Nearly half the sample favoured maintenance prescription of injectable opiates. The inevitable outcome of such practices is illicit selling on of drugs, with accidental overdoses and deaths. Indeed, much prescribed oral methadone ends up on the streets and already seems to play a part in accidental deaths.

The emergence of "dual diagnosis" patients, with both mental disorder and substance misuse, highlights the importance of psychiatric input to drug services.

Although the *Health of the Nation* recommends consultation with users and carers, it does not suggest that services should be designed solely to satisfy consumers' preference or convenience.⁵ Planning of services should be based on good evidence and sound clinical practice.

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Methadone maintenance treatment

EDITOR,—Our review of methadone maintenance treatment¹ elicited a range of views.² Wolfgang Poser and colleagues correctly comment that methadone treatment protects against death due to an overdose of opiates³—an effect that we have reviewed elsewhere.⁴ The problem of deaths due to overdoses of diverted methadone, however, is considerable in Britain and Denmark⁴ and merits further work.

Diversion of methadone is an important if underrecognised problem in Britain. It indicates poor quality treatment and can probably best be addressed through policies and guidelines on the supervision of consumption of methadone, facilities for consumption off site, and attention to lack of compliance with treatment. The issue of compliance is important and poorly studied in Europe.⁴ Kim Wolff and colleagues advocate regular measurement of plasma methadone concentrations.² We do not agree that this is the most direct method of addressing diversion of methadone. The role of urine and blood analysis in addressing poor compliance is unclear, and such tests constitute a substantial cost burden for service providers. On balance, we suspect that the cost of these procedures outweighs the benefits, but further study is needed to clarify this issue.

Frederick H Meyers's comments are not consistent with the research literature, and he cites no evidence to support his opinions.² Steve Kempney² and N J Shaw² express concern about methadone withdrawal in neonates. Methadone treatment for pregnant women who are dependent on heroin has been shown to protect maternal and neonatal wellbeing.⁵ Withdrawal of neonates is often substantially complicated, however, by mothers' use of more than one drug—for example, cocaine and benzodiazepines.

On balance, methadone treatment does have drawbacks, like many interventions in medicine do. Nevertheless, overall, properly delivered methadone treatment confers important benefits

for people who are dependent on opiates and for the community.

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Environmental lead and children's intelligence

Studies included in the meta-analysis are not representative

EDITOR,—Stuart J Pocock and colleagues' meta-analysis of 26 studies of asymptomatic exposure to lead in children concludes that "the evidence strongly supports an inverse association between body lead burden and child IQ."¹ The authors then urge caution before the relation is accepted as causal. They do this on the basis of the following considerations: Are the studies representative? Is confounding adequately dealt with? Is selection bias at work? Is causality reversed: does low IQ cause ingestion of lead?

These questions have been asked and answered before. The criticism of non-representativeness could readily be applied to Pocock and colleagues' analysis, which includes five studies reporting null effects. Three (in Bucharest, Budapest, and Sofia) have not been published independently in peer reviewed journals.² Two had major methodological difficulties severe enough to disqualify them. In one study of small sample size (n=135) half of the mothers of the infants misused a potent neurotoxin, alcohol.³ Another study had serious difficulties in analysing blood lead concentrations and was forced to change the analytical method in the middle of the investigation.⁴ On the other hand, two tooth studies that showed significant effects were excluded for arbitrary reasons.

Pocock and colleagues suggest that there may be many other unpublished and undiscovered studies that find no effect of lead and mention a single case in point. A non-anecdotal way to deal with this question is to estimate the number of unpublished null studies required to vitiate the observed joint result. This technique, known as "file drawer analysis," has in fact been applied to lead.⁵ The number of unpublished null studies required is between 26 and 82.

In their discussion of confounding the authors ignore the phenomenon of shared variance and assign the decrease in effect size when confounders are controlled exclusively to the confounder. Confounders can have a direct effect on outcome; they can also operate through increasing lead concentration. Poverty affects cognition. It also results in families living in homes with higher lead

content and surfaces that have deteriorated. Lower parental intelligence and poor care taking, in addition to directly affecting the cognition of offspring, may lead to increased absorption of lead. Ignoring this property may result in a type II error.

Selection bias of subjects was specifically evaluated in the two Boston studies and shown not to be consequential.

The issue of reverse causality dissolves under close inspection. Umbilical cord blood lead concentrations, measuring the intrauterine lead burden, are associated with later IQ deficits. Subhuman primates systematically given small doses of lead show clear deficits in learning and attention. To dismiss these studies on the basis of species differences is to ignore one of Bradford Hill's canons of causal inference: biological plausibility. There is no clearer example than the coherence of these two groups of epidemiological and experimental investigations.

Modern critics of the hypothesis linking lead with cognitive deficit should be required to provide fresh reasons for their scepticism and not use worn caveats that have been amply addressed over the past decade and a half.

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Britain must replace its lead pipes to meet WHO standards for drinking water

EDITOR,—As Stuart J Pocock and Marjorie Smith have been members of official British advisory committees, their acknowledgement that there is convincing evidence that blood lead concentrations down to 10 µg/dl (0.48 µmol/l) produce adverse neurological effects in children should have important implications for public policy.¹ The United States government set a target maximum childhood blood lead concentration of 10 µg/dl in 1991, but the British "action level" has remained at 25 µg/dl (1.2 µmol/l). The British government should now lower its target to, or preferably below, 10 µg/dl and then take steps to meet that target.

The problem of raised lead concentrations in drinking water is widespread in Britain. In Blackburn, to cite an extreme example, about 20 000 households receive water containing greatly increased concentrations—some as high as 50 000 µg/l (24 mmol/l) or even 300 000 µg/l (144 mmol/l); in 1990 almost a fifth of samples analysed by British water companies had lead concentrations above 10 µg/l.² If drinking water in Britain is to meet the World Health Organisation's new maximum lead contamination limit of 10 µg/l the only practical solution will be the replacement of all lead pipes.

Too little attention is being paid in Britain to the problem of old leaded paint and the consequent contamination of household dust. The United States has a comprehensive strategy for identifying and reducing housing stock contaminated with lead. In Britain the Department of Health has

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 circumcuspulpal 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