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SIR,—It has recently become fashionable to question the value of continuous treatment with β agonists in asthma. Constant P van Schayck and colleagues' study is one of few studies supporting a nihilistic attitude towards this principle of treatment and extends it to other bronchodilators.[1] The authors conclude that bronchodilators should be used only on demand, with additional corticosteroid treatment if necessary. The study, however, shows a very small decline in forced expiratory volume in one second (FEV_1) in the continuously treated groups (salbutamol and ipratropium bromide). This decline borders on significance ($p=0·05$) when confounding factors are considered, and it is stated that the decline was 0·029 (SE 0·036) l/year less during the year in which salbutamol was used than during the year in which ipratropium bromide was used; this must mean that no significant decline occurred during salbutamol treatment (the combined analysis showed a decline of 0·072 l/year during continuous treatment and 0·020 l/year during treatment on demand. Did the statistical power of the study really permit the inference that the two drugs had equal effects in this respect?

We also believe that there are methodological problems with the study: firstly, a fairly heterogeneous group of patients was studied, with about two thirds having chronic bronchitis; secondly, the drop out rate was high as only 144 out of 223 patients were included in the key analysis; and, thirdly, baseline FEV_1 in the groups receiving continuous and on demand treatment differed more than did the yearly changes observed (approximately 0·2 litres in favour of the group receiving on demand treatment). The only possible difference with regard to histamine sensitivity was a transiently reduced sensitivity in patients with asthma treated on demand. This does not seem logical.

The authors' main conclusion, that continuous treatment should not be used, is thus not supported by convincing data. A study by Sears *et al*, which is quoted in support, cannot be properly evaluated owing to a lack of primary data in the published paper.[2] Current opinion in Sweden and other countries favours the use of continuous treatment with β agonists only in combination with inhaled steroids. Thus van Schayck and colleagues' main conclusion is based on weak data from a study not designed according to presently accepted treatment strategies. Their warning against using long acting β stimulants (see their discussion) seems even more far fetched: they were not even studied.

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1 Van Schayck CP, Dompeling E, van Heerwarden CLA, Folgerin H, Verbeek ALM, van der Hoogen HJM, *et al*. Bronchodilator treatment in moderate asthma and chronic bronchitis: continuous or on demand? A randomised controlled study. *BMJ* 1991;**303**:1426-31. (7 December.)
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AUTHORS' REPLY,—In their letter[1] about our article[2] C J Hilton and R W Fuller report an improved FEV_1 in 55 patients who received 200 μg salbutamol regularly for 12 months. They claim that continuous use of salbutamol does not decrease lung function. We wonder what daily dose of salbutamol these 55 patients actually received. Our 83 patients who were treated on demand used an average daily dose of 240 μg salbutamol for two years. The decline in FEV_1 was only 0·020 l/year. The 61 patients who were treated continuously in our study received 1600 μg salbutamol daily for two years and had a decline in FEV_1 of 0·072 l/year ($p=0·05$). We assume that the 55 patients reported on by Hilton and Fuller received considerably less than 200 μg salbutamol eight times a day. To support the claim that regular use of salbutamol alone does not worsen the disease a randomised comparison should be made with treatment on demand, preferably over a period long enough for effects on the decline in lung function and not the immediate effects of giving the drugs to be studied.

Hilton and Fuller suggest that the difference between their and our findings may be related to the effect of stopping anti-inflammatory drugs. Previous treatment was not, however, a confounder in our randomised trial. The patients who stopped using anti-inflammatory drugs were equally distributed over the two treatment regimens. Hilton and Fuller further suggest that our results can be explained by more severe asthma in our continuously treated patients, but the decline was corrected for potential confounding variables such as initial FEV_1 and symptoms. After this correction the decline in continuous treatment remained three to four times greater than that in treatment on demand. The estimated influence (β) of stopping anti-inflammatory drugs on the decline in lung function in patients treated continuously (−0·015 l/year) was comparable with that in patients treated on demand (−0·016 l/year).

