should be considered among a growing list of techniques for restoring pulse oximeter function.

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Pacemaker Interactions with Transcutaneous Cardiac Pacing

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BECAUSE of electrical interference, patients with permanent or temporary pacemakers are at increased risk in the operating room and critical care environment.[1] We describe two cases of interaction of noninvasive transcutaneous pacing with an implanted demand (VVI) transvenous pacemaker and temporary epimyocardial pacing, respectively. Whereas in the first case, this interaction could be exploited therapeutically to inhibit ventricular pacing by the implanted transvenous pacemaker, resulting in resumption of sinus rhythm with improved hemodynamics, it evoked asystole by suppression of epimyocardial pacing in the second case.

Case Reports

Case 1

An 84-yr-old woman (53 kg body weight, 160 cm) with femoral neck fracture was scheduled for total hip arthroplasty. Past medical

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† Oberarzt.

history included sick sinus syndrome with several episodes of syncope, resulting in right infraclavicular implantation of a ventricular pacemaker (Siemens, Munich, Germany; programmed mode VVI, rate 65 beats/min) 3 yr ago, congestive heart failure (New York Heart Association classification grade 3), hypertension, and diabetes. Except for bilateral tibial edema and discrete rales over both lower dorsal lung fields, preoperative physical examination, vital signs, and laboratory studies were unremarkable. Medications included digitoxin, verapamil, nifedipine, isosorbide dinitrate, furosemide, glyburide, and L-thyroxine. A chest x-ray showed left heart enlargement, Kerley-B lines, and somewhat prominent interstitial markings. A 12-lead electrocardiogram revealed sinus rhythm (85 beats/min) with regular ventricular pacing (65 beats/min) during application of a magnet.

Following placement of electrocardiogram leads (II and V5), radial artery, and basilic vein central venous catheters, anesthesia was induced with divided doses of thiopental (total 100 mg intravenously), fentanyl (total 200 μg), and vecuronium (6 mg), and after tracheal intubation, maintained with enflurane (0.6–1%) in nitrous oxide/oxygen (50%/50%). Arterial pressure decreased from a baseline of 160/90 to 120/60 mmHg, with sinus rhythm at a rate of 80–100 beats/min. However, over the next 20 min, heart rate decreased progressively, and when ventricular pacing ensued at the programmed demand rate of 65 beats/min, arterial pressure precipitously decreased to 70/35 mmHg (mean 46 mmHg).

To improve hemodynamics, the anesthesiologist intended to increase the heart rate using a transcutaneous pacemaker (Pace 500 D, Osypka, Grenzach, Germany) connected to circular electrodes (diameter 13 cm, effective electrode area 50 cm²) that had been taped to the precordial and left paravertebral skin to be used in case the implanted pacemaker would malfunction during electrocautery. Transcutaneous stimulation (pulse width 40 ms, rate 80 beats/min) was begun with a current of 10 mA when it was noted that this maneuver evidently inhibited the implanted pacemaker and resulted in recovery of sinus rhythm at a rate of 60 beats/min (fig. 1). Under these conditions, arterial pressure immediately increased to 98/45
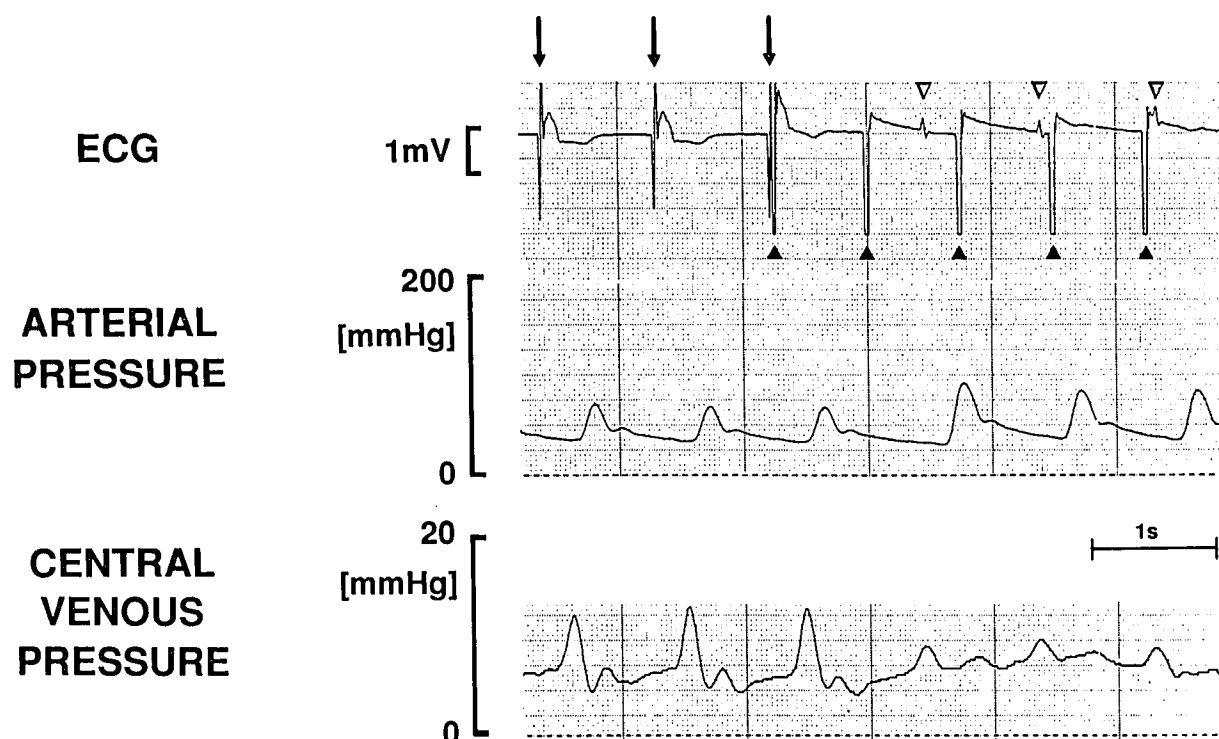
CASE REPORTS

