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the opposite results since the distal artery is likely hypoperfused from obstruction of the distal axillary artery itself by the cannula.

Last, the others favor a left axillary approach. Through our experience in more than 80 cases of axillary cannulation, particularly in cases of type A dissection or the need for elective cerebral protection, we favor the right side. Contrary to their opinion, we believe the right-sided approach anatomically allows a very efficient means for cerebral protection since no additional cannulation is needed and the perfusion is simply lowered to the calculated rate.² Brain pressure monitoring during circulatory arrest time is realized through the right radial line. We agree with Neri and colleagues to avoid manipulation of the common carotid and the innominate ostia. These are never clamped at the time of circulatory arrest until they have been visualized directly or have been seen previously through epiaortic echocardiographic reading.³ The significant flow obtained with excellent retrograde washing effect through the common carotid, innominate, and even left subclavian arteries through the vertebral-basilar system allows excellent washout effect against any embolic debris, particularly in the case of a significantly atherosclerotic aorta or aneurysm.

Our own experience involves 17 patients with type A dissection over a period of 3 years who were treated with perfusion through a graft interposition on the right axillary artery, 16 having cerebral perfusion through the same graft at the time of circulatory arrest. The mean age was 67.7 years and there were 8 women and 9 men. Mean extracorporeal circulation time was 266.2 minutes, with a mean crossclamp time of 80 minutes and a mean circulatory arrest time of 52 minutes. Sixteen of those 17 patients had antegrade cerebral perfusion, and 1 patient (left axillary approach because of pulseless right upper extremity) had retrograde cerebral protection. All had replacement of the ascending aorta, 5 having an aortic valve replacement and 3 having an arch replacement as well. Ten patients were treated on an emergency basis, with 2 being in shock on admission. Results showed 1 operative stroke with no delayed stroke after the operation and 5 deaths. Average length of stay was 28.3 days.

In summary, we congratulate the authors for their excellent results and agree with the targeted site for cannulation in aortic dissection. We still favor the graft interposition technique, which allows easy closure of the artery and makes safe a more medial approach to the artery, thus avoiding the cervical nerve roots. The additional benefit of superior antegrade brain protection, without any additional cannulation, makes us favor the right-sided approach as well.

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Cinefluoroscopic assessment of human mitral anulus after mitral valvuloplasty

To the Editor:

I read with great interest the article "Mitral Annular Size and Shape in Sheep With Annuloplasty Rings" by Glasson and associates.¹ I am very much impressed with their complete analysis. The mitral anulus is a complex structure. Complexity derives from its composition, its geometric relationships, and its pathophysiology. The literature contains ample evidence about the maintained movements of the mitral anulus even after annuloplasty.

Okada and associates² have shown that the motion of the mitral anulus during the cardiac cycle after mitral valve repair for chronic mitral regurgitation due to degenerative disease is affected by the type of annular device used. Of most importance, however, is that the diastolic blood flow across the mitral orifice during exercise was better in patients in whom a flexible ring was used. Mitral annuloplasty has always been a target for the cardiac surgeon. Pathophysiologic findings prompted many authors to develop flexible rings to avoid rigid fixation of the mitral anulus.

I would like to report the experience of my colleagues and me,³ which is similar to that of the authors (as a method but not as a conclusion). In 1991 we experimented with a new technique using autologous pericardium to obtain a flexible ring that could preserve the physiologic mitral annular motion after valvuloplasty. In a subgroup of patients (n = 20)who underwent mitral valvuloplasty because of degenerative disease, a long strip of pericardium was prepared, marked with a metal clip, and rolled up in a tubular fashion with the serosal surface on the outside. The pericardial tube was apposed on the posterior anulus just beyond the commissures. Postoperative Doppler echocardiographic analysis showed nearly normal transmitral flow indexes (flow velocity peak: 1.06 ± 0.2 ; P = no significant difference from normal indexes). Cinefluoroscopic examination was used for assessing annular motion with the metal clips used as radiopaque markers. Planimetry of the hemi-area showed a narrowing of annular size during ventricular systole (mean $8.5\% \pm 6.4\%$). Even long-term results seems to be good.⁴ These findings, as corroborated by others authors, demonstrate that the flexible properties of the mitral orifice are preserved equally well after this type of annuloplasty in man.

The aim of this letter is not to criticize. However, I would suggest that the authors' "surprising results" must be considered with extreme caution and additional studies should be required to establish the overall effectiveness of their method both in the animal model and in human beings.

