Developmental Outcome at 6.5 Years After Acidosis in Term Newborns: A Population-Based Study

WHAT'S KNOWN ON THIS SUBJECT: Conflicting results exist concerning long-term outcome in healthy infants with metabolic acidosis at birth.

WHAT THIS STUDY ADDS: Neonates who appear well after perinatal metabolic acidosis do not have an increased risk of neurologic or behavioral problems in need of referral actions or pedagogic arrangements at the age of 6.5 years.

abstract

OBJECTIVES: Infants who develop encephalopathy after perinatal asphyxia have an increased risk of death and adverse neurologic outcome. Conflicting results exist concerning outcome in healthy infants with metabolic acidosis at birth. The aim of the current study was to evaluate whether metabolic acidosis at birth in term infants who appear healthy is associated with long-term developmental abnormalities.

METHODS: From a population-based cohort (14,687 deliveries), 78 infants were prospectively identified as having metabolic acidosis (umbilical artery pH < 7.05 and base deficit in the extracellular fluid >12.0 mmol/L). Two matched controls per case were selected. The child health and school health care records were scrutinized for developmental abnormalities.

RESULTS: Outcome measures at 6.5 years of age for 227 of 234 children (97%) were obtained. No differences were found concerning neurologic or behavioral problems in need of referral action or neurodevelopmental diagnosis in comparison of control children with acidic children who had appeared healthy at birth, ie, had not required special neonatal care or had no signs of encephalopathy.

CONCLUSIONS: Infants born with cord metabolic acidosis and who appear well do not have an increased risk for neurologic or behavioral problems in need of referral actions or special teaching approaches at the age of 6.5 years. Pediatrics 2012;129:e1501–e1507
Perinatal asphyxia is known to be a major cause of neonatal and childhood morbidity and mortality. Infants who develop encephalopathy have an increased risk of death and adverse neurologic outcome. Perinatal asphyxia causes fetal acidosis manifested as low umbilical cord pH at birth. Analysis of umbilical cord pH has become part of routine care and serves as a quality measure of obstetric care and provides some risk assessment for the infant. In a Swedish study, 1 6.5% of acidotic infants had cerebral complications in the neonatal period, comparable with the rate of infants with cord artery pH < 7.05. This study also included the infants with encephalopathy.

The extensive review by Malin et al on the association between umbilical cord pH and perinatal long-term outcome corroborates these findings but also highlights the lack of high-quality, long-term follow-up studies for other measures of adverse outcome than cerebral palsy. The association between low cord pH in a low-risk population and long-term outcome is still unclear. In this meta-analysis, an initial search of 5690 citations gave 51 primary articles, but only 17 addressed long-term outcome measures, where 7 of these studies examined the association between arterial cord pH and long-term outcome beyond cerebral palsy.

Odd et al described that infants who were resuscitated but who remained well afterward had an increased risk for low IQ score compared with those not requiring resuscitation, but they found no difference in the aspect of neuropsychological functioning. These studies did not report the infants' IQ level. In the study of Ingemarsson et al, infants with acidemia at birth had more problems with speech development at 4 years of age than controls. This study also included the infants with encephalopathy. Dennis et al compared infants with acidemia at birth with nonacidotic infants having normal Apgar scores and low Apgar scores for performance in neurodevelopmental tests at age 4.5 years. They found no association between acidosis and neurodevelopmental function. The retrospective cohort study by Svirko et al correlated arterial pH at birth with childhood tests for nonverbal intelligence, grammar comprehension, and literacy. Although a negative correlation was found, only at extreme levels of acidosis was an increased incidence of overt brain injury established. Wilschut et al found that, in a sample of infants with a large variation in umbilical artery pH and without severe neonatal neurologic abnormalities, acid-base status at birth and quality of general movements at 3 months of age are not predictive of motor milestone achievement and cognitive and behavioral functioning at 4 years.

