WPW:
What’s still true?
What’s new?

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UTHSCSA and STVAHCS

I have no conflicts of interest related to this presentation.
Relevant References

• ACC/AHA/ESC Guidelines for the management of patients with supraventricular arrhythmias, 2003.
• Braunwald’s Heart Disease, 8th ed, 2008.
What’s Still True?

• How things developed
History of WPW - 1

• 1893: Kent described muscular AV connections, considered them to provide normal AV conduction
• 1914: Mines suggested that Bundle of Kent might mediate reentry
• 1930: Leon Wolff and Paul Dudley White in Boston and Sir John Parkinson in London published 11 cases with bizarre ventricular conduction and short PR intervals “Bundle Branch Block with Short PR Interval in Healthy Young People Prone to Paroxysmal Tachycardia” Am Heart J. 1930;5:685-704.
• 1944: Segers connected short PR interval, wide QRS and prolonged upstroke and arrhythmias into a syndrome

“Briefly the phenomena in question are concerned with the passage of the wave of contraction over the auriculo-ventricular groove, an explanation being required of the mode in which an auricular contraction is able on arriving at the groove to initiate a contraction of the ventricle”
Dr. Kent’s Data

- AV groove in monkey. “At the lower part of the figure on the right, a stellate mass of auricular muscle is seen, some of the fibers of which become continuous with some of the scattered branched muscle cells lying in the fibrous tissue.”

Dr. Kent’s Conclusion

• “The passage of the contraction over the cardiac tissue of the heart then appears to occur as a simple muscular wave, and the transmission of the contraction across the auriculo-ventricular groove appears to be of a similar nature.”

The American Heart Journal

Vol. V
AUGUST, 1930
No. 6

Original Communications

BUNDLE-BRANCH BLOCK WITH SHORT P-R INTERVAL
IN HEALTHY YOUNG PEOPLE PRONE TO
PAROXYSMAL TACHYCARDIA

LOUIS WOLFF, M.D., BOSTON, MASS., JOHN PARKINSON, M.D., LONDON,
ENG., AND PAUL D. WHITE, M.D., BOSTON, MASS.
The Three Original Authors
Paul Dudley White
1886-1973
WPW Exercise, Atropine

Fig. 1.—(Case I) Right bundle-branch block. The P-R interval is 0.1 second. The rate is 72. Time intervals for this and succeeding figures = 0.2 second. Horizontal lines cut off intervals of $10^{-4}$ volt.

Fig. 2.—(Case I) Immediately after exercise (running up and down four flights of stairs). Sino-auricular tachycardia, rate 140 to 120. The ventricular complexes are normal, the P-waves are better marked, and the P-R interval is 0.16 second.

Fig. 3.—(Case I) One hour after the subcutaneous injection of $1/20$ grain of atropine sulphate. The rate is 140, the ventricular complexes are normal, and the P-R interval is 0.15 to 0.16 second.
WPW and Exercise

Fig. 4.—(Case II) Intraventricular block. The P-R interval is 0.1 second. Rate 98.

Fig. 5.—(Case II) Immediately after exercise. The ventricular complexes are normal except for deformity of the S wave and S-T interval by artefact (high resistance, resulting in over-shooting). The T-wave is upright. The P-R interval is 0.15 second. Rate 90.
Original Article, Case III, Intermittent Pre-excitation
Original Article,
Case IV,
Intermittent

Fig. 9.—(Case IV) Spontaneous reversion from bundle-branch block curves to normal ones. The form of the P-wave remains unaltered, but the P-R interval changes from 0.09 second to 0.15 second.

Fig. 11.—(Case IV) Bundle-branch block. The P-R interval is less than 0.1 second. The P-waves are identical in Figs. 10 and 11; note the peculiar notching of the P-waves.

Fig. 10.—(Case IV) Normal physiological complexes. P-R interval 0.15 second. In Lead II there is a transition to the abnormal form and short P-R interval.

