SEVENTH EDITION

Roberts and Hedges'

CLINICAL PROCEDURES

in Emergency Medicine and Acute Care

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SEVENTH EDITION

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Dedicated to Lydia, Matthew, Martha, Eleanor, and Liam.

J.R.R.

To my son, Nicholas, and to the memory of my daughter, Lauren, who have taught me that the most important things in life are not found in a book. To my brother, E. Douglas Bomberger, PhD, whose teaching, research, writing, and editing in Historical Musicology inspire me and many others.

C.B.C.

To Jim and Cathy for including me again on this edition. It is an honor and privilege to work with you. To Gary Setnik for all that you have done for me, and for emergency medicine, throughout your career. To my parents, Alfred and Beverly Thomsen, for everything. And to Cristine, Henry, and Cole for your support and understanding at home while this project took me away from you.

T.W.T.

I hope to dedicate this edition to the first generation of emergency physicians who helped to cultivate our specialty. I would also like to thank my family, especially my parents; sisters; wife, Karen; and children, Sydney, William, and Nathan, who were so patient and endured so much while this field was developing so rapidly. Most importantly, may this book be a useful reminder to all who practice emergency medicine in all its variations that this science is constantly evolving and the search for excellence in training, education, and service is its own reward.

A.S.C

I dedicate the 7th edition of this book to what I hold most dear … my family. To my beautiful wife and best friend, Marcy, who binds us all together. And to my children, Adam, who protects our freedom; Arielle, who heals the sick; and Allison, who shapes young minds … I am a very fortunate man indeed.

C.R.C.

To Karen, Joshua, and Zachary—thanks for your inspiration and patience that has allowed me to pursue this educational passion. To my peers, residents, students, nursing staff, and patients who champion a mission to provide care for all.

P.M.C.D.

To my wife, Sejal, and my three children, Nikhil, Eleena, and Kamran, for giving me purpose and inspiration. To my colleagues and my mentors for all that they have taught me through the years. To Jim Roberts for continuing to be a driving force behind this text. And to emergency physicians around the world who continually care and advocate for their patients despite the toughest of times and circumstances.

A.M.

To Joyce, Barry, Eunice, and Moses. To Jim and Cathy. To the residents, faculty, nurses, and patients of the Los Angeles County/USC Medical Center.

S.P.S.

I would like to thank my wife, Erika, and my wonderful children, Hayden, Emma, Taylor, and Olivia, for their selfless love, support, and encouragement. You are my world. A special thanks also to the faculty and residents in the Department of Emergency Medicine at the University of Maryland. It is humbling to work with such amazing and talented physicians each day.

M.E.W.

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HOW THIS MEDICAL TEXTBOOK SHOULD BE VIEWED BY THE PRACTICING CLINICIAN AND THE JUDICIAL SYSTEM

The editors and authors of this textbook strongly believe that the complex practice of medicine, the vagaries of human diseases, the unpredictability of pathologic conditions, and the functions, dysfunctions, and responses of the human body cannot be rigorously defined, explained, or rigidly categorized by any written document. *Therefore it is neither the purpose nor the intent of our textbook to serve as an authoritative source on any medical condition, treatment plan, or clinical intervention; nor should our textbook be used to rigorously define a standard of care that should be practiced by all clinicians.*

Our written word provides the physician with a literature-referenced database and a reasonable clinical guide that is combined with practical suggestions from individual experienced practitioners. Some of the content is merely personal opinion of the authors. We offer a general reference source and clinical roadmap on a variety of conditions and procedures that may confront clinicians who are experienced in emergency medicine and critical care practice. This text cannot replace physician judgment; cannot possibly describe every possible aberration, nuance, clinical scenario, or presentation; and cannot define unwavering standards for clinical actions or procedures. *Every medical encounter must be individualized, and every patient must be approached on a case-by-case basis.* No complex medical interaction can possibly be reduced to the written word. *In addition, the treatments, procedures, and medical conditions described in this textbook do not constitute the total expertise or knowledge base expected to be possessed by all clinicians. Just because a certain procedure or technique is discussed, this does not mean that every clinician should be skilled in it, or even consider including it in their practice.*

Finally, many of the described complications and adverse outcomes associated with implementing or withholding complex medical and surgical interventions may occur, even when every aspect of the intervention has been performed correctly and as per any textbook or currently accepted standards.

