IN THE SPOTLIGHT: Too Little, Too Late

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I had just started my job as an Anesthesia Tech in 2011 when our patient died in the operating room. After a relatively minor procedure for removal of a jaw mass, we transferred the 55-year-old patient from the operating table to his bed. Noting circumoral cyanosis, the anesthesiologist resumed masking the patient, but the pulse oximeter showed continued desaturation. Checking the cable connection and calling other anesthesia personnel into the room delayed our journey to the cardiac cath lab, where an emergent procedure found complete occlusion of the patient’s LAD. The patient had died almost instantly, as soon as an embolus had lodged in his coronary artery, but this did not absolve the anesthesiologist overseeing the case. Afterward, the main focus was on why the patient’s EKG leads had been removed before being transferred to his hospital bed. I heard that a formal investigation followed, and that the patient’s family was devastated, but no party was deemed at fault because, in all likelihood, it was inevitable that the patient would experience a myocardial infarction eventually due to his atherosclerosis. In all likelihood.

Examples of medical error by omission are more rarely cited than those of commission [1]. Double-dosing of medication, operating on the wrong surgical site – such errors have a clear discernable origin, but this is not always so with errors of omission. The physician who administers too much medication provides a better teaching example than the physician who forgets it altogether, thus news articles and case reports tend to focus on the former. In fact, “errors” in which medical professionals apparently didn’t “do enough” aren’t always considered errors. This is because modern medicine is often tasked with preventing the natural history of disease, and lack of intervention might as well be the same outcome as an ineffective intervention. The anesthesiologist cited above might have monitored the patient’s EKG more closely; he or other health care providers caring for him could have taken a more thorough history of cardiac health; the OR team could have moved more swiftly to transfer the patient to the cath lab, but none of those things happened, and we don’t know how the outcome might have changed otherwise. So is this a case of medical error? We’ll never quite know, confounding the definition of medical error by omission, and further explaining why such examples are less often cited.

Regardless of fault, patients and their families want explanations when tragedies occur. Often times there are no explanations. If I was family to the patient with a jaw mass, I would have a hard time believing that his death was not directly caused by the surgery. Couldn’t someone have done more to prevent his death? How can we even tell the difference between the natural progression of disease vs. iatrogenic cause when the two overlap chronologically? These questions eventually erode patient faith in medical professionals, regardless of the outcome.
My mother has repeatedly told me that she doesn’t want me to become an emergency medicine physician. Her opinion stems from a 2006 visit to the ED in Burnet, Texas after she was bitten by a feral cat. Upon cleaning and bandaging this minor wound, the young physician commenced discharge from the ED. “Don’t I need a rabies vaccine?” asked my mom, but the doctor quickly dismissed her concern. Unassured, my mother contacted the State Health Department an hour later. The non-physician who answered escalated my mother’s fear, telling her that Burnet had the highest incidence of rabies in the state. Though no adverse outcome followed (my mom was vaccinated the next day in Austin), I am reminded constantly by my mother how incompetent and negligent that particular emergency doctor was. Without knowing the full details of the event or the protocol for vaccination, I have contended that “negligence” is an unfair and uninformed conclusion, but my mother remains steadfast in her criticism not only of that doctor but of the specialty as a whole.

In the ACA-era of overspending in healthcare, it sounds strange to emphasize the examples of medical error by omission. As previously stated, this under-emphasis is derived from the fact that less identifiable, less concrete and less detectible acts of omission. It is difficult to determine if an act of omission is indeed, a medical error, just as it is difficult to tell if a hospitalized patient’s death is attributable to their disease or some intervention. Nonetheless, those looking for answers – especially patients and their families, who physicians should concern themselves with most – cannot discern” errors of omission from errors of commission.” Once the unfortunate outcome is final, they only know that successive efforts are too little and too late to matter.

Disclosure statement: The authors have no conflicts of interest to disclose.

References:
STRATEGIC PLANNING UPDATE

In April 2015, ISHLT launched a strategic planning process to look at the direction the field is moving and to develop a new Strategic Framework to guide the Society over the next 5 years. The initiative is being led by a steering committee of six: Duane Davis, ISHLT President; Maryl Johnson, ISHLT President-Elect; Stuart Sweet, ISHLT Secretary/Treasurer; David Taylor, ISHLT Past President; Andreas Zuckermann, ISHLT Member; and Amanda Rowe, ISHLT Executive Director. The first phase of this process has been to gather data and best thinking from multiple sources primarily within the organization: ISHLT members, Board Members, and Council Members, as well as from a few leaders in the field. The collected data came from 404 responses to a membership survey, 15 one-on-one interviews with leaders in the field, and the engagement of the 11 Scientific Councils.

The output of this process was a Findings Report generated by the strategic planning consultant, Susan Meier. The Report summarized the consensus ideas expressed by many as well as some thoughtful ideas presented by individuals. In October, the Board of Directors, the Strategic Planning Task Force, and several past presidents and former Board members gathered in Durham, NC to participate in a half-day strategic planning retreat at which the Findings Report was explored and from which key areas of focus were identified. These areas of focus will serve as the basis for the work of a Strategic Planning Retreat in January 2016.

Some of the significant findings are shared with you below. Additional information will be shared following the January Strategic Planning Retreat.

Current Perceptions of ISHLT

Through all of the methodological avenues, a consistent and clear message was heard that ISHLT has been increasingly effective in recent years in providing platforms for education and networking for members to engage, learn, and exchange relevant and timely knowledge and practice. No one indicated that ISHLT has been focusing on the wrong types of work or issues or that there have been any crises or flagrant problems to note.

ISHLT’s reputation was uniformly praised, with a number of individuals expressing deep, long term affinity for the Society. A few interviewees expressed concerns about the challenges of expansion into developing areas (geographically and professionally) on the forefront of the Society’s mandate. Many others focused more on the challenge of being a truly integrated Society when the structure of the Society is reflective of more of a silo-ization by specialties.

There is a high level of satisfaction with the current mission and purposes of the Society. In terms of the value proposition of ISHLT membership, the single most valued offering is clearly the Annual Meeting. And, yet, a key challenge facing the Society is how to address the increasing demands to offer the highest quality program that satisfies a growing diversity of interests and specialties, especially given limited resources.

