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Spring 2021

The Academy Newsletter

Thank you!

To everyone who participated in the annual meeting – THANK YOU!

A huge note of appreciation to anyone who tuned into our recent virtual symposium. Whether you registered for one session or all six, your support made the event possible and I venture to guess that our success as the first meeting of this "season" will inspire our fellow Perfusion entities to go big as they undoubtedly plan their inaugural all-virtual events!

We had an overwhelming outpouring of support from our Council and committee members, presenters, panelists and moderators. Our sponsors were extremely generous and understanding of our limitations. CMIAV is the company that coordinated the vast array of services from laying out our format, setting up a recording schedule, teaching our many presenters and moderators, editing the presentations, broadcasting the event and troubleshooting where they could….. we could never have done this without them!

We are all hoping for an in-person meeting in Austin. The next few months of vaccinations, continued social distancing and "spread-stopping" will arguably be the most tenuous of modern time. I wish you health, happiness and peace as we navigate 2021.

Warmest regards,

Bill Riley
President, American Academy of Cardiovascular Perfusion

Inside this issue

Bill Riley
President, AACP

Welcome to New Members

The American Academy of Cardiovascular Perfusion would like to welcome the following individuals whom were voted into membership.

Fellow Member
Edward Delaney

Members
Alexander, Kiera
Anderson, Kelsey
Archer, Zachary
Clausen, John
Crum, Jayson
Ebel, Tanya
Gill, J. Quinn
Graham, Justine
Jubak, Bob
Lojovich, Sarah
Nanduri, Sravani
Oldeen, Molly
Puis, Luc
Rider, Kyle
Scullion, Mark
Sharma, Navriti
Williams, Alicia
Wolak, Amanda
Yamin, Adam

Students
Anderson, Kyle
Ansari, Nausherwan
Applewhite, Shanon
Baker, Rebecca
Barrios, Barbara
Beatty, Brent
Beletti, Courtney
Biazzola, Filipi
Blackwell, Daniel
Breckon, Jocelyn
Breshears, Lathen
Bullington, Ann Margaret
Butterbaugh, Rebecca
Canchola, Stephanie
Chapman, Eric
Chau, Siu
Cherichella, Robert
Cheyne , Jason
Ciarlo, Timothy
Cudd, Matthew
Delgado, RoseAnne
Drake, Leslie
Eash, Morgan
Evangelista, Anthony
Evans, Shelby
Fehrenbacher, Regan
Fischer, Kailah
Frankowski, Emily
Gadille, Sarah
Geisinger, Kaitlyn
Geurts, Brandon
Gonzales, Dallas
Goss, Lydia
Gray, Hannah
Hatcher, Nikah
Hayes, McKenzie
Ingiaimo, Cierra
Jacobson, Kenneth
Joyner, Jimmy
Karaduman, Melisay
Koerten, Kathryn
Larson, Joshua
Lee, Jihyun (Jane)
Leonor, Alexander
Lockwood, Courtney
Lohbusch, Breana
Luan, Liming
Magowan, Benjamin
Martin, Payton
Mendoza, Victor
Michael, Langston
Millikan, Trevor
Montgomery, Tairyn
Morgan, Shane
Ng, Man
Olson, Kaylee
Parker, Jack
Patterson, Benjamin
Perry, Edda
Pritchard, Paige
Quiambao, Laurice
Rager, Steeley
Scatena, Wayne
Scegolev, Irina
Schell, Kaylee
Schultz , Rachel
Seals, Marshall
Seefeldt, Cassandra
Short, Sydney
Sierra Aquino, Manuel
Smith, Michael
Solano, Megan
Spencer, Jeffrey
Strang, Meagan
Syquia, Jose Antonio
Tam, Andrea
Tetuik, Maciej
Tidwell, Kelvin
Torres, Ramiro
Tran, Sarah
Vanderbleek, Jordan
Varghese, Bency
Wadia, Aarish
White, Shawn
Wixom, Somer
Wong, Ethan
Wright, Anthony
Zachary, Melissa

