## Reply to the Letter to the Editor

### Reply to Apostolakis et al.

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We would like to thank Dr Apostolakis for his valuable comment on our recently published results on procalcitonin (PCT) in the postoperative course after decortication in case of pleural empyema [1,2].

The point of the study was the observation of the *postoperative* course of PCT *in comparison to CrP*. We registered that PCT seems 'to reflect the postoperative clinical course more accurately than CrP' and has had 'a good correlation with the postoperative course in case of SIRS or sepsis' [2]. On the other hand low preoperative levels of PCT, as was the case in two patients (9.1%), do not exclude a localized (without systemic signs) infection. The patient with the postoperative recurrent pleural effusions showed a preoperative level of 0.35 ng/ml. This is not a low level but rather a sign of an infection, yet not necessarily a systemic one (serum or plasma PCT concentrations of healthy persons are measured with the Kryptor-assay are below 0.06 ng/ml [3]).

If there was only the sneaking suspicion that a pleural empyema was due to tuberculosis (clinic, anamnesis) this patient was excluded from the trial from the beginning of treatment. So not one of the patients in the presented study had tuberculosis.

We are sure that in case of an earlier availability of the PCT values most of the patients with an antibiotic treatment after discharge would not have received antibiotics (up to 85.7%). 'So the use of PCT instead of CrP in the postoperative course after decortication could save at least 7 days of antibiotic treatment' [2]. This is in accordance with other trials [4,5].

Indeed, the number of patients is small, in this prospective, but not randomized trial. And it is known that PCT levels show a wide variation but: 'In general dynamics of PCT levels, rather than the absolute values, may be more important for identifying patients with persistent infection or infectious complications after surgery' [2].

Therefore we also think it is very important to have randomized studies with more patients, maybe in a multicenter trial. These studies should determine the value of PCT as a promising marker in thoracic surgery not only after decortication in case of pleural empyema.

### References

[1] Apostolakis E, Prokakis C, Dougenid D. Are procalcitonin levels sufficient for the follow up of patients undergoing lung decortications for pleural empyema? Eur J Cardiothorac Surg 2009;35:194.
[2] Carboni G, Fahrner R, Gazdhar A, Printzen G, Schmid RA, Hoksch B. Comparison of procalcitonin and CrP in the postoperative course after lung decortications. Eur J Cardiothorac Surg 2008;33(5):777—80.
[3] Morgenthaler NG, Struck J, Fischer-Schulz C, Seidel-Mueller E, Beier W, Bergmann A. Detection of procalcitonin (PCT) in healthy controls and patients with local infection by a sensitive ILMA. Clin Lab 2002;48(5—6):263—70.
[4] Hochreiter M, Köhler T, Schweiger AM, Keck FS, Bein B, von Spiegel T, Schröder S. Antibiotic treatment of surgical intensive care patients: procalcitonin to guide duration of therapy. Anaesthesist 2008;57(6):571—7.
[5] Stolz D, Crist-Crain M, Bingisser R, Leuppi J, Miedinger D, Mueller C, Huber P, Mueller B, Tamm M. Antibiotic treatment of exacerbations of COPD: a randomized, controlled trial comparing procalcitonin-guidance with standard therapy. Chest 2007;131(1):9—19.

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## Letter to the Editor

### Anterior leaflet augmentation to address tethering-induced functional tricuspid regurgitation

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We read with great interest the article by Dreyfus et al. on tricuspid leaflet augmentation in functional tricuspid regurgitation [1]. We congratulate the authors for their innovative approach to relieve tethering-induced functional tricuspid regurgitation and their excellent results. As mentioned by the authors, inadequate tricuspid leaflet coaptation has been shown to arise from annulus dilatation greater than 40 mm and tethering of the valve leaflets in functional tricuspid regurgitation. Annuloplasty addresses annular dilatation, however it has not been shown to improve leaflet tethering. Several authors have proposed valve repair techniques to address severe tethering, such as a tricuspid version of the Alfieri repair or 'clover' technique [2] or right ventricular remodeling [3]. The method described by Dreyfus et al. is innovative, in that it is the first to increase leaflet mobility and coaptation in the dilated right ventricle. However, paraphrasing the author's own remarks on functional mitral regurgitation repair at the 22nd EACTS meeting, this remains a valvular approach to a ventricular problem. Furthermore, Park et al. recently showed in a real-time 3D echocardiographic study of 54 patients with functional tricuspid regurgitation [4] that tethering was predominantly due to the septal leaflet, and not the anterior leaflet. These results are counter-intuitive,

as right ventricular free wall dilatation would appear to be a more logical explanation for tethering, resulting in predominantly anterior leaflet tethering.

