1st Virtual Congress of Cardiology

[Scientific Activity - Actividad Científica] [Brief Communications - Temas Libres]

Exceptional Patterns of the Wolff-Parkinson-White Syndrome in Rhythm and AV Nodal Tachycardia that Confirm the Prinzmetal's Theory of Accelerated Conduction

Quintiliano H. de Mesquita; Claudio A. S. Baptista

Clinicas de Cardiologia
São Paulo – Brasil

Abstract

This study present cases of nodal AV Rhythm and NAV paroxysmal tachycardia – with QRS -> P inverted and retrograde pattern – developing WPW syndrome with the Delta wave characterized by aberrant QRS complex with thicken R or S initial deflection in D1 and aVL x D3 and aVF, according the A or B types. In cases of NAV tachycardia with A and B types was recorded progressive block in NAV-A conduction with atrial failures in 3:2 and 4:3 types, but with a R-R regular cadence indicative of supraventricular origin. The WPW syndrome mechanism with AV nodal origin confirm the theory by Prinzmetal et al: dual pathway of AV conduction functionally dissociated in the specific muscular system – AV node, bundle of His and right and left branches - respectivelly in B and A types of WPW syndrome, through the conduction of stimulus by accelerated AV pathway responsible for the ventricular pre-excitation (Delta wave) and completed by the ventricular activation from the same stimulus by normal and slow AV pathway.

Top

Introduction: In 1952 Prinzmetal et al (1, 2) in experimental studies about the Wolff-Parkinson-White syndrome preconized one accelerated pathway conduction in the specific muscular system, as responsible for the ventricular pre-excitation (Delta wave), completed by the activation of the remainder ventricular mass, through the same stimulus conducted slower by the normal AV pathway conduction.

In 1955 we admitted the theory from Prinzmetal et al in cases of coronary sinus rhythm with WPW patterns (3) and in 1961 new cases came to reinforce such findings when we also presented curious recordings of extrasystoles and nodal AV escapes with normal QRS complexes in cases of sinusal rhythm with WPW syndrome (4), justified by the use of only one AV pathway.

Consequently, these observations leaded us to publish 2 cases of paroxysmal AV nodal tachycardia (PAVNT) from the present series, without elements to consider the suspected aberrancy of QRS complex as WPW syndrome and with special highlight to the progressive, retrograde and rare blocking in AVN-A conduction (5).

Meanwhile, the 2 cases of AV nodal rhythm (AVNR) with aberrant QRS observed thereafter, supplied us the

necessary elements to the diagnostical conviction about the WPW syndrome developed by AV nodal stimulus (QRS -> P(-), according the ruling of Prinzmetal et al theory (1), and gave consistency to our present paper.

Objectives: To present the WPW syndrome with AV nodal origin (type 3) and to demonstrate its mechanism according the theory of Prinzmetal et al.

Top

Material and Methods: In 1985 (6) we described the AV nodal rhythm (AVNR) as originary of only one site recognized as follows: type 1 characterized by retrograde and negative P wave with short PR interval (P(-) -> QRS) and considered as the AV nodal pattern; recognizing also the type 2 (P + QRS) and the type 3 (QRS-> P(-) as variations of type 1 in consequence of disturbance of AV nodal-Atrials conduction (AVN-A), as well the rarely noted conduction in the direction of AVN-Ventricles represented by elongated PR interval or by blocked P wave recorded like extrasystolic phenomenon.

The ventricular pre-excitation syndrome (WPW) in cases of AVNR and PAVNT types presents the following morphological sequence: Delta wave including and thickening the initial deflection of wide and aberrant QRS complex followed by inverted and retrograde P wave.

According Prinzmetal et al (1) diagrams are presented at this paper representing the types A and B of WPW syndrome originated from the AV node (Figs. 4 and 6) highlighting the 2 pathways in the own specific muscular system – accelerated and slow pathway in AV node, His bundle and branches.

