View metadata, citation and similar papers at core.ac.uk
brought to you by CORE
provided by Diposit Digital de Documents de la UAB

CHEST
Original Research
SIGNS AND SYMPTOMS OF CHEST DISEASES

Noninvasive Assessment of Acute Dyspnea in the ED

Xaime García, MD; Peter Simon, MD; Francis X. Guyette, MD; Ravi Ramani, MD; Rene Alvarez, MD; Jorge Quintero, MD; and Michael R. Pinsky, MD, FCCP

Background: **We compared the ability of noninvasive measurements of cardiac output (CO) and thoracic fluid content (TFC) and their change in response to orthostatic challenges to diagnose acute decompensate heart failure (ADHF) from non-ADHF causes of acute dyspnea in patients in the ED.**
Methods: **Forty-five patients > 44 years old presenting in the ED with dyspnea were studied. CO and TFC were monitored with a NICOM bioreactance device. CO and TFC were measured continuously while each patient was sitting, supine, and during a passive leg-raising maneuver (3 min each); the maximal values during each maneuver were reported. Orthostatic challenges were repeated 2 h into treatment. One patient was excluded because of intolerance to the supine position. Diagnoses obtained with the hemodynamic measurements were compared with ED diagnoses and with two expert physicians by chart review (used as gold standard diagnosis); both groups were blinded to CO and TFC values. Patient's treatment, ED disposition, hospital length of stay, and subjective dyspnea (Borg scale) were also recorded.**
Results: **Sixteen of 44 patients received a diagnosis of ADHF and 28 received a diagnosis of non-ADHF by the experts. Baseline TFC was higher in patients with ADHF ($P = .001$). Fifteen patients were treated for ADHF, and their Borg scale values decreased at 2 h ($P < .05$). TFC threshold of 78.8 had a receiver operator characteristic area under the curve of 0.81 (76% sensitivity, 71% specificity) for ADHF. Both ADHF and non-ADHF groups were similar in their increased CO from baseline to PLR and supine. Pre- and posttreatment measurements were similar.**
Conclusions: **Baseline TFC can discriminate patients with ADHF from non-ADHF dyspnea in the ED.**
CHEST 2013; 144(2):610–615

Abbreviations: ADHF = acute decompensated heart failure; BNP = brain natriuretic peptide; CO = cardiac output; PLR = passive leg raising; ROC = receiver operator curve; SV = stroke volume; SVI = stroke volume index; TFC = thoracic fluid content

The diagnosis of acute decompensated heart failure (ADHF) in nonselected patients presenting with acute dyspnea is challenging. Symptoms and physical examination are nonspecific and lack the sensitivity to make an accurate and reliable diagnosis of heart failure in dyspneic patients presenting acutely to a medical facility.[1] Although helpful at times, commonly available adjunct testing including ECG, chest radiography, and serum troponin does little to improve the diagnosis of heart failure.[1-4] Although brain natriuretic peptide (BNP) does improve the diagnosis of heart failure in some settings, it is affected by age, sex, medications, and has large indeterminate range. BNP may not be diagnostic in cases of flash pulmonary edema, mitral regurgitation, or obesity.[5,6] Furthermore, the clinical assessment correlates poorly with either BNP levels or patient outcomes[7] and serial BNP levels are of limited value in patient management.[7,8]

Additional diagnostic accuracy may be obtained from echocardiography or invasive hemodynamic monitoring but these tools are expensive and may not be available in the acute care. The combination of a wide variation in hospital admission rates for heart failure[9-13] and inaccurate physician estimates of heart failure patient risk of death[7] suggest that a more accurate means of diagnosis in these patients might improve the appropriateness of admission and treatment decisions. Indeed, the ED physician inaccuracy in making the diagnosis of ADHF delays appropriate treatment.[7] The ideal adjunct for the diagnosis of ADHF would be noninvasive, inexpensive, able to increase the accuracy of diagnosis, and provide real-time information about

both the severity of the illness and its response to emergent treatment.

