ID 569. OBSERVED RANGES OF NEONATAL BLOOD PRESSURE BY GESTATIONAL AGE: A SYSTEMATIC REVIEW

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**BACKGROUND:**
Neonatal blood pressure is known to vary according to gestational age and to rise within the postnatal period. However, determination of standard values is particularly complicated for premature neonates considering many receive blood pressure modifying agents or have established conditions that affect blood pressure. Therefore, a systematic review was undertaken to determine the observed ranges of blood pressures by gestational age throughout the first 3 months postnatal age, when not treated with blood-pressure modifying agents nor affected by complicating pathology.

**METHODS:**
A systematic literature search was conducted within MEDLINE, Pubmed, Embase, Cochrane Library, and CI-NAH from 1946-2017 regarding blood pressure in neonates <3 months of age. (PROSPERO registration ID CRD42018092886).

**RESULTS:**
3587 non-duplicate manuscripts were screened, with 623 extracted for in-depth review. Of these, 123 were deemed relevant to our primary question and were reviewed for full data extraction, of which 24 studies were included. 3 manuscripts contained data for extremely premature neonates (<28 weeks), 4 manuscripts for very premature neonates (28 to 31+6 weeks), 2 manuscripts for moderately premature neonates (32 to 33+6 weeks), 5 manuscripts for late preterm neonates (34 to 36+6 weeks), and 19 manuscripts for term neonates (37+ weeks). Several manuscripts were excluded due to purely graphical presentation of data. Collated mean blood pressure (mmHg) was calculated from observed means for preterm vs term babies at 1-23h (Mean=44 vs 49), day1 (41 vs 45), day2 (43 vs 46), day3 (44 vs 54), day7 (48 vs 63), and 1 week – 3 months (66 vs 56).

**CONCLUSION:**
There remains a paucity of published data related to the assessment of neonatal blood pressures according to gestational age, particularly for prematurity. Example demonstrative statistics showed marked discrepancy between preterm and term blood pressure. However, meta-analysis of studies is complicated by variability in study summary statistics presented. Future multi-collaborative large-scale studies are needed to improve the evidence base for standard blood pressure values within all gestational age ranges.

No conflicts of interest.
ID 57. Echocardiography-guided ductus arteriosus treatment: A Randomized Controlled Trial on two prescription strategies to reduce the incidence of necrotizing enterocolitis

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Background. Patent ductus arteriosus (PDA) approach remains controversial due to uncertainties about treatment benefits versus harms. We aim to evaluate whether a treatment scheme on echography-guided (EchoG) PDA closure (to reduce drug exposure) and 24h-continuous ibuprofen infusion (24h-IB) (to reduce peak concentration of ibuprofen), compared to EchoG PDA closure plus conventional bolus ibuprofen treatment (bolus-IB), reduces severe bowel adverse event rate in infants below 33 weeks’ gestation with hemodynamically significant (hs) PDA.

Methods: Multicentre, blinded randomized controlled trial. Infants with less than 28 weeks’ gestation underwent routine echocardiographic assessment between 18h to 72h of birth; infants between 28 and 33 weeks were screened only in case PDA was suspected clinically. hsPDA was considered if ductal diameter was larger than 1.5 mm and indicators of pulmonary overflow, systemic hypoperfusion, or both were present.

Results. One hundred forty six infants (median gestational age 26 [25-28] weeks; median birth weight 881 [704-1100] g) were randomized to 24h-IB (n=70) or bolus-IB (n=76) study group at 86 (58-140) h from birth. Groups were comparable in terms of perinatal or neonatal relevant clinical data with the exception of higher prevalence of male sex in the bolus-IB group (p=0.004). Treatment effectiveness was also similar with 53% (24h-IB) and 47% (bolus-IB) of the infants showing no ductal flow after ibuprofen treatment with similar total number of doses. Severe bowel adverse event rates were also similar [10% (24h-IB); 2.6% (bolus-IB), p=0.1], although those in bolus-IB group reached full enteral nutrition earlier (p=0.03). Postnatal age (p=0.02) and peripheral SaO2 (p=0.004) at treatment start, and pulmonary hemorrhage (0.03) before PDA treatment were associated to the development of severe bowel events independently of treatment group allocation.

Conclusions. Ibuprofen intravenous continuous infusion compared to bolus infusion in preterm infants with hsPDA shows similar rates of success and does not reduce the prevalence of severe bowel events.

Funding source: This study was supported by the Spanish Health Ministry (PI16/00644) and Mutua Madrileña Foundation (AP163272016). The authors did not receive any form of payment to perform the trial.
Background: We planned this study to evaluate the incidence, spectrum, and outcomes of shock among neonates.

Methods: The study was done in a tertiary-care referral hospital of a low-and-middle-income country (LMIC) between 1st January 2018 and 31st December 2019. We enrolled all neonates developing shock during this period. We retrieved the data from the electronic database of our unit, examined case record summaries and case record files. We compared survivors and non-survivors to find independent predictors of mortality.

