The use of heart rate variability measures to assess autonomic control during exercise

G. R. H. Sandercock1,2, D. A. Brodie2

1School of Health and Human Sciences, London Metropolitan University, London, UK, 2Research Centre for Health Studies, Buckinghamshire Chilterns University College, Buckinghamshire, UK

Corresponding author: Gavin R. H. Sandercock, School of Health and Human Sciences, London Metropolitan University, London, UK. E-mail: g.sandercock@londonmet.ac.uk

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Heart rate variability (HRV) is a non-invasive indicator of cardiac autonomic modulation at rest. During rhythmic exercise, global HRV decreases as a function of exercise intensity. Measures reflecting sympathovagal interactions at rest do not behave as expected during exercise. This makes interpretation of HRV measures difficult, especially at higher exercise intensities. This problem is further confounded by the occurrence of non-neural oscillations in the high-frequency band due to increased respiratory effort.

Alternative data treatments, such as coarse graining spectral analysis (CGSA), have demonstrated expected changes in autonomic function during exercise with some success. The separation of harmonic from fractal and/or chaotic components of HRV and study of the latter during exercise have provided further insight into cardioregulatory control. However, more research is needed. Some cross-sectional differences between HRV in athletes and controls during exercise are evident and data suggest longitudinal changes may be possible.

Standard spectral HRV analysis should not be applied to exercise conditions. The use of CGSA and non-linear analyses show much promise in this area. Until further validation of these measures is carried out and clarification of the physiological meaning of such measures occurs, HRV data regarding altered autonomic control during exercise should be treated with caution.

During exercise, heart rate (HR) increases linearly with work rate; individual training status influences this chronotropic response of the heart. The interaction of the sympathetic and parasympathetic branches of the autonomic nervous system (SNS and PNS) and their control over HR has been of interest to researchers for over 100 years. Surgical and pharmacological procedures using animal models (Hughson et al., 1977; Ordway et al., 1982) and humans (Robinson et al., 1966; Sutton et al., 1967; Lewis et al., 1980; Katona et al., 1982) have been employed to quantify the influence of the SNS and PNS on HR at rest and in response to both acute and chronic exercise. Such procedures are both invasive and expensive, making the idea of a non-invasive method of studying autonomic control very appealing.

Heart rate variability (HRV) is an established non-invasive tool which can be used to study the autonomic control of HR (Akselrod et al., 1981; Pagani et al., 1986; Malliani et al., 1991; Dreifus, 1993; Taskforce, 1996). Recent technological advances have made the application of HRV measures accessible to many researchers. The appealing prospect of gaining a non-invasive representation of autonomic nervous system (ANS) activity during exercise has lead to researchers in various fields attempting to use HRV methods and the number of such studies is increasing.

Behind the relatively simple acquisition of RR interval data for HRV analysis there is a minefield of potential data treatment and interpretation problems. There are many data treatment methods for the simple RR interval data obtained at rest or during exercise (time domain, frequency domain, and non-linear methods). A full list of abbreviations and brief descriptions of selected HRV measures appearing in this review can be found in Table 1.

HRV has classically been used to measure resting autonomic control (Pomeranz et al., 1985; Pagani et al., 1986, 1997; Malliani et al., 1991; Montano et al., 1994; Warren et al., 1997; Goldberger, 1999; Houle & Billman, 1999; Stein & Kleiger, 1999). These measures were developed and validated under resting conditions and few studies have attempted to validate their use during exercise. Most HRV measurements are predominantly influenced by PNS activity and the action of the baroreflex. The actions of these two controllers are drastically modified during exercise. It would, therefore, seem intuitive that
a measure, which is predominantly modulated by the PNS, may have little value under conditions where PNS withdrawal is known to occur.

