Hypotension

By
Nik Nikam, M.D.

Hypotension within the first 24 hours following cardiac bypass surgery is very common. It is noted is almost all of the patients after coronary bypass surgery. It occurs in 9% of the patients undergoing carotid endarterectomies(1). It can occur from a variety of causes ranging from transient unexplained hypotension to overt left ventricular failure from a massive myocardial infarction. So, hypotension could be a benign, routine passing symptom or it may signal the beginning of a saga in that patient's life. Consequently, hypotension deserves careful, thorough, and prompt consideration and treatment. If you remember the lessons from the ACLS, you will recall that human brain can only withstand four minutes of hypoxia before severe brain damage occurs. Even transient hypotension could lead to acute renal failure. That means, you have less than a few minutes to recognize the problem, identify the diagnosis, and institute appropriate treatment to preserver tissue perfusion, avoid brain, and or kidney damage. Let us look at some common causes of hypotension following cardiac surgery.

Transient hypotension

Transient hypotension occurs frequently after heart surgery. It may repeat itself 2 to 4 times during the first 24 hours. Generally, there is no identifiable cause. There is great deal of autonomic changes occurring with in the first 24 hours. Even though we do not routinely measure the catecholamine levels, yet we can see their effects in terms of heart rate and blood pressure. As the anesthetic effect wears off, the patient may begin to experience pain. A patient may not be able to tell us that he or she is hurting. However, their autonomic system would definitely respond to such stimuli and one of such effects could be hypotension. Similarly, the presence of E-T tube in the throat of a conscious patient could be another source of stimulation of the autonomic nervous system. Endotracheal suction could set of violent bouts of cough, changes in heart rate, and blood pressure.

This type of hypotension usually responds to volume expansion with normal saline. We also need to pay special attention to what the conscious patient may be feeling and address issues such as pain and sedation if necessary. The transient blood pressure problems generally subside after the first 24 to 36 hours. This type of hypotension may not be associated with significant changes in the oxygen level or EKG changes. If simple volume expansion fails to maintain adequate mean arterial pressure greater than 60 mm Hg, a small dose of dopamine infusion may stabilize the blood pressure. We generally start the dopamine at 5 mcg/kg/min which also helps to maintain renal perfusion. If these simple measures fail to raise the blood pressure then you better not take that coffee break. You need to look at other causes of hypotension.

Bleeding

Bleeding from the chest cavity is very common following cardiac surgery. Normally, the chest bleeding may range in amount from 50 to 100 ml per hour. However, when the bleeding from the chest tube exceeds 300 ml per hour it should raise the suspicion of an arterial bleeding

spot inside the chest cavity. Bleeding problems relating to thrombolytics and antiplatelet agents deserves special attention and they will be covered in detail at a later time. Chest bleeding could result from several causes. The amount of heparin received by the patient during cardiopulmonary bypass surgery may not be completely reversed. It is not uncommon to see prolonged PT and PTT's in patients coming out of the operating room. Extensive dissection involved in harvesting the internal mammary artery can be another source of widespread blood oozing. Occasionally, there may be arterial bleeding arising at the suture lines where the vein graft and the coronary arteries are attached. I have seen bleeding at a rate of 700 ml per hour resulting from one such situation. Infrequently, the bleeding may be so diffuse and relentless that it may force the surgeon to leave the chest cavity open with just a plastic sheet on top of it.

Usually, excessive bleeding (>300 ml/h) may signal a return trip to the operating room. If the surgeon can identify a bleeding spot, it can easily be fixed. However, if the bleeding is a result of the generalized oozing of the blood, the return trip to the operating room may not be beneficial. In fact, it may add extra burden to an already compromised cardiovascular system. Patients with open chest wounds have to be taken back to the operating room for chest closure when the bleeding problem is controlled.

Bleeding also can result form depletion of coagulation factors during cardiac surgery. Under these circumstances simple replacement of the packed red blood cells alone may not correct the problem. A thorough coagulation profile may aid in identifying problems such as low platelet count, defective platelets, or depletion of coagulation elements. Replacement of platelets or fresh frozen plasma could correct the coagulation deficiencies.