Fig. 1. Interaction between an implanted ventricular demand pacemaker and stimulation with a transcutaneous pacemaker. (*Left*) Right ventricular transvenous pacing accompanied by arterial hypotension and retrograde ventriculoatrial conduction as shown by the cannon-a-waves in the central venous pressure tracing. (*Right*) Following inhibition of the implanted pacemaker by noncapturing transcutaneous stimulation with a transcutaneous pacemaker, a spontaneous cardiac rhythm resumes, accompanied by a marked increase in arterial pressure and disappearance of the cannon-a-waves. Arrow = stimulation spike of implanted ventricular pacemaker followed by a branch block type QRS complex; closed triangle = spike of transcutaneous pacemaker without ventricular capture; open triangle = spontaneous cardiac activity.

mmHg. In contrast, transcutaneous ventricular pacing with capture of the heart (current 45 mA, rate 80 beats/min) resulted in a blood pressure of only 80/40 mmHg. Accordingly, transcutaneous stimulation was continued at the minimum adjustable current (10 mA) and pulse width (5 ms) that was effective in inhibiting the implanted pacemaker and maintaining sinus rhythm. Fifteen minutes later, when sinus node frequency had increased to 72 beats/min, resulting in suppression of the implanted pacemaker, transcutaneous stimulation was terminated. The patient's remaining anesthesia and further hospital course were uneventful, and a postoperative evaluation of the implanted pacemaker showed its proper function.

Case 2

A transaortic myectomy was carried out during cardiopulmonary bypass in a 48-yr-old man suffering from hypertrophic subaortic cardiomyopathy and atrial fibrillation. Because of development of a 3rd-degree atrioventricular block during his postoperative stay in the ICU, his heart was paced (Biotronik, model EDP 30, mode VVI, rate 90 beats/min) using the temporary right ventricular epimyocardial wires brought out through the skin in the epigastrium. Since the

pacing threshold was already high and sometimes can progressively increase exceeding maximum pacemaker output, resulting in loss of ventricular capture, pacing electrodes (diameter 13 cm, effective electrode area 50 cm^2) were prophylactically placed to the precordial and left paravertebral skin and connected to a transcutaneous pacemaker (Pace 500 D) to be used as a back-up system should epimyocardial pacing fail. To check the feasibility of transcutaneous pacing, a brief stimulation (stimulus width 40 ms, rate 95 beats/min) that progressively increased the current up to 40 mA was carried out. However, this failed to capture the heart and resulted in inhibition of epimyocardial pacing and a brief (6 s) asystole (fig. 2). The transcutaneous pacemaker was switched off immediately, resulting in resumption of pacing by the epimyocardial pacemaker. No further attempts were made to evaluate the transcutaneous pacing threshold in this awake patient, and he eventually recovered spontaneously from the 3rd-degree atrioventricular block several days later.