Fig. 12.—(Case IV) Simultaneous electrocardiogram and jugular and radial tracings. Bundle-branch block curves are present. The a. c. v. h. sequence is normal.
History of WPW - 2

- 1940 Richard F Ohnell (*Cardiologia* 1940) described a patient with WPW and SCD
Fibrous Skeleton of the Heart

Posterior view
The mitral annulus is generally thicker, more robust and fibrous than the tricuspid annulus.

Ventricular Pre-excitation

- 1/500 individuals
- Residual tissue after segmentation of the embryonic cardiac tube into atrial and ventricular chambers
- Fibers usually resemble ordinary myocardium*
- The fiber course from atrium to ventricle may be oblique to the long axis of the ventricle

WPW and Structural Disease

- Most have structurally normal hearts
- Some have abnormalities
  - Ebstein’s anomaly
  - HCM
  - MV prolapse
  - Genetic abnormalities PRKAG2 mutation (cardiac structure)
  - Mutation in BMP gene (neurologic)
Pathway Locations

60% left free wall

25% septal

15% right free wall
ECG Diagnosis

- Short PR interval
- Delta wave (slurred upstroke)
- Long QRS
- ST-T abnormality generally discordant from the delta wave and main QRS
- More subtle in left free wall pathways (the most common)
- Tachyarrhythmia is required for WPW syndrome
ECG Localization of Pathway

- There are many classification schemes by ECG
- All require manifest pre-excitation and up to 50% of pathways conduct only retrograde ("concealed")
- None is perfect
- I leave the details to the electrophysiologist
Localizing the Accessory Pathway - 1
(To be sure, must do EP study)

- Left free wall: negative delta wave in I, aVL, or V6 and “pseudo-RBBB” with Rs in V1
- Right anteroseptal (early ventricular activation near His bundle): positive delta wave in 2, 3, aVF, and low R/S in V1-V3 and late R wave transition
- Posteroseptal: negative or isoelectric delta waves in 2, 3, aVF and rapid R wave transition V1-V2

Localizing the Accessory Pathway - 2
(To be sure, must do EP study)

- Right free wall: positive delta wave in I and pseudo-LBBB
- Generally loss of a positive delta wave from leads 3 to aVF to 2 as the pathway location moves from anterior septal to posterior septal site around either AV ring
- For right-sided pathways, a positive delta wave occurs sequentially in V1 to V4 as the pathway location moves from anterior to posterior around the TV ring

Left posterior or paraseptal
Right posteroseptal – sudden transition from V1-V2, negative in II, III, and aVF
Intermittent preexcitation implies long refractory period of AP, so low risk of AF-RVR-VF

Right posterior paraseptal
Left lateral
Left lateral, patient with orthodromic AVRT
No preexcitation, P following QRS onset by 0.13 sec,
P upright in inferior leads
WPW

From Surawicz; Chou, 2001, p. 472
Orthodromic AVRT

- Most common tachycardia in WPW

Marriott, 9th ed, p. 107
Orthodromic AVRT

• If P wave is visible
  – Inverted in I – left lateral accessory pathway
  – Unfortunately, frequently impossible to discern
Left lateral or anterolateral
Left lateral or anterolateral with preexcited tachycardia (AF)
Atrial fib with long RR is low risk, high risk if RR<250 ms
NSR and WPW Pattern
WPW and Atrial Fibrillation
FIG. 10-45. Direction of slant of bypass tracts. The direction of slant between atrial and ventricular bypass tracts. From NASPE/ACC electrophysiology board review course, with permission.
Ventricular Pre-excitation

• Conduction is usually rate-independent like ventricular muscle
• Rate-dependent (decremental conduction) conduction to some degree has been found in about 7-8% (“PJRT”)
  – Antegrade decremental conduction more in right side
  – Retrograde decremental conduction anywhere

