Blood Damaging Effects in Cardiotomy Suction Return

Richard A. Malinauskas, Robert M. Sade, James P. Dearing, Francis G. Spinale, Frederick A. Crawford and Andreas F. von Recum

Medical University of South Carolina
Department of Surgery
Charleston, SC

Clemson University
Department of Bioengineering
Clemson, SC

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Abstract

(J. Extra-Corpor. Technol. 20[1]: p. 40-45, 44 references, Spring Issue) **Over the past three decades improvements have been made to many of the components of the extracorporeal circulation system resulting in decreased blood damage. However, the cardiotomy suction return sub-circuit, recognized as one of the most injurious components of heart-lung bypass, has remained relatively unchanged. The purpose of this study was to investigate the amount of blood damage that occurred with different cardiotomy suction designs. The five test designs compared two different suction tips with various air-mixing techniques, *in vitro*. Significant blood damage was observed in the air-containing designs as compared to the airless and control circuits. A minimal flow rate capable of excluding air was demonstrated as being the most important factor for blood protection, irrespective of aspirator tip design.**

Introduction

Blood damage during heart-lung bypass is largely due to the trauma of cardiotomy suction return.[1-3] This essential subcircuit returns approximately 10% of the cardiac output through the coronary and bronchial veins but must sometimes accommodate at least 50% of the perfusion flow.[4] Mixed with air, an average of 21.0 L/hr of blood is returned via the cardiotomy suction lines in standard coronary artery bypass operations.[5]

The major components of the cardiotomy suction system are an aspirator tip, approximately twelve feet of tubing, a roller pump for suction, and a cardiotomy reservoir with blood defoaming capabilities. Blood damage may include platelet injury,[6-9] blood protein denaturation,[10] and red blood cell (RBC) injury and lysis.[4,6,11-16]

Studies of aspirator tips,[1] blood handling tubing,[17] blood pumps,[18,19] and different oxygenator designs[20,21] have produced no long-lasting modifications of the cardiotomy suction return circuit.[4,22,23] Moreover, the complex flow relationship of the air/blood mixture is not well understood, even though damage to RBCs and platelets by shear stresses[24-30] and blood component interactions during air/blood mixing in tubular flow have been studied.[5,10,11,31-33]

Therefore, we have evaluated the blood damaging elements of the cardiotomy suction systems, emphasizing aspirator tip design and degree of air admixture with blood.

Materials and Methods

Standard clinical materials and equipment (extracorporeal tubing, connectors, and roller pumps) were used to create five similar cardiotomy suction circuits (Figure 1). Eight experiments were conducted utilizing heparinized blood from a different donor dog for each experiment. To facilitate the use of only one dog per experiment, blood aliquots of 300 ml were adjusted to a hematocrit (Hct) of 25% with physiological saline and placed into each of the five polypropylene "patient"

Direct communications to: Dr. Andreas F. von Recum, Department of Bioengineering, 301 Rhodes Engineering Research Center, Clemson University, Clemson, SC 29634-0905.

cups. The blood was then aspirated by either an intracardiac (IC) sucker[a] or an identical length of 1/4" internal diameter (ID) polyvinyl chloride (PVC) tubing.[b]

Circuits 1 and 2 aspirated only blood from the "patient" by using the two different aspirators with a pump speed of 400 ml/min (Figure 2). The remaining three circuits introduced a mixture of approximately 50% air and 50% blood into the systems. Circuits 4 and 5 aspirated air and blood continuously (800 ml/min) at the blood pool surface by using a PVC and an IC tip, respectively. Circuit 3 utilized a fully immersed IC tip in the blood pool while air was drawn directly into the tubing at a "Y" connector (Figure 2).

To adjust for the reduced volume (300 ml) available for each circuit while maintaining clinical tubing lengths (total of 18 ft. of 1/4" PVC tubing required a blood volume of approximately 175 ml), we fabricated a smaller version of the clinical cardiotomy reservoir. The modified cardiotomy reservoir, utilized in all of the circuits, consisted of a 250 ml polypropylene cup which had a section of PVC (1/4" ID) tubing cemented in the bottom for "debubbled" blood to flow through. The lids of the reservoirs had two sections of PVC tubing cemented in them: one to direct inflow, and one to vent air. To recreate the defoaming capabilities of the reservoir, a clinical method which was utilized until disposable cardiotomy reservoirs became available in the early 1970's was applied (Personal Communication: Mr. James P. Dearing). Hence, Tuffy[c] polypropylene sponges, which had been coated with a silicone defoaming agent,[d] were used as defoamers in the cardiotomy reservoirs. The flow rate of defoamed blood from the modified cardiotomy reservoirs back to the "patient" pools was fixed at 400 ml/min, such that, the total volume of pumped blood was the same for all five circuits (24.0 L).

