PHOENIX RISING SPECIAL EDITION

A Layman's Guide to the

The 2007 P.A.N.D.O.R.A. International IACFS Patient Conference
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Part II: Dr. Bell and Sleep / Dr. De Meirleir, the Gut and RNase L / What's New in CFS? – A Talk With the Experts / The Sand Castle Awards / Dr. Cheney's Keynote Address

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David Bell, MD Sleep Disorders are they the Cause or the side effect of CFS?
An engaging talker with a sly sense of humor Dr. Bell showed an impressive grasp of the sleep issues in CFS. He listed nine clinical sleep abnormalities that can be found in CFS and FM. Importantly Dr. Bell noted that studies have shown that 18% of 'CFS patients' don't have CFS: they have a sleep disorder that can, if identified, be treated successfully. He pointed out that sleep studies have shown that sleep architecture in CFS – the way patients move from one stage of sleep to another – is not dramatically different from that of healthy people. However, Dr. Bell is not a big fan of sleep studies. He noted one patient who didn't sleep at all during his stay – a fact noted at the end of the sleep lab report – but nevertheless tested out "normal"! *We will see evidence of parasympathetic nervous system activation during sleep in the Professional Conference – is this the missing element? Dr. Natelson is also studying cytokine production during sleep.*

Dr. Bell stated that sleep problems, at least so far as we understand them, are not the cause of CFS. He indicated that virtually everyone with CFS has sleep problems that can be treated fairly well but that doing so does not resolve their CFS. The most common sleep problem is the 'tired but wired' state in which the patient is exhausted but still can't get to sleep. He believes this problem is a by-product of the bodies attempt to increase blood flows to the brain through production of the stimulant adrenaline (epinephrine). Dr. Bell believes that this adrenaline production – which is appropriate when the CFS patient is upright – fails to turn off they lie down, and this prevents them from relaxing. He stated that benzodiazepines such Xanax, valium, etc. can helpful if used properly. (*Benzodiazapine - A class of compounds with antianxiety, hypnotic, anticonvulsant, and skeletal muscle relaxant properties. Stedman's Med. Dictionary).* He noted that while the over-the-counter medication Benadryl cannot be used continuously it can be used once in a while to go to sleep.

Some CFS patients have the opposite problem, hypersomnia, or excessive sleep. Dr. Bell stated that stimulants such as Ritalin, if used cautiously, can be quite helpful. *A poster in the professional conference will suggest the modafinil can work as well.* Proper sleep behavior or 'sleep hygiene' can be very helpful. This involves going to bed and waking up at certain times, not lying in bed before bedtime, and not lying in bed if you can't get to sleep.

Questions: One patient asked about waking up early in the morning and not being able to get back to sleep.

Dr. Bell suggested that some people don't need as much sleep as they think and that the person consider whether they are one of those people.

Q. One patient said that after going into a hot tub she woke up well rested for the first time in years and asked why.

Dr. Bell noted that a hot shower dilates the blood vessels in the legs and this draws blood down from the brain causing drowsiness and thus can be helpful with sleep for some CFS patients. He believes the alcohol intolerance in CFS is due to the same process.

This discussion got Dr. Bell into a short discourse on low blood volume, a subject he has great interest in. Eighty percent of the patients in Dr. Bell's practice are about a quart low on blood – a rather significant finding given that losing 1 ½ quarts of blood in an auto accident can leave you near death. He doesn't know why blood volume is low, would love to find out but emphatically stated it is not due to altered red blood cell shape. Just as with sleep he has medications he can use to increase blood volume but it does not help with CFS. He did note the intriguing case of patients improving on IV saline but does not recommend it. *We will see a poster on IV saline treatment and a fascinating presentation on a low blood volume study in the professional conference.*

Kenny De Meirleir, MD, PhD - Integrative & Complementary Medicine in CFS

Dr. De Meirleir gave an overview of his RNase L/PKR theory of cellular dysregulation in CFS. I was able to talk with Dr. De Meirleir several times – he is a delight to talk with, with an impish sense of humor. At one point he joked that he was getting bored with CFS, that he'd figured it out and was looking for another disease to study. His theory is quite complex but is well worth studying as it is almost uncanny how many aspects of CFS it has come to incorporate over time.

