JECT. 2007;39:267–270
The Journal of The American Society of Extra-Corporeal Technology

Cannulae and Cell Saver Design: Do They Make a Difference?

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Presented at the Perfusion Downunder Meeting, Hayman Island, Queensland, Australia, August 9–11, 2007.

Abstract: In the evolution of cardiopulmonary bypass (CPB), it is becoming increasing obvious that minimizing microembolization is critical in protecting the brain. Every component of the CPB circuit and ancillary apparatus must be evaluated and, if necessary, re-engineered with the reduction of microemboli a major focus. Cardiotomy suction has been identified as a major source of lipid microemboli. However, is the alternative blood treatment apparatus, the cell saver, capable of reducing the lipid embolic load and are all cell savers equally efficient? In the event that microemboli do make it to the aorta, is it possible to divert them away from the brain to more robust vascular beds through clever design of the aortic cannula? Is the venous cannula a source of microgaseous emboli? The answer is yes to both questions. Emboli can be directed away from the brain by the positioning and design of the aortic cannula and the venous cannulae may be a source of gaseous microemboli delivered to the oxygenator by the venous line but careful practice will prevent this type of embolic formation. **Keywords:** brain injury, microemboli, cardiopulmonary bypass. *JECT. 2007;39:267–270*

INTRODUCTION

Embolization is a major cause of morbidity secondary to cardiopulmonary bypass (CPB) and cardiac surgery procedures. Careful monitoring reveals when embolization occurs (1–4) and suggests methods to reduce the number of emboli that reach the brain (Figure 1), such as changing clamping procedures (5,6), using improved cannula, and changing cardiotomy suction (7) and cell saver (8,9) protocols.

The return of contaminated shed blood from the thoracic cavity during CPB, through the cardiotomy reservoir, is associated with lipid (LME) and gaseous (GME) microembolization. The passage of deformable GME or LME, through the vessels of the brain, results in a break down of the blood–brain barrier with associated brain swelling (10). Furthermore, the level of inflammatory mediators and the systemic inflammatory response (SIR) is increased through contact activation caused by the blood being damaged by suction and prolonged contact with non-biocompatible surfaces (11,12). Transfused blood products also contribute to SIR, partly because the route of administration is through the cardiotomy reservoir where the fresh blood products are mixed with contaminated suctioned blood.

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AVOID THE RETURN OF CARDIOTOMY SUCTION BLOOD

Defining the term shed blood has been difficult. However, it is well documented that blood in the thoracic cavity is contaminated with lipid material from the cut surfaces of the sternum (7). Suctioning causes gaseous microemboli to be coated with lipid and protein material and become particulate emboli with a gaseous core. The passage of these emboli through the micro-vasculature contribute to the breakdown of the blood–brain barrier and potentially the systemic inflammatory response syndrome.

IS THE CELL SAVER THE ANSWER

Patients who stay in the intensive care unit for >2 days receive a third more cell saver blood than patients discharged in <48 hours (Table 1). Is it the shed blood or the increased blood loss that causes the more problematic outcome? The studies of Aldea et al. (13), as well as Dr. Hammon's data (personal communication; Table 1), supports the contention that minimizing blood loss results in better outcomes.

It seems that treating shed blood through the cell saver may not be the panacea expected. Kincaid et al. (8) performed a series of experiments in a canine model of CPB to determine whether different cell savers and filters handled lipid emboli equally well as shown in the cerebral

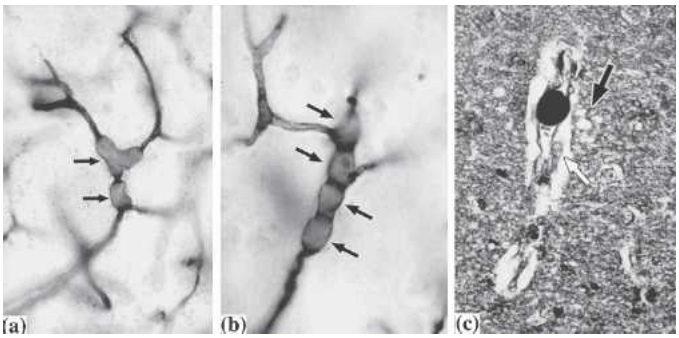

Figure 1. Lipid microemboli from human autopsy after CPB. High-magnification photomicrograph of microlipid emboli from patients who died after CPB. Original magnification, ×50. A and B, Microemboli at bifurcation points (arrows) in 100-mm-thick celloidin sections with AP microvascular staining. C, A microembolus stained black with osmium indicates that it is lipid. Swollen astrocytic end-feet (white arrow) and vacuolization in the adjacent neurophil (black arrow) indicate tissue injury. This is a paraffin-embedded, 5-mm-thick osmium-fixed section (courtesy Professors D. Moody and D. Stump, Wake Forest University School of Medicine, Winston-Salem, NC).

