Heart failure can be confusing and nebulous, so we will do our best to break this down into the key definitions and clinical pearls you'll need when seeing heart failure in clinic.

A lot of today's information will be coming from the Canadian cardiovascular society. They have comprehensive guidelines from 2017 and an update in 2021. They have a very useful app called iCCS with the most recent HF guidelines and an algorithm for the diagnosis of heart failure in the ambulatory care setting.

Heart failure is Just as it sounds! The heart is not pumping as effectively as it should, resulting in insufficient oxygen delivery to meet the body's needs. It's typically classified and treated according to ejection fraction.

That is, the amount of blood that is pumped out of the left ventricle during systole and reported as a percentage. A normal ejection fraction is 50-70% remembering that even a healthy heart doesn't pump out 100% of blood! Of course, the lower the number, the worse the heart failure is. The lowest I've seen is 15%!!

Heart failure is classified as either preserved ejection fraction or "HFpEF", sometimes also called "diastolic HF" with EF greater than 50%. This accounts for about ⅓ of HF patients.

The other type is heart failure with reduced ejection fraction, "HFrEF", or also called "systolic heart failure" with an EF of less than 40% but can go up to 49%, making up ⅔ of patients.

Just to further confuse matters, in recent years a new category called " mid-range" or HFmrEF has been proposed that is defined as EF 41-49%. It is unclear if this truly represents a distinct clinical entity and research is ongoing to better characterize this group. However, it is referenced in some guidelines.

1. **In patients with newly diagnosed heart failure determine the underlying cause, as treatment will differ.**
2. **In an older patient presenting with fatigue, include heart failure in your differential diagnosis.**
3. **In a patient with symptoms suggestive of heart failure and a normal ejection fraction do not exclude this diagnosis.**

The symptoms of heart failure, and possible patient presentations, include:

- progressive breathlessness,
- fatigue,

- leg swelling,
- orthopnea,
- paroxysmal nocturnal dyspnea (highest likelihood ratio for HF!, usually occurs 1-2 hours after falling asleep, relieved with sitting or standing up),
- generalized weight gain, as well as
- confusion + fatigue especially in the elderly.

There are a number of signs that we can look for on our exam. These include:

- decreased air entry to the lung bases,
- lung crackles,
- elevated JVP with positive abdominal jugular reflux,
- a displaced Apex,
- presence of 3rd (Highest likelihood ratio) or
- 4th heart sound,
- a heart murmur,
- low blood pressure, and
- a heart rate greater than 100 BPM.

If you are POCUS savvy you may even notice a pleural effusion or two.

The most common causes of heart failure include tachyarrhytmias, valvular disease, coronary artery disease (ischemic heart disease) and left ventricular hypertrophy (HTN).

Ischemic cardiomyopathy (⅔ HFrEF)
Nonischemic CM (⅓ HFrEF)

- Load - HTN, valvulopathy
- Arrhythmia
- Myocardium (numerous causes). A mnemonic I find useful is TIMINGS:
  - T - Toxic - EtOH, cocaine, radiation, chemotherapy (anthracyclines, trastuzumab, 5-FU)
  - I - infiltrative (amyloid, sarcoid, hemochromatosis, etc)
  - M - metabolic
    - Endocrine - thyroid, pheochromocytoma
    - Nutritional - thiamine, selenium
  - I - Inflammatory
    - Infectious - HIV, viral myocarditis
    - Autoimmune - GCA, RA, SLE
  - G - genetic (HOCM, muscular dystrophy, hemochromatosis, ARVC)
  - S - stress (Takutsubo, sepsis)

If a patient presents in new heart failure, you need to ask yourself two questions:

1. What is the underlying cause of the heart failure (i.e. the numerous causes listed above) and

2. What is causing this particular exacerbation of heart failure? We will address this in point #5 → we address this in point #5, would suggest just sticking to diagnosing HF + determine cause. These causes might be the same (e.g. atrial fibrillation with RVR) or different (e.g. Ischemic heart disease causing HFrEF with an exacerbation secondary to missing a week of furosemide).

Your initial heart failure workup should include ruling out the most common etiologies. Be sure to order an ECG (to assess for ischemia or arrhythmia), CXR (to support a diagnosis of heart failure + assess for cardiomegaly), and blood work including - CBC, electrolytes, Creatinine Glucose, troponin, + TSH as your initial work up.

If you're suspecting some of these other causes be sure to order tests to specifically rule those out. This may include a ferritin, or formal/ point of care ultrasound for Pericardial disease, or other bloodwork like an hgA1C

On ECG we might see q waves, signs of LVH, or a left bundle branch block. We may see tachycardia or atrial fibrillation.

Chest X-ray may reveal cardiomegaly, pulmonary Venous redistribution, pulmonary edema or plural effusion.

If after our physical exam and initial workup we're still not sure, we can look to the BNP for some additional guidance

To strictly follow the CCS guidelines here, if BNP >50pg/mL or NT proBNP >125 pg/mL they say an echo SHOULD be ordered

Is the thresholds differ for BNP versus NT pro BNP.

