CHAPTER 4
Heart Failure

Heart failure accounts for 5% to 10% of hospitalizations among adults throughout sub-Saharan Africa.[1-8] Surveys dating back to the 1950s show that these numbers have not changed substantially for at least the past half-century. In the 1980s and 1990s, the introduction of two-dimensional echocardiography helped clarify the causes of heart failure in this setting.[9] Reports from referral centers on the continent showed that most cases of heart failure were due to non-ischemic cardiomyopathies, congenital heart disease, rheumatic heart disease, or hypertensive heart disease rather than coronary artery disease (see **TABLE 4.1**). Even in urban centers with an increasing prevalence of vascular risk factors, non-ischemic etiologies of heart failure still dominated.[10-18]

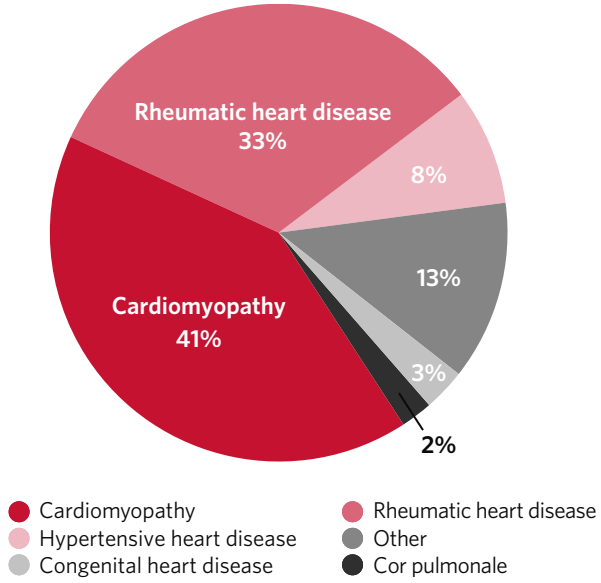
TABLE 4.1 Causes of Heart Failure Reported by Selected Echocardiographic Referral Centers in Sub-Saharan Africa

Authors	Country	n	Age†	RHD* (%)	DCM‡ (%)	EMF§ (%)	HTN HD†† (%)	ICM‡‡ (%)	CHD€ (%)
Amoah et al. 2000[19]	Ghana	572	42	115 (20)	65 (11)	22 (4)	122 (21)	56 (10)	57 (10)
Freers et al. 1996[9]	Uganda	406	N/A	58 (12)	40 (8)	99 (22)	38 (8)	4 (1)	75 (15)
Kingue et al. 2005[20]	Cameroon	167	57	41 (25)**	46 (27)	5 (12)	91 (55)	4 (3)	N/A
Thiam et al. 2002[21]	Senegal	170§§	50	76 (45)**	12 (7)	0 (0)	58 (34)	30 (18)	N/A

† Age = Mean Age; * RHD = Rheumatic heart disease; ‡ DCM = Dilated cardiomyopathies; § EMF = Endomyocardial fibrosis;
†† HTN HD = Hypertensive heart disease; ‡‡ ICM = Ischemic cardiomyopathy ; € CHD = Congenital heart disease
** These series reported total valvulopathies. §§ Patients were double counted if they had two etiologic processes.

In Rwanda, we have found a similar distribution of causes of heart failure at rural district hospitals (see **FIGURE 4.1**). Roughly half the cases are potential surgical candidates.

FIGURE 4.1 Distribution of Causes of Heart Failure in a Rwandan NCD clinic (n = 134)



In the past decade, ultrasound has become increasingly available in sub-Saharan Africa. This has opened up the possibility of decentralizing heart failure diagnosis and treatment from the referral centers to district-level facilities. However, even where the technology is available, few district hospitals have personnel trained in echocardiography. One reason for this is the complexity of traditional cardiology training models.[22]

This chapter offers a simplified approach to heart failure diagnosis and medical management at the district-hospital and health-center levels. The approach is based on four years of experience with more than 300 patients in this setting. It relies on the fact that heart failure in this population is due to a limited number of causes that result in dramatic findings on physical examination or on echocardiography. The approach is designed for rapid training of advanced nurses, clinical officers, and generalist physicians. We have been using this model and teaching it to district-level clinicians in Rwanda since 2007. We have found that with some mentorship and training, Rwandan NCD nurses and district doctors have mastered the required skills quickly. District-level nurses acquire these skills through a three-month integrated training on diagnosis and management of NCDs. Our internal monitoring and evaluation has shown that in almost all cases, clinicians have been able to follow the algorithms, and with them, have been able to independently manage heart failure patients effectively.[23-25]

4.1 Defining Categories of Heart Failure

In resource-poor settings, it is often difficult to confirm a diagnosis of heart failure, let alone differentiate between the many different types of heart failure. This can lead to both over- and underdiagnosis of heart failure as well as initiation of inappropriate treatments.

Our approach teaches district-level clinicians to categorize heart failure into one of five different groups (see **TABLE 4.2**). We have designed these categories to be narrow enough to guide appropriate treatment, yet broad enough to require only minimal echocardiographic and diagnostic skills for classification. Groupings are based on the following findings: evidence of cardiomyopathy, mitral stenosis, a large right ventricle or dilated inferior vena cava (IVC) on echocardiography, or presence of a murmur, cyanosis (in a child or young adult), or severe hypertension on physical exam (see **TABLE 4.4** and **TABLE 4.5**). Each of the groupings created by this approach contain a variety of types of heart failure. However, within each group, the appropriate short-term medical management of the conditions is the same. This approach also identifies patients who may be surgical candidates and therefore prioritizes them for evaluation by a cardiologist.

In this model, heart failure treatment initially takes place at the district-level NCD clinic and not at health centers. One reason for this is that echocardiography should be available at district but not health center–level facilities. Furthermore, heart failure cases are often more complex than patients with other NCDs, requiring a greater level of clinician training for appropriate classification. In some cases, patients who are on stable therapy for many months may be referred back to the health center–level integrated chronic care clinic for routine follow-up.

The role of the cardiologist in this model is restricted to supervising and mentoring the district-level clinicians, and evaluating patients who are potential surgical candidates. Ideally, this secondary evaluation by a fully trained echocardiographer will happen within six months of initial diagnosis.

TABLE 4.2 lists the categories of heart failure important for medical management at the district hospital level. In the following sections, we explain in detail, first, how to recognize each diagnostic finding and, second, how to manage each category of heart failure.

Age is an important consideration in making a diagnosis of heart failure. Some types of heart failure, such as congenital or rheumatic heart disease (excluding mitral stenosis), are more common in children. Mitral stenosis often affects young or middle–aged adults. Hypertensive heart

disease occurs more frequently in older adults and is virtually nonexistent in children. Cardiomyopathies and isolated right–sided heart failure can occur at any age, but are more common in the adult population. **TABLE 4.3** lists the most common cause of heart failure by age.

TABLE 4.2 Important Diagnostic Categories in Heart Failure

1. Cardiomyopathy	
Diagnostic criteria	1. Moderately to severely depressed left ventricular function (ejection fraction < 40%)
2. Hypertensive heart disease (not applicable to children)	
Diagnostic criteria	1. Severe hypertension 2. Shortness of breath 3. Normal to mildly depressed left ventricular function (ejection fraction ≥ 40%)
3. Mitral stenosis (rare before age 25)	
Diagnostic criteria	1. Mitral valve that doesn't open well with typical hockey-stick or elbow deformity 2. Normal to mildly depressed left ventricular function (ejection fraction ≥ 40%)
4. Other valvular heart disease (including rheumatic and congenital)	
Diagnostic criteria	1. Normal to mildly depressed left ventricular function (ejection fraction ≥ 40%) 2. No mitral stenosis 3. Blood pressure ≤ 180/110 mmHg (in an adult) 4. Dramatic heart murmur or cyanosis (in a child or young adult)
5. Isolated right heart failure	
Diagnostic criteria	1. Ejection fraction ≥ 40% 2. No mitral stenosis 3. Blood pressure ≤ 180/110 mmHg (in an adult) 4. No heart murmur 5. Large right ventricle or dilated inferior vena cava on echocardiography

TABLE 4.3 Common Causes of Heart Failure by Age

0–5 years	1. Congenital heart disease (most common) 2. Cardiomyopathy (rare)
6–15 years	1. Congenital heart disease (most common) 2. Acute rheumatic fever (peak age) 3. Rheumatic heart disease (mostly regurgitant lesions, mitral stenosis very rare) 4. Cardiomyopathy (rare)
16–30 years	1. Rheumatic heart disease (most common; regurgitant lesions more frequent than mitral stenosis) 2. Cardiomyopathy (less common, mostly viral, HIV or peripartum) 3. Congenital (less common)
31–40 years	1. Rheumatic heart disease (both mitral stenosis and regurgitant lesions) 2. Cardiomyopathy (as common as rheumatic heart disease, multiple etiologies) 3. Congenital (rare)
> 40 years	1. Cardiomyopathy 2. Rheumatic heart disease (both mitral stenosis and regurgitant lesions) 3. Hypertensive heart disease All etiologies roughly equal in prevalence
Isolated right-sided heart failure can occur at any age because of its many etiologies, but is more common in older patients.	

4.2 Physical Exam Findings for Classification of Heart Failure

Heart failure patients can have a wide variety of physical exam findings. Our diagnostic algorithms use only blood pressure (in adults) and the presence of dramatic murmurs or cyanosis (in a child) to classify patients. Other findings, such as signs of fluid overload, tachycardia, and increased respiratory rate, are important in determining medication adjustments and disposition within each diagnostic algorithm.

Blood pressure in our clinics is obtained with automatic machines. Providers are taught the importance of proper cuff size, and often small adult-sized cuffs are necessary for this low-BMI population. We define severe hypertension in adults as blood pressures greater than 180 mmHg systolic or 110 mmHg diastolic.

Murmurs can be difficult to detect, even with expensive stethoscopes and highly trained ears. However, we ask our clinicians to be able to recognize only the very obvious, loud murmurs, since these are the ones most likely to reflect serious valvular pathology aside from mitral stenosis. We also do not dwell on the characterization of the type or location of the murmurs.

Persistent or intermittent cyanosis in a child outside of acute respiratory illness is often a presenting sign of congenital heart disease. Some of these children will have lesions that do not cause murmurs.

TABLE 4.4 Physical Exam Findings in Classification of Heart Failure

1. Dramatic murmurs
2. Blood pressure ≥ 180 mmHg systolic or 110 mmHg diastolic (in adults)
3. Cyanosis (in a child or young adult)

4.3 Echocardiography for Classification of Heart Failure

Echocardiography is essential to confirm and characterize the diagnosis of heart failure. Our diagnostic and treatment algorithms rely on the clinician's ability to master two echocardiographic views of the heart: the parasternal long axis view and the subcostal view (see below). In those views, clinicians are asked to be able to recognize only four different abnormalities: (1) moderately to severely decreased ejection fraction (EF); (2) mitral stenosis; (3) a very large right ventricle; and (4) an enlarged inferior vena cava. The technical approach to obtaining ultrasound images is described in more detail in the *PIH Manual of Ultrasound for Resource-Limited Settings*.[24]

TABLE 4.5 Echocardiographic Findings in Classification of Heart Failure

1. Moderately to severely decreased EF (< 40%)
2. Mitral stenosis
3. Very large right ventricle (much larger than the aortic root or left atrium)
4. Clearly enlarged and non-collapsing vena cava (≥ 2.5 cm) in an adult

4.3.1 The Parasternal Long Axis View

The parasternal long axis view is obtained in the following manner: with the patient lying on the left side, the probe is placed to the left of the sternum, in the 4th or 5th intercostal space, with the probe marker pointed toward the right shoulder. The mitral valve and aortic valve should be clearly seen (see **FIGURE 4.2** and **FIGURE 4.3**).

FIGURE 4.2 Parasternal Long Axis View

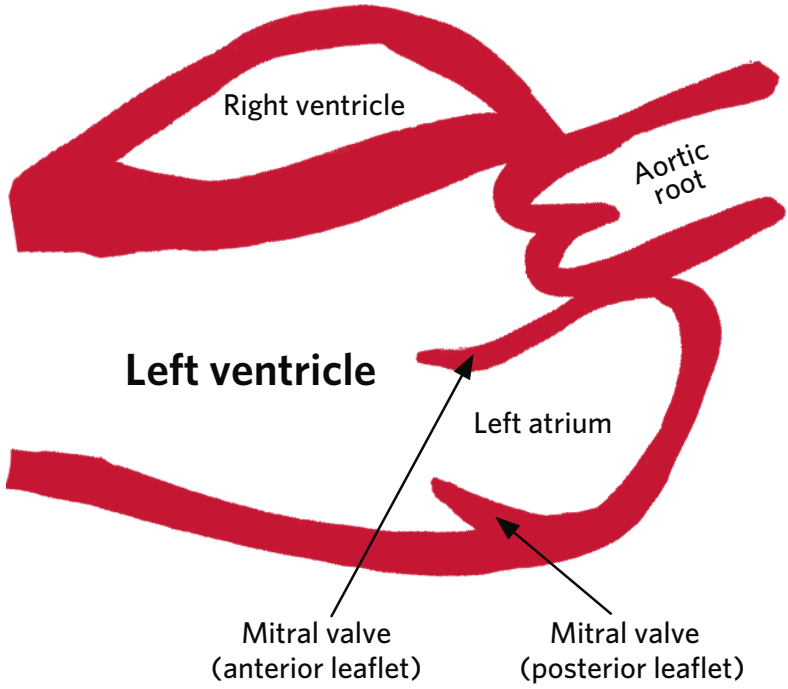
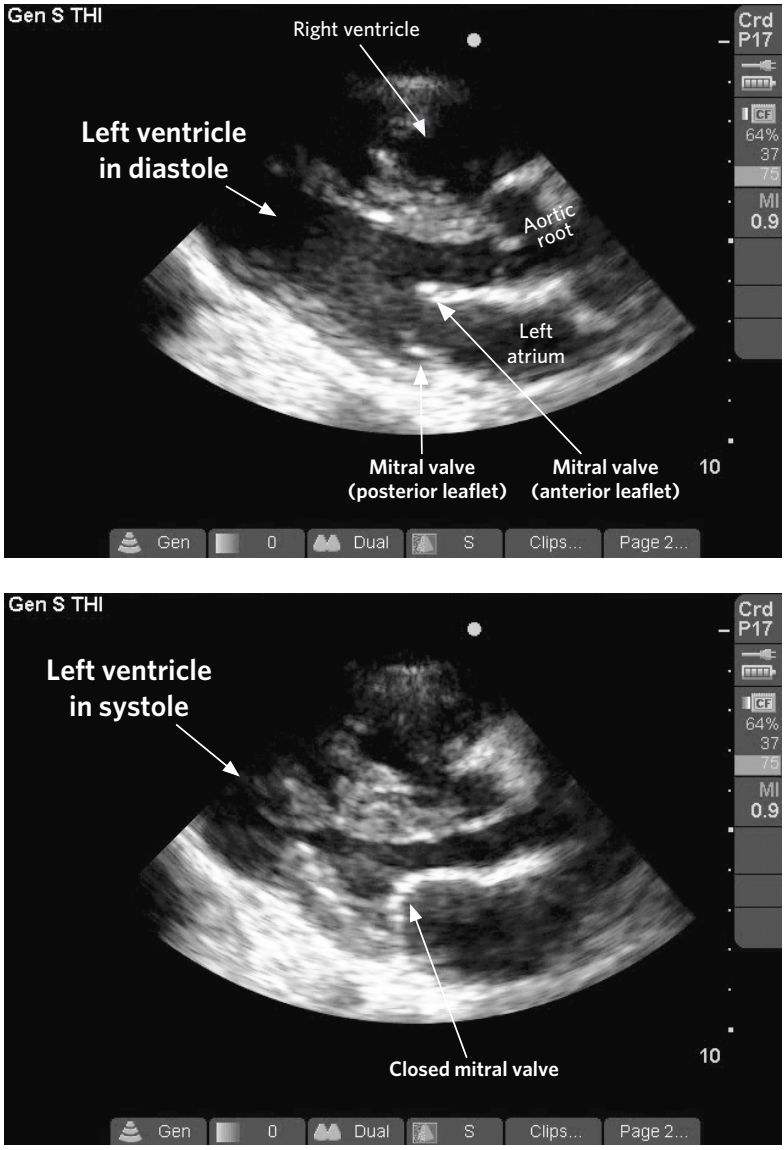


FIGURE 4.3 Normal Parasternal Long Axis View on Echocardiography (Diastole, top; Systole, bottom)



4.3.2 The Subcostal View

The subcostal view is obtained with the probe placed under the xiphoid process. The image generated is of the heart flipped upside down, with the liver seen at the top of the screen (see **FIGURE 4.4**). The IVC is seen as a black tube entering the right ventricle (see **FIGURE 4.5**). It should be measured 2 cm from its entrance into the right ventricle.

FIGURE 4.4 Subcostal View

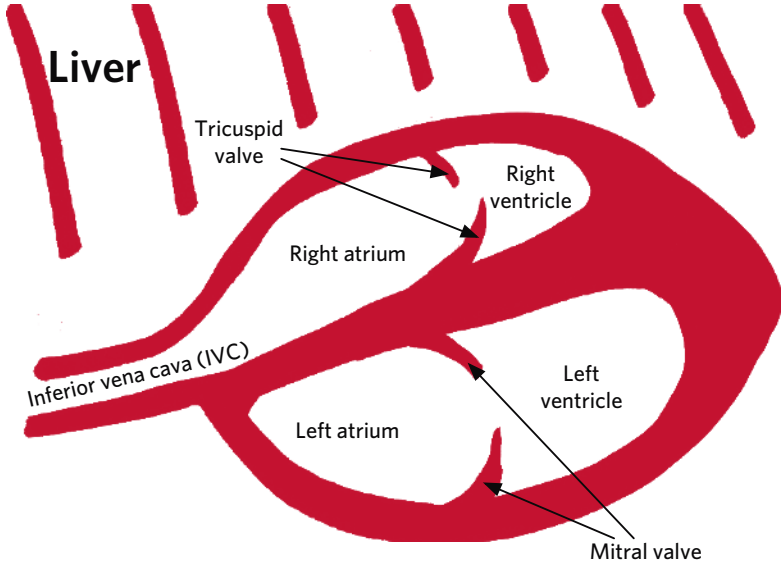
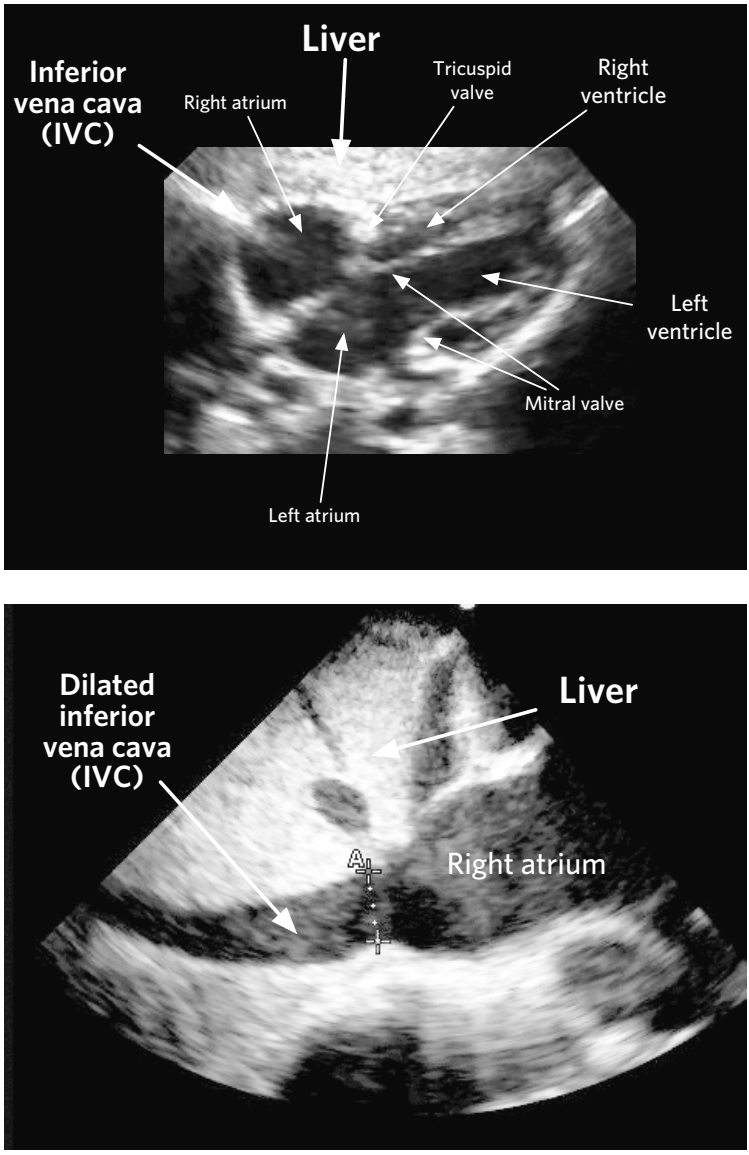


FIGURE 4.5 Subcostal View with Normal IVC (top) and Dilated IVC (bottom)



We have found that as clinicians become more adept at these views, many take it upon themselves to learn the other basic echocardiographic views and findings. This level of echocardiography may be helpful but is not essential in determining appropriate medical management of heart failure. For more in-depth instruction on echocardiography techniques, please refer to the *PIH Manual of Ultrasound for Resource-Limited Settings*.[24]

4.3.3 Ejection Fraction and Fractional Shortening

Evaluation of ejection fraction (EF) is the first and perhaps most important diagnostic skill in categorizing heart failure. EF refers to the amount of blood that the heart pumps out with each beat. Ejection fraction is a measure of the heart's systolic function: how well the heart works during the squeezing phase of the cardiac cycle. A normal EF is between 55% and 65%, meaning that the normal heart pumps about half of the blood in the left ventricle out into the aorta with each beat.

