

CARDIOVASCULAR AUTONOMIC FLEXIBILITY BEYOND RESTING HEART RATE VARIABILITY - A NARRATIVE REVIEW

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ABSTRACT

Resting heart rate variability (HRV) describes cardiac interval modulation under a particular physiological state; it does not establish how effectively cardiovascular control adapts when that state changes. This narrative review integrates evidence on physical activity and exercise training across HRV, blood pressure variability (BPV), and baroreflex sensitivity (BRS). We use cardiovascular autonomic flexibility to describe stimulus-appropriate adjustment and subsequent recovery of cardiovascular regulation. Exercise interventions often increase resting vagally mediated HRV, whereas evidence for BPV and BRS depends on population, measurement method, and recording timescale. Evidence that these resting adaptations translate into improved challenge responses is less consistent, including randomised evidence of improved fitness without corresponding changes in cardiovascular reactivity or recovery. Interpretation requires distinguishing cardiac vagal modulation from sympathetic vasomotor regulation, spontaneous cardiac BRS from complete baroreflex function, and beat-to-beat BPV from ambulatory or longer-term variability. Neither a larger response nor lower variability is inherently favourable. We propose an assessment framework comprising resting state, response to a standardised challenge, coupling among simultaneously recorded signals, and recovery kinetics. Isometric exercise, postural or central-volume changes, and controlled breathing provide complementary experimental demands but require protocol-specific interpretation. Cardiovascular autonomic flexibility is presented as a research framework rather than a validated biomarker. Its value depends on demonstrating reproducibility, mechanistic specificity, responsiveness to training, and incremental prediction beyond resting measures and conventional cardiovascular risk factors.

KEYWORDS: autonomic nervous system; physical activity; heart rate variability; blood pressure variability; baroreflex sensitivity; exercise.

INTRODUCTION

Regular physical activity is one of the most consistent non-pharmacological determinants of cardiovascular health, with benefits extending from resting blood pressure and lipid metabolism to neural control of the circulation. Exercise training can increase resting vagally mediated HRV and improve cardiac baroreflex sensitivity in selected populations.^[1,2,3,4,5] These effects do not establish a uniform reduction in sympathetic activity or improvement in every component of cardiovascular regulation. These observations have driven widespread use of HRV as a non-invasive marker of cardiovascular autonomic health.

A predominantly resting-HRV view, however, is physiologically incomplete. The autonomic nervous system does not maintain cardiovascular variables at fixed set points; it continuously adjusts cardiac chronotropy, contractility, vascular resistance, venous capacitance, and reflex gain as posture, respiration, exercise, thermal load, emotion, and circulating volume change. A healthy response to exercise may require rapid vagal withdrawal and baroreflex resetting, even though higher vagal modulation is favourable at rest. Conversely, high resting HRV does not guarantee an appropriate response to physiological stress, and a small fall in HRV during a challenge may reflect either stability or impaired recruitment, depending on the stimulus and the subsequent recovery trajectory.

This distinction motivates separating **resting autonomic status**, which is the pattern of measured modulation under defined resting conditions, from autonomic flexibility, i.e., the capacity to produce an appropriately directed, proportionate, coordinated, and reversible response when conditions change. In cardiovascular physiology, this response is distributed across at least three measurable domains: HRV (sinoatrial timing modulation), BPV (the integrated neural, vascular, humoral, respiratory, and mechanical influences on arterial pressure), and BRS (dynamic coupling between arterial pressure and cardiac-interval responses).^[6,7,8,9]

This narrative review has four aims: (1) to distinguish resting autonomic status from dynamic autonomic flexibility; (2) to synthesize the effects of physical activity and exercise training across HRV, BPV, and BRS; (3) to explain how physiological challenge and recovery testing add information beyond resting recordings; and (4) to propose a standardized framework for future research and eventual clinical translation.

Review approach and scope

This is a focused narrative synthesis of exercise, cardiovascular variability, and baroreflex physiology. The initial set of literature was the bibliography of the source manuscript. During revision, targeted web searches using article titles, author names, and identifiers were used to retrieve selected PubMed records and publisher pages. The checks focused on exercise meta-analyses, challenge-and-recovery experiments, BPV studies, and sources that inform physiological interpretation. This process was a targeted appraisal of the existing literature, not an exhaustive database search or a systematic update using a defined publication cutoff.

