J Cardiol 2006 Jun; 47(6): 301–306

Tricuspid Valve Stenosis Related to Subvalvular Adhesion of Pacemaker Lead: A Case Report

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Abstract

Endocardial pacemaker leads may cause tricuspid valve regurgitation, but only four cases of tricuspid stenosis without endocarditis have been reported. A 77-year-old woman had received three endocardial leads when aged 44, 57, and 72 years. One lead pushed up the septal leaflet of the tricuspid valve from below the valve, then adhered to the leaflet, and was positioned against the ventricular septum. Tricuspid valve stenosis and moderate regurgitation were separately detected by transthoracic echocardiography. The tricuspid valve orifice area was 0.93 cm^2 at cardiac catheterization. An excessive loop of a ventricular lead, especially a subvalvular loop, can cause opening limitation of the tricuspid valve, and the entangling of the lead in the subvalvular structures can easily induce reactive fibrosis and adhesions.

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Key Words

■**Tricuspid valve (stenosis)** ■**Pacemaker, artificial (pacemaker lead complication)**
■**Echocardiography, transthoracic, transesophageal**

INTRODUCTION

Tissue reactions at the pacemaker electrode-tissue interface can develop through the wall of the subclavian vein or the superior vena cava. Within the heart, adhesion between the tricuspid valve and an electrode may lead to tricuspid valve regurgitation. However, only six cases of tricuspid valve stenosis have been reported. Two of the cases were associated with infectious endocarditis, and two were caused by perforation of the valve leaflets by atrial or ventricular pacemaker leads. We report a case of severe tricuspid valve stenosis caused by adhesion between a ventricular electrode and lifted valvular structures.

CASE REPORT

A 77-year-old woman had a VVI pacemaker implanted in 1972, at the age of 44 years, because of complete atrioventricular block. The pulse generator was replaced, and a new ventricular lead was implanted in 1985, when the patient was aged 57 years. At that time, the original lead could not be removed and was left in situ. The insulation material of the second lead was silicone (Pacesetter AB M/N 411S). In January 2000, when the patient was aged 72 years, the second pulse generator battery had become depleted and the lead showed low impedance, indicating the need for lead replacement. A third lead (Medtronic M/N 5054 silicone) and generator were placed via the right cephalic vein. In April 2005, the patient was admitted to our

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Manuscript received August 4, 2005; revised December 12, 2005; accepted December 13, 2005

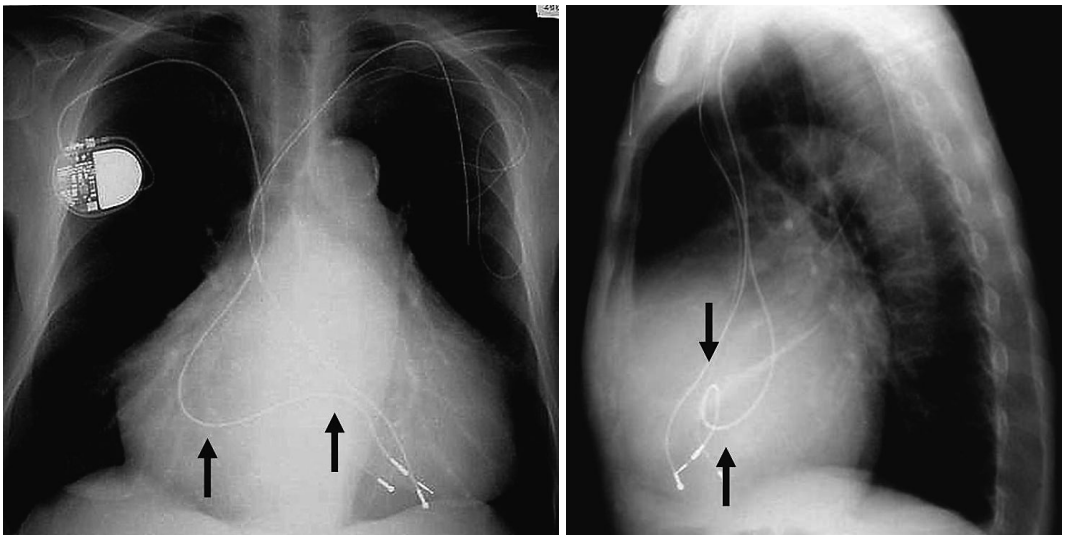

Fig. 1 Chest radiographs demonstrating three leads
The redundant lead made two loops (*arrows*) in the right atrium and the ventricle.
Left: Anterior. *Right*: Lateral.

