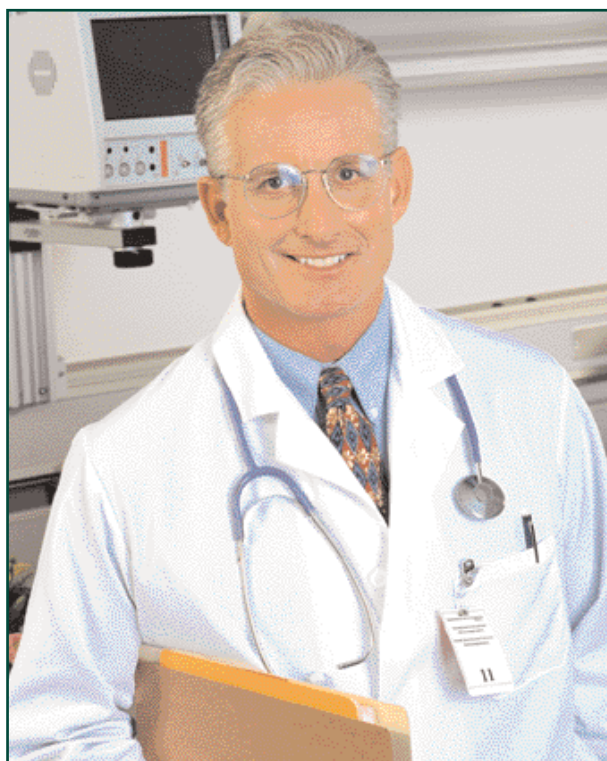


Special Report

The ACC 50th Annual Scientific Session

Part One

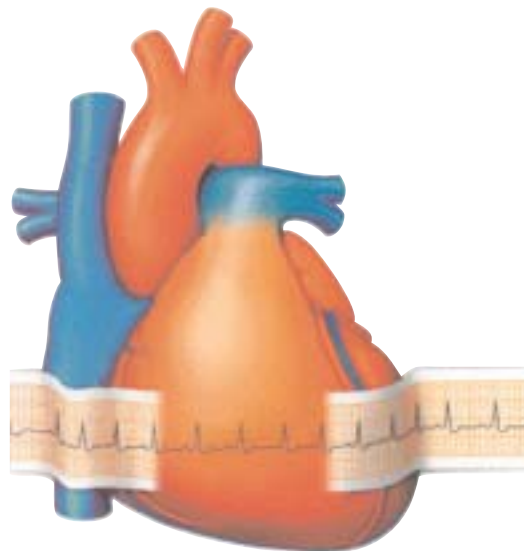


From March 18 to 21, 2001, physicians from around the world gathered to learn, to teach and to discuss at the American College of Cardiology 50th Annual Scientific Session.

Over the next two issues of *Perspectives in Cardiology*, we bring you summaries of four presentations we believe will be both interesting and useful for the Canadian family doctor. In this issue we feature:

- The Diagnosis of Diastolic Heart Failure; and
- Medication Doses Following PTCA.

The Diagnosis of **Diastolic** **Heart Failure**



D iastolic heart failure was one of hundreds of topics discussed at the American College of Cardiology's 50th Annual Scientific Session. The session on diastolic heart failure included presentations on epidemiology, pathophysiology, underlying mechanisms and treatment. This summary, however, will focus on the complex diagnosis of this syndrome.

The basic definition of heart failure is cardiac output insufficient to meet metabolic demands. Diastolic left ventricular (LV) dysfunction is the inability of the LV to generate end-diastolic volume in spite of normal or increased LV filling pressure. In spite of this simple definition, diagnosis can be complicated. The syndrome is incompletely characterized, definitions vary and the hemodynamics are complex. There are no signs or symptoms that reliably differentiate between systolic and diastolic dysfunction as a cause of heart failure.

The diagnosis of diastolic heart failure, however, is by no means impossible. In the presentation on which this summary is based, Dr. Dalane W. Kitzman of the Wake Forest University School of Medicine in Winston-

Salem, North Carolina, listed four steps to follow. First, establish the underlying syndrome of congestive heart failure. Then establish that LV function is normal, exclude other causes of the patient's signs and symptoms and, finally, evaluate diastolic dysfunction.

