The Academy NEWSLETTER

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FALL 2012

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Remembering 911: One Perfusionist's Perspective

Perfusion On the morning of September 11, 2001, I arose as I do every morning at 4:30 am to travel into New York City to my job as a perfusionist at the New York Presbyterian Medical Center. Located on the upper west side of Manhattan, walking across a bridge between hospital buildings there is a perfect view of the Empire State Building, and the World Trade Center (WTC) just beyond. It was a beautiful clear day and I remember that I was preparing for a Thoratec Heartmate training in our animal facility; coincidentally we were training a team from Boston.

Our operating rooms were in full swing. We had patients in all of our 24 ORs, and the cardiac patients had perfusionists preparing the heart-lung machine for their open heart procedures. Everyone has a task and the movement is choreographed some by music, and some by the everyday set up sounds of an operating room suite. By the time hospital personnel realized that this day would change the world as we know it, it was too late to retract the momentum.

At 8:46:30, Flight 11, originating from Boston crashes into the north face of 1WTC between floors 93 and 99. I heard from the operating room lounge where a television usually has news on in the early morning, a small plane must have run into the one of the towers of WTC. Everyone was stunned. There was a hole in the building and honestly from the view it looked rather small compared to the massive tower. The immediate thought running through my head was "Oh my God someone must have had a heart attack and crashed into the tower." (perfuionist thinking) We all had no idea that in fact the powerful shock wave from a hijacked plane that crashed into the tower ran down to the ground and then up the entire building trapping everyone above the 93rd floor. Immediately the building core is on fire. I continue to the animal lab to give my lecture to our trainees.

At 9:03:02, Flight 175, originated from Boston crashes into the south face of 2WTC between floors 77 and 85. All 65 people on board the aircraft die instantly on impact, and unknown hundreds in the building perish as well. Now I am visibly stunned. The word is out that the United States is under terrorist attack. I begin my lecture to the Boston team

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that has come to New York on a plane that preceded the hijacked one. The hospital goes into disaster mode and preparations begin to disseminate thousands of injured from the WTC disaster. Our command center contacts every employee by email, telephone, and beeper to give instructions for the disaster. Immediately, we are told to finish our first cases and the remaining day's schedule is cancelled. It's obvious that our duties as perfusionists are to stay with our patients. In the midst of our own fears and wanting to flee to safety, we are preparing for terrible injuries; none of which I had ever experienced in my lifetime. I was terrified. But first I had to finish the training.

All of our heart lung machines were set up in anticipation of injured that may need them. We had no idea what kind of injuries we would find. Every piece of equipment that might help save a life was prepared for use. Our autologous blood savers were ready, and we discussed how any of us could be used in other areas if needed. We were on standby and were not allowed to leave the hospital for any reason.

Two other flights, Flight 77 and Flight 93, crash into the Pentagon and Somerset County, Pennsylvania respectively. More lives lost. My heart is broken, the staff is in shock. No one can believe what has happened. In just over an hour, the south tower has collapsed, in another hour and a half the north tower would be collapsed. It was estimated that 50,000 people work in those buildings each day. The hospital is on red alert. We wait, and we wait, and we wait the entire day. No one is coming to us. We don't understand.

Finally at 7:00 pm a meeting is called of all employees by administration. We assemble dazed and confused after listening and watching the coverage on radio and television since early this morning. Everyone is sick at the thought that so many have perished. Trying to reach loved ones since the disaster was impossible as the lines of communication were gone. The WTC held the antennae for most of the communication systems in New York. The hospital then announced that everyone was to head home for today, but return tomorrow at 7 am. They stated, "The reason we have no injured patients is that there were none to be found so far. Most we think are gone." The rescue efforts will continue but no one will be coming to New York Presbyterian this evening.

The rest is history and everyone has been touched by the annual remembrances of the events of 911. Each of us has some connection to the event, either by knowing a close friend, relative, or acquaintance that perished that day. Our children are growing up in a new world and the impact of possible future terror attacks remain in their lives every day. For me, that day was surreal. The fact that I am a perfusionist that helps to save lives and was unable to perform any duties that day to help even one injured patient will remain in my heart forever. And as I participated in artificial heart training for the Thoratec Corporation in New York, their vice-president, Thomas E. Burnett, VP, COO, was among the passengers on Flight 93, able to resist the hijacking that could have killed hundreds more, only to perish in a field in Somerset, Pennsylvania.

Once again, the Freedom Tower (WTC) is the tallest building in New York. It has been rebuilt with the utmost respect and realization that we will not live in fear.

Linda B. Mongero, CCP

President,
American Academy of Cardiovascular Perfusion

AACP Announces Three New Programs For Student Perfusionists

For the past several years, the American Academy of Cardiovascular Perfusion (AACP) has been developing new programs to assist students in becoming more involved in our society. Past programs that have been incorporated within the annual AACP Symposium have included an exclusive student-only session, a reception for students to network with Fellows and clinical employers, financial assistance with conference expenses and much, much more. These programs have given students better insight into professional organizations as well as make them more well-rounded prospective clinicians. The AACP would like to inform you of three new programs that are soon to be implemented. It is our hope that these three new programs will further strengthen the foundation for students to become successful professionals. After several years of deliberation with these projects they will be implemented at the 2013 AACP Annual Symposium to be held in Los Angeles from January 24-27, 2013.

