Leslie T. Cooper, MD, Has Been Named to Chair Department of Cardiology at Mayo Clinic in Florida

Leslie T. Cooper, MD, has been named chair of the Department of Cardiology at Mayo Clinic in Florida. Dr Cooper, director of the Gonda Vascular Center at Mayo Clinic in Rochester, Minnesota, since 2010, earned a medical degree from the University of Pennsylvania. He completed his residency at Stanford University, a fellowship in cardiology at the University of California, San Diego, and a fellowship in vascular medicine at Mayo Clinic in Rochester. He is board certified in internal medicine, cardiovascular diseases, vascular medicine, and heart failure and transplantation.

A professor of medicine at Mayo Clinic College of Medicine, Dr Cooper is on the faculty of Mayo Clinic’s Clinical Immunology and Immunotherapeutics Program and the Translational Immunovirology and Biodefense Program. Dr Cooper has been principal investigator for more than a dozen research studies and has authored more than 110 peer-reviewed articles in addition to a book and numerous book chapters. He has lectured around the world about inflammatory cardiac and vascular diseases, including myocarditis and vasculitis. He is the founder and president of the Myocarditis Foundation.

Heart Failure With Preserved Ejection Fraction—HFpEF: More Than Diastolic Dysfunction

Nearly half of all patients with heart failure have a normal ejection fraction (EF). The prevalence of this syndrome, termed heart failure with preserved ejection fraction (HFpEF), continues to increase in the developed world, likely because of the increasing prevalence of common risk factors, including older age, female sex, hypertension, metabolic syndrome, renal dysfunction, and obesity. Hypertension in particular is a strong risk factor; 80% to 90% of patients with HFpEF are hypertensive. Historically, HFpEF was termed diastolic heart failure; however, recent investigations suggest a more complex and heterogeneous pathophysiology (Figure on page 2). Ventricular diastolic and systolic reserve abnormalities, chronotropic incompetence, stiffening of ventricular tissue, atrial dysfunction, pulmonary hypertension, impaired vasodilation, and endothelial dysfunction are all implicated. Frequently, these abnormalities are noted only when the circulatory system is stressed.
Pathophysiology

At a cellular level, cardiac myocytes in patients with HFrEF are thicker and shorter than normal myocytes, and collagen content is increased. Recent histologic studies have shown reductions in myocardial capillary density that may contribute. At the organ level, affected individuals may have concentric remodeling with or without hypertrophy, although many people have normal ventricular geometry. Increases in myocyte stiffness are mediated in part by relative hypophosphorylation of the sarcomeric molecule titin, due to cyclic GMP (cGMP) deficiency thought to arise primarily as a consequence of increased nitrosative stress induced by comorbid conditions such as obesity, metabolic syndrome, and aging. Cellular and tissue characteristics may become more pronounced as the disease progresses.

“Most studies suggest that the rate of left ventricular (LV) pressure decay during isovolumic relaxation is prolonged, increasing LV and left atrial (LA) pressure, especially with elevated heart rates, as during exercise,” according to Barry A. Borlaug, MD, a cardiologist at Mayo Clinic in Rochester, Minnesota. Normal ventricular filling is achieved in large part by ventricular suction, the early active component of diastole, which is generated by intraventricular pressure gradients, mitral annular longitudinal motion, early diastolic LV “untwisting,” and elastic recoil induced by contraction to a smaller end systolic volume in the preceding contraction cycle. Each of these 4 elements is impaired in patients with HFrEF, especially with stress, so filling becomes dependent on high LA pressure to actively push blood into the left ventricle, as opposed to the action of a normal left ventricle, which “pulls” blood in during early diastole. Passive LV end-diastolic stiffness (Ees) is quantified by the slope and position of the diastolic pressure-volume relationship. Eed increases with normal aging, but this increase is exaggerated in individuals with HFrEF in most, but not all studies.

Although systolic function is relatively preserved, individuals with HFrEF typically exhibit subtle abnormalities in systolic performance, which become more dramatic during exercise. Limited stroke volume reserve and chronotropic incompetence markedly limit cardiac output in response to exercise. Mechanical dyssynchrony is common even though electrical dyssynchrony is not. Atrial fibrillation is extremely common in HFrEF (seen at some point in two-thirds of patients) and poorly tolerated because of the importance of LA contractile function in maintaining adequate LV chamber filling.