REMEMBERING THE WAUD PRINCIPLE

Kelly D. Hedlund, MS, CCP

The University of Kansas Health System

HaysMed

Hays, Kansas

Introduction

Russell Amos Waud was born in 1893 and grew up in the Canadian province of Ontario. He was a tinkerer at heart, and was enamored with any kind of scientific gadget. In 1921 he achieved his medical degree from the University of Western Ontario. Following an internship at Victoria Hospital in London, Ontario, he opened a practice in family medicine. This was short-lived however, as the prospect of doing research consumed him. He undertook postgraduate studies in physiology, obtaining a Master's degree in 1925 and a Ph.D. in 1927. He then returned to Western and headed up the Departments of Physiology and Pharmacology until his retirement in 1958 (1).

Early Research

In 1902, the British physician Sir James Mackenzie devised the first polygraph to record arterial and venous pulse tracings (2). Seeking to improve upon Mackenzie's design, Waud added additional electronic and magnetic circuitry (see Figure 1). His efforts, published in 1924 (3), resulted in tracings much more discernible and greater in amplitude (see Figure 2).

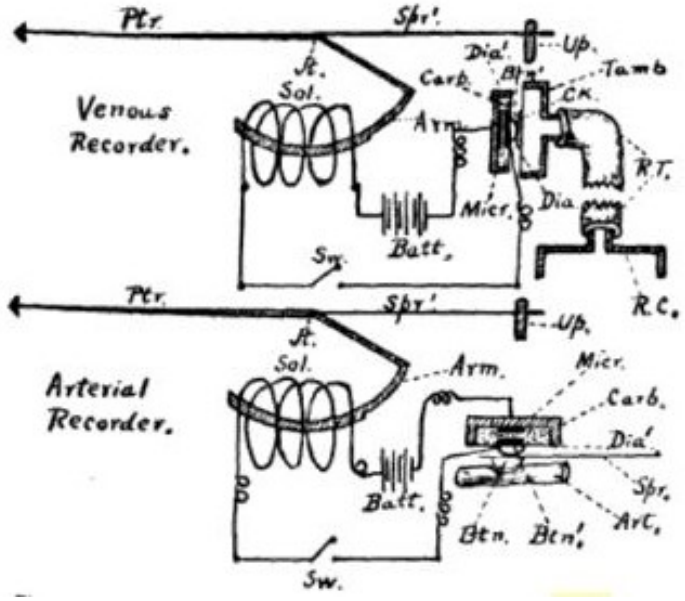

Figure 1. Schematic of Waud's polygraph for recording arterial and venous pulse tracings (circa 1924).

Waud also studied the effects of respiration on the venous pulse (4). In 1927, as part of his doctoral thesis, Waud theorized that the fall in blood pressure during shock was caused by a transient reduction in the blood's viscosity. His research, using rabbits, was lauded as historic and published in the *American Journal of Physiology* (5). As

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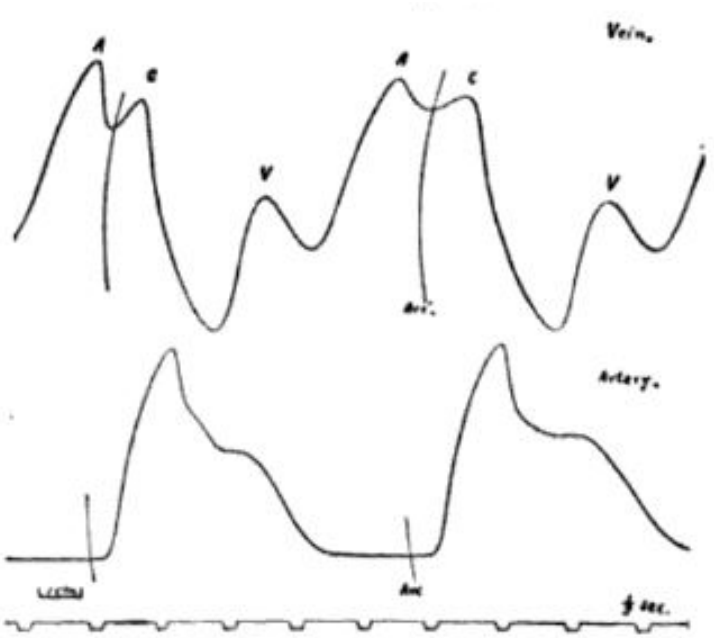

