The Relationship Between Cardiothoracic Ratio and Left Ventricular Ejection Fraction in Congestive Heart Failure

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Background: Left ventricular ejection fraction (EF) is a valuable prognostic index in patients with congestive heart failure (CHF). Although EF can be readily measured, many clinicians use roentgenographic heart size as a clue to differentiate systolic from diastolic dysfunction, even in the absence of solid supportive data.

Objective: To test the hypothesis that the cardiothoracic ratio (CTR) measured from the chest roentgenogram can be used to estimate left ventricular EF in individuals with CHF.

Methods: To answer this question, the database of the Digitalis Investigation Group trial was used. The CTR, determined using the Danzer method, and quantitative EF, measured locally using angiographic, radionuclide, or 2-dimensional echocardiographic techniques, were compared in 7476 patients with clinical CHF (New York Heart Association functional classes I-IV) due to acquired left-sided cardiac disease of ischemic, hypertensive, idiopathic, and alcohol-related causes.

Results: Mean (±SD) CTR for the cohort was 0.53±0.07. Mean (±SD) EF was 31.7%±12.2%. A weak, negative correlation between CTR and EF was observed (r=−0.176). Similar findings were obtained when the results were stratified by cause of CHF, presence of clinically defined right ventricular dysfunction, and method of EF measurement. Categorical analysis failed to yield a CTR cutoff point that facilitated useful segregation of individuals with an EF greater than 35% or 35% and below; greater than 40% or 40% and below; and greater than 45% or 45% and below in any patient group.

Conclusions: Although a weak, negative correlation exists between CTR and EF, this relationship does not allow for accurate determination of systolic function in individuals with CHF. Considering the morbidity and mortality associated with CHF, and the clinical implications of systolic function in this syndrome, direct measurement of EF is recommended.

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MEASURES OF left ventricular contractile function, including ejection fraction (EF), are relevant to diagnosis,1-4 prognosis,5-11 and treatment1-4,7,11 in patients with congestive heart failure (CHF). The EF is inversely related to mortality,1-11 and is perhaps the best prognostic index in heart failure. Thus, experts recommend that left ventricular systolic function be determined in patients with CHF.3,12,13 Although EF can be readily measured using angiographic, radionuclide, or echocardiographic techniques, these tests are expensive and may not be readily available in all clinical settings. A simple, convenient, noninvasive, and inexpensive method of accurately estimating left ventricular EF would be of great value in such patients.

Cardiac enlargement is associated with an adverse outcome in patients with heart disease.8,14,15 The size of the heart can be estimated from a chest roentgenogram by expressing it as a proportion of the thoracic diameter, the cardiothoracic ratio (CTR).16 Most cases of chronic CHF due to acquired left ventricular systolic dysfunction are associated with chamber dilation.17,18 In this context, one would expect an association between overall heart size, or CTR, and EF. However, one study demonstrated only a weak relationship between CTR and measured EF in patients with CHF.19 Furthermore, earlier work has shown a weak or inconsistent relationship between radiographic indexes and left ventricular size19-21 or function,20,22 although these studies may have been marred by small sample size and patient selection bias. Despite the absence of solid supportive data, clinicians continue to use roentgenographic heart size as a clue to differentiate systolic from diastolic dysfunction in patients with CHF.1,4

The Digitalis Investigation Group (DIG) Trial was a study of the effects of digoxin use on mortality and morbidity in
PATIENTS AND METHODS