Pulmonary hypertension is common in patients with HFrEF. Increased LA pressure adds in series with increased resistive and pulsatile pulmonary arterial loading to increase RV afterload. This then leads to RV dysfunction, which seems to be tightly correlated with the development of atrial fibrillation. With RV failure, progressive systemic congestion occurs, manifested by malabsorption, congestive hepatopathy, cardiorenal syndrome, systemic inflammation, and cardiac cachexia. Increased RV and LA size and subsequent increases in total cardiac volume can lead to pericardial restraint, preventing additional preload recruitment during exercise or saline loading and contributing to elevation in filling pressures and cardiac output plateau.

Diagnosis

The most common complaints at presentation are exertional dyspnea and fatigue. Currently, 3 criteria must be met to establish the diagnosis: 1) clinical symptoms consistent with heart failure; 2) preserved EF (at least 50%); and 3) evidence of cardiac dysfunction. “Objective evidence of cardiac dysfunction is the most controversial point and a topic of intense investigation,” says Dr Borlaug. Potential findings that demonstrate cardiac dysfunction include evidence of congestion on physical examination or chest x-ray; atrial fibrillation; echocardiographic evidence of diastolic dysfunction (LA enlargement, engorged inferior vena cava, pulmonary hypertension, or elevated E/e’ filling velocity); and elevated brain natriuretic peptide (BNP).

If the diagnosis remains uncertain after testing, invasive hemodynamic evaluation may be revealing. Elevated filling pressures at rest support the diagnosis of HFrEF; however, many individuals demonstrate hemodynamic compromise only with stress. In these patients, it is critical to perform hemodynamic measurements during exercise.
Treatment

Clinical trials have not yet identified effective treatments for HFpEF (Table). Thus, recommendations are based on expert consensus opinion and focus on treatment of hypertension and maintaining appropriate intravascular volume. Aldosterone antagonists were not beneficial in a large multicenter trial overall, but a subgroup analysis of patients enrolled in the Americas showed some benefit on the basis of an elevated BNP level. Because HFpEF is associated with reduced cGMP availability, it was thought that treatment with phosphodiesterase-5 inhibitors, which increase cGMP levels via reduced metabolism, might be helpful. Unfortunately, the multicenter RELAX trial found no benefit for sildenafil compared with placebo in terms of exercise capacity, cardiac function, or clinical status.

Many treatment aspects have not yet been evaluated in a controlled fashion. Although atrial fibrillation is very common in patients with HFpEF, it is not known whether a rate or rhythm control strategy is preferential. It is also not known whether aggressive revascularization in those individuals with both coronary artery disease and HFpEF affects outcomes, although a recent single-center observational study showed improved survival in patients receiving complete revascularization. Some studies have also shown a beneficial response to statins in HFpEF, although this has not been evaluated in this