Dr. De Meirleir's major problem is getting funding. He said he could whip out several clinical studies a year, but doing basic research was expensive. He felt he could make major progress in this disease very quickly if he had the funding available to him. He has recently moved to the U.S. in an attempt to drum up more funding. I believe he will be associated with Reno Research Center. For the time being, legal restrictions prevent him from practicing in the U.S.

(Click here to get extensive overviews of this intriguing model of CFS pathophysiology.) In this talk he extended the RNase L model to two areas that have not really been highlighted before; the ability of nitric oxide upregulation to cause muscle weakness by binding to the ryanodine receptors on muscles, and the ability of PKR upregulation to decrease CRH hormone production and, in turn, cause the low cortisol levels we see in CFS.

Dr. De Meirleir has found that CFS patients have higher than normal reactivities to heavy metals particularly nickel. When their lymphocytes are exposed to these substances they proliferate like mad, a reaction that suggests that these substances could be at in part the source of the immune activation in some CFS patients. High heavy metal loads can be treated using IV drips of EDTA, DMSA, HMP and antioxidants (?). He said that because long term DMSA causes kidney damage he prefers using chelators such as glutathione and herbs.

Dr. De Meirleir is focusing heavily on the gut now. He believes there is an underlying weakness in gastrointestinal functioning that predisposes CFS patients to their disease. He has found high levels of antibodies (IgA, IgM) to harmful bacteria in the blood of CFS patients. This indicates that these bacteria have penetrated the lining of the gut and made their way into the blood. The antibodies indicate that B-cells in the immune system have been fighting them off.

Later on in the conference he will note that 70% of the immune surfaces in the body are found in the gut. He believes these harmful bacteria initiate an immune response that can explain many problems in CFS. Initially bacterial proteins called lipopolysaccharides (LPS) activate the toll-like receptors on cells. These receptors then trigger the PKR enzyme to produce prostaglandin (PGE2) and activate the inducible nitric oxide enzyme to produce nitric oxide. The PGE2 prostaglandin causes inflammation, peripheral vasoconstriction (low blood supply to the tissues) and increased blood viscosity. Increased nitric oxide levels cause impaired memory, low NK cell activity, low neutrophil levels, herpes virus reactivation, slowed gastric emptying and low blood pressure. *Note that the probiotics Dr. De Meirleir uses are designed to introduce 'good' bacteria into the gut to replace the harmful.*

I asked him how the process of RNase L dysfunction gets started. He believes that high amounts of cell suicide (apoptosis) allow cellular debris to essentially overwhelm the body's clean-up mechanisims. (Several studies have, in fact, found increased rates of apoptosis in CFS patients and we will see more evidence of its importance in CFS in the professional conference.)

Over evolutionary time a great deal of retroviral DNA has become inserted in our DNA and Dr. De Meirleir believes that apoptotic activity releases these fragments. These are very small nucleotide chains that prompt the 2-5OAS enzyme to produce a peculiar type of 2-5A which binds to the RNase L enzyme but leaves it vulnerable to attack. If this occurs when inflammatory enzymes such as elastase are present they will break RNase L into fragments some of which are 6-10x's more active than normal.

RNase L's job is to cut up RNA and Dr. De Meirleir believes one RNase L fragment cuts up enough human RNA to keep immune cells from functioning properly. One fragment that appears able to affect the ion channels that transport substances in and out of the cells could cause muscular problems, pain sensitization, and immune dysfunction, and also impair heavy metal elimination and glutathione homeostasis. When he put one RNase L fragment into healthy cells he found that they died at lower levels of mercury exposure.

Some proteins produced during this process target cellular thyroid receptors for destruction which could cause the metabolic problems and weight gain seen in some CFS patients. Activation of the PKR system from infections, heavy metals or other processes could cause inflammation, Th2 dominance, nitric oxide upregulation (70% of CFS patients) and increased apoptosis.

Treatment -Dr. De Meirleir notes there are a lot of subgroups in CFS. His general treatment regime focuses on restoring immune competence, the Th1/Th2 ratio, hormonal balance and intestinal flora, and in treating metal allergy. He often uses a short course of antibiotics (1 week) followed by three months of probiotics. He also uses digestive enzymes, omega-3, vitamin C and B12 and others (lipoceutical glutathione). On average, this results in a 74% decrease in levels of the pro-inflammatory enzyme elastase. Almost 60% of CFS patients experience improvement on the regime.