Figure 2. Venous (top) and arterial (bottom) tracings recorded from Waud's polygraph (circa 1924).

a professor, Waud constantly sought ways to better educate his students. In 1930, he constructed an amplifier for listening to heart sounds (see Figure 3). At the time, the carbon microphones used for auscultation hissed and popped. Waud's amplifier, considered revolutionary, used condensers to filter out unwanted white noise and distortion (6). Having never stopped refining his polygraph, Waud in 1936 replaced rubber parts with metal, and included the electrocardiogram as a simultaneous tracing – a major advancement (7).

Figure 3. Waud (far left in lab coat) instructs students about heart sounds using his amplifier (circa 1930).

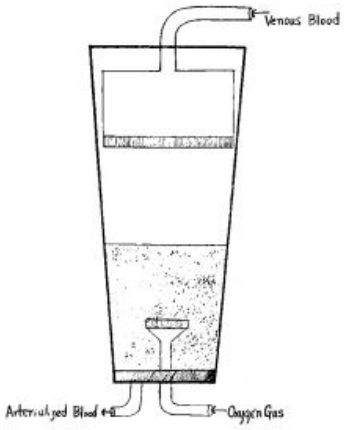

Figure 4. Schematic of Waud's foaming oxygenator (circa 1948).

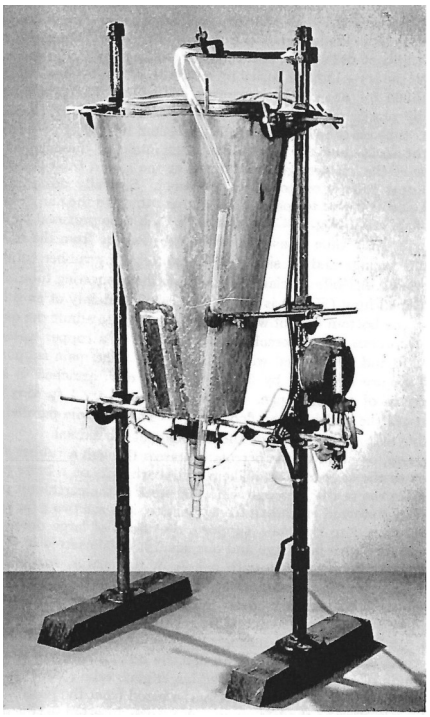

Figure 5 . Photo of Waud's foaming oxygenator. Venous blood enters the top of the glass vessel (housed inside the copper water bath) and oxygen enters the bottom in countercurrent fashion. The resulting foam layer provides a cushion for gas exchange to occur. The arterial blood outlet is seen exiting the bottom of the oxygenator (circa 1951).

Heart-Lung Machine

In 1948, Waud began work on a heart-lung machine to aid his study on the effects of drugs on the circulatory system. Unlike Gibbon, who at the time was still experimenting with the revolving cylinder oxygenator, Waud chose to build a foaming device for gas exchange (see Figure 4). The Russian experimenter Brukhonenko is generally credited with designing the first foam oxygenator in the 1930s. It's not likely that Waud knew of Brukhonenko's accomplishments for two reasons. First, the political climate of the time prevented Brukhonenko's research from being published in North American periodicals. Second, and perhaps more importantly, Waud never referenced any of Brukhonenko's articles or credited him in any way. Waud's oxygenator was an 8-liter glass percolator contained within a copper water bath of similar shape and size (see Figure 5). Venous blood entered the top of the percolator through a perforated showerhead-like manifold. This ensured a downward bloodstream with an evenly distributed pattern. Oxygen, in counter flow fashion, was blown upward from the bottom of the percolator. The resulting foam served as the gas exchange surface for the incoming venous blood. Once arterialized, blood exited the bottom of the percolator through a port adjacent to the oxygen inlet. Waud used two 50 mL glass syringes, a specially designed cam, and an electric motor as his right- and left-sided pumps. In 1951, Waud's heart-lung machine was featured in the *Calgary Herald Newspaper* (9). In 1952, Waud published a lengthy description of his device in the *Canadian Journal of Medical Sciences*, reporting that his apparatus had been used successfully in over 100 dog experiments (8).

