Autonomic nervous system status and responsiveness and the levels of anxiety in a normal population

We live in times of deadlines and information overload. Many individuals are mentally and emotionally overextended with conscious - or subconscious - feelings of apprehension and worry chronically present. The increased heart rate associated with certain psychological states and disorders has, until recently, primarily been ascribed to increases in the activity of the sympathetic nervous system. However, it is now accepted that decreases in parasympathetic control, that is, decreases in the autonomic 'brake' on heart rate, are much more important than previously assumed.[1,2] Perhaps of greater concern with regard to anxiety disorders are indications of an autonomic inflexibility or decreased responsiveness in the face of a challenge.[1,2] In view of the importance of the autonomic nervous system (ANS) to instantaneously regulate many functions in line with mental and physical needs, lower responsiveness could have a significant influence on physical and psychological well-being. The question is whether anxiety, within the normative range, has an impact on resting ANS functioning and on the ANS responses to moderate everyday challenges.

This is a report on the association between resting ANS status and responsiveness, and the levels of anxiety in a group of healthy volunteers. Eight adult volunteers, without previous complaints of anxiety or burnout and not on antidepressants or any medication that influences cardiovascular functioning, took part in the pilot study. Clearance was obtained from the Ethics Committee, Faculty of Health Sciences, University of Pretoria and all volunteers signed informed consent. ANS function was assessed by heart rate variability (HRV) techniques.[3,4] Tachogram recording sessions consisted of a stabilization period of 10 minutes, a 5 min baseline recording, a 5 min recording during a cognitive challenge and a 5 min recording over a recovery period after the cognitive challenge. Focussed attention, elicited and monitored by means of Infinity biofeedback equipment, was employed as the cognitive challenge. It involved an activity screen with a bowling ball animation, with the movement of the ball determined by the extent to which the subject increased the relative power of his/her sensorimotor rhythm. Anxiety was assessed by the State-Trait Anxiety Inventory for Adults.[5]

Mean state and trait anxiety for the group fell within the normal range with mean state anxiety at the 48th and mean trait anxiety at the 79th percentile of normal. Negative correlations were seen between parasympathetic control of the heart and levels of state anxiety in the baseline recordings (HF ms^2: $r = -0.714$, $p = 0.047$; HF%: $r = -0.643$, $p = 0.086$), as well as during the recovery period after the cognitive challenge (HFms2: $r = -0.857$, $p = 0.007$). Indications of a negative correlation between trait anxiety and parasympathetic control were seen with time domain and Poincare analyses ($r \leq -0.635$, $p \leq 0.091$). These results, in individuals with anxiety levels within the normal range, suggest lower parasympathetic cardiac control with subtle increases in anxiety levels. The findings are in line with the decreased parasympathetic activity reported in a number of anxiety disorders and with the vagal-suppressive effect described when subjects are subjected to stimuli that induce anxiety.[1,2]

No significant correlations were found between anxiety and indicators of sympathetic functioning. This uncertain outcome with regard to anxiety and sympathetic control, mirrors that of reports on anxiety disorders and excessive worrying that vary from decreased, to increased, to no different from normal sympathetic cardiac control.[1,6] Results of the study thus suggest that moderate levels of anxiety in the normal healthy population can reduce the inhibitory vagal tone to the sino-atrial node and, by implication, increase resting heart rate.

When the direction of the ANS response to the cognitive challenge was examined, a marginally significant increase (HF%: $p=0.0516$; HFnu: $p=0.0549$),) was seen from baseline to challenge in the parasympathetic, and a significant decrease (LF%: $p =0.0185$) in sympathetic, cardiac control. These results should be viewed against the type of stressor applied. Although the ANS response to physical stressors is largely understood, some controversy still exists about the effect of cognitive stressors. The response to a cognitive stressor appears to be influenced by the intensity, as well as by the type, of stressor. In this study focussed attention was applied as the cognitive stressor. However, different responses are known to occur depending on the type of attention. The so-called open attentional stance is said to lead to an ANS shift in favour of cardiac deceleration, while the closed attentional stance appears to induce an autonomic shift that results in heart rate acceleration.[7] The differences between the autonomic effects of the open and closed stances can be equated to that between the orienting and the defensive response, respectively.[7] The cognitive task in this study, i.e., moving objects on the screen through

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concentration, was that of orienting and one would therefore expect an autonomic nervous system response in line with cardiac deceleration. In the ANS response from baseline to challenge this was indeed observed in indications of an increase in the parasympathetic and a decrease in sympathetic control during the challenge.

When the magnitude of ANS responsiveness to the cognitive task was compared to baseline anxiety levels, time domain and Poincare indicators of parasympathetic control showed significant negative correlations with state anxiety ($r \leq -0.738$, $p \leq 0.037$), while frequency domain analysis showed a weak negative correlation with trait anxiety ($r = -0.647$, $p = 0.083$). The results thus suggest that, even in the normal healthy population with anxiety levels within the normative range, ANS flexibility varies inversely with resting anxiety levels. This is in contradiction to the erstwhile concept of an anxiety-associated autonomic lability and hyperreactivity that was an outflow of the work of Cannon[8], but in line with current concepts of high variability and flexibility as a reflection of coherence and viability.[2,9]

In conclusion, higher levels of anxiety, but still within the normal range, are associated with lower parasympathetic cardiac control and diminished ANS flexibility. Higher levels of anxiety, still within the normal range, may therefore contribute to a faster resting heart rate and a weakened ANS responsiveness to every day challenges. In view of the wide spectrum of functions controlled by the ANS, this decreased responsiveness (flexibility) could diminish the appropriate reactions to challenges. The major limitation of this pilot study is the small number of individuals investigated. A moderate cognitive stressor was used and it is feasible to expect more significant effects with anxiety-provoking stressors.

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