Original Article

The Impact of Mean Arterial Pressure during Cardiopulmonary Bypass on Postoperative Outcomes in Coronary Artery Bypass Graft Surgery

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Abstract

Background: *Maintaining an optimal mean arterial pressure (MAP) is vital for cerebral blood flow and the overall health of patients undergoing coronary artery bypass graft (CABG) surgery. This study investigated the impact of low MAP on cerebral blood flow and in-hospital clinical outcomes in CABG candidates at our hospital.*

Methods: *We compared a convenience sample of 55 patients with a low MAP (55 mmHg) and 54 patients with a high MAP (75 mmHg) who underwent CABG at Tehran Heart Center in 2023. We recorded the patients' demographic and clinical characteristics by measuring cerebral oximeter readings and lactate levels pre- and post-operation. We then compared the baseline and postoperative characteristics between the 2 groups and determined the impact of MAP on their postoperative changes.*

Results: *The groups were statistically similar at baseline, except for a lower pH in the low MAP group (55 mmHg) (P=0.016). The preoperative and postoperative measurements (including lactate levels and cerebral oximeter readings) did not differ between the groups. However, heart rate was significantly higher in the high MAP group (75 mmHg) (P=0.034). The adjusted effect of baseline MAP on selected postoperative characteristics showed that it could significantly but inversely influence heart rate (β coefficient=-5.952; P=0.017) and had a borderline effect on troponin (β coefficient=1.734; P=0.051) and HCO_3 (β coefficient=0.785; P=0.062).*

Conclusion: *MAP did not significantly influence clinical outcomes in our study. Nonetheless, maintaining an optimal MAP is essential for preventing cerebral injury and ensuring adequate cerebral oxygenation in CABG patients.*

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Introduction

Mean arterial pressure (MAP), the average measured pressure in an individual's arteries during a single cardiac cycle, is a critical factor in ensuring adequate blood flow and oxygen supply to vital organs.[1] In the context of coronary artery bypass graft surgery (CABG), maintaining an optimal MAP is crucial since it can significantly influence the outcomes of this procedure.[1] A low MAP may lead to inadequate perfusion to the vital organs, potentially resulting in organ dysfunction. Several studies have indicated that maintaining MAP within a specific range during and after CABG can improve patient outcomes.[2-4] For instance, a MAP range of 60–80 mmHg is often targeted during the procedure to balance the risks of organ hypoperfusion and excessive bleeding. Therefore, careful monitoring and management of MAP are essential in patients undergoing CABG.

Furthermore, a low MAP can significantly impact the outcome of CABG, particularly in the central nervous system.[5] Targeting MAP during cardiopulmonary bypass (CPB) using cerebral autoregulation monitoring may help decrease the incidence of postoperative complications related to cerebral perfusion, ranging from early memory loss to delirium. However, in patients undergoing subacute or elective CABG and/or aortic valve replacement surgeries randomized to either a target MAP of 40–50 mmHg or a target MAP of 70–80 mmHg during CPB, the differences in levels of biomarkers of neurological injury are insignificant.[6]

In recent decades, near-infrared spectroscopy has been used as a noninvasive method for cerebral oximetry to monitor regional cerebral oxygen saturation during surgery and as an indirect indicator of cerebral perfusion.[7, 8] In the context of CABG patients, cerebral oximetry is crucial in monitoring and managing cerebral oxygen supply/demand imbalance, associated with a high risk of cerebrovascular complications.[9] Cerebral oximetry in CABG patients has been linked to various surgical outcomes. Studies have shown that intraoperative management guided by cerebral oximetry is associated with a reduced incidence of postoperative cognitive dysfunction and a significantly shorter length of stay in the ICU.[10] Nevertheless, the use of intraoperative cerebral oximetry has not been found to influence the overall hospital length of stay and the incidence of postoperative delirium.[10] While some studies have demonstrated improved outcomes among cardiac surgical patients with the utilization of cerebral oximetry,[11] others have reported that its use during cardiac surgery has not been associated with improvements in neurocognitive outcomes, morbidity, or mortality.[12] Thus, the evidence supporting interventions for acute desaturation is equivocal, and the relationship between cerebral oxygen saturation and postoperative outcomes is not fully understood.

