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Winter 2019

The Academy Newsletter

The Roaring 20's Invade the 2020 AACP!

Please join us for The 41st Annual Seminar of the American Academy of Cardiovascular Perfusion being held in Reno, NV Feb 5 -8th, 2020 at the Grand Sierra Resort. Pre-meeting registration and hotel registration ends January 9, 2020.

Scientific sessions will be filled with exciting topics spanning all genres and guaranteed to interest all. Don't miss out on the ever popular interactive Fireside Chats. We are offering some exciting new topics this year to include Low fidelity simulation-buckets, hoses & duct tape: Making the most of what you've got, Pump On: the first five years, as well as Pump Off: making the most of the last 5 years! We also have two specials guests, Dr. Ross Ungerleider, and Jamie Dickey Ungerleider, PhD, who will be co-moderating along with Ian Shearer a fireside chat on "Dealing with stress, finding work/life balance, & communication". This session promises to have you on the edge of your seats AND walk away with some take-away tools to utilize in your daily life. We have a very unique Friday afternoon session planned that you will not want to miss. We'll start with a special session called "Heart Matters: Life beyond the Pump Run", where you

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will hear extraordinary stories of courage. Come meet our featured motivational speaker, Craig Cunningham. What you may not know is that one thousand people die ***each day*** from sudden cardiac arrest. On Nov 19, 2016, NHL hockey player Craig Cunningham was nearly one of them. *Craig Cunningham suffered a cardiac arrest on the ice before a hockey game with the Tucson Roadrunners. With the heroic efforts from medical experts and world renowned cardio-thoracic surgeon, Dr. Zain Khalpey, Craig defied the odds and fought his way back to life. Now, Craig and Dr. Khalpey want to make a difference and created a foundation with the goal of preventing sudden cardiac arrest.* Please visit the website (https://www.allheartfoundation.org) to learn more and see how you can help.

This will be followed by Aimee Mooney sharing her perspective on the joys and challenges of raising a child with HLHS. Finn Mooney, now 18, will be joining his mom in sharing their story.

Topping off the early afternoon session will be Ross Ungerleider and Jamie Dickey Ungerleider. They will be speaking on Integrating Life Skills for Creating Resilience to Burnout and Reconnecting with Joy in your Personal and Professional Life.

We are honored to have Dr. Ross Ungerleider as our 2020 Memorial Reed Lecturer. Dr. Ungerleider has been an incredible supporter of perfusion education and has been an unwavering friend to perfusion spanning the 41 years of his surgical career. His talk is titled "That's How the Light Gets In".

And if you aren't already excited enough, there's more! As you may have seen on our website and social media posts, **The Roaring 20's Invade the 2020 AACP** with a **Boardwalk Empire themed Sponsors' Workshop** on Thursday, February 6th and **The Great Gatsby Gala Induction Dinner** on Friday, February 7th! Both evenings will be chock-full with fun. The focus of the Sponsor's Workshop will be on multigenerational teams bonding with each other and encouraging generous face time with sponsors. Without revealing too much, casino games will be incorporated into this event! All participants are encouraged to wear 1920's and/or Boardwalk Empire themed attire if so desired. This can be as simple or "decked" out as you prefer. See the posted flyer (Visit Link) on the Academy website for examples.

This is not to be outdone by the Induction Dinner on Friday night. This will still be kept in a formal format with the added **1920s or Great Gatsby themed attire for the Gala Induction Dinner!** Enjoy the strolling magician performing sleight of hand manipulations during cocktail hour followed by a DJ and dancing after dinner. Just a reminder that the Friday night Gala Induction Dinner is open to **ALL** meeting attendees. Please remember to RSVP during registration. If you neglected to check the RSVP box, it is not too late to attend. Please just email David Palanzo, office@theaacp.com

As I look back at 2019, I am reminded that the only constant in life is change. Change is often met with trepidation about the uncertainties that is brought about. However, if we embrace it, change can lead to exciting new opportunities that we never thought possible. The goal of my presidency year was to encourage volunteerism, encourage more participation and inclusiveness, and increase transparency. This cannot be done without being open to change and trying new approaches. I believe we have taken several steps in this direction. We are continuing with improvements of the AACP website, adding additional features for council, increasing transparency and accountability among fellows and members by posting meeting attendance and participation. The goal is to assist all fellows and members to more easily track their activities. Our hope is that this will encourage increased participation and inclusion of all members

and invite new opportunities for those who may not be as comfortable being vocal early on.

We had a great response to the **"The AACP SEES YOUR VALUE"** volunteerism flyer written by Ashleigh LeBlanc. Alex Gum wrote a terrific informative three-part series on uses of social media to increase our social media presence and help attract our younger generation of perfusionists. Ashleigh and Alex are two examples of academy members who have risen to the occasion and used their talents to become more involved in the academy, and they have set the bar high! Allison Weinburg has been a rock star as the new chair of the Fireside Chat committee this year. We've had an infusion of enthusiasm by several new fellow members and re-engagement of seasoned fellows working together. I would be remiss in not mentioning co-chairs of the Social Media/IT Committee, Christine Chan and Kenny Fung; Student Liaison Chair, Molly Bryant; Membership Chair, Vince Olsholve; Sponsor's Committee Chair, Rich Melchior; the Awards/Manuscript Committee, and ALL committee members in their efforts. Finally, I owe a special debt of gratitude to the entire Program Committee; Bill Riley, Tami Rosenthal, Tom Klein, Allison Weinburg, Greg Smigla, Rich Melchior, Bob Grimmett, Desiree Bonadonna, Molly Bryant, Michael Brewer, Alex Gum, Dave Fitzgerald, Jimmy Beck, Justin Resley, Karen Smith, Mat Tyndal, and Ashleigh LeBlanc, for working tirelessly on what will be an EPIC meeting this year.

Wishing you all a safe and happy Holiday Season. See you in Reno!

Carmen Giacomuzzi
President, AACP

THE CRAIG CUNNINGHAM

ALL HEART FOUNDATION

TO PROMOTE PREVENTION AND SMART SCREENING TO PREVENT SUDDEN CARDIAC ARREST

Craig Cunningham suffered a cardiac arrest on the ice before a hockey game with the Tucson Roadrunners. With the heroic efforts from medical experts and world renowned cardiothoracic surgeon, Dr. Zain Khalpey, Craig defied the odds and fought his way back to life.

Now, Craig and Dr. Khalpey want to make a difference. This foundation was created with the goal of preventing sudden cardiac arrests. With our cutting edge technology and your help, we can all make a difference and start saving lives.

