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HAPS EDUCATOR

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Greetings
From Your President

The Victoria Conference is now in the history books! And WOW what an impression it had on all who attended, including the numerous first-timers. The venue was spectacular and our Canadian hosts were the friendliest ever! Victoria was charming and offered great seafood, lovely parks, the harbor, the museum, Butchart Gardens, shopping, street entertainment, and plenty of sightseeing in our free time. The update speakers, banquet speaker, and workshops provided us with abundant “take-home” material useful in educating our students and sharing with our colleagues who couldn’t attend. The exhibitors and vendors continue to be a significant part of our society – both professionally and personally. We thank them for their continued and generous support. In a change from past conferences, this year the posters were set up in the exhibit hall, which allowed significantly more visitors to the posters and was appreciated by all. I only wish that more anatomy and physiology educators could attend our annual conference and experience the enriching activities and dialogues with our colleagues from throughout North America. Thanks to the conference committee for making this great event happen: Peggy Hunter, Thuy Nevado, Larry Anthony, Tony Webster, Lori Zehr, Kirstin Lane, Jennifer Giuliani, and Doug Panton.

The business side of the meeting brought about spirited discussion on the bylaw amendments, with both amendments passing. Thanks to all of the Presidents Emeriti for being so bashful about your opinions! Our face-to-face Board meetings in Victoria were extremely productive. We have accomplished much on our Strategic Plan and will continually update it as our goals are met. Because of the economic situation at many of our institutions, we will strive to have more regional conferences this year so that more of our colleagues may experience what HAPS has to offer. The Marketing Committee is starting to develop a plan to market HAPS on a broader range. At Camosun College, the Board participated in a leadership workshop; plans are to continue this process through selected readings and discussion. In addition, there was a workshop for HAPS attendees on leadership. Our desire is to make it easier to assume leadership positions in our society. It always saddens me not only to leave my colleagues at the end of the annual conference, but also to say farewell to members of the Board who are transitioning off. John Waters joins the Presidents Emeriti advisory board. Wanda Hargroder, Secretary, and Craig Clifford, Southern Regional Director, will also be leaving the Board as of July 1. I have nothing but the utmost respect and gratitude for their roles on the Board. I am certain that all of these Board members will find many avenues for their talents in continued service to HAPS.

On the other side of the coin, I welcome new members to the Board of Directors: Dee Silverthorn as President-Elect, Lucia Tranel as Secretary, and Jason LaPres as Southern Regional Director. I look forward to a fruitful working relationship with them as I move to Past-President.

As I transition from President to Past-President, I want to dedicate the remainder of this column to thank ALL of the Board and Steering Committee members who have helped make this past year a success. I truly couldn’t have done this job without them. Without the commitment of all of these volunteers, HAPS wouldn’t be possible. Some of these leaders are long-time “veterans” of HAPS and have served in many capacities over the years – so long, in fact, that some of the committee names have changed. Others on the Board and Steering Committee are relative “newbies” who hopped on board right away in their volunteer capacities. I’m amazed at the breadth and depth of their commitment. Collectively, the HAPS Board has 113 years of HAPS membership and the Steering Committee 296 years, with many of those years spent in leadership roles or as part of HAPS Committees. I salute you all!

(Continued on next page)
Board Members:

Past-President: **John Waters** joined HAPS in 1995 and has attended 15 annual conferences. He was the first HAPS webmaster. John has also been our president and treasurer, has served on the Animal Use Committee, and is co-chair of HAPS-I. He is a master at "coaxing" members to become involved in committees and the Board! He can be found at most conferences hangin’ with Bill Perrotti and Kevin Petti. John believes that if there is a heaven, it will look like a HAPS Annual Conference, minus the sad good-byes when it’s over!

President-Elect: **Don Kelly** joined HAPS in 2001 and hasn’t missed an annual conference since Philadelphia in 2003. Don has been chair of the Animal Use Committee, chair of the Nominating Committee, and will become HAPS President July 1. He teaches at Mohawk Valley Community College (MVCC) in Utica, NY, and helped the college transition to a cadaver-based human anatomy and physiology lab facility in 2000. MVCC is one of two colleges who have had two HAPS Presidents (the other being Stark State College, Ohio, where I teach)! Busy people only get busier as evidenced by Don’s service to several of his local community organizations such as the Library Board of Trustees and county planning board.

Secretary: **Wanda Hargroder** has been a HAPS member for 8 years and has attended seven annual conferences. She was a co-chair of the annual conference in New Orleans and has served on the Cadaver Use Committee (Chair), Membership Committee, and was a recent HAPS-I instructor. She lights up a room, and has thus been given the very appropriate nickname “The Sun” by John Waters.

Treasurer: **Elizabeth Becker** has been a member since 1989 and has served on the Membership Committee, Safety Committee, and was Central Region Director for two terms. She currently is serving her second term as Treasurer. She has been to 18 annual conferences and several regional conferences. Her passion is horseback riding, which has provided her with some interesting x-rays to use in class!

Western Regional Director: **Anne Geller** first experienced a HAPS annual conference in Portland, OR, and has missed only a few since then. She helped organize the last San Diego Annual Conference in 2007. She has rarely met a glass of wine she didn’t like or a dance floor that was too small. If there’s dancing anywhere within walking distance of a HAPS conference, Anne’s gorgeous red hair will be flying as fast as her feet!

Eastern Regional Director: **Amy Way** joined HAPS in 2002. Her first conference was in Phoenix and she has attended every conference but one since then. She has been on the Grants and Scholarship Committee (Chair) and is in her second term as Eastern Regional Director. She was a past recipient of a HAPS scholarship, which helped bring her to her first HAPS Conference!

Central Regional Director: **Pat Bowne**’s first HAPS conference was Toronto in 1997, and she has attended the annual conference every year but one since then. For years she has served on the HAPS-ED advisory committee, regularly writing summaries of update speakers for HAPS-ED. Pat has also served on the Public Relations Committee and has been key in discussions about HAPS’ presence on Facebook and Twitter. Pat concentrates best when knitting – and the Board is witness to what an accomplished knitter she is!

(Continued on next page)
Southern Regional Director: **Craig Clifford** joined HAPS in 1991 and his first conference was Greenville, SC. Since then, he has only missed one annual conference. Besides being our Southern Regional Director, Craig has served on the following committees: Animal Use, Cadaver, as in “Cadaver Use” and HAPS-ED. He has contributed regularly to HAPS-ED for years. His southern charm, easy-going demeanor, and wide smile are infectious.

**Steering Committee:**

**Tom Lehman**, chair, joined HAPS in 2000. He has attended every conference since Maui in 2001. He has hosted a regional conference, has been the Web Committee chair, and has served as chair of the Steering Committee since 2004. He is this year’s recipient of the President’s Medal. Tom loves to tie-dye and brings tie-dyed shirts to every annual conference committee coordinator!

**Nick Despo**, Animal Use, joined HAPS in 1998 and his first annual conference was Fort Worth, TX, that same year. Although he missed a few conferences, he has attended every conference since Austin (2006). He has served on the Animal Use Committee since 2007 and has been chair since 2010.

**Izak Paul**, Annual Conference, became a HAPS member in 1992 and has attended over a dozen HAPS annual conferences. He has served as a Regional Director (Western) and hosted a wonderful HAPS annual conference in Calgary in 2004 (where a highlight for many of the women was picture-taking with the Royal Canadian Mounties!). He has served as chair of the Annual Conference Committee since 2005 and has taken it to new levels. As of July 1, he will step down as chair and get some much deserved rest!

**Leslie Day**, Cadaver Use, joined HAPS in 2003 and attended her first conference in St. Louis in 2005. She is a more recent committee member and has served as chair of the Cadaver Use Committee for two years. She has taught for 14 years and was awarded her university’s Excellence in Teaching award in 2009!

**Ron Gerrits**, Curriculum and Instruction, became a HAPS member in 2002 during the Phoenix Annual Conference. It was suggested by a colleague that he join HAPS because Ron had started a new full-time teaching position. He became “hooked” in Phoenix and joined the Curriculum and Instruction Committee at that meeting! He has stated that people were so nice to him as a first-timer that he wishes he could wear a first-timer’s badge at every conference!! He has missed only two conferences since Phoenix.

**Valerie O’Loughlin**, Foundation Oversight, was lured into HAPS by the Maui conference in 2001! She has attended every conference since. She has served on the Cadaver Use Committee, Partner Associations Committee, Membership Committee, and the search committee for our management group. She is also a liaison to AAA. Valerie’s background in biological anthropology is shared with HAPS ters through aging and sexing skeleton workshops at HAPS conferences (with Elizabeth Pennefather-O’Brien). She is also a textbook co-author who apparently doesn’t require sleep!

**Mike Kopenits**, Grants and Scholarship, has been a HAPS member for six years and his first conference was in St. Louis in 2005. He hasn’t missed a conference since. He has been the chair of the Grants and Scholarship Committee for three years. From his remarks at the Victoria Business Meeting, he noted that his last name presents some interesting spelling challenges for students who may inadvertently remove the “i” or add an extra “i”!

