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CASE REPORT

Blocked Atrial Bigeminy as an Unusual Cause of Bradycardia: A Case Report

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Video Abstract

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Abstract: The diagnosis of blocked atrial bigeminy may be challenging. It can easily be misinterpreted as sinus bradycardia or sinus exit block leading to unnecessary pacemaker implantation. We report a case of frequent blocked premature atrial contractions leading to episodes of marked bradycardia. A 66-year-old man disturbed with low pulse (42 bpm) measured by an automated blood pressure monitor sought medical attention. The patient also had untreated grade I arterial hypertension. Ambulatory ECG monitoring was performed and very frequent blocked premature atrial contractions were seen. The P wave of the atrial premature atrial contractions was always superimposed on the ST segment or T wave simulating a bifid T wave. Blocked atrial quadrigeminy, blocked atrial trigeminy, and blocked atrial bigeminy were observed. The minimum heart rate associated with blocked atrial bigeminy was 37 bpm. There were multiple episodes of regular ventricular rhythm during blocked atrial bigeminy. NT-proBNP concentration was within normal limits. Echocardiography showed only mild left ventricular hypertrophy, mild left atrial dilation and type I diastolic dysfunction. Therapy with flecainide and amiodarone separately was not effective. Option of radio-frequency catheter ablation was discussed with the patient, but was not accepted due to procedural risks and absence of severe symptoms related to blocked PACs. The patient was reassured and regular follow-up was recommended. The target values of blood pressure were achieved with candesartan at a dose of 32 mg. Thus, blocked atrial bigeminy is a rare cause of bradycardia with a regular ventricular rhythm. Recognition of the P wave superimposed on the ST segment or the T wave may be a clue for a correct diagnosis. Physicians should be aware of this condition to avoid unnecessary pacemaker implantation.

Keywords: ambulatory ECG monitoring, premature atrial contractions, extrasystoles, antiarrhtyhmic agents, supraventricular bigeminy, low heart rate

Introduction

Premature atrial contraction (PAC) is defined as the depolarization of the atria that originates from atrial tissue other than the sinoatrial node prematurely. These ectopic beats are often detected in the general population and are more prevalent than previously understood; studies indicate that PACs are observed in approximately 60.8% of healthy individuals.[1] In instances where PACs do not induce significant symptoms or discomfort, medical intervention is frequently deemed unnecessary, and the most common approach in clinical practice is to provide reassurance to the patients regarding the benign nature of their condition.[2]

Historically, PACs were regarded as harmless, with little to no impact on cardiovascular health; however, recent studies have demonstrated that sustained or frequent PACs can contribute to adverse atrial structural remodeling,[3] which may, in turn, predispose patients to more serious cardiovascular complications such as atrial fibrillation, an increased risk of stroke, heart failure, a decline in renal function, and ultimately, mortality.[2,4–8] Additionally, the phenomenon of PAC-induced cardiomyopathy has been extensively documented, revealing that the irregularity of ventricular contractions plays a significant role in its development.[9,10]

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The specific site from which PACs originate significantly influences the potential consequences associated with PACs. For instance, PACs that originate from the pulmonary veins have been found to have a higher propensity to induce atrial fibrillation compared to those that arise from other atrial sites.[11]

In terms of therapeutic options, it is well established that beta-blockers, particularly when administered at low dosages, may alleviate symptoms associated with PACs and are associated with a nearly 40% reduction in mortality risk.[12] Furthermore, the use of class IC antiarrhythmic agents, such as flecainide and propafenone, has been explored, with data suggesting that flecainide may prove to be more effective in suppressing supraventricular ectopy than propafenone.[13] For patients who do not respond adequately to pharmacological treatment, catheter ablation can be alternative.[14]

