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Manual of Cardiovascular Diagnosis and Therapy

Fourth Edition

配英汉索引

心血管疾病诊断与治疗手册

Edited by
Joseph S. Alpert
James M. Rippe

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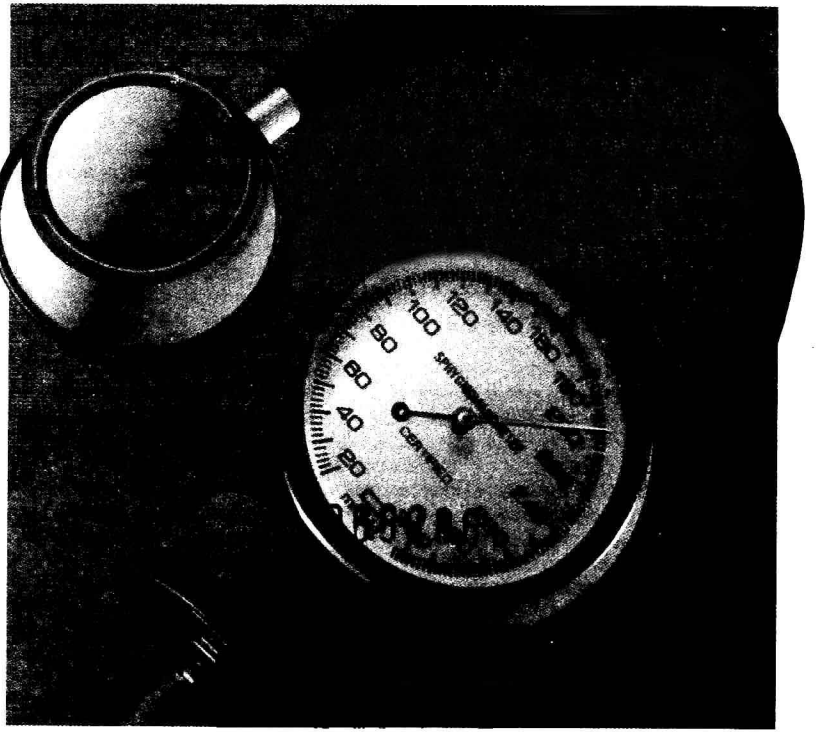

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天津科技翻译出版公司出版

著作权合同登记号:图字:02-2002-65

图书在版编目(CIP)数据

心血管疾病诊断与治疗手册 = Manual of Cardiovascular Diagnosis and Therapy/(美)阿尔伯特(Alpert, J.S.),(美)里佩(Rippe, J.M.)编著 . - 影印本 . - 天津:天津科技翻译出版公司,2003.1

(SPIRAL® MANUAL 系列丛书)

ISBN 7-5433-1605-6

Ⅰ. 心... Ⅱ. ①阿... ②里... Ⅲ. 心脏血管疾病-诊断-手册-英文 Ⅳ. R54-62

中国版本图书馆 CIP 数据核字(2002)第 080578 号

授权单位: Lippincott Williams & Wilkins Inc.
出　　版: 天津科技翻译出版公司
出 版 人: 邢淑琴
地　　址: 天津市南开区白堤路 244 号
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印　　刷: 天津市蓟县宏图印务有限公司印刷
发　　行: 全国新华书店
版本记录: 900×1168　32 开本　13.25 印张　400 千字
2003 年 1 月第 1 版　2003 年 1 月第 1 次印刷
定价:33.00 元

(如发现印装问题,可与出版社调换)

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Fourth Edition

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Little, Brown and Company
Boston New York Toronto London

Fourth Edition

Previous editions copyright © 1988, 1985 by Joseph S. Alpert and James M. Rippe; 1980 by Little, Brown and Company (Inc.)

