Intravascular ultrasound guided PTCA: a way to escape stent mania?

See page 137 for the article to which this Editorial refers

In 1995, a total of 278,982 percutaneous coronary interventions were carried out in Europe alone[1]. In the United States, the corresponding figure for the same period was approximately 500,000, and these figures continue to increase worldwide, year after year. To date, plain old balloon angioplasty remains the strategy of choice in the majority of cases, even though a myriad of different devices have been conceived, developed and tested in percutaneous coronary interventions. However, up to now, no other single device has seen the same spectacular explosion on the market as the stent, and none has managed to oust the angioplasty balloon from its position at the top of the polls.

Introduced in 1986, stents spread relatively slowly until the publication of large multicentre trials demonstrating the efficacy of this device in the prevention of restenosis[2,3]. Since then, stents have spread like wildfire, and their diffusion has been all the more encouraged by improvements in the prevention of subacute occlusions. In 1997, the average rate of stenting in Europe was 51%, with figures ranging from 25% in Eastern Europe to more than 60% in Western European nations[4]. Since this report was compiled, the rate of stenting has continued to grow, and is probably around 70% now.

The most common indications for stenting are abrupt vessel closure, suboptimal result defined by residual stenosis >30%, coronary dissection of grade C or higher and treatment of restenosis on a native artery, not to mention the very broad indications for stenting in venous bypass grafts. Without a doubt, stenting makes it possible to control abrupt occlusions and to optimize the result of the conventional angioplasty procedure. In short, the stent has made angioplasty safer and easier, with improved immediate results. However, it is worth noting that all these indications for stenting are based on observational studies only. The only indication in which the efficacy of stents has been proven is still complied with relatively rarely, namely the prevention of restenosis in STRESS-BENESTENT-type lesions, i.e. in lesions less than 15 mm in length, and in arteries with a diameter >2.8 mm and <3.5 mm[4].

Furthermore, it would appear that stenting is becoming systematic in certain centres, irrespective of vessel size — large or small, irrespective of the final result after conventional angioplasty, and the aspect of the lesion site — dissected or not, dissection with limited or non-limited flow. Direct stenting is the other representative example of growing stent-mania. This consists in deliberate stent deployment as the primary treatment, without previous dilation of the vessel, in the hope of reducing both the risk of vessel trauma and of dissection.

All in all, the attitude of extensive stenting prevails today despite the fact that it has never been demonstrated that stenting reduces immediate complications after angioplasty[2,3]. In particular, stents have never been shown to reduce the rate of post-procedure minor enzyme release, the prognosis of which is uncertain[4]. Widespread stenting persists, despite the considerable cost induced by their mass use, and despite the risk of intra-stent restenosis, which is estimated to be around 25%, and for which no satisfactory solution has yet been found[6].

In this context, the article by Schroeder et al.[7] in this issue, in which they analyse the immediate and long-term evolution of arterial dissections after intravascular ultrasound guided PTCA, provides us with an alternative view of angioplasty in the age where the stent is king. Does their view focus on the past, or look to the future? One must not forget the limitations of this paper, which presents the experience of a single centre in a non-randomized study. Intravascular ultrasound represents a major advance in coronary imaging because it allows a very precise picture of the exact size of the lumen, and also of the vessel structure, plaque burden and presence of calcification. So far, intravascular ultrasound imaging has been principally used to optimize stent deployment[8–10], and to demonstrate the reality of arterial remodelling post-angioplasty[11]. It has now become an indispensable tool for the analysis of the intimal mechanisms of endoluminal coronary interventions.
Moreover, recent studies would tend to prove that intravascular ultrasound allows for a physiological approach to coronary flow.[12] Furthermore, Abizaid et al. have shown that a stenosis is not haemodynamically significant when the cross-sectional area of the narrowing is >4 mm².[13] Using intravascular ultrasound to guide angioplasty offers the possibility of optimizing balloon size by adapting it to the real diameter of the artery as measured from external elastic lamina to external elastic lamina, and not according to the vascular lumen size as is the case in quantitative angiography. This results in a balloon/artery ratio considerably greater than 1, and is probably one of the reasons why residual stenosis of <30% at the end of the procedure, a so-called ‘stent like result’ can be more frequently obtained with intravascular ultrasound guided PTCA than in traditional angiography guided PTCA. Contrary to previous reports stating that oversizing resulted in more frequent complications, the rate of major adverse cardiac events in this report does not seem to be increased compared to conventional balloon angioplasty without oversizing.[14] Finally, intravascular ultrasound also allows for a close analysis of dissection, and shows that these latter are in fact very common, occurring in more than two-thirds of all cases of angioplasty. This confirms that dissections are an integral part of the mechanism of action of angioplasty.

