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Welcoming another new Associate Editor to *The Journal*

— William F. Balistreri, MD

When we began the search last year to replace Alan H. Jobe, MD, PhD, as an Associate Editor, it was immediately apparently that he left big shoes to fill. As mentioned in the December 2015 issue of *The Journal*, one of those shoes was filled by Robin Steinhorn, MD, in September. After careful consideration, we have now chosen someone to fill the other shoe, someone who is an expert in her field, highly regarded by her colleagues, and has an interest in journal editing.

We would like to welcome and introduce Raye-Ann deRegnier, MD, as *The Journal's* newest Associate Editor. Dr deRegnier began handling new manuscripts in mid-January.

Dr deRegnier is an Associate Professor of Pediatrics at Northwestern University's Feinberg School of Medicine and practices neonatology at the Ann & Robert H. Lurie Children's Hospital of Chicago and Northwestern Memorial Hospital in Chicago. She is a member of the American Pediatric Society and serves on the American Board of Pediatrics Sub-board of Neonatal Perinatal Medicine. She served as a Neonatal Fellowship Director for 11 years and is a member of the Perinatal Advisory Committee for the Midwest Conference on Perinatal Research.

Dr deRegnier's clinical and academic interests have been focused primarily on the cognitive and motor outcomes of neonatal and cardiac intensive care graduates and the effects of growth and nutrition and critical illness on development. She also has been clinically interested in systems of perinatal care and perinatal regionalization. She is newly married and has one adult daughter. Dr deRegnier believes that *The Journal* is currently the best journal for neonatology and looks forward to working with *The Journal's* leadership to continue to attract the highest-quality research.

The first step in preventing suicide is to ask

— Catherine Forster, MD

Adolescent suicide is a significant public health problem, representing the second leading cause of death in youth 10-21 years of age. Further, a national survey revealed that 8% of 9th to 12th grade students reported attempting suicide within the past year (*MMWR* 2014;63:1-168). Suicide can be preventable, but in order for prevention strategies to be successful, it is imperative to identify high-risk teens. However, opportunities to screen adolescents for risk of suicide within the medical system are frequently missed. Ross et al performed a qualitative-methods study to determine the opinions of medically ill adolescent inpatients regarding suicide risk screening. The majority of the patients (83%) were supportive of nurses screening inpatients for suicide risk. The major salient themes that emerged from those supportive of screening were: the potential of suicide prevention, elevated risk of hospitalized patients, emotional benefit to being asked, provider responsibility, and lack of harm in asking. Despite the concerning prevalence of adolescent suicide, the authors found that 62% of their cohort reported that they never had been asked about suicide in any setting. This finding is concerning, yet one that is relatively easy to change. The report by Ross et al provides data that, overall, adolescents support screening for suicidal thoughts. In order to affect change in the rate of teen suicide, it is critically important to first identify the population at risk.

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Surfactant treatments for RDS improves oxygenation

— Alan H. Jobe, MD, PhD

This title is simply the well-known observation from animal and human studies demonstrating that the major acute effect of surfactant treatments for infants with respiratory distress syndrome (RDS) is a large increase in arterial oxygen tensions. This clinical response results from the surfactant rapidly spreading to the distal lung airspaces to decrease the opening pressures for unexpanded (fluid filled) or collapsed airspaces. The decreased airway opening pressures result in increased functional residual volumes (FRC), increased lung gas volumes at end expiration, and a more uniform distribution of gas throughout the lung. These remarkable functional changes in the RDS lung are difficult to measure clinically, and, for practical purposes, the improved oxygenation is the clinical signal that a surfactant treatment has been effective. The demonstrations of the surfactant treatment responses for RDS were done with intubated animals or infants. Noninvasive approaches to surfactant treatments are being promoted that also depend on oxygenation responses to demonstrate rapid effects. These approaches use different types of fine catheters placed in the proximal airway with or without attempts to maintain continuous positive airway pressure (CPAP). In this issue of *The Journal*, van der Burg et al report on surfactant treatment with a 1- to 3-minute tracheal infusion through an umbilical artery catheter with the infant in the supine position while maintaining CPAP. They monitored the lung gas volumes by electrical impedance tomography. As anticipated, end-expiratory lung volumes increased in dependent and then nondependent lung regions as oxygenation improved. The lung volumes increased relatively homogenously. This report demonstrates again the "magic" of surfactant spreading from the trachea throughout the lungs with spontaneous breathing of the infant, even without changing the position of the infant. In practice, the clinician simply needs to get the surfactant into the proximal airways, and the combination of the surface properties of the surfactant and a spontaneously-breathing infant will finish the job. However, too rapid treatments can block airways and too slow treatments can result in nonhomogeneous distributions of surfactant.

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An uncommon condition may provide insight into a common problem

— Denise M. Goodman, MD, MS

Our understanding of genetic syndromes continues to grow, including those of the large group called RASopathies. These clinical syndromes have some phenotypic overlap and include Noonan syndrome, neurofibromatosis type 1, cardio-facio-cutaneous syndrome, and Costello syndrome. Although the conditions are varied, all of these syndromes are caused by germline mutations in genes related to components of or regulation of the RAS/MAPK pathways. These signal transduction pathways control cell proliferation, differentiation, and survival.

Patients with Costello syndrome have a mutation causing the *HRAS* gene to be permanently activated. *HRAS* is a recognized proto-oncogene also known as transforming protein p21. One hallmark of Costello syndrome is failure to thrive, and this growth failure has heretofore been attributed to feeding difficulties (eg, swallowing problems, weak suck, gastroesophageal reflux). In this context, the findings of Leoni et al in this issue of *The Journal* are intriguing and potentially far-reaching. The investigators compared 11 children with Costello syndrome with 11 age- and sex-matched controls. They found that despite high energy intake, the children with Costello had poor growth, the most striking difference compared with unaffected children was an increased resting energy expenditure.

Although Costello syndrome is uncommon (affecting 1 in 1.29 million children [*Am J Med Genet A* 2012;158A:1083-94]) this cohort provides an opportunity to link critical cellular functions with significant clinical findings. If the alteration in resting energy expenditure is confirmed, this may provide a clue to the metabolic derangements subserving altered cell growth and differentiation. These findings, in turn, may be applicable to a broad range of illnesses and provide insight into the dysregulation of growth typical in both genetic syndromes and tumor proliferation.

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