### Table. Comparison of Clinical Characteristics and Risk Factors in Recent Larger Clinical Trials

<table>
<thead>
<tr>
<th>Trial name</th>
<th>Drug</th>
<th>Age, y</th>
<th>Women,%</th>
<th>HTN,%</th>
<th>CAD, %</th>
<th>DM, %</th>
<th>Primary end-point</th>
<th>Overall result</th>
</tr>
</thead>
<tbody>
<tr>
<td>RELAX</td>
<td>Sildenafil</td>
<td>69</td>
<td>48</td>
<td>85</td>
<td>39</td>
<td>43</td>
<td>Peak $V_o_2$</td>
<td>Neutral</td>
</tr>
<tr>
<td>TOPCAD</td>
<td>Spironolactone</td>
<td>69</td>
<td>52</td>
<td>91</td>
<td>59</td>
<td>32</td>
<td>CV death, cardiac arrest, CHF hospitalization</td>
<td>Neutral</td>
</tr>
<tr>
<td>ALDO-DHF</td>
<td>Spironolactone</td>
<td>67</td>
<td>52</td>
<td>92</td>
<td>40</td>
<td>17</td>
<td>Peak $V_o_2$, diastolic function</td>
<td>Neutral</td>
</tr>
<tr>
<td>I-PRESERVE</td>
<td>Irbesartan</td>
<td>72</td>
<td>60</td>
<td>88</td>
<td>48</td>
<td>27</td>
<td>All-cause mortality, CV hospitalization</td>
<td>Neutral</td>
</tr>
<tr>
<td>CHARM-preserved</td>
<td>Candesartan</td>
<td>67</td>
<td>40</td>
<td>64</td>
<td>60</td>
<td>28</td>
<td>CV death, CHF hospitalization</td>
<td>Neutral</td>
</tr>
<tr>
<td>DIG-PEF</td>
<td>Digoxin</td>
<td>67</td>
<td>41</td>
<td>60</td>
<td>56</td>
<td>29</td>
<td>CHF mortality, CHF hospitalization</td>
<td>Neutral</td>
</tr>
<tr>
<td>PEP-CHF</td>
<td>Perindopril</td>
<td>75</td>
<td>55</td>
<td>79</td>
<td>27</td>
<td>21</td>
<td>All-cause mortality, CHF hospitalization</td>
<td>Neutral</td>
</tr>
<tr>
<td>SENIORS</td>
<td>Nebivolol</td>
<td>76</td>
<td>50</td>
<td>78</td>
<td>77</td>
<td>24</td>
<td>All-cause mortality, CV hospitalization</td>
<td>Neutral</td>
</tr>
</tbody>
</table>

Abbreviations: CAD, coronary artery disease; CHF, congestive heart failure; CV, cardiovascular; DM, diabetes mellitus; HTN, hypertension.

Adapted, with permission of the publisher, from Andersen and Borlaug, Curr Cardiol Rep. 2014 Jul;16(7):501.
Incomplete understanding of the pathophysiology of HFrEF, the likelihood that there is substantial pathophysiologic heterogeneity among affected patients, and the interplay of various risk factors have all been barriers in the development of effective treatments, underscoring the need for expanded research initiatives, given the rapidly increasing number of patients with this form of cardiac failure.

Clinical Trials in HFrEF at Mayo Clinic

**Inhaled Nitrite to Treat HFrEF:** This is a randomized, double-blind, placebo-controlled trial of acute inhaled sodium nitrite compared to placebo in people with HFrEF. This study is performed in the cath lab enrolling patients referred for invasive assessment to rule out HFrEF. Participants undergo the standard rest-exercise hemodynamic cath and then receive study drug, with repeat assessment of resting and low-level exercise hemodynamics. The idea is that, compared to placebo, nitrite therapy will reduce exercise filling pressures and improve cardiac output reserve.

- **Inclusion Criteria:** Scheduled for RH cath, clinical symptoms of dyspnea and fatigue, normal LVEF (>50%), and elevated LV filling pressures at cardiac catheterization (defined as resting PCWP>15 mm Hg and/or PCWP>25 mm Hg during exercise).

- **Exclusion Criteria:**

  - Systolic BP<120 mm Hg, prior nitrate therapy (within previous 2 weeks), G6PD deficiency, other “non-HFrEF” specific causes of heart failure such as significant valvular disease (>moderate left-sided regurgitation, >mild stenosis), severe pulmonary disease, unstable coronary disease or coronary spasm, primary renal or hepatic disease, constrictive pericarditis, or infiltrative, restrictive, or hypertrophic cardiomyopathies, pregnancy.

**RAPID-HF:** This is a randomized, double-blind crossover study testing rate adaptive atrial pacing in people with HFrEF and chronotropic incompetence. Everyone enrolled receives a pacemaker and then undergoes cardiopulmonary exercise testing after 1 month of device, on vs of. The order of device programming is randomized between the 2 crossover periods. The idea is that compared to device off, restoration of chronotropic reserve with pacing will improve exercise capacity in people with HFrEF.

