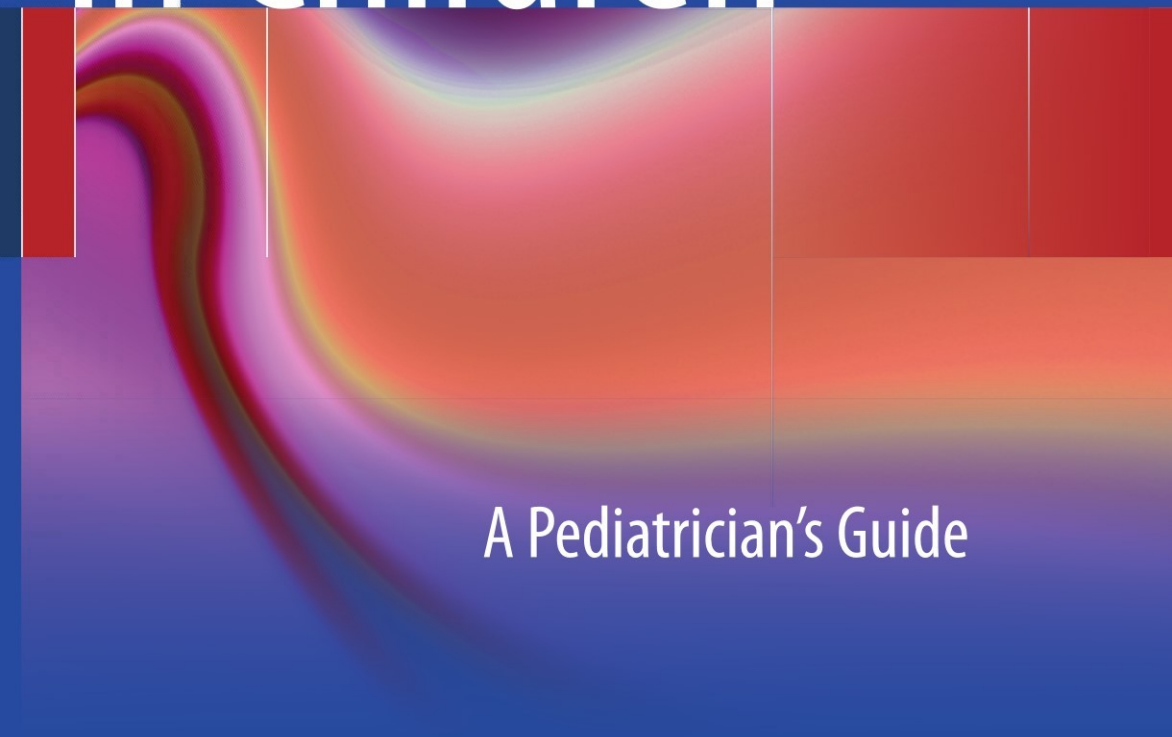
Ra-id Abdulla *Editor*

Heart Diseases in Children



A Pediatrician's Guide

Springer

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To my late father Muhammed, my mother Zaineb, wife Janaan and children Muhammed, Zaineb and Maryem.

One of my earliest childhood memories is of my parents with a book in their lap, reading and later relating and debating their literary experience. This passion for reading was one of the most potent factors in shaping my life. Growing up in a home where reading was as normal as having meals and books crowding shelves and piling high on tables in every room enriched my mind and soul. My parents' interest in what I read and write led me to a parallel universe where I can experience lives I have never lived and through my own writing create a private world to satisfy my imagination. My wife and children amplified this love of books through their own passion and dozens of books they added to our home library. To all of them I am indebted for this gift and much more.

Knowledge we spend a lifetime cultivating dies with our mortality and only escape this unfortunate fate through our recorded words.

