MTWA value as positive. Although this analysis operates on this dichotomy to generate the "positive" and the "negative" response of a patient to MTWA testing, thus creating a qualitative context, there is a quantitative issue as its underpinning (i.e., a patient has to reach the threshold to qualify as "positive"). Moreover, current work indicates that the magnitude in $\mu V$ of MTWA may be of importance, and higher values of calculated MTWA may imply worse prognosis (3,4), or therapy with various drugs, particularly beta-blockers, may attenuate the magnitude of MTWA (5). However, more work is needed to substantiate the claim that the quantitative assessment of MTWA may have advantages over the qualitative evaluation. In the meantime, it is important to deal with some possible confounders of the quantitative employment of the MTWA, 1 of which may be its T-wave amplitude dependence (6). Accordingly, in the presence of ventricular conduction delays, which often are associated with T waves larger in amplitude than the ones encountered in association with normal intraventricular conduction, a greater magnitude of MTWA in the former than in the latter may not mean a higher risk for sudden cardiac death or malignant ventricular arrhythmia, but merely an enhancing effect of the taller T waves on the magnitude of MTWA in patients with wide QRS complexes. Probably this was the reason for the increased rate of false-positive MTWA tests in patients with wide QRS complexes ($\geq 120$ ms) (specificity 22% in the patients with a wide QRS and 40% in the patients with a narrow QRS) in the authors’ other report (2). In that report they categorized their patients to those with QRS duration of $<120$ ms and $\geq 120$ ms (2). In their present contribution (1), by employing 3 categories of patients in terms of QRS duration (Table 1) (QRS $<110$ ms, QRS 110 ms to 120 ms, and QRS $>120$ ms), they provide an opportunity to evaluate the effect of the amplitude of the T waves on the magnitude of MTWA in a “dose response” fashion. What is needed for this is a comparison of the values of MTWA in $\mu V$, and the T-wave amplitudes in mV in the above 3 categories of patients, according to their QRS duration. Could the authors provide us with these data? Evaluation of possible determinants of the MTWA and potential adjustment via a MTWA index (6) may upgrade both the currently used “qualitative” assessment and a future adoption of quantitative assessment of MTWA testing.

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REFERENCES


Percutaneous Intervention in Saphenous Vein Bypass Graft Disease

Case Against the Use of Drug-Eluting Stents

Saphenous vein grafts (SVGs) tend to degenerate over time, and almost one-half develop significant stenosis and nearly 40% are completely occluded within 1 decade (1). Use of balloon angioplasty alone without stenting for treatment of SVG disease is associated with poor short-term and long-term outcome (2). Use of bare-metal stents (BMS) is associated with restenosis rates as high as 50% at 6 months (3). Lately, drug-eluting stents (DES) have been used for the treatment of SVG disease.

In contrast to the native coronary arteries, in which restenosis after percutaneous coronary intervention (PCI) is mainly due to intimal hyperplasia, restenosis in SVG involves cellular hyperplasia, progression of atherosclerosis, local inflammatory reaction to stent struts, and thrombosis. These observations suggest that it may not be wise to extend the seemingly better short- and midterm results seen with PCI for treatment of native coronary artery disease to the treatment of SVG disease. We have recently looked at the data in our institution in 109 patients with SVG disease who received BMS or DES. During a follow-up period of 33 months, we found that the incidence of

![Kaplan-Meier Survival Curves for Freedom From MACE at Almost 3 Years Follow-Up](image-url)
major adverse cardiac events (MACE) was much higher in patients treated with DES. We found that there was initially a benefit in terms of MACE in patients who received DES compared with the benefit seen in patients receiving BMS, but thereafter we noticed a late catch-up phenomenon (Fig. 1).

In this regard, Chu et al. (4) in a small study showed that although there were no differences in DES and BMS used for the treatment of SVG disease, the incidence of MACE increased from 6 months to 1 year after PCI in the BMS group, and the DES group had a much more pronounced increase in MACE in the same time frame. The secondary post-hoc data from the delayed RRISC (Reduction of Restenosis In Saphenous vein grafts with Cypher Stent) trial (5) also showed that over a median follow-up of 32 months, use of BMS was associated with significantly lower long-term mortality than the use of DES in SVG disease. The use of DES in the SVG disease needs to be re-examined.

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Reply

We really welcome the additional data provided by Dr. Bansal and colleagues over the mid- and long-term outcome of drug-eluting stents (DES) in diseased saphenous vein grafts (SVGs). We also appreciate the words of caution expressed by the authors and focused on a more careful use of DES in this type of lesion. Indeed, their data, as well as our long-term data from the DELAYED RRISC (Death and Events at Long-Term follow-up AnalYsis: Extended Duration of the Reduction of Restenosis In Saphenous vein grafts with Cypher stent) trial (1), and the data from Chu et al. (2), all point out that in the long term DES seem not to maintain the possible advantages shown in the midterm. Another additional study (a sub-study of the large prospective STENT [Strategic Transcatheter Evaluation of New Therapies] registry), recently presented as an abstract, also showed no differences between DES and bare-metal stents in SVGs (3). To our knowledge, while there are some data showing benefit of DES over bare-metal stents in the midterm (up to 6 to 9 months), there are no registries showing the same benefit in the long term (>1 year). The only long-term study is the one previously mentioned, and all show a similar trend without clear advantage of DES over bare-metal stents.

However, we have to underline some issues related to all these analyses. On the one hand, our data are focused only on sirolimus-eluting stents and not on other types of DES (1). On the other hand, the data of Dr. Bansal and colleagues, of Chu et al. (2), and of the STENT registry (3) were based on analyses encompassing different types of DES. Whether their results were mainly driven by a suboptimal performance of one type of DES over the others or whether there is a “class-effect” of DES in SVG, this cannot be evinced by the data provided. New prospective studies are undergoing in order to also offer additional data on other types of DES, such as polymeric paclitaxel-eluting stents (4). In addition, as all of these studies analyzed de-novo lesions in SVGs, we have a total lack of data on the way DES perform in restenotic SVG lesions.

While waiting for a conclusive large and well-powered randomized trial of DES versus bare-metal stents in SVGs, in our opinion the use of DES in this lesion subset in daily life clinical practice should be discouraged (unless prospectively evaluated in a study). If there is a willingness to implant a DES in a diseased SVG, this should be firmly discussed with the patient, and a careful assessment of the possible advantages and risks related to the implantation of this device should be cautiously evaluated. Moreover, we welcome interventional cardiologists that implanted DES in SVGs to try to collect long-term data on their patients in order to provide additional data to the scientific community, like Dr. Bansal et al. remarkably did in their letter.

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