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The Role of Thoracic Ultrasonography for Evaluation of Patients With Decompensated Chronic Heart Failure

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OBJECTIVES This study examined the usefulness of thoracic ultrasonography for evaluation of fluid accumulation in patients with decompensated chronic heart failure (CHF) in comparison with physical signs, upright posteroanterior chest X-ray and echocardiography.

BACKGROUND Decompensated CHF is frequently accompanied by pleural effusion, suggesting that pleural effusion is a useful marker for confirming the diagnosis of the uncontrolled stage of CHF. Thoracic ultrasonography seems to be adequate for this purpose.

METHODS Patients with uncontrolled CHF and an interpretable physical examination, chest X-ray, ultrasonogram for the heart and thorax and thoracic X-ray computed tomographic (CT) scan were enrolled in the study (n = 60). Patients free from thoracic and cardiovascular diseases served as a control (n = 22). Thoracic CT scan was used as the gold standard for the presence or absence of pleural effusion. Variables used to predict body fluid accumulation included the following: pulmonary rales, jugular venous distension or peripheral edema, roentgenographic evidence of pulmonary edema or pleural fluid, pericardial or pleural effusion on ultrasonographic study.

RESULTS The reported incidence of pleural effusion detected by thoracic ultrasonography was high (91%). The incidence of physical signs and roentgenographic signs of body fluid accumulation, however, was modest (56%) to low (33%). The best clinical variable for identifying patients with decompensated CHF was the detection of pleural fluid by thoracic ultrasonography (91% predictive accuracy). This variable also had high interobserver agreement (95% overall agreement, kappa = 0.70). There was only 41% to 65% predictive accuracy of other clinical variables, with 72% to 95% agreement (kappa = 0.400–0.848).

CONCLUSIONS Thoracic ultrasonography is a simple, sensitive and accurate method for the evaluation of body fluid accumulation in patients with decompensated CHF. This technique can be used to assist in making the diagnosis of decompensated CHF if other causes of pleural effusion have been clinically ruled out. (J Am Coll Cardiol 2000;35:1638–46) © 2000 by the American College of Cardiology

Although there are several reports (1,2) that cardiac dysfunction can occur without florid signs of cardiac decompensation, congestion is generally considered to be a prerequisite for the diagnosis of decompensated chronic heart failure (CHF) (3,4). Optimal clinical management of CHF requires monitoring of the symptoms and signs of congestion in order to prevent exacerbation due to fluid accumulation (5). Our recent experience using X-ray thoracic computed tomography (CT) for the evaluation of heart failure patients has shown that many decompensated CHF patients presented with pleural effusions, suggesting that pleural effusion is a useful marker for confirming exacerbation in patients with established CHF.

Thoracic CT in the supine position is the method of choice for accurate diagnosis of the presence or absence of pleural effusion. As a rule, however, CT study cannot be emergently performed in orthopneic patients with uncontrolled CHF. In emergency situations in which CT study is not available or is not available quickly enough, ultrasonography might make a crucial contribution to the diagnosis of pleural effusion. Some previous studies (6,7) used thoracic ultrasonography for detection of pleural effusion in heart failure patients, but these reports did not define the accuracy of thoracic ultrasonography for evaluation of such patients. Furthermore, there is no comparative study on the evaluation of body fluid accumulation in uncontrolled CHF patients among thoracic ultrasonography and traditional clinical tests. Thus, this study was performed to ascertain the accuracy and usefulness of thoracic ultrasonography for evaluation of fluid accumulation in patients with decom-

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Abbreviations and Acronyms

CHF = chronic heart failure
CT = computed tomography
ECG = electrocardiogram
NYHA = New York Heart Association

pensated CHF in comparison with physical signs, upright posteroanterior chest X-ray and echocardiography.