**Inclusion Criteria**

1. Age >18 years and able to provide informed consent to enroll in the trial, or consent through a legal guardian or power of attorney.
2. Previous clinical diagnosis of HF with current NYHA Class II–III symptoms.
3. At least one of the following:
   - Hospitalization for decompensated HF
   - Acute treatment for HF with intravenous loop diuretic or hemofiltration
   - Chronic treatment with a loop diuretic for control of HF symptoms + left atrial enlargement on echocardiography
   - Resting PCWP >15 mm Hg or LV end-diastolic pressure >18 mm Hg at catheterization for dyspnea, and/or exercise PCWP/LV end-diastolic pressure >25 mm Hg
4. LVEF ≥50% within 12 months with clinical stability.
5. Stable cardiac medical therapy for ≥30 days.
7. Chronotropic incompetence on recent (within 6 months) clinical exercise test, defined as heart rate reserve (HRR) <0.80 or <0.62 if on β-blockers.
   - HRR = [observed peak HR – observed rest HR]/[predicted peak HR – observed rest HR]
   - Predicted peak HR will be calculated using the formula (220-age)
8. Meet both screening criteria on clinically performed CPX within 12 months:
   - Peak VO₂ ≤60% age/sex-adjusted normal value + peak respiratory exchange ratio (RER) ≥1.00
   - One of the following:
     - NT-proBNP ≥400 pg/mL
     - NT-proBNP <400 pg/mL, with rest PCWP >20 mm Hg and/or >25 mm Hg with exercise

**Exclusion Criteria**

1. Inability to exercise or noncardiac condition that precludes exercise testing.
2. Any contraindication to a pacemaker system.
3. Noncardiac condition limiting life expectancy to less than 1 year.
5. Hypertrophic cardiomyopathy.
6. Infiltrative or inflammatory myocardial disease (amyloid, sarcoid).
7. Pericardial disease.
10. Acute coronary syndrome or revascularization within 60 days.
11. Other clinically important causes of dyspnea.
13. PR interval >210 msec.
14. Resting heart rate >100 bpm.
15. A history of reduced EF (<50%).
16. Advanced chronic kidney disease (GFR <20 mL/min/1.73 m² by modified MDRD equation).
17. Women of child-bearing potential without negative pregnancy test and effective contraception.
18. Severe anemia (hemoglobin <10 g/dL).
19. Severe hepatic disease.
20. Complex congenital heart disease.
21. Listed for cardiac transplantation.
22. Other Class I indications for pacing.

For information about either of these clinical trials, please contact:
Barry A. Borlaug, MD, Principal Investigator
Kathlyn E. Cook, Study Coordinator, Cardiovascular Research Unit
Phone: 507-255-2200 E-mail: cook.katlyn@mayo.edu
RECOGNITION

The Mayo Clinic Hospital in Phoenix, Arizona, has again been recognized as a “top hospital” by the Leapfrog Group. This honor is given for achievement in standards of patient safety, quality of care, and efficiency. Qualifying organizations must meet criteria for patient outcomes, hospital resources, and management practices.

The Women in Cardiology Committee of the American Heart Association awarded Trainee Awards of Excellence to Carolyn Larsen, MD, Rachel Le, MD, Ana Kazkhodayan, MD, and Tanya Tajouri, MD, all cardiovascular trainees at Mayo Clinic in Rochester, at the 2014 Scientific Sessions. This highly prestigious award recognizes outstanding academic and clinical performance in women cardiology fellows during cardiovascular-related specialty training.

Samuel J. Asirvatham, MD, has received the Mayo Distinguished Educator Award for 2014. The Distinguished Educator Award recognizes Mayo faculty who have, over time, demonstrated excellence, leadership, and an unusually high level of commitment to the learner in their educational activities. Award recipients have been recognized for their ability to challenge and inspire their students and colleagues.

Andre Terzic, MD, PhD, has received the American Heart Association 2014 Basic Research Prize. Dr Terzic is the Michael S. and Mary Sue Shannon Director, Center for Regenerative Medicine, the Marriott Family Professor of Cardiovascular Diseases, and a Professor of Medicine and Pharmacology at Mayo Clinic in Rochester, Minnesota. The prize was awarded for pioneering applications of emerging technologies to advance the diagnosis and treatment of cardiovascular disorders by deciphering molecular pathways of disease susceptibility and repair and transforming these findings into individualized solutions for patients. Dr Terzic’s multidisciplinary program is focused on deciphering molecular pathways of disease susceptibility and the identification of the genetic basis for maladaptation in human cardiovascular disease, with the goal of identifying targets for personalized diagnostics and therapy. This program draws on expertise in population sciences, genomics and proteomics, and experimental and translational medicine. It exemplifies the integration of emerging technologies for the express and ultimate purpose of transforming health care solutions, notably, the ability to detect for each individual early in the patient’s course the defining basis of disease risk and to personalize treatment, even to the extent of targeting the repair of affected tissue.

