guideline from the Royal College of Obstetricians and Gynaecologists that there should be one specialist for 500 deliveries will be tenable only if the trend towards sub-specialisation accelerates. A small referral base does not provide enough complications to maintain skills.⁵

Apart from stipulating a greater role for midwives, the report tries not to be too prescriptive; it recognises that purchasers and providers may agree many different models of care. The result, however, is that much of the report reads like a 100 page mission statement. It also seems to contain some internal contradictions. For instance, it argues that women should have the option of a planned home birth, especially as any increased risk is small (at least in absolute terms) and even those who perceive a risk may make a trade off in order to realise other gains—just as we do when transporting our families by road. Only a parentalist could argue against the report on this point, but the information that the report suggests should inform this choice was biased, although the report argues throughout that couples should be given unbiased information. The report states that any exposition on the risk of an unexpected emergency at home should be balanced by evidence from quoted studies showing higher intervention rates in hospital; the problem is that these studies are confounded by different patterns of midwifery care. The report advocates, however, that, irrespective of where she chooses to deliver, a woman's intrapartum care should be the responsibility of the same midwife. Perhaps this just shows that unbiased information, for which the report makes repeated pleas, exists only in those few areas which can be resolved empirically.

The usual slogan about choice appears in the report, but it is not backed up by thoughtful discussion. The report comes to the one sided conclusion that restrictions are more likely to be justified in response to a "demand for intervention rather than a request to avoid it." Thus the report argues against induction of labour purely at a woman's request when "there

are no clinical grounds," but "clinical grounds" are a function, among other things, of a person's wishes. Some women may have a value system whereby, given the probabilities of the various outcomes, induction of labour (or even elective caesarean section) on request is entirely appropriate. Those who are sincere about women's autonomy and whose real motivation is to foster choice rather than particular choices must avoid any hint of double talk. (I could not help noticing that day centres for fetal assessment are condemned without even a mention of relevant research.)

The proposed reorganisation has considerable implications for midwives, who as independent practitioners will be directly exposed to criticism and litigation. Some will relish the opportunities for personal and professional development, while others may be reluctant to work irregular hours or accept greater responsibility. As midwives become more autonomous and assertive it will be important that they do not abuse their power and become a barrier to the involvement of other professionals. Women should be free to consult their general practitioners or obstetricians or to avail themselves of hospital clinics. The report emphasises that no provider group should monopolise the provision of services.

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"Clusters" of anophthalmia in Britain

Difficult to implicate benomyl on current evidence

The campaign led by the press about a possible link between the pesticide benomyl and alleged clustering of cases of anophthalmia—babies born with no eyes—has prompted the government to commission research.¹ The government's dilemma is whether to pursue the question of pesticides and clustering or to investigate all the main causes of anophthalmia and how they might be prevented.

Whatever is decided, precise case definition will be of prime importance. Anophthalmia is part of a range of defects that includes microphthalmia. Differentiating between anophthalmia and severe microphthalmia and between microphthalmia and normal eye size may require expert examination and agreement on the limits of normal eye size. Isolated eye defects need distinguishing from those associated with other malformations as the underlying causes may differ. Overall, between 40% and 73% of cases reported to the national congenital malformation surveillance scheme (CMSS) in England and Wales and to the European congenital malformations registries (EUROCAT) had other malformations (B Botting and H Dolk, unpublished data).²

Anophthalmia and microphthalmia may result from fetal damage occurring up until mid-pregnancy due to a range of chromosomal, genetic, and environmental causes. Of cases of anophthalmia and microphthalmia reported to the European

registries, 16% had associated chromosomal abnormalities and 13% had other symptoms including genetic syndromes.² Fewer than 10% of cases notified to the surveillance scheme in England and Wales and 16% notified in Scotland had chromosomal or genetic factors reported (B Botting and H Dolk, unpublished data).³ This is partly due to registration under other malformations. Although a genetic aetiology is suspected in most cases of anophthalmia or severe microphthalmia, this can be shown only in the minority of cases where there is a detachable chromosomal abnormality or a family history. No genetic markers are yet available.

