Concealed Conduction

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1. Introduction

Concealed conduction is a phenomenon which could be rarely seen on the surface of electrocardiography (ECG). The surface ECG must be evaluated carefully for determination of concealed conduction. Because concealed conduction manifests itself in different shape and level. This evaluation could prevent misdiagnosis and unappropriate treatment. In this chapter we want to contribute to understanding this phenomenon.

2. Historical perspective

Concealed conduction of sinus impulse at the level of the AV node was documented in animal models in 1925 (1). Nearly 20 years after the animal models, concealed conduction was shown in human heart by Langendorf et al (2). Initial animal and human models showed that concealed conduction occurred in the sinus node, around the sinus node, atrium, AV node and Hiss-Purkinje system (3-5). Following years many studies was published about the effect and electrophysiological mechanism of concealed conduction.

3. Concealed conduction mechanism

A number of investigators had since observed similar phenomena but it was not until the studies of Langendorf and Pick that the concept of concealed conduction was firmly established in electrocardiology (6,7). A wide variety of phenomenology has been described as consequences of penetration of impulses that do not emerge from the AV node (8). Some of these: delay of conduction of a succeeding propagated response, block of a succeeding atrial impulse that occurs at a time when the AV node should have been excitable, delay of the expected discharge of an AV nodal pacemaker. When an impulse penetrates the AV node but it does not traverse completely, at one point it becomes subthreshold for cells located distal to the site of block. This subthreshold event may be manifested in the distal cells as an electrotonic depolarization at a distance that depends on the membrane resistance and the degree of electrical coupling among cells.

It was demonstrated initially by Antzelevitch and Liu (9,10) many of the phenomena described in terms of concealed conduction may be a consequence of the inhibitory action of subthreshold electrotonic potentials on subsequent responses in the Purkinje fiber and numerical experiments in models of AV nodal cells. Thus, electrotonic inhibition explain the

clinically observed phenomenon of concealed AV conduction. Subthreshold potentials in the AV node may result in delay of excitation or even failure of responses occurring later in time, that at least some variants of concealed AV conduction may be explained by electrotonically mediated reduction of $I_{Ca.T}$ (10).

Electrotonic inhibition and concealed conduction are widespread in cardiac tissues. However, we have very little knowladge about ionic mechanisms of electrotonic inhibition and concealed conduction. Electrotonic inhibition of excitability has been demonstrated in ventricular myocytes. Indeed, repetitive stimulation in myocytes, with depolarizing current pulses of threshold amplitude may elicit action potentials in a one-to-one manner. Furthermore, subthreshold pulse during a single diastolic interval may lead to a transient decay in excitability and even to complete failure of subsequent excitation.

Liu et al, demonstrated that the specific role of $I_{Ca.T}$ in the dynamic modulation of AV nodal excitability by premature inputs and suggest a plausible ionic mechanism for the regulation of action potential propagation through the AV nodal conducting system (10). Premature depolarization can modulate the amplitude of the transient calcium current, the time course of such modulation is compatible with the diastolic time window during which electrotonic inhibition is permissible. The modulation is directly associated with a decrease in $I_{Ca.T}$ (10). I_F may be responsible for electrotonic inhibition in Purkinje fibers (9). Previous experiments in pig ventricular myocytes have shown that diastolic excitability is modulated by the long time course of deactivation of I_K (11). If conditioning pulse intervals were much longer than expected intervals for complete deactivation of I_K , electrotonic inhibition occurred. Under such conditions, the basis of changes in the kinetics of $I_{Ca.T}$ may easily explain this phenomenon.

4. Concealed conduction

The concealed conduction, is an extra impulse from the heart which penetrate the electrical system of heart on refractory period, regardless the impulse change characteristics of conduction system, it does not cause any contraction. The effect on subsequent events is an important part of the definition of concealed conduction because it differentiates the concept of concealed conduction from other forms of incomplete conduction, such as conduction blocks. It should be recalled that although the ECG reflects the electrophsiological properties of the myocardium, alterations of conduction more often reflect electrophysiological changes in the specialized tissues, the activity of which is not recorded on the surface ECG.

The concept of concealed conduction, was introduced before (6,7), an explanation for the effects of incomplete penetration of an impulse into a portion of the A-V conduction system. The phenomena could observe on the surface ECG, but that was incompletely penetrating impulse and not directly reflected on the surface ECG; hence the term *concealed*.

The manifestations of concealed conduction are numerous, because of variations of the site of impulse formation, the different effects of the anatomical site, the direction of the concealment (e.g., the heart rate, automatic tone, drugs, or electrolyte balance).

The manifestations of concealed conduction include;

a.   prolongation of conduction,
b.   failure of propagation of an impulse,
c.   facilitation of conduction by "peeling back" refractoriness, directly altering refractoriness, and/or summation (12,13), and
d.   pauses in the discharge of a spontaneous pacemaker.

