Letters to the Editor

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Reply to the Editor:

We are grateful to Drs Gelsomino and Romano for their interest in our article.[1] They raise several important issues related to the adequacy of investigating the cardiovascular system by means of the pressure recording analytical method (PRAM) and to the methods used to attenuate the risk of underdamping/resonance artifacts in vivo, potentially responsible for an incorrect measurement of hemodynamic parameters.[1,2]

Dynamic response artifacts are often observed in patients receiving peripheral monitoring based on pulse contour analysis.[3] In particular, underdamping effects are frequently observed whenever the stiffness of the cardiovascular system, including the apparatus for pulse pressure "transduction," is augmented.[3,4] Therefore, conditions such as systemic arterial hypertension, severe calcified ascending aorta, diffuse atherosclerosis of the aortic arch, systemic vasoconstriction, hypovolemic conditions, and a stiff transduction system (related to the use of numerous stopcocks, stiff tubes, and arterial transduction catheters) all contribute to increase the risk for dynamic response artifacts.[3,4]

Although transducers ad hoc manufactured to avoid underdamping artifacts exist on the market,[3] they were not available at our institution at the time of the study and therefore were not used. Accordingly, our detection system might have been unable to recognize and consequently "clean" possible dynamic artifacts.

However, as for any detection system based on pulse contour analysis, the recorded variables were validated by an expert operator. For a correct interpretation of any data, the PRAM method undeniably requires a proper recognition of the dicrotic notch (which can be easily misrecognized by the machine and in cases of underdamping/resonance artifacts). Therefore, back to Gelsomino and Romano's question, no data were collected in our study unless validated by a fully trained operator. Therefore whenever artifacts existed, the detecting system was "restarted" to achieve a correct recognition of the pulse wave, thus collecting data only when the system properly interpreted the pulse.

An additional troubleshooting maneuver was to move to a different arterial site to derive a "correct" pulse wave (mainly the common femoral arterial access, where artifacts were less commonly encountered in our experience). Furthermore, the transduction system was a priori optimized by using only 1 stopcock and flexible tubing/arterial catheters, and only 20F catheters for radial transduction or 18F catheters for femoral transduction.

These problems were rarely encountered during the study because of the setting and the peculiar hemodynamic pattern of the enrolled population: Patients weaned from an intra-aortic balloon pump (IABP) are usually vasodilated (because of the IABP effect or the concomitant use of vasodilating drugs); moreover, patients with severe atherosclerotic aortic arch or thoracic descending aorta did not undergo transfemoral IABP because of institutional policies (and thus were not enrolled in this study). Given the peculiar design of the trial, based on the hemodynamic monitoring and surveillance of a potential perioperative low cardiac output state after weaning, all patients were maintained at proper preload (central venous pressure maintained at 8-12 mm Hg) and afterload states: thus, conditions such as vasoconstriction and hypovolemia, potentially favoring underdamping/resonance artifacts, were avoided. On the basis of this approach, it can be easily recognized that the most common causes of underdamping were prevented but overall rare in this specific patient population.

In addition, we want to underscore 3 major validation aspects of our study:

1. Before using PRAM, we performed a "cross-check" validation of data via a pilot study. We analyzed the reliability of hemodynamic indices derived from PRAM compared with those derived by the traditional Swan–Ganz thermodilution method. As in other reports,[5] this preliminary study demonstrated a perfect agreement between PRAM and Swan–Ganz data, unless paroxysmal high-rate atrial fibrillation occurred. However, a new onset of high-rate atrial fibrillation did not occur in our patients during the weaning trial. Of note, a new onset of high-rate atrial fibrillation at our institution is considered a contraindication to the progression of an IABP-weaning trial.

2. Our trial was designed to investigate at 360 degrees the impact of the 2 weaning strategies on the entire cardiovascular system. Accordingly, hemodynamic and biochemical indices were collected, such as troponin I and lactate, the former showing the myocardial perfusion and the latter showing the adequacy of peripheral "oxygenation." Again, peripheral lactate confirmed the superiority of the "volume-deflation" method versus the "rate-reduction" strategy in terms of peripheral perfusion.

