Objectives: Evaluate the risk of severe intraventricular hemorrhage, in the first week of life, in preterm infants undergoing early interhospital transport.

Design: Retrospective cohort study.

Setting: Tertiary neonatal centers of the Trent Perinatal Network in the United Kingdom.

Patients: Preterm infants less than 32 weeks gestation, who were either born within and remained at the tertiary neonatal center (inborn), or were transferred (transported) between centers in the first 72 hours of life.

Interventions: None.

Measurements and Main Results: Multivariable logistic regression models adjusting for key confounders were used to calculate odds ratios for intraventricular hemorrhage with 95% CIs for comparison of inborn and transported infants. Cranial ultrasound findings on day 7 of life. Secondary analyses were performed for antenatal steroid course and gestational age subgroups. A total of 1,047 preterm infants were included in the main analysis. Transported infants (n = 391) had a significantly higher risk of severe (grade III/IV) intraventricular hemorrhage compared with inborns (n = 656) (9.7% vs 5.8%; adjusted odds ratio, 1.69; 95% CI, 1.04–2.76), especially for infants born at less than 28 weeks gestation (adjusted odds ratio, 1.83; 95% CI, 1.03–3.21). Transported infants were less likely to receive a full antenatal steroid course (47.8% vs 64.3%; p < 0.001). A full antenatal steroid course significantly decreased the risk of severe intraventricular hemorrhage irrespective of transport status (odds ratio, 0.33; 95% CI, 0.2–0.55). However, transported infants less than 28 weeks gestation remained significantly more likely to develop a severe intraventricular hemorrhage despite a full antenatal steroid course (adjusted odds ratio, 2.84; 95% CI, 1.08–7.47).

Conclusions: Preterm infants transported in the first 72 hours of life have an increased risk of early-life severe intraventricular hemorrhage even when maternal antenatal steroids are given. The additional burden of postnatal transport could be an important component in the pathway to severe intraventricular hemorrhage. As timely in-utero transfer is not always possible, we need to focus research on improving the transport pathway to reduce this additional risk. (Pediatr Crit Care Med 2019; 20:638–644)

Key Words: cerebral intraventricular hemorrhage; infant, preterm; newborn; perinatal care; transport

Centralization of neonatal intensive care has improved preterm mortality; however, morbidity such as neurodisability remains unchanged (1). Preterm infants have an increased risk of intraventricular hemorrhage (IVH) (2, 3); although the etiology is multifactorial, the principle cause is due to the fragility of the germinal matrix and fluctuations in cerebral blood flow (4). However, this risk can be significantly reduced by administration of antenatal steroids (ANS) prior to delivery (5). Severe IVH (grades III and IV) is associated with increased mortality and an estimated 70% of survivors develop cerebral palsy or cognitive impairment (6). This has a significant impact not only on quality of life but on society with an estimated lifetime cost per child with cerebral palsy of $1.3 million (£1 million) (7, 8). Furthermore, infants with mild IVH have lower developmental scores compared with those with no IVH (6, 9).
The majority of IVHs start within the first hours and days of life (3), coincident with the period when interhospital transport frequently occurs (10) and, in many instances, grade 1 or 2 hemorrhages extend to the more severe grade (11). This is of particular concern as one in six preterm infants less than 32 weeks gestation (~1,400 infants) born in the United Kingdom are transported in the first 72 hours of life (10) and up to one in five are transported in Canada (12). The EPICure 2 study found only 7% of extremely preterm infants transported on the first day of life, survived without significant morbidity, lower than those born and cared for in level 2 (17%) and level 3 units (15%) (11). Many historical cohort studies have reported an increased risk of severe IVH with postnatal interhospital transport (8, 13–15), but others have not (16, 17). These have a number of limitations including exclusion of high-risk patients and omission of important confounders including the role of ANS as a potentially neuroprotective agent and proxy of good quality antenatal care. More importantly, none account for the lack of differentiation between early perinatal (in the first week) and later brain injury; the latter could be attributed to other risk factors associated with longer-term neonatal care.

