Large Right Atrial Thrombi Surrounding Permanent Transvenous Pacemakers

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SUMMARY

Transvenous pacemakers offer advantages but may also lead to complications not seen with transthoracic pacemakers. We have seen two patients in whom at autopsy large right atrial thrombi were found around the catheter. The first patient had signs of increasing right heart failure and oliguria and died 48 hours after implantation of the pacemaker, which functioned normally throughout. At autopsy 80% of the right atrium was filled by a thrombus. This thrombus had caused inadequate filling of the right atrium and seemed responsible for the deterioration and death of the patient. The second patient died suddenly 2 months after insertion of the pacemaker after suprapubic prostatectomy. At autopsy a well-organized thrombus encircled the catheter between the junction of the azygos vein and the superior vena cava to the tricuspid valve. The thrombus may have caused signs of right heart failure before death. Since right atrial thrombi may be detected by angiography and treated by surgery, it is important to be aware of this rare complication of transvenous pacing.

Additional Indexing Words:
Atroventricular block Endocardial electrode

Although transvenous pacing offers many advantages over the transthoracic approach, it may lead to problems not seen with the epicardial leads. Thromboembolic complications have been rare, and there have been only two documented cases of pulmonary embolism related to the intracardiac electrode.1, 2 In the present report we record two cases with transvenous pacemakers in which large thrombi formed around the catheter and led to cardiac inflow obstruction. Awareness of this complication is important as it may be diagnosed by angiography and treated by surgery.

Report of Cases

Case 1

A 75-year-old white man was admitted because of increasing shortness of breath. During the few weeks before admission he had complained of lightheadedness and had one documented syncopal episode. He had not received digitalis or diuretics. Electrocardiograms over the previous 5 years had shown first degree A-V block. On examination he had blood pressure of 110/55 mm Hg and pulse rate of 50/min. Jugular venous distention and moderate pitting edema of the ankles were noted. A grade II/VI systolic ejection murmur was heard at the apex and at the aortic area. The electrocardiogram taken at admission showed an atrial rate of 250/min and a varying slow ventricular response at about 48/min. Four days following admission, because of persistent failure and slow ventricular rate, a permanent fixed-rate transvenous pacemaker was implanted. During the procedure the patient had a brief period of respiratory arrest which required endotracheal intubation. During this short period the pulse rate fell to 35/min and external cardiac compression was performed. Endocardiac pacing was begun immediately and the patient was returned to the recovery room in good condition. The cardiac monitor revealed pacemaker-induced QRS complexes at 74/min without competition. During the subsequent 24 hours and despite administration of digitalis and diuretics the patient developed further increase of jugular

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venous pressure, oliguria, and finally anuria. He expired 48 hours after insertion of the pacemaker, which functioned normally until death.

At postmortem examination the tip of the pacing catheter was seen to be embedded beneath the trabecular network of the right ventricle near its apex. A large recent thrombus was found around the catheter in the right atrium (fig. 1A and B). The thrombus, which obliterated approximately 80% of the right atrial cavity, extended through the tricuspid valve into the right ventricle and cephalad into the superior vena cava. The pulmonary arteries were free of thrombi or emboli. There was moderate coronary atherosclerosis with scars in the inferior wall and the apex of the heart. Severe passive congestion of the liver, ascites, and bilateral pleural effusions were noted.

The patient was in heart failure before the pacemaker was inserted. Postoperatively, despite effective pacing, digitalis and diuretics, venous pressure rose, and urine output fell. These probably resulted from poor right heart filling because of the massive thrombus in the right atrial cavity.

Case 2

A 69-year-old white man was admitted because of shortness of breath and malaise. On admission, examination of the heart revealed regular pulse of 38/min and blood pressure of 210/100 mm Hg. The jugular veins were not engorged, and there was no peripheral edema. A grade II/VI systolic ejection murmur was present over the aortic area. The admission electrocardiogram revealed Mobitz type II A-V block with an atrial rate of 76 and a ventricular rate of 38/min. Two days following admission a permanent fixed-rate transvenous pacemaker was inserted. The postoperative electrocardiogram revealed pacemaker stimuli of 72/min with a 1:1 ventricular response and no competition. The patient was discharged a week later.