PJRT: Permanent Form of Junctional Reciprocating Tachycardia (Coumel)

- Incessant or nearly so, esp. seen in young
- Almost all cases due to retrograde conduction over accessory pathway, so better term is PAVRT, and accessory pathway has decremental retrograde conduction
- P waves are usually broad, inverted in 2, 3, and aVF
- RP longer than PR ("Long RP tachycardia")

The circuit involves 2 pathways with slow conduction, so giving a large "excitable gap"

PJRT: Permanent Form of Junctional Reciprocating Tachycardia

- **Initiation** of arrhythmia is with sinus beat, not PAC
- **Rate** of arrhythmia is sensitive to autonomic tone and physical activity with modulation of both RP and PR intervals
- **Transient termination** of arrhythmia through block in retrograde limb (no P wave)
- Retrograde limb sensitive to β-blockade, vagal maneuvers and calcium blockade, but arrhythmia is often refractory to medication

History of WPW Ablation

- 1967 – temporary ablation of pathway at surgery using procaine injection
- 1968 – ablation of pathway at surgery (Duke: Will Sealy, John Boineau, Galen Wagner, Andrew Wallace)
- 1984 – ablation of pathway with 200J DC shock
- 1989 – ablation of pathway with radiofrequency current
- 2001 – ablation of pathway with cryotherapy
Assessment of Risk in WPW

- Risk of SCD is higher in some subgroups
  - RR interval in AFib <250 msec
  - History of symptomatic tachycardia
  - Multiple accessory pathways
  - Ebstein’s anomaly
  - Familial WPW (rare)

- Risk of SCD is lower in some subgroups
  - Intermittent absence of delta wave
  - Asymptomatic ECG abnormality in pt >40 yo
Therapy for WPW Pattern on ECG (not syndrome)

• Asymptomatic patients with incidental preexcitation – no further eval or mgmt UNLESS … poss high-risk occupation such as bus driver, pilot, scuba diver, police, military, competitive athletics
Acute therapy for WPW

- Acute therapy for stable orthodromic AVRT: IV adenosine is highly effective (caution, it may result in atrial fibrillation in up to 12%) - - if asthmatic, IV calcium blocker
- Acute therapy for stable “preexcited tachycardia” (incl. antidromic AVRT, AT, Aflutter, AFib, AVNRT): IV procainamide or ibutilide
- Acute therapy for unstable tachycardia: DC cardioversion
Chronic therapy for WPW

- Infrequent well-tolerated episodes possible to use “pill-in-the-pocket” approach with AV nodal blocking agent
- Recurrent episodes, medical therapy with beta-blocker or calcium blocker VS ablation
- If persistent episodes, may add propafenone, flecainide, sotalol, or amiodarone to block accessory pathway
Catheter Ablation for WPW

- Radiofrequency for most, cryoablation for tracts near the conduction system or near coronary arteries (in coronary sinus)
- Cryoablation allows for “ice mapping”, technique of temporary freezing to evaluate result – if result is desirable, can perform permanent freeze
Lerman BB et al. NEJM. 2003;349:1787.
Unusual Pathway Locations

Catheter Ablation for WPW

• Success rate 98%
• Repeat procedure rate 2.2%
• Serious complication rate 0.6%
  – Tamponade, AV block, coronary artery injury, retroperitoneal bleed, stroke
• Mortality 0.02%
• (Overall annual SCD risk in WPW is 0.05-0.5%)
Indications for Catheter Ablation for WPW

- WPW syndrome with symptomatic arrhythmias, well tolerated (I)
- WPW syndrome with AF and rapid-conduction or poorly tolerated AVRT (I)
- AVRT, poorly tolerated, no pre-excitation (I)
- Single or infrequent AVRT episodes, no pre-excitation (IIa)
- Asymptomatic pre-excitation (IIa)
Future in WPW

• Progress in genetic associations and pathogenesis/embryogenesis
• Incremental improvements in ablation technology
  – High intensity focused ultrasound
Pathway Locations