A recent Canadian study, of almost 3,000 infants less than 29 weeks, found outborn (transported) infants were more likely to have a poor neurodevelopmental outcome at 2 years old (12). The authors postulated that the outborn infants were sicker initially but not 12 hours after transfer and suggested the illness severity after delivery and during transport may have a major impact on these outcomes. However, no data were presented on the day of transfer or risk of severe IVH in the first week of life, which could better reflect early perinatal risks including interhospital transport.

Understanding the prevalence of IVH and the associated perinatal factors in the first week of life could provide useful data for studies aimed at reducing this during the period of greatest risk. Our primary aim was to evaluate the relationship between neonatal transport, early severe IVH, and ANS administration in preterm infants born less than 32 weeks gestation who were transferred within the first 72 hours of life.

**MATERIALS AND METHODS**

**Study Design and Participants**

This retrospective cohort study used prospectively collected anonymized clinical data from a validated online national U.K. database (18), BadgerNet (Clevermed, Edinburgh, United Kingdom), between 2007 (the start of the database in this network) and 2016 as well as local clinical records where appropriate. Data were collected on all preterm infants born less than 32 weeks gestational age (GA), who were either born in (inborn) or transferred into (from regional centers of any care level) or between one of the two Nottingham University Hospitals (NUHs) tertiary neonatal ICUs (NICU). These are the two regional tertiary referral centers for the U.K. Trent Perinatal Network.

BadgerNet creates a single record of care for every newborn admitted to the NICU and includes information on obstetric care and subsequent postnatal management. In order to assess IVH potentially related to transport within 72 hours of birth, we included infants who had a cranial ultrasound scan (CrUSS) on day 7 (± 1) after birth as per the standard tertiary center protocol; those who died or were transported out of NUH before this scan were excluded. Ethical approval was given by the School of Medicine Ethics Committee, University of Nottingham.

### Outcome

The primary outcome was CrUSS findings taken on day 7 (±1) of life. All CrUSs used a standardized protocol with a predefined series of anatomical views obtained. These were then evaluated by a consultant pediatric neuroradiologist or neonatal consultant and graded using the classification by Papile et al (19). Infants were categorized as having no IVH or any grade of IVH, which was further divided into no or mild IVH (grade 1 or 2) and severe IVH (grade 3 or 4).

### Statistical Analysis

Infants were grouped according to transport status: inborn versus transported. The two groups were further divided into less than 28 week and 28–32 week GA subgroups. Initial assessment of the association between transport, within 72 hours of birth, and IVH outcome was conducted using a chi-square test. Associations between demographic and clinical variables with transport and with IVH were assessed using chi-square tests for categorical data and Mann-Whitney U test for nonnormally distributed continuous data.

Multivariable logistic regression was used to calculate the adjusted odds ratio (aOR) for the association between transport and IVH, controlling for confounding factors. A priori confounders (gender, gestation, and birth weight) were included in the logistic model in addition to any variable that had a statistically significant association with both exposure (transport) and outcome (IVH) at the 5% level. Variables that were not statistically significant on univariate testing were individually added back into the model and included as potential confounders if there was a change in the aOR for IVH in either direction by greater than or equal to 10%. Confounding factors evaluated for inclusion in the regression model were mode of delivery, intrauterine growth restriction (based on serial fetal ultrasound), maternal infection risk (maternal IV antibiotics or sepsis, prolonged rupture of membranes > 18 hr, maternal pyrexia, and Group B Streptococcus), antepartum hemorrhage, maternal recreational drug use, intubation at birth, surfactant administration, chest compressions at birth, delivery room adrenaline, Apgar scores at 1 and 5 minutes, and NICU inotrope treatment. Similar models were created with the main outcome severe IVH only, with the baseline group including those with no or mild IVH.

