

Letters to the Editor

Heat syncope and sustained ventricular tachycardia

Dear Sir,

Heat is responsible for several different clinical conditions^{2,6}, syncope being the most common. Its prognosis is generally benign. Peripheral vasodilatation occurs which triggers cerebral hypoperfusion and causes blackouts. This is common among athletes who exercise in hot temperatures and humid conditions, as well as in patients with cardiovascular disease and who are being treated with diuretics^{2,3}.

The following case involves a patient who was exercising in warm conditions and suffered from syncope and sustained ventricular tachycardia (VT).

This is the case of a 42-year-old woman who was a tourist from Germany and ran marathons. She had been in the country for less than 24 hours and had no relevant medical history or toxic habits. After 10 minutes of continuous physical exercise at a night time temperature of 32°C, she presented with a syncopal episode and spontaneously regained full consciousness. She was attended by our Emergency Medical Unit and during the first examination, the following points were noted: she was conscious and showing severe symptoms; tachypnoea (16 breaths per minute), sweating, signs of poor peripheral perfusion, blood pressure was 85/52 mmHg, her oxygen saturation level was 90%, her heart rate was fast at over 200 beats per minute, capillary glucose was 98 mg/dl and her body temperature was 37°C. We began with oxygen therapy which was given at a high flow rate of 15 litres per minute via an oxygen mask (Ventimask), cardiac monitoring and cannulation of two peripheral veins for fluid resuscitation with normal saline. An ECG was obtained (Figure 1) which showed regular broad QRS complex tachycardia at 250 beats per minute, typical of monomorphic ventricular tachycardia (VT). Given the patient's haemodynamic instability⁸⁻¹⁰ we opted for synchronised electrocardioversion to treat the VT. The patient was sedated and administered analgesia beforehand with 15 mg of midazolam and 0.5 mg of intravenous morphine chloride. An electric shock of 50 joules was given (biphasic defibrillator/monitor) restoring the tachycardia to a sinus rhythm of 50-60 beats per minute (Figure 1). The patient maintained this rhythm and remained haemodynamically stable while she was transferred to the hospital. She arrived at the hospital with a blood pressure reading of 115/60 mmHg, normal respiratory rate, normal colour, well perfused and her oxygen saturation level was 99%. She remained sedated. While she was being transferred to the hospital she was given 1000 ml of normal saline.

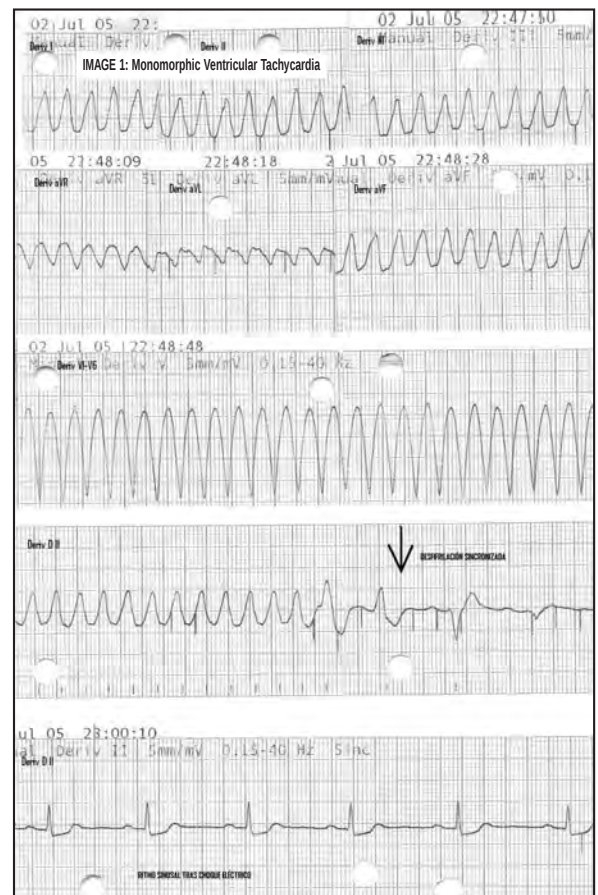


Figure 1. The patient's ECG results show ventricular tachycardia which returned to sinus rhythm after electrocardioversion.

The initial tests carried out after the patient was admitted to hospital (full blood count, biochemistry including electrolytes and cardiac enzymes, renal function, liver function and coagulation) showed no abnormalities. The chest x-ray was also normal and the ECG continued to show sinus rhythm at 62 beats per minute with no acute changes. The tests carried out on admission into hospital did not show any structural cardiac abnormalities which could indicate the possible cause of the ventricular tachycardia and she did not present any further tachycardic episodes.