Typically, on ECG, PAC is manifested as a premature P wave followed by a QRS complex with a morphology that is consistent with that of sinus beats. The P wave morphology in PACs differs from the one seen in sinus beats, which can be important for correct diagnosis. However, there are instances where the premature P wave may be followed by a wide QRS complex that has a morphology distinct from sinus beats; this phenomenon is referred to as aberrant conduction. Aberrant conduction occurs when there is a delay in the conduction in one of bundle of His, causing the QRS complex to widen, which can confuse the diagnosis and need precise differentiation from premature ventricular contractions. Moreover, blocked PACs are characterized by a premature P wave that is not followed by a QRS complex. This can create additional diagnostic challenges, as the P wave can sometimes be superimposed on the T wave of the preceding beat, making it difficult to discern the underlying rhythm on an electrocardiogram. Blocked atrial bigeminy is a rhythm abnormality in which every normal sinus beat is followed by a PAC that fails to conduct to the ventricles. Frequent occurrences of blocked PACs can lead to symptomatic bradycardia, where the heart rate drops significantly, potentially causing dizziness, fatigue, or other concerning symptoms.[15–17]

The challenge of accurately diagnosing blocked PACs cannot be understated, as these conditions can often be misdiagnosed. In some cases, patients experiencing frequent blocked PACs may be mistakenly referred for pacemaker implantation.[18]

Case Report

A 66-year-old man, experiencing a notably low pulse rate of 42 beats per minute as measured by an automated blood pressure monitor, sought medical attention. The patient had no symptoms. He had a history of untreated grade I arterial hypertension. Upon examination in the clinic, his heart rate registered within the normal range. A 24-hour ambulatory ECG monitoring was performed. High frequency of blocked premature atrial contractions (PACs) was found. The P wave of atrial PACs always superimposed on the ST segment or T wave creating a misleading appearance of a bifid T wave. There were blocked atrial quadrigeminy (Figure 1A), blocked atrial trigeminy (Figure 1B), blocked atrial bigeminy (Figure 1C–E). Notably, during episodes of blocked atrial bigeminy, the minimum heart rate plummeted to 37 beats per minute, as depicted in Figure 1F. There were multiple episodes of regular slow ventricular rhythm associated with blocked atrial bigeminy episodes, as shown in Figures 1D and F. Also, the episodes of atrial bigeminy with aberrant conduction were found (Figure 2). No episode of atrial fibrillation was found during ambulatory ECG monitoring. The concentration of NT-proBNP was measured at 115 pg/mL, which fell within the normal range. An echocardiogram revealed only mild left ventricular hypertrophy, mild left atrial dilation, and the presence of type I diastolic dysfunction. These abnormalities were sought to be caused by arterial hypertension.

In an effort to manage the patient's arrhythmia, he was initially prescribed flecainide at a dose of 200 mg daily. However, during a follow-up appointment on day five, atrial bigeminy was seen again on a conventional ECG. Consequently, amiodarone was administered at a dose of 400 mg daily. Despite this intervention, the patient continued to exhibit frequent PACs upon subsequent ECG evaluations, indicating a lack of response to both class IC and III antiarrhythmic agents. Given the persistent nature of the arrhythmia and the ineffective response to pharmacological treatments, the option of radiofrequency catheter ablation was discussed with the patient. However, considering absence of severe symptoms related to PACs, absence of PAC-induced cardiomyopathy signs, procedural risks, and cost of procedure, the option of radiofrequency catheter ablation was not accepted. He was reassured that his condition was not life-threatening. It was emphasized that he should engage in regular check-ups which would include monitoring his blood

