The survival rate of cancer patients has increased in the last 25 years. In the United States, the 5-year relative survival rate of patients diagnosed with cancer between 1975 and 1977 was 50%, but it has increased to 68% for patients diagnosed between 1999 and 2005. The introduction of more successful anticancer treatments has contributed to improved survival. Currently, there are more than 12 million cancer survivors in the United States alone. However, with longer survival, the long-term adverse treatment effects have become increasingly important.

"Cardiotoxicity is a common adverse effect of many treatments, and it may affect patient survival and quality of life independent of the oncological prognosis," according to Farouk Mookadam, MB, BCh, a cardiologist at Mayo Clinic in Arizona. "Given the prevalence of cancer and heart disease, it is common for cardiovascular comorbidities to influence the choice of cancer treatment."

Virtually all anticancer drugs that target tumor cell death may result in collateral injury to other healthy tissues. Bone marrow suppression and gastrointestinal toxicities associated with chemotherapy are well recognized and accepted side effects for the benefits that may accrue from the chemotherapy. "Much less appreciated, however, are the cardiotoxic effects of cancer treatment," says Donald W. Northfelt, MD, a medical oncologist at Mayo Clinic in Arizona. These adverse effects can include:

- Direct cytotoxic effects of chemotherapy resulting in systolic and diastolic dysfunction
- Cardiac ischemia
- Cardiac arrhythmia
- Pericarditis
- Chemotherapy-induced repolarization abnormalities

Furthermore, radiation therapy without adequate cardiac shielding may result in coronary artery disease, valvular heart disease, pericardial injury, and myocardial disease from fibrotic changes that occur postradiation. It is not uncommon for chemotherapy to follow radiation treatment, and in some organs such as the heart, this dual and serial insult can result in a higher likelihood of cardiotoxicity.
Common Cardiotoxic Chemotherapeutic Agents

Generally, chemotherapeutic cardiac toxicity is classified into 2 types. Type 1 chemotherapy-related left ventricular (LV) systolic dysfunction is caused by agents such as doxorubicin, epirubicin, idarubicin, cyclophosphamide, and docetaxel; it is usually dose related and not reversible. Type 2 chemotherapy-mediated cardiotoxicity results from agents such as trastuzumab, lapatinib, sunitinib, imatinib, and bevacizumab; it is generally not dose related and may be associated with reversible myocardial dysfunction.

Anthracyclines

Anthracyclines such as doxorubicin or epirubicin have been key components of many cytotoxic regimens since their introduction in the 1960s. Anthracyclines cause inhibition of DNA polymerases and DNA fragmentation. Cardiotoxic effects of anthracyclines are believed to be related to myocyte injury by oxygen free radicals and lipid peroxidation, resulting in left ventricular dysfunction and congestive heart failure (CHF).

"The occurrence of CHF is dose and schedule dependent, increases in incidence over time, and occurs in up to 20% of patients in some reports," says Joerg Herrmann, MD, a cardiologist at Mayo Clinic in Rochester. "In the past, the risk was believed to increase significantly with a cumulative doxorubicin dosage of 550 mg/m², but there is solid evidence that this threshold may be lower or even nonexistent." The cardiotoxic effects of anthracycline therapy appear to be increased in patients with preexisting heart disease or advanced age. Late-onset anthracycline cardiotoxicity, presenting as left ventricular dysfunction a year or more after chemotherapy, is increasingly recognized. The risk of cardiotoxicity may also be increased when anthracyclines are used in combination with other potentially cardiotoxic chemotherapeutic agents, such as kinase inhibitors (the "dual hit" theory).

