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HOW SEX HORMONES AFFECT THE HEART'S CONDUCTING SYSTEM

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The cardiac conduction system is responsible for generating and coordinating the heart's electrical activities. Recent studies have shown that sex hormones particularly estrogen, progesterone, and testosterone–profoundly influence conduction system function through multiple molecular mechanisms (Tse et al., 2021). Women show faster resting rates while men develop more fibrosis-related disorders (Vijayaraman et al., 2023). Significant variations in estrogen and progesterone levels during pregnancy result in detectable alterations in atrioventricular conduction and sinoatrial node function, making pregnancy a special physiological model of hormonal impact (Tan & Tan, 2019).

It is therefore more crucial than ever to comprehend these hormone-conduction interactions in order to create sex-specific pacing and arrhythmia treatment strategies.

The analysis reveals estrogen enhances sinoatrial (SA) node function via estrogen receptor alpha (ERα)-mediated funny current (If) while reducing conduction system fibrosis (Long & Fiset, 2020; Kurokawa et al., 2022). Progesterone prolongs ventricular repolarization through inhibition of the rapid delayed rectifier potassium current (IKr) but may exert protective effects during pregnancy (Odening et al., 2018). Testosterone shortens the QT interval (measured on electrocardiogram) yet promotes fibrotic remodeling of conduction tissue (Bidoggia et al., 2017). Clinically, women show higher resting heart rates and greater susceptibility to long QT syndrome (LQTS) and other arrhythmias, while men develop more progressive conduction system disorders with aging (Yang et al., 2016; Vijayaraman et al., 2023). Pregnancy demonstrates these hormonal influences through characteristic increases in heart rate and occasional atrioventricular (AV) conduction changes (Tan & Tan, 2019). These findings highlight critical clinical considerations. While estrogen appears broadly protective through its effects on SA node automaticity and anti-fibrotic actions, progesterone and testosterone demonstrate complex, context-dependent effects (Bidoggia et al., 2017). The progesterone paradox - being simultaneously arrhythmogenic (through IKr inhibition) and potentially cardioprotective - warrants investigation (Odening et al., 2018; Tan & Tan, 2019). Similarly, testosterone's dual effects on ventricular repolarization (QT shortening) versus conduction system fibrosis necessitate careful clinical consideration (Lester et al., 2021). Current limitations include predominant reliance on animal models and few human intervention trials (Tse et al., 2021). Promising translational approaches include sex-specific cardiac resynchronization therapy (CRT) and targeted hormonal modulation (Wu et al., 2024), though more clinical validation is required (Vijayaraman et al., 2023).

In conclusion Sex hormones critically regulate conduction through multiple mechanisms. Priority research areas include clinical trials of hormonal therapies, mechanistic studies of channel interactions, and sex-specific treatment guidelines.