Bleeding from the gastrointestinal tract can be a rare source of hypotension. This complication may go undetected for a while unless the patient has a nasogastric tube. A third of these UGI bleeders have a history of peptic ulcer disease. Careful history must be taken with reference to any UGI bleeding in the past. These patients must be treated with H2 receptor blockers in the postoperative period. **Norton** et

al. did a retrospective study of UGI hemorrhage following CABG. Fifty-five of 10,573 patients (0.5%) suffered a major UGI hemorrhage (as defined by need for transfusion or presence of melaena or hematemesis associated with hypotension). Majority of these patients bled from duodenal ulceration (82%). Eight patients underwent surgery as initial therapy (20%). They concluded that endoscopy was safe in this patient group and those who underwent surgery had a surgical mortality similar to those without bleeding(2).

Cardiac Tamponade

Fortunately, cardiac tamponade is a rare cause of hypotension. This can result from accumulation of blood within the pericardial cavity. There is no difference in the incidence of cardiac Tamponade whether the pericardial sack is closed or left open. Most often, the surgeon loosely closes the pericardial layer. Hence, the majority of the symptoms related to tamponade are more likely related to the accumulation of blood around the heart in the mediastinal cavity, that progressively compromises the left ventricular filling.

Suddenly, when the chest tubes stop draining blood that should raise your suspicion. You grease your fingers and try to milk the last drop of blood from the chest tubes. You get fingers cramps but not a drop of blood.

Look for the clinical features of tamponade. Watch for pulses paradoxus on the monitor, elevated central venous pressure, dwindling urine output, and a drop in oxygen saturation level. You need a prompt diagnosis. At our institution, we have the benefit of anesthesiologists who are well trained in performing transesophageal echocardiography. In addition, they are available at bedside 24 hours a day if needed. A quick slip of TEE scope down the throat can establish the diagnosis in a few minutes.

You try volume expansion to no avail. The real challenge may be getting the surgeon to come at 2 O'clock in the morning. However, returning the patient back to the operating room and clearing all the clots from around the

heart is of paramount importance. Identifying and fixing the bleeding spots may also make a difference between day and night for that patient and to the surgeon also who has to explain to the family the next day on the new developments during the past night.

Cardiac tamponade is a mechanical problem. It is an easily correctable problem compared to left ventricular pump failure resulting from myocardial damage. Cardiac tamponade is a medical and surgical emergency. Hence, a clear knowledge of the clinical picture and high degree of suspicion of such a condition can save vital time and prevent target organ damage resulting from prolonged hypotension.

Sometimes, all the classic signs of tamponade may not be clearly evident at bedside. Neck vein distension may be difficult to evaluate in a patient who has central lines or a short neck. A good central venous pressure in excess of 16 to 18 cm of water may be an indication of Tamponade if associated with clear lung fields and a lack of pulmonary congestion. Chest X-ray may reveal widened mediastinum which is a common finding after cardiac surgery and it may not be helpful to pinpoint the diagnosis. The blood count could be reasonably within the normal range of 9-11G% immediately following cardiac surgery.

Access your patient's situation quickly. Check complete vital signs and listen to the patient's chest. Measure the urine output. See what has changed since the last time the patient was in stable condition. Review the lab tests such as CBC, EKG, ABG, and chest X-ray. Get an echocardiogram or TEE whatever it is available if you suspect cardiac tamponade. Do not send the order through the computer for an echocardiogram to be done first thing in the morning.

Bommer et al. evaluated the sensitivity of classic echocardiographic criteria in detecting cardiac tamponade in patients who had undergone cardiovascular surgery. Of 848 consecutive patients who underwent cardiovascular surgery, 14 patients were selected for the study if they had clinical or hemodynamic deterioration and had undergone an echocardiogram just before a successful pericardiocentesis or a surgical evacuation of pericardial blood or clot. In these patients classic echocardiographic criteria were seen

infrequently: chamber collapse of the right atrium (6 of 14 patients) and right ventricle (4 of 14); Doppler flow variation (2 of 5); and swinging heart (0 of 15), whereas increased pericardial separation (> or = 10 mm) was seen in all (14 of 14) the patients(3).

Left ventricular failure

Hypotension related to Left ventricular failure can be difficult to access and a challenge to treat. It will be covered in detail elsewhere.

