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REVIEW

The effect of hyperkalaemia on cardiac rhythm devices

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In patients with pacemakers, hyperkalaemia causes three important abnormalities that usually become manifest when the K level exceeds 7 mEq/L: (i) widening of the paced QRS complex from delayed intraventricular conduction velocity, (ii) Increased atrial and ventricular pacing thresholds that may cause failure to capture. In this respect, the atria are more susceptible to loss of capture than the ventricles, and (iii) Increased latency (usually with ventricular pacing) manifested by a greater delay of the interval from the pacemaker stimulus to the onset of depolarization. First-degree ventricular pacemaker exit block may progress to second-degree Wenckebach (type I) exit block characterized by gradual prolongation of the interval from the pacemaker stimulus to the onset of the paced QRS complex ultimately resulting in an ineffectual stimulus. The disturbance may then progress to 2 : 1, 3 : 1 pacemaker exit block, etc., and eventually to complete exit block with total lack of capture. Ventricular undersensing is uncommonly observed because of frequent antibradycardia pacing. During managed ventricular pacing, hyperkalaemia-induced marked first-degree atrioventricular block may induce a pacemaker syndrome. With implantable cardioverter-defibrillators (ICDs) oversensing of the paced or spontaneous T-wave may occur. The latter may cause inappropriate shocks. A raised impedance from the right ventricular coil to the superior vena cava coil may become an important sign of hyperkalaemia in the asymptomatic or the minimally symptomatic ICD patient.

Keywords Hyperkalaemia • Cardiac pacemaker • Implantable cardioverter-defibrillator • Pacemaker failure • Oversensing by ICD

Hyperkalaemia may affect ~8% of hospitalized patients in the USA. It is common in older patients undergoing intensive treatment for heart failure (with potassium-retaining drugs) especially if there is coexisting renal insufficiency.[1–6] Hyperkalaemia is the most common electrolyte or drug abnormality to cause loss of capture by a cardiac rhythm device. In patients with pacemakers, hyperkalaemia causes two important clinical abnormalities: (i) widening of the paced QRS complex (and paced P-wave if it is seen) on the basis of delayed myocardial conduction (*Figure 1*). When the K level exceeds 7 mEq/L, the intraventricular conduction velocity is usually decreased and the paced QRS complex widens. Other common causes of a wide paced QRS complex include amiodarone therapy and severe myocardial disease. (ii) Increased atrial and ventricular pacing thresholds with or without increased latency. Failure to capture is usually seen when the K level reaches 7 mEq/L and occasionally at a lower level especially in the presence of heart disease.[8–11]

Electrophysiology

The potassium concentration gradient between extracellular and intracellular sites is the most important factor that controls the resting membrane potential. According to the Nernst equation or its simplified equivalent: 61 log K_i/K_o (with I=intracellular and o=extracellular) the resting membrane potential or ionic equilibrium is directly related to the ratio of intracellular to extracellular K concentration [normally about 30 (*Figure 2*)]. As the external K increases, the ratio will decrease causing partial depolarization of the cellular membrane to a less electronegative value (*Figure 2*). The threshold potential (critical value of depolarization that can provoke an action potential) also becomes progressively less negative with a rising extracellular K concentration.[12,13] The decrease in the d*V*/d*t* max or the slope of the upstroke of the action potential (Phase 0) is a major determinant of conduction velocity, which is counterbalanced by the difference between the resting membrane potential and the threshold potential.[12,13] This difference diminishes as the K level rises so that the d*V*/d*t* max of Phase 0 of the action potential becomes the dominant abnormality whereupon it produces a net reduction of conduction velocity. The duration of the action potential is decreased with shorter Phases 2 and 3 and the descent is more rapid (*Figure 2*). The faster repolarization causes tenting of the T-wave in the surface electrocardiogram (ECG).[12,13]

Ventricular pacing

It is well-known clinically that the administration of oral or intravenous (IV) potassium may decrease the ventricular pacing threshold

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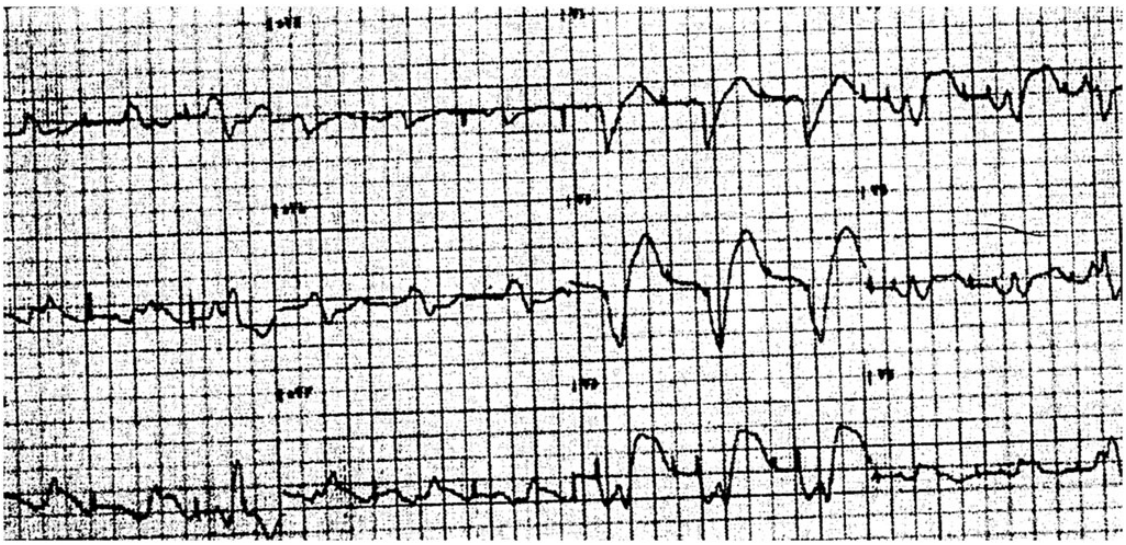

Figure 1 Twelve-lead ECG during hyperkalaemia ($K = 7.2$ mEq/L) showing loss of atrial capture and marked increase in the duration of the paced QRS complex (0.34–0.36 s). Some of the leads show pseudolatency of ventricular stimulation because of the initial isoelectric activation. Note the ST elevation in the inferior leads simulating an acute inferior myocardial infarction. There was no evidence of acute myocardial infarction and angiography revealed normal coronary arteries. Such ST elevation may occur in hyperkalaemia usually involving the right precordial leads and less commonly in the inferior leads.[7] Convex ST elevation in the inferior leads during ventricular pacing has not been previously reported. (Reproduced from Barold SS[30] with permission.)