Table 1. Cell saver volume and the length of intensive care unit stay.

ICU Stay	*n*	Cell Saver Amount (Mean ± SD)
<24	43	631 ± 234
24–48	55	680 ± 256
>48	40	829 ± 344

*$p < .05$.
ICU, intensive care unit.
(With permission Professor John Hammon, MD, Cardiothoracic Surgery, Wake Forest University School of Medicine, Winston Salem, NC).

vasculature by small arteriolar dilatations (SCADs). The wide variability in cell saver performance is shown in Figure 2.

THE SOLUTION

Most important is a consensus definition of what is shed blood and/or waste blood and how to quantify the volume of blood either returned or processed. Actual quantification often leads to improved blood management.

Reducing the quantity of shed blood that must be processed and returned to the patient through the cardiotomy reservoir or cell saver is a first priority. Improving the quality of the returned blood through better filtration and blood management is also critical. This can be accomplished by instituting several measures to either reduce bleeding or the volume of blood displaced from the patient:

1. Aggressive surgical techniques to minimize bleeding as it occurs.
2. Aprotinin should be used, when appropriate, to reduce blood loss and protect the patient from inflammatory processes.

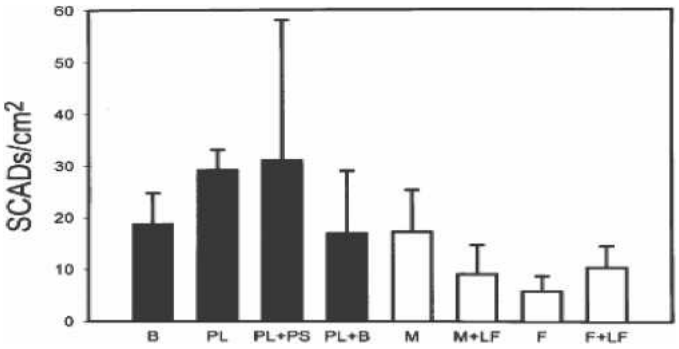

Figure 2. Mean small capillary and arteriolar dilation (SCAD) density ± SE by filter or processed salvaged blood (cell saver) group. Closed bars represent arterial filter group; open bars represent cell saver group. $p < .05$ for cell saver vs. arterial filter groups; $p > .05$ for all other intergroup comparisons. (B, Bentley Duraflow II AF-1025D; PL, Pall Leukoguard AL; PS, Pall Stat Prime; M, Medtronic Autolog Cell Saver; LF, Pall RCXL 1 leukocyte removal filter; F, Fresenius Continuous Autotransfusion System) (from Kincaid EH, Jones TJ, Stump DA, et al. Processing scavenged blood with a cell saver reduces cerebral lipid microembolization. Ann Thorac Surg. 2000;70:1296–300, with permission).

3. Possibly, microcircuits (or minimizing the current system) should be used to minimize blood dilution and the need to return cardiotomy suction shed blood.
4. The arterial filter should not be purged to the cardiotomy reservoir.
5. A 20-μm gravity filter placed between the cardiotomy reservoir and the CPB circuit greatly reduces the number of gaseous microemboli.
6. A 20-μm arterial line filter is superior in reducing the number of detectable microemboli coming from the CPB circuit.

Suctioned blood from the thoracic cavity is contaminated with lipid and gaseous microemboli and surgical debris. The contents are dilute with saline and cardioplegia solution, as well as being rich in inflammatory mediators and low in red blood cells. Shunting a fairly large volume of clean blood from the arterial filter and mixing it with the contaminated cardiotomy suction blood almost insures that the contents of the cardiotomy reservoir will have to be returned to the patient. The lipid and gaseous contents of the reservoir also degrade the performance of the arterial filter. Our perfusionists use a venous bag and return blood products through a closed system.

CAN CANNULA DIVERT EMBOLI FROM THE CIRCUIT AWAY FROM THE HEAD VESSELS?

We have performed extensive tests for industry to determine whether changes in aortic cannula can reduce the number of emboli detected in the left carotid artery during simulated CPB and during human coronary artery bypass grafting (CABG) procedures, as well as canine models. We participated in the development of the Cardeon Cobra cannula, which segmented the aortic arch with a physical

barrier that did reduce embolization, as well as provided differential cooling for the head and the body (14). In addition, we have performed extensive testing for the Edwards Embol-X System, which deploys a filter in the aorta to trap emboli during clamping (15). The laboratory has been extensively involved with Medtronic (Medtronic, Minneapolis, MN) in the development of the 3-D cannula that uses an innovative porting system to carry emboli away from the head vessels. The simulated methodology is as follows.