For a simple BNP: less than 100pg/ml means heart failure is unlikely, 100 to 400 is possible and greater than 400 implies heart failure is likely.

If we look at the NT pro BNP we see that less than 300 pg/ml is unlikely to be heart failure.

For 50 to 75 year olds 300 to 900 suggests heart failure is possible but it's likely if over 900. For those over 75, the range of 300 to 1800 means heart failure is possible and over 1800 heart failure is likely.

OF note, BNP can be difficult to interpret in renal failure (i.e. artificially elevated) so interpret these with caution/

An echocardiogram will of course tell us whether the left ventricular ejection fraction is diminished, as well as look at wall motion abnormalities (suggestive of ischemia), impaired diastolic function, left ventricular hypertrophy, valvular dysfunction and elevated pulmonary artery pressure.

4. **In patients with heart failure periodically assess functional impairment using validated tools (e.g., New York Heart Association class, activities of daily living).**

NYHA classification Can be useful here to gauge how well a patient is doing and their response to treatment.

This is a 4 level classification scale.

Class 1 involves no limitations of physical activity and ordinary activity does not cause fatigue, palpitation or dyspnea .

Class 2 includes slight limitations to physical activity where the patient is comfortable at rest, but ordinary activity results in fatigue, palpitations or dyspnea.

Class 3 includes marked limitations to activity; however the patient is still comfortable at rest.

Class 4 includes symptoms at rest and the inability to carry out any physical activity without discomfort or symptoms.

Class I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation or dyspnoea.
Class II	Slight limitation of physical activity. Comfortable at rest but ordinary physical activity results in fatigue, palpitation or dyspnoea.
Class III	Marked limitation of physical activity. Comfortable at rest but less than ordinary activity results in fatigue, palpitation or dyspnoea.
Class IV	Unable to carry out any physical activity without discomfort. Symptoms at rest. If any physical activity is undertaken, discomfort is increased.

NYHA: New York Heart Association.

The Canadian cardiovascular society suggests follow up intervals for the different classifications. For class 1 or 2 they suggest follow up every 6 to 12 months.

Class 4 heart failure should be followed up as frequently as every 1 to 4 weeks as needed.

For individuals with class 3 symptoms follow up should be tailored to the patient and their stability, As often as every couple weeks to as little as every 6 months.

Other more comprehensive questionnaires, such as the “Kansas City Cardiomyopathy questionnaire” can be used to have a better bird eye’s view of how your patient is doing in day-to-day life.

5. **To guide your management of a patient with an exacerbation of heart failure:**
    a. **Identify possible triggers (e.g., infection, arrhythmia, adherence, diet, ischemia)**

    b. **Consider comorbid conditions (e.g., renal failure)**

This really goes back to those first objectives and that long list of possible causes and triggers. Anytime you see a patient’s heart failure worsening, whether it’s acute or indolent, go through the list and ensure each of those conditions is either ruled out, or optimized. Then ask yourself whether there is an interplay with another condition like renal failure, pulmonary hypertension, or liver failure.

A Mnemonic I like to use to assess for triggers of exacerbation is “Failures”

- F - forgot to take meds (VERY COMMON!) can address reasons for non-adherence, for example cost issues (i.e. newer medications like entresto can be expensive)
- A- Arrhythmia, like AF - either new or itself exacerbated (remember our PIRATES mnemonic in episode 7? Self plug ;))
- I - ischemia or infarction
- L - lifestyle choices - high salt intake (side note, recent study SODIUM- HF published in the Lancet in 2022 consuming 2 g of salt has similar outcomes to further restriction down to 1.6 g, be kind to your patients!), alcohol, excessive fluid intake, obesity
- U - upregulation (pregnancy, hyperthyroidism)
- R - renal failure (acute, progression of CKD, missed hemodialysis)
- E - embolus (PE)
- S - stenosis (worsening)

These can all exacerbate heart failure, and will have specific, and sometimes more complicated, treatment plans. Specialist advice is warranted in these cases, if not already sought.

6. **When treating heart failure:**
    a. **Identify the type of heart failure (e.g., systolic, diastolic) because the treatment is different**

    b. **Appropriately prescribe medications to reduce mortality as well as treat the symptoms of congestive failure (e.g., diuretics, beta-blockers, ACE inhibitors, digoxin)**

This is where that echo becomes extremely useful.

For heart failure with reduced ejection fraction
we're looking at ARNI or Acei/arb medications, Beta blockers, aldosterone receptor antagonists, SGLT2 inhibitors

The ARNI or Angiotensin Receptor-Neprilysin Inhibitor available for use currently in Canada is sacubitril-Valsartan combination. Any of the ACEI or ARB medications are fine if an ARNI is not an option.

The aldosterone receptor antagonists include spironolactone and eplerenone.

Diuretics like furosemide can be used to relieve congestion and should be titrated to a minimum effective dose to maintain euvolemia. However, these have not been shown to affect mortality.

Be sure to monitor renal function and electrolytes, and replace as necessary (I'm looking at your potassium!)