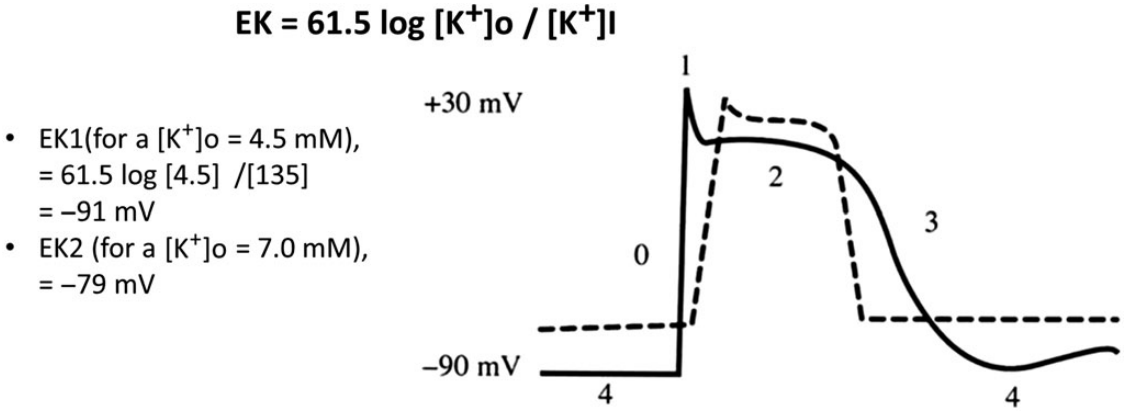

Figure 2 Electrophysiological effects of hyperkalaemia. The left side on top shows the simplified Nernst equation. EK=resting membrane potential (EK1 with a normal K level and EK2 with a high extracellular K level). Note the reduction of the resting membrane potential with hyperkalaemia from −91 mV ($K = 4.5$ mM) to −79 mV ($K = 7$ mM). The right side shows a diagrammatic representation of the normal action potential (solid line) and the action potential in hyperkalaemia (interrupted line). The rate of rise of Phase 0 is directly proportional to the resting membrane potential at the onset of Phase 0. This decrease in V_{max} causes slowing of the conduction. Hyperkalaemia causes shortening of the action potential by increasing the slope of Phases 2 and 3 of the action potential. This corresponds to the shortening of repolarization.

even if the serum K is normal.[14–16] The initial decrease is followed by an increase in the threshold as the K level rises. The level of hyperkalaemia causing clinical changes in the pacing threshold varies from patient to patient but when the serum K exceeds 7.0 mEq/L, there will almost always be an increase in the pacing threshold but not necessarily failure to capture.[17–22] The presence of heart disease in clinical situations associated with hyperkalaemia probably explains the greater susceptibility of the human heart to hyperkalaemia. Another factor may be the slower development of clinical hyperkalaemia vs. IV infusion of KCl. Other metabolic variables may influence

the sensitivity of cardiac tissue to hyperkalaemia because a modest elevation of the K level (e.g. 6.5 mEq/L) may sometimes cause failure of atrial and/or ventricular capture. These factors include other types of electrolyte imbalance, acid–base abnormalities, oxygen saturation, the rate of change of plasma K level, the intracellular–extracellular gradient, the type of antiarrhythmic drugs, and the aetiology and severity of heart disease. Types I and III antiarrhythmic drugs potentiate the effect of hyperkalaemia, which also means that hyperkalaemia promotes drug toxicity.[23–25] For this reason, the cardiac manifestations of hyperkalaemia in the clinical setting tend to occur at much lower K levels than those measured during an experimental infusion of potassium. In this respect, McVenes *et al.*[26] studied the effect of hyperkalaemia ($K = 8.3$ mEq/L) in dogs with chronic transvenous right ventricular (RV) leads in the RV. Hyperkalaemia caused a 23–29% decrease in the threshold voltage with no change in the impedance in contrast to an increase in the atrial pacing threshold. This study confirmed the clinical belief that the effect of hyperkalaemia is less pronounced in the ventricles of normal or slightly abnormal hearts. The greater susceptibility may be due to the slower development of clinical hyperkalaemia compared with a relatively rapid infusion of KCl. This concept was illustrated by the unimpressive changes in the ventricular pacing threshold in the study of McVenes *et al.*,[26] which was conducted in dogs without heart disease (12 weeks after lead implantation).

QRS configuration and duration

The paced QRS complex increases in duration reflecting slower intramyocardial conduction. A marked increase in QRS duration with reported durations of 400 ms is rare.[27] The wide QRS complex may predispose to far-field atrial sensing of the paced QRS complex.[28] The unusual initial part of the paced QRS configuration (1 : 1 capture) during regular ventricular pacing has been incorrectly interpreted as showing the failure of ventricular capture (*Figure 4*).[29]

