showed diffuse thickness of the left ventricular wall and severe stenosis of the hardened aortic valve, and the continuous-wave Doppler echocardiogram presented a more than 100 mm Hg systolic pressure gradient across the aortic valve. Aortography and computed tomography showed the ascending and aortic arch aneurysm of maximum 11 cm and its compression of the left main bronchus. Pulmonary arteriography revealed its compression of the right pulmonary artery with total occlusion of the upper branch, and pulmonary scintigrams presented a severe ventilation-perfusion mismatch (Fig 1).

Prior to operation, tracheal intubation and extracorporeal bypass from femoral vein to artery was performed with the patient in a half sitting position. When the patient was placed in the supine position, however, the venous drainage and arterial oxygen saturation decreased with a drop in blood pressure and pulse rate. Median sternotomy was accomplished immediately with topical head cooling, but this happened to injure the aneurysm and right ventricle that thickly adhered to the sternum because of a redo operation. Plugging the ruptured wall of the aneurysm with a finger, the venous cannula was inserted through the torn right ventricle into the superior vena cava, and a rapid core cooling was started. At the rectal temperature of 19°C, aneurysmal wall was incised and a low-porosity woven Dacron graft was anastomosed distally to the descending aorta and its continuing wall giving rise to the major three vessels of the arch under circulatory arrest for 38 min. During this time, myocardial protection was performed simultaneously using retrograde coronary perfusion without aortic cross clamp. With clamping the graft and resumption of extracorporeal circulation, the stenosed bicuspid aortic valve was replaced with a 23-mm St. Jude prosthesis and the graft was anastomosed proximally to the ascending aorta. The extracorporeal bypass was removed after repair of the right ventricle and wrapping the graft with aneurysmal wall.

Respiratory support was required for 33 postoperative days. After extubation, arterial blood gas showed PaO₂ of 78 mm Hg, PaCO₂ of 43 mm Hg, and SaO₂ of 96 percent under room air in a supine position, and breath sound was well audible over the bilateral lung fields. Postoperative aortography showed disappearance of aneurysm and good flow of three branches of the arch, and pulmonary arteriography revealed a complete release of compression of the right pulmonary artery. Pulmonary scintigrams also showed resolution of ventilation-perfusion mismatch. Pathologic findings were cystic medial necrosis of the aneurysmal wall and mucoid degeneration of the aortic valve.

Now, ten months after the operation, he is living a normal and active life with no neurologic deficit.

DISCUSSION

Because the aortic arch is in close proximity to the trachea, pulmonary artery, esophagus, and superior vena cava, its aneurysm often compresses these organs.4,5 The aneurysm in our patient, which caused severe ventilation-perfusion mismatch by compression of the left main bronchus and right pulmonary artery selectively, was very rare. Also, it was considered to be formed by poststenotic dilatation due to aortic restenosis derived from the imperfect previous commissurotomy in addition to fragility of the aortic wall that was demonstrated as cystic medial necrosis by the postoperative pathologic findings. Kampmeier noted that patients with aortic arch aneurysm had an average time duration of only 6.4 months from onset of symptoms to death, and so our patient required an urgent operation for survival because of the nine-month duration from onset to hospital admission.

This case presented a peculiar mechanism of hypoxia by large aortic arch aneurysm, and moreover, this operation was a redo operation. Therefore, a variety of additional procedures were employed and some useful suggestions were obtained from the operation. In patients with severe hypoxia due to aneurysmal compression of the trachea, pulmonary artery, or both, employment of femoro-femoral bypass prior to operation is very useful, and then a venous cannula should be inserted into the right atrium to obtain enough flow as is recently utilized for an emergency cardiopulmonary bypass support.4 In aortic arch replacement, aortic cross clamp technique under circulatory arrest with deep hypothermia and myocardial protection using retrograde coronary perfusion is preferable and useful, especially in patients with severe arteriosclerotic change of the aortic wall.

REFERENCES

Pacemaker-Induced Friction Rub and Apical Thrill*

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A patient with acute myocardial infarction developed a loud systolic sound and apical thrill. Doppler ultrasound excluded interventricular septal rupture and significant mitral or tricuspid regurgitation. Auscultatory abnormalities disappeared after removal of a temporary pacing electrode, suggesting that the friction it created with intracardiac structures was responsible for these findings.

