Colour Cardiology

(A Primer)

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Ver 3.0

Dedication Ver 1.0

I dedicate this booklet to the Pharmaceutical Industry. If it were not for their duplicity, self-interests, and profit driven actions I would not have this healthy mistrust of anything pharma related (I just wish they would stop doing such a good job of it), and I encourage all residents to seek their own knowledge and not believe biased pharma reps.

Dedication Ver 3.0

Version 3 of this Primer is dedicated to the medical students, residents and doctors that have all struggled through the COVID-19 pandemic. You do not have unions, you do not have overtime or hazard pay, and you do not have breaks. You work, tireless, selfless, for the patients. You have donned and doffed, and donned and doffed, and donned and doffed…used alcohol till your skin cracked, pulled your masks down for a few seconds of fresh air when no one was looking, and maybe sometimes teared up a bit when you were alone. You risked your lives, and risked your loved ones lives, but always working, exhausted, never taking time off, never saying no when you are needed, beaten and abused by this virus but never defeated. I dedicate this booklet to all of you.

Ver 1.0 June 2016
Ver 2.0 June 2018
Ver 3.0 June 2021

Contents

1. Introduction

Welcome to Cardiology!

This booklet was written with the intent of providing residents, especially our new residents, with a preliminary source to help guide them through their Cardiology rotation at Abbotsford Regional Hospital and Cancer Center (ARHCC).

You may find that some areas of the primer are oversimplified and we've designed it that way to help orient new residents to the content as well as address the tiny gaps in knowledge that one may have on the topic. We hope that you use this primer as a reference for review prior and during your rotation experience.

By the end of your Cardiology rotation, it is the department's intention that you will have a broad and multifaceted understanding of this specialty and be able to better manage patients and act in critical situations.

Let's begin.

2. Anatomy of Cardiology at ARHCC

Your role in Cardiology is the cardiology consult service resident, in addition to outpatient clinics. Other than your day role, you are also expected to do call as the cardiology resident.

The Cardiac Care Unit (CCU) is located on Yale 2. Right across from the CCU is the Emergency Department, and between those 2 locations is where you will be spending most of your rotation. Having said that, consults can originate from anywhere in the hospital, and it would be a good idea to familiarize yourself various wards as you may be needed to get there STAT.

We also rotate the residents through various Cardiology clinics that we have (details below).

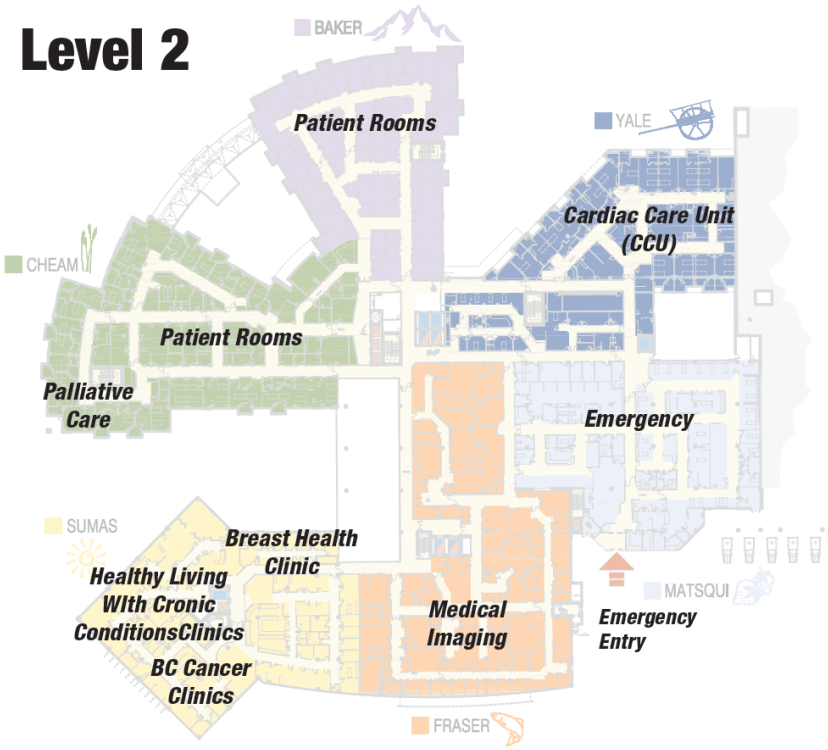

Figure 1

This would be the Chest Pain Clinic, the Cardiac Rehab Clinic (both on the 3rd floor of the Fraser Wing in Diagnostic Services, one floor above Medical Imaging) as well as the Heart Function Clinic (2nd floor in the Sumas Wing in the "Healthy Living with Chronic Conditions" Clinic) (see Figure 1).

What will you do?

2A. Consult Service

The consult service aims to provide prompt and effective Cardiology consultation to the Emergency Department, the various wards throughout ARHCC, as well as telephone consults from community physicians and other hospitals in the region (from Langley to Hope).

- The consult service is available from Monday through Friday from 0800 - 1700. Cardiology services are provided outside those hours by the on-call Cardiologist.

- From 0800 -1200 everyday your Attending will be the Cardiologist on the consult service. However, from 1200 -1700 the CCU Cardiologist will be cross covering the consult service as well. Please keep that in mind during your rotation, and of course we will advise you before the start of your time with us.

The goal is your education! If you have any questions or concerns, please take the initiative and contact me or any Cardiologist in our group. Our goal is to support your learning journey and to build your confidence in Cardiology as this will contribute to your practice in helping patients everywhere (mired with as few lawsuits as possible!).

Your Day on the Unit

- As the Cardiology resident on the consult service, you are expected to arrive at 0800 and call/text/page your Attending to see if there are any consults left over from the night before. If this is your first contact with that Attending it is good idea to introduce yourself and discuss what you hope to get out the rotation as well as how the Attending would like the week to run.

- Get the patient's information (name, location, reason for consult…etc.) and go see them. Conducting a thorough history and physical exam is key. Then formulate a plan and contact your Attending when you are done.

- One of the things that we hope to teach you during your rotation is **prioritization**. You will often receive more than one consult at a time and you need to determine who needs to be seen first. STEMI, Cardiogenic Shock, active chest pain…obviously should be seen before the 90-year-old who had some mild dizziness 4 days ago.

- Another key to success in your rotation is **time management**. We expect thoroughness and a focus on relevant information. Previous cardiac investigations and diagnosis are certainly relevant…the fact that the patient has a stable rash for the past 12 years not so much.

- The majority of consults you will see on the consult service are Afib, ACS, Syncope, and CHF. Refer to the primer's Appendix II for articles on how to approach these conditions.

- If there are no consults, I encourage you to go to your Attending and ask for teaching. Pick a topic, anything you want, and ask that they go over it with you. Be patient though; it can be very busy on cardiology and often the Attending is tied up with other patients. However, whatever quiet time you have you should use that to expand your mind to the wonderful nuances of the myocardium!

- At the appropriate time you can also ask your Attending to visit the CCU or in any of the specialized Cardiology Clinics we have to learn more.

- Your day is done (assuming you are not on call) by 1700. However, like any busy service, there will be times when you find yourself staying late to finish dictating or completing admission orders. There will be no more new consults after 1700.

Finally, though some of the patients can be exquisitely sick, always know that YOU ARE NOT ALONE. The Attending will always back you up. Your job is to assess the patient, make decisions you are comfortable with, and then call someone. They are NOT your responsibility and you will only be blamed for something if you don't call!!

Important points when on service

- **Touch base promptly at 0800 with your Attending.**
- **Prioritize your patients. See the sicker ones first.**
- **Good history and physical exam are key!**
- **Always ask questions!**

Good luck!

2B. Cardiac Care Unit (CCU)

The CCU at ARHCC is where the sicker, more acute patients end up. Some residents do electives with us in CCU, but usually they are R2 and above, and will hopefully by the end of their rotation gain some confidence in dealing with more acute cardiac cases than you would typically see on the consult service.

CCU rounds usually start around 8:30-9 am. There is a different Cardiology Attending on CCU than the consult service physician, but the CCU physician covers both CCU and the consult service in the afternoons (as above). Our CCU has 24 beds, with varying degrees of illness and acuity.

If you have some down time, and would like to round with the CCU Attending, it is a great opportunity to listen to interesting murmurs, and perhaps assist in various interventions like central lines, temporary pacemakers, and arterial lines.

Notes:

2C. Clinics

Heart Failure Clinic

The Heart Failure Clinic is located on the 2nd Floor in the Sumas Wing. It is a great clinic run by the Cardiologists as well as a Nurse Practitioner, RN's, in addition to dietitian and pharmacist. As the name implies, we deal with OUTPATIENTS who have heart failure, a chronic and debilitating disease. The goal of the clinic is to optimize anti-failure medications, provide education and resources, as well as minimize patient presentations to the ER by providing them with a relatively quick access to a health care professional with expertise in Heart Failure management. The Cardiologists attends the clinic on Wednesdays, so if you have some quiet time you are welcome to come by and join. It will give you excellent exposure to the various medications we use, as well as some experience in looking at the JVP!

Chest Pain Clinic and Cardiac Rehab

Both clinics are in the 3rd floor Diagnostic Services (Fraser Wing), and run in parallel on Tuesdays and Thursdays from 1230 until about 1600 (Chest pain only without cardiac rehab are Mondays, Wednesday and Fridays). The Chest Pain clinic is a "rapid access" clinic for any patients that are seen in the ER and deemed "not sick enough" to be seen by the Cardiology consult service, but "too sick" to be followed in the routine outpatient clinical scenario (that can sometimes take months). They are usually seen within 24-48 hours in the Chest Pain clinic from discharge from the ER.

The Cardiac Rehabilitation Clinic is a very popular clinic dealing with cardiac rehabilitation for patients post ACS, PCI, bypass/valvular surgery, as well as stable angina and heart failure. I strongly recommend that residents join if they are able to as the community family physicians often use the rehab clinic as a resource and this is a good opportunity to understand and observe what happens to your patients when they are referred to the clinic. On Tuesdays and Thursdays, we risk stratify them with an exercise stress test to determine their eligibility for the program, in addition to providing education about the various exercises they are able to perform. The rest of the week the patients are educated about medications, diet, as well as advised on psychological and group support. A cardiac event is a very scary thing and it's important that we provide as much support as we can to our patients surround them with a good support system.

2D. On Call

Ah yes, the joy of being on call. Remember the rules of being on call:

1. Always call the Attending with anything you are uncomfortable with.
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3. Always call the Attending with anything you are uncomfortable with.
4. Prioritize your consults – sicker patients are seen first.
5. Eat when you can, pee when you can.
6. Very sick patients in CCU that you are dealing with take priority over ER.
7. Always call the Attending with anything you are uncomfortable with.
8. Call starts at 1700 and ends at 2300 (you are not in house overnight).

When you start your call, be in contact with your Attending at 1700 and ask if there are any leftover consults from the day and go from there.

When you are on call, your responsibility is any consults to cardiology (which is mainly emerg) as well as the CCU. One of the key things you will learn from being on call is to prioritize. Sick patients need to be seen first of course, regardless of where they are. Having said that, CCU is your priority because you are the resident for that unit; the rest of the hospital and emerg have physicians available to take care of their patients. If a patient is hemodynamically stable and is pain free, they can wait until you address the sicker ones. The good news is you will rarely be called to deal with a sick patient in CCU. Usually the patients have been stabilized by the CCU Attending during the day and most issues the on-call Cardiologist can deal with over the phone.

We do our best to get patients moving from emerg to the ward/CCU. Unfortunately, often we have no beds across the hall, so they have to wait in emerg. Please make sure that these patients have all their notes written and all their orders put in, including echo reqs, angio reqs, and prn meds, so that the emerg nurses have clear guidance how to care for the patient until morning.

You will likely get several consults from emerg during your night on call; remember to prioritize. Discuss with your Attending beforehand how you would like to manage the patients (i.e. discuss each case with your Attending individually or see a few then call and review them together…etc.).

Finally, I would like to mention the **Code Blue process**. While we are not expected to go to any Code Blue that occurs, it's a great teaching opportunity and I would encourage you to gain exposure to the Code if you are not with any consults at the time. The exception is if it is a Code Blue in CCU – then we EXPECT you to be there and to assist. I'm not suggesting running the Code but use it as a learning opportunity and assist the ER doc (who runs the Code) and call your Attending if we are not there.

Important points when on call

- **Always call the Attending with anything you feel uncomfortable handling.**
- **PRIORITIZE! Ongoing chest pain, hemodynically unstable, see immediately!**
- **Be present at Code Blue if it occurs in CCU. But don't worry you won't have to run it (unless you want to!)**

Notes:

3. The Cardiac Physical Exam

One of the finest points of Cardiology is the ability to tell so much from the cardiac physical exam. Unfortunately, it is becoming a lost art, as echo becomes more and more accessible.

Having said that, this is your opportunity to refine your physical exam skills of the heart.

- Examine every patient thoroughly.
- Report on what you see/feel/hear.

Don't worry yet about its significance, just be able to recognize that there is a murmur, and describe it properly. Over time, the picture will fall into place and you will have a better understanding of what it all means. I will only discuss two points of the cardiac physical exam here; the rest I encourage you to look up!

3A. JVP

Though pulmonologists don't examine the lungs, and I can't remember the last time a gastroenterologist examined for the spleen, no Cardiologist can get away with not looking at the JVP. If I had to pick the most important component of the cardiac physical exam for the resident to master it would be interpretation of the JVP. No more is it acceptable to say the JVP is "elevated". You need to know how high, which wave is dominant, and comment on the x and y descent of it. Nothing will save your neck more in the middle of the night than a proper assessment of the JVP.

How to examine:

Though I recommend reading up on this, I will give a short primer on the important points of the JVP exam. Please ask your Attending to demonstrate for you and keep doing it with every patient (and anyone else you meet!) and you will master it.

Before we begin though, please take time to understand what you are seeing as shown in Figure 2.

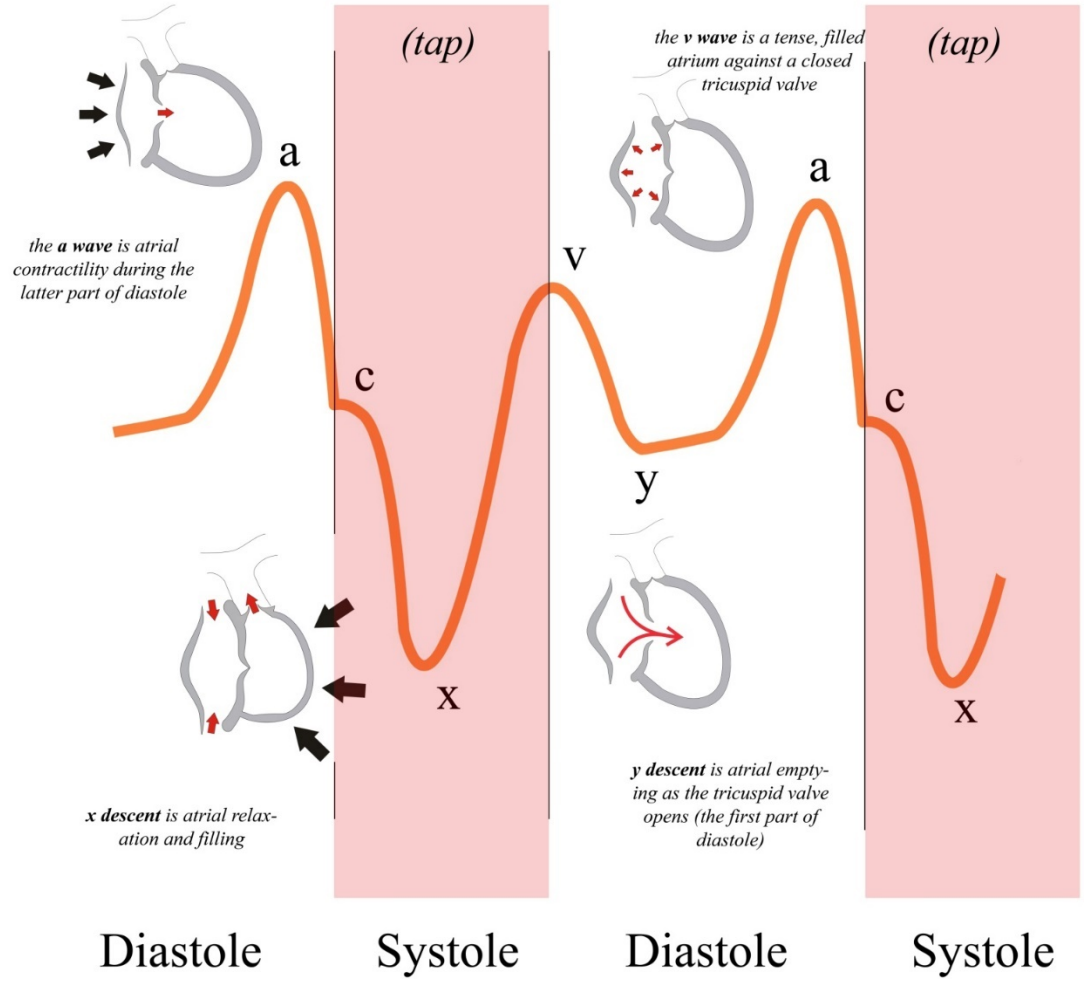


Figure 2

- Bed at 45 degrees.
- Stand to the right of the patient.
- Pt head should be tilted up and slightly away from you.
- Proper lighting.
- Good visualization of the neck.
- Look at between the two heads of the sternomastoid and the clavicle.
- I know you know all this.