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Preface

The seventh edition of *Roberts and Hedges' Clinical Procedures in Emergency Medicine and Acute Care* continues the book's original concept of providing complete, very detailed, and up-to-date descriptions of many common, and some uncommon, procedures encountered during emergency medicine and acute care practice. The novice may find the discussions and figures devoted to the many procedures somewhat daunting or overwhelming at first; but it is hoped that most will eventually appreciate the simple discussion and complex verbiage contained in the text. The goal is to describe clinical procedures—from simple Steri-Strip application, to loop drainage of an abscess, to tracheal intubation and mechanical ventilation, to skull trephination—as though each were the nascent clinician's first exposure to the concept, but with a depth and attention to detail that the seasoned operator would also deem helpful.

In previous editions it was difficult to find figures or photographs that conveyed the details or elucidated the vagaries to the extent one might want. The newly added color photographs, mostly digital quality, and a cornucopia of additional figures were a much needed update and morphed this edition into an obvious improvement over previous iterations. To make the text more user friendly, procedure boxes have been created, comprising a mini-atlas that allows the clinician to see the entire procedure at a glance. One can even bring the text to the bedside, viewing a single page of sequential images, the quintessential teaching tool for house staff and students. Many of the photographs were taken by me over 42 years of emergency department shifts or created or supplied by Todd W. Thomsen, MD. Some illustrations were borrowed from other sources, such as the wonderful text by Catherine B. Custalow, MD, PhD. This edition has more than 3500 images, half of which are new. More than 70 percent of the new images are the result of the artistic genius of graphics editor Dr. Thomsen. Frank Netter, watch out for Dr. Thomsen; he is rapidly attaining your status and may have already surpassed it in emergency medicine parlance. No doubt Dr. Thomsen has found his calling, blending amazing original art and electronic and digital prowess with equally impressive clinical medicine expertise. Please see Expert Consult for some bonus procedure videos from Todd.

The addition of the ultrasound-guided sections, presented in easily found and readily deciphered boxes, is the result of a gargantuan effort from our new ultrasound editor, Christine Butts, MD, an ultrasonographer extraordinaire. Another achievement of this edition is the inclusion of additional video procedures. Only wished for in other texts, many sections now reference online content that allows the reader to view videos of the procedures actually being performed. "See one, do one, teach one" has taken on new meaning with this text. This edition is now available electronically on such devices as the Kindle and iPad and is still fully searchable online at expertconsult.com.

There are, of course, many ways to approach any patient or any procedure, so this text is not a dictum and is not truly authoritative. This book does not attempt to define standard of care. The clinician should remember these caveats if involved in a medicolegal scenario. It contains practical hints and successful tactics gleaned from the literature and by years of practice, adeptly described by skilled clinicians. As with previous editions, this version also significantly incorporates the personal opinions and experience of the authors and editors. But this text is simply a clinical guide, not a legal document. Do not reference this book if you testify in court, for either the defense or the plaintiff. Today's dogma too often becomes tomorrow's heresy, and physician hubris is worse than incompetence. Simply stated, emergency medicine and acute care, and the human body too often defy the written word, personal opinion, or local custom and humble even the venerable and the universally praised gray-haired professor.

Many new authors have been added, as well as a number of new concepts and approaches. All procedures have been tweaked. As an example one will not find the novel loop abscess drainage technique, sedation techniques, or ENT and ophthalmology techniques so nicely described elsewhere.

