Location	House dust ($\mu\text{g/g}$)	Soil ($\mu\text{g/g}$)	Estimated % of children with blood lead	
			> 10 $\mu\text{g/dl}$	> 25 $\mu\text{g/dl}$
Mean value in national survey†	561	266	26.9	0.5
Range	5-36 900	13-14 100		
London	1010	654	55.5	3.9
Range	5-36 900	60-13 700		
Birmingham	424	313	21.1	0.3
Derbyshire: old mining villages	1870	5610	97	50.1
Range	606-7020	1180-22100		
Brighton houses:				
Built 1870-1919	1874	1014	81.1	14.5
Built 1960-1986	241	131	8.3	<0.05

*Measurements in Brighton were taken in September 1986.

†Including London, but excluding old mining areas.

Source for dust and soil samples: ref 4. Dietary lead intake assumed to be 15.71 $\mu\text{g/day}$ on basis of figures provided by Ministry of Agriculture, Fisheries and Food.¹ Water lead concentrations were conservatively assumed to be on average 4 $\mu\text{g/l}$ and ambient air lead concentrations to be 0.1 $\mu\text{g/m}^3$.

consistently denied that British children are at risk from this source of exposure to lead, and we can find no evidence that survey work is taking place.³ Research suggests, however, that the concentrations of lead in dust and soil in Britain may be as high as, or higher than, those found in the United States.⁴ If the blood lead concentration was lowered to 10 $\mu\text{g/dl}$ changes would have to be made in the methods by which buildings constructed before 1945 are maintained and re-decorated; after that date the use of leaded paints in Britain diminished rapidly.

Despite the lack of available data on blood lead concentrations in Britain we have used the US Environmental Protection Agency's computer model of the uptake and biokinetics of lead to estimate probable concentrations in children aged under 6 years by inference from data on the levels of environmental contamination (integrated exposure uptake biokinetic model for lead in children, version 0.99d). The results, given in the table, suggest that appreciable numbers of young children have raised blood lead concentrations and that adverse effects are therefore occurring.

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Authors' reply

EDITOR,—We endeavoured to include all relevant epidemiological studies in our meta-analysis and believe that others expert in this field would object to Herbert L Needleman's proposed deletions. The European multicentre study was one publication with consistent methodology relating to field work in several countries. In this complex epidemiological field, methodological concerns exist for many of the studies. In our view this is not best handled by deleting studies.

Regrettably, we had to exclude the two studies that measured circumcupal dentine lead. They cannot be combined with other tooth lead studies, and one study's regression results were not available. This left insufficient material for systematic review.

Publication bias is of concern with all systematic

reviews. While Needleman's "file drawer analysis" is interesting, it should be noted that even a few negative unpublished studies inevitably contribute bias to inflate the magnitude of observed association. We appreciate Needleman's observations on selection biases in the Boston studies and regret that more studies do not undertake such inquiries.

Needleman's concern about whether covariates are confounders or partial surrogates for body lead burden is a valuable point. Besides simply debating the issue, however, it seems impossible to quantify such "shared variance."

Needleman's lack of enthusiasm for reverse causality is not surprising. But intelligence clearly affects children's behaviour, and how this contributes to differentials in lead uptake is a serious point. For instance, in Boston, who knows if children of exceptionally high IQ play outdoors less than others?

While animal studies contribute to biological plausibility, they cannot directly help in determining the existence and magnitude of neuropsychological links to low level exposure to lead in children.

We acknowledge that Needleman is more convinced by the observational evidence than we are. When inferring causality we argue that the studies of lead and IQ are on weaker ground than studies in many other areas of epidemiological inquiry. Full scale IQ is a marker only for less identifiable specific potential neuropsychological deficits, other determinants of IQ are difficult to quantify, their role as confounders is unclear, the markers of body lead burden contain substantial measurement error as regards true exposure, and the statistical magnitude of any putative causal relation is small. This is not a satisfying area for deriving firm causal conclusions, which is why debate continues so long. Far from being "worn caveats," we think our caution in interpreting statistical associations is an appropriate stance for scientific investigation.

Hence, in answer to Erik Millstone and John Russell, we do not acknowledge that there is convincing evidence that blood lead concentrations down to 10 $\mu\text{g/dl}$ produce adverse neurological effects in children. Such causal inference relating to such a specific threshold of effect is not well founded.