The first program that is to be implemented is the *AACP Student Ambassadorship*. As an ambassador, the student will assist the AACP with future programs and suggest topics directly relevant to their perfusion education program. The Student Society division of the AACP kindly asks each perfusion education program director, if you have done so already, to nominate one of your students to be your program's AACP *Student Ambassador* (please include his/her CV & Biography). We hope to have each student that is nominated as a Student Ambassador of the AACP to be in attendance at the 2013 annual symposium.

The second program that will be instituted at this year's AACP Symposium will be the AACP Student Council. The AACP Student Society has selected the AACP Student Council for this year. The following students will make up the inaugural AACP Student Council, President -Seana Hall - University of Arizona, Vice President - Claire Jara - State University of New York, Secretary - Whitney Western - University of Nebraska Medical Center, Treasurer - Krishna Phifer – RUSH. These positions will play an important role during the Student-Only Fireside Chat at the annual AACP Symposium.

The third program that is to be implemented is the AACP *Student Mentorship*. This program will enable students to partner with a current AACP Fellow prior to graduation. It will allow a student to develop a professional relationship with a Fellow within the AACP. This bond will allow each student to be exposed to the knowledge and experience that each of our Fellows possess along with an important networking opportunity. This program will help students both in their first years in the profession as well as throughout their careers.

The AACP Student Society is very excited to offer both programs to all students and we look forward to developing a professional relationship with each of them.

Please feel free to contact ***Richard Melchior****, AACP Student Liaison Committee Chairman, with any questions and/or concerns you may have regarding the programs at melchiorr@email.chop.edu or 267-973-1951.*

2013 Annual Academy Meeting

Los Angeles, California
January 24 - 27, 2013

Thursday, January 24, 2013

9:00 AM – 1:00 PM	Council Meeting
10:00 AM – 3:00 PM	REGISTRATION
2:30 PM – 4:30 PM	Fireside Chats
	Computers in perfusion, assisted bypass, electronic records & data management
	ECMO, the old, the new, the next generation
	"Students Only" Forum
	Troubleshooting venous return, VAVR, air & emboli, peripheral cannulation & more
4:30 PM – 5:30 PM	REGISTRATION
5:00 PM	Opening Business Meeting
	Fellow, Member, Senior and Honorary Members
5:30 PM – 8:00 PM	Sponsor's Hands-On Workshop & Reception

Friday, January 25, 2013

7:00 AM	REGISTRATION
8:00 AM – 9:30 AM	Scientific Session
9:30 AM – 10:00 AM	Break
10:00 AM – 11:30 PM	Scientific Session
11:30 PM – 1:00 PM	Lunch
1:00 PM – 3:30 PM	Special Scientific Session (Panel)

Myocardial Protection

Co-Moderators: James MacDonald, CPC (R) and Kevin Charette, CCP

Myocardial Protection: Historical Perspective - Gerald Buckberg, MD

Del Nido Cardioplegic Solution - Stacy O'Blenes, MD

Hyperpolarizing Vs Depolarizing and Additive Effects - Geoffrey P. Dobson, MS, PhD, FAHA

Custodial HTK Solution - Claus Preusse, MD, PhD

3:30 PM – 5:30 PM Fireside Chats
Hemostasis management: blood use, anticoagulation, heparin resistance and more
Mechanical and assist therapies, VADs , nitric oxide, IABP
Pediatrics, is your circuit small enough, they are not just little people
Perfusion safety, implementing protocols, accidents and outcomes, disasters and anecdotes

6:30 PM Induction Dinner
Fellow, Senior, Honorary Members & Guests

Saturday, January 26, 2013

7:00 AM REGISTRATION
8:00 AM – 9:30 AM Scientific Session
9:30 AM – 10:00 AM Break
10:00 AM – 11:30 AM Memorial Session
Update on GME - David Stump, PhD
Charles C. Reed Memorial Lecture
Dr. Vladimir Kucera - Czech Republic
Thomas G. Wharton Memorial Lecture
Linda B. Mongero, CCP - President, AACP

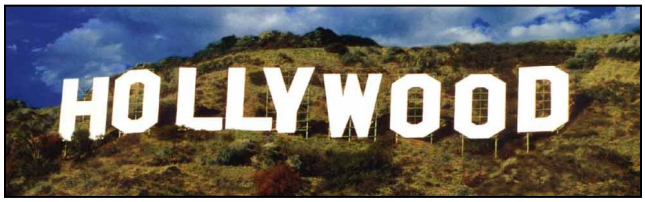
11:30 AM – 1:00 PM Lunch
1:00 PM – 3:30 PM Special Scientific Session (Panel)
ECMO Update
Co-Moderators: John Toomasian and William Harris, CCP
ECMO Update:2013 - Robert Bartlett, MD
Cannulation and Anticoagulation - D. Scott Lawson, MS, CCP
Ambulatory ECMO - Matthew Bacchetta, MD
ECMO for Respiratory Failure: What's New? - Daniel Brodie, MD

