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Case Report

Serial changes of L wave according to heart rates in a heart failure patient with persistent atrial fibrillation

Daisuke Morisawa (MD PhD), Yuko Ohno (MD), Yoshihiro Ohta (MD), Yoshiyuki Orihara (MD), Kumiko Masai (MD PhD), Akiko Goda (MD PhD FJCC), Masanori Asakura (MD PhD FJCC)*, Masaharu Ishihara (MD PhD FJCC)

Division of Cardiovascular Medicine, Hyogo College of Medicine, Hyogo, Japan

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ABSTRACT

Mid-diastolic forward flow velocity of transmitral flow (L wave) is known as a marker of diastolic dysfunction and is occasionally observed in patients with fluid retention, low heart rate, and atrial fibrillation (AF). However, how hemodynamic condition affects L wave is still unknown. An 81-year-old woman who underwent implantation of a DDD pacemaker due to complete atrioventricular block 38 years previously suffered from congestive heart failure and was admitted to our hospital. At the time of admission, electrocardiogram showed new-onset AF resulting in mode switch to VVI, and echocardiography showed a giant L wave. At the mid-term of the treatment, AF was converted to sinus rhythm resulting in mode switch to DDD, and pacemaker check-up was performed at pre- and post-cardioversion. During the pacemaker check-ups, L wave was assessed in various pacing rates. As pacing rate was increased, L wave altered according to heart rates and disappeared at 85 bpm in VVI with AF, whereas at 75 bpm in DDD. Through the treatment, L wave got smaller as fluid retention was improved and finally disappeared at the time of discharge. This case suggests that L wave is highly variable and affected by fluid volume, heart rate, and heart rhythm.

<**Learning objective:** Although L wave is known as a marker of diastolic dysfunction and occasionally observed in patients with high left ventricular filling pressure, low heart rate, and atrial fibrillation, how hemodynamics affects L wave is still unknown. In this case, it was observed that L wave is highly variable in the therapeutic process of heart failure and affected by fluid volume, heart rate, and heart rhythm. L wave potentially can be a useful indicator to evaluate treatment efficacy for heart failure.>

Introduction

L wave is usually defined as mid-diastolic forward flow velocity of transmitral flow whose velocity is more than 20 cm/sec and has been reported as a marker of diastolic dysfunction and/or elevation of left ventricular (LV) filling pressure, especially in patients with LV hypertrophy [1,2]. Some previous clinical studies have reported that L wave is more likely to occur in lower heart rate (HR) and atrial fibrillation (AF) [2–4]. However, L wave is not necessarily observed in patients with those hemodynamic conditions. No previous report has clarified how L wave was affected by heart rhythm and serial HR change and how L wave varies through treatment of heart failure. In this case report, we successfully detected serial changes in L wave according to various pacing rates of a pacemaker in a patient with as well as without AF. In addition, we observed how L wave altered through the treatment of heart failure.

Case report

An 81-year-old woman who underwent implantation of a DDD pacemaker due to complete atrioventricular block 38 years previously presented with dyspnea on effort and leg edema for one month, and was admitted to our hospital based on the diagnosis of decompensated congestive heart failure. Her body weight was 57.3 kg on admission (Fig. 1), which was a 7 kg gain compared to that of three months previously. Electrocardiogram showed new-onset AF and all ventricular pacing (the pacing rate was 60 bpm). Pacemaker check-up at the time of admission

* Corresponding author at: Division of Cardiovascular Medicine, Hyogo College of Medicine, 1-1 Mukogawa-cho, Nishinomiya, Hyogo, Japan.
E-mail address: ma-asakura@hyo-med.ac.jp (M. Asakura).