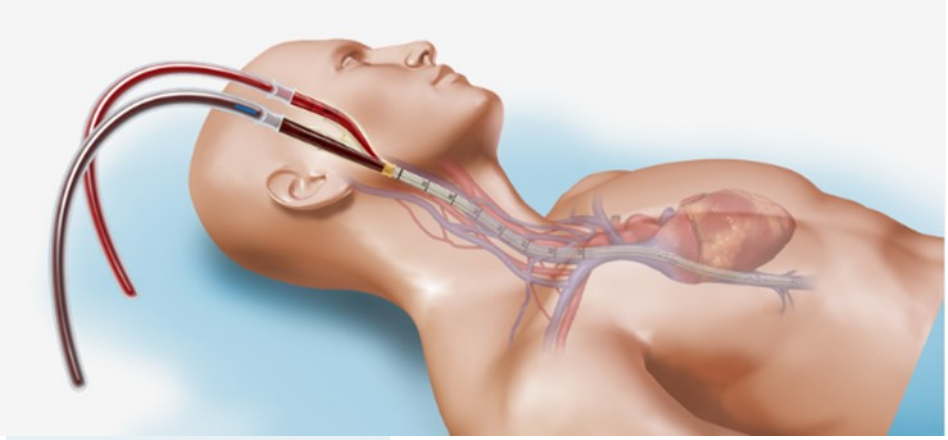
Designed for ECMO, Why Long-term Testing is Important

Rio Foster
Vice President of Marketing
MC3 Cardiopulmonary

Extracorporeal Membrane Oxygenation (ECMO) is the most advanced form of life support, allowing gas exchange through blood circulated outside of the body, in place of, or in combination with, conventional ventilation via the airway. When a physician prescribes ECMO, the clinical team unites to safely initiate the therapy. Physician may order ECMO (as illustrated by reports from the ELSO Registry and the FDA indication) for a broad set of indications with expected durations ranging from days to weeks.

Historically, ECMO has not been administered utilizing equipment repurposed from adjacent therapies such as Cardiopulmonary Bypass (CBP). These devices have been critically important to the evolution of ECMO. Some of the biggest innovations have come from the advancement of material science such as the development of poly-methyl pentene (PMP) fiber, which is ideal for ECMO and not especially suitable for CPB. Essentially all oxygenator assembly components, from connectors, to housings, to their flow path orientations were initially designed for CPB and repurposed for ECMO by adding PMP.

Our collective mission in the medical device industry is to bring forth meaningful innovation clinicians can use to save lives. Designing ECMO products for their intended clinical use will drive development processes that adequately test for long-term duration and follow important verification and validation protocol. The additional level of testing, not required for adjacent technologies, will drive safer, more effective technology for patients suffering from acute respiratory and cardiac failure.

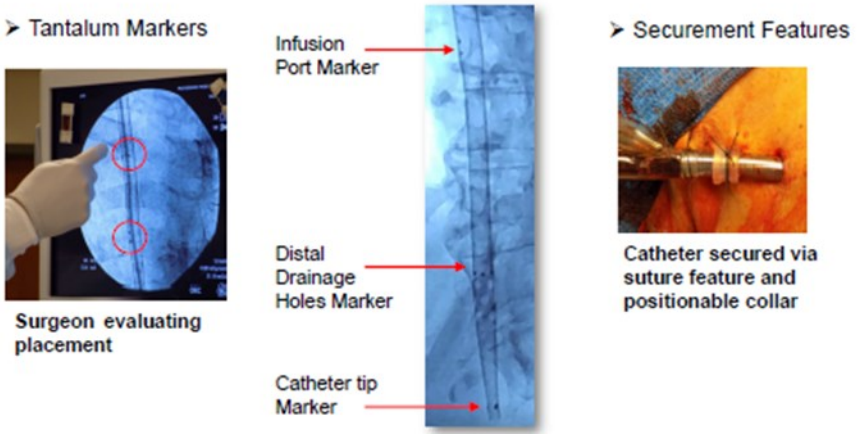


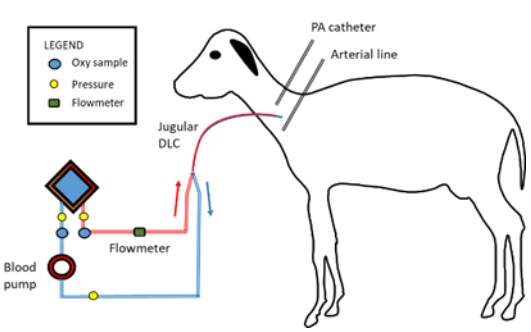
By basing the design process based on the intended clinical use, both the needs of the patient and user become integral to the development process. ECMO is lifesaving, but also a procedure of last resort. The duration of support may vary from days to weeks and beyond. The extended use of ECMO devices poses additional risk factors that need to be addressed in the design, verification and validation process. International standards for long-term testing and the FDA's final order for the reclassification of ECMO devices outlines the process for mitigation measures to address identified risks of ECMO devices. Substantial equivalence analysis includes comparison to the special controls, 81 FR 7451, Feb. 12, 2016, as well as comparison to a Secondary Clinical Reference Device if no device has been cleared under regulation 21 CFR 870.4100 to date.

The first device cleared by the FDA and marketed in the United States under the new indication for ECMO was the Crescent® Jugular Dual Lumen Catheter. MC3 Cardiopulmonary developed an extensive testing program to demonstrate that the performance of devices designed for ECMO sufficiently satisfies the needs of the users, demonstrates that the devices are safe and effective, and mitigates as far as possible all

risks identified for ECMO devices. Crescent was designed using best in class engineering and preclinical studies to verify and validate the design for extended ECMO use, and the rigorous process of testing for its intended purpose made it a better device.

The development team began by interviewing users all over the world, working with them to map their clinical workflow and identify the biggest limitations to the tools they currently used. Using these inputs, User Needs were created which later turned into device requirements that were verified and validated in a series of preclinical studies. Risks associated with dual lumen ECMO catheters were identified through the FDA identified health risks, complaint history in the FDA MAUDE database, voice of customer and extensive literature review. Design features were incorporated into the catheter to address known shortcomings in current products, including improving flow performance, additional kink resistance, visualization markers, added tip length and securement features to avoid placement and migration issues. In addition, materials and manufacturing processes were selected for long-term biocompatibility and durability. The new reinforced wire and dipping processes created a catheter that is flexible and kink resistant for long-term use.





The design concept was confirmed through a series of initial confidence studies, including engineering bench studies, a feasibility cadaver lab, and a usability in vivo study. Full design verification studies were conducted on finished, conditioned, sterile devices. Additional testing beyond that typical for adjacent technologies (e.g. CPB) included simulated use testing for 30 days, biocompatibility testing for prolonged use (24 hours to 30 days), as well as a multi-day, in vivo study to demonstrate that the catheters were safe for ECMO use. Final validation studies in a cadaver model by experienced surgeons, intensivists and allied clinicians on the ECMO team were used to demonstrate that all user needs were satisfied.

Designing ECMO products for their intended clinical use will drive development teams to adequately test for long-term duration and follow important verification and validation protocol. The additional level of testing, not required for adjacent technologies, will drive safer, more effective technology for patients suffering from acute respiratory and cardiac failure. With the widespread adoption of Crescent throughout the US and positive feedback from its users, the clinical acceptance supports the underlying premise that good development practice with rigorous testing for its intended purpose can create better, safer devices ready for clinical use.

