ing the importance of a careful history and physical examination in the evaluation of cyanotic newborn infants. Indeed, the diagnostic and therapeutic approach that we outlined makes sense only in circumstances in which the responsible physician is appropriately using the information obtained from the history and physical examination. We also concur that careful documentation and communication of this important information to the neonatologist is essential. In agreement with Dr. Pleasure, we reiterate the words of Sir William Osler, who remarked, "Observe, record, tabulate, communicate. Use your five senses."[1] The ability to observe well through taking a medical history and performing a physical examination and communicating well, may serve as the basis for someone else's advice to the primary care physician, as well as for the primary care physician's own decisions about the care of the patient. The presentation of our practical approach to diagnosis and immediate care of the cyanotic neonate[2] reflects our respect for our colleagues who provide primary care, their knowledge, skills, and dedication to their profession, and serves as evidence for our firm belief that they "can think and act in a very sophisticated manner" in stabilizing and preparing a cyanotic neonate for transfer to a Level III nursery. In fact, their job is "to know just what has to be done and to do it . . ."[3] Therefore, our discussion primarily addresses the appropriate use and interpretation of additional diagnostic and therapeutic maneuvers that may allow confirmation of etiologic diagnoses suggested by the history or physical examination. We hope that our discussion might allow the practicing pediatrician to better understand the rationale for and interpretation of the tests and interventions that are requested by the consulting neonatologist during the period prior to transport of the cyanotic neonate. We would certainly not wish to suggest that the pediatrician practicing in a community hospital should complete all of the recommended studies prior to requesting transfer to an intensive care nursery. On the contrary, we would hope that this approach could provide the framework for an ongoing collaboration between the primary pediatrician and the consulting neonatologist during this critical transitional period.

Similarly, we concur that the electrocardiogram and chest radiograph are frequently valuable in elucidating causes of refractory cyanosis in the newborn. These studies and the additional tests suggested by Dr. Pleasure often do suggest specific diagnoses, but we emphasize the importance of recognizing that two or more pathophysiologic causes underlying cyanosis may coexist in a single newborn infant. We believe that a diagnostic and therapeutic approach based upon a careful assessment of pathophysiology, rather than upon identification of the most easily diagnosed disease, is most likely to allow recognition of these situations.

Finally, we wish to assure Dr. Pleasure that we are indeed both neonatologists. We believe that provision of a pathophysiologic approach to diagnosis of the cyanotic infant, whose diagnosis is frequently elusive and often complex, will provide a common ground for collaboration between neonatologists and cardiologists, thereby preventing the altercations to which she refers.—DAVID K. STEVENSON, MD, *Associate Professor of Pediatrics and Associate Director of Newborn Nurseries, and* WILLIAM E. BENITZ, MD, *Assistant Professor of Pediatrics, Stanford University School of Medicine, Stanford, California.*

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Iron Supplementation in Cyanotic Congenital Heart Disease

To the Editor:

Iron deficiency has been associated with stroke and congestive heart failure in infants with cyanotic congenital heart disease. These patients have greater iron requirements than normal infants because they have 1) higher hemoglobin concentrations; 2) larger blood volumes; 3) lower birth weights; and 4) lower iron stores.[1] Thus, they are at greater risk for outstripping their iron endowment and becoming iron-deficient. Using previously published data on measurement of iron stores, iron losses, and iron endowment at birth, we calculated the total amount of dietary iron required to maintain iron sufficiency when cyanotic infants double their birth weights.[2] These theoretical calculations suggested that iron-supplemented formula could meet the iron needs of cyanotic patients if these patients had enhanced iron absorption.

We, therefore, postulated that infants with cyanotic congenital heart disease fed iron-supplemented formula would have enhanced iron absorption and would be able to maintain normal iron stores. To test this hypothesis, we assessed iron stores and absorption in five infants with cyanotic congenital heart disease (arterial PaO_2 = 41 ± 5 mmHg, hemoglobin = 17.3 ± 1.6 g/dl).

Iron stores were measured in all five infants at 7.9 ± 1.3 months of age. All infants received iron-supplemented formula as their only dietary source of iron. Normal iron stores for age were present in each (ferritin, 78 ± 43 ng/ml; transferrin saturation, 18 ± 9%; mean corpuscular volume, 88 ± 2 fl).

Though it has been known for many years that patients with cyanotic congenital heart disease have increased iron requirements, the best means of sup-

plying dietary iron has not been established. Our theoretical calculations of iron requirements as well as clinical observations in full term acyanotic infants suggest that non-supplemented formulas are inadequate.[3] We have demonstrated adequate iron absorption and iron sufficiency in cyanotic patients fed iron-supplemented formula. These data suggest that iron-supplemented formulas, without additional iron supplements, are an appropriate source of dietary iron for infants with cyanotic congenital heart disease.

We recommend all infants with chronic hypoxemia receive iron-supplemented formula or breast milk with additional prescribed iron.—SAMUEL S. GIDDING, MD, *Division of Cardiology, The Children's Memorial Hospital, Chicago, Illinois, and* AMNON ROSENTHAL, MD, *Director, Pediatric Cardiology, C.S. Mott Children's Hospital, Ann Arbor, Michigan.*

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Middle Ear Effusion and Airplane Trips

To the Editor:

I read with interest, and a sense of joy, the practical article in the November issue of *Clinical Pediatrics* entitled, "May Children With Otitis Media Safely Fly?"[1] My extensive experience echos the conclusions of Weiss and Frost. However, some small percentage children with acute otitis media will develop exquisite pain on descent of the aircraft even when the middle ear had fluid and the eardrum demonstrated limited mobility.

My instructions to the forewarned parents are to take paregoric or acetominophen with codeine on the airplane and to purchase and learn how to use a 1-oz infant nasal aspiratory with a rigid plastic conical tip. The aspirator will allow ventilation of atelectatic middle ears when used to gently insufflate air under positive pressure through one nostril while the opposite nostril is occluded. This politzer procedure should be used only when the nasal passages are free from thick mucus or purulent secretions. Prior use of vasoconstrictive nosedrops may be necessary for "stuffy noses." This politzer procedure may be repeated during descent of the aircraft every 10,000 feet or so and overcomes Eustachian tube "locking." Thus, cooperative children with retracted eardrums who are old enough to hold a sip of beverage in their mouth and swallow it on command may safely fly.—RICHARD SCHWARTZ, MD, *410 Maple Avenue West, Vienna, Virginia.*

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