The aims of the present article are to review the changes in HRV measurements during acute bouts of exercise and to establish the degree of accuracy with which they represent changes in ANS activity. A brief review is also provided of cross-sectional differences in resting HRV measures between groups with differing activity levels and the longitudinal effects of exercise training on resting HRV. Detailed recent reviews are available elsewhere (Aubert et al., 2003; Carter et al., 2003a)

**Differences in HRV measures between active and inactive subjects**

Surgical and pharmacological studies of autonomic regulation have lead to the classical view that the resting bradycardia observed in trained individuals was due to increased PNS activity (Frick et al., 1967; Ekblom et al., 1973; Smith et al., 1989). This view is not, however, universally accepted and contradictory evidence to refute this claim also exists (Sutton et al., 1967; Lewis et al., 1980). Katona et al. (1982) stated that several authors (Raab et al., 1960; Frick et al., 1967; Ekblom et al., 1973) may have misinterpreted pharmacologically derived data. In general, it seems that global HRV measures and spectral measures related purely to vagal control of HR are higher in trained individuals than untrained individuals (Kennedy, 1985; Maciel et al., 1985; Reiling & Seals, 1988; Gallagher et al., 1992; Goldsmith et al., 1992; Janssen et al., 1993; Puig et al., 1993; Shin et al., 1995; Davy et al., 1996; Macor et al., 1996; Bonaduce et al., 1998; Tulppo et al., 1998) although mixed (Sacknoff et al., 1994) and contradictory data also exist (De Meersman, 1993; Melanson, 2000; Migliaro et al., 2001; Buchheit et al., 2005).

**Longitudinal changes in HRV due to exercise training**

The use of spectral analysis to monitor longitudinal changes has also created equivocal results. Many studies show no significant alterations in HRV measures despite increased $\dot{V}_{O_{2,\text{max}}}$, and resting bradycard-
dia (Boutcher & Stein, 1995; Davy et al., 1997; Bonaduce et al., 1998; Loimaala et al., 2000; Myslivecek et al., 2002; Perini et al., 2002). Results are, however, mixed and in contrast to the above findings, a number of studies report significant alterations in autonomic balance measured by spectral analysis following exercise interventions (al-Ani et al., 1996; Schuit et al., 1999; Hedelin et al., 2000; Melanson & Freedson, 2001; Portier et al., 2001; Tulppo et al., 2003).

Even within studies that report statistically significant alterations in some HRV measures, not all changes are in the expected direction. One problem when reviewing these data is the sheer volume of results reported by authors. A single ECG trace can be treated in many ways and authors often lose the important or relevant outcomes in the myriad of results presented. This leads to studies giving mixed results (Schuit et al., 1999; Hedelin et al., 2001; Melanson & Freedson, 2001; Portier et al., 2001; Myslivecek et al., 2002; Carter et al., 2003b; Iwasaki et al., 2003). Small sample sizes, a paucity of a priori power calculations and varied data treatments also contribute to the heterogeneity of findings. Our recent meta-analysis of a large number of these studies (Sandercock et al., 2005) found significant increases in RR interval and the vagally mediated HRV measurement of high-frequency (HF) spectral power following exercise training. However, few data were obtained from randomized controlled trials and recent, long term data from two such trials showed non-significant changes in HRV (Uusitalo et al., 2004; Tuomainen et al., 2005).

**HRV during exercise**

The behavior of HRV measured during rhythmic exercise has been studied using either a single intensity steady-state exercise (Arai et al., 1989; Kamath et al., 1991; Dixon et al., 1992; Myslivecek et al., 2002), a number of steady-state intensities (Perini et al., 1990, 2000, 2002; Breuer et al., 1993; Gregoire et al., 1996; Macor et al., 1996; Piepoli et al., 1996; Tulppo et al., 1996), incremental stages (Yamamoto et al., 1992; Casadei et al., 1995), ramping (Yamamoto & Hughson, 1991; Nakamura et al., 1993; Tulppo et al., 1998) and even simulated altitude (Yamamoto & Hughson, 1991). Static, isometric exercise has also been used (Taylor et al., 1995; Raymond et al., 1997; Iellamo et al., 1999) although these protocols will not be discussed here.