BLASTOMYCOSIS: PATHOPHYSIOLOGY, MANAGEMENT AND TREATMENT OF A UNIQUE EPIDEMIC DISEASE IN NORTH AMERICA

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While most endemic diseases are directly correlated with environmental factors or climate factors, there are also others that do not demonstrate the aforementioned factors. One such example is that of blastomycosis, also known as North American blastomycosis, which is a fungal infection primarily associated with the soil in the Great Lakes Basin, encompassing territory from Southern Canada throughout most of the Midwest United States [1][2]. The clinical manifestations of pulmonary blastomycosis range from asymptomatic infection to severe pulmonary infection, which can lead to the development of acute respiratory distress syndrome (ARDS) requiring the use of extracorporeal membrane oxygenator (ECMO) [5]. In addition, pulmonary blastomycosis can also cause extrapulmonary manifestations, also known as disseminated blastomycosis, which in turn can lead to infections within the skin, bone, and central nervous system [3][4]. The pharmacological treatment of pulmonary and disseminated blastomycosis is directly dependent upon the degree of severity, the host immune status, and whether the central nervous system has been implicated. This paper will explore the new guidelines in regards to the treatment of blastomycosis, as well as the mechanisms of action of the drugs involved in its treatment.

A disease unique to the Midwest yet little known among its residents, the geographical region impacted by blastomycosis extends eastward along the south shore of the St. Lawrence River Valley, southward along the central Appalachian Mountains in the east, and to the Mississippi River Valley in the west (Figure 1) [4].

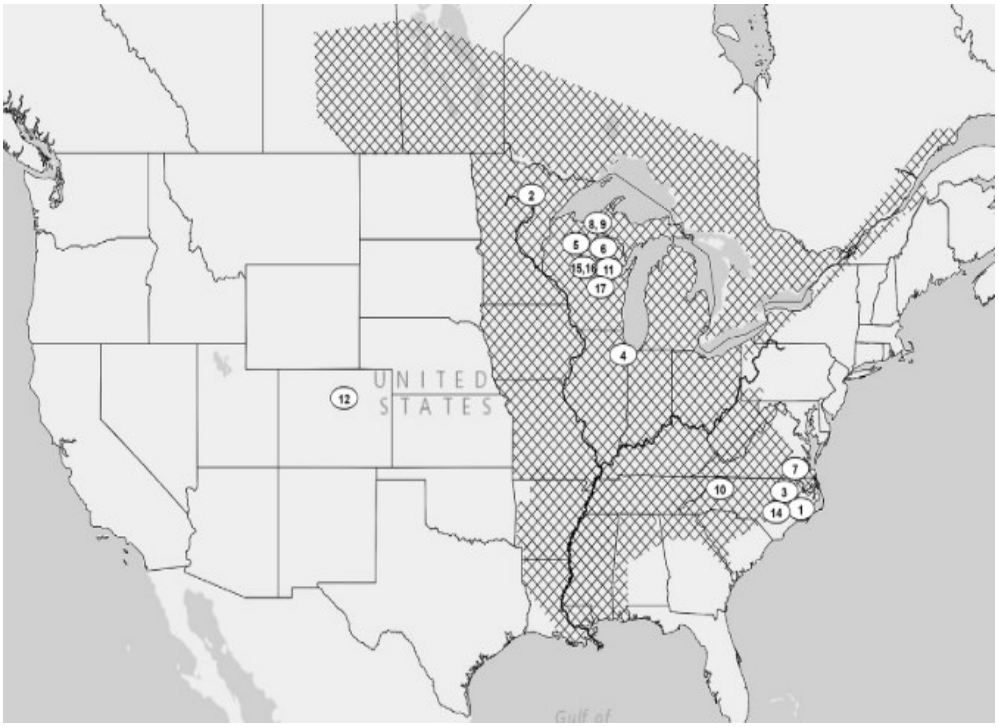

Figure 1 – Map of Distribution of Endemic Blastomycosis in North America. The numbers represent the distribution of the cases [4].

Blastomycosis is caused by dimorphic microfungus blastomyces *dematitidis*, a member of the *ascomycota* phylum, within the *ajellomycetaceae family* [7][8]. In order to begin to explore how blastomycosis is treated, it is necessary to first explore the pathogenesis of this infection. Recent analyses categorized blastomyces into either the *blastomyces dermatitidis* category or the *blastomyces gilchristii* category, which are both present in the soil and are aerosolized during activities that cause soil disruption [3][4][6]. When blastomyces are present within the soil, they undergo critical transformations when soil temperatures reach 22°C to 25°C, and later begin to produce infectious spores known as conidia. Conidia can be converted into yeast when exposed to temperatures ranging from 35°C to 37°C, which is what occurs in affected patients when inhaled [3][4]. When the conidia is inhaled, it is phagocytized by lung macrophages and neutrophils, and the portion of the conidia that survives at phagocytosis matures into yeast, as the lungs provide the perfect environment for conversion [3]. The transformation of the conidia into yeast is crucial, as it also causes a change in the composition of the cell membrane, increasing the amount of α (1-3) glucan within the cell wall [3][4]. In addition, blastomycosis yeast have high virulence and immune evasion from the innate and adaptive immune system. The transformation of the conidia into yeast allows the infection to transcend into the lungs parenchyma and vascular system, which in turns allows it to spread to many other vital organs. Also, when blastomycosis presents in the form of yeast, it is also able to inhibit host cell cytokine production, impair $CD4^+$ T-lymphocyte activation, and suppress nitric oxide production [3][4]. It is also important to note that blastomycosis yeast is resistant to reactive oxygen species produced by neutrophils and macrophages. For this reason, blastomycosis yeast is very difficult to treat within the human body, and it often causes organ damage in those affected ranging from asymptomatic subclinical infection to fulminant pulmonary infection [3][4].

The lungs are the primary entry point for the blastomycosis conidia, and pulmonary infection is typically reported in more than 79% of all documented cases [3]. Generally, the incubation period can vary from two to six weeks, during which time patients are asymptomatic. On the other hand, when patients begin to exhibit symptoms there is a high risk the patient has developed extrapulmonary dissemination, which generally involves an infection in the skin, bones, genitourinary tract, and central nervous system [3][4]. The spectrum of pulmonary infection can vary from subclinical pneumonia to ARDS. Although the treatment of pulmonary blastomycosis is highly case dependent, the Infectious Diseases Society of America (IDSA) recommends that all affected patients receive treatment, specifically antifungal therapy, regardless of the presence of symptoms [7]. Hospitals generally adopt two different treatment protocols in the treatment of blastomycosis: one for patients with mild or moderately pulmonary or disseminated blastomycosis, and a second protocol for patients with moderate-severe or severe pulmonary or disseminated blastomycosis with symptoms [3][4][7]. Generally, the first-line treatment for mild and moderate forms of pulmonary or disseminated blastomycosis is the use of the drug itraconazole, although it can be substituted by the less active drugs such as fluconazole or voriconazole. Differently, moderate-severe to severe forms of pulmonary or disseminated blastomycosis, as well as cases involving the central nervous system are generally treated with the administration of amphotericin B, which can be substituted with itraconazole or voriconazole in case of intolerance to itraconazole [4][7]. Pulmonary blastomycosis represents both a diagnostic and therapeutic challenge, due to its non-specific and wide spectrum of illness that can vary from asymptomatic infection to acute respiratory distress syndrome. The development of pulmonary blastomycosis in ARDS frequently is treated with specific antifungal drugs coupled with the use of ECMO [5]. The IDSA guidelines released in 2008 highly recommend the use of lipid amphotericin B as a first-line treatment for patients with moderate-severe to severe pulmonary blastomycosis, as well as several triazole drugs for patients with mild to moderate blastomycosis [7]. Despite more than fifty years of documented outbreaks, the IDSA guidelines are only based on clinical experiences, de-

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scriptive studies, and reports of expert committees. A clinical trial has not yet been conducted to develop deeper knowledge of the infection, mainly due to the infrequency of outbreaks and small population size affected [7]. Despite the rarity of outbreaks, further research must be conducted to improve treatment plans, due to the relatively high mortality rate ranging from 4% - 6% associated with the infection [10]. In fact, the mortality rate can reach up to 89% in patients with ARDS, even when the appropriate antifungal treatment is received [10]. Continued research on blastomycosis and its therapeutic treatments is promising, and it will remain a fascinating infection due to its rarity and localization to a contained region of North America.