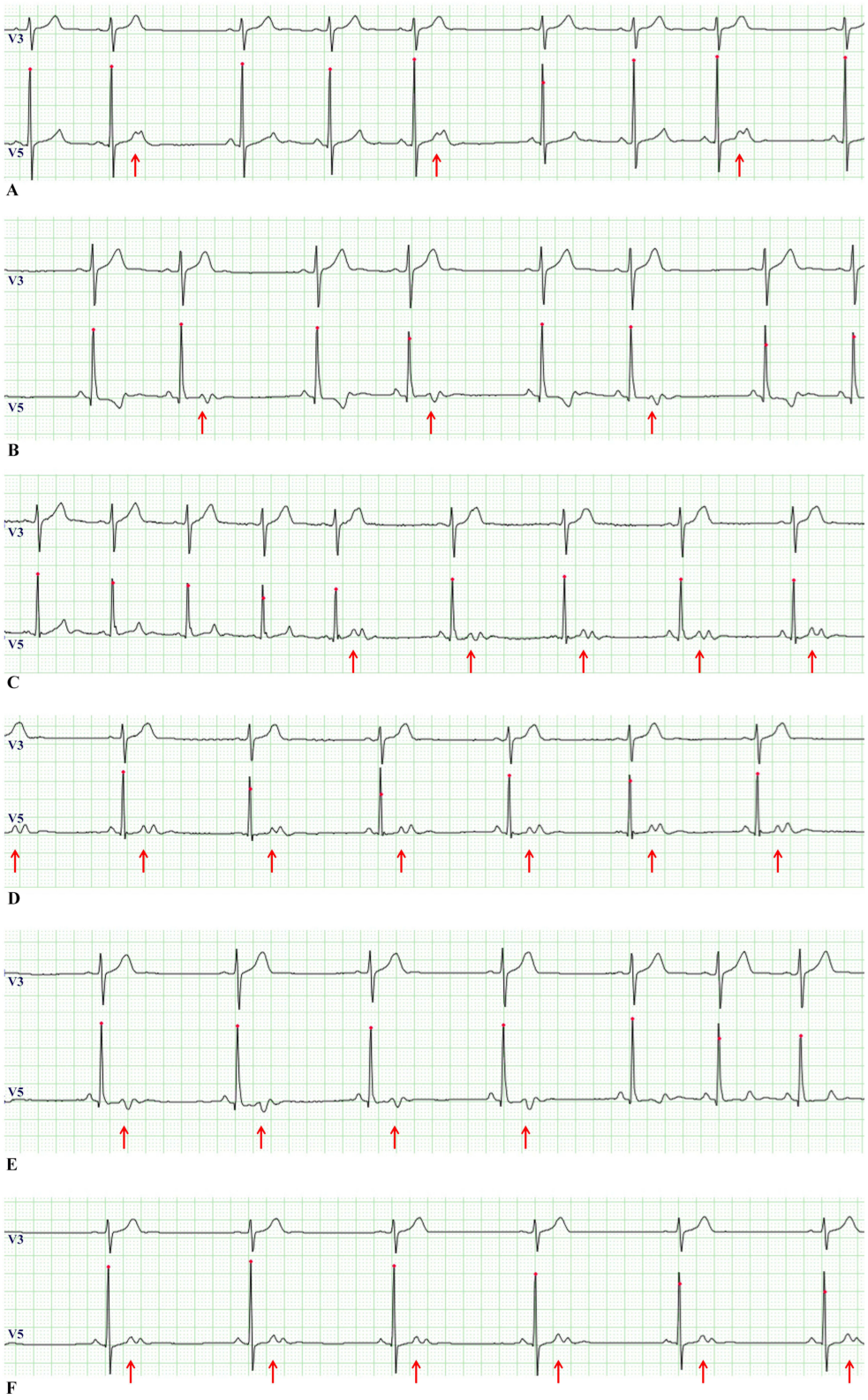

Figure 1 Blocked premature atrial contractions: blocked atrial quadrigeminy (**A**) blocked atrial trigeminy (**B**) initiation of blocked atrial bigeminy (**C**) blocked atrial bigeminy (**D**) termination of blocked atrial bigeminy (**E**) minimum heart rate associated with blocked atrial bigeminy (**F**). P waves of non-conducted PACs are arrowed.

