port lead to low patient attendance at urban primary care emergency centres (p 1627).¹⁸ Such centres are never likely to become realistic alternatives to home visits in rural areas either.

The government has offered to reinstate a fixed allowance for night visits (scrapped by the 1990 contract) to be funded by the abolition of the two tier visit fee and replacing it with a single reduced fee of one fifth the current higher rate. Given a fivefold difference in rates of night visiting across Britain¹¹ and the diversity with which visits are currently accomplished—by principals, general practitioners from small or large rotas, or deputising services—the GMSC is right to reject proposals that were insensitive to performance and which would have unfairly benefited practices with low visiting requirements.¹⁹ The government's encouragement of out of hours general practitioner cooperatives amounts to no more than a diversion of funds already due to general practitioners. The

Department of Health's proposal for night visit pay is completely inadequate: a "nil cost option" that offers a pittance, however parcelled out as incentives and allowances, of 2.8p per patient per week.²⁰

The demands of the general practitioner's day now include health gain and health maintenance, health promotion and disease prevention, target hitting and budget balancing, and provision of sophisticated clinical care. The government must understand that most general practitioners, whatever their age, whether rural or urban, and whether single handed or in group practice, have finally had enough of the unamended night time contract of the 1940s. The "primary care led NHS" requires a properly funded, well organised night time service.

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Fire retardants, biocides, plasticisers, and sudden infant deaths

The message of the "back to sleep" campaign holds until the chemistry has been worked out

Recent television programmes linking the sudden infant death syndrome to the antimony added to the plastic of cot mattresses has concerned the public and health care professionals alike. Unfortunately, the programmes and their fallout have been stronger on opinion and invective than on accurate information. There is a danger that the message of the government's "back to sleep" campaign, which has been followed by a dramatic fall in sudden infant deaths in Britain in the past three years,¹ may be obscured by incomplete, inaccurate, and sensationalist reporting.

Barry Richardson, a consulting scientist, proposed in 1989 that fire retardants in cot mattresses might contribute to the sudden infant death syndrome.² The essential component of his hypothesis was that, under the right conditions of warmth and humidity and in the presence of traces of organic material (for example, from sweat or urine), certain fungi (such as *Scopulariopsis brevicaulis*) can metabolise constituents of infants' mattresses (phosphorus, arsenic, or antimony) and produce the highly toxic trihydrides—phosphine, arsine, and stibine. These trihydrides, by acting as anticholinesterases, might then kill infants by inducing cardiac or respiratory failure of rapid onset.

S brevicaulis is common and can degrade nitrogen-con-

taining compounds in organic material (for example, in meat, cheese, and leather), so that ammonia (nitrogen trihydride) is released. Phosphorus, arsenic, and antimony are—like nitrogen—in group V/Vb of the periodic table of the elements, and Richardson's hypothesis is that their trihydrides may be similarly produced. Such a degradation process was recognised in the 19th century as leading to deaths from arsine poisoning. (In damp conditions *S brevicaulis* degraded arsenic contained in wallpaper pigments and paste.³) Although cot mattresses do not usually contain arsenic, they commonly contain organophosphates and antimony trioxide, which are added during manufacture to the polyvinylchloride coverings (as plasticisers and fire retardants respectively). Although the foam and woven fabrics used in cot mattresses do not contain antimony, they often contain phosphates.^{3,4}

This hypothesis is compatible with many of the features of the sudden infant death syndrome in Western countries, particularly the association with the prone sleeping position and heavy wrapping, and with the pronounced falls in incidence when infants do not sleep prone.^{5,6} Stibine, being heavier than air, would be most likely to cause toxicity to infants sleeping face down under heavy wrapping. The association of the sudden infant death syn-

drome with social disadvantage and high maternal parity is explained by the suggestion that such families are more likely to use old mattresses (which are said to be more heavily colonised with *S brevicaulis*).