One such adjunct may be noninvasive assessment of cardiac output (CO) and thoracic fluid content (TFC) using bioreactance, based on measurement of phase shift in a high-frequency, low-voltage current conducted into the chest cavity though a series of external electrodes. Bioreactance uses changes in chest fluid content associated with changes in thoracic capacitance and inductive properties to estimate CO and TFC. This noninvasive method of CO monitoring was compared in several studies to thermodilution using a pulmonary artery catheter[14,15] documenting good correlation ($R = 0.82$) and minimal bias ($\pm 4\%$) (11) and a three times faster response rate.

Because ADHF may present with a wide range of individual CO values, knowing absolute values may not be sensitive or specific enough to discriminate among other causes of dyspnea. Using functional hemodynamic monitoring such as TFC and the dynamic changes in CO and TFC in response to a calibrated orthostatic challenge, such as passive leg-raising maneuver (PLR), should increase the utility of the bioreactance measures in identifying acute decompensation and discriminating among other noncardiac dyspnea etiologies.

We hypothesized that dynamic changes in CO and TFC induced by PLR would be quantitatively different between patients with cardiac and noncardiac causes of dyspnea. Specifically, patients with ADHF would display a higher TFC and decreased variability in both CO and TFC than patients who do not have ADHF dyspnea.

Materials and Methods

After University of Pittsburgh institutional review board approval (IRB0701090), we performed a prospective observational study of patients presenting with a chief complaint of dyspnea to the ED of an academic tertiary care center. All subjects signed informed consent to participate in the study, and no untoward events occurred as a result of subject participation.

Study Population

Patients ≥ 45 years of age presenting to the ED with the primary or significant complaint of dyspnea or difficulty breathing were included. We chose this age threshold since 97% of acute heart failure admissions occur in this age segment and heart failure in younger patients is often accompanied by other structural concerns not present in this older population. Furthermore, excluding patients < 45 years of age enhances screening efficiency because this younger age group accounts for approximately two-thirds of all ED visits but only 3% of patients in the ED diagnosed with heart failure.[16]

Exclusion criteria included transfer from another ED or hospital, known pregnancy that would preclude the use of a PLR maneuver, shortness of breath from known traumatic cause, limited code status, clinically unstable patients at the moment of ED admission (systolic BP < 85 mm Hg, heart rate over 1 min > 120, or $Sp_{O_2} < 92\%$), and having the primary clinical team giving the patient the diagnosis of acute coronary syndrome. Although new or worsening cardiac ischemia may be the underlying cause for new onset or an acute exacerbation of heart failure in patients with ADHF, usually clinical practice for patients presenting to the ED with chest pain and dyspnea suspected of cardiac origin is managed in accordance with hospital chest pain protocols that preclude the time required for the completion of our study. Patients were included consecutively when the research team was available (business days, from 8:00 AM to 12:00 PM).

Bioimpedance/Bioreactance Measurements

Mean and PLR-induced changes in CO and TFC were obtained using a NICOM device (Cheetah Medical Holdings). The NICOM uses four electrodes pairs in both midclavicular lines and both lower rib margins. The system requires 2 min to calibrate and has no detrimental effects on the patient. Following informed consent, initial CO and TFC measures were made in the first 15 min after ED admission while the patient was seated (hips flexed to 60° or more). These measures were interpreted as baseline values. We then performed a PLR maneuver for 3 min by placing the patient in a supine position or as flat as tolerated (at least $\leq 30°$) and with legs raised to 45°. Finally, patients were placed in the supine position without leg raising for another 3 min, and another measurement was taken (Fig 1). Those patients diagnosed by their ED physician as having ADHF received disease-specific treatment, which typically included a combination of nitrates, diuretics, angiotensin-converting enzyme inhibitors, and continuous or bilevel pressure ventilation. To assess the hemodynamic effect of this treatment, an additional set of three orthostatic measures was taken 2 h after the initial measures. To assess the subjective effect of the treatment, patients quantified their dyspnea severity using the Borg modified scale that measures the shortness-of-breath subjective feeling on a numeric scale from 0 (nothing) to 10 (maximal).

