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PCI vs. CABG for Multivessel CAD

ABSTRACT & COMMENTARY

Synopsis: *In a group of multivessel disease patients, percutaneous coronary intervention resulted in better survival and freedom from MI than did coronary artery bypass graft surgery.*

Source: Rodriguez A, et al. *J Am Coll Cardiol* 2001;37:51-58.

Eraci ii (estudio randomisado argentino cirugia angioplastia II) is a randomized, multicenter trial comparing percutaneous coronary intervention (PCI) to coronary artery bypass graft (CABG) surgery for revascularization in patients with multivessel coronary artery disease (CAD). The study was performed at seven centers in Argentina and enrolled patients between 1996 and 1998. Patients were eligible if they had severe angina (CCS class III or IV, unstable or postinfarction angina) despite "maximal" medical therapy, or a large area (> 2 vascular distributions) of myocardium at risk by perfusion imaging. Angiographic entry criteria included: two-vessel disease (one vessel with > 70% stenosis, remainder with > 50% stenosis) with significant lesions being amenable to either PCI (> 3 mm vessel) or CABG, and when functional revascularization was felt to be achievable by either as surgical or a PCI-based strategy.

The primary end point was the composite of death, nonfatal Q-wave MI, stroke, or need for repeat revascularization at 30 days. Additional follow-up at one, three, and five years was planned. Secondary end points included angina status and completeness of revascularization, assessed anatomically (by angiography or intraoperatively), as well as functionally (by perfusion imaging within 30 days), and cost.

A total of 5619 patients undergoing diagnostic catheterization were screened; of these, 1076 met entry criteria and 450 were ultimately randomized. The remaining eligible patients were not randomized due to patient or physician preference. A total of 225 patients were randomized to PCI and 225 were randomized to CABG. There were no significant differences in baseline clinical or angiographic characteristics between the groups. PCI patients received ASA, ticlopidine, and periprocedural heparin. Twenty-eight percent received abciximab and 315 Gianturco-Roubin II (GR-II) stents were implanted. There was a 92% success rate in planned vessels treated (chronic total occlusion were generally not treated), resulting in an angiographically complete revascularization rate

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INSIDE

of 50%. There were no periprocedural deaths in the PCI group and only three patients crossed over to CABG (all nonemergent) in the in-hospital phase of follow-up. Patients randomized to CABG using arterial or reversed saphenous vein conduits with standard surgical techniques, result in an anatomically complete revascularization rate of 85%. There were three perioperative deaths and 16 patients randomized to CABG that crossed over to early PCI.

The composite end point (death, Q-wave MI, stroke or need for repeat revascularization at 30 days) occurred in 3.6% of PCI patients vs. 12.3% of CABG patients ($P = 0.002$). This included significantly lower rates of death (0.9% vs 5.7%; $P < 0.013$) and Q-wave MI (0.9% vs 5.7%; $P < 0.013$) for patients randomized to PCI. Rodriguez and colleagues note that mortality was highest in patients with unstable angina, particularly those with Braunwald class III or C.

There were no differences between the groups with respect to "functionally" complete revascularization as assessed by perfusion imaging within 30 days. Late clinical follow-up at 18.5 ± 6.4 months (9-33 months) showed that PCI patients had persistently better survival (96.9% vs 92.5%; $P < 0.017$), as well as survival free from Q-wave MI (97.7% vs 93.7%; $P < 0.017$). However, as in previously published studies, patients undergoing CABG had higher rates of freedom from angina (92% vs 84.5%; $P = 0.01$) and freedom from repeat revascularization (95.2% vs 83.2%; $P < 0.001$) with 11 PCI patients (4.8%) crossing over to undergo CABG. Economic analysis revealed no differences in cost at one year. Rodriguez et al concluded that in this high-risk group of multivessel disease patients, PCI resulted in better survival and freedom from MI than did CABG.

