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OF
CARDIOVASCULAR PERFUSION
515A EAST MAIN STREET
ANNVILLE, PA 17003
(717) 867-1485
OFFICEAACP@AOL.COM
HTTP://WWW.THEAACP.COM

Fall 2022

The Academy Newsletter

Inside this issue

A Message from the President

Fall 2022

Here we are with Labor Day a distant memory, and no one is wearing white, unless you work at the Cleveland Clinic. This is a test to see if any CCF pumpers are reading this. Anyway, it seems like weeks are flying by as fast as days. I have been extremely busy these last few months traveling all over the country. Everywhere I go I try and tell perfusionists I work with about the AACP meeting coming up in Savannah, GA! Hope you are all doing the same. If you have a paper, you would like to submit please do not hesitate to reach out to any member of the AACP. Also, if you or someone on your team is interested in presenting or speaking at a Fireside Chat please reach out to myself justin.resley@gmail.com or the office@theaacp.com

In my current position I have been to many meetings and listened to and spoken to many perfusionists and surgeons around the country. One consistent thing I hear is that perfusion and data are a big part of our patients' outcomes. That might seem obvious to us, but it reminds me of when we didn't have video cameras everywhere. Before video doorbells or cameras, we didn't know what was happening. Like that old saying goes "does a tree make a sound when it falls in the forest if

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there is nobody there to hear it" Now that we have all kinds of technology specific to perfusion data capture, we can hear the tree falling! I recently heard a talk by Luc Puis of the Tiny Perfusion Letter where he spoke of how fast knowledge is doubling. I can't quote him exactly but in the year 1900 human knowledge doubled every 100 years! Now it doubles every 12 months and in the very near future it is expected to double every 12 hours! As we move into the future in our profession, we can expect things to change more rapidly than they ever have before. I would encourage you to come to the AACP meeting and double your perfusion knowledge in just a few days, February 1st – 4th , 2023!

See you in Savannah!

Sincerely,

Justin Resley
President, American Academy of Cardiovascular Perfusion

Important Academy Dates

The ACADEMY ANNUAL MEETING DEADLINES

ABSTRACT DEADLINE	**November 15, 2022**
MEMBERSHIP DEADLINE	**December 1, 2022**
PRE-REGISTRATION	**January 6, 2023**
HOTEL REGISTRATION	**January 6, 2023**
2023 ANNUAL MEETING	**February 1-4, 2023**

Hillary Dressler, BSN, RN, CNOR

Texas Heart Institute
Houston, TX

Hillary Dressler is a perfusion student at the Texas Heart Institute, graduating in December 2022. Her previous experience includes six years as a CVOR Registered Nurse in Evansville, IN where she will return as a staff perfusionist upon completion of the program.

Treating Vasoplegia and the Use of Methylene Blue

Impact on Perfusion

Vasoplegia has become an increasingly prominent issue in cardiac surgery within the past few years. Vasoplegia can become a problem for perfusion throughout the entire operating room stay. The most common times for the perfusionist to be extremely vigilant in communication of vasoplegic issues happens during bypass, weaning and terminating CPB support. Communication with anesthesia for medication administration is vital to combat the issue before it becomes a final effort to save the patient.

When running a hemoconcentrator during a cardiopulmonary bypass case where the methylene blue is given, be aware that methylene blue will come out in the ultrafiltrate, in which not all the drug is getting to the patient. The patient may have a bluish green tinged urine if being treated for methylene blue for vasoplegia.

Review of Literature

Vasoplegia is known under many different names including distributive shock, vasoplegic syndrome, and "Chameleon Shock" (Masud et al., 2021). There is no universal consensus on a clinical definition; however, factors such as: a decreased preload, a low SVR, a high cardiac index, and a supranormal cardiac output can lead to a diagnosis of vasoplegia (McCartney et al., 2017). These patients also have little to no response to vasopressors, even at exceedingly high doses; and sources have evaluated that vasoplegia can be seen in 5-50% of cardiac surgery populations (Arevalo & Bullerwell, 2018). Having a diagnosis of vasoplegia can increase a patients mortality risk by up to 5 times (Mehaffey et al., 2017).

