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CASE REPORT
Arrhythmias/electrophysiology

Coughing as a potentially effective induction method of atrial tachycardia: a case report

Reina Tonegawa-Kuji , Kenichiro Yamagata *, and Kengo Kusano

Division of Arrhythmia and Electrophysiology, Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center, 6-1, Kishibe-Shimmachi, Suita, 564-8565 Osaka, Japan

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Background Cough-induced atrial tachycardia (AT) is extremely rare and its electrical origin remains largely unknown. Atrial tachycardias triggered by pharyngeal stimulation, such as swallowing or speech, appears to be more common and the majority of them originate from the superior vena cava or right superior pulmonary vein (PV). Only one case of swallow-triggered AT with right inferior pulmonary vein (RIPV) origin has been reported to date.

Case summary We present a case of a 41-year-old man with recurring episodes of AT in the daytime. He underwent electrophysiology study without sedation. Atrial tachycardia was not observed when the patient entered the examination room and could not be induced with conventional induction procedures. By having the patient cough periodically on purpose, transient AT with P-wave morphology similar to the clinical AT was consistently induced. Activation mapping of the AT revealed a centrifugal pattern with the earliest activity localized inside the RIPV. After successful radiofrequency isolation of the right PV, AT was no longer inducible.

Discussion In the rare case of cough-induced AT originating from the RIPV, the proximity of the inferior right ganglionated plexi (GP) suggests the role of GP in triggering tachycardia. This is the first report that demonstrates voluntary cough was used to induce AT. In such cases that induction of AT is difficult using conventional methods, having the patient cough may be an effective induction method that is easy to attempt.

Keywords Atrial tachycardia • Cough-induced tachycardia • Vagal-mediated arrhythmia • Ablation • Case report

Learning points

- Coughing induces atrial tachycardia (AT) via vagal-mediated mechanism in rare cases.
- It is important to recognize many options to induce atrial arrhythmias for successful mapping and ablation of the arrhythmia.
- In such cases that induction of AT is difficult using conventional methods, having the patient cough may be an effective induction method that is easy to attempt.

Introduction

Cough-induced atrial tachycardia (AT) is rare. Our literature review identified only four cases of cough-triggered atrial tachyarrhythmia that the patients were aware of cough as the trigger, but none of the reports examined the ectopic focus.[1,2] Atrial tachycardias triggered by pharyngeal stimulation appear to be more common, and have been reported in about 50 cases of swallowing and <10 cases of vocalization-triggered AT. Only one case of swallow-triggered AT with right inferior pulmonary vein (RIPV) origin has been reported to date, while the majority of the swallow or speech-induced AT cases originated from the superior vena cava or right superior pulmonary

* Corresponding author. Tel: +81-6-6170-1070, Email: look.cardiology@gmail.com
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vein (PV), and a few cases from the left superior PV and the right atrium.[3–6]

Timeline

6 months prior to presentation
- Patient reported daytime palpitations with no obvious triggering episodes.

At presentation
- The 24-h Holter monitoring revealed paroxysmal atrial tachycardia (AT) that lasted for up to 90 s.

Electrophysiology study and radiofrequency ablation (4 h)
- Atrial tachycardia was not induced by conventional induction procedures but by having the patient cough.
- Activation mapping was performed while coughing, revealing the ectopic focus of the AT in the right inferior pulmonary vein (PV).
- After successful electrical isolation of the right PV, AT was no longer inducible.

Follow-up at 6 months
- The patient remained asymptomatic without any arrhythmias.

Case presentation

A 41-year-old male with no significant medical history presented with recurring palpitations. This was associated with slight chest tightness occurring several times a day. He did not notice any triggering episodes. His physical examination, laboratory studies, and echocardiography results were normal. Premature atrial contractions (PACs) were frequently seen on electrocardiogram (*Figure 1*). The P-wave morphology of the PAC on 12-lead electrocardiogram was positive in V1 and not bifid in V1 or II, and sinus rhythm P-wave morphology was ± in V1, indicating that the PAC originated from the right PV.[7] The 24-h Holter monitoring showed that 14 250 out of 112 000 heart beats (12.7%) were extrasystolic, and paroxysmal narrow complex tachycardia at a rate of 200 b.p.m. lasted for up to 90 s. This was associated with his symptoms and occurred predominantly during the daytime, suggestive of AT. Aprindine and pilsicainide therapies did not affect the frequency of palpitations. Dabigatran, 300 mg bid, was started in anticipation of possible left atrial access during electrophysiology study (EPS) and radiofrequency ablation.

