EM CASES SUMMARY

Episode 197 Acute Heart Failure Risk Stratification and Disposition

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Accuracy of initial evaluation findings in the diagnosis of acute heart failure

Table 5. Accuracy of Initial Evaluation Findings in Diagnosing Heart Failure

Ruling in heart failure

Finding has conclusive effect	Positive likelihood ratio > 10	Specificity
Displaced cardiac apex*	16	0.95
Third heart sound	11	0.99
Chest radiography: interstitial edema	12	0.97
Chest radiography: venous congestion	12	0.96

Finding has moderate effect	Positive likelihood ratio of 5 to 10	Specificity
History of heart failure	5.8	0.90
Hepatojugular reflex	8.4	0.96
Jugular venous distension	5.1	0.92

Finding has small effect	Positive likelihood ratio of 2 to 5	Specificity
Framingham criteria for systolic heart failure	4.57	0.79
Framingham criteria for heart failure	4.35	0.79
Framingham criteria for diastolic heart failure	4.21	0.79
Initial clinical judgment	4.4	0.86
History of myocardial infarction	3.1	0.87
Rales (crackles)	2.8	0.78
Murmur	2.6	0.90
Paroxysmal nocturnal dyspnea	2.6	0.84
Peripheral edema	2.3	0.78
Orthopnea	2.2	0.77
Elevated BNP level	2.92	0.66
Elevated N-terminal pro-BNP level	2.67	0.65
Chest radiography: cardiomegaly	3.3	0.78
Chest radiography: pleural effusion	3.2	0.92
ECG: atrial fibrillation	3.8	0.93
ECG: new T-wave change	3.0	0.92
ECG: any abnormality	2.2	0.78

Ruling out heart failure

Finding has conclusive effect	Negative likelihood ratio < 0.1	Sensitivity
Framingham criteria for systolic heart failure	0.04	0.97

Finding has moderate effect	Negative likelihood ratio of 0.1 to 0.2	Sensitivity
Framingham criteria for heart failure	0.1	0.92
Framingham criteria for diastolic heart failure	0.13	0.89
Reduced BNP level	0.1	0.94
Reduced N-terminal pro-BNP level	0.14	0.92

Finding has small effect	Negative likelihood ratio of 0.2 to 0.5	Sensitivity
Dyspnea on exertion	0.48	0.84
Chest radiography: cardiomegaly	0.33	0.97
Chest radiography: venous congestion	0.48	0.96
ECG: normal*	0.27	0.84

Note: Items are listed by the following groups: clinical criteria, history, physical examination, laboratory tests, chest radiography, and electrocardiography.
BNP = B-type natriuretic peptide; ECG = electrocardiography.
*—Findings from research evaluating only systolic heart failure.

Value of NT-pro-BNP in risk stratification of acute heart failure remains controversial

Based on our in depth review of the world's literature in 2018 in **this Journal Jam podcast**, and as detailed on **First10EM,** BNP has limited, if any, value in risk stratification of acute heart failure in the ED. However, a subsequent study and guidelines suggest that NT-pro-BNP is highly accurate at the extremes (NT-pro-BMP <300 highly unlikely acute heart failure and NT-pro-BNP ≥5,000 highly likely acute heart failure). There remains an argument to be made that in patients who obviously do *not* have acute heart failure clinically or obviously *do* have heart failure clinically, a BNP is not going to shift your diagnostic certainty significantly, and for the intermediate cases where BNP would be most valuable, it is seldom discerning, and may be misleading.
Cardiology guidelines from Europe and Canada seem to have settled on NT-pro-BNP <300pg/ml as "rule-out" threshold, while implementing an age-adjusted "rule-in" threshold:

- Rule out – <300
- Rule in ("consider admission")
  - <50y – >450
  - 50-75 – >900
  - 75+ – >1800
- Rule in ("admit, close monitoring")
  - ≥5,000

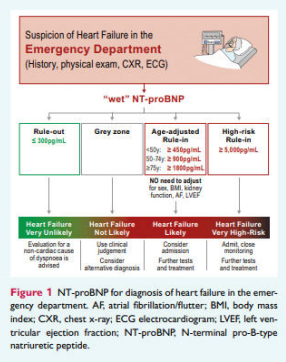
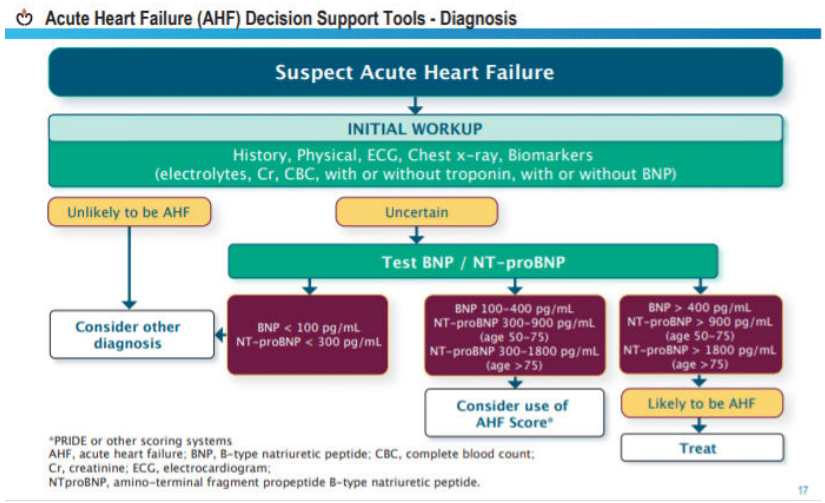


Figure 1 NT-proBNP for diagnosis of heart failure in the emergency department. AF, atrial fibrillation/flutter; BMI, body mass index; CXR, chest x-ray; ECG electrocardiogram; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide.