The focus of this paper is vasoplegia experience before, during, or after cardiac bypass surgery, with a small emphasis on cardiogenic shock due to the patient case study evaluated. While all these types of vasoplegia show signs of hypotension, each of these different categories have different mechanisms for the hypotension. Distributive shock, as seen as the most common form found in cardiac surgical patients is pronounced by the decreased preload, low SVR, and supranormal cardiac output; while the hypovolemic, cardiogenic, and obstructive forms are found much less frequently in patients and is evidenced by a low cardiac output and increased SVR (Masud et al., 2021).

While the pathophysiology of vasoplegia is still not heavily understood, there are factors that make a patient more apt to developing distributive vasoplegia. These can include preoperative medication concerns such as the use of ACE inhibitors within a few days prior to surgery, the use of heparin, calcium channel blockers, diuretics, amiodarone, as well as protamine (Fitzsimons & Nussmeier, 2022). Patient health related and comorbidity concerns including dialysis

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dependency or having a recent MI, and having procedural factors: LVAD insertion, valvular dysfunction of sorts, or having pre bypass hemodynamic instability can also put the patient at risk for distributive vasoplegia (Arevalo & Bullerwell, 2018).

Some comorbidities automatically increase a person's risk for developing vasoplegia, with dialysis dependency increasing this risk the highest (Masud et al., 2021). It is important to note, that many dialysis patients also have many of these other comorbidities, increasing the likelihood that they may experience vasoplegia throughout the course of their operation. In terms of procedural risks, VADs increase the risk of distributive shock by twelve and a half, with heart transplants coming in second, and valve replacements increasing a patients risk as well (Masud et al., 2021). Cardiac surgical procedure factors, such as, longer bypass and cross clamp times can play a role in a patient developing vasoplegia (Masud et al., 2021).

As vasoplegia has been studied, different algorithms have been created to try to not only diagnose vasoplegia, but to combat the hypotension aspect, while in turn treating the other signs of vasoplegia. Dr. Chatterjee of Baylor St Lukes Health in Houston, Texas gave a video presentation for the American Society of Thoracic Surgeons on vasoplegia and how vasoplegia is treated in the CV recovery ICU (Chatterjee, 2020). A patient with hypotension is usually given norepinephrine or vasopressin to try to constrict the vessels and increase the blood pressure. When a patient has refractory hypotension to these two medications, other avenues to stabilize the patient should be considered as the patient may be experiencing vasoplegia. The next treatment option can include calcium, gluco and mineral corticoids, ascorbic acid, hydroxocobalamin, and methylene blue (Chatterjee, 2020). Angiotensin II is also considered when the patient is still experiencing vasoplegic symptoms (Chatterjee, 2020).

This paper will focus on methylene blue as a treatment option for vasoplegia. Methylene blue is a nitric oxide synthase - (NOS) and guanylate cyclase inhibitor; vasoplegia is thought to be contributed from the NOS and cyclic guanosine monophosphate (the cGMP) pathway) (Petermichl et al., 2021). Several studies have found methylene blue improves the hemodynamics of vasoplegia related to endothelial dysfunction (Levin et al., 2009). For distributive vasoplegia, the most common dosage would be an IV bolus of 1-2mg/kg over anywhere from 10 to 60 minutes; with the possibility of a continuous infusion of 2mg/kg/hour for up to 72 hours to achieve hemodynamic stability and endothelial function (Leyh et al., 2003).

Cardiopulmonary bypass propagates the inflammatory response by the blood contact leading to the production of inflammatory activation: including tumor necrosis factor, and interleukins 1 and 6 (Masud et al., 2021). These are cytokines that increase the nitrous oxide synthase leading to increased regulation of nitrous oxide, which leads to nitrous oxide induced vasoplegia and hemodynamic dysfunction of the endothelial cells in the vessels (Masud et al., 2021). This causes the potassium channel to become ATP sensitive in one pathway, lowering calcium levels, which is the reason calcium is included in the algorithm to treat vasoplegia (Chatterjee, 2020). The decreased calcium and increased cyclic GMP lead to the decreased regulation of the calcium signal in smooth muscle (AKA the myosin dephosphorylation), resulting in endothelial vasodilation in patients with vasoplegia (Masud et al., 2021).