3:30 PM – 5:30 PM Fireside Chats
Communicating with the generations
Expanding the role of perfusion: Cath Lab, EP Lab, ER, etc.
Myocardial protection strategies
Women in Perfusion

5:30PM Closing Business Meeting
Fellow, Senior and Honorary Members Only

Sunday, January 27, 2013

8:00 AM – 10:00 AM Scientific Session
10:00 AM – 12:00 PM Fireside Chats
Budget management techniques, cost savings, administration
Managing perfusion, leadership issues, team building
Patient management, "What pressure, flow, temperature, etc. are we good?"
Publish or perish, reading the literature, preparing a presentation



The Student Section

Where do you place the air bubble detector?

Tyler Kelting BS

SUNY Upstate
Syracuse, NY

Where do certified clinical perfusionists (CCPs) place the air bubble detector (ABD) during the conduct of cardiopulmonary bypass (CPB)? This is the question I began to ask as I went on clinical rotations and observed a wide variety of very different locations used by CCPs. As a student, I was curious to know if there was any consensus on ABD placement and to understand the rationale for the specific placement on the circuit.

To answer these questions, we conducted a comprehensive survey of the perfusion community (Table 1) to determine where the positional use of the ABD was and the justification behind it. The results showed that the routine use of ABD during CPB was reported by 96.8% of CCPs. Of these, 74.6% set the ABD to servo-regulate the arterial pump, while 25.4% use the device in audible alarm only mode (Table 1). The responses regarding the specific placement of the bubble detector is displayed in Figure 1. These locations were claimed to be standardized at their departments by 89.2% of CCPs (40.1% written protocol & 48.8% unwritten consensus), while 11.0% stating that ABD placement not standardized at their institution. Users of an oxygenator with an integrated arterial line filter (ALF) tended to place the ABD in locations before the oxygenator inlet when compared to CCPs who use standard oxygenators (51.9% vs. 36.1% respectfully).

Survey Stats	
Time Frame:	
June - August 2011	
Participants:	
559 C.C.P.s	
40.7% Chief Perfusionists	
52.4% Staff Perfusionist	
6.9% Other	
Methods:	
On-line survey	
www.surveymonkey.com	

TABLE 1

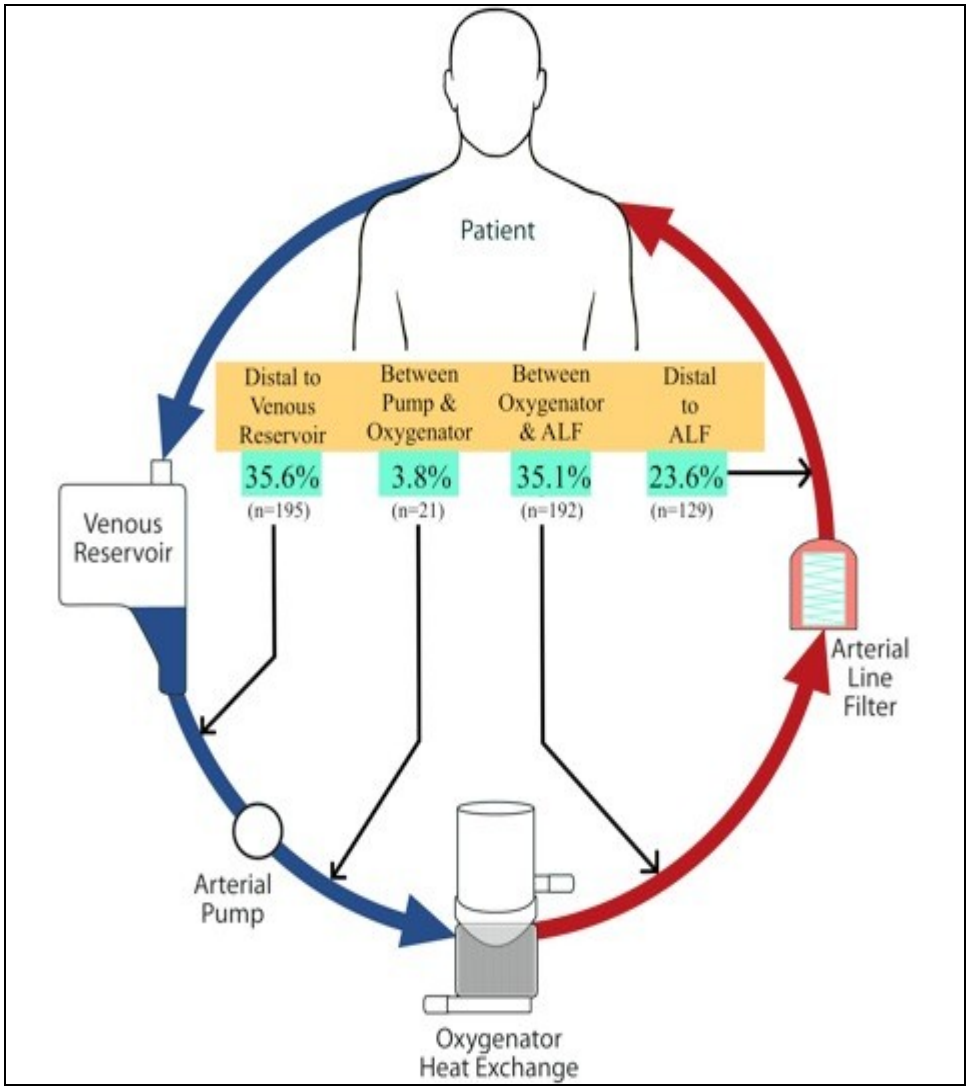
There were no major differences in ABD locations when comparing centrifugal pump to roller pump users. Primarily pediatric CCPs tended to place the ABD distal to the arterial line filter more often than their adult only counterparts (33.0% vs. 19.0%).

