SHORT REPORTS

Value of measuring cord blood bilirubin concentration in ABO incompatibility

Risemberg *et al*¹ suggested that "the risk to infants with ABO incompatibility and cord blood bilirubin levels greater than 68 $\mu\text{mol/l}$ (4 mg/100 ml) is so consistent that frequent re-evaluation is mandatory." As this was not our impression we studied cord blood bilirubin concentrations in 2117 consecutive births at Hope Hospital.

Methods and results

A 2-ml sample of cord blood was obtained without milking the cord and stored in a heparinised plastic tube. When necessary the sample was kept overnight for not more than 10 hours in a refrigerator protected from light. The bilirubin concentrations were measured in the hospital biochemistry department by an American Optical Corporation No 10200 bilirubinometer, a direct reading bilirubinometer that correlates well with spectrophotometric methods.² The mother's blood was grouped on the following indications: (1) jaundice on the first day; (2) a cord blood bilirubin concentration of 68 $\mu\text{mol/l}$ (4 mg/100 ml) or more; (3) severe hyperbilirubinaemia—that is, 273 $\mu\text{mol/l}$ (16 mg/100 ml) at 12–36 hours of age. Blood was taken from the infant for haemoglobin and direct Coombs test at the same time.

Out of the 2117 infants 212 (10%) required treatment for jaundice. ABO incompatibility was defined as follows³: mother group O and infant group A, B, or AB; mother group A and infant group B, or vice versa; presence of anti-A or anti-B haemolysins.⁴ Haemolysins were present in 48 (22.6%) jaundiced infants, all of whom had anti-A haemolysin. Other possible causes of jaundice were excluded as far as possible. None of the 212 infants had a cord blood bilirubin concentration above 68 $\mu\text{mol/l}$, range 2 $\mu\text{mol/l}$ to 51 $\mu\text{mol/l}$, mean ($\pm$ SD) 8 $\mu\text{mol/l} \pm 6.0$ $\mu\text{mol/l}$ (range 0.10 mg/100 ml to 3 mg/100 ml ± 0.35 mg/100 ml), but two had concentrations of 51 $\mu\text{mol/l}$ (3 mg/100 ml) and 47 $\mu\text{mol/l}$ (2.75 mg/100 ml) respectively. The infant with cord blood bilirubin of 51 $\mu\text{mol/l}$ required one exchange transfusion when his serum bilirubin was 346 $\mu\text{mol/l}$ (20.25 mg/100 ml). He was the only one who required an exchange transfusion in this group (table). The subsequent bilirubin concentrations in these 212 infants ranged from 150 $\mu\text{mol/l}$ to 346 $\mu\text{mol/l}$ (8.75 mg/100 ml to 20.25 mg/100 ml), mean 198.5 $\mu\text{mol/l} \pm 46.4$ $\mu\text{mol/l}$ (11.50 mg/100 ml ± 2.75 mg/100 ml). The Coombs test was positive in 15 (31.2%) infants with ABO incompatibility.

Relationship between cord blood bilirubin concentrations in 48 infants and degree of hyperbilirubinaemia (insignificant, moderate, or severe) at 36–48 hours, Coombs test, and exchange transfusion

	Serum bilirubin concentrations ($\mu\text{mol/l}$), range and mean ($\pm$ SD)		
	150–171 (163 $\pm$ 6.4)	171–257 (203.7 $\pm$ 23.1)	273 (328 $\pm$ 25.2)
No with cord bilirubin concentration:			
<68 $\mu\text{mol/l}$	10	36	2
>68 $\mu\text{mol/l}$	—	—	—
No Coombs test positive	—	13	2
Exchange transfusion	—	—	1

Conversion: SI to traditional units—Bilirubin: 1 $\mu\text{mol/l} \approx 0.06$ mg/100 ml.

Comment

There are no definite criteria of ABO haemolytic disease that could predict immediately after birth which infants may require treatment for hyperbilirubinaemia, yet increased pressure on hospital beds and early discharge policies make early diagnosis important. Risemberg *et al*¹ suggested that a cord blood bilirubin concentration of 68 $\mu\text{mol/l}$ was significant. Robinson *et al*⁵ thought that a concentration of 51 $\mu\text{mol/l}$ was highly suggestive of ABO haemolytic disease. In our 48 cases no baby had a concentration over 68 $\mu\text{mol/l}$ and only one infant had a concentration of 51 $\mu\text{mol/l}$. There were 38 (79.1%) infants who had concentrations under 51 $\mu\text{mol/l}$ who required treatment, which we gave as phototherapy. In all infants who had insignificant bilirubinaemia—that is, 171 $\mu\text{mol/l}$ (10 mg/100 ml) or

less—the Coombs test was negative. We consider that the cord blood bilirubin level is unreliable for predicting hyperbilirubinaemia due to ABO incompatibility.

I thank the late Mr Frank Warburton, of the biochemistry department, Sister McDermontt, Mr Allan Ashurst, and Dr D I K Evans for help in preparing this report, and Miss Susan Morris for typing the manuscript.