**Marsha Sousa**, HAPS-ED, joined HAPS in 1991 and has attended over 10 conferences. She has been the Editor of HAPS-ED since 2008 and has helped usher it into the digital form. Marsha found HAPS a lifeline for her when she was an adjunct instructor and the sole instructor of anatomy and physiology at her University.

**Ellen Arnestad**, HAPS-Institute, joined HAPS during the Calgary Annual Conference. She has been involved in HAPS-I, beginning with its feasibility study. Since then she has been part of HAPS-I as a committee member, director, co-chair, and instructor/course designer. Ellen has been to every HAPS Annual Conference since Calgary and rode her motorcycle to this year’s conference in Victoria!

**Javni Mody**, Marketing, has attended 13 HAPS conferences and has been a HAPS member since 1995. She has served in many capacities, including a member of the Regional Conference Committee and Marketing Manager, where she has played a vital role as a liaison with our vendors. She updated the Regional Conference Guide and the policies and procedures manual for regional conferences. Last year she received the President’s Medal for her contributions to HAPS.

**Gary Johnson**, Marketing, was one of the original founding members of HAPS and is a President-Emeritus – and he is STILL involved! He has missed only one meeting in 25 years – the very first meeting at Triton College (IL) in 1987. Even though he has retired from teaching, he still attends HAPS conferences. You can identify Gary in a crowd of HAPsters by his Hawaiian shirts or his HAPS t-shirts. Like many HAPS members, Gary entered teaching anatomy and physiology in a round-about way. As far as I know, he is one of only two HAPS members who began their careers working in a zoo (the other being Kevin Patton)! Some might comment that is why Gary gets along so well with HAPS colleagues!

(Continued on next page)
Elizabeth Hodgson, Membership, became a “HAPSter” at the Philadelphia annual conference in 2003 and since then has missed just one annual conference. Elizabeth has been on the Web Committee and is currently co-chair of the Membership Committee, serving as chair of the committee for a year. Elizabeth was a bit bleary-eyed in Victoria as she had just arrived from a cruise prior to the Victoria conference.

Elizabeth Pennefather-O’Brien, Membership, is one of our relative “newbies” on the Steering Committee as she has been a HAPS member since 2005; she has been to six conferences since joining. In her short period of time as a HAPS member, Elizabeth has served on the Safety Committee and is currently the co-chair of Membership. I would venture to say that Elizabeth probably has one of the most misspelled names in HAPS (folks want to put an extra “e” in Pennefather)!

Margaret Weck, Partners Association, has been involved in HAPS since it became an official organization in 1989 in Reno, NV. Margaret has missed only two HAPS annual conferences since its inception – one because she was on a wildlife tour in Australia and the other because she was on her honeymoon! Now Margaret’s husband Jamie can be found accompanying Margaret to our annual conferences. Margaret’s service to HAPS is without precedence. She served as secretary/treasurer when it was one combined position and was HAPS President (2007-2008). Margaret has been chair of the following committees: Grants and Scholarship Committee, Partner Associations, and Presidents Emeriti Advisory Board. In addition she has served (or currently serves) on the Core Curriculum Committee, Testing Committee, Annual Conference Committee, and Steering Committee. She regularly gives workshops at annual conferences, attends regional meetings, and was conference coordinator the second time the HAPS annual conference was in St. Louis (2005).

Kevin Petti, President Emeriti Advisory Board, joined HAPS in 1994. Kevin rarely misses a HAPS conference, although he missed the Victoria conference this year because his daughter graduated high school. Kevin has been a HAPS President, annual conference coordinator for the 2007 San Diego conference, presented HAPS workshops, and taught a HAPS-I course. Some years ago, Kevin was a one-man marketer of HAPS – developing a strategy to market HAPS through current members and creating a unique package to send to new HAPS members, including a vertebra mug. Kevin has garnered national and international attention and given many lectures on the links of historically relevant anatomical art to teaching. He has travelled Italy researching centuries-old anatomical art and its use in medical education.

David Evans, Public Affairs, joined HAPS in 1992. He has been to 17 annual conferences as well as to multiple regional conferences. David has been actively involved in the HAPS-ED Advisory Board, Publications Committee, Web Committee, Nominating Committee, and Steering Committee. He has served as the HAPS Public Affairs Officer and chair of the Public Affairs Committee. He is the editor for the extremely popular “What’s New” column that is regularly sent to us. David contributes regularly to the listserv as David in Darkest PA! His students claim he is “old and mean” but we know otherwise!

Ewa Gorski, Regional Conference, has been a HAPS member for 10 years and has attended every annual conference except Calgary since she joined HAPS. Ewa has served as the chair of the Regional Conference Committee for the past six years. If Elizabeth Pennefather-O’Brien and Mike Kopenits have the most misspelled name in HAPS, Ewa probably has the most mispronounced name!

Karen McMahon, Safety, joined HAPS in 1997 at the Toronto meeting. She hasn’t missed a conference since. Along with Sandy Lewis, Karen helped originate the Safety Committee, and she was instrumental in publishing the HAPS Safety Guidelines. Karen’s next big project is hosting the 2012 Annual Conference in Tulsa, OK. From the presentation she gave in Victoria, we are extremely excited to be having a conference in Tulsa!

Linda Nichols, Safety, began attending HAPS conferences in Portsmouth, NH, in 1994, and has been to most conferences since then. Linda recently retired from 35 years as a teacher and/or administrator. But that hasn’t stopped her from volunteering her time for HAPS! Since retirement, she has also been involved in three other jobs, all dealing with education. These include supporting high school Health Academies, offering camps promoting science to high school freshmen, and working with a private company on medical and nursing education using a new virtual platform.

Eric Sun, Testing, had his first HAPS annual conference experience in Phoenix in 2002. He hasn’t missed a conference since. As soon as he joined HAPS, Eric became involved in the Testing Committee as both chair and then co-chair. He helped organize a regional conference in Macon, GA, in 2009, and has conducted four webinars entitled “HAPS Objectives and Guidelines”. Eric loves animals and has a menagerie consisting of a horse, donkey, parrot, parakeet, cat, dog, and fish. He also is an avid gardener and loves cooking Chinese food.

(Continued on next page)
Curtis DeFriez, Testing, has been to six HAPS annual conferences. He has been on the Testing Committee for five years – first as a member, now as a co-chair. He's taught both anatomy and physiology and pharmacology at Weber State University (Ogden, UT) for 10 years. He recently finished a three-year plastination project of a cadaver; these organs and specimens are now used regularly in his anatomy and physiology courses.

Tom Lancraft, Web, has attended 16 conferences, with the first being Beaumont, TX, in 1993. Since joining HAPS, Tom has served as chair of the Web/Technology Committee, wrote the HAPS Position Paper on Distance Learning, has been the Web Editor for two years, and is the Instructional Design Coordinator for HAPS-I. He's one of our go-to guys for anything technology! Although Tom lives in Florida, he won't be found on the beach; his idea of a good time is to take short walks in the mall where there is air conditioning!

Peggy Hunter, 2011 Annual Conference Coordinator, has been a HAPS member for six years. This was Peggy's first foray into HAPS committee work – and boy did she pick a doozy! The smoothness of the Victoria conference attests to her organizational and people skills. We will give her some time to decompress from the conference and then get her involved in other committees!!

There you have it! Thirty-one of the most talented, generous individuals I have met. In addition to the Board and Steering Committee folks, Larry Spraggs (Executive Director), Shanan Molnar and Robin Hurst (from HAPS Headquarters) help to implement Board actions and manage our day-to-day activities.

Thanks to ALL of you for accompanying me on our HAPS journey this year!

Caryl Tickner
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Oh Canada! You know you are in a special place when everyone thanks the bus driver as they exit the bus. And what an amazing conference! Victoria was only my second conference with HAPS, but after Denver and now Victoria how can it keep getting better? I’m finding that HAPS Conferences are something unique. The learning opportunities, the networking opportunities, and the chance to enjoy the company of friends are outstanding. Peggy Hunter and her committee, as well as the HAPS Headquarter’s staff, deserve so much credit for their organization of this event. From my own experiences and those of everyone I talked to, everything went perfectly and smoothly. I think a new standard has been set for “herding cats.”

The annual conference is also the opportunity to conduct much of the business of HAPS. The Board of Directors meets for one of their two face-to-face meetings, the committees all meet, and the Annual Business Meeting is conducted. I have worked with numerous organizations over my career and I have to say that the active participation of the membership in the business of HAPS is commendable. You should all feel proud that the Board of Directors takes their job in governance very seriously and the membership at large cares deeply about the well being of HAPS as shown by the spirited, yet respectful, discussion of business items at the Annual Business Meeting. I think HAPS has a bright future that will be sustained by an active and caring membership. Stay involved with HAPS all year long, not just at the Conference, as this is the sign of a truly great organization.

