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Editorial

Basic Concepts and Applications of Heart Rate Variability: Recent Advances and Clinical Perspectives

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HRV: THE PHYSIOLOGICAL SIGNAL

The sinus node is continuously influenced by vagal and sympathetic efferents. Rapid vagal effects account for much beat-to-beat fluctuation, especially the respiratory sinus arrhythmia in which heart rate rises during inspiration and falls during expiration. Baroreflex feedback, vascular and respiratory oscillations, circulating hormones, temperature and intrinsic pacemaker properties add further variation. Consequently, Heart Rate Variability (HRV) is an output of an interacting cardiovascular control system, not a direct recording of autonomic nerve traffic. A low value can reflect altered modulation, but its cause cannot be identified from HRV alone. Pal's account of autonomic control and cardiovascular reflexes provides a useful physiological starting point for interpreting these responses in context.[1]

The distinction between tonic autonomic activity and oscillatory modulation matters. Resting heart rate may fall after an intervention while a particular HRV index changes little; conversely, a change in breathing can markedly alter respiratory HRV without proving a corresponding change in baseline vagal outflow. Mean heart rate, age, posture, respiratory frequency and depth, sleep, physical fitness, medication and disease influence the estimates. A comparison between patients, or between visits in one patient, must therefore account for these conditions. Recent guidance explicitly rejects the conventional interpretation of the Low-Frequency/High-Frequency (LF/HF) ratio as a quantitative measure of sympathovagal balance.[2,3]

What should be measured?

The starting material is a series of Normal-to-Normal (NN) intervals, usually derived from a quality-controlled Electrocardiogram (ECG). Ectopic beats, atrial fibrillation, pacing, missed detections and movement artifacts disrupt the series and require explicit handling. Time-domain measures are readily understood: SDNN is the standard deviation of NN intervals and summarizes overall variability over a specified recording period; RMSSD is the root mean square of successive differences and reflects relatively fast beat-to-beat modulation. These are not interchangeable. In particular, SDNN from a 5-min recording should not be compared directly with SDNN from a 24-hr Holter trace because the latter incorporates slower daily rhythms and changing activities.[2,3]

Frequency-domain analysis partitions fluctuations by oscillation rate. In conventional short recordings, High-Frequency (HF) power spans 0.15-0.40 Hz and commonly tracks respiration-related cardiac vagal modulation; Low-Frequency (LF) power spans 0.04-0.15 Hz and includes baroreflex-related and other influences. LF power is not a pure marker of sympathetic activity. Very-low-frequency power in a brief recording has less secure physiological interpretation. Recording duration, stationarity, ventilation and analysis choices should accompany every reported spectral value. Slow breathing may shift respiratory oscillations from the HF band into LF, producing an apparent LF rise without the physiological change often assumed.[2,3] Nonlinear methods, including Poincaré plots, entropy and detrended fluctuation analysis, examine patterns that mean and spectral power cannot fully describe. They can reveal altered organization of heartbeats in disease and may improve prediction in selected datasets. Their attraction should not obscure the need for adequate record length, reproducible preprocessing, transparent model development and comparison with simple clinical predictors. A complex metric gains clinical value only when it changes a decision or improves an outcome, not merely when it separates two retrospective groups.[3]

Where HRV helps clinically

The most established prognostic example is the association of reduced HRV after myocardial infarction with increased mortality. Landmark Holter studies demonstrated this relation, but modern reperfusion, pharmacotherapy and device treatment have changed the population in which a new prediction rule must work. HRV can add information about risk; an isolated low result should neither trigger an implantable defibrillator nor override ventricular function, rhythm findings and contemporary guideline-based assessment. In heart failure, diminished variability has also been linked to adverse outcomes, while congestion, arrhythmias, beta-blockers and

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pacing complicate interpretation. Serial HRV may be informative in a defined research or monitoring protocol, but routine treatment adjustment on that basis awaits stronger evidence.[4]