The Challenges ISHLT Faces

Continual, and at times dramatic, changes have become the norm in the environment in which we live and work. Associations and other organizations have learned that being adaptive to change is integral to remaining vital and relevant. This is further emphasized by the complexity and technological advances occurring in the advanced heart and lung disease field. Even though it is impossible to predict the future, it is essential that ISHLT leaders anticipate both the challenges and opportunities that are most likely to arise in future years.
Those who contributed to the information gathering process identified a number of challenges that ISHLT and/or the field in general will be facing over the next 10-15 years. The identified challenges offer both possibilities and strategic choices for ISHLT as it explores what its priorities should be in this increasingly complex field. How does the Society balance its unique ability to convene professionals involved with heart and lung transplantation with the respective interests of the many specialties within the field? How might the Society better attract and engage younger generations? How can ISHLT effectively extend its reach to parts of the world not well represented in its membership? How will ISHLT continue to distinguish itself from other societies? How can ISHLT best ensure long term sustainability with limited resources? These are just a few of the questions ISHLT is facing as it embarks on developing a strategic plan.

We look forward to the continued engagement of our members as we explore these challenges and opportunities. Thank you again for all of your input. We will keep you updated through the LINKS on the progress of this important process.
LAST CHANCE TO SUBMIT YOUR ABSTRACT

SUBMIT AN ABSTRACT

Deadline: November 3, 2015

Notification of Acceptance or Rejection of all submitted and finalized abstracts will be sent via email to the primary author in January 2016. For more information about abstract submission, visit ishlt.org/meetings/abstracts.asp

ISHLT Grants & Awards Applications Now Online – APPLY TODAY!

The 2016 Grants & Awards applications are now available online at http://ishlt.org/awards. The deadline for applications is Friday, January 15, 2016. Grants will be awarded at the ISHLT 36th Annual Meeting and Scientific Sessions, April 27-30, 2016 in Washington, DC.
Would You Take This Donor?

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Selecting the appropriate donor for a given recipient is perhaps the most crucial factor in determining post-transplant outcomes. Conferences, statements, and manuscripts abound yet still have not clarified the nuances of optimal donor and recipient matching.

With little data to guide us, we often formulate decisions based on prior experience and use the multiple hit theory to provide rationale when a donor just doesn’t seem right, “Well…it’s a female into a male, she used cocaine three years ago, and was hypertensive. One small hit is ok, but three small hits? Maybe we shouldn’t take it.” Then we poll additional colleagues, “Would you have taken this donor?”

The HFTX Council is launching a Donor Dilemmas blog to allow exchange of experience and expertise within the broader heart transplant community. We anticipate this to be a highly engaging and informative forum. Presenters will post a case only with donor and recipient information. Participants will be asked to discuss the case and vote on whether they would accept the donor. The outcome of the case will be posted after the voting period.

Bojan Vrtovec, Education Workforce Leader, and Michael Pham, Vice-Chair, are working diligently to develop this into an interactive and engaging learning experience. Video clips and real time voting are currently being investigated.

The launch for this site is set for December 1 and cases will be posted every other month. We hope all will participate in the discussions, and we are seeking brave volunteers to provide cases. These can be forwarded to Bojan Vrtovec (bojan.vrtovec@gmail.com).

Disclosure statement: The author has no conflicts of interest to disclose.
Desensitization Strategies In Heart Transplantation – Where are we now?

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Hyperacute rejection, although fortunately rare, is still the most feared complication after heart transplantation due to its rapidity of onset, severity and almost universally fatal outcome. The predominant risk factor for its development is the presence of circulating donor-specific cytotoxic antibodies in the recipient at the time of transplantation. Even in the absence of hyperacute rejection, sensitized patients are at increased risk of rejection, development of allograft vasculopathy and graft loss [1-3]. The challenge of the sensitized patient awaiting transplantation is that in order to avoid risk of rejection, the donor pool is limited to only compatible donors. This results in a prolonged and often prohibitive waiting time on the wait-list and a consequent increase in wait-list mortality [4]. Risk factors for sensitization include blood transfusion, infection, prior transplantation, prior surgery with homograft, presence of mechanical circulatory support, gender and race. Females, due to prior pregnancies, are twice as likely to be sensitized as males. There is also a higher rate of sensitization in African-Americans [5]. The number of patients active on the UNOS wait-list continues to increase [6] and there has been a co-incidental rise in the number of sensitized patients on the list such that about a third of the patients on the list are now sensitized [7].

The development of solid phase assays for the detection of circulating antibodies has been a major advance in the field. These technologies not only allow rapid high-resolution detection and specification of single HLA Class I and II IgG antibodies, but also quantification (strength) of these antibodies which correlates with in vitro cytotoxicity. This ability to detect individual potentially cytotoxic HLA antibodies has allowed the development of the “virtual” crossmatch, which has facilitated an expansion of the donor pool for sensitized patients by obviating the need to perform a prospective crossmatch.

Given the challenges faced by an increasing number of sensitized patients awaiting heart transplantation, a consensus conference was held to discuss the topic in 2009 [8]. Of 23 centers reporting, 362 (8%) of 4640 patients referred for heart transplantation underwent treatment for sensitization using a variety of therapies. On average, 45% of treated sensitized patients had a significant reduction (>50%) in circulating antibodies and 73% of treated sensitized patients underwent successful heart transplantation.

There have been no prospective randomized studies to determine the most effective treatment for sensitization. Desensitization therapies often involve the use of combined strategies involving antibody removal (using plasma exchange or immunoabsorption), therapies to alter antibody production by B cell modulation (rituximab), plasma cell depletion (bortezomib or carfilzomib) or immunomodulation with intravenous immunoglobulin (IVIG). Peri-operatively, T-cell responses may
be suppressed by cytolytic therapy (antithymocyte globulin) and complement blockade with eculizumab is currently under investigation.

