Background: Abdominal complications being rare but results in high mortality, commonly due to splanchnic organ hypoperfusion during the perioperative period of cardiac surgery. There are no feasible methods to monitor intraoperative superior mesenteric artery blood flow (SMABF). Hence, the aim of this study was to evaluate the feasibility and to measure SMABF using transesophageal echocardiography (TEE) during cardiac surgery under hypothermic cardiopulmonary bypass (CPB).

Methodology: Thirty-five patients undergoing elective cardiac surgery under CPB were enrolled. Heart rate, mean arterial pressure (MAP), cardiac output (CO), SMABF, superior mesenteric artery (SMA) diameter, superior mesentric artery blood flow over cardiac output (SMA/CO) ratio and arterial blood lactates were recorded at three time intervals. T0: before sternotomy, T1: 30 min after initiation of CPB and T2: after sternal closure.

Results: SMA was demonstrated in 32 patients. SMABF, SMA diameter, SMA/CO ratio and arterial blood lactates were recorded at three time intervals. T0: before sternotomy, T1: 30 min after initiation of CPB and T2: after sternal closure. Results: SMA was demonstrated in 32 patients. SMABF, SMA diameter, SMA/CO ratio and arterial blood lactates were recorded at three time intervals. T0: before sternotomy, T1: 30 min after initiation of CPB and T2: after sternal closure. Results: SMA was demonstrated in 32 patients. SMABF, SMA diameter, SMA/CO ratio and arterial blood lactates were recorded at three time intervals. T0: before sternotomy, T1: 30 min after initiation of CPB and T2: after sternal closure. Results: SMA was demonstrated in 32 patients. SMABF, SMA diameter, SMA/CO ratio and arterial blood lactates were recorded at three time intervals. T0: before sternotomy, T1: 30 min after initiation of CPB and T2: after sternal closure.

Conclusion: Study shows that there is decrease in SMABF during CPB and returns to baseline after CPB. Hence, it is feasible to measure SMABF using TEE in patients undergoing cardiac surgery under hypothermic CPB. TEE can be a promising tool in detecting and preventing splanchnic hypoperfusion during perioperative period.

Key words: Cardiopulmonary bypass; Superior mesenteric artery; Transesophageal echocardiography

INTRODUCTION

Gastrointestinal (GI) complications are reported to be <1% in cardiac surgical patients, but these carry a very high mortality of 14%. Acute mesenteric ischemia (AMI) accounts to majority of these complications around 10–67% with a case fatality rate of 70–100%. Blood supply to GI tract is by splanchnic circulation which includes celiac trunk, superior mesenteric artery (SMA) and inferior mesentric artery (IMA). Cardiopulmonary bypass (CPB) induces regional hypoperfusion commonly involving splanchnic circulation leading to mesenteric ischemia. Transesophageal echocardiography (TEE) is a useful tool in monitoring splanchnic mesenteric blood flow.
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to GI complications. SMA supplies majority of small and large intestine and monitoring the SMA blood flow during cardiac surgery would help in early diagnosis of mesenteric ischemia. There are various invasive and noninvasive modalities to image SMA, gold standard being angiography. Altered liver and renal function with unstable hemodynamics limit the use of angiography in AMI. Other noninvasive methods such as computed tomography and abdominal ultrasound can diagnose SMA ischemia but lacks sensitivity and specificity. However, none of the methods are feasible in operating theater.

Transesophageal echocardiography (TEE) has been routinely used during cardiac surgery for monitoring ventricular and valvular function. Demonstration of branches of abdominal aorta using TEE has been reported. This study was conducted for the feasibility of SMA visualization and quantification of SMA blood flow in on-pump cardiac surgical patients using TEE.