Vasodilatory Hypotension

Systemic Inflammatory Response Syndrome (SIRS), is a well known complication following cardiac surgery. The incidence of this syndrome varies from 8% to 44% (4,13). This wide variation in the incidence may reflect different criteria used by different authors in defining this syndrome. It is characterized by peripheral vasodilatation and hypotension. It is associated with a breakdown of capillary membranes and accumulation of excess interstitial fluid. These patients also show increased oxygen extraction. Several mechanisms for the development of this syndrome have been hypothesized. One of these theories is that the ischemic injury in the gastrointestinal tract disturbs the gut barrier function and allows enteric bacterial endotoxins to pass into the circulation producing sepsis-like symptoms. Other theories relate to the release of cytokines associated with CPB (5). Some of these patients also have elevated levels of tumor necrosis factor, and interleukin-6 (13).

If not recognized and treated promptly, it can have adverse outcome. This vasodilatory shock is known be present when the mean arterial pressure is less than 70 mm Hg associated with high cardiac index [>2.5 L/m^2], low urine output, and low filling pressures and increased oxygen consumption (7) . Volume expansion and usual vasopressor may fail to restore normal blood pressure and urine output. It responds well to intravenous arginine vasopressin. Arginine vasopressin is a powerful vasoconstrictor. Argenziano et al. noted such hypotension in 8% of 145

people who underwent cardiopulmonary bypass. Hypotension was more frequent in patients who had LVEF of <35% (RR 9.1) and those who had been on ACE inhibitors (RR 11.9) in the preoperative period(8). These patients also had very low levels of arginine vasopressin. When these patients were treated with arginine vasopressin, it increased their mean arterial pressure and decreased the catecholamine pressor requirements.

On the other hand, arginine vasopressin is known to be elevated in patients with congestive heart failure and especially in those patients with massive myocardial infarction leading to congestive heart failure. Hence, if the hypotension is a manifestation of low cardiac output or acute perioperative myocardial infarction then administration of arginine vasopressin could lead to further vasoconstriction and worsening of symptoms.

Gomez et al from Brazil, reported a similar clinical presentation in sixteen patients that they called vasoplegic syndrome. The mean CPB time was 121 minutes, ranging from 80 to 210 minutes. Their patients presented with severe hypotension, tachycardia, normal or elevated cardiac output, low systemic vascular resistance and decreased filling pressures. The characteristics of vasoplegic syndrome were similar to those observed in septic shock, where the alterations were mediated by cytokines and tumor necrosis factor-alpha. Fluid administration alone was not capable of restoring hemodynamic parameters. Normal capillary filling at the extremities although oliguria and hypotension were observed. These patients failed to respond to high doses of vasoconstrictor drugs (norepinephrine) for blood pressure(9).

Vascular tone and volume alterations

When cardiac surgery patients come out of the operating room their body temperature is 35 degrees centigrade. So, they may have significant peripheral vasoconstriction. As the body temperature approaches normal, there is associated peripheral vasodilatation that can cause relative changes in the intravascular volume that can lead to hypotension. This is more evident in those

patients who are also on vasodilators for control of hypertension. If careful attention is not paid, the blood pressure could change from hypertension to hypotension in a very short period. Hence, it is important to pay attention to several factors working at the same time while controlling hypertension or hypotension.

Cardiac Arrhythmias

Cardiac tachyarrhythmias such as AF with rapid ventricular response or VT may result in sudden hypotension. Similarly, complete heart block with loss of synchronized atrial activity or severe bradycardia could also lead to hypotension. Prompt cardioversion may be needed in severe tachyarrhythmias if immediate pharmacological intervention fails to restore adequate blood pressure and regular cardiac rhythm. On the other hand, severe bradycardia and complete heart block must be treated with electrical pacing, preferably A-V sequential pacing.

Orthostatic Hypotension

It is defined as a decline of 20 mm Hg systolic blood pressure or 10 mm Hg diastolic blood pressure upon upright posture. Even though this may not be a problem in patients in the intensive care units where they are bed ridden, it may become apparent once the patients start ambulation.

Mechanism: When a person assumes erect posture from a supine position there is pooling of 500 to 700 ml of blood in the lower extremities primarily due to failure of automatic vasoconstriction in the leg vessels. This decrease in venous return in turn stimulates the carotid, aortic, and cardiac baroreceptors. This in turn activates the sympathetic outflow leading to increased heart rate and peripheral vasoconstriction. Occasionally, these reflexes may be blunted or abnormal in some patients.