It has been suggested that neonatal complications are associated with metabolic rather than respiratory acidosis. Respiratory acidosis arises in the early stages of impaired blood supply to the fetus, and hypoxemia and hypercapnia occur that lead to a reduction in pH with a normal base deficit. If hypoxia is prolonged, anaerobic metabolism results, and the base deficit rises secondary to the presence of lactic acidosis and consumption of buffering substances.

The Child Health Service (CHS) program and the school health program assess the health and development of all children living in Sweden at specified ages. The first examination in school health care is made at the age of about 6.5 years. This evaluation is done by the school physician and the school nurse. The children are examined, and parents are interviewed. An educational assessment made by the child’s teacher and a specified parent report are included. The school health records routinely contain a summary of the child's preschool health service record (CHS) record.

A written summary is made on the basis of these records. Data from these assessments have previously been used in follow-up studies. The aim of the current study was to take advantage of all such data to evaluate whether metabolic acidosis at birth in term infants who appear healthy is associated with developmental abnormalities up to the age of 6.5 years.

**METHODS**

The definition of metabolic acidosis in this study is umbilical artery pH < 7.05 and base deficit in the extracellular fluid (BDecf) > 12.0 mmol/L. This definition was chosen in accordance with other studies that used the combination of pH and BDecf. A cutoff value of 7.00 is often used when cord artery pH has been the single definition of acidosis. In this situation, however, the low pH is most often a result of the combination of respiratory and metabolic acidosis. BDecf was calculated according to Wiberg et al.

As part of the introduction of the new STAN methodology for intrapartum fetal surveillance, combining an automatic continuous evaluation of the ST segment of the fetal electrocardiogram (STAN) with conventional fetal heart rate analysis (cardiotocography [CTG]), a prospective observational study covering the total population of deliveries at term in the city of Gothenburg and surrounding areas was conducted over 2 years (October 2000 to September 2002). Of the 14 687 term deliveries, 78 cases had cord artery metabolic acidosis (group A). Control cases (group B) were identified as the 2 adjacent deliveries monitored with the same technique CTG or CTG and ST analysis in combination, delivered at the same gestational age, of the same gender, having a cord artery pH of > 7.10 and in whom there was no need of care in the neonatal ward. A flowchart is shown in Fig 1.
The infants with metabolic acidosis were assigned to 4 groups according to their need of treatment in the neonatal period (groups A1 and A2) and planned level of follow-up (groups A3 and A4). Figure 1 shows the categorization. Healthy is defined as not admitted to the special care baby unit (SCBU) (group A2) or being assigned only for ordinary health care follow-up (group A4). Acidotic infants who initially had been admitted to the SCBU but not assigned to any extra follow-up were also included in group A4. The physician in charge decided the level of neonatal care (SCBU or not) based on clinical judgment and prevailing routines and likewise chose the level of follow-up (special development assessment or not).

The children were identified by using the Swedish national registration system and the school health database system. The records were scrutinized for developmental data, referral actions, or proceedings for specific educational support by one and the same experienced school health nurse (S.E.) with the use of a specific protocol. Data extraction from these protocols, evaluations, and categorization (Table 1) were performed independently by 2 experienced pediatricians (M.H., I.K.) blinded to the conditions at birth.

The Regional Ethical Review Board of the Faculty of Medicine, University of Gothenburg, approved the current study.

In the prospective power analysis based on data from 70 index and 140 control children, a significance level of 5% and a power of 0.88 were achieved when 20% of the index and 5% of the control population demonstrated developmental abnormalities. If the numbers were instead 15% and 5%, respectively, the power was estimated to be 0.63.

Because 11 of 78 allocation groups were not complete, a matched-pair analysis was omitted, and the difference between the groups was calculated by using the Mann–Whitney nonparametric test. The results are given as median values and range.

The $\chi^2$ distribution test was used to analyze the frequency of the different categories in the different acidotic groups (A, A2, and A4) and the control group (B). The STATISTICA 6.0 statistical package was used.