It is important to realize that BNP may be *elevated* by advanced age, renal dysfunction, ACS, ARDS, lung disease with right heart failure, pulmonary embolism, high output states and atrial fibrillation, and *lowered* by obesity, cardiac tamponade and pericardial constriction.

Importance of co-diagnoses and underlying causes and triggers of acute heart failure in risk stratification

The most common cause of acute heart failure decompensation is medication/dietary non-compliance on a background of known heart failure (up to 20% in North America). Despite our best efforts to identify a precipitant, about 30% may have no precipitants identified.
Important co-diagnoses and/or underlying triggers/causes to identify in the ED which help inform disposition decisions include:

- Cardiac
  - ACS
  - Dysrhthmias *atrial fibrillation with a rate that is relatively well controlled, specifically a HR < 110 can still trigger heart failure; BBB, LVH with strain and paced rhythm portend higher risk (see EHMERG score below)
  - Acute myocarditis
  - Endocarditis
  - Mechanical cause (VSD, acute MR, cardiac tamponade)
- Pulmonary
  - COPD exacerbation or asthma
  - Pulmonary embolism
  - Pneumonia
- Other
  - Hypertensive emergency
  - Thyroid dysfunction
  - Anemia
  - Cardio-renal syndrome
  - Acute aortic syndromes
  - NSAID use
  - Active cancer (high risk feature included in EHMERG score – see below)

Importance of mildly elevated troponin in informing disposition decisions in acute heart failure

About 75% of acute heart failure patients will have a high sensitivy troponin (hsTnT) value at or above the 99th percentile reference limit. While a slightly elevated hscTnT has little prognostic value in the acute heart failure patient**, a hscTnT of ≥35-45 ng/L portends a significant increase in 30 day mortality** in patients with acute heart failure, and this should help guide disposition decisions.

Outcomes and acceptable event rates of discharged patients diagnosed with acute heart failure

In the U.S., among ~1 million annual ED visits for acute heart failure, 80%–90% are hospitalized. In Canada, 40-60%. About 10% of our discharged patients will return and be admitted within 2 weeks of discharge. The 30-day mortality rate in Canada is ~4% and 23% at 1 year.
Our experts suggest that a reasonable acceptable event rate of discharged patients is a predicted risk of 0.5% or less within 7 days or 1% 30-day

mortality. For repeat ED visit rates, bounceback rates from the ED for cardiovascular causes should be 15 to 20 or less, however there is little data to back these numbers.
A recent study proposed acceptable event rates in discharged ED AHF patients:

Table 3 Proposed discharge rates and event rates in ED patients with AHF

	ED able to provide an observation frame time (%)	ED unable to provide an observation frame time (%)
Discharge rate	>40	>20
30-day mortality	<2	<1
7-day ED revisit	<10	<5
30-day ED revisit or hospital admission	<20	<15

AHF, acute heart failure; ED, emergency department.

Acute heart failure validated risk stratification tools

While risk scores may help support decision making (and in certain cases are shown to be more accurate than physician judgement of mortality – e.g. in OHFRS study), they should not be used in isolation. This is the Level B recommendation of the American College of Emergency Physicians (ACEP) – that physicians should not rely on current risk stratification tools in isolation to determine disposition for these patients. There are 3 validated ED-specific acute heart failure risk stratification tools, the Ottawa Heart Failure Risk Score, the Emergency Heart Failure Mortality Risk Grade score and the **MEESSI score**. The MEESSI score was only briefly included in our discussion as it is exceedingly complicated to calculate (as it includes a separate calculation of the **Barthel Index**) and includes vague factors such as 'low output symptoms' and thus impractical. Of note, however, it is the best predictor of 30 day mortality of the 3 scores.