Causes: Postural hypotension may be a preexisting condition in some elderly patients. It may be aggravated during the postoperative period due to poor oral intake, inadequate hydration, or vigorous diuresis. Some antihypertensive drugs in the presence of low intravascular

volume also can lead to postural hypotension. Rarely, it could be related to adrenal insufficiency.

Clinical Findings: Patients may experience weakness, dizziness, or fatigue especially during the early morning hours or immediately following meals.

Management: Blood pressure must be measured in supine and upright posture whenever a patient complains of weakness, dizziness, or fatigue. Diuretics and antihypertensive medications should be withheld until the condition is stabilized. If the orthostatic hypotension is a chronic preexisting condition, then administration of 1 to 2 grams of extra salt per day may help. Some patients may benefit from peripheral vasoconstrictors such as midodrine 10 mg 3 to 4 times a day. However this medicine has to be taken every four hours during daytime. Supine and standing blood pressure should be monitored regularly during treatment. Precaution must be taken to avoid midodrine during supine position to avoid hypertension. Ephedrine a sympathomimetic drug in doses of 25 mg 1 to 4 times daily also has been found to be useful. Ephedrine releases endogenous norepinephrine from its storage sites. Minerolocorticoids such as fludrocortisone 0.1 to 0.4 mg once daily has been found to be useful in ameliorating the symptoms of orthostatic hypotension. It helps to retain sodium.

RV dysfunction

Right ventricular dysfunction following cardiac surgery is a rare complication that can lead to hypotension. It is covered under in a separate heading. It is manifested by dilatation of the right ventricle, elevated central venous pressure, relatively clear lung fields and normal left ventricular function. These patients may also have low cardiac and urine output. Sometimes it may be related to acutely elevated pulmonary pressure(15). They respond to volume expansion with saline or colloids combined with vasopressor such as dopamine or norepinephrine.

Iatrogenic

During the hospital course following cardiac surgery, patients receive several drugs and blood products that can cause hypotension. Protamine given to reverse the effect of heparin is a well know agent to cause hypotension. Similarly, beta adrenergic agents administered for control of heart rate in AF can lead to hypotension. Amiodarone also has been associated with hypotension. Mair et al., reported on eighteen transfusion reactions that occurred in 16 patients. Sixteen of the reactions were hypotensive and occurred in 14 patients transfused with platelets through negatively charged bedside leukocyte reduction filters. All 14 patients had received angiotensin-converting enzyme (ACE) inhibitors prior to transfusion(10).

Other causes

Very rarely adrenal insufficiency has been associated with persistent hypotension. Simple measure of cortisol levels may uncover this problem that can be easily corrected with steroids(11, 12). Rivers et al. found adrenaline insufficiency in 8.7% of the adult patients, older than 55 years who underwent major surgery. All of these patients had serum cortisol levels of less than 20 mcg/dL. In the same population 24% of the patients had serum cortisol levels of less than 30 mcg/dL. Patients with adrenal insufficiency also has a significant increase in absolute eosinophil count. Their patients received hydrocortisone (100 mg IV q8 h.). The group that received hydrocortisone had a much better survival rate compared to those who did not receive hydrocortisone treatment ($p < 0.01$) (16).

Sepsis in the first two weeks following surgery could lead to hypotension and low cardiac output.

Treatment of hypotension

Treatment of hypotension is more than replacing volume or pumping vasopressors. It very much depends on the cause of hypotension. Hence, each hypotensive episode

must be evaluated individually and carefully while instituting prompt treatment.

Let us look at some common clinical situation and what kind of treatment would best address such situations.

If the patient has a normal LV function, normal oxygen saturation level, and normal urine output without significant bleeding problem, then a challenge of 500 to 1000 ml of normal saline may restore the blood pressure to acceptable levels.

If the patient has a vasodilatory hypotension as manifested by low CVP or PCW pressure, cardiac index or 2.5 L/min or more, low urine out put, normal oxygen saturation level then a trial of intravenous epinephrine or norepinephrine is in order. Some of these patients, especially those who have been on ACE inhibitors may have low levels of vasopressin(5). These patients should respond better to an infusion of arginine-vasopressin(5). It is important to recognize this condition early since these patients may not respond to norepinephrine as well as they would to arginine-vasopressin.

