Thoracoscopic patch insulation to correct phrenic nerve stimulation secondary to cardiac resynchronization therapy

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Aims
Cardiac resynchronization therapy is an established therapy for heart failure, improving quality of life and prognosis. Despite advances in technique, available leads and delivery systems, trans-venous left ventricular (LV) lead positioning remains dependent on the patient’s underlying venous anatomy. The left phrenic nerve courses over the surface of the pericardium laterally and may be stimulated by the LV pacing lead, causing uncomfortable diaphragmatic twitch. This paper describes a video-assisted thoracoscopic (VATS) procedure to correct phrenic nerve stimulation secondary to cardiac resynchronization therapy.

Methods and results
Most current ways of avoiding phrenic stimulation involve either electronic reprogramming to distance the phrenic nerve from the stimulation circuit or repositioning the lead. We describe a case where the phrenic nerve was surgically insulated from the stimulating current by insinuating a patch of bovine pericardium between the epicardium and native pericardium of the heart thus completely resolving previously intolerable and incessant diaphragmatic twitch. The procedure was performed under general anaesthesia with single-lung ventilation and minimal use of neuromuscular blocking agents. Surgical patch insulation of the phrenic nerve was performed using minimally invasive VATS surgery, as a short-stay procedure, with no complications. No diaphragmatic twitch occurred post-surgery and the patient continued to gain symptomatic benefit from cardiac synchronization therapy (New York Heart Association Class III to II), enabling return to work.

Conclusions
In cases where the trans-venous position of a LV lead is limited by troublesome phrenic nerve stimulation, thoracoscopic surgical patch insulation of the phrenic nerve could be considered to allow beneficial cardiac resynchronization therapy.

Keywords
Cardiac resynchronization therapy • Phrenic stimulation • Twitch • Phrenic nerve insulation • Thoracoscopic surgery

Introduction
The course of the left phrenic nerve on the pericardium is typically in close relation to the great cardiac vein and forms variable relations with the lateral vein, posterior branches of the anterolateral vein, and anterior branches of posterolateral cardiac vein.¹ These anatomical considerations result in a high probability of conflict between optimal site for left ventricular (LV) stimulation and intolerable phrenic nerve stimulation. Phrenic nerve stimulation causes uncomfortable diaphragmatic twitch for the patient, resulting in loss of cardiac resynchronization therapy (CRT) in 1–2% of patients.² We report a case of successful CRT lead placement complicated by phrenic nerve stimulation. Existing approaches to resolve the twitch are discussed, and a minimally invasive thoracoscopic method of surgical patch insulation of the phrenic nerve is described.
**Patient and history**

A 38-year-old woman with congenital complete heart block underwent initial permanent pacemaker implantation at the age of 22. The passive right ventricular lead was apically positioned and she was 100% ventricularly paced.

The patient presented with progressive dyspnoea and tiredness on exertion; echocardiography revealed deteriorating LV systolic function with LV ejection fraction of 24%. In the absence of any other identifiable cause for poor LV function, systolic dysfunction was attributed to long-term persistent right ventricular apical pacing. Despite optimal medical treatment, her symptoms and LV systolic function continued to deteriorate [New York Heart Association (NYHA) functional Class III] and when pacemaker generator replacement was indicated, an upgrade to CRT was planned.

**Cardiac resynchronization therapy**

A venogram confirmed a patent subclavian venous system and access was achieved via a fluoroscopically guided axillary vein puncture. A Medtronic MB2 catheter was used to access the coronary sinus and a venogram performed using a balloon-occlusion catheter. Only two potential branches of the coronary sinus were identified for LV lead placement; a posterolateral branch and an anterolateral branch. The posterolateral branch was explored initially because it was anatomically the most attractive option which would provide pacing of the posterolateral, non-apical area of LV myocardium. A problem of lead stability was encountered given the large calibre of the vein. Both a Boston Scientific bipolar passive-fixation Easytrak 3 lead (6F) and a Medtronic passive-fixation bipolar 4194 lead (6F) were advanced as far as possible into the terminal branch, but both displaced on slitting the catheter (Figure 1).

