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CARDIAC MEMORY: A CASE REPORT AND REVIEW OF THE LITERATURE

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☐ **Abstract—Background: A variety of clinical syndromes can cause T-wave inversion (TWI), ranging from life-threatening events to benign conditions. One benign cause of TWI is cardiac memory, which is characterized by the transient inversion of T-waves following abnormal activation of the ventricles, commonly due to intermittent left bundle branch block (LBBB), tachydysrhythmias, electrical pacing, or ventricular pre-excitation. Case Report: A 72-year-old man presented to the emergency department with chest pain, nausea, vomiting, and headache. Upon arrival, his electrocardiogram (ECG) showed new-onset LBBB with appropriate secondary ST-T wave changes. A subsequent ECG showed disappearance of LBBB and newly inverted T-waves in precordial leads V1–V5, followed by a repeat ECG that again showed LBBB. Serial troponin testing was unremarkable. During hospitalization, echocardiogram and nuclear perfusion stress test were normal. The transient TWIs in this patient were believed to be due to cardiac memory. We performed a literature review and identified 39 published cases of cardiac memory. The most common etiology for cardiac memory was after cardiac pacemaker placement, followed by intermittent LBBB (as was seen in our patient), and post-tachydysrhythmia. Patient ages ranged from 21 to 88 years, with an equal number of cases reported in men and women. Why Should an Emergency Physician Be Aware of This?: Cardiac memory is a poorly understood, rarely observed phenomenon that can occur in the setting of intermittent LBBB. Testing for acute cardiac ischemia and underlying coronary artery disease is still recommended, as the diagnosis of cardiac memory can only be made after negative workup.** © **2019 Elsevier Inc. All rights reserved.**

☐ **Keywords—cardiac memory; electrocardiogram; T-wave inversion; intermittent left bundle branch block**

INTRODUCTION

Although there are multiple causes of electrocardiographic precordial T-wave inversions (TWIs), this finding should initially prompt a consideration for the diagnosis of acute cardiac ischemia or underlying significant coronary artery disease (e.g., Wellens' electrocardiogram [ECG] pattern). A lesser understood, rare, alternative cause of precordial TWIs is cardiac memory, which is characterized by a transient inversion to T-waves following abnormal activation of the ventricles (1). This can be seen in a variety of settings, such as following tachydysrhythmias, intermittent left bundle branch block (LBBB), electrical pacing, or ventricular pre-excitation, such as in Wolff-Parkinson-White syndrome (2–5). Cardiac memory is believed to be a benign finding, as it does not indicate acute cardiac ischemia, and is not related to underlying coronary artery disease. However, it is an important phenomenon for the emergency physician to be aware of, as it can provide an alternative explanation for concerning ECG findings in the correct clinical context. Here we present a case of intermittent LBBB followed by a narrow QRS rhythm with newly inverted T-waves in precordial leads V1–V5 in a 72-year-old male due to cardiac memory.

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Additionally, we seek to review the contemporary published medical literature regarding this unusual condition.

CASE REPORT

A 72-year-old man with a history of hypertension presented to the emergency department (ED) with sudden-onset dizziness, substernal chest pain radiating to the left neck, headache, nausea, and vomiting. The patient reported having been "pistol whipped" in the head the evening prior, for which he was evaluated in a different ED with a negative computed tomography (CT) scan of the head. On arrival, blood pressure was 196/97 mm Hg, otherwise his vital signs were normal. Physical examination was remarkable for left temporal abrasion; tenderness over the temple and left lateral neck; and unremarkable neurologic, cardiac, and pulmonary examinations.