Library of Congress Cataloging-in-Publication Data

Alpert, Joseph S.
Manual of cardiovascular diagnosis and therapy / Joseph S. Alpert, James M. Rippe. — 4th ed.
p. cm.
Includes bibliographical references and index.
ISBN 0-316-03531-9
1. Cardiovascular system — Diseases — Handbooks, manuals, etc.
I. Rippe, James M. II. Title.
[DNLM: 1. Cardiovascular Diseases — diagnosis — handbooks.
2. Cardiovascular Diseases — therapy — handbooks. WG 39 A456ma 1996]
RC667.A39 1996
616.1 — dc20
DNLM/DLC
for Library of Congress 95-24885
CIP

Printed in the United States of America

SEM

Second Printing

Editorial: Tammerly J. Booth, Richard L. Wilcox
Production Editor: Kellie Cardone
Copyeditor: Ann Calandro
Indexer: Beta Indexing
Production Supervisor: Mike Burggren
Cover Designer: Ethan Thomas

Preface

The first three editions of the *Manual of Cardiovascular Diagnosis and Therapy* have been enthusiastically received by physicians in practice and in training and by nurses and physicians' assistants. This fourth edition continues the process employed in earlier editions by updating diagnostic and therapeutic strategies for patients with various cardiovascular diseases.

Most of the chapters and tables have been significantly altered and expanded to accommodate the vast amount of new literature and the technologic advances that have become available since the publication of the third edition. In addition, new chapters have been added containing material that was mentioned only briefly in earlier editions. The manual is still focused so as to aid harried physicians, house officers, and medical students involved in the daily care of patients with cardiovascular disease. The introductory chapters review salient features of cardiovascular physiology, the cardiovascular physical examination, and the numerous tests available for cardiovascular diagnosis. Following these initial chapters, common complications of patients with cardiac disease, such as arrhythmias, are reviewed. Finally, specific cardiovascular disease entities are dealt with in the longest section of the manual. Each chapter is written in an outline format for easy scanning. Chapters on specific disease entities follow identical formats, dealing first with various aspects of diagnosis and second with approaches to medical and surgical management. A brief annotated bibliography is appended to each chapter.

We gratefully acknowledge the assistance and inspiration of the following colleagues and secretaries: James E. Dalen, M.D., Gordon Ewy, M.D., Barbara Raney, Marie G. Striednig, and Beth Porcaro.

J.S.A.
J.M.R.

CONTENTS 目 录

Introduction to the Cardiovascular System

Notice. The indications and dosages of all drugs in this book have been recommended in the medical literature and conform to the practices of the general medical community. The medications described do not necessarily have specific approval by the Food and Drug Administration for use in the diseases and dosages for which they are recommended. The package insert for each drug should be consulted for use and dosage as approved by the FDA. Because standards for usage change, it is advisable to keep abreast of revised recommendations, particularly those concerning new drugs.

1 Physical Examination of the Heart

I. Introduction. Careful physical examination of the heart provides important information about the cardiovascular system. Together with a thorough history, the physical examination provides the initial data base and suggests further diagnostic tests and therapeutic maneuvers. In many conditions, careful physical examination can yield information as important as that obtained by more complex and costly procedures.

It is also important to recognize the complex interplay between cardiac disease and other systemic illnesses or conditions. A common mistake made by the noncardiologist is ignoring the cardiac manifestations of a systemic disease process. Conversely, the cardiologist may fail to recognize the effects of cardiac disease on other organ systems. For these reasons every patient suspected of having cardiac abnormalities must be given a thorough physical examination.

This chapter focuses on diagnostic aspects of physical examination of the heart and provides general guidelines for evaluating physical findings. Descriptions of the actual techniques of physical examination and theories concerning the origins of heart sounds are beyond the scope of this manual. Detailed descriptions of physical examination findings for various cardiac diseases are found in the chapter for each entity.

II. Observations, palpation, and percussion

A. Jugular venous pulse (JVP). Two types of information are obtained from the JVP: the quality of the wave form and the central venous pressure (CVP).

