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Association between postoperative mean arterial blood pressure and myocardial injury after noncardiac surgery

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Abstract

Background: Myocardial injury after noncardiac surgery is common, although the exact pathophysiology is unknown. It is plausible that hypotension after surgery is relevant for the development of myocardial injury. The authors evaluated whether low mean arterial pressures (MAPs) after surgery are related to an increased incidence in postoperative cardiac-troponin elevation.

Methods: A prospective cohort of 2211 patients aged ≥60 yr, undergoing major or moderate noncardiac surgery in The Netherlands, was retrospectively analysed for the occurrence of postoperative cardiac-troponin elevation [high-sensitive troponin T (hsTnT) >14 ng L^{-1}]. Blood pressures after surgery were recorded and divided into quartiles based on the lowest MAP prior to peak troponin recording. The association between MAP and extent of postoperative cardiac-troponin elevation was analysed.

Results: The patients were divided into quartiles based on their lowest MAP in the period preceding the peak hsTnT, ranging from a median of 62 in the lowest quartile to 94 in the highest quartile. Postoperative hsTnT elevation was present in 53.2% of the population. An association between MAP quartile and postoperative peak hsTnT was predominantly observed in the lowest quartile ($P<0.001$): median hsTnT 17.6 (10.3–37.3), 14.9 (9.4–24.6), 13.8 (9.1–22.5), and 14.0 (9.2–22.4). The multivariable logistic-regression analysis showed an increased risk for postoperative cardiac-troponin elevation with decreasing MAP thresholds.

Conclusions: Lower postoperative blood pressure is associated with an increased incidence of postoperative cardiac hsTnT elevation, irrespective of pre- and intraoperative variables.

Keywords: hypotension; observational study; surgery; troponin

Myocardial injury after noncardiac surgery is common, and is an independent risk factor for 30-day mortality and for 1 yr mortality in patients undergoing noncardiac surgery.[1–3] Although the exact pathophysiology of myocardial injury is unknown, similar pathways probably exist between postoperative myocardial injury and postoperative myocardial infarction.[4] Tachycardia, anaemia, hypoxaemia, and

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Editor's key points

- The role of postoperative hypotension in myocardial injury after noncardiac surgery is unclear.
- A retrospective analysis of a single-centre cohort of 2211 patients found a significant association between low mean arterial pressure and elevation of cardiac troponin.
- Further study is required to determine if postoperative hypotension is a modifiable risk factor for myocardial injury.

hypotension are all common after surgery, and can result in the development of myocardial injury because of a supply-and-demand mismatch.[5] Studies in the previous decade have mainly focused on the role of postoperative heart rate rather than postoperative blood pressure. In contrast, intraoperative hypotension has been studied extensively in patients undergoing surgery. Although various definitions of intraoperative hypotension have been used, it appears to be an important risk factor for myocardial injury, stroke, acute kidney injury, and mortality.[6–12] An important limitation in studying the effects of intraoperative arterial pressure is the confounding effect of surgical stimuli, pain, intravascular volume status, anaesthetics, and use of inotropic or vasopressive agents. In the postoperative phase, many of these limitations are overcome, and postoperative arterial pressure is the result of the combined response to anaesthetic effects, volume status, cardiac depressant effects, and inflammatory response.

As troponin release is most prominent in the early hours and 1st day after surgery, (early) postoperative hypotension could play a crucial role in the development of myocardial injury. The effects of postoperative hypotension on myocardial injury and outcome after noncardiac surgery have not been studied previously. In this study, we investigated the association between postoperative arterial pressures and postoperative cardiac-troponin elevation after noncardiac surgery.