COMMENT BY SARAH M. VERNON, MD

The efficacy of PCI vs. CABG for revascularization of patients with multivessel CAD has been evaluated in several large randomized clinical trials, most of which were conducted and published in the early to mid-1990s. All of these trials demonstrated equivalent safety and survival (with the exception of diabetic patients in BARI), but there were higher rates of recurrent angina and repeat revascularization in patients treated with PCI than in those receiving CABG. As a whole, these studies evaluated highly selected patient populations with relatively stable clinical syndromes, low-risk baseline characteristics, and with a predominance of two-vessel coronary disease. These studies were performed before the widespread use of glycoprotein IIb-IIIa inhibitors and coronary stents, which have been shown to reduce acute complications and restenosis rates in contemporary PCI procedures. In addition, surgical techniques and outcomes have also improved over the same time frame. All of these limitations make application of randomized clinical trial data to a given patient in 2001 somewhat problematic.

ERACI-II is the first published study in the "stent era" to compare PCI with CABG for the treatment of patients with multivessel CAD and, therefore, might be expected to be more reflective of current clinical outcomes. However, this study has many significant limitations. ERACI-II included a somewhat higher risk patient population than previously studied, including higher proportions of elderly, unstable angina, and three-vessel disease. However, as in previous studies, this remains a highly selected population (16% of patients screened were enrolled) that excluded many patients who would need to be considered for revascularization in clinical practice. While the PCI procedures included a relatively high rate of stenting, the Gianturco-Roubin II stent used preferentially in this trial, has subsequently been shown to be associated with high rates of restenosis and is, therefore, no longer used in the United States. In addition, the rate of GP IIb-IIIa inhibitor use during PCI (28%), while higher than in earlier published trials, remains significantly lower than most interventional laboratories today. More concerning however, is the relatively high rate of perioperative (5.7%) mortality seen in the patients undergoing CABG. Rodriguez et al attribute this to the higher proportion of high-risk patients included in the study, and indeed subgroup analysis, albeit of a rela-

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tively small sample size, suggests that adverse events may have been more common in patients with unstable angina. However, there are other potential contributors such as the longer delay between randomization to treatment in the CABG group. If perioperative mortality had been more in keeping with the majority of previous randomized controlled trails, the advantage for a PCI approach would have been less notable. Lastly, and perhaps most importantly, the choice of a 30-day primary end point may be construed as "stacking the deck" in favor of a percutaneous revascularization strategy. It is well established that surgical revascularization carries an inherently higher "up-front" risk, while the limitations of percutaneous revascularization may not become evident until later, when restenosis becomes clinically manifest. The longer-term "softer" secondary outcomes in this trial, including increased need for repeat revascularization and higher frequency of angina among patients randomized to PCI, are entirely in keeping with previously published studies.

Because of these limitations, the results of ERACI II, while intriguing, do not really demonstrate the superiority of a PCI based strategy or help to guide us in the selection of the most appropriate revascularization strategy in for a given patient. However, there is promise for continued improvement in outcomes of PCI procedures including reduction of acute complications and prevention and treatment of restenosis. Stent design, deployment technique, and adjuvant antiplatelet therapy have continued to improve in the four years since ERACI-II was initiated. This, in combination with more recent advances such as pharmacologically coated stents and brachytherapy, may "raise the bar" again and may ultimately make PCI the preferred strategy for revascularization in a larger number of patients with multivessel disease. ❖

Biphasic Shocks Compared with Monophasic Shocks in Cardiac Arrest

ABSTRACT & COMMENTARY

Synopsis: *The use of an automatic external defibrillator with a biphasic waveform is more effective for defibrillating with out-of-hospital cardiac arrest.*

Source: Schneider T, et al. *Circulation* 2000;102: 1780-1787.