Ottawa Heart Failure Risk Score (OHFRS)

*Not intended to be used to determine disposition until *after* ED intervention (in the study, OHFRS was assessed 2-8 hours after initial ED treatment)

Exclusion criteria

- Resting O_2 sat <85% on room air on normal home O_2 for >20 minutes
- Heart rate >120 on arrival
- Systolic BP <85 mmHg on arrival
- Confusion, disorientation, or dementia
- Ischemic chest pain requiring nitrates on arrival
- ST segment elevation on EKG
- Death expected within weeks from chronic illness
- Nursing home or chronic care facility resident
- On chronic hemodialysis

Items	Points
1. Initial Assessment	
a) History of stroke or TIA	(1) ___
b) History of intubation for respiratory distress	(2) ___
c) Heart rate on ED arrival ≥ 110	(2) ___
d) Room Air SaO_2 < 90% on EMS or ED arrival	(1) ___
2. Investigations	
a) ECG has acute ischemic changes	(2) ___
b) Urea ≥ 12 mmol/L	(1) ___
c) Serum CO_2 ≥ 35 mmol/L	(2) ___
d) Troponin I or T elevated to MI level	(2) ___
e) NT-ProBNP ≥ 5,000 ng/L	(1) ___
3. Walk test* after ED treatment	
a) SaO_2 < 90% on room air or usual O_2, or HR ≥ 110 during 3-minute walk test, or too ill to walk	(1) ___
Total Score (0 - 15):	____

Heart Failure Risk Categories for Serious Adverse Events within 14 days

Total Score	Risk	Category
0	2.8%	Low
1	5.1%	Medium
2	9.2%	Medium
3	15.9%	High
4	26.1%	High
5	39.8%	Very High
6	55.3%	Very High
7	69.8%	Very High
8	81.2%	Very High
9	89.0%	Very High

Figure 1. **Ottawa Heart Failure Risk Scale (OHFRS)** to identify ED patients with acute heart failure at high risk for serious adverse events. ECG = electrocardiogram; MI = myocardial infarction; OHFRS = Ottawa Heart Failure Risk Scale; TIA = transient ischemic attack.

*Can be used without NT-proBNP without sacrificing accuracy significantly – including BNP improves sensitivity while worsening specificity

Outcome

Serious Adverse Events = 30-day all cause mortality *or* within 14 days any of the following: hospital admission, NIPPV/intubation, MI, Major procedure (CABG, PCI, cardiac surgery, hemodialysis)

Scoring

Cut-off of >1 or >2 to recommend admission for monitoring/further treatment (>3 was associated with sensitivities for serious adverse events markedly worse than current practice)

- In validation study, "current practice" was as follows:
  - With BNP – Sensitivity 69.8%, Specificity 41.1% – Admission rate 60.8%
  - Without BNP – Sensitivity 71.8%, Specificity 45.5% – Admission rate 57.2%
- >1 – increases sensitivity for serious adverse events but increases admission rates
  - With BNP – Sensitivity 95.8%, Specificity 13.6% – Admission rate 88%
  - Without BNP – Sensitivity 91.8%, Specificity 24.9% – Admission rate 77.6%
- >2 – similar sensitivity to current practice with reduction in admission rates
  - With BNP – Sensitivity 79.8%, Specificity 40.5% – Admission rate 63%
  - Without BNP – Sensitivity 71.2%, Specificity 55.9% – Admission rate 48.3%

*Green – >5% better than current practice, Red – >5% worse than current practice

Criticisms of Ottawa Heart Failure Score

- May have some selection bias due to convenience sampling
- Explicitly excludes patients who are "too ill" via its many exclusion criteria
- Due to method of scoring, a patient with a history of TIA will be given the same weight as a patient with SpO2 <90%
- Similar to EHMRG, may not be as applicable to other settings due to Canada's relatively low rate of AHF admissions
- The patients studied using the Ottawa Heart Failure Risk score were healthier than heart failure patients than we see in community practice, since there was approximately 3.7% mortality at 30 days in the validation study for this score, compared to 7% in the COACH trial
- Urea is not routinely obtained for heart failure patients in many EDs
- There is a question of whether the lowest risk patients are truly low risk. Using the Ottawa HF risk score, the lowest total score is 0, but this confers a 2.8% risk of adverse events at 14 days. Is this sufficiently low risk to warrant sending a patient home directly from the ED?

Emergency Heart Failure Mortality Risk Grade score (EHMRG)

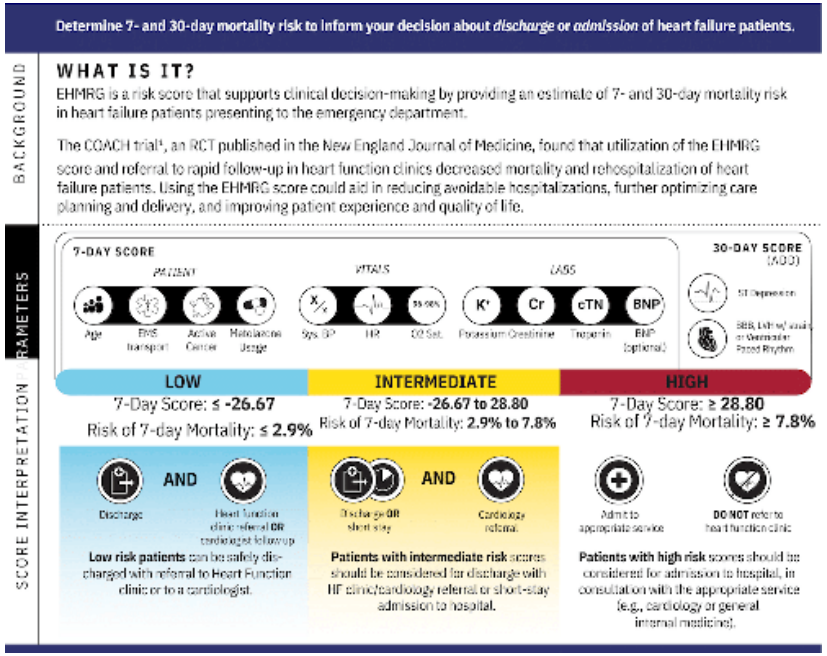
EHMRG estimates 7-day mortality of acute heart failure patients in the ED (another version of same model estimates 30-day mortality) to aid in disposition decisions.

