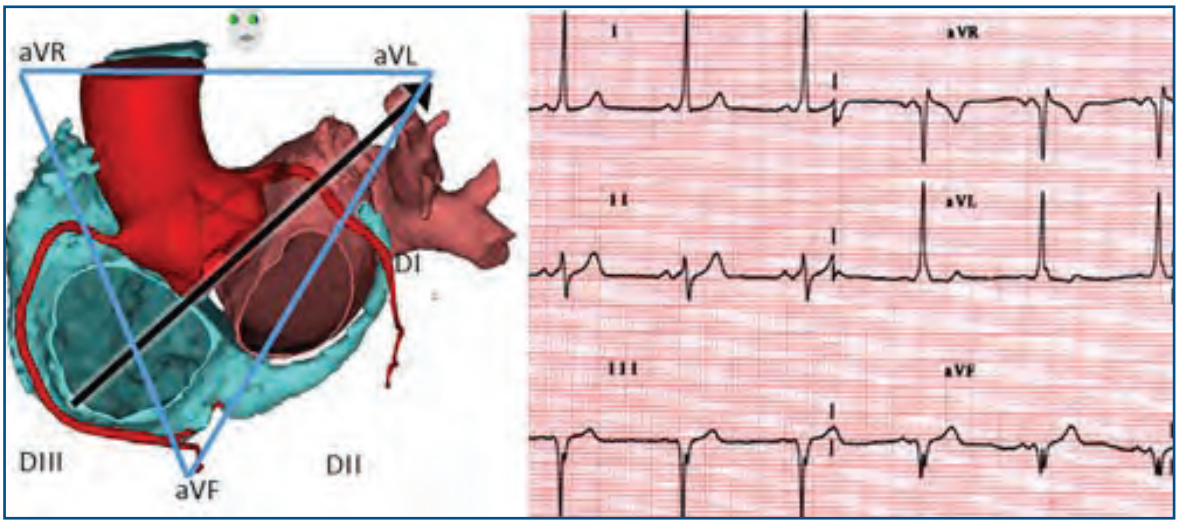
FIORENZO GAITA • CARLO PAPPONE

Ventricular preexcitation
WPW • PJRT • MAHAIM
From ECG to therapy



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CHAPTER 1

CARDIAC PREEXCITATION

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The term "preexcitation" indicates the premature activation of a portion of myocardium by an impulse traveling an anomalous pathway, able to conduct electrical impulses faster than the normal conduction pathways. Usually such anomalous, accessory pathways connect the atria to the ventricles and allow an impulse originated in one chamber (atrium or ventricle) to reach the other without the physiological delay elicited by the atrioventricular node. The accessory pathways are constituted in most cases by fast, sodium-dependent fibers with rapid conduction capacity. Differently, the atrioventricular node is constituted by calcium-dependent fibers with a slow conduction capacity. The term "preexcitation" is not a synonymous of "ventricular preexcitation": in fact, in the presence of an accessory pathway, the atria can be pre-excited by an impulse originating from the ventricles without any effect on the ventricular activation.

NOMENCLATURE AND CLASSIFICATION OF THE DIFFERENT FORMS OF PREEXCITATION

Since the last century numerous anatomical studies (Kent, 1893) have shown the presence of accessory pathways connecting the atrium to the ventricle, but only in 1930 Wolff, Parkinson and White demonstrated for the first time the clinical role of the preexcitation and the possible related arrhythmias. These Authors published a study entitled "Bundle branch block with short PR interval in healthy young people prone to paroxysmal tachycardia". In this article, the fundamental elements of the **"Wolff-Parkinson-White or WPW syndrome** (from the initials of the three Authors)" (short PR interval <0,12 sec, "wide" QRS complex >0,08 sec, initially interpreted as a bundle branch block, and paroxysmal tachycardia) were underlined. These three elements still constitute the most common presentation of the syndrome.

