Neurologic Activity of Infants Following
Anesthesia for Cesarean Section

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Elective cesarean section was performed in a consecutive series
of 30 patients with full-term pregnancies who were not in labor.
Epidural (lidocaine, 1.5 per cent, with epinephrine, 1:200,000)
and general anesthesia (thiopental, nitrous oxide–oxygen, su-
cinylcholine infusion) was used alternately. Neonatal acid–base
values and Apgar scores showed no significant difference between
the two anesthetic groups, and most infants were vigorous at birth.
The neurologic recoveries of the infants showed no significant dif-
cference between the two groups. In the group receiving epidural
anesthesia, there was a significant correlation between maternal
hypotension and weak rooting and sucking reflexes of the infants
during the first two days. All infants of high-risk obstetric patients
in the series, independent of anesthetic technique used, had ab-
normal neurologic activity, as evidenced by either depression of
muscle tone and the reflexes or all the tested variables. Neurologic
assessment as followed in this series is a sensitive indicator of
the effects of fetal stress factors acting during cesarean section. (Key
words: Anesthesia, obstetric; Surgery, cesarean section; Anes-
thetic techniques, peridural, balanced; Toxicity, neurotoxicity.)

There has recently been an increasing interest in exploring the subtle neonatal effects of drugs used
for maternal sedation or analgesia during labor.†–§
These studies have been done in groups of healthy
mothers and babies after normal vaginal deliveries.
To our knowledge, there has been no similar study of
infants born by cesarean section. However, both the
operative trauma and the anesthetic stress of an ab-
dominal delivery far exceed those of vaginal delivery,
as shown by Apgar scores, neonatal acid–base values,
and neonatal morbidity and mortality figures.⁶–⁸

This study compares the effects of epidural and
general anesthesia on neonatal neurologic activity
during the first week of life. We studied a series of
elective abdominal deliveries from our routine prac-
tice.

Methods and Materials

The patients were selected during an approximately
three-month period from parturients at term who

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were scheduled to have elective cesarean section. Epidural and general anesthesia were used in alternate
patients. The day before operation, the anesthesiologist visited the patient, explained the study, and
obtained her consent.

Most of the 30 patients (15 in each group) were
healthy, with uncomplicated full-term pregnancies.
Three patients in either group had antenatal stress
factors (mild toxemia, diabetes, or hypertension) and
one patient who had general anesthesia had partial
placenta previa and transverse lie. The membranes
were intact and the patients were not in labor. Most
of the patients were having cesarean section because
of fetopelvic disproportion. In each case, the infant
was a singleton. One of the infants in each group
was small for date. Gestational age was estimated using
the first day of the last menstrual period, and in cer-
tain cases, ultrasound analysis and amniotic fluid sam-
ping for determination of the lecithin/sphingomyelin
ratio were performed. Routine pediatric examination
immediately after delivery showed no premature in-
fant in the series. Other pertinent data are presented in
table 1.

Before induction of anesthesia, during preparatory
steps, the patient spent about 30 min on the operating
table with a wedge under the right hip and the uterus
placed to the left. A radial-artery cannula was
placed percutaneously during local anesthesia for re-
cording blood pressure and obtaining blood samples.
Mean arterial pressure (MAP) was recorded using a
Statham pressure transducer and a Hewlett-Packard
multichannel recorder. Maternal Electrocardiogram
and heart rate were also recorded continuously.

All anesthesia were administered by the authors,
and all operations were performed by the same group
of surgeons. Before epidural anesthesia, the parturi-
tent received a fluid load of gelatin solution (Haemacel),
500 ml of 3.5 per cent, within 10 to 15 min, fol-
lowed by lactated Ringer's solution, 500 ml (first nine
patients in the series) or 1,000 ml (last six). Epidural
block was done with the patient in the lateral position
using the L3–L4 interspace. Lidocaine, 18 ml of 1.5
per cent, with epinephrine, 5 μg/ml, was injected
through a Tuohy needle after a 2-ml test dose was
given. This resulted in a satisfactory (T5–T8 level)
operative block in every case. After the block, the
mother breathed oxygen (2 l/min) continuously.