METHODS

Study population. Between April 1997 and January 1999, 94 patients were consecutively admitted to our institution with a diagnosis of exacerbation of stable CHF or acute heart failure (8) requiring diuresis to improve their symptoms and signs of congestion and were initially considered for recruitment to this study. The patients had dyspnea upon mild to moderate exertion or at rest (New York Heart Association [NYHA] class II to IV) with objective evidence supporting the diagnosis of decompensated CHF: documented heart disease, cardiogenic pulmonary or peripheral edema of cardiac origin. Patients with acute coronary syndrome (n = 9), pericardial disease (n = 3) or acute valvular regurgitation were not included in this study. Patients with complicating inflammatory pulmonary or pleural disease (n = 6), renal failure (n = 4), hepatic cirrhosis (n = 2) or pleural exudates of unknown cause (n = 3) were also excluded from the study. Of the 67 remaining patients fulfilling the selection criteria, plain chest radiographs or ultrasonographic images were of low quality in four patients, and thoracic CT study could not be performed in three other patients. Thus, 60 patients with exacerbation of established CHF and an interpretable physical examination, upright posteroanterior chest X-ray, ultrasonogram for the heart and thorax and thoracic CT results were enrolled. In addition to the study group, a total of 22 patients (13 men and 9 women; mean age of 67.8 ± 22.3 years) with suspected pulmonary or thoracic aortic diseases, no cardiovascular disease and who underwent thoracic CT study and were ultimately proven to be free from significant CT findings served as a control to evaluate the diagnostic accuracy of the traditional clinical tests and thoracic ultrasonography for detection of body fluid accumulation. None of the study patients had any discernible renal impairment, with the serum creatinine values of all subjects being below 2 mg/dl. Plasma thyroidal hormone was measured in patients with pericardial effusion or atrial fibrillation to confirm a euthyroidal state.

Methodology. Detailed data were collected prospectively at the time of the diagnosis in study patients meeting criteria for heart failure. Each patient provided written informed consent before participating in the study. Data obtained immediately after admission included a thorough cardiovascular history and physical findings, blood chemistry, electrocardiogram (ECG), upright posteroanterior chest X-ray and ultrasonographic examination for the heart and thorax. To confirm myocardial failure, a serum assay for atrial (normal range: ≤43 pg/ml) and brain natriuretic peptide (≤18.4 pg/ml) was performed (9,10). After completion of admission data collection, all subjects underwent chest X-ray CT study as early as possible (41 [68%] within 2 h, 12 [20%] within 12 h and 7 [12%] within 24 h after admission). Diagnostic thoracentesis and pericardiocentesis were performed appropriately to confirm the cause of effusion (11). Patients were initially treated with some combination of oxygen, digoxin, nitrate, diuretics, sympathomimetic agents and synthetic human alpha atrial natriuretic peptide. Clinical, serum natriuretic peptides, ECG, plain chest X-ray and ultrasonographic examinations were repeated during a follow-up period.

Bedside physical examinations. Pulmonary crackles or rales were recorded as present or absent. Jugular venous distension was deemed present if venous pulsations were visible at 45° from the horizontal plane (12). The presence or absence of peripheral edema and of a third heart sound was also noted.

Chest radiography. All radiographs were evaluated for the presence or absence of cardiomegaly (cardiothoracic ratio more than 0.5), venous redistribution (upper lobe vessels more prominent than lower), interstitial edema (hilar haziness, peribronchial cuffing, Kerley B lines) or alveolar pulmonary edema and pleural effusion (13–15).

Cardiac ultrasonography. Echocardiography was performed using a commercially available real-time wide-angle phased-array system incorporated with a color-Doppler system (Aloka SSD-2000, Aloka Co. Ltd., Tokyo, Japan). Standard imaging views were obtained with the patient lying in the left lateral decubitus position. Full two-dimensional, M mode, color flow and Doppler studies were performed on each patient. Pericardial effusion was defined to be present when an echo-free space was clearly visualized between the epicardium and pericardium and was associated with flattening of the pericardial echo relative to the epicardial echo (16). An anterior echo-free space in the absence of a significant posterior echo-free space was not considered indicative of pericardial effusion (16).