Foreword

Heart Diseases in Children: *A Pediatrician's Guide* fills an important need in pediatrics and occupies a valued spot on the bookshelves of practicing cardiologists and pediatricians. This outstanding new reference work is in part the byproduct of my colleague Dr. Ra-id Abdulla's decade of editorship of the journal *Pediatric Cardiology*, his creation of one of the most visited internet Web sites in his field, and his leadership of outstanding fellowship training programs at the University of Chicago and Rush University. Dr. Abdulla is a consummate educator in the field of pediatric cardiology. His mastery is evident in the abundance of understandable illustrations, images of actual cases, and personal observations of real life practice that fill this book.

The management of children with heart disease – whether asymptomatic or symptomatic, diagnosed or undiagnosed, congenital or structural, corrected or palliated, acute or chronic – requires collaborative teamwork between the pediatric cardiologist and the primary care pediatrician. With this in mind, each of the chapters in this book has the dual authorship of an academic cardiologist and a practicing general pediatrician, a format which is unique among textbooks in the pediatric subspecialties. Many of the pediatric coauthors are recent graduates of our categorical Pediatrics and Internal Medicine/Pediatrics residencies at Rush. Their contributions provide a fresh and practical viewpoint that reflects their experiences in the hospital and in practice. The result is a text whose content is both authoritative and relevant.

This book proves useful as an accessible resource for teaching the fundamentals of pediatric cardiology, a handy resource for both cardiologists and pediatricians, and a rich trove of illustrative materials. As a pediatric chairman who knows most of the authors personally in their roles as faculty and trainees at Rush Children's Hospital, this book fills me with a sense of scholarly (and fatherly) pride. Its authors have tried to create a useful contribution to the care of children with heart disease and their families. I think they have succeeded admirably.

Chicago, IL — Kenneth M. Boyer, MD

Foreword

Going back to my days when I was a medical student at Jordan University and later as a house officer in pediatrics at Yale New Haven Hospital, it was hard for me to find the proper book for learning everything I needed in pediatric cardiology. Over the last decade or so, the field of pediatric cardiology has evolved causing many pediatric residents to develop great interest in pursuing this specialty. A major part of the reason for this interest is the great advancement in imaging (echocardiography, MRI, CT), interventional cardiology, and electrophysiology. Such advancements contributed to the improved survival of children with congenital cardiac defects.

This book provides a comprehensive review in pediatric cardiology, starting with an approach to heart disease in children and the interpretation of cardiac symptoms. Further, this book provides detailed discussion on how to interpret chest radiographs and the role of echocardiography and catheterization in diagnosing congenital heart disease. The beauty and elegance of this book is the case scenarios discussed in detail in every chapter. Such scenarios teach the reader (be it a student or resident) the flow of the case and how to reach a proper diagnosis.

All forms of congenital cardiac defects are discussed in detail in a systematic fashion, starting with incidence, pathology, pathophysiology, clinical manifestations, laboratory findings, and management. Each defect discussed is followed by case scenario. This format should teach the reader how to think and go about the case.

For the students and practitioners today, the information in this book provides a wealth of practical material, which is invaluable for the current management of congenital heart disease and also provides a systematic approach to each cardiac defect. This book should be a reference for all those who are interested in taking care of patients with congenital heart disease.

Chicago, IL — Ziyad M. Hijazi, MD, MPH, FAAP

Preface

The role of pediatricians in caring for children has become daunting. The ever expanding knowledge in disease processes and the wide and complex therapeutic options available makes keeping up with all nuances of the management of childhood diseases exceedingly difficult. As the subspecialty fields expand, the role of pediatricians change as they work with subspecialists in caring for children with ailments, such as heart diseases. Pediatricians are the primary care providers for children and are entrusted with the discovery of early signs of heart diseases, particularly in the newborn period when presentation is frequently obscure and occasionally with devastating consequences if not discovered and managed promptly.

The issue of how much a pediatrician should know about diseases typically managed by subspecialists is frequently raised. Educators in charge of training pediatric residents as well as regulating bodies providing certification of educational competency to pediatricians continue to emphasis the need for pediatricians to acquire and be considerably proficient in issues relating to heart diseases in children. This is primarily because pediatricians are the frontline practitioners who could identify early signs of heart diseases and are the primary care providers who follow children with ongoing cardiac diseases undergoing medical and surgical management.