The Waud Principle

Using Waud's device as a forerunner, numerous investigators built similar foam oxygenators, each with slightly higher gas transfer rates or enhanced safety features. One of Waud's close colleagues, Peter Salisbury, who himself built a foam oxygenator, coined the phrase *"the Waud principle"* in 1955 (10). Essentially, the Waud principle refers to a foaming device where the oxygen and venous blood flow countercurrent, thus producing a continuously-renewing layer of foam for gas exchange to occur. Salisbury credits Waud with the design, and specifically mentions him in at least three of his publications (10, 11, 12). Reference to the Waud principle is also made in a Master of Science thesis project in Canada in 1958 (13), and by a Japanese researcher studying extracorporeal circulation in 1962 (14).

Later Years

In 1954, Waud was selected as part of an esteemed group of physicians to help form the American Society of Artificial Internal Organs. Other members of this group included Charles Bailey, Leland Clark,

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Clarence Dennis, Forest Dodrill, John Gibbon, Jr., Willem Kolff, Walt Lillehei, and of course his old friend Peter Salisbury. Waud presented papers at the first two meetings – both focusing on the effects of drugs in the artificial heart and lung preparation (15, 16). In 1958, he retired, and spent his golden years gardening and sailing the nearby Great Lakes. He died peacefully at the age of 79 just a few miles from where he was born.

Though his name is relatively obscure, Russell Waud's medical achievements are significant and deserve to be remembered.

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ADVANCING ECLS THERAPY

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Notes on Cold Agglutinin Disease for Cardiac Perfusion Practice

Liming Luan

School of Perfusion Technology
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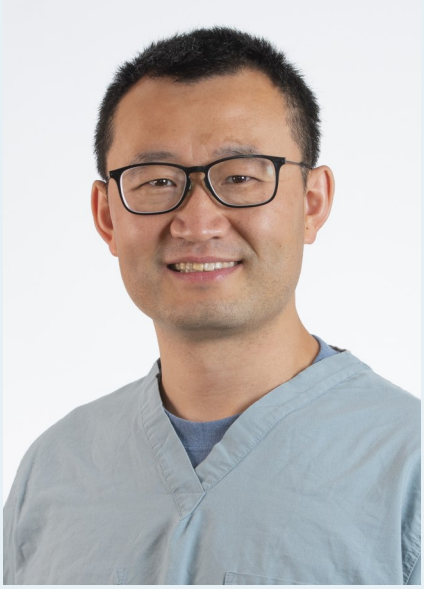

Cold agglutinin disease (CAD), a subtype of autoimmune hemolytic anemia, is a rare (approximately 1:300,000) disorder that is characterized by the pathological destruction of red blood cells (RBCs) by circulating cold-sensitive antibodies, also known as cold agglutinins (CAs). These antibodies, usually immunoglobulin M (IgM), are present in most people but are rarely of clinical significance at normothermia.[1] The antibodies become active at temperatures <30 °C and then bind RBCs into clumps (agglutination), which eventually cause microvascular occlusion and systemic hemolysis.[2] Therefore, CAD can lead to anemia and other related symptoms including tiredness, dizziness, headaches, cold hands and feet, pale skin, dark urine, chest pain, and even heart failure. CAD is also called cold hemagglutinin disease, cold agglutinin syndrome, and cold hemagglutinin syndrome.

