Utility of Impedance Cardiography to Determine Cardiac vs. Noncardiac Cause of Dyspnea in the Emergency Department

Determining the correct diagnosis of patients with dyspnea can be challenging. Early and accurate determination of the cause of dyspnea is vital in instituting timely and appropriate interventions. Hemodynamic parameters may aid in the evaluation of dyspnea, but are difficult to assess by physical exam. Impedance cardiography (ICG) is a newly validated method of determining hemodynamic parameters noninvasively. The purpose of this study was to determine the accuracy in differentiating cardiac from noncardiac causes of dyspnea utilizing ICG-derived hemodynamic parameters compared with that of emergency department (ED) physicians after initial history, physical, and laboratory tests. The final diagnosis, which was made retrospectively after review of the patient’s hospital record by a senior ED physician blinded to the ICG data, was compared with the treating ED physician’s and the ICG diagnoses. Thirty-eight patients who presented with dyspnea to a community ED were included in the study. There were significant differences in values of cardiac index by ICG (2.2 vs. 3.1; p<0.0001), systolic time ratio (0.52 vs. 0.37; p<0.01) and velocity index (32.9 vs. 42.7; p<0.01) between the cardiac and noncardiac groups, respectively. ICG measurements demonstrated greater sensitivity (92 vs. 83%), specificity (88 vs. 77%), and positive and negative predictive values (79 vs. 63% and 96 vs. 91%, respectively) compared with the ED physician in distinguishing cardiac from noncardiac cause of dyspnea. ICG can aid ED physicians in making more rapid and accurate determinations of cardiac vs. noncardiac cause of dyspnea. (CHF. 2004;10(2 suppl 2):14–16) ©2004 CHF, Inc.

Shortness of breath, or dyspnea, is the seventh most frequent cause of emergency department (ED) visits in the United States, accounting for more than 2.5 million visits per year. To identify the appropriate therapy for patients presenting with dyspnea, a determination must be made whether symptoms are due to a cardiac or noncardiac etiology. The evaluation of such patients is often challenging, especially when they have a history of both cardiac and pulmonary disease. Patients with cardiac disease may present with an altered hemodynamic state for which treatment needs to be targeted, but obtaining hemodynamic data via pulmonary artery catheterization is associated with cost and risk and is not standard care in the ED management of such patients. Studies have demonstrated that hemodynamic data cannot be reliably estimated by physician examination. Therefore, rapid and objective hemodynamic measurements in the ED may provide significant value in clinical decision making.

Impedance cardiography (ICG) is a validated, noninvasive method of determining hemodynamic parameters. Previous studies of ICG have demonstrated utility in the differential diagnosis of dyspnea, the prediction of hospital charges and length of stay, and the ability to change the real-time diagnosis and treatment decisions in dyspneic patients.

Methods

Study Design. A prospective, blinded study was conducted to compare the ability of ICG hemodynamic measurements and ED physician diagnosis to determine cardiac or noncardiac cause of dyspnea.

Setting and Population. A convenience sample of patients presenting to the ED at a community hospital was enrolled. Patients were included if they met one or more of the following criteria: complaints of trouble breathing or shortness of breath, respiratory rate >20/min, or hypoxia (arterial oxygen concentration <90 on room air). Patients were excluded if they were <18 years of age, <3 ft 11 in or >7 ft 8 in tall, <66 lb or >341 lb, identified as trauma patients, unconscious or unable to speak, or pregnant.

