Vascular Brachytherapy
Boon or Bust?
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In the present issue of Circulation, Drs Teirstein and King1 and Waksman and Weinberger2 provide a chronology for the evolution in concept and practice of vascular brachytherapy (VBT). The documentation for durable efficacy of VBT in suppressing recurrent in-stent restenosis (ISR) has been provided by multiple randomized controlled clinical trials. The importance of extending the length of radiated arterial segments well beyond the zone of procedural endoluminal injury has become evident. Similarly, the relative clinical and angiographic safety of VBT (versus conventional therapies for ISR) has been demonstrated, and specific procedural as well as adjunctive pharmacological protocol modifications have evolved. These have included the avoidance of restenting and the prolonged administration of clopidogrel therapy, which have collectively effectively eliminated the previously alarming issue of late thrombosis after VBT.

Finally, an effective and safe therapy for restenosis, the “Achilles heel” of percutaneous stent deployment, was available. Just as many interventional programs are successfully implementing multidisciplinary VBT programs, the primary indication for VBT (ISR) has been apparently “cured.” Drs Teirstein and King1 pose the question, “Will drug-coated stents transform vascular brachytherapy into a cure without a disease?” Their expert opinion answer to this question is “No.” Even in a study patient population composed largely of single-vessel, single-lesion coronary disease, angiographic binary restenosis rates after Cypher drug-eluting stent (DES) deployment were 8.6% overall, 18% in diabetics (35% for insulin-treated diabetics), and 16% in small vessels. Higher rates of restenosis (≥25%) have been reported for more complex patient subsets, such as those with branch vessel stenoses. If, as Drs Teirstein and King1 point out, drug-coated stents appreciably increase the number of patients undergoing stent implantation, even the relatively small percentage of patients who experience late DES failure will translate into a “substantial absolute number of candidates for brachytherapy.” Furthermore, they admonish, “At present, coated stents have no track record for [the treatment of] in-stent restenosis.” The zeal for coated stents is sobered by multiple questions for which evidence-based answers are not currently available. For example, what data define the safety and efficacy of DES deployment for treatment of ISR that follows bare-metal stent placement? Will diffuse versus focal patterns of ISR respond differently, and what is the relative safety/efficacy of DES (versus VBT) treatment by ISR pattern? What procedural and/or adjunctive pharmacological protocol modifications must evolve to provide optimal late outcomes? Although late failure of DES (ISR) in clinical practice will likely be more frequent than suggested by available clinical trials, how will this novel disease process respond to treatment with either VBT or the deployment of another DES? More specifically, how will the nonresorbable polymer coat be altered by ionizing radiation or another DES? What is the optimal strategy for restenting with another DES? What type and/or duration of adjunctive pharmacology will be required to ensure stability of this novel “stent-on-stent, polymer-on-polymer” complex? We don’t know the answers to these questions presently. How quickly and definitively will answers be forthcoming from registry experiences using historical controls? The answer to the last question is intuitively obvious: more slowly and less definitively than answers provided by randomized, controlled clinical trials. We must
acknowledge both the current gaps in evidenced-based support for DES treatment of multiple patient cohorts as well as the data derived from randomized controlled clinical trials of VBT that document therapeutic efficacy across a broad spectrum of target vessel size, lesion length, and patient demographics. The considerable efforts expended to objectively establish safety and efficacy of VBT for treatment of ISR cannot be subjugated to the self-interests of operator convenience. The challenges of coronary heart disease are many, and therapy for this protean disease process will not likely be defined by a single technology. Both approaches (DES and VBT) to limit neointimal proliferation and the specific role of VBT to treat established neointimal scar tissue secondary to stent deployment are likely to be complementary in the foreseeable future.

References
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