Intermittent Left Bundle Branch Block: An Overlooked Cause of Electrocardiographic Changes That Mimic High-Grade Stenosis of the Left Anterior Descending Coronary Artery

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Context: The electrocardiographic (ECG) pattern of high-grade stenosis of the left anterior descending coronary artery (LAD) is important clinically because of the high risk of myocardial infarction and cardiac death if the pattern is not recognized. Although the recognition of this pattern is currently widespread, false-positive ECG changes that mimic this pattern are infrequently reported.

Objective: To demonstrate that ECG changes from intermittent left bundle branch block (LBBB) and cardiac memory can mimic anterior ischemia.

Methods: Medical record review of cardiology patients in whom ECG tracings showed intermittent LBBB and anterior T-wave changes during normal QRS conduction. Patients were included if ECG changes suggestive of high-grade LAD stenosis in leads V_2 and V_3 met the following criteria: (1) the QRS conduction was essentially normal during periods of absent LBBB; (2) the ST segment took off from an isoelectric point or only slightly elevated from baseline; and (3) the ST segment sloped up gradually with an abrupt and sharp down stroke leading to terminal T-wave inversion. Additional criteria were little or no ST segment elevation, no loss of precordial R waves, and ECG changes suggestive of high-grade LAD stenosis demonstrated in precordial leads V_2 and V_3. All patients demonstrated intermittent LBBB, and patients were excluded if a ventricular pacemaker was present. The case series began in 2003 and continued until 2011.

Results: Sixteen patients (3 male) with intermittent LBBB were identified with ST- and T-wave changes during normal ventricular conduction that matched the pattern described by Hein J.J. Wellens, MD. Of these patients, none had evidence of clinically substantial coronary artery disease. Eleven patients had stress testing with myocardial perfusion imaging, and 5 patients underwent cardiac catheterization. In 1 patient whose ECG pattern showed high-grade LAD stenosis but normal coronary arteries at catheterization, a stress test was later performed, which provoked LBBB. All other patients had spontaneous, intermittent periods of LBBB and normal conduction.

Conclusion: The ECG pattern of high-grade LAD stenosis has proven to be an important marker of high-risk patients with chest pain. This pattern may also be seen in patients with a right ventricular pacemaker on resumption of native QRS conduction. Intermittent LBBB is a less obvious cause of a similar ECG pattern that may mimic anterior ischemia due to high-grade stenosis.

In 1982, Hein J.J. Wellens, MD, described characteristic patterns on electrocardiograms (ECG) that are indicative of critical stenosis of the left anterior descending coronary artery (LAD) and impending myocardial infarction.[1] This pattern was found when patients were not experiencing chest pain, and it consists of symmetrical T-wave inversion that begins in the terminal portion of the T wave (*Figure 1*). These ST- and T-wave changes are typically present in leads V_2 and V_3, but they can involve other precordial leads as well. This pattern is known as *Wellens syndrome* or *Wellens warning*.

Cardiac memory is another term describing characteristic ECG changes involving the ST and T waves. The concept was first reported in 1969 in relation to ventricular pacing.[2] The term, however, was first introduced in 1982 by Rosenbaum et al.[3] Cardiac memory involves reversible ST- and T-wave changes and is induced by an abnormality of the electrical activation pattern such as ventricular pacing or intermittent left bundle branch block (LBBB).[1,4,5]

Wellens syndrome and the ECG pattern of LBBB-induced cardiac memory can appear similar, if not identical. However, one of these patterns carries the prognosis of an impending myocardial infarction, while the other is benign in nature. Therefore, it is clinically relevant to distinguish these 2 ECG patterns, when possible. The current case series review was conducted to identify how ECG changes from intermittent LBBB can mimic anterior ischemia.

Figure 1.
Example of the classic electrocardiographic pattern described by Hein J.J. Wellens, MD. This pattern indicates critical stenosis of the left anterior descending coronary artery.