Study Protocol. The Institutional Review Board approved the study protocol and informed consent was obtained for all study participants. All patients received a history and physical examination by an ED physician, as well as the following standard tests: electrolytes, kidney and liver function, complete blood count, electrocardiogram,
Table I. Summary of Hemodynamic Statistics [N=38]*

<table>
<thead>
<tr>
<th>CHARACTERISTIC</th>
<th>CARDIAC FINAL DX (n=12)</th>
<th>NONCARDIAC FINAL DX (n=26)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SI [mL/min/m²]</td>
<td>31.8 (25.3–38.4)</td>
<td>35.9 (32.9–38.9)</td>
<td>0.24</td>
</tr>
<tr>
<td>CI [L/min/m²]</td>
<td>2.2 (1.9–2.5)</td>
<td>3.1 (2.9–3.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>SVRI (dyne x s x cm² x m⁴)</td>
<td>2742 (2066–3420)</td>
<td>2106 (1903–2309)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>VI (/1000/s)</td>
<td>32.9 (25.9–39.9)</td>
<td>42.7 (38.4–47.1)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>STR</td>
<td>0.52 (0.37–0.68)</td>
<td>0.37 (0.33–0.41)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

DX=dx; SI=stroke index; CI=cardiac index; SVRI=systemic vascular resistance index; VI=velocity index; STR=systolic time ratio; *values are expressed as mean ± standard deviation.

Table II. Summary Diagnosis Statistics [N=38]

<table>
<thead>
<tr>
<th>METHOD</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICG</td>
<td>92</td>
<td>88</td>
<td>79</td>
<td>96</td>
</tr>
<tr>
<td>ED physician</td>
<td>83</td>
<td>77</td>
<td>63</td>
<td>91</td>
</tr>
</tbody>
</table>

Sensitivity=probability that a person with a true cardiac cause will test cardiac by the method; specificity=probability that a patient with a true noncardiac cause will test noncardiac by the method; PPV=positive predictive value, the probability that a person who tests cardiac is truly a cardiac cause; NPV=negative predictive value, the probability that a person who tests noncardiac is truly a noncardiac cause.

chest radiograph, and arterial blood gas. When deemed medically necessary, an echocardiogram was performed. The treating physician was blinded to noninvasive hemodynamic monitoring by ICG (BioZ ICG Monitor, CardioDynamics, San Diego, CA), which was collected on all patients. ICG utilizes four dual sensors on the neck and chest to apply a low amplitude, high frequency alternating electrical current to the patient’s thorax. Pulsatile changes in blood volume and velocity are measured as impedance changes and then applied to electrocardiogram and arterial blood pressure measurements to automatically calculate hemodynamic parameters such as cardiac output/cardiac index (CI), systemic vascular resistance index, and contractility and fluid indices.13

The ED physician used the standard tests to diagnose the patient. A research assistant monitored patient enrollment, performed all ICG monitoring tests, and documented all data points.

Data Analysis. All data were entered into a spreadsheet (Microsoft Excel, Microsoft Corp, Redmond, WA). Following discharge from the hospital, each patient’s medical record was reviewed by a board-certified ED physician who was blinded to ICG results and not involved in the treatment of any study patients. Using all data recorded in the medical record, the reviewing physician determined a final hospital diagnosis, including whether the dyspnea was of cardiac or noncardiac origin.

ICG data were evaluated retrospectively and criteria for ICG-derived cardiac cause of dyspnea were defined as either a CI ≤2.4 or systolic time ratio (STR) ≥0.55 concurrent with a CI <3.0. For each patient, final diagnosis was compared with ICG-derived and ED physician diagnosis. Continuous variables are expressed as mean ± standard deviation. Sensitivity, specificity, positive predictive value, and negative predictive value were calculated.

Results

Forty patients were enrolled in the study. Two were excluded due to an inability to obtain ICG data. No patients died during the course of the study. There were 16 men and 22 women included; the average age was 67.2±15.2 years. Twelve patients had a final diagnosis of cardiac cause of dyspnea, 26 had a noncardiac cause. Echocardiograms were deemed medically necessary in 24 patients, and results were provided in advance of ED physician diagnosis in 10 of these patients.

Patients with a cardiac cause of dyspnea had significantly (p<0.05) lower CI and velocity index, and higher STR and systemic vascular resistance index. A comparison of hemodynamic values of the cardiac and noncardiac groups is shown in Table I.

