

dissections is attractive in selected cases (adverse anatomy, small vessels, type A-B dissections). However, we believe that only properly designed studies will be able to determine whether this strategy is superior to stenting in most patients experiencing nonocclusive dissections. In the interim, accepting the potential risk of vessel closure and the logistic implications (prolonged observation or even repeat angiography) inherently associated with the conservative strategy should be weighted against the results of coronary stenting using currently available stent designs. Although we sympathize with the words of caution against the indiscriminate use of stents, it would appear more reasonable to challenge first the systematic use of “elective” stenting in clinical/angiographic settings where its efficacy—as compared with PTCA—remains largely unsettled.

#### Fernando Alfonso, MD, PhD, FESC

Interventional Cardiology Department  
San Carlos University Hospital  
Madrid, Spain

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#### REFERENCES

1. Cappelletti A, Margonato A, Rosano G, et al. Short- and long-term evolution of unstented nonocclusive coronary dissection after coronary angioplasty. *J Am Coll Cardiol* 1999;34:1484-8.
2. Leingruber PP, Roubin GS, Anderson HV, et al. Influence of intimal dissections on restenosis after successful coronary angioplasty. *Circulation* 1985;72:530-50.
3. Ellis SG, Roubin GS, King SB, Douglas JS, Cox WR. Importance of stenosis morphology in the estimation of restenosis risk after elective percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1989;63:30-4.
4. Ozaki Y, Violaris AG, Kobayashi T, et al. Comparison of coronary luminal quantification obtained from intracoronary ultrasound and both geometric and videodensitometric quantitative angiography before and after balloon angioplasty and atherectomy. *Circulation* 1997;96:491-9.
5. Alfonso F, Hernandez R, Goicolea J, et al. Coronary stenting for acute coronary dissection after coronary angioplasty: implications of residual dissection. *J Am Coll Cardiol* 1994;24:989-95.

#### REPLY

Dr. Alfonso asks why two patients in our study (1) with occlusive dissection after percutaneous transluminal coronary angioplasty (PTCA) were excluded and when these dissections occurred. As it is clearly stated in the article these two type E dissections evolved toward complete artery occlusion during the procedure and how they caused an acute myocardial infarction immediately after the procedure. Because the study reported the results of nonocclusive unstented dissections, they were excluded from the analysis at the beginning.

As far as the second point is concerned, we have acknowledged the higher prevalence of lesions A and B in the unstented group, but this limitation derives from the later stage in which the stented patients were assessed, when the easy availability of stenting allowed higher inflation pressures. However, although unstented patients had a higher prevalence of dissections grades A and B (namely 85% vs. 56% at 24 h), the restenosis rate for stented and unstented patients was similar for each dissection grade ( $p = NS$ ).

What we would like to stress in our study is that in this stenting era, where there is a growing and widespread use of these devices (2), the “minor” dissections (type A and B), most frequently

occurring during PTCA, are associated with a very low risk of complications and restenosis, suggesting a more conservative approach.

Finally, Dr. Alfonso states that “the large lumen diameter of the dissected segments indicates that the dissection image was fully included into the lumen measurements.” However, as clearly shown in Table 1 of our article, the mean lumen diameter post-PTCA in dissected vessels was not  $3.23 \pm 0.65$  mm but  $3.11 \pm 0.89$  mm, a lower value than that of the mean reference artery diameter pre-PTCA ( $3.18 \pm 0.7$  mm) in the same vessels. We do agree that the methodology of quantitative coronary angiography is technically demanding, especially for the analysis of dissected segments. Therefore, we are promoting in our Institute new and different tools for quantitative analysis, such as intracoronary ultrasound (IVUS), coronary Doppler evaluation, and myocardial fractional flow-reserve measurement.

#### Alberto Cappelletti, MD

#### Alberto Margonato, MD

Division of Cardiology  
Istituto Scientifico H.S. Raffaele  
Milan, Italy

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#### REFERENCES

1. Cappelletti A, Margonato A, Rosano G, et al. Short- and long-term evaluation of unstented nonocclusive coronary dissection after coronary angioplasty. *J Am Coll Cardiol* 1999;34:1484-8.
2. Rankin JM, Spinelli JJ, Carere RG, et al. Improved clinical outcome after widespread use of coronary artery stenting in Canada. *N Engl J Med* 1999;341:1957-65.

### Coenzyme Q10 as an Adjunctive Therapy in Patients With Congestive Heart Failure

Lack of effect from treatment with coenzyme Q10 in congestive heart failure is not an objective title or conclusion for the study by Watson et al. (1) in which the main limitation obviously is their sample size and its lack of study patients. Even so, the investigators state in their introduction that previous studies with coenzyme Q10 “lack credibility because of small sample sizes, lack of controls, etc.”

The majority of the 27 study patients, who were not classified according to the New York Heart Association (NYHA), were seemingly at late-stage disease (mean length of symptoms 3.4 years). Mean patient age was 55 years, which is compatible with predominantly ischemic origin. This was also recently confirmed at an International Conference in Sydney, Australia—“Oxidative Pathways in Health and Disease”—in a lecture by one of the co-authors, Nicholas Bett (2). However, according to the Watson et al. (1) study, in the Patients’ Demographics in Table 1, 77% of the patients were listed as having dilated cardiomyopathy. This is a patient clientele that is, at least partially, prone to respond either spontaneously or to medical intervention with subsequent improvement of myocardial function.

Conversely, it is well-known that changes—and not least improvements—in echocardiographic parameters of left ventricular (LV) function are minimal in late-stage disease, especially in heart failure due to ischemic heart disease. This is why the