Since the maximum hemodynamic effect of a PLR maneuver is usually seen in the first 1 to 2 min,[17] we reviewed the CO and TFC data over the entire PLR maneuver and reported the highest CO and TFC values as the PLR effect. We compared absolute values and the changes in TFC and CO between baseline, PLR maneuver, and sitting to supine position for their ability to discriminate ADHF from non-ADHF diagnoses.

Additional Tests

We also collected physical examination data, ECG, and laboratory (including BNP if done) results, the Rapid Emergency

Manuscript received July 4, 2012; revision accepted January 10, 2013.
Affiliations: From the Department of Critical Care Medicine (Drs García, Simon, and Pinsky), University of Pittsburgh, Pittsburgh, PA; Department of Intensive Care Medicine (Dr García), Hospital of Sabadell, CIBER Enfermedades Respiratorias, Institut Universitari Parc Taulí–Autonomous, University of Barcelona, Sabadell, Spain; and Department of Emergency Medicine (Drs Guyette and Quintero) and Department of Cardiovascular Diseases (Drs Ramani and Alvarez), University of Pittsburgh, Pittsburgh, PA.
Funding/Support: This work was supported in part by National Institutes of Health [Grants HL67181 and HL073198].
Correspondence to: Michael R. Pinsky, MD, FCCP, 606 Scaife Hall, 3550 Terrace St, Pittsburgh, PA 15261; e-mail: pinskymr@upmc.edu
© 2013 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians. See online for more details.
DOI: 10.1378/chest.12-1676

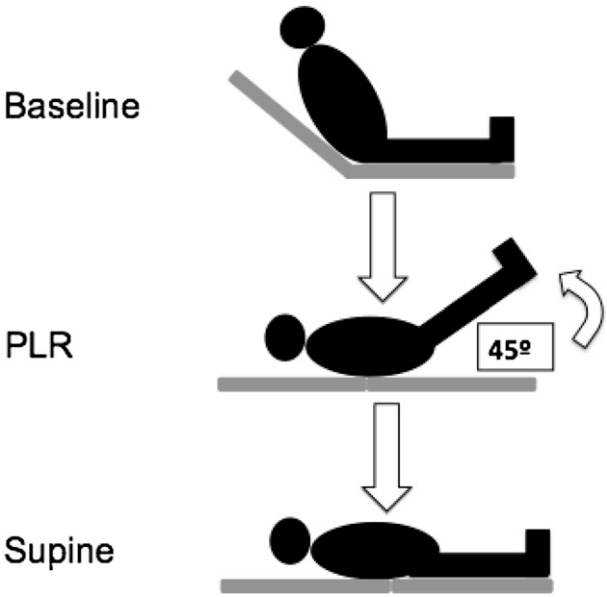

FIGURE 1. Orthostatic maneuvers. PLR = passive leg raising.

Medicine Score, and any additional inpatient data related to ADHF diagnosis.

ED physicians were blinded to the NICOM data. We collected the ED discharge diagnosis, hospital admission or ICU admission diagnosis and inpatient deaths, serious medical complications that occurred before hospital discharge, and hospital discharge date used to calculate hospital free days (over 30). Inpatient death was considered 0.

Outcome Adjudication

Diagnosis as ADHF or non-ADHF was determined by two cardiologists expert in heart failure diagnosis, after a retrospective review of patient's medical chart with full access to all the adjunctive tests made to the patient during hospital stay, but blinded to both the CO and TFC results and the ED physician's diagnosis. These independent reviewers provided scores obtained using the Framingham[18] and National Health and Nutrition Examination Survey heart failure clinical score[19] to identify patients with heart failure. Both cardiologists had to agree in one diagnosis after a separated review of medical records. In case of disagreement, the experts were asked to meet and reach a common diagnosis. In practice, both cardiologists reached the same diagnosis on all patients during their independent reviews. The ED physician's diagnosis reflects the diagnosis for ED discharge or hospital admission as made by the attending ED physician.

