Subdural Hemorrhage and Hypoxia in Infants With Congenital Heart Disease

BACKGROUND AND OBJECTIVES: It has been suggested that there is a causal relationship between hypoxia and subdural hemorrhage (SDH) in infancy. The purpose of this study was to review the incidence of SDH in infants with congenital heart disease and explore the relationship between SDH and hypoxia.

METHODS: Review of data collected for a prospective longitudinal cohort study of infants undergoing surgery for congenital heart disease in New Zealand and Australia. Infants underwent serial MRI scans of the brain in the first 3 months of life. All oxygen saturation recordings and MRI results were extracted and infants assigned to categories by degree of hypoxia. The data were then examined for any statistically significant relationship between hypoxia and SDH.

RESULTS: One hundred fifty-two infants underwent MRI scans, and 66 (43%) had 145 loci of SDH. New SDH was seen in 12 infants after cardiac surgery. Of the loci of SDH, 63 (43%) were supratentorial and over the convexities. Small infratentorial SDHs may persist for ≤90 days. In young infants with congenital heart disease, an association between hypoxia and SDH could not be demonstrated.

CONCLUSIONS: Asymptomatic neonatal SDH is common, resolves within 4 weeks, and is typically infratentorial or posterior when supratentorial. Subdural hematomas may occur after cardiac surgery in infancy. Some hypothesize a causal relationship between hypoxia and SDH in infancy.

WHAT’S KNOWN ON THIS SUBJECT: Asymptomatic neonatal subdural hemorrhage (SDH) is common, resolves within 4 weeks, and is typically infratentorial or posterior when supratentorial. Subdural hematomas may occur after cardiac surgery in infancy. Some hypothesize a causal relationship between hypoxia and SDH in infancy.

WHAT THIS STUDY ADDS: Asymptomatic neonatal SDH is often supratentorial and over the convexities. Small infratentorial SDHs may persist for ≤90 days. In young infants with congenital heart disease, an association between hypoxia and SDH could not be demonstrated.
Subdural hemorrhage (SDH) in infancy is associated with nonaccidental injury. SDH is typically considered traumatic in origin and is a key diagnostic feature of the condition once known as “shaken baby syndrome”. Nowadays, the American Academy of Pediatrics prefers the term “pediatric abusive head trauma” (AHT), in recognition of the fact that in infants who do sustain nonaccidental head injury, there are a variety of possible traumatic mechanisms.

The relationship between infantile SDH and trauma has been challenged in recent years. One series of neuropathological postmortem studies concluded by speculating that in a susceptible infant, “subdural and retinal bleeding might result from any event that initiated apnoea or significant hypoxia, with brain swelling.” Thus, in some infants with unexplained SDH a presumptive diagnosis of AHT might be incorrect. Although controversial, this “unified hypothesis” quickly appeared in court, culminating in a judicial review of criminal prosecutions for AHT in the United Kingdom, where deficiencies in the hypothesis were acknowledged. Several subsequent postmortem studies have found no association between hypoxia and SDH in infancy but other authors continue to suggest a strong association between SDH and hypoxia in the presence of additional factors.

Another factor to consider in young infants is the possibility of SDH resulting from birth trauma. Symptomatic SDH from birth trauma is uncommon and usually associated with complicated or instrumental deliveries. However, MRI revealed that asymptomatic SDH at birth is common. Some studies report a prevalence as high as 50%, although no study so far reports persistence of SDH present at birth beyond 4 weeks. The purpose of this study was to review the incidence of SDH in newborns with congenital heart disease undergoing MRI brain scans as part of a perioperative research protocol and to explore the relationship between SDH and hypoxia.

METHODS

The Hearts and Minds Study is a prospective longitudinal cohort study of infants undergoing surgery for congenital heart disease before 2 months of age at Starship Children’s Hospital, Auckland, New Zealand and the Royal Children’s Hospital Melbourne, Australia. The study protocol is described in detail elsewhere. In brief, participants underwent serial perioperative MRI scans, with collection of physiologic data including oxygen saturations during the primary hospital admission. The study was approved by the institutional review boards for both hospitals. Three MRI scans were performed: before surgery (MRI 1), 7 to 14 days after surgery (MRI 2), and finally at ~2 to 3 months of age (MRI 3).

