Atrial fibrillation and atrial flutter are commonly seen in patients with WPW syndrome. In these patients ventricular rates may rise to near 300bpm or above, this can be serious due to the nature of the conducting accessory pathways. Care must be taken to stop the atrial fibrillation and flutter causing the ventricles to fibrillate as this can have life threatening consequences.

Atrial flutter is a very small reentry circuit inside the right atrium at the base near to the AV node. Ablation is again the preferred treatment as antiarrhythmic drugs have little effect.

Atrial fibrillation originates from a very small reentry wave of energy circulating in the great venous structures. The use of ablation to cure fibrillation is difficult and success rates vary.

## DEFINITIONS

Retrograde; the direction of travel of the impulse is from the ventricle to the atrium.

Anterograde; the direction of travel of the impulse is from the atrium to the ventricle.

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SADS UK played a significant role in a campaign to bring about a new chapter in the Department of Health's National Service Framework for Coronary Heart Disease. Chapter Eight, Arrhythmias and Sudden Cardiac Death was published on 4th March 2005.

**The chapter includes the following recommendations for good practice for initial treatment.**

Patients should receive a hard copy of their ecg which shows their arrhythmia & patients surviving cardiac arrest or having presented with pre-excited atrial fibrillation should be assessed by a heart rhythm specialist.

The following patients should be **urgently assessed** by a heart rhythm specialist:-

Patients with either syncope suggesting an arrhythmia, symptoms of arrhythmia, a personal history of structural heart disease, a family history of early sudden death, recurrent syncope with palpitations, 3rd degree AV block, or with ventricular tachycardia.

The following should be **referred** to a heart rhythm specialist:-

People with suspected ventricular tachycardia, patients with WPW Syndrome, recurrent SVT not controlled by medication, recurrent atrial flutter, symptomatic atrial fibrillation not controlled by medication, first degree relatives of victims of sudden cardiac death who died below the age of 40 years, or patients with inexplicable recurrent falls.

**The Recommendations for Ongoing Treatment are:-**

Patients with sustained or compromising arrhythmias receive timely referral for appropriate treatment. Those identified at being high risk or with life-threatening ventricular arrhythmias should be considered a candidate for an implantable cardioverter defibrillator (ICD). For patients with sustained SVT catheter ablation should be considered. An outpatient care plan is devised between patient, GP and arrhythmia care team, when further hospital treatment is not recommended.

**Please make a donation to SADS UK to enable us to support those affected by cardiac arrhythmia and sudden cardiac death –**

**SADS UK, 22 Rowhedge, Brentwood, Essex CM13 2TS**


Grateful thanks to Dr Andrew Grace for helping compile this leaflet.



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Ashley Jolly sad Sudden Adult Death Trust

# SADS UK

**Supporting those affected by Cardiac Arrhythmia**

# The WPW Syndrome

## Supraventricular tachycardia (SVT) and Wolf Parkinson White Syndrome (WPW)

## A Guide for Patients and Health Care Providers


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When the heart is beating normally the beat (electrical impulse) starts from the sinus node which is situated in the right atrium (upper chamber) and passes through the atria, along the His bundle and its branches to activate the ventricles. The atria and the ventricles are electrically insulated from each other except at a single point, the AV (atrioventricular) node. The His bundle passes through the AV node at this point and conducts the electrical signal from the atria to the ventricles. The sinus node may be referred to as the hearts natural pacemaker as it controls the heart rate. As the electrical impulse travels through the bundle branches and over the ventricles the muscle contracts forcing the ventricles to pump the blood around the body including to the lungs and brain.

When extra myocardial tissue is present in the electrically insulated region (atrioventricular ring) between the atria and the ventricles, a short circuit may be caused. This extra tissue is called an accessory pathway and the presence of such tissue changes the electrical flow between the atria and the ventricles. The pathway may pass current in one direction only or in both directions. A pathway can be concealed and its effect on the electrical impulse signal cannot be seen by an ECG machine. A concealed pathway only allows the impulse to travel from the ventricles to the atria, with the WPW syndrome the accessory pathway permits the impulse to travel in both directions.

When an accessory pathway is present the impulse from the sinus node activates the ventricles in the normal way via the His bundle and also more rapidly through the accessory pathway. The faster signal passing through the accessory pathway prematurely activates the ventricular muscle tissue, causing the pre-excited WPW ECG appearance. Any tachyarrhythmia that is initiated by the presence of accessory pathway myocardial tissue breaching the insulating layer (atrioventricular ring) is called Supraventricular tachycardia SVT.

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Supra means above; Tachycardia is a heart beat above 100 beats per minute; the atria chambers are above the ventricular chambers; thus an arrhythmia starting in the atria is termed supraventricular tachycardia.

The type of SVT that a patient has is classified according to the way in which the electrical impulse travels from the atria.

Orthodromic atrioventricular re-entrant tachycardia is the most common supraventricular tachycardia in patients with WPW syndrome. The re-entry circuit starts normally with the impulse from the sinus node, the impulse travels correctly towards the ventricles down the His bundle past the AV node and over the ventricles via the His bundle branches and Purkinje fibres. Due to the presence of the accessory pathway the impulse is able to return (re-entrant) to the atrium via the accessory pathway.

With orthodromic tachycardia the impulse has started normally via the His bundle so ventricular depolarisation (initiation of muscle contraction) is normal. The QRS complexes are thus narrow and a retrograde P wave follows shortly after the QRS complex, a delta wave is not seen.

Antidromic atrioventricular re-entrant tachycardia occurs in a small number of WPW syndrome patients. The impulse travels from atrium to ventricle via the accessory pathway instead of travelling down the His bundle. The impulse then re-enters the atrium travelling the wrong way up the His bundle and through the atrioventricular node to the atrium. The QRS complexes are broad, an exaggeration of the delta wave. Antidromic atrioventricular tachycardia is more common when multiple accessory pathways are present, this arrhythmia can be mistaken to be of ventricular origin rather than originating in the atria.



Symptoms of supraventricular tachycardia can be light-headedness, dizziness, palpitations, dyspnoea, diaphoresis, angina, shortness of breath and possibly chest discomfort. If the patient has symptoms then they need an electrophysiologic study to determine the presence and number of accessory pathways.

The symptoms can be so subtle that the patient is unaware and does not seek treatment. Syncope (fainting/loss of consciousness) is not normally associated with WPW syndrome.

WPW syndrome can be inherited; it is believed that the gene for some patients with WPW is located on chromosome 7.

If the patient has SVT whilst connected to an ECG machine, a distinctive change to the trace will be seen. Where the symptoms occur randomly over the day a 24hr Holter may be used to record the heart trace. Where the occurrence is infrequent a loop recorder should be used. The ECG pattern for WPW syndrome was identified in 1930 by Dr Herold Wolf, Paul White and Sir John Parkinson.

The preferred treatment is to destroy the accessory pathway tissue with a short pulse of radio frequency energy. A probe (catheter) is inserted usually into the femoral vein near the groin or alternatively into a vein near the collarbone and is carefully steered to the atrium. The tip of the probe is guided to the pathway tissue, which is then quickly burned away. These currently used ablation techniques were pioneered in the early 1980s and have been in widespread use over the last decade.

Calcium channel blockers or digitalis should not be used as the only method of treatment, beta-blockers have little or no effect and antiarrhythmic drugs can be a possible risk.

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