Are Cardiologists good for Cardiology?

On a snowy Manhattan day in 1949, 14 cardiovascular physicians met to form a "*revolutionary"* new society called the American College of Cardiology thus paving the way for a new *subspecialty* and the first "sanctioned" cardiologists.[1] But was this a good thing? When looking at data over the past 35 years, we note that survival from a heart attack has improved from half the patients having an acute myocardial infarction (AMI) dying, to a third, however the overall number of AMI's (all sizes) has increased to around 1.5 million annually.[2] The number of AMI's increasing may be a deceiving number though as it is not age properly adjusted for the increase in population. But what is not deceiving is the number of patients with arrhythmia's and congestive heart failure. Lets look at atrial fibrillation. An estimated 2.66 million people had atrial fibrillation in 2010 and is on the rise. If projections hold true, as many as 12 million people will have the condition by 2050.[3] The estimated cost of the treatment of atrial fibrillation in 2005 was a staggering $6.65 billion per year, which included costs of hospitalization, in- and outpatient physician care, and medications.[4] One can only imagine what is will be in 2050. In the case of CHF, it is also on the rise with 550,000 new diagnoses per year.[5] Nearly 5 million Americans are currently living with congestive heart failure (CHF).[6] CHF is the first-listed diagnosis in 875,000 hospitalizations, and the most common diagnosis in hospital patients age 65 years and older.[7]

Naturopathy

The deep-rooted philosophy's of Naturopathic medicine include a concept that suppression of one disease can cause another. I ran in to this exact issue during a meeting with three interventional cardiologists. The topic came up about the alarming increase in heart failure. One of the interventional cardiologist stated that even if they where saving a life with a stent placement or bypass (which is highly, highly debatable) they where just trading a fast death for a slow one due to the fact that heart failure, post tent and bypass is on the rise. The two other cardiologists agreed. I stated the possibility that by suppressing one disease (therefore by definition not curing it), you where allowing it to manifest in another way. They all agreed. It was an interesting moment when a Naturopathic

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[1] http://www.cardiosource.org/en/ACC/About-ACC/Who-We-Are/History.aspx, Home Page, 2013
[2] http://www.buzzle.com/articles/heart-attack-statistics-by-age.html, Feb 2013
[3] These are figures from a variety of sources, all compiled in Lloyd-Jones D, Adams RJ, Brown TM, et al. Heart Disease and Stroke Statistics—2010 Update: a report from the American Heart Association. *Circulation*. 2010;121:e91.
[4] Coyne KS, Paramore C, Grandy S, Mercader M, Reynolds M, Zimetbaum P. Assessing the direct costs of treating nonvalvular atrial fibrillation in the United States. *Value Health*. 2006 Sep–Oct;9(5):348–56
[5] http://www.emoryhealthcare.org/heart-failure/learn-about-heart-failure/statistics.html, Feb 2010
[6] IBID
[7] IBID

cardiologist and three conventional ones who practice an almost exclusively suppressive and invasive system of medicine, agreed on a Naturopathic concept. But how do we take this concept and define it? How to we move a philosophy into practice? The key is the linking of the body's subsystems into an all-inclusive supersystem, or in other words "despecialize" the body.

Neuro-Endo-Immuno-Inflam Cardiology

Lets shorten this to "NeuroCardio" for ease of reading and define our new viewpoint. In the recent past Dr. Gottfried Kellerman has pioneered the concept of the NEI supersystem. In this system, the nervous, endocrine, and immune system are viewed as an integrated model versus a sectioned off for "specialty" model. This model is measurable using standard labs along with adrenal and neurotransmitter testing along with inflammatory and anti-inflammatory cytokines. The technology allows the imbalances between the systems to be identified and treated. One of the keys here is the opinion that norepinephrine plays in the balancing and control of norepinephrine. When the brain (locus coeruleus) and the adrenal medulla decrease the output of norepinephrine, the body's ability to manage and monitor the cytokine system falters.[8] Interestingly enough is the fact that the immune system can also produce norepinephrine, so the overall measurement of norepinephrine can be elevated, while the brain and medulla are still low. Norepinephrine in the inflamed or depressed or tired patient when measured high, may be an indication of an actual norepinephrine deficiency, in an inflamed patient.

