Factors Affecting Blood Pressures in Newborn Infants

SUMMARY Blood pressure (BP) was measured in 837 newborn infants ranging in age from 1-6 days and their mothers in Boston, Massachusetts, and Providence, Rhode Island. The BP increased significantly with age in days, birth weight, weight on day of measurement, arm circumference, and ponderal index. There was no relation of sex, race, pulse rate, type of feeding, or Apgar scores to BP in the neonatal period. Significant correlation was found between maternal and infant BP (r = 0.196 for systolic (SBP) and 0.157 for diastolic (DBP) pressures, p < 0.001). Preliminary analysis shows a trend for resemblance of 1-month and newborn BP. (Hypertension 2 (suppl I): I-99-I-101, 1980)

KEY WORDS • newborn • familial aggregation • blood pressure tracking

In the past decade, increased activity has been directed to studies of blood pressure (BP) in children. Since the etiology of essential hypertension in adults is not known and it is impossible to pinpoint accurately the onset of this disease, it is logical to examine early in life those factors that relate to BP. Several studies in adults have suggested that BPs in the third and fourth decades are predictive of BP in later life,1-2 and these observations have been extended into childhood.

In 1967 we began a prospective study of a cohort of 721 normal children in 192 families in the Boston area.8 Blood pressures were measured in the homes using an automated BP recorder. These studies demonstrated for the first time that BP levels were aggregated in families of children. This cohort has been followed for 8 years, and the familial aggregation persists. The BP levels at the second and third surveys were significantly positively related to BP measured at the initial survey when the children were 2-14 years old.4 Thus, it is possible that those children with relatively higher BP at age 2, 6, or 12 years will be at greater risk of developing essential hypertension later in life.

Since the factors responsible for familial aggregation of BP are operating by age 2, it is reasonable to assume that there are powerful influences on BP beginning earlier in life. With the availability of devices for the measurement of newborn BP,5 it has become possible to study large populations of newborns and to seek relative variables operational in the earliest days of life.

This report describes the design and preliminary results of a long-term follow-up study of BP in normal newborn infants.

Materials and Methods

The two cohorts of newborn infants drawn for this study include 412 infants born at the Boston City Hospital and 425 born at the Women & Infants Hospital of Rhode Island in Providence. Following an uncomplicated vaginal or cesarean delivery, mothers were invited to participate in this program. Approximately 4% of the total deliveries during the time of study were included in the survey. The participants represent 62% of the total number of mothers invited. Written informed consent was obtained, and BP was measured on the infant and one or both parents in the hospital during the first days of life. The Roche Arteriosonde ultrasonic device (Roche Medical Electronics, Cranbury, New Jersey) was used to measure BP, and in the Boston cohort this measurement was recorded on a paper tracing by a portable automated recorder (Roche Medical Electronics, Cranbury, New Jersey).4 Appropriate cuff sizes were used for adults as recommended by the American Heart Association,7 and the cuff covered two-thirds of the arm above the
elbow in the neonates. Usually, the infant was examined on the same day as the mother but not in her presence. The following determinations were also recorded: sex, race, arm circumference, arm length (measured with a tape measure from the acromion to the distal end of the humerus), birth weight, weight on day of BP measurement, condition at the time of measurement (quiet sleep, active sleep, awake and quiet, crying; as described by Lee et al.), pulse rate, and time of day. Also, the educational level and socioeconomic status of the parents were determined by a questionnaire and the modified Hollingshead index. The BP levels were measured at home visits in participating families at 1 week, 1 and 6 months, and 1 year after discharge from the hospital.

Results

The results are presented as preliminary analyses of the merged Boston (412 infants) and Providence (425 infants) cohorts. As there was no significant difference in BP after adjusting for age (in days) and other significant variables in the two populations, the samples have been merged for some analyses. Follow-up data have thus far been analyzed only in the Providence cohort of 425 infants.

Age in Days

Blood pressures increase significantly during the first 5 days of life, and the increase may be as large as 10 mm Hg (table 1). For example, in the Boston cohort, the mean systolic blood pressure (SBP) at 1-2 days in 247 infants was 70.7 ± 9.2 mm Hg; at 3 days, in 132 babies, 75.3 ± 9.9 mm Hg, and at 4 days, 76.1 ± 9.0 mm Hg in 33 infants.

Race and Sex

Blood pressures did not differ significantly by race (for SBP, 74.1 ± 9.2 mm Hg in 380 white babies and 75.1 ± 11.2 mm Hg in 26 black babies; for diastolic blood pressure (DBP) 51.3 ± 9.0 and 51.3 ± 10.6 mm Hg respectively in the Providence cohort). Similar data were found in the Boston and combined cohorts. Also, no difference was found according to sex of the infant.

Type of Feeding

Neonatal BP in the hospital did not differ in breast- or bottle-fed babies. Mean SBP and DBP in 154 breast-fed babies were 74.2 ± 9.6 mm Hg and 50.9 ± 9.0 mm Hg compared with 74.2 ± 9.0 and 51.6 ± 9.1 mm Hg in 264 bottle-fed infants.