To evaluate whether ANS administration modified the effect of transport within 72 hours of birth on IVH, infants were stratified by whether they had received no or an incomplete course of ANS or a complete course of ANS at least 24 hours prior to delivery. Logistic multivariable models were also...
used to evaluate the association between transportation and IVH or severe IVH adjusting for confounding factors, depending on ANS status. All statistical analyses were performed using Stata SE (Version 14; StataCorp, College Station, TX).

**RESULTS**

A total of 1,114 preterm infants met the inclusion criteria (inborn, n = 713 and transported, n = 401; Fig. 1). Sixty-five infants died prior to day 7 CrUSS, one infant had a missing CrUSS, and one infant was transferred outside of the network before day 7, leaving 1,047 for analysis. Extreme prematurity and respiratory causes were identified as the main factors contributing to the cause of death in infants who died before day 7 in both groups (Table S1, Supplemental Digital Content 1, http://links.lww.com/PCC/A933; Table S2, Supplemental Digital Content 2, http://links.lww.com/PCC/A934; and Table S3, Supplemental Digital Content 3, http://links.lww.com/PCC/A935). Both groups had similar median birth weights and GA at birth (Table 1). Overall, 51.9% (n = 203) of transfers occurred between level 3 units (n = 71 < 28 wk GA). Transported infants were more likely to be male, have a greater prevalence of maternal infection risk factors, and were more likely to be intubated or receive surfactant compared with inborn infants. Over the period of the study, the prevalence of mild and severe IVH did not change (data not shown).

Irrespective of transport group, infants less than 28 weeks GA were significantly more likely to develop any IVH (58.6% vs 25.4%; p < 0.001) and severe IVH (12% vs 3%; p < 0.001) compared with those born 28–32 weeks GA. The prevalence of any grade IVH was similar between transported (n = 165; 42.2%) and inborn infants (n = 264; 40.1%) and the OR was not statistically significant before (OR, 1.08; 95% CI, 0.84–1.4; p = 0.51) or following adjustment (aOR, 0.99; 95% CI, 0.75–1.35; p = 0.96). Overall, when including transfer status, transported infants were more likely to develop severe IVH compared with inborns (5.8%) (Table 3). Furthermore, of the 38 transported infants with severe IVH, 35 occurred in those transferred in the first 48 hours of life with 27 of these infants less than 28 weeks GA (Table S4, Supplemental Digital Content 4, http://links.lww.com/PCC/A936).

For the assessment of ANS, 19 infants were excluded as they had missing data, leaving 1,028 for analysis. Of these, 610 infants (58.2%) received a full course of ANS (Fig. S1, Supplemental Digital Content 5, http://links.lww.com/PCC/A937; legend, Supplemental Digital Content 7, http://links.lww.com/PCC/A939), 228 (37.4%) were subsequently diagnosed with an IVH with 26 (4.3%) developing severe IVH. Inborn infants were significantly more likely to receive a full course of ANS compared with transported infants (p < 0.001) (Table 1). Irrespective of transport status, a full course of ANS was associated with a 33% decrease in odds of any IVH (OR, 0.67; 95% CI, 0.52–0.87; p = 0.003) and 67% reduction of severe IVH (OR, 0.33; 95% CI, 0.2–0.55; p < 0.001). Overall, transported infants who received no or an incomplete course of ANS were at increased odds of developing any IVH (OR, 1.47; 95% CI, 1.10–2.17; p < 0.05) although this was not statistically significant following multivariable adjustment (aOR, 1.37; 95% CI, 0.88–2.14).