Caveat: different authors use different orientations of the mitral and tricuspid orifices in their illustrations

Surawicz, Chou, 2001, p. 478
Braunwald, 2005, p. 740

Figure 20–19. The heart as viewed in the left anterior oblique projection. Nomenclature used to describe accessory pathway locations. RA = right anterior; RAL = right anterolateral; RL = right lateral; RPL = right posterolateral; RP = right posterior; PSTA = posteroseptal tricuspid annulus; CSOs = coronary sinus ostium; MSTA = mid-septal tricuspid annulus; AS = anteroseptal; RAPS = right anterior paraseptal; MCV = middle cardiac vein (coronary vein); CS = coronary sinus venous anomaly (coronary sinus diverticulum); PSMA = posteroseptal mitral annulus; LP = left posterior; LPL = left posterolateral; LL = left lateral; LAL = left anterolateral; HB = His bundle. (From Arruda MS, McClelland JH, Wang X, et al: Development and validation of an ECG algorithm for identifying accessory pathway ablation site in Wolff-Parkinson-White syndrome. J Cardiovasc Electrophysiol 9:2, 1998, by permission.)
Ablation of Oblique Pathway

Locations of Accessory Pathways
(Finer subdivisions are also used)

- Right anteroseptal (less common)
- Right free wall (third most common, 10-20%)
- Posteroseptal (second most common, 20-30%)
- Mid-septal (between His bundle and coronary sinus, less common)
- **Left free wall** (most common, 50-60%)
- Multiple in 5-20% of patients
  - Particularly posteroseptal and right free wall: consider Ebstein’s
  - More in patients resuscitated from VF

Names of Fibers

- **Kent**: AV connection
- **James**: atrium to distal or compact AVN
- **Brechenmacher**: atrium to His bundle
- **Mahaim**: His to ventricle (commonly used for atriofascicular fiber, original description was nodofascicular connection which is much less common than atriofascicular, e.g., atrium to right bundle branch along the lateral tricuspid annulus only capable of anterograde connection)

Types of Fibers

Hurst, 11th ed. P. 856.

Site of prior successful ablation of AVNRT

Brechenmacher

James

Mahaim

Kent

FIGURE 30-1  Structure of the AV node. A. Heart specimen from patient with AVNRT. Koch's triangle is formed by tendon of Todaro, coronary sinus (CS), ostium, and septal attachment of tricuspid valve (TV). Arrow represents site of successful ablation. IAS = interatrial septum, RV = right ventricle, FO = fossa ovalis, RAA = right atrial appendage. (From Olgin et al.16 With permission) B. Schematic drawing depicting the three zones of the AV node and various types of perinodal and atrioventricular bypass tracts. (From McManus BM, Harji S, Wood SM. Morphologic features of normal and abnormal conduction systems. In: Singer I, ed. Interventional Electrophysiology, 2d ed. New York: Lippincott Williams & Wilkins; 2001:23. With permission.)
Types of Ventricular Pre-Excitation

- Atrioventricular pathway (Kent bundle) – most common
- Mahaim fibers
  - Atriofascicular (Brechenmacher tract is to His bundle), antegrade only and decremental so reciprocating tachycardia is LBBB
  - *Nodoventricular (and nodofascicular) – clinical significance is controversial, reciprocating tachycardia does not require atrium (so can dissociate)
  - Fasciculoventricular - has not been demonstrated to cause
Types of Ventricular Pre-Excitation

A. Atrioventricular pathway (Kent bundle) – most common
B. Atriohisian is very uncommon, might give LGL, unproved (but atriofascicular does exist and gives preexcitation)
C. Nodovoventricular, original concept
D. Fasciculoventricular, not thought to be important in genesis of arrhythmia
E. Current concept of nodovoventricular – accessory pathway with AV nodal properties

Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005
Localization of Bypass Tract

FIG. 10-42. Schema of bypass tract locations used for ECG analysis. On the left the heart is opened at the midatrial level, and on the right the atria have been removed. The regions we find useful for ECG classification of bypass tracts are shown on the left. Region 1 is left lateral, region 2 is left posterior free wall, region 3 is posterior septal, region 4 is right free wall, and region 5 is anterior septal. The area between 3 and 5 along the tricuspid valve incorporates what are now referred to as mid-septal pathways. See text for discussion.
Preexcitation Syndromes


Table 1. Spectrum of Preexcitation Syndromes

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>No. Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accessory atrioventricular connections (WPW)</td>
<td>163</td>
</tr>
<tr>
<td>With associated EAVN*</td>
<td>20</td>
</tr>
<tr>
<td>AP conducts only retrograde</td>
<td>12</td>
</tr>
<tr>
<td>EAVN (LGL or variant) alone</td>
<td>11</td>
</tr>
<tr>
<td>Nodoventricular fibers</td>
<td>2</td>
</tr>
<tr>
<td>Fasciculovenicular fibers</td>
<td>6</td>
</tr>
<tr>
<td>EAVN plus fasciculovenicular fiber (mimicking WPW)</td>
<td>4</td>
</tr>
</tbody>
</table>

* EAVN, enhanced atrioventricular node conduction.

Table 2. Proposed Terminology for Anatomic Substrates of the Preexcitation Syndromes

<table>
<thead>
<tr>
<th>Proposed Terminology</th>
<th>Previous Terminology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accessory AV connection</td>
<td>Kent bundle (in septum also called Paladino tract)</td>
</tr>
<tr>
<td>Atriofascicular bypass tract</td>
<td>Atrio-Hisian fiber</td>
</tr>
<tr>
<td>Intranodal bypass tract*</td>
<td>James fiber</td>
</tr>
<tr>
<td>Nodoventricular connection</td>
<td>Mahaim fiber</td>
</tr>
<tr>
<td>Fasciculovenicular connection</td>
<td>Mahaim fiber</td>
</tr>
</tbody>
</table>

* Enhanced AV conduction through the AV node may be equally well explained by an AV nodal malformation or functional states of conduction unaccompanied by abnormal anatomic substrates.
Localization of Bypass Tract

Localization of Pathway

Precordial Lead R-Wave Transition*

Dominant R wave or transition at V1

Between V1-2 or at V2

Between V2-3 or at V3

Between V3-4

At or after V4

Left-sided

no

R in lead I >S by >1mV

yes

Septal

no

Δ wave in II<1mV

yes

Right-sided

*If the R/S ratio in any lead is nearly 1, the transition is at that lead. If R/S is <1 in one lead and >1 in the following lead, then the transition is between those leads.

Zipes and Jalife, 3rd ed, 2000. Ch. 95, p. 1081
Localization of Pathway

**Precordial Lead R-Wave Transition**

- **Dominant R wave or transition at V1**
  - No
    - Left-sided

- **Between V1-2 or at V2**
  - R in lead I ≥S by >1mV
    - Yes
      - Septal
        - Δ wave in II<1mV
          - Yes
            - Right-sided
              - +Δ wave aVF or R wave III ≥0mV
                - No
                  - Posterolat

- **Between V2-3 or at V3**
  - No
    - Midsept

- **Between V3-4**
  - Yes
    - Anterosept

- **At or after V4**
  - Yes
    - Anterolat

**For separation of septal pathways, the sum of the delta wave polarities in the inferior leads is considered, where a positive delta wave = 1, a negative delta wave = -1, and an isoelectric delta wave = 0.**

