therefore be a particular aspect of a wider problem. Established pre-eclampsia may also be associated with a high maternal serum α -fetoprotein concentration,^{25 26} although earlier reports suggested an overall reduction in values.^{27 28}

The small number of cases available for study limits interpretation of the results. It appears that pre-existing hypertension is not a predisposing factor in the development of severe early onset pre-eclampsia. The risk factors that we have been able to identify are: a previous history of pre-eclampsia, a history of infertility, a history of headaches—particularly migraines—and a raised serum α -fetoprotein concentration at the time of screening for neural tube defects.

References

- Butler NR, Bonham DG. *Perinatal mortality*. Edinburgh: E and S Livingstone Ltd, 1963:86-100.
- Chamberlain G, Philipp E, Howlett B, Masters K. *British births*. London: William Heinemann Medical Books Ltd, 1970:80-107.
- Chesley LC. *Hypertensive disorders in pregnancy*. New York: Appleton-Century-Crofts, 1978:427.
- Billewicz WZ, Kemsley WFF, Thomson AM. Indices of adiposity. *British Journal of Preventive and Social Medicine* 1962;16:183-8.
- Stewart A, Hewitt D. Toxaemia of pregnancy and obesity. *Journal of Obstetrics and Gynaecology of the British Empire* 1960;67:812-8.
- Lowe CR. Toxaemia and pre-pregnancy weight. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1961;68:622-7.
- Butler NR, Alberman ED. *Perinatal problems*. Edinburgh: E and S Livingstone Ltd, 1969:36-9.
- Baird D. Epidemiological aspects of hypertensive pregnancy. *Clin Obstet Gynaecol* 1977;4:531-48.
- Rotton WN, Sachtleben MR, Friedman EA. Migraine and eclampsia. *Obstet Gynecol* 1959;14:322-30.
- Wainscott G, Sullivan FM, Volans GN, Wilkinson M. The outcome of pregnancy in women suffering from migraine. *Postgrad Med J* 1978;54:98-102.
- Walters WE, O'Connor PJ. Epidemiology of headache and migraine in women. *J Neurol Neurosurg Psychiatry* 1971;34:148-53.
- Walters WE. Headache and blood pressures in the community. *Br Med J* 1971;i:142-3.
- Bulpitt CJ, Dollery CT, Carne S. Change in symptoms of hypertensive patients after referral to hospital clinic. *Br Heart J* 1976;38:121-8.
- Pickering GW. *High blood pressure*. London: Churchill, 1968:324.
- Gant NF, Daley GL, Chand S, Whalley PJ, Macdonald PC. A study of angiotensin II pressor response throughout primigravid pregnancy. *J Clin Invest* 1973;52:2682-9.
- Franchi G, Anselmi B, Michelacci S. Change in serotonin and catecholamines vascular reactivity during migraine attack. *Res Clin Stud Headache* 1972;3:304-9.
- Appenzeller O, Davison K, Marshall J. Reflex vasomotor abnormalities in the hands of migrainous subjects. *J Neurol Neurosurg Psychiatry* 1963;26:447-50.
- Hanington E, Jones RJ, Amess JAL, Wachowicz B. Migraine: a platelet disorder. *Lancet* 1981;ii:720-3.
- Redman CWG, Bonnar J, Beilin LJ. Early platelet consumption in pre-eclampsia. *Br Med J* 1978;i:467-9.
- Miller D, Waters DD, Warnica W, Szlachcic J, Kreeft J, Theroux P. Is variant angina the coronary manifestation of a generalised vasospastic disorder? *N Engl J Med* 1981;304:763-6.
- Coffman JD, Cohen RA. Vasospasm—ubiquitous? *N Engl J Med* 1981;304:780-2.
- MacGillivray I. Some observations on the incidence of pre-eclampsia. *Journal of Obstetrics and Gynaecology of the British Empire* 1958;65:536-9.
- Long PA, Abell DA, Beischer NA. Fetal growth retardation and pre-eclampsia. *Br J Obstet Gynaecol* 1980;87:13-8.
- Wald NJ, Cuckle HS, Boreham J, Turnbull AC. Maternal serum alpha-fetoprotein and low birthweight. *Br J Obstet Gynaecol* 1980;87:860-3.
- Clayton-Hopkins JA, Oslen PN, Blake AP. Maternal serum alpha-fetoprotein levels in the pregnancy complicated by hypertension. *Prenatal Diagnosis* 1982;2:47-54.
- Kahlil FK, Bonnet M, Guibaud S, Combet A, Thoulon JM, Dumont M. Alpha-fetoprotein levels in placenta, maternal and cord blood in normal and pathologic pregnancy. *Obstet Gynecol* 1979;54:117-9.
- Rodeck CH, Campbell S, Biswas G. Maternal plasma alpha-fetoprotein in normal and complicated pregnancies. *Br J Obstet Gynaecol* 1979;83:24-32.
- Garoff L, Seppälä M. Toxemia of pregnancy: assessment of fetal distress by assaying estriol and circulating human placental lactogen and alpha-fetoprotein levels. *Am J Obstet Gynecol* 1976;126:1027-33.

