function are presented in the table. The patient was restudied four months later, and the results with analgesics were very much the same.

Comment

Airways obstruction was strikingly relieved by aspirin in the patient studied. Similar beneficial effects were produced by three other cyclo-oxygenase inhibitors, that is, by indomethacin, mefenamate, and fenoprofen, but not by two remaining analgesics—salicylamide and benzydamine—which do not inhibit PG biosynthesis (Vane, 1976; Szczeklik *et al,* 1977). It was, therefore, logical to assume that the pharmacological removal of a product of arachidonic acid (AA) cyclo-oxygenation from his respiratory tract helped our patient to overcome the airways obstruction. Perhaps this product was a bronchoconstrictor $PGF_{2\alpha}$, TXA_2, or other as yet unknown metabolite. Whatever the metabolite was, it could not have been a physiological one, since we have found that bronchodilator PGI_2 and PGE_2 are two major products of AA transformation in the lungs (Gryglewski *et al,* 1978).

In our patient the suspected abnormality in arachidonate metabolism was restricted to the respiratory system. Detailed platelet function studies, particularly those related to arachidonic pathway, failed to show any differences from the normal. The transient petechiae were most likely due to increased vascular fragility secondary to triamcinolone administration.

The number of asthmatic patients who might benefit from aspirin is not known. Trial of aspirin treatment might seem warranted in asthma, since it could allow the steroid dose to be reduced, as in our case. Great care, however, would be necessary at the beginning of such treatment, and the initial dose of aspirin should not exceed 20–40 mg, as the same cyclo-oxygenase inhibitors, which proved to be so efficient in relieving bronchoconstriction in our patient, may produce bronchoconstriction in other asthmatics suffering from so-called aspirin-induced asthma (Szczeklik *et al,* 1975; 1977). Thus two opposite reactions can be caused in asthmatics by the same specific inhibitors. This indicates that different mediators, even derived from the same precursor, might play a different role in various types of asthma.

Discussion

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Corrections

1 Warren, C P W *et al.* Mechanical properties of the lung in extrinsic allergic alveolitis. *Thorax*, 1978, **33,** 315–321.
The caption to figure 1 should show (a) four weeks and (b) one week.

2 Battesti, V P *et al.* Chronic cor pulmonale in pulmonary sarcoidosis. *Thorax*, 1978, **33,** 76–84.
The authors regret that they omitted to acknowledge Professor J Turiaf, who originally studied the patients.