- ¹ Risemberg, H M, *et al*, *Archives of Disease in Childhood*, 1977, **52**, 219.
- ² Evans, R T, and Holten, J B, *Annals of Clinical Biochemistry*, 1970, **7**, 104.
- ³ Honig, G R, and Schulman, Irving, in *Pediatrics*, ed A M Rudolph, 16th edn, p 1198. New York, Appleton-Century-Crofts, 1977.
- ⁴ Dacie, J V, and Lewis, S M, *Practical Haematology*, 5th edn, p 542. Edinburgh, Churchill Livingstone, 1975.
- ⁵ Robinson, G C, Dunn, H G, and Wong, L C, *Acta Paediatrica*, 1960, **49**, suppl 120, 53.

(Accepted 27 September 1978)

Department of Neonatal Paediatrics, Hope Hospital, Salford M6 8HD

K N HAQUE, MRCP, DCH, registrar (now assistant professor of paediatrics, Medical College Riyadh, PO Box 2925, Saudi Arabia)

Spontaneous pneumomediastinum complicating anorexia nervosa

We report two cases of apparently spontaneous pneumomediastinum occurring in girls with anorexia nervosa.

Case reports

(1) A 15-year-old girl presented as an emergency to the casualty department with weight loss and weakness. She weighed 52 lb (23.6 kg) and was 61 in (1.53 m) tall. Anorexia nervosa had been diagnosed six months previously at another hospital, but she had defaulted from outpatient follow-up after three months, when she weighed 70 lb (31.8 kg). She had many features of the condition—cachexia, scaphoid abdomen, preservation of the breasts, lanugo, erythema ab igne, bradycardia (pulse rate 44/min), hypotension (blood pressure 80/50 mm Hg), pretibial petechial rash, and denial of illness. The diagnosis was consistent with Feighner's criteria.¹

Chest radiographs 24 hours after admission showed pneumomediastinum and pneumopericardium. Later surgical emphysema of the neck developed. Meanwhile, a nasogastric feeding tube had been passed without difficulty. There was no history of lung disease, hyperventilation, or self-induced vomiting and barium swallow and meal radiographs were normal. The nasogastric tube was left in situ, she remained asymptomatic, and within five days the surgical emphysema had resolved and the x-ray pictures were normal. She made a good recovery and weighed 84 lb (38 kg) when transferred to a psychiatric unit six weeks later. Within seven weeks normal menstruation had resumed.

(2) A 17-year-old girl was admitted to hospital with a four-month history of weight loss, amenorrhoea, anorexia, and vomiting and many of the classical features of anorexia nervosa. Her weight was 91 lb (41.4 kg) and her height 61 in (1.53 m). The diagnosis was in accordance with Feighner's criteria. Surgical emphysema of the neck was noted and a chest radiograph showed mediastinal emphysema. Barium swallow and meal radiographs and panendoscopy of the respiratory tract and oesophagus showed no abnormality. She recovered without further complication. The emphysema resolved both clinically and radiologically within seven days. She weighed 93 lb (43.8 kg) when discharged to psychiatric outpatients.

Comment

We conclude, in the absence of other causes, that in the first case pneumomediastinum developed spontaneously. Vomiting may have been a contributory factor in the second case. One case of emphysema, pneumomediastinum, and pneumoretroperitoneum in functional anorexia has been reported,² but, in an extensive search, we have not

found any other reports of pneumomediastinum as a complication of anorexia nervosa, starvation, or wasting diseases. Studies on rats of lung ultrastructure and mechanics after prolonged starvation have shown an increase in alveolar surface forces and a decrease in tissue elasticity, with fewer lamellar bodies in pulmonary granular pneumocytes (the storage site for surfactant) and a decrease in volume density of mitochondria in the same cells.³ Perhaps comparable changes occur in the human lung after starvation and account for this complication in anorexia nervosa.

We thank Dr J R Rees, United Bristol hospitals, and Dr A D Perrett, Royal Cornwall Hospital, for permission to report these cases under their care.

¹ Feighner, J P, *et al*, *Archives of General Psychiatry*, 1972, **26**, 57.

² Al-Mufti, N S, and Bevan, D H, *British Journal of Clinical Practice*, 1977, **31**, 160.

³ Sahebji, H, Vassallo, C L, and Wirman, J A, *American Review of Respiratory Disease*, 1978, **117**, 77.

(Accepted 12 September 1978)

Department of Medicine, British Royal Infirmary, Bristol BS2 8HW

ANITA J DONLEY, MB, MRCP, senior house officer (present address: Southmead Hospital, Bristol)

Royal Cornwall Hospital, Triliske, Truro, Cornwall

T J KEMPLE, MB, DCH, senior house officer in medicine

Strokes and hypertension: contribution of poor blood pressure control

There is a well-established relation between the incidence of strokes and the adequacy of blood pressure control in hypertension.¹ There is, however, little information available on the reasons for poor control in hypertensive patients who have a stroke,² and we have investigated the problem further.

