mentary oxygen with the Air-Viva lowered the inspired carbon dioxide concentration in a stepwise fashion, although the concentration did not reach zero even with 10 l added oxygen/min.

Comment

Resuscitator bags refill with air during the patient's expiratory phase. To prevent rebreathing it is essential that the air intake and expired gases are separated. The valve of the Air-Viva, however, has a common port for both, which leads to expired gas being drawn into the bag during refilling.

Supplementary oxygen, by displacement, reduces the amount of expired gas that enters the bag, so that the inspired carbon dioxide concentration falls as the flow of oxygen is increased. Nevertheless, as we found that the Air-Viva valve tended to obstruct when an oxygen flow of above 4 l/min was added it is impracticable to prevent rebreathing by using high flows of oxygen.

The use of an International Standards Organisation model lung with added carbon dioxide is a standard method for demonstrating rebreathing in anaesthetic breathing systems, and the results obtained are comparable with clinical events.[1] We were able to confirm these findings in two patients during the routine use of the Air-Viva bag. In both cases the inspired carbon dioxide concentration rose to 2% within 20 seconds.

Resuscitation bags are used for the resuscitation and during the transport of critically ill patients. In both of these circumstances a raised inspired carbon dioxide concentration may have serious deleterious effects, such as raised intracranial pressure and peripheral vasodilatation. Rebreathing also, of course, implies a reduction in the inspired oxygen concentration. We believe, therefore, that the Air-Viva resuscitation bag should be withdrawn from use. Although it is no longer marketed (having been superceded by the Air-Viva 2), there are many such bags in general use. The Air-Viva 2 is of completely new design and performed satisfactorily in this test.

We thank Professor C M Conway, Westminster Hospital, London, and Dr P J Simpson, Frenchay Hospital, for their help in this project.

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Autoimmune thrombocytopenia in pregnancy: new approach to management

Autoimmune thrombocytopenia may cause haemorrhagic complications in pregnant women, and transplacental transfer of IgG platelet autoantibodies often leads to neonatal thrombocytopenia. Early splenectomy, corticosteroids, and plasma exchange have all been used to provoke remission, and elective caesarean section is often recommended. Newland *et al* recently confirmed that most adults with autoimmune thrombocytopenia show a substantial and predictable response after infusion of high dose monomeric IgG (Sandoglobulin). We report our experience with this agent in a pregnant woman with severe thrombocytopenia. To our knowledge this has not been documented previously.

Case report

A 26 year old Japanese primigravida who had had thrombocytopenia since the age of 10 presented in the 11th week of pregnancy. Investigations at antenatal booking showed haemoglobin concentration 14·5 g/dl, white cell count $6{\cdot}8\times10^9$/l, and platelets 87×10^9/l. A plasma coagulation screen was normal, and an autoantibody screen to tissue antigens yielded negative results. In the third trimester her platelet count fell to 17×10^9/l. Bone marrow examination at this time showed increased megakaryocytes. A direct immunofluorescence test for platelet antibodies yielded positive results, and platelet associated IgG was raised at 62 ng/10^6 platelets (enzyme assay n = 2·5 – 15·8). Antiplatelet antibody was not detected in serum.

Autoimmune thrombocytopenia was diagnosed, and in view of persistently low platelet counts of about 10×10^9/l and recurrent epistaxis prednisolone 40 mg daily was started. After seven weeks of treatment her platelet count was still dangerously low at 7×10^9/l, and in the 38th week of pregnancy IgG infusion (Sandoglobulin) was started at a dose of 0·4 g/kg/day for five days, according to the manufacturer's instructions. On the fifth day of infusion she had a spontaneous rupture of membranes and 28 hours later was delivered of a normal baby girl, whose platelet count was normal at 254×10^9/l. The maternal platelet count had risen marginally to 25×10^9/l and by the following day was 175×10^9/l. Maternal blood one and seven days post partum showed considerably raised concentrations of platelet associated IgG (80 ng and 130 ng/10^6 platelets, respectively), and antiplatelet antibody was detected in maternal serum. Cord blood concentrations of platelet associated IgG were also raised at 50 ng/10^6 platelets. The puerperium was uneventful, but the maternal platelet count decreased and three weeks after delivery was 49×10^9/l.

Comment

Reports since 1950 on 91 patients with autoimmune thrombocytopenia and 138 pregnancies have shown a high incidence of maternal (2%), fetal (11%), and neonatal deaths (5%). Maternal haemorrhagic complications occurred in 22% of patients in one series,[2] and neonatal thrombocytopenia occurred in 43% in another.[3] Territo *et al* recommended caesarean section in patients with platelets counts below 100×10^9/l to avoid fetal cerebral trauma during vaginal delivery, as the incidence of neonatal thrombocytopenia in this group was 79%.[4] Neonatal thrombocytopenia due to placental transfer of platelet autoantibodies may occur, however, when maternal counts are normal,[3 4] and Ayromlooi suggested obtaining platelet counts via fetal scalp vein sampling.

High dose immunoglobulin crosses the placental barrier and ensures normal platelet levels in both the mother and fetus. Precisely how a remission is induced is still not clear and was reviewed by Newland *et al*.[1] In contrast to their findings, however, we showed a rise in platelet associated IgG in the mother after infusion of IgG. Furthermore, although antiplatelet antibody was not found in maternal serum before infusion, it was detected after delivery, and this might be because further antibody binding on the maternal platelets had been prevented. Thus in addition to macrophage Fc receptor blockade[1] a more direct but unexplained effect on platelets themselves by infused IgG may play a part.

IgG infusion was probably started a few days later than it should have been in our patient. Although considerable rises in platelet counts occur in most patients with autoimmune thrombocytopenia on the fifth day of infusion, peak increases are usually reached four days after the end of infusion and the response is maintained thereafter for a mean of 24 days.[1] We suggest that infusion of IgG should be started 10-15 days before the expected date of delivery. This has the added advantage that caesarean section, if indicated, may be undertaken with relative safety.

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