Letter to the Editor

Which are the exact guidelines for more rationale intervention concerning beta-blockers administration in coronary patients preoperatively?

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This interesting study [1] confirms a well-established knowledge: the over-stimulation of the sympathetic system has unfavorable consequences on early and late results in a coronary patient [2,3]. Especially, the combination of decreased parasympathetic and increased sympathetic system activity reduces further the threshold of ischemia, ventricular tachycardia or fibrillation and sudden cardiac death in post-CABG patients [3]. The above mechanism involving the catecholamine elevated levels derived from the corresponding increased stimulation of sympathetic system favors the induction of endothelial injury and the further activation of platelets [2]. However, both the methodology and evaluation of your results raise several questions:

(a) Initially, the methodology concerning the measurement of the two parameters (HR and PP) is unclear and unequal in your material. Especially, when are the measurements performed? What was the interval between them? When are the measurements performed in regard with the operation, where the stress is much higher, and beta-blocker administration time respectively? Examining Fig. 1(1), it is evident that as far as the patients with bpm ≥70 are concerned, more than 50% did not receive beta-blockers (and reversely) despite the fact that in your material 65% of patients had three-vessel disease and 17% had stem disease. Consequently, what were the guidelines for the beta-blocker administration?

(b) Of note, you enrolled patients according to an arbitrary limit of EF: 0.40. Did all patients with EF >0.55 have the same ‘pattern’ of HR and PP parameters compared to them with EF >0.40?

(c) How could the paradox that the primary outcomes were associated with PP but not also with the mean PP be interpreted?

(d) In addition, you did not clarify if any patients were receiving specific regiments before the measurements (e.g. premedication of tranquilizers as diazepam by the anesthetist). This protocol is not stable but is based on the anesthetist’s estimation and on ‘sentimental profile’ of the patient.

(e) What was the preoperative PP distribution in patients, in regard with beta-blocker administration? What finally was the correlation between mortality and morbidity in relation to the preoperative administration of beta-blockers? The preoperative beta-blocker administration is associated with much lower morbidity and mortality after CABG [4]. Fact that is also supported by your outcomes: despite the definition of an arbitrarily high ’cTnI concentration threshold’ (cTnI >20 μg/l), a relatively high percentage of postoperative MI (12.1%) occurred in comparison to other studies [5].

In our opinion, the European Society of Cardiology might determine specific guidelines concerning the rationale administration of anti-anginals and especially beta-blockers and tranquilizers. In addition, the individualization of the dose of beta-blockers according to HR, BP, and disease severity, and on the other hand the corresponding of the tranquilizers based on the autonomous nervous system pattern (increased sympathetic activity tone); would ‘stabilize’ the preoperative hemodynamic profile of the patients, contributing to the further improvement of early and maybe late results.

References


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Reply to the Letter to the Editor

Reply to Apostolakis et al.

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We thank Dr Apostolakis et al. for their regular comments in this journal. Their comments regarding our paper [1] are
focused on the highly debated usefulness of beta-blockers prior to cardiac surgery [2]. The poorer prognosis of our patients with high HR could potentially be interpreted as beneficial effects of beta-blocking therapy [1]. To date, trials on beta-blocker therapy prior to coronary surgery provide conflicting results [3].

It should be stressed that our study is descriptive [1], emphasising that rest heart rate (HR) and pulse pressure (PP) are two independent predictive markers of perioperative events, which should be considered in risk scores for coronary surgery. While we adjusted our findings to the use of preoperative HR lowering medications, primarily beta-blockers, because of their evident influence on HR and to some extent on PP values, the analysis of events according to preoperative beta-blocker therapy is beyond the scope of this observational study. Such analysis would have required a randomised controlled trial, or at least a propensity score analysis. Yet, the aim and design of our study could not support such conclusions. Of note, we did not find any significant difference regarding PP distribution according to beta-blocker administration.

We consider the issue raised about 'mean PP' unclear. The conditions for HR assessment are important. We measured HR systematically the day before surgery, at patient’s admittance. Pulse pressure was calculated according to systolic and diastolic blood pressure (BP) values averaged from a single BP measure during the anaesthetist’s outpatient visit one month prior to surgery and from two consecutive BP measures upon admission. The assessment being strictly preoperative, any interference with specific premedication can reasonably be excluded. Consequently, we consider the conditions for HR and PP measurement in this study as realistic, as usually performed in clinical routine. With the aim of using these two parameters for perioperative risk stratification, we consider that they should be measured in a steady state, long before surgery.

Defining perioperative myocardial damage during heart surgery by any cut-off for troponin-I concentration remains indeed arbitrary. In a consensus statement, it has been acknowledged that ‘the situation for patients undergoing CABG is too complex to define simple cut-off values’ [4]. Hence, the troponin-I threshold used, although rather high, is consistent with findings in the literature [5], with similar rates of clinical complications. Of note, we found similar trends when we used a lower cut-off (10 μg/l), comparable to reported results with the 20 μg/l threshold. In conclusion, we consider evidence regarding the systematic use of beta-blockers in the setting of CABG, as well as the use of biomarkers to assess perioperative myocardial damage still insufficient to be subject to consensual guidelines. Further adequate clinical trials and meta-data analyses are required to respond to these daily clinical concerns.

References


[2] Apostolakis EE, Koniaris IC, Tsigkas GG. Which are the exact guidelines for more rationale intervention concerning beta-blockers administra-


Letter to the Editor

Is the use of at least one internal thoracic artery (ITA) directed associated with increased long-term cardiac-specific survival?

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Even though, the study by Mohammadi et al. [1] comes up with significant conclusions as to the use of at least one ITA (only as far as the late outcome is considered), it seems to be lacking the liability of a perspective randomized study. Retrospective examination of results of a non-designed study suffers major flaws as the element of early outcome is absent. And it is these early outcomes that identify and strictly differ between the 3 subgroups of NITA, SITA or BITA patients and eventually determine the surgeon’s decision. Clinical experience has shown that NITA subgroup of patients suffer from more severe coronary heart disease comparing to the other two (SITA and BITA) [2,3]. Specifically:

(A) In the population of cardiac-death, sudden as well as unknown death is included. This inevitably bears a high index of error. A patient with chronic renal failure, who will most possibly suffer a sudden death (due to hyperkalemic arrest or pulmonary edema), will, according to the author’s methodology, be falsely included in cardiac related death.