and the community medicine specialist should decide the policy; prophylaxis should certainly include "kissing contacts" and household contacts, who have an attack rate 500-800 times that of the general population.⁵ Medical and paramedical attendants do not require prophylaxis unless there has been intimate exposure, such as mouth to mouth resuscitation.⁶

When a schoolchild develops meningitis there is usually great concern in the local community, but it is essential that general practitioners do not take individual initiatives without discussion with the community medicine specialist, who may in turn wish to take further advice.⁶ Certainly he or she should arrange for the head teacher and local general practitioners to be informed of the antibiotic policy. Unfortunately it is impossible to give advice that is universally applicable. In general, school contacts are not given prophylaxis unless there has been particularly close contact or more than one related case at the school. Prophylaxis should be considered for staff and children attending nursery schools or day care centres, which may function in "household fashion," often with close contact among staff and children.

Patients with meningococcal meningitis may continue to harbour the organism in their throats after treatment with penicillin, and this carriage should be eradicated by giving a "prophylactic" antibiotic before discharge from hospital.

Rifampicin is the best antibiotic to use for prophylaxis⁶ as it reduces nasopharyngeal carriage rates by 80-90%.⁷ The current *British National Formulary* recommendation for prophylaxis is rifampicin 600 mg every 12 hours for two days; children 10 mg/kg (those aged 3 months-1 year 5 mg/kg) every 12 hours for two days.

In normal circumstances meningococcal vaccination has no part to play after a sporadic occurrence of meningococcal infection because protection does not start immediately,

children under 2 years respond poorly to vaccination, and most isolates in Britain are group B strains and are not preventable by the available vaccines. If cases occur despite correct prophylaxis or if there is a rapidly evolving epidemic, especially in institutions, then vaccination may have some part to play.⁶ It may also prevent secondary cases, especially delayed emergence of infection in household contacts,⁸ which may occur many days later—well after the protection given by antibiotic prophylaxis will have disappeared. Accordingly, we believe that close contacts of patients over the age of 2 with group A or group C infection should be vaccinated. Whatever form of prophylaxis is undertaken a close contact should be instructed to seek urgent medical advice if he or she develops any illness.

When dealing with meningococcal infection at least one telephone call, either seeking help or giving information, is an essential part of management.

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Congenital virus infections

More viruses are now known to infect the fetus

The list of viruses that may cause congenital infections is growing. In addition to rubella virus,¹ cytomegalovirus,² and varicella zoster virus³ the human immunodeficiency viruses (HIV-I and HIV-II)⁴ and human parvovirus B19⁵ may infect the developing fetus. Transplacental infection with Japanese encephalitis⁶ and Lassa virus⁷ has also been reported, as has occasionally that with hepatitis B virus.⁸ Herpes simplex virus, varicella zoster virus, enteroviruses, hepatitis B virus, HIV, and human T cell lymphotropic virus (HTLV-I) may cause perinatal infections.

By contrast with other maternal virus infections that sometimes induce congenital anomalies primary rubella almost invariably affects and damages the fetus if acquired in the first trimester. Some obstetricians who counsel patients are not aware of this and base their advice that the risks are lower on the results of earlier prospective studies.¹ Congenital defects, usually deafness, occur less frequently (in roughly 15% of cases) when maternal infection occurs between 13 and 16 weeks' gestation and are rare after infection at between 17 and 20 weeks. A recent study of 38 cases showed that rubella before conception was not associated with fetal infection.⁹ Fetal malformations may very rarely result from reinfection in the first trimester, but the risks have not been measured.¹⁰ Prospective studies to estimate this and determine why only some pregnancies have an adverse outcome will be of value in

providing advice to obstetricians and their patients. The rubella vaccination programme has substantially reduced the susceptibility among women of child bearing age, and giving measles, mumps, and rubella vaccine to preschool children should reduce the circulation of virus in the community—68 cases of rubella in pregnant women were, however, reported to the Communicable Disease Surveillance Centre in 1989. For some time to come women and their doctors will continue to ask for advice about the risks associated with both primary rubella and reinfection.

When rubella occurs in pregnancy there is usually an illness with a rash or a history of contact with a rubella-like illness. By contrast there is little to pinpoint primary infection with cytomegalovirus as infection in immunocompetent hosts is usually subclinical. Transplacental transmission of virus occurs less frequently (30-40% of cases) than with rubella, and only some 10% of infected fetuses are severely damaged. Damage may result from infection occurring at any stage of pregnancy. Nevertheless, congenital cytomegalovirus is still the most common microbial cause of mental retardation. Recurrent cytomegalovirus infection only very rarely causes congenital anomalies. All this adds up to the conclusion that screening pregnant women for evidence of recent or current cytomegalovirus infection would be of little value. The birth of between 250 and 400 babies damaged by cytomegalovirus a

year in Britain might be prevented, however, if an effective vaccine was available—but such a development has not been given much emphasis. Live attenuated vaccines would not be acceptable today,¹¹ particularly in pregnancy, but the use of DNA recombinant technology, already applied to the development of such vaccines as those against hepatitis B, HIV, and Lassa fever, may well result in an effective cytomegalovirus vaccine.