If bleeding is the primary cause of hypotension, then we not only need to replace the red blood cells but also attempt to correct the coagulation problems. Please refer to the chapter on bleeding for a complete discussion of this topic.

If hypotension is due to left vetricular pump failure it poses even a greater challenge. This condition is manifested by low blood pressure, increased PCW pressure in excess of 18 mm Hg, low cardiac index (2.0L/min/cm2), and increased systemic resistance. In addition, there may be low urine output, pre-renal azotemia, cold and clammy extremities. It is discussed in length in the 'left ventricular pump failure' chapter.

If hypotension is associated with pulmonary congestion, hypoxia, and adequate left ventricular function [ARDS], then consider adult respiratory disease, severe broncho-pneumonia, and atelectasis. These patients may have normal PCW pressure in the presence of elevated right heart pressures. In these patients, a cautious administration of colloids such a 5% albumin (250-500 ml)

or Hispan (500 ml) along with a small dose of a diuretic (furosemide 20-40 mg IV may help).

If the patient has mild hypoxia, low red blood cell count (Hg < 8.0 G/dl), and volume contraction then transfusion of packed red blood cells would not only correct the hypotension, but also improve the hypoxia by increasing the blood oxygen carrying capacity.

After you have addressed the underlying problem if the patient still remains hypotensive, then consider intravenous dopamine starting at a renal perfusion dose of 5 mcg/kg/min. Some patients may need epinephrine or norepinephrine to maintain adequate blood pressure.

The name of the game is act quickly and positively. If you are contemplating on putting an IABP in the morning, why not do it now? It's better to do it now so you can sleep better at night.

[Kuralay et al. discovered that those who underwent. posterior pericardiotomy (longitudinal incision was made parallel and posterior to the left phrenic nerve, extending from the left inferior pulmonary vein to the diaphragm, n=100) had lower incidence of AF (6% V. 34%) compared to those who had anterior pericardiotomy. Early and late pericardial effusion were 54% and 21%, respectively, in anterior pericardiotomy group. However, neither early nor late pericardial effusion were noted in the posterior pericardiotomy group (P =.00001). Delayed pericardial tamponade was also significantly lower in posterior pericardiotomy group I (0% vs 10%; P =.001). ( ) ps: move to hypotension.]

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Hypotension Treatment Choices

Fluids	Dosage
Normal Saline	500-1000 ml IV bolus
Packed red blood cells If the Hg is <8 G% or if patient has COPD	One to several units depending on bleeding. Consider auto-transfusion if the bleeding is profuse
Albumin 5%	250-500 ml IV
Hispan	250-500 ml IV
Fresh Frozen Plasma	Depending on Coagulation status
Platelets	10-20 units to correct platelet dysfunction and or count
Cryoprecipitate	
Aminocaproic Acid [Amicar]	Loading dose is 4—5 grams IV over one hour, followed by a continuous infusion at 1 gram/hour IV for eight hours. The maximum dose is 30 grams.
Dopamine	Start at 0.5 to 2.0 mcg/kg/min Renal perfusion dose 5 mcg/kg/min. Max: 30mcg/kg/min
Epinephrine	1 µg/min as a continuous IV infusion, titrated to desired hemodynamic response (usually 2—10 µg/min).
Phenylephrine alpha-adrenergic agonist [Neo-Synephrine]	Initial: 0.2 mg IV (range: 0.1—0.5 mg), q 10 to 15 min. Infusion: Initially, 100—180 µg/min. maintenance 40—60 µg/min.
Norepinephrine	0.5 to 1 µg/min as an IV infusion, titrated to a maintenance dose usually 2—4 µg/min. Max 8—30 µg/min.
Arginin-vasopressin	
Milrinone for Class III & IV CHF	IV loading dose of 50 µg/kg slowly followed by a continuous infusion at 0.375 to 0.75 µg/kg/min <48 hours
Metaraminol [Aramine] Adrenergic agonists	10—300 µg/min IV infusion
Intra aortic balloon pump [IABP]	Left ventricular failure

Petersen DA. **Managing hypotension after cardiac surgery: an algorithm for treatment.** Crit Care Nurse 2000 Apr;20(2):36-41, 43-6, 48-9

Memorial Hospital, Gulfport, Miss, USA.