It has been a pleasure working with President Caryl Tickner and the Board of Directors over the last year. A lot has been accomplished and the stage has been set for more advances in HAPS. I look forward to working with our new President, Don Kelly, and the new board this year. Don has already set an ambitious agenda and I’m excited about our future.

Remember…Learn, Discover, Share with HAPS.

Cheers,

Larry

Dr. Laurence Spraggs
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Summary of Update Seminar I
Brain Insulin: A Sweet Deal for Normal Baroreflex Function

Presenter: Virginia Brooks, Ph.D.
Department of Physiology and Pharmacology
Oregon Health and Science University
Portland, OR

Summarizer: Pat Bowne, Ph.D.
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All Anatomy and Physiology courses deal with the normal baroreflex control of heart rate, in which carotid sinus and aortic arch baroreceptors send impulses to the brain through the glossopharyngeal and vagus nerves, respectively. The cardioregulatory and vasomotor centers in the medulla oblongata then send either sympathetic (SNS) impulses (via sympathetic efferents) or parasympathetic (PNS) impulses (via the efferent fibers of the vagus) to the heart, adjusting heart rate to return blood pressure (BP) to within normal range. All A&P students learn that when BP decreases, the SNS will be activated and increase heart rate. This update seminar took us farther into the details of the response and its alterations in a variety of pathological situations also associated with insulin resistance.

First, SNS activity is not ‘turned off’ at normal BP levels, as our students are often prone to believe. It is tonic; that is, it is active at a low level during normal BP levels and in fact, like heart rate and BP, it varies during the respiratory cycle. For example, when BP drops significantly during inspiration, activation of the SNS obscures this variation.

Second, the relationship between HR and BP, or SNS activity and BP, is sinusoidal (Fig. 1). That is, there is a maximum HR/SNS which cannot be exceeded, no matter how high BP drops. There is a minimum HR/SNS at which further decrease stops, no matter how high BP rises. Between these two extremes is the effective range of the baroreflex, a range in which a change of BP will be met with a change in HR/SNS activation. The slope of the line relating BP to HR/SNS in this physiological range is the baroreflex gain and represents the effectiveness of the reflex. If the baroreflex gain is high, this means that a small change in BP is met with a large compensatory change in HR/SNS.

The baroreflex is impaired in both normal physiological conditions (pregnancy, aging) and pathological conditions (obesity, type II diabetes mellitus, metabolic syndrome, Alzheimer’s disease, hypertension, and congestive heart failure). Yet it is not immediately obvious that these conditions share anything which would explain the baroreflex impairment. Dr. Brooks gave credit for the insight that all of these conditions involved insulin resistance to her former graduate student Daisy Daubert, a HAPS attendee. The subsequent research project has been carried on by both Dr. Daubert and a large number of graduate and undergraduate students, and was noted as a testimonial to the effectiveness of undergraduate research internship programs.

The investigation of this hypothesis began by comparing changes in the baroreflex and in insulin sensitivity in pregnant rabbits. Pregnant rabbits show a noticeable change in the baroreflex response, with decreases in both maximum HR and baroreflex gain at the end of gestation (Fig. 2). Insulin sensitivity also decreased during the latter half of pregnancy and was highly correlated with baroreflex gain (r²=0.65).

(Continued on next page)
Treatment with rosiglitazone returned both insulin resistance and baroreceptor gain to pre-pregnancy levels, while having no effect on control animals.

Having established a relationship between insulin sensitivity and baroreflex function in pregnant animals, the research turned toward obesity. An animal model of human obesity was constructed by feeding rats a high-fat diet. Some rats, the obesity-prone, became overweight and developed insulin resistance. These rats also exhibited decreased baroreflex gain as measured by HR responsiveness, and again both conditions were improved by rosiglitazone. Given that insulin sensitivity appears to affect baroreflex effectiveness, at what point in the baroreflex does it exert this effect? The team began to test each component of the reflex, beginning with the afferent pathways from the baroreceptors to the brain.

The glossopharyngeal nerve showed similar activity in control, pregnant, and obese animals. When the glossopharyngeal nerve was cut and directly stimulated, to remove any variations in baroreflex neuron firing, obese and pregnant animals still showed a blunted change in HR, suggesting that the problem arose somewhere in the brain or in the efferent pathways from the brain to the heart. Indeed, neurons in the vasomotor centers showed decreased activation in the pregnant and obese animals, causing the team to refocus on the brain.

Insulin crosses the blood-brain barrier via active transport by an as yet unidentified molecule which is suspected to resemble the insulin receptor. If this is the case, it would make sense that conditions which decrease receptor-based insulin sensitivity in other tissues would also interfere with the transport of insulin into the cerebrospinal fluid. Once insulin has entered the brain, it can attach to receptors in areas involved with the baroreceptor reflex. As a result, increased brain insulin improves baroreflex gain. Importantly, the ability of increased insulin (during feeding) to improve baroreflex gain has recently been demonstrated in humans by Young et al. (2010). However, in insulin resistant conditions, like pregnancy or obesity, the fall in brain insulin would depress baroreflex gain.

In support of this idea, during pregnancy, the insulin content of rabbit CSF was found to decrease by approximately 50%, at exactly the time during pregnancy when both insulin resistance and baroreflex insufficiency appeared. The decrease in baroreflex gain could be abolished by injecting insulin into the CSF, but the decrease in maximum HR remained, suggesting that it was mediated by a different mechanism (probably allopregnanolone produced during pregnancy).

Infusing insulin into the brains of obese animals also increased baroreflex gain, heart rate, and SNS activity. In an unexpected finding, however, insulin infusion into the CSF of obese rats increased maximum HR and baroreflex gain above the normal, suggesting that these rats' brains had become hypersensitive to insulin.

Where in the brain is insulin exerting these effects? Insulin receptors are found in several areas involved in the baroreflex – most notably the rostroventrolateral medulla, the paraventricular nucleus in the hypothalamus, and the arcuate nucleus. While blocking the activity of the paraventricular nucleus reversed the effects of insulin, injecting insulin directly into that area or into the medulla had no effect. The arcuate nucleus, which also responds to leptin, appears to be the target area through which insulin exerts its effect on the baroreceptor reflex. One neurotransmitter that could relay the signal from the arcuate nucleus to the paraventricular nucleus could be inhibition of neuropeptide-Y-releasing neurons in the arcuate nucleus, as experimental neuropeptide Y blockade in this region restores baroreflex gain and maximum heart rate to normal levels.

HAPSters greatly enjoyed this lively presentation of a fast-moving research program, and had a variety of questions ranging from the roles of diabetic PNS neuropathy, possible cardiac insensitivity to SNS stimulation, and orexin in the development of baroreflex insensitivity. We all especially applauded the excellent opportunities Dr. Brooks has provided for so many undergraduate interns.

Note: Dr. Brooks has graciously allowed HAPS to post a copy of her presentation on the HAPS website, www.hapsweb.org. Click on the annual conference link and follow through to Speaker Presentations.

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BIRD FLU! Still a Serious Threat Worldwide
Education remains our best weapon

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With the 2009 H1N1 (Swine Flu) pandemic now declared officially ended by the World Health Organization, the world’s pandemic influenza threat is over, right? Wrong. H5N1 (Bird Flu) which originated in 1997, disappeared, and then re-emerged in 2003, continues to work its way around the world and remains a serious threat to humanity. Education, and the global sharing of information remains, in my opinion, our greatest defense against potential pandemic viruses, and in particular, the influenza virus. As educators, we can make a huge difference toward this effort.

Before we begin addressing the issue at hand, it is important to clarify terminology used in this article’s presentation of influenza and its continuing threat to the world. The following terms will be used when referring to the various influenzas discussed here: H5N1 virus (bird or avian flu) which emerged in 1997, 2009 H1N1 virus (swine flu), 1918 pandemic influenza virus, and the seasonal flu virus (which changes each year and is usually a combination of various types and subtypes of influenza viruses).

It is also important to list a few quick facts regarding the H5N1 Bird Flu before proceeding:

1. The H5N1 Avian Flu virus is NOT currently a pandemic virus, but has the POTENTIAL to become a pandemic virus.
2. The current H5N1 virus that we will discuss is very similar to the 1918-19 pandemic influenza virus which killed 50-100 million people worldwide (Johnson et al. 2002).
3. Over 538 people have become infected with the current H5N1 bird flu virus since it first appeared in 1997 (WHO, Global Alert and Response 2011).
4. The current mortality rate for H5N1 infections among humans is 60%, making the current H5N1 bird flu virus one of the deadliest viruses on our planet (WHO, Global Health and Response 2011). Mortality rate for humans who became infected with the 1918-19 pandemic virus was about 2.5% (Crosby 1989).
5. So far, human to human transmission has been very limited. Nearly all human infections of H5N1 bird flu have come from handling infected birds or eating undercooked infected poultry (WHO Influenza Fact Sheet 2011).
6. Vaccine research and development is underway, but is difficult. In addition, there is uncertainty regarding how effective a vaccine would be if the current H5N1 bird flu virus mutated to become a pandemic virus. Also uncertain is our ability to produce enough vaccine in a timely manner to meet world-wide need if such a demand occurred.

