ABERRANT VENTRICULAR CONDUCTION TYPES AND CONCEALED CONDUCTION

Others denominations: Aberrancy, ventricular aberration.

Aberrant ventricular conduction definition: It is a term applied to alterations in QRS contour of supraventricular beats resulting from impulse transmition during periods of physiologic refractoriness and/or depressed conductivity. The supraventricular electrical impulse is conducted abnormally through the ventricular conducting system. This results in a wide QRS complex that may be confused with a ventricular ectopic beat.

RELATIVE FREQUENCY OF EXPERIMENTAL ABERRATION[1]

Aberrant ventricular conduction was induced in 44 subjects by introduction of atrial premature beats through a transvenous catheter-electrode. Multiple patterns of aberrant ventricular conduction were obtained in 32 patients and, in the whole group, 116 different configurations were recorded. Of these, 104 showed a classical pattern of mono- or biventricular conduction disturbance. Right Bundle Branch Block (RBBB) 24%, RBBB combined with Left Anterior Fascicular Block (LAFB) 18%, LAFB 15%, RBBB combined with Left Posterior Fascicular Block (LPFB) 10%, LPFB 9%, LBBB 5%, Incomplete LBBB (ILBBB) 6%, trivial changes of the QRS contour 6% and marked anterior displacement or Prominent Anterior Forces(LSFB) 4%.

Totals: RBBB: 52, LAFB: 33, LPFB: 19, LBBB: 14, trivial modifications of the QRS contour in 7 of them. In the other 5 instances, aberrant conduction manifested itself by a conspicuous anterior displacement of the QRS loop (Prominent Anterior Forces = PAF), with increased duration of anterior forces. The latter observation is worthy of notice, as it indicates that, in the differential diagnosis of the VCG pattern characterized by, conduction disturbances should be considered a possible etiological factor in addition to right ventricular hypertrophy, and true posterior wall myocardial infarction (or lateral MI in the new Bayes de Luna nomenclature concept).

RBBB aberrancy pattern is observed in 80 % to 85% of cases. In sick population LBBB aberrancy is near 33% of cases[2].

The mechanisms of aberrancy with changing cycle length are[3; 4]:

1) Premature arrival of the supraventricular impulse before full recovery of the right bundle branch (RBB);
2) Inadequate or unequal refractoriness of conducting tissue resulting in local delay or block of dromotropism;
3) Prolongation of Action Potential (AP) secondary to lengthiness of the preceding cycle duration;
4) Unsuccessful of restitutions of transmembrane electrolyte concentration during relaxation and dilatation of the ventricles;
5) Failure of the refractory period to shorten in response to acceleration of the heart rate(HR);
6) A reduced take-off potential secondary to diastole depolarization;
7) Concealed transseptal conduction with delay or block of bundle branch conduction and
8) Diffuse depression of Intraventricular conduction including that of specialized as well as contractile myocardial.

The three forms of ventricular aberration

Type A: It is the common form and due to fascicular refractoriness. It is caused by supraventricular (atrial or junctional) premature beat or accelerated of the sinus rhythm. The early impulse reach the RBB when still in refractory period and it has been unable to respond and conduct

Type B: It is due to anomalous supraventricular activation

Type C: It is due to paradoxical critical rate.

In cases of stable wide QRS-complex tachycardia the evaluation of the ECG without further information in prehospital emergency-medicine leads to unsatisfactory results. The correct diagnosis in wide QRS-complex tachycardia can be improved by using additional data but the diagnostic accuracy is still low.

Therefore, the differential diagnosis of stable wide QRS-complex tachycardia in preclinical emergency-medicine cannot be recommended. Until proven otherwise, any stable wide QRS-complex tachycardia should be managed as if it were VT[5].

I) ABERRANCY SECONDARY TO GOUAUX-ASHMAN PHENOMENON OR ASHMAN PHENOMENON[6]

Although most Premature Atrial Contractions (PACs) or Premature Junctional Contractions (PJCs) (premature supraventricular beats) are conducted to the ventricles normally (i.e., with a narrow QRS complex), this is not always the case. Instead, PACs or PJCs may sometimes occur so early in the cycle as to be "blocked" (i.e., non-conducted), because the conduction system is still in an absolute refractory period (ARP). Other times, premature beats may occur during the relative refractory period (RRP), in which case aberrant conduction (with a widened QRS) occurs. Practically speaking, aberrant conduction is most likely to take the form of some type of bundle branch block (BBB)/fascicular pattern most commonly RBBB. Attention to QRS morphology may help to distinguish between aberrancy and ventricular premature contractions(VPCs).

The refractory period in cardiac physiology is related to the ion currents which, in cardiac cells as in nerve cells, flow into and out of the cell. The flow of ions translates into a change in the voltage of the inside of the cell relative to the extracellular space. As in nerve cells, this characteristic change in voltage is referred to as an AP. Unlike nerve cells, the cardiac AP duration is closer to 100 ms (with variations depending on cell type, autonomic tone, etc.). After an AP initiates, the cardiac cell is unable to initiate another AP for some duration of time (which is slightly shorter than the "true" action potential duration). This period of time is referred to as the refractory period.