Results: We recorded 3271 neonatal admissions during the study period. Of them, 415 episodes of neonatal shock were recorded in 392 neonates. The incidence of neonatal shock was 12.0% [95% confidence interval (CI): 10.9-13.2%]. Of 415 episodes of neonatal shock, 237 (57%) episodes were identified as septic shock, 67 (16%) episodes as cardiogenic shock, and six (1.4%) episodes as obstructive shock. Rest 105 (25%) episodes had overlapping features of various forms of shock. The incidence of culture-proven septic shock was 32% (n=132; 95% CI: 27%-37%). Acinetobacter baumannii was the most common pathogen (n=58). There were 256 non-survivors in our series. The case fatality rate in our cohort was 62% (95% CI 57%-67%). The overall adjusted mortality rate of our unit during the corresponding period was 2.3% (after removing major congenital malformations, received compassionate care, and HIE–III). Characteristics of survivors and non-survivors are presented in Table 1. On univariate analysis, gestational age, birth weight, female gender, hyaline membrane disease, early-onset sepsis, Acinetobacter sepsis, and cardiogenic shock were significantly different between survivors and non-survivors. SGA neonates showed a trend of association with mortality. On multivariable logistic regression analysis, four variables—gestational age, small for gestational age, female gender, and Acinetobacter sepsis—showed an independent association with mortality in neonatal shock.

Conclusions: We observed a 12.0% incidence of shock among neonates admitted in a tertiary care referral hospital of an LMIC country. The incidence of septic shock was significantly higher than cardiogenic shock. The neonatal shock was associated with a high case fatality rate. Gestational age, small for gestational age, female gender, and Acinetobacter sepsis independently predicted mortality in neonatal shock.
None of the authors have any conflict of interest to resolve.

### Table 1: Characteristics of Survivors and Non-Survivors

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Characteristics</th>
<th>Non-Survivors (n=256)</th>
<th>Survivors (n=159)</th>
<th>Unadjusted OR (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Gestational age (wk)*</td>
<td>31.3 ± 4.0</td>
<td>33.2 ± 4.1</td>
<td>0.89 (0.85, 0.94)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2.</td>
<td>Birth weight (g)*</td>
<td>1175 (904, 1605)</td>
<td>1576 (1120, 2372)</td>
<td>0.99 (0.99, 1.00)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3.</td>
<td>Small of gestational age (%)</td>
<td>117 (46)</td>
<td>59 (37)</td>
<td>1.4 (1.0, 2.1)</td>
<td>0.1</td>
</tr>
<tr>
<td>4.</td>
<td>Female Gender (%)</td>
<td>119 (47)</td>
<td>55 (35)</td>
<td>1.6 (1.1, 2.4)</td>
<td>0.02</td>
</tr>
<tr>
<td>5.</td>
<td>Multigravidity (%)</td>
<td>58 (23)</td>
<td>34 (22)</td>
<td>0.9 (0.6, 1.7)</td>
<td>0.9</td>
</tr>
<tr>
<td>6.</td>
<td>Caesarean section/ instrumental delivery (%)</td>
<td>125 (49)</td>
<td>71 (45)</td>
<td>1.2 (0.8, 1.7)</td>
<td>0.5</td>
</tr>
<tr>
<td>7.</td>
<td>1-min Apgar &lt;7</td>
<td>151 (59)</td>
<td>79 (50)</td>
<td>1.4 (0.9, 2.1)</td>
<td>0.2</td>
</tr>
<tr>
<td>8.</td>
<td>1-min Apgar &lt;3</td>
<td>49 (19)</td>
<td>25 (16)</td>
<td>1.2 (0.7, 2.1)</td>
<td>0.4</td>
</tr>
<tr>
<td>9.</td>
<td>5-min Apgar &lt;3</td>
<td>4 (2)</td>
<td>5 (3)</td>
<td>0.5 (0.1, 1.8)</td>
<td>0.3</td>
</tr>
<tr>
<td>10.</td>
<td>Hyaline membrane disease (%)</td>
<td>86 (34)</td>
<td>31 (19)</td>
<td>2.1 (1.3, 3.3)</td>
<td>0.003</td>
</tr>
<tr>
<td>11.</td>
<td>Early onset sepsis (%)</td>
<td>150 (59)</td>
<td>74 (47)</td>
<td>1.6 (1.1, 2.4)</td>
<td>0.02</td>
</tr>
<tr>
<td>12.</td>
<td>Culture-proven sepsis (%)</td>
<td>89 (35)</td>
<td>43 (27)</td>
<td>1.4 (0.9, 2.2)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Any culture-positive (%)</td>
<td>45 (18)</td>
<td>13 (8)</td>
<td>2.8 (1.4, 5.8)</td>
<td>0.008</td>
</tr>
<tr>
<td></td>
<td>Acinetobacter sepsis (%)*</td>
<td>47 (18)</td>
<td>33 (21)</td>
<td>1.0 (0.6, 1.6)</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td>Other than Acinetobacter (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>Patent ductus arteriosus (%)</td>
<td>63 (25)</td>
<td>29 (18)</td>
<td>1.5 (0.9, 2.4)</td>
<td>0.1</td>
</tr>
<tr>
<td>14.</td>
<td>Pulmonary arterial hypertension (%)</td>
<td>18 (7)</td>
<td>19 (12)</td>
<td>0.6 (0.3, 1.1)</td>
<td>0.1</td>
</tr>
<tr>
<td>15.</td>
<td>Air Leaks (%)</td>
<td>30 (12)</td>
<td>14 (9)</td>
<td>2.0 (0.5, 7.4)</td>
<td>0.3</td>
</tr>
<tr>
<td>16.</td>
<td>Postnatal age at onset of shock (d)</td>
<td>3 (2.4)</td>
<td>3 (1.5)</td>
<td>1.0 (0.9, 1.0)</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>Septic shock</td>
<td>153 (60)</td>
<td>84 (53)</td>
<td>1.3 (0.9, 1.9)</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>Cardiogenic shock</td>
<td>31 (12)</td>
<td>36 (23)</td>
<td>0.5 (0.3, 0.8)</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td>Obstructive shock</td>
<td>4 (2)</td>
<td>2 (1)</td>
<td>1.2 (0.2, 6.8)</td>
<td>0.8</td>
</tr>
<tr>
<td></td>
<td>Mixed</td>
<td>69 (27)</td>
<td>36 (23)</td>
<td>1.2 (0.8, 2.0)</td>
<td>0.4</td>
</tr>
<tr>
<td>17.</td>
<td>Type of Shock (%)</td>
<td>23 (9)</td>
<td>8 (5)</td>
<td>2.0 (0.6, 6.2)</td>
<td>0.3</td>
</tr>
</tbody>
</table>

