Letters to the Editor

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Prediction of neurological outcome after cardiopulmonary resuscitation

The Editorial of Hijdra1 regarding our article2 deserves some critical comments because it contains several errors and misinterpretations. Hijdra errs in the definition of poor outcome in our study which was ‘persistent coma within 6 months’ but not ‘death or persistent coma’. This distinction is important because cardiac arrest victims may suffer death unrelated to neurological causes which are not predictable by determination of neuron-specific enolase (NSE). Hijdra doubts the prospective nature of our study because physicians were not blinded to the biochemical test results, a limitation that we already described in our article. Contrary to the opinion of Hijdra, our study design was clearly prospective as documented by the existence of two interim reports presented in 2000 and 2002.3,4 Moreover, the best argument that our treatment decisions during the study period were not influenced by the known NSE values is our resulting cut-off value of 80 ng/mL which was considerably higher (but not equal or lower) than the widely proposed cut-off value of 33 ng/mL that was already published before the start of our investigation.5 Just with the growing number of patients over time, we realized the most important finding of our study that the true cut-off value of NSE to predict persistent coma was much higher than initially presumed. Hijdra declares that data on patients with severe disability were not separately reported. However, this information is depicted in Figures 1 and 3 of our article showing that a distinct cut-off value of NSE with 100% specificity for the prediction of severe disability did not exist. Therefore, we strongly warn against using a cut-off value of 33 ng/mL to predict death, persistent coma, or severe disability in clinical practice as suggested by Hijdra in Table 1 of his editorial.

References


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Gray zone problem in athletes

Sudden death is the most unpleasant complication of hypertrophic cardiomyopathy (HCM), which is the leading cause of cardiac death in athletes. The accurate diagnosis of HCM is a major diagnostic problem.1,2 The level of outflow tract obstruction. Recently, an easily measured tissue Doppler index was proposed as a potentially useful method for distinguishing athlete's heart from structural heart disease.

In conclusion, it is clear that there is a significant problem of left ventricular hypertrophy which is not an accurate diagnostic criterion, theophylline spectrum may be different among individuals with the same mutation.6 It is also well recognized that some gene carriers may not have abnormal ECGs or echocardiograms.

Indeed, based on the data of the Italian national pre-participation screening programme, it may be argued that ECG is an effective tool for identifying young athletes with HCM. In addition, according to The European Society of Cardiology Consensus Statement,7 the 12-lead ECG has been proposed as a simple and cheap test for detecting cardiovascular abnormalities. Furthermore, the 12-lead ECG shows a broad range of abnormal patterns in trained athletes; however, the determinants and clinical significance of these abnormal ECG patterns in trained athletes are still uncertain. Also, Pelliccia and Maron8 have demonstrated abnormal ECGs in 40% of the 1005 athletes tested, but structural cardiac diseases were identified in only 5% of these. A specific finding in this study is a lower incidence of athlete's heart and mild morphological forms of HCM overlap.

Although echocardiography may not be cost-effective, it is a valuable non-invasive method for differentiating cardiac pathologies other than athlete's heart.9 Echocardiography is not only helpful for accurate diagnosis of HCM, but also facilitates its risk stratification, such as the level of outflow tract obstruction. Recently, an easily measured tissue Doppler index was proposed as a potentially useful method for distinguishing athlete's heart from structural heart disease.

In conclusion, it is clear that there is a still a long way to go for the discrimination of these two entities. However, it appears that evaluation of myocardial function by new echocardiographic techniques may be useful in solving this problem.

References

Implantation of cardioverter defibrillator in post-myocardial infarction patients: when and how?

We read with great interest the recently published guidelines in the European Heart Journal ‘for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death’. In Chapter XIV, page 2115, it is recommended (class I) that an implantable cardioverter defibrillator (ICD) should be implanted at least 40 days post myocardial infarction (MI) in patients with left ventricular dysfunction with an ejection fraction (EF) of 30–40% and NYHA functional class II or III. We presume that this recommendation is based on the results of MADIT II trial.

MADIT II trial clearly demonstrated that the Kaplan-Meier estimates of survival in the two groups began to diverge at approximately 9 months and continued their separate paths thereafter favouring the group who received an ICD. In our opinion, there is no obvious or reasonable explanation for this delay. If we wanted to follow strictly the outcome of MADIT II, we would recommend an ICD implantation not earlier than 9 months post MI. There is strong evidence to support this hypothesis although its mechanism is not entirely clear. Furthermore, according to the results of MADIT I trial, clear benefit from ICD implantation was documented in post MI patients with NYHA functional class I, II, or III, a left ventricular EF <0.35, documented episodes of asymptomatic non-sustained ventricular tachycardia (VT) and inducible non-suppressible VT on electrophysiologic study. Therefore, based on the results of these two large-scale trials, it seems reasonable to treat post MI patients according to MADIT I data during the first 9 months, and according to MADIT II data thereafter. This strategy may reduce the number of unnecessary ICD implantations.

In view of the recent developments in the treatment of acute MI, it is more than evident that ischaemic cardiomyopathy is an evolving area. Does this mean that new studies are required to resolve this issue?

References


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Letters to the Editor

Role of delayed enhancement MRI in patients with acute coronary syndrome and unobstructed coronary arteries

We read with interest the paper by Assomull et al. on the use of magnetic resonance