Impact of Impedance Cardiography on Diagnosis and Therapy of Emergent Dyspnea: The ED-IMPACT Trial

W. Frank Peacock, MD, Richard L. Summers, MD, Jody Vogel, MD, Charles E. Emerman, MD

Abstract

Background: Dyspnea is one of the most common emergency department (ED) symptoms, but early diagnosis and treatment are challenging because of multiple potential causes. Impedance cardiography (ICG) is a noninvasive method to measure hemodynamics that may assist in early ED decision making.

Objectives: To determine the rate of change in working diagnosis and initial treatment plan by adding ICG data during the course of ED clinical evaluation of elder patients presenting with dyspnea.

Methods: The authors studied a convenience sample of dyspneic patients 65 years and older who were presenting to the EDs of two urban academic centers. The attending emergency physician was initially blinded to the ICG data, which was collected by research staff not involved in patient care. At initial ED presentation, after history and physical but before central lab or radiograph data were returned, the attending ED physician completed a case report form documenting diagnosis and treatment plan. The physician then was shown the ICG data and the same information was again recorded. Pre- and post-ICG differences were analyzed.

Results: Eighty-nine patients were enrolled, with a mean age of 74.8 ± 7.0 years; 52 (58%) were African American, 42 (47%) were male. Congestive heart failure and chronic obstructive pulmonary disease were the most common final diagnoses, occurring in 43 (48%), and 20 (22%), respectively. ICG data changed the working diagnosis in 12 (13%; 95% CI = 7% to 22%) and medications administered in 35 (39%; 95% CI = 29% to 50%).

Conclusions: Impedance cardiography data result in significant changes in ED physician diagnosis and therapeutic plan during the evaluation of dyspneic patients 65 years and older.

ACADEMIC EMERGENCY MEDICINE 2006; 13:365–371 © 2006 by the Society for Academic Emergency Medicine

Keywords: dyspnea, hemodynamics, impedance cardiography, bioimpedance, cardiac output, systemic vascular resistance, noninvasive

Dyspnea is one of the most common emergency department (ED) symptoms in older patients.[1] Various conditions, including heart failure, chronic obstructive pulmonary disease, pneumonia, pulmonary embolus, and acute coronary syndromes, may occur alone or in combination in a given patient, adding uncertainty to diagnosis and treatment. In patients with both cardiac and pulmonary disease, the initial assessment and therapy in the ED are challenging.

Because cardiovascular disease, specifically decompensated heart failure (HF), is a relatively common cause of dyspnea in elders, an assessment of hemodynamics,

From the Department of Emergency Medicine, Cleveland Clinic (WFP), Cleveland, OH; Department of Emergency Medicine, University of Mississippi (RLS), Jackson, MS; Wayne State University (JV), Detroit, MI; and Department of Emergency Medicine, Case Western Reserve University (CEE), Cleveland, OH.
Received September 6, 2005; revision received November 7, 2005; accepted November 15, 2005.

Supported by GE Medical Systems (Milwaukee, WI), which provided devices and disposables for this study, and by CardioDynamics (San Diego, CA), which provided a study grant for support of research assistants. In addition, CardioDynamics participated in the creation of the educational process for the participating physicians before study commencement. The sponsor had no role in data collection or statistical analyses, and the manuscript is the sole responsibility of the authors. W.F.P. and R.L.S. received honoraria in 2003 for speaking for CardioDynamics.

Presented as a moderated poster at the American College of Emergency Physicians Research Forum, October 2003.

Address for correspondence and reprints: W. Frank Peacock, MD, The Cleveland Clinic Foundation, Department of Emergency Medicine, Desk E-19, 9500 Euclid Avenue, Cleveland, OH 44195. Fax: 216-445-4552; e-mail: peacocw@ccf.org.

doi: 10.1197/j.aem.2005.11.078
ISSN 1069-6563
PII ISSN 1069-6563583

including cardiac output, systemic vascular resistance, and fluid status, may provide important information and aid decision making beyond what is possible from history and physical examination alone. Unfortunately, hemodynamic parameters cannot be accurately determined by patient history or physical examination.[2–5] Until recently, hemodynamic data could only be obtained by pulmonary artery catheterization. Because this invasive procedure is not practical in the ED, physicians typically are left to make diagnosis and treatment decisions without reliable information about a patient's hemodynamic status.