Study design: multicenter, prospective validation study of patients with acute heart failure at 9 hospitals of previously derived EHMERG score

Definition of acute heart failure used in the study: Framingham Criteria and pragmatic independent final discharge diagnosis

Exclusion criteria

- Dialysis-dependent patients
- DNR patients
- Palliative patients
- BNP <100 or NTproBNP <300



EHMRG Score components include:

Vitals

- Systolic BP at triage (subtracted from total score – higher BP = lower score – max 160)
- HR at triage (added to total score – higher HR = higher score – min 80, max 120)
- SpO2 at triage (multiplied by 2 and subtracted from total score – higher O2 = lower score – max 92%)

Labs

- Troponin elevation (+60)
- Creatinine (divide by 88.4 to convert to mg/dL and multiply by 20)
- Potassium (<4 = +5, >4.5 = +30)

History

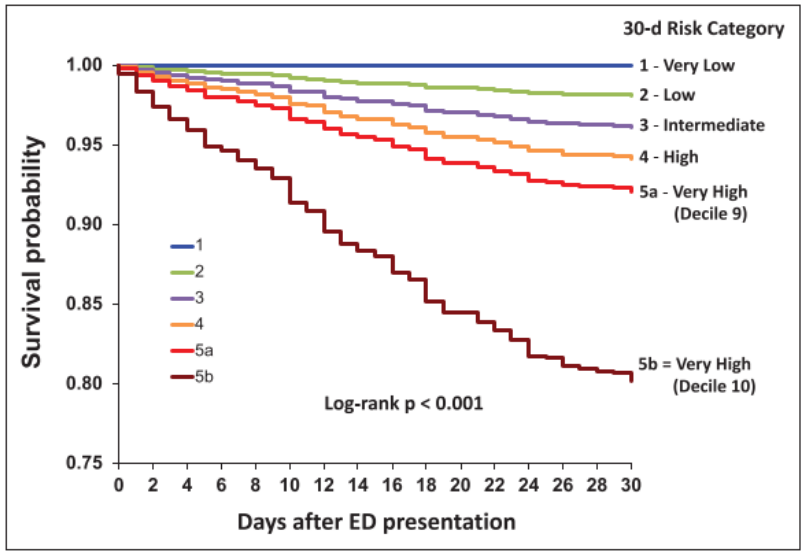
- Age (multiplied by 2 and added to total score – higher age = higher score)
- Transported by EMS to ED (+60)
- Active Cancer (+45)
- On outpatient metolazone (+60)

ECG *****_EHMRG 7-day model was extended to 30d with inclusion of additional ECG criteria of any presence of ST Depression or BBB/LVH with strain/paced rhythm_

***Metolazone** is a drug used in the setting of diuretic resistance and it's use is a significant high risk prognostic feature in acute heart failure

[EHMRG Score 7 day outcomes calculator]
[EHMRG Score 30 day outcomes calculator]

Results:



Key take home points from the EHMRG validation study

The EHMERG validation study compared using the decision tool to physician gestalt which is often missing in emergency decision tools. In the validation study, physicians *overestimated* risk in low-risk patients and *underestimated* risk in high-risk patients, so that low risk patients were

over-admitted and high risk patients were under-admitted. Physician estimated risk for predicting the outcome of a patient with heart failure is not bad, but it is also fallible because unless we use a risk score, we tend to think that healthier patients look worse than their score would predict, and the sickest patients look healthier than they actually are. This could lead to bad decisions being made in the ED, where some high risk patients could be discharged home and could die after leaving the hospital.

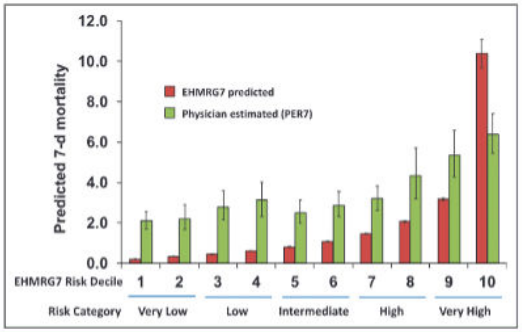


Figure 4. Physician estimated risk vs EHMRG7 risk score deciles.
EHMRG7 indicates Emergency Heart failure Mortality Risk Grade for 7-day mortality; and PER7, physician-estimated risk of 7-day mortality.