But the characteristic age distribution of infants dying of the sudden infant death syndrome (peaking between 2 and 4 months), the seasonal incidence (with, until recently, a pronounced winter peak), and the strong association with parental cigarette smoking are not well explained by this hypothesis. Haemolysis, the most common finding in acute stibine toxicity in adults,⁴ is not seen in the sudden infant death syndrome. In Richardson's laboratory, incubation of pieces of polyvinylchloride from mattresses with *S brevicaulis* on malt agar plates has been shown to produce colour changes in silver nitrate indicator papers, which he claims indicates the presence of stibine.

The Turner committee, set up in 1990 to investigate this hypothesis, commissioned studies in the laboratory of the government chemist; attempts to replicate these observations were unsuccessful.⁴ A controlled study commissioned by the Foundation for the Study of Infant Deaths (C F Simpson, personal communication), closely following Richardson's methods, failed to show evidence of production of stibine. While the studies in the laboratory of the government chemist have been criticised on methodological grounds, there has to date been no independent replication of Richardson's observations (B Richardson, personal communication).

Antimony's role

The most recent data produced in support of the hypothesis consists of the television description⁷ of concentrations of antimony in postmortem liver and serum from infants who were said to have died of the sudden infant death syndrome and from control infants who died of other causes, including trauma, infections, prematurity, and malformations (S Variend, personal communication). These data have not been published, but a summary of the results has been made available to us (A Taylor, personal communication) and shows detectable concentrations of antimony in the livers of 22 of the 41 children dying of the sudden infant death syndrome compared with only one of the 15 controls. Liver and blood concentrations correlated poorly; the infant with the highest liver concentration had no detectable antimony in the blood.

No evidence exists that the concentrations that have been found are toxic, and the presence of antimony in liver suggests long term rather than short term exposure. Published results from women occupationally exposed to antimony have shown urine concentrations more than 1000 times higher than the serum concentrations observed in these infants.⁸ Several of the control infants died in hospital and may have received supported ventilation, which may have reduced environmental exposure. The presence of antimony in infants' hair⁹—if confirmed—represents evidence of environmental exposure but does not indicate toxicity. The concentrations quoted in the television programme are close to those previously reported for antimony in hair and much lower than those recorded after drugs containing antimony have been used to treat parasitic diseases.¹⁰

The sudden infant death syndrome is not a "new" condition. Babies have died suddenly and unexpectedly in all societies since antiquity.¹¹ Until this century such deaths most commonly occurred in bed with a parent and were attributed to overlying.¹² In 1953 a committee was established by the Ministry of Health, which undertook studies

on sudden death in infancy. The interim report published in 1957 estimated that 1400 such deaths occurred annually in England and Wales,¹³ a similar figure to that recorded in 1990, before the "back to sleep" campaign began.¹ The dramatic fall in the incidence of the sudden infant death syndrome in many Western societies after "back to sleep" campaigns means that the incidence of the syndrome in these countries (in which mattresses covered with polyvinylchloride are widely used) is now similar to or lower¹⁶ than that reported from countries such as Japan and China where these mattresses are not commonly used.^{3,14,15} In Britain the incidence or the syndrome fell in the few years after many manufacturers increased the amount of antimony added to the polyvinylchloride coverings of mattresses (believing that this was necessary to meet the 1988 Furniture and Furnishings Fire Safety Regulations). Thus potential environmental exposure to antimony increased as rates of the syndrome fell.^{13,16}

The Richardson hypothesis has some biological plausibility and fits some, but not all of the known epidemiological features of the syndrome. Stibine, the trihydride of antimony, is highly toxic, and without compelling evidence of benefit from the addition to cot mattresses of (reportedly non-toxic) antimony trioxide we agree with the suggestion in the Turner report that its continued use should be seriously questioned.⁴ While Richardson's observations of release of stibine from polyvinylchloride have not been confirmed independently, the concentrations of antimony in infants suggest an environmental source. The dietary content of antimony is generally low,¹⁶ concentrations in tobacco are higher,¹⁷ and antimony is widely used as a fire retardant—in paper products as well as plastics¹⁶; information on its uptake and excretion in infants, however, is scarce.