Data Collection

Scans were performed with a 1.5- or 3.0-T Magnetom Avanto scanner (Siemens, Erlangen, Germany). Standardized sequences included coronal 3-dimensional fluid-attenuated inversion-recovery T1-weighted images (1-mm slice thickness), coronal and axial T2-weighted dual-echo, fast spin-echo images (2-mm slice thickness), and axial diffusion-weighted imaging (12–20 directions, 4-mm slice thickness). In addition, T2 gradient-weighted imaging (3-mm slice thickness) was performed at Starship Children’s Hospital, and susceptibility-weighted imaging (1.5-mm slice thickness) was performed in a Royal Children’s Hospital subset. Scans were reported independently by 2 neuroradiologists blinded to clinical data. The presence, size, location, and description of any SDH were recorded. Oxygen saturation (SaO2) recordings were collected from clinical records by investigators blinded to cardiac diagnosis. For each infant an individual dataset of SaO2 recordings was created: hourly in the PICU and 6 hourly on the ward. Any low SaO2 recorded at any time between regular recordings was also included. Recordings were separated into 3 time intervals: admission until MRI 1 (T1), between MRI 1 and MRI 2 (T2), and between MRI 2 and MRI 3 or discharge (T3).

SaO2 data were analyzed for each individual and categorized as follows for each time interval (T1, T2, T3):

1. No persistent hypoxia, average SaO2 ≥ 91%
2. Moderate persistent hypoxia, average SaO2 81% to 90%
3. Severe persistent hypoxia, average SaO2 ≤ 80%

In addition, acute hypoxia was classified as any acute deviation of ≥15% below that infant’s average SaO2 during that specific time interval.

Statistical Analysis

Data were analyzed by using JMP V10.0 Software (SAS Institute, Inc, Cary, NC). Categorical data were examined by using Fisher’s exact test and continuous data using Wilcoxon’s rank-sum test. When we tested for any relationship between SDH and hypoxia, our null hypothesis was that there is no difference in the proportion of SDH between hypoxia groups, and the 2-tailed test for a difference in proportion in any direction was appropriate. We also applied the 1-tailed test looking specifically for a difference in proportion in the direction of hypoxia, that is, testing for evidence of a positive relationship between SDH and hypoxia.

RESULTS

MRI

One hundred fifty-two infants with MRI scans are included in this analysis. Of these, 12 had 1 MRI scan, 32 had 2, and
108 had 3. Sixty-six infants (43%) had SDH on ≥1 scan. All were neurologically asymptomatic. Ninety-one (60%) had an unassisted vaginal delivery, 16 (11%) had an assisted vaginal delivery, and 45 (29%) had a cesarean delivery. Data on the duration of the stages of labor was not collected. The risk of SDH was not collected. The risk of SDH on MRI 1 differed by mode of delivery (P < .001), with a gradient of risk from cesarean delivery (lowest risk) to assisted vaginal delivery (highest risk) (Table 1). There was no association between SDH on MRI 1 and Apgar score (Table 2). The number of scans by age and the prevalence of SDH are shown in Table 3.

In 7 infants with SDH on MRI 2, findings could not be compared with those of MRI 1 because there was no MRI 1 (4) or T1-weighted images on MRI 1 were absent or incomplete (3). Seven infants with no SDH on MRI 1 had new SDH on MRI 2 (Fig 1). Five infants with SDH on MRI 1 had SDH in new locations on MRI 2. No new SDHs were seen on MRI 3. Infants often had SDH in >1 location. One hundred forty-five loci of SDH were seen in the 66 infants with SDH; 111 loci in 52 infants on MRI 1 and 34 more loci in 19 infants on MRI 2. In those with SDH, other bleeds on MRI 1 included cerebellar hematoma (1), cephalhematoma (2), intraventricular hemorrhage (2), and subgaleal hemorrhage (3). Isolated intraparenchymal petechial hemorrhages were seen in 3 infants on MRI 2. No infants had any of the typical signs of hypoxic-ischemic injury on either diffusion-weighted imaging or T2 scans.

