Heart rate and pulse pressure at rest are major prognostic markers of early postoperative complications after coronary bypass surgery

Victor Aboyans, Michael Frank, Karine Nubret, Philippe Lacroix, Marc Laskar

Abstract

Objective: There is substantial evidence to consider both heart rate (HR) at rest and pulse pressure (PP) as significant markers of cardiovascular prognosis in the general population. Despite this, neither of these two parameters has been taken into consideration in the design of modern coronary artery bypass risk prediction scores, and little data on their early postoperative prognostic value are currently available. We aimed to assess the predictive value of preoperative HR and PP in the 30-day postoperative period. Methods: We prospectively enrolled all patients referred to our institution for non-urgent coronary artery bypass grafting. We measured HR on ECG at admittance. Preoperative pulse pressure was obtained by the difference of the mean of three consecutive systolic and diastolic blood pressures. The primary outcome combined the 30-day postoperative mortality, myocardial infarction (new Q-waves on ECG or Troponin-I) was obtained by the difference of the mean of three consecutive systolic and diastolic blood pressures. The secondary outcome corresponded to clinical events only (stroke or death). Statistical analysis was performed by usual methods.

Results: We enrolled 1022 patients (age 66.9 ± 9.2 years). Those meeting the primary outcome (n = 146) had a significantly higher HR (69.9 ± 14.3 bpm vs 64.9 ± 13.2 bpm, p < 0.0001) and a higher proportion presented a PP >70 mmHg (17.1% vs 10.2%, p < 0.03). After adjustments for age, gender, systolic blood pressure, preoperative beta-blocker therapy, left ventricular ejection fraction <0.40, unstable cardiac status, redo surgery, peripheral arterial disease, renal failure, and combined vascular surgery, both HR (OR = 1.17 per 10 bpm, p < 0.03) and PP >70 mmHg (OR = 1.99, p = 0.03) remained significant risk predictors. Similar results were found when considering only clinical events.

Conclusion: This prospective study highlights the usefulness of HR and PP as preoperative risk markers in CABG candidates.

1. Introduction

One of the most challenging tasks in the management of coronary patients is the risk assessment of different revascularization strategies. When coronary artery bypass grafting (CABG) is considered, several predictive models are currently available to estimate the patient's surgical risk [1,2]. This risk prediction is critical as during the last two decades the proportion of high-risk patients referred to cardiac surgery has steadily increased. The quality of these predictive scores depends not only on their ability to classify accurately high- and low-risk patients, but also on their applicability, especially by using easy-access variables.

In several epidemiological studies in the general population, it has been evidenced that a high heart rate (HR) at rest is predictive of death; especially, but not exclusively, of cardiovascular death [3]. It is still unclear whether HR is a contributing factor or an integrative marker, as a result of the imbalance between the sympathetic and parasympathetic systems. The association between HR and mortality is independent of almost all other conditions, which could potentially be considered as confounding bias (e.g. heart failure, smoking). In general surgery, preoperative HR is predictive of perioperative cardiovascular events [4]. In vascular surgery, recent studies suggest that the intentional perioperative HR lowering could prevent adverse cardiovascular events. Surprisingly, the association between preoperative HR and the postoperative outcome of patients undergoing CABG has been poorly studied, and this parameter has never been taken into consideration in any of the available predictive risk scores for cardiac surgery [1,2].

Similar to HR, pulse pressure (PP), defined as the difference between the systolic and diastolic blood pressures (PP = SBP – DBP), is another very accessible variable, which has been widely studied and confirmed as a cardiovascular risk marker in the general population [3]. This clinical
parameter, representing the pulsatile component of blood pressure, is an indirect marker of central arteries stiffness. Almost all longitudinal cohort studies are concordant to show that PP is associated with cardiovascular mortality [3], but the ability of PP to predict post-CABG adverse events is unknown.

In this cohort study, we aimed to assess HR and PP as potential prognostic factors in patients undergoing CABG. We hypothesized that both factors are related to an excess risk of postoperative adverse events, and that this association is independent of other major known risk factors.