Differential effect on atrial vs. ventricular myocardium

In a dual-chamber device, hyperkalaemia may cause failure of atrial capture associated with the preservation of ventricular pacing (*Figure 3*). The effect of K^+ infusion on the atrial myocardium in the canine study of McVenes *et al.*[26] was variable and showed that when K^+ exceeded about 7 mEq/L, half the leads developed atrial exit block at $\geq$10 V with various pulse durations. The other leads continued to pace satisfactorily. There was no change in the atrial pacing impedance (12 weeks after implantation). The presence of steroid elution in one case did not prevent exit block. This differential effect on atrial and ventricular excitability (pacing) correlates with the well-known clinical and experimental observations that the atrial myocardium is more sensitive to hyperkalaemia than the ventricular myocardium.[27,30–32] Loss of atrial capture should be suspected in hospitalized pacemaker patients with severe heart failure, who develop relatively sudden decompensation with hypotension after exclusion of major causes of hypotension such as myocardial infarction, pulmonary embolism, and so on. This situation requires an immediate ECG that will almost always show a marked increase in paced QRS duration compared with previous recordings. Successful atrial capture may not be discernible because hyperkalaemia attenuates the P-wave ECG. Loss of atrial capture should be demonstrated at the maximum programmable atrioventricular (AV) delay to rule out increased atrial latency and recording the ECG at double standardization to confirm the loss of atrial activity (*Figure 4*). Electrocardiographic evaluation of atrial capture should focus on lead V1 because successful atrial capture may be visible only in this particular lead, which should be recorded at double standardization to bring out the low-amplitude P-waves induced by hyperkalaemia. Failure to appreciate this fact with hyperkalaemia has led to the erroneous diagnosis of failure of atrial capture (*Figure 5*).

Undersensing

The amplitude of the atrial electrogram in the canine study of McVenes *et al.*[26] decreased significantly (about 70%) and its slew rate dropped proportionately by 64% from 2.8 ± 1.6 to 1.0 to 0.5 V/s. Hyperkalaemia caused marked attenuation of the atrial signal for sensing. The frequency of P-wave undersensing (related to a depressed amplitude and slew rate of the intracardiac electrogram) has not been studied perhaps because atrial pacing occurs often as the spontaneous atrial rhythm is often suppressed without any visible spontaneous atrial activity. This difficulty is compounded by the reduced amplitude and eventual disappearance of the surface P-wave with increasing hyperkalaemia. The lower amplitude of the atrial electrogram seems to correlate with the reduction of the P-wave amplitude on the surface ECG.

In terms of ventricular sensing, McVenes *et al.*[26] in their canine study found no significant change in the amplitude and slew rate of the ventricular electrogram. In this respect, Haberen *et al.*,[33] who studied the effects of KCl infusion on acute ventricular sensing in canines (with epicardial and endocardial leads) post-myocardial infarction, found a significant decrease in ventricular amplitude and slew rate. It seems therefore that the effect of hyperkalaemia on ventricular signal amplitude and slew rate may be of clinical significance at least if sensing is marginal to begin with. Ventricular undersensing with the preservation of ventricular pacing has been rarely reported in hyperkalaemia probably because the paced rhythm suppresses the underlying bradycardia induced by hyperkalaemia (*Figure 5*).[34,35] However, ventricular undersensing (reversible after therapy) may become manifest when hyperkalaemia causes failure of ventricular capture. Friedman *et al.*[36] reported hyperkalaemia-induced QRS undersensing by an implantable cardioverter-defibrillator (ICD), a complication potentially life-threatening.

Oversensing

Oversensing of the T-wave induced by hyperkalaemia has been reported in ICDs but not in pacemakers because the ventricular sensitivity of ICDs is so high.[35] Far-field sensing of a prolonged QRS complex (beyond a 300 ms post-ventricular atrial refractory period) has been reported to cause far-field endless loop tachycardia in a patient with hyperkalaemia producing a wide paced QRS complex extending beyond the post-ventricular atrial refractory period.[28]