The literature shows that methylene blue is safe to use in vasoplegic patients; however, there are some considerations before administering it in every situation. Methylene blue is a MAOI, so it should not be used with patients taking monoamine oxidase medications or it can cause serotonin syndrome, those with liver issues should be evaluated for the drug as methylene blue is metabolized in the liver and inhibits cytochrome P450 isozymes (McCartney et al., 2017). Although used in many patients with renal issues, it must be evaluated prior to use, as it is excreted in the kidneys (McCartney et al., 2017). Methylene blue should be used with caution for both pregnant patients and neonates and should not be used in G6PD patients due to the decreased ability of the metabolizes to diminish (McCartney et al., 2017).

Dosages are important to ensuring the expected patient results. While very few facilities are known to give doses over the 2mg/kg, toxicity levels are important to know. Dosages greater than

7mg per kilogram can cause hemolysis, methemoglobinemia, chest pain, and hypertension (2017). Dosages greater than 20 milligrams per kilograms can cause intravascular hemolysis, hyperbilirubinemia, and even death (2017). Toxicity can also be seen in the form of cardiac arrhythmias, deterioration in gas exchange, increased pulmonary vein resistance and pulmonary vein pressure, and decreases in renal and mesenteric blood flow (2017).

Conclusion

In conclusion, it is valuable to identify preoperative risk factors for patients who may become vasoplegic. Identifying refractory hypotension and vasoplegic signs and symptoms is also important to begin treatment earlier in the process. Each facility should have protocols in place for hemodynamic instability while on pump and for post bypass. Hopefully in the next few years more clinical research will be done on the pathophysiology of vasoplegia and how it can best be treated; as well, to reveal if methylene blue with continue as a helpful treatment option to maintain hemodynamic stability for patients with vasoplegia.

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THE ACADEMY TO OFFER LIVE WEBCAST AGAIN THIS YEAR

The American Academy of Cardiovascular Perfusion will again be offering a live webcast of our 2023 Annual Meeting in Savannah, Georgia. The General Sessions of the meeting and two Fireside Chats each day will be broadcast in high quality streaming video. There will also be an opportunity for attendees to ask questions, thus qualifying for Category I CEUs from the American Board of Cardiovascular Perfusion.

The National Office of the Academy has moved.

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Mitigating the Effects of Plasma Free Hemoglobin Toxicity on CPB & ECMO

Jonathan Maxwell French
Texas Heart Institute
Houston, TX

How important is it for clinicians to pay attention to plasma free hemoglobin levels post-cardiopulmonary bypass (CPB) and during ECMO? I'd argue that toxic levels of plasma free hemoglobin have contributed to a lack of stability in many patients following CPB. Plasma free hemoglobin may promote a hypertensive and hypercoagulable state. This is a review of methods perfusionists may employ to mitigate the deleterious influence of plasma free hemoglobin on patients that undergo CPB, ECMO, and cell salvage.

Plasma Free Hemoglobin (PFH) simply describes hemoglobin found in the extracellular space where it usually exists in trace quantities at around 5mg/dL or less. PFH is a known endothelial nitric oxide (NO) scavenger and a source of oxidative stress. At concentrations exceeding 10mg/dL, the potent inhibition of nitric oxide mediated vasodilation is observable. (8) Elevated PFH may be caused by several scenarios ranging from Sickle Cell Disease, intense exercise, and CPB. CPB-related hemolysis caused by non-endothelial surface contact and mechanically induced red blood cell (RBC) lysis account for the marked increase in PFH both during and post-bypass. Furthermore, over-occlusive roller heads, high negative pressure from vacuum assist, cell salvage, and sheer stress from blood flowing throughout the circuit all contribute to the post-bypass apoptosis of sub-lethally damaged RBCs. Post-bypass apoptosis is delayed which is indicated by the sharp increase in PFH which usually peaks around two hours of reperfusion. (1) This was shown in a study that included 54 patients with bypass times that ranged from 1 to 6 hours. (1) The average PFH measured two hours after reperfusion began was 22.5 mg/dL. Keep in mind that a PFH measurement above 10 mg/dL is considered PFH toxicity.

