Influence of CRT on sudden cardiac death risk factors

Heart rate variability (HRV) is usually reduced in patients with heart failure. This alteration is related to the catecholaminic stress imposed by the haemodynamic impairment. The catecholaminic stimulation may promote life-threatening ventricular tachyarrhythmias with a high risk of sudden cardiac death. HRV measurement is an elegant and reliable method to evaluate the vagal-adrenergic balance of patients, and is part of the assessment of sudden cardiac death risk factors.

Lawo (24-1) used the implanted device to record continuously HRV after implantation of the CRT device. As for any parameter continuously monitored by an implanted device, precision of instantaneous information is poor, but long-term surveillance of the parameter is feasible with an implanted system but with incomparable significance of the collected data. Lawo showed that HRV increases after CRT, suggesting that HRV measurement is an indirect parameter to evaluate the decrease in the catecholaminic stress, in parallel with haemodynamic data assessment.

Zanon (24-2) confirms these results in his patient population. The increase in HRV correlates with the improvement in quality of life score.

According to these findings, the patients implanted with a CRT device should show a reduction in ventricular arrhythmic episodes, which leads to the discussion of the use or the non-use of implantable cardioverter defibrillators (ICDs) in CRT. This topic is discussed in the next three abstracts of this session with conflicting results, and thus, promoting an interesting discussion.

Rucinski (24-3) analysed Holter recordings of CRT patients during the 2-week period after implantation and comparing bi-ventricular pacing and right ventricular pacing. He did not find any difference between the two pacing modes with regard to the number of premature ventricular complexes (PVCs), doublets and non-sustained ventricular tachyarrhythmias (VTs).

Vogt (24-4) analysed the outcome of his CHF patient population with respect to ventricular arrhythmias, patients being implanted with a CRT-pacemaker (PM) or a CRT-ICD according to accepted indications for ICDs. He found that 19% of PM patients showed malignant arrhythmias after CRT. Consequently, Vogt is in favour of an ICD support in CRT patients.

Nägèle (24-5) reports 15 out of 105 patients (mostly treated with beta-blockers and amiodarone) presenting with sudden cardiac death or symptomatic VT during a 1.6-year follow-up (27% patients having asymptomatic VTs in the device memory). This author also advocates routine use ICDs for CRT.

These three latter abstracts are relevant to the COMPANION trial results. This trial suggested that mortality rate is reduced in the CRT population compared with the rate in the population treated with maximal medical therapy only. The subgroup implanted with a CRT-ICD showed a further reduction in the mortality rate compared with the group implanted with a CRT-PM. Vogt and Nägèle did find results going in the same direction, suggesting that CRT-ICD devices will probably be the standard method in the future. However, Rucinski did not find any significant difference in terms of PVCs and non-sustained (NS) VTs using the conventional 24-h Holter method. Due to the brevity of assessment by this method, it seems possible that this explains the lack of difference.

If CRT-ICD devices are to be systematically used for this type of therapy, I foresee two major and another additional problems. The first concerns the economic impact of this decision. Who were the patients who died in the CRT-PM group of COMPANION? Were there patients who had conventional indication for ICD in the PM population? As a matter of fact, due to the policy of the Ministry of Health in France, for example, which still does not accept reimbursement of ICDs, no one can imagine why use of ICDs systematically in candidates for CRT in this country!

The second aspect is the haemodynamic impact of the use of ICDs compared with pacemakers. In theory, there should not be any difference between the methods. In practice, the implantation of a CRT-PM does not hesitate to place the right ventricular lead at unconventional sites within the right ventricle (RV), but the same physician may be reluctant to implant the right ventricular lead at another site than the right ventricular apex for defibrillation threshold purposes. Consequently, as Kutarski suggested in the session on mechanisms of action of CRT, the concept of the systematic implantation of the RV lead at the RV apex for ICDs may not be optimal for all CRT-ICD patients and should be revisited.

The last problem of the systematic use of ICDs is the probable higher risk of infection. The already prolonged implant procedure for CRT-PMs compared with the implantation time of conventional PMs, is even longer because of the defibrillation threshold tests that are performed at the end of the procedure. These tests may impose a lead configuration change in order to achieve safe defibrillation in those patients with damaged and dilated ventricles. As infection rate is correlated with the duration of operation, and as ICDs are still larger than PMs, infection rate may increase dramatically, with subsequent increase in cost of therapy, issues that have not previously been addressed.

According to Fernandez Lozano (24-6), bi-ventricular antitachycardia pacing seems to be more efficient over time compared with the heart failure population which was implanted with a conventional device. However, this discrepancy may be due to the better haemodynamic condition of the CRT-ICD population, again a matter for discussion of the use of ICD in CRT. In our experience, very few patients received shocks from their devices and VT episodes became much less numerous when the patient was haemodynamically improved by the CRT system.