**METHODOLOGY**

After obtaining institutional ethics committee clearance and informed consent from each patient, 35 adult patients scheduled for elective on-pump cardiac surgery were enrolled in the study. Exclusion criteria included acute myocardial infarction (<7 days), preoperative hemodynamic instability, preoperative or intraoperative intraaortic balloon pulsation, Doppler angle >20° and contraindication for insertion of TEE probe.

All patients were premedicated with injection midazolam 0.05 mg/kg intravenously. Radial artery was cannulated under local anesthesia for invasive monitoring. General anesthesia was induced with injection fentanyl 5 mcg/kg, titrated doses of injection propofol and injection vecuronium 0.1 mg/kg to facilitate muscle relaxation. Following endotracheal intubation, patients were mechanically ventilated and maintained with injection fentanyl, midazolam and vecuronium. A 5 MHz Biplane TEE probe (Philips En Visor CHD, Bothell, Washington 98041, USA) was inserted after induction of anesthesia. A triple lumen central venous catheter in the right internal jugular vein and femoral arterial catheter were inserted for invasive monitoring.

Heparin sulfate 4 mg/kg was administered before CPB to maintain an activated clotting time of at least 480 s. CPB was conducted with roller pump using a membrane oxygenator and nonpulsatile perfusion (at a flow rate of 2–2.4 L/min/m²). Antegrade intermittent cold blood cardioplegia was used for myocardial protection. Systemic temperature was maintained between 28°C and 32°C, which was monitored using nasopharyngeal temperature probe. Mean arterial pressure (MAP) was kept at 50–80 mmHg. Anesthesia during cardiopulmonary bypass was maintained using injection midazolam, fentanyl and vecuronium. Heparin sulfate was reversed with a corresponding dose of protamine sulfate at the end of surgery.

SMA was visualized by TEE starting from mid esophageal aortic short axis view and by advancing the probe into the stomach, with appropriate rotation and anteflexion to keep the image of the aorta at the center of the screen. The 1st branched vessel was visualized at 1 o’clock position-ceeliac trunk/artery [Figure 1a and Video 1]. The color Doppler signal detection scale was lowered to get a clear vision of the vessel and the blood flow signal. The distinguishing feature of the celiac trunk was its distal bifurcation. The probe was further advanced until the 2nd branched vessel at 3 o’clock position appeared on the screen, i.e. SMA [Figure 1b and Video 2].

SMA velocities, i.e., peak systolic velocity (Vs), end diastolic velocity (Vd) and velocity time integral
RESULTS

Thirty-five patients were enrolled in the study protocol. SMA was successfully demonstrated in 32 patients with a success rate of 91.4%. Three patients were excluded as they could not complete the study. Two patients had a Doppler angle correction >20°, hence were excluded. Statistical analysis was done for thirty patients only. There were 13 female patients and 17 male patients with age ranging from 20 years to 54 years. Average CPB time was 66 ± 18 min. There were seven patients who underwent mitral valve replacement (MVR), ten patients aortic valve replacement, two patients atrial septal defect closure, three patients for coronary artery bypass surgery with MVR, one patient for tetralogy of fallot correction and seven patients for mitral valve repair. All patients required inotrope or vasoconstrictor while weaning from CPB.

There was statistically significant decrease \((P < 0.0001)\) in SMABF, SMA diameter, MAP, CO, superior mesentric artery blood flow over cardiac output \((\text{SMA}/\text{CO})\) ratio between T0 (before sternotomy), T1: 30 min after initiation of CPB, T2: after sternal closure.

During CPB, MAP was replaced by perfusion pressure, CO by pump flow rate and SMA blood flow was calculated using Vm and diameter.

The patient was followed up for 48 h postoperatively for any adverse events.

Statistical analysis

Hemodynamic and echocardiographic variables were compared by means of Student’s t-test for paired data on pump and off pump. A two-tailed probability level of <0.05 was considered significant. All results were expressed as mean ± standard deviation. Statistical analysis was performed using MedCalc software version 12.2.1. (Ostend, Belgium).
There was a progressive, significant increase in lactate levels over time ($P \leq 0.0001$) from T0 to T2 [Table 1].

