Pulmonary Embolism Complicating Permanent Cardiac Pacing

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SUMMARY

Pulmonary embolism arising from intracardiac electrodes in permanently-paced patients, is fortunately rare. The serious nature of the complication is illustrated by a case report.

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Since the introduction of artificial cardiac pacing many different complications of this procedure have been reported. The initial problems of a mechanical and electronic nature, such as lead fracture and premature failure, have to a large extent been overcome by improvement in the design of the pacing systems, and the more interesting complications are those related to disturbances of rhythm caused by electronic and other interferences.[1-5]

In view of the tremendous amount of permanent transvenous pacing in use today (about 33 000 units are sold per month by American and Canadian manufacturers[6]) it is remarkable how infrequently serious pulmonary embolism arising from thrombosis on the endocardial lead, is reported.[7-11]

In the case reported here this complication was present and contributed significantly to the patient's death.

CASE REPORT

A 65-year-old woman was admitted to hospital because of Stokes-Adams attacks experienced with increasing frequency over the previous 3 years. For 8 years she had been aware of intermittent episodes of slow or fast heart rates. Ten years previously she had undergone an abdominal hysterectomy, which was complicated by pulmonary embolism. The clinical examination, apart from arrhythmias and marked obesity, revealed no pertinent findings. Urinalysis was normal. Continuous electrocardiographic monitoring showed the bradytachycardia syndrome[12] with changes between normal sinus rhythm, sinus bradycardia, ectopic atrial rhythm, nodal rhythm with 1:1 retrograde conduction and 2:1 retrograde block, escape-capture bigeminy, sinus arrest, ventricular ectopic beats, atrial flutter and fibrillation, and ventricular fibrillation. After electrical ventricular defibrillation and temporary transvenous pacing, a stable ventricular rhythm was established and the implantation of a permanent unipolar pacing system was made. She experienced an episode of electrode displacement on the 10th day after implantation which was rectified by repositioning the electrode. She then developed fast atrial fibrillation which was controlled with digitalis and verapamil.

Three months later she was admitted in a lethargic state with diabetes mellitus, established for the first time. The pacemaker was functioning normally. On the 10th day she developed pulmonary embolism, confirmed by radioisotope scanning. On the 20th day her lower abdomen and her legs suddenly became blueish-white, the femoral pulses were absent, urination ceased, and she died.

At postmortem examination the aorta showed moderate atherosclerosis, and proximal to the renal arteries there was a big atheromatous plaque with fresh thrombus extending into both renal arteries; and an atheromatous ulcer just distal to the renal arteries. Both coronary arteries were atheromatous and calcified, and narrowed by about 50 - 75%.

The intracardiac part of the pacing electrode was covered in an endothelialised sheath and the tip was firmly attached to the septum near the right ventricular apex. There was an organised thrombus, 1,5 by 3 cm, in the right atrial appendage, attached by a thin strand to the fibrous sheath covering the electrode at its entrance from superior vena cava into the right atrium (Fig. 1). There was also a smaller, organised thrombus attached to the electrode in the right ventricle (Fig. 2). Smaller, organised mural thrombi were present in the left atrium. A big embolus was situated in the right branch of the pulmonary artery and there was extensive infarction of the right lung.

DISCUSSION

After the introduction of pacemaking catheters into the heart, they become covered with a thin layer of fibrous material within a remarkably short period of time.[13]

Thrombosis on the wall of the chamber and on the surface of the catheter is a known reaction and, apparently contributes to the eventual formation of the encapsulating fibrous sheath or so-called neo-endocardium.[14]

In view of the fact that thrombosis may even occur on autologous fibrous strands as reported by Paloheimo,[15] it appears all the more remarkable that serious thrombotic complications of pacemaking catheters are not more common. Paloheimo's case had sinus rhythm, and stasis of blood could thus not be implicated in the formation of the thrombus, whereas in many paced patients, especially those

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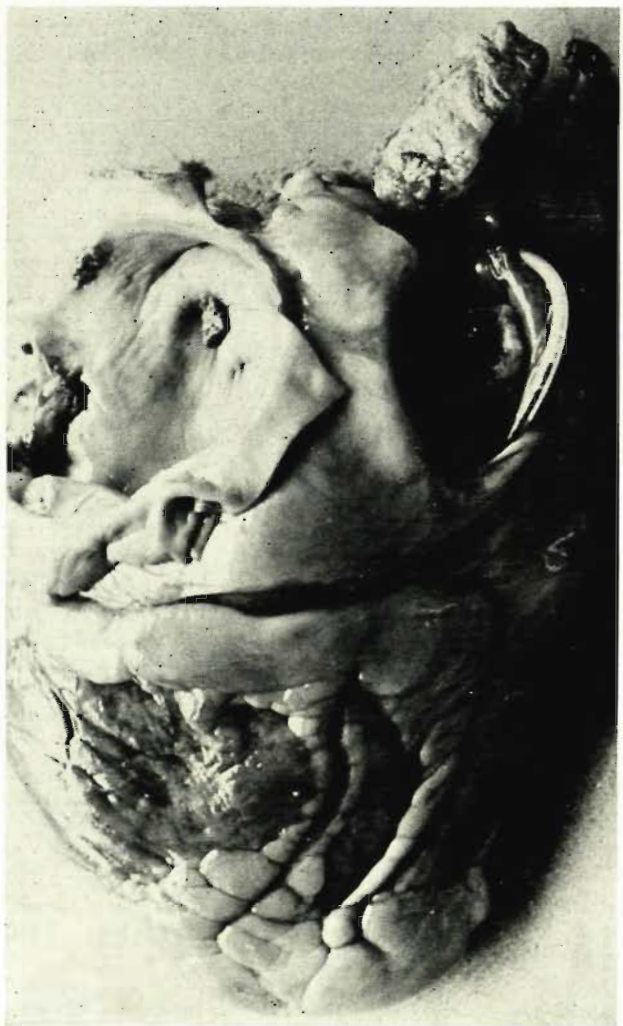

