Heart rate

Heart rate is the speed of the heartbeat measured by the number of contractions of the heart per minute (bpm). The heart rate can vary according to the body's physical needs, including the need to absorb oxygen and excrete carbon dioxide. It is usually equal or close to the pulse measured at any peripheral point. Activities that can provoke change include physical exercise, sleep, anxiety, stress, illness, and ingestion of drugs.

The normal resting adult human heart rate ranges from 60–100 bpm.

Tachycardia is a fast heart rate, defined as above 100 bpm at rest.

Bradycardia is a slow heart rate, defined as below 60 bpm at rest. During sleep a slow heartbeat with rates around 40–50 bpm is common and is considered normal. When the heart is not beating in a regular pattern, this is referred to as an arrhythmia. These abnormalities of heart rate sometimes indicate disease.

The vagus nerve is a cranial nerve that plays a significant role in the parasympathetic nervous system, which is responsible for regulating various bodily functions, including heart rate.

Reduced heart rate (HR) during vagus nerve stimulation (VNS) is associated with therapy for heart failure, but stimulation frequency and amplitude are limited by patient tolerance. An understanding of physiological responses to parameter adjustments would allow differential control of therapeutic and side effects. To investigate selective modulation of the physiological responses to VNS, Huffman et al. quantified the effects and interactions of parameter selection on two physiological outcomes: one related to therapy (reduced HR) and one related to side effects (laryngeal muscle EMG).

They applied a broad range of stimulation parameters (mean pulse rates (MPR), intra-burst frequencies, and amplitudes) to the vagus nerve of anesthetized mice. They leveraged the in vivo recordings to parameterize and validate computational models of HR and laryngeal muscle activity across amplitudes and temporal patterns of VNS. We constructed a finite element model of excitation of fibers within the mouse cervical vagus nerve.

HR decreased with increased amplitude, increased MPR, and decreased intra-burst frequency. EMG increased with increased MPR. Preferential HR effects over laryngeal EMG effects required combined adjustments of amplitude and MPR. The model of HR responses highlighted contributions of ganglionic filtering to VNS-evoked changes in HR at high stimulation frequencies. Overlap in activation thresholds between small and large modeled fibers was consistent with the overlap in dynamic ranges of related physiological measures (HR and EMG).

The present study provides insights into physiological responses to VNS required for informed parameter adjustment to modulate selectively therapeutic effects and side effects [1)].

Autonomic impairment, as measured by heart rate variability and baroreflex sensitivity, is significantly associated with increased mortality after traumatic brain injury. These effects, though partially interlinked, seem to be independent of age, trauma severity, intracranial pressure, or

autoregulatory status, and thus represent a discrete phenomenon in the pathophysiology of traumatic brain injury. Continuous measurements of heart rate variability and baroreflex sensitivity in the neuromonitoring setting of severe traumatic brain injury may carry novel pathophysiological and predictive information [2].

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2)
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