The synthesis distinguishes between intervention evidence and cross-sectional associations, and separates established measurement principles from the proposed flexibility framework. Published meta-analyses inform the discussion of resting adaptation; individual human studies inform stimulus-specific responses. No new pooled estimate, duplicate study screening, formal risk-of-bias assessment, or certainty grading was undertaken. The principles of clear aims, transparent source selection, and reasoned interpretation are consistent with SANRA's purposes, although no formal SANRA score is claimed.^[10]

From resting autonomic status to cardiovascular autonomic flexibility

Defining the construct

Cardiovascular autonomic flexibility is operationally defined as the capacity of the cardiovascular control system to: (1) maintain an appropriate resting operating state; (2) generate a stimulus-congruent change in cardiac and vascular autonomic modulation; (3) reset reflex control without losing effective blood pressure regulation; (4) coordinate heart rate, arterial pressure, respiration, and vascular resistance; and (5) return

efficiently toward the pre-challenge state once the stimulus ends.

This definition is deliberately directional rather than a claim that "more variability is better." During static exercise, a fall in RMSSD and spontaneous cardiac BRS may accompany an appropriate response. Vagal withdrawal and baroreflex resetting accommodate higher heart rate and pressure; resetting does not, by itself, require a reduction in maximal reflex gain. During cardiopulmonary baroreceptor loading or slow breathing, an increase in vagally mediated oscillation or BRS may be appropriate. During recovery, delayed vagal reactivation may indicate incomplete restoration even when resting values obtained on a separate day are normal.^[11] Flexibility is therefore the ability to vary appropriately, not the tendency to vary more.

Resting capacity and response amplitude

Resting differences influence the apparent amplitude of a challenge response. An individual with a high baseline RMSSD has more observable room for vagal withdrawal than one whose baseline is already low; a larger absolute decrease can therefore coexist with a more favourable resting state. Conversely, a small change in a sedentary participant could reflect a limited measurable range, a weaker effective stimulus, or a different operating state. The change alone cannot identify impaired adaptation. This reserve-and-response interpretation parallels other physiological systems in which baseline capacity and recruited response must be evaluated jointly.

Change scores can, however, be distorted by regression to the mean, mathematical coupling to baseline, and heart-rate dependence. Dynamic studies should therefore report baseline values, condition-specific estimates, absolute and relative changes, and group-by-condition interactions, rather than relying on within-group significance alone. Mixed-effects models are well suited to this purpose, as they retain repeated observations, estimate differential trajectories, and account for within-person dependence.

Evidence that challenges the training hypothesis

Improved aerobic fitness does not necessarily produce altered autonomic reactivity. In a randomised trial of 149 healthy sedentary adults, Sloan and colleagues compared 12 weeks of aerobic and strength training. Aerobic capacity improved selectively with aerobic training, but no between-group differences in heart rate, R-R variability, or blood pressure responses to psychological and orthostatic challenges were observed.^[12] This result limits any claim that exercise reliably improves dynamic cardiovascular regulation. A useful flexibility framework must accommodate null findings and specify which response, challenge, population, and training exposure are expected to change.

Heart rate variability

Short-term HRV remains the best-established non-invasive window into cardiac autonomic modulation. RMSSD and SD1 predominantly reflect short-term, vagally mediated beat-to-beat variability, whereas SDNN over a five-minute epoch reflects overall variability within that recording. High-frequency (HF) power is strongly influenced by respiratory sinus arrhythmia and is interpretable as vagally mediated only when breathing frequency and tidal pattern are taken into account.^[6,13]

Low-frequency (LF) HRV is not a direct measure of cardiac sympathetic activity; it contains baroreflex-mediated and parasympathetic contributions, and its meaning varies with posture, respiration, recording duration, and normalisation. The LF/HF ratio should not be presented as a quantitative measure of "sympathovagal balance."^[14] Normalised units are compositional — an increase in HF normalised power can occur because LF falls, even when absolute HF power does not rise. Reviews of exercise training should therefore prioritise RMSSD, SDNN, absolute HF power, and underlying heart rate, treating normalised spectral indices as contextual rather than decisive.

