Differentiation of Cardiac and Noncardiac Dyspnea Using Bioelectrical Impedance Vector Analysis (BIVA)

ANTONIO PICCOLI, MD, DSc,1 MARTA CODEGNOLTO, MD,1 VITO CIANCI, MD,2 GIANNA VETTORE, MD,2 MARTINA ZANINOTTO, PhD,3 MARIO PLEBANI, MD,3 ALAN MAISEL, MDFACC,4 AND W. FRANK PEACOCK, MD5

Padova, Italy; San Diego, California; and Cleveland, Ohio

ABSTRACT

Background: There is no gold standard for the differential diagnosis of acute dyspnea despite the usefulness of N-terminal pro–B-type natriuretic peptide (NT-proBNP) and lung ultrasound. No study has evaluated the contribution of bioelectrical impedance vector analysis (BIVA) in discriminating between cardiac and noncardiac dyspnea. We sought to determine whether a relationship exists between ultrasound detection of lung congestion, NT-proBNP, and BIVA in patients with acute dyspnea.

Methods and Results: Eligible patients were between 50 and 95 years, with an estimated glomerular filtration rate of ≥30 mL min⁻¹ 1.73 m⁻², who presented to an emergency department with dyspnea. Dyspnea was classified by reviewers blinded to BIVA as cardiac or noncardiac based on physical examination, electrocardiogram, chest X-ray, NT-proBNP, and B-lines of lung congestion on ultrasound. Overall, 315 patients were enrolled (median age 77 years, 48% male). An adjudicated diagnosis of cardiac dyspnea was established in 169 (54%). Using BIVA, vector positions below −1 SD of the Z-score of reactance were associated with peripheral congestion ($\chi^2 = 115; P < .001$). BIVA measures were reasonably accurate in discriminating cardiac and noncardiac dyspnea (69% sensitivity, 79% specificity, 80% area under the receiver operating characteristic curve).

Conclusions: In patients presenting with acute dyspnea, the combination of BIVA and lung ultrasound may provide a rapid noninvasive method to determine the cause of dyspnea. (J Cardiac Fail 2012;18:226–232)

Key Words: Congestion, heart failure, lung ultrasound, NT-proBNP.

Acute dyspnea is a common cause of emergency department admission. Although tools to assist in accurate diagnosis include natriuretic peptides (NPs) and lung ultrasound (LUS), the differential diagnosis can be challenging. The interpretation of elevated N-terminal pro–B-type natriuretic peptide (NT-proBNP) can be confounded by the presence of renal injury and obesity, therefore more objective measures of volume overload are needed. In the setting of elevated NT-proBNP, thoracic ultrasound can detect pulmonary fluid and thereby aid in diagnosis. The presence of sonographic artifacts, known as B-lines, suggests thickened interstitia of interlobular septa or fluid-filled alveoli. LUS has been shown to have greater diagnostic accuracy in differentiating the causes of acute dyspnea in emergency settings compared with the traditional methods. Its major advantages are the absence of ionizing radiation, speed, and insensitivity to the patient’s breath-hold limitations or agitation. It also allows distinction between solid and fluid lesions (consolidation vs effusion) and provides dynamic information of moving structures in real time.1–3

A newer technology, bioelectrical impedance vector analysis (BIVA) evaluates hydration status.4 Body impedance is a combination of resistance (R) (ie, the opposition to flow of an alternating current through intra- and extracellular ionic solutions) and reactance (Xc) (ie, the capacitative component of tissue interfaces, cell membranes, and organelles).4–6 With BIVA, the impedance measurements of R and Xc are normalized by the subject’s height (H)
and then expressed graphically (Fig. 1) as R/H and Xc/H on the x and y axes, respectively.4,7–9

Body impedance is 90% determined by soft tissues of limbs, so peripheral congestion is specifically detected.6,7 Only 10% is generated by the trunk, owing to its large cross-sectional area and heterogeneity of tissues and materials. Therefore the presence of pleural effusions, fluid in the lung, ascites, etc. does not contribute to the measured impedance. Whole-body bioimpedance and LUS provide complementary information: Bioimpedance cannot detect lung fluid status, and LUS (acoustic impedance) cannot detect peripheral congestion.