Methods

Study design

This was a retrospective study, selected from an ongoing prospective registry of patients undergoing noncardiac surgery at the Erasmus Medical Centre, Rotterdam, The Netherlands. Consecutive patients aged ≥60 yr who are scheduled for intermediate- or high-risk noncardiac surgery are under postoperative surveillance for postoperative cardiac-troponin elevation during the first 3 postoperative days through (high-sensitive) troponin T (hsTnT) measurements. All patients, regardless of the type of anaesthesia or setting (elective of emergency), are included in this registry. For this observational study, we included all patients between the start of the surveillance protocol in July 2012 through July 2014. The institutional approval for this study was obtained. This study was not subject to the Dutch Medical Research Involving Human Subjects Act. Therefore, the ethics committee of the Erasmus Medical Centre waived the requirement for a written informed consent. This study complies with the Declaration of Helsinki on research ethics.[13] The primary outcome was occurrence of postoperative cardiac-troponin elevation (hsTnT above the 99th percentile: 14 ng L^{-1}). The secondary outcomes included myocardial infarction (based on the third universal definition[14]) and 30-day mortality.

Data collection

Data were extracted from the electronic-hospital patient information system. Baseline data included age, sex, type of surgery, and past medical-history variables, including hypertension, diabetes mellitus, coronary heart disease (history of angina pectoris, previous myocardial infarction, percutaneous coronary intervention, or coronary artery bypass grafting), chronic heart failure, previous cerebrovascular disease (cerebrovascular accident or transient ischaemic attack), chronic obstructive pulmonary disease, renal failure (preoperative creatinine concentration above 177 μmol L^{-1}), and peripheral arterial disease. Additionally, the preoperative use of beta blockers, statins, angiotensin-converting-enzyme (ACE) inhibitors, angiotensin receptor blockers, diuretics, calcium-channel blockers, nitrates, aspirin, and oral anticoagulants was retrieved from the electronic medical files. Survival status was completed in all patients by means of the institution's medical records, or was ascertained by inquiry from the civil registries.

Haemodynamic data and troponin measurements

Arterial blood pressures and heart rates measured on the ward on the day of surgery or the evening before surgery were used as baseline preoperative haemodynamic data. Intraoperative arterial pressures and heart rates were extracted from the anaesthesia information monitoring system, and were cleaned using an algorithm previously described.[15] Blood loss was noted at the end of the operation, as reported by the anaesthesiologist. The most recent haemoglobin concentration before surgery and the lowest haemoglobin concentration within 30 days after surgery were noted, irrespective of the peak hsTnT concentration. After surgery, all patients were admitted to the post-anaesthesia care unit, or were transferred immediately to a high-dependency ward/intensive care unit (ICU), depending on the patients' health status and the perioperative course of the surgery. All blood pressures, recorded on the ward and in the high-dependency ward/ICU up to 3 days after discharge from the post-anaesthesia care unit, were retrieved from the hospital information system. As per hospital protocol, non-invasive blood pressure is measured at least once every 8 h on the ward, or is recorded at 5 min intervals for patients with invasive blood-pressure monitoring. Heart rate was recorded simultaneously with all blood pressures. Measurements of hsTnT were routinely obtained in the morning during the first 3 postoperative days, unless discharged earlier, or whenever clinically indicated by the treating physician using the Cobas e602 Troponin T hs STAT assay (Roche Diagnostics, Mannheim, Germany). For each patient, the highest value of all routine hsTnT measurements in the 3 postoperative days was used in the analysis. An hsTnT <14 ng L^{-1} is considered normal and the 99th percentile. We defined hsTnT of 14–50 ng L^{-1} as low elevation, hsTnT of 50–150 ng L^{-1} as moderate elevation, and hsTnT >150 ng L^{-1} as high elevation.[16]

Statistical analysis

The patients were divided into quartiles based on their lowest mean arterial pressure (MAP) prior to peak hsTnT.

For patients who had a peak hsTnT on the 1st postoperative day, the MAP recorded on the evening of the surgery was used for the analysis. We did not choose a threshold for hypotension because there is no widely accepted definition. Baseline characteristics were described as counts and percentages (dichotomous variables), or means and SDs (continuous variables). HsTnT is presented as median and inter-quartile ranges because the data distribution was skewed. Differences in baseline characteristics between MAP quartiles were determined using Pearson's χ^2 analysis and ANOVA or Kruskal–Wallis test, where appropriate. Normality was assessed both visually with histograms and Q–Q plots, and with the Kolmogorov–Smirnov test. HsTnT was log transformed because of non-normal distribution. To account for the unknown direction of the effect (i.e. hypotension causing postoperative cardiac-troponin elevation or postoperative cardiac-troponin elevation causing hypotension), correlation analyses were repeated with the lowest MAP within the first 3 postoperative days, irrespective of the peak hsTnT measurement.

