Reproducibility of cerebral artery Doppler measurements

B Moorthy, P R Colditz, K N Ives, D G Rees, W G van’t Hoff, P L Hope

Abstract

Intraobserver and interobserver variability were calculated for the measurement of cerebral artery pulsatility indices by five observers who made two recordings on each of 12 stable preterm infants. Variations in pulsatility indices of up to 0.21 were found; no measurement was below 0.55.

There have been many studies of the cerebral circulation of newborn infants with Doppler techniques since the report by Bada et al in 1979, in which they used the Pourcelot or pulsatility index.1 Pulsatility index is defined as (S-D)/S, where S is systolic and D is diastolic flow velocity.2 It has been suggested that a low pulsatility index (<0.55) during the first five days of life is a useful prognostic sign, with a high sensitivity for the prediction of death or disability, in infants who have been subjected to severe birth asphyxia.2 The aim of this study was to assess the reproducibility of measurement of pulsatility indices by clinical staff using a commercial duplex scanner.

Patients and methods

Twelve stable preterm infants were each studied within a period of one hour by five observers, using a duplex Doppler scanner (Ultramark 4, Advanced Technical Laboratories). A real time image was obtained in the parasagittal plane, and a Doppler signal obtained at 5 MHz from the anterior cerebral artery anterior to the corpus callosum. Each observer made two separate recordings from the anterior cerebral artery of each infant, and stored the flow velocity waveforms on a hard copy. The hard copies were kept by one of the authors who, after collection of the data was complete, issued each observer with the 24 recordings he had made from the 12 infants. Observers were therefore unaware which were paired results from the same infant. Systolic and diastolic velocities were measured by rulor on the hard copy. Measurements were made to the nearest millimetre and the pulsatility index was calculated as the mean (S-D)/S of at least three waveforms.

Analysis of variance was used to study the 60 first observations by each observer on each infant.3 Estimates of the SD of both the intraobserver and interobserver variability were calculated, making the reasonable assumption of no interaction between baby and observer.

Results

The table shows the medians and ranges of values obtained by the five observers from each of the 12 infants. There was considerable variation between observers, the most extreme being between 0.72 and 0.93. None of the observers recorded a pulsatility index below the 0.55 threshold suggested by Archer et al.4 The estimates of the SDs associated with the intraobserver and interobserver variances were 0.046 and 0.030, respectively. These values may be interpreted as follows: (1) Of the differences between two independent measurements made by the same observer on the same patient, 95% will be less than 0.13 (=2.83×0.046) or 17% when expressed as a percentage of the mean pulsatility index.4 (2) Of the differences between two independent measurements made by two observers on the same patient, 95% will be less than 0.16 (=2.83×√0.046²+0.030²), or 20% as a percentage of the mean pulsatility index.

Analysis of the differences between the duplicate determinations by each observer showed that these differences were less than would be predicted if each was subjected independently to the calculated degree of intraobserver variability. The two measurements were therefore not truly independent, and the duplicate measurements were not used in the analysis.

Discussion

Previous studies of the reproducibility of measurement of pulsatility indices have been limited to comparison of results obtained by two or three experienced observers.5 6 As cerebral artery Doppler studies are now useful clinically

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Median and range of values for pulsatility indices of 12 infants

<table>
<thead>
<tr>
<th>Case No</th>
<th>Pulsatility indices</th>
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<td></td>
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<tr>
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<tr>
<td>11</td>
<td>0.75</td>
</tr>
<tr>
<td>12</td>
<td>0.78</td>
</tr>
</tbody>
</table>

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Virilisation of female preterm infants

P C Midgley, D Azzopardi, N Oates, J C L Shaw, J W Honour

Abstract
Two cases of hypertrophy of the clitoris in premature girls are reported; this was associated with persistently high concentrations of adrenal fetal zone androgens.

Isolated hypertrophy of the clitoris in neonates is known to be associated with congenital adrenal hyperplasia, administration of androgens to the mother in the second and third trimesters of pregnancy, and maternal androgen secreting tumours. We report two cases of progressive hypertrophy of the clitoris developing after birth, and associated with persistent excretion of high concentrations of 3β-OH-5-ene steroids, produced by the fetal adrenal zone and which are known to be weak androgens. This association has not previously been reported.

Case reports
Case 1
A girl, birth weight 640 g, was one of twins delivered at 24 weeks' gestation for antepartum haemorrhage; the mother was aged 24, para 1+1. The second twin died aged 11 hours.

The surviving twin developed hyaline membrane disease, and was ventilated for five weeks, remaining oxygen dependent for a further seven weeks. Apnoea of prematurity was treated with theophylline from 4 to 10 weeks. She had a patent ductus arteriosus, which failed to close with indomethacin, and led to treatment with digoxin and frusemide from 9 weeks until spontaneous closure at 11 weeks. She had recurrent hyponatraemia and required sodium supplements.

Clitoromegaly was noted at 4 weeks of age (fig 1A). She had a left inguinal hernia with a palpable gonad (a macroscopically normal ovary was later found at herniotomy). Ultrasound showed a normal uterus and adrenals. The karyotype was normal female. Plasma 17α-OH progesterone was 27-2 nmol/l at 7 weeks, 61-8 nmol/l at 9 weeks, and 11-2 nmol/l at 20 weeks. Adrenocorticotropic hormone (ACTH) was 4-8 pmol/l at 11 weeks and 6-6 pmol/l at 20 weeks (immunoradiometric assay, Eurodiagnostics, Netherlands). The plasma cortisol on five occasions, when not receiving hydrocortisone, ranged from <69 nmol/l to 316 nmol/l and after corticotrophin stimulation increased to >1380 nmol/l at 60 minutes. Plasma dehydroepian-

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