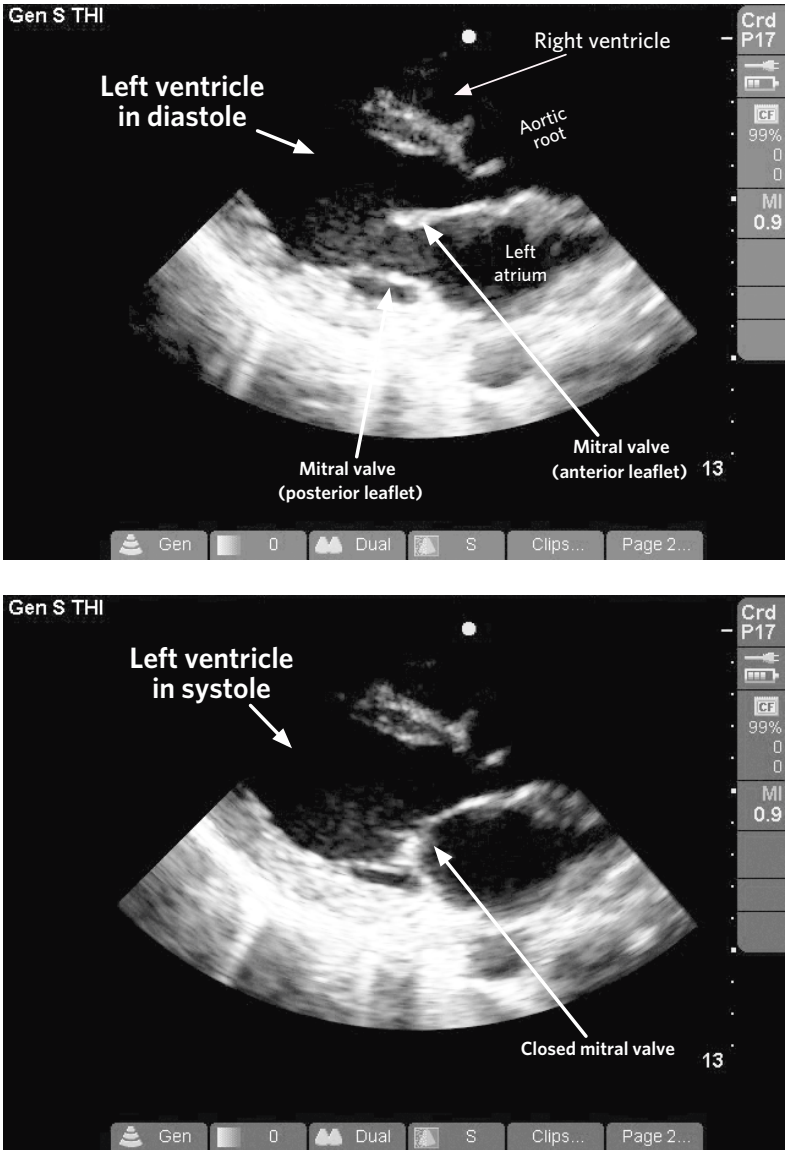
EF is generally classified as normal (≥ 55%), mildly reduced (40%–55%), and moderately to severely reduced (< 40%). A moderately to severely reduced EF means a patient has a cardiomyopathy, a failure of the heart muscle. While echocardiography machines can generate measurements of EF, qualitative visual assessment is more reliable. In a cross-sectional view, this translates into how much the walls of the ventricle are seen to move toward each other with each heartbeat. This distance is called the fractional shortening. A normal ejection fraction of 55% corresponds to a fractional shortening of about 25%. This means that when the normal heart squeezes in systole, the distance between the walls narrows by only 25%.

We teach our clinicians to evaluate EF in the parasternal long view. It can also be assessed in the parasternal short axis, and apical 4-chamber views.

FIGURE 4.3 shows the normal relationship between the heart in diastole (relaxation) and systole (contraction). Notice that the volume of the left ventricle decreases in systole, and the left ventricular walls become thicker as the ventricle contracts and squeezes the blood out into the aorta.

FIGURE 4.6 shows how the left ventricular walls do not move together very much in a heart with a cardiomyopathy—the left ventricle remains about the same size contracted as relaxed, and the walls do not thicken much. Compare this to the normal heart in **FIGURE 4.3**.

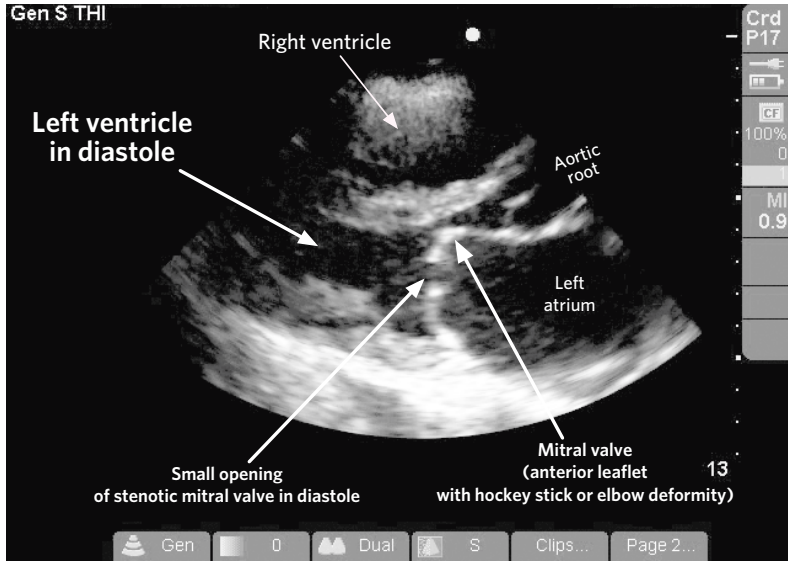
FIGURE 4.6 Cardiomyopathy in Parasternal Long Axis View (Diastole, top; Systole, bottom)



4.3.4 Assessment for Mitral Stenosis

In patients with rheumatic mitral stenosis, the mitral valve has a strikingly characteristic appearance in the parasternal long axis view (see **FIGURE 4.7**). The features of typical rheumatic mitral stenosis include: (1) a small opening of the mitral valve in diastole (see **FIGURE 4.3** and **FIGURE 4.6** for comparison); (2) the anterior leaflet of the mitral valve has a hockey stick or elbow deformity; (3) left ventricular function is typically normal.

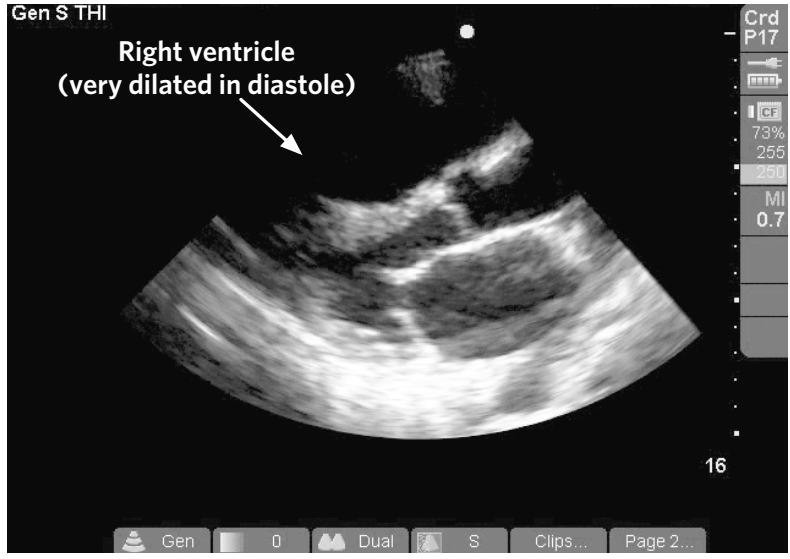
FIGURE 4.7 Mitral Stenosis in Diastole



4.3.5 Assessment of Right Heart Size

In cases of right heart failure, the right ventricle may appear enlarged on the parasternal long axis view (see **FIGURE 4.8**). Normally, the right ventricle is roughly the same size as the aortic root and the left atrium.

FIGURE 4.8 Right Ventricular Enlargement (Parasternal Long View)



4.3.6 Assessment of the Inferior Vena Cava

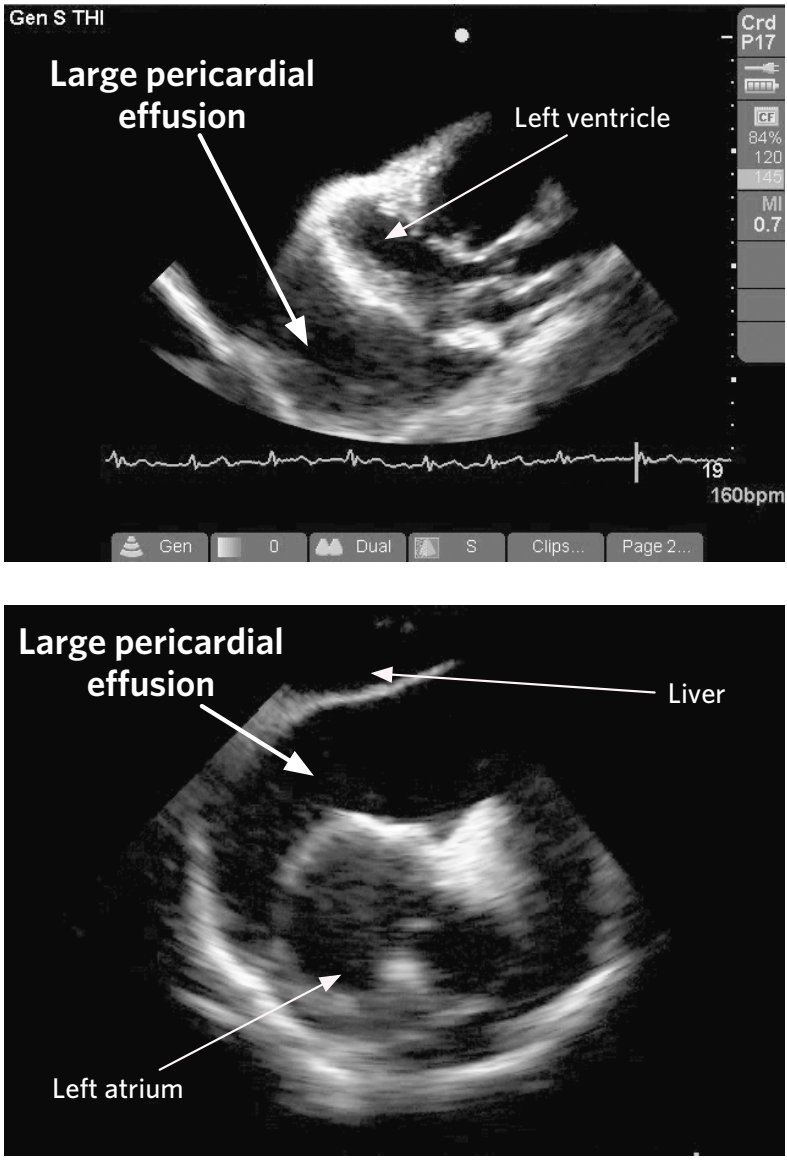
The inferior vena cava in the subcostal view should be small and collapse by 50% with each inspiration. An IVC greater than 2 cm in adults that doesn't collapse is strongly suggestive of some element of right-sided heart failure (see **FIGURE 4.5**).

4.3.7 Assessment of Pericardial Effusion

Pericardial effusions can occur in many different types of heart failure. They can also occur in patients with no history of heart failure, such as those with cancer or renal failure. They are listed here as a key echocardiographic finding because recognition of a very large pericardial effusion (≥ 3 cm in diastole in an adult) in a patient with decompensated heart failure should prompt immediate admission to the district hospital for possible pericardiocentesis. It is not, however, one of the findings we use as a diagnostic criteria for heart failure categorization.

Since fluid appears black on ultrasound, pericardial effusions appear as an extra stripe of black around the heart. This can be seen best in the subcostal view, but can also be seen in the parasternal long axis view (see **FIGURE 4.9**). Many patients may have small pericardial effusions.

FIGURE 4.9 Pericardial Effusion (Parasternal View, top; Subcostal View, bottom)



4.4 Initial Recognition and Referral of the Heart Failure Patient

Heart failure patients may present to any level of the health care system. All patients with suspected or confirmed heart failure should eventually be referred to the district NCD clinic.

At health-center level, heart failure patients often present with non-specific symptoms. In our anecdotal experience, most patients with

clinically important heart failure in Rwanda will have shortness of breath at a decreased level of activity. Clinicians should refer patients to the NCD clinic who do not have an infectious process to explain their shortness of breath or who have any of the findings in **TABLE 4.6** (see below) that suggest an underlying cardiac etiology. **CHAPTER 10** outlines the acute care health center approach to shortness of breath.

TABLE 4.6 Common Signs and Symptoms of Heart Failure (Usually Present in Addition to Shortness of Breath)

1. Lower-extremity edema
2. Loud murmurs
3. Orthopnea (shortness of breath when lying prone)
4. In children: otherwise unexplained cyanosis, lethargy, poor feeding, or poor weight gain

Another subset of patients will have dramatic edema or ascites without shortness of breath. These patients may have isolated right-sided heart failure. They may alternatively have renal or liver failure. All such patients should be referred to the district NCD clinic for echocardiography and lab testing to determine the cause of the edema.

Integrated chronic care providers at health centers may also see patients with symptoms that should prompt referral to the district NCD clinic, such as those with severe hypertension who develop shortness of breath or edema, or patients with wheezing who are not improving with asthma therapy. Triggers to refer to the district-level clinic are addressed in chapters covering these topics.

Very young children with heart failure will present with non-specific symptoms, such as poor feeding, cyanosis, irritability, hypotonia or poor growth. Most children with heart failure will have a valvular or congenital lesion accompanied by a loud murmur. A smaller subset will present with cyanosis without a murmur. Heart failure should be considered in any child with these signs or symptoms.

Another group of heart failure patients will be referred to the outpatient district NCD clinic from the district hospital inpatient units. Many patients with heart failure only seek medical care once they have become very ill and require hospitalization. Algorithms for recognition and management of heart failure patients in the inpatient district hospital are included in the WHO's forthcoming district clinician manual for adult and adolescent care. The WHO algorithms are similar to those presented here for use in the outpatient setting, with an added emphasis on stabilization of the severely decompensated patient. Patients are started on appropriate outpatient therapy prior to discharge and given a follow-up appointment

in the district-level NCD clinic. In some cases, the NCD clinic nurses may see hospitalized heart failure patients together with their physicians to help ensure good continuity of care.

Suspected heart failure patients referred to the district NCD clinic should receive a complete history, physical exam, creatinine testing, and echocardiography. Patients will be classified into one of five heart failure diagnostic categories or receive an alternate, non-cardiac diagnosis.

Clinicians will then determine the initial treatment based on the treatment algorithm for that diagnostic category (see **PROTOCOL 4.1**).

Patients with unstable vital signs, severe symptoms, or dramatic fluid overload are classified as having decompensated heart failure and will be referred for admission to the district hospital for stabilization and diuresis (see **PROTOCOL 4.2**).

Patients are then assessed for the presence or absence of a cardiomyopathy. All patients with an estimated EF of less than 40% are defined as having a cardiomyopathy. Regardless of other abnormalities, these patients will be treated according to the cardiomyopathy treatment guidelines (see **SECTION 4.8**).

Once cardiomyopathy has been ruled out, patients are assessed for the presence of mitral stenosis on echocardiography. These patients are treated as having mitral stenosis, regardless of the presence of other valvular lesions (see **SECTION 4.10**).

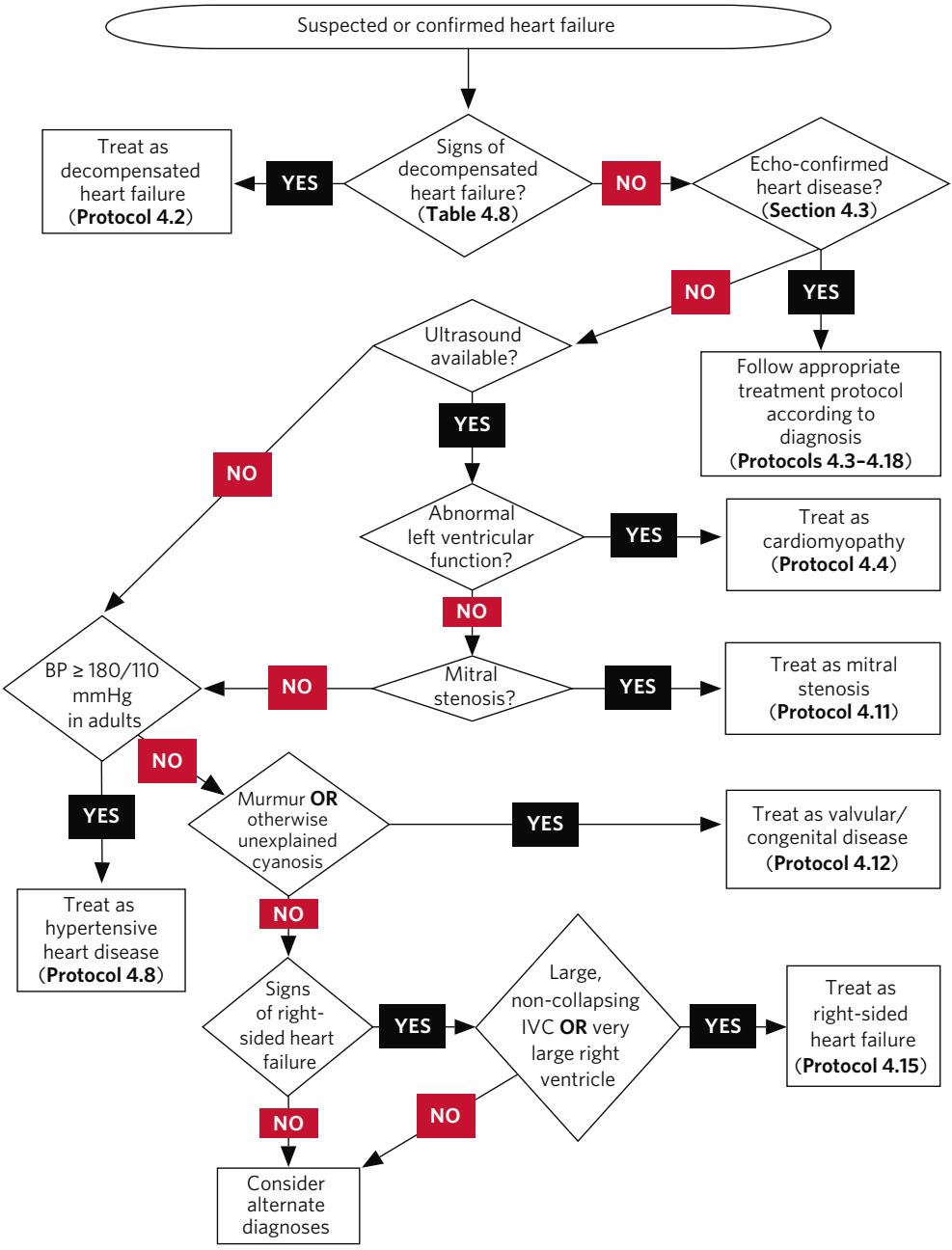
Adult patients without cardiomyopathy or mitral stenosis are then assessed for presence of severe hypertension. If present, they are categorized as having hypertensive heart disease (see **SECTION 4.9**). Hypertensive heart disease is extremely rare in children.

Remaining patients are then assessed for presence of a heart murmur or cyanosis. Those with loud, obvious murmurs without cardiomyopathy, mitral stenosis, or hypertensive heart disease are categorized as having some variety of congenital or rheumatic valvular disease apart from mitral stenosis (see **SECTION 4.11**). Cyanosis with or without a murmur is a common presenting sign of congenital heart disease in children and young adults. Patients with cyanosis without another explanation (e.g., severe respiratory illness) are also placed in this heart failure category. In infants and toddlers, almost all heart failure will be due to congenital lesions. Rheumatic heart disease rarely affects children less than 5 years old.

The remaining patients are evaluated for signs of isolated right heart failure, such as a very large right ventricle or a dilated IVC on echocardiography. These patients are classified as having isolated right heart failure (see **SECTION 4.12**).

Patients with none of the preceding findings are evaluated for other non-cardiac causes of their symptoms, such as renal or liver failure.

PROTOCOL 4.1 Initial Diagnosis and Management of Heart Failure



Next, all treatment algorithms begin with an assessment of the patient's symptom severity and volume status. This assessment will be discussed in the following sections.