1. Technique of examination. The JVP is best observed in the right internal jugular vein. With normal CVP, the JVP is assessed with the patient's trunk raised less than 30 degrees. With elevated CVP the patient's trunk must be raised higher, sometimes to as much as 90 degrees. The JVP is accentuated by turning the patient's head away from the examiner and shining a flashlight obliquely across the skin overlying the vein.

2. Wave form of the JVP. Two waves per heartbeat are generally visible in the JVP: the A wave and the V wave. The A wave appears as a brief "flicker" and represents increased venous pressure resulting from atrial contraction. The V wave is a longer surge that follows the A wave and represents increased venous pressure transmitted during ventricular contraction. The drop in pressure following the A wave is called the X descent, and the fall in pressure after the V wave is denoted as the Y descent. The JVP waves should be timed with simultaneous palpation of the carotid artery. The A wave immediately precedes the carotid pulse; the V wave follows the pulse.

Occasionally, difficulty may be experienced in differentiating venous and arterial pulsations in the neck. Several observations and findings may be helpful: (1) the arterial pulse is more localized and will forcefully strike examining fingers, whereas palpation will often obliterate a venous pulse; (2) compression of the base of the neck will not alter arterial pulses but will obliterate the venous ones; (3) arterial pulses do not change with patient position while venous pulses will often disappear when the patient assumes an upright posture (either sitting upright or standing).

The diagnosis of a variety of pathologic states is assisted by observation of abnormalities in the JVP wave forms (Table 1-1).

Table 1-1. Clinical information derived from abnormal jugular venous pulsations

Finding	Comment/significance
Markedly raised central venous pressure, accentuated X and Y descents	? Cardiac tamponade ? Constrictive pericarditis ? Endocardial fibroelastosis ? Severe right-heart failure
Large A waves	? Pulmonary valvular stenosis ? Hypertension ? Various arrhythmias where atria contract against closed AV valve (e.g., junctional rhythm, AV dissociation)
Absent A wave	Atrial fibrillation
Large V wave	Tricuspid regurgitation

3. **Determination of CVP.** CVP can be estimated by observing the vertical distance from the top of the V wave to the right atrium. In the individual with normal CVP, the V wave rises 1–2 cm above the sternal angle. When the V wave rises to more than halfway to the angle of the jaw in a patient who is not recumbent, elevated CVP is present. In some pathologic conditions (e.g., cardiac tamponade, constrictive pericarditis), CVP may be so high that A and V waves are above the angle of the jaw. In this setting, exaggerated X and Y descents may suggest the diagnosis. As a rule of thumb, for a patient sitting upright, a JVP visible at the sternal angle represents a CVP of approximately 10 mm Hg.

   During inspiration the height of the JVP typically declines (although amplitude of the X and Y descents will increase). In certain pathologic conditions such as chronic constrictive pericarditis and occasionally tricuspid stenosis, congestive heart failure, right ventricular dysfunction, or infarction the JVP actually increases with inspiration. This important clinical finding is known as **Kussmaul's sign.**

B. Arterial pressure pulse. The central arterial pressure pulse is characterized by a rapid rise to a rounded shoulder peak with a less rapid decline. Information about the adequacy of ventricular contraction and possible obstruction of the left ventricular outflow tract may be assessed by palpation of the carotid artery. By the time the pulse wave is transmitted to peripheral arteries, much of this initial information is lost; however, pulsus alternans is best evaluated in peripheral arteries.

A variety of pathologic conditions alters the characteristics of the carotid pulse. These conditions, and the corresponding modifications of the carotid pulse, are listed in Table 1-2. In patients with unexplained hypertension, simultaneous palpation of radial and femoral arterial pulses helps to rule out coarctation of the aorta.

C. Precordial palpation. Information concerning the location and quality of the left ventricular impulse is available through precordial palpation. In addition, intensity of murmurs may be gauged by palpating associated thrills.

