Redefining respiratory sinus arrhythmia as respiratory heart rate variability: an international Expert Recommendation for terminological clarity

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Abstract

The variation of heart rate in phase with breathing, known as respiratory sinus arrhythmia (RSA), is a physiological phenomenon present in all air-breathing vertebrates. RSA arises from the interaction of several physiological mechanisms but is primarily mediated by rhythmic changes in cardiac parasympathetic (vagal) activity, increasing heart rate during inspiration and decreasing heart rate during expiration. RSA amplitude is an indicator of autonomic and cardiac health; RSA is diminished or absent in common pathological

conditions such as chronic heart failure and hypertension. In this Expert Recommendation, we argue that the term 'RSA', although historically important, is semantically inaccurate and carries a misleading pathological connotation, contributing to misunderstanding and misinterpretation of the origin and physiological importance of the phenomenon. We propose replacing 'RSA' with the term 'respiratory heart rate variability' (RespHRV), which avoids pathological connotations and emphasizes the specific respiratory contribution to HRV. We clarify that RespHRV encompasses respiratory-related heart rate variations in both the low-frequency and high-frequency bands traditionally defined for HRV and should not be misconstrued as a measure of vagal tone. Adoption of the proposed term 'RespHRV' is expected to unify understanding and promote further experimental and clinical studies into the physiological mechanisms and functional importance of this phenomenon.

[H1] Introduction

We often refer to a person's heart rate (HR) as a certain number of beats per minute, but recordings show substantial variability in HR under most physiological conditions, even at rest. Multiple factors contribute to and influence HR variability, including emotional state[1], cardiorespiratory fitness[2] and breathing[3]. The focus of this Expert Recommendation is the physiological variation of HR in phase with breathing, first observed in 1733 by the Reverend Stephen Hales, reported in 1847 by Carl Ludwig[4–6], and subsequently named 'respiratory sinus arrhythmia' (RSA). The primary mechanism underlying RSA involves rhythmic changes in the parasympathetic (vagal) cardioinhibitory influence that increases during expiration, resulting in a decrease in HR, and decreases during inspiration, leading to an increase in HR[7–18] (Fig. 1). Changes in the frequency and depth of breathing strongly influence the amplitude (the absolute difference in HR between inspiration and expiration) of RSA[19,20] (Fig. 2a). RSA is evolutionarily conserved and has been observed across all species

of air-breathing vertebrates studied so far, including mammals, reptiles, birds, amphibians and also lungfishes (one of the earliest obligate air-breathers in the vertebrate evolutionary record, which increase HR at each surface air breath[21,22]. The physiological function of RSA in terrestrial vertebrates has been debated, with suggestions that, in mammals, RSA improves pulmonary gas exchange, reduces cardiac energy expenditure while helping to maintain homeostasis of the arterial partial pressure of CO_2, and/or stabilizes arterial blood pressure and systemic blood flow[2,3,17,23,24]. In humans, the amplitude of RSA is larger in young and healthy individuals and gradually decreases with ageing[25]. RSA is greatly reduced or absent in common pathological conditions, such as chronic heart failure and hypertension[2,26,27] (Fig. 2). Reinstating RSA by rhythmic pacing of the heart in patients with heart failure is a promising therapeutic strategy that is being tested in clinical trials, after showing benefit in preclinical studies[26,27]. Slow, deep breathing practices aimed at amplifying RSA to promote relaxation and meditation are ancestral and common, spanning from yogic pranayama to 'cardiac coherence' and 'boxed' breathing[3,28–30]. However, when the physiological mechanisms and potential health benefits underlying these practices are explained, RSA is rarely mentioned. In this Expert Recommendation, we argue that the term 'RSA' is semantically incorrect and intuitively conveys a pathological connotation, often leading to confusion and aberrant interpretations. Attempts to circumvent this problem by substituting the term 'RSA' with the term 'high-frequency heart rate variability' (HRV) overlook a vital aspect of this physiological phenomenon: its inherent dependence on respiratory activity[17,31–33].