Discussion

The cases presented demonstrate that inhibition of an implanted transvenous pacemaker as well as of tem-
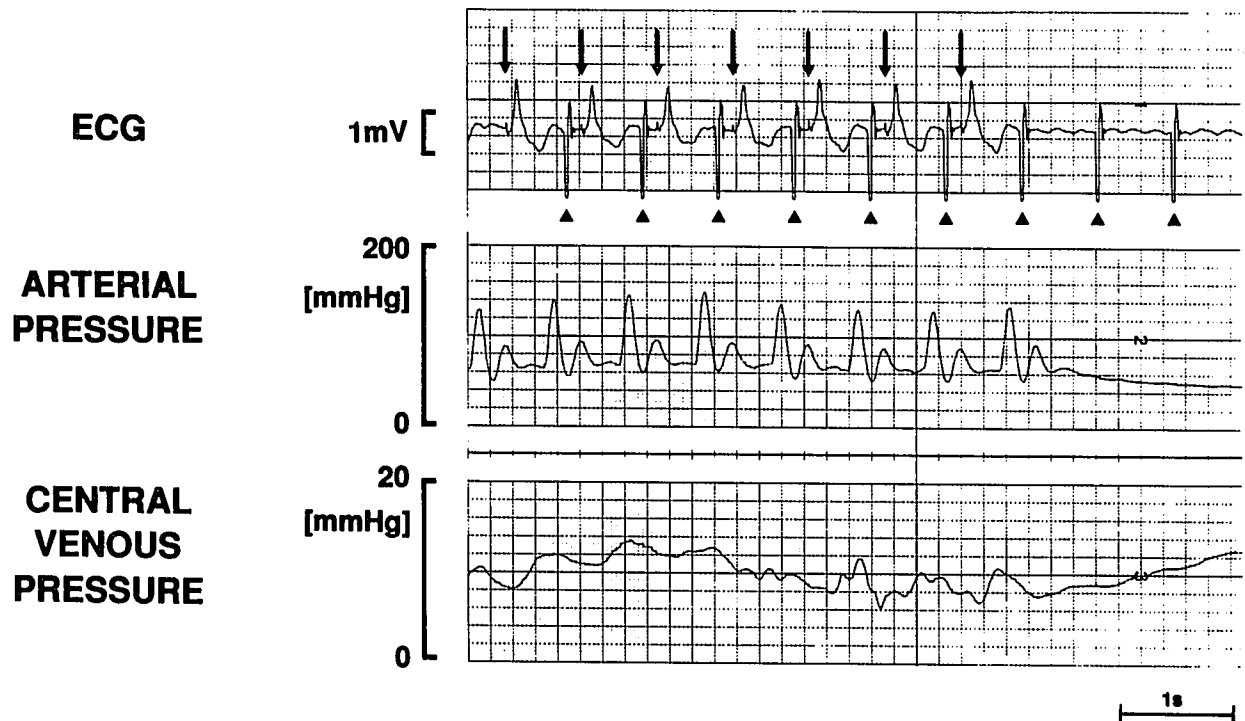
CASE REPORTS

Fig. 2. Interaction between epimyocardial (VVI) pacing and stimulation *via* a transcutaneous pacemaker in a patient with 3rd-degree atrioventricular block following transaortic resection of subvalvular tissue in a case of hypertrophic cardiomyopathy. Following inhibition of epimyocardial pacing by stimulation with a transcutaneous pacemaker and progressively increasing current, a brief asystole ensues because of a lack of immediate generation of a ventricular escape rhythm. Arrow = stimulation spike of epimyocardial ventricular pacing followed by a branch block type QRS complex; closed triangle = spike of transcutaneous pacemaker. Beginning with the 7th spike, the demand pacer is inhibited without ventricular capture, resulting in transient asystole.

porary epimyocardial pacing is readily possible *via* interference with their sensing function by a standard noninvasive transcutaneous pacemaker. Accordingly, this type of pacemaker interaction with a transcutaneous pacemaker, which to our knowledge has not been described previously, must be added to the list of circumstances leading to potential interference with normal pacemaker function in the operating room and critical care environment.[1]

