Selected abstracts from presentations to the 18th Autumn Meeting of the Association of Cardiothoracic Anaesthetists

16 November 2001, London, UK

Association of Cardiothoracic Anaesthetists

The publication of these abstracts was made possible by an unrestricted educational grant from Bayer plc

Published online: 7 November 2001
Critical Care 2001, **5** (Suppl 4):1-8

1 Serum N-terminal pro brain natriuretic peptide (NTproBNP) in perioperative cardiac surgical patients

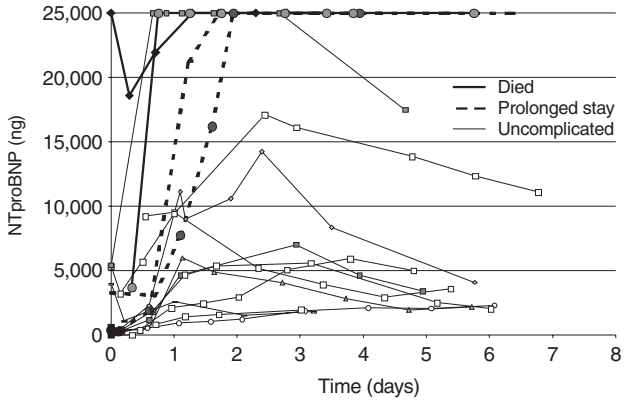
A Crerar-Gilbert, A Dewhurst, SC Barnes, PO Collinson and GR McAnulty
Anaesthetic Department and Department of Clinical Biochemistry, St George's Hospital, London, UK

Introduction: Brain natriuretic peptide (BNP) is released from stretched ventricular wall. BNP particularly the N-terminal portion of proBNP (NTproBNP) is a sensitive marker of congestive heart failure and predictor of outcome [1]. Variations in NTproBNP in cardiac surgical patients are not well described [2]. We investigated changes in NTproBNP in relation to clinical progress in open heart surgery patients.

Methods: We measured serum concentrations of NTproBNP in 15 perioperative cardiac surgical patients in a pilot observational study using an electrochemiluminescent sandwich immunoassay (Elecsys 2010, Roche Diagnostics: interassay c.v. 5.0% at 380 ng/l, 4.4% at 8700 ng/l, 5.0% at 13 000 ng/l, detection limit 20 ng/l, upper measuring limit 25,000 ng/l). We collected samples on induction of anaesthesia, at the end of the surgery, 12 hourly for 3 days, then daily for 3 days.

Results: Two patients of 15 died. One had preoperative right ventricular failure and a baseline NTproBNP of >25,000 ng/l falling to 18 613 ng/l postoperatively but rising to >25,000 ng/l until death. The other with left ventricular failure and NTproBNP of 3720 ng/l had sustained postoperative levels of >25,000 ng/l until death. Two patients were hemofiltered and NTproBNP rose to >25,000 ng/l postoperatively. Eight patients with a baseline NTproBNP <700 ng/l had uncomplicated recoveries. Of five patients with a baseline NTproBNP >1000 ng/l two died and two had prolonged ICU stays. Baseline NTproBNP was missing for two patients, one of whom died (Fig. 1).

Figure 1



NTproBNP concentrations in 15 heart surgery patients.

Conclusions: Changes in serum NTproBNP in cardiac surgical patients may predict clinical course.

References

1. Bettencourt P, Ferreira A, Dias P, Castro A, Martins L, Cerqueira-Gomes M: **Evaluation of brain natriuretic peptide in the diagnosis of heart failure**. *Cardiology* 2000, **93**:19-25.
2. Morimoto K, Mori T, Ishiguro S, Matsuda N, Hara Y, Kuroda H: **Perioperative changes in plasma brain natriuretic peptide concentrations in patients undergoing cardiac surgery.** *Surgery Today* 1998, **28**:23-29.

