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*Tel.: +44 141 201 0269.
E-mail address: sgunpat@hotmail.com

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Reply to the Letter to the Editor

Reply to Shanmugam

Young Tae Kim*, Jun Sung Kim
Department of Thoracic and Cardiovascular Surgery, Xenotransplantation Research Center, Seoul National University Hospital, Cancer Research Institute, Seoul National University College of Medicine, 28 Yongon-Dong, Chongro-Gu, Seoul 110-744, South Korea

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Keywords: Congenital cystic adenomatoid malformation; Surgery; Complication; Early postnatal period

We would like to express our appreciation to Mr Shanmugam for his letter regarding our article on congenital cystic adenomatoid malformation (CCAM) [1]. He pointed out that the paper failed to provide a new strategy for deciding the surgical timing in CCAM. His comments on the traditional strategy for CCAM are appropriate and we agree with them. As he mentioned, many patients will have antenatally diagnosed CCAM in the future, but many of them may not be present with any significant respiratory symptoms. These patients would not have been diagnosed antenatally in the past until a chest radiograph was taken with/without respiratory symptoms at some point of their life in order to make a diagnosis. Traditionally, we have recommended surgical resection for CCAM, diagnosed either incidentally or with minimal symptoms, at the time of diagnosis for various reasons mentioned in the article. However, there is no clear guideline of management strategy for antenatally diagnosed CCAM patients. Should we recommend surgery for those patients? If so, when is the best timing for surgery?

We reviewed our experience, focusing on the timing of surgery and postoperative complications. Although, we found that minor complication rates increased with the patient's age at the time of operation, the odd ratio per month increase of age was only 1.024. This indicates that the risk of complication will not be significantly increased even if the operation is delayed several months. We also suggested that waiting several years may result in a substantial increase of risk and such a lengthy delay should be avoided. In our experience, we did not find operative mortality nor morbidity among cases operated during the neonatal period. This may not be generalized for every hospital. However, in the major centers where neonatal surgeries have been established and performed routinely, we believe that surgery for CCAM can be performed during the neonatal period without a substantial increase of risk. Considering these factors, we recommended to perform elective surgery at 2–3 months of age for asymptomatic CCAM patients.

With regards to the need of intensive care unit care, there will be many patients who will not need such care. However, until we are clearly able to predict the postnatal outcome of antenatally diagnosed CCAM, at least close observation for any respiratory symptoms should be mandatory.

Adzick's classification is based on the ultrasound size and appearance whereas Stocker's classification is based on the histopathologic findings. It should be emphasized that there is a poor correlation between ultrasound classification and resected histology. It has been suggested that macrocystic lesions tend to be stable but can enlarge and mostly require postnatal surgical excision, whereas microcystic disease may diminish or resolve during pregnancy and, if so, can simply be followed-up and investigated appropriately [2]. However, in general, we agree with Mr Shanmugam's comments about the macrocytic and microcytic lesions and acknowledge possible misunderstanding of our manuscript.

We would like to thank Mr Shanmugan for his informative comments on our paper.

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*Corresponding author. Tel.: +82 2 760 3161; fax: +82 2 765 7117.
E-mail address: ytkim@snu.ac.kr (Y.T. Kim)

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Letter to the Editor

Surgery for ischemic mitral valve prolapse

A. Ruchan Akar*, Umit Ozyurda
Department of Cardiovascular Surgery, Heart Center, Ankara University School of Medicine, Dikimevi, 06340 Ankara, Turkey

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Keywords: Ischemic mitral regurgitation; Mitral valve repair

We read the original article by Jouan and colleagues [1] with great interest, highlighting the mechanisms of ischemic mitral valve prolapse and implications for mitral valve

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repair, which appeared in the December 2004 issue of EJCTS. The results of this retrospective observational study suggest that ischemic mitral valve prolapse (Carpentier type-II) represents one-third of the cases in their series. We wish to congratulate the authors on a very interesting paper; however, we feel that certain issues must be addressed.