Schneider and associates compared the efficacy of automatic external defibrillators (AEDs) using either biphasic or monophasic shock waveforms in patients with out-of-hospital cardiac arrest. The study involved the emergency medical services (EMS) in four cities: Mainz and Hamburg in Germany, Brugge in Belgium, and Helsinki, Finland. All EMS units involved in the trial carried either a biphasic AED that delivered a 150 joule, impedance compensated biphasic waveform shock or a monophasic AED that used either a monophasic truncated exponential or a monophasic damped sine defibrillation waveform. A daily schedule of randomly selected AED types was used in each city. After arrival at the scene, the AED appropriate for that day was placed on each victim of sudden, out-of-hospital cardiac arrest whenever defibrillator application was felt to be clinically indicated. Manual defibrillators were available for back-up in case the AED was unsuccessful. The sequence of shocks was 200, 200, and 360 joules for the monophasic units and three 150 joule shocks for the biphasic units. If three consecutive shocks failed to defibrillate or if the AED did not advise a shock be delivered, then local life support protocols were followed. The primary end point of the study was the percentage of patients with ventricular fibrillation (VF) as the initial monitored rhythm who were defibrillated in the first series of three shocks or less. Defibrillation with one or two shocks, return of spontaneous circulation, neurologic status at discharge, and survival to hospital admission and discharge were secondary end points.

The study enrolled 338 patients with out-of-hospital cardiac arrest and, of these, 246 were eligible for randomization. Ventricular fibrillation was the initial monitored rhythm in 115 individuals and these subjects formed the final study group. The groups were comparable in terms of age, gender, weight, cardiac diagnosis, and cause of cardiac arrest. Eighty-eight percent of the arrests were witnessed by bystanders. Forty-four percent of the patients received bystander cardiopulmonary resuscitation. The time from the emergency call to delivery of the first shock was 8.9 ± 3 minutes overall and this interval was not different in the two treatment arms.

Four patients in the monophasic shock group were not treated with an AED due to low amplitude VF that was not detected and identified as VF by the AED. The first shock efficacy was 59% for patients who received a monophasic shock and 96% for patients who received an initial biphasic shock. Three shocks or less defibrillated 69% of the patients in the monophasic AED group vs. 98% in the biphasic AED group. Return of spontaneous circulation was seen in 54% of the monophasic group and 76% of the bipha-

sic group (P = 0.01). Fifty-one percent of the monophasic group vs. 61% of the biphasic group survived to hospital admission (P = 0.27). There was no difference in survival to hospital discharge with 31% of the monophasic group surviving vs. 28% of the biphasic group. At the time of discharge however, cerebral performance was rated as good in 87% of the patients who were treated with the biphasic AED vs. 53% of the monophasic AED. Schneider et al conclude that the use of an AED with a biphasic waveform is more effective for defibrillating patients with out-of-hospital cardiac arrest. Larger studies are indicated to see if survival rates to hospital admission and discharge will be improved with the use of these new units.

COMMENT BY JOHN P. DiMARCO, MD, PhD

There have been numerous recent advances in external defibrillators. Biphasic waveforms were first shown to have advantages in terms of energy requirements, size, and weight in implantable defibrillators. More recently, studies using external defibrillators have also shown an advantage for the biphasic waveform in patients with both supraventricular and ventricular arrhythmias. This well-organized trial confirms the superiority of the biphasic waveform in automatic external defibrillators used by emergency medical services personnel in the field.

It is somewhat disappointing that the higher defibrillation efficacy did not translate into increased survival to hospital discharge. It is likely that delay from the time of collapse to deliver of the first shock using any waveform remains the most critical variable in survival to hospital discharge. The small amount of time required to deliver multiple shocks and the myocardial dysfunction from any additional shocks required may be much less important than the time to defibrillation. However, the increased defibrillation efficacy and the increased rate of return of spontaneous circulation are important parameters to assess in evaluating a complete resuscitation algorithm and the data presented here are quite convincing.

Although it will involve considerable expense, it is clearly time that hospitals and emergency medical systems begin a replacement cycle for their monophasic defibrillators. This study, as well as other studies, is unanimous in supporting the enhanced efficacy of biphasic units. Unfortunately, time to defibrillation of any type still remains the primary parameter influencing survival in out-of-hospital cardiac arrest. ❖

Neurally Mediated Hypotension in Chronic Fatigue Syndrome

ABSTRACT & COMMENTARY

Synopsis: *The therapy of neurally mediated hypotension with fludrocortisone does not provide reliable symptomatic improvement in patients with chronic fatigue syndrome.*

Source: Rowe PC, et al. *JAMA* 2001;285:52-59.