My personal thanks are hereby conveyed to those who contributed to previous editions. The updated chapters often merely refine or further manipulate the scholarly work of others who originally assisted us. The current contributors include an enviable blend of friends and colleagues, some former students of mine, up-and-coming rising stars in their own right, and my prior mentors and role models—all are accomplished physicians and leaders in their own milieu. Many of the editors' names are well known to anyone who reads the literature or attends a continuing medical education activity. All of the associate editors portray and embody the pinnacle of emergency medicine and acute care excellence. Most of the contributors, and all the associate editors, probably know more than I know, and most are likely infinitely more capable and facile with procedures. All are capable of writing a text themselves, and some have already done so; however, some are now enlightened and eschew that primal urge because they now know how difficult it is to write even a single chapter. My able and erudite associate editors, all from prestigious academic teaching programs in emergency medicine, are Arjun S. Chanmugam, MD, MBA; Carl R. Chudnofsky, MD, FACEP; Peter M.C. DeBlieux, MD; Amal Mattu, MD; Stuart P. Swadron, MD, FRCPC, and recently added Michael Winters, MD, FACEP, FAAEM. They provided the bulk of the original editing, but senior editor, Dr. Custalow, read every single word and reviewed every table and figure. Dr. Custalow is a more tenacious editor than the proverbial honey badger in regard to dealing with details, grammar, organization, and style. In the end, my personal bias may be evident, but Dr. Custalow was the fire and fuel for the book's framework. As already stated, Dr. Thomsen made the text come to life with images. I would also like to thank Robert Orman, MD, and Scott D. Weingart, MD, FCCM, who served as video editors on the previous edition.

If any of our editing changed, altered, or misinterpreted the original thoughts of the contributors (and I know in some instances it must have), we apologize; but hard decisions had to be made, and waffling was rarely an option. Our book simply tells you what to do and how and when to do it, but no book can always fit every individual situation. We attempted to squarely address such omnipresent vague topics as prophylactic antibiotics, local customs, and variations in style, and accepted the fact that not all foreign bodies or tendon lacerations will be identified in the heat of the moment by even the most skilled clinician. The prescient and sagacious clinician knows that the ability to practice medicine from a book is limited, and one learns best from past experiences; and, for certain, the most instructive past experience is one that was not always textbook perfect.

James R. Roberts, MD, FACEP, FAAEM, FACMT

Foreword

Every shift in the emergency department has the potential to take us into the great unknown. Frequently a patient's presentation challenges our capabilities, and an uncommon procedure may be the only answer. Throughout my training and career, this book you are now holding has been my unwavering companion.

On a recent night shift, I encountered a patient needing two separate procedures, both well within my capabilities as an emergency physician. Yet, I found myself asking, "Do I remember exactly how to do the procedure? What is the best approach? Where are my landmarks?" I was almost positive I could recall the technique correctly, but before breaking skin, I decided a review of *Roberts and Hedges* was in order. Taking this step dispels any doubt and gives me confidence to get the job done well.

Whether it's tapping a septic elbow, checking a compartment pressure, or performing a lateral canthotomy, the refrain is always the same: "How, exactly, do I do this?" becomes "Right, oh yes, I've got this down!"

As we move further into the world of online and on-demand education, some may say that textbooks are a vestige of the past, but I disagree in this instance. Even after decades in print, *Roberts and Hedges* continues to shine as the guiding light of clinical emergency medicine.

Rob Orman, MD
June 1, 2017

Acknowledgments

Gargantuan efforts, clairvoyant and perceptive suggestions, and decidedly prescient and sagacious contributions of many individuals have brought this work to fruition. Not the least of whom were the individual authors who toiled over tedious manuscripts and answered countless queries about the vagaries and vicissitudes of seemingly straightforward clinical procedures.

This book's current edition was championed by Elsevier's Senior Content Strategist, Kate Dimock. All of the initially submitted work was culled, corrected, and collated by the very able Senior Content Development Specialist, Jennifer Ehlers. Senior Project Manager, Rachel McMullen, also toiled over every aspect of the text. My sincere gratitude to them is warmly extended with this acknowledgment. If any reader is contemplating developing their own textbook, snag this team of publishing aficionados if you can.

