

Case Report

Dual-Chamber Pacemaker Malfunction Mimicking Atrial Capture by the Ventricular Electrode

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A 78-year-old man came for a scheduled check up one month after the implantation of a permanent dual-chamber pacemaker for symptomatic transient atrioventricular conduction disturbances (intermittent Mobitz II atrioventricular block). The patient's ECG indicated a loss of atrial capture by the atrial electrode with 1:1 atrioventricular conduction. A subsequent X-ray examination showed that both electrodes were well placed and in their correct sites. A detailed check of the pacemaker using the programmer, together with an echocardiographic examination, revealed the true nature of the malfunction: there was a significant delay between the atrial capture and atrial depolarisation and systole, as well as a loss of ventricular capture because of an acute increase in the threshold. Normal pacemaker function and pacing ECG were restored through modification of the pacemaker's functional parameters.

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Implantation of a permanent pacemaker for the treatment of symptomatic or life-threatening bradyarrhythmias is an invasive procedure that can often lead to problems, such as periprocedural complications, or findings of pacemaker malfunction during the early or late stage of follow up. As a rule, malfunction of the pacing system is apparent on the surface ECG, especially after the application of a magnet with the consequent unsynchronised (DOO) pacemaker function at the levels of the predetermined magnetic frequency. In the case of pacemaker malfunction, a detailed check should be carried out immediately using the pacemaker's programmer in order to establish the precise nature and extent of the problem, and to determine whether the malfunction can be handled effectively by modification of the parameters of the pacemaker device or whether a new intervention is needed to correct the problem.

Here we present the case of a patient with a pacemaker malfunction, where the

initial ECG evaluation gave a completely wrong impression of a severe malfunction requiring invasive treatment.

Case presentation

A 78-year-old man, with a history of hypertension under treatment with angiotensin-converting enzyme inhibitors, underwent an uncomplicated procedure for the implantation of a permanent DDD pacemaker (Vitatron C70A4) because of occasional episodes of sudden dizziness that were attributed to atrioventricular conduction disturbances, with bifascicular block on the resting ECG (Figure 1A) and intermittent Mobitz atrioventricular block on 24-hour Holter monitoring. The atrial electrode was placed in the auricle of the right atrium and the ventricular electrode at the right ventricular apex, both with passive fixation, with absolutely satisfactory sensing and capture parameters (atrial electrode: A-wave 1.5 mV, pacing threshold 0.4 V / 0.4 ms; ventricular elec-

trode: R-wave 8 mV, pacing threshold 0.6 V / 0.4 ms). The pacemaker was programmed as follows: DDD 60/130 beats/min; unipolar pacing electrodes, output 3.5 V / 0.4 ms; bipolar atrial/ventricular sensing 0.25/2 mV, respectively; sensed/paced atrioventricular delay 160/180 ms, respectively. The patient remained free of symptoms during the first month after the procedure, at the end of which he returned for a programmed pacemaker check.

At that time, the resting ECG showed pacemaker malfunction, where the initial finding was a lack of atrial capture by the atrial electrode and atrial capture by the ventricular electrode with 1:1 atrioventricular conduction (Figure 1B). Application of a magnet confirmed the presence of atrial capture, but cast doubt on the initial impression that its source was the ventricular electrode (Figure 1C). The patient was admitted to our clinic for further examinations and treatment. The chest X-ray, anteroposterior and lateral, showed that both electrodes were correctly

placed in their respective cardiac cavities (Figure 2).

A pacemaker check was then performed using the programmer, giving the following findings. Atrial pacing (AAI mode) at various rates demonstrated normal atrial capture by the atrial electrode, but with an extremely long delay (up to 200 ms) between the appearance of the atrial spike and the P-wave on the ECG (Figure 3A). Ventricular pacing (VVI mode) showed normal ventricular capture, but with a high threshold (4 V / 0.4 ms), slightly higher than the programmed output of the ventricular electrode, which was 3.5 V / 0.4 ms (Figure 3B). Also, DDD pacing with a high ventricular electrode output showed ventricular capture and an apparent loss of atrial capture, possibly through fusion of the P and QRS waves (Figure 3C).