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through a nasal catheter. Mean induction-to-delivery
time was 24 min (range 15–45 min). Hypotension was
treated with ephedrine, 5 to 25 mg, intravenously.

In the general anesthesia group, atropine, 0.5 mg,
was given intravenously before induction of anes-
thesia. Oxygen was administered by mask, after which
thiopental, 4 mg/kg, was injected and endotracheal
intubation was accomplished with the aid of succinyl-
choline, 1 mg/kg. Anesthesia was maintained with a
mixture of nitrous oxide and oxygen 1:1 and succinyl-
choline infusion. Glucose solution was not given dur-
ing anesthesia. Ventilation was controlled with an
Engström respirator using a nonrebreathing tech-
nique (respiratory rate 16/min, minute volume 9–10
l/min). The mean induction-to-delivery time was 8 min
(range 6–11 min).

Maternal arterial blood samples were obtained im-
mediately before anesthesia and at delivery to deter-
mine \( \rhoH \), \( P_{\text{a}} \), and \( P_{\text{CO}_2} \), base deficit, and blood glucose.
At the time of delivery, the umbilical cord was clamped
and samples of umbilical artery (\( U_a \)) and umbilical
vein (\( U_v \)) blood were drawn for determination of
acid-base values, \( P_{\text{a}} \), and \( U_v \) lidocaine concentrations.
Blood samples from infants were obtained from ar-
terialized heel punctures 15 and 30 min, 1, 2 and 12
hours after delivery for determination of \( \rhoH \), \( P_{\text{CO}_2} \),
base deficit and blood glucose. The blood samples
for acid-base and \( P_{\text{a}} \) measurements were drawn in
glass syringes whose dead space was filled with hepa-
rin. Samples were taken anaerobically and analyzed
immediately. \( \rhoH \), \( P_{\text{CO}_2} \), and \( P_{\text{a}} \) were determined with a
Radiometer BMS 2 analyzer. Base deficit was cal-
culated using the Sigggaard-Andersen nomogram.
Samples for glucose determination were taken in
ethylenediaminetetra-acetic acid tubes and analyzed
immediately using a glucose oxidase method.†† The
lidocaine concentration in \( U_v \) plasma was analyzed
by gas chromatography..

†† Boehringer Mannheim GmbH Diagnostica, Mannheim West
Germany: Blood sugar, GOD-perid method. Gemsaeq centrifugal

Neurologic assessment of infants was based on
standard testing as developed by Prechtl and Bein-
tema (see Appendix). The examination involves as-
essment of infant motility, muscle tone, reflexes and
general reactions. The examination was performed
with the infant in optimal condition. The examiner
(M.K.) was unaware of the anesthetic or obstetric man-
agement and was not involved in the care of the
infant. All 30 infants were examined at the ages of 2, 4,
and 8 hours, and 1, 2, 3, 4, and 7 days. Altogether,
240 separate examinations were made. Student's \( t \) test
and the chi-square probabilities for \( 2 \times 2 \) tables were
used for statistical analyses. \( \dagger \) \( P < 0.05 \) was con-
sidered significant.

