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

only modest. After surgical annuloplasty to reduce annular size, significant mitral stenosis develops beyond annular size reduction by limited leaflet opening with diastolic tethering.^{1,6} Dr Frater's previous investigations, demonstrating the presence of leaflet tethering in diastole,³ provide conceptual support and explanation for the development of functional mitral stenosis after surgical annuloplasty for ischemic mitral regurgitation.^{1,6} These concepts have clear therapeutic implications regarding the need to address subvalvular tethering for repair of both systolic and diastolic dysfunction of the mitral apparatus.

The observation in Dr Frater's letter regarding the heterogeneous influences on diastolic leaflet mobility from papillary muscle displacement is of special interest. He observed that papillary muscle separation with its lateral and posterior displacement causes greater impairment on diastolic mitral leaflet opening than does papillary muscle apical displacement. Although we have not performed 3-dimensional analysis of papillary muscle position, we agree with his observation, because papillary muscle tip position by echocardiography seems to be displaced laterally and posteriorly but not apically in many patients. We referred to "outward displacement" as "lateral and posterior displacement" in our publications.^{1,5} We expect that "outward" or "lateral and posterior" displacement of papillary muscle will cause tensed and posteriorly displaced chordae to limit anterior leaflet opening, leading to functional mitral stenosis in diastole, whereas apical displacement may not displace chordae posteriorly and resultant effects to restrict leaflet opening may be only modest. The direction and degree of papillary muscle displacement are key factors to address subvalvular tethering and provide comprehensive surgical relief for ischemic mitral regurgitation and/or stenosis. We look forward to Dr Frater's publication of his

research on this topic to stimulate further investigations and clinical improvements.

Yutaka Otsuji, MD^a

Ryuzo Sakata, MD^b

Kayoko Kubota, MD^c

Masaaki Takeuchi, MD^a

Robert A. Levine, MD^d

Chuwa Tei, MD^c

^aUniversity of Occupational and
Environmental Health

Kitakyushu, Japan

^bKyoto University

Kyoto, Japan

^cKagoshima University

Kagoshima, Japan

^dMassachusetts General Hospital
Boston, Mass

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 regurgitation: 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. Otsuji Y, Gilon D, Jiang L, He S, Leavitt M, Roy MJ, et al. Restricted diastolic opening of the mitral leaflets in patients with left ventricular dysfunction: evidence for increased valve tethering. *J Am Coll Cardiol.* 1998;32:398-404.
5. Levine RA, Otsuji Y, Schwammenthal E, Sakata R, Tei C. Ischemic mitral regurgitation: from new understanding to new solutions role of echocardiography. *J Echocardiogr.* 2006;4:1-18.
6. Magne J, Sénéchal M, Mathieu P, Dumesnil JG, Dagenais F, Pibarot P. Restrictive annuloplasty for ischemic mitral regurgitation may induce functional mitral stenosis. *J Am Coll Cardiol.* 2008; 51:1692-701.

doi:10.1016/j.jtcvs.2010.10.018

A SELF-INTRODUCED SHARP METALLIC FOREIGN BODY MIGRATING TRANSCUTANEOUSLY TO THE INTERVENTRICULAR SEPTUM, MISTAKEN AS AN INHALED PIN To the Editor:

I enjoyed reviewing the article on cardiothoracic imaging titled "Sew-

ing needles embedded in the cardiac interventricular septum and chest wall."¹

This case reminds me of a 17-year-old girl admitted to our center 4 years ago with a chest x-ray film suggestive of an inhaled pin or sewing needle. A routine quick history was taken by a junior staff member, and an examination was done with a rigid bronchoscope, but no foreign body was found. Emergency left thoracotomy revealed a small hematoma on the surface of the pericardium with no significant effusion or foreign body. On fluoroscopy, the foreign body was still in place but its exact relationship to the cardiac structures could not be delineated. An urgent echocardiogram and computed tomographic chest scan with intravenous contrast localized the foreign body in the interventricular septum. An open heart operation was performed and the foreign body was removed from the septum through a right atriotomy. Postoperatively, detailed history-taking showed that the patient was confused and was being treated for major depression. She confessed that she had stabbed herself in the chest with a syringe needle to commit suicide.

From the teaching point of view, this case raises some important points:

1. A full thorough and detailed history should be taken from the patient and/or the family.
2. Inhaled foreign bodies can be mistaken for swallowed and or self-inflicted foreign bodies.
3. A chest x-ray film with the upper half of the body unclothed is recommended.²

The heart may be severely damaged by objects of unimpressive size or appearance. Pins and needles can injure the heart, resulting in life-threatening cardiac tamponade.^{3,4}

Perrotta, Perrotta, and Lentini,⁵ in their best evidence medicine article, stated that early diagnosed needle injury to the heart should be treated surgically, regardless of the presence of