REVIEW ARTICLE

Old teaching tools should not be forgotten: The value of the Lewis ladder diagram in understanding bigeminal rhythms

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Abstract

As medical education evolves, some traditional teaching methods often get forgotten. For generations, the Lewis ladder diagram (LLD) has helped students understand the mechanisms of cardiac arrhythmias and conduction disorders. Similarly, clinicians have used LLDs to communicate their proposed mechanisms to their colleagues and trainees. In this article, we revisit this technique of constructing the LLD and demonstrate this process by describing the mechanisms of various bigeminal rhythms.

KEYWORDS

bigeminal rhythm, escape-capture bigeminy, Lewis' diagram, Wenckebach phenomenon

1 | INTRODUCTION

The Lewis ladder diagram (LLD) is a technique of graphically representing the electrical activation sequence of the heart. It helps clinicians and learners visualize and communicate mechanisms of arrhythmia. The invention of ladder diagrams is often credited to Thomas Lewis; however, Lewis derived his illustration using existing diagrams as templates (Surawicz, 2003). Similar diagrams were originally used by many scholars including Auguste Chauveau in France, Theodore Wilhelm Engelmann in The Netherlands, Wenckebach in Germany, and Thomas Mackenzie in England. A

thorough review of the history of the LLD has been previously published (Johnson & Denes, 2008).

A bigeminal rhythm is defined as a regularly irregular rhythm wherein the ECG demonstrates pairs of QRS complexes in which a short R-R interval is followed by a long one. This sequence repeats successively, sometimes over a long period of time. (Baranchuk, Miranda, Rana, & Michael, 2012; Antoni Bayés de Luna & Baranchuk, 2017; López Díez, Femenía, & Baranchuk, 2010; Sadiq & Baranchuk, 2016; Varriale, Gul, Ali, Glover, & Baranchuk, 2016). In this article, we will use the Lewis ladder diagram (LLD) to illustrate the electrophysiologic mechanisms behind five different

bigeminal rhythms that we encountered in our clinical practice (Johnson & Denes, 2008; Lewis, 1913).

2 | CONSTRUCTION OF THE LLD

In order to apply the LLD to rhythm disturbances, it is first necessary to identify the atrial and ventricular activation waves and postulate how the electrical stimulus is conducted from one chamber to the next (Bayés de Luna & Baranchuk, 2017; Johnson & Denes, 2008; Lewis, 1913) Figure 1.

A basic LLD usually consists of 3 lines that represent 3 areas of the heart through which the stimulus is conducted (A = atria, V = ventricles, and AV = AV junction) (Figure 1a). LLD can be expanded to emphasize certain electrophysiologic structures. For example, additional lines can be inserted to represent the sinus node (SN) and the sinoatrial node junction (SA-J) (Figure 1b). Similarly, the AV junction can be divided into the AH and HV zones (Figure 1c). The ventricular zones can also be further expanded into the right branch (RB) and left branch (LB) to illustrate bundle branch conduction abnormalities. The origin of the electrical current is usually indicated with a point for both sinus and ectopic rhythms (Figure 1d).

3 | LLDS OF BIGEMINAL RHYTHMS

In this section, we will illustrate various bigeminal rhythm patterns utilizing the LLD.

3.1 | Sinus rhythm and atrial extrasystoles

The ectopic P wave (P') has a different morphology than the one of sinus origin (Figure 2). The key to recognizing this rhythm is to identify the ectopic premature P wave. It is important to note that the ectopic focus can originate in the crista terminalis, adjacent to the sinus node, producing P waves that have similar morphology to the sinus P waves. Premature P waves commonly hide in, or fuse with, the T waves of the preceding QRS complexes. One must learn to

detect hidden P waves in deformed T waves that look different than the T waves of flanking beats, as exemplified in this tracing. Most commonly, ectopic atrial impulses conduct into the sinus node area and reset the sinus node action potential. This results in a "noncompensatory pause" that typically follows a PAC, as seen in Figure 2.

Figure 2 also demonstrates right bundle branch aberrancy following a shorter R-R interval. This is a well-described phenomenon caused by a long R-R interval in the preceding cycle. The longer R-R interval prolongs the refractory period of the bundles. The right bundle branch often has a longer refractory period than the left. The PAC that occurs in the subsequent cycle (short R-R interval) activates both bundles, but the right bundle has not yet recovered and fails to conduct resulting in the right bundle branch block pattern. Such RBBB aberrancy after a long-short sequence is commonly known as the Ashman phenomenon (Lakusić, Mahović, & Slivnjak, 2010).