The EHMRG is very good at identifying low risk patients. In the prospective validation study, they found that the lowest two risk quintiles had 0% mortality at 7 days and 0% mortality at 30 days. Essentially, this means that the sensitivity of a low risk individual was 100%.

COACH Trial (Comparison of Outcomes and Access to Care for Heart Failure): The EHMRG Score + rapid outpatient follow-up

Rationale: Acute heart failure is responsible for a large number of admissions annually and 30-day readmission/mortality outcomes have not decreased substantially over time – one barrier to early discharge of low-risk patients is lack of access to rapid outpatient follow-up which may also contribute to readmissions.
Design: Randomized trial in Ontario, Canada of hospital-level intervention (randomized at hospital level – control and intervention phases for each hospital including academic and community sites)

Designed by same team that developed the EHMRG score – examining question of whether use of a strategy to support clinicians in making decisions about discharging or admitting patients, coupled with rapid follow-up in an outpatient clinic, would affect outcomes of HF patients
Exclusion criteria:

- No HF diagnosis (per Framingham criteria)
- Negative BNP (<100)
- End-stage disease/palliative (not eligible for EHMRG scoring)
- Unable to attend outpatient clinic visits (nursing-home, dementia, limited mobility)
- No fixed address
- LAMA

Intervention: 5452 adults presenting to ED with acute heart failure randomized to clinical judgment alone or EHMERG score to inform decisions regarding early discharge (in ≤3 days) or hospital admission. During the intervention phase, patients discharged early were referred to standardized transitional care at an outpatient clinic for 30 days; otherwise, patients received non-standardized routine follow-up after discharge.
Outcomes: The coprimary outcomes were a composite of death from any cause or hospitalization for cardiovascular causes within 30 days after presentation and the composite outcome within 20 months.
Result: absolute statistically significant difference (1-2.5%) in coprimary outcome
Composite (mortality/readmission)
30d Hazard Ratio – 0.88 (0.78-0.99) – at 30d, 12.1% (intervention) vs 14.5% (control)

- Hospitalization for heart failure – 0.81 (0.69-0.95) – 6.1% vs 8.0%
- Hospitalization for cardiovascular causes – 0.85 (0.74-0.98) – 8.1% vs 10.6%
- All-cause mortality – non-significant – 5.9% vs 6.6%
- Composite of ED visit, all-cause mortality, hospitalization – non-significant – 27.7% vs. 28.6%

Key take home points from COACH Study

- Patients in the high-risk group were much less likely to be discharged from the ED with use of the algorithm compared to physician gestalt alone

- While re-hospitalization rates were significantly improved with the algorithm, all cause mortality was *not* independently significantly changed
- The absolute primary outcome event rates were 12.5% in the intervention arm (both the risk stratification method and the rapid follow-up clinic were both utilized) and 14.5% in the control, a significant difference in the combined outcome of risk of death or CV hospitalization within 30 days.

Implementation of EHMRG score in EDs

Our experts recommend that the EHMRG score be automated in the electronic medical record so the clinician does not have to calculate it themselves.
Implementation of EHMERG score into EMR guide/checklist

The value of PoCUS in the diagnosis, risk stratification and disposition of acute heart failure patients with Ian Chernoff

Where PoCUS really shines in HF is diagnostically, and as 80% of all patients hospitalized for AHF are admitted through the ED, getting this diagnosis right really matters. The majority of the diagnostic yield from PoCUS in HF comes from Lung US alone. In general, the best PoCUS exams are those that are fast and easy to obtain, easy to interpret, and significantly impact patient care; and LungUS in HF ticks all of those boxes.

Lung Ultrasound vs CXR +/- BNP for diagnosis of Acute Heart Failure

In every single study that has compared the 2, Lung US outperforms CXR +/- BNP. Highlighting this, in a 2019 Italian ED RCT that compared the 2 head-to-head, clinical work-up + Lung US produced a whopping positive LR of 20.9 and negative LR of .07 for the diagnosis of AHF compared to LR's of 8.0 and .17 for clinical work-up + CXR+BNP, *and*, the time to diagnosis was 5 minutes for Lung US versus 104.5 minutes for CXR+BNP.

A 2015 multicenter prospective study showed that following clinical evaluation with physical exam, ECG and blood gases, using Lung US produced a positive LR of 14.1 and negative LR of 0.1 for the diagnosis of HF, versus LRs of 3.9 and 0.4 for CXR. In addition, the use of Lung US in this study led to a change in diagnosis in 19%, or 1 out of every 5, patients. A 2014 systematic review and meta-analysis showed Lung US provided a pooled positive LR of 12.4 and negative LR of .06 for the diagnosis of HF.

Furthermore, in a 2022 ACEP Clinical Policy statement on the evaluation and management of HF in the ED, the diagnostic use of Lung US in AHF was endorsed as a Level B recommendation. Lung US was the single diagnostic test for HF that there was adequate evidence for ACEP to include in this Clinical Policy Statement. This Level B recommendation endorsed "the use point-of-care lung ultrasound as an imaging modality in conjunction with medical history and physical examination to diagnose acute heart failure syndrome when diagnostic uncertainty exists as the accuracy of this diagnostic test is *sufficient* to direct clinical management". That same policy statement also examined the evidence for the therapeutic use of both nitrates and diuresis in AHF, 2 therapies that we all hold as sacred, and found it was only sufficient to justify Level C recommendations for their use.