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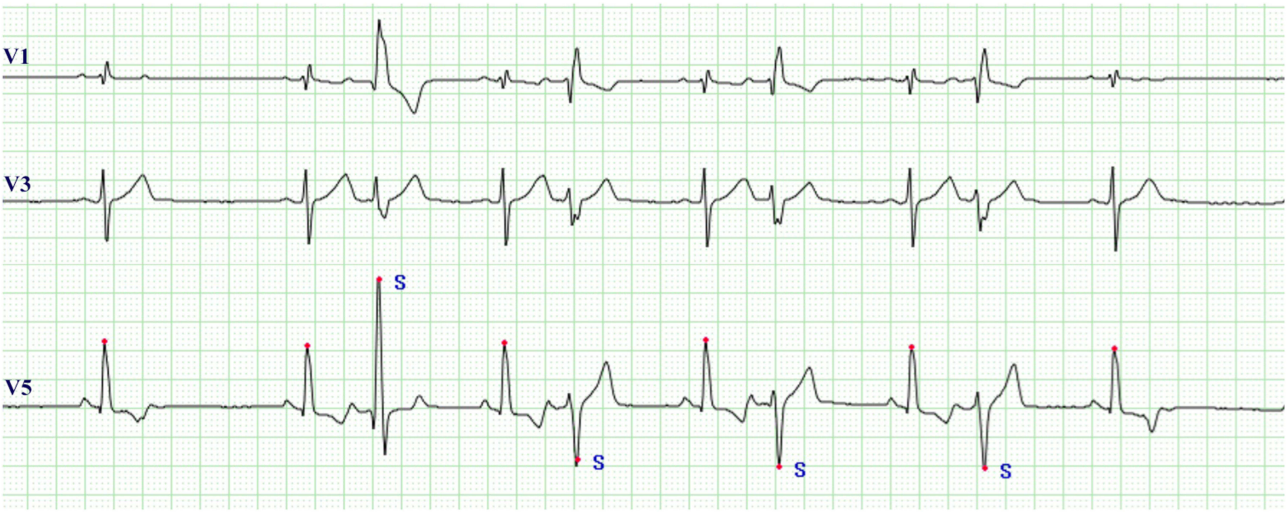

Figure 2 Atrial bigeminy. Premature atrial contractions after the first and last (sixth) sinus beats are blocked. Premature atrial contractions with aberrancy (marked with letter "S") are observed after the second, third, fourth, and fifth sinus beats.

pressure and pulse, along with repeated conventional ECG and ambulatory ECG monitoring as well as echocardiography. For arterial hypertension, the patient was prescribed candesartan at a dose of 32 mg daily. Regular follow-up visits were scheduled.

Discussion

It may be difficult to distinguish blocked atrial bigeminy from sinus bradycardia due to the subtle similarities they exhibit in heart rhythm patterns. However, a sudden change in heart rate and the distinct morphology of the T wave, characterized by the superimposition of the P wave before and after this change, serve as key diagnostic features that can aid in accurate differentiation (Figures 1C and E). It is crucial not to refer patients exhibiting bradycardia associated with blocked PACs for permanent pacemaker implantation prematurely. Clinical evidence suggests that antiarrhythmic agents classified as class IC,[19] along with radiofrequency catheter ablation,[15,18,20] can provide symptomatic relief in cases of bradycardia resulting from frequent blocked PACs. However, in the specific instance presented herein, the patient was entirely asymptomatic, with the sole concern being a low pulse reading observed on an automated blood pressure monitor. However, the patient and his relatives were significantly disturbed by bradycardia. In this context, a thorough evaluation was conducted, which included ambulatory ECG monitoring, echocardiography, and the measurement of NT-proBNP concentrations, alongside conventional tests.

The administration of antiarrhythmic agents or the consideration of radiofrequency catheter ablation should only be entertained if the patient begins to experience symptoms that significantly impact their quality of life. Furthermore, regular follow-up with repeated ECGs, ongoing ambulatory ECG monitoring, and echocardiograms will be performed to identify any potential structural or functional changes within the heart. Additionally, measuring NT-proBNP concentrations is performed on follow-up visits.

