
The Role of Colloid Osmotic Pressure in Post Bypass Pulmonary Dysfunction

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Abstract

Determination of colloid osmotic pressure (COP), arterial blood gases (ABG) and weight were made in 25 consecutive patients undergoing cardiopulmonary bypass (CPB) with hemodilution. COP measurements were made prior to CPB, at 30 minute intervals during perfusion, and at 6, 12, 24 and 48 hours following CPB. Arterial blood gases were measured at 6, 12, 24 and 48 hours post-operatively. COP decreased abruptly with the initiation of bypass from 25.4 ± 3.4 mmHg to 16.8 ± 4.6 mmHg ($p < 0.05$) and remained at this level throughout the duration of bypass. All COPs returned to baseline levels, 23.6 ± 4.1 mmHg, 48 hours after surgery. Maximum decrease in COP was compared to the increase in calculated alveolar-arterial O_2 gradient at 6, 12, 24 and 48 hours post-operative, and no significant correlation could be found. Similarly, the decrease in COP did not correlate with the weight gain present at 24 and 48 hours. COP is significantly altered by CPB and undoubtedly results in changes in fluid compartment dynamics. However, changes in COP do not seem to play a role in ventilatory problems following CPB in the routine patient.

Introduction

Respiratory problems remain the most significant cause of morbidity following cardiopulmonary bypass.¹ Wet lung or mild pulmonary edema is the most common clinical problem following perfusion.

When lung biopsies are taken after shock, trauma, or cardiopulmonary bypass, the morphologic changes are similar and consist of interstitial edema, perivascular hemorrhage, and miliary atelectasis.¹ Several mechanisms of lung injury have been postulated leading to this common histologic presentation.² Hypoxia of the lung parenchyma may be one of the factors contributing to this lesion. Decreased pulmonary blood flow during cardiopulmonary bypass may result in precipitation products produced by extracorporeal circulation, since circulation of blood through a bubble oxygenator results in protein denaturation, the release of free lipids and red blood cell agglutination.¹

Hemodilution and decreased plasma oncotic pressure resulting from non-blood priming (physiological saline, etc.) may contribute to the formation of interstitial pulmonary edema. Autopsy studies confirm the presence of pulmonary edema despite only small increases in pulmonary extravascular water content. Pulmonary edema occurs when there is an imbalance of pressure in the following equation.^{3,4}

$$V = Kt(P_c - P_{IF}) - (\pi_p - \pi_{IF})$$

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Presented at AmSECT's 20th International Conference. Hollywood, Fla., April 27, 1982.

This equation defines the transport of fluid across capillaries, where V is the rate of liquid movement; K_t is the capillary filtration coefficient; P_c is the hydrostatic pressures in the capillary; P_{IF} the hydrostatic pressures in the interstitial fluids; π_p the plasma oncotic pressure; π_{IF} the interstitial fluid oncotic pressure.

The predominant force which filters water out of circulation to the surrounding tissue (interstitial space) is blood pressure. The predominant force which reabsorbs this water into the circulation (intravascular) is plasma colloid oncotic pressure. It is the gradient of intravascular and interstitial pressures that dictate the movement and balance of fluid exchange. It has been shown that in the normal individual colloid oncotic pressure is rarely the cause of pulmonary edema. However, marked decreases in colloid oncotic pressure have precipitated pulmonary edema.¹ The present study was designed to determine the role of altered colloid oncotic pressure in the development of post-cardiopulmonary bypass respiratory insufficiency.

