



# Evaluation of pre-operative bladder contractility as a predictor of improved response rate to a staged trial of sacral neuromodulation in patients with detrusor underactivity

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## Abstract

**Purpose** Sacral neuromodulation (SNM) is one of the few management options shown to improve outcomes in patients with detrusor underactivity (DU). This original research will investigate if preserved bladder contractility can predict a successful treatment with SNM.

**Methods** This is a retrospective study of a prospectively collected database of consecutive patients with DU, who had a staged SNM trial from January 2013 to December 2018, with a minimum of 12 months follow-up. The primary outcome was the success of stage 1 SNM trial.

**Results** In total, 69 patients with DU were followed. The median age was 67 [interquartile range (IQR) 74–55], median baseline bladder contractility index (BCI) 18 (IQR 67–0), and median post-void residual 200 mL (IQR 300–130). There were 35 patients (51%) that responded to a SNM trial. At a median follow-up of 23 months (IQR 39–12), three were removed for poor efficacy. In patients with detrusor acontractility (DAC), six responded (33%), compared to 29 patients (57%) with BCI > 0. This was statistically significant, *p* value 0.03. Younger age was also a predictive factor for SNM response, *p* value 0.02. There were no differences noted in those with gender, neurogenic history, previous pelvic surgery, diabetes, or pre-operative voiding history.

**Conclusion** Our study showed that patients with preserved bladder contractility are more likely to respond to a trial of SNM compared with those that have DAC. Younger age was also predictive of SNM response. UDS is the only method to accurately identify DAC patients. This information will help in patient selection and pre-operative counselling.

**Keywords** Underactive bladder · Detrusor underactivity · Urodynamics · Neuromodulation · Sacral neuromodulation

## Abbreviations

BCI	Bladder contractility index
DAC	Detrusor acontractility
DU	Detrusor underactivity
ICS	International Continence Society
LUTS	Lower urinary tract symptoms
OAB	Overactive bladder

PVR	Post void residual
SNM	Sacral neuromodulation
UAB	Underactive bladder
UDS	Urodynamics

## Introduction

Impaired emptying of the bladder is becoming a well-recognized cause of lower urinary tract symptoms (LUTS). However, this condition can be hard to manage, with both diagnostic and treatment challenges. Many different terms have been used in the past to describe impaired bladder emptying secondary to poor bladder contractility. The International Continence Society (ICS) has recently updated this terminology [1]. Underactive bladder (UAB) is a clinical symptom complex characterized by a slow stream, hesitancy, and straining to void, with or without a feeling of

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incomplete emptying. While detrusor underactivity (DU) is a sign based on urodynamics (UDS) findings of reduced contraction strength or duration, resulting in prolonged bladder emptying or incomplete bladder emptying. DU is often under-recognized, poorly understood, and has a long latency to diagnosis and treatment. Due to the variation in awareness and diagnosis, appropriate treatment may be fragmented and not based on evidence-based practice.

One of the treatments being investigated for the treatment of DU is sacral neuromodulation (SNM). SNM has shown improvement in many patients with various forms of voiding dysfunction, but those with urinary retention may have a lower response compared to other forms of voiding dysfunction [2]. Indeed, there has been mounting evidence that patients with DU can respond well after SNM implant, with sustained results [3–6]. However, predictors of which patients will respond to SNM implant are unclear at this point.

We believe there are patient characteristics that can be identified on UDS that differentiate between those more likely to respond to SNM. The objective of our study is to identify predictive factors in patients with DU prior to SNM implantation. This would allow better patient selection and enable improved counselling of potential outcomes and expectations. We investigate on the role of having preserved detrusor function and contractility in predicting successful response to SNM implant. The primary outcome was the success of the stage 1 SNM trial.

## Methods

This was a retrospective study of prospectively collected data on all consecutive patients who underwent sacral neuromodulation implantation from January 1, 2013 to December 31, 2018. Patients had a pre-operative evaluation with history, examination, UDS, questionnaires, and a voided volume diary. UDS was performed according to the ICS standards. Inclusion criteria are at least a 6-month duration of symptoms, minimum 18 years of age, and pre-operative evaluation with UDS confirmed DU. For the purposes of our study, we defined DU as having a bladder contractility index ( $BCI = PdetQ_{max} + 5Q_{max}$ )  $< 100$ . Exclusion criteria included concomitant pelvic organ prolapse, bladder outlet obstruction, Fowler's syndrome, stress urinary incontinence, or any contraindication to having a SNM implant.