hospital because of swelling of both lower extremities and loss of appetite. On physical examination, the pulse rate was 70 beats/min, the respiratory rate was 12 breaths/min, and blood pressure was 142/80 mmHg. Cervical veins were dilated, and the liver was palpable 3 finger-breadths below the right costal margin. A pacemaker was present in the right infraclavicular space, and old leads were present in the left infraclavicular space. A high-frequency ejection systolic murmur and a diastolic murmur at the 4th left intercostal space along the sternal margin were appreciated. Both lower extremities were edematous.

Chest radiography films demonstrated pleural effusion at the left costal-phrenic angle, and the cardiothoracic ratio was 79% (**Fig. 1**). The ventricular lead that was connected to the pacemaker passed from the superior vena cava into the right atrium with a small loop, then made a second loop under the tricuspid valve, and was positioned at the apex of the right ventricle. The two other leads had no loops but stretched across the tricuspid valve. Electrocardiography showed atrial stand still with ventricular pacing.

Transthoracic echocardiography showed an enlarged right atrium with normal-sized right and left ventricles (**Fig. 2−left**), and a small amount of pericardial effusion. In the enlarged right atrium, the three leads crossed. Continuous Doppler flowmetry of the right ventricular inflow tract demonstrated a peak diastolic flow velocity of 1 m/sec (**Fig. 2−right**). There was no stenosis or regurgitation of the mitral valve. Transesophageal echocardiography showed limited mobility of the leaflet of the tricuspid valve which appeared fixed and thickened. The lead attached to the pulse generator had lifted the septal leaflet of the tricuspid valve from the subvalvular direction (**Fig. 3**). Another lead depressed the free wall leaflet of the tricuspid valve, causing moderate tricuspid regurgitation (peak V flow velocity 2.47 m/sec). Valve stenosis and regurgitation were separately detected (**Fig. 4**).

Laboratory data were normal, except that the level of brain natriuretic peptide was 61.8 pg/m*l*. The patient received diuretic therapy and her appetite improved, but edema of the extremities persisted. She was offered surgical correction, but she refused. In November 2005, the patient was readmitted because of facial edema. She received diuretic therapy again and underwent cardiac catheterization. The mean right atrial pressure was 16 mmHg, and right ventricular systolic pressure was elevated to 44 mmHg (**Fig. 5**). The mean diastolic pressure gradient across the tricuspid valve was 8 mmHg. The thermodilution cardiac index was 4.27 *l*/min. The tricuspid valve area estimated with the Gorlin equation was 0.93 cm^2, confirming the diagnosis of tricuspid stenosis.

DISCUSSION

Tricuspid regurgitation is a well-documented

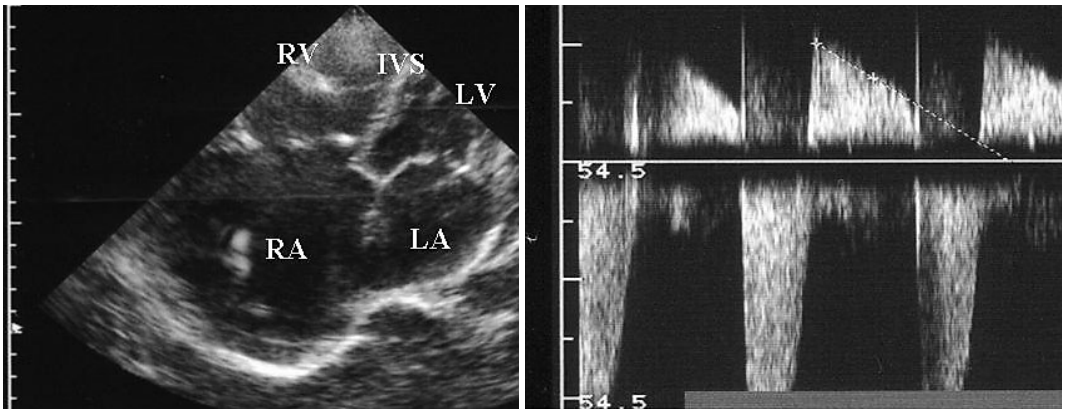

Fig. 2 Transthoracic echocardiograms
Left: Enlarged right atrium with normal right and left ventricles. Four-chamber view.
Right: Continuous Doppler flowmetry of the right ventricular inflow tract showed a peak diastolic flow velocity of 1 m/sec, and pressure half time of 232 msec.
RV = right ventricle; IVS = interventricular septum; LV = left ventricle; RA = right atrium; LA = left atrium.

