The term "risk ratio" is used incorrectly in this paper. For "risk ratio" please substitute "odds" throughout.

Jaundice in the Healthy Newborn Infant: A New Approach to an Old Problem

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From the Division of Newborn Medicine, Department of Pediatrics, The Pennsylvania State University College of Medicine, Hershey, and the Department of Statistics, The Pennsylvania State University, University Park

ABSTRACT. We measured the serum bilirubin concentrations in 2,416 consecutive infants admitted to our well baby nursery. The maximal serum bilirubin concentration exceeded 12.8 mg/dL (221 µmol/L) in 147 infants (6.1%), and these infants were compared with 147 randomly selected control infants with maximal serum bilirubin levels ≤12.9 mg/dL. A serum bilirubin concentration >12.9 mg/dL was associated strongly with breast-feeding (P = .0000) and percentage of weight loss after birth (P = .0001), as well as with maternal diabetes, oriental race, decreased gestational age, male sex, bruising, and induction of labor with oxytocin. Risk ratios and the risk of jaundice were calculated for hypothetical infants in the presence and absence of these variables. These calculations show that, in certain infants, "nonphysiologic" jaundice is likely to develop and its presence in such infants might not require laboratory investigations. In others, a modest degree of hyperbilirubinemia could be cause for concern. An awareness of these factors and their potential contribution to serum bilirubin levels permits a more rational approach to the action levels used for the investigation of jaundice in the newborn. We need a new definition of physiologic jaundice. Pediatrics 1988;81:500–511; jaundice, hyperbilirubinemia, bilirubin, newborn infant, breast-feeding.

To the pediatrician and family practitioner, jaundice remains the most common and, perhaps, the most vexing problem in the well baby nursery. Although the vast majority of jaundiced full-term infants appear completely healthy, standard textbooks of pediatrics and newborn medicine mandate diagnostic investigations to rule out "pathologic" jaundice in full-term infants whose serum bilirubin concentrations exceed a level of 12 to 12.9 mg/dL (205 to 221 µmol/L) = [mg/dL] × 17.10 = [µmol/L]. Some suggest that bilirubin levels in excess of 10 mg/dL (171 µmol/L) deserve further investigation. Whatever level is chosen, there is little doubt that the presence of pathologic (perhaps better termed "nonphysiologic") jaundice engenders substantial anxiety in parents and physicians alike. In a previous study, however, we could find no cause for the jaundice in 58% of infants whose serum bilirubin concentrations exceeded 12 mg/dL. If such jaundice is truly pathologic, it seems strange that a cause is found in fewer than one half of these infants. Furthermore, as no investigations are considered necessary in infants with "physiologic" jaundice, it is important to define more precisely what is, and what is not, physiologic.

The largest prospective study of neonatal jaundice was performed by the National Collaborative Perinatal Project in which serum bilirubin concentrations were measured prospectively from samples obtained from more than 35,000 infants. Of infants with birth weights >2,500 g, 6.1% of white infants and 4.51% of black infants had serum bilirubin levels >12.8 mg/dL. In an earlier study of 106 infants (90% white infants), 13.2 mg/dL (226 µmol/L) represented the 90th percentile. In more recent studies, the incidence of serum bilirubin concentrations >12 mg/dL has ranged from 8% to 20% during the first week of life. However, numerous factors are known to affect serum bilirubin levels and these may vary among different populations. For example, although the strong association between breast-feeding and jaundice in the early newborn period has been questioned in the past, recent studies leave absolutely no doubt of that association.
Thus, the importance of documenting the prevalence of breast-feeding, in a population for whom one is establishing norms for serum bilirubin levels, is obvious. Because investigations for jaundice increase the cost of care as well as parental anxiety, it seems important to establish reasonable action levels for the investigation of otherwise healthy jaundiced newborn infants. In this study, we investigated the association between serum bilirubin concentrations and variables previously reported to affect bilirubin levels in the newborn infant.

**PATIENTS AND METHODS**

We measured the serum bilirubin concentrations in 2,416 consecutive infants (95% white, 95% >2,500 g) admitted to our well-baby nursery between Jan 1, 1976, and Dec 31, 1980. The total serum bilirubin level was measured in every case on the second or third hospital day (according to a standard clinical protocol), and measurement was repeated if clinically indicated. If the bilirubin concentration exceeded 12.9 mg/dL, the following additional investigations were performed: blood typing, direct and indirect Coombs tests, reticulocyte count, hemostocrit, WBC and differential cell count, smear for RBC morphology, and total and direct serum bilirubin concentrations. The maximal serum bilirubin level exceeded 12.9 mg/dL in 147 (6.1%) infants. These infants constituted our test group. From the remaining 2,269 infants with serum bilirubin levels ≤12.9 mg/dL, we randomly selected a control group of 147 infants (using a table of random numbers) and compared them with the test group for the variables listed in the Figure. ABO and Rh incompatibility were not included in this comparison because blood typing and Coombs tests were not performed in the low-bilirubin group. Information concerning feeding, birth weight, and weight loss was obtained from a nursery log maintained for every baby. The rest of the clinical information was obtained by retrospective review of mothers' and infants' charts.

