Facts and principles learned at the 32nd annual Williamsburg Conference on Heart Disease

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The December 2004 conference on heart disease in Williamsburg, Virginia, was the 32nd such annual conference to be held in that city. The conference has been directed by one of the authors (WCR) since it began in 1972. It usually attracts about 200 enrollees, most of whom are repeat attendees. The conference provides 20 hours of continuing medical education credit, and nearly all of the speakers are nationally and internationally recognized. It is one of the longest-running cardiology courses sponsored by the American College of Cardiology. Its unique feature is that each presentation is 90 minutes, which allows most speakers time to discuss more than one topic and answer questions.

The proceedings of the December 2002 and the December 2003 conferences were summarized in previous issues of Baylor University Medical Center Proceedings, and this article summarizes the proceedings of the 2004 conference. Unfortunately, the American College of Cardiology has decided to discontinue its sponsorship of cardiology conferences beginning in 2005. Moreover, the Williamsburg Conference Center will undergo extensive renovation in 2005 and therefore will not be available for meetings until 2006. Thus, there will be no 2005 Williamsburg heart disease meeting, and the 2006 meeting, if held, will be sponsored by an organization other than the American College of Cardiology.

PERCUTANEOUS CORONARY INTERVENTION IN ACUTE MYOCARDIAL INFARCTION

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Percutaneous coronary intervention (PCI) vs thrombolysis in acute myocardial infarction (AMI). Most studies of patients with acute myocardial infarction have shown that the survival rate is greater in patients treated with PCI than those treated with thrombolytic therapy if the interval from symptom onset to arrival in the cardiac catheterization laboratory is short. A metaanalysis of 11 trials carried out between 1989 and 1996 (before use of coronary stents) in patients with AMI randomized within 12 hours following symptom onset showed that the mortality rates 1 and 6 months after treatment were 4.3% and 6.2%, respectively, for 1348 patients randomized to undergo PCI, and 6.9% and 8.2%, respectively, for 1377 patients assigned to receive thrombolytic therapy (absolute risk reductions, 2.6% and 2%; relative risk reductions, 38% and 23%) (1). Another study of 840 AMI patients randomized within 6 hours of symptom onset between June 1997 and September 2000 disclosed the 30-day occurrence of death, nonfatal AMI, or nonfatal disabling stroke to be 6.2% in the PCI group and 8.2% in the prehospital fibrinolysis group (2). The 30-day mortality rate was 4.8% in the PCI group and 3.8% in the prehospital fibrinolysis group. Thus, many but not all studies favor PCI over thrombolysis for AMI.

Prediction of delay from symptom onset to PCI in AMI. Angeja and colleagues (3) studied 40,017 consecutive patients with AMI from June 1994 to April 2000. The door-to-balloon time ranged from 84 to 152 minutes (median, 111). The proportion of patients with delays >2 hours (as compared with <2 hours) was greater among those aged ≥65 years (49% vs 41%), women (50% vs 42%), patients with a contraindication to fibrinolysis (60% vs 41%), those without chest pain on admission (61% vs 43%), those transferred from another hospital (87% vs 43%), and those who presented outside the hours of 8 AM to 4 PM. The shorter the interval was from onset of symptoms of AMI to either PCI or thrombolytic therapy, the greater the survival rate (4, 5). The European Society of Cardiology recommends PCI as the preferred reperfusion therapy for AMI if 1) the interval from symptom onset to reperfusion is <90 minutes, 2) the patient is in shock; 3) the patient has a contraindication to thrombolysis, or 4) thrombolysis has failed (6, 7).

Cardiogenic shock in AMI. Goldberg and associates (8) studied 9076 patients with cardiogenic shock (systolic blood pressure [BP] <80 mm Hg) secondary to AMI during the period from 1975 to 1997 and found that the incidence of shock remained relatively stable during the 23-year period, averaging 7%. The in-hospital mortality rate in those with shock was 72%, while the rate in those without shock was 12%. Cotter et al (9), in an evaluation of the effect of a nitric oxide synthase inhibitor (N^G-nitro-L-arginine methyl ester, or L-NAME) in 30 patients with refractory cardiogenic shock, found that 4 of the 15 patients (27%) who received L-NAME died within 30 days, while 10 of the 15 control patients (67%) died within 30 days. Thus, this report supports treatment of patients who have cardiogenic shock with a nitric oxide synthase inhibitor.
SUDDEN CARDIAC DEATH

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Frequency of sudden cardiac death (SCD) in the USA. The exact frequency of SCD in adults in the USA is variable and dependent on the definition of SCD, the location, and the methods used (prospective or retrospective) to obtain the data. Zheng et al (10), for example, examined mortality data from US vital statistics from the period 1989 to 1998, defining SCD as death occurring out of the hospital or in the emergency department or patients who are “dead on arrival” with previous known heart disease. Of nearly 720,000 cardiac deaths in the USA in 1998, 456,000 (63%) were classified as SCD. Of the latter, 62% were considered to be due to coronary artery disease (CAD). The rate of SCD increased with age, was higher in men than women, and was higher in blacks than whites.

Chugh and colleagues (11) prospectively evaluated SCD among all residents in a single county in Oregon during a 1-year period. Of the county’s 660,486 residents, 353 had SCD (53/100,000; median age, 69; 57% male), accounting for nearly 6% of the overall mortality. Resuscitation was attempted in 237 cases (67%) and was successful (defined as survival to hospital discharge) in 28 (8%). A retrospective review of death certificates yielded 1007 cases (153/100,000; median age, 81 years; 51% male). Thus, retrospective death-certificate–based surveillance resulted in significant overestimation of SCD incidence.

Bunch and associates (12) studied all residents of Rochester, Minnesota, who from 1985 to 2002 presented with a ventricular fibrillation (VF) out-of-hospital cardiac arrest identified and treated by emergency medical services personnel. The overall incidence of treated VF out-of-hospital cardiac arrest was 17/100,000: 26/100,000 in the period 1985 to 1989, decreasing to 8/100,000 in the period 2000 to 2002. During the same time period, placement of intracardiac defibrillators (ICD) increased: from 5/100,000 during the period from 1990 to 1994 to 21/100,000 during the period from 2000 to 2002. Thus, the incidence of VF out-of-hospital cardiac arrest appears to be declining.

Gorgels and colleagues (13) studied 492 victims of SCD during the period from January 1997 to December 2000 in the Maastricht area of the Netherlands. SCD represented 19% of all deaths in that time period. The yearly incidence of SCD was 92/100,000 inhabitants. In 52% of the men and 59% of the women, SCD was the first manifestation of heart disease. Of the 492 SCD victims, 77% were known to have CAD and 72% to have had an AMI that had healed. Most of the SCD victims had never had evidence of heart failure (HF), although the frequency of SCD was increased among patients with HF.

Since the rate of overall mortality from coronary heart disease in the USA has been decreasing, it follows that the incidence of out-of-hospital cardiac arrest must also be decreasing. A study from Finland described a marked decrease in frequency of out-of-hospital cardiac arrest during a 5-year period (1994–1999), and another study in Sweden showed a decrease during a 17-year period. Cobb and colleagues (14) compared out-of-hospital cardiac arrest cases during a 21-year period (1979–2000) in Seattle, Washington. The annual incidence of cardiac arrest with VF as the first identified rhythm decreased by about 56% during this period, and the decrease was most evident in men. When all treated arrests with a presumed cardiac etiology (including asystole) were considered, the incidence decreased by 43% in men but negligibly in women. Thus, in Seattle and we hope in other parts of the country, there has been a major decline in the incidence of out-of-hospital VF and in all cases of cardiac arrest presumably due to heart disease.

Risk factors for SCD. Zipes and Wellens (15) suggested, from findings of another study that contrasted with their results already mentioned, that 80% of victims of SCD have underlying CAD and therefore the risk factors for SCD are essentially those of atherosclerosis. Thus, the incidence of SCD increases with age in both men and women and in whites and nonwhites. Among patients with CAD, however, the proportion of CAD deaths that are sudden decreases with age. Of SCD victims in the USA, ~7% are men and ~25% are women.

In addition to CAD, other risk factors for SCD include prior cardiac arrest with successful resuscitation, prior ventricular arrhythmia, depressed left ventricular ejection fraction, previous coronary event (AMI, angina pectoris), elevated serum lipid levels, premature ventricular complexes, atrial fibrillation (AF), cigarette smoking, obesity, diabetes mellitus, inactivity, elevated C-reactive protein (CRP) level, season of the year (rate higher in winter), time of day (AM > PM), week of the month (rate high in first week), various other electrocardiographic abnormalities (e.g., prolonged QT interval, widened QRS complex, heart-rate variability, increased QT dispersion, T-wave alternans), drugs (any antiarrhythmic agent and some nonantiarrhythmic agents), increased resting heart rate, systemic hypertension, emotional stress, depression, left ventricular hypertrophy, and any underlying heart disease (e.g., cardiomyopathy, valvular disease, congenital disease, primary electrical abnormality) (15).

Drugs vs ICD in decreasing mortality rate. At least 7 multicenter trials (MADIT, MUSTT, MADIT II, AVID, CASH, CIDSD, and DINAMIT) have demonstrated that ICDs are more effective than any antiarrhythmic drug in decreasing the mortality rate (15, 16). The SCD-HeFT trial, which included patients with either ischemic or idiopathic dilated cardiomyopathy and left ventricular ejection fractions ≤35%, demonstrated that those patients receiving an ICD had a 23% lower all-cause mortality rate than either the placebo group or a group receiving amiodarone (17). A recent ruling now allows reimbursement for implantation of an ICD in patients with ischemic cardiomyopathy, healed myocardial infarction, or a left ventricular ejection fraction ≤30%.