Two types of CAD

First described in the 1950s,[3,4] CAD has traditionally been classified as either primary or secondary. The term "cold" is derived from the immunological rather than the clinical features of CAD. Primary CAD is a chronic condition and does not involve an underlying disease. A characteristic clinical manifestation of primary CAD is excessive B lymphocyte proliferation, which causes production of CAs. Production of these antibodies and the resulting symptoms often occur in those 50 years of age and peak in the 70s and 80s.[2] Our knowledge of primary CAD is incomplete. Secondary CAD is the more common form and usually results from an underlying infectious disease such as mycoplasma pneumonia or human immunodeficiency virus (HIV).[5] Drug-induced secondary CAD is uncommon.[2] In both forms of CAD, autoantibodies bind to RBCs, and the antigen-antibody complex induces activation of the complement system and hemolysis.

CAD and COVID-19

In December 2019, coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged in Wuhan, China. By March 2020, COVID-19 had spread worldwide, leading to an ongoing pandemic. Patients often present with fever, cough, shortness of breath, fatigue, and loss of smell and taste. CAs have been reported to be of clinical significance in some COVID-19 patients despite the presence of minimal *in vivo* hemolysis, given their frequent need for renal replacement therapy.[6] Because of the high incidence of disseminated intravascular coagulation in critically ill COVID-19 patients[7] and the literature associating CAD with thrombotic events,[8] some investigators have speculated that SARS-CoV-2 infection may cause

CAD.[9, 10] However, more research is needed to better understand the relationship between SARS-CoV-2 and CAD, including complete blood work evaluation in these patients.

CAD and cardiopulmonary bypass

Cardiopulmonary bypass (CPB) and hypothermia provoke a significant physiological disturbance in patients undergoing cardiac surgery, particularly in patients with CAD. The presence of CAs creates a significant risk of morbidity during CPB requiring systemic hypothermia and cold cardioplegia. Recent studies suggest that dysfunction of the myocardium and other end organs, such as the liver and kidneys, is attributed to CAs in patients undergoing hypothermic CPB.[11] Mainly, this occurs because cold temperatures activate CAs, which in turn cause massive hemagglutination, followed by complement fixation, catastrophic hemolysis, and microvascular thrombosis upon rewarming. This situation can lead to intracoronary thrombosis, visible agglutination, and high line pressures in the cardioplegia circuit, as well as insufficient cardioplegia delivery, during cardiac operations.

The incidence of CA-related complications during cardiac surgery is reported to be ~0.8% to 4%.[11] Numerous published case reports have outlined intraoperative management strategies for CAD patients with coronary artery disease (for both off-pump[12] and on-pump[13] procedures), coronary sinus ostial abnormalities,[14] aortic[15] or mitral[16] valve defects, aortic arch aneurysm,[17] and aortic dissection,[18] and for those undergoing organ transplantation.[19, 20] In a few case reports, cardiac surgery in pediatric CAD patients with congenital heart disease has been described.[21, 22] Although intraoperative management strategies remain controversial, CAD patients require extra attention and individualized planning for managing CPB and myocardial protection, and maintaining CAD patients within a categorically safe temperature range is crucial throughout the procedure.

CAD screening before cardiac surgery

As it is essential to know who is at risk due to CAD, every patient scheduled for a cardiac procedure should undergo preoperative screening for CAD before surgery. In addition to the age and infection factors mentioned above, CAD is 1.5 times more common in women than in men.[5] Preoperative symptoms and signs of hemolytic anemia (eg, pallor, jaundice, and hemoglobinuria) are good indicators for laboratory tests such as reticulocyte count and a peripheral blood smear, which can provide a preliminary diagnosis of CAD. Unfortunately, some patients who have no symptoms or signs still could be at risk of agglutination and hemolysis due to non-physiological hypothermic conditions during cardiac surgery. In emergency situations, it is critical to empirically treat patients with signs of CAD, especially if the patient had clotted blood in initial laboratory draws.