Palpation is best accomplished using the fingertips, with the patient either supine or in the left lateral decubitus position. Simultaneous auscultation can aid in the timing of events. A list of abnormalities detected by precordial palpation and their significance is found in Table 1-3.

III. Auscultation

A. S1. The first heart sound (S1) occurs at the time of closure of the mitral and tricuspid valves. It is probably generated by the closure of the valves. S1 is frequently split (with mitral closure preceding tricuspid), but this event is often hard to appreciate and of little clinical relevance. More important is variation

Table 1-2. Clinical information derived from abnormalities in carotid pulse

Finding	Comment/significance
Pulsus bisferiens (2 systolic peaks)	Found in aortic regurgitation and hypertrophic obstructive cardiomyopathy
Pulsus parvus (small, weak pulse)	Any condition causing diminished left ventricular stroke volume or narrow pulse pressure (hypovolemia, mitral/aortic valve stenosis, restrictive pericarditis, recent myocardial infarction); may also be caused by atherosclerosis of the carotid artery or diseases of the aortic arch
Pulsus tardus (delayed systolic peak of pulse)	Aortic outflow obstruction
Pulsus paradoxus (larger than normal decrease in systolic arterial pressure during inspiration)	Pericardial tamponade, airway obstruction, superior vena caval obstruction; may be seen in asthma or COPD
Pulsus alternans (consistent alternation in pulse pressure amplitude despite regular rhythm)	Severe left ventricular decompensation for any reason; following paroxysmal tachycardia; for several beats following a premature beat

Table 1-3. Clinical significance of abnormalities in precordial palpation

Finding	Comment/significance
Left ventricular thrust	Left ventricular hypertrophy
Displacement of left ventricular pulse downward and to the left	Left ventricular dilatation; left ventricular failure; volume overload (aortic regurgitation or decompensated mitral regurgitation)
Presystolic impulse	Pressure overloaded states (hypertension, aortic stenosis)
Double systolic impulse	Hypertrophic obstructive cardiomyopathy
Systolic bulge (dyskinetic impulse)	Coronary artery disease, recent myocardial infarction (most commonly felt above and medial to the point of maximal impulse)
Parasternal lift	Mitral regurgitation (occurs after the left ventricular apical impulse); right ventricular dilatation (mitral stenosis, pulmonary embolism)
Thrills	Aortic stenosis, pulmonic stenosis; ventricular septal defect, severe mitral regurgitation

Table 1-4. Clinical information derived from abnormalities in S1

Finding	Comment/significance
Loud S1	Short P–R interval
Loud "snapping" S1	Mitral stenosis (pliable valve)
Variation in intensity of S1	Complete heart block
Diminished intensity of S1	Mitral regurgitation, slow heart rate (long P–R interval), poor conduction of sound through chest wall, slow rise of left ventricular pressure, mitral stenosis (rigidly calcific valve), severe or acute aortic regurgitation

in intensity of the first sound. S1 varies with the P–R interval of the ECG. The shorter the P–R interval, the louder the S1. The best example of S1 variation with P–R interval occurs in complete heart block, in which atrial and ventricular contractions are dissociated.

S1 may be loud and "snapping" in quality in mitral stenosis, indicating both that the valve is pliable and that it remains wide open at the beginning of isovolumic contraction. Conversely, a diminished or absent S1 in mitral stenosis suggests a rigidly calcified valve that cannot "snap" shut.

Other situations in which S1 may be diminished include mitral regurgitation, slow heart rates (long P–R interval), poor sound conduction through the chest wall, and a slow rise of left ventricular pressure. A summary of clinical information derived from variations in S1 is found in Table 1-4.

B. S2. In contrast to S1, in which splitting is less important than changes in intensity, S2 reveals variations in both splitting and intensity that provide important clinical information.

