Bronchoconstriction in response to suggestion

SIR,—Dr J E Neild and Professor I R Cameron (2 March, p 674) found no evidence that bronchoconstriction in their asthmatic patients, produced in response to suggestion, was due to airway cooling. Although their findings appear at first sight to contradict our own,[1] there is a major difference in methods. In preliminary studies without suggestion we found that, in addition to heating the nebuliser solution and its driving gas, it was necessary to heat and humidify the auxiliary air entrained during aerosol inhalation to prevent bronchoconstriction. Heating the nebuliser solution and its driving gas alone, which appears to have been the method used by Dr Neild and Professor Cameron, or heating without humidifying the auxiliary air, did not prevent bronchoconstriction after inhalation of isotonic saline. Our findings are in keeping with recent studies in which heated air at ambient humidity was a potent stimulus to bronchoconstriction,[2 3] the bronchoconstriction being closely related to the water content of the inspired air. It is unlikely that the degree of evaporation of aerosol droplets which takes place between generation and inhalation is sufficient to saturate the inspired air at the temperature in these studies.

The findings of Dr Neild and Professor Cameron agree with those of two controlled studies in which bronchoconstriction occurred when inhalation of isotonic saline was accompanied by the suggestion that the solution would cause bronchoconstriction and did not occur when no suggestion was made.[4 5] The suggestion of "bronchoconstriction" is likely to cause hyperventilation unless ventilation is controlled, and the resulting increase in airway water loss would be a sufficient stimulus to cause bronchoconstriction in some patients. By controlling respiratory rate in our study we probably prevented this increase in ventilation in response to bronchoconstriction suggestion.

In our study nine out of 30 patients with asthma developed bronchoconstriction in response to suggestion irrespective of whether it was suggested that the solution would bronchoconstrict or bronchodilate. In the eight patients we were able to restudy none developed bronchoconstriction when the solutions were given at 37°C and 100% humidity. We therefore attributed the bronchoconstriction to the effect of airway cooling, though airway water loss is probably more relevant. Our findings do not exclude the possibility that suggestion can induce bronchoconstriction, but this can only be said with confidence to be unrelated to airway cooling if subjects suffer bronchoconstriction when the inspired air is at 37°C and 100% humidity.

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**The authors reply below.—ED, *BMJ*.

SIR,—Dr Lewis and his colleagues have pointed out the importance of ensuring that the air inhaled during saline challenge is entirely composed of warm, saturated air. Their criticism of our experiments is based on the hypothesis that because the asthmatic subjects would be likely to hyperventilate on suggestion of bronchoconstriction they might entrain extra air which was not fully saturated and warmed. This could account for our observation that some asthmatics undergo bronchoconstriction on inhaling saline if told that it is a bronchoconstrictor substance.

If Dr Lewis and others are correct and there is a differential response in ventilation between those who bronchoconstrict and those who do not this would be a valid criticism. Neither we nor they measured ventilation so we cannot know. In other experiments in which we have exposed asthmatics and normal subjects to stress the increase in measured ventilation has been the same in the two groups.

Lastly, Dr Lewis and his colleagues point out that in their study ventilation was controlled. Their subjects breathed at 30 breaths per minute. Their experiments were performed therefore during controlled hyperventilation, another substantial difference between our two groups.

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Informed consent from the mentally ill

SIR,—Dr R L Palmer is to be congratulated for raising the issue of informed consent from the mentally ill who participate in television programmes (23 March, p 931). The only patients who ever consent to such exposure are those whose prognosis is poor; and that prognosis is worsened, even if marginally, by such an event. In every mentally ill person, even those most wildly psychotic, there remains an intact awareness of the degradation of his personal predicament. Public exposure then serves only to emphasise his personal despair—indeed he may welcome it for just such a purpose—and further erodes such fragments of self esteem he possesses.

These patients will do almost anything to oblige their doctors, nurses, and others, especially if they believe it will enhance the immediate security of their care. A patient of mine offered herself to a television company for a programme in the belief this would help "save the hospital from closure." Her charge nurse objected to this "consent," fearing her appearance would damage the delicate relationship which had been established, directed towards the enhancement of the patient's self esteem. I listened to both nurse and patient and, following much discussion, supported the nurse, to the manifest relief and pleasure of both nurse and patient.

I do not believe it is proper that ill patients, however willing, should be used as instruments of social change. Moreover, it needs to be understood, in every instance when the television camera intrudes on and exhibits personal grief and mental pain of an overwhelming order, that none of us, *in our right minds*, would ever wish so to be publicly exposed.

I do not seek to impugn the motives of any interested party—television, medical, or nursing—but merely to draw attention, as I think Dr Palmer does too, to what is betrayed. Are there not other ways, even if less evocative and arresting to the television viewer, of stimulating a public debate of these very important social issues? Some do not think there is any other way. But if we are not willing to explore other possibilities, then surely we owe it to ourselves and our professional integrity that we acknowledge the nature of the sacrifice made of the immediate welfare of some patients in the interests of many.

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Coeliac disease as a cause of osteomalacia and rickets in the Asian immigrant population

SIR,—Coeliac disease is a relatively common cause of osteomalacia.[1] It should be suspected at any age[2] and confirmed by a small intestinal biopsy showing atrophic mucosa. The symptoms of coeliac disease may be precipitated by pregnancy[3] so it is surprising that Dr P Dandona and colleagues (16 March, p 837) did not perform a biopsy of the small bowel to exclude coeliac disease as the cause of osteomalacia. Coeliac disease can cause florid rickets in immigrant children.[4]

A 13 year old non-vegetarian north Indian girl who had been born in England but had spent seven years in India presented with a three week history of diarrhoea, abdominal discomfort, and listlessness two years after returning to England. She had had vague ill health while in India. Preliminary investigations showed florid rickets on wrist radiography; iron deficiency anaemia; haemoglobin 6·5 g/dl; and normal erythrocyte sedimentation rate, white cell count, and reticulocytes. Rickets was confirmed by a very high alkaline phosphatase of 1618 U/l, skeletal in origin. Further investigations showed malabsorption with abnormalities in xylose absorption and barium meal. Serum vitamin B_{12} was normal and serum folate concentration was low normal.

Tropical sprue was considered as a differential diagnosis,[5] and small bowel biopsy was postponed for a few months. Her anaemia improved rapidly with oral iron and the rickets was treated with oral vitamin D supplements. She recovered quickly in hospital and had no symptoms, but a few months later, though very well, serum alkaline phosphatase values remained high and a small bowel biopsy showed subtotal villous atrophy consistent with coeliac disease. A gluten free diet was introduced, and a year later a small bowel biopsy (before challenge with gluten) was performed. The appearances of the second biopsy were essentially the same as those seen in the previous biopsy but much milder. We suspected that she was not satisfactorily sticking to her gluten free diet. Four months after strict gluten free diet, small bowel biopsy was virtually normal, confirming the diagnosis of coeliac disease. Her rickets had also healed and she was leading a normal life.

This case illustrates the value of small bowel biopsy in diagnosing osteomalacia and rickets. It also illustrates that the symptoms of coeliac disease may disappear on nutritional supplements such as oral iron and vitamin D but that serial small bowel biopsies are needed for diagnosis and to monitor progress. As