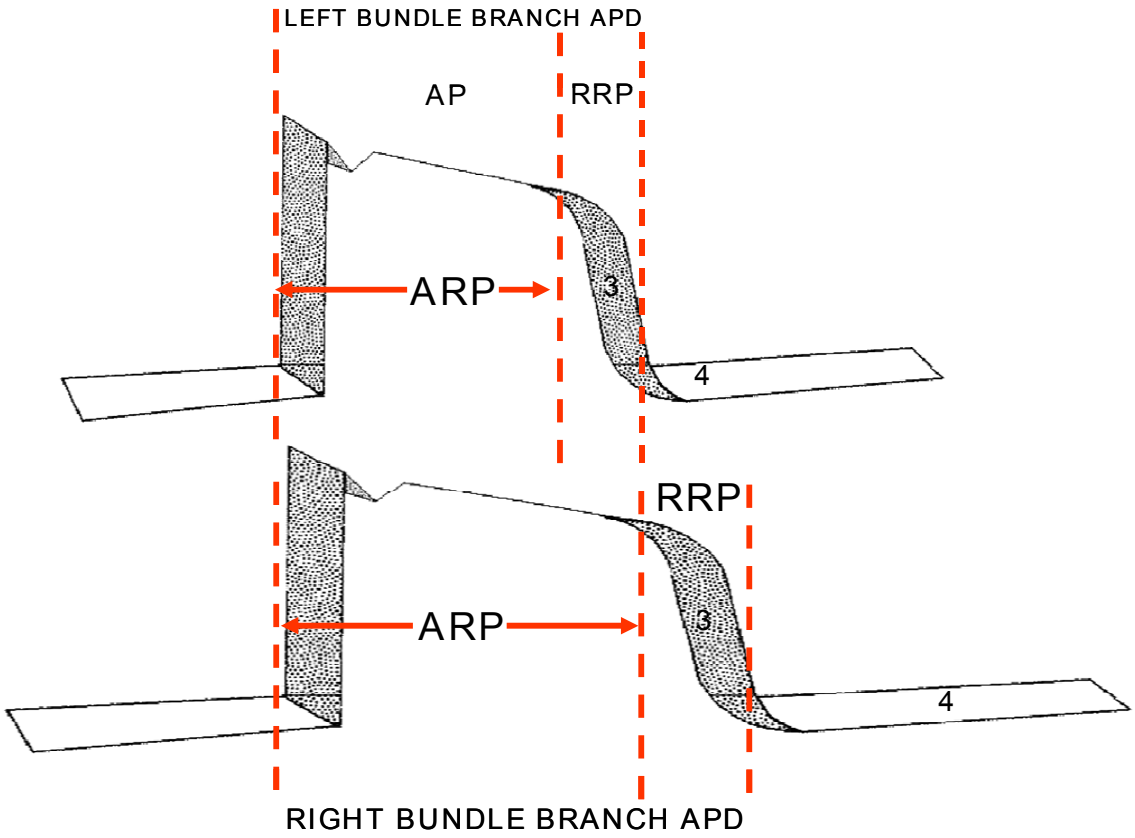
Classically, the cardiac refractory period is separated into an ARP and a RRP. During the ARP, a new AP cannot be elicited. During the RRP, a new AP can be elicited under the correct circumstances. During RRP a second AP can be evoked, but only if the stimulus strength is increased.

The aberrant conduction depends on the RRP of the conduction tissues. The refractory period depends on the heart rate (HR). Action potential duration (APd) (ie, refractory period) changes with the R-R interval of the preceding cycle; shorter duration of action potential (AP) is associated with a short R-R interval and prolonged duration of AP is associated with a long R-R interval. A longer cycle lengthens the ensuing refractory period, and, if a shorter cycle follows, the beat ending it is likely to be conducted with aberrancy.

Aberrant conduction results when a supraventricular impulse reaches the His-Purkinje system while one of its branches is still in the RRP or ARP. This results in slow or blocked conduction through this bundle branch and delayed depolarization through the ventricular muscles, causing a bundle-branch block configuration pattern on the surface ECG, in the absence of bundle-branch pathology. A RBBB pattern is more common than a LBBB pattern because of the longer refractory period of the RBB.

Figure 1

DIFFERENTIAL CHARACTERISTICS OF AP ON RBB AND LBB



RRP: Relative Refractory Period. **ARP:** Absolute Refractory Period.

APD: Action Potential Duration.

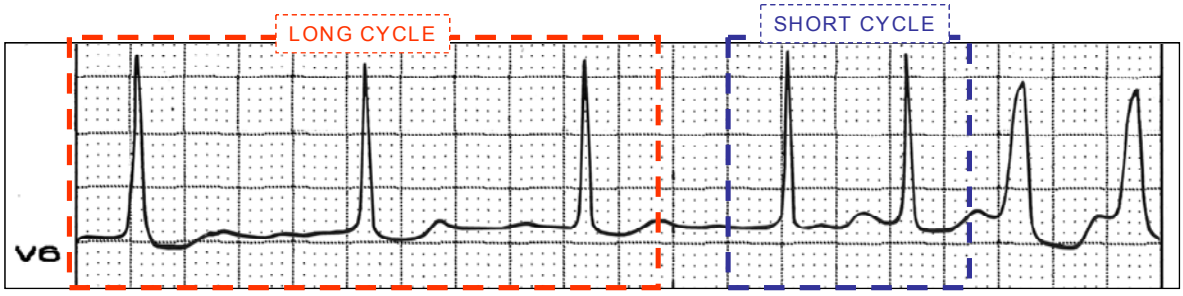
Commentaries: RBB has refractoriness periods (RRP and ARP) longer than the LBB.

Several studies have questioned the sensitivity and specificity of the long-short cycle sequence. Aberrant conduction with a short-long cycle sequence has also been documented.

Gouaux-Ashman phenomenon or Ashman phenomenon[6] is an intraventricular conduction abnormality restricted to the His-Purkinje system, caused by a change in the HR. This is dependent on the effects of rate on the electrophysiological properties of the heart and can be modulated by metabolic and electrolyte abnormalities and the effects of drugs. Conditions causing an altered duration of the refractory period of the bundle branch or the ventricular tissue cause Ashman phenomenon. These conditions are commonly observed in:

1) Atrial fibrillation(AF): Figure 2;
2) Atrial tachycardia;
3) Premature Atrial Contractions.

Figure 2



In this case aberration occurs when a short cycle follows a long one. Aberration follows a long-short sequence during AF. The last 2 complex are aberrant with LBBB pattern: Gouaux-Ashman phenomenon or Ashman phenomenon.