Noninvasive hemodynamic monitoring by impedance cardiography (ICG) has been used in more than four million patients. Cardiac output (CO) by ICG has been shown to correlate well with CO obtained by invasive methods in hospitalized patient populations with correlation coefficients for CO by ICG and thermodilution ranging from 0.76 to 0.89.[6–10] ICG also has been used as an alternative to invasive monitoring in the critical care setting.[11] In the ED setting, ICG has been studied for the differential diagnosis of dyspnea[12–14] and the identification of pulmonary edema[15,16] and provides prognostic information about hospitalization costs and length of stay.[17] ICG results are available within a few minutes, allowing more rapid patient evaluation than that afforded by radiographic or laboratory studies.

Given the high rate of morbidity, mortality, and hospital readmissions for patients with dyspnea and acute decompensated HF, there is an urgent need to examine technologies that could lead to improvements in care in the ED. The present study examines an aspect of therapeutic efficacy[18] as it relates to ICG and the acutely dyspneic emergency patient, and not simply the performance of ICG as a testing modality. Put into context, previous studies of commonly used ED tools, such as pulse oximetry[19,20] and B-type natriuretic peptide (BNP) testing,[21] suggest that a 5% to 11% rate of change in diagnosis, or 10% rate of change in therapy, is clinically relevant. The effect of ICG-derived hemodynamics on diagnosis and treatment of dyspnea in the ED is not yet known. The purpose of this study was to determine the rate of change in diagnosis and therapy resulting from the availability of ICG data during the initial evaluation of older ED patients presenting with dyspnea.

METHODS

Study Design

This was a prospective study of dyspneic patients that was designed to determine the frequency of change in the ED physician's initial diagnosis and therapeutic plan after physician access to noninvasive ICG hemodynamic data. The study was approved by each hospital's institutional review board. All patients gave informed consent before enrollment in the study.

Study Setting and Population

The setting was two large urban academic EDs with experience in using noninvasive hemodynamic monitoring. A convenience sample was obtained from patients age 65 years or older who were presenting with a chief complaint of dyspnea or symptoms of HF, as determined by the ED physician. Because ICG is not currently recommended (per U.S. Food and Drug Administration guidelines) for the diagnosis of acute coronary syndromes, including acute myocardial infarction (MI), patients were excluded if electrocardiogram (ECG) or serum markers were positive for acute MI. Additional exclusions included the following: if ICG monitoring was not possible because of inability to place electrodes, if the patient's weight was greater than 341 pounds, or if the patient had an activated minute ventilation pacemaker (which uses an impedance signal). Also, although severe aortic regurgitation that could give a falsely elevated ICG CO is rare and generally evident on ED evaluation, we excluded those with aortic regurgitation by past history, and those with the typical diastolic murmur. Last, because the treatment and disposition actions for patients needing immediate intubation and mechanical ventilation are generally well defined from the emergency physician point of view, and because it was our intent to study the diagnostically most challenging patients, those requiring urgent intubation and mechanical ventilation upon presentation to the ED were excluded.

Study Protocol

Project coordinators screened candidates and an independent research nurse, not involved in the diagnosis or treatment of the patient, obtained hemodynamic data. Hemodynamic data were collected by using the BioZ ICG monitor (CardioDynamics, San Diego, CA), as has been described elsewhere.[22] ICG data are obtained by the following technique: four dual sensors (each sensor consisting of two electrodes) are placed on the patient, as shown in Figure 1, on opposite sides of the neck at a level between the ears and shoulders and on either side of the chest in the mid-axillary line at the level of the xiphoid process. The outer electrodes in each sensor transmit a low-amplitude, high-frequency current (2.5 mA, 70 kHZ), and the inner electrodes detect thoracic voltage changes. Changes in voltage are used to calculate changes in impedance (Z). Baseline, static impedance is indicative of chest fluid volume, and dynamic impedance

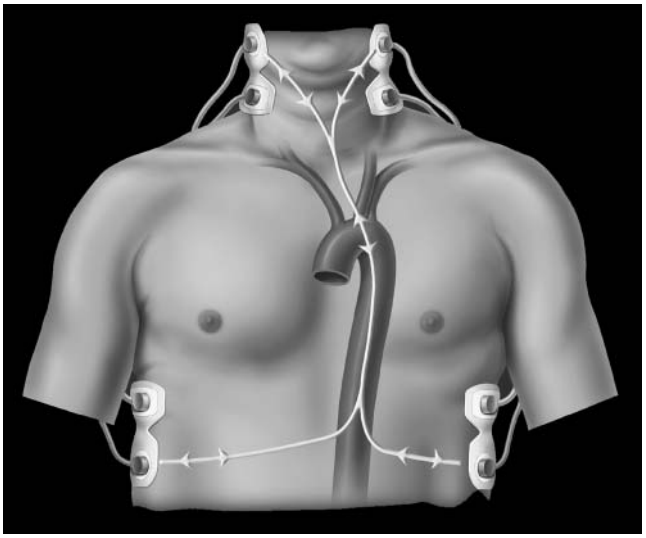

Figure 1. Front view of impedance cardiography method.