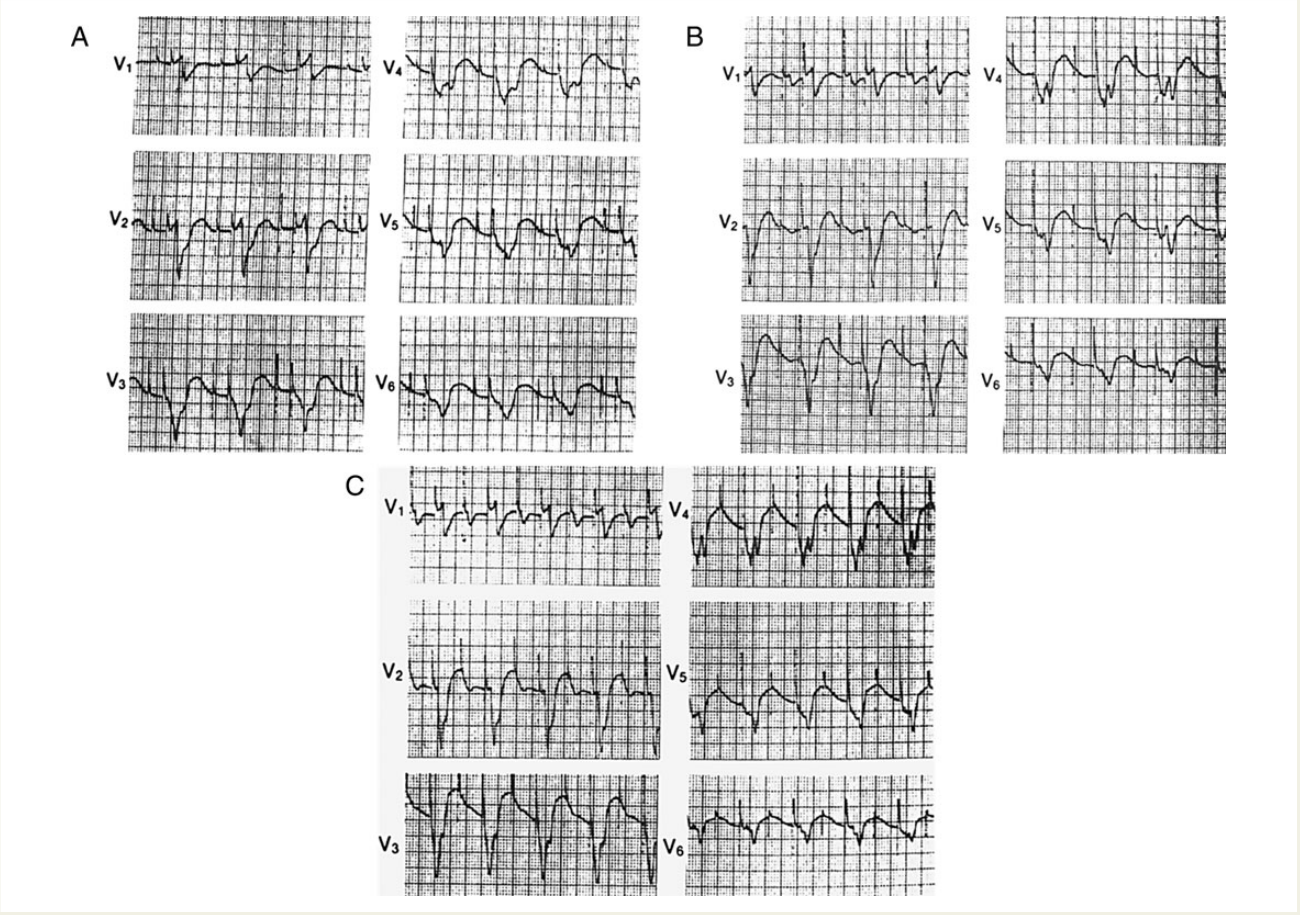

Figure 3 Hyperkalaemia-induced loss of atrial capture. (*A*) Electrocardiogram (leads V1–V6) showing the failure of atrial capture during DDD pacing in a patient with severe congestive heart failure ($K = 6.3$ mEq/L). The paced QRS complex is widened to 0.36 s. Pacemaker variables: lower rate = 70 ppm, AV delay = 200 ms, atrial output = 8.1 V at a 1.0 ms pulse duration, and ventricular output = 5.4 V and 0.6 ms pulse duration. (*B*) Electrocardiogram showing restoration of atrial capture a few minutes after initial treatment of hyperkalaemia. The pacemaker variables are the same as in (*A*). The duration of the QRS complex has shortened to 0.30 s. The interval from the atrial stimulus to the isoelectric segment of the PR interval measures ~0.22 s, and represents the delay in interatrial conduction. (*C*) Electrocardiogram recorded 24 h after (*A*). Pacemaker variables: lower rate = 90 ppm (increased from 70 ppm because of congestive heart failure and ventricular ectopy), AV interval = 300 ms, atrial output = 5.4 V at a 0.6 ms pulse duration, ventricular output = 5.4 V at a 0.6 sms duration. The QRS complex has further shortened to 0.24 s. The interval from the atrial stimulus to the isoelectric segment of the PR interval has shortened to 0.16 s. (Reproduced from Barold SS *et al.*[31] with permission.)

Latency

The delay from the pacing stimulus to the onset of ventricular (or atrial) depolarization is called latency. An isoelectric onset of the QRS complex in one or a few leads can mimic latency. Latency can only be evaluated by looking at a 12-lead ECG (preferably at a fast recording speed) to rule out an isoelectric initial part of the QRS complex. The normal values for right-sided stimulation measure <40 ms. Prolonged latency is also known as first-degree pacemaker exit block. Latency occurs in hyperkalaemia, severe myocardial disease, myocardial infarction (such as RV infarction), antiarrhythmic drug toxicity, and variant (Prinzmetal) angina. First-degree ventricular pacemaker exit block in hyperkalaemia can progress to second-degree Wenckebach (Type I) exit block characterized by gradual prolongation of the interval from the pacemaker stimulus to the onset of the paced QRS complex ultimately resulting in an ineffectual stimulus (*Figure 5*).[37–40] The pacing disturbance may then progress to 2 : 1, 3 : 1 exit block, etc., and eventually to complete exit block with total lack of capture. Total unresponsiveness to ventricular stimulation has been reported with a potassium level of only 6.6 mEq/L. This response can only be interpreted against the background of a severely diseased heart. Comparable elevated serum potassium values can be seen on a daily basis in patients undergoing dialysis with no major consequences in the absence of advanced heart disease. Hyperkalaemia-induced forms of ventricular pacemaker exit block are potentially reversible (as is antiarrhythmic drug toxicity especially with Types I and III agents) unlike many other causes of increased latency that occur predominantly in severe or terminal myocardial disease often with a combination of ischaemia, acidosis, hypoxia, antiarrhythmic drugs, and also hyperkalaemia. Exit block (increased

