Hearts ‘too bad’ to survive or ‘too good’ to die

See page 1251 for the article to which this Editorial refers

Sudden death is unconsciously perceived by most physicians, especially in Latin countries, as a matter of destiny. However its abrupt occurrence is in strong opposition to deaths resulting from long-standing diseases, in which the outcome is the unavoidable consequence of progressive deterioration of vital functions. Sudden death resulting from cardiac causes may also be the final event of successive and multiple occlusions of the coronary arteries, depriving the musculature of the heart of its basic nutriments and oxygen, indispensable to maintain its proper function. Although these ischaemic episodes can be clinically silent, autopsy studies have demonstrated that most of these hearts were ‘too bad’ too survive . . . On the other hand, there are also patients whose hearts were considered ‘too good’ to die because they had unexpected minor lesions. These deaths, mostly due to ventricular fibrillation despite preserved cardiac mechanical function, appear unacceptable to clinical electrophysiologists in view of their extensive current knowledge[1]. Such unexpected deaths without underlying severe myocardial lesions were originally observed in patients who died of lack of elaboration or conduction of electrical influxes. The replacement of the failing natural pacemaker and conduction system by an implanted device was later established as the most suitable medical tool. It is now clear that the pacemaker patient has almost a normal life expectancy.

The occurrence of a syncopal episode used to be the clinical event leading to pacemaker implantation. With time, specific ECG markers were identified permitting the prophylactic implantation of the device before the occurrence of the first symptom.

Sudden death due to ventricular fibrillation

Unfortunately, primary ventricular fibrillation, albeit much more frequent, is less easy to manage. Most episodes of ventricular fibrillation are successfully treated by prompt defibrillation. Surviving a first attack of primary fibrillation is certainly less likely than surviving a first episode of asystole, which is frequently followed by spontaneous resumption of electrical activity or conduction. Ventricular fibrillation occurs most frequently as a consequence of coronary heart disease with severe underlying lesions. There is no doubt that the multiple forms of coronary artery diseases, ranging from congenital anatomical anomaly to ischaemia and/or necrosis related to atherosclerosis, are the most frequent conditions leading to sudden death[2]. In some cases, acute ischaemia is the cause of severe ventricular failure. Those hearts are ‘too bad’ to survive . . .

However, in coronary artery disease as well as in cardiomyopathies including arrhythmogenic right ventricular dysplasia or acute as chronic myocarditis, some hearts seem ‘too good’ to die[3-4]. In these cases, sudden death may appear as the result of a reversible acute phenomenon. Such ventricular fibrillation occurs as a transient electric storm, although a limited mass of myocardial tissue is involved[1]. In these cases there are currently few predictors of the catastrophe[5]. The presence of late potentials, obtained after major amplification of the terminal phase of the QRS complexes studied in the time as well as the frequency domains was found to be good predictors of ventricular tachycardia but not sudden death. Epidemiological studies have shown that out-of-hospital cardiac arrest is often the first presentation of a cardiac disease without any recognized warning symptom[6,7]. Only a limited number of patients at risk can be identified[5]. Therefore the only practical approach to this problem is to rescue the victim from ventricular fibrillation as fast as possible[8].

The valuable contribution by Herlitz et al.[9] published in this issue, stresses the advances but also the limits of the efforts presently obtained in the treatment of out-of-hospital cardiac arrest over a 17-year period in the city of Göteborg (Sweden). Overall results, based on the number of patients discharged alive from the hospital, improved in the early years of the study, but returned to the initial situation thereafter. This deceptive final result was observed despite the concentrated efforts of well-equipped rescue teams. The interval between collapse and defibrillation decreased over time from 9 to 6 min, an improvement mostly obtained by the participation of bystanders[10]. However, the cause of the later decrease in success rate seems due to an increasing age and related co-morbidity. Unfortunately systematic autopsies were not performed to identify the proportion of these hearts ‘too bad’ to survive despite early intervention[11].

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Implantable rescue localizer and calling system

Possible advances in fighting sudden death are probably possible with the help of modern communication technologies. Our experience has shown that patients collapsing during jogging were efficiently rescued, thanks to an early call to paramedics by someone using a cellular phone.

From these examples it seems that we have now at hand the technology and the knowledge to identify those patients in acute arrhythmic distress who can be rescued by appropriate medical actions. As properly stressed by Herlitz et al., citizens educated in cardiopulmonary resuscitation is a key factor in rapid intervention. Such training should start in the secondary school. This strategy also requires the wide distribution and availability of automated external defibrillators.