The positional rationale of the ABD varied among CCPs. Those placing the ABD distal to the venous reservoir (35.6%) predominately argued that an emptied venous reservoir was the most likely place to introduce air into the circuit and, therefore, was the best location. Those who placed the ABD between the oxygenator and arterial

line filter (35.1%) commonly reasoned that this placement protects against air exiting the membrane and also allows removal of air through the ALF purge. Those placing the ABD distal to the ALF (23.6%) most often cited that this protects the patient from all possible entry points of air. Several CCPs stated that the use of multiple ABD would be ideal to allow for more than one placement location. A false alarm from an ABD during a case (within the last 2 years) was reported by 36.1% of CCPs.

Author	Publication Year	Response	Bubble Detector Use
Kurusz, M[1]	1986	524	47.9%
Mejak, B[2]	2000	552	87.8% (63%)
Kelting, T	2012	559	96.8% (74.6%)

TABLE 2. Parenthesis indicate ABD linked to arterial pump servoregulation.



There are two principle findings of this study; (1) the use of ABD by CCPs during CPB is now nearly universal (96.8%) and (2) there is currently a lack of consensus by CCPs on the specific ABD placement on the circuit. The respondents offer strong rationales for the different ABD placements and this may suggest that the adoption of multiple ABD may offer the greatest comprehensive protection against air emboli. As medical technology changes, a future follow-up survey is recommended to see the transformation of use and placement of bubble detectors in the field of perfusion.

Kurusz M, Conti VR, Arens JF, Brown JP, Faulkner SC, Manning JV Jr. Perfusion accident survey. Proc Am Adad Cardiovasc Perfusion 1986; 7: 57-65.

Mejak BL, Stammers A, Rauch E, Vang S, Viessman T. A retrospective study on perfusion incidence and safety devices. Perfusion 2000; 15: 51-61.

This study was presented at the AACP's 33nd Annual Seminar on Cardiovascular Perfusion, 2012, New Orleans, LA. The full manuscript has been submitted to the journal ***Perfusion*** *for review.*

The Student Section

Review of Adenosine Enhanced Cardioplegia

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Abstract

Adenosine enhanced cardioplegia has been gaining momentum as an arrest and additive agent since the early 1990s. Several studies have been published investigating whether adenosine is beneficial, but results have been inconclusive. However, many papers have compared cardioplegia on an unbalanced scale, where clearly the cardioplegia technique may affect the effectiveness of adenosine as a cardioprotective agent. This review provides a short synopsis of adenosine in terms of chemistry, as an arrest and additive agent, and provides some insight as potential reasons why adenosine cardioplegia efficacy varies.

Introduction

A variety of medications may be administered as part of cardioplegia, such as adenosine. The use of this particular drug is not currently standard. However, there is evidence that adenosine cardioplegia has protective effects on the heart. According to the FDA, the use of adenosine is for fast acting treatment of cardiac arrhythmias, such as supraventricular tachycardia (1). Since the early 1990s, adenosine has gained popularity in the perfusion community as an additive and arrest agent. Many manufactures have not tested the efficacy of adenosine as a cardioplegia arrest or additive agent despite the rising prevalence in perfusion. As more studies are being published, it is evident that there is not yet a perfect formula for adenosine enhanced cardioplegia. Several clinical studies have indicated adenosine can benefit patients in several ways; from preventing depletion of cardiac energy supplies, to faster cardiac arrest and swift resumption of cardiac activity post -cross clamp (2-5). Conversely, there are many studies that conclude adenosine offers no advantage to myocardial protection or recovery (6). On a closer analysis, it is apparent that adenosine cardioplegia techniques varied widely, and must contribute to the inconsistent results.

