During Induction and Dispersion of Repolarization

First, the apolipoprotein E polymorphism may act as a susceptibility mutation for ischemic heart disease; such mutations need certain contexts before their impact on risk of disease is expressed. Perhaps a diet rich in saturated fat can explain part of a higher prevalence in cardiovascular disease associated with the epsilon4 allele in Northern Europe compared with Southern Europe.

Second, as the study by Batalla et al. included only 220 patients and 200 controls it is also possible that lack of power in that study explains the negative findings: The power in that study given a two-sided p value <0.05 to exclude the 40% and 60% increases in risk of ischemic heart disease associated with epsilon43 and epsilon44 (as observed in our study), was only 30% and 10%, respectively.

Finally, our observations of increased risk associated with epsilon43 and epsilon44 genotypes could represent chance findings. However, we find this unlikely because 1) epsilon43 and epsilon44 were (in our study and many other studies) also associated with increases in both cholesterol and triglyceride levels, explaining the increased risk of ischemic heart disease, and 2) our observations of increased risk of ischemic heart disease associated with the epsilon4 allele in men agrees with that of a previous meta-analysis including mainly men (4).

REFERENCES


Dispersion of Repolarization During Induction and Termination of Ventricular Fibrillation

I read with great interest the article by Moubarak et al. (1). The authors found that a high postshock dispersion of repolarization (PSDR) following a T-wave shock is associated with induction of ventricular fibrillation (VF); although following a defibrillating shock, it is associated with its failure and the continuation of VF. However, the authors did not mention anything about the antiarrhythmic drugs (ADs) used by the patients. The drugs were not specified and described in the study. We do not know whether the patients who failed to induce VF with T-wave shock were taking amiodarone. Amiodarone provides homogeneous prolongation of ventricular repolarization (VR) and prevents the development of reentrant circuits. During testing of the implantable cardioverter defibrillator (ICD) it is sometimes very difficult to induce and perpetuate VF with T-wave shock in patients taking amiodarone.

It is well known that different drugs, especially class 1A, class 1C, and class 3 prolong VR in a spatially heterogeneous manner, which results in increased dispersion of VR (2). In a report about propofol, which was used for general anesthesia during ICD testing, it was mentioned that propofol decreased QT interval and dispersion (3).

In light of these findings it is reasonable to suggest that different ADs used by the patients in this study (1) might have caused repolarization changes. We also have no idea about baseline QT measurements. Some patients might have demonstrated high baseline QT dispersions. Hence, patients taking amiodarone or class 1A drugs might show high or low QT intervals and dispersion, which might affect PSDR and inducibility as well as terminability of VF. Thus, without knowing which patient took which drug and baseline QT dispersions, it is not rational to attribute a high PSDR to inducibility or terminability of VF.

Other factors such as disease state, transient ischemia, electrolyte abnormalities, changes in autonomic tone, and hemodynamic stress may also modulate the PSDR and also the success rate of shock attempts. It might be quite helpful to know the PSDR between shock attempts in one patient, because PSDR can be variable between shock attempts in one particular patient or among different patients according to the factors mentioned above. In other words, one can terminate or fail to terminate VF with the same shock strength according to the probability nature of the defibrillation success curve. It would be more reliable to withhold the drugs at least five half-lives before the procedure to minimize the potential effect of drugs on preshock and postshock repolarization times.

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