SPECTRAL ANALYSIS OF BIOFEEDBACK INDUCED HEART RATE DECREASES IN SINUS TACHYCARDIA

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Self-control of heart rate has been one of the most extensively studied areas in biofeedback research. Overall, the impression nowadays is that when given exteroceptive feedback of cardiac activity, most subjects can gain control to some extent over their heart rate. In this Chapter, we will first give an outline of the role that respiratory and somatic factors have played in human heart rate feedback research. We will then discuss what impact this has had on the development of heart rate feedback as a tool in clinical practice, and more specifically, its application for sinus tachycardia. Furthermore, background information is given on symptoms, etiology, and conventional therapy of sinus tachycardia. Subsequently we will present some recent findings on application of heart rate feedback for this arrhythmia from our own laboratory. Lastly, we will show how the application of spectral analysis may contribute towards unravelling some of the neuro-physiological mechanisms involved in sinus tachycardia and its treatment with heart rate feedback.

RESPIRATORY AND SOMATIC FACTORS IN EXPERIMENTAL HEART RATE FEEDBACK

Of any single factor that has been suggested to be of influence for the efficacy of heart rate feedback training, somatic influences appear to be capable of producing the most significant heart rate changes. Within somatic mediation, respiratory influences have received far more attention than effects due to muscular changes (Williamson and Blanchard, 1979; McCanne and Sandman, 1976). This is understandable since it has been known for some time that there are sizable cyclic variations in heart rate that occur in phase with respiration: respiratory sinus arrhythmia (RSA). Moreover, it has been demonstrated several times that alterations in respiratory patterns can cause appreciable changes in heart rate (Davis and Neilson, 1967; Sroufe, 1971; Johns, 1970). In the emerging area of heart rate feedback, an important question to be answered was whether subjects could learn to control their heart rate directly, obviously underscoring the necessity to control for respiration. This often was done through some sort of pacing procedure, but instructed variations and correlational techniques were also applied (Holmes et al., 1980). The results of these studies with controlled procedures were that subjects could still acquire

* In fond memory of my mother.
control of heart rate, but with smaller effects as compared to free respiration. Obrist and his colleagues have convincingly demonstrated that the fewer the constraints on subjects, the better their ability to change heart rate (Obrist, 1976; Obrist, Galosy, Lawler, Gaebelein, Howard and Shanks, 1975). As constraints were added to respiration and skeletal muscle manoeuvres, smaller changes could be reached. However, even in the most restricted condition, subjects could still reliably show some control of heart rate. Furthermore, since the amount of control was of limited magnitude even in the free respiration condition, serious doubts were raised as to the practical usefulness of heart rate feedback per se. This was especially true for biofeedback assisted heart rate deceleration where 'overall' positive results have not been as numerous or of the same magnitude as with heart rate acceleration. This has brought about the bizarre situation of heart rate feedback being perhaps the most intensively researched biofeedback area in experimental psychology, in which evidence on parameters relevant to outcome of training continues to be built up, but without resulting in the development of significant clinical applications.

CLINICAL HEART RATE FEEDBACK: THE CASE OF SINUS TACHYCARDIA

This paradoxical situation probably could have been circumvented if in discussing the magnitude of treatment effects on heart rate, there had been greater consideration of the biological limits of the cardiovascular system, i.e., the range of its possible variations which contribute towards the healthy homeostatic functioning of the organism. The subjects in most of these feedback studies have been young (college age) and free of disease. Since the normal resting heart rate is usually about 70 beats per minute (bpm), the potential range of change to be expected for heart rate acceleration and deceleration would be on the order of 100 bpm and 15 bpm respectively (Cheatle and Weiss, 1982). Thus as for deceleration effects, the observed magnitude of effect should always be evaluated against this limited range in which decreases in heart rate can still be said to be biologically meaningful. Also, since clinical populations may have resting heart rates that may well differ from 70 bpm, no definite inferences on the therapeutic potential of heart rate feedback can be made from these studies with nonclinical populations.

This point becomes especially pungent if one considers the several reports that demonstrate the potential of learned deceleration of heart rate for sinus tachycardia (Engel and Bleecker, 1974; Scott, Blanchard, Edmundson and Young, 1973; Vaitl, 1975; Janssen, 1983). Sinus tachycardia refers to an abnormally increased heart rate in which the rhythm still generates from the sinoatrial node. Hence the electrocardiogram has normal QRS complexes, except that the heart rate is considerably higher than normal. Three independent general causes of tachycardia are increased body temperature, toxic conditions of the heart, and stimulation of the heart by the autonomic nerves (Guyton, 1981). In healthy individuals, sinus tachycardia occurs as an adaptation mechanism for adjusting cardiac output to the demands of the organism, e.g., in physical exercise or emotion (Robles de Medina et al., 1980), or in cognitive-informational tasks (Mulder and Mulder, 1981). In cases of chronic or recurrent sinus tachycardia, if differential diagnosis excludes organic pathology, psychosocial and psychodynamic factors can be found to be involved. Symptoms that often accompany the tachycardia include strong precordial pulsation, respiratory distress, chest symptoms, and jerking pulse (Garnier, 1981). Thus tachycardia may constitute a frightening, burdensome and taxing experience to the person, and one way for it to persist is when its interpretation (or cognitive attribution) induces a state of anxiety in the subject. The physiological changes thus brought about will intensify the cardiac sensations, and this will anchor the subject to his interpretations and anxiety, i.e., a vicious circle as described by Liebhart (1974).