instrumental deliveries. The figure shows that this would logically demand attendance at all spontaneous vaginal deliveries with fetal distress, since this group has a higher proportion of low or medium Apgar scores than non-rotational forceps deliveries without fetal distress. This decision would entail attendance at 37% of term deliveries but would yield 78% of all term infants with low or medium Apgar scores. Including preterm deliveries, this policy would mean attending 19 deliveries a week in our hospital studied, out of about 2300 deliveries a year. Attending 10% and 20% of term deliveries as outlined previously would mean attending a total of eight and 12 deliveries a week respectively.

The results may be used to guide the reader in which categories of delivery should be attended to produce the most effective work for the manpower available.

Two other aspects of our study require discussion.

Rotational forceps—Chiswick and James reported a similar number of rotational forceps deliveries to ours.³ The incidence of fetal distress in both studies was 40%. In their study the incidence of delayed respiration at birth was considerably increased when fetal distress had been present, whereas in our study the high incidence of a low or medium one minute Apgar score (16%) was unaffected by fetal distress. Our findings are supported by other recent work.⁴ Since we have shown that our definition of fetal distress is associated with a decreased Apgar score in other forms of delivery, this finding suggests that a mid-cavity rotational forceps delivery is inherently associated with a decreased chance of a high Apgar score. This could be a risk associated with the failure to rotate spontaneously, or with the procedure itself, rather than the indication for its use.

Caesarean section—When infants with fetal distress were excluded the incidence of low one minute Apgar scores in our study was similar whether the caesarean section was performed under general or epidural anaesthesia. Our findings and our overall rate of low Apgar scores are similar to those reported.⁵ The interval between induction and delivery, which is an important factor,⁶ was not analysed in either study, but the

findings suggest that increasing care is being taken to avoid prolonged general anaesthesia.

Breech delivery was associated with an increased incidence of low Apgar scores in our study. Breech presentation and delivery are hazardous,⁷ and clearly these deliveries have priority for paediatric attendance.

The overall incidence of low or medium Apgar scores after spontaneous vertex deliveries in our study was 4.2%. This is similar to the 5.3% reported in a large American collaborative study,⁸ but less than the findings of another study, where 7.3% of infants had an Apgar score of below 6 at one minute after a spontaneous vaginal vertex delivery.⁴ Since this group had a policy of less intervention in the second stage of labour the results are not strictly comparable.

We did not set out to compare the benefit or risk to comparable infants of different modes of delivery. Our aim was to define "at risk" groups in obstetric practice that can be detected antenatally, so that a data base can be provided for rational decisions about perinatal paediatric services.

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References

- 1 Robson E, Hey E. Resuscitation of preterm babies at birth reduces the risk of death from hyaline membrane disease. *Arch Dis Child* 1982;57:1184-6.
- 2 Anonymous. The value of the Apgar score [Editorial]. *Lancet* 1982;i:1393-4.
- 3 Chiswick ML, James DK. Kielland's forceps: association with neonatal morbidity and mortality. *Br Med J* 1979;ii:7-9.
- 4 Cardozo LD, Gibb DHF, Studd JW, Cooper DJ. Should we abandon Kielland's forceps. *Br Med J* 1983;287:315-7.
- 5 Zagorzycki MT, Brinkman CR III. The effect of general and epidural anaesthesia upon neonatal Apgar scores in repeat caesarean section. *Surg Gynecol Obstet* 1982;155:641-5.
- 6 Stenger V, Blechner J, Prystowsky H. A study of prolongation of obstetric anaesthesia. *Am J Obstet Gynecol* 1969;103:901-7.
- 7 Calvert JP. Intrinsic hazards of breech presentation. *Br Med J* 1980;281:1319-20.
- 8 Drage JS, Kennedy C, Schwarz BK. The apgar score as an index of neonatal mortality. A report from the collaborative study of cerebral palsy. *Obstet Gynecol* 1964;24:222-30.

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SHORT REPORTS

Ulnar nerve palsy in a haemophiliac due to intraneural haemorrhage

Peripheral nerve lesions are a common complication of haemophilia, occurring in 10-13.6% of patients.^{1,2} The aetiology of these lesions has been reported to be intramuscular or intra-articular haemorrhage with subsequent extrinsic nerve compression.³ We report a case of ulnar nerve palsy due to intraneural haemorrhage and consider the implications of this finding for diagnosis and treatment.

Case report

A 9 year old boy with haemophilia presented after receiving a bump at play to the right elbow. He had severe haemophilia with a factor VIII concentration of 1-2% and required regular factor VIII replacement treatment on a home treatment programme. The results of regular screening tests for factor VIII inhibitors were negative. He had experienced few previous haemarthroses affecting the elbows. On examination the elbow joint was normal and there was no local bruising or swelling. The ulnar nerve was clinically intact. A radiograph of the elbow was normal. Treatment with antihemophilic factor was given and he was allowed home.

