stitute treatment programme comparable to methadone maintenance for opiate addicts does not seem promising as there is no obvious candidate drug for the substitute. The American use of methylphenidate seems wrong for Britain, where there is a more extensive history of intravenous abuse of this drug. One of the more promising approaches to treatment is using tricyclic antidepressants such as desipramine, which may correct disturbances in dopaminergic pathways and alter receptor supersensitivity resulting from regular cocaine use.^{15 16} Such drugs may thus help to correct the depression or anhedonia which colour the let down from cocaine and which if uncorrected may invite further cocaine use. Alternatively, a drug might be given which would produce cocaine blockade (just as naltrexone does with heroin), but no such drug has yet been reliably identified. There have now been reports that bromocriptine may reduce postwithdrawal craving for cocaine, but these first reports need to be investigated further.¹⁷ General treatment approaches used in rehabilitating those addicted to other drugs should be relevant. The use of a structured approach for preventing relapse should help in identifying alternative coping strategies.¹⁸ Systemic exposure

to stimuli that promote craving may also be useful—as it has been with other addicts.¹⁹⁻²¹ Most important of all may be the non-specific and low key help based on the wide availability of a friendly and knowledgeable person who may inform, support, and cajole. Treatment for those addicted to cocaine and crack must depend on strengthening existing services rather than on creating a new crack specialty.

So is crack the tiger or the toy poodle? In the light of the scientific evidence it would be wrong to portray the use of crack as innocent fun. And yet to give crack the image of a mythological monster and portray it sensorially as something uniquely diabolical will only add to its allure. The hype is dangerous as well as the drug.

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Diagnosing rubella

May be difficult in patients without symptoms

One of the government's aims in introducing the mumps, measles, and rubella vaccine for all children in October 1988 is to eliminate rubella.¹ But this will not happen for years, and some pregnant women will still be exposed to rubella. Primary infection with rubella in the first four months of pregnancy carries a major risk of fetal damage,² and correct diagnosis is thus vital. The infection is usually clinically apparent but may be subclinical; fetal damage is likely whether or not the infection is apparent.³ This means that all patients with rubelliform illnesses and exposures to rubella in early pregnancy must be investigated.

Clinical diagnosis is unreliable because other viral infections, particularly those with parvovirus B19, may present as a rubelliform illness.⁴ Viral culture is unhelpful, and serological investigation is the only valid method of diagnosis. The characteristic serological pattern of primary rubella is that the patient develops maximum concentrations of antibody within 10 to 14 days after the onset of illness. The antibodies include high concentrations of specific IgM that persist for at least a month⁵: if the samples of serum are taken more than a few days after the onset of the illness (as is usually the case) and rubella antibody is present then the detection of specific IgM is taken to indicate recent infection. There are

reliable and sensitive assays for detecting specific IgG and IgM,⁶ and usually it is straightforward to confirm or refute a diagnosis of recent primary rubella. Unfortunately, however, there are several pitfalls.

All assays for specific IgM occasionally give false positive results. Low concentrations may occur in infectious mononucleosis,⁷ in infection with parvovirus B19⁸ and cytomegalovirus (M Supran, personal communication), and if rheumatoid factor is present. Nevertheless, most false positive results cannot be explained. Experience has shown that if a positive result in one assay is not confirmed in a different assay of similar sensitivity then the result is likely to be non-specific and hence negative.

A further problem is to distinguish primary rubella from reinfection. Reinfection is diagnosed when an antibody response is shown in someone who has previously had natural rubella or successful immunisation. If the patient is in early pregnancy and has not had symptoms the distinction is crucial. There have been only a few proved cases of asymptomatic reinfection with transmission to the fetus and consequent damage,^{9 10} although further cases await reporting. As rubella is endemic in children, however, probably only a few of the asymptomatic reinfections that occur in pregnancy

are recognised. Although the data are not available, it seems that the risk to the fetus of asymptomatic reinfection is small,^{3,11} and certainly it is far less than that of primary rubella. In the rare cases of symptomatic reinfection viraemia may be presumed to have occurred, putting the fetus at appreciable risk.⁹

