Original Article

# Infusion of low-dose vasopressin improves left ventricular function during separation from cardiopulmonary bypass: A double-blind randomized study

**Ahmed Said Elgebaly, Mohab Sabry[1]**

Departments of Anesthesia and SICU, [1]Cardiothoracic Surgery, Faculty of Medicine, Tanta University, Egypt

## ABSTRACT

We aimed to investigate whether low-dose vasopressin administered to patients undergoing coronary artery bypass grafting (CABG) surgery with preexisting mild to moderate systolic dysfunction can produce sustained improvement in cardiac function. This double-blind randomized study was conducted in a hospital where a single anesthetic and surgical team performed elective CABG. Twenty patients aged 32–61 years who underwent elective CABG between January 2007 and December 2007 were enrolled in this study. The patients randomly received either vasopressin 0.03 IU/min (Group A) or normal saline (Group B) in equal volume for 60 min after cardiopulmonary bypass (CPB). The cardiac output, cardiac index, stroke volume index, fractional area of contraction and systemic vascular resistance index were significantly higher in Group A than in Group B. Adrenaline (mean dose: 0.06 μg/kg•min–1) was required in seven patients from Group B but in none of the Group A patients on initial separation from CPB ($P< 0.05$). Of the 10 patients in Group B, five required phenylepherine to maintain the mean arterial pressure (MAP) >65 mmHg, whereas none of the Group A patients required phenylephrine for MAP regulation ($P< 0.05$). We conclude that Infusion of low-dose vasopressin for patients with mild to moderate left ventricular systolic dysfunction during separation from CPB is beneficial for the postoperative hemodynamic profile, reduces the catecholamine doses required and improves left ventricular systolic function.

Received: 29-11-11
Accepted: 03-03-12

**Key words:** Cardiopulmonary bypass, left ventricular function, transesophageal echocardiography, vasopressin

## INTRODUCTION

Ventricular dysfunction and pulmonary hypertension are significant risk factors that may increase the incidence of morbidity and mortality in cardiac patients undergoing cardiac surgery.[1] Reduced left ventricular function in patients undergoing coronary artery bypass grafting (CABG) with cardiopulmonary bypass (CPB) may lead to persistent hypotension.[2] Profound hypotension is a life-threatening condition that cannot be managed easily by administration of fluids, inotropes or even vasopressor catecholamines.[3] Infusion of catecholamines during surgery in fact often complicates cardiovascular stabilization by producing arrhythmias.[4,5] Previous studies have introduced vasopressin as an adjunct to catecholamines in cases of cardiac arrest and advanced vasodilatory shock, and it has even been demonstrated to be more effective than catecholamines.[5-7]

Several case reports and studies have demonstrated that administration of nonpressor doses of vasopressin restores the blood pressure in normal subjects who experience septic shock[7,8] and in postcardiotomy shock

Access this article online
Website: www.annals.in
DOI: 10.4103/0971-9784.95076
Quick Response Code:

**Address for correspondence:** Dr. Ahmed Elgebaly, Department of Anesthesia and SICU, Faculty of Medicine, Tanta University, 43 IBN Elfard Street, Elgharbia, Tanta, Egypt. E-mail: elgebaly_13@hotmail.com

patients with documented hyporesponsiveness to catecholamines.[9] In most of these cases, vasopressin was administered as a last resort, after large doses of norepinephrine had failed to significantly increase blood pressure. Vasopressin has also been reported to improve the mean arterial pressure (MAP) and reduce the required norepinephrine dose when used in adjunct with norepinephrine.[6]

This study aimed to determine whether administration of low-dose vasopressin in patients with preexisting mild to moderate systolic dysfunction undergoing CABG surgery produced sustained improvement in cardiac function. We used transesophageal echocardiography (TEE) to continuously assess left ventricular function after infusion of low-dose vasopressin, as described in a previous study.[10]

## MATERIALS AND METHODS

After obtaining the approval from the Ethics and Research Committee of the Department of Anaesthesia and written informed consent from all the study participants, the study was conducted in the Cardiothoracic Surgery Department.