The next day he developed paraesthesiae on the medial border of the right hand but did not present again until four days after the original injury. At this time there was reduced sensation over the medial two fingers of the right hand and ulnar surface of the palm. The muscles of the hand innervated by the ulnar nerve were weak and he had developed a claw hand. No bruising, swelling, or tenderness was detected at the elbow or elsewhere in the arm. Factor VIII replacement treatment was started and surgical ulnar nerve decompression undertaken. Under tourniquet control a posteromedial incision was made and the cubital tunnel exposed. No free blood was in

the cubital tunnel when it was opened, but the nerve was hyperaemic for 2-3 cm and had a bluish discoloration. The epineurium was divided, with escape of free blood.

After surgery the wound healed well with no further bleeding. The next day normal sensation in the ulnar nerve distribution and a full range of finger movements had returned. Factor VIII treatment was continued for 10 days. At the time of his discharge he had full ulnar nerve function on clinical testing. About two weeks after surgery nerve conduction studies showed that motor conduction across the right elbow segment was slightly slower than normal and that on the left side. Ulnar sensory action potentials were present at the wrist but not at the elbow on the right side. Repeat studies three months later showed some remaining relative delay in cross elbow motor conduction but with considerable improvement since the first study.

Comment

Reported causes of peripheral nerve lesions in haemophilia include intramuscular, intra-articular, and perineural soft tissue haemorrhage and nerve compression from large pseudotumours. The nerve affected most often is the femoral, in association with an iliacus haematoma,⁴ followed by the ulnar nerve, which accounts for 17.2% of peripheral nerve lesions.¹ The cause of these lesions is thought to be extrinsic compression. This is the first report of a peripheral nerve lesion due to intraneural haemorrhage in a haemophiliac. The ulnar nerve at the elbow is susceptible to direct trauma, but in this case there were no local findings to confirm this as the site of nerve injury. The lack of localised swelling, bruising, or bony injury should not detract from the diagnosis, and the spontaneous, isolated development of an ulnar nerve palsy in a haemophiliac should prompt investigation for possible intraneural haemorrhage.

Previous reports of peripheral nerve lesions in haemophilia have only rarely recorded or recommended surgery. Ehrman found that 37.5% of peripheral nerve lesions in his study showed only partial or no recovery.¹ The rapid and complete clinical recovery shown in this case suggests that when intraneural haemorrhage is possible early surgical decompression with adequate factor VIII replacement is the treatment of choice.

¹ Ehrman L, Lechner K, Mamoli B, Novotny C, Vos K. Peripheral nerve lesions in haemophilia. *J Neurol* 1981;225:175-82.

² Silverstein A. Neuropathy in hemophilia. *JAMA* 1964;190:554-5.

³ Duthie RB, Matthews JM, Rizza CR, Steel W. Peripheral nerve lesions. In: *The management of musculoskeletal problems in haemophiliacs*. Oxford: Blackwell Scientific Publications, 1972:63-74.

⁴ Goodfellow J, d'Afearn CB, Matthews JM. Iliacus haematoma, a common complication of haemophilia. *J Bone Joint Surg [Br]* 1967;49:748-56.

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Diabetogenic effects of nifedipine

Nifedipine is widely used to treat angina pectoris and hypertension. Some studies of its effects on carbohydrate metabolism and insulin secretion have shown it to be potentially diabetogenic,^{1,2} although another study found that it had no appreciable effect on glucose tolerance.³ We report on one patient in whom diabetes deteriorated and another in whom it developed during the use of nifedipine; in both cases these effects disappeared after withdrawal of the drug. We have not been able to find any other such reports, and neither the adverse drug reaction monitoring team in Kuwait nor the manufacturers of nifedipine have been notified of such side effects.

Case reports

CASE 1

A 56 year old man was diagnosed as having diabetes mellitus in 1982. He had had hypertension and type IV hyperlipidaemia since 1969 and a myocardial infarction in 1978. His diabetes was controlled by diet and he was prescribed propranolol 320 mg/day, chlorthalidone 50 mg thrice weekly, isosorbide dinitrate 30 mg/day, and clofibrate 2 g/day. In August 1983 his fasting plasma glucose concentration was 7.9 mmol/l (142.4 mg/100 ml), and oral nifedipine 30 mg/day was started to control his hypertension and mild angina. Ten days later he complained of fatigue and polyuria, and his fasting plasma glucose concentration was found to be 30.8 mmol/l (555 mg/100 ml). He was admitted to hospital, and insulin injections were started.

Examination did not show any abnormality. His blood pressure was 120/80 mm Hg and his temperature normal. Blood tests showed: white cell count $7.5 \times 10^9/l$, erythrocyte sedimentation rate 61 mm in the first hour, blood urea concentration 19 mmol/l (114 mg/100 ml), creatinine concentration 336 $\mu\text{mol/l}$ (3.8 mg/100 ml), and normal serum electrolyte concentration. Urine and throat cultures were sterile.