NeuroCardio- Coronary Artery Disease

Inflammation and oxidation are the two top underlying causes of heart disease per most experts. However, we have had very little in the way of laboratory analysis to diagnose these underlying causes. CRP- HS has been a good start, but is far from giving us a clear avenue for preventative treatment. Recently a new test for oxidative LDL has come to market, which is a huge step in the right direction. But what about the inflammation? Currently we have definitive research, which demonstrates that pro-inflammatory cytokines can lead to predictable imbalances in both cortisol and neurotransmitter levels.[9] Performing a cellular cytokine analysis along with neurotransmitter testing can diagnose the underlying inflammatory mechanisms of that individual patient. Once discovered, these neurotransmitters and cytokines can be balanced through a variety of modalities including nutrition stress management, and nutritional supplementation. In addition, depressed patients have been found to have higher levels of pro-inflammatory cytokines, acute phase proteins, chemokines and cellular adhesion molecules.[10] Pro-inflammatory cytokines have been definitively linked

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[8] Rommelfanger KS and Weinshenker D. *Biochemical Pharmacology*. 2007 (74) 177-190.

[9] Brain Behav Immun. 2003 Oct;17(5):373-83.
Cortisol responses to mild psychological stress are inversely associated with proinflammatory cytokines.Kunz-Ebrecht SR, Mohamed-Ali V, Feldman PJ, Kirschbaum C, Steptoe A.Department of Epidemiology and Public Health, University College London, 1-19 Torrington Place, London WC1E 6BT, UK. kunz@ift.de

[10] Cytokines sing the blues: inflammation and the pathogenesis of depressionCharles L. Raison, Lucile

to lower GABA and serotonin expression resulting in depression.[11] [12] Depression is recognized as one of the leading risk factors for developing CAD.[13] Simply put, a happy patient is a happy heart. Balancing to the correct levels of serotonin and GABA may not only improve their quality of life but improve the quantity of life as well. In looking specifically at women, healthy estrogens are necessary for healthy mucosal linings including the intima layer of a coronary vessel.[14] Emerging science demonstrates that estrogen levels are directly related to serotonin levels, so therefore the measurement and the restoration of serotonin is vital to the prevention and treatment of cardiovascular disease in women.[15] From now on, looking at cardiovascular products for patients need to be researched to not only work as an antioxidant and an anti-inflammatory (looking at the reduction of CRP-HS, and oxidized LDL) but also in reducing inflammatory cytokines such as IL-6, and TNF α. [16] To date, the best data appears to be coming in the from proprietary lycopene/tomato extracts coming out of Israel.
In a "NeuroCardio" world, urine neurotransmitter testing, along with cellular cytokine analysis (looking for abnormal pro-inflammatory markers) while establishing appropriate cortisol levels throughout the day and night, will allow for a truly integrative and highly effective approach for preventing CAD and AMI.

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[11] Differential Regulation of AMPA Receptor and GABA Receptor Trafficking by Tumor Necrosis Factor α David Stellwagen1, Eric C. Beattie2, Jae Y. Seo2, and Robert C. Malenka1

[12] *Neuropsychopharmacology* (2006) 31, 2121–2131. doi:10.1038/sj.npp.1301029; published online 1 February 2006 Preclinical ResearchThe Proinflammatory Cytokines Interleukin-1beta and Tumor Necrosis Factor-Alpha Activate Serotonin Transporters Chong-Bin Zhu1, Randy D Blakely1,2,3 and William A Hewlett1,2 [1]Department of Pharmacology, Vanderbilt University School of Medicine, Nashville, TN, USA Department of Psychiatry, Vanderbilt University School of Medicine, Nashville, TN, USACenter for Molecular Neuroscience, Vanderbilt University School of Medicine, Nashville, TN, USACorrespondence: Dr WA Hewlett, Department of Psychiatry/Pharmacology, Vanderbilt School of Medicine, Nashville, TN 37232-8645, USA. Tel: +1 615 343 0795; Fax: +1 615 322 5298; E-mail: william.a.hewlett@vanderbilt.edu Received 2 September 2005; Revised 16 November 2005; Accepted 28 November 2005; Published online 1 February 2006.

