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Dysfunctional Hypoglossal Nerve Stimulator After Electrical Cardioversion: A Case Series

Adam P. Vasconcellos, MD; Colin T. Huntley, MD; Amy E. Schell, MD; Ryan J. Soose, MD; Maurits S. Boon, MD

Objectives/Hypothesis: Upper airway stimulation has demonstrated marked improvements in apnea-hypopnea index, oxygen desaturation index, and quality-of-life measures in patients with moderate to severe obstructive sleep apnea (OSA) who cannot tolerate continuous positive airway pressure. Cardiac arrhythmias are common in patients with OSA and can require electrical cardioversion. We describe the first four reported cases of hypoglossal nerve stimulator (HGNs) dysfunction after electrical cardioversion and illustrate our operative approach to device troubleshooting and repair.

Study Design: Retrospective case series.

Methods: A retrospective review of 201 HGN implantations performed at two academic institutions revealed four cases of HGN device dysfunction after electrical cardioversion requiring surgical revision. Preoperative and postoperative device performance metrics and electrical cardioversion specifications were retrospectively assessed and compiled for this case series. The senior authors (R.J.S., M.S.B.) detail operative planning and approach for HGN implantable pulse generator (IPG) replacement.

Results: At least two patients with HGNs device dysfunction had received cardioversion via anterolateral electrode pad placement. Three patients had received multiple shocks. All four patients experienced a change in device functionality or complete cessation of functionality after electrocardioversion. Operatively, each patient required replacement of the IPG, with subsequent intraoperative interrogation revealing proper device functionality.

Conclusion: Counseling for patients with HGNs undergoing external electrical cardioversion should include possible device damage and need for operative replacement. Anteroposterior electrode pad placement should be considered for patients with HGNs who require electrocardioversion. Operative replacement of an HGN system damaged by electrocardioversion begins with IPG replacement and intraoperative device interrogation.

Key Words: Hypoglossal nerve stimulator, upper airway stimulation, obstructive sleep apnea.

Level of Evidence: 4

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INTRODUCTION

Upper airway stimulation (UAS) has demonstrated marked improvements in apnea-hypopnea index (AHI), oxygen desaturation index, and quality-of-life measures in patients with moderate-to-severe obstructive sleep apnea (OSA) who are unable to tolerate continuous positive airway pressure (CPAP).1–4 There have been few documented procedure or device-related adverse events. These have included tongue soreness, discomfort with stimulation, and device migration.1,2

Many patients with OSA have cardiovascular comorbidities, including a variety of cardiac arrhythmias that can be treated medically but often require electrical cardioversion. Previous reports indicate that external electrical cardioversion risks damaging an indwelling cardiac pacemaker.7–9 However, there has been no documentation of risk of cardioversion to hypoglossal nerve stimulator (HGNs) hardware. We present the first four documented cases of external electrocardioversion resulting in dysfunction of implanted HGNs.

MATERIALS AND METHODS

The senior authors (R.J.S., M.S.B.) of this case series have performed a total of 201 HGN implants. HGN databases at both institutions were queried and revealed a total of four cases in which HGN devices were damaged as a result of electrical cardioversion and required surgical revision. Clinical indications for HGNs; preoperative, postoperative, and postrevision AHI or respiratory event index (REI); device generation (3024 or 3028); and electrical cardioversion shock type, number, and positioning were retrospectively assessed and compiled for this case series. The senior authors detail operative planning and approach for HGNs IPG replacement as performed in these four cases.
RESULTS

Patient 1

A 65-year-old male presented with severe OSA with AHI 53. The patient described persistent atrial fibrillation (Afib) despite prior cardioversion. The patient could not tolerate CPAP and met clinical indications for HGNS, including drug-induced sleep endoscopy findings. The patient underwent implantation of HGNS and had successful postoperative titration of the device with AHI 13. Seven months later, routine electrical cardioversion was performed for Afib. Two biphasic shocks were performed, first at 120 joules and subsequently at 150 joules. Electrode pad positioning was not reported. That evening, the patient reported an inability to activate HGNS, and device interrogation showed dysfunction of the internal pulse generator (IPG). Operative exploration via the patient’s chest wall incision revealed intact leads and a damaged IPG, which was replaced. After replacement, device function was evaluated and the HGNS was programmed to precardioversion parameters. Postoperative 2-night unattended home polysomnography yielded REI of 6.6 and 11.3.