Initial laboratory results, including troponin, complete blood count, prothrombin time/international normalized ratio, and comprehensive metabolic panel were unremarkable, except for a potassium of 3.3 mEq/L, which was repleted. First ECG (Figure 1), recorded upon arrival to the ED, showed a new-onset LBBB with appropriate secondary ST-T wave changes compared with previous ECG from 2012. Chest x-ray study was notable for mild cardiomegaly. Additional imaging, including CT head, CT angiography of the neck, CT of the cervical spine and magnetic resonance imaging of the brain, was negative for any acute intracranial and cervical abnormalities. A repeat ECG (Figure 2) 4 h after arrival showed disappearance of the LBBB and new deep TWIs in the anterior precordial leads. A third ECG (Figure 3), obtained 6 h after arrival, showed reappearance of the LBBB. Figure 4 shows a baseline ECG of sinus bradycardia from 2012. Given the dynamic changes on ECG and concern for acute cardiac ischemia or underlying significant coronary artery disease, the patient was admitted for further cardiac evaluation.

The patient was given 325 mg aspirin and sublingual nitroglycerin for ongoing chest pain. Cardiology recommended blood pressure control with the patient's home antihypertensives, amlodipine 5 mg, enalapril 10 mg, and isosorbide dinitrate 30 mg. Blood pressure improved to 136/87 mm Hg and chest pain resolved. Echocardiogram demonstrated normal systolic function with an ejection fraction of 50–55% and grade I diastolic dysfunction. A regadenoson (Lexiscan®; Astellas Pharma, Tokyo, Japan) nuclear stress test was performed and negative for any stress-induced perfusion defects. Given reassuring studies and resolution of chest pain, cardiac catheterization was not performed and the patient was discharged on hospital day 4 with complete resolution of symptoms.

DISCUSSION

Although this patient's presentation was concerning for cardiac ischemia, his workup ultimately did not identify any underlying significant coronary artery disease or acute myocardial infarction. The precordial T-wave changes seen on his ECG were attributed to cardiac memory following his intermittent LBBB. In a study looking at patients with intermittent LBBB, the incidence of TWIs seen during normal conduction was 72% and

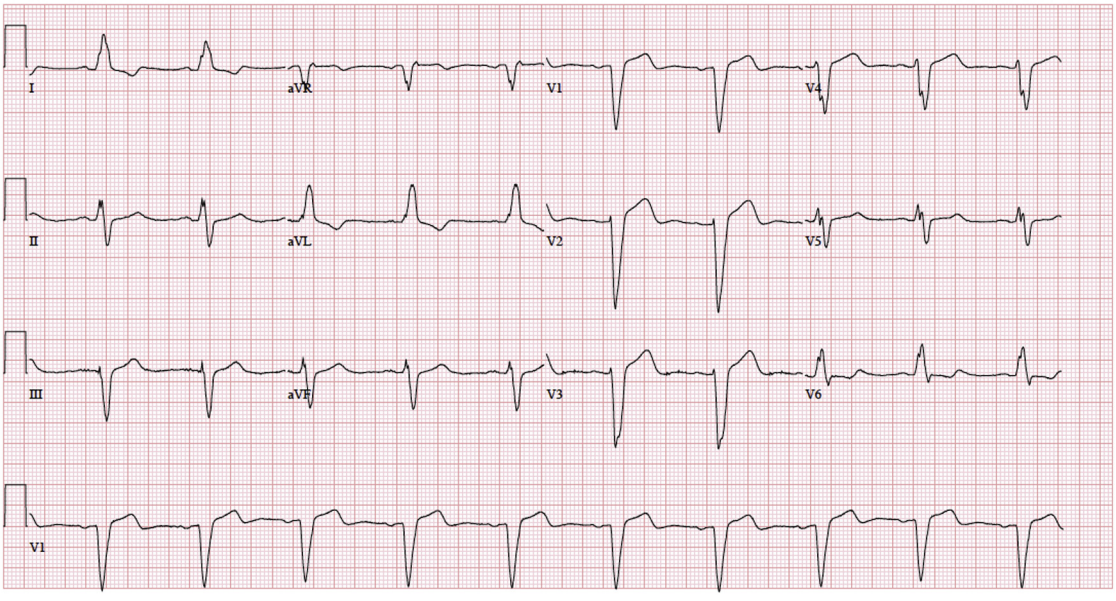

Figure 1. Initial electrocardiogram upon arrival to the emergency department shows left bundle branch block with T-wave inversion in I and aVL.