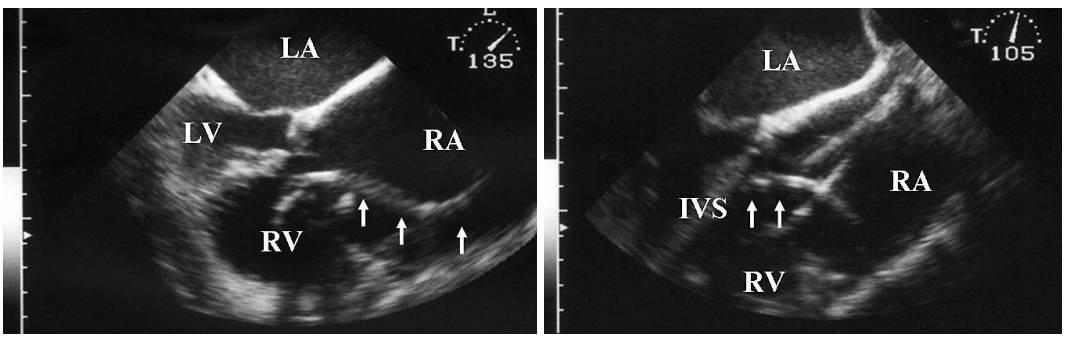

Fig. 3 Transesophageal echocardiograms
Left: The redundant lead crosses from the lateral wall of the large right atrium to the ventricular septum (*arrows*).
Right: The lead lifts the leaflet of the tricuspid valve (*arrows*) and creeps along the ventricular septum.
Abbreviations as in Fig. 2.

complication of endocardial right ventricular leads caused by adhesion and valve fixation in the open position[1,2]. However, tricuspid valve stenosis related to endocardial pacing leads is rare. Other than two cases of tricuspid valve stenosis induced by endocarditis[3,4], only four cases have been described. The first report, from 1980[5], was in an 80-year-old man who had received two endocardial leads. At surgery for tricuspid valve replacement, the anterior and posterior leaflets of the tricuspid valve were found to be fused, and infravalvular stenosis was found to have been caused by fusion of the chordae tendineae and papillary muscles. The second reported case from 1989[6], was in a 60-year-old man who had undergone VVI pacemaker implantation 14 years earlier. The single endocardial ventricular lead looped in the right ventricle adjacent to the tricuspid valve. Transthoracic echocardiography demonstrated that the leaflet of the tricuspid valve had markedly diminished mobility and appeared fixed in the closed position. The subvalvular structures appeared thickened and adherent to the endocardial pacemaker lead.

Two cases of tricuspid stenosis were associated with the perforation of a tricuspid valve leaflet by a pacemaker lead[7]. The first case was in a 46-year-old woman who had four endocardial leads. At surgery, one of the atrial leads was found to have perforated the tricuspid valve leaflets, then looped back to the interatrial septum. The massive fibrosis of the leaflets and subvalvular apparatus caused severe tricuspid stenosis. The second case was in a