The second heart sound (S2) occurs at the time of closure of the aortic and pulmonic valves. In normal circumstances, aortic closure precedes pulmonic closure (A2 followed by P2). Under normal circumstances, the split in S2 is maximal at the end of **inspiration** and minimal at the end of **expiration.** This phenomenon reflects an underlying movement of P2 with respect to a relatively constant A2. During inspiration, right ventricular filling increases and P2 is delayed, causing the widely split S2. During expiration, less right ventricular filling occurs and P2 "closes" toward A2, causing a diminished split in S2. This "normal splitting" of S2 is invariably present in individuals under 30 years of age, provided heart rates are not markedly accelerated. It is best appreciated over the "pulmonic area" and can be heard with either the bell or the diaphragm.

1. **Fixed splitting of S2.** The most common abnormality of S2 is failure of splitting to close at the end of expiration. This "fixed splitting" occurs for either of two reasons: P2 is delayed or A2 is early. A split of S2 on expiration may also represent a normal variant. In the latter setting, however, some difference in the degree of split should occur between inspiration and expiration.

   Fixed splitting of S2 due to delayed P2 is found in four clinical settings: (1) acute right-heart pressure overload (e.g., pulmonary embolism), (2) right bundle branch block, (3) atrial septal defect (ASD), (4) pulmonic stenosis.
2. **Paradoxical splitting of S2.** Paradoxical splitting of S2 is said to be present when S2 splits on expiration and closes on inspiration. Although fixed splitting denotes delay in normal closure of the pulmonic valve, paradoxical splitting denotes delayed closure of the aortic valve. This important clinical sign never occurs in the absence of cardiac disease. The most common states in which paradoxical splitting is encountered are aortic stenosis and left bundle branch block. Paradoxical splitting takes place in about 25% of individuals with these conditions.

Paradoxical splitting may occur in patients with coronary artery disease or hypertension or both. In these individuals a closely split S2 may be observed to close to a single sound at midinspiration. A similar finding is often made in early stages of aortic stenosis or in incomplete left bundle branch block. Alterations in the intensity of S2 can also yield important clinical information. A2 is frequently decreased in aortic stenosis. The presence of a normal A2 when aortic stenosis is clinically suspected raises the question of outflow obstruction at a site other than the valve. P2 may be augmented in pulmonary hypertension and diminished in pulmonic stenosis. Finally, P2 may appear unusually loud in thin-chested individuals without cardiac disease.

A summary of clinical information derived from alterations in S2 is found in Table 1-5.

C. S3. The third heart sound (S3, or ventricular gallop) is low-pitched and best heard at the apex with the stethoscope bell. The S3 is probably the result of rapid filling and stretching of an abnormal left ventricle. The cadence of the S3 has been likened to the *y* in *Kentucky*. An S3 may be heard in any condition resulting in rapid ventricular filling. It is frequently an early sign of left ventricular failure. Third heart sounds may also be present in atrial septal defect, mitral or aortic insufficiency, ventricular septal defect, and patent ductus arteriosus. An S3 can also be a normal variant, particularly in young adults. A loud, early diastolic sound is often heard in constrictive pericarditis. This "pericardial knock" may be mistaken for an S3.

D. S4. The fourth heart sound (S4, atrial gallop, presystolic gallop) is also the result of altered ventricular compliance. Its cadence has been likened to the soft *a* of *appendix*. It is a low-pitched sound, best heard with the stethoscope bell. It is loudest at the apex and may be accentuated by placing the patient in the left lateral decubitus position. The presence of an S4 implies effective atrial contraction; it is never heard in atrial fibrillation. An S4 may be heard in any condition causing reduced ventricular compliance: aortic stenosis, systemic or pulmonary hypertension, coronary artery disease, hypertrophic cardiomyopathy, acute mitral regurgitation, and myocardial infarction.

