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Review Article

The implantable automatic defibrillator: an update for emergency physicians

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ABSTRACT

The implantable automatic defibrillator (IAD) represents a recently-acquired therapeutic option for the prevention and management of ventricular arrhythmias and cardiac sudden death. A significant increase of the indications for its use has occurred over the last few years and, considering the increasing number of patients in whom an increased risk of these most severe complications may today be identified, there has been also an exponential increase in the number of device implantations. Finally, the currently most advanced devices incorporate further functions beyond that of triggering electric discharges, such as arrhythmia event recording, anti-tachycardia or anti-bradycardia stimulation functions, or cardiac resynchronisation therapy. It is thus of utmost importance for the emergency physician to know the possibilities of this new therapeutic tool in all acute-phase clinical scenarios. The present review paper addresses those aspects with the greatest interest for the emergency physicians in the context of clinical and electrophysiological background, device function and current IAD indications.

Key Words: *Defibrillator. Emergencies. Emergency clinics. Device update. Device functions. Indications.*

RESUMEN

El desfibrilador automático implantable: actualización para médicos de urgencias

El desfibrilador automático implantable (DAI) constituye una opción terapéutica de reciente adquisición para el tratamiento y prevención de las arritmias ventriculares y la muerte súbita de origen cardíaco. Durante los últimos años se ha producido una relevante ampliación de sus indicaciones y, dado el creciente número de pacientes en los que hoy se puede identificar un riesgo elevado de padecer estas gravísimas situaciones, también a un aumento exponencial del número de implantes de dispositivos. Por último, los dispositivos más actuales incorporan otras funciones además de la posibilidad de liberar descargas de corriente, como la del registro de eventos arrítmicos, las funciones de estimulación antitaquicardia o antibradicardia y la función de resincronización cardíaca. Todo ello ilustra la importancia de conocer esta nueva arma terapéutica en los escenarios clínicos de la fase aguda. En la presente actualización se discuten los aspectos de mayor interés para los médicos de urgencias relativos a los fundamentos clínicos y electrofisiológicos, funcionamiento e indicaciones actuales de los DAI.

Palabras clave: *Desfibrilador. Urgencias. Revisión. Funciones. Indicaciones.*

INTRODUCTION

The implantable automatic defibrillator (IAD) has become the treatment of choice for patients with a high risk of developing ventricular tachyarrhythmias which could endanger a patient's life. Technological improvements, a lack of confidence in the universal efficacy of pharmacological treatments and the body of evidence provided by numerous clinical trials have led to expansion in the number of indications for such devices, with an exponential growth in the number of implants (over 100,000 per annum in the United States)[1]. As a result, in the near future physicians treating acute phases will come

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across an ever increasing number of patients fitted with these devices, and will thus, need to have updated knowledge thereof specially geared for this area of care. To this end, the IAD Work Group of the Spanish Cardiology Society and the Cardiac Arrhythmia Group of SEMES have prepared this review, dealing with the current features, operation and indications of the device.

IAD COMPONENTS

An IAD has two main components: the generator and the wire(s). The generator houses the battery and all circuits required for the release of electrical impulses, the generation of electric shocks, the filtering and analysis of signals, as well as data storage. The duration of the life of an IAD generator varies considerably depending on the number of discharges and the frequency of use of anti-bradycardial stimulation, ranging between 5 and 7 years. Battery depletion is the most common replacement indicator. Given that the battery (a combination of lithium-silver and vanadium oxide) cannot release the amount of energy required in the short time frame of a defibrillation discharge, the charge must be accumulated in a condenser before it can produce an electric shock. The maximum amount of energy that it is able to store ranges between 30 and 45 Jules. A significant percentage of space within the generator is taken up by the condenser, the size of which has been become increasingly smaller over the last 10 years. The latter, and the development of lower energy biphasic discharges, has led to current IADs being implanted transvenously, in an almost identical way to conventional pacemakers, with the generator in the pectoral area (Figure 1). The latest generation of IADs weigns around 70 g. with an approximate volume of 30 cc., far from the 175 cc of the first devices[2].

The defibrillator wires transmit electrical signals from the endocardial surface of the right ventricular apex to the generator for analysis, releasing stimulation and discharge pulses to the heart. The discharge defibrillation vector is currently found between the coils located in the wires in the right ventricle and very often in the superior vena cava and the generator casing itself (Figure 1, arrows). Where appropriate, these devices may also offer an additional possibility of stimulation and sensing in the right atrium via a second lead. Devices equipped with a third lead implanted in a tributary of the coronary sinus (close, therefore, to the left ventricle) have been recommended in the last few years, seeking to achieve cardiac resynchronization via biventricular stimulation[2].

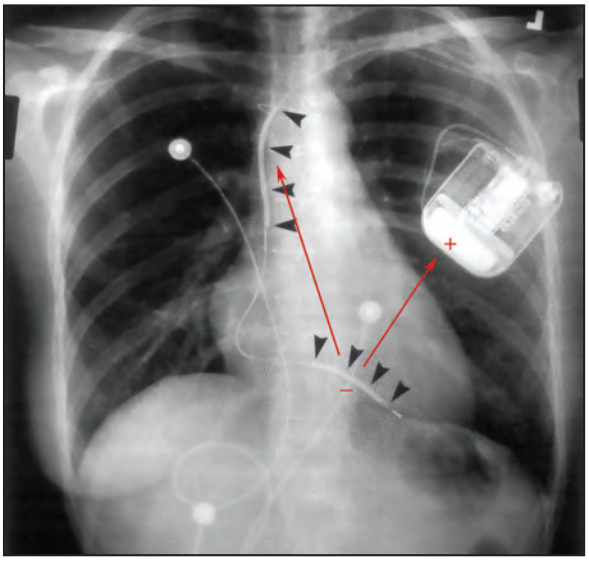

Figure 1. Image of an IAD and its components. It is composed of a generator and wires (grey arrows) which generate the defibrillation wave fronts (red arrows).

FUNCTIONS OF AN IAD

The first IAD was implanted in a human being in 1980[3], and even though originally conceived for defibrillation, improvements in the device have included an increase in the number and effectiveness of its functions. As such, synchronized electrical cardioversion, anti-bradycardial stimulation (pacemaker function), anti-tachycardial functions (overstimulation) and the possibility of registering and storing arrhythmia episodes have been added to the device. Furthermore, aside from cardiac frequency - the only initial criterion for deciding whether an arrhythmia episode merited treatment - other detection criteria have been implemented to increase diagnostic specificity, seeking above all to avoid action in the case of non-ventricular tachycardia (what is known as inappropriate therapies).