Of course, the entire work was infused with vim and vigor from Catherine B. Custalow, MD, PhD, and every image was created, beautified, or otherwise superbly orchestrated by Todd W. Thomsen, MD. The final editing of Arjun S. Chanmugam, MD, MBA; Carl R. Chudnofsky, MD, FACEP; Peter M.C. DeBlieux, MD; Amal Mattu, MD; and Stuart P. Swadron, MD, FRCPC, and Michael Winters, MD, FACEP, FAAEM, completed the task. One might think that these folks have a lot of free time on their hands or, more likely, they burned gallons of midnight oil for the project. It has been an honor to be associated with such icons in the field. The contributing authors are certainly also to be congratulated on a stellar performance.

Thank you all for accomplishing a goal that was once thought, even by me, to be nothing more than a seemingly good idea, but a task too difficult to even contemplate, let alone wantonly attempt. It is hard to believe that the first edition of this book was done in 1985 on an IBM electronic typewriter, before email, scanning, fax, or Microsoft Word. We have come a long way for sure.

James R. Roberts, MD, FACEP, FAAEM, FACMT

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SECTION I

Vital Signs and Patient Monitoring Techniques

CHAPTER 1

Vital Signs Measurement

Jillian L. McGrath and Daniel J. Bachmann

Measuring the temperature, pulse, respiratory rate (RR), blood pressure, and pulse oximetry is generally recommended for all emergency department (ED) patients, in addition to assessment of pain in the appropriate patient population. For very minor problems or for some fast-track patients (e.g., suture removal), a full set of vital signs may not be required, and this is best decided on a case-by-case basis rather than by strict protocol. Vital signs may indicate the severity of illness and also dictate the urgency of intervention. Although a single set of abnormal values suggests pathology, findings on triage or the initial vital signs may be spurious and simply related to stress, anxiety, pain, or fear. It would be incorrect and not standard of care to attribute initial triage blood pressure, RR, or pulse rate to specific pathology or to retrospectively assume that diagnostic or treatment interventions should have been initiated based solely on these readings. The greatest utility of vital signs is in their continued observation and trends over time. Deteriorating vital signs are an important indicator of a compromised physiologic condition, and improving values provide reassurance that the patient may be responding to therapy. When a patient undergoes treatment over an extended period, it is essential that the vital signs be repeated as appropriate to the clinical scenario, particularly those that were previously abnormal. In some clinical circumstances, it is advisable to monitor the vital signs continuously.[1]

Vital signs should be measured and recorded at intervals as dictated by clinical judgment, the patient's clinical state, or after any significant change in these parameters. Adhering to strict protocols or disease categories is not useful or productive. An abnormal vital sign may constitute the patient's entire complaint, as in a febrile infant, or it may be the only indication of the potential for serious illness, as in a patient with resting tachycardia.[2]

Emergency medical service (EMS) personnel begin assessment of the patient's status and vital signs in the prehospital setting. Surges of epinephrine and norepinephrine commonly occur during transport by the EMS, and these catecholamines are known to alter vital signs and lead to increases greater than 10% in the heart rate.[3] Vagal influences may also influence EMS-derived vital signs. Prehospital vital signs should always be interpreted with the entire clinical scenario in perspective.

Blood pressure and pulse are frequently evaluated together as a measure of blood volume. Capillary refill is discussed as an assessment of overall perfusion, circulatory volume, and blood pressure. Although body temperature is usually the last vital sign measured during resuscitation, it has special importance for patients suffering from thermal regulatory failure. With these considerations in mind, the current chapter is organized according to the priorities of patient resuscitation and evaluation. Assessment of pain as a vital sign is gaining acceptance and is discussed briefly at the end of this chapter.

Background
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NORMAL VALUES

The range of normal resting vital signs for specific age groups must be appreciated by the clinician to enable identification of abnormal values and their clinical significance. The normal ranges for vital signs are also influenced by gender, race, pregnancy, and residence in an industrialized nation. These ranges have not been validated in ED patients, who have many reasons for abnormalities in vital signs, including anxiety, pain, and altered physiology as a result of their disease states. Ranges of normal vital signs, commonly quoted as normal or abnormal in other settings, serve only as a guide and not an absolute criterion for diagnosis, treatment, further observation, or intervention in the ED.

