# CORRESPONDENCE

## Research Correspondence

### Modified Temporary Cardiac Pacing Using Transvenous Active Fixation Leads and External Re-Sterilized Pulse Generators

**To the Editor:** Transcutaneous temporary pacing can be unreliable, and the stimulation can be uncomfortable; epicardial pacing requires thoracotomy (1,2). Transvenous percutaneous pacing systems placed "blindly," fluoroscopically, or electrocardiographically have therefore become the mainstay of temporary pacing (1). However, temporary pacing leads have a significant risk of dislodgement, infection, loss of capture, or even chamber perforation (3). In addition to risk of infection with prolonged central venous access, patient immobilization ("bed rest") can result in additional complications and economic burden.

As a result, use of temporary pacing systems is limited to no more than a few days (1). However, prolonged temporary pacing may sometimes be required. Treatment of infection in a pacemaker-dependent patient (4), post-operative recovery from cardiac valve surgery (5), or Lyme myocarditis with transient atrioventricular (AV) block (6) are representative of cases where prolonged pacing support may be necessary.

We present an alternative approach for modified temporary pacing support to address these scenarios. An active fixation pacing lead is placed fluoroscopically via percutaneous venous access and connected to a re-sterilized external pulse generator. This paper describes our clinical experience with this pacing system.

All patients who received modified temporary pacing support by the arrhythmia service at Brigham and Women's Hospital from July 2000 through November 2004 were included in the analysis. The facility is a regional referral center for lead extraction, resulting in a significant number of patients admitted with infected devices. Patient clinical characteristics, details of the implantation procedure, and follow-up results were evaluated.

If the commonly accepted indication for pacing (7) was potentially reversible but likely to persist for more than one or two days, or if the patient had a treatable contraindication to *permanent* hardware implantation, a modified temporary pacing system was considered.

New standard active-fixation leads were selected at physician discretion. Pulse generators previously explanted from patients without known infection were re-sterilized and utilized. In a sterile procedure room, venous access was achieved using standard percutaneous technique (without cutdown) (Fig. 1). The pacing lead was advanced under fluoroscopic guidance, fixed to the myocardium, and tested in a standard fashion. The lead was then sutured to the skin using the anchoring sleeve (Fig. 1). After connection to the lead, the pulse generator (programmed SSI) was secured to the skin surface (Fig. 2).

After implantation, patients were returned to their hospital beds and, unless contraindicated, were encouraged to ambulate. Plain film chest radiography and interrogation were performed the following day to exclude acute dislodgment. Patients were followed clinically to determine if a permanent device was still indicated and/or if concurrent infection or illness had resolved sufficiently to allow *permanent* device implantation. At the time of permanent implantation, the external pulse generator was detached followed by retraction of the active fixation mechanism from the myocardium under fluoroscopic guidance.
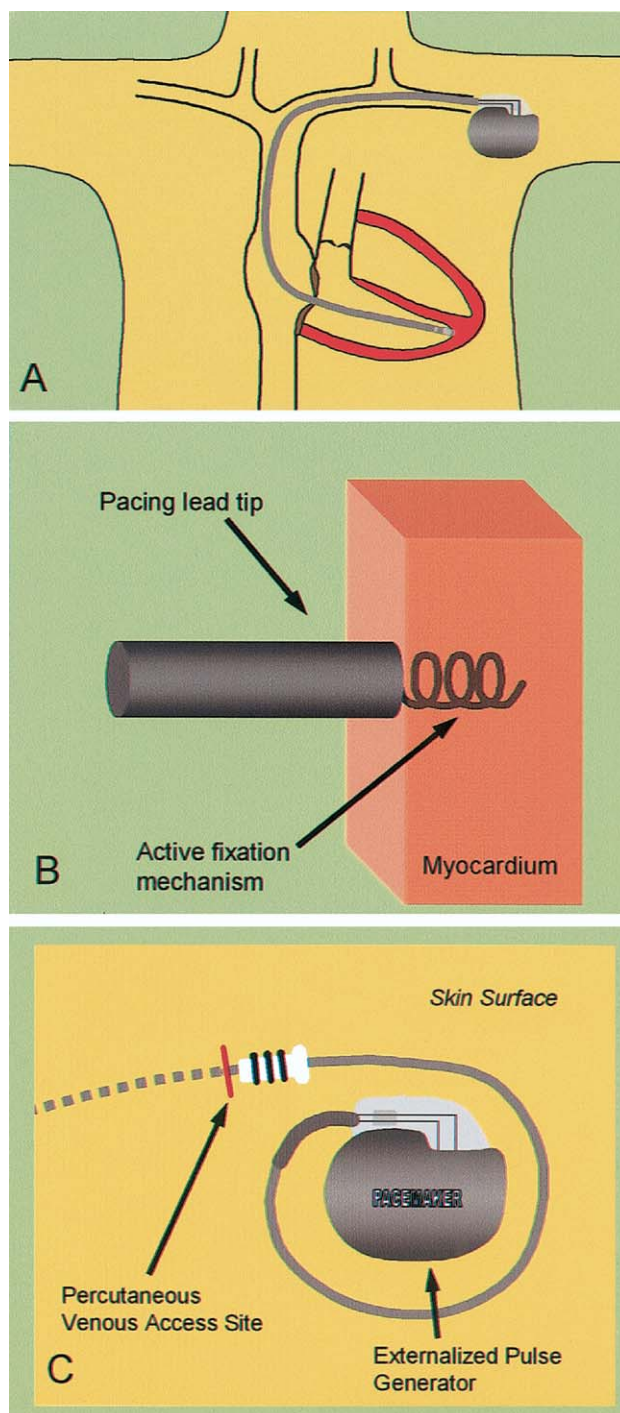