Rowe and colleagues conducted a study of the effect of fludrocortisone acetate in patients with neurally mediated hypotension in the setting of chronic fatigue syndrome. Patients who met Center For Disease Control (CDC) criteria for chronic fatigue syndrome were screened for other reversible causes of fatigue and had four or more of the following symptoms: impaired memory or concentration, sore throat, tender adenopathy, muscle pain, multi-joint pain, headaches, unrefreshing sleep, and postexercise malaise. All patients had had symptoms for at least six months. A Beck Depression Inventory (BDI) was completed by all patients on two occasions before the initial tilt study. A BDI score of 65 or less on the unidimensional global wellness scale was required for entry into the study. Patients then underwent tilt table tests using, in stage I, an upright tilt of 70° for up to 45 minutes. Vital signs were monitored noninvasively. If patients completed stage I of the test without symptoms or hypotension, they proceeded to stage II, which involved head-up tilt during a 2 mcg/min infusion of isoproterenol for a maximum of 15 minutes. In order to enter the study, all patients were required to manifest neurally mediated hypotension as defined as a 25 mm Hg reduction in systolic blood pressure with no associated increase in heart rate and associated symptoms of presyncope or syncope.

One hundred patients were eventually randomized in the study—50 to fludrocortisone and 50 to placebo. The mean age for the group was 37 years. Seventy-eight percent of the patients were older than age 30. Their mean weight was 69 kg. Ninety-eight percent of the patients were white and 66% were women. The mean duration of chronic fatigue syndrome was 6.4 years and 71% of the patients had a duration of symptoms greater than three years. Patients were randomized to receive fludrocortisone acetate titrated to a dose of 0.1 mg/d. Repeat tilt table testing was performed during the ninth week of treatment. Multiple measures

of depression, medical fatigue, and medical symptoms were made in the days before the first and second tilt table study and the mean for these measurements during seven days prior to the first and second study were used for the primary outcome.

There was no significant difference in the proportion of subjects with at least a 15-point improvement in wellness scores over the course of the study. Fourteen percent of the subjects in the fludrocortisone group showed this degree of improvement compared with 10% of those in the placebo group. Age, duration of therapy, and compliance with medications did not identify responders. There were no significant changes in any of the secondary outcomes as well. At the second tilt table study, a normal response was seen in nine of 48 placebo patients vs. four out of 42 patients in the fludrocortisone group.

Rowe et al conclude that therapy of neurally mediated hypotension with fludrocortisone does not provide reliable symptomatic improvement in patients with chronic fatigue syndrome.

COMMENT BY JOHN P. DiMARCO, MD, PhD

Chronic fatigue syndrome is a frustrating condition with no identifiable cause. Patients with this syndrome have a number of other complaints in addition to fatigue and the system can be physically and psychologically disabling. Several years ago, the groups at Johns Hopkins reported that a large proportion of patients with chronic fatigue syndrome had neurally mediated hypotension. The hypothesis that therapy of neurally mediated hypotension would reverse the symptoms of chronic fatigue syndrome led to the present study. Unfortunately, as shown by the data here, fludrocortisone did not have a statistical effect on either mental status or tilt table responses in patients with this syndrome.

Rowe et al admit that there are some limitations to their trial. Higher doses of fludrocortisone may have been required but many patients cannot tolerate higher doses. Some patients were excluded from participation because of either prior therapy or because of primary psychiatric or medical conditions. However, if neurally mediated hypotension was the primary physiologic abnormality in patients with chronic fatigue syndrome, one would have expected a positive result to this study.