**Global HRV measurements**

Several common patterns clearly emerge from the study of HRV during exercise. Firstly, when expressed in either the frequency domain (as TP) or in the time domain (such as by SDNN), HRV is greatly reduced during exercise (Arai et al., 1989; Furlan et al., 1990; Perini et al., 1990, 2000; Dixon et al., 1992; Breuer et al., 1993; Casadei et al., 1995; Macor et al., 1996). Incremental exercise protocols show that the decrease in global HRV occurs as a function of exercise intensity (Perini et al., 1990; Breuer et al., 1993; Casadei et al., 1995; Tulppo et al., 1996, 1998). More importantly, the decrease in variability may quickly reach the limit of resolution of some analysis systems (Rimoldi et al., 1990; Pagani et al., 1995). Methodologically this means that time and frequency domain measures of HRV can be difficult to interpret, especially at higher exercise intensities.

The behavior of short-term spectral HRV measurements

The physiological interpretations of LF and HF spectral power have been derived from both pharmacological data at rest (Hedman et al., 1992; Jokkel et al., 1995; Nakata et al., 1998) and measurements during manoeuvres such as head up tilting (Pagani et al., 1986, 1997; Furlan, 1987; Montano et al., 1994; van de Borne et al., 1997). Such data have lead to the assumption that HF is vagally mediated and that LF is of mixed sympathovagal genesis. The LF:HF ratio has been proposed as a measure of sympathovagal balance (Pagani et al., 1986), although experimental data to support this supposition are limited (Goldberger, 1999). In fact the existence of any “balance” between the two branches as well as the efficacy of LF:HF to measure it have both been repeatedly challenged (Eckberg, 1997; Goldberger, 1999).

Pharmacological data have shown that vagal withdrawal; followed by sympathetic activation, bring about the changes in HR observed during exercise. Given this knowledge, and assuming that the behavior of HRV measures at rest and during orthostatic challenge (e.g. head up tilt) can be extrapolated to exercise conditions, a decrease in HF and greater proportion of TP in the LF band would be expected. Such a pattern is, however, rarely reported.

**Changes in spectral power expressed in raw units**

When expressed in raw units (ms²), both LF and HF decrease exponentially as a function of exercise intensity (Arai et al., 1989; Perini et al., 1990, 2000, 2002; Rimoldi et al., 1992; Casadei et al., 1995; Tulppo et al., 1996, 1998). Observed decreases in HF have lead to the suggestion that it may be a suitable marker of vagal modulation during exercise. However, several problems with this assumption become evident at higher exercise intensities. These are discussed below. The often near total elimination
of raw LF and HF power mean little information can be gained from the study of these frequency components when expressed in this way. To gain insight into the changes in autonomic control associated with exercise, researchers have used one or more of the following data treatments:

i. the expression of LF and HF as percentages of total spectral power (LF%, HF%),

ii. normalized units (LFnu, HFnu), and

iii. the LF:HF ratio.

Changes in LF% and HF% during exercise
During a single bout of steady-state exercise (50% $V_{O2\,max}$), Kamath et al. (1991) found that LF% was greater when compared with measurements made in the supine position, but unchanged when compared with standing values. Dixon et al. (1992) also found that LF% was greater during exercise than when the subject lay supine, but unchanged when compared with standing values (Kamath et al., 1991; Dixon et al., 1992). HF% also remained unchanged compared with standing values.

Using incremental absolute exercise intensities, LF% and HF% were found to be similar to standing values when cycling at 50 W but then to fall rapidly at 100 and 150 W (Perini et al., 1990). Similarly, Casadei et al. (1995) found LF% increased slightly whereas a slight decrease in HF% at 110 W was found. LF% and HF% both then rapidly decreased in an almost inverse linear pattern with exercise intensity up to 221 W. A similar decline in LF% was found in healthy, older subjects (Perini et al., 1990). This decline in LF% was accompanied by an almost linear reduction in HF% power over exercise intensities equating to oxygen consumption values from 5 to 25 mL/kg/min.

During supine cycling, LF% initially increased at 20% $V_{O2\,max}$, remaining elevated at 40% and 60% of $V_{O2\,max}$, compared with resting values (Macor et al., 1996). HF% was reduced (25-fold) at 20% $V_{O2\,max}$ and by >100 fold at 60% $V_{O2\,max}$. Using LF% and HF% to study autonomic balance during exercise, there is some evidence to suggest the expected augmentation and reduction of LF% and HF%, but only at low-to-moderate intensities. LF% and HF% decrease dramatically as TP is reduced and a greater proportion of TP moves into the VLF waveband (Perini et al., 1990; Casadei et al., 1995).