Logistic-regression analyses were performed with postoperative cardiac-troponin elevation as the dependent variable and different MAP threshold values as the independent variable. Multivariable analyses included age, sex, diabetes mellitus, coronary heart disease, chronic heart failure, previous cerebrovascular disease, chronic obstructive pulmonary disease, renal failure, type of surgery, emergency surgery, preoperative haemoglobin concentration, intraoperative MAP, and heart rate at the time of MAP measurement. A sensitivity analysis was performed in patients who were admitted to a high-dependency ward.

A two-sided *P*-value of <0.05 was considered significant. Analyses were performed using RStudio and IBM SPSS 21.0 statistical software (SPSS Inc., Chicago, IL, USA). Figures were made using GraphPad Prism, version 6.00 (GraphPad, San Diego, CA, USA).

Results

A total of 3176 patients aged ≥60 yr were included in our cohort in the specified period. Patients were excluded if no troponin measurements (N=954) were available in the first 3 postoperative days. Of the 2222 eligible patients aged above 60 yr with troponin measurements, 11 patients (0.5%) had no blood-pressure recordings in the hospital information system before the peak hsTnT was recorded and were excluded from the analysis. The final study sample consisted of 2211 patients (Fig. 1).

The patients were divided into quartiles based on their lowest postoperative MAP prior to peak hsTnT. The peak