Another major limitation of this study is the fact that both chronic fatigue syndrome and neurally mediated hypotension are quite difficult to treat. In this study, fludrocortisone did not effectively reverse either changes during tilt table testing or improve measures of fatigue or mental health. If the causal relationship between the two syndrome is to be ruled out, it would have been necessary to normalize the tilt table responses without affecting the patients symptoms. Thus, it remains possible that truly effective treatment of neurally mediated hypotension might have some effect in this condition. ❖

Accuracy of Color Doppler for MR

ABSTRACT & COMMENTARY

Synopsis: *The semi-quantitative color flow jet characteristic method for estimating the severity of MR could separate more severe from less severe regurgitation but could not determine the actual hemodynamic load in the more severe grades of MR.*

Source: Pu M, et al. *Am J Cardiol* 2001;87:66-70.

Semi-quantification of the severity of mitral regurgitation (MR) by color flow Doppler echocardiography is routinely used in most echocardiography laboratories because of its simplicity and intuitiveness. However, serious disconnects between the color jet characteristics, the patient's symptoms, or heart chamber sizes frequently occur and make most of us uneasy about the accuracy of using jet characteristics for quantitating MR. On the other hand, measuring regurgitant volume or orifice area by echocardiography is tedious and demanding. Since the relationship between these semiquantitantive and quantitative measures is not well characterized, Pu and colleagues studied 113 patients undergoing clinically indicated intraoperative transesophageal echocardiography (TEE) who had color Doppler evidence of MR. All patients had a right heart catheter from which cardiac output was measured by thermodilution. Mitral annular stroke volume was determined by pulsed Doppler at the mitral annulus (area times velocity integral) and was subtracted from the left ventricular stroke volume by catheter to derive the regurgitant stroke volume. Mitral regurgitant orifice area was determined by dividing the regurgitant stroke volume by the mitral velocity integral. These measurements were compared to the semiquantitation of MR from the color Doppler jet characteristics using a 1-4 scale with 0.5 increments. The results showed that the relationship between the quantitative and semi-quantitative measures was exponential with a steep increase in regurgitant volume or orifice area at 3+ MR or more. At MR grades less than 3+, regurgitant stroke volume was consistently less than 50 mL, but with grades more

than 3+ it ranged up to more than 200 mL. Also, there was considerable variability in the regurgitant stroke volume at all grades of MR, but especially in the higher grades: at 2+ MR, it ranged from near zero to 50 mL; at 3.5+, it ranged from 40 to almost 200 mL. Thus, at grades 3-4+, MR regurgitant stroke volume ranged four-fold and regurgitant orifice area ranged six-fold. Pu et al concluded that the semi-quantitative color flow jet characteristic method for estimating the severity of MR could separate more severe from less severe regurgitation but could not determine the actual hemodynamic load in the more severe grades of MR.

COMMENT BY MICHAEL H. CRAWFORD, MD

This study confirms what many of us who read a lot of echoes and see patients with MR have long suspected; the semi-quantitation of MR by color flow Doppler is seriously flawed. It should be pointed out that this is a TEE study; there is no reason to believe that transthoracic echo would be any better and might well be worse. There are two major findings in this study. First, the exponential relationship between the color flow grade and regurgitant volume or orifice area. Grades 0.5-2.5+ are almost indistinguishable with regards to regurgitant volume, which ranges from zero to 60 mL and shows a similar variability at all 0.5 increments in grade. According to these data to say that someone with 1+ MR has less regurgitation than someone with 2+ MR is ridiculous. However, someone with 3-4+ MR probably has a larger regurgitant volume than someone with 1+ MR. Also, the data suggest that two grade differences correlate with real differences in regurgitant volume, but there is too much overlap between adjacent grades to ascribe meaningful differences in regurgitant volumes. The second major finding is that there is a large variation in regurgitant volumes in grade 3-4+ MR (4-fold) such that it is impossible to accurately define the hemodynamic load of regurgitation in these grades. This undoubtedly explains the variability in symptoms, physical findings, and cardiac chamber size and function in patients with grade 3-4+ MR. This finding suggests that other criteria should be used for determining the hemodynamic significance of MR such as left atrial size, left ventricular size and function, pulmonary artery pressure, and symptoms. In fact, this is what research and derived guidelines on the timing of surgery for MR suggest. Among patients with severe MR by echo or angio, surgery should be considered if other clinical or echocardiographic findings are present. The estimation of regurgitation severity by regurgitant jet characteristics alone is not sufficient reason to operate. Thus, color flow grades 1-2 MR are indistinguishable and hemodynamically insignificant. These patients have a good prognosis and need not be followed closely. Grades 3-4+ MR are usually hemodynamically significant. These patients should be followed more closely and measurements of left heart chamber size and function should be performed serially.