Otherwise, the atrial pacing threshold was entirely satisfactory (0.5 V / 0.4 ms), while atrial and ventricular sensing parameters were within acceptable limits (A-wave 0.4 mV, R-wave 4 mV), while the impedances measured for both electrodes were normal. In light of

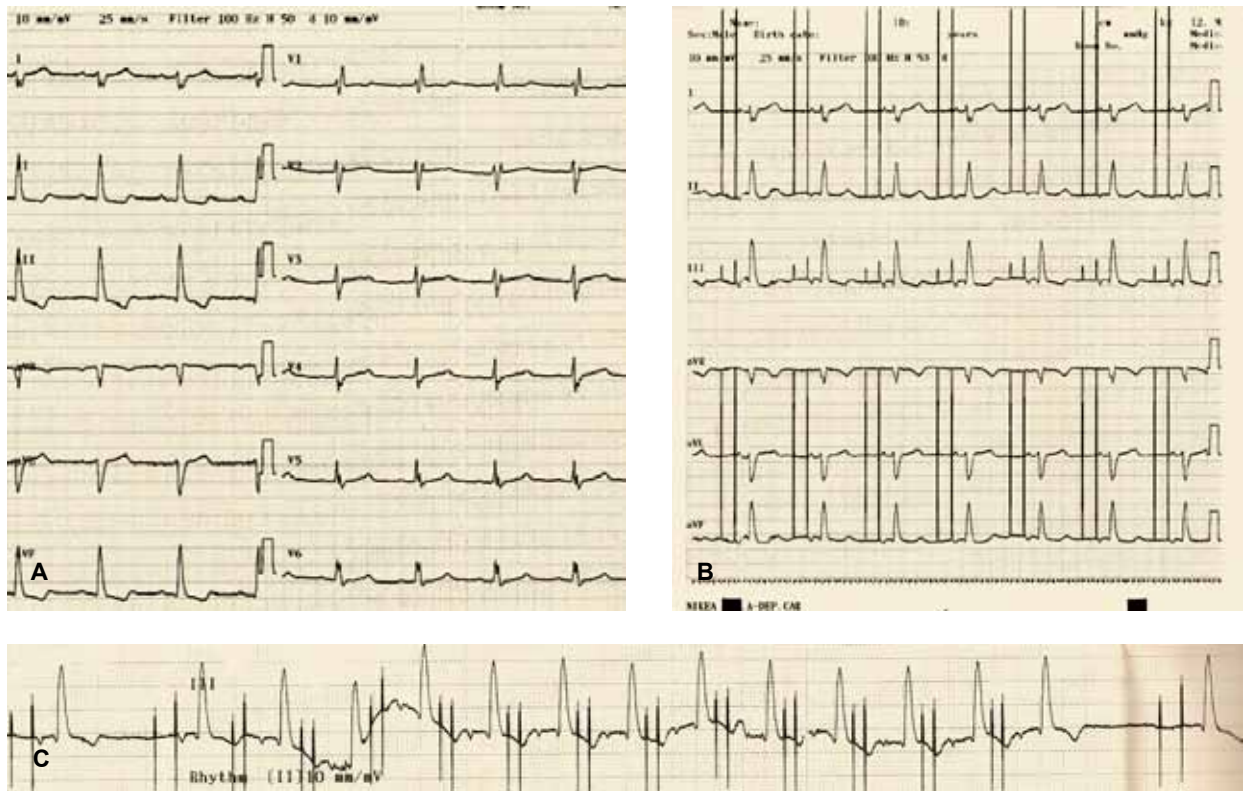


Figure 1. A. Resting ECG before pacemaker implantation, showing bifascicular block (right bundle branch block and left posterior hemiblock). B. Pacing ECG one month after implantation of a dual-chamber pacemaker. The initial (but erroneous) interpretation was a loss of atrial capture by the atrial electrode and atrial capture by the ventricular electrode with 1:1 atrioventricular conduction. C. Pacing ECG with the application of a magnet. Atrial capture is confirmed (intrinsic QRS rate equal to paced rate). Shortening of the atrioventricular delay during magnet application was associated with separation of the produced P-wave from the atrial spike, while the distance between the atrial spike and the P-wave remained constant. From the findings so far it is not clear whether the atrial capture is caused by the atrial or the ventricular spike.