2. Methods

2.1. The cohort and preoperative data

The cohort constitution, data collection and variables definition have already been described in detail elsewhere [5,6]. Our aim in this prospective observational study was to detect perioperative event rates as well as factors associated with these events in order to improve risk assessment and prevention. In brief, we prospectively enrolled all consecutive patients referred to our department for CABG from August 1998 to January 2002. Patients requiring urgent surgery were excluded from the study, if physical examination was incomplete or procedure was delayed. Preoperative data included cardiovascular history, cardiovascular risk factors as well as coronary artery disease extent and left ventricular ejection fraction (LVEF). Patients were considered as smokers if actively smoking or having discontinued smoking within 2 years prior to surgery. Diabetes was defined by fasting blood glucose at entry >11.1 mmol/l or the use of oral anti-diabetic drugs and/or insulin. Preoperative systolic and diastolic blood pressures (SBP and DBP) used for the determination of pulse pressure, was an average of three consecutive blood pressure measurements. Pulse pressure (PP = SBP – DBP) and mean blood pressure (MBP = 1/3SBP + 2/3DBP) were calculated from the mean of these three measurements. Hypertension was defined by any history of hypertension with use of anti-hypertensive drugs for that purpose. Hypercholesterolemia was defined by a fasting blood cholesterol level at entry >11.1 mmol/l. The ankle-brachial index was systematically measured preoperatively, and peripheral arterial disease (PAD) was defined according to clinical history and/or an ankle-brachial index <0.85 or >1.50. A history of cerebrovascular disease combined stroke, transient ischemic attack (TIA) or carotid revascularization reported in the patient’s chart. Heart rate was recorded at admission and at rest from the preoperative ECG. Supraventricular arrhythmia was defined as the presence of sustained atrial fibrillation or flutter. The presence of beta-blockers in the patient’s treatment list at admission was noted.

The left ventricular ejection fraction (LVEF) was determined according to the ventriculography. Low LVEF was defined when <0.40. Significant coronary stenosis was considered when the stenosis exceeded 70% or 50% for the left main coronary artery.

2.2. Perioperative data

Perioperative data consisted of the number of bypasses performed, whether the revascularization was complete or not, the use of cardiopulmonary bypass or off-pump surgery, and the performance of any combined surgery (i.e. concomitant valvular or carotid surgery).

2.3. Outcomes

The postoperative outcomes were collected within the first month following surgery. The primary outcome was composite, including death, non-fatal myocardial infarction, non-fatal stroke or TIA. Non-fatal myocardial infarction was defined by the occurrence of a Q-wave in at least two contiguous ECG derivations and/or a postoperative Troponin-I (cTnI) peak >20 µg/l. Non-fatal strokes or transient ischemic attacks (TIA) were diagnosed by any physician taking care of the patient during this period, and attested by a neurologist in borderline cases requiring confirmation. The clinical events (death and stroke or TIA) and cTnI peak were also studied separately as secondary outcomes.

2.4. Statistical methods

Data are reported as mean ± SD. In the univariate analysis, risk factors for the occurrence of the primary outcome were analyzed using the χ²-test for discrete variables and Student’s t-test for continuous variables. Multiple logistic regression analysis was used to determine the model with independent predictive factors. For this purpose all factors were initially introduced in the multivariate model (initial model). Using a stepwise descending procedure, all the factors with a p > 0.15 were excluded from the analysis leading to a final model. Age, sex, SBP, the use of beta-blockers and a low LVEF were systematically forced into the model as adjusting variables. All the models have been repeated once after the exclusion of patients who underwent combined coronary and valvular surgery. For all the tests, a p-value <0.05 was considered as significant. These statistical analyses were performed using the Statview 5.0 software (SAS institute, Cary, NC).

