STEP-UP to MEDICINE

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Steven Agabegi
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Step-Up SERIES

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The authors and publisher would like to thank the following student reviewers who provided valuable input in revising the new edition, as well as the many student readers who have provided ongoing feedback since the book's original publication.

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PREFACE

It is hard to believe that it has been 14 years since the first edition of *Step-Up to Medicine* was published. Now in its fifth edition, the success of this book has always been linked to its in-depth, yet concise coverage of every medical topic that a student will encounter during the clinical years of medical school and corresponding NBME shelf examinations.

This fifth edition of *Step-Up to Medicine* has been extensively revised and edited based on constructive feedback from students, residents, and faculty. To this end, we recruited a stellar team of medical professionals, led by Dr. Mark Duncan and Dr. Kelley Chuang, to update every topic and enrich the content of each chapter. We would like to acknowledge and sincerely thank these physicians for offering their expertise and valuable time in this endeavor. The quality of this edition of *Step-Up to Medicine* is the result of their tireless efforts.

We hope you find this new edition of *Step-Up to Medicine* a valuable study tool during your clinical years of medical school. We welcome any feedback or suggestions you may have for future editions. Please email us at agabegs@ucmail.uc.edu.

Steve and Liz Agabegi

CONTENTS

APPENDIX

1

Diseases of the Cardiovascular System

Dhananjay Chatterjee

Ischemic Heart Disease

Stable Angina Pectoris

A. General Characteristics

1. Stable angina pectoris is due to fixed atherosclerotic lesions that narrow the major coronary arteries. Coronary ischemia is due to an imbalance between blood supply and oxygen demand, leading to inadequate perfusion. Stable angina occurs when oxygen demand exceeds available blood supply.
2. Major risk factors
   a. Diabetes mellitus (DM)—worst risk factor
   b. Hyperlipidemia—elevated low-density lipoprotein (LDL)
   c. Hypertension (HTN)—most common risk factor
   d. Cigarette smoking
   e. Age (men >45 years; women >55 years)
   f. Family history of premature coronary artery disease (CAD) or myocardial infarction (MI) in first-degree relative: Men <55 years; women <65 years
   g. Low levels of high-density lipoprotein (HDL)
3. Less common risk factors include end-stage renal disease (ESRD) on hemodialysis, human immunodeficiency virus (HIV) infection, history of mediastinal radiation. Minor risk factors (less clear significance) include obesity, sedentary lifestyle (lack of physical activity), stress, excess alcohol use.
4. Prognostic indicators of CAD
   a. Left ventricular function (ejection fraction [EF])
      - Normal >50%
      - If <50%, associated with increased mortality
   b. Vessel(s) involved (severity/extent of ischemia)
      - Left main coronary artery—poor prognosis because it supplies approximately two-thirds of the heart
      - Two- or three-vessel CAD—worse prognosis

Quick **HIT**

CAD can have the following clinical presentations:
- Asymptomatic
- Stable angina pectoris
- Unstable angina
- Myocardial infarction (MI)—either NSTEMI or STEMI
- Sudden cardiac death