[13] Litchman JH, et al. Depression and coronary heart disease: Recommendations for screening, referral and treatment. A science advisory from the American Heart Association Prevention Committee of the Council on Cardiovascular Nursing, Council on Clinical Cardiology, Council on Epidemiology and Prevention, and Interdisciplinary Council on Quality of Care and Outcomes Research: endorsed by the American Psychiatric Association. Circulation, Oct 21 2008. 118(17):1768-1775.

[14] Intimal Estrogen Receptor (ER)β, But Not ERα Expression, Is Correlated with Coronary Calcification and Atherosclerosis in Pre- and Postmenopausal Women

Rose C. Christian, Peter Y. Liu, Sean Harrington, Ming Ruan, Virginia M. Miller, and Lorraine A. Fitzpatrick

[15] *Menopause,* The Journal of the North American Menopause Association: Relationship between Estrogen, Serotonin, and Depression. Archer, Johanna S. M. VMD, MS, MD, http://journals.lww.com/menopausejournal/Abstract/1999/06010/Relationship_between_Estrogen,_Serotonin,_and.15.aspx posted December 9, 2014

[16] Riso, P, Visioli, F., Grande, S., Guarnieri, S., Gardana, C., Simonetti, P., and Porrini, M J Agric Food Chem, 54: 2563-2566, 2006.

NeuroCardio- Congestive Heart Failure

There is a debate in CHF circles that the hallmark of diagnosis, the "ejection fraction", may be incomplete.[17] Markers such as BNP and others are becoming more prominent for risk stratification, and prognosis. As a clinician and professor I teach the neurohormonal response the body creates in response to a lower EF. (Ejection Fraction) This includes activation of the sympathetic system (epinephrine and norepinephrine) and the renin-angiotensin aldosterone system.[18] Pro-inflammatory cytokines, in particular tumor necrosis factor-a, have tremendous regulatory effects on energy metabolism, immune function, neuroendocrine and hormonal function.[19] These inflammatory cytokines are known to contribute to the progression of heart failure, and have been related to patients' prognosis.[20] Advanced heart failure can be considered a state of chronic (low-grade) inflammation.[21] In the "NeuroCardio" world we will diagnose the "low grade inflammation" component of the patients CHF through urine neurotransmitter and stimulated cellular cytokine testing in an attempt to risk stratify and create a prognosis. For example, if catecholamine's such as norepinephrine and epinephrine are elevated, and the all important for the heart inhibitory neurotransmitter taurine is low, than it may lead to a poor prognosis for the patient. Or if a patient's interleukins, cytokines, and TNF numbers are demonstrating a "low grade inflammation", than the expectation for patient improvement may be low. Remember however that supporting norepinephrine naturally in heart failure patients may still be needed, despite the fact that overall catecholamine production is elevated due to the anti-inflammatory cytokines that need norepinephrine signaling to work properly. In my opinion, most CHF patients need catecholamine support, to help calm the overproduction of catacholamines. But this is purely anecdotal. Again, many modalities can be used to bring these numbers into balance; so all forms of medicine are inclusive as treatment options. By linking the "supersytem" of neurology, endocrinology and immunology to inflammation we can focus our treatments on calming the neurohormonal response to heart failure. This may lead to fewer medications, and an improved prognosis. Currently we tend to give uniform treatments to almost all heart failure patients. With accurate neurotransmitter and cellular cytokine analysis, we can individualize the patient's treatment to optimize their current quality of life and their prognosis.