The anatomical basis of the WPW syndrome is the presence of a muscular bundle (named Kent bundle) directly connecting the atria to the ventricles. Later, other forms of preexcitation have been described: for example, the **Lown-Ganong-Levine or LGL syndrome** (Lown *et al.* 1952; Coumel *et al.* 1972; Castellanos *et al.* 1982, Ho, 2008), that is the combination of short PR interval, narrow QRS complex without delta wave (i.e. the initial slowing of the QRS complex corresponding to ventricular preexcitation) and paroxysmal tachycardia. In this case, the "James bundle", an accessory pathway connecting the atrium to the most distal portion of the atrioventricular node, was later proposed as anatomical substrate. At

present, however, the role of the James bundle in the genesis of arrhythmias has not been demonstrated, and the association of short PR interval, normal QRS complex and paroxysmal tachycardias has not been proved. For these reasons the term "LGL syndrome" should not be used and should be replaced by "accelerated atrioventricular nodal conduction".

A third form of preexcitation has been described and attributed to the **Mahaim fibers**, node-ventricular or node-fascicular (rarely fasciculo-ventricular) bundles directly connecting the atrioventricular node to the right ventricular myocardium or to the right bundle branch (Mahaim *et al.* 1970). Given their electrophysiological features of calcium-dependent fibers, characterized by a slow decremental conduction, other accessory pathways connecting the right atrium to the right ventricle identified with these properties, have been named **"pseudo-Mahaim",** due to their electrophysiological affinity to the "true" Mahaim fibers (Haissaguerre *et al*, 1995).

Another subtype of accessory pathways connecting the right ventricle to the right atrium have been reported, characterized by only retrograde slow and decremental conduction. These accessory pathways, described as long, curvy calcium-dependent fibers, usually located in the proximity of the atrioventricular node (Critelli *et al.*, 1984) represent the anatomical substrate for the **Permanent Junctional Reciprocating Tachycardia (PJRT)**, also known as Coumel reciprocating tachycardia), a rare syndrome characterized by incessant tachycardia episodes (Coumel, 1975; Critelli *et al.*, 1985).

CONDUCTION THROUGH THE ACCESSORY PATHWAYS IN WPW

The conduction through the Kent bundle is usually rate-independent and takes place with "all-or-none" modality (Josephson, 1993), i.e. the conduction time is independent from the heart rate and an impulse can be conducted or blocked, but in case of conduction it cannot be slowed through the accessory pathway.