Table 1. Characteristics of the Study Population

<table>
<thead>
<tr>
<th></th>
<th>Anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Epiglottal</td>
</tr>
<tr>
<td></td>
<td>( n = 15 )</td>
</tr>
<tr>
<td>Maternal age</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>27.1</td>
</tr>
<tr>
<td>Range</td>
<td>21–43</td>
</tr>
<tr>
<td>Parity</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>1.8</td>
</tr>
<tr>
<td>Range</td>
<td>1–4</td>
</tr>
<tr>
<td>Gestational week</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>38.9</td>
</tr>
<tr>
<td>Range</td>
<td>37–40</td>
</tr>
<tr>
<td>Infant birth weight (g)</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>3390</td>
</tr>
<tr>
<td>Range</td>
<td>2,600–6,200</td>
</tr>
<tr>
<td>Infant sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>7</td>
</tr>
<tr>
<td>Female</td>
<td>8</td>
</tr>
<tr>
<td>Apgar score</td>
<td></td>
</tr>
<tr>
<td>1 min</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>9.0</td>
</tr>
<tr>
<td>Range</td>
<td>8–10</td>
</tr>
<tr>
<td>5 min</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>9.5</td>
</tr>
<tr>
<td>Range</td>
<td>8–10</td>
</tr>
<tr>
<td>15 min</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>9.6</td>
</tr>
<tr>
<td>Range</td>
<td>8–10</td>
</tr>
</tbody>
</table>

Results

Following epidural anesthesia, MAP decreased
from 105 ± 3.0 (SE) torr to 91 ± 5.3 torr during the
first 10 min after block, and increased thereafter. Nine
of 15 patients had decreases in MAP of more than 17
torr. In six patients, MAP decreased to below 70 torr.

\( \dagger \) Goyette D, Mickey MR: Chi square probabilities for \( 2 \times 2 \) ta-
bles. Technical Report No. 15, Health Sciences Computing Facility,
University of California, Los Angeles, California 90024.
With prior infusion of lactated Ringer's solution, 1,000 ml, and Hemaccel, 500 ml, in the last six patients, hypotension did not occur. In the patients given general anesthesia, MAP increased from 113 ± 3.9 torr to 138 ± 6.1 torr at the time of incision of the skin. Infant heart rate, respiratory rate and rectal temperature showed no difference between the two groups during the first 12 hours of life. The infants were clinically healthy and vigorous, having Apgar scores of 8 or more with two exceptions. The infant of a mother with a placenta previa had Apgar scores of 3, 7, and 8 at 1, 5, and 15 min. This infant's trachea was intubated and ventilation was controlled and assisted for 15 min. She needed oxygen, 40 per cent, for four hours, but the later course was uneventful. Another mother, a 35-year-old multigravida who had low pre- and intraoperative PaO₂ values, delivered an infant with a low U₃, PaO₂ value (17 torr) and Apgar scores of 6, 9, and 9 at 1, 5, and 15 min. In the epidural-anesthesia group, there was one infant who had an Apgar score of 8 at 1 min who had aspirated clear liquid during delivery and needed intubation of the trachea to clear the airway; this infant received oxygen, 40 per cent, for eight hours. The significantly higher maternal PaCO₂ and PaO₂ values during general anesthesia (table 2) were due to controlled ventilation with a preset volume and a higher inspired concentration of oxygen. The slightly higher U₃, P₃CO₂ values following general anesthesia represented the only significant difference between U₃ and U₃ values, and reflected the higher maternal PaCO₂ value in that group.

There was no statistically significant difference in neonatal mean-acid–base and glucose values during the first 12 hours of life (table 3). There were steady increases in pH due to steady decreases in P₃CO₂ values in both groups. The mean U₃ lidocaine concentration was 0.76 μg/ml ± 0.06.

In four infants whose mothers had hypotension after epidural anesthesia, pH values 15 min after delivery were below 7.20. Two of the infants of mothers given general anesthesia had pH values below 7.20 during the first 30 min after birth. One of the mothers had a placenta previa. The other mother had mistakenly been allowed to lie on the operating table with-