Pediatricians are not expected to come up with precise diagnoses of cardiac anomalies in a child; instead, their role is one of identifying the possibility of cardiac anomalies and their potential urgency, or lack of. Furthermore, pediatricians are expected to understand issues relating to ongoing therapy or staged interventional procedures to provide general pediatric care that augments the therapeutic measures underway for the cardiac lesion. Perhaps a good example of the latter includes the knowledge of lesions requiring subacute bacterial endocarditis prophylaxis or the management of a child requiring anticoagulation therapy.

The purpose of this textbook is to provide comprehensive, yet easy to understand details of heart diseases in children. Therefore, the construction of this reference was based upon three principals: Provide comprehensive details of most heart lesions encountered in this field, detail pathophysiological principals of each lesion so as to provide the reader with knowledge that could apply to a wide spectrum of

presentations of the same lesion, and finally illustrate each concept and lesion through case scenarios and images.

The art of teaching is a fascinating process. To be able to convey knowledge in a clear and meaningful way is not always easy. Educators should be well versed in the material they intend to teach; but perhaps more importantly is their ability to gauge what the audience already knows and how to build upon their existing knowledge to what is desired. To achieve this, we have followed a unique model in authoring each chapter. Topics were initially written by a pediatric cardiologist knowledgeable in the issues presented; this was then reshaped by a second author, a pediatrician, to suit the needs of the generalist, rather than the specialist. Each chapter traveled back and forth between specialist and generalist until a satisfactory format was reached providing ample information and packaged to what a pediatrician may need.

Significant effort was made in producing the large sum of illustrations in this book. The heart diagrams depicting various congenital heart diseases were based on a normal heart diagram created by Jeremy Brotherton, a talented medical illustrator. Jeremy crafted a normal heart diagram using a computer-based drawing program, thus allowing me to alter it to depict the various congenital heart disease illustrations in this text. The ECG rhythm strips were generated through a computer drawing program which I designed some time ago and found very useful in showing typical electrocardiographic rhythm strips for teaching purposes. The 12 lead ECG images were of actual patients, however, edited to enhance the pathological features without excessive annotations. The chest X-ray images were enhanced to clarify subtleties of abnormalities of cardiac silhouette or pulmonary vasculature though illustrations inserted over the original chest X-ray image providing clarity and details difficult to do with annotations. Variations of many of the images used in this book were previously used in the pediatric cardiology teaching Web site I constructed at Rush University (http://www.pedcard.rush.edu). The echocardiographic images in this book were limited to those which provide a clear understanding of how echocardiography is used in assessing children with congenital heart diseases. The purpose of these illustrations was to demonstrate the different tools available through this imaging modality. The echocardiographic images were made by Stephen Stone, MD who during an elective at Rush University showed an uncanny understanding of the 3-dimension nature of the heart as depicted through 2-dimension images of echocardiography. Furthermore, his ability to illustrate what echocardiographic images produced is a collection of illustrative images which he used in the chapter he coauthored.

Teaching pediatric cardiology to the noncardiologist is an exciting endeavor which I learned to love from my mentor, Dr. William Strong. I witnessed him during my fellowship at the Medical College of Georgia lecturing medical students the principals of pathophysiology in congenital heart diseases, I was awestricken. Dr. Strong captured their attention from the first word he uttered to the conclusion of his talk when he was always warmly applauded by the medical students who were finally able to put all the basic knowledge they have attained in synch with

the clinical sciences they are striving to learn. Once I became a faculty member, I too embraced his approach of tracing back cardiac symptoms and signs to their pathophysiological origins, thus demystifying clinical presentations and investigative studies of children with heart diseases. I have experienced many masters of education, but non like Bill Strong, a true scientist, thinker, orator, and above all a remarkable teacher to whom I owe much of what I have learned.