Subgroup analysis of infants less than 28 weeks GA showed a complete course of ANS was associated with significantly reduced odds of both any grade IVH by 41% (OR, 0.59; 95% CI, 0.40–0.86; p < 0.001) and severe IVH by 63% (OR, 0.37; 95% CI, 0.21–0.66; p < 0.001) irrespective of transport status. However, inclusion of ANS in the multivariable regression model demonstrated transported infants less than 28 weeks remained significantly more likely to have severe IVH despite a full course of ANS (Fig. 3 and Table 4). Transferred infants 28–32 weeks GA, irrespective of maternal ANS treatment, had a greater proportion of severe IVH overall (2.3% vs 1.2%), but this was not statistically significant (Table 4; Fig. S1, Supplemental Digital Content 5, http://links.lww.com/PCC/A937; and Fig. S2, Supplemental Digital Content 6, http://links.lww.com/PCC/A938, respectively [legend, Supplemental Digital Content 7, http://links.lww.com/PCC/A939]).
### TABLE 1. Comparison of Demographic and Clinical Variables Between Inborn and Transported Infants

| Variables                      | Inborn, \( n = 656^a \) | Transported, \( n = 391^a \) | Missing, \( n \) (%) | \( p^b \) |
|-------------------------------|--------------------------|-----------------------------|----------------------|--------|
| Gestation                     | 28.4 (26.4–29.9)         | 28.1 (26.4–29.7)            | 0                    | 0.72   |
| Birth weight                  | 1,050 (810–1,285)        | 1,090 (860–1,300)           | 28 (2.7)             | 0.07   |
| Male                          | 336 (51.2)               | 226 (57.8)                  | 0                    | 0.03   |
| Intrauterine growth restriction| 81 (12.3)                | 39 (10)                     | 6 (0.6)              | 0.24   |
| Maternal infection risk       | 78 (11.9)                | 72 (18.4)                   | 10 (1)               | 0.003  |
| Antepartum hemorrhage         | 116 (17.7)               | 45 (11.5)                   | 6 (0.6)              | 0.008  |
| Antenatal steroid             |                          |                             |                      |        |
| None/incomplete               | 222 (33.8)               | 196 (50.1)                  | 19 (1.8)             | <0.001 |
| Complete                      | 423 (64.5)               | 187 (47.8)                  |                      |        |
| Mode of delivery              |                          |                             |                      |        |
| Normal vaginal delivery       | 306 (47.0)               | 194 (49.6)                  | 10 (1)               | 0.16   |
| Emergency C-S                 | 293 (45.0)               | 175 (44.8)                  |                      |        |
| Elective C-S                  | 36 (5.5)                 | 11 (2.8)                    |                      |        |
| Instrumental                  | 16 (2.5)                 | 8 (2.0)                     |                      |        |
| Intubated first 72 hr         | 537 (81.9)               | 341 (87.2)                  | 1 (0.01)             | 0.01   |
| Surfactant                    | 535 (81.6)               | 342 (87.5)                  | 0                    | 0.009  |
| Chest compressions            | 36 (5.5)                 | 26 (6.6)                    | 0                    | 0.43   |
| Adrenaline                    | 10 (1.5)                 | 4 (1)                       | 0                    | 0.5    |
| Apgar 1 min                   | 6 (4–8)                  | 6 (4–8)                     | 148 (14.4)           | 0.36   |
| Apgar 5 min                   | 9 (7–9)                  | 8 (7–9)                     | 161 (15.7)           | 0.16   |
| Intropes                      | 118 (18)                 | 113 (29)                    | 0                    | <0.001 |
| Days to first extubation\(c\) | 2 (1–4)                  | 3 (1–6)                     | 49 (4.7)             | <0.001 |
| Mortality after day 7         | 39 (5.9)                 | 27 (6.9)                    | 0                    | 0.52   |

\( ^a \)Data are \( n \) (%) or median (interquartile range).

\( ^b \)Categorical data analyzed using \( \chi^2 \) test; nonnormally distributed continuous data analyzed using Mann-Whitney \( U \) test.

\( ^c \)Extubation without consequent re-intubation within the following 72 hr.