Exercise interventions generally improve resting HRV, though the magnitude and consistency depend on population and protocol. A 2024 meta-analysis of 16 randomised trials in healthy adults reported increases in SDNN, RMSSD, and absolute HF power, moderated by age, sex, and exercise type.^[1] A separate 2024 synthesis of adults with cardiovascular disease reported beneficial effects on HRV but focused on a clinical population rather than healthy adults generally.^[2] A 2025 synthesis of 34 studies also reported changes in HRV, including a lower LF/HF ratio.^[3] This ratio should not be used to infer restoration of autonomic balance, and differences in subgroup significance do not, by themselves, establish effect modification. These findings support training-related enhancement of resting cardiac autonomic modulation but do not, by themselves, establish generalised improvement in dynamic cardiovascular control.

Blood pressure variability

Beat-to-beat variability over minutes, ambulatory variability across a day, day-to-day home variability, and visit-to-visit variability arise from overlapping but distinct mechanisms.^[7,15] Very short-term BPV reflects respiratory mechanics, stroke-volume fluctuations, arterial and cardiopulmonary reflexes, sympathetic vasomotor rhythms, vascular compliance, and measurement characteristics. Low-frequency arterial pressure oscillations are associated with vasomotor modulation and Mayer waves, whereas high-frequency pressure oscillations are substantially influenced by respiration and its mechanical effects on venous return and stroke volume.^[16,17,18]

BPV should therefore be interpreted in relation to its recording timescale and physiological context. Higher ambulatory and visit-to-visit variability have been associated with adverse outcomes, but those relationships do not define an optimal beat-to-beat response during exercise.^[7,15] A reduction in short-epoch BPV may reflect stronger buffering, reduced oscillatory input, altered breathing, or measurement effects. It does not alone establish either improved regulation or constrained responsiveness. Mean pressure, symptoms, stimulus intensity, and accompanying hemodynamic changes are necessary for interpretation.

Evidence that physical activity modifies BPV is emerging. A 2023 meta-analysis evaluated 24-hour ambulatory BPV and found reductions in systolic variability after exercise training; its evidence included single-arm studies and a smaller randomised subset.^[19] These findings should not be extrapolated directly to beat-to-beat fluctuations during a five-minute laboratory challenge. In 92 healthy young adults, greater moderate-intensity activity and less time in sedentary bouts exceeding one hour were associated with lower supine beat-to-beat systolic average real variability.^[20] These findings are important but do not yet establish an ideal beat-to-beat BPV phenotype under physiological stress; isometric exercise studies demonstrate protocol-dependent haemodynamic changes.^[21] In a sham-controlled handgrip study, post-exercise systolic BPV differed by sex and time, but no trial-condition effect was detected.^[22] The observed recovery pattern, therefore, cannot be attributed specifically to the active handgrip condition.

Baroreflex sensitivity (BRS)

Cardiac BRS quantifies the change in cardiac interval associated with a change in arterial pressure, usually expressed in ms/mmHg. It is one component of arterial baroreflex function; sympathetic baroreflex sensitivity requires an appropriate measure of sympathetic output. Pharmacological methods remain important mechanistic standards, but spontaneous sequence and spectral methods allow non-invasive assessment at rest and during physiological manoeuvres.^[8,9] The sequence method identifies concordant systolic pressure and R–R interval ramps at a specified lag. Down sequences refer to falling pressure accompanied by shortening R–R intervals, rather than a negative slope by definition. Spectral alpha indices use the square root of the ratio of R–R power to systolic pressure power within a frequency band; coherence and other methodological criteria govern interpretation.^[8,9]

BRS is often dominated by cardiac vagal efferent effects at rest, but should not be described as a pure parasympathetic measure. Its value depends on the baroreflex operating point, arterial mechanics, afferent transduction, central integration, efferent responsiveness, sinoatrial transduction, respiration, and the statistical properties of spontaneous pressure fluctuations. Exercise

can reset baroreflex control toward higher pressures while maintaining pressure regulation. Experimental carotid baroreflex work demonstrates that cardiac and vasomotor components need not change together.^[23] A lower spontaneous cardiac gain cannot, therefore, be equated with loss of total baroreflex buffering or used as evidence of resetting without additional measurements.