We now know that the great influenza pandemic of 1918-1919 was caused by an H1N1 virus similar to the bird-flu virus which originated in 1997 in Southeast Asia and has now spread around the world. This information was made possible due to the patience and diligent research efforts of Dr. Jeffery Taubenberger, a pathologist for the United States Armed Forces Institute of Pathology, and his team. The team began the slow process of sequencing small viral RNA fragments, obtained from archived influenza autopsy materials which were collected from deceased soldiers in the fall of 1918. The goal was to identify the entire genomic structure of the 1918 influenza virus (Taubenberger et al. 1997). Using tissue samples from three deceased soldiers, it took over a year to finally complete the project. One may well ask “where did we ever find tissue samples from 1918?” As it happens, the US Government has been obtaining tissue samples from armed forces personnel for about the past 150 years and cataloging and storing them in an obscure warehouse (NOVA 2005).

Influenza is an infection of the respiratory tract caused by a virus and transmitted primarily through contact with infected secretions (touching contaminated items, shaking hands, putting hands in mouth, direct sneezing, etc.). It has an incubation period of about 1-5 days from exposure to onset of symptoms, with the majority of influenza types most communicable around 1-2 days BEFORE the onset of symptoms and up to 5 days after symptoms. This makes pandemic influenza viruses a serious concern, since we could be exposed to influenza from seemingly healthy people who have no symptoms at all, just as we may be exposed to a seasonal flu virus. However, data for H5N1 infections indicate that an incubation period of 2 to 8 days is common and an incubation period of up to 17 days is possible with this virus (WHO Influenza Fact Sheet 2011). Thus, the potential for rapid spread of this virus is most concerning.

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Influenza viruses are named according to a very precise protocol. In order to understand the naming protocol, let us use a simple virus as an example: A/Beijing/32/91 (H3N1). “A” denotes the virus type (we will compare type A and type B viruses later in this article). “Beijing” denotes the geographical origin of the virus. Most influenza viruses contain an Eastern Asian name because of a number of factors. First, in many areas of Eastern Asia, humans live in very close proximity to other humans and animals, especially domestic birds such as chickens, and there are very large populations of domestic birds as well as people in this area of the world. Next, the prevailing winds affect the migratory patterns of wild birds, such as ducks and geese. Since wild birds are the primary carriers of influenza viruses, their migratory patterns usually bring the virus from eastern Asia toward the west. In our example virus, “32” denotes the strain number, and “91” the year the virus was isolated. In parentheses “H” represents hemagglutinin. Hemaglutinin is the protein that allows the influenza virus to enter the host epithelium. It determines the virus subtype. “N” represents neuraminidase, the enzyme that allows the replicated viruses to exit the host epithelium and infect other healthy cells.

There are three major types of influenza viruses: types A, B, and C. Types A and B are most relevant to this discussion. Therefore, we will only compare these two types of viruses. Type A viruses can affect both animals and humans. This is the type of virus capable of causing epidemics and pandemics. Major pandemics are more likely to cause higher mortality rates and serious complications among younger, healthier adults ages 18-40 and pregnant women (Figure 2, W curve, Simonsen 1998). Type B viruses affect humans only and generally cause only milder epidemics. Persons at greatest risk of complications resulting from type B viruses include the elderly, infants, immunocompromised individuals, and pregnant women. (Figure 2, U curve, Simonsen 1998)

The problem with type A influenza is that hemagglutinin and neuraminidase periodically change. A minor change, referred to as antigenic drift, causes a mutation in the genes that code for the flu virus’s surface antigens, enabling a more common seasonal virus (type A and/or B) to escape or bypass the body’s defense system. This is why we need to produce new flu vaccines each year for the anticipated seasonal flu virus. Seasonal flu vaccines contain vaccines against both A and B viruses. Antigenic shift, on the other hand, is a major change that occurs in type A influenza viruses, which are genetically labile, and can easily reassort, swap, scramble, or exchange genetic material between virus subtypes from different species. Poultry and pigs are good vessels to mix the genetic material from human and bird viruses. As a result, new subtypes emerge that may have the potential to cause a pandemic. High Pathogenicity Avian Influenza (HPAI) outbreaks have, to date, been caused by antigenic shift resulting in subtypes H5 and H7.

What are the basic differences between pandemic and seasonal flu? Pandemic flu is caused by a type A novel virus and generally begins in wild birds, such as geese and ducks, or in pigs. It then spreads to domestic birds, such as chickens, and then finally jumps species to become easily transmitted from human to human. We have little or no immunity to such viruses and there are few antiviral medications available. Vaccine production for these viruses is much more challenging than vaccine production for seasonal flu viruses. Seasonal flu is caused by type A and/or B viruses, generates milder symptoms in humans, and spreads only among humans. It responds more effectively to antiviral medications, and vaccine production is easier than pandemic virus vaccine production resulting in greater vaccine availability.

In general, all pandemics appear to follow similar patterns of development and spread. The World Health Organization has established 6 phases of a pandemic. The interpandemic period includes two phases. In phase 1, no new flu virus subtypes are detected in humans; there may be some in animals, but risk to humans is very low. In phase 2, no new viruses are seen in humans but circulating animal subtypes pose substantial risk to humans. Next is the pandemic alert period which includes phases 3, 4, and 5. In phase 3, human infections with new subtypes have occurred, but there has been only rare or no human to human spread of the virus. Right now, H5N1 remains classified in phase 3. In phase 4, only small clusters of human to human transmission occur. During phase 5, large clusters of human to human transmission are seen, but human to human transmission is still localized. However, the virus is clearly better adapted to humans. The pandemic period includes phase 6, the pandemic phase, when there is increasing and sustained transmission in the general population throughout the world. The 2009 H1N1 flu was declared to be in the pandemic phase on June 11, 2009. While the 2009 H1N1 virus was highly contagious, the virulence of the virus was, fortunately, far less than originally anticipated, and mortality was minimal.

The pandemic phase may occur in peak waves of transmission which can be weeks apart. This was clearly seen in the 1918-19 pandemic (Figure 1, Jordan, 1927). Since it is possible that a pandemic caused by H5N1 could follow this pattern, it is important for people to know this in order to understand why official orders to close public facilities and ban public gatherings may continue even though the threat of human to human spread appears to have ended.
The extremely high mortality rate of the H5N1 virus in humans is alarming. It is currently approximately 60%, as reported to the World Health Organization since 2003 (WHO Global Alert and Response 2011). In contrast, consider that 50-100 million people died during the 1918-19 pandemic, with a mortality rate of only 2.5%. It is no wonder there is so much concern within the World Health Organization, the US Centers for Disease Control, and most governments around the world regarding the H5N1 bird flu virus and its potential of mutating to become a pandemic virus. The virus, in its current highly lethal genetic form, is circling the globe, carried by migrating birds such as ducks and geese. Worldwide, millions and millions of chickens have been culled, particularly in Southeast Asia, causing serious economic concern. H5N1 infections in domestic poultry result in very high death rates (up to 100% within 48 hours), while most wild birds carrying the virus show little to no evidence of the illness (WHO Avian Influenza Fact Sheet 2011).

Also of huge concern, and a good topic for discussion during immune system lectures in A&P classes, is the fact that people, aged 18-40 are at greatest risk for major complications and death from a pandemic virus, compared to their lower risk for complications from a seasonal influenza virus. Explain to students that strong immune systems elicit a very strong immune response, especially where there is very little to no immunity whatsoever (as is the case when a person becomes infected with a "novel" influenza virus). Since our immune systems generally peak in function between about ages 18-40, strong immune responses can cause a "cytokine storm" where the immune response is so vigorous that it actually destroys the body's healthy tissue as it destroys the pathogen. I like to use the example that we often times develop a fever when we become ill from an invading pathogen. In most cases, the elevated body temperature actually helps destroy the pathogen. However, if the fever gets too high, the fever itself can kill us. Unfortunately, many of our healthiest and most productive members of society are most at risk of death from a pandemic virus, as evidenced during the 1918-19 pandemic (Simonson et al. 1998). Are leaders of our armed forces concerned? Of course. Our military consists primarily of people in the most vulnerable age group. Unfortunately, not all federal policy makers in this country are willing to take "concern" to "action" and appropriately fund research and education related to the H5N1 virus and its pandemic potential. Unlike other disasters, where infrastructure is seriously damaged or destroyed (consider Hurricane Katrina, for example), a pandemic doesn’t have to affect infrastructure if we are prepared, educated, and well-funded. For example, if a certain number of doctors, truck drivers, airline mechanics and pilots, electrical linepersons, computer and technology experts, communication experts, educators, etc. become ill, we must have a plan in place for temporarily replacing them. We also need a network of individuals who can care for children and dependents of our health-care workers, first responders, etc.