Q. In general how confident are you that you can help a CFS patient?

De Meirleir – If they're under 30, about 80% have acceptable results. If they're over 50, then results are usually poorer. It depends on how much damage has occurred. He said CFS physicians were still missing important factors in this disease.

What's New in CFS & FM Science, Treatment and Demographics

Introduction: **Nancy Klimas, MD**, Moderator: **Eleanor Hanna**, Panel: **Dharam V. Ablashi, DVM, MS, Dip. Bact., Kenny De Meirleir, MD, PhD, Birgitta Evengård, MD, PhD, Leonard A. Jason, PhD, Hirohiko Kuratsune, MD, DMedSci. & Gudrun Lange, PhD**

Dr. Klimas had high praise for Dr. Hanna of the Office for Research Into Women's Health (ORWH) in the NIH, calling her a hard worker endowed with passion for her work, and a real hero for the CFS movement. Dr. Hanna led this discussion. Throughout it she pitched the need for innovative, multi-disciplinary research proposals from CFS researchers to the NIH.

Dr. Kuratsune laid out the very intriguing state of CFS research in Japan. Chronic fatigue research in Japan is booming. The Japanese recognize that chronic fatigue in their population is a real threat not only medically but economically and the Japanese government has initiated a large research program to uncover the causes of chronic fatigue and to uncover treatments. The Japanese government has gone so far as to require that anyone who works over a certain number of hours a month be checked by an 'industrial physician' for signs of chronic fatigue. They estimate that a third of Japanese workers suffer from some form of chronic fatigue and that the problem costs the economy over $10 billion dollars a year.

Kuratsune's research team has marched down a lot of false trails but believes they have found a biomarker for CFS using spectroscopic analysis of serum samples. Dr. Kuratsune was unable to respond to Rich Van Konynenburg question regarding which proteins the analysis revealed because of patent issues, but he seemed quite excited about the finding. He is presently trying to develop an international collaboration to develop the test for world-wide use.

Dr. Gudrun Lange – stated that fMRI studies show that CFS patients can generally function fairly well but, to do so, have to use different parts of their brains from those healthy people use. In general patients' brains appear to be functioning in ways similar to those of people who are much older. Its unclear right now if there is a dysfunction in just one part of the brain or if it's systemic; CFS patients show signs of both kinds of dysfunction.

Dr. Kenny De Meirleir – believes that most problems in CFS originate in the gut and then branch out from there. Approximately 70% of the immune system surface in the body is found in the gut – a system that covers about the size of a football field. Eighty percent of CFS patients exhibit maldigestion of certain substances. Regular findings of antibodies to intestinal bacteria in the blood indicate that disturbed barrier functioning in the gut has allowed intestinal bacteria to make their way into the blood. CFS patients also exhibit increased uptake of heavy metals with almost 60% of them having an allergy to nickel, an immune system inducer.

Dr. De Meirleir believes predisposing factors are present in CFS but that the triggering factor is often an infectious agent.

Dr. Hanna asked about the efficacy of SSRI's given the large number of serotonin receptors in the gut. Dr. De Meirleir replied that some CFS patients improve on them, some don't, and some get worse.

Dr. Leonard Jason – noted how important the IACFS was becoming as an organization. He felt it had been revitalized in recent years and will provide a strong impetus for research. The IACFS developed a Pediatric Definition that indicates that kids with CFS have somewhat different symptoms than do adults. He noted that since 4-5% if the population has six or more months of fatigue, it is critical that researchers be able to develop homogenous groups to sample. He also

noted that the escalation of the war in Iraq will deprive medical researchers of much needed funds.

Dr. Daram Ablashi – is trying via the HHV-6 Foundation to assess the prevalence of HHV-6A in multiple sclerosis and CFS. They have not been able thus far to find antibodies that differentiate HHV-6A and B. The Foundation has looked at a lot of antiviral compounds but most have not worked.

QUESTION PERIOD

Q. Rich Von Konynenburg asked if glutathione had been effective in treating HHV-6. (The HHV-6 Foundation has been testing numerous substances against HHV-6 in test tubes. Early tests suggested that lipoceutical glutathione might be quite effective)

Dr. Ablashi – stated that while lipoceutical glutathione worked on some tests, it did not appear to affect HHV-6 in cells.

Q. What is the current thinking on Epstein-Barr Virus?

Dr. Klimas – EBV reactivation is possible in CFS. Dr. Glaser believes that EBV may be giving the cell DNA that is gumming up the works.

