Self-Regulation of Respiratory Sinus Arrhythmia

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Respiratory sinus arrhythmia (RSA) — the peak-to-peak variations in heart rate caused by respiration — can be used as a noninvasive measure of parasympathetic cardiac control. In the present study four strategies to increase RSA amplitude are investigated: (1) biofeedback of RSA amplitude, (2) biofeedback of RSA amplitude plus respiratory instructions, (3) respiratory biofeedback, and (4) respiratory instructions only. All four procedures produce a significant increase of RSA amplitude from the first physiological control trial compared to baseline. This increase is faster for the groups that received respiratory biofeedback and respiratory instructions only than for the two groups that received biofeedback of RSA amplitude, the increases being equivalent for the four groups in the third session. All subjects of the group that received biofeedback of RSA amplitude only reported respiratory strategies in order to achieve the increase in RSA. Possible clinical implications of these results for parasympathetic cardiac control and cardiovascular disorders are discussed.

KEY WORDS: Biofeedback; Respiratory sinus arrhythmia; Parasympathetic cardiac control; Respiratory rate; Respiratory amplitude.

Respiratory sinus arrhythmia (RSA) consists of the cooccurrence of cyclical fluctuations in heart rate in close correspondence with the respiratory cycle: increases in heart rate during inspiration and decreases during expiration. These fluctuations depend on specific respiratory patterns. Respiratory patterns characterized by low rates and high amplitudes increase RSA to the

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maximum, while the patterns characterized by high rates and low amplitudes reduce it drastically (Eckberg, 1983).

Traditionally it is believed that the magnitude of RSA changes with age, the highest amplitudes being demonstrated in children and the lowest in older people (Waddington, MacCulloch, & Sambrooks, 1979). However, more recent studies have found relatively high levels of RSA among older individuals who are healthy and active (Hirsch & Bishop, 1981) and low levels among children who suffer from psychosis and hyperactivity and adults with diabetes or cardiovascular illnesses (Hilsted & Jensen, 1979; Johnston, 1980; Piggott, Ax, Bamford, & Fetzner, 1973). This suggests that the relation of age and sinus arrhythmia can be partially confounded by the higher incidence of disease among the older population (Grossman, 1983).

Although the physiological mechanisms responsible for RSA imply complex interactions among different physiological processes, both peripheral and central, the generators of the RSA are cyclical vagal discharges due to the inhibition of vagal efferent activity during inspiration (Katona, Poitras, Barnett, & Terry, 1970). The administration of atropine and vagotomy eliminates the RSA, and variations in the heart rate linked to breathing can be predicted based on the efferent cardiac vagal activity (Katona, Poitras, Barnett, & Terry, 1970). Given this vagal modulation of the RSA, its amplitude can be used as a noninvasive index of parasympathetic cardiac control (Katona & Jih, 1975).

At a clinical level, it is considered that normal parasympathetic responsiveness fulfills a protective function for the heart. This has been shown both in human clinical studies and in animals. Several studies show a clear association between reduced parasympathetic cardiac control and cardiovascular dysfunction (Eckberg, Drabinsky, & Braunwald, 1971; Eckberg, 1980; Eckholdt, Bodmann, Pfeifer, & Schubert, 1976; Grassman & Blomquist, 1977; Johnston, 1980). This is consistent with data from other studies which predict illness and death by cardiovascular failure from reduced levels of RSA amplitude, while patients with cardiovascular problems who at the same time show normal RSA have a greater life expectancy than patients whose heart rates do not change with respiration (Hinkle, Carver, & Plakun, 1972; Kleiger, Miler, Bigger, & Moss, 1987). Similar associations have been reported with respect to diabetes (Hilsted & Jensen, 1979; Ewing, Campbell, & Clarke, 1980; Kitney, Byrne, Edmonds, Watkins, & Roberts, 1982). As regards the possible physiological alterations responsible for this reduction in parasympathetic cardiac control, the data suggest a loss of sensitivity of the baroreceptor reflex (Eckberg, 1980; Bertinieri et al., 1987). At an experimental level, different animal studies clearly indicate that parasympathetic activation can prevent the development of fatal