PATIENTS

The design of the DIG Trial has been previously reported. In brief, patients with stable symptoms of CHF (New York Heart Association functional classes I-IV) who had normal sinus rhythm were enrolled in this study. Previous treatment with digitalis was allowed. Investigators were encouraged to prescribe angiotensin-converting enzyme inhibitors or other vasodilators prior to randomization. Exclusion criteria included age younger than 21 years and the presence of complex congenital heart disease, unstable or refractory angina, atrial fibrillation or flutter, cor pulmonale, constrictive pericarditis, acute myocarditis, hypertrophic cardiomyopathy, recent myocardial infarction, and uncorrected severe valvular heart disease. The underlying primary cause of CHF was determined locally by the DIG investigators as either ischemic, hypertensive, valvular, idiopathic, alcohol-related, or ‘other’ using common clinical definitions. Participants in the DIG study with valvular and other causes of CHF, representing only 3.6% of the cohort, were excluded from the current analysis. In addition, DIG participants with missing or potentially erroneous data (ie, CTR, <0.16 or >0.84; EF, <6% or >79%) were also excluded from the current analysis. For the purpose of this study, clinical right ventricular dysfunction was considered present when patients had elevated jugular venous pressure and/or peripheral edema within 1 month of enrollment in the DIG study. The DIG Trial was approved by the institutional review boards of all the participating centers and all patients signed informed consent forms.

MEASURING CTR AND LEFT VENTRICULAR EF

Prior to randomization in the DIG Trial, patients had a chest roentgenogram performed to measure CTR. Patients also underwent assessment of left ventricular EF. Both studies could be performed within 6 months of randomization. In cases in which clinical events occurred in the interim that might have affected the test results (such as myocardial infarction or revascularization), these studies were repeated prior to randomization and the most recent results recorded in the database. Vital patient data, including CTR and EF, were conveyed to the DIG Trial’s data coordinating center prior to randomization. When investigators reported a CTR greater than 0.70 or less than 0.40 or an EF greater than 70% or less than 6%, they were asked by the data coordinating center to confirm the validity of these data.

Posteroanterior chest roentgenograms were performed using standard radiologic techniques. The CTR was determined using the Danzer method (Figure). Left ventricular EF was also measured locally using either angiographic, radionuclide, or 2-dimensional echocardiographic techniques, with the choice being left to the investigators. However, quantitative measurements, not visual estimates, were required. For those patients undergoing echocardiographic measurement, the area-length method, modified Simpsons rule, or some other standard equation was specifically required.

DATA MANAGEMENT AND STATISTICAL ANALYSES

Data archival and statistical analyses were performed using SAS software. Student t test and analysis of variance methods were used to compare the results of continuous variables between groups. The Pearson product moment correlation coefficient was used to evaluate the relationship between CTR and EF. After the relationship between CTR and EF was examined as continuous variables, categorical analysis was performed to test the predictive value of CTR for EF in individual patients. Specifically, a series of CTR cutoff points from 0.45 and below to 0.62 and higher were examined for their ability to differentiate patients with EF greater than 33% or 35% and lower; greater than 40% or 40% and lower; and greater than 45% or 45% and lower.

In addition to statistical analyses for the entire study group, results were stratified by the method of EF measurement, the cause of CHF, and the presence of clinical right ventricular dysfunction. Because the anatomical pattern of cardiac chamber enlargement (cavity dimension and wall thickness) might differ between patients with and without hypertensive conditions, separate analyses were performed for all patients without hypertensive heart disease combined into the all nonhypertensive cohort. In this article, results are presented as mean (±SD) unless otherwise noted.

RESULTS

Between February 1991 and August 1993, a total of 7788 patients were enrolled in the DIG Trial at 302 centers in the United States and Canada. Of the total population, 254 participants were excluded from this analysis on the basis of the presence of valvular or other heart disease.
right ventricular dysfunction had a slightly higher CTR. The mean CTR was similar among the 3 EF method groups (angiography, 0.54±0.08; radionuclide, 0.53±0.07; and echocardiography, 0.54±0.08).

For the entire cohort, age was weakly related to CTR ($r=0.08$) and EF ($r=0.107$). For the entire sample, weight was not related to CTR ($r=-0.033$) or EF ($r=0.039$). Moreover, body mass index (calculated as the weight in kilograms divided by the square of the height in meters) was not related to CTR ($r=-0.002$) or EF ($r=0.021$). Similar findings were observed within each of the causal subgroups.