Table 1-5. Clinical information derived from abnormalities in S2

Finding	Comment/significance
Abnormalities in timing	
Fixed splitting	Acute right-heart overload (e.g., pulmonary embolism) Right bundle branch block Atrial septal defect (often widely split) Pulmonic stenosis
Paradoxical splitting	Aortic stenosis Left bundle branch block
Closely split with closure at midinspiration (variant of paradoxical splitting)	Coronary artery disease Hypertension
Abnormalities in intensity	
Increased A2	Hypertension Aortic dilatation
Increased P2	Pulmonary hypertension Normal finding in thin-chested individual
Decreased A2	Aortic stenosis
Decreased P2	Pulmonic stenosis

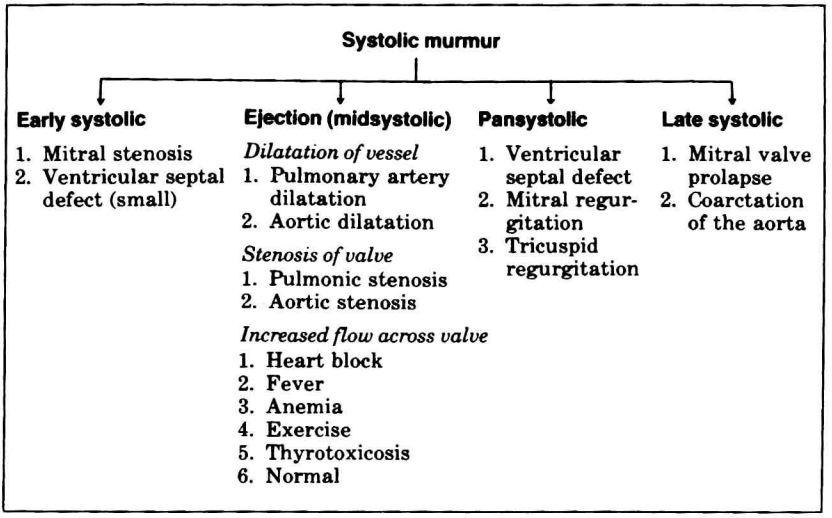

Fig. 1-1. Differential diagnosis of systolic murmurs.

E. Snaps, clicks, and other adventitious sounds

1. **Opening snap (OS).** An opening snap of the mitral valve is frequently heard in mitral stenosis. The opening snap arises from the stiff mitral valve's snapping toward the left ventricle in early diastole. The opening snap is best heard in the fourth intercostal space halfway between the apex and the left sternal border. The interval between S2 and the OS is related to the severity of mitral stenosis. The more severe the stenosis, the shorter the S2–OS interval.
2. **Ejection clicks.** Ejection clicks are high-pitched sounds occurring in early systole. They are associated with stenosis of either the aortic or the pulmonic valve, with hypertension or dilatation of either the aorta or the pulmonary artery or both. Aortic clicks are best heard at the apex, while pulmonic clicks are most audible at the left upper sternal border. Pulmonic clicks vary with respiration and are best heard during expiration. Aortic clicks do not vary with respiration.
3. **Midsystolic (nonejection) clicks.** Midsystolic clicks often accompanied by a late systolic murmur occur in patients with prolapse of the posterior leaflet of the mitral valve. The clicks may result from sudden tensing of the chordae tendineae or snapping of the prolapsing leaflet. The clicks may be single or multiple and may occur at any time during systole, although they generally come later than ejection clicks.

F. Systolic murmurs. Systolic murmurs are classified according to their time of occurrence, sound quality, and duration. The most fundamental distinction is between systolic ejection murmurs and pansystolic murmurs. Ejection murmurs ordinarily occur in midsystole. Early and late systolic murmurs also occur and should be distinguished from ejection murmurs. Ejection murmurs begin after S1 and are usually crescendo-decrescendo ("diamond-shaped"), ending before S2. Pansystolic murmurs begin with S1, extend throughout systole, and are characteristically uniform in intensity. Systolic ejection murmurs have been likened to the chug of a steam engine laboring up a hill, while pansystolic murmurs have been likened to the high-pitched wail of the engine's whistle.