Blood samples were drawn and refrigerated after each hour run of the five experimental circuits. Two room temperature control samples were drawn for each experiment: one after hemodilution of the blood to Hct = 0.25 (Initial Control, C: time = 0 hrs) and one at the completion of all five circuit runs (Final Control, F: time = 2.5 hrs).

The hematological variables measured by a Coulter Counter[e] included: hematocrit (Hct), red blood cell (RBC) count, platelet count (PLT), total cellular hemoglobin (Hgb), and mean corpuscular hemoglobin

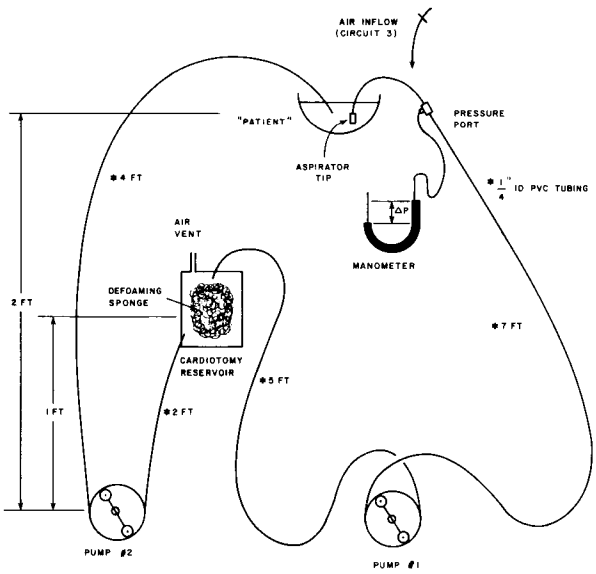

Figure 1: Cardiotomy Suction Circuit: Basic Testing Configuration

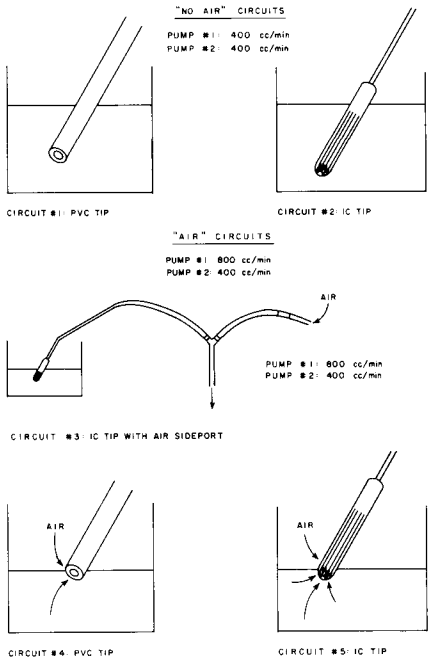

Figure 2: Specific Treatment Designs

(MCH). Spectrophotometric hematological measurements included the evaluation of free plasma hemoglobin (FPH),[34]a sensitive marker for blood damage, and red cell osmotic fragility.[35] Blood smears, prepared with Wright's stain, were also used to analyze the extent of morphological damage to the red blood cells.

Schlichting's modified equation[39] for the evaluation

a Model 999, Sarns Corp., Ann Arbor, MI 48106
b American Bentley, Irvine, CA 92714
c Miles Laboratories Inc., Elkhart, IN 46515
d Dow Corning Silicone Stopcock Grease, Dow Corning Corp., Midland, MI 48640
e Model 7-90, Coulter Electronics, Hialeah, FL 33010

of shear stresses in a turbulent jet was applied to the "air" circuits.

$$\tau = 0.03\ (2\triangle P/\varrho),$$

where,

$\triangle P$ = pressure drop across the aspirator (mmHg)

and,

ϱ = calculated density of measured air (53%) and blood (47%) mixture (gm/ml).

The statistical computer package BMD[f] was used for analysis of the hematological variables by performing the Wilcoxon Signed-Rank Test for Paired Observations, the Kruskal-Wallis One-way Analysis of Variance Test, and Spearman's Rank-correlation Coefficient Test.[36] The levels of probability are presented in the text.

Results

All blood aliquot control values were within the normal ranges for dogs.[19,37,38] The values for RBC count and Hct remained within these hemodiluted control limits at all times in all circuits. However, measurements of FPH revealed distinct differences between the circuits, as is evident in Figure 3. The mean values (± S.D.) of FPH increased from the initial control value of 28.9 ± 9.4 mg% to 41.1 ± 15.7 mg% ($p<0.05$) after the blood had been stored at room temperature for 2.5 hours (final control). The mean values for circuits 1 and 2 were 52.9 ± 25.9 mg% and 60.2 ± 54.8 mg% revealed no difference. FPH in circuits 3, 4, and 5 increased to 761 ± 1002 mg%, 859 ± 1023 mg%, and 991 ± 1339 mg%, respectively, which was significantly more than in the "no air" circuits ($p<0.01$). No differences appeared among the "air" circuits, but circuit 5 was greater than circuit 3 ($p<0.07$).