He underwent EPS without sedation, as most of the AT episodes were documented during the daytime. Premature atrial contraction was not observed when the patient entered the examination room. Heparin (14 000 units) was administered to maintain the activated clotting time above 300 s during the procedure. Tachycardia could not be induced with conventional induction approaches: 2–6 μg of isoproterenol being injected seven times, atrial burst pacing (cycle

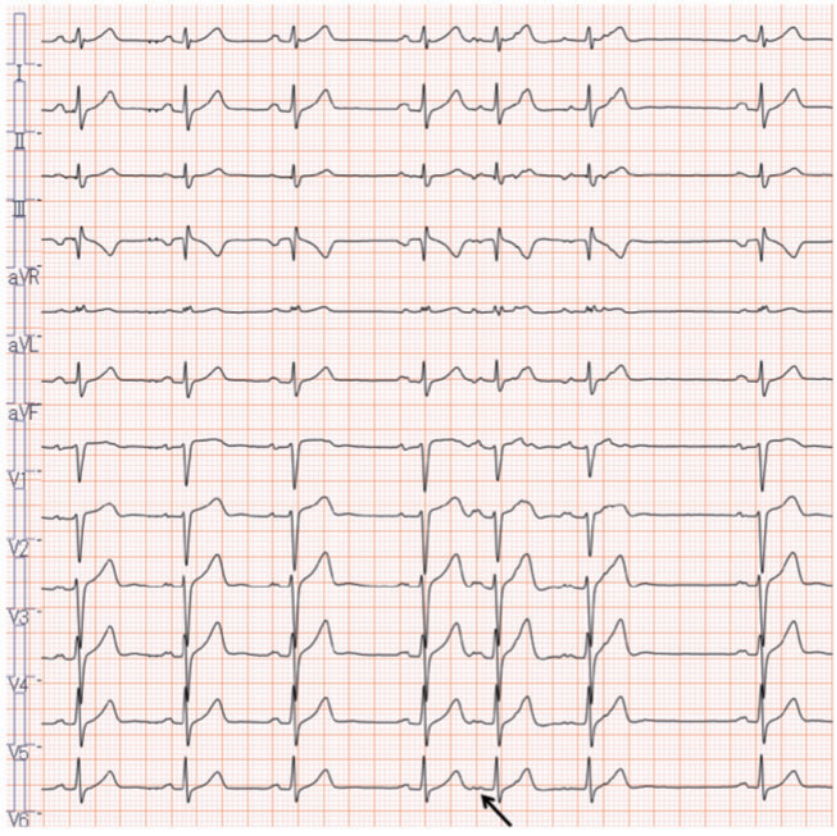

Figure 1 A 12-lead rhythm strip of the patient with a premature atrial contraction (arrow).