Diagnosis of tachycardia via an IAD

The device detects ventricular stimulation by means of a VD electrode. Initially, the only available parameter for deciding whether a patient was having tachycardia was ventricular frequency, which implied shock administration (only existing therapy at that time) whenever the ventricular frequency passed the cut-off frequency with the ensuing treatment of all types of tachycardia irrespective of their origin[3]. Improvements made in the IADs include programmable cut-off frequencies and more sophisticated discrimination criteria:

1) Criterion of sudden start: aims to differentiate sinusal tachycardia, which is usually of a progressive nature, from ventricular tachycardia, usually with a sudden onset[4]. 2) Frequency stability criterion: aims to differentiate episodes of atrial fibrillation, with variable RR intervals, from those of ventricular tachycardia, usual with fairly static RRs[4-6]. 3) Discrimination criteria based on the morphology or width of the electrogram: that is, whether the signal detected is similar to that of a sinusal rhythm, which would be suggestive of supraventricular tachycardia, or different, which would be suggestive of a ventricular origin. These criteria are evidently programmable both in terms of activation and differentiation parameters, which prove to be of great importance: poor programming may lead to non-detection of tachycardia of ventricular origin[7,8]. 4) In defibrillators with atrial and ventricular electrodes, additional criteria may also be activated, based on the presence or absence of atrio-ventricular dissociation or on the position of atrial activation in relation to ventricular activation[8,9].

Anti-bradycardial stimulation

The first IADs did not incorporate a pacemaker function and in those patients who needed stimulation and thus needed both devices, certain interferences generated serious malfunctions in both devices. Current IADs include the pacemaker function and such interferences no longer exist. VVI (single –chamber) was the only possible stimulation in the beginning. Nowadays there are IADs with single, dual and even triple chamber stimulation, as described in the section below. Programming the bradycardial function is similar to that of the pacemaker, and in fact, the stimulation parameters following an IAD electric shock can be programmed independently in order to avoid post-shock loss of capture due to threshold increase.

Anti-tachycardial stimulation

The first IADs only provided electric shocks. Nowadays they can provide programmable frequency stimulation sequences and number of heartbeats faster than detected tachycardia, in order to attempt to end it without resorting to shock, which is known as tachycardial stimulation (Figure 2). This therapeutic model has proven to be very useful in the treatment of ventricular tachycardia, being able to convert up to 90% thereof to a sinusal rhythm[10-12], with the added benefit that, compared with shocks this treatment is painless.

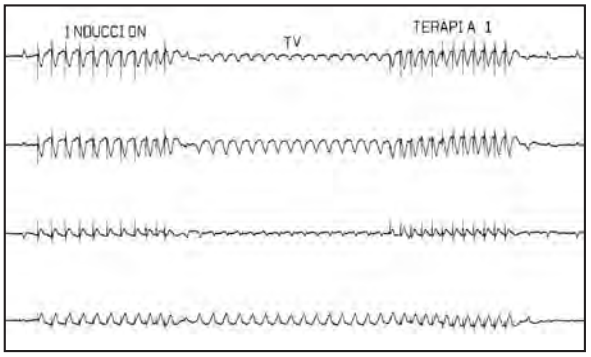

Figure 2. Electrocardiographic derivations II, III, aVR and aVL (from top to bottom). Induction of monomorphic ventricular tachycardia (TV in the picture) is shown via the device itself by means of the programmer with a stimulation train. Therapy 1 is another stimulation train which is slightly faster than the tachycardia which ends it. The action of the IAD would have been the same in the event of spontaneous clinical tachycardia.

Cardioversion and defibrillation

Defibrillation is the main function underlying the original design of the IAD to avoid sudden death of cardiac origin due to ventricular fibrillation (Figure 3). From the beginning, all efforts have focussed on increasing its efficacy, that is, to ensure that the patient will always be defibrillated. Consequently, defibrillation systems (these initially consisted of patches sutured into the epicardium via thoracotomy, and currently consist of endocardial electrodes and the generator casing itself)[13], the direction of the current emitted or the shape of the wave thereof (currently biphasic, more effective than the former monophasic wave) have been modified. Current IADs, using a single lead in the right ventricle, an activated casing and biphasic shocks, enable defibrillation of most patients (over 95%) with energy levels under 18 Jules, leaving a safety level of over 10 Jules to program the shocks maximum level of energy of the IAD. The efficacy of the therapy is thus ensured, as defibrillation thresholds in clinical practice are not accurate. This also provides a safety level of a certain number of Jules above the energy level proven to be effective[14]. Current IADs are also able to perform a cardioversion function in ventricular tachycardia (administering shocks in synchrony with the QRS) and even atrial arrhythmias (dual chamber devices which perform stimulation or shocks in the right atrium), occasionally decided by the patient (placing a device near the generator when for instance, an atrial fibrillation episode fails to subside)[15].

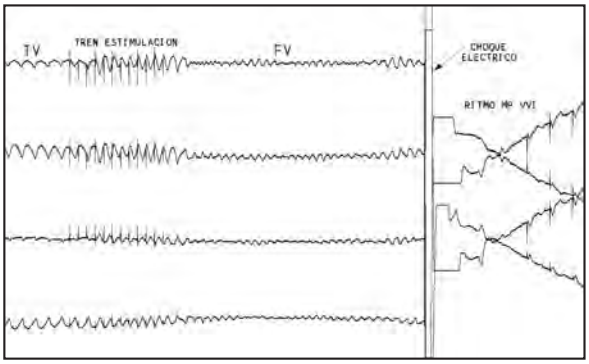

Figure 3. II, III, aVR and aVL electrocardiographic derivations (top to bottom). The figure shows how a stimulation train accelerates arrhythmia in ventricular fibrillation (FV in the graph) during a monomorphic ventricular tachycardia. In response, the IAD charges the condensers and administers an electric shock at the programmed energy level. The shock defibrillates the patient who, after the event, will have a basal frequency lower than the programmed frequency in the anti-bradycardial function. As a result of this lower frequency and for its duration, ventricular stimulation appears (pacemaker VVI in graph).