**RESULTS**

All of the 234 children were identified by the Swedish national registration system. Four had died, and 4 had emigrated from Sweden. Of the remaining 226 health records that were possible to find, 223 (98.7%) were found and surveyed. Two records from the control group and 1 from the index group could not be located (Fig 1). These 11 missing records came from different allocation groups. Both controls were found for the remaining 67 index cases.

Data from the examinations done at 6.5 years of age were obtained in all but 6 of the studied files. Four of these had a neurodevelopment diagnosis and were followed at a habilitation center according to the CHS (category 4, Table 1). 1 had been referred to a neuropsychiatric assessment (category 3), and
a normal educational assessment at 6.5 years of age as well as a normal report were obtained in 1 child, allowing us categorize this child to category 1.

All but 1 infant had arterial umbilical cord pH in this study, and all but 3 had arterial umbilical BDecf values recorded at birth. At the single occasion when both arterial values were missing, umbilical venous samples were available. This made it possible to categorize this infant to the acidotic group. The child was treated at the SCBU and followed at the special development assessment clinic.

Table 2 provides a detailed description of the characteristics of the study population.

There were in all 13 children with a poor outcome: 4 died (category 5) and 9 had abnormal neurodevelopment (category 4). Of the children who died (1 boy, 3 girls) in the perinatal period, all had severe metabolic acidosis (range for pH 6.62–6.97, and for BDecf 15.3–19.9) and low Apgar scores measured at 1, 5, and 10 minutes (range, 0–2, 0–7, and 0–8). Three of the children had a severe encephalopathy according to Sarnat and Sarnat criteria,15 and the fourth (with Apgar score 2, 7, 8) died in *Escherichia coli* sepsis. The 4 infants in the acidotic group, who were later assigned a neurodevelopmental diagnosis, all had an affected Apgar score (range, 0–5, 0–6, and 4–7), low pH (range, 6.87–7.02), and high BDecf (range, 13.1–15.3). Clinical signs of encephalopathy15 were seen in 3 of these infants, and the fourth had meconium aspiration. This child later received a diagnosis of atypical autism. Four of these 8 infants were delivered by cesarean delivery, 2 by vacuum extraction, and the remaining 2 by spontaneous cephalic delivery. The infants treated at the SGBU received care according to the current routines at the time, eg, no hypothermia was given for encephalopathy.

None of the 5 children in the control group who, at 6.5 years of age, had a neurodevelopmental diagnosis was in the need of hospital care during the neonatal period. They had no sign of asphyxia (Apgar score range, 7–9, 9–10, and 9–10), had a normal pH (range, 7.17–7.31), and BDecf (range, —3.92 to 3.70), and no clinical sign of encephalopathy. At 6.5 years of age, 2 had Ehler-Danlos syndrome, both with known heredity, 1 had Asperger syndrome, 1 had attention-deficit/hyperactivity disorder in need of medical treatment, and the fifth had a generalized mild developmental delay.

A flowchart of the population, including the distribution between the different groups, is shown in Fig 1. Table 3 shows the distribution of cases in the different categories.

All infants born with metabolic acidosis who appeared healthy at birth and who were not treated in the SCBU (A2) were without neurodevelopmental diagnosis at 6.5 years of age, but 8 (8/33 = 24%) were categorized as having a divergence for speech, language, behavior, or motor problems where referral action was taken or had specific educational arrangements in the school system (category 3).

Four of the 13 infants who were followed at the special assessment clinic (A3) developed a neurodevelopmental diagnosis (category 4); of the remaining 9, 3 were classified to category 3. In total, 54% of the children in this group had special needs (7/13). None of the 59 infants with metabolic acidosis who only followed the normal CHS program and the school health program (A4) developed a neurodevelopmental diagnosis (category 4), but 13 infants fulfilled the criterion for category 3 (13/59 = 22%).

In the control group (B), referral actions or educational arrangements had been done in 31 of the 151 (21%) (the 5 with a neurodevelopmental diagnosis included) and, in the total acidotic group, 24 of 76 (A) (32%) had an adverse outcome (dead, neurodevelopmental diagnosis, or referral actions).

