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NEUROHUMORAL MECHANISMS OF FUNCTIONAL RECOVERY FOLLOWING CATHETER ABLATION FOR VENTRICULAR ARRHYTHMIAS

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Background. The sympathoadrenal system plays an important role in the development of certain forms of ventricular arrhythmias. By acting on adrenergic receptors in cardiac cells, adrenaline and noradrenaline enhance calcium cycling and may facilitate the generation of premature impulses. Although radiofrequency catheter ablation aims to eliminate the arrhythmogenic focus, subsequent functional recovery may also be associated with changes in neural and hormonal regulation. Clarifying this relationship is important for accurately interpreting the clinical outcomes of the procedure.

Study objective. To elucidate the potential mechanisms and clinical significance of sympathoadrenal changes following radiofrequency catheter ablation for ventricular arrhythmias.

Materials and methods. This abstract was prepared as a narrative review. The relationship between the arrhythmogenic effects of catecholamines, the autonomic response following ablation, and functional recovery was assessed based on clinical and experimental studies. Observations in humans and findings from animal models were interpreted separately.

Results. Activation of beta-adrenergic receptors increases calcium loading in cardiac cells and may cause spontaneous calcium release from intracellular stores. The inward electrical current generated through sodium–calcium exchange can give rise to a delayed afterdepolarization and an additional impulse. This mechanism explains the sensitivity of certain idiopathic ventricular arrhythmias to adrenergic stimulation. Since ablation targets the arrhythmogenic focus, the arrhythmia may decrease even without a reduction in catecholamine levels [1].

In the study by Yu et al., the disappearance of premature ventricular contractions following successful ablation was accompanied by a decrease in the mean 24-hour heart rate and changes in autonomic regulation. However, these functional findings did not constitute direct measurements of adrenaline and noradrenaline secretion [2]. Therefore, clinical improvement cannot be equated with complete normalization of the sympathoadrenal system.

In a study conducted in dogs by Tan et al., left ventricular ejection fraction recovered after the induction of premature ventricular contractions was discontinued, whereas increased sympathetic nerve activity persisted. Although this was not a catheter ablation study, it demonstrates that cardiac contractile function and neural regulation may recover at different rates [3]. Thus, arrhythmia burden, cardiac function, the medication regimen, and follow-up duration should be considered when assessing the sympathoadrenal response following ablation. Direct clinical evidence of sustained normalization of catecholamine levels remains limited.

Conclusion. Clinical recovery following radiofrequency catheter ablation begins with elimination of the arrhythmogenic focus and may be accompanied by changes in neurohumoral regulation. Although reduced sympathoadrenal activity is a potential mechanism of recovery, it has not been demonstrated consistently across all patients. A reduction in arrhythmia burden and improvement in cardiac function do not, in themselves, confirm normalization of catecholamine secretion. When investigating this mechanism, it is important to assess clinical outcomes alongside changes in adrenaline and noradrenaline levels under consistent testing conditions. Repeated follow-

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up assessments help clarify the duration of sympathoadrenal changes and their relationship with functional recovery.

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