Registration and storage of episodes

IADs have a 'memory' to enable them to store patient events including all arrhythmia episodes, the date, duration, therapy which achieved reversion, R-R intervals thereof and even electrograms (as if it were an ECG of the tachycardia). This, for instance, enables one to analyse whether the episode has or has not been ventricular (may have been a supraventricular arrhythmia or an interference) and helps to program the device to avoid the release of shocks in response to supraventricular arrhythmias[16,17].

Other functions

IADs electrophysiological studies to be undertaken in patients without catheter insertion: by means of the programmer, the heart can be stimulated or shocks emitted to induce arrhythmia and verify the efficacy of the device (Figure 2). IADs also have an alert function; this consists of a series of programmable alerts which emit beeps in response to certain anomalies (excessive charge time, low battery, impedance of stimulation outside the permitted range) or in response to certain clinical situations (such as tachycardia which has needed various therapies) which will make the patient consult the arrhythmia specialist for assessment of the situation. Lastly, the placement of a magnet above the generator allows deactivation the IAD necessary (for instance, a patient with inappropriate discharges in response to a non-controllable rapid atrial fibrillation). The response magnet of the varies according to the manufacturer and; generally as opposed to what happens with pacemakers, the bradycardial function is not usually modified.

PROGRAMMING AND OPERATION OF THE IAD

Irrespective of the reason for which it was implanted, patients fitted with an IAD, and particularly those with a structural cardiopathy, can present with rapid ventricular tachycardia episodes, slower ventricular tachycardia episodes and, obviously, ventricular fibrillation[18]. For this reason, up to 3 different areas of detection may be programed with different frequencies and different therapies in each area. Thus, in response to a slow ventricular tachycardia episode anti-tachycardial stimulation is programed seeking, above all, to avoid the administration of electric shocks (particularly unpleasant for a probably fully conscious or even asymptomatic patient). If anti-tachycardia stimulation does not succeed, cardioversion shock will come into play, initially of low energy and increasing in strength if this still fails to prove effective. In the area of ventricular fibrillation, maximum energy shocks are logically programmed from the start. In tachycardia oscillating from one frequency zone to another, either spontaneously or as a result of therapy, therapies programmed for the new zone are implemented.

Time and number of beat criteria to be fulfilled by the tachycardia in order to be detected and treated are also programmed. Additionally, in the event of shocks, the time and number of beats post-charge before administering the charge (reconfirmation) must be programmed to avoid treating episodes which end spontaneously. The number of therapies to be administered by the device must also be programmed (when the IAD exhausts the therapies in a zone due to ineffectiveness thereof and tachycardia persists, the device ceases to administer therapy). In some types of tachycardia detection only and no therapy (Holter function) may likewise be programed: the IAD stores the episode information for analysis at the next review. On the other hand, this detection criteria, electrogram storage derivation alerts and IAD response to magnet acceleration are also programmed according to patient characteristics.

Lastly, the pacemaker function must be programmed: if the patient is not pacemaker-dependent, a low frequency VVI

TABLE 1. Haemodynamic consequences of left bundle branch block and congestive heart failure

A. Intraventricular asynchrony
1. Reduction in cardiac contractibility
a. Reduction in LV ejection fraction (LVEF)
b. Reduction in cardiac expenditure
2. Alteration in function of papillary muscles
a. Increase in degree of mitral failure
B. Interventricular asynchrony
1. Reduction in RV ejection volume
C. Atrio-ventricular asynchrony
1. Reduction in ventricular filling
a. Increase in pressure in right atrium
b. Increase in LV telediastolic volume

LV: left ventricle. RV: right ventricle.

is programmed to facilitate the patient's own rhythm as much as possible, thus avoiding the damaging effect that the asynchrony of ventricular contraction (caused by stimulation from right ventricle) may have over ventricular function[19], although dual and triple chamber IADs are available for atrio-ventricular sequential stimulation and ventricular resynchronization, respectively.

Cardiac resynchronization

Cardiac resynchronization (CR) is a new therapy indicated for patients with congestive heart failure (CHF) who present with a wide QRS, mainly due to left bundle branch block (LBBB). This therapy has proven to have two beneficial effects: improvement of the symptoms with an ensuing reduction in the number of hospitalizations and a reduction in the mortality rate in these patients[20,21].

Physiopathology

LBBB causes a lack of time coincidence (asynchrony) in the contraction of both ventricles (interventricular asynchrony): the contraction of the left ventricle (LV) is delayed in comparison to that of the right ventricle (RV). Furthermore, intraventricular asynchrony also takes place in the LV: the lateral wall contracts later than the interventricular septum. Likewise, atrio-ventricular asynchrony also takes place, with partial contraction of the atrium during ventricular contraction[22]. The haemodynamic consequences of such anomalies in contractibility are considerable (Table 1) and are associated with the clinical deterioration and medium to long term prognosis.

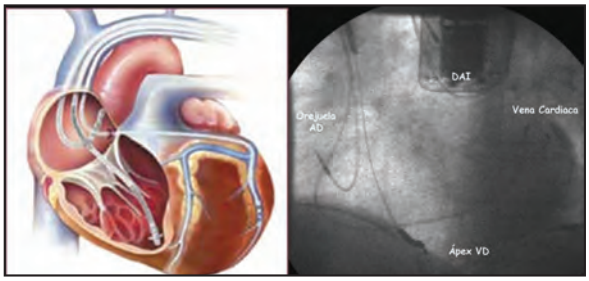

Figure 4. Distribution of electrocatheters within the cardiac cavity. On the right, radioscopic image.

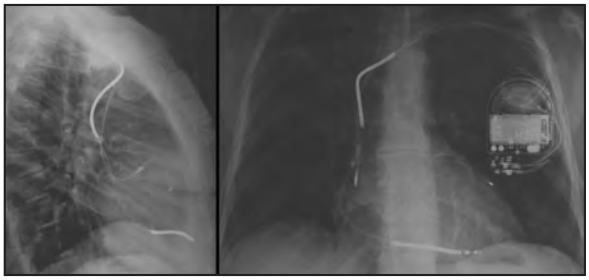

Figure 5. Lateral and posterio-anterior thorax x-ray of a patient with IAD+Resynchronizer.

Cardiac resynchronization devices

CR is achieved by simultaneous stimulation of both ventricles (biventricular stimulation) by placing an electrocatheter in the RV apex and in the LV (the latter via a lead inserted into the coronary sinus (CS) tributary). Auricular sensing, vital to achieve physiological stimulation, is done via placement of an electrocatheter in the right atrium (RA) appendage. The device is then fitted with a generator which is implanted subcutaneously in the upper right area of the chest. The devices are therefore known as triple chamber devices, given that electrodes are implanted in the RA, RV and CS (Figure 4). In patients with atrial fibrillation (AF) electrodes are only implanted in RV and SC. Lastly, CR with biventricular stimulation may be combined with endocavity defibrillation therapy (IAD) by incorporating coils in the RV electrocatheter for shock release (Figure 5).