STUDY GOALS

Compare and contrast various cannula designs on the behavior of aortic GMEs.

- Document the behavior of GMEs exiting the cannula into the aortic arch via videotape; i.e., spiraling of GME, aggregation of GME, formation of macro-air bubbles, etc.
- Measure the transit time of GMEs and macroemboli through the aortic arch through videotape.
- Count the number of GMEs that transit the left carotid using the EDAC embolus detection system.

METHODS

A water-glycerol solution [42% glycerol (Sigma Aldrich G7757] with water solution (~30 L) was prepared in a black plastic tub (17 in. width × 30 in. length × 14 in. depth) with a viscosity similar to blood and was circulated through a model aorta, closely resembling the human arterial system. The aorta model was configured using 3/8" tubing with a ½″ tubing segment through the roller head pump (700 MDX, Sarns, Ann Arbor, MI). Hoffman clamps were used to regulate the outflow and pressure of the aortic model to regional physiologic levels associated with CPB. The completed circuit was warmed (36.5–38.3°C) as it was constantly circulated through a Biotherm Heat Exchanger (61399400964; Medtronic) connected to a Sarns Cooler/Heater (11160; Sarns, Ann Arbor, MI). Aortic pressure was monitored from the left iliac artery site. The proximal aortic arch was videotaped during each trial to visualize the distribution of GMEs of each cannula within the arch to the major vessels directed towards cerebral blood flow.

Air (5 mL) was introduced after the roller-pump and 50 cm before the cannula as a rapid bolus or as constant streaming air (30 seconds @ 0.16 mL/s) at flow rates of 4 and 6 L/min. The streaming air was delivered using a Harvard syringe pump connected to the circuit through 60″ small bore tubing (priming volume = 1.7 mL). Streaming air was delayed reaching the circuit because it was necessary for the compressing air to overcome the perfusion pressure of the circuit. The syringe pump was turned off 30 seconds after streaming air began to enter the arch.

GMEs were counted using an embolus detection and classification (EDAC) (16) transducer positioned onto the left carotid ~25 cm distal from the aortic arch (Figure 3).

EDAC data were collected for a 2-minute period either beginning 1 second before each 5-mL air bolus injection or beginning 1 second after the appearance of streaming GMEs within the aortic arch.

We tested 14 different cannula, not all of which are commercially available, and the results were quite revealing. It is absolutely possible to minimize brain embolization with clever cannula design. Any porting is superior to just a straight J, but porting must be done with view toward more than just changing the pressure gradient. There was a >2-fold decrease in the number of emboli detected in the left carotid with the best performing cannula. However, some cannula accomplished this by diverting most of the emboli up the right carotid. Others shredded large bubbles in to many small ones, resulting in higher counts of clinically less significant GMEs. Lipid microemboli handling characteristics are not the same as GME handling attributes. Therefore, we await the outcomes of the clinical trials.

While monitoring emboli counts from the arterial filter, we also documented emboli returning from the venous line. The question was had these emboli completely passed through our canine model? We determined that when the siphon caused the vena cava to collapse around the venous cannula, the system went from being compliant, with the patient essentially being a collapsible bag, to the venous line becoming a fixed volume container. As the siphon created a negative pressure, significant levels of out gassing were shown and replicated in human studies.

Further research is needed to better define the relative contribution of each of the "improvements" brought online by industry and academic investigators. We must continuously question how and why we perform certain actions and whether they are habits from early training that may not be appropriate today.

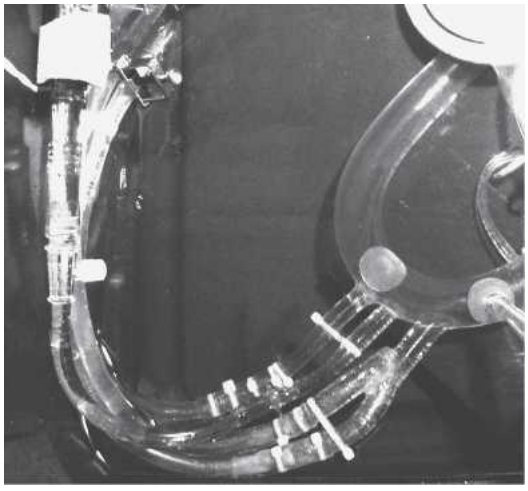

Figure 3. EDAC transducer positioned on simulated left carotid.

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