

Changes in Peripheral Arterial Blood Pressure After Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) in Non-traumatic Cardiac Arrest Patients

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Abstract

Background

Resuscitative endovascular balloon occlusion of the aorta (REBOA) may be an adjunct treatment to cardiopulmonary resuscitation (CPR). Aortic occlusion may increase aortic pressure and increase the coronary perfusion pressure and the cerebral blood flow. Peripheral arterial blood pressure is often measured during or after CPR, however, changes in peripheral blood pressure after aortic occlusion is insufficiently described. This study aimed to assess changes in peripheral arterial blood pressure after REBOA in patients with out of hospital cardiac arrest.

Methods

An observational study performed at the helicopter emergency medical service in Trondheim (Norway). Eligible patients received REBOA as adjunct treatment to advanced cardiac life support. Peripheral invasive arterial blood pressure and end-tidal CO₂ (EtCO₂) was measured before and after aortic occlusion. Differences in arterial blood pressures and EtCO₂ before and after occlusion was analysed with Wilcoxon Signed Rank test.

Results

Five patients were included to the study. The median REBOA procedural time was 11 minutes and median time from dispatch to aortic occlusion was 50 minutes. Two patients achieved return of spontaneous circulation. EtCO₂ increased significantly 60 seconds after occlusion, by a mean of 1.16 kPa ($p = 0.043$). Before occlusion the arterial pressure in compression phase were 43.2 (12 – 112) mmHg, the mean pressure 18.6 (4 – 27) mmHg and pressure in the relaxation phase 7.8 (-7 – 22) mmHg. Two minutes after aortic occlusion the corresponding pressures were 114.8 (23 – 241) mmHg, 44.6 (15 – 87) mmHg and 14.8 (0 – 29) mmHg. The arterial pressures were significant different in the compression phase and as mean pressure ($p = 0.043$ and $p = 0.043$, respectively) and not significant in the relaxation phase ($p = 0.223$).

Conclusion

This study is, to our knowledge, the first to assess the peripheral invasive arterial blood pressure response to aortic occlusion during CPR in the pre-hospital setting. REBOA application during CPR significantly increase the peripheral artery pressures. This likely indicates improved central aortic blood pressure and warrants studies with simultaneous peripheral and central blood pressure measurement during aortic occlusion.

Trial registration

The study is registered in ClinicalTrials.gov (NCT03534011).

Background

Resuscitative endovascular balloon occlusion of the aorta (REBOA) may increase the aortic pressure during cardiopulmonary resuscitation (CPR) and can possibly be an adjunct treatment in non-traumatic cardiac arrest patients. The use in preclinical and human studies has recently been reviewed (1, 2) and the procedure is shown feasible during pre-hospital CPR (3). However, the effects of REBOA on central aortic or peripheral blood pressure during CPR is scarcely described (4–8). Case reports and preclinical studies indicate that REBOA may increase aortic pressure and subsequently the coronary perfusion pressure (CPP). Studies on CPR without REBOA suggest that changes in radial arterial pressure may indicate changes in central pressure (9, 10).

The central aortic blood pressure is the main determinant for the CPP during resuscitation. Increase in CPP is associated with return of spontaneous circulation (ROSC) in humans (11). The REBOA catheter currently in use in Norway is not approved for aortic blood pressure measurements, which prevents the direct measure of central aortic pressure. The CPP is the difference between the relaxation phase pressure in the aorta and the right atrial pressure. Hence, to increase the relaxation phase aortic pressure by applying REBOA will likely increase the CPP. We hypothesise that REBOA during CPR may enhance this increase in CPP and may increase the cerebral blood flow during CPR.

The newly commenced REBOARREST trial is a randomised controlled trial (RCT) that aims to investigate the efficacy of REBOA as an adjunct treatment to advanced cardiopulmonary resuscitation (ACLS) (12). The current study was initiated to substantiate the physiological rationale for REBOA in non-traumatic cardiac arrest patients. The aim of this study was to investigate the peripheral arterial pressure response to aortic occlusion during CPR in patients suffering from non-traumatic out of hospital cardiac arrest (OHCA).

