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Haemodynamic impact of chest compressions in the delivery room

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Brief Report

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Abstract

Among 65 neonates with encephalopathy undergoing cooling, 30 (46.1%) received chest compressions during delivery room resuscitation. Despite differences in encephalopathy severity, early (<24 hours) biventricular function on echocardiogram (fractional area change, myocardial performance indices, systolic to diastolic duration ratios, tricuspid annular plane systolic excursion) was comparable between groups with and without chest compressions. Epinephrine receipt was associated with abnormal tricuspid annular plane systolic excursion.

The onset of breathing immediately after birth triggers the normal haemodynamic transition process in neonates, with a decrease in pulmonary vascular resistance, and an increase in left ventricular end-diastolic dimensions and stroke volume.[1] Birth asphyxia, or the failure to establish breathing at birth, accounts for nearly a quarter of all neonatal deaths worldwide and is associated with hypoxic–ischaemic encephalopathy. Cooling has demonstrated benefits in reducing death and disability in neonatal encephalopathy.[2] Up to 60–80% of neonates with encephalopathy have biventricular dysfunction, reduced cardiac output, and pulmonary hypertension on echocardiograms, compared to controls.[3–5] Asphyxiated infants with reduced cardiac output have associated reduced coronary flow.[6,7]

Neonatal resuscitation algorithms include sequentially, positive pressure ventilation, chest compressions, and epinephrine, and improve outcomes in asphyxiated neonates. Among infants with encephalopathy undergoing cooling, 39–63% receive chest compressions and epinephrine in the delivery room.[8,9] Whether chest compressions and epinephrine in the delivery room portend ventricular dysfunction remains unclear. We evaluate the effects of resuscitation on biventricular function on early echocardiograms, among infants with encephalopathy undergoing cooling.

Methods

This was a single centre retrospective review of medical records and echocardiograms in infants (gestational age ≥ 36 weeks) with moderate or severe encephalopathy, based on standard criteria, who were cooled to 33.5°C for 72 hours. The study groups comprised infants who received (a) chest compressions for ≥30 seconds and (b) epinephrine in the delivery room. Infants ≤ 1800 g birth weight, admitted after 12 hours of age, with major congenital abnormalities, CHD other than a patent ductus arteriosus, in whom aggressive treatment was limited, or who did not undergo an echocardiogram within 24 hours of age were excluded.

The following echocardiographic measurements were performed offline by a single experienced central reader (SA, paediatric cardiologist):

Right ventricular systolic function:

- Tricuspid annular plane systolic excursion was measured from 2D M-mode apical four-chamber view, with the cursor placed at the free wall of the tricuspid annulus, aligning the cursor as vertically as possible to the cardiac apex.[10]
- Fractional area change was calculated as the difference between areas in diastole and systole divided by the area in diastole.

Global myocardial function:

- Myocardial performance index was assessed separately for each ventricle by dividing the sum of isovolumic contraction and relaxation times by the ejection time.[3]
- Ratio of right ventricular systolic to diastolic duration was measured offline in triplicate from the best Doppler signal of tricuspid regurgitation on either apical four-chamber or parasternal long axis view. The systolic duration was calculated from the onset to the termination of tricuspid regurgitation and diastolic duration as the time between two jets. Heart rate was calculated from R–R interval.[5]

Table 1. Comparison of echocardiographic characteristics in the groups of neonates with encephalopathy who did and did not undergo delivery room chest compressions.

Mean (SD) or n (%)	Chest compressions (n = 30)	No chest compressions (n = 35)	p-value
Heart rate (bpm)	116 (19)	120 (28)	0.461
TAPSE	0.56 (0.25)	0.64 (0.24)	0.172
Abnormal TAPSE	18 (60%)	15 (42.9%)	0.216
LV MPI	0.43 (0.15)	0.41 (0.14)	0.553
RV MPI	0.50 (0.22)	0.51 (0.24)	0.868
Abnormal RV MPI	5 (16.7%)	5/33 (15.2%)	1.000
RV S/D	1.50 (0.41)	1.61 (0.51)	0.375
Abnormal RV S/D	13 (43.3%)	15 (42.9%)	1.00
RV fractional area change	30.3 (10.5)	33.6 (10.9)	0.224
Abnormal RV fractional area change	8 (26.7%)	10 (28.6%)	1.000
RV systolic pr (mm Hg)	(n = 18) 45 (14)	(n = 18) 49 (13)	0.434
Moderate or large PDA	20 (66.7%)	19 (54.3%)	0.053
PDA R-L/bidirectional	19/25 (76%)	16/26 (61.5%)	0.266
Moderate or large ASD	14 (46.7%)	10 (28.5%)	0.132
ASD R-L/bidirectional	11 (36.6%)	10 (28.5%)	0.487

ASD = atrial septal defect; LV = left ventricle; MPI = myocardial performance index; PDA = patent ductus arteriosus; R-L = right to left shunt; RV = right ventricle; S/D = ratio of systolic to diastolic durations; TAPSE = tricuspid annular plane systolic excursion

A high parasternal ductal view was utilised to assess presence, size, and direction of ductal shunt. Pulse Doppler at the junction of ductus and main pulmonary artery was used to assess the pattern of shunt.

Demographic data and clinical characteristics were collected.

Statistical analysis: Mean (SD) or median (IQR) echocardiographic data were compared between groups using t−tests, Mann–Whitney U, and chi-square tests, as appropriate. Statistical analysis were performed using SPSS Version 19 (SPSS, Chicago, Illinois, United States of America).