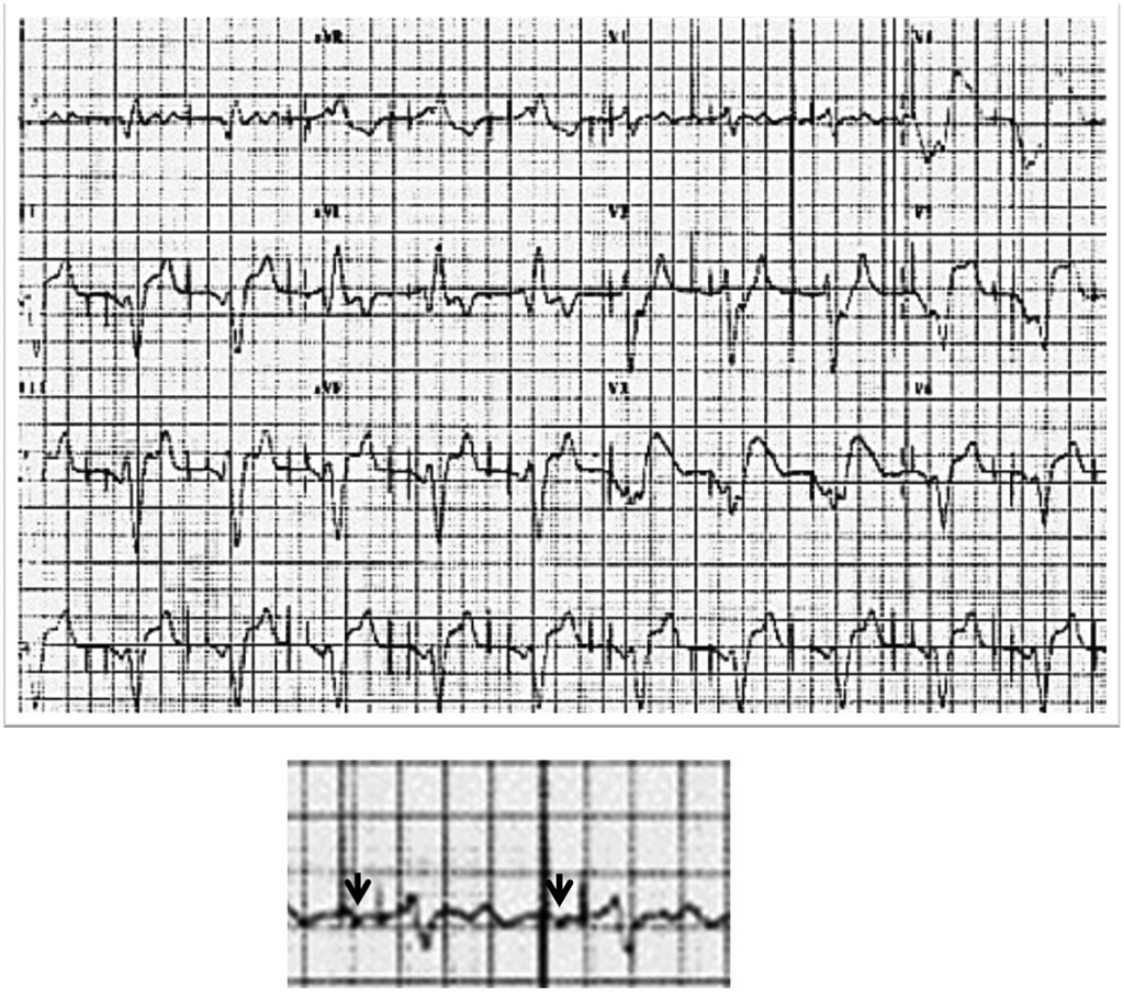

Figure 4 Twelve-lead ECG considered to show pacemaker failure to capture in the atrium and ventricle during hyperkalaemia ($K = 8.3$ mEq/L). There is apparent loss of atrial capture best seen in the lower rhythm strip. However, magnified lead V1 at the bottom shows successful atrial capture. Atrial capture is difficult to determine because of P-wave attenuation and atrial conduction delay caused by hyperkalaemia. There is consistent ventricular capture with a very wide paced QRS complex (0.36 s). In the precordial leads, the QRS complex exhibits an initial negative, shallow, and wide deflection followed by an rS deflection. The unusual early part of the QRS complex is typical of hyperkalaemia-induced delayed intraventricular conduction in hyperkalaemia which was interpreted as failure of ventricular capture. (Reproduced from Kahloon *et al.*[29] with permission).

latency) with Wenckebach or second-degree Type I exit block is always abnormal during conventional RV pacing at physiological rates. In cardiac resynchronization with otherwise uncomplicated biventricular pacing, latency linked to left ventricular (LV) pacing from the coronary venous system (unrelated to other causes of the pacemaker exit block such as hyperkalaemia, metabolic disorders, or drug toxicity) may produce suboptimal haemodynamics associated with an ECG showing the pattern of RV pacing because LV depolarization is delayed and overshadowed by RV stimulation.[41,42] Left ventricular pacing exit block may occasionally progress to second-degree type I pacemaker exit block. The electrical and haemodynamic problem of LV pacing exit block can often be corrected by advancing LV stimulation by programming the interventricular (V–V) delay.

During hyperkalaemia, an RV pacemaker stimulus near the capture threshold excites the surrounding myocardium more slowly than a suprathreshold stimulus. This latency may be related to non-homogeneous propagation of excitation, local changes in conduction near the electrode, failure of impulse propagation due to depression of intra-atrial or intraventricular conduction, or an increase in refractoriness. Increased latency usually depends on the amplitude and rate of stimulation (*Figure 6*). Consequently, with the first-degree exit block, an increase in the pacing rate often leads to prolongation of the latency interval. An increase in the amplitude of the stimulus may shorten the latency interval and convert Type I second-degree to first-degree pacemaker exit block (*Figure 6*). These responses are consistent with the concept of a virtual electrode.[43,44]

Changes in spontaneous intraventricular conduction

Evaluation of spontaneous beats, if any, may yield important clues for the diagnosis of hyperkalaemia. The development of bundle branch or bifascicular block generally reflects the progression of disease. However, when there is a spontaneous rhythm, the recent occurrence of typical right or left bundle or bifascicular block (even fascicular block) hyperkalaemia should be considered in the differential diagnosis of an intraventricular conduction block in the heart failure patient, who often takes potassium-retaining drugs and especially if the conduction disorder occurs suddenly.[45–48] The diagnosis of true bundle branch block may be helped by remembering that hyperkalaemia affects the entire QRS complex in contrast to the bundle branch block, which affects only a specific part of the QRS complex.

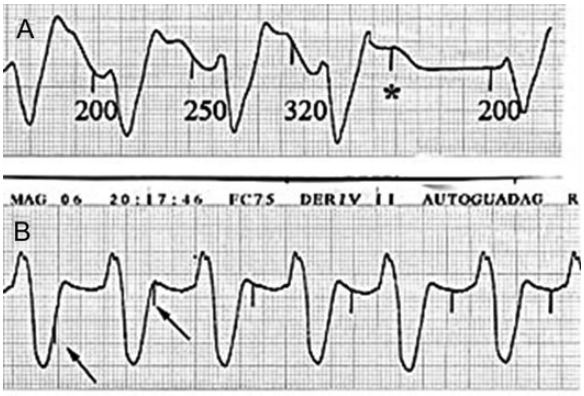

Figure 5 Effect of hyperkalaemia on pacemaker function at $K = 9.1$ mEq/L. Note the very wide QRS complex. (*A*) Wenckebach type I pacemaker exit block showing gradual increase of the stimulus to the QRS interval (shown in milliseconds) until there is failure to capture (asterisk). This is followed by shortening of the spike to the QRS interval in the beat after the ineffectual stimulus. (*B*) Ventricular undersensing with spike on the T-wave. (Reproduced from Shiraldi *et al.*[34] with permission.)