Zipes and Jalife, 3rd ed, 2000. Ch. 95, p. 1081
• Left free wall: negative delta wave in I, aVL, or V6 and “pseudo-RBBB” with Rs in V1
• Generally loss of a positive delta wave from leads 3 to aVF to 2 as the pathway location moves from anterior septal to posterior septal site around either AV ring
• Right anteroseptal (early ventricular activation near His bundle): positive delta wave in 2, 3, aVF, and low R/S in V1-V3 and late R wave transition
• Posteroseptal: negative or isoelectric delta waves in 2, 3, aVF and rapid R wave transition V1-V2
• Right free wall: positive delta wave in I and pseudo-LBBB
• For right-sided pathways, a positive delta wave occurs sequentially in V1 to V4 as the pathway location moves from anterior to posterior

Figure 2. Algorithm for the diagnosis of eight different sites of implantation of accessory pathways based on the polarity of the QRS complex during sinus rhythm on the surface ECG. An accurate diagnosis was possible in 128 of 140 (92%) patients with a single and anterogradely conducting accessory pathway. + = predominantly positive QRS complex; — = predominantly negative QRS complex; +/- = isodiphasic QRS complex; AS = anteroseptal; LL = left lateral; LP = left posterior; LPS = left posteroseptal; MS = mid-septal; PS = posteroseptal; RL = right lateral; RPS = right posteroseptal.
Figure 1. Schematic drawings showing a cross-section of the atroioventricular ring on the 30° left anterior oblique projection. The possible anatomical localizations of the accessory pathways are shown. 1 = left lateral accessory pathway; 2 = left posterior accessory pathway; 3 = left paraseptal accessory pathway; 4 = posteroseptal accessory pathway; 5 = right paraseptal accessory pathway; 6 = right lateral accessory pathway; 7 = anteroseptal accessory pathway; 8 = mid-septal accessory pathway; AV = aortic valve; MV = mitral valve; PV = pulmonic valve; TV = tricuspid valve.
FIGURE 2. Schematic representation of the accessory pathway location in the best left anterior oblique projection, illustrating the division of the 13 regions. The coronary sinus and great cardiac vein are depicted encircling the mitral annulus, with the ostium demarcated by the venous phase of the left coronary arteriography, routinely performed in this laboratory before electrophysiologic study. The numbers of accessory pathways in each location of the 182 patients are shown in parentheses. LAL = left anterolateral; LL = left lateral; LP = left posterior; LPL = left posterolateral; LPS = left posteroseptal; MS = midseptal; RA = right anterior; RAL = right anterolateral; RAS = right anteroseptal; RL = right lateral; RP = right posterior; RPL = right posterolateral; RPS = right posteroseptal.
FIGURE 3. Delta wave axis in the frontal plane of the initial 182 patients. The axis of the delta waves for each region showed much overlap, and was not very helpful for differentiation. Left lateral (LL)/left anterolateral (LAL) (range +60° to +120°); left posterior (LP)/left posterolateral (LPL) (range −60° to +30°); left posteroseptal (LPS) (range −60° to +30°); midseptal (MS) (range −30° to +30°); right anterolateral (RAL) (range +15° to +45°); right anteroseptal (RAS)/right anterior (RA) (range +30° to +60°); right lateral (RL) (range −30° to +30°); right posterior (RP)/right posterolateral (RPL) (range −60° to −15°); right posteroseptal (RPS) (range −75° to +15°).
TABLE II  Difference in Precordial R/S Ratio Between Group I and II Pathways

<table>
<thead>
<tr>
<th>Group I</th>
<th>Group II</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_1 ) R/S ( \geq 1 )</td>
<td>68</td>
<td>1</td>
<td>61.3</td>
</tr>
<tr>
<td>( V_2 ) R/S ( \geq 1 )</td>
<td>105</td>
<td>3</td>
<td>94.6</td>
</tr>
<tr>
<td>( V_3 ) R/S ( \geq 1 )</td>
<td>111</td>
<td>31</td>
<td>100</td>
</tr>
</tbody>
</table>
Left Free Wall Pathway