A standard protocol for SNM was followed; all patients underwent an initial staged SNM trial with a 28 cm quadripolar tined lead model 3889 (Interstim, Medtronic, Minneapolis, USA). No patients had a peripheral nerve evaluation during this study, as responses in patients with DU often take longer to manifest. If patients had  $> 50\%$  response (which included a voiding volume diary, uroflowmetry, and

post-void residual (PVR) during the 2-week trial, a second stage implantable pulse generator was performed with the Medtronic neurostimulator device model 3058.

Follow-up for all patients was at least 12 months. The primary outcome measure was the success of the stage 1 SNM trial. Comparison groups for outcome differences were those with and without preserved detrusor contractility, with and without neurogenic history, and those able to void and non-voiders. This investigation obtained ethics board approval with Australian New Zealand Clinical Trials Registry (ANZCTR), trial number 126160015764.

Chi-square statistics were calculated to compare the proportion of patients with response to stage 1 SNM, among the categorical variables gender, neurogenic history, diabetes mellitus history, pelvic surgery history, pre-procedure voiding function, and detrusor acontractility. Wilcoxon rank-sum tests were calculated to compare median values of age, pre-voiding volume and pre-procedure PVR for patients with and without response. Odds-ratios (ORs) were generated to assess the association of patients with response to stage 1 SNM, using a multivariable logistic regression model. The covariates used for the model were: age, gender, neurogenic history, diabetes mellitus history, previous pelvic surgery, pre-procedure voiding function, BCI, and detrusor acontractility. Predicted probability of response after stage 1 SNM was graphed for any significant adjusted predictor variables using the generated regression model. BCI was analysed as both a continuous and a dichotomous variable, to further characterise and determine if a cut-off value may predict response to stage 1 trial. Data were analysed using StataIC v15.1 computer software (College Station, TX, USA). In this study, statistical significance was set at 0.05.

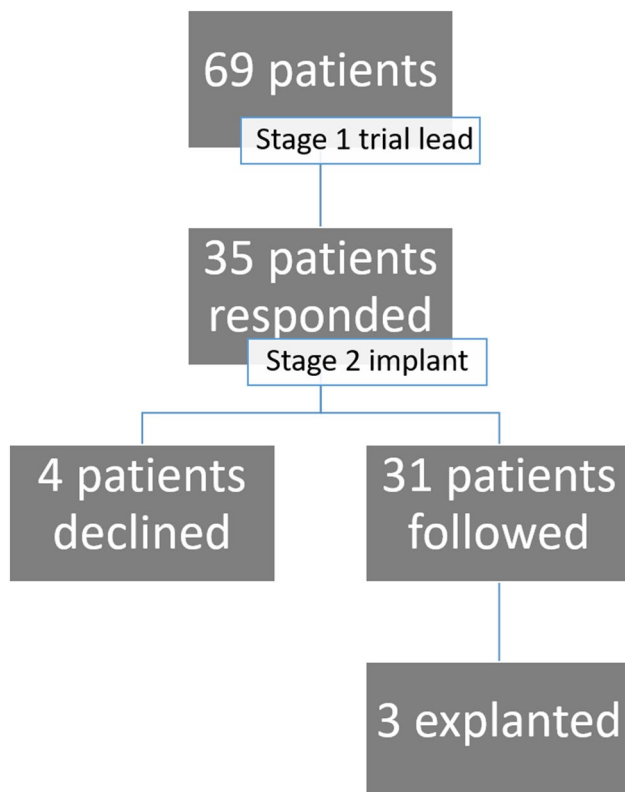
## Results

In total, we identified 69 patients over a 5-year period that fit our inclusion criteria (see Table 1). There were 50 female, and 19 male patients. The median age of patients was 67 (IQR 74–55), median baseline BCI was 18 (IQR 67–0) and median PVR was 200 mL (IQR 300–130). All patients were offered SNM stage 1 trial tined lead implant (Fig. 1). From these, 35 patients (51%) had a favourable response to the trial phase, defined by at least 50% improvement in symptoms, PVR, and voided volume bladder diary data. These patients proceeded to a final stage 2 SNM implant. This significant improvement from SNM responders is reflected in the difference between groups as seen in the post-SNM parameters in Table 2. There were four patients who had a response but declined stage 2 implant. During the median follow-up period of 23 months (IQR 39–12), three patients had their permanent implant removed for subsequent poor efficacy.