However, not all factors regarding a potential H5N1 bird flu pandemic are gloom and doom. Certainly we have already learned much from the recent H1N1 “swine flu” outbreak. Public awareness and understanding (albeit at a very elementary level) have improved, although the potential for complacent attitudes if there is a major change in H5N1 bird flu virus could be problematic. But research continues in vaccine and antiviral drug development, funding is improving, global reporting protocols have been established, and even digital connections are being developed to assist in containment of the virus at the source. The College of Natural Science and Mathematics at the University of Alaska Fairbanks has reported on work being done there by the Center of Excellence for Influenza Research and Surveillance (CEIRS). Important aspects of that work include the digital component which will allow for high-precision, in-time information during an influenza outbreak. Such a sophisticated digital component to avian influenza monitoring, research, and reporting has vast global implications and is most encouraging (Huettmann 2008).

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It is also encouraging that pandemic education and preparations are becoming more organized in many countries, including ongoing efforts to educate people about proper respiratory hygiene and the dangers of handling or consuming under-cooked infected birds. In the United States, we have made some progress in educating first responders, health care workers, certain government and industry leaders, and some educators regarding pandemic preparations, but we still fall far short of where we should be. With appropriate mass education, we can prepare for a potential pandemic, whether it is the H5N1 avian influenza or some other pandemic pathogen yet to emerge.

My final thoughts and questions regarding this challenge we may face at some time in the future include the following:

1. We have never in history been more physically connected to each other globally than we are today.
2. Our health care system cannot support our current health care needs. What would happen in the case of a pandemic?
3. Many more people today have asthma, allergies, lung and other cancers, and serious chronic conditions such as obesity, diabetes, and heart disease than in 1918. How will a pandemic affect these people?
4. Finally, consider the fact that there are many more people here on earth today than there were in 1918!

How do we stay as safe as possible during a pandemic? Follow official recommendations for vaccinations, hygiene, masks, etc., and isolate yourself and your family as much as possible. Remember: influenza pandemics are always temporary. The virus ultimately destroys itself by killing the organism it invades, or the organism gains the capability to kill the virus and remain alive.

How do we best prepare for and survive a pandemic? Education! It’s our best weapon. I strongly suggest you check out pbs.org/wgbh/rxfor survival, an excellent DVD series on infectious diseases sponsored in part by the Bill and Melinda Gates Foundation. Look for Bird Flu: How Safe Are We? There are other resources online regarding this issue with compelling stories and information your students would love to learn about. We have already been given a gift of knowledge regarding influenza pandemics. People in 1918 could not own that gift. It is our responsibility to use it.

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Like a metronome, the beating heart paces the background music of our lives, speeding up or slowing down to accompany life’s constantly changing rhythms. The right AV valve, the pulmonary valve, the left AV valve and the aortic valve help maintain a one-way flow of blood through the heart and ensure that the heartbeat is regular and dependable. When disease affects any of these valves, the ability of the heart to function as a pump is compromised. Heart valve disease characteristically takes one of two forms. It can lead to the development of leaky valves, causing regurgitation, or it can result in valves that do not open widely enough, a condition known as stenosis. Either condition can cause troublesome symptoms that may lead to a reduction in a person’s overall quality of life or, in extreme cases, affect a person’s ability to survive.

Currently, valve repair is still considered to be the gold standard when it comes to surgical treatment of heart valve disease because of its ability to preserve natural heart tissue, decrease the risk of post-surgical infection, and decrease the need for anticoagulation medication. Valve repair is limited, however, by the tendency of conditions such as valve disease related to rheumatic fever to progress and become more symptomatic as the body ages (Sarralde et al. 2010, Omeroglu et al. 2004). Common valve repair procedures include commissurotomy, decalcification, resection of valve leaflets, and mitral valve repair. A commissurotomy is done to separate fused valve leaflets and widen the valve opening thereby delaying or negating the need for valve replacement (Omeroglu et al. 2004). Decalcification is a process of removing age-related calcium deposits from the valve leaflets to allow more normal flexibility and mobility of the leaflet. Resection, reconstruction, or repositioning of a valve leaflet are all possible procedures and can be done to correct a variety of valve pathologies. Illustrative of the type of valve repair being done today, Urbanski reported in 2010 that he obtained the best long-term results for aortic valve reconstruction of calcified leaflets by sewing autologous pericardial patches, taken from the patient’s own pericardium, into the frame of the native valve leaflet once the diseased area of the leaflet had been resected. In an array of other procedures, holes in valve leaflets can be surgically patched, leaflets can be enlarged by the addition of patches, and in some cases, papillary muscles themselves can be surgically relocated and chordae tendinae can be cut to achieve proper valve closing and lessen the amount of valve regurgitation (Kron et al. 2002, Rendon et al. 2002, Urbanski 2010).

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When repair is not an option and heart valves must be replaced, there are two basic types of replacement valves in use today: mechanical valves and biological valves. Mechanical valves are totally man-made valves that are generally well tolerated by the body. The most common type of mechanical valve is a bileaflet valve that consists of two carbon leaflets covered with polyester knit fabric. Valves of this type have been used successfully for 30 years. They are designed to last a lifetime and are very durable. However, patients who receive these valves will require treatment with anticoagulant medications for the rest of their lives to prevent formation of blood clots on the mechanical parts of the valve. Biological valves, also called bioprosthetic valves, are made of human tissue or, more commonly, the tissue of pigs or cows. Pig valves, either mounted on frames called stents, or stentless, have been in use for 30 years. Biological valves made from the pericardial tissue of cows have been used successfully in the U.S. since 1991 (Gillinov 2011). Biological valves that are made from animal tissue are called xenografts. Biological valves that are made from human donated tissue are known as homografts or allografts. The term autograft is used when human tissue is moved from one place to another in the same human heart. For instance, in the Ross Procedure, a patient’s own pulmonary valve is used to replace his aortic valve and the patient’s pulmonary valve is then replaced by a pulmonary homograft (Gillinov 2011, Takkenberg 2009).

It is estimated that 300,000 people have surgery each year to replace heart valves that have ceased to function properly and cannot be adequately repaired. The majority of these people will have a post-surgical dependency on anticoagulation medications and/or face additional surgery later in life as transplanted valves wear out and have to be replaced. A very active field of investigation today centers on developing heart valves made of human tissue that would have some capacity for repair and remodeling in response to stress and would free patients from the lifelong dependency on anticoagulants or subsequent valve replacements that characterize conventional heart valve replacements. Much progress is being made in the development of autologous heart valves, made from the patient’s own cells, through the application of tissue engineering (Flanagan et al. 2007, Sutherland et al. 2005).

The challenge for researchers who are trying to make autologous heart valves is multidimensional. They are striving to create a valve that is compatible with human tissues, does not induce thrombogenesis, and is capable of growth and remodeling in vivo. The valve must lend itself to implantation using currently acceptable surgical techniques, it must perform with hemodynamic perfection and it must be sturdy enough to last a lifetime and substantially improve the patient’s overall quality of life. To achieve these goals researchers must determine the best and most practical ways to form and condition autologous heart valves. They must develop evaluation standards for the valves and devise non-invasive ways to monitor the valves in living animal systems. It is a highly complex undertaking; if successful, it will allow patients to overcome the fundamental drawbacks of mechanical and bioprosthetic heart valves (Apte et al. 2011).

Tissue engineering combines the biological principles governing cell growth and cell proliferation with engineering know-how to produce new living tissues that are identical or very close to their biologically derived counterparts. There are two basic approaches to building the desired valve architecture, which is known as a valve construct. In one approach researchers design a biodegradable, polymer-based support scaffold or matrix which can be fabricated in the exact shape of the desired valve. In another approach, researchers start with decellularized biological tissue of human or animal origin which provides a natural matrix. From either starting point, the second step is to seed the scaffold or decellularized tissue with autologous cells that have been harvested and grown in the laboratory. The autologous cells attach themselves to the microstructure of the scaffold or decellularized tissue, and the whole unit is then subjected to various forms of stimulation that encourage proper tissue growth and maturation. The goal is to promote maximum tissue strength coupled with maximum functionality (Schumann et al. 2009, Zimmermann et al. 2003, Weber 2011). As the biodegradable scaffolding disintegrates, the seeded cells begin to form their own extracellular matrix, which allows them to proceed to tissue formation. Scaffolding is also frequently used to provide appropriate growth factors to the proliferating cell populations (Khademhosseini et al. 2005, Howard et al. 2008, Weber et al. 2011).
Scaffolds and decellularized tissues both have a range of features that can affect their efficacy. The architecture of the scaffold has the ability to influence the action of the cells that are growing on it. For instance, scaffold engineering can cause mineral deposits to accumulate in certain regions of the scaffold, and altering the size of the scaffold microstructure from nanoscale to microscale can cause changes in the formation of the cytoskeleton of the forming cell (Meredith et al. 2007). One of the major challenges in working with biodegradable scaffolds is to match the appropriate cells to the proper form of scaffolding. In addition, the chemicals that are added to the biodegradable scaffold polymer must be carefully monitored since they are intended to modify the scaffold in specific ways (Howard et al. 2008). Decellularized valves that have had their native cell populations removed by detergents and trypsin are thought to have fewer rejection issues than biodegradable scaffolds. However, they are believed to be more immunogenically active, and since they are derived from live tissue and can be diseased, the possibility of the spread of disease from donor to host cannot be discounted. Allogenic decellularized scaffolds tend to evoke fewer immunogenic responses because they originate from the same species as the recipient. However, allogenic donors are limited, and xenogenic donors are commonly used so the recipient’s immune system must be consistently monitored (Dohmen and Konertz 2009). Because there are so many potential variables associated with both scaffolding and decellularized valve tissues it is important to keep experimenting with new scaffold structures and decellularized tissues until the best materials can be identified for each purpose (Howard et al. 2008).

