lower lobe pulmonary vein into the left atrium, the catheter became trapped and looped at the level of the atrial septal defect; injection of contrast material revealed its tip to be floating freely in the left atrium (Fig 1). Attempts at extraction involved the use of guide wires in an effort to straighten the catheter, pressure injections of saline solutions to dislodge the trapped portion, and any nitrate inhalation to relax possible vascular spasm associated with the entrapment. Simple traction on the catheter was without result, and produced considerable chest pain. Because the exact site and cause of entrapment was unknown, the catheter was left in place, and connected to a rapidly flowing heparinized saline infusion in order to prevent thrombus formation within the catheter. Continuous overnight traction was applied to the catheter by means of a specially designed weighted pulley system, and was of no avail. On the following day, another attempt to remove the catheter under fluoroscopic control was unsuccessful.

As the hemodynamic data indicated a significant left-to-right shunt, and as the catheter could not be extracted, thoracotomy was performed on the third post-catheterization day.® With the patient under full cardiopulmonary bypass, right atriotomy was made. The tip of the Courmand catheter was discovered to be curled upon itself and caught in the strands of a Chiari network in the atrial septum (Fig 2). The Chiari strands, together with the catheter, were excised and the atrial septal defect closed by direct suture. There were no complications.

Discussion

Chiari's networks are fenestrated membranes in the right atrium, resulting from incomplete resorption of the valves of the inferior vena cava (Eustachian) and coronary sinus (Thesenian). Such membranes are occasionally the site of thrombus formation, thereby providing a potential source of pulmonary emboli. Usual murmurs may also result from these anomalous bands. The fibers of the network have wide attachments, either to the crista terminalis of the right atrium or to the interatrial septum. In the presence of an atrial septal defect, the network might therefore be found anywhere within the defect, thereby representing a potential difficulty, as well as a hazard to transseptal catheterization.

*Surgery performed by Dr. Frank Gerbode.

In our patient, such a web did indeed lead to difficulty in crossing the interatrial defect, a procedure usually performed with ease.

The presence of a Chiari network was not initially suspected as the cause of catheter entrapment. As injection of contrast material demonstrated that the tip of the Courmand catheter was free in the left atrium, serious consideration was given to the possibility of a catheter defect. Cracking of a cardiac catheter might conceivably extricate it by catching a portion of tissue. If the tissue obstructs the crack, injection of contrast material might fail to visualize the defect. Catheter defects as a cause of complications of intracardiac catheterization were not reported in a recent cooperative study involving over 12,000 patients. However, such a defect has been known to cause entrapment in a coronary artery, necessitating thoracotomy for its removal (Dr. Abraham Rudolph, personal communication). However unusual, the possibility of a faulty catheter must be borne in mind, as attempts to extricate it are potentially hazardous in that tissue might be avulsed.

Early thoracotomy is warranted in all cases of catheter entrapment not responding to routine maneuvers.

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Ventricular Demand Pacemaker

Inhibition by An Atrial Fixed Rate Pacemaker

KASSANOFF, SYMBAS AND WENGER

[Image of pacemaker]

In an attempt to improve cardiac function, a pacemaker is often used to stimulate the heart. The ventricular demand pacemaker is activated only when the atrial rate is too slow. This pacemaker is designed to maintain a fixed rate, ensuring a consistent heartbeat. The pacemaker's effectiveness can be monitored through various means, including ECG recordings and pacemaker interrogation.

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VENTRICULAR DEMAND PACEMAKER INHIBITION

Pacemaker function may be suppressed or interfered with by electrical fields from external sources such as microwave ovens, television sets, and radio stations. Recent reports have also emphasized intrinsic inactivation; for example, a failing implanted permanent pacemaker inhibited a temporary demand pacemaker and an atrial P wave inhibited a transvenous pacemaker in the coronary sinus. Recently we observed inhibition of an epicardial ventricular demand pacemaker by an epicardial atrial fixed rate pacemaker. This inhibition was evident during periods of atrioventricular block.

CASE REPORT

A 56-year-old woman had heart disease of unknown cause for approximately five years, manifested by congestive heart failure, sinus bradycardia (as slow as 40 beats/minute), intermittent atrioventricular Wenckebach phenomenon, angina pectoris, and exertional syncope. In December, 1969, bradyarrhythmias occurred more frequently in association with the above symptoms, and a ventricular demand pacemaker was thought indicated. Temporary ventricular demand pacing at 70-80 beats/minute by a transvenous pacemaker catheter did not prevent congestive heart failure; physical activity was accompanied by shortness of breath, a ventricular diastolic gallop, and neck vein distention. Because of these findings despite ventricular pacing, in February, 1970, an atrial fixed rate pacemaker was implanted on the epicardial surface of the left atrium to afford an "atrial kick."