Managed ventricular pacing

The goal of managed ventricular pacing (MVP) is to achieve 'functional' atrial pacing in the safe context of dual-chamber pacing. In the Medtronic MVP system, the AS–VS or the AP–VS intervals (AP, atrial paced event; AS, atrial sensed event and VS, ventricular sensed event) can be very long and do not terminate with a ventricular paced event. Lin and Francisco[49] described a patient whose MVP pacemaker showed AP–VS intervals of 560 ms during hyperkalaemia ($K = 7.7$ mEq/L). Correction of hyperkalemia restored the basic AP–VS interval of 360 ms. This example highlights how hyperkalaemia can induce or aggravate spontaneous first-degree or Wenckebach second-degree AV block during MVP which may cause pacemaker syndrome or degrade LV function.[50,51] These abnormalities are probably uncommon because hyperkalaemia-induced bradycardia promotes ventricular pacing and pacemaker-induced AV block should not be interpreted as a form of latency.

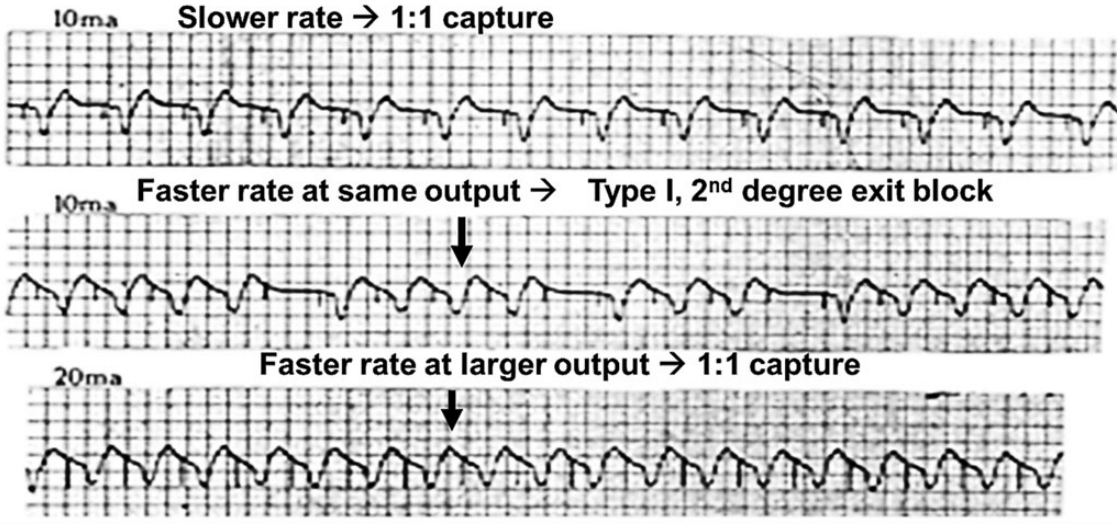

Figure 6 Pacemaker latency and type I pacemaker exit block during temporary pacing in a patient with hyperkalaemia. Top: At a pacing rate of 75 ppm at 10 mA. There is latency which was present in all the 12-ECG leads. Middle: When the pacing rate was increased to 107 ppm, and the output maintained at 10 mA, Type I Wenckebach pacemaker exit block occurred with gradual prolongation of spike to the QRS interval ultimately resulting in an ineffectual stimulus. Bottom: At the identical rate of 107 ppm, when the output was increased to 20 mA, 1 : 1 capture supervened. The tracings show the importance of the pacing rate and the output in the development of the pacemaker exit block during hyperkalaemia (Reproduced from Barold SS *et al.*[22] with permission.)

Managed ventricular pacing often induces functional AAI (R) pacing so that the new development of bundle branch block in the conducted QRS complex may be revealed to allow its relation to hyperkalaemia to be determined.

Biventricular pacing

Hyperkalaemia-induced T-wave oversensing during biventricular pacing will reduce the 'dose' of cardiac resynchronization therapy (CRT).[52] In the acute setting, when a decrease in sensitivity to an ICD system may be undesirable, T-wave oversensing may be abolished by programming to monochamber LV pacing or changing the V–V interval.[53,54]

In CRT patients with hyperkalaemia-induced delayed intraventricular conduction and/or T-wave oversensing may disrupt ventricular resynchronization, and precipitate or aggravate heart failure which is reversible upon restoration of the normal potassium level.

Implantable cardioverter-defibrillators

Inappropriate shocks

Oversensing of the spontaneous and/or the paced T-wave has been reported in patients with ICDs designed with a high ventricular sensitivity (*Table 1*).[35,54–58] Oversensing and the electrographic abnormalities disappear with normalization of potassium levels. In one case with inappropriate shocks, the amplitude of the spontaneous T-wave in the ventricular electrogram was measured and found to be higher during hyperkalaemia compared with the normal situation (*Figure 7*). Oversensing of the paced T-wave is benign but sensing of the spontaneous T-wave may cause the inappropriate delivery of a shock (*Figure 8*) (*Table 1*). The ventricular electrogram may show reversible fractionation related to hyperkalaemia-induced conduction delay. Triple counting (two QRS deflections and one T-wave signal) has been reported with hyperkalaemia.[58] On this basis, hyperkalaemia with a deranged ventricular electrogram should be ruled out with the new development of double QRS counting or triple counting that results in shock delivery.[59,60]

High-voltage impedance

Kuriachan *et al.*[61] recently reported that hyperkalaemia can increase the superior vena cava (SVC) to RV coil high-voltage impedance. In one patient with unimpressive symptoms, the SVC to RV coil impedance rose from 74 to 118 Ω when the potassium level was 8.2 mmol/L. The elevated SVC to RV coil impedance triggered an audible lead integrity alert (>100 Ω) but the pacing and defibrillation impedances were raised but not enough to trigger an alert. The elevated SVC to RV coil impedance trended back to normal levels as the potassium levels normalized. A second patient showed an increase of the SVC to RV coil impedance from 40 to 76 Ω with a potassium level of 6.5 mEq/L when the other impedance parameters were normal. This patient also showed a downward trend in the SVC to RV coil impedance as the potassium levels normalized. Kuriachan *et al.*[61] postulated that the basically selective rise of the SVC to RV coil impedance (as opposed to other impedances) was related to the presence of cardiac tissue and blood between the two coil defibrillation electrodes. Their observations suggest that it might be possible to diagnose hyperkalaemia in patients with a dual-coil ICD when they are either asymptomatic or minimally symptomatic. This measurement is only possible in patients with a dual-coil-defibrillation lead. With a dual-coil lead, there are two ways of obtaining the measurement: (i) The RV–SVC impedance is automatically displayed by the programmer together with the impedance of the programmed shock vector or (ii) the shock vector has to be programmed to a new vector from RV to SVC to determine the 'SVC' impedance. This unusual impedance response to hyperkalaemia should be studied in a large number of patients before advocating it for routine use.