The anterolateral branch was smaller and a Medtronic 4196 passive-fixation dual cathode lead (4F) was advanced as far as possible. This was a suboptimal anatomical position, and was also unstable (Figure 1). Finally, a Medtronic 4195 active-fixation unipolar LV lead with deployable lobes was therefore positioned in the posterolateral branch, achieving acceptable parameters (threshold 1.2 V at 0.4 ms, impedance 1258 Ω, sensing 22 mV) and stability (Figure 1). There was no diaphragmatic twitch at 10 V, either

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**Figure 1** (A) Boston Scientific bipolar passive-fixation Easytrak 3 lead in the posterolateral branch of the coronary sinus. (B) A Medtronic passive-fixation bipolar 4194 lead in the terminal branch of the posterolateral vein. (C) A Medtronic 4196 passive-fixation dual cathode lead positioned in the anterolateral branch of the coronary sinus. (D) A Medtronic 4195 active-fixation unipolar left ventricular lead with deployable lobes positioned in the posterolateral branch of the coronary sinus.
palpably or fluoroscopically. This stable position was maintained throughout sitting and removal of the sheaths and indeed, thereafter.

Phrenic stimulation

Intolerable diaphragmatic twitch occurred post-operatively when the patient sat up. The Medtronic 4195 being a unipolar lead offered limited recourse to electronic repositioning, to no avail and the LV lead was turned off.

Options considered to counteract phrenic stimulation

Phrenic stimulation is often not detected at implantation because it occurs only in the left lateral or sitting position in a significant number of patients. Electronic repositioning is the least invasive option and usually the first intervention attempted when diaphragmatic twitch is encountered. This was unsuccessful in our case due to limited options. A further attempt at LV lead reposition was not considered due to anatomical limitations.

His-bundle pacing has been reported as providing a more physiological pacing site, resulting in a narrower QRS. This was not felt to be satisfactory for a patient with significant, symptomatic LV impairment, and may also result in more frequent generator replacement requirements due to the higher thresholds necessary for true His-bundle pacing. Endocardial LV lead placement has been reported but would require life-long anticoagulation due to the high risk of systemic thromboembolism. This was not an attractive option for a young woman.

Epicardial placement of the LV lead may also be considered in patients suitable for CRT but with unsuitable venous anatomy, with some authors reporting excellent results. Others have reported a higher incidence of lead failure compared with endocardial pacing and thresholds are usually higher resulting in frequent generator replacements. In addition, most epicardial leads are placed anteriorly due to the difficulty of lateral exposure during surgery. The position of the LV lead may thus be limited, especially if a minimally invasive approach is utilized and thus optimal CRT is less guaranteed compared with LV pacing via a non-apical posterolateral branch of the coronary sinus. For these reasons, alternative ways to eradicate diaphragmatic twitch while maintaining the otherwise optimal existing lead position were considered.

A bipolar or quadripolar lead would allow electronic repositioning with the potential to avoid phrenic stimulation, if sufficient lead stability could be achieved. Currently, the only commercially available quadripolar lead is device specific, suitable only for the St Jude Medical QUADRA device (high voltage, CRT-D). While this may reduce the incidence of phrenic stimulation, the use of a high-voltage device in this patient was not felt to be warranted. At the present time adaptors for use in CRT-P devices are not available.

Surgical insulation of the phrenic nerve

A minimally invasive video-assisted thoracoscopic (VATS) procedure to insulate the phrenic nerve was proposed. This technique would preserve the optimal lead position for CRT and avoid instrumentation of pacemaker site. The patient was counselled regarding the uncertain outcome and the potential of injury to the phrenic nerve.

Specific anaesthetic management

The procedure was performed under general anaesthetic, in the right lateral decubitus position. Standard general anaesthesia would suppress any diaphragmatic movement and so specific anaesthetic management was crucial to visualize the twitch and its subsequent abolition. After induction of anaesthesia, a small dose of a neuromuscular blocking agent (a-tracurium) combined with a potent opioid (remifentanil) was used to facilitate placement of the double-lumen tube. This allowed surgical access to the left thorax with single-lung ventilation of the right lung. A peripheral nerve stimulator was placed over the patient’s facial nerve and full return of neuromuscular function was present prior to skin incision. The LV lead was turned on, with immediate and obvious diaphragmatic twitch.

