1991 and 1998 Framingham algorithms had only slightly different discriminatory power in our populations (c-statistics of 0.68 and 0.69, respectively in Belfast and France for the 1991 algorithm as compared to 0.66 and 0.68 for the 1998 one). This suggests in practice that the variability in the measurement of LDL-cholesterol only partially contributed to the “quality” of the algorithms.

Second, the author pointed out the problem of the concordance between estimated individual’s probability of CHD events and the actual outcomes. Although we did not develop this point in our paper, we gave c-statistics (area under the receiving operative characteristics curve) at Table 4, which precisely represents this concordance for the different algorithms (Framingham and PROCAM) and populations (Belfast and France).

Finally, we agree with Dr Wierzbicki that individual risk calculation by algorithm may be largely spurious and should be interpreted critically in the light of clinical judgment.

References


J.-P. Empana
INSERM, U 508, 1 Rue du Pr Calmette
59000 Lille, France
Tel.: +33-1-45-59-51-00
Fax: +33-1-47-26-94-54
E-mail address: empana@vjf.inserm.fr

Cardioversion threshold, IRAF and beyond

We read with great interest the report of De Simone et al. in the August 2003 issue of the journal.1 The main finding of their study was that verapamil, added to flecainide or amiodarone reduced recurrences of atrial fibrillation after cardioversion. They also report that patients on flecainide required less shocks and had a lower mean cumulative energy to defibrillate than patients on amiodarone. They state that this illustrates the favourable effect of flecainide on defibrillation energy requirements. However, on the basis of their definition of successful cardioversion (i.e. at least three beats of sinus rhythm, probably indicating 3 or more atrial complexes irrespective of the site of origin in the atria) it is impossible to correctly determine and compare defibrillation thresholds in the treatment groups. In stead, absence of electrical activity in the atria immediately after a shock should have been used. The definition of De Simone et al. gauges cardioversion threshold, resumption of sinus rhythm and suppression of hyperacute immediate recurrence of atrial fibrillation (IRAF), all at the same time. In other words, the higher ‘cardioversion threshold’ may in fact have been due to a more marked depressing effect of amiodarone on atrial pacemaker activity compared to flecainide. Such an effect may impede resumption of normal sinus rhythm and facilitate IRAF. Considering the above, the authors could have taken the opportunity to report the effects of various drug combinations on threshold as well as on IRAF, i.e. recurrences occurring within 1–2 min after cardioversion.2 The fact that verapamil added to amiodarone was not associated with increased energy requirements is not discussed, but suggests a favorable effect due to reduction of IRAF.3 We feel it is important to dissect the different drug effects on threshold, resumption of normal pacemaker activity and IRAF.4 Considering the meticulous monitoring of the heart rhythm around the cardioversion, the study by De Simone et al. could have added valuable information in this respect. Such information may enhance a targeted use of antiarrhythmic drugs in the suppression of consecutive recurrence mechanisms in patients undergoing cardioversion of persistent atrial fibrillation.

References


K.L. Merckx
R.G. Tieleman
H.J.G.M. Crijns
Department of Cardiology
University Hospital Maastricht
P.O. Box 5800, 6202 AZ
Maastricht, The Netherlands
Tel.: +31-433-875-093
Fax: +31-433-875-104
E-mail address: hcri@cardio.azm.nl
(H.J.G.M. Crijns)