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HEART OF THE MATTER

A Lull in the War on Heart Disease

BY SIDNEY GOLDSTEIN, M.D.

By all appearances, we have reached a plateau in our attack on heart disease, and the recent meeting of the American Heart Association in Dallas provided ample evidence of this.

Numerous presentations and clinical trials were reported at the meeting, and some are included in this issue of CARDIOLOGY NEWS. But the findings reported have not moved us very far forward. Clinical trials spanning a variety of targets failed to show any significant benefits. Much of what was reported compared one drug or device with another, with an eye to safety rather than showing any improvement in mortality or morbidity.

In many cases, investigators examined new stents and antithrombotic drugs, but provided no reason to deviate from current practice. Some studies suggested that one stent might be better than another, but were often conducted in differing patient populations, which modulated most of the observed benefits and negated the differences.

In the device area, a variety of new techniques directed at improving the marginal benefits of ablation therapy for atrial fibrillation were reported. Several studies focused on trying to identify those patients who benefit the most from biventricular pacing.

More surprisingly, the results of the Multicenter Automatic Defibrillator Implantation Trial II (MADIT II) indicated that heart failure mortality increased significantly after a single discharge of the defibrillator. These observations suggested that the MADIT II patients were trading sudden death for progressive heart failure. There was no ready explanation for these observations, which generated considerable discussion among attendees about the trade-off.

The results of two clinical trials with levosimendan, a calcium-sensitizing drug for heart failure that is available in Europe but not approved in the United States, were also reported. Although patients seemed to experience improvement in symptoms acutely, morbidity and mortality were not significantly improved. It seems that current heart failure therapy with intravenous inotropic drugs supports patients through the acute episode, only to expose them to repeat and progressive failure. The cost of this support is further myocardial damage. It is becoming clear that there is a need for drugs that will improve cardiac function without causing cell damage in the setting of acute heart failure, a syndrome that is drawing increasing interest from investigators.

In the area of lipid therapy, fenofibrate was studied in patients with type 2 diabetes. There was a significant decrease in nonfatal MIs, but also a nonsignificant increase in total mortality.

With some certainty, our major therapeutic tools have stood the test of examination during the last year, but they have not provided a window to any new therapies. It seems that we have lowered cardiac mortality to a level that might make it difficult to demonstrate drug benefit.

Perhaps these observations will become more pertinent to the future of cardiology as costs and reimbursements begin to play an even greater role in our decision making. It is possible that we may be pricing ourselves out of the market as the numbers in our uninsured population burgeon. ■

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