Impedance Cardiography Changes Therapy in Dyspneic Emergency Department Patients: The ED-IMPACT Trial

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Introduction

Dyspnea is one of the most common Emergency Department (ED) symptoms in older patients. Multiple confounding 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, determining the cause of the current ED presentation is challenging. Hemodynamic data may offer benefit to ED clinical decision making above and beyond that obtained by the history, physical examination, and routine ED testing.

Impedance Cardiography (ICG) is a rapid noninvasive monitoring technology that provides real-time hemodynamic data. Hemodynamic information could assist in early ED decision making, but little is known about whether the addition of ICG data in the ED will change diagnosis and treatment toward improved patient outcomes. Given the high rate of morbidity, mortality, and hospital readmissions for patients with dyspnea and acute decompensated heart failure, there is an urgent need to examine ED technologies that could lead to improvements in care.

Objective

The purpose of the study was to determine the rate of diagnostic and therapeutic changes that result from adding ICG data to standard clinical evaluation in ED dyspneic patients at risk for acute decompensated heart failure.

Methods

Setting: Two urban academic EDs.

Participants: A convenience sample of patients presenting to the ED was included if they met the following criteria: 1) age 65 or greater, 2) admitted to the ED with a chief complaint of dyspnea or symptoms suggestive of heart failure, per the ED physician.

Impedance Cardiography: An independent research nurse, not involved in the diagnosis or treatment of the patient, obtained hemodynamic data using the BioZ® ICG monitor (CardioDynamics, San Diego, CA). Briefly, four dual sensors (each sensor consisting of two electrodes) are placed on the patient, as shown in Figure 1. Two sensors are placed on opposite sides of the neck at a level between the ears and shoulders; the other two sensors are placed 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 current (2.5mA, 100kHZ) and the inner electrodes detect the corresponding drop in voltage in the thorax. Pulsatile changes in blood volume and velocity are measured as impedance changes and then applied to electrocardiogram and blood pressure measurements to automatically calculate hemodynamic parameters.

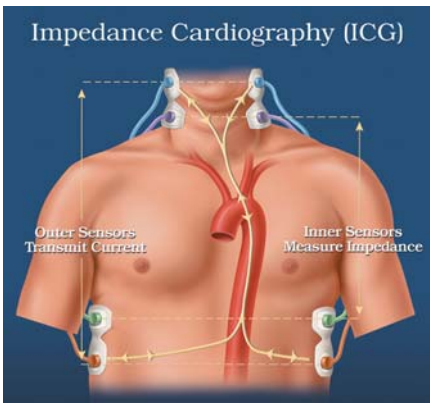


Figure 1.
ICG Method

Study Method: At the initial Emergency Department presentation, following the performance of the history and physical, but before central lab or X-ray data returned, the attending ED physician completed a case report form documenting their diagnosis, planned medication orders, and anticipated disposition. The ICG data was then unblinded, and the attending ED physician again completed another identical case report form. Pre and post-ICG case report forms were compared for differences.

Statistical Methods: Statistical analysis was performed using SAS® Software (Cary, NC). The size of the study was based on the number needed to detect a 5% rate of change in diagnosis or 10% change in therapy with 95% confidence.

Results

A total of 89 patients participated in the study. The mean age of the subjects was 74.8 ± 7.0 years (61 to 94). Baseline characteristics are defined in Table 1.

Table 1. Patient Population Characteristics, N=89

PATIENT CHARACTERISTIC	N (%)
Male	42 (47%)
Female	47 (53%)
Black	52 (58%)
White	36 (41%)
Other	1 (1%)
History of Heart Failure	43 (48%)
History of Chronic Obstructive Pulmonary Disease or Asthma	34 (38%)
History of Heart Failure and COPD	11 (12%)
History of Cardiovascular Disease	64 (72%)

The primary diagnosis changed after ICG data was shown to the ED physician in eleven (12%) of the patients. In those eleven patients, the final diagnosis was the same as the post-ICG diagnosis in seven (64%) and the pre-ICG diagnosis in four (36%). When the post-ICG and final diagnoses were categorized as either cardiac or noncardiac, the post-ICG diagnosis was the same as the final diagnosis in eight (73%).