In this International Expert Recommendation we propose adopting and using the term 'respiratory heart rate variability', abbreviated to 'RespHRV', which overcomes these shortcomings. This name change represents more than a semantic improvement; we propose a fundamental reorientation that is expected to have profound implications for basic and

clinical research involving HRV analysis, which has often neglected the crucial role of respiratory modulation of HR. We argue that this oversight has contributed to common misunderstanding of the importance of the respiratory influence on HR. One consequence of this misunderstanding is the frequent use of imperfect proxies for RSA, such as high-frequency HRV, and ambiguous terms such as 'cardiac coherence' that have begun to permeate the public domain, further perpetuating confusion. We contend that the inadequacies of the term 'RSA' as a descriptor lie at the core of these issues and should be addressed. The adoption of the proposed term 'RespHRV' by the clinical and the basic research communities would be expected to promote accurate interpretations of HRV metrics and improve our understanding of this physiological phenomenon (Box 1).

[H1] Incorrect and misleading RSA terminology

Scientific terms and definitions evolve to reflect the development of ideas and advancement in understanding. The term 'RSA' probably emerged in the early twentieth century as knowledge of autonomic control expanded, particularly with the advent of electrocardiography and more precise physiological measurements. However, the first explicit use of the name 'RSA' in published research is uncertain. Semantically, 'arrhythmia' is derived from the Greek 'a-' (without) and 'rhythmia' (rhythm), implying a complete lack of rhythm. However, 'arrhythmia' has come to mean any abnormal heart rhythm, whether too fast, too slow or irregular and, therefore, the word carries a negative, pathological connotation. Any search for the definition of the term 'arrhythmia' leads to descriptions of abnormal, pathological rhythms of the heart, from specialist sources such as the US National Institutes of Health[34] or the American Heart Association[35], to popular sites such as Wikipedia[36]. Therefore, the term 'RSA' does not immediately point to an important physiological phenomenon but instead suggests a pathological condition. Moreover, the word

‘sinus’ in ‘RSA’ refers to the sinoatrial node, which generates the heartbeat (therefore the term ‘sinus rhythm’), but even this meaning is often misunderstood. A common misconception is that ‘sinus’ in RSA refers to the sinusoidal shape of the HR profile representing its variations with respiration. A clear understanding of the term ‘RSA’ has never reached the public and, in our experience, the term is often confusing even to clinical scientists and medical practitioners. When teaching students or engaging in community outreach and public events, use of the term ‘RSA’ requires careful and detailed explanation, which is counterproductive.

[H1] RSA metrics and (mis)interpretations

The idea that variation in HR is a sign of cardiac and general health is now widespread[37]. The term ‘heart rate variability’ (HRV) has gained popularity across a wide range of applications, from clinics to smartwatch and smartphone devices[31,33,38]. HRV can be assessed from simple recordings of cardiovascular activity, such as an electrocardiogram or blood pressure, followed by time-domain and/or frequency-domain analyses[33,39]. Its widespread accessibility, together with the tantalizing promise of a non-invasive and ready-to-use solution to measure autonomic balance and health status, have led to oversimplifications and overinterpretations of HRV metrics. The prominent respiratory component in HRV (which is RSA) is often oversimplified as the high-frequency HRV band, overinterpreted as a measure of parasympathetic influence on the heart and, by extension, overinterpreted as a measure of overall vagal activity, autonomic balance and general health[12,17,19,31,32]. These aspects are discussed below.

