

Supraventricular tachycardia. ECG diagnosis and anatomy

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Abstract:

This paper reviews the anatomical substrates responsible for the induction and maintenance of supraventricular tachycardia and discusses the ECG findings associated with these tachycardias. The normal anatomy of the supraventricular conducting system, particularly within the atria, is complex with conduction proceeding along preferential pathway, which are in turn determined in part by the anisotropic properties of the atrial myocardium. There appear to be at least dual inputs to the atrioventricular node, a posteriorly situated slow pathway and an anterior fast pathway. It is sometimes possible to relate ECG findings directly to anatomical substrates; for example, in some cases of atrial tachycardia the site of the atrial focus (left or right, superior or inferior) can be determined by the polarity of the P wave. The anatomical substrates responsible for intra-atrial re-entry, atrial flutter and atrial fibrillation relate to anatomical barriers to impulse propagation and areas of slow conduction. In atrial flutter the crista terminalis, Eustachian valve, inferior vena cava, coronary sinus os, and tricuspid annulus have been identified as anatomical barriers to conduction around which a macro re-entrant circuit within the right atrium may conduct, usually in a counter-clockwise direction. Clockwise direction of conduction, and other mechanisms of tachycardia, occur in some of the less typical forms of atrial flutter. Atrial fibrillation is caused by multiple wavelets which randomly conduct through the atrial myocardium and are responsible for the irregular 'fibrillation waves' on the ECG. Supraventricular tachycardia presents as a narrow complex tachycardia unless pre-existing or rate-related bundle branch block is present. Less common causes for a broad complex tachycardia occurring in supraventricular tachycardia include an accessory atrioventricular or atriofascicular pathway conducting antegradely during tachycardia, or accessory pathway participation as a bystander during supraventricular tachycardia. ECG features which can help to distinguish between atrioventricular nodal re-entrant tachycardia and atrioventricular re-entrant tachycardia include: (1) the presence of a delta wave during sinus rhythm which is highly suggestive of atrioventricular re-entrant tachycardia as the mechanism of supraventricular tachycardia; (2) the finding of a pseudo s (lead II) or pseudo r' (lead V1) during tachycardia in atrioventricular nodal re-entrant tachycardia; (3) lengthening of the tachycardia cycle length in cases of atrioventricular re-entrant tachycardia when bundle branch block occurs ipsilateral to the accessory pathway and (4) the finding of QRS alternans during tachycardia which is suggestive of atrioventricular re-entrant tachycardia. 'Long RP' tachycardia may be caused by an atrial tachycardia due to an inferiorly situated area of abnormal automaticity, atypical atrioventricular nodal re-entrant tachycardia with slow retrograde conduction, or atrioventricular re-entrant tachycardia with an accessory pathway conducting slowly from ventricle to atrium during tachycardia.