3. Results

A total of 1022 patients were enrolled during the inclusion period. Table 1 displays general characteristics of the population. Mean HR in the study population was at 65.8 ± 13.5 beats per minute (bpm). The distribution of HR in the population, both overall and according to the use of beta-blocking therapy, is displayed in Fig. 1. Heart rate at entry was >80 bpm for 11.7% of patients (5% and 22.3% respectively in those with and without beta-blockers, p < 0.05). Mean PP in the study population was at 21.4 ± 10.9 mmHg. The distribution of PP in the population is displayed in Fig. 2. Pulse pressure exceeded 70 mmHg in 11.3% of patients (13.3% and 9.9% respectively in those with and without a history of hypertension, p = ns).

During the first postoperative month, 33 patients (3.2%) died. Thirty-seven patients (3.6%) experienced stroke or TIA.
Postoperative myocardial infarction occurred in 118 patients (12.1%). Overall, 146 patients (15.0%) experienced the primary composite outcome. They were then compared to the remaining 876 event-free patients (Table 2). In the univariate analysis, the following factors differed significantly between these two groups: sex ratio, renal failure, NYHA stages III or IV at admission, low LVEF (<0.40), unstable cardiac status at admission, combined valvular surgery and heart rate. Of note, the mean PP did not differ significantly between the two groups. However, while a steady positive association was observed between HR and the primary outcome (Fig. 3), the association between PP and primary outcome presented a ‘U-shape’ pattern, with the highest rates of events for those with PP <40 or >70 mmHg (Fig. 4). This specific pattern was also noted when the three types of events (death, stroke/TIA, and myocardial infarction) were studied separately. As a consequence, in the following analyses, PP has not been assessed as a continuous variable but three categories were defined: low PP (<40 mmHg), normal PP (40—70 mmHg) and high PP (>70 mmHg). Fig. 5 presents the postoperative events rates according to preoperative HR and pulse pressure.

The multivariate model exploring the association of all potential variables at risk and the primary outcome are displayed in Table 3. The final model was obtained after excluding stepwise all the non-significant variables. Heart rate was significantly associated with the occurrence of the primary outcome, even after adjusting for age, sex, SBP, low LVEF and the use of beta-blockers. Similarly, in the same model, high PP was significantly associated with the primary outcome, while only a trend was noted for low PP. Likewise, when only clinical endpoints (death, stroke or TIA) were combined as a secondary outcome (Table 3), high PP

### Table 1
Baseline characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean value or prevalence</th>
</tr>
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<tbody>
<tr>
<td><strong>Preoperative data</strong></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>66.9 ± 9.2</td>
</tr>
<tr>
<td>Female sex</td>
<td>186 (18.2%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>297 (29.1%)</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>610 (60.0%)</td>
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<tr>
<td>Hypertension</td>
<td>492 (48.2%)</td>
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<tr>
<td>Diabetes</td>
<td>268 (26.3%)</td>
</tr>
<tr>
<td>Body mass index (BMI) &gt;30 kg/m²</td>
<td>179 (17.7%)</td>
</tr>
<tr>
<td>Renal failure</td>
<td>48 (4.7%)</td>
</tr>
<tr>
<td>NYHA stages III or IV</td>
<td>157 (15.4%)</td>
</tr>
<tr>
<td>Supraventricular arrhythmia</td>
<td>89 (8.7%)</td>
</tr>
<tr>
<td>Low ejection fraction (&lt;40%)</td>
<td>98 (9.6%)</td>
</tr>
<tr>
<td>redo surgery</td>
<td>48 (4.7%)</td>
</tr>
<tr>
<td>Left main coronary artery stenosis &gt;50%</td>
<td>173 (17.0%)</td>
</tr>
<tr>
<td>Triple-vessels disease</td>
<td>661 (64.8%)</td>
</tr>
<tr>
<td>Unstable cardiac status</td>
<td>161 (15.9%)</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
<td>143 (14.0%)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>98 (9.6%)</td>
</tr>
<tr>
<td>Beta-blocker therapy</td>
<td>623 (60.1%)</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>133.4 (14.8)</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>75.4 (7.6)</td>
</tr>
<tr>
<td><strong>Operative data</strong></td>
<td></td>
</tr>
<tr>
<td>Off-pump surgery</td>
<td>136 (13.3%)</td>
</tr>
<tr>
<td>Number of bypasses</td>
<td>3.0 ± 1.0</td>
</tr>
<tr>
<td>Complete revascularization</td>
<td>872 (85.4%)</td>
</tr>
<tr>
<td>Combined with valve surgery</td>
<td>160 (15.7%)</td>
</tr>
<tr>
<td>Combined with vascular surgery</td>
<td>33 (3.2%)</td>
</tr>
</tbody>
</table>