Crit Care Nurse. 2000 Apr;20(2):36-41, 43-6, 48-9. Related Articles, Links

Managing hypotension after cardiac surgery: an algorithm for treatment.

Petersen DA.

Memorial Hospital, Gulfport, Miss, USA.

Publication Types:

- Review
- Review, Tutorial

AACN Clin Issues. 1997 Aug;8(3):303-18. Related Articles, Links

Hypotension.

Hravnak M, Boujoukos A.

University of Pittsburgh School of Nursing, Pennsylvania, USA.

Advanced practice nurses are responsible for diagnosing and treating patients with acute onset hypotension. The potential diagnostic hypotheses for hypotension are related to a wide variety of pathophysiologic processes. These processes are

represented by the acronym VINDICATE--Vascular (and cardiac), Inflammatory, Neoplastic, Degenerative, Intoxication/Iatrogenic, Congenital, Allergic/Autoimmune, Traumatic, Endocrine/Metabolic However, acute onset hypotension experienced by the adult patient in the hospital is likely to be caused by the vascular (and cardiac) processes of absolute hypovolemia, relative hypovolemia, and pump failure. Developing the differential diagnosis for acute onset hypotension involves making a series of clinical decisions in a stepwise manner. The clinician bases these decisions on information contained in a subjective and objective database and on recognizing patterns in the central findings. However, treatment of hypotension may be necessary before or during the diagnostic process, depending on the severity of the patient's symptoms.

Publication Types:

- Review
- Review, Tutorial

Anaesth Intensive Care. 2001 Dec;29(6):591-4. Related Articles, Links

Human serum albumin induced hypotension in the postoperative phase of cardiac surgery.

Howard G, Downward G, Bowie D.

Department of Intensive Care Medicine, Christchurch Hospital, New Zealand.

Hypotension associated with the rapid infusion of human serum albumin products was first recognised in Australasia in the early 1970s. An association with the angiotensin converting enzyme inhibitor class of drugs (ACE-I) followed, leading to a proposed mechanism involving bradykinin

generation through pre-kallikrein activator (PKA) presence in the infused fluid. The newer generation albumin products (Albumex) contain very low concentrations of PKA and are generally thought safe to use in most patient populations. Anecdotal reports of paradoxical hypotension with rapid infusion of 4% albumin in our department led to an audit of practice over three months. Four out of 36 patients (11%) who received 4% albumin intravenously experienced paradoxical hypotension. Three of these patients were taking ACE-I preoperatively (P=0.04). There was no observed hypotension associated with intravenous infusion of crystalloid fluid. We believe 4% albumin should be used with caution, particularly in those patients receiving ACE-I preoperatively.

ASAIO J. 2001 Nov-Dec;47(6):673-6. Related Articles, Links

Prostaglandin synthesis inhibitor improves hypotension during normothermic cardiopulmonary bypass.

Takewa Y, Seki T, Tatsumi E, Taenaka Y, Takano H.

Department of Artificial Organs, National Cardiovascular Center Research Institute, Suita, Osaka, Japan.

Hypotension is a major systemic side effect during cardiopu monary bypass (CPB), especially at normothermia. We previously reported that prostaglandin (PG) might play a substantial role in hypotension. The purpose of this study was to clarify whether a PG synthesis inhibitor (PGSI) could improve hypotension during CPB. Thirty-six patients undergoing cardiac surgery with normothermic CPB (35-37 degrees C) were divided into two groups: a PGSI group (n = 18), whose members wer given a PGSI before and during CPB, and a control group (n = 18). In both groups, perfusion flow was sufficient and pressure was maintained at above 45 mm Hg by infusion of metaraminol, a vasoconstrictor. The mean arterial pressure throughout CPB was

significantly higher in the PGSI group than in the control group (57 +/- 4 vs. 48 +/- 3 mm Hg, p < 0.01), whereas the dose of infused metaraminol was significantly lower in the PGSI group (13 +/- 7 vs. 21 +/- 6 mg, p < 0.01). The blood base excess was not significantly different (1.0 +/- 1.6 vs. 1.7 +/- 1.9 mmol/L, p = 0.28), and urine output was significantly higher in the PGSI group (503 +/- 179 vs. 354 +/- 112 ml/hr, p < 0.01). In conclusion, PGSI can improve hypotension during CPB and increase urine output without impairing peripheral circulation.