is affected by aortic blood volume and velocity. Beat-to-beat changes in thoracic impedance are processed to calculate blood flow per heartbeat (stroke volume) and per minute (cardiac output). By using standard equations, other hemodynamic parameters, such as systemic vascular resistance, are calculated. The reciprocal of baseline thoracic impedance can provide an index of intrathoracic fluid and is termed *thoracic fluid content* (TFC). TFC has been used to identify intravascular and extravascular fluid changes[23,24] and to titrate diuretic therapy.[25]

Before study initiation, participating physicians received instruction regarding the interpretation of the hemodynamic values obtained by the ICG device. Attending physicians, all of whom were board-certified or board-eligible in emergency medicine, were given a description of ICG technology and hemodynamic parameters provided on the ICG report, including definitions and normal values for cardiac index (CI), resistance, thoracic fluid content, and measures reflecting left ventricular performance. This was performed at departmental grand rounds and at the monthly attending-physician staff meeting. Additional information was disseminated in hardcopy by mailing and was duplicated by e-mail. The pathophysiology of HF and hemodynamic findings most suggestive of dyspnea caused by decompensated HF (reduced CI, elevated systemic vascular resistance, and increased TFC) were described. The expected effects of various medications on hemodynamic parameters were discussed, including use of diuretics, vasodilators, and drugs affecting contractility. Additionally, physicians were provided personal reference cards for use at their discretion that detailed normative values for all ICG data. Copies of the data card were also kept fixed to the ICG device. These data were also shown at the time of ICG unblinding. For any given parameter, ICG data are presented as a bar indicating the normal human range. The average result and the currently measured data point then are indicated on this bar, such that variations from normal are readily apparent.

All staff involved with patient care were blinded to the ICG data until after the initial history and physical examination by the attending physician. After the initial history and physical exam, but before initiation of therapy (other than supplemental oxygen), and before obtaining any central laboratory or radiographic data, the attending physician completed a case report form indicating his or her working diagnoses and short-term therapeutic plans. The physician was then immediately shown the ICG hemodynamic data and was asked to complete the case report form again, this time with consideration of the ICG data. All patient care then proceeded according to usual ED routine. Blood tests, including electrolytes, blood urea nitrogen (BUN), serum creatinine (Cr), and BNP levels, were obtained in the majority of cases. Although these data were not mandated as part of the protocol, they were used in most cases to determine final ED diagnosis.

Measures

The two primary endpoints were 1) the rates of change in working diagnosis and 2) medical therapy after the addition of ICG data to the physician's initial clinical assessment and therapeutic plan. In the cases in which the diagnosis changed on the basis of ICG data, a comparison to the final diagnosis was made to determine whether the pre- or post-ICG diagnosis was more consistent with the final ED diagnosis. The final ED primary diagnosis was defined as the principal diagnosis at the end of the ED visit after all diagnostic testing was completed and reviewed by the ED physician responsible for disposition. A change in therapy was defined as the addition or subtraction of a drug or procedure. Changing the dose of a previously ordered drug was not considered a therapeutic change. Adverse events were defined as cardiac arrest, intubation for respiratory failure, urgent cardioversion, or blood transfusion.

Data Analysis

The size of the study was prospectively determined on the basis of the number needed to detect a 5% rate of change in diagnosis or therapy. Given an alpha of 0.05 and a beta of 0.20, a sample size of 100 was needed to detect a statistically significant change. Data were analyzed by an independent statistician using SAS Software (Cary, NC).

Demographic data are reported descriptively. Continuous variables are reported as mean ± standard deviation (SD). Rates of change were calculated by dividing the number of patients in whom diagnosis or therapeutic plan changed by the total number of patients and were reported as percentages. An analysis of variance was performed to assess for differences among vital signs and ICG parameters in the final diagnosis categories.

RESULTS

Eighty-nine patients, cared for by 31 ED staff physicians, were enrolled from December 2001 through July 2003 and are included in the analysis. No adverse event, defined as cardiac arrest, intubation, cardioversion, or blood transfusion, occurred during the course of the ED observation during this study.

