take less time. Tearing of the mesosalpinx is a recognised, if infrequent, complication and it can usually be controlled by reapplication of the ring. Postoperative lower abdominal pain has been a problem, and although only six of the patients at follow-up commented on its severity, it clearly caused them considerable distress in the immediate postoperative period. We now apply 2% lignocaine gel to the tubes at operation in an attempt to prevent this complication. The site of application of the ring on the tube seems to have no effect on the incidence or severity of the pain.

Advocates of this method of sterilisation claim advantages over tubal diathermy in terms of increased chance of reversibility. This claim has not yet been substantiated but might be understandable since there would appear to be less tubal damage than with diathermy. The pregnancy rate in the published series²⁻⁴ is extremely low, and although our follow-up is short (the study began in April 1976), no pregnancies have so far been reported.

Laparoscopic tubal diathermy is associated with an increased incidence of menstrual problems in patients seen up to 28 months after procedure.⁵ Long-term follow-up of patients sterilised using diathermy and a comparison with a similar series of patients sterilised using the Silastic-band technique is underway, and may provide data that is helpful in understanding this late complication of female sterilisation.

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Coxsackie B2 virus infection and acute-onset diabetes in a child

There is increasing interest in the possible role of viruses in the aetiology of juvenile-onset diabetes. There is controversial epidemiological evidence that Coxsackie B viruses, particularly Coxsackie B4 virus, may be associated with juvenile-onset diabetes, but direct evidence is lacking.¹ We report a case of acute-onset diabetes associated with Coxsackie B2 virus infection in an 18-month-old child.

Patient, methods, and results

An 18-month-old boy was admitted to hospital on 24 September 1975 with a one-week history of mild diarrhoea and polyuria. He was a well-developed, intelligent, active child and the only clinical abnormalities were fever (37.8°C), acetone on the breath, and glycosuria. Initial investigations showed: blood glucose 14.2 mmol/l (256 mg/100 ml), blood urea 8.3 mmol/l (50 mg/100 ml), carbon dioxide total 19 mmol/l (8.5 ml/dl); other electrolytes were normal. The total white cell count was $19.3 \times 10^9/l$ ($19\,300/mm^3$) with 76% lymphocytes ($14.6 \times 10^9/l$; $14\,600/mm^3$), 20% polymorphs ($3.9 \times 10^9/l$; $3900/mm^3$), and 4% monocytes ($0.8 \times 10^9/l$; $800/mm^3$). A midstream urine specimen showed an increased pus cell count and coliforms were cultured.

The day after admission the blood sugar rose to 25 mmol/l (450 mg/100 ml), and soluble insulin (15–20 units daily) has been necessary to control his diabetes since then.

Serum samples were taken at one, 15, and 32 days and 14 months after admission, and were tested for antibody to infectious agents possibly associated with diabetes. Coxsackie group B virus antibody was titrated in a two-hour neutralisation test at 37°C. The reciprocals of antibody titres to Coxsackie B2 virus were 2560, 2560, 1280, and 1280, and Coxsackie B1, B3, B4, B5, and B6 antibody titres were <10. Titres of mumps, cytomegalovirus, complement-fixing antibody, and rubella haemagglutination-inhibiting antibody were all <10. *Mycoplasma pneumoniae* complement-fixing antibody titre was 20. For the titration of Coxsackie B immunoglobulin M (IgM) the indirect fluorescent antibody test was used with acetone-fixed monkey kidney cell monolayers infected with Coxsackie B1–6 viruses, patient's sera absorbed with Vero cells and heat aggregated human immunoglobulin, and sheep antihuman IgM conjugated with fluorescein isothiocyanate and absorbed with Vero cells. In the acute-phase sera the Coxsackie B2 IgM titre was 20, but staining of the other Coxsackie B virus types also occurred. This test also showed a fibrillar anticeellular IgM titre of 20 in the patient's acute-phase sera that could be removed by absorption with human myometrium. Coxsackie B2 IgM and the fibrillar anticeellular IgM titres were both <5 in the serum taken 14 months after onset.

There were two siblings in the family. The brother aged 6 years was 4 years old when he developed diabetes in June 1973, but a sister aged 5 years and the father and mother were not diabetics. Serum samples were taken from the family six weeks after the patients' admission to hospital and they had antibody to most or all of the six Coxsackie B virus types. Coxsackie B2 antibody titres in the brother, sister, father, and mother were 640, >1280, 160, and 320, respectively. The brother had rubella and cytomegalovirus antibody titres of <10; mumps and *M. pneumoniae* antibody titres were 20. The tissue type of the patient was HLA-A2, HLA-BW40, HLA-B12, and that of his brother was HLA-A2, HLA-BW40.

Comment

The combination of lymphocytosis and high titres of Coxsackie B2 neutralising antibody and Coxsackie B IgM with acute diabetes in an 18-month-old child suggests that the diabetes was associated with a recent Coxsackie B2 virus infection. Coxsackie B IgM and fibrillar anticeellular IgM were not detected 14 months after onset, which shows that the acute phase of the viral infection had ended. Coxsackie B virus neutralisation tests are type-specific, while Coxsackie B fluorescent antibody tests are not and are less sensitive.² Fibrillar anticeellular IgM autoantibody is found during the acute phase of other virus infections,³ and insulin release has been shown to be dependent on the integrity of the microtubular-microfilamentous system.⁴ Coxsackie B2 neutralising antibody was also present in the diabetic brother and in his sister, father, and mother, which shows that they were all infected with this virus at some time in the past. Cytomegalovirus, rubella, or mumps viruses and *M. pneumoniae* have also been implicated in juvenile-onset diabetes, but the patient and his brother had no evidence of recent infection with these agents. The tissue types of the patient and his brother were not those associated with juvenile-onset diabetes.

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