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أوراق خاصة جدًا

Pharmacotherapy 1

Heart Failure

(Updated March 2026; **updates are in RED**)

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Topic outline:

- ✓ Definition
- ✓ Epidemiology
- ✓ Etiology
- ✓ Normal Cardiac Function
- ✓ Pathophysiology
- ✓ Factors Precipitating/Exacerbating Heart Failure
- ✓ Clinical Presentation
- ✓ Laboratory Tests
- ✓ Diagnosis
- ✓ Treatment of Chronic Heart Failure

اللهم علمنا ما ينفعنا
وانفعنا بما علمتنا
وزدنا علما ♥

➢ Definition

✓ Heart failure (HF) is a progressive clinical syndrome caused by inability of the heart to pump sufficient blood to meet the body's metabolic needs.

✓ HF can result from any abnormality in cardiac structure or function that impairs the ability of the ventricle to fill with (diastolic dysfunction) or eject blood (systolic dysfunction).

✓ Diastolic dysfunction (restriction in ventricular filling): HF with preserved LVEF (HFpEF) or normal systolic function (normal LVEF)- patients typically are elderly, female, and obese, and have HTN, atrial fibrillation, or diabetes.

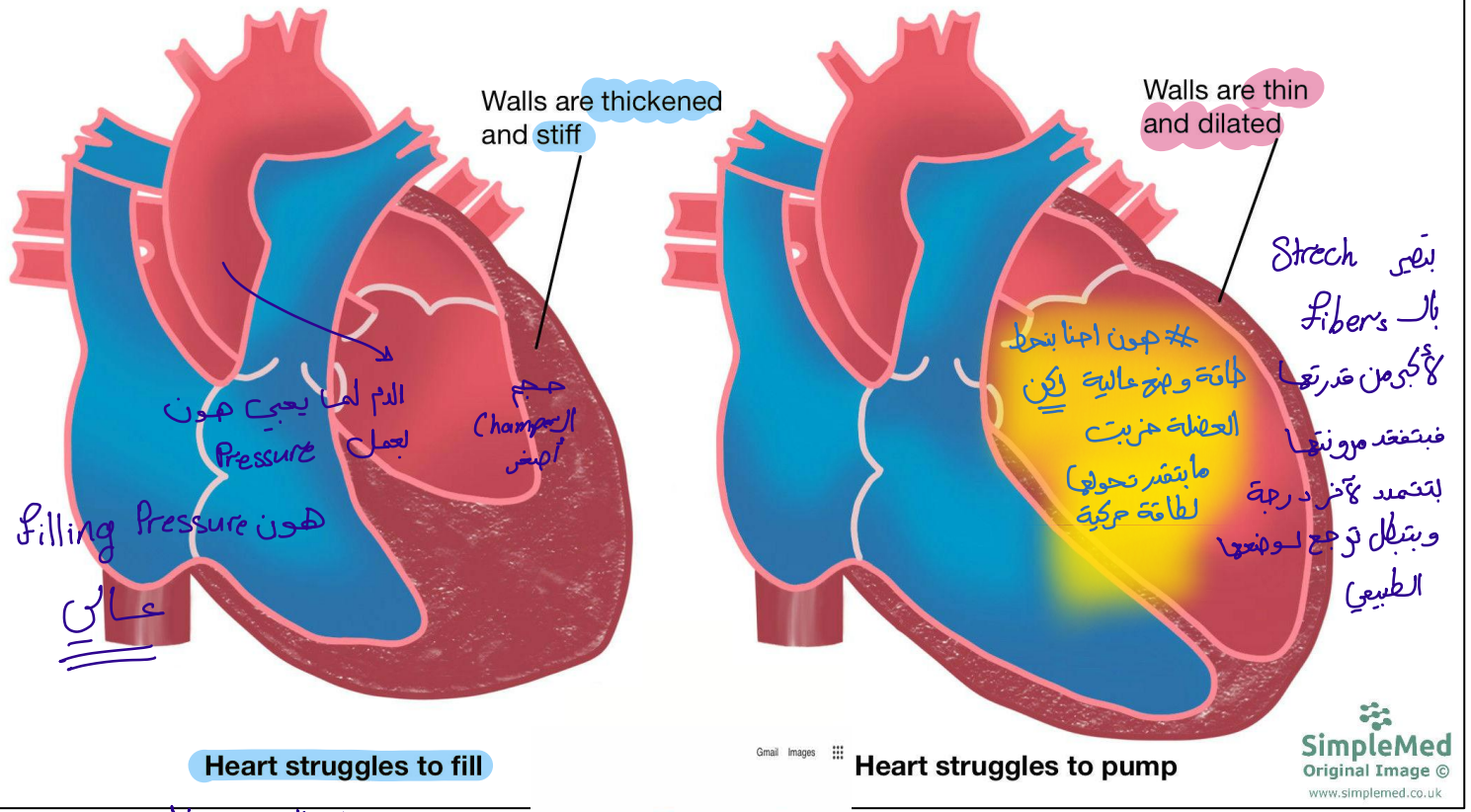
✓ Systolic dysfunction (decreased contractility): HF with reduced ejection fraction (HFrEF)- the classic, more familiar form of the disorder and is often caused by previous MI

العضلة بهلت مثل البالون
كانت بالأول تتمدد و تعبي جوواتها
هسا صارت Stiff

Systolic & بشبه البلوزة
اللي بتكون Stretch لوهي Size M
واللي بلبسها سايزه M راح تكون مناسبة
عليه ومرتبة ، لو لبسها حدا سايزه 2XL
راح ال Fibers تبع البلوزه يحرب مع انها
راح تدخلا فيه عادي بس راح يصير مش
مرتبه على اللي سايزه M صارت Dilated
& Thin
+ مثال المعلبة

Heart failure with preserved ejection fraction
(Diastolic heart failure)

Heart failure with reduced ejection fraction
(Systolic heart failure)



الدم لما يجي هون Pressure
حجم الchamber اصغر
Filling Pressure هون عالي
Systolic الضغط Pressure عالي
بس مختلفين عن بعض
Diastolic
حجم الدم راح يكون اقل
Regid

* هون احنا بنحط طاقة وضغط عالية لكن العضلة هربت مابتقدر تحولهن لطاقة حركية

Stretch بتصير بال Fibers لأكبر من قدرتها فبتفقد مرونتها بتتمدد لآخر درجة وبتبطل ترجع لوضعها الطبيعي

Systolic —
حجم الدم أكبر (حتى انتبجوا على شكل CLV)
بس كمان ال Blood Pressure بعمل
والقلب موعارف يطلع الدم منه و لايكبي الحجم الكتير

هاد الفيديو عرضناه بالسمينار بشرح شويصي بالعضلة وكيف ال Heart muscle بال Fiber بتعمل Stretch لدرجة معينة وبتخزن طاقة وضغط بعدين بتحولها لطاقة حركية
طاقة الوضع = الطاقة الحركية

Google
Google Search I'm Feeling Lucky

الارقام هون حفظ!

TABLE 39-1

Classification of Heart Failure According to Left Ventricular Ejection Fraction

HF with reduced EF (HFrEF)	HF with LVEF ≤40% (0.4)
HF with mildly reduced EF (HFmrEF)	HF with LVEF 41%-49% (0.41-0.49)
HF with preserved EF (HFpEF)	HF with LVEF ≥50% (0.5)
HF with improved EF (HFimpEF)	HF with baseline LVEF ≤40% (0.4) and subsequent measurement of LVEF >40% (0.4)

With optimal treatment, Life Style & Control Risk factors

اول مرة كان 40% او اقل

بعدين تحسن

EF > 40%

بعاي الحالة مابنحط المريض انه HFmrEF يصنف انه HFimpEF

شو بتتوقعو يكون العلاج ؟!

Continue

مابنقلل الجرعات بنخليه يكمل على كل العوامل الي ساعدته

➢ Epidemiology

دراسة قديمة نوعًا ما
out of the date

- ✓ In Jordan, the prevalence of HF is approximately 100,000; the estimated incidence is 8251 annually *
- ✓ Improved survival after MI → increased incidence & prevalence of HF
- ✓ Current estimates of annual expenditures for HF: > $30 billion (mainly on hospitalized patients)
- ✓ The overall 5-year survival is approximately 42% for all patients with a diagnosis of HF.
- ✓ Death is classified as sudden in about 40% of patients, due to serious ventricular arrhythmias as the underlying cause.

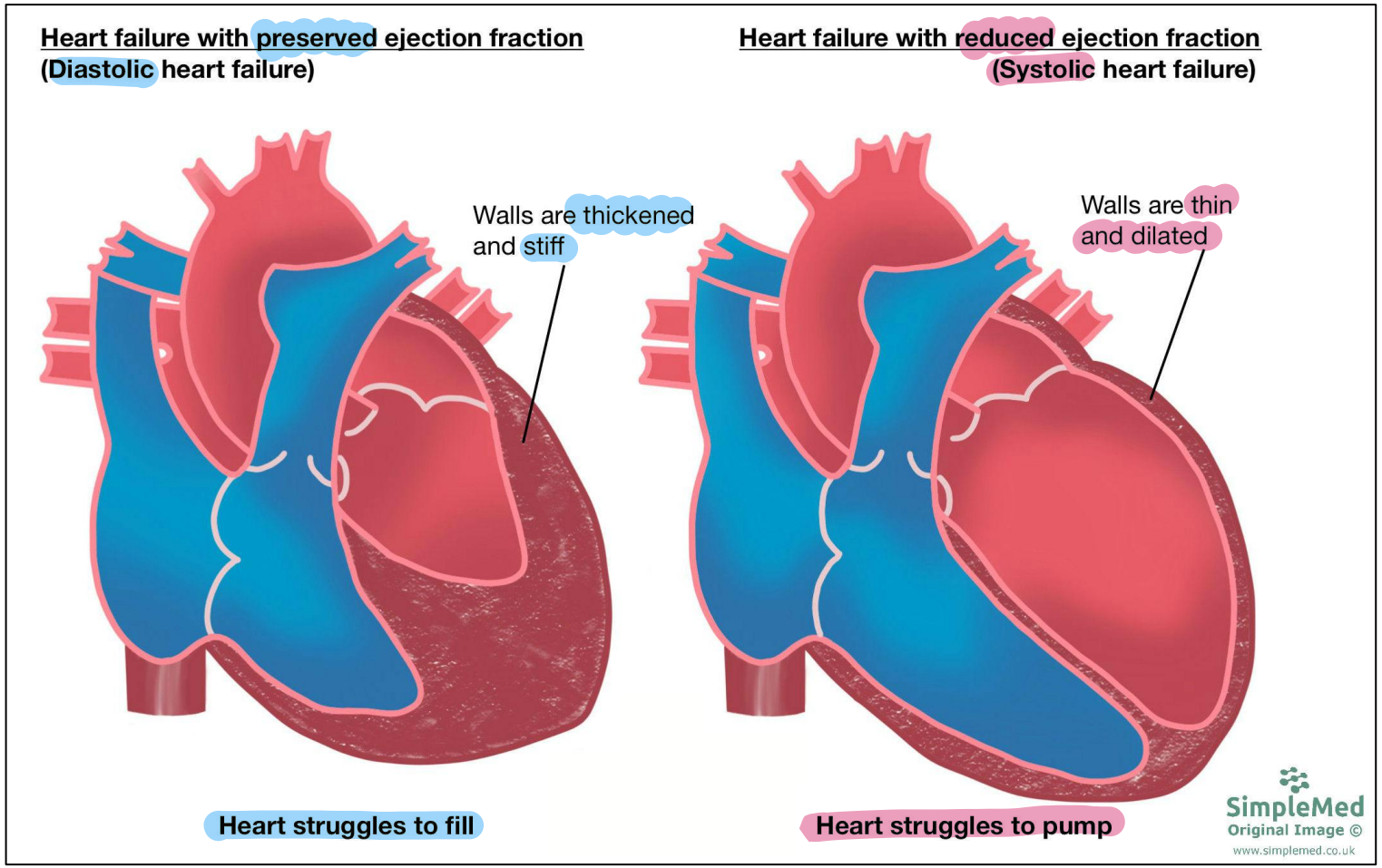
*: Abu Hayeah, Haneen & Saifan, Ahmad & Aburuz, Mohannad & Aljabery, Mohannad. (2017). Health-Related Quality of Life in Heart Failure in Jordan from Patients perspectives. IOSR Journal of Nursing and Health Science. 06. 14-21. 10.9790/1959-0601031421.

➢ Etiology

※ الرسمة انزحت فوق ※

- ✓ The most common causes of HF are: هم كتار لكن اختصرناهم بأهم 3

1. CAD resulting in an acute MI (75% of cases) Systolic
2. HTN ← Diastole غالبًا بعمل
3. Cardiomyopathies العم كتير انواع منهم بكون منذ الولادة



دكتورة إنعام مرأت
اللي عندهم صح وحكت راح يمروا معنا بسلايدات بعدين
دكتورة تهاني مرأت
الجدول كامل بمافيه الأقواس

TABLE 39-2

Common Causes of Chronic Heart Failure

- CAD (eg, myocardial infarction [MI] or ischemia)
- HTN
- Metabolic disorders (eg, obesity, diabetes)
- Valvular heart disease (eg, aortic stenosis)
- Heart rhythm disorders (eg, AF, tachycardia-induced cardiomyopathy)
- Chemotherapy and other cardiotoxic medications (eg, alcohol, stimulants)
- Infiltrative myocardial disease (eg, amyloidosis, sarcoidosis)
- Dilated cardiomyopathies (eg, genetic heart disease, viral infections, peripartum)
- Pericardial disease (eg, pericarditis, pericardial tamponade)
- Ventricular hypertrophy (eg, hypertrophic cardiomyopathy [HCM])

➢ Normal Cardiac Function

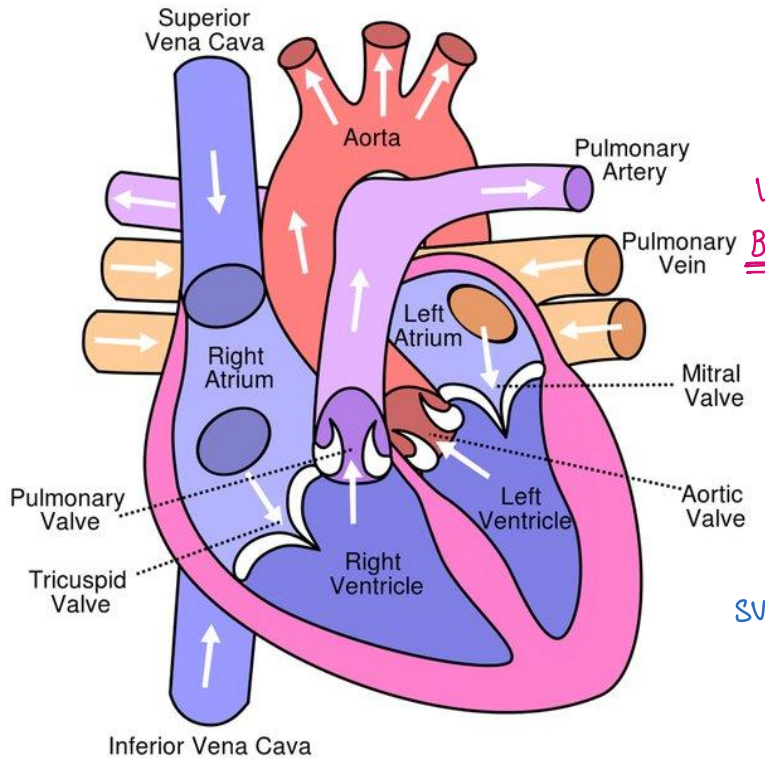
✓ CO is the volume of blood ejected per unit time (L/min) and is the product of HR & SV

$CO = HR \times SV$ راح يأثر عليه

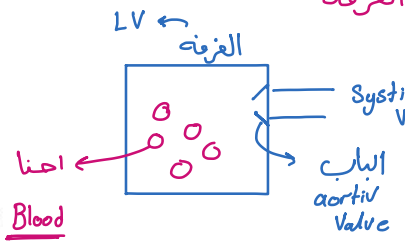
$BP = CO \times$ systemic vascular resistance (SVR)

✓ SV, or the volume of blood ejected during systole, depends on **preload**, **afterload**, & **contractility**.

كل ما كانت النبضة الواحدة بتطلع دم اكثر (SV↑)
يعني انه الدم اللي راح يطلع دم اكثر بالدقيقة الواحدة CO ↑



الدكتورة لما حكت مثال العزفة
بتسأل عن العلاقات ※



العزفة (LV) انقبضت ← راح تدفشنا (Blood) البرا
كل ما كان الانقباض اقوى ← بقوة اكبر بدل ما يطلع 80 ممكن يطلع 50

※ الـ Contractility بتأثر على الـ SV وكمان كم عدد الناس اللي جوا (كمية الـ Blood)

لو الباب مسكر ← راح يطلع جزء لانه في قوة بس عدد اقل SV↓
لو في Bodyguards وفي مساحة انه نطلع بس بصير SV↓

يعني SV ← بزيد اذا الـ Preload اكبر
بقل اذا الـ afterload كان اكبر

HFpEF هفبف ← بالفيديو كانوا يحكوه "هف رف"

➢ Compensatory Mechanisms in HFrEF

- ✓ HFrEF is a progressive disorder initiated by any event that impairs the ability of the heart to contract resulting in a decrease in CO.
  - متفقين انه CO↓ عشان الجسم يحافظ على BP ← TPR↑
- ✓ A decrease in CO results in activation of compensatory responses to maintain the circulation.
  - بتسميه
- ✓ Compensatory responses evolved to provide short-term support to maintain circulatory homeostasis after acute reductions in BP or renal perfusion.
  - هي مفيدة صح لكن لوقت قصير مجرد ما طال الـ Disorder على بتصير مضرة long term
  - ↑ RAAS activity
  - الجسم بحاول يصلح الوضع لانه بنكره اشي دخلي لكن الي طارئ كان Permenant Structural or Functional Change
- ✓ The persistent decline in CO in HF triggers long-term activation of these compensatory responses resulting in the complex functional, structural, biochemical, and molecular changes important for the development and progression of HF.
  - النتيجة لـ
  - لـ بالتالي القلب بدل ما يرتاح بحمل Load زيادة
  - هون لحالهم
  - لو طار MI على الـ Coronary Art والتسكير طول وما لحقنا نعمل Revascularization بسرعة وصارت الـ iscemia طولت على العضلة وجزء من العضلة ماتت وقدرتها على الـ Contraction قلت

✓ Compensatory mechanisms:

1. Tachycardia and increased contractility (sympathetic nervous system activation)
2. The Frank–Starling mechanism (increased preload increases stroke volume)
3. Vasoconstriction
4. Ventricular hypertrophy and remodelling

القلب بعمل Strech كتير

بتضخم

بتغير شكلها

✓ Our current understanding of HFrEF pathophysiology is best described by the neurohormonal model. Activation of endogenous neurohormones including norepinephrine (NE), angiotensin II, aldosterone and numerous proinflammatory cytokines plays an important role in ventricular remodeling and the subsequent progression of HF.