**Table 1** Patient demographics

	<i>N</i>
Total patients who had stage 1 implant	69
Median age (IQR)	67 (74–55)
Male	19 (28%)
Female	50 (72%)
Voiders	52 (75%)
Non-voiders	17 (25%)
Neurogenic history	34 (49%)
Diabetes mellitus	8 (12%)
Pelvic surgery	12 (17%)
Median BCI (IQR)	23 (68–0)
Median voided volume, mL (IQR)	140 (239–70)
Median PVR, mL (IQR)	200 (300–130)
Detrusor acontractility	18 (26%)
Patients who had response to stage 1 trial	35 (51%)
Patients who had stage 2 implant	31 (45%)
Median follow up in months (IQR)	23 (39–12)

*BCI* bladder contractility index, *IQR* interquartile range, *PVR* post void residual

**Fig. 1** Patient response and implantation outcome

Increasing age significantly predicted a lower response rate to SNM trial on multivariable logistic regression (OR

0.94, 95% CI 0.895–0.990,  $p$  value = 0.02). Based on the predictive margins generated for SNM response by age, patients aged 50 [probability (Pr) = 0.674, 95% CI 0.524–0.825] were roughly twice as likely to respond to SNM trial as someone aged 80 (Pr = 0.317, 95% CI 0.150–0.484) (Fig. 2). Analysis of age as a dichotomous variable did not significantly predict response, likely contributed by small sample size.

There were 18 patients found on UDS to have detrusor acontractility (DAC): five males and 13 females (Tables 2, 3). DAC is the UDS finding of absent contractility with failure to empty when given permission to void in the absence of EMG abnormalities. Only six patients (33%) with DAC had a favourable response to a stage 1 trial and long term improvement with stage 2 implant. In contrast, 29 patients (57%) who had BCI > 0 had a favourable response to the stage 1 trial and long term improvement without removal. A difference in response to stage 1 trial was not significant on chi-square testing ( $p$  = 0.08). However, on multivariable logistic regression, DAC significantly predicted response to stage 1 (OR 0.107, 95% CI 0.014–0.79,  $p$  value = 0.03) (Table 5 and Fig. 3).

Based on available history, patients were classified on neurological history and voiding ability. There were 34 patients that had a possible neurogenic history that may have contributed to their LUTS, these patients are summarized in Table 4. Comparing those with or without any neurogenic history, there was no difference in SNM trial response outcome on multivariate analysis:  $p$  value 0.31 (Table 5). We identified 49 patients on history and UDS who were voiding prior to implantation, and 20 patients who were non-voiders. In our patient population, voiders are those that are still able to generate enough detrusor contraction or abdominal straining (Valsalva) to void. On multivariable logistic regression, patients' pre-op voiding status did not show any significant difference in response to SNM trial:  $p$  = value 0.75. There were also no differences noted in gender, history of diabetes, prior pelvic surgery, voided volume, PVR, or BCI as a continuous variable (Table 2). BCI as a dichotomous variable was not significantly associated with SNM response using chi-square testing, with cutoffs of 25 or 50 (Tables 2, 6).

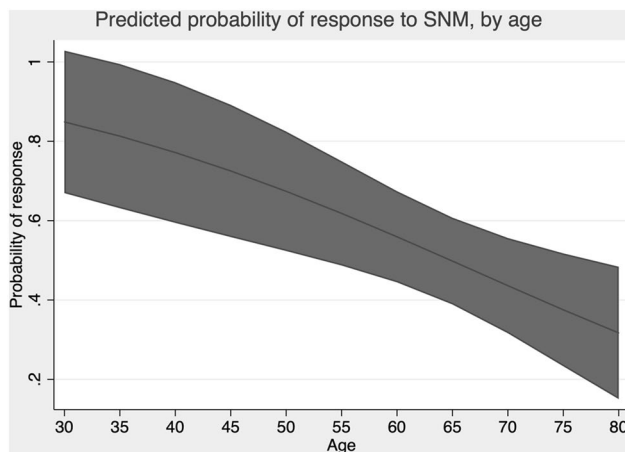
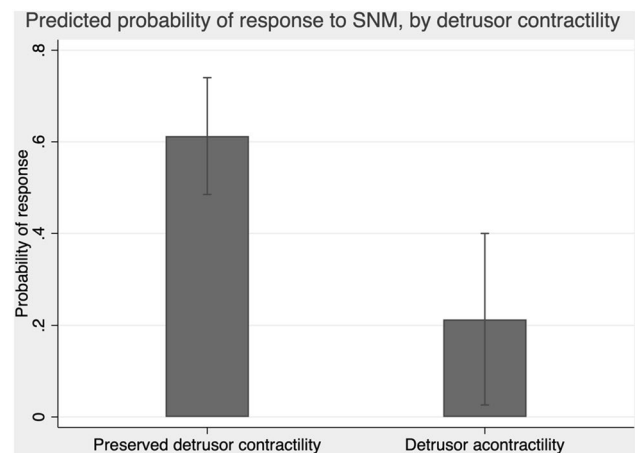
## Discussion