Retrospective studies do show that these therapies are often effective at reducing antibody burdens sufficiently to allow successful cardiac transplantation with acceptable outcomes. In a study of 21 patients awaiting heart transplantation with panel reactive antibodies (PRA)>10% treated with combination therapy including plasmapheresis, intravenous immunoglobulin and rituximab, circulating antibody levels decreased from a mean PRA of 70% to 30% [9]. Patients subsequently had a negative donor-specific cross-match and underwent successful heart transplantation. Compared to an untreated sensitized cohort and unsensitized control group, treated sensitized patients had similar five year survival and freedom from cardiac allograft vasculopathy, although there was a significantly lower freedom from any treated rejection in the first year compared to the other groups. However, despite these promising results, many patients remain refractory to these therapies. In a follow up study of seven patients who did not respond sufficiently to the above regimen, the combination of plasmapheresis and bortezomib further reduced calculated PRA from 62% to 35% [10]. In an extended cohort of 30 patients [11], plasmapheresis and bortezomib was effective at reducing HLA antibody burden in a majority of sensitized patients, including patients with high levels of antibodies as determined by 1:8 dilution or complement binding ability (C1q). Patients who received prior desensitization therapies with plasmapheresis. IVIG and rituximab appeared to have a greater response suggesting combination therapies appear to be more effective. The majority of patients were able to undergo transplant with excellent one-year survival and low treated rejection rates. One-year actuarial freedom from treated infection was 33% and other adverse effects were infrequent. These data appear congruent with similar results in the renal transplant literature to suggest that desensitization therapy is effective but requires combination therapy for greatest efficacy. There remains a long road ahead to satisfactorily address this increasingly challenging group of patients and further studies are urgently needed to determine optimal efficacy and long term outcomes. To address several issues in sensitization, a consensus conference on circulating antibodies in heart transplantation is planned for April 2016.

Disclosure statement: The author has no conflicts of interest to disclose.

References:


Cardiogenic Shock - In the Era of Advanced Short Term Mechanical Circulatory Support

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If not treated in a sufficient and timely manner cardiogenic shock (CS) is known to have still a high morbidity and mortality instead of improvements in the therapy eg of acute myocardial infarction (MI) and CS [1].

In 1967 Killip classified the severity of derangement after myocardial infarction into four groups: no heart failure, heart failure, severe heart failure with frank pulmonary edema, and cardiogenic shock (CS): signs including hypotension with a systolic blood pressure (BP) 90 mmHg or less, evidence of peripheral vasoconstriction: oliguria, cyanosis, diaphoresis, and heart failure - often with pulmonary edema [2].

The Shock Trail confirmed CS complicating acute MI by clinical and hemodynamic criteria which have been used since then. Clinical: hypotension (systolic BP <90 mmHg for at least 30 minutes or the need for supportive measures to maintain a systolic BP of >90 mmHg) and end-organ hypoperfusion (cool extremities or a urine output of <30 ml per hour, and a heart rate of >= 60 beats per minute – paced rhythm included). Hemodynamic criteria: cardiac index (CI) of no more than 2.2 l/min/m2 of body-surface area and a pulmonary-capillary wedge pressure (PcW) of at least 15 mmHg. Thrombolytic therapy and intraaortic balloon counterpulsation were recommended [3].

Over time new aspects have been added into the assessment of CS: change of mental status, presence and degree of lactic acidosis, systemic inflammatory response syndrome (IL-6, TNF-a, NO)[4,5,6,7].

Acute myocardial ischemia is only one of many reasons that a patient may end up with a profound CS refractory to conventional therapy (PCSref). PCSref can have many etiologies presenting as low- or high-cardiac output syndrome compromising macro- and microcirculation with severe reversible or irreversible multi organ failure and finally causing a patient’s death [5,8,9].

Our therapeutic tool box should provide the right tool ready for immediate use in our proactive attempt to break the vicious cycle to reestablish a sufficient circulation with tissue perfusion and oxygenation. In PCSref time is tissue especially brain tissue.

So which device should be at the top of possible recommendations if time is a crucial component for successful immediate effective circulatory support in true cardiogenic shock like PCSref to safe a
patient’s life and organ function, to gain circulatory stability and time for detailed diagnostic and specific therapies like coronary revascularization? Such a device should be easy to be initiated when and where ever needed (inhospital and in out-of-hospital scenarios) in patients with PCSref without the risk to lose time and tissue by moving these patients to the cath lab or operating room from one hospital to another or the Critical Care Unit, emergency room or even the scene in the field. Compared to other percutaneous inducible devices the veno-arterial extra corporeal membrane oxygenation (VA-ECMO) seems to be the device of choice in patients with PCSref – where minutes count - because tissue (brain) is the major concern [10,11,12].

VA-ECMO seems to be the quickest applicable and most effective tool to restore full circulatory support with 4 to 5 l/min and to provide sufficient tissue oxygenation in patients with PCSref. In those patients VA-ECMO serves as a bridge-to-decision and bridge-to-treatment device. Reported data mostly from utilized older technologies and less advanced critical care settings reflecting real life usage not generated by randomized trials [13].

Any delay to reestablish a sufficient circulation eg due to an additional transport from place A to B will add higher risk to the patient’s odds. A door-to-ECMO implantation time <30 minutes significantly improves the 30-day outcome in patients with out-of-hospital cardiac arrest [14,15].

The use of nondurable mechanical support, including the use of percutaneous and extracorporeal ventricular assist devices has recently been recommended as Class IIA in the 2013 ACCF/AHA Guideline for the Management of Heart Failure [16]. The definition of cardiogenic shock has been modified in the 2015 SCAI/ACC/HFSA/STS Clinical Expert Consensus Statement together with a statement that in cases of biventricular failure VA-ECMO is the mechanical circulatory support device of choice for patients in cardiogenic shock and impaired oxygenation [17].

Nevertheless way to go: current CS classifications and definitions, especially for PCSref do not classify sufficiently the different aspects and possible therapeutic consequences in regard to “which device when” related to: underlying etiology of the PCSref with expected or observed cytokine release, immune response, metabolic derangements instead of one, two or more inotropic substances, the degree of mitochondrial and/or neuro hormonal involvement, and reversibility of end-organ dysfunction: brain, lungs, kidneys, liver, etc.

Disclosure statement: Dr. Michael M. Koerner has no conflicts of interest to disclose. Dr. Aly El-Banayosy reports receiving speaker fees from Thoratec, Inc., Pleasanton, CA, USA.

References:


Updates from the Pathology Council: Now, Our Precious Efforts are CLAD in AMR!

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The Pathology Council has been busy in 2015! As in recent years, antibody-mediated rejection (AMR) has remained a strong focus of our Council’s discussions and research efforts. Over the last year, we have seen a number of important clinical studies from members of our Council and others, further refining our understanding of the biology of AMR in the heart and lung, and the utility, reproducibility, and limitations of various biopsy modalities for diagnosing AMR. There is also considerable and growing interest in the pathology of chronic lung allograft dysfunction (CLAD), and “mixed rejection” in the heart. These topics will likely remain in focus over the coming year and beyond.