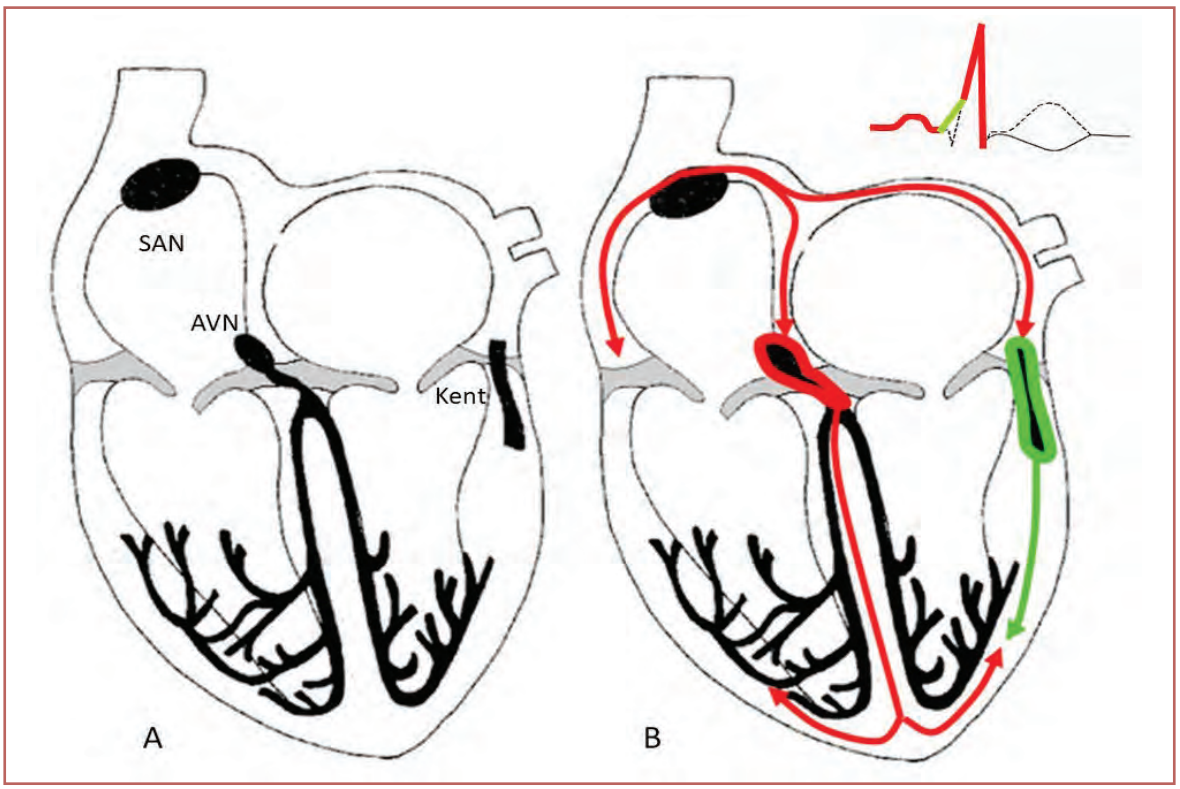

Figure 1.1 • Schematic representation of a Kent bundle (A) and conduction responsible for ventricular pre-excitation (B). SAN: sino-atrial node. AVN: atrio-ventricular node.

THE ELECTROCARDIOGRAM IN THE PRESENCE OF WPW PREEXCITATION

In the presence of a sinus or atrial rhythm, if a portion of the ventricles is pre-excited by an impulse traveling through an accessory pathway, typical electrocardiographic characteristics can be observed, indicating that the supraventricular impulse is conducted through the Kent bundle. In this situation the preexcitation is defined as "manifest" and the following elements can be observed at surface ECG: short PR interval (<0,12 sec), delta wave, wide QRS complex (>0,08 sec), secondary repolarization abnormalities (**Figure 1.1**). A Kent bundle can conduct the impulses in both directions (from the atrium to the ventricle and vice versa). In 20% of the cases, only retrograde conduction is possible, while in 10% of the cases only anterograde conduction is evident. When the conduction is exclusively retrograde, there is no evidence of ventricular preexcitation because the accessory pathway is not able to conduct the impulse from the atria to the ventricles, but an atrial preexcitation in the presence of a ventricular rhythm may occur (Gallagher *et al.* 1976; Sung *et al.* 1976; Tonckin *et al.* 1975; Przybylski *et al.* 1980; Gaita *et al.* 1992 c; Josephson, 1993; Prytovsky, 1990; Wellens *et al.* 1983, 1990 a, 1990 b; Yee *et al.* 1990); in this situation the accessory pathway is defined "concealed". When the accessory pathway has an anterograde conduction capacity, however, the electrocardiographic signs of ventricular preexcitation may sometimes not be present during sinus rhythm: the reason is that the impulse conducted through the normal pathway activates the ventricles before the same impulse reaches the accessory pathway: this phenomenon is most commonly observed when the Kent bundle is located far from the sinus node, as in the case of left lateral pathways. This condition is defined "non-manifest preexcitation" and is characterized by the absence of electrocardiographic signs of preexcitation in a subject with a Kent bundle with an anterograde conduction capability. In these cases, if the atrioventricular nodal conduction is slowed with appropriate maneuvers (for example vagal maneuvers as the carotid sinus stimulation, the use of drugs able to slow the atrioventricular conduction as beta-blockers or adenosine), ventricular preexcitation (delta wave) may become manifest (**Figure 1.2A, 1.2B, 1.2C**).

In some subjects, ventricular preexcitation is intermittent and can disappear for long periods according to the autonomous nervous system activation or can emerge at a regular pace (i.e. bigeminal, that is an alternance between a normal beat and a pre-excitated one; or trigeminal) (Satullo *et al.* 1988).