Q. CFS research generates about 300 papers a year vs. about 15,000 on breast cancer. What's going on here? (This question generated applause.)

Dr. Hanna – stated that there is a great deal known about breast cancer and that it's been studied extensively for years. CFS will not get large amounts of funding until there is a critical mass of basic science to build on that will help CFS researchers put the pieces together.

Dr. Klimas – noted how frustrating this problem was. She said CFS vitally needed to have more scientists interested in studying it.

(The breast cancer analogy was an interesting. Dr. Pinn has stated that breast cancer research funding was very low when she became head of the ORWH in the early 1990s. One wonders if one of CFS's sister diseases, fibromyalgia, is reaching that critical mass as we're seeing a lot of fibromyalgia studies now. *A poster in the conference will speak to this.* If this is true it demonstrates that the tide can turn even for controversial diseases.)

Q. A representative of the Great Plains Laboratory asked Dr. De Meirleir to comment on the fact that they are seeing some similar markers in autism and CFS, FM and multiple sclerosis.

Dr. De Meirleir – Mothers with CFS have 3-4 times perhaps even 10 times more chance of having autistic children. He's collecting data on this now.

Dr. Hanna closed the session by imploring CFS researchers to be creative in looking for funding right now. She noted that there's a lot of money going into autism research right now and creative CFS researchers could tap into it. When I talked with her later she said it's very possible to have a 'CFS study' that doesn't mention CFS in the title but has a CFS patient control group. She appeared somewhat frustrated that some CFS researchers felt they had to have CFS in the title of their paper.

The P.A.N.D.O.R.A. Sand Castles Awards

Next we had the dinner and during it the Sand Castle Awards and the Keynote Address. The winners of the Sand Castle Awards were

Life Time Achievement: Marc Iverson
Outstanding Male Advocate of the Year – John Herd
Outstanding Female Advocate of the Year – Pat Fero
Pioneer Spirit Awards – Paul Cheney, Nancy Klimas, David Bell, Dan Peterson
Author of the Year – Dorothy Wall – '*Encounters with the Invisible'*
Founder's Award – Rebecca Artman

Dorothy Wall was unable to attend the conference but did provide an address via video. For an interview with this stimulating author **click here**.

KEYNOTE ADDRESS - DR. PAUL CHENEY – The State of Art of CFS

Dr. Cheney's address was both exasperating and exciting. After a long conference day and a full meal, patients were hardly ready to take in a lot of technical information but they sure got it. Around my table I saw eyes rolling and people getting up and heading for the door. I had trouble keeping my eyes open as well but every now and then, as he always does, Dr. Cheney would drop in something absolutely riveting. Because I was unable to keep up with Dr. Cheney's rapid fire presentation parts of it will be missing. My comments are in italics.

CFS Patients Are Functionally Hypoxic - Dr. Cheney started off in characteristically dramatic fashion showing a slide of Mt. Everest (29,028 ft.) with 1/3rd of the oxygen at sea level and announcing that the problems CFS patients face are very similar to those that climbers at the top of Mt. Everest face.

Hypoxia: Decrease below normal levels of oxygen in inspired gases, arterial blood, or tissue (Stedman's Medical Dictionary)

Next he jumped to picture of a fetus noting that a fetus gestates at an effective altitude of 29,000 ft. because it does not have the physiology to handle oxygen. Although oxygen plays a central role in the aerobic energy production process it is not a benign substance. Oxygen is delivered into the mitochondria of cells by red blood cells where it functions as an electron carrier in the electron transport chain. As oxygen becomes charged it turns into a free radical called superoxide which can, if it is not quickly degraded, be turned into two free radicals: peroxynitrite or the hydroxyl radical. Superoxide degradation is so critical that it constitutes the most rapid enzymatic reaction that takes place in the body. Energy production is synonymous with free radical production.

Dr. Cheney believes an injury to the antioxidant system that degrades superoxide is the central facet of CFS and that the 'body' down-regulates ATP production in order to spare it from free radical injury. *Although the outward manifestations of Dr. Cheney's theory have changed dramatically, this aspect has not – it has been a central part of his theory for many years. His explorations with whey protein were an attempt to boost the antioxidant properties of the cell.*

Early in their development fetuses grow under hypoxic conditions but later on grow under normal oxygen levels. The foramen ovale allows oxygen rich maternal blood to bypass the lungs as it travels from the right to the left side of the fetuses heart. The foramen ovale is closed at birth by increased blood pressure on the left side of the heart.