Surgical method

A minimally invasive VATS approach was utilized, with three small (10 mm) ports created, enabling thoracoscopic access to the left pleural cavity. The left phrenic nerve was visualized on the lateral surface of the pericardium. At this point, the blue tip of the Medtronic 4195 lead could also be seen through the pericardium beneath the course of the phrenic nerve. The twitching diaphragm was also directly visualized. Using an image intensifier, the approximate location of the tip of the LV pacing lead was then established. A small 2–3 cm longitudinal pericardial opening was created using Endoshears. A small wad of Surgicel was placed thoracoscopically inside the pericardial window, just underneath the course of the phrenic nerve, in order to insulate the nerve from the underlying pacing lead (Figure 2). The diaphragmatic twitch stopped immediately, even when pacing at 10 V.

Figure 2 Surgicel and bovine pericardial patch placed thoracoscopically inside the pericardiotomy, to lie between the tip of the coronary sinus lead and the phrenic nerve isolating the nerve from the pacing lead tip.
Results

Three-month follow-up

The patient reported improved symptoms, with less breathlessness and fatigue (NYHA Class II), and had returned to work. Her surgical wounds had healed (Figure 3) and all pacing parameters were satisfactory. She had not felt any further diaphragmatic twitching during recovery.

Discussion

With the establishment of CRT as a non-pharmacological treatment modality for heart failure patients, and expected expansion of its indication to less symptomatic patients, interventions to increase success are crucial. Diaphragmatic twitch is a well-recognized and common complication of cardiac resynchronization therapy, due to the phrenic nerve crossing the path of the target vein. In some cases, this may not be apparent during the implant procedure, masked by posture, sedation, or by failure to test at high-voltage outputs.

Surgical implantation of an epicardial LV lead results in similar haemodynamic and symptomatic benefits to endocardial leads, but with increased risk of device infection, haemorrhage and arrhythmias, and limited lead positioning with most leads placed anteriorly. A recent publication also reported a small but significantly increased mortality in patients undergoing isolated surgical LV lead placement compared with trans-venous lead placement.

van Steenberghe et al. reported surgical insulation of the phrenic nerve via left anterolateral thoracotomy with successful abolition of phrenic stimulation at 6 months of follow-up. The authors pointed out that a thoracoscopic approach might be less traumatic. We describe a thoracoscopic surgical patch insulation of the phrenic nerve itself, hence preserving the optimal target vein.

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With concern that the Surgicel would degrade with time, and that the twitch might then recur, we elected to add a folded rectangle of bovine pericardium which was similarly introduced through the pericardiotomy to lie between the tip of the coronary sinus lead and the phrenic nerve isolating the nerve from the pacing lead tip. Tisseel haemostatic sealant (10 mL) was then sprayed liberally, using a dedicated thoracoscopic delivery system, around the bovine pericardial patch as an adhesive to prevent migration of the patch. The patch was further anchored in place by tacking it to the edge of the pericardium with a 4/0 prolene suture. There was no further twitch at 10 V. A 28 Fr Argyle drain was left in the pleural space and after re-inflating the non-ventilated left lung, the port sites were closed with Vicryl sutures. The surgery was completed with no immediate complications and took ~90 min in total.

Post-operative pacing checks were satisfactory and chest radiograph confirmed no paralysis of the left hemidiaphragm. The patient was discharged home after 48 h, with no complications during recovery.

Conclusions

Although phrenic stimulation is a common complication of cardiac resynchronization therapy, it may not always be apparent during the implant procedure. In such cases, a second intervention may be required to allow effective CRT. In these situations, as well as those where no site for effective LV site pacing without twitch can be found, phrenic nerve insulation may be considered. This can be performed using minimally invasive thoracoscopic surgery, as a short-stay procedure and results in optimal CRT with no phrenic stimulation.

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References

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