**Progression and Regression of SDH**

Most SDH resolved over time. Of 52 infants with SDH on MRI 1, 50 underwent MRI 2 and 41 underwent MRI 3. Of 108 loci of SDH on MRI 1, 89 (24%) completely resolved by MRI 2 and 55 (21%) completely resolved by MRI 1. In 41 infants who underwent MRI 1 and MRI 3, 83 of 87 loci of SDH present on MRI 1 (95%) resolved by MRI 3 (38 out of 41 infants). Ten of 12 infants with new loci of SDH on MRI 2 also underwent MRI 3. In these infants, 17 out of 18 loci (94%) resolved by MRI 3 (9 out of 10 infants). Five infants still had SDH on MRI 3, all in the posterior fossa. Table 4 presents all 8 infants who had SDH on MRI 1 that persisted beyond 28 days of life.

**Location of SDH**

Of the 145 loci of SDH, 63 (43%) were supratentorial and 82 (57%) infratentorial. Supratentorial SDH were inter-hemispheric, parafalcine, subfalcine, or midline (25); parietal, tempoparietal (18), temporal or subtemporal (12), and occipital or suboccipital (8) (Fig 2). Infratentorial SDH were described as posterior cranial fossa (PCF) (38), below the tentorium (27), in relationship to the cerebellum (16), or below the straight sinus (1) (Fig 3).

**Size of SDH**

Most SDHs (88 out of 145, 61%) were small (≤2 mm deep), 43 (30%) moderate (3–4 mm deep), and 14 (10%) large (≥5 mm deep). None caused a mass effect or necessitated intervention. In 2 infants, SDH enlarged slightly between MRI 1 and 2, but both resolved by MRI 3. One other infant had a 4-mm midline PCF SDH on MRI 2 that was 6 mm on MRI 3. All other SDHs were stable or smaller on serial scans. We did not observe a pattern of increase in SDH size with time.

**Oxygen Saturation**

During T1 and T2, SaO2 recordings were available for most infants with MRI (140 out of 143 infants during T1, 132 out of 138 infants during T2). During T3, recordings were available in only 39 out of 119 infants because most were discharged from the hospital. Therefore, we did not include T3 in the hypoxia analysis. To account for the probability that preoperative and postoperative physiology might differ significantly (eg, surgical correction of a cyanotic congenital heart defect), relationships between hypoxia and SDH in T1 and T2 were examined separately.

**SaO2 During T1 (Preoperative Period)**

There were 83 infants in group 1 (no persistent hypoxia: average SaO2 = 95%), 41 in group 2 (moderate persistent hypoxia: average SaO2 = 86%), and 16 in group 3 (severe persistent hypoxia: average SaO2 = 78%). There was no significant positive association between SDH and hypoxia, whether persistent or acute (Table 5).

**SaO2 During T2 (Before MRI 2)**

There were 68 infants in group 1 (average SaO2 = 97%), 48 in group 2

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**TABLE 1** Relationship Between SDH on MRI 1 and Mode of Delivery

<table>
<thead>
<tr>
<th>Mode of Delivery</th>
<th>SDH + N (%)</th>
<th>SDH − N (%)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elective cesarean</td>
<td>0 (0)</td>
<td>22 (100)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Emergency cesarean</td>
<td>6 (27)</td>
<td>16 (73)</td>
<td></td>
</tr>
<tr>
<td>Normal vaginal</td>
<td>38 (46)</td>
<td>45 (54)</td>
<td></td>
</tr>
<tr>
<td>Assisted vaginal</td>
<td>8 (50)</td>
<td>8 (50)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>91</td>
<td></td>
</tr>
</tbody>
</table>

* Fisher’s exact test.

