In: Cardiac pacing. Proc. of the 4th international symposium on cardiac pacing Groningen, 1973. Ed. H.J.Th. Thalen. Assen, Van Gorcum, 1973.

Electrocardiograms of patients with (mal)functioning pacemakers

Diagnostic and therapeutic implications

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Understanding and/or interpretation of electrocardiograms of patients with (mal)functioning pacemakers require(s) knowledge of human electrocardiography and the electronic properties of the pacemaker systems used.
At present the following pacemaker systems are being used clinically:
1. Fixed rate pacemakers,
2. Demand pacemakers, to be divided into R-wave inhibited and R-wave synchronized,
3. Atrial triggered pacemakers and
4. Sequential pacemakers.

Fig. 1 demonstrates the electrocardiogram of a 69-years old patient with a fixed rate pacemaker. It can be seen that the pacemaker of which the stimulus artefact is indicated by the arrows, functions independently of autochthonous cardiac activity. In this case a stimulus artefact (third arrow) falls in the refractory period of a preceding premature beat and hence is not followed by a propagated response. It is possible for the pacemaker impulse to coincide with the vulnerable period and to cause ventricular tachycardias or even ventricular fibrillation. For that reason the demand pacemaker has definite advantages over the fixed rate type.
Fig. 2 shows the electrocardiogram of a 25-years old patient who was treated with a pacemaker for congenital complete heart block and Adams-Stokes attacks. The ECG shows bigeminal rhythm and the demand properties of the pacemaker used. Again the arrows point to the stimulus artefacts caused by the pacemaker. A striking feature of this bigeminal rhythm is the fact that the premature beats have virtually the same configuration as the complexes originated by the electronic pacemaker. Evidently the stimulus causes a re-entry mechanism within a very small area adjacent to or identical with the site of the epicardial electrodes.
The ECG of a patient (70-years old female) with an R-wave synchronized demand pacemaker is demonstrated in Fig. 3. It turns out that the pacemaker is incapable to detect each R-wave. This depends on the duration of the preceding R-R interval(s). Accurate analysis of this electrocardiogram reveals that the pacemaker is only able to synchronize after a preceding R-R interval of 480 ms or longer. An early QRS-complex (460 ms) as occurs in this case of atrial fibrillation