The patient characteristics and vital signs are summarized in Table 1. The mean (±SD) age of the subjects was 74.8 (±7.0) years. Fifty-eight percent of the patients were African American, and 61% had a history of HF,

Table 1
Patient Characteristics and Vital Signs

Patient Characteristic	Value
Male, *n* (%)	42 (47)
Ethnic background, *n* (%)	
African American	52 (58)
White	36 (41)
Other	1 (1)
Medical history, *n* (%)	
History of heart failure	43 (48)
History of chronic obstructive pulmonary disease or asthma	34 (38)
History of heart failure and COPD	11 (13)
Vital signs (mean ± SD)	
Temperature (ºC)	36.5 ± 0.7
Respiratory rate (min^{-1})	22.2 ± 5.1
Heart rate (min^{-1})	84.6 ± 18.8
Systolic blood pressure (mm Hg)	145.6 ± 29.3

N = 89.

Table 2
Hemodynamic Values at Presentation

Hemodynamic Parameter	Mean ± SD	Normal Range
Thoracic fluid content ($kOhm^{-1}$)	34.2 ± 10.4	30–50 (male) 21–37 (female)
Systemic vascular resistance (dyne sec cm^{-5})	1,685 ± 579	742–1,378
Cardiac index ($L/min/m^2$)	2.6 ± 0.6	2.5–4.2
Stroke index (mL/m^2)	31.8 ± 10.1	35–65

N = 89.

including 13% with a history of both HF and chronic lung disease. The prevalence of chronic lung disease, including asthma, was 38%. The average respiratory rate was 22.2 (±5.1) min^{-1} with systolic BP and heart rate of 145.5 (±29.3) mm Hg and 84.6 (±18.8) min^{-1}, respectively. The hemodynamic values for the population as a whole are listed in Table 2.

Patients could be categorized by final primary diagnosis at the time of ED discharge or hospital admission into three major groups: 1) HF (n = 43); 2) chronic obstructive pulmonary disease (COPD; 20); and 3) "other" (26). The *other* group included other cardiovascular and lung conditions not included in the HF or COPD groups: atrial fibrillation (n = 4), bronchitis (4), hypertension (2), pneumonia (2), pulmonary hypertension (2), anemia (1), influenza (1), lung cancer (1), palpitations (1), upper respiratory infection (1), atypical chest pain (1), hypoxia (1), intra-abdominal abscess (1), non-cardiac shortness of breath (1), pulmonary fibrosis (1), vertigo (1), and dehydration (1).

Chest radiographs and ECG results were recorded by the ED physician in 85 patients (96%). The various ECG and radiographic findings are summarized in Table 3. ECG findings were described as normal or nonspecific in the vast majority (82%), and the chest radiograph was normal or nondiagnostic in nearly half, with only 16% showing either HF or upper zone redistribution consistent with pulmonary venous congestion.

Table 3
Electrocardiographic and Chest Radiographic Findings

Parameter	n (%)
Electrocardiographic findings	
Normal, nonspecific, or "nondiagnostic ECG"	70 (82)
Left ventricular hypertrophy	15 (18)
Atrial fibrillation	11 (13)
Prior myocardial infarction	8 (9)
Left bundle branch block	3 (4)
Relevant chest radiography findings	
Normal or no acute disease	42 (49)
Increased cardiothoracic ratio (>0.5) or cardiomegaly	13 (15)
Pleural effusion	8 (9)
Pulmonary edema or "heart failure"	8 (9)
Upper zone redistribution	6 (7)
Pulmonary infiltrate	5 (6)
Atelectasis	2 (2)
Hyperinflation	1 (1)

For each finding group, n = 85. Because of multiple findings, total for ECG diagnoses is greater than 85.

Diagnosis Changes

A summary of the rates of diagnosis and therapy change that resulted from ICG data are presented in Figure 2. ICG data changed the working diagnosis in 12 (13%; 95% CI = 7% to 22%). When diagnoses were categorized as either cardiac or noncardiac, the post-ICG diagnosis was the same as the final diagnosis in 8 of 12 patients in whom ICG resulted in a change (67%, 95% CI = 35% to 90%). Of the four patients in whom a change in diagnosis after ICG did not match the final ED diagnosis, one who was ultimately diagnosed with a cardiac cause of dyspnea had normal hemodynamic parameters, suggesting a pulmonary cause. In another patient, altered hemodynamic parameters suggested cardiac dyspnea that was later attributed to an exacerbation of COPD. One patient with lung cancer had hemodynamic findings consistent with diastolic HF. Finally, one patient who initially was thought to have pulmonary dyspnea had altered hemodynamic findings that were believed to be nondiagnostic by the evaluating physician; that patient was ultimately treated for fluid overload and discharged home.

