QAOS (QUICK ASSESSMENT OF SYNCHRONY) PROTOCOL: AN ECHOCARDIOGRAPHIC STRATEGY FOR THE ASSESSMENT OF PACEMAKER-INDUCED CARDIAC DYSSYNCHRONY

PROTOCOLO QAOS (AVALIAÇÃO RÁPIDA DA SINCRONIA): UMA ESTRATÉGIA ECOCARDIOGRÁFICA PARA A AVALIAÇÃO DA DISSINCRONIA CARDÍACA INDUZIDA POR MARCAPASSO

PROTOCOLO QAOS (EVALUACIÓN RÁPIDA DE LA SINCRONÍA): UNA ESTRATEGIA ECOCARDIOGRÁFICA PARA LA EVALUACIÓN DE LA DISINCRONÍA CARDÍACA INDUCIDA POR MARCAPASOS

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ABSTRACT

The ventricular activation front generated by a pacemaker resembles that of advanced left bundle branch block, with delayed electromechanical activation of the left ventricular free wall. This leads to contraction dyssynchrony and, eventually, heart failure. Evidence supports the existence of a distinct clinical entity known as Paced-Induced Cardiomyopathy (PIC), defined by the 2023 HRS/APHRS/LAHRS guidelines as a reduction in left ventricular ejection fraction (LVEF) to below 50%, with an absolute decrease in LVEF greater than 10%, a pacing burden of at least 20%, and no other identifiable causes of heart failure following device implantation. The author proposes a protocol named QAOS (Quick Assessment Of Synchrony) for evaluating patients with an existing pacemaker, to enable follow-up and early diagnosis of PIC; and for patients undergoing resynchronization or physiological pacing (His bundle or left bundle branch), to assess whether synchrony has been restored. The protocol consists of 10 steps that follow the sequence of a standard echocardiographic study and does not require highly specialized techniques for its execution. The primary objective of this publication is to encourage the development of pilot studies across different centers devoted to the care of patients with pacemakers and, if their value and clinical applicability are demonstrated, to enable the protocol to be evaluated in clinical trials.

Keywords: Pacemaker. Cardiac Dyssynchrony. Left Ventricular Dysfunction. Echocardiography.

RESUMO

A frente de ativação ventricular gerada por um marcapasso se assemelha àquela observada no bloqueio avançado do ramo esquerdo, com atraso na ativação eletromecânica da parede livre do ventrículo esquerdo. Isso leva à dissincronia da contração e, eventualmente, à

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insuficiência cardíaca. Evidências sustentam a existência de uma entidade clínica distinta conhecida como Cardiomiopatia Induzida por Estimulação (CIE), definida pelas diretrizes HRS/APHRS/LAHRS de 2023 como a redução da fração de ejeção do ventrículo esquerdo (FEVE) para valores inferiores a 50%, com uma diminuição absoluta da FEVE superior a 10%, uma carga de estimulação de pelo menos 20% e ausência de outras causas identificáveis de insuficiência cardíaca após o implante do dispositivo. O autor propõe um protocolo denominado QAOS (Quick Assessment of Synchrony – Avaliação Rápida da Sincronia) para a avaliação de pacientes com marcapasso já implantado, possibilitando o acompanhamento e o diagnóstico precoce da CIE, bem como para pacientes submetidos à terapia de ressincronização ou à estimulação fisiológica (feixe de His ou ramo esquerdo), a fim de avaliar se a sincronia foi restaurada. O protocolo consiste em 10 etapas que seguem a sequência de um estudo ecocardiográfico padrão e não requer técnicas altamente especializadas para sua execução. O objetivo principal desta publicação é incentivar o desenvolvimento de estudos piloto em diferentes centros dedicados ao cuidado de pacientes portadores de marcapasso e, caso seu valor e aplicabilidade clínica sejam demonstrados, possibilitar que o protocolo seja avaliado em ensaios clínicos.

Palavras-chave: Marcapasso. Dissincronia Cardíaca. Disfunção do Ventrículo Esquerdo. Ecocardiografia.

RESUMEN

El frente de activación ventricular generado por un marcapasos se asemeja al observado en el bloqueo avanzado de la rama izquierda, con retraso en la activación electromecánica de la pared libre del ventrículo izquierdo. Esto conduce a disincronía de la contracción y, eventualmente, a insuficiencia cardíaca. La evidencia respalda la existencia de una entidad clínica distinta conocida como Cardiomiopatía Inducida por Estimulación (CIE), definida por las guías HRS/APHRS/LAHRS de 2023 como una reducción de la fracción de eyección del ventrículo izquierdo (FEVI) por debajo del 50%, con una disminución absoluta de la FEVI superior al 10%, una carga de estimulación de al menos el 20% y ausencia de otras causas identificables de insuficiencia cardíaca tras el implante del dispositivo. El autor propone un protocolo denominado QAOS (Quick Assessment of Synchrony – Evaluación Rápida de la Sincronía) para la evaluación de pacientes con marcapasos implantado, que permita el seguimiento y el diagnóstico precoz de la CIE, así como para pacientes sometidos a terapia de resincronización o a estimulación fisiológica (haz de His o rama izquierda), con el fin de evaluar si se ha restablecido la sincronía. El protocolo consta de 10 pasos que siguen la secuencia de un estudio ecocardiográfico estándar y no requiere técnicas altamente especializadas para su ejecución. El objetivo principal de esta publicación es fomentar el desarrollo de estudios piloto en distintos centros dedicados a la atención de pacientes portadores de marcapasos y, si se demuestra su valor y aplicabilidad clínica, permitir que el protocolo sea evaluado en ensayos clínicos.

Palabras clave: Marcapasos. Disincronía Cardíaca. Disfunción del Ventrículo Izquierdo. Ecocardiografía.