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Emergency care in general practice

EDITOR,—Andrew Dicker's assertion that "there is an urgent need for a nationwide reactive and effective emergency service, staffed by properly trained paramedics, to respond to medical emergencies at home" encouraged us to collate and analyse some relevant data that we have been collecting.¹ We wrote to all chief ambulance officers in the United Kingdom seeking their views on tertiary education for paramedics. To date, 44 (86%) of 51 have responded. Forty supported the introduction of a nationally recognised degree or diploma in ambulance technology, while the concepts of a General Paramedical Council and state registered paramedics were both supported by 39. Reaccreditation and the provision of a national qualifying exam were both supported by 35 respondents. There was not support, however, for the regulation of entry numbers to protect career progression or for the suggestion that people should not be able to hold senior management positions in the service unless they had a paramedic degree.

These data show a willingness by the leaders of the ambulance service to formalise the acquisition of skills and to professionalise the service, thus bringing the United Kingdom into line with other countries such as the United States and South Africa. We believe that such developments should be encouraged. C Brittain and colleagues' statement that "other health care professionals such as those in the ambulance service... are only too keen to take on this work to enhance their status" puts them in danger of being accused of professional protectionism.² We are sure that this is not the position of the British Association for Immediate Care and that, like us, it sees the best interests of patients being served by the coexistence of a highly trained and skilled paramedic service with general practitioners who have benefited from the excellent courses that it and others run.^{3,4}

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Nicotine patches

EDITOR,—May R Arnaot suggests that nicotine patches may not be safe.¹ I agree that nicotine could aggravate cardiovascular disease through the haemodynamic consequences of sympathetic neural stimulation or systemic release of catecholamines, or both.² I believe, however, that the risk of treatment with nicotine patches is negligible compared with that of cigarette smoking.

Cigarette smoking delivers many potentially toxic chemicals, including carbon monoxide. It produces a hypercoagulable state, which is thought to be a primary mechanism for acute cardiovascular events related to smoking. Use of nicotine patches seems not to enhance coagulation.³ Even though nicotine is potentially toxic, the

risks of nicotine replacement treatment are likely to be much less than those of cigarette smoking for several reasons.⁴ For most smokers the doses of nicotine obtained by cigarette smoking exceed those delivered by currently marketed nicotine replacement treatments. The cardiovascular effects of nicotine are more intense when delivered rapidly by cigarette smoking than when delivered more slowly, as by nicotine patches. The relation between the dose of nicotine and cardiovascular response is flat, such that the effects of cigarette smoking combined with nicotine from other sources are similar to those of cigarette smoking alone.⁵ At a minimum, the risk of nicotine replacement treatment is likely to be much less than that of cigarette smoking, even in patients with coronary heart disease. These risks are substantially outweighed by the potential benefits of the increased likelihood of stopping smoking by using nicotine replacement treatment.

With respect to the case reports, relatively few adverse cardiovascular events have been reported despite millions of patients having used nicotine patches, many of whom are presumed to be at considerable risk of acute cardiovascular events. Any middle aged or older person who has smoked long term is at increased risk of myocardial infarction or sudden death; and some of these events are bound to occur by chance in the first few weeks or months after that person has stopped smoking, whether or not nicotine patches are used.

I agree that further research into the risks of nicotine replacement treatment in patients with cardiovascular disease is needed. Meanwhile, I would much prefer my patients with coronary heart disease to use nicotine patches than to smoke cigarettes.

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*Professor Benowitz has been a consultant to many of the pharmaceutical companies that manufacture or market nicotine replacement products, including nicotine patches.

Limitations of randomised controlled trials

EDITOR,—T C B Dehn has commented on the problems of accruing patients into randomised controlled trials, noting the understandable reluctance of patients to enter some trials ("I want the best treatment, doctor") in which the patient's outcome (for example, time to death) is the main end point and, consequently, the patient is unable to benefit from the study directly.¹ This raises a fundamental issue concerning the usefulness of randomised controlled trials in reliably identifying new clinical knowledge. Less recognised, but equally important, is inconsistent clinical enthusiasm to offer the study to all patients presenting during the study period, which results in patient selection bias.