The adverse effects of PFH toxicity mainly stem from PFH's ability to decrease nitric oxide bioavailability. PFH is capable of scavenging NO approximately 1000x faster than hemoglobin found intracellularly (2). Nitric oxide is our most important endogenous vasodilator. The decreasing bioavailability of NO is a major issue when considering higher degrees of hemolysis. Thus, PFH toxicity can lead to microcirculatory disfunction. The rapid loss of endothelial NO Stores can develop into a hypertensive, hypercoagulable, and proinflammatory state (2). Hemolysis also results in the release of an enzyme called arginase 1. Arginase 1 converts l-arginine, the substrate for nitric oxide synthesis, to ornithine. In this way, hemolysis not only causes scavenging of nitric oxide but also theoretically prevents new nitric oxide formation. (1) The acute microvasculature damage caused by the increase in hemolysis can cause damage to several organs including the kidneys and brain. (3)

So, what can the perfusion team do to reduce hemolysis? Shorter duration of CPB, shorter tubing lengths, and less negative pressure utilized by cell salvage/suckers are all relatively simple methods to reduce hemolysis. Also, avoiding allogenic blood transfusions as much as possible is another

Max is a graduate of Texas Tech University, prior to perfusion school at Texas Heart Institute he was a perfusion assistant in San Angelo, Texas. Max will graduate from THI in December 2022 and will join the perfusion team at University Medical Center in Lubbock, Texas.

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subject to consider concerning autoimmune hemolytic anemia. (1) Reduction of hemolysis on ECMO involves avoiding venous line chatter and staying below 3,000 RPMs in the pump head. If the venous line chatters or is momentarily kinked while a high rotation speed is set (> 3,000 rpm), negative pressure within the pump head can exceed –700 mm Hg which can cause cavitation and severe hemolysis. To fix this issue, reduce the pump speed and correct the triggering factors which may include hypovolemia, kinked venous line, or venous cannula positioning. (5) If pump head thrombosis is suspected via a considerable rise in LDH & PFH, replacement of the pump head is imperative. (5)

The treatment options for PFH toxicity are quite varied and some are still experimental. Nitric oxide inhalation is already a treatment for sickle cell patients in crisis because it increases bioavailable NO and oxidizes PFH into methemoglobin which inactivates its pro-oxidant nature. This method can also stimulate intrapulmonary NO formation. (1) Nitrite supplementation is another method. Nitrite can become a NO donor during acidic and hypoxemic states . (1) Human haptoglobin and hemopexin administration have also been proposed as methods of sequestering and neutralizing PFH and free heme. Haptoglobin is a plasma protein primarily produced by the liver and is responsible for inactivating PFH from lysed red cells in vivo. Due to haptoglobin levels being depleted in the presence of large amounts of PFH, decreased haptoglobin is another possible measurement of hemolysis. A study in Japan showed that six grams of haptoglobin added to CPB prime solution showed a marked decrease in renal tubular enzymes, a marker for renal damage. ($p<0.0.5$) (6) Haptoglobin is difficult and expensive to manufacture which has limited its clinical viability. A human-plasma derived haptoglobin product was produced by the Benesis Corporation. In 2013, it cost $540 for a two-gram bolus. (2)

One method that has significant appeal is administering NO via oxygenator while on CPB. A study published in 2020 concluded that NO administration at 40ppm via oxygenator in patients at moderate risk of renal complications undergoing elective cardiac surgery with CPB was associated with a lower incidence of acute kidney injury. (7) However, this method does not reduce PFH, it only increases the amount of bioavailable NO. A concerning aspect of this method is the high price of nitric oxide gas.

Plasma free hemoglobin toxicity post-CPB and ECMO continues to contribute to multiple pathologic states due to its ability to reduce bioavailable NO. Although several methods are currently in use to mitigate this issue, an appealing method is the administration of NO via oxygenator while on CPB. This method

does have its limitations, and further considerations should be addressed in the future concerning PFH toxicity and the bioavailability of endogenous nitric oxide.

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Abstract Deadline for the 2023 Meeting has been extended to November 15, 2022.