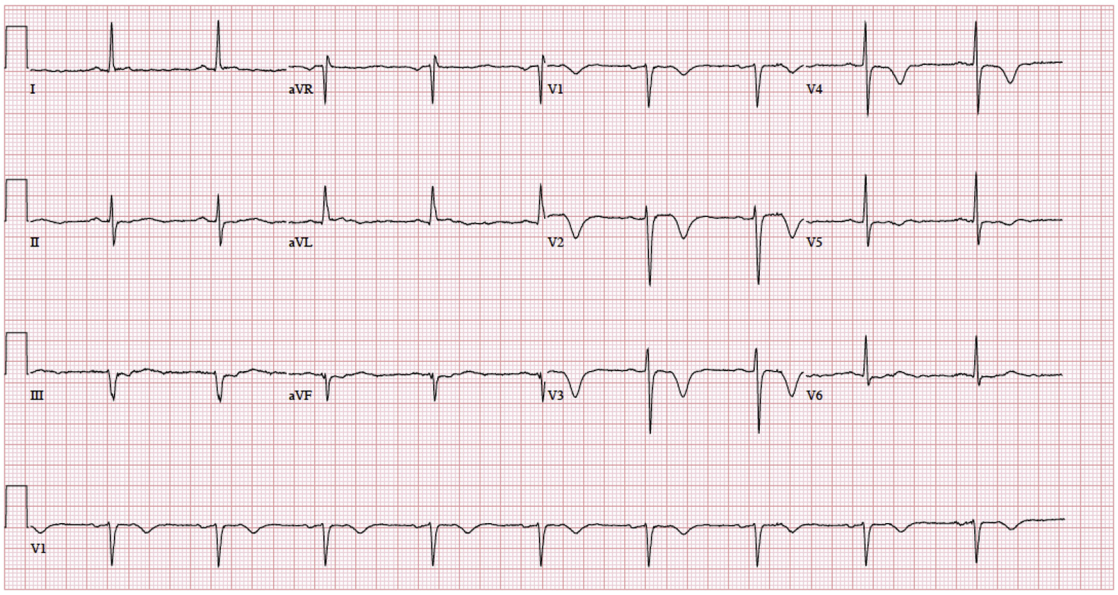

Figure 2. Repeat electrocardiogram 4 h after arrival shows normal sinus rhythm with T-wave inversion in V1–V5.

approximately half of those patients had no clinical evidence of coronary artery disease (6). This would suggest that cardiac memory is not an uncommon finding in intermittent LBBB.

Cardiac memory (also termed *Chatterjee phenomenon*, *post tachycardia T-wave inversion*, or *post-pacing T-wave inversion*) was first reported in the 1930s (7). Chatterjee et al. showed that transient TWIs could be induced in human subjects solely by electrical pacing, and found that the duration of TWIs was proportional to the time of electrical pacing. Transient TWIs lasted for 15 min when paced for 10 min, but lasted as long as 18 months when paced for 2 years (8). Rosenbaum coined the term *cardiac memory* in 1982 and demonstrated that abnormal activation of the ventricles, such as during electrical pacing or intermittent LBBB, was responsible for the transient TWIs (1). He used the term *memory* to describe how the T-wave remembers the leads with the greatest magnitude of QRS during the abnormal activation. The TWIs seen during sinus rhythm occurred in