In the present study, we compared peri- and postoperative outcomes, including cerebral oxygenation parameters, in patients who underwent CABG at our center between those with a low and normal MAP at baseline.

Methods

In this study, we enrolled a convenience sample of 55 patients with a low MAP (55 mmHg) and 54 patients with a high MAP (75 mmHg) who were candidates for elective CABG alone at Tehran Heart Center. The inclusion criteria were an average age of 40-75 years, elective CABG, and consent to participate in the study. The exclusion criteria included a history of previous cardiac surgery, a history of syncope or neurological diseases, and a previous cerebrovascular accident. The study protocol was approved by the internal ethics committee's research board (IR.TUMS.SPH.REC.1402.122), and all the patients provided informed consent. The study population underwent elective on-pump CABG by expert cardiac surgeons at Tehran Heart Center's operating rooms following current protocols. The patients' blood pressure was measured in the operating room before anesthesia induction using a brachial cuff while the patient was in a supine position. MAP was calculated using the following formula:

$$MAP = \frac{\text{Systolic blood pressure} + \left(2 \times \text{diastolic blood pressure}\right)}{3}$$

For cerebral oximetry monitoring, adhesive pads were attached to both sides of the forehead while the patient was in a supine position before surgery. Measurements were then recorded from the monitor. A venous blood sample was obtained at baseline to measure creatinine, urea, electrolytes, blood glucose, lipid profile, blood cell count, troponin, and lactate levels. A similar measurement was performed postoperatively after the patients were transferred to the ICU. Additionally, an arterial blood sample was taken before surgery and repeated postoperatively to measure arterial blood gas characteristics, including pH, PCO_2, PaO_2, and HCO_3. All the patients were monitored during surgery and their ICU stay. Heart rate and blood pressure were recorded at the beginning of the operation before anesthesia induction and at the time of ICU admission. Categorical

data were described as frequencies (percentages) and compared between the 2 study groups using the χ^2 test. Quantitative data were checked for normal distribution using the Kolmogorov-Smirnov test. Normally distributed data were described using mean and standard deviation, while non-normally distributed data were described using medians and interquartile ranges. Variables with normal distribution were compared between the study groups using the Student *t*-test, while non-normally distributed data were compared using analysis of variance. To identify the effect of MAP on postoperative measurements, we employed an adjusted generalized linear regression model, adjusting for baseline variables with a P value <0.100 as confounding factors. All the statistical analyses were performed using SPSS, version 23.0 (IBM, NY, USA), with a P value <0.05 considered statistically significant.

Results

In this study, we compared 55 patients with a low MAP (55 mmHg) and 54 patients with a high MAP (75 mmHg). There were no significant differences in demographic and baseline clinical variables between the groups. However, pH was significantly lower in the low MAP group, although PCO_2 and HCO_3 levels did not differ. The comparison of baseline variables is presented in Table 1.

The operation was successful for all the patients, and no complications or mortality occurred. Regarding peri- and postoperative characteristics, no significant differences existed in ventilation time, ICU stay, laboratory parameters, and blood gas between the groups. Only heart rate was significantly higher in the high MAP group. Additionally, no significant differences were observed in cerebral oximetry, and none of the patients developed hypoxia. The peri- and postoperative measurements are summarized in Table 2.

The adjusted impact of baseline MAP on selected postoperative characteristics revealed that it significantly but inversely influenced heart rate and had a borderline effect on troponin and HCO_3 levels (Table 3).