Using the R versus Xc graph allows the presentation of impedance vectors, the terminus of which may be presented in relation to ellipses of the 50th, 75th, and 95th percentiles of vectors derived from a normal population (Fig. 1). Vectors that project into the upper poles of the ellipses indicate decreased tissue fluid volume according to $-1,-2,$ and $-3$ dehydration ranks, and conversely, vectors that terminate in the lower poles are associated with increased tissue fluid volume according to $+1,+2,$ and $+3$ fluid accumulation ranks. The lower pole of the 75% ellipse has been identified as a threshold for apparent edema in renal patients, with 100% sensitivity and 92% specificity. Edema is not usually detectable until the interstitial fluid volume has risen to about 30% above normal (4 to 5 kg of body weight).10 BIVA can detect increased fluid volume before pitting edema is present, ie, when rank is $+1.4,5$ Vectors falling in the left halves of ellipses indicate more soft tissue mass (eg, obese and athletic subjects) compared to vectors falling in the right halves (which suggest lean and malnourished subjects).4,11–16

The purpose of the present study was to determine if there is an association between ultrasound detected lung congestion, NT-proBNP, and BIVA in patients presenting with acute dyspnea.

**Materials and Methods**

**Experimental Design**

This was a prospective observational cross-sectional study of patients presenting to the ED of Padua University Hospital with acute dyspnea. The study was approved by the local Ethics Committee. Caucasian ED patients with a chief complaint of dyspnea were eligible for inclusion if they were between 50 and 95 years of age, had a body mass index (BMI) between 15 and 40 kg/m² and estimated glomerular filtration rate (eGFR) $\geq 30$ mL min $^{-1}$ 1.73 m $^{-2}$ (calculated according to the National Kidney Foundation’s Kidney Disease Outcomes Quality Initiative recommendations17).

Patients were excluded if they were unconscious, seizing, had a presentation secondary to trauma, acute coronary syndrome, ascites, edema secondary to vein disorders, lymphedema, or hypoalbuminemia.

In the ED, after obtaining informed consent, we recorded the clinical history and physical examination. Blood samples were taken for the routine laboratory determinations. NT-proBNP was determined using the PBNP method (Dade-Behring, Newark, New Jersey) with results available in 1 hour. Electrocardiogram (ECG), chest X-ray, and LUS were also performed. The ultrasound examination consisted of bilateral scanning of the anterior and lateral chest wall, performed on the patient in a supine or near-supine position by 1 operator. A MyLab50 machine (Esaote, Genova, Italy), equipped with a convex 3.5-MHz probe, was used. According to Volpicelli et al, an examination for lung interstitial syndrome (which includes cardiogenic pulmonary congestion but cannot differentiate other conditions) is considered to be positive if $\geq 2$ positive scans per

![Fig. 1.](image-url)
side are observed when the examination is performed on 8 chest areas (2 anterior and 2 lateral per side). Each scan is considered to be positive when ≥3 close B-lines is visualized (maximum distance between adjacent B-lines of 7 mm).  

BIVA measurements were obtained with standard tetrapolar bioelectrical impedance electrodes at a frequency of 50 kHz using a phase-sensitive analyzer (BIA-101; Akern-RJL Systems, Florence, Italy) as described elsewhere.  

The 2 vector components R and Xc were recorded and divided by the subject’s height.  

BIVA results were blinded to the clinician caring for the patient. All BIVA measurements were performed by 1 operator.  

Sex-specific reference intervals were available for the Italian healthy population as 50%, 75%, and 95% tolerance ellipses on the R-Xc graph.  

The values of R and Xc in Ohm/m were transformed into bivariate Z-scores using the mean and the SD of the sex-specific reference healthy population: Z(R) = (R/H – mean R/H)/SD (ie, [R/H - 371.9]/49 if female and [R/H - 298.6]/43.2 if male) and Z(Xc) = (Xc/H – mean Xc/H)/SD (ie, [Xc/H - 34.4]/7.7, if female and [Xc/H - 30.8]/7.2, if male), thus defining one set of tolerance ellipses (50%, 75%, and 95%) independent of sex.  

Individual vectors were plotted on the Z-score graph. Mean vectors of groups were represented as point vectors with their 95% confidence ellipse on the same Z(R)-Z(Xc) plane.  