A total of 26 patients aged 32–61 years were screened from January 2007 to December 2007 and 20 were enrolled in the study based on the following inclusion criteria: presence of mild to moderate left ventricular systolic dysfunction (ejection fraction [EF] = 35–50%) and underwent elective CABG. Patients with an EF less than 30% and those who were in shock or in a critical hemodynamic state, as confirmed by TEE, were excluded. Further, patients with the appearance of shock or severe hemodynamic instability that seemed "intractable" in simple preload manipulations (fluid infusion) and that occurred in combination with simultaneous (observed by TEE) impairment of left ventricular function during the operation and in the first 2 h postoperatively were excluded. Other reasons for exclusion were confirmed as hepatic and/or renal, thyroid and/or adrenal disease; significant carotid stenosis or any event of intraoperative brain ischemia as documented by continuous transcranial $SvO_2$ (INVUS); significant peripheral obstructive arteriopathy; documented pulmonary hypertension expressed by a systolic pulmonary artery pressure of >35 mmHg; and chronic obstructive pulmonary disease, as confirmed by preoperative spirometry, thorax X-ray and blood gas analysis. A total of six such cases were excluded from the study.

In all 20 patients, right internal jugular vein was catheterized with a three lumen central venous catheter and a Swan-Ganz catheter for continuous measurement of pulmonary artery pressure, cardiac output (CO) and mixed venous blood saturation. A urinary catheter was introduced for measurement of hourly diuresis. In addition, a transesophageal ultrasound probe was introduced for intra- and postoperative estimation of cardiac function. The two right internal jugular vein catheters and the urinary catheter were retained for 24 h.

### Anesthesia

The patients were premedicated on the morning of the operation by intramuscular (i.m.) injection of 0.01 mg/ kg morphine; and received 3–5 mg midazolam in the preparation room. Anesthesia was induced with 2–4 mg/kg sodium thiopental, 5–10 μg/ kg fentanyl and 0.1 mg/kg pancronium for muscle relaxation. Ventilation was controlled manually using 100% oxygen delivered through a face mask. Tracheal intubation was carried out 3–5 min later. Anesthesia was maintained using 1.0–1.5% isoflurane in oxygen.

### TEE data

The transgastric midpapillary short axis view was monitored during the study period by a separate anesthesiologist other than the one responsible for the conduct of anesthesia. The left ventricular endocardium was traced at the end of the diastole to obtain the end-diastolic area and end-diastolic circumference. The endocardium and epicardium were also traced at the end of the systole to obtain the end-systolic area, with the papillary muscle included, end-systolic circumference, and total area enclosed by the left ventricular epicardium and the right side of the septum.

### Surgery

The patients were operated upon through standard median sternotomy. Establishment of cardiopulmonary bypass was done using direct aortic cannulation of the ascending aorta and single venous cannula through right atrial appendage. Also, aortic root cannula was inserted for antegrade cardioplegia and deairing of the heart after surgery The protocol of coronary revascularisation was to graft the left anterior descending artery (LAD) using the left internal mammary artery (LIMA) and grafting of all other coronary arteries using saphenous vein grafts (SVG) All distal anastomoses were done first then proximal anastomoses of the SVG to the ascending aorta were done on beating heart with partial aortic clamping.

### Parameters

The CO, cardiac index (CI), stroke volume index and systemic vascular resistance index (SVRI) were measured in this study. The pulmonary artery systolic pressure was measured using the Swan-Ganz catheter. Contractility was assessed by estimating the fractional area of contraction (FAC).

TEE analysis was conducted at the following time intervals: 20 min after induction of anesthesia, 20 min after weaning from CBP and at 40 and 60 min after CBP and at the end of surgery.