3.2 | Second-degree 3:2 Wenckebach AV block

This tracing represents bigeminy wherein progressive prolongation of the PR interval is seen until a P wave is not conducted to the ventricles (Nadeau-Routhier & Baranchuk, 2016) (Figure 3). This is the most common bigeminal rhythm after atrial and ventricular extrasystoles. This tracing also demonstrates the *sine qua non* of AV Wenckebach: The recovery PR interval after the nonconducted P wave is always shorter than the last conducted PR interval just before the nonconducted P wave.

3.3 | Sinus rhythm and ventricular extrasystoles

Premature ventricular complexes (PVCs) commonly occur in a bigeminal pattern as shown in Figure 4. If the AV node is capable of retrograde conduction, retrograde P waves may deform the ST segment or T wave of the PVC, as seen in this tracing. When the ambient sinus rate is slow, retrograde conduction may rarely reset the sinus node, resulting in a noncompensatory pause. More commonly, this does not occur and PVCs are followed by compensatory pauses that indicate that the sinus node automaticity has not been affected by retrograde conduction. In the setting of bigeminy, as in this figure,

FIGURE 1 Examples of the Lewis ladder diagrams (LLDs). A, atria; AV-J, atrioventricular junction; EF, ectopic focus; SA-J, sinoatrial node junction; SN, sinus node; V, ventricles

FIGURE 2 Sinus rhythm, atrial extrasystoles, and Ashman phenomenon (Courtesy: Dr. G Veenhuyzen)

FIGURE 3 Second-degree 3:2 Wenckebach AV block

FIGURE 4 Sinus rhythm and ventricular extrasystoles

the effect of PVCs on the underlying sinus automaticity may be impossible to judge because sinus impulses are too few to accurately measure their pattern.

3.4 | Unusual bigeminy formed by escape QRS complexes followed by a sinus P wave

This tracing shows three pairs of ventricular complexes in a bigeminal pattern (Sadiq, Chiale, Bayes de Luna, Conde, & Baranchuk, 2014) (Figure 5). The first QRS complex of each pair is not preceded by a P wave and is likely an escape complex originating in the AV node because it is narrow and is preceded by a long pause. The first QRS complex of each pair is followed by a P wave that is very likely of sinus origin ($\pm$ in lead V1). These P waves follow the escape complexes at variable coupling intervals, indicating they may not be related mechanistically. The P waves are also regular (see equal A-B and B-C intervals) and represent a very slow baseline sinus rate of 25 bpm. The second QRS in the first and third pair (A and C), likely represent conducted sinus beats with a long PR interval. The P wave in the second QRS pair (B) is followed by a shorter PR

interval and a QRS complex of different morphology. We propose that this QRS complex is likely an ectopic beat that came early, depolarized the ventricles, and terminated in the AV node when it met the descending sinus wavefront. In this example, we use a LLD to communicate our proposed mechanism. However, we do acknowledge that other explanations are possible to explain this tracing.

3.5 | 3:2 Sinoatrial block versus para-sinus atrial bigeminy

This tracing demonstrates bigeminy wherein the shorter R-R interval (right side of the tracing) exactly matches the basal rate (left side of the tracing) (Bayés de Luna et al., 1991) (Figure 6). Also, the longer R-R interval of the bigeminy is twice the interval of the shorter R-R interval. This suggests that the sinus node likely fired within the longer R-R interval (i.e., C and D), but failed to exit the sinus region into the atria, hence the P wave is absent. The next sinus activation exits to the atria, producing a P wave that comes exactly on schedule. The activation of the sinus node does not manifest on the

FIGURE 5 Unusual bigeminy formed by escape QRS complexes followed by a sinus P wave

FIGURE 6 Sinoatrial block (Bayés de Luna et al., 1991)

surface ECG, and we must infer its activation by measuring P-wave intervals. Note that the subsequent P wave follows on schedule without interruption.

Figure 6 also demonstrates an interesting phenomenon. It appears that the P-P intervals progressively shorten before the sinus exit block takes place. This likely represents a Wenckebach phenomenon of the conduction between the sinus node and the atrium. Just like R-R interval decrements in AV nodal Wenckebach, sinoatrial exit Wenckebach manifests as a decrement in the P-P interval.

4 | DISCUSSION AND CONCLUSION

The LLD is an effective way to diagram the proposed mechanisms of arrhythmias observed on ECG tracings. In this work, we showcase various bigeminal rhythms and illustrate the proposed mechanism of each rhythm using LLDs. Often the exact mechanism cannot be determined using a surface tracing. In such cases, a differential diagnosis must be formulated and narrowed as additional information is gathered. Some cases require intracardiac recordings to delineate the mechanism. The LLD has stood the test of time and should not be forgotten as electrocardiographers hasten to use electronic methods to teach new learners interpretation of this century-old technology (Antipervitch et al., 2018; Nadeau-Routhier & Baranchuk, 2016).

CONFLICTS OF INTEREST

None to declare.

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