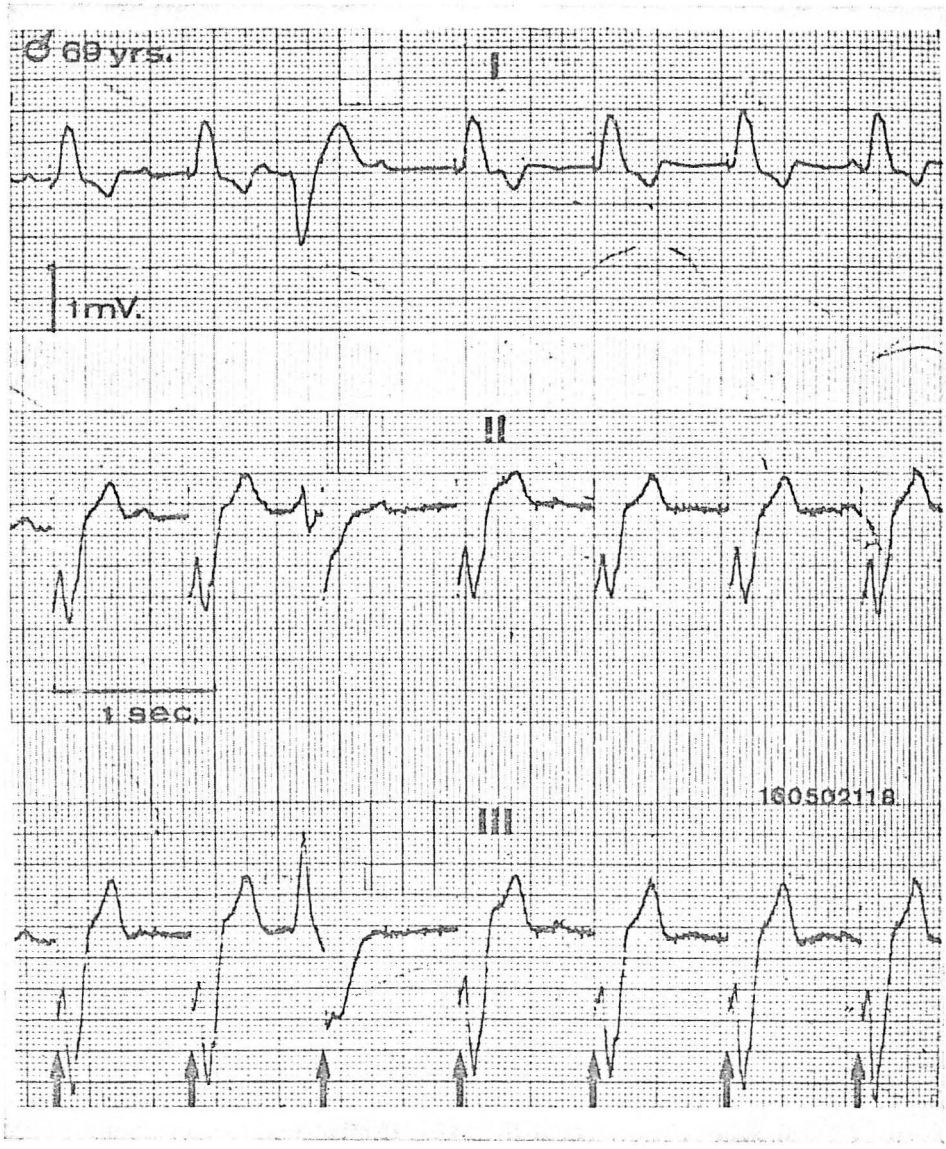

Fig. 1 Fixed rate pacemaker.

has not been detected by the pacemaker and thus did not prevent the subsequent pacemaker stimulus. In this way, despite the demand mode, a pecemake. stimulus may again faill within the vulnerable period and cause ventricular arrhythmias, in this case because of an extremely long "refractory period" of the pacemaker.