The concealed conduction usually occur in AV node and/or His-purkinje system. The conduction of AV node is silent, so that does not occur any deflexion on surface ECG. The conduction of AV node can be determined by measuring duration of PR. Sometimes, an extra impulse is originated from atria or ventricle which incompletly penetrate in AV node because of refractoriness of the AV node. The incomplete penetration of extra impulse to AV node cause prolongation of the PR interval or blockage of AV conduction (figure 1,2,3,4).

A common example may be interpolated premature ventricular contraction (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 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. (figure 1,3).

Fig. 1. Sinus rhythm and bigeminal PVC. P waves which follow PVC are blocked, Lewis diagram: A: Atrium, AV: Atrio ventricular, V: Ventricle.
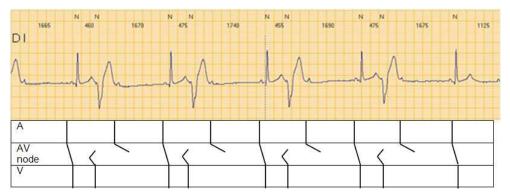

Fig. 2. First and second narrow QRSs are followed by PVCs with progressive prolongation of the PR interval and the blockage of the third sinus beat (Mobitz type I AV block). The third sinus beat is obscured by the T wave. The third and fourth narrow QRSs are also followed by PVCs and PR interval shows progressive prolongation. Since the fifth narrow QRS is not followed by a PVC, this prolongation does not end with the blockage of the next (7th) P wave, which is conducted with a normal PR interval. First, third, fourth, and fifth PVCs are interpolated, Lewis diagram: A: Atrium, AV: Atrio ventricular, V: Ventricle.
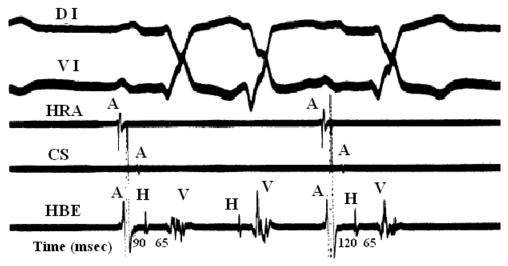
Fig. 3. Because of retrograde concealed conduction by a fascicular extrasystole (second beat) the next sinus impulse A-H interval is longer than before.
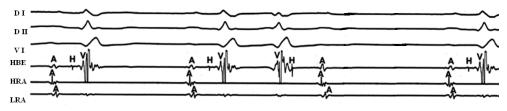

Fig. 4. Concealed conduction by PVC (third beat) the subsequent sinus complex is a block in the A-V node (third atrial beat).

Another example on concealed conduction concept is seen in atrial flutter and fibrillation. Some of the atrial activity fails to get through the AV node in an anterograde direction as a result of the rapid atrial rate. A long RR interval after repeated concealments is usually followed by an AV junctional escape impulse. If the RR is longer than the expected junctional escape interval, a concealed impulse of the junctional pacemaker could be suspected.

In some cases, concealed conduction (extra impulse is orginated from atria or ventricle which has incompletly penetrated in AV node) repair the AV node refractory period and subsequent impulse conduct with normal or short duration of PR. This phenomen is called ''peeling'' or peeling back of the refractory period (figure 5).

Concelaed conduction may be seen in bundle branch system. Functional bundle branch block may be seen as a result of rapid increase of ventricular rate and bundle branch block could continue until ventricular rate return to baseline heart rate. The functional bundle branch block may be explained by transseptal concealed conduction (figure 6). Atrial impulse is conducted to the ventricle via a branch which is in non refractory period and the other branch of conduction system is stimulated via transseptal concealed conduction which

is in refractory period during atrioventricular conduction. The next atrial impulse is conducted by the same branch because of ongoing refractory period of the other branch. This stituation causes vicious cycle. This vicious cycle has been continued until heart rate decreases to critical ventriculer rate which elicits refractory period of all branches of cardiac conducting system.
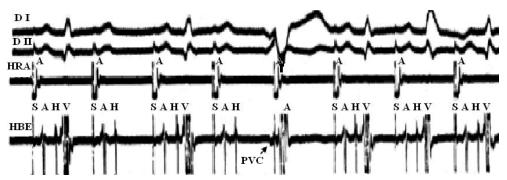

Fig. 5. During atrial pacing at a cycle length of 400 msec, 2:1 block below the His bundle occurs. After a PVC (arrow, the fifth complex), the P wave that block in the 2:1 sequence conducts to ventricle as other conducted complexes. The PVC repair the AV node refractory period and subsequent impulse conduct with normal duration. (S: stimulation, A: atrium, H: His, V: Ventricle).
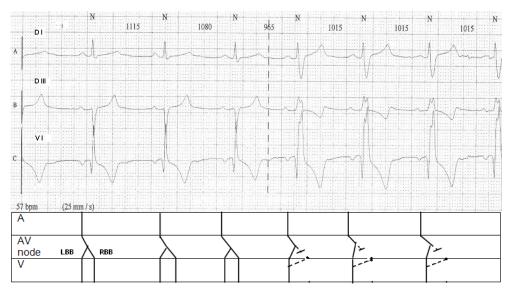