If you get anything out of this booklet get the following sentence:

To properly examine the JVP, ALWAYS palpate the opposite carotid artery simultaneously as you look at the JVP.

This is key. You are taught all the time of the five different ways to differentiate between carotid and JVP. Well, sure, that is ok, but a much easier way to do it is to palpate the opposite carotid, and as you feel that "tap-tap-tap" of the carotid with your fingers you can observe the JVP and easily differentiate between the two pulsations (arterial carotid vs. venous jugular). You can also easily differentiate between the a wave and v wave that way. Of course, patients with multiple TIAs or carotid endarterectomy or significant vagal pauses with carotid massages, well…you may want to just go to the old five ways method!

As you look at the patient's neck (always palpating the opposite carotid!), focus on that pulsation, and ask yourself the following questions:

- How high is it? Always have a ruler. We want accurate numbers not just "high."
- Which wave is dominant? A or V?
- How about that x and y descent? Present, absent, blunted?

The answer to the first question (how high is the JVP, or is it flat?) is one of the best clinical clues that you will have when you have a patient in distress on the ward (any ward) or CCU.

- Do you give furosemide or fluid?
- Is the patient dyspneic from pulmonary edema or COPD/pneumonia?

The JVP is key. And I will tell you something that every Cardiologist will deny and don't say it out loud or they will chew you out, but…there is actually very good evidence that assessing the EXTERNAL jugular (which is waaaaay easier than assessing the internal jugular vein) is just as good when establishing the height of the JVP. So you can just look at the external jugular (usually a bulging vein across the neck) to assess height of JVP, but NOT wave dominance nor descent...sorry, for that you need to be a purist.

It is easy if you are timing it with the "tap-tap-tap" of the opposite carotid. The A wave is prior to the tap, the V wave is after the tap. Which is larger? Over time you will be proficient in telling them apart.

What about the descent? How can I tell one from the other? The easiest way would be to time it with S2 on auscultation (x descent occurs WITH S2, while y descent occurs AFTER S2), but the BEST way is to (you guessed it) time it with the carotid pulsation that you are palpating. Each "tap" will coincide with the x descent, while the y descent will occur after the tap. Though you may feel this is complex and overwhelming it just requires practice, keep at it and ask the Attending with you to show you time and time again. You will get it, and you will then be a king amongst men when it comes to physical examination skills!

Important points when examining the JVP

- **ALWAYS palpate the opposite carotid**
- **Comment on JVP height, wave dominance, and x and y descents**

I won't go into detail here about special maneuvers, but take the time to read about hepato-jugular reflex and Kussmaul's sign…common questions on rounds and also helpful with your physical exam.

3B. Auscultation

Describing cardiac auscultation and the various murmurs/heart sounds is beyond the scope of this booklet. There are a few important points I would like to mention and that is an approach and description of what you hear during your cardiac examination.

You should have a systematic approach to listening to the heart.

- Patient lying at 45 degrees supine
- Apply the bell to the skin at the apex.
- During auscultation listen both specifically and selectively for heart sounds and then for murmurs, first during systole and then during diastole.
- In each area examined (you know, aortic, pulmonic, tricuspid, and mitral), you should listen first for the S1.
- This is followed by listening for the S2, noting the presence of splitting and variation with respirations.
- Then extra sounds are searched for and carefully listened to, first in systole and then in diastole.
- Instruct the patient to roll onto the left side. and selectively listen to diastole and the low-frequency range.
- Then have the patient in the sitting position. Re-examine the 4 precordial areas. While the patient leans slightly forward during quiet respiration, you can optimally appreciate splitting of S2.
- With the patient's breath held in deep expiration, examine the aortic and pulmonic areas with the diaphragm, selectively tuning in to the high-frequency range in an effort to hear the faint blowing diastolic murmur of AR or, if the clinical situation warrants, the presence of a pericardial friction rub.

As for murmurs, when you hear it, describe it. Don't worry about putting it all together; just describe what you hear. Mainly, comment on the following 4 things:

- **Location:** Where is it heard loudest and where does the sound radiate to?
- **Timing:** Early diastolic, pan systolic, etc.
- **Grade** or **Intensity**: Rank from 1 to 6.
- **Quality** and **Shape** of the heart sound: Is there a musical crescendo, harsh snap, etc.?

Important points when auscultating

- **Describe accurately what you hear.**
- **Have a thorough, routine method for all patients.**
- **Identify location, timing, grade or intensity, and quality and shape of the heart's sound.**
- **The more you examine patients, the better you will understand what you hear.**

A good physical examination book will help but practicing your listening is your best teacher.

Notes:

4. Electrocardiogram

The ECG is an extremely useful tool in the Cardiologists arsenal. It is cheap, non-invasive, and reproducible. It also provides us with a great deal of information.

I'm assuming that you have some basic knowledge of interpreting ECGs. By your rotation's completion, the expectation is that you will have developed a reasonable ability at recognizing some key findings. No one expects you to detect Brugada Syndrome but if you miss a STEMI you're in deep dog poop.

As pointed out, the goal of this booklet is to create a starting point to help you through this rotation, and give you some pointers that should help you on the wards/CCU and while on call.

Please find the time to:

- Read a good ECG book
- Attend ECG rounds (usually once a month during your academic half day)
- Be prepared to ask us plenty of questions. This is your best opportunity to understand something that you will be seeing for the rest for your medical career.

Let's get started!

4A. Vectors, Axis, blah blah blah

This is a complex concept of ECG, and I will simplify it to the best of my ability, insofar as much as you need for your rotation. If you want to do Cardiology then expand on this, but if you just want to survive, this will get you through the night.

What is it and why do we care? Electricity is conducting through the heart. It starts at the SA node, to the AV node, the bundle of His, down the left and right bundle branches, and finally to the Purkinje fibers. When the SA node depolarizes, that electrical current goes to both the atria, as well as down to the AV node. The Vector is simply the sum direction of that current (see Figure 3).

As you can see from the diagram, the small green lines represent the various depolarizations across the left and right atrium. The large solid green line is the sum of those vectors. As you know from medical school, as the vector goes towards a lead, the deflection will be positive (deflect up). As it goes away, it will be negative (deflect down). If perpendicular, it will be biphasic.

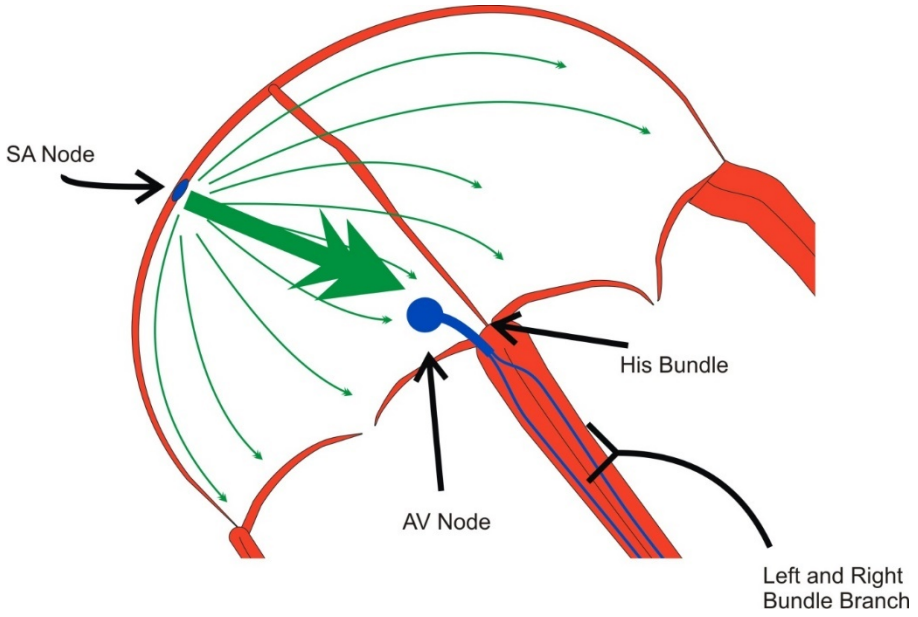


Figure 3

Next question! **How do we determine if the heart rate is *SINUS* or not (i.e. originates from the SA node)? How can we tell?** We look at the vectors and the effect they have on the ECG tracing (See Figure 4).

As the Vector of the P wave travels from the SA node to the AV node, you can see that it is travels away from aVR, and towards leads I and II. On the surface ECG, if the P wave is positive (upright) in I and II, and negative in lead aVR, then you have a sinus rhythm. Sinus rhythms originate from the SA node or near there.

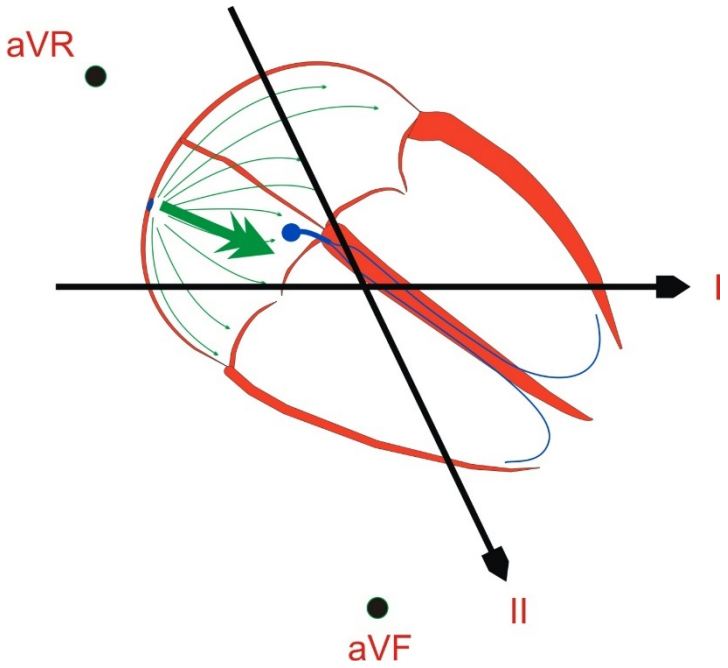


Figure 4

You will notice that the P wave vector is also going towards aVF. In sinus rhythm, the P wave is often upright in lead aVF also. However, if the P waves are NOT upright in I and II, then we know it is NOT originating from around the area of the SA node; therefore, it is NOT a sinus rhythm. The impulse still may reach the AV node, and still may conduct one to one, but it is NOT sinus.

For those of us who decided to make a career out of this confusion, by using the basic concept of knowing what normal is, then seeing the abnormal P and QRS waves, we can tell, to a certain degree, where the impulse is originating in the heart. For example, in ECG rounds, you will hear us talk about where the VT is originating…all we are doing is seeing which leads is the QRS positive (i.e. the vector of the VT is going towards those leads) and which ones are negative (i.e. the vector is going away from those leads) and making an approximate estimate of where the foci of the VT is.

Axis is similar in concept to estimating the Vector for the P wave, but it is for the whole heart (the bulk of which are the ventricles). As in the diagram, the normal axis (i.e. the sum of all the electrical vectors is generally in this direction) is -30 to + 120 degrees. That is normal. Some disease states (and some normal states) will cause the general axis of the heart to rotate to the left or to the right (i.e. left axis deviation (LAD) or right axis deviation (RAD)).

Why do we care? Because it will help us to determine what is going on with the heart, and though not a diagnosis on its own, the axis is a piece of the puzzle that helps us solve the problem. Examples of LAD are normal variants like obesity or the elderly, and abnormal states like LBBB, Wolf-Parkinson-White, and congenital lesions. Examples of RAD include normal variants, RBBB, RVH, and left ventricular ectopic beats.

How to calculate the axis? There is the simple way and the complex way. Let's stick with the simple way (see Figure 5).

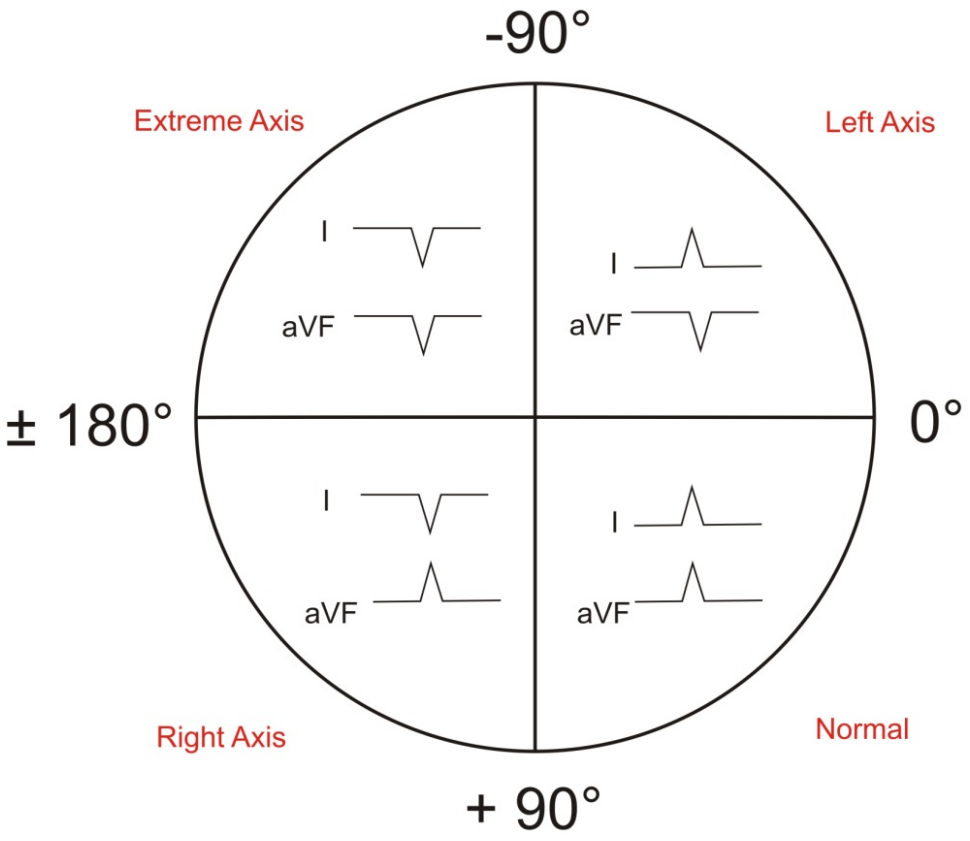


Figure 5

- If the QRS in leads I and AVF are both upright then the Axis is normal.
- If lead I is upright and lead AVF is downward the Axis is Left.
- If lead AVF is upright and lead I is downward then the Axis is Right.
- If both leads are downward then the Axis is extreme Right axis (no man's land).

Ok, because I am ultimately a Cardiologist I will also tell you the complex way, as it is more accurate (+/- 10-15 degrees) and what is generally taught. To understand this method properly you need to memorize the axis diagram (see Figure 6). If you have this memorized then it is easy.