Anterior leaflet augmentation should increase leaflet coaptation and decrease tethering height and tenting volume. However, given the new data provided by Park et al., this repair will significantly displace the coaptation plane closer to the septum, with a possible risk of dynamic RVOT obstruction, and it will not restore a completely physiological function to the tricuspid valve. The risk of lesions to the atrioventricular node precludes septal leaflet enlargement similar to the technique described. The fate of untreated autologous pericardium in the long-term should be assessed, as deterioration could lead to decreased anterior leaflet mobility and repair failure. Another possible strategy to treat tethering would be to suspend the septal leaflet free edge to the native anterior annulus or anterior annuloplasty ring, as we have reported in posterior leaflet retraction of the mitral valve [5] and as we have used successfully in two patients with tricuspid septal leaflet tethering in Ebstein's anomaly.

We congratulate the authors on their innovative and thoughtful approach to a complex problem. Further research into the mechanisms of functional tricuspid regurgitation is warranted and surgical strategies to treat them should be adapted. We humbly suggest, given the latest echocardiographic data on the mechanisms of tricuspid tethering, that perhaps septal leaflet augmentation would provide a more physiological solution, although it does not appear realistically feasible. Anterior leaflet augmentation should be considered a reasonable alternative, keeping in mind the alterations to the function of the tricuspid valve.

## References

[1] Dreyfus GD, Raja SG, John Chan KM. Tricuspid leaflet augmentation to address severe tethering in functional tricuspid regurgitation. Eur J Cardiothorac Surg 2008;34(4):908—10.
[2] De Bonis M, Lapenna E, La Canna G, Grimaldi A, Maisano F, Torracca L, Caldarola A, Alfieri O. A novel technique for correction of severe tricuspid valve regurgitation due to complex lesions. Eur J Cardiothorac Surg 2004;25(5):760—5.
[3] Kappert U, Tugtekin SM, Ouda A, Alexiou K, Schmeisser A, Schoen S, Matschke K. Right ventricular reduction as an adjunct procedure in tricuspid valve repair. Ann Thorac Surg 2008;85(5):e27—9.
[4] Park YH, Song JM, Lee EY, Kim YJ, Kang DH, Song JK. Geometric and hemodynamic determinants of functional tricuspid regurgitation: a real-time three-dimensional echocardiography study. Int J Cardiol 2008;124(2):160—5.
[5] Sierra J, Lahlaidi NH, Christenson JT, Kalangos A. Mitral valve insufficiency: suspension of the posterior leaflet to the mitral annuloplasty ring. Ann Thorac Surg 2008;85(2):e6—8.

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# Reply to the Letter to the Editor

## Reply to Myers et al.

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We are grateful to Myers et al. [1] for their appreciation of our innovative technique [2] and more importantly for providing us the opportunity to address key issues relevant to the understanding of this technique as well as the pathophysiology of functional tricuspid regurgitation (FTR).

Septal leaflet lacks a well-defined papillary muscle and is attached to the interventricular septum through chordae, each of which has its own rudimentary papillary muscle. Augmentation of septal leaflet is unlikely to achieve the desired results of elimination of residual/recurrent regurgitation as increase in surface area of the septal leaflet will be negated by its limited excursion due to lack of papillary muscle. Moreover, when tethering is present the septal shift is minimal as opposed to movement of the anterior free wall of the right ventricle (RV) that is the major contributor to this process.

Suspension of the septal leaflet free edge to the native anterior annulus or anterior annuloplasty ring is no doubt a potential strategy but unfortunately not an ideal strategy to tackle severe leaflet tethering. This is for two reasons. Firstly, this technique puts the tissues under increased tension and restricts leaflet motion. Secondly and more importantly it fails to increase the coaptation surface, which is the primary pathology in tethering. On the contrary, our technique is unique amongst existing strategies as it not only avoids these aforementioned drawbacks but also preserves the functional relationship of the tricuspid valve leaflets by ensuring coaptation not at its theoretical level but as deep as needed into the RV cavity.

We acknowledge the comment of Myers et al. [1] regarding the fate of untreated autologous pericardium in the long-term. However, we will take this opportunity to clarify that use of untreated autologous pericardium is not by choice but due to health and safety regulations in United Kingdom that prohibit use of glutaraldehyde in the work place.

As for dynamic right ventricular outflow tract obstruction (RVOTO) after the use of our technique, we will like to mention that RVOTO is impossible due to simple anatomical facts, which differentiate the right ventricle from the left ventricle. Firstly, there is a lack of continuity between the anterior leaflet of tricuspid valve and pulmonary valve annulus unlike the anterior leaflet of mitral valve and its continuity with the aortic valve annulus [3]. Secondly, the inflow tract and outflow tract in the right ventricle are distinct due to the presence of supraventricular crest unlike the left ventricle where no such distinction is present. Thus, due to these anatomical arrangements whereas dynamic left