There was no postoperative GI complication or mortality observed in the study patients with maximum postoperative Intensive Care Unit stay being 2 days.

**DISCUSSION**

SMA being demonstrated in 91.4% of the patients, the present study showed it is feasible to visualize and quantify SMABF intraoperatively by transesophageal approach of abdominal arteries using duplex ultrasound. This technique is simple and reproducible by an experienced operator.\[1\] Accurate velocity measurement can be obtained when there is correct estimation of the vessel diameter, proper Doppler interrogation using two-dimensional mode and recommended Doppler angle. In this study, patients with Doppler angle >20° were excluded and the insonation angle was kept same during T0, T1, and T2 VTI measurements.

Abdominal complication following cardiac surgery is rare but results in high mortality which is due to ischemia/reperfusion of splanchnic organs.\[6,9\] Splanchnic ischemia causes damage of the mucosal barrier resulting in gut translocation of endotoxin leading to systemic inflammatory response and later multi-organ failure in cardiac surgical patients.\[8,10\] AMI is due to arterial embolus, arterial thrombosis, splanchnic vasoconstriction, known as nonocclusive mesenteric ischemia (NOMI) and venous thrombosis. NOMI is the most common cause of AMI following CPB.\[11-13\]

Results of present study show significant decrease in SMABF ($170.80 \pm 117.46$ ml/min) after 30 min of initiation of CPB (T1) as compared to baseline ($343.10 \pm 204.50$ ml/min) (T0). There was significant decrease in CO, MAP and diameter ($P < 0.0001$) during CPB, which may have resulted in decrease in SMABF statistically significant increase in SMABF ($P < 0.0001$), SMA diameter ($P < 0.0001$), MAP ($P = 0.001$), CO ($P < 0.0001$), SMA/CO ratio ($P = 0.0002$), between T1 (30 min after initiation of CPB) and T2 (after sternal closure) [Table 1b].

There was no significant change in SMABF ($P = 0.25$), SMA diameter ($P = 0.18$), MAP ($P = 0.21$), CO ($P = 0.19$), SMA/CO ratio ($P = 0.14$) and R.I ($P = 0.58$) between T0 and T2 [Table 1c].

**Table 1a:** Intraoperative hemodynamic parameters and superior mesenteric artery blood flow data - T0 versus T1

| Parameter | T0 (Before sternotomy) | T1 (30 min after initiation of CPB) | $P$ Value |
|-----------|------------------------|-----------------------------------|-----------|
| MAP (mm Hg) | 73.53±12.86 | 58.27±10.59 | <0.0001 |
| CO (l/min) | 5.24±0.66 | 3.95±0.44 | <0.0001 |
| SMABF (ml/min) | 343.10±204.50 | 170.80±117.46 | <0.0001 |
| SMA diameter (cms) | 0.51±0.18 | 0.37±0.15 | <0.0001 |
| SMA/CO (%) | 6.56±3.82 | 4.25±2.71 | <0.0001 |
| Arterial lactates (mmol/l) | 1.13±0.42 | 2.83±0.88 | <0.0001 |

- MAP: Mean arterial pressure, CO: Cardiac output, SMA/CO: Superior mesenteric artery blood flow, CO: Cardiac output ratio, SMA/CO: Superior mesenteric artery blood flow over cardiac output ratio.

