

Reversal of a Case of Advanced Coronary Artery Disease with Unstable Angina Using Pulsed Electromagnetic Field (PEMF) Cellular Exercise *by Martin Milner, ND*

It is wonderful to both the patient and physician when, after years of failed trials in both conventional and alternative medicine, a safe, natural method of cellular exercise makes dramatic change in a case of serious chronic disease. This case is an extraordinary example of reversing end-stage coronary artery disease with pulsed electromagnetic field cellular exercise (PEMF). The case also elucidates critical monitoring and decision-making horizons throughout patient management.

The Case

SH, a 65-year-old, very pleasant white Caucasian female, presented to our clinic with advanced coronary artery disease, diabetes, hypertension, and obesity. Her cardiac history began in 1996, when she went into cardiac arrest and was successfully defibrillated and brought back to life. She did lose sensation in two of her toes at discharge from this hospitalization. This loss of sensation was presumed to be a complication of chest defibrillation. During this hospitalization, significant ischemic heart disease was diagnosed on cardiac catheterization, and two stents were deployed into the left anterior descending and right circumflex coronary artery.

Progression to Advanced Coronary Artery Disease

As time progressed, her disease advanced, and a second angiogram involved the deployment of a third stent in her left anterior descending coronary artery. Her ischemic heart disease progressed further, and in 2005 she underwent three vessel coronary artery bypass graph surgery where the LAD stents were bypassed along with bypass surgery of the left circumflex and bypassing a new occlusion in the right anterior descending coronary artery. At the time of this

hospitalization, she was diagnosed with non-insulin- dependent diabetes mellitus and hypertension. Her diabetes advanced, and she became insulin dependent in 2008.

Cardiac Selective Beta 1 Blocker Affecting Asthma

SH began seeing me in January 2006. At that time, her biggest concern was to be able to reduce her prescription drug load. She had a history of asthma and was being prescribed atenolol for hypertension. Even though atenolol is a cardiac-selective beta 1 blocker, it may aggravate asthma in sensitive patients. Recall that beta 1 cardiac-specific blockers do not affect beta 2 receptors and in general do not exacerbate asthma, unlike beta nonselective blocking drugs such as propranolol. The uncommon effect of beta 1 blockers' aggravating asthma may be due to their tendency to reduce sympathetic tone overall beyond the heart causing bronchial constriction.

Drug Side Effects and Gradual Weaning of Atenolol

Additional side effects that she was experiencing included leg cramps from Lipitor and a dry cough from lisinopril. She was taking bioidentical hormones via her obstetrician. Through 2006 she was very gradually weaned off Atenolol over a six-month period. She experienced great difficulty getting off the last 6.5 mg, with rebounding rapid heart rate. It is important to always wean any cardiac arrhythmic or coronary artery disease patients off beta blockers gradually. The rebounding tachyarrhythmia induced by too-abrupt weaning can be life threatening. Once we successfully brought her down to 6.25 mg, her asthmatic breathing resolved. Apparently, in this patient, the cardiac specificity of Atenolol crossed over and somehow affected beta 2 adrenal receptors in the bronchial tubes, aggravating her asthma.

Modifiable Metabolic Markers of Heart Disease

Our initial work-up included a comprehensive profile of modifiable metabolic markers of heart disease, including a lipid panel with lipoprotein A and lipid fractionation, homocysteine, CRP-HS, fibrinogen, and bleeding time. Her homocysteine had not been formerly measured and was 11.4 in January 2006, reduced to less than

6 since June 2006 with routine homocysteine-lowering B vitamin therapy, including B6, B12, and folic acid.

Advanced Coronary Artery Disease Progressing to Congestive Heart Failure, then Remitting

This first year of management also focused on better control of her hypertension. However, she developed progressive unstable anginal chest pain at rest. An April 2007 angiogram reveals severe obstructive disease involving a nonrevascularized diagonal branch of the LAD with the native bypass graft unchanged. A high-grade distal lesion at LAD evolved into worsening chest pain.

Continuous Nitrate Prescribing Adjustments from Arginine to Isosorbide

During SH's enhanced external counterpulsation (EECP) treatment, the referring cardiologist in collaboration with our office adjusted the nitroglycerine management. I had initiated the prescribing of arginine 900 mg, 2 t.i.d. with gamma-tocopherol 200 IU, b.i.d. in March 2007. This was unsuccessful in controlling chest pain and was discontinued in September 2007. Concurrent nitrate therapy was added with isosorbide dinitrate ER, 40 mg q.d. in April 2007 to further aid in the management of her ischemia and unstable chest pain at rest. This was increased to 40 mg b.i.d. and ultimately one every six hours after arginine was discontinued in September 2007 to adequately control her progressing unstable chest pain at rest. At this point, SH was completely disabled, with unstable chest pain at rest with no activity.