In the case of this patient, the heat probably triggered the syncope given that the patient was not used to the Spanish climate^{3,5,6}, although other factors like physical exercise may have triggered the development of the VT. The patient often ran marathons and had never had this kind of episode in the past. The cardiac investigations ruled out a structural abnormality as the cause of the arrhythmia and therefore the VT was idio-

pathic, sustained and related to physical exertion¹. The mechanisms associated with the development of tachyarrhythmia are the following; an increase in automatism, abnormal automatism and circular or re-entry stimulation which is the most common and comes from the right ventricle outflow tract in up to 80% of cases^{1,8}. VT related to exercise is generally benign, however, if associated with precipitating factors such as cardiomyopathy, structural or organic heart disease or abnormal physiological conditions, it can be potentially serious. However, it must be said that physical exercise should not be considered an arrhythmogenic factor and generally does not induce ventricular arrhythmia. There are also no significant differences with respect to its prevalence among the general population^{1,7}.

Therefore we can conclude by saying that in this case, different elements such as the fact that the patient was not used to the Spanish climate, exercise and very warm conditions came together and probably contributed to this clinical picture.

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Subclavian crush syndrome, a serious complication caused by a permanent pacemaker

Dear Sir,

In 2005, 28,724 pacemakers were implanted into patients in Spain. This translates into 651.3 generators per million people¹, and this figure is on the increase. In 1994 a large scale survey showed at that time that intensive care physicians were responsi-

ble for 40% of pacemaker insertions in Spain. Details of their work was collected annually and entered in the MAMI2 (intensive care medicine data base) register. This means that emergency physicians should have access to information about the basic characteristics of pacemakers and the complications involved^{3,4}. The following case describes an incident involving a pacemaker.

This case involves a 67-year-old man who came to the emergency department at our hospital complaining of pain in his right shoulder that had developed over several days. He had a permanent VDD pacemaker which had been implanted three years before because of a bifasicular block and paroxysmal symptomatic episodes of a complete atrioventricular heart block (AVB).

The results of the examination suggested the presence of a fluctuating haematoma without any signs of inflammation in the area of the pacemaker but this was ruled out beforehand. The ECG showed a sinus rhythm with a first degree block AVB (0.24 m/sec) and the chest x-ray (Figure 1) showed a disruption in the continuity of the ventricular electrode. A suspected broken pacemaker lead was diagnosed and the intensive care department, which implants and checks the devices in our hospital, was consulted. The broken electrode was confirmed using thoracoscopy while the analysis with the programmer showed how three months before the patient had come to the emergency department with an increase in the electrode impedance to 23,986 ohms (Ω) which automatically reverted the polarity of the stimulation to unipolar with a decrease in impedance to 3,000 Ω in later automatic checks. There were no clinical symptoms.

A new lead was inserted. Retrieval of the old electrode showed a clean cut 20 cm from the connector. Exploration further up to the subclavian vein showed that the remaining fragment was not at the distal end that was in the intravascular space. Since we did not have the technical means to extract the electrodes or a cardiovascular surgery department in our hospital, we contacted another centre in the area which did have the necessary facilities to deal with the case.

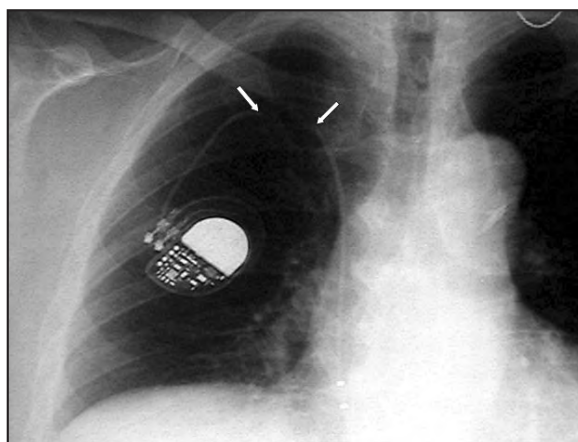


Figure 1. Chest x-ray which shows the pacemaker and its broken electrode (highlighted by the arrows).

After evaluating the case, a more conservative approach was adopted, the patient was reviewed frequently and no related complications appeared until that point (three years later).

Subclavian crush syndrome causes the deterioration of electrodes that are implanted via the subclavius. The lead is severed due to the compression and insertion of the cable between the first rib and the subclavius. The incidence rate varies between 1% and 2.5%⁵ and cases involving the complete section of the electrode are even less frequent. Most breaks occur when the implantation is in the middle section of the clavicular region. According to other reports, ways to avoid this include implantation via other subclavian channels or locating the subclavian vein beforehand with a contrast guided approach to the extrathoracic portion⁸. The medical consequences for patients who depend on their pacemakers can be very serious leading to syncope and even sudden death. When the patient does not depend on the device, the malfunction may be discovered by chance.