The large bowel cancer project's randomised

controlled trial recorded the presence of such an "eligible but not randomised" subpopulation, the proportion of which varied among participating clinicians.² Furthermore, the clinical outcome in this group differed from that in the trial's control arm, confirming the problem of selection bias. This factor, and other forms of bias, limit the value of randomised design for prospective studies.³ If, however, we are committed to generating reliable new knowledge using the format of randomised controlled trials then the clinical scientists conducting these studies, as well as the grant giving bodies supporting them, should recognise the existence of patients who are eligible but not randomised in all such trials.

We propose two strategies to address this problem. Firstly, all patients with the diagnosis of interest in the study should be documented and followed up whether or not they become part of the randomised controlled trial. Secondly, after a positive study result has been obtained documentation should continue during the implementation phase of the new treatment to show that the expected positive outcome for the population at large does occur. Without such post-trial surveillance it is difficult to know that the predicted value of the study materialises in an unselected population. In addition, the low rates of unexpected sequelae (either complications or additional benefits) may not be recognised. This additional documentation should become part of the plans for the randomised controlled trial. Pollock concluded that consecutive audit has value in this context.⁴

The high cost of randomised controlled trials (money, resources, and professional commitment) and the trust that patients place in us when they enter such trials mandate that all efforts are geared to improving patients' care. An increased awareness of the group in randomised controlled trials who are eligible but not randomised, the need for post-trial surveillance, and the need for eventual audit will help the process of responsible implementation of new treatment. These issues not only are the responsibility of clinical investigators but should be crucial concerns for funding leaders and medical publishers.

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Childhood antecedents of schizophrenia

EDITOR,—Andrew D Paterson is correct to point out the abundant evidence of a genetic contribution in the aetiology of schizophrenia, but I take exception to his conclusions regarding treatment.¹ He makes a basic error in assuming that just because a biological factor can be shown to be of primary importance in the aetiology of a condition then it must follow that suitable treatments must also be limited to the biological. Even if, as seems the case, environmental factors are of minimal

importance in the aetiology of schizophrenia, we must not neglect the psychological and social aspects of management. We should remember, for example, that it is a simple dietary intervention that is required in phenylketonuria, an autosomal recessive disorder whose genetics are far better understood than schizophrenia.

Paterson's statement that "it is questionable whether psychotherapeutic intervention will have any influence on the expression of the disease" is surprising in the light of the fact that in his previous paragraph he quoted McGuffin *et al* as stating, "there is good evidence that psychosocial factors, such as high expressed emotion at home or life events, can hasten relapses or precipitate onsets."² Surely it is now time to move on from this Platonic dualism separating soul and body, mind and brain, to a medicine that recognises and treats the whole person with whatever interventions prove to be effective.

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Palliative care in terminal cardiac failure

Hospices cannot fulfil such a vast and diverse role

EDITOR,—I agree with Sarah Jones's comments about the neglected needs of patients with terminal cardiac failure¹ and the fact that the poor prognosis is not appreciated as being the same as that in cancer.² Ironically, this benefits patients with cardiac failure, who are spared the taboos of cancer, its particular stresses, and the distancing of health professionals. The infrequency of requests to hospices regarding cardiac failure could reflect the ability and willingness of hospitals and primary care teams to continue care. The original need for hospices was to redress this imbalance.

Palliative care for all is an admirable goal, and palliative medicine's basic principles apply to those dying of any illness. It is naive, however, to pretend that present hospices could fulfil such a vast and diverse role.

Hospices offer intensive specialist input, historically to patients with cancer plus motor neurone disease and multiple sclerosis and more recently to those with HIV infection. Patients are seen at any time of their illness according to need, with end stage care being only part of hospices' role. Hospices' skill lies in actively controlling symptoms and providing support through day centres, outpatient visits, and short inpatient stays averaging 14 days,³ with discharge in most cases. This does not qualify hospice doctors as specialists in the many other terminal diseases that need hospice-type care. Nor are hospices in a position to offer medium term or long term care, which is a common request for patients with non-malignant diseases. As with cancer, this would be a waste of resources, and provisions are made elsewhere.

Over three quarters of hospice beds are funded by charities and bound by mission statements, which may single out cancer for individual attention. The reason for donations must be honoured.

Palliative medicine is acknowledged to be advancing rapidly while concentrating on this specific range of patients⁴; broadening its remit would dilute the benefits of specialising and hence slow progress. The hospice philosophy provides staff with a clear approach in a stressful specialty. This may be less appropriate for diseases with