To the Editor—Recently Urbanowicz et al.1 described the potential for esophageal damage with transesophageal echocardiography (TEE). We have two questions and related comments that pertain to this study: 1) Does esophageal pressure (ESP) as measured by the modified probe accurately reflect the pressure produced by contact between a conventional TEE probe and the esophagus? 2) What is the clinical significance of ESP? Urbanowicz et al. studied six patients and three dogs with a modified TEE probe designed to measure ESP generated by probe flexion. The transducer tip of the modified probe was fitted with a water-filled Silastic balloon connected to an external pressure monitor. The balloon tip disperses the force generated by probe flexion onto a larger surface area of the esophagus with a resultant decrease in esophageal surface pressure. Therefore, this study may significantly underestimate the force generated by a conventional TEE probe, and hence may underestimate risk of esophageal injury.

Urbanowicz et al. attempted to simulate the worst clinical scenario with continuous probe flexion and could not demonstrate esophageal injury, either histologically (animals) or symptomatically (humans). None of the animals sustained "abnormally" high ESP, but one of the patients did, despite no history of preexisting esophageal disease. It would be informative to know if the investigators noticed any resistance to probe flexion in this patient.

The clinical significance of ESP is appreciated by gastroenterologists who use esophageal tamponade and dilatation. To minimize injury during esophageal tamponade, inflation pressures are limited to 35–45 mmHg.2-4 Perhaps an animal model can be developed to establish a relationship between elevated ESP during TEE and esophageal injury. If elevated surface pressure was associated with esophageal injury, then this measurement may have significant clinical implications in patients undergoing TEE.

Ultrasound transducers not only produce pressure at the site of contact but also generate heat and place the patient at risk for thermal injury. The water-filled balloon on the modified probe used in this study may function as an insulator and protect the esophageal mucosa from injury. If maximum power output was maintained for up to 8 h in the animal studied with the conventional TEE probe (i.e., without a balloon), it is of interest that no evidence of thermal injury was detected on histologic examination of the esophageal mucosa. Most TEE instruments designed for intraoperative use have a thermocouple to monitor transducer tip temperature and possess an automatic shutdown mechanism if a preset threshold (42°C) is reached. Thermal monitoring is clinically relevant only when the instrument is used for extended periods or when adjacent structures are deliberately cooled (e.g., posterior left ventricle with cold cardioplegia).

Urbanowicz et al. should be applauded in addressing this issue. Their study confirms previous animal studies that reported no evidence of esophageal injury after TEE.4 Although continuous compression of tissue blood supply may result in ischemia and necrosis, the consequences of a routine TEE evaluation with intermittent compression are poorly understood and are unlikely to be associated with significant injury. However, we believe that the approach used in this study significantly underestimates the force that is transmitted onto the esophageal wall by a conventional TEE probe.

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contact with a pressure producing surface.* It is unlikely that within this biologic system a pressure "point" occurs which is of higher pressure and is dissipated by virtue of the surface area of the pressure transducer. The issue is a reasonable one to consider for the future, but there is no reason to think it is a problem in the current study.

It is not clear what level of pressure would be clinically significant for short-term or long-term compression of the esophagus by the transesophageal probe. When designing this experiment, we theorized that pressures of less than 35 mmHg would be unlikely causes of tissue necrosis as a result of ischemia. Savino and Weiss seem to have come to the same hypothesis. Although we did not sense any increased resistance to probe flexion in our patient with "abnormally" high esophageal surface pressure, the results of our study in this patient have made us cautious in using the transesophageal probe in patients with preexisting thoracic disease around the esophagus.

Most transesophageal echocardiography equipment does have a transducer-tip temperature monitor with an automatic shut-down mechanism as mentioned by Savino and Weiss. Nevertheless, in the one animal in whom the probe was left in place for 8 h, at maximum power transmission settings and without the pressure transducer interposed between the probe and the esophagus, there was no evidence of thermal or mechanical damage. This may be more important if there is no blood flow to stabilize the tissue temperature in the face of even minor heating from the ultrasonic device. This could be true in situations of circulatory arrest. In the current series, there was no evidence of esophageal damage.

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