**Figure 1.** Implant technique schematic. **(A)** The pacing lead is inserted via percutaneous access and placed in the right ventricle (or atrium). **(B)** An extendable-retractable active fixation lead is used to reduce migration. **(C)** The proximal lead remains external to the patient, is anchored via suture sleeve to the skin, and attached to the external pulse generator.
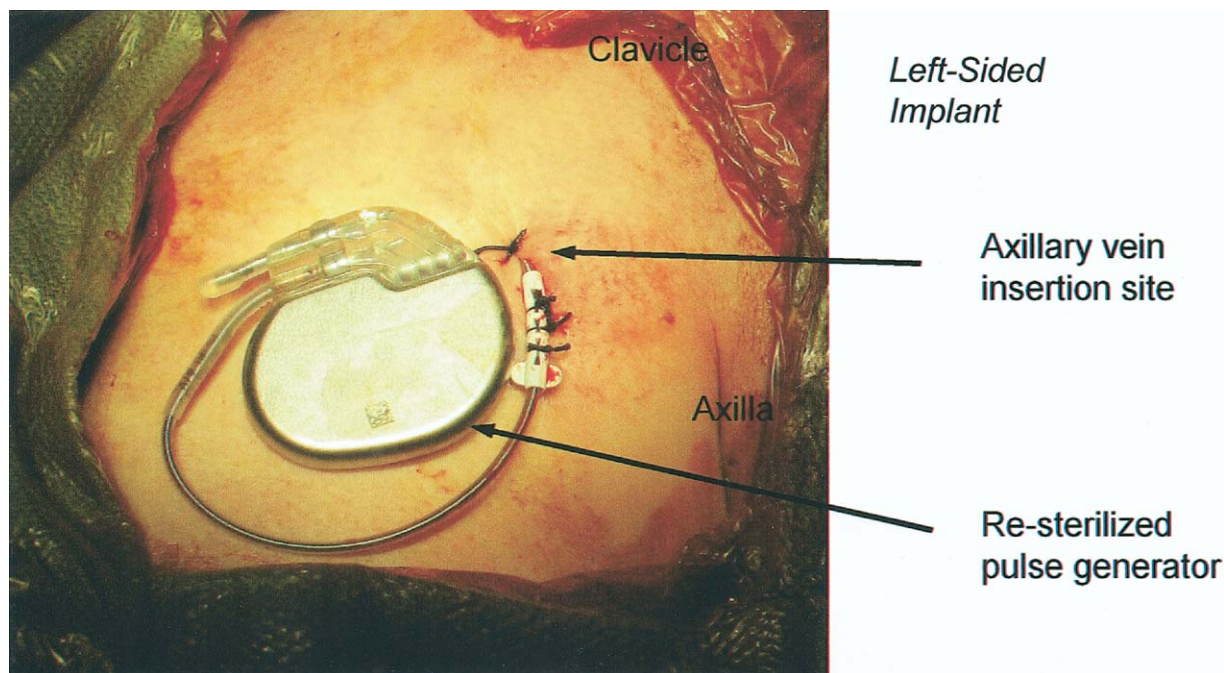
**Figure 2.** A newly implanted modified temporary pacing system is shown, with central venous access via axillary vein puncture. The proximal lead is external and sutured to the skin. A pulse generator is attached and then taped to the skin.