Fig. 6. Functional bundle branch because of increased heart rate and concealed transseptal conduction from LBB to RBB, resulting RBBB. Lewis diagram: A: Atrium, AV: Atrio ventricular, V: Ventricle, LBB: left bundle branch, RBB: right bundle brunch.
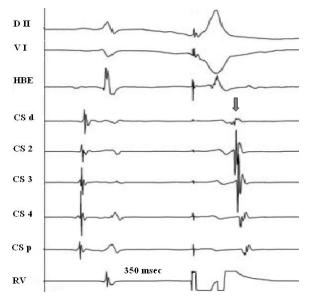
Fig. 7. Right ventricular extrastimulus with 350 msec coupling interval showing retrograde conduction over a left-sided accesory pathway (arrow) (because of short coupling interval the concealed conduction can not arrive to accesory pathway and accesory pathway can be suitable for retrograde conduction).
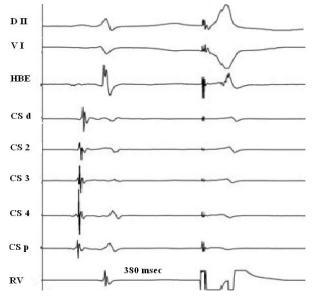

Fig. 8. Right ventricular extrastimulus with 380 msec coupling interval showing no retrograde conduction (the concealed conduction to accesory pathway prevents the retrograde conduction which cause resiprocating tachycardia).

One of circumstance related with concealed conduction is WPW sendrome. Atrial stimulation is conducted to the ventricle by passing AV node and lead to resiprocating tachycardia result of recycling retrograde conduction via accesory pathway (figure 7). However, concealed conduction to accesory pathway causes refractory period of accesory pathway and inhibition of retrograde conduction of accesory pathway, so the concealed conduction to accesory pathway prevents resiprocating tachycardia (figure 8).

The concealed conduction to the sinus node because of an extra atrial impulse (the impulse changes sinus node pacemaker function) lead to delay in the next stimulus of the sinus node. As a result, this event represents itself with a longer duration of PP than the previous duration of PP on surface ECG (figure 9).

Fig. 9. Lewis diagram: Concealed conduction to sinus node. A: Atrium, AV: Atrio ventricular, V: Ventricle.

5. Summary

Heart conduction system sites and mechanisms of concealed conduction.

- Sinüs node:
  Changing the sinüs node pacemaker function by concealment of an extra atrial impulse; lead to delay the next stimulus.
- AV node:
  1-Anterograde AV node concealment manifest with;
  a.   Delay of AV conduction
  b.   Block of AV conduction
  c.   Block of retrograde VA conduction
  d.   Slowing of ventricular rate due to concealment of atrial fibrillatory waves
  e.   Peeling back of the AV node refractory period and subsequent impulse conduct with normal or short duration of PR.
  2-Retrograde VA concealment manifest with;
  a.   Delay of AV conduction
  b.   Block of AV conduction
  c.   Peeling back of the AV node refractory period and subsequent impulse conduct with normal or short duration of PR.
- Bundle branch system:

Functional bundle branch block because of rapid ventricular rate and the other branch of conduction system is stimulated via transseptal concealed conduction.
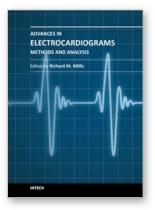
- Accesory pathway:
  Concealed conduction to accesory pathway inhibition the retrograde conduction of accesory pathway. The concealed conduction prevents tachycardia which is related with accesorf pathway.

6. Disclosure

Authors have no relevant conflict of interest to disclose

7. References

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Electrocardiograms are one of the most widely used methods for evaluating the structure-function relationships of the heart in health and disease. This book is the first of two volumes which reviews recent advancements in electrocardiography. This volume lays the groundwork for understanding the technical aspects of these advancements. The five sections of this volume, Cardiac Anatomy, ECG Technique, ECG Features, Heart Rate Variability and ECG Data Management, provide comprehensive reviews of advancements in the technical and analytical methods for interpreting and evaluating electrocardiograms. This volume is complemented with anatomical diagrams, electrocardiogram recordings, flow diagrams and algorithms which demonstrate the most modern principles of electrocardiography. The chapters which form this volume describe how the technical impediments inherent to instrument-patient interfacing, recording and interpreting variations in electrocardiogram time intervals and morphologies, as well as electrocardiogram data sharing have been effectively overcome. The advent of novel detection, filtering and testing devices are described. Foremost, among these devices are innovative algorithms for automating the evaluation of electrocardiograms.

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