

# Systematic Approach on Postoperative Care of the Cardiac Surgical Patients

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**Summary:** With the medical advances and rising expectations among cardiac surgeons and patients, older and sicker patients now undergo ever more complex operations. However, fortunately, postoperative care of cardiac surgical patients also have shown important changes parallel to the surgical improvements.

Although, the events within the operating room are accepted as the main determinants for the faith of the patient, some patients, who are very ill upon arrival in the intensive care unit, may have a good long-term prognosis when postoperative care is meticulously provided. Conversely, patients who are doing well as they leave the operating room may be put at significant risk for complications by poor postoperative management.

In this paper, we draw an outline of systematic approach of intensive care of cardiac surgical patients and summarize a practical manual for physicians. By applying this approach effectively, it should be expected that, the clinician is able to recognize an impending disaster earlier, initiate the proper treatment timely, and increase patient's chances of survival. (*Anadolu Kardiyol Derg, 2003; 3: 156-161*)

**Key Words:** Cardiac surgery, postoperative care, surgical complication

## Introduction

Postoperative care of cardiac surgical patients have shown important changes parallel to the surgical improvements in recent years. These changes have resulted in intensive care unit (ICU) containing a greater proportion of older, sicker patients who have undergone more major surgery.

The real challenge, for all physicians who deal with patients who may become critically ill, is to develop a system of practice that will allow the early identification and correction of complications before it happens.

In the US, more than 300.000 cases involving cardiopulmonary bypass (CPB) are performed every year and all of these patients require postoperative intensive care (1). The main determining factors of success of a cardiac operation are the events that occur within the operating room. However, some patients, who are very ill upon arrival in the ICU, may have

a good long-term prognosis when postoperative care is meticulously provided. Conversely, patients who are doing well as they leave the operating room may be put at significant risk for complications by poor postoperative management. It is crucial in all circumstances, an expert interdisciplinary postoperative approach should be provided 24 hours a day in order to ensure a successful outcome.

The postoperative cardiac surgical patient is in a special biologic situation due to the effects of CPB. Early after the operation there is a whole body inflammatory response with leaky capillaries and all its possible attendant problems. The physician must be keenly aware of this basic premise in caring for these patients.

There are several key points, as outlined by Cogle that should be remembered (2):

- a- Postoperative course is determined by intra-operative events.
- b- Management in the early postoperative period is determined by the multisystem effects of CPB.
- c- Managing cardiovascular function is key to recovery in other systems.
- d- Respiratory care is directed at restoring lung volume and minimizing lung water.

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e- Preservation of renal function is essential in minimizing postoperative morbidity.

f- Coordination of care is required to facilitate immediate and timely interventions and to incorporate input from surgeons, anesthesiologists, cardiologists, cardiac intensivists, and consultants.

The type of surgery plays an important role in determining postoperative hemodynamic management and outcome. Valve replacement patients are more at risk for complications than coronary bypass patients. Those with mitral insufficiency seem to be at the highest risk for postoperative complications, followed by those with aortic stenosis or insufficiency, and finally, mitral stenosis (3). The goals in the immediate postoperative period include: monitoring and assurance of hemodynamic and respiratory stability, restoration of normal body temperature, monitoring of fluid, electrolytes and blood loss from mediastinal and pleural chest tubes (4). It is important to intervene in a timely fashion to manage agitation and shivering as the patient awakens from anesthesia. There are several important problems that must be addressed rapidly to prevent complications (5):

- 1- Inadequate perfusion pressures
- 2- Low cardiac output
- 3- Dysrhythmias
- 4- Ischemia
- 5- Excessive bleeding
- 6- Cardiac tamponade

The approach to these patients is best directed by dividing their problems into the systems:

## I- Cardiovascular System

For the routine uncomplicated patient with relatively normal preoperative function. The tendency is toward a slow increase in systemic vascular resistance (SVR) in the early postoperative period. As warming occurs the patient tends to vasodilate, and volume loading and the use of a vasopressor may be needed. It is important not to administer any more volume than necessary in this period. With increased preload and decreased afterload, cardiac output (CO) may reach or surpass preoperative values.

Intravascular overload secondary to overaggressive fluid resuscitation has always been a primary concern with large-volume fluid administration. In addition to intravascular overload, the myocardium itself may become edematous and lead to impaired contractility and compliance.