Thoracic ultrasonography. After completion of the echocardiography, the patient was asked to assume a sitting position. The thoracic ultrasonographic study (6,7,17–21) consisted of echographic investigation of each hemithorax using a 3.5 MHz sector transducer through the intercostal space avoiding the ribs. The liver and spleen, and occasionally the kidney, were used as guides to thoracic ultrasonography for the detection of pleural fluid. The ribs and the interface of lung and pleura are readily identified, with the latter reflecting most of the acoustic energy and appearing as a bright white line often associated with distal reverberation

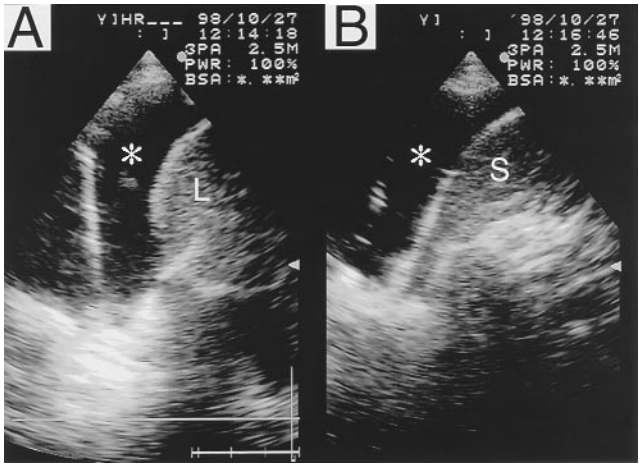

Figure 1. Representative example of the thoracic ultrasonograms of a decompensated heart failure patient with bilateral pleural effusion. Accumulation of pleural effusion (*) is noted adjacent to the liver **(L)** in the right hemithorax **(panel A)** and adjacent to the spleen **(S)** in the left hemithorax **(panel B)**.

echoes. Identification of a smaller amount of the pleural fluid in heart failure patients is ordinarily accomplished by applying the transducer on the posterolateral thoracic wall where the most dependent portion of the costophrenic sulcus in the pleural space can be detected in the upright position. As the pleural effusion accumulates, it gradually extends from this portion to the lateral and eventually reaches the anterior costophrenic sulcus. Shallow respiration enhances the ability of ultrasonography to detect fluid collection, which appears as an echo free space between the lung and the adjacent abdominal organ (i.e., the liver in the right hemithorax and the spleen in the left hemithorax). Figure 1 shows a representative example of thoracic ultrasonographic findings in a CHF patient with bilateral pleural effusion.

X-ray thoracic CT. For the evaluation of pleural effusion, a whole body CT scanner (Toshiba Medical Co., Tokyo, Japan; CT scanner X speed), which utilized a continuously rotating gantry and pulsed anode with X-radiation collimated to form a thin fan-shaped beam, was used. At least two separate windows of varying contrast were viewed at each level, including the areas of water density, for the analysis of pleural effusion. The volume of thoracic effusion was calculated by measuring areas of pleural effusion on a tomogram and totaling the areas in each tomogram obtained by slicing the thorax to a thickness of 1 cm (22). The mean difference in interobserver agreement of right or left pleural effusion by two different reviewers was 51.1 ± 38 ml ($r = 0.97$; standard error of estimate 23.5 ml; $p < 0.0001$; $n = 20$ comparisons) and 42.6 ± 42.7 ml ($r = 0.97$; SEE 16.9 ml; $p < 0.0001$; $n = 20$), respectively.

Study variables. *Variables used to predict body fluid accumulation included the following:* presence of pulmonary crackles, rales or wheezing, jugular venous distension or peripheral edema on bedside physical examination, roentgenographic evidence of pulmonary interstitial and alveolar edema or pleural fluid and presence of pericardial effusion or pleural effusion on ultrasonographic study for the heart and thorax. Two observers independently evaluated each patient for the presence or absence of these study variables. Consensus agreements were reached when there was disagreement between two observers.