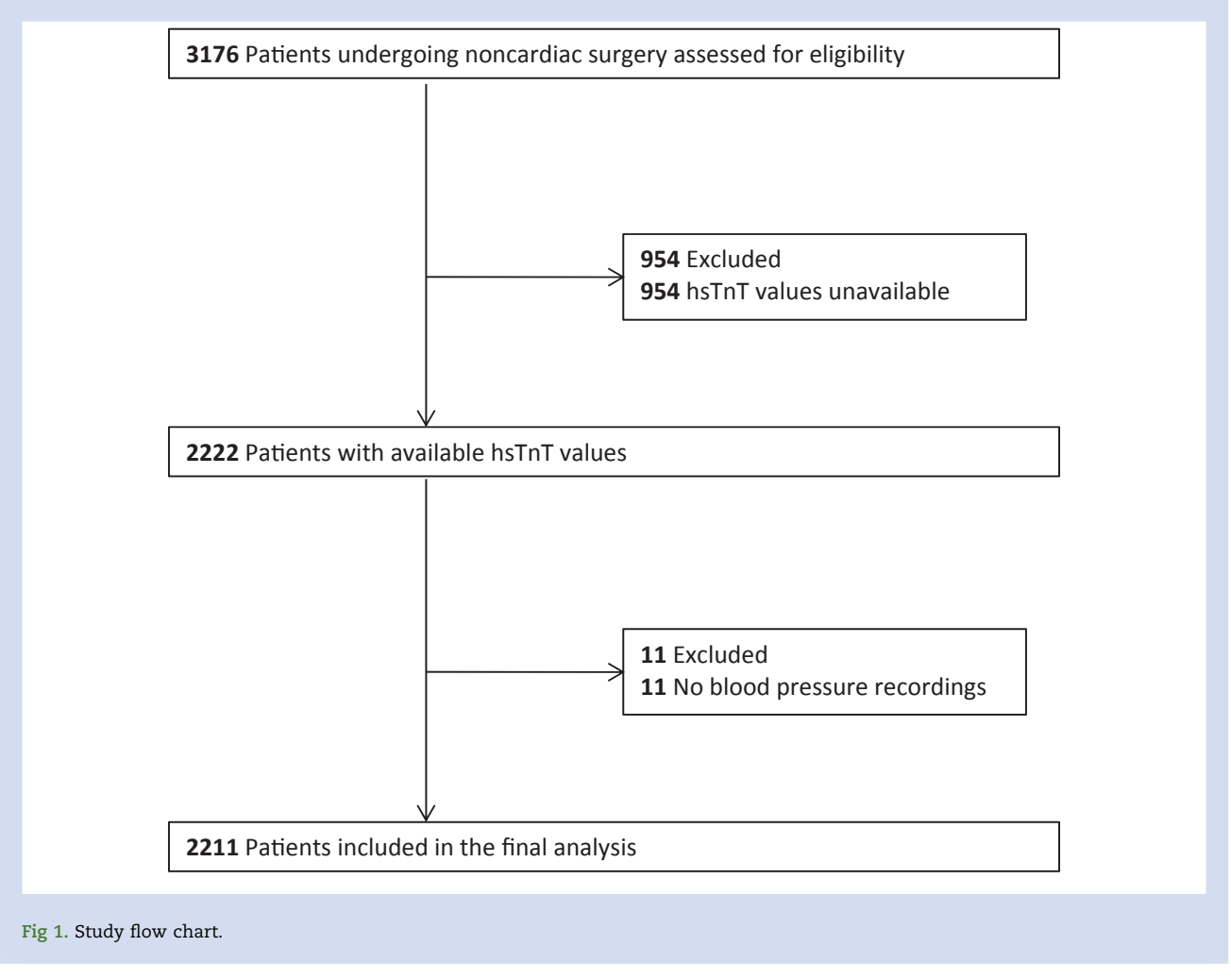

Fig 1. Study flow chart.

hsTnT during the first 3 postoperative days was recorded on the 1st postoperative day in 61% of the patients. In 26% of the patients, the peak hsTnT was found on the 2nd postoperative day, and in 13%, it was found on the 3rd day. In 79% of patients, the lowest MAP prior peak hsTnT was measured 1 day before hsTnT peaked. The baseline characteristics are presented in Table 1. Patients in the lowest quartile had a higher incidence of previous myocardial infarction, coronary artery disease, chronic heart failure, diabetes mellitus, and chronic obstructive pulmonary disease ($P<0.05$ for all). Renal failure was more prevalent in the highest quartile ($P<0.001$). Relevant pre-, intra-, and postoperative haemodynamic data; hsTnT data; and clinical end points are shown in Table 2.

An association between MAP quartiles and postoperative peak hsTnT was predominantly observed in the lowest quartile ($P<0.001$): median hsTnT 17.6 (10.3–37.3), 14.9 (9.4–24.6), 13.8 (9.1–22.5), and 14.0 (9.2–22.4). The 30-day mortality was highest in the lowest MAP quartile (Table 2).

MAP, as a continuous variable, was independently associated with postoperative cardiac hsTnT elevation [odds ratio (OR)=0.991, 95% confidence interval (CI), 0.983–0.999]. A sensitivity analysis in patients admitted to a high-dependency ward revealed comparable results (OR=0.984, 95% CI, 0.970–0.998), whilst significance was lost in patients admitted to a regular ward (OR=−0.994, 95% CI, 0.983–1.006). To graphically represent the OR as a function of different threshold values for low MAP, the associations between a wider range of low MAP thresholds and postoperative cardiac hsTnT elevation were calculated with univariable and multivariable logistic-regression analysis (Fig. 2).

Discussion

In this retrospective single-centre cohort study, we investigated the possible association between postoperative hypotension and postoperative increases in high-sensitive troponin T (hsTnT) as a marker for perioperative myocardial

Table 1 Patient baseline characteristics. Values are numbers with percentages unless otherwise specified. *P*-values were calculated with χ^2 test for categorical variables, and ANOVA or Kruskal–Wallis test for continuous variables. *Lowest MAP before peak HsTnT. MAP, mean arterial pressures