Patient 2

A 72-year-old male presented with moderate OSA with an AHI of 20. The patient similarly described persistent Afib despite prior cardioversion. The patient could not tolerate CPAP and met clinical indications for HGNS, including drug-induced sleep endoscopy findings. The patient underwent implantation of HGNS and had successful postoperative titration of the device with AHI 0. Four months later, routine electrical cardioversion was performed for Afib. The patient received a single synchronized biphasic 150-joule shock delivered to the heart using adhesive pads positioned in the anteroposterior orientation (see Fig. 1),10 which restored sinus rhythm. HGNS was interrogated postprocedure and was found to be working appropriately. The patient subsequently received placement of an implantable subcutaneous loop recorder. Repeat electrocardioversion was performed 1 year later for recurrent Afib, consisting of a biphasic 150-joule shock in the anteroposterior orientation. Postprocedure interrogation of HGNS revealed that the device had been reset to 0 stimulation but was reprogrammable. Three months later, however, cardioversion was again performed, this time a 200-joule shock in the anterolateral orientation (see Fig. 1).10 HGNS device interrogation subsequently revealed dysfunction of the IPG. Operative exploration via the patient’s chest wall incision revealed intact leads and a damaged IPG, which was replaced. After replacement, device function was evaluated and the HGNS was programmed to precardioversion parameters. Postoperative device titration via in-house polysomnography yielded an AHI 1.3.

Patient 3

A 70-year-old female presented with severe OSA with AHI 39. The patient’s sleep apnea was similarly...
diagnosed in the context of Afib, for which the patient had received prior cardioversions. The patient could not tolerate CPAP and met clinical indications for HGNS, including drug-induced sleep endoscopy findings. The patient underwent left-sided implantation of HGNS because the patient had right-sided silicone breast implant from prior reconstruction after mastectomy. The patient had successful postoperative titration of the device with AHI 11. Five months later, the patient underwent elective electrical cardioversion for Afib. The patient received a synchronized biphasic 150-joule shock with pads positioned in the anterolateral orientation, followed by a second biphasic shock at 250 joules. The patient subsequently reported an inability to activate HGNS, and device interrogation showed dysfunction of the IPG. Operative exploration via the patient's chest wall incision revealed intact leads and a damaged IPG, which was replaced. After replacement, device function was evaluated and the HGNS was programmed to precardioversion parameters. Postoperative device titration has yet to be performed for this patient.

**DISCUSSION**

OSA is present in an estimated 40% to 50% of patients with Afib. Along with other comorbid conditions such as hypertension and valvular heart disease, OSA has been suggested to both potentiate Afib and foster its progression from paroxysmal Afib to chronic Afib. Furthermore, patients with untreated OSA are more likely to have recurrence of Afib after cardioversion and cardiac ablation.11,12 Treatment of concurrent OSA is thus essential for patients with Afib.

UAS is a modern surgical approach to moderate-to-severe OSA for patients who cannot tolerate CPAP.1,13 With surgical success defined as AHI reduction > 50% and AHI ≤ 20, reported success rates for UAS have ranged from 68% to 96%.14–16

<table>
<thead>
<tr>
<th>Patient</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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<tr>
<td>Preop AHI</td>
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<td>20</td>
<td>39</td>
<td>24.5</td>
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<tr>
<td>Postop AHI</td>
<td>13</td>
<td>1.3</td>
<td>11.3</td>
<td>2</td>
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<td>Generation of original IPG</td>
<td>3024</td>
<td>3024</td>
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<td>3024</td>
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<tr>
<td>Generation of re-implanted IPG</td>
<td>3024</td>
<td>3028</td>
<td>3028</td>
<td>3028</td>
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<tr>
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<td>Left</td>
<td>Right</td>
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<td>Implantation to device failure (months)</td>
<td>7</td>
<td>16</td>
<td>5</td>
<td>10</td>
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Afib = atrial fibrillation; AHI = Apnea-Hypopnea Index; IPG = internal pulse generator.
The authors of this case series have performed a total of 201 HGNS implants among two institutions and report four cases of HGNS dysfunction after external electrocardioversion for Afib (see Table I). In at least two cases, dysfunction of the device was noted after anterolateral placement of electrode pads with delivery of biphasic charges. Three patients received multiple shocks. Three patients had a history of electrocardioversion prior to HGNS device implantation, and all experienced a change in device functionality or complete cessation of functionality with attempts to turn on their respective devices after electrocardioversion.