Methods

This was an observational study, performed by the physician-manned helicopter emergency medical service (HEMS) in Trondheim, Norway, with a catchment population of approximately 700 000. The personnel at the HEMS base has previously been educated in the use of the REBOA technique during ACLS (13) and a pilot feasibility study was performed at the base (3). Patients were included between December 2019 and April 2021. Due to the Covid-19 pandemic, inclusion of patients was halted in most of 2020 until primo 2021, and the study was prematurely stopped before the intended 10 patients had been included.

All patients that met inclusion/exclusion criteria were resuscitated on scene according to the current ACLS guideline published by the Norwegian Resuscitation Council (14). Patients were endotracheally intubated and received mechanical chest compressions (LUCAS CPR, Physio Control-Inc, Lund, Sweden). Invasive arterial blood pressures were measured via the left radial or brachial artery and registered at one-minute intervals. If the physician failed to achieve peripheral arterial access, the patient was excluded and

did not receive REBOA as an adjunct treatment, and standard ACLS was provided as per routine. The REBOA procedure was performed in sterile conditions under ultrasound guidance (iViz, FUJIFILM SonoSite, WA, USA) via the femoral artery. A catheter (7 Fr, 20 mm, Reboa Balloon Kit, Reboa Medical AS, Norway) was inserted to a length of 50 cm for an aortic zone 1 occlusion. The detailed procedure has previously been reported (3) and is available at www.reboarrest.com.

Patients between 18 and 75 years of age, with non-traumatic cardiac arrest and CPR commenced within 10 minutes were included. Exclusion criteria were suspected or known pregnancy, known terminal illness, accidental hypothermia, drowning, strangulation and suspected intracerebral haemorrhage.

Data collection

Demographic and cardiac arrest variables and characteristics were obtained through a semi-structured interview with the performing physician and by the Utstein template for CPR related variables (15). Time of HEMS dispatch, arrival, procedure duration and ROSC were obtained from the emergency medical communications central database and a specifically designed checklist chart (Supplemental material 1).

End-tidal CO₂ (EtCO₂) values were obtained (Corpuls3, GS, Germany) and measured before balloon inflation, directly after inflation, 30, 60 and 90 seconds after inflation, and after ROSC of any duration. Invasive arterial blood pressure measurements were obtained and registered every minute.

Statistical analysis

Data was analysed with IBM SPSS Statistics 27 and R version 3.6.0. The R package 'ggplot2' was applied for visualization. Continuous variables are reported as mean or median with range, as appropriate. Categorical variables are described as count and/or proportion. Differences in arterial blood pressures and EtCO₂ before and after occlusion was analysed with Wilcoxon Signed Rank test. A *p* value of < 0.05 was regarded as statistically significant.

Results

During the study period, HEMS was dispatched 157 times due to OHCA, of which 69 were during a pause in study due to the Covid-19 pandemic. Of the remaining 88 patients, 22 were older than 75 years and 14 had obtained ROSC before the arrival of the HEMS crew. Thirty-five patients were not eligible for REBOA due to other exclusion criteria. In four patients the physician was unable to achieve peripheral arterial access (Fig. 1). In total, seven patients received REBOA as an adjunct treatment to ACLS. All procedures were performed indoors. Two of these were subsequent excluded from the study due to extra-arterial placement of the peripheral arterial line. The arterial pressure was measured in the left radial artery in four patients and in the left brachial artery in one patient. The baseline details for these five patients are described in Table 1.

Table 1
 Baseline characteristics of patients. OHCA indicates out of hospital cardiac arrest; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia.

Baseline Characteristics	
Male, n (%)	5 (100)
Age, median (range)	63 (45–71)
Time of OHCA	
Daytime (08–23), n (%)	3 (60)
First monitored rhythm	
Asystole, n (%)	2 (40)
PEA, n (%)	2 (40)
VF/VT, n (%)	1 (20)

The REBOA procedure was successful in all five patients at first cannulation attempt. In all patients the cannulation was performed during a 10–20 second pause in chest compressions. No procedural complications such as excess bleeding at puncture site, equipment malfunction or resistance to introduction of the equipment occurred. Relevant procedural data are presented in Table 2.

Table 2
 Procedural times for resuscitative endovascular balloon occlusion of the aorta. Occlusion time and time from dispatch to ROSC are only indicated for the two patients with ROSC. ROSC indicates return of spontaneous circulation; REBOA, resuscitative endovascular balloon occlusion of the aorta.