Consequences of biventricular stimulation

The first feature observed upon activation of ventricular pacing is the narrowing of the QRS (Figure 6) compared with basal QRS and QRS produced by stimulation from the RV apex (Figure 7). This is followed by an acute improvement in functional class of all haemodynamic parameters altered by ventricular asynchrony[23]. This improvement is sustained over time and leads to an improvement in functional class, an in-

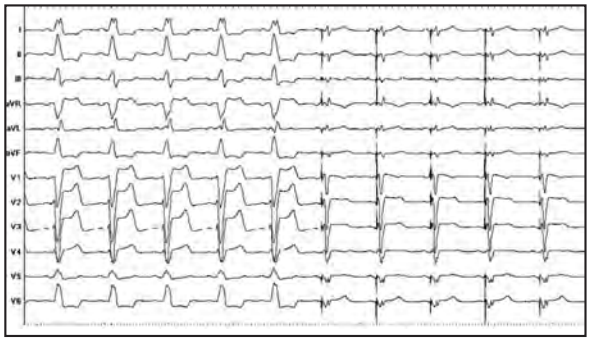

Figure 6. ECG with 12 derivations, initially LBBB image and QRS narrowing upon activation of biventricular pacing.

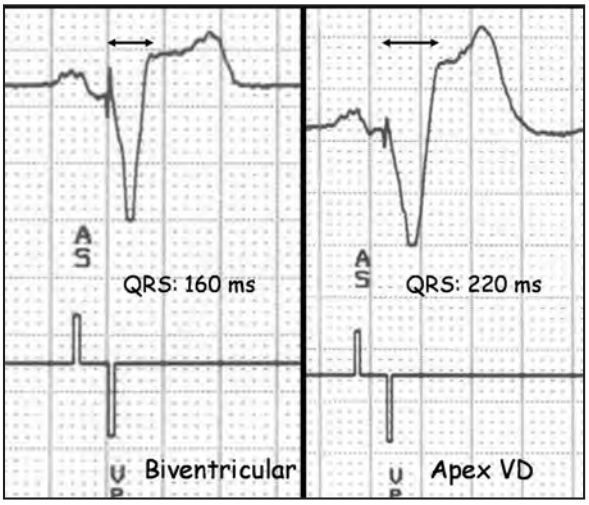

Figure 7. Telemetry of the IAD and the ECG in a channel. QRS interval with biventricular stimulation (left) of 160 ms developing to 220 ms with pacing in right ventricle apex (right).

crease in distance covered in a 6-minutes test, an increase in LV ejection fraction (LVEF), a reduction in the number of hospital admissions and a reduction in overall mortality[24-26].

Furthermore, patients with a lowered LVEF, regardless of cause, and heart failure are at high risk of sudden death due to ventricular arrhythmia[27]. This is why the combination of CR and IAD has been shown to improve the survival rate in these patients[28,29].

In order to obtain such benefits, biventricular pacing must be performed for the longest possible time throughout the day. In patients with sinusal rhythm this is achieved by suitable programming of the IAD parameters (programming an AV interval lower than the PR interval), but in patients with atrial fibrillation the aim is to achieve a cardiac frequency as low as possible so that biventricular pacing becomes the predominant activity, usually by means of β-blockers and/or digoxin.

Indications for cardiac resynchronization therapy

CR therapy is currently accepted by the American Cardiological Society as a IIa class indication in patients with dilated or ischaemic cardiomyopathy, QRS>130 msec, and LVEF ≤ 35% in patients with a NYHA III-IV functional classification, despite optimum pharmacological treatment[29]. The European Society of Cardiology recommends CR therapy for patients with reduced EF, QRS ≥ 120 ms. which remain symptomatic (NYHA II-IV), despite optimum pharmacological treatment (Ace inhibitors, spironolactone, diuretics and β-blockers) to improve symptoms (Class 1A), to reduce hospitalizations (Class 1A) and reduce mortality (Class 1B)[30].

IAD+CR therapy is indicated in patients who would benefit from an IAD and whose characteristics meet CR criteria. Nevertheless, it is not rare to find that CR is indicated in patients with a reduced LVEF with or without LBBB who need a definite pacemaker, given that pacing from the RV apex might worsen the clinical condition of such patients[31]. Among ECG alterations in which CR therapy has proven to be effective is LBBB with sinusal rhythm. In other anomalies such as atrial fibrillation or right bundle branch block, the efficacy of CR is not as clear, although some favourable data have recently been published[32,33]. In our country the rate of implants of IAD+CR is 14% of all IADs implanted in the year 2004[34]. However, an increase in this percentage is foreseeable over the next few years.

CURRENT INDICATIONS OF IAD THERAPY

Secondary prevention

In recent years several clinical trials have studied the efficacy of IAD compared to anti-arrhythmia medication (amiodarone, metoprolol, sotalol, propafenome) in patients who had ventricular arrhythmia. These studies included patients who had recovered from cardiac arrest by ventricular fibrillation (VF) or ventricular tachycardia (VT) or had presented with syncopal or poorly tolerated VT (symptoms or signs or haemodynamic compromise such as presyncope, shock, heart failure, severe angina, etc) and also had a LVEF under 25-40% (most had structural cardiopathy, largely previous myocardial infarction). After an average follow-up of 2 to 3 years, the group of patients treated with IADs showed a higher survival rate than those treated with medication[35-40]. Several sub-studies have highlighted that pa-

tients with a LVEF lower than or equal to 25% had most benefitted from the IAD, whereas in those with a relatively conserved LVEF (above 35%), the IAD failed to show any advantage over amiodarone. Patients with moderate to severe ventricular dysfunction were found to most benefit from an IAD[41-43].

However, some patients with polymorphic VF or VT may not have an apparent structural cardiopathy and may have a completely normal ventricular function, but show primary electrical alterations which tend towards development of sudden cardiac death. This is the case in patients with idiopathic ventricular fibrillation, Brugada syndrome, Long QT syndrome and short QT syndrome. In all these groups, the indication for patients who have been resuscitated from cardiac arrest secondary to such malignant ventricular arrhythmias is the implantation of a defibrillator[44].