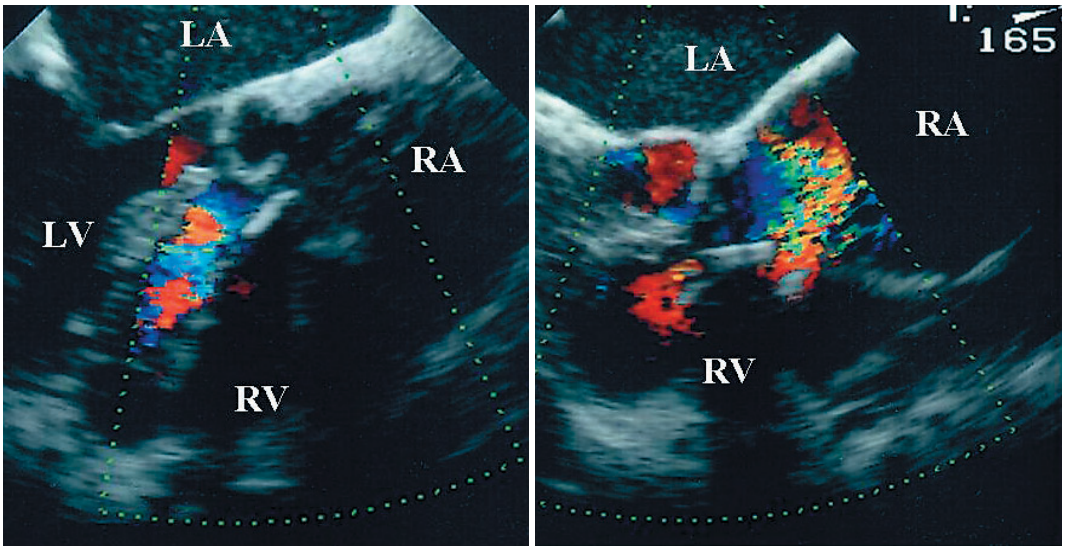

Fig. 4 Transesophageal echocardiograms
Left: The tricuspid stenosis appeared in the diastolic phase in the direction of the ventricular septum. *Right*: The tricuspid regurgitation was detected from another orifice of the valve in the systolic phase. Abbreviations as in Fig. 2.

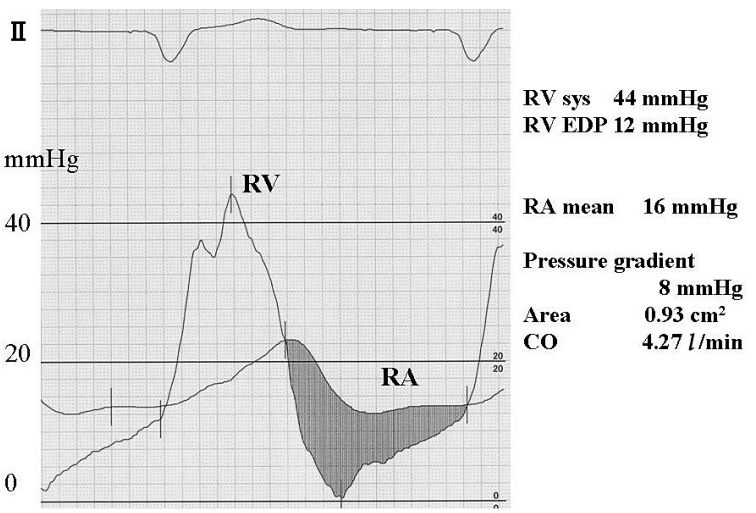

Fig. 5 Simultaneous recording of right atrial and right ventricular pressures obtained at cardiac catheterization
RV sys = right ventricular systolic pressure; RV EDP = right ventricular end-diastolic pressure; CO = cardiac output. Other abbreviations as in Fig. 2.

woman who had received a DDD pacemaker. The ventricular pacing lead had perforated the septal leaflet of the tricuspid valve. Notable fibrosis of the valve and subvalvular apparatus with severe tricuspid valve stenosis had resolved by the time of surgery. In these two cases, the endothelial injury, such as fibrosis, calcification and valvular stenosis, was believed to have been caused by perforation of a valve leaflet. However, it is difficult to perforate the leaflet during lead insertion. The course of the lead on chest radiography was not described in these two cases, but we believe that a redundant loop or subvalvular trapping of the lead can cause "whiplash injury" to the leaflet, destroying its edge, leading to inflammatory reaction, and embedding the electrode in the valve.

In our case, the tricuspid valve orifice area will be underestimated by the moderate regurgitation of tricuspid valve. However, the diastolic pressure gradient of 8 mmHg indicates tricuspid stenosis. We postulate that an excessive loop of the ventricular pacing lead lifted the tricuspid valve and adhered to the valvular structures. Additionally, opening limitation and whiplash injury to the leaflet of the tricuspid valve by the lead may have caused inflammation with endocardial fibrosis.