Table 1. Comparisons of the baseline characteristics between the study groups

Characteristic	MAP=55 (mmHg)	MAP=75 (mmHg)	P*
Age (y)	58.12±11.63	61.64±10.22	0.097
Male sex, n (%)	41 (47.5)	39 (72.2)	0.831
BMI (kg/m^2)	26.32±3.84	25.61±3.44	0.295
Diabetes mellitus (n)	36 (65.5)	36 (66.7)	0.894
Hypertension n (%)	23 (41.8)	27 (50.0)	0.391
Systolic blood pressure (mmHg)	115.70±15.07	116.15±17.78	0.908
Diastolic blood pressure (mmHg)	69.36±11.85	67.73±13.22	0.506
EF (%)	49.42±5.11	48.81±6.04	0.539
Na (mg/dL)	135.82±7.84	136.47±6.32	0.637
K (mg/dL)	4.05±0.43	4.06±0.68	0.886
Blood glucose (mg/dL)	125.46±35.09	126.31±38.03	0.867
Lactate (mg/dL)	7.86±2.52	7.67±2.79	0.713
Triglyceride (mg/dL)	127.95±52.80	150.75±72.97	0.064
Cholesterol (mg/dL)	149.75±71.44	159.08±39.45	0.231
BUN (mg/dL)	22.99±9.85	23.88±11.40	0.661
Creatinine (mg/dL)	1.06±0.31	1.01±0.26	0.351
Hemoglobin (g/dL)	13.46±1.60	13.57±1.67	0.725
Hematocrit (%)	38.72±6.05	37.63±7.06	0.396
Troponin (ng/L)	5.09±7.80	3.28±2.80	0.112
pH	7.39±0.07	7.42±0.05	0.016
$PaCO_2$ (mmHg)	38.09±7.94	35.04±9.45	0.071
PaO_2 (mmHg)	263.14±98.75	312.40±91.16	0.008
HCO_3 (mg/dL)	24.84±2.70	24.30±2.99	0.317
Heart rate (bpm)	66.54±13.98	67.82±13.34	0.638
Cerebral oximetry 1 %	62.43±7.30	62.72±9.04	0.819
Cerebral oximetry 2 %	63.41±6.53	63.64±9.33	0.938

*P<0.005 was statistically significant.

BMI, Body mass index; EF, Ejection fraction; Na, Sodium; K, Potassium; BUN, Blood urea nitrogen; pH, Power of hydrogen; $PaCO_2$, Partial pressure of carbon dioxide; PaO_2, Partial pressure of oxygen; HCO_3, Bicarbonate

Table 2. Comparisons of the pre and postoperative characteristics between the study groups

Characteristics	MAP=55 (mmHg)	MAP=75 (mmHg)	P*
Systolic blood pressure (mmHg)	113.62±11.27	114.1±9.94	0.809
Diastolic blood pressure (mmHg)	63.73±10.08	66.0±9.87	0.234
Ventilation time (h)	7.20±2.63	7.02±3.04	0.7
Discharge time from ICU (h)	32.61±10.04	32.57±12.52	0.95
Na (mg/dL)	133.19±17.14	136.52±6.58	0.185
K (mg/dL)	4.40±0.73	4.53±0.67	0.347
Blood glucose (mg/dL)	150.13±29.72	156.49±43.68	0.383
Lactate (mg/dL)	14.29±5.13	14.24±4.22	0.957
Triglyceride (mg/dL)	131.42±47.39	140.31±56.570	0.373
Cholesterol (mg/dL)	147.08±37.36	156.48±35.14	0.182
BUN (mg/dL)	23.38±8.47	23.26±7.86	0.938
Creatinine (mg/dL)	1.25±1.14	0.98±0.19	0.096
Hemoglobin (g/dL)	10.66±1.50	10.65±1.68	0.979
Hematocrit (%)	28.36±4.83	29.53±4.68	0.18
Troponin (ng/L)	8.11±5.15	6.63±3.96	0.096
pH	7.22±0.95	7.35±0.07	0.331
$PaCO_2$ (mmHg)	36.00±7.88	34.36±6.84	0.252
PaO_2 (mmHg)	194.43±88.87	216.55±96.19	0.215
HCO_3 (mg/dL)	22.47±2.43	21.72±1.93	0.074
Heart rate (bpm)	81.13±13.20	86.46±12.40	0.034
Cerebral oximetry 1 (%)	63.52±9.56	61.91±8.58	0.337
Cerebral oximetry 2 (%)	64.91±7.80	62.69±9.22	0.163

*P<0.005 was statistically significant.
BMI, Body mass index; EF, Ejection fraction; Na, Sodium; K, Potassium; BUN, Blood urea nitrogen; pH, Power of hydrogen; $PaCO_2$, Partial pressure of carbon dioxide; PaO_2, Partial pressure of oxygen; HCO_3, Bicarbonate

Table 3. Adjusted impact of baseline mean arterial pressure on the selected postoperative characteristics

Characteristic*	β	95% CI	P**
Heart rate	-5.952	-10.849 - 1.054	0.017
Creatinine	0.218	-0.095 - 0.532	0.172
Troponin	1.734	-0.008 - 3.476	0.051
HCO_3	0.785	-0.041 - 1.610	0.062

*The effect was adjusted for age and baseline pH.
**P<0.05 was statistically significant.
HCO_3, Bicarbonate

Discussion

In this study, we observed that a low MAP (55 mmHg) in CABG patients did not significantly impact in-hospital outcomes, except for its significant influence on postoperative heart rate and a modest effect on postoperative troponin and HCO_3 levels.