Ashman phenomenon is an aberrant ventricular conduction due to a change in QRS cycle length. In 1947, Gouaux and Ashman reported that in AF, when a relatively long cycle was followed by a relatively short cycle, the beat with a short cycle often has RBBB morphology. This causes diagnostic confusion with premature ventricular complexes (PVCs). If a sudden lengthening of the QRS cycle occurs, the subsequent impulse with a normal or shorter cycle length may be conducted with aberrancy.

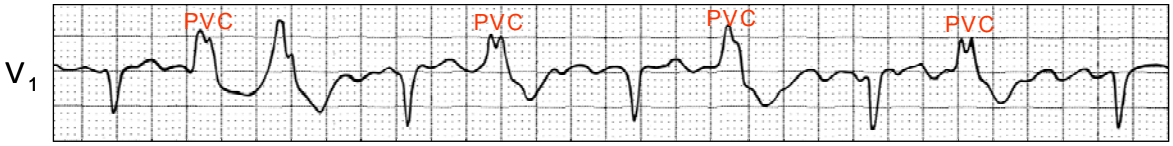
Studies emphasize the importance of HR in the genesis of ventricular arrhythmias during myocardial ischemia. In 20 dogs subjected to acute myocardial ischemia and crushing of the SA node, standard ECG leads were recorded, as well as His bundle and epicardial electrograms from the normal and ischemic areas. Abrupt pauses in regular atrial pacing did not cause arrhythmias prior to the onset of ischemia; however, during ischemia, atrial pacing with intermittent abrupt pauses resulted in the induction of ventricular arrhythmias beginning after the second conducted beat following each pause (VPCs, 20/20; VT, 19/20; and VF, 8/20). Onset of the arrhythmia was associated with increased delay in activation of ischemic epicardium and fractionation of the electrogram potential of the second conducted impulse. Typical Gouaux-Ashman phenomenon was an incidental observation. Unlike the Gouaux-Ashman phenomenon, which is restricted to the His-Purkinje system, the phenomenon the authors observed originated within ischemic myocardium. In vitro studies indicate that the underlying mechanism may be related to postrepolarization refractoriness induced by ischemia[7].

Transitory BBB at the onset of an SVT is noted in 14% of the population, is more frequent in patients with accessory pathway reentrant tachycardia, but is helpful for this diagnosis in only 12% of cases. A regular tachycardia with permanent left or right bundle branch morphology induced by atrial stimulation in a patient without heart disease and without BBB during atrial pacing is due to a VT even if this patient has also narrow complex tachycardias. This mechanism does not affect the excellent prognosis of this population[8].

Differential diagnosis

1) Atrial fibrillation and premature ventricular contractions

Figure 3



Atrial fibrillation with PVCs. The coupling intervals are fixed, the PVCs complex are very wide (190ms) and monophasic. In this case, there are not marked compensatory pause after PVCs.

Table 1

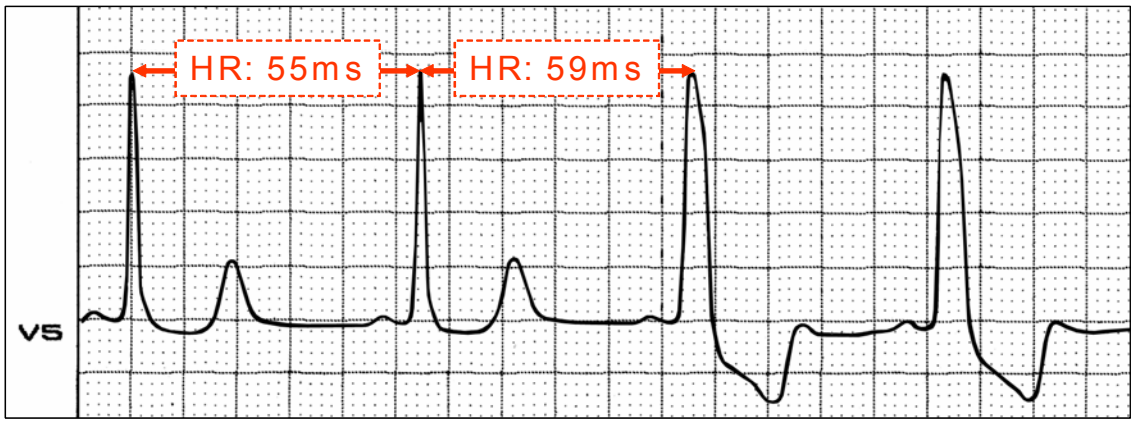
Electrocardiographic differentiations between AF with aberrant ventricular conduction and AF with premature ventricular contractions (PVCs)

	AF with aberrant ventricular conduction	**AF with PVCs**
QRS duration	Between 120 to150ms.	Very wide: 150 to 200ms.
QRS pattern	rR' pattern in V_1-V_2. Variable degrees of widening and varying of BBB.	Bizarre mono or diphasic complexes. Occasionally BBB pattern.
Coupling interval	A varying interval between the widening QRS complex and the previous complex.	Fixed between the broad QRS complex and previous narrow complexes.
Compensatory pause	Absence of "compensatory post extrasystolic pause"	Presence of a marked "compensatory post extrasystolic pause" caused by concealed AV conduction.

II) ACCELERATION-DEPENDENT ABERRANCY, TACHYCARDIA-DEPENDENT, IN PHASE 3 ABERRANCY, OR PHASE 3 ABERRATION

Resulting from the occurrence of impaired intraventricular conduction as the heart attains a specific critical rate. At a critical HRs, impaired ventricular conduction results in aberrancy. The appearance and disappearance often depends on very small changes in cycle length. Aberrancy often appears at relatively slow rates, frequently below 75 beats/min.