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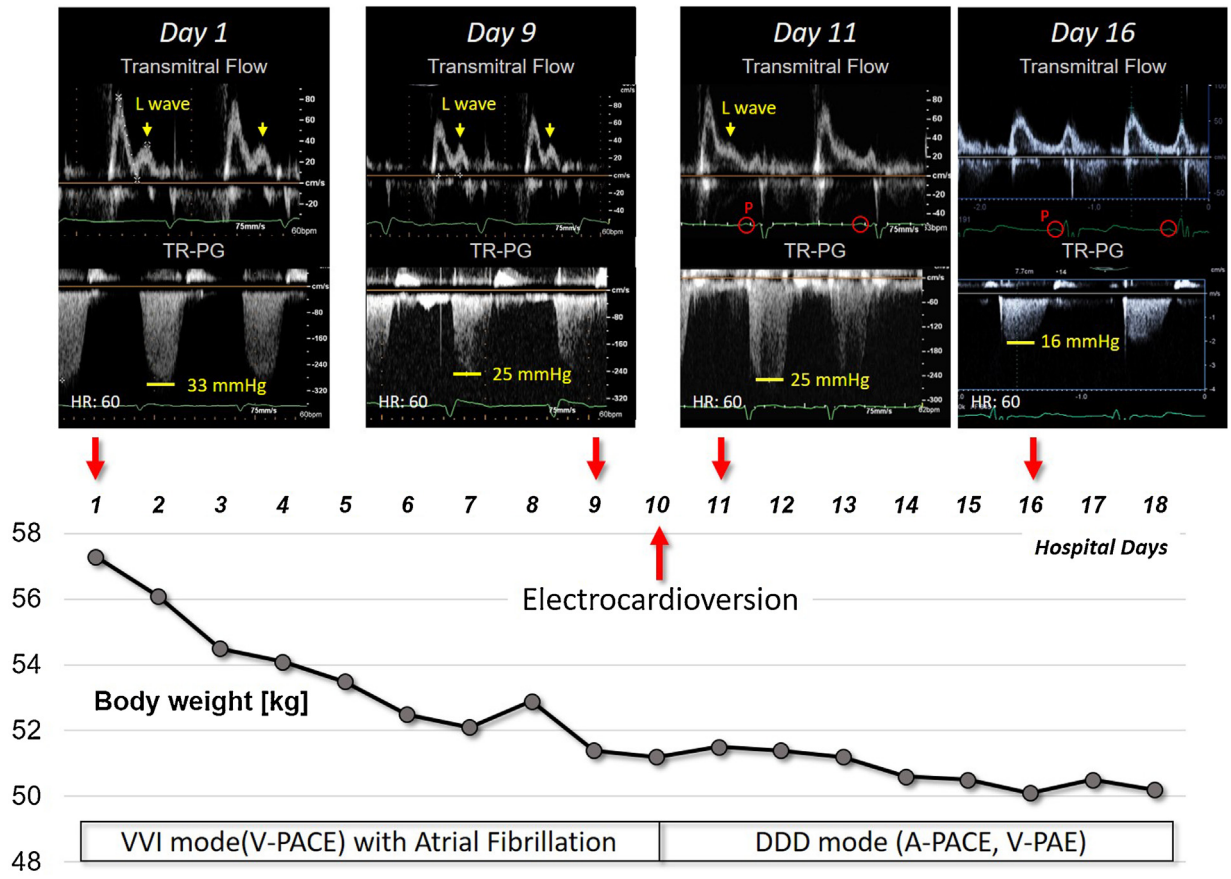

Fig. 1. Time course of body weight and echocardiography. Echocardiography was performed at four different points, the day 1, 9, 11, and 16. Transmitral flow and tricuspid regurgitation peak pressure gradient (TR-PG) are described in the upper panel. Body weight is described in the lower panel.

showed the pacing mode had been switched from DDD to VVI mode. AF had not been confirmed during pacemaker check-up performed three months previously. Echocardiography showed an ejection fraction of 55%, LV end diastolic diameter of 48 mm, left atrial volume index of 56 ml/m^2, tricuspid regurgitation peak pressure gradient (TR-PG) of 33 mmHg (Fig. 1), and diameter of inferior vena cava of 23 mm without respiratory change, which indicated heart failure due to volume overload. Severe valve dysfunction was not detected in the examination. In addition, pulse-wave Doppler showed a giant L wave of 37 cm/sec peak velocity in transmitral flow which looks like late mitral flow (A wave) (Fig. 1). We administered a loop diuretic for one week, which led to the body weight loss of 5.9 kg. On the 9th day after the admission, echocardiography was performed, and TR-PG was calculated as 25 mmHg which suggested that volume overload still remained (Fig. 1). We also assessed the form of L wave in seven settings of VVI pacing rate, 60, 65, 70, 75, 80, 85, and 90 bpm. At the baseline pacing rate of 60 bpm, an obvious L wave was still observed (Fig. 2A). As the pacing rate was increased, the height of L wave was getting lower and finally its velocity became less than 20 cm/sec which means out of L-wave definition at 85 bpm (Fig. 2B-G). The next day cardioversion was performed, and AF was successfully converted to sinus rhythm. Pacemaker mode was switched from VVI to DDD. On the day after the cardioversion (day 11), we assessed the L wave under DDD mode during pacemaker check-up in the same manner as pre-cardioversion. Triphasic transmitral flow i.e. early mitral flow (E wave), L wave, and A wave, was confirmed at the baseline pacing rate of 60 bpm, and L wave partially merged with E wave (Fig. 2H). As the pacing rate was increased, the fusion of E and L wave progressed, and A wave was getting closer to L wave (Fig. 2J). Subsequently the pacing rate was increased to 75 bpm, L wave was finally absorbed into E and A wave. At that time, transmitral flow altered triphasic to biphasic shape and L wave became undetectable (Fig. 2K). As the pacing rate was increased up to 90 bpm, the fusion of E and A wave progressed (Fig. 2K-N). The echocardiography performed on day 11 showed 25 mmHg of TR-PG which was the same as before cardioversion (Fig. 1). We continued the administration of the diuretics, and the body weight eventually decreased to 50.1 kg (Fig. 1). At the time of discharge, echocardiography was performed, and we confirmed that the L wave completely disappeared at the pacing rate of 60 bpm and TR-PG of 16 mmHg indicating a sufficient improvement in fluid retention (Fig. 1).

