Measurement of Thoracic Fluid Content in Heart Failure

The Role of Impedance Cardiography

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Marjorie Funk, PhD, RN, FAHA, FAAN

Abstract

Current guidelines for assessing the fluid status of patients with heart failure include subjective physical findings, which often occur late in decompensation, and objective pulmonary artery catheter measurements, whose use is controversial in patients with heart failure. Impedance cardiography, which assesses cardiac function by measuring the opposition to an alternating electric current in the thorax, indicates the amount of fluid in the thorax. It is used externally by employing electrodes on the thorax but can also be used internally by electrodes within a cardiac pacemaker. Recent studies have suggested that one of its direct measurements, thoracic impedance, may identify patients at risk for decompensation of heart failure, but because these measurements inconsistently correlate with pulmonary artery catheter measurements and study sample sizes are small, further research is required before its use can be recommended. Keywords: heart failure, impedance cardiography, thoracic fluid

According to the American College of Cardiology and the American Heart Association 2005 guidelines, heart failure is defined as “a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. The cardinal manifestations of heart failure are dyspnea and fatigue ... and fluid retention.” Approximately 5 million patients in the United States have heart failure, and more than 550,000 patients are diagnosed with heart failure for the first time each year. The disorder is the primary reason for 12 to 15 million physician office visits and 6.5 million hospital days each year. From 1990 to 1999, the annual number of hospitalizations increased from approximately 2.4 to 3.6 million for heart failure as a primary or secondary diagnosis. In 2001, nearly 53,000 patients died from heart failure as a primary cause. Fluid retention that leads to pulmonary congestion is one of the cardinal manifestations of heart failure. Pulmonary congestion and poor organ perfusion result in dyspnea, fatigue, and activity intolerance initially but can rapidly lead to respiratory and cardiac arrest or the failure of other organs. Therefore, it is necessary to be vigilant in assessing and managing a patient’s fluid status.

The purpose of this article is to review manifestations of fluid overload, specifically thoracic fluid content and its measurement.

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with an emphasis on impedance cardiography. We then summarize and critique the published research related to the use of noninvasive and invasive direct thoracic impedance for assessing thoracic fluid content in patients with heart failure. We specifically examine the accuracy and reproducibility of direct thoracic impedance for assessing thoracic fluid content in patients with heart failure. The article concludes with implications for the use of impedance cardiography in practice.

**Fluid Overload**

Symptoms related to fluid overload that can lead to hospitalization for heart failure usually occur late in the course of a patient's decompensation. Clinical signs of alveolar congestion develop only after the interstitial fluid volume increases approximately 6-fold or more. Heart failure–related hospitalizations are frequently for fluid overload. Treatment of acute heart failure at its preclinical stage of interstitial edema may prevent its progression or alleviate its clinical impact. To treat the patient in a timely manner and prevent hospitalization, healthcare professionals must be able to recognize the preclinical phase; determination of increased thoracic fluid content is a key component at this phase.

Thoracic fluid content is composed of intravascular, intra-alveolar, and interstitial fluid within the thorax. For patients with multiple comorbidities, focusing on thoracic fluid content during a time of possible heart failure decompensation may help differentiate between cardiogenic and other causes of dyspnea. Thoracic fluid content is a subset of a patient’s overall fluid status. The American College of Cardiology and the American Heart Association 2005 guidelines for fluid status assessment include monitoring body weight changes, orthostatic blood pressures, jugular venous distension and the hepatojugular reflex, the severity of organ congestion (pulmonary rales and hepatomegaly), the magnitude of edema in the legs, abdomen, presacral area, and scrotum, and ascites in the abdomen. However, a patient’s weight is not specific to fluid gain only. Muscle atrophy or hypertrophy and adipose tissue changes cannot be easily factored into a patient’s weight change. It is difficult to differentiate thoracic fluid changes from total body weight changes. All of these benchmarks are highly subjective and therefore vary according to the healthcare professional doing the assessment. While in the hospital, these parameters are assessed daily, but at home, patients may rely only on weight changes for self-assessment of their fluid status.

