Supraventricular tachycardia

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Supraventricular tachycardia (SVT) is defined as an abnormally rapid heart rhythm, with a narrow complex (QRS < 120 milliseconds) having an electropathologic substrate, essential for sustaining the arrhythmia, emerging in atrial or atrioventricular nodal tissue. Most types of SVT are triggered by a reentry mechanism that may be induced by premature atrial or ventricular ectopic beats and are classified according to the location of the reentry circuit. The incidence of SVT is about 35 cases per 100 000 population per year, with a prevalence of 2.29 cases per 1000 population.[1] Excluding the atrial fibrillation and atrial flutter as specific individual arrhythmic entities, supraventricular tachycardia is categorized based on the length of PR interval. First line treatment of patients presenting with sustained SVT is usually to slow conduction through the atrioventricular node, rarely, the arrhythmia is poorly tolerated that immediate electrical cardioversion is needed. Atrioventricular node conduction can be slowed by vagal stimulation with carotid sinus massage or the Valsalva maneuver. If vagal maneuvers are unsuccessful then, recommended first line medication in slowing atrioventricular conduction is intravenous administration of adenosine, unless the patient suffers from chronic obstructive pulmonary disease. The effectiveness of intravenous verapamil seems similar to that of adenosine in terminating SVT, with slightly lower rates of adverse effects such as hypotension associated with adenosine. Major therapeutic goal for SVTs should be improvement of patients quality of life according to symptomatology and patient preferences. Long-term treatment should be offered to patients which suffer from recurrent symptomatic episodes, prolonged medical treatment or catheter ablation. Catheter ablation provides a definitive management option for SVT and after the procedure, most patients can return to their normal activities very quickly.

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LITERATURE

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