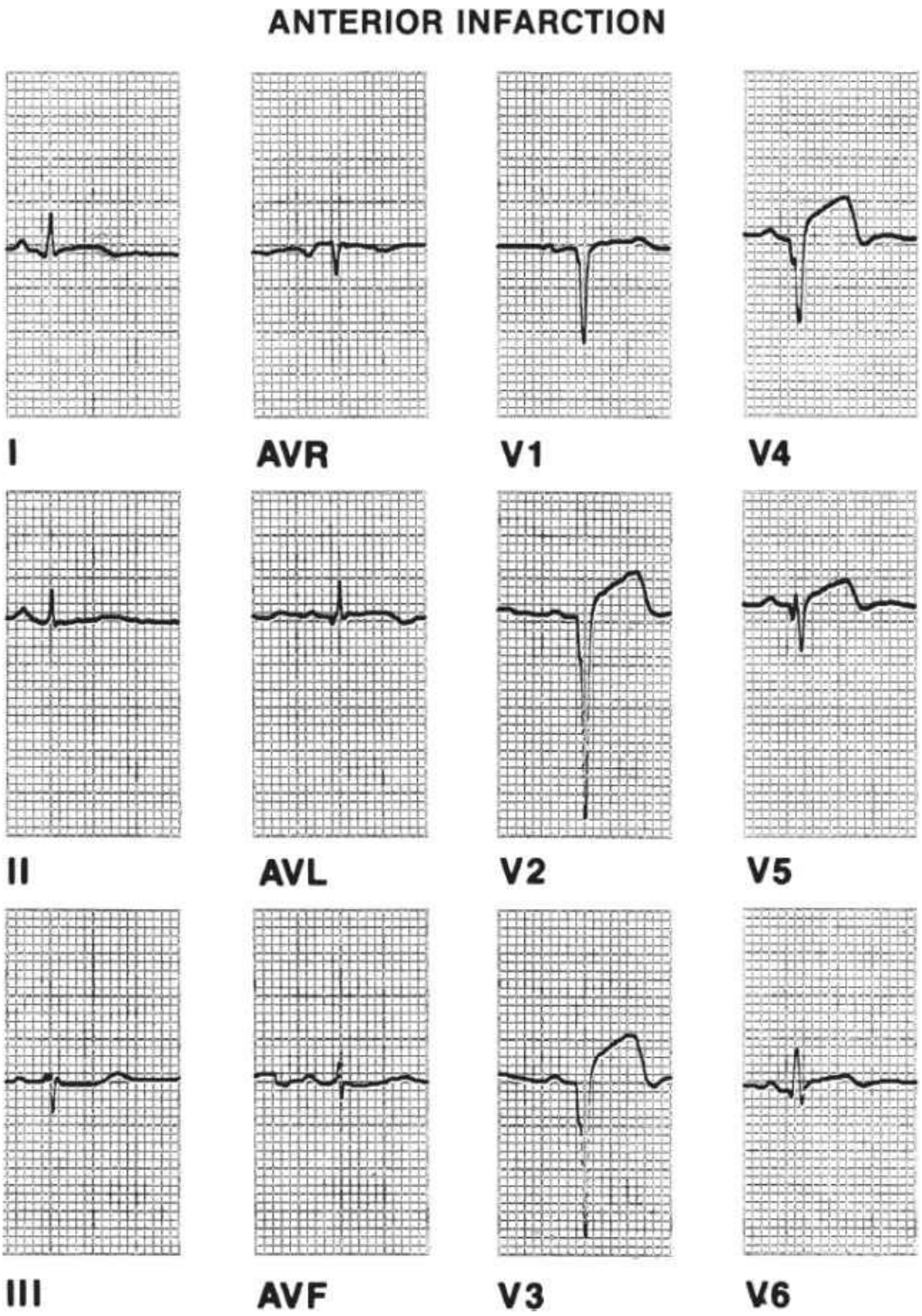

FIGURE 1.2 **ECG showing anterior wall myocardial infarction—all 12**

Congestive Heart Failure

A. General Characteristics

1. CHF is a clinical syndrome resulting from the heart's inability to meet the body's circulatory demands under normal physiologic conditions. It is the final common pathway for a wide variety of cardiac diseases (see also Clinical Pearl 1-7).
2. Pathophysiology (Figure 1-5)
   a. Frank–Starling relationship
      - In a normal heart, increasing preload results in greater contractility.
      - When preload is low (at rest), there is little difference in performance between a normal and a failing heart. However, with exertion a failing heart produces relatively less contractility and symptoms occur (Figure 1-6).

***Quick* HIT**

Often, both systolic and diastolic dysfunctions are present simultaneously.