Procedural data	Median	Range	No.
Dispatch to arrival on scene, min	29	10–38	5
Dispatch to occlusion, min	50	39–72	5
Dispatch to ROSC, min	53.5	50–57	2
Procedure time REBOA, min	11	8–22	5
Occlusion time, min	8.0	5–11	2

The median time from dispatch to aortic occlusion was 50 minutes (range 39–72). Two of the five patients achieved ROSC and one patient was admitted to hospital. No patients survived to day 30.

The mean EtCO₂ value before start of aortic occlusion was 3.00 kPa and increased by a mean of 1.08 kPa ($p = 0.104$) 30 seconds after occlusion. From baseline to 60 seconds after occlusion, the EtCO₂ increased by a mean of 1.16 kPa ($p = 0.043$).

The peripheral artery pressures changes after aortic occlusion are demonstrated in Fig. 2. Two minutes before occlusion the arterial pressure in compression phase (“systolic”) were 43.2 (12–112) mmHg, the mean pressure 18.6 (4–27) mmHg and pressure in the relaxation phase (“diastolic”) 7.8 (-7–22) mmHg. The corresponding pressures two minutes after occlusion were 114.8 (23–241) mmHg, 44.6 (15–87) mmHg and 14.8 (0–29) mmHg. Arterial pressure two minutes before occlusion and two minutes after occlusion were significant different in the compression phase (“systolic”) and as mean pressure ($p = 0.043$ and $p = 0.043$, respectively) and not significant in the relaxation phase (“diastolic”) ($p = 0.223$).

One patient showed signs of CPR-induced consciousness (16) after aortic occlusion, to the extent that sedation was needed. The tertiary hospital was consulted for possible extracorporeal membrane oxygenation treatment but was declined due to long duration of cardiac arrest. The resuscitation efforts were then abandoned.

Discussion

To our knowledge, this is the first study to report changes in peripheral arterial pressure due to aortic occlusion in humans suffering from OHCA. Our findings demonstrate that aortic occlusion during ACLS increases peripheral arterial pressure. The REBOA catheter in use is not approved to perform aortic pressure recordings, hence we were not able to measure the central aortic blood pressure. However, we find it likely that an increase in the radial or brachial arterial pressures during CPR also indicate an increase in central aortic blood pressure.

During spontaneous circulation, the blood pressure is amplified and augmented from the aorta to the peripheral arteries, where the systolic pressures increase and the diastolic pressure and mean pressures may decline (17–19). The ventricular systole creates a forward pressure wave that is transmitted along the elastic aorta. The wave hits zones of impedance mismatch, e.g. arterial bifurcations, arterial tapering, changes in arterial stiffness and impedance (17, 20) and is reflected retrograde towards the heart. It then meets the forward pressure wave, and creates an augmented and amplified pressure wave (17).

During cardiac arrest, the scenario is different. Chest compressions pump the blood through the aorta, mimicking the ventricular systole. It was initially proposed as a result of the direct compression of the heart between the sternum and the vertebrae, “the cardiac pump” theory (21). A later explanation is that an increase in intra-thoracic pressure beyond the extra-thoracic pressure is the cause of blood flow during CPR (9, 22), “the thoracic pump” theory. Another possible mechanism is the “respiratory pump” theory (23) where the negative intrathoracic pressure during the relaxation phase (the “diastole”) cause improved return of blood to the heart (e.g. impedance threshold device). It is however possible that it is the sum of different simultaneous mechanisms that produces the forward blood flow (24, 25).

Few studies describe simultaneous radial arterial and central aortic pressures during CPR. One human study (10) demonstrated that the radial arterial pressure correlated with the aortic pressure during CPR. During the compression phase the radial arterial pressure was significantly lower than the aortic pressure, both at baseline and after epinephrine was administered. During the relaxation phase the radial arterial pressure was significantly higher than aortic pressure and the femoral arterial pressure was comparable to the aortic pressure. Another study (9) found that the peak compression phase right atrial pressure (RAP) was slightly higher than the radial arterial pressure, and the relaxation phase gradient between the radial artery and right atria was found to be approximately 11 mmHg. In two case reports the radial arterial compression phase, relaxation phase and mean pressures increased after aortic occlusion (4). In these, RAP was measured simultaneously, and the coronary perfusion pressure increased from - 2 to 8 mmHg and 15 to 18 mm Hg, respectively. These studies indicate first that changes in radial arterial pressures may indicate changes in central aortic pressures, and second that aortic occlusion may increase aortic pressure and subsequently the CPP.