For the entire study group, a weak negative correlation between CTR and EF was observed ($r=-0.176$). Because of the large number of patients studied, this correlation coefficient was of high statistical significance ($P<.001$). A regression equation for the prediction of EF from CTR was derived:

$$EF = (-29.9 \times CTR) + 47.5$$

with an SE of the estimate of 12.0%. Similar results were observed when the data were stratified by clinical right ventricular dysfunction (Table 2 and Table 3), cause of CHF, and method of EF measurement (Table 3). Among those causal–right ventricular dysfunction–EF method subgroups with 100 or more patients, a correlation coefficient larger than $-0.35$ was observed in only 1: the group with idiopathic causes of CHF and right ventricular dysfunction who underwent radionuclide studies ($r=-0.388$).

With categorical analysis, increasing proportions of patients were shown to have low EF at incrementally higher CTR values, consistent with the negative correlation between CTR and EF. However, no CTR value was precise in the prediction of EF greater than 35% or 35% and below; greater than 40% or 40% and below; or greater than 45% or 45% and below in any patient group. For example, among the entire study population, an EF of 35% and below was present in 56% of patients with a CTR of 0.45 and lower, 59% of patients with a CTR lower than 0.50, 64% of patients with a CTR of 0.55 and lower, 65% of patients with a CTR lower than 0.60, and 75% of pa-

Table 1. Clinical Characteristics of Patients With Congestive Heart Failure*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>63.9±10.8</td>
</tr>
<tr>
<td>Sex, F</td>
<td>1805 (24.1)</td>
</tr>
<tr>
<td>Race, white</td>
<td>6410 (85.7)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>79.4±17.5</td>
</tr>
<tr>
<td>Height, cm</td>
<td>169.8±11.7</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>4835 (64.7)</td>
</tr>
<tr>
<td>Current angina</td>
<td>2056 (27.5)</td>
</tr>
<tr>
<td>Duration of congestive heart failure, mo</td>
<td>29.6±36.3</td>
</tr>
<tr>
<td>New York Heart Association classification</td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>1048 (14.0)</td>
</tr>
<tr>
<td>Class II</td>
<td>4089 (54.7)</td>
</tr>
<tr>
<td>Class III</td>
<td>2187 (29.3)</td>
</tr>
<tr>
<td>Class IV</td>
<td>147 (2.0)</td>
</tr>
<tr>
<td>Left ventricular ejection fraction, %</td>
<td>31.7±12.2</td>
</tr>
<tr>
<td>Cause of congestive heart failure</td>
<td></td>
</tr>
<tr>
<td>Ischemic</td>
<td>5343 (71.5)</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>793 (10.6)</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>1106 (14.8)</td>
</tr>
<tr>
<td>Alcohol-related</td>
<td>234 (3.1)</td>
</tr>
<tr>
<td>Medication use at enrollment</td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>3214 (43.0)</td>
</tr>
<tr>
<td>Potassium-sparing diuretics</td>
<td>568 (7.6)</td>
</tr>
<tr>
<td>Other diuretics</td>
<td>5819 (77.8)</td>
</tr>
<tr>
<td>Potassium supplements</td>
<td>2093 (28.0)</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitor</td>
<td>6999 (93.6)</td>
</tr>
<tr>
<td>Nitrates</td>
<td>3202 (42.8)</td>
</tr>
<tr>
<td>Hydralazine hydrochloride</td>
<td>152 (2.0)</td>
</tr>
<tr>
<td>Other vasodilators</td>
<td>90 (1.2)</td>
</tr>
</tbody>
</table>

*Categorical variables are shown as number of patients with percentage in parentheses. Continuous variables are shown as mean (±SD).

tients with a CTR of 0.60 and higher. Among patients with hypertensive disease, an EF of 45% and below was present in 51% of patients with a CTR of 0.45 and lower, 55% of patients with a CTR lower than 0.50, 65% of patients with a CTR of 0.55 and lower, 69% of patients with a CTR lower than 0.60, and 81% of patients with a CTR of 0.60 and higher. The sensitivity, specificity, and posi-


Our data demonstrate a weak negative correlation between CTR and left ventricular EF among a large and diverse population with chronic, stable heart failure. Although statistically significant due to the cohort’s large size, this correlation is not clinically useful because it does not allow for the accurate prediction of EF in individual patients or specific subgroups.