When HRV analysis is performed in the frequency domain, the power spectral density at the respiratory frequency provides a measure of the respiratory variation in HR (Fig. 2b). In adult humans, respiratory frequency is usually between 0.15 and 0.4 Hz. In this frequency

range, the respiratory variation in HR is, indeed, the main component of the HRV power spectrum, usually appearing as a distinct peak. This understanding led to the segmentation of the 0.15–0.4 Hz range of HRV as the high-frequency band of HRV. However, efficient lung ventilation can be achieved at lower respiratory rates, with a substantial percentage of breaths in healthy humans under resting conditions occurring below 0.15 Hz[40]. Additionally, voluntary slow, deep breathing is typically practised at approximately 0.1 Hz (6 breaths per minute). At these lower breathing rates, the respiratory-related HRV merges with other HRV components in the low-frequency band (0.04–0.15 Hz), complicating its accurate measurement. Nevertheless, because the high-frequency band is the most selectively quantifiable HRV band and covers predominant respiratory frequencies, this band is now commonly used as a measure of the respiratory modulation of HR[32,33]. The term 'high-frequency HRV' is frequently used as a synonym for 'RSA', which is erroneous because it conflates the physiological phenomenon with the method used to capture it — and only a fraction of it (the high frequencies) is captured. The use of the term 'high-frequency HRV' further introduces an important analytical and interpretative issue, primarily because it fails to acknowledge the inherent respiratory origin of the phenomenon it is aimed to describe. This oversight has led to important misinterpretations, a problem that has been discussed in detail in the field of psychophysiology[32,41].

Given that the respiratory variation in HR is primarily generated via parasympathetic modulation, it has been used as a marker of cardiac vagal activity[17,42,43]. This interpretation is often extended to the high-frequency band of HRV. It is important to understand that cardiac vagal parasympathetic activity comprises both rhythmic and tonic components[12,18]. The parasympathetic activity that contributes to generation of the respiratory variation in HR includes only the rhythmic, respiratory-related component. RSA, or more appropriately RespHRV, describes and approximates the amplitude of variations in cardiac vagal activity

about its mean value, rather than the tonic level of cardiac vagal activity, across a measurement period. This variation probably serves a very specific and evolutionarily conserved function of coordinating ventilation and circulation, which is particularly important in non-mammalian vertebrates with undivided circulations[17,21]. Furthermore, the respiratory variation in HR is predominantly but not exclusively generated by rhythmic changes in cardiac vagal activity; additional contributing factors include the respiratory modulation of cardiac sympathetic activity[17,44] and respiratory-related changes in venous return to the heart[17,45] (Fig. 1). Within-individual variations in respiratory parameters have been shown to exert profound effects on the amplitude of the respiratory variation in HR, independently of overall changes in cardiac vagal activity[17,19]. Therefore, the respiratory variation in HR results from several concurrent mechanisms and is neither a selective nor an absolute marker of cardiac vagal activity[17].

When the respiratory variation in HR and the high-frequency band of HRV are interpreted as markers of cardiac vagal activity, they are then often generalized as measures of overall vagal tone, autonomic balance and general health[17,19,42,43]. Similar generalizations are frequently applied to the interpretation of common time-domain metrics of HRV, such as the RMSSD (root mean square of successive RR interval differences) and, to a lesser extent, the SDNN (standard deviation of NN intervals). These generalizations are at odds with the organization and functioning of the autonomic nervous system, which controls physiological functions in an organ-specific, tissue-specific and context-dependent manner[46–48]. We are not aware of any experimental evidence suggesting that the amplitude of respiratory variation in HR reflects the activity of parasympathetic efferent innervation of any organ other than the heart. However, strong evidence does indicate that physiological HR variability (which can be estimated by the analysis of HRV) is associated with improved outcomes[37], and a growing number of commercial companies offer solutions that claim to increase vagal tone and