In this modern era of information overload, it can sometimes be challenging to stay up-to-date while maintaining a busy clinical practice. In the spirit of aiding our Council members and others to stay abreast of new developments in our rapidly evolving field, a few of the key pathology publications from the last year are highlighted below, although an incomplete list, as many other studies worthy of review.

Progress in Heart Transplantation

Data on cardiac AMR and mixed rejection continues to accumulate. Although macrophage accumulation within capillaries is a well-recognized feature, the significance of other inflammatory cells in cardiac AMR is poorly understood. It remains unknown whether mixed rejection is a distinct entity, or simply the sum of acute cellular rejection (ACR) and AMR, as this issue continues to be hotly debated.

In February, our European colleagues with the Association for European Cardiovascular Pathology (AECVP) published a multi-institutional pilot study evaluating inflammatory infiltrates in cardiac AMR (http://www.ncbi.nlm.nih.gov/pubmed/25612500). In their study, inflammatory burden was a constant feature of AMR and correlated with AMR grade, C4d positivity, and DSA positivity. In addition, T-cells and macrophages were observed in equivalent numbers in AMR, both within and outside the vasculature. This interplay between inflammatory components led this group to conclude that mixed rejection may represent a distinct entity.

On the other hand, data from the UTAH Cardiac Transplant Program (recently accepted for publication in JHLT, soon to be released electronically) suggests that mixed rejection may simply represent the sum of ACR and AMR occurring concomitantly, rather than a distinct entity. In mixed rejection, some interplay was observed over time between cellular rejection and AMR scores, although this interplay was unequal, with the course of AMR being more likely to proceed independently, leading the authors to favor the "sum of two" hypothesis.
For now, the nature of mixed rejection remains unsettled, but hopefully ongoing efforts will resolve these questions in the near future. Regardless, we can look forward to stimulating discussions with our colleagues on this controversial topic at the ISHLT Annual Meeting next spring!

Other interesting reads include a recent study of ectopic lymphoid structures surrounding epicardial coronary arteries in cardiac allograft vasculopathy (CAV), further suggesting an immunologic mechanism in the pathogenesis of CAV (http://www.ncbi.nlm.nih.gov/pubmed/25655346), as well as a study suggesting that phosphorylated S6 kinase and S6 ribosomal protein may represent useful adjunctive diagnostic markers for cardiac AMR (http://www.ncbi.nlm.nih.gov/pubmed/25511749).

And lastly, for those of us asked to evaluate liver biopsies from patients with long-standing heart failure as part of a pre-cardiac transplant work-up, a recent paper in Modern Pathology (http://www.ncbi.nlm.nih.gov/pubmed/25793895) may be of interest. What is the significance of bridging fibrosis in these patients, you ask? In this study, the authors examined liver biopsies and explants in this context, and suggest that patients with bridging fibrosis may still be viable candidates for isolated heart transplantation, as the pattern of fibrosis due to passive congestion is highly variable. The take-home message is that a diagnosis of cirrhosis should only be made with great caution in such a biopsy.

**Progress in Lung Transplantation**

As in the heart, data on pulmonary AMR continues to accumulate as we seek to understand this phenomenon and define the most reliable modalities for its diagnosis. The anecdotal experience of most members of the Council is the infrequent demonstration of C4d staining of interstitial capillaries in the setting of circulating de novo DSA and clinical evidence of graft dysfunction. The reason for the relative insensitivity of C4d in the lung is unclear, but is likely multifactorial including technical, interpretative, and mechanistic considerations.

Late last year, Anja Roden and her colleagues at Mayo Clinic published their experience with C4d in lung allografts, examining the reproducibility of scoring and correlation with clinical findings and DSA status (http://www.ncbi.nlm.nih.gov/pubmed/25149365). Not surprisingly, reproducibility was suboptimal among the four pathologists, validating what we all suspected, although the reproducibility of immunofluorescence scoring was superior to that of immunohistochemistry.

Not to be outdone, the same group continued their momentum and published the first series comparing transbronchial cryobiopsies to conventional transbronchial biopsies for evaluation of lung allografts (http://www.ncbi.nlm.nih.gov/pubmed/26488148). As expected, they observed that cryobiopsies tended to be larger, with less crush artifact, although complications tended to be more frequent. Reassuringly, there was no significant difference between the types of biopsies with respect to the reviewers’ agreement on grades of rejection. Certainly more studies on cryobiopsies are needed, and it remains to be seen which modality will be favored in the coming years.
AMR hasn’t been the only hot lung topic in 2015. The lung session at the recent Banff allograft pathology meeting in Vancouver, co-chaired by Carol Farver and Dean Wallace, focused on the pathology of chronic lung allograft dysfunction (CLAD). Our understanding in this area has been relatively underdeveloped, likely due to the deficiencies of the transbronchial biopsy to evaluate features of CLAD. The session looked at the current state of pathology of obstructive chronic rejection (BOS), restrictive chronic rejection (RAS), and chronic vascular changes. The summary conclusion was a proposal for a multi-institutional study utilizing allograft explants to begin to address some of the outstanding issues in this area and improve our understanding of CLAD. The lung session for the next Banff meeting, scheduled for 2017 in Barcelona, will be co-chaired by David Hwang and Elizabeth Pavlisko. For those looking for something a little sooner, CLAD will also be a key topic at the upcoming ISHLT Annual Meeting this spring.

Other News

One of our ongoing challenges in transplant pathology remains dissemination of knowledge about the practical application of rejection criteria to practicing pathologists on the “front lines,” many of whom are not ISHLT members and most of whom evaluate these specimens as a small component of their broader duties in surgical pathology. In recent years, members of the Pathology Council developed an online tutorial on cardiac ACR and AMR for pathologists, in partnership with the Society for Cardiovascular Pathology and Association for European Cardiovascular Pathology (http://scvp.net/acr/index.html).

This online tutorial has been well received, with nearly 25,000 page views and over 20,500 unique page views in 2015 alone, and the tutorial pages are among the top hits on the SCVP website overall. To expand upon these educational efforts, this year a quiz component has been added to the ACR tutorial, and a similar quiz component is planned for the AMR tutorial soon. This resource continues to undergo updates, and we welcome feedback and suggestions from anyone who uses it.