**Low output syndrome (LOS):** LOS is defined as a cardiac index (CI)  $<2.2$  L/min/m<sup>2</sup>. It is associated with a high incidence of respiratory failure, renal fa-

ilure, disseminated intravascular coagulation (DIC), central nervous dysfunction, gastrointestinal (GI) bleeding and death (6). Acute left ventricular dysfunction may occur in the early post-operative period or later during periods of stress such as weaning from mechanical ventilation. One must remember that a thermodilution CI of 2.2 L/min/m<sup>2</sup> must be interpreted with respect to the patients age, preoperative status, and end-organ function as manifest by mental function, acid-base status, lactate production, mixed venous oxygen saturation, urine output and signs of peripheral hypoperfusion. If all systems are functioning normally and all other values are within acceptable parameters, one must not get "locked" in to a thermodilution CO value. Conversely, when it is clear that a greater CO is needed, preload and afterload must be optimized first, then the addition of an inotrope such as dopamine, dobutamine, amrinone or epinephrine is warranted. However, if the patient is bradycardic increasing the heart rate with a pacemaker maybe effective (7).

Unlike congestive heart failure, the perioperative LOS often does not respond to vasodilators alone. In addition to this, the need for inotropic support after CPB does not significantly reduce outcome expectations, as it does in medical congestive heart failure (8).

**Arrhythmias:** Arrhythmias are frequent in the perioperative period and there appears to be two peaks in their incidence (9). The first occurs in the operating room usually during induction of anesthesia or during weaning from CPB. The second peak occurs between the 2nd and 5th postoperative day. The mechanism underlying postoperative arrhythmias, is not completely understood.

Bradycardias suggest chronotropic incompetence related to drugs, structural abnormalities, or reflex-mediated responses such as seen in severe hemorrhagic shock and acute inferior wall myocardial infarction. Tachycardias may reflect underlying cardiac disease, pharmacologic activity, or physiologic stress and the need for compensatory mechanisms to maintain CO and organ perfusion (10).

Supraventricular arrhythmias (SVA) occur in 20-50% of patients after cardiac surgery (1). Atrial fibrillation is common and is usually easily controlled with digoxin. Therapy with beta-adrenergic blocking drugs has been shown to be effective in both preventing and treating SVA.

Ventricular ectopy is increased in postoperative cardiac surgery patients and some advocate lidocaine and magnesium to suppress these as prophylaxis against more serious ventricular arrhythmias. The benefits of prophylactic anti-arrhythmia therapy postoperatively

have never been adequately addressed in a randomized clinical trial. If ventricular tachycardia occurs, arrhythmic treatment with lidocaine is warranted. However, this treatment should not last longer than 24 hours, at which time it should be discontinued and the patient observed for any further arrhythmias (11).

**Cardiac tamponade:** Signs of tamponade may be very different than in the patient with chest trauma or constrictive or restrictive pericardial disease. The diagnosis should always be suspected when the patient's hemodynamic status worsens and does not respond to interventions, especially when accompanied by elevation and equalization of end diastolic pressures or marked decrease in chest tube output.

**Postoperative hypertension:** Hypertension occurs in up to 40% of patients and may be part of a hyperdynamic state (12). The possible etiology is multifactorial:

- Hypoxia
- Hypercapnia
- Increased sympathetic tone
- Emergency from anesthesia
- Surgical manipulation of the great vessels

After assuring a normal arterial blood gases, in most cases treatment is aimed at reducing the SVR (13). Sodium nitroprusside will lower blood pressure by causing a decrease in SVR as well as causing an increase in heart rate and CO. In contrast, labetalol, a combined alpha and beta blocker, causes a decrease in blood pressure primarily by decreasing CO and heart rate with little change in SVR (14). Hydralazine is a good alternative for hypertensive patients with bradycardia, as it does not effect the heart rate. Smooth blood pressure control should be achieved by manipulating CO, afterload and preload.

## II- Respiratory System

The provision of adequate oxygenation and ventilation while recovering from anesthesia and surgery are primary goals. Secondary goals are avoidance of barotrauma, volutrauma and infection. Once the patient is awake, responsive and hemodynamically stable, weaning and extubation are usually accomplished. The most common pulmonary abnormality after cardiac surgery is loss of lung volume with subsegmental, segmental or lobar atelectasis. Expected changes in pulmonary function after a median sternotomy and CPB include a decrease in vital capacity by approximately 50%, which nadirs on the second to third post-operative day (15). There is also an accompanying decrease in functional residual capacity. Chest X-Ray evidence of atelectasis is present in 98%

of patients and a significant number of patients have pleural effusions (left>right). Internal mammary artery grafting is accompanied by greater pulmonary impairment than with saphenous vein grafts alone.