* mean ± standard deviation, * median (25th, 75th centile), * Acinetobacter sepsis include 6 additional strains identified in blood culture growing ≥1 organism.
Introduction: Pulmonary hypertension (PH) in infants with Down Syndrome (DS) has an incidence of up to 30% according to retrospective reports with multifactorial aetiology. However, the contribution of diastolic dysfunction to PH severity in this population is currently unexplored. We hypothesise that infants with DS exhibit early diastolic dysfunction that is related to the degree of PH during the early neonatal period.

Methods: This was a prospective study of 70 infants with DS and 60 controls who underwent comprehensive echocardiography evaluations over the first 3 days following delivery. Left ventricular (LV) diastolic function was measured using mitral valve inflow velocities and LV lateral wall tissue Doppler imaging to assess left atrial pressure (Ee'}). Speckle tracking echocardiography (STE) was used to assess LV and right ventricular (RV) systolic and diastolic function. Pulmonary vascular resistance (PVR) was assessed using pulmonary artery acceleration time (PAAT) indexed to right ventricular ejection time (RVET), and LV eccentricity index (EI).

Results: Infants with DS had a lower gestation (37.7 ± 2.1 vs. 39.6 ± 1.2 weeks, p<0.01) and birthweight (3.02 ± 0.68 vs. 3.56 ± 0.42 Kg, p<0.01). Infants with DS had higher markers of PVR throughout the study period (Figure). LV and RV systolic function was also lower in infants with DS (Figure). There was a negative correlation between PVR and RV function measured with STE. Lower LV diastolic function was associated with higher PVR (Figure).

Conclusions: Infants with DS exhibit LV diastolic dysfunction during the early neonatal period that may contribute to the evolution of PH in this population. STE-derived diastolic function measures are relatively load independent suggesting that diastolic function is contributing to increasing PVR in this population.
Figure: Values are presented as means (diamonds) and 1 standard error (whiskers). * p < 0.05 between groups. LV: Left ventricle; RV: Right ventricle; PAAT:RVET: Pulmonary artery acceleration time to right ventricular ejection time ratio.

- **PVR Measurement (lower = ↑ PVR)**
  - Day 1: *
  - Day 2: *
  - Day 3: *

- **LV Systolic Function**
  - LV global longitudinal strain (%)
  - Day 1: *
  - Day 2: *
  - Day 3: *

- **RV Systolic Function**
  - RV free wall strain (%)
  - Day 1: *
  - Day 2: *
  - Day 3: *

- **LV Early Diastolic Strain Rate (1/s)**
  - LV Eccentricity Index
  - r=0.33, p<0.01

- **RV Systolic Strain Rate (1/s)**
  - LV Eccentricity Index
  - r=0.43, p<0.01

- **PAAT:RVET**
  - LV Early Diastolic Strain Rate (1/s)
  - r=0.41, p<0.01

- **Ee’ Ratio**
  - 5 to 30
  - r=0.32, p<0.01

- **Controls**

- **Down Syndrome**
Assessment of pulmonary hypertension and myocardial function over the first week of age in infants with DS and controls
There are no conflicts of interest to declare