Chemistry

The chemical properties of adenosine advocate the potential advantages as a cardioplegia additive. Chemically, adenosine is simply an endogenous nucleoside, synthesized in the degradation process of the cell's main energy source; adenosine triphosphate (7). Adenosine has numerous physiological actions, depending on the receptor cell type. In the coronary vessels, adenosine binds to receptor type 2A and 2B, which initiates muscle relaxation that dilates the coronary arteries (8, 9). Activation of the 1A receptor in cardiac smooth muscle excites potassium related ATP channels to open, causing an influx of potassium that hyperpolarizes the cell membrane (7-9). The sinoatrial node has A1 receptor sites that respond with a negative chronotropic and dromotropic effects to reduce spontaneous firing of action potentials (8). Furthermore, the A1 receptors also respond with negative inotropic and anti-beta adrenergic effects of adenosine (9). The half-life of adenosine is extremely short, a mere 10 seconds, as the drug is quickly converted to inosine by enzyme adenosine deaminase (8, 9). The effects on the coronaries, smooth muscle and pacemaker cells would be advantageous in cardioplegia to preserve myocardial function (7-9).

Adenosine as an Additive

Adenosine as an additive has many potential advantages. First, adenosine

has been shown to slow the rate of intracellular calcium loading, which is beneficial to prevent the calcium paradox (10). Mentzer published a study in 1999 demonstrating adenosine is safe and well tolerated by patients undergoing coronary artery bypass grafting surgery (11). Likewise, the study indicated patients that received adenosine cardioplegia required less inotropic support, had faster hemodynamic recovery, and less adverse events such as myocardial infarction, intra-aortic balloon support and death (11). Studies have indicated that adenosine has been linked to reduced ischemic injury due to cross clamp time and cardiopulmonary bypass, decreased ischemic reperfusion injury, preservation of myocardium energy supplies, and better global myocardial protection (2, 3, 5, 12). Adenosine cardioplegia has been linked to beneficial effects outside of myocardial protection. A study by Mentzer et al in 1996 demonstrated that adenosine cardioplegia decreased post-operative bleeding, and consequently transfusion requirements for cardiac patients (13). Current understanding of adenosine cardioplegia benefits may only be the tip of the iceberg. Future studies pinpointing an ideal protocol may reveal other benefits and the full value of adenosine as an additive.

Adenosine as an Arrest Agent

As an arrest agent, adenosine has been shown to reduce time to arrest, faster resumption of cardiac activity post cross clamp, and improved post ischemic recovery (14). In a 2004 study with Dobson and Jones, adenosine was shown to be as effective as a hyperkalemic solution (14). Potassium channels are activated by adenosine receptors, causing a hyperpolarization, which is the natural resting state the heart assumes (14). The arrested heart in a hyperpolarized state may reduce cellular destruction by slowing metabolic processes that would be occurring faster in a depolarized arrest state (14). The authors revealed that hyperkalemic cardioplegia is linked to arrhythmias, microvascular damage, and left ventricular dysfunction, all of which have not been linked to adenosine use (14). In addition to adenosine preventing the destruction caused by potassium, adenosine offers protection from ischemic reperfusion injury (14). The experimental method was a normothermic state with ischemic time of 20 minutes between cardioplegia doses (14). Cohen published a study that stated adenosine as an arrest agent produced a “predictable and sustainable hyperpolarized cardiac arrest that is reversible by reperfusion” (15). The study concluded electrical and mechanical function was completely persevered with no ischemic damage due to adenosine cardioplegia when compared to a standard hyperkalemic induced arrest (15). Many studies have used adenosine in conjunction with hyperkalemic arrest agents, which demonstrated adenosine decreased the speed of depolarization and intracellular loading of calcium (5). Adenosine clearly offers potential advantages as a cardioplegia arrest agent.

Methods of Administration

There are many influential factors contributing to the effectiveness of adenosine enhanced cardioplegia. Some studies determined ischemic preconditioning with adenosine is perhaps the most advantageous method (16, 17). However, some studies have examined the use of adenosine just in the initial and final doses (18); while other studies had adenosine in every cardioplegia dose (11). Ratio or microplegia methods differ in concentration and rate of adenosine delivered to the myocardium, which could make a difference when determining if adenosine benefited the patient. The ideal dosage of adenosine has not been thoroughly tested. Several studies have examined the safety and tolerance of adenosine, such as a study by Lee et al in 1995 (6). Despite the important conclusion of adenosine doses that were safe and well tolerated by patients, the study had the limitation of only examining patients undergoing three vessel coronary bypass grafting (6). Perhaps patients undergoing valve repair or replacement would require a different dosage of adenosine for cardioprotective effects as compared to patients having only myocardial revascularization. The difference between antegrade and retrograde add even more variability to myocardial protection. It is known that retrograde delivery does not completely perfuse the right ventricle, preventing global protection of the myocardium (19).

Temperature of delivery is another factor that possibly plays a role in adenosine efficacy. A study done by Mentzer in 1999 administered cold adenosine cardioplegia that exhibited a statistical difference, however a study by Ahlisson in 2011 revealed cold adenosine cardioplegia had no difference as compared to a placebo (7, 11). Additional factors that contribute to cardioplegia variability are temperature, cardioplegia volume, ischemic time between cardio-

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plegia doses, total cross clamp time, dosage of additives, and rate of adenosine administered. All studies done on adenosine enhanced cardioplegia vary on these factors, and this contributes to limited reliable statistical results.