A Wilcoxon sign-rank test performed on the FPH, Hgb, and MCH data substantiated the significant differences between the "no air" and the "air" circuits. However, there were no significant differences ($p<0.05$) between the two trips within each flow group ("no air": circuits 1 and 2, and "air": circuits 4 and 5).

Similar results were obtained from the osmotic fragility tests (Figure 4). The non-osmotic fragility test also correlated well ($r=0.83$, $p<0.01$) with the FPH data, indicating that the amount of sublethal damage to the RBCs was directly related to the amount of hemoglobin released into the plasma. Similarly, in the Wright stained blood smears, as the suspending plasma darkened due to increased amounts of released hemoglobin, distinct morphological changes in the RBCs (crenation, ghost cells) also appeared.

The PLT count decreased ($p<0.01$) most in the circuits with air-blood mixing (Figure 5). There was also an inverse correlation between the FPH and PLT count ($p<0.01$).

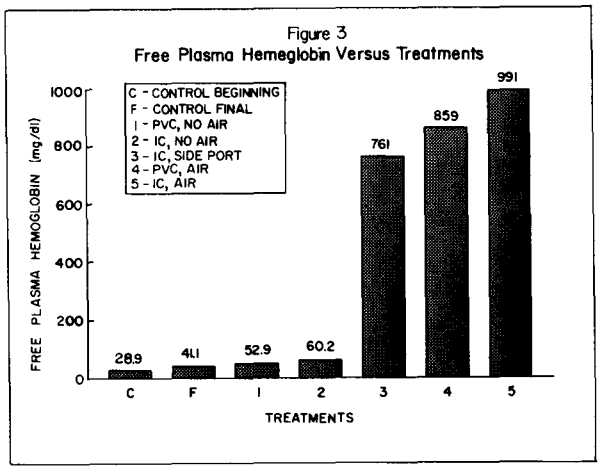

Figure 3: Free Plasma Hemoglobin Versus Treatments

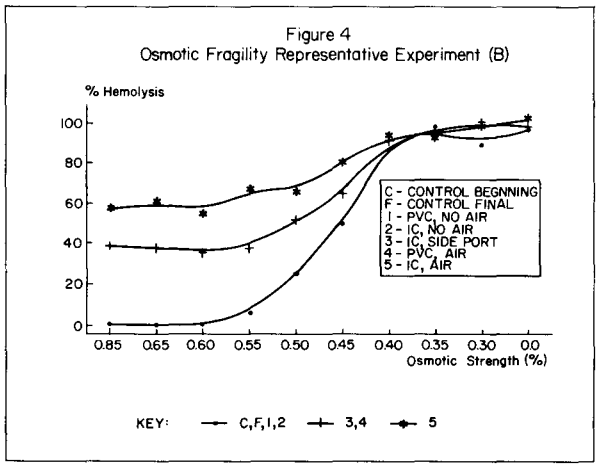

Figure 4: Osmotic Fragility Representative Experiment (B)

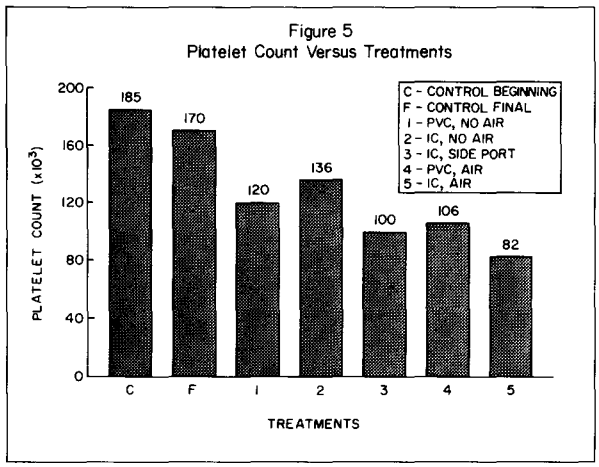

Figure 5: Platelet Count Versus Treatments

f UCLA, Los Angeles, CA 90024

Discussion

Patients undergoing cardiopulmonary bypass exhibit a slight decrease (1%) in Hct and RBC count.[4] This is consistent with the results presented here which show that RBCs are damaged rather than destroyed, resulting in an immediate increase in FPH and a subsequent decrease in RBC lifetimes,[6,7] manifested as postoperative anemia.