Machines known as bioreactors are used in the process of tissue engineering. They are essential for providing the known chemical and physical conditions that mimic the environment of living cells. They are structured to provide a means of assessing the behavior of an array of biological substitutes to a variety of biochemical cell signals and variable mechanical forces (Alberti, 2009). Specifically, bioreactors are used to control essential factors such as temperature, oxygen levels, osmolarity, density and pH. They are also used to stimulate or regulate physical conditioning factors such as tissue compression, application of shear forces, the rate of interstitial blood flow and variations in fluid pressure (Weber 2011). Bioreactors are extremely important in the development of heart valve tissue because fibroblasts and endothelial cells have growth responses to mechanical signals as well as to chemical signals. The forming heart valve becomes stronger as it is mechanically stimulated. The bioreactors most commonly used in heart valve engineering are called pulsatile bioreactors. The machine’s inner chamber houses a unit that can generate pulsatile flow and provides an area where a culture medium exchange can be set up to provide growth factors to the forming cells (Sarraf et al. 2003, Mol et al. 2006).

A great deal of study has been done to determine what cell populations will work best for seeding the scaffold of decellularized tissue. The heart loses its ability to produce cardiac myocytes very soon after birth, and it does not have a large number of stem cells or reserve cells of any type from which to grow a suitable population of cells for seeding purposes. In the past, researchers have attempted to grow the patient’s own healthy heart cells in vitro for seeding purposes but these cells have been shown to be invasive and a high percentage of them have proven to be damaged or diseased. Consequently, it is essential to find cells that mimic heart cells for use in seeding scaffolds or decellularized valve tissues (Li et al. 2008, Howard et al. 2008). Today researchers are turning to stem cells as sources for seeding; exploring adult marrow-derived cells, embryonic stem cells, bone marrow-derived mesenchymal stem cells, resident cardiac stem cells, endothelial progenitor cells, and umbilical cord blood stem cells (Li et. al. 2008). It is currently believed that in order to closely duplicate the natural characteristics of a heart valve, it is essential to match the myofibroblast-fibroblast cell type as a part of the process. Myofibroblast-fibroblast cells normally produce the extracellular matrix of heart cells and are responsible for the opening and closing mechanics of the valve. Endothelial cells are another necessary cell type since endothelial cells

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significantly improve quality of life and life expectancy. Researchers believe that valve replacement can significantly improve quality of life and life expectancy. The number of patients needing valve replacements could top 850,000 per year by 2050 with the greatest increases projected to be in third world countries where access to valve replacement has historically been very limited. Researchers believe that valve replacement can significantly improve quality of life and life expectancy. For instance, without valve replacement a 60-year-old man who suffers from severe unrepaird aortic stenosis has perhaps 4 years to live. His life expectancy can be increased by 13 years with a successful valve replacement. As the quality of valves improves, his life expectancy could perhaps be extended the full 18 additional years that a 60-year-old man with no heart problems can expect to enjoy (Yacoub et al. 2005).

Creating a perfect valve is a daunting proposition since normal heart valves perform incredibly complicated functions. They have a direct effect on myocardial function and they help to regulate blood flow into the coronary vessels, the lungs, and the aorta. Researchers have shown that valve movement precedes the movement of blood from one heart chamber to another in anticipation of hemodynamic changes. During the cardiac cycle the various valve components change their size and shape over and over again in response to sympathetic, parasympathetic, and sensory innervations. A vast array of vasoactive substances constantly interacts with heart tissue. These functions are all possible because of patient-specific gene expression patterns in different areas of the valve. Valve cells secrete unique components of the extracellular matrix and the matrix in turn influences the valve's physical properties. Valve cells constantly communicate with the extracellular matrix to influence the generation of appropriate valve tension, polarity and response to constantly changing chemical and physical stimuli (Yacoub et al. 2005). The quality, quantity, and complexity of a heart valve's interaction with its environment is staggering. Autologous heart valves that can ultimately replicate all or most of the biological functions of normal heart valves will change the landscape of treatment for valve diseases and give new hope to thousands who wish for a better quality of life and a longer life span.

* Megan Placido graduated from Arcadia University in May of 2010 with a major in Biology. Excerpts from her Senior Thesis "The Use of Stem Cells in Autologous Heart Valves" appear in this article.

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The Vitruvian Man Exercise: Preparing Exercise Professionals to Detect Segmental Deviations

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Background

Historically, the teaching and coaching of exercise has included an element of interpretive anatomy with three distinct applications. The most recognized and obvious is the selection of exercises based on anatomical structures in which adaptation is desired, be it cardiovascular, pulmonary, skeletal, neural, or muscular. Failure to understand structure weakens an exercise professional’s ability to induce appropriate adaptations, or in other words, improve fitness.

A second application of anatomical knowledge is the use of anthropometric data in the identification of individuals with physical dimensions associated with success in sport (Bourgois 2000, Claessens 1998, Grimston 1986, Kansal 1980, Pipes 1977). It has become rather common to see sport coaches select potential athletes for a sport or a specific position within a sport based upon physical dimensions. Most frequently this occurs in developmental, school-age, and high school sport, where the coach is initially presented with no other means of selection or information other than stature – a simplistic application of anthropometric measurement.

A third, less understood, but important application, is an aptitude in visual evaluation of an individual’s unique anatomical structure in order to place the trainee in a position that [1] is correct for producing efficient movement through a task appropriate range of motion and [2] provides a foundation for safety. Development of this third application is an area of weakness in virtually all university courses related to anatomy. Traditional anatomy courses do not address the concept that mastery of anatomy is essential for developing competency in the analysis of human movement. Anatomy and physiology courses designed for a wide range of allied health professionals similarly do not present information within this topic area, as it is not central to all of the allied health professions utilizing such courses. Even “functional” anatomy courses created specifically for exercise professional preparatory programs rarely provide for student competency in this important ability.

There are a growing number of exercise professionals and academics who believe that there exists an anatomical orientation of the body that creates efficient movement unique to individuals and individual exercises. For example, it has been demonstrated that a vertical alignment of the navicular bone, bar of the barbell, and scapular spine will produce a linear bar path in the deadlift exercise (Kilgore 2009). Deviations from this alignment induce curvilinear, inefficient motion. Given the variation in the length of body segments encountered across human populations, the composite of the joint angles contributing to this alignment produces a different appearance between individuals (Fig. 1). It has also been suggested that body segmental variations alter ambulatory exercise performance. And in fact there is data demonstrating that segmental differences alter gait and velocity transitions (Monteiro 2010).

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Fig. 1. At the start of the deadlift the navicular bone, the bar, and the most medial and inferior aspect of the scapular spine are aligned in order to produce a straight bar path during ascent. A simple variation in arm length effectively changes the constituent joint angles required to create that alignment and changes the appearance of the exercise position even though each is correct (reprinted from Kilgore, 2010).

There exists a dearth of modern literature relevant to this topic, and this combination of curricular and literary omission affects millions of average gym-goers each year. Their trainers or coaches have never been provided any guidance on how to adapt exercise positions for individual variations in body dimensions among other exclusions (Stacey 2010). As such, it is quite common to see the one-size-fits-all approaches to teaching exercise positions printed in a majority of the authoritative professional literature (National Strength & Conditioning Association 2008) adopted by practitioners. This is a limitation affecting both the exercise professional’s competency and the quality of teaching and coaching received by the trainee.

So a curricular content inclusion question arises: how does one approach presenting students with a means to detect anatomical variations and how to accommodate them? The first step is to facilitate determination of whether someone has longer than normal legs, shorter than normal arms, longer than normal trunk, etc. For this to occur, one must have a reference standard.

Is there a reference standard that can be used in this application? A review of the scientific and exercise professional literature does not provide a direct answer – unless we consider the works of Leonardo da Vinci. One of the easiest methods of determining if an individual deviates from “normal” anthropometry is to use the historical concept of normal human dimensions created by da Vinci (circa 1487). Virtually everyone is familiar with the “Vitruvian Man”, da Vinci’s map of human proportions, center of mass, and center of gravity (Fig. 2). While exercise professionals may not be familiar with the utility of the illustration, it is a convention used in art instructional units around the world as a method towards creating proportional representations of the human body. In application within the exercise arena, one can simply use an individual’s head length, as did da Vinci, as a basis for body segmental analysis. Using the Vitruvian map, field practitioners can visually, and rapidly, determine if a body segment is different from da Vinci’s prototypical human male.

