lapsed into a drowsy state. Lumbar puncture at another hospital had shown heavily blood-stained cerebrospinal fluid with xanthochromia. On admission she was drowsy and confused, and showed a severe dysphasia. She had a mild right hemiparesis. Her blood pressure was 140/80 mm Hg. Bilateral carotid angiogram showed a small irregularly-shaped aneurysm arising from the supraclinoid part of the left internal carotid artery close to the point of origin of the ophthalmic artery (Fig 2). There was no cross-flow between the two sides of the head. Because of the evidence of severe left hemisphere damage no surgical treatment was carried out. She remained drowsy, confused, incontinent, and dysphasic for three weeks, after which time her condition steadily improved. She was discharged home on 14 October. When last seen as an outpatient in March 1972 she had fully recovered apart from a very mild expressive dysphasia. She was running her home and doing a part-time office job.

Comment

Although subarachnoid haemorrhage from a ruptured intracranial aneurysm is a common condition, and a congenital weakness in the arterial wall is often postulated as an aetiological factor, there is little evidence that genetic influences play any part in the production of the disease. Chambers *et al.*, (1954) were the first to report the occurrence of ruptured intracranial aneurysms in two members of a family, a father and son. There have been 14 further reports of familial incidence of ruptured intracranial aneurysm. The literature is well reviewed by Bannerman and Ingall (1970) and also by Kak *et al.* (1970). In all, these reports are of a total of 45 patients, not a large number when one considers how common the disease is in the community.

There is only one reference to the condition in twins. O'Brien (1942) described a 34-year-old man who had died from subarachnoid haemorrhage from a middle cerebral artery aneurysm. His identical twin had died eight years earlier from an acute cerebral illness. Although the diagnosis given on his death certificate was brain abscess, it seems very likely from the history that he too had had a subarachnoid haemorrhage. No postmortem had been done so that the diagnosis was never proved.

The two cases reported here are, therefore, the first proved example of subarachnoid haemorrhage occurring in twins. There seems no doubt that they are identical twins. They have always looked alike and had similar personalities. Blood group tests (including ABO, MNS, P, Rh, Kell, and Duffy groups) showed identical reactions, indicating a 99·5% chance of their being monozygous twins (W. J. Jenkins, 1969, personal communication). A further parallel in these two patients is the fact that both aneurysms ruptured during the same decade of life, indeed within a time interval of just over two years. Neither of them suffered from hypertension, co-arctation of the aorta, or polycystic disease of the kidneys, conditions which are known to predispose to the formation of intracranial aneurysms. Finally, both patients had aneurysms at the same point on the Circle of Willis—that is, at the junction of the internal carotid artery and the ophthalmic artery. This is an uncommon site for aneurysm formation. Only 57 such aneurysms were found out of a total of 2,951 ruptured intracranial aneurysms collected in a recent co-operative study (Locksley, 1966), an incidence of 5·2%.

The likelihood that all these circumstances were coincidental seems so remote that it must be concluded that in this pair of identical twins some common genetic factor was involved.

I would like to thank Dr. W. J. Jenkins, director of the North-east Metropolitan Regional Blood Transfusion Centre at Brentwood, for his help in studying the blood groups of these patients.

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Bilateral Pneumothoraces and Subcutaneous Emphysema: A Complication of Internal Jugular Venepuncture

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The problem of taking blood from babies is well known. When a superficial vein cannot be found, as is often the case, alternative sites are sought—such as the superior saggital sinus, femoral veins, internal and external jugular veins, and the subclavian veins. Hazards are encountered at all these sites, and we report one unexpected complication of internal jugular venepuncture.

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Case Report

A 2-day-old Indian boy was transferred to this hospital because of umbilical bleeding. He had been born normally at term, weighing 6 lb 13 oz (3,090 g). The mother was a healthy primigravida with an uneventful antenatal course. The baby required no resuscitative measures at birth.