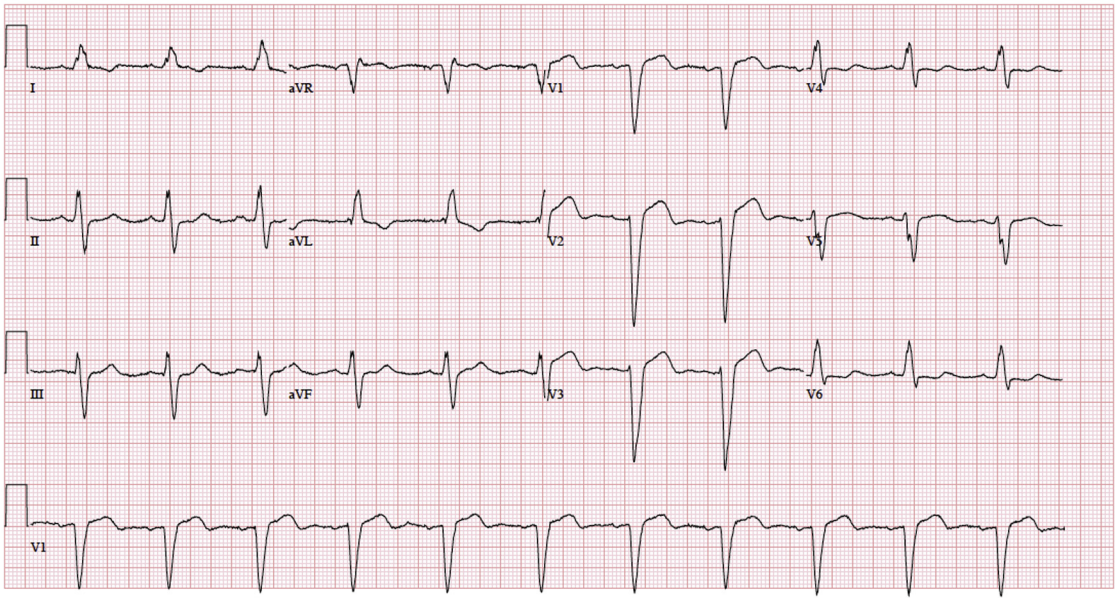

Figure 3. Electrocardiogram 6 h after arrival shows return of left bundle branch block morphology and T-wave inversion in I and aVL.

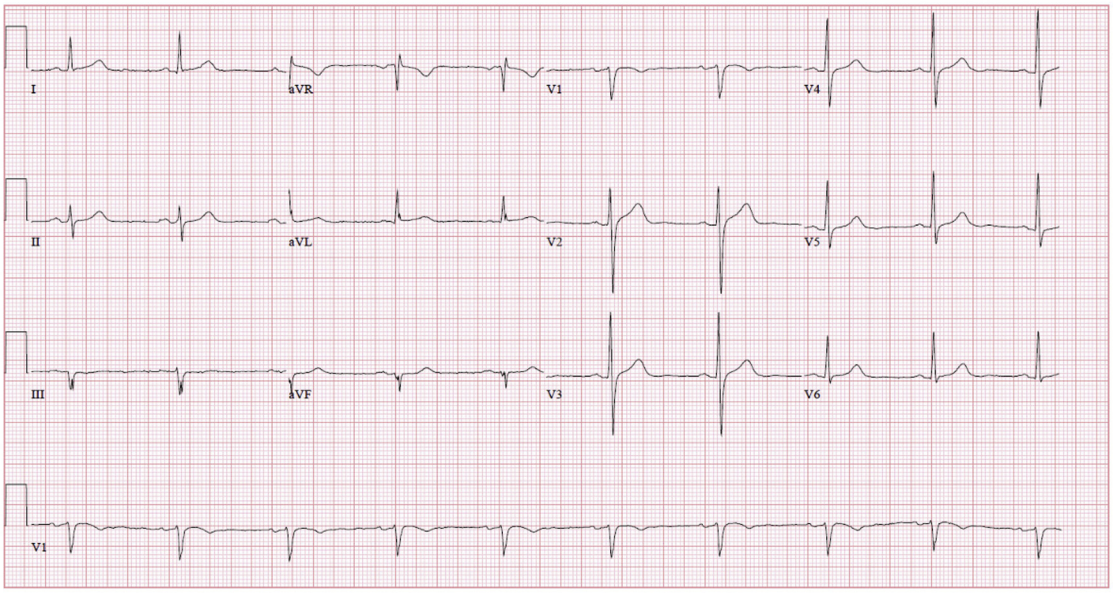

Figure 4. Baseline electrocardiogram from 2012 shows sinus bradycardia with T-wave inversion in V1.

the same leads as the deeply negative QRS complexes during electrical pacing or the largely positive QRS complexes in intermittent LBBB. Rosenbaum also described the property of "accumulation" in cardiac memory cells, where the magnitude of the T-wave change increased with repetitive activation by pacing or LBBB and persisted for longer periods of time with increasing duration of the altered activation (1).