improve health by amplifying respiratory variations in HR. These providers do not use the term ‘RSA’ and have developed alternative terms, such as ‘cardiac coherence’. The term ‘coherence’ is used in its signal processing sense, denoting the relationship between respiration and HRV in the frequency domain. Similar to ‘high-frequency HRV’, the term ‘cardiac coherence’ is largely based on a methodology rather than the physiological phenomenon it is intended to describe. Claims are made that one can be either in a state of cardiac coherence or not, creating a sense of dichotomy, and that the state of cardiac coherence can be achieved by practising voluntary slow, deep breathing, typically at 6 breaths per minute (0.1 Hz). In reality, the state of cardiac coherence represents an amplified respiratory variation in HR, in which the respiratory-related band merges with other low-frequency HRV components, including one that is probably related to the operation of the baroreflex and potentially induced by blood pressure oscillations at about 0.1 Hz (Mayer waves)[49–51]. Conversely, the concept of cardiac coherence does not account for the basal level of respiratory variation in HR at higher (and much more common) respiratory rates in what would then be considered ‘incoherent’ states, nor does it consider that the respiratory variation in HR can also be amplified in a state-dependent manner at these higher breathing frequencies (Fig. 2). It is concerning that the concept of ‘cardiac coherence’ is now used by many practitioners, with the health benefits claimed that are far-reaching and based on misunderstanding and misinterpretation of the underlying physiology and recorded measures.

Therefore, in our opinion, there is a clear need for a more appropriate and scientifically accurate term than ‘RSA’ to describe the respiratory variation in HR that would accurately reflect the underlying physiological mechanisms and help to prevent inaccurate interpretations. The new term must be easily understandable and usable by researchers, medical practitioners and the general public.

[H1] The proposed new name: RespHRV

The term HRV is already widely recognized and used, from scientific and medical literature to consumer applications. We propose to use the three-letter abbreviation 'HRV' as the core of the new name. As discussed, although the high-frequency component of HRV is commonly used as a measure of RSA, it is not an accurate measure because it does not take into account the respiratory-related modulation of HR at lower breathing rates. In addition, interpretation of RSA and of the high-frequency component of HRV as markers of cardiac vagal activity and, by extension, of overall vagal tone is problematic, particularly in the psychobiological literature, in which the amplitude of RSA is almost exclusively used as a vagal index[17,21,43,46]. The explicit inclusion of the term 'respiratory' in the new name could help to prevent these issue, and would also be entirely appropriate from a physiological perspective, given that the respiratory variation in HR is the only component of HRV with a well-defined origin (that is, respiratory activity), emerging predominantly from the rhythmic modulation of cardiac vagal preganglionic neuron activity by the neighbouring respiratory network and afferent feedback[7–18] (Fig. 1).

Therefore, we propose the term 'respiratory HRV' (RespHRV) to replace the term 'RSA'. This term is characterized by its explicit, intuitive and unambiguous nature, which should serve to circumvent the issues with the current terminology discussed above. Some publications[52] have already used this term and, in our opinion, its broader adoption and use by researchers, clinicians and the community could help to unify our understanding and interpretation and promote further experimental and clinical studies designed to better understand the physiological mechanisms and functional importance of this phenomenon (Box 1).

[H1] Conclusions

Brain–body interactions are in the spotlight, garnering substantial research funding and heightened media attention. The specific topic of brain–heart interactions occupies a particularly prominent position in this focus[53,54], with breakthroughs in our understanding of autonomic cardiac regulation and cardiac interoception[55–60]. In this context, where the autonomic control of the heart is gaining increased exposure but remains entangled in ambiguous and inaccurate terminology that can be challenging for non-specialists, we recommend replacing the historically important, but inaccurate and misleading term 'RSA' with the term 'RespHRV'. We are confident that adopting the name 'RespHRV' to describe respiratory modulation of HR would benefit not only the research community but also the general public (for example, users of smartwatch applications that estimate HRV) by providing a clear term that accurately reflects the underlying physiological phenomenon to facilitate basic science and clinical research in this area and promote public understanding of the physiology discipline.

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Author contributions

C.M. researched data for the article. C.M., A.B-T., A.M.A., D.G.S.F., E.W.T., J.F.R.P., J.B., P.G., T.R. and A.V.G. discussed the content of the article. C.M. and A.V.G. wrote the

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Competing interests

The authors declare no competing interests.