Atrial contraction during end diastole increases ventricular filling and thus, by increasing preload, contributes to maintenance of cardiac output and arterial pressure.[2] It is only during profound myocardial failure and with an increase in left ventricular end-diastolic pressures to 20–30 mmHg or greater, that atrial contraction does not substantially increase end-diastolic left ventricular volume.[3] In contrast, loss of a timely atrial contraction, as with ventricular pacing or during AV junctional rhythm, can markedly decrease cardiac

output, particularly under conditions of myocardial hypertrophy and decreased ventricular compliance (hypertension, valvular aortic stenosis, hypertrophic cardiomyopathy) or with mitral valve stenosis.[4] Furthermore, in half of the patients, ventricular pacing leads to retrograde (ventriculoatrial) conduction, with atrial contraction against the closed AV valves,[5] as shown by cannon-a-waves in the central venous pressure tracing (fig. 1). This may further diminish ventricular filling. Finally, during right ventricular pacing, the interventricular septum contracts in an asyncronous fashion, compared to the left ventricular free wall in most patients increasing end-systolic volume at the expense of stroke volume.[6,7] Accordingly, the precipitous decline in arterial blood pressure with the onset of ventricular pacing as seen in case 1 is not unexpected and required prompt treatment.

Treatment options to be considered in this situation include intravenous administration of atropine or β-

adrenergic agonists, transesophageal atrial pacing,[8] or reprogramming of the pacemaker by a cardiologist using special equipment. While the pharmacologic approach, especially in patients with coronary artery disease, is often either undesirable, because of potential tachycardia and arrhythmia, or ineffective in restoring sinus rhythm in patients with sick sinus syndrome, the latter options were not readily available. Reprogramming the pacemaker to a higher rate, either in the VVI or the VVO mode, may not be optimal to appropriately cover the patient's hemodynamic needs for the duration of anesthesia and surgery and, at the same time, avoid potential interference by electrocautery.

Inhibition of transvenous and epimyocardial pacing due to transcutaneous pacing most likely was caused by interference with the sensing function of the former pacemakers. Transcutaneous pacing delivers a substantial current to the chest wall,[9] which may be sensed *via* the transvenous or epimyocardial leads of another pacemaker and mistaken by their sensing circuits as a spontaneous cardiac depolarization when the sensing threshold is exceeded.

Though discovered by chance, termination of ventricular pacing *via* the implanted pacemaker by transcutaneous pacing in case 1 restored sequential atrioventricular conduction and, therefore, represented a causal treatment approach. In fact, minimum current output and stimulus duration of the transcutaneous pacemaker already were sufficient to suppress the implanted pacemaker, allowing resumption of sinus rhythm accompanied by hemodynamic improvement.

The type of pacemaker interaction described may be beneficial or detrimental to the patient. On one hand, use of a transcutaneous pacemaker as a tool for suppressing an internal pacemaker has the definite advantage that reliable pacing[9] at a higher rate can immediately be instituted if desired, *e.g.*, in case that electrocautery induces malfunction, unintentional reprogramming, or switch to a fixed low-rate backup mode of the implanted pacemaker,[1,10] or if the implanted pacing leads must be disconnected from the pacer for surgical reasons. In this regard, the use of transcutaneous pacemaker stimulation to inhibit an implanted transvenous pacemaker appears preferable to a hand-held nerve stimulator applied to the skin overlying a pacemaker.[11]

On the other hand, as shown in case 2, interference with a pacemaker *in situ* by stimulation *via* transcutaneous pacing electrodes is not without risk, because

asystole may result in those hearts being unable to generate an escape rhythm. Nevertheless, this should be easily recognizable and is managed rapidly either by termination of transcutaneous stimulation, with resumption of effective pacing by the implanted pacemaker, or by increasing the output of the transcutaneous pacemaker until the heart is captured.

In conclusion, a transcutaneous pacemaker, with its stimulating electrodes taped to the skin in the standard position and with minimum current output and duration, can interfere with the sensing function of other transvenous or epimyocardial pacemakers and result in inhibition of demand pacing. In selected cases, this interaction may be used to suppress undesired ventricular pacing by an implanted pacemaker, potentially allowing cardiac performance to improve.

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