Establish the Underlying Syndrome

The diagnosis of heart failure should be done at the bedside based on signs and symptoms with which we are all familiar and supplemented by such laboratory tests as chest X-ray, creatinine and liver function tests and, in some cases, brain natriuretic peptide (BNP) rapid assay.

Currently, there are a number of published methods that set scoring criteria, based on the above components, to decide if heart failure exists. Most of these methods, however, have a systolic dysfunction bias. Some require a low ejection fraction (EF) for a diagnosis of heart failure, while most simply make the diagnosis of heart failure difficult to fulfill without a low ejection fraction.

Establish That LV Systolic Function is Normal

An echocardiogram is the most practical and easily available test to establish normal LV systolic function. This test will also provide a great deal of other information regarding segmental wall motion abnormalities, valvular function and pericardial function.

There is some question as to what EF cutoff should be used to determine what is normal and what is not. Some favor EFs as low as 35%, whereas Dr. Kitzman prefers to use an EF of 50% and term those with an EF between 35% and 50% as having mixed heart failure. Dr. Kitzman argues the higher the EF cutoff, the greater the specificity.

A significant subset of heart failure patients don't come close to this mildly abnormal group. Instead they have high normal, and even supernormal, EFS in the 70% and above range, thus excluding them from the subset of patients with systolic LV heart failure.

Exclude Other Possible Causes of Signs and Symptoms

Dyspnea and rales could be due to chronic obstructive pulmonary disease (COPD). Pedal edema could be caused by benign edema and fatigue could be caused by edema, hypothyroidism, extreme obesity or deconditioning. Aortic stenosis, mitral stenosis, regurgitation, constrictive pericarditis and ischemic heart disease all result in heart failure with a normal EF. The elderly often have multiple confounding conditions that can cause heart failure, however, aging alone does not cause dyspnea or exertional fatigue under everyday conditions.

Although the above conditions may result in the same signs and symptoms as diastolic heart failure, they must be differentiated as each condition has a specific treatment.

Evaluate Diastolic Function

Following the three steps given above will allow a diagnosis of isolated diastolic heart failure—a diagnosis largely of exclusion. An evaluation of diastolic function is the next logical step.

A direct measure of simultaneous diastolic pressure versus volume is definitive but impractical in most patients with clinical conditions. An elevated area diastolic pressure or pulmonary wedge pressure alone is strongly suggestive.

Doppler seems to be the most practical way to evaluate diastolic function, however, there are some drawbacks. Mitral flow patterns, for example, are helpful but are load dependent and change with circumstances. Early deceleration time is related directly to chamber stiffness, making it useful. In addition, it is of prognostic value in certain patient subsets. Unfortunately, there is a significant variability in measurement under clinical conditions.

Tissue Doppler, though a newer technique, looks very promising. It is relatively load independent, quick, simple and it is relatively unidirectional.

Conclusion

The diagnosis of diastolic heart failure can be difficult. It can be done, however, by ensuring heart failure is, indeed, the cause of the symptoms, by ensuring LV systolic function is normal and by eliminating any other possible causes for the signs and symptoms. Finally, evaluating diastolic function may help confirm the diagnosis, however, given more recent data, this may not be a mandatory criterion.

Medication Doses Following PTCA



There is a great deal of knowledge available about what drugs are being used to treat coronary artery disease (CAD), how effective these drugs are and whether the correct drugs are being used in clinical practice. Most of the research that has given us this information used drug dosages equal to, or very close to, the maximum recommended dose for a given drug. There is very little knowledge, however, about what medication dosages are being presented in the clinical setting.

Sacha R. Bhatia, a second-year medical student at McGill University, and Dr. Mark Eisenberg, assistant professor of medicine at McGill and staff cardiologist at Sir Mortimer B. Davis Jewish General Hospital in Montreal, examined this issue. They presented a poster at the American College of Cardiology's 50th Annual Scientific Session that examined medication use and dosage in patients who have undergone percutaneous transluminal coronary angioplasty (PTCA).