Figure 4

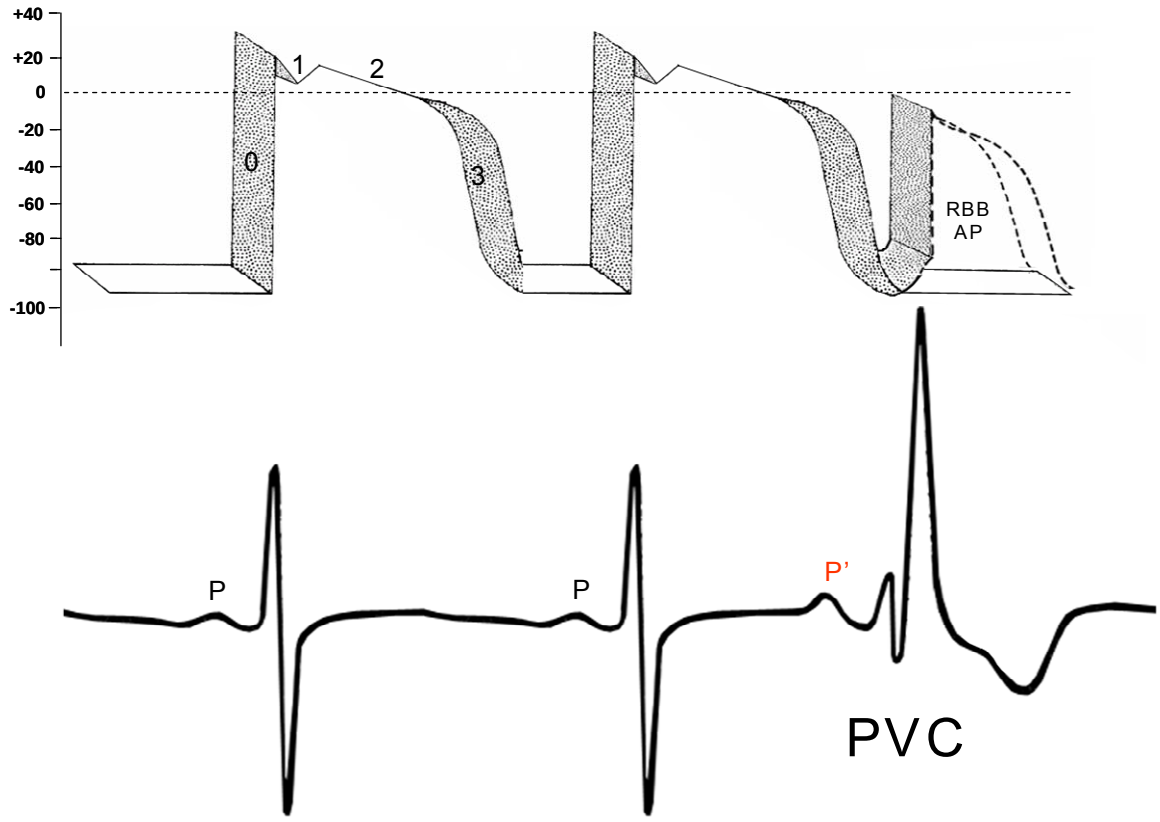


Aberration appear with minimal or imperceptible cycle length shortening

An very early premature beat arrives to the branches during the RRP(phase 3 period) of the RBB and produces an AP that does not propagate. The aberrations occurs because the stimulus reaches the RBB during phase 3 when the membrane potential is -65 mV. The impulse is successful conducted down the LBB to produce RBBB pattern. This is the common form of aberration.

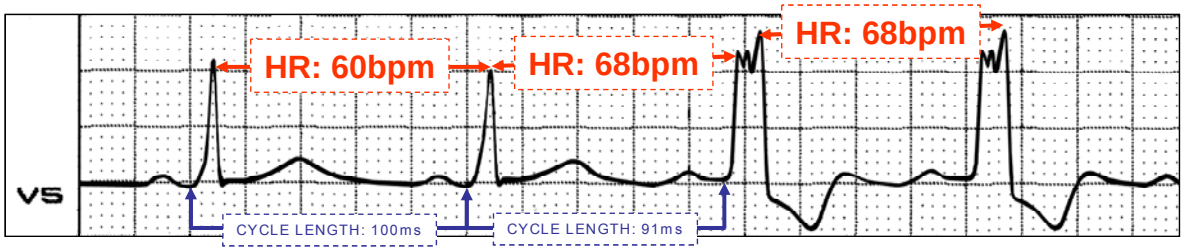
Phase 3 aberration may also occur pathologically if the RRP is abnormally prolonged and the involved fascicle is stimulated at relatively fast rate.

Figure 5



APs corresponding ECG representation of Phase 3 aberration. Early atrial PVC arrives during the RRP of the RBB and produces AP with RBBB pattern.