An antibody response may be confidently ascribed to reinfection if two or more previous positive results for rubella antibody have been obtained. One previous positive result must be treated with scepticism as errors may have occurred.¹² Hence the Royal College of Obstetricians and Gynaecologists and a working party of the Public Health Laboratory Service advise that every woman should be tested for rubella antibodies in every pregnancy.⁶ A history of rubella immunisation also means that primary rubella is unlikely, but vaccination failures are well recognised.¹² If a patient has a rising titre of rubella antibody reinfection may be discriminated serologically from primary rubella if specific IgM is not found or is found only in low concentration.^{3,11,13} The recent finding of raised concentrations of specific IgM in confirmed reinfections (unpublished observations) adds to the problem. If only late serum samples are available and there is no history of previous testing or immunisation then specific IgM (even at low concentration) may reflect recent reinfection or primary rubella some weeks previously. Recently developed assays that measure specific IgG subclasses and avidity may allow this distinction to be made, but the tests have not yet been validated in routine use.¹⁴⁻¹⁶

A reliable and safe method of prenatal diagnosis of fetal infection would be valuable when there is diagnostic uncertainty or proved reinfection (although fetal infection does not necessarily mean fetal damage). Approaches include culturing amniotic fluid for rubella virus, which is time consuming and unreliable, and detecting specific IgM in fetal blood, which has to be delayed until 23-24 weeks of pregnancy.

Detecting the rubella virus genome and antigen in chorionic villus samples is promising but requires further assessment.¹⁷

Correct diagnosis of primary rubella or reinfection in patients without symptoms may thus be difficult and lead to much anxiety about how to manage the pregnancy. It is thus essential for diagnosis that full information is given to the laboratory on symptoms, date and type of contact, previous rubella immunisation, and dates and results of previous tests for rubella antibody.

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Inquiry into stillbirths and infant deaths

We must have comparable regional surveys

The government in its recent response to the report of the Social Services Committee on perinatal, neonatal, and infant mortality has accepted that because infant mortality is a key indicator of the nation's health the recent levelling off in its rate of decline represents a problem.¹ It has rejected the committee's recommendations that funds should be earmarked centrally for allocation to areas of social deprivation and to those health authorities whose maternity and neonatal services are below standards outlined by the committee. It has also declined to prescribe the structure of neonatal intensive care services, reiterating its view that responsibility for the detailed planning and delivery of maternity and neonatal services must lie with regions and districts.

In some other respects, however, the government has responded positively to the committee's recommendations. It has agreed that improved understanding of why babies die must be an essential component of any strategy to reduce perinatal and later infant deaths. The government will consider the scope for further research and has already commissioned the Medical Research Council to undertake a review of the sudden infant death syndrome and advise on what new research is needed.

Informed assessment of the pathological causes of stillbirths and infant deaths is required, and this cannot happen

without improved specialist pathology services. The NHS Management Executive has therefore been asked to ensure that all regions have at least one paediatric pathologist in post by 1991. Also endorsed is the committee's recommendation that procedures should be established for informing health authorities of the outcome of coroners' inquests.

These developments should improve the quality of the information provided by those who certify the causes of stillbirths and infant deaths and thus enhance the value of the vital statistics based on these certificates and published regularly by the Office of Population Censuses and Surveys. In addition, improved pathological investigation of stillbirths and infant deaths is needed for regionally based epidemiological surveys. Over the past decade there have been repeated calls for these developments,²⁻⁴ but only a minority of English regions have responded; the Northern region has the longest established survey⁵ and the only one now covering stillbirths and all infant deaths routinely. The government has now asked the NHS Management Executive to ensure that all regions have surveys in place by April 1991.

Regions should adopt the schemes for classifying and tabulating stillbirths and neonatal deaths which have been agreed and published jointly by representatives of the surveys in the Northern region and Scotland.⁶⁻⁸ For its part the