Osmotic Pressure of the Serum Proteins

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ABSTRACT

The osmotic pressure of the serum proteins (colloid osmotic pressure [COP] or "oncotic" pressure) is only one of the four Starling forces (plus the capillary permeability coefficient) which affect the net filtration of fluid from the capillaries. The COP will vary with the concentration of total serum proteins, but more so with the specific pattern or composition of the protein components, especially albumin. The use of formulas utilizing total protein (or albumin/globulin) to calculate COP is not warranted. COP should be determined; this is easy at the present time with the advent of a compact commercial instrument. Generalized or localized edema (e.g., pulmonary edema or ascites) has been associated with low serum albumin and low COP values. This is not always so since cases of analbuminemia do not necessarily exhibit edema. The study of COP is warranted but precautions are necessary in proper interpretation of the causes of "edema,"—the Starling forces and hemodynamic factors, capillary permeability, lymphatic return, etc., are all involved in the phenomenon.

Introduction

In 1964, as part of the previous seminar on serum proteins, Kupke30 stated that "Osmotic pressure means different things to different persons...." To the purist, osmotic pressure is the difference in the ability of solvent molecules (e.g., water) to pass through a semipermeable membrane.30 A thermodynamic definition is the additional pressure, at constant temperature and volume, necessary to raise the potential or activity (α) of the diffusible component in the solution phase to equal that in the pure solvent phase (for pure water, α = 1).29 The potential, conveniently a pressure difference, is called the osmotic pressure (π). A practical definition of osmotic pressure is that hydrostatic pressure on a solution containing a non-diffusible solute (e.g., protein) which just prevents a net flow of solvent across a semipermeable membrane separating the solution and the solvent.29

The fraction of the total osmotic pressure, owing to macromolecules such as protein, is termed the colloid osmotic pressure (COP) or "oncotic" pressure. Both medical38 and non-medical66 dictionaries have similar definitions for osmotic pressure and for osmometer; however, medical dictionaries also define an
osmometer as a device for measuring the acuteness of smell. Webster defines oncotic pressure as the osmotic pressure exerted by the plasma proteins, etc.; the medical dictionaries do not define oncotic pressure. Both types of dictionaries define "oncometer" as an instrument to measure variations in size or volume of the internal body organs; this is related to oncology, the study of tumors, and to oncotomy, the incision of a tumor or swelling.

If the non-diffusible component possesses a net charge (e.g., serum proteins exist as anions), there is a greater concentration of the smaller diffusible cations in the side containing the greater concentration of protein. Thus, a greater potential or pressure is necessary for equilibrium when charged macromolecules are present; the extra pressure due to the excess diffusible ions is termed the Donnan pressure or the Gibbs-Donnan equilibrium. The higher the concentration of the proteins, the more progressively significant does the Donnan equilibrium become.

In table I are illustrated some of these differences between the intravascular fluid (IVF) and interstitial fluid (ISF). The cation concentration (millimoles or millimoles) is greater in the plasma (side with more protein) than in the interstitial compartment. In contrast, the chloride concentration (and total anions) is greater in the interstitial fluid in contrast to the plasma. The total concentration of mil-

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>Illustrative Theoretical Values of Milliosmole Content of Body Compartments*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substance</td>
<td>Intravascular (IVF)</td>
</tr>
<tr>
<td></td>
<td>mosmol/l</td>
</tr>
<tr>
<td><strong>ELECTROLYTES</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Cations</strong></td>
<td></td>
</tr>
<tr>
<td>Sodium</td>
<td>142</td>
</tr>
<tr>
<td>Potassium</td>
<td>5</td>
</tr>
<tr>
<td>Calcium</td>
<td>2.5</td>
</tr>
<tr>
<td>Magnesium</td>
<td>1</td>
</tr>
<tr>
<td><strong>Sub-totals</strong></td>
<td>150.5</td>
</tr>
<tr>
<td><strong>Anions</strong></td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>26</td>
</tr>
<tr>
<td>Chloride</td>
<td>103</td>
</tr>
<tr>
<td>Phosphate</td>
<td>1</td>
</tr>
<tr>
<td>Sulfate</td>
<td>0.5</td>
</tr>
<tr>
<td>Organic acids</td>
<td>5.5</td>
</tr>
<tr>
<td>Proteinate</td>
<td>2</td>
</tr>
<tr>
<td><strong>Sub-totals</strong></td>
<td>138</td>
</tr>
<tr>
<td><strong>TOTAL ELECTROLYTES</strong></td>
<td>288.5</td>
</tr>
<tr>
<td><strong>NON-ELECTROLYTES</strong></td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>5</td>
</tr>
<tr>
<td>Urea</td>
<td>4</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>Others</strong></td>
<td>-</td>
</tr>
<tr>
<td><strong>TOTAL NON-ELECTROLYTES</strong></td>
<td>9.2</td>
</tr>
<tr>
<td><strong>TOTAL MILLIOSMOLES</strong></td>
<td>297.7</td>
</tr>
<tr>
<td><strong>Corrected millimoles</strong></td>
<td>-</td>
</tr>
<tr>
<td><strong>Osmotic pressure (mmHg)</strong></td>
<td>-</td>
</tr>
</tbody>
</table>

