Inhibition of a Demand Pacemaker by Electrosurgery

John Kevin O'Donoghue

Chest 1973;64;664
DOI 10.1378/chest.64.5.664

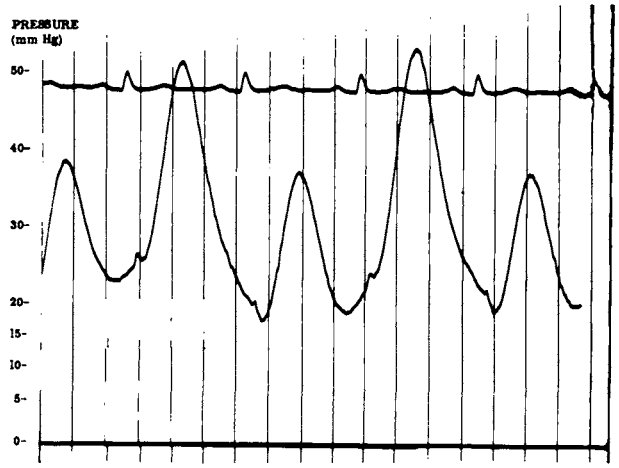

FIGURE 1. Pulmonary artery pressure tracing showing alternans pattern.

the region of the right upper lobe. The right heart catheterization revealed an end-diastolic pressure in the right ventricle of 9 mm Hg, supporting the diagnosis of right ventricular failure. A pulmonary artery tracing demonstrated the pattern of pulsus alternans (Fig 1). The respiratory rate remained stable at about 24 per minute. A carotid artery pulse tracing was normal (Fig 2). The patient was treated with intravenously administered heparin and discharged on therapy with warfarin sodium, as it was later discovered that his level of anticoagulation was intermittently suboptimal. He has remained well since discharge.

DISCUSSION

The actual physiologic basis of pulsus alternans is unknown. A rhythmic alternation of cardiac strength has been attributed to disease or fatigue of large numbers of cardiac fibers, resulting in varying excitability of refractoriness.[7,8]

It has been suggested that fibers that are activated but do not contract are potentiated toward a stronger contraction during the next cycle (a mechanism similar to that of paired pulsation).[9] Another theory explains the phenomenon of pulsus alternans on the basis of alternating ventricular end-diastolic volume and/or myocardial fiber length.[10,11]

Pulmonary embolization may have several consequences, among which are pulmonary hypertension, cardiac arrhythmia, hypoxia, and coronary vasoconstriction. These factors may lead to the development of right ventricular dilatation and failure, and, as demonstrated in the present report, may be associated with pulsus alternans of the pulmonary circuit.

Pulsus alternans, seen in the lesser circuit at cardiac catheterization, should call to mind the possibility of pulmonary embolism.

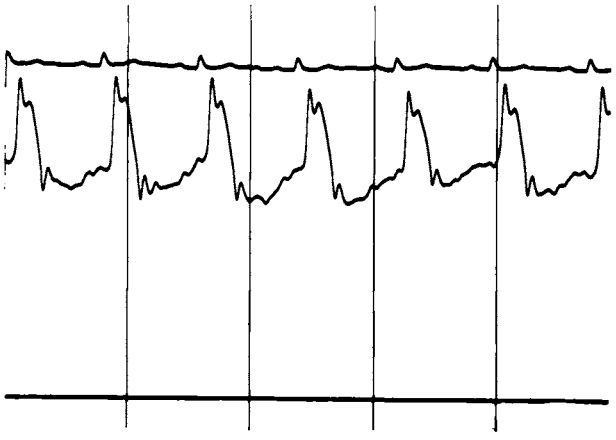

FIGURE 2. Normal carotid artery pulse tracing.