In Hilton and Fuller's study the number of patients who dropped out seems comparable with the number in our study who used an average dosage of 240 μg salbutamol daily and dropped out after 12 months: eight out of 63 (13%) in their study versus 14 out of 110 (13%) in our study.

Andy Lawton and Maria Teresa Lopez-Vidriero are probably unaware of our other article, which shows the influence of, for example, bronchial hyperresponsiveness on decline in lung function.[3] This study was carried out in the same study population as that used in our study reported in the *BMJ*.[2] The two groups of patients—51 asthmatic patients and 93 patients with chronic bronchitis—were analysed separately, and thus each group was homogeneous. There were similar intervals of six months between measurements, and FEV_1 was always measured at exactly the same time of the day to avoid diurnal variation. Bronchodilator drugs were stopped for at least eight hours before the start of the measurements.

Our article shows that the measurements of FEV_1 clearly fit a linear model. This model explained a variation of more than 70%. We did not use autoregression analysis except afterwards to reanalyse our data. In doing this we took only equally spaced time points.

We are surprised that Kjell Larsson and Paul Hjemdahl consider the decline in FEV_1 in the continuously treated group to be very small. The crossover design for the two drugs and the parallel design for the two treatment regimens does not allow a simple comparison as suggested. Both drugs were given to all 144 patients for one year and compared within patients. There was no significant difference in the decline in lung function between the two drugs ($p=0·41$).

Only 23 patients dropped out from the study for reasons unrelated to the drug treatment, such as lack of motivation. This is low for a two year study. Forty patients dropped out because the treatment with bronchodilators was not sufficient. In this group twice as many patients were treated continuously. This is an important finding.

Our findings seem to support the current opinion in Sweden that continuous β_2 agonists should be used only in combination with inhaled steroids. We showed that patients receiving continuous bronchodilator treatment were unaware of an increased decline in lung function. Therefore we suggested that continuous bronchodilation without anti-inflammatory treatment masks the decline in lung function and suppresses the subjective need for additional anti-inflammatory treatment. As long acting β_2 agonists seem even more effective in suppressing symptoms such as morning dyspnoea we suggest that patients may be more misled by the apparent wellbeing produced by these long acting bronchodilators.

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1 Hilton CJ, Fuller RW. Bronchodilator treatment in asthma: continuous or on demand? *BMJ* 1992;**304**:121. (11 January.)
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Coronary heart disease

SIR,—J McMurray and H J Dargie put forward a compelling case for including heart failure in the initiative *The Health of the Nation*.[1] They point out that the Framingham study shows that the annual incidences of heart failure in subjects aged 65 and over and subjects aged under 65 are only slightly lower than those of myocardial infarction and higher than those of stroke. The Framingham study was begun in 1949 and refers to an American population in which the causes (particularly hypertension) and the treatment of heart failure were different from those today. There is a dearth of epidemiological information on heart failure not only in the United Kingdom but throughout the world, largely because epidemiologists have concentrated on coronary heart disease manifest by sudden death, myocardial infarction, or angina. We recently studied the prevalence of heart failure in three general practices[2] and the impact of heart failure on workload in a district general hospital.[3]

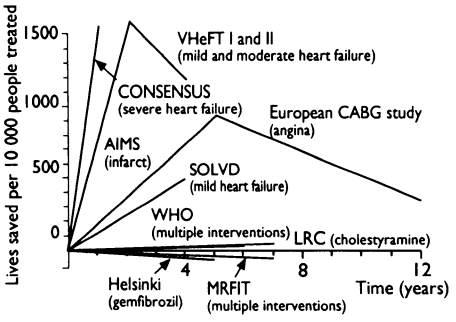
The prevalence of heart failure in a population of 30 204 people in north west London was 0·4%.[2] The prevalence was 0·06% in those aged under 65 and 2·8% in those aged 65 and over (mean 73). Heart failure was determined by an analysis of prescriptions for diuretics and a clinical definition. Hypertension at any time was identified in only 6% of those with heart failure.