The findings in this study suggest that we may want to consider modifying how we grade MR by echo. Some have suggested we use words such as trivial, mild, moderate, severe, but this is not much different from grade 1-4 and would have the same drawbacks. Others have maintained a 1-4 grading system but have added trivial which results in five grades. Perhaps three grades are enough; insignificant (grades 0.5-2); significant (grades 3-4 without chamber enlargement); and hemodynamically significant (grades 3-4 with chamber enlargement). Unfortunately, in this study, no data on chamber sizes and function were presented, so we do not know how such a classification system would mesh with the data in this study. Lacking a universally agreed upon system, I suggest that each laboratory agree on a system that makes sense to them, use it consistently, and educate your physician users on how to translate it to the clinical arena for patient decisions. ❖

Sudden Death in Hypertrophic Cardiomyopathy

ABSTRACT & COMMENTARY

Synopsis: *Analysis of five easily measurable variables can effectively predict sudden death in patients with hypertrophic cardiomyopathy. Patients with multiple risk factors may have a sudden death probability high enough to justify prophylactic therapy.*

Source: Elliott PM, et al. *J Am Coll Cardiol* 2000;36: 2212-2218.

Elliott and colleagues at st. george's hospital in London report on predictors of outcome in a cohort of 368 patients with hypertrophic cardiomyopathy. The influence of five variables on survival was analyzed. These five clinical variables were: nonsustained ventricular tachycardia on a 48-hour ambulatory ECG, history of syncope, exercise blood pressure response, family history of sudden death, and left ventricular wall

thickness. Patients in the study were between the ages of 14 and 65. Patients were excluded if they had a documented sustained ventricular arrhythmia or out-of-hospital cardiac arrest, or if they received amiodarone during more than 50% of their follow-up. Other forms of therapy including dual chamber pacing, calcium channel blockers, beta blockers, and other antiarrhythmic drugs were permitted. Follow-up data were collected using a questionnaire sent to all patients' general practitioners. Additional information, if required, was obtained by direct communication with the patients. An abnormal blood pressure response was measured during either treadmill exercise testing or bicycle ergometry and was defined as a failure of the blood pressure to rise or an actual fall in blood pressure during exercise. Based on prior observations, blood pressure response was classified as a risk factor only in patients less than or equal to 40 years of age.

The final group included 368 patients of whom 239 were men. The mean age at diagnosis of hypertrophic cardiomyopathy was 33 ± 14 years. In this group, 57 patients (16%) had a history of syncope, 90 (25%) had a family history of sudden death, 138 (38%) had an abnormal blood pressure response with exercise, 64 (17%) had nonsustained ventricular tachycardia during Holter monitoring, and 44 (12%) had a left ventricular wall thickness of 30 mm or more. There was no relationship between the pattern of hypertrophy (asymmetric, concentric, apical or eccentric) and survival.

The mean follow-up was 3.6 ± 2.5 years. There were 36 (9.7%) deaths among the 368 patients. Twenty-two deaths (61%) were sudden, five (14%) were from progressive heart failure, four (11%) were from other cardiovascular causes, and five (14%) were from noncardiac causes. The mean age at death or transplantation was 39 ± 16 years. Of the twenty-two patients who died suddenly, 15 were younger than 40 years old.

