Aspirin “Allergy” and Resistance

In a recent article, Gum et al. (1) discussed aspirin resistance, and Eikelboom et al. (2) wrote an editorial comment regarding this topic. We have seen several patients who reported that they seemed to have “allergic” reactions to aspirin. These “allergic” reactions consisted primarily of asthma-type attacks. Careful study revealed that these patients are not “allergic” to aspirin in the classical way; inhibition of cyclooxygenase by 325 mg of aspirin shifts the arachidonic acid cascade to the lipo-oxygenase branch (Fig. 1). This results in the production of more leukotriene C_4, D_4, and E_4, which together are the “slow-reacting substance of anaphylaxis” and powerful bronchoconstrictors. These patients then refuse to take aspirin and claim that they are “resistant” to and have “allergies” to aspirin.

On the other hand, when patients are given only 81 mg of aspirin, cyclooxygenase production recovers rapidly, including prostaglandin i_2 synthesis, which is platelet-aggregation inhibitory and a vasodilator. Inhibition of the platelet-aggregation inducer thromboxane A_2 appears to persist for several hours. It is generally accepted that low-dose aspirin (81 mg one time daily) should be recom-