4. EFFECTS OF AUTONOMIC TONE ON TACHYCARDIAS

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The pioneering work of Bernard [70B], Gaskell [40G], Langley [12L], Cannon [12C], Herring [76H], and Heymans and Neil [84H, 85H] established that the autonomic nervous system plays a major role in regulating cardiac function, arterial and venous tone, blood pressure, and heart rate [127S]. In the last 30 years, beginning with a classification of adrenergic receptors by Alquist in 1948 [38A], there has been intense research into every facet of autonomic control of cardiovascular function [1A, 118B, 89K, 44V]. This has included exacting anatomic descriptions of afferent and efferent pathways [4A, 91H], central interconnections [87K], cardiac localization of receptors and nerves [3J, 39K], physiologic description and measurement of diverse reflexes [99C, 106D, 47F, 88K, 58L, 92L], measurement of neurotransmitters [3O], the development of selective alpha- and beta-receptor agonists and antagonists [22D, 182W], characterization of the mechanisms of modulation of the release and reuptake of neurotransmitters [24I, 10L, 12V, 5Y], and elucidation of the mechanisms of binding and action of agonists and antagonists on cell receptors [33L, 34L, 35R, 172S, 228S, 36W, 37W]. Interest in the autonomic nervous system has now extended to major issues in clinical cardiology such as hypertension [46F] and cardiac arrhythmias [115C, 96H, 59L, 134L, 13M, 77S, 20Z]. In this chapter we will describe selected human and animal data which illustrate the potential role played by the autonomic nervous system in the diagnosis, onset, modulation, and termination of tachycardias.

Changes in Autonomic Tone Resulting from a Tachycardia

Tachycardias, irrespective of type, alter cardiovascular function because of their effects on cardiac filling and stroke volume [10N]. Blood pressure and pulse pressure decline in direct relationship to the rate, atrioventricular (AV) synchrony, and cardiac function, thereby decreasing the input to the pressure-sensitive baroreceptors. The resultant reduction in afferent nerve traffic to the vasomotor centers raises sympathetic efferent tone and withdraws vagal efferent tone [106D, 89K]. Tachycardias beginning abruptly (i.e., reentrant type) cause the maximum blood pressure fall at their onset, and thus the peak stimulus for sympathetic compensation and vagal withdrawal occurs at this time. While autonomic tone may not

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play a role in initiating a given tachycardia, once the arrhythmia is established, changing neural tone may exert significant effects, depending on the locus of the tachycardia circuit or pacemaker cells and the innervation and responsiveness of these regions to autonomic tone. This is readily illustrated in some cases of paroxysmal supraventricular tachycardia where, after the tachycardia onset, reflex changes in neural tone may cause the arrhythmia to end spontaneously. By monitoring the dynamic effects of such tachycardias on a beat-to-beat basis [63W], one can identify the mechanisms which may lead to spontaneous termination of these tachycardias [56W]. In a recent study, hypotension at the onset of the tachycardias led to augmented sympathetic tone, which accelerated the rate and raised blood pressure above control levels. The elevated blood pressure then increased vagal tone, which caused tachycardia slowing in all and termination in nine out of 20 cases (see Figure 4-1) [56W]. Since most cases of paroxysmal supraventricular tachycardia involve the AV node, a structure richly innervated, one easily observes the dynamic effects of altered neural tone on the tachycardia. The extent of reflex change following the onset of a tachycardia depends on the intensity of the hypotensive stimulus, the integrity of the neural reflexes, and the capacity of the heart and peripheral circulation to respond. For example, in persons with congestive heart failure, tachycardias do not elicit powerful cardiac and circulatory responses to altered autonomic tone as their hearts are catecholamine-depleted [60C], have a reduced beta-receptor density, a reduced level of isoproterenol-stimulated adenylcyclase, a reduced contractile response to isoproterenol [128B], and are poorly responsive to changes in vagal tone [5E]. Reflex alterations in autonomic tone following the onset of a tachycardia may transform the arrhythmia. The onset of ventricular fibrillation during atrial fibrillation with rapid conduction over a Wolff-Parkinson-White (WPW) pathway may be secondary to marked elevations in sympathetic tone. Similarly, a reflex increase in sympathetic tone following the onset of ventricular tachycardia may be responsible for its transformation to ventricular fibrillation. In this regard, one notes that ventricular fibrillation when documented is very often preceded by several seconds of organized ventricular tachycardia [21, 76]. Equally, in cases of ventricular tachycardia with adequate hemodynamic compensation, sympathetic tone may fall while vagal tone rises and this may contribute to termination (spontaneous or otherwise) of the tachycardia (see below).

During tachycardias such as paroxysmal supraventricular tachycardia and atrial fibrillation, the atrial pressure rises and activates stretch-sensitive receptors [91L, 262]. These receptors send afferent impulses via the vagus nerves, causing diuresis by inhibiting antidiuretic hormone secretion [35T] and a rise in sympathetic tone [91L]. The diuresis is often an important clue in diagnosing an arrhythmia as being supraventricular [199W] and may cause intravascular volume contraction which can aggravate the hypotension caused by the tachycardia. The increase in sympathetic tone elicited through these receptors may further increase the rate of tachycardia [91L] (see below).

Termination of Paroxysmal Supraventricular Tachycardia

TERMINATION OF PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA BY INCREASED VAGAL TONE

Work dealing with termination of paroxysmal supraventricular tachycardia by the autonomic nervous system has focused on the