All five risk factors analyzed were univariate predictors for mortality. However, there was a significant interaction between family history of sudden death and syncope. In the absence of the other factor, each of these factors was associated with a relative risk of less than one. When they were combined however, the univariate risk for the combined factor of family history of sudden death and syncope was 8.2. Multivariate survival analysis was also performed. The multivariate sudden death risk ratios for the four risk factors were: 1.8 for an abnormal exercise blood pressure response, 5.3 for a family history of sudden death and syncope, 1.9 for nonsustained ventricular tachycardia, and 2.9 for left ventricular wall thickness greater than 30 mm. When family history of sudden death and syncope were considered as a single risk factor, 203 (55%) patients had no risk factors. One, two, and three risk factors were seen in 122 (33%), 36 (10%), and 7 (2%) patients, respectively. No patient had four risk factors. If a patient had no risk factor, the estimated six-year survival rate without sudden death was 95%. The survival estimates for patients with one, two, and three risk factors were 93%, 82%, and 36%. Thus, the 43 patients with two or three risk factors had a relative risk of 5.6 for sudden death compared to the 325 patients with zero or one risk factor.

Elliott et al conclude that analysis of five easily measurable variables can effectively predict sudden death in patients with hypertrophic cardiomyopathy. The presence of two or more risk factors was associated with a 4-5% estimated annual sudden death risk. Therefore, patients with multiple risk factors may have a sudden death probability high enough to justify prophylactic therapy. Recommendations about the most appropriate prophylactic therapy, amiodarone, or implantable defibrillators, cannot be made from this study.

COMMENT BY JOHN P. DiMARCO, MD, PhD

In this paper, Elliott et al describe easily identifiable clinical variables for sudden death in a large group of patients with hypertrophic cardiomyopathy. They identified a subgroup of 12% of the patients with two or more risk factors who had an annual sudden death risk of about 5%. Patients with none or only one risk factor had a much lower risk of sudden death.

Patients with hypertrophic cardiomyopathy are often difficult to manage. Even in the absence of significant hemodynamic symptoms, sudden death may occur. It is particularly tragic that sudden death is more common in young individuals who may have few other symptoms. Among prophylactic therapies, only amiodarone and defibrillator implantation are thought to be effective. Unfortunately, the value of invasive studies including electrophysiologic testing and hemodynamic measurements for predicting sudden death has been controversial and an easy method to identify a group that justifies these interventions has not been widely accepted. Elliot et al describe a fairly simple system to identify a group with a 5% annual sudden death risk and this value approaches the level at which the hazards and costs of possible prophylactic therapy would be acceptable.

A major limitation of this study is the fact that about half of all the patients seen with hypertrophic

cardiomyopathy at Elliott et al's institution were excluded for one reason or another. Many of these were excluded because of age or a history of amiodarone use. However, the data should apply to patients seen for initial diagnostic evaluation by clinical cardiologists. Data such as these could be used to initiate a comparative trial of interventions to prevent sudden death. ❖

CME Questions

11. In multivessel disease patients PCI vs. CABG results in:
a. less mortality at 30 days.
b. less MI at 30 days.
c. more repeat revascularization.
d. All of the above

12. New biphasic external defibrillators vs. monophasic results in:
a. increased first shock efficacy.
b. increased survival to hospital discharge.
c. improved cerebral function.
d. A and C

13. Color flow Doppler grading of mitral regurgitation:
a. accurately reflects regurgitant stroke volume.
b. determines the hemodynamic load.
c. separates mild from severe regurgitation.
d. All of the above

14. Fludrocortisone treatment of hypotension in chronic fatigue syndrome:
a. improves subjective well being.
b. normalizes the tilt table response.
c. improves exercise tolerance.
d. was ineffective.

15. Which of the following is *not* predictive (risk ratio < 2.0) of mortality in hypertrophic cardiomyopathy?
a. Family history of sudden death and syncope
b. Abnormal exercise BP response
c. LV wall thickness of more than 3 cm
d. All of the above

Readers are Invited. . .

Readers are invited to submit questions or comments on material seen in or relevant to *Clinical Cardiology Alert*. Send your questions to: Melissa Lafferty, *Clinical Cardiology Alert*, c/o American Health Consultants, P.O. Box 740059, Atlanta, GA 30374. For subscription information, you can reach the editors and customer service personnel for *Clinical Cardiology Alert* via the internet by sending e-mail to melissa.lafferty@ahcpub.com. ❖

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