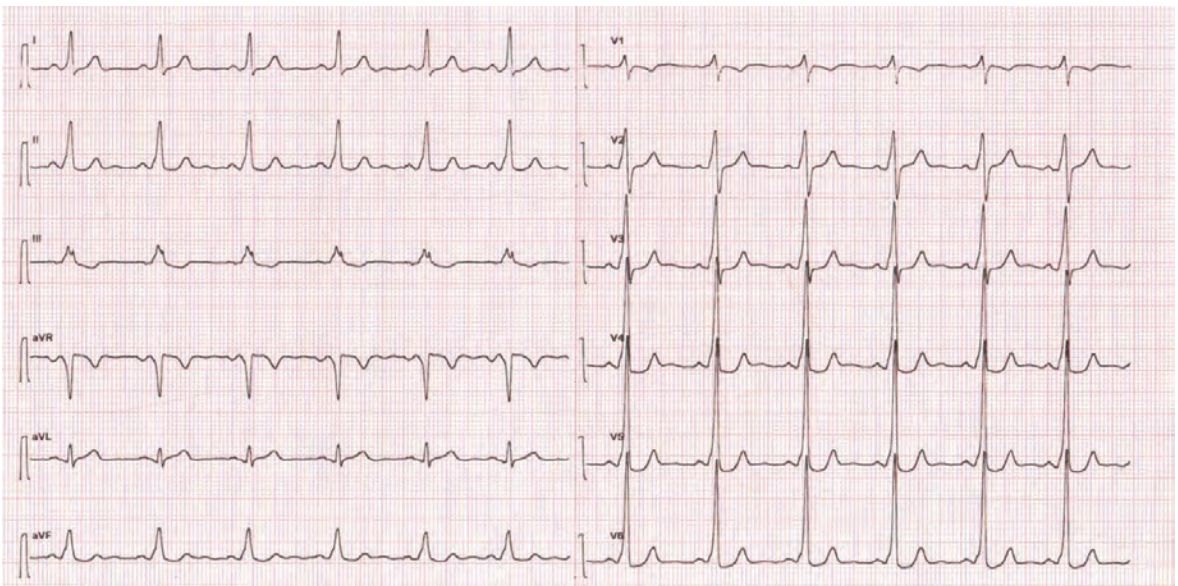

Figure 1.2A • Manifest preexcitation. Delta wave is clearly evident at 12-leads ECG.