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**TABLE 2** Relationship Between SDH on MRI 1 and Apgar Score

<table>
<thead>
<tr>
<th>Apgar Score</th>
<th>SDH + N (%)</th>
<th>SDH − N (%)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 min</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>8</td>
<td>8</td>
<td>.75</td>
</tr>
<tr>
<td>Maximum</td>
<td>10</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Minimum</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>51</td>
<td>87</td>
<td></td>
</tr>
<tr>
<td>5 min</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>9</td>
<td>9</td>
<td>.59</td>
</tr>
<tr>
<td>Maximum</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Minimum</td>
<td>7</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>48</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

* Wilcoxon rank-sum test.
Of the infants analyzed in Table 6, 45 had persistent SDH and 7 had new SDH. Because this was a mixed group, we repeated the analysis in 3 ways. First, we analyzed only the 123 infants who had both MRI scans. The outcome was similar to that of the total group (SDH in 52% without hypoxia versus 31% with hypoxia, $P = .03$). Second, we compared only those with persistent SDH with those who never had SDH ($n = 112$). The outcome was similar (49% vs 31%, $P = .06$). Third, we compared only those with new SDH with those who never had SDH ($n = 73$), and again SDH was more common in infants without hypoxia (15% vs 3%, $P = .09$).

Other Factors

We analyzed T1 data for relationship between cardiac diagnosis and hypoxia, using standard diagnostic groups (2 ventricle, 2 ventricle with aortic arch obstruction, single ventricle, and single ventricle with aortic arch obstruction).$^{22}$ Patients in the 2-ventricle group were more likely to have low SaO$_2$ before surgery. However, there was no relationship between diagnostic group and SDH ($P = .32$).

We also analyzed T1 data for relationship between SDH and the lowest preoperative pH (no relationship, $P = .99$) or the highest preoperative lactate (no relationship, $P = .25$).

**DISCUSSION**

This study is the largest published series describing the prevalence of SDH on MRI scans in newborns. We have shown that SDH was present in 36% of infants during the early neonatal period and present in only 4% at 2 to 3 months of age and that SDH was strongly associated with vaginal rather than cesarean delivery but was not associated with Apgar score at birth or acute or chronic postnatal hypoxia. The rate of asymptomatic neonatal SDH in published literature ranges from 8% to 45%.$^{17–19,21}$ One previous study of SDH in a much smaller cohort of newborns with congenital heart disease identified preoperative SDH in 11 out of 21 (52%).$^{20}$ It is usually suggested that these SDHs originate from tearing of the dura or bridging veins by molding of the cranium during birth.$^{17–19,23–25}$ Others suggest that they originate from “intradural bleeding or congestion of the abundant venous sinuses” rather than from torn bridging veins.$^{14}$

The location of the SDH seen in our cohort was diverse. Previous literature found that asymptomatic neonatal SDHs are usually below the tentorium, or low and posterior when above it. Holden et al$^{17}$ described 4:3 along the falx and 1 at the tentorial notch. Whitby et al$^{18}$ described 9:7 cerebellar, 1 parietal, and 1 multiple (parietal, occipital, and cerebellar). Looney et al$^{19}$ described 16, all infratentorial or low in the occipital or temporal areas. Rooks et al$^{21}$ described 46 infants with supratentorial SDH, mostly in the posterior hemispheric fissure, in the posterior occipital, and over the tentorium. Twenty of the 46 (43%) also had infratentorial SDH. In a study of neonates with congenital heart disease, 11 bled along the inferior surface of the tentorium, and 6 out of 11 also had small supratentorial SDH “along the tentorium and falx or more laterally.”$^{20}$ Although we confirmed that many SDHs are infratentorial and posterior interhemispheric, we also found that many supratentorial SDHs are parietal or temporoparietal.