Methods

This retrospective review of medical records was exempt from institutional review board approval. All patients were from the same institution. Some patients had multiple ECGs. The ECGs chosen were those that showed LBBB and those that best demonstrated the characteristic changes when the LBBB terminated. A single cardiologist (F.J.R.) collected 12-lead ECGs from patients with intermittent L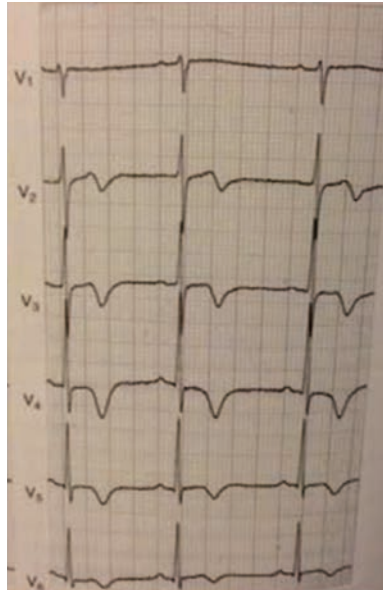BBB and anterior T-wave changes consistent with ischemia during normal QRS conduction. Patients were included if ECG changes suggestive of high-grade LAD stenosis in leads V_2 and V_3 met the following criteria: (1) the QRS conduction was essentially normal during periods of absent LBBB; (2) the ST segment took off from an isoelectric point or only slightly elevated from baseline; and (3) the ST segment sloped up gradually with an abrupt and sharp down stroke leading to terminal T-wave inversion. Additional critera were little or no ST segment elevation and no loss of precordial R waves. Patients with ventricular pacemakers were excluded. The standard definition of LBBB was used[6] (QRS duration >120 milliseconds, eliminating normal septal Q waves in lateral leads; tall R waves are produced in the lateral leads [I, V_5-V_6] and deep S waves in the right precordial leads [V_1-V_3]; and broad or notched R waves in the lateral leads).

Clinical information was obtained from the medical records of clinic or hospital patients who met the study criteria and were examined between 2003 and 2011.

Results

Sixteen patients were included, all of whom presented with or were referred for chest pain. Of these 16 patients, 13 were women. The ages ranged from 29 to 87 years. All patients had been evaluated for ischemia with either myocardial perfusion imaging or cardiac catheterization. Of the 16 patients, 13 did not have previously diagnosed intermittent LBBB, and the intermittent LBBB was often not present on the same day that the ECG changes consistent with ischemia were present. Three patients had no history of intermittent LBBB until stress testing provoked the intermittent or rate-dependent LBBB. One patient demonstrated classic ischemic ST- and T-wave changes after resolution of the LBBB, but on the next occasion of intermittent LBBB resolution, the ST- and T-wave changes were not present. Owing to the spontaneous and intermittent nature of the LBBB, we were unable to observe the natural history, such as the duration of LBBB necessary for these changes to manifest. Eleven of the 16 patients were evaluated with myocardial perfusion imaging to assess for ischemia. The remaining 5 patients underwent cardiac catheterization. The findings of those procedures were normal for all 16 patients. The ejection fractions ranged from 50% to 70%. The ECG changes can be seen in *Figure 2*.

Discussion

T-wave inversions on ECG may be indicative of ischemia. Alteration in ventricular activation or depolarization, as seen with intermittent LBBB, ventricular pacing, or preexcitation can also cause these T-wave changes. Cardiac memory describes the development and cessation of T-wave changes with intermittent or transient ventricular activation.[1,5] The study by Chatterjee et al[2] was one of the earliest to demonstrate that artificial pacing was the cause of T-wave inversions without QRS changes in a nonpaced beat. He also found that the duration of pacing influenced the duration of T-wave changes. In normal conduction, ventricular repolarization is in the opposite direction of ventricular depolarization, resulting in the polarity of the T wave and QRS being the same. The T-wave inversions that occur in cardiac memory with normal QRS duration are in the direction of the previous altered QRS complex, resulting in opposite polarity of the QRS and T wave.[7]

Although no clear definition exists, there are certain criteria, such as those by Rosenbaum et al,[3] who noted 3 elements: (1) T wave with vector during sinus rhythm approaching that of the paced or widened QRS; (2) accumulation of the size of the T wave with abnormal activation of the ventricle; and (3) persistence of T-wave changes for a variable period even after restoration of sinus rhythm.[1,8] Rosenbaum[3] referred to the T-wave changes as pseudoprimary. Initially, the T-wave changes were thought to be secondary because they were initiated by a change in the QRS complex. Yet the T-wave changes persisted after the QRS had normalized, mimicking a primary change.