Extubation can be accompanied frequently within 4-6 hours of arrival in the ICU if the patient meets the criteria along with hemodynamic stability and an alert mental status (16). If the patient is more complex or has had >100 minutes of CPB, it is probably prudent not to extubate early (17).

### Factors predisposing to prolonged ventilation:

- Re-exploration for bleeding or tamponade
- Emergency procedure
- Left ventricular dysfunction
- Renal failure
- Fluid overload
- Neurological injury (stroke, encephalopathy)
- Infection (sepsis, pneumonia, mediastinitis, etc.)
- Malnutrition
- Bronchospasm
- Acquired respiratory distress syndrome (ARDS)
- Systemic inflammatory response syndrome/multiorgan dysfunction syndrome
- Phrenic nerve injury
- Advanced age

## III- Gastrointestinal System

Acute dysfunction of the GI subsystem is uncommon after cardiac surgery occurring in only about 1% of patients (18). However, important dysfunction is followed by death during hospitalized period in more than 50% of patients. The following problems can occur after an open heart operation (19):

- Postoperative ileus
- Upper GI bleeding
- Pancreatitis
- Cholecystitis
- Hyperbilirubinemia
- Bowel perforations and infarcts

Risk factors:

- CPB time
- Concurrent renal failure
- Respiratory failure requiring prolonged ventilatory support
- Previous history

Jaundice occurs in up to 20% of patients with bilirubin levels approximately 3 mg/dL. Moderate or severe jaundice, with more than 6 mg/dL bilirubin, occurs only in about 5% of patients (20, 21).

Jaundice maybe related to:

- Severity of preoperative right atrial hypertension

- Hypoxia during operation
- Early postoperative hypotension
- Multiple blood transfusions
- CPB time

**Infection:** Wound infections, sepsis, pneumonia, mediastinitis, urinary tract infections all occur as complications after cardiac surgery. The specific organisms seen are influenced by the antibiotic prophylaxis chosen. Treatment should be guided by culture and sensitivity reports and be organism specific. However, most institutions use either cefazolin or vancomycin, and therefore most superinfections are frequently with resistant organisms such as *Pseudomonas aeruginosa*, *Klebsiella*, *Serratia*. Sternal wound infections occur in approximately 1% of patients (22). Predisposing factors for wound infection following cardiac surgery are:

- Diabetes mellitus
- LOS
- Use of bilateral Internal Mammary Artery
- Reoperation for bleeding or tamponade
- Prolonged intubation

Sepsis is the most common cause of death in the surgical ICU's. Mortality ranges from 20% to 60% (23). Therefore, Gram-negative organisms are responsible for 50-80% of all cases of septic shock, while 6-24% of cases result from gram-positive organisms.

The source of bacteremia leading to sepsis includes the urinary, respiratory and GI tracts. However, in about 30% of patients, the source is unknown.

**Bleeding:** Increased blood loss following CPB maybe either "surgical" or "non surgical". Surgical bleeding is characterized as blood loss from a specific anatomic site. Mediastinal blood loss of >300 ml in the first hour, >250 ml in the second, and >150 ml thereafter has been correlated with surgically correctable bleeding (24). When bleeding is suspected to be surgical, the patient should be re-explored and bleeding controlled. Blood should not be allowed to collect within the mediastinum. Because it is likely to induce a local and systemic fibrinolytic state which can cause a vicious cycle of diffuse non surgical bleeding. Non surgical blood loss during the first four hours after surgery is directly related to the duration of CPB and the lowest systemic temperature reached (25). Adequate preoperative hematologic assessment, meticulous surgical technique and efficient prompt re-warming are imperative in the reduction of postoperative blood loss.

Persistent bleeding after cardiac surgery is one of the most common problems in the early postoperative period. The causes of postoperative bleeding are multiple and include (26):

- Heparin rebound
- Acquired, qualitative and quantitative platelet defects due to CPB and drugs
- Dilutional thrombocytopenia and factor deficiency
- Local and systemic fibrinolysis
- DIC
- Pre-existing coagulopathies

Interestingly, in our recent study, we have shown that the patients underwent open heart surgery during summer months had a higher incidence of bleeding (27). Therefore, it is also possible that clotting system might show some types of seasonal fluctuations.

