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**BONANNO'S CATHETER: A LESS INVASIVE AND COST-EFFECTIVE ALTERNATIVE FOR DRAINAGE OF PLEURAL EFFUSION**  
**To the Editor:**

It was with great interest that I read a response letter by Asopa and Patel<sup>1</sup> to an article by Chetty and colleagues.<sup>2</sup> Their article describes the safe use of a Bonanno catheter for drainage of a pleural effusion. Asopa and Patel called for modifications to the catheter before it was considered for general use after an incident at their center in which the left ventricle was inadvertently pierced during catheter insertion.

Traditionally, the Seldinger technique has been used to insert a pigtail drain into the intercostal space to allow the safe drainage of a pleural effusion.<sup>3</sup> Many risks and technical difficulties come with the use of this technique. Often the needle is misplaced when the aspiration syringe is being removed, leading to drain insertion outside the chest cavity or indeed the needle being advanced into the heart or large vessels.<sup>4</sup>

The use of the Bonanno catheter as described by Chetty and associates<sup>2</sup> is an innovative use of a suprapubic catheter and, in my experience, a simple and safe way to insert a chest drain. The technique is fully described in their article, and there are several steps that ensure that the procedure is carried out as safely as possible and the complication risk is minimized. Asopa and Patel<sup>1</sup> believed that the

length of the catheter and the lack of external markings caused the method to become unsafe compared with the Seldinger technique. I disagree. If the method of insertion laid out clearly and concisely in the article by Chetty and associates is followed carefully, complications can be avoided. It is recommended that during local anesthetic infiltration, the parietal pleura be breached and a test aspiration carried out. This allows the operator to feel and see how far the catheter will need to be advanced; the test will ensure that the effusion is present at that intercostal level and that it is large enough to be drained safely. If there are any problems with this test aspiration, the procedure should be abandoned. Second, when advancing the catheter, one should be aspirating all the time so that as soon as the pleura is breached and the effusion is aspirated, it is apparent that the catheter is in the cavity and the operator can stop advancing the needle. The technique is then extremely simple; the catheter is advanced while the needle is being held still, similar to the technique for venous cannulation, until the catheter has been fully inserted.

I see no reason why one should lose track of how far the needle has been advanced. If the operator is able to aspirate fluid with a local anesthetic needle, then the distance the catheter and tracer need to be advanced will be relatively small. If the operator is aspirating the entire time, then the equipment should not be advanced beyond the effusion and into the heart or large vessels.

I appreciate the concerns that Asopa and Patel have expressed, and indeed markings on the catheter may prove to be helpful to some, but if Chetty and colleagues' comprehensive guidance is followed, no problems should be encountered.

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**HYPOXEMIC REPERFUSION OF ISCHEMIC STATES PREVENTS MYOCARDIAL INJURY, ATTENUATING THE OXIDATIVE AND INFLAMMATORY RESPONSE**  
**To the Editor:**

We read with great interest the recent article by Abdel-Rahman and colleagues<sup>1</sup> on the favorable effect of "hypoxic reoxygenation" in the attenuation of myocardial ischemia-reperfusion injury after cardioplegic arrest. The importance of progressive oxygen re-entry into ischemic tissues has been shown since 2001 in the brains of a porcine model of cardiac arrest.<sup>2,3</sup> The favorable effect of the strategy was documented through better overall neurological performance, less lipid peroxidation,<sup>2</sup> and attenuation of the brain morphologic changes.<sup>3</sup> Because of malondialdehyde (MDA) increase, oxidative stress has been theoretically implicated, though not confirmed by the measurement of reactive oxygen species (ROS).

With regard to the current study, 3 comments are to be addressed. First, the authors deduced oxidative myocardial injury by determining MDA concentrations in coronary sinus blood without comparative reference MDA concentrations at the distal site. Nevertheless, ROS seem to be directly cardiotoxic, independent of the site of their