Invasive hemodynamic monitoring and laboratory values add another dimension to assessing a patient’s fluid status. Arterial and central venous catheters provide numerous measurements, such as arterial blood pressure, central venous and pulmonary artery pressures, and cardiac output and index. Pro-B-type natriuretic peptide is a laboratory value sometimes used to estimate the presence and severity of a patient’s heart failure. However, current published research is inconclusive regarding the utility of these measurements for patients with heart failure. Clinical judgment based solely on these measurements is inadequate for a reliable estimate of cardiopulmonary status in critically ill patients. The risks associated with placing and maintaining central catheters also discourage their use. Therefore, the many patients without invasive devices must be evaluated using only traditional clinical methods.

**Impedance Cardiography**

Although developed in 1940, examination of the use of impedance cardiography in the clinical setting began in the 1980s. According to some researchers, it is now becoming the new standard for noninvasive hemodynamic monitoring. Impedance cardiography is used by more than 8000 physicians in the office setting and by numerous clinicians in approximately 500 hospitals and clinics throughout the United States. Although research on it is limited, impedance cardiography has been used to identify fluid accumulation in the lungs and to titrate diuretic therapy. Also known as bioimpedance monitoring, impedance cardiography assesses cardiac function by using a high-frequency, low-amplitude current to measure the resistance to the flow of the alternating electric current. Electricity travels better through fluid than through bone, tissue, or air, and less resistance or “impedance” is measured in patients in a hypervolemic state in comparison with euvoletic or hypovolemic states. Impedance cardiography measures pulsatile and baseline (also known as reference or raw) impedance. Pulsatile impedance changes are generated by variations in blood volume in the ascending
aorta. Pulsatile impedance decreases during systole as a result of increased blood volume and flow velocity and increases during diastole as flow is reduced. These pulsatile impedance changes directly reflect ascending aortic flow and therefore represent left ventricular function. Baseline impedance is a calculation of the continuous pulsatile impedance changes for a given period, providing an “average” impedance measurement of the thorax for that time.\(^9,10\) Because “baseline” describes a time that impedance is measured and the discussion of changes from one baseline to another can create confusion, baseline impedance is discussed as “thoracic impedance” in this article. When thoracic impedance and pulsatile impedance values are correlated with the patient’s electrocardiogram, the data are used to calculate various hemodynamic values, such as stroke volume and cardiac output.\(^5,10\) Changes in the patient’s thoracic impedance from hour to hour or day to day are used to assess the patient’s fluid volume in the thorax. Thoracic impedance should decrease when the patient’s lungs are “wet” as a result of worsening heart failure and increase after diuresis.\(^10\)

Impedance cardiography can be applied invasively or noninvasively. The more traditional noninvasive impedance cardiography, also known as transthoracic impedance, uses 4 or 6 external skin electrodes to measure resistance changes in the thorax. This form of impedance cardiography provides many indirect measurements, including stroke volume, cardiac output, and contractility indices, through the manipulation of direct measurements, such as volume of electrically participating tissue, impedance modulating aortic compliance, and baseline and pulsatile thoracic impedance.\(^12\)

Invasive impedance cardiography, also known as intrathoracic impedance or implantable hemodynamic monitoring, uses electrodes within a cardiac pacemaker or an implantable defibrillator. A constant current is sent through the tissue from one electrode and measured by another, resulting in a voltage measurement of thoracic impedance.\(^4\) Stored impedance data can be obtained by device interrogation in an office setting or downloaded at the patient’s home and transmitted via a standard telephone line to a secure Web site, where it can be reviewed by a clinician.\(^4\) Both noninvasive and invasive thoracic impedance have been used as a measure of thoracic fluid content.