The use of simple laboratory tests generally can determine which particular cause is principally responsible for the bleeding problem. This should allow for specific therapies to be administered. However, one should keep in mind that bleeding as a consequence of one etiology could quickly lead to impairment in other components of the coagulation cascade (28).

Adequate reversal of heparin should be ensured, especially if heparin rebound is suspected, by monitoring ACT, PTT or both, and administering additional protamine as indicated. Platelet consumption and dilution as a consequence of CPB maybe a common cause of bleeding. Therefore, if the patient continues to bleed significantly after adequate rewarming, fresh platelet transfusions are to be administered. Antiplatelet therapy, such as aspirin, is common in antiangiinal regimens that many patients' platelets are ineffective even with a normal preoperative platelet count. Continued prolongation of the PT and PTT should prompt infusion of fresh frozen plasma. Low levels of fibrinogen should be corrected with cryoprecipitate. Generally, blood transfusion is considered when hemoglobin is less than 8 g/dL, and hematocrit is less than 24% (23). However, transfusion of red blood cells should also be guided by rate of blood loss, underlying medical problems in the patient, and by the indicators of oxygen delivery. The re-infusion of up to 1000 ml of mediastinal blood shed through chest tubes postoperatively (auto re-transfusion system) has been shown to be safe and effective.

Pharmacologic interventions to control diffuse non surgical bleeding should include the use of protamine as described above and possibly  $\epsilon$ -aminocaproic acid, an inhibitor of fibrinolysis. Tranexamic acid is an isomer of  $\epsilon$ -aminocaproic acid with 7-10 times its inhibitory activity. Desmopressin acetate (DDAVP) has also been used to help attenuate bleeding, especially in patients with renal insufficiency. Aprotinin, a protease inhibitor may have a role when administered in high doses before and during CPB.

## IV- Renal System

Oliguric renal failure (RF), a very serious problem, has been reported to occur in 0.7-4.3% of patients (29). The incidence of RF requiring dialysis rises when postoperative creatinine is more than 1.7 mg/dL.

The most accurate simple test to discriminate established acute RF from other causes of oliguria is the fractional excretion of sodium (30). Urinary sodium, if low (<20mmol/L), will suggest active tubular conservation of sodium and further prompt circulatory therapy. Otherwise, following the serum urea and creatinine concentrations will adequately document the progress of renal function.

Mortality rates for RF ranged up to 90% in older reports (31), although, some evidences now indicates that early dialysis may reduce this rate significantly (32). There has been a trend toward early application of continuous arteriovenous hemofiltration in an attempt to remove excess fluid, thereby facilitating extubation and decreasing the incidence of respiratory failure (33).

Risk factors:

- Preoperative elevation of creatinine
- LOS
- Hypotension
- Advanced age
- Need for postoperative circulatory support
- Multiple blood and blood product transfusions
- CPB time

When RF occurs it generally follows three patterns (34):

**1- Abbreviated RF:** Occurs after an isolated insult at the time of surgery. Creatinine peaks around day four and then recovers.

**2- Overt RF:** The acute insult is accompanied by prolonged circulatory failure. Recovers in 2-3 weeks.

**3- Protracted RF:** Same as the previous one. But, as recovery occurs a second insult (sepsis, massive gastrointestinal bleeding, acute myocardial infarction, etc) ensues and permanent RF may occur. This is usually accompanied by ARDS or multiorgan dysfunction syndrome.

## V- Neuropsychiatric System

Cerebral complications of cardiac surgery are being recognized with increasing frequency. A subgroup of neuropsychologic complications, consisting of changes in memory, concentration and visual motor skills are directly attributable to CPB (35, 36). Transient postoperative delirium is seen in approximately 7% of patients on postoperative day 1, but either re-

solves spontaneously or after appropriate treatment by day 6 (37). Embolism and hypoperfusion have been implicated since the earliest days of cardiac surgery to be etiologic factors for strokes (38). New techniques to evaluate microemboli (Doppler detection, fluorescent retinal angiography, and conventional and diffusion magnetic resonance imaging) offer promise in the evaluation of these complications. The use of intraoperative ultrasound to detect atheromatous changes in the ascending aorta may help improve neurologic outcome.