The investigators looked at 698 patients from 11 clinical centers in five countries, all of whom

were part of the Routine versus Selective Exercise Treadmill Testing after Angioplasty (ROSETTA) registry. All patients enrolled had undergone successful PTCA, defined as at least one stenosis equal to or greater than 60%, reduced to less than 50% with a minimum absolute reduction equal to or greater than 20%. Detailed medication data, including the drug name and dosage, were obtained at discharge.

The investigators focused on five different drug classes—acetylsalicylic acid (ASA) beta blockers, calcium channel blockers (CCBs), angiotensin converting enzyme (ACE) inhibitors and anti-lipid agents. The investigators then calculated what percentage of the maximum recommended dose (MRD) was prescribed in each case. They calculated this measure by dividing the dose given by the MRD, as found in the Compendium of Pharmaceuticals and Specialties (CPS), and multiplying it by 100.

Most of the patients involved in the study were elderly men with multiple risk factors for CAD. Thirty-five per cent of these

Table 1

Medication Doses Following PTCA

Drug Class	Percentage of Patients With Prescriptions	Median Dosage	Patients With Less Than 100% MRD
ALL PATIENTS			
ASA	92%	46% of MRD	64%
Beta Blockers	56%	25% of MRD	92%
CCBs	42%	—	86%
ACE Inhibs	32%	—	92%
Anti-Lipid Agents	49%	—	91% (75% on < 50% of MRD)
PREVIOUS MI			
ASA	—	—	73%
Beta Blockers	57%	—	94%
DIABETES MELLITUS			
ACE Inhibs	57%	—	87%
HYPERLIPIDEMIA			
Anti-Lipid Agents	76%	—	75%

patients had undergone a previous revascularization procedure.

The results indicated that the doses of medications shown to be efficacious in clinical trials are not being prescribed in the clinical setting (see Table 1). Instead, the doses being prescribed are significantly less than the MRD. Figure 1, which shows the use of ACE inhibitors at discharge after PTCA, provides an example of the prescribing patterns for one class of drug. The fact that patients are often not prescribed the MRD may mean that patients are not benefitting as much from their medications as clinical trials would indicate they should.

There were limitations to the study. There have been a number of trials since patients were recruited into the ROSETTA registry and the results, therefore, may not reflect the latest practice guidelines. In addition, the study was underpowered to correlate outcomes with medication dosages. *PCard*

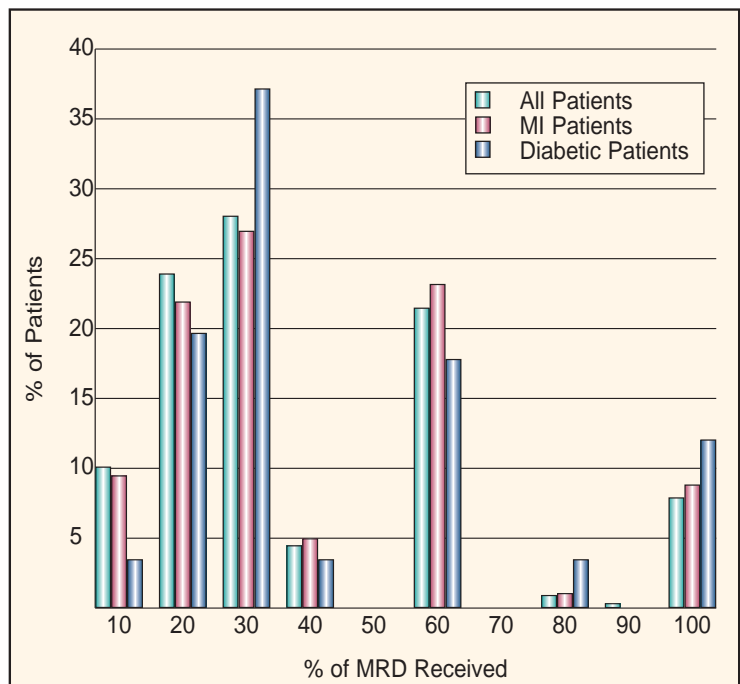


Figure 1. Use of ACE inhibitors at discharge after PTCA.