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LETTERS TO THE EDITOR

Alternative and complementary medicine for asthma

Readers of the review by D J Lane and T V Lane (November 1991;46:787–97) might conclude that hypnosis has little to offer asthmatic patients. Although the report of Ewer and Stewart[1] is quoted as showing improvement in symptom scores and some peak expiratory flow rates and decreased use of bronchodilators, no mention is made of a 74·9% improvement ($p < 0·01$) in the degree of bronchial hyperresponsiveness to a standardised methacholine challenge test. These authors state that "while our hypnotic technique does not eliminate bronchial hyperresponsiveness it does provide a clinically useful and non-toxic adjuvant to drug treatment that might benefit about half of the asthmatic population." This approach could well reduce the use of the toxic drugs, such as troleandomycin, gold, azathioprine, and methotrexate, mentioned as steroid sparing agents by Shiner and Geddes.[2]

The British Tuberculosis Association's study[3] did not report negative results as stated in the review. On the contrary, "independent clinical assessors considered the asthma to be much better in 59% of the hypnosis groups and in 43% of the control group, the difference being significant." These results were obtained by using only direct suggestion under hypnosis plus autohypnosis daily; more advanced methods, such as reciprocal inhibition, were not used.

In my own study[4] it was possible to withdraw oral prednisolone or to reduce the dose in 14 of the 16 patients treated by hypnosis. The number of hospital admissions during the first year of hypnotherapy fell to 13, compared with 44 during the previous year. This represented a reduction of 249 hospital days, which, at 1988 costs (£170 per day), saved the NHS £42 330. As some 55 000 adults are admitted each year for asthma, savings to the NHS could be considerable if hypnotherapy were to be used more widely.

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AUTHOR'S REPLY We thank Dr Morrison for his interest in our article. On the question of bronchial hyperresponsiveness, he seems to have missed our discussion of this on page 794. The changes recorded by Ewer and Stewart, though significant statistically, are unlikely to make much difference clinically. We know of no work on the use of hypnosis in the sort of chronic persistent asthma that might be treated with the "toxic drugs" he mentions.

Perhaps we were unfair to dismiss the British Tuberculosis Association's study of 1968 as producing "negative results." In fact, the details of the recorded wheezing score, use of bronchodilators, and forced expiratory volume, divided by sex (their table IV), showed a difference between treated and control groups only for wheezing score in females (that is, five out of six comparisons showed no difference). The paper gives no details of the methods used for the independent clinical assessments other than that they "were made by a physician unaware of the patient's treatment."

Dr Morrison's own study gave impressive results, but it is a pity that the comparative control period had to be retrospective. Careful attention to many aspects of the care of asthmatic patients can produce a reduction in corticosteroid treatment and admissions. As we stated in our review, if hypnosis is to be advocated as a means of obtaining these ends there is a need to establish both a reliable method of screening for those likely to be susceptible to hypnosis and a standardised form of treatment acceptable to patients over long periods.

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We read the editorial entitled "Alternative and complementary medicine for asthma" by Dr DJ Lane and TV Lane (November 1991; 46:787–97) with great interest. Patients suffering from asthma often demand complete relief of their symptoms and therefore are not fully satisfied with present medications, which although highly effective are not curative. We have also observed an interest in alternative medicine among asthmatic patients in Turkey. We summarise the findings of an investigation both on the attitude towards "alternative" treatment and on the previous therapy practices of asthmatic patients who presented to the outpatient clinic of the department of chest diseases, Hacettepe University School of Medicine, Ankara, during 1991.

Of the 205 patients who were included in this study, 92 (45%) reported that they had either tried or were still using one or more of the alternative therapies recommended for asthma. Herbal medicine (48), speleotherapy (treatment based on visiting caves: 10), wearing bracelets (6), Turkish baths (5), rabies vaccine (3), and syrups containing various trace elements (3) had been used by these patients. Acupuncture was practised by only two patients; and other methods, such as yoga, hypnosis, and homeopathy, were not used by any.

We have collected 33 different prescriptions for herbal medicine from 48 patients. Numerous types of plants, leaves of trees, fruits, plant roots, and spices have been used either alone or in combination, usually mixed with honey. Though their benefit to the patient is questionable, they are regarded as harmless except for one containing oleander. Quail eggs, the only animal derived protein in these prescriptions, had been used by almost half of the patients.