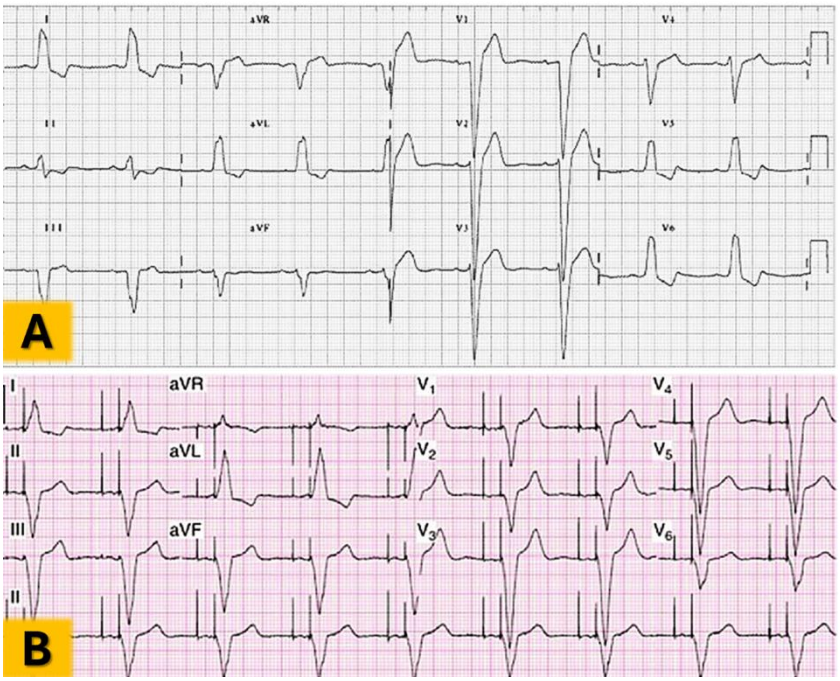
1 INTRODUCTION

The primary goals of permanent cardiac pacemaker implantation are, first and foremost, to save lives, and in less critical cases, to improve quality of life. More recently, there is growing consensus around a third, equally important objective: to preserve cardiac synchrony—or, alternatively stated, to prevent or treat established dyssynchrony—in order to avoid left ventricular dysfunction leading to heart failure. Optimal heart mechanical performance requires an intact impulse generation and conduction system, both structurally and functionally. Any condition affecting this system results in non-physiological cardiac activation, thereby reducing global mechanical efficiency[1].

Right ventricular chronic artificial stimulation has been common practice for several reasons: There is a direct and safe venous access, the implantation procedure is relatively simple, and essential ventricular stimulation is guaranteed. However, this approach comes at a cost—especially when the lead is positioned at the right ventricular apex because the ventricular activation front generated by a pacemaker resembles that of advanced left bundle branch block, with delayed electromechanical activation of the left ventricular free wall (Figure 1).

Figure 1

Comparison of the electrocardiogram from a patient with left bundle branch block (Panel A) and the tracing from a patient with a pacemaker showing right ventricular apical pacing (Panel B)



Dyssynchronous contraction ultimately leads to heart failure[2] through various mechanisms—even in dual-chamber pacing modes[3]. Advanced imaging techniques, as speckle-tracking echocardiography (STE), has been useful to depict the extent of such dyssynchrony (see Figures 2 and 3), but these applications demand expensive equipment that are not widely available, particularly in developing countries.

The first study specifically designed to demonstrate pacemaker induced ventricular dysfunction dates back to 2006: the HOBIPACE trial[4], which focused on ventricular function, exercise tolerance, and quality of life. The BLOCK-HF study, involving patients with a baseline left ventricular ejection fraction (LVEF) below 50%, showed that biventricular pacing led to reduced cardiac remodeling, heart failure, and overall morbidity and mortality compared to exclusive right ventricular pacing[5]. Gage, Burns, and Bank reached similar conclusions in a 2014 publication[6].

Figure 2

Left ventricular polar maps. Pacemaker-induced left intraventricular dyssynchrony. Acute changes are shown with speckle-tracking strain echocardiography, both in native rhythm (left) and during pacing (right ventricular apical lead, right). Mechanical dispersion (PSD: Peak strain deviation) increased from 53 ms (normal) to 177.7 ms (severely abnormal), confirming new dyssynchrony caused by pacing

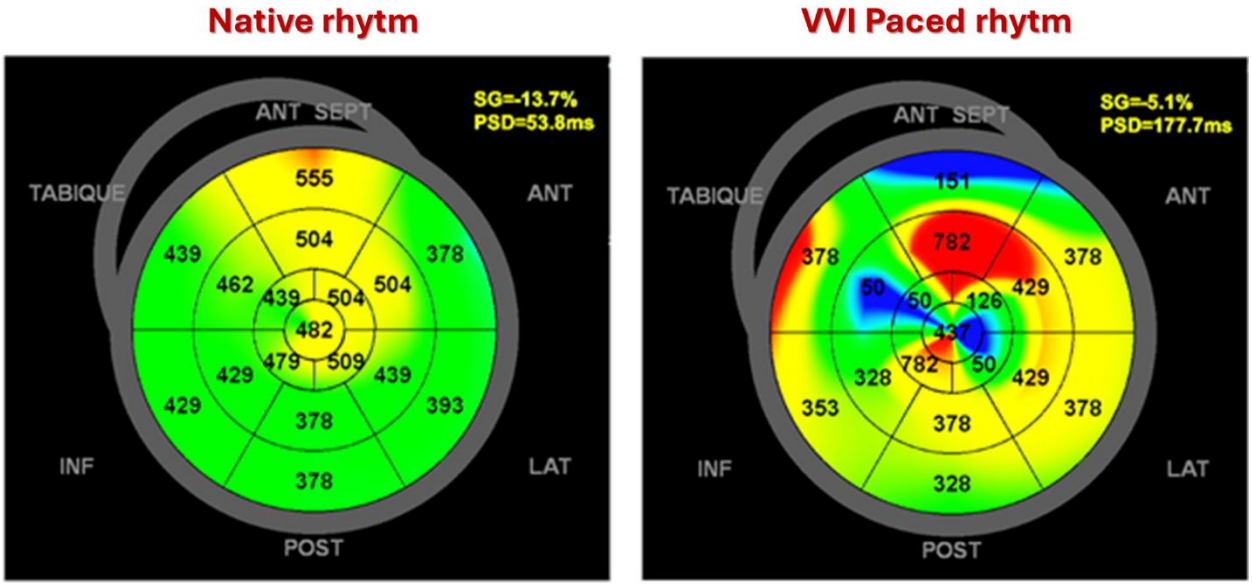
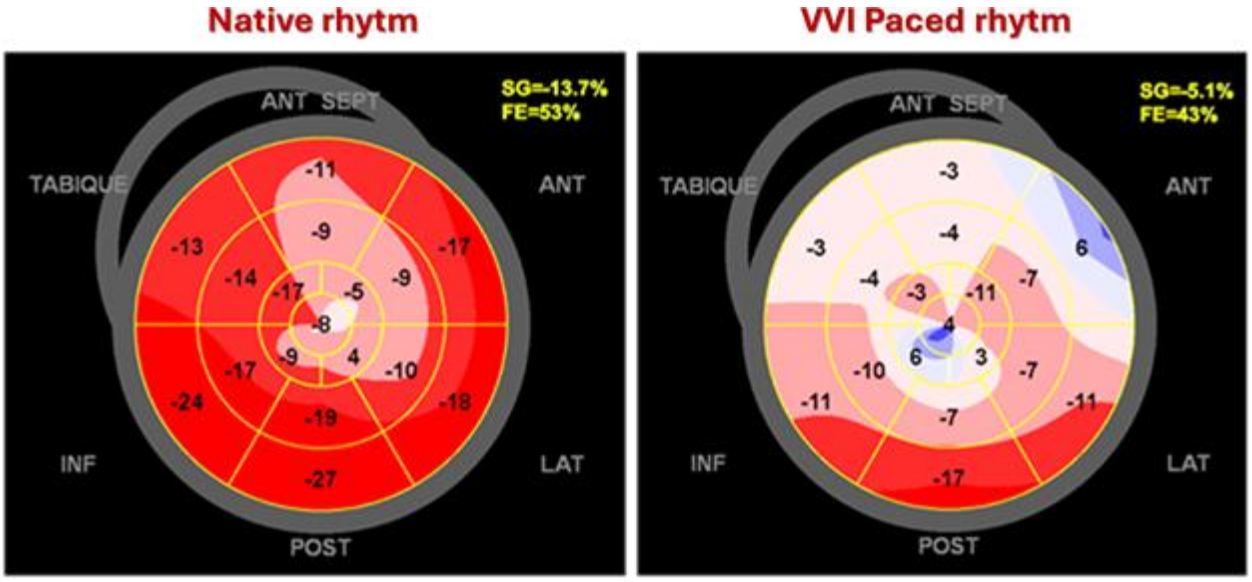