Published vital sign norms for children are not as well accepted as those for adult patients. Table 1.1 and Table 1.2 report heart rate and RRs by age grouping and percentile for children from birth to 18 years of age. This data represents a large cross-sectional study using 6 months of nurse-documented heart rates and RRs from the electronic records of 14,014 children on general medical and surgical wards at two tertiary-care children's hospitals. Up to 54% of heart rate observations and up to 40% of RR observations in this sample were outside textbook reference ranges.[8] During the newborn period, normal arterial blood pressure rises rapidly. Values for pulse and respiration in children older than 3 years reflect an average of male and female values for 0- to 1-, 3-, 9-, and 16-year-old populations. The values for blood pressure reflect an average of male and female values for the 1- to 6-month-old and the 3-, 9-, and 16-year-old populations.[9] Other studies have assessed the reference values for RR in children demonstrating the same variability of pediatric "normal" vital sign ranges.[10–12]

BACKGROUND

Early recognition of vital signs dates to the fourth century BC, when Herophilus first described sphygmology, or palpation of the pulse in terms of size, frequency, force, and rhythm. Chinese clinicians (second century BC) timed the pulse by the RR of the examiner in the belief that four pulsations per respiration was normal for adults. The study of pulses was greatly influenced by Galen, who expanded the subject into a rather complex and obscure art form and wrote 18 books on the subject.[4]

Blood pressure was first measured directly in 1733 by Hales, who recorded arterial pressure in a mare by cannulation with a brass pipe and a blood-filled glass column.[5] Frank used large-bore catheters connected to a rubber membrane in a 1903 manometer.[6] The invention of inflatable cuff manometers (Riva-Rocci, 1896) and discovery of the arterial phase sounds (Korotkoff, 1905) allowed the development of indirect measurement of blood pressure.[5,6]

Clinical thermometry was introduced by Sanctorius in 1625, with early thermometers being filled with alcohol. Mercury column thermometers were introduced by Fahrenheit in 1714. Although their routine use was supported by Boerhaave, thermometry was not established as routine clinical practice until the 1870s.[7]

TABLE 1.1 Pediatric Heart Rate Limits Based on Age Group and Percentile

AGE GROUP	5TH PERCENTILE	50TH PERCENTILE	95TH PERCENTILE
0–3 months	113	140	171
3–6 months	108	135	167
6–9 months	104	131	163
9–12 months	101	128	160
12–18 months	97	124	157
18–24 months	92	120	154
2–3 years	87	115	150
3–4 years	82	111	146
4–6 years	77	106	142
6–8 years	71	100	137
8–12 years	66	94	129
12–15 years	61	87	121
15–18 years	57	82	115

Adapted from Table 3 of Bonafide CP, Brady PW, Keren R, et al: Development of heart and respiratory rate percentile curves for hospitalized children, *Pediatrics* 131(4):e1150–e1157, 2013.

TABLE 1.2 Pediatric Respiratory Rate Limits Based on Age Group and Percentile

AGE GROUP	5TH PERCENTILE	50TH PERCENTILE	95TH PERCENTILE
0–3 months	27	41	62
3–6 months	25	38	58
6–9 months	23	35	54
9–12 months	22	33	51
12–18 months	21	31	48
18–24 months	20	29	45
2–3 years	18	27	42
3–4 years	18	25	40
4–6 years	17	24	37
6–8 years	16	23	35
8–12 years	15	21	31
12–15 years	13	19	28
15–18 years	13	18	26

Adapted from Table 4 of Bonafide CP, Brady PW, Keren R, et al: Development of heart and respiratory rate percentile curves for hospitalized children, *Pediatrics* 131(4):e1150–e1157, 2013.