3. Romagnoli and colleagues[3] have demonstrated that whenever underdamping/resonance effects exist, significant differences are recorded between hemodynamic data derived from "conventional" PRAM and those derived from underdamping/resonance-corrected PRAM by means of specific transducers. As we mentioned, these transducers were not available at the time we started the study. Furthermore, an

important "delta" was recorded in that study in terms of systemic arterial pressure and dP/dt$_{MAX}$ (variables not collected in our study and not considered as end points of the trial), but a less important "delta" (although statistically significant) was recorded for cardiac index and cardiac cycle efficiency (magnitude ∼0.24 L/min for cardiac index and 0.018 units for cardiac cycle efficiency, respectively).[3] However, these "deltas" were registered in an animal model during systemic hypertensive conditions (mean values of systolic blood pressure ranging from 160 to 174 mm Hg),[3] a hemodynamic pattern distant from that of a vasodilated patient in stable hemodynamic condition, as in our hemodynamically oriented IABP-weaning trial. We strongly believe that the magnitude of these "deltas" in patients with an overall good hemodynamic performance, especially when confirmed by other hemodynamic, biochemical, and clinical data, should be considered negligible from a clinical perspective.

Finally, we assume that the measurement of any biological process is truly a difficult task, and that the trend over time of a hemodynamic index, rather than its absolute value at a single time, is important in clinical practice, as in the case when evaluating 2 different IABP-weaning strategies. Accordingly, considering that most of the mentioned mechanisms responsible for a "high" cardiovascular stiffness are unavoidable and constant in a given patient, provided that a good transduction setting is used and that preload and afterload have been adequately corrected, we still consider that the traditional PRAM method (in the absence of ad hoc manufactured transducers) can be reliable in clinical practice, as long as a proficient human validation phase of the PRAM-derived indices is provided.

We are pleased that our study confirmed, for the first time in humans, the important findings found by Gelsomino and colleagues[6] in a swine model of acute ischemia, in which hemodynamic indices and cardiac contractile efficiency parameters were similarly derived by conventional PRAM methodology, showing the same inadequacy of 1:2 and 1:3 IABP assisting rates on hemodynamic recovery.

We hope that these endeavors, taken together, can open the way to a new paradigm when weaning patients from IABP support.

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GUIDED OR NONGUIDED ENDOCARDECTOMY DURING SURGICAL VENTRICULAR RECONSTRUCTION?
To the Editor:

In a recent article in the *Journal*, Babokin and colleagues[1] presented results after surgical ventricular reconstruction (SVR) together with endocardectomy along radiofrequency ablation–induced markings. The objective was to evaluate the efficacy of a new approach for endocardectomy during SVR in patients with postinfarction left anteroseptal ventricular aneurysms. The extent of the endocardectomy was guided by preoperative electroanatomic mapping and markings produced by radiofrequency ablations. The design was a retrospective single-center study, and 168 patients were included and categorized in 2 groups: 74 patients who underwent SVR including endocardectomy and 94 patients who underwent SVR. Whether or not endocardectomy should have been added to SVR was decided by the surgeon. All patients also underwent coronary artery bypass grafting. In the SVR + endocardectomy group and SVR only group, early mortality was 1% and 6%, respectively, and 1-year mortality was 5% and 13%, respectively. Cardiac function and volumes were similar between the groups both before and after surgery. In a subgroup of patients who underwent both pre- and postoperative electrophysiologic studies, Babokin and colleagues[1] found a lower incidence of spontaneous and induced ventricular tachycardia (VT) after surgery in patients who underwent SVR + endocardectomy compared with SVR only. There were also fewer implantable cardioverter-defibrillators (ICDs) in the SVR + endocardectomy group.

Patients are at risk for malignant arrhythmias after SVR; however, whether the risk is high[2] or low[3] is unclear. One strategy would be to use ICDs routinely after SVR. However, there are downsides to ICD use, for