Table 1. Demographic Data and NYHA Functional Classification (n = 60)

Age (yr)	
<65	10 (17%)
65–85	36 (59%)
>85	14 (24%)
Gender	
Male	23 (38%)
Female	37 (62%)
NYHA functional	
Class II	18 (30%)
Class III	18 (30%)
Class IV	24 (40%)
Blood chemistry	
Hemoglobin (g/dl)	12.2 ± 1.6
Range	9.8–16.7
Albumin (g/dl)	3.9 ± 0.42
Range	2.7–4.8
Creatinine (mg/dl)	1.09 ± 0.33
Range	0.5–1.9
Atrial natriuretic peptide (pg/ml)	172.8 ± 22.0
Range	15–862
Brain natriuretic peptide (pg/ml)	691.5 ± 638.0
Range	83–3,000

NYHA = New York Heart Association.

Statistical analysis. Continuous variables were reported as mean ± SD and compared using Student *t* test. Categorical variables were expressed as the percentage and compared using the chi-square test or McNemar's test, appropriately. The agreement between two clinicians was assessed by calculating the Kappa value; the strength of agreement is "poor" when the value of kappa is less than zero, "slight" from 0 to 0.20, "fair" from 0.21 to 0.40, "moderate" from 0.41 to 0.60, "substantial" from 0.61 to 0.80 and "almost perfect" from 0.81 to 1.00 (23). Sensitivity was defined as 1 − number of false negatives/number of true negatives; specificity was defined as 1 − number of false positives/number of true negatives; and predictive accuracy was defined as number of correct tests/number of patients tested (24). A p value of less than 0.05 was considered to be statistically significant.

RESULTS

Table 1 summarizes the demographic data and NYHA functional classification. Table 2 lists various causes of decompensated CHF in the 60 patients (mean age of

Table 2. Etiology of CHF

Etiology	Number of Patients
Cardiac valve disease	
Mitral regurgitation	9
Aortic regurgitation	5
Aortic stenosis	1
Tricuspid regurgitation	2
Combined regurgitant valve disease	7
Mitral stenosis	2
Arterial hypertension	12
Chronic ischemic heart disease	9
Hypertrophic cardiomyopathy	4
Idiopathic dilated cardiomyopathy	1
Rhythm disturbance	
Atrial fibrillation	4
Atrioventricular block	1
Combined*	3
Total	60

*Combined: more than one etiology primarily involved.

76.0 ± 12.9 years). Forty percent of the patients were in NYHA class IV. Serum brain natriuretic peptide on admission was elevated in all, and atrial natriuretic peptide was elevated in 49 patients (82%). Cardiac valve disease was the most frequent etiology (43%) followed by arterial hypertension (20%) and chronic ischemic heart disease (15%). Atrial fibrillation was present in 25 patients, the incidence being high among the patients with valvular heart disease (14/26, 54%).

Detection of pleural effusion by thoracic ultrasonography versus plain chest radiograph. Table 3 summarizes the comparison of the presence of signs of pleural effusion between plain chest radiograph and thoracic ultrasonography related to changes in the volume of pleural effusion in 22 normal controls and 60 heart failure patients. On X-ray thoracic CT, 52 of 60 heart failure patients (86.6%) and none of the control subjects demonstrated pleural effusions.

Table 4. The Frequency of the Detection of Body Fluid Accumulation in 60 Patients With Decompensated CHF by Two Clinicians

	A (%)	B (%)	Combined (%)
Physical findings			
Rales or wheezing	35 (58)	32 (53)	33.5 (56)
Jugular venous distension	25 (42)	20 (33)	22.5 (38)
Peripheral edema	20 (33)	20 (33)	20 (33)
Any	35 (58)	39 (65)	37 (62)
Chest roentgenogram			
Pulmonary edema	28 (47)	26 (43)	27 (45)
Pleural effusion	23 (38)	26 (43)	24.5 (41)
Any	34 (57)	39 (65)	36.5 (61)
Ultrasonography			
Pericardial effusion	13 (22)	12 (20)	12.5 (21)
Pleural effusion	55 (92)	54 (90)	54.5 (91)
Any	55 (92)	54 (90)	54.5 (91)