3. Heart Failure with reduced Ejection Fraction (HFrEF) or systolic dysfunction
   a. Owing to impaired contractility (reduced EF <40%)
   b. Causes include:
      - Ischemic heart disease or after a recent MI—infarcted cardiac muscle does not pump blood (decreased EF)
      - idiopathic
      - HTN

CLINICAL PEARL 1-7

High-Output Heart Failure

- In high-output heart failure, an increase in cardiac output is needed for the requirements of peripheral tissues for oxygen.
- Causes include:
  - Chronic anemia
  - Pregnancy
  - Hyperthyroidism
  - AV fistulas
  - Wet beriberi (caused by thiamine [vitamin B_1] deficiency)
  - Paget disease of bone
  - MR
  - Aortic insufficiency
- The conditions listed above rarely cause heart failure by themselves. However, if these conditions develop

in the presence of underlying heart disease, heart failure can result quickly.

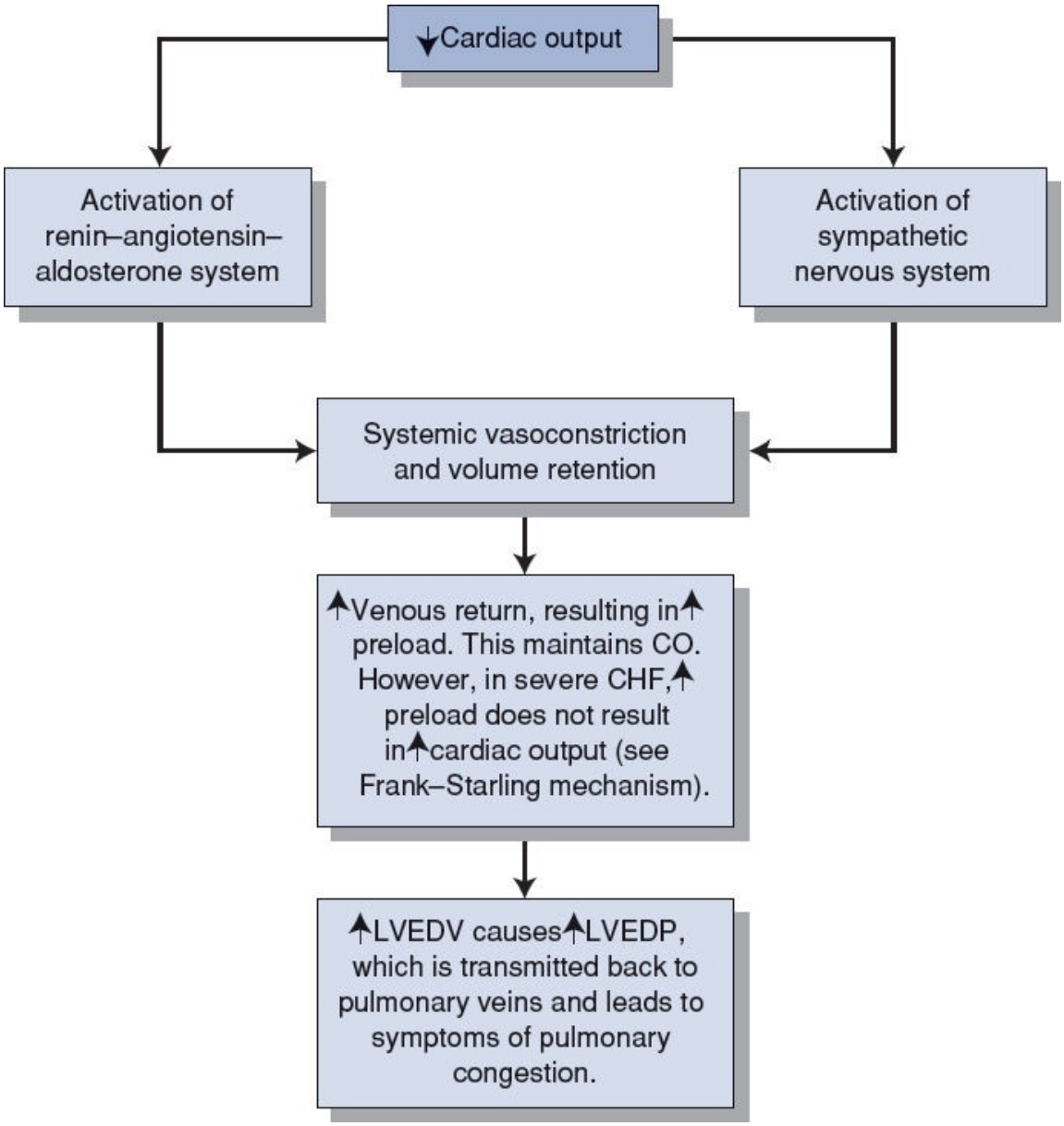

FIGURE 1.5 Pathophysiology of CHF.

- Myocarditis (postviral, giant cell, autoimmune)
- Drugs: Alcohol, cocaine, methamphetamines, chemotherapy (anthracyclines and trastuzumab)
- Infiltrative disease (amyloidosis, sarcoidosis, hemochromatosis, Wilson disease)
- Radiation therapy
- Thyroid disease
- Peripartum cardiomyopathy

- Infectious disease (Chagas disease, HIV, endocarditis causing valvular disease)
- Valvular heart disease (usually MR, aortic stenosis or regurgitation)
- High-output heart failure (severe anemia, due to AV fistulas, pregnancy, severe thiamine deficiency)
- Congenital/hereditary