2016 Annual Meeting & Scientific Sessions in Washington, DC.

Please join us for the following pathology-oriented sessions in Washington next spring:

Unraveling “Chronic Rejection” in the Heart, where the controversy and ambiguity surrounding chronic rejection in the heart will be discussed. What does “chronic” really mean? And are the changes we observe truly rejection? Speakers in this session will address the myocardial alterations (beyond CAV) seen after years of repetitive rejection episodes, novel mechanisms of CAV development, the role of complement and other mechanisms in potentiating late graft damage, analogues of chronic rejection in other organs, and animal models of late graft loss.

Controversies in Heart Transplantation: Past, Present and Future, where the current status of rejection surveillance and utility of endomyocardial biopsy will be hotly debated.

Big Data to Answer Big Questions: Biobanking to “Omics” to Personalized Medicine in Thoracic Organ Transplantation, where renowned investigators will discuss the process of
biobanking and proper utilization of banked specimens for research, and the promise of Big Data as a tool that will revolutionize future clinical practice.

**A 2016 Focused Update on AMR in Cardiac Transplantation: Immunologic Diagnostics and the Treatment of Refractory AMR**, where the current state of cardiac AMR will be addressed, including a discussion of endomyocardial biopsy features in AMR and emerging molecular technologies for detection of AMR.

**Endotypes of CLAD and Novel Treatment Strategies**, where our current understanding of the different CLAD phenotypes will be reviewed, including their clinical diagnosis, radiology, pathology, prognosis, and treatment.

For more details about these and other exciting symposia, please visit the 2016 Preliminary Program at [http://www.ishlt.org/meetings/annualMeeting.asp](http://www.ishlt.org/meetings/annualMeeting.asp).

See you all in Washington!

Disclosure statement: The author has no conflicts of interest to disclose.
THE ASPEN LUNG CONFERENCE 2016: “Lung Transplantation: Opportunities for Repair and Regeneration” June 8-11, 2016 @ The Gant Conference Center, Aspen, Colorado

Dear ISHLT membership:

We are pleased to announce that the 59th Thomas L. Petty Aspen Lung Conference will be devoted to lung transplantation! The Aspen Lung Conference is one of the most respected and innovative meetings in the North American lung research community at large: With a focus on clinical problems affecting the lung, the Aspen Lung Conference blends cutting-edge basic and clinical research in a setting that facilitates extensive discussion amongst participants. This is the very first time that lung transplantation will be the topic of discussion. The conference will be organized around a central theme of the life cycle of a lung transplant, spanning donation, explantation, preservation, implantation and finally accommodation. Emphasis will be placed on integration of basic, translational and clinical sciences. The program will be organized into a series of thematic sessions focusing on (i) new concepts in lung allograft preservation and reconditioning utilizing ex-vivo lung perfusion, (ii) adaptive immunity including new arenas in T cell and B cell biology, (iii) host response/innate immune mechanisms, (iv) airway/allograft remodeling and (v) strategic approaches to translating scientific advances into meaningful therapies including molecular phenotyping, novel immunosuppression and development of novel diagnostic and monitoring techniques. The overall objective is to assemble thought leaders and learners of transplantation to educate the next generation of scientists and define the next big steps to be taken in the field. We hope many of you will consider submitting an abstract (which can overlap your ISHLT, AST, or ATS submissions). Abstract deadline is February 14, 2016. For more information, contact: Jeanne Cleary, E-Mail: Jeanne.Cleary@ucdenver.edu or visit our website at www.aspenlungconference.org. We hope to see you in Aspen!

Martin Zamora (Chair)
Mark Nicolls & Tereza Martinu (Co-Chairs)

Disclosure statement: The author has no conflicts of interest to disclose.
How U.S. Presidents Got Thanksgiving Together

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Every November in the United States, people all over the country take a break from their busy routines to celebrate a holiday laced with tradition known as Thanksgiving. On the fourth Thursday of the month, family and friends from near and far will gather together for a grand feast with a menu classically composed of roasted turkey, mashed potatoes, gravy, stuffing, cranberry sauce, green bean casserole, and pumpkin pie. From a romanticized perspective, Thanksgiving is a time to cherish time with loved ones and to reflect on all of life’s blessings. However, in modern times, the concept of Thanksgiving has expanded beyond traditional values and has become very much commercialized. For example, months before the actual holiday, culinary retailers will begin advertising roasters, pumpkin-shaped soup bowls, turkey-ornamented platters, and other Thanksgiving-themed kitchenware. On the day after Thanksgiving, infamously known as Black Friday, die-hard consumers eager to get a head start on their Christmas shopping will lurk outside retailers' doors for hours, and when opening time finally arrives, will stampede in like cattle to take advantage of the limited time and highly reduced prices. And for those who are especially preoccupied with fitness, runs known as Turkey Trots will enable participants to burn some extra calories prior to the day’s feast. With all of these “traditions” dominating the modern American culture, it can be all too easy to forget the historical basis of how this holiday came to be.

In 1492, Christopher Columbus “sailed the ocean blue” and made history by being the first person to successfully sail across the Atlantic Ocean from Europe to the Americas [1]. Columbus’s journey instigated a race among European nations to establish colonies in the Americas with the hopes of finding gold and other riches that could generate wealth for the mother country. This race was dominated by Spain for most of the 1500s, but England was an eager contender. In 1607, the Jamestown colony in Virginia became the first permanent English settlement in North America. On December 4, 1609, a group of 38 English settlers arrived and settled a land grant along the James River that came to be known as the Berkeley Hundred (aka Berkeley Plantation) [2]. The journey across the Atlantic Ocean was long and perilous, so upon their arrival, these settlers observed a prayer of Thanksgiving for their safe passage to the New World [3]. The Berkeley Plantation, the grounds where the first Thanksgiving took place, is a site of historical significance in the United States. This plantation is where Benjamin Harrison, signer of the Declaration of Independence, was born and lived. His son William Henry Harrison (“Tippecanoe”) was elected the 9th President of the United States, and his great-grandson Benjamin Harrison (“Little Ben”) was elected the 23rd President.