Systolic blood pressure can differ significantly between the central and peripheral arteries (17). The radial or brachial arterial pressure is commonly measured in patients and the mean arterial pressure is used as a substitute to tissue perfusion (26). This may create erroneous assumption of the patient's central aortic blood pressure. Few studies, and with small sample sizes, report intra-arterial systolic blood pressure differences between the brachial and radial artery (27). One study demonstrated that on average, radial arterial systolic pressure was 5.5 mmHg higher than brachial arterial systolic pressure. Most patients had systolic radial arterial BP > 5 mmHg higher than brachial and as much as 14% of the patients had radial arterial systolic BP > 15 mmHg higher than brachial. This so-called "Popeye phenomenon" (27) clearly demonstrates that there is not equivalence between brachial and radial arterial pressure. Further, it is shown that brachial cuff BP measurements systematically underestimate the true intra-arterial brachial pressure by 5.7 mmHg (28). Combined with the difference of > 15 mmHg from brachial to radial arterial pressure as shown (27), the difference from brachial cuff-measured systolic pressure and invasive radial pressure may be above 20 mmHg. How, and where, arterial pressure is measured are therefore important to consider in clinical practice.

If spontaneous circulation is achieved, it is not known how the lumen-occluding balloon will affect the forward wave or the subsequent reflection wave. One may speculate that a stunned heart would deteriorate under the afterload created by the inflated balloon. However, it may also be that a potential increase in aortic pressure will contribute to improved myocardial perfusion and subsequent improvement of contractility (29). This may possibly be mediated through the Greggs phenomena (30), where improved coronary perfusion results in increased oxygen uptake followed by increased cardiac strength. The cause of this is not clear, but may involve restoration of adequate subendocardial oxygen supply (ischemia effect) (31) or "the garden-hose effect", where the increased blood flow through coronary vasculature stretches the surrounding myocardium (sarcomere stretch), with an increased left ventricular contractility due to the Frank-Starling mechanism (32, 33). It is also not known if, and by how much, the Anrep effect (34) contribute to the haemodynamic situation after ROSC. The Anrep effect is a positive inotropic effect after a sudden increase in systolic pressure. This enhancement in left ventricular

function may contribute to improved post ROSC circulation, even after the deflation of the REBOA balloon. If this effect, or Gregg's phenomena, is at all valid during CPR, is unknown.

Limitations

First, the small number of patients cause the data to be regarded as preliminary findings. Second, it was a single-centre study, with few physicians and paramedics involved. Third, all the physicians were board-certified anaesthesiologist with considerable experience with the use of ultrasound and Seldinger technique, and the results may not be relevant to other settings where cardiac arrest patients are treated. Fourth, the arterial pressures are measured with one-minute sampling rate. The blood pressure may vary during this time interval, hence further studies may benefit from the use of higher sampling rate, or continuous sampling. Finally, this study primarily increase knowledge on the hemodynamic changes caused by REBOA during CPR and cannot conclude about the potential clinical benefit from a REBOA intervention.

Conclusion

To our knowledge, this is the first study to investigate the peripheral invasive arterial blood pressure response to aortic occlusion during non-traumatic OHCA patients. REBOA as an adjunct treatment during resuscitation significantly increase the peripheral artery pressures and it is likely that this indicates improved central aortic blood pressure. Our findings warrant studies with simultaneous peripheral and central blood pressure measurement during aortic occlusion.

List Of Abbreviations

ACLS Advanced cardiovascular life support

BP Blood pressure

CPP Coronary perfusion pressure

CPR Cardiopulmonary resuscitation

HEMS Helicopter emergency medical service

OHCA Out of hospital cardiac arrest

RAP Right atrial pressure

REBOA Resuscitative endovascular balloon occlusion of the aorta

ROSC Return of spontaneous circulation

Declarations

Ethics approval and consent to participate

The study was approved by the Regional Committees for Medical and Health Research Ethics (reference 2018/51/REK Midt) and is registered in ClinicalTrials.gov (NCT03534011). The patient's next-of-kin were given study information and provided an oral and written consent.