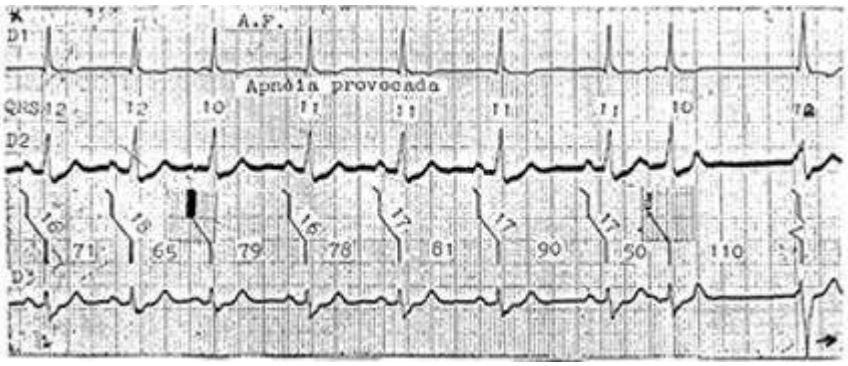
The inverted and retrograde P wave, posterior to the QRS complex, serve only to characterize the type 3 of AVNR and PAVNT, independently of the phenomena responsible for the WPW syndrome.

Top

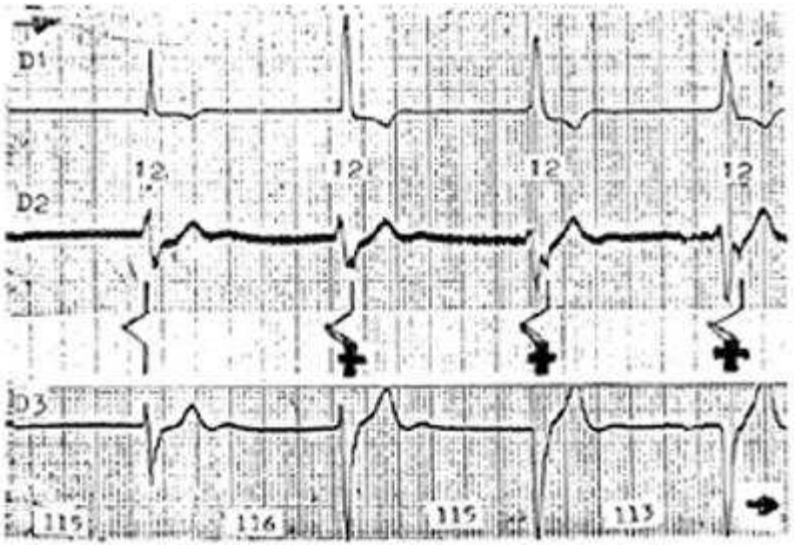
Results: The case 1 shows particular electrocardiographical aspects starting from the sinusal rhythm which depressed and by interference of atrial extrasystoles give rise to the installation of AVNR type 3 with WPW syndrome type B, recording the ventricular extrasystole without to alter the AVNR + WPW, followed by the return to the reactivated sinusal rhythm as spontaneous transition and with the same ECG initial morphological characteristics: disturbance of intraventricular conduction and of ventricular repolarization without branch block pattern (Fig. 1).

Case 1 – AF, 65 y., patient with chronic coronary heart disease and arterial hypertension with a discrete left ventricular hypertrophy.

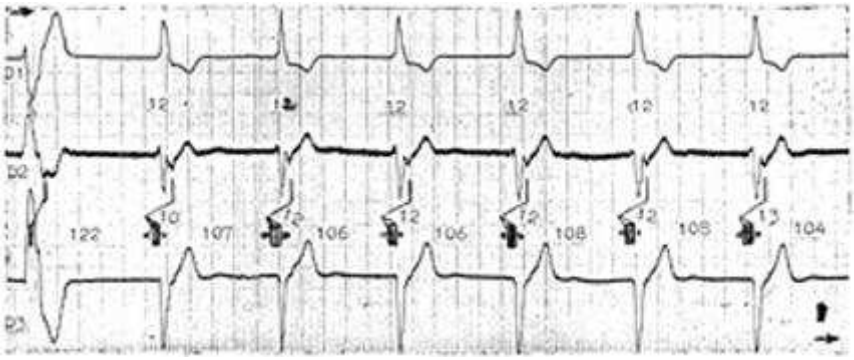
Figure 1 – 28/07/71: Continuous ECG (4 strips); simultaneous L1, L2 and L3: during provoked apnea. **Strip 1**: sinusal rhythm with isoelectric P wave in L1, QRS complex with 0,10 – 0,12 sec.characterized in L1 by "qR"with notches and enlargement of terminal branch R, S2 and S3 with notches and enlarged, Atrial extrasystoles; 3º and 8º cycles; AV nodal escape with sum of sinusal P wave (9º cycle)