Statistical Methods

We compared both the ED physician diagnosis and those assigned based on initial CO and TFC values against the diagnosis obtained by two independent reviewers by χ^2 testing. α was defined as $P < .05$. Baseline characteristics used mean values and proportions. We compared ED physician diagnosis based on initial impression and with the addition of laboratories and clinical data to the orthostatic CO and TFC data using a two-sample t test. χ^2 testing and ORs were computed to compare the orthostatic CO and TFC data vs the diagnosis defined by the expert reviewers. We generated receiver operator curves (ROCs) for physician assessment, orthostatic CO, orthostatic TFC, and combined orthostatic CO and TFC. We used the Cohen κ index to assess the agreement between expert diagnosis of ADHF and ED physician diagnosis.

RESULTS

Forty-five patients were included in the study over 6 months (April-August 2010). One patient was excluded after initial inclusion due to intolerance to the orthostatic challenge maneuvers. Dynamic (but not baseline) TFC changes in the first 15 patients were discarded due to technical problems during the orthostatic challenges with the initial Bioreactance software program. The expert reviewers classified post hoc 16 patients as ADHF and 28 as non-ADHF. Table 1 shows the patient demographic data.

Although baseline heart rate was higher in patients without ADHF, there were not significant differences in stroke volume (SV) and stroke volume index (SVI) with the orthostatic challenges among the two diagnostic groups (Table 2) nor in the difference between maneuvers (Δ baseline to PLR and Δ baseline to supine) (Table 3).

TFC was significantly higher at baseline in patients with ADHF ($P = .001$) (Fig 2) and remained higher during the orthostatic maneuvers. The baseline TFC value showed ROC area under the curve of 0.81 that was higher than the ED physician accuracy for ADHF diagnosis (0.74). We found a cutoff TFC value of 78.8 1/kW in baseline, having 76% sensitivity, 71% specificity, positive likelihood ratio of 2.6, and a negative likelihood ratio of 0.3 (Fig 3).

We found a moderate agreement (77%) between ED physicians and the expert reviewers (κ, 0.46), indicating that the accuracy of ADHF diagnosis by ED physicians remains limited. Those patients who were correctly diagnosed and treated as ADHF showed a significant decrease in the Borg modified scale dyspnea subjective perception (from 5 ± 2 to 3 ± 2, $P < .05$). There were no significant differences in the hemodynamic parameters.

BNP was measured in seven patients. However, it was more frequently measured in those patients with

Table 1—*Demographic Data Separated by Expert Diagnosis of Dyspnea*

	Expert Diagnosis	
Demographics	ADHF (n = 16)	Non-ADHF (n = 28)
Age, y	65 ± 11	60 ± 12
Sex, M (F)	9 (7)	11 (17)
BSA, m²	1.95 ± 0.28	1.83 ± 0.27
Heart rate, bpm	74 ± 16	88 ± 18
Respiratory rate, breaths/min	22 ± 4	19 ± 3
Systolic BP, mmHg	140 ± 29	134 ± 27
SaO_2, %	96 ± 2%	97 ± 2%
Borg scale	4 ± 3	5 ± 2
REMS score	6.8 ± 2.2	5.6 ± 2.4

Values (except sex) expressed in mean ± SD. ADHF = acute decompensated heart failure; bpm = beats per min; BSA = body surface area; F = female; M = male; REMS = Rapid Emergency Medicine Score; SaO_2 = pulse oximetry oxygen saturation.

Table 2—*Hemodynamic Values at Baseline (30° Trunk Elevation), PLR, and in Supine Position*

	Baseline			PLR			Supine		
Value	ADHF	Non-ADHF	*P* Value	ADHF	Non-ADHF	*P* Value	ADHF	Non-ADHF	*P* Value
Heart rate, bpm	74 ± 16	88 ± 18	.02	75 ± 16	87 ± 18	.04	75 ± 16	86 ± 18	.06
SV, mL	79 ± 28	72 ± 25	.36	91 ± 43	78 ± 26	.23	86 ± 33	77 ± 25	.30
SVI, mL/m²	40 ± 11	39 ± 10	.07	45 ± 17	42 ± 11	.21	43 ± 13	42 ± 11	.17
TFC, 1/kW	94 ± 22	71 ± 31	.001	93 ± 21	72 ± 33	.002	94 ± 22	73 ± 39	.002

Values expressed in mean ± SD. PLR = passive leg raising; SV = stroke volume; SVI = stroke volume index; TFC = thoracic fluid content. See Table 1 legend for expansion of other abbreviations.

a final diagnosis of ADHF (37.5% vs 3.6%). In addition, 71.4% of patients not diagnosed with ADHF were admitted, as well as 93.3% of those diagnosed by the experts as ADHF. There was no significant difference in hospital-free days between ADHF and non-ADHF groups (25 ± 5 days vs 26 ± 5 days).

