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Invited Commentary

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Since Macklin's early observations, the "air-block syndrome" has been a problem of considerable concern, especially to physicians dealing with the neonate [1]. In the past decade, the development of highly sophisticated infant pressure and volume ventilators has resulted in an increased incidence of "air-block syndrome" and its sequelae. This syndrome is characterized by alveolar overdistention with subsequent rupture of alveoli adjacent to the capillary bed and dissection of air into the pulmonary interstitium. Under a continued pressure gradient (which may be expected when ventilator support and positive end-expiratory pressure [PEEP] are employed), air dissects along the perivascular venous sheath towards the hilum of the lung and may pierce the mediastinal or visceral pleura, resulting in pneumomediastinum, pneumothorax, or both. Pneumopericardium is the most serious se-

quelae of the "air-block syndrome" and is associated with a triad of cardiovascular collapse (due to air tamponade), hypotension, and bradycardia. In the present era, pneumopericardium is almost always a complication of therapy and is usually seen in the smallest infants (often weighing less than 1.0–1.5 kg) receiving vigorous ventilatory support. While PEEP is useful in keeping the alveoli expanded, it may also reduce the infant's cardiac output. Levels of PEEP above 7.5 cm of water pressure often result in alveolar rupture and the onset of air-block. Experimental data from our laboratory suggest that alveolar rupture is dependent on both increased inspiratory pressures and the duration of pressure [2]. In an animal model, pneumopericardium occurred at pressures above 60 cm of water and resulted in hypotension, bradycardia, and cardiovascular collapse similar to the clinical data presented by Emery and his associates. Anatomic studies suggest that air enters the pericardium along the pulmonary perivascular sheath at the site of entry of the pulmonary veins. Coronary artery and systemic air emboli have also been noted in experimental animals following severe hypotension related to pneumopericardium [2].

We would strongly agree with Emery et al. regarding the urgent need to evacuate pericardial air to relieve air tamponade, and we made similar recommendations in 1970 [3]. Failure to relieve air tamponade results in the rapid demise of an already seriously ill infant. The rapid percutaneous subxiphoid passage of a silicone Angiocath® results in pericardial decompression and at least temporary improvement of the patient's condition. The present report by Emery and associates emphasizes some important clinical observations, namely, that recurrence of pneumopericardium may occur if the pericardial catheter is prematurely removed, especially if the infant is on PEEP, and that percutaneous catheter insertion may, on occasion, result in complications such as pneumothorax (if not already present) and myocardial laceration.

Over the past 2 years, 12 premature infants with pneumopericardium have been treated at the James Whitcomb Riley Hospital for Children in Indianapolis. Catheter decompression was employed in all but 2 instances and was successful in 8 patients in relieving the initial symptoms. Recurrent pneumopericardium occurred if the catheter was removed prior to discontinuation of PEEP therapy. Catheter decompression was superior to attempts at simple needle aspiration of the pericardium. In 3 instances, the catheter was unsuccessful in achieving appropriate pericardial decompression. Operative placement of a multi-eyed catheter into the pericardium under direct vision, as Emery and associates suggest, may obviate both complications related to percutaneous catheter insertion and failure to adequately relieve air tamponade. The authors' technique may be useful in selected cases of pneumopericardium and represents a potential improvement in care. Performance of this procedure, however, "on the ward" is of some concern, as appropriate lighting, operative equipment, and the actual field conditions regarding sterility may be in question. This is of particular importance in the small premature infant with relative deficiencies of IgM, the C_3 component of complement, opsonization, and a decreased phagocytic ability. It must also be kept clearly in mind that despite removal of air from the pericardium, the mortality rate associated with this complication remains quite high (> 50%). Mortality may be directly attributable to pneumopericardium or may be indirectly associated with severe underlying pulmonary pathology [4].

The improved survival in neonates with respiratory distress syndrome in this decade has been attributed to referral of such infants to tertiary neonatal intensive care facilities staffed by highly skilled personnel. Improvements in monitoring, the use of miniaturized ventilator support systems, and serial arterial pH and blood gas tension sampling have played a direct role in this effort. Pediatric physicians (both internists and surgeons) should maintain a high index of suspicion for the development of complications of aggressive ventilatory treatment. The use of fiberoptic transilluminators and frequent monitoring of the chest x-ray in patients who demonstrate deterioration of either blood gas tensions or respiratory efforts are recommended. Close attention to these details will result in early diagnosis of pneumothorax, pneumomediastinum, and pneumopericardium and allow more rapid therapy in hopes of achieving greater survival in complicated instances.

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