4.5 Heart Failure Severity Classification

Patients with heart failure of any etiology fall along a common continuum of symptom severity. These are categorized into four groups, based on the New York Heart Association classification system (see **TABLE 4.7**). Understanding this classification system allows the NCD clinician to clearly document a heart failure patient's clinical course. Sometimes patients may fall between two classes. This classification system is not as useful for very young children.

Mild abnormalities of heart structures in many cases never lead to symptoms of heart failure. Patients with moderate to severe abnormalities may also be asymptomatic or mildly symptomatic (class I or II) for months to years, thanks to the body's compensatory mechanisms. However, as heart failure progresses, these compensatory mechanisms generally fail and patients develop more symptoms, placing them in a higher heart failure class. Heart failure treatment can slow and sometimes even reverse this progression, allowing some patients to move to a lower heart failure class.

TABLE 4.7 New York Heart Association Heart Failure Classification

Class I	Patients with cardiac disease but no resulting limitation of physical activity. Ordinary physical activity does not cause symptoms.
Class II	Patients with cardiac disease resulting in mild limitation of physical activity. Patients are comfortable at rest. Ordinary physical activity (farming, running, carrying water, climbing a hill) causes symptoms.
Class III	Patients with cardiac disease resulting in moderate limitation of physical activity. Patients are comfortable at rest. Less than ordinary physical activity (light housework, walking on flat ground) causes symptoms.
Class IV	Patients with cardiac disease resulting in severe limitation of physical activity. Patients have symptoms at rest. Any physical activity causes symptoms.

4.6 Decompensated Heart Failure

When a patient's body is no longer compensating well for the degree of heart dysfunction, volume status and functional ability will worsen. This is termed decompensated heart failure. Clues that a patient has entered this state are abnormal vital signs (such as low blood pressure and fast heart rate) and severe symptoms of dyspnea and orthopnea (see **TABLE 4.8**). Again, children may present with different signs and symptoms, such as lethargy, cyanosis, or hypotonia. Vital sign parameters are also different in children (see **APPENDIX E**). These patients are too sick to be treated in the community and require inpatient management of their fluid status and other symptoms. **PROTOCOL 4.2** provides guidance on how to evaluate and initiate treatment for patients with decompensated heart failure prior to transfer to the inpatient unit.

TABLE 4.8 Signs and Symptoms of Decompensated Heart Failure

Vital signs (if child, see **APPENDIX E** for normal vital sign ranges by age)	Blood pressure	Very low (SBP ≤ 80 mmHg in adults) Very high (SBP ≥ 180 mmHg in adults)
	Pulse	Very low (≤ 40 bpm in adults) Very high (≥ 120 bpm in adults)
	High respiratory rate	≥ 24 breaths/minute in adults
	Low oxygen saturation	Saturation ≤ 90% (note patients with a cyanotic congenital lesion may be stable at a low oxygen saturation)
Symptoms	Inability to lie down flat Severe dyspnea at rest **In children ≤ 5 years:** hypotonia, new or worsened cyanosis, lethargy, poor feeding	

Assessment of vital signs and the patient's overall condition is the first step in evaluating any heart failure patient. This initial assessment should determine whether or not the patient is decompensated and in need of hospitalization for treatment. Refer to **APPENDIX E** for a guide to normal vital signs by age in children.

The following vital signs should be collected on every visit: heart rate, blood pressure, and weight. Oxygen saturation, respiratory rate, and temperature should be checked if a patient seems ill or in distress.

Patients with heart failure can often be in a stable condition with low blood pressures (as low as systolic blood pressure of 80 mmHg in adults). If a patient feels well and is not tachycardic, a low blood pressure can be tolerated. However, these patients are more fragile than others and medication adjustments should thus be made even more carefully.

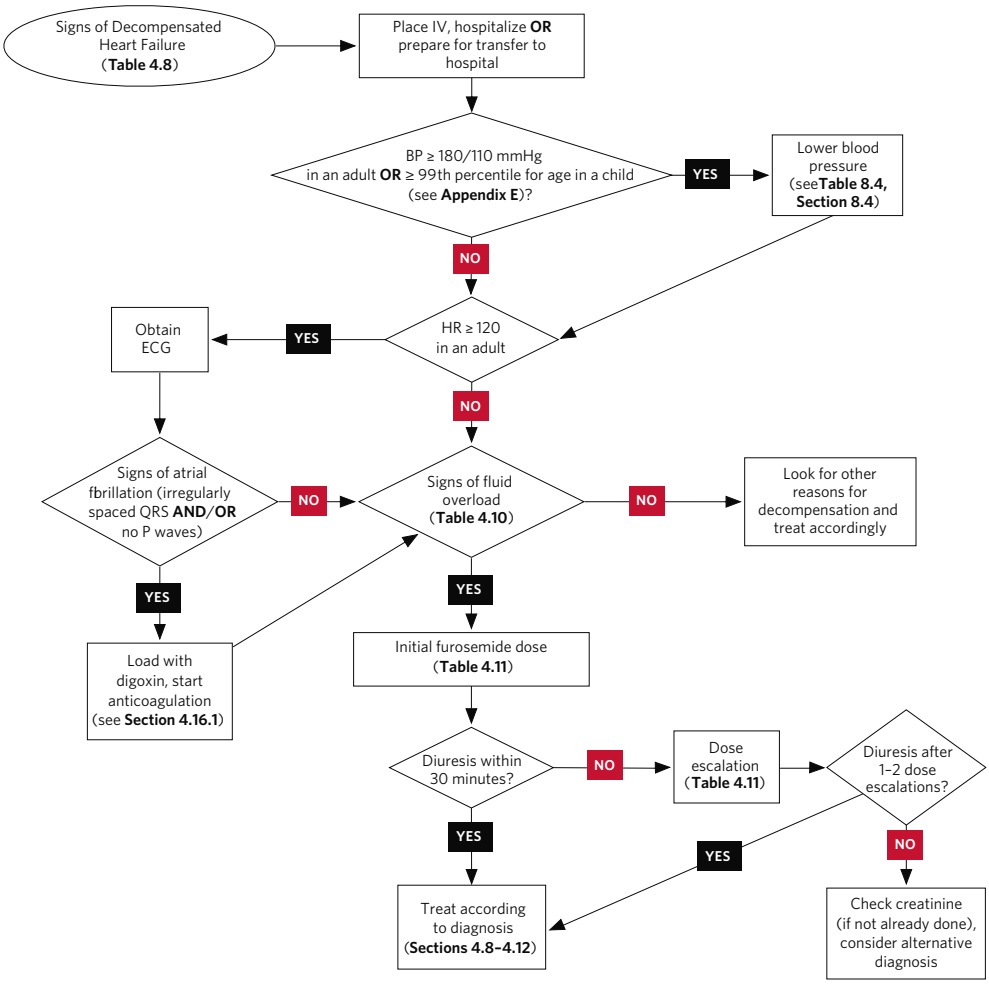
TABLE 4.9 lists some of the common reasons for acute worsening of a heart failure patient. A very high blood pressure (≥ 180/110 mmHg in an adult) can result in severe decompensation of the heart failure patient. Acute increases in blood pressure may cause the heart to stiffen and a subsequent backup of fluid into the lungs, resulting in acute pulmonary edema. Heart failure patients with signs of decompensation and very high blood pressures should receive immediate interventions to lower blood pressure (see **SECTION 8.4** and **TABLE 8.4**).

Large changes in vital signs or very abnormal values should trigger (1) investigation of the cause; (2) initial attempts to stabilize the patient; and (3) preparations for transfer to a higher level of care. See **PROTOCOL 4.2** for the algorithm for initial management of decompensated heart failure.

TABLE 4.9 Reasons for Acute Decompensation in Patients with Heart Failure

1. Medication nonadherence or recent changes in medications
2. Change in diet (e.g., increase in salt intake)
3. Acute illness (e.g., pneumonia, rheumatic fever, endocarditis)
5. Worsening valvular disease
6. Pregnancy Exacerbates all types of heart failure, but especially mitral stenosis (2nd and 3rd trimester) and peripartum cardiomyopathy.
7. Arrhythmia Especially common in patients with cardiomyopathies and valvular disease, particularly mitral stenosis.
8. Worsened hypertension Very high blood pressure can cause acute stiffening of the heart muscle and back up of fluid into the lungs (pulmonary edema).

PROTOCOL 4.2 Management of Decompensated Heart Failure



4.7 Fluid Status Assessment

After assessment of vital signs, assessment of fluid status is the next step in management of any patient with known or suspected heart failure.

Fluid retention occurs because the kidneys, to compensate for poor cardiac function, hold on to extra fluid to try to maintain adequate tissue blood flow. At a certain point, this compensatory mechanism fails, as the extra fluid leaves the blood and goes into tissue, leading to congestion of the lungs and/or the extremities and abdomen. This extra fluid results in many of the symptoms of heart failure. Moving this fluid out of the tissues and out of the body through the urine is called diuresis, and medications that promote this process are called diuretics. Furosemide (trade name Lasix) is the most commonly used diuretic in heart failure.

Assessing volume status accurately can be difficult. There is no one test or finding that accurately diagnoses volume imbalances, but rather a collection of physical findings and symptoms that need to be taken into consideration in determining the nature and degree of fluid imbalance. With practice, clinicians should be able to make a reasonable assessment of fluid status, with the warning that even experts sometimes get this assessment wrong. Here as in most of medicine, if a therapy does not produce the expected result, the diagnosis should be reconsidered.

Heart failure patients will fall into three broad volume status categories: (1) hypovolemic, meaning the patient has too little fluid in the body; (2) euvolemic, meaning the patient is in good fluid balance (neither too much nor too little) and is at a dry weight; or (3) hypervolemic, meaning the patient has an excess of fluid in the body.

Within the hypervolemic category, there are three subcategories based on severity of fluid overload: mild, moderate, and severe. Categorization is based on assessment of symptoms, physical findings, and comparison of current weight to the patient's dry weight, if known.

Assessing fluid status involves (1) asking the patient about symptoms of fluid overload (i.e., orthopnea, dyspnea); (2) examining the patient, making special note of whether the neck veins are distended, whether there are crackles on lung exam, and whether there is any lower-extremity edema or ascites; and (3) measuring creatinine if the patient is new to the clinic and on a regular basis thereafter.

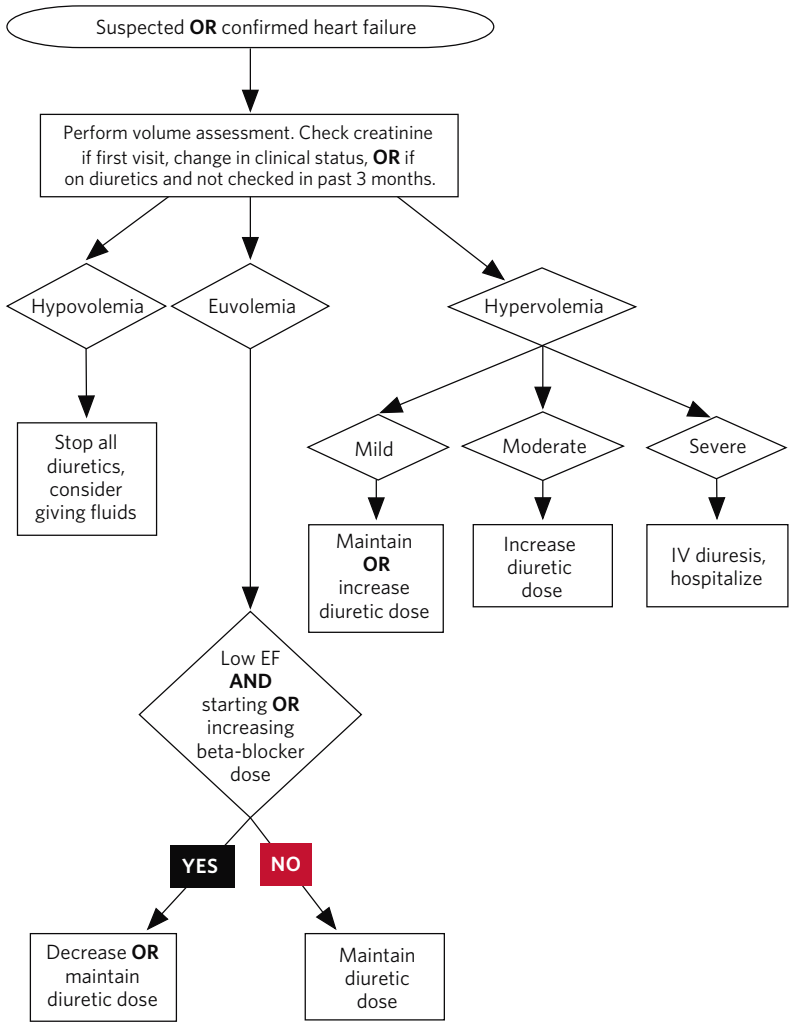
TABLE 4.10 Volume Status Categories and Diuretic Adjustment

Category	Hypovolemic	Euvolemic	Hypervolemic		
			Mild	*Moderate*	*Severe (decompensated)*
Weight	Less than dry weight	At dry weight	≤ 5 kg above dry weight (in an adult)	≥ 5 kg above dry weight (in an adult)	≥ 5 kg above dry weight (in an adult)
Symptoms	Variable	Class I–II	Class I–II	Class II–III	Class III–IV
Vital signs	Tachycardia Hypotension	Normal	Normal	Mild tachycardia	Tachycardia, tachypnea, hypoxia, hypotension, or hypertension
Physical exam	Does not always correlate with severity. Signs of fluid overload include distended neck veins, lung crackles, louder murmurs, hepatomegaly, ascites, and lower-extremity edema. However, can be intravascularly hypovolemic and still have signs of hypervolemia in lungs, extremities, and abdomen.				
Creatinine	Increased	Stable or decreased	Stable or decreased	Can be increased (due to hypoperfusion of the kidneys), stable or decreased.	
Diuretic adjustment	Stop all diuretics	Reduce or maintain dose (if starting beta-blocker, do not reduce diuretic)	Maintain or increase dose	Increase dose	Start IV furosemide and prepare for transfer to hospital if possible

With diuresis, these symptoms and physical findings should improve (see **TABLE 4.10**). However, patients with low serum protein, significant right-sided failure, or renal failure may continue to have residual edema despite intravascular euvolemia. As a patient gets close to his dry weight, the creatinine may start to rise. This is one sign that it may be time to decrease a patient's diuretic dose. All patients with suspected heart failure should have a creatinine checked on the initial visit and then at least every three months if they are on diuretics. A creatinine should also be measured if there is a change in the patient's clinical status.

When a patient is deemed to be fluid-overloaded, the key clinical decision is whether the patient requires hospitalization for fast, intravenous diuresis, or whether the fluid can be taken off more slowly with oral diuresis as an outpatient. A patient who appears decompensated due to fluid overload should be hospitalized for intravenous diuresis. **PROTOCOL 4.3** presents an algorithm for appropriate management of diuretics according to fluid status assessment.

PROTOCOL 4.3 Volume Status Assessment and Diuretic Adjustment



Patients with renal failure pose a special challenge for diuretic management. If a patient is very fluid-overloaded, removal of fluid can improve the heart's ability to pump and increase the amount of blood delivered to the kidneys. Renal function will improve, with a resulting drop in creatinine levels. However, if the patient is not fluid-overloaded or if too much fluid is removed, blood flow to the kidneys will decrease and renal function will decline, with a resulting rise in creatinine levels. Therefore, it is important to pay attention to the trend in creatinine as a clue to the patient's current volume status.

In fact, because the kidneys do not benefit from any of the fluid stored outside of the blood vessels and in the tissues, a patient can have too

much fluid stored in the feet, abdomen, and lungs, yet not enough fluid in the blood. This situation will also lead to increased creatinine. For this reason, we generally recommend that clinicians err on the side of keeping patients slightly fluid-overloaded.

In this chapter, we use a creatinine cutoff of 200 µmol/L in adults or 2x normal creatinine for age in children as a definition of renal failure. These values should fall within class 3 kidney disease (CKD 3) and correspond with a glomerular filtration rate (GFR) between 30 and 60 mL/min. **CHAPTER 6** discusses the diagnosis and management of renal failure in more detail.

4.7.1 Guidelines for Initiating Intravenous Diuresis

All patients who are decompensated with any signs of hypervolemia will require intravenous diuresis. As the body becomes increasingly fluid-overloaded, the tissues become edematous. This includes the gut, which can lead to poor absorption of oral medications. For this reason, intravenous furosemide is the preferred medication for patients with severe fluid overload.

Intravenous diuresis should be initiated immediately upon diagnosis of severe fluid overload while preparing for transfer to the hospital. Furosemide should work quickly, with an average onset of 15 minutes from administration. If the patient does not respond, it may be that the threshold for diuresis has not been reached and a higher dose is needed. If the patient does not urinate within 30 minutes of the first dose, the dose can be doubled. **TABLE 4.11** provides suggested starting, escalation, and maximum doses of intravenous furosemide. Intravenous furosemide lasts about 2 hours and should be dosed 2–4 times per day.

TABLE 4.11 Intravenous Furosemide Dosing

<table>
<tr><th colspan="2">Initial dosing</th><th>Escalation</th><th>Maximum dose</th></tr>
<tr><td rowspan="2">Patient does not already take furosemide</td><td>Adult:
40 mg IV 2x–3x/day</td><td rowspan="3">Double every 30 minutes until response or maximum dose achieved</td><td>Adult:
100 mg IV 2–4x/day</td></tr>
<tr><td>Pediatric (≤ 40 kg):
0.5 mg/kg IV 2x/day</td><td rowspan="2">Pediatric:
4 mg/kg IV
2–4x/day</td></tr>
<tr><td>Patient already takes furosemide</td><td>Double effective outpatient dose and give as IV</td></tr>
</table>

Furosemide is twice as effective when given intravenously as when given orally. It also acts faster and wears off more quickly. It is important to keep this in mind when transitioning patients from one form of the drug to another (see **TABLE 4.12**).

TABLE 4.12 Equivalent Oral and IV Furosemide Doses

Oral	IV
80 mg PO	40 mg IV

Once a patient has been diuresed effectively, it is very important to make note of the dry weight—the weight at which the patient is euvolemic. Any weight gain over that can be considered fluid gain, unless there is reason to believe that the patient has other reasons to gain weight (i.e., a growing child). When a patient is discharged from the hospital, this value should be communicated to the outpatient provider.

4.7.2 Guidelines for Initiating and Titrating Oral Furosemide at Health-Center Level

Most patients with heart failure will have a tendency to store too much extra fluid. It may be possible to wean some patients off all diuretics with proper therapy, but most will require a low long-term maintenance dose. This means that patients who were previously hypervolemic but become euvolemic after diuresis will still often require diuretics to prevent reaccumulation of fluid. In these patients and in those who are mildly hypervolemic, oral diuretics can be used to maintain good fluid balance and to control symptoms. **TABLE 4.13** outlines the recommended dosing of oral furosemide.

TABLE 4.13 Oral Furosemide Dosing

Dose adjustment					
Hypovolemia	**Euvolemia**	**Mild hypervolemia**	**Moderate hypervolemia**	**Severe hypervolemia**	
Stop all diuretics, consider giving fluid	May attempt to decrease dose unless starting or increasing beta-blocker	Keep dose the same	Increase current dose (by no more than double)	Admission and intravenous diuresis (see **TABLE 4.11**)	
Adult dosing					
Initial dose: 40 mg 1x/day. **Maximum dose:** 80 mg 2x/day.					
Pediatric dosing	**Starting doses**				
	< 10 kg	**10 kg**	**15 kg**	**20 kg**	**30 kg**
40 mg tablet	See mg/kg dosing*	10 mg ¼ tab 1x/day	10–20 mg ¼–½ tab 1x/day	20 mg ½ tab 1x/day	20–40 mg ½–1 tab 1x/day
Initial dose: 1 mg/kg 1x/day. **Increase by:** 0.5–1 mg/kg per dose or increase frequency of dosing. **Maximum dose:** 4 mg/kg 2–4x/day.					

* May require crushing pills and diluting to achieve appropriate dose. This should only be done under the supervision of an experienced clinician.

4.7.3 Guidelines for Use of Other Diuretics

Other oral diuretics besides furosemide may be used to help achieve and maintain euvolemia (see **TABLE 4.14**). These medications are generally added to furosemide to increase the strength of diuresis. While they may be very effective, these medications can also increase the chances of electrolyte abnormalities and other side effects of furosemide, and should be used with caution.

4.7.3.1 Spironolactone

Spironolactone (trade name Aldactone), like furosemide, acts on the kidneys to increase urination. While furosemide tends to cause hypokalemia, spironolactone will maintain or increase blood potassium levels. Spironolactone can be particularly effective in patients with ascites. Because spironolactone can increase blood potassium levels, it should only be used in the setting of normal renal function (defined here as a creatinine ≤ 100 µmol/L in adults or within the normal range for age in children (see **APPENDIX E** for pediatric ranges)).

4.7.3.2 Hydrochlorothiazide

Hydrochlorothiazide is a weaker diuretic than furosemide, but acts in much the same way. When taken 30 minutes prior to furosemide, hydrochlorothiazide can make furosemide work better, causing a larger diuresis. A very small number of patients should require this. This combination can cause massive diuresis and loss of vital electrolytes, and therefore should only be considered in consultation with a physician and with very close monitoring of electrolytes.

