PAPERS AND ORIGINALS

Perinatal Coagulopathies Complicating Fetal Blood Sampling

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Summary

In a neonatal postmortem histological study of scalp stabs made with a fetal blood sampling blade the aponeurosis was punctured in up to 38% of cases even when it was more than 2 mm deep. This and other indirect and direct evidence discussed suggest that five of the seven reported cases of major bleeding after fetal blood sampling in labour were due to blood clotting defects and that these should be considered urgently in the management of future cases.

Introduction

Coagulation disorders in the newborn are being recognized with increasing frequency. Disseminated intravascular coagulation resulting in a consumptive coagulopathy accounts for many deaths in babies of low birth weight, but can also complicate perinatal asphyxia in mature infants (Chadd *et al.*, 1971; Markarian *et al.*, 1971). Haemorrhagic disease (*British Medical Journal*, 1971), due specifically to deficiency of the vitamin-K-dependent coagulation factors, usually occurs a few days after birth when these factors are at their lowest. When it occurs earlier it may be due to maternal anticonvulsant therapy (Evans *et al.*, 1970; Mountain *et al.*, 1970) but is usually spontaneous. Even healthy babies have a demonstrable deficiency of these factors at birth (Lanzkowski *et al.*, 1967; Bonnar *et al.*, 1971) and a case has been described of massive haemorrhage due to haemorrhagic disease occurring after fetal blood sampling in labour (Hull and Wilson, 1972).

Major haemorrhage after fetal scalp blood sampling occurs rarely, though fatal cases have been reported. Beard *et al.* (1966) attributed the cause of death in one of their two cases primarily to vascular damage beneath the epicranial aponeurosis. To avoid aponeurotic puncture most obstetricians use a blade only 2 mm long when taking a fetal blood sample. The purpose of this paper is to show that possible aponeurotic puncture is of relatively slight importance when excessive bleeding occurs

after fetal blood sampling, but that the bleeding is more likely to be due to a coagulation defect on which the obstetrician and paediatrician must focus their urgent attention.

To assess the significance of aponeurotic puncture its incidence was sought in postmortem scalp stabs made in a small series of babies who had died shortly before or after delivery near term. This could reasonably be assumed to be the incidence in fetal blood sampling practice and would be inversely related to the danger of such injury, since major complications are rare.

Method

A series of stabs in the scalp were made in a transverse line over the vertex using a standard blade as for fetal blood sampling no more than 2 mm wide and projecting 2 mm from a protective plastic shoulder. The stabbed tissue was examined microscopically after standard histological preparation and staining with haematoxylin and van Gieson. Sections were examined from multiple levels through each block of tissue. Some stabs were absent, in part or whole, from some sections; stabs seen in all sections were counted. Partly for this reason the apparent stabs outnumbered the actual stabs, but also it was possible to calculate only the minimum not all the actual stabs examined because some could not be separately identified.

Results

The bodies of four babies were used but one was excluded because the aponeurosis was separated from the skin by a haematoma due to difficult forceps delivery.

Case 1.—A male West Indian Negro baby weighing 3,800 g was delivered shortly after intrauterine death due to placental abruption at 39 weeks' gestation. The average thickness of the scalp from the epidermis to the aponeurosis was 2.7 mm (2.1-3.0 mm). The aponeurosis was punctured (see Fig. 1) in 39 out of 102 stabs seen (38%), from a minimum of 35 stabs made, up to a depth of 3.0 mm.

Case 2.—A white female baby weighing 2,670 g was born normally at 38 weeks' gestation and died within a few minutes. She had gross exomphalos and spinal deformity. The average scalp thickness was 2.3 mm (1.8-3.2 mm) and the aponeurosis was punctured (see Fig. 2) in 22 out of 106 stabs seen (21%), from a minimum of 46 made, up to a depth of 2.8 mm.

Case 3.—This was a white male baby weighing 2,350 g. He was the second of twins born normally at 39 weeks' gestation com-

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plicated by hypertension. He had a large diaphragmatic hernia and died within two hours. The average scalp thickness was 1.6 mm (1.2-1.8 mm) and the aponeurosis was punctured in 58 out of 77 stabs seen (75%), from a minimum of 44 made.