No statistical differences were found when the control group (B) was compared with acidotic infants who appeared healthy at birth and were not admitted to the SCBU (A2) nor in a comparison of the control group with the acidotic infants who had only ordinary health care follow-up (A4). In this comparison, the 26 infants who had initially been admitted to the SCBU but who were only followed up at the regular CHS are included. A statistical difference was found when all children who had been acidotic at birth (A) were compared with the control group (B).

**DISCUSSION**

This study shows that term infants with severe metabolic acidosis in cord blood who appear healthy at birth do not have an increased risk of neurologic or behavioral problems in need of referral actions or educational support at the age of 6.5 years. Likewise, acidotic newborns that were transferred for supervision to the SCBU but did not exhibit symptoms of encephalopathy did not have increased risks of problems at 6.5 years.

However, a difference emerges when the entire cohort of acidotic children in this population is compared with the control group. In the acidotic group, 10.6%, and, in the control group, 3.3% exhibited a neurodevelopmental diagnosis or died. This difference is related to increased risks for children with acidosis and neonatal encephalopathy.

Only a few studies have addressed the question of long-term outcome in healthy infants with metabolic acidosis at birth. Malin et al3 conclude that low cord pH is substantially associated with neonatal mortality and morbidity and cerebral palsy in childhood and that these outcomes justify increased surveillance of infants born with a low cord pH. The studies published have
TABLE 2 Characteristics of Study Population

<table>
<thead>
<tr>
<th>Measure</th>
<th>SCBU, A1</th>
<th>Maternity Ward, A2</th>
<th>Special Developmental Assessment, A3</th>
<th>Child Health Clinic, A4</th>
<th>Control Group, B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>43</td>
<td>35</td>
<td>13</td>
<td>61</td>
<td>156</td>
</tr>
<tr>
<td>Gender: boys/girls (% boys)</td>
<td>30/13 (70)</td>
<td>17/18 (49)</td>
<td>9/4 (69)</td>
<td>37/24 (61)</td>
<td>94/62 (60)</td>
</tr>
<tr>
<td>Gestation: wk + d</td>
<td>40±5, NS (37±6 to 43±1)</td>
<td>41±0, NS (37±6 to 42±2)</td>
<td>41±2 NS (37±6 to 42±2)</td>
<td>40±6 NS (37±6 to 43±1)</td>
<td>40±6 (37±0 to 43±1)</td>
</tr>
<tr>
<td>Method of birth ****</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Spontaneous cephalic, n (%)</td>
<td>18 (42)**</td>
<td>29 (83)</td>
<td>7 (54)</td>
<td>39 (64)</td>
<td>115 (74)</td>
</tr>
<tr>
<td>Caesarean delivery, n (%)</td>
<td>18 (42)**</td>
<td>5 (14)</td>
<td>3 (23)</td>
<td>9 (15)</td>
<td>25 (16)</td>
</tr>
<tr>
<td>Instrumental (vacuum extraction), n (%)</td>
<td>7 (16)**</td>
<td>1 (3)</td>
<td>3 (23)</td>
<td>13 (21)</td>
<td>16 (10)</td>
</tr>
<tr>
<td>Birth weight, g</td>
<td>3540 NS (2580–4920)</td>
<td>3555 NS (2580–4935)</td>
<td>3480 NS (2615–4120)</td>
<td>3540 NS (2580–4935)</td>
<td>3650 (2650–5630)</td>
</tr>
<tr>
<td>Umbilical artery, Bdecf</td>
<td>14.73**** (12.27–19.89)*</td>
<td>12.65**** (12.20–18.57)*</td>
<td>15.04**** (12.27–19.59)*</td>
<td>13.35**** (12.10–17.74)**</td>
<td>3.63 (3.92 to 10.17)</td>
</tr>
<tr>
<td>Apgar score at 1 min 3**** (0)</td>
<td>2**** (0)</td>
<td>2**** (0)</td>
<td>2**** (0)</td>
<td>2**** (0)</td>
<td>2**** (0)</td>
</tr>
<tr>
<td>Apgar score at 5 min 6**** (0)</td>
<td>6**** (0)</td>
<td>6**** (0)</td>
<td>6**** (0)</td>
<td>6**** (0)</td>
<td>6**** (0)</td>
</tr>
<tr>
<td>Apgar score at 10 min 8**** (0)</td>
<td>8**** (0)</td>
<td>8**** (0)</td>
<td>8**** (0)</td>
<td>8**** (0)</td>
<td>8**** (0)</td>
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<tr>
<td>Outcome assessment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ordinary health care follow-up, n</td>
<td>26</td>
<td>35</td>
<td>0</td>
<td>61</td>
<td>156</td>
</tr>
<tr>
<td>Special development assessment, n</td>
<td>13</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>Mortality, n</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lost to follow-up, n</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lead circumference at 18 mo (SDS)</td>
<td>0.13, NS (–0.76 to 2.51)</td>
<td>0.64 NS (–1.44 to 2.41)</td>
<td>0.13 NS (–0.76 to 1.06)</td>
<td>0.24 NS (–2.15 to 2.51)</td>
<td>0.13 (–2.89 to 2.35)</td>
</tr>
<tr>
<td>Weight at 6.5 y (SDS)</td>
<td>–0.28** (–2.66 to 2.04)*</td>
<td>0.74 NS (–1.58 to 4.71)</td>
<td>–0.67** (–2.60 to 1.41)*</td>
<td>0.03 NS (–2.66 to 4.71)</td>
<td>0.31 (–2.94 to 5.11)</td>
</tr>
<tr>
<td>Height at 6.5 y (SDS)</td>
<td>–0.21** (–2.71 to 1.87)**</td>
<td>0.48 NS (–1.65 to 1.97)</td>
<td>–0.58** (–2.47 to 0.61)**</td>
<td>0.18 NS (–2.71 to 1.97)</td>
<td>0.34 (–2.87 to 3.38)</td>
</tr>
<tr>
<td>BMI</td>
<td>15.53 NS (13.15–19.72)</td>
<td>16.57 NS (13.98–25.41)</td>
<td>14.86 NS (13.15–19.23)</td>
<td>15.90 NS (13.31–25.41)</td>
<td>15.84 (12.01–24.68)</td>
</tr>
</tbody>
</table>