### Table 2
Predictive factors of occurrence of postoperative cardiovascular event (primary outcome)

<table>
<thead>
<tr>
<th>Factors</th>
<th>Event-free group (n = 876)</th>
<th>Event group (n = 146)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>66.8 ± 9.3</td>
<td>68.1 ± 8.8</td>
</tr>
<tr>
<td>Female sex</td>
<td>133 (16.1%)</td>
<td>38 (21.9%) ***</td>
</tr>
<tr>
<td>Smoking</td>
<td>240 (29.1%)</td>
<td>43 (29.5%)</td>
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<tr>
<td>Hypercholesterolemia</td>
<td>492 (59.7%)</td>
<td>87 (60.0%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>401 (48.5%)</td>
<td>67 (45.9%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>215 (26.0%)</td>
<td>37 (25.5%)</td>
</tr>
<tr>
<td>Low ejection fraction (&lt;40%)</td>
<td>68 (5.7%)</td>
<td>20 (13.8%)</td>
</tr>
<tr>
<td>Renal failure</td>
<td>27 (3.3%)</td>
<td>12 (8.2%) **</td>
</tr>
<tr>
<td>NYHA stages III or IV</td>
<td>115 (13.1%)</td>
<td>34 (23.3%) ***</td>
</tr>
<tr>
<td>Supraventricular arrhythmia</td>
<td>63 (7.6%)</td>
<td>18 (12.3%)</td>
</tr>
<tr>
<td>Low LVEF (&lt;40%)</td>
<td>68 (8.2%)</td>
<td>20 (13.8%)</td>
</tr>
<tr>
<td>redo surgery</td>
<td>34 (4.1%)</td>
<td>11 (7.5%)</td>
</tr>
<tr>
<td>LMCA stenosis &gt;50%</td>
<td>135 (16.3%)</td>
<td>27 (18.5%)</td>
</tr>
<tr>
<td>Triple-vessels disease</td>
<td>525 (63.4%)</td>
<td>103 (70.5%)</td>
</tr>
<tr>
<td>Cardiopulmonary bypass</td>
<td>716 (86.5%)</td>
<td>130 (89.0%)</td>
</tr>
<tr>
<td>Complete revascularization</td>
<td>731 (85.4%)</td>
<td>101 (85.6%)</td>
</tr>
<tr>
<td>Unstable cardiac status</td>
<td>120 (14.5%)</td>
<td>32 (21.9%)</td>
</tr>
<tr>
<td>Beta-blocking therapy</td>
<td>526 (63.5%)</td>
<td>83 (57.2%)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>78 (9.4%)</td>
<td>17 (11.6%)</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
<td>108 (12.3%)</td>
<td>26 (17.8%)</td>
</tr>
<tr>
<td>Combined valvular surgery</td>
<td>109 (13.2%)</td>
<td>42 (28.8% ***</td>
</tr>
<tr>
<td>Combined carotid surgery</td>
<td>24 (2.9%)</td>
<td>8 (5.5%)</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>64.9 (13.2)</td>
<td>69.9 (14.3% ***</td>
</tr>
<tr>
<td>Systolic pressure (mmHg)</td>
<td>133.1 (13.5)</td>
<td>133.3 (16.0%</td>
</tr>
<tr>
<td>Diastolic pressure (mmHg)</td>
<td>75.7 (10.8)</td>
<td>74.9 (8.3)</td>
</tr>
<tr>
<td>Mean pressure (mmHg)</td>
<td>94.8 (10.0)</td>
<td>94.3 (9.7)</td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td>57.4 (12.9)</td>
<td>58.4 (12.8)</td>
</tr>
</tbody>
</table>

Univariate analysis. *p < 0.05; **p < 0.01; ***p < 0.0001.
presented a significant association with this endpoint, independent from other risk factors. In the same model, HR presented a borderline association ($p = 0.07$). Similar trends were found when outcomes were defined by each of the events used in the primary outcome separately, but the number of outcomes was too low to reach statistical significance. Of note, when replacing PP by mean blood pressure in the models, no significant association was found between the latter and outcome (data not shown).