① ② ③ ④

بدنا اياه بكل اشي عدا القلب

✓ Importantly, pharmacotherapy targeted at antagonizing this neurohormonal activation has slowed the progression of HFrEF and improved survival.

* من هدول الفرق بنكتشف العلاج بدنا نقلل الـ fibrosis اللي بعمله ①②③④

عن طريق انه اعطي ادوية تعاكس شغلهم Aldactone , ACEI's, ARB's, β-Blocker

بقلل انه المريض يصير عنده مضاعفات

* هون الجواب

نحسن اداء عضلة القلب ⇐

↑ EF = المؤشر على التحسن ←

عن طريق اني اقلل الـ fibrosis و الـ Remodeling

لـ بحاي الطريقة بوقف الـ neurohormonal Disorder

وبقلل الـ Structural & functional HF

طيب الديجوكسين : مابشتغل على وظيفة

ما بقلل الـ morbidity & mortality او الـ Survival

بس بحسن الـ QOL بقلل Hospitalization + Relive Symptoms

* بده دراسة على ارض الواقع على عكس الـ Theory انه اعنا هون بنتوقع

وبلازم يكون في Clinical تثبت انه توقعنا صحيح او لا

Figure 39-1 Relationship between stroke volume and systemic vascular resistance.

In an individual with normal left ventricular (LV) function, increasing systemic vascular resistance has little effect on stroke volume. As the extent of LV dysfunction increases, the negative, inverse relationship between stroke volume and systemic vascular resistance becomes more important (B to A).

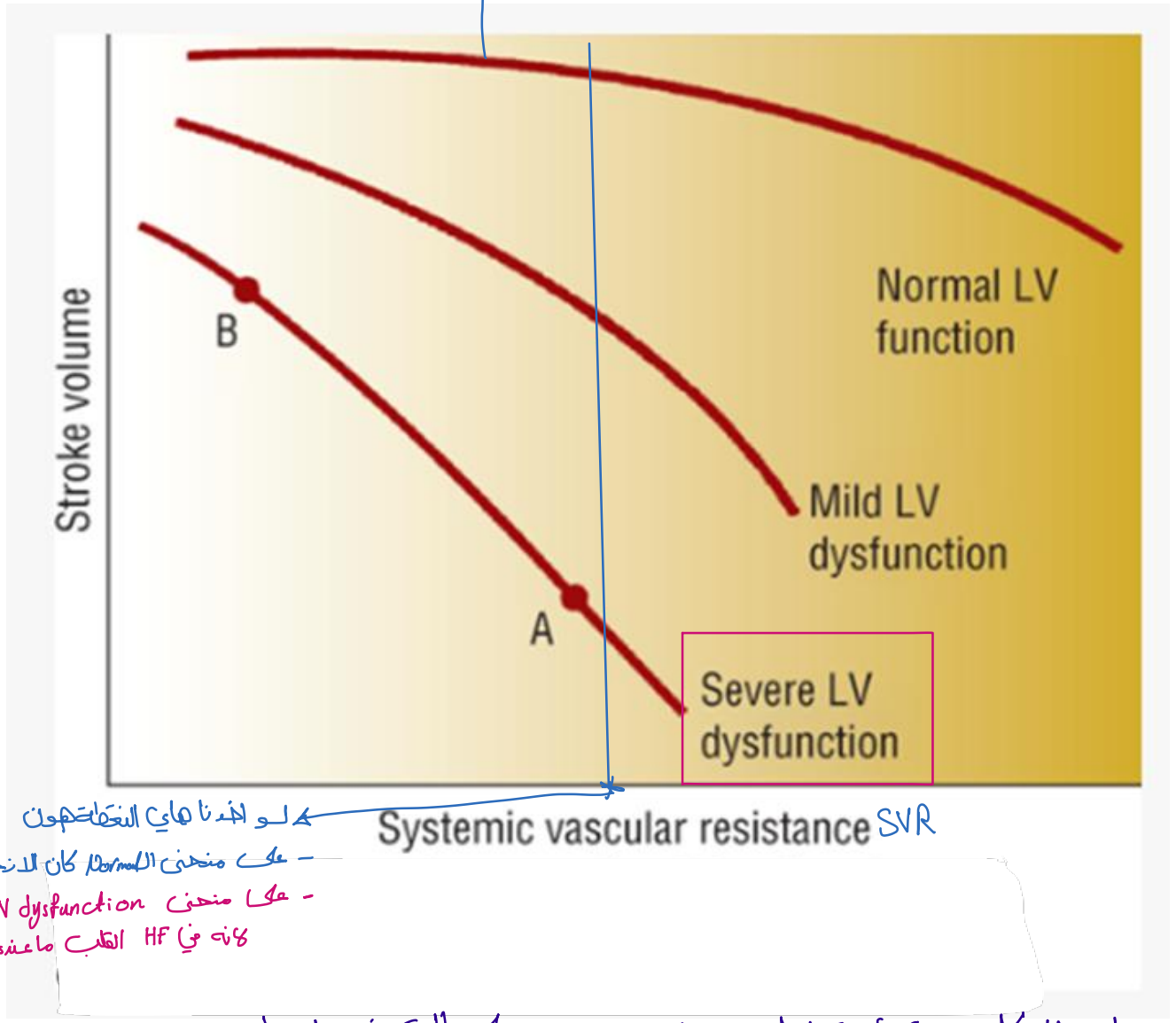


Figure 39-1 Explanation

This figure illustrates the relationship between stroke volume (SV) and afterload, represented by systemic vascular resistance (SVR). In individuals with normal left ventricular function, increases in afterload have only a minimal effect on stroke volume because the heart can compensate by generating sufficient contractile force to maintain forward blood flow. However, as left ventricular dysfunction develops, this relationship becomes increasingly inverse. In mild dysfunction, increases in SVR begin to reduce stroke volume modestly. In severe left ventricular dysfunction, even small increases in afterload result in a marked decrease in stroke volume, reflecting the inability of the weakened ventricle to overcome resistance. Clinically, this explains why patients with heart failure, particularly HFrEF, are highly sensitive to afterload, and why reducing afterload with therapies such as ACE inhibitors or vasodilators can significantly improve cardiac output and overall hemodynamics.

شرح الشكل 39-1

يوضح هذا الشكل العلاقة بين حجم الضربة (Stroke Volume - SV) والحِمْل البعدي (Afterload)، والذي تم تمثيله بـ المقاومة الوعائية الجهازية (SVR).

في الأفراد الذين يتمتعون بوظيفة طبيعية للبطين الأيسر، يكون لزيادة "الحمل البعدي" تأثير طفيف فقط على "حجم الضربة"؛ وذلك لأن القلب يمكنه التعويض عن طريق توليد قوة انقباضية كافية للحفاظ على تدفق الدم إلى الأمام.

ومع ذلك، ومع تطور خلل الوظيفة البطينية اليسرى (Left Ventricular Dysfunction)، تصبح هذه العلاقة عكسية بشكل متزايد:

- في حالة الخلل الطفيف: تبدأ الزيادات في المقاومة الوعائية الجهازية (SVR) في خفض حجم الضربة بشكل متواضع.
- في حالة الخلل الشديد في البطين الأيسر: تؤدي حتى الزيادات الطفيفة في "الحمل البعدي" إلى انخفاض حاد وملحوظ في "حجم الضربة"، مما يعكس عدم قدرة البطين الضعيف على التغلب على المقاومة.

سريرياً (إكلينيكياً): يفسر هذا سبب حساسية مرضى قصور القلب (خاصة المصابين بـ قصور القلب مع انخفاض الكسر القذفي - HFrEF) العالية تجاه "الحمل البعدي"، ولماذا يمكن للعلاجات التي تقلل من هذا الحمل (مثل مُثبطات الإنزيم المحول للأنجيوتنسين - ACE inhibitors أو موسعات الأوعية الدموية) أن تحسن بشكل كبير من النتاج القلبي (Cardiac Output) والديناميكا الدموية الشاملة.

أهم المصطلحات الواردة:

- Stroke Volume: حجم الضربة (كمية الدم المقذوفة في الانقباضة الواحدة).
- Afterload: الحِمْل البعدي (المقاومة التي يجب أن يضخ القلب ضدها).
- SVR: المقاومة الوعائية الجهازية.
- HFrEF: قصور القلب مع كسر قذفي منخفض.
- Hemodynamics: الديناميكا الدموية (حركية الدم).

RAAS

الدكتورة هامرت عليها
كله انشرحت
بال HTN

Figure 39-2 Physiology of the renin-angiotensin-aldosterone system.

Renin converts angiotensinogen to angiotensin I (AT1). AT1 is cleaved to angiotensin II (AT2) by the angiotensin-converting enzyme (ACE). AT2 has a number of physiologic actions that are detrimental in HF. Note that AT2 can be produced in a number of tissues, including the heart, independent of ACE activity. ACE is also responsible for the breakdown of bradykinin. Inhibition of ACE results in the accumulation of bradykinin that, in turn, enhances the production of vasodilatory prostaglandins.

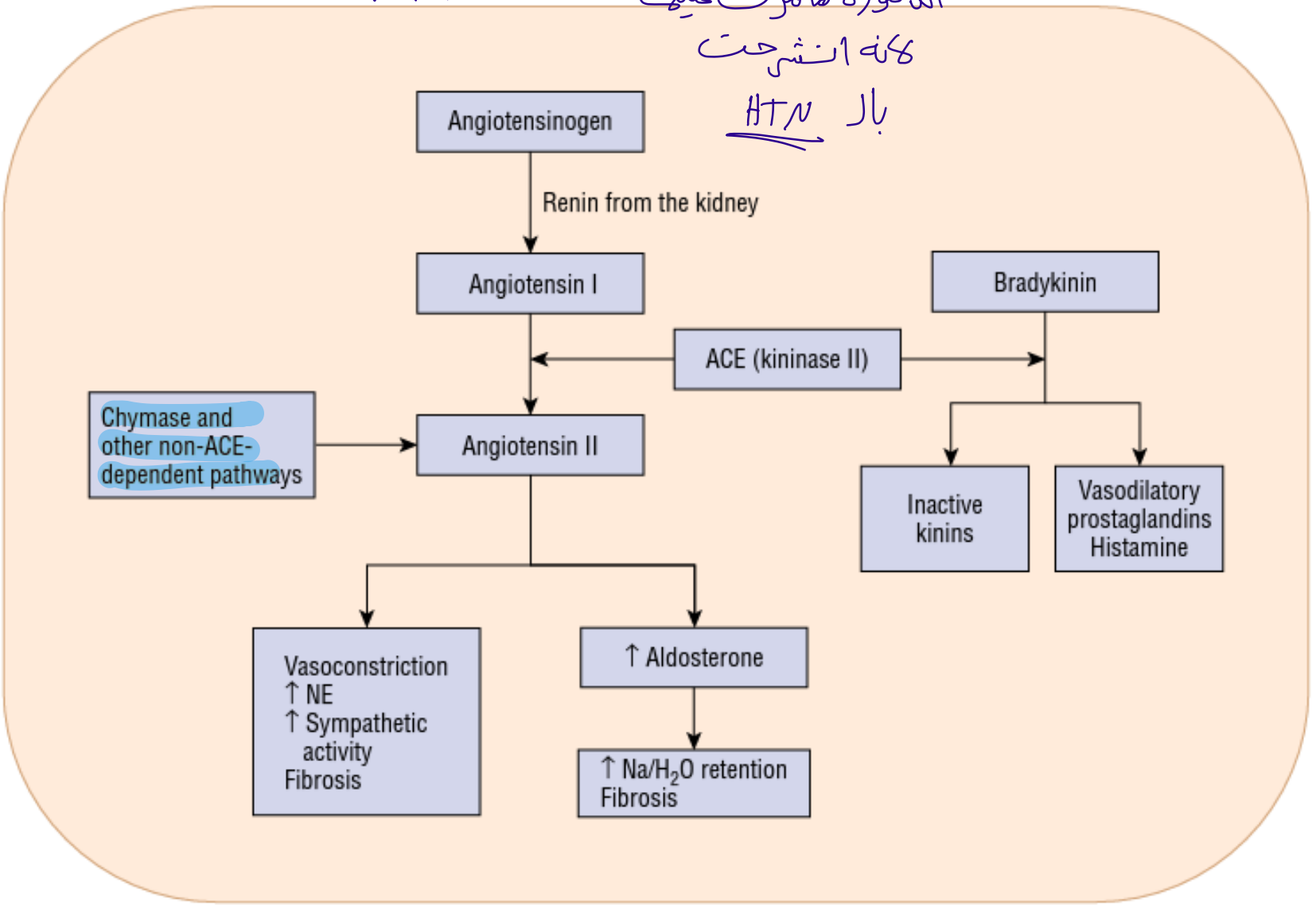
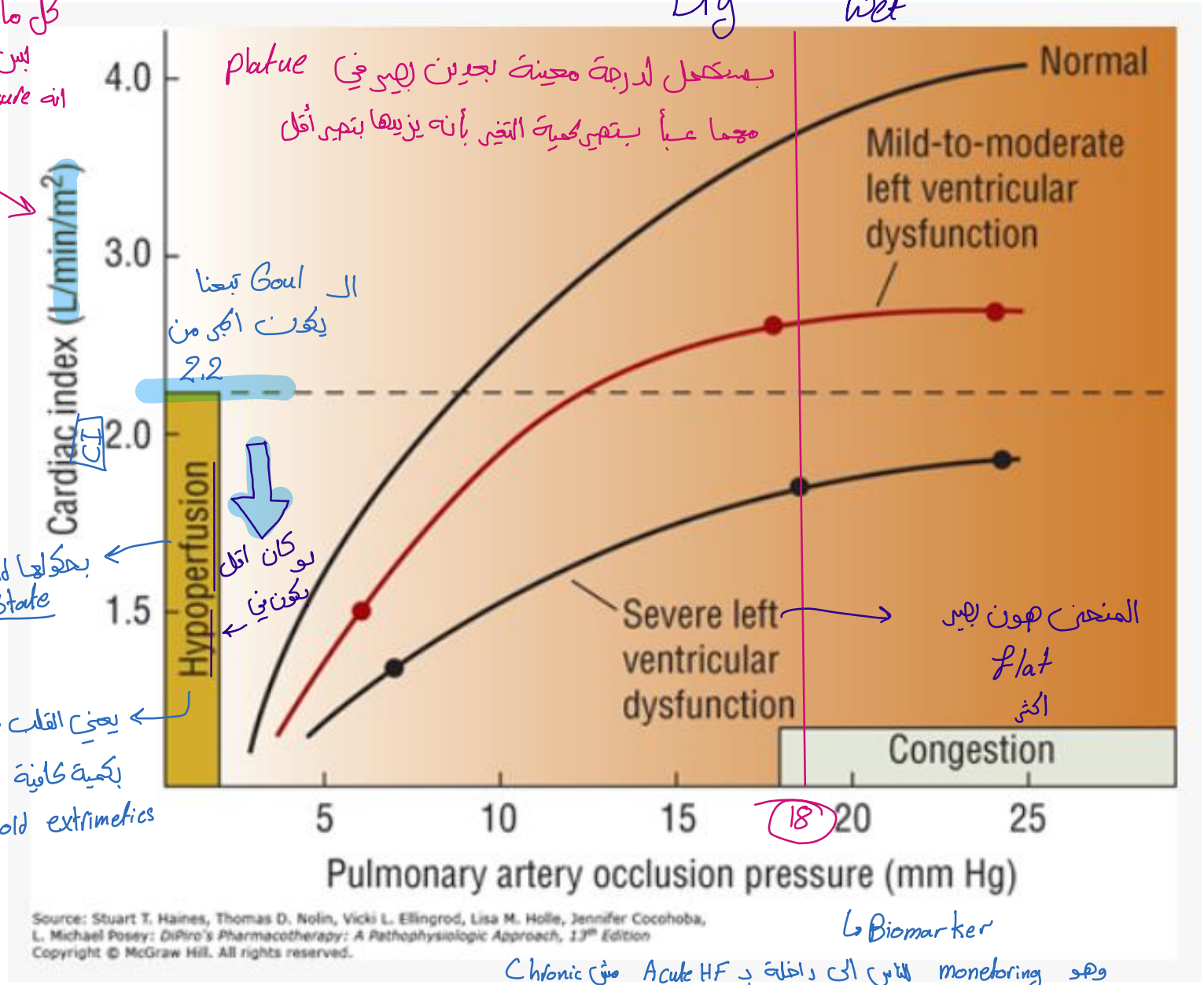


Figure 39-3

Relationship between cardiac output (shown as a cardiac index which is CO/body surface area) and preload (shown as pulmonary artery occlusion pressure).



This figure demonstrates the relationship between cardiac output (cardiac index) and preload, represented by pulmonary artery occlusion pressure (PAOP), and reflects the clinical Frank–Starling mechanism. In a normal heart, increasing preload leads to a substantial increase in cardiac output, indicating an efficient response to ventricular filling. However, as left ventricular dysfunction develops, this relationship becomes progressively impaired. In mild-to-moderate dysfunction, increases in preload produce only modest improvements in cardiac output. In severe dysfunction, further increases in preload result in minimal or no increase in cardiac output, while significantly elevating filling pressures. Clinically, this creates two important states: at low preload, patients may experience hypoperfusion due to insufficient cardiac output, whereas at high preload, patients develop congestion due to elevated pressures, leading to symptoms such as pulmonary edema and dyspnea. Therefore, in heart failure, especially advanced disease, increasing preload is no longer beneficial and instead worsens congestion, which explains why therapies such as diuretics are used to reduce preload and optimize the patient's position on the curve.