How to utilize lung ultrasound simply and efficiently in the diagnosis of acute heart failure

How rapidly and how easily can Lung US diagnose acute heart failure? A positive Lung US for HF is defined by the presence of at least three B lines in two bilateral lung zones. We also know that B-lines first appear in the bases of the lungs then progress to involve the upper lungs as heart failure worsens. So a 2019 study asked if we could just look at the lower lung zones alone with Lung US to diagnose HF.

They compared the diagnostic yield of a 2 lung zone protocol that looked just at the lower lungs through a single probe position on either side of the chest, to a 4 lung zone protocol that examined both the upper and lower lungs bilaterally. They defined a positive Lung US as either greater than 3

B-lines per lung zone or a pleural effusion *bilaterally*. The lower lungs were examined by placing the probes laterally in each 5th intercostal space in the midaxillary lines. The upper lung zones by placing a probe anteriorly in each 2nd intercostal space in the midclavicular lines. They found that looking at the lateral zones alone provided a sensitivity of 100% and specificity of 83% for the diagnosis of AHF, and that adding the anterior zones added no significant diagnostic value. So, they showed that examining the lateral zones alone with Lung US is enough to diagnose AHF. Not only that, they showed that this could be accomplished within 30 seconds, which is less time than it takes me to log into our EMR and order a CXR.

How much pre-existing ultrasound skill do you need to be able to use Lung US to diagnose HF? I would say little to none. A 2018 paper showed that Lung US for HF could be competently done by junior medical students with little ultrasound experience, by paramedics and by nurses on cardiology wards.

Even more impressively, a 2021 paper examined the ability of patients with HF to perform Lung US on *themselves* to monitor their number of B-lines at home. The patients were provided with a 15-minute instructional video on 4-zone Lung US, then shown how to perform the exam on themselves with a handheld Butterfly ultrasound probe and an iPad. They were sent home, and their images were subsequently uploaded to a cloud to be interpreted by emergency physicians. High school was the highest level of education that 70% of these patients had attained. How successful were these patients in performing Lung US? 85% of the images they produced were adequate for interpretation, and 98% of the patients said they felt comfortable performing the technique.

So if Lung US can be learned by laypeople, there truly should be little barrier to Emergency Physicians giving it a try. In doing Lung US for HF, looking bilaterally in the 5th intercostal space, mid-axillary line should be adequate to make the diagnosis, and this should easily be able to be achieved in well under a minute.

Monitoring therapy in acute heart failure with PoCUS B-lines

B-lines change very dynamically as HF worsens or improves. In acute HF, the number of B-lines will decrease within 3 hours of initiation of treatment, such as giving a diuretic intravenously. B-lines can be used to monitor response to therapy, and have prognostic value. In one study, a high residual number of B lines at discharge identified patients with a 5-fold greater risk for HF readmission or death. In another conducted in outpatient HF clinics, high numbers of B-lines were associated with nearly a four-fold increased risk of 6-month HF rehospitalization or death.

Differential diagnosis of B-lines

Nothing is perfect, though, including Lung US for HF. There are entities other than HF that can produce diffuse pulmonary B-lines such as ARDS, diffuse pneumonia or pulmonary fibrosis. In a small proportion of patients with HF, there will be no B-lines to be found. Lung US is not a standalone test for HF, just like anything else in medicine, but a data point that needs to be incorporated into the rest of the patient's presentation. And if the diagnosis remains unclear after Lung US, there is more that can be done with PoCUS to help determine whether a patient is in AHF.

PoCUS JVP assessment for the diagnosis of acute heart failure

In keeping with the theme of easy-to-perform PoCUS applications that are high yield, the next task for PoCUS after looking at the lungs is to look at their JVP. As was mentioned in the podcast, the JVP undeniably provides useful information in HF, but also is frequently difficult to accurately identify on physical exam, regardless of the expertise of the examiner. With ultrasound, the height of the JVP can be directly visualized every time. There have been numerous ways of examining the JVP with ultrasound that have been described, but the one that I find the easiest was recently described in an article in the Annals of Internal Medicine: the patient's bed is inclined at 45°, and a high frequency ultrasound probe is placed over the sternocleidomastoid muscle just above the clavicle to identify the carotid artery and internal jugular vein in cross-section. The probe is then slid cranially until the IJ is of smaller diameter than the carotid throughout the

respiratory cycle, and where the probe is positioned on the neck to achieve this is the height of the JVP.