Figure 6



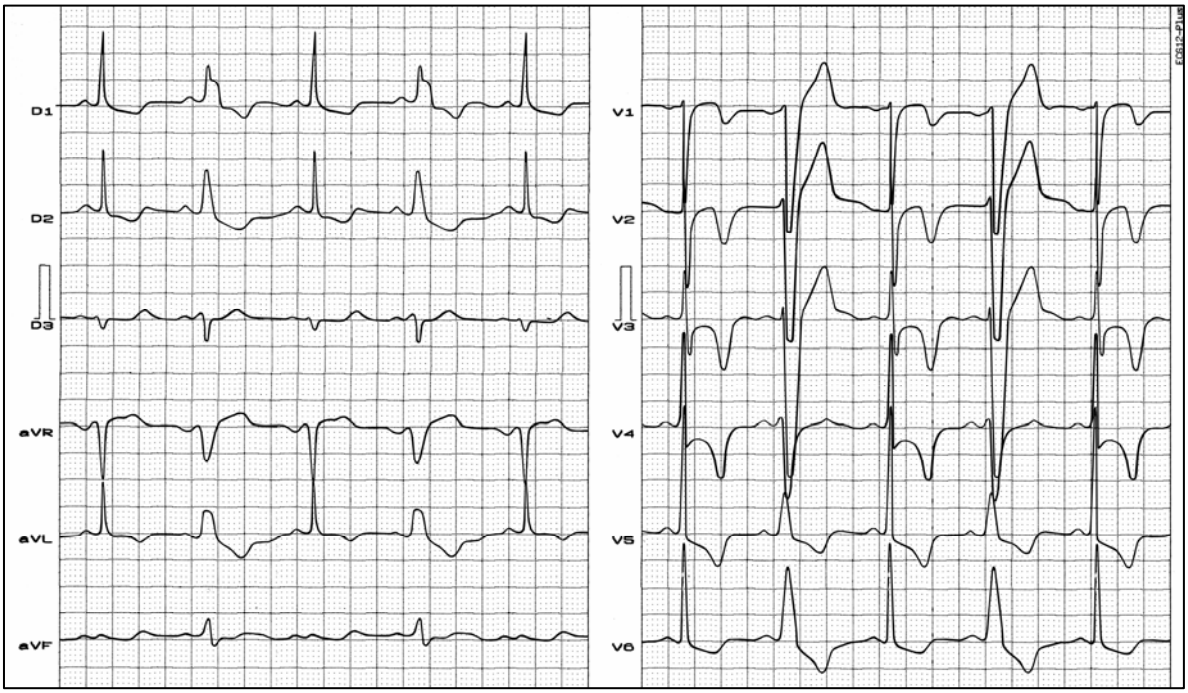
LBBB appears when the HR > 60bpm and cycle length <100ms.

Patients who present with atypical chest pain in whom rate-dependent LBBB develops on the treadmill are significantly less likely to have coronary artery disease than patients who present with classic angina. The onset of LBBB at a HR of ≥125 beats/min is highly correlated with the presence of normal coronary arteries, regardless of patient presentation. Patients with angina in whom both chest pain and LBBB develop during exercise may have normal coronary arteries[9].

The LBBB itself can produce T-wave inversions in the right precordial leads during the normal conduction phase which may simulate an AMI. Persistent deep T wave inversions are seem after return of normal depolarization. The phenomenon is named cardiac memory-persistent T wave changes[10;11;12]

Figure 7

Name: VGA. Sex: Male. Age: 54 y. Race: Caucasian. Weight: 70 Kg
Height: 1.75 m Date: 02/05/2006.



Clinical diagnosis: Coronary insufficiency. Acute myocardial infarction.

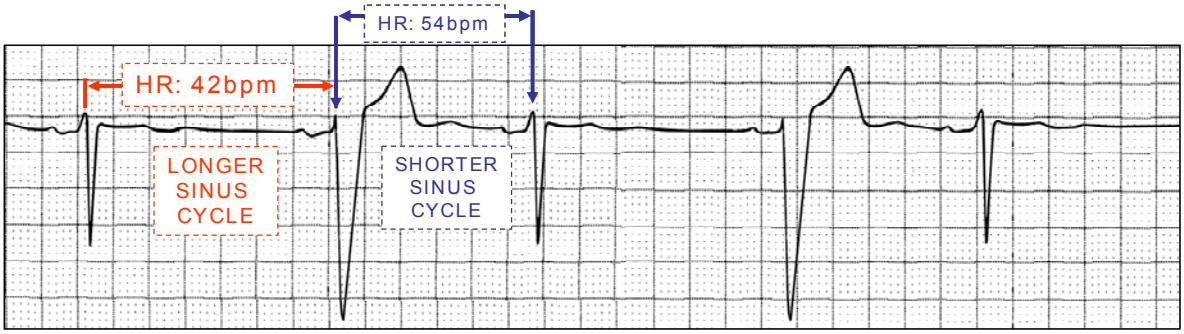
ECG diagnosis: Intermittent LBBB independent from heart rate with bigeminy sequential beat-to-beat: normal conduction and LBBB alternatively. During the normal conduction, ischemic changes masked by the LBBB are seen. Elevated value of biochemical markers was observed.

When the LBBB is intermittent it might be possible to diagnose the AMI during those periods when the conduction is normal. Patients with clinical picture of AMI associated with high HR and LBBB pattern, on the assumption that the block might be rate-dependent, carotid massage with secondary diminution of HR, the LBBB eventually disappear and during the normal conduction ischemic changes masked by the LBBB are clearly seen[13].

III) BRADYCARDIA-DEPENDENT, PHASE 4 ABERRANCY OR DECELERATION-DEPENDENT ABERRANCY

Resulting from the occurrence of impaired intraventricular conduction after long pauses or slowing of the heart to a critical rate. This form of aberrancy is due to a gradual loss transmembrane resting potential during a prolonged diastole with excitation from a less negative take-off potential. During a long pause the fibers of the His-Purkinje system begin to depolarize in an effort to reach threshold potential. By the time the late sinus beat reaches the ventricles, not at all His-Purkinje fibers are negative enough to propagate. Phase 4 block occurs late in diastole and is associated with the cyclical reduction in resting membrane potential that is typical of latent pacemacker cells. In figure 3 the fibers of RBB are activated by the supraventricular impulse but not propagate, which leaves the task of activating the ventricles to the LBB. As a result, an RBBB patter is produce on the ECG.

Figure 8



Phase 4 aberration is observed in 2nd and 4th QRS complex. The longer sinus cycle end with LBBB pattern and the shorter sinus cycle is followed by a narrow QRS complex.

Phase 4 aberration is rare and associated with organic heart disease. Phase 4 aberrancy needs one or more of following situations:

1) The presence of slow diastolic depolarization which need not be enhanced;
2) A shift in threshold potential toward zero.
3) A deterioration in membrane resposiveness so that significant conduction impairment develops at -75mV instead of -65mV;
4) Hypopolarization (the lost of maximum diastolic potential)

RBBB block occurring on alternate beats during regular sinus rhythm, can disappeared during hyperventilation induced increase in HR, and reappeared with slight slowing of the sinus rate due to carotid sinus massage an be caused by bradycardia-dependent RBBB[14].