In the 22 control subjects, one patient showed signs of pleural effusion on the left thoracic ultrasonographic examination. In the 60 patients with decompensated CHF, thoracic ultrasonography was highly accurate for identifying the pleural effusion when compared with plain chest radiograph; even when there was a very small amount (≤100 ml) of pleural effusion, it was detected by thoracic ultrasonography in as many as 80% (left hemithorax) to 87% (right hemithorax) of the patients. In contrast, plain chest X-ray examination detected only 13% of the cases in which there was a very small amount of pleural effusion in the right hemithorax. In total, thoracic ultrasonography identified significantly more pleural effusion in each hemithorax than did plain chest radiograph: 92% in the right hemithorax and 93% in the left hemithorax and only 48% ($p < 0.001$) and 26% ($p < 0.001$) in the plain chest radiograph, respectively.

Incidence of clinical signs of body fluid accumulation. Table 4 shows the frequency of clinical signs of body fluid

Table 3. Comparison of the Presence of Signs of Pleural Effusion Between Plain Chest X-ray and Thoracic Ultrasonogram in Normal Controls and Patients With Decompensated CHF

	Right Hemithorax			Left Hemithorax		
Pleural Effusion by CT	n	X-ray	Echo	n	X-ray	Echo
Normal controls (n = 22)						
Absent	22	0	0	22	0	1 (5)
Patient group (n = 60)						
Absent	10	0	1 (10)	14	0	2 (14)
Present (ml)						
Very small (≤100)	15	2 (13)	13 (87)	15	0	12 (80)
Small (≤400)	20	9 (45)	18 (90)	23	6 (26)	23 (100)
Moderate (≤700)	8	6 (75)	8 (100)	6	4 (67)	6 (100)
Large	7	7 (100)	7 (100)	2	2 (100)	2 (100)
Total	50	24 (48)	46 (92)	46	12 (26)	43 (93)

Numbers in parentheses are percentages.
Echo = thoracic ultrasonography.

Table 5. Observer Disagreement on Clinical Tests for Identifying Body Fluid Accumulation in 60 Patients With Decompensated CHF

Observations of Observer A	Number of Observations by Observer B		Agreement (%)	Kappa Coefficients
	Present	Absent		
Physical findings				
Rales or wheezing				
Present	29	3		
Absent	6	22	85	0.697
Jugular venous distension				
Present	14	6		
Absent	11	29	72	0.400
Peripheral edema				
Present	17	5		
Absent	6	32	81	0.609
Chest roentgenogram				
Pulmonary edema				
Present	19	7		
Absent	9	25	73	0.462
Pleural effusion				
Present	21	5		
Absent	2	32	88	0.759
Ultrasonography				
Pericardial effusion				
Present	11	1		
Absent	2	46	95	0.848
Pleural effusion				
Present	53	1		
Absent	2	4	95	0.700

accumulation in 60 patients with decompensated CHF by two clinicians. Reported incidence was excellently high in the variable of the pleural effusion detected by thoracic ultrasonography (90% to 92%). The incidence of rales or wheezing was modest (53% to 58%) but that of other physical signs of jugular venous distension (33% to 42%) and peripheral edema (33%) was low. On chest radiograph, the frequency of the presence of pulmonary edema (43% to 47%) or pleural effusion (38% to 43%) was low, but the presence of two or more variables moderately predicted body fluid accumulation (57% to 65%).

Interobserver agreement on the presence or absence of clinical signs of body fluid accumulation. Table 5 summarizes the interobserver agreement on the reported clinical tests for identifying body fluid accumulation in patients with decompensated CHF, which varied widely and ranged from 72% to 95%. Among study variables, there was substantial to almost perfect agreement between the two observers on the results of the thoracic ultrasonography (95% overall agreement, kappa = 0.70), echocardiography (95% overall agreement, kappa = 0.848) and on the presence or absence of pleural effusion on plain chest radiograph (88% concordance, kappa = 0.759). There was also substantial agreement between the two observers for pulmonary rales (85% overall agreement, kappa = 0.697). There was only 72% agreement on interpretations for the presence or absence of jugular venous distension (kappa = 0.40), 81% agreement on recognizing peripheral edema (kappa = 0.609) and 73% concordance in identifying pulmonary interstitial edema on chest radiograph (kappa = 0.462).