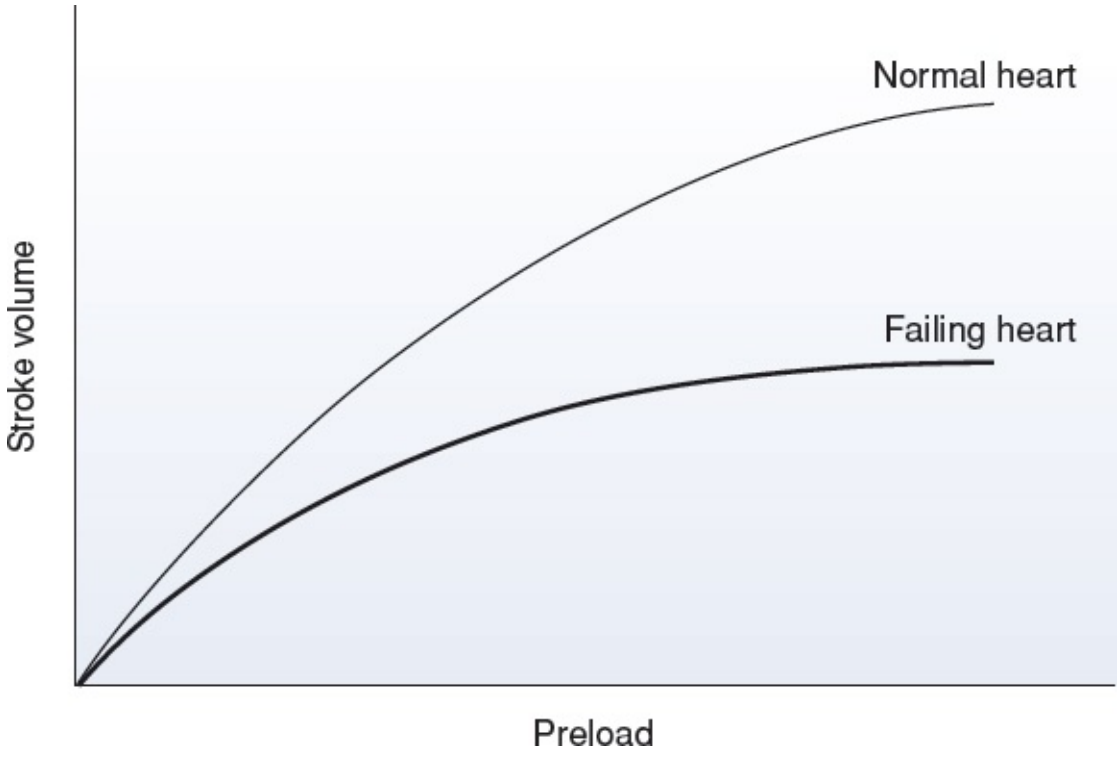

FIGURE 1.6 **Frank–Starling relationship.**

4. Heart Failure with preserved Ejection Fraction (HFpEF) or diastolic dysfunction
   a. Owing to impaired ventricular filling during diastole (either impaired relaxation or increased stiffness of ventricle or both).
   b. Echocardiogram shows impaired relaxation of left ventricle.
   c. Causes include:
      - HTN leading to myocardial hypertrophy—most common cause of diastolic dysfunction
      - Valvular diseases such as aortic stenosis (AS), mitral stenosis, and aortic regurgitation
      - Restrictive cardiomyopathy (e.g., amyloidosis, sarcoidosis, hemochromatosis in their early phases)

B. Clinical Features

1. Symptoms of left-sided heart failure (see also Clinical Pearl 1-8)

Spinal Cord Diseases

Syringomyelia

Central cavitation of the cervical cord due to abnormal collection of fluid within the spinal cord parenchyma (Figure 5-5).
Most commonly associated with Arnold–Chiari malformation. Other causes are posttraumatic, postinfectious, tethered cord, intramedullary tumors.