Several impedance cardiography products are used in practice today. Some noninvasive impedance cardiography products provide direct thoracic impedance values, whereas others provide variations of direct thoracic impedance, such as thoracic fluid content values or net impedance values. Because only slight variations exist in the measurement of direct thoracic impedance and their values are comparable, studies that assess these various types of measurements are reviewed in this article. All noninvasive direct thoracic impedance and the variations have been discussed as “direct thoracic impedance.” Table 1 provides a list of noninvasive impedance cardiography devices and an explanation of their direct thoracic impedance measurements. Invasive thoracic impedance is measured as thoracic impedance only.

**Use of Impedance Cardiography in Heart Failure: What Is the Evidence?**

A large body of research exists on complications due to fluid overload in patients with heart failure as well as a smaller but substantial body of research about assessing their fluid

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**Table 1: Noninvasive Impedance Cardiography Devices**

<table>
<thead>
<tr>
<th>Device</th>
<th>Description</th>
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<tbody>
<tr>
<td>IQ (Wantagh Inc and Renaissance Technologies, currently Noninvasive Medical Technologies, Las Vegas, Nevada)</td>
<td>provides direct thoracic impedance measurements called Zo. For this product, a high Zo is supposed to indicate that the patient has low thoracic fluid content.(^22)</td>
</tr>
<tr>
<td>BioZ (CardioDynamics, RS Medical Monitoring Ltd, Jerusalem, Israel)</td>
<td>provides measurements that are the inverse of direct thoracic impedance measurements called TFC; TFC increases as impedance decreases. For this product, a high TFC is supposed to indicate that the patient has high thoracic fluid content.(^23)</td>
</tr>
<tr>
<td>RS-205 (RS Medical Monitoring Ltd, Jerusalem, Israel)</td>
<td>provides net impedance measurements that equal direct thoracic impedance minus skin-electrode impedance. For this product, a high net impedance is supposed to indicate that the patient has low thoracic fluid content.(^3)</td>
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Impedance cardiography has been examined in numerous patient populations, from hemodialysis to trauma to pulmonary patients. Within the population of patients with heart failure, the impedance cardiography measurement of cardiac output is the most studied, and there are few studies specifically focused on the direct measurement of thoracic impedance.

Current methods of assessing thoracic fluid content are subjective. Because no devices directly measure thoracic fluid content, researchers compare direct thoracic impedance with subjective measurements of fluid accumulation related to heart failure, such as lung sounds, degree of dyspnea, chest radiographs, overall fluid balance, and whether the patient required hospitalization. Comparability and reproducibility of these studies is nearly impossible due to the level of subjectivity involved. Other investigators have correlated direct thoracic impedance with objective measurements, such as pulmonary capillary wedge pressure, right atrial pressure, left ventricular diastolic pressure, and pro-B-type natriuretic peptide values. Reproducibility of this research using more objective measures for comparison is more feasible; however, whether pulmonary artery catheter values and pro-B-type natriuretic peptide values are appropriate measurements of thoracic fluid content in heart failure is debatable.

**Noninvasive Impedance Cardiography**

In studies comparing direct thoracic impedance with objective values derived from a pulmonary artery catheter, only one study showed that direct thoracic impedance correlated with a pulmonary artery catheter value (Table 2). Albert et al\(^1\) found a positive, but weak, correlation of direct thoracic impedance with left ventricular diastolic pressure. Both Funk et al\(^2\) and Drazner et al\(^3\) showed no correlation of direct thoracic impedance and pulmonary artery catheter measurements; however, left ventricular diastolic pressures were not measured. The small sample size in these studies indicates a need for a larger study examining noninvasive direct thoracic impedance in relation to all relevant pulmonary artery catheter measurements\(^4\)–\(^6\).