Other Variables

Correlation coefficients were calculated by the method of Rosner et al. using raw BP in the Providence cohort (table 2). Both SBP and DBP increased with increasing arm circumference, weight at examination, birth weight, body length, and ponderal index (weight/length2). There was no significant relationship of BP to pulse rate or arm length or to 1- and 5-minute Apgar scores at birth. The BP increased with increasing activity in the newborn at the time of the measurement. In the Providence cohort, sleeping babies had lower BP than crying babies (73.9 ± 9.2 vs 76.6 ± 11.2 mm Hg for SBP), but quiet, awake babies had pressures resembling those of sleeping infants (73.2 ± 8.3 vs 73.9 ± 9.2 mm Hg). Similar data have been published for the Boston cohort.

Maternal-Infant Resemblance

In the combined cohort, there was a significant but small relation between maternal and neonatal SBP and DBP. The pairwise interclass correlation coefficients in 576 maternal-infant pairs were 0.196 for SBP and 0.157 for DBP (p < 0.001). Maternal-infant analyses are in progress.

Follow-Up Studies

In the combined cohort, home visits have been made to 451 families at 1 week, 580 at 1 month, and 435 at 6 months after birth. Preliminary analyses are available only on the Providence cohort for SBP scores adjusted for age in days and the other variables listed in table 2 in 111 infants studied in the hospital and at 1 month at home. The adjusted score represents the residual score after removing effects of the listed variables. The regression coefficient for 1 month SBP adjusted scores on initial postpartum score was 0.241, p = 0.07. This coefficient increased to 0.272, p = 0.04 when change in pulse rate from birth to 1 month was added in a multiple regression analysis.

Discussion

The burgeoning literature in the past 10-15 years gives testimony to the increased interest in BP in children. Studies have been directed toward uncovering clinical cases of essential hypertension in teenage and older children, and toward predicting essential hypertension in adults by studies of BP in children. It is not possible at present to identify an individual child who, if untreated, is destined to develop sustained hypertension later in life; but it is possible to identify some of the variables that affect BP in children.

<table>
<thead>
<tr>
<th>Age (day)</th>
<th>n</th>
<th>SBP ± SD (mm Hg)</th>
<th>DBP4 ± SD (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>72.4 ± 6.1</td>
<td>50.5 ± 9.7</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>68.6 ± 6.5</td>
<td>47.6 ± 7.3</td>
</tr>
<tr>
<td>3</td>
<td>214</td>
<td>72.6 ± 8.7</td>
<td>49.7 ± 8.3</td>
</tr>
<tr>
<td>4</td>
<td>98</td>
<td>78.3 ± 8.1</td>
<td>54.3 ± 8.6</td>
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<tr>
<td>5</td>
<td>39</td>
<td>83.4 ± 10.8</td>
<td>59.2 ± 10.5</td>
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<td>6</td>
<td>98</td>
<td>79.4 ± 8.0</td>
<td>58.2 ± 10.3</td>
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SBP = systolic blood pressure; DBP4 = diastolic blood pressure, 4th Korotkoff phase; SD = standard deviation.
Our present study examines several variables affecting BP in newborn infants. Although multivariate analyses have not yet been performed, the significant association of birth weight, arm circumference, ponderal index, age in days, are similar to other published studies. The familial association of maternal and neonatal BP present within the first few days of life is seen for both SBP and DBP. Lee et al. have previously reported a significant maternal-infant correlation for DBP in a sample of this population. Hennekens et al. have reported a significant aggregation of infant-sibling BP by 1 month of age, but de Swiet and Shinebourne did not find parent-infant association of birth weight, arm circumference, analyses have not yet been performed, the significant association of BP measurements. Nonetheless, it appears that some degree of familial influence on BP is present very early in life.

Several studies in older children have reported that BP tracking begins in childhood. This finding implies that, once elevated, a given BP remains relatively elevated with the passage of time. The magnitude of the “tracking coefficient” appears to increase with increasing age and reaches 0.6-0.7 by adulthood.

Detailed analyses of follow-up BP in infants are limited in number. Schachter et al. reported recently that SBP and DBP in 115 infants showed significant correlations of readings at 6 months with those at 15 months but not with pressures obtained earlier. Studies by de Swiet and colleagues showed a significant relation of BP at 4-6 days and at 5-7 weeks in 272 newborns in the United Kingdom. Our preliminary tracking data at 1 month are consistent with this finding, and suggest that BP status may be set very early in life.

In this and in other series, neonatal BP did not differ by type of feeding. Even if bottle or breast feeding were determined of elevated BP, it would likely take several months or years for any effect to be noticed or deciphered. Similarly, considerations of other dietary variables, such as sodium intake, require detailed chemical studies and intensive follow-up before being assigned any positive or negative role.

The findings of significant tracking correlations and familial aggregation of BP in infancy do not provide solutions for the etiologic enigmas of hypertension. The studies reported do not isolate all the possible risk factors that may act early in life to elevate BP. Obviously, there are genetic and environmental factors that must interact over a lifetime in the course of development of essential hypertension. Nevertheless, a significant familial influence can be discerned early in life, and the familial aggregation effect is almost of the same magnitude as that seen in adults.

References

Factors affecting blood pressures in newborn infants.
S H Zinner, Y H Lee, B Rosner, W Oh and E H Kass

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