- Most common
- Negative delta waves in I and L and positive delta in inferior leads and all precordial leads (Josephson p. 356)
- Negative delta waves in I, aVL, or V6 and a “pseudo-right bundle branch block” QRS complex appearance with positive QRS complex (Rs wave) in V1 (Prystowsky in Zipes p. 873)
- Step 1: If the delta wave in lead I is (-) or (+-) or the R/S in lead V1 is >1, a left free-wall AP is present (Surawicz p. 479 after Arruda)
Wolff-Parkinson-White Syndrome (WPW)

- Short PR interval (<0.12 sec) in 75-90%
- Wide QRS complex (≥ 0.11 sec) in 65-75%
- Slurred initial forces of QRS - Delta wave
- Secondary ST segment and T wave abnormality (discordant to Delta wave)
- Frequent association of paroxysmal tachycardia, usually supraventricular
Wolff-Parkinson-White Syndrome (WPW) - 2

- Every beat is a fusion beat
  - Part of QRS from AV node and normal His-Purkinje system
  - Part of QRS from conduction through the accessory AV connection (“Bundle of Kent”) from atrial muscle to ventricular muscle
  - Variable conduction depends on how much of the ventricle is excited from the normal versus the
Wolff-Parkinson-White Syndrome (WPW) - 3

- Tachycardia is often due to electrical activity travelling in a circular pathway
- One Possible Pathway
  - AV node
  - Atrial muscle
  - Accessory pathway
  - Ventricular muscle
- “Circus movement”
- Atrioventricular reentrant tachycardia (AVRT)
Tachycardias in WPW Syndrome

• Accessory pathway integral to circuit
  – Orthodromic AVRT (most common)
    • With or without functional bundle branch block (ipsilateral, slows rate)
  – Pre-excited reciprocating tachycardias
  – Antidromic AV reentrant tachycardias
  – AVRT with multiple pathways

• Accessory pathway passive, not essential
  – AVNRT
  – AVRT with second bystander accessory pathway
  – Aflutter or Fibrillation
  – VT

Tachycardias in WPW Syndrome

- Accessory pathway integral to circuit
  - Orthodromic AVRT (most common)
    - With or without functional bundle branch block (ipsilateral, slows rate)
  - Pre-excited reciprocating tachycardias
  - Antidromic AV reentrant tachycardias
  - AVRT with multiple pathways
- Accessory pathway passive, not essential
  - AVNRT
  - AVRT with second bystander accessory pathway
  - Atrial flutter or fibrillation
  - VT

Orthodromic AVRT and BBB

Josephson ME. Clinical Cardiac Electrophysiology 2002; p. 370.
PJRT, or PAVRT

FIGURE 94.5  ■ Surface electrocardiogram of a patient with the permanent form of junctional reciprocating tachycardia.  A, The 12-lead electrocardiogram illustrates the essential features of the tachycardia with typical negative P waves in leads II, III, and aVF and an R-P interval longer than the P-R interval.  B, The tachycardia was transiently terminated by right carotid sinus massage.  Termination without a retrograde P wave indicated block occurred in the retrograde limb and illustrated the atrioventricular node–like behavior of the accessory pathway.  With acceleration of the sinus rate, there was spontaneous resumption of tachycardia.

• Left lateral or anterolateral pathway
Right anteroseptal accessory pathway, characteristic inferior axis, delta wave is negative in V1 and V2, upright in I, II, aVL, and aVF, isoelectric in III, negative in aVR

Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005
Right posteroseptal accessory pathway. negative delta in II, III, and aVF, upright in I and aVL localize the pathway to posteroseptal, and negative delta in V1 and rapid transition in V2 pinpoints to right posteroseptal; AFib

Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005
AF and WPW becoming VF

Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 836, 2005
Left lateral accessory pathway – positive delta in anterior precordial leads and in II, III, and aVF, positive or isoelectric in leads I and aVL, and isoelectric or negative in V5 and V6 is typical of a left lateral accessory pathway. Coronary sinus pacing was used to enhance preexcitation.

Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005
Right free wall accessory pathway – predominantly negative delta in V1 and axis more leftward
Right posteroseptal – sudden transition from V1-V2 and negative in II, III, and aVF
Left lateral
Read as WPW but questionable … short PR is because rhythm is not sinus
Right anterior paraseptal
Left posterior or posterolateral
Left anterolateral or lateral
Left anterolateral or lateral
Right anteroseptal or anterior paraseptal
Left posterior
Left lateral
No delta wave – merely enhanced AV node conduction (LGL)
Not very pre-excited, left lateral
Left posterior or posterolateral
References

• Chugh A and Morady F, Ch. 58, “Atrioventricular reentry and its variants” Cardiac Electrophysiology, From Cell to Bedside, 5th ed. 2009.
Original Article, Cases V and VI

Fig. 13.—(Case V) Intraventricular block. The P-waves are normal and upright in all leads. The P-R interval is well under 0.1 second.

Fig. 14.—(Case VI) Intraventricular block. The P-waves are normal and upright in all leads. The P-R interval is well under 0.1 second.
Fig. 15.—(Case VII) Left bundle-branch block. The P-waves are normal and upright in all leads. The P-R interval is well under 0.1 second.

Fig. 16.—(Case VIII) Right bundle-branch block. The P-R interval is well under 0.1 second.

Fig. 17.—(Case IX) Intraventricular block. The P-R interval is 0.1 second. Time intervals = 0.2 and 0.04 seconds.

Fig. 18.—(Case IX) Three years later. Normal physical curves. The P-R interval is 0.16 second. The P-waves are identical in Figs. 16 and 17.
Fig. 19.—(Case X) Intraventricular block. The P-R interval is 0.1 second.

Fig. 20.—(Case XI) Intraventricular block. The P-R interval is less than 0.1 second.
Note: In this paper bundle-branch block when mentioned is referred to according to the old nomenclature of right bundle-branch block for upright widened Q-R-S waves in Lead I and inverted widened Q-R-S waves in Lead III, and left bundle-branch block for inverted widened Q-R-S waves in Lead I, and upright widened Q-R-S waves in Lead III according to the newly revised nomenclature, which is probably correct, these designations would be changed, so that one should read "left bundle-branch block" for "right" and "right bundle-branch block" for "left" in this paper.

REFERENCES

Anatomical landmarks of the triangle of Koch. This triangle is delimited by the tendon of Todaro superiorly, which is the fibrous commissure of the flap guarding the openings of the inferior vena cava and coronary sinus, by the attachment of the septal leaflet of the tricuspid valve inferiorly, and by the mouth of the coronary sinus at the base.

Tendon of Todaro is absent in about 2/3 of hearts; it originates in the central fibrous body and passes through the atrial septum to continue with the eustachian valve.

The compact portion of the AVN becomes the penetrating portion of the His bundle at the point where it enters the central fibrous body.

- In 85-90% of human hearts the arterial supply to the AVN is from the RCA originating at the posterior intersection of the AV and interventricular grooves (crux).

The Bundle of His (penetrating portion of the AV bundle) continues from the central fibrous body through the annulus fibrosis and penetrates the membranous septum.

Anatomical landmarks of the triangle of Koch. This triangle is delimited by the tendon of Todaro superiorly, which is the fibrous commissure of the flap guarding the openings of the inferior vena cava and coronary sinus, by the attachment of the septal leaflet of the tricuspid valve inferiorly, and by the mouth of the coronary sinus at the base. Stippled area adjacent to the central fibrous body is the approximate site of the compact atrioventricular node.

Braunwald, 2005, p.656.
Sections through the atrioventricular (AV) junction show the position of the AV node (arrowhead) within the triangle of Koch (A) and the penetrating AV bundle of His (arrowheads) within the central fibrous body (B).