Such findings support the parasympathetic genesis of HF. Reductions in HF% that appear to be a function of exercise intensity may reflect vagal withdrawal at lower intensities. Such findings do not, however, support the sympathetic evolution of LF%.

Changes in LFnu, HFnu and LF:HF ratio during exercise
To exclude the influence of VLF (which cannot be measured accurately from short-term recordings) and to control for the massive decreases in TP, LF and HF have been reported in normalized units and using the LF:HF ratio. When calculated as the proportion of spectral power in the LF or HF bands divided by the total power in the two bands (see Table 1), LFnu and HFnu are quantitatively similar to the LF:HF ratio.

The use of the LF:HF ratio as a measure of sympathovagal balance is supported by evidence from orthostatic stress tests and pharmacological manipulation (Pagani et al., 1986, 1997; Furlan, 1987; Montano et al., 1994; van de Borne et al., 1997). However, further critical experimental evidence suggests the circumstances under which the LF:HF ratio and the balance between sympathetic and vagal SA node modulation is limited (Goldberger, 1999). As orthostatic and pharmacological sympathetic stimulation increase LF:HF, one would expect that LF:HF would also increase during exercise, perhaps as a function of exercise intensity.

Kamath et al. (1991) found standing values for LF:HF remained unchanged during steady-state exercise. A common behavior of LF:HF during incremental exercises is an increase at lower intensities followed by a decline as exercise intensity increases (Arai et al., 1989; Rimoldi et al., 1992; Breuer et al., 1993; Casadei et al., 1995; Tulppo et al., 1996).

This pattern of change is not, however, universally reported (Perini et al., 1990). During incremental exercise, Perini et al. (1990) found LF:HF ratio decreased steadily up to 50% $V_{O2\,max}$ then increased, although not significantly at 70% $V_{O2\,max}$. Additionally, in older subjects (Perini et al., 2000) LF:HF remained unchanged during low intensity exercise before decreasing at higher intensities due mainly to decreased LF power.

Using exercise to elicit HRs of 100 and 150 (beats/min), Breuer et al. (1993) found raw LF and HF decreased dramatically at both intensities and that along with plasma catecholamines and lactate, LF:HF ratio increased from 2.0 (at rest) to 3.3 at 100 beats/min, lactate and catecholamine levels increased again but the LF:HF ratio was significantly reduced. In a separate protocol, the authors found that an atropine-induced increase in
HR was accompanied by similar reductions in raw LF and HF but with an opposite effect on LF:HF.

Such findings lend support to the notion that vagal activity modulates both LF and HF power but do not support the use of LF or LF:HF as markers of sympathetic modulation or sympathovagal balance. The dissociation between plasma catecholamines, lactate and LF, or LF:HF during exercise, challenges the usefulness of these measures under such conditions. The contradictory findings from pharmacological and exercise-induced HR changes may suggest that changes in LF:HF obtained under pharmacological manipulation in resting subjects cannot be accurately extrapolated to measurements made during exercise.

To our knowledge, only one study has shown changes in LF and HF that coincide with the known rearrangements in PNS and SNS activities (Yamamoto et al., 1991). Using data from six, 14 min, steady-state exercise stages, Yamamoto et al. (1991) found that HF oscillations declined steadily from rest to 60% of ventilatory threshold ($T_{vent}$). Above $T_{vent}$, the LF:HF ratio increased. The use of a LF spectral bandwidth of 0.00–0.15 Hz means power usually found in the VLF waveband was categorized as LF. These findings are not comparable with those of other studies using standard LF bandwidths. They have, to our knowledge, never been replicated.

