Every piece is important to build a puzzle. What can we learn from early experiences with balloon PTCA?

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Percutaneous transluminal coronary angioplasty (PTCA) was introduced into clinical practice more than 20 years ago, for the treatment of very selected anatomical and clinical situations. The procedure quickly became accepted as an alternative to the medical and surgical treatment of coronary artery disease. Progress made over the years has been tremendous and was unimaginable when PTCA was first used. Progress has also been quick, in spite of the reduced number of prospective randomized clinical trials comparing PTCA to medical or surgical treatment, for single or multivessel disease, with or without stents. Guidelines have been written, but they soon become, like clinical trials, outdated, because they might not represent the ‘state of the art’ of existing treatment modalities.

In order for the clinician to decide what is best for his patient, every single publication is important. We should not forget, however, that all available treatments for coronary artery disease are palliative, and that the decision to revascularize, often made on the spot, should aim first to improve symptoms and quality of life and, ultimately, improve prognosis, by altering the natural history of the disease.

The paper by van Domburg et al.[1], reports on the longest follow-up (17 years), ever published, of balloon angioplasty performed in a single reputed centre. The data probably represent the worst possible scenario after PTCA, if we compare the conditions available in 1980–1985 with those existing today. It could also be considered as the natural history of balloon PTCA, if we ignore the fact that this type of revascularization was being applied to coronary artery disease patients.

What can we learn from this early experience with PTCA? Several aspects regarding the natural history of coronary artery disease, when the first option of revascularization was balloon angioplasty, can be discussed. The population selected at the time had a mean age of 56 years (6% more than 70 years), 47% had unstable angina or acute coronary syndromes and patients had predominantly single vessel disease (37% with 2–3 vessels disease) and good ventricular function (17% with an ejection fraction <50%).

Information that came to the fore initially was that the four most important independent predictors of long-term mortality were advanced age (>60 years), diabetes, multivessel disease and impaired left ventricular function (ejection fraction <55%). Mortality at 17 years was 42% and event-free survival 19%. There was an impressive difference between survival in single-vessel disease with good ventricular function (75%) compared to multivessel disease with impaired function at 15 years (28%). Diabetics had a worse outcome with survival curves diverging in the first 7 years and a 15% benefit for non-diabetics. The authors, however, in selecting a group of patients (26% of the total population) aged less than 60 years who were, non-diabetic, with single vessel disease and good ventricular function, found the prognosis to be similar to the general Dutch population.

The second piece of important information is that, although 59% of patients required reinterventions (34% repeat PTCA, 42% surgery, 24% PTCA + surgery), 32% of the new revascularizations occurred within the first year. Afterwards, the annual coronary reintervention incidence was 2–3% and stable, beyond the 10 years. Of the 87% of patients who do not require a reintervention in the first year, only 24% had further interventions in the follow-up.

Finally, and somewhat surprisingly, is the fact that the prognosis of patients with failed PTCA (59% survival at 15 years) is similar to those in whom it succeeds (64%). The lack of further information in the paper would make any discussion speculative.

Increased age is expected to have a strong impact on survival, the bigger the group of patients followed, particularly as a result of co-morbidities and ageing. Multivessel disease and impaired left ventricular function helped to favour coronary surgery in the randomized trials of the 1970s, comparing surgery to medical treatment. The risk of future events should increase with the extent of atherosclerosis present at the time of revascularization, particularly if we do not control adequately the ongoing atherosclerosis process. In the paper of van Domburg et al.[1], cholesterol-lowering drugs were given in only 18% of patients at hospital discharge. Reduced ventricular function, the consequence of previous infarctions or the extent of the disease, has been always recognized as important for survival. Diabetes, however, only recently became an important marker of accelerated atherosclerosis with implications for the choice of revascularization type. In the study[1], only 12% of patients were diabetic, but the finding that diabetes...
was an important predictor of mortality only confirms present knowledge.

Regarding the revascularization option, it is important to see confirmation of the importance of the first year after balloon angioplasty. Further revascularizations, by either form, run steadily after this first year, with a very low incidence of 2–3%, corresponding to the expected disease progression. Assuming that we could control the restenotic process, we should improve long-term survival.

As mentioned above, the current practice of PTCA is completely different from 15–20 years ago. In a recent paper by Williams et al. [2] from the National Heart, Lung, and Blood Institute, comparing the 1985–1986 Registry with the 1997–1998 Dynamic Registry, patients are now older and more often female, angioplasty is performed more often for acute myocardial infarction, for more severe lesions with thrombus and calcium, and stents are used in 71% of cases. Interestingly, although most patients had multivessel disease, only one lesion was attempted in two-thirds of patients, which means the maintenance of a rather conservative approach. Procedural success is now higher, with lower combined rates of hospital death, myocardial infarction and emergency surgery (4.9% vs 7.9%, P=0.001). PTCA is safer in spite of being used in a more severe population, but the most striking difference is the 41% reduction in the need for additional revascularization, either percutaneous or surgical, during the first year of follow-up. This reduction of repeat procedures may reflect better restenosis rates, although the analysis found no difference between stented and non-stented patients.