Results Grouped by Final Diagnosis

A summary of the patient vital signs and hemodynamic characteristics grouped by final ED diagnosis is listed in Table 4. No diagnosis group had vital sign data that were significantly different from those of any other group (p = 0.1332). Of the hemodynamic parameters, cardiac index, systemic vascular resistance index, and thoracic fluid content had one diagnosis group that differed significantly from the other two (p < 0.02). HF patients had greater amounts of lung water, as reflected by a mean TFC (38.5 ± 12.3 $kOhm^{-1}$) that was significantly higher than that of the other two diagnosis groups (30.0 ± 6.17 and 30.4 ± 5.6 for the COPD and *other* groups, respectively). Patients with COPD had higher CI (3.08 ± 0.57 vs. 2.39 ± 0.56 and 2.48 ± 0.65) and lower SVR (1,361 ± 407 vs. 1,772 ± 565 and 1,789 ± 638) than did patients in the HF or *other* groups, respectively.

Laboratory measurements, including electrolytes, BUN, Cr, WBC, Hgb, and BNP were analyzed by final diagnosis. Of the laboratory measurements, only BNP, measured in 72 patients, exhibited a statistically

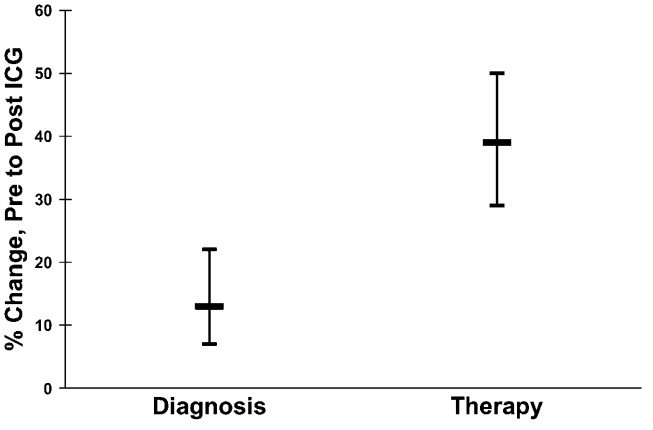

Figure 2. Category rate of change from pre-ICG to post-ICG (mean values with 95% confidence intervals).

Table 4
Initial Vital Signs and Selected Hemodynamic Parameters by Final Diagnosis

Parameter	Final Diagnosis: Heart Failure (n = 43)	Chronic Obstructive Pulmonary Disease (n = 20)	Other (n = 26)
Temperature (ºC)	36.4 ± 0.7	36.6 ± 0.8	36.7 ± 0.5
Systolic blood pressure (mm Hg)	149.5 ± 30.5	135.6 ± 19.1	146.8 ± 32.2
Diastolic blood pressure (mm Hg)	80.2 ± 18.0	75.7 ± 13.0	78.5 ± 21.2
Heart rate (min^{-1})	84.1 ± 18.0	88.2 ± 19.4	82.9 ± 20.1
Respiration rate (min^{-1})	22.1 ± 5.7	23.8 ± 3.8	21.0 ± 4.6
Thoracic fluid content ($kOhm^{-1}$)	38.5 ± 12.3*	30.0 ± 6.17	30.4 ± 5.6
Systemic vascular resistance (dyne sec cm^{-5})	1,772 ± 565	1,361 ± 407*	1,789 ± 638
Cardiac index ($L/min/m^2$)	2.39 ± 0.56	3.08 ± 0.57*	2.48 ± 0.65
Mean arterial pressure (mm Hg)	100.0 ± 20.0	98.5 ± 14.2	100.7 ± 21.8
Stroke index (mL/m^2)	29.7 ± 9.9	36.3 ± 9.7	31.6 ± 10.0

Data are mean ± SD.
* Different from other two categories, $p < 0.02$.

significant difference ($p < 0.0001$) among the three diagnosis groups. The HF group had a significantly higher mean BNP level (940 pg/mL) than did the other diagnosis groups (137 pg/mL, and 357 pg/mL for COPD and *other* groups, respectively).

Treatment Changes

Changes in planned medication orders, occurring after ICG information was revealed (and without other input to the treating physician), are shown in Table 5. Thirty-five patients (39%, 95% CI = 29% to 50%) had a total of 54 changes in the medication plan after initial assessment and review of ICG data. Use of diuretics most often was altered based on ICG findings, suggesting fluid overload or cardiac cause of dyspnea. When looking at medication changes by category of final diagnosis, there were 17 medication changes in the 43 patients with a final diagnosis of HF, 20 medication changes in the 20 patients with a final diagnosis of COPD, and 17 medication changes in the 26 patients in the *other* group. Twenty-three of 54 (43%) medication changes that resulted from the availability of hemodynamic information were changes in the use of diuretics or bronchodilators.