After medical evaluation and stabilization in the ED, the gold standard diagnosis of cardiac or noncardiac dyspnea was made by physicians using all of the clinical data, including physical examination, ECG, chest X-ray, NT-proBNP, and LUS, but blinded to BIVA results. European Society of Cardiology criteria were used for an HF diagnosis, which is based on the following operational definition: “The patient has symptoms typical of heart failure (breathlessness at rest or on exercise, fatigue, tiredness, ankle swelling) and signs typical of heart failure (tachycardia, tachipnea, pulmonary rales, pleural effusion, raised jugular venous pressure, peripheral edema, hepatomegaly) and objective evidence of a structural or functional abnormality of the heart at rest (cardiomegaly, third heart sound, cardiac murmurs, abnormality on the echocardiogram, raised natriuretic peptide concentration).”  

Assuming that peripheral congestion is specifically detected by BIVA and lung congestion by LUS, we defined “peripheral congestion without lung congestion” as the condition in which a patient had a negative LUS and an impedance vector below 1 SD of Z(Xc).  

The receiver operating characteristic (ROC) curve analysis on Z-scores and the calculation of common statistical tests were performed with the SPSS statistical package (v 18; Chicago, Illinois). Results of NT-proBNP (log-normal distribution) are reported as geometric means.  

Results  

Characteristics of patients are reported in Table 1. Mean age was 77 years and 48% were male. Of 315 patients, 169 (54%) received a gold standard diagnosis of cardiac dyspnea due to acute HF. Noncardiac dyspnea was diagnosed in 146 patients (46%), due to decompensated chronic obstructive pulmonary disease (58%; n = 85), asthma (30%; n = 44), anxiety (10%; n = 15), or pneumonia (3%; n = 4). Dyspnea category, NT-proBNP, LUS, BIVA results, and final clinical state are depicted in Figure 2. In the cardiac dyspnea group, the concentrations of troponin-I and NT-proBNP were higher and the impedance vector components Z(R) and Z(Xc) were significantly decreased.  

Group Mean Vectors  

In Figure 3, mean Z-scores vectors are depicted as point vectors with their 95% confidence ellipse with coordinates in SD units. Separate confidence ellipses indicate a significant difference in mean vector position.  

As shown in Figure 1, the mean vector of the group with noncardiac dyspnea (group a) was close to 0 SD of both components [Z(R) = −0.38; Z(Xc) = −0.37 SD], indicating a normal body hydration with impedance close to the mean value of the healthy population.  

In the cardiac dyspnea group without edema on physical examination (group b in Figure 3), the mean vector was displaced along the major axis and close to the lower pole of the 50% tolerance ellipse [Z(R) = −0.92; Z(Xc) = −0.86 SD] and was consistent with a mild peripheral congestion. Peripheral congestion with pitting edema on physical examination was detected by BIVA (Fig. 1). The mean vector from the cardiac dyspnea and pitting edema group (65 out of 169 patients, 38%) (group c in Fig. 3) was close to the lower pole of the 95% tolerance ellipse [Z(R) = −2.48; Z(Xc) = −1.80 SD], indicating peripheral edema.  

Individual Vectors  

Most vectors from patients with noncardiac dyspnea lay above −1 SD of Z(Xc) (79% specificity), and the majority with cardiac dyspnea were below this cutpoint (69% sensitivity). The optimal cutoff for differentiating cardiac and noncardiac dyspnea as identified by ROC curve analysis was a vector that terminated below −1 SD of Z(Xc) (sensitivity 69%, specificity 79%, area under the ROC curve 80%).  

Lung and Peripheral Congestion  

NT-proBNP mean levels were used to aid in interpreting BIVA results, based on the principle that the greater the NT-proBNP, the higher the likelihood of a HF diagnosis. Using this standard, there was a weak significant inverse correlation between the vector terminus and log NT-proBNP.
This was consistent with our finding that patients with a physical examination of “edema with pulmonary congestion” had an NT-proBNP 3 times higher than “edematous patients without lung congestion” (3,890 vs 1,318 ng/L; \( P \leq .001 \)).

Figure 2 demonstrates the combined classification of the 315 patients by clinical diagnosis, LUS, BIVA, and NT-proBNP. Among the 146 patients with a gold standard diagnosis of noncardiac dyspnea, 95% had a negative LUS. Of these, 78% had vectors exceeding the \(-1\) SD of \( Z(Xc) \) and were consistent with a non-HF state (BIVA dry). Representing the “no congestion” cohort (ie, neither lung nor peripheral), this group had the lowest NT-proBNP concentration (419 ng/L). A minority (30/139, 22%) of patients with a negative LUS had a BIVA consistent with increased fluid volume. In this group, the increase in NT-proBNP (815 ng/L) was twofold higher than the “no congestion” group, suggesting that BIVA may contribute to the evaluation of volume status above and beyond that of LUS alone.