**Table 1b:** Intraoperative hemodynamic parameters and superior mesenteric artery blood flow data - T1 versus T2

| Parameter | T1 (30 min after initiation of CPB) | T2 (After sternal closure) | $P$ Value |
|-----------|-----------------------------------|---------------------------|-----------|
| MAP (mm Hg) | 58.27±10.59 | 70.37±11.94 | 0.001 |
| CO (l/min) | 3.95±0.44 | 5.13±0.65 | <0.0001 |
| SMABF (ml/min) | 170.80±117.46 | 325.50±200.18 | <0.0001 |
| SMA diameter (cms) | 0.37±0.15 | 0.49±0.16 | <0.0001 |
| SMA/CO (%) | 4.25±2.71 | 6.22±3.86 | 0.0002 |
| Arterial lactates (mmol/l) | 2.83±0.88 | 3.47±1.24 | 0.0001 |

- MAP: Mean arterial pressure, CO: Cardiac output, SMA: Superior mesenteric artery, SMA/CO: Superior mesenteric artery blood flow, CO: Cardiac output ratio, SMA/CO: Superior mesenteric artery blood flow over cardiac output ratio.

**Table 1c:** Intraoperative hemodynamic parameters and superior mesenteric artery blood flow data - T0 versus T2

| Parameter | T0 (Before sternotomy) | T2 (After sternal closure) | $P$ Value |
|-----------|------------------------|---------------------------|-----------|
| MAP (mm Hg) | 73.53±12.86 | 70.37±11.94 | 0.21 |
| CO (l/min) | 5.24±0.66 | 5.13±0.65 | 0.19 |
| SMABF (ml/min) | 343.10±204.50 | 325.50±200.18 | 0.25 |
| SMA diameter (cm) | 0.51±0.18 | 0.49±0.16 | 0.18 |
| SMA/CO (%) | 6.56±3.82 | 6.22±3.86 | 0.14 |
| R.I | 0.78±0.06 | 0.79±0.07 | 0.58 |
| Arterial lactates (mmol/L) | 1.13±0.42 | 3.47±1.24 | <0.0001 |

- R.I: Resistivity index MAP: Mean arterial pressure, CO: Cardiac output, SMA: Superior mesenteric artery, SMA/CO: Superior mesenteric artery blood flow, CO: Cardiac output ratio, SMA/CO: Superior mesenteric artery blood flow over cardiac output ratio, CPB: Cardiopulmonary bypass.

Splanchnic hypoperfusion involving mesenteric arterial trunk (which has adrenergic innervations)\[14\] is due to regional vasoconstriction caused by catecholamine,
sympathetic and renin-angiotensin stimulation\textsuperscript{[15]} and hypothermia during CPB. This can result in increased resistance,\textsuperscript{[16]} decrease diameter and hence decreased flow in SMA.

SMABF reduced to a greater extent than CO, T0 versus T1 (50.21\% vs. 24.62\%), with a decrease in fractional output flowing through SMA from 6.56 \pm 3.82\% to 4.25 \pm 2.71\%, \(P < 0.0001\) (T0 vs. T1). During stages of shock, there is less amount of CO directed to splanchnic organs, with greater reduction in visceral organ perfusion compared to CO.\textsuperscript{[17]} Blood flow to heart and central nervous system is maintained by decreasing blood flow to visceral organ microcirculation.\textsuperscript{[18]}

There was an increase in SMABF at T2 (325.50 \pm 200.18) as compared to T1 and insignificant change as compared to T0 (343.10 \pm 204.50 ml/min). This could be due to increase in CO, MAP and SMA diameter (\(P > 0.05\)) between T0 and T2 [Table 1c]. Previous studies have shown sudden reactive hyperemia after sustained reduction in mesenteric artery blood flow which is due to stimulation of constrictor fibers or mechanical occlusion.\textsuperscript{[19,20]}

However, in this study, increase in SMABF and diameter may have been because of return of CO, MAP and resistivity index (0.78 \pm 0.06 vs. 0.79 \pm 0.07) close to baseline value (T0). T2 values were obtained on an average after 1–1.5 h weaning from CPB, where reactive hyperemia causing an increase in SMABF is unlikely. All patients were weaned from CPB with injection dopamine or dobutamine or a vasodilator which are known to dilate the mesenteric bed. Only three patients had a vasoconstrictor (injection adrenaline).

