plication of a paralyzed hemidiaphragm are at a higher-than-usual risk for protein-losing enteropathy or other modes of eventual Fontan failure, despite having enjoyed an uncomplicated early postoperative recovery. Notwithstanding the potential for recovery of function of a paralyzed hemidiaphragm, as well as the important observations of Kumar and colleagues[1] regarding the potential benefit of prior plication in terms of mitigation of short-term post-Fontan morbidities, it is a fact that the best strategy regarding phrenic nerve injury and diaphragm paralysis is to avoid it completely.

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Commentary: Brains over brawn: Do strong diaphragm muscles matter?

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CENTRAL MESSAGE

Diaphragm plication for diaphragm paresis does not adversely affect pulmonary hemodynamics or progression through staged palliation for single-ventricle patients.

Subramanyan and colleagues[1] take an important look at the effects of diaphragmatic plication for diaphragm paresis and paralysis on successful completion of staged palliation in a population of patients with single-ventricle anatomy. Phrenic nerve injury during the course of congenital heart surgery, especially in children younger than age 2 years, is a significant source of morbidity. Most of the time, the phrenic nerve stays out of harm's way with meticulous technique and conscious avoidance of nerves. Various centers have reported anywhere from 2.2% to 4.8% occurrence of diaphragm paresis/paralysis.[2,3] The loss of function of a hemidiaphragm can reveal itself as respiratory compromise, such as failure to wean from ventilator or nonsustainable increased work of breathing, especially in neonates and infants.[4] Various published studies examine the need for and timing of diaphragm plication in treatment of a paretic diaphragm. There is relative agreement on early intervention in symptomatic patients and patients with single-ventricle anatomy.[5] A paretic hemidiaphragm's recovery does not seem to be negatively influenced by plication.[6] Diaphragm plication does not seem to negatively influence pulmonary hemodynamics.[1]

In 2008, Baker and colleagues[7] reported that approximately 70% of paretic diaphragms recover. In this contemporary look from the same institution, 63% of patients regained some function of their diaphragm by the next staged surgery. Patients underwent a pre-Glenn and pre-Fontan cardiac catheterization. They found no difference

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in pulmonary hemodynamics or hospital course compared with controls.[1] This is encouraging, crucial information to guide treatment for this physiologically sensitive patient population with diaphragm muscle dysfunction.

Perhaps this work is also a missed an opportunity to study diaphragm paresis more thoroughly. Understanding pulmonary function and exercise testing post Fontan in this subset of patients could help give insight into expected mid- and long-term outcomes. Lemmer and colleagues[8] report a tendency toward lower lung function values in patients with diaphragmatic plication, but no change in exercise capacity. Fluoroscopic imaging and ultrasonography are less precise measurements of injury or recovery. There have been other reports that use more sensitive methods of evaluation, such as phrenic nerve latencies, depolarizing afterpotential, and afterhyperpolarization to quantify nerve recovery. Reporting more detailed recovery data would be additive and contribute to understanding diaphragm paresis versus paralysis and predicting recovery potential. Amin and colleagues[3] performed a similar study and found the incidence of ascites, prolonged pleural effusions and chest tube duration, readmission, length of hospitalization, and postoperative Fontan pressures significantly higher than controls. Georgiev and colleagues[9] did not show benefit to an aggressive approach to plication (after a single unsuccessful extubation). It will be important to look more closely at the reasons for these differences.

Diaphragm paresis remains a rare, important complication of congenital heart surgery with significant morbidity. Diaphragm plication has shown not to adversely affect progression through the multiple stage palliation for single-ventricle physiology. Perhaps it is neither brain (phrenic nerve) nor brawn (strong diaphragm) that determines clinical outcomes, but rather timely plication of a weakened diaphragm.

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