Speleotherapy, the second most commonly used method, is used not only in Turkey but also in centres in Hungary, Poland, Czechoslovakia, Switzerland, and Italy. Though there have been international meetings and an increasing number of articles on speleotherapy, there have been no controlled studies.[1-4] Some articles have discussed the temperature, humidity, volume, electrical characteristics, types of air flow, and gas content of the indoor environment, but no objective benefit of speleotherapy has been documented.[5,6] Ten patients in our study group visited Damlatas cave in the south of Turkey for three to four weeks in the summer, and all stated that they had felt comfortable for several months after speleotherapy, being able to decrease their bronchodilatator drug dosage. Further controlled and objective studies are needed on this subject.

Bracelets and Turkish baths are two methods of alternative medicine that have not previously mentioned in published reports. The "bracelet" epidemic spread from south east Asia to Turkey, and asthmatic patients as well as those with rheumatological problems began using bracelets. Six patients in the study group were wearing bracelets for the relief of their pulmonary symptoms.

Alternative medicine has emerged as a consequence of continuing efforts for more effective treatment for breathlessness. Some practices have arisen through experience that has accumulated over hundreds of years and have become traditional. Others have resulted from individual trials. We hope that public interest in alternative medicine will diminish in time with both progress in research for more efficient treatments and the realisation by patients of the effectiveness of conventional treatment.

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Air pollution and respiratory morbidity

We read with interest the article by Dr J Britton (May 1992;47:391–2). This raises a number of important points but perpetuates confusion over EC limit values, EC guide values, and WHO air quality guidelines; this confusion is present also in the paper of Sunyer *et al*[1] to which Dr Britton refers.

EC limit values and guide values are often expressed in terms of percentiles, with which individual measurements should not be compared. For example, the EC limit value for

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nitrogen dioxide is defined as: "The 98th percentile of hourly means should not exceed 104·6 ppb." The underlying reason for specifying an upper percentile rather than an absolute limit is that extreme values are liable to be erratic, occurring in a highly local or transient manner or even as a result of instrument malfunction. In one year there are 8760 hours; the 98th percentile of hourly means is defined as the 176th highest hourly mean measured. Only if this exceeds 104·6 ppb is the relevant ED directive contravened. The limit value, designed to safeguard health with regard to exposure to nitrogen dioxide, was set with knowledge of the likely log-normal distribution of hourly mean concentrations. Though hourly means could exceed 104·6 ppb on 175 occasions per year, with occasional maxima up to three or four times that value, damage to health would not be expected.

In discussing WHO air quality guidelines for sulphur dioxide and smoke Dr Britton fails to point out that safety factors of 2 for effects on morbidity and mortality and of 1·5 for decrements of lung function were included when the guidelines for combined exposure to sulphur dioxide and particulate matter were defined.[2]

It should also be noted that the data on which the joint guideline was defined did not permit separation of sulphur dioxide and black smoke in terms of effects; it would thus be more appropriate to quote the number of days when the joint guidelines were exceeded than each one separately. Furthermore, WHO guidelines for black smoke[2] have not been defined on an hourly basis and the 24 hour guideline is currently quoted[2] as 125 $\mu g/m^3$ rather than 100 $\mu g/m^3$. The figure of 100 $\mu g/m^3$ occurs in the World Health Organisation report of 1979.[3] In that report a range of 100–150 $\mu g/m^3$ was defined.

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The above are the opinions of the authors and should not be taken as representing those of the Department of Health.

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AUTHOR'S REPLY I thank Drs Maynard and Waller for their comments on my article, and their clarification of the various guidelines and limits on atmospheric pollution levels. I suspect that I am not alone in finding them a little confusing. The point I wanted to make in the article is that in the study by Sunyer *et al* (their ref 1) everyday variations in usual levels of atmospheric pollution had small but measurable effects on respiratory morbidity. The vast majority of this variation was well within all of the guidelines and limits. We need guidelines for monitoring and enforcing pollution controls, but if there was any doubt before it is surely now evident that pollution that does not contravene existing guidelines still causes respiratory morbidity. The question is whether this degree of morbidity is an acceptable or necessary price to pay for economic development and, if it is, how much society should be prepared to accept.