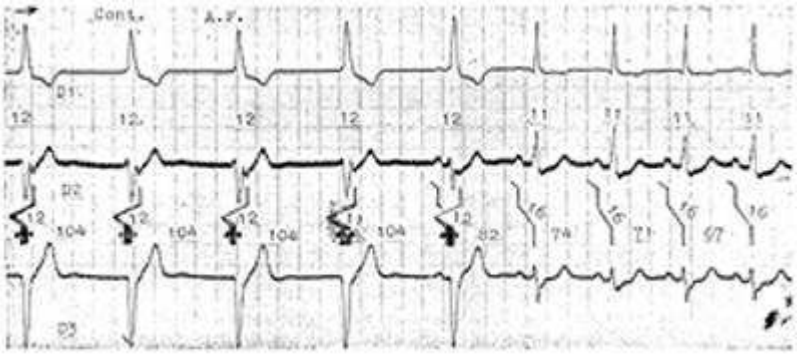
Strip 2: sinusal depression and installation of AVNR: 1º AV nodal cycle type 2 (present "qR": without WPW), 2º AV nodal cycle type 2 + WPW; 3º and 4º AV nodal cycles type 3 + WPW type B, with Delta wave including"q1 and r3" and responsible for the thickening of initial deflections R1 and S3



Strip 3: Ventricular extrasystole without repercussion on the AVNR type 3 + WPW type B, with Delta wave (+) in L1 and (-) in L3. QRS complex with WPW = 0,12 sec., RP interval = 0,12 sec.



Strip 4: Reversion of AVNR + WPW for active sinusal rhythm with QRS complex identical to strip 1



The case 2 characterized by the ECG with AVNR type 3 + WPW type B (Fig. 2) is represented by the graphic showed at Fig. 4; in confrontation with the normal ECG recorded 18 days after (Fig. 3).

Case 2 – OG, 26 y., normal heart

Figure 2 – 19/10/90: Conventional leads of limbs and multiple precordials. AVNR type 3 + WPW type B. Positive Delta wave in L1, aVL and from V2-V6 and negative in L2, L3, aVR, aVF, better characterized in V1. QRS complex = 0,12 sec.; RP interval = 0,13 sec.