	Total cohort (*N*=2211)	First MAP quartile (*N*=543)	Second MAP quartile (*N*=551)	Third MAP quartile (*N*=569)	Fourth MAP quartile (*N*=548)	*P*-value
Lowest MAP,* median	76.7	62.0	71.7	81.3	94.3	
Lowest MAP, minimum–maximum	31.0–122.3	31.0–67.0	67.3–76.3	76.7–86.0	86.7–122.3	
Age (yr), mean (SD)	70.5 (6.9)	71.5 (7.1)	70.7 (6.9)	70.0 (6.9)	69.8 (6.8)	<0.001
Sex, male	1298 (58.7)	296 (54.5)	321 (58.3)	340 (59.8)	341 (62.2)	0.070
Hypertension	1211 (54.8)	305 (56.2)	292 (53.0)	309 (54.3)	305 (55.7)	0.716
Coronary artery disease	378 (17.1)	105 (19.3)	112 (20.3)	97 (17.0)	64 (11.7)	0.001
Myocardial Infarction	276 (12.5)	88 (16.2)	79 (14.3)	56 (9.8)	53 (9.7)	0.001
Chronic heart failure	118 (5.3)	46 (8.45)	33 (6.0)	29 (5.1)	10 (1.8)	<0.001
Cerebrovascular accident or transient ischaemic attack	344 (15.6)	95 (17.5)	103 (18.7)	69 (12.1)	77 (14.1)	0.008
Diabetes mellitus	528 (23.9)	152 (28.0)	131 (23.8)	126 (22.1)	119 (21.7)	0.060
Renal failure	168 (7.7)	33 (6.2)	27 (5.0)	37 (6.6)	71 (13.0)	<0.001
Chronic obstructive pulmonary disease	308 (13.9)	89 (16.4)	92 (16.7)	79 (13.9)	48 (8.8)	<0.001
Peripheral artery disease	272 (12.3)	73 (13.4)	73 (13.2)	61 (10.7)	65 (11.9)	0.470
Beta blockers	839 (37.9)	227 (41.8)	214 (38.8)	211 (37.1)	187 (34.1)	0.066
Statins	979 (44.3)	252 (46.4)	255 (46.3)	246 (43.2)	226 (41.2)	0.241
ACE inhibitor	529 (23.9)	149 (27.4)	126 (22.9)	123 (21.6)	131 (23.9)	0.128
Angiotensin II antagonists	409 (18.5)	95 (17.5)	104 (18.9)	118 (20.7)	92 (16.8)	0.338
Diuretics	637 (28.8)	165 (30.4)	167 (30.3)	157 (27.6)	148 (27.0)	0.468
Calcium-channel blocker	456 (20.6)	98 (18.0)	119 (21.6)	112 (19.7)	127 (23.2)	0.171
Nitrates	137 (6.2)	43 (7.9)	45 (8.2)	24 (4.2)	25 (4.6)	0.005
Aspirin	706 (31.9)	185 (34.1)	185 (33.6)	182 (32.0)	154 (28.1)	0.137
Oral anticoagulant	252 (11.4)	79 (14.5)	57 (10.3)	60 (10.5)	56 (10.2)	0.069
Emergency surgery	188 (8.5)	58 (10.7)	47 (8.5)	37 (6.5)	46 (8.4)	0.100
Type of surgery						<0.001
Orthopaedic	391 (17.7)	74 (13.6)	108 (19.6)	126 (22.1)	83 (15.1)	
General	266 (12.0)	107 (19.7)	78 (14.2)	44 (7.7)	37 (6.8)	
Urologic of gynaecologic	343 (15.5)	71 (13.1)	84 (15.2)	78 (13.7)	110 (20.1)	
Vascular	513 (23.2)	138 (25.4)	127 (23.0)	137 (24.1)	111 (20.3)	
Other	698 (31.6)	153 (28.2)	154 (27.9)	184 (32.3)	207 (37.8)	
Type of anaesthesia						
General anaesthesia	2105 (95.2)	528 (97.2)	531 (96.4)	535 (94.0)	511 (93.2)	0.005
Epidural	149 (6.7)	85 (15.7)	44 (8.0)	8 (1.4)	12 (2.2)	<0.001
Spinal	72 (3.3)	8 (1.5)	16 (2.9)	18 (3.2)	30 (5.5)	0.003
Regional anaesthesia	190 (8.6)	71 (13.1)	59 (10.7)	43 (7.6)	17 (3.1)	<0.001
Local anaesthesia	29 (1.3)	3 (0.6)	10 (1.8)	7 (1.2)	9 (1.6)	0.263
High-dependency ward	932 (42.3)	382 (70.7)	271 (49.2)	179 (31.5)	100 (18.3)	<0.001