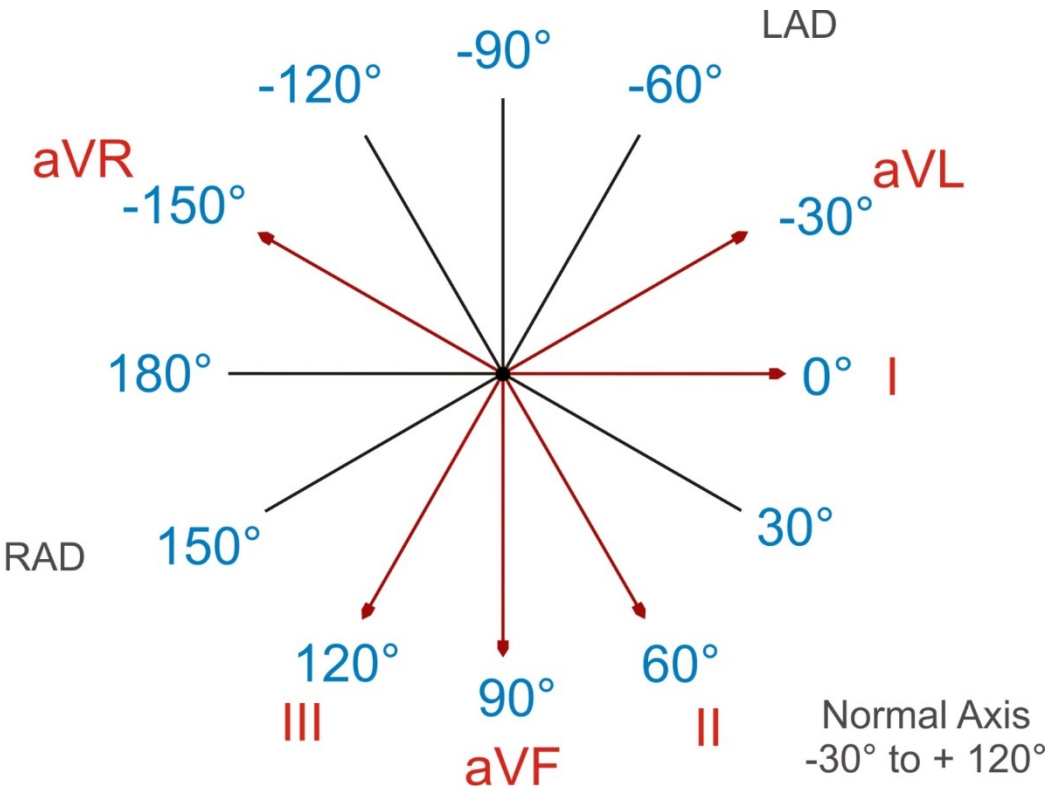


Figure 6

Say you want to know the axis of a certain ECG in front of you. What you do is you look at the limb leads on the ECG (I,II,III, aVR, aVL, aVF) and you see which one is the most equiphasic QRS complex (i.e. you count the small boxes above the baseline and subtract from the small boxes below the baseline…try for 0 or close to it). For example, let's say lead II is equiphasic.

Then, if you look at the diagram, you see which leads are perpendicular to it, and that is essentially your axis. In this case it is aVL…but is the axis -30 or + 150? Simply look at the QRS in aVL; if it is predominantly positive, then the axis is towards the positive pole (i.e. + 150), if it is predominantly negative, then it will be towards the negative pole (i.e. -30). As you can see it is a little more complex, but if you have the axis diagram memorized in your head you will be able to easily get a very good approximation to what the axis is. Clear as mud?

4B. Chamber Enlargement

The only really important one though is Left Ventricular Hypertrophy. Commonly seen in HTN, HCM, and AS. There are several criteria for diagnosing LVH, but remember that ECG is specific, not sensitive (i.e. if there is LVH on ECG, it is likely that, if there is no LVH on ECG, it doesn't necessarily mean there is no LVH). So, pick one criteria that you find easy and go with it. The Romhilt-Estes criteria is the more complex yet the best of them, but I think it is easiest to stick with the simpler ones, for example Cornell which simply states R in AVL + S in V3 > 24mm in men and 28mm in women is positive for LVH.

4C. VT or SVT with Aberrancy

You will likely come across a situation during your Cardiology rotation(s) where you will have a wide complex tachycardia ECG in your hands and you will need to make a decision on your course of action. WCT's are either VT or SVT with an aberrant pathway (or SVT with a bundle branch block); both require different treatments, and treatment for one may worsen the other. However, BOTH respond quite nicely to ZAPZAPZAP. Electrical cardioversion for any patient with WCT that is UNSTABLE is appropriate. Please keep that in mind; don't waste time fumbling with an ECG if your patient is hemodynamically unstable or losing consciousness or having chest pain. ZAPZAPZAP!

Management of VT vs SVT is something that you can review elsewhere, but how to differentiate is what we will go over here.

There are a couple of practical considerations here. First, the history is much more useful than ECG dissection in diagnosing WCT's! If there is a prior history of MI, there is a ~ 98% chance it is VT. If there is a history of structural heart disease, there is a > 90% chance it is VT[1]. Second, there are 3 things if you see that are considered "diagnostic" of VT on ECG; AV dissociation (where you see P waves independent of the wide QRS), capture beats (where in the midst of the wide QRS you see one narrow QRS) and fusion beats (QRS patterns that are essentially a "fusion" between the wide QRS of VT and the narrow QRS of the capture beat…they don't look quite like the wide QRS nor quite like the narrow one, but something in between).

If you don't see any of the "diagnostic" patterns on ECG for VT then proceed to the Brugada criteria[2] for differentiating between VT and SVT (insert groan here) (see Figure 7).

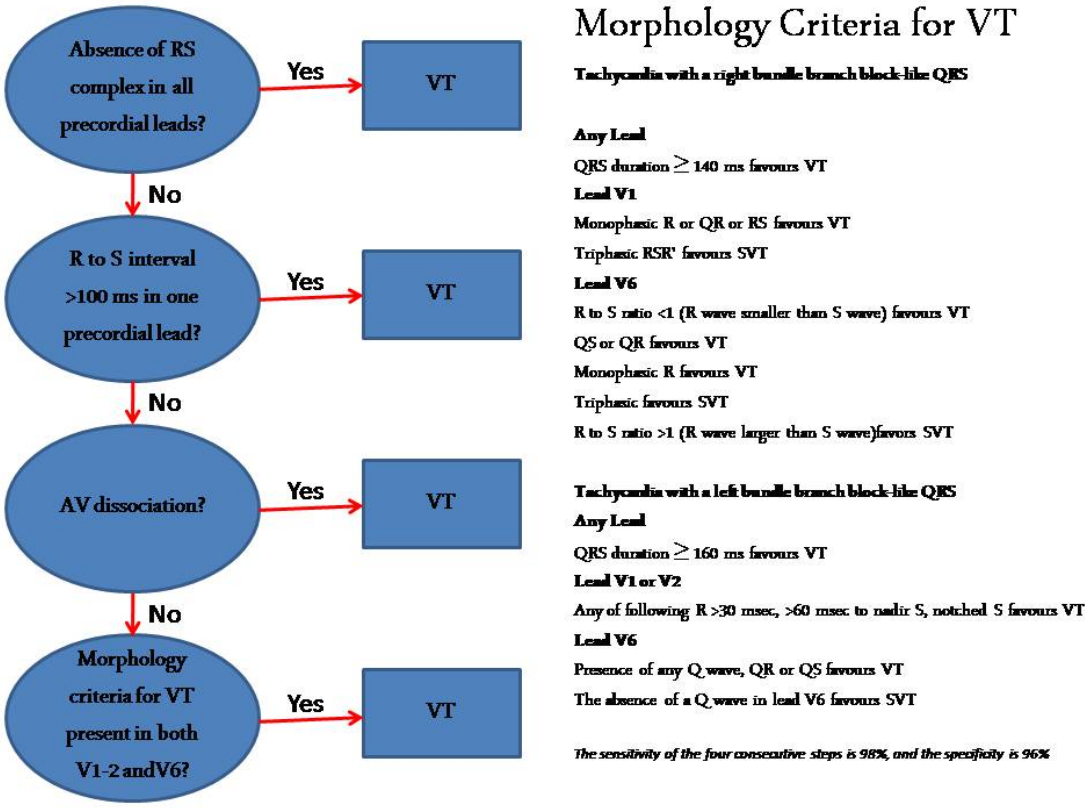


Figure 7

4D. Ischemia

Well, this is by far the commonest and also most important thing that you will be searching for when you look at the ECG in the middle of the night. Are there ischemic changes? Is that ST elevated? Will anyone be upset if I miss the ST elevation? (The answer to that last one is "yes").

Anyway, when it comes to ischemic changes the most important thing is to look at prior ECGs (as mentioned later)! That is a key point in establishing whether these changes are new or simply residual changes from prior episodes.

Now, it is best to differentiate what you are looking for into two large categories: ST elevation and then everything else. ST elevation means act now, act quickly. Anything else usually means you have time to breath. Let's go through ST elevation.

Of course, as with anything in medicine, treat the patient not the test result. If you see ST elevation in the proper setting (i.e. patient having chest pain or similar complaint) then time is muscle, and every minute counts. In this scenario, you need to have the Attending notified immediately, and you can proceed from there. The questions are:

What is true ST elevation? And what are mimickers of that?

Well the classic textbook evolution of ST elevation usually occurs in 4 stages. We will go through them but keep in mind that 1/3 or patients have electrically silent MI's, and that this is the textbook description but of course is not always the case.

See Figure 8.

- Stage 1: Is the "hyperacute" stage where the T wave peaks in size and broadens, almost "pushing" the ST segment up with it. This is a subtle change and is often short-lived.
- Stage 2: Shows the conventional ST elevation, (with the beginning of Q wave formation) often the "frown" look (i.e. the concavity is upwards). I'll stop here for a second to point out that you compare ST elevation with the PRIOR T-P segment; that is the baseline that you compare with. Also, your reference point of ST elevation is the J point, which is the sharpest angle that you see as the S becomes the ST segment.
- Stage 3: Q waves become bigger, the ST elevation becomes maximum, and the T waves begin to invert. The T waves evolve as the ST returns to baseline.
- Stage 4: The ST elevations resolves to baseline or near baseline, the T wave returns upright, and usually the Q waves are persistent. Remember pathological q waves are q waves deeper than 2 small blocks and wider than 1 small block.

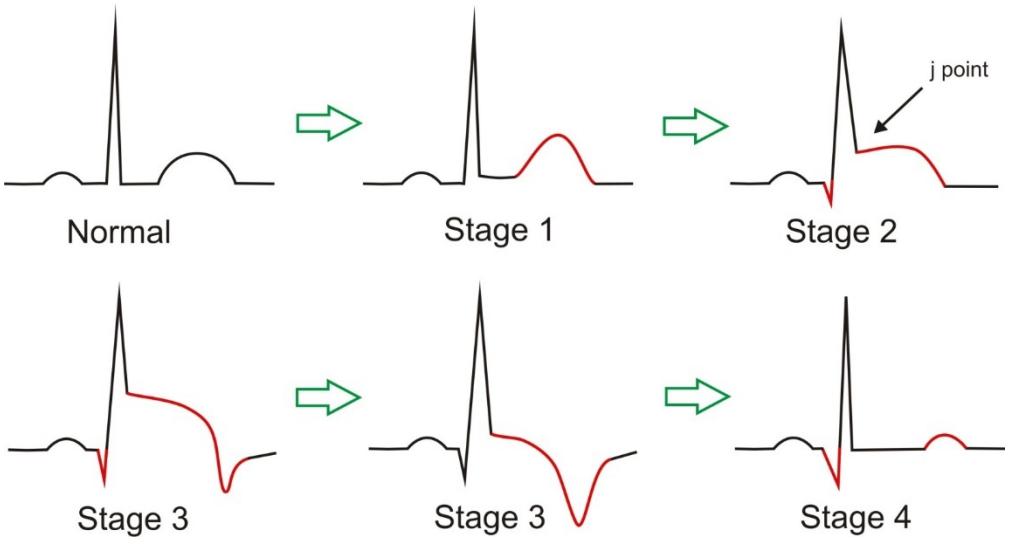
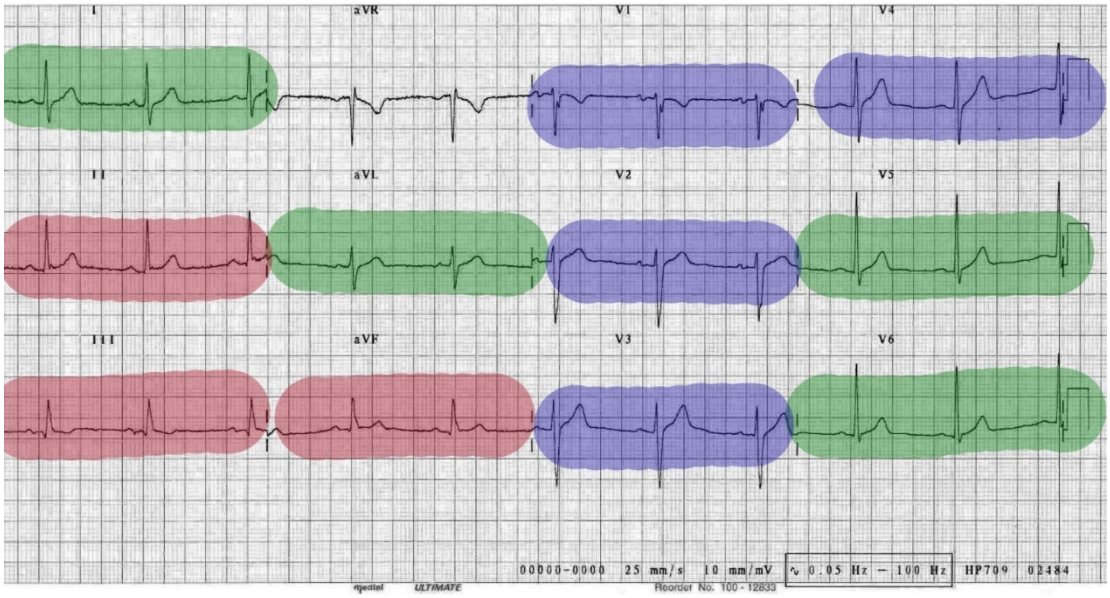


Figure 8

When you look at ischemic changes on an ECG you must look at the anatomical picture as well. What I mean by that is the various leads on the ECG correspond to one of the 3 coronary arteries (see Figure 9). If you see ST elevation in leads II and III, that fits with the RCA territory.

What if I see ST elevation in multiple territories? Well, in that case either the patient is really unlucky, or there is more to it. You have to look closer at the ECG. Is it truly ST elevation in multiple territories? Is something else going on, perhaps pericarditis? In fact, one of the key ways to differentiate pericarditis and ischemia is the distribution of the ST elevation. Remember, it must follow anatomical patterns, or else there is something that you are missing!



Red = Inferior Leads looking at the Right Coronary Artery (RCA)
Green = Lateral Leads looking at the Left Circumflex Artery (LCx)
Blue = Anterior/Antero-septal Leads looking at the Left Anterior Descending Artery (LAD)

Figure 9

Finally, you must look for reciprocal changes. What that means is as you recall from my little vector explanation is that as the current of injury goes towards one area of the heart you get ST elevation, but when it is simultaneously going away from another area of the heart so you will get ST depression in that area. An example is ST elevation in the inferior leads will give you reciprocal depression in I and aVL. Another very important example is if you see ST depression in the anterior leads (V1-4), this may in fact be an ST elevation in the posterior part of the heart! If you see that quickly ask for a 15 lead ECG that places extra leads across the patients back and looks at the posterior portion of the heart. If it is elevated you will look very smart to your Attending. If it is negative the ECG tech will give you an ugly look but that is ok.

What constitutes ST elevation MI? Well, according to the guidelines you need ST elevation of 1 mm or more in 2 or more contiguous leads. Contiguous means 2 leads beside each other in one anatomical territory...Leads I and AVR are beside each other on the ECG but not anatomically...leads II, II, and AVF are contiguous leads in the inferior (RCA) territory.

Other causes of ST elevation? Well, LBBB is the commonest you will see. Others would include LVH (and repolarization changes), an aneurysm, hyperkalemia, and hypothermia.

Everything else:

Any other ischemic ECG changes seen on an ECG is considered a NSTEMI in the setting of positive enzymes. These include ST depression, TWI, TW flattening, and ST elevation less than one small block.

Keep in mind that these changes are NOT indicative of anatomy. You might see ST depression in I, AVL, and V5-6 but that does not mean the circumflex artery is the culprit. It just means that there is ischemia going on.

One last piece of advice! Diffuse ST depression, with elevation in aVR is BAD. It usually means left main disease or left main equivalent. Call your Attending and try to prevent the patient from sneezing.

