antifungal) and the duration of therapy (lifelong antibiotic therapy?).

In our opinion, the open questions remain: (1) Which antibiotic therapy should be performed in patients with ABF treated with thoracic endovascular aneurysm repair? (2) How long should antibiotic therapy be continued?

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Reply to the Editor:

Bozzani and colleagues¹ state that surgical correction of an aortobronchial fistula, particularly open correction of a thoracic aneurysm, carries a fairly high postoperative incidence of stent-graft infection. To the contrary, minimal infection rates were observed after endovascular stent placement. The authors question whether antibiotic therapy should be administered after this minimally invasive operational procedure.^{1,2} There is scarce literature on immunologic consequences after stent implantation in humans. According to immunologic data obtained from patients undergoing heart operations with cardiopulmonary support and abdominal surgery, any operation performed in humans induces a state of immune suppression in vivo. Therefore, patients undergoing heart surgery (cardiopulmonary support)

should receive aggressive 5-day antibiotic treatment in accordance with the insight of an induced “systemic immune suppression” after heart surgery.^{3,4} In regard to the ongoing discussion of antibiotic treatment after endovascular stent implantation, the following approach seems to be feasible. Studies have to be initiated to investigate the immunologic consequence of open and endovascular stent implantation in humans (eg, abdominal aorta aneurysm repair, open, closed), and “yes,” antibiotic treatment should be applied for 4 to 5 days after endovascular stent placement to potentially “prohibit” pain from infection (local, systemic) in patients.

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A COMPLEMENTARY TECHNIQUE TO CARBON DIOXIDE DE-AIRING IN OPEN CARDIAC OPERATIONS?

To the Editor:

In a recent study Al-Rashidi and associates¹ concluded that “bilateral ...

pulmonary collapse and successive filling of the lungs with ... concomitant increase in mechanical ventilation during de-airing of the left side of the heart significantly reduces the number of systemic MES [microembolic signals]... and ... air emboli.”

Inasmuch as the study's limitations were not mentioned, we would like to discuss a few:

1. Inasmuch as the patients were alternately allotted to control and study groups, the principle of randomization was ignored. Moreover, the principle of unbiased assessment was also compromised inasmuch as the single surgeon, who actively followed the degree of de-airing via transesophageal echocardiography (TEE) during the surgical de-airing maneuvers before the end of cardiopulmonary bypass (CPB), thus participated in the evaluation of the technique, which he himself had proposed.
2. The authors enumerate 9 exclusion criteria, including accidental opening of the pleural cavity and chronic obstructive lung disease, although all operations were performed by one highly experienced surgeon. Consequently, the generalizability of the results cannot but be very limited.
3. According to the Methods section, TEE and transcranial Doppler (TCD) measurements were only performed *after* CPB. However, in the Results section, the authors make comparisons with the number of MES *before* the end of CPB. Moreover, “The aortic root was de-aired” before release of the crossclamp only in the study group.
4. The possible risks of a left ventricular vent inserted through the apex of the heart (eg, bleeding and arrhythmias), a prerequisite of the technique, have not been mentioned.
5. In contrast to common practice as well as the study group, the control group patients had their lungs ventilated continuously during

crossclamping and then fully already after release of the cross-clamp. This may have negatively influenced the number of detected MES and TEE-detected air emboli in the control group.

6. The authors claim that “their results are similar, if not better, to those described with carbon dioxide insufflation” and refer to a study by us.² We find this comparison inappropriate. Continuous carbon dioxide insufflation of the open cardiothoracic cavity during open cardiac surgery with an effective device creates a local atmosphere of 100% carbon dioxide,^{3,4} whereby only carbon dioxide and not air can enter the heart and the vessels directly. Thus, any gaseous microemboli detected with TEE or TCD during and after CPB must contain carbon dioxide and not air, unless air is introduced indirectly via cannulas. As expected, we found that the TEE-detected microemboli were fewer and disappeared much quicker in our treatment group receiving carbon dioxide.² In contrast, the new surgical de-airing technique¹ did not eliminate the risk of air embolism, inasmuch as air emboli were still present in the left side of the heart and MES containing air still occurred. Furthermore, our study² randomized patients, all 6 surgeons were blinded to TEE findings, the apex of the heart was not cannulated, and we did not have exclusion criteria.

In conclusion, if proven able to reduce air embolization in a correctly performed randomized trial, the described technique¹ may be a complement to de-airing with carbon dioxide only, if air has been introduced into the left heart and the great vessel directly, by use of an inappropriate carbon dioxide insufflation technique, or indirectly, through cannulas.

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Reply to the Editor:

We thank Peter Svenarud and his colleagues for their valuable comments and questions about our article in the *Journal*.¹ We will try to answer their queries in the order of their appearance in their letter to the Editor.

In our prospective controlled study, the de-airing was performed in both groups under intraoperative transesophageal echocardiographic (TEE) control. When air bubbles ceased to appear in the left side of the heart, the de-airing was stopped and the de-airing time noted. During this period, microembolic signals (MES) were also recorded by transcranial echo-Doppler (TCED) on line in both groups. The surgeon obviously could not influence these data in favor of one or the other group. Subsequent analysis of the data showed that, in addition to the significantly longer de-airing time ($P < .001$), the number of MES recorded were also significantly higher in the control group ($P < .002$). We also found a good correlation existing between the TEE and TCED measured air emboli in both groups (during the first 10 minutes after weaning patients

from cardiopulmonary bypass), so a bias in favor of the technique proposed by us is unlikely.

The clinical study under discussion is the second in a series of studies under way. Patient inclusion and exclusion criteria were purposely kept strict to allow us to draw definite conclusions from the small number of patients included in the study. It is, however, too early to predict the real limitations to the de-airing technique proposed by us.

Our study has focused on two end points: (1) “the de-airing time” based on the cessation of air emboli on intraoperative TEE and (2) the “residual microemboli” during the first 10 minutes after termination of cardiopulmonary bypass as assessed by intraoperative TEE and TCED. In addition, the number of MES as recorded by online TCED during the “de-airing time” itself were also analyzed. During the de-airing period itself, assessment of the magnitude of the air emboli on TEE is not only difficult but also fraught with numerous errors. Moreover, a major amount of these emboli are being evacuated by the left ventricular vent. That is why TCED data alone were considered for assessing the magnitude of systemic air emboli in both groups during the de-airing period itself.

In the control group, the aortic root was de-aired by filling it passively with blood from the left ventricle before final closure of the aortotomy. Thereafter, the left side of the heart was manually de-aired through the left ventricular apical vent under continued passive filling of the lungs with blood and full ventilation of lungs with 100% oxygen. The aortic clamp was released first thereafter. In the study group, on the other hand, the aortic root was de-aired by filling it passively with blood by release of the aortic clamp under low systemic blood pressure over a short period of time before final closure of the aortotomy. The aortic clamp was released