Therapy

If hyperkalaemia is strongly suspected, specific therapy that includes device reprogramming should be initiated as soon as blood is drawn

Table 1 Reports of hyperkalaemia-induced T-wave oversensing by ICDs

Reference	K level (mEq/L)	Sensing paced T-wave	Sensing spontaneous T-wave	Inappropriate shocks	EGM fractionation	Comments
Arthur and Kaye[35]	6.7	+	No	No	–	–
Hosaka *et al.*[55]	8.3	+	+	+	No	VEGM showed larger T-wave
Koul *et al.*[56]	6.1	+	+	+	No	CRT-D device
Oudit *et al.*[57]	8	No	Slow VT (100–125 b.p.m.)	+	+	Programmed VF therapy: 176 b.p.m.
Xu *et al.*[54]	6.2	–	+	+	No	Less prominent T-wave in VEGM after correction of hyperkalaemia
Khan *et al.*[58]	7.6	–	+	+	Wide VEGM	Triple counting (two QRS signals and one T-wave)

ICD, implantable cardioverter-defibrillator; V, ventricular; VT, ventricular tachycardia; EGM, electrogram; VEGM, ventricular electrogram; CRT-D, cardiac resynchronization therapy-defibrillators; VF, ventricular fibrillation.

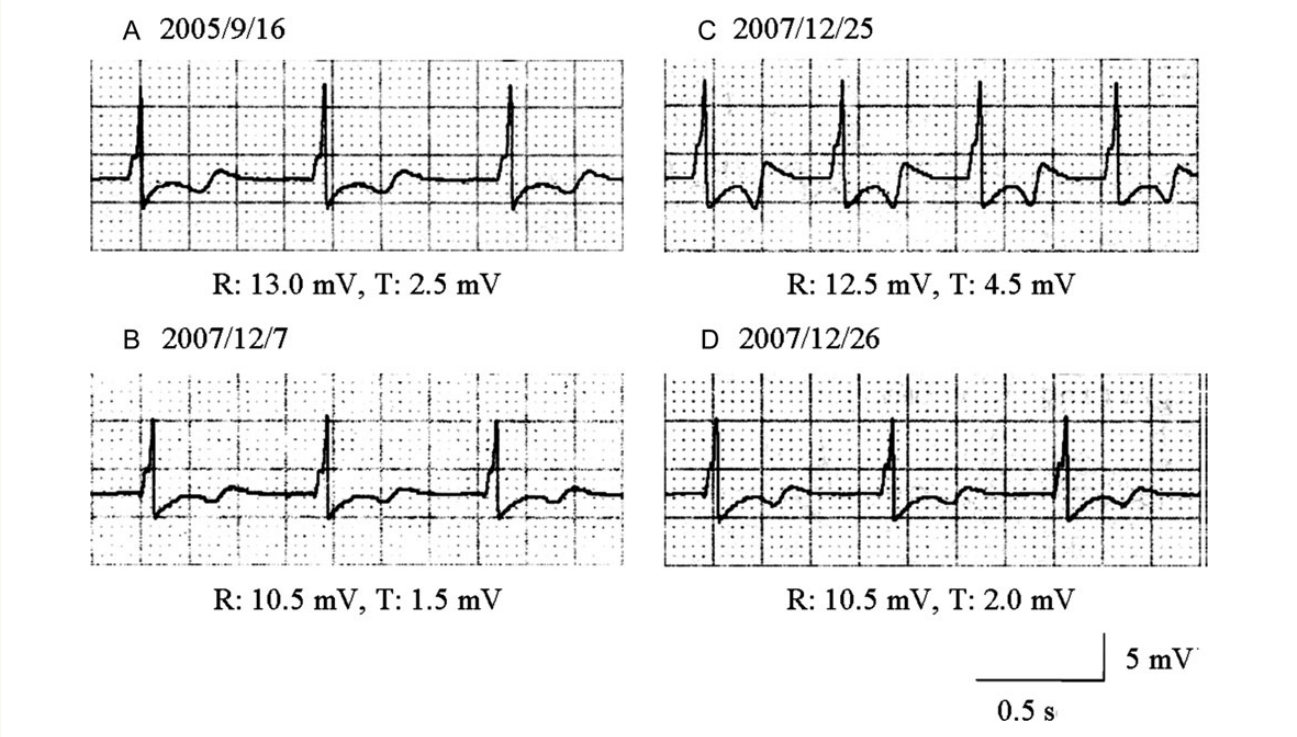

Figure 7 Electrograms recorded from an ICD during hyperkalaemia. (*A*, *B*) Before the onset of hyperkalaemia-induced inappropriate shocks, the R- and T-waves were stable and no T-wave sensing was observed. (*C*) At the time of admission, to the hospital, ($K = 8.3$ mEq/L), the T-wave amplitude was increased and peaked, while the R-wave was stable. (*D*) After treatment of hyperkalaemia, the T-wave configuration returned to baseline. (Reproduced from Hosaka *et al.*[55] with permission.)