A ventricular demand pacemaker was implanted on the epicardial surface of the left ventricle because of the possible progression to complete atrioventricular block. The atrial pacemaker was set at 94 beats/minute to sustain complete capture. The patient responded well to atrial pacing without recurrence of the prior symptoms of angina pectoris, syncope, or congestive heart failure. In May, 1970, adequate ventricular demand pacemaker function was documented during a ten-hour magnetic tape electrocardiographic recording (Fig. 1). The ventricular demand pacemaker functioned when atrial pacing impulses were not conducted to the ventricle.

In August, 1970, seven months after implantation of the two pacemakers, routine ECG showed an increase in the atrial pacing rate from 94 beats/minute at implantation to 105 beats/minute. This increased rate was considered an indication of impending battery failure of the atrial pacemaker, and the atrial fixed rate pacemaker battery was replaced. At the time of battery replacement, the atrial fixed rate pacemaker was again set at 94 beats/minute, but it was necessary to increase the amperage to 6 ma from 1.5 ma to sustain complete capture.

In December, 1970, a ten-hour magnetic tape recording showed several instances of the failure of conduction of the atrial pacing impulses to the ventricle (Fig 2). During this episode of "atrial pacemaker-ventricular block," the atrial fixed rate pacemaker continued to fire at 94 beats/minute. The ventricular demand pacemaker, set to fire at 78 beats/minute, failed to function even when the RR interval was 1.2 sec, equivalent to a ventricular rate of 50 beats/minute. It was thought either that the ventricular demand pacemaker battery had failed, or that the increased amperage of the atrial fixed rate pacemaker was ceased by the ventricular demand unit, which was suppressed by this signal despite the failure of atrioventricular conduction. To differentiate between these possibilities, the atrial fixed rate pacemaker was turned off percutaneously to determine if the ventricular demand pacemaker would function. This indeed occurred, supporting the thesis that the high milliamperage of the atrial fixed rate pacemaker had suppressed the ventricular demand pacemaker (Fig 3).

We decided to manage the patient with only the ventricular demand pacemaker. The atrial fixed rate pacemaker was left in place, turned off, and the patient now tolerated the intermittent absence of an "atrial kick." Her basic cardiac rhythm was a sinus mechanism, with rare periods of atrioventricular Wenckebach.

Three months later, in March, 1971, the rate of the ventricular demand pacemaker slowed from 78 to 50 beats/minute. This slower rate was thought to represent battery failure, and the ventricular demand pacemaker battery was replaced without incident. She subsequently had several episodes of angina pectoris and dizziness, associated with intermittent sinus bradycardia and nodal bigeminy (Fig. 4). The nodal premature beats suppressed the ventricular demand pacemaker, but apparently did not afford adequate systemic, cerebral, or cardiac perfusion; these premature beats did not produce a palpable pulse in the radial artery.

The ventricular demand pacemaker rate was increased to 94 beats/minute, the rate at which the atrial fixed rate pacemaker had previously functioned. The patient has since remained asymptomatic with the atrial fixed rate pacemaker turned off and only the ventricular demand pacemaker operative.

*Medtronic units 5870 and 5841—atrial fixed rate pacemaker and ventricular demand pacemaker respectively.

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**Figure 1.** Atrial pacing at 94/minute. Functioning ventricular demand pacemaker at 78/minute.

**Figure 2.** Atrial pacing impulse at 94/minute, with intermittent failure of conduction of the atrial pacing impulse to the ventricle. Ventricular demand pacemaker not functioning.

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Simultaneous Excision of Bilateral Neurogenic Tumors of the Mediastinum

Raymond G. Armstrong, Lt Col, USAF (MC), FCCP, Evan F. Lindberg, Col, USAF (MC); George Tresler, Maj, USAF (MC); William Stanford, Col, USAF (MC); and Byron N. Dooley, M.D.*

Bilateral adjacent neurogenic tumors of the mediastinum were excised in a 20-year-old man through a single left thoracotomy. The presence of bilateral lesions was indicated by the discrepancy between excised tumor and the mass on x-ray examination. Two distinct histologic patterns resulted in one mass representing a ganglieneuroma, and the second, a neurofibroma.

In most reported series of mediastinal masses1,2 the incidence of neurogenic tumor is between 25 and 30 percent and represents the most common mediastinal tumor. In spite of the bilateral nature of neural tissue from which these tumors could arise, cases of multiple neurogenic tumors occurring bilaterally and simultaneously have rarely been reported.1,3 In contrast, the large tumor extending across the midline from one thoracic cavity to the other has been frequently described.

The case to be reported illustrates one possible approach to this unique situation.

CASE REPORT

An asymptomatic 20-year-old man was referred to Wilford Hall USAF Medical Center in February, 1969 because of a mediastinal radiodensity found on a routine chest film. The physical examination was negative except for multiple cafe-

*From the Thoracic Surgery Service, Department of Surgery, Wilford Hall USAF Medical Center (AFSC), Lackland AFB, Texas.

Reprint requests: Lt Col Armstrong, Wilford Hall USAF Medical Center (SGHST), Lackland AFB, Tex 79136.

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