The article by Schroeder et al.[7] shows above all that the evolution of non-stented arterial dissections, provided they are not flow-limiting, is favourable in the majority of cases, with the rate of major adverse cardiac events in this series not exceeding that attained in other series with extensive stenting. This result seems to be achievable thanks to significant over-sizing of the balloon compared to the angiography-determined lumen size, resulting in major enlargement of lumen size so that arterial dissections do not compromise the flow. The final result is that the rate of the stenting in this series will probably be considered by most interventional cardiologists as being ridiculously low, at less than 5%. In addition, the long-term evolution indicates that a low restenosis rate can be achieved without the use of stents. Of course, to be validated, these results need to be confirmed by further studies, and reproduced by other investigators.

All in all, intravascular ultrasound is one of the tools developed for and by angioplasty, but which did not experience the same widespread use as some of the other devices at the disposal of interventional cardiologists. This lack of popularity is most likely due to the relatively high price of the intravascular ultrasound consoles which are necessary to operate the probes, and of course, the price of the actual intravascular ultrasound catheter itself, even though this latter is still considerably cheaper than a stent. In addition, the use of intravascular ultrasound increases the complexity of the procedure, whereas stent implantation results in over-simplification of the procedure, and increased comfort for the physician, explaining the relative lack of interest by interventional cardiologists in this imaging technique.

In any case, this article clearly shows that angioplasty carried out in a different way, with the choice of balloon size guided by accurate intravascular ultrasound measurement of vessel size, makes it possible to achieve results which compare favourably with systematic stenting. The time has come to carry out further, randomized studies in this area. Some are already ongoing in Europe. It remains to be seen whether it is possible to escape from the ‘pensée unique’ that coronary angioplasty must systematically involve stent implantation.

J.-P. BASSAND
University Hospital Saint-Jacques, Besançon, France

References

Diagnosing primary diastolic heart failure

With the increasing refinement of methods to uncover early phases of cardiac failure, we have, over the past two decades, witnessed the emergence of diastolic dysfunction and diastolic failure of the heart as separate, widely recognized clinical entities. Whereas the majority of the conditions related to diastolic dysfunction and failure are the mere consequence of systolic cardiac failure, there also exists a distinct primary form of diastolic failure. Primary diastolic failure has been commonly defined as a condition with classic findings of congestive heart failure with normal ventricular systolic function, but with predominantly diastolic dysfunction. It has been observed in a large variety of clinical conditions and was believed to occur more commonly—at least in the elderly population—than previously thought, accounting for about 30% to 40% of all patients with congestive heart failure.

In an excellent review, Vasan et al.\(^\text{[1]}\) surveyed 31 studies on diastolic failure published in the period January 1970–March 1995. From their critical analysis, the authors were astonished to find that the prevalence of primary diastolic heart failure, i.e. patients with congestive heart failure and normal ventricular systolic performance, varied widely from 13% to 74%. Despite the many possible causes, interpretations and warnings suggested by these authors, it is surprising that their conclusions were not taken more seriously. Similar criticisms were recently raised by Caruana et al.\(^\text{[2]}\). In a Letter to the Editor in the European Heart Journal (20/5), Caruana et al. responded to a report entitled ‘How to diagnose diastolic heart failure’ by the European Study Group on Diastolic Heart Failure\(^\text{[3]}\). In the Working Group’s Report, it was stated that a diagnosis of primary diastolic heart failure requires three obligatory conditions to be satisfied simultaneously: (1) presence of signs or symptoms of congestive heart failure; (2) presence of normal or only mildly abnormal left ventricular systolic function; (3) evidence of abnormal left ventricular relaxation, filling, diastolic distensibility or diastolic stiffness. Using echocardiographic examination in patients with dyspnoea but no apparent left ventricular systolic dysfunction, Caruana et al. observed a prevalence of primary diastolic dysfunction of 3–5% when using an E/A ratio in association with deceleration time, but of 27% if isovolumic ventricular relaxation time was used. There was poor overlap between subjects found to be ‘abnormal’ by each of the two different criteria, with only 2–3% when both indices were combined. From this, the authors concluded (i) that different measures of diastolic dysfunction give different prevalences of primary diastolic failure, and (ii) that there is no simple echocardiographic means of reliably diagnosing diastolic dysfunction.

As previously stated by Vasan et al.\(^\text{[1]}\), only two reasons could possibly account for this somewhat absurd wide variation in clinical prevalence of primary diastolic heart failure. Either there is no agreement of what should be considered as normal-systolic function and how it should be measured, or there is no clear, generally agreed definition of diastolic dysfunction or failure. Moreover, as many conditions may clinically resemble primary diastolic failure, one should first exclude all non-cardiac causes.

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