Splanchnic circulation which supplies GI tract includes celiac trunk (gastric, hepatic, pancreatic), SMA and IMA. There are many methods to measure splanchnic blood flow like –using Fick’s principle, laser Doppler flowmetry and hepatic venous oxygen saturation. Gastric tonometry, splanchnic lactate extraction and D-dimer can be used to detect splanchnic ischemia. However, among all these methods only few are feasible intraoperatively. Hepatic vein blood flow (HBF) has been demonstrated using TEE during cardiac surgery with CPB. The authors have concluded that there is decrease in HBF during hypothermic CPB and not in normothermic CPB as compared to baseline with an increase in HBF 4 h after termination of CPB.\textsuperscript{[21]} Previously TEE has been used to demonstrate SMABF\textsuperscript{[22,23]} but there is limited literature on the use of TEE to assess SMABF during hypothermic CPB. Doppler recordings from the TEE give us the advantage of obtaining the data that is almost an online picture of changes in SMABF. A study by Fiore \textit{et al.}\textsuperscript{[23]} demonstrated SMA using TEE in off-pump coronary artery bypass grafting patients and concluded that there is decrease in SMABF during decrease in CO, which occurred while revascularization of obtuse marginal and right coronary artery. SMABF increased at the end of surgery after normalization of CO. Another study by Straub \textit{et al.}\textsuperscript{[5]} demonstrated an increase in resistive and pulsatility index after CPB indicating rigidity in mesenteric vascular bed with decrease in perfusion. However, in their study, measurements were done using Doppler sonography 30 min after induction of anesthesia and 60 min after end of the surgery. They concluded that increase in resistivity index was due to systemic inflammatory response due to CPB. However, in this study, SMABF measurement was done using TEE intraoperatively.

Lactates showed a progressive increase from T0 to T1, which could be accounted for a poor splanchnic perfusion with normal physiological CPB pump flows and perfusion pressures. There is decrease in hepatic clearance leading to pyruvate accumulation resulting in increased lactate\textsuperscript{[24]} or increased intestinal production of lactate secondary to transient intestinal ischemia\textsuperscript{[25]} during and after CPB, which could have further led to progressive increase in lactate from T1 to T2.

Limitations of this study were that - SMABF obtained by TEE was not compared with SMA angiography and also mucosal microcirculation was not assessed.

\textbf{CONCLUSION}

This study demonstrated that TEE is feasible to monitor SMABF during hypothermic CPB. SMABF decreased during CPB as compared to baseline value, which returned to near baseline value after CPB. Hypoperfusion although transient might result in subclinical ischemia causing GI complication. Further studies are required to compare SMABF between hypothermic and normothermic CPB and if an intervention could treat NOMI caused due to mesenteric hypoperfusion during CPB.

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\textbf{Conflicts of interest}
There are no conflicts of interest.
REFERENCES

1. Abboud B, Daher R, Boujaoude J. Acute mesenteric ischemia after cardio-pulmonary bypass surgery. World J Gastroenterol 2008;14:5361-70.

2. Lazenby WD, Ko W, Zelano JA, Lebowitz N, Shin YT, Isom OW, et al. Effects of temperature and flow rate on regional blood flow and metabolism during cardiopulmonary bypass. Ann Thorac Surg 1992;53:957-64.

3. Trompeter M, Brazda T, Remy CT, Vestring T, Reimer P. Non-occlusive mesenteric ischemia: Etiology, diagnosis, and interventional therapy. Eur Radiol 2002;12:1179-87.

4. Orihashi K, Matsuura Y, Sueda T, Shikata H, Morita S, Hirai S, et al. Abdominal aorta and visceral arteries visualized with transesophageal echocardiography during operations on the aorta. J Thorac Cardiovasc Surg 1998;115:945-7.