Materials and Methods

Twenty-five consecutive male patients (mean age 58.3 ± 7.1 S.D. years) who underwent coronary artery bypass surgery were studied. Perfusion employed a bubble oxygenator, a two-roller pump, and crystalloid prime. Each patient was perfused with the crystalloid prime, Plasmalyte 148 (composition per liter: 140 mEq of sodium ion, 5 mEq of potassium ion, 3 mEq of magnesium ion, 98 mEq of chloride ion, 27 mEq of acetate ion and 23 mEq of gluconate ion), 25 grams mannitol, 44.6 mEq of sodium bicarbonate and 75 units of beef lung heparin per kg body weight. The volume of Plasmalyte needed to produce a Hct of 25% was determined from each patient's weight and red cell volume according to the following equation:

$$\frac{BV \times \text{Hct} (\%) }{25\%} - BV = \text{Dilution Volume (L)}$$

where BV = blood volume (estimated to be 75 ml per kg body weight), supplemental blood transfusions were used only when the hematocrit fell below 25 percent. Transfusion, arterial line and cardiotomy suction filters were used in all cases.

Blood samples were drawn according to the following protocol: intraperfusion, 30, 60, 90 min-

utes and 6, 12, 24 and 48 hours post-perfusion. The blood gas determinations were made by Instrumentation Laboratory 813 blood gas analyzers.^a Plasma for COP determinations was collected and stored at 4°C and assayed in batch to minimize intra-assay variability. COPs were measured by an Instrumentation Laboratory Model 186 Weil Oncometer.^b Data were analyzed using the Student's t test with $P \leq 0.05$ accepted as significant.

Results

The COP dropped significantly from 25.4 ± 3.4 (SD) to 16.8 ± 4.6 (SD) mmHg ($p \leq 0.05$) following hemodilution as shown (Figure 1). COP started to rise in a linear manner during cardiopulmonary bypass and returned to normal ranges within 48 hours after surgery.⁶ The average weight gain at 24 hours after surgery was 6.41 kg, but at 48 hours the net mean weight gain was down to 3.95 kg.

The mean alveolar arterial oxygen difference ((A-a)DO₂) did not show significant improvement from post-perfusion levels in the first 48 hours post-perfusion (Figure 2). Although (A-a)DO₂ was significantly elevated in the post-perfusion period, this elevation did not correlate with the changes in COP (Figure 3).

Discussion

The patient undergoing cardiopulmonary bypass, unlike the seriously ill patient, has a very sudden drop in colloid oncotic pressure.^{2,5} This sudden pressure drop occurs within seconds or minutes whereas the pressure drop that occurs during the course of acute illness usually is observed within hours or days.⁷ When the colloid oncotic pressure drops to 14 mmHg, the prognosis for the critically ill patient is less than 50 percent survival and 60 percent develop pulmonary edema with mortality of 55 percent.⁸⁻¹⁰ Data from the present study demonstrates a rapid drop in COP and a return to normal levels 48 hours after coronary artery bypass surgery. In addition, we noted that elevated alveolar arterial oxygen differences did not improve over a period of 48 hours following perfusion,

^a Instrumentation Laboratory Inc., Lexington, Massachusetts 02173.

