The extrapolation is based on Castro and colleagues’ observation of a single death among 114 patients (70% confidence limits 0.1%-3.0% mortality) in the concurrent setting of 543 patients deemed not in need of aortic root enlargement; the latter sustained a mortality of 4.1% with 70% confidence limits of 3.2% to 5.1%. These confidence limits and the context caution against concluding that widespread application of the proposed algorithm is likely to lower operative mortality.

Small sets of data pose two dangers, identifying spurious relations in small subgroups and failing to identify relations because of lack of statistical power. These concerns motivated collaborators in the multi-institutional study to contribute more than 13,000 patients with 70,000 patient years of follow-up.

Disclaimer

In responding to this letter, we must acknowledge that we reside within the institution with the highest volume of heart valve operations on the North American continent. The practice at The Cleveland Clinic Foundation has not been to avoid using small-sized prostheses in small aortic roots. Yet hospital mortality for 881 primary isolated aortic valve replacements in the last 5 years was 1.2%, and for 996 primary combined aortic valve replacements with coronary artery bypass grafting it was 2.0%. In some instances, left ventricular outflow tract myectomy is performed for obstruction at that level, but rarely is aortic root enlargement performed.

We remain interested in performance of prostheses, but we believe that available evidence suggests that other factors have more impact on long-term all-cause mortality than does prosthesis-patient size, however expressed.

Eugene H. Blackstone, MD
A. Marc Gillinov, MD
Delos M. Cosgrove, MD
Department of Thoracic and Cardiovascular Surgery
The Cleveland Clinic Foundation
Cleveland, OH 44195

References


Epidemiologic contrast of predictors’ trends for outcomes of coronary artery bypass grafting: Heart failure versus ventricular function versus left main disease

To the Editor:

I read with interest the article by Davierwala and colleagues in the November 2003 issue of the Journal. The work analyzed differential change in predictors of hospital mortality after coronary artery bypass grafting. The study elegantly demonstrated the diminishing statistical significance for left ventricular function greater than 20% as a predictor for mortality during a 12-year period. Several explanations are provided related to patient comorbidity, interventional cardiology, surgeon experience, and intensive patient care. It is well established that one of the major determinants of morbidity during and after coronary artery bypass grafting is low left ventricular ejection fraction. The results of numerous coronary artery bypass grafting trials performed in the 1970s and 1980s show that despite this increased morbidity, the benefits of this procedure for patients with multivessel coronary artery disease and low left ventricular systolic function in many cases outweigh the risks. The article does not contain data for the actual causes of death in this large group of patients, which would be epidemiologically relevant. Given the data provided, however, it is difficult to observe a contrast in the trends between congestive heart failure (CHF) and left ventricular function as predictor variables. Davierwala and colleagues also stated in their discussion that from studying the data again in Table 1 it is clear to the reader that CHF held an increasing proportion as morbidity in the patient cohort (7.8% vs 9.4% vs 9.4%) during the interval (1990-1993 vs 1994-1997 vs 1998-2001). Moreover, according to the original article’s Table 3, after a multivariate analysis CHF showed an increasing trend for odds ratio by year group (1.9 vs 3.6). This contrast of predictor outcome trends, the authors should have included other explanations. In simple terms, the CHF increasing trend could reflect an increase in diastolic heart failure, assuming that the proportions of new patients in each time interval were significant. In other words, the decline of the predictor value in left ventricular dysfunction was not related to interyear group patient death. The authors stated that left main disease in a “somewhat counterintuitive finding” was “unmasked” in the last time cohort, with an odds ratio of 1.7 in Table 3 of the original article. From their data, both CHF and left main disease increased in prevalence, yet worsening ventricular dysfunction declined. This constellation may reflect a change in the ventricular dysfunction function from systolic to diastolic biometrically.

Furthermore, the typical risk factors and comorbidities of female gender, diabetes, and hypertension together increased significantly during the entire period of their study. This association has now been found to be the same with diastolic dysfunction. As we know now, there is growing appreciation of diastolic heart failure as a distinct
entity. There are 4.6 million people in the United States with heart failure today, and 550,000 new cases are being reported annually by the American Heart Association. Approximately 30% to 50% of patients with heart failure have a normal or nearly normal left ventricular ejection fraction. CHF is a leading cause of cardiac morbidity and mortality from cardiovascular disease. Although left ventricular diastolic dysfunction occurs in all patients with systolic dysfunction and CHF, a third of patients have CHF with isolated diastolic dysfunction. In the general population, the mortality among patients with diastolic heart failure is 4 times that among persons without heart failure but half that among patients with systolic heart failure. It is widely known that many physicians underappreciate diastolic dysfunction in heart failure.

Reinterpreting the data in the context of the discussion asks for other explanations. In the present era of advancements in echocardiography, diastolic dysfunction needs to be taken into consideration, especially when symptoms of congestive heart failure are present. If that is the case in this study, left ventricular dysfunction remains a predictor of outcome but with a change from systolic to diastolic. Finally, I ask Davierwala and colleagues to take a second look at their data in the light of this forgotten discussion.