Figure 3

Left ventricular polar maps. Pacemaker-induced ventricular dysfunction. Acute changes are shown in the same patient from Figure 3, assessed with speckle-tracking strain echocardiography. Left ventricular function is compared in native rhythm (left) and during pacing from a right ventricular apical lead (right): Left ventricular ejection fraction (EF) decreases from 53% to 43%, and global longitudinal strain (SG) drops dramatically from –13.7% to –5.1%



The cited evidence supports the existence of a distinct clinical entity: Paced-Induced Cardiomyopathy (PIC), defined by the 2023 HRS/APHRS/LAHRS guidelines as a reduction in left ventricular ejection fraction (LVEF) to below 50%, with an absolute decrease in LVEF greater than 10%, a ventricular pacing burden of at least 20%, and no other identifiable causes of heart failure following device implantation[7]. Kim et al. argue that while the drop in LVEF is a key criterion, the emergence of new heart failure symptoms, hospitalization, and/or new-onset atrial fibrillation should also be considered in the PIC concept, although PIC may occur in the absence of overt left ventricular failure symptoms[8].

Regarding its incidence, a recent multicenter study by Somma et al.—a meta-analysis of 26 studies including approximately 57,993 patients—reported a 12% incidence of new PIC cases (range: 6–39%) over a follow-up period ranging from 0.7 to 16 years[9]. Reported incidence rates are expected to vary depending on the criteria used to define PIC and the duration of follow-up[8].

Several risk factors have been studied in relation to PIC development[8]:

- Advanced age.
- Male gender.[10]
- Pre-implantation atrial fibrillation.[11]

- Reduced LVEF.
- Prolonged QRS duration.[10]
- Diastolic dysfunction.
- Abnormal global longitudinal strain.
- Complete AV block as the indication for pacemaker implantation.[11]
- Ventricular pacing burden greater than 40%.
- Chronic kidney disease and myocardial infarction have also been proposed as contributing risk factors[12].

Regarding pacing burden, Kiehl et al. suggest that heart failure risk increases with a pacing burden of 40%, showing a linear correlation between pacing percentage and the onset of atrial fibrillation—even when AV synchrony is preserved with DDD pacing[13]. Sweeney et al. also propose 40% as a threshold for increased heart failure risk (OR: 2.5)[3].

Relative risks of all-cause mortality and new-onset heart failure in PIC patients have been compared to those in pacemaker recipients without PIC (Cho et al.[14]). Auger et al.[15] conducted a similar analysis using a dyssynchrony index. Both studies independently demonstrated significant increases in all-cause mortality and heart failure. The DAVID trial[16] also showed that unnecessary DDD pacing led to higher morbidity and mortality compared to backup-only pacing.

Although conduction system pacing has emerged as a physiological strategy aimed at preserving normal ventricular activation through left bundle branch capture (left bundle branch pacing – LBBP), recent studies warn that this modality is not exempt from the risk of inducing ventricular dyssynchrony. In a contemporary cohort, a 3.75% incidence of newly developed left ventricular dysfunction following LBBP was reported (Ponnusamy et al.[17]). Moreover, the distinction between left ventricular septal pacing (LVSP) and LBBP—in terms of synchrony—is not always clinically evident, although parameters such as QRS area reduction suggest a slight superiority of LBBP in achieving more physiological ventricular activation (Heckman et al.[18]).

Accumulated evidence on pacing-induced cardiomyopathy (PIC) reveals that electrical and mechanical dyssynchrony—typically associated with “conventional” right ventricular pacing—can trigger ventricular remodeling, fibrosis, and progressive deterioration of left ventricular ejection fraction, even in patients without preexisting structural heart disease (Mizner J[19], Ferrari ADL[20]). Therefore, given the diversity of techniques and outcomes within conduction system pacing—particularly in the left bundle branch region—it is essential not to assume that physiological pacing guarantees complete synchrony.