### Table 2. Maternal Arterial, Umbilical Arterial, and Venous Blood pH, P₃CO₂, PaO₂ and Base Deficit, Mean ± SD

<table>
<thead>
<tr>
<th></th>
<th>Maternal Arterial before Anesthesia</th>
<th>Maternal Arterial at Delivery</th>
<th>Umbilical Venous</th>
<th>Umbilical Arterial</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epidural anesthesia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.41 ± 0.04</td>
<td>7.39 ± 0.04</td>
<td>7.34 ± 0.03</td>
<td>7.29 ± 0.03</td>
</tr>
<tr>
<td>P₃CO₂ (torr)</td>
<td>31 ± 2</td>
<td>30.0 ± 4.5</td>
<td>30.0 ± 3.8</td>
<td>48.8 ± 5.3</td>
</tr>
<tr>
<td>Base deficit (mmol/l)</td>
<td>4.1 ± 2.4</td>
<td>5.3 ± 2.7</td>
<td>4.8 ± 1.8</td>
<td>4.5 ± 2.2</td>
</tr>
<tr>
<td>P₃O₂ (torr)</td>
<td>96 ± 19</td>
<td>118.5 ± 30.0</td>
<td>30.8 ± 6.8</td>
<td>18.3 ± 4.5</td>
</tr>
<tr>
<td><strong>General anesthesia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.45 ± 0.03</td>
<td>7.36 ± 0.04*</td>
<td>7.33 ± 0.04</td>
<td>7.29 ± 0.04</td>
</tr>
<tr>
<td>P₃CO₂ (torr)</td>
<td>26 ± 4</td>
<td>34.5 ± 3.8*</td>
<td>42.3 ± 5.3</td>
<td>50.8 ± 5.3*</td>
</tr>
<tr>
<td>Base deficit (mmol/l)</td>
<td>4.0 ± 2.1</td>
<td>5.1 ± 2.7</td>
<td>4.0 ± 2.1</td>
<td>4.3 ± 2.2</td>
</tr>
<tr>
<td>P₃O₂ (torr)</td>
<td>96 ± 14</td>
<td>159 ± 38.3*</td>
<td>31.5 ± 7.5</td>
<td>17.3 ± 5.3</td>
</tr>
</tbody>
</table>

* P < 0.05.

### Table 3. pH, P₃CO₂, Base Deficit and Blood Glucose (Means ± SD) in Newborn Infants after Epidural and General Anesthesia for Cesarean Section

<table>
<thead>
<tr>
<th></th>
<th>15</th>
<th>30</th>
<th>60</th>
<th>120</th>
<th>Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epidural anesthesia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.21 ± 0.07</td>
<td>7.24 ± 0.08</td>
<td>7.33 ± 0.06</td>
<td>7.35 ± 0.05</td>
<td>7.34 ± 0.04</td>
</tr>
<tr>
<td>P₃CO₂ (torr)</td>
<td>64.4 ± 10.0</td>
<td>59.9 ± 11.0</td>
<td>45.0 ± 9.0</td>
<td>45.0 ± 6.8</td>
<td>42.0 ± 6.0</td>
</tr>
<tr>
<td>Base deficit (mmol/l)</td>
<td>3.1 ± 3.7</td>
<td>3.8 ± 4.3</td>
<td>1.5 ± 3.6</td>
<td>0.7 ± 3.1</td>
<td>3.1 ± 2.5</td>
</tr>
<tr>
<td>Blood glucose (mmol/l)</td>
<td>2.5 ± 0.9</td>
<td>2.4 ± 0.8</td>
<td>2.3 ± 1.0</td>
<td>2.6 ± 0.7</td>
<td>3.0 ± 0.5</td>
</tr>
<tr>
<td><strong>General anesthesia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.23 ± 0.07</td>
<td>7.29 ± 0.09</td>
<td>7.30 ± 0.07</td>
<td>7.35 ± 0.06</td>
<td>7.36 ± 0.04</td>
</tr>
<tr>
<td>P₃CO₂ (torr)</td>
<td>65.0 ± 12.0</td>
<td>53.8 ± 10.0</td>
<td>47.3 ± 10.0</td>
<td>42.8 ± 9.0</td>
<td>42.0 ± 6.0</td>
</tr>
<tr>
<td>Base deficit (mmol/l)</td>
<td>2.8 ± 5.2</td>
<td>1.8 ± 5.8</td>
<td>2.3 ± 4.0</td>
<td>0.9 ± 3.0</td>
<td>2.2 ± 2.3</td>
</tr>
<tr>
<td>Blood glucose (mmol/l)</td>
<td>2.3 ± 0.4</td>
<td>2.0 ± 0.4</td>
<td>2.2 ± 0.7</td>
<td>2.5 ± 0.4</td>
<td>2.6 ± 0.3</td>
</tr>
</tbody>
</table>
out lateral tilt before the start of anesthesia. Her systolic blood pressure decreased to 80 torr but was corrected to more than 95 torr by turning her to a lateral position. The Uₐ and Uₐ-Pₐₐ values were in the normal range, and Apgar scores were 9, 9, and 9 at 1, 5, and 15 min.