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THE ACADEMY TO OFFER LIVE WEBCAST

The American Academy of Cardiovascular Perfusion will again be offering a live webcast of our 2020 Annual Meeting in Reno, Nevada. The General Sessions of the meeting and one Fireside Chat 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.

There will not be any on-site registration in Reno this year so please pre-register. We have extended the deadline to January 12, 2020.

SUSPECTED HEPARIN INDUCED THROMBOCYTOPENIA AND ECMO PATIENTS, CONSIDERATION OF CVVH FILTER

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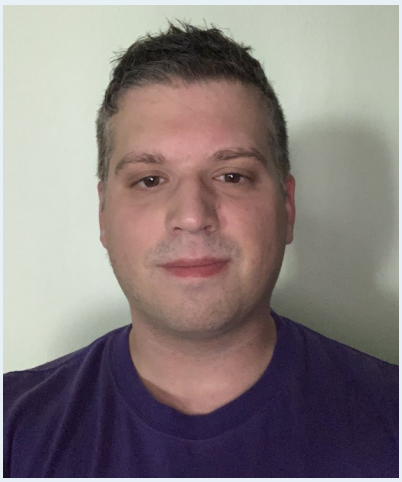

Platelet counts drop expectedly following cardiac surgery and cardiopulmonary bypass. More complex and critically ill patients may require extracorporeal membrane oxygenation (ECMO) from failure to wean from the cardiotomy or from some sort of respiratory condition unrelated to a postoperative period. The stress due to the patient's condition and comorbidities may also lead to the acute renal failure requiring use of continuous veno-venous hemofiltration (CVVH). Considering the patient is being supported by both an ECMO circuit and CVVH, platelet counts decrease. Such a large drop in platelet count can lead to suspicion of heparin induced thrombocytopenia, as the ECMO circuit requires some form of anticoagulation and the patient possibly is recovering from a procedure involving cardiopulmonary bypass using heparin [1–3].

Thrombocytopenia in the critically ill is common and can occur due to decreased production, consumption, or destruction of platelets. Naturally, a patient on ECMO checks many of the risk factors for thrombocytopenia including, sepsis, bleeding, various medications, hemodilution, the ECMO circuit, and possibly heparin induced thrombocytopenia [4].

Heparin induced thrombocytopenia (HIT) is a severe anti-body mediated reaction leading to thrombotic state and greatly increased morbidity and mortality. In addition it causes a longer ICU stay and failure to wean from ECMO [5]. While it is often suspected, it is rarely confirmed. Various retrospective studies of patients on ECMO showing significantly less confirmed cases in relation to suspected cases [5,6]. Heparin induced thrombocytopenia is believed to only affect approximately 0.5-3% of individuals receiving unfractioned heparin, however it is suspected far more often [7].

While it sounds simple in concept, a patient has a positive PF4 or serotonin release assay and the care team switches the patient over to a direct thrombin inhibitor and the patient is able to eventually recover, it can be more difficult. Serological tests for HIT are not resulted immediately. Thus, if a patient were to be suffering from HIT while still on heparin the consequences could be disastrous. A direct thrombin inhibitor is also a finite and expensive resource and waste results in an astronomical cost to hospitals. Argatroban waste can generate a cost of up to half a million in pure drug waste alone of unused medication, not including unnecessary use [8].

A large decrease in platelet count can be an indicator of heparin induced thrombocytopenia, requiring the immediate removal of heparin and replacement with a direct thrombin inhibitor such as argatroban [9]. These drugs are irreversible and more expensive, as well as often being instituted prior to detection of PF4 antibodies from laboratory tests. Argatroban, because of its clearance by the liver, makes it a great choice in a patient with compromised renal function [2]. Though it must be considered that argatroban is costly, and quite often a great amount of it is wasted placing extra cost on the patient and hospital [8].

Complicating this dilemma for physicians determining an anticoagulant to use on ECMO, is when a patient is on CVVH. Literature has shown that there could be a drop-in platelet count for a patient just undergoing a renal replacement therapy as high as 48%, which would immensely complicate the potential determination of a significant drop in platelet count for a patient on ECMO [10–12]. A patient on just CVVH may not be exposed to any

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heparin at all and still express a large decrease in platelet count, which is why it would be beneficial to compare this drop-in platelet count to those that could be seen in a patient being treated with ECMO. The culprit has been found to likely be the CVVH filter, which causes a decrease in platelet count almost instantly and increases over time [12]. I plan to investigate this connection between the CVVH filter and the ECMO circuit through a retrospective study for my thesis project.

When looking at the ECMO circuit in itself, a 2016 study showed that the duration of ECMO had no discernable impact on platelet count whatsoever, and that drops in platelet count are merely associated with severity of illness and the platelet count at time of cannulation [13]. Another study from 2015 examining 119 patients on ECMO showed that suspected HIT occurred in 19% of the subjects with only one having a confirmed laboratory diagnosis [5]. HIT is rare, but the fear of it occurs often, resulting in alternative treatments being used that may not be necessary.

Overall, it is worth noting that critically ill patients being treated with ECMO may have other devices that can substantially impact anticoagulation from a perfusionist perspective. It could be worth it to increase awareness of other forms of extracorporeal devices. It is important to be cognizant of the entire patient's plan of care and recognize that a significant drop in platelet count is worth investigating, but to consider all potential causes.

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Considerations for Renal Protection and Management: CPB

Renal impairment is frequently a complication of CPB, often associated with unfavorable prognoses for patients. Depending on how renal failure is defined, incidence varies between 2.5% to 30% of all cardiac surgery patients [8]. In patients that develop acute kidney injury (AKI), the mortality rate can be as high as 60%, and about 1% of all routine coronary artery bypass grafting (CABG) patients require dialysis [3,12,16]. Moreover, renal impairment post-CPB has a complicated and multifaceted etiology. Even with the progress made in its management, post-CPB renal impairment persists. By identifying high-risk patients, using preoperative lab values, and improving protective and therapeutic measures, perfusionists can effectively detect and manage CPB related renal impairment.