Risk factors (39):

- Emergency procedure
- Severe left ventricular dysfunction
- Advanced age
- Peripheral vascular disease
- Intraaortic balloon pump
- CPB time > 142 minutes
- Valve procedure or ventriculotomy
- Carotid bruits

In cardiac surgical intensive care, early recognition of an impending disaster and initiation of treatment increase patient's chances of survival, and at the very least prevent further complications. This can be ensured by systematic approach. The body consists of different systems and all of them integrate and interact with each other. If there is something wrong with one component, one should expect some problems in the other systems.

The scope of cardiac surgical critical care includes prediction and prevention of problems as well as investigation and intervention. However, preventing deterioration is more effective than attempting salvage at a later stage. In addition, prompt simple actions can save lives and prevent complications. Therefore, the best critical care is simple and preventive. Late, heroic interventions generally are less successful.

## References

1. Kirklin JW, Barratt-Boyes BG: Cardiac Surgery. 2nd ed. New York: Churchill Livingstone; 1993. p.195-247.
2. Coyle JP. Sedation, pain relief, and neuromuscular blockade in the postoperative cardiac surgical patient. *Semin Thorac Cardiovasc Surg* 1991; 3:81-7.
3. Braunwald E. Valvular heart disease. In: Braunwald E, editor. *Heart Disease: A Textbook of Cardiovascular Medicine*. 4th ed. Philadelphia:WB Saunders; 1992. p.1007-77.
4. Weeks KR, Chatterjee K, Block S, Matloff JM, Swan HJ. Bedside hemodynamic monitoring. Its value in the diagnosis of tamponade complicating cardiac surgery. *J Thorac Cardiovasc Surg* 1976; 71:250-2.
5. Shoemaker WC. Shock states: pathophysiology, monitoring, outcome prediction, and therapy. In: Schoema-