يوضح هذا الشكل العلاقة بين **النتاج القلبي (Cardiac Output)** —أو **المشعر القلبي (Cardiac Index)**— و**الحِمْل القبلي (Preload)**، والذي يمثله **ضغط انحباس الشريان الرئوي (PAOP)**، كما يعكس آلية **فرانك-ستارلينج (Frank–Starling mechanism)** السريرية.

في القلب الطبيعي، تؤدي زيادة "الحمل القبلي" إلى زيادة كبيرة في "النتاج القلبي"، مما يشير إلى استجابة فعالة لامتلاء البطين. ومع ذلك، ومع تطور **خلل الوظيفة البطينية اليسرى**، تضعف هذه العلاقة تدريجياً:

- **في الخلل الطفيف إلى المتوسط:** تؤدي الزيادات في "الحمل القبلي" إلى تحسن طفيف فقط في "النتاج القلبي".
- **في الخلل الشديد:** تؤدي الزيادات الإضافية في "الحمل القبلي" إلى زيادة طفيفة جداً أو معدومة في "النتاج القلبي"، بينما ترفع ضغوط الامتلاء بشكل كبير.

سريرياً، يؤدي هذا إلى حالتين هامتين:

1. **عند انخفاض الحمل القبلي:** قد يعاني المرضى من **نقص التروية (Hypoperfusion)** بسبب عدم كفاية النتاج القلبي.
2. **عند ارتفاع الحمل القبلي:** يصاب المرضى بـ **الاحتقان (Congestion)** نتيجة ارتفاع الضغوط، مما يؤدي إلى أعراض مثل **الوذمة الرئوية (Pulmonary edema)** و**ضيق التنفس (Dyspnea)**.

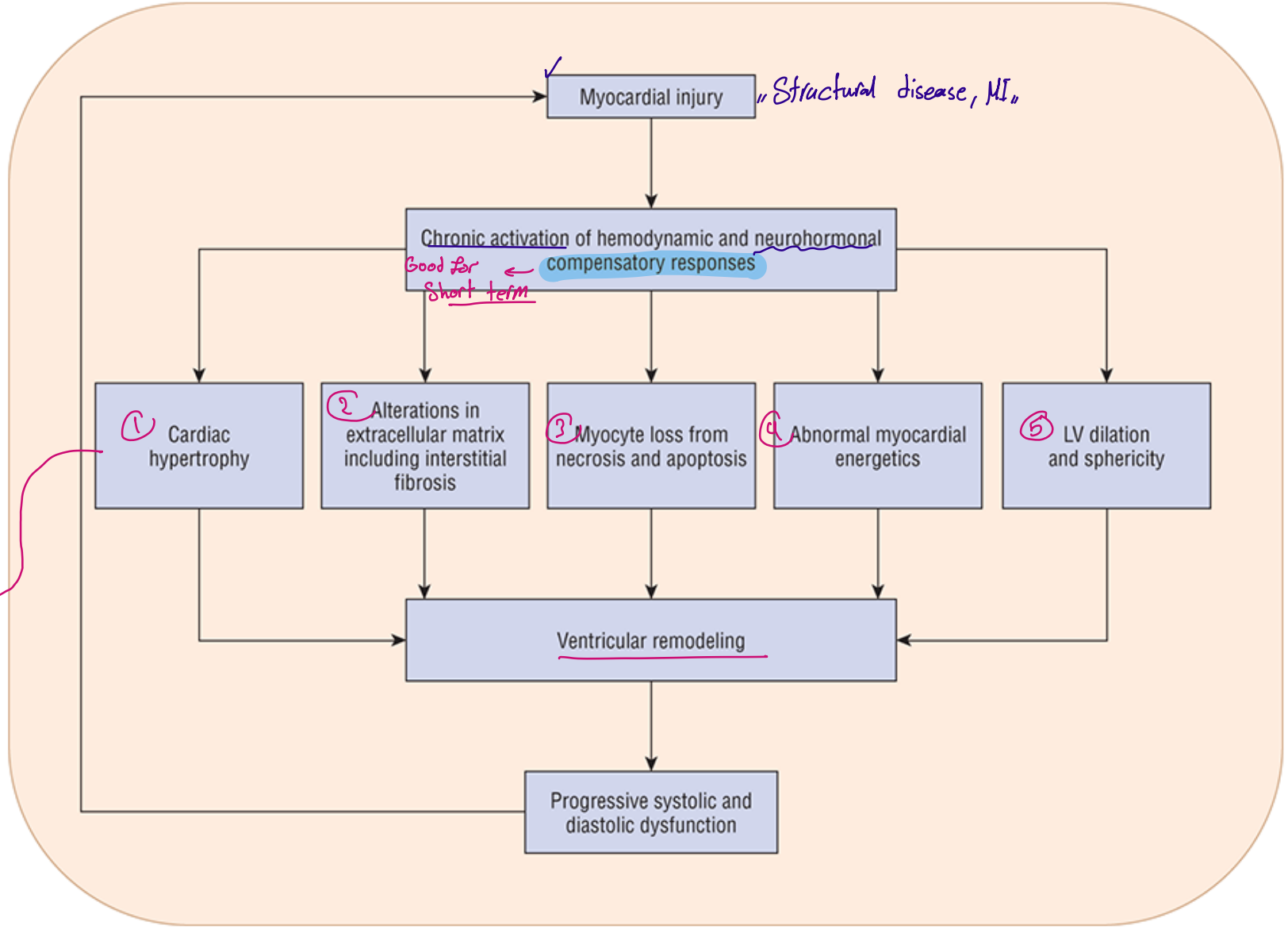
لذلك، في حالات قصور القلب (خاصة الحالات المتقدمة)، لم تعد زيادة "الحمل القبلي" مفيدة، بل على العكس، تزيد من سوء الاحتقان. وهذا يفسر سبب استخدام علاجات مثل **مدرات البول (Diuretics)** لتقليل "الحمل القبلي" وتحسين وضع المريض على منحنى "فرانك-ستارلينج".

ترجمة AI

Cardiac Remodeling ... weird

Figure 39-4 Key components of the pathophysiology of cardiac remodeling.

Myocardial injury (eg, MI) results in the activation of a number of hemodynamic and neurohormonal compensatory responses in an attempt to maintain circulatory homeostasis. Chronic activation of the neurohormonal systems results in a cascade of events that affect the myocardium at the molecular and cellular levels. These events lead to th changes in ventricular size, shape, structure, and function known as ventricular remodeling. The alteration in ventricular function result in further deterioration in cardiac systolic and diastolic function that further promote the remodeling.



Source: Stuart T. Haines, Thomas D. Nolin, Vicki L. Ellingrod, Lisa M. Holle, Jennifer Cocohoba, L. Michael Posey: *DiPiro's Pharmacotherapy: A Pathophysiologic Approach, 13th Edition*

TABLE 39-3

Beneficial and Detrimental Effects of the Compensatory Responses in Heart Failure

Compensatory Response	Beneficial Effects of Compensation	Detrimental Effects of Compensation
Tachycardia and increased contractility	Increased NE (SNS activation) Helps maintain CO	Shortened diastolic filling time β_1-receptor downregulation, decreased receptor sensitivity Precipitation of ventricular arrhythmias Increased risk of myocardial cell death Increased MVO_2
Vasoconstriction and increased afterload	Increased NE (SNS activation) Increased AT_2 (RAAS activation) Increased BNP Maintains BP despite reduced CO Shunts blood from nonessential organs to brain and heart	Increased afterload decreases SV and further activates the compensatory responses Increased MVO_2
Fluid retention and increased preload	Increased aldosterone (RAAS activation) Increased BNP, AVP, SGLT2 Sodium and water/fluid retention Optimizes SV via Frank-Starling mechanism	Pulmonary and systemic congestion and edema Increased MVO_2
Ventricular hypertrophy and remodeling	Increased NE (SNS activation) Helps maintain CO Reduces myocardial wall stress Decreases MVO_2	Diastolic dysfunction Systolic dysfunction Increased risk of myocardial cell death and ischemia Increased arrhythmia risk Fibrosis

AII, angiotensin II; AVP, arginine vasopressin; BP, blood pressure; CO, cardiac output; MVO_2, myocardial oxygen demand; NE, norepinephrine; RAAS, renin-angiotensin-aldosterone system; SGLT2, sodium-glucose cotransporter-2; SNS, sympathetic nervous system.

شرح شات GPT

💡 أولاً: شو الفكرة الأساسية؟

لما القلب يضعف (Heart Failure ↓CO)، الجسم يخاف إنه الأعضاء ما يوصلها دم كفاية
→ فيشغل آليات تعويض (Compensatory mechanisms)

🔥 1. أول رد فعل: الجهاز العصبي السمبثاوي (SNS)

⬆️ Norepinephrine (NE)
→ يعمل:
- ↑ Tachycardia (زيادة النبض)
- ↑ Contractility (قوة الانقباض)

✅ الفائدة:
- يحافظ على Cardiac Output (CO)
- يوصل الدم للأعضاء المهمة (brain + heart)

❌ المشكلة:
- ⏱️ يقصر وقت الامتلاء (diastole) → يقل SV مع الوقت
- القلب يصير → 📉 Downregulation للـ β-receptors أقل استجابة
- ⚡ Arrhythmias
- 💀 ↑ موت خلايا القلب (myocardial cell death)
- يزيد الضغط → (استهلاك الأوكسجين) ↑ MVO_2 🔥 على القلب

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2. ثاني رد فعل: نظام الرينين-أنجيوتنسين-ألدوستيرون (RAAS)
⬆️ Angiotensin II + Aldosterone

شو يعمل؟

- 🩸 Vasoconstriction → ↑ Afterload
- 💧 احتباس سوائل (Na^+ + water) → Preload ↑

✅ الفائدة:

- يحافظ على Blood Pressure
- يزيد الامتلاء → يحسن الضخ حسب Frank-Starling mechanism 👉
SV \propto EDV

❌ المشكلة:

- 💦 Fluid overload → edema + pulmonary congestion
- 🪨 ↑ Afterload → القلب يتعب أكثر → ↓ SV
- 🔁 يدخل في حلقة مفرغة (vicious cycle)
- 💀 ↑ cell death + ischemia

3. هرمونات إضافية 🧠

📌 BNP
- يفرز عشان يخفف الضغط على القلب
- يعمل:
  - ↓ preload
  - ↓ afterload
  - ↓ MVO_2

يعني هو محاولة إنقاذ من الجسم 👉

📌 AVP + SGLT2
- يزيدوا احتباس السوائل 😬

---

4. النتيجة النهائية: Remodeling 🏗️

بسبب الضغط المستمر:
- 💪 Ventricular hypertrophy
- 🪨 Fibrosis
- 💔 Systolic dysfunction
- 🫀 Diastolic dysfunction

🔄 الخلاصة (أهم ربط لازم تفهميه):
1. ↓ CO
2. الجسم يفعل:
- SNS → ↑ HR & contractility
- RAAS → ↑ fluid + vasoconstriction
3. بالبداية 👍 (يحافظ على الحياة)
4. مع الوقت 👎:
- ↑ afterload → ↓ SV
- ↑ MVO_2 → ischemia
- ↑ arrhythmias
5. → يدخل القلب في حلقة تدهور مستمرة

➢ Factors Precipitating/Exacerbating HF

✓ Appropriate therapy can maintain patients in a "compensated" state → relatively symptom-free

✓ Many aggravating or precipitating factors may cause a previously compensated patient to develop worsened symptoms necessitating hospitalization:

1 ➢ Cardiac events (MI, atrial fibrillation, uncontrolled HTN)

2 ➢ Noncardiac events (pulmonary infections, pulmonary embolus, diabetes, worsening renal function, hypothyroidism, and hyperthyroidism)

3 ➢ Nonadherence with prescribed HF medications or with dietary recommendations (eg, sodium intake and fluid restriction)

4 ➢ A number of drugs can precipitate or exacerbate HF by one or more of the following mechanisms: (a) negative inotropic effects; (b) direct cardiotoxicity; or (c) increased sodium and/or water retention.

Table 39-4 - Part 1

Selected Drugs That May Precipitate or Exacerbate Heart Failure

Negative Inotropic Effect

- Antiarrhythmics (disopyramide, dronedarone, flecainide, propafenone, sotalol)
- β-Blockers (eg, propranolol, metoprolol, carvedilol)
- Calcium channel blockers—nondihydropyridine type (verapamil, diltiazem)
- Itraconazole

Cardiotoxic

- Alcohol
- Amphetamines (eg, cocaine, methamphetamine)
- Antiarrhythmics (proarrhythmic actions—disopyramide, flecainide, propafenone, sotalol)
- Cancer Therapies (various)
  - Alkylating agents (eg, cyclophosphamide, ifosfamide, melphalan)
  - Anthracyclines (eg, doxorubicin, daunorubicin, epirubicin, idarubicin)
  - Antimetabolites (eg, fluorouracil, capecitabine, fludarabine, decitabine)
  - Antimicrotubules (eg, docetaxel, paclitaxel)
  - BCR-ABL inhibitors (eg, bosutinib, dasatinib, imatinib, ponatinib)
  - BRAF inhibitors (eg, dabrafenib)
  - Hormonal therapy (eg, aplalutamide, bicalutamide, darolutamide, nilutamide)
  - Human epidermal growth factor receptor inhibitors (eg, lapatinib, osimertinib)
  - Human epidermal growth factor receptor 2 inhibitors (eg, pertuzamab, trastuzumab)
  - Immune checkpoint inhibitors (eg, nivolumab, ipilimumab, pembrolizumab)
  - Immunomodulators (eg, lenalidomide, pomalidomide, thalidomide)
  - MEK inhibitors (eg, binimetinib, cobimetinib, trametinib)
  - Mitomycin
  - Mitoxantrone
  - Vascular endothelial growth factor inhibitors (eg, axitinib. bevacizumab, cabazantinib, lenvatinib, pazopanib, sorafenib, sunitinib, vandetanib)
  - Miscellaneous (eg, entrectinib, fedratinib, ripretinib, tretinoin)
- Carbamazepine

Table 39-4 - Part 2

Selected Drugs That May Precipitate or Exacerbate Heart Failure

Sodium and Water Retention
Androgens and estrogens
Cyclooxygenase-2 inhibitors
Glucocorticoids (systemic)
NSAIDs
Pioglitazone and rosiglitazone
Salicylates (high dose)
Sodium-containing drugs (eg, carbenicillin disodium, ticarcillin disodium)
Uncertain Mechanism
Alpha-1 blockers (eg, doxazosin)
Dipeptidyl peptidase-4 inhibitors (eg, saxagliptin, alogliptin)
Gabapentin, pregabalin
Tumor necrosis factor-α inhibitors (eg, adalimumab, infliximab, etanercept)

➢ Clinical presentation

✓ Patient presentation may range from asymptomatic to cardiogenic shock.

المريض بشكي منها

✓ Symptoms

Dyspnea, particularly on exertion	Orthopnea	Paroxysmal nocturnal dyspnea
Exercise intolerance (بمشي خطوتين وبتعب)	Tachypnea	Cough
Fatigue	Nocturia	Hemoptysis
Abdominal pain	Anorexia	Nausea
Bloating	Ascites	Poor appetite, early satiety
Mental status changes	Weight gain or loss	

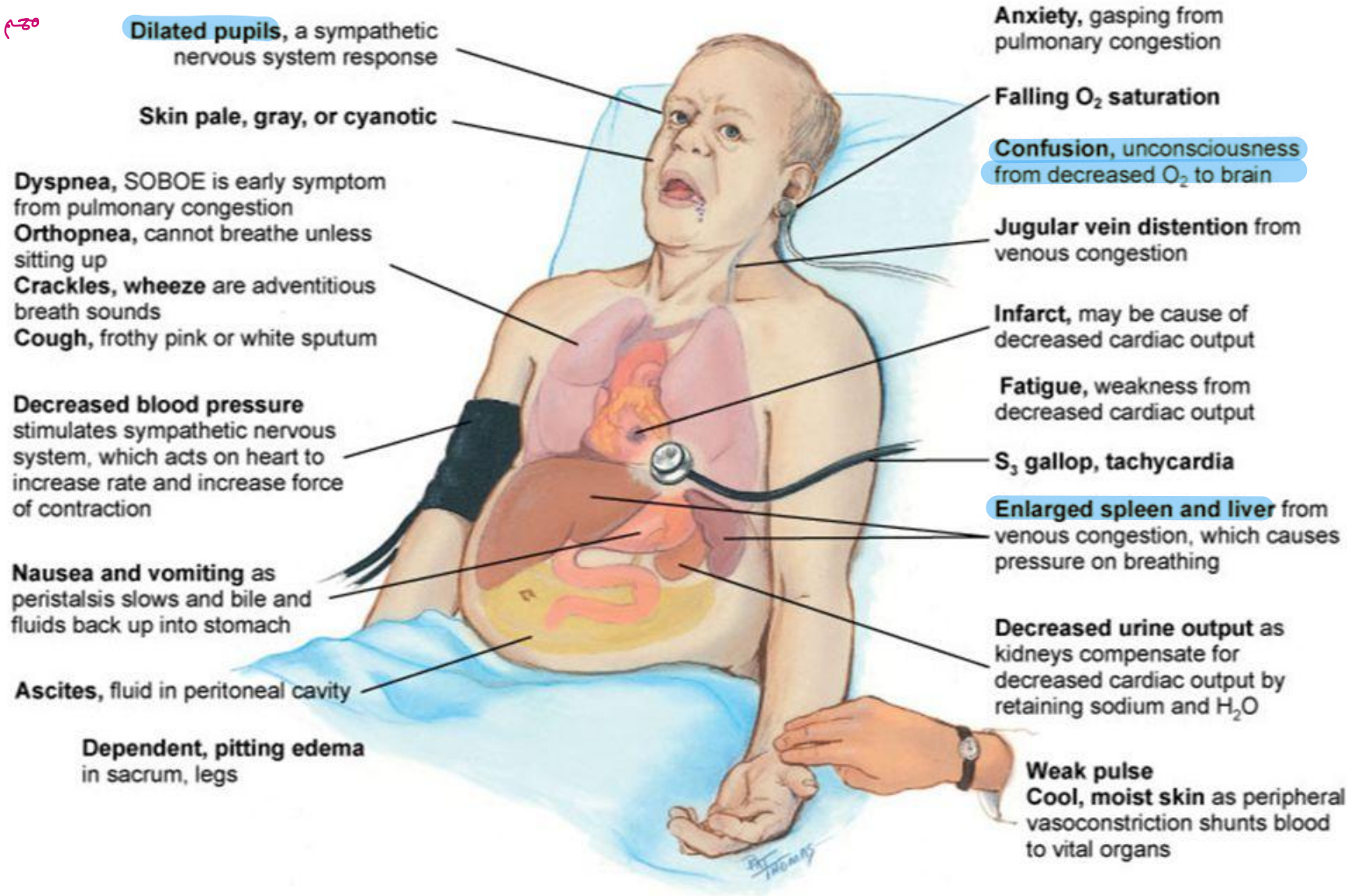
الدكتور بميزها

✓ Signs

Pulmonary rales (بسمعها بالسماعة)	Pulmonary edema	S3 gallop (ECG & سماعة)	Cool extremities
Pleural effusion → Chest x-ray	Narrow pulse pressure	Cardiomegaly	Tachycardia
Peripheral edema	Jugular venous distention	Hepatomegaly	

نفس الي فوق بس في توضيحات
حلو انه تقرأوه و تعرفوا ليه؟!
مهم وبكل الي فوق

Clinical Portrait of Heart Failure



Dilated pupils, a sympathetic nervous system response

Skin pale, gray, or cyanotic

Dyspnea, SOBOE is early symptom from pulmonary congestion
Orthopnea, cannot breathe unless sitting up
Crackles, wheeze are adventitious breath sounds
Cough, frothy pink or white sputum

Decreased blood pressure stimulates sympathetic nervous system, which acts on heart to increase rate and increase force of contraction

Nausea and vomiting as peristalsis slows and bile and fluids back up into stomach

Ascites, fluid in peritoneal cavity

Dependent, pitting edema in sacrum, legs

Anxiety, gasping from pulmonary congestion

Falling O_2 saturation

Confusion, unconsciousness from decreased O_2 to brain

Jugular vein distention from venous congestion

Infarct, may be cause of decreased cardiac output

Fatigue, weakness from decreased cardiac output

S_3 gallop, tachycardia

Enlarged spleen and liver from venous congestion, which causes pressure on breathing

Decreased urine output as kidneys compensate for decreased cardiac output by retaining sodium and H_2O

Weak pulse
Cool, moist skin as peripheral vasoconstriction shunts blood to vital organs

➢ Laboratory tests

- ✓ B-type natriuretic peptide (BNP)>100 pg/mL and NT-proBNP >300 pg/mL: assist in differentiating dyspnea caused by HF from other causes.