**Figure 1**

A. Heart opened from the posterior aspect to show the right atrial and right ventricular cavities. The massive thrombus in the right atrium may be seen surrounding the catheter and extending through the tricuspid valve. SVC = superior vena cava; CS = coronary sinus; Th = thrombus; TV = tricuspid valve. B. Same view as A. The tricuspid ring and right atrial wall are drawn together to illustrate the obliteration of the right atrial cavity by the thrombus.
later without medication, the dyspnea and malaise having subsided. He was seen again 2 weeks later, at which time he continued to remain free of symptoms.

Two months after insertion of the transvenous pacemaker the patient was readmitted with dysuria and frequency of urination. Although he denied dyspnea at that time, physical examination revealed jugular venous distention, hepatomegaly, and moderate edema of the feet and ankles. Electrocardiogram showed pacemaker stimuli at 72/min with 1:1 ventricular response and no competition. Benign prostatic hypertrophy was diagnosed and the patient underwent suprapubic prostatectomy with good immediate postoperative course. Two days following surgery he was found dead in bed.

At autopsy the tip of the catheter was embedded beneath the trabeculae of the right ventricle near the apex. A large thrombus surrounded the catheter from the junction of the azygos vein and superior vena cava into the right atrium as far as the tricuspid valve (fig. 2). The transverse diameter of the thrombus ranged from 1.5 to 3 cm. The pulmonary arteries were free of thrombi. There was marked coronary atherosclerosis with old anterior and inferior myocardial infarcts. Bilateral pleural effusions and passive congestion of the liver were present. There was a large suprapubic hematoma under the surgical incision.

The exact mode of death in this patient is uncertain and does not appear related to the intracardiac thrombosis. Signs of right-sided congestion first detected 8 weeks after insertion of the catheter suggest that the thrombus, although smaller than in the first case, was of hemodynamic significance.

Discussion

In several large studies of cases dealing with permanent transvenous pacemakers, there were no embolic complications. However, two cases of pulmonary embolism, one associated with a prolapsed loop of the catheter into the main pulmonary artery, have been reported. Three instances of asymptomatic pulmonary embolies have also been found in a pathologic study, but they could not be attributed with certainty to the endocardial pacing catheters. Insignificant small thrombi around the catheter were seen in one of 21 autopsied cases and in another series in two of seven autopsied cases. By contrast, in our patients the thrombi were large enough to produce symptoms. In the first the thrombus obliterated most of the right atrium, and the clinical signs were consistent with severe inflow obstruction. In the second patient signs of right-sided congestion were more discrete and unrelated to his demise, but their development could be timed to the period following insertion of the pacemaker. There was no obvious explanation for the formation of these large thrombi except, possibly, for the brief period of external cardiac compression in the first patient.

Thrombi large enough to produce hemodynamic impairment can be detected by angiocardiography. This diagnostic procedure should be considered if signs of severe right heart failure develop or become worse in a patient with a normally functioning transvenous pacemaker. We hope that the present report will stimulate awareness of this rare but
dangerous complication since it can be diagnosed and corrected.

Acknowledgment
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References

Emotion and the Pulse Rate
Avicenna 10th Century A.D.
On Love
Avicenna says:
"And hereby it is possible to arrive at the identity of the beloved person, if the patient will not reveal it, such knowledge affording one means of treatment. The device whereby this may be effected is that many names should be mentioned and repeated while the finger is retained on the pulse, and when it becomes very irregular and almost ceases, one should then repeat the process. I have tried this method repeatedly, and have discovered the name of the beloved. Then, in like manner, mention the streets, dwellings, arts, crafts, families and countries, joining each one with the name of the beloved, and all the time feeling the pulse, so that when it alters on the mention of any one thing several times, you will infer from this all particulars about the beloved as regards name, appearance and occupation. We have ourselves tried this plan, and have thereby arrived at knowledge which was valuable. Then, if you can discover no cure except to unite the two in such wise as is sanctioned by religion and law, you will do this. . . ."—From Edward G. Browne: Arabian Medicine: Being the Fitzpatrick Lectures Delivered at the College of Physicians in November 1919 and November 1920. Cambridge, the University Press, 1921, p. 86.
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