Respiration and Pulse Pressure Influence Cerebral Circulation in Infants with Peri-intraventricular Haemorrhage – Transcephalic Electrical Impedance and Mar-modelling Approach

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Abstract: Cerebral circulation was monitored cot-side in 42 neonates during the first day of life using transcephalic electrical impedance. Mechanical ventilation and pulse pressure compromised cerebral circulation in infants who developed peri-intraventricular haemorrhage by the end of the fourth day of life. Transcephalic impedance seems to provide a useful method for cot-side monitoring of cerebral circulation.

INTRODUCTION

The incidence of peri-intraventricular haemorrhage (PIVH) in infants below 1500 g and below the gestational age of 35 weeks has declined over the past decade from 55% to 26% but it still remains one of the most severe catastrophes confronting preterm neonates [1–4]. Thus the prevention of PIVH is a major goal of modern perinatal research and development. An ideal prerequisite for the early identification and intervention of the babies in danger for developing PIVH would be a quantitative, continuous and non-invasive, cot-side monitoring method based on parameters yielding an early warning of cerebral bleeding or ischaemia. A method allowing this is transcephalic electrical impedance plethysmography in combination with spectral analysis and multivariate autoregressive modelling of cardiorespiratory signals [5].

MATERIAL AND METHOD

Transcephalic electrical impedance (ΔZ), heart rate, arterial blood pressure (aBP) and respiration were intermittently studied at 8 hour intervals in 42 preterm (GA<36 weeks) infants during the first day of life [6]. The variability of the signals and the interrelations between them were analysed off-line in an IBM 486 PC using multivariate autoregressive (MAR) modelling (model order 30) to find parameters related to cerebral circulatory catastrophes [5]. Severe peri-intraventricular haemorrhage (PIVH) developed to 10 infants by the fourth day of life and echodensities to eight, two of whom developed to cystic periventricular leukomalacia. Echodensities were found in 8 newborns during the first week of life. The remaining 24 infants served as controls in this study. Statistical analysis between the three groups and between the time points was done with the unbalanced repeated measures model analysis of variance with structured covariance matrices (BMDP 5V) using restricted maximum likelihood. Multiple comparisons between the groups were done with Bonferroni and Tukey multiple comparisons tests (BMDP 7D).

RESULTS

The high-frequency (frequency of hear rate) pulsatile variability of ΔZ was lower in the PIVH group (207, SEM 33) than in the controls (740, SEM 202) (p=0.047) during the first day of life.

At the low-frequency range (0.07–0.18 Hz) the cross-contribution of pulse pressure to ΔZ was higher in the PIVH group (18%, SEM 4%) than in the controls (11%, SEM 1%) (p=0.042).

The cross-contribution of respiration to ΔZ corresponding to the rate of mechanical ventilation (0.18–0.80 Hz) was higher in the PIVH group (15%, SEM 1%) than in the controls (13%, SEM 1%) (p=0.046).

DISCUSSION

Rapidly fluctuating arterial blood pressure mediated to pressure passive cerebral circulation and leading to increased fluctuations of cerebral blood flow has been suggested as one of the primary causes of PIVH [7]. We could not entirely agree with this. The magnitude of rapid fluctuations in cerebral circulation was lower in the PIVH group than in the control group at the frequencies corresponding to the heart beat, which also agrees with our previous results [6]. An explanation for the decreased variability in the cerebral blood volume could be the increased intracranial pressure in infants with PIVH. Secondly, the decreased variability could be caused by the "no reflow phenomenon" described as severely decreased cerebral blood flow after ischaemic insult of the brain in neonatal animals and newborns [8, 9]. The "no reflow phenomenon" may be caused by arterial vasospasm, oedema or tissue necrosis.

CONCLUSION

We conclude that in infants with PIVH the neonatal cerebral circulation seemed to respond to
changes induced by respiration and pulse pressure more prominently than in infants without PIVH. This and the low amount of the rapid fluctuations of the cerebral circulation in the infants with PIVH could be related to the circulatory adjustments caused by the haemorrhage. Transcephalic electrical impedance together with MAR-modelling seems to provide a useful method for monitoring and detecting pathological changes in neonatal cerebral circulation.

REFERENCES