- ✓ Electrocardiogram may be normal or it could show numerous abnormalities including acute changes from myocardial ischemia, atrial fibrillation, bradycardia, left ventricular hypertrophy

- ✓ Serum creatinine: It may be increased due to hypoperfusion. Preexisting renal dysfunction can contribute to volume overload

- ✓ Complete blood count useful to determine if heart failure due to reduced oxygen carrying capacity

- ✓ Chest X-ray: Useful for detection of cardiac enlargement, pulmonary edema, and pleural effusions

- ✓ Echocardiogram: Used to assess LV size, valve function, pericardial effusion, wall motion abnormalities, and ejection fraction

- ✓ Hyponatremia: serum sodium < 130 mEq/L is associated with reduced survival and may indicate worsening volume overload and/or disease progression

➢ Diagnosis

بدنا نجمع معلومات عنه + الـ History

✓ HF is often initially suspected in a patient based on symptoms.

✓ But, signs and symptoms lack sensitivity for diagnosing HF (they are frequently found with many other disorders).

HF ممكن الـ Shortness of Breath واحد عنده مشاكل من موضوع صحي , مش منطقي نحكيله عندك

✓ In general, HFpEF cannot be distinguished from HFrEF on the basis of the 1- history, 2- physical examination, 3- chest x-ray, and 4- ECG alone.

Echocardiogram بدنا فقط 1- 2- 3- 4- صعب نفرق بينهم عن طريق EF بتطلعلي فكرة عن

✓ A complete history and physical examination targeted at identifying cardiac or noncardiac disorders that may cause HF development or progression are essential in the initial patient evaluation.

✓ A careful medication history should also be obtained (ethanol, tobacco, NSAIDs).

- ✓ The patient's volume status should be documented by assessing the body weight, JVD, and presence or absence of pulmonary congestion and peripheral edema.

- ✓ Although the history, physical examination, and laboratory tests provide important insight into the underlying cause of HF, the **echocardiogram** is the **single** most useful test in the evaluation of the patient. https://www.youtube.com/watch?v=Kirg2GuESsE

- ✓ The echocardiogram is used to assess abnormalities in cardiac structure and function and should include evaluation of the pericardium, myocardium, and heart valves, and quantification of the LVEF to determine if systolic or diastolic dysfunction is present.

- ✓ Sudden cardiac death, primarily due to ventricular tachycardia and fibrillation, is responsible for 40% to 50% of the mortality in patients with HFrEF.

جهاز صغير ينزرع داخل الجسم (تحت الجلد)، غالباً في الصدر.

وظيفته: ⚡

يراقب نبض القلب بشكل مستمر، وإذا صار في:
- ❗ اضطراب خطير في النبض (Arrhythmia)

مثل:
- Ventricular tachycardia
- Ventricular fibrillation

يقوم مباشرة بـ: 👉
- ⚡ إعطاء صدمة كهربائية (shock)

→ ترجع النبض للوضع الطبيعي

⇒ Part 2

الدكتورة عملت مراجعة صغيرة بالبداية
- improved EF وكيف بنعالجه
- Compensatory حكت كمان mechanism
- figures : SV & afterload , RAAS Frank-Starling law "Co index & preload"
- factors , lab test , Diagnosis

➢ Treatment of Chronic Heart Failure

low CO

✓ Desired Outcomes

The goals of therapy in management of chronic HF are:

1. improve the patient's quality of life
2. relieve or reduce symptoms (Free → minimal)
3. prevent or minimize hospitalizations
4. slow progression of the disease ("Remodeling process")
5. prolong survival ⇌ ↓ mortality

One way direction: A → B → C → D (ما في رجعة منه مجرد B مستحيل يرجع C ما بيرجع)

✓ The ACC/AHA guidelines for the evaluation and management of HF utilize a staging system that not only recognizes the **evolution and progression** of the disorder but also emphasizes **risk factor modification and preventive treatment** strategies (Next Figure).

Control لازم اعمل HTN كيف امنع تطور المرض عن طريق اني اتحكم بالسبب لو السبب

Stage 1 لسا ما بان HF

✓ The general principles used to guide the treatment of HFrEF are based on numerous large, randomized, double-blind, multicenter trials.

Diastolic عكس ال ← evidance "قوي جدا"

Stage C

✓ With HFpEF (EF is ≥50%) with signs and/or symptoms of HF: Most of the pharmacotherapies known to benefit patients with HFrEF have been less beneficial in HFpEF. Targeting the underlying cause, most commonly uncontrolled HTN, has been the primary strategy for managing patients with HFpEF. However, recent trials have identified treatments that benefit patients with HFpEF as SGLT2 inhibitors, ARBs, spironolactone, & ARNI.

female + HTN + obese + Arrythmia

(17) guid line جديدة

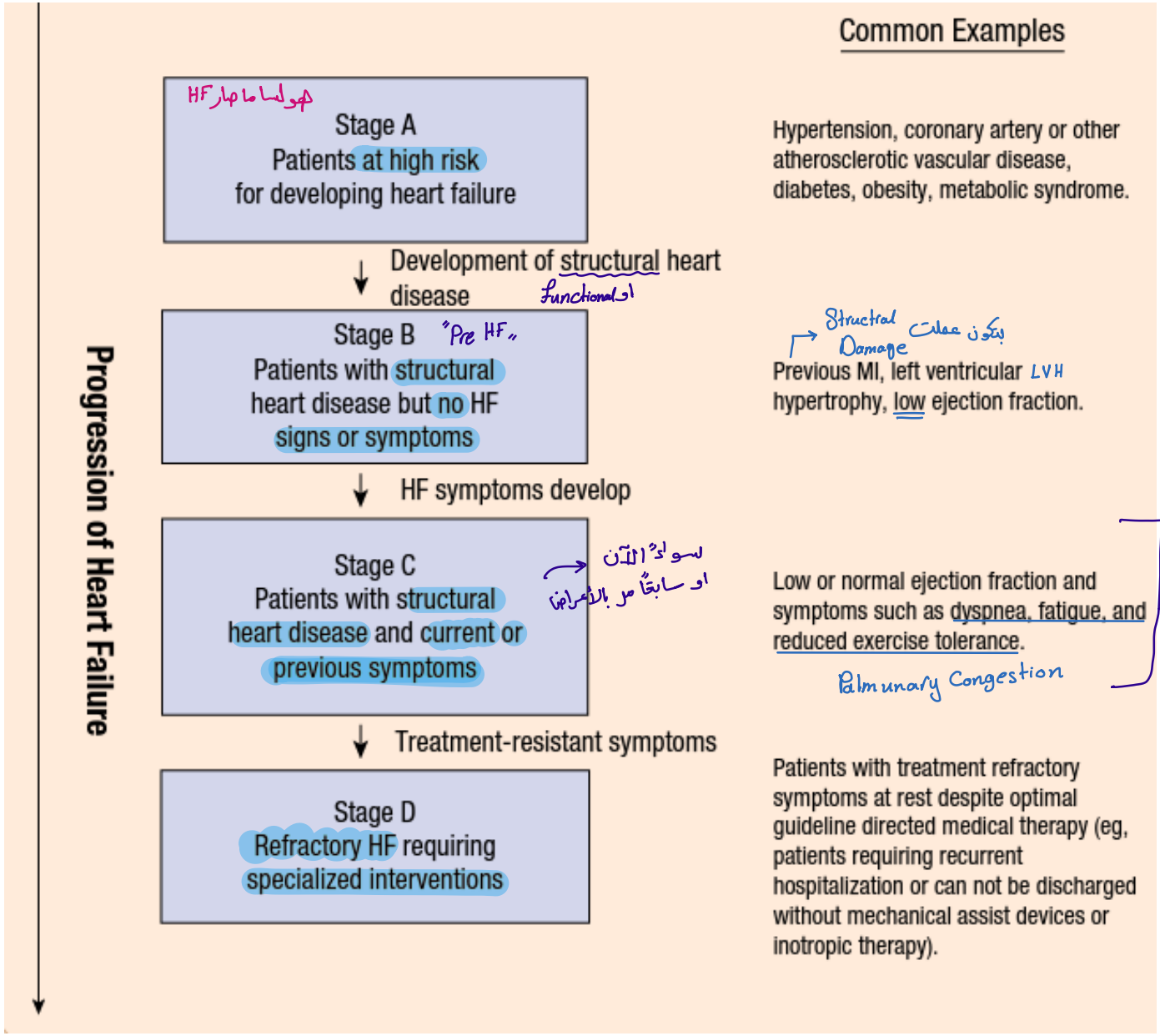
Systolic بشبه ال

مش ACEI ← (2) (3) (4)

For your reference

ACC/AHA Heart Failure Staging System

بفرق عن Class نركز



Updated figure in the next slide

الدكتورة بتحب تخلي القديم والجديد لحتى نقرأوا ونقارنوا الطلاب ليلة الامتحان :-

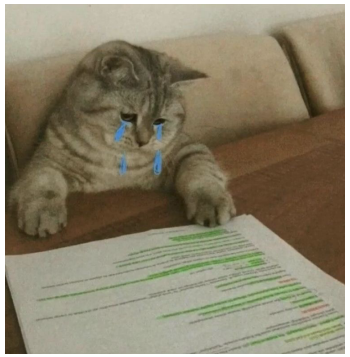
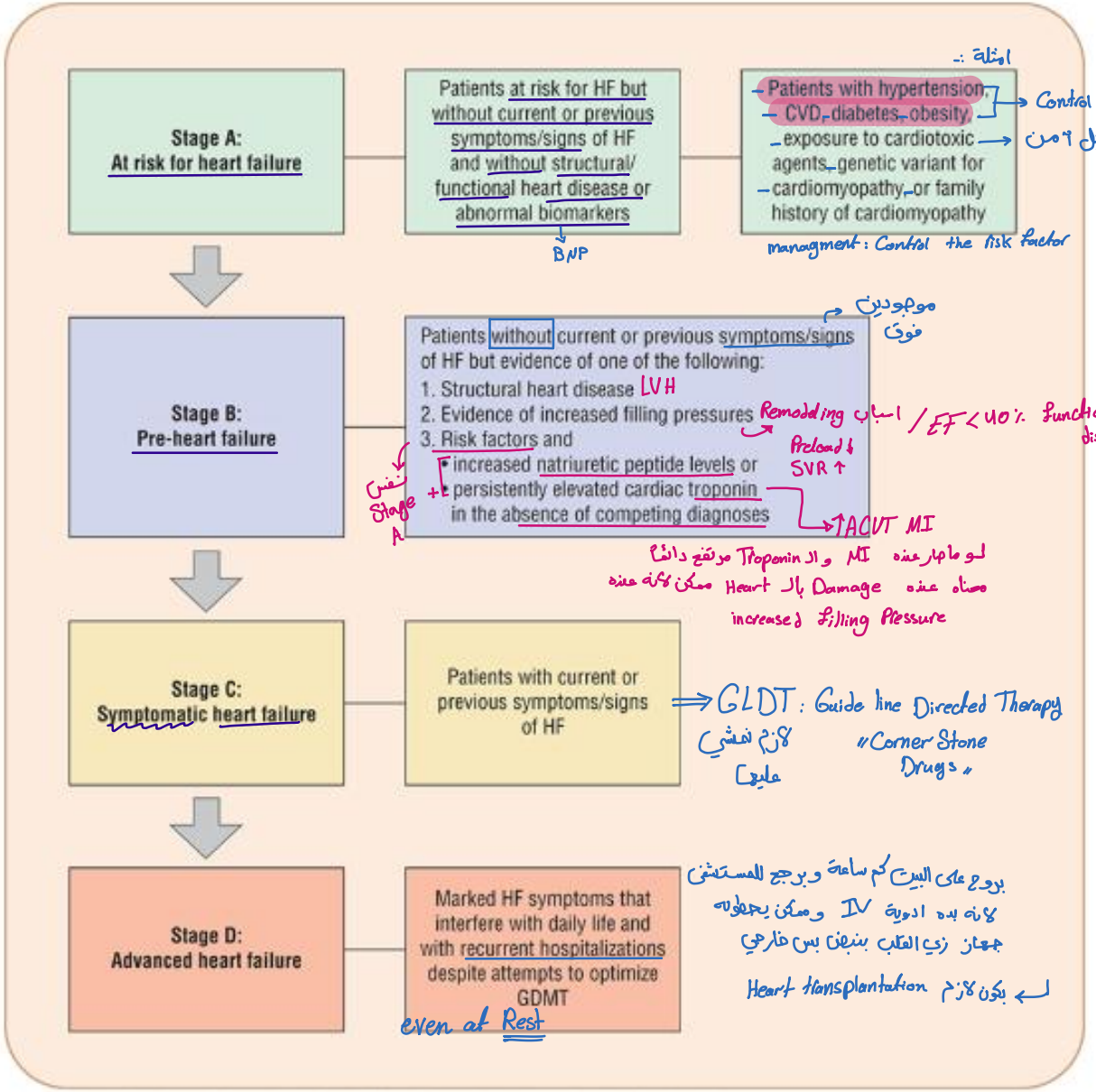


Figure 39-5 Stages of heart failure.

(CVD, cardiovascular disease; GDMT, guideline-directed medical therapy; HF, heart failure.)
(Reprinted, with permission, from Circulation. 2022;145:e876-e894; ©2022 American Heart Association, Inc.)



Source: Stuart T. Haines, Thomas D. Nolin, Vicki L. Ellingrod, Lisa M. Holle, Jennifer Cocohoba, L. Michael Posey: *DiPiro's Pharmacotherapy: A Pathophysiologic Approach, 13th Edition*

- ✓ The four stages differ from the NYHA functional classification with which most clinicians are familiar (useful for monitoring patients).

NYHA FC ≠ ACC/AHA Stages

- ✓ The NYHA system is primarily intended to classify symptoms according to the clinician's subjective evaluation and does not recognize preventive measures or the progression of the disorder.

→ مافي معيار معين يعرف عن طريقه شو درجة الـ Symptoms راح تختلف من دكتور للتاني لانه كل واحد حسب كيف شاف الاعراض

عكس الـ Staging

→ FC 3 : Sever Shortness of Breath
↳ Palmonary edema بسبب
→ FC 1 : بتروح الـ edema وبيي منيح ← optimizing بجعله diuretic

Tow Way *

- ✓ A patient's symptoms can change frequently over a short period of time due to changes in medications, diet, intercurrent illnesses, etc.

لو صار عنده او infection AF

- ✓ For example, a patient with ACC/AHA Stage C HF with NYHA class IV symptoms such as marked volume overload could rapidly improve to class I to II with aggressive diuretic therapy.

4

- ✓ In contrast, and consistent with the progressive nature of HF, a patient's ACC/AHA HF stage could not improve (eg, go from Stage C to Stage B) even though the patient's symptoms could fluctuate from NYHA class IV to I.