Most ratings of coronary artery disease disability follow the following table from the New York Heart Association.

New York Heart Association (NYHA) Cardiac Disability Rating Scale:

I = no symptoms

II = symptoms with ordinary activities of daily living

III = symptoms with less than ordinary activity

IV = symptoms at rest

End Stage Coronary Artery Disease

It doesn't get any more disabling than persistent unstable angina at rest. Having failed EECB and progressing to unstable angina with extensive prior CABG (coronary artery bypass graft) and stent deployments, conventional as well as alternative medicine interventions seemed to be used up. Although intravenous chelation was discussed, I questioned its ability to improve end stage disease and suggested we begin a trial of pulsed electromagnetic field (PEMF) cellular exercise.

Living Cells Are Direct Current Systems – Treating the Electrical Cause of Disease

Our living cells are electrical direct current (DC) systems. In fact, all life generates an electrical DC charge. This natural charge is created by the movement of ions in and out of cell membranes, creating and maintaining a membrane charge of approximately 70 mV. Any challenge to the cell, such as oxygen/ nutrient deficiency, toxicity, tissue changes, or inflammation alters ion movement, and the charge on the cell membrane changes. This altered charge profoundly affects metabolic nutrition into and waste products out of the cell.

Pulsed Electromagnetic Field Cellular Exercise

PEMF takes alternating current (AC) and transforms it into DC, producing variations ranging from low to high voltage. This voltage is passed through a coil, generating a safe, pulsed magnetic field around the coil. As the magnetic field is pulsed on, electrons are excited, and cells exposed to the magnetic field are exercised and expanded. The electrically charged cell membrane is gently pulled by the pulsing magnetic field, and the matter as well as the space around matter is recharged. On the off phase of the pulse, the cells relax. This is profoundly beneficial cellular exercise and cellular rehabilitation. As cells expand and relax, they rehabilitate, ion movement improves, and the membranes' electrical charge begins to return to optimal. As cells recharge themselves, they heal and return to optimal function. High-intensity PEMF is not a medical device in the US. It simply produces a pulsed magnetic field of varying strengths functioning as a cellular

exerciser. It is not intended for the treatment, diagnosis, or prevention of any disease or condition.

Recovery of Toe Paresthesia After One Session

We started using PEMF with SH in June 2008. After the first session of approximately 10 minutes over her chest and heart, she fully regained the sensation in the two toes of her left foot that had permanently lost sensation for the last 12 years. This lack of sensation has not returned as of this writing (February 2010). While this may sound miraculous, it actually makes sense, since it was hypothesized that the defibrillation during her cardiac arrest induced the nerve damage. PEMF could very well recover that nerve damage. Nerve and heart cells are both extraordinarily ionically sensitive cell structures and respond exquisitely to the cellular exercise of a pulsed magnetic field.

Remission of Unstable Angina at Rest

SH continued with PEMF session of 30 to 60 minutes two to three times a week. She became able to perform activities of daily living without chest pain after the first month of PEMF and was no longer experiencing chest pain at rest. Her isosorbide dose was lowered from three times daily back to twice daily. Her BNP dropped from a high of 699 to 126 by December 2008, confirming resolution of ischemic heart failure. **Partial Relapse Followed by Remission** She experienced a partial relapse with reduction of PEMF sessions from three times weekly to once weekly. However, upon purchasing her own machine in June 2009 and increasing the sessions to one to two hours daily, her ischemia improved further. She improved again to the point of never getting chest pain at rest or with mild activities of daily living. She was able to mildly exercise without chest pain, and her BNP was low at 134 as of July 2009.

Toward the end of December 2009 and thereafter, the patient upgraded her PEMF machine with enhancement in its pulse pattern. There were no other changes in her health-care regime. A BNP in Feb. 2010 came back very low at 63, indicative of further improvement in heart failure from the new pulse upgrade to the PEMF machine. She

remains well as of this writing (February 2010) with no unstable chest pain episodes at rest.

Brain Natriuretic Peptide (BNP) as a Marker of CHF

SH developed congestive heart failure in April 2007 due to advanced coronary artery disease verified with a mild elevated BNP of 342 pg/mL. BNP levels are the best blood marker for heart failure. For patients with heart failure, BNP values will generally be above 100 pg/mL. A BNP above 100 pg/mL has a sensitivity of 90% and specificity of 76% for heart failure. A more conservative interpretation of the BNP is a normal value less than 50 pg/mL with a diagnostic “gray area” between 100 and 500 pg/mL, for which the test may be considered inconclusive. Values above 500 pg/mL are generally considered to be positive for heart failure. This gray zone has been addressed in several studies referenced below. It is best to combine BNP findings with clinical history, physical signs, and symptoms of heart failure along with periodic echocardiogram findings to aid in stratifying diagnostic severity. We were able to pull her out of heart failure for the entire second half of 2007 with levels well below 100 pg/mL.