Most studies comparing direct thoracic impedance with heart failure symptoms showed a positive correlation (Table 3). Campos et al\(^7\) reported that the direct thoracic impedance measurement in patients with decompensated

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Model of Impedance Device</th>
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<th>Conclusions</th>
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<tbody>
<tr>
<td>Drazner et al (2002)</td>
<td>BioZ, Cardiodynamics (RS Medical Monitoring Ltd, Jerusalem, Israel)</td>
<td>50 patients with heart failure, 86% of whom had an EF of &lt; 45%, in a cardiac catheterization laboratory (10- to 30-beat measurements, averaged per patient)</td>
<td>TFC did not correlate with pulmonary capillary wedge pressure (r = 0.05, P = .71)</td>
</tr>
<tr>
<td>Albert et al (2004)</td>
<td>BioZ, Cardiodynamics (RS Medical Monitoring Ltd, Jerusalem, Israel)</td>
<td>29 patients with heart failure with an EF of &lt; 35% in an intensive care unit (30-beat measurements over 10 to 20 minutes, averaged per patient)</td>
<td>TFC correlated with left ventricular diastolic pressure (r = 0.39, P = .02)</td>
</tr>
<tr>
<td>Funk et al (2005)</td>
<td>IQ, Wantagh Inc.</td>
<td>39 patients with a mean EF of 19.6% ± 8.9% in a coronary care unit or cardiac catheterization laboratory (78 sets of measurements)</td>
<td>Zo did not correlate with right atrial pressure (r = -0.185, P = .110)</td>
</tr>
</tbody>
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Abbreviations: EF, ejection fraction; TFC, thoracic fluid content calculation by impedance cardiography; Zo, direct thoracic impedance by impedance cardiography.
heart failure (heart failure signs and symptoms at rest) indicated significantly higher thoracic fluid content than in patients with compensated heart failure (absent signs and symptoms at rest). Shochat et al,3 using the presence and severity of rales as benchmarks, also showed that direct thoracic impedance could differentiate between levels of heart failure. After diuresis, direct thoracic impedance also correlated with net fluid balance. The investigators

<table>
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<tbody>
<tr>
<td>Peacock et al (2000)</td>
<td>IQ, Renaissance Technologies</td>
<td>139 patients with dyspnea and suspected heart failure in an emergency department (measurement details not published)</td>
<td>Zo was significantly lower in patients with abnormal pulmonary fluid by CXR than in patients without pulmonary fluid by CXR (P &lt; .0002) Mean Zo of 23.4 ± 5.4 ohm for grade 0 CXR Mean Zo of 17.5 ± 5.5 ohm for grade 1 or 2 CXR Mean Zo of 17.2 ± 4.2 ohm for grade 3 CXR</td>
</tr>
<tr>
<td>Campos et al (2005)</td>
<td>BioZ, Cardio-Dynamics (RS Medical Monitoring Ltd, Jerusalem, Israel)</td>
<td>14 inpatients and outpatients with decompensated heart failure,4 dilated cardiomyopathy, and a mean EF of 24% ± 5% (measurements collected in triplicate over 15 minutes)</td>
<td>TFC of 39 ± 11 kohm⁻¹ during decompensated heart failure⁴ (P &lt; .001) TFC of 32 ± 6 kohm⁻¹ during compensated heart failure⁴ (P &lt; .001)</td>
</tr>
<tr>
<td>Shochat et al (2006)</td>
<td>RS-205, RS Medical Monitoring, Jerusalem, Israel</td>
<td>403 patients admitted with acute coronary syndrome without initial clinical signs of acute heart failure; 70 patients developed heart failure signs (measurements done every 30 minutes for 72 hours)</td>
<td>Net impedance decrease of 12% is 98% specific and 97.5% sensitive for predicting the development of rales 30 minutes before their onset Net impedance decreased by a mean of 19.9% from baseline (baseline is 58.6 ohm [95% CI = 34.4–82.8 ohm]) when rales were only at the lung bases (95% CI = 12.9%–26.9%) Net impedance decreased by a mean of 35.8% ± 7.5% after achieving fluid balance of −4278 ± 890 mL</td>
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<tr>
<td>Packer et al (2006)</td>
<td>BioZ, Cardio-Dynamics (RS Medical Monitoring, Jerusalem, Israel)</td>
<td>212 patients with chronic heart failure (NYHA II–IV) with a mean EF of 27% ± 14% (2316 measurements. Measurements done every 14 days for 26 weeks. 77 study visits preceded a heart failure event with in 14 days and 2239 study visits did not.)</td>
<td>TFC index⁴ correlated with a heart failure event⁴ within 14 days (P = .0034) TFC of 33.1 ± 9.2 kohm⁻¹ not preceding heart failure event (n = 2239) TFC of 37.4 ± 11.8 kohm⁻¹ preceding heart failure event (n = 77) (P = .002)</td>
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(continues)
concluded that direct thoracic impedance correlated with the presence and severity of rales and with diuresis. In a recent study of patients presenting to the emergency department with shortness of breath, Peacock et al compared direct thoracic impedance with the final diagnosis of heart failure. The direct thoracic impedance values in patients with heart failure indicated higher thoracic fluid content than in patients with chronic obstructive pulmonary disease. The PREDICT trial showed that direct thoracic impedance indicated that patients had higher thoracic fluid content during office visits preceding a heart failure event than office visits not preceding a heart failure event. The small sample size in most of these studies indicates a need for a larger study examining the relationship between direct thoracic impedance and heart failure symptoms. Although a larger study would be helpful, the subjectivity of grading symptoms (ie, a heart failure event occurrence) is not an ideal comparison.