All patients were operated under moderately hypothermic CBP (nasopharyngeal temperature: 28°C). Blood cardioplegia (one part blood, three parts crystalloid) was administered for myocardial protection via aortic root every 20–30 min. Toward the end of the CBP, patients were randomly divided into two groups. Group A patients (the vasopressin group) received continuous infusion of 0.03 IU/min vasopressin solution (Pitressin, Pfizer, Karlsruhe, Germany) at the rate of 22 mL/h. The infusion was initiated 10 min before separation from CPB, and was continued throughout the operation until 60 min after termination of the CPB intravenously through a central line. Similarly, Group B patients (the placebo group) received normal saline solution administered at the same rate for the same duration. Both the solutions were prepared by a nurse. Except for the nurse, all other staff present in the operating room, including the surgeon and anesthetist, were blinded to the type of infused solution given to each patient. Dynamic measurements and FAC were estimated using TEE within 20 min after weaning, 40 min after CBP and at the end of operation. Hypertensive crisis or elevated pulmonary artery pressure was not observed in any of the patients. If the CI was less than 2.2 L/min/m$^2$, the patients received an initial dose of 0.05 μg/kg/min adrenaline. This dose was increased up to 0.1 μg/kg/min if required, until the patient was hemodynamically stable. Phenylephrine was administered at 50–100 μg intravenously (i.v.) to maintain the MAP above 60 mmHg.

### Statistical analysis

Hemodynamic and echocardiographic data were analyzed using one-way and repeated measure ANOVA, as appropriate. The adrenaline and phenylephrine requirements were analyzed using the $\chi^2$ test. Comparisons between bypass time and number of grafts were analyzed using the Student's *t* test. $P<0.05$ was considered significant. All the analyses were performed using SPSS 16 statistical package.

## RESULTS

Table 1 shows the demographic and clinical data. There was no significant difference between the two groups with respect to age, sex, weight, height, preoperative EF, hypertension, incidence of congestive heart failure, diabetes mellitus, number of grafts done, medications before the operation, CPB time and ICU and hospital stay times. All the patients were successfully separated from CBP in the initial attempt. Seven patients in the placebo group [Table 2] required adrenaline (mean dose: 0.06 μg/kg/min, whereas none of the patients in the vasopressin group required adrenaline infusion on initial separation from CBP ($P<0.05$). Forty minutes

**Table 1: Demographic data, mean (SD), ratios and number (percentage) of patients**

| | Vasopressin group (*n*=10) | Placebo group (*n*=10) |
|---|---|---|
| Age (years) | 60.9 (9.3) | 58.1 (8.6) |
| Sex (M/F) | 5/4 | 6/4 |
| Weight (kg) | 59.4 (9.4) | 57 (7.8) |
| Height (cm) | 164 (8.3) | 168 (9.1) |
| EF (%) | 47.4 (8.5) | 46.6 (9.5) |
| Hypertension (Y/N) | 6/4 | 5/5 |
| Congestive heart failure (CHF) | 10% | 20% |
| Diabetes mellitus (Y/N) | 5/5 | 7/3 |
| Number of grafts | 2.7 (0.6) | 2.8 (0.6) |
| CPB (min) | 174 (57.3) | 172.4 (66) |
| β-blockers | 9 | 8 |
| Calcium channel blockers | 8 | 9 |
| ACE inhibitors | 3 | 2 |
| ICU stay (days) | 5.5 (3) | 5.9 (2.9) |
| Hospital stay (days) | 10.7 (6.2) | 12.2 (8.7) |

EF - Ejection fraction; CPB - Cardiopulmonary bypass; ACE inhibitors - Acetylcholine esterase inhibitors; ICU - Intensive care unit

**Table 2: Number of patients separated from CPB and requiring adrenaline or phenylephrine infusion**

| | Vasopressin group (*n*=10) | Placebo group (*n*=10) |
|---|---|---|
| Successful separation from CPB on the initial attempt | 10 | 10 |
| Patients required adrenalin infusion on initial separation from CPB with mean dose of 0.06 μg/kg/min | 0 | 7 |
| Patients required adrenalin infusion 40 min after the end of CBP with mean dose of 0.05 μg/kg/min | 3 | 7 |
| Patients required phenylephrine infusion with mean dose of 80 μg | 0 | 5 |

CPB - Cardiopulmonary bypass

after separation from CBP, three patients from the vasopressin group and seven patients in the placebo group required 0.05 μg/kg/min adrenaline until the end of the surgery. Of the 10 patients in the placebo group, five required phenylepherine (mean dose, 80 μg) to maintain MAP at >65 mmHg, whereas none of the patients in the vasopressin group required phenylephrine ($P<0.05$).