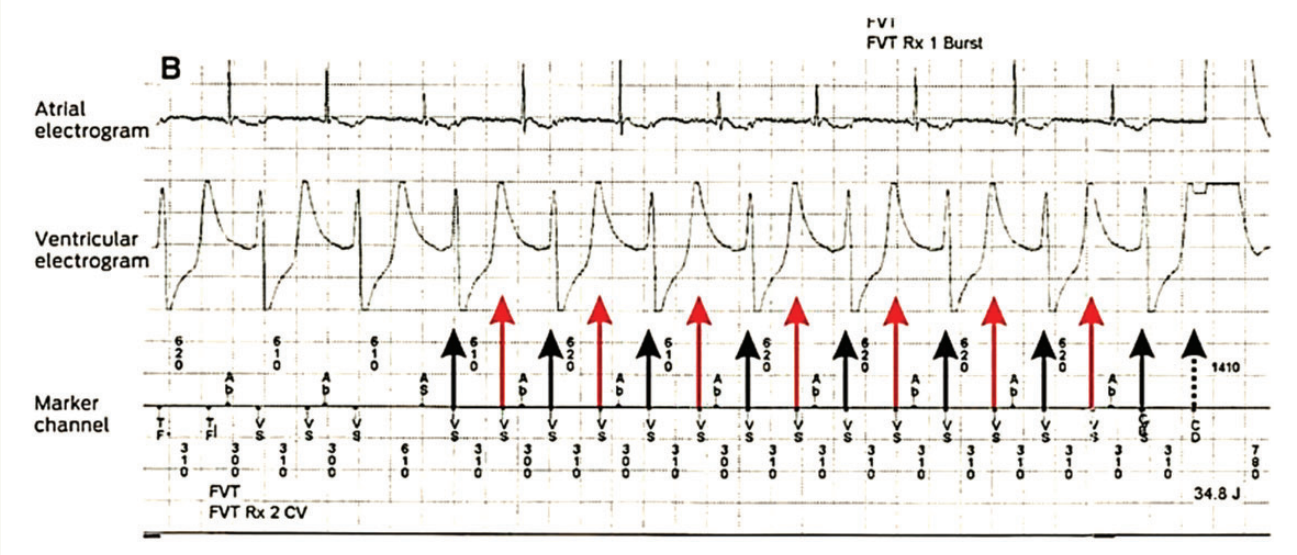

Figure 8 Hyperkalaemia-induced T-wave sensing by an ICD ($K = 6.2$ mEq/L) resulting in double counting and the delivery of an inappropriate shock (34.8 J). The red arrows point to the oversensed T-waves. VS, ventricular sensed event; Ab, atrial event in the blanking period; CD, delivery of shock (Reproduced from Xu *et al.*[54]).

for K determination without waiting for the result. Failure to capture or substantial latency requires temporary programming of the atrial and ventricular outputs to maximum voltage and a wider pulse duration. T-wave oversensing requires temporarily programming a lower ventricular sensitivity (higher numerical value). The status of atrial capture during hyperkalaemia can be difficult to determine especially with a relatively short programmed AV delay. If there is lack of atrial capture, device telemetry can be diagnostically helpful by showing atrial deflections unrelated to atrial stimuli. Evaluation of atrial capture may require programming a longer AV delay and recording the ECG at double standardization. With prolonged atrial latency, the AV delay may have to be temporarily prolonged to provide better haemodynamics by improving the AV relationship.

Conclusion

Based on the typical electrocardiographic manifestations of hyperkalaemia such as a very wide paced QRS complex in the absence of other obvious causes, the diagnosis becomes relatively simple. Programming higher atrial and ventricular outputs may be helpful in temporary high-threshold situations and increased latency. Hyperkalaemia-induced T-wave oversensing may cause inappropriate ICD shocks. Measurement of impedance from the RV coil to the SVC may become important in the detection of hyperkalaemia in asymptomatic or minimally symptomatic patients. Pacemaker patients suspected of having hyperkalaemia should be treated immediately including reprogramming of the cardiac rhythm device after drawing blood for testing without waiting for the result of the K level because their condition can deteriorate quickly especially when there is no atrial capture in patients with heart failure and poor LV function.

Conflict of interest: none declared.

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Remote magnetic ablation of atrial fibrillation is safe and feasible in the presence of a left atrial appendage closure device

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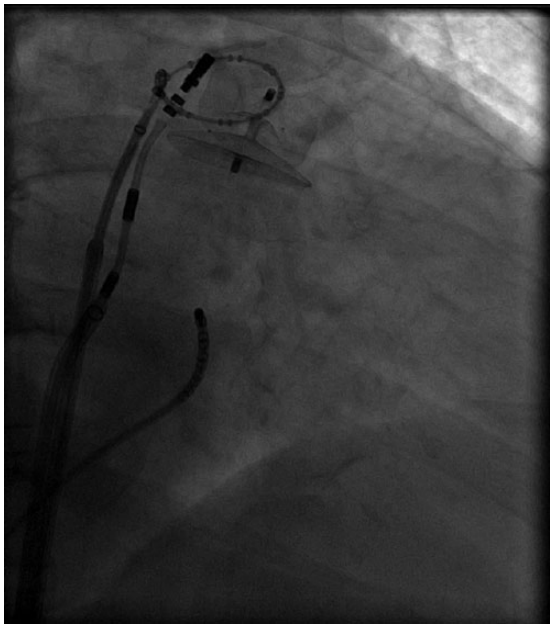
A 75-year-old female patient ($CHA_2DS_2VAS_c = 4$) was referred to our institution for recurrent atrial tachycardia (AT) after two previous radiofrequency persistent atrial fibrillation (AF) ablation procedures. Owing to severe epistaxis under a vitamin K antagonist, an Amplatzer Cardiac Plug ACP (St. Jude Medical, Inc.) was implanted in the left atrial appendage. Three months later, because of drug-refractory paroxysmal AT, a remote magnetic navigation (RMN) (Epoch, Stereotaxis Inc.) ablation procedure consisting of successful pulmonary vein reisolation (Navistar RMT Thermocool, Biosense Webster), and mitral isthmus (MI)-dependent AT ablation, led to AT interruption and MI block completion (*Figure*). Transoesophageal echocardiography did not reveal any leak around the ACP device at the end. On the next day, the patient was discharged under aspirin alone, and was free from any atrial arrhythmia and embolic event during the follow-up after 6 months.

Remote magnetic navigation is characterized by a soft magnetic catheter tip. One of the most serious complications associated with ACP is device embolization (1.9%), especially in the early months after implantation. Remote magnetic navigation for RF AF/AT ablation is feasible in the presence of an ACP and may avoid unexpected mechanical deterioration of the ACP by safe manipulation.



The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/remote-magnetic-ablation.pdf.