The 62 patients receiving modified temporary pacing were 68.4 ± 14.2 years of age (n = 37, 59.7% male), with a mean left ventricular ejection fraction of 47 ± 2%. Twenty-six patients (41%) had known coronary artery disease. The median hospital length of stay was 18 days. Indications for pacing were symptomatic AV block (n = 39, 62.9%), sick sinus syndrome (n = 17, 27.4%), and one instance of drug-related torsades de pointes.

Reasons for delaying permanent device implantation included bacteremia (n = 27, 43.5%), early post-cardiac surgery (n = 17, 27.4%), concurrent medical illness (n = 6, 9.7%), serologically confirmed Lyme disease (n = 3, 4.8%), and miscellaneous (n = 9, 14.5%). Among patients with bacteremia, most were elective admissions for lead and generator extraction for infection (n = 10, 37.0%) or primary diagnoses of endocarditis (n = 3, 11.1%).

Central venous access was via the internal jugular (n = 31, 47.7%), axillary (n = 21, 32.3%), subclavian (n = 11, 16.9%), cephalic (requiring cutdown, n = 1, 1.5%), and femoral (requiring bed rest, n = 1, 1.5%) veins. The majority of leads were placed in the right ventricular apex (n = 59, 95.2%), although three patients received right atrial pacing support.

The median duration of modified temporary pacemaker implantation was 7.5 days. Approximately two-thirds (n = 41, 66.1%) received permanent device implantation during index hospitalization. Of the 21 patients not felt to be candidates for permanent pacemaker implantation, 9 (42.9%) no longer required pacing support. All Lyme myocarditis patients recovered AV conduction, and none required permanent pacing, with time to recovery between 6 to 11 days. Among the post-operative patients, two died from non-arrhythmic causes during hospitalization, and five (23.8%) recovered conduction, obviating the need for permanent pacing. Among the remaining patients, nine (42.9%) died of non-arrhythmic causes during hospitalization, and three were transferred to the referring hospital for further treatment, primarily for infection, followed by permanent device implantation there.

No deaths were due to arrhythmia. No implant-related complications were observed, including lead dislodgment, perforation, pericardial effusion, or device infection. In those patients initially in an intensive care setting due to pacing support requirement, providers were encouraged to transfer the patient to a "step-down" or telemetry ward.

In the largest case series to date, we demonstrate an alternative approach to provide intermediate-term pacing (8). The advantages include lead position stability, allowance for full treatment of infections before permanent device placement, and an extended period of observation for recovery of normal conduction. Patients with significant risk of mortality are allowed to follow a natural history without the expense of permanent hardware implantation. These re-sterilized pulse generators are free of known infection and only contact the patient's skin, resulting in minimal additional infection risk.

This series represents patients that are generally high risk, referrals, and have a high incidence of device infection. Patients with acute myocardial infarction requiring temporary pacing are generally treated in our cardiac catheterization laboratory; this important subset of patients is not addressed. These points may limit the generalizability of our findings. Other approaches to intermediate duration pacing have been described utilizing specialized pacing wires with active fixation mechanisms (9). Comparisons to these other temporary pacing systems should not be inferred and are beyond the scope of this analysis.