Chicago, IL Ra-id Abdulla, MD

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Part I
Approach to Heart Diseases in Children

Chapter 1
Cardiac History and Physical Examination

W. Reid Thompson and Surabhi Mona Mehrotra

Key Facts

- In most instances, history and physical examination provide crucial information when determining if a child has heart disease
- Heart disease should be suspected if history reveals:
  - Shortness of breath without wheezing
  - History of central cyanosis
  - Easy fatigability
  - Failure to thrive
  - Family history of heart disease or sudden cardiac death
- Heart disease should be suspected if physical examination reveals:
  - Central cyanosis, clubbing of digits
  - Poor capillary refill and pulses
  - Delayed and weak femoral pulse when compared to brachial pulse
  - Hyperactive precordium, thrill
  - Murmurs louder than 2/6, diastolic murmurs
  - Single S2, fixed splitting of S2, additional heart sounds

Introduction

The wide application of fetal echocardiography in the United States has changed the most common presenting symptom of the neonate in many centers from cyanosis or tachypnea to "history of abnormal fetal screen." While this may be

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advantageous to those newborns, the skills needed to detect heart disease presenting without a fetal diagnosis, as a direct result, are increasingly in danger of being lost. Detection of previously undiagnosed heart disease in infants and children usually begins with a careful history and physical examination appropriate for the age of the child and the likely diseases that may present at that time. Knowledge of the classic presenting symptoms and signs of heart disease and skill in distinguishing the abnormal from the normal physical exam is crucial for the general pediatrician, and remains the primary screening tool for children of all ages.

Cardiac History

Consideration of heart disease as a possible diagnosis is usually prompted by one of a small list of symptoms or signs, including otherwise unexplained tachypnea, with or without failure to thrive, cyanosis, abnormal heart sounds or murmur, chest pain, or syncope. Congestive heart failure (CHF) due to excessive pulmonary blood flow is characterized by "quiet tachypnea," meaning the patient is not distressed, but is breathing rapidly (>60 breaths/min in the infant). A careful feeding history should be taken to ascertain how many ounces of formula are taken per feeding and per 24-h period, how long the typical feeding takes, whether the feeding is interrupted by frequent stops for breathing and ends with apparent fatigue, and whether it is accompanied by diaphoresis. CHF usually results in decreased formula intake because of tachypnea despite increased caloric demands, resulting in failure to thrive. Anomalous origin of the left coronary, presenting usually between 2 and 4 months, is typically associated with apparent discomfort during feedings. When asking about cyanosis, a distinction should be drawn between peripheral acrocyanosis, involving only the distal extremities, and central cyanosis, expressed as blueness of the lips and mucous membranes. Cyanosis with crying may be a sign of tetralogy of Fallot. However, visible cyanosis requires at least 3 g of desaturated hemoglobin per deciliter of blood, thus is relatively more difficult to detect in infants with lower hemoglobin values (for a given arterial oxygen saturation). Frequent and more serious respiratory illnesses may indicate predisposing cardiac pathology. Swallowing difficulties and/or stridor may be a sign of a vascular ring.

The older child is more likely to have either an occult congenital defect, such as an atrial septal defect, coronary anomaly, cardiomyopathy, or valve disease that was asymptomatic and difficult to detect on physical exam in infancy, or an acquired disease (e.g., myocarditis). The history should include questions about physical activities including exercise-induced chest pain, dizziness or shortness of breath, decreased exertional tolerance, or syncope. Most chest pain that occurs at rest in children is noncardiac, with the exception of myopericarditis. Heart racing or palpitations that occur at rest, with sudden onset and resolution, in a nonanxious youngster may indicate supraventricular tachycardia.

Family history of congenital heart disease or arrhythmia should be determined. History of premature death, sudden or otherwise, or significant disability from

cardiovascular disease in close relatives under 50 years old may put the child or adolescent at increased risk for familial cardiomyopathy or premature atherosclerotic disease. Specific diagnoses should be inquired about, including hypertrophic or dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia, long Q-T syndrome, and Marfan's syndrome.