The exact underlying mechanisms that lead to cardiac memory are unclear. Some consider it a reversible form of electrical remodeling.[9] There are data suggesting that modification of specific potassium channels,[4,10] modification of calcium channels,[11,12] angiotensin II production,[13,14] myocardial stretch,[15,16] transient outward current It0,[3,10] transcription factors,[17] and alterations in the phosphorylation of the cyclic adenosine monophosphate–responsive element binding protein are all involved in cardiac memory.[4,17-19]

It is unknown how long an intermittent LBBB or ventricular pacing needs to be present for cardiac memory to develop. In the canine heart, cardiac memory was induced after 20 minutes of right ventricular pacing.[10] In humans, T-wave changes developed within 1 week.[5] Studies on patients with intermittent LBBB demonstrate the incidence of T-wave inversions during sinus rhythm range from 72% to 83%.[20,21] The cardiac memory pattern on ECG can last for minutes to hours, or as long as weeks to months.
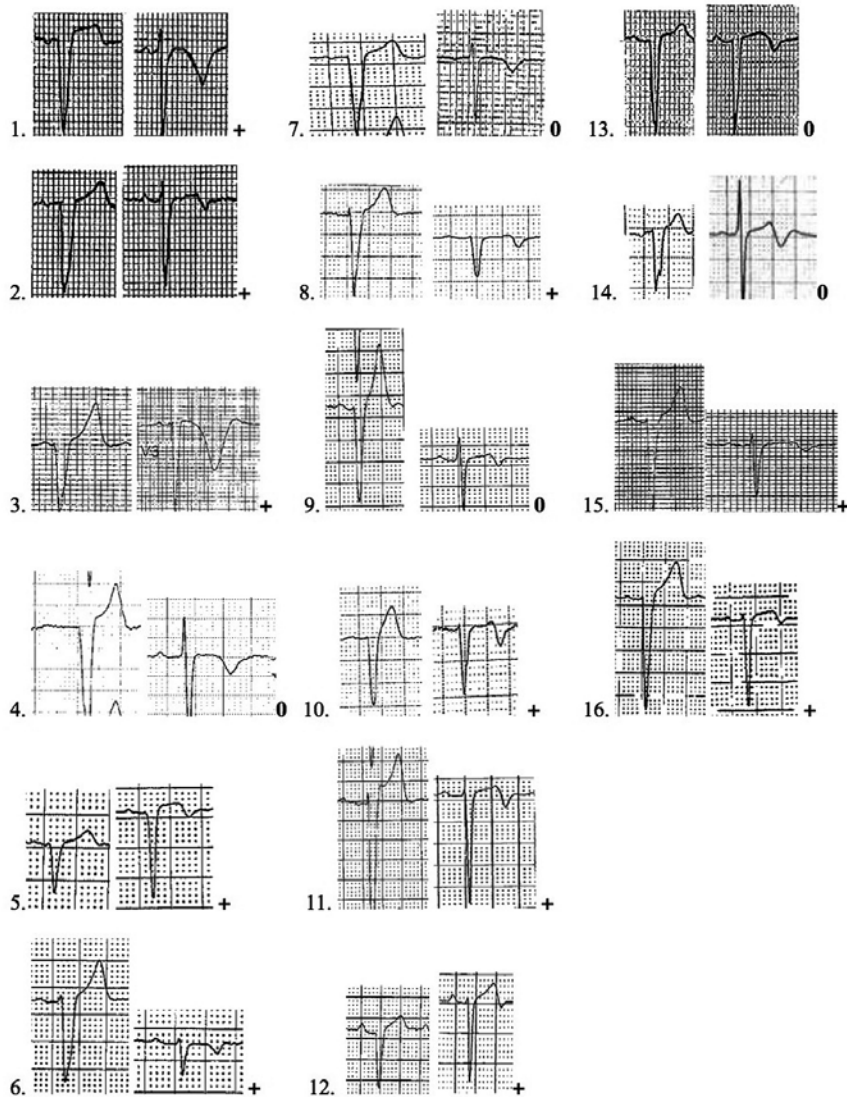
Figure 2.
Electrocardiograms from all 16 patients. The plus sign indicates normal perfusion per myocardial perfusion imaging, and 0 signifies normal coronary arteries per cardiac catheterization.