In El Salvador, since January 2024, we have selected 35 patients of both sexes (15 female, mean age 74.2+13.5 years, 7 with cardiac resynchronization devices, and two with left bundle branch pacing) for a prospective pilot study on the incidence of PIC. The protocol is carried out during device follow-up visits, scheduled at least annually, except in cases of heart failure symptoms. Follow-up is expected to continue at least through January 2027; however, 8 patients has already diagnosed with PIC (22.9% of incidence), one of them required an upgrade from DDDR to left bundle branch pacing, the other is still waiting for upgrade; we have registered one death, not related with cardiac disease. The implementation, under the author's supervision, has proceeded efficiently and without significant obstacles.

2 OBJECTIVES

This is an extended version of an original paper published in January 2026, in the Romanian Journal of Cardiology21. The author proposes a protocol for all patients who already have an implanted device, regardless of modality with the aim of identifying criteria for lost and/or restored synchrony and documenting potential impairment and/or recovery of clinical markers and left ventricular function. This protocol has been named QAOS (Quick Assessment Of Synchrony), with the intention to promote design and carry out local pilot projects and, subsequently, if future clinical trials demonstrate it to be an effective and efficient procedure, to pursue its definitive implementation.

3 METHODS

To implement the protocol, a data collection instrument (see Table 1) has been designed to capture demographic, clinical, pacing burden, left ventricular function parameters, hemodynamic data, and atrioventricular, interventricular, and left intraventricular synchrony metrics. All measurements and calculations has been reported as useful tools for dyssynchrony evaluation (see specific references). This instrument may be applied prospectively as needed and should be included in each patient's medical record. The minimum required clinical data fields are:

- Healthcare facility.
- Evaluation date.
- Patient identification/demographics: Sex, Date of birth / age.
- Blood pressure (mmHg).
- Pacemaker implantation date and indication.
- Pacemaker brand and model, as well as pacing modality.

- Right ventricular pacing burden.

Regarding ECHO data, if left ventricular function indices are normal during pacing, it is not necessary to repeat the measurements in native rhythm. However, if there is any evidence of left ventricular dysfunction in patients who are not fully pacemaker-dependent, measurements and calculations should be performed both during pacing and in native rhythm, in order to confirm whether pacing itself is responsible for the dysfunction. Technical support from the device manufacturer may be requested to assist with programmer use.

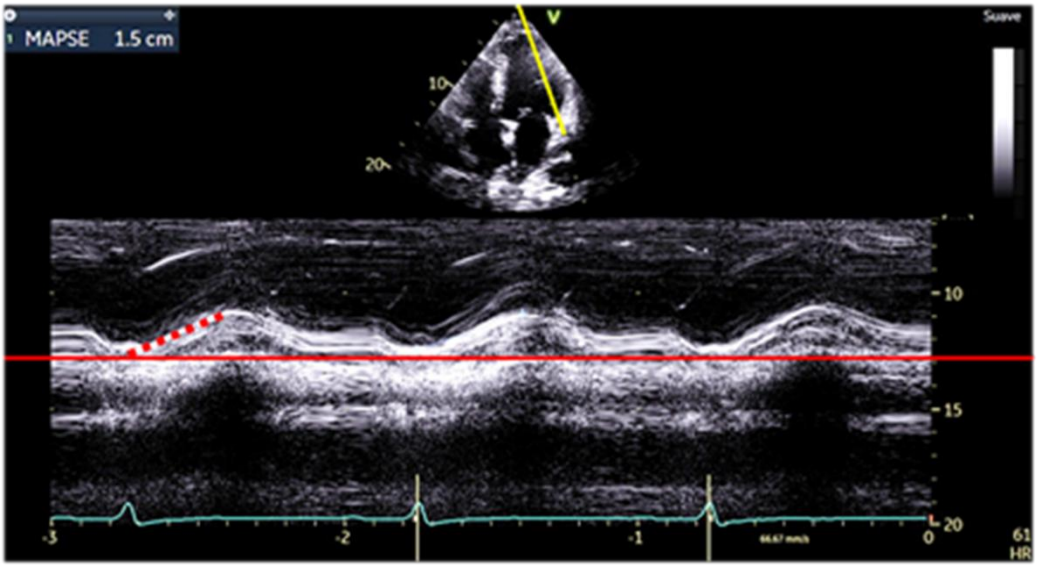
3.1 CALCULATIONS AND MEASUREMENTS

1. Assessment of Left Ventricular Systolic Function Parameters.

- MAPSE (Mitral Annular Plane Systolic Excursion): This parameter correlates strongly with global longitudinal strain22. It is measured in the apical four-chamber view (Ap4C) using M-mode (Figure 4).

Figure 4

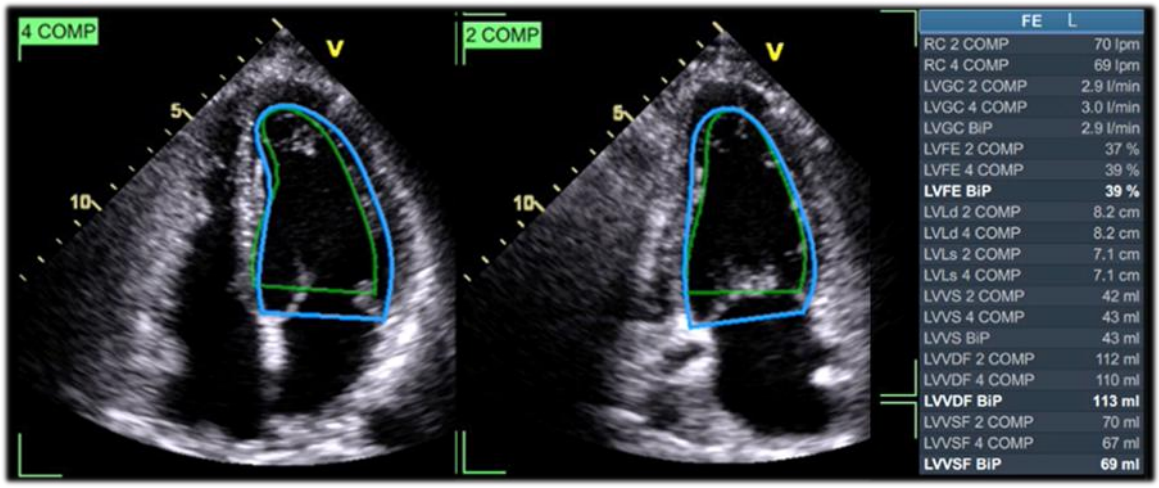
MAPSE, M-Mode, mitral lateral annulus



- Left Ventricular Ejection Fraction (LVEF): Ideally assessed using biplane apical or three-dimensional (3D) echocardiography. Automated methods allow averaging over three consecutive cardiac cycles (Figure 5).