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44th Annual Seminar of The American Academy of Cardiovascular Perfusion

The Desoto Savannah

15 East Liberty Street
Savannah, Georgia
February 1-4, 2023

(Tentative Program)

Wednesday, February 1, 2023

11:00 AM – 4:00 PM	REGISTRATION
3:30 PM - 4:00 PM	Opening Business Meeting *Fellow, Member, Senior and Honorary Members*
4:00 PM – 7:00 PM	Manufacturers' Breakout Rooms

Thursday, February 2, 2023

7:00 AM	REGISTRATION
7:00 AM – 8:00 AM	Video Presentations
8:00 AM – 09:30 AM	Scientific Paper Session
9:30- AM – 11:30 AM	Fireside Chats *Everything ECMO* *Myocardial Preservation from the OR to DCD* *Pediatrics* *Perfusion Accidents* *Student Only Forum*
11:30AM - 1:00PM	Lunch (Historical Videos)
1:00 PM – 3:00 PM	**Special Scientific Panel Session**
3:00 PM – 3:30PM	Break
3:30PM – 5:00PM	**Special Scientific Panel Session**
06:00PM	Sponsor's Hands-On Workshop & Reception

Friday, February 3, 2023

7:00 AM	REGISTRATION
7:00 AM – 8:00 AM	Video Presentations
8:00 AM – 9:30 AM	Scientific Paper Session

9:30- AM – 11:30 AM	Fireside Chats *Pediatric ECMO* *Pediatrics* *Simulation: From Low to High Fidelity* *What they didn't teach us in school.* *Women in Perfusion*
11:30AM - 1:00PM	Lunch (Historical Videos)
1:00 PM – 3:00 PM	**Special Scientific Panel Session**
3:00 PM – 3:30PM	Break
3:30 PM – 5:30 PM	Memorial Session ***Charles C. Reed Memorial Lecture*** ***Thomas G. Wharton Memorial Lecture*** *(Justin Resley, CCP)*
6:30 PM	Induction Dinner *All Attendees and Guests*

Saturday, February 4, 2023

7:00 AM	REGISTRATION
7:00 AM – 8:00 AM	Video Presentations
8:00 AM – 9:30 AM	Scientific Paper Session
9:30 AM – 10:00 AM	Break
10:00 AM – 11:30 AM	**Special Scientific Panel Session**
11:30 AM – 1:00 PM	Lunch (Historical Videos)
1:00 PM – 3:00 PM	**Special Scientific Panel Session**
3:00 PM – 5:00 PM	Fireside Chats *Dealing with stress, finding work/life balance, team building and communication* *ECMO and VAD challenges: scenarios and transports* *Electronic Medical Records: The good, the bad, and the glitchy* *Industry and CCP*
5:00 PM	Closing Business Meeting *Fellow, Senior and Honorary Members Only*

There will <u>not</u> be any on-site registration in Savannah this year so please pre-register.

2023 AACP Fireside Chats

Thursday, February 2, 2023

Student Only Forum
Students only, a forum to meet and greet

Pediatrics
An open forum to discuss standards of care and new practices in the field

Myocardial Preservation from the OR to DCD
Protecting the heart is no longer just every 20 minutes.

Everything ECMO
Collaborate with colleagues about the intricacies of what goes into all parts of ECMO.

Perfusion Accidents
If you can think of it, it has either happened to someone else or will to you. Let's share and learn.

Friday, February 3, 2023

What they didn't teach us in school.
Round table discussion on situations you never anticipated after graduation, and how to navigate

Pediatric ECMO
What's new, what's not and what struggles do we face as the specialty moves forward.

Women in Perfusion
Collaborate with some special situations and challenges of other women in the field.

Simulation: From Low to High Fidelity
See what other centers are doing to build and grow this important technique.

Pediatrics
Open forum to discuss what centers are doing, standards of care, and any new topic as well

Saturday, February 4, 2023

Electronic Medical Records: The good, the bad, and the glitchy
See how centers are tackling and incorporating this evolving standard of care

Industry and CCP
What it's like to go from clinical to industry, combine both, and maybe back again with all the challenges faced.

Dealing with stress, finding work/life balance, team building and communication
How to find a balance in a high stress work environment

ECMO and VAD challenges: scenarios and transports
Let's learn from each other as transports (intra and inter hospital) are becoming their own new specialty.

2023 Annual Meeting

Savannah, Georgia
February 1-4, 2023

Our Host Hotel
The Desoto Savannah
15 East Liberty Street, Savannah, GA, 31401

Reservations: 800-239-5118

Single/Double Occupancy: $199.00

Remember to mention that you will be attending the Annual Conference of The American Academy of Cardiovascular Perfusion (AACP).

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