Discussion

L wave is occasionally detected in patients with heart failure and regarded as an indicator of diastolic dysfunction [2,5]. Some previous researchers have reported that regardless of ejection fraction, L wave is more likely to be observed in heart failure patients with lower HR, higher LV filling pressure and AF, and the presence of L wave is associated with poor prognosis [3,4,6]. However, in these reports, L wave was observed not for a certain duration but at a specific point of time, e.g. at time of discharge or outpatient clinic visit. Therefore, to date, no clinical research has elucidated how L wave serially varies according to HR change or how L wave is affected by heart rhythm and fluid volume. We report for the first time in this paper that there were serial changes

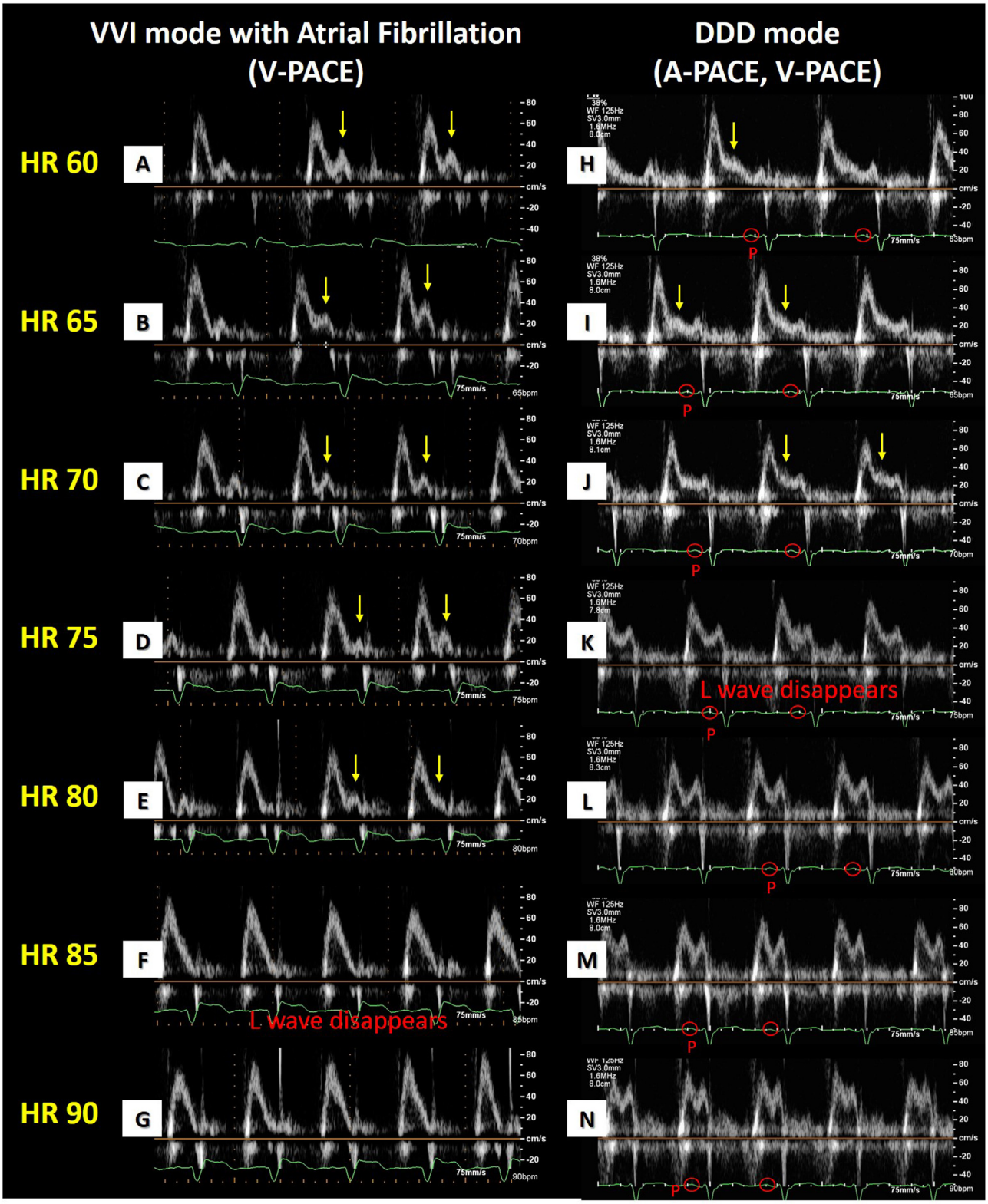

Fig. 2. Serial changes in transmitral flow in six settings of pacing rate, 60–90 bpm. (A–G) VVI mode with atrial fibrillation. (H–N) DDD mode. Yellow arrows indicate L wave.

of L wave according to HR change with or without the presence of A wave. In addition, L wave alteration was observed through the improvement of heart failure.

In this case, as the pacing rate was increased, L wave was gradually getting smaller and finally became undetectable. This suggests that HR elevation led to L wave disappearance even in the same degree of volume overload. Furthermore, L wave disappeared at pacing rate of 85 bpm under VVI mode with AF rhythm, whereas at the pacing rate of 75 bpm under DDD mode with the presence of A wave. There were 10 bpm difference between the presence and