In a second set of analysis, we excluded the 160 patients who benefited simultaneously from a valvular surgery; both HR and PP remained associated to the primary outcome, but the lower number of cases led to borderline results ($p < 0.10$, data not shown).

4. Discussion

In this study, we show that both HR and PP are predictive of poor postoperative prognosis, independent of other common risk factors usually used in different predictive scores. While HR presented a positive correlation with postoperative events, the association between PP and events was non-linear; with higher rates of CV events in those with a high-PP ($>70$ mmHg). Heart rate and PP were both independent predictors of adverse postoperative events.

A majority of epidemiological studies describe a significant relationship between a high resting HR and mortality, especially for cardiovascular mortality (for review see [3]). The analysis of these studies leads to a global estimation of 30–50% mortality excess for every 20 bpm increase of the resting HR. The more recent studies not only confirmed these results but also excluded confounding factors such as smoking, sedentary lifestyle, obesity, breathing disorders or blood abnormalities, which can be associated to HR acceleration.

Heart rate at rest is a marker of the autonomic nervous system balance, and high HR at rest reflects predominance of the sympathetic over the parasympathetic tone. The sympathetic nervous system is highly activated during cardiac surgery, favoring ischemia by increasing myocardial oxygen supply/demand mismatch, and also increasing platelet aggregation and decrease fibrinolytic activity, both facilitating coronary thrombosis [7]. While a higher perioperative HR is already considered as a risk factor for perioperative MI [4], and a tight HR control by the use of beta-blockers is of prognostic interest in non-cardiac surgery [8], especially in vascular surgery [9], little interest has surprisingly been made for preoperative HR in the risk stratification of patients undergoing cardiac surgery. Reviewing computerized operative data, Reich et al. [10] found that HR $>100$ bpm during the precardiopulmonary bypass phase was an independent risk factor for postoperative myocardial infarction, but not postoperative stroke or mortality. Puddu et al. [11] found similar results using a HR cut-off at 130 bpm. In another study, Fillinger et al. [12] found that a pre-induction HR $>80$ bpm was associated with increased mortality. All these studies used HR recorded during a period very close to CABG surgery, in the operating room. Conversely, our study used HR as recorded at patient’s admittance in our department, a period more adapted to the estimation of patient’s surgical risk.

While this observational study is not able to demonstrate that lowering HR preoperatively might be of prognostic benefit, it nevertheless underlines the usefulness of HR as a prognostic marker. Currently, none of the predictive scores used for risk stratification in cardiac surgery include HR as a variable. This might be explained by the fact that higher HR can also reflect other situations such as heart failure or unstable hemodynamic status, which are already included in
those risk scores. One of our major findings in this study is that even adjusting for these potential confounding biases, including the use of beta-blockers, preoperative HR remained an independent predictor of postoperative events.

Basically, PP is determined by the interaction of ventricular ejection with large-artery stiffness and with the wave reflection [13]. Several large-scale population-based studies showed PP as an independent predictor of fatal and non-fatal CV events, with a special note in hypertensive subjects (for review see [3]). Nawrot et al. [14] found that PP brought additive prognostic information to that provided by the Framingham score, and they even proposed to include the former in the latter, in order to enhance its predictive power. Pulse pressure is also strongly correlated with HR and PP as a marker of arterial stiffness, since PP is determined by the interaction of HR with large-artery stiffness and with the wave reflection [13].