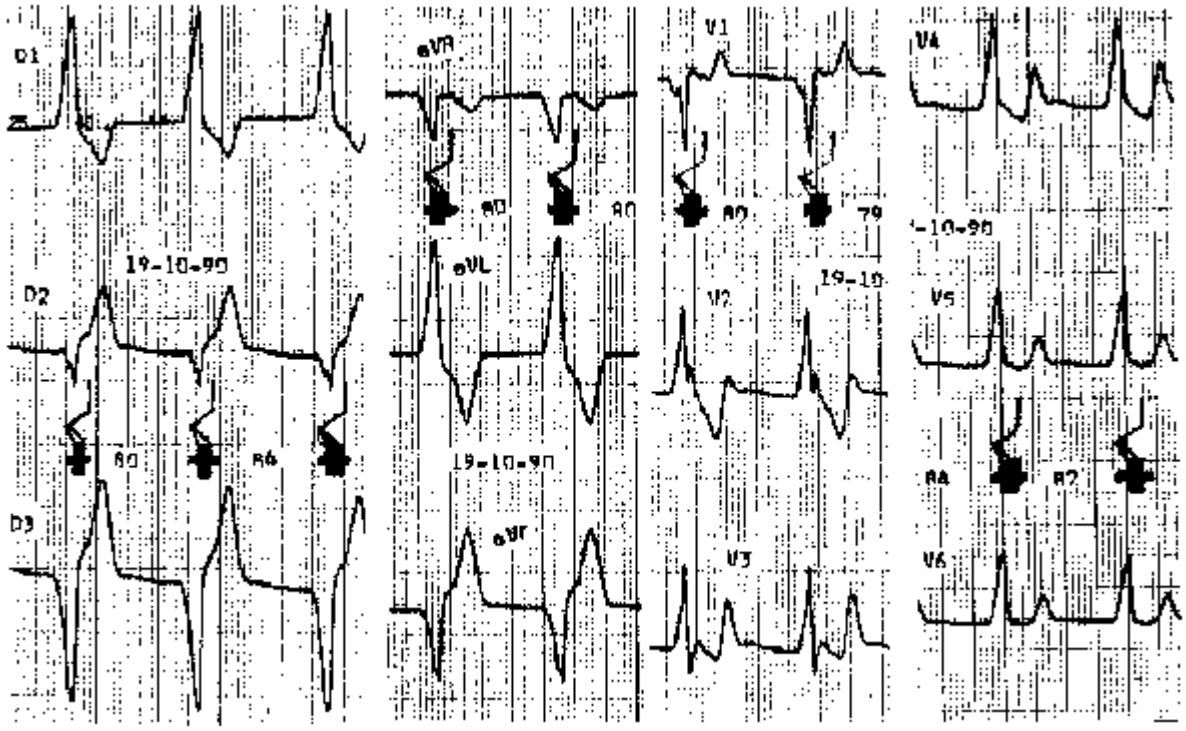


Figure 3 – 06/11/90: Same leads recorded earlier: sinusal rhythm. Normal ECG

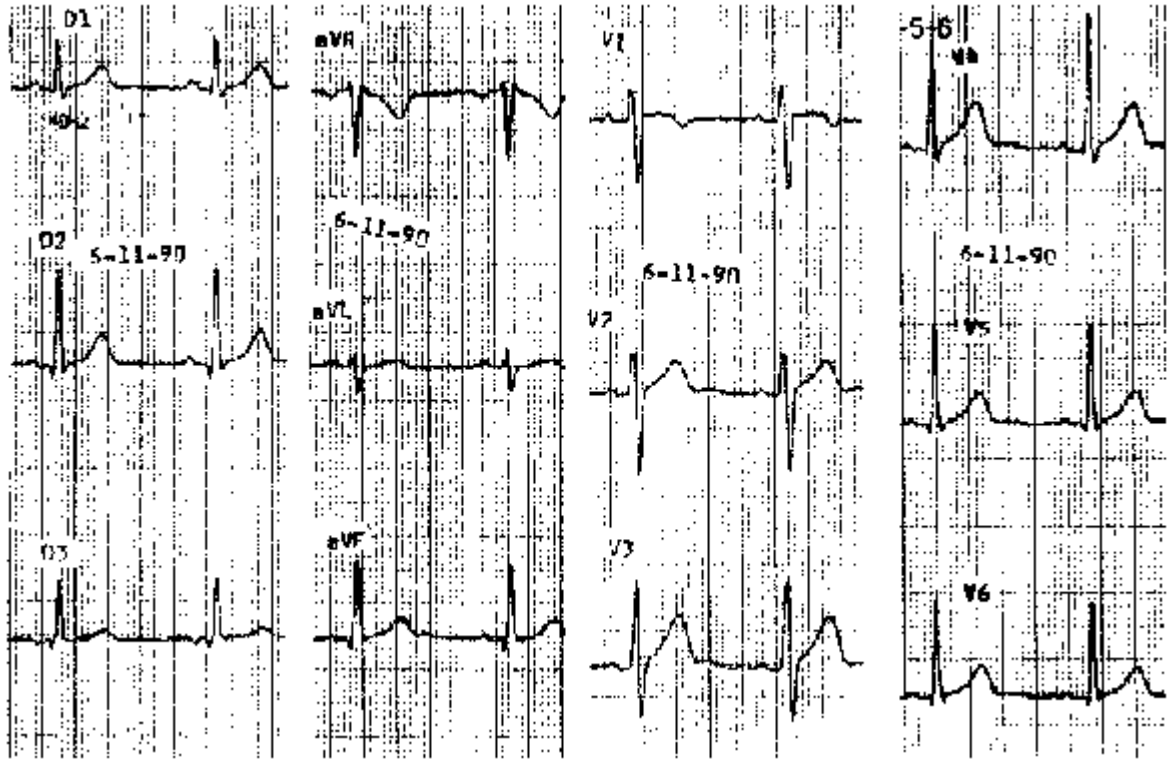
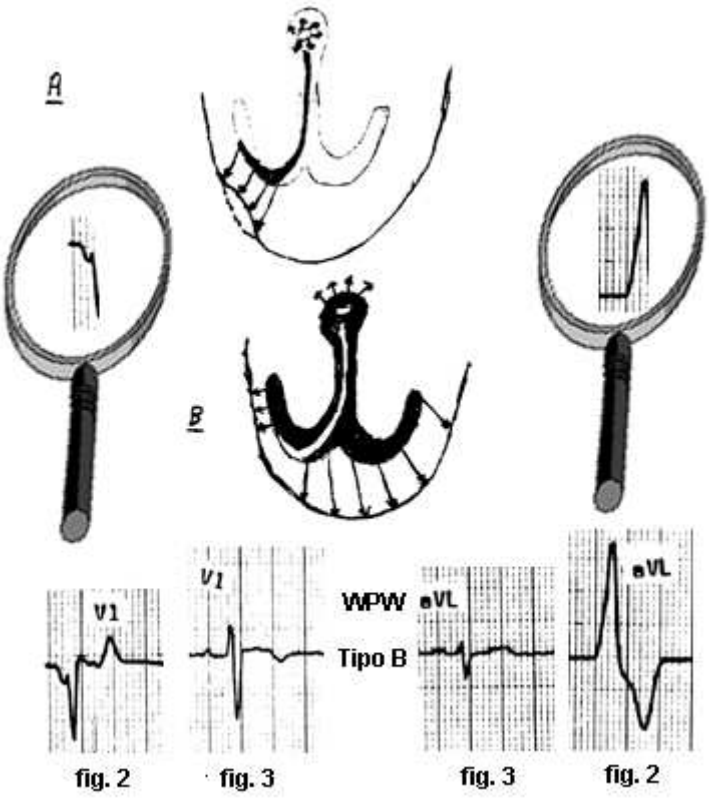