There are several different mechanisms thought to be involved in the development of cardiac memory. Current research suggests that altered activation causes mechanical stretch and strain in cardiac myocytes, leading to increased angiotensin II (AT II) synthesis and release (9). One of the roles of AT II in the heart is to regulate the transient outward potassium current (I_{to}) and cell-surface expression of potassium channels (10). AT II causes internalization of the AT-1 receptor and a subunit of the potassium channel on the cell surface, leading to their degradation. The overall effect is a reduction in the density of functioning potassium channels and a reduction in current (11). I_{to} is normally responsible for the fast outward flow of potassium from myocytes immediately following depolarization, and also contributes to the repolarization gradients in the ventricles; therefore, disruptions in this process will understandably lead to changes in T-wave morphology (4). Several studies support this theory, as pharmacological blockade of potassium channels, AT-1 receptors, and angiotensin-converting enzymes affect cardiac memory or prevent it altogether (10,12,13). Additionally, calcium currents, other types of potassium currents, and connexins, which are the main proteins making up gap junctions, also appear to be involved in development of cardiac memory (14–16).

The TWIs seen with cardiac memory can be difficult to distinguish from the TWIs often seen with Wellens' ECG pattern. Shvilkin et al. found that the combination of 1) positive T-wave in aVL, 2) positive or isoelectric T-wave in lead I, and 3) largest TWI in the precordial leads that is greater than the TWI seen in lead III, was 92% sensitive and 100% specific for cardiac memory (17). Another set of criteria used a combination of 1) positive T-wave in aVL, 2) negative or isoelectric T-wave in II, 3) negative T-wave in V4-V6, or 4) QTc <430 ms, and found a sensitivity of 100% and specificity of 96% for cardiac memory (18). The results from these small case series require prospective validation before clinical decision-making should be altered.

Cardiac memory is generally thought to be a benign finding, however, recently this idea has begun to be challenged. Cases of torsades de pointes (TdP) have been reported in patients with abnormal ventricular activation and cardiac memory changes. Haverkamp et al. describe a patient in whom TWIs from cardiac memory were seen following successful ablation of atrioventricular re-entrant tachycardia. This patient was then given sotalol, a potassium channel blocker, and subsequently developed prolonged QT and TdP (19). Although sotalol is a risk factor for developing prolonged QT syndrome and TdP, the possibility of an interaction while the patient was experiencing cardiac memory (i.e., a reduction in the potassium channel receptors, see above) may have contributed to the development of

