TFT and Heart Rate Variability Claims Demystified

I came to realize that many people choose scientific beliefs the same way they choose to be Methodists, Democrats, or Chicago Cubs fans. They judge science by how well it agrees with the way they want the world to be.

--Robert Park, Voodoo Science

For more information on Heart Rate Variability read: Herbert, J. D., & Gaudiano, B. A. (2001,October) "The search for the Holy Grail: Heart rate variability and Thought Field Therapy." Journal of Clinical Psychology.

Recently, Callahan reported that he has observed positive changes in the autonomic nervous system due to TFT treatment as measured by heart rate variability (HRV). HRV is a noninvasive test of cardiovascular autonomic regulation. Specifically, HRV is a measurement of the interaction between sympathetic (i.e., "fight or flight" energy mobilization) and parasympathetic (i.e., the opposite of the sympathetic activity or "relaxation" response) activity in autonomic functioning (i.e., the nervous system that controls the heart, intestines, and other organs). There are two main HRV approaches: time domain analysis and frequency domain analysis. One common measure in time domain analysis of HRV is SDNN (i.e., standard deviation of normal to normal intervals). Frequency domain analysis, also called power spectrum density (PSD), produces high frequency (parasympathetic activity) and low frequency (sympathetic activity) power values; a total power (sympathetic/parasympathetic balance) can also be derived. In general, higher HRV is desirable; lower HRV has been found to be a significant predictor of cardiac mortality and morbidity (see Cohen, Matar, Kaplan, & Kotler, 1999 for a further explanation of HRV).

HRV is a promising yet complicated measurement that still has many unresolved issues. While the high frequency (HF) component is fairly well established as a measure of parasympathetic activity, there is still some controversy regarding how accurately the low frequency (LF) measurement signifies sympathetic activity. In addition, different measurements of HRV and data collection methods have lead to conflicting results and conclusions from researchers (Cohen et al. 1999). HRV measurements must be done in standardized conditions in order for the data to be reliable and valid. For example, HRV is influenced by many factors such as age, sex, position, breathing, smoking, hour of the day and medications. Sexual intercourse has also been shown to produce changes in HRV.

Inconsistent results in HRV have been found in depressives (Moser et al., 1998; Yeragani, Balon, Pohl, & Ramesh, 1995). Some researchers find that depressives have lower HRV than controls, while others have been unable to replicate this finding. The findings are more consistent for anxiety disorders and anxious patients usually demonstrate lower HRV when compared to controls (Cohen et al., 1998; Kawachi, Sparrow, Vokonas, & Weiss, 1995; Klein, Cnaani, Harel, Braun, & Ben-Haim, 1995). These results have been demonstrated in patients with panic disorder and posttraumatic stress disorder; however, it is not known whether HRV markers can differentiate between the anxiety disorders or if these results simply indicate increased stress and anxiety. None of these findings would be unexpected because an anxiety/stress reaction is physiologically

demonstrated in the "fight or flight" response, resulting in increased sympathetic and decreased parasympathetic activity.

Callahan & Callahan (2000; pp. 231-251) have presented uncontrolled case studies in which he claims that HRV was improved in patients following TFT treatment. However, it is unclear if standard measurement procedures were followed because details of the recording protocols are not provided. Controlled conditions are particularly important when calculating HRV because if such elementary variables as respiration rate are not standardized, results are invalid (Cohen, et al., 1997). For example, Pomeranz et al. (1985) state: "This HI-FR [HF] peak is affected by depth of breathing and varies in frequency with variations in respiratory rate. It constitutes a direct measure of the well-known respiratory sinus arrhythmia" (H152). Specifically, it is possible that simply slowing down breathing from the first reading to the second will result in a higher HF (parasympathetic activity) reading and subsequently higher HRV (Hirsch & Bishop, 1981). Therefore, what Callahan could simply be measuring is the fact that individuals are more relaxed after treatment than when the measurements were originally taken due perhaps to the passage of time (Kline, personal communication).

Callahan frequently asserts that HRV is not influenced by placebo. However, his understanding of placebo is overly simplistic. A placebo is simply an "inactive" treatment (Lohr, Lilienfeld, Tolin, & Herbert, 1999). For example, a sugar pill is a placebo because it is not known to have medicinal benefits. Why a placebo "works" is an entirely different question. It is reasonable to view tapping as a placebo because of a lack of empirical support at this time, meaning that it does not, in and of itself, produce reductions in psychological distress. However, the placebo effect can produce benefits with some individuals for a multitude of non-related reasons, only one of which involves suggestion. As mentioned, slowing respiration (which may not be related to tapping) could result in the "benefits" shown from TFT treatment.

Callahan often quotes from a study by Kleiger et al. (1991) to support his assertion that HRV is not susceptible to "placebo". This study investigated the properties of HRV measurements over time in normal subjects. Subjects were assessed at baseline and after being given a pill placebo. They found that HRV measurements after pill placebo were nearly identical to those taken at baseline. At face value, such findings would seem to substantiate Callahan's claims of the lack of a placebo effect on HRV. However, this conclusion is completely misguided and not applicable in the case of Callahan's use of HRV. The subjects in this study were normal controls who therefore had no medical or psychological problem for which they expected to be treated. In addition, because these subjects were controls in good health, they were not assumed to have the autonomic deregulation in the first place. Furthermore, the placebo pill was not claimed by the researchers to be beneficial in any way. It is a well established fact that the autonomic nervous system (ANS) can be influenced by the person and therefore in conjunction with a placebo (i.e., inactive) treatment (Ross & Buckalew, 1985). For example, Cowan, Kogan, Burr, Hendershot, and Buchanan (1990) found that cardiac patients could cognitively increase their HRV through biofeedback training. If the ANS is altered, this result will be shown in HRV measurements. Therefore, Callahan's claims about HRV are not substantiated by this study.

Unfortunately, Callahan has not conducted a proper study using HRV as an outcome measure comparing those treated with TFT to a control group. Such data is necessary before claims can be taken seriously; uncontrolled case studies are not sufficient to substantiate this claim.

In conclusion, the claims being made by Callahan regarding HRV are not backed up by credible data from controlled research. Callahan's statements about the immunity of HRV to "placebo" stem from a misguided and simplistic understanding of the placebo effect. Furthermore, demonstrating increased HRV following treatment does not necessarily provide evidence of the efficacy of tapping because other reasonable explanations are more likely. Of course, one might expect that a treatment that would be related to an improvement in anxiety symptomatology would be associated with increased HRV. For example, benefits in HRV have been found from psychotropic medications which correlate to symptom reduction (Roose et al., 1998; it should be noted that drugs with anticholinergic effects actually decrease HRV, while still being associated with a reduction in symptomatology). In other words, HRV data should be treated as a physiological indication of improvement, which should be one component of a broad, multi-domain assessment. Cohen et al. (1999) warn that HRV is still a controversial method for use in psychiatric populations and further research is needed to clarify what conclusions can be drawn from the data. Therefore, TFT's claims about HRV at this point seem to be overblown and not based on clear evidence.

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