### Cardiac Dyspnea Group

The majority (91%) of 169 patients with a gold standard diagnosis of cardiac dyspnea also had a positive LUS and the highest NT-proBNP levels (4,501 and 4,217 ng/L). Of

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**Table 1. Characteristics of Patients by Dyspnea Group**

<table>
<thead>
<tr>
<th></th>
<th>Noncardiac (n = 146)</th>
<th>Cardiac (n = 169)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SE</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Age, y</td>
<td>74.7 ± 0.85</td>
<td>79.0 ± 0.69</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>25.8 ± 0.38</td>
<td>26.3 ± 0.33</td>
</tr>
<tr>
<td>Creatinine, µmol/L</td>
<td>96.0 ± 2.92</td>
<td>111.6 ± 2.8</td>
</tr>
<tr>
<td>eGFR, mL min⁻¹ 1.73 m⁻²</td>
<td>70.3 ± 2.26</td>
<td>57.6 ± 1.84</td>
</tr>
<tr>
<td>Troponin-I, µg/L</td>
<td>0.04 ± 0.01</td>
<td>1.11 ± 0.45</td>
</tr>
<tr>
<td>NT-proBNP, ng/L</td>
<td>407.4 ± 0.06</td>
<td>3236.0 ± 0.04</td>
</tr>
<tr>
<td>Z(R)</td>
<td>−0.4 ± 0.11</td>
<td>−1.5 ± 0.10</td>
</tr>
<tr>
<td>Z(Xc)</td>
<td>−0.3 ± 0.07</td>
<td>−1.2 ± 0.06</td>
</tr>
</tbody>
</table>

**BMI**, body mass index; eGFR, estimated glomerular filtration rate; NT-proBNP, n-terminal pro-B-type natriuretic peptide.

* Student t test for unpaired data.

1The mean values of NT-proBNP are geometric means.
patients with “lung and peripheral congestion,” 71% had a vector that was below −1 SD of Z(Xc) (BIVA wet). Of patients with “lung congestion without peripheral congestion” 29% had a positive LUS and a BIVA vector exceeding −1 SD of Z(Xc) of the normal population. However, the vectors of most of these patients exceeded −1 SD of Z(Xc), because lung congestion is not detected by BIVA.

Despite a gold standard diagnosis of cardiac dyspnea, 9% of LUS examinations were negative. Of these patients, 57% had a BIVA measurement below −1 SD of Z(Xc), with an NT-proBNP that was 2 times increased versus the “no congestion” BIVA patients (from 899 to 1,809 ng/L). Overall, peripheral congestion as determined by BIVA, without ultrasound evidence of pulmonary congestion was associated with an increased NT-proBNP level 2.3 times that of patients without peripheral congestion (1,072 vs 457 ng/L; P < .007).

Renal Dysfunction

We found that there was a weak, inverse, linear correlation (r = −0.45; P = .001) between log NT-proBNP and eGFR which accounted for less than 20% of the NT-proBNP variability. As reported in Table 1, patients with a gold standard diagnosis of cardiac dyspnea had 12 mL/min/1.73 m² eGFR less than patients with noncardiac dyspnea. The difference is statistically significant but not relevant for the explanation of the higher NT-proBNP concentrations in patients with cardiac dyspnea. Furthermore, the differences in eGFR between patients with a wet versus dry BIVA pattern were not statistically significant in both cardiac and noncardiac dyspnea. In patients with “peripheral congestion without pulmonary congestion,” the eGFR was the same as in patients without peripheral congestion (67.1 vs 67.6 mL min⁻¹ 1.73 m⁻²). Patients with a physical examination of edema and with pulmonary congestion had an eGFR similar to that of edematous patients without lung congestion (61 vs 64 mL min⁻¹ 1.73 m⁻²).