Patients, methods, and results

All patients under the age of 75 who were admitted to hospital over eight months with a completed stroke (signs persisting for more than 24 hours) were seen by one of us (PK). Whether or not the patient was hypertensive was assessed on the basis of clinical history and examination, chest x-ray and electrocardiograph, reference to previous hospital notes, and interviews with relatives. General practitioners who did not know the aims of the investigation were asked by letter about previous blood pressure readings and treatment. The response rate was 88%. Relatives were also asked about how regularly the patient took his drugs. A diastolic blood pressure of 110 mm Hg or more was the criterion used for hypertension, and in most of the previously diagnosed cases several readings were available. Patients were considered to have had good blood pressure control if a diastolic blood pressure of 100 mm Hg or less had been recorded within the previous six months, and a history was obtained of regular medication until the stroke.

Of the 65 patients investigated, 35 (27 men) proved to be hypertensive (age 47-74, median 66), and only three of these were not previously known to have hypertension. Twelve (38%) of the 32 known hypertensive patients were believed to have had well-controlled blood pressure before the stroke. No fewer than 11 patients had discontinued hypotensive medication on the advice of their doctor (seven patients) or spontaneously (four patients) up to six years beforehand and in five cases within two weeks to a month before the stroke. Two other patients admitted to taking their drugs only intermittently, and three patients had never had treatment. Four patients had had unsatisfactory blood pressure control in spite of taking regular medication.

Comment

The assessment of previous blood pressure control is difficult even in patients regularly attending hypertension clinics, which only four of our patients were doing. Our figure of 62% for the incidence of unsatisfactory blood pressure control in patients who suffer hypertensive strokes is, however, close to the figure of Beevers *et al* of 70%.¹ In a review of the "obstacles to controlling hypertension" Stason *et al*³ found that failure to start treatment and to maintain it

were the most important. The clinician must also accept some responsibility for the non-compliance of patients to prescribed medication. The need for and the importance of life-long treatment is often not emphasised enough, unnecessarily complicated drug regimens are prescribed and too little attention is paid to side effects.

Our study was started after one of the patients at our hypertension clinic suffered his second stroke because of non-compliance and poor blood pressure control; we had failed to recognise that this was due to impotence from methyldopa. Pharmacological resistance was also noted, but it appears to be less important than the other reasons for poor control.

In our study nearly one-sixth of the hypertensive strokes occurred within one month of stopping treatment. A relation between stroke and stopping hypotensive therapy has a theoretical basis in loss of autoregulation of cerebral blood flow with fairly acute short-term rises in blood pressure.⁴ This suggests that our observation may be important clinically. Nevertheless, it seems unreasonable to think that the potential danger of stopping hypotensive treatment should discourage the initiation of treatment when otherwise indicated.

Only three of our patients presented with completed strokes and undiagnosed hypertension. These findings provide evidence that more effort needs to be paid to ensuring adequate treatment of patients already known to have hypertension than to finding new cases. In every hypertensive patient who suffers a stroke the previous management of the hypertension should be reviewed. In this way local obstacles to controlling hypertension can be identified and possibly overcome and the incidence of stroke reduced. The means of ensuring adequate treatment need to be widely publicised.⁵

We thank our colleagues in hospital and general practice for their helpful collaboration.

¹ Beevers, D G, *et al*, *Postgraduate Medical Journal*, 1973, **49**, 905.

² World Health Organisation, *Hypertension and Stroke Control in the Community*. Geneva, WHO, 1976.

³ Stason, W B, Weinstein, M C, and Shepard, D S, in *Hypertension: A Policy Perspective*, ed M C Weinstein and W B Stason, p 111. Cambridge, Mass, and London. Harvard University Press, 1976.

⁴ Russell, R R W, *Lancet*, 1975, **2**, 1283.

⁵ Stamler, R, *et al*, *Lancet*, 1975, **2**, 1227.

(Accepted 27 September 1978)

Whittington Hospital, London N19

P G E KENNEDY, MB, MRCP, medical registrar

B I HOFFBRAND, DM, FRCP, consultant physician

Cardiac pain referred to site of previously experienced somatic pain

Cardiac pain may be referred at times to the site of pain from lesions in the head and neck. For example, the pain of angina may be felt in the same distribution as pain from cervical spondylosis or from the teeth (for instance, a periapical abscess).^{1 2} More rarely, cardiac pain may be atypically referred to the site of pain previously experienced in the chest or upper limbs. We describe three such cases.

Case reports

(1) A 56-year-old man, a clerk, presented with asymptomatic essential hypertension in 1960 and was treated with methyldopa. In 1965 he complained of back pain at the level of T4 which was strictly related to exertion and was relieved by rest and by trinitrin. He had no precordial pain. An ECG showed inferior ischaemic changes. In 1968 he had an inferior myocardial infarction, but even then pain was confined to the dorsal region over T4. A radiograph showed an old compression fracture of T4. On direct questioning he said he had injured his back in falling from a horse 20 years previously. The patient died suddenly after his discharge home. There was no necropsy.

(2) A 56-year-old solicitor had had left thoracic herpes zoster 30 months previously. He had mild postherpetic neuralgia, but was otherwise well. In September 1975 his neuralgic pain suddenly worsened and was accompanied by sweating and nausea but no precordial pain. Because the pain was similar, though more severe, he thought it was due to an exacerbation of his herpes zoster. He attended the emergency department, where he was found