Table. Cumulative Incidence of CHF and/or Cardio-myopathy with Select Chemotherapeutic Agents

<table>
<thead>
<tr>
<th>Cumulative Incidence (%)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthracycline</td>
<td>1.2</td>
<td>2.0</td>
<td>2.7</td>
<td>3.5</td>
<td>4.3</td>
</tr>
<tr>
<td>Trastuzumab</td>
<td>3.6</td>
<td>5.8</td>
<td>7.8</td>
<td>9.9</td>
<td>12.1</td>
</tr>
<tr>
<td>Anthracycline + Trastuzumab</td>
<td>6.2</td>
<td>9.8</td>
<td>13.2</td>
<td>16.5</td>
<td>20.1</td>
</tr>
<tr>
<td>Other agents</td>
<td>1.3</td>
<td>2.1</td>
<td>2.9</td>
<td>3.7</td>
<td>4.5</td>
</tr>
<tr>
<td>None</td>
<td>0.9</td>
<td>1.4</td>
<td>1.9</td>
<td>2.5</td>
<td>3.1</td>
</tr>
</tbody>
</table>

(Reproduced, with permission, from Journal of the National Cancer Institute 2012;104:1293-1305.)

Kinase Inhibitors

Protein kinases in healthy cells may act as tumor suppressors under normal circumstances. Dysfunctional signaling of various kinases is linked to tumorigenesis and tumor angiogenesis. Kinase inhibitors have been specifically developed by "rational drug design." The goal of targeted therapy is to improve antitumor activity with a lower risk of adverse effects than traditional anticancer therapies. Kinases are also present in cardiac myocytes and are responsible for phosphorylating the amino acids threonine, serine, and tyrosine. Cardiac toxicity from the kinase inhibitors may be the result of either of 2 mechanisms interfering with myocyte kinase activity: "on-target" toxicity resulting when the tyrosine kinase also serves an important role in normal cardiomyocyte function, and "off-target" toxicity occurring when an agent inhibits a kinase not intended to be a drug target.

Tyrosine kinase inhibitors are common antineoplastic agents and are of 2 types: small molecule kinase inhibitors, which target both receptor and nonreceptor kinases, and monoclonal antibodies, which typically target growth factor receptor tyrosine kinases. For example, imatinib (Gleevec®; Novartis AG, Basel, Switzerland) is a small molecule tyrosine kinase inhibitor used in the treatment of chronic myelogenous leukemia. Many of the small molecule kinase inhibitors induce hypertension in a significant proportion of patients. The development of hypertension can be acute and severe, resulting in mitigation of the chemotherapy either by dose reduction or schedule change—or even cessation, with obvious consequences.

Trastuzumab (Herceptin®; Genentech, South San Francisco, California) is a monoclonal antibody targeting human epidermal growth factor receptor 2 (HER2, also known as ErbB-2). It has led to a significant breakthrough in the treatment of breast cancers that overexpress HER2 receptors (up to 30% of all breast cancer cases), a variant of the disease traditionally associated with a poor prognosis. "Unlike anthracyclines, cardiotoxicity associated with trastuzumab is not dose dependent, and ultrastructural changes typical of anthracycline toxicity are not seen on cardiac biopsy specimens," says Carrie A. Thompson, MD, a medical oncologist at Mayo Clinic in Rochester. "The clinical outcome of patients with trastuzumab-induced cardiotoxicity is more favorable than that associated with anthracyclines; cardiac function usually recovers after drug withdrawal and initiation of CHF therapy."

Combination Adjuvant Anthracyclines and Trastuzumab

Data from randomized controlled trials in women with HER2-positive disease provide clear evidence of disease-free survival and overall survival benefits when trastuzumab is added to standard anthracycline chemotherapy in the adjuvant setting. Mortality is reduced by approximately one-third, but the risk of heart toxicity is 5 times more likely for women receiving trastuzumab than for women receiving standard...
therapy alone (Table). Furthermore, concurrent anthracycline/trastuzumab chemotherapy strategies consistently lead to higher adverse event rates compared to sequential treatment strategies.