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The use of a temporary transvenous cardiac pacemaker may be responsible for auscultatory findings which include presystolic clicks, systolic murmurs, and friction rubs. When patients with acute myocardial infarction require temporary transvenous pacing, misinterpretation of pacemaker-induced sounds can result in the erroneous diagnosis of major structural changes such as rupture of the interventricular septum or disruption of a papillary muscle. We present findings of a pacemaker-induced friction rub and an apical thrill which disappeared after pacemaker removal. Recognition of these or similar findings is crucial for the appropriate management of patients with transvenous pacemakers.

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Case Report

A 58-year-old man with an acute inferior wall myocardial infarction was treated with tissue plasminogen activator. He received a temporary right ventricular pacemaker introduced through the brachial vein for second- and third-degree atrioventricular block associated with hypotension. Two days later, his physician detected a prominent new systolic sound and arranged transfer to our hospital for treatment of suspected acute mitral regurgitation or interventricular septal rupture.

The patient was hypotensive (blood pressure: 80/54 mm Hg) but asymptomatic. There was jugular venous distension with an "a" wave visible 2 cm above the clavicle at 90 degrees. The relationship of "a" and "v" waves was normal. Cardiac examination by multiple observers revealed a distinct apical thrill. A systolic vibratory sound of medium frequency was heard over the apex, loud in sinus rhythm but almost absent in paced rhythm.

An electrocardiogram revealed demand ventricular paced rhythm with proper sensing and capture. Echocardiography revealed inferior wall hypokinesia, mild enlargement of the right ventricle with a pacemaker lead visible within the cavity, and no pericardial effusion. Doppler ultrasound showed no evidence of interventricular septal rupture and only nonsignificant mitral and tricuspid regurgitation.

Heart catheterization revealed the following pressures: right atrial mean pressure; 11 mm Hg; right ventricle pressure, 43/5 mm Hg; with end-diastolic pressure, 13 mm Hg; pulmonary artery pressure, 37/15 mm Hg; and pulmonary capillary wedge pressure, 17 mm Hg. There was no step-up in oxygen saturation in the right-sided cardiac chambers. Left ventriculography demonstrated no mitral regurgitation or ventricular septal defect.

The patient did well, and on the eighth hospital day the transvenous pacing wire was removed. The apical thrill and systolic sound vanished when the pacing wire was withdrawn. He had no symptoms on follow-up 2.5 years later. Cardiac examination was normal.

Discussion

New systolic sounds following pacemaker lead placement have been variously described as being "vibratory," "scratchy," and "squeaky," occurring in mid-to-late systole and heard best at the lower left sternal border. The mechanism of production of pacemaker-induced systolic sounds is not known. They have been attributed to tricuspid insufficiency induced by insertion of the cardiac electrode into the right ventricle and by vibration of the pacing wire in this chamber. Our findings of a systolic sound which was louder in sinus rhythm and nearly absent in paced rhythm are similar to those in four cases reported by Shirato and Ishikawa, but unlike individual case reports by Gibson et al. and Gupta and Taguchi in which the sounds were unaffected by rhythm. Shirato and Ishikawa suggested that the sound may reflect vibration of the pacemaker lead within the right ventricular cavity or an "endocardial friction rub" created by the pacing electrode sliding over the endocardial surface, as originally described by Glassman et al. The accentuation of the sound in sinus rhythm in some patients may reflect the enhanced ventricular volume and contractility resulting from atrial systole. A more forceful contraction with greater wall excursion may result in more prominent vibration of the pacemaker lead.

It is well known that myocardial perforation may complicate transvenous pacemaker placement. Perforation may be accompanied by the development of a friction rub, but a pacemaker-induced friction rub may occur in the absence of perforation. Our patient, likewise, had pacemaker-induced auscultatory findings with no evidence of myocardial perforation.

Although Gibson et al. described a pacemaker-related murmur which was classified as grade 4/6, no specific mention was made regarding the presence of a cardiac thrill. Our case is significant in that there were impressive audible and palpable findings at the same time that Doppler ultrasound revealed only insignificant tricuspid regurgitation. No previous reports have shown the absence of significant tricuspid regurgitation in patients with pacemaker-induced sounds of this nature. This unique finding lends further support to the hypothesis that auscultatory findings may be explained by vibration induced by pacemaker leads rubbing against intracardiac surfaces. The presence of a systolic sound after pacemaker placement should not be considered as sufficient evidence for the presence of tricuspid valve dysfunction, but should prompt further investigation as clinically indicated. In addition, in the setting of acute myocardial infarction requiring temporary cardiac pacing, the clinician should keep in mind that the differential diagnosis for new auscultatory findings in systole includes pacemaker-related sounds as well as disruption of intracardiac structures.

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