2 Insensible fluid loss during cardiac surgery

Y Rajalingam, S Tourney and HJ McFarlane
Department of Anaesthesia, Aberdeen Royal Infirmary, Aberdeen, UK

Introduction: Insensible losses normally occur by diffusion through the skin and evaporation from the respiratory tract. Total losses per day at ambient temperature are 700 ml [1]. When a body cavity is breached during surgery evaporative losses increase and are difficult to measure. However underestimation of insensible losses has led to a reappraisal of fluid requirements perioperatively to prevent oliguria [2,3]. With invasive monitoring most clinicians prefer to titrate fluid replacement to measured variables. Cardiac patients leave theatre in a positive balance but as the early post bypass period is associated with a diuretic phase, relative fluid balance is usually achieved within a few hours. However we have observed a reluctance amongst trainee and nursing staff to give additional fluid when urine output is falling if the measured pressures are satisfactory and calculations continue to indicate a positive balance. Measurement of central venous pressure is insensitive and measurable changes only follow intravascular volume changes of 750 ml or more [3]. The prescription of a diuretic at this stage may be erroneous. This pilot study sought to quantify insensible losses during cardiac surgery.

Methods: Eight male and two female patients undergoing routine cardiac surgery (two mitral valve replacemenst, one aortic valve replacement + grafts, one mitral valve replacement + grafts) gave informed consent to the study. On arrival in the theatre suite patients were weighed using the Arjo Maximove (Arjo Ltd, Gloucester UK). This was calibrated according to manufacturer's recommendations and has an accuracy of ±0.2 kg within the normal adult range of weight. Postoperatively, once stable, and within 30 min of arrival in the ICU patients were weighed a second time. Fluid gains and losses were charted accurately during surgery including priming volumes, residual pump volumes, irrigation fluids and infusion volumes from all sources. Blood loss was estimated by conventional weighing and suction with the addition of 25% to the total. All other additions including endotracheal tube, cannulae, catheter, drains and dressings were weighed separately and added where appropriate.

Results: Results are expressed as mean ± SD. Time on bypass was 1.5 ± 0.66 hours. Weight before operation was 78.97 ± 8.8 kg. Weight increased in all patients. The expected weight gain was 3.04 ± 0.75 kg. The observed weight gain was 1.86 ± 0.73 kg. The difference between the expected and observed weight gains was significant ($P<0.002$, Student's t test). The estimated insensible loss in all patients was 1.18 ± 0.18 l equivalent to 15 ± 2.9 ml per kg.

Conclusions: We conclude that during routine cardiac surgery in adults, insensible losses of 1 l are to be expected. These losses should be taken into account in any subsequent estimation of fluid balance.

References

1. Guyton AC, Hall JE: *Textbook of Medical Physiology, 9th Edition.* Philadelphia: WB Saunders, 1996.
2. Campbell IT, Baxter JN, Tweedie IE, Taylor GT, Keens SJ: **IV fluids during surgery.** *Br J Anaesth* 1990, **65**:726-729.
3. Sweny P: **Is postoperative oliguria avoidable?** *Br J Anaesth* 1992, **67**:137-145.

3 Fentanyl and myocardial protection: is there a preconditioning mechanism?

EJS Taylor, RO Feneck and DJ Chambers*
*Departments of Anaesthetics and *Cardiac Surgical Research, The Rayne Institute, St Thomas' Hospital, London, UK*

Introduction: Ischaemic preconditioning (IPC) was first reported by Murry and colleagues . Other triggers of preconditioning have subsequently been identified and there is now substantial evidence for involvement of the opioid receptor in preconditioning of rat hearts . Kato and Foex demonstrated that fentanyl, given as a pretreatment and during reperfusion, improved postischaemic recovery of function, and suggested that a preconditioning mechanism may be involved. In this study, we set out to examine whether fentanyl acts to induce a preconditioning protection or whether it influences ischaemia-reperfusion effects.