REFERENCES

1 Traube L: Ein Fall Von Pulsus bigeminus nebst Bemerkungen über die Leberschwellungen bein Klappenfehlern und über acute Leberatrophie. Klin Wochenschr 9:185, 1872
2 Gould L: Pulmonary artery alteration with atrial fibrillation. JAMA 207:1515, 1969
3 de Rabago P, Kohout FW, Katz LN: An unusual case of pulsus alternans recorded during cardiac catheterization from the pulmonary and systemic blood vessels. Am Heart J 49:472, 1955
4 Ferrer MI, Harvey RM, Cournand A, et al: Cardiocirculatory studies in pulsus alternans of the systemic and pulmonary circulations. Circulation 14:163, 1956
5 Segers M, Hendrickx J: Exploration électrokymographique d'un case de WPW intermittent. Acta Cardiol (Brux) 8:643, 1953
6 Weissel W, Salzmann VH: Pulmonary capillary arterial pressure pulse in man. Br Heart J 14:47, 1952
7 Spodick DH, St Pierre Jr: Pulsus alternans: Physiologic study by noninvasive techniques. Am Heart J 80:766, 1970
8 Noble RJ, Nutter NO, Crumly HJ Jr: The demonstration of alternating contractile style in pulsus alternans. J Clin Invest 49:1166, 1970
9 Guntheroth WG, Morgan BC, McGough GA, et al: Alternate deletion and potentiation as the cause of pulsus alternans. Am Heart J 78:669, 1969
10 Gleason WL, Braunwald E: Studies on Starling's law of the heart. Circulation 25:841, 1962
11 Mitchell JH, Sarnoff SJ, Sonnenblick EH: The dynamics of pulsus alternans: Alternating end-diastolic fiber length as a causative factor. J Clin Invest 42:55, 1963

Inhibition of a Demand Pacemaker by Electrosurgery*

John Kevin O'Donoghue, M.D.

A case of intermittent asystole due to suppression of the output of a Medtronic 5942 demand pacemaker by a Bovie electrocautery used during prostatic surgery is reported. Despite the claims of the manufacturer, the physician should be aware of this potential hazard. Recommendations are made for minimizing the incidence and risk of this complication.

*Instructor in Medicine (Cardiology), University of Chicago School of Medicine.
Reprint requests: Dr. O'Donoghue, 950 East 59th Street, Box 423, Chicago 60637

Inhibition of demand pacemakers by radio frequency waves has been recognized since the earliest noncompetitive (demand) pacemakers were implanted.[1] Electrocautery,[2] distributors of combustion engines,[3] microwave ovens[4] and radar[5] have all been known to cause temporary suppression of the unit. The manufacturers of later models have incorporated various features into the pacemaker to try to prevent this suppression: shielding the pacemaker unit by a titanium cover and incorporating a high-frequency filter into the circuit are two approaches.

In addition, in order to prevent dangerous situations at surgery, it has been recommended that the electrosurgical tip and the ground plate be localized as far as possible away from the pacemaker.[6] Thus, for prostatic surgery, the ground plate should be positioned under the left gluteal region. Constant electrocardiographic monitoring has also been advocated to demonstrate that electrosurgery of the prostate is being accomplished without pacemaker suppression.

We recently have encountered a patient who developed suppression of his demand pacemaker, despite the precautionary measures advised above.

Case Report

A 67-year-old black man was admitted first to Billings Hospital in 1969 because of central retinal artery occlusion, and was shown in addition to have complete right bundle branch block, left axis deviation (left anterior hemiblock) and sinus bradycardia (rate 45/min), with a PR interval of 0.19 sec. Despite a full medical regimen including isoproterenol hydrochloride (Proternol) and propantheline bromide therapy (Pro-Banthine), he developed an episode of syncope, and a permanent transvenous bipolar electrode was therefore inserted through a jugular vein into the right ventricle, and a Medtronic 5942 demand pacemaker placed in a subcutaneous pocket in the right anterior chest wall. Threshold for capture was 0.5 ma, and the chest x-ray film confirmed proper location of the electrode.

Six months later, the patient underwent a transurethral resection of the prostate gland. The ground of the Bovie CSV was placed under the patient's left buttock. As expected continuous ECG monitoring was impossible during electrosurgery, because of the electrical interference. On two occasions following four to five seconds of electrocutting, the patient was noted to have tonic-clonic movements of the head and arms, and the carotid pulse could not be felt at this time. A magnet was placed over the pacemaker converting it into a fixed rate unit, and no further episodes of inhibition occurred during the remainder of the operation. The patient had a good surgical result, and no sequelae from the temporary asystole.

Following surgery, the electrocardiographic monitoring equipment and the Bovie CSV were both checked for current leakage with the equipment grounded; none was found.

