be a blowout at the previous aortotomy site, because there was no evidence of aortic wall infection intraoperatively, and histologic examination and cultures taken from the pseudoaneurysm remained sterile.

The patient was discharged 4 weeks later. Heart and prosthetic valve function were normal, and computed tomography scan showed no abnormality (Figure 1). We think operation for ascending aortic pseudoaneurysms can be safely performed on femorofemoral bypass without hypothermic cardiac arrest.

Jiang Sheng-li, MD
Li Bo-jun, MD
Gao Chang-qing, MD
Department of Cardiovascular
Surgery of Chinese PLA General
Hospital
Chinese PLA Cardiac Surgery
Institute
Beijing, PR China

References

1. Emaminia A, Amirghofran AA, Shafa M, Moaref A, Javan R. Ascending aortic pseudoaneurysm after aortic valve replacement: watch the tip of the cardioplegia cannula! *J Thorac Cardiovasc Surg.* 2009;137:1285-6.
2. Sabri MN, Henry D, Wechsler AS, DiSciascio G, Vetrovec GW. Late complications involving the ascending aorta after cardiac surgery: recognition and management. *Am Heart J.* 1991;121:1779-83.
3. Sullivan KL, Steiner RM, Smullens SN, Griska L, Meister SG. Pseudoaneurysm of the ascending aorta following cardiac surgery. *Chest.* 1998;93:138-43.
4. Katsumata T, Moorjani N, Vaccari G, Westaby S. Mediastinal false aneurysm after thoracic aortic surgery. *Ann Thorac Surg.* 2000;70:547-52.
5. Apaydin AZ, Posacioglu H, Islamoglu F, Telli A. A practical tool to control bleeding during sternal reentry for pseudoaneurysm of the ascending aorta. *Ann Thorac Surg.* 2003;75:1037-8.

doi:10.1016/j.jtcvs.2010.07.100

ACUTE KIDNEY INJURY AFTER CORONARY ARTERY BYPASS GRAFTING: DOES RHABDOMYOLYSIS PLAY A ROLE?

To the Editor:

In the August 2010 edition of this *Journal*, Perioperative Management section, the following article was published: "Acute kidney injury after cor-

onary artery bypass grafting: Does rhabdomyolysis play a role?" by Benedetto et al.

The study has hypothesized that rhabdomyolysis may play a role in the development of acute kidney injury after coronary artery bypass grafting. However some important considerations have been ignored in the study. (1) The use of depolarizing muscle relaxant suxamethonium is not mentioned. Suxamethonium has been previously shown in literature as a potential cause of muscle injury and rhabdomyolysis. This is even more important when given to obese patients. (2) The use of antifibrinolytic medications aminocaproic acid (Amicar) and tranexamic acid (Cyclokapron) is also not mentioned. Several case reports have associated rhabdomyolysis with their concurrent use in cardiac surgery. (3) The use of the antiarrhythmic drug amiodarone has not been addressed. Amiodarone has also been associated with acute renal injury, especially when used along with statins. (4) All statins carry a risk of rhabdomyolysis. However, the Food and Drug Administration authority has said that the risk of rhabdomyolysis developing is more pronounced when simvastatin is combined with amiodarone than it is with other statin medications. The study has not provided the different statin medications the patients were receiving. (5) The study failed to mention whether the statin therapy was optimized before the operation. (6) The anesthetic technique used is totally ignored. Use of several inhalational agents could potentially increase the likelihood of renal injury developing, especially in combination with aforementioned drugs. Was total intravenous anesthesia technique used in any of the patients in the study? (7) No information was given about concurrent use of mannitol (especially during cardiopulmonary bypass).

It is very important to consider all possible causes of rhabdomyolysis that can occur during coronary artery

bypass grafting. I wish to know from Benedetto and associates the information regarding these questions.

Srikanth Ramarapu
Oklahoma University
Department of Anesthesiology
Oklahoma City, Okla

doi:10.1016/j.jtcvs.2010.09.060

Reply to the Editor:

We appreciate the interest in our work¹ and are pleased to address the aroused considerations.

First, we use cisatracurium, and not suxamethonium, as a muscle relaxant. We do not use aminocaproic acid or tranexamic acid, which is why these drugs are not mentioned in our article.¹ Amiodarone, which is known to play a role in rhabdomyolysis development,² was entered in the multivariable correction, but no statistically significant correlation was found ($P = .75$). Unfortunately, we could not retrieve information on the different types of statins that patients were taking because there were several and it was a retrospective study; however, we do know that atorvastatin, simvastatin, rosuvastatin, fluvastatin, and pravastatin were used. Nevertheless, consistent with the latest guidelines,³ statin therapy was optimized before surgery to achieve a low-density lipoprotein cholesterol level less than 100 mg/dL, and this was achieved in 85% of patients. Anesthetic induction was obtained by means of sevoflurane, and propofol was used as maintenance. We believe these agents do not increase the hazard of renal impairment. Finally, mannitol is routinely administered during cardiopulmonary bypass at a small dose of 200 to 250 mg.