Comparative diagnostic value of study variables. Table 6 shows the comparative data of sensitivity, specificity and predictive accuracy of study variables for the diagnosis of body fluid accumulation. The best clinical variable for identifying patients with decompensated CHF was the detection of pleural fluid by thoracic ultrasonography (91% predictive accuracy). There was only 41% to 65% predictive accuracy of the other clinical variables examined.

Follow-up data. After a follow-up period of 16.2 ± 4.5 days (range 10–30 days), 54 patients with decompensated CHF on admission showed definite clinical improvement after effective diuresis, as documented by clinical examinations in addition to a net negative fluid balance or significant weight loss and serum assay of natriuretic peptides; the serum level of atrial and brain natriuretic peptides decreased to 54.0 ± 38.9 pg/ml and 204.6 ± 215.5 pg/ml, respectively. Both were significantly different from admission

Table 6. Comparative Diagnostic Value of Traditional Clinical Signs and Ultrasonographic Test for Identifying Body Fluid Accumulation in Decompensated CHF

	Sensitivity (%)	Specificity (%)	Predictive Accuracy (%)
Physical findings			
Rales or wheezing	33/60 (55)	20/22 (91)	53/82 (65)
Jugular venous distension	24/60 (40)	19/22 (86)	43/82 (52)
Peripheral edema	22/60 (37)	21/22 (95)	43/82 (52)
Chest roentgenogram			
Pulmonary edema	27/60 (45)	20/22 (91)	47/82 (57)
Pleural effusion	26/60 (43)	22/22 (100)	48/82 (59)
Ultrasonography			
Pericardial effusion	12/60 (20)	22/22 (100)	34/82 (41)
Pleural effusion	54/60 (90)	21/22 (95)	75/82 (91)

levels ($p < 0.0001$ in each). In the remaining six patients with decompensated CHF, the clinical course deteriorated during the follow-up period; four patients died from advanced heart failure, one patient refractory to heart failure treatment, and another case was complicated by a serious cerebrovascular accident.

DISCUSSION

Because heart failure is a clinical syndrome that is caused by abnormal cardiac function, the major goals in the evaluation of patients with heart failure are:

1) to identify the nature and severity of the cardiac abnormality,
2) to characterize the nature and severity of the patient's functional limitation, and
3) to assess the presence and severity of fluid retention (5).

With regard to assessment of the presence and severity of fluid retention, results of this study indicate that thoracic ultrasonography is a simple, sensitive and accurate method for evaluation of patients with decompensated CHF. When compared with traditional physical and chest radiographic examinations, this method proves to be very useful for identifying the signs of body fluid accumulation.

Body fluid retention in CHF. Chronic heart failure resulting from diminished cardiac function is invariably caused by an abnormality of the muscle, rhythm, valves or pericardium. The syndrome of CHF is the response of the body to the heart's inability to maintain an adequate blood supply at a rate commensurate with the requirements of the metabolizing tissues. The primary problem in CHF is cardiac, but the clinical syndrome is characterized by secondary multisystem dysfunction, which ultimately leads to a terminal state of multiorgan failure (25). The reduction in cardiac output that occurs in the failing heart activates the sympathetic nervous system (26,27), resulting in an increased heart rate and vasoconstriction. A decrease in renal perfusion results in activation of the renin-angiotensin-aldosterone system, leading to production of the powerful vasoconstrictor angiotensin II and, ultimately, sodium retention through the actions of aldosterone (28,29). Water retention is also augmented by vasopressin production from the posterior pituitary (30). Other potent vasoconstrictors, such as endothelin, contribute to an increase in peripheral vascular resistance (31,32). These changes might not be sufficiently counteracted by the cardiac endocrine system, which produces natriuretic and vasodilating atrial and brain natriuretic peptides (9,10,33,34). As a result, this sequence of the spiraling course of heart failure induces more and more retention of sodium and water and is ultimately punctuated by acute exacerbations (so-called decompensated CHF) that require hospital admission.