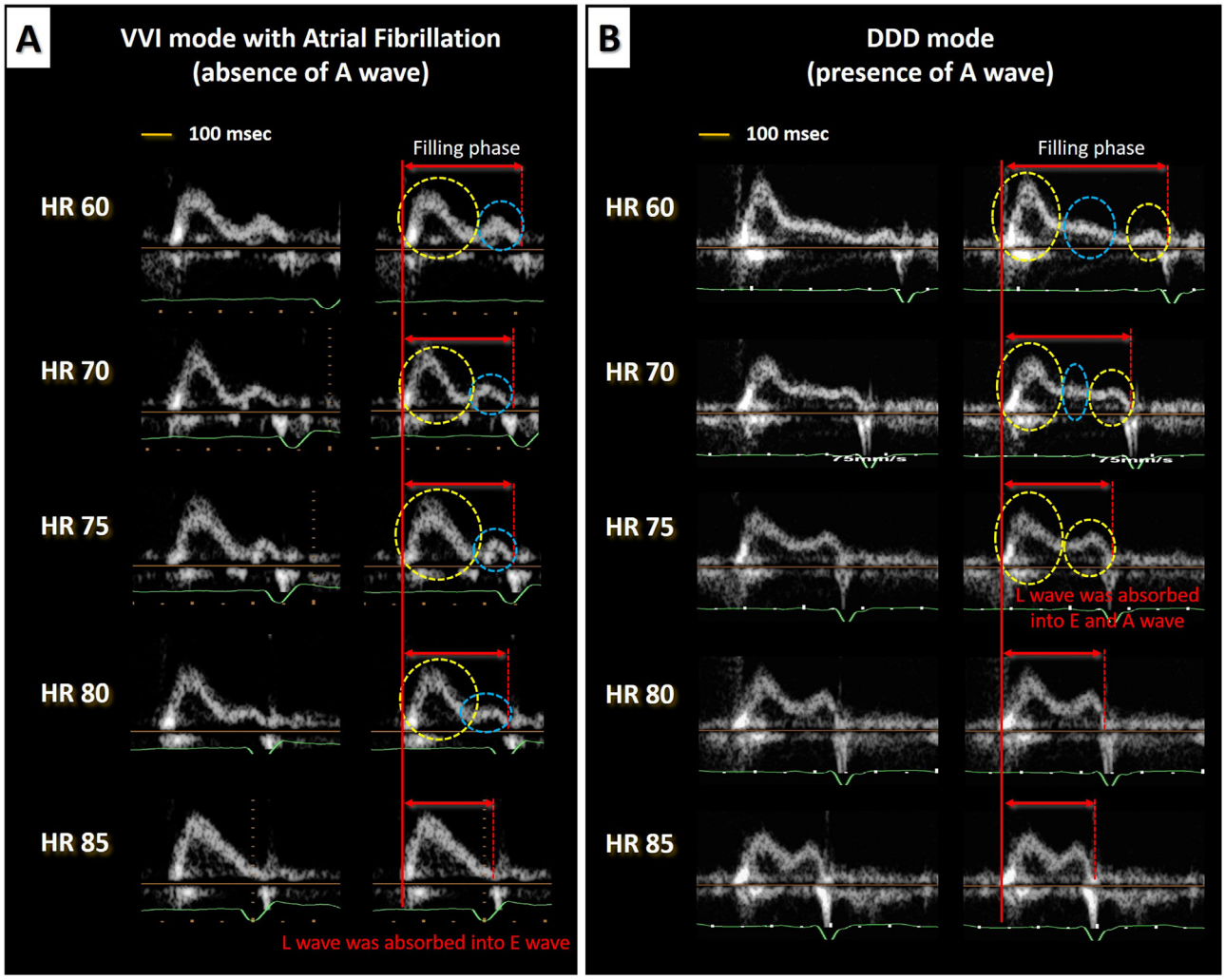

Fig. 3. Serial change in L wave and duration of filling phase in pacing rate of 60, 70, 75, 80, and 85 bpm. (A) VVI mode with atrial fibrillation. Yellow and blue circles indicate E and L wave, respectively. (B) DDD mode. Yellow circles indicate E and A wave. Blue circles indicate L wave. Red arrows indicate left ventricular filling phase.

the absence of A wave in the HR of L wave disappearance. These findings are consistent with the previous reports describing that L wave is more likely to be observed in patients with AF and lower HR [3,4]. In the condition with the presence of A wave, A wave occupied late diastolic phase and shortened the time for L wave existence (Fig. 3B). As HR was increased, LV filling phase was shortened (Fig. 3B), and E and A wave got closer to each other, which eventually led to absorption of L wave into E and A waves (Fig. 3B). On the other hand, in the condition with AF, longer time was secured for L wave existence in late diastole due to the absence of A wave (Fig. 3A). This is possibly a reason why L wave can exist at higher HR in AF than in sinus rhythm.

Through the treatment of heart failure, echocardiography was performed at four different points, i.e. the time of admission, the mid-term of the treatment, pre/post-cardioversion, and the time of discharge. In this serial echocardiographic assessment, we found that L wave was getting smaller and finally disappeared as the fluid retention was being improved. L wave was previously reported to be likely to appear in patients with elevated LV filling pressure. In this case, it is possibly said that improvement in fluid retention resulted in decline of LV filling pressure, leading to disappearance of L wave. From these points, the detection of L wave can potentially be a useful indicator to evaluate treatment efficacy for heart failure. In particular, we may safely describe that disappearance of L wave through treatment in patients with fully controlled HR indicates a sufficient achievement of fluid management and LV filling pressure control.