Emiliano Angeloni, MD
Umberto Benedetto, MD
Riccardo Sinatra, MD
Department of Cardiac Surgery
University of Rome "La Sapienza"
Policlinico S. Andrea
Rome, Italy

References

1. Benedetto U, Angeloni E, Luciani R, Refice S, Stefanelli M, Comito C, et al. Acute kidney injury after coronary artery bypass grafting: does rhabdomyolysis play a role? *J Thorac Cardiovasc Surg.* 2010;140:464-70.
2. Borders-Hemphill V. Concurrent use of statins and amiodarone. *Consult Pharm.* 2009;24:372-9.
3. Grundy SM, Cleeman JJ, Merz CN, Brewer HB Jr, Clark LT, Hunninghake DB, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Circulation.* 2004;110:227-39.

doi:10.1016/j.jtcvs.2010.10.016

FUNCTIONAL MITRAL STENOSIS AFTER SURGICAL ANNULOPLASTY FOR ISCHEMIC MITRAL REGURGITATION: IMPORTANCE OF SUBVALVULAR TETHERING IN THE MECHANISM AND DYNAMIC DETERIORATION DURING EXERTION

To the Editor:

Kubota and associates¹ have presented detailed information of the interactions between mitral valve anatomy and function in the presence of mitral insufficiency caused by ischemic ventricular mechanism. They emphasize restriction of flow through the tethered mitral valve at leaflet edge level, which is aggravated by the presence of annuloplasty. The possibility of further aggravation by exercise is well discussed. I find that observations made by me and my colleague, Dr Edward Yellin,²⁻⁴ between 1959 and 2009 provide support for the excellent work described by Kubota and associates.

The normal valve transmits normal flow with a minimal gradient with less than full possible opening. When the normal diastolic annular expansion is fixed by annuloplasty, there is always a small gradient and a reduction of the mitral area to less than the annular area. However, as Kubota and associates found, the echo-derived mitral areas are much less than the geometric area of the annuloplasty rings used. They attribute this "functional mitral

stenosis" to leaflet tethering at the free edge with a reduced separation of the central anterior and posterior leaflet tips. A paper presented at the July 2009 meeting of the Society for Heart Valve Disease in Berlin (Frater, Papillary Displacement: Effect on Chordae and Leaflets) supports and confirms this opinion.

This was an in vitro anatomic observation of the effects of papillary displacement on the anterior and posterior leaflets in 6 pig and 3 human hearts with fixed reducing annuloplasties. In normal diastole the papillary muscles move apically, posteriorly, and apart.⁵ These are also the papillary displacements seen in myopathic ventricular enlargement. Apical displacement by itself puts the second-order chordae under tension, leaving the free edge first-order chordae loose, and allows the leaflet to retain flexibility. Separation of the papillary muscles, by contrast, puts both the second-order and the free edge first-order chordae under tension. Tautness of the free-edge chordae reduces all flexibility of the major parts of the anterior leaflet and the central scallop, rendering them resistant to opening by transvalvular flow. Whereas the normal separation of papillary muscles is 1 cm in systole and 2 cm in diastole, in a ventricle with a 65-mm diastolic dimension, the papillary separation may be 4 cm. The anterior leaflet is a taut sheet pointing posteriorly and the stiffened posterior scallops are parallel with the ventricular wall. The findings of this in vitro study provide clear confirmation of the mechanism of mitral stenosis at free edge level postulated by Kubota and associates.

Robert W. M. Frater, MD
Montefiore Medical Center
Department of Cardiothoracic
Surgery
Bronx, NY

References

1. Kubota K, Otsuji Y, Ueno T, Koriyama C, Levine RA, Sakata R, et al. Functional mitral stenosis after surgical annuloplasty for ischemic mitral regur-

itation: importance of subvalvular tethering in the mechanism and dynamic deterioration during exertion. *J Thorac Cardiovasc Surg.* 2010;140:617-23.

2. Frater RWM. Mitral valve anatomy and prosthetic valve design. *Proc Staff Meet Mayo Clin.* 1961;36:582-92.
3. Yellin EL, Peskin C, Yoran C, Koenigsberg M, Matsumoto M, Laniado S, et al. Mechanism of mitral valve motion during diastole. *Am J Physiol.* 1980;241(Heart Circ. Physiol 10):H389-400.
4. Yellin EL, Nikolic S, Frater RWM. Left ventricular filling dynamics and diastolic function. *Prog Cardiovasc Dis.* 1990;32:247-71.
5. Babburi H, Oommen R, Brofferio A, Ilcail A, Frater RWM, Shirani J. Functional anatomy of the normal mitral apparatus: a transthoracic two-dimensional echocardiographic study. *J Heart Valve Dis.* 2003;12:180-5.

doi:10.1016/j.jtcvs.2010.10.017

Reply to the Editor:

We appreciate the comments of Dr Robert Frater on our recent manuscript¹ and are honored to reply to them. He has been a pioneer in recognizing that mitral leaflet diastolic opening, not only systolic closure, is controlled by the chordal apparatus.^{2,3} He and his colleagues observed only limited influence from changes in mitral inflow volume on mitral leaflet opening excursion.³ They further observed that papillary muscle rupture clearly increases mitral leaflet opening, indicating limited leaflet opening by chordal attachments.³ Therefore, they demonstrated flow-independent opening of the mitral leaflets as well as diastolic restriction or tethering of mitral leaflet opening by chordal attachments and tension, subsequently confirmed in patients by our group.⁴

In dilated ventricles, papillary muscle displacement causes greater chordal tension and restricts leaflet mobility in both systole and diastole. It leads to mitral regurgitation in systole and reduced leaflet opening in diastole.^{4,5} Therefore, subvalvular tethering by papillary muscle displacement is the central pathophysiology restricting mitral leaflet mobility and function. Of note is that ventricular dilatation alone causes significant mitral regurgitation whereas resultant mitral stenosis is