Despite the 2-mm blade used it is clear that the aponeurosis was very frequently punctured even beyond this depth, and it occurred up to 3.0 mm in Case 1. This was presumably due to the compression of the intervening fat, which is illustrated by the frequency with which the stab tracks appeared to be distorted or discontinuous (Figs. 1 and 2).

FIG. 1—Case 1. Photomicrograph of scalp. Aponeurosis punctured (below) at depth of 2.7 mm. Note apparently discontinuous track in subcutaneous layer due to distortion by compression. (Haematoxylin and van Gieson. $\times 26$.)

FIG. 2—Case 2. Photomicrograph of scalp. Adjacent stabs showing aponeurotic puncture in one (right) and apparently not in the other at depth of 1.9 mm. (Haematoxylin and van Gieson. $\times 26$.)

Discussion

There have been few, mostly minor, complications of fetal blood sampling (Kubli *et al.*, 1967; McDonald, 1967; Nelson *et al.*, 1971). Since aponeurotic puncture must occur very

frequently, as shown by the present study, it is likely to be of little significance. It is therefore possible to speculate some other cause of death in the case in which this injury was found by Beard *et al.* (1966). They described a second case in which they suspected a coagulation defect, but tests were made after death and following blood transfusion. Only one other fatal case has appeared in the literature but was not described (Kubli and Berg, cited by Saling, 1965). Balfour *et al.* (1970) described three cases of moderately severe bleeding; the two that they tested both had clotting defects, and one of these was fatal and was associated with severe perinatal asphyxia. Finally, there is the case of a massive subaponeurotic haematoma occurring after suture of bleeding stab sites due to haemorrhagic disease in a Negro baby described on p. 321 (Hull and Wilson 1972).

This last case is unique, but it shares many of the features of other cases of neonatal scalp haematomas severe enough to require blood transfusion and not associated with cranial damage. Only 58 have been reported (Aballi *et al.*, 1959; Leonard and Anthony, 1961; Pachman, 1962; Van der Horst, 1963; Valentine, 1964; Kozinn *et al.*, 1964; Kozinn *et al.*, 1965; Barrow and Peters, 1968; Robinson and Rossiter, 1968; Ahuja *et al.*, 1969). They invariably occurred within 24 or 48 hours of birth. The bleeding in nearly all cases was beneath the epicranial aponeurosis, where a very large volume can collect unnoticed, so that blood transfusion was often massive and amounted to half or more of the normal blood volume. The commonest clotting defect in those tested was prolongation of the prothrombin time, suggesting haemorrhagic disease. Where race was specified most were Negro, of whom half were delivered normally. By contrast the only cases of white babies affected occurred after vacuum extraction (Ahuja *et al.*, 1969).

The predominance of early haemorrhagic disease in Negro babies in the cases reviewed suggests strongly that this was the cause of bleeding in the two cases, both Negro, of Beard *et al.* (1966). Thus of the seven reported cases of major bleeding after fetal blood sampling a clotting defect was found in all three properly tested and a presumptive diagnosis of haemorrhagic disease can be made in two others.

The present analysis suggests that when excessive bleeding occurs after fetal blood sampling attention should be diverted from the stab and focused instead on a possible coagulation defect. In the absence of asphyxia this is likely to be deficiency of the vitamin-K-dependent clotting factors, particularly if the baby is Negro or if the mother is receiving anticonvulsant therapy.

In labour the possibility that vaginal bleeding may be due to fetal blood sampling must be tested promptly, and to this purpose endoscopy seems preferable to the tablet test of Chamberlain and Fosker (1970). If fetal bleeding is confirmed then it would be wise to have fresh blood cross-matched against the mother and to avoid instrumental delivery, particularly vacuum extraction.

At birth placentofetal blood transfusion should be encouraged and cord blood taken for haemoglobin estimation, cross-matching, and coagulation studies. If the prothrombin time is prolonged and blood is not required to be given, it might be advisable to infuse fresh frozen plasma (10 ml/kg) immediately because of the delayed action of vitamin K₁. This was recommended by Ahuja *et al.* (1969), who found that prophylactic vitamin K₁ acted too slowly to reduce the incidence of major and fatal haematomas occurring after vacuum extraction. Finally, the bleeding must be controlled only by prolonged pressure over the stab sites, which should not be sutured at least until a coagulation disorder has been excluded. Whether blade or needle has started the bleeding under the aponeurosis the sutures will certainly confine it there and massive haemorrhage may be hidden from view.