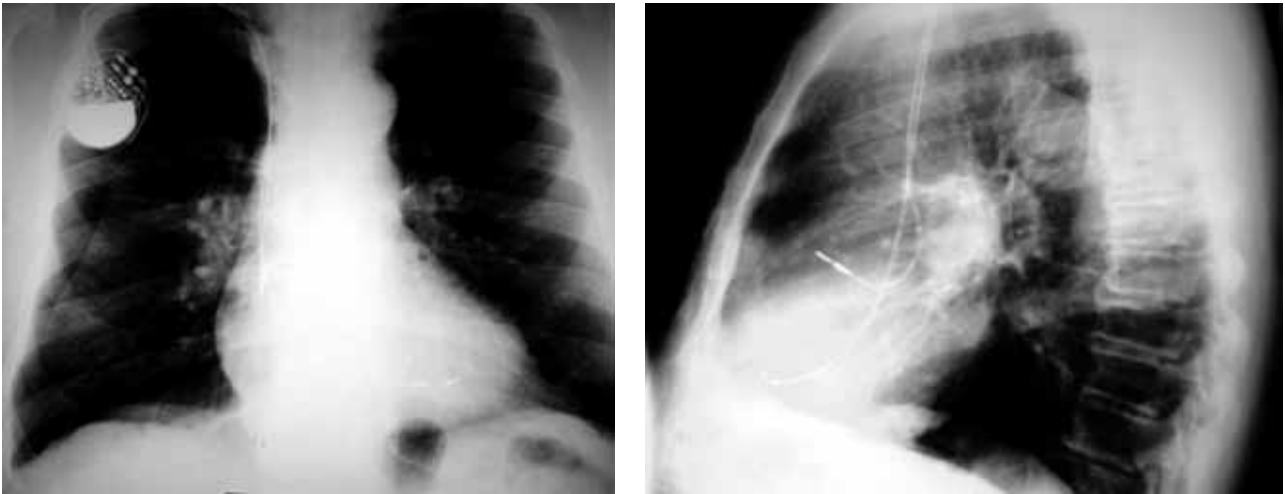


Figure 2. Chest X-ray, anteroposterior and lateral, showing the normal position of the two electrodes.

these data, the ECG picture of pacemaker malfunction had an entirely different interpretation from the obvious, but completely erroneous initial impression: the only real capture of the atrium was by the atrial electrode, but the timing of the ECG phenomena falsely suggested capture by the ventricular electrode.

The patient underwent a full echocardiographic study. The two-dimensional study was within normal limits. Then, a pacemaker check was performed simultaneously with spectral Doppler evaluation of the timing of the electrical versus mechanical components of atrioventricular depolarisation. Various pac-

