Outflow Obstruction of Soft-Shell Venous Reservoir Bags: A Preliminary Investigation

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ABSTRACT

Potential problems have occurred during cardiopulmonary bypass where the area directly above the outlet port of the soft-shell venous reservoir containing a screen has collapsed similar to when the bag is empty, although there are still several hundred milliliters in the bag. A preliminary investigation was conducted to rule out the possibility of this outflow obstruction being attributable to poor flow design through the reservoirs. The test circuit consisted of a cardiotomy reservoir, a centrifugal pump, 3/8 and 1/2 in PVC tubing, and one of the test venous reservoirs (BARD S-2116, Medtronic 1385 and MVR-1600, Sarns 4858). The circuit was primed with outdated packed red blood cells, platelets, human albumin, normal saline, and 10,000 units of sodium heparin in concentrations to simulate routine bypass. Each reservoir was tested at 6.5–7.0 L/min flows for at least 20 min each.

None of the reservoirs tested demonstrated the phenomenon that had been observed clinically. This dismisses flow design flaws as a possible cause. Further clinical investigation must be performed to identify the possible cause or causes of this problem.

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INTRODUCTION

Recently, we have noticed an occasional problem with the newer collapsible venous reservoir bags containing screen filters. Similar reports have also been filed from other institutions concerning other brands of reservoirs. The phenomenon manifests itself in the venous reservoir bag collapsing above the outlet port as if the bag was empty, even when there may be several hundred milliliters still in the bag. While this phenomenon is occurring, the flow rate is greatly affected, making it almost impossible to maintain adequate flows. This problem was observed in our institution in two models of venous reservoir bags manufactured by Medtronic, Inc., model #1375 and MVR-1600. Similar reports have also been filed from other institutions concerning other makes of reservoirs including the BARD S-2116 reservoir (personal communication). In most cases, the problem resolves upon warming, allowing for resumption of full flow. Monitoring of adequate anticoagulation was done using activated clotting times (ACT). All ACT values were maintained at greater than 500 sec. Gross examination of the venous bag post bypass revealed no visible fibrin or cellular deposits on the filter mesh. This investigation was conducted to rule out the possibility of this outflow obstruction being the result of poor flow design through the reservoirs.

MATERIALS AND METHODS

A test circuit was designed to study the flow pattern through several soft-shell venous reservoirs. (Figure 1) The circuit consisted of a centrifugal pump (BP80 Bio-Pump), a flow probe (TX50 Bio-Probe), a “patient” reservoir (BARD H-3700), 3/8 in and 1/2 in polyvinyl chloride tubing, and one of the study reservoirs. (Table 1)

The circuit prime consisted of outdated packed red blood cells, platelets, human albumin, normal saline, and 10,000 units of porcine mucosal sodium heparin in concentrations to simulate routine bypass.

Three reservoirs of each type were tested at flow rates of 6.5–7.0 L/min for at least 20 min each. The study reservoirs were monitored during the trials to visualize if any obstruction was occurring above the outlet port. Manipulation of both the venous reservoir bags and their holders was also done to observe its effect on flow through the reservoir.

RESULTS

At no time during the trial runs was any obstruction to flow noticed within any of the study reservoirs. Even with extreme manipulation and torquing of the outlet port connectors, reservoir holder faceplate, and venous reservoir bag itself, the obstruction observed during clinical use could not be duplicated in the laboratory.

DISCUSSION

When this venous outlet obstruction phenomenon occurs clinically, it can be quite dramatic. The inability to achieve full flow can become a serious problem for the patient. We were unable to reproduce this phenomenon in the laboratory. This dismisses flow design flaws as a possible cause. The problem arises only with direct patient involvement. The scenario is very similar to that seen in hollow-fiber membrane oxygenators with the high transoxygenator pressure phenomenon, whether it be caused by cryofibrinogen or something else de-
A number of articles have reported flow obstruction mainly in membrane oxygenators. Blombäck and co-workers reported pressure increases occurring on the inlet side of the oxygenator during the cooling phase of bypass (3). They concluded that some patients exhibited a tight fibrin gel network in conjunction with immediate cooling once on bypass.

Bearss and colleagues reported numerous accounts of flow impedence attributed to cooling with resolution during warming. Those oxygenators involved had a noticeable quantity of fibrin and/or clotting when they were examined postbypass. They concluded that cryofibrinogen was the cause and offered preventative measures to identify and possibly prevent this phenomenon. Those measures included the addition of albumin to the prime, slowing the rate of cooling, monitoring oxygenator inlet pressure, and rewarming if the problem arises.

In a 1996 study from our institution, investigators reported an unexplained elevation in transoxygenator pressures resulting in less than adequate blood flows. On one occasion, the decrease in blood flow was so severe that oxygenator replacement was required. In cases of unexpected decreases in platelet counts and decreased blood flows, the oxygenators were carefully flushed and dissected postbypass. Fibrin was observed clinging to the heat exchanger.

The following year, we tested oxygenators with uncoated surfaces with and without albumin in the prime along with coated oxygenators. The phenomenon was identified using decrease in circulating platelet counts. This study showed that uncoated surfaces posed the greatest threats for cryofibrinogen formation. Coated surfaces came in second, with better results than the uncoated oxygenators (not statistically significant). The best performers were surfaces coated with albumin in the pump prime.

A more recent investigation at our institution demonstrated that as little as three milliliters of 25% normal serum albumin was adequate to preserve platelet counts and effectively prevent fibrin gel formation.

At the 1999 Annual Seminar of the American Academy of Cardiovascular Perfusion, Jennifer Schaadt presented a lengthy review presenting case reports and publications, both domestic and international, detailing this phenomenon.

Our preliminary suspicions are that this same phenomenon seen in oxygenators and earlier in filters now surfaces on venous reservoir bags containing screen filters. The fibrinogen that is deposited attracts platelets to that surface. These aggregates then occlude portions of the filter mesh decreasing the surface area to the point of restricting flow through the venous bag. This causes the area of the reservoir bag just above the outlet port to collapse, even when the bag had volume in it proximal to the filter sock. (Figure 2)

Further support for this phenomenon occurring is the fact that the problem is not seen clinically when a venous reservoir bag without a filter is used. In addition, it does not occur when albumin is used in the prime or the filter media within the venous reservoir bag is coated.

To reinforce this theory, animal studies must be conducted. The test circuit should include a heat exchanger, enabling investigators to cool or warm the perfusate. Moreover, the elimination of albumin from the prime may aid in formation of cryoprecipitate, which the authors feel is the culprit in this phenomenon.

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REFERENCES