Notes

4E. Bundle Branch Block

This is easy. Simply put, a bundle branch block occurs when one of the 2 bundle branches (right or left) get blocked, usually associated with age or prior ischemia (see Figure 10)

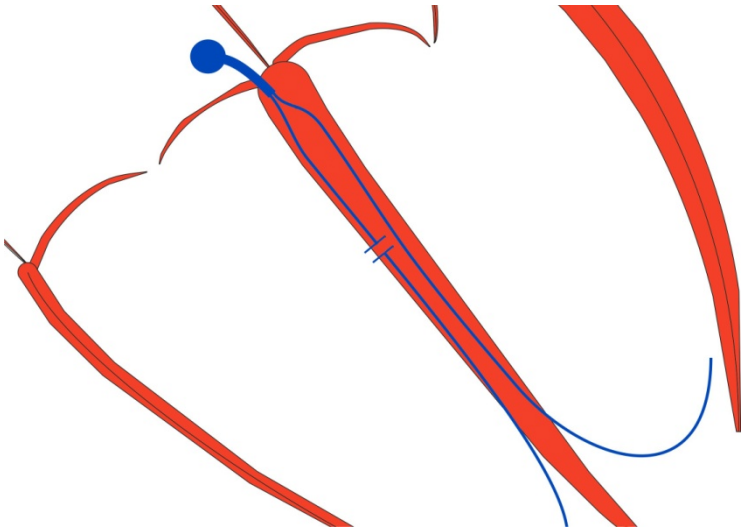


Figure 10

How can you tell on ECG? Well, look at the QRS.

- Less than 100 msec is normal.
- 100-120 msec is called interventricular conduction delay.
- 120 msec is called bundle branch block.

To differentiate between Left and Right is really easy.

- Look at V1.
- Do you see the RsR' pattern? Also called "rabbit ears"? If you do, it is a right bundle branch. Anything else is considered left bundle branch.
- And that is that.

4F. Heart Block

As you recall (or should recall, read the Axis part again if you don't) the electrical pathway of the heart starts at the SA node and ends with the Purkinje fibers. But along the way it can slow down, be interrupted, or simply stop at the AV node.

The heart block patterns that you need to know are 1st degree (slowing of the impulse through the AV node, so you get a P-R interval of more than 1 large square), 2nd degree type 1 (Wenckebach, another benign rhythm that causes a longer P-R interval with each consecutive beat until you get a P wave without a subsequent QRS, only another P wave and it resets itself), 2nd degree type 2 (a more worrisome rhythm, where you just suddenly get dropped beats), and finally 3rd degree (the worst, need admission and a pacemaker) which you will see p waves marching along completely independent of the QRS.

4G. AFib/Flutter

Very common rhythm that you will see. An easy way to recognize it is if you see QRS that are not regular but irregular (i.e. very chaotic with no recognizable pattern to them), your differential is almost always AFib. However, it can also be multifocal atrial tachycardia (in which case you have to be able to point out 3 different p wave morphologies) or atrial flutter with an irregular response.

Atrial flutter is really the same as AFib only it is often at a ventricular rate of 150 and usually you will see the classic saw tooth pattern of the baseline of the ECG. Many patients have both.

5. Approach to Common Clinical Scenarios

There are certain common clinical scenarios that you will come across in cardiology. You need to know what to do, and why you are doing it. First, you need to have a basic understanding of the common drugs we use in cardiology. The medications are often used in UA/NSTEMI and STEMI situations as well as CHF. Of course, each situation is unique, and you must carefully assess the need of each drug in each situation. ***You can cause more harm with your pen then any surgeon with his scalpel.*** Please think before you write a medication order.

5A. Common Medications

A) Beta Blockers: These drugs are an essential cornerstone in the management of ACS and CHF. They act through decreasing O2 demand to the heart, improving contractility, decreasing occurrence of VFib, increasing diastole (thereby increasing coronary blood flow) and even slow the rate of coronary atherosclerosis. These benefits have been shown in the pre-thrombolytic era (mortality benefit of 10-15%) and in the Reperfusion era (studies show up to 10% overall mortality benefit). Though there are multiple trials that have shown this benefit, a review of MIAMI and ISIS-1 trials is a good place to start. In your cardiology rotation, you do not need to memorize trials or know the details but if you are familiar with the big ones you will be a star.

B) ACE I: Most randomized, placebo-controlled trials have demonstrated that ACE inhibitor therapy within 24 hours to 16 days following an acute MI improves the left ventricular ejection fraction (LVEF) at one month to one year. The effects are most noticeable in low-EF patients (as has been shown in the study SAVE and AIRE). However, smaller mortality benefits are noted for all comers who receive ACE inhibitors within 24 hours of chest pain onset (GISSI-3, ISI-4). Finally, ACEI have been shown to decrease ventricular arrhythmia (VHeFT II) and AFib (SOLVD) in post MI patients. While there is no evidence that ACE inhibitors have direct antiarrhythmic effects, blockade of the renin-angiotensin system, reduction in ventricular and atrial wall stress, and the decrease in sympathetic tone resulting from improved left ventricular function may reduce atrial and ventricular arrhythmia.

C) ASA: The totality of evidence from basic research, clinical investigations, observational epidemiologic studies, and randomized clinical trials has provided strong support for ASA Despite this, it is still woefully underused in MI patients. It is cheap and well tolerated, it saves lives, please give it. 'Nuff said. But if you are really keen, and want the evidence, I recommend Antithrombotic Trialists' Collaboration, GUSTO-1 and 3, The Canadian Multicenter Trial, and the RISC trial.

D) Clopidogrel: The anti-platelet medication, Clopidogrel, is associated with modest reductions in the incidence of severe or refractory in-hospital ischemia, in-hospital heart failure, and need for a revascularization procedure (CURE). However, there is a significant increase in major bleeding (3.7% versus 2.7%) but not in life-threatening bleeding or hemorrhagic stroke. For patients going to the cath lab within 12 hours, the interventional Cardiologists like them to get loaded with 600 mg. Otherwise, dosing is 300 mg then 75 mg daily. Important point to keep in mind however is the bane of the cardiac surgeon's existence is this drug; unless it is an emergency they refuse to operate on patients having taken it within the last 7 days. If the patient is on the ward awaiting CABG or valvular surgery, please don't administer it.

E) Ticagrelor: This is a platelet $P2Y_{12}$ receptor blocker and works by blocking the binding of adenosine diphosphate to a specific platelet receptor $P2Y_{12}$, thereby inhibiting platelet activation (just like Clopidogrel, but has a more rapid onset and more pronounced platelet inhibition). The PLATO trial (2009) compared Ticagrelor with Clopidogrel in ACS patients included patients who underwent PCI, were referred for CABG, or were managed medically. The composite primary end point (cardiovascular death, MI, or stroke) occurred less often in patients receiving Ticagrelor (9.8 versus 11.7%). While there was no significant difference in the rates of major bleeding between the Ticagrelor and Clopidogrel groups, Ticagrelor was associated with a significantly higher rate of major bleeding not related to CABG (4.5 versus 3.8 percent). As it is a newer drug, it is quite expensive so Clopidogrel is a reasonable alternative, except in STEMI patients where Ticagrelor (or Prasugrel) is the preferred anti-platelet (in addition to aspirin).

F) Statin: There are a multitude of trails (unfortunately mostly funded by the drug-industry) that has shown the clear benefit of reducing your LDL and increasing your HDL on CV mortality/morbidity. The mechanism of action of statins is not clearly understood, but it seems that plaque stabilization, reversal of endothelial dysfunction, and decreased thrombogenicity all play a role. Whatever the reason, statin is beneficial post MI, even if patients have "normal" LDL levels (the fact that they had an MI means it needs to be lower). Though statins have been shown to be the best antilipid therapy, I recommend you familiarize yourself with other lipid-lowering drugs, because it is not uncommon to have someone who is intolerant of statins for whatever reason (check LFTs!). Some good statin trails include 4S, TNT, CARE, MIRACL, and PROVE IT-TIMI 22.

G) PCSK-9 Inhibitors: Proprotein convertase subtilisin kexin 9 (you need to be able to say this quickly 5 times in a row to pass your rotation!! – jk) is a serine protease produced predominately in the liver that leads to the degradation of LDL receptors and increased LDL levels. Therapies that lower circulating PCSK9 levels significantly lower LDL levels. As of this writing, there are currently 2 drugs approved in Canada (Evolocumab and Alirocumab, both given SQ q2weeks). This category of lipid lowering therapy appears promising in a range of clinical situations but are ridiculously expensive (thank you big pharma!), costing about $7,000 a year, compared to $400 a year for statins. Due to cost and ongoing trials these drugs have not reached mainstream yet.

F) Heparin: Thrombosis is central to coronary occlusion and infarct. Rupture of an atheromatous plaque starts a cascade that ends with thrombosis formation. Patients who have not been reperfused (i.e. neither successful thrombolytic nor PCI with stent) benefit from heparin administration. The choice of UFH vs LMWH depends on patients age and kidney function, and though trials do not show an obvious benefit of one vs another (ASSENT-3, ExTRACT-TIMI 25), two recent meta-analysis showed reduction in death and nonfatal MI with LMWH. The ACC/AHA in 2007 recommended the use of either heparin, but it was considered reasonable to prefer LMWH. The duration for LMWH was maximum hospital stay (up until 8 days) for LMWH and 48 hours for UFH.

G) Angiotensin Receptor-neprilysin inhibitor (ARNI): A novel combination drug LCZ696 (Entresto) (a fixed-dose combination of valsartan, an angiotensin receptor blocker (ARB), and sacubitril, a neprilysin inhibitor prodrug), is the most recent significant development in the HFrEF arena (see Figure 11 below).

The PARADIGM-HF trial compared the long-term effects of LCZ696 with enalapril in patients with HF (EF <40%, NYHA II-IV). The trial demonstrated the superiority of LCZ696 over enalapril in reducing the primary outcome of death from cardiovascular causes or hospitalization for HF (21.8 versus 26.5%; HR 0.80; 95% CI 0.73-0.87). The LCZ696 group had higher proportions of patients with hypotension and non-serious angioedema but lower proportions with renal impairment, hyperkalemia, and cough. Again, thanks to the profit driven (and not patient care driven) pharmaceutical industry, this drug is quite expensive; however it is currently covered by the BC government with special authority.

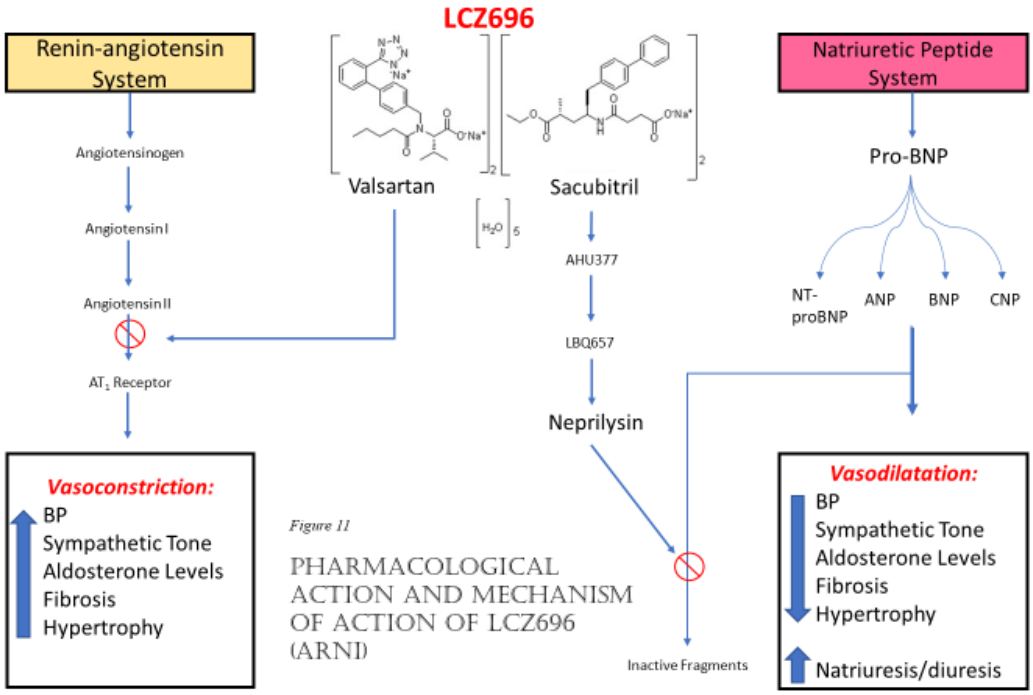


Figure 11.

H) SGTL2 Inhibitors:

Another exciting class of medications that is showing significant improving in heart failure populations are the sodium-glucose cotransporter 2 (SGLT2) inhibitors. Both DAPA-HF and EMPEROR-Reduced trials show that, *irrespective of diabetes status*, this class of medications significantly reduced hospitalizations due to heart failure and cardiovascular death. SGLT2 inhibitors promote osmotic diuresis and natriuresis in patients with and without diabetes, and thus may reduce preload, in addition to the vascular effects (including improving endothelial function) that promote vasodilation and thus may also reduce afterload.

5B. ST Elevation Myocardial Infarction (STEMI)

In 1971, Eugene Braunwald, MD, postulated a revolutionary hypothesis: ***time is muscle***. He proposed that acute MI is a dynamic process and that its clinical outcome is determined largely by infarct size. When he and his colleagues tested this hypothesis, they concluded: "Of greatest interest, from the clinical point of view, is the finding that the severity and extent of myocardial ischemic injury resulting from coronary occlusion could be radically altered not only by pretreatment but also by an appropriate intervention as late as 3 hours after the coronary occlusion".[3] This hypothesis has been proven true over and over. In STEMI patients, ***time is muscle.***

Given this, what are the times we care about?

Percutaneous coronary intervention (PCI)

The 2013 ACC/AHA Guidelines for the Management of Patients with STEMI recommended the use of primary PCI for any patient with an acute STEMI who can undergo the procedure ***within 90 minutes of first medical contact*** by persons skilled in the procedure, as long as symptom onset is less than 12 hours. If symptom onset is 12 to 24 hours, PCI is reasonable if the patient has severe HF, hemodynamic instability, or persistent ischemic symptoms. PCI > 24 hours after symptom onset is of little benefit and in fact may be harmful.

Thrombolytics

The 2013 ACC/AHA update recommend the use of fibrinolytic therapy in the following patients:

- Any patient with an STEMI who presents within 12 hours of symptom onset and has no contraindications for fibrinolysis, and presents to a facility without the capability for expert, prompt intervention with primary PCI within 90 minutes of first medical contact.
- Patients who present to a facility in which the relative delay necessary to perform primary PCI (the expected door-to-balloon time minus the expected door-to-needle time) is greater than one hour (i.e. if it will take more than an hour for PCI, give lytic).

The time interval from first patient contact to initiation of fibrinolytic drug infusion (door to needle) should be less than 30 minutes.

The survival benefit is greatest when fibrinolytic agents are administered within the first 4 hours after the onset of symptoms, particularly within the first 70 minutes (see Figure 11).

Important points about STEMI

- **Time is myocardium.**
- **Target of first medical contact to balloon is 90 minutes.**
- **Target of first medical contact to needle is 30 minutes.**
- **Risk stratify post PCI.**

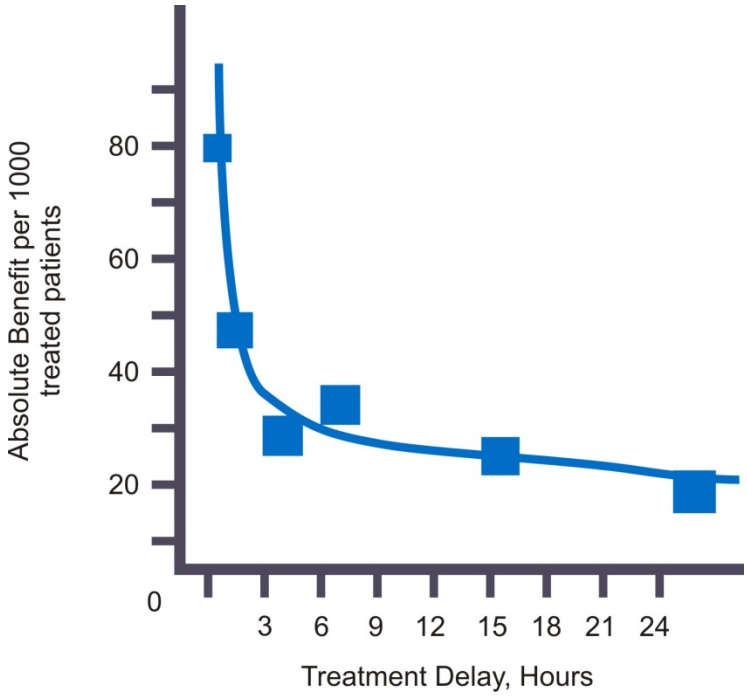


Figure 12

At ARHCC, the ER physician typically identifies the STEMI patients. These patients are sent as a primary immediately. However, keep your eyes and ears open, as you may be called upon to assess a patient with chest pain who in fact has a STEMI that is missed. If you are suspicious of this, please call your Attending right away.