Training studies generally suggest improved baroreflex function, particularly in older adults and in cardiometabolic disease. Exercise training has improved BRS in older adults with type 2 diabetes and other cardiovascular risk factors^[4,5], and earlier mechanistic work demonstrated training-related changes in baroreceptor control of sympathetic nerve activity.^[24] Studies in older sedentary and trained participants illustrate that observed baroreflex differences depend on the population and protocol.^[25,26] Such findings cannot establish a universal training effect in young healthy adults. Resting BRS should therefore be interpreted alongside challenge-induced resetting and recovery rather than in isolation.

The effect of physical activity on autonomic flexibility

Physical activity may influence autonomic flexibility through several mutually reinforcing mechanisms. Repeated exercise exposes the circulation to transient elevations in heart rate, cardiac output, arterial pressure, ventilation, muscle afferent activity, and thermal stress. Recovery then requires withdrawal of sympathetic drive, vagal reactivation, vascular redistribution, and restoration of pressure control; repeated transitions of this kind may condition not only the resting state but also the kinetics and coordination of the response.

Cardiac adaptations. Training-associated increases in RMSSD and absolute HF power support changes in vagally mediated cardiac modulation under comparable recording conditions.^[1,2,3] They do not directly quantify tonic vagal nerve traffic. Because interval-based variability depends partly on mean heart period, a lower resting heart rate can contribute to between-group differences. Reporting heart rate with HRV and prespecifying sensitivity analyses helps separate these related effects.

Vascular and reflex adaptations. Training studies support changes in cardiac BRS in selected clinical groups and in baroreflex control of sympathetic outflow.^[4,5,24] However, these outcomes assess different

pathways. A change in pressure–interval gain could arise from altered arterial mechanics, afferent processing, central integration, or cardiac responsiveness; spontaneous BRS alone cannot localize the adaptation.^[8,9] Skeletal-muscle metaboreceptor and mechanoreceptor sensitivity, central command, and cardiopulmonary receptor loading further shape how a training-related change in resting tone is expressed during a specific physiological challenge, including isometric exercise, orthostatic stress, and central-volume manipulation.^[26,27,28,29,30]

Coordination and recovery. Because HRV, BPV, and BRS reflect overlapping but non-identical control processes, an accurate picture of flexibility requires examining their coupling rather than any single index in isolation. Discordance across domains is not necessarily an error: for example, a change in a successive-difference BPV metric alongside non-significant changes in individual spectral bands need not be contradictory. The measures weight the signal differently, and non-significance is not evidence of unchanged spectral power, and a rise in alpha-LF BRS can occur when LF pressure power falls more than LF R–R power, because the alpha index is the square root of a power ratio. Reporting the numerator, denominator, and coherence of such indices prevents an apparent increase in "sensitivity" from being misread as generalized enhancement.

Mild head-down tilt and thermoneutral immersion should not be treated as selective switches for parasympathetic activity. Chouchou and colleagues found similar changes in cardiac interval, HF power, and spontaneous BRS during immersion, 6° head-down tilt, and supine control.^[29] A resting control condition is therefore essential. The sympathetic response to head-down tilt also depends on the imposed gravitational and central-volume stimulus.^[30,31]

During slow breathing at near-0.1 Hz, respiratory sinus arrhythmia lies within the conventional LF band rather than the HF band. An increase in LF under those conditions need not indicate sympathetic activation. Respiratory rate should be recorded, and respiratory oscillations should be interpreted at their actual frequency.^[6,13,14] Controlled breathing is itself an intervention and should not be assumed equivalent to spontaneous breathing.

Table 1: Standardised perturbations for assessing cardiovascular autonomic flexibility.

Perturbation	Primary autonomic demand	Information gained
Isometric handgrip/leg exercise	Central command and muscle mechanoreflex and metaboreflex activation	Vagal withdrawal, pressor response, baroreflex resetting under load
Active stand / head-up tilt	Orthostatic reflex compensation for reduced venous return	Sympathetic activation, vagal withdrawal, BRS resetting, and recovery speed
Head-down tilt / water immersion	Central blood volume redistribution and cardiopulmonary receptor loading	Assess loading responses against supine control; responses depend on angle, duration, and

		immersion temperature
Controlled/slow breathing	Respiratory-gated vagal modulation	Respiratory–cardiac coupling; slow breathing may move respiratory power into LF, precluding a fixed HF interpretation
Post-exercise recovery	Withdrawal of exercise-related sympathetic drive and reactivation of vagal tone	Recovery kinetics of HRV, BPV, and BRS as an index of restorative capacity

Expected responses depend on the comparison condition and stimulus dose. Slow breathing and passive postural changes are not selective measures of a single autonomic pathway.

