(rSO_2) during hypothermia after birth asphyxia. Contrary to earlier reports,[2–4] rSO_2 was not suitable to study the effects of additional neuroprotective therapies. The authors postulated that the late timing of rSO_2 measurement contributed to this finding and that hypothermia could have equally slowed down cerebral metabolism (less O_2 utilization) in infants with and without reperfusion injury. We may have an alternative explanation for the divergent results. The range of rSO_2s during hypothermia in asphyxiated infants with adverse outcome measured with the small adult sensor of Somanetics (Troy, MI) is between 82% and 95%.[2–4] The highest value of rSO_2 is limited at 95% (set by manufacturer). Since the neonatal sensor of Somanetics read rSO_2 values 10% higher on average compared to the small adult Somasensor,[5] this range has been compressed to between 92% and 95%. The authors do not mention the actual numbers of rSO_2 but it is possible that the use of the neonatal sensor may have contributed to the divergent results concerning efficacy of rSO_2 as a prognostic biomarker.

Author Response: Renee A. Shellhaas, John Barks, Ann Arbor, MI: Our Dutch colleagues highlighted an important point: the rSO_2 value depends on the sensor employed. Their data[5] were published after ours went to press, but we cited a personal communication with Drs. Lemmers and Toet in our Discussion. A strength of our study was that we used a clinically available device (Invos 5100C, Somanetics Corporation). We also utilized the US Food and Drug Administration–approved neonatal cerebral and somatic sensors, which is the equipment most likely to be used clinically in an American NICU and in future clinical research. However, published articles do not often list which sensor was utilized. Future near-infrared spectroscopy research must consistently report the exact equipment and sensor type.

In our study,[1] the mean cerebral rSO_2 immediately before and during rewarming was 81.9 ± 3.39 (somatic rSO_2 was 71.8 ± 7.15). The Dutch data demonstrated divergent cerebral rSO_2 and fractional cerebral tissue oxygen extraction values in the first 36 hours of life for those with adverse vs favorable outcome.[4] However, the absolute differences decreased thereafter, with no significant difference at 60 hours, the timeframe most comparable to our study. Therefore, the combination of their and our data suggests that cerebral rSO_2 on the third day of life may not differentiate those destined for favorable or adverse outcomes.

1. Shellhaas RA, Thelen BJ, Jayapalli R, et al. Limited short-term prognostic utility of cerebral NIRS during neonatal therapeutic hypothermia. Neurology 2013;81:249–255.
2. Toet MC, Lemmers PMA, van Schelven LJ, van Bel F. Cerebral oxygenation and electrical activity after birth asphyxia: their relation to outcome. Pediatrics 2006;117:333–339.
3. Ancora G, Maranella E, Grandi S, et al. Early predictors of short term neurodevelopmental outcome in asphyxiated cooled infants: a combined brain amplitude integrated electroencephalography and near infrared spectroscopy. Brain Dev 2013;35:26–31.
4. Lemmers PMA, Zwanenburg RJ, Benders MJ, et al. Cerebral oxygenation and brain activity after perinatal asphyxia: does hypothermia change their prognostic value? Pediatr Res 2013;74:180–185.
5. Dix LM, van Bel F, Baerts W, Lemmers PM. Comparing near-infrared spectroscopy devices and their sensors for monitoring cerebral oxygen saturation in the neonate. Pediatr Res 2013;74:557–563.

CORRECTION

Summary of evidence-based guideline update: Prevention of stroke in nonvalvular atrial fibrillation: Report of the Guideline Development Subcommittee of the American Academy of Neurology

In the article "Summary of evidence-based guideline update: Prevention of stroke in nonvalvular atrial fibrillation: Report of the Guideline Development Subcommittee of the American Academy of Neurology" by A. Culebras et al. (*Neurology*® 2014;82:716–724), there was an error on page 720 of the print article and on pages 24 and 58 of the full-length article (see data supplement e-1) regarding the recommended dosage for apixaban. The text should read: "Apixaban 5 mg twice daily (if serum creatinine <1.5 mg/dL), or 2.5 mg twice daily if *any 2* of the following criteria are present: serum creatinine >1.5 mg/dL and <2.5 mg/dL; body weight ≤60 kg; age ≥80 years." The authors regret the error.

Author disclosures are available upon request (journal@neurology.org).