External electrical cardioversion has previously been demonstrated to pose risk to indwelling cardiac pacemakers. Electrode pad positioning may influence risk of damage to these implantable devices. Anteroposterior pad orientation (see Fig. 1) in which one pad is placed anteriorly over the left precordium and one is placed posteriorly on the upper back may be considered relatively device-protective. This is in contrast to anterolateral pad orientation (see Fig. 2), which positions one pad in the upper right chest wall and a second pad in the lower left chest wall and may direct current through the implantable device. Our case series describes two episodes of HGNS dysfunction after anterolateral electrode pad placement. Regarding electrode pad placement, similar precautions to those provided to patients with other implantable medical devices must be considered for patients with HGNS, and patients should be informed of the risk of possible device damage with external electrocardioversion.

For each case detailed above, HGNS is first formally interrogated in the office and deemed nonfunctional. Once a diagnosis of a nonfunctional device is made, the next step is surgical replacement of the IPG. A preoperative chest X-ray is performed to assess lead placement. The surgical approach begins with incision at the chest wall site of prior IPG implantation. Particular caution is taken in approaching the IPG to ensure that leads are not sitting superficial in the pocket because damage to any of the leads would necessitate complete replacement. One of the senior authors (R.J.S.) advises approaching the IPG via the lateral aspect of the incision to minimize risk of damage to the leads. Dissection is carried through the subcutaneous tissues, and a dense fibrotic capsule is entered that encompasses the IPG. The IPG is disconnected from the respiratory sensor and the stimulation lead and subsequently is removed (see Fig. 2). A new IPG is connected to the respiratory sensor and stimulation lead (see Fig. 3), placed back into the fibrous pocket, and secured with suture. Device interrogation is performed. In each case described in this article, replacement of the IPG alone resulted in excellent tongue protrusion and respiratory sensing. However, if persistent device failure had been noted after replacement of IPG, subsequent exploration of the respiratory sensor and stimulation leads would be necessary via lateral chest wall and cervical incisions, respectively. Due to the dense fibrosis that has been anecdotally noted by the senior author (M.S.B.) with the stimulation lead in revision procedures, consideration would be given to approaching the contralateral neck and placing a new stimulation lead, which could then be tunneled to the new IPG.

In three cases, the patient had a 3024 generation IPG exchanged for a newer 3028 generation IPG. The newer generation IPG is 40% smaller and 18% thinner than the previous generation IPG, and magnetic resonance imaging is conditional to allow imaging of the brain and extremities. There is also report from the manufacturer that the newer generation device may be more resistant to damage from cardioversion. Further evaluation is necessary to determine whether the IPG type would influence dysfunction after electrocardioversion.

The manufacturer of the HGNS system’s guidelines are that pharmaceutical cardioversion be performed whenever possible to avoid risk of damage to the HGNS system. When electrical cardioversion is necessary, biphasic waveform is preferred over monophasic waveform, and total energy delivered should be minimized if possible. Paddles should be placed as far from device components as possible, and the HGNS device should be tested after electrocardioversion to ensure continued functionality.

CONCLUSION
The possibility of HGNS device damage from cardioversion should be included in preoperative counseling prior to device implantation, particularly in patients with known Afib. Patients with HGNS undergoing external electrical cardioversion should be counseled regarding possible device damage and need for operative replacement. Anteroposterior electrode pad placement should be considered for patients with HGNS who require electocardoversion. Operative replacement of HGNS system damaged by electrocardioversion begins with IPG replacement and intraoperative device interrogation.

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