Symptoms of heart failure. The classical symptoms of heart failure are dyspnea, ankle edema and fatigue. Dyspnea on exertion is common in the general population, particularly in patients with respiratory disease or in the obese, and, therefore, it cannot be used as the selection criterion for the diagnosis of heart failure (25). Orthopnea and paroxysmal nocturnal dyspnea are less common in the general population than dyspnea alone, but less sensitive, for the diagnosis of heart failure (2,25,35–38). Additionally, there are many asymptomatic or minimally symptomatic patients who are existing even in the uncontrolled stage of CHF, particularly among aged patients in whom the prevalence increases sharply, affecting perhaps as many as 5% to 10% of individuals over the age of 65 (39,40). In this study, the incidence of mild subjective symptoms of heart failure (NYHA class II) tended to be higher in patients ≥75 years (15 of 40 patients, 38%) than in younger patients (3 of 20, 15%; $p < 0.1$). The most probable reasons for the lack of subjective heart failure symptoms in the aged are their low level of daily activity or high rate of complicating cerebrovascular disease, which should obscure complaint of the dyspneic sensation.

Classical clinical signs of body fluid accumulation in CHF. An accurate objective measurement of the presence of body fluid retention is necessary in order to evaluate patients with decompensated CHF. The difficulty in clini-

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cally defining such patients, however, stems from the fact that no simpler or more objective sign of body fluid accumulation is currently available because there is no clear cut-off sign of fluid accumulation that can be used reliably to identify subjects with decompensated CHF. Previous experiences indicate that some patients with decompensated CHF have no classical clinical or radiographic signs of congestion, despite markedly elevated pulmonary filling pressures and decreased cardiac output (12,41).

Body fluid retention in decompensated CHF is reflected in physical signs of congestion, such as ankle edema, jugular venous distension and pulmonary crackles. Plain chest radiograph is also a traditional standard method of choice to obtain objective evidence of body fluid accumulation. At least seven studies (2,12,35–37,42,43) have examined the sensitivity, specificity and predictive value of physical signs for diagnosis of heart failure (25). These studies suggest that none of the signs of fluid retention (i.e., peripheral edema, jugular venous distension and pulmonary rales) is particularly sensitive, though specificity varied among studies (25). Additionally, some studies have shown that there is disagreement among several clinicians regarding recognition of classical physical signs of heart failure (44,45). The results of this study are consistent with these previous observations; the incidence of classical physical signs for identifying body fluid accumulation and the agreement between two observers on these signs, except for pulmonary rales, were only modest.

Radiography is still a standard method for evaluating heart failure patients. Two important specific markers for fluid accumulation on plain chest radiograph are pleural effusion and interstitial edema (hilar haziness, peribronchial cuffing, Kerley B lines) or alveolar pulmonary edema. Radiographic identification of pleural effusion (15,17), however, is reported to be insensitive and detects only moderate to large accumulation of pleural fluid. Indeed, this study demonstrated that detection of the fluid in either hemithorax using plain X-ray occurred in only 38% to 43% of patients and was confirmed by using X-ray thoracic CT, though concordance by two observers in identifying this sign was substantial. The incidence of roentgenographic signs of pulmonary congestion was also not high, and the interobserver agreement of this sign was only modest, which is consistent with previous observations (12,46). Previous reports suggested that chronically high left-sided heart pressures permit compensatory mechanisms to correct the fluid shift that has occurred, which can mask clinical and radiographic evidence of the underlying hemodynamic derangements. Increased lymphatic drainage will clear flooded alveoli, and radiographic evidence of edema might be absent (12,47,48).