In parts of tropical Africa and Asia and many urban areas in the United States increasing numbers of women of child bearing age are infected with HIV, and 30-40% transmit infection to their infants in utero or perinatally. Transmission of the virus occurs more often among mothers with more advanced HIV disease.¹² There is no convincing evidence, however, that HIV induces congenital malformations. Some 25-40% of infected infants die before their second birthday.^{12,13} Because antiviral treatment may be beneficial if started in infancy early diagnosis is important.⁴ Isolation of virus from peripheral blood lymphocytes is the only established method of diagnosis in the neonate, but this is a time consuming method limited to a few specialised laboratories. Gene amplification techniques, however, such as the polymerase chain reaction,¹⁴ seem promising, and techniques for detecting HIV specific IgM deserve further evaluation.¹⁵

Varicella (chicken pox) and parvovirus B19 infection in pregnancy give less cause for concern. In Britain there are now more women susceptible to varicella (5-7%) than to rubella (2-3%); indeed, we are now consulted as frequently about the risks of fetal damage after varicella as rubella. The characteristic constellation of defects known as the "congenital varicella syndrome" is fortunately rare, occurring in only about 3% of pregnancies (a figure that should be set against the risk of major malformations in uninfected pregnant women of 2-3%). The risk associated with zoster in pregnancy is so small that it can be safely ignored. Parvovirus B19 infection in adults may be asymptomatic or produce a rubella-like rash and arthralgia. A recent study showed that when infection occurs in pregnancy transplacental transmission of virus may occur in up to a third of cases. Most pregnancies proceed to term normally without evidence of congenital malformation, but in about 9% of cases infection may result in miscarriage or stillbirth associated with hydrops fetalis, the risk of fetal loss being greatest in the second trimester (p 1166).

Against the background of those data on the common viruses can anything be offered to pregnant women by prenatal virological diagnosis employing techniques such as the detection of virus specific IgM in the fetal blood and detection of virus or viral components in amniotic fluid or chorionic villus biopsy specimens? Fetal blood sampling is possible after 18 weeks' gestation, but techniques for detecting virus specific IgM may not be sufficiently sensitive to detect the low levels of specific IgM present in fetal blood even at a time when therapeutic abortion may still be considered. For example, when this technique was used for the prenatal diagnosis of congenital rubella virus infection false negative results were obtained even at 23 weeks' gestation.¹⁶ Cytomegalovirus has been detected in the amniotic fluid of patients presenting with symptomatic primary infection (K Hayes and J A Leydon, European Society Against Virus Diseases and European Group for Rapid Viral Diagnosis joint meeting, Groningen, 1989). As most mothers do not have symptoms and because only about one third of the fetuses infected will be damaged the technique is of limited value, though a negative result is reassuring. Detection of viral nucleic acid in chorionic villus biopsy specimens by nucleic acid hybridisation or the polymerase chain reaction may be useful for assessing the risk to

pregnancies in which maternal rubella in the first trimester has been suspected but cannot be confirmed¹⁷ but would be of little value in assessing the risk of rubella after the first trimester as fetal infection without damage is common.¹⁸ Perhaps such techniques are more likely to be of value for assessing the risks of rubella virus reinfection, when detection of the viral genome would be of importance as placental infection would not be expected to occur in most patients immune to rubella virus. Although detection of varicella zoster virus specific IgM in fetal blood or the viral genome in chorionic villus biopsy specimens might be evaluated, the rarity of the congenital varicella syndrome would make this difficult. Perhaps ultrasonography should be used more frequently to monitor women who have acquired varicella in the first 20 weeks of pregnancy, as it is the only method for detecting defects induced by varicella such as severe cicatricial scarring and limb deformities. Pregnant women may be monitored by ultrasonography for evidence of hydrops fetalis induced by B19, and fetal blood tested for evidence of B19 viraemia and anaemia, which may result from infection with B19. Specific IgM is not usually produced. Intrauterine blood transfusion has been used to treat a few such patients, but this high risk procedure has not been evaluated.¹⁹

Fetal blood sampling and amniocentesis cannot be used to determine whether intrauterine infection with HIV has occurred as such techniques might increase the risk of infecting the fetus. By sampling fetal blood and amniotic fluid from infected women before therapeutic abortion, however, it might be possible to determine how commonly intrauterine infection occurs in early pregnancy. Although using the polymerase chain reaction to detect virus in fetal blood may seem an attractive proposition, this may not be reliable as by this reaction one molecule of DNA may be amplified a millionfold; that means that contamination by the minutest quantity of maternal blood may give a false positive result.

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