Table 2 Pre-, intra-, and postoperative haemodynamic values and outcome data. MAP, mean arterial pressures

Characteristics		Total cohort (*N*=2211)	First MAP quartile (*N*=543)	Second MAP quartile (*N*=551)	Third MAP quartile (*N*=569)	Fourth MAP quartile (*N*=548)	*P*-value
Preoperative							
Haemoglobin (mmol L^{-1})	2188	8.3 (7.4–9.0)	8.0 (7.1–8.8)	8.3 (7.4–9.0)	8.3 (7.4–9.1)	8.5 (7.6–9.1)	<0.001
Heart rate	2163	73.0 (65.0–83.0)	74.0 (65.3–84.0)	73.0 (66.0–83.0)	72.0 (64.0–82.0)	73.5 (65.0–82.0)	0.278
MAP	2166	99 (89.7–108.3)	96.3 (86.0–106.5)	96.0 (87.6–105.4)	99.3 (90.76–107.8)	103.7 (94.3–112.0)	<0.001
Intraoperative							
Lowest MAP	2103	55 (47–62)	50.0 (43.0–58.0)	54.0 (48.0–61.0)	57.0 (50.0–64.0)	59.0 (51.0–67.0)	<0.001
Median MAP	2103	81.0 (74.0–88.0)	77.0 (71.0–83.0)	80.0 (74.0–86.0)	81.0 (75.3–88.0)	85.0 (79.0–92.0)	<0.001
Maximal heart rate	2103	94 (81–110)	98.0 (84.0–114.0)	93.0 (81.0–111.0)	93.5 (81.0–108.0)	92.0 (79.8–107.3)	<0.001
Median heart rate	2103	62 (56–71)	63.0 (57.0–72.0)	62.0 (55.0–71.0)	62.0 (55.0–70.8)	62.0 (56.0–70.0)	<0.001
Blood loss (ml)	2206	150 (0–472)	300 (50.0–850.0)	200 (9–500)	150 (0–400)	100 (0–250)	<0.001
Postoperative							
Heart rate	2211	73.0 (64.0–84.0)	70.0 (61.0–81.0)	72 (64.0–82.0)	73.0 (63.0–83.0)	75.0 (67.0–87.0)	<0.001
Haemoglobin (mmol L^{-1})	1954	6.5 (5.5–7.5)	5.9 (5.1–6.9)	6.6 (5.5–7.4)	6.8 (5.8–7.6)	7.0 (6.0–7.8)	<0.001
Peak hsTnT (ng L^{-1})							
<14		1035 (46.8)	207 (38.1)	261 (47.4)	292 (51.3)	275 (50.2)	
14–50		917 (41.5)	229 (42.2)	229 (41.6)	229 (40.2)	230 (42.0)	
50–150		186 (8.4)	72 (13.3)	46 (8.3)	38 (6.7)	30 (5.5)	
>150		73 (3.4)	35 (6.4)	15 (2.7)	10 (1.8)	13 (2.4)	
hsTnT (median, 25th–75th percentile)		14.9 (9.4–25.8)	17.6 (10.3–37.3)	14.9 (9.4–24.6)	13.8 (9.1–22.5)	14.0 (9.2–22.4)	<0.001
Outcome data							
Myocardial infarction		62 (2.8)	26 (4.8)	16 (2.9)	8 (1.4)	12 (2.2)	0.005
30-day mortality		80 (3.6)	38 (7.0)	16 (2.9)	11 (1.9)	15 (2.7)	<0.001

injury in patients above 60 yr who underwent noncardiac surgery. We found that the incidence of postoperative hypotension was high and that postoperative hypotension early after surgery on high-dependency units was associated with postoperative cardiac hsTnT elevation.

Low blood pressures after surgery were very common in our study population, with half of patients having an MAP <77 mm Hg. Postoperative cardiac hsTnT elevation was also common in our study population with 53% of the patients having a value above the reference value (i.e. >14 ng L^{-1}) and 12% >50 ng L^{-1}. Both cut-off values showed a similar relationship between MAP and postoperative hsTnT elevation. After multivariable adjustments, the postoperative MAP proved to be an independent risk factor for postoperative cardiac-troponin elevation after noncardiac surgery.