Our study showed that preserved bladder contractility predicted higher response to a trial of SNM compared with those with acontractility. Detrusor acontractility (DAC) may signal the end stage of bladder function, deficient of proper central and peripheral afferents to be stimulated by neuromodulation. We found that any preserved bladder function was more important than a specific magnitude of BCI. BCI cut-offs for response were not predictive, but rather if there was any preservation of detrusor function (Fig. 3 and

**Table 2** Patient Response to stage 1 trial on univariate analysis

	<i>N</i> = 69	Total	No response ( <i>n</i> = 34)	Response ( <i>n</i> = 35)	<i>p</i> value
Age, years (median, IQR)		67 (74–55)	70 (77–61)	64 (72–46)	0.03
Males ( <i>n</i> , %)		19 (28)	12 (50)	7 (49)	0.35
Past history ( <i>n</i> , %)					
Neurogenic history		34 (49)	20 (59)	14 (40)	0.12
Diabetes mellitus		8 (12)	3 (9)	5 (14)	0.48
Pelvic surgery		12 (17)	5 (15)	7 (20)	0.56
Pre-SNM bladder function ( <i>n</i> , %)					
Voiders		52 (75)	24 (71)	28 (80)	0.36
Detrusor acontractility		18 (26)	12 (35)	6 (17)	0.08
Pre-SNM parameters (median, IQR)					
Voided volume, mL		140 (239–70)	130 (250–50)	150 (220–73)	0.87
PVR, mL		200 (300–130)	230 (300–132)	187 (300–125)	0.50
BCI		18 (67–0)	23 (70–0)	15 (65–5)	0.98
Post-SNM parameters (median, IQR)					
Voided volume, mL		160 (300–100)	117 (200–50)	250 (350–150)	<0.001
PVR, mL		123 (230–30)	230 (400–150)	45 (150–21)	<0.001

*BCI* bladder contractility index, *DAC* detrusor acontractility, *DU* detrusor underactivity, *IQR* interquartile range, *PVR* post void residual, *SNM* sacral neuromodulation

**Fig. 2** Predicted probability of response to SNM by age**Fig. 3** Predicted probability of response to SNM by detrusor contractility**Table 3** DAC patients

	<i>N</i>
Total patients with DAC	18
Non-voiding	13 (72%)
Valsalva voiding	5 (28%)
Median PVR (IQR) for voiders	300 mL (300–200)
Neurological history	10 (56%)
DAC patients who had response to stage 1 trial	6 (33%)

*DAC* detrusor acontractility, *IQR* interquartile range, *PVR* post void residual

**Table 4** Neurogenic etiology

	<i>N</i>
Total patients with neurogenic history	34
Spinal cord pathology	29 (85%)
Parkinson's disease	2 (6%)
Cerebral vascular event	3 (9%)

Table 6). Although the exact reason why SNM is effective is not proven, one likely explanation is via negative feedback and downregulation of the sphincter and guarding reflex [7, 8]. Accepting this, SNM requires functioning and intact