To date, the mechanisms of L wave have not been elucidated. Keren et al. previously mentioned a hypothesis of the mechanism that refilling of pulmonary vein flow into left atrium (LA) at the end of E wave derives atrioventricular pressure gradient which generates an extra reaccelerating mitral flow, namely L wave [7]. Based on this hypothesis, it can be said that occurrence of L wave requires sufficient fluid volume and duration of diastole. However, up to now, no previous study has demonstrated this hypothesis. In particular, the mechanisms of relationship between HR and L wave have not fully been clarified. In this case report, HR elevation leading to the shortening of LV filling phase seems to directly affect the disappearance of L wave. Meanwhile, the shortening of diastole may cause inadequate conditions for L wave appearance, e.g. less blood filling of LA and/or pulmonary vein. In addition, under AF condition, L wave existence may be influenced by not only the prolongation of the time for L wave existence but also the LA stunning leading to blood stagnation and/or the loss of

blood transference from LA to LV which possibly causes delayed extra reaccelerating mitral flow. Based on Keren's hypothesis, L wave can potentially be influenced by various hemodynamic conditions, and this case report possibly reinforces this hypothesis of the mechanisms. Further studies are needed to elucidate the mechanisms of the phenomena observed in this report.

Conclusion

This case suggests that L wave is highly variable in the therapeutic process of heart failure and strongly affected by fluid volume, heart rate, and heart rhythm. Furthermore, L wave even disappears by improvement of fluid retention and occasionally becomes undetectable by alteration of specific conditions, i.e. HR elevation and conversion to sinus rhythm.

Conflict of interest

The authors declare that they have no conflict of interest.

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