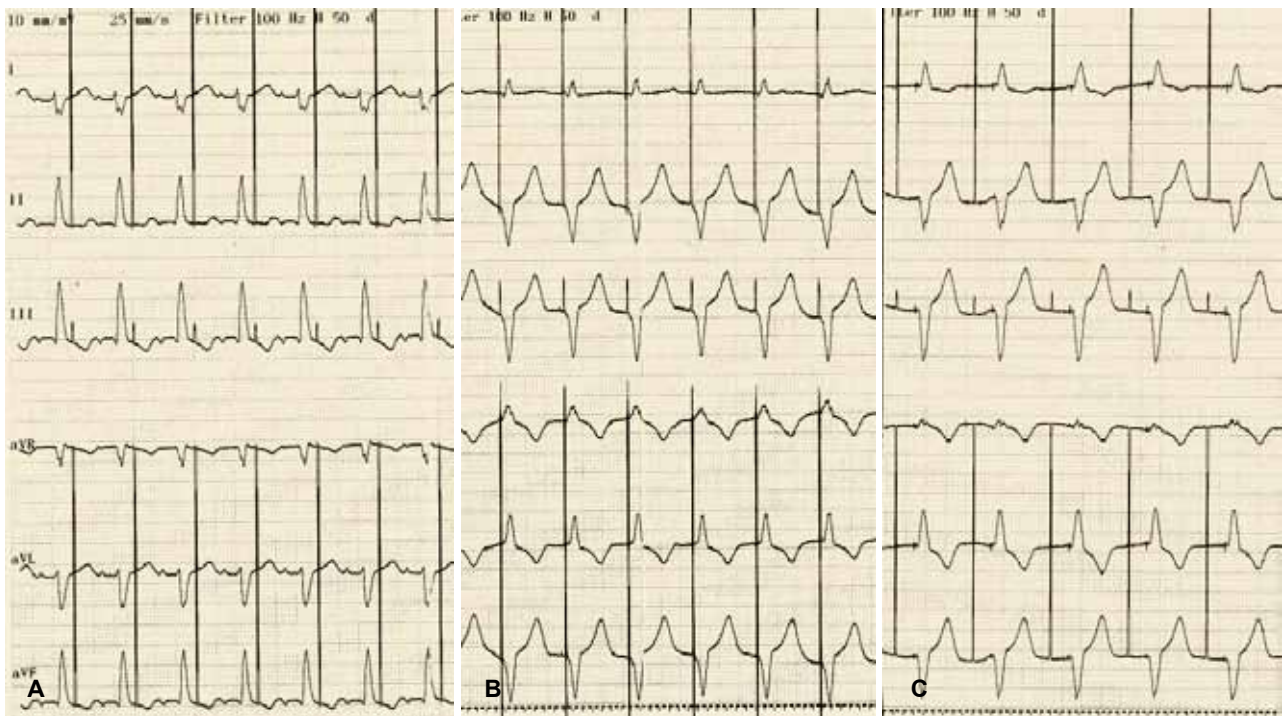


Figure 3. A. Pacing ECG under AAI mode, confirming the presence of atrial capture by the atrial electrode, with a very long delay between the appearance of the atrial spike and the P-wave, around 200 ms. B. Pacing ECG under VVI mode with high ventricular electrode output in order to achieve ventricular capture. C. Pacing ECG under DDD mode with an atrioventricular delay of 180 ms, also with high ventricular electrode output. Based on the above it is likely that the P-waves have fused with the QRS.

ing modes with different atrioventricular delays were investigated and it was found that the mechanical phenomenon of atrial systole faithfully followed the appearance of the P-wave (intrinsic or paced), while there were also findings of normal or abnormal timing of the atrial in relation to the ventricular systole that were absolutely predictable from the patient's ECG in each case (Figure 4A-F). In addition, imaging of the A-wave of mitral or tricuspid flow documented the practically simultaneous systole of the left and right atria under atrial pacing (Figure 4G, H). The latter finding showed that the material delay of the stimulus did not arise during its interatrial conduction, but rather around the electrode implantation site.

After the diagnostic examinations were completed, the pacemaker was programmed in VDD mode with ventricular electrode output 7 V / 0.4 ms, at a basic rate of 60 beats/min with hysteresis 50 beats/min and atrioventricular delay 80 ms, in order to ensure as far as possible appropriate atrioventricular synchrony. The patient was given corticosteroids *per os* and on re-examination one week later there was an improvement in the ventricular electrode parameters (pacing threshold 3.5 V / 0.4 ms, V-wave 5.5 mV), while for the atrial electrode the values of the spike-to-P-wave interval (around 200 ms) during atrial pacing and the A-wave (0.4 mv) remained stable.

Discussion

In patients who have a permanent cardiac pacemaker, the resting ECG before and after the application of a magnet provides important information about the two basic functions of the electrodes (sensing and capture) and allows a swift general evaluation of pacemaker function. In the case of findings of pacemaker malfunction, the assessment of whether safe pacemaker function is possible or not requires, first, a detailed check of the data from the pacing system (generator, electrodes) with a suitable programmer, and second, sufficient understanding of the interactions between the system and the electrical phenomena of cardiac function. The chest X-ray is often judged essential for the determination of the precise positions of the electrodes in their respective cardiac cavities, sometimes supplemented by echocardiography or a computed tomographic scan of the chest when there is suspicion that an electrode may have perforated the myocardium.¹

Disturbances of intra- or interatrial conduction

are often observed in patients with a pathological atrial myocardium (e.g. dilation, fibrosis, ischaemia, hypertrophy, taking of antiarrhythmic drugs, previous myocarditis) and are related with the probability of occurrence of atrial fibrillation, while in patients with a permanent pacemaker they are related to the site of the atrial electrode (site of stimulus production).²⁻⁶ These disturbances are manifested electrocardiographically as an increase in the height and/or duration of the two components of the P-wave, which indicate the depolarisation of the right and left atria.

As regards the pathology of stimulus transmission from its source, the stages (or degrees) of sinoatrial block are well known, from a delay to the complete inability of the stimulus to exit from the tissue that surrounds the sinus node. In the case described here, the electrical stimulus from the tip of the atrial electrode within the auricle of the right atrium, recorded as a spike on the surface ECG, together with the subsequent delayed onset of the P-wave, demarcate the exit block of the pacing stimulus, which corresponds to first degree sinoatrial block. The duration of both the intrinsic and the paced P-wave were within normal limits, giving a picture of normal interatrial stimulus conduction. Based on the above, one would expect to find normal timing of the electrical depolarisation in relation to the mechanical systole of the atria, as was confirmed in practice by the echocardiographic study. Also, a corresponding exit block was found, in the form of delayed sensing by the pacemaker of the intrinsic sinus depolarisation. Given the ventricular electrode capture, this entry-exit block in atrial electrode function can create a false picture of a loss of atrial capture under conditions of atrial and ventricular pacing (A pace – V pace, Figure 3C). Clinically, the provoked desynchronisation between atrial and ventricular systole can lead to the manifestation of pacemaker syndrome. In the theoretical case of entry-exit block by way of second degree sinoatrial block, the ECG picture would probably be even more complicated and uninterpretable.