More commonly, you may be asked to see a patient who has come back from PCI. Ensure they are on all the right medications. These patients should undergo early and late risk stratification soon after presentation. Early risk stratification provides the patient and family with some sense of what the future holds. Late risk stratification attempts to identify patients who are at increased risk for late arrhythmic or nonarrhythmic death. There are several validated risk prediction models that include the most important predictors of outcome. A good one is the GRACE registry, a Global Registry of Acute Coronary Syndrome (ACS) patients from almost 250 hospitals in 30 countries. Risk of both in-hospital and six-month mortality is assessed, looking at 8 independent risk factors found to account for almost 90 percent of the prognostic information:

- Age
- Killip class (look it up!)
- Systolic blood pressure
- Presence of ST segment deviation
- Cardiac arrest during presentation
- Serum creatinine concentration
- Presence of elevated serum cardiac biomarkers
- Heart rate

You can access the GRACE calculator online (www.outcomes-umassmed.org/grace) as well as download the app.

5C. Unstable Angina and non-ST Elevation Myocardial Infarction

The far majority of your consults on the ward or through emergency will be of this type of patient. The UA/NSTEMI patient population is quite varied, with their presentation and their prognosis just as varied. When you approach ACS patients in emerg there are 3 aspects of the clinical situation that we focus on.

First is the story; **is this legitimate chest pain or not? Are their cardiac risk factors? How long does it last? And is it brought on by exertion/stress and relieved by rest/NTG?** It is actually very important to listen carefully to the patient's description of his/her chest pain because that is a key element in the decision-making progress.

The second aspect we look at is the ECG. **Are there ECG changes?** As in reviewing x-rays, the best thing to do when looking at an ECG is to compare to a previous one. **Are there new changes? Are there dynamic changes with chest pain?** Finally, we look at enzymes. CK and Troponin. If it is strongly positive that usually is a slam-dunk NSTEMI, and you should further risk stratify them for management. However, there are also a multitude of other clinical phenomena that can cause elevated enzymes, so please keep that in mind.

Unstable Angina is a clinical diagnosis. There are often no ECG changes, and no enzyme rise (if there was an enzyme rise it would be a NSTEMI). The story is even more relevant in this situation, and you must look at duration of chest pain, aggravating factors and relieving factors. However, UA is managed in a similar fashion as NSTEMI.

Once you have established a diagnosis of UA or NSTEMI, what are we going to do now? Will they all get angiograms? Will they all get GXT's? The answer to that depends on your clinical assessment of the patient but like STEMI, there are also multiple scoring systems that can help you decide between the low risk patients (get GXT and if negative follow up as outpatient) or higher risk (proceed with admission and angiogram). The scoring system that I find works best is the TIMI scoring system (there is one for both UA/NSTEMI and STEMI). It comprises 7 points, and if the patient scores 3 or more, they are high risk enough to be admitted and should get an angiogram. There is an easy was to memorize the TIMI scoring system.

1. >1 mm ST depression on ECG
2. Two or more episodes of CP in the past 24 hours
3. Three or more cardiac risk factors
4. +ve enzymes
5. 50% stenosis on previous angiogram
6. Sixty-five years old or older
7. ASA use in the past 7 days

Easy? If you have not figured out WHY this is an easy way to memorize please consider another career choice (And yes, I know the 4th one is the exception to the easy memorization rule). The last thing I want to say is there are some legitimate criticisms of the TIMI scoring system, so please use it as a guide, not a hard and fast rule.

Important things about UA and NSTEMI

- **Unstable angina is a clinical diagnosis**
- **Taking a good history of cardiac pain is critical**
- **If you can count to 7, you know the TIMI risk stratification system**

Notes

5D. Atrial Fibrillation (Drs O Gusbi and S Rezazadeh)

- Atrial Fibrillation (afib) is most common sustained tachyarrhythmia
- Associated with:
    1. Increased cardiovascular morbidity and mortality
    2. Preventable Stroke
    3. 1/3 of cardiac hospitalization for cardiac rhythm disturbances
- The incidence and prevalence increase with age.

Classification

1. **Paroxysmal AFib:** (also self-terminating or intermittent):
   AFib that terminates spontaneously or with intervention within 7 days of onset.
2. **Persistent AFib:**
   AFib that fails to self-terminate within 7 days.
   Episodes often require pharmacologic or electrical cardioversion
3. **Long standing AFib:**
   AFib that has lasted for more than 12 months.
4. **Permanent AFib:**
   Used to identify patients with AFib where a joint decision by the patient and the physician to no longer pursue rhythm control strategy.
5. **Lone AFib:**
   This is used to describe patients with AFib without clinical or echocardiographic evidence of cardiopulmonary disease

Valvular Vs Non-Valvular AFib

Valvular AFib is AFib in the presence of ANY MECHANICAL HEART VALVE, or in the presence of moderate-severe mitral stenosis (rheumatic or nonrheumatic).

This is a very important notion as CHADS score does not apply to these patients and they should be on WARFARIN and not DOAC.

Clinical Presentation

1. Asymptotic
2. Palpitations, Dizziness
3. Fatigue, diaphoresis
4. Dyspnea, pulmonary edema
5. Chest pain
6. Syncope
7. Thromboembolic Stroke
8. Increased urination (elevated ANP)

Differential Diagnosis on ECG

1. Multifactorial atrial tachycardia (MAT)
2. Frequent premature atrial contractions
3. Atrial flutter with variable block

Etiology

1. Advanced age
2. Hypertension
3. Valvular heart disease
4. CHF
5. CAD
6. Cardiac surgery (highest in MV surgery)
7. Obesity/OSA
8. Hypertrophic cardiomyopathy
9. Congenital heart disease

These entities result in left atrial fibrosis, pulmonary vein dilatation and reduced atrial contractility. This leads to altered sympathetic innervation, leading to increased ectopic atrial activity and in turn leading to development of micro-reentrant circuits.

Investigations

1. 12 lead ECG
2. Echocardiography (structural heart disease)
3. TSH, GFR, LFT`s, CBC, PT/PTT, fasting lipid profile/glucose
4. ETT (in selected patients)
5. Sleep study (in selected patients)
6. 24 hrs. ambulatory BP monitoring
7. 24 hrs. Holter monitor

Treatment

A. Unstable patient:

The term unstable should include the patient who is:

1. Highly symptomatic:
    - Chest pain
    - Pulmonary edema
2. Hemodynamically unstable

***** Note:*** *Treatment of unstable patients is immediate DC Cardioversion*

B. Stable patient:

1. Control of ventricular response
2. Minimization of thromboembolic risk
3. Restoration and maintenance of sinus rhythm

1. Control of ventricular response:

Generally controlled through drugs that slow conduction through the AV node.

*****Note:*** *AFib in the setting of WPW with excitation on ECG is treated differently than AFib conducting down the AV node alone. The following medications are contraindicated:*

- *Calcium channel blockers*
- *Beta-blockers*
- *Adenosine*

(These medications facilitate conduction down the accessory pathway causing acceleration of the ventricular rate, hypotension and ventricular fibrillation). In hemodynamically stable patients, CLASS I ANTIARRHYTHMIC medications such as PROCAINAMIDE may be administered intravenously (diminishes anterograde conduction down the accessory pathway and decreases the degree of pre-excitation and may convert the AFib entirely)

(Rate Control Strategies)

A) Beta Blockers:
    a. Rapid onset of action (IV forms take 5 minutes)
    b. Used cautiously in pts with HF or low BP
    c. Amiodarone and Sotalol have Beta blocker properties
B) CCB:
    a. Diltiazem and Verapamil
    b. Rapid onset in the IV form
    c. Contradicted in low EF
C) Digitalis:
    a. IV and PO form slow onset between 1-4 hours
    b. Ideal for pts with:
        i. Low EF
        ii. Low BP
        iii. Bronchospastic disease

(Rhythm Control Strategies)

D) Other antiarrhythmics:
    a. Dofetilide (not readily available in Canada) and Ibutilide are effective for CV but not effective for control of ventricular rate
    b. Propanone and Flecainide are effective in CV in structurally normal hearts but require concomitant AV nodal blocking agents.
    c. Amiodarone should be used (IV or oral) when there is structural heart disease or it`s unknown.

2. Minimization of thromboembolic risk:

Factors associated with risk of stroke in pts with afib include:

A) Previous thromboembolism (CVA, systemic emboli)
B) Age 65 or greater
C) CAD
D) CHF
E) Female
F) HTN
G) DM
H) Renal insufficiency

***Note:** According to the 2014 focused update of the CCS guidelines for the management of AF, the CHADS2 score has changed to CHADS65 (A for Age was changed from 75 to 65 years old).*

Stroke Risk Stratification with CHADS65 (Ischemic stroke rate % per year)

0 = 0.6%

1 = 3%

2 = 4.2%

3 = 7.1%

4 = 11.1%

5 = 12.5%

6 = 13%

Patients with a CHADS 65 of one or greater require oral anticoagulation. Those with no risk factors require no anticoagulation nor anti thrombolytic therapy.

***Note:** The new (novel) oral anticoagulants are preferred over warfarin for non-valvular atrial fibrillation. These include rivaroxaban, apixaban, dabigatran, and edoxaban.*

***Note:** patients who have been in atrial fibrillation for more than 48 hours and are hemodynamically stable should not be cardioverted unless they become unstable. These patients should be anticoagulated with oral anticoagulants for 3 weeks then cardioverted. Alternatively, the patient may be placed on heparin and a TEE is performed. If no intracardiac thrombus is seen, the patient can be cardioverted.*

***Note:** Post cardioversion, cardiac output may decrease in 33% of patients for as long as one week. This may lead to pulmonary edema as soon as 3 hours post cardioversion. Atrial function also declines after cardioversion (spontaneous, pharmacological or electrical). Cardiac output returns to normal in approximately four weeks (the risk of thromboembolism is thus still increased during this period). Oral anticoagulation should continue for four weeks post cardioversion even in patients with a CHADS65 of zero.*

***Note:** Appropriate rate control during atrial fibrillation is resting heart rate of less than 100 bpm.*

3. Restoration and maintenance of sinus rhythm:

There is debate whether restoration of normal sinus rhythm is beneficial in asymptomatic patients as compared with controlling ventricular rate plus reducing of thromboembolic risk. As per the AFFIRM trial (Multicenter RCT of 4060 pts, looking at rhythm vs. rate control) rate control was found to not be inferior to rhythm control and possibly superior in elderly and co-morbid patients.

Special Considerations

A. Post-op atrial fibrillation:

- Incidence of post-op afib depends on the type of surgery. The highest incidence is post cardiac surgery (between 20 and 50%).
- Usually occurs in the first five days.
- Risk factors for perioperative atrial fibrillation include age, history or atrial fibrillation, COPD, valvular heart disease, atrial enlargement, perioperative heart failure, and withdrawal from a beta blocker or ace inhibitor.
- Usually sell limited. DC cardioversion is usually not needed.
- If afib persist for more than 48 hours anticoagulation is required
- A rhythm control or rate control strategy are both acceptable

***Note:** The new oral anticoagulants have not been studied in post op atrial fibrillation, thus warfarin is preferred.*

B. Afib in Acute MI

- Incidence of afib following an acute MI varies (10 to 20% to 30 days).
- Patients have worse outcome at 30 days than patients with normal sinus rhythm.
- Stroke rates are higher.

Treatment includes:

- Urgent electrical cardioversion,
- IV beta blocker to reduce oxygen demand,
- Digoxin,
- Post MI ACEI reduces the incidence of atrial fibrillation in patients with significant LV dysfunction.

C. Afib in COPD.

- Afib commonly develops in patients with COPD exacerbations.
- Treatment includes treating the underlying lung process, correction of hypoxia and acid base imbalances, and ventricular rate control with non-dihydropyridines (low dose betablockers can also be used that are B1 selective)
- Medications commonly used to treat bronchospastic airway disease (beta agonists, theophylline) precipitate afib.

Important things about Afib

- **Treatment of unstable patients is immediate DC Cardioversion**
- **CHADS65 replaces CHADS2**
- **Oral anticoagulation should continue for four weeks post cardioversion even in patients with a CHADS65 of zero**
- **AFFIRM!**

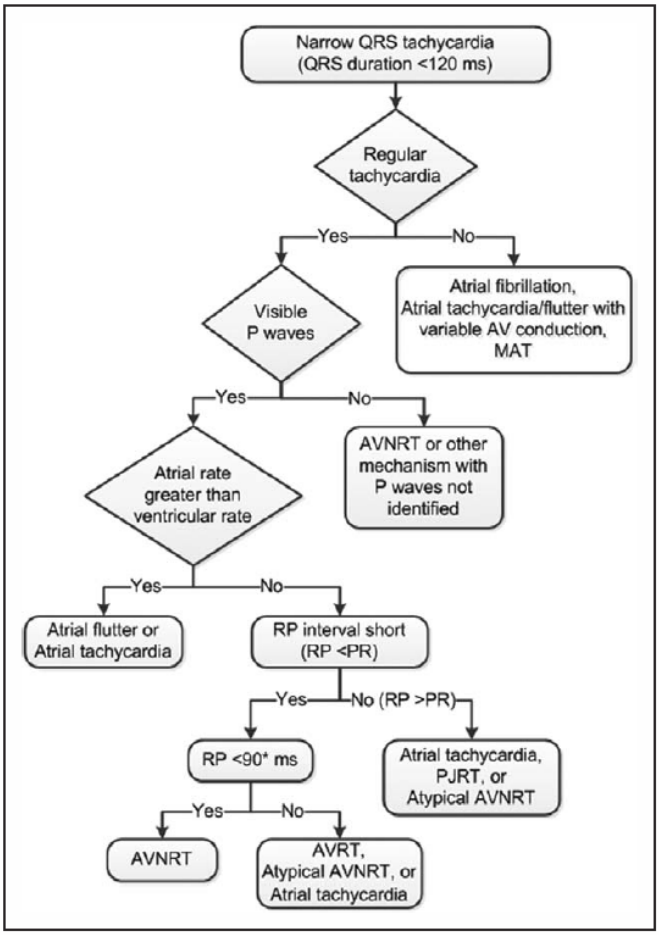
Notes

5E. Approach to SVT (Dr S Rezazadeh)

If a patient is in a narrow complex tachycardia (QRS <120 ms), then the first distinction to be made after ensuring your patient is not distressed is whether or not the rhythm is regular or not. If irregular, then the likely cause is atrial fibrillation, atrial flutter with variable AV conduction, or multifocal atrial tachycardia.

If the ventricular rhythm is regular, then try to look for P waves. If you can find them and if the atrial rate is greater than ventricular rate then the likely diagnosis is atrial flutter or atrial tachycardia. If the atrial rate is the same as the ventricular rate then look at RP (R to the next P wave duration) interval. If the RP interval is shorter than PR interval and RP interval is less than 90 ms, then AVNRT is the culprit. If RP interval is greater than 90 ms, then the diagnosis is likely AVRT or atypical AVNRT or atrial tachycardia. If the RP interval is long (RP > PR), then it is likely AVRT or atypical AVNRT.

If you can't find a clear P wave, then the likely cause is AVNRT.



Acute management:

- Vagal maneuvers should be your first line as they can often terminate the arrhythmia.
- If the patient **does not have pre-excitation** then adenosine is indicated. Make sure you have the crash cart ready and the pads on the patient.
- If the patient is hemodynamically unstable then they should be immediately cardioverted.
- If ineffective and the patient is hemodynamically stable, then IV beta-blockers, or IV diltiazem or verapamil. If ineffective then synchronized cardioversion is indicated.

Chronic management:

If preexcitation is present, then patient should be discussed with electrophysiology and plan for ablation. Avoid AV nodal agents.

If there is no evidence of pre-excitation, then beta-blockers or CCBs are the cornerstones of medical management. If they prove to be ineffective, then antiarrhythmic agents such as flecainide or propafenone can be trialed. Patients should be referred for consideration of ablation.