Conclusion

The studies published in the last two decades have come to varying conclusions about the benefits of adenosine; quite possibly because of different surgeon protocols, and most importantly, inconsistent technique of cardioplegia administration, dosing, and composition (5). Because current data have been collected from studies that were not well controlled, further research is needed to explore the valuable effects of adenosine during cardiac surgery. Adenosine has shown some evidence to be advantageous as an arrest or additive agent. Undoubtedly there are benefits to adenosine cardioplegia based on the chemistry of adenosine alone, but the method for adenosine cardioplegia to maximize these advantages is not yet clear. Hopefully future research will demonstrate the ideal method of adenosine cardioplegia to provide better myocardial protection and faster resumption of cardiac active postoperatively.

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Abstract Deadline for the 2013 AACP Seminar is October 30, 2012

The Student Section

Tissue-Engineered Heart Valves: An Overview and Update

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Abstract

There are approximately 300,000 heart valve replacements performed around the world each year, this number is expected to triple by 2050. Tissue-engineered heart valves (TEHV) have the potential to overcome many of the short falls of current valve replacement options, including the need for life-long anticoagulation therapy, valve degeneration or calcification, and the inability to repair, grow or remodel. TEHVs are autologous living tissue valves created from a scaffold material seeded with autologous stem cells. Creating these valves is not simple task due largely to the complexity of the native heart valve tissue structure and composition. Many challenges need to be overcome before TEHVs become clinically available; however, the field has come a long way in the last 20 years.

Introduction

There are approximately 300,000 heart valve replacements performed around the world each year, this number is expected to triple by 2050 (1, 2). Of the 90,000 valve replacements done in the US each year approximately 43% involve mechanical valves, 50% porcine or bovine tissue valves, and 7% are human tissue valves, allografts or due to Ross procedures (3). The thrombogenicity of mechanical valves requires lifelong anticoagulation therapy which increases the risk of hemorrhagic complications. Tissue valves do not require anticoagulation, however, they are prone to progressive degeneration often caused by calcification which leads to structural failure and requires subsequent surgeries within 10-15 years. Current valve options have led to decreased mortality and morbidity for heart patients around the world. However, these current options fall short when compared to the native healthy heart valve. These inadequacies are especially prevalent in the care of the nearly 20,000 children born each year with congenital heart defects (3). The ideal graft would work lifelong without further intervention, be easily implantable, have perfect blood flow properties, would not require anticoagulation, would not degenerate, be noninflammatory, be non-thrombogenic, and most importantly be capable of growth, repair, and remodeling (1, 2, 4). The tissue-engineered heart valve (TEHV) would, theoretically, be able to accomplish all of this with the use of autologous cells to create a living heart valve.

Natural Heart Valve Structure

To understand the challenges of TEHV creation, it is vital to understand the structure of the natural heart valve. The valve consists of three main layers of tissue; Ventricularis, Spongiosa, and Fibrosa, which are then surrounded on either side with endothelium. Ventricularis is located at the inflow surface of the valve, it is made up of dense elastin fibers and is mainly responsible for the ability of the tissue to recoil, or return to its original shape after opening. Spongiosa is the middle layer; it consists of a loose collagen scaffold and is responsible for accommodating for shear stresses and absorbing shock during the cardiac cycle. Fibrosa is located at the outflow surface; it is made of collagen and valvular interstitial cells (VIC) and provides strength and stiffness for bearing the diastolic stresses enduring when the valve is closed (2, 4, 5). Each of these layers is formed and remodeled to handle changes in stresses experienced by the valve. Endothelial cells, VICs, fibroblasts, smooth muscle cells and nerve cells are all involved in proper functioning of the native heart valve. The goal of TEHV is to form this same trilaminar histology in order to best recreate a healthy valve (1, 5).

Methods for TEVH Design

The general process for creating a TEHV has four basic steps: harvest progenitor cells, seed scaffold, proliferation and differentiation of cells, removal of the scaffold (1). Progenitor cells will ideally be harvested in the least invasive possible

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manner, after which they may be expanded in a tissue culture, or concentrated before being used to seed the selected scaffold. At this point the scaffold may be placed in a bioreactor to allow for cell proliferation and differentiation under desired conditions, or it may be placed directly in to a patient where the same process will occur. Over time as the cells grow and produce the appropriate extra cellular matrix (ECM) to create a functional valve the scaffold will be dissolved until only autologous tissue remains.