Fig. 1. Posterior view of the opened atria, showing the mural left atrial thrombi and the big thrombus removed from the right atrium.

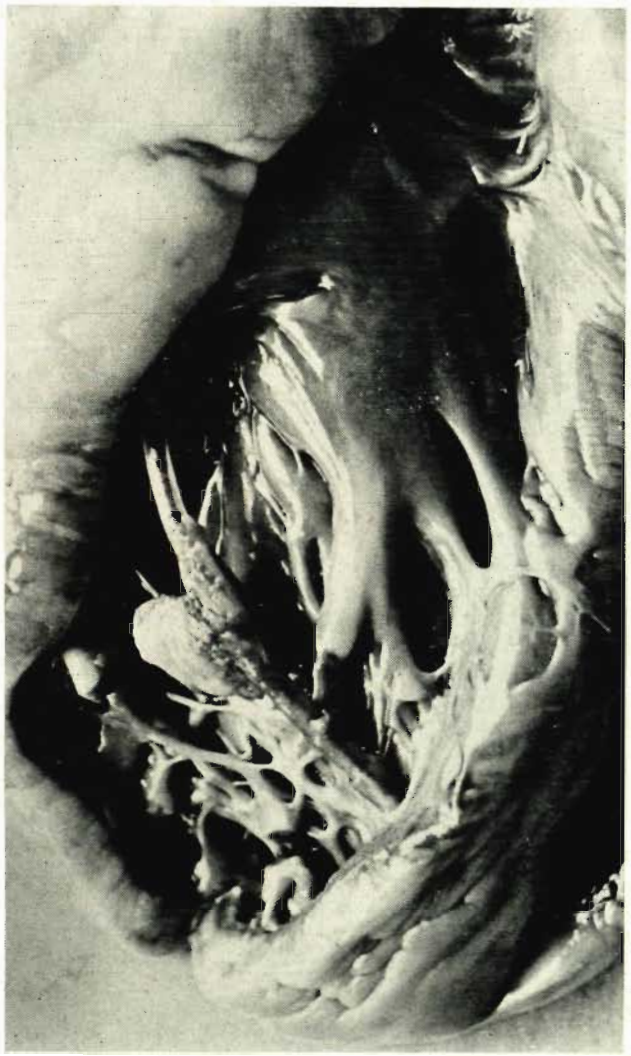

Fig. 2. Thrombus attached to the intraventricular pacing lead.

in whom the bradytachycardia syndrome is the indication for pacing, abnormal atrial rhythms do exist and can, conceivably, contribute significantly to the formation of thrombosis. It appears that the presence of an abnormally situated strand in the heart is important in the formation of a thrombus, and not only the presence of foreign material as represented by the pacemaker lead.

The question of anticoagulant therapy for paced patients arises, but this appears not to offer any advantage,[16,17] and when the reported incidence of complicating pulmonary embolism is compared with the number of patients paced, this conclusion appears to be highly valid.

Another relevant factor in the formation of thrombosis could be the polarity of the intracardiac electrode, for it is stated that the anode is inclined to attract the negatively charged blood elements and so contribute to the formation of a thrombus. However, this does not appear to be important in the case of unipolar endocardial stimulation,[17] though it might contribute to a rising threshold of stimulation in the presence of bipolar endocardial electrodes. In this case it was not a factor of importance, as the thrombi were formed on the unipolar catheter electrode some distance from its tip, i.e. in the right atrium and the right ventricular inflow tract. Additionally, there were small mural thrombi in the left atrium and it is even more probable that the varying atrial rhythm (predominantly atrial fibrillation) could be blamed for the formation of thrombi in 3 of the cardiac chambers.

Small thrombi have been noted in the left ventricles of paced patients[18] and although the authors deny any relationship, these thrombi may represent some mechanical or electrical reaction to stimulation on the right ventricular surface of the septum, which is, however, not relevant to the present case.

In conclusion, it is suggested that pulmonary embolism in a paced patient should always raise the suspicion that

thrombosis on the pacing electrode is the origin of any pulmonary emboli. Angiography of the veins and cardiac chambers traversed by the electrode may reveal filling defects, but the treatment of such cases presents problems because any attempt at removal of these electrodes, other than by open heart surgery, is almost certain to dislodge further emboli. Even if the electrode is successfully removed without surgery and without dislodging thrombi, the formed sheath and thrombus will remain to threaten the pulmonary arterial system of the patient.

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