Plasma titers of CA and the thermal amplitude test should be used to screen for CAD-positive patients, because activation of CAs closely depends on those two factors. The cold agglutinin titer blood test, also known as the direct Coombs test or the direct antiglobulin test (DAT), is a reliable diagnostic laboratory test for CAD. Healthy individuals often have low serum levels of CAs (about 1:16), but they can safely undergo CPB with little change in practice. Lower CA titers (up to 1:40) are not considered clinically significant[11] and may not pose a risk of agglutination during surgery. However, with higher titers (>1:128), CA activation is more likely to occur.[5] A hematologist's evaluation should be sought in patients with a significantly higher titer. Hemolysis is rarely seen when CA titers are below 1:1000.[13] Patients with CAD would present with laboratory evidence of hemolysis (eg, high lactate dehydrogenase, high bilirubin, and low haptoglobin) and a positive direct Coombs test. In addition to CA levels in plasma, the temperature below which CA activation occurs should be determined preoperatively, thus providing the surgeon and anesthesiologist with an appropriate temperature range for the patient.

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Born and raised in China, Liming Luan was a staff scientist in the Department of Anesthesiology at Vanderbilt University Medical Center (VUMC) before being accepted as a student at Texas Heart Institute School of Perfusion. During his tenure at VUMC, Liming not only actively maintained his own projects, but also served as a key facilitator, collaborating with each member of the research team. Liming was nominated for the Roger England Research Award, which he won at the 13th Annual Department of Anesthesiology Research Symposium in 2017 at VUMC. Currently, Liming is a student ambassador to the American Academy of Cardiovascular Perfusion.

Management and Treatment

Because of the rarity of CAD, no systematic studies have been conducted to assess its optimal treatment. The primary strategy is to treat the underlying infection and largely depends on the severity of the condition. For clinical perfusionists, CAD patients with high CA titers and a wide thermal range for antibody activation present challenges during CPB. Unfortunately, little is known on how to treat such patients who have severe, life-threatening hemolytic anemia. A common intraoperative management strategy for CAD patients during cardiac surgery is to maintain normothermic CPB or keep systemic perfusion temperature above the thermal threshold of agglutinin activity, while continuously delivering warm blood cardioplegia or cold crystalloid cardioplegia above the thermal amplitude. In addition, anesthetic agents and fluids (including priming fluids) should be warmed, the operating room temperature should be increased, and a warming blanket can be applied.

For patients who require deep hypothermic arrest, preoperative high-volume plasmapheresis[13] (also known as plasma exchange therapy) in combination with intravenous immunoglobulin infusion[23] can be used to significantly reduce CA titers. Previous reports have described multiple preoperative management strategies, such as the use of steroids,[24] rituximab,[25] cyclophosphamide,[26] chlorambucil,[11] high-dose IgG,[27] and eculizumab.[28] These medical therapies are suggested to reduce CA titer or reactivity, but the details of their underlying mechanisms are unclear.

Because CAD is rare and still underdiagnosed in many hospitals, it is difficult to design and conduct randomized clinical trials with adequate statistical power. Nevertheless, several B-cell-directed therapies, such as rituximab, are now available. Novel complement-directed therapies are currently being investigated and appear promising. Potential life-threatening events such as heart attack and stroke caused by CAs warrant better awareness and improved screening for individuals suspected of having these antibodies. As evidence-based therapies for CAD patients are urgently needed, we as perfusion students and perfusionists need to stay abreast of new developments in the field and be vigilant in managing patients during our practice.

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Meta-Analysis on ANH & RAP on Reducing Allogeneic Blood Transfusions

Maria Plomondon

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Hamden, CT

Abstract

Background: Perioperative allogeneic red blood cell transfusions have been associated with increased hospital costs, morbidity, and mortality in patients post cardiac surgery. It was hypothesized that acute normovolemic hemodilution and retrograde autologous prime can decrease allogeneic blood transfusions in cardiac surgery patients in the perioperative period.

Methods: A meta-analysis was done to determine the efficacy of ANH in reducing perioperative allogeneic blood transfusions. Seven studies were chosen and included adult and pediatric cardiac surgery to assess the efficacy of ANH with a total sample size of 38,360 participants. A second meta-analysis was done to determine the efficacy of RAP in reducing perioperative allogeneic blood transfusions in adult cardiac surgery patients. Seven studies were chosen with a total sample size of 23,868 participants. The article search was conducted through the Quinnipiac University's, Arnold Bernhard, and Netter Health Science Library from May 2020 to September 2020.