Serum bilirubin measurements were performed by a modified diazo method using an automatic clinical analyzer (ACA III Instruction Manual; EI Dupont de Nemours Co, Clinical Systems Division, Wilmington, DE). At serum bilirubin levels of 2.5, 4.2, and 19.4 mg/dL (Omega Chemistry Control Sera; Hyland Diagnostics Corp, Bannockburn, IL), 30 repeated determinations showed standard deviations of 0.1, 0.18, and 0.3 mg/dL with coefficients of variation of 4.6%, 4.2%, and 1.5%, respectively.

The data were analyzed using the Fisher exact test for nominal data and a pooled t test for continuous data. In addition, analysis of the two groups was performed using a stepwise logistic regression procedure. The logistic regression model allows us to estimate the probability that an infant with given characteristics (eg, breast-fed, oriental) will have a serum bilirubin level greater than 12.9 mg/dL (the risk ratio). When there are several characteristics that are known or thought to be related to the occurrence of this event, the stepwise logistic regression approach is especially useful in selecting a subset of these characteristics that can be used in a logistic regression model. The characteristics selected by this procedure, the order in which they are selected, their associated _P_ values, and the performance of the fitted logistic regression model in correctly classifying the study subjects as patients (indicated by high estimated probabilities of the event) or controls (indicated by low estimated probability of the event) provide useful information to the investigator in making a final selection of a logistic regression model.

The logistic regression analysis used ignores patients with any missing data. Thus, for this analysis, choioamnionitis was not included because too few placentas were examined. Also eliminated were black infants (n = 6) and infants who were both breast- and bottle-fed (n = 11). Because blood typing was not performed in control infants we could not consider Rh or ABO incompatibility in the model. We entered only those variables that were significant by the univariate analysis or identified by the multivariate model to further refine the data using logistic regression analysis. The coefficients in the final model were estimated.
using 135 observations in the low-bilirubin group and 135 observations in the high-bilirubin group.

RESULTS

The results are shown in Tables 1 through 4. A total of 147 infants (61.1%) had a serum bilirubin concentration >12.9 mg/dL. Compared with the control group, a serum bilirubin concentration >12.9 mg/dL was significantly associated with breast-feeding, percentage of weight loss after birth (a particularly important association [Table 3]), maternal diabetes, oriental race, induction of labor with oxytocin, decreasing gestational age, and male sex. Of infants in whom no cause for hyperbilirubinemia was found, 82.7% were breast-fed vs 46.9% in the control group (P < .00001). Breast-feeding was significantly associated with hyperbilirubinemia, even in the first three days of life. No significant association was found with the method of delivery, anesthesia, maternal hypertension, maternal alcohol use, augmentation of labor with oxytocin, or Apgar score. The distribution of maximal serum bilirubins in the 2,297 infants weighing >2,500 g at birth has been presented in detail. In 66 of the 147 infants with hyperbilirubinemia (45%), an apparent cause of the jaundice was found (five had Rh and ABO incompatibility; other causes included polycythemia, maternal diabetes, bruising/cephalohematoma, polythemia, asphyxia, cholestasis, and gestation ≤35 weeks).

DISCUSSION

Our data confirm the association between neonatal jaundice and several factors previously identified. We recently documented a striking association between breast-feeding and hyperbilirubinemia and, in contrast to previous observations, we also found a highly significant association between weight loss and jaundice (Tables 2 and 3), suggesting an important role for caloric (or fluid) intake in the regulation of serum bilirubin. A relationship between caloric intake and serum bilirubin concentrations has been demonstrated previously in adults and animals, and