Dr Shuhaiber correctly points out that left ventricular (LV) diastolic dysfunction is frequently superimposed on either normal or reduced systolic function in patients undergoing coronary artery bypass grafting. The importance of diastolic dysfunction in the development and progression of congestive heart failure (CHF) has been illustrated previously and is highlighted by the divergent trends in the prevalence of LV systolic dysfunction and CHF in the series on which we reported. Early echocardiographic signs of impaired LV relaxation include decreased early transmitral LV filling and greater dependence on atrial contraction. Pseudonormalization of LV filling is observed as LV stiffness and left atrial pressures continue to increase, and finally severe diastolic dysfunction is marked by rapid early filling, extreme LV stiffness, and elevated diastolic pressures. This diastolic dysfunction has been shown to predict outcomes in patients with CHF independently of systolic function.

Despite this, echocardiographic characterization and quantitation of LV diastolic dysfunction is not performed and recorded for all patients undergoing coronary surgery. This may be due in part to logistical issues and in part to underappreciation of its prognostic significance. Like systolic function, diastolic function may also vary significantly with time in patients with intermittent ischemia or a maturing myocardial infarction.

Our institutional database, initiated in 1982 and now encompassing approximately 40,000 surgical patients, does not as yet contain uniform data on diastolic function, nor does the much larger Society of Thoracic Surgeons database. Thus, although one may reasonably speculate that the prevalence of diastolic dysfunction in patients undergoing coronary artery bypass grafting is increasing in parallel with other risk factors, as we have reported in this series, our data do not currently permit evaluation of the independent effect of diastolic function on mortality and morbidity. These measures of diastolic dysfunction will have to be obtained routinely for all patients undergoing surgery and incorporated into institutional and regional databases before we be able to determine whether the decreasing influence of LV systolic dysfunction on outcomes has unmasked relatively more subtle predictors, including CHF and left main stenosis as we have reported or diastolic dysfunction as Dr Shuhaiber suggests.

References

Reply to the Editor:

Dr Shuhaiber correctly points out that left ventricular (LV) diastolic dysfunction is frequently superimposed on either normal or reduced systolic function in patients undergoing coronary artery bypass grafting. The importance of diastolic dysfunction in the development and progression of congestive heart failure (CHF) has been illustrated previously and is highlighted by the divergent trends in the prevalence of LV systolic dysfunction and CHF in the series on which we reported. Early echocardiographic signs of impaired LV relaxation include decreased early transmitral LV filling and greater dependence on atrial contraction. Pseudonormalization of LV filling is observed as LV stiffness and left atrial pressures continue to increase, and finally severe diastolic dysfunction is marked by rapid early filling, extreme LV stiffness, and elevated diastolic pressures. This diastolic dysfunction has been shown to predict outcomes in patients with CHF independently of systolic function.

Despite this, echocardiographic characterization and quantitation of LV diastolic dysfunction is not performed and recorded for all patients undergoing coronary surgery. This may be due in part to logistical issues and in part to underappreciation of its prognostic significance. Like systolic function, diastolic function may also vary significantly with time in patients with intermittent ischemia or a maturing myocardial infarction.

Our institutional database, initiated in 1982 and now encompassing approximately 40,000 surgical patients, does not as yet contain uniform data on diastolic function, nor does the much larger Society of Thoracic Surgeons database. Thus, although one may reasonably speculate that the prevalence of diastolic dysfunction in patients undergoing coronary artery bypass grafting is increasing in parallel with other risk factors, as we have reported in this series, our data do not currently permit evaluation of the independent effect of diastolic function on mortality and morbidity. These measures of diastolic dysfunction will have to be obtained routinely for all patients undergoing surgery and incorporated into institutional and regional databases before we be able to determine whether the decreasing influence of LV systolic dysfunction on outcomes has unmasked relatively more subtle predictors, including CHF and left main stenosis as we have reported or diastolic dysfunction as Dr Shuhaiber suggests.

Piroze M. Davierwala, MD
Terrence M. Yau, MD, MSc
Division of Cardiovascular Surgery
Toronto General Hospital
Toronto, Ontario, Canada

Extracapsular lymph node involvement in esophageal cancer and number of involved nodes

To the Editor:

We thank Dr DeMeester for his valuable comments on our article “Extracapsular Lymph Node Involvement Is a Negative Prognostic Factor in T3 Adenocarcinoma of the Distal Esophagus and Gastroesophageal Junction.” We fully agree with Dr DeMeester that the number and characteristics of lymph nodes are very important determinants of survival in esophageal cancer, and we endorse his plea for taking the number of involved nodes into account in the TNM staging system of esophageal cancer.

In his editorial, Dr DeMeester made some points that we would like to clarify. With regard to the survival in patients without nodal involvement versus patients with intracapsular nodal involvement, there was no significant difference in survival.