Block in non-conducted PACs may be either supra-hisian[20] or infra-hisian.[21] In the present case, electrophysiological study was not performed; therefore, the level of block could not be established. The clinical importance of the level of block is uncertain, and blocked PACs in itself does not necessary reflect a disorder of the conduction system of the heart.[22]
The coupling interval (the interval between the previous sinus beat and PAC) predetermines one of three conditions:[23]

1. If PAC occurs very early after the previous QRS complex, it is blocked (non-conducted), as the AV-node, the bundle of His, or infra-hisian conduction tissues may still be in their refractory period. This early occurrence indicates that these critical structures have not yet recovered from the previous electrical impulse, thereby preventing the propagation of the new impulse to the ventricles.

2. Conversely, if the PAC occurs later after the previous QRS complex but still before one of the bundle branches of His (typically the right bundle branch) has exited its refractory period, aberrant conduction of the PAC is observed. This aberrancy results in a distinctive morphology of the QRS complex.
3. Finally, if the PAC appears significantly later, when the AV node, bundle of His, and infra-hisian structures have all exited their refractory periods, a normally conducted PAC is seen. In this scenario, the PAC is able to successfully propagate through the conduction system, resulting in a morphology similar to that of the normal sinus rhythm.

Blocked atrial bigeminy should be differentiated from sinus bradycardia, sinus exit block, type 2:1 second-grade atrioventricular block, escape junctional and ventricular rhythms. It's important that each condition belongs to "true" bradyarrhythmias and may require pacemaker implantation.

The morphology of the P wave in PACs usually differs from that of sinus beats, reflecting the distinct spreading electrical activity associated with these ectopic beats. The morphology of the P wave makes it possible to roughly determine the localization of the PAC origin.[24] The PR interval in PACs is usually normal or slightly prolonged (due to conduction via the slow pathway of the atrioventricular node); a short PR interval in PACs is rare and occurs when PAC originates from the area close to the atrioventricular node.[24] In such cases, the proximity allows for rapid conduction, resulting in a shorter interval between the P wave and the following QRS complex.

Although PACs were previously considered to be harmless, there is evidence that frequent PACs are strongly associated with atrial fibrillation.[2] Research indicates that frequent PACs not only heighten the risk of developing atrial fibrillation but are also linked to an increased risk of ischemic stroke, which can occur even in the absence of diagnosed atrial fibrillation. Also, stroke is found to be the first clinical manifestation rather than the development of atrial fibrillation itself.[25] A meta-analysis performed by Meng L. et al (2020) showed that patients with frequent PACs have a 2.2 times higher risk of atrial fibrillation, a 2.2 times higher incidence of stroke, and a 1.6 times higher all-cause mortality.[26] The frequency of PACs increases with age.[27] Additionally, there is two-fold greater number of PACs in patients with established cardiovascular disease.[27]

Intense atrial ectopy is sometimes called "excessive supraventricular ectopic activity" (ESVEA), although there is no consensus on a definition.[28] The most common definition of ESVEA is having ≥30 PACs per hour or any episode with run of ≥20 PACs.[28]

Nowadays, it's considered that the main cause of atrial fibrillation and frequent PACs is atrial cardiomyopathy. According to the international expert consensus, atrial cardiomyopathy is defined as any complex of structural, architectural, contractile or electrophysiological changes affecting the atria with the potential to produce clinically relevant manifestations.[29]

This condition is not merely a singular ailment but rather a multifaceted syndrome that encompasses a variety of alterations within the heart's atrial tissues. Atrial cardiomyopathy is associated with primarily cardiomyocyte changes, fibrotic changes, and primarily non-collagen deposits.[29] Plenty of conditions and diseases may contribute to the atrial cardiomyopathy, including aging, arterial hypertension, diabetes, obesity, heart failure, valvular heart disease, ischemia, myocarditis, systemic inflammation, certain medications, endocrine disorders, genetic factors, and obstructive sleep apnoea.[30]

The patient had arterial hypertension, a significant aspect that could potentially be attributed to atrial cardiomyopathy and lead to the frequent PACs. Suppression of supraventricular ectopic activity, whether through pharmacological intervention or ablation techniques, presents both significant benefits and disadvantages that must be carefully considered by the healthcare team as well as the patient. The benefits include: 1) Prevention of PAC-induced cardiomyopathy; 2) Prevention of atrial fibrillation and stroke; 3) Prevention of left atrial remodeling; 4) Reverse myocardial remodeling. The disadvantages for pharmacological treatment include: 1) Side effects of antiarrhythmic drugs; 2) The need for daily medication intake. The disadvantages for ablation include: 1) Risks related to the procedure; 2) Cost of the procedure.