Methods: Isolated rat hearts were Langendorff-perfused with Krebs Henseleit bicarbonate buffer (KHB) and function (left ventricular developed pressure: LVDP) measured with a fluid-filled LV balloon during a 15 min stabilisation period. Hearts (n=at least six per group) were then randomly assigned to one of five treatment groups and then subjected to 25 min global 37°C ischaemia followed by 60 min reperfusion (when recovery of function, expressed as percent of pre-ischaemic function, was measured). The five groups were: (a) Control: 30 min additional KHB perfusion, (b) Ischaemic preconditioning (IPC); three episodes of 5 min global ischaemia and 5 min reperfusion, (c) Fentanyl preconditioning (FPC); three episodes of 5 min KHB with 470 nmol/l fentanyl and 5 min KHB reperfusion, (d) Fentanyl pretreatment and reperfusion (FPT); 15 min KHB alone and 15 min KHB with 470 nmol/l fentanyl, (e) Fentanyl reperfusion (FREP); KHB reperfusion with 470 nmol/l fentanyl. Differences between groups were assessed by ANOVA and Dunnett's t-test (for multiple comparisons); $P<0.05$ was considered significant.

Results: At the end of 60 min reperfusion, recovery of LVDP was 31 ± 3.5%, 52 ± 2.8%*, 31 ± 3.4%, 47 ± 1.9%* and 46 ± 3.8%* for groups (a)–(e) respectively [*$P<0.05$ compared to group (a)].

Conclusions: We were unable to demonstrate that fentanyl, at the concentration used in this study, exerted a myocardial preconditioning protection. However, when added during reperfusion, fentanyl was beneficial and this appeared unrelated to any antiischaemic effect induced by pretreatment. The mechanism of action for this effect is currently unknown.

References

1. Murry CE, Jennings RB, Reimer KA: **Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium.** *Circulation* 1986, **74**:1124-1136.
2. Schultz JE, Gross GJ: **Opioids and cardioprotection.** *Pharmacol Ther* 2001, **89**:123-137.
3. Kato R, Foex P: **Fentanyl reduces infarction but not stunning via d-opioid receptors and protein kinase C in rats.** *Br J Anaesth* 2000, **84**:608-614.

4 Methylprednisolone exacerbates porcine pulmonary dysfunction induced by infrarenal aortic ischaemia-reperfusion

RC Baker, MA Armstrong*, SJ Allen†, FC Campbell, AAB Barros D'Sa and WT McBride†
*Departments of Surgery, *Immunobiology and †Clinical Anaesthesia, The Queen's University of Belfast, Belfast, UK*

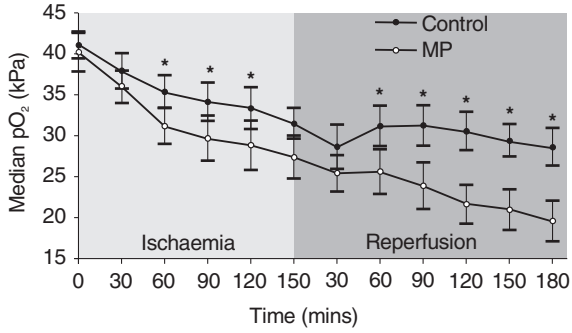
Background: Methylprednisolone (MP) at cardiac surgery has been shown to reduce perioperative proinflammatory cytokine responses, reduce neutrophil adhesion molecule expression and increase antiinflammatory interleukin-10 [1]. This effect has been linked with renal protection [2]. Paradoxically, MP administration has been associated with impaired pulmonary function [3]. The mechanism is unknown. To begin elucidating this, we investigated the hypothesis that methylprednisolone would significantly exacerbate porcine pulmonary dysfunction induced by infrarenal aortic ischaemia-reperfusion.