<table>
<thead>
<tr>
<th>TABLE 1.</th>
<th>Univariate Analysis: Discrete Variables*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Serum Bilirubin (&lt;12.9 mg/dL)</td>
<td>High Serum Bilirubin (&gt;12.9 mg/dL)</td>
</tr>
<tr>
<td>Total Observations</td>
<td>No. (%) of Subjects</td>
</tr>
<tr>
<td>Breast-feeding</td>
<td>147</td>
</tr>
<tr>
<td>Maternal diabetes</td>
<td>147</td>
</tr>
<tr>
<td>Oxytocin induction</td>
<td>147</td>
</tr>
<tr>
<td>Male sex</td>
<td>147</td>
</tr>
<tr>
<td>Oriental race</td>
<td>147</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>125</td>
</tr>
<tr>
<td>Epidural anesthesia</td>
<td>147</td>
</tr>
<tr>
<td>Apgar score &lt;5 at 1 min</td>
<td>147</td>
</tr>
<tr>
<td>&lt;7 at 5 min</td>
<td>147</td>
</tr>
<tr>
<td>Bruising/cephalohematoma</td>
<td>147</td>
</tr>
<tr>
<td>Maternal hypertension</td>
<td>146</td>
</tr>
<tr>
<td>Black race</td>
<td>147</td>
</tr>
<tr>
<td>Method of delivery</td>
<td>147</td>
</tr>
<tr>
<td>Vaginal</td>
<td>147</td>
</tr>
<tr>
<td>Instrumental</td>
<td>147</td>
</tr>
<tr>
<td>Cesarean section</td>
<td>147</td>
</tr>
<tr>
<td>Oxytocin augmentation</td>
<td>121</td>
</tr>
</tbody>
</table>

* Numbers <147 indicate missing or inadequate data.

<table>
<thead>
<tr>
<th>TABLE 2.</th>
<th>Univariate Analysis: Continuous Variables*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Bilirubin (&lt;12.9 mg/dL)</td>
<td>Serum Bilirubin (&gt;12.9 mg/dL)</td>
</tr>
<tr>
<td>Wt loss (%)</td>
<td>4.69 ± 2.69</td>
</tr>
<tr>
<td>Gestational age (wk)</td>
<td>39.63 ± 1.96</td>
</tr>
<tr>
<td>Birth wt (g)</td>
<td>3,358 ± 556</td>
</tr>
<tr>
<td>Gravidity</td>
<td>2.24 ± 1.49</td>
</tr>
</tbody>
</table>

* N = 147. All values are means ± standard deviation.
TABLE 3. Stepwise Logistic Regression Procedure*  

<table>
<thead>
<tr>
<th>Variable</th>
<th>β</th>
<th>SE</th>
<th>χ²</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>2.355</td>
<td>3.3232</td>
<td>2.52</td>
<td>.1124</td>
</tr>
<tr>
<td>% wt loss</td>
<td>19.844</td>
<td>5.8399</td>
<td>11.16</td>
<td>.0008</td>
</tr>
<tr>
<td>Breast-fed</td>
<td>1.3064</td>
<td>0.3889</td>
<td>14.86</td>
<td>.0001</td>
</tr>
<tr>
<td>Oriental race</td>
<td>2.6382</td>
<td>1.1212</td>
<td>6.41</td>
<td>.0114</td>
</tr>
<tr>
<td>Gestational age</td>
<td>0.2001</td>
<td>0.0846</td>
<td>5.57</td>
<td>.0182</td>
</tr>
<tr>
<td>Oxytocin induction</td>
<td>1.4056</td>
<td>0.7304</td>
<td>3.70</td>
<td>.0543</td>
</tr>
<tr>
<td>Brusing</td>
<td>0.6217</td>
<td>0.3308</td>
<td>3.33</td>
<td>.0697</td>
</tr>
<tr>
<td>Maternal diabetes</td>
<td>1.5977</td>
<td>0.8385</td>
<td>3.72</td>
<td>.0538</td>
</tr>
<tr>
<td>Male sex</td>
<td>0.4467</td>
<td>0.2891</td>
<td>2.39</td>
<td>.1224</td>
</tr>
</tbody>
</table>

* Note that the P values obtained in this procedure are different from the P values obtained in the univariate analyses and are to be regarded as the P value associated with a particular variable when the present set of variables is used in estimating the probability of the event.

† Each of the estimated β values has its associated estimated asymptotic standard deviation. The ratio of the estimated β to its standard deviation is a standard normal random variable if in fact the real β is zero (the null condition for which a P value is desired). The square of a standard normal random variable is a χ² random variable with 1 df.