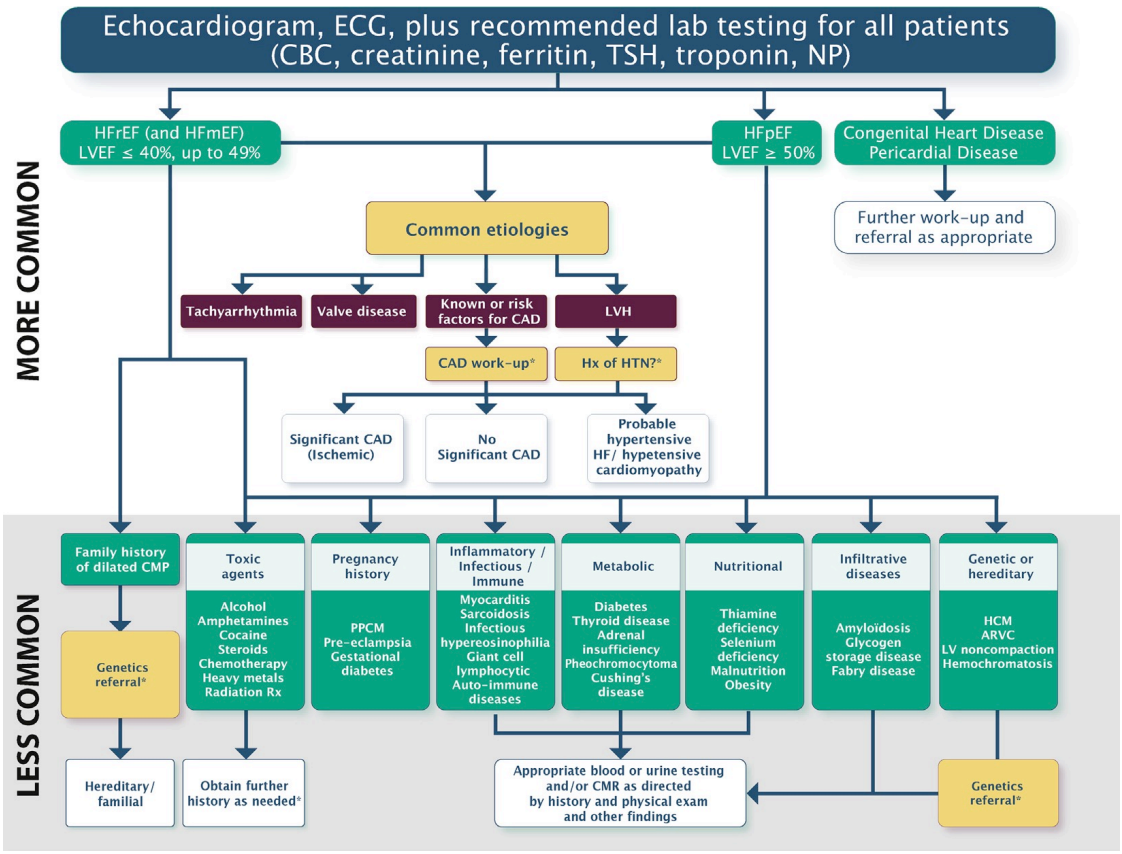
Notes

5F. Heart Failure (Dr P Bains)

Heart failure is a clinical syndrome characterized by symptoms of exercise intolerance (dyspnea, fatigue) and/or signs of fluid retention (peripheral edema and pulmonary congestion) due to a variety of pathological processes that affect normal cardiac function. Approximately 50% of patients present with Heart Failure with Preserved Ejection Fraction (HFpEF) and a left ventricular ejection fraction of >40%; the remaining half are found to have Heart Failure with Reduced Ejection Fraction (HFrEF) in which they have a clinical diagnosis of heart failure with reduced left ventricular systolic function ≤ 40%. The burden of heart failure represents a significant problem worldwide with annual mortality rates reaching ~20% annually.

Diagnosis

A thorough clinical history and physical exam should aid in the selection of additional investigations. A detailed family history is invaluable in patients who are young or who do not have an obvious history. More common etiologies (coronary artery disease, hypertension) should be considered first and testing for rarer conditions reserved for times when no cause is found. The Canadian Cardiovascular Society recommends the following approach[4]



Management

Acute Heart Failure:

In 1977 Forrester created a classification of heart failure with respects to hemodynamic severity and pulmonary congestion. This classification is predictive of cardiovascular mortality and aids in the acute treatment of heart failure[5]:

Profile	Mortality 3 months (%)	Mortality at 1 year (%)
A: No evidence of congestion or hypoperfusion	5	18
B: Congestion with adequate perfusion	13	36
C: Congestion with hypoperfusion	46	53
L: Hypoperfusion without congestion	6	19

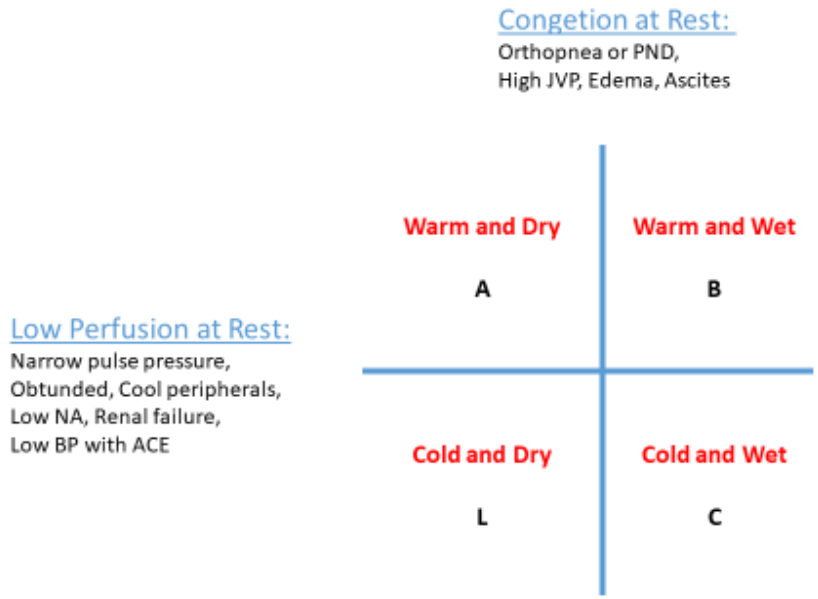


Figure 12.

The treatment algorithm for acute heart failure outlined by the Canadian Cardiovascular Society is based on the principles volume status and hemodynamic profile. In general patients who are admitted to hospital with heart failure require intravenous diuretic to relieve congestion. In patients who have new onset heart failure or who are not on chronic diuretics frusemide is administered at a dose of 20-40mg 2-3x daily if the GFR >60mL/min/1.73m2 or 20-80mg 2-3x daily if the GFR<60mL/min/1.73m2. In patients who have established heart failure or are on chronic oral diuretic therapy the initial intravenous dose is an equivalent of the oral dose.

Chronic Heart Failure:

The treatment of HFpEF and HFrEF follow similar principles but the evidence surrounding treatment strategies differs for both.

There is a complex interplay of medications that are used in managing these patients. It is very important that this point be stressed – these patients are VERY SICK, and we need to manage them carefully.

This includes non-pharmacological treatment like self-management with decreased fluid and salt intake, self-monitoring of vital sings, and daily weight assessment. These self-management strategies have been shown to reduce hospitalization at one year by 40%.

As far as medical management, please refer to Appendix IV. I would STRONGLY encourage that you have a Cardiologist or a Heart Function Clinic involved with your heart failure patients, and along those lines I again encourage you to spend some time in our heart failure clinic.

You will see that it is important to use these medications, with slow and gradual up titration, as much as possible, to allow for the best chances for improvement in EF and clinical outcomes.

In dealing with community physicians, there are 2 practical tips that I would like to pass on to you. First, we often see with some patients that physicians will decrease the medications because the patients' blood pressure or heart rate is low – DON'T! It is very important that they be on maximum tolerated therapy. Of course, if they are very symptomatic with orthostatic hypotension or presyncope/syncope, that is different. But even if changes are necessary, I encourage having a discussion with the Cardiologist before you make any drastic changes. The second tip I would give is to try to put patients on low doses of the 4 major classes of medications (beta blockers, ACE/ARB/ARNI, MRA, and SGLT2) rather than maximize one or two.

5G. Lipidology (Dr O Gusbi)

(See also Appendix V)

Editor's Note: If any of you bothered to read my Ver 1.0 dedication, you can see I am not a fan of pills. Having said that, I know that medications saves lives, and in the correct clinical situation, medications are absolutely necessary!

With that in mind, probably the most common medication class I discuss (i.e. argue) with patients about are statins. "Why do I need to be on a statin?" (Because you had a heart attack), "My cholesterol has always been fine!" (Yet you had a heart attack), "For how long do I have to be on a statin?" (Until we invent time travel and we can go back in time and prevent your heart attack), "I don't want to be on a statin" (Ummm…heart attack?) and on and on.

There are lots of myths surrounding statins, and despite claims of dementia, prostate cancer, and ALS, statins are, generally speaking, quite safe and well tolerated. Certainly, the commonest side effects are muscle aches, though this can sometimes be alleviated with co-enzyme Q10 and Magnesium supplements. However, often times you do indeed need to decrease the dose or switch to a different statin, or another class altogether (PCSK9 inhibitors).

What is also quite interesting is often I find patients OVER medicated with statins, in particular for primary prevention, where diet and exercise is usually sufficient and there is no need for statin initiation. So, please read on to get a good understanding of WHO to screen and HOW to screen, with an emphasis on secondary prevention.

Finally a couple of practical tips – of the various anti-lipid medications (niacin, fenofibrates…etc.) the best evidence really is the addition of ezetimibe to a statin. So if a patient is only tolerating a low dose statin consider the addition of ezetimibe 10 mg po qd. Second, if patients completely fail statin treatment, they may be a candidate for PCSK-9 inhibitors. A great class of injectable medications that significantly reduce LDL, but horribly expensive (because instead of $15 billion in profit pharma wants to make $16 billion), so please refer to a Cardiologist if you have hit that wall with a patient and sometimes we can facilitate to get the cost covered…sometimes!

WHO to screen for dyslipidemia in adults at risk.

1. Men ≥40 years of age
2. Women ≥40 years of age (or post-menopausal)
3. Consider earlier in ethnic groups at increased risk such as South Asian or Indigenous individuals.

All patients with any of the following conditions, regardless of age:

- clinical evidence of atherosclerosis
- abdominal aortic aneurysm (AAA)
- diabetes mellitus
- arterial hypertension
- current cigarette smoking
- stigmata of dyslipidemia (corneal arcus, xanthelasma, xanthoma)
- family history of premature CVD†
- family history of dyslipidemia
- chronic kidney disease (eGFR ≤60 mL/min/1.73 m2 or ACR ≥3 mg/mmol)
- obesity (BMI ≥30 kg/m2)
- inflammatory diseases (RA, SLE, PsA, AS, IBD)
- HIV infection
- erectile dysfunction
- COPD
- history of hypertensive disorder of pregnancy

† Men younger than 55 years of age and women younger than 65 years of age in first degree relatives. CVD = cardiovascular disease; eGFR = estimated glomerular filtration rate; ACR = albumin-to-creatinine ratio; BMI = body mass index; RA = rheumatoid arthritis; SLE = systemic lupus erythematous; PsA = psoriatic arthritis; AS = ankylosing spondylitis; IBD = inflammatory bowel disease; HIV = human immunodeficiency virus; COPD = chronic obstructive pulmonary disease

HOW to screen for dyslipidemia in adults at risk

For all:

1. History and physical examination
2. Standard lipid profile†: TC, LDL-C, HDL-C, non-HDL-C*, TG
3. FPG or A1c
4. eGFR
5. Lipoprotein(a) – once in patient's lifetime, with initial screening

Optional:

1. Apolipoprotein B (ApoB)
2. Urine ACR (if eGFR <60 mL/min/1.73 m2, hypertension, or diabetes)

TC = total cholesterol; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; TG = triglycerides; FPG = fasting plasma glucose; A1c = glycated hemoglobin; ACR = albumin-to-creatinine ratio; eGFR = estimated glomerular filtration rate.

† Non-fasting lipid testing is recommended in most adults for screening; however, for individuals with a history of triglyceride levels >4.5 mmol/L, measurement of fasting lipid levels are recommended.

* it is now generally preferable to follow non-HDL-C or ApoB levels over LDL-C when interpreting lipid results, particularly when TG is ≥1.5 mmol/L.

SECONDARY PREVENTION patients shown to derive the largest benefit from intensification of statin therapy with the addition of a PCSK9 inhibitor.

1) **Recent acute coronary event (ACS)**
   - i- Hospitalized index ACS to 52 weeks post index ACS

2) **Clinically evident ASCVD and any of the following:**
   - i- Diabetes mellitus or metabolic syndrome
   - ii- Polyvascular disease (vascular disease in ≥2 arterial beds)
   - iii- Symptomatic PAD
   - iv- Recurrent MI
   - v- MI in the past 2 years
   - vi- Previous CABG surgery
   - vii- LDL-C ≥ 2.6 mmol/L or heterozygous FH
   - viii- Lipoprotein (a) ≥ 60 mg/dL (120 nmol/L)

ASCVD = atherosclerotic cardiovascular disease; PAD = peripheral arterial disease; MI = myocardial infarction; CABG = coronary artery bypass graft; LDL-C = low density lipoprotein cholesterol; FH = familial hypercholesterolemia

6. Common Cardiology Investigations

6A. Exercise Stress Test (GXT or ETT):

A GXT indirectly detects myocardial ischemia. It is a common tool in our investigatory repertoire and you will often use it to risk stratify your patients, mainly in dealing with emerg/outpatient type of patients.

What is it?

Patients scheduled for a GXT are essentially hooked up to a 12-lead ECG for continuous monitoring while they attempt to reach their target heart rate (220 - the age of the patient = target heart rate) on a treadmill. We have the patient walk on the treadmill until that target heart rate, and analyze the patient's symptoms, blood pressure, and the ECG tracing.

They exercise on the treadmill using various "protocols"; the commonest and most validated is the Bruce protocol that is divided into successive three-minute stages, each of which requires the patient to walk faster and at a steeper grade. The modified Bruce protocol is used for risk stratification of patients after an ACS and in sedentary patients in whom the standard Bruce protocol may be too strenuous.

There is also the Cornell and Naughton protocols, details of which you don't need to know as they are not commonly used at ARHCC.

When do we order it?

Indications: According to the 2002 ACC/AHA guidelines, it is a class I indication (see Table 1) to perform a GXT to diagnose coronary heart disease in patients who have an intermediate (variably defined as between 25% and 75% or between 10% and 90%) pretest probability.

Table 2 is based on the classic Diamond-Forrester study that assessed pretest probability based on age, gender, and symptoms, with subsequent angiography. It is a great tool to help you establish the pretest probability of coronary heart disease in the patients you assess in emerg/outpatient setting.

ACC/AHA guideline summary: Exercise ECG testing without an imaging modality for the diagnosis of obstructive coronary heart disease	
Class I	An intermediate pretest probability of CHD based upon age, gender, and symptoms, including patients with complete right bundle branch block or less than 1 mm ST depression, in the absence of the exceptions listed in class IIb and class III.
Class IIa	Suspected variant (vasospastic) angina.
Class IIb	A high or low pretest probability of CHD. Digoxin therapy and less then 1 mm of ST segment depression at baseline. Electrocardiographic evidence of left ventricular hypertrophy and less then 1 mm of ST segment depression at baseline.
Class III	Patients with the following baseline ECG abnormalities: 1. Preexcitation (Wolff-Parkinson-White) syndrome. 2. Electronically paced ventricular rhythm. 3. More than 1 mm of ST segment depression at rest. 4. Complete left bundle branch block. An established diagnosis of CHD due to prior myocardial infarction or coronary angiography. However, testing may be warranted in such patients to assess functional capacity and prognosis.
Table 1. Class I: There is evidence and/or general agreement at the usefulness, Class IIa: The weight of evidence or opinion is in favor, Class IIb: The usefulness is less well established, Class III: There is evidence and/or general agreement that it is not useful.	
Circulation 2003; 107:149	

Pretest probability of coronary heart disease in patients with chest pain according to age, gender, and symptoms						
Age	**Nonanginal pain**		**Atypical angina**		**Typical angina**	
	Men	**Women**	**Men**	**Women**	**Men**	**Women**
30-39	4	2	34	12	76	26
40-49	13	3	51	22	87	55
50-59	20	7	65	31	93	73
60-69	27	14	72	51	94	86
Table 2. *N Engl J Med 1979; 300:1350; and N Engl J Med 1979; 301:230*						

6B. Echocardiography:

So, you know, I spent 2 years after my Cardiology Fellowship to train in the nuances and beauty of Echocardiography. As such, if you are rounding with me, no matter the clinical question, know that if your answer is "patient should get an echo!", you will pass the rotation!!

