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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