This coincidental relationship between United States Presidents and the origins from which Thanksgiving sprang forth is only one of many. The holiday, unique to our country and celebrated
by few others, has indeed been contorted and perhaps tainted by commercialization, most notably in 1939 [4]. President Franklin D. Roosevelt, attempting to drag the country out of the depths of the Depression, moved the Thanksgiving holiday from the last Thursday of the month to the fourth Thursday, thereby extending the duration of Christmas shopping season and appeasing such capitalists as Frank Lazarus, Jr. who were lobbying the economic benefits. Many political spectators criticized this decision as an abandonment of the core values behind the meaning of Thanksgiving (a harbinger to the Black Friday and Cyber Monday sequelae of contemporary Thanksgiving). Supporters of Roosevelt, however, pointed toward their President’s resourcefulness in times of turmoil, using this uniquely American holiday to re-strengthen America. In fact, historical evidence shows that United States Presidents have long depended on the Thanksgiving holiday during times of national tumult in order to stabilize and unify the country.

Abraham Lincoln is credited with the establishment of Thanksgiving as annual national holiday, but credit is further due to Sarah Josepha Hale, an author and advocator for concordance between the North and the South [5]. She wrote the President and four others predating him, promoting Thanksgiving as an instrument by which to establish national solidarity. Her efforts were realized in 1863 when Lincoln signed legislation which established Thanksgiving as a national holiday (before that of Christmas or Easter). "Giving thanks" and appreciation was symbolically intended to promote the national identity of our young country at a time when we were fractured along geopolitical lines, and many preferred to look past the gruesome consequences of The Civil War. Take a moment to review the ISHLT’s dedication to Lincoln’s Gettysburg Address and what it means to us. ISHLT Links November 2011. Like Roosevelt, Abraham Lincoln valued Thanksgiving for its secondary purpose during a period of national strife, recognizing that it was both emblematic and singular to our country.

When the United States was in its infantile beginnings, George Washington proclaimed “a day of public thanksgiving and prayer” in 1789 [6]. The idea was to offer "an opportunity to all the citizens of the United States of joining, with one voice, in returning to Almighty God their sincere thanks”. This notion, surprisingly, was met with significant resistance from Congress, some members of which thought that a designated day for giving thanks was too forced, or that it overstepped boundaries between church and state. Reluctantly, it was signed into bill, the idea promoted as “a laudable one in and of itself”. After all, what ulterior motive can overshadow the good intentions behind demonstrating appreciation towards one’s life blessings?

Herein lies the true meaning of Thanksgiving. Sure it has been exploited, some purposes more noble than others, but many of our nation’s greatest presidents came to fully appreciate the holiday’s symbolism. By recognizing our country’s beginnings and being thankful, Thanksgiving allows us to unify, even under less-than-ideal circumstances. This concept has been called “nationalism” by some, but viewed under a less cynical lens, could more appropriately be called harmony. Thanksgiving continues to be taken advantage of in the commercial industry. Black Friday and all that it represents are not exactly flattering representations of the modern United States, but at least we can celebrate these “holidays” together.

Disclosure statement: The authors have no conflicts of interest to disclose.
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As the holidays draw ever nearer, perhaps people all over the world will brace themselves for a grueling season of feasting. On Thanksgiving Day in the United States, kitchens will be overflowing with the edible goodness of roasted turkey, stuffing, gravy, cranberry sauce, mashed potatoes, sweet potatoes, green bean casserole, and Grandma's famous pumpkin pie. After uncomfortably tight waistbands herald the end of the feast, the preparers of the meal will be faced with a new challenge: what to do with all the leftovers. Purists will argue that leftovers must be consumed in their original form whereas the more adventurous eaters may transform the ingredients into a new dish entirely. Regardless of how they are consumed, if too much time elapses, any uneaten vittles may become the subject of a biology project. Although the appearance of blue fuzz typically indicates that a food is destined for the trash can, it could also be the first sign of a medical breakthrough.

Like many great discoveries in science, the antibiotic penicillin was discovered serendipitously. In 1928, Alexander Fleming, a microbiologist on the faculty at St. Mary's hospital in London, neglected to clear away his Petri dishes of Staphylococcus before heading out for a weekend vacation [1]. When he returned to his lab after the weekend, he was greeted by both a messy lab bench and a peculiar finding. Inspection of his bacterial plates revealed that the plates had been contaminated with mold and that the bacteria growing closest to the mold were undergoing lysis. Thrilled by this finding, Fleming eagerly attempted to repeat the experiment, but it turned out this exact strain of Penicillium chrysogenum mold was more difficult to grow than he had anticipated. It was Fleming's colleague Ronald Hare who figured out that the mold grew and produced this “bacteriolytic substance” optimally at a low temperature of 20°C [1,2]. In subsequent experiments, Fleming took the “mould broth filtrate” from this special P. chrysogenum strain and added it to various bacterial strains that were pathogenic in humans to assess its antimicrobial activity. For the sake of convenience when publishing his results, Fleming substituted the lengthy phrase “mould broth filtrate” for “penicillin,” and hence gave this now ubiquitous antibiotic its name [2]. Fleming’s experiments yielded the findings that penicillin was ineffective at killing Gram negative bacilli, but was very effective at killing Gram positive cocci such as Staphylococcus aureus, Streptococcus pneumoniae, and Streptococcus pyogenes [2].

As significant as Fleming’s findings were, there existed the practical issue of generating enough penicillin from the mold to make it available for mass distribution. As World War II encroached on Europe, interest in finding treatments for infections and other illnesses that befell soldiers grew, so Dr. Howard Florey, a professor of pathology at Oxford University, and several of his colleagues accepted the challenge of making penicillin more widely available [3]. In the summer of 1941, shortly before the United States entered the war, Florey and his colleagues traveled to Peoria, Illinois to continue their research. They were particularly interested in finding a P. chrysogenum strain that
was capable of producing copious amounts of penicillin, so they solicited international assistance by asking for donations of lab samples, moldy fruit, grains, and vegetables [1]. After much searching, the solution finally arrived in the form of a moldy cantaloupe brought in by a Peoria housewife. With laboratory manipulation of this P. chrysogenum strain, penicillin production jumped from a humble 4 units/mL to 250 units/mL. Today, industrial P. chrysogenum strains can produce 50,000 units/mL of penicillin, and these strains are still believed to be derivatives of the strain isolated from the Peoria cantaloupe. In the war, penicillin significantly reduced mortality related to pneumococcal infection, and in 1945, Fleming, Florey, and Dr. Ernest Chain, a chemist on Florey’s Oxford team, were awarded the Nobel Prize in Medicine [3].