This study was performed on patients who were undergoing previously planned right heart catheterizations, so they were able to assess how their ultrasound-JVP correlated with Right Atrial Pressures (RAP); they found that it correlated well with an AUC of 0.84. In this same study, they first estimated the height of the JVP via physical exam prior to assessing it with ultrasound, and found that ultrasound examination of the JVP was superior. Previous studies have also shown that ultrasound examination of the JVP correlates well with RAP, including a 2018 article in the Journal of the American Heart Association. They found an elevated JVP on ultrasound correlated with elevated RAP with a PPV of 87%. Furthermore, they showed that ultrasound examination of the JVP had prognostic value – patients whose ultrasound-JVP had normalized at discharge had a 91% PPV for avoiding 30-day readmission. Similar to Lung US for HF, one of the main selling points for ultrasound of the JVP is its ease of use, and I think that it is very underutilized.

Additional PoCUS tools beyond B-lines and JVP for the diagnosis and risk stratification of acute heart failure – IVC diameter, Ejection Fraction, atrial pressures

In many ways, I think that the discussion about using PoCUS in the ED for HF should stop here, with 2 quick bedside tests that are easy to do, easy to interpret, and provide really useful clinical information; but other PoCUS tools can help with the diagnosis and treatment of HF in the ED that are worth learning.
Ultrasound of the IVC is another exam that has been incorporated into numerous PoCUS protocols to evaluate patients for HF. It's not quite as easy as looking at the lungs or JVP with ultrasound, but is of a similar level of difficulty to doing a subcostal view of the heart, and is obtained from the same window, so should be within the skill set of most Emergency Physicians. But the same patient factors that can lead to difficulty in obtaining a subcostal view of the heart – obesity, bowel gas – can similarly impede your ability to visualize the IVC.

Similar to the JVP, the IVC diameter correlates with right atrial pressure. Does looking to the IVC with ultrasound add anything to looking at the JVP in HF? There have been few studies that have compared the two in HF, but my guess is that the majority of the time, maybe not. However, a 2023 study found that in patients hospitalized with HF exacerbations, those with reduced Ejection Fraction (EF) were more likely to have elevated JVP than those with preserved EF, whereas IVC performed the same in both the reduced and preserved EF groups. This study is amongst others that show that IVC also has prognostic value in HF – patients with IVC>2.07cm on their inpatient ECHO's had a significantly elevated risk of HF rehospitalization within the next year with a HR of 2.44.

PoCUS Cases video on IVC for volume assessment with Rob Simard

What about the heart itself? Is being able to estimate an EF also a key skill for emergency physicians to possess to evaluate patients in the ER with heart failure? To determine *why* they are in heart failure, especially if it is a new or surprising diagnosis, knowing how to do a focused ECHO exam with parasternal long- and short- axis views, apical 4-chamber and subcostal views can obviously be invaluable – maybe they have a new viral cardiomyopathy with markedly reduced ejection fraction leading to their HF, or maybe they have torrential MR.
To determine *if* they are in HF, being able to estimate the EF has its limitations. First, as 50% of all HF has a preserved EF, focusing on the EF alone is going to miss all of those patients. Second, even if the EF is reduced, this may be a chronic finding, and doesn't necessarily mean that the cause of their presentation is acute heart failure.
Regardless of whether a patient presenting in AHF has a reduced or preserved EF, a common pathway to pulmonary edema developing in both groups is the development of increased left atrial (LA) pressures, so a focused heart protocol that allows for the evaluation of LA pressures should be more sensitive for diagnosing AHF than protocols that evaluate EF alone. However, being able to evaluate LA pressures requires some knowledge of how to perform diastolic measurements, and admittedly, learning how to do this may not be the highest educational priority for many emergency physicians.

If you're an Emergency Physician whose ultrasound skills are such that you already feel comfortable with Lung US, JVP and IVC assessment, and are able to reliably generate all the basic views of the heart including an apical 4-chamber view, dabbling with diastology may be a great place to spread your wings.
A 2017 study developed a rapid cardiothoracic ultrasound protocol for the diagnosis of heart failure in the ED. This protocol involved doing 4-zone Lung US, plus measuring LA pressure using a E/e' ratio. An E/e' ratio involves doing 2 separate PW Doppler measurements via an apical 4-chamber view. The investigator in this study, admittedly an experienced one, was able to do a 4-zone Lung US plus generate an E/e' ratio within 3 minutes, and this protocol had a sensitivity of 100% and specificity of 95.8% for the diagnosis of AHF. This study also found the incidence of reduced EF was actually higher in the *non-HF* group at 52%, versus 42% in the HF group.This difference was not statistically significant, but highlights my earlier point about the limitations of EF in ED PoCUS protocols for the diagnosis of HF.