Several models for examining high-risk patients developing renal failure post-CPB have been created [3,12,16]. The studies have pointed to several significant factors improving the ability to predict renal impairment. Possibly the most evident factor for developing post-CPB renal impairment is pre-existing renal dysfunction [11,13]. Renal dysfunction is often noted by elevated serum creatinine. Creatinine is a byproduct of creatine phosphate catabolism in muscle cells. Once filtered in the kidneys, creatine is minimally reabsorbed or secreted. Therefore, serum creatinine can approximate glomerular filtration rate (GFR) – predictive of renal failure. In patients with preoperative renal impairment, the incidence for post-CPB AKI was greater than 20% in addition to a greater than 50% mortality rate [11,13].

Advancing age is also considered a risk factor [9]. Older patients tend to have some degree of senile degeneration of nephrotic mass because of genomic changes that culminate with age [9]. Specifically, vascular endothelial growth factor gene expression decreases over time, increasing the risk of ischemic insult [9]. Furthermore, renal autoregulation is impaired significantly [3]. Renal autoregulation is a myogenic response the kidneys use to maintain GFR over a wide range of blood pressures (i.e., 70-180 mmHg). Patients greater than 70 years of age have a 2-fold increase in risk for renal autoregulation impairment, and those 80 years of age, a 4-fold increase in risk [3].

Another risk factor incurs before surgery when patients have angiograms done. The contrast dyes used for cardiac catheterization increase BUN levels preoperatively leading to azotemia [2,4]. Patients with azotemia are particularly vulnerable to developing post-CPB AKI [2,4]. Many times, surgery is postponed until BUN levels return to normal. Furthermore, contrast agents can also bind to calcium channels in the renal medulla and induce a vasoconstrictive response [2,4]. This potentially puts the patient at risk for upcoming surgery and/or exacerbates any medullary ischemia already present.

Other factors in a patient's chart that should be considered include a history of moderate to severe congestive heart failure (CHF), ejection fraction (EF) < 35%, previous surgeries (especially valvular), peripheral vascular disease, diabetes mellitus (DM), or a history of rheumatic fever [3,12,16]. Many of these factors indicate impaired circulation or cardiac function. Because of this, there is the possibility of decreased renal perfusion and pre-operative renal impairment.

After taking into consideration the patient's medical history and pre-

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Continued from Page 11

operative lab values, protecting the kidneys is the next priority. One important CPB factor affecting renal perfusion is hemodilution. Hemodilution lowers blood viscosity, increasing systemic blood flow, and perfusion [7,10]. Microcirculatory improvement reduces afterload (decreasing shear stress on the arterial side) and increases venous return resulting in an improved cardiac output [7]. This includes improved renal perfusion and urine output. Moreover, hemodilution lowers plasma oncotic pressure, thus maintaining GFR at low perfusion pressures. During CPB, this translates into an increased urine output and decreased creatinine and sodium clearance. By reducing urine osmolarity via hemodilution, renal tubule constitution is protected [7,8,10]. Hemodilution also generates less hemolysis when compared to blood prime [14].

Hemolysis results in hemoglobin release into the plasma [5]. If the level of filtered hemoglobin supersedes the transport maximum for reabsorption, hemoglobinuria may manifest [5]. Precipitate casts from the hemoglobin then form within the renal tubules, altering their ability to function and possibly leading to acute tubular necrosis [5,14]. Albeit, nephrotoxic levels of hemoglobin are rarely reached during CPB [5]. It could be considered, however, in high-risk patients to send cardiotomy suction to be processed (washed and concentrated) via cell saver before returning it to the extracorporeal circuit in an effort to prevent this from occurring.

Lastly, using a crystalloid prime rather than blood prime reduces the incidence of adverse homologous blood syndrome reactions [6]. Historically, homologous blood syndrome was blamed for intraoperative and postoperative bleeding diathesis in addition to post-CPB cerebral, pulmonary, and renal dysfunction [6]. It was thought to be a result of incompatibility from cross-reactions between multiple units of donor blood that mixed together in the extracorporeal circuit during priming [6]. However, now that blood prime is less frequently used, the incidence of homologous blood syndrome and associated renal dysfunction has reduced. Another relatively antiquated CPB protection consideration is the elimination of bubble oxygenation [15]. As membrane oxygenators and filters have become the standard of care, the incidence of emboli has gone down, including any ensuing renal impairment because of this.

Once protective measures have been deliberated, management strategies for patients on CPB that are presenting with renal impairment are considered more closely. Urine output for patients should be 1 cc/kg/hour [8]. This is the simplest indicator for renal function during CPB. It should be noted that variable perfusion pressures, hypothermic techniques, and mannitol in pump prime all commonly alter urine output, possibly making it an inaccurate predictor of renal perfusion [1,8].

Regardless, oliguria needs to be addressed immediately when present [8]. Technical problems should be ruled out first. Patency of the urinary catheter tubing, catheter tip obstruction with gel, or disconnected tubing are all possibilities [8]. Following this, CPB flow rates may need to be increased, ideally maintaining a mean pressure of 65 mmHg with vasodilators and pump flows at 30 to 50 mL/kg to maintain GFR [8]. Drugs that compromise renal function (e.g., phenylephrine, norepinephrine) should be eliminated if possible [8]. Hydration should be maintained by an infusion of a balanced electrolyte solution, ensuring appropriate intravascular volume [8]. If at this point the patient is still producing inadequate urine output, renal vasodilation and diuretic therapy should be considered.

To attenuate renal vascular resistance, dopaminergic agents such as dopamine, dopexamine, and fenoldopam are frequently used. Of the three, fenoldopam seems the most efficacious [17]. Dopamine stimulates both DA-1 and DA-2 receptors which have opposing effects on renal blood flow, sodium, and water secretion, making it difficult to predict how it will interact on a given patient [17]. Dopexamine is less receptor specific (i.e., active at DA-1, DA-2, and β_2 receptors) [17]. Fenoldopam is a specific DA-1 receptor agonist, having minimal effects on systemic blood pressure while increasing RBF, urine output, and decreasing renal vascular resistance [17].

Diuretics can also be given to maintain renal tubular flow [8]. Osmotic diuretics such as mannitol function by raising plasma and tubular fluid osmolarity. Mannitol is a pharmacologically in-

ert sugar with a low rate of metabolism in the body [17]. Mannitol is readily filtered with nominal reabsorption, limiting tubular water and electrolyte reabsorption, thus maintaining GFR and urine output [17]. However, if GFR is severely depressed and tubular necrosis supervenes, the kidney will be unable to form urine even with the osmotic load [17]. At this point, mannitol is contraindicated for because of its capability to cause plasma volume expansion which can lead to pulmonary edema and congestive heart failure [17].

If mannitol is no longer an option, high ceiling (loop) diuretics may be considered (e.g., bumetanide and furosemide) [17]. Loop diuretics function by inhibiting sodium reabsorption in the ascending limb of Henle, proximal convoluted tubule, and distal convoluted tubule, causing additional water and chloride excretions [8,17]. Furthermore, it is active in patients with relatively severe renal failure and has a rapid onset of 2-10 minutes with a duration of 1-2 hours, making it an excellent therapeutic choice in managing AKI [8,17]. While administering loop diuretics, electrolytes should be monitored closely as metabolic alkalosis may result [17].