Discussion

We found a significant difference in baseline TFC but not CO between patients diagnosed with ADHF and those not diagnosed with ADHF presenting to the ED with acute dyspnea. However, we also found no difference in either ΔTFC or ΔCO in response to an orthostatic challenge between ADHF and non-ADHF groups. Although CO increased with supine and PLR maneuvers compared with baseline, the increase was not different between patient groups. That only one patient of 45 did not tolerate the supine position and PLR maneuvers documents that these orthostatic challenges are safe for patients with dyspnea presenting to the ED.

Our data are only in partial agreement with those previously reported by Engineer et al.[20] They found a similar increased baseline TFC in patients with ADHF compared with their patients with COPD who did not have ADHF. Presumably, these differences reflect differences in methodologies. First, we used both the supine position and a PLR maneuver to provoke the dynamic changes in venous return, while Engineer et al[20] used only the dynamic change from sitting (hips flexed to 90°) to supine position or as flat as tolerated. Still, in our study, TFC remained significantly higher in patients with ADHF during both challenges.

Our reference ADHF diagnosis was retrospective chart review by two expert cardiologists; Engineer et al[20] used either ED physician diagnosis combined with BNP, or only BNP levels > 500 pg/mL. Regrettably, ED physician diagnosis accuracy is not higher than 80% and even combined to BNP is only 82.5%.[6] Our study corroborates their findings because our ROC showed accuracy for ED physician diagnosis of 0.74, although BNP was rarely measured in the subjects. This low accuracy and the moderate agreement found between ED physician and expert reviewer diagnosis underscores the necessity of diagnostic tools to help ED physicians accurately make the ADHF diagnosis. The fact that, in our study, those patients who received mismatched treatment of ADHF had less symptom improvement further emphasizes this necessity. Our study demonstrates that baseline TFC could be such a valid tool.

Another important difference between our study and the one by Engineer et al[20] is that we had very restrictive exclusion criteria regarding clinical instability, while in their study even patients with the greatest severity of illness were not excluded. Thus, it is possible that their patients with ADHF were more severe than in our sample, and this could explain why their patients with ADHF had the lower cardiac index response to the orthostatic challenges.

Study Limitations

Although the NICOM CO and TFC measurements are already validated, CO and TFC measures use different analyses. CO measures are based on bioreactance (phase shift in the oscillating electrical field), whereas TFC is based in bioimpedance. Bioimpedance

Table 3—*Variation in Bioreactance/Biompedance Parameters With Orthostatic Changes*

	Δ From Baseline to PLR			Δ From Baseline to Supine		
Parameter	ADHF, %	Non-ADHF, %	*P* Value	ADHF, %	Non-ADHF, %	*P* Value
SV	11 ± 16	9 ± 14	ns	8 ± 14	9 ± 22	ns
SVI	11 ± 16	9 ± 15	ns	9 ± 22	9 ± 15	ns
TFC	0 ± 2	1 ± 4	ns	0 ± 2	3 ± 6	ns

Values expressed in percentage ± SD. ns = not significant. See Table 1 and 2 legends for expansion of other abbreviations.