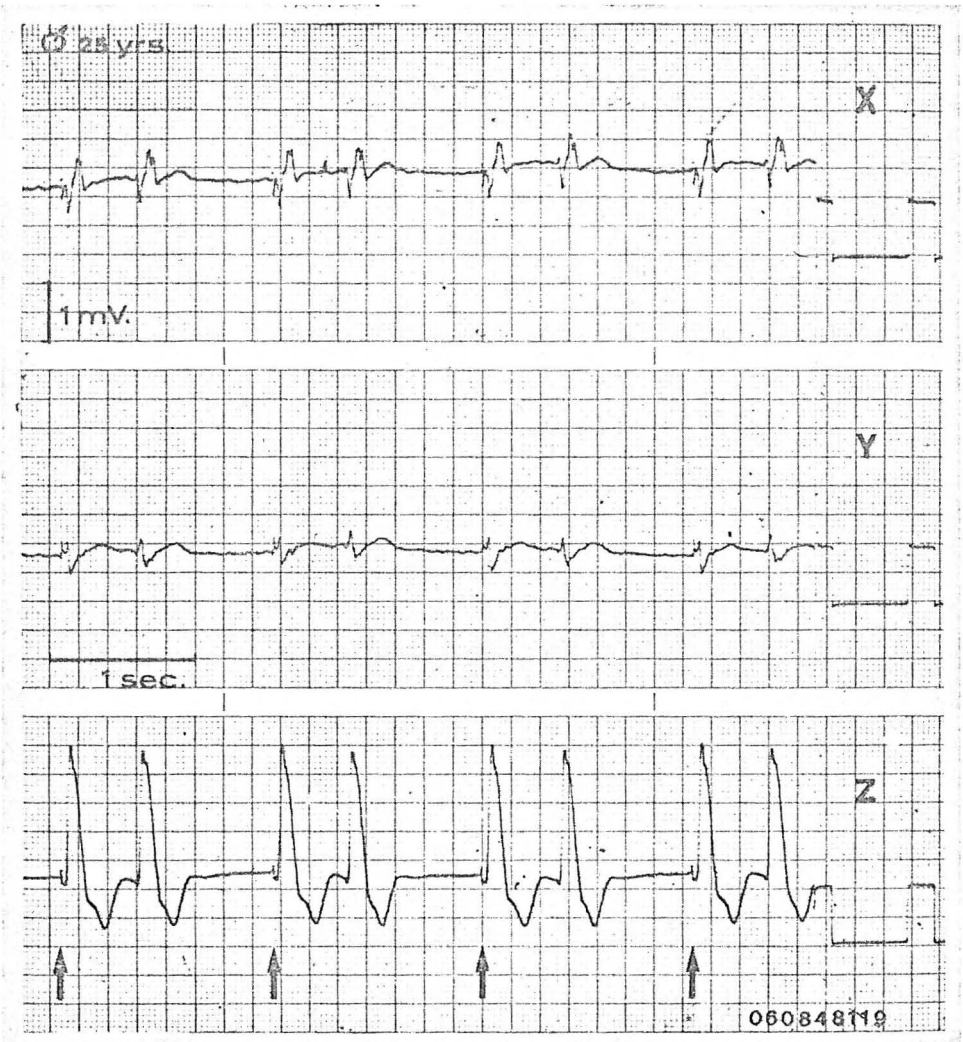

Fig. 2 Demand, R-wave inhibited, pacemaker.

Atrial triggered and sequential pacemakers are not being used clinically in most departments. Examples of their functioning and mal-functioning can be demonstrated in animal experiments.

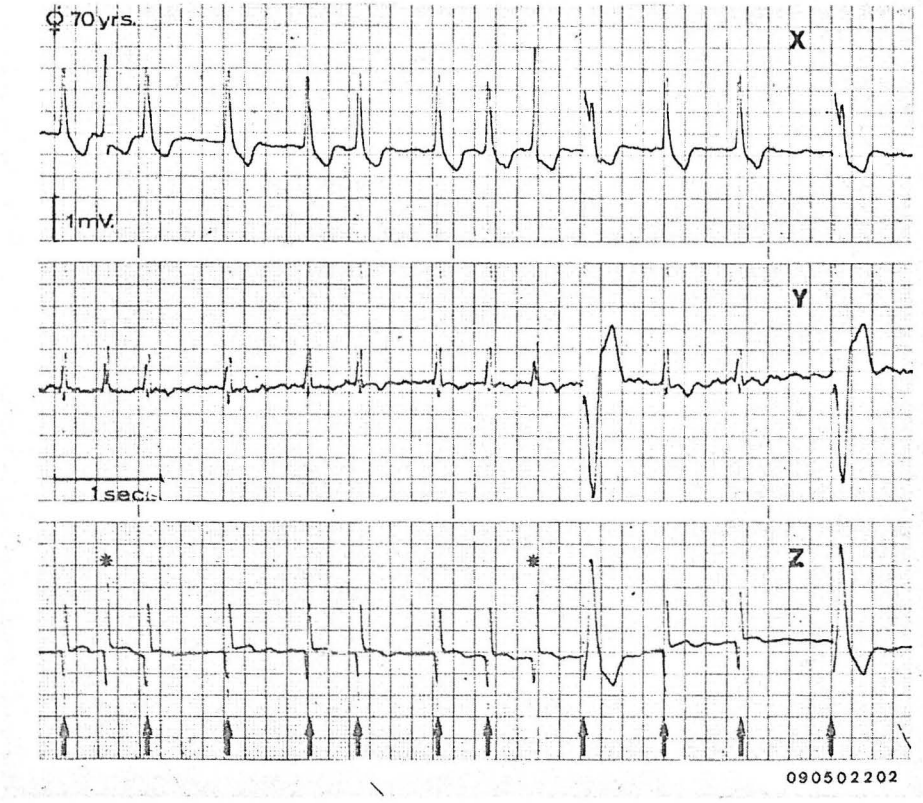

Fig. 3 Demand, R-wave synchronized, pacemaker.