Cardiotoxicity is a particularly important issue given the large number of women with breast cancer receiving combination anthracycline/trastuzumab therapy in the adjuvant setting. On average, 17% of patients receiving this treatment for the most aggressive forms of breast cancer have to stop therapy due to cardiac complications. Cardiotoxicity is reversible in the majority of patients if the treatment is stopped immediately. Several strategies to reduce anthracycline/trastuzumab-induced cardiotoxicity have been proposed (but are yet unstudied in controlled clinical trials), with key elements including establishing stringent left ventricular ejection fraction (LVEF) criteria for patient selection, monitoring cardiac function during therapy, discontinuing potentially cardiotoxic therapy when cardiotoxicity arises, and instituting heart failure medications early.

Monitoring for Toxicity
In clinical oncology practice, an asymptomatic decrease in LVEF is the most commonly encountered form of cardiotoxicity. However, echocardiographic LVEF assessment has shown low diagnostic sensitivity and low predictive power in detecting subclinical myocardial injury. Newer tests of systolic function, such as myocardial strain and strain rate, are sensitive measures of tissue deformation that have been shown to be useful in detecting subclinical disease in a number of disease settings. Strain rate imaging is a noninvasive technique that can quantitatively analyze myocardial mechanics by detecting speckles from the myocardium with 2-dimensional echocardiography (Figure). Directional motion is analyzed in an angle-independent manner, unlike tissue Doppler imaging. In general, a reduction of longitudinal strain greater than 10% from baseline after 3 months may be able to predict future cardiac injury with a sensitivity of about 79% and a specificity of about 79%.

Specialty Integration
The objectives of an interdisciplinary and integrative management approach to cancer patients with cardiovascular risks or who develop cardiovascular injury are:

- Early detection of patients at risk of cardiotoxicity
- Early institution of cardioprotective agents
- Preventing treatment mitigation of the chemotherapeutic agent
- Eliminating as much of the cancer as possible while minimizing collateral damage, such as cardiotoxicity

The risk of delayed toxicity may be less relevant for patients with advanced disease and a limited lifespan. However, in the setting of neoadjuvant and adjuvant treatment, the laudable goal should be that a cancer survivor of today does not become the heart failure patient of tomorrow. To that end, Mayo Clinic has developed specialty cardio-oncology clinics to provide collaborative evaluation and care of these patients. The objectives of these clinics are to provide expert advice and guidance for patients who will undergo cancer treatment and aid in the prevention, monitoring, and management of cardiovascular toxicities. “With the proliferation of new antineoplastic agents, the development of new combination therapy protocols, longer patient survival, and the large population of cancer patients with preexisting heart disease, formal specialty collaboration is increasingly recognized as the optimal practice model to deliver integrated care to this population of patients,” says Dr Herrmann. Furthermore, this collaborative effort will facilitate basic, translational, and clinical research efforts in the attempt to optimize clinical outcomes.

For more information about cardio-oncology at Mayo Clinic, visit http://www.theheart.org/columns/mayo-clinic-chair/cardioncology-what-every-cardiologist-should-know.do.

ANNOUNCEMENTS

Mayo Clinic in Arizona is sponsoring Rally for Red, a cardiac health campaign this year on ABC15 TV in Phoenix. The campaign includes monthly segments on Sonoran Living Live featuring Mayo Clinic heart specialists. To watch the debut segment featuring Robert Scales, director of the Cardiac Rehabilitation Program at Mayo Clinic in Arizona, visit http://www.youtube.com/watch?v=q-cVyxz5Pms.
During the past 3 decades, the implantable cardioverter-defibrillator (ICD) has evolved from a "last-ditch effort" for the treatment of patients with malignant ventricular arrhythmias to a widely used treatment option for patients who are at risk of sudden cardiac death. Since ICD treatment requires surgery and implantation of a complex and exquisitely engineered device, it is sometimes difficult to decide whether ICD implantation is appropriate in an individual patient. Randomized clinical trials are designed to identify a relatively homogeneous population at risk of the treatment endpoint of interest to evaluate the effects of a specific therapy. "While clinical guideline statements developed by professional societies are extremely important for synthesizing scientific data and expert consensus and thus provide general guidance to the clinician for best practices, they cannot account for all of the variables in clinical medicine," says Fred Kusumoto, MD, an electrophysiologist at Mayo Clinic in Florida.