Cardiac Examination

The comprehensive cardiac examination in the infant or child should begin with a period of observation, prior to interacting with the patient. Note the respiratory rate and pattern, whether or not accessory muscles are being used or flaring is present (usually more consistent with pulmonary disease or airway obstruction), and what degree of distress the patient is in. Note also the general nutritional status, the color of the mucous membranes, the presence of clubbing of digits (Fig. 1.1a, b), and the peripheral perfusion. Also take note of any specific dysmorphic features that might be associated with known syndromes. Next, carefully assess the vital signs and compare with age appropriate normal data, in the context of the potentially anxiety-provoking examination experience. Blood pressures should be obtained in all four extremities with appropriate size cuffs (Fig. 1.2 a, b). Pulse oximetry should be performed in every newborn and, if ductal dependent left-heart obstruction is possible, upper and lower extremity pulse oximetry should be compared. Lungs should be auscultated, listening for wheezing or rales, though these findings are relatively uncommon in infants with CHF. Also take note of any stridor, especially with crying, that may indicate a vascular ring. The abdominal exam should include careful assessment of the liver position and distance of the edge relative to the costal margin. Hepatomegaly is a reliable feature of significant heart failure in infants (Fig. 1.3). A more central liver edge is found in heterotaxy syndrome. Peripheral edema is relatively uncommon in pulmonary over-circulation lesions (e.g., VSD), and in infants in general. Likewise, jugular venous distention is not usually appreciated in infants.

Cardiac auscultation begins with a general assessment of the chest, looking for signs of hyperdynamic precordium. When palpating the chest, check for the apical impulse which represents the left ventricular (LV) impulse. LV impulse is at the apex of the heart, approximately in the fourth or fifth intercostal space at the midclavicular line. In addition, the right ventricular (RV) impulse can be elicited at the right lower sternal border (Fig. 1.4). Decrease or increase in either or both LV and RV impulse may indicate hypoplasia or hypertrophy (respectively) of that ventricle. Palpation of the chest may reveal the presence of a lift or heave of increased right ventricular pressure or thrill associated with a grade 4 or higher murmur. Use the appropriate stethoscope for the patient's size and listen systematically to each part of the cardiac cycle and at each area on the chest. Start by listening to the first and second heart sounds. S1 is best heard at the apex and marks the beginning of systole, whereas S2 is best heard at the mid to upper sternal border

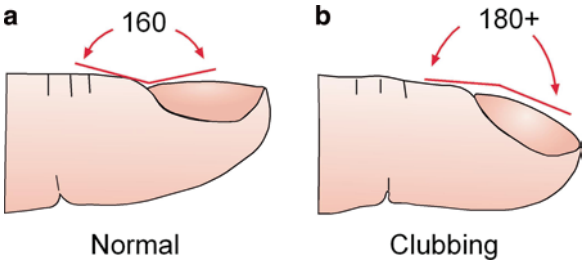

Fig. 1.1 Clubbing. (**a**) The angle at the junction of skin with the nail at the dorsal surface of digits is normally around 160°. (**b**) In children with cyanotic congenital heart diseases this angle becomes wider and may exceed 180°. This is the result of hypoxia in peripheral tissue, which causes the opening of normally collapsed capillaries to better perfuse the hypoxic tissue. Perfusion of these collapsed capillaries will result in expansion of the volume of these peripheral tissues (tips of digits) resulting in clubbing. This phenomenon is seen in other lesions causing hypoxia of peripheral tissue, such as with chronic lung disease and chronic anemia (causing hypoxia through reduction of level of hemoglobin and therefore reduction of oxygen carrying capacity) such as with ulcerative colitis, Crohn's disease, and chronic liver disease