Secondly, an inverse relationship was noted between heart rate and postoperative cardiac hsTnT elevation. Given the fact that myocardial injury can result from an imbalance between myocardial oxygen supply and demand in patients with pre-existing stable coronary artery disease, low blood pressure

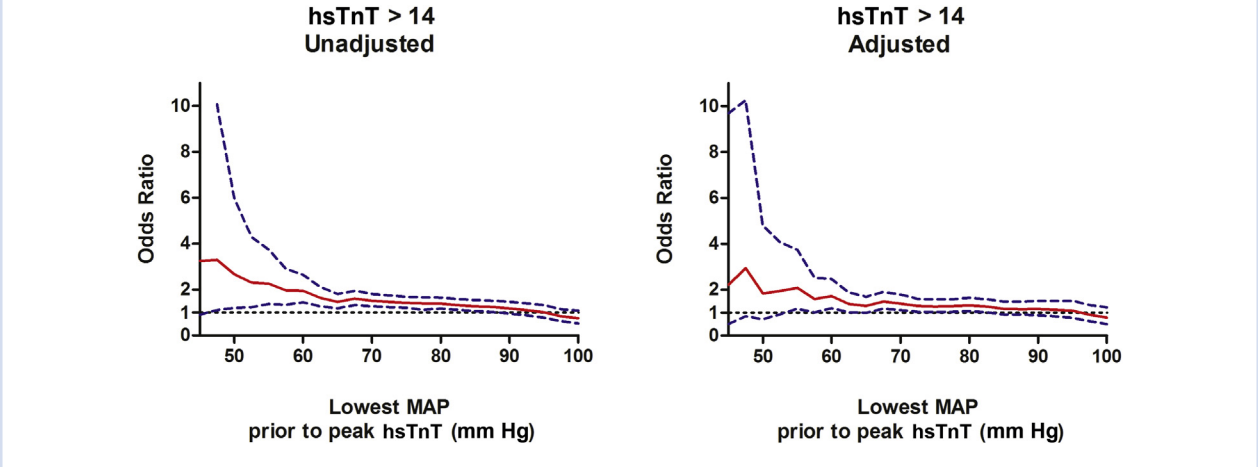

Fig 2. Risk of myocardial injury as a function of different mean arterial pressures (MAP) threshold values. Multivariable analyses included age, sex, diabetes mellitus, coronary heart disease, previous myocardial infarction, chronic heart failure, previous cerebrovascular disease, chronic obstructive pulmonary disease, and renal failure. Dashed lines represent the 95% CI.

during and after surgery is more likely to contribute to the presence and extent of myocardial injury than elevated postoperative heart rate.

Intraoperative hypotension has been shown to be an important risk factor for adverse outcome.[7,10,17,18] However, postoperative hypotension has not been carefully studied as a possible predictor of myocardial injury. Furthermore, there are no uniform definitions for either intra- or postoperative hypotension.

After reviewing 130 publications, Bijker and colleagues[11] identified 140 different definitions of intraoperative hypotension. The most frequently used definitions were a reduction of systolic arterial pressure <80 mm Hg, or a relative decrease of the systolic arterial pressure by >20% of the preoperative baseline value. Other definitions refer to the total or relative reduction in MAP.[10]

This raises an important question as to whether we should focus on systolic or mean arterial blood pressure. Arterial elastance can be approximated from knowledge of total peripheral resistance, total arterial compliance, aortic characteristic impedance, and systolic and diastolic time intervals; parameters that can be derived from pressure; and flow measured in the ascending aorta. It shows a clear dependence on age. With increased age, the compliance of the arterial system is reduced and elastance is increased. Consequently, increased age increases the dependency of systolic arterial pressure on stroke volume, and thus pulse pressure is elevated. As autoregulation of the most important organs is primarily dependent on MAP and systolic arterial pressure is not directly proportional because of the described relationships, the MAP should be used instead of systolic arterial pressure from a pathophysiological perspective. Therefore, we studied the mean instead of the systolic arterial pressures. Furthermore, we looked at MAP as a continuous variable rather than converting it to a dichotomous number.