Ischaemic mitral regurgitation (IMR) is a common complication of coronary artery disease caused by partial or complete obstruction of one or more coronary arteries [2]. By definition, patients with IMR have structurally normal valve leaflets and chordae but valvular incompetence usually occurs as a complication of regional or global LV dysfunction [3]. As described by Steven Bolling, 'IMR is a ventricular disease, not a valvular disease'. Exception to this might be the patients with chronic mitral valve prolapse without mitral regurgitation (a subgroup of patients with Barlow's disease), the most common valve abnormality involving approximately 3–5% of the adult population [3]. If the patients in this subgroup develop mitral regurgitation only after a myocardial infarction, they can be included in IMR series. Patients exhibit different pathological features, clinical presentation and outcome than those having MR of other aetiology. Taking this into account, it is essential that the authors demonstrate previous ischemic myocardial injury for all patients in their series. However, the authors reported no wall motion abnormality in 22.7% of their patients [1]. Furthermore, the mean ejection fraction in this patient population was $54.9 \pm 13.8\%$ and end diastolic left ventricular diameter was 60.9 ± 8.9 mm [1]. The authors also failed to mention the duration between the myocardial infarction and surgery for their series, although 34.1% were operated upon within 60 days following acute myocardial infarction. They did not investigate histological or ultra-structural analysis of mitral valve specimens in patients undergoing quadrangular resection, either. However, they overcame this limitation by discussing mechanisms of papillary muscle dysfunction after ischemic injury and detailed description of papillary muscle anatomy, which deserves attention.

Thus, some of Carpentier's geneous mitral valve repair techniques, including quadrangular resection and chordal transposition are questionable when applied to IMR. Better understanding of the complex inter-relationship of the obstructed coronary artery, left ventricular muscle and competency of the mitral valve would certainly help to develop new management strategies for IMR, which is a subject of intense debate. Certain mitral valve pathologies and concomitant coronary artery disease continue to create a dilemma for diagnosis of IMR and reporting outcome in this patient population.

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*Corresponding author. Tel.: +90 5055279680; fax: +90 3123625639.
E-mail address: rakar@medicine.ankara.edu.tr (A.R. Akar)

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Reply to the Letter to the Editor

Reply to Akar and Ozyurda

Christophe Acar*
Hopital Pitié-Salpétrière, 50-52 Bd Vincent Auriol, 75013 Paris, France

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Keywords: Mitral repair; Ischemic mitral insufficiency; Mitral prolapse

The aim of this article was to emphasize the complexity of the mechanism of ischemic mitral valve insufficiency, which is practically the sole situation in which the three functional Carpentier types can be encountered. This series underlines the difficulty in recognizing a leaflet prolapse when located at a commissure and the variability of the papillary muscle distribution leading to a vast panel of possible injuries. As opposed to Akar, we feel that the distinction between MR related to degenerative mitral valve insufficiency associated with a coronary disease and ischemic MR is often simple. Most patients with degenerative MR have a myxomatous involvement of the leaflets and chordae (Barlow) which can easily be recognized. In those cases with isolated chordal rupture, we believe that the triggering factor could have been the change in regional wall motion due to the acute ischemic process resulting in an untolerable load on leaflets and chordae (fibroelastic deficiency). Because the cause of MR was probably two-fold and because they raised common surgical issues, we chose to group all Carpentier type II non-myxomatous valves which ultimately represented one-third of all ischemic MR in this series.

The absence of regional wall motion abnormality in the basal conditions does not preclude the onset of regional ischemic MR at stress. Although stress test was seldom available here, we acknowledge that it could have been useful on occasional cases to strengthen the indication. An anatomic sample was rarely available for pathological study in this series (papillary muscle rupture, leaflet excision), however, on the whole we did not find that this test was of any informative value in most ischemic MR.

*Tel.: +33 1 42 16 56 85; fax: +33 1 42 16 56 78.
E-mail address: c.acar@psl.aphp.fr

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