Pediatric RESEARCH

COMMENT

Editorial Commentary: Inter-observer concordance in applying the Sarnat Grading Scale of neonatal encephalopathy to mildly preterm infants

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Validation is essential to evidence-based science and medicine. The study by Pavageau et al. makes a laudable attempt to demonstrate concordance between neonatologists in applying a mild modification of the Sarnat Grading Scale of neonatal encephalopathy in term neonates to 102 mildly preterm infants of 32–36 weeks GA, using strictly clinical criteria.[1] The original 1976 publication, before any types of neuroimaging or of EEG monitoring were available, provided the basis for the modifications proposed by Pavageau et al.[2] They found good agreement in most criteria except muscle tone and Moro reflex. The authors are all neonatologists; no paediatric neurologists contributed to the study.

Techniques of neurological examination in neonates are important so that standardised manoeuvres are used by all observers to establish criteria of decreased muscle tone and of eliciting the Moro reflex. This technique is not described in Methods. For a study such as that of Pavageau et al.[1] that compares the results of several examiners, uniformity of examination technique is primordial, and perhaps variation in the methods of assessing tone and of eliciting the Moro reflex might account for the lack of agreement between examiners in these items.

The original publication of 1976 was intended as a relatively small prospective pilot study that utilized clinical neurological examination, the only special supplementary technique being a standard EEG by paper recording as the standard of practice of that time. This study has endured over more than four decades because its design was the first to objectively address a common neonatal neurological problem, because its application was easily utilized by any physician examining neonates without special research tools, and because confirmation of the study by other unrelated investigators confirmed its validity. Though most of the infants in the original study had perinatal hypoxic/ischaemic encephalopathy or intrapartum asphyxia, the title of the article referred simply to *neonatal encephalopathy following fetal distress*, specifically avoiding the more specific aetiological designation of hypoxia or ischaemia because the grading scale potentially could be applied to other neonatal encephalopathies as well, of metabolic, genetic, toxic or infectious origin.

Preterm neonates usually have decreased muscle tone that might be considered hypotonia in the term infant, a well-recognized physical finding by neonatologists and other paediatricians caring for newborns, as well as by paediatric neurologists. The present manuscript by Pavageau et al. is a clinically descriptive paper and provides a comparison between different observers with similar expertise, but does not discuss the developmental pathophysiology of muscle tone and other neuromuscular functions in terms of central or peripheral nervous system maturation. I attributed the changes in muscle tone with maturation early in life to an extrapolation from animal experiments, particularly the work of Lawrence and Kuypers in the neonatal monkey in the late 1960s.[3] Postures, both normal and abnormal, often are secondary to imbalances between flexor and extensor muscle tone, unrelated to strength and assuming the absence of a primary myopathy or peripheral neuropathy. Posture and muscle tone in the neonate are closely related.

Descending cerebrospinal pathways can be divided into three groups:[4,5] the medial and lateral subcorticospinal pathways, which are groups of mostly small bulbospinal tracts such as the vestibulospinal, olivospinal, rubrospinal and reticulospinal, and the large but later maturing corticospinal tract. Tract maturation from the neuroanatomical perspective is the arborization of terminal axonal contacts, synaptogenesis and myelination, but also includes maturation of the distant neurons of origin that must synthesize and transport within long axons neurosecretory chemical transmitters. Functionally, the medial subcorticospinal and corticospinal pathways are antagonistic: the medial subcorticospinal subserves proximal extension and distal flexion; the corticospinal tract subserves proximal flexion and distal extension. Brainstem structures from which the subcorticospinal pathways originate are more resistant to hypoxic or noxious influences and the balanced antagonism is lost. Furthermore, the corticospinal tract is an inhibitory pathway that selectively sculpts muscle tone and posture by selectively suppressing massive excitation. This exemplifies why knowledge of pathophysiological mechanisms is so much more rewarding than simple clinical observation alone, particularly in the immature nervous system.

In sum, the paper by Pavageau et al. is a good start to find clinical criteria for determining encephalopathy in mildly preterm neonates, but future studies that include strict criteria for the evaluation technique for each item might contribute to an even better inter-examiner concordance. Discussion of the neuroanatomical and neurophysiological basis for each criterion will provide a greater and in-depth understanding of why abnormalities occur, and of their neuroanatomical localization.

ADDITIONAL INFORMATION

Competing interests: The author declares no competing interests.

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