Figure 4 – Diagram explaining the WPW syndrome type B with stimulus originated from the AV node indicating the accelerated conduction mechanism in the ventricular pre-excitation syndrome based in longitudinal dissociation – AV node, His bundle and right branch – allowing the conduction of part of the impulse to pass through the fast pathway to activate the small area of right ventricle (A) – Delta wave – being completed the remainder ventricular activation by the normal slow AV pathway, asynchronically - aberrant QRS complex - (B) notched and slurring QS in V1 and slurring R in aVL



The case 3 of PAVNT type 3 + WPW syndrome type A (Fig. 5); represented by the graphic of Fig. 6 presents special electrocardiographical recordings of AVN-A block as Wenckebach phenomenon types 3:2 and 4:3.

Figure 5 – 22/12/50: Leads of limbs, precordials of CF type according Pardee technique (CF4R – EF2). PAVNT type 3 + WPW type A with negative Delta wave in L1, aVR and aVL and positive in L2, L3, aVF, CF4R to EF2 and aberrant QRS complex with 0,15 sec., enlarged L3: positive Delta wave (+) in L3 with aberrant QRS complex. Regularity in R-R distance (0,36 sec.) with frequence of 166 stimulus/min.. Wenckebach phenomenon in AVN-A conduction assuming blocks types 3:2 and 4:3 with recordings of fusions of P and T waves, showing atrial failures which provide the recording of T waves morphologically pures

