BRIEF COMMUNICATIONS

VASOPRESSIN FOR REFRACTORY HYPOTENSION DURING CARDIOPULMONARY BYPASS

Martin P. Talbot, MD, Isabelle Tremblay, MD, André Y. Denault, MD, FRCP(C), and Sylvain Bélisle, MD, FRCP(C),
Montreal, Quebec, Canada

A 72-year-old, 42-kg woman with aortic stenosis was referred for coronary arteriography and hemodynamic evaluation. Her history included aorta-bifemoral bypass, hypothyroidism, and non–Q wave myocardial infarction (Killip III) after a carotid endarterectomy. She was on a regimen of ramipril (discontinued on admission), metoprolol, aspirin, oral nitrates, furosemide, levothyroxine sodium (Synthroid), and pravastatin.

Coronary arteriography showed severe tritruncular disease; hemodynamic evaluation and transthoracic echocardiography showed severe aortic disease and grade 2/4 mitral regurgitation. Severe diastolic dysfunction and reduced ejection fraction (35%) with diffuse hypokinesis were present. The electrocardiogram showed signs of left ventricular hypertrophy. A revascularization operation and aortic valve replacement were scheduled. Preoperative work-up revealed cold agglutinins at 24°C.

On arrival in the operating room, blood pressure was 134/42 mm Hg and the heart rate was 58 beats/min. General anesthesia was induced with sufentanil 60 μg, midazolam 5 mg, and pancuronium 5 mg. Hemodynamics remained stable until institution of cardiopulmonary bypass (CPB).

At the beginning of CPB, electrical activity persisted despite the infusion of large amounts of warm cardioplegic solution (total 2100 mL); use of hypothermia was limited by the cold agglutinins. During CPB, the patient became hypotensive (mean arterial pressure ~40 mm Hg), and with each bolus of cardioplegic solution, hypotension worsened. In addition to multiple 100 μg- and 200-μg boluses, a phenylephrine infusion was started, soon followed by norepinephrine. Doses were increased—phenylephrine up to 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ plus several 200-μg boluses and norepinephrine 12 μg/min—without substantial improvement. Because of refractory hypotension, 1 unit of vasopressin (20 U/mL; Fujisawa Canada Inc, Markham, Ontario, Canada) was given. Mean arterial pressure normalized at about 80 mm Hg, allowing us to taper the phenylephrine and norepinephrine infusions. A second dose of vasopressin was given during CPB for recurring hypotension. Subsequently, the procedure was uneventful (Fig 1). The hemoglobin value rose from 34 mg/dL at the onset of CPB to 54 mg/dL with administration of 2 units of blood (after vasopressin boluses). Blood gas levels during CBP are given in Table I. The patient was transferred to the intensive care unit receiving norepinephrine 4 μg/min and nitroglycerin 17 μg/min. Vasopressors were stopped and the patient was extubated on postoperative day 1. She was discharged from the hospital on postoperative day 6, without any apparent sequel.

Discussion. Vasopressin is a potent endogenous vasoconstricting agent, acting through the increase of arteriolar systemic resistances. Many recent studies have shown vasopressin to be effective in the treatment of refractory hypotension.

Interest has been focused on treatment of hypotension resulting from distributive shock, as in sepsis or post-CPB states. These states are characterized by low peripheral vascular resistances, catecholamine-resistant hypotension, altered peripheral perfusion, and high cardiac output. Landry and associates[1] showed that the blood levels of vasopressin in septic shock were abnormally low. This finding supports the hypothesis that those patients had depleted vasopressin stocks and/or dysfunctional baroreflexes that would cause insufficient vasopressin secretion. They also reported instances of effective treatment of hypotension with vasopressin in septic shock.[2] After the administration of vasopressin, the catecholamine needs were reduced and diuresis improved.

Similar to septic shock, CPB causes an inflammatory response leading to a vasoplegic state, in which Argenziano and colleagues[3] showed that blood levels of vasopressin were lower than those in patients with cardiogenic shock. Their retrospective analysis of 40 cases of vasodilatory shock after cardiac surgery treated with vasopressin showed no rebound hypertension or mesenteric or peripheral ischemia, along with an improvement of mean arterial pressure and a diminution of catecholamine needs. An ejection fraction below 35% and long-term treatment with angiotensin-converting enzyme inhibitor predicted vasodilatory shock after CPB.