To quantify the possible role of methodological disparities in the heterogeneity of findings, Casadei et al. (1995) assessed HRV during incremental exercise and reported their findings in raw units, normalized units and as a percentage of TP. Raw LF initially increased at 110 W but had disappeared at 221 W. HF was dramatically reduced at 110 W but remained unchanged until the end of exercise. When expressed as normalized units, LFnu increased initially, but then fell linearly until disappearing at 221 W. HFnu showed an initial decrease, but then increased in parallel with exercise intensity until it accounted for 80.4% of spectral power at 221 W. LF% initially increased but then disappeared and HF% remained unchanged throughout the protocol. The percentage of power in the VLF band remained unchanged to 147 W but then increased dramatically during more intense exercise. The use of LF and HF in raw units and in normalized units during exercise was described as misleading as the former is modulated very strongly by the reduction in TP that accompanies the shorter RR intervals associated with exercise. The increase in HFnu was attributed to non-neural factors such as stretch and atrial node activity.

Casadei et al. (1995) concluded that spectral analysis was unable to provide adequate assessment of PNS or SNS activity. Decreased activity in the LF and HF bands has been interpreted as a withdrawal of vagal activity but gives no additional information to that which could be gained from analysis of a more simple measure such as SDNN or even HR. The use of LF or LFnu was also said to be misleading and unable to reflect sympathetic activation as both disappear at higher workloads where adrenergic activity is increased.

It appears that one confounding factor in the use of power spectral analysis during exercise is the non-neural genesis of some HF oscillations (Bernardi et al., 1990; Casadei et al., 1996). Casadei et al. (1996) quantified this non-neural contribution by comparing HRV at rest and during exercise in healthy subjects and found that the proportion of HF power due to non-neural mechanisms was increased to 32% (range 17–75%) during exercise. Evidence for the existence of non-neural oscillations comes from distinct HF peaks in patients with fixed atrial pacing (Lombardi et al., 1996) and in denervated hearts of transplant patients (Bernardi et al., 1990).

It seems, therefore, that oscillations in the LF band are exclusively neurally mediated but that non-neural mechanisms contribute to the HF. Although the contribution of non-neural factors at rest is negligible, during moderate intensity exercise it becomes significant. This leads to specific problems associated with the use spectral analysis of RR interval as an autonomic marker during exercise.

Alternative data manipulation techniques have also been suggested in an attempt to rectify the unexpected behaviors of standard HRV measures and ratios using muscarinic, β-receptor and combined blockade at rest and during exercise (Warren et al., 1997). During several low-to-moderate exercise intensities, ratios of HF/TP and LF/TP were found to be unrepresentative of sympathetic and vagal changes when standard frequency domain measures were used. HF (0.1–1.0 Hz) provided a reliable index of vagal activity during exercise. Its behavior corroborated HR response and was supported by pharmacological data. No satisfactory sympathetic marker could be provided.

**HRV response to exercise: findings from studies using coarse graining spectral analysis (CGSA)**

Applying CGSA allows the separation of harmonic, linear and non-linear (fractal or chaotic) features of RR interval data (Yamamoto & Hughson, 1991; Yamamoto et al., 1991, 1992; Nakamura et al., 1993). CGSA improves resolution of short-term HRV spectra by removing the fractal component of the time series (Saul et al., 1988), leaving only harmonic components which exist at frequencies from 0.0003 to 0.01 Hz (10h–10s) and may affect assessment of what some researchers (Yamamoto & Hughson, 1991) term sympathovagal balance.

The harmonic LF and HF bands are commonly reported as LO (0.0–0.15 Hz) and HI (0.15–1.0 Hz)
although this is not universal. The LF:HF ratio becomes the LO:HI ratio. HI is commonly referred to as the PNS indicator, LO and LO/HI were referred to as the SNS indicator (Yamamoto & Hughson, 1991; Yamamoto et al., 1991). The ratio of HI/TP (also called PNS indicator) has also been reported (Gregoire et al., 1996; Amara & Wolfe, 1998; Myslivecek et al., 2002).