Reduced restenosis rates have been confirmed with the use of stents [3,4]. Better acute results were also obtained with the current antiplatelet agents [5] and long-term results after stenting are now starting to appear [6,7]. It is anticipated that these results will improve in the future, with new stent designs. The group from Rotterdam [6] reported the results of stenting in 1000 patients between 1986–1996, with an 86% survival at 5 years. The group from Toulouse [7] reported a 9-year follow-up after Palmaz–Schatz stents for single-vessel disease. The event-free survival rate was 60% and freedom from death, myocardial infarction and bypass surgery was 81.7%. Diabetes was the only predictive factor of major adverse events in this last study.

More recent data from the EAST [8] and BARI [9] trials reported survival rates of 79.3% and 80.9% for the multivessel angioplasty groups, with 8 and 7 years follow-up, respectively. Inspite of similar results in terms of mortality with angioplasty or surgery for multivessel disease, this was only observed in non-diabetic patients in the BARI trial. The presence of treated diabetes gave significantly better results with surgery in this trial and worse results in those treated initially with angioplasty in the EAST trial, after 6 years.

It seems evident that angioplasty with the current use of stents and antiplatelet agents should give better long-term results than those of van Domburg et al. [1], and that the future looks even more promising. Diabetes, however, has emerged, in almost all studies, as one of the most important predictors of survival. This fact should turn our attention more to the patient and to atherosclerosis progression than exclusively to the revascularization strategy [10].

On suspicion, or after angiographic confirmation of the severity and extension of coronary artery disease, established medical therapies such as aspirin, beta-blockers, statins and angiotensin-converting enzyme inhibitors, if tolerable, should be used first. All have proved to reduce mortality and myocardial infarction in the long term and, sometimes, the need for revascularization. Treatment of hypertension, control of diabetes, smoking cessation, etc. should be part of any medical regimen, which, if possible, should remain for life.

The crucial question then, is whether to improve clinical symptoms or to increase survival? There is no doubt that some form of revascularization should be offered to improve symptoms, but I am not sure if the same applies to increased survival, particularly without aggressive medical therapy and with the exception of some anatomical subsets of lesions.

For those who perform diagnostic and therapeutic procedures, the choice of revascularization is usually based on the feasibility, with its high acute success rate, of angioplasty, at least of the culprit lesion. If not feasible, the patient is a surgical candidate, or at least a candidate for a hybrid procedure. The willingness of the patient, the presence of diabetes, the state of the distal vessels, particularly of the left anterior descending coronary artery, the opinion of the referring physician, etc., are all important factors to take into consideration in the final decision. We should always remember that for the individual patient, all available pieces of the puzzle, that are progressively accumulating, are important, and that we should aim not only to relieve symptoms but also to improve survival.

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References

[1] van Domburg RT, Foley DP, de Feyter PJ et al. Long-term clinical outcome after coronary balloon angioplasty:
Importance of control of fluid volumes in heart failure

See page 942 for the article to which this Editorial refers

During the last three decades physicians have witnessed substantial improvements in health outcomes emanating from advances in the diagnosis, management and treatment of patients with heart failure. Drugs have become available which have been demonstrated unequivocally to delay death, avoid hospitalization and improve the quality of life assessed by exercise capacity and symptom questionnaires. In the 1970s heart failure was perceived as a disease whose symptoms were closely allied to haemodynamic disequilibrium. In simple terms, breathlessness was believed to be largely attributable to a raised left atrial pressure (backward failure in old terminology) and fatigue to a low cardiac output (forward failure in old terminology). These abnormalities, it was thought, could be rectified by the use of vasodilating drugs (afterload reduction) which did bring about a lowering of left atrial pressure and increase of cardiac output. Enthusiasm for these ideas waned with the realization that some drugs which reduced afterload were not associated with any clinical benefit and that the origin of symptoms in chronic heart failure was not related in a simple manner to haemodynamic findings[1–3].

In recent years the most important developments in the treatment of heart failure have been the introduction of first the angiotensin converting enzyme (ACE) inhibitors and then beta-blockers. The efficacy of ACE inhibitors in heart failure was initially tested by the use of exercise tests, often in short-term crossover studies. The idea behind this type of experiment was that maximum exercise was a measure of the limitations of a patient with heart failure and that any improvement in exercise capacity would necessarily correlate with changes in prognosis and quality of life. This attractive notion is undoubtedly true in the context of acute heart failure which responds rapidly to drugs such as loop diuretics[4]. But once patients have been stabilized in terms of fluid overload and central haemodynamics by the use of powerful diuretics, exercise capacity has been shown not to be a simple surrogate of outcome in patients with heart failure. Large changes of exercise capacity are likely to be indicative of benefit, but a small change or no change in exercise capacity does not necessarily mean no benefit. Improvements in morbidity and quality of life have consequently become the goal of many cardiovascular physicians. Numerous questionnaires have been designed to measure quality of life. These have not fulfilled expectations because they are difficult to apply, time consuming, lack reproducibility, lack sensitivity and there is often patient resistance. In addition debate continues about what constitutes morbidity or quality of life. Hospitalization has come to be regarded as a measure of morbidity. Worse, in