Scaffold Materials

The scaffold must provide a three dimensional structure to mimic the native layer of the valve, and allow for the seeding of the necessary cells into the appropriate layers. It must also be reabsorbable, noninflammatory when seeded with cells, and facilitate cell adhesion and proliferation as well as ECM production (1, 2). It is also important that the scaffold be able to maintain adequate hemodynamic competence once implanted and throughout in vivo remodeling until the autologous cells are capable of supporting this function. Natural and synthetic materials are being examined as possible scaffold materials for TEHVs. Natural materials include decellularized biological valves and fibrin or collagen bases. Synthetic materials include synthetic polymers such as: polyglycolic acid (PGA), polylactic acid (PLA), polyhydroxyalkanoates (PHA), and polyhydroxybutyrates (PDB) (1, 2). Due to problems with inflammation, fibrosis, and degradation of biological valves and unpredictable variations in cell densities of fibrin and collagen based scaffolds, biodegradable synthetic scaffolds are the main approach used in current research.

Cell Sources

Cultured cell lines, autologous cells, and donor cells have all been investigated as possible options for creating TEHVs. Due to risks of infection and rejection autologous cells are the focus of current research. Circulating endothelial progenitor cells (cEPC), mesenchymal stem cells (MSC), and amniotic fluid stem cells (AFSC) are all autologous cell options (1, 6). Circulating EPCs can be harvested directly from cord blood or the circulation after being mobilized form the bone marrow via vascular endothelial growth factor (VEGF) administration. The implantation of a pure scaffolding capable of attracting and catching cEPCs would allow for shelf-ready TEHVs at a lower cost compared to other methods of seeding scaffolds. MSC can be obtained via bone marrow aspiration, and have been shown to create a natural-like heart valve structure under certain circumstances (1). AFSC and cEPC of the cord blood would be ideal options for correcting congenital heart disease with minimal risk to the mother or fetus/ newborn.

Bioreactors

Bioreactors simulate native circulatory environment by applying stresses with pulsatile flow of a dynamic cell culture through the immature valve allowing it to grow and remodel as needed before implantation. In current studies, this process normally takes three to four weeks to develop an acceptable tissue formation. Bioreactors may not be a necessary step for some TEHVs that are intended to be implanted immediately after seeding; this relies on the body to act as an appropriate bioreactor in which this stage can occur (1, 2).

Where are we now?

To date, at least eight synthetic materials or combinations of materials, with a large variety of cells, have been implanted into lambs and sheep since 1995 (3). Since 2000, seven natural materials or combinations of materials, with a variety of cells, have been implanted into rats, beagles, and sheep (3). These in vivo animal studies have opened the door for human trials. TEHVs with autologous, xenogeneic, and allogeneic components have been implanted in pediatric and adult patients in the pulmonary position with promising results (1).

Table 1 consists of seven in vivo studies involving the implantation of TEHVs into various hosts. Though results are promising there are some recurring problems including: stenosis, regurgitation, and leaflet thickening. These studies have shown that cell proliferation and differentiation as well as ECM development and valve growth are possible with TEHVs. Many of these studies are hindered by small sample sizes and a limited time in vivo. Though imaging technology allows valve function and structure to be determined the valves must be explanted to study cell and ECM development, thus limiting the time allotted for the valves to develop in vivo.

Conclusion

For the thousands of children born each year with congenital heart defects, as well as the hundreds of thousands of adult each year facing valve replacement procedures, TEHV seems like a dream come true. By eliminating the need for anticoagulation therapy and diminishing the need for future replacement procedures, there is no doubt that TEHVs have the potential to revolutionize patient care around the world. However, the development of TEHVs into clinical practice still faces many challenges. Recurring themes of leaflet thickening, valve insufficiency, valve stenosis, and limited evidence of true valve growth and remodeling must be resolved before this dream can become a reality.

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Year	Author	Experimental Aspects	Results
1995-1996	Shinoka et al (7/8)	Pulmonary Leaflet implant	Severe stenosis when applied to a trileaflet design
2000	Stock, Soian, Hoerstrup (1)	In vivo pulmonary valve	Regurgitation, mild stenosis, leaflet thickening
2005	Sutherland et al (9)	Longest in vivo (8 months) Pulmonary valve	Minor regurgitation, leaflet thickening, small sample size
2006	Cebotari et al (10)	Seeded pulmonary allografts, 2 tetralogy of Fallot pediatric patients	Trivial regurgitation, valve diameter did increase with patient's somatic growth at 3.5 years post-operative
2007	Dohmen et al (11/12)	Porcine and allograft scaffolds, pulmonary position, ross procedure, 23 patients	No significant regurgitation, no calcifications, no thrombogenesis, 3.5-5 years follow-up
2010	Gottleib et al (13)	Largest in vivo study, 19 sheep, pulmonary valve	Regurgitation, leaflet thickening
2011	Weber et al (14)	Pulmonary valve, 6 Chacma Baboons	Cell growth and proliferation occurred, pilot study, short and small

Table 1. Summary of recent in vivo TEHV studies.

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Gottlieb D, Kunal T, Emani S, Aikawa E, Brown DW, Powell AJ, et al. In vivo monitoring of function of autologous engineered pulmonary valve. J Thorac Cardiovasc Surg 2010 Mar;139(3):723-731.