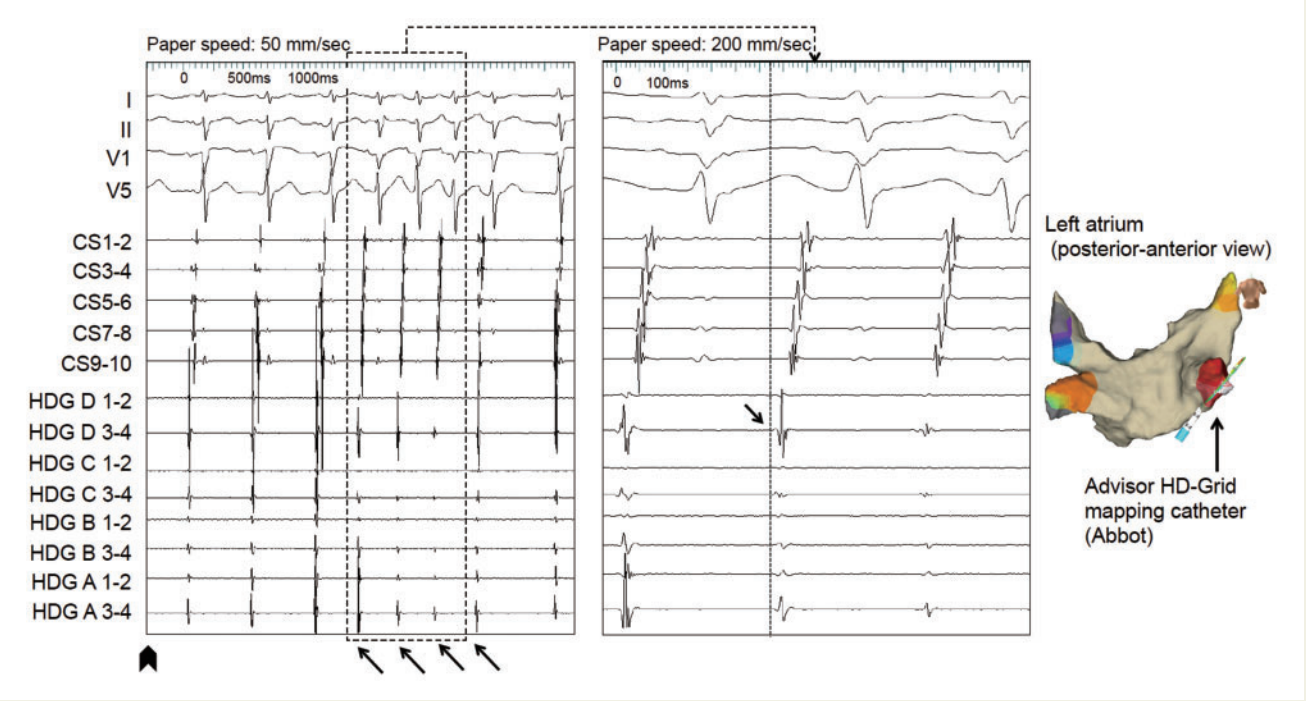

Figure 2 Surface and intracardiac electrograms of cough-induced arrhythmia. Top four leads (I, II and V1, V5) reflect surface electrogram tracings. A–D lines reflect intracardiac electrograms acquired using the Advisor HD-Grid Mapping Catheter (Abbot, USA) located in the RIPV. CS1-10 reflects intracardiac electrograms originating from the coronary sinus. The patient is in normal sinus rhythm until intentionally coughing (arrowhead), followed by four beats of non-sustained atrial tachycardia (bottom arrows). The first three beats of atrial tachycardia are shown in slower paper speed in the middle panel. The earliest activation is documented in proximal D-line of HD-grid located in the RIPV (arrow). Left atrium 3D activation map of the atrial tachycardia and the location of the Advisor HD-Grid mapping catheter at the timing of the intracardiac electrograms shown in the figure is shown in the right panel. AT, atrial tachycardia; CS, coronary sinus; RIPV, right inferior pulmonary vein.

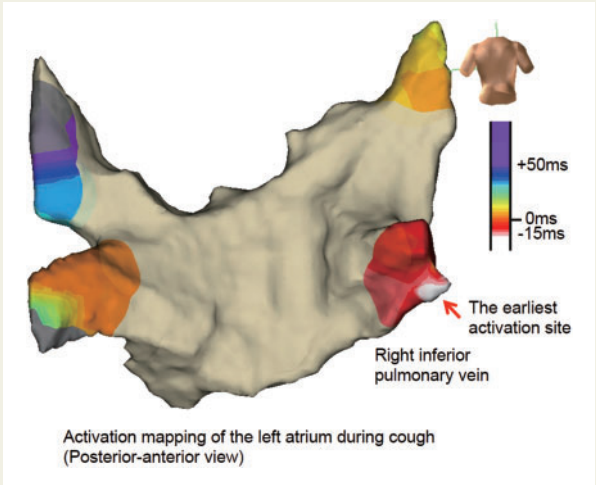

Figure 3 Left atrium 3D activation map of the atrial tachycardia.

length 200–400 ms) or atrial single extra stimulation up to effective refractory period being attempted multiple times following isoproterenol injections, and 40 mg of adenosine triphosphate disodium hydrate being injected twice. Furthermore, rare but reported induction methods that we routinely attempt in PAC induction, such as holding the breath, handgrip, speech, or swallowing, failed to induce clinical tachycardia. Having the patient cough periodically was the only way to consistently induce transient AT with P-wave morphology similar to the clinical AT (*Figure 2*). EnSite Precision™ three-dimensional cardiac mapping system (Abbott, Saint Paul, MN, USA) was used with decapolar catheter (Abott, Saint Paul, MN, USA) placed in the coronary sinus and Advisor HD-Grid mapping catheter (Abott, Saint Paul, MN, USA) for mapping. Right atrial mapping revealed the earliest activity of AT to be localized in the septum, and by approaching the left atrium by transseptal puncture, the activation map of the PAC revealed a centrifugal pattern with the earliest activity localized to the RIPV (*Figure3*). In order to avoid the risk of PV stenosis by ablating inside the vein, we performed right PV isolation. Radiofrequency currents were delivered for 30 s at a power of 30 W with a 4-mm irrigated-tip Tacticath Sensor Enabled contact force ablation catheter (Abott, Saint Paul, MN, USA) through an Agilis sheath (Abott, Saint Paul, MN, USA). After electrical isolation of the right PV, tachycardia did not recur despite repeated coughing as well as electrical stimulation or isoproterenol infusion. The total procedure time was 225 min with a left atrial dwell time of 61 min. The patient was discharged without any antiarrhythmic drugs including beta-blockers or calcium-blockers. At 6 months of follow-up, the patient remained asymptomatic without any palpitations, and dabigatran was