Discussion

BIVA provided a threshold for discrimination between cardiac and noncardiac dyspnea with 69% sensitivity and 79% specificity. Using a BIVA threshold of 1 SD below normohydration suggests that patients with HF and clinically apparent normovolemia may have an excess of fluid causing dyspnea. In patients with dyspnea and a negative LUS, the mean NT-proBNP level was twofold higher in patients with peripheral congestion compared with those without peripheral congestion (wet vs dry BIVA pattern). Therefore, peripheral congestion without lung congestion may result in heart overload with increased (wet) NT-proBNP. When the LUS was positive, the NT-proBNP reached the maximal concentration in either BIVA pattern.

Both BIVA and LUS can be performed at the bedside and results are available almost immediately, which has the potential to improve ED decision making regarding therapeutically intervention and disposition.

It is well established that HF patients present with a variety of clinical manifestations. Though some may present with only lung congestion or only peripheral congestion, others may manifest both peripheral and lung congestion. It is recommended that patients presenting to emergency services with dyspnea should undergo a history, physical examination, chest X-ray, ECG, and blood sampling for NP and renal function measurements.

In the present analysis, we evaluated the association between BIVA and LUS as compared to a gold standard diagnosis. LUS has been validated in the general ED population (not only dyspneic patients) for lung interstitial syndrome, which includes cardiac pulmonary congestion. Obviously, LUS cannot detect peripheral congestion. BIVA operates as a stand-alone procedure using soft tissue electrical properties and is independent of body weight. BIVA is potentially superior to conventional BIA that is based on regression equations that include body weight. Indeed, conventional BIA may yield inaccurate volume estimates when tissue hydration exceeds 73%, owing to the weight of trunk fluid added to the body weight.

LUS for the detection of lung congestion was positive in 91% of patients with cardiac dyspnea and negative in 95% of patients with noncardiac dyspnea. Our findings were similar to those recently reported in the literature. Prosen et al found 100% sensitivity and 94% specificity of LUS in discriminating between cardiac versus noncardiac dyspnea in a prehospital setting. LUS was performed in 1 minute on the arrival of the patient. Because in a number of our patients LUS has been performed after some delay due to
stabilization, some LUS examination could have led to false negative results, as suggested in the literature. Dyspnea without pulmonary congestion could also have been caused by low-flow hemodynamic states. In 57% of these patients, a wet BIVA pattern was found, suggesting peripheral congestion without lung congestion. This result can be interpreted as a false positive result of the wet BIVA pattern in relation to negative LUS or as a true positive result in relation to cardiac dyspnea. NT-proBNP concentration in patients with negative LUS and dry BIVA pattern (“no congestion”) was lowest among patients with cardiac dyspnea. When LUS was positive, the NT-proBNP was at the highest concentration regardless of peripheral congestion (either dry or wet BIVA pattern).

Interestingly, 22% of patients with noncardiac dyspnea without lung congestion (negative ultrasound) had a peripheral congestion as diagnosed by BIVA (ie, a wet BIVA pattern; Fig. 2). In these patients, NT-proBNP concentration was about double than in patients without peripheral congestion. Because there is no gold standard diagnostic test for cardiac dyspnea, our results can be interpreted as either that the wet BIVA pattern was a false positive or, conversely, that it indicated “peripheral congestion without lung congestion” and was causal for NT-proBNP release. In these dyspneic patients, treatment with diuretics would be reasonable if not contraindicated by the clinical status. Unfortunately, we cannot comment on an interpretation of the BIVA pattern in the 5% of patients with a positive LUS and noncardiac dyspnea, owing to insufficient sample size (Fig. 2).

Latent Peripheral Congestion

Peripheral congestion with pitting edema is easy to identify. Peripheral congestion without pitting edema is difficult to establish, because edema is not usually detectable until the interstitial fluid volume has risen to ~30% above normal (4–5 kg of body weight).10

Fluid accumulation and removal in hemodialysis patients is a good model for validation of BIVA in peripheral congestion.15,16,21 The reference method is the ultrafiltration volume that is measured every 30 minutes with a bed scale. Changes in ultrafiltration are mapped to changes in impedance vector position. In a clinical validation study in hemodialysis patients free from edema (1,116 patients), we previously demonstrated that after ultrafiltration (mean 3 L), impedance vectors migrate from lower to upper poles of the reference tolerance ellipses according to a wet-dry cycle of impedance that is associated with a wet-dry body weight cycle. BIVA is not suitable to detect pulmonary congestion due to heterogeneity of soft tissue in the trunk and to the wide cross-sectional area of the trunk (see introductory section).