Automatic electrical defibrillators in public places and in private homes. About 80% of cardiac arrests occur in the home. Survival after cardiac arrest decreases by 10% per minute after arrest. Automatic electrical defibrillators are already in airports, commercial air carriers, many public places (15, 18), and, in a few neighborhoods, lock boxes.

The biologic pacemaker. The biologic pacemaker involves use of viral gene transfer to convert quiescent heart-muscle cells into pacemaker cells and successful generation of spontaneous, rhythmic electrical activity in the ventricle in vivo. Early research suggests that genetically engineered pacemakers could be developed as a possible alternative to implantable electronic devices (19).
PREOPERATIVE RISK STRATIFICATION OF PATIENTS UNDERGOING NONCARDIAC SURGERY
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Cardiac risk index. In 1977, Goldman and colleagues (20) published a multifactorial index of cardiac risk in patients undergoing noncardiac surgical procedures. In 1999, Lee and associates (21), including Goldman, revised their previous cardiac risk index to include 6 independent correlates of major cardiac complications: 1) high-risk type of noncardiac surgery; 2) evidence of coronary heart disease; 3) evidence of HF; 4) history of cerebrovascular disease; 5) insulin therapy for diabetes mellitus; and 6) serum creatinine >2.0 mg/dL. Rates of major cardiac events in patients with 0, 1, 2, or ≥3 of these factors were approximately 0.5%, 1%, 7%, and 11%, respectively.

Preoperative results of dobutamine stress echocardiography and single-photon emission computed tomography imaging do not add to the predictive value of the index. Importantly, coronary angiography just before the planned elective noncardiac operation actually added to the operative risk rather than subtracted from it. Patients who had undergone coronary artery bypass grafting (CABG) in the past appeared to have lower 30-day rates of postoperative noncardiac mortality (1.7%) and AMI (0.8%) than individuals with similar coronary narrowing who had not undergone CABG (33% and 2.7%).

Usefulness of prophylactic beta-blocker therapy before elective noncardiac surgery. In a study by Boersma et al (22), a beta-blocker (bisoprolol) taken at least 7 days preoperatively decreased 30-day rates of cardiac death and nonfatal myocardial infarction in patients with <3 cardiac risk correlates from 2.3% to 0.8%, and in patients with ≥3 cardiac risk correlates, from 13.0% to 6.2%.

Usefulness of statins before elective noncardiac surgery. At least 3 studies have shown statins to be effective in reducing rates of death and nonfatal myocardial infarction after elective noncardiac surgery (23–25). Poldermans and colleagues (23) found the risk of 30-day mortality in patients undergoing major noncardiac vascular operations was 4.5 times lower among statin users than among statin nonusers, despite a much lower use of both beta-blockers and aspirin in the statin group. Durazzo et al (24) found in 100 patients randomized to receive either atorvastatin 20 mg daily or placebo for 30 days before noncardiac vascular surgery that the statin patients had more than two thirds fewer cardiac events than the placebo group (8% vs 26%) in the 6-month postoperative period. Lindenauer and associates (25) studied 780,591 adults who underwent major noncardiac surgery and found that the 77,080 who had received preoperative lipid-lowering therapy had a lower mortality rate (2.1% vs 3.1%) than those who did not receive preoperative lipid-lowering therapy.

INDICATIONS FOR CARDIAC VALVE REPLACEMENT OR REPAIR
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Dr. Bonow was chairman of the American College of Cardiology/American Heart Association guidelines committee for the management of patients with valvular heart disease. The first report of this committee appeared in 1998 and consumed 103 published pages (26). The second report is due to be published in the spring of 2005. Unlike coronary heart disease and HF, for which results of many trials allow specific guidelines for management, valvular heart disease has yielded no prospective randomized trials with definitive results and, therefore, the guidelines are based only upon consensus of experts (27). Unfortunately, opinions among the “experts” in management of these patients differ widely. As a consequence, the threshold for proceeding with a valve operation varies tremendously from place to place depending upon the results expected from the surgeons and the skill of the referring cardiologists.

Aortic stenosis. Aortic stenosis is the most common valvular condition in patients referred for a cardiac valve operation in the western world, even though mitral regurgitation (either from mitral valve prolapse or from papillary muscle dysfunction secondary to myocardial ischemia) is a more common condition. Most patients with mitral regurgitation never require a mitral valve operation, whereas most patients with aortic stenosis do require an aortic valve operation if they live long enough. Although Bonow and colleagues (26) state that “the absolute valve area (or transvalvular pressure gradient) is not usually the primary determinant of the need for aortic valve replacement,” we believe that it is the severity of the stenosis, not its presence, that determines whether symptoms result and therefore whether valve replacement is indicated. For example, a patient who has a peak systolic transvalvular gradient of 10 mm Hg has aortic stenosis, but the degree of obstruction is minimal and therefore symptoms cannot logically be attributed to the stenosis. A recent study by Roberts and Ko (28) demonstrated that the transvalvular peak systolic gradient correlated much better with the weight of the operatively excised aortic valve than did the aortic valve area, which changed insignificantly as the weight of the valve increased. (The valve’s weight is determined mainly by the quantity of calcium in the valve, and it is the quantity of calcium that mainly determines the degree of stenosis across the valve.)

Aortic valve replacement for aortic stenosis is usually not performed until the patient develops symptoms (angina pectoris, syncope, or HF), irrespective of the degree of obstruction across the valve. Some centers, however, perform aortic valve replacement if the degree of obstruction is severe despite an asymptomatic state. The definition of “severe,” moreover, appears to vary among medical centers.

A major complicating factor in evaluating adults aged ≥20 years who have aortic stenosis is that about 50% of them also have severe narrowing of one or more major epicardial coronary arteries and, of course, CAD also can produce angina and dyspnea, the most common manifestations of HF. The decision for aortic valve replacement at the time of CABG can be difficult. The degree of obstruction across the aortic valve to warrant its replacement is usually considerably less than in the patient with aortic stenosis and insignificant coronary narrowing. Generally, a peak systolic gradient >25 mm Hg in a patient undergoing CABG probably is sufficient to warrant aortic valve replacement at the same operation. If the stenotic aortic valve is not replaced at the time of CABG, reoperation later to replace the stenotic valve is, of course, complicated by pericardial adhesions and the presence of the bypass conduits attached to the ascending aorta.
Aortic regurgitation. Aortic regurgitation is more complex than aortic stenosis in that it can result from disease of the aortic valve (bicuspid structure, infective endocarditis, rheumatic disease), from disease of the aorta (Marfan syndrome or forme fruste varieties, syphilis, aortic dissection), or from both (ankylosing spondylitis). The latter conditions may require replacement of a portion of ascending aorta in addition to the aortic valve, a far more complex procedure than aortic valve replacement alone. Furthermore, in contrast to aortic stenosis, aortic regurgitation may be acute (active infective endocarditis, aortic dissection) or chronic, whereas aortic stenosis is always a chronic state in that calcific deposits develop over a long period of time and the degree of aortic stenosis is roughly proportional to the quantity of calcium on the valve.

Although most patients with pure aortic regurgitation (no element of valve stenosis) are not referred for operation until symptoms develop, the appearance of left ventricular dilation, particularly when progressive, and diminished or diminishing left ventricular ejection fraction in the absence of symptoms generally is indicative of need for operation. These criteria include left ventricular peak systolic dimension >55 mm and/or diastolic dimension >75 mm and/or a left ventricular ejection fraction <50%.

Mitrais stenosis. The prevalence of mitral valve stenosis has decreased enormously in the western world but continues to plague residents of the developing world. Mitral valvotomy (either via the percutaneous or operative route) is generally indicated when symptoms (mainly dyspnea) reach New York Heart Association functional class II status (i.e., patient is comfortable at rest but ordinary physical activity results in fatigue, dyspnea, palpitations, and/or angina pectoris) (26). Other criteria include a mitral valve orifice area of ≤1.0 cm²/m² body surface area or <1.7 cm² in normal-sized adults. Valvulotomy is generally indicated when the peak systolic pulmonary arterial pressure is >60 mm Hg or the pulmonary artery mean pressure is >25 mm Hg during exercise. Valvulotomy can be performed only when the stenotic mitral valve leaflets are devoid or nearly devoid of calcific deposits and mitral regurgitation is absent or minimal. Otherwise, mitral valve replacement is required. A mitral valve procedure is usually indicated in patients with a previous symptomatic embolus, even when they are asymptomatic.

Mitrais regurgitation. In contrast to mitral stenosis, which in adults is virtually always secondary to a single etiology (i.e., rheumatic heart disease), mitral regurgitation has several causes. Its management, therefore, being dependent to a large extent on its cause, is more complex than that of mitral stenosis. Pure mitral regurgitation (no element of stenosis) is most commonly due to mitral valve prolapse, probably a congenital condition resulting from weakened collagen, or to papillary muscle dysfunction after healing of a myocardial infarct that resulted from severe CAD.

Because operative interventions for mitral regurgitation secondary to mitral valve prolapse and for that due to myocardial ischemia or infarction secondary to CAD are quite different, the presenter focused on operative indications for chronic mitral regurgitation secondary to mitral valve prolapse. Surgical treatment should be considered for patients with functional disability and/or for patients with no symptoms or only mild symptoms but with progressively increasing left ventricular dimensions as determined by noninvasive studies. Generally, a mitral valve operation, preferably a reconstructive procedure, is indicated in patients with symptoms, a left ventricular peak systolic dimension >45 mm or a serial increase in this dimension, a left ventricular ejection fraction <60% or a serial decrease in the ejection fraction, significant pulmonary hypertension, possibly the presence of AF, or evidence of right ventricular dysfunction. In asymptomatic patients with severe mitral regurgitation, a lower threshold for operation is probably warranted with progressive left atrial enlargement (>45 mm) in which successful mitral valve repair appears highly likely.