Methods: Forty-two male, 10–12 week old, pigs underwent pentobarbitone anaesthesia followed by tracheostomy and mechanical ventilation. The inspired oxygen concentration was maintained at 70% throughout the procedure and all animals had invasive monitoring of their systemic and pulmonary pressures. At laparotomy the infrarenal aorta was cross clamped for 150 min and then released to allow 180 min of reperfusion. The animals were randomly allocated to treatment ($n=21$) or control groups ($n=21$). After a baseline arterial blood sample was taken the treatment group received 30 mg/kg of MP and the control group a saline placebo. Arterial blood samples were obtained after 30, 60, 90, 120 and 150 min of ischaemia. Further samples were collected at 30, 60, 90, 120, 150 and 180 min into reperfusion.

Results: During the ischaemia-reperfusion period all animals showed a time dependent deterioration in arterial oxygen tension ($P<0.05$; Wilcoxon signed rank test). The PaO_2 was lower ($P<0.05$) in the treatment group compared to the control group (Mann Whitney U test) during ischaemia at time points 60, 90 and 120 min and during the reperfusion period at 60, 90, 120, 150 and 180 min (Fig. 1).

Conclusions: This porcine model demonstrates MP induced exacerbation of ischaemia reperfusion related pulmonary dysfunction. This model will allow elucidation of the relative protective or deleterious effects of MP administration as well as mechanisms of action.

Figure 1



$P<0.05$ (Mann–Whitney U test) for between group differences. Error bars display interquartile ranges. MP, methylprednisolone.

Acknowledgements

This research is supported by the British Heart Foundation.

References

1. Hill GE, Alonso A, Thiele GM, Robbins RA: **Glucocorticoids blunt neutrophil CD11b surface glycoprotein upregulation during cardiopulmonary bypass in humans.** *Anesth Analg* 1994; **79**:23-27.
2. Baker RC, Armstrong MA, Barros D'Sa AAB, Campbell FC, McClean E, McBride WT: **The effect of methylprednisolone on urinary N-acetyl-β-D-glucosaminidase/creatinine ratios in porcine vascular surgery.** *Br J Anaesth* 2001; **87**:661P.
3. Chaney MA, Nikolov MP, Blakeman B, Bakhos M, Slogoff S: **Pulmonary effects of methylprednisolone in patients undergoing coronary artery bypass grafting and early tracheal extubation.** *Anesth Analg* 1998; **87**:27-33.

5 A comparison of two methods of estimating systemic carbon dioxide production during cardiopulmonary bypass

NJ Glassford, M Torrie and RP Alston
Department of Anaesthesia, Critical Care and Pain Medicine, Royal Infirmary of Edinburgh, Edinburgh, UK

Introduction: The gold standard for assessing the efficacy of cardiopulmonary bypass (CPB) is systemic oxygen uptake (VO_2) as estimated by the Fick Principle [1]. Systemic carbon dioxide production (VCO_2) may also be determined in this way as another estimate of the aerobic efficacy. Both techniques, however, are error prone due to compounding of individual measurement errors during their arithmetical calculation. Also, they are mathematically coupled to predictor variables, such as flow rate, so invalidating statistical analysis of their relationships. VCO_2 may also be estimated as the oxygenator's CO_2 output which should be a robust and independent measure. The aim of this study was to compare the measurement of VCO_2 using the Fick Principle with that by oxygenator carbon dioxide production.

Methods: Blood was aspirated from the arterial and venous lines during CPB at initial cooling, stable hypothermia and rewarming (35°C). Samples were analysed on a blood gas analyser and CO_2 content was then estimated from its partial pressure using Kelman's algorithm [2]. The product of the arterio-venous difference in carbon dioxide content and pump flow rate was used to obtain VCO_2. VCO_2 was also estimated as the product of CO_2 concentration exhausting from, and fresh gas flow through, the oxygenator. Method comparison analysis was used to compare the techniques [3].

Results: A total of 186 measurements were made in 51 patients. The differences between results obtained by the two methods plotted against the mean of the paired results demonstrated poor agreement between the methods (limits −23, 105 ml/min). VCO_2 as predicted by the Fick Principle overestimated that determined by the oxygenator output technique with an average bias of 41 ml/min (95% CI 32–50). The bias was proportional ($r=0.75$, 95% CI 0.55–0.95). Similar relationships between the techniques were found at each individual time point during CPB.