Peacock et al compared direct thoracic impedance with chest radiograph results in patients presenting to the emergency department with shortness of breath and suspected heart failure. Although the purpose of this study was to differentiate among different grades of chest radiographs and correlate each grade with direct thoracic impedance, the only statistically significant result was between normal and abnormal chest radiographs, but not among the grades. They concluded that direct thoracic impedance is able to differentiate between normal and abnormal chest radiographs; however, it does not differentiate across radiograph grades. Although a larger study is necessary to have adequate power to detect associations, the subjective grades of chest radiographs are not ideal comparisons.

### Invasive Impedance Cardiography

In studies of invasive impedance cardiography, researchers analyze thoracic impedance and discuss it as a measure of thoracic fluid content. All of the published research that compared thoracic impedance with objective measures of heart failure (pulmonary catheter wedge pressure, pro-B-type natriuretic peptide, and fluid loss) showed a positive correlation. Yu et al showed that thoracic impedance trended downward, meaning an increase in thoracic fluid content, between 11 and 15 days before hospital admission for heart failure. The first occurrence of worsening heart failure symptoms was 2.5 to 3 days before admission. Thoracic impedance indicated a significant increase in thoracic fluid content 1 day before hospital admission.

### Table 3: Noninvasive Impedance Versus Subjective Clinical Measurements (continued)

<table>
<thead>
<tr>
<th>Author</th>
<th>Model of Impedance Device</th>
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<tbody>
<tr>
<td>Peacock et al (2006)</td>
<td>BioZ, CardioDynamics (RS Medical Monitoring Ltd, Jerusalem, Israel)</td>
<td>89 patients with dyspnea in an emergency department (89 measurements: final diagnosis of heart failure in 43, COPD in 20, and other in 26)</td>
<td>Patients with heart failure had higher TFC than patients without heart failure ($P &lt; .02$). Patients with heart failure had a mean TFC of 38.5 $\pm$ 12.3 kohm. Patients with COPD had a mean TFC of 30.0 $\pm$ 6.17 kohm. Patients with other diagnoses had a mean TFC of 30.4 $\pm$ 6.6 kohm.</td>
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</table>

Abbreviations: COPD, chronic obstructive pulmonary disease; CXR, chest radiograph; EF, ejection fraction; grade 0 CXR, normal pulmonary vascular distribution; grade 1 CXR, vascular redistribution and basilar vasoconstriction without radiographic evidence of pulmonary edema; grade 2 CXR, vascular redistribution and radiographic evidence of early or late interstitial pulmonary edema; grade 3 CXR, vascular redistribution and cardiogenic perihilar alveolar pulmonary edema; NYHA, New York Heart Association functional class of heart failure; TFC, thoracic fluid content calculation by impedance cardiography; $TFC = 1000 \times TFC/Direct Thoracic Impedance$; Zo, direct thoracic impedance.