Two seminal trials established the ICD as an effective therapy that reduces mortality. In the Multicenter Automatic Defibrillator Implantation Trial II (MADIT-II), patients with an ejection fraction < 30% due to prior myocardial infarctions were randomized to best medical therapy or best medical therapy with an ICD. After 2-year follow-up, patients randomized to receive an ICD had a 20% reduction in risk of death. Using a slightly different patient population, the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) randomized patients with an ejection fraction < 35% due to either ischemic or nonischemic cardiomyopathy, and NYHA Class II or Class III heart failure, to receive an ICD, amiodarone, or placebo. After 46-month follow-up, patients who received an ICD had a 23% reduction in mortality compared to the placebo and amiodarone groups.

When applying the results of clinical trials to management of individual patients, it is important to clearly understand the inclusion and exclusion criteria for the trial and carefully examine the characteristics of the patients who were actually enrolled. In MADIT-II and SCD-HeFT, there were no upper age limits, but the average age of patients enrolled in both studies was 60 to 64 years, and both included mostly men (84% in MADIT-II and 76% in SCD-HeFT). Between 2000 and 2010 in the United States, the population older than 65 years grew at a faster rate (15%) than the total population (10%), and there are now nearly 6 million people in the United States who are older than 85 years. In a recent analysis of the National Cardiovascular Data Registry for ICDs, 47% of ICD implants were in patients > 70 years of age, and 16% were in patients > 80 years of age. In addition to underrepresenting the elderly and women, clinical trials have not enrolled patients with potentially terminal medical illnesses. This represents a significant gap in our understanding of the clinical application of ICD therapy. For example, in patients with stage I to III breast cancer, 5-year survival rates can range from 45% to 95%. In a patient with breast cancer who also has cardiomyopathy and is at risk of sudden cardiac death, is an ICD an appropriate treatment option?

Careful examination of MADIT-II and SCD-HeFT can provide some insight into these important questions. The survival benefit from ICD therapy appears to increase over time, with the survival curves beginning to diverge at 1 year in MADIT-II and 2 years in SCD-HeFT, so that by 3 years there is an absolute 6% to 9% reduction in mortality associated with ICD implantation (Figure). To put this data into perspective, chronic beta-blocker therapy after myocardial infarction was associated with an absolute 2.5% to 3.5% reduction in mortality in the Carvedilol Post-Infarct Survival Control in left Ventricular Dysfunction (CAPRICORN) study and the Beta-Blocker Heart Attack Trial (BHAT).

There is some information available on the use of ICDs in the elderly. In a prespecified sub-analysis of SCD-HeFT, ICD therapy was not
trials actually underestimated the possible benefits of ICD therapy," says Dr Kusumoto.

It is obvious that the decision whether to implant an ICD in an older patient or a patient with a potentially terminal condition is highly nuanced and requires a sophisticated and balanced approach. The decision is best served by close collaboration among a group of physicians who have expertise in different medical specialties and taking care of different patient groups. Once implanted, the ICD must be programmed carefully and based on individual patient characteristics to maximize the benefit of therapy. While clinical guidelines and algorithms are extremely useful for providing broad recommendations, the decision to implant an ICD in the patient "sitting in front of you" requires thoughtfulness and consideration of the clinical situation and the patient's wishes.