**Table 5** Logistic Regression for response to stage 1 trial

Dependent variable	Predictors	<i>b</i>	SE of <i>b</i>	<i>p</i> value	Odds ratio	95% CI for OR
Response to stage 1	Age	- 0.060	0.026	0.02	0.94	0.895–0.990
	Male	- 0.381	0.704	0.59	0.68	0.172–2.715
	Neurogenic history	- 0.569	0.560	0.31	0.57	0.189–1.698
	Diabetes mellitus history	1.023	0.846	0.23	2.78	0.530–14.584
	Pelvic surgery history	0.002	0.764	0.99	1.00	0.224–4.480
	Voiders	0.303	0.941	0.75	1.35	0.214–8.566
	Detrusor acontractility	- 2.235	1.020	0.03	0.11	0.014–0.790
	BCI	- 0.012	0.011	0.29	0.99	0.967–1.010
	Constant		4.872	1.904	0.01	130.537

*BCI* bladder contractility index

**Table 6** BCI as dichotomous variable response rate to stage 1 trial

<i>N</i> =69	Total	No response ( <i>n</i> =34)	Response ( <i>n</i> =35)	<i>p</i> value
BCI> 50 ( <i>n</i> , %)	31 (45)	15 (48)	16 (52)	0.90
BCI> 25 ( <i>n</i> , %)	33 (48)	17 (53)	16 (49)	0.72

*BCI* bladder contractility index

afferent pathways to improve LUTS, which may no longer be present in those with end-stage detrusor function and subsequent DAC. This hypothesis of the feedback loop effect on detrusor function is suggested by the results of one small study that showed that SNM may increase bladder emptying by inhibition of the sphincter and pelvic floor tone and thus allowing normal bladder contraction [9]. In this paper by Everaert et al. patients who showed improvement in bladder emptying after SNM were those with detrusor underactivity (preserved contractility), and impaired sphincter relaxation or sphincter hypertonicity [9]. These findings suggest the role that SNM may play in re-integrating the lower urinary tract system and circuitry.

While we found that some DAC patients still responded, this was significantly less than those with any preserved detrusor function. The minority of patients with DAC who responded may be very early in the course of their detrusor failure or had such low tonicity that UDS was unable to measure the contractility during the study. The use of ambulatory UDS may be able to better diagnose true DAC patients and separate those that conventional UDS may misattribute impaired pelvic floor relaxation or a 'shy bladder' as DAC, as these may still respond favourably to SNM. However, ambulatory UDS has its own barriers to implementation, including cost, availability and interpretation difficulties. Voiding is an integrative process that requires detrusor contraction and outlet relaxation, and the total process can still be modulated by SNM, since those with some intact function may reincorporate some additional control with

SNM stimulation. One recent study highlighted this idea; a study of 18 men with DU found that a bladder contractility (Maastricht-Hannover) nomogram could possibly predict response to SNM [10]. The authors showed that only 20% of patients below the 10th percentile responded, but 86% of men between the 10 and 25th percentiles of the nomogram were treated successfully with SNM. The same group identified in a different very heterogeneous cohort that despite differences in detection of DU with conventional UDS compared with ambulatory UDS, those with acontractility responded much less to a SNM trial and subsequent implantation [11]. Indeed, very few studies have since addressed this important finding further, and ours represents the largest cohort of these patients with confirmatory results. A recent systematic review of DU and UAB found less than 50 specific articles addressing these topics [12]. This update highlights the important emerging area of research still needed to understand this complex problem.

The concept of using a patient's pre-operative ability to void, albeit with incomplete bladder emptying, as a guide to response to a trial of SNM is an interesting one. After all, if there is a direct association, it would be an easy predictor that can be used to counsel patients preoperatively. Our study is the first to investigate this and we showed that a patient's pre-operative voiding status does not predict improved response with a trial of SNM ( $p=0.75$ , Table 2). A possible explanation is that patients who are "voiders" may include those who void spontaneously with some preserved detrusor function, but also those who void with significant abdominal straining (Valsalva voiders). Many of these patients are unaware they are Valsalva voiders and are straining subconsciously when voiding. The only reliable way to distinguish between these two groups is to perform pre-operative UDS. UDS is useful not only in diagnosing DU (which can be difficult to do clinically due to the overlap of symptoms with other diagnoses like the overactive bladder and bladder outlet obstruction), but is also helpful for counselling and discussion about trial expectations in patients with urinary retention [13].