1 Campos et al defined decompensated heart failure as heart failure signs and symptoms at rest.
2 Campos et al defined compensated heart failure as heart failure signs and symptoms absent at rest.
3 Net Impedance = Direct Thoracic Impedance – Skin-Electrode Impedance.
4 TFC Index = TFC/Body Mass Index.
5 Packer et al defined heart failure event as death from any cause or hospitalization or emergency department visit for worsening heart failure that required intensification of treatment.

Peacock et al defined decompensated heart failure as heart failure signs and symptoms at rest. They concluded that direct thoracic impedance correlated with the presence and severity of rales and with diuresis. In a recent study of patients presenting to the emergency department with shortness of breath, Peacock et al compared direct thoracic impedance with the final diagnosis of heart failure. The direct thoracic impedance values in patients with heart failure indicated higher thoracic fluid content than in patients with chronic obstructive pulmonary disease. The PREDICT trial showed that direct thoracic impedance indicated that patients had higher thoracic fluid content during office visits preceding a heart failure event than office visits not preceding a heart failure event. The small sample size in most of these studies indicates a need for a larger study examining the relationship between direct thoracic impedance and heart failure symptoms. Although a larger study would be helpful, the subjectivity of grading symptoms (ie, a heart failure event occurrence) is not an ideal comparison.

Peacock et al compared direct thoracic impedance with chest radiograph results in patients presenting to the emergency department with shortness of breath and suspected heart failure. Although the purpose of this study was to differentiate among different grades of chest radiographs and correlate each grade with direct thoracic impedance, the only statistically significant result was between normal and abnormal chest radiographs, but not among the grades. They concluded that direct thoracic impedance is able to differentiate between normal and abnormal chest radiographs; however, it does not differentiate across radiograph grades. Although a larger study is necessary to have adequate power to detect associations, the subjective grades of chest radiographs are not ideal comparisons.
hospital admission for heart failure exacerbation. In addition, thoracic impedance correlated with pulmonary capillary wedge pressure and net fluid loss. Vollmann et al. reported a correlation between thoracic impedance and pro-B-type natriuretic peptide. They also reported a positive correlation between thoracic impedance and heart failure symptoms. Although Abraham et al. and Knackstedt et al. concluded that as heart failure symptoms begin, thoracic impedance decreases, meaning an increase in thoracic fluid content, the published abstracts did not describe the statistical analyses. The small sample size of these studies

Table 4: Invasive Impedance Versus Objective Measurements

<table>
<thead>
<tr>
<th>Author</th>
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<tbody>
<tr>
<td>Yu et al (2005)</td>
<td>OptiVol, Medtronic (Marquis, Minneapolis, Minnesota)</td>
<td>33 patients with a history of significant heart failure (NYHA III or IV within the prior 12 months)</td>
<td>Mean impedance decreased by 12.3% ± 5.3% from baseline 1 day before admission for heart failure ($P &lt; .001$)</td>
</tr>
<tr>
<td>Vollman et al (2006)</td>
<td>OptiVol, Medtronic (Marquis, Minneapolis, Minnesota)</td>
<td>52 patients with heart failure (26 patient alerts for an impedance value of ≤ 60 ohm, 7 patients presented with decompensated heart failure without a patient alert)</td>
<td>Impedance of ≤ 60 ohm indicated heart failure with 68% sensitivity and 67% specificity Individual impedance changes inversely correlated with individual pro-B-type natriuretic peptide changes ($r = -0.49, P &lt; .001$)</td>
</tr>
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</table>

Abbreviation: NYHA, New York Heart Association functional class of heart failure.

Vollmann et al. did not define decompensated heart failure in their study.