During the last 5 years, several studies have documented and studied the relationship between unnecessary shocks and clinical outcomes. In the recently published Multicenter Automatic Defibrillator Implantation Trial-Reduce Inappropriate Therapy (MADIT-RIT), careful programming of the ICD to reduce inappropriate therapy was compared to standard programming. Programming that required higher tachycardia rates or longer periods of time in tachycardia before a shock was delivered were associated with a 45% to 55% reduction in mortality. In MADIT-RIT, approximately 50% received an ICD that could also provide cardiac resynchronization therapy, and the 2-year mortality of the conventionally programmed group (10%) was lower than the 2-year mortality of the ICD group in MADIT-II (16%). "The findings of MADIT-RIT do bring up the intriguing notion that the large original ICD trials actually underestimated the possible benefits of ICD therapy," says Dr Kusumoto.

In contrast, in a post hoc analysis of MADIT-II, mortality reductions with ICD therapy were similar in patients < 75 years and ≥ 75 years. A recent meta-analysis studied the effects of age by combining data from MADIT-II and SCD-HeFT with three smaller primary prevention trials, the Multicenter Unsustained Tachycardia Trial (MUSTT), the Defibrillators in Nonischemic Cardiomyopathy Treatment Evaluation (DEFINITE) trial, and the first Multicenter Automatic Defibrillator Implantation Trial (MADIT-I). When these studies are combined, 597 patients > 75 years old were identified (12% of the total number of patients enrolled), and the ICD remained beneficial for reducing all-cause mortality (HR 0.73; 95% CI, 0.51-0.97; p = 0.03).

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New Clinical Trial Evaluates Choice of Treatment for Left Main Coronary Disease

The development of coronary artery bypass grafting (CABG) in 1967 and percutaneous coronary interventions (PCI) in 1977 were seminal events in the evolution of the treatment of coronary artery disease (CAD). Multiple clinical trials in the intervening years have helped to clarify appropriate utilization of each approach. Meanwhile, improvements in anesthesia, operative technique, and postoperative care, as well as technological advances in percutaneous systems, have resulted in reduced hospital stays and improved outcomes and durability for both procedures. "However, these advances challenge the medical community to continually reevaluate the safety, efficacy, and indications for these treatment modalities," according to Richard C. Daly, MD, a cardiovascular surgeon at Mayo Clinic in Rochester.

Traditionally, individuals with significant stenosis of the left main (LM) coronary artery were treated with CABG rather than PCI due to the large area of myocardium at risk, the dire consequences of abrupt periprocedural closure, and concerns regarding durability of PCI. The introduction of drug-eluting stents, and now second-generation drug-eluting stents, has prompted reappraisal of the possible role of PCI for some patients with LM disease. There is now evidence to suggest that PCI with drug-eluting stents may be appropriate treatment for some patients with LM disease, especially when the total burden of coronary disease is not high.

Although the randomized Synergy Between PCI With TAXUS and Cardiac Surgery (SYNTAX) trial found that PCI with the first generation of paclitaxel-eluting stents in patients with 3-vessel and LM disease was inferior to CABG for the composite primary endpoint of death, myocardial infarction (MI), stroke, or revascularization at 1 year, a post hoc analysis suggested that subgroups with low anatomic SYNTAX scores had similar outcomes with PCI compared to CABG.

Second-generation drug-eluting stents have been shown to demonstrate lower restenosis and stent thrombosis rates than first-generation stents. The Comparison of the Everolimus-Eluting XIENCE-V Stent With the Paclitaxel-Eluting TAXUS LIBERTE Stent in All-Comers (COMPARE) trial was a single-center "real-world" prospective randomized study conducted in the Netherlands comparing the clinical performance of paclitaxel- and everolimus-eluting stents. The rate of major adverse events (all death, nonfatal MI, and target vessel revascularization) was significantly higher in the paclitaxel group than in the everolimus group at 1 year (9.1% vs 6.2%; p = 0.023) and 2 years (13.7% vs 9.0%; p = 0.0016). The everolimus group also had lower rates of stent thrombosis at 1 year (2.6% vs 0.7%; p = 0.002) and 2 years (3.9% vs 0.9%; p = 0.0001), primarily due to a reduction in early stent thrombosis.
bosis. However, only 2% of the patients in this trial had unprotected LM stenting.