TABLE 4.14 Other Oral Diuretic Dosing

Drug	Initial dosing	Notes	Maximum dose
Spironolactone	**Adult:** 25 mg 1x/day	Add if patient has significant ascites and normal renal function	50 mg/day
	Pediatric (≤ 40 kg): 2.5 mg/kg 1x/day		3 mg/kg/day
Hydrochlorothiazide	**Adult:** 12.5 mg 1x/day	Can be used if furosemide is not available or added to furosemide if greater diuresis is desired.	25 mg/day
	Pediatric (≤ 40 kg): 1 mg/kg/day		12 mg/day

4.7.4 Dangers of Diuresis

Diuretics are powerful medications. If used in excess, they can cause excessive water loss and resulting damage to the kidneys. This in turn can cause dangerous electrolyte imbalances and even death.

For this reason, it is important to err on the side of using smaller doses and keeping patients slightly hypervolemic. Patients must be educated on the dangers of overdiuresis and instructed to stop their diuretic

medications if they develop fever or diarrhea, which in combination with medications can lead to serious dehydration.

When adding two diuretics together, the effect on electrolytes is increased. Combinations should be used only in the setting of normal creatinine. If possible, sodium, potassium, and creatinine should be monitored at regular intervals in these situations.

4.8 Cardiomyopathy

The term cardiomyopathy encompasses several types of heart disease, all of which lead to a similar outcome of heart muscle dysfunction. The common feature of all these diseases is a low ejection fraction, usually less than 40% (fractional shortening less than 20%).

There are many different causes of cardiomyopathy (see **TABLE 4.15**). As stated above, some types of heart failure begin as a cardiomyopathy. Others, however, such as valvular or hypertensive heart disease, can progress to a cardiomyopathy over time. Any patient with an ejection fraction less than 40%, regardless of the cause, should be treated primarily as a cardiomyopathy. Cardiomyopathies affect patients of every age group but are rare in children.

TABLE 4.15 Etiology of Cardiomyopathy in Rwanda

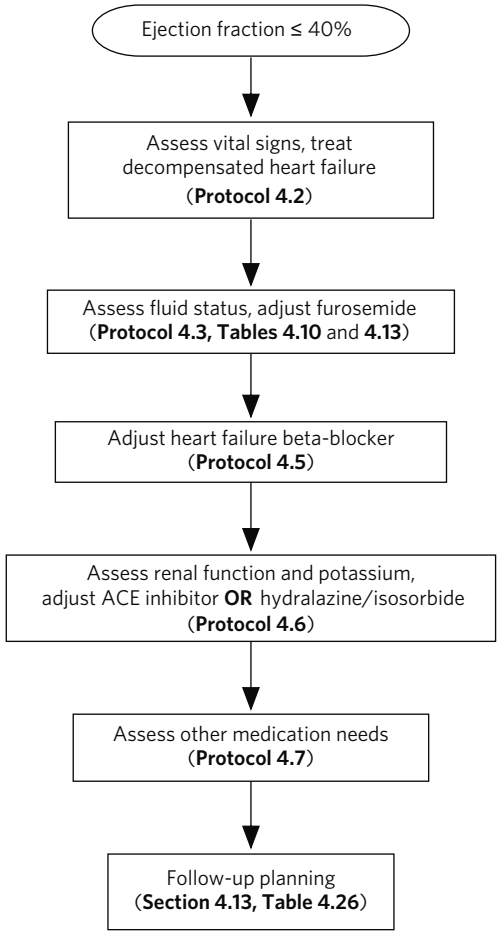
Idiopathic	Most of the cases of cardiomyopathy are caused by an unknown process. Many may be due to a previous viral infection.
HIV cardiomyopathy	HIV can cause direct cellular damage to heart muscle. ARVs can delay or reverse this process.
Alcohol-related cardiomyopathy	Chronic alcohol use can be toxic to the heart muscle. If the patient stops drinking alcohol, the function may improve with time.
Peripartum	Women can develop a cardiomyopathy in the last month of pregnancy and up to six months postpartum. This often improves with appropriate management and avoidance of subsequent pregnancies.
Viral myocarditis	Many different viruses can infect the heart muscle and cause dysfunction. This often improves with time, but may be permanent.
Anemia	Severe anemia (sometimes due to cancer or poor nutrition) can lead to cardiomyopathy. This can improve when the anemia is treated.
Advanced valvular disease	Most valvular conditions causing abnormal flow of blood within the heart can lead to a cardiomyopathy. This is particularly true with regurgitant lesions. This is not the case for pure mitral stenosis, in which the left ventricle is protected, while the right ventricle ultimately suffers.

In many cases, identifying the cause of the cardiomyopathy is not important, because the clinical management will be the same. However, several causes can be corrected with proper additional therapy. Patients

with anemia-induced cardiomyopathy may improve as their anemia is corrected. Likewise, ARV therapy may reverse some of the systolic dysfunction in patients with HIV-induced cardiomyopathy as well as prevent non-cardiac HIV-related complications. For these reasons, all patients with a newly diagnosed cardiomyopathy should have their hemoglobin and HIV serotest checked. In addition, all patients with a newly diagnosed cardiomyopathy should be asked about their alcohol intake. Cardiomyopathy is a rare cause of heart failure in children, but may occur as a result of a viral infection or from congenital or rheumatic valvular lesions.

All cardiomyopathies, regardless of etiology, are managed with diuretics and with medications, such as beta-blockers and ACE inhibitors, which have been proven in clinical trials to decrease risk of death in adults and improve symptoms over the long term. **PROTOCOL 4.4** provides an outline for cardiomyopathy management.

PROTOCOL 4.4 Cardiomyopathy Management



4.8.1 Vital Sign Assessment

As in all types of heart failure, cardiomyopathy management begins with an assessment of the patient's vital signs, weight, and overall condition. Patients with cardiomyopathies will often have low blood pressures. However, heart failure patients with systolic low blood pressures (below 80 mmHg in an adult or below normal range for age in a child), along with other signs of decompensation (tachycardia, respiratory distress, altered mental status), should be hospitalized.

4.8.2 Fluid Status Assessment

The next step in the management of cardiomyopathy is evaluation of fluid status and titration of diuretics. In the case of a cardiomyopathy, diuretic doses should not be reduced if beta-blockers are going to be added or increased at the same visit. This is because, in the short-term, beta-blockers can exacerbate fluid retention.

4.8.3 Titration of Mortality-Reducing Medications

Beta-blockers, ACE inhibitors, spironolactone, and the combination of hydralazine and isosorbide dinitrate have all been shown to decrease morbidity and mortality among adult cardiomyopathy patients when used at doses proven effective in large trials.[26] The provider's goal is to increase the doses of these drugs until reaching a target dose. However, this increase must be achieved gradually, and there are certain factors that make even small increases of these drugs dangerous. If side effects develop, the provider must be able to identify what drug is the culprit. Therefore, only one drug should be increased at each visit. In general, if the patient is euvolemic and stable, it is best to increase only the patient's beta-blocker at the first visit. If there are contraindications to increasing the beta-blocker, the ACE inhibitor can be increased first. At the second visit, the ACE inhibitor may be increased. In this manner, medications are increased one at a time until goal doses are achieved or dose-limiting side effects occur.

These medications are not as well–studied in children. It is difficult to conduct randomized trials for patients with a rare disease. However, there is reason to think that these medications may confer similar benefits in children as in adults. Additionally these medications have been found to be safe for use in childhood cardiomyopathy if appropriately low doses are administered.

There is no good data on the use of isosorbide and hydralazine in children. However, the clinician might consider its use in a child with cardiomyopathy who also has renal failure precluding the use of an ACE inhibitor. This should be done in close consultation with a pediatrician or cardiologist as the hemodynamic effects of these medications in children can be unpredictable.

TABLE 4.16 Mortality-Reducing Medications for Cardiomyopathy in Adults

Beta-blocker			
	Starting dose	**Dose change**	**Target dose**
Carvedilol	3.125–6.25 mg 2x/day	3.125–6.25 mg 2x/day	25 mg 2x/day
Atenolol*	12.5 mg 1x/day	12.5 mg 1x/day	50 mg 1x/day
ACE inhibitor			
	Starting dose	**Dose change**	**Target dose**
Lisinopril	5 mg 1x/day	5 mg 1x/day	20 mg 1x/day
Captopril	12.5 mg 3x/day	12.5 mg 3x/day	50 mg 3x/day
Enalapril	2.5 mg 2x/day	2.5 mg 2x/day	10–20 mg 2x/day
Hydralazine/isosorbide dinitrate (Contraindications to beta-blocker and/or ACE inhibitor)			
	Starting dose	**Dose change**	**Target dose**
Hydralazine	25 mg 3x/day	25 mg 3x/day	50 mg 3x/day
Isosorbide	10 mg 3x/day	10 mg 3x/day	30 mg 3x/day
Potassium-sparing diuretic			
	Starting dose	**Dose change**	**Target dose**
Spironolactone	12.5–25 mg 1x/day	12.5 mg 1x/day	25 mg 1x/day

* Only if no carvedilol or other heart failure beta-blocker available.

TABLE 4.17 Medications for Cardiomyopathy in Children (≤ 40 kg)

Beta Blockers					
Starting doses	**< 10 kg**	**10 kg**	**15 kg**	**20 kg**	**30 kg**
Carvedilol 6.25 mg tablet	See mg/kg dosing*	See mg/kg dosing*	See mg/kg dosing*	1.5 mg ¼ tab 2x/day	1.5 mg ¼ tab 2x/day
Initial dose: 0.05 mg/kg/dose 2x/day. **Maximum dose:** 0.4 mg/kg/dose 2x/day **OR** 25 mg 2x/day.					
Atenolol 50 mg tablet	See mg/kg dosing*	See mg/kg dosing*	12.5 mg ¼ tab 1x/day	12.5–25 mg ¼–½ tab 1x/day	25 mg ½ tab 1x/day
Initial dose: 0.5–1 mg/kg as one daily dose. **Maximum dose:** 2 mg/kg as one daily dose.					
ACE Inhibitors					
Starting doses	**< 10 kg**	**10 kg**	**15 kg**	**20 kg**	**30 kg**
Lisinopril 10 mg tablet	See mg/kg dosing*	See mg/kg dosing*	See mg/kg dosing*	2.5 mg ¼ tab 1x/day	2.5 mg ¼ tab 1x/day
Initial dose: 0.07 mg/kg/day as one daily dose. **Maximum dose:** 0.6 mg/kg/day or 20 mg as one daily dose. **Notes:** Do not use in children with a creatinine ≥ 2x the normal value for age (see **APPENDIX E** for normal ranges). Not safe for use in pregnancy.					
Captopril 25 mg tablet	See mg/kg dosing*	6.25 mg ¼ tab 2x/day	6.25 mg ¼ tab 3x/day	6.25–12.5 mg ¼–½ tab 2x/day	12.5 mg ½ tab 3x/day
Initial dose: 0.3–0.5 mg/kg/dose 2–3x/day. **Maximum dose:** 2 mg/kg/dose 2–3x/day. **Notes:** Do not use in children with a creatinine ≥ 2x the normal value for age (see **APPENDIX E** for normal ranges). Not safe for use in pregnancy.					
Hydralazine/Isosorbide Dinitrate (Contraindications to B-blocker and/or ACE inhibitor)**					
Starting doses	**< 10 kg**	**10 kg**	**15 kg**	**20 kg**	**30 kg**
Hydralazine 50 mg tablet	See mg/kg dosing*	See mg/kg dosing*	See mg/kg dosing*	See mg/kg dosing*	12.5 mg ½ tab 3x/day
Initial dose: 0.25–0.3 mg/kg/dose 3x/day. **Maximum dose:** 1.7 mg/kg/dose 3x/day or 25 mg 3x/day.					
Isosorbide dinitrate 10 mg tablet	See mg/kg dosing*	See mg/kg dosing*	2.25 mg ¼ tab 3x/day	2.25 mg ¼ tab 3x/day	5 mg ½ tab 3x/day
Initial dose: 0.15 mg/kg/dose 3x/day. **Maximum dose:** 0.5 mg/kg/dose 3x/day or 10 mg 3x/day.					

Potassium-Sparing Diuretic					
Starting doses	**< 10 kg**	**10 kg**	**15 kg**	**20 kg**	**30 kg**
Spironolactone 25 mg tablet	See mg/kg dosing*	12.5 mg ½ tab 1x/day	12.5–25 mg ½–1 tab 1x/day	25 mg 1 tab 1x/day	25–50 mg 1–2 tabs 1x/day
Initial dose: 1 mg/kg as one daily dose. **Maximum dose:** 3 mg/kg/dose given once a day or 50 mg as one daily dose. **Notes:** Do not use in children with a creatinine > the normal value for age (see **APPENDIX E** for normal ranges). Consider cutting the dose by half if patient is also on furosemide.					

* Note that dosing medications for small children may require crushing pills and diluting. This should only be done under the supervision of an experienced clinician.

** There is no good evidence of the efficacy of this medication combination for the treatment of heart failure in children. We recommend initiating these medications only in consultation with a pediatrician or cardiologist.

4.8.3.1 Beta-Blockers in Heart Failure

Beta-blockers are a class of drugs that work on receptors in the heart to slow heart rate and reduce the work the heart muscle must do with each squeeze. In large randomized trials, certain types of beta-blockers have been shown to reduce the risk of death in adult patients with cardiomyopathies by around 30%.[27-31] Beta-blockers shown to prolong life in these heart failure patients include carvedilol, metoprolol succinate, and bisoprolol.

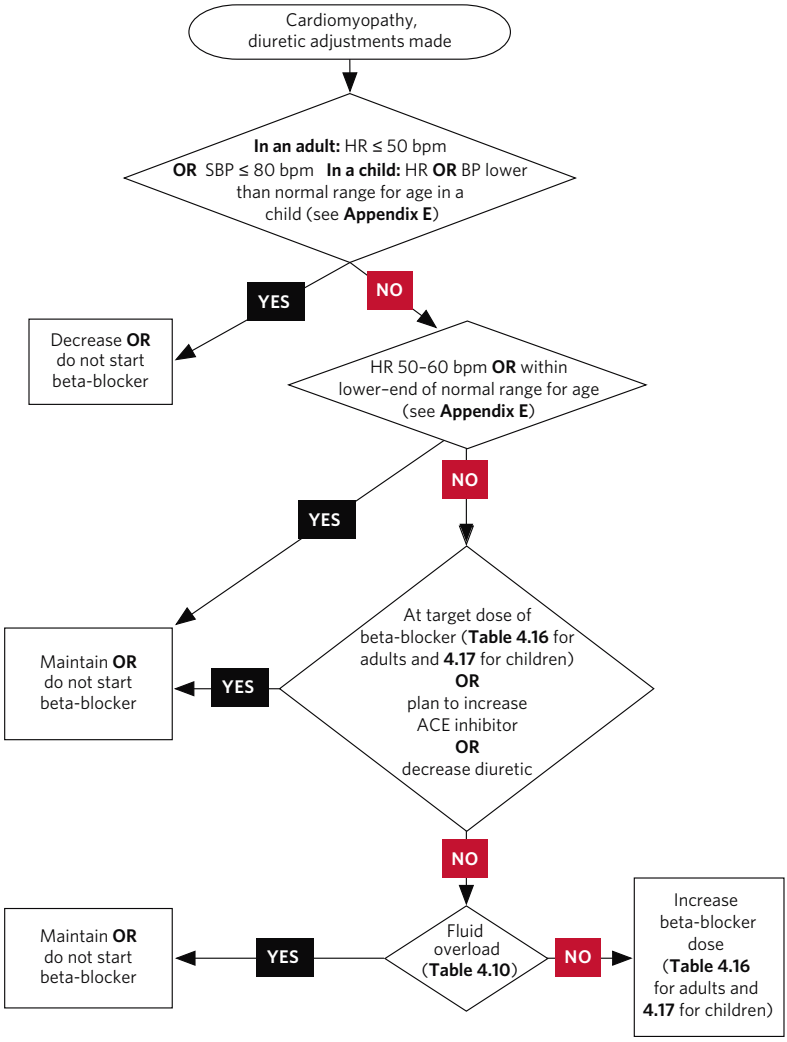
Atenolol is the beta-blocker most often available in low-resource settings. This drug is very inexpensive and is mentioned specifically in the 2008 WHO model drug formulary (though as a treatment for angina, not for heart failure).[32] Unfortunately, there is no prospective, randomized data to support its use in patients with cardiomyopathies.[33]

In comparing the price of heart failure beta-blockers (all generic at this point), we have found the price of carvedilol to be the lowest through our usual distributors (see **APPENDIX B**). We have judged that the benefits of carvedilol (or another heart failure beta-blocker) are probably significant enough to justify its addition to the national formulary. Atenolol can be used if no heart failure beta-blocker is available.

Beta-blockers should be used with caution in patients with cardiomyopathies. In the short-term, beta-blockers can actually cause fluid overload even though they stabilize volume status over the long term. Beta-blockers should be started only when a patient is euvolemic and should not be started or increased at the same time that diuretics are reduced. Likewise, they should not be started or increased in the setting of low heart rate (≤ 55 beats per minute or lower than normal range for age in a child (see **APPENDIX E**), or very low systolic blood pressure (≤ 80 mmHg or lower than normal range for age in a child (see **APPENDIX E**), or in the case of a patient with severe asthma. Side effects of beta-blockers include aggravation of asthma symptoms, bradycardia, and hypotension.

PROTOCOL 4.5 provides guidance on beta-blocker titration in cardiomyopathy.

PROTOCOL 4.5 Beta-Blocker Titration in Cardiomyopathy



4.8.3.2 ACE Inhibitors

ACE inhibitors are a class of medication that work on the kidneys, blood vessels, and heart muscle to decrease blood pressure, decrease fluid retention and prevent harmful remodeling of heart muscle. They have been shown to reduce mortality by around 30% in adult patients with cardiomyopathies.[34-37] Although all ACE inhibitors are acceptable for use, lisinopril has the lowest cost and requires only once per day dosing (see **TABLE 4.16** for adults and **TABLE 4.17** for children and **APPENDIX B**).

In the presence of renal failure, ACE inhibitors can cause a dangerous rise in potassium. Patients with an elevated creatinine (≥ 200 µmol/L (≥ 2.3 mg/dL) in adults) or ≥ twice the normal value for age in children (see **APPENDIX E**) should not be placed on an ACE inhibitor unless potassium levels can be easily monitored at regular intervals. ACE inhibitors also cause birth defects and should never be used in women who are pregnant. Like beta-blockers, ACE inhibitors can cause low blood pressure and should be avoided in patients with a low systolic blood pressure (less than 90 mmHg in adults or outside of the normal range for age in children).

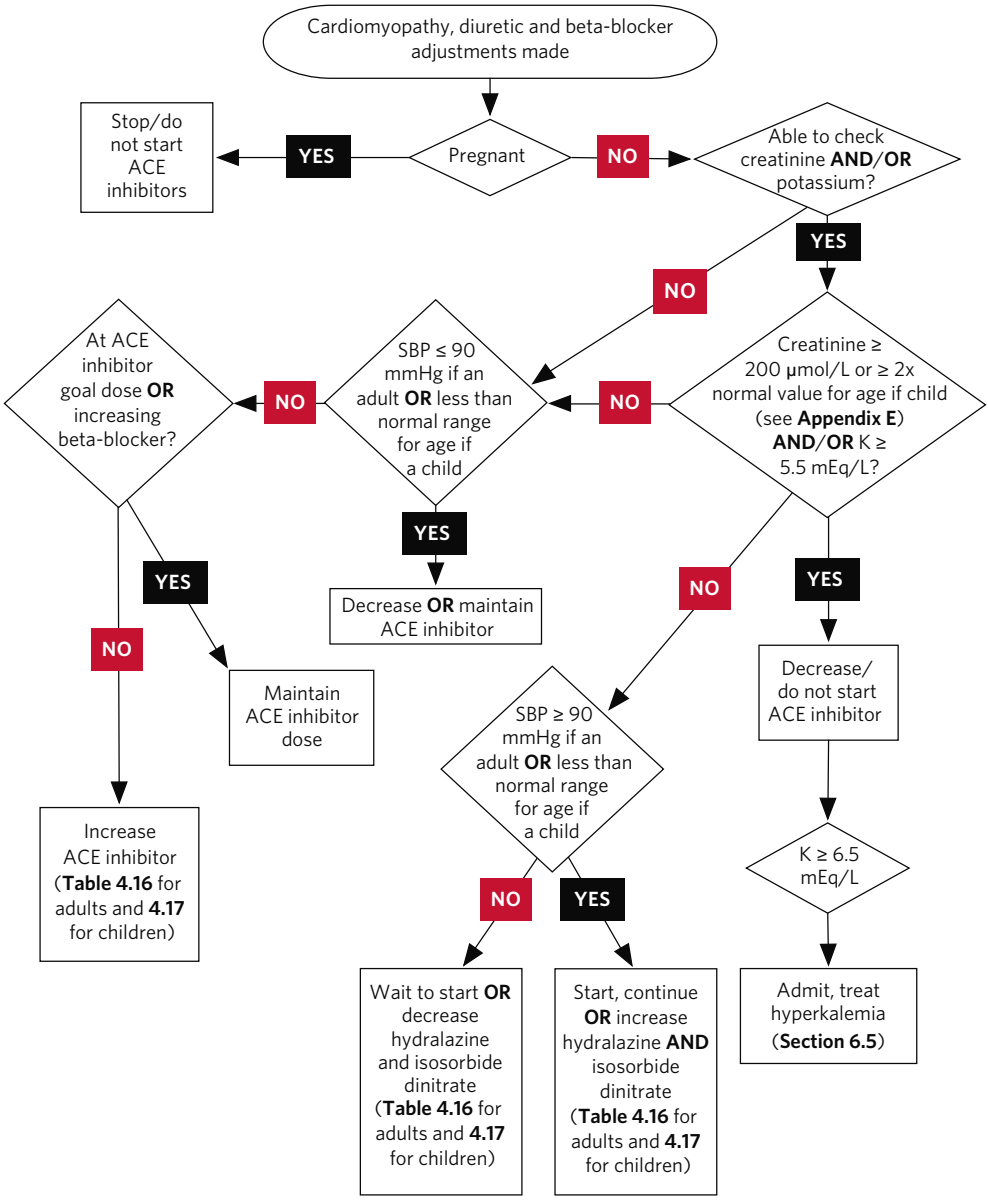
PROTOCOL 4.6 provides guidance on titration of ACE inhibitors in cardiomyopathy.