Table 1. Summary of Findings From Literature Review on Cardiac Memory

Year	First Author	Clinical History	Cardiac Memory TWI on ECG	CAD Ruled Out*	Cardiac Memory Etiology
1952	Myerson (21)	32 yo M with palpitations and narrow complex tachycardia on presenting ECG	V2–V5	None	Post-tachydysrhythmia
1952	Myerson (21)	26 yo M with SOB, palpitations, weakness, and narrow complex tachycardia on presenting ECG	II, III, aVF, V5–V6†	None	Post-tachydysrhythmia
1969	Kernohan (22)	21 yo M with tachyarrhythmia and new TWIs on ECG following cardioversion	TWI in II, III, aVF, V3–V6	Normal cardiac biomarkers; Non-obstructive CAD	Post-tachydysrhythmia
1980	Gould (23)	70 yo F with dizziness and LBBB on presenting ECG	III, V2–V3	Normal cardiac biomarkers; Non-obstructive CAD	Intermittent LBBB
1985	Michelotti (24)	54 yo M who was denied medical insurance after ECG during routine medical examination showed new TWIs	III, V2–V4	Non-diagnostic echo; Non-obstructive CAD	Intermittent LBBB
1995	Katz (25)	67 yo M on digoxin p/w SVT treated with carotid massage	V4#	Non-obstructive CAD	Post-tachydysrhythmia
1998	Haverkamp (19)	66 yo M with SVT requiring ablation and temporary RV pacing	V2–V3	Normal cardiac biomarkers	Ventricular pacing
2002	Iida (5)	38 yo M presenting for elective knee arthroscopy found to have new TWIs on preoperative ECG	II, III, aVF	Normal cardiac biomarkers; Non-diagnostic echo	Ventricular pre-excitation
2002	Kolb (4)	28 yo M with pacemaker for sick sinus syndrome and aflutter p/w chest pain, nausea, vomiting	II, III, aVF, V2–V6	Normal cardiac biomarkers	Ventricular pacing
2002	Kolb (4)	77 yo F with pacemaker for CHB who had a routine ECG 1 wk after hip surgery showing new TWIs	II, III, aVF, V1–V6	Normal cardiac biomarkers; Non-diagnostic echo	Ventricular pacing
2003	Gautschi (26)	85 yo F with dizziness, DOE, and syncope from sick sinus syndrome who had new TWIs on ECG following ventricular pacemaker placement	II, V2–V4	Normal cardiac biomarkers	Ventricular pacing
2006	Shenoy‡ (27)	48 yo M with intermittent chest discomfort and new TWIs on presenting ECG	V1–V4	Non-obstructive CAD	Intermittent LBBB
2007	Monahan (28)	60 yo M p/w ICD discharge for multiple episodes of NSVT after a bee sting	I, II, aVL, V5–V6, biphasic t-waves in V2–V4	Normal cardiac biomarkers, non-obstructive CAD	Post-tachydysrhythmia
2007	Siddiqui (29)	78 yo F p/w acute pulmonary edema and initial ECG showing LBBB	V1–V5	Non-obstructive CAD	Intermittent LBBB
2007	Sovari (30)	61 yo F with afib p/w palpitations, nausea, dizziness, chest pain, and aflutter with variable AV block on presenting ECG	I, II, III, aVF, V3–V5	Normal cardiac biomarkers; non-diagnostic echo; non-obstructive CAD	Post-tachydysrhythmia
2007	Wylie (31)	74 yo F on propafenone for afib p/w lightheadedness, malaise, nausea, anorexia and wide complex tachycardia on presenting ECG	V1–V5, II, III, aVF	Normal cardiac biomarkers; non-diagnostic echo; non-obstructive CAD	Propafenone toxicity
2008	Erdogan (32)	58 yo F with RVOT pacemaker for second-degree AV block presenting for routine pacemaker interrogation	I, aVL, V2–V4	None	Ventricular pacing
2008	Rottensteiner (33)	26 yo M with jaw pain and sensation of lump in throat following initiation of high-dose methylprednisolone for orbital pseudotumor	II, III, aVF, V4–V6	Normal cardiac biomarkers, non-diagnostic echo, non-obstructive CAD	Methylprednisolone
2008	Sucu (2)	36 yo F 20 wk pregnant with monomorphic VT and LBBB on presenting ECG	III, aVF, biphasic t-waves in II, V3–V5	Non-diagnostic echo	Post-tachydysrhythmia

(*Continued*)