NEW YORK HEART ASSOCIATION (NYHA) HEART FAILURE CLASSIFICATION

CLASS I → asympotomatic مع الوضع الطبيعي

✓ NO LIMITATION OF PHYSICAL ACTIVITY; ORDINARY PHYSICAL ACTIVITY DOES NOT CAUSE SYMPTOMS

CLASS II

SLIGHT LIMITATION OF PHYSICAL ACTIVITY; ✓ COMFORTABLE AT REST; ORDINARY PHYSICAL ACTIVITY CAUSES SYMPTOMS بسيطة

لو صارت اسوء →

CLASS III

MARKED LIMITATION OF PHYSICAL ACTIVITY; ✓ COMFORTABLE AT REST, BUT LESS THAN ORDINARY ACTIVITY CAUSES SYMPTOMS اكثر من II

CLASS IV

SEVERE LIMITATION AND DISCOMFORT WITH ANY PHYSICAL ACTIVITY; SYMPTOMS PRESENT EVEN AT REST

WWW.MEDCOMIC.COM

لو هو بمشي كل يوم ساعة وما بتعب لو كان ساعة راح يتعب

→ Asymptomatic on ordinary activity

نفس كلام الرسمة

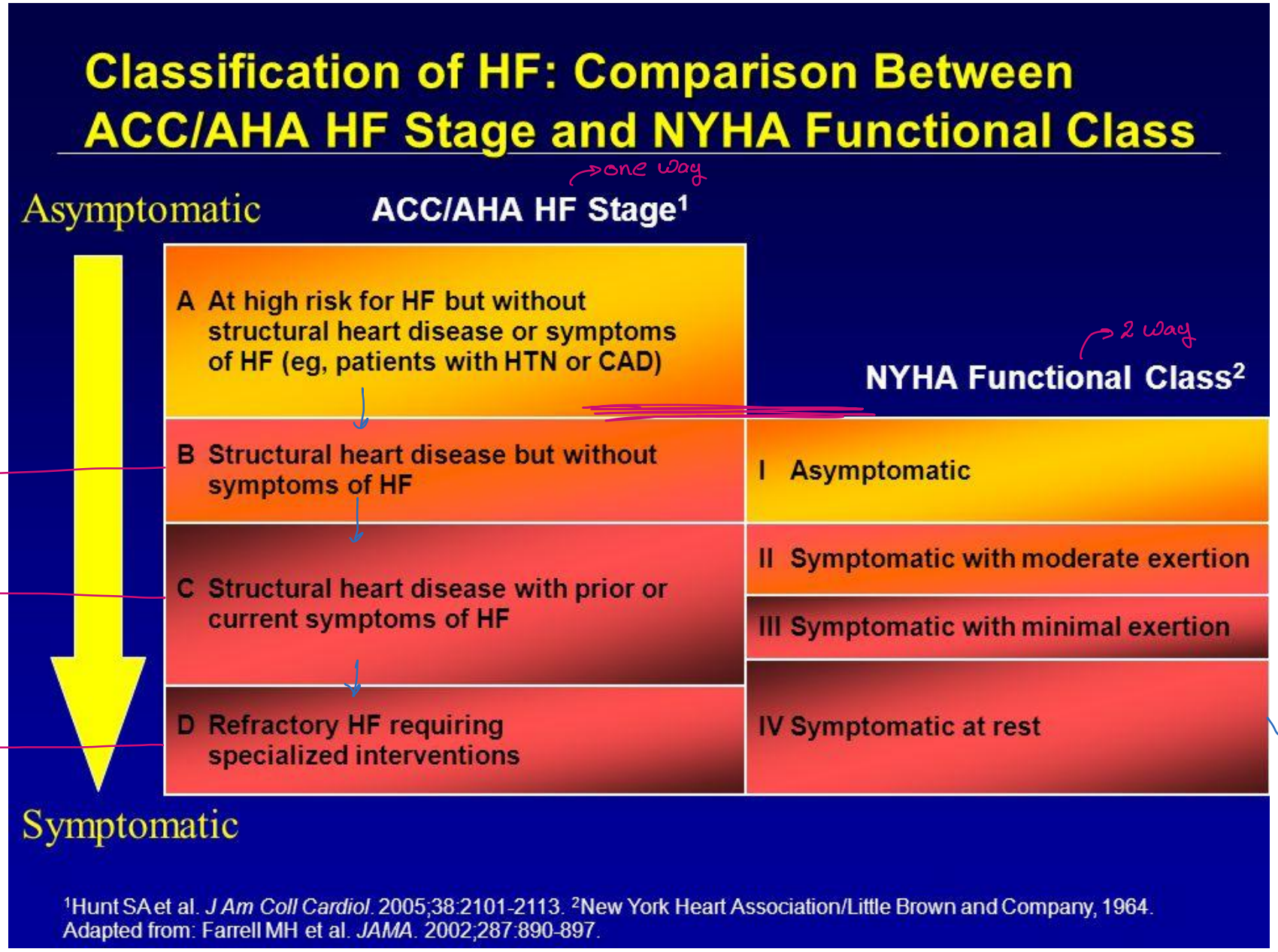
✓ New York Heart Association Functional Classification Functional class:

I. Patients with cardiac disease but without limitations of physical activity. Ordinary physical activity does not cause undue fatigue, dyspnea, or palpitation.

II. Patients with cardiac disease that results in slight limitations of physical activity. Ordinary physical activity results in fatigue, palpitation, dyspnea, or angina.

III. Patients with cardiac disease that results in marked limitation of physical activity. Although patients are comfortable at rest, less than ordinary activity will lead to symptoms.

IV. Patients with cardiac disease that results in an inability to carry on physical activity without discomfort. Symptoms of congestive heart failure are present even at rest. With any physical activity, increased discomfort is experienced.



B & I
ما عندهم اعراض
C & II or III
D & IV

➢ General Measures (including nonpharmacological management)

treat the underlying cause ※ دائمًا

- ✓ The first step in management of chronic HF is to determine the etiology/precipitating factors.
- ✓ Revascularization or anti-ischemic therapy in patients with CHD may reduce HF symptoms.
  ↳ IHD CCD
- ✓ Drugs that aggravate HF should be discontinued if possible.
- ✓ Restriction of physical activity reduces cardiac workload and is recommended for virtually **all** patients with **acute congestive symptoms**. However, once the patient's symptoms have stabilized and excess fluid is removed, restrictions on physical activity are discouraged. ↳ لازم نشجعه انه يقوم بعض الحركة
  لازم يرتاح — Chronic مش
- ✓ Exercise training may improve functional status and quality of life (supported by current guidelines to improve functional status).

- ✓ Restriction of dietary sodium and fluid intake is an important lifestyle intervention for both HFrEF and HFpEF.

لو وزنه زاد خلال يوم ١ كيلو
← سوائل بتكون من دهون

لازم يبعد عن الأكل المالح

- ✓ Mild (less than 3 g/day) to moderate (less than 2 g/day) sodium restriction, in conjunction with daily measurement of weight, should be implemented to minimize volume retention and allow use of lower and safer diuretic doses.

- ✓ Patients should avoid adding salt to prepared foods and eliminate foods high in sodium (eg, salted snack foods, pickles, and processed foods).

← بنترجمها (شيبس، مكرات، أكل جاهز، محلل وزيتون)

Dilution ← من ال

- ✓ In patients with hyponatremia (serum Na less than 130 mEq/L) or those with persistent volume retention despite high diuretic doses and sodium restriction, daily fluid intake should be limited to 2 L/day from all sources.

← الشوربة والعصير والدمتياء اللي فيها سوائل

← اللي منبح عشان edema
Dehydration, Hypotension & kidney function بس ممكن يصير عنده

- ✓ Excessive restriction (Na and fluid) can lead to hypotension, low-output state, and/or renal insufficiency.

صابدنا المريض يوصلك Parameters بحكيله انه ينتبه على محبة انه يقلل ال restriction في

- ✓ Dietary and lifestyle factors that decrease the risk of development of CAD and HTN should be encouraged.

- ✓ Other important general measures include patient and family counseling on the signs and symptoms of HF, detailed written instructions on the importance of appropriate medication use and compliance, activity level, diet, discharge medications, weight monitoring, continuity of care, and the need for close monitoring and follow up to reinforce compliance and minimize the risk of HF exacerbations and subsequent hospitalization.

- ✓ These activities are now referred to as **self-care** and constitute an important means to improve such important outcomes as hospitalization and quality of life.

For your reference

Updated figure in the next slide

هون القديم

Treatment algorithm for patients with ACC/AHA Stage A and B heart failure

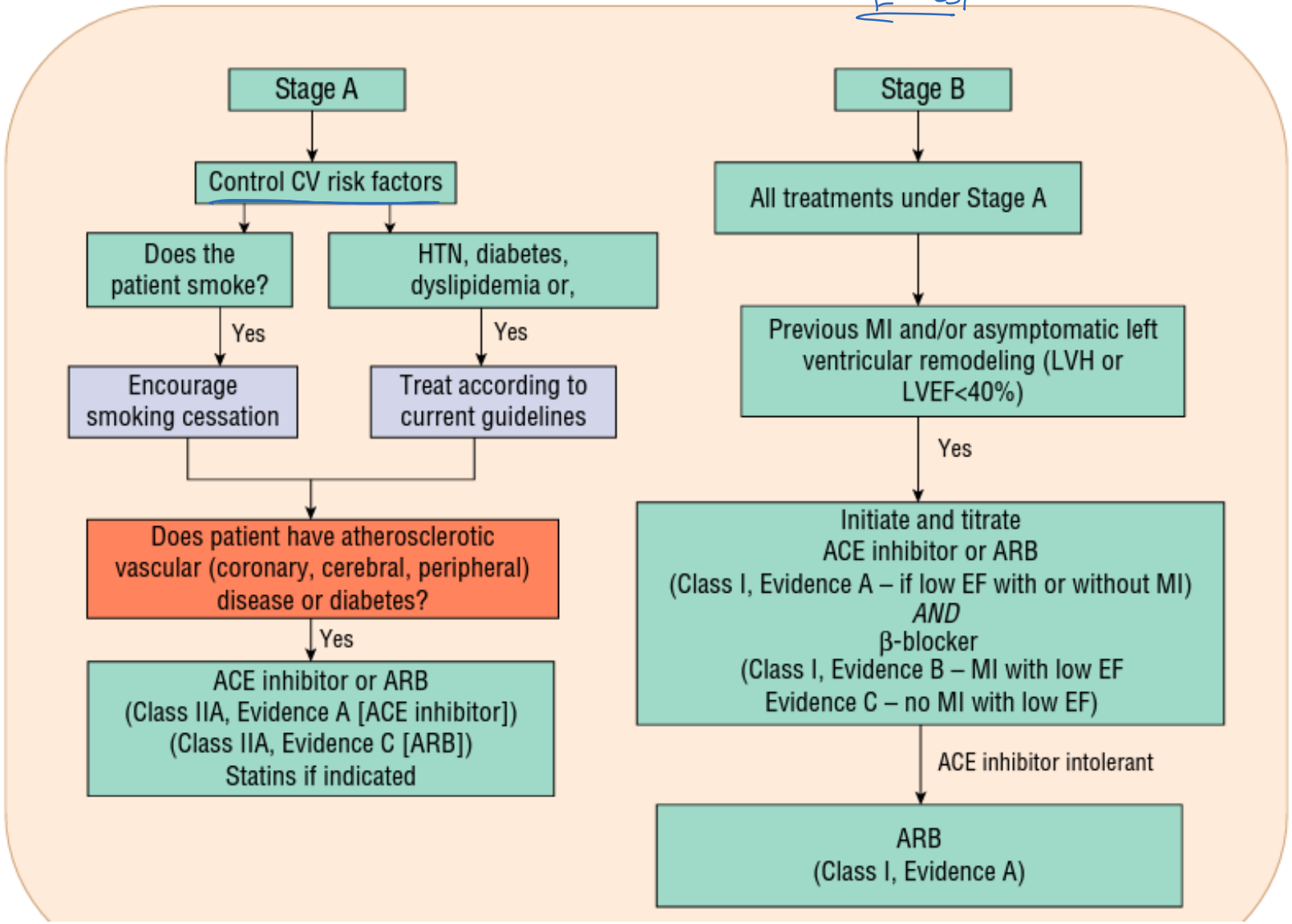
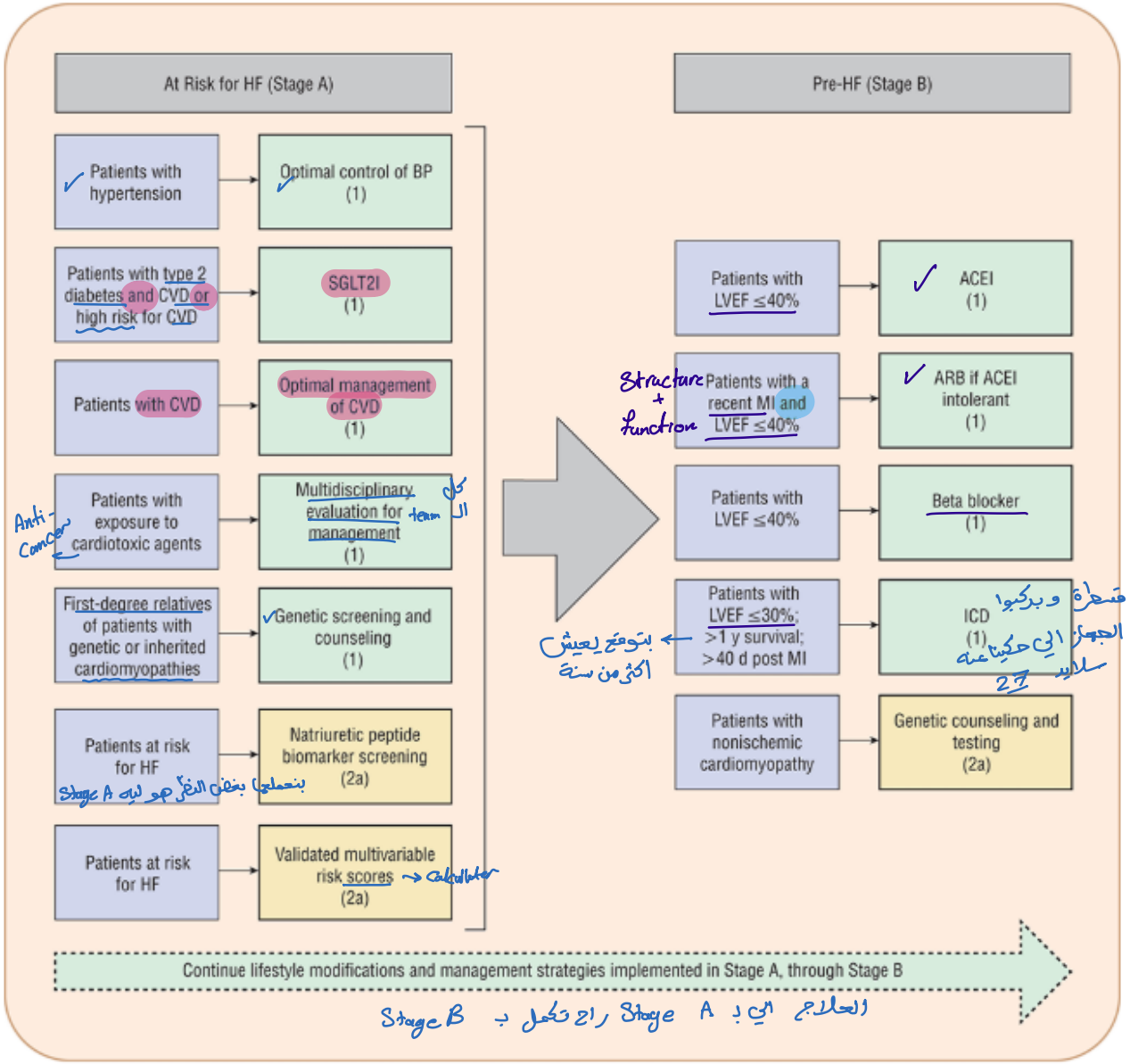


Figure 39-6 Treatment of HFrEF stage A (at Risk for HF) and stage B (pre-HF).

See ACC/AHA/HFSA guidelines for class of recommendation definitions provided under each recommendation (eg, 1, 2a). In brief, **class 1** recommendation refers to conditions for which there is evidence or general agreement that a given procedure/treatment is useful and effective. **Class 2** refers to conditions with conflicting evidence with **2a** having a greater weight/usefulness than **2b**, and **class 3** is for conditions where there is evidence or general agreement that treatment or procedures are not useful/effective or may be harmful. (ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BP, blood pressure; CVD, cardiovascular disease; HF, heart failure; HFSA, Heart Failure Society of America; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; MI, myocardial infarction; SGLT2I, sodium-glucose cotransporter 2 inhibitor.)



هون كل الكلام مكتوب ※

➢ Treatment of Stage A Heart Failure:

- ✓ Patients are at high risk for developing HF because of the presence of risk factors.
- ✓ The emphasis here is on risk factor **identification** and **modification** to prevent the development of structural heart disease and subsequent HF.
- ✓ Commonly encountered risk factors include HTN, dyslipidemia, diabetes, obesity, metabolic syndrome, smoking, and coronary artery disease.
- ✓ Effective blood pressure control reduces the risk of developing HF by approximately 50%; thus, current HTN treatment guidelines should be followed.
- ✓ Although treatment must be individualized, ACE inhibitors or ARBs are recommended for HF prevention in patients with **multiple vascular risk factors**. All DM patients should take a SGLT2 inhibitor for HF prevention.

➢ Treatment of Stage B Heart Failure:

- ✓ Patients in Stage B have structural heart disease, but do not have HF symptoms.
- ✓ This group includes patients with left ventricular hypertrophy, recent or remote MI, valvular disease, or LVEF less than 40%. These individuals are at risk for developing HF, and treatment is targeted at minimizing additional injury and preventing or slowing the remodeling process.
- ✓ In addition to the treatment measures outlined in Stage A, SGLT2 inhibitors (if DM present), ACE inhibitors or ARBs and β-blockers are important components of therapy.
- ✓ All patients with a reduced LVEF should receive an ACEI or ARB and a β-blocker to prevent development of HF, whether or not they have had an MI.
- ✓ Patients with a previous MI and reduced LVEF should also receive an ACEI or ARB, evidence-based β-blockers, and a statin.