The two year case-control study on all sudden unexpected deaths in infancy in three health regions (with a population of about 17 million), funded by the Department of Health, is due to complete the collection of data early next year and will provide important information on any association between particular types or ages of mattress and the risk of the sudden infant death syndrome. Further studies are needed of the concentrations of antimony in infants and the relations to various environmental sources. Of particular urgency is the need for independent replication of Richardson's observations of the generation of stibine from polyvinylchloride.

For the immediate future, it is important to stress that the advice contained in the "back to sleep" campaign of 1991 still holds and has been followed by a remarkable fall in the incidence of the sudden infant death syndrome in Britain and elsewhere.¹⁶

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Internal market rules OK

New rules take account of market behaviour

When the internal market was introduced into the NHS health economists pointed out that it would have to meet several conditions if it was to achieve the ends that its promoters hoped.¹ Analysis of its early operation by the King's Fund and others suggested that it was more successful than might have been expected in meeting most of these conditions.² However, several unresolved issues remained, particularly regarding the competitive structure of the market. Economists at the Department of Health became increasingly aware of these problems as the market evolved, and they have used the experience of the past four years of market operations to develop a set of guidelines, or rules, for operating the internal market.

These guidelines, *The Operation of the Internal Market: Local Freedoms, National Responsibilities*, were published earlier this week.³ They concern four areas: mergers between providers and joint ventures; mergers between purchasers; managing change when providers are in difficulty (a euphemism for reconfiguring hospitals, including closures); and collusion between providers and purchasers, or between providers.

Mergers between providers present difficulties for markets. On the one hand, the creation of bigger units may result in efficiency savings through economies of scale. On the other hand, these large units may monopolise the market, operating against the interests of purchasers and hence ultimately of patients.

The guidelines propose that, in general, mergers will not be challenged by the NHS Executive so long as they do not require existing trusts to be dissolved and the resulting single organisation provides less than half of services in a market. The market is defined as the area around each provider in terms of travel time (14 to 19 minutes for accident and emergency services; 30 minutes for all other services). If a proposed merger would result in a single organisation providing more than half the services in the market the executive would examine the proposal in detail, comparing the benefits, through exploiting economies of scale, with the costs in terms of reduced competition. Although the guidelines' authors would undoubtedly deny any such implication, their tone implies that the cases where the benefits exceed the costs would be rare and therefore that few mergers of this kind are likely to be permitted.

Detailed examination of the costs and benefits should be welcomed. Far too often health authority purchasers have been unduly impressed by the potential that merger or

"rationalisation" of providers offers for short term savings, totally ignoring the merger's impact on purchasers' ability to switch services away from expensive or unsatisfactory providers, and the consequent long term power that this would give to the huge units that would appear after the merger. The only problem with this proposal is that the detailed examinations may prove costly and will also be subject to political and other manipulation. Care would have to be taken that whatever arm of the executive was making the examinations was not "captured" by the relevant interests.

Are mergers in the public interest?

Mergers between purchasers, again often justified on the grounds of economies of scale, also present difficulties for the operation of internal markets. The problems here concern the distancing of the purchaser from local people and from local general practitioners, and the consequent weakening of the purchaser's ability properly to take account of all the diverse needs and wants of the people on behalf of whom it is supposed to be acting. Also, patient choices may be limited through standardisation or the use of block contracts with a single provider. Accordingly, the guidelines suggest that mergers can be considered only where measures are in place to ensure that patients' and general practitioners' views are heard and that patients are actually offered choices where appropriate.

The guidelines could have taken a more robust approach on this point, not only discouraging mergers but actively encouraging demergers. Many areas, dissatisfied with the performance of centralised purchasing and impressed by the success of fundholding general practices, are looking at ways of decentralising their purchasing decisions through locality purchasing, total fundholding, and the like. Demerging would be another way of achieving this. The weight of experience favours smaller, not larger, purchasers, and it would have been useful if the guidelines had reflected this.

An important difficulty with the operation of the internal market so far has been the absence of any strategy for convincing "provider exit." For markets to have incentives for quality and efficiency it must be possible for providers to fail; if providers who get into financial difficulty know that they always will be bailed out, their incentive to be responsive to purchasers' requirements is substantially reduced, if not eliminated. Yet the pressures to bail out