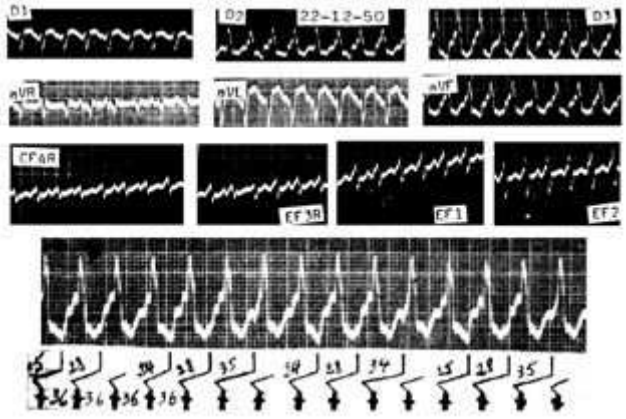
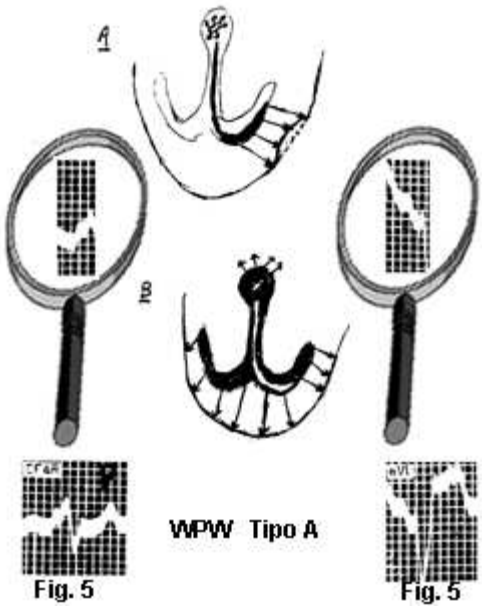
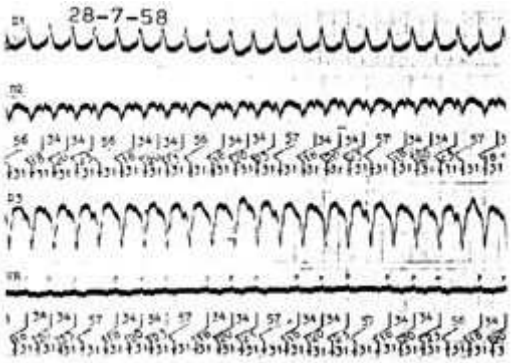


Figure 6 – Diagram explaining the WPW syndrome type A with stimulus originated in the AV node using the fast pathway – His bundle and left branch – developing the activation of small area of left ventricle (A) – Delta wave – being completed the remainder ventricular activation by the slow pathway of normal AV conduction, asynchronically, responsible for the aberrant QRS complex (B): Notched and slurring QS in aVL and R notched and slurring in CF4R



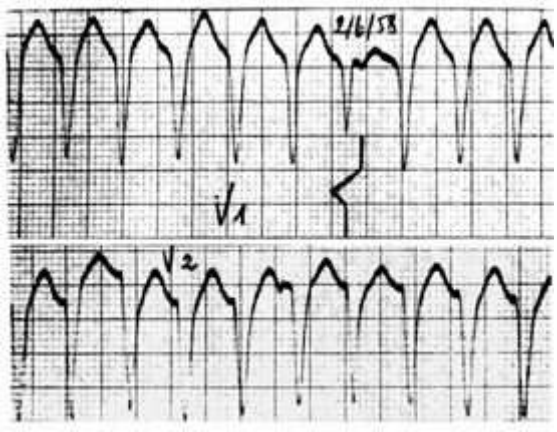
The case 4 of PAVNT type 3 + WPW type B, presents AVN-A block with Wenckebach phenomenon type 4:3 (Fig. 7).

Figure 7 – 28/07/58: simultaneous L1, L2, L3 and VR of Wilson. PAVNT type 3 + WPW type B with positive Delta wave in L1 and negative in the rest of leads; aberrant QRS complex with 0,12 sec. ; the PAVNT shows progressive periods of slow down in the RP interval, terminating in atrial failure (Wenckebach phenomenon): regular P-P distance with 0,34 sec. but with periodical atrial failures (P-P from 0,56 – 0,57 sec.) resulted from AVN-A 4:3 blockade; regular R-R distance of 0,31 sec. (193/min.) Recording the sum of P and T waves in complete cycles Type 3: QRS-P) and pure T waves in the cycles with atrial failures



Important recordings of enlarged V1 and V2 leads (Fig.8) to emphasize the WPW syndrome with negative Delta wave. In V1 the 7º QRS complex of normal duration happens from the syncronic ventricular activation followed by negative P wave; as observed the negative P wave in V2 after the 5º QRS complex.