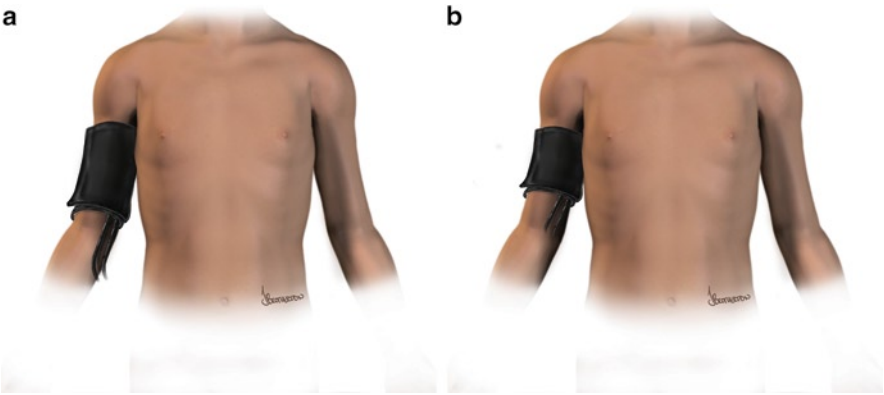

Fig. 1.2 Appropriate size BP cuff. Cuffs used to measure blood pressure should be appropriate in size for the child. (**a**) The breadth of the cuff should extend 2/3rd to 3/4th the distance of the forearm (or leg). (**b**) Smaller blood pressure cuffs will cause falsely elevated blood pressure measurements

and marks the beginning of diastole. By identifying S1 and S2, the systolic versus diastolic intervals can likewise then be distinguished, even though they may be of equal duration (at higher heart rates). In the case of mesocardia or dextrocardia, the apical impulse will be displaced rightward.

S1 is usually single, though in reality is the result of multiple low frequency events, which can often have at least two detectable components ("split S1"). This normal finding is relatively common in older children or adolescents, and is

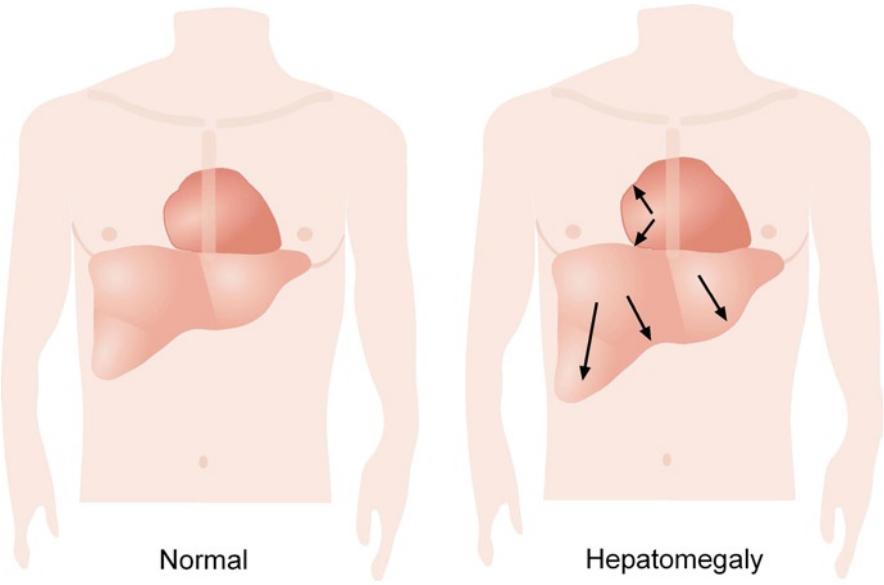

Fig. 1.3 Hepatomegaly. Increased blood flow in the right heart such as seen in patients with atrial or ventricular septal defects will cause dilation and increase in right atrial pressure. This will eventually lead to congestion of organs draining blood into the right atrium such as the liver, leading to its enlargement