Figure 5

Apical 4-chamber and 2-chamber views in a patient with non-ischemic systolic dysfunction. Automated (with manual correction) measurement of left ventricular cavity volumes for calculation of the ejection fraction (LVEF)



- Global Longitudinal Strain (GLS): If speckle tracking is available, both average strain and mechanical dispersion should be recorded (Figure 6). If not, the HUNT method23 may be used, with a lower cutoff value (–16%, Figure 7).

Figure 6

Longitudinal strain by 2D speckle-tracking. Strain-versus-time curves and polar map. A: Four-chamber strain curves; B: Two-chamber strain curves; C: Parasternal long-axis strain curves. Note the different timings of peak deformation and the limited negative deflection, reflecting dyssynchrony and reduced strain in a patient with advanced heart failure. D: Polar map: Color heterogeneity reflects heterogeneous strain; pale tones indicate subnormal values

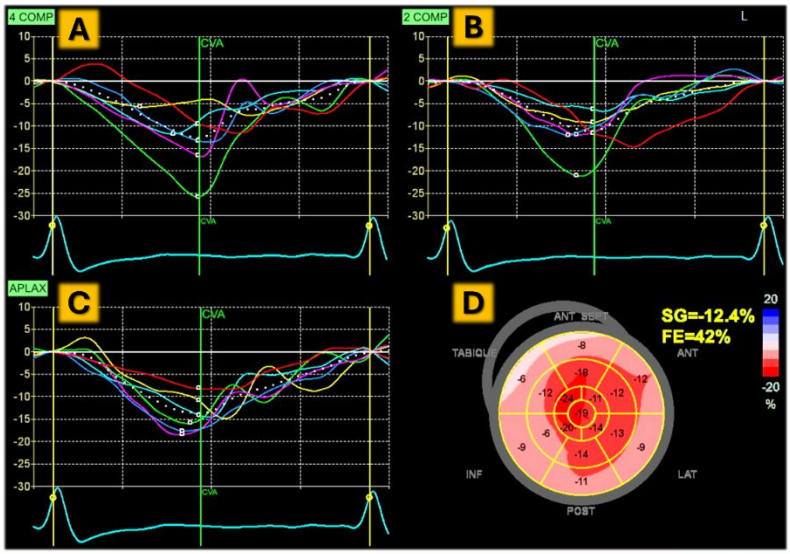
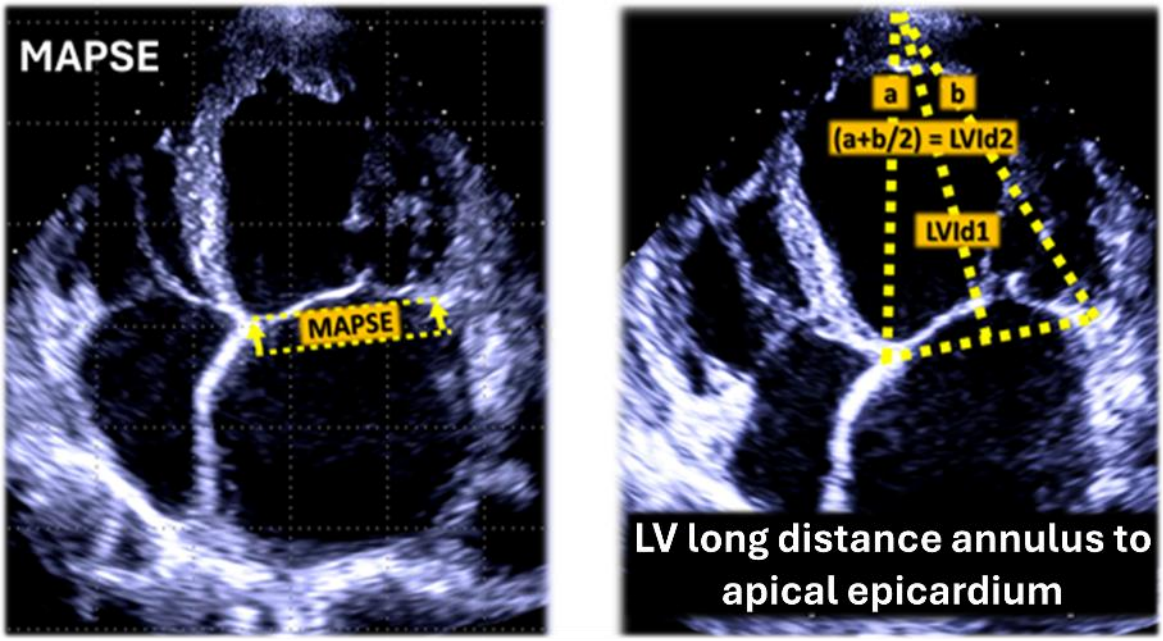


Figure 7

Measurement of MAPSE and the left ventricular length from the mitral annular plane to the apical epicardium (LV length) are required for longitudinal strain calculation with the HUNT method