USEFULNESS OF BETA-BLOCKERS IN CORONARY ARTERY DISEASE

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Although their benefit in patients who have experienced AMI is well established (82 studies, 54,234 patients), beta-blockers are still underutilized in these patients. Thirty-four beta-blockers are available in the USA, but both calcium antagonists and nitrates are prescribed more often for patients with CAD than beta-blockers. Wang and Stafford (29) found that, even after eliminating patients with atrioventricular conduction defects or bronchospasm (from asthma or chronic obstructive lung disease), ambulatory patients with healed myocardial infarcts were receiving a beta-blocker in only 37% of 5.5 million office visits, and patients with HF were receiving a beta-blocker in only 6.5% of 4.3 million office visits. In a study sponsored by the Health Care Financing Administration of medical records of 201,752 patients with healed myocardial infarcts, Gottlieb and colleagues (30) found that only 34% of the patients received a beta-blocker. In the myocardial infarct patients with no comorbid conditions, treatment with a beta-blocker was associated with a 40% reduction in mortality during the 2 years following the infarct. Even among the patients with left ventricular ejection fraction <20%, HF, chronic obstructive lung disease, serum creatine levels >1.4 mg/dL, or diabetes mellitus, those treated with a beta-blocker had significantly lower mortality rates in each of these subgroups than those who did not receive a beta-blocker.

Beta-blockers are at least as good as calcium antagonists in relieving episodes of myocardial ischemia. Heidenreich and associates (31) did a metaanalysis of 90 studies comparing the relative efficacy and tolerability of beta-blockers and calcium antagonists in patients with stable angina pectoris. They found that the rates of cardiac death and myocardial infarction were similar for those receiving either a beta-blocker or a calcium antagonist, but those who received a beta-blocker had fewer episodes of angina pectoris per week, and beta-blockers were discontinued because of adverse events less often than calcium antagonists.

The effectiveness of beta-blockers in patients with chronic HF is well appreciated (7 favorable trials). Pool-Wilson and colleagues (32) compared carvedilol to metoprolol in just over 3000 patients with left ventricular ejection fraction ≤35% and found in the nearly 5-year follow-up period that the all-cause mortality rate was significantly lower in those randomized to receive carvedilol than in those who received metoprolol (34% vs 40%). Sliwa and
Several studies have shown that CABG provides better long-term outcome than PCI in patients with severe narrowing of all 3 major coronary arteries, in those in whom the left main coronary artery is severely narrowed, and in those with subnormal left ventricular function. The BARI trial (37) compared patients with multivessel CAD randomly assigned to an initial treatment strategy of CABG or PCI, monitoring their condition for an average of 5.4 years. The respective in-hospital event rates for CABG and PCI were 1.3% and 1.1% for mortality, 4.6% and 2.1% for Q-wave AMI, and 0.8% and 0.2% for stroke. The 5-year survival rates were 89.3% for those assigned to CABG and 86.3% for those assigned to PCI. The respective 5-year survival rates free from Q-wave AMI were 80.4% and 78.7%. By 5 years after entering the study, 8% of the patients assigned to CABG and 54% of those initially assigned to PCI had undergone additional revascularization procedures, and 31% of those initially assigned to PCI subsequently underwent CABG. The diabetic patients being treated with insulin or oral hypoglycemic agents at baseline had a 5-year survival rate of 80.6% in the CABG group and 65.5% in the PCI group. Thus, an initial strategy of percutaneous transluminal coronary angioplasty did not significantly compromise 5-year survival in patients with multivessel CAD, although subsequent revascularization was required more often with this strategy. For patients with diabetes, the 5-year survival rate was significantly better after CABG than after PCI.

Drug-eluting stents have performed much better than bare metallic stents in patients with CAD. Moses and colleagues (39) compared the sirolimus-eluting stent with a standard stent in 1058 patients. The rate of failure of the target coronary artery was reduced from 21% with a standard stent to 9% with a sirolimus-eluting stent—a reduction that was driven largely by a decrease in the frequency of need for revascularization of the target coronary narrowing (17% in the standard stent group vs 4% in the sirolimus-eluting stent group). Thus, the sirolimus-eluting stent yielded lower rates of restenosis and associated clinical events than the standard stent. Orlic and colleagues (40) also found the sirolimus-eluting stent to be very useful in patients with multivessel CAD. Lemos and colleagues (41) found that the sirolimus-eluting stents were much more effective in patients with AMI than conventional bare stents. The sirolimus-eluting stents were not associated with an increased risk of stent thrombosis (unlike conventional bare stents) and were effective in reducing the incidence of adverse events after 300 days in patients with ST-segment elevation AMI.

Statin drugs, of course, have been found to be enormously effective in preventing first atherosclerotic events and in preventing repeat events in patients who have had a first atherosclerotic event. Serruys and colleagues (42) studied 16,077 patients who had stable or unstable angina pectoris or silent myocardial ischemia following successful completion of their first PCI and total serum cholesterol levels between 135 and 270 mg/dL. The patients were randomly assigned to receive fluvastatin 80 mg/day...
or matching placebo at hospital discharge for 3 to 4 years. The median time between PCI and first dose of fluvastatin was 2.0 days, and the median follow-up interval was 3.9 years. Survival time free from major adverse cardiac events was significantly longer in the fluvastatin group (n = 844) than in the placebo group (n = 833). A total of 181 (21%) in the fluvastatin group and 222 (27%) in the placebo group had at least one major adverse cardiac event. The authors concluded that fluvastatin treatment in patients undergoing their first successful PCI significantly reduced the frequency of major adverse cardiac events in the next 3 to 4 years (42).

A previous study showed that CABG was more effective than PCI in diabetic patients with multivessel CAD. Abizaid and colleagues (43) on behalf of the ARTS investigators randomly assigned patients to undergo stent implantation (n = 600 patients, 112 of whom were diabetic) or CABG (n = 605 patients, 96 of whom were diabetic). After 1 year, diabetic patients treated with stenting had the lowest event-free survival rate (63%), because of a higher incidence of repeat revascularization) compared with either diabetic patients treated with CABG (84%) or non-diabetic patients treated with stents (76%). Diabetic and nondiabetic patients treated with CABG had similar 1-year event-free survival rates (84% and 88%). The authors concluded that diabetic patients with multivessel CAD treated with stenting had a worse 1-year outcome than patients who underwent CABG or nondiabetics treated with stenting. The strategy of stenting was less costly than CABG, however, regardless of diabetic status (43).

It is well known that patients with left ventricular dysfunction as a consequence of CAD are at high risk of cardiac arrest. No benefit from prophylactic ICD therapy was observed, however, in a group of patients with left ventricular dysfunction who underwent CABG (the CABG PATCH trial). Veenhuyzen and colleagues (44) studied modes of death in 5410 patients with ischemic left ventricular dysfunction enrolled in the Studies Of Left Ventricular Dysfunction (SOLVD) trial. The outcomes of patients undergoing earlier CABG and those not undergoing earlier CABG were compared by baseline left ventricular ejection fractions. Prior CABG was associated with a 25% reduction in death rate and 46% reduction in sudden death rate independent of ejection fraction and severity of HF symptoms. When these results were applied to a group of patients with left ventricular dysfunction who had not undergone prior CABG, the annual rates of death (8.2%) and sudden death (2.4%) were similar to those in the CABG group (7.9% and 2.3%). The authors concluded that, in patients with ischemic left ventricular dysfunction, prior CABG is associated with a significant independent reduction in mortality. These results appear to account for the lack of benefit from ICD therapy in the CABG patients.

Several studies have shown that levels of specific systemic markers of inflammation (CRP, interleukin 6, tumor necrosis factor) usually are increased in patients with acute myocardial ischemic syndromes and after coronary revascularization procedures. Lincoff and colleagues (45) collected serum samples at baseline (before revascularization) and 24 to 48 hours and 4 weeks after administration of abciximab or placebo to 160 patients. Between baseline and 24 to 48 hours, CRP level increased 32% less, interleukin 6 level increased 76% less, and tumor necrosis factor level increased 100% less in the abciximab therapy group than in the placebo group. By 4 weeks, most marker levels had returned to baseline, with no significant differences between placebo and abciximab groups. Thus, the systemic markers of inflammation increase in the first 24 to 48 hours after PCI, and the magnitude of that rise is diminished by periprocedural abciximab. Some of the long-term clinical benefit derived from this agent may be related to its antiinflammatory effect.

Statin drugs have many benefits, but a new study has shown that administration of a statin before PCI reduces the frequency of adverse events following PCI. Pasceri and colleagues (46) studied 153 patients with chronic stable angina pectoris who had not received previous statin therapy. Patients scheduled for elective PCI were randomized to receive atorvastatin (40 mg/day) or placebo starting 7 days before the procedure. Detection of markers of myocardial injury above the upper limit of normal was significantly less frequent in the statin group than in the placebo group: 12% vs 35% for creatine kinase-MB, 20% vs 48% for troponin I, and 22% vs 51% for myoglobin. After PCI, AMI as defined by creatine kinase-MB level was detected in 5% of patients in the statin group and in 18% in those in the placebo group. Post-procedural peak levels of creatine kinase-MB, troponin I, and myoglobin were also significantly lower in the statin group (2.9 ng/mL, 0.09 mg/mL, and 58 mg/mL) than in the placebo group (7.5 ng/mL, 0.47 mg/mL, and 81 mg/mL). Thus, pretreatment with atorvastatin 40 mg/day for 7 days significantly reduced the frequency of procedural myocardial injury in elective PCI.