The figures are shown as median (range). The statistical calculations are made in comparison with the control group (B). SDS, SD score (z score); NS, not significant. *** P < .0001, ** P < .001, * P < .01, P < .05. * Significant results are indicated with *.

TABLE 3 Distribution of Cases (n, %) in the Different Categories

<table>
<thead>
<tr>
<th>Category</th>
<th>Complete Acidotic Group, A</th>
<th>Acidotic SCBU, A1</th>
<th>Acidotic, No SCBU, A2</th>
<th>Acidotic, Special Developmental Assessment, A3</th>
<th>Acidotic, Ordinary Health Care, A4</th>
<th>Control Group, B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41 (53.9%)</td>
<td>22 (51.2%)</td>
<td>19 (57.6%)</td>
<td>5 (38.5%)</td>
<td>36 (61.0%)</td>
<td>95 (61.6%)</td>
</tr>
<tr>
<td>2</td>
<td>11 (14.5%)</td>
<td>5 (11.8%)</td>
<td>6 (18.2%)</td>
<td>1 (7.7%)</td>
<td>10 (16.9%)</td>
<td>27 (17.9%)</td>
</tr>
<tr>
<td>3</td>
<td>16 (21.1%)</td>
<td>8 (18.6%)</td>
<td>8 (24.2%)</td>
<td>3 (23.1%)</td>
<td>13 (22.0%)</td>
<td>26 (17.2%)</td>
</tr>
<tr>
<td>4</td>
<td>4 (5.3%)</td>
<td>4 (9.3%)</td>
<td>0</td>
<td>4 (30.8%)</td>
<td>0</td>
<td>5 (3.3%)</td>
</tr>
<tr>
<td>5</td>
<td>4 (5.3%)</td>
<td>4 (9.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lost to follow-up</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Sum</td>
<td>78</td>
<td>43</td>
<td>35</td>
<td>13</td>
<td>61</td>
<td>156</td>
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<tr>
<td>χ² test</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Significance</td>
<td>P &lt; .0001***</td>
<td>NS</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