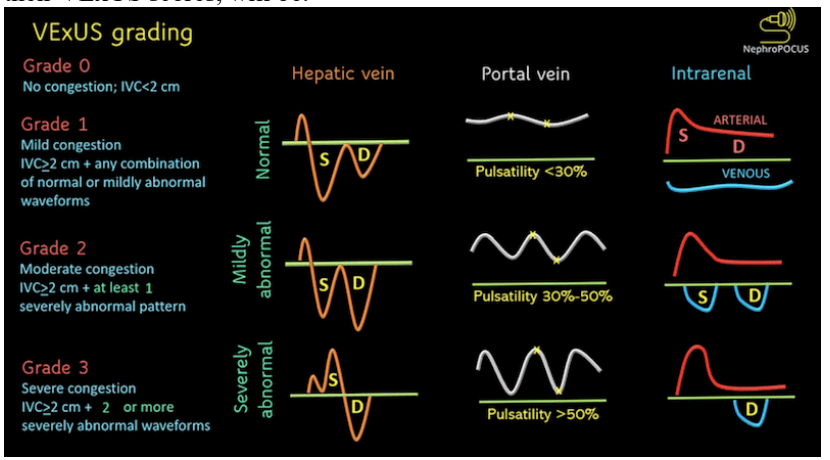
PoCUS Cases video on LV Dysfunction with Rob Simard

The role of PoCUS in assessing venous congestion, guiding management of cardiorenal syndrome and prognosis of acute heart failure – The VEXUS score

The last thing that I wanted to touch on was the evaluation of venous congestion with PoCUS. Dr. Lee talked about the dilemma of renal venous congestion in HF management and cardiorenal syndrome in this podcast. When the creatinine begins to climb in patients with HF who are being managed with diuretics, the knee jerk way of thinking of this previously has been that these patients might be receiving too much diuresis, are too dry, and their kidneys aren't seeing enough fluid. However, we now know that this is not always the case, and it may be that their declining renal function is instead due to renal venous congestion from under-treated HF. Such patients need *more* diuresis to decongest their kidneys to improve their kidney function, not *less.* Dr. Lee mentioned that one way to show if renal venous congestion is the cause of a rising Creatinine in cardiorenal syndrome is to be more aggressive with the diuresis of these patients, and in in whom renal venous congestion is the underlying cause, their Creatinine will begin to improve with such therapy.

PoCUS also provides a mechanism for evaluating for venous congestion in HF patients through a protocol called VExUS, or the Venous Excess Ultrasound score. VExUS was first described in a post-op cardiac surgery population, and although the PoCUS skills needed to do VExUS are a bit more complex, the principles underlying it are reasonably straightforward. As patients with HF become more fluid-overloaded, venous congestion in organs such as their liver and kidneys increases. As venous congestion gets worse, it alters the way blood flows through the veins of these organs in a predictable way, and these alterations in flow mechanics can be detected with PoCUS by assessing the IVC, then using PW Doppler to interrogate venous flow patterns in the renal veins of the kidney, and the hepatic and portal veins of the liver.

PW Doppler of the renal, portal and hepatic veins can reveal normal flow patterns, or as venous congestion increases, either mildly abnormal or severely abnormal patterns. The more severe the venous congestion in patients with HF, the more abnormal their flow patterns, and thus the higher their VExUS scores, will be.



Source: NephroPOCUS

VExUS is relatively new, and reasonably complicated, but it has been met with relatively wild enthusiasm amongst Critical Care, Nephrology and Emergency Medicine PoCUS enthusiasts for the promise it seems to hold for better evaluating the overall fluid status of a patient with ultrasound. So in addition to its use in HF, perhaps VExUS will be able to help guide fluid management decisions in resuscitation, for instance. And although it may not yet be within the reach of the average Emergency Physician, I think VExUS is something that we are all going to be hearing more and more about over time.

As it pertains to the matter at hand, the use of VExUS was shown by a 2023 Spanish study to potentially yield important prognostic information in the management of patients with HF. This was a small study, but of a sick population, 14% of whom died within the course of hospital stay and 26% within 90 days. This study showed that patients with higher levels of venous congestion at admission documented by VExUS had significantly higher rates of inpatient mortality. For instance, a severely abnormal renal VExUS pattern predicted inpatient mortality with a sensitivity of 90%, specificity of 81%, positive predictive value of 43%, and negative predictive value of 98%. Furthermore, when these patients had a VExUS exam repeated in an outpatient clinic in follow-up, the severely abnormal renal VExUS pattern predicted need for HF readmission with a sensitivity 92% and a specificity of 67%, and, a distended IVC in outpatient follow-up also predicted need for readmission.

Take home points for the role of PoCUS in the diagnosis, risk stratification and disposition of acute heart failure patients

- The test that has the single best characteristics for the diagnosis of HF, PoCUS or otherwise, is Lung US. As a starting point for Emergency Physicians who want to incorporate PoCUS into their evaluation of patients with HF, simply taking 30 seconds to look bilaterally in their 5th intercostal spaces, mid-axillary lines, for B-lines is going to markedly improve your diagnostic accuracy in HF.
- If you take an additional minute or two to look at their JVP with ultrasound, you'll get even more diagnostic clarity.
- For emergency physicians who are already proficient in generating basic cardiac views, and want to grow their PoCUS skill set, consider learning how to evaluate LA pressures with elements of diastology. This is less complicated than it sounds, and can yield dynamic information as to how a patient is responding to diuresis.
- The number of B-lines found on LUS, a persistently elevated JVP distended IVC, and the demonstration of higher grades of venous congestion with VExUS have all been shown to have prognostic value in HF patients, and it seems very likely that we will see the inclusion of many of these PoCUS measures in future HF risk stratification scores.
- Future research in acute heart failure decision tools should incorporate PoCUS

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