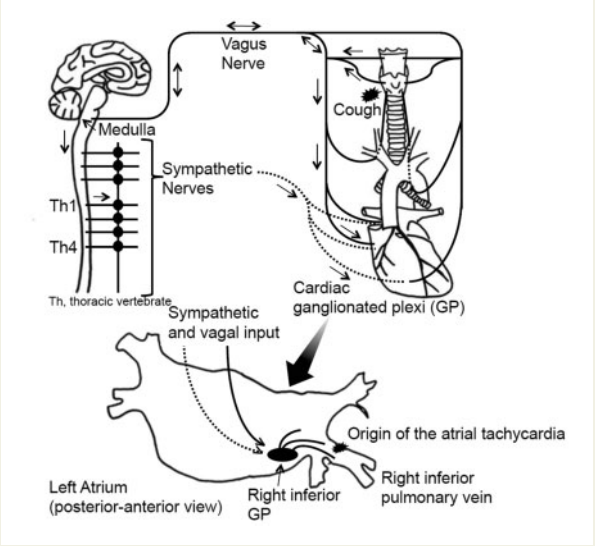

Figure 4 A schematic illustration of the anatomical relationship between the pharyngolaryngeal system, vagus nerve, and cardiac autonomic nervous system.

discontinued after observing no AT and only few PACs [25 out of 117 383 heart beats (0.02%)] during 24-h Holter monitoring.

Discussion

We present a case of focal AT originating from the RIPV that could only be induced by having the patient cough during EPS. To our knowledge, this is the first report that voluntary cough was used to induce AT. Identifying this highly reproducible trigger was critical for successful mapping and ablation of the arrhythmia.

Cough-induced atrial tachyarrhythmia is rare.[1,2] Atrial tachycardias triggered by pharyngeal stimulation appears to be more common, however, only one case of swallow-triggered AT with RIPV origin has been reported to date.[3] Therefore, our patient with AT induced by cough with RIPV as the focus presents an extremely rare manifestation.

We speculated that the induction of AT in this patient involved vagal-mediated response of cardiac ganglionated plexi (GP) provoked by cough, which were supported by the following observations. Coughing may directly stimulate pharyngolaryngeal vagal receptors and subsequently vagal afferent sensory nerves.[8] Activated afferent vagal nerves transmit signal to autonomic centres including nucleus tractus solitarius, which in turn sends vagal efferent output to the cardiac GP. Inferoposterior root of the RIPV has been reported to be a major location of left atrial autonomic GP, known as inferior right GP, extending epicardial nerves to the RIPV.[9,10] The possible role of the GP in initiating AT in this case is supported by the consistent location of the inferior right GP and extending nerves with the site of ectopy. The cooperative activation of the vagal and adrenergic elements has been shown to induce triggered activity and subsequent atrial arrhythmogenic foci by the abbreviation of the local action potential duration and increased intracellular calcium transient currents via the GPs, particularly in vagally mediated ones (*Figure 4*).[11,12] In our patient, the time lapse from the coughing to the onset of AT was as short as 2 s, making this mechanism a reasonable explanation. Ablation of the inferior right GP at the time of right PV isolation led to the resolution of symptoms in this patient.

With the recent advancement of multipolar mapping catheter such as the Adviser HD-Grid Mapping Catheter used in this case, it is possible to take multiple simultaneous activation points from a single atrial potential. Still, it is important to recognize many options to induce AT or PACs to attain the precise activation map to identify the precise origin by reproducible method. Our report demonstrated that cough was an effective option in inducing atrial arrhythmia.

Conclusion

Cough-induced tachyarrhythmia is extremely rare. In the case of RIPV ectopy, the proximity of the inferior right GP suggests the role of GP in triggering tachycardia. In such cases that induction of AT is difficult using conventional methods, having the patient cough may be an effective induction method that is easy to attempt.

Lead author biography

Dr Reina Tonegawa-Kuji is a cardiologist working in the National Cerebral and Cardiovascular Center in Suita, Japan. She acquired her medical degree at Nagoya University, Japan and completed training in cardiology in Saitama, Japan. Her principal field of interest is electrophysiology, arrhythmias, ablation, and cardiovascular implantable electric devices.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report, including images and associated text, has been obtained from the patient in line with COPE guidance.

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