Current indications for IAD implantation as secondary prevention are shown in Table 2. These indications are based on the clinical guidelines of various scientific societies, mainly the American College of Cardiology (ACC), the American Heart Association (AHA), the North American Society of Pacing and Electrophysiology (NASPE, currently Heart Rhythm Society), the European Society of Cardiology (ESC) and the Spanish Cardiology Society[29,45-47]. Although recommendations for an IAD are uniform in patients who have been resuscitated from sudden death by VF or VT, and in patients with a poorly tolerated VT with structural cardiopathy, the indication in patients with a well-tolerated VT and previous infarction is controversial, especially in patients who do not have severe ventricular dysfunction.

Primary prevention

In patients with a high risk of developing malignant ventricular arrhythmias, the implant of an IAD may be indicated. In patients with a previous infarction and ventricular dysfunction, the MADIT study reported a relevant increase in survival with the AID compared to conventional treatment in patients with a LVEF below 35% showing a: reduction in 2-year mortality of 54%[48,49]. In the MUSST study (patients with previous infarction, non-sustained VT in the Holter and LVEF < 40%) it was also proven that patients who were fitted with an IAD had less sudden and overall mortality than the rest[50]. Several years later, the MADIT II study (post-infarction patients with LVEF $\leq$ 30%)[51], showed a reduction in the mortality of patients with a LVEF < 25% with an implanted IAD versus those who had received optimum pharmacological treatment, although such data were not confirmed in the DINA-MIT study[52]. These results, together with a sub-analysis of the MDIT II study, which highlights that the benefit of an IAD is greater the longer patients are included after an infarction[53], underline the lack of benefits of an IAD in primary prevention, during the first weeks following an acute myocardial infarction.

In patients with dilated cardiomyopathy, several studies have failed to prove the benefit of the defibrillator versus conventional medical treatment or amiodarone treatment[54-56]. This was probably due to the fact that routine use of medication such as beta-blockers, which have shown to reduce sudden and overall mortality, was not yet common practice. Nevertheless, the latest study published on primary prevention, the SCD-HeF[57], has shown an increase in the survival of IAD carriers compared to isolated medical treatment, which, together with a recently performed meta-analysis on the IAD and heart failture[58], suggests that the IAD may be indicated in primary prevention of patients with structural cardiopathy, severe LV dysfunction and heart failure. If, in addition, patients present LBBB with a QRS > 120 msec., the combination of a defibrillator and a cardiac resynchronization device has been shown in the COMPANION study to help achieve a reduction in overall mortality and the number of hospitalizations for any cause, compared with medical treatment exclusively or associated with cardiac resynchronization therapy without a defibrillator[59], an indication which has been accepted by the European Society of Cardiology[30].

Indications for IADs in Primary Prevention are shown in Table 3 (it is important to mention that the results of the MADIT II, DINAMIT, DEFINITE, COMPANION and SCD-HeF were published after these guidelines, and thus the recommendations do not take into account the important results of these studies). Table 4 summarizes the recommendation classes of the different therapeutic indications and the levels of scientific evidence sustaining them.

The applicability of such results is discussed by various authors and whilst some defend the generalization of the MADIT II type indication (patients with previous infarction and LV ejection fraction below 30%), and the SCD-HeFT (patients with structural cardiopathy, ejection fraction below 35% and heart failure), others prefer to assign this indication to patients in whom the most benefit has been shown in such studies and sub-analyses thereof, bearing in mind other data of the clinical profile of the patient such as age or associated comorbidity.

In the case of hypertrophic cardiomyopathy, the indication for an IAD as primary prevention is controversial, and the decision must be carefully taken on an individual basis taking into account risk factors and patient clinical profile[60]. In the case of Brugada syndrome, the indications in primary preven-

TABLE 2. IAD indications. Secondary prevention

1) Cardiac arrest

Class I

- Patients resuscitated from cardiac arrest by VF or VT recorded on ECG. Not due to a reversible transitory cause (Level of evidence A).
- Patients resuscitated from cardiac arrest with no VF or VT recordings via ECG, but are assumed as cause due to effective defibrillation or to induction of sustained ventricular arrhythmia in the electrophysiological study (Level of evidence B).

Class III

- VF or VT due to transitory, reversible or treatable causes such as those taking place within the first 48 hours of the acute phase of a myocardial infarction, acute iscaemia, Wolf-Parkinson-White syndrome (Level of evidence C).
- Limited life expectancy (less than 6 months) (Level of evidence C)*
- Patients with serious psychiatric disturbances which may be aggravated by an IAD implant or prevent proper patient follow-up (Level of evidence C)*.
- Patients with severe neurological sequelae following cardiac arrest (Level of evidence C).

2) Ventricular tachycardia not associated with cardiac arrest

Class I

- Patients with sustained VT and severe haemodynamic compromise: syncope, presyncope, shock, heart failure or severe angina (Level of evidence A).

Class II

- Sustained VT without haemodynamic compromise in patients with ejection fraction ≤ 40% (level of evidence B).
- Sustained VT without haemodynamic compromise in patients with ejection fraction >40% (level of evidence C).

Class III

- Continuous VT (level of evidence C).
- Idiopathic VT (level of evidence C).
- Patients who are not high risk, with VT, who have undergone effective catheter or surgical ablation (level of evidence C).

3) Syncope without ventricular arrhythmia documented throughout

Class I

- Induction of VT or VF in electrophysiological study, with severe haemodynamic compromise (syncope, presyncope, shock, heart failure or severe angina) and structural cardiopathy with ejection fraction ≤ 40% (level of evidence B).
- Syncope not attributable to other causes and ECG pattern of Brugada syndrome (Level of evidence C).

Class II

- Induction of TV or VF by electrophysiological study, with severe haemodynamic compromise (syncope, presyncope, shock, heart failure or severe angina) and structural cardiopathy but with ejection fraction > 40%. (level of evidence C).
- Syncope not explained by other causes associated with structural cardiopathies (hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy of RV) or electrical (long QT syndrome) with a risk of developing sudden cardiac death (level of evidence B).

Class III

- Syncope without structural cardiopathy (level of evidence C).
- Syncope secondary to transitory, reversible or treatable causes (level of evidence C).