For your reference

هون الصح

Updated figure in the next slide

Guideline-directed treatment algorithm for patients with ACC/AHA Stage C heart failure with reduced ejection fraction

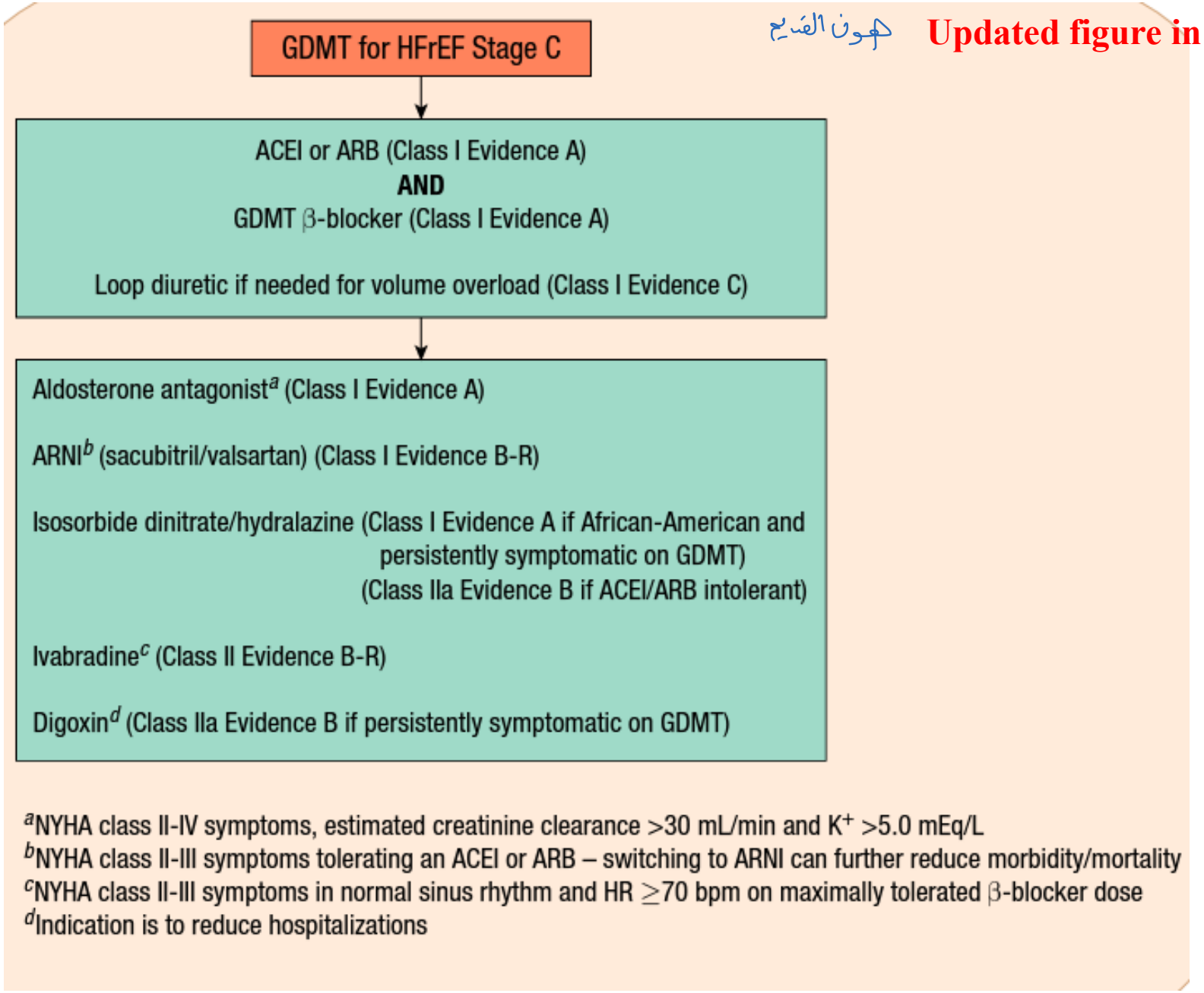
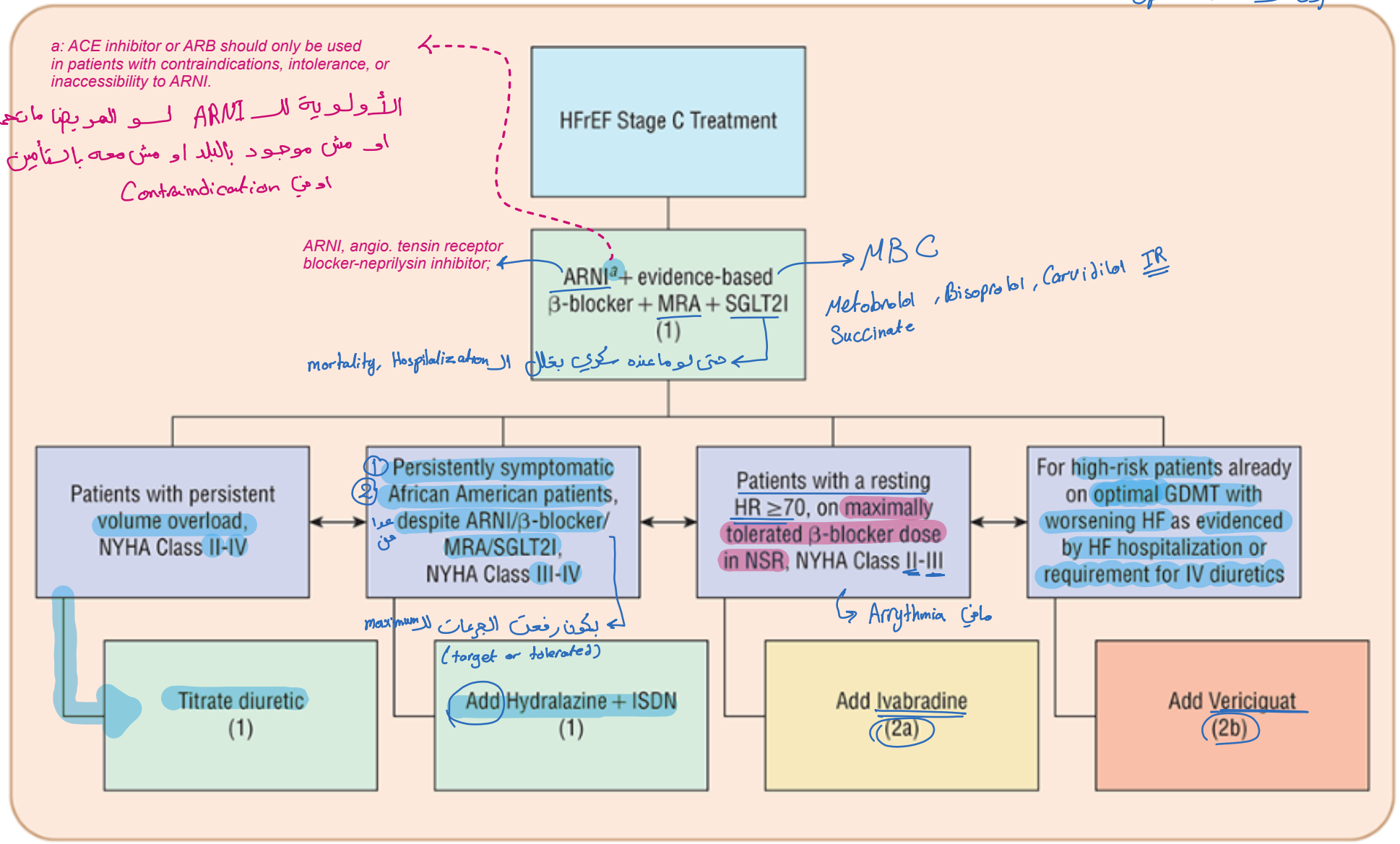


Figure 39-7
Treatment of HFrEF stage C.



Source: Stuart T. Haines, Thomas D. Nolin, Vicki L. Ellingrod, Lisa M. Holle, Jennifer Cocohoba, L. Michael Posey: *DiPiro's Pharmacotherapy: A Pathophysiologic Approach, 13th Edition*

Figure 39-7 Treatment of HFrEF stage C.

See ACC/AHA/HFSA for class of recommendation definitions provided under each recommendation (eg, 1, 2a). In brief, **Class 1** recommendation (green) refers to conditions for which there is evidence or general agreement that a given procedure/treatment is useful and effective. **Class 2** refers to conditions with conflicting evidence with **2a** (yellow) having a greater weight/usefulness than **2b** (orange), and **Class 3** is for conditions where there is evidence or general agreement that treatment or procedures are not useful/effective or may be harmful. **a:** ACE inhibitor or ARB should only be used in patients with contraindications, intolerance, or inaccessibility to ARNI. Evidence-based β-blocker refers to metoprolol succinate, bisoprolol, and carvedilol. (ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor blocker-neprilysin inhibitor; GDMT, guideline-directed medical therapy; HFrEF, heart failure with reduced ejection fraction; ISDN, isosorbide dinitrate MRA, mineralocorticoid receptor antagonist; NSR, normal sinus rhythm; NYHA, New York Heart Association; and SGLT2I, sodium-glucose cotransporter 2 inhibitor.) (Reproduced with permission from Thomas M. Maddox et al. 2024 ACC Expert Consensus Decision Pathway for Treatment of Heart Failure with Reduced Ejection Fraction: A Report of the American College of Cardiology Solution Set Oversight Committee. JACC. 2024;83(15);1444-1488.)

➢ Treatment of Stage C HF:

- ✓ Patients with structural heart disease and previous or current symptoms are classified in Stage C and include both HFrEF and HFpEF.

- ✓ Patients should be routinely treated with guideline directed medical therapy (GDMT) that includes an ARNI (sacubitril/valsartan), ACEI or ARB, an evidence-based β-blocker, SGLT2 inhibitor, MRA, and diuretic therapy.

- ✓ The benefits of these medications on slowing HF progression, reducing morbidity and mortality, and improving symptoms are clearly established.

- ✓ Hydralazine-ISDN can also be used in these patients.

- ✓ Digoxin can also be considered in selected patients (to decrease hospitalizations in patients with HFrEF that remain symptomatic despite GDMT or added during initial treatment of patients with severe symptoms while GDMT is started), as can newly approved medications, ivabradine & others.

- ✓ Nonpharmacologic therapy with devices such as an implantable cardioverter-defibrillator (ICD) is also indicated in certain patients with HFrEF in Stage C.

- ✓ Other general measures noted earlier are also important as is careful follow up and patient education to reinforce dietary and medication compliance to prevent clinical deterioration and reduce hospitalization.

- ➢ **Treatment of Stage D HFrEF:**

- ✓ Stage D HF includes patients receiving maximally tolerated GDMT that have persistent symptoms.

- ✓ This is often referred to as advanced, refractory, or end-stage HF.

- ✓ These patients often undergo recurrent hospitalizations or cannot be discharged from the hospital without special interventions, have a poor quality of life, and are at high risk for morbidity and mortality.

- ✓ These individuals have the most advanced form of HF and specialized therapies including mechanical circulatory support, continuous IV positive inotropic therapy, and cardiac transplantation can be considered in addition to standard treatments outlined in Stages A to C.

24 hr IV infusion
Dobutamine, Dopamine
Milrinone

- ✓ Discussions with the patient and family members regarding prognosis, patient priorities for minimizing symptoms versus prolonging survival and end-of-life and hospice care should be initiated.

نعدّل الـ Remodeling

* لازم الدكتور يحكي مع عيلة المريض
ويفهمهم وضعه + يحكيلهم شو بعملو للمريض

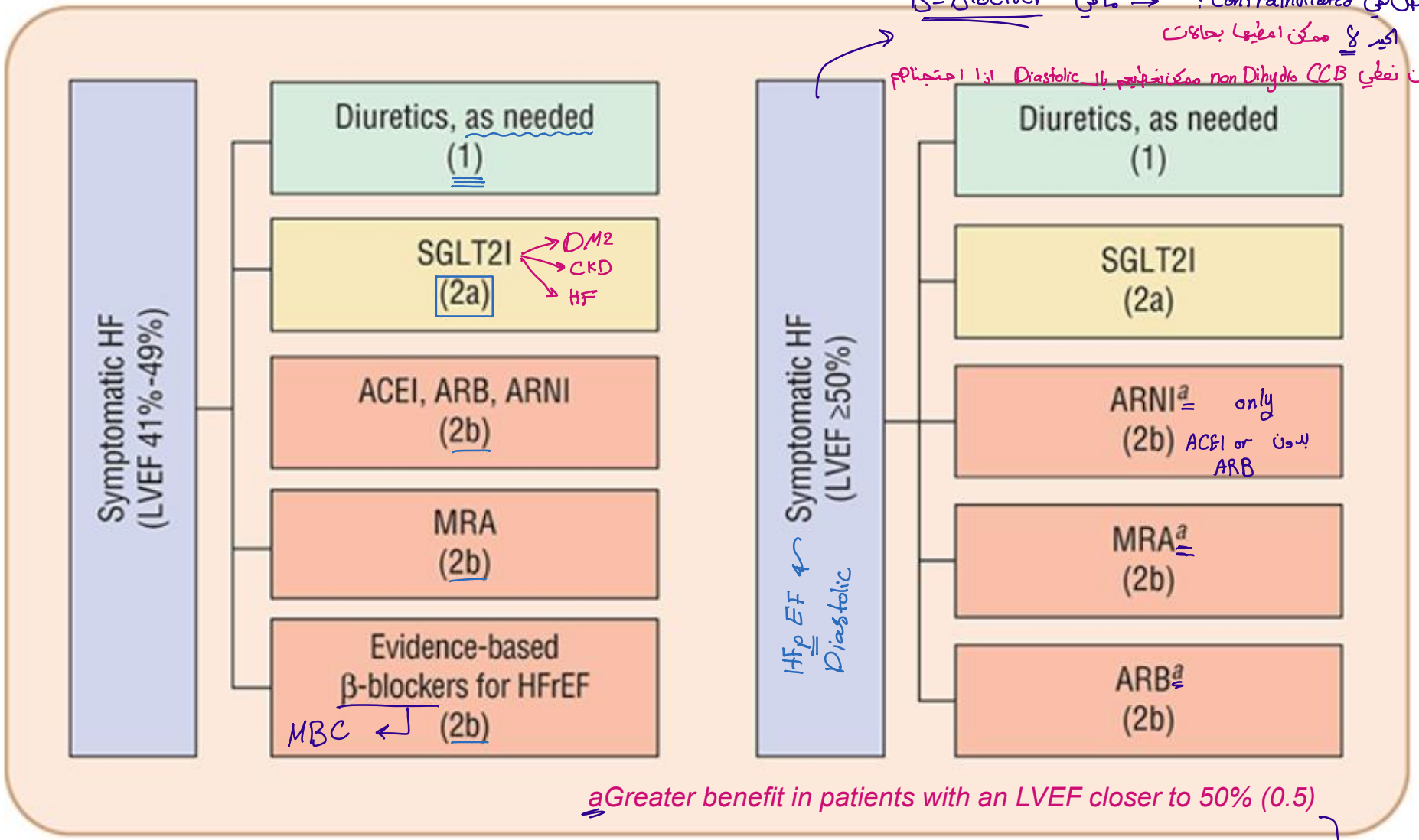
شو هو الجهاز؟ 🫀

👉 *Intra-aortic balloon pump*

جهاز مؤقت لدعم القلب في الحالات الحرجة.

📍 وين بنحطه؟
•قسطرة فيها بالون
•تدخل من الشريان (غالبًا *femoral artery*)
•وتوصل لـ الشريان الأورطي (*Aorta*)

Figure 39-8
Treatment of HFmrEF and HFpEF.



Source: Stuart T. Haines, Thomas D. Nolin, Vicki L. Ellingrod, Lisa M. Holle, Jennifer Cocohoba, L. Michael Posey: *DiPiro's Pharmacotherapy: A Pathophysiologic Approach, 13th Edition*

Figure 39-8 Treatment of HFmrEF and HFpEF.

See ACC/AHA/HFSA guidelines for class of recommendation definitions provided under each recommendation (eg, 1, 2a). In brief, **class 1** recommendation refers to conditions for which there is evidence or general agreement that a given procedure/treatment is useful and effective. **Class 2** refers to conditions with conflicting evidence with **2a** having a greater weight/usefulness than **2b**, and **class 3** is for conditions where there is evidence or general agreement that treatment or procedures are not useful/effective or may be harmful. aGreater benefit in patients with an LVEF closer to 50% (0.5). Evidence-based β-blocker refers to metoprolol succinate, bisoprolol, and carvedilol. (ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; HF, heart failure; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; SGLT2I, sodium-glucose cotransporter 2 inhibitor.)

End of Part 2

oral agents; MOA: increase Glucose secreation in urine Suffix: gliflozin

Summary

اول اشي كان لـ DM2

بعدين هرنا نستخدمه
لـ HF و لـ CKD

TABLE. Recommendations for Use of SGLT2 inhibitors in HF[1]

Stage of HF	Recommendation	COR
Stage A, at risk for HF	To prevent HF hospitalization in patients with T2D who have CVD or are at high risk for CVD	1
Stage C, symptomatic HF		
HFrEF	To reduce HF hospitalization and CV mortality in patients with symptomatic chronic HFrEF, regardless of the presence of T2D	1
HFpEF	To reduce HF hospitalizations and CV mortality in patients with HFpEF	2a
HFmrEF	To reduce HF hospitalizations and CV mortality in patients with HFmrEF	2a

COR, class of recommendation; CVD, cardiovascular disease; HF, heart failure; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; SGLT2, sodium-glucose cotransporter-2; T2D, type 2 diabetes

= Benefits :-

درجة

إذا ماني مابنعطيه بنعطيه اذا في DM2

كمان بـ Stage B بنستخدمها
بنفس الاشي

نفس الاشي

لونها اهبل

For your reference

ACC/AHA/HFSA 2022 Update

※ هدول الدكتورة حكت عنهم نفس اليوم فوق وانه حلو نشوفهم

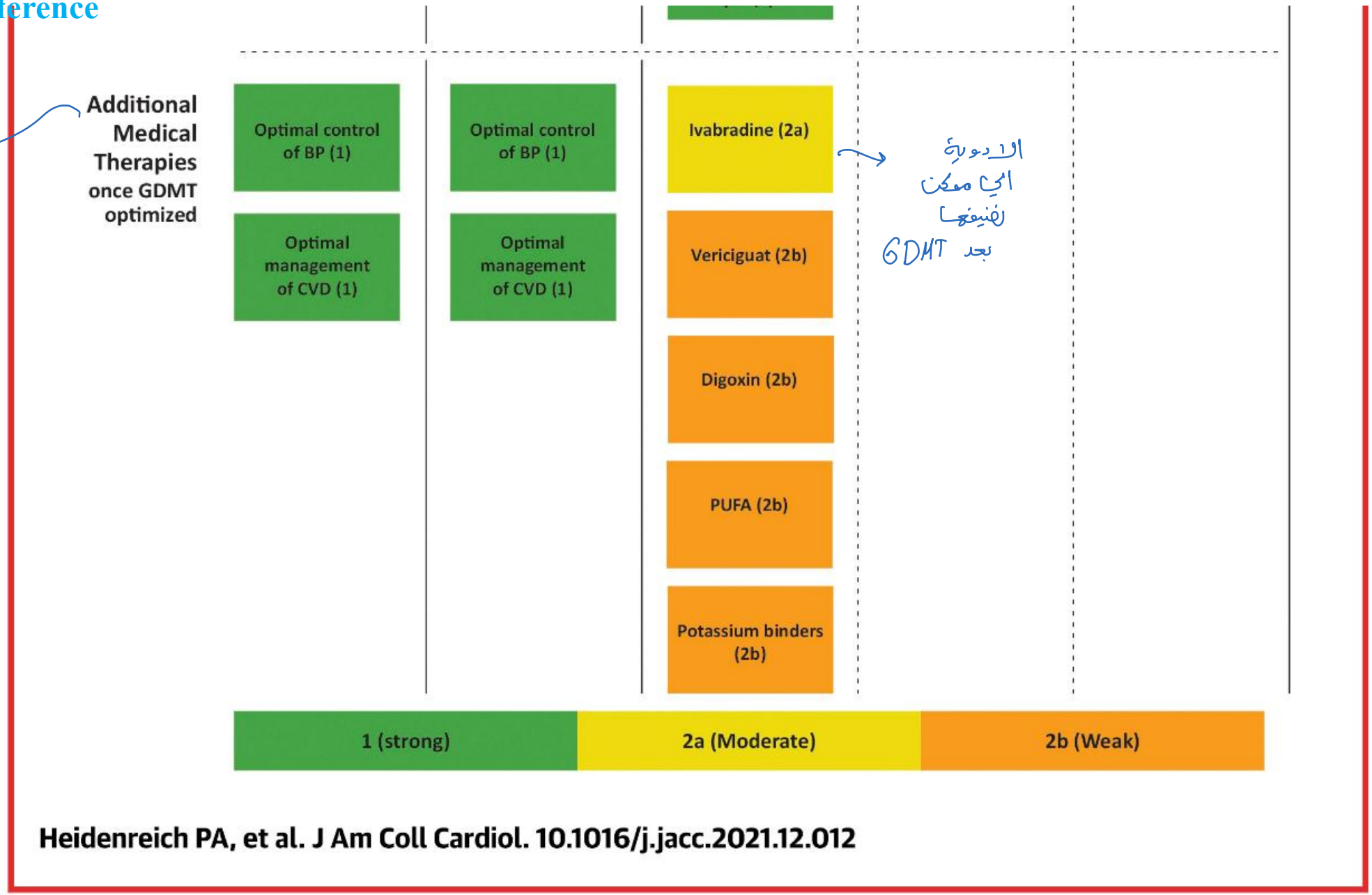
CENTRAL ILLUSTRATION: 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure

Guideline Directed Medical Therapy Across Heart Failure Stages

Use this tool to reference guideline directed medical therapy (GDMT) across the four ACC/AHA stages of Heart Failure (HF) as outlined in the 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure. See the guideline for specific patient population criteria.