Energy Production and the Heart – Dr. Cheney noted that most of the energy consumed by the heart occurs not when it pumps out the blood during the systolic phase but during the relaxation or diastolic phase in which it (the left ventricle) fills up with blood. This means that if someone has an energy deficit – as he believes CFS patients do – they're not going to have a problem with pumping the blood out but with drawing blood into the heart. The filling phase has two parts; first the mitral valve opens to allow blood in – this allows some filling but the heart needs more. In the second phase, a contraction forces more blood into the ventricle.

A Personal Tragedy Results in New Insights – Dr. Cheney then shared how his own experience with heart failure contributed to his new look at CFS. As he began to recover from his heart transplant Dr. Cheney felt he was recapitulating many of the symptoms that his CFS patients had suffered from. It was then that someone handed him the just-published Peckerman paper which suggested CFS patients that cardiac problems in CFS were accentuated while they were lying down – a sign of diastolic dysfunction. *Even though their cardiac output was higher when the CFS patients were lying down the difference between their output and that of the healthy controls was actually greater then. The weak heart is actually under more stress when lying down because it has to deal with more blood than when one is standing. Heart failure patients end up standing upright 24 hours a day in order to spare their heart.* Approximately 50% of the CFS patients in Dr. Peckerman's study had low cardiac output. Dr. Cheney noted that more of his patients did (@80%) probably because he had a more disabled patient population.

Dr. Cheney calls the heart problems in CFS "CFS Associated Cardiomyopathy" or "CAD". *In an earlier talk Dr. Cheney accentuated that CFS patients were in "heart failure".* Here he noted that 25% of heart failure patients die within a year but that none of his CFS patients had ever reached that point. *There is a difference between diastolic dysfunction and diastolic heart failure. In diastolic dysfunction the heart is behaving improperly but it has not reached the point at which the heart muscle has entered a precipitous decline. One researcher has postulated that diastolic heart failure is reached when the patient is exhibiting symptoms of blood backing up into the lungs (pulmonary congestion). Since this is not usually observed in CFS patients, it seems that diastolic dysfunction is a more appropriate term for the process occurring there. As I remember this is the term Dr. Cheney usually used.*

One of the questions regarding Dr. Cheney's theory of heart failure in CFS has been that CFS patients rarely exhibit the key sign of heart failure, namely "shortness of breath". Dr. Cheney explained this conundrum by saying that diastolic heart failure patients more commonly evidence orthostatic intolerance – a condition often found in CFS – than shortness of breath. *Dr. Cheney's slide of typical heart failure signs/symptoms (fatigue, exercise intolerance and others) did not include several other symptoms also commonly found with heart failure, including morning edema in the ankles, distinctive heart and lung sounds and heart enlargement. All the websites from major medical organizations still associate diastolic heart failure with shortness of breath upon exertion. One site furnished findings from a study that indicated that shortness of breath was more commonly found in diastolic than in systolic heart failure. Perhaps Dr. Cheney has information that they do not.*

Dr. Cheney then introduced an intriguing slide showing that diastolic heart failure rates among women had risen dramatically since about the mid 1980s – or about the time CFS showed up on the scene. He said that researchers had been unable to account for this rise. *He didn't explain why he showed this slide but it suggested he thought that there was a commonality between the two.*

Dr. Cheney then went through a variety of different cardiac findings in CFS and concluded that most CFS patients display a pattern of blood flow through the heart called 'pseudonormalization.' which is indicative of an intermediate case of diastolic dysfunction. He noted that almost all CFS patients had at least one abnormal reading in four different tests of blood flow. He then introduced an interesting table that looked at population-wide averages of diastolic dysfunction in older Americans.

Opinions differ on what constitutes diastolic dysfunction (DD). Several of the tests of diastolic dysfunction, including some but not all used by Dr. Cheney, have not been found to correlate well with fitness or with more stringent measures of cardiac functioning and this has lead some to question their importance. This is presumably why Dr. Cheney introduced what he called a conservative measure of diastolic dysfunction created by the Mayo Clinic and then compared how many CFS patients versus older patients in the general population met the criteria for diastolic dysfunction. *Diastolic dysfunction has been believed to be primarily an older person's disease. Intriguingly it is also primarily a women's disease.* This was, I thought, one of the more convincing arguments for its association with CFS. Unfortunately my notes are not clear as to percentages but while a subset of CFS patients did not meet the Mayo criteria for DD, most of his patients met the criteria for either mild or intermediate DD.