Some academics advocate yearly chest x-rays to check that the electrodes are intact and in place, especially for patients who are pacemaker dependent and whose pacemakers have been inserted via the subclavius⁹. It is thought that in cases where a malfunction is detected during checks it could be a useful tool for the differential diagnosis, just as it is for patients who come to the emergency department with symptoms that suggest that the device is not working properly.

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Antibiotic treatment of community-acquired pneumonia

Dear Sir,

We read Moya Mir's¹ article on empirical antibiotic treatment of community-acquired pneumonia (CAP) with great interest, in particular the part about oral beta-lactam antibiotics, where the author recommends cefditoren pivoxil as an oral treatment with beta-lactam antibiotics. We believe that such conclusions should be explored further and in certain cases, reconsidered.

Current guidelines that recommend empirical treatment which covers atypical pathogens in patients with CAP are based on studies with level III evidence². Two recent meta-analyses of randomised studies which compared treatment with and without coverage for atypical pathogens (*Mycoplasma pneumoniae*, *Chlamydia pneumoniae* and *Legionella pneumophila*) showed that there were no advantages in terms of survival or clinical effectiveness for hospitalised patients with mild/moderate CAP^{3,5}. In our hospital, the incidence rate of CAP caused by *Legionella* spp. stands at 1.5% in patients who have undergone urinary antigen detection (Binax NOW8) to diagnose pneumonia caused by *L. pneumophila* and using nonconcentrated urine. Therefore, given the low incidence rate of pneumonia caused by *Legionella* spp. in our hospital, we understand that the use beta-lactam antibiotics as the only treatment is adequate for managing CAP, at least in patients who do not need to be admitted to an intensive care unit⁶, when the antigen *Legionella* spp. test is negative^{7,8}. This argument is also supported by medical data and epidemiologists^{7,9}.

We do not agree with Moya Mir's point that within the group of oral beta-lactam antibiotics, cefditoren pivoxil has the same indications as acid in patients with CAP. The effectiveness of an antibiotic is measured by assessing the minimum inhibitory concentration (MIC) according to clinically significant susceptibility cut-off points or the pharmacokinetic/pharmacodynamic (PK/PD) parameters^{10,11}. Cefditoren pivoxil works well *in vitro* against penicillin susceptible *S. pneumoniae* or *S. pneumoniae* with medium susceptibility (MIC₉₀ ≤ 0.5 µg/ml) and produces mixed results against penicillin resistant *S. pneumoniae* (MIC₉₀ is 0.5-2 µg/ml)¹², but there are no clinically significant susceptibility cut-off points for cefditoren in the treatment of *Streptococcus pneumoniae*¹². However, according to the PK/PD parameters, the treatment objective of beta-lactams is to obtain more time than MIC₉₀ by at least a 40% dosage interval for *S. pneumoniae* which is not susceptible to penicillin. Based on these parameters, the average times for the plasma concentrations of cefditoren are ≥ 1 or ≥ 2 µg/ml in 6 hours (50% of the dosage interval) and in 3.8 hours (31.6% of the dosage interval), respectively, after a dose of 400 g¹³ and therefore it will

be effective in many patients with *S. pneumoniae* with medium susceptibility to penicillin. However, it is doubtful that it is effective in microorganisms with a MIC ≥ 2 $\mu\text{g/ml}$ such as *S. pneumoniae* which is resistant to penicillin. In two randomised CAP studies quoted by Moya Mir¹, the effectiveness of cefditoren pivoxil was evaluated (200 mg or 400 mg every 12 hours) compared with amoxicillin/clavulanic acid (875 mg/125 mg every 12 hours) and cefpodoxime proxetil (200 mg every 12 hours) with no significant clinical or microbiological differences. However, these two studies are not valid from a microbiological standpoint because less than 10 patients with penicillin-resistant *S. pneumoniae* were evaluated. Furthermore, the uncertain effectiveness of cefpodoxime against penicillin-resistant *S. pneumoniae* (after showing MIC₉₀ of 2 against *S. pneumoniae* and based on the PK/PD parameters), makes us doubt the methodology of this study and the clinical value of the patient's sputum with *S. pneumoniae*. In fact, in the U.S and Europe, cefditoren pivoxil has been approved to treat infections caused by penicillin susceptible *S. pneumoniae* or *S. pneumoniae* or with reduced susceptibility to penicillin¹⁴ but not to treat penicillin-resistant *S. pneumoniae* (MIC ≥ 2 $\mu\text{g/ml}$). Therefore, from a PK/PD perspective, of the oral beta-lactam antibiotics, only high doses of amoxicillin/clavulanic acid (875 mg/125 mg every 8 hours or 2000 mg/125 mg every 12 hours) would provide the best coverage against *S. pneumoniae*, including those strains not susceptible to penicillin^{10,11,15,16}, as well as fight against the development and spread of resistant pathogens¹⁰.

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