4.8.3.3 Hydralazine and Isosorbide Dinitrate

Hydralazine is an arteriolar dilator, and isosorbide dinitrate is a venodilator. When used in combination, they have been shown in large clinical trials to decrease the risk of death in adult patients by about 30% and improve heart failure symptoms, although they are not as effective as ACE inhibitors.[38-40] Hydralazine and isosorbide have the advantage of affecting heart rate and renal function less than beta-blockers and ACE inhibitors. They are safe to use when bradycardia limits beta-blocker use and/or when renal function prohibits ACE inhibitor use. However, this drug combination is expensive, it requires inconvenient three-times-a-day dosing, and it can result in significant side effects such as headaches, which may limit patient adherence. For these reasons, we generally reserve the use of hydralazine/isosorbide dinitrate for patients with contraindications to beta-blockers and/or ACE inhibitors. Caution should be exercised when starting or increasing these medications in patients with low systolic blood pressure (less than 90 mmHg in adults or lower than normal range for age in children). As mentioned above, this drug combination is not well studied in the pediatric population and for this reason should be used with caution in children with cardiomyopathies.

PROTOCOL 4.6 also provides guidance on titration of hydralazine/isosorbide in cardiomyopathy.

PROTOCOL 4.6 ACE-Inhibitor Titration and Use of Hydralazine/Isosorbide in Cardiomyopathy



4.8.3.4 Spironolactone

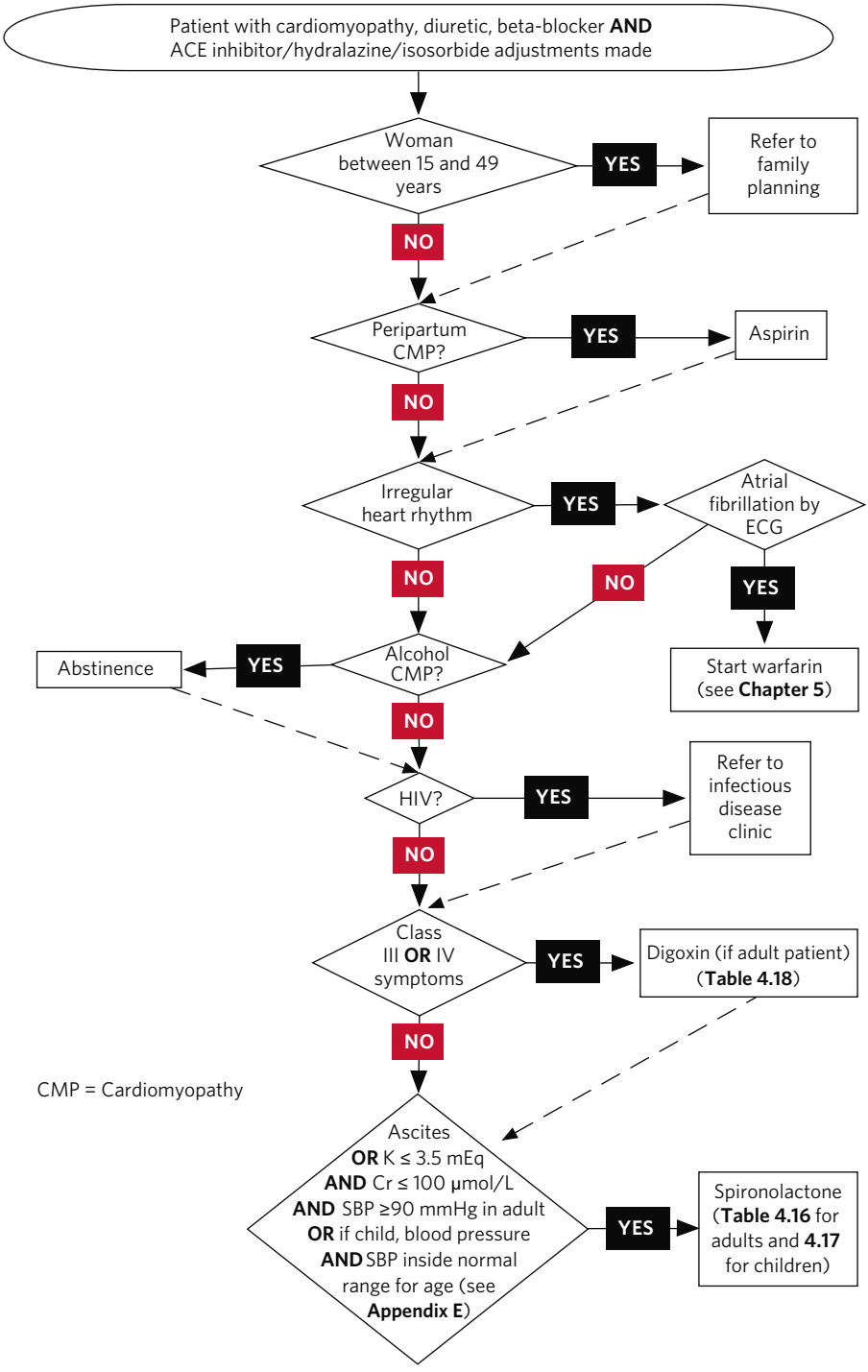
As described in **SECTION 4.7.3**, spironolactone can be a useful adjunctive diuretic in cases of ascites. Like ACE inhibitors, spironolactone can cause a rise in serum potassium, especially in the presence of renal failure. It should only be used if the creatinine is normal (< 100 µmol/L or < 1.1 mg/dL in an adult or normal range for age in a child). On the other hand, it may be a useful adjunct in patients with chronically low potassium

secondary to the use of furosemide or hydrochlorothiazide. Spironolactone has also been shown to reduce mortality by 30% in adult patients with left ventricular dysfunction who are already taking ACE inhibitors.[41,42] **TABLE 4.16** and **TABLE 4.17** show recommended dosing.

4.8.4 Other Medications in Cardiomyopathy Management

Depending on the suspected etiology of cardiomyopathy and/or disease cofactors, certain adjunctive medications may be indicated. **PROTOCOL 4.7** provides guidance on using these other medications.

PROTOCOL 4.7 Other Medication Needs for Cardiomyopathy Patients



4.8.4.1 Digoxin (Digitalis)

Digoxin is one of the oldest and most widely available medications for the treatment of heart failure. It acts to increase the pumping function of the heart and can also slow down the heart rate if atrial fibrillation is present. Using digoxin can improve symptoms in patients with heart failure. However, in a large clinical trial, it did not decrease mortality.[43] Also, at high doses, toxicity can develop, which will result in visual changes, dizziness, or nausea, and can lead to dangerous arrhythmias. Toxicity will occur if digoxin is used in patients with renal failure, so this drug should be used only if the creatinine level is ≤ 100 µmol/L.[44] We reserve digoxin for patients with class III or IV heart failure symptoms. For patients with known atrial fibrillation, digoxin may be started to help with rate control. We do not discuss digoxin use in children. In Rwanda, we currently do not have dose formulations small enough for children available at district level and pills cannot be crushed.

TABLE 4.18 lists recommended digoxin dosing.

TABLE 4.18 Adult Digoxin Dosing

Starting dose	Maximum dose
0.125 mg/day	0.25 mg/day

4.8.4.2 Aspirin

When the heart does not pump well, blood tends to sit still inside the heart and may form clots, which can then embolize (travel to other parts of the body), causing stroke or other catastrophic events. Peripartum cardiomyopathy patients are at particularly high risk for this, as well as for venous clots. They should be started on 100 mg of aspirin daily to help thin the blood. Patients with evidence of an active clot will need stronger anticoagulation (see **CHAPTER 5**).

4.8.4.3 Birth Control

All female patients of childbearing age should be counseled on the dangers of pregnancy in the setting of a cardiomyopathy. The physiologic changes during pregnancy, including increased fluid retention and smaller lung volumes, mean that heart failure symptoms almost always worsen. In the case of peripartum cardiomyopathy, subsequent pregnancies can be lethal. All women should be advised to use birth control. Clinicians should work with family-planning staff to coordinate efforts and ensure that women at risk receive timely and appropriate birth control. See **CHAPTER 3** for further discussion of birth control for women with heart failure.

4.8.4.4 Antiretrovirals (ARVs)

Untreated HIV infection frequently leads to cardiomyopathy.[45,46] Predictors of HIV cardiomyopathy are opportunistic infection and length of time since HIV diagnosis. All patients with a newly diagnosed cardiomyopathy should be tested for HIV. If a patient is HIV positive, regardless of the CD4 count, the patient should be treated with ARVs according to local protocols.

4.9 Hypertensive Heart Disease Etiology and Diagnosis

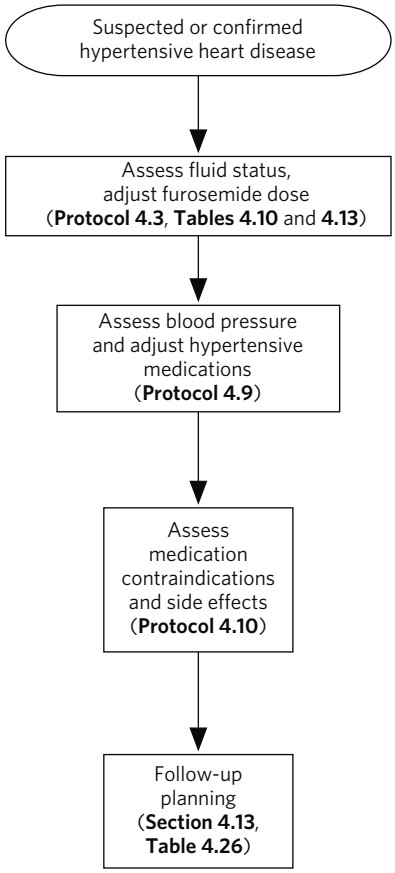
Hypertension creates stress on the heart, which has to work harder to push blood into a high-pressure system. In response to this workload, the heart muscle gets thicker (hypertrophy) and stiffer. This change defines hypertensive heart disease. The thick, stiffened walls are not able to relax as normal heart walls do, making it difficult for the heart to fill with blood. The common echocardiographic finding is a thickened heart wall. Usually, patients with hypertensive heart disease will have normal or only mildly depressed systolic function (ejection fraction between 40% and 50%) with left ventricular hypertrophy, but some can progress to very low ejection fractions.

Hypertensive heart disease results from long-standing, severe hypertension. The hypertension itself may be idiopathic (which is most common) or secondary to another problem (such as a kidney or endocrine disorder). In young patients (≤ 40 years old) a secondary cause should be suspected—severe renal failure in particular—although hypertension itself can cause renal failure. As with all types of heart failure, new patients should be screened for renal failure with a creatinine.

Hypertensive heart disease is an extremely rare cause of heart failure in children. This chapter refers only to adult diagnostic criteria and medication dosing.

Treatment of hypertensive heart disease includes control of volume status and blood pressure. **PROTOCOL 4.8** provides an outline for hypertensive heart disease management.

PROTOCOL 4.8 Management of Suspected or Confirmed Hypertensive Heart Disease



4.9.1 Vital Sign Assessment

As with all types of heart failure, management of hypertensive heart disease begins with an assessment of the patient's vital signs and overall condition. Decompensation in patients with hypertensive heart disease is often caused by uncontrolled, very high blood pressure (SBP ≥ 180 mmHg), resulting in stiffening of the heart. As described in **CHAPTER 8**, the approach in this situation is to lower the blood pressure gradually—by approximately 25% in the first 2 to 4 hours.

4.9.2 Fluid Status Assessment

The next step in the management of hypertensive heart disease is the evaluation of fluid status and titration of diuretics. See **SECTION 4.7**, **TABLE 4.10**, and **PROTOCOL 4.3**. All patients should have a creatinine checked regularly to assess renal function. Renal failure is common in patients who have had long-standing, severe hypertension.

4.9.3 Titration of Antihypertensives

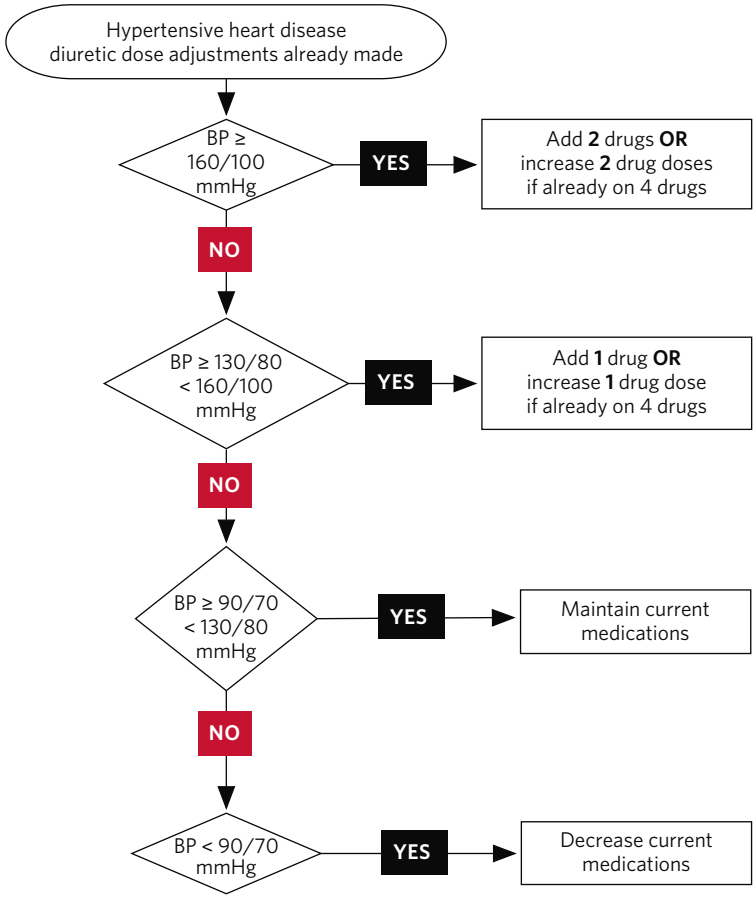
In order to prevent worsening heart failure symptoms, antihypertensives should be titrated to achieve a blood pressure goal of ≤ 130/80 mmHg. See **PROTOCOL 4.9**, **PROTOCOL 4.10**, and **TABLE 4.19**. In general, an ACE inhibitor is first-line therapy for these patients unless contraindications exist (creatinine ≥ 200 µmol/L or pregnant/breastfeeding). If the blood pressure is not controlled or if ACE inhibitors are not appropriate for the patient, a thiazide diuretic may be started. After that, amlodipine, a peripherally acting calcium-channel blocker, should be used. Beta-blockers and hydralazine are third- and fourth-line medications, respectively.

TABLE 4.19 Antihypertensives for Hypertensive Heart Disease

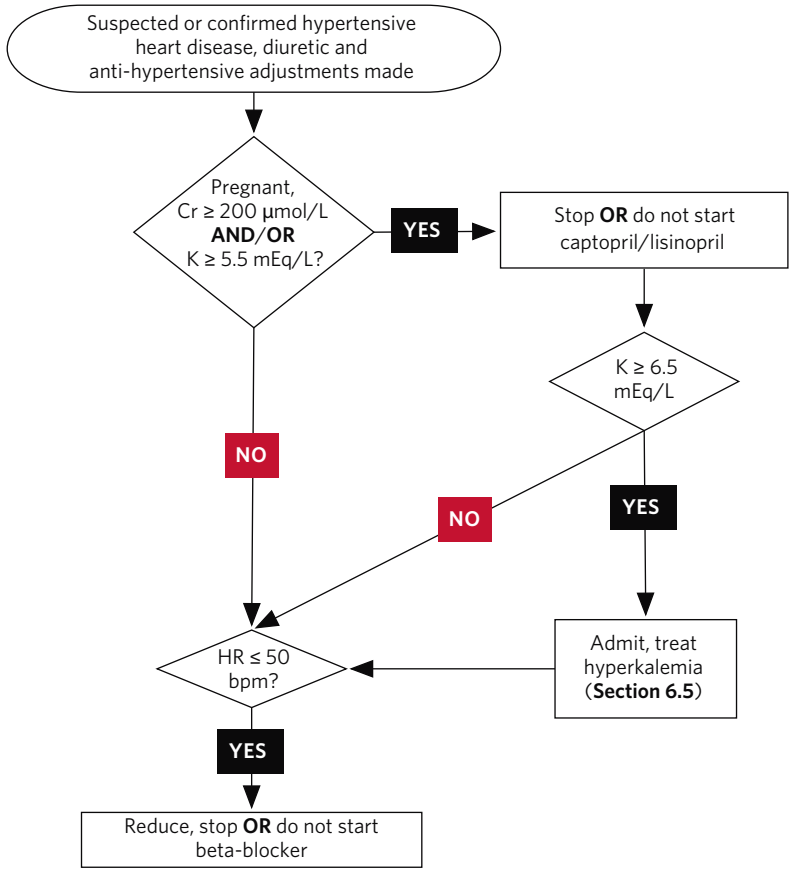
First-line drug (ACE inhibitor)	Initial dose	Dose increase	Maximum dose	Side effects/cautions
Lisinopril	5 mg 1x/day	5 mg 1x/day	20 mg 1x/day	1. Birth defects (contraindicated in pregnancy) 2. Chronic non-productive cough 3. High potassium 4. Do not use if creatinine ≥ 200 µmol/L
Captopril	12.5 mg 3x/day	12.5 mg 3x/day	50 mg 3x/day	
Second-line drug (thiazide)	**Initial dose**	**Dose increase**	**Maximum dose**	**Side effects/cautions**
Hydrochlorothiazide	12.5 mg 1x/day	12.5 mg 1x/day	25 mg 1x/day	Lowers potassium
Third-line drug (CCB)*	**Initial dose**	**Dose increase**	**Maximum dose**	**Side effects/cautions**
Amlodipine	5 mg 1x/day	5 mg 1x/day	10 mg 1x/day	Lower-extremity swelling
Nifedipine (sustained release)	20 mg 2x/day	20 mg 2x/day	40 mg 2x/day	
Fourth-line drug (beta-blocker)	**Initial dose**	**Dose increase**	**Maximum dose**	**Side effects/cautions**
Atenolol	25 mg 1x/day	25 mg 1x/day	50 mg 1x/day	1. Bradycardia 2. Renally excreted 3. Decrease dose for renal failure
Fifth-line drug (vasodilator)	**Initial dose**	**Dose increase**	**Maximum dose**	**Side effects/cautions**
Hydralazine	25 mg 3x/day	25 mg 3x/day	50 mg 3x/day	1. Headache 2. Tachycardia 3. Lower-extremity swelling

* CCB = Calcium-channel blocker

PROTOCOL 4.9 Antihypertensive Management in Hypertensive Heart Disease



PROTOCOL 4.10 Assessment of Medication Contraindications and Side Effects in Management of Hypertensive Heart Disease

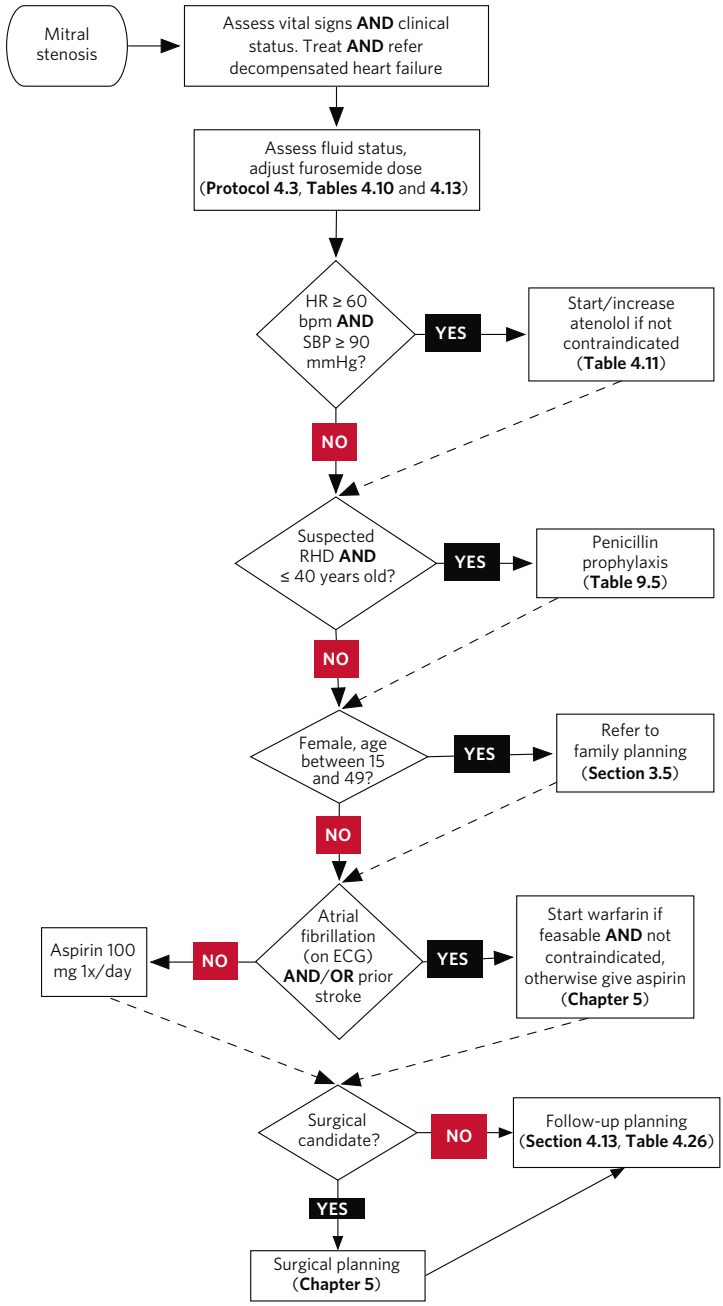


4.10 Mitral Stenosis

Mitral stenosis is a special type of valvular disease with a treatment pathway that differs from that of other valvular diseases. Untreated, mitral stenosis leads to left atrial enlargement, pulmonary hypertension, and right ventricular failure. The stretched left atrium is particularly prone to developing electrical abnormalities, leading to atrial arrhythmias. Mitral stenosis almost always results from untreated rheumatic heart disease. Mitral stenosis is most common in people between 30 and 50 years old. It may sometimes occur in younger or older patients. However, it is very rarely seen in young children. Therefore, all vital sign parameters and dosing information in this chapter refer only to adults. Often women will develop symptoms in the middle of their pregnancies. Patients with mitral stenosis are also at high risk of atrial fibrillation, which can cause rapid decompensation. Basic echocardiography can easily identify a stenotic mitral valve, which has a highly recognizable pattern in the parasternal long view.