Consent for publication

All patients' next of kin provided oral and written consent for publication.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

Dr Brede and Dr Jørstad Krüger are partly funded by the Norwegian Air Ambulance Foundation for research purposes. Dr Nordseth has received research funding from the same organization. The other authors have no disclosures.

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Authors' contribution

JRB and AJK designed the study. JRB drafted the manuscript. PK and ES aided in the design of the study and revised the manuscript. TN prepared the figures and aided in revision of the manuscript. All authors have read and approved the final manuscript.

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Supplemental Material

Supplemental Material 1 is not available with this version.

Figures

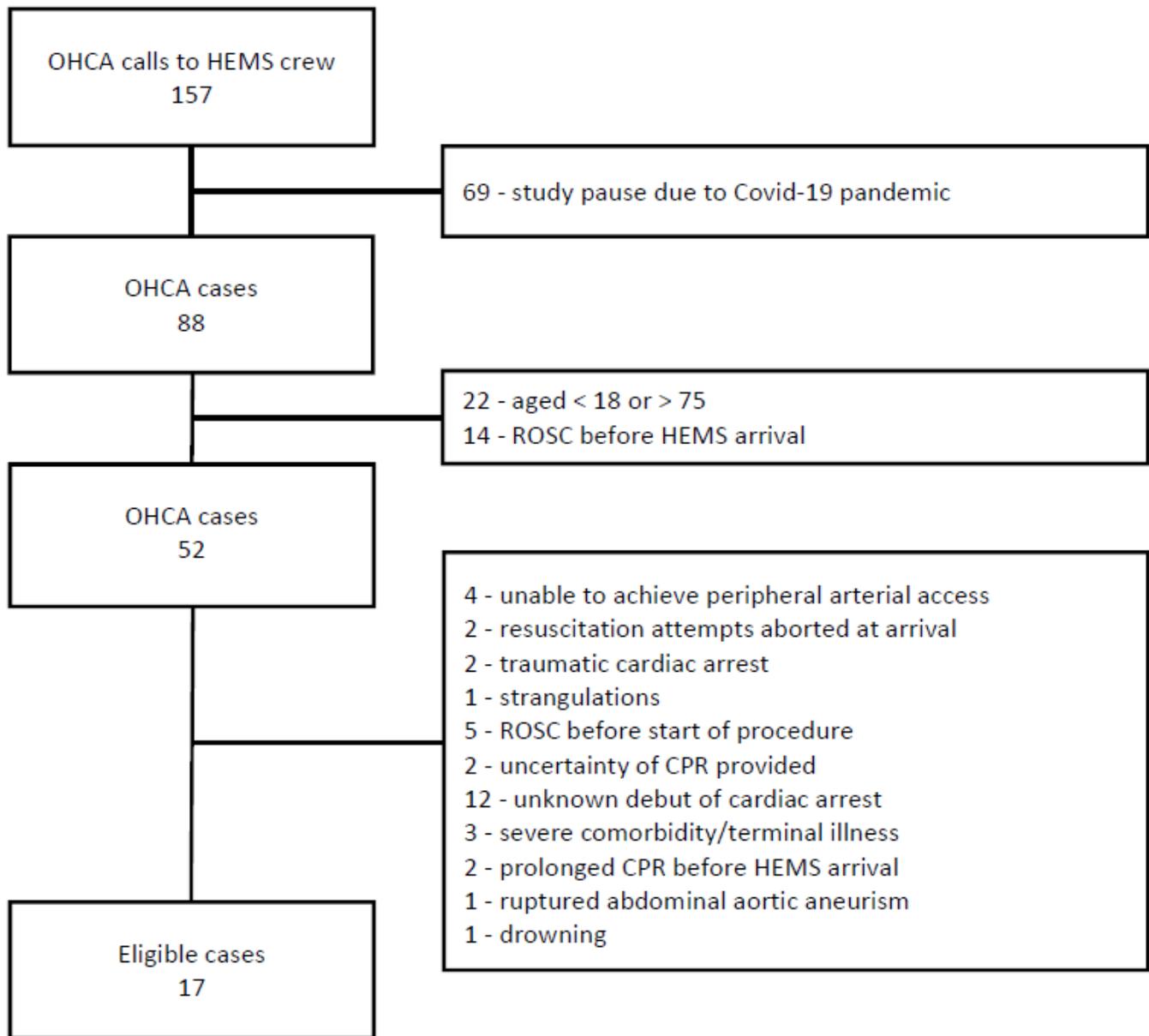


Figure 1

Flowchart of patient inclusion. Flowchart of patients eligible for pre-hospital REBOA with simultaneous peripheral invasive arterial blood pressure measurements during cardiac arrest. OHCA indicates out of hospital cardiac arrest; HEMS, helicopter emergency medical service; ROSC, return of spontaneous circulation; CPR, cardiopulmonary resuscitation; REBOA, resuscitative endovascular balloon occlusion of the aorta.

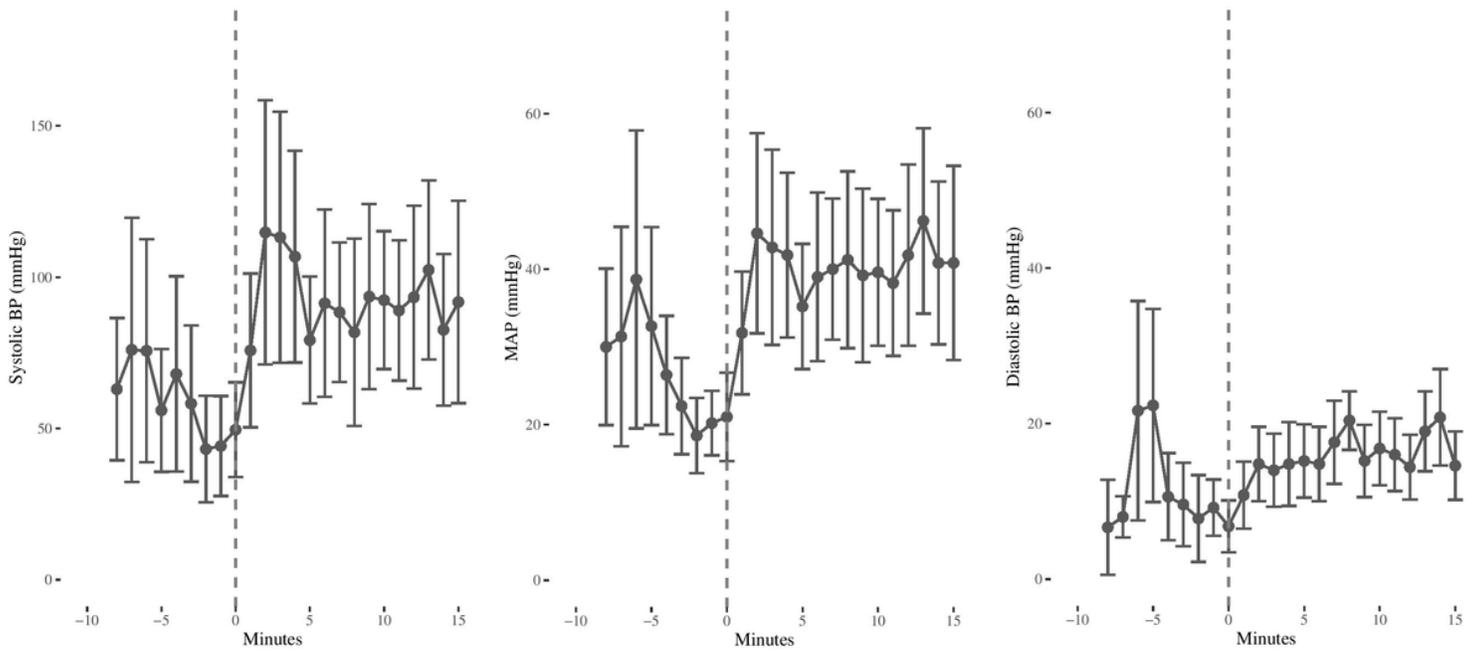


Figure 2

Peripheral blood pressure changes after aortic occlusion. Peripheral artery pressure changes after aortic occlusion, mean values +/- standard error. Occlusion is at 0 minutes. BP indicates blood pressure; MAP, mean arterial pressure.