A proposed framework for future studies

A flexibility-oriented study design should capture four complementary components: resting state, challenge response, cross-signal coupling, and recovery kinetics (Figure 1). The primary endpoint should be specified before analysis. Candidate endpoints include the group-by-condition interaction for log-RMSSD, the joint trajectory of RMSSD and sequence BRS, time to recovery of vagal indices, or a multivariate latent response derived jointly from HRV, BPV, and BRS. A single composite "flexibility score" is premature until directionality, reliability, and prognostic value have been externally validated.

The challenge dose should be defined before interpreting group differences. Handgrip studies should report relative intensity, absolute force, contraction duration, fatigue, and whether breath-holding occurred. Equal percentages of maximal voluntary contraction do not necessarily impose equal metabolic stress. Postural studies should specify starting posture, tilt angle, duration, stabilization, and condition order.

Counterbalancing and adequate recovery reduce order and carryover effects.

Signal transitions also need separate treatment from steady epochs. Conventional spectral analysis assumes sufficient stationarity, so pooling stimulus onset and steady challenge into one window can obscure the very dynamics under investigation.^[6] Recovery should be sampled at prespecified times, with physiological and statistical reasons for the chosen duration. There is no universally optimal return time: temperature, ventilation, metabolites, and the preceding exercise dose can sustain a normal post-exercise response.^[11]

For inference, group differences require a direct interaction or contrast, not a comparison of separate within-group *P* values. Log-transformed outcomes should be back-transformed and labelled consistently with the model. Missing observations, failed BRS sequences, and inadequate spectral coherence should be reported rather than silently discarded. Multivariable adjustment for heart rate or respiration should be justified because these variables may participate in the response pathway. Sensitivity analyses can distinguish alternative interpretations without treating one adjustment strategy as definitive.

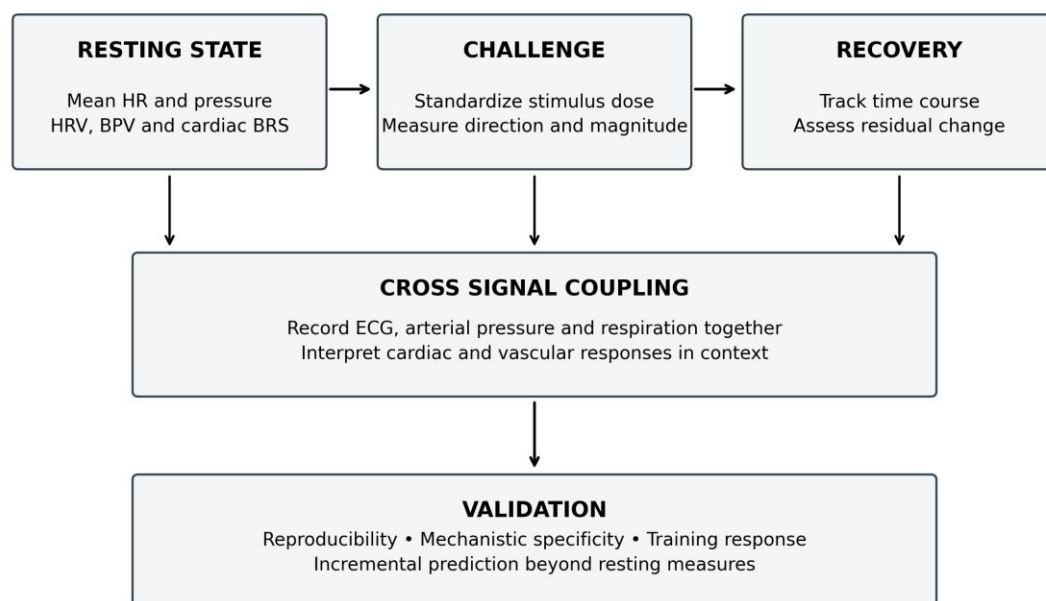


Figure 1 Cardiovascular autonomic flexibility as a testable framework.