Although the exact mechanism is still unknown, the duration of cardiac memory is somewhat proportionally related to the length of abnormal ventricular activation.[1,9,18] The morphology of the QRS/T ratio with LBBB determines the magnitude of the T-wave vector change with cardiac memory.[18]

There are still unanswered questions regarding cardiac memory and the mechanisms involved. If intermittent LBBB is unknown or not observed, is it possible to differentiate ischemic T-wave changes from cardiac memory T-wave changes? Shvilkin et al[22] developed 3 criteria to differentiate cardiac memory from ischemia:
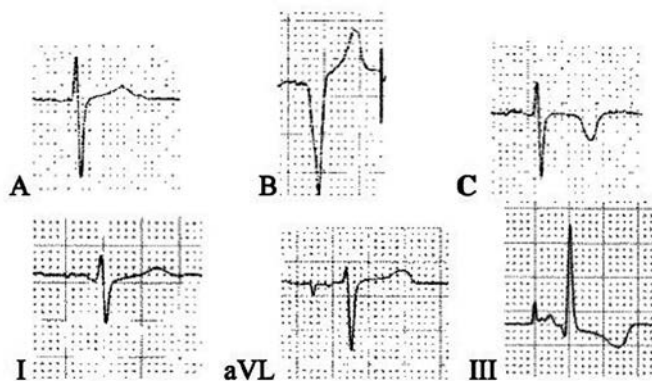
(1) positive T wave in aVL; (2) positive or isoelectric T wave in lead I; and (3) maximal precordial T-wave inversion is greater than the T-wave inversion in lead III. These criteria were 92% sensitive and 100% specific for detecting cardiac memory as the cause of T-wave changes[22] (*Figure 3* and *Figure 4*). Fifteen of the 16 patients in our medical record review met the above criteria for cardiac memory. Therefore, it appears the criteria developed by Shvilkin et al[22] can be accurately used to differentiate between ischemia and cardiac memory.

Conclusion

The ECG pattern of high-grade LAD stenosis has proven to be an important marker of high-risk patients with chest pain.[1] A primary concern for patients presenting with chest pain is myocardial ischemia. Physician awareness and recognition of cardiac memory and its benign nature, however, can help avoid unnecessary hospital admissions and procedures.

Author Contributions

Drs Kershaw and Rogers provided substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; drafted the article or revised it critically for important intellectual content; and gave final approval of the version of the article to be published.

Figure 3.
Patient with a permanent pacemaker who presented for a stress test. Resting electrocardiogram with normal QRS in lead V$_2$ (A) compared with the same lead with ventricular pacing (B), and a return to normal QRS as pacing resolves (C). If the criteria of Shvilkin et al[22] are applied, the positive T waves in leads I and aVL and the T-wave inversion in lead III support changes due to cardiac memory as opposed to myocardial ischemia.
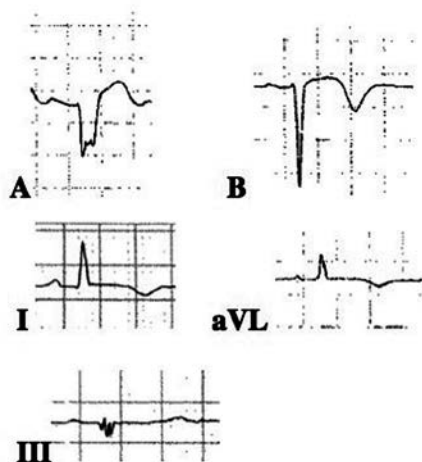

Figure 4.
This patient presented with acute coronary syndrome and intermittent left bundle branch block (LBBB). The troponin rose to 2.16 ng/mL, and cardiac catheterization demonstrated 90% proximal stenosis in the left anterior descending coronary artery. Panel A shows the LBBB pattern in lead V$_2$, and panel B shows the T-wave inversion in V$_2$ after resolution of LBBB. In contrast with a patient whose T-wave changes are due to cardiac memory, this patient shows T-wave inversion in leads I and aVL.

References

1. de Zwaan C, Bar FW, Wellens HJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. *Am Heart J*. 1982;103(4-2):730-736.

2. Chatterjee K, Harris A, Davis G, Leatham A. Electrocardiographic changes subsequent to artificial ventricular depolarization. *Br Heart J*. 1969;31:770-779.