As was noted earlier most CFS patients have a pseudonormalized pattern of blood flow. This would indicate that the left ventricle has stiffened sufficiently to prevent it from filling properly during the second (a) phase of the diastolic pulse. My reading of medical papers indicates that this phase is usually accompanied by hypertrophy of the heart and shortened breath during exertion – two problems Dr. Cheney did not say were present in his patients. Patients with this condition should also demonstrate abnormalities during the Valsalva maneuver – again something studies have not found in CFS. The diastolic dysfunction in CFS appears to be different than normally found?

Functional hypoxia – A Key Aspect of CFS – Dr. Cheney then popped open eyes that had begun to glaze over by showing, this time using a hyperbaric chamber, that if you put young men in an environment with really low oxygen levels their hearts begin to look very much like those of CFS patients (going to back to the reference to theHimalaya's). He went through this pretty quickly but the confluence in test results between the two was striking. This suggested that heart problems in CFS may be due to a low oxygen environment.

Patent Foramen –Dr. Cheney reported that his CFS patients exhibit very high rates (90%) of an abnormality called a 'patent foramen'. Remember the valve (patent ovale) that is closed in a fetus once it is born? The patent foramen (ovale) (foramen = hole) is what that valve is called if it opens in adults (= becomes patent).

Every time the patent foramen opens it allows some CO2/O2 to escape into an artery. Some of these gases may find their way into the brain and Dr. Cheney indicated that patent foramens may be implicated in stroke and migraine. But what is causing this problem in CFS? Dr. Cheney showed that simply giving CFS patients oxygen through the nose caused the patent foramen to close off in a significant subset of his patients. He concluded that the patent foramen and the other cardiac problems in CFS are due to an 'oxygen dependent diastolic dysfunction' and that this is a hallmark of CFS.

Patent foramen (ovale) – the patent foramen ovales may be more prevalent in CFS but is it in itself a significant medical problem? This is another grey area in medicine. Approximately 25% of the population has one and physicians differ as to their significance. Patent foramens differ in severity; some open only when one is lifting something or coughing, others are open more

frequently. Because they can be induced some believe physicians are finding more of them simply because they are looking harder for them. Recent studies suggest they are not implicated in stroke but this is still a grey area. There is e is disagreement about how aggressively they should be treated or, indeed, if they should be treated at all. Patent foramen ovales are not associated with any symptoms. See the professional conference for Dr. Cheney's theory on why they are so commonly present in CFS.

Treating a CFIDS Associated Cardiomyopathy (CAD) - We now come full circle to Dr. Cheney's thesis that a functional hypoxia is central in CFS. *This is not your normal garden variety of hypoxia – CFS patients don't appear to have even mild systemic hypoxia.* Dr. Cheney again stated that he believed the problem lay in the inability of the antioxidant enzyme system (glutathione peroxidase/catalase) to degrade the free radicals produced during mitochondrial activity. He noted that some viruses can inhibit these enzymes and that the glutathione peroxidase enzyme that produces the master detoxifier glutathione may be particularly vulnerable to mitochondrial problems because it requires a lot of energy. Now Dr. Cheney came to the treatment.

Here the address almost came to a halt as Dr. Cheney was informed he had used up his time! The patient outcry, of course, was rather intense as they saw their reward for sitting through a highly technical lecture after a really full day about to be dashed, and Dr. Cheney was allowed to finish.

The detoxifying enzymes (catalase, glutathione peroxidase) are produced by the liver and Dr. Cheney's strategy is to boost their levels by boosting liver production. He is attempting to do this by using, if my notes are correct, adolescent porcine liver extract ???, hawthorn and low amounts of human growth hormone to jumpstart the process. He said that at 90 days he saw about 30% improvement in one common diastolic abnormality (IVRT).

This was a fascinating lecture. It is still unclear how important diastolic dysfunction is to the pathophysiology of CFS, i.e. whether it is a primary or secondary feature, but Dr. Cheney is presenting evidence that it is present and should be investigated further. What was most interesting to me as a layman were the connections between Dr. Cheney's findings and others presented at the conference. These connections will be explored further in the upcoming Professional Conference overview.