Increasing age was also shown in our study to be an important predictor of poor response to SNM trial. Interestingly, a previous study showed that age was not predictive of SNM response regardless of indication, but this same study found a difference in gender, with females more likely to respond to a stage 1 trial [14]. This was contrary to our findings, which suggest no difference between gender responses [14]. This may be partly explained by our patient population containing only patients with UAB, whereas theirs was a mixture of all SNM indications, including the overactive bladder (OAB). Additionally, with increasing age, DAC may also increase and result in poor outcomes in our patient cohort. This is the first study with all DU patients to underline the significant relationship of age and SNM response.

Although SNM is not a well-established treatment in neurogenic patients with OAB, several previous studies have shown efficacy and acceptable safety in these patients [15–18]. One systematic review found from over 250 pooled neurogenic patients, the response rate to stage 1 testing was around 68% [15]. As the SNM response rates in idiopathic OAB patients are generally higher compared to neurogenic ones, we thought it would be interesting to see if this holds true in patients with urinary retention. Interestingly, our study found that those with a neurological history had similar outcomes to those without in patients with urinary retention (Tables 1, 2). Much less is known about how SNM works in neurogenic patients, but potential theories are a multifactorial effect including neural plasticity, reactivation of previous neural pathways, and preservation of existing reflex arcs [15, 16, 19]. The limitation with these previous studies, and to an extent our study, is that many of these patients with a neurogenic history are heterogeneous and at various time points in their neurogenic pathology. Due to these heterogeneous patients, some clinicians advocate for classification of the neurogenic bladder to be based on UDS findings rather than on neurogenic history, which may have inconsistent effects on the lower urinary tract.

One of the limitations of this study is that BCI is not well studied in women, and is validated for use only in men with DU. However several other recent studies have used BCI in women [20–22]. To maintain consistency during our trial period, BCI was used as there were no other available validated nomograms to evaluate DU in women. Other proposed standards for DU such as the Watts factor (WF) were considered but it is a complex calculation compared with BCI. Watts factor is also not a precise measurement in women and there is no widely accepted gender-specific cut off value of WF to determine DAC or DU. Given these issues for calculation of bladder contractility, we opted for a simple BCI measurement not as a continuous scale measure or range, but rather as a simple confirmation of bladder function. Our study stresses that the important point is not the amount of bladder function that matters, but if there is any at all.

We showed that patients tend to respond unfavourably when there is no function, and BCI was a simple measurement of that fundamental idea. Therefore, regardless of the parameters of BCI measurement in women, any contractility rather than increasing levels of contractility determined response.

Another weakness of this study may be related to how subjective reporting the SNM trial response can be (> 50% improvement), but this would have been partially mitigated by using other objective parameters (PVR, voided volume bladder diary). There may also be limitations with selection bias from our complex patients and tertiary referral bias, which might explain the lower average success rate with SNM stage 1 trial (51%) compared with other studies [23]. However, some published success rates can be misleading as many studies include OAB and other heterogeneous patients with noted higher SNM success rates. Furthermore, outcomes were not always reported uniformly for both the stage 1 trial phase, and stage 2 implantation [7]. The high proportion of women is also noted in our cohort, but this may be related to a referral bias of our female urology centre. Most recently, Hartigan et al. found that women might also have a disproportional prevalence of DU, finding that up to 45% of older neurologically normal women had evidence of DU on UDS [24].

This objective UDS finding is encouraging and suggests that other predictive factors may exist. Suggestions for future studies include assessing pre and post-operative UDS SNM findings in DU, evaluating changes in detrusor function and BCI among other objective responses. Larger prospective studies may be needed for further evaluation of these.

## Conclusion

In properly selected patients with voiding dysfunction, SNM can significantly improve quality of life. Our study showed that in patients with DU, younger patients and those with preserved bladder contractility are more likely to respond to a trial of SNM compared with those without any detrusor function. Other pre-operative characteristics such as neurogenic aetiology and voiders vs non-voiders, were non-predictive. UDS can help in identifying those with preserved contractility. This information is invaluable in the pre-operative selection of patients for SNM as well as counselling and management of patient expectations. Future studies should focus on determining other predictive factors to guide patient selection and treatment.

**Author contributions** GC: project development, data collection, analysis and manuscript writing. LGQ: data analysis and manuscript writing. JG: project development, data collection, and manuscript writing.

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## Compliance with ethical standards

**Conflict of Interest** None.

**Ethical approval** All procedures performed in this retrospective study involving human participants were in accordance with the ethical standards of the institutional review board of Austin Health with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

**Informed consent** Not applicable for this retrospective analysis.

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