From a scientific standpoint, Penicillium chrysogenum could be considered a multitalented fungus because it has shown potential in synthesizing a variety of products. Penicillin, its most famous product, falls under the classification of beta-lactam antibiotics, a large group that also includes cephalosporins, carbapenems, and monobactams [4]. These drugs vary widely in their spectrum of coverage, but they share a common chemical structure of the beta-lactam ring as well as a common mechanism of inhibiting the synthesis of the bacterial peptidoglycan cell wall. Penicillin was once the treatment of choice for the common pathogens Streptococcus pneumoniae and Staphylococcus aureus, but due to growing resistance over the years, alternative antibiotics are now frequently used. Despite this growing resistance, penicillin is still the treatment of choice for conditions associated with Streptococcus pyogenes infection (e.g. Streptococcal pharyngitis, rheumatic fever, scarlet fever, toxic shock syndrome, necrotizing fasciitis, and erysipelas), meningococcal disease, syphilis, actinomycosis, gas gangrene, and Pasteurella multocida. In addition to its well-documented utility in the synthesis of an antibiotic, P. chrysogenum has shown potential in the production of the lipid-lowering drug pravastatin [5]. Breaking out from the field of medicine, P. chrysogenum may also have a future in the production of sweet wine [6].

At the conclusion of his Nobel lecture in 1945, Fleming summarized the beginning of his work with penicillin by describing P. chrysogenum as “a mould which was not wanted...[that] contaminated one of my culture plates...[and] produced an effect which demanded investigation” [7]. In Fleming’s case, seeing value in this unwanted organism led to the development of a drug that has significantly impacted medical practice. From this historical event, we can see that sometimes the things that are undesirable may prove to be more valuable than we anticipate. With this principle in mind, we should approach this season of feasting with a new mentality: Bring on the leftovers!

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EDITOR’S CORNER: From Little Ben and Uncle Jumbo to a Teddy Bear and a Big Lub

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Benjamin Harrison was born in North Bend, Ohio on August 20, 1833. Despite being from one of America’s oldest and most distinguished families, he grew up on a modest farm. He attended Miami University in Oxford, Ohio and studied law in Cincinnati. He was nicknamed, “Little Ben” during the Civil War because he stood only 5’6” tall. Little Ben was discharged as a breveted brigadier general following the Union victory in 1865. He became a brilliant lawyer in Indianapolis, Indiana and was elected to the United States Senate in 1880 where he championed the rights of Native Americans, Civil War Veterans and homesteaders and advocated for high tariff. Harrison unseated the incumbent President Grover Cleveland and became the 23rd President of the United States while winning the Electoral Vote but losing the popular vote. Cleveland held the umbrella for Harrison during the rainy inauguration on March 4, 1889 (see here). He earned another nickname, “Centennial President” because he was in office during the 100th-anniversary celebrations of George Washington’s inauguration. Harrison believed the future prosperity of the U.S. was linked to international trade and cooperation. He sponsored the first Pan-American Congress. In domestic affairs, he supported increased import duties, a new Pension Act, the Sherman Anti-Trust Act which regulated monopolies and the Silver Purchase Act thus authorizing the minting of additional silver coins. Other notable events included the addition of six new states joining the Union which was more than under any other President. Also, Congress authorized the first billion-dollar budget and tariffs in a single session which gave Harrison’s legislative branch the nickname “the Billion Dollar Congress.” He was the only President who was the grandson of a President, William Henry “Tippecanoe” Harrison. In addition, his great-grandfather, also named Benjamin Harrison, signed the Declaration of Independence in 1776. Little Ben died of pneumonia at his home in Indianapolis, Indiana on March 13, 1901. He left us this quote, “No other people have a government more worthy of their respect and love or a land so magnificent in extent, so pleasant to look upon, and so full of generous suggestion to enterprise and labor.”

As stated above, Grover Cleveland won the popular vote, but lost the Electoral College vote and therefore the election to Benjamin Harrison in 1888. He remained active in politics and criticized Harrison’s policies. He was against the Sherman Silver Purchase and McKinley Tariff Acts of 1890. Because of his political stances and stature as a former President he faced Harrison again in the 1892 election and won both the popular and Electoral College votes to become the only former President to regain the office when he was elected 24th President of the United States. Shortly after his inauguration in 1893, America was faced with the financial “Panic of 1893” with high unemployment and a severe economic depression. Cleveland blamed the Sherman Act and managed to have it repealed. During his second term as President from 1893 to 1897 his greatest success was his forceful efforts at putting an end to the strike by the Pullman Company railroad workers in Chicago. This strike had paralyzed commerce and mail delivery in the Midwest. He became renowned for his firm
William McKinley was born in Niles, Ohio on January 29, 1843. He attended Allegheny College in Pennsylvania, became a teacher then enlisted in the Union Army during the Civil War. He was promoted to Major and served under Rutherford B. Hayes during the War. After the War, he became a lawyer. With Hayes’ encouragement, he was elected to the House of Representatives at the age of 34 where he served 14 years and sponsored the protectionist McKinley Tariff Act of 1890. This act raised the average duty on imports to nearly 50% and was designed to protect domestic industries and foreign competition. McKinley earned the reputation as a champion of people’s rights against private interests and for protecting American businesses by creating tariffs on imported goods. Supported by a wealthy businessman, he was twice elected Governor of Ohio. He was nicknamed the “Idol of Ohio” and was chosen as the 1896 Republican Presidential candidate. From his “front porch” in Ohio, he spoke of a “full dinner pail.” His opponent, William Jennings Bryan from Nebraska was against McKinley’s high tariff and gold standard. Bryan appealed to farmers and the less fortunate and advocated “free silver” to increase the money supply. Because of the economic upturn, McKinley won the 1896 election and took the oath of office in 1897 as the 25\textsuperscript{th} President. The economic depression which started in 1893 had ended and the nation was on the path to prosperity. The period from 1897 and into the 20\textsuperscript{th} century was the time of prosperity and growth which became known as the “Gilded Age.”

Might I remind you of Mark Twain’s satirical point of view from all of his famous books including The Gilded Age.