Mayo Clinic in Rochester is participating in the Evaluation of XIENCE Prime or XIENCE V Versus Coronary Artery Bypass Surgery for Effectiveness of Left Main Revascularization (EXCEL) clinical trial, which will clarify the role of second-generation drug-eluting stents for LM disease. The EXCEL trial will randomize 2600 patients at 165 sites globally; 1300 will receive stenting, and 1300 will undergo CABG. Inclusion and exclusion criteria are outlined in box 1 and box 2. In addition to clinical endpoints (box 3), the study will evaluate quality-of-life scores and analyze procedural and follow-up costs for the full study period. For information about enrolling a patient in the EXCEL trial at Mayo Clinic in Rochester, please contact study coordinators Mary E. Peterson (507-255-4080) or Deborah A. Rolbiecki (507-255-5027).

Box 1. Main Inclusion Criteria
- Unprotected LMCA disease with angiographic stenosis ≥ 70% requiring revascularization as assessed by both interventional cardiologist and cardiac surgeon
  OR
- Unprotected LMCA disease with angiographic stenosis ≥ 50% but < 70% requiring revascularization as assessed by both interventional cardiologist and cardiac surgeon AND one of the following:
  - Noninvasive evidence of ischemia referable to a LM lesion
  - IVUS MLA ≤ 6.0 mm²
  - FFR ≤ 0.80
- Significant LM equivalent disease: ostial lesions of LAD and CFX ≥ 70%, OR
- One or both of the ostial LAD and CFX lesions ≥ 50% and < 70% AND one of the following:
  - Noninvasive evidence of ischemia
  - IVUS MLA ≤ 4.0 mm²
  - FFR ≤ 0.80
- Clinical and anatomic eligibility for both PCI and CABG
- Silent ischemia, stable angina, unstable angina, or recent MI

Box 2. Main Exclusion Criteria
- Prior PCI LM at any time
- Prior CABG at any time
- Prior PCI any coronary artery within past year
- Need for other cardiac surgery
- Need for any other surgery within next year
- Inability to tolerate dual antiplatelet therapy for at least 1 year
- Noncardiac comorbidities with life expectancy of ≤ 3 years
- Other investigational studies
- Pregnancy

Box 3. Trial Endpoints
Primary Endpoints
- Composite all-cause mortality, MI, CVA at 3 years
Secondary Endpoints
- All-cause mortality, MI, CVA at 30 days
- Unplanned revascularization within 3 years
- Quality-of-life measures and treatment costs for the full follow-up period

RECOGNITION

Richard Van Praagh, MD (left), a cardiac pathologist at Boston Children’s Hospital—the pediatric teaching hospital of Harvard Medical School—was the seventh annual David J. Driscoll, MD, Visiting Lecturer at Mayo Clinic in Rochester. Dr. Driscoll is the former chair of the Division of Pediatric Cardiology at Mayo Clinic in Rochester.

Brian P. Shapiro, MD, received the 2012 Department of Medicine Outstanding Teaching Award at Mayo Clinic in Florida.

Joseph L. Blackshear, MD, received the 2012 Department of Medicine Outstanding Researcher Award at Mayo Clinic in Florida.

ANNOUNCEMENTS

New from Mayo Clinic
Pediatric Cardiology Board Review

Pediatric Cardiology Board Review

Cardiovascular Surgery
Mayo Clinic in Rochester, Minnesota
Joseph A. Dearani, MD, Chair
Harold M. Burkhart, MD
Thomas T. Carmody, MD*
Richard C. Daly, MD
Kevin L. Greason, MD
Lyle D. Joyce, MD, PhD
Soon J. Park, MD
Alberto Pochettino, MD
Hartzell V. Schaff, MD
John M. Stulak, MD
Rakesh M. Suri, MD, DPhil
Robert J. Wiechmann, MD*

ROBOTIC HEART SURGERY
Harold M. Burkhart, MD
Richard C. Daly, MD
Rakesh M. Suri, MD, DPhil

PEDIATRIC CARDIOVASCULAR SURGERY
Harold M. Burkhart, MD
Joseph A. Dearani, MD

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For additional information, visit www.mayo.edu/cme/cardiovascular-diseases, email our Education Department at cvcme@mayo.edu, or call us at 800-283-6296, 507-266-0677, or 507-266-6703.