From the Department of Anesthesia and Reanimation, Montreal Heart Institute, University of Montreal, Montreal, Quebec, Canada.

Received for publication Jan 6, 2000; accepted for publication March 16, 2000.

Address for reprints: André Denault, MD, Montreal Heart Institute, Department of Anesthesia and Reanimation, 5000 Belanger St East, Montreal, Quebec, Canada H1T 1C8 (E-mail: denault@videotron.ca).

J Thorac Cardiovasc Surg 2000;120;401-2

0022-5223/2000 $12.00 + 0 **12/54/107208**
doi:10.1067/mtc.2000.107208

The Journal of Thoracic and
Cardiovascular Surgery
August 2000

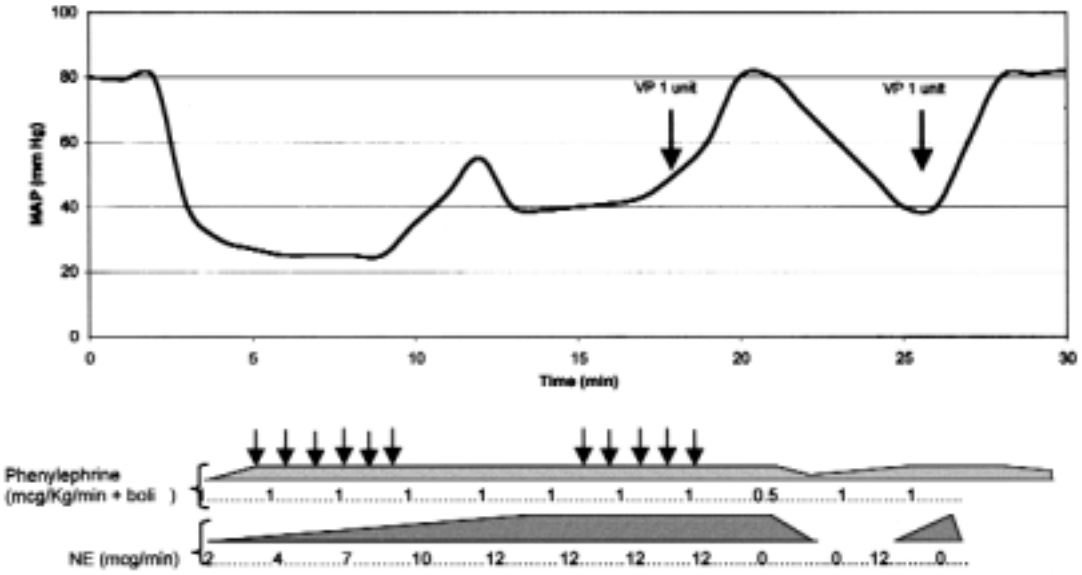

Fig 1. Evolution of mean arterial pressure *(MAP)* and medication given after onset of cardiopulmonary bypass (CPB). *NE,* Norepinephrine.

Table I. *Blood gas values during CPB*

Blood gas values				
pH	7.41	7.43	7.33	7.39
Arterial P_{CO_2} (mm Hg)	30	29	37	33
Arterial P_{O_2} (mm Hg)	318	310	323	285
Base excess (mmol/L)	–4.6	–4.2	–4.8	–3.4
Lactate (mg/dL)	5.8	6.0	7.2	6.6

We report here the case of a patient with refractory hypotension at the beginning of CPB. Hemodilution at the onset of CPB, high circulating potassium concentrations from the cardioplegic solution, and long-term use of ramipril and diuretics are the causes. Use of a 1-unit bolus of vasopressin in this patient with an important history of vascular disease permitted a rapid correction of the mean arterial perfusion, while allowing us to taper doses of other pressors without any noticeable side effect. Efficiency and safety of this promising new pressor agent during CPB will need confirmation through a randomized trial.

REFERENCES

1. Landry DW, Levin HR, Gallant EM, Ashton RC, Seo S, D'Alessandro D, et al. Vasopressin deficiency contributes to the vasodilation of septic shock. Circulation 1997;95:1122-5.
2. Landry DW, Levin HR, Gallant EM, Seo S, D'Alessandro D, Oz MC, et al. Vasopressin pressor hypersensitivity in vasodilatory septic shock. Crit Care Med 1999;25:1279-82.
3. 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.