Discussion: Either of the methods could be responsible for the poor agreement. As discussed above, the Fick method is error prone. Also, our assumption that CO_2 can accurately be estimated from its partial pressure may also be invalid in this setting. Thermal gradients across the oxygenator caused by the heat exchanger will have altered the blood solubility of CO_2 so decreasing and increasing oxygenator CO_2 output during cooling and rewarming, respectively. Also, because of the respiratory quotient, the flow of gas exhausting from, will be less than the fresh gas entering, the oxygenator. In conclusion, the two methods of VCO_2 cannot be used interchangeably.

References

1. Alston RP, Singh M, McLaren AD: **Systemic oxygen uptake during hypothermic cardiopulmonary bypass – effects of flow rate, flow character and arterial pH.** *J Thorac Cardiovasc Surg* 1989, **98**:57-768.
2. Kelman GR: **Digital computer procedure for the conversion of PCO_2 into blood CO_2 content.** *Resp Phys* 1967, **3**:111-115.
3. Bland MJ, Altman DG: **Statistical methods for assessing agreement between two methods of clinical measurement.** *Lancet* 1986, **1**:307-310.

6 'Alveolar recruitment strategy' improves arterial oxygenation after cardiopulmonary bypass

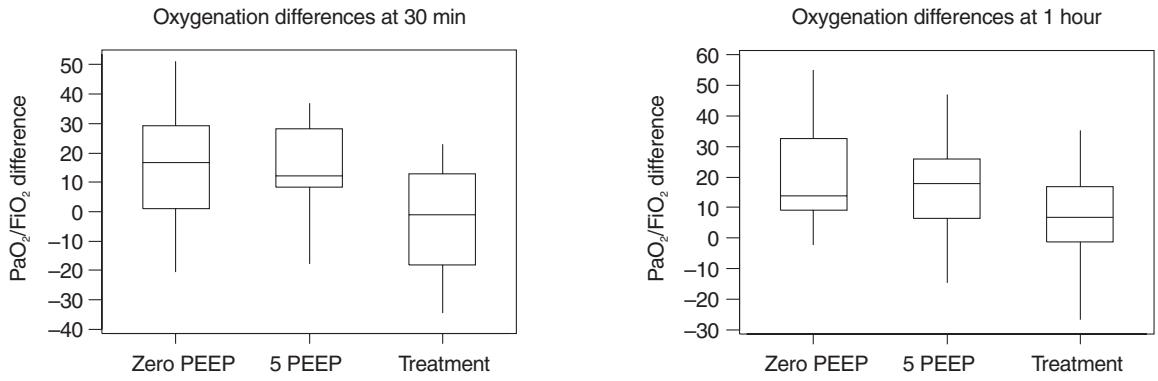
B A Claxton, J Berridge, P Morgan, H McKeague and A Mulpur
Yorkshire Heart Centre, Leeds General Infirmary, Leeds, UK

Introduction: Atelectasis occurs during general anaesthesia [1]. During cardiopulmonary bypass, this atelectasis is exacerbated by the physical collapse of the lungs. As a result, poor arterial oxygenation is often seen post-operatively. Studies using an alveolar recruitment strategy have improved oxygenation after non-cardiac surgery [2].

Methods: We tested the effect of an 'alveolar recruitment strategy' on arterial oxygenation in a prospective, randomised, controlled study of 78 patients undergoing cardiopulmonary bypass. Divided equally into three groups of 26, Group 'No PEEP' received standard post bypass manual lung inflation, and no PEEP applied until on ICU. Group '5 PEEP' received standard post bypass manual inflation, and then 5 cmH_2O PEEP applied and maintained until extubation on ICU. The third group 'treatment group', received a pressure controlled stepwise increase in PEEP up to 15 cmH_2O and tidal volumes of 18 ml/kg or a peak inspiratory pressure of 40 cmH_2O was reached. This was maintained for 10 cycles, and the PEEP of 5 cmH_2O was maintained until extubation on ICU. Arterial blood samples were analysed at 30 min post induction of anaesthesia, and then at 30 min, 1, 2 and 6 hours post bypass. The length of ICU stay, hospital stay and incidence of chest infections was recorded.