Walsh and colleagues[10] showed that the duration of intraoperative hypotension (MAP <55 mm Hg) as an independent factor is associated with the onset of acute kidney injury, perioperative myocardial ischaemia, and increased mortality. An additional harmful effect was found if the duration of hypotension was taken into account, in patients who had a MAP of <55 mm Hg. Above a threshold of 55 mm Hg, this relationship was found only for acute kidney injury.

Because of their interventional nature, the Perioperative Ischemic Evaluation (POISE) trials have shown the possibility of a causal relationship, although mediated through beta blockers and clonidine, between hypotension and poor outcome.[17,18] Both studies show the relationship between various cardiovascular events (i.e. death, myocardial infarction, and stroke) and hypotension. Whilst a more subjective definition for hypotension was used in these trials, both POISE 1 and 2 reported an increased risk for myocardial infarction for clinically important hypotension, with hypotension being most prevalent during surgery. Our results extend these findings from previous studies and reveal new insights regarding the magnitude and relevance of hypotension in the perioperative period.

Our study differs in various ways from previous work. First, we chose to study postoperative instead of intraoperative hypotension. In contrast to intraoperative and early postoperative hypotension in the post-anaesthesia care unit, where blood pressures are measured on regular short intervals, blood-pressure measurement on the ward is often limited to single measurements every 8 h on non-high-dependency wards. Consequently, we were not able to take the duration of hypotension into account. Because of the much lower frequency of blood-pressure measurement and the absence of vasoactive treatment on general wards, it is likely that low blood pressures are tolerated for much longer periods of time.

Whether the recorded levels are accurate and illustrative of true physiology is debatable. There was no significant relationship in a sensitivity analysis of patients admitted to a regular ward, whilst the relationship remained significant in patients admitted to a high-dependency ward. Unfortunately, we cannot conclude from this that the relation no longer exists. It might as well be that the relationship is still present, but we are unable to show the relationship because of the low frequency of blood-pressure recordings on the regular ward, especially when the patients are asleep and blood pressure is usually low. Second, the blood-pressure value indicating an increased risk differs from previous work. Whilst critical intraoperative arterial pressure from the literature is an MAP of ~55 mm Hg,[7,10] the critical value for postoperative arterial pressure in our study seems to be around 70–80 mm Hg.

Our study has several important limitations. First, in our population, arterial pressures were measured in various ways and at various intervals. Patients admitted to a high-dependency ward more often had invasive pressure monitoring with more frequent recording compared to patients on the ward. This might lead to inaccuracy and bias in recording hypotension. Secondly, protocol adherence was suboptimal in the beginning, resulting in missing troponin values. Thirdly, because we did not measure troponin before surgery, pre-existing elevated troponin could not be taken into account. This is important, because elevated troponin concentrations can be present before operation in patients with renal failure, and are common in patients with different cardiovascular conditions.[19,20]

Conclusions

This study shows that low MAP after surgery is independently correlated to postoperative cardiac-troponin elevation. Given the multifactorial causes of postoperative hypotension, it remains to be determined if hypotension is a modifiable risk factor for myocardial injury, and if more frequent postoperative arterial-pressure monitoring and proactive treatment of hypotension will improve outcomes. Our data suggest that we should monitor patients at risk continuously and treat hypotension promptly using the most appropriate therapy based on the most likely cause.

Authors' contributions

F.v.L. and S.H. had full access to all the data in the study, and take responsibility for the integrity of the data and the accuracy of the data analysis.
Study concept and design: F.v.L., F.W., H.B., S.H.
Acquisition, analysis, or interpretation of data: all authors.
Drafting of the manuscript: F.W., S.H., V.L., F.v.L.
Critical revision of the manuscript for important intellectual content: S.H., J.W.P., F.G., R.J.S., F.v.L.
Statistical analysis: F.W., S.H., H.B., F.v.L.
Administrative, technical, or material support: F.W., S.H., V.L.
Study supervision: S.H., F.v.L.

Declaration of interest

None declared.

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