

22. Opel DJ, Mangione-Smith R, Robinson JD, Heritage J, DeVere V, Salas HS, et al. The influence of provider communication behaviors on parental vaccine acceptance and visit experience. *Am J Public Health* 2015;105:1998-2004.
23. Chamberlain AT, Limaye RJ, O'Leary ST, Frew PM, Brewer SE, Spina CI, et al. Development and acceptability of a video-based vaccine promotion tutorial for obstetric care providers. *Vaccine* 2019;37:2532-6.

50 Years Ago in *THE JOURNAL OF PEDIATRICS*

When the Newborn Remains Blue

Lees MH. Cyanosis of the newborn infant. Recognition and clinical evaluation. *J Pediatr* 1970;3:484-98.

It has been almost 100 years since Lundsgaard et al described that clinical cyanosis is dependent on the absolute concentration of reduced hemoglobin in the blood, as well as on the difference between central and peripheral cyanosis. It was recognized that organs such as the tongue with a high blood flow and a small arteriovenous oxygen difference might not appear cyanotic as readily as organs with a low blood flow and a large arteriovenous oxygen difference, such as skin of cool hands and feet. This was the background of the concept of central and peripheral cyanosis.¹

Fifty years ago, Martin H. Lees published this comprehensive and critical review of cyanosis. He criticized the then-current dogma in medical textbooks that a 5-g reduction of hemoglobin in arterial blood is required before central cyanosis becomes visibly detectable. He argued that if this were the case, then an infant with a total hemoglobin content of 15 g per 100 mL of blood would be visibly cyanotic only at an arterial oxygen saturation of $\leq 67\%$, and pointed out that central cyanosis is detectable by inspection of the tongue and mucous membranes at an arterial saturation of 75%-88% at a 3-g decrease of hemoglobin in arterial blood. He disputed the absolute distinction of central and peripheral cyanosis and noted that newborns with high fetal hemoglobin concentration may need a serious reduction in oxygen tension before central cyanosis is clinically apparent. The article reviews the relationship of cyanosis, oxygen saturation, and PaO₂; discusses the clinical spectrum and how to diagnose central cyanosis; and systematically summarizes 11 major causes of cyanosis in the newborn.

This review is fascinating reading because it provides insight into how our present knowledge in this field is based on meticulous studies by previous colleagues. It reminds us how privileged we are today when we can simply screen all newborn babies for cyanosis with a pulse oximeter. Lees' article 50 years ago still contains valuable clinical information and can still be recommended as a perspective on newborn cyanosis.

Jannicke H. Andresen, MD, PhD

Department of Neonatology
Oslo University Hospital
Oslo, Norway

Ola Didrik Saugstad, MD, PhD

Department of Pediatric Research
University of Oslo
Oslo, Norway

Ann and Robert H. Lurie Children's Hospital of Chicago
Northwestern University Feinberg School of Medicine
Chicago, Illinois

Reference

1. Lundsgaard C, Van Slyke D, Abbott ME. Cyanosis. *Can Med Assoc J* 1923;13:601-4.