*Modified from Guyton.
†Phosphocreatine, carnosine, adenosine triphosphate (ATP) and hexose monophosphate.
§Based on "activity" of about 90 percent (owing to interionic and intermolecular attraction and/or repulsion).
×Osmotic pressure (at 37°) in mmHg = 19.3 x (mosmol per kg of water).
liosmole is corrected for an activity of about 90 percent, resulting in a net difference of 1.3 mosmol between the IVF and ISF; if only the protein concentration were considered, the difference would be 2.1 minus 0.2, which equals 1.9 mosmol. Multiplying the milliosmoles by 19.3 gives the osmotic pressure at 37°; the difference, based on total osmoles, between the IVF and the ISF (and intracellular fluid [ICF]) is 25 mmHg, the “colloid” osmotic pressure. The colloid osmotic pressure is less than 0.5 percent of the total osmotic pressure of plasma (table I).

Capillary Fluid Exchange

The osmotic pressure of the serum protein is only one of the four Starling factors regarding fluid movement across a capillary. The Starling hypothesis can be expressed as

\[ F = K_f [(P_c - P_{isf}) - (\pi_{pl} - \pi_{isf})] \]

in which \( F \) is the net capillary filtration, \( K_f \) is the capillary (ultra)filtration or membrane permeability coefficient (amount of fluid filtered per unit surface area of capillary per unit pressure difference across the capillary wall), \( P \) is the hydrostatic pressure, \( \pi \) is the colloid osmotic pressure, \( \epsilon \) is capillary, \( \pi_{pl} \) is plasma, and \( \pi_{isf} \) is interstitial fluid.

Water and solutes of small molecular weight leave the systemic capillaries and return to the circulation by reentering venous capillaries. The Starling hypothesis is an oversimplification of a complex phenomenon. Rather than simple filtration at the arteriolar end and simple reabsorption at the venular end of a capillary, the relative duration of the constrictor and dilator phases of vasomotion (rhythmic contraction and relaxation of the precapillary sphincters and of the smooth muscle of the terminal arterioles and metarterioles) controls the flow of fluid.71

During the dilator phase (rapid blood perfusion) fluid filters out along the entire length of the capillary; during the constrictor phase (blood flow ceases) fluid enters the capillary throughout its length.71 The variation in dilator/constrictor phases is from less than one to ten per minute. The tissue or blood \( P_{O_2} \) serves as an autoregulator of vasomotion.81 In the presence of a decreased \( P_{O_2} \) the dilator phase occurs more frequently and for longer periods; the increased blood flow allows the passage of fluid (and nutrients and oxygen) out along the entire capillary.9 If the blood flow is extremely rapid, not all of the oxygen may be released to the tissues.51 In the presence of elevated \( P_{O_2} \) values the capillary is constricted and fluid reabsorption can occur along its length. The vasoconstrictor effect of oxygen can be reversed by the concomitant administration of carbon dioxide.32

In contrast to the vasodilator effect of hypoxia on smooth muscle (systemic and cardiac blood vessels), both alveolar hypoxia and pulmonary vessel hypoxia cause constriction of the small arteries and arterioles in the lung.19,23 Elevated \( P_{O_2} \) values cause pulmonary vasodilatation, with concomitant outward passage of fluid,—a possible factor contributing to the pulmonary pathology seen in “oxygen toxicity.”