TABLE 4. Risk Ratios for Hypothetical Infants With (+) and Without (−) Variables Shown*  

<table>
<thead>
<tr>
<th>Factors</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestation (wk)</td>
<td>39</td>
<td>40</td>
<td>38</td>
<td>42</td>
<td>38</td>
</tr>
<tr>
<td>Percent weight</td>
<td>5</td>
<td>7</td>
<td>15</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Oriental</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Oxytocin induction</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Maternal diabetes</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Breast-fed</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Male sex</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Risk ratio</td>
<td>0.014</td>
<td>0.0626</td>
<td>0.720</td>
<td>2.032</td>
<td>5.335</td>
</tr>
<tr>
<td>Jaundice risk (%)</td>
<td>1.4</td>
<td>5.9</td>
<td>42</td>
<td>67</td>
<td>84</td>
</tr>
</tbody>
</table>

* Risk ratio = (probability of serum bilirubin >12.9 mg/dL)/(probability of serum bilirubin ≤12.9 mg/dL). Jaundice risk = Probability of having serum bilirubin >12.9 mg/dL = (risk ratio/(1 + risk ratio)). Example: Baby C is a breast-fed boy and has a 15% weight loss. The odds of baby C having a serum bilirubin >12.9 mg/dL are 0.720:1 and the risk of jaundice is 42%. Note: In our population the prevalence of hyperbilirubinemia (>12.9 mg/dL) is 6.1%. If no information is available on an infant, the risk ratio is 0.065 and the risk of jaundice is 6.1%. (Confidence limits 5% to 7%.)

results of studies in newborn infants suggest a similar association. A negative association between maternal smoking and neonatal hyperbilirubinemia has also been observed by others. Although our data suggested such an association, the results did not reach statistical significance (P = 0.094). Smoking did not enter the logistic model, perhaps because our retrospective review of the mothers' charts did not permit us to quantify accurately the number of cigarettes smoked by each mother. Thus, smoking could be introduced only as a categorical and not a continuous variable. Linn and coworkers found that women who smoked at least one pack of cigarettes per day had a lower risk of having a child with hyperbilirubinemia than those who had smoked less. In a preliminary analysis of the data from the Collaborative Perinatal Project, R. L. Naeye (personal communication, 1987) has found a similar strong, negative association between smoking and neonatal jaundice. Our finding that hyperbilirubinemia was associated with the induction but not augmentation of labor with oxytocin is similar to the observations of others. Many of the variables are closely associated and thus confounding. For example, 15% of breast-feeding mothers smoked v 41% of bottle-feeding mothers (P < .00001). We found an association between maternal diabetes and hyperbilirubinemia, but 87.5% of diabetic mothers breast-fed their infants v 84.4% of nondiabetic mothers (P = 0.45). Breast-fed infants lost an average of 8.86 ± 2.97% of their birth weight, whereas bottle-fed infants lost 4.24 ± 2.88% (P < 0.02). The relationship between diabetes and oxytocin is also confounding. In 31% of diabetic women, labor
was induced with oxytocin v 5.6% of non-diabetic women (P = .003).

It is evident, therefore, that in addition to the usual univariate analyses we should consider the joint effects of the variables in the study on the occurrence of an elevated serum bilirubin level. The logistic regression model is commonly used to do this (see "Appendix"). The logistic regression equation permits calculation of the risk ratios and the risk of jaundice in individual infants in the presence or absence of certain variables (Table 4 and "Appendix"). Breast-feeding and maternal diabetes increase the risk ratio for jaundice 3.7 and 4.9 times, respectively, and an increase in postnatal weight loss of 5% increases the risk ratio 2.7-fold. A breast-fed male infant of a diabetic mother, born following labor induced by oxytocin, has an 84% risk of hyperbilirubinemia (Table 4).

It should be noted that the prevalence of hyperbilirubinemia in the logistic model was 50%, whereas in the total population the prevalence was 6%. The expansion of risk estimates to a larger population is discussed in the "Appendix."

What are normal serum bilirubin levels? Inherent in this question is the definition of "normal," a matter of more than philosophical interest. As we suggested, the definitions of normal bilirubin values found in current texts have, in essence, created a class of healthy jaundiced babies with "nondisease." To address this, we might designate as abnormal those levels that are commonly associated with disease, but, by itself, hyperbilirubinemia is a relatively common event in certain groups of otherwise healthy infants. If we knew at what level the serum bilirubin represented a threat to the well-being of the infant, we might consider that level as abnormal or we could define a "therapeutic" normal level. This is the level beyond which therapy has been shown to do more good than harm. Unfortunately, we have little data on which to base such a definition in a population of healthy, term infants without erythroblastosis fetalis. In many jaundiced infants, known risk factors (Tables 2 to 4) are present, and significant pathology such as hemolytic disease can be ruled out frequently by a review of the mother's history and blood type and careful physical examination of the newborn. In such infants, a policy of watchful expectancy (with repeat serum bilirubin determination when indicated) seems justified rather than a battery of laboratory tests. On the other hand, an apparently normal (physiologic) serum bilirubin level (eg, 12.0 mg/dL) in a low-risk infant such as baby A (Table 4) might arouse suspicion and require further investigation.