Results: In both the zero PEEP and 5 PEEP groups there was a decrease in arterial oxygenation at 30 min post bypass (mean decrease of 15.5 and 15.0 respectively), however in the treatment group there was an increase in oxygenation compared to baseline at 30 min (mean increase of 1.9). The difference between the treatment group and the other two control groups was very significant at 30 min with $P<0.001$. At 1 h post bypass the difference was significant at $P=0.002$ for the no PEEP group and $P=0.04$ for the 5 PEEP group (Fig. 1). No significant difference was found between the two control groups. At 2 and 6 hours post bypass

Figure 1



there was not a significant difference between the three groups. There was no significant difference in ICU stay, hospital stay or incidence postoperative chest infections. No complications due to the alveolar manoeuvre occurred.

Conclusions: We conclude that the application of an alveolar recruitment strategy improves arterial oxygenation up to one hour after cardiopulmonary bypass surgery. We hypothesize that this improvement is lost after one hour due to routine ICU nursing care which includes open circuit suctioning and disconnections from the ventilator thus removing the PEEP. This would allow atelectasis to reoccur. The results of this study suggest that the early impairment of gas exchange that occurs postoperatively in cardiac surgery can be reversed, thus allowing for earlier extubation.

References

1. Lindberg P, Gunnarson L, Tokics L, Secher E, Lundquist H, Brismar B, Hedenstierna G: **Atelectasis and lung function in the postoperative period.** *Acta Anaesthesiol Scand* 1992, **36**:546-553.
2. Tusman G, Bohm SH, Vasquez de Anda GF, do Campo JL, Lachmann B: **'Alveolar recruitment strategy' improves arterial oxygenation during general anaesthesia**. *Br J Anaesth* 1999, **82**: 8-13.

7 Measurement of respiratory nitric oxide in patients undergoing cardiopulmonary bypass

L Pardeshi, J Hayden-Smith, T Small*, P Arnold, A Fisher* and P Corris*
*Department of Cardiothoracic Anaesthesia and Cardiothoracic Centre and *Department of Respiratory Medicine, Freeman Hospital and University of Newcastle, Newcastle upon Tyne, UK*

Introduction: Aim of the present study was to study the effects of cardiopulmonary bypass (CPB) on endogenous nitric oxide (NO) from the lungs and to exclude mass gas flow by measuring the NO levels in the trachea held during end inspiration [1,2].

Method: Twenty adult patients undergoing elective surgery involving CPB under mild hypothermia were prospectively investigated. Patients likely to have pre-existing pulmonary hypertension, cardiac failure or lung disease (including controlled asthma) were excluded. Measurements were recorded before and after CPB. After preoxygenation with 100% oxygen for 3 min, a non-Teflon catheter was introduced into the endo-tracheal (E-T) tube via an airtight valve so that the tip of the catheter was within 3 cm of the E-T tube tip. Respiration was held during end inspiration until a plateau was seen on the nitric oxide and carbon dioxide traces. Exhaled NO was measured using a rapid highly sensitive chemiluminescence analyser (LR2000, version 2.2; Logan Research, Rochester, UK).

Results: Two-way analysis of variance, with patients and times as factors showed that the peak concentration of NO in the major airways of patients following CPB was significantly lower as compared to pre-bypass samples. End-inspiratory NO levels decreased from 7.89 ± 0.5 ppb (mean ± STD) to 4.63 ± 0.32 ppb ($P < 0.05$).

Conclusions: There is a decrease in the plateau level of exhaled nitric oxide in the major airways following CPB. The reason for these reduced levels remains uncertain. The results suggest a reduction in endogenous nitric oxide production from the lungs.