5. Straub U, Winning J, Greilach P, Isringhaus H, Kalweit G, Huwer H. Alterations of mesenteric blood flow after cardiopulmonary bypass: A Doppler sonographic study. J Cardiothorac Vasc Anesth 2004;18:731-3.

6. Yang PL, Wong DT, Dai SB, Song HB, Ye L, Liu J, et al. The feasibility of measuring renal blood flow using transesophageal echocardiography in patients undergoing cardiac surgery. Anesth Analg 2009;109:1418-24.

7. Fujimura J, Camilleri M, Low PA, Novak V, Novak P, Opfer-Gehrking TL. Effect of perturbations and a meal on superior mesenteric artery flow in patients with orthostatic hypotension. J Auton Nerv Syst 1997;67:15-23.

8. Hessel EA 2nd. Abdominal organ injury after cardiac surgery. Semin Cardiothorac Vasc Anesth 2004;8:243-63.

9. Khan JJ, Lambert AM, Habib JJ, Broce M, Emmett MS, Davis EA. Abdominal complications after heart surgery. Ann Thorac Surg 2006;82:1796-801.

10. Warltier D. The systemic inflammatory response to cardiac surgery: Implications for the anesthesiologist. Anesthesiology 2002;97:215-52.

11. Pinson CW, Alberty RE. General surgical complications after cardiopulmonary bypass surgery. Am J Surg 1983;146:133-7.

12. Wilson C, Gupta R, Gilmour DG, Imrie CW. Acute superior mesenteric ischaemia. Br J Surg 1987;74:279-81.

13. Hasan S, Ratnatunga C, Lewis CT, Pillai R. Gut ischaemia following cardiac surgery. Interact Cardiovasc Thorac Surg 2004;3:475-8.

14. Gelman S, Mushlin PS. Catecholamine-induced changes in the splanchnic circulation affecting systemic hemodynamics. Anesthesiology 2004;100:434-9.

15. Jakob SM. Splanchnic blood flow in low-flow states. Anesth Analg 2003;96:1129-38.

16. Texter EC Jr., Merrill S, Schwartz M, Van Derstappen G, Haddy FJ. Relationship of blood flow to pressure in the intestinal vascular bed of the dog. Am J Physiol 1962;202:253-6.

17. Vatner SF. Effects of hemorrhage on regional blood flow distribution in dogs and primates. J Clin Invest 1974;54:225-35.

18. Takala J. Determinants of splanchnic blood flow. Br J Anaesth 1996;77:50-8.

19. Granger DN, Richardson PD, Kvietys PR, Mortillaro NA. Intestinal blood flow. Gastroenterology 1980;78:837-63.

20. Fatehi-Hassanabad Z, Parratt JR, Furman BL. Endotoxin-induced inhibition of mesenteric vasodilator responses to acetylcholine, bradykinin, and post-occlusion hyperemia in anesthetized rats. Shock 1996;6:371-6.

21. Gärdebäck M, Settergren G, Brodin LA. Hepatic blood flow and right ventricular function during cardiac surgery assessed by transesophageal echocardiography. J Cardiothorac Vasc Anesth 1996;10:318-22.

22. Orihashi K, Sueda T, Okada K, Imai K. Perioperative diagnosis of mesenteric ischemia in acute aortic dissection by transesophageal echocardiography. Eur J Cardiothorac Surg 2005;28:871-6.

23. Fiore G, Brienza N, Cicala P, Tunzi P, Marraudino N, Schinosa Lde L, et al. Superior mesenteric artery blood flow modifications during off-pump coronary surgery. Ann Thorac Surg 2006;82:62-7.

24. Manthous CA, Schumacker PT, Pohlman A, Schmidt GA, Hall JB, Samel RW, et al. Absence of supply dependence of oxygen consumption in patients with septic shock. J Crit Care 1993;8:203-11.

25. Landow L. Splanchnic lactate production in cardiac surgery patients. Crit Care Med 1993;21 2 Suppl:S84-91.