*∑ Applicable as general counter-indications both in primary and secondary prevention.
Abbreviations: IAD: implantable automatic defibrillator; VT: ventricular tachycardia; VF: ventricular fibrillation; ECG: Surface electrocardiogram; RV: right ventricle.

tion are also controversial[61-64], and we thus advise following the indications agreed by the European and American cardiological societies[65]. Lastly, in the cases of Long and Short QT syndromes, there are no clear indications for primary prevention: the IAD would be indicated in patients with recurrent syncopes not due to other causes despite pharmacological treatment, especially if they have a family history of sudden death at a young age[44].

CONCLUSION

It may, thus, be concluded that nowadays an IAD is a first line therapeutic tool in the diagnosis and treatment of ventricular arrhythmias and sudden death of cardiac origin. Thanks to its versatility and its new features, the number of implants will increase exponentially in the near future, which is why familiarization with IAD operation and indications

TABLE 3. IAD indications. Primary prevention

Class I

- Non-sustained VT in patients with previous myocardial infarction, LV ejection fraction < 40% and sustained VT inducible in the electrophysiological study (level of evidence B).
- IAD with cardiac resynchronization capacity in patients with cardiopathy, LV ejection fraction ≤ 35%, functional class III or IV and QRS > 120 ms (level of evidence B).

Class II

- Patients with previous myocardial infarction and ejection fraction below 30% (level of evidence B).
- Patients with ischaemic or dilated cardiomyopathy, with ejection fraction below 35% and functional class II or >III (level of evidence B).
- Hypertrophic cardiomyopathy with risk factor of cardiac sudden death (syncope of unclear aetiology, family history of sudden death, non-sustained VTs in the Holter, septal thickness > 30 mm. or hypotense tensile response during strength test) (level of evidence B).
- Brugada syndrome with type I ECG pattern and family history of cardiac sudden death and/or inducibility of malignant ventricular arrhythmias in electrophysiological study (level of evidence B).

Class III

- Non-sustained VT not associated with structural cardiopathy (level of evidence C).
- Non-sustained VT with structural cardiopathy and ejection fraction > 40% (level of evidence C).
- Non-sustained VAT in patients with ischaemic cardiopathy and EF > 30& without sustained VT inducible in electrophysiological study (level of evidence C).
- Asymptomatic Brugada ECG pattern with no pathological spontaneous pattern or medical history of syncope (level of evidence C).

Hypertrophic cardiomyopathy in patients without sudden death risk factors (level of evidence C).

TABLE 4. Levels of evidence and recommendation classes

Class I: Conditions for which there is a general consensus or evidence that a procedure or treatment is beneficial and effective.

Class II: Conditions for which there are conflicting data and/or discrepancies regarding the usefulness or efficacy of a given procedure or treatment.

Class III: Conditions for which there are evidence or general agreement that a given treatment or procedure is not useful or effective and may even be damaging in some cases.

Level of evidence A: Based on the information of various randomised clinical trials in a large number of patients.

Level of evidence B: Data based on one or two randomised clinical trials in a small number of patients or in non-randomized studies.

Level of evidence C: Data based on records, observational studies or general agreement among experts.

has become imperative. Despite its efficacy in the secondary prevention of sudden death (or seriously symptomatic ventricular arrhythmias), its clinical usefulness in cardiac resynchronization and the benefits of its anti-bradycardial function, its indication in primary prevention of malignant ventricular arrhythmias remains unclear. Lastly, multi-disciplinary management of these potentially high risk patients is of great importance, and this is where emergency medicine can contribute towards their improvement during the acute phase of their illness.

REFERENCES

1- Stevenson WG, Chaitman BR, Ellenbogen KA, Epstein AE, Gross WL, Hayes DL, et al. Clinical assessment and management of patients with implanted cardioverter-defibrillators presenting to nonelectrophysiologists. Circulation 2004;110:3866-9.

2- Glikson M, Friedman PA. The implantable cardioverter defibrillator. Lancet 2001;357:1107-17.

3- Mirowski M, Reid PR, Mower MM, Watkins L, Gottu L, Schanble J, et al. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. N Engl J Med 1980;303:322-4.

4- Swerdlow CD, Chen PS, Kass RM, Allard JR, Peter CT. Discrimination of ventricular tachycardia from sinus tachycardia and atrial fibrillation in the tiered-therapy cardioverter defibrillator. J Am Coll Cardiol 1994;23:1342-55.

5- Neuzner J, Pitschner HF, Schlepper M. Programmable VY detection enhancements in implantable cardioverter defibrillator therapy. PACE 1995;18:539-47.

6- Higgins S, Lee R, Kramer R. Stability: an ICD detection criterion for discriminating atrial fibrillation from ventricular tachycardia. J Cardiovasc Electrophysiolo 1995;6:1081-8.

7- Saeed M, Link MS, Mahapatra S, Morded M, Tzeng D, Jung V, et al.

Analysis of intracardiac electrograms showing monomorphic ventricular tachycardia in patients with implantable cardioverter-defibrillators. Am J Cardiol 2000;85:580-7.

8- Hintringer F, Schwarzacher S, Eibl G, Pachinger O. Inappropriate detection of supraventricular arrhythmias by implantable dual chamber defibrillators: a comparison of four different algorithms. Oacing Clin Electrophysiol 2001;24:835-41.

9- Sinha AM, Stellbrink C, Schuchert A, Möx A, Jordaens L, Lamaison D, et al. Clinical experience with a new detection algorithm for differentiation of supraventricular from ventricular tachycardia in a dual-chamber defibrillator. J Cardiovasc Electrophysiol 2004;15:646-52.

10- Saksena S, Chandran P, Sha Y, Boccadamo R, Pantopoulos D, Rothbart SY, et al. Comparative efficacy of transvenous cardioversion and pacing in patients with sustained ventricular tachycardia: a prospective, randomized, crossover study. Circulation 1993;87:1889-96.

11- Newman D, Dorian P, Hardy J. Randomized controlled comparison of antitachycardia pacing algorithms for termination of ventricular tachycardia. J Am Coll Cardiol 1993;21:413-8.

12- Heisel A, Neuzner J, Himmrich E, Pitschner HF, Liebrich A, Jung J, et al. Safety of antitachycardia pacing in patients with implantable cardioverter defibrillators and severly depressed left ventricular function. PACE 1995;18:137-41.

13- Saksena S, An H, Merha R, De Groot P, Krol RB, Burkhardt E, et al. Prospective comparison of biphasic and monophasic shocks for implantable cardioverter defibrillators using endocardial leads. Am J Cardiol 1992;70:304-10.