The cardiac silhouette on a chest roentgenogram encompasses all the contents of the pericardium. As shown in the Figure, the transverse dimension of the cardiac silhouette, which forms the numerator of the CTR, is predominantly affected by right atrium size, the internal dimension of the left ventricle, the thickness of the left ventricular wall, pericardial thickness, and the contents of the pericardial space. In the majority of patients with chronic left-sided CHF, left ventricular systolic function de-

**COMMENT**

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**Table 2. Relationship Between Cardiothoracic Ratio and Left Ventricular Ejection Fraction for All Patients With Congestive Heart Failure and for Subgroups Based on Cause and Presence or Absence of Clinical Right Ventricular Dysfunction (RVD)**

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>No. of Patients</th>
<th>Cardiothoracic Ratio*</th>
<th>Left Ventricular Ejection Fraction, %*</th>
<th>r†</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>7476</td>
<td>0.53±0.07</td>
<td>31.7±12.2</td>
<td>−0.176</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>2019</td>
<td>0.55±0.08</td>
<td>31.1±12.9</td>
<td>−0.182</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>5457</td>
<td>0.52±0.07</td>
<td>31.9±11.9</td>
<td>−0.170</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Ischemic cause</td>
<td>5343</td>
<td>0.52±0.07</td>
<td>31.5±11.3</td>
<td>−0.163</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>1336</td>
<td>0.54±0.07</td>
<td>30.7±11.9</td>
<td>−0.176</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>4007</td>
<td>0.52±0.06</td>
<td>31.7±11.1</td>
<td>−0.152</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypertensive cause</td>
<td>793</td>
<td>0.55±0.08</td>
<td>37.3±15.6</td>
<td>−0.235</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>309</td>
<td>0.56±0.09</td>
<td>37.3±15.5</td>
<td>−0.239</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>484</td>
<td>0.54±0.08</td>
<td>37.4±15.8</td>
<td>−0.235</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Idiopathic cause</td>
<td>1106</td>
<td>0.55±0.08</td>
<td>29.3±12.1</td>
<td>−0.230</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>304</td>
<td>0.56±0.06</td>
<td>29.0±12.7</td>
<td>−0.216</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>802</td>
<td>0.54±0.07</td>
<td>29.7±11.9</td>
<td>−0.229</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Alcohol-related cause</td>
<td>234</td>
<td>0.55±0.08</td>
<td>27.5±11.3</td>
<td>−0.191</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>With RVD</td>
<td>70</td>
<td>0.57±0.08</td>
<td>25.4±11.1</td>
<td>−0.033</td>
<td>.79</td>
</tr>
<tr>
<td>Without RVD</td>
<td>164</td>
<td>0.54±0.08</td>
<td>28.4±11.3</td>
<td>−0.237</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>All nonhypertensive causes</td>
<td>6683</td>
<td>0.53±0.07</td>
<td>31.0±11.5</td>
<td>−0.186</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>1710</td>
<td>0.55±0.08</td>
<td>30.0±12.1</td>
<td>−0.188</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>4973</td>
<td>0.52±0.07</td>
<td>31.3±11.3</td>
<td>−0.178</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Values reported are mean (±SD).
†r indicates Pearson correlation coefficient.