Unblinding ICG data resulted in 54 medication changes in 35 patients (39%). In the 38 patients with a final diagnosis of congestive heart failure, ICG changed therapy in twelve (33%). In the 51 patients without a final diagnosis of congestive heart failure, post-ICG therapy differed from pre-ICG therapy in 23 (45%).

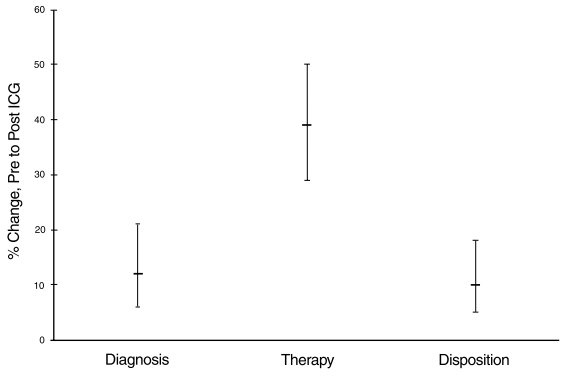
Table 2. Final Diagnosis N=89

FINAL DIAGNOSIS	N (%)
Heart failure	38 (43%)
COPD	20 (23%)
Other	14 (16%)
Atrial fibrillation	4 (5%)
Bronchitis	4 (5%)
Hypertension	2 (2%)
Pneumonia	2 (2%)
Anemia	1 (1%)
Influenza	1 (1%)
Lung cancer	1 (1%)
Palpitations	1 (1%)
Upper respiratory infection	1 (1%)

Table 3. Comparison of changes in medications from pre- to post-ICG.

MEDICATION CLASS	Post-ICG vs. pre-ICG medication changes 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%)
Totals	54

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



Discussion

ED physicians have a variety of laboratory and diagnostic tools available to assist with proper diagnosis of dyspnea. Most recently, BNP testing has become a valuable tool to aid diagnosis of emergent dyspnea.

ICG may aid in determining a cardiac versus noncardiac diagnosis for patients presenting with dyspnea. ICG may assist physicians in medication decision making for those at risk for acute decompensated heart failure.

However, even with the availability of point-of-care lab testing, real time diagnosis and treatment can be delayed due to staff availability and processing time. In these cases, the ED physician can choose to wait for additional tests before diagnosing the patient and initiating treatment, or can make a preliminary diagnosis based on history and physical and start treatment.

Unfortunately, this results in either delayed or incorrect therapy in a significant number of patients. Therefore, a real-time monitor that could aid in the initial and ongoing assessment of the patient with dyspnea would be a welcome new tool for the ED physician.

The results of this study indicate that the addition of ICG results in significant changes in the ED physician's diagnosis, treatment plan, and disposition of the ED patient with dyspnea. The use of ICG was able to change diagnosis or disposition in about one in ten patients. More significantly, ICG-derived hemodynamic information resulted in a change in therapy in four out of every ten patients.

Limitations: In a diverse patient population, with potentially many confounding diagnoses, we used the final diagnosis as the gold standard for diagnostic accuracy. It is conceivable that this diagnosis was incorrect.

In addition, we allowed the ED physician to interpret the ICG data without pre-set criteria in parameter values for the diagnosis of heart failure. It is possible that established standards for ICG-aided diagnosis could have improved the accuracy of the post-ICG diagnosis. Importantly, the study did not evaluate the effects of the changes that occurred as a result of ICG data, only that the changes occurred. It is conceivable that some changes improved patient care while others did not.

Conclusions

ICG data results in significant changes in ED physician diagnosis, therapeutic plan, and disposition of dyspneic patients 65 years and older. Based on the changes in patient management that result, ICG has the potential to improve outcomes in patients presenting to the ED with dyspnea.