However, we have to develop specific equipment which will allow monitoring of cardiac electric signals and function by ECG and impedancemetry, respectively. Alarm signals from this implanted device, requiring only minimal invasive intervention, have to be transmitted to radiofrequency receivers linked automatically to the closest dispatching centre. The latter transmits to the closest rescue team the nature of the alarm and the precise location of the patient.

Since many cases of sudden death occur during sleep it is conceivable that the automatic opening of doors and turning on of lighting could facilitate emergency access to the individual in distress. After basic cardiopulmonary resuscitation by bystanders the professional rescue team would intervene quickly with a portable automated external defibrillator and other assist equipment. It is also conceivable that defibrillation could be attempted by trained individuals. Automated defibrillators with ready-to-use adhesive patches hanging on the walls should be available in all strategic places. The bedroom of already identified patients at risk should be also equipped. All of these tools should facilitate early defibrillation and provide a comprehensive report of the events stored in defibrillator memory. These data are useful for optimization of rescue management. This approach is likely to save many lives. It is also encouraging to think of the changes in the mentality of trained people more inclined to take charge of the life of others.

The risk of a new episode is a well-documented possibility. However, here again the answer is at hand with the implantable defibrillator.

Implanted networking monitoring device

Implanted monitoring equipment to evaluate permanently ECG and haemodynamic parameters is likely to be the most realistic and cheapest tool to identify patients at risk. An alarm signal would be automatically transmitted to a surveillance centre or could be directly perceptible by the patient, when dangerous parameters are detected. This approach would permit the most appropriate prophylactic implantation of defibrillators following in that respect the original vision of Michel Mirowski, with whom we discussed this matter during his first visit to France a few months after the successful implantation of the device in an animal.

Therefore, we can predict that it will be increasingly possible with time, as with pacemakers, to prevent the unacceptable fatal outcome of all of these hearts which are ‘too good’ to die . . .

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References
Ventricular pacing: a promising new therapeutic strategy in heart failure. For whom?

At present long-term therapy for heart failure is entirely targeted to the modulation of neurohormonal systems, in particular the renin-angiotensin-aldosterone and the autonomic nervous systems. No therapy specific to the heart is available. Resynchronization of ventricular activity by electrical pacing might be such a therapy, but for whom? For a few selected patients or for many? And how can potential responders be recognized? These are obviously very important questions and are not specific to ventricular pacing. We must recognize that our therapeutic approach to heart failure is rather empiric. For instance, we do not use specific criteria to select patients to be treated with widely accepted classes of drugs such as ACE-inhibitors and beta-blockers. It is somewhat paradoxical that we base the prognostic stratification of our heart failure patients on a set of functional parameters which does not include neurohormonal variables, whereas the drugs we use are mainly neurohormonally active. However the definition of indicators outlining the potential responders to pacing therapy is particularly important because this therapy requires an invasive, disturbing procedure, is costly and is irreversible. We cannot be approximate; negative or even neutral effects would not be easily tolerated. Moreover the available clinical evidence shows that treated patients have extremely heterogeneous responses, at least acutely[1,2], which underlines the need for accurate selection of candidates for the treatment. Thus the question of how to select the patients is of primary importance.

The rationale of pacing interventions is that inter-and intra-ventricular conduction delays which can occur in advanced heart failure, desynchronize the mechanical activity of the ventricles thus affecting their pump performance. In fact three major types of myocardial asynergy can occur in heart failure patients. One is a progressive loss of integrity of the myocardial collagen matrix, typical of the familial cardiomyopathies but common to all dilated cardiomyopathies. The myocardial fibrillar collagen weave ensures structural integrity of adjoining myocytes and provides the means by which myocyte shortening is translated into overall left ventricular pump function. Disruption of the collagen network by altering the coordinated mechanical response of the ventricles. The consequences are both prolongation of the QRS and loss of mechanical efficiency. Another type of ventricular asynergy is intraventricular conduction delay, generated by bundle branch blocks which most frequently impair conduction through the left bundle branch. A further type of ventricular asynergy is that of regional wall motion abnormalities typical of ischaemic heart disease. Uncoordinated ventricular contraction alters regional workload and stress. The region of early activation contracts against minimal load, rapid early systolic shortening does not translate into pressure because the rest of the