3.2 QAOS PROTOCOL PARAMETERS

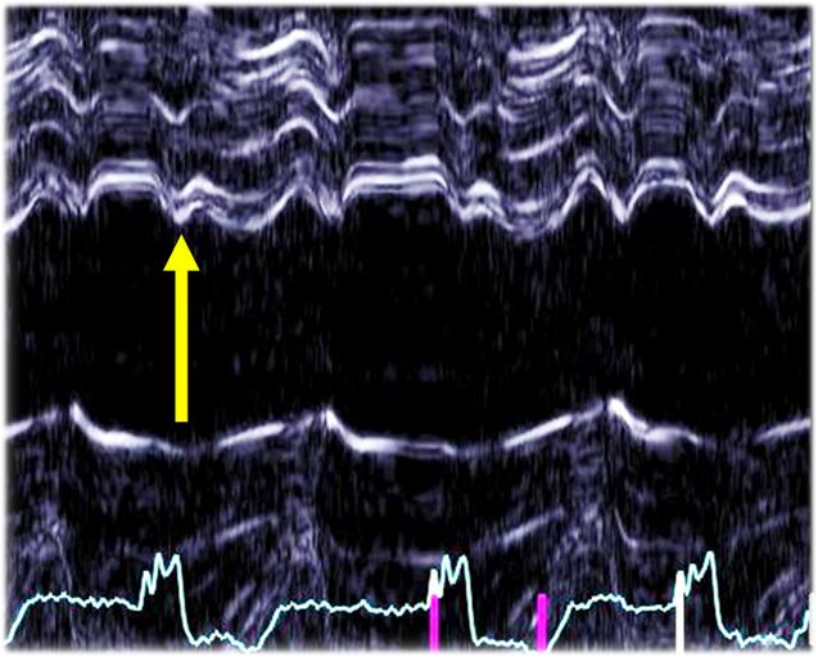
The protocol consists of 10 steps:

- Step 1: Septal Flash Detection.

Septal flash: Brief septal movement during isovolumetric contraction, typical of left bundle branch block, also observed in right ventricular pacing (Figure 8). It is documented using M-mode of the anterior septum in the parasternal long-axis view (PLAX) and serves as a reliable dyssynchrony marker24.

Figure 8

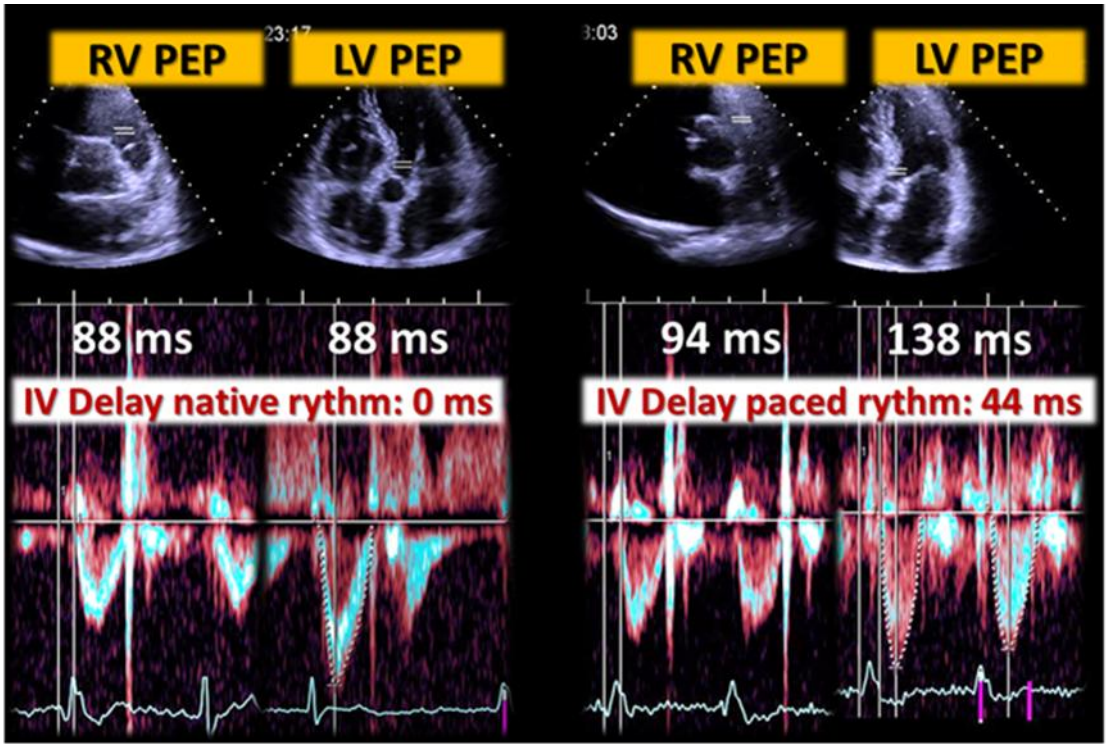
Septal flash: Inward–outward septal motion occurring during isovolumetric contraction (arrow). The patient has a left bundle branch block



- Step 2: Measurement of Left Ventricular Outflow Tract (LVOT) and calculation of LVOT Area (A-LVOT). These measurements (also in PLAX) are needed to calculate stroke distance and volume.
- Step 3: Right Ventricular Pre-Ejection Period. Using pulsed-wave Doppler in the right ventricular outflow tract (RVOT) in the parasternal short-axis view of the great vessels (PSAX), the interval from QRS onset to the start of systolic flow is measured in milliseconds (ms, Figure 9).
- Step 4: Left Ventricular Pre-Ejection Period. Using pulsed-wave Doppler in the LVOT, the interval from QRS onset to the start of systolic flow is measured in ms (Figure 9).
- Step 5: Interventricular Delay Calculation. To diagnose interventricular dyssynchrony, subtract the right and left pre-ejection periods—ideally in both native and paced rhythm (Figure 9). Delay >40 ms confirms dyssynchrony25.