**INDICATIONS FOR CORONARY ARTERY BYPASS GRAFTING AND CARDIAC VALVE REPLACEMENT**

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Dr. Charles Roberts briefly described cardiothoracic and vascular procedures done at the Winchester Medical Center. At that center, 569 cardiac, 410 noncardiac vascular, and 155 general thoracic operations were done in 2003. Of the cardiac procedures, 385 (80%) were CABG, 78 (16%) were valve operations, and 19 (4%) were other types of cardiac operations. The 3 most common, of course, were CABG, aortic valve replacement, and mitral valve repair. Most of the valve operations were performed by Dr. Roberts. The in-hospital mortality rate overall for the 569 cardiac operations was 2.5%. For isolated CABG, the hospital mortality rate was 2.1%. Of the 385 CABG procedures, 340 were on-pump and 45 were off-pump. Of the 78 valve operations, 38% were combined aortic valve replacement and CABG, 26% were isolated aortic valve replacement, 22% were isolated mitral valve repair or replacement, 12% were mitral valve repair or replacement plus CABG, and 2% were combined mitral and aortic valve replacement. The operative mortality rate for the valve operations was 1.3%. Of the 410 noncardiac vascular operations, 162 (40%) were carotid endarterectomy, 76 (19%) were aortic procedures, 76 (19%) were bypass procedures in the legs, 68 (16%) were amputations, and 28 (6%) were miscellaneous procedures. Of the 64 patients who underwent operation for abdominal aortic aneurysm, none died in the hospital.

In his presentation, Dr. Roberts asked 10 questions, as follows:

1. What is the role of the left ventricular ejection fraction in determining the appropriateness of CABG versus percutaneous coronary intervention (PCI)?
2. What are the indications for CABG in diabetic patients?
3. How does CABG compare with PCI in terms of procedural myocardial injury?
4. What is the role of statins in reducing procedural myocardial injury?
5. What are the indications for cardiac valve replacement?
6. How do CABG and valve replacement compare in terms of mortality?
7. What are the indications for peripheral arterial bypass grafting?
8. How do surgical and endovascular procedures differ in terms of complications?
9. What are the long-term outcomes of CABG and PCI in diabetic patients?
10. What role does inflammation play in determining the success of PCI?
1) Which patients should undergo CABG? In 2004, the American College of Cardiology/American Heart Association provided a guideline update to answer this question (47). Dr. Roberts compared results of CABG to those of drug-eluting stents and discussed CABG as therapy for patients with HF or AMI. He emphasized that results improved considerably if CABG was delayed until at least 48 hours after onset of AMI. He also presented results of CABG in patients with the metabolic syndrome.

2) On-pump CABG vs off-pump CABG: Do outcomes differ? Dr. Roberts discussed the prevalence of each technique and relevant randomized trials. He also discussed the effect of coronary pulmonary bypass on the inflammatory response and means of reducing that response. He prefers the on-pump procedure.

3) Can neurologic outcomes be improved in CABG patients? The usefulness of apomorphine, epiaortic ultrasound, intravascular filtration, and ligation of the left atrial appendage were all discussed.

4) Should saphenous venous grafts ever be used, or should only arterial conduits be used? He discussed the use of 2 internal thoracic arteries rather than just one and compared use of the radial artery and the right internal thoracic artery and use of the radial artery and saphenous vein as grafts.

5) What criteria for aortic valve replacement in aortic stenosis should be used in the patient undergoing CABG? For the patient ≥70 years of age, the peak transvalvular systolic pressure gradient should be >30 mm Hg. For the patient <70 years of age, it appears to be useful to add 2 mm Hg for every year after that. He also addressed which substitute valve to use and discussed the patient with a small aortic dimension.

6) When is operation indicated for ascending aortic aneurysm? Dr. Roberts distinguished between the patient with a 3-cuspid aortic valve and the patient with a 2-cuspid aortic valve. The evidence is minimal in both circumstances, but in the patient with the bicuspid aortic valve, Marfan syndrome, or the forme fruste varieties of that syndrome, the aortic diameter should be >4.5 cm, and in the patient with a tricuspid aortic valve, the aortic diameter should probably be >5.5 cm. He compared the benefits of aortic reduction or aortoplasty vs replacement and suggested, as have others, that aortic reduction might be a particularly good procedure in the patient with the bicuspid aortic valve (48, 49).

7) What criteria should be used for mitral valve repair or replacement for mitral regurgitation in patients undergoing CABG? The cause of mitral regurgitation—whether secondary to myocardial ischemia or infarction or to mitral valve prolapse—has considerable effect on what should be done. For ischemic mitral regurgitation, mitral valve anuloplasty may be enough (“ring and run”). For mitral valve prolapse, partial resection of a portion of the posterior leaflet with anuloplasty appears to be preferred. The edge-to-edge repair is coming into focus since it is being done in some places percutaneously. Little postoperative evaluation data are available on these patients. The approach for mitral regurgitation associated with end-stage ischemic cardiomyopathy also was discussed (50).

8) Who should have ablation for AF? The intraoperative radiofrequency Maze ablation procedure and the standard modified Cox-Maze procedure were discussed (51–53). When developing a treatment strategy for AF in patients undergoing CABG or a valve procedure, surgeons should consider whether AF is continuous or intermittent preoperatively. It appears that the Cox-Maze procedure does not improve survival rate.

9) Which procedure should be done first: CABG, abdominal aortic aneurysm repair, or carotid endarterectomy? If the abdominal aortic aneurysm is >5.5 cm in diameter and can be repaired by endovascular technique, it should probably be addressed first. In the stable cardiac patient, carotid endarterectomy should probably be done first if the diameter reduction is >70% in the symptomatic and asymptomatic patient.

10) Do higher volumes mean better outcomes? He discussed the relation between surgeon volume and outcome, the relation between institutional volume and outcome, and the issue of public disclosure of operative results.

THE CHANGING WORLD OF CARDIAC SURGERY
Charles S. Roberts

Oversupply of cardiothoracic surgeons. The frequency of CABG is diminishing, but the number of cardiovascular surgeons being trained is not diminishing (54). Thus, new trainees are not getting the workload that previous trainees received and, after completion of their training, their workload is not as great as in the past. As a consequence, technical expertise in cardiothoracic surgery may not be as good in the future as it is presently. A survey of cardiothoracic surgeons showed that among cardiac surgeons who perform cardiac procedures on adults and also perform general thoracic surgery, the median number of cardiac cases in 1999 was 174. As this number drops, patients face the probability of a higher operative risk because of the surgeons’ lower annual volumes.

While CABG numbers have declined, outcome expectations for all cardiothoracic operations have risen. Cardiothoracic outcomes, particularly CABG outcomes, are now feverishly analyzed, perhaps more so than any results in the history of surgery. In this milieu, 1 year with a high complication numerator and a low volume denominator may be ruinous for a young surgeon or even a seasoned one. To train many residents for a specialty that has few openings in practice is unfair to trainees. The January 2004 issue of the Annals of Thoracic Surgery listed only 2 “situations available,” whereas the January 1994 issue listed 21. Nevertheless, about 140 cardiothoracic residency positions remain available each year in the USA. The answer is to decrease the number of physicians training in cardiothoracic surgery in the USA. Dr. Roberts concluded that “in the face of an unrelenting decline in CABG numbers, prompt correction in the oversupply of cardiothoracic surgeons is in the best interest of patients.”

HYPERTRYPHIC CARDIOMYOPATHY
Barry J. Maron, MD, Director of the Cardiovascular Research Division, Heart Institute Foundation, Minneapolis, Minnesota

Hypertrophic cardiomyopathy (HC) was first described by Dr. Donald Teare, the medical examiner in London, United Kingdom, in 1957, when he described 10 patients who died suddenly in that city. He showed in that first article that the ventricular septum was thicker than the left ventricular free wall and that the myocardial fibers in the ventricular septum had a disorganized pattern. The next major development in this condition took place at the National Institutes of Health, when Drs. Eugene Braunwald and Andrew G. Morrow described clinical, hemodynamic, and
operative findings in a large group of patients with HC. Since about 1980, Dr. Maron has been the leading HC investigator. He has published several hundred articles on this condition.

In a review of this topic in 2002 (55), Maron found 968 articles on HC in the English language published between 1966 and 2000. Approximately 1 in 500 persons, or 2000/1,000,000 population, has HC. Thus, there are nearly 600,000 persons with this condition in the USA, making it the most common genetic cardiovascular disease. It is inherited as a mendelian autosomal dominant trait and is caused by mutations in at least 10 genes comprising over 400 sarcomeric protein mutations and some nonsarcomeric mutations. Three of the HC-causing mutant genes predominate, namely, those for a beta-myosin heavy chain, cardiac troponin, and myosin-binding protein C. The other genes, which account for a minority of HC cases, include those for cardiac troponin I, regulatory and essential myosin light chains, tipin, alpha-tropomyosin, gamma-actin, and gamma-myosin heavy chain. Although DNA analysis for mutant genes is the definitive method for establishing the diagnosis of HC, genotyping is not yet a routine clinical strategy because it is complex, time-consuming, and expensive and therefore should be confined to research-oriented investigations.