IV) CRITICAL RATE BUNDLE BRACH BLOCK

This situation is defined as the rate at which BBB develops during acceleration or disappears during slowing. At the fast rate the refractory period shortens; normal conduction tends to be preserved because of this response.

V) CONCEALED INTRAVENTRICULAR CONDUCTION

Concealed Intraventricular conduction is defined as the manifestations of concealed conduction into the bundle branch system or the effect of a non-propagated impulse conduction of a subsequently propagated impulse. Conduction of an impulse through a part of the heart without directs evidence of its presence in the ECG; conduction is inferred only because of its influence on the subsequent cardiac cycle.

Concealed conduction in the human heart usually occurs in the AV node or Hiss-Purkinje system or both. Tissue stimulation without direct effect (such as causing contraction in another chamber), but little is know about the underlying mechanisms.

In AV node concealed conduction acts as a resetting mechanism of the excitability cycle in the slow and fast pathways similar to that expected form a conducted beat[15].

A common example would be interpolated PVC during normal sinus rhythm; the PVC does not cause an atrial contraction, because the retrograde impulse form the PVC does not completely penetrate the AV node. However this AV node stimulation (which is not visible no ECG by itself, hence “concealed”) can cause a delay in subsequent AV conduction by modifying the AV node´s subsequent conduction characteristic. Hence, the PR interval after the PVC is longer than the baseline PR interval[16].

Anterograde concealed conduction into the concealed accessory AV pathway has been postulated to be one of the factors preventing the reciprocicating process via the accessory pathway in patients with the concealed Wolff-Parkinson White syndrome[17]. In these cases, the parallel accessory tract is only capable of conducting the stimulus in a retrograde fashion; i.e. ventriculo-atrial, which overshadows the presence of pre-excitation, given that ventricular activation is processed by the normal Nodo-Hisian normal pathway, originating a normal-duration PR interval. It is important to know the WPW syndrome, because it may predispose the appearance of Supraventricular Paroxysmal Tachycardia runs of the orthodromic macro-reentry type, which use the Node-Hisian system in anterograde fashion and the parallel pathway in retrograde fashion (narrow QRS complexes).

Finally, another variation on concealed conduction concept is seen in atrial flutter. As a result of the rapid atrial rate, some of the atrial activity fails to get through the AV node in an anterograde direction but can alter the rate at which a subsequent atrial impulse is conduced. In this circumstance, an alteration on the “F” wave to QRS relationship is seen.

The following are the possible mechanisms [18]:

1) Trans-septal retrograde concealed intraventricular conduction responsible for:

(1a) Perpetuation of functional BBB initiated by a premature supraventricular impulse;

(1b) Alternation of aberrant ventricular conduction in supraventricular bigeminy;

(1c) Normalization of intraventricular conduction with acceleration or rate in bradycardia-dependent BBB, and

(1d) Prevention of the manifestation of Wenckebach periods of conduction in a bundle branch or fascicle.

2) Anterograde concealed intraventricular conduction responsible for

(2a) Prevention of expected aberrant ventricular conduction when a short cycle follows a long one, and;

(2b) Exceptions to the "rule of bigeminy".

3. Retrograde concealed intraventricular conduction of a ventricular escape in association with unidirectional bundle branch or fasciular block responsible for:

(3a) Resumption of AV conduction in "paroxysmal AV block" with BBB;

(3b) Facilitation (due to supernormality) of conduction in type II AV block due to bilateral BBB.

4. Concealed intraventricular conduction of a premature ventricular impulse responsible for

(4a) Initiation or termination of a re-entrant ventricular tachycardia;

(4b) Resetting of an idioventricular pacemaker, and

(4c) Pseudo-intraventricular or pseudo-AV block.