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Reply to the Editor:

Dr Scrofani and his colleagues in Milan are to be congratulated for carrying out these fluoroscopic studies of implanted radiopaque mitral annular markers in human subjects. Scrofani's letter alludes to the radiographic results in 20 patients who had a few, relatively large (3 mm) metallic markers inserted into a posterior mitral annuloplasty tube of autologous pericardium, which were published in 1991¹ and 1996.² The Institutional Review Board at Ospedale "L. Sacco" and these courageous investigators deserve credit for conducting the only human investigation that I am aware of which employed intracardiac radiopaque markers; it is notable that no adverse complications of the research study were noted. When we began our myocardial marker studies in human patients in the 1970s, we used only external or left ventricular mid-wall myocardial markers. Our more recent investigations of the mitral ventricular-valvular complex, which now include upward of 10 miniature gold markers inserted in the mitral leaflets per se in addition to the 8 annular markers, have all been performed in animal models, including the article concerning the mitral annular ring in sheep, which prompted Scrofani's letter.³

Both similarities and differences between the normal sheep and the human postmitral repair studies are noteworthy, including the timing and amplitude of annular motion. This Italian group attempted to reproduce in human beings the seminal pioneering work performed by Tsakiris and colleagues⁴⁻⁶ in dogs at the Mayo Clinic in the 1960s. Importantly, only single-plane fluoroscopy was used, as was the case in Tsakiris's experiments, which means that cardiac motion into and out of the plane of reference and other internal motion (translation and rotation) were not detectable. This implies that the observed motion could possibly have not been true internal cardiac deformation, but corrupted by artifact resulting from cardiac translation or rotation. The high speed, simultaneous biplane cinefluoroscopic techniques that we use eliminate such ambiguity, because the true 3-dimensional coordinates of each marker are identified every 17 ms with respect to a fixed, external laboratory reference system, with an accuracy and reproducibility of less than 1 mm.

Scrofani and associates noted that fractional area change of the posterior mitral "hemiarea" during the cardiac cycle was small (8.5% \pm 6%) after a partial posterior pericardial annuloplasty; importantly, this is not dissimilar to the total mitral annular area change of $11\% \pm 2\%$ reported in our study in control sheep.³ On the other hand, we found no mitral area change after implantation of either a complete Duran ring (Medtronic, Inc, Minneapolis, Minn) or a Carpentier-Edwards Physio complete annuloplasty ring (Baxter Healthcare Corp, Irvine, Calif).³ Given the marked differences in annuloplasty methods employed, analytical techniques used, the experimental conditions, and the species, there is little point in debating which measurement is more correct than the other. Much more noteworthy is that the Italian investigators observed a much smaller magnitude of dynamic motion of the human annular orifice after mitral repair than did the Japanese team of Okada and associates,7 who used extrapolated 2-dimensional echocardiographic imaging to make their measurements. Okada's group reported mitral fractional area reduction of 26% \pm 4% in patients who had received a Duran flexible annuloplasty ring,7 which was similar to that reported echocardiographically in normal human beings by Ormiston and coworkers8 in 1981. Again, these echocardiographic methods cannot differentiate true internal cardiac deformations from rotation and/or translation, cannot track the motion of discrete cardiac foci, and are limited in terms of temporal resolution, similar to the limitations inherent in contrast left ventriculography, nuclear multigated acquisition scanning, and magnetic resonance imaging (without radiofrequency tagging of discrete structural elements). Indeed, the advent of 3-dimensional echocardiography may even have complicated this issue more, as a recent abstract from the Cleveland Clinic Foundation reported mitral area changes of $25\% \pm 10\%$ in normal human beings and $28\% \pm 11\%$ in those who had received a Cosgrove-Edwards annuloplasty band (Baxter Healthcare Corp, Irvine, Calif) during mitral repair.9 These large fractional mitral area changes were associated with calculated end-diastolic mitral annular areas of 10.1 \pm 3.9 cm^2 in normal subjects and $11.5 \pm 2.7 \text{ cm}^2$ in patients who had an annuloplasty, clearly much larger than one would expect. Indeed, the normal mitral area is only 4 to 6 cm² as calculated from the Gorlin formula and, pathologically, measures only 6 to 8 cm²; therefore, these 3-dimensional echo estimates of mitral area actually are supraphysiologic. This leads one to question what the 3-dimensional echo techniques are actually measuring. We all have been dazzled by beautiful 3-dimensional echo images of mitral valve leaflets opening and closing and wire-frame reconstructions of other cardiac structures, but it must be remembered that quantitative 3-dimensional echo measurements of discrete cardiac dimensions and dynamics have not yet been validated. Comparison of 3-dimensional marker data obtained from simultaneous