The clinical diagnosis of HC is established most easily and reliably by 2-dimensional echocardiography, although magnetic resonance imaging may be the best method in the future. HC is characterized by a hypertrophied left ventricular wall but a nondilated left ventricular chamber in the absence of another cardiac or systemic disease (e.g., systemic hypertension or aortic stenosis) capable of producing a similar magnitude of hypertrophy. The condition may be initially suspected because of a precordial murmur, positive family history, new symptoms, or an abnormal electrocardiographic pattern. Because about 75% of HC patients do not have left ventricular outflow obstruction, most do not have a precordial murmur. The left ventricular wall thickness ranges from 13 to 60 mm, the greatest thickness of any condition. The 12-lead electrocardiographic pattern is abnormal in nearly 95% of HC patients. A wide variety of abnormal patterns are observed. Electrocardiographic voltages are only modestly associated with the magnitude of left ventricular hypertrophy (56). Nevertheless, electrocardiograms raise suspicion of HC in family members without left ventricular hypertrophy on echocardiogram and are used in targeting athletes for echocardiography as part of pre-participation screening.

HC is characterized morphologically by left ventricular hypertrophy; in approximately 80% of patients, the ventricular septum is thicker than the left ventricular free wall (57). Most commonly, the thickest portion of ventricular septum is midway between the bases of the aortic valve cusps and the apex of the left ventricle. In patients with a left ventricular outflow pressure gradient, the ventricular aspect of the anterior mitral leaflet is thickened and the mural endocardium in the ventricular septum in direct apposition to the anterior mitral leaflet is also thickened by fibrous tissue. This anatomic equivalent of anterior septal motion of the anterior mitral leaflet is diagnostic of HC. The right ventricular cavity is not dilated, and its wall is thickened. The most common anatomic finding in patients with HC is dilatation of both atria (57). One of the authors (WCR) has examined over 300 hearts with HC at necropsy, and all had dilated atria.

On histologic examination, transverse sections of the ventricular septum commonly show disorganization of myocardial fibers and, in about 50% of cases, abnormal intramyocardial coronary arteries, mainly those in the ventricular septum. The abnormal intramyocardial coronary arteries are primarily in patients with focal scars in the ventricular septum. When the ventricular septum is quite hypertrophied, focal scars are commonly observed in the absence of any narrowing of the epicardial coronary arteries. Scarring in the left ventricular free wall and right ventricular free wall is also common in these individuals in the absence of narrowing of the epicardial coronary arteries; scarring in the left ventricular free wall is primarily in the subepicardial portion rather than in the subendocardial portion, as is characteristic of ischemia. The myocardial cells in the ventricular wall and the ventricular septum are usually quite hypertrophied and vary considerably in shape. The abnormal intramyocardial coronary arteries appear to be larger than normal, more numerous that normal, and have lumens that are much smaller than normal. HC is associated with larger myocardial masses than any other condition; the myocardial mass may “outgrow” its blood supply, and myocardial necrosis and fibrosis can be a consequence. Heart weight occasionally exceeds 1000 g (58). Some patients with HC develop considerable ventricular septal and left ventricular wall scarring, with resulting dilatation of the ventricular cavities, a situation simulating idiopathic dilated cardiomyopathy. When this situation occurs, cardiac transplantation may be necessary (59).

The clinical course of HC is enormously variable and unique among cardiovascular diseases by virtue of its potential for clinical presentation during any phase of life (from infancy to >90 years of age). Misperceptions about HC have prevailed because of its relatively low prevalence in cardiac populations, its extreme heterogeneity, and skewed patterns of patient referral that have created important selection biases. Indeed, much of the data assembled during the past 45 years has been disproportionately generated by a few tertiary centers and has largely represented patients preferentially referred because of their high-risk status or severe symptoms requiring specialized care such as operative therapy. As a consequence, the earlier publications on HC were dominated by descriptions of the most adverse consequences of the condition, namely SCD and severe HF, while patients whose disease was clinically stable or asymptomatic and elderly patients were underrepresented. Thus, the risks of HC were probably overestimated early on by dependence on frequently cited, ominous mortality rates of about 5% annually. Recent publications from less-selected regional or community-based HC patient cohorts cite annual mortality rates of about 1%, not dissimilar to that for the general adult US population. Thus, the view has switched from HC's being a bad disease to its being a disease that can be bad. Patients >75 years of age probably constitute as much as 25% of the total HC population, but only a minority have severe manifestations of HF. Left ventricular outflow obstruction is present in about 40% of HC patients of advanced age, suggesting that subaortic gradients may be well tolerated for long periods without adverse consequences.

HC patients with poor prognosis fall primarily into 3 categories: 1) those with a high risk of sudden death, 2) those in whom symptoms progress rather rapidly, some to the end stage wherein dilation of the cardiac ventricles results in left ventricular systolic...
Sudden death. Sudden death is the most common mode of death in patients with HC and the most devastating and unpredictable complication of the condition. Sudden death is the initial and only symptom of HC in a few patients. Although sudden death occurs most commonly in children and young adults, it may occur in midlife and beyond. It occurs most commonly during vigorous physical exercise. Indeed, HC is the most common cause of cardiovascular sudden death in young people, including trained competitive athletes. Those with increased risk of sudden death appear to constitute about 15% of the total HC population. Markers of the highest risk for sudden death in HC are the following: 1) prior cardiac arrest or spontaneous sustained ventricular tachycardia; 2) family history of premature HC-related death, particularly if sudden and in close relatives; 3) syncope and near syncope, particularly exertional or recurrent; 4) multiple and repetitive or prolonged bursts of nonsustained ventricular tachycardia on ambulatory electrocardiographic monitoring; 5) hypotensive BP response to exercise; and 6) extreme left ventricular hypotrophy with maximal wall thickness >30 mm, particularly in adolescents and young adults. Surprisingly, there is no independent linkage between sudden death and left ventricular outflow obstruction.

At present, there are no conclusive data supporting use of beta-blockers, calcium antagonists, or antiarrhythmics, including amiodarone, as preventers of cardiac arrest. An ICD appears to be an effective treatment in these high-risk patients. The ICD is most strongly warranted for prevention of sudden death in patients with prior cardiac arrest or sustained spontaneous ventricular tachycardia and in those with multiple risk factors for sudden death. Intense physical exertion constitutes a sudden death trigger in some individuals. Therefore, to reduce risk, it has been prudently recommended that athletes with unequivocal evidence of HC be disqualified from most competitive sports.

Atrial fibrillation. AF is the most common sustained arrhythmia in HC, accounting for unexpected hospital admissions and unscheduled work loss, and therefore usually justifies aggressive therapeutic strategies. Paroxysmal episodes of AF or chronic AF ultimately occur in about 25% of HC patients; the incidence increases with age and with enlargement of the left atrium. AF is reasonably tolerated by about one third of patients with HC and is not an independent determinant of sudden death. AF is associated with embolic stroke, however, which in itself may lead to death and disability. Amiodarone may be effective in reducing AF recurrences in these patients. In patients with chronic AF and HC, beta-blockers or verapamil effectively control the ventricular rate, although atrioventricular node ablation with permanent ventricular pacing is occasionally necessary.

Heart failure. Dyspnea, whether during exertion or at night, and fatigue are the most common symptoms in patients with HC, and they occur characteristically in the presence of normal or supranormal left ventricular contractility and independent of the presence of outflow obstruction. Symptoms of HC-related HF are usually deferred until adulthood but may occur at any age. Symptoms progress markedly to New York Heart Association class III or IV in about 20% of patients with HC, but such exertional disability evolves at varying rates, and deterioration is often gradual and punctuated by long periods of stability. The symptoms of HF in HC appear to be largely the consequence of left ventricular diastolic function and compromised left atrial systolic function, leading to elevated left atrial and left ventricular end-diastolic pressures with reduced stroke volume and cardiac output.

Chest pain suggestive of myocardial ischemia (with angiographically normal epicardial coronary arteries), either typical or atypical of angina pectoris, is a symptom commonly associated with exertional dyspnea. The role of myocardial ischemia in risk stratification in these cases is unresolved. Treatment of the HF usually begins with a beta-blocker or verapamil independent of whether outflow obstruction is present. Some investigators favor disopyramide over verapamil.

Operative treatment. If severe HF-related symptoms become unrelenting and refractory to pharmacological treatment, affecting lifestyle unacceptably, subsequent therapeutic choices are determined largely by whether the peak pressure gradient between the left ventricle and aorta is >50 mm Hg. Ventricular septal myotomy-myectomy (the Morrow procedure) has been the standard therapeutic option for adults and children with obstructive HC and severe drug-refractory symptoms. These operative candidates represent probably about 5% of the overall HC population. The myotomy-myectomy procedure usually lessens symptoms and increases exercise capacity for years after the procedure. The outflow gradient disappears, although it may take 6 months or so for it to disappear completely. The consistent relief of severe symptoms following myotomy-myectomy is evidence that marked outflow gradient and increased left-ventricular systolic pressure are of clinical significance to many patients. Some patients tolerate large gradients for long periods, however, with no or little disability. The outflow gradient, moreover, is typically labile and hemodynamically sensitive to alterations in ventricular volume and systemic vascular resistance.