Discussion

Intermittent heart block,[8] the sick sinus syndrome,[9] incomplete trifascicular block,[10] and the use of permanent pacemakers in conjunction with drug therapy to suppress cardiac dysrhythmias have all resulted in an increased use of demand pacemakers. These units function by sensing and recognizing the intracardiac ventricular R wave and thereafter inhibiting the electrical output of the pacemaker.[11] Intracardiac R-wave electrograms of 1.5 mv or more will be sensed, allowing the unit to recognize premature ventricular contractions, which infrequently generate endocardial potentials less than this critical value.[3] This high sensitivity does, however, place the generator in the danger of being "fooled" by external electrical fields, especially if they are pulsed.

Placing the indifferent electrodes of the cautery under the buttocks of the patient should confine the current field to an area away from the pacemaker or endocardial electrodes, allowing electrosurgery to be carried out in safety,[2,7,12] but as occurred in this patient, induction currents of sufficient magnitude to be picked up by the electrodes and inhibit the pacemaker can unfortunately still be generated.[13]

Recently, during prostatic electrosurgery, endocardial potentials have been measured at levels of 8-16 volts with a temporary bipolar pacing electrode, which are of more than sufficient amplitude to be sensed by the pacemaker, and thus cause inhibition of its output, despite there being 30-40 cm from the surgical operation field to the pacemaker.

The electrocardiograph, being a sensitive voltmeter, will not function during the high voltages used during electrosurgery, and will, therefore, give no indication of the electrical activity of the heart during this time. An intraarterial cannula or indwelling needle should be used during electrosurgery to monitor continuously the pulse of these patients, and the surgeon should limit electrocautery to not more than 2-3 sec intervals at a time if any suppression of the pacemaker is produced. If continuous electrosurgery is necessary, a magnet should be applied over the pacemaker to convert it to a fixed rate modality by flipping the internal reed switch.

In a previous report that dealt with pacemaker suppression by electrosurgery, the authors[14] indicated that the unit failed a few weeks after surgery and considered the possible relationship between cautery and battery failure but could find no evidence for a causal relationship. Recently Schwingshackl[15] has demonstrated that partial battery depletion could occur after use of electrosurgery for 45 minutes. He recommends, therefore, using electrosurgery only when the batteries are at full capacity, advice which may be difficult to achieve under all circumstances. Demand pacemakers may be more vulnerable than fixed-rate units to high levels of electrical energy, due to their low impedance input amplifier circuitry.[16] Although the manufacturers have attempted to reduce the inherent risk of electrical interference by electrosurgery or other sources of pulsed current, this case report and others in the literature indicate that the shielding and modified circuits currently used are insufficient to protect the patient against dangerous output suppression at these times, and that further effort in this regard is urgently needed. A recent article[17] reiterates the belief that the Medtronics 5942 will convert to a fixed rate when confronted with external interference.

This is not always the case. Therefore we recommend the following:

1) The ground of the cautery should be positioned under the patient's buttocks, increasing the distance of the resultant electrical field from the pacemaker.

2) ECG monitoring may be fallacious, owing to electrical interference of the cautery, and an intraarterial cannula or indwelling needle should be used to monitor mechanical activity of the heart throughout.

3) Electrocautery should be limited to not more than bursts of 2-3 sec if any pacemaker suppression is produced, or the pacemaker should be converted to a fixed-rate modality using the magnet provided.

4) Prolonged electrocautery (45 min or longer) may drain the life of the batteries of the pacemaker, requiring earlier elective change of the unit.

5) Pacemaker manufacturers should be encouraged to develop better electrical shielding.

ACKNOWLEDGMENT: The author wishes to gratefully acknowledge the assistance and encouragement received from Dr. Leon Resnekov in the preparation of this manuscript.