We identified new SDHs in 12 out of 138 infants after cardiac surgery. New SDHs have been described previously in a minority of patients after neonatal cardiac surgery.$^{20,23,24}$ Complex multivariable analyses of perioperative factors that may be associated with brain

### Table 3: Presence and Number of SDH by Age

<table>
<thead>
<tr>
<th>MRI Scan</th>
<th>MRI n</th>
<th>SDH + n (%)</th>
<th>Age at Time of Positive Scan, d</th>
<th>Loci of SDH, n</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mean</td>
<td>Median</td>
</tr>
<tr>
<td>1</td>
<td>143</td>
<td>52 (36%)</td>
<td>7.2</td>
<td>6.5</td>
</tr>
<tr>
<td>2</td>
<td>138</td>
<td>59 (43%)</td>
<td>18.2</td>
<td>15.0</td>
</tr>
<tr>
<td>3</td>
<td>119</td>
<td>5 (4%)</td>
<td>53.0</td>
<td>49.0</td>
</tr>
</tbody>
</table>

**FIGURE 1**

Two midline sagittal T1-weighted MRI scans from the same patient. A, MRI 1. B, MRI 2 (shows a small new SDH lying below the tentorium in the midline.)
injury have been published, but SDH is either excluded from analysis or regarded as a lesser manifestation of injury. None have singled out SDH for separate analysis. However, typical hypoxic–ischemic watershed patterns of brain injury are not usually seen.

One article that included SDH found no relationship between sustained perioperative cerebral desaturation and any form of brain injury, despite many infants having >4 hours with regional cerebral SaO2 <45%.

Most SDHs had completely resolved by the final MRI scan at 2 to 3 months of age, but some posterior fossa SDHs persisted. This has not previously been described. Only 2 previous studies have followed the natural history of asymptomatic neonatal SDH over time. Whitby

<table>
<thead>
<tr>
<th>Day</th>
<th>MRI</th>
<th>Description When Last Seen, Depth in mm</th>
<th>Resolution (d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>29</td>
<td>2</td>
<td>PCF bilateral 3, right subtemporal 2, smaller</td>
<td>Unknown (last scan)</td>
</tr>
<tr>
<td>30</td>
<td>2</td>
<td>PCF right tent leaf 9 × 11 × 2, smaller</td>
<td>MRI 3 (57)</td>
</tr>
<tr>
<td>34</td>
<td>2</td>
<td>Right posterior temporal, almost completely resolved</td>
<td>Unknown (last scan)</td>
</tr>
<tr>
<td>35</td>
<td>3</td>
<td>PCF midline, very small</td>
<td>Unknown (last scan)</td>
</tr>
<tr>
<td>35</td>
<td>2</td>
<td>PCF left and midline, smaller 1–2</td>
<td>MRI 3 (69)</td>
</tr>
<tr>
<td>37</td>
<td>2</td>
<td>PCF midline/right, posterior parietal, smaller</td>
<td>MRI 3 (83)</td>
</tr>
<tr>
<td>49</td>
<td>3</td>
<td>PCF midline 6, right cerebellar convexity 3–4</td>
<td>MRI 4 (65)</td>
</tr>
<tr>
<td>90</td>
<td>3</td>
<td>PCF midline, was 1–2 mm on MRI 1, now smaller</td>
<td>Unknown (last scan)</td>
</tr>
</tbody>
</table>

PCF, posterior cranial fossa.

FIGURE 2
Supratentorial SDH on T1-weighted MRI scans (A and B from 1 child, C and D from another). A, Axial, interhemispheric. B, Coronal, posterior left hemispheric convexity. C, Axial, right temporo-occipital convexity. D, Coronal, same SDH as C and posterior fossa SDH (right and under the cerebellum near the midline).
et al\textsuperscript{18} described complete resolution by 4 weeks of age in 9 out of 9 newborns, and Rooks et al\textsuperscript{21} reported resolution in 15 out of 16 by 1 month. Interpretation of our observations must take into account the fact that all patients in our cohort had congenital heart disease, cardiac surgery (with heparinization), and intensive care, and some acquired new SDH. One might expect the same factors that induce new SDH to delay the resolution of neonatal SDH present before surgery. Despite this, by MRI 3 SDH had resolved in 93% who had SDH on MRI 1 and in 90% who had SDH first noted on MRI 2.