14- Fromer M, Brachmann J, Block M, Siebels J, Hoffman E, Almendral J, et al. Efficacy of automatic multimodal device therapy for ventricular tachyarrhythmias as delivered by a new implantable pacing cardioverter-defibrillator. Results of a European Multicenter Study of 102 implants. Circulation 1992;86:363-74.

15- Boodhoo L, Mitchell A, Ujhelyi M, Sulke N. Improving the acceptability of the atrial defibrillator: patient-activated cardioversion versus automatic night cardioversion with and and without sedation (ADSAS 2). Pacing Clin Electrophysiol 2004;27:910-7.

16- Hook BG, Marchlinski FE. Value of ventricular electrogram recordings in the diagnosis of arrhythmias precipitating electrical device shock therapy. J Am Coll Cardiol 1991;17:985-90.

17- Marchlinski FE, Gottlieb CD, Sarter B, Finkle J, Hooke B, Callans D, et al. ICD data storage: value in arrhythmia management. PACE 1993;16:527-34.

18- Mont L, Valentino M, Sambola A, Matas M, Aguinaga L, Brugada J. Arrhythmia recurrente in patients with a healed myocardial infarction who received an implantable defibrillator: analysis according to the clinical presentation. J Am Coll Cardiol 1999;34:351-7.

19- Steinberg JS, Fischer A, Wang P, Schuger C, Daubert J, McNitt S, et al. The clinical implications of cumulative right ventricular pacing in the multicenter automatic defibrillator tiral II. J Cardiovasc Electrophysiol 2005;16:359-65.

20- McAlister FA, Ezekowitz JA, Wiebe N, Rowe B, Spooner C, Crumley E, et al. Systematic Review: Cardiac Resynchronization in patients with symptomatic heart failure. Ann Intern Med 2004;141:381-90.

21- Leclercq C, Hare JM. Ventricular Resynchronization: Current State of the Art. Circulation 2004;109:296-9.

22- Hernández-Madrid A, Escobar-Cervantes C, Blanco-Tirado B, Marín-Marín I, Moya-Mur JL, Moro C. Resincronización cardíaca en la insuficiencia cardíaca: bases, métodos, indicaciones y resultados. Rev Esp Cardiol 2004;57:680-93.

23- Auricchio A, Stellbrink C, Block M, Sack S, Vogt J, Bakker P, Klein H, et al. Effect of pacing chamber and atrioventricular delay on acute systolic function of paced patients with congestive heart failure delay. Circulation 1999;99:2993-3001.

24- Cazeau S, Leclercq C, Lavergne T, Walker S, Varma C, Linde C, et al. Effects of multisite biventricular pacing in patients with heart failure and intraventricular conduction delay (MUSTIC). N Engl J Med 2001;344:873-80.

25- Abraham WT, Fisher WG, Smith AL, Delurgio DB, Leon AR, Loh E, et al. Cardiac resynchronization in chronic heart failure (MIRACLE). N Eng J Med 2002;346:1845-53.

26- Cleland JGF, Daubert JC, Erdman E, Freemantle N, Gras D, Kappenberger L, et al. The effect of cardiac resynchronization on morbidity and mortality in Heart Failure (CARE-HF). N Eng J Med 2005;352:1539-49

27- Narang R, Cleland JG, Erhardt L, Ball SG, Coats AJ, Cowley AJ, et al. Mode of death in chronic heart failure. A request and proposition for more accurate classification. Eur Heart J 1996;17:1390-403.

28- Bristow MR, Saxon LA, Boehmer J, Krueger S, Kass DA, De Marco T, et al. Cardiac-Resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure (COMPANION). N Engl J Med 2004;350:2140-50.

29- Gregoratos G, Abrams J, Epstein AE, Freedman R, Hayes D, Hlatky M, et al. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to update the 1998 Pacemaker Guidelines). Circulation 2002;106:2145-61.

30- Swedberg K, Cleland J, Dargie H, Drexler H, Follath F, Komajda M, et al. Guidelines for the diagnosis and treatment of chronic Herat failure: full text (update 2005). The Task Force for the diagnosis and treatment of CHF of the European Society of Cardiology. Eur Hear Journal 2005;26:1115-40.

31- Leon AR, Greenberg JM, Kanuru N, Baker CM, Mera FV, Smith AL, et al. Cardiac resynchronization in patients with congestive heart failure and chronic atrial fibrillation: effect of upgrading to biventricular pacing after chronic right ventricular pacing. J Am Coll Cardiol 2002;39:1258-63.

32- Molhoek SG, Bax JJ, Bleeker GB, Boersma E, van Erven L, Steendijk P, et al. Comparison of response to cardiac resynchronization therapy in patients with sinus rhythm versus chronic atrial fibrillation. Am J Cardiol 2004;941:506-9.

33- Egoavil CA, Ho RT, Greenspon AJ, Pavri BB. Cardiac resynchronization therapy in patients with right bundle branch block: Analysis of pooled data from the MIRACLE and Contak CD trials. Heart Rhythm 2005;2:611-5.

34- Peinado R, Arenal A, Arribas F, Torrecilla EG, Álvarez M, Ormaetxe J, et al, en representación del Grupo de Trabajo de Desfibrilador Implantable. Registro Nacional de Desfibrilador Automático Implantable. Periodo 2002-2004. Rev Esp Cardiol 2005;58:1435-49.

35- The Antiarrhythmic Versus Implantable Defibrillators (AVID) Investigators. A comparison of Antiarrhythmic-Drug Therapy with Implantable Defibrillators in patients resucitated from near-fatal ventricular arrhythmias. N Engl J Med 1997;337:1576-93.

36- Kuck K, Cappato R, Siebels J, Rüppel R. Randomized comparison of antiarrhyhtmic drug therapy with implantable defibrillators in patients resucitated from cardiac arrest: the Cardiac Arrest Study Hamburg (CASH). Circulation 2000;102:748-54.

37- Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon R, et al. Canadian Implantable Defibrillator Study (CIDS): a randomized trial of the implantable cardioverter defibrillator against amiodarone. Circulation 2000;101:1297-302.

38- Connolly SJ, Hallstrom AP, Cappato R, Schron E, Kuck K, Zipes D, et al. Meta-analysis of the implantable cardioverter defibrillator secondary prevention trial. Eur Heart J 2000;21:2071-8.

39- Ezekowitz JA, Armstrong PW, McAlister FA. Implantable cardioverter defibrillators in primary and secondary prevention: a systematic review of randomized, controlled trials. Ann Intern Med 2003;138:445-52.