Kidding aside, Echocardiography is a diagnostic procedure that uses ultrasound waves to create images of the heart and the blood vessels that supply it. When you order an echo for a patient, you should have a specific question in mind, and be looking for a specific answer. Make sure before you have completed your rotation that you have reviewed a few echocardiograms with your Attending. It's quite interesting to witness the cardiac hemodynamics and cardiac structures real time.

You will find that inpatient echoes are done fairly quickly; if not same day then next day at the latest. Please do not abuse that, as we have limited resources, so there are appropriate and inappropriate indications for echo (Appendix III) but nothing replaces clinical judgment and your assessment.

6C. MIBI:

This nuclear scan is done at ARH, and is a noninvasive tool that enables evaluation of cardiac perfusion and function at rest and during dynamic exercise or pharmacologic stress for the diagnosis and management of patients with known or suspected coronary heart disease.

It has increased sensitivity and specificity (89 and 77%, respectively) compared to regular stress testing (68 and 77%), and allows for risk stratification pharmacologically for those unable to exercise or with underlying ECG abnormalities that prevent accurate assessment of ischemia (like LBBB).

However, it does involve radiation, and there is an environmental cost with the nuclear material used. It is a great tool to use but make sure when ordering it that it is truly indicated.

6D. Stress Echocardiography:

Similar to MIBI scans, stress echocardiography is used to detect hemodynamically significant CAD. The basic concept of stress echocardiography is the detection of ischemia through the development of new regional wall motion abnormalities or worsening of preexisting regional wall motion abnormalities.

Stress echo has the added advantage of adding insight regarding hemodynamics during stress, allowing for assessment of valvular disease, diastolic dysfunction, and pulmonary hypertension at peak stress.

6E. Cardiac Catheterization

Coronary catheterization is the direct visualization of the coronary arteries under fluoroscopy using dye. This is often followed by placing a stent in any significant narrowing in the arteries. In the cath lab we can also measure pressures in the LV (and right sided chambers including wedge pressures when undergoing a "right heart cath") in addition to assessing aortic and mitral valve disease (though echo is the preferred method of doing so).

We do not have a cath lab at ARHCC, but will send patients to the Royal Columbian Hospital, or occasionally to St. Paul's Hospital or VGH. Though it is an easy answer to cath all patients who present with chest (since cath is the "gold standard") it is not without its complications (it is, after all, an invasive procedure). Having said that, as per guidelines, most NSTEMI and STEMI patients will undergo a cath at some point.

Before sending a patient to the cath lab, ensure appropriateness for the cath, look at the patient's blood work (like creatinine, as they will be receiving a dye load), any anticoagulation medications (barring emergencies, INR <1.7 is preferred), and examine to ensure good femoral and/or radial pulses.

Do you know what the Allan test is?

Notes:

Appendix I (Rounds/Clinics)

Schedule of Rounds/Clinics

Cardiology Educational Rounds:

When: Every 1st and 3rd Wednesday of the month, from 1200-1300

Where: CCU conference room located off of the CCU

Chest Pain Clinic:

When: Every Monday through Friday from 1230-1600

Where: 3rd Floor Diagnostic Services (Fraser Wing)

Cardiac Rehab Clinic:

When: Every Tuesday through Thursday from 1230-1600

Where: 3rd Floor Diagnostic Services (Fraser Wing)

Heart Failure Clinic:

When: Every Wed from 0900-1600

Where: 2nd Floor Healthy Living with Chronic Conditions Clinic (Sumas Wing)

ECG Rounds:

When: TBD; once a month from 0800-0830, Thursdays before academic half day

Where: CCU conference room

Echocardiography Rounds:

When: Every 4th Wednesday of the month, from 1200-1300

Where: CCU conference room

Appendix II (Topics to Review)

Topics That You Need To Know

Hatoum T, Sheldon R. A Practical Approach to Investigation of Syncope. Canadian Journal of Cardiology. 2014; Jun;30(6):671-674.

King M, Kingery J, Casey B. Diagnosis and Evaluation of Heart Failure. *American Family Physician*. 2012; 85(12):1161-1168.

Kumar A, Cannon CP. Acute Coronary Syndromes: Diagnosis and Management, Part I. *Mayo Clinic Proceedings*. 2009;84(10):917-938.

Kumar A, Cannon CP. Acute Coronary Syndromes: Diagnosis and Management, Part II. *Mayo Clinic Proceedings*. 2009;84(11):1021-1036.

Macle L, Cairns JA, Andrade JG, Mitchell LB, Nattel S, Verma A. 2014 Atrial Fibrillation Guidelines. *Canadian Journal of Cardiology.* 2015;Oct;31(10):1207-18.

Yaxley JP, Thambar SV. Resistant hypertension: an approach to management in primary care. *Journal of Family Medicine and Primary Care*. 2015;4(2):193-199. doi:10.4103/2249-4863.154630.

Appendix III (Echo Appropriateness Criteria)

Appropriateness for Echocardiography

These are the Appropriateness Criteria for Transthoracic and Transesophageal Echocardiography as set by the ACCF/ASE/ACEP/ASNC/SCAI/SCCT/SCMR in 2007. Scores of 1-3 are deemed generally inappropriate (I) and 7-9 are deemed appropriate (A).

Table 8. Appropriate Indications (Median Score 7–9)

	Indication	Appropriateness Score (1–9)
	General Evaluation of Structure and Function—Suspected Cardiac Etiology—General	
1.	Symptoms potentially due to suspected cardiac etiology, including but not limited to dyspnea, shortness of breath, lightheadedness, syncope, TIA, cerebrovascular events	A (9)
2.	Prior testing that is concerning for heart disease (i.e., chest X-ray, baseline scout images for stress echocardiogram, ECG, elevation of serum BNP)	A (8)
	General Evaluation of Structure and Function—Adult Congenital Heart Disease	
3.	Assessment of known or suspected adult congenital heart disease including anomalies of great vessels and cardiac chambers and valves, or suspected intracardiac shunt (ASD, VSD, PDA) either in unoperated patient or following repair/operation	A (9)
	General Evaluation of Structure and Function—Arrhythmias	
6.	Patients who have sustained or nonsustained SVT or VT	A (8)
	General Evaluation of Structure and Function—LV Function Evaluation	
8.	Initial evaluation of LV function following acute MI	A (9)
9.	Re-evaluation of LV function following MI during recovery phase when results will guide therapy	A (8)
	General Evaluation of Structure and Function—Pulmonary Hypertension	
10.	Evaluation of known or suspected pulmonary hypertension including evaluation of right ventricular function and estimated pulmonary artery pressure	A (8)
	Cardiovascular Evaluation in an Acute Setting—Hypotension or Hemodynamic Instability	
11.	Evaluation of hypotension or hemodynamic instability of uncertain or suspected cardiac etiology	A (9)
	Cardiovascular Evaluation in an Acute Setting—Myocardial Ischemia/Infarction	
12.	Evaluation of acute chest pain with suspected myocardial ischemia in patients with nondiagnostic laboratory markers and ECG and in whom a resting echocardiogram can be performed during pain	A (8)
13.	Evaluation of suspected complication of myocardial ischemia/infarction, including but not limited to acute mitral regurgitation, hypoxemia, abnormal chest X-ray, VSD, free-wall rupture/tamponade, shock, right ventricular involvement, heart failure, or thrombus	A (9)
	Cardiovascular Evaluation in an Acute Setting—Respiratory Failure	
14.	Evaluation of respiratory failure with suspected cardiac etiology	A (8)
	Cardiovascular Evaluation in an Acute Setting—Pulmonary Embolism	
16.	Evaluation of patient with known or suspected acute pulmonary embolism to guide therapy (i.e., thrombectomy and thrombolytics)	A (8)

	Indication	Appropriateness Score (1–9)
	Evaluation of Valvular Function—Murmur	
17.	Initial evaluation of murmur in patients for whom there is a reasonable suspicion of valvular or structural heart disease	A (9)
	Evaluation of Valvular Function—Mitral Valve Prolapse	
18.	Initial evaluation of patient with suspected mitral valve prolapse	A (9)
	Evaluation of Valvular Function—Native Valvular Stenosis	
20.	Initial evaluation of known or suspected native valvular stenosis	A (9)
22.	Routine (yearly) evaluation of an asymptomatic patient with severe native valvular stenosis	A (7)
23.	Re-evaluation of a patient with native valvular stenosis who has had a change in clinical status	A (9)
	Evaluation of Valvular Function—Native Valvular Regurgitation	
24.	Initial evaluation of known or suspected native valvular regurgitation	A (9)
26.	Routine (yearly) re-evaluation of an asymptomatic patient with severe native valvular regurgitation with no change in clinical status	A (8)
27.	Re-evaluation of native valvular regurgitation in patients with a change in clinical status	A (9)
	Evaluation of Valvular Function—Prosthetic Valve	
28.	Initial evaluation of prosthetic valve for establishment of baseline after placement	A (9)
30.	Re-evaluation of patients with prosthetic valve with suspected dysfunction or thrombosis *or* a change in clinical status	A (9)
	Evaluation of Valvular Function—Infective Endocarditis (Native or Prosthetic Valves)	
31.	Initial evaluation of suspected infective endocarditis (native and/or prosthetic valve) with positive blood cultures or a new murmur	A (9)
33.	Re-evaluation of infective endocarditis in patients with any of the following: virulent organism, severe hemodynamic lesion, aortic involvement, persistent bacteremia, a change in clinical status, or symptomatic deterioration	A (9)
	Evaluation of Intracardiac and Extracardiac Structures and Chambers	
34.	Evaluation for cardiovascular source of embolic event (PFO/ASD, thrombus, neoplasm)	A (8)
35.	Evaluation of cardiac mass (suspected tumor or thrombus)	A (9)
36.	Evaluation of pericardial conditions including but not limited to pericardial mass, effusion, constrictive pericarditis, effusive-constrictive conditions, patients post-cardiac surgery, or suspected pericardial tamponade	A (9)
	Evaluation of Aortic Disease	
37.	Known or suspected Marfan disease for evaluation of proximal aortic root and/or mitral valve	A (9)
	Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Hypertension	
38.	Initial evaluation of suspected hypertensive heart disease	A (8)
	Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Heart Failure	
41.	Initial evaluation of known or suspected heart failure (systolic or diastolic)	A (9)
43.	Re-evaluation of known heart failure (systolic or diastolic) to guide therapy in a patient with a change in clinical status	A (9)
	Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Pacing Device Evaluation	
44.	Evaluation for dyssynchrony in a patient being considered for CRT	A (8)
45.	Patient with known implanted pacing device with symptoms possibly due to suboptimal pacing device settings to re-evaluate for dyssynchrony and/or revision of pacing device settings	A (8)
	Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Hypertrophic Cardiomyopathy	
46.	Initial evaluation of known or suspected hypertrophic cardiomyopathy	A (9)
48.	Re-evaluation of known hypertrophic cardiomyopathy in a patient with a change in clinical status to guide or evaluate therapy	A (9)
	Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Cardiomyopathy (Other)	
49.	Evaluation of suspected restrictive, infiltrative, or genetic cardiomyopathy	A (9)
50.	Screening study for structure and function in first-degree relatives of patients with inherited cardiomyopathy	A (8)
	Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Therapy With Cardiotoxic Agents	
51.	Baseline and serial re-evaluations in patients undergoing therapy with cardiotoxic agents	A (8)
	Use of TEE as the Initial Test—Common Uses	
52.	Evaluation of suspected acute aortic pathology including dissection/transsection	A (9)
53.	Guidance during percutaneous noncoronary cardiac interventions including but not limited to septal ablation in patients with hypertrophic cardiomyopathy, mitral valvuloplasty, PFO/ASD closure, radiofrequency ablation	A (9)
54.	To determine mechanism of regurgitation and determine suitability of valve repair	A (9)
55.	To diagnose/manage endocarditis with a moderate or high pre-test probability (e.g., bacteremia, especially staph bacteremia or fungemia)	A (9)
56.	Persistent fever in patient with intracardiac device	A (9)
	Use of TEE as the Initial Test—Common Uses—Atrial Fibrillation/Flutter	
57.	Evaluation of patient with atrial fibrillation/flutter to facilitate clinical decision-making with regards to anticoagulation and/or cardioversion and/or radiofrequency ablation	A (9)

Table 10. Inappropriate Indications (Median Score 1–3)

Indication		Appropriateness Score (1–9)
General Evaluation of Structure and Function—Adult Congenital Heart Disease		
4.	Routine (yearly) evaluation of asymptomatic patients with corrected ASD, VSD, or PDA more than 1 year after successful correction	I (3)
General Evaluation of Structure and Function—Arrhythmias		
5.	Patients who have isolated APC or PVC without other evidence of heart disease	I (2)
General Evaluation of Structure and Function—LV Function Evaluation		
7.	Evaluation of LV function with prior ventricular function evaluation within the past year with normal function (such as prior echocardiogram, LV gram, SPECT, cardiac MRI) in patients in whom there has been no change in clinical status	I (2)
Cardiovascular Evaluation in an Acute Setting—Pulmonary Embolism		
15.	Initial evaluation of patient with suspected pulmonary embolism in order to establish diagnosis	I (3)
Evaluation of Valvular Function—Mitral Valve Prolapse		
19.	Routine (yearly) re-evaluation of mitral valve prolapse in patients with no or mild MR and no change in clinical status	I (2)
Evaluation of Valvular Function—Native Valvular Stenosis		
21.	Routine (yearly) re-evaluation of an asymptomatic patient with mild native AS or mild-moderate native MS and no change in clinical status	I (2)
Evaluation of Valvular Function—Native Valvular Regurgitation		
25.	Routine (yearly) re-evaluation of native valvular regurgitation in an asymptomatic patient with mild regurgitation, no change in clinical status, and normal LV size	I (2)
Evaluation of Valvular Function—Prosthetic Valve		
29.	Routine (yearly) evaluation of a patient with a prosthetic valve in whom there is no suspicion of valvular dysfunction and no change in clinical status	I (3)
Evaluation of Valvular Function—Infective Endocarditis (Native or Prosthetic Valves)		
32.	Evaluation of native and/or prosthetic valves in patients with transient fever but without evidence of bacteremia or new murmur	I (2)
Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Hypertension		
39.	Routine evaluation of patients with systemic hypertension without suspected hypertensive heart disease	I (3)
40.	Re-evaluation of a patient with known hypertensive heart disease without a change in clinical status	I (3)
Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Heart Failure		
42.	Routine (yearly) re-evaluation of patients with heart failure (systolic or diastolic) in whom there is no change in clinical status	I (3)
Evaluation of Hypertension, Heart Failure, or Cardiomyopathy—Hypertrophic Cardiomyopathy		
47.	Routine (yearly) evaluation of hypertrophic cardiomyopathy in a patient with no change in clinical status	I (3)
Use of TEE as the Initial Test—Common Uses—Atrial Fibrillation/Flutter		
58.	Evaluation of a patient with atrial fibrillation/flutter for left atrial thrombus or spontaneous contrast when a decision has been made to anticoagulate and not to perform cardioversion	I (3)

Appendix IV (HF Management)