Table 1. Continued

Year	First Author	Clinical History	Cardiac Memory TWI on ECG	CAD Ruled Out*	Cardiac Memory Etiology
2009	Chen (34)	74 yo M presenting for routine investigation of pacemaker for afib and sick sinus syndrome found to have new TWIs on ECG	II, III, aVF, V3–V6	Non-obstructive CAD	Ventricular pacing
2010	Brown (35)	39 yo F with Ebstein anomaly p/w palpitations, chest discomfort and narrow complex tachycardia on presenting ECG	I, II, III, aVF, V2–V6	Non-diagnostic echo; non-obstructive CAD	Post-tachydysrhythmia
2010	Byrne (3)	57 yo M p/w chest pain, and LBBB on presenting ECG	V1-V4	Non-obstructive CAD	Intermittent LBBB
2011	Van de Heyning (36)	63 yo M p/w atypical chest pain and bronchitis and initial ECG showing LBBB alternating with NSR	V2-V5	Normal cardiac biomarkers; non-obstructive CAD	Intermittent LBBB
2012	Costantini (37)	23 yo M p/w intermittent precordial pain and new TWIs on presenting ECG	III, aVF, V2–V3	Normal cardiac biomarkers; non-diagnostic echo	Intermittent LBBB
2013	di Cori (38)	34 yo F with congenital long-QT syndrome presenting for ICD replacement	V2–V6§	None	Ventricular pacing
2014	Littmann (39)	83 yo F with pacemaker for intermittent CHB p/w fatigue and leg swelling and initial ECG showing LBBB with new TWIs	II, III, aVF, V3–V6	Non-diagnostic echo; non-obstructive CAD	Ventricular pacing
2014	Vijayaraman (40)	88 yo F with His bundle pacemaker placement for afib, sinus bradycardia, and LBBB	II, III, aVF, V1–V5	None	Ventricular pacing
2016	Waks (41)	72 yo F with pacemaker placement for afib, aflutter, and tachy-brady syndrome	II, III, aVF, and V3–V6	Non-diagnostic echo	Ventricular pacing
2016	Yoshida (20)	71 yo F with long QT-syndrome and LBBB p/w faintness and Holter monitor showing LBBB transitioning into sinus rhythm with TWIs and TdP.	V5‖	None	Intermittent LBBB
2017	Danon (42)	70 yo F p/w DOE and RBBB and LAFB on presenting ECG	II, III, aVF, V4-V5	Non-obstructive CAD	Alternating trifascicular block
2017	di Matteo (43)	68 yo F with pacemaker for paroxysmal CHB p/w epigastric pain	II, III, aVF and V3–V6	Normal cardiac biomarkers; non-obstructive CAD	Ventricular pacing
2017	di Matteo (43)	85 yo M with pacemaker for sick sinus syndrome p/w intermittent stinging chest pain	II, III, aVF, V3–V4	Normal cardiac biomarkers; non-obstructive CAD	Ventricular pacing
2017	Hoyme¶ (44)	82 yo F with pacemaker for intermittent afib p/w chest pain, nausea, vomiting, and initial afib with new TWIs on presenting ECG	II, III, aVF, V3–V6	Normal cardiac biomarkers; non-obstructive CAD	Ventricular pacing
2017	Manne (45)	81 yo F p/w left arm and shoulder pain and new TWIs on presenting ECG	V1–V3	Normal cardiac biomarkers; non-diagnostic echo; non-obstructive CAD	Intermittent LBBB
2018	Bhandari (46)	69 yo F p/w syncope and CHB with new pacemaker placement	V2–V4	Non-diagnostic echo; non-obstructive CAD	Ventricular pacing
2018	Bode** (47)	59 yo M with cardiomyopathy p/w syncope after being shocked by ICD for VT	II, III, aVF	Non-diagnostic echo; non-obstructive CAD	Ventricular pacing

(*Continued*)

Table 1. Continued

Year	First Author	Clinical History	Cardiac Memory TWI on ECG	CAD Ruled Out*	Cardiac Memory Etiology
2018	Ibarrola (48)	46 yo M p/w stable angina for 1 y and LBBB on presenting ECG	III, V2–V5, biphasic t-wave in V6	Non-obstructive CAD	Intermittent LBBB
2018	Peck (49)	60 yo M p/w pleuritic chest pain and LBBB on presenting ECG	II, III, aVF, V2–V5	Normal cardiac biomarkers; Non-obstructive CAD	Intermittent LBBB
2018	Seibolt (50)	50 yo F presenting for knee arthroscopy with LBBB on presenting ECG	V4–V6	Non-obstructive CAD	Intermittent LBBB