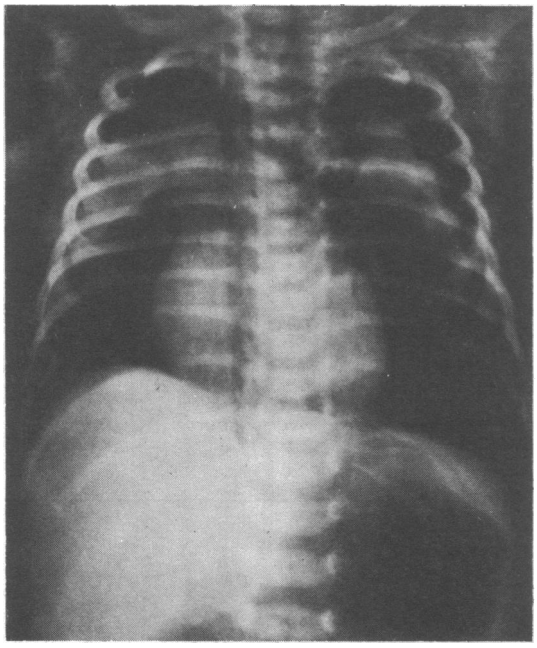
On admission he was found to be a normal child with some venous oozing from the umbilicus. Right internal jugular venepuncture was performed by an experienced operator without incident using the following procedure. The baby's head was extended and turned to the left, his shoulders being firmly held on a flat surface. A Leur disposable 21 S.W.G. needle, with attached syringe, was introduced deep to the lateral border of the right sternomastoid at the point where it is crossed by the superficial jugular vein. The needle was directed towards the suprasternal notch.

Blood cultures taken at the time proved to be sterile, and haemoglobin, white cell count, and film were within normal limits. Over the subsequent two days he was treated with pressure dressings to the umbilicus and 0·5 mg phytomenadione (vitamin K) intramuscularly. The oozing stopped.

On the third day of admission a further venous sample of blood was required to check the prothrombin time, and an attempt was made to obtain this by the same operator and by the same route.

Air was aspirated initially, the needle was withdrawn, and a swab was placed over the puncture site. Seconds later a diffuse swelling characteristic of surgical emphysema appeared in the neck, beginning in the suprasternal notch. There were no signs of respiratory distress and the baby seemed otherwise well. Two hours later the child became dyspnoeic. It was thought that this was due to trachael compression from the emphysema. Despite there being no stridor, an endotracheal tube was inserted. By this time respiratory efforts were poor and the child was manually ventilated for about 10 minutes with good response. Adequate spontaneous respiration then resumed with the tube in place, but over the next 15 minutes his condition deteriorated and was not improved by further ventilation. Decreased chest movement on the right side suggested the possibility of a tension pneumothorax on that side and an apical chest drain was rapidly inserted with immediate improvement. A chest *x*-ray picture five minutes later (see Fig.) showed bilateral pneumothoraces. A second drain was therefore inserted on the left side. The subcutaneous emphysema was also apparent.

Intrapleural air was extracted with a low suction (2-3 mm Hg) Robert's pump connected to the air space above the underwater drain. Whenever the suction was discontinued during the first 48 hours the pneumothoraces recurred.



Radiograph showing bilateral pneumothoraces.

The child's initial improvement was maintained and within two hours he was able to feed normally with no respiratory embarrassment. The intercostal drains were removed at 72 hours and 96 hours on the left and right respectively. He made an uneventful recovery, suffering no permanent sequelae.

Comment

Unilateral pneumothorax has been reported as a complication of internal jugular vein cannulation (English *et al.*, 1969) and of supraclavicular subclavian venepuncture (Walker and Sanders, 1969). Bilateral pneumothorax is a known complication of tracheostomy (Parikh, 1965) and mediastinoscopy (Furgang and Saidman, 1972). Massive subcutaneous emphysema and pneumothorax has been described after transincothyroid bronchography (Won *et al.*, 1967).

It is also well known that bilateral pneumothorax can be produced by overforceful ventilation (Rastogi and Wright, 1969; Miller and Hamilton, 1970) but we think it is unlikely in this case because respiratory distress was present before intubation and ventilation.

We feel that the most likely sequence of events here was that the mediastinal trachea was entered and slightly lacerated by the needle. This resulted in subcutaneous emphysema in the neck together with mediastinal emphysema, and when the intramediastinal pressure had risen sufficiently rupture of both mediastinal pleurae occurred producing bilateral pneumothoraces. This would agree with some of the experimental hypotheses of Macklin and Macklin, 1937. It is thought possible that the needle could have pierced the right apical pleura causing subcutaneous emphysema and a right pneumothorax, and the left could have been secondary to intermittent positive-pressure ventilation. Our impressions are that there were two key factors in the management of this case. Firstly, rapid reassessment of the diagnosis when it became clear that ventilation was not effective on the second occasion, and, secondly, recognition that underwater drains in neonates are not often successful without suction (Ramchandra and Russell, 1972)—a feature learned by anxious experience in the present case.

Internal jugular venepuncture is widely used in paediatric practice and we feel it is important to re-emphasize some of the dangers of this technique.

We should like to thank Mr. E. T. Murray, under whose care this patient was admitted, for permission to report this case and for helpful criticism.

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