Aberrant ventricular conduction types and concealed conduction

Others denominations: Aberrancy, ventricular aberration - 2009

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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 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) Excitation prior to completion of repolarization;
2) Unequal refractoriness of conducting tissue resulting in local delay or block of conduction;
3) Prolongation of Action potential (AP) due to prolongation of the preceding cycle length;
4) Failure of restitutions of transmembrane electrolyte concentration during diastole;

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 oif bundle branch conduction and
8) Diffuse depression of Intraventricular conduction including that of specialized as well as contractile myocardial.

I) ABERRANCY SECONDARY TO GOUAUX-ASHMAN PHENOMENON OR ASHMAN PHENOMENON[5]

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 beats.

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 right bundle branch.

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[5] 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;
2) Atrial tachycardia;
3) Premature Atrial Contractions.

Ashman phenomenon is an aberrant ventricular conduction due to a change in QRS cycle length. In 1947, Gouaux and Ashman reported that in atrial fibrillation, 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[6].

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[7].

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.

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 2

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[4]. The LBBB itself can produce T-wave inversions in the right precordial leads during the normal conduction phase which may simulate an AMI[5]. Persistent deep T wave inversions are seem after return of normal depolarization. The phenomenon is named cardiac memory-persistent T wave changes[10;11]

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[12].

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.

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)

Figure 3

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[13].

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 direct 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[14].

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[15].

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[16]. 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 [17]:

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[18].

In severe hyperpotasemia (serum potassium between 8 to 9 mEq/l) 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[19].

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.

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