References

1 Carleton RA, Sessions RW, Graettinger JG: Environmental influences on the implanted cardiac pacemaker. JAMA 190:160-162, 1964
2 Smith BR: Pacemaker malfunction from urethral electrocautery. JAMA 218:256, 1971
3 Furman S, Parker B, Krauthamer M, et al: The influence of electromagnetic environment on the performance of artificial pacemakers. Ann Thoracic Surg 6:90-95, 1968
4 King GR, Hamburger AC, Forough P, et al: Effects of microwave ovens on implanted cardiac pacemakers. JAMA 212:1213, 1970
5 Yatteau RF: Radar induced failure of a demand pacemaker. N Engl J Med 280:34, 1969
6 Fein RL: Transurethral electrocautery procedure in patients with cardiac pacemakers. JAMA 202:101-103, 1967
7 Greene LF, Meyers GH Jr, McCallister BD: Transurethral resection of the prostate in patients with cardiac pacemakers. Br J Urol 41:572-578, 1969
8 Bilitch M, Lau FVK, Cosby RS: Demand pacemaker inhibition by radio frequency signals. Circulation (Suppl 2): 35:II-68, 1967
9 Easley RM, Goldstein S: Sino atrial syncope. Am J Med 50:166-177, 1971
10 Trivino AJ, Beller BM: Conduction disturbances of the left bundle branch block system and their relationship to complete heart block. Am J Med 51:374-382, 1972
11 Sowton E, Gray K, Preston T: Electrical interference in non-competitive pacemakers. Br Heart J 32:626-632, 1970
12 Feil RL: Transurethral resection of the prostate with an in situ internal cardiac pacemaker. J Urol 97:137-139, 1967
13 Mansfield PB: On interference signals and pacemakers. Am J Med Elec 5:61-63, 1966
14 Wajszczak WJ, Mowry FM, Dugan NL: Deactivation of a demand pacemaker by transurethral electrocautery. N Engl J Med 280:34, 1969
15 Schwingshackl H, Mauser R, Amor H: Interfering influence of low frequency alternating currents on asynchronous and controled pacemaker systems during the use of electrosurgical devices. Schweiz Med Wochensch 101:46-52, 1971
16 Lau FYK, Bilitch M, Weintraub HJ: Protection of implanted pacemakers from excessive electrical energy of D.C. shock. Am J Cardiol 23:244-249, 1969
17 Greene LF, Merideth J: Transurethral operations employing high frequency electrical currents in patients with demand cardiac pacemakers. J Urol 108:446-448, 1972

Arrhythmias Associated with Hysteresis Ventricular Inhibited Pacing*

Seymour Furman, M.D., and Doris J. W. Escher, M.D.

Hysteresis rate ventricular inhibited pacing permits a longer period to elapse from a spontaneous cardiac contraction to the first pacemaker impulse, than between succeeding impulses during continuous pacing. It allows a cardiac rate, conducted or idioventricular, slower than the paced rate to inhibit the pacemaker. In the first of two patients, an increase in idioventricular rate paradoxically slowed the total cardiac rate from 71-62 during activity. In the other, false recycling from a P wave permitted an asystolic period of 1.4 seconds.

Of the variety of pacing modes, the most widely used in the past few years has been ventricular inhibited (demand/standby), designed to inhibit pacemaker function whenever spontaneous ventricular activity appears at a QRS-to-QRS interval shorter than the pulse generator escape interval (a rate greater than the pacemaker rate of emission). One of the refinements of ventricular inhibited pacing has been the development of two different intervals, one for pacing the heart, *ie*, the interval between two successive paced beats, the other and longer, the interval between a spontaneous cardiac contraction and the next paced beat. This design is called "rate hysteresis." As commonly set, continuous pacing is at an interval of 0.86 sec (rate 70 per minute) while intervals of less than 1.0 sec between spontaneous complexes keep the pacemaker inhibited. In effect, any spontaneous rate greater than 60 per minute inhibits the pacemaker.

When hysteresis rate pacing was introduced as a variation of ventricular inhibited pacing its presumed effects of allowing a slower sinus than paced rate, reduction of battery drain and consequent increase in pulse generator longevity, were cited as potential benefits. These have not been clearly demonstrated during clinical evaluation. The arrhythmias associated with this variety of ventricular inhibited pacing are described and

*From the Cardiothoracic Service, Division of Surgery and the Cardiology Service, Division of Medicine, Montefiore Hospital and Medical Center, Bronx, N.Y. Supported in part by USPHS Grant No. HE04666-13.
Reprint requests: Dr. Furman, 111 East 210th Street, Bronx 10467

Inhibition of a Demand Pacemaker by Electrosurgery

John Kevin O'Donoghue

Chest 1973;64; 664
DOI 10.1378/chest.64.5.664