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PRE-REGISTRATION FORM

The 2013 Annual Meeting of
The American Academy of Cardiovascular Perfusion

MEMBER	**FEE**	**Amount**
Registration Fee	$340.00	_____
2012 Annual Dues	$145.00	_____
Adult Guest to Workshop	$25.00	_____
NON-MEMBER	**FEE**	**Amount**
Registration Fee	$400.00	_____
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STUDENT PERFUSIONIST	**FEE**	**Amount**
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***MUST** *include a letter from the school director with registration.*		
To take advantage of the Student rate of $30.00, you must be a current Student Member of The Academy.		
FELLOW or SENIOR MEMBER	**FEE**	**Amount**
Registration Fee	$400.00	_____
2012 Annual Dues	$170.00	_____
Guest to Induction Dinner	$100.00	_____
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FIRESIDE CHAT REGISTRATION
(make your first three choices each day)

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Choices will be assigned in the order they are received. Each Fireside Chat is limited to 30 attendees per session each day.

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Message From The Academy's Student Council President

To the Members of The Academy:

I am proud to announce the arrival of the first ever Student Council of the American Academy of Cardiovascular Perfusion! The AACP Student Council will be working with the Student Ambassadors from each perfusion education program to develop a voice from the Student Society within the Academy. The Student Ambassadors and the Student Council are looking forward to developing this relationship with the AACP Council and other members of the Academy.

Not only is this an exciting time for new students joining The Academy, but for long-time members as well. We are all members of this great professional society because of our enthusiasm for furthering research and education of our field. Knowledge is not meant to be stowed away, but shared with others in an altruistic manner. It is my hope that this Student Council will personify this idea and promote collaboration between perfusion students and experienced perfusionists for years to come. The Student Council will also give perfusion students a way to become involved at the earliest point of their career and establish their lifelong support of the AACP.

As President of the Student Council, I am especially honored to be a part of its beginning. A lot of work has gone into its creation and it would not be possible without the continued support of The Academy. I look forward to seeing its success in the future and its debut at next year's Annual meeting in Los Angeles!

Sincerely,

Seana G. Hall
Student Council President, American Academy of Cardiovascular Perfusion

INSTRUCTIONS and INFORMATION

o Complete each appropriate section of this form by printing or typing.
This form may be copied, but must include both pages.
o Members must pay their 2013 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 15, 2013--bring it with you to the meeting.
o No pre-registration will be processed after January 3, 2013.
-- **After this date you must register at the meeting**.
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 $145 for the 2013 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 12, 2013.

o **Make checks payable to AACP (US dollars). Mail completed pre-registration form and check to:**

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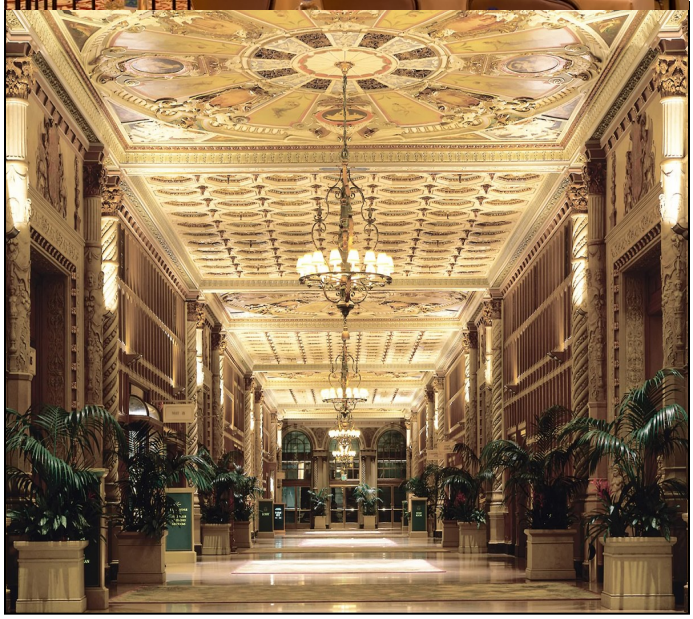
Reservations

800-245-8673
Single/Double Occupancy (One King Bed)
$179 per night

When making your reservations mention that you will be attending The American Academy of Cardiovascular Perfusion (AACP) Conference.



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The ACADEMY ANNUAL MEETING DEADLINES

ABSTRACT DEADLINE	**October 15, 2012**
MEMBERSHIP DEADLINE	**November 24, 2012**
PRE-REGISTRATION	**January 3, 2013**
HOTEL REGISTRATION	**January 3, 2013**
2013 ANNUAL MEETING	**January 24 - 27, 2013**

Others Meetings

21st Century Treatment of Heart Failure
Synchronizing Surgical and Medical Therapies for Better Outcomes
October 18-19, 2012
InterContinental Hotel & Bank of America Conference Center, Cleveland, Ohio
In cooperation with Cleveland Clinic Kaufman Center for Heart Failure and the American Association for Thoracic Surgery
Website: **www.ccfcme.org/heartfailure12**

Third International Congress on ECMO Therapy
Wednesday November 14, 2012
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