forming the loss of resistance test with saline or local anesthetic rather than air.

There are two aspects of their epidural technique that may have contributed to the high incidence of air embolism. First, they used the "hanging drop" sign to identify the epidural space. I would propose that if carefully investigated, this method for identifying the epidural space would be associated with a higher incidence of entry of the epidural needle tip into an epidural vein than if the space is identified by "loss of resistance" in which continuous pressure is applied to the plunger of a small syringe filled exclusively with fluid, either saline or local anesthetic. Second, it has been shown that the incidence of insertion of an epidural catheter into an epidural vein is greater when the epidural space is not first expanded by the injection of fluid, than when it is.[2] I believe that the incidence of air embolism from epidural anesthesia reported by Naulty *et al.* would have been far lower if greater care had been taken to push the epidural venous plexus out of the way by the use of continuous pressure with fluid as the needle was being inserted and by expansion of the space with 7 to 10 ml of fluid before inserting the catheter. Further, I suggest that these simple precautions are more important than hydration or posture for minimizing this complication.

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In reply:—The "hanging drop" sign is used at our institution as a training device for residents, and since this is our standard technique, this is what we evaluated in the study. We noted in the paper that the possibility of air embolism would be reduced by using the loss of resistance technique "with saline or local anesthetic rather than air." However, I feel that if 7 to 10 ml of "fluid" are used to distend the epidural space prior to insertion of the epidural catheter, the fluid should *not* be local anesthetic, since this large volume given intrathecally or intravenously could cause obvious difficulties. *All* aspects of technique in epidural anesthesia are critical.

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Pulmonary Artery Catheters in Eisenmenger's Syndrome: Many Risks, Few Benefits

To the Editor:—I read with both interest and chagrin the case report by Devitt *et al.*[1] recounting the fatal outcome of pulmonary artery monitoring in a parturient with Eisenmenger's syndrome. Although their discussion of the risks was entirely reasonable, the discussion of the benefits of a PA line in this disease demands further consideration.

A number of potential benefits for pulmonary artery catheterization in this patient could be postulated:

1) Pulmonary artery catheterization allows measurement of PA occluded pressure to evaluate left ventricular volume status. Eisenmenger's syndrome is characterized by obliterative pulmonary vascular disease in which PAOP may not truly reflect LVEDP or LVEDV. In this syndrome, the right ventricle, not the left, is at highest risk for dysfunction. Right atrial pressure should provide an adequate assessment of vascular volume.

2) The authors' discussion of PA catheterization for evaluation of shunting should be reevaluated. In the patient with pulmonary hypertension and a right-to-left shunt, the degree of shunting is easily evaluated by serial analysis of arterial blood gases and by observing the clinical signs of increasing tachypnea, cardiac rhythm

disturbances, and peripheral cyanosis. I think that evaluation of peripheral oxygen saturation and symptoms is a far easier and an equally accurate method for determining the magnitude of shunting in this disease.

3) The authors do not mention the use of PA pressure monitoring for the diagnosis of an acute event resulting in the increase of already elevated pulmonary arterial pressure (*e.g.*, amniotic fluid embolus). In this event, symptoms of circulatory collapse and profound arterial oxygen desaturation would allow one to make the diagnosis.

4) Finally, a pulmonary artery catheter could be useful for infusing therapeutic vasoactive agents directly into the pulmonary artery in an attempt to reduce peripheral systemic effects. Unfortunately, the use of vasoactive agents is unreliable in this syndrome. Spinnato describes a patient with Eisenmenger's syndrome who underwent an uneventful anesthetic for cesarean section.[2] Pulmonary artery pressures were monitored and remained stable during anesthesia only to rise acutely in the early postoperative hours. This event was characterized by bradycardia, premature ventricular contractions, diaphoresis, cyanosis, and systemic hypotension. Vasoactive substances (atropine and phenylephrine) were not useful. The therapy of choice is oxygen for this problem and in the case just mentioned, it was immediately successful at restoring pulmonary arterial pressure to previous levels and eliminating symptoms.

I believe that the authors' notion that the site of the shunt should dictate use of a pulmonary artery catheter in Eisenmenger's syndrome is spurious. Pulmonary hypertension in this disorder is due to continuous, torrential pulmonary blood flow. When the left-to-right shunt occurs distal to the tricuspid valve, pulmonary flow is delivered at systemic pressures and causes accelerated, irreversible pulmonary vascular disease. In order for an atrial septal defect to cause the syndrome, it must be very large or there must be left atrial outflow obstruction such as mitral stenosis. No matter what the site of the shunt is, using pulmonary artery pressures to assess shunting serves primarily to focus the anesthesiologist's attention on the monitor rather than the patient.

Because of the above considerations, and with the outcome of this case in mind, it is my view that the use of PA catheterization in the patient with Eisenmenger's syndrome carries definite risks and very few benefits. This places me in a position directly opposed to those who recommend using the catheter as routine monitoring in these patients.[3]

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In reply:—We thank Dr. Robinson for his comments on our paper.[1] We agree wholeheartedly with his concluding sentence that PA catheters not be used as a routine monitoring procedure with this syndrome. However, we disagree on several points.

The degree of shunting can be evaluated with serial blood-gas analysis, which was done, and continuous oxygen saturation monitoring is valuable. However, clinical signs of tachypnea and cyanosis are not of much value since they are always present, at least in critically ill patients with this syndrome. If we wait for cardiac rhythm disturbances to tell us of hypoxemia, we may have sealed the patient's fate.

Dr. Robinson correctly emphasizes the value of the high inspired oxygen levels chosen in our patient.

Our case report was meant to emphasize the point that risk-benefit ratios of inserting a Swan Ganz® catheter are not the same in all patients with Eisenmenger's syndrome. Patients with a large atrial septal defect (ASD) "giving a functionally common atrium" and patients with interventricular communications have few benefits derived from inserting a PA catheter. Here we agree that PA catheters should not be used. But these patients should not be lumped together with patients with smaller ASDs or aorto-pulmonary communications with a pressure gradient. Here the PA catheter gives