Despite twice daily injections of insulin zinc suspension (Monotard) and neutral insulin (Actrapid), his plasma glucose concentrations during the day were 19.6-22.4 mmol/l (353-404 mg/100 ml) with no ketonuria. Three days later nifedipine was stopped. His plasma glucose concentrations fell gradually and the doses of insulin were tapered down. He was discharged, taking a small dose of insulin, when his fasting plasma glucose concentration was 8.2 mmol/l (148 mg/100 ml). During follow up insulin was withdrawn, and two months later his fasting plasma glucose concentration was 5.4 mmol/l (97 mg/100 ml) without any medication.

CASE 2

A 67 year old man, hypertensive since 1973 and taking propranolol 180 mg/day, had suffered a myocardial infarction in the same year. His fasting plasma glucose concentration was 5.5 mmol/l (99 mg/100 ml) in December 1982, and oral nifedipine 20 mg/day was started for angina. A month later he started to complain of polyuria, and his fasting plasma glucose concentration was 10.4 mmol/l (187 mg/100 ml). No cause could be found for this sudden development of diabetes mellitus. During the next few months his fasting plasma glucose concentration remained between 8.8 and 9.8 mmol/l (159 and 177 mg/100 ml). Nifedipine was stopped in November 1983 when his symptoms became worse. Three days later his fasting plasma glucose concentration was 5.2 mmol/l (94 mg/100 ml) and he stopped complaining of polyuria, although his angina worsened.

Comment

The administration of nifedipine to both patients was associated with raised plasma glucose concentrations, which returned to normal after this drug was withdrawn. We could not find any other factor in either patient to explain these changes. In view of reports that nifedipine may be diabetogenic,^{1,2} we conclude that it was responsible for the deterioration in glucose homeostasis in these two patients. Verapamil, another calcium antagonist, has been experimentally shown to inhibit the release of insulin.⁴ Although more studies are needed to confirm the effect of nifedipine on glucose tolerance, we suggest that plasma glucose concentrations should be monitored in diabetic and non-diabetic patients receiving this drug.

¹ Giugliano D, Torella R, Cacciapuoti F, Gentile S, Verza M, Varricchio M. Impairment of insulin secretion in man by nifedipine. *Eur J Clin Pharmacol* 1980;18:395-8.

² Charles S, Ketelslegers J-M, Buysschaert M, Lambert AE. Hyperglycaemic effect of nifedipine. *Br Med J* 1981;283:19-20.

³ Donnelly T, Harrower ADB. Effect of nifedipine on glucose tolerance and insulin secretion in diabetic and non-diabetic patients. *Curr Med Res Opin* 1980;6:690-3.

⁴ Devis G, Somers G, Obberghen EV, Malaisse WJ. Calcium antagonists and islet function. I. Inhibition of insulin release by verapamil. *Diabetes* 1975;24:547-51.

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Traumatic dislocation of the hip in mini rugby

Traumatic dislocation of the hip in children is rare and seldom presents to an individual surgeon. Most reports have combined the experience of several surgeons. We report two cases of traumatic dislocation of the hip incurred in mini rugby, both of which presented to the same orthopaedic surgeon within six months.

Case reports

Case 1—A 10 year old boy was kneeling, having been tackled, when he was knocked forwards on to his hands and knees by another player who then sat on his right buttock. The posterior dislocation thus caused was reduced with ease under general anaesthesia within a few hours. He then spent four weeks in skin traction, and the joint remained non-weightbearing for a further two months, when he used crutches. His recovery was without complication: within eight months he had regained a full and painless range of movement of the hip and was back in the rugby team.

Case 2—A 10 year old boy was injured after scoring a try. He was on his hands and knees when another player jumped on his back, causing a posterior dislocation of the hip. Reduction was achieved with ease within three hours of injury. He was immobilised in fixed traction with a Thomas splint for four weeks, and the joint remained non-weightbearing for a further two months, when he used crutches. Six months after the injury his hip was normal, both clinically and radiologically.

Comment

During games of mini rugby both these boys sustained a posterior dislocation of the hip without any associated fracture; neither showed any complication during the subsequent nine months. Posterior dislocation of the hip usually occurs when a force is directed proximally up the shaft of the femur from the knee to the flexed hip. This can happen in younger children who fall on to their knees or in older children who suffer greater trauma—for example, by falling from a height or in road traffic accidents. In our cases the knee was pressed to the ground and the dislocating force was applied to the pelvis via another player's body weight. This mechanism of injury has been described previously in a child aged under 2 who was on his knees and elbows when his father fell on his back.¹

The major complications of traumatic dislocation of the hip include avascular necrosis of the femoral head, traumatic arthritis, coxa