Fig. 1 | Physiological mechanisms underlying the generation of RespHRV. Schematic representation of the main mechanisms contributing to respiratory heart rate variability (RespHRV). The brain controls the heart via branches of the autonomic nervous system, with cardiac parasympathetic and sympathetic activities being modulated by respiration[9,12,16–18,44]. RespHRV emerges from rhythmic modulation of the activities of cardiac vagal preganglionic neurons and presympathetic neurons in the brainstem by inputs from the respiratory central pattern generator (neural circuit oscillator (∿) that generates the respiratory command), as well as cardiorespiratory afferents, including pulmonary stretch receptors, arterial baroreceptors and veno-atrial stretch receptors. These interactions result in opposing rhythmic patterns of cardiac parasympathetic and sympathetic activities[9,12,16,18,44], which converge at the cardiac pacemaker to increase heart rate during inspiration and decrease heart rate during expiration. Respiratory-related changes in venous return further contribute to RespHRV and arise from changes in intrathoracic pressure affecting the pulmonary circulation, and variations in systemic arterial blood pressure in phase with respiration (Traube–Hering waves). These mechanical effects contribute to RespHRV via the Bainbridge effect, which is an increase in heart rate during inspiration mediated by veno-atrial stretch receptor feedback and direct stretch of the sinoatrial node[45,61–63]. Among all the mechanisms, rhythmic changes in cardiac parasympathetic activity are of primary importance for the generation of RespHRV, as evidenced by near-complete loss of RespHRV following muscarinic receptor blockade, vagotomy, heart transplantation or brainstem death[2,3,12,14,21,22,64,65].

Fig. 2 | Assessment and quantification of RespHRV applied to representative physiological and pathological conditions. a, Quantification of respiratory heart rate variability (RespHRV), measured in beats per minute (bpm), across four schematized human

conditions: quiet breathing at 12 breaths per minute; voluntary slow, deep breathing at 6 breaths per minute; quiet breathing plus oxytocin to represent a condition in which RespHRV is amplified independently of changes in respiratory activity, as shown in humans (such as during parent–infant bonding)[66,67] and in animals (such as during recovery from stress)[15]; and heart failure, when RespHRV is largely absent. The quantification of RespHRV, as a variation of heart rate (in bpm) in phase with breathing, enables the assessment of RespHRV within a single respiratory cycle, which can be averaged across cycles. This requires simultaneous measurements of respiratory activity and HR and is available as an open-source Python toolbox[68]. **b**, Quantification of RespHRV in the frequency domain, using heart rate variability (HRV) spectral analysis from the same conditions shown in panel **a**. This type of analysis measures RespHRV as a spectral density. At low frequencies of breathing around 0.1 Hz (6 breaths per minute), as shown for the voluntary slow, deep breathing condition, the RespHRV spectral density merges with other low-frequency oscillatory components in HRV. Spectral analysis of RespHRV involves segmenting the heart rate signal over several minutes (typically 5-min windows in humans), during which RespHRV is averaged. This type of analysis is routinely performed in clinical studies, often using heart rate measurements alone. However, without the simultaneous recording of respiratory activity, it is not possible to accurately identify the RespHRV spectral density, especially if the participant is breathing at a low rate. In the assessment of RespHRV using HRV spectral analysis, it is recommended to simultaneously record respiratory activity (respiratory rate and tidal volume) to determine the frequency (or frequencies) of the RespHRV spectral density (or densities). a.u., arbitrary units.

Box 1 | Expert recommendations

- Avoid using the term ‘arrhythmia’ when referring to an evolutionarily conserved physiological phenomenon that reflects functional coupling between central nervous mechanisms of autonomic cardiovascular and respiratory control.
- Adopt and use the term ‘respiratory heart rate variability’ (RespHRV) instead of ‘respiratory sinus arrhythmia’ (RSA) to describe the respiratory modulation of cardiac pacemaker activity by autonomic mechanisms, manifested as rhythmic changes in heart rate in phase with breathing.
- Use the term RespHRV when referring to respiratory-related variations in heart rate in both the low-frequency and high-frequency bands traditionally defined for heart rate variability, to prevent misinterpretation of the HRV metrics.
- Clarify that RespHRV is not a measure of total cardiac vagal activity or overall vagal tone.
- Promote clinical translation of basic research findings into the mechanisms and functional importance of RespHRV.