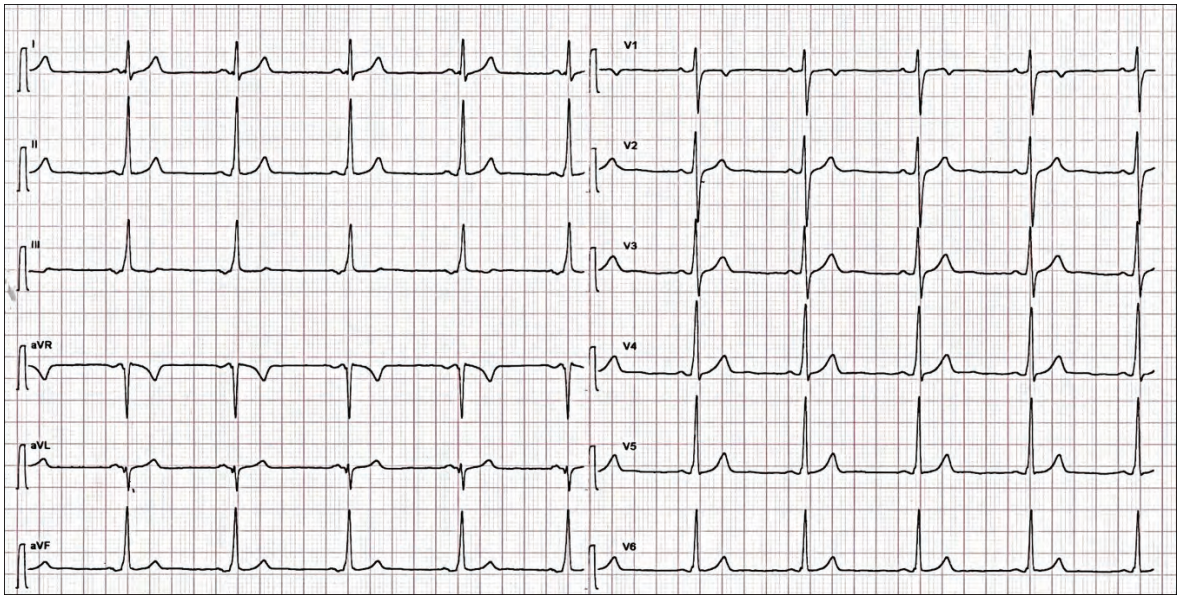

Figure 1.2B • Manifest preexcitation. Delta wave is less pronounced than in Figure 2.2A.

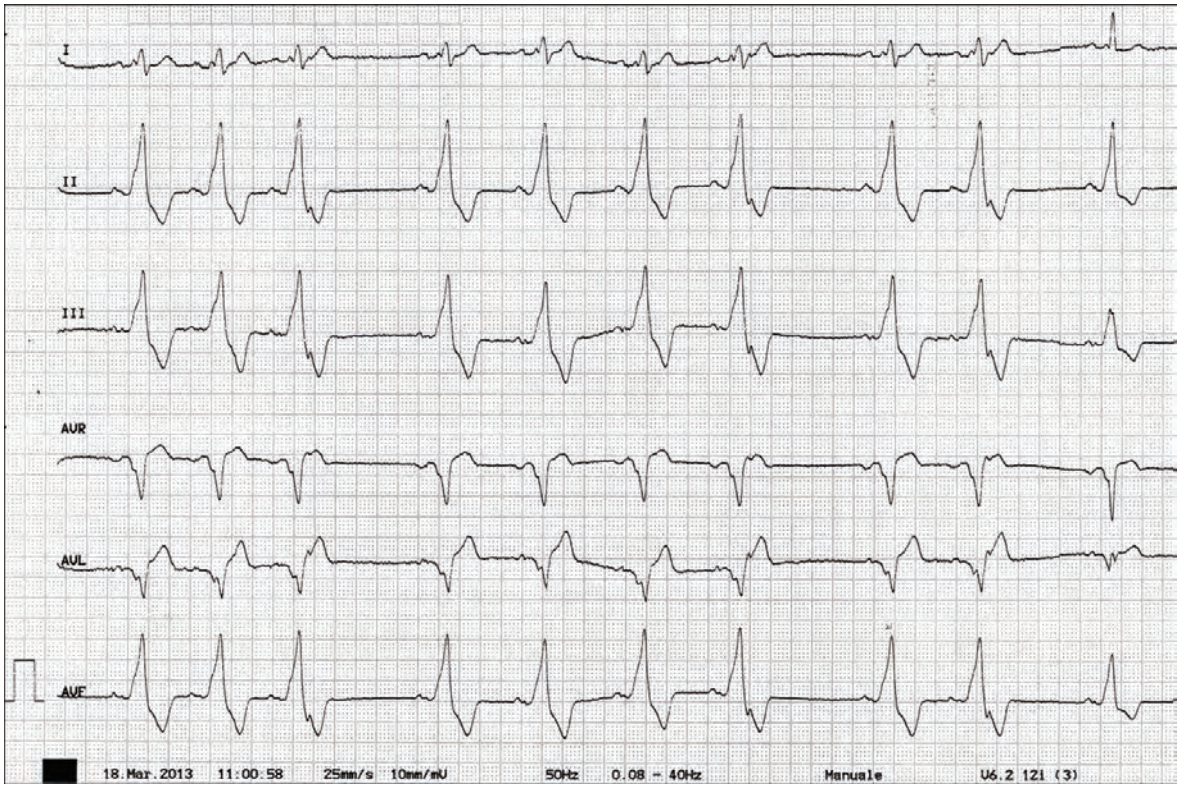

Figure 1.2C • Delta wave unmasking during administration of i.v. adenosine.