Mckinley was the last Civil War veteran elected President and the first incumbent President to win re-election since Grant in 1872. A defining event of his reign was the Spanish-American War, ignited by the sinking of the U.S.S. Maine in Havana Harbor on February 15, 1898. The United States blamed Spain for the attack and sunk the Spanish fleet which led to a swift victory for America with territorial gains in Puerto Rico, Guam and the Philippines. McKinley set the stage for an “open door” trade policy with China and sponsored annexation of the Hawaiian Territory in 1898. Because of his popularity he was easily re-elected over Bryan in 1900. Just six months into his second term, he was shot by anarchist Leon Czolgosz while attending the Pan-American Exposition in Buffalo, New York. He died on September 14, 1901 and was replaced by Vice President Theodore Roosevelt. McKinley left us these words, “That’s all a man can hope for during his lifetime – to set an example – and when he is dead, to be an inspiration for history.”

Theodore Roosevelt (Teddy) was born to wealth and privilege in New York City on October 27, 1858. He was an ill, frail, near-sighted and asthmatic child who transformed himself into a robust and vigorous leader with indefatigable dedication to exercise and progress for himself and America. He was a historian, an author, an egotist, a braggart, an explorer, a hunter, a cowboy, a war hero, a naturalist, an aggressive and progressive reformer, an activist, a statesman, an expansionist, a Nobel Peace Prize winner, a founder of the National Collegiate Athletic Association (NCAA) and a
leading conservationist who established national parks, forests and monuments. His face rests between Jefferson and Lincoln alongside Washington on Mount Rushmore in the Black Hills of South Dakota. He was also one of the original members of the American Institute of Arts and Letters and a president of the American Historical Association. Most of all, Teddy Roosevelt embodied the strains of America’s transformation from a rural, agrarian, and relatively isolated society into a modern industrialized nation placing it among the great world powers; no different than how he transformed himself from that of a sickly child to nearly an indestructible man of masculinity and vitality.

He received a Harvard education, studied law and entered politics in 1882 as the youngest member of the New York State Assembly. He ran as a Republican, because he believed the Democrats supported Tammany Hall, the infamous and corrupt political machine of New York City. Personal tragedy struck early in his career. On Valentine’s Day of 1884, shortly after the birth of their daughter, Alice Lee, aka “Baby Lee,” his wife Alice Hathaway Lee Roosevelt died in the arms of the 25-year-old Theodore Roosevelt. To make matters worse, several hours before his young wife’s death, he had already said a final goodbye to his mother, Martha who died of Typhoid in the same house. In his diary that day, he left a big “X” and wrote, “The light has gone out of my life.” Afterward he spent two years in solitude on his ranch in the Dakota Territory. As an aside if you care to dare, you will not be disappointed if you read about his oldest daughter, Alice Roosevelt Longworth.

You will be quite amused about this socialite who graced the White House from her father’s administration into the Carter administration and was considered “The Other Washington Monument.” Among the many quips about her, President Carter wrote, “She had style, she had grace, and she had a sense of humor that kept generations of political newcomers to Washington wondering which was worse – to be skewered by her wit or to be ignored by her.” It was believed that Teddy Roosevelt could do anything except one. One day in the Oval Office following many interruptions by his daughter Alice, Teddy threatened to throw her out the window and stated, “I can either run the country or I can attend Alice, but I cannot possibly do both.”

Following his return from solitude, Teddy became police commissioner of New York City then Assistant Secretary of the Navy during the McKinley administration. When the Spanish-American War broke out in 1898 he organized his “Rough Riders” Calvary unit and led his troops into battle at San Juan Hill, Puerto Rico. He returned to New York and became governor and then Vice President of the United States. Following McKinley’s assassination he became the 26th President, the youngest President in American History. He won re-election in 1904. As President, Roosevelt launched a “trust-busting” campaign against big business. In foreign affairs, his greatest achievement was the Panama Canal which began under his leadership, and also he sent the Navy’s “Great White Fleet” on a world journey to demonstrate America’s military strength. He was awarded the Nobel Prize for Peace for helping end a war between Japan and Russia. During his terms, the Wright Brothers made their first flight and the Ford Model T was introduced. He created his own “Bull Moose” Party in an unsuccessful bid for a third term as President in 1912. It was during this campaign when he was shot by John Shank in Milwaukee, Wisconsin. TR’s metal eyeglass case and the manuscript for his speech protected his heart. Teddy delivered his speech with his blood stained manuscript before being rushed to the hospital. He died on January 6, 1919 at his Sagamore Hill home in Oyster Bay, New York either from a pulmonary embolus or a stroke. Nicknamed and affectionately known as T.R., he left us with, “I
have always been fond of the West African proverb: 'Speak softly and carry a big stick: you will go far.'"

William Howard Taft, whose father served as a judge and later as Attorney General and Secretary of War under Grant’s administration, was born in Cincinnati, OH on September 15, 1857. A graduate of Yale, he attended Law at the University of Cincinnati. After working as a lawyer and a judge, he was dean of the University of Cincinnati Law School. President McKinley named him Governor of the Philippines, and President Teddy Roosevelt appointed him Secretary of War. Ardently supported by TR, “Big Bill” Taft won the Presidential nomination in 1908. Roosevelt recognized he could influence the affable Taft but not mold him in his own image. In any event, political critics dubbed Taft (TAFT) as Take Advice From Teddy. Roosevelt was a dynamo, vigorous and visible, Taft was more restrained. Taft defeated William Jennings Bryan and was inaugurated the 27th President. Weighing in over 330 lbs, he was the heaviest President. He was derisively nicknamed “Big Lub” in reference to his weight during his childhood. This nickname stuck with him. During his presidency, TR’s trust-busting policies continued, the 16th amendment passed allowing Congress to collect federal income tax and the Boy Scouts of America was formed. In 1910, Taft started the tradition of throwing out the first ball of the baseball season. After his presidency, Taft became a professor of law at Yale until he was appointed Chief Justice of the United States in 1921. He has been the only President to serve on the Supreme Court and considered it his greatest honor. Taft later commented, "I don’t remember that I was ever President," a job that intimidated him. He died in Washington, D.C. on March 8, 1930 in coma as a consequence of cardiovascular disease. He was the first President buried in Arlington National Cemetery and he leaves us with “A government is for the benefit of all the people.”

Disclosure statement: The author has no conflicts of interest to disclose.

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