3. Rosenbaum MB, Blanco HH, Elizari MV, Lazzari JO, Davidenko JM. Electronic modulation of the T wave and cardiac memory. *Am J Cardiol*. 1982;50(2):213-222.

4. Gautschi O, Naegeli B. Cardiac memory mimicking myocardial ischaemia. *J R Soc Med*. 2003;96(3):131-132.

5. Wecke L, Gadler F, Linde C, Lundahl G, Rosen MR, Bergfeldt L. Temporal characteristics of cardiac memory in humans: vectorcardiographic quantification in a model of cardiac pacing. *Heart Rhythm*. 2005;2(1):28-34.

6. Wagner GS. *Marriott's Practical Electrocardiography.* 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001:107-109.

7. Wecke L, Rubulis A, Lundahl G, Rosen MR, Bergfeldt L. Right ventricular paging-induced electrophysiological remodeling in the human heart and its relationship to cardiac memory. *Heart Rhythm.* 2007;4(12):1477-1486.

8. Kolb JC. Cardiac memory-persistent T wave changes after ventricular pacing. *J Emerg Med.* 2002;23(2):191-197.

9. Bierhuizen MF, Vos MA. The molecular labyrinth of electrical remodeling: making sense of cardiac memory. *Heart Rhythm.* 2010;7(7):971-972. doi:10.1016/j.hrthm.2010.04.003.

10. Geller JC, Rosen MR. Persistent T-wave changes after alteration of the ventricular activation sequence: new insights into cellular mechanisms of 'cardiac memory.' *Circulation.* 1993;88(4 pt 1):1811-1819.

11. Moss AJ. The heart does not have Alzheimer's disease: electrical and mechanical cardiac memory after ventricular pacing. *J Am Coll Cardiol.* 2004;44(9):1889-1890.

12. Plotnikov AN, Yu H, Geller JC, et al. Role of L-type calcium channels in pacing-induced short-term and long-term cardiac memory in canine heart. *Circulation.* 2003;107(22):2844-2849.

13. Ozgen N, Lau DH, Shlapakova IN, et al. Determinants of CREB degradation and KChIP2 gene transcription in cardiac memory. *Heart Rhythm.* 2010; 7(7):964-970. doi:10.1016/j.hrthm.2010.03.024.

14. Rosen MR. Why T waves change: a reminiscence and essay. *Heart Rhythm.* 2009;6(11 suppl):S56-S61. doi:10.1016/j.hrthm.2009.07.022.

15. Jeyaraj D, Wilson LD, Deschenes I. Stretch the heart: get memory. *Heart Rhythm.* 2008;5(1):114-115.

16. Sosunov EA, Anyukhovsky EP, Rosen MR. Altered ventricular stretch contributes to initiation of cardiac memory. *Heart Rhythm.* 2008;5(1):106-113.

17. Patberg KW, Plotnikow A, Giannulin R, et al. Cardiac memory is associated with alterations in the cAMP responsive element binding protein and its phosphorylation form. *PACE.* 2001;24:645.

18. Shvilkin A, Bojovic B, Vajdic B, Gussak I, Zimetbaum P, Josephson ME. Vectorcardiographic determinants of cardiac memory during normal ventricular activation and continuous ventricular pacing. *Heart Rhythm.* 2009;6(7):943-948.

19. Rosen MR, Cohen IS. Cardiac memory...new insights into molecular mechanisms. *J Physiol.* 2006;570(pt 2):209-218.

20. Engel TR, Shah R, DePodesta LA, Frankl WS, Krause RL. T wave abnormalities of intermittent left bundle branch block. *Ann Intern Med.* 1978;89(2):204-206.

21. Denes P, Pick A, Miller RH, Pietras RJ, Rosen KM. A characteristic precordial repolarization abnormality with intermittent left bundle-branch block. *Ann Intern Med.* 1978;89:(1)55-57.

22. Shvilkin A, Ho KK, Rosen MR, Josephson ME. T-vector direction differentiates postpacing from ischemic T-wave inversion in precordial leads. *Circulation.* 2005;111(8):969-974.

23. Yu H, Mckinnon D, Dixon JE, et al. Transient outward current, Ito1, is altered in cardiac memory. *Circulation.* 1999;99(14):1898-1905.

24. Wellens HJ, Conover MB. *The ECG in Emergency Decision Making.* 2nd ed. Philadelphia, PA: Saunders; 2005:54-59.

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