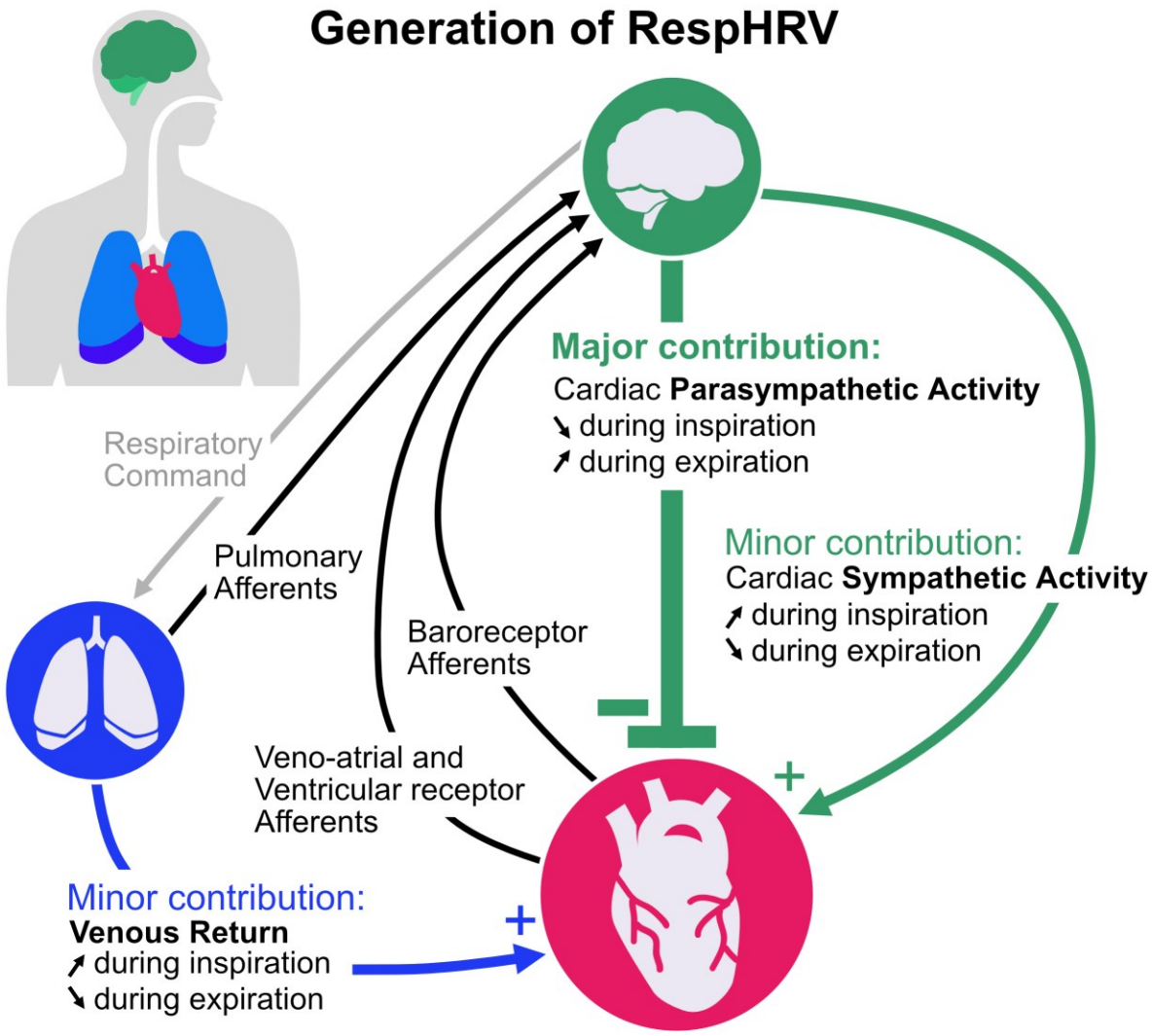
Generation of RespHRV
Major contribution:
Cardiac Parasympathetic Activity
↘ during inspiration
↗ during expiration
Minor contribution:
Cardiac Sympathetic Activity
↗ during inspiration
↘ during expiration
Respiratory Command
Pulmonary Afferents
Baroreceptor Afferents
Veno-atrial and Ventricular receptor Afferents
Minor contribution:
Venous Return
↗ during inspiration
↘ during expiration
+
+