From the time of ED admission, chest radiographs were completed in 1 hour 22 minutes (±48) minutes, and ED physician diagnosis in 2 hours 57 minutes (±1 hour 14 minutes). Compared with the final diagnosis, the overall diagnostic accuracy by the ED physician was 79% (30/38) compared with 89% (34/38) for ICG. The ED physician diagnosed 10/12 patients correctly with a final diagnosis of cardiac cause, and 20/26 with noncardiac cause. ICG correctly diagnosed 11/12 patients with cardiac cause, and 23/26 with noncardiac cause. ICG demonstrated superior sensitivity, specificity, positive predictive values, and negative predictive values when compared with the ED physician in the final diagnosis of cardiac vs. noncardiac cause of dyspnea (Table II).

Discussion

Today’s ED environment challenges physicians to make decisions more quickly with greater reason to fear liability for
mistakes. Even with the laboratory and diagnostic tests available to ED physicians today, an accurate initial determination of the underlying cause of emergent dyspnea remains challenging. Therefore, both the theoretical and practical benefits of a real-time monitor that would aid in the differential diagnosis and treatment of dyspnea are clear.

Our goal was to ascertain if ICG-derived hemodynamic data could be used to distinguish between cardiac and noncardiac causes of dyspnea in an adult population presenting to the ED. In this study, retrospective evaluation with ICG was able to accurately differentiate between cardiac- and noncardiac-related cause of dyspnea with greater sensitivity, specificity, and positive and negative predictive value compared with an ED physician using conventional methods.

In this study we independently evaluated ICG data and the treating ED physician’s diagnosis. However, in clinical practice ICG would not be utilized as a stand-alone test but by an ED physician in conjunction with other available laboratory and diagnostic tests. McCullough et al. reported that the accuracy of ED physicians in diagnosing cardiac vs. noncardiac cause of dyspnea was 74% in a study of 1586 patients. While our population was significantly smaller, the diagnostic accuracy of ED physicians was similar (79%). In the study by McCullough et al., they also sought to determine if overall diagnostic accuracy could be improved by adding a screening test for B-type natriuretic peptide (BNP). The addition of BNP testing increased the overall diagnostic accuracy to 81.5%, but it was also concluded that BNP testing had little impact on medical decision making for patients who were already given a primary diagnosis of heart failure.

ICG information can be easily obtained by a nurse or cardiovascular technician in 3–5 minutes and does not require blood draw or time for laboratory analysis. This study did not compare ICG to BNP or evaluate the ability of ICG to change medical decision making. However, results from the Emergency Department Impedance Cardiography-aided Assessment Changes Therapy (ED-IMPACT) trial demonstrated that rapid provision of ICG hemodynamic information resulted in a change in treatment plan in 24% of dyspnic patients, while a similar study with BNP resulted in an 11% change. ICG measures have been shown to reliably reflect various aspects of cardiac function including blood flow (stroke volume and cardiac output), afterload (systemic vascular resistance), contractility (velocity index and STR), and changes in fluid status (thoracic fluid content). This information, available in real time, will likely allow a more targeted approach to correcting the hemodynamic abnormalities that may exist. In heart failure patients, for example, the ability to monitor cardiac output/CI or systemic vascular resistance in addition to blood pressure can identify vasconstriction that would otherwise be unrecognized. In a dyspneic patient with normal blood pressure, low CI, and high systemic vascular resistance, an ED physician may be more likely to administer a vasodilating agent to treat the underlying hemodynamic cause of the symptoms, reducing afterload and improving cardiac output.

Data from the Acute Decompensated Heart Failure (ADHERE) registry demonstrate a clear mortality benefit in early administration of nitroglycerin or nesiritide in acute decompensated heart failure, and it is quite conceivable that ICG data could help increase the utilization of these two agents.

This study’s limitations include its small sample size and retrospective criteria for ICG diagnosis. A prospective trial with a larger set of subjects is needed to establish with greater confidence which hemodynamic parameters of ICG have the greatest value in assessing patients with dyspnea in the ED.

ICG can aid physicians in the differential diagnosis of dyspnea in a more accurate, timely, and cost-effective manner. This provides a sufficient rationale to consider ICG an important and emerging new tool in the diagnosis and treatment of dyspnea.

**References**