Hemodynamic and echocardiographic data for both groups are shown in Tables 3 and 4. There were no significant differences between the two groups with respect to the heart rate and MAP at all the times investigated. The CI, FAC and SVRI were significantly higher in the vasopressin group than in the placebo group at 20 min after CPB, 60 min after CBP and at the end of surgery. The increases in CI and FAC at 20 and 60 min after CPB and at the end of surgery in the vasopressin group were significantly higher ($P< 0.05$) than the respective baseline values measured at 20 min after induction of anesthesia. On the other hand, no such changes in the CI or FAC as compared with the baseline values were observed in the placebo group.

## DISCUSSION

Several previous studies have demonstrated that the administration of vasopressin restores vascular tone in patients following CPB, especially in cases that are refractory to norepinephrine.[11-13] These results could be attributed to the known action of vasopressin. Earlier Morales *et al* had shown that vasopressin is important in the maintenance of vascular tone after CPB and that a deficiency in vasopressin contributes to vasodilatory shock. The correction of this deficiency with low-dose vasopressin "replacement" therapy dramatically improves hemodynamics in post-CPB vasodilatory shock.[14]

The increased CI is attributed not only to the preload and afterload changes[12,13,15,16] but also to the increased myocardial contractility. In fact, vasopressin infusion tends to improve myocardial performance by increasing the intramyocardial calcium concentrations and producing coronary artery vasodilatation in combination with the increase in myocardial blood flow due to increased systemic perfusion pressure.[4,7]

**Table 3: Hemodynamic data of patients in the two study groups**

| Group | Parameter | 20 min after induction of anesthesia | 20 min after the end of CBP | 40 min after the end of CBP | 60 min after the end of CBP (end of surgery) | *P* value |
|---|---|---|---|---|---|---|
| Vasopressin group (*n*=10) | HR (beat/min) | 78.0 (14) | 100.6 (3.2) | 105.8 (2.1) | 102.2 (6.1) | 0.085 |
| | CVP (cm $H_2O$) | 5.0 (2.5) | 7.0 (3.1) | 6.5 (2.1) | 7.0 (2.3) | 0.075 |
| | MAP (mmHg) | 70.8 (5.1) | 69.4 (4.3) | 68.0 (3.2) | 70.2 (4.5) | 0.066 |
| | PASP (mmHg) | 31.0 (2.1) | 22.3 (6.1) | 23.2 (5.1) | 23.2 (5.1) | 0.084 |
| Placebo group (*n*=10) | HR (beat/min) | 80.0 (5.1) | 94.8 (2.2) | 95.2 (8.3) | 101.6 (6.2) | 0.538 |
| | CVP (cm $H_2O$) | 4.0 (6.8) | 8.3 (2.3) | 7.5 (4.2) | 7.2 (3.1) | 0.762 |
| | MAP (mmHg) | 71.8 (3.2) | 68.1 (6.3) | 71.2 (2.1) | 69.8 (4.3) | 0.538 |
| | PASP (mmHg) | 30.2 (6.2) | 28.3 (4.8) | 29.1 (3.1) | 29.5 (5.8) | 0.699 |

Values are presented as mean (SD). $P<0.05$ was considered statistically significant