	Stage A	Stage B	Stage C & D		
			Stage C: Symptomatic Heart Failure & Stage D: Advanced Heart Failure		
	At-Risk for Heart Failure	Pre-Heart Failure	HFrEF LVEF ≤40%	HFmrEF LVEF 41-49%	HFpEF LVEF ≥50%
GDMT of major medication classes	SGLT2i in pts with DM (1)	SGLT2i in pts with DM (1)	ARNI in NYHA II-III; ACEi or ARB in NYHA II-IV (1)	Diuretics, as needed (1)	Diuretics, as needed (1)
		ACEi (1)	Beta blocker (1)	SGLT2i (2a)	SGLT2i (2a)
		ARB if ACEi intolerant (1)	MRA (1)	ACEi, ARB, ARNi (2b)	ARNi (2b)
		Beta blocker (1)	SGLT2i (1)	MRA (2b)	MRA (2b)
			Diuretics, as needed (1)	Beta blocker (2b)	ARB (2b)
			Hydral-nitrates for NYHA III-IV, in African American pts (1)		

For your reference



Heidenreich PA, et al. J Am Coll Cardiol. 10.1016/j.jacc.2021.12.012

For your reference

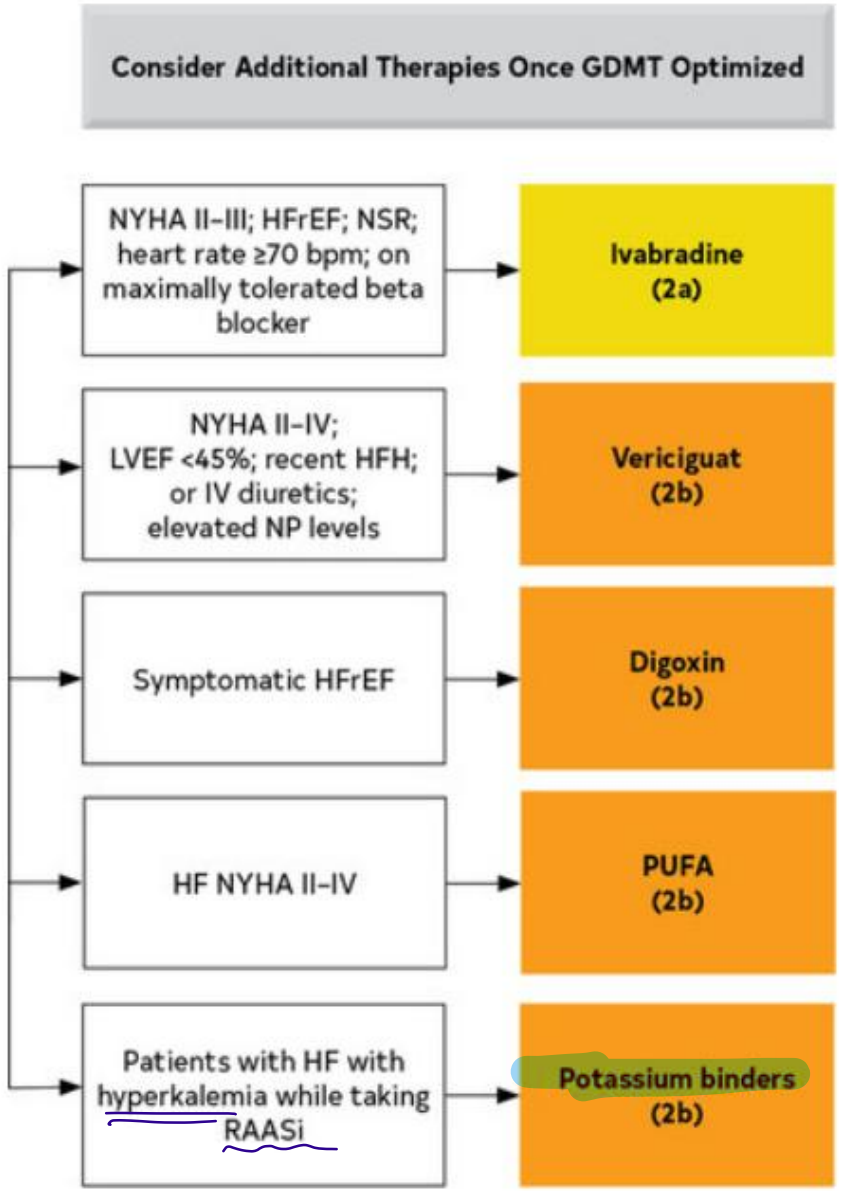


Figure 7. Additional Medical Therapies for Patients With HFrEF. Colors correspond to COR in Table 2. Recommendations for additional medical therapies that may be considered for patients with HF are shown. GDMT indicates guideline-directed medical therapy; HF, heart failure; HFH, heart failure hospitalization; HFrEF, heart failure with reduced ejection fraction; IV, intravenous; LVEF, left ventricular ejection fraction; LVESD, left ventricular end systolic dimension; MV, mitral valve; MR, mitral regurgitation; NP, natriuretic peptide; NSR, normal sinus rhythm; NYHA, New York Heart Association; and RAASi, renin-angiotensin-aldosterone system inhibitors.

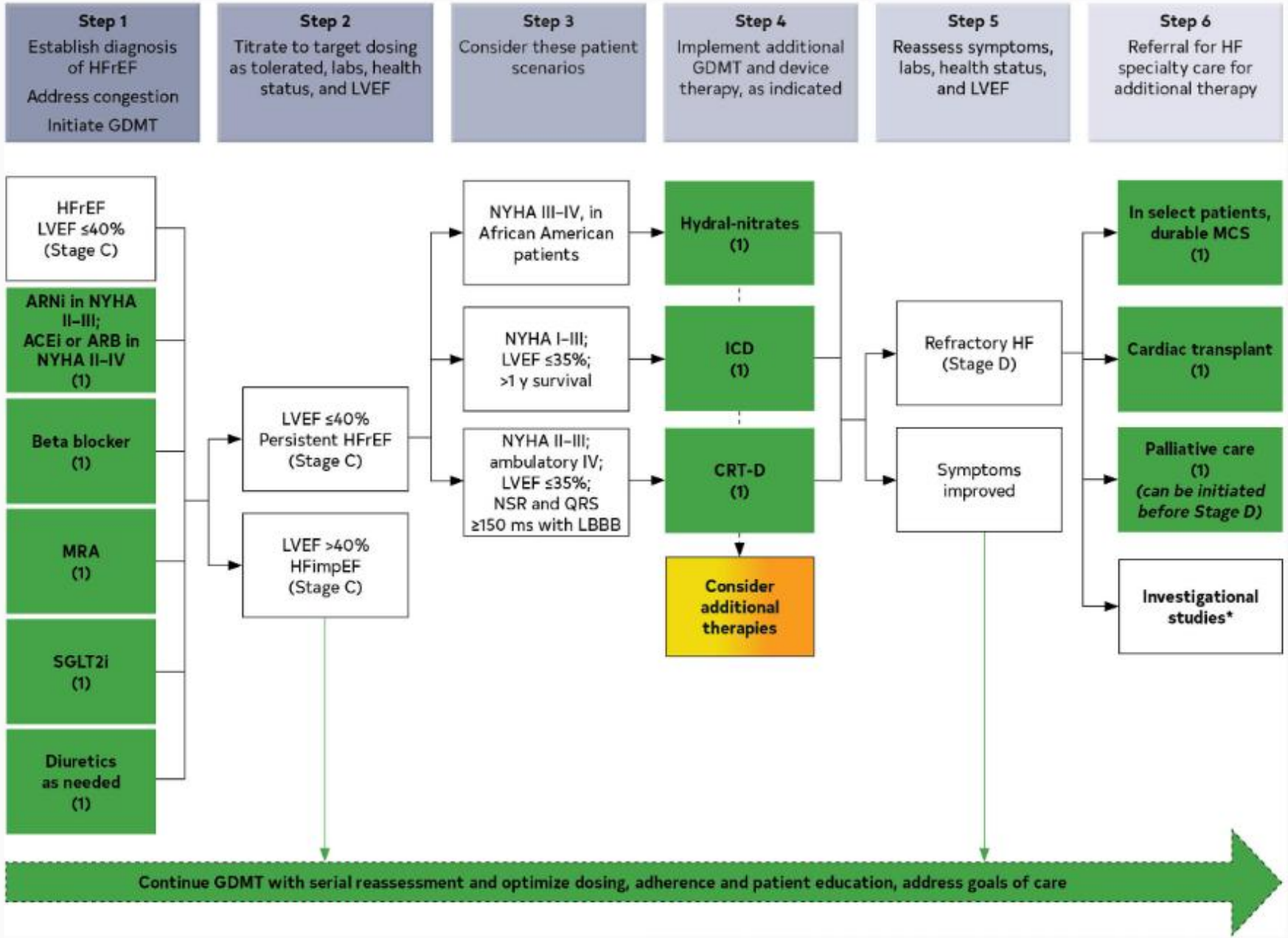
Oral soluble guanylyl cyclase stimulator directly binds and stimulates sGC and increases cGMP production (vasodilation, improvement in endothelial function, decrease in fibrosis and remodeling of the heart).

Omega-3 polyunsaturated fatty acids

الدكتورة رجعت عليهم من الجدول

Patiromer and sodium zirconium cyclosilicate (SZC) - remove potassium by exchanging cations

For your reference



Treatment of HFrEF Stages C and D. Colors correspond to COR in Table 2. Treatment recommendations for patients with HFrEF are displayed. Step 1 medications may be started simultaneously at initial (low) doses recommended for HFrEF. Alternatively, these medications may be started sequentially, with sequence guided by clinical or other factors, without need to achieve target dosing before initiating next medication. Medication doses should be increased to target as tolerated. ACEi indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNi, angiotensin receptor-neprilysin inhibitor; COR, Class of Recommendation; CRT, cardiac resynchronization therapy; GDMT, guideline-directed medical therapy; ICD, implantable cardioverter-defibrillator; hydral-nitrates, hydralazine and isosorbide dinitrate; HFrEF, heart failure with reduced ejection fraction; LBBB, left bundle branch block; MCS, mechanical circulatory support; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; NSR, normal sinus rhythm; NYHA, New York Heart Association; and SGLT2i, sodium-glucose cotransporter 2 inhibitor. *Participation in investigational studies is appropriate for stage C, NYHA class II and III HF.

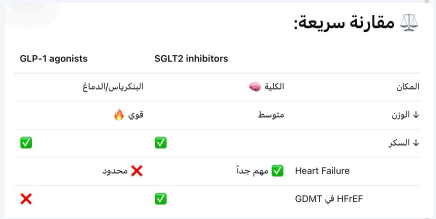
وحملت الدكتورة هون ✳
← الدكتورة ركزت على اشياء كانت حاكيتها

Table 39-8 Cardiovascular and Noncardiovascular Comorbidities – Part 1

CV Comorbidity +HF	Recommendations and Comments
1) Hypertension	• Treat according to ACC/AHA guideline for the prevention, detection, evaluation, and management of high BP in adults • Target BP <130/80 mm Hg, unless evidence for symptomatic hypotension, labile BP, or observed impact on kidney dysfunction • Preferred therapies: diuretic agent, ARNI, ARB, MRA • In HFrEF, addition of hydralazine/ISDN or a dihydropyridine CCB (amlodipine, felodipine) may be considered • Both verapamil and diltiazem should be avoided in HFrEF but can be safely used in HFpEF; however, use caution with aggressive HR lowering
2) Dyslipidemia	Treat according to current AHA/ACC/Multi-society guideline on the management of blood cholesterol and the ACC ECDP on the role of nonstatin therapies for LDL-cholesterol lowering in the management of ASCVD risk
3) Coronary artery disease CAD	• Treat according to current ACC/AHA/SCAI guideline for coronary artery revascularization and the ACC/AHA/ASE/CHEST/SAEM/SCCT/SCMR guideline for evaluation and diagnosis of chest pain • Surgical revascularization in appropriate patients • β-Blockers and nitrates are preferred antianginals for patients with both HFrEF and CAD • ✓Amlodipine and felodipine are safe in HFrEF, while nondihydropyridines and CCBs (verapamil, diltiazem) should be avoided • β-Blockers and verapamil are safe and have neutral effects on outcomes in HFpEF; however, use caution with aggressive HR lowering given low SV at rest and poor SV reserve during exertion • In patients with HFpEF, routine use of nitrates or phosphodiesterase-5 inhibitors to increase activity or QOL is ineffective, and long-acting nitrates appear to hinder activity level • Optimize therapies for secondary prevention of coronary and atherosclerotic vascular disease
4) Atrial fibrillation AT	• Treat according to the current ACC/AHA/ACCP/HRS guideline for the management of patients with AF • Anticoagulation as indicated • Rate vs rhythm control guided by HF symptoms • AF ablation recommended, if symptoms attributable to AF and HFrEF or HFpEF (*less evidence in HFpEF*) • AV node ablation with CRT implantation recommended, if LVEF ≤50% (0.5) and rhythm control strategies fail or are undesirable (*less evidence*) • In HFrEF, β-blockers are more effective for rate control than digoxin and improve morbidity and mortality. Combination of β-blockers and digoxin may be more effective at rate control. Nondihydropyridine CCBs (verapamil, diltiazem) should be avoided • In HFpEF, β-blockers and nondihydropyridine CCBs are often considered first-line agents for HR control; however, as described above, aggressive rate control should be avoided • Exercise caution when treating with digoxin • In HFrEF, amiodarone and dofetilide are preferred for rhythm control, and avoid class I antiarrhythmics and dronedarone

Table 39-8 Cardiovascular and Noncardiovascular Comorbidities – Part 2

Non-CV Comorbidity	Recommendations and Comments
5) Diabetes	• Treat according to the current ACC ECDP on novel therapies for CV risk reduction in patients with T2DM and ADA standards of medical care in diabetes • Consider consulting with endocrinologist • SGLT2 inhibitors as first-line therapies for comorbid T2DM and HF regardless of EF • GLP-1 receptor agonists are an option in individuals with high CV risk and/or obesity → ozempic • Finerenone in diabetic + kidney disease *Mineralocorticoid Receptor Antagonists (MRAs) Non-steroidal* ⭐ لكن • Avoid thiazolidinediones and DDP-4 inhibitors (saxagliptin, alogliptin) Edema بعملوا
6) Chronic kidney disease	• Treat according to KDIGO clinical practice guidelines for the evaluation and management of CKD and guideline for diabetes management in CKD • Optimize RAAS inhibitor therapy ACEI, ARB, ARNI • Use hydralazine/ISDN if an ARNI/ACE inhibitor/ARB cannot be used بديل العجم + Add on لـ African American • Treat with SGLT2 inhibitor if GFR allows • Consider potassium binders for hyperkalemia → Chronic Kidney disease + الادوية - Hyperkalemia → local بكونوا K Binders بنعطي in GI → K بنعطوا امعاهم • Consider nephrology consult



ACC, American College of Cardiology; ACCP, American College of Clinical Pharmacy; ACE, angiotensin-converting enzyme; ADA, American Diabetes Association; AF, atrial fibrillation; AHA, American Heart Association; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; ASCVD, atherosclerotic cardiovascular disease; ASE, American Society of Echocardiography; AV, atrioventricular; BP, blood pressure; CCB, calcium channel blockers; CHEST, American College of Chest Physicians; CKD, chronic kidney disease; CRT, cardiac resynchronization therapy; CV, cardiovascular; DDP-4, dipeptidyl peptidase-4; ECDP, Expert Consensus Decision Pathway; GDMT, guideline-directed medical therapy; GFR, glomerular filtration rate; GLP-1, glucagon-like peptide-1; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; HR, heart rate; HRS, Heart Rhythm Society; ISDN, isosorbide dinitrate; KDIGO, Kidney Disease Improving Global Outcomes; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; MRA, mineralocorticoid receptor antagonist; QOR, quality of life; RAAS, renin-angiotensin-aldosterone system; SAEM, Society for Academic Emergency Medicine; SCAI, Society for Cardiovascular Angiography & Interventions; SCCT, Society for Cardiovascular Computed Tomography; SCMR, Society for Cardiovascular Magnetic Resonance; SGLT2, sodium-glucose cotransporter-2; T2DM, type 2 diabetes mellitus.