During ramped exercise (15 W/min), Yamamoto et al. (1991) found power in LO and HI spectral peaks declined and were eliminated by the time “moderate” exercise intensity was reached. Using the RR data described previously from six, 14 min, steady-state exercise stages, the PNS indicator declined steadily from rest to 60% of ventilatory steady-state exercise stages, the PNS indicator decreased and were eliminated by the time (Yamamoto et al., 1992) and interpreted as evidence of increased SNS activity above T_vent. However, this should be treated with caution for a number of methodological reasons. There was actually a large reduction in SNS indicator at the final work rate (40 W above T_vent) and the values for the PNS indicator increased at intensities 30–40 W above T_vent. Also the shallow ramping protocol failed to provide respiratory indicators that T_vent was reached.

Decreased PNS and increased SNS indicators have also been observed during exercise at 50 and 100 W (Gregoire et al., 1996) and also at a relative exercise intensity of 40% HR_{max} (Myslivecek et al., 2002). Both these studies used very low intensities of exercise and it should be borne in mind that general spectral analyses show very similar changes at such intensities.

Warren et al. (1997) make the point that certain RR interval treatments give results that may be coincidental with established theories of autonomic response. Validation of CGSA harmonic components under pharmacological blockade and using tilt are somewhat equivocal (Blaber et al., 1996; Tulppo et al., 2001) and incongruencies between CGSA-derived measures of central sympathetic outflow and direct microneurographic estimates exist (Notarius et al., 1999). Recent studies assessing changes in PNS and SNS indicators during very high-intensity exercise are notable by their absence and it is unclear why investigations (Gregoire et al., 1996a, b; Myslivecek et al., 2002a, b) have limited the use of CGSA to very low exercise intensities.

Non-linear HRV measured during exercise

Nakamura et al. (1993) reproduced previous SNS and PNS indicator results (Yamamoto et al., 1992) using an identical ramping protocol and investigated further the changes in fractal HRV. The fractal component (D_f) was estimated as the slope of the linear regression (β) assessed by plotting the log frequency vs log power and represents the complexity of a given time series with values of between 1<β<3 indicating a fractal signal. The more complex the time series the higher the value of β. Mathematically, β is representative of the number of independent oscillators responsible for the generation of any given time series (Yamamoto et al., 1992). Physiologically, β is representative of the number of inputs which affect the cardiovascular control centers and ultimately HR (Goldberger, 1990).

Nakamura et al. (1993) found values for D_f increased to ~3 during mild exercise (increasing complexity) but then decreased linearly at moderate to high intensities. β increased (>2) linearly at these intensities simultaneously with decreased PNS and increased SNS indicator values at high-intensity exercise where β was reduced to <2. This suggests the presence of a single dominant controller of HR. Coincidence of this point and a marked lactacidaemia led the authors to hypothesize that arterial chemoreceptor afferents may be the dominant input.

Measures of the fractal properties and the overall complexity of HR tend to show a biphasic response. At low-to-moderate intensities there is an increase in fractal correlation properties and increased complexity of HR dynamics. At higher workloads, these measures decline almost linearly as a function of exercise intensity (Yamamoto & Hughson, 1994; Yamamoto et al., 1995, 1996; Tulppo et al., 1998, 2001; Perkiomaki et al., 2002; Hautala et al., 2003).

Poincaré plot analysis

Non-linear dynamics of HR can be assessed both qualitatively and quantitatively using Poincaré plots. Values for SD1 and SD2 in a standard Poincaré plot give information regarding fast and slow changes in RR interval, respectively. Major advantages of Poincaré analyses over spectral measures are their simplicity and being applicable to non-stationary data sets.

Using combined exercise and pharmacological blockade (Tulppo et al., 1996) markers of vagal modulation were found to decrease progressively until the T_vent. Then sympathetic activation was reflected by significantly altered SD1:SD2 ratio. Harmonic variables were unable to illustrate such changes. The authors concluded that two-dimensional vector analysis of Poincaré plots could provide useful information on vagal modulation of RR interval dynamics during exercise that are not easily detected by linear summary measures of HRV. A similar sensitivity of SD2 to changes in RR interval regulation during exercise was later confirmed in patients using moxonidine (De Vito et al., 2002).
Cross-sectional and longitudinal studies examining differences in the HR response to exercise

Few studies have compared the HRV response to exercise between groups of subjects with different levels of aerobic fitness. This is undoubtedly due, in part, to unresolved methodological issues accompanying the use of HRV during exercise. The few data that exist are at best, equivocal.

