Letters to the Editor

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A Randomized Comparison of External and Internal Cardioversion of Chronic Atrial Fibrillation

We thank Dr. Ewy1 for his editorial comment on our study2 and would like to respond to the issues he raised. The first comment refers to the low success rate that we obtained with external shocks compared with the pioneering reports of Lown et al.1,3 Their initial report1 included only 10 patients who were undergoing mitral valve operation; sinus rhythm was restored in eight of these. The second report4 was a lecture, and the results of cardioversion are difficult to compare with the results of other reports. The duration of atrial fibrillation, number of sessions of cardioversion, and number of shocks per patient were not detailed; the energy delivered was not standardized. Whereas a majority of patients in our study had coronary artery disease, hypertensive heart disease, or dilated cardiomyopathy, valvular heart disease was the cause of atrial fibrillation in 70% of patients in the Lown report.4 Note that 35% of the patients in the external cardioversion group and 45% in the internal group in our study had a history of congestive heart failure, and 36% of patients in the external cardioversion group had failed previous electrical cardioversion attempts.2 Note also the long duration of atrial fibrillation in many patients in our study, with 43% of patients in the external cardioversion group having had atrial fibrillation for >2 years.

Dr. Ewy found our description of the external cardioversion technique not detailed enough. The paddles were standard size (3×5 in.) and were applied to the chest wall manually with firm pressure. The paddles were positioned in an anterolateral fashion and not apicoposteriorly as stated in his editorial. The purpose of our study was not to compare the “optimal technique of external cardioversion” (which to our knowledge has not been universally defined) with internal cardioversion but instead to compare a conventional method of external cardioversion with internal cardioversion. We are aware that electrode position, electrode size, and other parameters may influence the results of external cardioversion.1,3 Nevertheless, our study demonstrated that internal cardioversion is more effective than and as safe as conventional external cardioversion in patients with chronic atrial fibrillation.

Transvenous internal high-energy cardioversion of chronic atrial fibrillation is not an alternative to external cardioversion. It has proved to be safe and effective and serves as an additional tool to convert patients with chronic atrial fibrillation to sinus rhythm if external cardioversion fails. We hope Dr. Ewy will try this technique in his patients with chronic atrial fibrillation who fail to respond to external cardioversion.

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References
5. Ewy GA: Effectiveness of direct current defibrillation: Role of paddle electrode size. II. Am Heart J 1977;93:674–675

Acute Reduction of Lipoprotein(a) by Tissue-Type Plasminogen Activator

We read with interest in the June 1992 Circulation the work of Hegele et al1 on lipoprotein(a) [Lp(a)] reduction in patients with unstable angina treated with tissue-type plasminogen activator (t-PA) and/or heparin. The authors attributed this effect to t-PA and excluded a possible influence of the heparin given intravenously at the same time in a first group of patients because they did not observe any significant lowering effect on Lp(a) in their group of patients treated only with heparin.

We propose a different interpretation to emphasize the value of the heparin, which is indirectly supported by another study2 that does not demonstrate any effect on Lp(a) of isolated thrombolytic therapy and is directly supported by our own previous and recent experience with the Lp(a) and heparin relation.

Table 1 summarizes the results of our latest study regarding the effect of intravenous heparin on Lp(a) in patients with coronary artery disease treated with PTCA.3 It is evident that heparin infusion produced a very marked decrease of Lp(a) (the same order of values as seen by Hegele et al in their group of patients treated with t-PA and heparin) whose statistical significance (p<0.001) persisted in blood samples taken at the end of PTCA and 4 hours later.

Similar reductions in Lp(a) values were first seen by us4 in patients with coronary heart disease who underwent coronary artery bypass graft surgery (studied before and after surgery) and, more recently, were also found in patients with chronic renal insufficiency during the course of hemodialysis. All these patients were given intravenous heparin; the resultant effect of the drug on Lp(a) was not dependent on the type of technical procedure and was possibly related to basal values of Lp(a).

We wonder whether the chance presence of cases previously treated with heparin might have had a part in the results reported by Hegele et al. In their 11 patients treated only with heparin, we noted significantly lower basal values of Lp(a) compared with those of the other group and four cases, noted by the authors,1 with a decrease of Lp(a), as we have observed in our patients, that might be ascribed only to the effect of heparin.

The interesting work of Hegele et al gives us the opportunity to propose and discuss our hypothesis, which is still in progress and requires further contributions. We hope that it will not be the object of merely speculative discussions and that the data hitherto collected will be considered sufficient for attributing to heparin this "Lp(a)-trapping" or "Lp(a)-clearing" effect, which is not yet well known to clinicians and which does not exclude a synergism with t-PA.
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