Figure 1

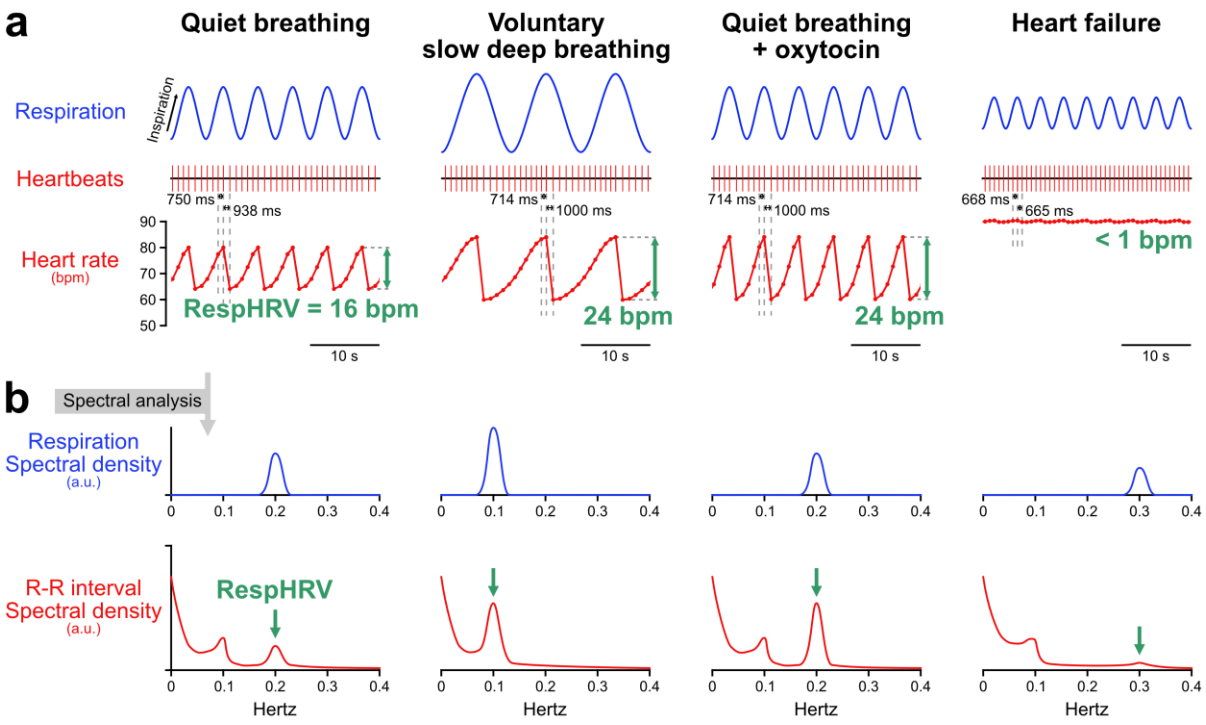
a
Quiet breathing
Voluntary slow deep breathing
Quiet breathing + oxytocin
Heart failure
Respiration
Inspiration
Heartbeats
Heart rate (bpm)
750 ms
938 ms
714 ms
1000 ms
714 ms
1000 ms
668 ms
665 ms
90
80
70
60
50
RespHRV = 16 bpm
24 bpm
24 bpm
< 1 bpm
10 s
10 s
10 s
10 s
b
Spectral analysis
Respiration Spectral density (a.u.)
R-R interval Spectral density (a.u.)
RespHRV
0 0.1 0.2 0.3 0.4
Hertz

Figure 2