Date	BNP pg/mL
May 2007	315
June 2007	699 Definitive ischemic CHF (congestive heart failure)
July 2007	319
Aug. 2007	44
Oct. 2007	61
Dec. 2007	31

BNP levels were brought to a low of 31 pg/mL in December 2007 using bed rest (essential in unstable heart failure), an array of nutritional support, no or minimal Atenolol, and a referral for concurrent 45, one-hour, EECP treatments from July 2007 through January 2008. While SH was better during the EECP sessions, she quickly relapsed after completion of the 45th session, and just three months later she developed a recurrence of heart failure in April 2008 with a BNP of 291 pg/mL.

Date	BNP pg/mL
April 2008	291 relapse a few months after EECF
Oct. 2008	233 PEMF started 1-3 times weekly July 2008
Dec. 2008	126
Apr 2009	180
Jul 2009	134 PEMF sessions increased to daily in June 2009
Oct. 2009	250
Nov. 2009	229
Feb. 2010	63 out of heart failure after one month of PEMF with upgraded technology

Conclusion

A review of the electrophysiological effects of pulsed electromagnetic fields and the presentation of this case compel the reader to consider PEMF as one of greatest hidden breakthrough technologies of cellular exercise and cellular rehabilitation of the last century. Over 5,000 studies have been published worldwide over the last 40 years. It definitely warrants further investigation.

Full Disclaimer

Pulsed electromagnetic fields (PEMF) are generators that produce a DC pulsed electromagnetic field of varying strengths delivered via insulated cables. No current of any kind comes into contact with the body. The magnetic field energy produced at the cable passes freely through living tissue for the purpose of cellular exercise to promote and support a sense of well-being. It is not a medical device. It has not been evaluated by the FDA. It is not intended for the diagnosis, treatment, or cure of any physical or medical condition. If you are experiencing the symptoms of a physical or medical condition, you should seek the advice of a medical professional before using PEMF as a form of cellular exercise.

References

- Atisha D, Bhalla MA, Morrison LK, et al. A prospective study in search of an optimal B-natriuretic peptide level to screen patients for cardiac dysfunction. *Am Heart J.* September 2004;148 (3): 518–523.
- Brenden CK, Hollander JE, Guss D, et al. Gray zone BNP levels in heart failure patients in the emergency department: results from the Rapid Emergency Department Heart Failure Outpatient Trial (REDHOT) multicenter study. *Am Heart J.* May 2006;151(5):1006–1011.
- Bibbins-Domingo K, Gupta R, Na B, Wu AH, Schiller NB, Whooley MA. N-terminal fragment of the prohormone brain-type natriuretic peptide (NT-proBNP), cardiovascular events, and mortality in patients with stable coronary heart disease. *JAMA.* January 2007;297(2):169–176.
- Fitzgerald RL, Cremo R, Gardetto N, et al. Effect of nesiritide in combination with standard therapy on serum concentrations of natriuretic peptides in patients admitted for decompensated congestive heart failure. *Am Heart J.* September 2005;150(3):471–477.
- Jerabek J, Pawluk W. *Magnetic Therapy in Eastern Europe: A Review of 30 Years of Research.* Rancocas, NJ: W. Pawluk; 1999.
- Malmivuo J, Plonsey R. *Bioelectromagnetism: Principles and Applications of Bioelectric and Biomagnetic Fields.* Oxford University Press; 1995.
- Strunk A, Bhalla V, Clopton P, et al. Impact of the history of congestive heart failure on the utility of B-type natriuretic peptide in the emergency diagnosis of heart failure: results from the Breathing Not Properly Multinational Study. *Am J Med.* January 2006;119(1):69.e1–11.
- www.BioMagneticRelief.com – link to worldwide research

Dr. Milner received his ND from National College of Natural Medicine in 1983. He has remained at his alma mater for the past 23 years as professor of cardiovascular and pulmonary medicine. Dr. Milner is also the CEO and medical director of the Center for Natural Medicine Inc. (1983–present) in Portland, Oregon. CNM is in its 11th year providing a clinical training ground for naturopathic medical student interns and resident doctors, supervised by Dr. Milner. He is well published in endocrinology, cardiology, pulmonology, oncology, and environmental medicine. Dr. Milner cofounded BioMagnetic Relief LLC, dedicated to researching pulsed electromagnetic field as a form of cellular exercise.

1330 SE 39th Avenue
Portland, Oregon 97214
503-232-1100
drmilner@biomagneticrelief.com
www.biomagneticrelief.com