Clinical features—most often asymptomatic and discovered incidentally on MRI obtained for other reasons. Symptoms may include **bilateral loss of pain** and temperature sensation over the shoulders in a **"cape-like" distribution** (lateral spinothalamic tract involvement), preservation of touch, thoracic scoliosis and muscle atrophy of the hands may occur.
Diagnosed by MRI.
Treatment depends on size of syrinx, symptoms, and associated findings (Chiari, tethered cord). Evaluation by neurosurgery recommended.

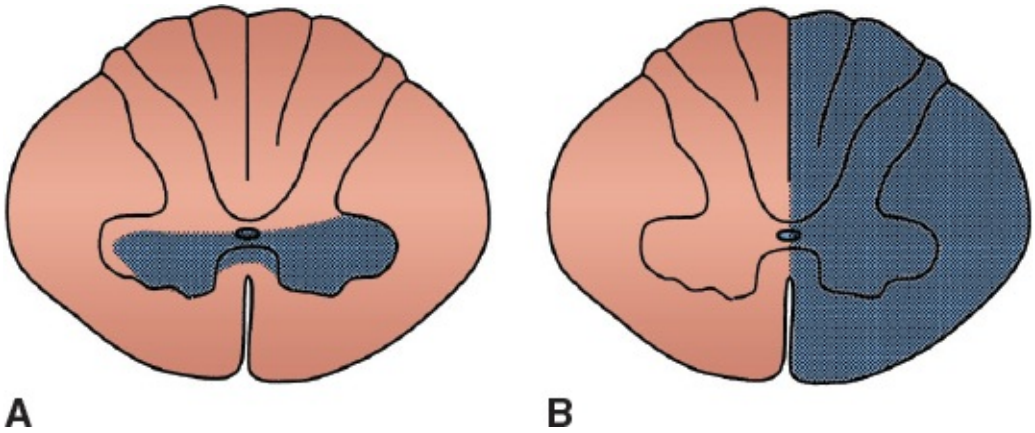

FIGURE 5.5 **Classic lesions of the spinal cord.** A**: Syringomyelia.** B**: Hemisection of the spinal cord (Brown-Séquard syndrome).**

(From Fix JD. *High-Yield Neuroanatomy*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2000:46, Figure 8-2H and E, respectively.)

d. Additional diagnostic tests may be indicated, and include ambulatory ECG monitoring, exercise testing, carotid sinus massage, neurologic studies such as EEG or neurovascular imaging, and electrophysiologic studies. Tilt-table testing was frequently used to diagnose vasovagal syncope in the past, but is now controversial.