Similar HRV measures were reported for athletes and controls during exercise at 50% max workload (Dixon et al., 1992) and during recumbent cycling at 20%, 40% and 50% $V_{O_{2max}}$ (Macor et al., 1996). Using incremental, upright cycling Tulppo et al. (1998) found that HF power during exercise from 50 to 100 W was significantly higher in an “average” fitness ($V_{O_{2peak}}$ 51 ± 4 mL/kg/min) compared with a “poor” fitness group ($V_{O_{2peak}}$ 34 ± 3 mL/kg/min). This difference was not evident at rest and disappeared at higher exercise intensities. Longitudinally, Perini et al. (2002) found no differences in LF(nu) or HF(nu) response to exercise after an 8 weeks aerobic training program.

Using CGSA, Gregoire et al. (1996) found that young, trained subjects showed greater preservation of TP and SDNN when cycling at 100 W compared with untrained controls, despite no differences in SNS or PNS indicators at rest or during exercise. Myslivecek et al. (2002) found evidence of preserved vagal modulation during exercise (40% HRmax) after an exercise training intervention.

HR dynamic and prognosis

Ambulatory and resting measures of HRV are significant prognosticators for adverse events in a number of patient populations (Kleiger et al., 1987; Guzzetti et al., 2005). No such relationship between exercise measures of HRV and adverse events has yet been demonstrated. Until the methodological issues highlighted above are resolved, such a study seems unlikely. Measures of HR dynamics such as HR recovery are already established as excellent markers of prognosis in some patient groups (Cole et al., 1999; Nissinen et al., 2003; Lipinski et al., 2005). Recently, the prognostic role of the ANS measured by observing HR dynamics during exercise has also been demonstrated (Falcone et al., 2005; Jouven et al., 2002). HRV analyzed during exercise may, in the future, provide further prognostic information than simple measures of HR.

Summary

Power spectral measures

Several authors (Perini et al., 1990; Casadei et al., 1995, 1996; Perini et al., 2000, 2002) agree that HRV is a useful measure for assessing the resting autonomic balance but that its use during exercise is limited. It is clear that appropriate treatment and representation of data is crucial as certain representations may be misleading. Insight into autonomic control during exercise from traditional spectral measures of HRV is severely limited. Results coincidental with alterations in sympathovagal interaction may be obtained at lower exercise intensities but the real physiological meaning of these is uncertain as few studies have attempted to validate findings with autonomic blockade (Warren et al., 1997). At higher intensities, the rearrangements in sympathovagal interaction that occur and the massive reduction in available spectral power render standard spectral measurements practically useless. Studies showing preserved HF power at high intensities due to mechanical stimuli support this viewpoint (Cottin, 2004) but give little information regarding autonomic control during high-intensity exercise.

CGSA

It seems that CGSA may be a more useful tool with which to observe the changes in autonomic function that accompany exercise. The abrupt increase in SNS indicator at or around the $T_{vent}$ is analogous to the known sympathetic response observed during incremental exercise using invasive methods (Wallin et al., 1992; Kingwell et al., 1994) and similar changes in SNS and PNS indicators have been replicated on several occasions using this method. However, the harmonic CGSA components during exercise have not yet been fully validated. Further concurrent microneurographic and/or pharmacologic validation of the PNS and SNS indicators is required and further work in this area is encouraged.

Methodological considerations

Different analytical techniques are, therefore, an undoubted source of variation in the results. In addition to using CGSA, standard spectral measures may also be analyzed parametrically (using a number of different order autoregressive models), or non-parametrically via Fourier type transformation (usually by the use of the regularly sampled interpolation of discreet event series).

What has long been recognized that the very large decreases in spectral power observed during intense exercise may quickly reach the limit of resolution of many analysis systems (Rimoldi et al., 1990; Pagani et al., 1995). Due to the small amounts of variation in RR interval during high-intensity exercise large amounts of high precision data are needed in order to obtain stable spectral estimates. There are also data to suggest that certain spectral power estimates
may be affected when calculated from different number of heartbeats (Yamamoto et al., 1994).