Four infants of mothers given epidural anesthesia and three of mothers given general anesthesia had hypoglycemic serum glucose values (1.0 to 1.6 mmol/l) during the first two hours of life.

The neurologic assessment showed that 55 per cent of the infants in both groups were normal except for some weak reflexes during the first few hours after birth. The most common finding in those infants evaluated as abnormal was absence or weakness of integrated reflexes. To summarize the observations of the five examinations during the first two days of life, there was a significantly greater number of infants who lacked or had weak sucking, rooting, and Palmar grasp reflexes following epidural anesthesia (table 4). In the infants of mothers given epidural anesthesia there was a significant correlation between maternal hypotension and weak rooting and sucking reflexes of infants at 1 and 2 days of age. After the third day of life, however, there was only one infant with absent or weak reflexes. Muscle tone, motility, and reaction type showed no significant difference between the two groups.

Following epidural anesthesia five of the six infants whose mothers had the greatest decrease in MAP showed abnormal neurologic responses. Where the duration of hypotension was longest, the infant was listless, hypotonic and hypokinetic, with weak or absent reflexes and slow recovery requiring seven days (table 5). The mother who had the shortest and least decrease of MAP below 70 torr (67 torr for 2 min with a systolic blood pressure above 100 torr) had an infant with no neurologic abnormality. Three of the mothers given general anesthesia had received clonidine treatment for hypertension. All three infants were hypotonic, and two were drowsy (table 6).

### Discussion

Clinically, nearly all of the infants in our series were vigorous and in good condition immediately after delivery. Mean Uₐ and Uₐ blood acid–base as well as Pₐₐ values were well within the range observed in other series where lateral tilt and comparable anesthetic techniques have been used.¹¹ Neonatal pH, Pₐₐ and base deficit values during the 12-hour follow-up period compared well with findings in a similar study,¹¹

#### Table 4. Numbers of Infants with Absent or Weak Reflexes during the First Week of Life Following Epidural or General Anesthesia (E = Epidural, G = General)

<table>
<thead>
<tr>
<th>Age at Examination</th>
<th>Sucking</th>
<th>Rooting</th>
<th>Moro</th>
<th>Palmar Grasp</th>
<th>Plantar Grasp</th>
<th>Stepping</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 h</td>
<td>E G</td>
<td>G E G</td>
<td>E G</td>
<td>E G E</td>
<td>E G E</td>
<td>E G</td>
</tr>
<tr>
<td>4 h</td>
<td>5 7 4 5</td>
<td>5 3 2 3</td>
<td>3 2 4 4</td>
<td>4 5 6 3</td>
<td>3 4 5 4</td>
<td>4 5</td>
</tr>
<tr>
<td>8 h</td>
<td>5 2 4 6</td>
<td>6 2 4 3</td>
<td>3 1 2 0</td>
<td>2 0 2 0</td>
<td>2 1 0 2</td>
<td>1 1</td>
</tr>
<tr>
<td>1 d</td>
<td>4 2 6 2</td>
<td>2 1 2 1</td>
<td>1 0 1 2</td>
<td>0 1 2 0</td>
<td>0 1 2 0</td>
<td>1 1</td>
</tr>
<tr>
<td>2 d</td>
<td>4 1 5 1</td>
<td>1 1 1 1</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 1</td>
</tr>
<tr>
<td>3 d</td>
<td>1 1 1 1</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 1</td>
</tr>
<tr>
<td>4 d</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 1</td>
</tr>
<tr>
<td>7 d</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 0 1 0</td>
<td>1 1</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>31 16 32 16 13 5 15 5 15 6 19 13</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Table 5. Cases in Which Infants had Abnormal Neurologic Activity Following Epidural Anesthesia