The patient was actively engaged in the decision-making process regarding their treatment options and discussed the benefits and disadvantages thoroughly. After careful consideration, antiarrhythmic agents were prescribed, with a particular preference for the IC class of antiarrhythmic agents due to their safety for long-term use. However,

neither flecainide nor amiodarone demonstrated any significant effect on conventional ECGs. Furthermore, the use of beta-blockers and calcium channel blockers was avoided in this case, as these medications carry the risk of exacerbating bradycardia.[20] Considering the potential risks associated with catheter ablation, this option was not accepted. This decision was made after weighing the potential benefits against the risks involved, especially since the condition was deemed non-life-threatening. However, either pharmacological suppression of frequent PACs or the option of ablation could play a critical role in preventing the development of new-onset atrial fibrillation. Additionally, these interventions could help prevent PAC-induced cardiomyopathy and promote reverse remodeling of the atrial myocardium.

Due to the non-life-threatening nature of the condition and the absence of significant structural cardiac abnormalities, dual antiarrhythmic therapy was not used. This option would be used with caution as an alternative to ablation if bradycardia were symptomatic or if PAC-induced cardiomyopathy were diagnosed. Flecainide combined with amiodarone is sometimes used for refractory arrhythmias, as it may be effective when neither drug is effective as a single therapy. It may also allow a reduction in dosage and side effects.[31] Additionally, there is evidence supporting the use of sotalol with flecainide.[32,33]

NT-proBNP concentration was measured to rule out chronic heart failure due to PAC-associated cardiomyopathy. However, NT-proBNP level is an important predictor of new-onset atrial fibrillation.[34] Elevated NT-proBNP levels are associated with a 3.8 times higher risk of the development of atrial fibrillation.[35] Every 1000 pg/mL increase in NT-proBNP is associated with a 16% increase in the risk of atrial fibrillation occurrence.[36] Furthermore, each doubling of NT-proBNP increases the risk of incident atrial fibrillation by 46%.[37]

It is very important to recognize the true cause of bradycardia. For some individuals, bradycardia may simply be a physiological phenomenon, such as during sleep when the body is in a state of rest and recovery, or in well-trained athletes. However, bradycardia can be also signal serious underlying pathological processes. Conditions such as sick sinus syndrome, coronary artery disease, myocarditis, and cardiomyopathy can all lead to bradycardia. Furthermore, bradycardia can arise as a result of increased intracranial pressure, or can be related to endocrine disorders like hypothyroidism. Additionally, environmental factors like hypothermia can lead to bradycardia, as can imbalances in electrolytes, such as hyperkalemia and hypocalcemia. Moreover, it is essential to consider the potential influence of medications on heart rate, as certain drugs can induce bradycardia as a side effect. Interestingly, not all cases of bradycardia come with overt clinical symptoms; some individuals may exhibit bradycardia without any noticeable symptoms, while others may experience symptoms such as dizziness, fatigue, or even fainting, which can be predictors of more severe consequences.

Conclusion

Blocked atrial bigeminy is a rare cause of bradycardia with regular ventricular rhythm. Recognition of the P wave superimposed on the ST segment or the T wave may be a clue for a correct diagnosis. Physicians should be aware of this condition to avoid unnecessary pacemaker implantation.

Abbreviations

ECG, electrocardiogram; ESVEA, excessive supraventricular ectopic activity; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PAC, premature atrial contraction.

Patient Consent and Institution Approval

The written consent for this case publication was obtained from the patient. The institutional approval for the case report is not required according to local guidelines.

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Disclosure

The authors report no conflicts of interest in this work.

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