The ECG picture also becomes more complex when the above findings are combined with a loss of capture by the ventricular electrode, as in the case described here, where the significant delay in the exit of the atrial stimulus (spike-to-P-wave interval about 200 ms) happens to be almost identical to the programmed paced atrioventricular delay (180 ms), so that the onset of the delayed paced P consistently coincides with the appearance of the inappropriate ventricular spike, creating a paradoxical and extremely unusual ECG

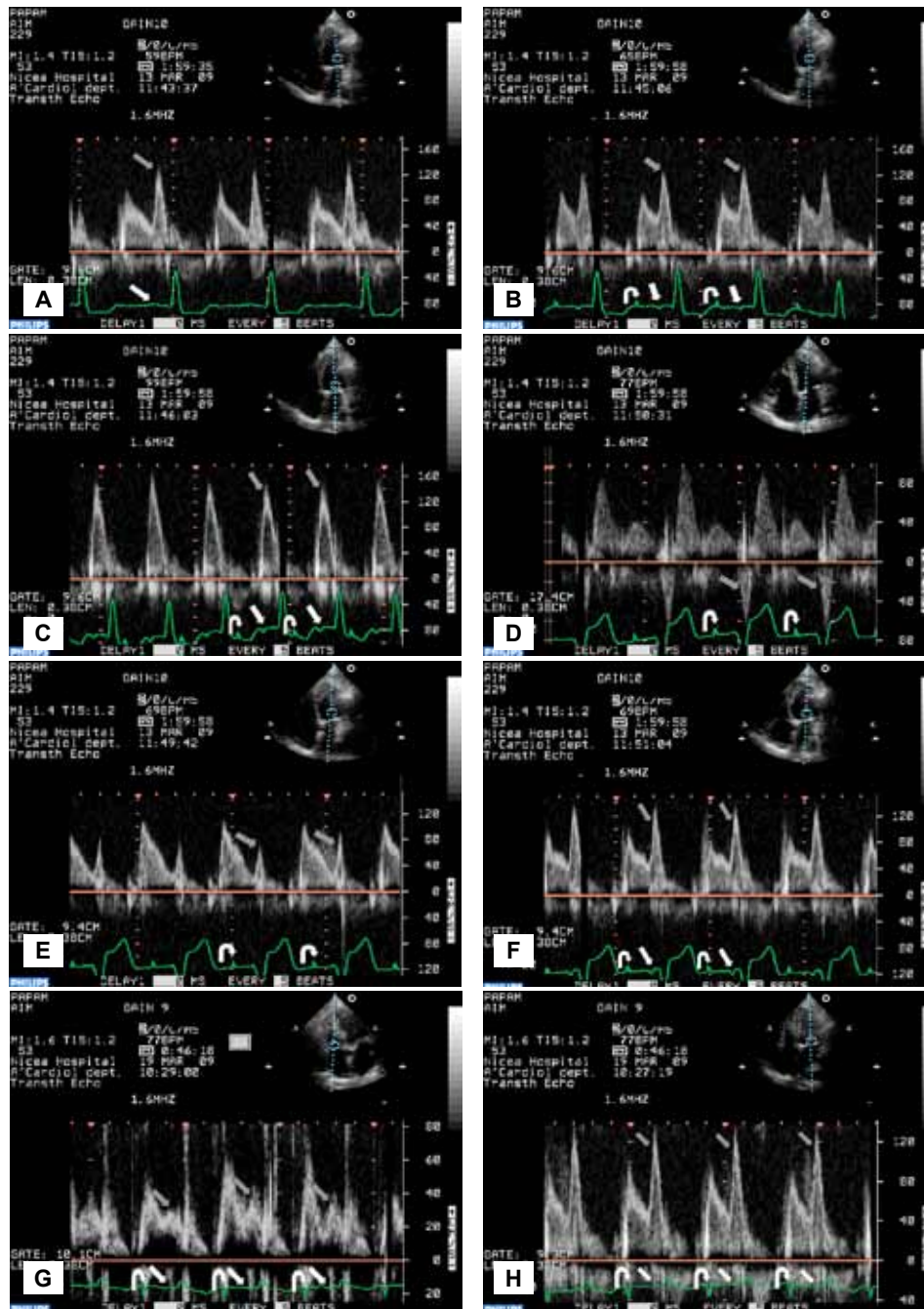


Figure 4. A. VVI 30 beats/min. Intrinsic sinus rhythm with a clear P-wave (white arrow), which is followed by the A-wave (grey arrow) on the Doppler study (end-diastolic left ventricular filling). B. AAI 70 beats/min. Atrial spike (curved arrow) followed by a significantly delayed atrial P-wave (white arrow), which then produces a Doppler A-wave (grey arrow). C. AAI 100 beats/min. Atrial spike (curved arrow) followed by a significantly delayed atrial P-wave (white arrow), and subsequently a Doppler A-wave, which, however, cannot be distinguished clearly because of the union of the E and A waves (grey arrow). D. DDD 70 beats/min, atrioventricular delay 200 ms. Doppler investigation of pulmonary venous flow. The pacing spike (curved arrow) is followed by an atrial wave, which, however, cannot be recorded because it is overwritten by the QRS of the pacemaker's ventricular depolarisation. An impressively large retrograde A-wave is shown (grey arrow), which arises from the left atrial systole while the mitral valve is closed by the simultaneous ventricular systole. E. DDD 70 beats/min, atrioventricular delay 200 ms. Pacing spike (curved arrow) producing a P-wave that is not visible because it coincides with the QRS. However, a relatively small A-wave is produced (grey arrow) slightly before the paced ventricular systole that follows. F. DDD 70 beats/min, atrioventricular delay 300 ms. Pacing spike (curved arrow) producing a P-wave (white arrow) that causes a clear A-wave (grey arrow) well timed in advance of the paced ventricular systole that follows. G, H. AAI 70 beats/min. Spectral Doppler investigation of tricuspid (G) and mitral (H) flow. Shown in order are the spike (curved arrow), the generated P-wave (white arrow) and the A-wave (grey arrow), which in both cases is produced immediately, documenting the simultaneous systole of the two atria.