Potential environmental causes include infective agents, toxins, and drugs. In humans, congenital rubella, influenza, varicella, and parvovirus B19 infection have been associated with multiple fetal malformations including microphthalmia.^{4,5} Cases of anophthalmia or microphthalmia have also been reported in association with antenatal exposure to lysergide, ethambutol, thalidomide, and vitamin A.^{4,6,7}

Experiments on animals suggest a possible link between benomyl and anophthalmia. Some 43% of the offspring of pregnant rats fed more than 1000 times the estimated dose of benomyl received by farm workers spraying pesticides developed malformations including microphthalmia.^{8,9} Experimental infection of pregnant cattle with pestivirus

(bovine viral diarrhoeal virus) causes fetal eye defects, including anophthalmia.¹⁰ In Britain 100-300 incidents of fetopathy thought to be due to congenital pestivirus infection in cattle are reported each year.¹¹

In England and Wales about nine new cases of anophthalmia and 11 of microphthalmia are reported to the congenital malformation surveillance scheme each year within the first 10 days of life. Although underreporting undoubtedly occurs, the combined prevalence of anophthalmia and microphthalmia at birth in England and Wales for 1981-5 and 1986-90 was 0.4 and 0.3/10 000 live births respectively.¹² These figures are similar to the prevalence of 0.5/10 000 live births for 1985-92 reported by the Northern regional fetal abnormality survey, which is based on notifications antenatally and up to any age postnatally (M Renwick, personal communication).

The prevalence based on notifications to the European congenital malformations registries is higher: for 1980-8 anophthalmia was reported in 0.3/10 000 births and microphthalmia in 1.4/10 000 live births.² The discrepancy between registries may be partly due to underreporting in Britain, differing case definitions, and overestimation of prevalence in centres with a particular interest in ocular abnormalities.

Data showing trends over time are less affected by underreporting. Since 1971, two years after benomyl was licensed in Britain, no increase in the reported prevalence of anophthalmia and microphthalmia has occurred: the reported prevalence for England and Wales was 0.4, 0.5, and 0.4/10 000 live births in 1971-5, 1976-80, and 1981-5 respectively.¹² Over the past 10 years the use of benomyl has fallen, but use of its metabolite, carbendazim, which also causes malformations in rats,¹³ has increased nearly 1000-fold since 1965 (M R Thomas, Ministry of Agriculture, Fisheries, and Food, personal communication). These data suggest that benomyl and carbendazim are unlikely to be major causes of anophthalmia or severe microphthalmia in humans.

A French population based study of 78 cases during 1979-88 found no trends with time or space-time clusters.¹⁴ The largest cluster reported by the *Observer* comprised nine cases born in north Lincolnshire over 12 years.¹⁵ No details of the proportion with anophthalmia or microphthalmia was presented.¹⁶ "Clusters" identified retrospectively, without a prior hypothesis and without predefined geographical, temporal, and diagnostic boundaries, require cautious statistical treatment.¹⁷ Cases within the cluster may not be causally related: the cluster may be due to chance or manipulation of boundaries.

Future research should be designed to benefit those at risk, and studies of clustering may not achieve this. Clustering could be consistent with fetal exposure to pesticides, pestivirus, or other environmental agents; genetic susceptibility to specific environmental agents; or even a genetic cause if affected family members lived in the same area. Conversely, the absence of clustering would not provide evidence against an association with a specific environmental factor, particularly if exposure to that factor is widespread.

Future epidemiological studies on anophthalmia and microphthalmia should include the detailed ophthalmological and dysmorphological assessment of affected patients and their families to define cases accurately and identify those of known cause. Such assessment would also provide the opportunity to investigate chromosomal and genetic factors, with potential benefits for genetic counselling and antenatal diagnosis.

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Paternal exposure to chemicals before conception

Some children may be at risk

The dangers to children of maternal exposure to a variety of chemicals and drugs is now well established. Interest in this area may be traced back to the discovery in the 1960s that giving thalidomide to women during the first trimester of pregnancy caused severe limb reductions in their offspring.¹ No similar dramatic discovery has been made of an effect of paternal exposure to drugs or environmental chemicals.

Over the past two decades, however, evidence has been accumulating that exposure of men to xenobiotics can impair their fertility and adversely affect their progeny. Such adverse outcomes may include pregnancy loss, malformations evident at birth, and problems that are detected only later in life, such as behavioural abnormalities and cancer. We focus here on the

risks associated with paternal exposure to chemicals; we will not discuss the extensive literature dealing with the effects of paternal exposure to radiation.

Epidemiological studies have examined the effects of paternal occupational exposures on fetal development and childhood cancers. The effects on fetal development range from spontaneous abortion and stillbirth to malformations at birth, preterm delivery, or delivery of an infant who is small for gestational age. Various paternal occupations have been associated with these adverse outcomes. Partners of motor vehicle mechanics have an increased incidence of spontaneous abortions.² Fathers employed in occupations associated with solvent exposures are more likely to have offspring with