The beta blockers of choice here are carvedilol, bisoprolol or metoprolol extended release, as these are the ones shown to have benefit in randomized trials.

With respect to heart rate: If the heart rate is still greater than 70 and sinus rhythm, ivabradine could be considered. If sub optimal rate control for atrial fibrillation then consider digoxin.

For HFpEF
less evidence is available, can consider starting spironolactone as it has been shown to decrease hospitalizations, and the recent EMPEROR-PRESERVED trial showed primarily decreased hospitalizations with an SGLT2 inhibitor.

Of course it is important to treat other comorbidities including their diabetes, atrial fibrillation, renal failure, COPD, etc..

7. **For patients with heart failure ensure you offer patient education and self-monitoring, such as routine self-weighing, healthy diet, medication adherence, smoking cessation, and exercise, to minimize exacerbations.**

   As mentioned above, new study shows sodium intake of 1,600 mg is similar to 2,000 mg in terms of outcomes, so don't need to severely restriction, but should monitor. I also like to ask patients not how much salt they use (because everyone always says, oh doc, i NEVER put salt on my food …) but rather how much take out they eat each week. Progessed food is the largest contributor of salt to the diet. Self-monitoring of weight is important as well, because often first sign things are going awry, and can offer a change to avoid hospitalization if increase diuretics and address why they are leaning toward an exacerbation!

8. **In a patient with heart failure recognize non-sustained response to treatment as an indicator of worsening prognosis.**

For patients with worsening condition such as NYHA class 3 or 4, consider referral for advanced heart failure therapy including mechanical circulatory support or transplant.

9. **In a patient with heart failure and a progressively deteriorating clinical course:**
    a. **Provide a realistic prognosis to patients and families**

    b. **Introduce palliative care principles when appropriate for the patient**

Broaching the subject of palliative care can be challenging with some patients and their families, so go back and review Episode 10 titled Bad News for an approach to this.
Also be aware, the prognosis for heart failure can be worse than some cancers! Patient's don't often appreciate this and can be shocked to learn heart failure is a life limiting condition. I've usually heart all comers about 50% survival rate at 5 years.

Medications that can be helpful include:

- PRN furosemide for symptom control, have even seen lasix infusion for end of life patients!
- Opioids for dyspnea
- ……

We're done with the objectives, now let's talk about donkeys!
This is an awesome analogy that I've heard and seen in a few places, but don't know where it originated, so our apologies if this was your invention! Some of this will apply more to acute exacerbations of congestive heart failure, but it should still be illustrative overall.

The heart is a muscle that obviously works very hard. It pumps blood from an area of lower pressure, the venous and pulmonary systems, to an area of higher pressure, the systemic system. Let's compare this to a donkey that needs to move a pile of sandbags up a hill.

If the donkey goes too quickly or works too hard it might get tired or injured, if it goes too slowly then those sandbags are going to start piling up at the bottom of the hill as they're made. See where this is going?

Now how can we help this poor donkey out? We could lower the height of the hill, maybe there's a better destination lower down – that's like reducing afterload on the heart. What can we use to do that?

To lower afterload we can use antihypertensives like ACEi and ARBs and hydralazine. As well, the new ARNI will have an effect here.

Ok, but how about starting up the hill closer to where we want to end up? In our donkey analogy we can improve our starting position by reducing the number of sandbags it needs to carry. For the heart that would be similar to optimizing pre-load.

I'm sure you all remember the Frank-Starling curve of cardiodynamics, and you'll also recall that many of these heart failure folks will be sitting in that far right area where more preload actually decreases cardiac output. (i.e the heart is already stretched, so stretching it further doesn't increase contractility)

So how do we reduce preload? We can use nitrates or diuretics like furosemide or spironolactone, or the ARNI again comes up here.

Alright so the terrain is optimized, we can't make this hill any flatter. Now what, our donkey still isn't moving enough sand-bags up the hill?
We can improve the efficiency of the cart for this donkey so it can slow down and relax a bit. For the heart, that corresponds to using beta-blockers, calcium channel blockers and digoxin to slow the heart rate, encourage positive cardiac remodeling, and improve contraction in the case of digoxin. SGLT2i would also fit here as they're thought to improve contraction, somehow, we still don't know.

But what if I need the donkey to bring those sandbags right away because they're all at the bottom of the hill and a low-pressure system is about to flood my house?

You mean like in an acute exacerbation of heart failure resulting in hypotension and pulmonary edema? Well, we could try to reward the donkey with a carrot, but this is a very intrinsically motivated donkey, so only a stick will do.

Right, we could beat our donkey, figuratively speaking, with an infusion of epinephrine, dobutamine or milrinone. Of course this isn't a good long term solution, but it might help weather the current storm.

Outro

Sources Used

The most recent CFP article was 2021 for mgmt of HFrEF
https://www.cfp.ca/content/67/12/915

HFrEF update 2021 https://www.onlinecjc.ca/article/S0828-282X(21)00055-6/fulltext

early palliative care can improve QOL and length of life
https://pubmed.ncbi.nlm.nih.gov/17349493/