(Accepted 24 May 1983)

SHORT REPORTS

Rebreathing during use of the Air-Viva resuscitation bag: a hazard

Self inflating resuscitator bags used for ventilatory support usually contain a valve arrangement to separate inspired from expired gases. On inspection the valve of the Air-Viva bag appeared inadequate to prevent the rebreathing of expired gas. We decided to test this hypothesis and also to test other readily available bags.

Methods and results

The following resuscitation bags were examined: Airox, Air-Viva, Air-Viva 2, Ambu Compact, Ambu Universal, Laerdal, Ohio, PMR, PMR 2, Penlon, Robertshaw, Sabre/Sussex, and Vitalograph. The bags were tested with an International Standards Organisation lung of standard specifications (compliance 0.02 l/cm H₂O and resistance 20 cm H₂O/l/s); carbon dioxide was introduced into the lung at 200 ml/min to simulate the patients' carbon dioxide production. The lung was then ventilated with each resuscitator bag. The respiratory flows produced were measured with a pneumotachograph (Hewlett Packard) and the signal integrated to give tidal volume. Carbon dioxide concentrations were measured continuously with a Datex infrared carbon dioxide meter with printer. The gas for this analysis was sampled from the patient port of the resuscitator. The bags were compressed at a fixed rate of 20 breaths/min for two five minute periods. During the first period the bags were squeezed with one hand to simulate normal use. To test the effect of increased tidal volume two hands were used for the second period. The Air-Viva bag was also tested over a longer period (15 mins) and with supplementary oxygen. Three other Air-Viva bags were picked at random from various departments of the hospital and submitted to the same tests.

When compressed with one hand all the bags produced tidal volumes in

the range of 400-650 ml; two hands increased the volume range to 500-800 ml. All the bags tested, apart from the Air-Viva, produced satisfactory results at both tidal volumes with zero carbon dioxide during the inspiratory phase (figure). With the Air-Viva, however, the inspired carbon dioxide concentration rose immediately—that is, rebreathing occurred—and after 20-30 seconds reached a plateau of 2%, which was then maintained throughout the five and 15 minute periods. Increasing the tidal volume did not influence this rise in inspired carbon dioxide. The three other Air-Viva bags tested showed the same pattern. Use of increasing flows of supple-

Top: Tidal carbon dioxide traces with Air-Viva and some satisfactory bags. Bottom: Effect of added oxygen on inspired carbon dioxide concentration with Air-Viva, oxygen flows 0-10 l/min.

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 superseded 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.

¹ Sykes M. Rebreathing during controlled ventilation with the Magill attachment. *Br J Anaesth* 1959;31:247-57.

(Accepted 12 May 1983)

Anaesthetic Department, Royal United Hospital, Bath, Avon BA1 3NG

STEPHEN L HILL, FFARCS, senior registrar
JENNIFER M EATON, FFARCS, senior registrar

Correspondence to: Dr S L Hill.

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 $6.8 \times 10^9/l$, and platelets $87 \times 10^9/l$. 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 $17 \times 10^9/l$. 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 $10 \times 10^9/l$ and recurrent epistaxis prednisolone 40 mg daily was started. After seven weeks of treatment her platelet count was still dangerously low at $7 \times 10^9/l$, 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 $254 \times 10^9/l$. The maternal platelet count had risen marginally to $25 \times 10^9/l$ and by the following day was $175 \times 10^9/l$. 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 $49 \times 10^9/l$.

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 $100 \times 10^9/l$ 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 blockade¹ 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.

We thank Dr T W Poole (Sandoz) for providing immunoglobulin for infusion.

¹ Newland AC, Treleaven JG, Minchinton RM, Waters AJ. High dose intravenous IgG in adults with auto immune thrombocytopenia. *Lancet* 1983;i:84-7.

² Heys RF. Child bearing and idiopathic thrombocytopenic purpura. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1966;73:205-14.

³ Noriega-Guerra L, Aviles-Miranda A, De la Cadena OA, Espinosa LM, Chavez F, Pizzuto J. Pregnancy in patients with auto immune thrombocytopenic purpura. *Am J Obstet Gynecol* 1979;133:439-48.

⁴ Territo M, Finkelstein J, Oh W, Calvin H, Kattlove H. Management of auto immune thrombocytopenia in pregnancy and in the neonate. *Obstet Gynecol* 1973;41:579-84.

⁵ Ayromlooi J. A new approach to the management of immunologic thrombocytopenic purpura in pregnancy. *Am J Obstet Gynecol* 1978;130:235-6.

(Accepted 17 May 1983)

Royal Sussex County Hospital, Brighton BN2 5BE

G R MORGENSTERN, MRCPATH, consultant haematologist
B MEASDAY, FRCS, FRCOG, consultant obstetrician and gynaecologist

Ealing Hospital, Southall, Middlesex UB1 2HW

U M HEGDE, MRCPATH, consultant haematologist

Correspondence to: Dr U M Hegde.