picture. It should also be stressed that, initially, the only logical, albeit incorrect, interpretation of the paradoxical ECG was that it showed a loss of atrial capture by the atrial electrode, at the same time as atrial capture by the ventricular electrode. This in turn could be due either to displacement of the electrodes from their initial position, with the atrial electrode not causing capture and the tip of the ventricular electrode being in contact with atrial tissue, or to the proximal ends of the two electrodes being wrongly (inversely) connected to the pacemaker generator in combination with a loss of sensing and capture by the ventricular electrode. It is obvious that both these cases of pacemaker malfunction can be treated only invasively by opening the surgical wound and replacing or reconnecting the electrodes, respectively.

In our case, both these possible explanations were ruled out during the subsequent examinations and the real malfunction, as explained above, could be treated adequately by modification of the pacing parameters alone. Both the discernible causes of malfunction that composed the unusual ECG picture in the case described here (exit block of the atrial spike by way of first degree sinoatrial block, and loss of ventricular capture because of an increase in threshold) were attributed to a strong, aseptic inflammatory reaction with subsequent oedema at the points of contact between the electrode tips and the atrial and ventricular endocardium, leading us to the decision to give our patient corticosteroids. As a result, there was an improvement in the functional parameters of the ventricular electrode, though not the atrial electrode. This finding suggested a slight displacement of the atrial electrode (not apparent on the chest X-ray) as the cause of its malfunction, without excluding the non-reversible development of fibrous tissue with extremely slow conduction at the

contact point of the atrial electrode within the auricle of the right atrium.

In conclusion, this is an extremely rare case of a pacing ECG that led to an entirely erroneous interpretation of pacemaker malfunction. Some degree of entry-exit block of the pacing stimulus should always be in our diagnostic thoughts in any case where the pacing ECG is difficult to interpret, with the knowledge that there may be significant delay in the electromechanical manifestation of atrial function after the appearance of the atrial spike on the surface ECG. Even in cases where the only apparent explanation of the paced ECG picture is a severe malfunction than cannot be treated conservatively, a full check of the pacemaker device should be carried out in order to determine the true nature of the malfunction before any therapeutic decision is taken.

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