Figure legend. Resting status, a standardized challenge, and recovery are interpreted together with coupling among simultaneously recorded cardiovascular and respiratory signals. A response is judged against the physiological demand, not its amplitude alone. Cross-signal coupling spans all phases rather than constituting a separate time epoch. Clinical use requires validation of reliability, mechanism, training responsiveness, and incremental predictive value. This original conceptual schematic contains no participant data, numerical thresholds, or validated diagnostic classifications.

Clinical relevance

A challenge-based framework may clarify why individuals with similar resting heart rate or HRV tolerate physiological stress differently. Potential applications include.

- **Hypertension prevention:** identifying impaired vascular–baroreflex adaptation before sustained hypertension develops.
- **Ageing and diabetes:** testing whether documented improvements in resting BRS translate into better challenge responses and recovery.^[4,5]
- **Cardiac rehabilitation:** distinguishing improvement in baseline tone from improvement in stress reactivity and recovery.
- **Autonomic disorders:** characterising whether symptoms arise from low resting capacity, inappropriate response direction, excessive response, or slow recovery.
- **Exercise prescription:** using recovery kinetics and stimulus-specific responses to individualise training intensity and load.
- **Deconditioning research (bed rest, immobilisation, spaceflight analogues):** tracking deterioration and restoration of cardiovascular control during deconditioning and countermeasure programmes.

Clinical translation requires caution. Short-term variability measures are sensitive to breathing, posture, device characteristics, signal editing, and analytical decisions. Normative values obtained under one protocol cannot be transferred uncritically to another, and "greater flexibility" should not be assumed beneficial without reference to the stimulus — excessive pressure reactivity may be maladaptive even when it demonstrates a large dynamic range.

Research gaps

Five gaps are especially important. First, studies disproportionately measure resting HRV, while dynamic beat-to-beat BPV and BRS receive comparatively little attention. Second, physical activity is often self-reported and treated as equivalent to fitness, although the two exposures can produce different associations with autonomic indices. Third, respiration is frequently unmeasured, limiting interpretation of HF-HRV, HF-BPV, and spectral BRS. Fourth, women, older adults, and clinical populations remain underrepresented in

tightly controlled mechanistic protocols, and sex-specific BPV responses have already been reported.^[22] Fifth, few studies connect laboratory response trajectories to prospective clinical outcomes.

Future work should use repeated standardized challenges, objective activity and fitness measures, and multilevel models of baseline, reactivity, and recovery. Reliability studies are needed to determine which response indices are stable enough for individual phenotyping. Prospective cohorts should test whether impaired autonomic flexibility predicts hypertension, orthostatic intolerance, exercise intolerance, cardiovascular events, or training response, beyond what resting HRV and conventional risk factors already predict.

Limitations

As a narrative rather than systematic review, study selection was not exhaustive and is subject to the authors' interpretive priorities; no formal risk-of-bias or certainty-of-evidence grading (e.g., GRADE) was applied, and the targeted source checks do not constitute a comprehensive literature update. The review synthesises evidence across substantially heterogeneous populations, protocols, and analytic conventions, which limits direct comparison across the domains discussed. The cardiovascular autonomic flexibility construct proposed here is conceptual and has not itself been empirically validated as a predictive biomarker; it is offered as a framework to guide future controlled studies, not as a ready clinical tool.

CONCLUSION

Resting HRV, BPV, and cardiac BRS describe related but non-equivalent aspects of cardiovascular control. Exercise can modify these measures, yet evidence for improved resting modulation is stronger than evidence for a general improvement in challenge reactivity and recovery. Cardiovascular autonomic flexibility provides a framework for testing that distinction through simultaneous signal recording across rest, a defined stimulus, and recovery. Its central criterion is appropriate regulation for the imposed demand, rather than a predetermined increase or decrease in variability. Reliability studies and controlled training interventions should precede clinical scoring, and prospective work must establish whether response trajectories add useful information beyond resting measures. Until those requirements are met, the framework should guide research rather than individual diagnosis or treatment.

Declarations

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Data availability: No new participant-level data were generated or analysed. The cited publications constitute the source material.

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