afib = atrial fibrillation; AV = atrioventricular; CAD = coronary artery disease; CCTA = coronary computed tomography angiography; CHB = complete heart block; DOE = dyspnea on exertion; ECG = electrocardiogram; echo = echocardiography; EF = ejection fraction; F = female; ICD = implantable cardioverter defibrillator; IVCD = intraventricular conduction delay; LAFB = left anterior fascicular block; LBBB = left bundle branch block; LPFB = left posterior fascicular block; M = male; NSVT = non-sustained ventricular tachycardia; NSR = normal sinus rhythm; p/w = presenting with; RBBB = right bundle branch block; RV = right ventricle; RVOT = right ventricular outflow tract; SOB = shortness of breath; STD = ST depression; STE = ST elevation; SVT = supraventricular tachycardia; TdP = Torsades de pointes; TWI = T-wave inversion; VT = ventricular tachycardia; yo = year old.
* Non-obstructive CAD was diagnosed through cardiac catheterization, nuclear stress test, pharmacologic stress test, treadmill stress test, stress echocardiography, cardiac magnetic resonance imaging, or coronary computed tomography angiography.
† No ECG provided.
‡ Poster presentation.
§ Only rhythm strip with V1–V6 provided.
|| Only rhythm strip with V5 provided.
¶ Original text in German.
# Only rhythm strip with V4 provided.
** Poster presentation.

this rhythm. A second case of TdP was related to repolarization abnormalities from cardiac memory in a patient with intermittent LBBB (20). The possible effects of cardiac memory on dysrhythmogenesis have not yet been fully studied; however, these cases suggest that cardiac memory may not be as harmless as previously thought.

We performed a literature review on PubMed using the phrase "cardiac memory." From 159 articles, we identified 30 case reports. Three of these articles contained multiple case reports, and an additional 6 case reports were included from other sources, for a total of 39 cases that we included in this review, as seen in Table 1. The earliest article came from 1952; however, the majority of articles were published in the last 10 years. There were 14 cases of cardiac memory involving electrical pacing, 12 from intermittent LBBB, 8 following tachydysrhythmias, and 5 with other causes (ventricular excitation, trifascicular block, catheter ablation, propafenone, and methylprednisolone). Patient ages ranged from 21 to 88 years old, with a relatively equal number of cases involving males and females. The TWI related to cardiac memory is most commonly seen in the anterior precordial leads, but can be seen in the inferior leads as well. The majority of cases involved patients presenting to the ED, however, other settings included cardiology office visits, routine pacemaker interrogation, and preoperative appointments.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Emergency physicians are trained to quickly scan an ECG for ST segment changes and TWIs in order to identify patients with myocardial ischemia/infarction or underlying significant coronary artery disease. Every effort is made to limit delay in getting these patients the definitive diagnosis and treatment they need, which is typically percutaneous coronary intervention. The 90-min "door-to-balloon" time is considered the standard of care for those hospitals treating patients with acute myocardial infarctions, which means that emergency physicians have only a few minutes to decide whether or not a patient is experiencing myocardial ischemia. Although precordial TWIs are most concerning for acute cardiac ischemia or underlying significant coronary artery disease, the emergency physician must be aware of other etiologies that can result in TWIs in leads V1–V3, such as benign TWI, digitalis effect, hypokalemia, pulmonary embolism, central nervous system event/increased intracranial pressure, apical variant hypertrophic cardiomyopathy, dysrhythmogenic right ventricular cardiomyopathy, Wolff-Parkinson-White syndrome,

right ventricular hypertrophy with strain, right bundle branch block, or cardiac memory. Additionally, in the patient presenting with palpitations, presyncope or syncope, and TWIs on ECG consistent with cardiac memory, a diagnosis of resolved tachydysrhythmia should be considered. Ultimately, the diagnosis of cardiac memory should only be considered after reliably excluding acute cardiac ischemia or significant underlying coronary artery disease.

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