Fluid may return into a capillary and it may also enter a lymphatic vessel; in addition, the intercellular junctions of the lymphatic endothelium allow passage of macromolecules and even particles the size of erythrocytes.56 Other factors affecting fluid flow are capillary permeability, distensibility (compliance) of the tissues, rate of lymphatic drainage, elastic recoil of connective tissue mucopolysaccharides, etc.

Vasomotion affects the filtration coefficient (\( K_f \)) and the intracapillary hydrostatic pressure (\( P_c \)) in equation 1. The body contains about 10 billion capillaries with a surface area of 500 m². The \( K_f \) varies in different tissues, being small in brain and muscle, moderate in subcutaneous tissue,
large in the intestines, and extreme in the liver. This is correlated to the different protein permeabilities, so that the interstitial fluid total protein concentration is 1.5 percent in muscle, 2 percent in subcutaneous tissue, 4 percent in intestines, and 6 percent in liver. Measurements of Kf have been studied by changes of limb volume27,28 or weight of isolated organs.12 The value for body Kf is 5.67 ml per min per mmHg and the average tissue Kf is 0.008 ml per min per mmHg per 100 g of tissue.19 The net rate of fluid flow for the entire body is 1.7 ml per min, which must be balanced by reabsorption via the capillaries and the lymphatics.

Measurements

The capillary hydrostatic pressure (Pc) has been estimated by direct cannulation of the arterial ends (30 to 40 mmHg) and the venous ends (10 to 15 mmHg) with an average of 25 mmHg in the middle. Indirect functional measurements (isogravimetric or isovolumetric) yield values of 17 mmHg.19

The interstitial hydrostatic pressure (Pistf) had been considered to be positive until 1960 when Guyton20,21 reported the values to be negative. The brilliant studies utilized the implantation of a perforated capsule in the tissues; after one month the measurements can be made.

Another technique is to suck the skin away from the looser subcutaneous tissue; measurements are made a day later. In both methods the Pistf is about -6 to -7 mmHg. More recently, Scholander48,49 has utilized a cotton wick method; the results are similar. Negative values for Pistf result in an additional force to foster outward flow of fluid for systemic and pulmonary capillaries, but not for the renal capillaries4,41 in which it is positive (table II).

The interstitial colloid osmotic pressure (πistf) was thought to be very low due to the very low protein concentration. The ISF protein has a disproportionately high albumin to globulin ratio since the capillary endothelium will pass albumin molecules 1.6 times as readily as the globulin molecules. Each gram of albumin exerts twice the osmotic pressure of a gram of globulin (respective average molecular weights are 69,000 and 140,000 daltons). Thus it is the albumin that exerts the major portion of osmotic pressure in the plasma, etc.

In every tissue the heterogeneous colloidal ground substance system, in spite of fluctuations in its state of hydration, is in equilibrium with the plasma. The water content is distributed between two phases,—a semi-solid gel phase in which the water is tightly bound to the dense matrix and a water sol phase contained in the submicroscopic inclusion vacuoles.18 The gel phase, which normally contains about 50 percent of the interstitial fluid volume, is not available to proteins; the free fluid phase, therefore, has an effective protein concentration about two times higher than the protein concentration reported for the total interstitial fluid space (gel plus water phases).69 The average “plasma” protein concentration in the interstitium is 1.7 percent, which could exert a colloid osmotic pressure of about 5 mmHg, whereas the lymph from practically every organ of the body contains 3 to 4 percent protein which could exert a colloid osmotic pressure of about 10 mmHg.61

### TABLE II

<table>
<thead>
<tr>
<th>Systemic</th>
<th>Pulmonary</th>
<th>Renal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>OUTWARD</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pc</td>
<td>17</td>
<td>7</td>
</tr>
<tr>
<td>πistf</td>
<td>5</td>
<td>16</td>
</tr>
<tr>
<td>Pistf (negative)</td>
<td>6.3</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>28.3</td>
<td>29</td>
</tr>
<tr>
<td><strong>INWARD</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pistf (positive)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>pl</td>
<td>28</td>
<td>28</td>
</tr>
<tr>
<td>Total</td>
<td>28</td>
<td>28</td>
</tr>
<tr>
<td><strong>NET OUTWARD PRESSURE</strong></td>
<td>0.3</td>
<td>1</td>
</tr>
</tbody>
</table>

*Symbols as for Equation 1.
†Based on data of 19,20,21,33,46.
Colloid Osmotic Pressure of Plasma

Theoretical and empirical formulas utilizing plasma protein concentration to calculate colloid osmotic pressure have been annotated. However, such calculations are only approximations, being inadequate especially when the protein fractionation pattern is abnormal. It is best to measure the colloid osmotic pressure directly.