Mitral stenosis requires a different treatment strategy than other forms of valvular heart disease. The goals of medical therapy are to manage fluid status and to minimize the negative effects of mitral stenosis on cardiac output by controlling heart rate. **PROTOCOL 4.11** outlines the steps in the medical management of mitral stenosis.

PROTOCOL 4.11 Management of Mitral Stenosis



4.10.1 Vital Sign Assessment

As with all types of heart failure, mitral stenosis management begins with an assessment of the patient's vital signs and overall condition. Patients with mitral stenosis are particularly sensitive to tachycardia and will not be able to tolerate fast heart rates well.

4.10.2 Fluid Status Assessment

The next step in the management of mitral stenosis is evaluation of fluid status and titration of diuretics. The principles here are the same as in other forms of heart failure. See **SECTION 4.7**, **TABLE 4.10**, and **PROTOCOL 4.3**.

4.10.3 Control of Heart Rate

When the mitral valve doesn't open well, less blood can move from the left atrium to the left ventricle during diastole. This, in turn, decreases the amount of blood the heart can pump out with each beat. When the heart rate increases, diastole (the heart's filling time) becomes even shorter, exacerbating the problem. For this reason, it is very important to control heart rate, preferably keeping it below 60 beats per minute, if permitted by blood pressure. We recommend the use of beta-blockers for this purpose. All beta-blockers work equally well in decreasing heart rate. We suggest using atenolol because of its once-a-day dosing, low cost, and wide availability. However, any agent that slows the heart rate may be used.

Atrial fibrillation, which is common in mitral stenosis, often causes very fast heart rates. Patients with mitral stenosis are unable to tolerate these fast heart rates and will become very sick or even die if the arrhythmia is not controlled. Beta-blockers (atenolol) are the first-line therapy. However, if good heart rate control in a patient with atrial fibrillation and mitral stenosis cannot be obtained with beta-blockers alone, or if blood pressure limits the use of beta-blockers, clinicians may prescribe digoxin if the patient has a creatinine < 100 µmol/L. (See **TABLE 4.20** and **SECTION 4.16.1**.)

TABLE 4.20 Medications to Reduce Heart Rate in Mitral Stenosis

Medication	Initial dose	Dose increase	Maximum dose
Beta-blocker *(for use in sinus tachycardia or atrial fibrillation)*			
Atenolol	12.5 mg 1x/day	12.5 mg 1x/day	50 mg 1x/day
Digoxin *(only if tachycardia and atrial fibrillation and creatinine < 100 µmol/L)*			
Digoxin	0.125 mg 1x/day	0.125 mg 1x/day	0.25 mg 1x/day

4.10.4 Other Medications in Management of Mitral Stenosis

4.10.4.1 Penicillin

Rheumatic heart disease causes almost all cases of mitral stenosis in resource-poor settings. As described in **CHAPTER 9**, prevention of further attacks of rheumatic fever through penicillin prophylaxis helps slow the progression of rheumatic valvular disease. Although rheumatic fever generally affects school-aged children, we recommend penicillin prophylaxis for all patients with rheumatic valvular disease under the age of 40. See **SECTION 9.2** for specific guidelines on penicillin dosing and administration.

4.10.4.2 Birth Control

As with cardiomyopathy, mitral stenosis presents an increased danger to women during pregnancy, and for some pregnant women, it can be lethal. Not uncommonly, women will first become symptomatic from mitral stenosis in the 2nd or 3rd trimester. Women between the ages of 15 and 49 years with mitral stenosis should receive family planning counseling and be offered an acceptable form of birth control (see **CHAPTER 3**).

4.10.4.3 Anticoagulation (Aspirin and Warfarin)

All patients with mitral stenosis are at very high risk of developing clots in their dilated, stagnant left atrium. These clots have a high likelihood of embolizing to the brain and causing stroke. Atrial fibrillation further increases the chances of clot formation and stroke, and patients with mitral stenosis are at very high risk of atrial fibrillation. For this reason, all patients with mitral stenosis should receive some type of blood-thinning agent. If a patient has clear documentation of atrial fibrillation, stronger anticoagulation in the form of warfarin should be initiated if there are no contraindications to warfarin therapy (see **CHAPTER 5**). If there is no ECG-documented atrial fibrillation, then aspirin should be prescribed.

4.11 Other Valvular and Congenital Heart Disease

This category encompasses the most diverse group of heart failure patients. Most children with heart failure will fall into this diagnostic category. Unlike the previously described heart failure categories, these diseases often require advanced echocardiography skills for differentiation. Fortunately, they also share a common initial approach in management (see **PROTOCOL 4.12**). We therefore argue that at the district health center level, these patients can be safely managed as an undifferentiated group until a more advanced echocardiographer can make a definitive diagnosis.

Valvular disease (here excluding mitral stenosis) means that some insult to the heart valve (such as rheumatic fever) has caused it to either not open (stenosis) or not close (regurgitation) properly. Afflicted patients

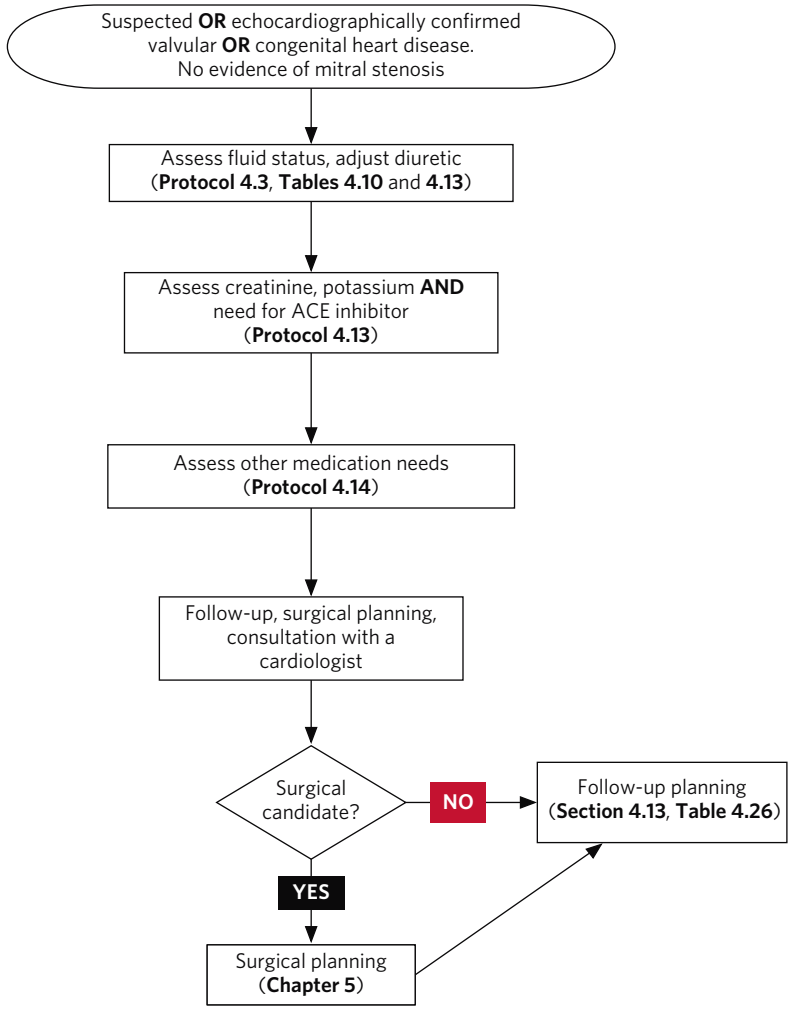
will most often have dramatic murmurs. These lesions can eventually cause dilation of the associated heart chambers, which in turn can lead to a cardiomyopathy. Valvular diseases are usually secondary to rheumatic heart disease, in which untreated streptococcal infection leads to an autoimmune response that results in valve destruction.

Congenital heart disease means that the heart was malformed at birth. Congenital disease comes in many varieties and often causes murmurs or cyanosis. It can be caused by a variety of genetic factors and/or teratogens (toxins during pregnancy).

This collection of heart failure diagnoses is both larger and more diverse than the previous categories. However, these diseases share a common pathway in their initial medical management. Moreover, differentiation among these diseases often requires a higher level of echocardiographic and other diagnostic skills than are reasonably attainable by generalist physicians or advanced nurses in resource-poor settings.

The common goals in the medical management of this group of heart failure patients include management of fluid status and reduction of the heart's workload. Definitive treatment for most congenital and valvular disease will be surgical, and thus all patients suffering this class of heart failure should be evaluated by a cardiologist, internist, or pediatrician experienced in echocardiography within 6 weeks to 6 months of preliminary diagnosis.

PROTOCOL 4.12 Valvular or Congenital Heart Disease Management (Excluding Mitral Stenosis)



4.11.1 Vital Sign Assessment

As with all types of heart failure, management of this collection of heart failure types begins with an assessment of the patient's vital signs, weight, and overall condition.

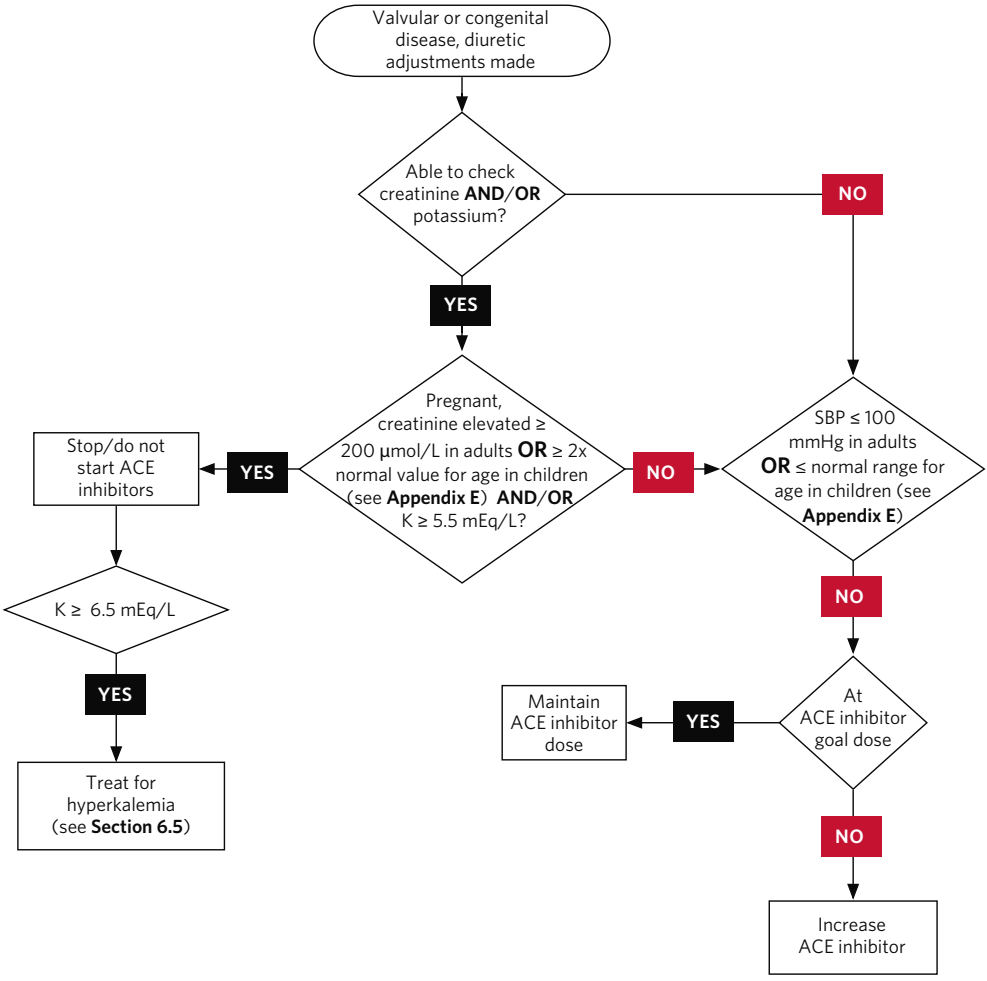
4.11.2 Fluid Status Assessment

The next step in management is evaluation of fluid status and titration of diuretics. See **SECTION 4.7**, **TABLE 4.10**, and **PROTOCOL 4.3**.

4.11.3 Titration of ACE Inhibitors

In patients with valvular heart disease (excluding mitral stenosis), it is important to reduce the workload of the heart by lowering the blood pressure. The heart was designed to pump blood in only one direction. However, regurgitant lesions cause blood to flow backwards with each cardiac cycle. This results in wasted work by the heart. Lowering blood pressure helps reduce some of this extra effort by reducing the amount of force the heart needs to pump blood in the forward direction. ACE inhibitors are the preferred anti-hypertensive agent (see **TABLE 4.20**). However, if a patient has renal failure or hyperkalemia, or is pregnant, furosemide alone should be used. A cardiologist or doctor may choose to add another medication (such as isosorbide dinitrate) to reduce cardiac workload. The goal systolic blood pressure for patients with valvular heart disease is 100–120 mmHg in adults, and within normal range for age in children (see **APPENDIX E**). **PROTOCOL 4.13** outlines an approach to initiating and titrating ACE inhibitors.

PROTOCOL 4.13 ACE-Inhibitor Titration for Valvular or Congenital Heart Disease Management (Excluding Mitral Stenosis)



4.11.4 Other Medications

4.11.4.1 Spironolactone

Patients with disease of any of the valves on the right side of the heart can develop symptoms of right heart failure, including massive ascites. In these patients, spironolactone can be a useful adjunct to furosemide (see **TABLE 4.21** and **TABLE 4.22**). Spironolactone should only be used in patients with normal renal function (Cr ≥ 100 µmol/L or ≥ 2.3 mg/dL in adults or ≥ normal range for age if a child (see **TABLE 6.4**)), because of the risk of hyperkalemia. Concurrent use with an ACE inhibitor can also cause dangerous hyperkalemia, and these patients should have potassium monitored every six months.

4.11.4.2 Penicillin

As described in **CHAPTER 9**, penicillin prophylaxis helps slow the progression of rheumatic valvular disease by preventing further attacks of rheumatic fever. Although rheumatic fever generally affects school-aged children, we recommend penicillin prophylaxis for all patients with rheumatic valvular disease under the age of 40. See **SECTION 9.2** for specific guidelines on penicillin dosing and administration.

TABLE 4.21 Medications for Valvular/Congenital Heart Disease

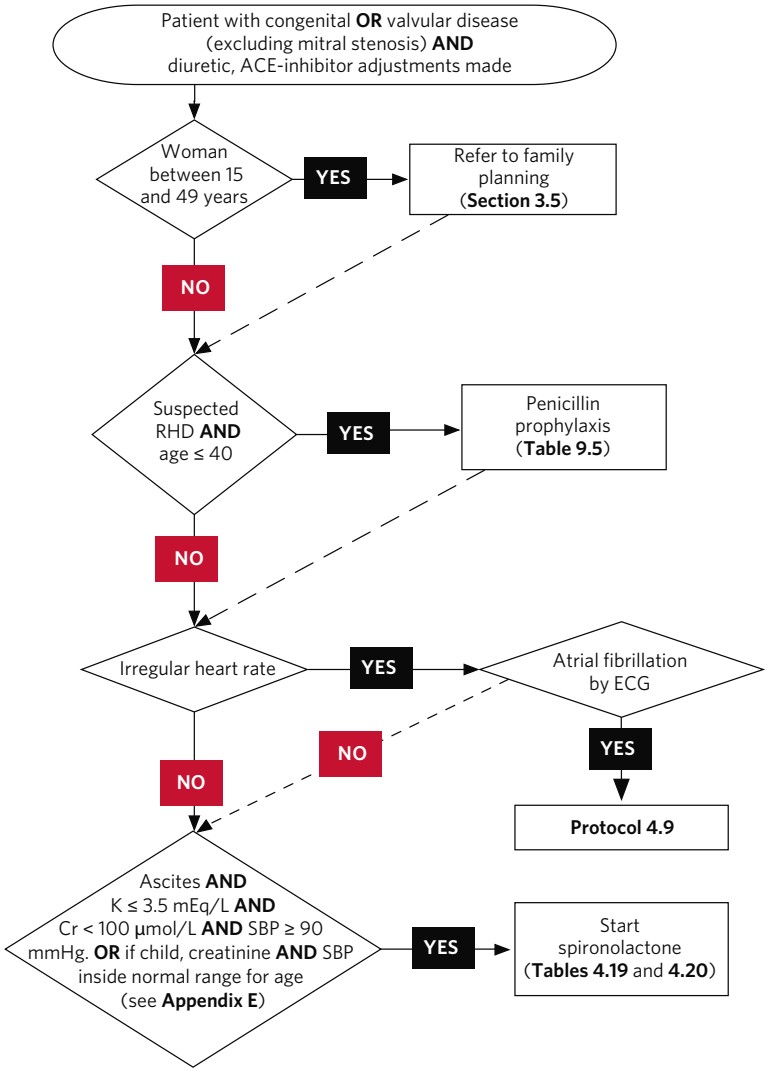
	Initial dose	Dose adjustment	Maximum dose
ACE inhibitor			
Lisinopril	5 mg 1x/day	5 mg 1x/day	20 mg 1x/day
Captopril	12.5 mg 3x/day	12.5 mg 3x/day	50 mg 3x/day
Potassium-sparing diuretic			
Spironolactone	12.5–25 mg 1x/day	12.5 mg 1x/day	25 mg 1x/day

TABLE 4.22 Medications for Valvular/Congenital Heart Disease in Children (≤ 40 kg)

ACE Inhibitors					
Starting doses	**< 10 kg**	**10 kg**	**15 kg**	**20 kg**	**30 kg**
Lisinopril 10 mg tablet	See mg/kg dosing*	See mg/kg dosing*	See mg/kg dosing*	See mg/kg dosing*	2.5 mg ½ tab 1x/day
Initial dose: 0.07 mg/kg/day as one daily dose. **Maximum dose:** 0.6 mg/kg/day or 20 mg as one daily dose. **Notes:** Do not use in children with a creatinine ≥ 2x the normal value for age (see **APPENDIX E** for normal ranges). Not safe for use in pregnancy.					
Captopril 25 mg tablet	See mg/kg dosing*	6.25 mg ¼ tab 2x/day	6.25 mg ¼ tab 2x/day	6.25–12.5 mg ¼–½ tab 2x/day	12.5 mg ½ tab 2x/day
Initial dose: 0.3–0.5 mg/kg/dose 2–3x/day. **Maximum dose:** 2 mg/kg/dose 2–3x/day. **Notes:** Do not use in children with a creatinine ≥ 2x the normal value for age (see **APPENDIX E** for normal ranges). Not safe for use in pregnancy.					
Potassium-Sparing Diuretic					
Starting doses	**< 10 kg**	**10 kg**	**15 kg**	**20 kg**	**30 kg**
Spironolactone 25 mg tablet	See mg/kg dosing*	12.5 mg ½ tab	12.5 mg ½ tab	25 mg 1 tab	25 mg 1 tab
Initial dose: 1 mg/kg as one daily dose. **Maximum dose:** 3 mg/kg/dose or 50 mg as one daily dose. **Notes:** Do not use in children with a creatinine > the normal value for age (see **APPENDIX E** for normal ranges). Consider cutting the dose by half if patient is also on furosemide.					