Whatever decisions are made regarding investigation, they are usually unrelated to treatment. Whether and when such infants should be treated to prevent possible bilirubin encephalopathy is an issue that cannot be addressed by this study. It must be emphasized, however, that additional serum bilirubin levels should be obtained, when necessary, to determine the progress of the hyperbilirubinemia and to suggest intervention when appropriate.

Our data confirm a strong association between breast-feeding and jaundice in the healthy newborn infant and suggest that caloric deprivation also plays an important role. Furthermore, several nonpathologic variables in pregnancy, labor, and the newborn period are significantly associated with neonatal hyperbilirubinemia. An awareness of these factors and their potential contribution to serum bilirubin levels permits a more rational approach to the action levels used for the investigation of jaundice in the newborn. We suggest that a new definition of what is and is not physiologic jaundice is needed.

Appendix

The logistic regression equation is of the form:

\[ P(x_1, \ldots, x_k) = \frac{1}{1 + e^{- (\beta_0 + \beta_1 x_1 + \cdots + \beta_k x_k)}} \]

where \( x_1, \ldots, x_k \) are the variables (discrete or continuous) being used to provide \( P(x_1, \ldots, x_k) \), the probability of the event in question. Generally, a data set of cases and controls is used to estimate the parameters (\( \beta \) values) in the model. Note that because the

\[ \text{Risk ratio} = \frac{P(\text{event})}{1 - P(\text{event})} \]

device which associated with the logistic regression model is simply exp(\( \beta_0 + \beta_1 x_1 + \cdots + \beta_k x_k \)).

Unfortunately, it is imperative, in the logistic regression procedure (Table 3) permits calculation of the risk ratio in the presence or absence of the listed variables.

Risk ratio

\[ \frac{\text{probability of serum bilirubin > 12.9 mg/dL}}{\text{probability of serum bilirubin ≤ 12.9 mg/dL}} \]

In: (risk ratio for sampled babies) = intercept + \( \beta_1 \) (fract. weight loss) + \( \beta_2 \) (breast-fed) + \( \beta_3 \) (gestational age) + \( \beta_4 \) (oxytocin induction) + \( \beta_5 \) (maternal diabetes) + \( \beta_6 \) (male)

To go from the risk ratio for sampled babies (RRSB) to the risk ratio for all babies (RRAB) the formula is in general is

\[ \text{RRAB} = \left( \frac{N_0}{N_D} \right) \text{RRSB} \]

where \( N_D \) is the total number of cases and \( N_0 \) is the
total number of noncases among all babies. For our
problem the factor
\[
\frac{N_D}{N_C} = \frac{147}{2289} = 0.06478
\]
Example:
Baby C (Table 4)—breast-fed, white boy, 38 weeks,
15% weight loss
in (risk ratio for sampled babies)
\[
5.28 + 19.84 (0.15) + 1.306 - 0.2 (38) + 0.446
= 2.408
\]
risk ratio (sampled babies)
\[
\phi = 2.408
\]
risk ratio (all babies)
\[
= 11.11 \times 0.0648 = 0.720
\]
may now calculate risk ratio for the two groups
of babies looking at characteristics of each group

\[P(\text{event}) = \frac{\text{(risk ratio)}}{1 + \text{(risk ratio)}}\]

The factor 0.0648 makes the proper adjustment for the
270 sampled (135 low-bilirubin and 135 high-bilirubin)
babies used in the case-control logistic analysis to pro-
vide a risk ratio for the population from which these
were selected.

The risk ratio is of interest in assessing the important-
ance of the variables. The probability of the event is
related to the risk ratio by the equation

Several points should be noted: (1) If \( n_D/n_C = N_D/N_C \),
then no adjustment is made. This would usually
mean that we spent too much of our effort studying too
many controls. (2) If \( n_D = n_C \), as is often the case, the
adjustment factor is the population risk ratio for the
disease. (3) If the \( n_C \) are selected by other than a pure
random process (eg, by matching with the \( n_D \)),
then the given adjustment is not correct. (4) Because the
adjustment factor is fixed, it follows that confidence in-
tervals for the risk ratio from the study group can be
converted directly into confidence limits for the risk
ratio for larger population. (5) If all of the data are
available for the larger population of interest, then they
should be used to develop estimates of the risk ratio for
a baby with given characteristics. It would be of interest
to see the degree to which this method would be su-
prior to simply expanding the estimates from the
study group. A simulated experiment for a variety of
sample sizes should provide the answers.

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