Various methodologies to determine colloid osmotic pressure have been reported. Hansen's capacitance manometer was a great advance and used by various groups. This was improved, incorporating a flow-through chamber, for whole blood colloid osmotic pressure. Modifications have been reported, but the only one currently available commercially is Weil's modification of the Prather et al instrument and which utilizes discrete 300 µl plasma samples at room temperature.

Ladegaard-Pedersen has reported various aspects of proper collection of a specimen and the conditions for determining the plasma colloid osmotic pressure. It is best to use an arterial or central vein specimen. If a peripheral vein is used the blood should be taken "immediately" after placement of the tourniquet or one should wait for three minutes after the tourniquet has been removed; waiting for only one minute of free-flow gives COP values about 4 percent higher than with the tourniquet in place. Allowing blood to stand for one day prior to separation of the cells resulted in a significant elevation of about 1 percent; however, plasma separated immediately had the same value even if kept refrigerated for up to 21 days. Freezing did not affect COP values. Venous blood (lower pH and higher P_co2 values) had COP values 0.5 percent higher than an arterial specimen. Fasting (and dehydration) for eight hours resulted in average elevations of 3.3 percent; non-fasting had more stable values. There was a daily variation of 10.1 percent for the fasting and 8.4 percent for the non-fasting condition in patients at bed-rest.

The most important variable is the position of the subject. Ambulation for only one hour gave higher values of 14.7 percent (range 5 to 25 percent). The lower the COP value and the older the patient, the greater the percentage increase after ambulation. The COP values during recumbency also varied by ±10 percent. Hemolyzed plasma specimens have increased COP values of about 5 percent in contrast to non-hemolyzed specimens. Sodium heparin does not affect the COP values if the concentration is less than 200 units per ml. Citrated or EDTA plasmas have falsely elevated, whereas oxalated plasmas have falsely decreased COP values. High plasma glucose values may interfere with the equilibration across the membrane.

Values for COP have been expressed in terms of millimeters or centimeters of water, torr or millimeters of mercury (1 mmHg equals 1.36 cm of water) and kilopascals (1 kPa equals 7.5 mmHg). Various normal values, utilizing different instruments at different temperatures, have been reported; the numbers for each group have not always been designated. In table III are listed various normal ranges for colloid osmotic pressure; for convenience, pressures reported as mm or cm H2O have been recalculated to mmHg. Most publications indicate that the COP was determined at room temperature. Skillman, however, corrected such values to 37°, based on the report of Soto-Rivera, who used the Hepp osmometer to determine COP for comparison against the plasma density. The van't Hoff approximation for osmotic pressure is