* Note that dosing medications for small children may require crushing pills and diluting. This should only be done under the supervision of an experienced clinician.

PROTOCOL 4.14 Other Medications for Valvular or Congenital Heart Disease Management (Excluding Mitral Stenosis)



4.11.5 Cardiac Surgery Evaluation

Definitive treatment for most valvular and congenital heart disease will be surgical, and thus all patients in this class of heart failure should be evaluated by a cardiologist or experienced internist within 6 weeks to 6 months of intake. Although advanced echocardiography is not needed to guide medical management of the patient, it is necessary to evaluate the specific type of surgery that is needed. Even patients who appear well-compensated may be candidates for surgical intervention before they begin to decompensate. At times, these are the best surgical candidates. See **CHAPTER 5** for more detail on cardiac surgery evaluation.

4.12 Isolated Right-Sided Heart Failure Etiology and Diagnosis

The terms right- and left-sided heart failure are often used to describe a patient's presenting symptoms. Most etiologies of heart failure can result from left-sided heart failure symptoms (such as shortness of breath and orthopnea), and patients with signs of left-sided heart failure will often have signs of right-sided heart failure. However, patients with isolated symptoms of right-sided heart failure (ascites, hepatomegaly, elevated jugular venous pressure [JVP], lower extremity edema) fall into a distinct category of diagnoses. (See **TABLE 4.23**.)

In resource-poor settings such as Rwanda, isolated right-sided heart failure most often results from chronic diseases of the lungs that have led to pulmonary hypertension or from diseases of the pericardium (see **TABLE 4.24**). Tuberculosis is by far the most common reason for constrictive pericardial disease in most of rural sub-Saharan Africa, and as such represents a treatable cause of heart failure.

TABLE 4.23 Clinical Presentation of Right-Sided Heart Failure

Symptoms	Physical exam findings
Weight gain	Elevated JVP
Abdominal swelling	Cardiac murmur (depending on cause)
Lower-extremity edema	Enlarged or pulsatile liver
Decreased appetite	Ascites
Right-upper-quadrant pain	Lower-extremity pitting edema

Our protocols ask practitioners to first exclude left-sided heart failure (such as cardiomyopathy, hypertensive heart disease, mitral stenosis, and other valvular disease) before considering pure right-sided heart failure as a diagnosis. As with other categories of heart failure, the precise diagnosis is neither easy nor essential to the initial management of the patient. Our protocol for right-sided heart failure therefore focuses on excluding non-cardiac causes of the presenting symptoms of edema and ascites, and on identifying those patients most likely to benefit from empiric tuberculosis treatment.

Patients with ascites caused by liver failure or renal failure can resemble patients with right-sided heart failure. It is clinically very difficult to distinguish between these patients. Echocardiography is essential in differentiating patients with right-sided heart failure from those with other etiologies of edema and ascites. In particular, echocardiography can be helpful as a way to exclude heart failure as a cause of ascites. If the patient has no murmurs and has an entirely normal-appearing basic echocardiogram (including a normal inferior vena cava and right

ventricle), they are unlikely to have a cardiac cause of their presenting symptoms (see **FIGURE 4.5** and **FIGURE 4.8**).

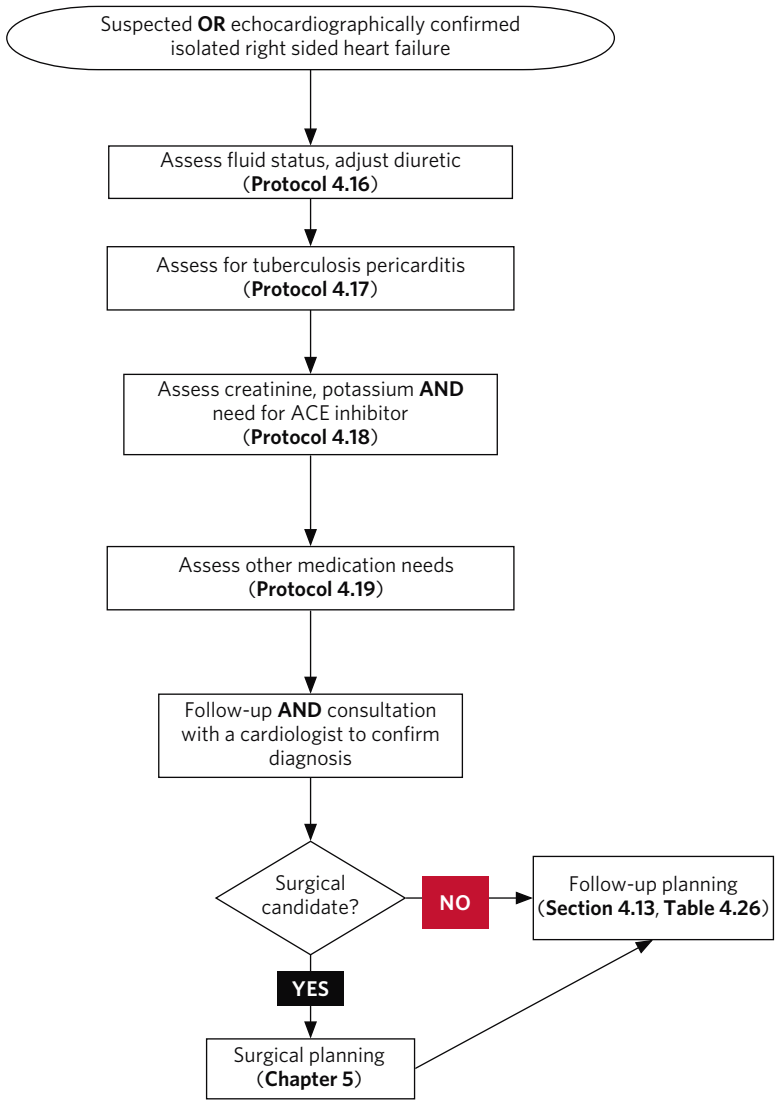
TABLE 4.24 Causes of Right-Sided Heart Failure

Cause	Notes
Left-sided heart failure	Left-sided heart failure is the most common cause of right-sided heart failure. Some common causes include cardiomyopathy, mitral stenosis, and mitral regurgitation
Tricuspid valve abnormalities	Tricuspid valve stenosis, tricuspid regurgitation, congenital heart disease
Pulmonary disease	Pulmonary TB, severe COPD
Pulmonary hypertension	HIV, congenital heart disease (commonly VSD, ASD), other
Constrictive pericarditis	Most commonly caused by TB
Endomyocardial fibrosis	Cause unknown

Treatment for patients with right-sided heart failure focuses first on identifying those patients who are likely to have disease caused by active tuberculosis. Patients with other types of right ventricular failure (usually due to pulmonary hypertension or endomyocardial fibrosis) will be mainly palliative and will mirror the approach to medical management of patients with valvular and other congenital heart disease.

PROTOCOL 4.15 outlines an approach to patients with isolated right-sided heart failure.

PROTOCOL 4.15 Management of Isolated Right-Sided Heart Failure



4.12.1 Vital Sign Assessment

As with all types of heart failure, management of suspected right-sided heart failure begins with an assessment of the patient's vital signs and overall condition. Patients with an effusion and hypotension should be presumed to have tamponade and should be referred to the district hospital for emergent pericardiocentesis. Likewise, patients with massive ascites that is causing respiratory distress should also be referred to the district hospital for paracentesis.

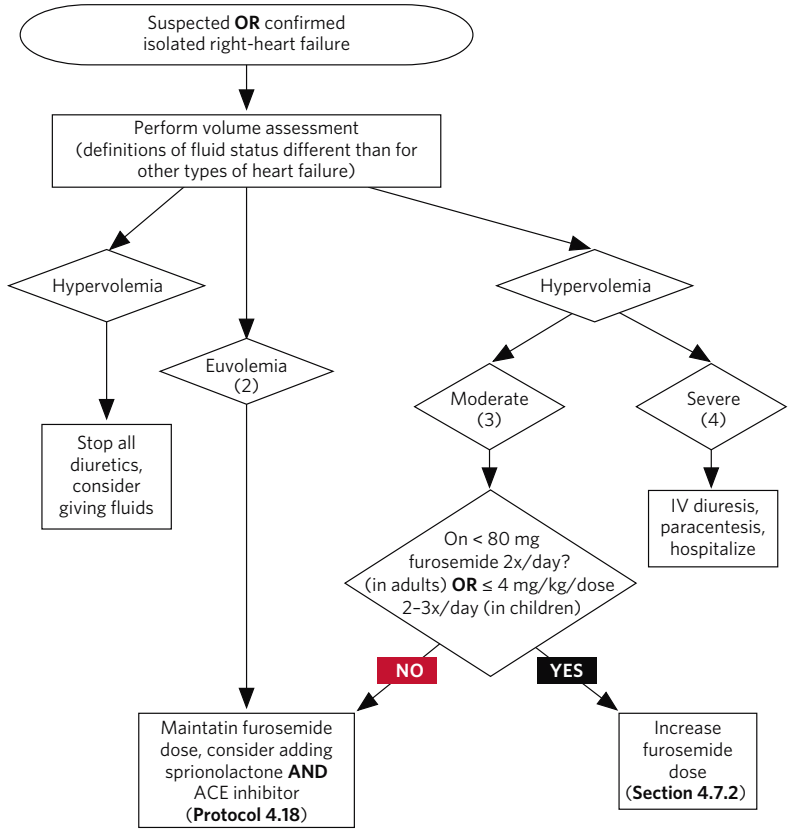
4.12.2 Fluid Status Assessment

The next step in the management of right-sided heart failure is evaluation of fluid status and titration of diuretics. These patients tend to have profound leg edema and ascites. In most cases, these patients will never reach euvolemia, and attempting to diurese them down to a dry weight will result in intravascular hypovolemia, causing renal failure, hypotension, and electrolyte imbalances. The goal should be to minimize the patient's discomfort from edema and abdominal distension.

As with other types of heart failure, furosemide is the main tool for controlling the patient's fluid status. See **SECTION 4.7.2**.

Spironolactone should be added to assist in diuresis of patients with normal renal function. In patients with severe discomfort or respiratory distress from ascites, paracentesis may be performed (see **SECTION 4.12.4**).

PROTOCOL 4.16 Fluid Status Management in Right-Sided Heart Failure



(1) Hypovolemia: Rising creatinine, low blood pressure; may still have significant edema or ascites
(2) Euvolemia: Comfortable, able to perform daily activities
(3) Hypervolemia, Moderate: Increasing ascites or edema, uncomfortable but able to do basic activities
(4) Hypervolemia, Severe: Rapid breathing, unable to walk

4.12.3 Pericardial Disease

Diseases of the pericardium fall into two main categories: (1) pericardial effusions; and (2) constrictive pericardial disease.[47] Both of these conditions can lead to heart failure by causing external compression on the heart, and by not allowing the heart to fill properly with blood.

4.12.3.1 Pericardial Effusions

Pericardial effusions are fluid collections around the heart. Small effusions (less than 1 cm in diastole) are common incidental findings in all forms of heart failure. However, large pericardial effusions without another cardiac finding are most often due to either tuberculosis (TB), cancers, or viral infections. TB is the cause of large pericardial effusions in about 90% of those infected with HIV, and in about half of those not infected with HIV.[47] On chest x-ray (CXR), the heart often appears large or globular. On an echocardiogram, the heart is surrounded by fluid (see **FIGURE 4.9**). If large enough, a pericardial effusion can cause life-threatening hypotension, which is known as cardiac tamponade. The treatment for this condition is drainage of the fluid with a needle (pericardiocentesis).

4.12.3.2 Constrictive Pericarditis

The diseases that cause a pericardial effusion can also lead to scarring and stiffening of the pericardium. This is known as constrictive pericarditis. The diagnosis of constrictive pericarditis can be difficult to make. Most patients will present with symptoms of right-sided heart failure, including ascites. Calcification around the heart on CXR is diagnostic for constrictive pericarditis, but is actually rare. Constrictive pericardial disease should be suspected in patients with signs of right-sided heart failure; a dilated, non-collapsing IVC; and no other obvious cardiac findings on echocardiography or physical examination.

Most constrictive pericardial disease in rural sub-Saharan Africa is probably due to TB. Echocardiography is helpful in the diagnosis of a pericardial effusion or constriction. However, evidence of TB infection is often subtle or nonexistent, making a definitive diagnosis unlikely in most cases. CXR demonstrates signs of pulmonary TB in only one-third of cases, and sputum smear for acid-fast bacilli is often negative.[47] Drainage or biopsy of the pericardium is difficult, expensive, and not usually available in community-based settings. Diagnosis must therefore be based on clinical presentation in the context of risk factors for TB and HIV.

4.12.3.3 Diagnosis and Treatment of Tuberculosis Pericarditis

Prompt initiation of anti-mycobacteria treatment can be lifesaving in cases of TB pericarditis. We therefore suggest that TB is the presumed diagnosis in cases of suspected constrictive pericardial disease. We

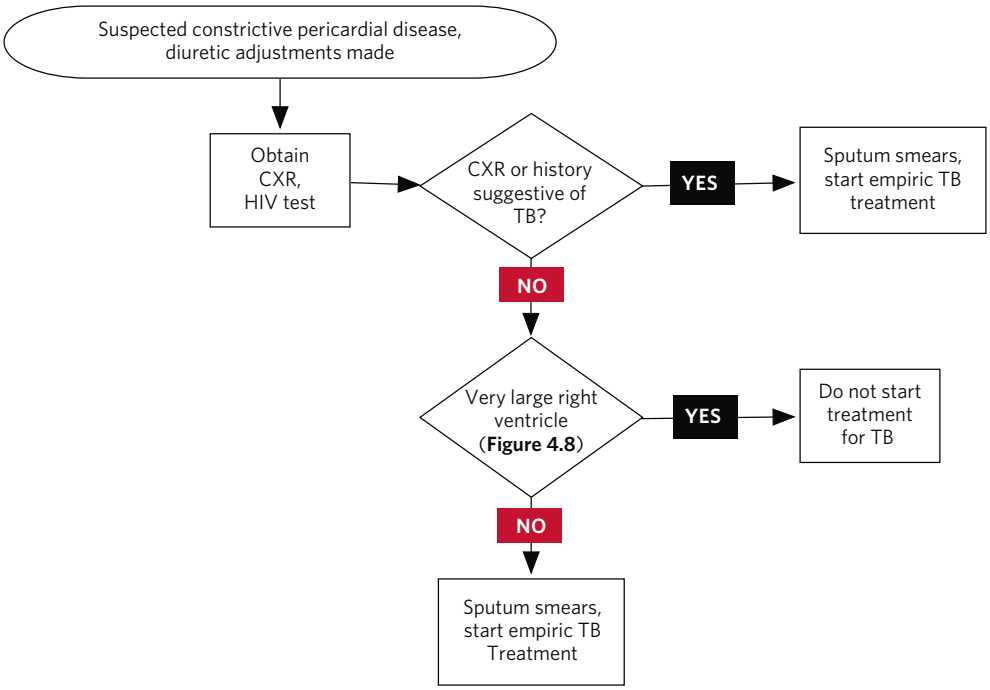
recommend starting empiric therapy in these cases. All patients started on TB treatment should have three sets of sputum collected. All patients started on treatment should also be seen by a cardiologist who will decide, in conjunction with the infectious disease clinic, whether to continue or stop the anti-tuberculosis medications.

Treatment of pericardial TB is the same as for pulmonary TB and consists of a 4-drug regimen given over 6 months: 2 months of daily isoniazid, rifampin, pyrazinamide, and ethambutol, followed by 4 months of daily isoniazid and rifampin. There is no need to prolong the course of treatment. The use of corticosteroids is controversial, but in several studies, the concomitant use of prednisone with anti-TB therapy has been shown to reduce mortality and the need for surgical interventions.[48,49] We therefore recommend prescribing a steroid taper for patients treated for suspected tuberculosis pericarditis (see **TABLE 4.25**). Because rifampin increases the metabolism of steroids, prednisolone is given at relatively high doses (around 1.5 mg/kg per day in adults).

TABLE 4.25 Treatment of Tuberculous Pericarditis

Medication	Adult Dose	Pediatric dose (< 40 kg)	Schedule
Isoniazid	5 mg/kg once daily **Maximum dose:** 300 mg daily	10–15 mg/kg once daily **Maximum dose:** 300 mg once daily	26 weeks
Rifampin	10 mg/kg once daily **Maximum dose:** 600 mg once daily	10–20 mg/kg once daily **Maximum dose:** 600 mg once daily	26 weeks
Pyrazinamide	20–25 mg/kg once daily **Maximum dose:** 2 g once daily		8 weeks
Ethambutol	15–20 mg/kg once daily **Maximum dose:** 2.5 g once daily	15–25 mg/kg once daily **Maximum dose:** 2.5 g once daily	8 weeks
Prednisone	40 mg twice per day	1 mg/kg 1x/day	4 weeks, followed by
	20 mg twice per day	0.5 mg/kg 1x/day	4 weeks, followed by
	10 mg twice per day	0.25 mg/kg 1x/day	2 weeks, followed by
	5 mg once daily	0.125 mg/kg 1x/day	1 week

PROTOCOL 4.17 Diagnosis and Treatment of Tuberculosis Pericarditis



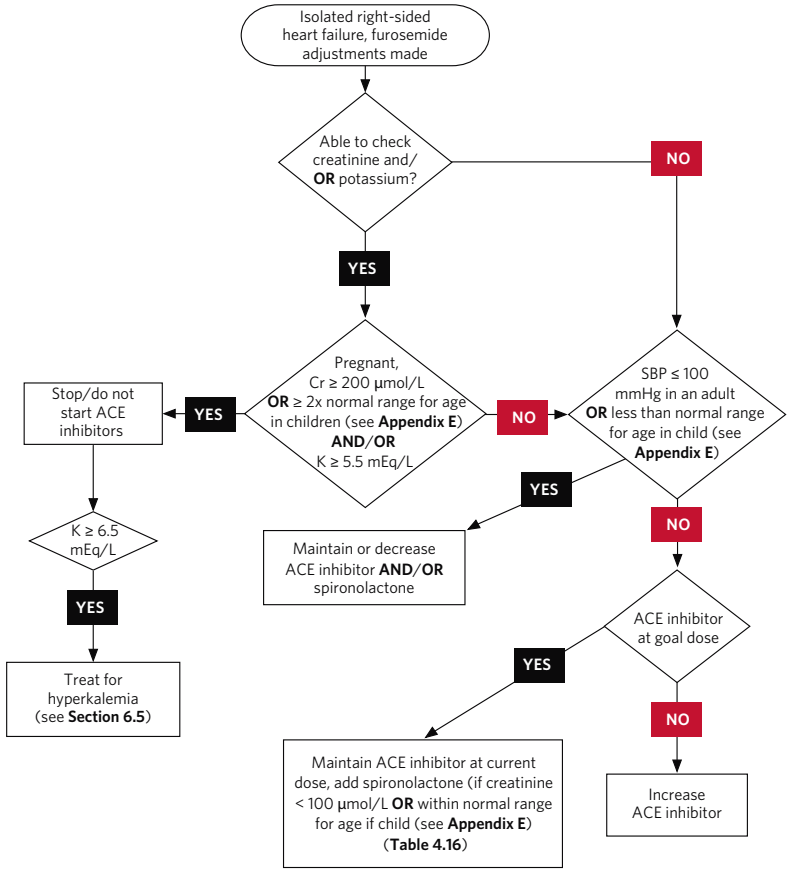
4.12.4 Titration of ACE Inhibitors and Spironolactone and Paracentesis

As described in **SECTION 4.11.4.1**, spironolactone can be used in addition to furosemide for patients with ascites. However, it should only be used in patients with normal renal function (creatinine < 100 µmol/L or < 1.1 mg/dL in adults or within normal range for age in children (see **APPENDIX E**)). When spironolactone is prescribed along with lisinopril, the patient should be monitored for hyperkalemia at least every 6 months. Spironolactone may be a useful adjunct in patients with chronically low potassium secondary to furosemide or hydrochlorothiazide use.

Patients with tense ascites may need periodic paracentesis to relieve abdominal discomfort or respiratory distress. However, without correction of the underlying cause, reaccumulation of ascitic fluid will occur. Moreover, frequent paracentesis puts patients at risk for peritoneal infections and loss of protein, which can in turn worsen the ascites.

After adding spironolactone, an ACE inhibitor may be added to the patient's regimen, if renal function and blood pressure permit. As in other types of heart failure, the ACE inhibitor can help reduce the heart's workload. In addition, patients with right-sided heart failure tend to have high levels of aldosterone, and the ACE inhibitor, like spironolactone, can counteract this.