Table 5: Invasive Impedance Versus Subjective Clinical Measurements

<table>
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<th>Measurements</th>
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</thead>
<tbody>
<tr>
<td>Abraham et al (2005)</td>
<td>OptiVol, Medtronic (Marquis, Minneapolis, Minnesota)</td>
<td>32 patients with heart failure (7 heart failure events)</td>
<td>Decreases in impedance correlates with decompensated heart failure* Increases in the difference between daily impedance and baseline impedance correlates with decompensated heart failure</td>
</tr>
<tr>
<td>Knackstedt et al (2006)</td>
<td>OptiVol, Medtronic (Marquis, Minneapolis, Minnesota)</td>
<td>42 patients with heart failure (NYHA III or IV and EF of &lt; 35%) (17 patient alerts for an unstated impedance value, 7 patients were clinically stable)</td>
<td>Impedance alert indicated signs of heart failure correctly in 59% of patients</td>
</tr>
</tbody>
</table>

Abbreviations: EF, ejection fraction; NYHA, New York Heart Association functional class of heart failure.

*Heart failure events was not defined in the study of Abraham et al.

*Decompensated heart failure was not defined in the study of Abraham et al.
indicates a need for a larger study of thoracic impedance as it relates to pulmonary catheter wedge pressure, diuresis, and heart failure symptoms. As stated earlier, the subjectivity of grading symptoms does not make an ideal comparison.

The data presented on the use of noninvasive and invasive direct thoracic impedance to assess patients' thoracic fluid content for the diagnosis of impending decompensation of heart failure show the need for further study. No devices currently exist that provide direct measurements of thoracic fluid content against which direct thoracic impedance can be objectively compared. Using pulmonary artery catheter measurements as a benchmark for direct thoracic impedance seems inappropriate because published studies remain inconclusive regarding their utility to determine thoracic fluid content in patients with heart failure. Subjectively grading chest radiographs and heart failure symptoms for comparison is also not ideal, but there are no other options.

Clinical Implications

The widespread use of impedance cardiography to determine trends in patients with heart failure throughout the course of their disease is not appropriate without statistical evidence that its measurements are accurate and reproducible. Current research does not warrant such use. Large trials comparing noninvasive and invasive direct thoracic impedance with pulmonary artery catheter values and heart failure symptoms could help determine whether a comparable objective measure exists, whether a significant correlation with symptoms exists, and whether the thoracic impedance measurements are accurate and reproducible.

Many possible scenarios exist where impedance cardiography monitoring would be beneficial if further research is supportive. Patients hospitalized with heart failure who may or may not require hemodynamic monitoring could benefit from continuous noninvasive impedance cardiography monitoring. In the outpatient setting (office, clinic, or home), patients with heart failure could benefit from impedance cardiography hemodynamic snapshots and trends over time to track heart failure progression, titrate medications, monitor medication and diet compliance, and differentiate among causes of dyspnea. A new small device is commercially available that can be used by patients at home, but no studies reporting the reproducibility or accuracy of its readings could be found. Patients with heart failure who require a pacemaker or an implantable cardiac defibrillator could benefit from the devices that have built-in impedance cardiography electrodes. This would allow for continuous hemodynamic monitoring with adjustable parameters that, when exceeded, would alert the patient to contact a healthcare professional.

It is not clear whether changing a patient's treatment plan based on thoracic impedance will improve clinical outcomes beyond that expected if healthcare professionals responded to clinical signals only. Although one study using invasive thoracic impedance values and another study using noninvasive direct thoracic impedance values showed a positive correlation with diuresis, further research is required before data from these devices can be recommended for titrating diuretics in practice.

In conclusion, these studies suggest that noninvasive and invasive direct thoracic impedance values represent a patient's thoracic fluid content and may identify patients with heart failure at risk of decompensation, but without a standard measurement of thoracic fluid content against which to compare them, they will never be validated. Noninvasive direct thoracic impedance correlates with heart failure symptoms, net fluid balance, and chest radiograph findings but not with pulmonary artery catheter measurements. Invasive thoracic impedance has been reported to correlate with heart failure symptoms, pulmonary catheter wedge pressure, pro-B-type natriuretic peptide, and fluid loss, but because of the lack of statistical detail published in the abstracts, the significance of these correlations cannot be determined. The clinical applicability of these findings needs to be tested in larger trials.

References


