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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 Patel¹ to an article by Chetty and colleagues.² 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.³ 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.⁴

The use of the Bonanno catheter as described by Chetty and associates² 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 Patel¹ 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 colleagues¹ 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.^{2,3} The favorable effect of the strategy was documented through better overall neurological performance, less lipid peroxidation,² and attenuation of the brain morphologic changes.³ 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

production. In fact, normoxemic reperfusion of a distal ischemic organ (eg, the intestine) has been shown to cause myocardial injury that was prevented with progressive oxygen re-entry during reperfusion.⁴

Second, in the above studies, similarly to the current one, with the exception of MDA measurement, the favorable effect of hypoxemic reperfusion was not substantiated by direct indices of oxidative stress alterations, such as ROS measurement. ROS and cytokines were recently assessed in a model of hypoxemic resuscitation of hemorrhagic shock in rabbits and found to be particularly diminished compared with those seen in the standard resuscitation mode of clinical practice.⁵ The weakening effect on cytokine production is an additional important achievement of hypoxemic resuscitation because these mediators lead to post-ischemia-reperfusion inflammatory injury.

Third, the authors have repetitively used erroneously the term “hypoxic reoxygenation” instead of “hyperoxic reoxygenation” to describe Pao₂ values of 250 to 350 mm Hg, which is usually applied in the clinical setting. Furthermore, we consider even more accurate the term “hyperoxemic” because it describes higher arterial oxygen content largely and easily modifiable by fraction of inspired oxygen changes; in contrast, the term “hyperoxic” describes tissue oxygen concentration dependent on addition to hemodynamic parameters. Correspondingly, the other treatment branch is represented by gradual reoxygenation by means of an initial “hypoxemic” instead of “hypoxic” period.

Our published data support the findings of Abdel-Rahman and colleagues¹ regarding the principal idea of the strategy for the prevention of reperfusion injury,²⁻⁴ the restoration of hemodynamic parameters,^{4,5} and the attenuation of oxidative stress.²⁻⁵ Additionally, these data provide a further insight into the potentially protective role of this strategy through the atten-

uation of the inflammatory response during reperfusion.⁵

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HYPOURICEMIC EFFECT OF STATINS: ANOTHER PLEIOTROPIC BENEFIT?

To the Editor:

We read with interest the recent trial by Hillis and associates,¹ demonstrating that hyperuricemia was associated with poorer survival after coronary artery bypass grafting (CABG), independently of other risk factors, including the European System for Cardiac Operative Risk Evaluation (EuroSCORE). Also, this increased risk was particularly evident among patients with levels in the upper quartile.

It has been showed that treatment with statins² can reduce serum uric

acid (SUA) levels; thus they would have an additional beneficial effect apart from that derived for lowering low-density lipoprotein cholesterol (LDL-c) levels. However, little information is available about the effects of high doses of statins on the SUA levels. The present study focuses on the possible effect of using a high dose of atorvastatin on SUA levels. We therefore hypothesized that the use of the highest dose of atorvastatin (80 mg/day) in high-risk patients under treatment with atorvastatin 40 mg/day would reduce SUA levels.

We studied 29 “high-risk” patients (inclusion criteria: severe triple vessel disease but rejected for bypass for extensive coronary disease, severe effort angina after coronary artery bypass, and premature coronary disease with ≥ 3 risk factors) with an abnormal lipid profile despite atorvastatin 40 mg/day at baseline and at 3 months after increasing the dose to 80 mg/day. Baseline results in patients were compared with those of 39 healthy controls. The lipid profile, C-reactive protein levels (CRP), SUA levels of patients and controls, and the changes after increasing doses of atorvastatin are shown in Table 1, A and B. At baseline, patients had higher levels of SUA ($P = .036$) compared with controls. Follow-up was completed in a total of 26 patients (2 patients withdrew voluntarily from the study and 1 patient had no samples at 3 months). After the dose of atorvastatin had been increased, a significant decrease in CRP ($P = .004$) and SUA ($P = .018$) levels were detected among treated patients. Also, these reductions did not show correlation with the changes of lipid profile (cholesterol, triglycerides, LDL-c, and high-density lipoprotein cholesterol). No adverse effects were observed in any patients.

The present study shows that increasing doses of atorvastatin at 80 mg reduces SUA levels compared with the 40-mg dose in high-risk patients with ischemic heart disease. Previous studies have shown that