“training” to sustain system pressures were reported 18 years ago^{2,3} and may be of special interest for the development of pulmonary artery banding devices. Ideally, the aim of training of a right ventricle to sustain systemic load should prevent any kind of myocardial damage and probably imitate clinical conditions such as development of pulmonary stenosis. To imitate this type of continuous load increase, we developed and studied a novel device.² The results demonstrate that training of the right ventricle follows very delicate characteristics, at least in pigs.² We found that a fast pressure increase of 3.4 mm Hg/d during 3 weeks was associated with a 20% to 30% reduction in right ventricular ejection fraction and a 100% increase in right ventricular end systolic volume. On the other hand, a slow pressure increase of 1.5 to 2.2 mm Hg/d was associated with less significant functional impairment of the right ventricle. Thus pulmonary artery banding may have harmful effects, which might be even more complex in children with heart disease. Therefore open questions should be addressed before the proposed clinical application of this new device, such as the appropriateness of stepwise load increase in comparison to continuous load increase, with special reference to the implications for right ventricular function. It should also be discussed whether the new device includes refined mechanisms that allow for sensitive adjustment of load increase.

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Reply to the Editor:

We thank Dr Sievers for his important comments. He is right in pointing out that pulmonary artery banding with the aim of training the subpulmonary ventricle is a delicate procedure that should be done with the utmost caution. The ability of the subpulmonary ventricle to adapt to the increased afterload imposed by the pulmonary artery constrictor depends on the age of the patient, baseline ventricular function, and the timing and level of afterload increase.^{1,2}

Dr Sievers points out a study by his group in which it was shown that a slow progression of pressure loading (1.5 mm Hg/d) results in a lesser increase of right ventricular end-diastolic pressure and minimal changes in ejection fraction and end-systolic volume relative to a fast progression (3.6 mm Hg/d), thus suggesting better preservation of myocardial function.³ In addition, these results were supported by the finding that slow pressure loading resulted in a steeper slope of the relationship between maximal pressure and end-systolic volume, thus reflecting improved contractile performance. The reported functional impairment after fast pressure loading in the study of Lange and colleagues³ may be related to the simple fact that a load-dependent index of cardiac performance (ejection fraction) was used to quantify myocardial function after chronic right ventricular pressure overload.

In another article,⁴ we have published the hemodynamic results of our banding study. In that article we showed that stroke volume as well as ejection fraction were decreased, whereas compensatory ventricular dilatation (Frank-Starling mechanism) was not observed after chronic right ventricular pressure loading. In the study, right ventricular pressure overload at systemic (aortic) level was produced within a period of 2 weeks. However, according to the slope and intercept of the end-systolic pressure-volume relationship, right ventricular contractile performance was found to be significantly enhanced in these hearts. In addition, we have shown that abnormalities in diastolic function, associated with the (normal) hypertrophic response to pressure loading, may be held responsible for functional impairment of cardiac function after chronic right ventricular pressure loading.⁵

Part of the difference in results between our study and that of Lange and col-

leagues³ may be explained by the ages at which the pressure overload was applied. In the latter study, young piglets with an average body weight of 22.3 kg (range 17-32 kg) were subjected to slow progressive right ventricular pressure overload. Apart from differences in species, it is likely that these animals, according to their weight, were substantially older (probably several months⁶) than the lambs used in our study (age 2-3 weeks, average body weight 6.4 ± 1.7 kg). This difference may have influenced the myocardial response to pressure loading. Finally, whereas the study by Lange and colleagues³ compared results of the banding in animals against their own prebanding values (each animal acted as its own control), we compared right ventricular function after 8 weeks of loading against an age-matched control group without banding.

We agree with Dr Sievers that adjustable constrictors should allow small and reproducible adjustments of the level of constriction. Precisely how small the adjustments can be made with our device cannot be inferred from our current results and needs further study.

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Lobar transplantation To the Editor:

In their communication detailing their observations of the detrimental effects of hyperinflation of transplanted lungs, Kozower and colleagues¹ speculate that patients receiving lobar transplants may be at even greater risk for this complication due to the greater potential for size disparity between lobe and hemithorax. This is indeed the case as we have recently reported.² We believe the mechanism to be as described by Kozower's group. We also believe this size disparity is even greater than would otherwise be predicted as the anterior-posterior dimension in cystic fibrosis patients increases because of lung hyperinflation during the period of bony development of the thorax.³ We have observed that this physiology is more apparent and the hemodynamics are more severely affected with increasing size disparity. Early in our experience, the breath stacking occasionally became severe enough to cause severe hemodynamic depression as a result of the altered respiratory mechanics.²

These observations have led us to modify our postoperative management for lobar transplantation as follows²:

1. In the operating room, 4 chest tubes are placed (2 in each hemithorax) and

connected to water-seal with no negative pressure.

2. Positive end-expiratory pressure is applied in the operating room (5-10 cm H₂O as tolerated) and continued in the intensive care unit.
3. Negative pressure is applied to the chest tubes in sequence: 5 to 10 cm H₂O to each tube, in 1-hour intervals, rotating for the first 24 hours. Later, each tube is placed on continuous suction that is gradually increased to -20 cm H₂O over the next 48 hours as tolerated.

Using the above procedure on all lobar transplant patients, we have minimized the hemodynamic consequences of the donor-to-recipient size discrepancies inherent in this procedure and have not seen similar acute deterioration in the last 90 to 100 lobar transplantation cases.

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Reply to the Editor:

We are grateful for the confirmation provided by the letter and the case report by Haddy and colleagues. Readers interested in this phenomenon are encouraged to read their article¹ for a clear and methodical description of an event that sounds identical to our "index case" in 1997. After several sporadic observations over 3 years, we attempted to capture the phenomenon in a

prospective fashion and the result is our own brief report.²

Our only disagreement with their letter is with the University of Southern California protocol for avoiding the stacking of breaths in transplant recipients with undersized grafts. In our experience, chest tubes placed to water-seal can still elicit the phenomenon when there is no air leak from the visceral pleural surface. This absence of air leaks may be uncommon after bilateral transplant, but we still begin with 1 tube on each side unconnected and thus open to atmospheric pressure. Also, the subsequent application of negative pressure to 1 of 2 chest tubes seems equivalent to applying it to both tubes: either the pleural pressure is negative or it is not. Two tubes on suction should only make a difference in pleural pressure if there are large air leaks and the removal of air from the pleural space is faster with 2 tubes on suction. If one tube is placed on suction and breath stacking does not happen immediately, our experience suggests that it will not occur.

Otherwise, we are reassured that our observations are validated by a simultaneous and independent report from a distinguished team of lung transplant experts.

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Brachial artery cannulation To the Editor:

In their report Galajda and colleagues¹ describe their technique of brachial artery cannulation for two patients with type A aortic dissection. One patient was cannu-