In Hillingdon Hospital, which serves roughly 155 000 patients, 2877 patients were admitted to the medical and geriatric services over six months.[3] Of these, 140 had heart failure as the main reason for admission, of whom 15 had heart failure as a complication of myocardial infarction. Twenty nine patients were aged under 65. Sixty two patients died within one year of admission. By comparison, during the same six months 89 patients were admitted to the coronary care unit with acute myocardial infarction and 52 with unstable angina. Of the patients with myocardial infarction, 55 were aged under 65. A few patients with these conditions might have been admitted directly to the wards, particularly the geriatric wards.

In his response to McMurray and Dargie, Hugh Tunstall-Pedoe is reticent about the importance of heart failure for four reasons.[4] Firstly, the main problem is in patients over the age of 65; that is

true, but the clinical problem below that age is still just over half that of myocardial infarction. Secondly, he says that mortality statistics underestimate deaths from heart failure, but he correctly attributes that to the method of classification.

Thirdly, he says that heart failure contributes as a "pathological mechanism"; so does atheroma in the coronary arteries. The fundamental cause of atheroma still remains obscure. Finally, he points out that the epidemiology of heart failure has been largely ignored because of difficulties of definition; it is quite possible to agree a definition such as the one we have used in our work. None of these arguments are persuasive reasons for ignoring an important cause of morbidity and death.



Lives saved per 10 000 patients treated for varying periods in several studies. CONSENSUS (the cooperative north Scandinavian enalapril survival study), VHeFT I and II (Veterans Administration cooperative vasodilator heart failure trials), and SOLVD (studies of left ventricular dysfunction) were trials of enalapril in heart failure of decreasing severity. AIMS (the anistreplase intervention mortality study) tested the efficacy of thrombolytic treatment in myocardial infarction. The European CABG study (coronary artery surgery study in stable angina) investigated the benefits of cardiac surgery in patients with angina pectoris. MRFIT (the multiple risk factor intervention trial) and the WHO (World Health Organisation) study were trials in which several risk factors for coronary heart disease were modified. The LRC (Lipid Research Clinics) trial (of cholestyramine) and the Helsinki trial (of gemfibrozil) assessed the value of lipid lowering drugs in reducing mortality

Heart failure is common, causes premature death, carries a poor prognosis, consumes considerable hospital resources, and can be identified in the population, and effective prevention and therapeutic interventions are available.[5,6] Heart failure fulfils all the criteria for inclusion in *The Health of the Nation*. It is being neglected because of a belief that prevention of the main cause, coronary heart disease, is more appropriate, although current evidence suggests that proper treatment of heart failure applied to the whole population would be more effective in terms of mortality and possibly morbidity (figure). These are not mutually incompatible approaches. Proper emphasis on coronary heart disease in *The Health of the Nation* should allow interventions other than primary or secondary prevention of coronary heart disease to be included.

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Action Asthma: privatising the airways?

SIR,—A recent press release described Action Asthma as "an educational initiative launched [in January 1991] . . . to improve the management of asthma through educational programmes with hospital doctors, GPs, practice nurses and patients." It is guided by 11 doctors with a special interest in asthma. So far a national survey on asthma has collected information on how 61 000 patients feel about their asthma. There is a national telephone asthma helpline, and a leaflet with 10 questions "to ask your doctor" is being distributed. The leaflet carries the logo of Allen and Hanburys, with the company's name and the slogan "Confidence for living with asthma." The company's name appears on all Action Asthma's material, and its inhaled product packs contain a form inviting patients to enrol in the Action Asthma patient service, which offers written information and advice. Almost 180 000 patients have enrolled.

Several issues arise. Though no criticism can be made of Action Asthma's worthy aims and motives, the likely effect would seem to be the development and strengthening of patients' and doctors' loyalties to one company. This may not be in their interest or in the interest of the NHS: decisions about treatment should be independent of company loyalty. It is also, in my view, unfair to competing firms.

It would be healthier if good causes did not provide commercial benefits for their sponsors. Such relations are liable to tarnish their image.[1] If other companies, not necessarily concerned with health, had joined Allen and Hanburys in supporting Action Asthma it would be less controversial.