Results: The ANH group received allogeneic blood transfusions an average of 69.8% of the time when compared to the control group which received them 100% of the time and was found to be statistically significant with a p-value of 0.004. It was determined the RAP group received allogeneic blood transfusions 14.6% of the time when compared to the control group which received them 30% of the time and was found to be statistically significant with a p-value of 0.03.

Conclusion: The results suggest that ANH and RAP are safe and effective blood conservation techniques for patients undergoing cardiac surgery in reducing allogeneic blood transfusions. A strong association between reduction in allogeneic blood transfusions in the perioperative period was appreciated with the use of ANH and RAP.

This is an abstract from Marie's presentation at the 42nd Annual Seminar of The American Academy of Cardiovascular Perfusion.
To view her full presentation, click here.

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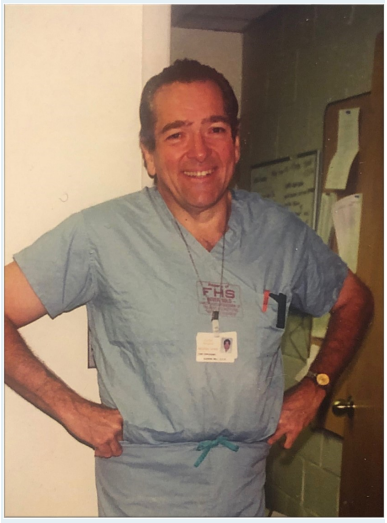
FRESENIUS MEDICAL CARE

Aaron G. Hill Research Grant



Aaron G. Hill was a pioneer in clinical perfusion and heavily involved in the establishment of the profession. He was truly a good friend, colleague and mentor to many of us in the field of Perfusion. A research grant has been established in his name.

If you are interested in applying for a research grant, click on this link.

Aaron G. Hill Research Grant Application

Purpose: To help support perfusion-related research

Requirements: Grant recipients are required to present their research findings at an Academy meeting. This includes submitting an original manuscript that can be sent to the journal *Perfusion* for possible publication.

Name: _____

Address: _____

Phone: _____ Email: _____

Institutional Affiliation: _____

Are you a Perfusionist or a Perfusion Student? _____

Does this investigation involve patients or patient data? YES or NO

If YES, do you have documented institutional IRB approval? YES or NO

IRB Number: _____

Estimated budget for your study: _____ Amount Requested: _____

On a separate sheet, give a short, detailed summary of your study, including the following: (1) title of your study; (2) an assessment of originality and how the study will contribute to the scientific literature; (3) expected start and finish dates for the research project; (3) names of co-investigators or senior advisors including their anticipated roles; (4) specifically, what will the grant award be used for such as laboratory supplies.

(NOTE: travel expenses are not covered by this grant)

I am the principle investigator on this project and I understand that if awarded a grant, I must present my research at an Academy meeting at my own expense and submit a manuscript suitable for potential publication in the journal Perfusion.

_____ _____

Print Name Signature

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Important Academy Dates

The ACADEMY ANNUAL MEETING DEADLINES

ABSTRACT DEADLINE	**October 15, 2021**
MEMBERSHIP DEADLINE	**December 9, 2021**
PRE-REGISTRATION	**January 14, 2022**
2022 ANNUAL MEETING	**February 9-12, 2022**

Other Meetings

The Maryland State Perfusion Society is presenting the (13th) Annual MidAtlantic VAD & ECMO Symposium - MAVES 2021

DATE: Saturday, April 24, 2021
TIME: 0700H to 1745H
LOCATION: LIVE VIRTUAL
ABCP CATEGORY 1 CEUs (for CCPs): 10
COST: $85.00 (All Attendees)
$35.00 (Active Military)

For Information & Registration:
https://mdperfusion.com/events/

For Questions:
treasurer@mdperfusion.com

2022 Annual Meeting

Lost Pines, Texas

February 9-12, 2022

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Single/Double Occupancy:
$239.00 (includes daily resort fee)

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