Andreas Gruentzig was not quite ready to accept patients with chronic total occlusion for coronary angioplasty when this indication slipped in through the back door, albeit, it was on his mind. The waiting list between diagnostic study and angioplasty occasionally produced "surprise occlusions" found at the angioplasty session in patients accepted for a non-total lesion, the lesion having closed silently after the diagnostic study. Patient, operator, and laboratory crew all being geared for angioplasty, the guiding catheter already introduced and the balloon prepared, an angioplasty attempt was irresistible. It succeeded in 62% of 16 such patients among the first 1,000 angioplasty cases of Andreas Gruentzig and coworkers. It seems odd at first that the crude, non-steerable material used in those times yielded a success rate in total occlusions that nobody would be ashamed of today. First, all occlusions were very recent. Second, the stiffness of the original angioplasty balloons were favorable for occlusions in contrast to the floppy wires and trackable balloons used currently. In fact, one of the balloon catheters advocated for occlusion angioplasty at present resembles the ancient Gruentzig balloon.2

See p 1003

Over the years, "occlusion angioplasty" has become commonplace, accounting for about 10% of the total angioplasty activity.3,4 Since 1983, considerable pertinent literature has accrued. The major reports represent more than 2,000 patients4-12 and yield an average success rate of 66% (range, 54-72%), emergency surgery rate of 2% (0-3%), infarction rate of 2% (0-18%), among which were only a very few Q wave infarctions, in-hospital death rate of 1% (0-2%), and recurrence rate of 62% (43-77%) among patients with success who were restudied, with a rate of complete reocclusion of only 17% (4-21%). There was no clear-cut increase in success rates over the years when looking at the different reports or looking at results of a single institution,4 although only the most recent three reports state more than 65% success. Yet, two of them include functional occlusions11,12 and the third describes a cohort with more than 25% occlusions fresher than a week. However, this does not mean that there have been no technical improvements and no learning curve. Their effect on success has simply been blunted by broader indications, including older and longer occlusions.

Technical Improvements

The conventional wire/balloon technique has remained the mainstay of occlusion angioplasty.1 It has been learned that stiffer wires succeed where floppy wires fail.7 A particularly robust ball-tipped wire (Magnum wire) showed superiority over conventional wires in a first randomized study comparing two technical approaches to chronic occlusions.13 Because even these wires are still quite flexible, bracing by advancement of the balloon catheter close to the tip is needed to enhance stiffness. Fixed-wire balloons have been rediscovered for chronic occlusions: the sturdy Omniflex as a single instrument2 and the more flexible Probe in conjunction with a probing catheter.14

The Rotacs system (smooth metallic tip rotating at <200 revolutions per minute) is the only drilling device used routinely for chronic occlusions that is unguided by a previously placed wire.15 The Kensey high-speed drill, rotating at up to 100,000 revolutions per minute, seems stuck in the investigational stage it entered in 1987.16

None of the various coronary atherecorty devices have been used to approach chronic occlusions without passing a wire first. They have, therefore, no value concerning the actual recanalization.

Two laser devices have been clinically tested for initial passage through chronic occlusions. The argon laser-heated hot-tip wire has been abandoned, despite some initial enthusiasm.17 The results of the bare argon laser instrument LASTAC18 are at best comparable with those achieved with less costly mechanical means, a fact that discouraged the investigators. There is clearly a mechanical component with these sturdy and blunt catheters, and a significant laser contribution to the recanalization is questionable. To take advantage of the uncontested potential of laser energy to create channels, lasers triggered by spectral analysis of the target tissue are being investigated.19 Such concepts are intriguing but unlikely to mature to routine clinical use.

A 20-kHz ultrasonic catheter successfully ablated human calcified atherosclerotic plaques in vitro and recanalized vessel occlusions in an animal model.20 This project is compromised by the fact that the elastic fibrous tissue that occlusions are predominantly made of is not a valid target for ultrasonic breakdown. It likely also resists the accompanying shock wave seen to remove fresh clots.

New Insights

The articles from Atlanta, Ga., recently published in Circulation,12 and from Rochester, Minn., appearing in

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this issue of *Circulation,*4 corroborate facts grasped previously and provide interesting new clinical notions concerning occlusion angioplasty.

The population accepted for catheter recanalization of chronic total coronary occlusions is characterized by an over-50% incidence of previous infarctions but well preserved left ventricular function. Indications for angioplasty are more lenient than those for surgery because only 36% and 58% of patients with failed angioplasty in Atlanta and Rochester, respectively, subsequently underwent bypass surgery. For the remainder, medical therapy was deemed sufficient.

The Atlanta study confirms that functional occlusions yield a higher success rate. Moreover, it demonstrates what has not been demonstrated before, that recurrences of functional occlusions are less frequently totally occlusive than those of initially complete occlusions. This definitely relegates functional occlusions into the realm of nontotal lesions, where they belonged in the first place. If nontotal lesion angioplasty had a follow-up occlusion rate anywhere near the 17% seen with occlusion angioplasty, one would witness an infarction rate of about 10% (some patients have collaterals) during the first months after routine angioplasty! Fortunately, restenosis after angioplasty is produced by tissue proliferation generating a smooth and elastic inner lining and not by recrudescence of the original plaque, with its propensity for rupture and acute thrombosis. This explains the blessed paucity of site-related infarcts during follow-up after routine angioplasty and the innocuousness of recurrent angina a few months after successful angioplasty in terms of risk for infarction (facts yet to be fully appreciated by the medical community).