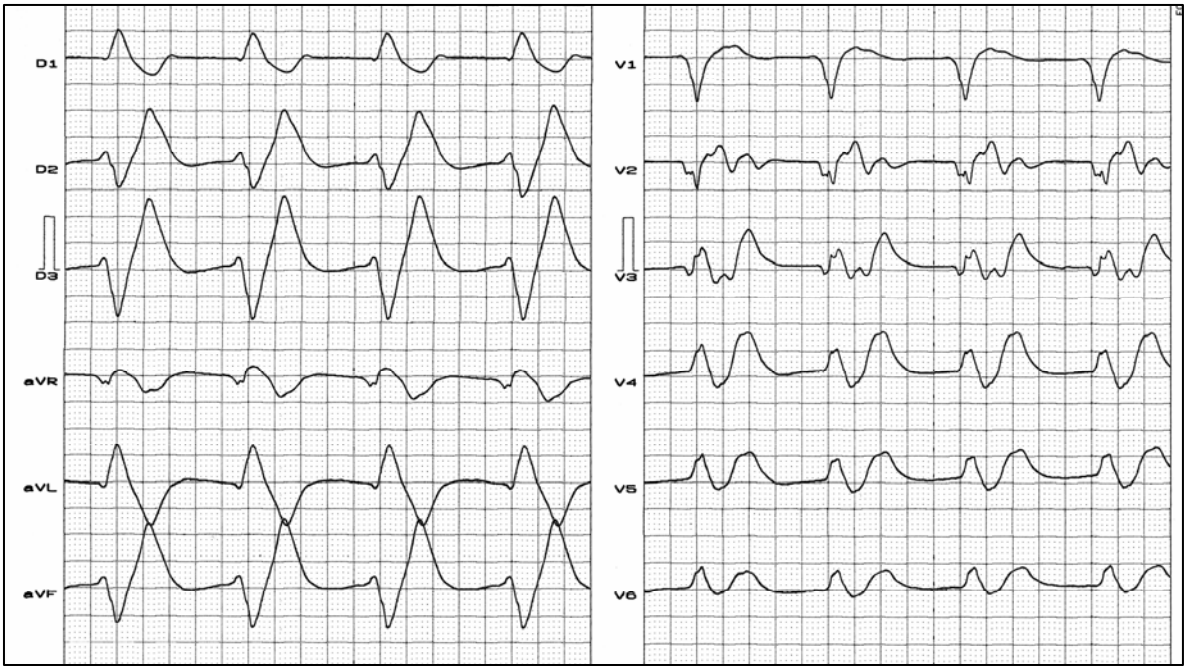
VI) ABERRANCY SECONDARY TO DRUGS AND METABOLIC OR ELECTROLYTE DISORDERS

Aberrant conduction is common during infusion of the I(kr)-blocker almokalant (Class III drugs) during AF, and seems to be more frequent in females and in patients with more advanced myocardial disease[19].

In severe hyperpotasemia (serum potassium between 8 to 9 mEq/l) is frequent observe progressive rhythm and conduction disturbances such as bradycardia, spiked and narrow T waves, widening QRS complex, widening and flattening P wave, disappearance of the P wave, and cardiac arrest. Diffuse QRS complexes widening, similar to left or RBBB, associated to anterior or posterior fascicular block by extreme shift of SAQRS in the FP to left or right is frequently observed. This QRS complex widening is differentiated of genuine branch blocks, because in them, the delay is final or middle, while in hyperpotasemia is always global or diffuse[20].

Figure 9

Typical example of ECG of patient with extremely high level of serum potassium.



Clinical diagnosis: chronic renal insufficiency in dialysis. The patient delayed 72 hours the dialysis session. Hyperpotasemia of 9 mEq/L.

ECG diagnosis: absence of P wave, sinoventricular rhythm, 57 bpm, morphology of bizarre intraventricular severe disorder (QRSd: 240 ms) that is similar to complete left bundle branch block. T waves with polarity matching with QRS from V3 to V6. Convergence of QRS with T wave that outlines smooth diphasic wave or sine curve.

VIII) POSTPAUSAL ABERRANCY OR POSTEXTRASYSTOLIC ABERRATION

This variant is caused probably to slow diastolic depolarization, unequal recovery of conducting or myocardial tissue, or increased diastolic volume.

VENTRICULAR TACHYCARDIA VERSUS SUPRAVENTRICULAR TACHYCARDIA WITH ABERRATION: WIDE-COMPLEX OR BROAD QRS TAQUICARDIAS

Wide-complex tachycardia (WCT) or broad QRS complex tachycardia is defined as a rhythm disturbance with a rate greater than 100 beats/min and a QRS complex duration of 120 ms or more in the adult patient in the pediatric patient, both rate and QRS complex width are age related and caused by various mechanisms, either supraventricular with aberrant intraventricular conduction or ventricular[21]. It is important to differentiate between ventricular (VT) and supraventricular (SVT) because it will determine treatment and prognosis of patients. Unconscious patients with wide complex tachycardia should be treated in a standard cardiac arrest approach. VT is frequently mistaken for supraventricular tachycardia with aberration (SVT). Ectopy is much more common than aberration.

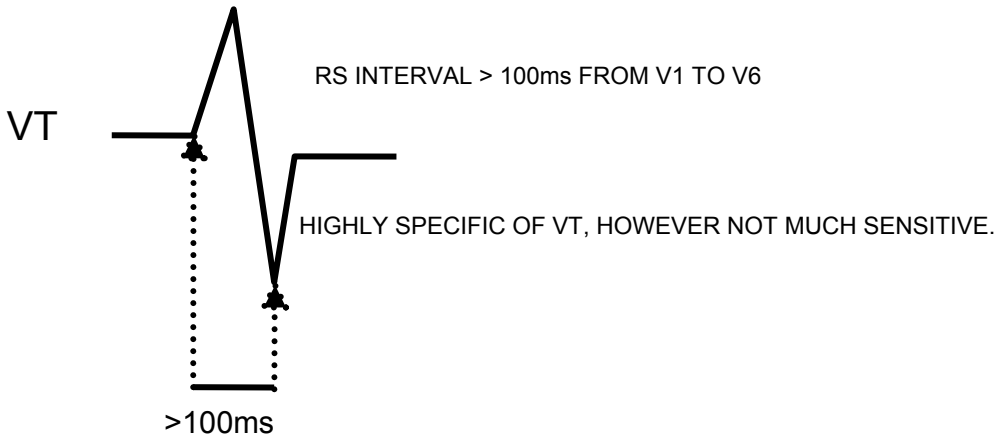
Although up to 10% of cases will defy differentiation, VT and SVT with aberrant conduction can be distinguished utilizing history, physical examination, and ECG criteria.

The positive features in favor of SVT with aberration are:

1) Signs of AV dissociation are infrequent in SVT. Infrequently SVT may also have AV dissociation when retrograde conduction occurs from the junctional focus. The signs of AV dissociation are: irregular cannon waves in the jugular pulse, varying intensity of the first heart sound, and best-to beat changes in systolic blood pressure.
2) Triphasic pattern rsR´ in V1. (monophophasic or diphasic QRS complex in V1 are suggestive of VT);