Management of Chronic Heart Failure

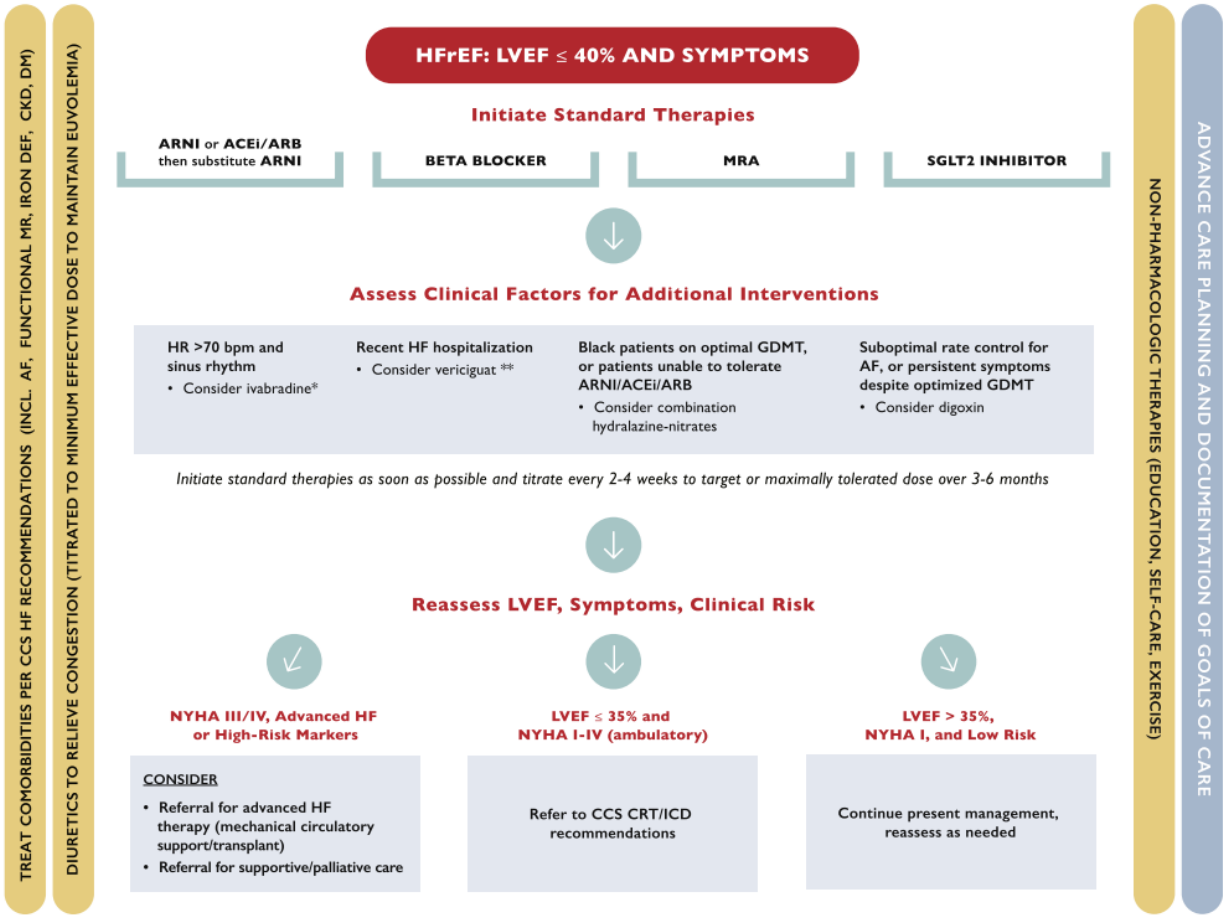
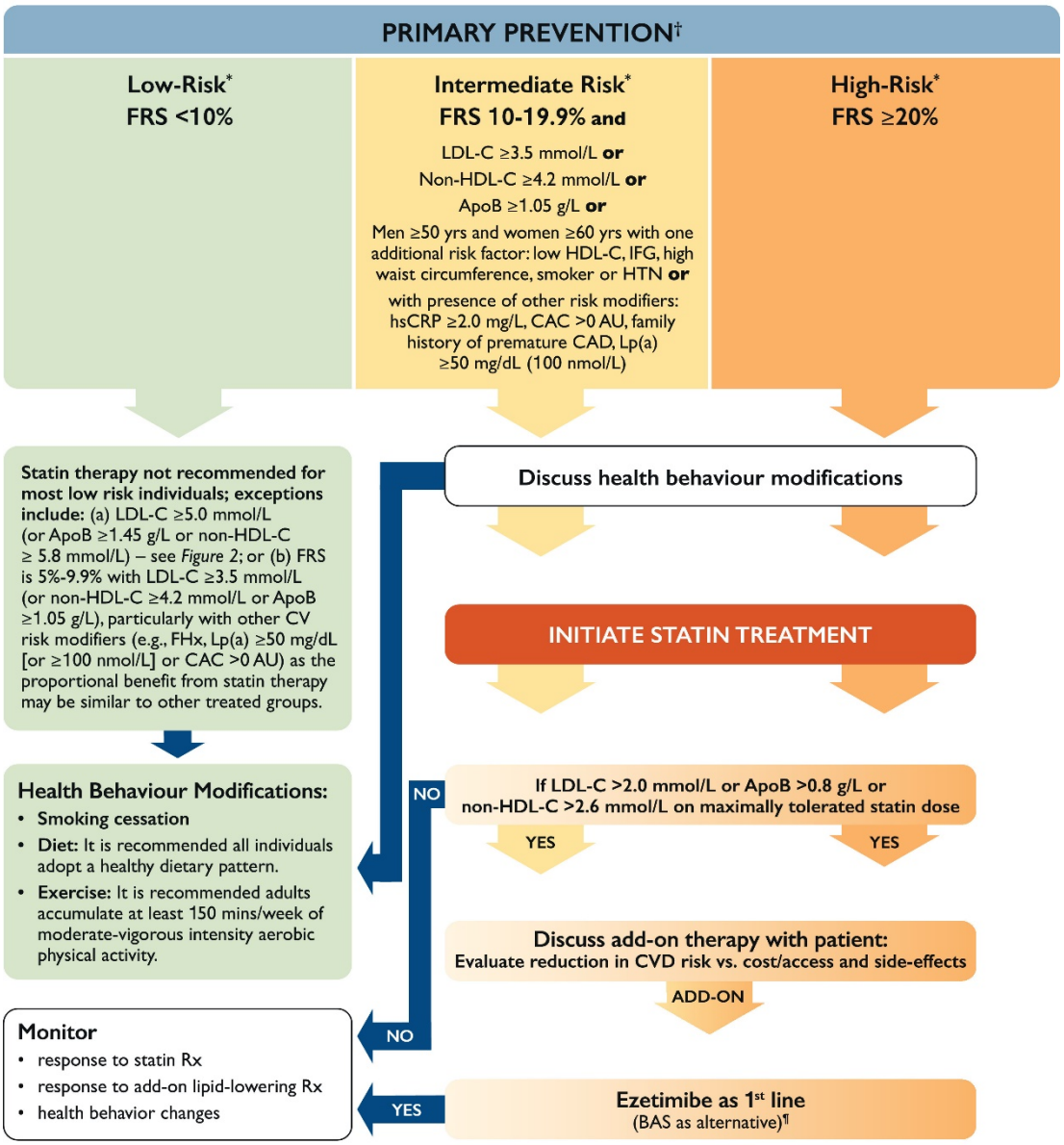


Figure 1. Simplified treatment algorithm for management of heart failure (HF) with reduced ejection fraction (HFrEF). Standard therapies are applicable to most patients with HFrEF for reducing cardiovascular mortality and hospitalization for HF. Additional, pharmacologic therapies should be individualized on the basis of clinical factors as outlined in the text. Every attempt should be made to initiate and titrate therapies with the goal of medication optimization by 3-6 months after a diagnosis of HFrEF. Throughout the patient journey, nonpharmacologic therapies should be prescribed, along with judicious use of diuretics to maintain euvolemia. Evidence also supports interventions to treat important comorbidities including iron deficiency, atrial fibrillation (AF), and functional mitral regurgitation (MR) in selected patients. ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; CCS, Canadian Cardiovascular Society; CKD, chronic kidney disease; CRT, cardiac resynchronization therapy; DM, diabetes mellitus; GDMT, guideline-directed medical therapy; ICD, implantable cardioverter defibrillator; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; NYHA, New York Heart Association; SGLT, sodium glucose transport. * Health Canada has approved ivabradine for patients with HFrEF and heart rate (HR) $\geq$ 77 bpm in sinus rhythm. ** Vericiguat is not yet approved for use in Canada.

Appendix V (Lipid Management)

Treatment Approach for Primary Prevention Patients (without a statin indicated condition‡)



‡Statin indicated conditions consists of all documented ASCVD conditions, as well as other high-risk primary prevention conditions in the absence of ACSVD, such as most patients with diabetes, those with chronic kidney disease and those with a LDL-C ≥5.0 mmol/L.

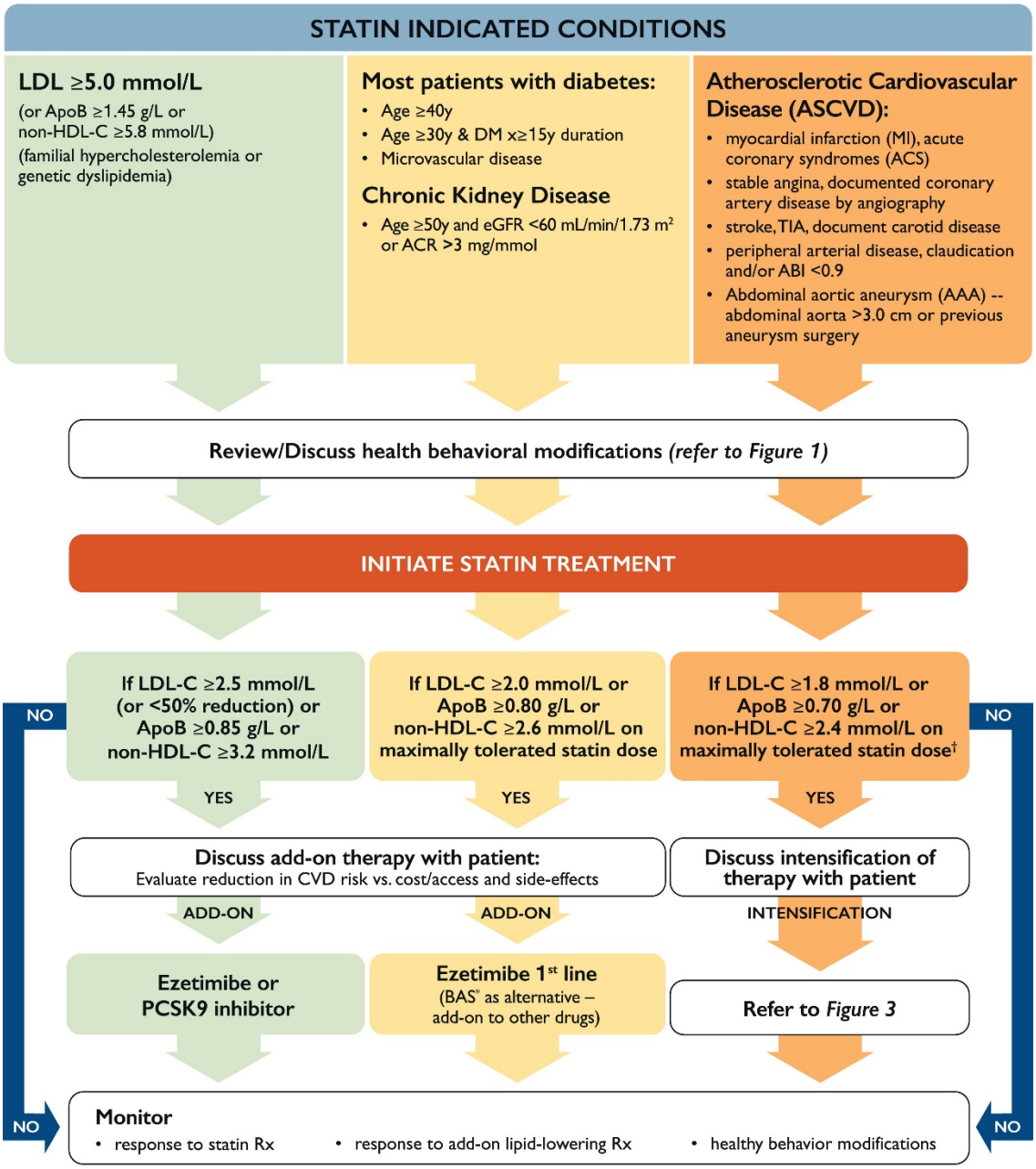
†Calculate risk using the Framingham Risk Score (FRS) – refer to the iCCS available on the App Store or on Google Play

*Screening should be repeated every 5 years for men and women aged 40 to 75 years using the modified FRS or CLEM to guide therapy to reduce major CV events. A risk assessment might also be completed whenever a patient's expected risk status changes.

¶ studies have evaluated the efficacy of BAS for the prevention of ASCVD, but results have been inconclusive.

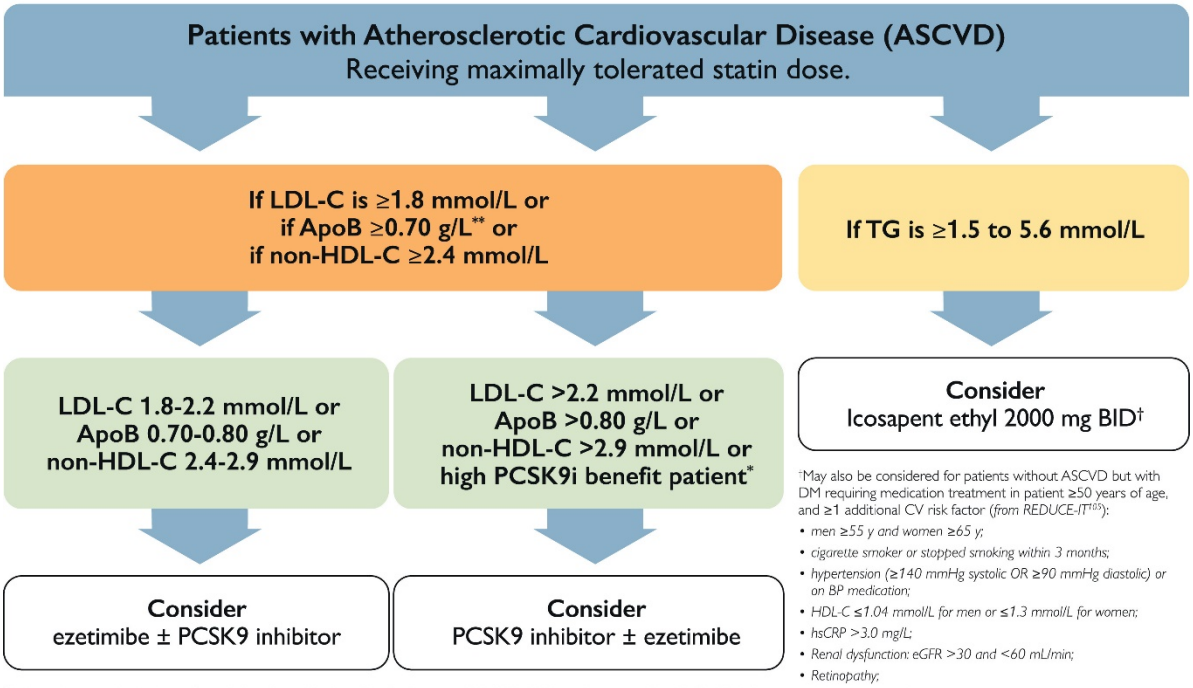
FRS = Framingham risk score; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; ApoB = apolipoprotein B; IFG = impaired fasting glucose; HTN = hypertension; hsCRP = high-sensitivity C-reactive protein; CAC = coronary artery calcium; AU – Agatston unit; Rx = prescription; BAS = bile acid sequestrant

Treatment Approach for Patients with a Statin Indicated Condition



eGFR = estimated glomerular filtration rate; ACR = albumin-to-creatinine; TIA = transient ischemic attack; ABI = ankle-brachial index.
†LDL-C threshold selected on the basis of the PCSK9-inhibitor clinical trials lipid inclusion parameters (references 91 and 92) with percentile equivalents used for ApoB and non-HDL-C (see supplement).
*studies have evaluated the efficacy of BAS for the prevention of ASCVD, but results have been inconclusive.

Treatment Intensification Approach for Patients with Atherosclerotic Cardiovascular Disease (ASCVD)



*Patients shown to derive largest benefit form intensification of statin therapy with PCSK9 inhibitor therapy are identified in Table 3.

**At low levels of LDL-C or non-HDL-C, measurement of apoB is more accurate than other markers.

†May also be considered for patients without ASCVD but with DM requiring medication treatment in patient ≥50 years of age, and ≥1 additional CV risk factor (*from REDUCE-IT*[105]):

- *men ≥55 y and women ≥65 y;*
- *cigarette smoker or stopped smoking within 3 months;*
- *hypertension (≥140 mmHg systolic OR ≥90 mmHg diastolic) or on BP medication;*
- *HDL-C ≤1.04 mmol/L for men or ≤1.3 mmol/L for women;*
- *hsCRP >3.0 mg/L;*
- *Renal dysfunction: eGFR >30 and <60 mL/min;*
- *Retinopathy;*
- *Micro- or macroalbuminuria;*
- *ABI <0.9 without symptoms of intermittent claudication)*

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[3] Maroko PR, Kjekshus JK, Sobel BE, et al. Factors Influencing Infarct Size Following Experimental Coronary Artery Occlusions. *Circulation*. 1971; 43:67-82.

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[5] Nohria, A et all JACC 41 10 May 2003