Our study is the first to examine the relationship between hypoxia and SDH in a cohort of living infants exposed to significant and prolonged hypoxia from birth and to incorporate its own controls (infants of the same age, needing a similar intensity of medical and surgical intervention but without significant hypoxia). The very nature of the “unified hypothesis” means that it cannot be tested in a randomized controlled trial in living infants. The wide variety of medical conditions associated with fatal hypoxia makes it difficult for postmortem studies accurately to reflect the population diagnosed with AHT. Therefore, it is important to find ways to examine this hypothesis in appropriate cohorts of living infants.

We did not demonstrate a relationship between hypoxia and SDH in newborn infants with congenital heart disease. One could speculate that such a relationship existed, but our sample size or datapoints were insufficient to detect it. Taken in isolation, the finding that SDH

\begin{figure}[h]
\centering
\includegraphics[width=	extwidth]{figure3.png}
\caption{Infratentorial SDH on T1-weighted MRI scans in 4 different patients. A, Axial, small SDH left cerebellar convexity. B, Axial, larger SDH left cerebellar convexity. C, Sagittal, small SDH below tent and posterior to cerebellum. D, Sagittal, small SDH below tent, off midline.}
\end{figure}
was present in 44% of patients with hypoxia compared with 32% of patients without hypoxia preoperatively (Table 5). This difference was not statistically significant (2-tailed *P* = .21, 1-tailed *P* = .12) could be considered to indicate a potential association. However, the same test postoperatively demonstrated an effect in the opposite direction (SDH present in 33% of patients with hypoxia, compared with 54% without, 2-tailed *P* = .01), and this was the same whether the SDH persisted from birth or was a new postoperative finding. So overall, in this study there was no apparent trend or consistent direction of effect. In addition, although SDH was common, MRI findings of hypoxic–ischemic encephalopathy were not seen in any infants.

The differences between our cohort and infants admitted with suspected AHT are an obvious limitation to our study. We analyzed a cohort of neurologically asymptomatic patients in the hospital from birth who underwent cardiac surgery in a controlled clinical environment. None sustained hypoxic ischemic brain injury, and none had clinical evidence of elevated intracranial pressure. It was therefore not possible to examine several elements of the pathophysiological cascade proposed in the unified hypothesis. However, our study has found that in a controlled environment, hypoxia of the severity and duration experienced by infants needing postnatal cardiac surgery is not associated with subdural hemorrhage.

Our prospective study also has the limitation that it was not specifically designed to examine the relationship between SDH and hypoxia, so data documenting SDH and hypoxia were analyzed in retrospect. Nevertheless, we believe there is merit to our analysis. The MRI technique was thorough, and the number of infants followed with serial scans was high. *SaO*₂ data were frequently and carefully recorded and almost universally accessible. The presence or absence of SDH was recorded by experienced neuroradiologists blinded to the clinical details, and the blinding of investigators accessing *SaO*₂ data minimized any risk of observer bias.

Subject to the limitations described in this article, we have used available data from living children to present what we believe is an uncomplicated and unbiased test of 1 aspect of the unified hypothesis: that in early infancy,
there may be an association between hypoxia and SDH. No evidence to support that aspect was found.

CONCLUSIONS

Asymptomatic SDH is common in newborns with congenital heart disease, at a frequency similar to that of healthy infants without congenital heart disease. Although infratentorial SDH is common, supratentorial SDH is also common and may occur in locations where it is seen in AHT (particularly the interhemispheric fissure and the cerebral convexities). These SDHs are usually small and resolve within the first 2 to 3 months of life, but small, asymptomatic posterior fossa SDH may persist in some infants. The implication of our findings is that hypoxia alone (of the severity seen in infants needing surgery for congenital heart disease) is not sufficient to cause SDH and that persistence of neonatal SDH is unlikely to explain symptomatic SDH presenting in early infancy.

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(Continued from first page)

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