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1 Erlichman J. Charity logo deal breaks new ground. *Guardian* 1991 Sep 14:3.

⁂We sent this letter to Action Asthma, who replied as follows.

SIR,—As Andrew Herxheimer states, Action Asthma is guided by a group of respiratory physicians and general practitioners with an interest in asthma. We were pleased to accept Allen and Hanburys' invitation to develop an educational service for patients, nurses, and doctors. The aim of the service is to complement the many other initiatives to reduce suffering from asthma. We believe that various strategies are needed if that goal is to be achieved. Since its launch in September 1990 Action Asthma has provided a range of educational aids and materials and arranged various meetings for doctors and nurses.

The chairman of the education committee of the National Asthma Campaign has attended Action Asthma's programme development board, and similar initiatives from other pharmaceutical companies, to ensure that advice is standardised and duplication of effort avoided. At the first meeting there was a commitment that the materials would not be promotional and that Action Asthma's most successful educational activities would be offered to the National Asthma Campaign for continuation.

Herxheimer suggests that the aims of Action Asthma are tied in to developing patients' and doctors' loyalties to one company. This is not the case. Action Asthma's services support and encourage the guidelines for management and treatment proposed by the British Thoracic Society. We do not promote specific drug treatment produced by Allen and Hanburys or any other pharmaceutical company. We refer only to types of treatment—"relievers," "preventers"—and the way in which they work. Membership of the Action Asthma patient service is open to all patients regardless of the treatment they receive.

Herxheimer's general concern about the funding of postgraduate education and patients' education is an important issue that extends well beyond respiratory disease. In the current climate, however, when the NHS has limited resources and the government offers little funding specifically for education about asthma, financial support for health education continues to be needed. At present this gap is often filled by pharmaceutical companies. We believe that Action Asthma's materials have been of considerable benefit to the patients who use them.

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Blood glucose concentrations and progression of diabetic retinopathy

SIR,—Olaf Brinchmann-Hansen and colleagues, reporting the progression of diabetic retinopathy at seven years in the Oslo study, suggest that improved glycaemic control is of benefit.[1] Their key data are presented in table III, which gives the score for the severity of retinopathy (mean (SD) numbers of microaneurysms and haemorrhages) at the beginning of, and seven years into, the study. The data are broken down arbitrarily into three groups: patients with "blood glucose concentration" (actually percentage glycated haemoglobin (HbA_1)) of <9·0% (20 patients), 9·1-10·0% (13 patients), and >10·1% (12 patients) at the examination at seven years.

† Testing of these data, however, yields no difference in the severity of retinopathy at seven years between the groups with HbA_1 concentrations of <9·0% and 9·1-10·0% or between the groups with concentrations of 9·1-10·0% and >10·1%. A significant difference ($p=0·021$) exists solely between the groups with HbA_1 concentrations of <9% and >10·1%, but this difference is, in principle, eroded by the Bonferroni correction required for situations of multiple comparison. Spearman's or Pearson's correlation of the seven year HbA_1 concentration with the severity of retinopathy (which would have been a more useful test of the hypothesis than a statistical procedure based on arbitrary subdivisions) was unfortunately not given, but log linear regression analysis of the risk of retinopathy (table V) seems to enter only the difference in mean HbA_1 concentration between the start of the study and at seven years. The authors also show that retinopathy at the beginning of the study was not correlated to HbA_1 concentration at that time. As individual severity of retinopathy at seven years correlated powerfully with retinopathy at the start of the study it seems that retinopathy is more closely related to the inherent severity of diabetic disease than to the long term effects of hyperglycaemia.

The Oslo study was originally established to determine the effects of insulin pumps, multiple insulin injections, and conventional treatment on the progression of complications. At the beginning of the study 45 patients were randomised into three equal groups: 15 used insulin pumps, 15 had a multiple injection regimen, and 15 received two insulin injections a day. At seven years, however, only 10 used the pump, 29 used multiple injections (insulin pens), and only six were using conventional treatment. These groups are not analysed separately for progression of retinopathy, and it is not clear, from the data presented, how these groups