**Table 3. Relationship Between Cardiothoracic Ratio and Left Ventricular Ejection Fraction for All Patients With Congestive Heart Failure and for Subgroups Based on Cause, Presence of Clinical Right Ventricular Dysfunction (RVD), and Method of Ejection Fraction Measurement**

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>No. of Patients</th>
<th>Angiography</th>
<th>Radionuclide</th>
<th>Echocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Patients</td>
<td>r</td>
<td>P</td>
</tr>
<tr>
<td>All patients</td>
<td>410</td>
<td>4904</td>
<td>−0.174</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>122</td>
<td>1207</td>
<td>−0.173</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>288</td>
<td>3697</td>
<td>−0.172</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Ischemic cause</td>
<td>257</td>
<td>3695</td>
<td>−0.165</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>70</td>
<td>870</td>
<td>−0.162</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>187</td>
<td>2825</td>
<td>−0.163</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypertensive cause</td>
<td>43</td>
<td>429</td>
<td>−0.199</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>21</td>
<td>144</td>
<td>−0.132</td>
<td>.12</td>
</tr>
<tr>
<td>Without RVD</td>
<td>22</td>
<td>285</td>
<td>−0.234</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Idiopathic cause</td>
<td>99</td>
<td>632</td>
<td>−0.290</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>28</td>
<td>151</td>
<td>−0.388</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>71</td>
<td>481</td>
<td>−0.243</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Alcohol-related cause</td>
<td>11</td>
<td>148</td>
<td>−0.169</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>With RVD</td>
<td>3</td>
<td>42</td>
<td>−0.044</td>
<td>.78</td>
</tr>
<tr>
<td>Without RVD</td>
<td>8</td>
<td>106</td>
<td>−0.248</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>All nonhypertensive causes</td>
<td>367</td>
<td>4475</td>
<td>−0.194</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>With RVD</td>
<td>101</td>
<td>1063</td>
<td>−0.201</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Without RVD</td>
<td>266</td>
<td>3412</td>
<td>−0.186</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>
tions defined using echocardiography had cardiomegaly on
jects with asymptomatic left ventricular systolic dysfunc-
tion in primary mitral stenosis and primary mitral regurgi-
tation. Patients with clinical right ventricular dysfunction
were studied separately from those without because of the
potential differences between these groups in the pattern of
tion in primary mitral stenosis and primary mitral regurgi-
tation. Patients with clinical right ventricular dysfunction
were studied separately from those without because of the
potential differences between these groups in the pattern of
right atrial and right ventricular chamber size and wall thick-
ness. 5,6 Subgroup analysis based on modality of EF mea-
surement was performed to control for variation in the ran-
don error of measurement among these 3 techniques.

Although the CTR was designed to compensate for such factors as sex and body habitus, it
does not account for respiration and cardiac cycle–specific variation in heart position and chamber volume, and is sub-
ject to variability as a function of the heart-to-film distance.
Second, a time lag may occur between the onset of left ven-
tricular systolic dysfunction and an increase in cavity dimen-
ion, or CTR. However, a time lag alone cannot account for
the reduced sensitivity in our series, since the presence of
right ventricular failure, which implies relative chronicity
of CHF, 7 did not improve the CTR-EF relationship. While
EF is known to be dependent on cardiac loading conditions 7,8
and can be altered by pharmacologic therapy in patients with
CHF, the CTR may be less responsive to these factors. Fi-
nally, EF measurement itself is subject to random variation, 9
although the size of our study sample should have ade-
quately compensated for such variations in EF assessment.

A few limitations of this study warrant comment. First,
failure to use a single laboratory for determination of CTR
and EF may have induced significant variability in these
measurements. Second, we cannot state whether our con-
clusions apply to those patients with CHF secondary to other
causes, such as valvular or congenital heart disease, acute
myocarditis, or cor pulmonale. Third, the proportion of pa-
tients in the DIG Trial with an EF greater than 40% or 45%
may be smaller than that encountered in a typical primary
care practice. 1,4 Thus, the positive and negative predic-
tive values reported in our study may not apply to populations
with a different prevalence of diastolic heart failure. Fourth,
because the database did not specify those medications taken
at the actual time of CTR and EF measurement, we can-
not state the degree to which drug use interfered with the
CTR-EF relationship. Fifth, we cannot discount the pos-
cesses, filling pressure increases, and the chamber di-
lates. 17,18 Dilation and/or hypertrophy of other cardiac cham-
bers may also occur. Thus, patients with CHF generally have
a larger CTR than healthy subjects. 6 In turn, clinicians have
extrapolated that the chest roentgenogram can be used to
predict systolic function in patients with CHF. 1,4