**Table 4: Transesophageal echocardiography data of the patients**

| Group | Parameter | 20 min after induction of anesthesia | 20 min after the end of CPB | 40 min after the end of CBP | 60 min after the end of CBP (end of surgery) | *P* value |
|---|---|---|---|---|---|---|
| Vasopressin group (*n*=10) | CO (L/min) | 4.1 (0.5) | 6.1 (1.5)*† | 5.8 (3.8)*† | 5.2 (2.5)*† | 0.042 |
| | CI (L/min/$m^2$) | 2.4 (0.2) | 3.2 (1.2)*† | 3.1 (2.3)*† | 3.0 (2.2)*† | 0.003 |
| | SVI (Ml/beat·$m^2$)$^{-1}$ | 25.0 (3.5) | 30.2 (3.8)*† | 31.8 (6.5)*† | 29.1 (6.2)*† | 0.004 |
| | FAC (%) | 33.4 (2.8) | 51.1 (3.4) *† | 50.8 (6.5)*† | 51.0 (6.2) *† | 0.001 |
| | SVRI (dyne.s/($cm^5$·$m^2$)$^{-1}$) | 1754.2 (163.1) | 2125.3 (101.3)*† | 2280.2 (119.2)*† | 2310.3 (231.0)*† | 0.002 |
| Placebo group (*n*=10) | CO (L/min) | 4.5 (3.2) | 4.8 (2.3) | 4.5 (3.2) | 4.8 (3.1) | 0.567 |
| | CI (L/min/$m^2$) | 2.2 (1.5) | 2.9 (1.3) | 2.8 (2.5) | 2.7 (2.4) | 0.566 |
| | SVI (Ml/(beat·$m^2$)$^{-1}$) | 26.1 (3.5) | 22.8 (2.1) | 25.2 (4.2) | 24.9 (3.10) | 0.619 |
| | FAC (%) | 36.2 (3.1) | 38.2 (2.4) | 37.8 (6.8) | 36.4 (5.2) | 0.352 |
| | SVRI (dyne.s/($cm^5$·$m^2$)$^{-1}$) | 976.9 (134.1) | 1004.2 (146.6) | 1198.6 (116.8) | 2110.1 (133.2) | 0.084 |

Values are presented as mean (SD). *$P<0.05$ compared with the placebo group. †$P<0.05$ compared with baseline readings (20 min after induction of anesthesia) in the vasopressin group

Several studies have demonstrated the augmented vasoconstrictor action of vasopressin, which persists for up to 2 h, in patients with hypotension who do not respond to high-dose norepinephrine, dopamine and fluid resuscitation.[17,18] Moreover, vasopressin has an important advantage in that the vasoconstriction in the coronary and cerebral circulation is less pronounced than in other circulations.[19] In addition to the double beneficial action of vasopressin in the myocardium and brain, it exerts protective action on the kidneys as well. Experimental studies on protocols for short and prolonged cardiac arrest have shown that vasopressin produced a significantly higher vital organ blood flow and cerebral oxygen delivery than did epinephrine.[20,21] The increased urine output is a remarkable result of infused vasopressin that, according to several studies, is caused by the increased MAP and, consequently, improves the glomerular filtration rate.[22,23]

In our study, patients receiving vasopressin showed a significant postoperative increase in the EF, our results are similar to those of Papadopoulos *et al*.[2] To the best of our knowledge, no other studies have recorded and evaluated this hemodynamic parameter. Our study showed a significant increase in the CO, CI, FAC and SVRI. Increased systemic vascular resistance mainly occurs because of vasopressin-induced systemic vasoconstriction, and observed in patients with shock rather than in those with a normal hemodynamic state.[14,24]

Our study also showed that pulmonary vascular resistance and mean pulmonary artery pressure were not affected by the vasopressin infusion. This may be attributed to the observed vasodilatory effect of vasopressin in the pulmonary vasculature,[12,16] which has already been experimentally confirmed and is due to the release of NO by the endothelial pulmonary capillaries.[24] Because of the above-described action, vasopressin has been successfully used by Tayama *et al*.[24] in patients with preoperative pulmonary hypertension who underwent cardiac surgery.