In recent years, alcohol septal ablation has been used to thin the portion of ventricular septal thickness at the site of necrosis (61). This procedure involves injection of approximately 2 mL of alcohol into a septal perforator coronary artery, producing AMI; generally it requires insertion of an ICD, and a repeat procedure is required in as many as 25% of these patients. The procedure is not always successful in patients with a high outflow gradient. Despite the relatively small amount of long-term follow-up data on the alcohol ablation procedure, which has been performed now for about 5 years and in >3500 patients, there is little evidence that it is as good as the myotomy-myectomy procedure performed in a medical center where this operation is performed frequently. The myotomy-myectomy procedure has been done for 45 years and in approximately 3000 HC patients, meaning that the alcohol ablation procedure has been performed 10 to 35 times more frequently in the last 5 years than the septal myotomy-myectomy procedure. At the Minneapolis Heart Institute where Dr. Maron performs the procedure, 725 HC patients are being monitored; during the past 12 years, myotomy-myectomy has been performed in 20 (3%) and the alcohol ablation procedure in 5 (1%). It may be useful to reserve the septal myotomy-myectomy procedure for those <55 years and the alcohol septal ablation procedure for those >55 years. Dual-chamber pacing has some role in patients who are not candidates for either of these 2 procedures.
SUDDEN DEATH IN YOUNG COMPETITIVE ATHLETES

Barry J. Maron, MD

The first study of sudden death in competitive athletes <35 years arose from the National Institutes of Health in the 1980s. When Maron moved to Minneapolis in 1993, he set up a registry which is called the Minneapolis National Registry of Sudden Death in Athletes. >800 patients have been included in that registry (62). The most common causes of sudden death in these athletes were HC (26%); commotio cordis (20%); coronary arterial anomalies (14%); left ventricular hypertrophy of indeterminate cause, probably also HC (8%); myocarditis (5%); ruptured aortic aneurysm (i.e., Marfan syndrome) (3%); arrhythmogenic right ventricular cardiomyopathy (3%); tunneled coronary artery (3%); aortic valve stenosis (3%); atherosclerotic CAD (3%); dilated cardiomyopathy (2%); mitral valve prolapse (2%); and miscellaneous causes (9%). Sudden death in young athletes appears to occur at a frequency of about 1 per 200,000 per year. Screening is customary practice for most high school and college athletes, and about 5% of new HC cases are diagnosed via screening. The screeners include MDs and DOs (35%), registered nurses (20%), nurse practitioners (20%), chiropractors (9%), and others (2%). The number to be screened is huge: it is estimated that about 7 million high school competitive athletes, about 0.5 million college athletes, and about 0.2 million professional athletes compete in the USA.

EFFECTIVE LONG-TERM WEIGHT MANAGEMENT AND ITS IMPORTANCE

John P. Foreyt, PhD, Director, Behavioral Medicine Research; Professor of Medicine, Baylor College of Medicine, Houston, Texas

Obesity in the USA. Two thirds of Americans are overweight, and half of them are obese (63). In 1976, 47% of Americans were overweight or obese; by 1988, 56%; by 1999, 64%; and by 2004, 67%. Overweight is increasing in the USA by 1% per year. By 2040, at this rate, 100% of Americans will be overweight. The average American is 25 lb (11 kg) heavier than his or her same-sex great-grandparent was at the same age. The average soldier in the Civil War (1861–1865) was 68 inches (172 cm) tall and weighed 146 lb (66 kg); the average American man today is also 68 inches tall but weighs 171 lb (77 kg). We all tend to overestimate our height and underestimate our weight. The percentage of body fat today is 25% in the average US woman and 20% in the average US man. The average model in the USA today is 70 inches tall and has a body mass index (BMI) of 16 kg/m²; the average nonmodel adult woman is 64 inches tall and has a BMI of 24 kg/m². The average dress size in adult women outside the USA is size 2; the average dress size in US adult women is size 14. In the past 10 years, the average American adult has increased fat intake by 26 g/day and now consumes about 3400 kcal/day.

Why is obesity increasing in the USA at such rapid rates? There are multiple reasons, of course, but long hours at work and long commuting times, which in turn decrease time available for exercise and preparing food, and the presence everywhere of high-fat, high-calorie foods certainly play a role. Seven percent of Americans eat at a McDonald’s restaurant every day. Children in the USA have seen about 10,000 commercials advertising various foods by the time they grow up. Portion sizes in all restaurants have increased considerably in the past 10 years. The average American adult today consumes 200 more calories each day than 10 years ago. That much of a daily increase amounts to 100 more pounds in 10 years. The food industry in the USA produces 3800 calories per person per day, and most adults need only 2000 calories per day. Most American adults now consume >3000 calories per day.

Metabolic syndrome. Over 50 million, and possibly 75 million, Americans meet criteria for the metabolic syndrome, namely any 3 of the following: 1) abdominal obesity (waist size >40 inches in men, >35 inches in women); 2) triglycerides ≥150 mg/dL; 3) high-density lipoprotein (HDL) cholesterol <40 mg/dL in men and <50 mg/dL in women; 4) systemic hypertension (BP >130/85 mm Hg); and 5) fasting blood glucose >110 mg/dL. Weight loss decreases BP and serum levels of LDL cholesterol, triglycerides, insulin, and glucose and increases HDL cholesterol level. Twenty-five percent of Americans aged 50 years or older have the metabolic syndrome, and 40% of those aged ≥60 years have it.

Weight loss. The key factors in development of obesity are excessive food portion sizes and lack of exercise. The change of developing diabetes mellitus falls nearly 60% with a low-fat diet and 150 minutes per week of exercise. It’s not fat, protein, or carbohydrates that make people obese, it’s calories. Fad diets work, but the weight usually returns because most dieters do not modify their lifestyle. One simple strategy for effective weight loss is the 100/100 plan: eat 100 fewer kcal per day (1 cookie or 3 bites of a hamburger) and increase exercise by 100 kcal per day (walk 20 minutes per day). Strength training works as well as aerobics for weight loss, and multiple short bouts of exercise are as good as a long bout for weight loss. (Longer bouts of aerobic exercise, however, are better for cardiovascular fitness.) The healthiest diet in the world is the “Mediterranean” one, which comprises approximately 50% carbohydrates, 25% fat, and 20% protein. The Atkins diet, in contrast, comprises 11% carbohydrates, 56% fat, and 33% protein. Skipping breakfast is not beneficial. More obese people skip breakfast than nonobese persons.

Behavioral strategies for weight loss. The most important behavioral strategy is self-monitoring, which includes daily weighing and daily recording of all calories ingested (food diary). The average adult underreports calories consumed by 33% and overreports exercise by 50%. The second most important is stimulus control: avoid snacking and eating too quickly or too slowly. Third is cognitive restructuring: expectations must be realistic, such as losing 10% of body weight (about 20 lb) as a first goal. The average person wants to lose 37% of body weight; this type of goal is unrealistic. The 100/100 plan is a realistic option. Fourth, relaxation techniques such as meditation can reduce stress and decrease snacking. Fifth is social support: eat healthily and exercise together with family or friends.

Medical treatment for obesity. Sibutramine (Meridia) 10 mg/day, a selective serotonin and norepinephrine reuptake inhibitor, enhances satiety in some patients. This drug increases BP; however, and should be taken for no more than 3 months (64, 65). Orlistat (Xenical) (120-mg capsule with each fatty meal) blocks fat absorption in the gut by about 30%. This drug eliminates about 200 calories/day. Both of these drugs provide a modest (10-lb) weight loss in most patients but are ineffective at achieving more significant weight loss. Gastric bypass is now the treatment of choice for morbid obesity (BMI >40 kg/m²). This operation car-
Systolic BP increases linearly throughout life, regardless of the starting level (68). Diastolic BP increases to age 50 years, is level from 50 to 60 years, and then declines after age 60 years. Isolated diastolic hypertension is most common in patients in their 30s. Contrary to older teachings, isolated systolic hypertension is not a normal part of aging and does need to be treated. In patients >60 years of age with isolated systolic hypertension (systolic BP >160 mm Hg and diastolic BP <90 mm Hg), lowering the systolic BP by about 10 mm Hg with medications decreases the relative risk of stroke by about 40%, of coronary artery disease by about 30%, and of HF by about 40% (69).

The decreased frequency of cardiovascular events in hypertensive patients who take antihypertensive drugs is due to the lowering of the BP, irrespective of which drug caused that lowering. In a recent large trial, a diuretic (chlorthalidone), an ACE inhibitor (lisinopril), and a calcium antagonist (amlodipine) produced similar degrees of BP lowering and similar decreases in frequency of cardiovascular events (70).

Obstacles to BP control include lack of awareness (only about 70% of patients are aware of their hypertension), lack of treatment (only about 60% are treated), and lack of adequate treatment (only about 30% are at BP goal) (71, 72). Despite 5 years of close follow-up in a recent trial, only 67% of patients achieved their BP goal of <140/90 mm Hg.

Diastolic BP is much easier to control than systolic BP. Only about 70% of patients younger than 60 years, 50% of those aged 60 to 75 years, and 35% of those older than 75 years have their peak systolic pressures lowered by antihypertensive drugs to <140 mm Hg.

If systemic hypertension were better controlled or eliminated, the frequency of chronic HF would drop about 50% (73, 74). HF developing after myocardial infarction is of the systolic type; that developing in the setting of systemic hypertension not associated with myocardial infarction, in contrast, is usually of the diastolic type (75–78). Of cases of chronic HF in adults, about 50% are of the systolic type (low ejection fraction) and about 50% of the diastolic type (normal ejection fraction). Thus, treating hypertension is one of the best means of preventing chronic HF. In a study by Levy et al (73) of 392 HF patients, 357 (91%) had had systemic hypertension before HF, and only 35 (9%) had not. Of the same 357 patients, 151 (42%) by history had a prior myocardial infarct and 206 (58%) did not. Although the systolic type of HF is readily treated (ACE inhibitor/angiotensin receptor blocker, beta-blocker, diuretic, aldosterone antagonist), there is no proven therapy for diastolic HF.