\[ \pi = CRT \]
**TABLE III**

Normal Ranges of Colloid Osmotic Pressure Values*

<table>
<thead>
<tr>
<th>Year</th>
<th>No.</th>
<th>Instrument Used</th>
<th>Temperature Type</th>
<th>COP Supine</th>
<th>COP Ambulatory</th>
</tr>
</thead>
<tbody>
<tr>
<td>1962</td>
<td>10</td>
<td>N.A.</td>
<td>0°</td>
<td>21-35</td>
<td></td>
</tr>
<tr>
<td>1967</td>
<td>31</td>
<td>Hansen</td>
<td>21-24°</td>
<td>18-31†</td>
<td>23-34‡</td>
</tr>
<tr>
<td>1970</td>
<td>55</td>
<td>Hansen</td>
<td>Room×</td>
<td>25-28</td>
<td></td>
</tr>
<tr>
<td>1971</td>
<td>11</td>
<td>Hansen</td>
<td>N.A.</td>
<td>23-36</td>
<td></td>
</tr>
<tr>
<td>1971</td>
<td>38</td>
<td>Intaglietta &amp; Zweifach</td>
<td>20°</td>
<td>18-35‡</td>
<td>25-40†</td>
</tr>
<tr>
<td>1973</td>
<td>65</td>
<td>Hansen</td>
<td>N.A.</td>
<td>21-29‡</td>
<td></td>
</tr>
<tr>
<td>1974</td>
<td>47</td>
<td>Intaglietta &amp; Zweifach</td>
<td>37°</td>
<td>24-31‡</td>
<td>(20-28)†</td>
</tr>
<tr>
<td>1975</td>
<td>7</td>
<td>N.A.</td>
<td>N.A.</td>
<td>(19-22)†</td>
<td></td>
</tr>
<tr>
<td>1976</td>
<td>24</td>
<td>N.A.</td>
<td>N.A.</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>1976</td>
<td>3</td>
<td>Hansen</td>
<td>N.A.</td>
<td>(24-33)†</td>
<td></td>
</tr>
<tr>
<td>1977</td>
<td>14</td>
<td>&quot;Weil&quot;</td>
<td>25-26°</td>
<td>23.5±0.4‡</td>
<td></td>
</tr>
</tbody>
</table>

*Values rounded off and/or 95 percent limits calculated from S.D.
†Original cm H₂O or mm H₂O recalculate to mmHg (1.36 cm H₂O = 1 mmHg).
§After one hour ambulation.
×Corrected to 37°.
% S.E.
N.A. Not available.
( ) Values for children.

Left ventricular filling pressure (LVFP) may be indirectly approximated by the pulmonary artery diastolic pressure (PADP) or the pulmonary artery wedge pressure (PAWP). Although intended for the treatment of acute myocardial infarction, a PAWP of 18 mmHg has also been adopted as the maximum since values above that level usually result in pulmonary edema. Stein et al report the development of pulmonary edema in patients with normal LVFP in the absence of cardiac failure but in whom the COP was reduced to 16 mmHg (average of 11 patients). They advocate the use of colloid rather than electrolyte (non-colloid) solutions in the therapy of hypovolemia. Morissette et al report that no patient with cardiopulmonary failure survived if the COP was less than 10.5 but all the survivors had values greater than 19 mmHg. Luz et al have expanded their studies on pulmonary edema and report the difference between COP and PAWP to be of greater significance in predicting the onset of pulmonary edema. In the absence of pulmonary edema, COP was 20.8 mmHg and reduced to 16.9 mmHg in those patients with pulmonary edema; both groups of patients had acute myocardial infarction. Normal supine patients averaged 20.6 mmHg, using the normal PAWP of 4 to 12 mmHg results in the COP-PAWP gradient of 9 to 17 mmHg, which normally favors the retention of fluid within the capillaries. In the patients with acute myocardial infarction without edema in contrast to those with pulmonary edema, the respective gradients had an average of 9.7 ± 1.7 S. E. versus 1.2 ± 1.3 S. E.

Not all of the reported research supports the conclusions of Weil and his associates. In a study on 43 patients undergoing hemodynamic resuscitation during and following abdominal aortic surgery, there was not a single example of pulmonary edema in the absence of an elevated hy-
drostatic pressure. Decreased COP or COP-PAWP gradient did not result in pulmonary edema if PAWP was not elevated, despite the presence of edema elsewhere. When the COP is low, the lung is seemingly protected by increased lymph flow, increased lymph albumin clearance to lower interstitial osmotic pressure and increased pulmonary interstitial pressure.

In a series of cases of reversible pulmonary edema without cardiac dilatation the COP and PAWP values were normal. The edema was probably due to increased filtration caused by increased alveolar capillary membrane permeability (Equation 1). Guyton explains the safety factor against pulmonary edema as the strong dehydrating force of the COP in the lungs; the pulmonary capillary pressure must rise to above 30 to surpass the 28 mmHg COP in the lungs (table II). Patients with chronically elevated pulmonary capillary pressures (even as high as 45 mmHg) may not develop pulmonary edema because of the extremely rapid run-off of the fluid to the lymphatics.

If the pulmonary capillary pressure remains elevated for more than two weeks, the pulmonary lymphatics enlarge as much as six to ten times, with an increased lymph flow of about 20 times above the resting level. In patients with pulmonary edema due to left ventricular failure, the COP was elevated; this was explained by the transudation of fluid which is low in protein content into the lungs. This is substantiated by the increase in hematocrit and concentration of plasma proteins following the onset of pulmonary edema.