Maintaining an optimal MAP in CABG patients is crucial for ensuring positive clinical outcomes.[5, 13, 14] A low MAP during CPB has been linked to adverse outcomes. A study found that stroke was strongly associated with a sustained MAP<64 mmHg during CPB.[14] For every 10 minutes of MAP between 55 and 64 mmHg, the adjusted odds ratio for stroke was 1.13, and for every 10 minutes of MAP less than 55 mmHg, the adjusted odds ratio for stroke was 1.16. A randomized controlled trial analyzed the effect of target blood pressure management during CPB on blood lactate levels following cardiac surgery.[13] The study revealed that maintaining a moderately higher MAP during CPB led to reduced blood lactate levels at the end of surgery, decreased epinephrine administration, and shortened extubation time and length of ICU stay. These findings highlight the importance of maintaining optimal MAP during CABG to prevent low MAP-related complications and improve patient outcomes. Nonetheless, the specific MAP threshold considered low may vary depending on individual patient characteristics and the specific context of the surgery.[14, 15]

In CABG patients, MAP and cerebral oximetry, often measured as regional cerebral oxygen saturation, are

interconnected and can significantly impact patient outcomes.[10] Cerebral oximetry provides a noninvasive estimate of cerebral oxygenation and perfusion based on near-infrared spectroscopy, reflecting the balance between cerebral oxygen supply and demand.[16] Impaired cerebral autoregulation has been observed in patients undergoing CPB, but its risks and associations with outcomes are not well understood.[16] A study revealed that targeting MAP during CPB using cerebral oximetry monitoring might help reduce the incidence of post-cardiac surgery delirium.[5] Delirium occurs in up to 52% of patients following cardiac surgery and may result from changes in cerebral perfusion.

Hyperlactatemia commonly occurs after cardiac surgery, affecting 10%–30% of patients during or shortly after CPB.[17] Increased lactate levels often result from anaerobic glycolysis due to tissue hypoperfusion, hypoxia, or both.[18] In patients undergoing CABG, MAP and lactate levels are interconnected and can significantly impact patient outcomes.[13, 18, 19] A study found that target blood pressure management during CPB can improve lactate levels after cardiac surgery.[13] The study divided patients into low MAP (50–60 mmHg) and high MAP (70–80 mmHg) groups. At the end of the surgery, the high MAP group had a significantly higher lactate level than the low MAP group. This finding suggests that maintaining a relatively higher MAP during CPB can decrease the blood lactate level at the end of surgery. Another investigation concluded that an elevated mean central venous pressure during the first 24 hours was correlated with worse outcomes in CABG patients.[19] The impact on the afterload of tissue perfusion of the potential mechanisms may alter the lactate levels and lactate clearance. These findings and our results underscore the significance of careful blood pressure management during CABG to optimize lactate levels and potentially improve patient outcomes. Still, individual patient characteristics, the specific context of the surgery, and the timing of the lactate elevation should always be considered when interpreting these associations.[18, 20, 21] While cerebral oximetry offers valuable information on the cerebral oxygenation status of CABG patients, its impact on surgical outcomes remains a subject of ongoing research. Further large-scale, ideally multicenter, randomized controlled trials are necessary to determine whether cerebral oximetry-guided management can positively influence postoperative outcomes.

This study has several limitations worth noting. Firstly, it was conducted in a tertiary heart center with a limited sample size, so the generalizability of the results should be approached with caution. Secondly, patient follow-up was limited to the duration of hospital stay, and we did not examine the potential long-term effects of baseline low MAP on patient outcomes.

Conclusion

Maintaining an optimal MAP is essential for minimizing post-CABG complications related to cerebral blood flow. However, the optimal MAP may vary based on individual patient characteristics and specific surgical contexts. Future research should focus on determining the optimal MAP range for each patient, considering factors such as age and comorbidities. Adopting this personalized approach could lead to further improvements in CABG outcomes.

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