### TABLE 2. Unadjusted and Adjusted Odds Ratios to Show the Association of Transportation With No/Mild Intraventricular Hemorrhage and Severe Intraventricular Hemorrhage for All Infants and by Gestational Subgroups

| Gestational Age Group | Outcome Comparison | OR (95% CI) | Adjusted OR (95% CI)\(^a\) |
|-----------------------|--------------------|-------------|---------------------------|
| All infants (\( n = 1,047 \)) | No/mild IVH vs severe IVH | 1.75 (1.09–2.80) | 1.69 (1.04–2.76) |
| < 28 wk (\( n = 492 \)) | No/mild IVH vs severe IVH | 1.63 (0.94–2.82) | 1.83 (1.03–3.21) |
| 28–32 wk (\( n = 555 \)) | No/mild IVH vs severe IVH | 2.02 (0.77–5.35) | 1.66 (0.61–4.52) |

\( ^a \)Adjusted for gender, gestation, birth weight, mode of delivery, intrauterine growth restriction, maternal infection, antepartum hemorrhage, maternal recreational drug use, intubation at birth, surfactant administration, chest compressions at birth, delivery room adrenaline, Apgar scores 1 and 5, and neonatal ICU inotropes.

Mild, grade 1 and 2; severe, grade 3 and 4.

Bold indicates statistical significance \( p < 0.05 \).
DISCUSSION

This study aimed to evaluate the association between early interhospital transport of preterm infants and severe IVH in the first week of life. We found transported infants, particularly those less than 28 weeks GA, were significantly more likely to develop severe IVH compared with inborns and this association remained following adjustment for major confounding factors. ANSs reduce the risk of IVH (5) but previous preterm transport studies have not been able to include these in their modeling (8, 14, 15). This UK regional network transport study is one of the largest to date and demonstrates a 67% reduction in severe IVH for all preterm infants following a full course of ANS as expected (19). However, infants less than 28 weeks GA undergoing early interhospital transport were still more likely to develop a severe IVH despite a full course of ANS.

This is the first study to demonstrate the association between early transport of preterm infants and the risk of significant brain injury in the first week of life. Our results are consistent with previous studies (2, 3, 8) demonstrating an increased risk of severe IVH at discharge in preterm infants transported early in life, however, none of these studies prove causation. The perinatal period is a high-risk window for the development of IVH, and our study raises the possibility that the postnatal transport pathway itself may contribute to the increased prevalence observed as we were able to adjust for many of the known obstetric and early neonatal risks. Our findings are similar to a recent Canadian study of tertiary center inborn and outborn infants, less than 29 weeks gestation, demonstrating significantly greater mortality, severe IVH and poorer long-term neurodevelopmental outcomes in outborn infants (12). Their study also controlled for a number of perinatal factors but did not report CrUSS findings in the first week of life, which may explain why the prevalence of severe IVH was higher than we report in the first week of life. Additional explanations for the difference could include later neonatal factors, such as sepsis, impacting on the progression of mild IVH into more severe IVH or the transport distances between Canadian centers are far greater than in the United Kingdom resulting in longer exposure to the noxious effects of transport including air transfer (20).

The mechanism for the association between IVH and transportation is not yet fully understood but is likely to be multifactorial due to suboptimal ventilation (8), temperature instability (21), and the ambulance environment. The preterm infant is exposed to many noxious agents including noise, handling, and vibration, which increase discomfort (22). Excess noise adversely impacts cardiorespiratory stability and significantly decreases cerebral oxygen saturations relative to baseline which could contribute to adverse neurologic outcomes through resultant changes in cerebral vasculature and blood flow (4, 23). Vibration is known to result in cerebral capillary wall thickening, constriction, and destruction as well as induce neuronal injury in animal models (24, 25). During neonatal ambulance transfer, the newborn’s head is exposed to excessive vibration far in excess of that deemed safe and known to cause illness in well adults (26). Combined with our data,
In this study, the higher prevalence of severe IVH in transported infants less than 28 weeks GA (4.9% vs 2.7%) for no or an incomplete ANS course lacked statistical significance. This could be as a consequence of the smaller overall numbers and a lower event rate of severe IVH in this group compared with those with a full ANS course.