Renal impairment persists as a complication of cardiovascular surgery and is a substantial contributor to post-CPB mortality. Geriatric patients with preexisting renal dysfunction may never recuperate kidney function following AKI, requiring a renal replacement therapy for the duration of their life. Recognizing vulnerable patients and being proactive along with aggressive intervention strategies vastly improves patient outcomes and is essential in addressing CPB induced renal impairment.

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AACP Social Media:
Part 3: Twitter

Authored by Molly Bryant and Alex Gum

Social media provide perfusionists with incredible opportunities to share, connect, and learn from one another. While meetings and other events give us the chance to share new research and best practices, social media can serve to bridge the gaps between the times we are able to meet face-to-face. Whether we're looking for new techniques, wanting to see what the last meeting was like, or we're just curious about the latest goings-on, social media supply us with a myriad of ways to connect with other perfusionists and organizations.

The three largest social media companies today are Facebook, a full-featured desktop and mobile platform, Instagram, a mobile photo/video-sharing platform, and Twitter, a mobile text/link-sharing platform. This article will focus on Facebook, stay tuned for parts 2 and 3 to learn more about Instagram and Twitter!

Twitter

What it's good for

Twitter is mobile-friendly platform that's good for quick communication with anyone, share photos/videos/links, and make announcements.

How to sign up

Navigate to www.twitter.com and click "Sign Up".

Follow the prompts for information.

If you choose an email address to sign up, you will receive an email with instructions to verify your identity. If you choose a mobile phone number, you will receive a verification code by call or text.

Select a username, which is unique to every user.

To use Twitter on a phone, download the app and log in with your credentials.

How to engage

Twitter loosely shares the hashtag-friendly structure found on Instagram. Text posts and link-sharing are also available, but text posts are limited to 280 characters. Rather than being a

place for robust discussion, Twitter provides a more mobile-friendly platform for sharing and discussing content more quickly than a platform like Facebook. While lacking dedicated group pages, profiles run by organizations are easy sources of information. This is particularly true while on the go or at an event, for instance.

Social Media at Meetings

Group pages and profiles become especially useful around perfusion meetings. Contributors can share photos of the meeting in real time, organize a dinner after the last scientific session, or connect with new acquaintances. These pages also serve as a scrapbook of past meetings as contributors share their photos and videos. Social media provide an easy way to get in touch with someone using only their name, which can complement the flow of getting to know new people without getting bogged down in exchanging contact information.

While at the AACP meeting in Reno, NV please share your pictures and posts on the social media platforms (Facebook, Instagram, and Twitter. You can also share pictures and posts on LinkedIn). Utilize **Hashtags** (#AACP, #Perfusion, #AACPReno2020, etc.) to allow those attending and not attending the meeting to see all of the fun activities and connections being made at the meeting. You can also **tag** individuals or groups on the post or in the comments to serve as a shorthand way of sharing the content with them.

Professional Use of Social Media

Working in a medical field often puts us at odds with the main purpose of social media. In general, hospitals craft social media policies to provide employees with a clear understanding of what they can and cannot share - these policies vary among institutions. Some common rules include:

- No HIPAA violations

o Posting any kind of information that in any way can be used to identify a patient constitutes a HIPAA violation. This information can be as cursory as a conversation picked up by your phone microphone while you film a short video of the pump.

o No misrepresenting yourself or your organization

o When sharing on social media, it can be prudent to make clear that what you share is your own opinion, and does not reflect on your institution in any way. Furthermore, take care to disclose your connection to the organization when discussing where you work and what you do - misrepresentation of what you do can reflect poorly on you and your institution/organization.

o Don't speculate

◊As a person associated with a hospital, professional organization, and perfusionists everywhere, using social media to engage in speculation or furthering rumors can have unpredictable and often negative effects on you and your associates.

This article is Part 3 of a three article series composed by the AACP Social Media Committee.

41st Annual Seminar of The American Academy of Cardiovascular Perfusion

Grand Sierra Resort
2500 East Second Street
Reno, Nevada
February 5-8, 2020

(Tentative Program)

Wednesday, February 5, 2020

9:00 AM – 2:00 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 6, 2020

7:00 AM	REGISTRATION
7:00 AM – 7:45 AM	Video Presentations
7:45 AM – 09:30 AM	Scientific Paper Session
9:30- AM – 11:30 AM	Fireside Chats *Clinical Instructor Session* *ECMO: Starting, Maintaining and Growing a Program (Webcast also)* *Perfusion Accidents* *Pump Off: Making the Most of the Last Five Years* *Student Forum Only*
11:30AM - 1:00PM	Lunch (Historical Videos)
1:00 PM – 3:00 PM	Special Scientific Panel Session **Extracorporeal Life Support** *Moderators: Allison Weinberg, Desiree Bonadonna* Pumpless ECMO Bridge to Transport: A Case Report: *Dorothy Garbin* EROCA Trial / ECPR: *Sage Whitmore, MD* Simulation Model for ECMO: *Sage Whitmore, MD* Venting the LV on ECMO: *Dana Apsell* Cleaning Out the Arterial Cannulas: *Kevin Charette* Panel Q&A
3:00 PM – 3:30PM	Break

3:30PM – 5:30PM	Special Scientific Panel Session **Hot Topics and Current Trends** *Moderators: Edward Delaney, Carmen Giacomuzzi* Double Lung Transplants: *Mat Tyndal* Terumo/Sarns Fellowship Experience: *Ashleigh LeBlanc* Vasoplegia: *Ryan Kleinman* Transmedics: *Kristina Iwai* Quantum QA/QI Initiatives/Best Practices: *James Beck* Panel Q&A
06:00PM	Sponsor's Hands-On Workshop & Reception

Friday, February 7, 2020

7:00 AM	REGISTRATION
7:00 AM – 7:45 AM	Video Presentations
7:45 AM – 9:30 AM	Scientific Paper Session
9:30- AM – 11:30 AM	Fireside Chats *Blood Conservation and Transfusion Triggers (Webcast also)* *Dealing with Stress, Finding Work/Life Balance, Team Building and Communication* *ECMO Scenarios* *Myocardial Preservation* *Pediatrics* *Pump On: The First Five Years*
11:30AM - 1:00PM	Lunch (Historical Videos)
1:00 PM – 3:00 PM	Special Scientific Panel Session **Heart Matters: Life Beyond the Pump Run** *Moderators: Carmen Giacomuzzi, William Riley* Motivational Speaker: Craig Cunningham The Heart of the Matter: A parent's perspective on raising a child with HLHS: *Aimee Mooney, OT, Assistant Professor* Integrated Life Skills for Creating Resilience to Burnout and Reconnecting with Joy in your Personal and Professional Life: *Ross Ungerleider, MD* and *Jamie Dickey Ungerleider, PhD* Panel Q & A
3:00 PM – 3:30PM	Break
3:30 PM – 5:30 PM	Memorial Session ***Charles C. Reed Memorial Lecture*** *(Ross Ungerleider, MD)* ***Thomas G. Wharton Memorial Lecture*** *(Carmen Giacomuzzi, CCP)*
6:30 PM	Induction Dinner *All Attendees and Guests*