Table 5
Therapeutic Changes Post- vs. Pre-ICG Listed by Medication Class

Medication Class	n (%) [95% CI]
Diuretics	12 (13) [7%–22%]
Nitroglycerin	4 (4) [1%–11%]
Bronchodilators	11 (12) [8%–24%]
Steroids	6 (7) [3%–14%]
Antibiotics	6 (7) [3%–14%]
Anticoagulants	5 (6) [2%–13%]
Other	10 (11) [6%–20%]

There were 54 total medication changes in 35 patients. Because of multiple therapeutic changes, total changes are greater than the number of patients.

DISCUSSION

In cases of elder dyspneic patients who may require urgent treatment, the ED physician must assess status, formulate a working diagnosis, and institute therapy, in many cases before all information is available. Hemodynamic information, which reflects the contribution of the cardiovascular system to the current presentation, may have an important impact on the process of care. Our results demonstrate that knowledge of ICG data leads to a change of working primary diagnosis in 13% of elder patients presenting with dyspnea to the ED. When changes in diagnosis were made, they were consistent with the final diagnosis at time of ED disposition in two-thirds of cases. In addition to changes in diagnosis, ED physicians made medication changes on the basis of ICG-derived hemodynamic information in 39% of cases. Finally, unlike vital signs, which were similar across the various diagnostic groups, hemodynamic data varied based on causes of dyspnea. These findings are consistent with the hypothesis that hemodynamic information is relevant and actionable in the ongoing evaluation and treatment of such patients.

Patients presenting with dyspnea are commonly at risk for exacerbation of either cardiac or pulmonary disease. Those with acute HF typically have reduced cardiac output and elevated vascular resistance. Those with a pulmonary or other noncardiac cause of their dyspnea typically have normal cardiac output and hemodynamic parameters. Because the accuracy and reproducibility of ICG have been validated in a variety of patient populations and settings, it is not surprising that physicians used this information to help guide diagnosis and treatment in dyspneic patients. Our finding of different values of hemodynamic parameters among the diagnostic groups is consistent with this paradigm. The relatively high rate of change of diagnosis, when ICG-derived information was revealed to treating physicians, suggests acceptance of the technical and diagnostic accuracy efficacies of the test. Not only does the information result in altered diagnosis, but the noninvasive hemodynamic data provided by ICG was applied by the physicians to therapeutic decision making, an indication of therapeutic efficacy, as defined by Pearl.[18] Thus, our results support the potential value of such information and support a practical role for this technology in the ED assessment of such patients.

Recently, BNP testing has been shown to be a useful bedside tool to aid in diagnosis of patients presenting with shortness of breath.[26] However, despite the availability of point-of-care laboratory testing, real-time diagnosis and treatment can be delayed. In fact, one large trial of cardiac markers found that even with point-of-care testing, the door-to-brain time (the time from ED arrival until cardiac marker results are available for the physician to act upon) exceeded one hour.[27] ICG data are available within several minutes. And, unlike the hemodynamic information obtained by a pulmonary artery

catheter, performance of ICG is noninvasive and can be readily accomplished in the ED without specialized training and at minimal risk to the patient.

The magnitude of the changes in diagnosis and treatment resulting from ICG-derived hemodynamic data can be compared with that from other technologies that are currently the standard of care in most EDs. Historically, changes in therapy on the order of 5% to 11% appear to define utility of testing in the ED. In one study, Summers et al.[19] reported that the ED physician assessment of patient severity of illness was changed by pulse oximetry in 3% of cases. Kosowky et al.,[21] evaluating BNP testing in patients older than 40 years of age, found that BNP data changed the diagnosis in 10%, and treatment in 11%, of cases. These trials suggest that the rate of change that resulted from ICG use in the present study would be clinically significant in the ED environment. Moreover, ICG can be performed concurrently with existing diagnostic and therapeutic strategies, such that the information is incremental in the decision-making process.

The changes in ED decision making from esophageal Doppler results, a more invasive and less common form of cardiac output measurement, have been studied elsewhere.[28] Those investigators found a change in management decisions in 31% of cases. Our results show a greater change in therapy alone, perhaps because of the incremental information provided by systemic vascular resistance and thoracic fluid content parameters. Although most ED physicians would not subject a patient to esophageal monitoring to obtain hemodynamic measurements, it is likely that many would consider the collection of ICG data, which requires little more time or inconvenience than obtaining an electrocardiogram. We did not measure the time required to obtain ICG data; however, in routine use, these data can be obtained in about 3 to 5 minutes and require 30 to 60 seconds to interpret. Because ICG provides early and accurate data, there is a potential for significant clinical impact from its use. We did not specifically study the financial effects of ICG in this study or how it might have affected length of ED stay or hospital admission rate. However, at a procedural cost for each test of less than $20 and with the cost of a day in intensive care at more than $1,000, the provision of ICG would be cost-effective even if, for example, it reduced hospital length of stay by only one day for every 50 patients monitored.