NeuroCardio- Atrial Fibrillation

There are two different factors involved in heart rate management: intrinsic and extrinsic controls. Intrinsic regulation of heart rate is the result of the unique nature of cardiac tissue - it is self-regulating and maintains it's own rhythm without direction. Extrinsic

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[17] CLEVELAND CLINIC JOURNAL OF MEDICINEVOLUME 69 • NUMBER 3 MARCH 2002

[18] Neurohormonal Activation in Congestive Heart Failure and the Role of Vasopressin

Kanu Chatterjee, Chatterjee Center for Cardiac Research, Division of Cardiology, University of California, San Francisco, San Francisco, California

[19] Immune and Neurohormonal Pathways in Chronic Heart Failure
Rakesh Sharma, BSc, MRCP, Stefan D. Anker, MD, PhD

[20] IBID

[21] IBID

controls are those that come from both hormonal responses as well as the commands from the nervous system: the central nervous system and the autonomic nervous system.[22] The SA node displays intrinsic automaticity at a rate of 100-110 action potentials ("beats") per minute. This "vagal tone" reduces the resting heart rate down to 60-80 beats/min.[23] Neurotransmitters come in to play in both the intrinsic and extrinsic heart rate control mechanisms. At present, research regarding the causes of A-fib present like a set of risk factors for heart disease but a little different. Risk factors include hypertension and alcohol use, but expand further to viral infections, pulmonary issues, and others.[24] But what is the underlying mechanism? What affect are these risk factors having on the neurotransmitters? Although not well-defined research shows that epinephrine, norepinephrine, and vagal tone are all implicated in the genesis of atrial fibrillation.[25] One odd fact is that in a patient who has prolonged atrial fibrillation the vagal tone and possibly the neurotransmitters normalize, which may be the underlying reason why atrial fibrillation is stubborn to resolve.[26] In clinical practice quite often we have warnings that a patient may be headed for atrial fibrillation, like episodes of palpitations, CAD, and high stress. In the "NeuroCardio" world we would immediately screen both cortisol and neurotransmitters to look for the "supersytem" imbalances before the try and prevent atrial fibrillation. We would also look at supplementation that not only helps reduce the biochemistry of stress, but also look to use supplements which support the calming or vagal tone aspects of the autonomic nervous system. A variety of modalities may be involved such as balancing the physical parts of the body through spinal manipulation, visceral manipulation of the stomach to try and allow for unimpeded vagal nerve tone, and relaxation techniques. Amino acid and methylation therapies may be used to help "nudge" the neurotransmitters back into alignment.

Are Cardiologists good for Cardiology?

It has been a debate since the beginning of specialization on whether this was a good idea or not. The idea of focusing on a specific part or system of the human body seams on the surface a good idea, but the results since the formation of have been mixed. It appears that we are reducing morbidity and mortality on the one hand, but possibly creating other morbidity mortalities on the other. Introducing the "NeuroCardio" approach to the world may be the total "supersytem" approach needed. By it's definition, specialization may not be capable of anything but suppression. Using the modern diagnosis of valid urine neurotransmitter testing, cellular cytokine analysis and saliva adrenal testing may be the "diagnostic" connection needed to validate approaches to these

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[22] http://www.heartmonitors.com/exercisetips/changing_heartrate.htm Feb 2013

[23] Weiss, S.A. Decker, "Anticoagulant Therapy and Antiarrhythmics", CNDA presentation, 2012

[24] Atrial Fibrillation Causes: http://www.mayoclinic.com/health/atrial-fibrillation/DS00291/DSECTION=causes1998-2013 Mayo Foundation for Medical Education and Research (MFMER). All rights reserved. A single copy of these materials may be reprinted for noncommercial personal use only. "Mayo," "Mayo Clinic," "MayoClinic.com," "EmbodyHealth," "Enhance your life," and the triple-shield Mayo Clinic logo are trademarks of Mayo Foundation for Medical Education and Research.

[25] **Role of the autonomic nervous system in vagal atrial fibrillation**

M P van den Berg,1 R J Hassink,1 C Baljé-Volkers,2 and H J G M Crijns3

[26] IBID

devastating medical conditions. In the world of “NeuroCardio” we would argue that this could and will create the opportunity for cure.