For definition of categorization definition see Table 1. NS, not significant.

conflicting results, varying inclusion criteria concerning infants with or without symptoms of encephalopathy, and varying definitions of acidosis (pH level) (see the introduction). The studies published have not had the same main goal as this study, namely to separate the “healthy” and “sick” acidotic infants. In light of this, our study brings new knowledge. We demonstrate that metabolic acidosis in cord blood is not associated with increased developmental problems at the age when children start school, unless acidosis is combined with encephalopathy.

The anthropometric data for the acidotic children defined as healthy in the newborn period (groups A2 and A4) and controls were similar. The significant difference seen for the other groups (A1 and A3) is expected, because they include the children with a neurodevelopmental diagnosis.

**Strengths of This Study**

We have a population-based study where the matching of controls was done prospectively at birth according to strict criteria. The incidence of metabolic acidosis in cord blood is very similar to that...
observed in another contemporaneous population-based material from West Sweden.13 In the acidotic group, 8 of 78 infants died in the neonatal period or developed a neurodevelopmental diagnosis. For 2 of these infants, no obvious clinical signs of encephalopathy were found. These figures correspond to those in clinical material from similar demographic situations1,13 and suggest that our material is representative.

Five children in the control group had a neurodevelopmental diagnosis. For the children with attention-deficit/hyperactivity disorder, Asperger syndrome, and mild mental retardation, this might be expected. Concerning the 2 children with Ehlers-Danlos syndrome, we have no other explanation than chance.

Very high follow-up rates were achieved. All but 3 school records possible to find were located and scrutinized. If we include the records of the 4 who died in the neonatal period, 227 of 234 (97%) records could be found and the outcome information retrieved. This was made possible by the Swedish national registration system in combination with the national CHS and school health program where all children have health and development assessments at defined ages.

The same nurse with long experience of the Swedish school health system scrutinized all records and filled in the protocols. Data extraction from these protocols and evaluation and categorization (Table 1) were performed by 2 experienced pediatricians who at that time were blinded to the conditions at birth. These factors increase the accuracy of the material.

The use of strictly defined metabolic acidosis levels instead of just pH levels increases the accuracy of the study. This is discussed in the introduction. All but 1 infant in this study had arterial cord pH values, and arterial BDeq values were recorded in all but 3. This was made possible by strict clinical routines in the delivery wards for this procedure. The advantage of also basing the study on umbilical arterial samples instead of mixed or venous samples is well documented.14

Weakness of This Study
The categorization of being healthy in the neonatal period (group A2 and A4) is partly a subjective judgment. However, all acidotic infants who developed a neurodevelopmental diagnosis were treated in the SCBU and assigned to this follow-up. We believe that the corresponding results in these 2 comparisons groups (A2 and A4) strengthen the result that metabolic acidosis in the perinatal period above all increases the risk for neurologic or behavioral problems or need of referral actions or educational arrangements at the age of 6.5 years if the infant already shows symptoms during the neonatal period.

We believe that the differences we obtain when including all acidotic infants in a comparison strengthen these results.

Outcome data were retrospectively collected. This is in part compensated by the fact the matching of controls at birth was done prospectively. The disadvantages of making chart reviews and not examining all children are obvious. The possibility that minor neurodevelopmental problems at the age of 6.5 and after only 0.5 to 1 year in the school system are not yet discovered or are not yet reported must be taken into consideration. This problem is assumed to be similar in the control group.

The reservation that a larger cohort of acidotic infants could possibly detect less frequent divergences must be made.

CONCLUSIONS
Infants born with cord metabolic acidosis and who appear well do not have an increased risk for neurologic or behavioral problems in need of referral actions or pedagogic arrangements at the age of 6.5 years.

ACKNOWLEDGMENTS
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