<table>
<thead>
<tr>
<th>Maternal Complicating Factors</th>
<th>Maternal Hypotension after Epidural</th>
<th>Duration of Abnormal Response to Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient 1</td>
<td>None</td>
<td>42 8</td>
</tr>
<tr>
<td>Patient 3</td>
<td>Mild toxemia; obesity; Pₐₐ 64 torr at delivery</td>
<td>58 4</td>
</tr>
<tr>
<td>Patient 9</td>
<td>Diabetes (class C)</td>
<td>55 3</td>
</tr>
<tr>
<td>Patient 7</td>
<td>None</td>
<td>52 1</td>
</tr>
<tr>
<td>Patient 4</td>
<td>None</td>
<td>88†</td>
</tr>
<tr>
<td>Patient 10</td>
<td>None</td>
<td>97‡</td>
</tr>
<tr>
<td>Patient 13</td>
<td>Diabetes (class D)</td>
<td>108</td>
</tr>
</tbody>
</table>

* White's classification.
† 25-torr decrease in MAP after block.
‡ MAP before block 87 torr.
Table 6. Cases in Which Infants had Abnormal Neurologic Activity Following General Anesthesia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Maternal Complicating Factors</th>
<th>Duration of Abnormal Response to Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>28</td>
<td>Diabetes (class D)* and mild toxemia; clonidine medication</td>
<td>7 days (3 days all variables)</td>
</tr>
<tr>
<td>16</td>
<td>35-year-old B-3, p-4; blood pressure 170/100 torr before induction; $P_{0.9}$ 75 torr† during induction–delivery interval</td>
<td>4 days (3 days all variables except motility)</td>
</tr>
<tr>
<td>21</td>
<td>Transverse lie, placenta previa</td>
<td>1 day (all variables)</td>
</tr>
<tr>
<td>26</td>
<td>Essential hypertension; clonidine medication</td>
<td>8 hours (all variables)</td>
</tr>
<tr>
<td>27</td>
<td>Healthy mother who had previously delivered three chondrodys trophytropic children; prolonged apnea after succinylcholine</td>
<td>8 hours (all variables)</td>
</tr>
<tr>
<td>23</td>
<td>Healthy mother who had supine hypotension before anesthesia; metabolic acidosis (base deficit 9.2 mmol/l) at delivery</td>
<td>8 hours (4 hours all variables)</td>
</tr>
<tr>
<td>24</td>
<td>Essential hypertension; clonidine medication</td>
<td>1 day (muscle tone and reflexes‡)</td>
</tr>
</tbody>
</table>

* White’s classification.
† $P_{0.9} = 0.5$.
‡ Small for date.

Table 6 continues...
enough to cause fetal hypoxia and subsequent depressed motor performance lasting longer than a few hours.

A severe long-lasting decrease in maternal blood pressure can lead to decreased interstitial blood flow, fetal hypoxia and hypotension, decreased cerebral perfusion, and neonatal cerebral insults. In two of our cases there was postepidural hypotension, which added to the obstetric stress factors acting on the fetus (table 5). The chronic decrease in placental blood flow caused by toxemia or diabetes added to the acute maternal hypotension could explain the neurologic abnormalities in these infants. That a shortlived hypotension alone might cause neurologic abnormalities in three of the full-term infants of healthy mothers was surprising. However, recent measurements of interstitial blood flow with a noninvasive intravenous 133Xe method have shown a decrease in interstitial blood flow with the decrease in maternal MAP after epidural anesthesia. Improvement in the interstitial blood flow lagged several minutes behind the normalization of MAP established with ephedrine given intravenously (Hollmen A, unpublished data). Also, in the present series, the short-lived hypotension probably caused a decrease of interstitial blood flow, which lasted several minutes after maternal hypotension had been corrected with ephedrine.