For the adult population, normal blood pressure values have been better established. Although systolic blood pressure increases with age, *normotensive* or *normal systolic blood pressure* is defined as 90 to 140 mm Hg, and normotensive or *normal diastolic blood pressure* is defined as 60 to 90 mm Hg. The recent literature suggests defining an "optimal" blood pressure as 115/75 mm Hg because values at or below this level have been associated with minimal vascular mortality.[13] It has been suggested that the definition of hypertension be further expanded to integrate a global cardiovascular risk assessment.[14,15] Although most patients have similar blood pressure in both arms, Pesola and colleagues found that 18% of their hypertensive population[16] and 15% of their normotensive population had a difference of greater than 10 mm Hg in systolic blood pressure between arms.[17]

Within the adult population, optimal definitions for normal systolic blood pressure probably vary with age, and particular differentiation should be made in regard to geriatric patients in the emergency setting. The recent literature suggests redefining values representative of hypotension in the elderly, especially in the setting of trauma. Systolic blood pressure readings ranging from approximately 90 to 120 mm Hg have been associated with occult hypoperfusion and increased mortality in geriatric trauma patients.[18–20]

In 1928 the New York Heart Association, by consensus, established the normal limits for the resting heart rate as 60 beats/min and 100 beats/min. More recent data indicate that 45 beats/min and 95 beats/min may better define the heart rate limits of normal sinus rhythm in adults of all ages. Spodick and colleagues recommended that the operational definition for the limits of the resting heart rate in adults should be 50 beats/min and 90 beats/min.[21,22] This view is widely supported among cardiologists,[23–25] but these ranges have not been validated in the ED setting. There is currently no consensus on what constitutes a normal adult RR; however, an RR range of 12 to 24 breaths/min is generally accepted in the existing literature as the norm for adults.[26,27]

Pregnancy results in alterations in the normal adult values of pulse and blood pressure. Pregnancy is characterized by significant increases in minute ventilation, and is thought to be due to the combined facilitatory effects of progesterone and estrogen on central and peripheral chemoreflex drives to breathe.[28] The resting pulse rate increases throughout pregnancy from 10% to 15% over baseline values. The norms for systolic and diastolic blood pressure are dependent on patient positioning. When a pregnant patient is sitting or standing, systolic pressure is essentially unchanged. Diastolic pressure declines until approximately 28 weeks' gestation, at which time it begins to rise to nonpregnant levels. When a pregnant patient is in the lateral decubitus position, both systolic and diastolic pressure decline until the 28th week and then begin to rise to nonpregnant levels (Table 1.3).[29]

RESPIRATION

Breathing is initiated and primarily controlled in the medullary respiratory center of the brainstem. The respiratory center is modulated by the pneumotaxic center, which limits the length of the inspiratory signal and greatly influences the RR and apneustic center in the pons.[30] Respiratory frequency reveals only a glimpse of the entire clinical picture. The pattern, effort, and volume of respiration may be more indicative of altered respiratory physiology. An abnormality in respiration may be

TABLE 1.3 Vital Signs During Pregnancy in the Lateral Decubitus Position (Means ± SD)

	Trimester		
PARAMETER	1ST	2ND	3RD
Pulse rate (beats/min)	77 ± 2	85 ± 2	88 ± 2
Systolic BP (mm Hg)	98 ± 2	91 ± 2	95 ± 2
Diastolic BP (mm Hg)	53 ± 2	49 ± 2	50 ± 2

BP, Blood pressure; *SD*, standard deviation.
Adapted from Katz R, Karliner JS, Resnik R: Effects of a natural volume overload state (pregnancy) on left ventricular performance in normal human subjects, *Circulation* 58:434, 1978. By permission of the American Heart Association.

a primary complaint or a manifestation of other systemic diseases. Increased RRs may be seen in patients with a variety of pulmonary or cardiac diseases, and acidosis, anemia, temperature, stress, and drugs (such as stimulants and salicylates) can significantly alter the RR in the absence of cardiopulmonary dysfunction.

Indications and Contraindications

The only contraindications to careful measurement of RR are the scenarios of respiratory distress, apnea, and upper airway obstruction, which require immediate therapeutic intervention. RR and respiratory effort should be assessed as soon as patient care demands allow.