Saturday, February 8, 2020

7:00 AM	REGISTRATION
7:00 AM – 7:45 AM	Video Presentations
7:45 AM – 9:30 AM	Scientific Paper Session
9:30 AM – 10:00 AM	Break
10:00 AM – 12:00 PM	Special Scientific Panel Session **Industry Partners: "Past, Present, and Future"** *Moderators: Richard Melchior, Giovanni Cecere* Edwards InvoSurg LivaNova Medtronic Spectrum Medical Terumo Panel Q&A
12:00 PM – 1:00 PM	Lunch (Historical Videos)
1:00 PM – 3:30 PM	Special Scientific Panel Session **Complex Congenital Heart Surgery** *Moderators: Tami Rosenthal, Kevin Charette* Clampless Cardioplegia: *Thomas Klein* Case Report; Gunshot to the Heart: *D. Brad Sanders* Circuit Miniaturization; Is it Worth the Effort: *D. Scott Lawson* Cord Blood: Possibilities and Challenges: *Bharat Datt* Antifibrinolytics for Pediatrics: *Isaac Chinnappan* Panel Q & A
3:30 PM – 5:30 PM	Fireside Chats *Evolving Scope of Practice: Past, Present and Future* *Low Fidelity Simulation: Buckets, Hoses and Duct Tape: Make the Most of What You've Got* *Pediatrics (Webcast also)* *Taking the Show on the Road: Transporting VADs and ECMO* *Using EMRs, Apps and New Technology to Improve Patient Safety and Standards of Care*
5:30 PM	Closing Business Meeting *Fellow, Senior and Honorary Members Only*

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CELEBRATING 50 YEARS WITH THE TOTAL ARTIFICIAL HEART

If you could have just one thing in this entire world, what would that be? Most of us could answer that question based on what we already have in front of us and what we have yet to achieve. Perhaps it is to have your dream car or job, to meet your favorite celebrity, or to travel to the one place you have always dreamed about.

Now imagine you have heart disease—that you are fighting for your life in a hospital bed. You are constantly in pain, having difficulty breathing, and feeling like a pincushion from each lab draw stick and IV start you need to help sustain your life. Your family members visit you constantly, but you still feel despondent because when their visit ends, you are unable to go with them. You feel helpless because you can barely find enough strength to get up and walk to the chair next to your bed.

Would your answer change then? Would you wish you were able to take another breath without hesitation, or perhaps for a perfect heart? But if that perfect heart was not available yet, would you at least want some sort of support that could give you one more day to spend with your loved ones? The further improvements of the total artificial heart (TAH) throughout the years have given hope to those patients in need.

Dustin Gitchell, BS
Leslie Gonzalez, BS, RRT
Kevin Do, BS
Sean Pollock, BS
Nicole Morency, BS
Tony Dohman, BS, RN
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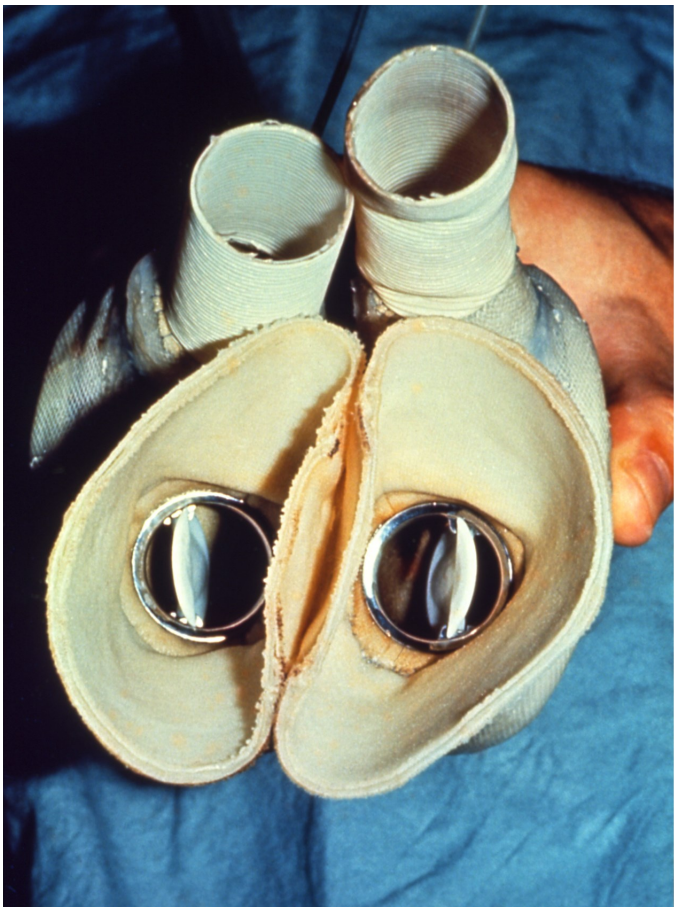
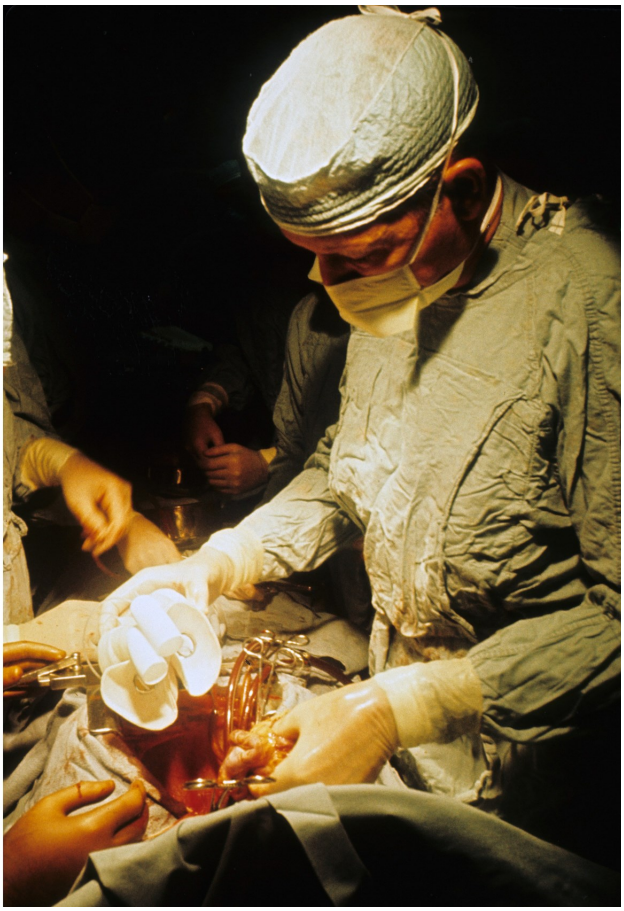
Texas Heart Institute School of Perfusion Technology

Houston, Texas

With every step that Dr. Denton A. Cooley and Dr. Domingo S. Liotta took toward the operating room on April 4, 1969, the gravity of the situation intensified; however, the composure of Dr. Cooley kept their feet as light as if they were walking on the low-gravity surface of the moon. Dr. Cooley was about to perform the first implantation of a TAH into a patient, marking a triumphant victory for humanity against a disease that ravages the world. Dr. Cooley executed this feat with poise and described it as being any other operation, and the results were indicative of that. The patient was in critical condition and had exercised every other option available before concluding that the newly developed TAH was his best chance of survival. Dr. Cooley performed this miracle not to embroider his name and legacy but simply to extend this patient's life. The patient lived with the TAH for three days until a donor heart was found. This sensational achievement was attributable to the patient's own courage and Dr. Cooley's bravery.