Figure 9

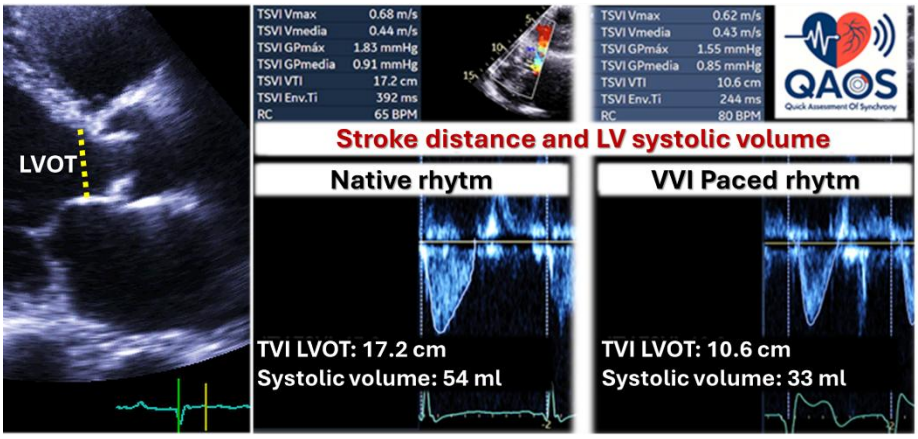
Pre-ejection periods of the outflow tracts of both ventricles (RV PEP: Right ventricular pre-ejective period, LV PEP: Left ventricular pre-ejective period). Left: Interventricular (IV) delay in native rhytm. Right: IV delay in paced rhytm, an interventricular dyssynchrony is diagnosed because the delay is longer than 44 ms



- Step 6: LVOT Stroke Distance (Time-Velocity Integral). By tracing the systolic jet contour in the LVOT, the stroke distance is obtained. Stroke volume is calculated by multiplying this distance by the LVOT area (Figure 10).

Figure 10

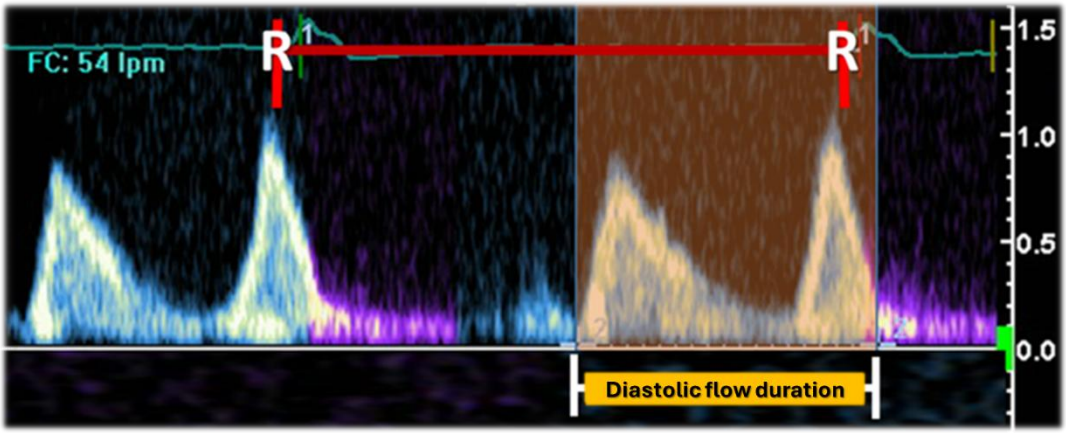
Left: LVOT measurement, in systole. Center and right: Time–velocity integral of the left ventricular outflow tract (LVOT VTI), in native and paced rhythm



- Step 7: Apical Rocking. Often associated with septal flash, is a well-recognized sign of left intraventricular dyssynchrony26, best assessed in the Ap4C view. In the PREDICT-CRT study apical rocking and septal flash added predictive power beyond clinical variables and QRS duration for identifying responders to cardiac resynchronization therapy. Its absence or failed reversal has been linked to poor survival outcomes27.
- Step 8: Cardiac Cycle Duration. Is the R–R interval, recorded in ms.
- Step 9: Diastolic Flow Duration. In the apical view, using pulsed Doppler, mitral inflow is traced from the onset of the E wave to mitral valve closure, regardless of A wave presence (Figure 11).
- Step 10: Diastolic Flow/R–R Ratio. Diastolic flow duration (in ms) divided by R–R interval, multiply by 100 to obtain a percentage. Normal value: >40%; any value below this threshold confirms AV dyssynchrony28 (Figure11).

Figure 11

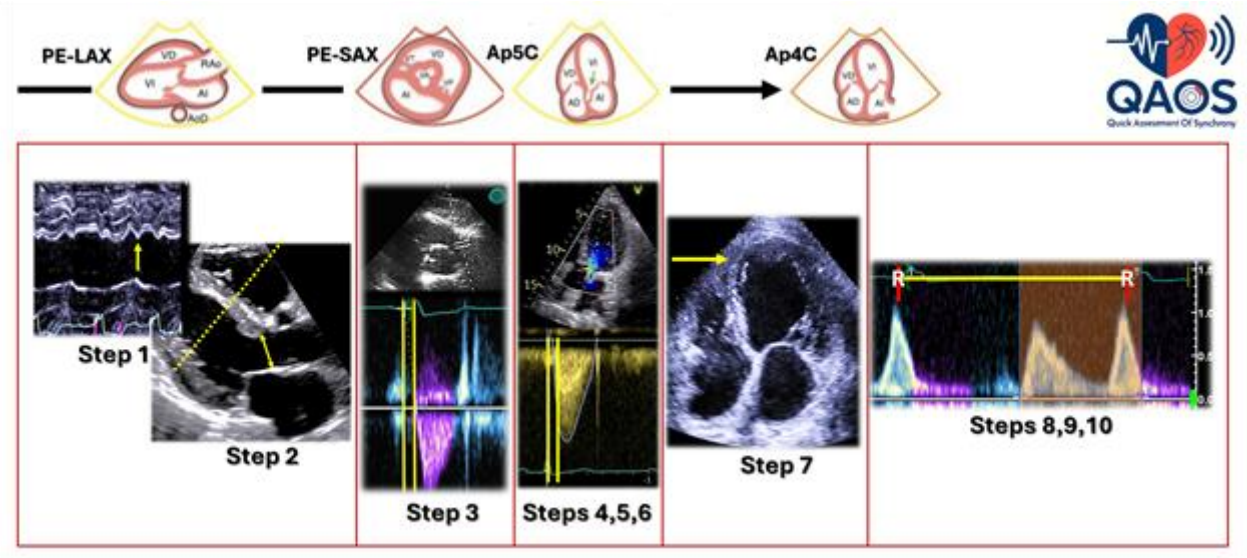
Assessment of atrioventricular synchrony. The diastolic flow percentage is 52.6%, confirming that synchrony is preserved



Finally, in summary Figure 12 shows the sequence of steps suggested for applying the QAOS protocol (Figure 12).