Role of ultrasonography for detection of body fluid accumulation. The findings of the X-ray thoracic CT in this study indicated that as many as 90% of decompensated CHF patients have pleural effusion, suggesting that CT scan might be very useful for confirming pleural effusion in the uncontrolled stage of CHF. The most frequently encountered problems with CT scans are, however, difficulty in transporting critically ill patients and compelling the orthopneic patient to assume a supine position during the examination, which makes X-ray thoracic CT scans inconvenient or impractical (19). This study clearly demonstrated that thoracic ultrasonography efficiently supplemented the role of CT scan for the diagnosis of pleural effusion in decompensated CHF. Most importantly, the best clinical sign for identifying patients with decompensated CHF among the study variables was the detection of pleural fluid by thoracic ultrasonography. In our experience, evaluation of CHF patients using this method often revealed unexpected body fluid accumulation and changed patient management. The use of ultrasonography to detect decreased pleural effusion could become one of the most useful markers for confirming the therapeutic effectiveness of decompensated CHF. Of course, it should be kept in mind that the signs of body fluid accumulation, including thoracic ultrasonography, have a limited value for revealing latent myocardial failure or predicting the prognosis of CHF patients, because these markers are evident only with acute exacerbation of CHF. The measurement of one or more natriuretic peptides (9,10,33,34) will be more useful for this purpose.

Echocardiography combined with Doppler study is rapidly establishing itself as the primary investigation in patients with suspected heart failure (49–52). Thoracic ultrasonography, however, is not currently used in the routine assessment of heart failure patients. This technique has several advantages: 1) high predictive accuracy in detecting body fluid accumulation, 2) ease of performance at bedside, 3) flexibility and short examination time compared with X-ray thoracic CT scan, and 4) lower cost. These advantages make thoracic ultrasonography a useful diagnostic tool with great potential for assisting decision making and management of patients with decompensated CHF.

Several disadvantages should also be pointed out:

1) technical difficulty in obtaining a high quality of image in obese patients,
2) restricted field of view and
3) operator dependence.

Though recent guidelines for the evaluation and management of heart failure reported from the European Society of Cardiology (51) or from the American College of Cardiology/American Heart Association (52) do not include thoracic ultrasonography as a diagnostic tool for the evaluation of heart failure patients, this technique should be performed at an early stage in the management of any patient with a suspected diagnosis of CHF in order to obtain objective evidence of fluid accumulation in the pleural cavity.

Study limitations. There are important limitations to this study:

1) The major limitation of this study is the small number of patients and the lack of an additional control group of outpatients with stable CHF.
2) It is necessary to have an objective measure of whether body fluid accumulation is truly present in order to establish diagnostic accuracy and interobserver agreement of any given symptom or sign. There is, however, no gold standard test for the objective measure of body fluid accumulation in a clinical setting.
3) A valvular etiology due to degenerative valve disease in the aged was most frequent in this study, which is different from recent literature that report coronary artery disease predominance (53). This probably reflects the peculiar fact that patients with ischemic heart disease were preferentially referred to other hospital coronary care units because our hospital does not have a coronary care unit. Any impact of the inclusion of many heart failure patients due to chronic ischemic heart disease on the present form of study remains to be determined.
4) Previous studies report the clinical usefulness of the abdominojugular test (12) in assessing CHF patients. This variable was not evaluated in this study.
5) The findings of this study on body fluid retention in decompensated "chronic" heart failure cannot be extrapolated to those observed in "acute" heart failure (8), because compensatory mechanisms to correct the body fluid accumulation would differ between these two clinical syndromes (12,41,45,54).

Conclusions. Diagnosis of decompensated CHF in general practice is likely to remain haphazard and will continue to require objective substantiation. Results of this study indicated that thoracic ultrasonography is a simple, sensitive and accurate method for evaluation of the body fluid accumulation in patients with decompensated CHF. This technique can be used to assist in making the diagnosis of decompensated CHF and in the investigation of CHF patients during the follow-up period, if other causes of pleural effusion have been clinically ruled out.

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