Results

Our cohort (n = 65) included 30 (46.2%) infants who received chest compressions or epinephrine in the delivery room and 35 (53.8%) who did not. The mean (SD) age at echocardiogram was 11.1 (6.3) hours. In the resuscitation group, 21 (70%) infants received epinephrine in addition to chest compressions. Infants in the resuscitation group had higher mean birth weight, were more often white, had severe rather than moderate encephalopathy and Apgar scores < 5 at 5 and 10 minutes more often (Table S1). Table 1 is a comparison of echocardiographic characteristics between groups who did and did not receive chest compressions. Right ventricular dysfunction was noted in 33 (50.8%) infants, all of whom had abnormal (<0.64) tricuspid annular plane systolic excursion. Fractional area change was <23 (more than 2 SD below the published norms) in 18 (27.7%) infants, and systolic to diastolic duration ratio was >1.45 in 28 (43.1%) infants and myocardial performance index > 0.72 in 10 (15.4%) infants. There were no differences in function between groups. Tricuspid annular plane systolic excursion was abnormal in 71% of infants (n = 21) who received epinephrine for resuscitation, compared to 21% of those (n = 44) who did not (p = 0.033) (Table 2). All other measures were comparable between groups.

Table 2. Comparison of echocardiographic characteristics in the groups of neonates with Hypoxic-ischemic encephalopathy who did and did not require Epinephrine for resuscitation.

Mean (SD) or n (%)	Epinephrine (n = 21)	No Epinephrine (n = 44)	p-value
Heart rate (bpm)	121 (16)	116 (27)	0.374
TAPSE	0.53 (0.24)	0.64 (0.24)	0.096
Abnormal TAPSE	15 (71.4%)	18 (20.5%)	0.033
LV MPI	0.44 (0.16)	0.40 (0.13)	0.238
RV MPI	0.52 (0.25)	0.51 (0.23)	0.728
Abnormal RV MPI	5 (16.7%)	5/33 (15.2%)	1.000
RV S/D	1.53 (0.42)	1.57 (0.48)	0.764
Abnormal RV S/D	12 (57.2%)	24 (54.5%)	0.568
RV fractional area change	29.7 (11.6)	33.2 (10.2)	0.223
Abnormal RV fractional area change	7 (33.3%)	11 (25.0%)	0.338
RV systolic pr (mm Hg)	(n = 14) 45 (12)	(n = 22) 50 (13)	0.064
Moderate or large PDA	17 (80.9%)	22 (50.0%)	0.01
PDA R-L/bidirectional	16/19 (84%)	19/32 (59.3%)	0.177
Moderate or large ASD	11 (52.4%)	13 (29.5%)	0.11
ASD R-L/bidirectional	9 (42.8%)	12 (27.3%)	0.261

ASD = atrial septal defect; LV = left ventricle; MPI = myocardial performance index; PDA = patent ductus arteriosus; R-L = right to left shunt; RV = right ventricle; S/D = ratio of systolic to diastolic durations; TAPSE = tricuspid annular plane systolic excursion

Discussion

Among 65 neonates with encephalopathy undergoing cooling, nearly half received chest compressions in the delivery room. Despite differences in encephalopathy severity, early (<24 hours) biventricular function on echocardiogram was comparable between groups with and without chest compressions. Epinephrine receipt was associated with abnormal tricuspid annular plane systolic excursion.

Post-resuscitation echocardiographic left ventricular dysfunction, including global systolic dysfunction (20%), regional wall motion abnormalities (7%), and apical ballooning involving all walls not limited to any single coronary territory (5%), is reported in 33–75% of adults with cardiac arrest.[11] However, left ventricular systolic dysfunction post-arrest has not consistently shown an association with outcomes and may simply reflect transient myocardial stunning.

The right ventricle has a different coronary supply, lower myocardial oxygen consumption, and the ability to increase oxygen extraction, which make it relatively ischaemia-resistant. In the current study in neonates, about half had evidence of echocardiographic right ventricular dysfunction. However, there were no

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significant differences between the groups with and without chest compressions, despite differences in the proportion of severe encephalopathy in the groups. This could be due to the small sample size or could suggest that the antecedent hypoxic–ischaemic event or cooling, rather than the resuscitation itself, is associated with ventricular dysfunction in term neonates. The resuscitation may have been relatively brief and recovery may have been related mainly to respiratory support. Infants who received epinephrine, possibly reflecting more prolonged resuscitation, were more likely to have abnormal tricuspid annular plane systolic excursion. Tricuspid annular plane systolic excursion is a robust measure of right ventricular free wall longitudinal deformation; we speculate that this abnormality may be an early marker of myocardial dysfunction, prior to changes in other measures of global right ventricular function.

We acknowledge the limitations of this study. While echocardiograms were done within 24 hours, timing did vary, and some were after induction of cooling. Right ventricular shape and geometry make it difficult to accurately quantify function. We did not have data available on the duration or quality of resuscitation. Nonetheless, a systematic echocardiographic investigation following resuscitation has not been previously undertaken in the neonate. We used newer quantitative load independent and reliable parameters, and a single central reader.

In conclusion, our data showed no significant difference in biventricular function with or without resuscitation, among neonates with moderate or severe encephalopathy.

Supplementary material. To view supplementary material for this article, please visit https://doi.org/10.1017/S1047951121005217

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Conflict of interest. None of the authors have any real or potential financial conflicts of interest to disclose.

Ethical standards. Research was conducted in accordance with the ethical standards of all applicable national and institutional committees and the World Medical Association's Helsinki Declaration.

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