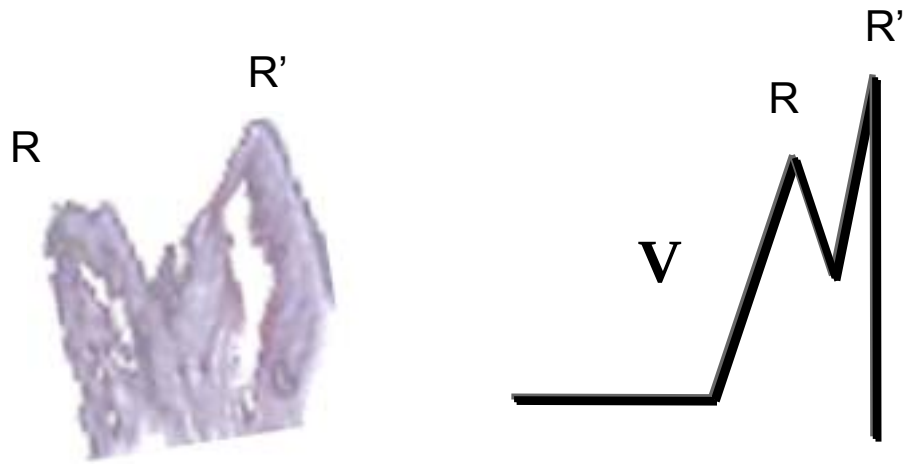
3) qRs pattern in V6;
4) Small, narrow r wave in V1 –V2 during LBBB aberration, if an r wave is present;
5) RS ration in V6 > 1: R>S. (RS ration in V6 < 1: R<S suggest VT.). If the R to S interval were >100 ms in some precordial lead, it is highly specific of VT; however, it is not much sensitive.

Figure 10



6) QRS complex < 140ms. CRBBB pattern, QRS >140 ms = VT < 140 ms = SVT with aberrant conduction. Pattern of CLBBB >160ms = VT. Duration < 160 ms = SVT with aberrant conduction[22; 23]. Unfortunately Measurement of QRS duration is difficult, mainly in the presence of obvious notching, slurrings and terminal slow vectors, can be operator dependent and influenced by the presence of conduction abnormalities which reduce its accuracy and reproducibility[24].
7) Early S nadir in V1-V2 ≤ 60ms form the onset of the QRS complex(In VT the distance form the onset of the QRS complex to the lowest point of the S wave(its nadir) is > 60ms);
8) Swift smooth S Dow stroke in V1-V2 reflecting conduction in the His-Purkinje system;
9) Absence of the rabbit ear clue: rR: Left peak taller than the right. The voltage of the R wave in V1 is less than R': "Rabbit Ears".

Figure 11



RR': R' > R SIGN OR RABBIT CLUE EARS.

10) Preceding atrial activity;
11) Initial deflection identical with that of conducted beats if RBBB;
12) Absence of negative complexes from V4 to V6 .
13) Absence of QR from V2 to V6.
14) SAQRS between < 60 degree and > 150degree is frequent in VT.
15) Second-in-the-row anomalous beat;
16) Alternating BBB patterns separated by single normally conducted beat;
17) Preexisting BBB with identical QRST pattern.
18) When in doubt about the origin use intravenous procainamida, do not use verapamil, because the last drug can to occasion hemodynamic deterioration.
19) It seems to be easy to distinguish the two forms in patients with preexisting BBB: if the QRS morphology during tachycardia is identical to those during at rest the tachycardia is supraventricular, if different, ventricular. It is generally assumed that if a wide QRS complex tachycardia has the same morphology on the 12-lead ECG as during sinus rhythm, the tachycardia is supraventricular. VT can occur with the same QRS complex morphology as occurs during sinus rhythm [25; 26].
20) The typical ECG criterion for a diagnosis of supraventricular tachycardia of BBB (left = rS or QS wave in leads V1 and V2, delay to S wave nadir <

70 ms, and R wave and no Q wave in lead V6; right = rSR' wave in lead V1 and an RS wave in lead V6, with R wave height greater than S wave depth) These criteria, which require only knowledge of typical bundle branch block patterns, were highly sensitive for the important diagnosis of VT[27].

Observation: Fascicular VT Left ventricular idiopathic VT and bundle branch reentry VT may also have the triphasic pattern in V1 and relatively narrow QRS complex.

There are several causes of broad-complex tachycardia, even in patients with previous myocardial infarction, and, where doubt exists, electrophysiologic studies should be performed. Antidromic atrioventricular reentrant tachycardia can to mimic a VT[28].

Atriofascicular pathway-mediated antidromic tachycardia using atriofascicular pathway as the antegrade limb and the atrioventricular nodal pathway as retrograde limb should be considered in differential diagnosis of all cases of wide complex tachycardia with LBB morphology and left axis[29]. The most common type of accessory pathway causing a wide complex tachycardia is the atrioventricular bypass tract. Distinguishing the accessory pathway-mediated tachycardia from VT or SVT with aberrancy is often difficult, but has important clinical consequences. Despite sophisticated atrial and ventricular pacing techniques used during electrophysiologic study, the exact mechanism of some wide-complex tachycardia remain with inconclusive results from diagnostic. In this situation, eliminating conduction from specific tissue and observing the effect on the tachycardia can be diagnostic. Such a maneuver can be performed using the technique of ice mapping, which entails reversible cooling of tissue to test function prior to delivery of a permanent ablation lesion[30].

If the ECG reveal wide QRS tachycardia with a narrow complex beat during a wide complex tachycardia suggests a capture or fusion beat in the setting of VT. However, there are situations where SVT can also manifest this way[31].

Patients who are misdiagnosed with VT because of ECG artifact may be subjected to unnecessary procedures.

Physicians (n = 766) were surveyed with a case simulation that included a two-lead electrocardiographic monitor tracing of artifact simulating a wide-complex tachycardia. The rhythm strip was not recognized as artifact by 52 of the 55

internists (94%), 128 of the 221 cardiologists (58%), and 186 of the 490 electrophysiologists (38%). 156 of the 181 electrophysiologists (88%), 67 of the 126 cardiologists (53%), and 14 of the 15 internists (31%) who misdiagnosed the rhythm as VT recommended an invasive procedure for further evaluation or therapy. ECG artifact that mimics VT may frequently result in patients being subjected to unnecessary invasive cardiac procedures. Physicians should include artifact in their differential diagnosis of wide complex tachycardias to minimize unneeded procedures[32].

References

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