LIMITATIONS

We acknowledge several limitations. This study evaluated the effect of ICG on working diagnosis and initial treatment plan before the results of chest radiograph, ECG, or BNP level. Thus, it is impossible to gauge the relative importance of the information obtained from ICG to that obtained by these other tests or to judge the additional contribution of ICG for cases in which the results of other tests were available before performing ICG. Although blood work and various ancillary testing such as chest radiography are part of the complete ED evaluation of such patients, the results are generally not available within the first few minutes of patient assessment. By design, this study evaluated ICG's effect on working diagnosis and therapy in a manner that would be consistent with clinical practice in the ED, where patients presenting with dyspnea might be evaluated with ICG either before or within minutes of the ED physician's initial assessment. Furthermore, as seen in our study, the findings of ECGs and chest radiographs are often normal or nonspecific and may not provide significant diagnostic certainty. Because ICG is not part of the diagnostic criteria for acute coronary syndromes, including acute MI, we did exclude patients with evidence of myocardial necrosis from analysis. Therefore, the role of ICG in providing possible clues in the evaluation of patients with dyspnea as a manifestation of MI cannot be assessed by the present study.

Our study was also limited by the use of the final ED diagnosis as the criterion standard for diagnostic categorization. Although it is possible that this diagnosis was incorrect or incomplete in some patients, this represents the real-life diagnosis based on current evaluation strategies during the patient's ED visit. It is also possible that a physician had the right diagnosis and treatment plan before reviewing ICG results and that ICG data resulted in inappropriate therapies. A larger prospective outcome-based study will be required to determine the potential for this to occur.

In our study, ICG data were available and likely contributed to the final ED diagnosis, thereby introducing possible bias. However, the goal of this study was not to assess technical accuracy of the technology, which has been evaluated in previous studies. In contrast, this study was designed to assess whether physicians would incorporate early hemodynamic information into the process of formulating an initial working diagnosis and treatment plan. In addition, the study design does not allow us to draw conclusions about the sensitivity or specificity of ICG criteria, or to compare diagnostic accuracy to other measures, such as BNP or chest radiography. The accuracy of the post-ICG diagnosis based on these hemodynamic criteria could only be verified by a more standardized diagnostic approach including cardiac imaging studies, blinded reviews of subsequent hospital records with adjudication of discordant diagnoses, and long-term follow-up, which were not within the scope of the current study.

CONCLUSIONS

Knowledge of ICG data early in the ED evaluation of patients older than 65 years of age presenting with dyspnea results in significant changes in diagnosis and treatment plan. Whether changes in diagnosis, diagnostic certainty, or therapy from ICG improve outcomes or are cost-effective will require a prospective, randomized clinical trial with longer periods of clinical follow-up.

The authors thank Gerard Smits, PhD, for his statistical assistance.

References

1. McCraig LF, Burt CW. National hospital ambulatory medical care survey: 2001 emergency department summary. Report from Centers for Disease Control