- ker WC, Ayres S, Grenvik A, Holbrook PR, Thompson WL, editors. Textbook of Critical Care. 2nd ed. Philadelphia:WB Saunders; 1989. p.977-93.
6. Kumon K, Tanaka K, Hirata T, Naito Y, Fujita T. Organ failures due to low cardiac output syndrome following open heart surgery. *Jpn Circ J* 1986; 50:329-35.
  7. Urzua J, Zurick AM, Starr NJ, Cosgrove DM, Yared JP, Estafanous FG. Enhanced cardiac performance following cardiopulmonary bypass. *J Cardiovasc Surg* 1985; 26:53-8.
  8. Zaloga GP, Prielipp RC, Butterworth JF, Royster RL. Pharmacologic cardiovascular support. *Crit Care Clin* 1993; 9:335-62.
  9. Estafanous FG, Urzua J, Yared JP, Zurick AM, Loop FD, Tarazi RC. Pattern of hemodynamic alterations during coronary artery operations. *J Thorac Cardiovasc Surg* 1984; 87:175-82.
  10. Secher N, Sander-Jensen K, Werner C, et al. Bradycardia during severe but reversible hypovolemic shock in man. *Circ Shock* 1984; 14:267-74.
  11. England MR, Gordon G, Salem M, Chernow B. Magnesium administration and dysrhythmias after cardiac surgery. A placebo-controlled, double-blind, randomized trial. *JAMA* 1992; 268:2395-402.
  12. Estafanous FG, Tarazi RC. Systemic arterial hypertension associated with cardiac surgery. *Am J Cardiol* 1980; 46:685-94.
  13. Fouad FM, Estafanous FG, Bravo EL, Iyer KA, Maydak JH, Tarazi RC. Possible role of cardiopulmonary reflex in postcoronary bypass hypertension. *Am J Cardiol* 1979; 44:866-72.
  14. Cruise CJ, Skrobik Y, Webster RE, Marquez-Julio A, David TE. Intravenous labetalol versus sodium nitroprusside for treatment of hypertension postcoronary bypass surgery. *Anesthesiology* 1989; 71:835-9.
  15. Berrizbetia LD, Tessler S, Jacobowitz IJ, Kaplan P, Budzilowicz L, Cunningham JN. Effect of sternotomy and coronary bypass surgery on postoperative pulmonary mechanics. Comparison of internal mammary and saphenous vein bypass grafts. *Chest* 1989; 96:873-6.
  16. Matthay MA, Wiener-Kronish JP. Respiratory management after cardiac surgery. *Chest* 1989; 95:424-34.
  17. Lemaire F, Teboul JL, Cinotti L, et al. Acute left ventricular dysfunction during unsuccessful weaning from mechanical ventilation. *Anesthesiology* 1988; 69:171-90.
  18. Hanks JB, Curtis SE, Hanks BB, Andersen DK, Cox JL, Jones RS. Gastrointestinal complications after cardiopulmonary bypass. *Surgery* 1982; 92:394-400.
  19. Leitman IM, Paull DE, Barie PS, Isom OW, Shires GT. Intraabdominal complications of cardiopulmonary bypass operations. *Surg Gynecol Obstet* 1987; 165:251-4.
  20. Chu C, Chang C, Liaw Y, Hsieh MJ. Jaundice after open heart surgery: A prospective study. *Thorax* 1984; 39:52-6.
  21. Collins JD, Ferner R, Murray A, et al. Incidence and prognostic importance of jaundice after cardiopulmonary bypass surgery. *Lancet* 1983; 1(8334):1119-23.
  22. Grossi EA, Culliford AT, Krieger KH, et al. A survey of 77 major infectious complications of median sternotomy: A review of 7949 consecutive operative procedures. *Ann Thorac Surg* 1985; 40:214-23.
  23. Jakowatz JG. Clinical care of the surgical patient. In: Wilson SE, editor. *Surgery*. Laguna Hills:Current Clinical Strategies Publishing; 2000. p.14-27.
  24. Michelson EL, Torosian M, Morganroth J, MacVaugh H 3rd. Early recognition of surgically correctable causes of excessive mediastinal bleeding after coronary artery bypass graft surgery. *Am J Surg* 1980; 139:313-7.
  25. Bick RL. Hemostasis defects associated with cardiac surgery, prosthetic devices, and other extracorporeal circuits. *Semin Thromb Hemost* 1985; 11:249-80.
  26. Harker LA, Malpass TW, Branson HE, et al. Mechanism of abnormal bleeding in patients undergoing cardiopulmonary bypass: acquired transient platelet dysfunction associated with selective alpha granule release. *Blood* 1980; 56:824-34.
  27. Konuralp C, Ketenci B, Özyay, B., et al. Effects of seasonal variations on coronary artery surgery. *Heart Surg Forum* 2002; 5:388-92.
  28. Wasnick JD: *Handbook of Cardiac Anesthesia and Perioperative Care*. Newton: Butterworth-Heinemann; 1998. p.318.
  29. Bhat JG, Gluck MC, Lowenstein J, Baldwin DS. Renal failure after open heart surgery. *Ann Int Med* 1976; 84:677-82.
  30. Ramsay G. Renal failure. In: Anderson ID, editor. *Care of the Critically Ill Surgical Patient*. London: Arnold Publ; 1999. p.86-95.
  31. Hilberman M, Myers BD, Carrie BJ, Derby G, Jamison RL, Stinson EB. Acute renal failure following cardiac surgery. *J Thorac Cardiovasc Surg* 1979; 77:880-8.
  32. Koning HM, Koning AJ, Leusink JA. Serious acute renal failure following open heart surgery. *Thorac Cardiovasc Surg* 1985; 33:283-7.
  33. Coraim FJ, Coraim HP, Ebermann R, Stelwag FM. Acute respiratory failure after cardiac surgery: Clinical experience with the application of continuous arteriovenous hemofiltration. *Crit Care Med* 1986; 14:714-8.
  34. Myers BD, Moran SM. Hemodynamically mediated acute renal failure. *N Engl J Med* 1987; 314:97-105.
  35. Breuer AC, Furlan AJ, Hanson MR, et al. Central nervous system complications of coronary artery bypass graft surgery: Prospective analysis of 421 patients. *Stroke* 1983; 14:682-7.
  36. Murkin JM. The role of CPB management in neuro behavioral outcomes after cardiac surgery. *Ann Thorac Surg*. 1995; 159:1308-11.
  37. Calabrese JR, Skwerer RG, Gullledge AD, et al. Incidence of postoperative delirium following myocardial revascularization: A prospective study. *Clev Clin J Med* 1987; 54:29-32.
  38. Reed GL 3rd, Singer DE, Picard EH, De Sanctis RW. Stroke following coronary artery bypass surgery. A case-control estimate of the risk from carotid bruits. *N Engl J Med* 1988; 319:1246-50.
  39. Slogoff S, Girgis KZ, Keats AS. Etiologic factors in neuropsychiatric complications associated with cardiopulmonary bypass. *Anesth Analg* 1982; 61:903-11.