1. **Systolic ejection murmurs (SEMs).** SEMs begin after the semilunar (aortic and pulmonic) valves open at the end of isovolumic systole. Their intensity parallels the amount of blood being ejected through the stenosis, peaking in

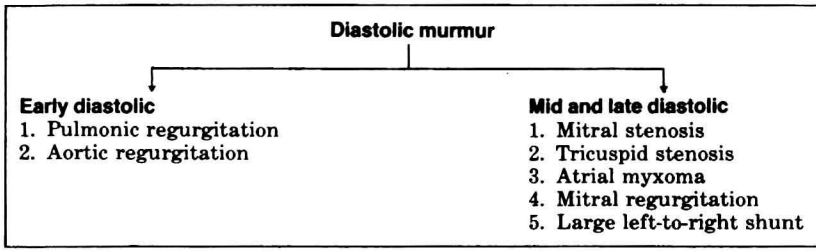

Fig. 1-2. Differential diagnosis of diastolic murmurs.

midsystole. SEMs arise in the following settings: (1) aortic or pulmonic stenosis, (2) dilatation of the aorta or pulmonary artery distal to the valve, (3) increased rate of ventricular ejection (heart block, fever, anemia, exercise, thyrotoxicosis), and (4) healthy individuals.

2. **Pansystolic murmurs.** Pansystolic murmurs occur when blood flows through a ventricular septal defect, or retrograde through the mitral or tricuspid valve. The even intensity and long duration of these murmurs reflect the large pressure difference across the orifice where the sound originates. The murmur continues as long as pressure in the chamber of origin exceeds that in the recipient chamber.
3. **Early and late systolic murmurs.** Early systolic murmurs begin with or shortly after S1 and end by midsystole. They have been reported in (1) mitral stenosis (etiology unclear, either coexistent mitral regurgitation or distortion of the mitral valve apparatus), (2) small ventricular septal defects, and (3) individuals without cardiac disease.

   Late systolic murmurs begin in midsystole and extend to or through S2. They may be heard in mitral valve prolapse (frequently accompanied by midsystolic clicks) or coarctation of the aorta.
4. **Summary.** Figure 1-1 summarizes systolic murmurs. Systolic murmurs arising from the right side of the heart generally increase with inspiration while those originating on the left side decrease or do not change. Many systolic murmurs are totally innocent (as in pregnant women, growing children, and individuals with abnormal chest configuration).

G. **Diastolic murmurs.** Diastolic murmurs are classified according to their position in diastole as early, mid, or late. An alternative classification emphasizes etiology: regurgitant murmurs from semilunar insufficiency versus ventricular filling murmurs. Regurgitant murmurs are generally early diastolic whereas ventricular filling murmurs occur in mid and late diastole.

1. **Early diastolic murmurs.** Early murmurs begin immediately after S2. The most common causes are aortic or pulmonic valve regurgitation. The murmur is usually high-pitched and blowing in quality with a decrescendo configuration. The intensity of the murmur reflects the size of the valvular leak, the acoustic properties of the chest, and the pressure difference across the valve. The distinction between pulmonic and aortic regurgitation may be extremely hard to make and may require catheterization for definitive determination.
2. **Mid and late diastolic murmurs.** Mid and late diastolic murmurs are produced by forward flow of blood through the AV (mitral and tricuspid) valves. They arise from either augmented blood flow or a stenosed valve. As a rule the murmur is low-pitched and rumbling in quality. It does not begin until the valve from which it originates opens (sometimes with an audible snap) and ventricular pressure has fallen below atrial pressure in early diastole. Conditions in which mid or late diastolic murmurs may arise include (1) mitral or tricuspid stenosis, (2) left atrial myxoma, (3) mitral regurgitation (increased flow), and (4) large left-to-right shunts (increased flow).

   A summary of murmurs that may occur in diastole is found in Figure 1-2.