Peripheral congestion with pitting edema on physical examination was detected by BIVA (Fig. 1). The mean vector from the cardiac dyspnea and pitting edema group (group c in Fig. 3) was close to the lower pole of the 95% tolerance ellipse, indicating peripheral edema. This is also consistent with the BIVA pattern observed in patients with nephrotic syndrome, edematous peritoneal dialysis patients and those with New York Heart Association functional classes III and IV HF.4,13,21,22 This suggests that BIVA may have a role for detecting latent peripheral congestion and for monitoring fluid removal in congested patients.

Optivolemic Status

It is established that prognosis improves in patients who are able to lower their NT-proBNP levels after treatment of congestion.19 Conceptually, the NP level of a patient is composed of 2 components, that of a baseline optivolemic (“dry”) NP level and that occurring from acute pressure or a volume overload (“wet”) NP level.19 At an acute presentation to the ED, the contribution of wet and dry components of NP is frequently unknown. Our findings of elevated NP being associated with a wet BIVA pattern suggest that there may be clinical value in considering the results of these tests in combination. Furthermore, we hypothesize that a decrease in NT-proBNP from wet to dry levels is associated with a vector migration from wet to dry BIVA pattern with disappearance of B-lines in LUS. Because LUS and BIVA carry different information, we propose that the definition of the optivolemic status may be considered when there is a dry BIVA pattern together with a negative LUS. In these conditions we documented the lowest NT-proBNP concentrations.

BIVA Threshold for Cardiac Dyspnea

Others have suggested that the BIVA threshold for cardiac dyspnea Z(Xc) = −1 SD lies just on the lower pole of the 50% tolerance ellipse, where the fluid volume and central venous pressure begin to increase.14 This indicates that patients with cardiac dyspnea cannot tolerate a still-normal fluid volume. In contrast, patients without HF (eg, renal patients) with vectors between the lower poles of 50% and 75% tolerance ellipses (Fig. 1: +1 rank) are asymptomatic and their modest fluid volume increase is not clinically detectable.10,15,21,22 They develop pitting edema as soon as their vectors migrate below the lower pole of the 75% ellipse (+2 rank or more, 100% sensitivity, and 92% specificity).4,13,14,22

A study on treatment of congestive HF patients with a diuretic load documented an impedance vector displacement similar to that observed in fluid removal with hemodialysis.20 Therefore, BIVA can be properly used for detecting latent peripheral congestion and for monitoring fluid removal from congested patients. Segmental bioimpedance measurements of the trunk have been proposed to detect lung congestion. Although there was a correlation with whole-body impedance, we cast doubts about the usefulness of impedance measurements from the trunk, where very low values, particularly of the Xc component (3–6 Ohm with SD 2–3 Ohm), are meaningless owing to a small signal-to-noise ratio.24
Study Limitations

In this study, we did not evaluate the consequences of interventions. Therefore, additional studies are needed to establish whether adequacy of fluid removal with diuretics or ultrafiltration can be achieved by bringing the BIVA vector above the "wet" threshold. Also, because physicians were blinded to the BIVA data, we cannot speculate on the consequences of outcomes from interventions based solely on BIVA and ultrasound results. Furthermore, using NT-proBNP level in the adjudication may have presented biases in any of the analyses that incorporated NT-proBNP. Next, although a noninvasive strategy with minimal direct costs is likely to be cost-effective, we did not evaluate the financial implications of this strategy in our protocol. Finally, our gold standard diagnosis was defined by clinical impression rather than an external adjudication committee. Additional studies using long-term end points may provide objective outcome data for these diagnostic modalities.

Conclusion

In the present study, immediately available BIVA results provided a threshold for discrimination between cardiac and noncardiac dyspnea with 69% sensitivity and 79% specificity. Using a BIVA threshold of 1 SD below normovolemia may have an excess of fluid causing dyspnea. When LUS was negative, the mean NT-proBNP level was twofold higher in patients with peripheral congestion than in those without peripheral congestion (wet compared to dry BIVA pattern). Finally, peripheral congestion without lung congestion may result in heart overload with increased (wet) NT-proBNP release. The optimal evaluation of a dyspneic patient may include the search for peripheral congestion by BIVA and lung congestion by LUS.

Disclosures

None.

References