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Massive Scalp Haemorrhage after Fetal Blood Sampling Due to Haemorrhagic Disease

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Summary

Massive subaponeurotic haematoma occurred in a baby after suture of bleeding scalp blood sampling stabs made before delivery. Eighteen hours after delivery blood samples showed marked prolongation of the prothrombin time. The condition was successfully treated with vitamin K₁ and blood transfusion.

Introduction

Haemorrhagic disease of the newborn (*British Medical Journal*, 1971) is usually the result of a dietary deficiency of vitamin K in breast-fed babies and seldom occurs less than a few days after birth. A unique example, however, is described below, believed to be the first reported case of proved haemorrhagic disease complicating fetal blood sampling in labour.

Case Report

The mother was a healthy 30-year-old Negress who had one previous baby without complication and a normal second pregnancy. Labour occurred spontaneously in the 42nd week in February 1971. Progress was accelerated by forewater rupture at 5 cm dilatation of the cervix and she delivered normally six hours later after a short second stage. The amniotic fluid became lightly stained with meconium and a scalp blood sample was taken for measurement of pH, which was 7.26 (maternal 7.37). The bleeding was not unusually heavy but would not stop. Nevertheless, it was not sufficient to speed the fetal heart before delivery 40 minutes later.

Two scalp stabs had been made 1 cm apart over a parietal bone and at least 1.5 cm from the sagittal suture. The blade used was less than 2 mm wide and protruded 2 mm from the protective plastic shoulder.

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The baby, a boy, weighed 3,800 g and had an Apgar score of 10. He behaved normally at birth but continued to bleed slowly from the scalp stabs despite prolonged pressure over the area. After three hours the stabs were underpinned with three sutures of fine silk mounted on an atraumatic cutting needle. These stopped the visible bleeding and the baby seemed well, but nearly 15 hours later oozing recurred, his head was swollen, and he was very ill. He was shocked, had a slow heart beat, and his respiratory rate was 60/min. There was pitting oedema of the scalp overlying a generalized swelling which extended to the limits of the scalp and across all suture lines, typical of a huge haematoma beneath the epicranial aponeurosis. Eighteen hours after delivery blood was taken from the umbilical vein for coagulation studies. The prothrombin time was 110 seconds; the various results are given below. The haemoglobin was 8.7 g/100 ml, and there was a pronounced metabolic acidosis, presumably due to blood loss and anaemia.

Subsequently vitamin K₁ (total 9 mg) and antibiotics were given, intravenously at first, and bicarbonate, dextrose, and electrolyte solutions were infused appropriately. Fresh blood transfusions totalling 240 ml were given within 48 hours of delivery. After the first 180 ml of blood the haemoglobin fell further to 7.6 g/100 ml, probably owing both to haemodilution and to continued bleeding into the scalp. The haemoglobin then rose rapidly to 10.7 g/100 ml (venous) on the fourth day, and 12.4 g/100 ml (capillary) on the twelfth. The prothrombin time at 42 hours was 40 seconds, at 48 hours 21 seconds, on the third day 18 seconds, and on the fourth day was normal, 15 seconds. After the first critical 48 hours the baby improved rapidly. He took feeds on the third day and behaved normally from the fourth. By the time he went home on the twelfth day there was only a little boggy oedema of the scalp and periorbital oedema. His progress at 12 months remained normal.

There are two additional clinical points worth noting. Firstly, the child's head circumference was 34 cm at birth and did not reach its maximum until the third day, when it was 39 cm, suggesting that bleeding continued long after treatment started. Secondly, he was never jaundiced. The highest serum bilirubin was 5.9 mg/100 ml after 42 hours.

The coagulation studies made at 18 hours were as follows: prothrombin time (one-stage method) 110 sec (control, 15 sec), partial thromboplastin time with kaolin 130 sec (control, 60 sec), platelets 174,000/mm³, fibrinogen (semiquantitative measurement by thrombin titre using Fibrindex) titre 1/32 (control, 1/32); there was no excess of fibrin/fibrinogen degradation products. The gross prolongation of the prothrombin