Only 30% of them failed only in one step while the rest made more than one error simultaneously. We also studied the kind of outpatient control, if any, devoted to these patients. As expected, the greatest percentage of errors was observed in the group of patients without any kind of medical advice (83%). However, practically the same proportion (79%) failed to use the MDIs properly despite the fact that they were under the supervision of medical outpatient care, either public or private. This proportion fell to 45% in our group of asthmatic subjects controlled by a member of our hospital staff.

Our results do show both that MDIs are extensively used in a wrong fashion and also that the majority of patients failed in more than one of the recommended steps, reflecting the importance of the hand-lung problem.

As suggested by Newman and Clarke, proper instruction is essential to teach the correct inhaler technique. However, since both reported series on the bronchodilator effect of the patient's own technique and that from supervised administration by the physician have been controversial, we suggest that a trial and error method may be the best way to achieve maximal bronchodilator response. In other words, if the asthmatic patient does feel the airway penetration of the nebulized particles, regardless of method, maximal therapeutic effect may probably be achieved, which essentially is the cornerstone of medical advice.

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To the Editor:

We are grateful for the comments made regarding our recent editorial on the proper use of metered dose inhalers (MDIs). Although we are aware of the theoretical advantages of the open mouth technique, we do not recommend it. We estimate that, with the mouthpiece 4 cm from the open mouth, the inhaler needs only to be held 1 cm off-center, or to be turned through an angle of 10 degrees upwards, downwards or to one side in order to spray the drug onto the lips and face. In our experience, the average patient could easily develop this fault. Thus we regard the open-mouth technique as a potential and undesirable source of further error in the use of MDIs. This may explain why one clinical trial3 has in fact shown less bronchodilatation by open-mouth inhalation compared to either closed-mouth inhalation or the use of a cylindrical spacer. Maximal bronchodilator response is achieved with the closed-mouth technique, provided that patients inhale slowly and then hold the breath for 10 seconds.4

After the MDI has been actuated, the metering chamber refills almost instantaneously, and the inhaler will spray again after only one second. However, the temperature of the actuator seating and valve stem falls by 10 to 15°C and does not return fully to ambient temperature for 30 to 40 seconds. The propellant vapor pressure, and hence the spray characteristics, depend upon temperature. A one minute interval between puffs removes any danger of changing the nature of the spray by actuating through a cold nozzle. When more than two puffs are to be taken, a shorter interval is obviously more convenient, and may well be just as effective.


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Selective IgA Deficiency and Sarcoidosis

To the Editor:

We recently described a case of selective IgA deficiency and sarcoidosis associated with recurrent sinopulmonary infections.1 We assumed that this association was coincidental as it has only been previously described in six patients with sarcoidosis, and the incidence of selective IgA deficiency in the general population is estimated to be one in 500. We suggested, however, some common immunologic mechanisms that may underlie both disorders and explain their possible association.2 Shortly thereafter, we encountered another similar case and encourage others to report similar cases to establish whether a real association exists.

Case Report

A 31-year-old male smoker was referred to us in February 1984 to undergo mediastinoscopy for bilateral hilar lymphadenopathy, originally seen on chest x-ray examination in 1966. The patient had suffered from recurrent sinopulmonary infections since the age of 3 years and was suspected to have bronchiectasis in the lower lobes of both lungs. Routine laboratory examination results were within normal limits. The tuberculin skin test (STU of PPD) proved nonreactive. Quantitation of serum IgG, IgA and IgM, using radial immunodiffusion technique on commercially available immuno-plates, showed complete absence of IgA. Serum levels of IgG and IgM were 2,200 and 75 mg/100 ml, respectively. Ouchterlony analysis of the patient's saliva showed complete absence of both IgA and IgM. Gallium-67 lung scan demonstrated increased uptake of the tracer in both hilar and lower lung regions. Serum angiotensin-I converting enzyme level was 3.45 mmoYml (normal range 2.56-3.81 mmoYml serum).3 The diagnosis of sarcoidosis was established through mediastinoscopy and biopsy of an enlarged hilar lymph node. Follow-up to late 1984 revealed no change in his clinical condition or the serum level of IgA, and no change was evident on the chest x-ray film. No corticosteroid therapy was given.

This patient constitutes the eighth reported case of sarcoidosis and selective IgA deficiency and the second reported case, to our knowledge, to be associated with recurrent sinopulmonary infections. In this patient, however, it may well be that selective IgA deficiency is congenital and sarcoidosis developed later.

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