Figure 12

The image acquisition process adheres to the conventional sequence of standard echocardiography. The QAOS steps are illustrated in the parasternal long-axis (PE-LAX), short-axis views (PE-SAX, at the level of the great vessels), apical five- (Ap5C) and four-chamber (Ap4C) views



In our ECHO Lab, after MAPSE and LVEF measurement, execution times for the 10 protocol steps were timed; after 35 procedures, the average duration was 5 minutes and 36 seconds ± 44 seconds.

Once all data are collected, they are entered into a specific instrument (see Table 1), providing specific answers to the following questions —both in native rhythm and under artificial pacing, if necessary:

- Is there interventricular dyssynchrony?
- Is there left intraventricular dyssynchrony?
- Is there AV dyssynchrony?
- Can PIC be diagnosed?
- Are there criteria for pacemaker upgrade to cardiac resynchronization therapy or physiological pacing?

To provide practical examples of the implementation of QAOS protocol, Table 1 presents a side-by-side comparison between two clinical cases of symptomatic patients with pacemakers.

Table 1

Comparative Analysis with QAOS Protocol: Two cases of patients with pacemakers.

QAOS Quick Assessment Of Synchrony	Case A Male, 85 y-o Ischaemic heart disease		Case B Male, 68 y-o Non ischaemic heart disease	
Heart failure symptoms?	NYHA Class II		NYHA Class II	
RV pacing %	62.3%		55%	
Heart Rythm	Native Sinus	VVI	Native Atrial Fib	VVI
LVEF (%)	58%	56%	48%	37.9%
Global Longitudinal Strain (%)	-16.4%	-12.5%	-24.2%	-15.5%
QAOS Step 1: Septal flash?: (Y/N/Not evaluable)	NO	YES	NO	YES
QAOS Step 2: LVOT Area (cm^2)	2.27 cm^2		3.14 cm^2	
QAOS Step 3: VD pre-expulsive period (ms)	55 ms	116 ms	88 ms	94 ms
QAOS Step 4: LV pre-expulsive period (ms)	87 ms	176 ms	88 ms	138 ms
QAOS Step 5: Interventricular delay (ms)	32 ms	60 ms	0	44 ms
Interventricular synchrony? (Y/N)	YES	NO	YES	NO
QAOS Step 6: LVOT VTI (cm)	25.2 cm	14.5 cm	17 cm	14 cm
LV stroke volume (ml)	56 ml	32 ml	51.5 ml	44 ml
QAOS Step 7: Rocking apex: (Y/N/Not evaluable)	NO	YES	NO	YES
Intraventricular synchrony? (Y/N)	YES	NO	YES	NO
QAOS Step 8: R-R interval (ms)	1005 ms	1000 ms	822	740 ms
QAOS Step 9: Mitral diastolic flow duration (ms)	614 ms	382 ms	391	254 ms
QAOS Step 10: Mitral diastolic flow duration ÷ R-R: (%)	61.1%	38.2%	47.6%	34.3%
A-V Synchrony? (Y/N)	YES	NO	YES	NO
Can PIC be diagnosed?	NO		YES	
There is a need for a pacemaker upgrade?	NO		YES	

Comments about Table 1:

- In Case A, the patient presents with ischemic structural heart disease and non-transmural scarring following a myocardial infarction; eventually the patient developed a complete A-V block and a unicameral pacemaker was implanted; to date, left ventricular ejection fraction (LVEF) is normal. The patient is in NYHA functional Class II. There is a high demand of ventricular pacing and has a markedly deleterious effect on synchrony and myocardial strain, but no effect in LVEF, which is why a pacing upgrade is not currently indicated.
- In Case B, the patient presents without structural ventricular damage but remains symptomatic. Pacing-induced dyssynchrony develops with a device dependency of 55%, accompanied by a 10% reduction in left ventricular ejection fraction (LVEF). In this context, pacing-induced cardiomyopathy (PIC) can be diagnosed, and an upgrade to physiological pacing is recommended.

4 DISCUSSION

It is recommended to incorporate tools for risk estimation, prevention, and detection of early, subclinical, or latent forms of dyssynchrony and/or PIC, both before and after device implantation:

- Serial electrocardiographic analysis (QRS morphology and duration).
- Monitoring of electrical parameters such as left ventricular activation time.
- Active surveillance in patients with high pacing burden (>20%), even with left bundle branch pacing.
- Imaging-based strategies to assess ventricular function and mechanical synchrony or dyssynchrony.

With respect to imaging strategies, in patients with permanent pacemakers, echocardiography is indicated based on specific clinical criteria. QAOS protocol (besides a complete echocardiographic study) could be implemented in the follow-up if a high burden of ventricular pacing (>40%) is associated with an increased risk of PIC, which justifies echocardiographic evaluations, every 6 to 12 months. Likewise, the emergence of new or progressive symptoms of heart failure constitutes an immediate indication for echocardiographic assessment. In patients undergoing cardiac resynchronization therapy (CRT) and conduction system pacing, echocardiography is also recommended 3–6 months after implantation to assess response28, QAOS can be a useful tool in these instances because all the measurements and calculations has been already validated.

5 CONCLUSION & CLINICAL RELEVANCE

There are clinical scenarios in which the QAOS protocol is of paramonutn interest and may have significant practical application:

1. In patients with right ventricular pacing, QAOS may be used to detect pacing-induced dyssynchrony and/or ventricular dysfunction. This should raise concern, given the natural history of such dyssynchrony often progresses toward heart failure[12], driven by a well-recognized cascade of functional deterioration. This underscores the need for periodic QAOS evaluations in dyssynchronous patients to identify the optimal timing for cardiac resynchronization therapy or conduction system pacing, beyond optimal medical management. The simplicity of the protocol allows for rapid assessment in both paced and native rhythm (when feasible), with a recommended interval of one to two minutes between evaluations.
2. QAOS may be a valuable tool—alongside clinical assessment—for verifying the effectiveness of cardiac resynchronization therapy or conduction system pacing, confirming whether the patient is a responder with improvement in symptoms and markers of synchrony and systolic function.

This protocol is published to authorize its free use in both routine clinical practice and research settings. Users are encouraged to adhere faithfully to the protocol, while remaining open to suggestions that may enhance its design. The development of a digital application for use on electronic devices is envisioned as a next step, should field results prove promising.

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