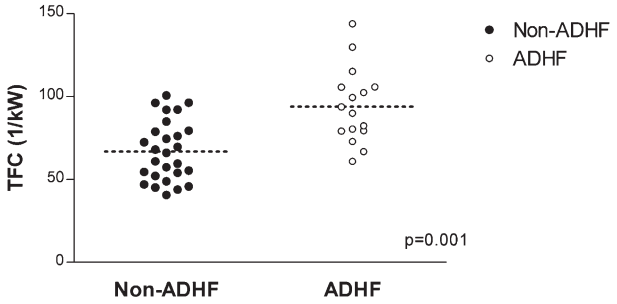

FIGURE 2. Baseline TFC differences between groups. Note that one outlier measurement has been removed from the non-ADHF group in this figure as it was > 2 SD from the mean value. ADHF = acute decompensated heart failure; TFC = thoracic fluid content.

is more likely to be affected by external interferences. Potentially, this may explain the lack of separation of ADHF from non-ADHF in response to orthostatic challenges. We doubt this because the TFC and CO standard deviation values at baseline for all patients were proportionally similar. Furthermore, we excluded clinically unstable patients who presumably would have more severe ADHF. Thus, our results may only be applicable to patients with less severe dyspnea. Finally, the absence of TFC data only after the orthostatic maneuver in the first 15 patients may reduce the power of the study to detect TFC changes, but not CO changes.

CONCLUSIONS

Baseline TFC is higher in patients with ADHF than in patients who do not have ADHF presenting to the ED with acute dyspnea. This noninvasive rapid measure can be a useful tool for discriminating dyspnea caused by ADHF in the ED.

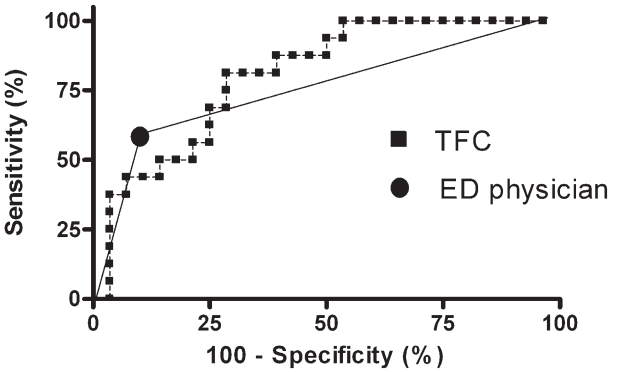

FIGURE 3. Receiver operator curve predicting ADHF diagnosis. ED physician area under curve was 0.74. TFC baseline area under curve was 0.81. Cutoff value was TFC 78.8 (sensitivity, 76%; specificity, 71%; LR+, 2.6; LR−, 0.3). LR− = mean negative likelihood ratio; LR+ = mean positive likelihood ratio. See Figure 2 legend for expansion of other abbreviations.

ACKNOWLEDGMENTS

Author contributions: Dr García is guarantor of the manuscript and takes responsibility for the integrity of the data and accuracy of the data analysis.
Dr García: contributed to the conception and design of the study, obtained research funding, recruited patients, performed the data management and analysis, and drafted the manuscript.
Dr Simon: contributed to the conception and design of the study, performed the data management and analysis, provided statistical advice, and contributed substantially to the manuscript's revision.
Dr Guyette: contributed to the conception and design of the study and revision of the manuscript.
Dr Ramani: contributed by performing the expert diagnosis and writing the manuscript.
Dr Alvarez: contributed by performing the expert diagnosis and writing the manuscript.
Dr Quintero: contributed by performing the expert diagnosis and writing the manuscript.
Dr Pinsky: contributed to the conception and design of the study, obtained research funding, supervised the conduct of the trial and data collection as well as quality control, and contributed substantially to the manuscript's revision.
Financial/nonfinancial disclosures: The authors have reported to *CHEST* the following conflicts of interest: Dr Pinsky has stock options from Cheetah Medical Holdings. Drs García, Simon, Guyette, Ramani, Alvarez, and Quintero have reported that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.
Role of sponsors: The sponsor had no role in the design of the study, the collection and analysis of the data, or in the preparation of the manuscript.