The 2015 Gerald Gau Lecturer was Robert H. Eckel, MD (right), professor of medicine in the Division of Endocrinology, Metabolism and Diabetes, and Cardiology Professor of Physiology and Biophysics. He holds the Charles A. Boettcher Endowed Chair in Atherosclerosis and is the program director of the Adult General Clinical Research Center. His talk was entitled “Beyond the 2013 ACC/AHA Guideline on Lifestyle Management to Reduce CV Risk.”
New Book Details Development of Modern Cardiac Care

Caring for the Heart: Mayo Clinic and the Rise of Specialization

by W. Bruce Fye, MD (New York: Oxford University Press, 2015), describes major developments in the diagnosis and treatment of heart disease and explains how Mayo Clinic became a prominent medical center. The book shows how scientific advances and technological innovations—along with national and international societies—helped create contemporary heart care and stimulate subspecialization. Caring for the Heart was written for health care professionals, historians, and policy analysts as well as the general public. Dr Fye spent more than a dozen years researching and writing it. In the process, he reviewed more than 15,000 printed and unpublished documents and interviewed 63 individuals, including physicians, surgeons, scientists, nurses, and technicians. Dr Fye, now an emeritus cardiologist at Mayo Clinic in Rochester, Minnesota, is a past president of the American College of Cardiology and the American Association for the History of Medicine. We are pleased to present highlights from this fascinating book as we conclude the year-long celebration marking the 100th anniversary of the inauguration of formal cardiac services at Mayo Clinic.

The Electrocardiograph and the Birth of Cardiology (Chapter 5)

Franklin Lane had served under Presidents Theodore Roosevelt, William Howard Taft, and Woodrow Wilson. During 1920, the beloved 56-year-old Lane became progressively disabled by chest pain that occurred with activity and at rest. He also had recurring upper abdominal pain. After an East Coast doctor diagnosed gallbladder disease, Lane decided to go to Mayo Clinic. Writing from Rochester in December, he said, “I am being ground and wound and twisted and fed into and out of the Mayo mill, and a great mill it is.” An ECG led to a sudden change in the treatment strategy; it revealed abnormalities during an episode of chest pain that suggested that elective abdominal surgery was too risky. Dr Will Mayo would not operate on Lane’s abdomen. Another Mayo surgeon took out his tonsils. Took out his tonsils? The decision to perform a tonsillectomy was based on the focal infection theory, which was proposed in an era when doctors thought bacteria might cause some noncontagious diseases.

Challenges and Changes During the Depression (Chapter 6)

Mayo clinical investigators pioneered the use of 2 blood thinners: intravenous heparin in 1938 and an oral agent that would be termed dicumarol 3 years later. The Mayo researchers had a unique opportunity to compare heparin and dicumarol because both anticoagulants were available in Rochester before almost any other clinical investigators had access to either one. Commercialized versions of heparin and dicumarol (and a similar drug that would be named warfarin) would become mainstays of therapy to prevent acute and chronic thrombosis and embolism. The ability of these drugs to prevent blood from clotting made them indispensable for the development of technologies that revolutionized lifesaving treatments after World War II, such as kidney dialysis and open-heart surgery.

President Roosevelt’s Secret Hypertensive Heart Disease (Chapter 7)