Recently we have used an externally programmable pacemaker in a 55-years old male patient with a bilateral bundle branch block and intractable ventricular tachycardias.

Fig. 4A shows the ECG of that patient during regular sinus rhythm prior to pacemaker implantation. Despite the pacemaker the patient got another episode of ventricular tachycardia which was terminated by a DC shock. After that episode the ventricular tachycardias appeared more often and the electrocardiogram

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Fig. 4 ECG of patient with periods of bilateral bundle branch block and intractable ventricular tachy cardias.
A. Uppertracing: ECG before pacemaker implantation.
B. Lower tracing: ECG after pacemaker implantation and DC shock (see text).

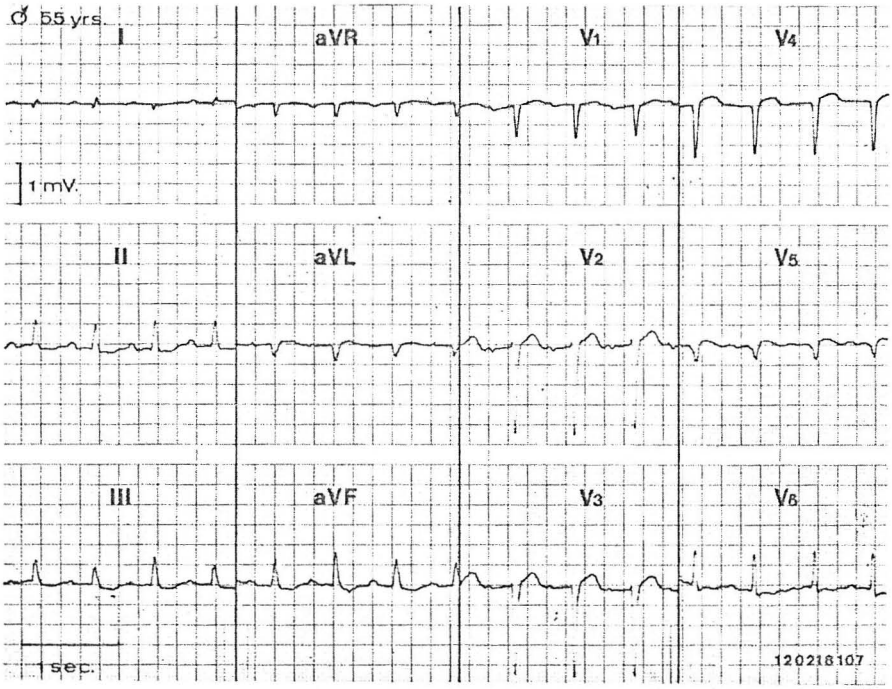
♂ 55 yrs.
I
aVR
V1
V4
1 mV.
II
aVL
V2
V5
III
aVF
V3
V6
1 sec.
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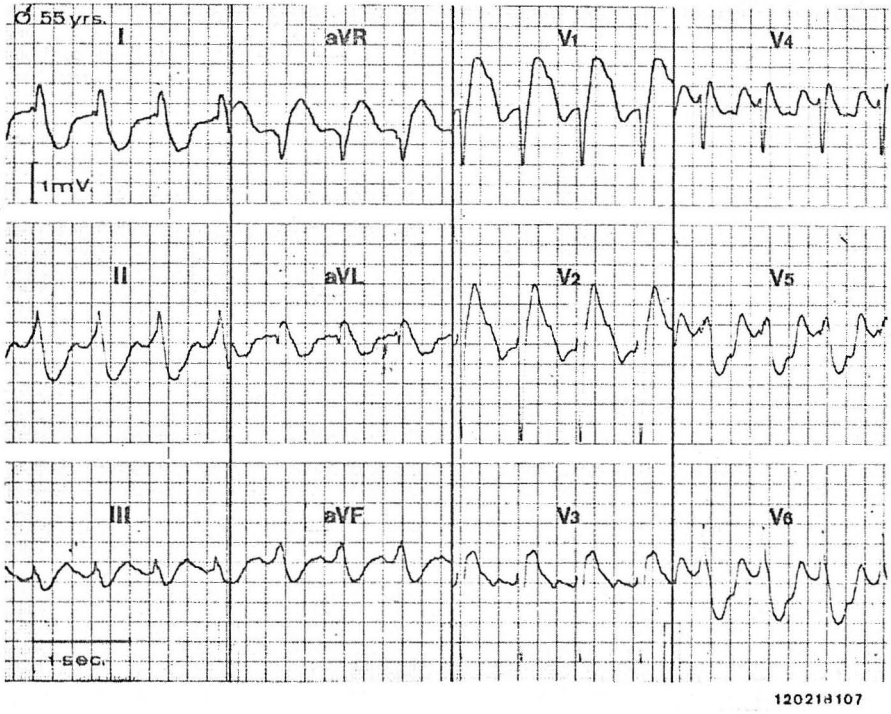
Fig. 4A and 4B.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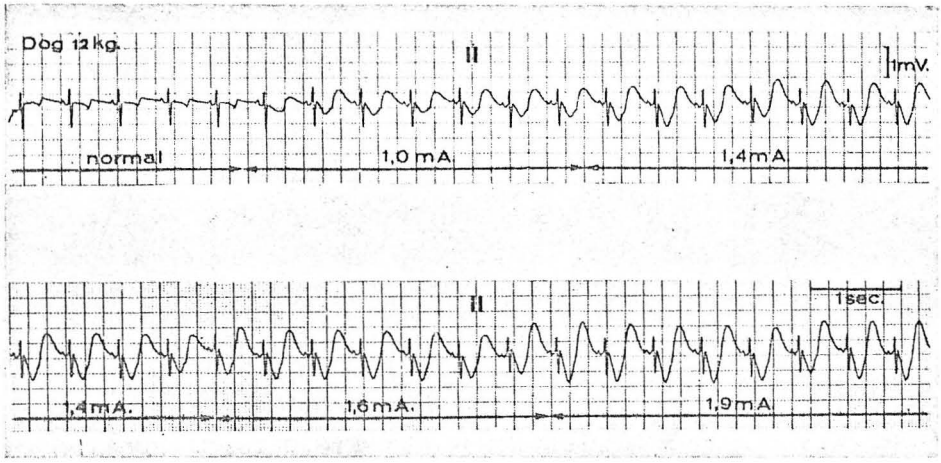

Fig. 5 Endocardial transvenous monopolar stimulation catheter carrying DC current. DOG experiment. See text.

showed a dramatic change (see Fig. 4B). The patient died during another attack of ventricular tachycardia. The pacemaker showed gross changes due to electrolysis caused by a continuous DC output which in turn was originated by the damage evidently caused by the DC shock.

The effect of DC output applied to the heart of a dog via an endocardial unipolar electrode in the right ventricle is shown in Fig. 5. This experiment proves that the ECG changes of our patient (see Fig. 4B) could indeed have been caused by the DC current of the damaged pacemaker. In this case the electronic properties of this particular pacemaker apparently made it vulnerable to DC counter shocks to which patients may be exposed. DC current applied directly to the heart over a longer period of time may cause untreatable and lethal ventricular tachycardias.

This example also demonstates that knowledge of pacemaker design and electronics and its effect upon the electrical activity of the heart is essential for the understanding of predictable and unpredicted side-effects of pacemakers.