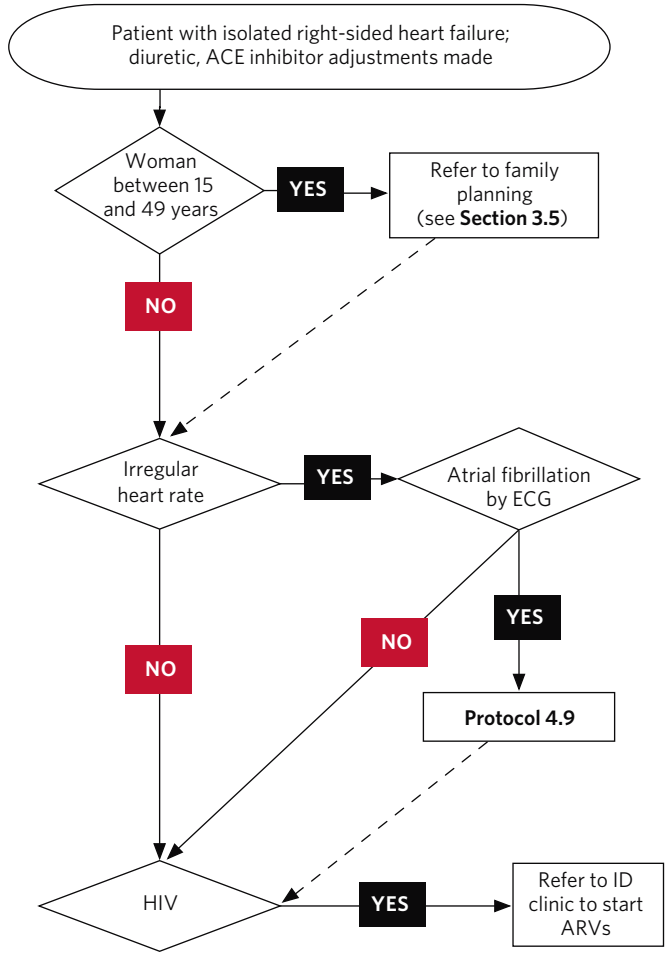
PROTOCOL 4.18 ACE-Inhibitor and Spironolactone Titration and Paracentesis for Isolated Right-Sided Heart Failure



4.12.5 Other Medication Needs

Patients with isolated right-sided heart failure may suffer from various comorbid conditions, some of which may be related to or worsen their heart failure. For instance, patients with HIV may develop pulmonary hypertension, which can cause right-sided heart failure. Women with right-sided heart failure should avoid becoming pregnant, as pregnancy may worsen their symptoms. Patients with evidence of atrial fibrillation should be rate-controlled and anticoagulated.

PROTOCOL 4.19 Other Medication Needs in Isolated Right-Sided Heart Failure



4.13 Heart Failure Patient Follow-Up

No heart failure intervention will be successful without good patient retention and follow-up. **CHAPTER 3** outlines the important role community health workers play in this effort. In addition, frequent medication adjustments require frequent clinic visits. Frequency of visits should be flexible and depend on the patient's condition. **TABLE 4.26** provides the guide used by NCD clinicians in determining how often to see patients in the clinic. Obviously, clinicians must rely on their own clinical judgment when determining how frequently a patient should be seen. In general, we encourage clinicians to see patients more frequently if they have any questions or concerns about a patient's condition or management. This allows for smaller, more frequent titration of medications and faster

recognition of a deteriorating condition. Children may need to be seen more frequently given that they may quickly grow out of their medication dose.

TABLE 4.26 Clinic Follow-Up Schedule for Heart Failure Patients

	Class I or Class II heart failure, euvolemic	Class III or Class IV symptoms, new renal failure, hypervolemic or any other clinical concern
Medication change	Return in 2 weeks–1 month	Return in 1–2 weeks
No medication change	Return in 1–2 months	Return in 2 weeks–1 month

In rural, resource-poor settings such as Rwanda, travel to a clinic can pose significant hardship to patients, especially those too ill to walk long distances. As a temporary measure, we have reduced barriers to clinic attendance by providing travel reimbursement for patients traveling long distances.

4.14 Potassium Management

Many of the medications used in heart failure management can affect potassium. For instance, potassium levels may decrease over the course of diuresis. Conversely, if patients have baseline renal dysfunction, potassium may rise to dangerous levels if renal function worsens. Potassium is an essential electrolyte that, when in imbalance, can cause significant cardiac arrhythmias and even death.

In general, potassium levels will not become dangerously high as long as creatinine is normal. Many facilities are able to check creatinine but not potassium. Accordingly, we have based our algorithms on creatinine alone. See **SECTION 6.5** for discussion of management of hyperkalemia.

4.15 Arrhythmia Diagnosis and Management

Patients with heart failure of any type are at increased risk of having abnormal heart rhythms. The field of arrhythmia diagnosis and management is vast and complex, and beyond the scope of this handbook. Here we aim to provide a basic guide for district-level clinicians in recognition and management of common arrhythmias.

4.16 Role of Electrocardiography in Rural Rwanda

Electrocardiogram (ECG) use is limited at rural district hospitals in Rwanda. Many district hospitals do not have an electrocardiogram machine, and few district hospital clinicians feel comfortable in the use or interpreta-

tion of ECGs. The goal in Rwanda is to have an ECG machine at each district hospital and to provide basic training in ECG interpretation.

The main utility of an ECG in rural Rwanda is to detect arrhythmias. In this setting, few patients have coronary artery disease, and the ability to detect myocardial infarction on ECG is a less useful skill.

Almost all arrhythmias occur among patients with heart failure, particularly among those with a reduced EF or mitral stenosis and among those who have had cardiac surgery. Atrial fibrillation is likely the most common arrhythmia in this setting. NCD clinicians with no prior ECG training can easily learn to identify its characteristic ECG pattern. Other arrhythmias are less common, harder to diagnose, and more difficult to treat. Unfortunately, many patients with ventricular arrhythmias never make it to the hospital. Patients with cardiomyopathy are at high risk for these types of rhythms, and beta-blocker use following the cardiomyopathy protocols will help reduce this risk. For these reasons, we focus our ECG teaching on the detection of atrial fibrillation among patients with established heart failure.

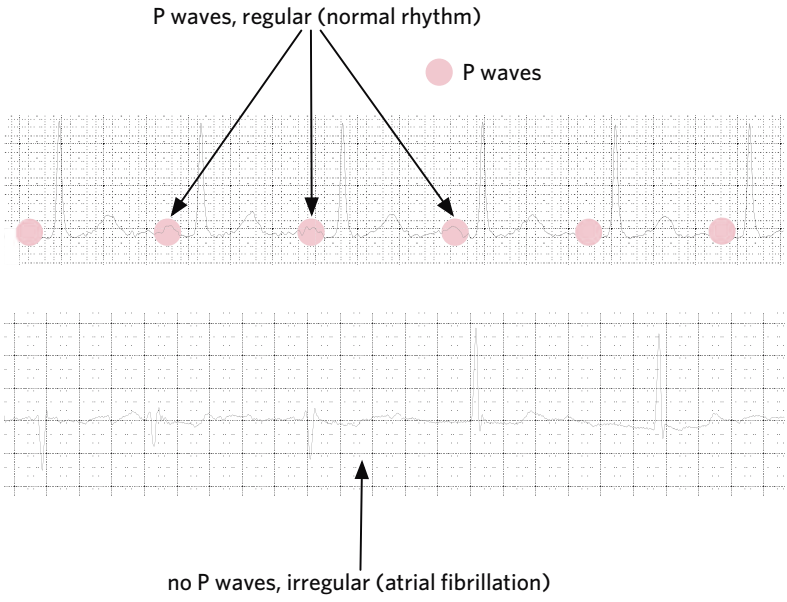
Identification of complete heart block is a second skill that can be useful. Heart block is relatively rare but potentially deadly, and it is treatable with pacemaker implantation.

All district hospitals should have an ECG machine. However, we have found that these are often cumbersome to use. Recently, some portable, one-lead ECG machines have come onto the market and may be easier to use.

4.16.1 Atrial Fibrillation Diagnosis and Management

Our protocol for atrial fibrillation diagnosis and treatment suggests that clinicians obtain an ECG on any patient with an irregular pulse or a very rapid heart rate (≥ 120 beats per minute in an adult). The NCD nurse may perform the ECG in the clinic or accompany the patient to the inpatient ward to have the ECG performed. NCD nurses should receive basic training in ECG interpretation. However, a district hospital physician should be available to help with interpretation if there are questions. The key ECG findings suggestive of atrial fibrillation are (1) absence of P waves; and (2) irregularly spaced QRS waves (see **FIGURE 4.10**).

FIGURE 4.10 ECG of Normal Rhythm (top) and Atrial Fibrillation (bottom)



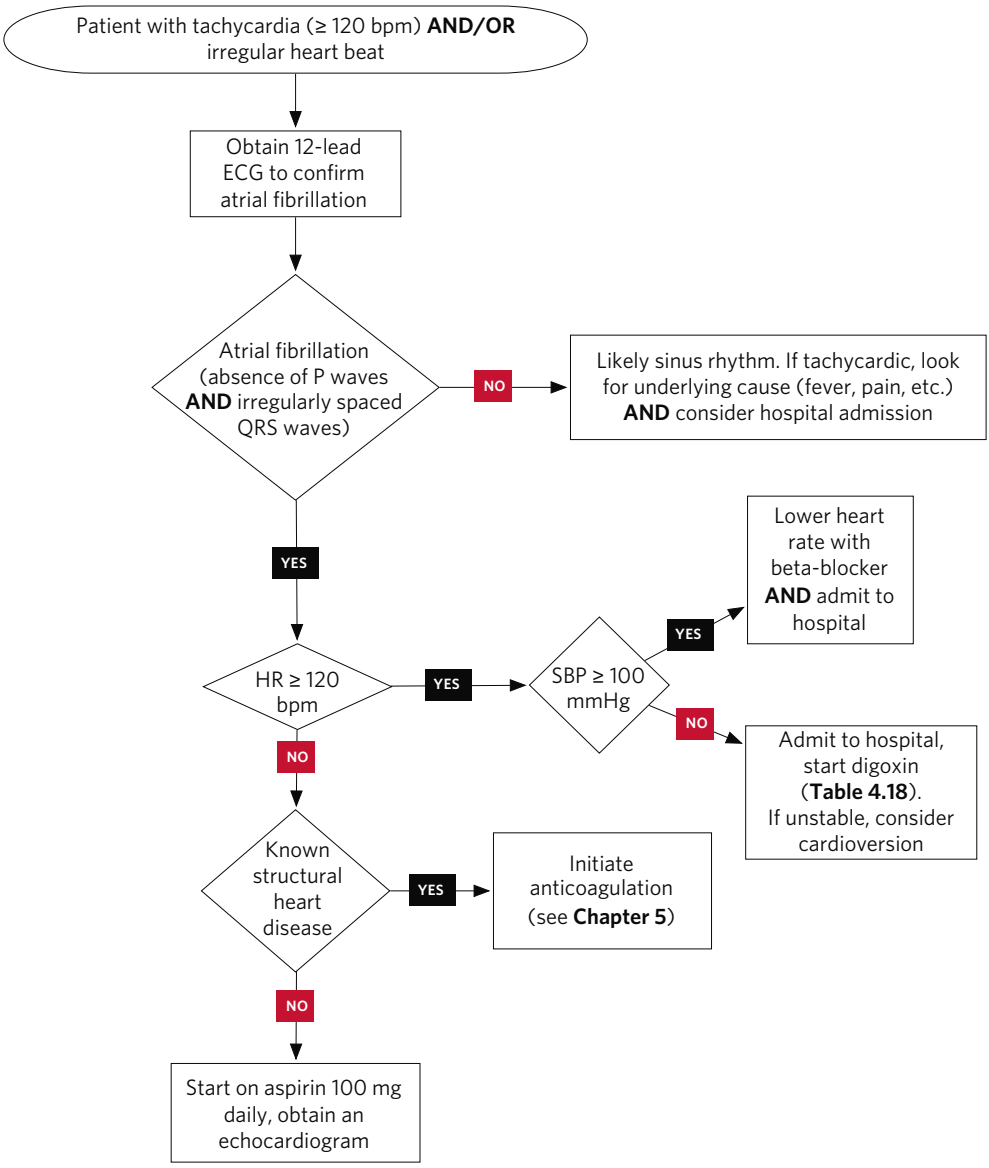
Patients with signs of decompensated heart failure should be admitted to the hospital. Patients with atrial fibrillation at a very rapid heart rate, even if it is asymptomatic, should also stay in the hospital until the rate is controlled. Adult patients with a heart rate ≥ 120 beats per minute should have their heart rate lowered if possible. Lowering the heart rate increases the time the heart has to fill and can therefore increase cardiac output. As a general rule, patients with a tachycardia (≥ 100 beats per minute in an adult) should receive a trial of beta-blocker treatment. Digoxin may also be given to help slow the heart rate and is a better choice in patients with borderline blood pressures and/or signs of decompensated heart failure. Digoxin generally takes up to a day to take effect. See **TABLE 4.18** for digoxin dosing.

Patients with decompensated heart failure, a low systolic blood pressure, and rapid atrial fibrillation may benefit from electrical cardioversion. Cardioversion machines should be available at all district hospitals.

Many of the patients who present with atrial fibrillation will have structural heart disease. Therefore, all patients found to have atrial fibrillation should have an echocardiogram.

Heart failure patients with atrial fibrillation who are rate-controlled and asymptomatic are still at high risk of stroke and should be anticoagulated. **CHAPTER 5** outlines initiation of warfarin therapy. Unlike patients who already have clots, these patients do not need to bridged with heparin therapy until they reach a therapeutic INR.

PROTOCOL 4.20 Diagnosis and Management of Atrial Fibrillation in Adults



4.17 Cardioversion

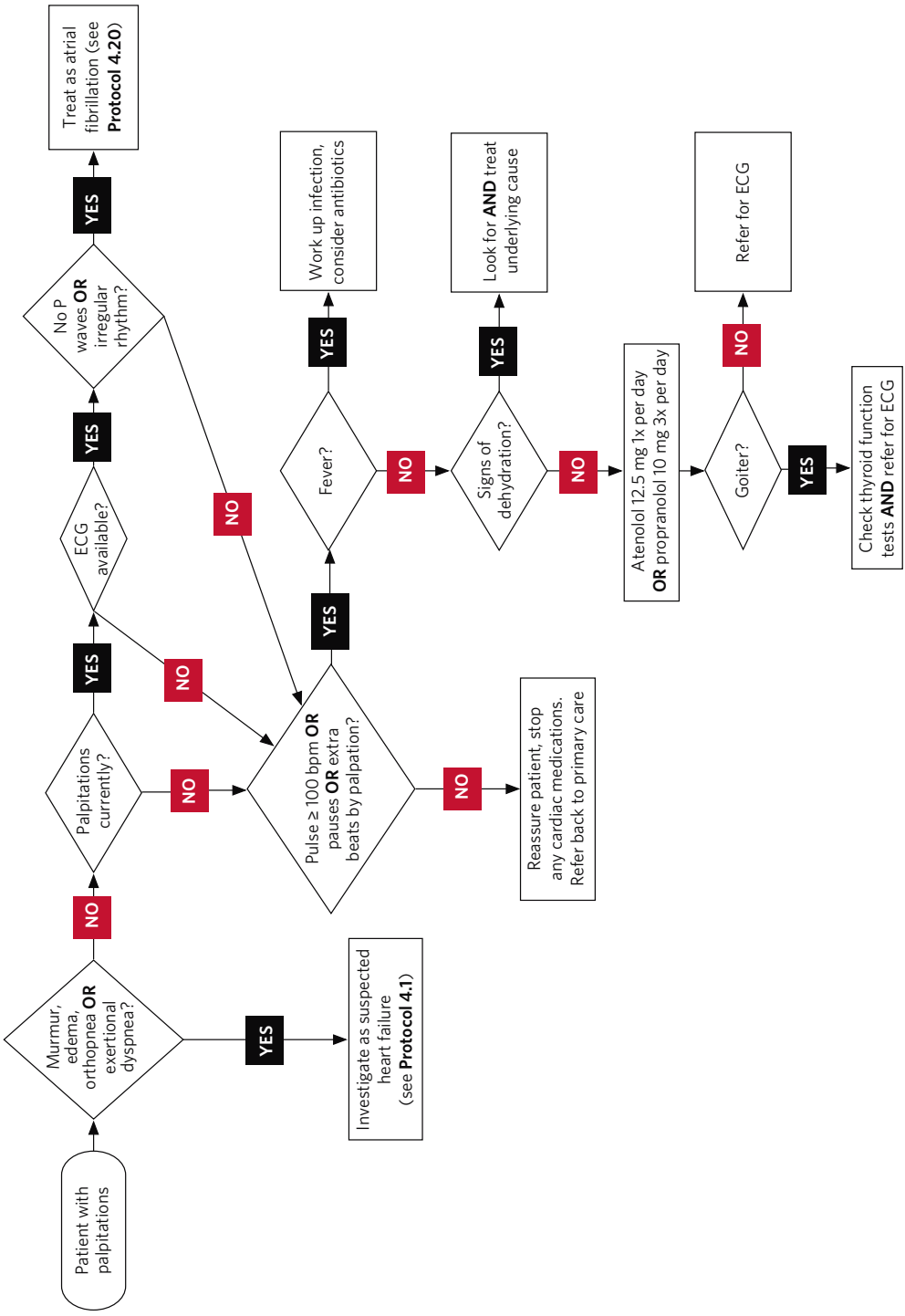
The main indication for cardioversion is cardiogenic shock in the setting of rapid atrial fibrillation. This most often occurs with mitral stenosis. Every district hospital should have a cardioversion machine. Patients who are potentially in need of cardioversion should be admitted to the hospital and managed by the inpatient physician.

4.18 Palpitations and Somatization

In Rwanda, many otherwise healthy patients seek medical care for palpitations. The vast majority of these patients have normal vital signs, physical exams, and echocardiograms. When asked in greater detail about their symptoms, many patients report that the palpitations began after the 1994 Rwanda genocide. Anecdotally, palpitations (like shortness of breath) seem to be common as a somatized expression of depression, grief, and stress.

We have developed a protocol for managing palpitations in conjunction with our mental health team (see **PROTOCOL 4.21**). If history and physical exam reveal no signs of heart failure or murmurs, and the patient has a regular heart rate and rhythm, arrhythmia is unlikely. The clinician reassures the patient and explains that there doesn't seem to be anything wrong with the heart. These patients should be asked about stressors and should be told that it is common for people to experience sadness or stress as physical complaints. These patients should be offered an appointment with the mental health team.

PROTOCOL 4.21 Palpitations in Adults



4.19 Palliative Care for Patients with Heart Failure

Palliative care is an essential part of heart failure treatment. A study comparing patients with symptomatic heart failure to patients with advanced cancers found that the two groups had similar numbers of physical symptoms and similar depression scores.[50] A study of patients dying of heart failure found that 63% experienced severe dyspnea.[51] A review of studies reporting symptoms in patients with end-stage heart disease, cancer, AIDS, chronic obstructive lung disease, and renal disease found that three symptoms—pain, dyspnea, and fatigue—had a prevalence of more than 50% in patients with each of the five diseases.[52] These physical symptoms often make it impossible for heart failure patients to work or even to be independent in self-care. Thus, symptom relief and psychosocial supports are as important for heart failure patients as they are for cancer patients.

Because heart failure typically has a relapsing and remitting course, recurrences of dyspnea and other symptoms should be anticipated and plans made—during an NCD clinic visit or prior to hospital discharge—to relieve them quickly wherever the patient might be, including in the home. When dyspnea occurs despite standard disease-modifying treatment, opioid therapy can be beneficial.[53] Morphine may improve exercise tolerance for patients with NYHA Class III disease and also can make comfortable patients with Class IV disease (see **CHAPTER 2, SECTION 2.2.4**). The usual starting doses can be used for opioid-naïve patients: 5 mg orally or in adults or 0.2–0.4 mg/kg/dose orally in children every 4 hours as needed or every four hours (see **CHAPTER 2, SECTION 2.2.3**). Because of its venodilating effect, morphine also is an effective treatment along with furosemide for acute exacerbations of pulmonary edema due to heart failure when the blood pressure is normal or elevated. In hypotensive or hypovolemic patients, however, it can cause a dangerous drop in blood pressure and thus should be used cautiously unless the only goal of care is comfort. When there is concern about side effects, morphine can be started at half the usual starting doses: 2.5 mg orally or 1 mg IV/SC in adults or 0.1–0.2 mg/kg/dose in children every 4 hours as needed.

Patients actively dying of heart failure sometimes have hours or days of dyspnea and other symptoms and require careful titration of morphine given around-the-clock to assure comfort. In some cases, health center nurses and community health workers may be able to provide this care in patients' homes once appropriate prescriptions have been written by a district-level physician. Patients with severe or complex symptoms may require end-of-life care at the health center or district level.

Patients with heart failure also may have sudden cardiac death. It may be appropriate to inform some families and patients of this possibility. Care should be taken whenever giving bad news to minimize emotional distress for the patient and family. This can be done in several ways: 1) by suggesting that support persons such as family members or friends be present when bad news will be discussed; 2) by taking the time to sit down with the patient or family and give them a chance to absorb the news; 3) by first exploring the patient's or family's understanding of the disease and gently correcting misconceptions; 4) by being prepared for strong reactions, such as anger or grief; 5) by offering to see the patient or family again soon to answer questions and provide emotional support.

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