Figure 8 – same case in 02/06/58: V1 and V2 enlarged; negative Delta wave characterizing the WPW syndrome type B. In V1, during other paroxysmal nodal AV tachycardia type 3 (RP = 0,12 sec.) with ocasional and spontaneous recording of normalization of QRS complex (0,10 sec.) with WPW pattern with negative Delta wave and followed by retrograde P wave (7º cycle), providing in this way the diagnostic of AV nodal origin (type 3) of paroxysmal tachycardia In V2, the 5º QRS complex is aberrant and followed by retrograde P wave with RP interval of 0,25 sec.



Top

Discussion: The cases of AVNR and PAVNT presenting WPW patterns types A and B are represented by the diagrams showed at Fig. 4 and 6 reproducing the mechanism preconized by Prinzmetal et al (1): longitudinal dissociation in the own specific muscular system with accelerated and slow pathways of AV conduction.

We are registering, with special interest, the Fig. 18 from the book of Prinzmetal et al (1) showing in the ECG the sinusal rhythm + WPW with AV nodal extrasystole type 3 + WPW, representing the only case published in bibliography according our review.

The concept about the necessary dual AV conduction to register the WPW syndrome is strengthened even more by the publication of Mahaim (7) through the case of agenesia of AV node, His bundle and the 2 branches, in presence of functioning aberrant atrioventricular bundle, responsible for the recording of long PR interval and enlarged QRS complex; characterizing the ventricular activation only by anomalous or aberrant pathway, without WPW pattern, representing in this way a significative and possible alternative to the anomalous bundle, in cases of total AV block.

In case 1 has been recorded (Fig. 1 strip 2: nodal AV escapes type 2 with and w/out WPW followed by the installation of AVNR type 3 with WPW type B); in the cases 3 and 4 the AVN-A blocks seems to make favourable to the WPW syndrome mechanism.

Conclusions: The cases of NAVR and PNAVT types 3 with NAV-A block, generating the WPW syndrome types A and B constitute exceptional and rare electrocardiographic aspects which are useful to confirm the theory of dual AV conduction – accelerated and slow – from Prinzmetal et al.

Top

References

1 - Prinzmetal, M; Kennamer, R; Corday, E; Osborne , JA; Fields, J e Allen, A: Accelerated Conduction. The Wolff-Parkinson-White syndrome and related conditions, New York, Grune & Stratton, Inc., 1952
2 - Prinzmetal, M; Corday, E; Brill, IC; Oblath, RW; Kruger, HE et al: The auricular arrhythmias, Springfield, Ill., Charles C. Thomas Pub, 1952
3 - Mesquita, QH de: Raros aspectos da sindrome de Wolff-Parkinson-White, Rev. Hosp. N. S. Aparecida, 1955; 8: 179-204
4 - Mesquita QH de: Manifestações do nó auriculo-ventricular e seio coronário com relação à sindrome de Wolff-Parkinson-White, Arq Bras Cardiol, 1961; 14: 363-372
5 - Mesquita, QH de: Taquicardia paroxística nodal AV inferior com QRS aberrante e bloqueio retrógrado progressivo nodal AV- auricular, Arq Bras Cardiol, 1961; 14: 213-220
6 - Mesquita, QH de : Sucessivas manifestações eletrocardiográficas dos 3 tipos de ritmo nodal AV, Rev Bras Med (Cardiologia), 1985; 4: 195-98
7 - Mahaim, I: Kent's fibers and the AV paraspecific conduction through the upper connection of the bundle of His-Tawara, Am Heart J, 1947; 33: 651

Questions, contributions and commentaries to the Authors: send an e-mail message (up to 15 lines, without attachments) to arritmias@listserv.rediris.es , written either in English, Spanish, or Portuguese.

Top

- **Dr. Armando Pacher**
President
Steering Committee

apacher@satlink.com
fac@fi.uner.edu.ar

- **Dr. Emilio Kuschnir**
President
Scientific Committee
polofriz@arnet.com.ar
conea@unc.edu.ar

Bioengineering
UNER
Update
Nov/14/1999