**ATRIAL FIBRILLATION**

Michael Ezekowitz, MD, Department of Medicine, Drexel University College of Medicine, Philadelphia, Pennsylvania

**Epidemiology.** In 1909, the affliction of *pulsus irregularis perpetuus* was captured electrographically by Einthoven’s galvanometer and linked to the fibrillations of the cardiac atria. AF is the most common sustained cardiac arrhythmia, affecting ≥2 million people in the USA. The increasing prevalence of AF is due largely to an increasing elderly population, comorbid conditions, and perhaps lifestyle changes. The prevalence of AF increases from 0.5% in persons aged 50 to 59 years to about 10% in persons >70 years. The rate of hospitalization for AF has increased by 2 to 3 times in the past 2 decades. AF is more frequent in men than women and more common in whites than blacks (79).

**Genomics.** Although AF is typically an acquired disease, as many as 5% of patients have a heritable form. Several studies of multigenerational kindreds have localized mutations to specific chromosomes, identified genes, and even demonstrated the associated functional protein product. This advancement in genomic delineation fortifies the notion that AF is both a multifactorial and a genetically heterogenous disease (80).

**Mechanism.** Many patients with AF have anatomically and histologically abnormal atrial walls. The atria are nearly always dilated. This results in a difference in refractory period within the atrial tissue and promotes reentrant mechanisms. A shorter effective refractory period promotes AF. One mechanism of AF is the fractionation of a mother wave into daughter wavelets; in the presence of an enlarged atrium, a short refractory period, and slow conduction, this leads to sustained AF. The other mechanism is the presence of foci that discharge either continuously, leading to sustained AF, or in short bursts, triggering AF that is sustained by the first mechanism. These foci are commonly found in patients with paroxysmal AF and a structurally normal heart and are a target for ablation therapy. Depending on the pattern, AF is classified as single episode, paroxysmal (recurrent but self-terminating), persistent (recurrent and not self-terminating), or permanent (present all the time).

**Associations.** AF can have a cardiac or noncardiac cause (Table). Elimination of the precipitating cause can lead to conversion to sinus rhythm (SR) without any cardiac intervention. When AF occurs in the absence of any demonstrable underlying disease, it is known as “lone AF.”

**Maintenance of sinus rhythm vs rate control and anticoagulation.** The most common approach to AF is conversion to and
cause arrhythmias such as torsades de pointes and bradycardia. Generally need to be initiated in the hospital. The drugs can cause cardioversion is less effective and more time consuming than pharmacological cardioversion. The main agents available for pharmacological cardioversion are the class Ic, III, and Ia antiarrhythmics. These agents are more effective in recent-onset AF than in persistent AF. Their efficacy may be related to the route of administration, dose, and rapidity of administration. It is difficult, however, to compare different agents because various trials have used different agents at different dosages.

Flecainide and propafenone are commonly used class Ic agents. Both drugs have similar efficacy, and their intravenous forms act more quickly than their oral forms. Propafenone is superior to oral amiodarone or quinidine. Class Ic drugs are the most efficacious drugs in cardioversion of recent-onset AF; however, their efficacy is much lower in cardioversion of persistent AF. Class Ic drugs should not be used in patients with structural heart disease or abnormal ventricular function because of the high risk of arrhythmias.

Amiodarone, dofetilide, and ibutilide are the commonly used class III drugs. Amiodarone and dofetilide are more useful in cardioversion in persistent AF. Although amiodarone is used frequently, it is only modestly effective in cardioversion (about 25%), and cardioversion may not occur for days to weeks. Its long-term use is associated with side effects that lead to withdrawal from treatment and requires regular monitoring, but it can be highly effective in some cases. Dofetilide is administered orally, and its efficacy for cardioversion is about 30%. In-hospital initiation is necessary so that QTc interval and arrhythmias can be monitored. Dofetilide acts faster than amiodarone (90% of patients cardiovert within 36 hours with dofetilide) and has been shown to be safe in patients with myocardial infarction or HF, the 2 groups that are at a higher risk of developing proarrhythmic side effects. Intravenous ibutilide is superior to procainamide and sotalol but has no advantage over amiodarone in terms of efficacy. Pretreatment with ibutilide prior to electrical cardioversion can lead to a very high rate of conversion to SR (100% in one study) and at the same time decrease the amount of energy needed for cardioversion.

Quinidine is a class Ia drug used orally after ventricular response has been controlled. It is effective in cardioverting 60% to 80% of episodes of recent-onset AF.

Clinical response may be expected 2 to 6 hours after administration. Quinidine is now used infrequently because it is associated with significant adverse effects (including sudden death), and drugs with similar efficacy and fewer side effects are now available. Esmolol, a short-acting intravenous beta-blocker, can be used in patients who have had recent surgery. Digoxin, calcium antagonists, and other beta-blockers have not been shown to be effective in cardioversion (51–53). Sotalol is currently not recommended for cardioversion.

Electrical cardioversion. Electrical cardioversion has been used since the 1960s for managing arrhythmias. Direct-current cardioversion involves delivery of an electrical shock synchronized with the intrinsic activity of the heart, usually by sensing the R wave of the electrocardiogram. Successful electrical cardioversion of AF depends on the nature of the underlying heart disease and the current density delivered to the atrial myocardium. Rates of elec-

### Table. Conditions associated with atrial fibrillation

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<td>Pericarditis and myocarditis</td>
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<td>Idiopathic dilation of the right atrium</td>
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maintenance of SR. The justification for this approach is that it should decrease symptoms, increase cardiac output, improve exercise tolerance, prevent tachycardia-induced myopathy, lower the risk of stroke and thromboembolism, reduce mortality, and improve quality of life. This traditional strategy is associated with a significant failure rate and side effects.

In patients likely to have recurrent AF, rate control is as effective as rhythm control. Attempts to maintain SR results in more adverse reactions and hospitalizations than the rate-control strategy. In both groups, most strokes occur after warfarin has been stopped or when anticoagulation is inadequate (i.e., subtherapeutic international normalized ratio). In patients who are likely to have recurrent AF, rate control and anticoagulation seem to be the most appropriate strategy.

Cardioversion. New-onset AF (24–48 hours) has the highest rate of spontaneous cardioversion to SR—as high as 50% to 70%—and may not require intervention for rhythm control. Patients with persistent AF have a low rate of spontaneous cardioversion and may benefit from electrical or pharmacological cardioversion. Electrical cardioversion is recommended if the patient’s condition is unstable because of hypotension, cardiac ischemia, or HF attributable to AF. It requires hospitalization and sedation and can cause myocardial injury, arrhythmias, and local side effects such as skin burns and muscle injury. Pharmacological cardioversion is less effective and more time consuming than electrical cardioversion. Pharmacological agents such as sotalol generally need to be initiated in the hospital. The drugs can cause arrhythmias such as torsades de pointes and bradycardia.
trical cardioversion of AF vary from 70% to 90%. Using higher initial energy (200 J for biphasic and 300–360 J for monophasic) results in fewer shocks and less energy use overall. Pretreatment with ibutilide just before cardioversion increases success rates to as high as 100%. Short duration of AF, presence of atrial flutter, and younger age are predictors of success, whereas left atrial enlargement, premature atrial complexes, underlying heart disease, and cardiomegaly are predictors of recurrence.

Transesophageal echocardiography. Transesophageal echocardiography (TEE) is the most sensitive and specific technique for detection of left atrial thrombus. Compared with transthoracic echocardiogram, TEE provides a much better view of the left atrial appendage. TEE is used in AF to stratify the risk of stroke and to guide cardioversion. Traditionally, patients receive anticoagulant for 3 to 4 weeks before cardioversion to prevent embolism. Besides the shorter wait between the presentation of AF and cardioversion, the advantages of TEE include a possible impact on atrial remodeling as a result of early cardioversion and risk stratification in case of an urgent cardioversion. All patients should receive anticoagulant therapy prior to, during, and for a variable period after cardioversion. Unless there is a contraindication or the patient is at low or intermediate risk for stroke, anticoagulation should be continued indefinitely.

Maintenance of sinus rhythm. AF, a chronic disorder, commonly recurs. Female gender, age ≥55 years, AF duration ≥3 months, atrial enlargement, rheumatic heart disease, underlying heart disease, and hypertension increase the risk of recurrence and the need for prophylactic treatment with antiarrhythmic drugs to maintain SR. Class III drugs, i.e., amiodarone and dofetilide, are the most effective agents in maintenance of SR. Amiodarone is significantly better than sotalol or propafenone, although it is associated with more adverse effects and withdrawal symptoms. Dofetilide has been shown to maintain SR in about 70% of patients with persistent AF at 1-year follow-up. Propafenone and flecainide are useful in maintaining SR in patients with no structural heart disease and are the first-choice agents in these patients. These 2 drugs and sotalol are associated with fewer side effects than other antiarrhythmic drugs. Now that newer, safer antiarrhythmics with equal or superior efficacy are available, class Ia agents are not commonly used. Beta-blockers, though only moderately effective in prevention, also control heart rate and reduce associated symptoms. In patients who develop AF only during exercise, a beta-blocker may be effective.

Surgical techniques. The Maze procedure controls AF in >90% of selected patients. The principle behind the procedure is that the creation of barriers to conduction in the atrium prevents the propagation of reentrant wavefronts, thereby inhibiting sustained AF. This is generally achieved by surgically creating scars in the left atrial wall and by isolating pulmonary veins. The Maze-III procedure is a more refined version that is currently used. The mortality rate of the isolated Maze operation is 1% to 2%. Sinus node dysfunction due to disruption of blood supply may occur, necessitating permanent pacemaker implantation. This procedure is indicated in highly symptomatic, drug-resistant AF or in patients with thromboembolism due to AF while on warfarin. It can also be done as an add-on to other cardiothoracic surgery.

