- ² Hathaway WE, Mahasandana C, Makowski EL. Cord blood coagulation studies in infants of high-risk pregnant women. *Am J Obstet Gynecol* 1975;**121**:51-7.
- ³ Condie RG. Components of the haemostatic mechanism at birth in pre-eclampsia with particular reference to fetal growth retardation. *Br J Obstet Gynaecol* 1976;**83**: 943-7.
- ⁴ Cederholm-Williams SA, Spencer JAD, Wilkinson AR. Plasma levels of selected haemostatic factors in newborn babies. *Thromb Res* 1981; **23**:555-8.
- ⁵ Ekelund H, Hedner U, Nilsson IM. Fibrinolysis in newborns. *Acta Paediatr Scand* 1970;**59**:33-43.
- ⁶ Biland L, Duckert F. Coagulation factors of the newborn and his mother. *Thromb Diath Haemorrh* 1973;**29**:644-51.
- ⁷ Kries RV, Jurgens H, Voss HV, Gobel U. The clinical relevance of factor VIII:C and factor VIII:A determination in newborns. *Eur J Pediatr* 1981;**137**:189-94.

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Expiration induced femoral flow in neonatal coarctation of aorta

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SUMMARY In a 3 day old infant with coarctation of the aorta a loud Doppler signal synchronous with expiration was present for some hours in the femoral vessels, with only barely audible signals synchronous with cardiac systole. It is suggested that in the presence of severe aortic constriction and temporary ductal closure, blood was pumped through the infradiaphragmatic arteries by increased intrathoracic expiratory pressure.

We believe that the following phenomenon may not have been reported before.

Case report

A baby girl aged 72 hours was transferred from another hospital because of pallor and pyrexia. Her birth had been normal, at term, and she weighed 3.82 kg. At 12.15 am the infant was slightly cyanosed, there was forceful grunting expiration at a rate of 56/min, her cardiac rate was 150/min, and her liver edge was palpable 4 cm below the costal margin in the right nipple line. Praecordial and epigastric pulsation were prominent but femoral pulsation was not detected. The blood pressure in both arms was 150 mm Hg systolic (Doppler ultrasound, 3.5 cm cuff). When the Doppler probe was placed sequentially over the femoral arteries a loud signal synchronous with expiration was heard over each artery and very faint signals synchronous with cardiac systole were audible on increasing the volume of the ultrasound. The expiration induced signal was heard at a lower intensity in the right popliteal fossa and was abolished by 20 mm Hg pressure in a 5 cm cuff on the thigh.

Digoxin, frusemide, and oxygen were given and arrangements were made for transfer to hospital

in Dublin later that morning. About 8 hours later blood pressure in the right arm was 120 mm Hg and the expiration induced signal had been replaced in the groins by one synchronous with systole. The femoral systolic artery pressure was 70 mm Hg. As she was being transferred cardiac arrest occurred.

At necropsy the essential findings were a hypoplastic aortic arch with a tight infantile coarctation immediately distal to the left subclavian artery. There was a patent but narrow ductus. The other relevant findings were right ventricular dilation and hypertrophy, anatomically patent but functionally closed foramen ovale, and normal thoraco-abdominal aorta. The aortic valve was tricuspid and no endocardial fibroelastosis was seen.

Discussion

During the initial observations blood flow in the femoral arteries coincided with expiration and virtually no flow synchronous with cardiac systole was detectable. We presume that at that time ductal closure occurred which added to the severe aortic constriction and severely restricted flow into the descending aorta. We suggest that the flow detected in the femoral arteries was induced by the pumping action of a high intrathoracic expiratory pressure on the descending aorta, sufficient to produce a pressure of at least 20 mm Hg. Since an expiratory intrathoracic pressure of 92 cm H₂O (67.6 mm Hg) has been recorded in a neonate¹ and pressure of about 240 cm H₂O (176.5 mm Hg) in an adult male undergoing physical stress,² it seems reasonable that with retrograde flow largely prevented by aortic and ductal constriction, high intrathoracic pressure could pump blood in the descending aorta downwards. This would imply that sufficient blood was

reaching the aorta through the coarctation, the ductus, and collateral vessels to be pumped caudally. When the final observations were made the more usual femoral flow synchronous with systole presumably resulted from intervening dilatation of the ductus. Expiration induced femoral flow may be useful as an indicator of ductal closure in coarctation.

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References

- 1 Milner AD, Saunders RA, Pressure and volume changes during the first breath of human neonates. *Arch Dis Child* 1977;52:918-24.
- 2 Altmann PL, Dittmer DS. *Respiration and circulation*. Federation of American Societies for experimental biology: Bethesda, 1971:86.

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Serum thyroxine and thyroid stimulating hormone values in unreferred children with short stature

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SUMMARY Of 449 children aged 6-9 years with heights below the first centile in a total population of 48 221, only 1 had previously undiagnosed hypothyroidism. In a community with well developed health services hypothyroidism is unlikely to be the cause of short stature among primary school children.

Primary hypothyroidism has been described as the commonest endocrine cause of short stature.¹ This was tested in a total population study designed to find the prevalence of growth hormone deficiency.

Subjects and methods

The screening of height in this group has been described.^{2,3} Many children were excluded because of previous negative studies (10), previous diagnosis of an organic cause (82), insufficient residual blood (6), and parental refusal (77). Two hundred and sixty seven children had thyroxine (T4) and 274 thyroid stimulating hormone (TSH) estimations. Serum T4 and TSH values were determined by radioimmunoassay with between assay coefficients of variation of 11.7 and 5.1 percent respectively. Bone age was determined by radiographic examination of the left wrist.

Results

Serum T4. The distribution of T4 concentrations is shown in Table 1. Values did not differ appreciably in boys and girls (mean (SD) 116.5 (21.4) nmol/l (9.1 (1.7) µg/dl) and 118.3 (17.9) nmol/l (9.2 (1.4) µg/dl)). Those with values exceeding 161 nmol/l had normal TSH values and were clinically euthyroid.

There was no correlation with sex, age, height, or bone age. Six children had values in the lower range for normal adults (75-80 nmol/l) and of these, 2 were being treated for coeliac disease. Another had a marginally raised TSH (7.3 mU/l), the upper limit of normal being 5.7 mU/l. He had been a 'light for dates' twin, of 42 weeks' gestation, and of below average intelligence. He was clinically euthyroid.

Serum TSH values. The distribution of TSH values shown in Table 2 approximates to that of a log normal curve.

One 7 year old girl had a value of 98.4 mU/l. Her parents were small. One paternal relative had thyrotoxicosis and 2 others were type I diabetics. Her hair was dry and she was often sleepy but there were no other features and her IQ was 107 (Wechsler intelligence scale). Bone age was 3 years retarded

Table 1 *Distribution of serum thyroxine (T4) values in 267 children aged 6-9 years*

Serum thyroxine (nmol/l)	Boys	Girls	Total
<70	—	—	—
71-80	5	1	6
81-90	9	7	16
91-100	22	8	30
101-110	29	22	51
111-120	28	24	52
121-130	28	27	55
131-140	12	14	26
141-150	13	7	20
151-160	2	3	5
>161*	5	1	6
Total	153	114	267

Conversion: SI to traditional units—thyroxine 1 nmol/l ≈ 0.08 µl/dl. *Five children had T4 values >162 nmol/l and were clinically euthyroid. Their T4 and TSH values (TSH normal value ≈ 2.2 mU/l), were; 170 (3.7), 172 (2.0), 175 (4.3), 190 (5.1), and 210 (5.4).