Following general anesthesia, where there were either pre- or perinatal stress factors acting together, neurologic depression of infants occurred in all but one case (table 6). In the three infants whose mothers had received clonidine, neurologic depression seems most likely to have been caused by multifactorial effects of maternal hypertension, drugs used for anesthesia, trauma of cesarean section, and possibly clonidine.

Chronic antenatal stresses such as toxemia, diabetes, and hypertension, which decrease placental blood flow during late pregnancy, combined with the acute stress of abdominal delivery, seem to depress the neurologic activity of an infant. In our series all infants of high-risk mothers showed abnormal neurologic recovery independent of anesthetic technique, whereas infants of mothers with no complicating factor generally did well.

There is a need for clinical trials comparing the effects of general versus epidural techniques upon infants of patients with uteroplacental insufficiency. The present study shows that in addition to Apgar score and acid-base balance, such studies should include a more sensitive indicator of the neonatal wellbeing, namely, the infant's neurologic recovery.

*** Kää R: Personal communication.

References
9. Tucker GT: Determination of bupivacaine (Marcaine) and Other anilide-type local anesthetics in human blood and plasma by gas chromatography. Anesthesiology 32:253–260, 1970
APPENDIX

Neurologic Examination

1. Motility

Motility was estimated from the speed, intensity and amount of spontaneous movements. Infants were classified as normokinetic when they had medium speed, intensity, and amount of movements; hyperkinetic when they had high-speed, very intensive movements.

2. Muscle Tone

Resistance against passive movement was tested by moving the neck, trunk, and upper and lower limbs slowly and repeatedly throughout the full range of motion. Also, head control with the infant in the sitting position was tested. Infants with complete lack of resistance or weak resistance were classified as hypotonic; moderate resistance, as normal; strong resistance, as hypertonic.

3. Reflexes and Responses

Reflexes evaluated include:

- **Sucking response.** The examiner elicited sucking movements by inserting a finger or nipple into the mouth of the infant. Responses were classified: absent, no response; weak, weak sucking movements; normal, strong sucking movements.

- **Rooting response.** Rooting response was elicited by touching the perioral skin at the corners of the mouth and the upper and lower lips with the top of the index finger. The following scale was used: absent, no response; weak response, lip movements only; normal response, lip movements and turning of the head toward the stimulated side.

- **Moro response.** The Moro response was elicited by a rapid drop of the infant of a few cm. Abduction at the shoulders, extension at the elbow and threshold for the Moro response were observed. Responses were graded as: absent, no response; weak response, slight abduction at the shoulders (less than 45 degrees) and extension at the elbow less than 90 degrees; normal response, abduction 45 degrees or more and extension 90 degrees or more.

- **Palmar grasp.** Palmar grasp was elicited by inserting the index fingers of the examiner into the hands of the infant from the ulnar side and gently pressing against the palmar surface. Grading was: absent, no response; weak, weak grasp; normal, strong grasp.

- **Plantar grasp.** The examiner elicited the plantar grasp by gently pressing the thumbs against the balls of the infant’s feet. Responses were classified as: absent, no response; weak, weak and unsustained plantar flexion of the toes; normal, good plantar flexion of the toes.

- **Stepping movements.** Stepping movements were elicited by holding the infant upright and allowing the soles of the feet to touch the surface of the examination table. Responses were graded as: absent, no stepping movements; weak, an attempt to make stepping movements or one step; good, at least two steps.

4. General Reactions

General reactions were assayed as listless, normal, or hyperexcitable. This included the observation and scoring of the infant’s awake and sleep states before and during testing, response to pain stimulus, alertness, and tremor, as well as intensity and threshold for reflexes and spontaneous motor activity.