Mayo Echocardiography Review Course for Boards and Recertification
May 4-7, 2013, Rochester, MN

NEW Basic to Advanced Echocardiography: From the Blue Ridge Mountains of Asheville
May 8-11, 2013, Asheville, NC

New Frontiers in Endovascular Therapy
May 17-18, 2013, Jacksonville, FL

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Cardiology Update 2013: The Heart of the Matter
Aug 2-4, 2013, Sedona, AZ

Success With Failure: Strategies for the Evaluation and Treatment of Heart Failure in the Clinical Practice
Aug 12-14, 2013, Incline Village, NV

Electrophysiology Review for Boards and Recertification
Transseptal and Epicardial Workshop
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Sep 8-11, 2013, Boston, MA

Internal Medicine Review for Nurse Practitioners, Physician Assistants and Primary Care Providers
Sep 18-20, 2013, Rochester, MN

18th Annual Mayo Cardiovascular Review Course for Cardiology Boards and Recertification
Pre-Course Echo Focus Session
Sep 20-26, 2013, Rochester, MN

10th Annual Mayo Clinic Interventional Cardiology Board Review
Sep 27-29, 2013, Rochester, MN

Challenges in Clinical Cardiology: An Annual Case-Based Update
Oct 4-6, 2013, Chicago, IL

29th Annual Echocardiography in Pediatric and Adult Congenital Heart Disease
Oct 13-16, 2013, Rochester, MN

Cases in Echocardiography, CT & MRI
Oct 23-26, 2013, Napa, CA

Coronary Artery Disease: Prevention, Detection and Treatment
Nov 2-4, 2013, Las Vegas, NV

Echo on Marco Island: Case-Based Approach
Dec 5-8, 2013, Marco Island, FL

2nd Annual Mayo Clinic Heart Rhythm Course: A Case-Based Approach
Dec 5-8, 2013, Scottsdale, AZ

National Meetings
American Society of Echocardiography
24th Annual Scientific Sessions

Symposium sponsored by Mayo Clinic
Echocardiography: Central Role in Decision-Making
Jun 28, 2013, Minneapolis, MN

American Heart Association Scientific Sessions 2013
Nov 16-20, 2013, Dallas, TX, www.heart.org

Symposia sponsored by Mayo Clinic
dates to be announced
Cardiac Rheumatology: Mechanisms and Management
An Integrated Approach to Evaluation and Management of Pulmonary Hypertension
Sports Cardiology Conference

International Meetings
Joint Mayo Clinic-Samsung Medical Center Cardiovascular Symposium
May 25, 2013, Seoul, South Korea
marriane.mun@samsung.com

16th Annual International Course on Cardiology and Cardiac Surgery
Aug 5-9, 2013, Vina del Mar, Chile
wright.scott@mayo.edu

British Cardiovascular Society: Cases, Controversies and Updates

Mayo Clinic International Vascular Symposium
Mar 27-29, 2014, Buenos Aires, Argentina
www.mayo.edu/cme/internationalvascular2014

Other Education Opportunities
Midwest Cardiovascular Forum: Controversies in Cardiovascular Disease
Jun 1-2, 2013, Minneapolis, MN
cme@umn.edu

Contact Us
Mayo Clinic welcomes inquiries and referrals, and a request to a specific physician is not required to refer a patient.

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800-533-1564

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