Editorial

RUBELLA IN EARLY PREGNANCY

PREGNANCY and german measles are both common conditions in general practice, but the combination can be disastrous. What should general practitioners do when a woman reports that she has missed her last period and that a few days previously she has been in contact with a child now thought to have rubella? Inevitably most such patients are not sure whether they have ever had german measles.

Most practitioners would agree that the first step should be to confirm pregnancy and in the early weeks this will have to be done by a urine test. Many practitioners would take blood for rubella antibody titre at this first attendance, but some might prefer to wait for confirmation of pregnancy. Laboratories vary but some will now report the rubella antibody titre by telephone within 24 hours.

A negative history of german measles is often obtained, but in practice 80 per cent of British women will be found to have a detectable titre of rubella HAI antibody in their sera. There is dispute about what level of titre offers adequate protection. Some believe that 1/32 is adequate while others believe that any level of detectable antibody will prevent fetal infection.

Most laboratories will state, on the basis of their own results, whether an individual patient should be considered susceptible or immune.

If pregnancy can be confirmed and if sufficient antibody is detected the patient can now be reassured that neither she nor her baby are going to be infected. The difficulties, however, arise when a mother has a low positive titre and she presents about two weeks after contact. She needs to be retested later.

If, however, no antibody can be detected what can be done to protect the mother and the fetus against infection? Until a few years ago the answer would have been easy—gamma-globulin. In 1970, however, a report by the Public Health Laboratory Service concluded that " the prophylactic use of immunoglobulin against maternal rubella . . . is based on premises that now appear largely unsound . . . Immunoglobulin has little effect in preventing maternal rubella and cannot therefore be expected to protect against the possibility of fetal abnormality."

Until something better is produced, however, and because in the Public Health Laboratory study only one out of 70 babies born to infected mothers who were given immunoglobulin showed evidence of a congenital abnormality at the time of the report, some practitioners will still administer normal human immunoglobulin to suspectible mothers, if possible by day six after exposure to rubella in early pregnancy, in the hope of protecting the fetus even if the mother herself does develop clinical or subclinical rubella. The usual dose is 750–1,500mg but this is probably still too low. Professor J. A. Dudgeon (1974) has been giving 1,500mg at the mother's first attendance and another 1,500mg about the sixth day after contact.

If no antibody is detected in the woman's first serum, provided it is taken within a few days of contact, it is clear that the mother must be retested—but when?

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Within an incubation period of about 17 days, she could not show any rise in HAI antibody titre in the first two weeks after contact. In the next two or three weeks, if infected, her antibody titre would rise significantly. Thus many practitioners now advise the patient to return for her second blood test four or five weeks after first contact.

If her second sample, taken five weeks after the last possible contact, still shows no rise in antibody titre she can be reassured that she has escaped infection and her baby will have been unharmed by that contact. If antibody negative, she should be offered a vaccination against rubella as soon as possible after the pregnancy, but with prevention of pregnancy for at least two months afterwards because a live vaccine is being used.

If, however, her second sample does show a fourfold or greater rise in rubella titre, the patient must have been infected, even if she has not appeared to suffer from illness nor has a rash appeared. The fetus unfortunately is now at risk.

If rubella infection of the mother is confirmed, the risk of the fetus being deformed varies with the stage of pregnancy, falling throughout the first four months to *nearly* zero. The probability is never 100 per cent and by the end of the third month is about 30 per cent. Thus two out of three babies so exposed will be normal.

Guiding parents to understanding these risks is always difficult, but worthwhile and essential. They, and they alone, will have to make the decisions which are in accordance with their beliefs and which they are least likely to regret in the future.

The choice lies between continuing pregnancy and bearing hopefully the months of waiting in doubt about the outcome, or undergoing a therapeutic abortion, not knowing if the fetus is damaged or not. A crumb of comfort can be offered to the mother who chooses abortion—the fetal remnants can be examined for rubella virus and if this is found the parents can be virtually certain not only that the fetus was infected, but was also damaged.

The challenge to general practice now must be to seek to avoid this situation and to develop methods of finding and immunising those who are susceptible before their first pregnancy occurs.

REFERENCES

Dudgeon, J. A. (1974). *British Medical Journal*, **2**, 723.
Public Health Laboratory Service Working Party on Rubella (1970). *British Medical Journal*, **2**, 497.

PROOF CONTAINERS

The authors consider that the development in mechanical methods of testing ' child-proof ' containers is highly desirable and they believe that the tests they have used are capable of development into such a method. The authors believe that further research into high-torque ratio caps might well yield a reasonably child-resistant closure quickly and at much less cost than any other form of packaging.

The authors also draw attention to the possibility that the upsurge in child poisoning from 1966–67 may have been caused by the introduction of the first plastic dispensing containers with push-off caps.

Jartside, R. & Carter, J. W. L. (1975). *The Pharmaceutical Journal*, **215**. No. 5832, 168.