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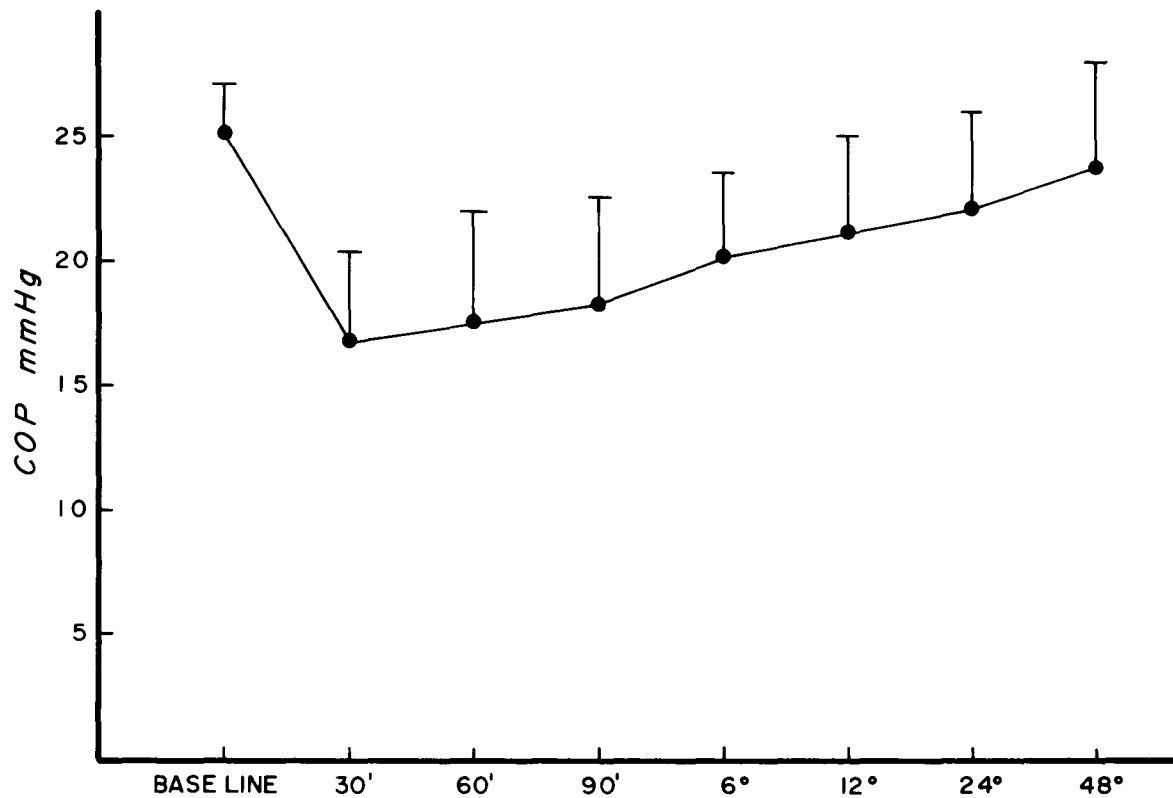


FIGURE 1. Changes in colloid oncotic pressure (COP) before (baseline), during (90 minutes) and after (48 hours) perfusion. Each point represents mean value \pm S.D. for 25 patients undergoing coronary artery bypass surgery.

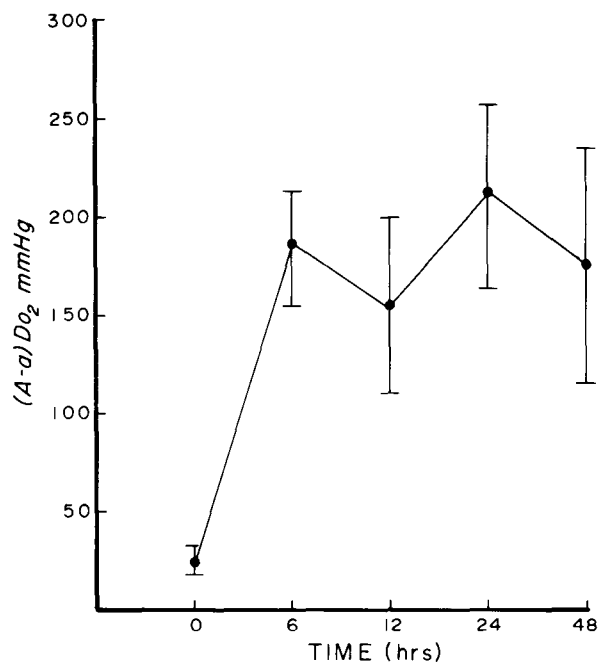


FIGURE 2. Changes in the alveolar arterial tension differences (A-a)DO₂ during 48 hours following perfusion. Each point represents mean value \pm S.D. for 25 patients undergoing for coronary artery bypass surgery.

despite an almost linear rise in oncotic pressure during this same time period. The presence of edema, as reflected in patient weight gain, was evident and did improve somewhat between 24 and 48 hours post bypass. Mills¹¹ has reported that most patients lose all extracellular water and return to pre-surgery weight levels five days following bypass.