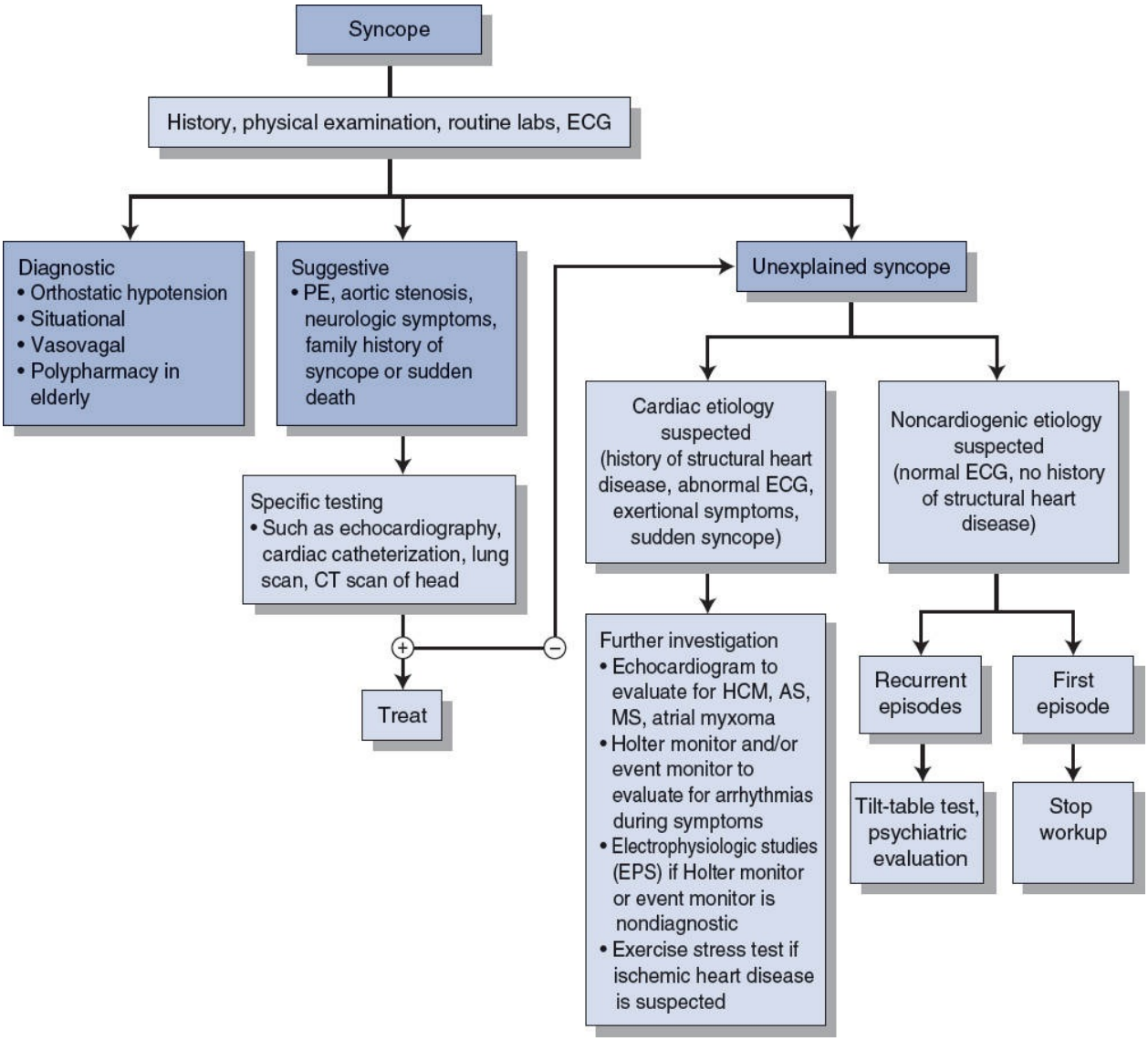

FIGURE 5.6 Syncope flowchart.

(Adapted from Heaven DJ, Sutton R. Syncope. Crit Care Med 2000;28(10 Suppl):118, Fig. 1.