The high incidence of complete reocclusions after occlusion angioplasty remains to be explained, because there is but one common mechanism of recurrence to all sorts of lesions (confirmed by only 1% late restenoses in the Atlanta study, in keeping with nontotal lesion restenosis). The reocclusions must be predominantly thrombotic occlusions occurring immediately after the recanalization and going clinically unrecognized, because they only reproduced the status quo. The question of whether the rare late reocclusions are more infarction prone or whether the former collaterals remain reliably and readily recruitable is answered by the Rochester study, reporting no death and no significant infarction in such patients, as well as by anecdotal reports on prompt recruitment of dormant collaterals as late as 6 years after successful recanalization.21-23

As for factors influencing success in true occlusions, the classical ones are mentioned in both studies; namely, age of occlusion (substantiated by data in the Rochester study), length of occlusion, visible stump (substantiated by data in the Atlanta study), and absence of bridging collaterals. A new variable is introduced by the Atlanta study (i.e., thrombus adjacent to the occlusion). This proved to be a favorable sign, perhaps because it is associated with recent occlusions. Irrespective of the technique used, patient selection alone may produce a wide range of success rates. They may even reach those of nontotal lesion angioplasty as shown by the 93% success with recent occlusions in the Rochester study.

The literature on occlusion angioplasty was free of major complications such as death and Q wave infarctions until 1990. Since then, several cases have been described.4,11,12,24 In no case so far has the isolated reocclusion of the recanalized site been indubitably identified as culprit for death or Q wave infarction, but two of the nine deaths in the Rochester study were due to manipulations at the occluded site. The majority of serious complications, however, are due to additional sites attempted simultaneously. Possible mechanisms for occlusion site–related complications leading to emergency surgery, infarction, or death include dissection of proximal vessels and embolization. The conceivable problem of impairment of proximal collateral outflow or distal collateral inflow has yet to be documented in a specific case. Even very long dissections created by subintimal pathways during recanalization attempts and reaching beyond the resumption of the distal lumen typically produce, at worst, modest creatine kinase elevations. The true lumen is pressurized by the distal collateral inflow, which tends to repaste the dissected layers. Finally, perforations are not a prominent clinical threat with conventional methods (none reported in the over 800 patients of the two studies discussed), although there may be unrecognized wire perforations.

The two studies shed no new light on the restenosis enigma. The high restenosis rate can be explained by the less-than-ideal results frequently achieved with recanalizations. This was a predictor of recurrence, according to the Atlanta study; or it may be explained by the good collateralization, mandatory for the indication of recanalization attempts, that is an even more potent predictor of recurrence (the collateral pressure persists after disappearance of the collaterals).25 Then again, the high restenosis rate may be artifactual because of the erroneous addition of almost 20% of unrecognized acute reocclusions and the low restudy rates (53% in Atlanta and 28% in Rochester, respectively). Clinical recurrences after recanalization are rarely compelling and never menacing. This markedly lowers the restudy rate and skews the results calculated from the few patients restudied who are mostly symptomatic.

The overall clinical gain emerging from these two studies is dominated by the stunning finding in the Atlanta cohort that successful recanalization yields a survival at 4 years of 99%, which is significantly superior to the 96% with a failed attempt. Not many times before has a study on coronary angioplasty demonstrated a beneficial influence on survival. However, the authors wisely took care not to overstate this finding. First, technical success or failure may indirectly be independent on the viability of patients. Second, failed angioplasty may compromise the patient’s chance to survive, which offsets the beneficial effect of angioplasty attempt as a whole. Third, bypass surgery carried out predominantly in failed patients might be responsible for the increased mortality, and longevity might have been better if the failed group had been treated nonsurgically. Surgery, on the other hand, seemed to have positively influenced symptomatology. It must account for the failure of both studies to confirm what was previously documented, namely, reduced symptoms during follow-up in patients with successful recanalization.26 Had none of the patients undergone bypass surgery, the
group with failed angioplasty would probably have remained more symptomatic.

The quite sobering message of both studies is that the only palpable benefit of successful balloon recanalization of coronary occlusion is a reduction in subsequent use of bypass surgery. This is not even a cardiac event. It represents an arbitrary decision aimed at patient comfort rather than survival; yet this message need not deter interventional cardiologists from continuing to dig to see the light at the end of the tunnel. It is rewarding enough to obtain a long-term course comparable with that after surgery in the patients with successful angioplasty, while knowing that a failed attempt is unlikely to jeopardize the patient. Moreover, in a rare patient, timely recanalization of an occluded coronary artery may prove to be lifesaving when the vessel originally providing collaterals occludes and the entire myocardium is subtended by the previously recanalized vessel supplying reversed collateral flow. There is indeed an incentive for endeavors to improve primary success that will entail widening of indications. After all, in one- and two-vessel disease, total occlusions are the prime reason to select bypass surgery over angioplasty.

**Outlook**

The clinical yield of chronic coronary occlusion angioplasty will never match that of coronary angioplasty for stenoses even if technical success can be improved. This is acceptable as long as recanalization angioplasty remains low risk and low cost, which disqualifies lasers and other sophisticated instruments. Hence, genuinely mechanical tools are likely to remain the backbone of catheter revascularization, starting with the simplest.

Although occlusion angioplasty is dealing with dead ends, it is itself not at a dead end. When it is used 1) reasonably by selecting patients, operators, and material augmenting the best chance of success, 2) prudently by knowing how to avoid pitfalls and when to quit, and 3) cost efficiently by combining the attempt with the diagnostic study, it may enjoy sustained use without being bothered too much by the notorious call for randomized proof of its right to exist.

**References**


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"Occlusion angioplasty". Light at the end of the tunnel or dead end?

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