Perceived reality is the enemy of imagination, and the stance of the few is often propagated as undeniable truth. Dr. Cooley's imagination and charisma challenged the stance that an artificial heart was merely science fiction, and he overcame that dogma. It has been fifty years since this accomplishment, and it has paved the way toward more feasible and prolonged modes of artificial cardiac support. Per Dr. Jack G. Copeland, a temporary TAH (SynCardia Systems, Inc., Tucson, AZ) has been implanted in more than 1,100 patients and is currently being used at approximately 100 different health care centers across multiple countries. Currently, the longest duration of support stands at 3.75 years, in a male patient who later received a heart transplant and is living well. Dr. Jack Copeland, an American cardiothoracic surgeon who specializes transplanting both human and artificial hearts, declares that out of the 1,100 pa-





tients, more than 47 have survived for more than a year with the artificial heart, and 72% of them received a heart transplant. The 1-year survival rate among these patients after transplantation was 80%-86%.

Although the TAH has continued improved since its genesis of, many challenge their usefulness, stating that ventricular assist devices can be and are superior. Further research, however, has suggested that most patients with biventricular failure require a TAH. As Jason A. Cook, an established author in cardiothoracic surgery, points out, "As experience with TAH advances, growing evidence supports its use in patients with biventricular heart failure. Patients with concurrent right ventricular failure in addition to left ventricular failure have poorer outcomes with left ventricular assist devices (LVAD) than patients with isolated left ventricular failure. Initial biventricular assist devices (BiVAD) for critically ill patients usually provides higher cardiac output at lower doses of inotropes, which can help resuscitate end organ malperfusion. One small retrospective study even showed no differences in mortality for patients with a TAH compared with BiVADs."

Although TAH implantation success and patient survival rate with the TAH is high at 80%-86%, there is still a chance for complications. The major complications of TAH implantation include strokes, infections, bleeding, renal failure, and chronic anemia. In an article in the *Journal of Thoracic Disease,* Jason Cook and others state that [1].[3]± of TAH-supported patients had strokes, 63.4% had infections (most commonly in the lungs and urinary tract) requiring treatment, and 42.6% had bleeding episodes of varying severity. These episodes included mediastinal bleeding requiring mediastinal exploration in 24.7% of patients, of whom 44% died within a month. Despite the complications of the

Continued on Page 22

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TAH, the necessity and inspiration to achieve success in this field is continuing to expand, and new ideas are being challenged and tested to further improve the field and rid it of obstacles.

Dr. O. H. "Bud" Frazier was a fellow pioneer in many of the advancements made in TAH devices and was a friend and colleague of Dr. Cooley. Our class had the opportunity to interview Dr. Frazier, and he provided us with valuable insights about the conception of the TAH and of what is to come with this technology. He further elaborated on the character of Dr. Cooley, and it became abundantly clear to us why these trailblazers achieved the level of success that they did. Dr. Frazier informed us that "the most important thing from conception until now is chance." He further elaborated that every achievement, whether past, present, or future, is driven by the chance of discovery and the motivation of the individual to act. Dr. Cooley dared to inspire by challenging the dogma of his time, and when given the chance, he capitalized by implanting the first TAH. While there are still challenges to overcome with the technologies of TAHs, thanks to Dr. Cooley humanity will continue to be inspired and take advantage of its chances until heart disease is overcome.

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Dr. O. H. "Bud" Frazier and the student perfusionists (authors).

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How long have you been in the perfusion field? _____

Will you be attending the Induction Dinner on Friday evening? YES NO
(Dark Suit and Tie Required / Black Tie Optional or join in the festivities and dress in the Great Gatsby style - visit our website for examples.)

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Credit card billing address if different from above.
ADDRESS _____

CITY _____ **STATE** _____ **ZIP** _____

Signature_____

** ***There will be a $25.00 service charge for any check returned for insufficient funds.***

INSTRUCTIONS and INFORMATION

o Complete each appropriate section of this form by printing or typing.
o *All attendees are invited to the Induction Dinner on Friday evening. Attire is dark suit and tie required.*
o Members must pay their 2020 Annual Dues along with their registration fees by completing that portion of the form.
o You will receive acknowledgment of your pre-registration by January 17, 2020--bring it with you to the meeting.
o No pre-registration will be processed after January 12, 2020
o Your receipt and meeting credentials will be available for you at the Pre-Registration desk at the meeting.
o There will be **NO ADMISSION to any Fireside Chat without proper admission credentials**.
o If you are joining The Academy with your registration you must:
1) complete appropriate areas of the form;
2) you MUST INCLUDE the membership application form;
3) include the $25 filing fee;
4) include $155 for the 2020 Annual Dues;
(Your membership begins with the closing business meeting)
o ONLY VISA/MasterCard credit cards are accepted - with VISA/MasterCard you may FAX your registration to (717) 867-1485
o The AACP Federal Tax ID Number: 63-0776991 (for hospital use only)
o Refund policy: Anyone that is pre-registered for this meeting and is unable to attend will receive a full refund minus $50.00 for handling, mailing, and processing upon written request before January 15, 2020.

o **Make checks payable to AACP (US dollars). Mail completed pre-registration form and check to:**
AACP
515A East Main Street
Annville, PA 17003

IF YOU HAVE QUESTIONS FILLING OUT THIS FORM, PLEASE CONTACT THE NATIONAL OFFICE (717) 867-1485.

o **If paying by VISA/MasterCard you may FAX this form to (717) 867-1485 or mail to above address.**

Contact Information for Our Sponsoring Partners

CARDIOQUIP
Phone: 979-691-0202 or 888-267-6700
Website: www.CardioQuip.com

INVOSURG
Fax: 617-507-6462
Website: www.invosurg.com

LIVANOVA
SORIN GROUP USA, INC.
Phone: 800-221-7943 or 303-467-6517
Fax: 303-467-6375
Website: www.soringroup.com

MEDTRONIC PERFUSION SYSTEMS
Phone: 763-391-9000
Websites: www.medtronic.com
www.perfusionsystems.com

QUEST MEDICAL, INC.
Phone: 800-627-0226 or 972-390-9800
Fax: 972-390-2881
Website: www.questmedical.com

SPECTRUM MEDICAL, INC.
Phone: 800-265-2331
Fax: 803-802-1455
Website: www.spectrummedical.com

TERUMO CARDIOVASCULAR SYSTEMS
Phone: 734-663-4145 or 800-521-2818
Fax: 734-663-7981
Website: terumo-cvs.com

Important Academy Dates

The ACADEMY ANNUAL MEETING DEADLINES

ABSTRACT DEADLINE	**October 31, 2019**
MEMBERSHIP DEADLINE	**December 5, 2019**
PRE-REGISTRATION	**January 12, 2020**
HOTEL REGISTRATION	**January 12, 2020**
2019 ANNUAL MEETING	**February 5-8, 2020**