and Prevention, National Center for Health Statistics. Advance Data Vital Health Stat. 2003; 335:18.
2. Eisenberg PR, Jaffe AS, Schuster DP. Clinical evaluation compared to pulmonary artery catheterization in the hemodynamic assessment of critically ill patients. Crit Care Med. 1984; 12:549–53.
3. Speroff T, Connors AF Jr, Dawson NV. Lens model analysis of hemodynamic status in the critically ill. Med Decis Making. 1989; 9:243–52.
4. Neath SX, Lazio L, Guss DA. Utility of impedance cardiography to improve physician estimation of hemodynamic parameters in the emergency department. Congest Heart Fail. 2005; 11:17–20.
5. Van De Water JM, Dalton ML, Parish DC, Vogel RL, Beatty JC, Adeniyi SO. Cardiopulmonary assessment: is improvement needed? World J Surg. 2005; 29(Suppl 1):S95–8.
6. Sageman WS, Riffenburgh RH, Spiess BD. Equivalence of bioimpedance and thermodilution in measuring cardiac index after cardiac surgery. J Cardiothorac Vasc Anesth. 2002; 16:8–14.
7. Drazner M, Thompson B, Rosenberg P, Yancy C. Comparison of impedance cardiography with invasive hemodynamic measurements in patients with heart failure secondary to ischemic or nonischemic cardiomyopathy. Am J Cardiol. 2002; 89:993–5.
8. Van De Water JM, Miller TW. Impedance cardiography: the next vital sign technology? Chest. 2003; 123: 2028–33.
9. Yung GL, Fedullo PF, Kinninger K, Johnson FW, Channick RN. Comparison of impedance cardiography to direct Fick and thermodilution cardiac output determination in pulmonary arterial hypertension. Congest Heart Fail. 2004; 10(2 Suppl 2):7–10.
10. Albert NM, Hail MD, Li J, Young JB. Equivalence of bioimpedance and thermodilution in measuring cardiac output in hospitalized patients with advanced, decompensated chronic heart failure. Am J Crit Care. 2004; 3:469–79.
11. Silver M, Cianci P, Brennan S, Longeran-Thomas H, Ahmad F. Evaluation of impedance cardiography as an alternative to pulmonary artery catheterization in critically ill patients. Congest Heart Fail. 2004; 10(2 Suppl 2):17–21.
12. Springfield C, Sebat F, Johnson D, Lengle S, Sebat C. Utility of impedance cardiography to determine cardiac vs. noncardiac cause of dyspnea in the emergency department. Congest Heart Fail. 2004; 10(2 Suppl 2):14–6.
13. Han J, Lindsell C, Tsurov B, Storrow A. The clinical utility of impedance cardiography in diagnosing congestive heart failure in dyspneic emergency department patients [abstract]. Acad Emerg Med. 2002; 9: 439–40.
14. Barcarse E, Kazanegra R, Chen A, Chiu A, Clopton P, Maisel A. Combination of B-type natriuretic peptide levels and non-invasive hemodynamic parameters in diagnosing congestive heart failure in the emergency department. Congest Heart Fail. 2004; 10:171–6.
15. Peacock WF, Albert NM, Kies P, White RD, Emerman CL. Bioimpedance monitoring: better than chest x-ray for predicting abnormal pulmonary fluid? Congest Heart Fail. 2000; 6(2):32–5.
16. Newman RB, Pierre H, Scardo J. Thoracic fluid conductivity in peripartum women with pulmonary edema. Obstet Gynecol. 1999; 94:48–51.
17. Milzman D, Morrisey J, Pugh C, Napoli A, Gerace T, Fernandez E. Occult perfusion deficits in heart failure patients: identification through noninvasive central hemodynamic monitoring [abstract]. Crit Care Med. 1999; 27:A88.
18. Pearl WS. A hierarchical outcomes approach to test assessment. Ann Emerg Med. 1999; 33:77–84.
19. Summers R, Anders R, Woodward L, Jenkins A, Galli R. Effect of routine pulse oximetry measurements on ED triage classification. J Emerg Med. 1999; 16:5–7.
20. Mower WR, Sachs C, Nicklin EL, Safa P, Baraff LJ. Effect of routine emergency department triage pulse oximetry screening on medical management. Chest. 1995; 108:1297–302.
21. Kosowsky JM, Weiner C, Morrissey JH. Impact of B-type natriuretic peptide testing on medical decision-making for older patients with dyspnea. Ann Emerg Med. 2003; 42:S11.
22. Summers R, Schoemaker W, Peacock WF, Ander D, Coleman T. Bench to bedside series: impedance cardiography (ICG). Acad Emerg Med. 2003; 10:669–80.
23. Luepker R, Michael JR, Warbasse JR. Transthoracic electrical impedance: quantitative evaluation of a noninvasive measure of thoracic fluid volume. Am Heart J. 1973; 85:83–93.
24. Van de Water JM, Mount BE, Chandra KM, Mitchell BP, Woodruff TA, Dalton ML. TFC (thoracic fluid content): a new parameter for assessment of changes in chest fluid volume. Am Surg. 2005; 71:81–6.
25. Taler SJ, Textor SC, Augustine JE. Resistant hypertension: comparing hemodynamic management to specialist care. Hypertension. 2002; 39:982–8.
26. Maisel AS, Krishnaswamy P, Nowak RM, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. N Engl J Med. 2002; 347:161–7.
27. Peacock WF, Roe MT, Chen AY, et al. Vein-to-brain time: an emergency department quality of care marker for non-ST-segment elevation acute coronary syndrome [abstract]. Acad Emerg Med. 2004; 11:569.
28. Urrunaga J, Rivers E, Mullen M, et al. Hemodynamic assessment of the critically ill: the clinician versus esophageal Doppler monitoring (EDM) [abstract]. Acad Emerg Med. 2000; 7:587b.