This study has several strengths compared with previous studies (8, 13–15). Our study included only infants who were less than 32 weeks gestation, transferred within the first 72 hours and had a standardized day 7 CrUSS. These infants are those most at risk of IVH as the fragile germinal matrix is most prominent in this GA group (4). Using early CrUSS for outcome analysis allows for the potential inflammatory process associated with the transportation process to evolve but minimizes the influence of exposure to other postnatal events with later brain injury. Multivariate logistic regression analyses allowed adjustment for major risk factors for IVH and any group imbalance. However, we acknowledge some residual confounding effect may remain due to variable data entry error or imprecision, such as inotrope use to represent hypotension rather than actual values.

The main limitation of studies comparing inborn to transported infants is selection bias, as critically ill infants who are not stable enough to survive transport are often excluded. Our study highlights this, as inborn infants who died before day 7 were more likely to die within the first 2 days of life than transported infants. These infants were extremely premature, died shortly after delivery, and if delivered in a nontertiary center may not have survived transportation. We aimed to minimize this bias by excluding infants who died in the first week of life as many died from causes related to extreme prematurity and respiratory conditions rather than IVH. However, we cannot exclude all bias due to the increased level of care offered in tertiary centers as inborn infants, who would otherwise be too unstable for transfer if born elsewhere, have an increased chance of survival and are more likely to develop severe IVH (1) although both groups analyzed were well matched for gestation and birth weight.

A further limitation of this study was the exact timing of occurrence of IVH could not be established, although the inclusion of all infants who survived to day 7 CrUSS decreased the chance of selection bias. A prospective study could obtain a pre-transfer CrUSS to aid with interpretation in this setting. Although we used a standardized CrUSS protocol, inter-assessor interpretation could introduce differences in grading of IVH. Pragmatically, this is what happens in clinical practice, but we did try to mitigate this by subgrouping into mild (grade 1 or 2) or severe IVH (grade 3 or 4).

The limitations of retrospective cohort studies also make it difficult to account for evolving practices over the timeframe of the study. For example, the gradual introduction of magnesium sulphate, known to reduce the prevalence of cerebral palsy (27), could not be assessed although this is unlikely to affect the prevalence of early IVH.

**CONCLUSIONS**

Our U.K. Trent perinatal network study highlights that, despite the increased survival observed with centralized neonatal care (28, 29), the early postnatal transport of extremely preterm infants is associated with an increased risk of severe IVH in the first week of life. This risk is not completely mitigated by the known neuroprotective effects of ANS, although receiving a full course of ANS was beneficial over having either no or an incomplete course. Women presenting with threatened preterm delivery should be given prompt ANS (30) and ideally transferred in-utero to an appropriate center, a measure that could be used as a quality benchmark for perinatal network delivery of care.

With the centralization of neonatal intensive care, we have seen investment in postnatal transport services. However, the in-utero transfer process remains a time-consuming process for healthcare staff potentially resulting in missed transfer opportunities (31). Development of a coordinated, in-utero transfer service, with both obstetric and neonatal services,
could result in not only better service provision and allocation of facilities based on clinical needs but could help reduce adverse outcomes associated with postnatal transfer. However, in-utero transfer is not always achievable, therefore the associated risks of postnatal transfer need to be explored and addressed where possible to improve the comfort of the infant and minimize any risk of neurologic insult. This could include reducing noxious environmental stimuli such as noise and vibration, improving monitoring and ventilation as well as considering the timing of postnatal transfer with the ultimate goal to reduce the significant long-term risk of neurodisability.

ACKNOWLEDGMENTS
We are grateful to Jim Thornton for reviewing this article.

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