Fig. 2. da Vinci’s Virtuvian Man (Uomo Vitruviano). From the collection of the Gallerie dell’Accademia, Venice, Italy.

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The Exercise

A hands-on laboratory exercise using da Vinci’s work as a basis was developed to help students grasp both the technique and utility of visual anthropometric assessment.

**Step 1** of the learning process is to create an understanding of the Vitruvian model. The students are provided with a page-sized copy of the da Vinci’s illustration (Fig. 2) along with a set of observations regarding segmental dimensions derived from his notes and illustration.

From the top of the head to the bottom of the chin is one-eighth of a man's height.

A man's height is four cubits, which conveniently is eight heads in overall length.

The length of the outspread arms (wing span or reach) is equal to his height, or eight head lengths.

The width of the shoulders is a quarter of a man’s height, or two head lengths.

The distance from the elbow to the armpit is one-eighth of a man's height, or one head length.

The distance from the elbow to the tip of the hand is a quarter of a man’s height, or two head lengths.

The torso, from sternal notch to the level of the hip joint, is two and a half head lengths.

The upper leg is two and a quarter head lengths.

The lower leg to the ground is two head lengths.

This widest point of the hips is one and a half head lengths.

**Step 2** is the identification and palpation of the anatomical sites and features associated with each segment described. Each segment is discussed with respect to appropriate anatomical landmarks that bound the ends of each segmental measure. Three aids are provided each student pair to go along with the discussion: [1] the Vitruvian Man worksheet (Fig. 3), [2] a life size articulated skeleton, and [3] a laboratory partner. By having all students physically locate each structure on the illustration, skeleton, and on a fellow student during the presentation, they become familiar with the anatomical terrain and become prepared to perform the upcoming measurements accurately.

**Step 3** is the creation of a subject-specific measurement device. Each student pair is provided two one-foot-long lengths of nylon cord. On this cord a set of marks is made. One represents the height of the head from the most inferior point of the chin to the highest and most superior point on the skull. Another mark is made at midpoint of the cord. The two resulting halves are bisected again to produce a measurement cord with increments of 0.25 head lengths.

**Step 4** is measurement of the Vitruvian segments on the student partner using the customized cord. The identical measures are taken again in centimeters with a standard measuring tape. This step reinforces familiarity with topographical anatomy, develops a sense of measurement accuracy, and begins to develop a visual sense of segmental dimensions and orientation. Each measurement is recorded on a data reporting form created on a large whiteboard. This form has spaces for the data from each student in the class. Once the complete data set is on the whiteboard, the students copy the data set.

**Step 5** involves simple statistical evaluation of the data using Microsoft Excel. Students are instructed briefly how to calculate means and standard deviations for each segmental measure for the entire class. This is assigned as out-of-class work in the form of a laboratory report. Five specific questions are included on the student’s report form:

Does the data from the class conform precisely to da Vinci’s model of human dimensions? If not, describe the variance. If they are similar (within one standard deviation of the class mean) can the model still be used as a guide?
Are there gender differences in the data? If so specify which dimensions display the greatest disparity.

Were any two subjects alike? What does this tell you about the value of being able to recognize segmental variation in your future trainees?

**Step 6** takes place the following class period where the data, as analyzed by faculty, is compared to student reports and the directed questions are discussed.

Generally, what the students find is that the modern human does not conform precisely to the Vitruvian template, rather they see variation among the body segment lengths. However, they do find their results to be within one standard deviation of da Vinci’s proposed segmental relationships. As such the template can still be cautiously applied.

Student feedback from this exercise has been exceptionally positive, with many reporting “Aha!” type moments when the connection between the presented information, the activity, and professional practice was realized. This laboratory, presented very early in the course schedule, has improved student participation in both lecture discussions and laboratory activities in subsequent portions of the course. Making a tangible connection between familiar art, learning, and professional practice has made the study of anatomy appealing to students who often look upon the study of anatomy as, at best, a necessary evil.

**Literature Cited**


■ *(Teaching Tips article 2 begins on next page)*
Excitation-Contraction Coupling in Skeletal Muscle: A Love Story?

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We would like to share with the HAPS community a novel teaching tool that is regularly used in our undergraduate human physiology course at the University of Wisconsin School of Medicine and Public Health. This five credit course is offered every semester (including the 8-week summer session) and is now enrolling over 1000 undergraduate students per year. Because this one semester course surveys the whole of human physiology, the teaching staff recognizes the need for teaching aids that turn complex, dynamic processes into engaging analogies with a “hook” that reminds a stressed student how to travel through the steps of the mechanism. An example of this can be readily seen in our skeletal muscle module wherein the students must learn and commit to memory the steps of excitation-contraction coupling and the cross bridge cycle. Something so elemental and multi-faceted has been greatly aided by turning the whole process into a narrative story which we now present.

(Continued on next page)
As we blush a little at their disregard for privacy, the exhibitionist $Ca^{2+}$ ions and TROPONIN (C) molecules bond for brief moments. But it’s enough: TROPONINS, dizzy in their ionic embraces, relax and slide away, dragging their strands of TROPOMYSIN away with them for a short respite. (If there were ever a time they would like a nicotine bolus, it would be now!)

As this crowd of molecules briefly disperses, ACTIN is again revealed. It sees its long-lost MYOSIN! MYOSIN, still primed for action and bound to ADP and $P_i$, rushes toward ACTIN.

They bind in an awkward group embrace! (ADP•P•M•A) MYOSIN bends its head in gratitude. The embrace is so strong, $P_i$ is released. (ADP•M•A + P)

MYOSIN and ACTIN become oblivious in their passionate embrace. The stubborn voyeur, ADP tries to hang on but eventually departs, leaving them alone. (M•A + ADP)

As myosin and actin cuddle, we hear their whispered giggles as they rejoice in their re-connection.

But what is this we behold next?

Why….it’s….oh, such sweet irony. It is the approach of another ATP molecule!

Over the past two years, our instructors, teaching assistants, and peer-led team facilitators have independently recognized that this story’s versatility is in fact its best attribute. The manner in which this story is utilized in a classroom can be easily adapted to best match both the instructor’s personality and the student group personality. Below are five representative comments from instructors who have used this narrative in their classroom:

1. “For me, the part where ATP binds is reminiscent of a game that I like called Mad Libs. It occurred to me, after I read the “playlet”, that if I omitted a few words, this would be a fun Mad Libs game for my students to complete.”

2. “I’ve done this kind of thing before in other settings—having students move around the room and act out the actions of molecules thus helping to prompt kinesthetic memories—and really enjoy it.”

3. “I copied it to distribute, but we went through it as a class. I led them through it verbally, reading very dramatically to make it more fun, like the soap opera it is, and they filled in the blanks verbally, though we did use the chalkboard in part to write down the words. When they got stuck, I used a list of word options on the chalkboard that they could choose from.”

4. “I purposefully use repetition to try to stick the key players (Actin, Myosin, Troponin, Tropomysin, Calcium, etc) in my own head, and thus hopefully stick them into the students’ heads! A wise salesperson knows this trick well.”

5. “In using this tool, some of the underlined words can be blanked out OR filled in as needed, to adjust the “playlet” to the level of the students being taught. Entire sections can be omitted as needed (e.g., the DHPR-RyR connection).”

Not surprisingly, student reaction to this teaching tool is always positive regardless of how or when it is utilized and regardless of an instructor’s dramatic abilities. Quantitative information on how it ultimately affects learning and performance on assessments is not yet available. This is due in part to the fact that the skeletal muscle unit is only one part of a larger exam unit covering muscle physiology and endocrinology. However, it is clear from conversations with students that they are definitely more engaged by this story, and thus they are more likely to remember the steps of skeletal muscle excitation-contraction coupling and the cross bridge cycle.
Acids, Bogs, Energy, and Tension Producing EDU-Snippets

EDU-Snippets – A column that survives because you - the members - send in your Snippets

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EDU-Snippets is a column designed to let you, the members of HAPS, share your “ways to make sure your students get it.” Since EDU-Snippets began, our members have been continuously amazed at how many teaching and demonstration ideas pop up and are easily transferred from one instructor to another through Snippets. This edition is no exception. Below you will see some great ideas – fast, fun, easy, and cheap! Those low-budget or no-budget ideas are sometimes the best!

When I put out the call for ideas for this issue, some interesting “magic moments” came through. Some are included here and some will be in later issues. Some other ideas were also generated from the Haps-I discussion listserv. Some of this issue did come either directly or indirectly from the discussion listserv, so some of you may recognize a few points. Meanwhile, the EDU-Snippets desk certainly encourages everyone to keep right on submitting! Please keep your wonderful ideas coming! This column thrives on what you do – both in lab and in lecture.

I. Acidic or Basic Snippets

A number of people were interested in acid-base understanding this time around. Here are three great ideas for going “one step beyond” in helping our students conceptualize the many facets of acid-base balance.