Blood smears paired with the osmotic fragility data (Figure 4) illustrate how sublethal RBC damage appears as FPH. Although percent hemolysis leveled off at 0.00-0.35% osmotic strength, there was no visible deformation of the RBCs in the 0.35% solution, while rampant ghost cells in a hemoglobin stained plasma field were evident in the distilled water solution (0.00%). This suggests that a large number of RBCs do not have to be destroyed to raise the FPH level substantially. Sufficient damage to the RBCs causes an increase in membrane porosity, which allows hemoglobin to leak out of the cell into the plasma.[40,41] This may subsequently lead to an osmotic influx into the cell creating large, deformed corpuscles (poikilocytes and spherocytes) with decreased lifetimes.[26]

Circuits 1 and 2, which aspirated blood but not air, produced little RBC damage. The laminar shear stresses (6.9 and 20.7 dynes/cm^2, respectively) calculated for both of these circuits were well below the trauma threshold of blood (500 dynes/cm^2)[11]. The osmotic fragility curve also confirms that these are minimally damaging circuits. This is an indication that the other components of the circuits (roller pumps, tubing, and cardiotomy reservoir) also only cause minimal blood damage.

Although large variances appeared in the FPH data between experiments in the "air" circuits, apparent conclusions could still be drawn. Air mixing circuits 3, 4, and 5 all showed appreciable blood damage with increased FPH levels and RBC fragility when compared to the controls and the "no air" circuits. Moreover, a marked increase in crenation, hemoglobin stained plasma, and ghost cells appeared in the blood smears of the heavily damaged "air" runs. Even though there was an increase ($p<0.07$) in the amount of FPH damage when the IC tip aspirated blood with air (Circuit 5) than when air was introduced directly into the tubing independent of the tip (Circuit 3), the damage caused by the tips was minimal when compared with the damage caused by the complex interactions of blood elements with air mixing.

Clinically, postoperative bleeding is related to a decrease in the number of functional PLT. The phenomenon of decreasing PLT occurs normally whenever blood is exposed to a foreign surface and platelets aggregate to that surface. However, PLT values decreased significantly between the control, "no air," and "air" test groups, further substantiating that air/blood mixing is responsible for the majority of blood damage in our mock test circuits.

Although the large range of FPH values may, in part, be masking the results, no statistical difference was found between the tips of circuits 4 and 5 when air was aspirated with blood; even though markedly different bubble flow patterns and suction pressures were found between the two tips. The IC tip of circuit 5 was observed to create a flow pattern with many bubbles of diameter 2-4 mm while the PVC tip and sideport caused a distinct separation of blood and air regions in forming a "bolus" type of flow. These differences occurred due to the decreased diameter of the IC tip handle and orifice area (increased flow resistance) and because bubble formation is a function of both the orifice size and air velocity.[42] We expected a difference between these two circuits for several reasons: the suction pressure created in circuit 5 (17.6 ± 1.7 mmHg) was greater ($p<0.001$) than in circuit 4 (5.6 ± 1.5 mmHg), the smaller bubbles of circuit 5 mixed and caused local areas of turbulence as they were broken and reformed, and the gas-blood surface interface was greater for circuit 5, which promoted a higher amount of blood protein denaturation.

The Schlichting[39] calculated tip shear stresses for these two circuits were 868 and 2730 dynes/cm^2, respectively. Although substantial RBC damage appears at 1500 dynes/cm^2, no difference was detected between these circuits. Factors which disrupt the application of this equation to our system include: 1) inaccuracy in measuring the density and viscosity of an air/blood mixture.[11] 2) the Reynolds numbers were below the accepted threshold of turbulence value of 2300. 3) the presence of wall impact[32] and bubble flow pattern effects, and 4) blood damage which can occur under the turbulence threshold.[11]

Although understanding of air/blood flow dynamics in tubing is inadequate and warrants further research, we can conclude that major improvement in the current cardiotomy suction systems must involve the elimination of their major source of damage: unstable air/blood mixing in tubular flow. We advocate continuing investigation of "no air" suction systems for clinical use.[5,31,33,43,44] Until the time when these systems can be widely applied, blood damage due to suction may be minimized by aspirating only pools of blood, avoiding tip occlusion, keeping the pump flow rate at lowest possible levels when in use, avoiding sloshing of air and blood by turning suction pumps off when not needed, and decreasing the length of cardiotomy tubing to avoid loops.[45] These observations also suggest avoidance of

other devices which promote air/blood interaction, such as, left ventricular decompression lines with vacuum relief valves that aspirate air directly into their return lines.

This study re-emphasizes that blood damage due to air/blood mixing continues to be a major design fault in extracorporeal circulation. Reduction or elimination of blood damage extend the safe application period of extra-corporeal circulation and improve patient recovery.

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