REFERENCES

1. Stevenson LW, Perloff JK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure. *JAMA*. 1989;261(6):884-888.
2. Remes J, Miettinen H, Reunanen A, Pyörälä K. Validity of clinical diagnosis of heart failure in primary health care. *Eur Heart J*. 1991;12(3):315-321.
3. Yealy DM, Hsieh M. BNP is not a value-added routine test in the emergency department. *Ann Emerg Med*. 2009;53(3): 387-389.
4. Collins SP, Lindsell CJ, Storrow AB, Abraham WT; ADHERE Scientific Advisory Committee, Investigators and Study Group. Prevalence of negative chest radiography results in the emergency department patient with decompensated heart failure. *Ann Emerg Med*. 2006;47(1):13-18.
5. Silver MA, Maisel A, Yancey CW, et al. BNP Consensus Panel 2004: a clinical approach for the diagnostic, prognostic, screening, treatment, monitoring, and therapeutic roles of natriuretic peptides in cardiovascular diseases. *Cong Heart Failure*. 2004;10(5 suppl 3):1-30.
6. McCollugh PA, Nowak RM, McCord J, et al. B-type natriuretic peptide and clinical judgment in emergency diagnosis of heart failure: analysis from Breathing Not Properly (BNP) Multinational study. *Circulation*. 2002;106(4):416-422.
7. Maisel A, Hollander JE, Guss D, et al. Primary results of the Rapid Emergency Department Heart Failure Outpatient Trial (REDHOT): a multicenter study of B-type natriuretic peptide levels, emergency department decision making, and outcomes in patients presenting with shortness of breath. *J Am Coll Cardiol*. 2004;44(6):1328-1333.
8. Singer AJ, Birkhahn RH, Guss D, et al. Rapid Emergency Department Heart Failure Outpatients Trial (REDHOT II): a randomized controlled trial of the effect of serial B-type

natriuretic peptide testing on patient management. *Circ Heart Fail.* 2009;2(4):287-293.
9. McMahon LF, Wolfe RA, Tedeschi PJ. Variation in hospital admissions among small areas. A comparison of Maine and Michigan. *Med Care.* 1989;27(6):623-631.
10. Roos NP, Wennberg JE, McPherson K. Using diagnosis-related groups for studying variations in hospital admissions. *Health Care Financ Rev.* 1988;9(4):53-62.
11. Wennberg JE, Greeman JL, Culp WJ. Are hospital services rationed in New Haven or over-utilised in Boston? *Lancet.* 1987;1(8543):1185-1189.
12. Rosenthal GE, Shah A, Way LE, Harper DL. Variations in standardized hospital mortality rates for six common medical diagnoses: implications for profiling hospital quality. *Med Care.* 1998;36(7):955-964.
13. Smith WR, Poses RM, McClish DK, et al. Prognostic judgments and triage decisions for patients with acute congestive heart failure. *Chest.* 2002;121(5):1610-1617.
14. Squara P, Denjean D, Estagnasie P, Brusset A, Dib JC, Dubois C. Noninvasive cardiac output monitoring (NICOM): a clinical validation. *Intensive Care Med.* 2007;33(7):1191-1194.
15. Raval NY, Squara P, Cleman M, Yalamanchili K, Winklmaier M, Burkhoff D. Multicenter evaluation of noninvasive cardiac output measurement by bioreactance technique. *J Clin Monit Comput.* 2008;22(2):113-119.
16. Schappert SM. Ambulatory care visits to physician offices, hospital outpatient departments, and emergency departments: United States, 1997. *Vital Health Stat 13*. 1999;(143):1-39.
17. Monnet X, Rienzo M, Osman D, et al. Passive leg raising predicts fluid responsiveness in the critically ill patients. *Crit Care Med.* 2006;34(5):1402-1407.
18. McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure: the Framingham study. *N Engl J Med*. 1971;285(26):1441-1446.
19. Schocken DD, Arrieta MI, Leaverton PE, Ross EA. Prevalence and mortality rate of congestive heart failure in the United States. *J Am Coll Cardiol*. 1992;20(2):301-306.
20. Engineer RS, Benoit JL, Hicks CW, Kolattukudy SJ, Burkhoff D, Peacock WF. Hemodynamic changes as a diagnostic tool in acute heart failure—a pilot study. *Am J Emerg Med*. 2012;30(1):174-180.