Combined measurements of pulmonary arterial wedge pressure (PAW) and COP have demonstrated that pulmonary edema developed rarely in those patients in whom the colloid oncotic pressure-pulmonary artery wedge gradient was greater than 6 mmHg.^{6,7} When pulmonary artery wedge pressures were measured in critically ill patients, it was found that pulmonary edema was present at low oncotic pressure at a lower pulmonary artery wedge pressure than in patients with normal COP.^{8,12} Clinically, low colloid oncotic pressure has been implicated as an important factor in the development of systemic and pulmonary edema in the critically ill patient.¹³

Although the perfusion times in this study were

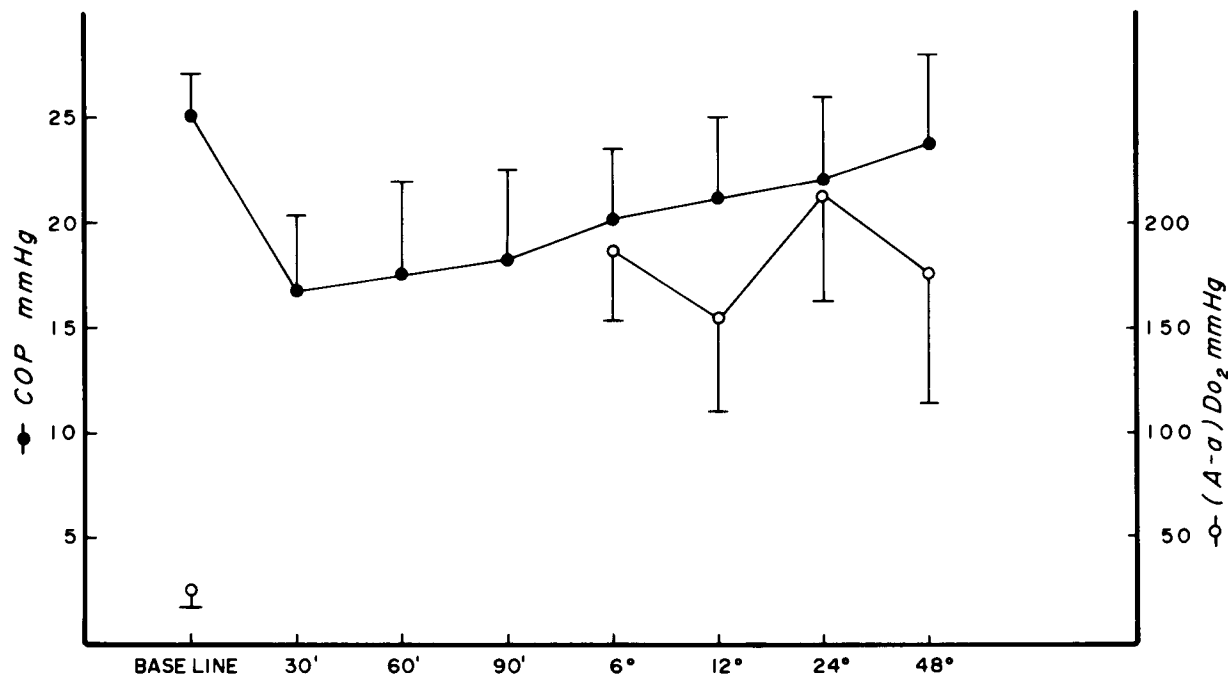


FIGURE 3. Changes in colloid oncotic pressure (COP) and alveolar-arterial tension differences ((A-a)DO₂) baseline, during (only COP), and after (48 hours) perfusion. Each point represents mean value \pm S.D. for 25 patients undergoing coronary artery bypass surgery.

relatively short, we did not see colloid oncotic pressures reduced to the levels usually associated with pulmonary edema. In all cases, the decrease in colloid oncotic pressure appeared to be self-correcting and not correlated with post-perfusion pulmonary dysfunction. We conclude that decreases in colloid oncotic pressures are not the cause of pulmonary dysfunction following relatively short cardiopulmonary bypass.

Acknowledgment

We wish to express our appreciation to Dr. F. T. Caldwell, Jr. for generously allowing us the use of the Weil Colloid Oncometer.

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