When Howard Bruenn, the chief of cardiology at the Naval Medical Center in Bethesda, first examined President Franklin Roosevelt on March 24, 1944, he discovered that his blood pressure was high (186/108 mm Hg), and he had worrisome findings, including mitral regurgitation and congestive heart failure. An ECG demonstrated dramatic T-wave inversions; New York cardiologist Harold Pardee had written 3 years earlier, “Follow-up reports of the Mayo Clinic show that when associated with other abnormalities of the cardiovascular system, T wave abnormality added considerably to the gravity of the prognosis, such patients having a shorter average duration of life than those with normal T waves.” During the first week of April 1944, President Roosevelt’s 9 blood pressure readings averaged 210/114 mm Hg. At the time, documenting hypertension was relatively easy, but treating it was almost impossible. Gossip regarding the president’s health circulated at Bethesda Naval Hospital in Maryland and at Mayo Clinic, prompting the White House to request FBI director J. Edgar Hoover to investigate. The timing and pace of this investigation reflected the fact that the election was just days away. One of the FBI agents recalled that he had been sent to Rochester “in what proved to be an attempt to prevent the information on FDR’s circulatory disease from becoming public knowledge.” The media had made no mention of the casual conversations in Rochester and Bethesda, and press secretary Steve Early wanted to keep it that way. White House physician Ross McIntire misled reporters about the president’s medical evaluations and never informed the press or the public that Roosevelt had significant hypertension or that a cardiologist had diagnosed heart failure. In fact, he continued to deceive the public in 1946, a year after Roosevelt died of a stroke. McIntire claimed, “There are some conditions, of course, in which
we think we can predict it, such as extremely high blood pressure and advanced general arteriolosclerosis although there is no certainty. President Roosevelt did not have either of these. His blood pressure was not alarming at any time.”

**Pioneering Open-Heart Surgery at the University of Minnesota and Mayo Clinic (Chapter 10)**

On March 22, 1955, surgeon John Kirklin led a team that helped him perform Mayo’s first open-heart operation on a 5-year-old girl who had been born with a large ventricular septal defect. Two months later, Kirklin reported the results of the first 8 open-heart operations. The patients, who ranged in age from 4 months to 11 years, were severely symptomatic from congenital heart disease and had a very poor prognosis. Half of the children died. Despite the high mortality rate, Kirklin concluded that the new Mayo-Gibbon heart-lung machine functioned well and “established excellent conditions for precise, unhurried intracardiac surgery.” In that year, open-heart surgery was done on a regularly scheduled basis in just 2 institutions in the world: the University of Minnesota and Mayo Clinic. Heart surgery pioneer Harris Shumacker later claimed that “the 1955 report of John Kirklin and his colleagues of their first clinical experiences was most important in the history in the development of contemporary cardiac surgery.”

**Creating Coronary Care Units and Empowering Nurses (Chapter 13)**

Clark Gable finished filming *The Misfits* with Marilyn Monroe on November 4, 1960, four days before John Kennedy was elected president. But the 59-year-old actor did not have a chance to celebrate the movie’s completion or cast his vote. Gable was admitted to the hospital with a heart attack on November 6. Former President Dwight Eisenhower, who had spent a month in bed after an acute myocardial infarction 6 years earlier, wrote to the actor, “I learned from the paper this morning that you have suffered a mild coronary thrombosis. I trust that your recovery will be rapid and complete.” Gable’s recovery went well until the 10th day, when he died suddenly in his hospital room. The actor was not connected to a heart monitor, and no one was aware that he had a cardiac arrest. Physicians recognized the lifesaving potential of the technologies clustered in specialized coronary care units during the 1960s, and their widespread establishment contributed to a very significant shift in the traditional relationship between doctors and nurses. Support for granting specially trained nurses authority to defibrillate patients grew during the second half of the decade. The practice of nurses using defibrillators also gained popularity because it represented an alternative to having a doctor less than 5 minutes away from vulnerable patients at all times. There simply were not enough physicians to provide instantaneous round-the-clock coverage, especially at community hospitals without interns or residents.

**Challenges and Opportunities Around the New Millennium (Chapter 20)**

In 2009, Boston surgeon and best-selling author Atul Gawande published “The Cost Conundrum” in *The New Yorker* magazine. The academic surgeon observed, “Americans like to believe that, with most things, more is better. But research suggests that where medicine is concerned it may actually be worse. For example, Rochester, Minnesota, where the Mayo Clinic dominates the scene, has fantastically high levels of technological capability and quality, but its Medicare spending is in the lowest fifteen percent of the country.” Gawande had drawn some of his conclusions from a trip to Rochester as a visiting surgeon. Characterizing Mayo as one of the highest-quality, lowest-cost health care systems in America, he described some of the human interactions he observed. Gawande was impressed with “how much time the doctors spend with the patients. There was no churn—no shuttling patients in and out of rooms while the doctor bounces from one to the other.”
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