In this study, care was taken to account for those clinical
variables that might interfere with the relationship be-
tween CTR and EF among patients with CHF. Patients were
stratified by CHF cause because the anatomical pattern of
chamber enlargement (cavity dimension and increased wall thickness) might differ among groups. For instance, an in-
creased CTR due to hypertrophy may be associated with low,
normal, or high EF in patients with CHF caused by hyper-
tensive heart disease. Patients with vaguely defined CHF
causes were excluded from this analysis. In addition, patients
with valvular heart disease, representing a very small por-
tion of the DIG Trial cohort, were excluded on the assump-
tion that they are a heterogeneous group with a varied and
complex relationship between CTR and EF. For example,
there are distinct differences between the classic patterns of
chamber enlargement and left ventricular contractile function
in primary mitral stenosis and primary mitral regurgi-
tation. Patients with clinical right ventricular dysfunction
were studied separately from those without because of the
potential differences between these groups in the pattern of
right atrial and right ventricular chamber size and wall thick-
ness. 5,6 Subgroup analysis based on modality of EF mea-
surement was performed to control for variation in the ran-
don error of measurement among these 3 techniques.

Table 4. Accuracy of Cardiothoracic Ratio Greater Than 0.55 for Prediction of Left Ventricular
Ejection Fraction of 35% or Lower in Patients With Congestive Heart Failure

<table>
<thead>
<tr>
<th>Patient Group*</th>
<th>No. of Patients</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>Positive Predictive Value, %</th>
<th>Negative Predictive Value, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>7476</td>
<td>36</td>
<td>74</td>
<td>74</td>
<td>36</td>
</tr>
<tr>
<td>Patients with RVD</td>
<td>2019</td>
<td>49</td>
<td>61</td>
<td>74</td>
<td>36</td>
</tr>
<tr>
<td>Patients without RVD</td>
<td>5457</td>
<td>31</td>
<td>78</td>
<td>74</td>
<td>36</td>
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<tr>
<td>Hypertensive cause</td>
<td>793</td>
<td>57</td>
<td>60</td>
<td>58</td>
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<tr>
<td>All nonhypertensive causes</td>
<td>6683</td>
<td>35</td>
<td>76</td>
<td>77</td>
<td>34</td>
</tr>
</tbody>
</table>

* RVD indicates clinical right ventricular dysfunction.

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sibility that CTR (or other roentgenographic findings) had important diagnostic or prognostic implications that were independent of their relationship to left ventricular EF. Finally, we cannot comment on the predictive value of other radiographic findings, such as pulmonary congestion, since these data were not recorded in the database.

Congestive heart failure is a common condition\(^1,3\) associated with a poor prognosis,\(^2,3,33\) Left ventricular systolic function has important implications for cause,\(^1,4\) prognosis,\(^3,11\) and treatment.\(^4,7,11\) In this disorder. For example, in diastolic heart failure, diuretics should be used with caution or avoided, although \(B\)-adrenergic and calcium channel-blocking agents may be effective.\(^1,3\) Evidence exists that some patients with CHF may receive suboptimal medical care.\(^3,34\) One aspect of this quality of care issue may be related to incorrect management decisions that result from inaccurate differentiation of systolic from diastolic dysfunction.\(^2\) Thus, our findings support the recommendations of expert panels\(^3,13\) that direct and quantitative methods should be used to distinguish systolic from diastolic heart failure.\(^3,36\) Considering the morbidity and mortality associated with CHF, estimating contractile function from the chest roentgenogram should be considered poor practice and be discouraged.

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