Catheter ablation. Arrhythmogenic foci in pulmonary veins, right atrium, left atrium, superior vena cava, or coronary sinus may initiate AF. The selection of catheter-based radiofrequency ablation of these foci, pulmonary vein isolation, or compartmentalization of the atrium using linear ablation depends on the underlying electrophysiology. Pulmonary vein isolation can be done by using either 3-dimensional nonfluoroscopic electroanatomical mapping or circular mapping. These procedures can be successful in as many as 80% of patients who have structurally normal hearts and undergo single-focus ablation or pulmonary vein isolation. The success rate in more persistent AF is about 60%. Because of the potential for recurrence, patients may continue to require antiarrhythmic medications after radiofrequency ablation. Complications of catheter ablation for AF include pericardial effusion and tamponade, systemic embolism, pulmonary vein stenosis, and phrenic nerve palsy.

Heart rate control. An alternative to maintenance of SR in patients with paroxysmal or persistent AF is control of the ventricular rate. The rate is considered controlled when it is between 60 and 80 beats per minute at rest and between 90 and 115 beats per minute during moderate exercise. The adequacy of rate control during AF can be determined from symptoms. Efficacy of rate-control therapy for AF depends mainly on suppression of conduction across the atrioventricular node, and hence drugs that prolong the node’s effective refractory period are the ones that generally are effective. Sinus bradycardia and heart block may occur in some patients, particularly the elderly, as an unwanted effect of pharmacological intervention with beta-blockers, digoxin, or calcium antagonists.

For rapid control of ventricular response in acute AF, short-acting intravenous agents are used. A calcium antagonist (generally diltiazem) or beta-blocker is the first choice, but digoxin and amiodarone are also used. The response to diltiazem is transient, and repeated doses or a continuous intravenous infusion may be necessary to maintain heart rate control. Intravenous beta-blockers may be particularly useful in patients in a state of increased sympathetic activity. Short-acting esmolol in particular helps careful titration of heart rate. Intravenous digoxin may effectively slow the ventricular rate at rest, but there is a delay in the onset of a therapeutic effect in most patients, and a peak effect does not develop for up to 6 hours. The combination of digoxin and atenolol can be used to control ventricular rate. Intravenous amiodarone is effective and well tolerated in critically ill patients and those with HF who develop rapid AF.

In persistent AF, when rate control is the mainstay of treatment, oral forms of beta-blocker, calcium antagonist, and digoxin are generally used. Because digoxin is unable to control heart rate during exercise, it is generally combined with a beta-blocker or calcium antagonist. Digoxin is no longer the first choice except in patients with depressed left ventricular systolic function.

Some patients have a variable heart rate (e.g., sick sinus syndrome) or develop bradycardia in response to rate-control treatment. These patients may be candidates for ventricular pacing combined with rate-controlling agents. Atioventricular nodal ablation with permanent pacemaker implantation is highly effective in controlling heart rate and improving symptoms. This approach is generally indicated for patients whose heart rate is inadequately controlled on drugs (refractory AF), who develop unacceptable side effects to medications, or who are noncompliant.
**Stroke prevention.** The most worrisome complication of AF is stroke. The risk of stroke in patients with AF is nearly 6 times greater than in patients in SR, and an estimated 60,000 people have strokes associated with AF each year in the USA. Approximately 70% of individuals with AF are between 65 and 85 years of age, and patients in this age group are at the highest risk of developing a stroke. The risk of stroke from AF increases significantly with age. In patients aged 50 to 59 years, the risk of stroke is about 1%; between 60 and 69 years, 3%; between 70 and 79 years, 10%; and between 80 and 89 years, nearly 25%. The risk of stroke is the same for paroxysmal and persistent AF. Individuals <65 years with lone AF do not have increased risk (0.5%–1.0% per year). AF is also associated with an increase in silent cerebral infarcts and may be associated with cognitive dysfunction.

Thromboembolism is the major cause of stroke in AF, and it is logical that anticoagulation would be beneficial in stroke prevention. The role of warfarin and aspirin in prevention has now been established beyond doubt. Not all patients with AF need anticoagulation, however. Several trials have revealed that only patients at a higher risk of stroke (AF with at least one risk factor) definitely benefit from warfarin, whereas low-risk patients need only aspirin. Patients with moderate risk (aged 65–75 years) can be treated with either warfarin or aspirin. In a pooled analysis of 5 primary trials, conventional-dose warfarin was associated with risk reduction of 68% (4.5%–1.4% per year). Warfarin decreased the risk of stroke by 85% in women and by 60% in men. A metaanalysis of 6 trials comparing aspirin with placebo found that aspirin reduced the relative risk of stroke by about 20%; the absolute risk reduction for primary prevention was 1.5% per year. In secondary prevention, warfarin yielded a 65% risk reduction (from 12% per year to 4%). Aspirin yielded a 30% risk reduction in outcomes (death from vascular disease, any stroke, myocardial infarction, or systemic embolism). An international normalized ratio range of 2.0 to 3.0 is currently recommended for all patients, including the elderly. After cardiovascular patients should receive anticoagulant therapy for life unless they have a contraindication, a low risk for stroke, or a low risk for AF recurrence. At present, only 50% of patients with AF who need anticoagulation are receiving anticoagulant therapy. Reasons for underutilization of warfarin include contraindications to anticoagulation, patient preference, and perception of a higher than actual rate of bleeding complications.

**Neuroprotective effects of statins.** Stroke continues to be the third most common cause of death in the USA after heart disease and cancer. Approximately 750,000 strokes (new or recurrent) occur each year. The direct cost of providing care for stroke victims in the USA is in the range of $17 billion per year. Although mortality rates for heart disease have decreased over the past 2 decades, stroke mortality rates have declined little since 1990.

There is a linear relation between the risk of coronary heart disease and high serum levels of total cholesterol and LDL cholesterol. The preponderance of evidence has failed to show a significant relation between the risk of stroke and total serum cholesterol concentrations. Trials of nonstatin lipid-lowering agents have failed to show a neuroprotective effect.

Despite the poor predictive values of total cholesterol and LDL cholesterol levels for stroke, statin therapy reduced stroke in the Myocardial Ischaemia Reduction with Aggressive Cholesterol Lowering with atorvastatin trial (MIRACL) and other trials. In MIRACL, which included 3086 subjects with unstable angina pectoris or non–Q-wave AMI and mean LDL cholesterol levels of 124 mg/dL, patients who received atorvastatin therapy had a 50% reduction in stroke incidence. A metaanalysis of 13 trials, including the Scandinavian Simvastatin Survival Study (4S), the Cholesterol And Recurrent Events (CARE) trial, and the West Of Scotland Coronary Prevention Study (WOSCOPS), studied the effects of statins (lovastatin, pravastatin, or simvastatin vs placebo) in 19,921 subjects. The stroke rate was reduced from 2.38% in the placebo group to 1.67% in the statin group. There was no association between the magnitude of cholesterol reduction and the relative risk of stroke.

Until now, the lipid-lowering properties of statins have dictated their use in clinical practice. Recent trials using statins (e.g., the MIRACL and HPS studies) have shown significant stroke reduction even in the absence of dyslipidemia. It is probably appropriate to prescribe statins as well as aspirin, clopidogrel, or a combination of dipyridamole and aspirin to all patients at high risk of stroke. Identifying which pleiotropic property of the statin is responsible for the reduction in stroke would be helpful in deciding which patients would benefit from this therapy.

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An outsider’s impression of the Williamsburg Conference

EDITOR’S NOTE: I asked my administrative assistant to write this article about her impressions. These notes could apply to any meeting. The main point is that we physicians represent the medical profession wherever we are.

As one of the women who helped at the registration desk, I was asked to write about my impressions of the conference. I was a bit intimidated about writing such an article. After all, I am just a recent college graduate in my first real job. Who am I to comment? I concluded that several of the physicians I have worked with might find it interesting to know what people like me take note of.

As physicians interacted with me at the registration desk, several were frazzled and would interrupt me or the other ladies in midsentence. They would talk over us and practically walk off as we were talking to them. The other ladies and I felt unimportant, but the physician was the one who looked unprofessional. By contrast, other physicians were in such high spirits that more than one spoke with us for a long time. I was able to meet fascinating people and hear wonderful stories.

While it was interesting seeing spouses and guests sneak into sessions, which were for registrants only, it was even more interesting to watch physicians sneaking out of the sessions! Leaving early may seem reasonable, but most attendees recorded the full number of continuing medical education hours. In addition, several presenters (who are paid an honorarium to speak and thus are not eligible for credit) claimed the meeting hours in their entirety. I was surprised, and the experience caused me to wonder: Are these physicians sometimes dishonest in other aspects of their profession?

At Baylor, the cardiologists and vascular surgeons appear to be extremely healthy; they “practice what they preach.” That was not the case with all the physicians who attended the meeting. I saw an obese cardiologist taking a smoke break from a conference on heart disease. If I were a patient of such a physician, I would not have much confidence in his advice about making lifestyle changes to improve my health.

This was my first experience planning faculty dinners, obtaining travel schedules from the presenters, and preparing the syllabus. Quite a few of the presenters were efficient in turning in material for the syllabus, but others waited until the day the syllabus had to be sent to the printer or submitted nothing at all—even after numerous follow-up requests. The syllabus was not a popular item on the evaluations. Attendees wanted everyone’s slides and not “publications that have nothing to do with the talk”; however, we could provide only the material we received. For the most part, the evaluations were very positive. Some people, though, always find something to complain about—the parking, the expensive dining options at the hotel, the crowds.

This program gave me a lot to think about and, more importantly, to learn. I know very little about cardiology, but I now know a little more about physicians in the field. Physicians are people just like everyone else, and they come from all walks of life. I think I speak for most of the attendees when I say that the conference was outstanding, and we hope to attend again.

—ANDREA BEGGS