A. Robert Rawding (Gannon University, rawding001@gannon.edu) has been concerned about the parameters of acid-base balance, so he introduced a very practical way to apply the principles. Maybe some of you can access similar equipment and/or modify the idea here to fit what you do have at your particular institution.

This coming fall semester, after coverage of acid-base balance in lecture, I will be utilizing our medical simulation center to show patients in one of these scenarios: metabolic or respiratory acidosis and metabolic or respiratory alkalosis. I will prepare the students by teaching them how to use a Davenport nomogram (a diagram used to predict disturbances in acid-base balance). I will then give the students a homework exercise consisting of six to eight cases in which I will have included for each “patient” the pH, pCO2, bicarbonate level, and so forth. Students will have to deduce the cause for the imbalances for each of the case patients. Many of my students are in nursing, but some are in pre-pharmacy, sports and exercise science, or nutrition. It is important that they understand the cause(s) of acidosis or alkalosis in order that they might be able to affect the proper treatment.

This is a nice hands-on lab exercise where students can vicariously observe patient symptoms and track blood gases on the bedside monitors. The visual experience definitely aids in understanding. You might even suggest that your students Google the Davenport nomogram and learn a bit about its history and development.

B. Meanwhile, Betsy Ott (Tyler Junior College, bott@tjc.edu) concentrated on the acidic properties of carbon dioxide. Betsy mentions setting up pH standards for more exact measurements. Putting Betsy’s ideas together with Bob Rawding’s experiment above, most of the ingenious members of HAPS should be able to come up with numerous very precise ways to vary specific parameters in acid-base measurements. These might include adding vinegar or NaCl to the water in specific or not specific quantities, or varying the amount of water (calibrated amounts) and calculating the time required for the color to change. Another idea (if you have access to more elaborate lab equipment) would be to do the experiment in a sealed container using known aliquots of CO2. You can basically make this experiment as simple or as complicated as you would like. And you can combine and graph the class results.

We developed a brief, visually-memorable demonstration to show the acidic effect of carbon dioxide. We add phenol red (readily

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available where swimming pool supplies are sold) to tap water, and have students exhale through a straw into the solution, timing how long it takes for the color to turn yellow. Then they add bicarbonate to water and repeat, showing the much longer time needed to make the buffered solution turn color. You can set up standards at various pHs, or just have a bottle of vinegar to show that acid turns phenol red to a yellow color.

C. And finally, Larry Anthony (Camosun College, anthonyl@Camosun.bc.ca) added one more interesting dimension – a dimension that might be very handy in working with the blood flow lessons. And, with usual HAPS ingenuity, many of you will certainly come up with numerous variations on what Larry has presented here.

Throw a thickening agent into the water to make it more viscous, use narrow and wide diameter straws, and you can add hemodynamics to the illustration!! The thickening agent can be anything from gelatine to corn starch. If you are not familiar with how to best utilize the thickening agent (or to best get an even suspension), you might want to practice before doing this in class. You can set up the standard liquid to approximate the viscosity of normal blood. You can then have solutions with slightly higher and slightly lower viscosities (calibrated, of course). Your students can measure the role viscosity may or may not play in acid-base balance. You can always ask them why a change did or did not occur. You can also chart the differences between the two straws in the various solutions.

Experiments involving acid-base variations are indeed numerous! They are simple, they are low-budget, and they are hands-on. Furthermore, the students can see the physiological principles.

II. Hierarchical “Rattlin’ Bog” Snippet

Tama Fox (South Seattle Community College, auntama@gmail.com) was surfing the biological web and was reminded about how jingles stick in our minds. She thought that introducing this little Irish ditty might be very useful in helping our students remember hierarchical arrangements.

This is an ear worm for students to remember the hierarchy from atoms to ecosystems. I apologize in advance if “Oh row the rattlin’ bog” gets stuck in anyone’s head. It really is an “ear worm” and it really will help the students remember what goes where in a list of how something goes – whether you are breaking down or building up a system. Furthermore, you can change the lyrics any way you wish to fit the particular unit or chapter you are covering.

The first website listed here is more straightforward and, when you play it, the lyrics are more easily understood. If you (or your students) are more into the jazzed up version, go with the second website.

http://www.youtube.com/watch?v=TTSO-edqI_0&NR=1&feature=fvwp

http://www.youtube.com/watch?v=xn v9GB8xvrw&feature=related

The first website follows the pattern you and then check the Internet for more versions. From there you can take this in any direction you want because you can put any logical sequence into a bog – anything from a human to a Venus fly trap to a food web to even any anatomical system.

III. Energetic Snippet

Dennis Kingery (Metropolitan Community College, dkingery@mccneb.edu) spent some time wondering how we could explain to our students how energy is extracted from food.

As a simple demonstration of the need for a controlled, step-by-tedious-step approach to extracting energy from food, I can suggest a couple of ideas. First, set a scrap of paper aflame in a non-flammable container – such as heat resistant glass beaker or plate. (An aluminum pan can be used because, although aluminum will burn in the atmosphere, the temperature and time for its disintegration must be higher than will be happening here.) As the paper burns, you can

(Continued on next page)
invite the nearest students to warm their hands or roast marshmallows, emphasizing the fact that there is a great heat loss. Soon the fire goes out and there is a fragment of ash left behind. Ask students “Although we often say we ‘burn’ our food, would we ever want to use a process like this one? They will readily respond that it would be too hot, too fast, and too wasteful for use in living cells.

(If you do this experiment, I urge you to take care that any safety sensors in your classroom are not so sensitive that they sprinkle the room to put out the fire!)

After the paper burning demonstration, present a plastic liter beaker filled with paper wads, identifying it as representing the energy content of a molecule of glucose. For example, propose that we need a flameless transfer of energy, and toss the beaker to a student in the second or third row. Whether it is caught or not, it is likely that paper wads will spill out. The analogy demonstrates again the unavoidable waste of energy involved in quick energy transfers.

Finally, produce another liter beaker of paper wads and pass the wads one by one, student to student, to that same student still holding the plastic beaker from the first toss. This stepwise process (analogous to cellular respiration) allows the student to fill the beaker (analogous to ATP) efficiently for quick access at some later time.

Now begin your review of cellular respiration. Don’t forget to clean up the floor! Where did that energy to pick up those paper wads come from?

IV. Tense Snippet

Another problem students often have – particularly in the early phases of their Anatomy and Physiology studies – is in understanding surface tension and subsequently in understanding the importance of surface tension in respiratory physiology. Sara Van Orden (Quinebaug Valley Community College, svanorden@Qvcc.commnet.edu) sent in the following idea.

To demonstrate the surface tension of water, I take several small (100mL) beakers and fill them with water. The students are in groups and I instruct one student from each group to carefully “drop” an insect pin onto the water’s surface to show how tough that surface is. As the students are watching the pin float, I go around to each group and carefully let a drop of dish detergent drip down the inside of the beaker. When the surfactant hits the water, it breaks the surface tension and the pin miraculously sinks. This seems to really drive home the necessity of surfactant in the lungs. I relate this to normal breathing as well as to one of the problems associated with premature babies. You can take the subsequent discussion in numerous directions and you can bring in the roles of surface tension and surfactant in quite a few other respiratory problems.

You can also suggest that the students do this experiment at home using a toothpick and a glass of water. Put a sketch on the board showing them how, literally, the water’s surface has an indent when an insect pin or a toothpick is gently placed on that surface. Invite the students to experiment with such concepts as the mass required to break the surface tension or the force required to cause the object (toothpick) to sink immediately. You, or your students, can think of countless other parameters. If you teach older than average students, tell them they can use this to teach their own children about a critical point in human physiology.

V. And We Hope You Will....

Keep those cards and letters coming! Thank you all for your EDU-Snippet contributions. The influx of Snippets has been great!
Victoria HAPS Scavenger Hunt winners!

Tom Lehman, Steering Committee Chair
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During the annual HAPS conference, the Steering Committee likes to make a direct connection with First-Time participants. The Steering Committee (made up of the Chairs of the various committees in HAPS) hosts a Scavenger Hunt where First-Timers must track down each committee chair and gain their signature. Along the way, they learn about the different committees and how they can become involved. It’s an incredible activity that spans the first two days of the conference, helping First-Timers to meet many new HAPSters and learn more about the Society and what they can gain from it.

This is the sixth year of the Scavenger Hunt and we have had tremendous feedback about the activity. It’s a blast to see First-Timers from previous years help newcomers to find Chairs and integrate them into the great big family of HAPS. AD Instruments has been very generous in offering prizes each year for First-Time participants (if you look closely next time, you’ll invariably see some HAPSters wearing caps from previous years). Of all of the participants who complete their Scavenger Hunt cards, one lucky winner receives free conference registration to the next year’s conference.

Congratulations to all of the First-Timers who successfully completed the Scavenger Hunt at the 2011 HAPS conference in Victoria! You demonstrated the enthusiasm and dedication that we like to see in our profession. We look forward to seeing each of you at Tulsa in 2012!

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