2- Dyslipidemia

Goals عندي

- Managment of Blood Cholesterol
  By: antiCholesterlemic agents
- " of ASCVD risk
  By: dyslipidemia agent „Statin"
  mortality بتقلل الكوليسترول وال ←

B- Blockers ←

لازم نبدأ بجرعة قليلة وتحمل بعدها

Titration up بعد مش اقل من اسبوعين

- ⛔ تقلل تأثير NE (SNS)
- 🔽 تقلل toxic overstimulation على القلب
- 🛡️ تمنع:
  - Remodeling
  - Arrhythmias
  - Cell death

📌 النتيجة:

- ↓ mortality ✅
- ↓ hospitalizations ✅
- تحسن EF مع الوقت ✅

يعني رغم إنها تقلل القوة بالبداية...
لكنها "تحمي القلب" على المدى الطويل

2. ليش Non-DHP CCBs (verapamil, diltiazem)
❌؟

👉 لأنها تعمل:

- ⛔ Negative inotropy (تقلل الانقباض) قوي
- ❌ بدون أي فائدة على:
  - SNS
  - RAAS
  - Remodeling

📌 النتيجة:

- 😬 أكثر ↓ contractility
- ↓ CO
- ممكن تزيد الحالة سوء

يعني:
تضعف القلب... بدون ما تعالج السبب

بحالة HFrEF ◦ Systolic
لكن ممكن نستعمله مع HFpEF ◦ diastolic

ولكن لازم اشبه كمان على HR انه ما يقل كثير

$CO = HR * SV$

وما بدي HR↓↓ → CO↓↓ حيث يعيد

Table 39-9 Specific Patient Subpopulations

Subpopulation	Recommendations and Comments
Pregnancy	• Multidisciplinary management and GDMT with reasonable safety profile • Pregnancy contraindicated for women with LVEF <30% (0.3) (NYHA class III/IV) or history of PPCM with LVEF <50% (0.5) (WHO class IV) • Baseline and monitor BNP/NT-proBNP • ACE inhibitors, ARB, ARNI, and MRA are contraindicated in pregnancy • Avoid hypotension and over-diuresis • Preferred HF agents: β-blockers, diuretics, ISDN, hydralazine • Following delivery, if breastfeeding then assess drug distribution of current HF therapies into breast milk
Patients with cancer	• Requires multidisciplinary management and treatment plan will vary based on the type of cancer, the cancer therapy used, and other factors specific to the patient • ARB, ACE inhibitor, and β-blocker GDMT for select asymptomatic patients with cancer therapy–related cardiomyopathy (LVEF <50% [0.5])
African American patients, self-identified	• GDMT • Angioedema risk possibly higher with ACE inhibitors and ARBs compared to White patients • Angioedema risk with ARNIs may not be different from White patients
Patients living with frailty per established criteria	• GDMT as tolerated • Uncertain response to GDMT, treat as tolerated • Possible increased risk of adverse drug reactions
Adults ≥75 years	• GDMT but recognize that this population is excluded from many trials supporting GDMT • Consider starting with lower doses of GDMT • Common potential risks: falls, worsening of kidney function, polypharmacy, comorbidity, depression, financial toxicity

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; GDMT, guideline-directed medical therapy; HF, heart failure; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; (NT-pro)BNP, (aminoterminal-prohormone) B-type natriuretic peptide; NYHA, New York Heart Association; PPCM, peripartum cardiomyopathy; WHO, World Health Organization

Drug Dosing Table

Drug	Initial Dose	Usual Range	Special Population	PK/PD Considerations, Key Drug Interactions, Other Concerns
Loop Diuretics				When used in combination with thiazide diuretics, there is an additive risk of hypokalemia, hypomagnesemia, and volume depletion with related renal dysfunction.
Furosemide	**20-40 mg once or twice daily**	**20-160 mg once or twice daily**	Clcr 20-50 mL/min: 160 mg once or twice daily Clcr<20 mL/min: 400 mg daily	Single doses exceeding those listed are unlikely to elicit additional response
Bumetanide	0.5-1.0 mg once or twice daily	1-2 mg once or twice daily	ClCr 20-50 mL/min: 2 mg once or twice daily Clcr<20 mL/min: 8-10 mg daily	Single doses exceeding those listed are unlikely to elicit additional response
Torsemide	10-20 mg once daily	10-80 mg once daily	ClCr 20-50 mL/min: 40 mg once daily Clcr<20 mL/min: 200 mg daily	Single doses exceeding those listed are unlikely to elicit additional response
Guideline-Directed Medical Therapy for HFrEF and HFpEF				
1) ACEIs:				All ACEIs: Additive risk of hyperkalemia with other medications that increase serum potassium (eg, potassium supplements). Additive risk of renal dysfunction with diuretics, NSAIDs, and other nephrotoxins
1)Captopril	**6.25 mg three times daily**	**50 mg three times daily***		

Drug	Initial Dose	Usual Range	PK/PD Considerations, Key Drug Interactions, Other Concerns
2) Enalapril	**2.5 mg twice daily**	**10-20 mg twice daily***	
3) Lisinopril	2.5-5.0 mg once daily	20-40 mg once daily*	
4) Ramipril	1.25-2.5 mg	5 mg twice daily*	
5) Fosinopril	5-10 mg once daily	40 mg once daily	Undergoes both hepatic and renal elimination
1) ARBs:			All ARBs: Additive risk of hyperkalemia with other medications that increase serum potassium (eg, potassium supplements). Additive risk of renal dysfunction with diuretics, NSAIDs, and other nephrotoxins
Candesartan	**4 mg once daily**	**32 mg once daily***	Candesartan is a prodrug
Valsartan	20-40 mg twice daily	160 mg twice daily*	
Losartan	25-50 mg once daily	150 mg once daily*	Prodrug
1) Angiotensin Receptor Blocker/Neprilysin Inhibitor:			
Sacubitril/ Valsartan (Entresto)	49/51 mg twice daily 24/26 mg twice daily if not taking or low dose of ACE inhibitor/ARB or if eGFR is <30 mL/min/1.73 m2	97/103 mg twice daily *	Discontinue ACE inhibitors at least 36 hours prior to initiation Additive risk of hyperkalemia with other medications that increase serum potassium (eg, potassium supplements) Additive risk of renal dysfunction with diuretics, NSAIDs, and other nephrotoxins

Drug	Initial Dose	Usual Range	Special Population	PK/PD, Key Drug Interactions, Other Concerns
2) β-blockers:				
Bisoprolol	1.25 mg once daily	10 mg once daily*		
Carvedilol IR	**3.125 mg twice daily**	**25 mg twice daily***	**Target dose for patients weighing >85 kg is 50 mg twice daily**	Should be taken with food
Carvedilol phosphate	10 mg once daily	80 mg once daily		Should be taken with food
Metoprolol succinate CR/XL	**12.5-25 mg once daily**	**200 mg once daily***		
3) MRAs: Aldosterone Antagonists				- If eGFR 30-49 mL/min/1.73 m2: reduce dose or frequency - Risk of hyperkalemia increases if serum creatinine >1.6 mg/dL (141 µmol/L). Avoid if baseline potassium is >5 mEq/L (mmol/L) - Additive risk of hyperkalemia with other medications that increase serum potassium (eg, ACE inhibitors, ARBs, ARNIs, potassium supplements)
Spironolactone	eGFR ≥50 mL/min /1.73m^2: **12.5-25 mg once daily**	**25-50 mg once daily***	eGFR 30-49 mL/min/1.73m^2: 12.5 mg once daily or every other day	
Eplerenone	eGFR ≥50 mL/min /1.73m^2: 25 mg once daily	50 mg once daily*	eGFR 30-49 mL/min/1.73m^2: 25 mg every other day	Eplerenone is a substrate of CYP3A4
Finerenone (Kerendia)	10 mg once daily	20 mg once daily		Finerenone is currently only FDA-approved for chronic kidney disease with type 2 diabetes

Drug	Initial Dose	Usual Range	PK/PD Considerations, Key Drug Interactions, Other Concerns
4) SGLT2 Inhibitors:			Increased risk of ketoacidosis and volume depletion in patients with diabetes بزيدوا الـ Diuresis ممكن يعملوا UTI و ممكن يعملوا Dehydration
Dapagliflozin (Farxiga)	10 mg once daily	10 mg once daily	Dapagliflozin should not be initiated if eGFR ≤25 mL/min/1.73 m2
Empagliflozin (Jardiance)	**10 mg** **once daily**	**10 mg** **once daily***	نفس الاشي
Sotagliflozin (Inpefa)	200 mg once daily	400 mg once daily	Sotagliflozin should not be initiated if eGFR ≤25 mL/min/1.73 m2 Sotagliflozin may increase digoxin exposure

Add on

Drug	Initial Dose	Usual Range	PK/PD Considerations, Key Drug Interactions, Other Concerns
Additional therapies for HFrEF or HFpEF or other cardiomyopathies			
Hydralazine-Isosorbide Dinitrate (Bidil) (fixed-dose combination) -or- Individual agents not in fixed-dose combination	Fixed-dose combination: 37.5-mg hydralazine plus 20-mg isosorbide three times daily Individual agents: 25-50 mg hydralazine plus 20-30 mg isosorbide dinitrate 3-4 times daily	75-mg hydralazine plus 40-mg isosorbide three times daily * 300-mg total hydralazine plus 120-mg total isosorbide dinitrate daily in divided doses	Indicated in conjunction with GDMT in patients self-identified as African American with NYHA class III-IV HFrEF to improve symptoms and reduce morbidity and mortality Indicated in current or previous symptomatic HFrEF who cannot be given first-line agents, such as ARNI, ACE inhibitor, or ARB, because of drug intolerance or renal insufficiency to reduce morbidity and mortality

Drug	Initial Dose	Usual Range	PK/PD Considerations, Key Drug Interactions, Other Concerns
Additional Tx for HFrEF or HFpEF or other cardiomyopathies			
Digoxin	**0.125-0.25 mg once daily**	**Titrate dose according to age, lean body weight, and renal function; individualized, variable dose to achieve serum digoxin concentration of 0.5-0.9 ng/mL (0.6-1.2 nmol/L)**	Indicated in conjunction with GDMT in symptomatic HFrEF despite GDMT (or who are unable to tolerate GDMT) to decrease hospitalizations for HF
Ivabradine	Initiate 5 mg twice daily, increase at 2 weeks to 7.5 mg twice daily to achieve a resting HR of 50-60 beats/min If HR <50 beats/min, may reduce dose to 2.5 mg twice daily Take with food		Indicated with GDMT in symptomatic, NYHA class II-III, stable chronic HFrEF (LVEF ≤35%) including a β-blocker at maximum tolerated dose, and who are in sinus rhythm with an HR ≥70 beats/min at rest, to reduce HF hospitalizations and cardiovascular death Contraindicated with strong CYP3A4 inhibitors Additive risk of bradycardia with other medications with negative chronotropic effects (eg, digoxin)

Drug	Initial Dose	Usual Range	PK/PD Considerations, Key Drug Interactions, Other Concerns
Additional Tx for HFrEF or HFpEF or other cardiomyopathies			
Ferric carboxymaltose دواء جديد (Injectafer) حديد	On day 1: Hgb ≤14 g/dL (140 g/L; 8.69 mmol/L): 1,000 mg, Hgb 14-15 g/dL (140-150 g/L; 8.69-9.31 mmol/L): 500 mg بتعتمد على قيمة Hgb وعلى الوزن At week 6: maintanance Dose بنعمل ال 12 وعلى الأسبوع يعني بعد 3 monthes If <70 kg, 500 mg if Hgb <10 g/dL (100g/L; 6.21 mmol/L), otherwise no dose If >70 kg, 1,000 mg if Hgb <10 g/dL (100g/L; 6.21 mmol/L), 500 mg if Hgb 10-14 g/dL (100-150 g/L; 6.21-8.69 mmol/L): 100, and no dose if Hgb 14-15 g/dL (140-150 g/L; 8.69-9.31 mmol/L)	Indicated for iron deficiency in adult patients with NYHA class II-III HF to improve exercise capacity Administer a maintenance dose of 500 mg at weeks 12, 24, and 36 if serum ferritin <100 ng/mL (mcg/L; 225 pmol/L) or serum ferritin 100-300 ng/mL (mcg/L; 225-674 pmol/L) and transferrin saturation <20% 6 بعد 3 اشهر 9 No dosing data available after 36 weeks or with Hgb ≥15 g/dL (150 g/L; 9.31 mmol/L) ما بنعطيه

*: Regimens proven in large clinical trials to reduce mortality.

* راح احطلكم جنبهم Highlight الدكتورة حطت , أغلبه مكرر + ملاحظات

Drug Monitoring Table

Drug Class	Adverse Effect	Monitoring Parameters	Comments
ACEIs	Angioedema, cough, hyperkalemia, hypotension, renal dysfunction	BP, electrolytes, BUN, and creatinine	* Contraindicated in patients with bilateral renal artery stenosis, history of angioedema, or pregnancy. Assess BP, BUN, creatinine, and electrolytes at baseline and 1-2 weeks after initiation or increase in dose. Goal is target dose from clinical trials or highest tolerated. ≠ Minimum Effective Dose (معهمة حكت عنها الدكتورة / ممكن المريض ما يتحمل)
ARBs	Hyperkalemia, hypotension, renal dysfunction	BP, electrolytes, BUN, and creatinine	Contraindicated in patients with bilateral renal artery stenosis or pregnancy. Assess BP, BUN, creatinine, and electrolytes at baseline and 1-2 weeks after initiation or increase in dose. Use with caution in patients with a history of ACEI-associated angioedema. Goal is target dose from clinical trials or highest tolerated.
Sacubitril/ valsartan	Angioedema, hyperkalemia, hypotension, dizziness, renal dysfunction	BP, electrolytes, BUN, and creatinine	* Contraindicated in patients with a history of angioedema associated with ACEI or ARB therapy or in pregnancy. Assess BP, BUN, creatinine, and electrolytes at baseline and 1-2 weeks after initiation or dose increase. Start with a low dose and double the dose every 2-4 weeks as tolerated based on BP, serum potassium, and renal function. Goal is target dose from clinical trials or highest tolerated.

Drug Monitoring Table

Drug Class	Adverse Effect	Monitoring Parameters	Comments
Aldosterone antagonists	Gynecomastia/breast tenderness/menstrual irregularities (spironolactone), hyperkalemia, worsening renal function	BP, electrolytes, BUN, and creatinine	Assess BP, BUN, creatinine, and electrolytes at baseline. Check potassium 3 days and 1 week after initiation and then monthly for the first 3 months. Change to eplerenone if gynecomastia develops with spironolactone.
β-blockers	Bradycardia, heart block, bronchospasm, hypotension, worsening HF	BP, HR, ECG, signs and symptoms of worsening HF, blood glucose	Start with low dose and titrate upward no more often than every 2 weeks as tolerated based on BP, HR, and symptoms. Goal is target dose from clinical trials or highest tolerated. Patients may feel worse before they feel better.
Digoxin	GI and CNS adverse effects, brady- and tachyarrhythmias	electrolytes, BUN, creatinine, ECG, serum digoxin concentration	Target serum digoxin concentration 0.5-0.9 ng/mL.
SGLT2i	UTIs, Genital mycotic infections, polyuria, polydipsia, hypotension, dehydration, bone fracture.	Renal function, BP, Lipids, Pregnancy test (before), HbA1c (DM)	Before initiation: 1. Assess renal function beforehand and periodically thereafter 2. Correct condition in patients with volume depletion

Drug Monitoring Table

Drug Class	Adverse Effect	Monitoring Parameters	Comments
Ivabradine	Bradycardia, hypertension, atrial fibrillation, luminous phenomena (phosphenes, transiently enhanced brightness in a portion of the visual field)	BP, HR, ECG	Start with 5 mg twice daily and after 2 weeks adjust dose to achieve a resting HR 50-60 BPM. Only use in patients in sinus rhythm.
Diuretics	Hypovolemia, hypotension, hyponatremia, hypokalemia, hypomagnesemia, hyperuricemia, renal dysfunction, thirst	BP, electrolytes, BUN, creatinine, glucose, uric acid, changes in weight, JVD	Dose should be adjusted based on volume status, renal function, electrolytes, and BP. Reassess these parameters 1-2 weeks after dose changes. Goal is lowest dose that maintains euvolemia.
Hydralazine	Hypotension, headache, rash, arthralgia, lupus, tachycardia	BP, HR	
Nitrates	Hypotension, headache, lightheadedness	BP, HR	
Ferric carboxymaltose	Hypersensitivity, hypophosphatemia, HTN, flushing, injection site reaction, erythema, rash, nausea, vomiting, dizziness, headache	Serum ferritin, Hgb, Hct, BP, blood phosphate levels	Observe hypersensitivity during and after administration for at least 30 minutes and until clinically stable following completion of each administration.

Class 1

➢ Diuretics

Rapid Symptomatic Reliver

- Diuretic therapy, in addition to sodium restriction, is recommended in **all** patients with clinical evidence of fluid retention.

non pharmaco

edema → pulmonary

- Once fluid overload has been resolved, many patients require chronic diuretic therapy to maintain euvolemia input = output

بدنا نحافظ على الوضع اللي وصلناه

mentainace dose

Lowest effective Dose → Rational: euvolemia + Safty + electrolites

- Diuretics do not prolong survival or alter disease progression, therefore are not considered mandatory therapy (patients who do not have fluid retention would not require diuretic therapy).

- The primary goal of diuretic therapy is to reduce symptoms associated with fluid retention, improve exercise tolerance and quality of life, and reduce hospitalizations from HF.

IV furosomide

- Diuretics accomplish this by decreasing pulmonary & peripheral edema (reduction of preload).

- Diuretic therapy must be used carefully because overdiuresis can lead to a reduction in CO, renal perfusion, and symptoms of volume depletion.

بالتوفيق + دعواتكم

لحد هون مادة الطب ✗ مادة السكند مكملين