It was thought that the presence or absence of ascites in patients with cirrhosis of the liver was determined by the concentration of serum albumin. Mankin and Lowell have demonstrated that there is a constant difference between the COP of the plasma and the ascitic fluid (e.g., approximately 18 and 6 mmHg, respectively) which was maintained despite alterations of the COP in each fluid. Reducing the serum protein concentration of rats (during development of aminonucleoside nephrosis) was followed by a reduction of the interstitial fluid protein concentration (and interstitial fluid osmotic pressure), with the result that edema formation did not occur in the presence of the hypoproteinemia. Bjømeboe had postulated that the serum protein concentrations were so adjusted that the COP was kept at a normal level.

If the globulins are increased owing to antibody production, the albumin is decreased; if the albumin is decreased owing to liver impairment, the globulins increase. Cases of hepatitis with ascites and edema are examples in which the regulatory mechanism has failed. Studies on perfused rat liver also reveal similar results.—COP regulates albumin synthesis. In children at risk to kwashiorkor, the early fall in serum albumin concentration (with a normal COP) may be a response to the high globulin concentrations caused by frequent infections.

Other studies report that the COP is the prime determinant of the hypercholesterolemia seen in the nephrotic syndrome. In normal subjects the total serum cholesterol has a positive correlation with the serum albumin concentration. In the nephrotic subjects (most with albumin levels below 2 g per dl) there is a negative correlation between the cholesterol and albumin concentrations. Conwill et al suggest that the continued loss of albumin and low molecular weight globulins in nephrotic subjects causes a fall in COP which serves as a nonspecific stimulus for synthesis of all hepatically derived serum proteins (e.g., \( \beta \)-lipoprotein increase accounting for the hypercholesterolemia).

Schultze and Heremans report that edema usually results if the COP falls below 18 to 20 mmHg (recalculated by present author). The normal values for
children were reported by Boman et al\textsuperscript{3} as 24 to 33 mmHg; yet a child with analbuminemia (zero albumin with 5.5 g of total protein) and a COP of about 15 mmHg had no signs of peripheral edema. Cormode et al\textsuperscript{7} report a similar case of analbuminemia in a neonate, confirming the congenital origin of the disorder. Baum and Harris\textsuperscript{1} report that infants with erythroblastosis fetalis had total protein and albumin concentrations commensurate with the normals for gestational age but had abnormally low values of COP. Their suggestion (without a firm conclusion) is that a given protein concentration exerts less of an effect on COP in erythroblastosis.\textsuperscript{1}

Controlled hemorrhage (15 percent of the measured blood volume) in normal men resulted in a significant drop in COP by six hr and continued to 24 hr.\textsuperscript{54} The albumin concentration did not drop, but there was a significant decrease in globulin concentration. It had been shown that after hemorrhage there is a decrease in capillary hydrostatic pressure, serving as a stimulus to transcapillary refilling of the blood volume by 48 hr after the acute blood loss.\textsuperscript{53} A control study in which the bloodletting was accompanied by a simultaneous autologous blood transfusion did not result in a drop of the COP.\textsuperscript{51}

Major surgery, however, will result in a loss of total protein (albumin) owing to sequestration of plasma in areas not accessible to the intervascular space; whole blood replacement is not sufficient to replace the protein losses.\textsuperscript{54} Surgical patients receiving whole blood had significant drops of total protein, albumin and COP in contrast to those receiving whole blood supplemented with albumin.\textsuperscript{24} However, in patients on dilutional cardiopulmonary bypass, the administration of salt poor albumin in addition to whole blood did not significantly increase the rise in postoperative COP when compared to whole blood alone.\textsuperscript{37} A comparison of patients in cardiopulmonary bypass—normothermic versus hypothermic (25 to 28°C)—revealed the hypothermic group to have normal COP values 90 min after cessation of bypass.\textsuperscript{37} Hypothermia per se can elevate colloid osmotic pressure.\textsuperscript{62} Possible mechanisms for the normalization include postbypass “cold diuresis” (intraoperative urine output three times that seen in normothermia), water shifts out of the blood, or intravascular translocation of interstitial protein.\textsuperscript{37}

References