Our data regarding the postoperative requirement of epinephrine were in agreement with the results reported by Papadopoulos *et al*.,[2] and showed that the percentage of patients requiring epinephrine was significantly lower in the vasopressin group than in the control group. Adrenaline (mean dose: 0.06 $\mu$g/kg/min) was required in seven patients in the placebo group, while none of the patients in the vasopressin group required adrenaline infusion on separation from CBP ($P< 0.05$). Forty minutes after separation from CBP, three patients in the vasopressin group required an infusion of 0.05 $\mu$g/kg//min adrenaline till the end of surgery. Of the 10 patients in the placebo group, five required phenylepherine (mean dose: 80 μg) to maintain MAP above 65 mmHg, whereas none of the patients in the vasopressin group required phenylephrine ($P<0.05$).

Our study has an important limitation of small sample size. The study included only 20 participants who fulfilled all the inclusion criteria and had undergone surgery for coronary artery disease. The sample size was restricted to 20 cases because of logistical reasons; we could only schedule one surgery per week. Moreover, the study drug vasopressin was provided free of cost to all the study participants, limiting the inclusion of more cases.

## CONCLUSIONS

In summary, infusion of 0.03 IU/min vasopressin towards the end of CPB and for the first hour after CABG in patients with low EF is a beneficial technique. It significantly reduces the required doses of catecholamines, resulting in a better hemodynamic profile for the first hour. The vasopressin infusion in low doses (0.03 IU/min) appears to prevent the incidence of postcardiotomy vasodilatory shock. Finally, low-dose vasopressin may decrease both the dose and the duration of catecholamine administration; therefore, it could be a useful agent for decreasing all the side-effects of CPB.

## ACKNOWLEDGMENT

We would like to thank Dr. Timor Mostafa Abdalla, MD. Department of Cardiology, Faculty of Medicine, Tanta University, Egypt, for his assistance in using TEE in this study. Also, we thank all of the nurses in our postanaesthesia care unit for caring our patients.

## REFERENCES

1. Khedr H, El-dian A. Assessment of left ventricular function by TEE after a single dose of milrinone during separation from cardiopulmonary bypass. Egypt J Anesth 2003;19:127-30.
2. Papadopoulos G, Sintou E, Siminelakis S, Koletsis E, Baikoussis NG, Apostolakis E. Perioperative infusion of low dose of vasopressin for prevention and management of vasodilatory vasoplegic syndrome in patients undergoing coronary artery bypass grafting: A double-blind randomized study. J Cardiothorac Surg 2010;5:17.
3. Laffey JG, Boylan JF, Cheng DC. The systemic inflammatory response to cardiac surgery: implications for the anesthesiologist. Anesthesiology 2002;97:215-52.
4. Luckner G, Dunser MW, Jochberger S, Mayr VD, Wenzel V, Ulmer H, *et al*. Arginine vasopressin in 316 patients with advanced vasodilatory