In diabetes, reduced heart-rate responses to deep breathing and other cardiovascular reflex tests can reveal cardiovascular autonomic neuropathy before overt resting tachycardia or orthostatic hypotension. The Toronto consensus places HRV within a structured assessment that includes heart-rate responses to standing and Valsalva manoeuvre and blood-pressure responses to standing. A reduced resting RMSSD alone does not establish diabetic autonomic neuropathy. Symptoms, glycaemic history, medications, competing causes of neuropathy and age-appropriate interpretation remain essential. The same principle applies in hypertension and obesity: altered HRV can characterize autonomic physiology or track change in a cohort, but it is not an individual diagnostic label.[5]

HRV is also being studied in critical care, perioperative monitoring, sleep disorders, mental health, exercise training and rehabilitation. These settings differ substantially in physiology and measurement conditions. For example, sedation, ventilation and inflammation may dominate an intensive-care signal; exercise and recovery change heart rate and respiratory pattern; sleep-stage distribution affects an overnight estimate. Findings from one setting cannot simply be transferred to another. An editorial priority for physiology journals is to report the context and mechanism alongside the association, and to distinguish a plausible biomarker from a validated clinical test.[3,6]

Intervention studies deserve similar care. Aerobic training, paced breathing and some stress-reduction programmes can alter HRV, but the measured change depends on breathing rate, fitness, treatment adherence and the time between the intervention and recording. A larger HF peak during slow, guided breathing does not establish a persistent increase in autonomic health; the breathing manoeuvre itself changes the spectrum. Trials should therefore measure participants under a common resting protocol in addition to any task-specific recording, and report clinically relevant outcomes independently of HRV. If an intervention improves symptoms, exercise capacity or blood pressure, HRV may help explain a mechanism. If only HRV changes, a patient benefit cannot be assumed. This distinction is especially pertinent when autonomic measures are proposed as endpoints for lifestyle or mind-body interventions.[2,3]

Recent advances and their limits

Wearable sensors now enable repeated measurements outside the laboratory. Chest devices with ECG electrodes can acquire electrical RR intervals; watches and rings often infer pulse intervals from Photoplethysmography (PPG). The latter produces pulse-rate variability, a useful but imperfect surrogate for ECG-derived HRV because vascular pulse transit, peripheral perfusion, motion and beat-detection algorithms influence the timing. Agreement may be acceptable at rest yet deteriorate during movement or arrhythmia. A personalized baseline collected at the same time of day and under comparable conditions may be more meaningful than a manufacturer's population score. Device-specific validation remains necessary, particularly in older adults and people with cardiovascular disease.[6,7]

Signal-processing advances can identify ectopic beats, quantify uncertainty, handle noisy segments and integrate HRV with activity, respiration and sleep. Machine learning can combine such longitudinal information with clinical data to estimate risk or recognize physiological transitions. However, algorithms trained on a convenient cohort can learn artifact, medication patterns or setting-specific behaviour instead of biology. Independent external validation, prespecified outcomes, calibration, explainability where feasible and assessment across sex, age and patient groups are needed before deployment. A 2026 European Society of Cardiology statement on digital biomarkers emphasizes technical and clinical validation, interoperability, privacy and equitable access. These requirements are part of measurement science, not obstacles to be addressed after an algorithm is marketed.[3,6]

A practical minimum standard follows from these lessons. State whether intervals came from ECG or PPG; specify recording length, time of day, position, breathing instructions and sampling method. Describe rhythm screening, exclusions, artifact correction and the fraction of data removed. Report heart rate alongside HRV, select metrics appropriate to the recording duration, and present absolute measures rather than relying on LF/HF. For longitudinal work, repeat measurements under similar conditions and ask whether the observed change exceeds within-person variation. For clinical prediction, compare an HRV-enhanced model with established predictors and show that its use improves decisions. These steps make findings reproducible and prevent a physiologically attractive signal from becoming an overinterpreted number.[2,3,7]

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CONCLUSION

HRV remains valuable precisely because it connects elementary cardiovascular physiology with changing human environments and disease. Its clearest clinical roles are as a component of autonomic testing and as a contextual prognostic signal in selected cardiovascular populations. Wearables and computational methods can extend observation from minutes to months, but their contribution will depend on signal quality, physiological restraint and prospective proof of clinical utility. The next advance is likely to come less from a new index than from asking a sharper question: whose variability is being measured, under what conditions, and what action should follow?

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