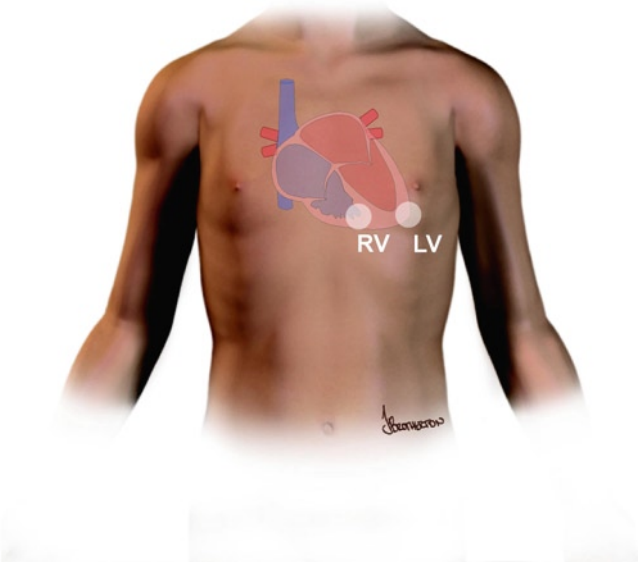

Fig. 1.4 RV and LV impulses. The left ventricular (LV) or apical impulse reflects the left ventricular apex, while the right ventricular (RV) impulse reflects the right ventricular apex. LV impulse is normally the left lateral most border of the heart, while the RV impulse is at the left lower sternal border. RV impulse is close in intensity to the LV impulse in the neonatal period. Later in life, the LV impulse is much more prominent

best heard at the left lower sternal border. This is sometimes mistaken for the presence of an early systolic ejection sound or "click," though a click is usually somewhat higher in frequency and intensity, slightly later in timing, and is well heard at the apex, where the split S1 is usually not heard. S1 may be softer than normal with first degree A–V block (long P–R interval on ECG), or variable in intensity with complete A–V block (A–V dissociation with slower ventricular rate on ECG). A louder S1 may be heard with a short P–R interval (W–P–W syndrome). These changes are due to the alteration in the time period blood can flow from the atria to the ventricles. A short P–R interval indicates less time between atrial and ventricular contraction, whereas a prolonged period P–R interval allows more time for blood to flow from atria to ventricles prior to ventricular contraction and closure of AV valves.

S2 is an important event to characterize in children, as it may be the only abnormal finding indicating serious pathology. S2 is best heard at the left mid to upper sternal border. Once pulmonary vascular resistance (PVR) has fallen and the pulmonary artery pressure has reached its normal levels, the second heard sound should exhibit "physiologic" splitting, becoming wider with inspiration as P2 lags behind A2 (Fig. 1.5), then narrower with expiration, and P2 intensity (reflecting pulmonary artery pressure) should be softer than A2 (reflecting systemic arterial pressure). The interval should close with expiration, at least in the sitting position, though may occasionally remain slightly split when supine, sometimes reflecting an incomplete right bundle branch block (normal variant). Wide, fixed splitting of S2 is a sign of right heart volume overload from an atrial septal defect or anomalous pulmonary return. A narrowly split (or single) S2, with increased intensity of P2 component is an important sign of pulmonary hypertension. Paradoxical splitting of S2 (widening of the interval with expiration, and closing with inspiration) is due to delayed closure of the aortic valve (A2) and is often found in aortic stenosis or left bundle branch block.

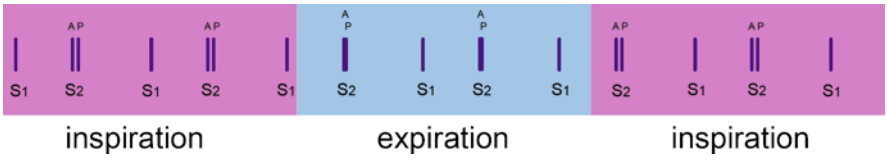

Fig. 1.5 Normal splitting of S2 with respiration. The first heart sound is typically single, reflecting closure of the tricuspid and mitral valves and occurs at the onset of systole. S2 is normally split, consisting of closure of the aortic valve, followed by the pulmonary valve. The aortic valve closes first due to the shorter left bundle branch of the His conduction system. This will allow the left ventricle to contract a few milliseconds before the right ventricle and therefore complete systole a few milliseconds before the right ventricle, hence aortic valve closes before pulmonary valve. This phenomenon is exaggerated during inspiration due to the increase in blood return to the right heart secondary to the sump effect of a negative intrathoracic pressure, thus leading to wider splitting of the second heart sound. The opposite is true in expiration, leading to approximation of the aortic and pulmonary components of the second heart sound, thus sounding as a single heart sound