40- Lee DS, Green LD, Liu PP, Dorian P, Newman D, Grant F, et al. Effectiveness of implantable defibrillators for preventing arrhythmic events and death: a metanalysis. J Am Coll Cardiol 203;41:1573-82.

41- Domanski MJ, Saksеena S, Epstein AE, Hallstrom AP, Brodsky MA, Kim S, et al. for the AVID Investigators. Relative effectiveness of the implantable cardioverter-defibrillator and antiarrhyhtmic drugs in patients with varying degrees of left ventricular dysfunction who have survived malignant ventricular arrhythmias. AVID Investigators. J Am Coll Cardiol 1999;34:1090-5.

42- Sheldon R, Connolly S, Krahn A, Roberts R, Gent M, Gardner M. Identification of patients most likely to benefit from implantable cardioverter defibrillator therapy: the Canadian Implantable Defibrillator Study. Circulation 2000;101:1660-4.

43- Bokhari F, Newman D, Greene M, Korley V, Mangat I, Dorian P. Long-term comparison of the implantable cardioverter defibrillator versus amiodarone. Eleven –year follow-up of a subset of patients in the Cana-

dian Implantable Defibrillator Study (CIDS). Circulation 2004;110:112-6.
44- Priori SG, Aliot E, Blomstrom-Lundqvist C, Bossaert L, Breithardt G, Brugada P, et al. Task Force on Sudden Cardiac Death of the European Society of Cardiology. Eur Heart J 2001;22:1374-450.
45- Hauer RNW, Aliot E, Block M, Capucci A, Lüderitz B, Santini M, et al. Study Group on Guidelines on ICDs of the Working Group on Arrhythmias and the Working Group on Cardiac Pacing of the European Society of Cardiology. Indications for implantable cardioverter defibrillator (ICD) therapy. Eur Heart J 2001;22:1074-81.
46- Priori SG, Aliot E, Blomstrom-Lundqvist C, Bossaert L, Breithardt G, Brugada P, et al. Update of the guidelines on suden cardiac death of the European Society of Cardiology. Eur Heart J 2003;24:13-5.
47- Pérez-Villacastín J, Carmona JR, Hernández Madrid A, Marín E, Merino JL, Ormaetxe J, et al. Guías de práctica clínica de la Sociedad Española de Cardiología sobre el desfibrilador automático implantable. Rev Esp Cardiol 1999;52:1083-104.
48- Moss AJ, Hall J, Cannom DS, Daubert JP, Higgins SL, Klein H et al. for the Multicenter Automatic Defibrillator Implantation Trial Investigators. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. N Eng J Med 1996;335:1933-40.
49- Moss AJ. Implantable cardioverter defibrillator therapy. The sickest patients benefit the most. Circulation 2000;101:1638-40.
50- Buxton AE, Lee KL, Fisher JD, Josephson M, Prytowsky E, Hafley G, et al. For the Multicenter Unsustained Tachycardia Trial Investigators. A randomized study of the prevention of suden death in patients with coronary artery disease. N Engl J Med 1999;341:1882-90.
51- Moss AJ, Zareba W, Hall WJ, Klein H, Wilber D, Cannom DS, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877-83.
52- Hohnloser SH, Kuck KH, Dorian P, Roberts RS, Tech M, Hampton JR, et al. Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. N Engl J Med 2004;351:2481-8.
53- Wilber DJ, Zareba W, Hall WJ, Brown MW, Lin AC, Andrews ML, et al. Time dependence of mortality risk and defibrillator benefit after myocardial infarction. Circulation 2004;109:1082-4.
54- Bansch D, Antz M, Boczor S, Volkmer M, Tebbenjohans J, Seidl K, et al. Primary prevention of sudden cardiac death in idiopathic dilated cardiomyopathy: the Cardiomyopathy Trial (CAT). Circulation 2002;105:1453-8.
55- Strickberger SA, Hummel JD, Barlett TG, Frumin HI, Schuger CD, Beau SL, et al. Amiodarone versus implantable cardioverter-defibrillator: randomized trial in patients with nonischemic dilated cardiomyopathy and asymptomatic nonsustained ventricular tachycardia-AMIOVIRT. J Am Coll Cardiol 2003;41:1707-12.
56- Kadish A, Dyer A, Daubert JP, Quigg R, Estes NA, Anderson KP, Calkins H, et al. Prophylactic defibrillator implantation in patients with nonischemic dilated cardiomyopathy. N Engl J Med 2004;350:2151-8.
57- Bardy GH, Lee KL, Mark DB for the Suden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive Heart failure. N Engl J Med 2005; 352:225-37.
58- Nanthakumar K, Epstein AE, Kay GN, Plumb VJ, Lee DS. Prophylactic Implantable Cardioverter-Defibrillator therapy in patients with left ventricular systolic dysfunction. J Am Coll Cardiol 2004;44:2166-72.
59- Bristow MR, Saxon LA, Boehmer J, Krueger S, Kass DA, De Marco T, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic Heart failure. N Engl J Med 2004;350:2140-50.
60- Maron B, McKenna WJ, Danielson GK, Kappenberger LJ, Kuhn HJ, Seidman CE, et al. American College of Cardiology/European Society of Cardiology Clinical Expert Consensus Document on Hypertrophic Cardiomyopathy. J Am Coll Cardiol 2003;42:1687-713.
61- Brugada J, Brugada R, Antzelevitch C, Tobwin J, Nademanee K, Brugada P. Long-term follow-up of individuals with the electrocardiographic pattern of right bundle branch block and ST-segment elevation in precordial leads V1 to V3. Circulation 2002;105:73-8.
62- Brugada J, Brugada R, Brugada P. Determinants of suden cardiac death in individuals with the electrocardiographic pattern of Brugada syndrome and no previous cardiac arrest. Circulation 2003;108:3092-6.
63- Eckardt L, Probst V, Smits JPP, Bahr ES, Wolpert C, Schimpf R, et al. Long-term prognosis of individuals with right precordial ST-segment-elevation Brugada Syndrome. Circulation 2005;111:257-63.
64- Priori SG, Napolitano C, Gasparini M, Pappone C, Della Bella P, Giordano U, et al. Natural history of Brugada syndrome: insights for risk stratification and management. Circulation 2002;105:1342-7.
65- Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada R, Corrado D, et al. Brugada Syndrome. Report of the Second Consensus Conference. Circulation 2005;111:659-70.