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FIGURE 1. This illustration shows the fiber orientation relationships of the right ventricle whereby oblique fibers arising from the descending and ascending segments of the apical loop form the septum. The septum is surrounded by fibers with transverse muscular orientation arising from the basal loop, which comprises the free right ventricular wall.

excursion and its velocity. This movement follows septal helical coiling; “the septum is the RV lion.”⁴ Moreover, the limited contribution of the free wall is demonstrated by absence of RV failure when the entire free wall is cauterized, or replaced with a patch, provided that the septum is uninjured.⁴ Conversely, septal damage causes RV failure, especially if pulmonary vascular resistance increases.

The long-term myocardial dilemma of PR is worsened by intraoperative septal muscle damage during tetralogy of Fallot repair. Chronic PR is not innocuous. Some septal dysfunction evolves from arterial fistulas after muscular ventriculectomy, but most damage stems from impaired myocardial protection. The incidence of intraoperative septal damage in children is unknown; however, 50% of 3300 adult patients were seen to develop septal paradox.⁵ This complication is completely prevented by integrated myocardial protection.² The septum is the issue, *not* the free wall. This finding is underscored by novel procedures that include septal restoration when chronic PR is corrected.⁴

Development of surveys of intraoperative septal function in children is needed, especially because PR accentuates RV dysfunction; volume loading makes the RV cavity and wall more spherical, impairing septal function because it develops a more horizontal fiber angulation. Preventing PR by inserting outflow tract conduits with a valve may prevent pulmonary insufficiency, but focusing upon septal protection during the tetralogy of Fallot repair is important. Current myocardial protection patterns should be followed if postoperative septum function is normal but must change if septal injury develops, mirroring the adult cardiac surgical sequence.

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Reply to the Editor:

I appreciate the interest in our recently published study by Buckberg and Hoffman in their letter to the Editor. In our study, no long-term benefits were demonstrated for limited right ventricular (RV) incision relative to conventional (longer) RV incision in transannular repair of tetralogy of Fallot (TOF) in terms of RV volume and function. Buckberg and Hoffman speculate that our finding was due to the limited contribution of the RV free wall to the overall RV function. They stressed the importance of interventricular septum in maintaining normal RV function according to the helical heart model of Torrent-Guasp. I admit the limited contribution of the RV infundibulum, in which a transannular incision is made, to global RV function. As discussed in previous paper, Geva and colleagues¹ have demonstrated that the

infundibulum contributes only 13% of the total RV stroke volume. Bodhey and colleagues² found the stroke volume of the infundibulum to account for 25% of the total RV stroke volume in healthy individuals. The fact that the RV infundibulum has limited contribution to global RV function, however, does not mean that the RV infundibulum is not important in maintaining RV function. In fact, there is good evidence leading us to speculate that RV infundibular contractility may play a key role in protection of the RV against deleterious consequences of the chronic volume overload related to pulmonary regurgitation after TOF repair.^{3,4}

One should remember that many factors other than the length of the RV incision could also influence late outcomes of patients with repaired TOF. These factors include the width of the transannular patch, the number of coronary arterial branches sacrificed, the extent of infundibular muscle resection, and the methods of myocardial protection. Poor myocardial protection and ensuing septal dysfunction can definitely affect the long-term outcomes of patients with repaired TOF, and we cannot rule out the possibility of poor myocardial protection in the historical cohort of patients who underwent TOF repair long ago and whose late outcomes are being studied today. In the current era of improved myocardial protection, however, serious septal dysfunction is too rarely observed after operation for TOF for us to reconsider our current method of myocardial protection.

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AWAKE THORACOSCOPIC SURGERY: SAFETY ISSUES IN DIFFICULT AIRWAY AND USE OF THORACIC EPIDURAL ANESTHESIA

To the Editor:

We read with great interest the article by Galvez and colleagues¹ that reported successful application of awake uniportal video-assisted thoracoscopic surgery (VATS) in a patient with metastases after nasopharyngeal cancer.¹ We congratulate Galvez and colleagues¹ and agree with them that a combination of uniportal and nonintubated VATS techniques would expand the armamentarium of thoracic surgical practice in an ambulatory setting.^{1,2} There are safety issues associated with nonintubated VATS, however, and these should be highlighted in their patient management.

First, a nonintubated technique for VATS is believed to benefit those patients who would be at high risk when undergoing an intubated single-lung ventilation.³ In this respect, patients with an anticipated difficult airway are appealing candidates; however, they should be selected more stringently, especially in the case of previous head and neck surgery or radiotherapy. In our own non-intubated VATS cohort, there still

were 4% of cases requiring conversion to tracheal intubation.³ Despite a conversion protocol, emergency management of difficult airway in such patients may be more challenging, and the increased risks of airway morbidity or mortality may outweigh the benefits of nonintubated VATS, especially when a lateral decubitus position is used.

Second, the use of thoracic epidural anesthesia for minimally invasive thoracic surgery is on the decline.⁴ Not only is thoracic epidural catheterization, time-consuming and technically demanding, we are also concerned that the single injection of 0.5% bupivacaine hydrochloride (INN bupivacaine) in a large dose (15 mL) may be excessive and can be associated with significant hemodynamic disturbances because of potentially complete sympathetic block. In addition, an unpredictable high-level blockade of phrenic nerves may occur and jeopardize the diaphragmatic function, which is essential in nonintubated VATS. In our experience, 8 to 10 mL of 2% lidocaine is sufficient to achieve a sensory block between the T2 and T9 dermatomes and provides a safer therapeutic window.³ With single-incision VATS, we believe that the requirement of local anesthetic can be reduced even further, because a sensory block between the T4 to T6 may be enough for a single fifth intercostal incision.

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