On post mortem analysis, the ventricular lead was found to have firmly adhered to the tricuspid

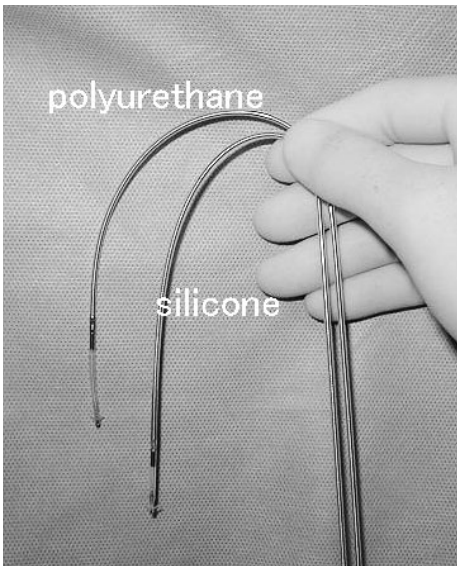

Fig. 6 Photograph showing the relative stiffness of the silicone and polyurethane electrode insulators

valve apparatus in 7 of 11 cases(64%)[8]. In 5 of these 7 cases, chordae tendineae were entrapped within a collagen sheath around the lead. Therefore, if the lead becomes entangled in the chordae tendineae, fibrotic adherence of the tissue with the lead will easily spread to the subvalvular structure. We could not determine whether the lead had trapped the chordae tendineae in our case, but transesophageal echocardiography showed the path of the ventricular lead along the ventricular septum. Infravalvular stenosis had been caused by fusion of the chordae tendineae and papillary muscles in one case[5]. We suggest that the redundant subvalvular curve of the lead will hold the tricuspid valve in a closed position and that entangling of the lead in the subvalvular structure will easily induce biological reactions between the lead insulator and the valve structures, causing various degrees of valvular stenosis by adherence to the tricuspid valve.

The relation of several ventricular leads and the tricuspid valve regurgitation are controversial[2,9]. However, we consider that the number of leads has no effect on tricuspid stenosis. The redundant subvalvular loop of single lead may play an important role in the development of this phenomenon.

The stiffness of the electrode insulator differs between leads made of silicone and those made of polyurethane(**Fig. 6**). The tension of the lead against the endocardial tissue will affect the reaction at the tissue-electrode interface. Biodegradation of the materials composing the electrode insulator may occur, such as stress cracking or fluid permeation[10,11]. However, differences in the tissue response or lead stiffness related to the composition of the electrode insulator have never been investigated. Encapsulation around the lead and adhesion to the cardiac structure were reported in 10 cases with silicone leads and one case with polyethylene[8]. However, the differences in the materials composing the lead insulator were not assessed.

In conclusion, to avoid tricuspid valve stenosis, endocardial leads should be carefully implanted with minimal redundancy to avoid entangling subvalvular structures. Additionally, tissue reactions at the lead/tissue interface and the stiffness of the composite materials of the lead insulator should be evaluated in the future.

要　　約

ペースメーカーリードの弁下部癒着による三尖弁狭窄症の1例

平　カヤノ　　鈴木明日美　　藤野　彰久
渡辺　達也　　荻生　徳寛　　芦川　紘一

心臓ペーシングリードによる三尖弁閉鎖不全の合併は比較的多くみられるが，三尖弁狭窄症はまれであり，6例の報告のうち2例は細菌性心内膜炎合併によるものである．今回我々は3本の心室内リードを有し，三尖弁狭窄をきたした症例を経験したので報告する．症例は77歳，女性．44歳時に完全房室ブロックによりVVIペースメーカー植え込み，57歳，72歳で電池交換およびリード再挿入術を受けている．両下肢のむくみと食欲不振で入院となった．心胸郭比は79%，経胸壁および経食道心エコー図法により右房の著明な拡大と少量の心　液が認められた．3本の心室リードのうち1本が右房外側から三尖弁を横切り，右室中隔を這うように接して心尖部に留置されていた．

三尖弁は弁下部よりリードに押し上げられて可動性が乏しく，高度の三尖弁狭窄と中等度の三尖弁閉鎖不全が認められた．心臓カテーテル検査では三尖弁の平均圧較差8 mmHg，弁口面積0.93 cm^2と高度の三尖弁狭窄が認められた．弁下部にリードのたわみがあることにより，リードが三尖弁を押し上げ可動性を制限し，さらにリードが弁下組織に接しているため，リード表面に生じる線維性皮膜や組織反応による癒着が起こりやすくなり，三尖弁狭窄をきたしたものと考えられた．

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