This pacing system requires no new technology and utilizes equipment already available to the implanting physician. It provides safe, stable, and reliable pacing in the individual who is immediately pacemaker-dependent but has reasons for delay in

permanent device implantation. We believe this system can be readily implemented in such clinical scenarios.

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# Brain Natriuretic Peptide Levels in Constrictive Pericarditis and Restrictive Cardiomyopathy

**To the Editor:** Constrictive pericarditis (CP) often is a challenging diagnosis despite sophisticated modalities (1–4). If a simple test could alert physicians to the possibility of CP in patients with evidence of heart failure, it would be valuable.

Recent data (5) suggest that brain natriuretic peptide (BNP), released in response to cardiac stretch (6), is normal or only minimally elevated in patients with CP and more elevated in patients with restrictive cardiomyopathy (RCMP). However, there are a variety of etiologies for CP. Many patients at our institution present after either mantle chest radiation and/or open heart surgical procedures (7). These patients may have additional myocardial damage that could raise BNP even when concomitant CP is present. To determine whether BNP levels in all patients with CP are different from those with restriction, we studied patients with well-documented CP and RCMP. We separated patients with CP into those with idiopathic disease and with CP due to secondary causes.

We reviewed 22 patients with surgically confirmed CP where BNP was measured pre-operatively. Patients were divided into those with idiopathic CP or secondary CP if there had been previous cardiac surgery or chest radiation. We identified patients with RCMP who had appropriately timed BNP values as a comparison group. The diagnosis of RCMP was made according to criteria by Hurrell et al. (4). These patients also had characteristic echocardiographic findings. The diagnosis of CP was confirmed at surgery.

Two-dimensional echocardiographic examinations were performed as previous reported (8); BNP measurements were performed with the Biosite assay (La Jolla, California) close to the time of the echocardiographic examinations and before surgical pericardiectomy. The interval between echocardiogram and BNP measurement was $1.3 \pm 7.4$ days, and in all patients except 6, the interval was 1 day.

Summary statistics are presented as frequencies (percentages) for categorical variables, as mean $\pm$ SD for the normal variables, and as median and first and third quartile (Q1, Q3) for skewed variables. The normal distribution of the variables was verified performing the Shapiro-Wilk W test. Only BNP was very right skewed. Log10-transformed values of BNP (Log10BNP) were used in the analysis to obtain a normal distribution of the values. Categorical variables were compared by Fisher exact test, continuous normal variables by analysis of variance (followed by Tukey's HDS post-test). All tests were two-sided, and for all analyses $p < 0.05$ was considered statistically significant. Data were analyzed with JMP Version 5 (SAS Institute Inc., Cary, North Carolina).

Twenty-two patients had surgically confirmed CP, 11 patients had idiopathic CP, and 11 secondary CP (8 with previous cardiac surgery and 3 with radiation therapy). They were compared to 11 patients with RCMP. Patient characteristics are reported in Table 1. There were no significant differences among groups. Median BNP was 80 (44 to 193) ng/l for idiopathic CP, 278 (118 to 526) ng/l for secondary CP, and 499 (361 to 606) ng/l for RCMP. The Log10-BNP was $1.9 \pm 0.3$ ng/l for idiopathic CP, $2.4 \pm 0.3$ ng/l for secondary CP, and $2.7 \pm 0.2$ ng/l for RCMP. There was a statistically significant difference between the BNP values with idiopathic CP and RCMP ($p < 0.05$) and between patients with idiopathic CP and those with secondary CP ($p < 0.05$). There were no significant differences in BNP between patients with secondary CP and RCMP (Fig. 1).

All patients with BNP <150 ng/l had CP. Higher BNPs were seen in patients with secondary CP and RCMP. The only differentiating finding with RCMP was a BNP >650 ng/l, which did not occur with CP.

Our data confirm and extend those recently published (5). Brain natriuretic peptide levels were significantly lower in patients with idiopathic CP than those with RCMP. However, BNP is not significantly different in patients with CP secondary to previous