shock. Crit Care Med 2005;33:2659-66.
5. Dunser MW, Wenzel V, Mayr AJ, Hasibeder WR. Management of vasodilatory shock: defining the role of arginine vasopressin. Drugs 2003;63:237-56.
6. Wenzel V, Krismer AC, Arntz HR, Sitter H, Stadlbauer KH, Lindner KH. A comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation. N Engl J Med 2004;350:105-13.
7. Dunser MW, Mayr AJ, Ulmer H, Knotzer H, Sumann G, Pajk W, *et al*. Arginine vasopressin in advanced vasodilatory shock: A prospective, randomized, controlled study. Circulation 2003;107:2313-9.
8. McGaw C, Scarlett M, Irvine R, Ramphal P. Vasopressin for refractory hypotension during cardiopulmonary bypass. West Indian Med J 2007;56:550-4.
9. Dunser MW, Mayr AJ, Ulmer H, Ritsch N, Knotzer H, Pajk W, *et al*. The effects of vasopressin on systemic hemodynamics in catecholamine-resistant septic and postcardiotomy shock: a retrospective analysis. Anesth Analg 2001;93:7-13.
10. Schluter M, Langenstein BA, Polster J, Kremer P, Souquet J, Engel S, *et al*. Transoesophageal cross-sectional echocardiography with a phased array transducer system. Technique and initial clinical results. Br Heart J 1982;48:67-72.
11. Morales DL, Gregg D, Helman DN, Williams MR, Naka Y, Landry DW, *et al*. Arginine vasopressin in the treatment of 50 patients with postcardiotomy vasodilatory shock. Ann Thorac Surg 2000;69:102-6.
12. Argenziano M, Choudhri AF, Oz MC, Rose EA, Smith CR, Landry DW. A prospective randomized trial of arginine vasopressin in the treatment of vasodilatory shock after left ventricular assist device placement. Circulation 1997;96(9 Suppl): II-286-90.
13. Masetti P, Murphy SF, Kouchoukos NT. Vasopressin therapy for vasoplegic syndrome following cardiopulmonary bypass. J Card Surg 2002;17:485-9.
14. Morales DL, Garrido MJ, Madigan JD, Helman DN, Faber J, Williams MR, *et al*. A double-blind randomized trial: Prophylactic vasopressin reduces hypotension after cardiopulmonary bypass. Ann Thorac Surg 2003;75:926-30.
15. Argenziano M, Chen JM, Choudhri AF, Cullinane S, Garfein E, Weinberg AD, *et al*. Management of vasodilatory shock after cardiac surgery: Identification of predisposing factors and use of a novel pressor agent. J Thorac Cardiovasc Surg 1998;116:973-80.
16. Argenziano M, Chen JM, Cullinane S, Choudhri AF, Rose EA, Smith CR, *et al*. Arginine vasopressin in the management of vasodilatory hypotension after cardiac transplantation. J Heart Lung Transplant 1999;18:814-7.
17. Leone M, Albanese J, Delmas A, Chaabane W, Garnier F, Martin C. Terlipressin in catecholamine-resistant septic shock patients. Shock 2004;22:314-9.
18. Novella S, Martínez AC, Pagán RM, Hernández M, García-Sacristán A, González-Pinto A, *et al*. Plasma levels and vascular effects of vasopressin in patients undergoing coronary artery bypass grafting. Eur J Cardiothorac Surg 2007;32:69-76.
19. Delmas A, Leone M, Rousseau S, Albanèse J, Martin C. Clinical review: Vasopressin and terlipressin in septic shock patients. Crit Care 2005;9:212-22.
20. Wenzel V, Lindner KH. Employing vasopressin during cardiopulmonary resuscitation and vasodilatory shock as a lifesaving vasopressor. Cardiovasc Res 2001;51:529-41.
21. Wenzel V, Lindner KH, Prengel AW, Maier C, Voelckel W, Lurie KG, *et al*. Vasopressin improves vital organ blood flow after prolonged cardiac arrest with postcountershock pulseless activity in pigs. Crit Care Med 1999;27:486-92.
22. Holmes CL, Walley KR, Chittock DR, Lehman T, Russell JA. The effects of vasopressin on hemodynamics and renal function in severe septic shock: A case series. Intensive Care Med 2001;27:1416-21.
23. Albright TN, Zimmerman MA, Selzman CH. Vasopressin in the cardiac surgery intensive care unit. Am J Crit Care 2002;11:326-30; quiz 331-2.
24. Tayama E, Ueda T, Shojima T, Akasu K, Oda T, Fukunaga S, *et al*. Arginine vasopressin is an ideal drug after cardiac surgery for the management of low systemic vascular resistant hypotension concomitant with pulmonary hypertension. Interact Cardiovasc Thorac Surg 2007;6:715-9.

**Cite this article as:** Elgebaly AS, Sabry M. Infusion of low-dose vasopressin improves left ventricular function during separation from cardiopulmonary bypass: A double-blind randomized study. Ann Card Anaesth 2012;15:128-33.

**Source of Support:** Nil, **Conflict of Interest:** None declared.