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Pulmonary function in chronic renal failure: effects of dialysis and transplantation

In their article (June 1991;**46**:424–8) Drs A Bush and R Gabriel referred to decreased carbon monoxide transfer (TLCO) in four groups of patients with chronic renal failure. Apparently, their TLCO values were corrected for haemoglobin, which was low in most of their patients. Thus the decreased TLCO should be attributed to causes other than a low haemoglobin pool. The authors speculated that it was due to interstitial lung fibrosis, caused by chronic subclinical oedema.

These authors' results differ from those of others. TLCO is usually reduced in uraemic patients because of coexisting anaemia; adjusting to a normal haemoglobin concentration therefore produces normal TLCO values.[1 2] In the population they studied, however, the underlying cause could not be determined from their data. Interstitial fibrosis with resulting deterioration of the membrane TLCO component (Dm) is the only possible factor. The authors did not perform chest radiography, which could have helped to resolve the issue. Moreover, there are even more sensitive tools for such evaluation—specifically high resolution computed tomography and a separate determination of Dm and the vascular component of the TLCO: pulmonary capillary blood volume (Vc) and the reaction rate of carbon monoxide with haemoglobin (theta, θ). According to the theory of Roughton and Forster—expressed as $1/TLCO = 1/Dm + 1/\theta Vc$—$\theta$ increases with increased haemoglobin. Without measuring the components of TLCO therefore, even after adjustment for haemoglobin, one cannot determine whether Dm or Vc predominantly affects the overall TLCO. There are also reports that haemodialysis (their group 3) per se affects TLCO,[1] which was not considered by the authors. In the sample that we studied[1] the effect of haemodialysis was a reduction in TLCO of about 10%, due entirely to a decrease in Vc of about 20%, which we attributed to a decrease in blood volume with consequent reduction in Vc.

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1 Dujić Ž, Tocilj J, Ljutić D, Eterović D. Effects of hemodialysis and anemia on pulmonary diffusing capacity, membrane diffusing capacity and capillary blood volume in uremic patients. *Respiration* 1991;**58**:277–81.
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AUTHOR'S REPLY We thank Professor Dujic and colleagues for their interest in our paper and for raising some interesting points. Our findings of a reduced TLCO even after correction for anaemia is in accord with the findings of other workers[1 2] not cited in their own paper.[3] We agree that the pathological cause of the physiological abnormalities could not be determined; we did not think that additional radiography was justified when we designed the study, but agree that in the light of our findings quantitative computed tomography[4] would be of great interest. The issue of the meaning (if any) of attempting to measure Dm and Vc separately has been debated at length elsewhere.[5 6] The theoretical problems include non-uniform DL/VA in the real lung, the dangers of extrapolating back to the intercept beyond the data points, and the profound effects that oxygen itself has on the pulmonary circulation, altering the variable one is trying to measure. There is also experimental evidence that Dm is insignificant.[7] Thus we believe that TLCO does indeed measure the amount of blood (or, strictly, haemoglobin) within the pulmonary capillary bed. We did consider in detail the acute effects of haemodialysis on the lungs[8 9]; the acute fall in TLCO takes place early in the dialysis, and it has reverted almost completely to predialysis values by the end of a six hour dialysis. We studied our patients within 24 hours of dialysis, to try to minimise the effects of accumulation of lung and body water between dialyses. Our final speculations are confirmed to some extent by pathological studies[10]; we agree that a new computed tomography, biopsy, or necropsy study is needed to determine the underlying pathology.

A BUSH

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Haematological effects of inhalation of *N*-formyl-methionyl-leucyl-phenylalanine in man

We read with interest the description by Dr M J Peters and colleagues (April 1992;**47**:284–7) of temporary peripheral blood leucopenia, in particular neutropenia, immediately following the inhalation of the tripeptide *N*-formyl-methionyl-leucyl-phenylalanine (FMLP) in normal subjects, accompanied by activation of peripheral blood neutrophils as measured by chemiluminescence.

We agree that the likely mechanism is

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