To our knowledge, this is the first study assessing the predictive role of PP in the setting of the perioperative period. In a prospective multicenter study, Aronson et al. [20] reported a 40% increase of perioperative morbidity in those with an isolated systolic hypertension (SBP >140 mmHg with DBP <90 mmHg, or in other words, PP >50 mmHg). In that study, it is unclear whether high SBP per se or high PP was actually predictive of perioperative events. In our study, beyond several confounders and other risk factors, PP was independently associated to mortality, stroke, and the combined primary outcome, even after adjustments for SBP.

While HR is correlated to PP both in general [21] and hypertensive [22] populations, there is no direct relationship between HR and PP as a marker of arterial stiffness, since PP is not affected when HR is artificially increased by pacing at 65 up to 120 bpm [23]. Therefore, PP is not determined by HR and the combination of both markers is interesting for the evaluation of perioperative risk.

Our study has some limitations. First, it is an observational study, and despite our efforts to adjust our results to potential confounding bias, we cannot definitely exclude any residual bias. Second, due to the limited size of our cohort, our primary outcome combines several events and we were not able to find significant results for separate endpoints. The definition of postoperative MI using the 20 μg/l threshold cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI threshold for the definition of myocardial damage. Salamonsen et al. [24] stratified patients according to postoperative cTnI levels, and found higher rates of adverse outcomes in those with a cTnI peak >40 μg/l. In addition, when we only focused our combined criteria to clinical endpoints (stroke and death), we obtained consistent results. The definition of postoperative MI using the 20 μg/l threshold cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI threshold for the definition of myocardial damage. Salamonsen et al. [24] stratified patients according to postoperative cTnI levels, and found higher rates of adverse outcomes in those with a cTnI peak >40 μg/l. In addition, when we only focused our combined criteria to clinical endpoints (stroke and death), we obtained consistent results. The definition of postoperative MI using the 20 μg/l threshold cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI threshold for the definition of myocardial damage. Salamonsen et al. [24] stratified patients according to postoperative cTnI levels, and found higher rates of adverse outcomes in those with a cTnI peak >40 μg/l. In addition, when we only focused our combined criteria to clinical endpoints (stroke and death), we obtained consistent results. The definition of postoperative MI using the 20 μg/l threshold cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI concentration is arbitrary, since there is no consensus regarding this cut-off. However, we used a higher cut-off than several other studies, in order to consider only a substantial increase of cTnI threshold for the definition of myocardial damage. Salamonsen et al. [24] stratified patients according to postoperative cTnI levels, and found higher rates of adverse outcomes in those with a cTnI peak >40 μg/l.
events in our study, our primary aim is to reveal PP as a risk marker, rather than a risk factor, suggestive of a direct causative relationship. Similarly, aortic valve diseases (regurgitation or stenosis) might affect PP, but the PP-adverse events relationship remained significant after adjusting for valve surgery, or when combined surgeries were excluded from the analyses. Finally, while we found that the association between HR and adverse events was independent of beta-blocker therapy, we are not able to confirm whether this was due to an inadequate dosage of this group of treatment, and whether a tighter HR control could reduce the postoperative events rates. In addition, the cohort size was not large enough to be able to detect different results according to beta-blocking agents particularities (e.g. intrinsic sympathomimetic activity) and/or discontinuation during the immediate postoperative period. Finally, even though we included cases with combined valvular and coronary surgery, our study has been focused on coronary surgery, and our results may not be applicable for non-coronary cardiac surgery (e.g. isolated valvular surgery).

5. Conclusion

In this study, we found that two clinical parameters, heart rate and pulse pressure, obtained very easily preoperatively in a patient at rest, are both associated to adverse postoperative outcomes, combining clinical (stroke, death) and biological (new Q-wave on ECG and/or cTnI raise) events. These associations are independent of other potential confounding factors, which are usually considered in different risk scores available for the assessment of perioperative risk in cardiac surgery. While further studies are required to clarify whether our findings are related to a causative relationship or only as a marker of other underlying conditions, we suggest that these two parameters should be considered in future attempts to improve risk prediction in patients who undergo coronary bypass surgery.

References


