Microneurographic recordings of human sympathetic nerve activity (SNA) have provided considerable information on reflex cardiovascular control in health and disease. Such measures have led to emerging hypotheses linking hyperadrenergic activity with cardiovascular stiffening and pathology. However, the mechanistic basis of this linkage is difficult to assess in humans because of the nature of the SNA signal and the complexities in neurovascular interactions. The SNA neurogram indicates a bursty pattern of postganglionic neural activation with variations in the rate and size of the integrated bursts. This burstyness appears to convey important information to the end organ as well as reflect important input information from multiple sensory sites. In humans, methods used to interpret the SNA neurogram generally fall into assessment of the SNA signal itself, or of the action of SNA patterns on the end organ, such as a change in vascular resistance. Yet, the methods used to quantify SNA can affect the interpretation of either reflexive inputs to the autonomic nervous system, and/or neurovascular interactions. Moreover, several circumstances can be discussed where SNA appears to have minimal impact on vasomotor control. Multiple neurotransmitters from sympathetic nerve endings, each with acute and chronic post-junctional actions and subject to local effects on bioavailability, further complicate studies into the linkage between SNA and end organ function. This lecture will discuss these challenges in the interpretation of SNA in humans. First, the nature and reflex specificity of SNA bursty behavior will be presented. These will be discussed within the context of the current differential release hypothesis that suggests SNA discharge patterns affect the release patterns of the multiple sympathetic co-transmitters. This will be followed by a discussion about SNA analysis methods and how these can lead to differing and confusing interpretations about sympathetic neurovascular control. Examples of analyses challenges include spectral analysis approaches and, in the integrated neurogram, whether or not focus should include only burst rate or burst area. Third, the impact of experimental design on SNA control will be addressed. This issue will raise important questions about the complexity of this controlled system and whether or not experimental methods interfere with normal physiology. For example, whether normal ventilation is allowed during the measures affects SNA discharge. The lecture will close with a brief discussion of new methodological approaches that are being developed to expose greater information from the sympathetic neurogram and for improved understanding of neurovascular interactions.

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