Difference in harmonic content was ≤0.4 mm Hg for the first eight harmonics, which contained 99.9% of cumulative power. Thus, the Millar tonometer provides a robust method for high-fidelity recording of the important features of the radial waveform.

The generalized transfer function used in sphygmoCor is virtually identical to that described by Lasance et al. (4), as well as by Karamanoglu et al. (5), and its utility was described by Chen et al. (6). Its validity has also been confirmed in a realistic model of the human upper limb (7). When this generalized transfer function was prospectively validated against invasively recorded aortic pressures in a large group of patients, the mean offset for systolic, diastolic, mean, and pulse pressures was <1 mm Hg (SD <4.5) (8). Differences were slightly greater (mean <4 mm Hg, SD <7) for augmented and end-systolic pressures (9) but still within the Association for the Advancement of Medical Instrumentation (AAMI) SP10 criteria for "substantial equivalence." Therefore, we believe that the validity of the transfer function itself is proven, and we understand that the Food and Drug Administration (FDA) has accepted use of the sphygmoCor system when combined with accurate intra-arterial manometer systems, and with the noninvasive Millar tonometer. Nevertheless, we have retrospectively compared augmentation index in the radial artery between the two groups in our original study (1). Radial augmentation was enhanced in the hypercholesterolemic subjects (66 vs. 78%; $p < 0.001$), suggesting that the observed differences in central augmentation were not due to the transfer function per se. Rather, the transfer function permits accurate, noninvasive determination of central pressures and thus left ventricular load.

Finally, Dr. Hope and colleagues question cuff calibration of peripheral waveforms. Although we agree that invasive radial waveforms were used in the most recent validation study (8), such an approach tests the validity of the transfer function itself. We also accept that not all automated sphygmomanometers are accurate. However, we used a device that meets both the British Hypertension Society and AAMI standards (Omron, 705CP), thus minimizing any potential source of error (10). The investigators should remember that such scaling issues also apply to other techniques—for example, when carotid waveforms are scaled to pressures recorded by sphygmomanometry in the arm (2,11). Moreover, such investigators have not always used validated devices (12). We trust we have put to rest the concerns voiced by Dr. Hope and colleagues about the transfer function, and we await with interest the results of the large-scale outcome studies using sphygmoCor currently in progress, as we are sure they do too.

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Enoxaparin After High-Risk Coronary Stenting

We read with interest the report by Batchelor et al. (1), published in the November 15, 2001, issue of the *Journal*. They conclude that, given the relative safety of enoxaparin and the potential to reduce the risk of subsequent infarction, a 14-day course of enoxaparin may be considered for carefully selected patients. We would like to comment on some aspects of their study.

The investigators' conclusion is based on the finding of a reduction of myocardial infarction at 30 days. This variable was neither the primary end point nor a predefined secondary end point in their study. The fact that the study was stopped prematurely owing to a low rate of events does not, in our opinion, allow one to conclude a therapeutic recommendation. Of course, it is possible that the end point was not achieved because of a beta error, as pointed out by the researchers, but we do not know what the outcome would be in a study including at least 3,590 patients, which was the recalculated sample size. Contrarily, Batchelor et al. (1) affirm that rates of major bleeding (3.3% for enoxaparin, 1.6% for placebo, $p = 0.08$) were comparable. In reality, the rate of this event with enoxaparin was two times higher than with placebo, and the p value was next to the conventionally accepted significance level. In this case, the lack of statistical significance could also be due to a beta error, but this is omitted by the investigators.

Some aspects of the discussion deserve comment. It is mentioned that enoxaparin's clinical superiority over unfractionated heparin (UFH) has been shown in patients with acute coronary syndromes (2–4) and that extended low-molecular-weight heparins (LMWHs) also reduce the short-term risk of thrombotic events, although benefits are not sustained at six months (5). In the same manner that it is mentioned that enoxaparin was superior to

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UFH, it should be specified that the benefit derived from extended LMWH use was obtained with dalteparin in the FRISC II study (5). Neither in the TIMI-11B (4) nor in the FRAXIS (6) studies, a benefit associated with the extended treatment of enoxaparin or fraxiparin, respectively, was not evidenced. As has been pointed out, LMWHs should not be regarded as interchangeable, but rather as distinct drugs with specific structural and functional profiles that require individual investigation (7). Thus, results obtained with one LMWH cannot be extrapolated to the other products.

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