Some authors do not report sampling frequencies or the total number of beats sampled; although the latter can often be estimated from mean HR and sample duration. By doing this, it becomes obvious that few studies using standard spectral analysis have sampled a sufficient number of heartbeats to provide stable spectral estimates. Spectral estimates tend to be made on samples ranging from 200 to 500 beats (Perini et al., 1990; Tulppo et al., 1996, 1998) with a common sample length being 256 or 300 beats (Kamath et al., 1991; Casadei et al., 1995; Brenner et al., 1997) recorded over periods of 3–5 min.

Conversely, studies using CGSA have commonly used 10-min sampling periods; equating to 1000–1500 beat samples (Yamamoto & Hughson, 1991; Yamamoto et al., 1991, 1992, 1996; Nakamura et al., 1993; Myslivecek et al., 2002). When Yamamoto and Hughson (1991) studied changes in LF, HF and LF:HF ratio using Fourier analysis of 10-min RR interval data samples (sampled at 1000 Hz), values obtained via CGSA and Fourier analysis were very similar. Additionally, the latter also gave a fair reflection of autonomic change during exercise. This may indicate that data recording length could influence spectral estimates made during exercise although there are presently insufficient data to examine this claim in detail. A further methodological problem is the decreased signal-to-noise ratio in ECG recordings made at higher exercise intensities. Gregoire et al. (1996) found that 50% of tachograms recorded at 110% $T_{vent}$ or at training HR could not be analyzed due to low signal-to-noise ratio. Increases in likelihood of non-sinus heartbeats movement artefacts and other “noise” in ECG recordings mean that very careful filtering and editing of the RR interval time series should be carried out before HRV analysis. Some automated beat rejection and interpolation algorithms devised for resting tachogram filtering may not be satisfactory for this purpose. Techniques should be reported fully, as should the percentage of beats and/or tachograms accepted or rejected before final analysis.

Non-linear dynamics

In addition to the information produced by the PNS and SNS indicators, the ability of CGSA to separate the harmonic and fractal components of the heart signal provides further insight into the nature of autonomic control of HR. The fractal component increases as a percentage of the total spectral power with increasing exercise intensity but the component’s chaotic nature at rest is simplified, approaching unity during high-intensity exercise. The parallels between this harmonizing effect and the changes in this component at time of death warrant further investigation.

Certainly, the biphasic responses of certain non-linear measures may indeed represent the number of inputs impinging on the SA node. Alterations in RR interval complexity from resting conditions (predominating vagal activity) to moderate exercise (mixed sympathovagal activity) may occur via altered baroreflex function. At rest it is known that baroreflex function modulates harmonic oscillations centered on 0.1 Hz that fall in the LF power spectral band. Of interest, LF (expressed as a percentage or in normalized units) also increases and then decreases with exercise intensity in a similar pattern to some non-linear measures. The further use of Poincaré analysis to investigate non-linear dynamics of HR should also be encouraged due to their simplicity, ease of interpretation and the ability to apply this measure to non-stationary data series.

Recommendations

Little information can be gained from further study of HRV during exercise using the spectral analysis techniques recommended (Taskforce, 1996) for use at rest or during ambulatory data collection. Harmonic measures from CGSA and measurements of fractal and/or chaotic behavior show much promise, as they appear to approximate the known alterations in sympathovagal interactions. More information and validation studies in this area are being published all the time (Tulppo et al., 2001, 2005; Perkiomaki et al., 2005). Researchers interested in gaining a non-invasive insight into autonomic function during exercise should be encouraged to apply these analyses to RR interval data recorded. It has been recognized that the use of such measures are still far away from clinical medicine (Perkiomaki et al., 2005). Additionally, the availability and the relative simplicity of applying standard time and frequency domain analysis to RR interval data, coupled with the familiarity with measures produced by these methods may be an obstacle. If further research is to be conducted using standard frequency methods, duration of data collection should be long enough to ensure stable spectral estimates can be made.

Keywords: heart rate variability, exercise, autonomic nervous system.
Heart rate variability and exercise

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