References

1. Hyde RW, Geigel EJ, Olszowka AJ, Krasney JA, Forster RE 2nd, Utell MJ, Frampton MW: **Determination of production of nitric oxide by lower airways of humans: theory.** *J Appl Physiol* 1997, **82**:1290-1296.
2. Brett SJ, Quinlan GJ, Mitchell J, Pepper JR, Evans TW: **Production of nitric oxide during surgery involving cardiopulmonary bypass.** *Crit Care Med* 1998, **26**:208-209.

8 Oesophageal Doppler monitoring of descending aortic blood flow velocity during off-pump coronary artery bypass surgery

SK Kodakat, M Delaguila, U Trivedi and R Kong
Departments of Anaesthesia and Cardiac Surgery, Sussex Cardiac Centre, Brighton, UK

Objective: Acute haemodynamic changes occur frequently during off-pump coronary artery surgery (OPCAB). Currently used haemodynamic monitors are not satisfactory during this procedure. We assessed the feasibility of oesophageal Doppler insonation of the descending aorta to measure cardiac output and other haemodynamic indices continuously during OPCAB.

Methods: Twenty-five consecutive patients scheduled to undergo OPCAB were studied prospectively. The ethical committee waived the need for informed consent because the oesophageal Doppler monitor (CardioQ, Deltex, UK) was already in routine use in our hospital. Haemodynamic parameters were recorded at multiple intervals after anaesthetic induction; before sternotomy; before, during and after each anastomosis and at the end of surgery. Stroke volume, cardiac output (CO), corrected flow time (FTc), peak velocity(PV) and mean acceleration were measured or calculated by the Doppler monitor. Mean arterial pressure (MAP) and central venous pressure(CVP) were measured from radial artery and internal jugular venous cannulae respectively.

Results: Satisfactory Doppler signals were not obtained in one patient. Two patients were converted to standard revascularisation using cardiopulmonary bypass because of technical considerations. One, 7 and 14 patients had one, two and three vessels grafted respectively. The haemodynamic data on these 22 patients are presented in Table 1 as mean and standard deviation.

Conclusion: The oesophageal Doppler was able to monitor cardiac output and other haemodynamic parameters continuously throughout OPCAB. Further studies are needed to determine whether or not active haemodynamic management assisted by oesophageal Doppler monitoring during OPCAB improves patient outcome.

Table 1

	CO (l/min)	MAP (mmHg)	CVP (mmHg)	FTc (s)	PV (cm/s)
Post Induction	4.28 ± 1.30	78 ± 21		325 ± 44	50 ± 12
Pre LAD	5.12 ± 1.41	72 ± 17	8 ± 4	355 ± 29	54 ± 17
During LAD	4.76 ± 1.26	68 ± 13	10 ± 4	353 ± 45	53 ± 16
Post LAD	5.57 ± 1.68	71 ± 15	9 ± 5	359 ± 45	55 ± 16
Pre Cx	5.46 ± 1.84	67 ± 12	13 ± 5	353 ± 60	53 ± 17
During Cx	4.19 ± 1.12	68 ± 10	16 ± 5	343 ± 50	48 ± 12
Post Cx	5.67 ± 1.69	70 ± 13	7 ± 4	375 ± 56	57 ± 13
Pre PDA	5.38 ± 1.39	83 ± 17	12 ± 4	375 ± 42	52 ± 17
During PDA	5.38 ± 1.15	73 ± 13	14 ± 3	355 ± 61	54 ± 16
Post PDA	5.14 ± 1.19	68 ± 13	9 ± 4	356 ± 39	53 ± 16
Chest closed	4.89 ± 1.34	67 ± 12	9 ± 3	338 ± 40	57 ± 15

LAD (left anterior descending artery) = 21 patients, Cx (circumflex artery) = 18 patients, PDA (posterior descending artery) = 15 patients.
Pre, during and post refer to immediately before, during and after anastomosis.