The respiratory status of both adults and children plays a crucial role in determining the overall assessment of illness. Although it is a sensitive yet nonspecific indicator of respiratory dysfunction, the RR can also predict nonpulmonary morbidity. Several prehospital and hospital-based illness or injury severity scores feature the RR as a cardinal value. A prehospital RR of less than 10 breaths/min or greater than 29 breaths/min is associated with major injury in 73% of children.[31] Using tachypnea alone as a predictor of pulmonary pathology, infants with an RR higher than 60 breaths/min are found to be hypoxic 80% of the time.[32] Pediatric studies have linked abnormal RRs to in-hospital mortality and the level of care required in the ED.[33,34] In a retrospective study exploring predictors of critical care admission for adult ED patients who were initially triaged as having low to moderate acuity, an abnormal RR at the first nursing assessment increased the odds of critical care admission by a factor of 1.66.[35] An RR higher than 25 breaths/min in prehospital trauma patients was associated with increased mortality.[36] Pre-arrest respiratory insufficiency (RR >36 breaths/min or pulse oximetry <90%) was an independent predictor of mortality (odds ratio [OR], 4.2) in patients with EMS-witnessed cardiac arrest.[37] Although some studies have associated abnormal RRs in adult ED patients with increased mortality,[38,39] a 2011 large prospective cohort study of adult patients found that an initial abnormal RR on triage in the ED was not an independent predictor of hospital mortality.[40]

Procedure

To measure RR (inspirations per minute), count the respirations when the patient is unaware that his or her breathing is being observed. Count for a full minute to most accurately determine the RR. The frequency of breathing is less regular than the pulse, and inaccurate measurement is more likely to occur if the count is taken for a shorter interval. It is common to measure respirations over 15 seconds and multiply by 4, but this can significantly alter the true RR per minute. An infant's RR can easily be determined by observing or palpating the excursion of the chest or the abdominal wall.[41] Infants should be observed for grunting respirations, which are produced by expiration against a partly closed glottis (an attempt to maintain positive airway pressure).

Interpretation

Respiratory Rate

The reproducibility of RR measurements may be limited by significant interobserver variability.[42,43] Clinicians should recognize this inherent variability and interpret the RR with caution. Rates obtained by nurses versus medical students varied significantly, as did those obtained by medical students versus residents and attending clinicians.[44] Interobserver variability may account for a difference of up to 6 breaths/min, and variability in the same observer may account for up to 5 breaths/min.[44] A study comparing RRs obtained by triage nurses with an electronic monitor found that neither provided an accurate measurement of the RR in the ED, suggesting that new clinical strategies for obtaining this vital sign may be necessary.[45] RR is an independent risk marker for in-hospital mortality in community-acquired pneumonia and should be measured when patients are admitted to the hospital with pneumonia and other acute conditions.[46]

Current texts vary considerably in their definition of a normal RR and cite published values that range from 8 to 24 breaths/min. In a study that specifically investigated normal RRs in an ED (afebrile ambulatory patients without respiratory complaints), females had a mean RR of 20.9 breaths/min and males had a mean RR of 19.4 breaths/min. The researchers concluded that a normal RR in the adult patient population was 16 to 24 breaths/min.[44] Other studies have provided additional information on normal resting and sleeping RRs in children younger than 7 years.[9–14] RRs obtained with a stethoscope were higher than those obtained by observation (mean difference, 2.6 breaths/min in awake and 1.8 breaths/min in asleep children). Smoothed percentile curves demonstrated a larger dispersion at birth (5th percentile, 34 breaths/min; 95th percentile, 68 breaths/min), whereas dispersion was less at 36 months of age (5th percentile, 18 breaths/min; 95th percentile, 30 breaths/min).

The RR will generally increase in the presence of fever. It is often difficult to determine whether tachypnea is a primary finding or is simply associated with hyperpyrexia. A study of children younger than 2 years in whom pneumonia was subsequently diagnosed found that age-appropriate limits for resting tachypnea in the presence of fever could be defined. A sensitivity of 74% and specificity of 77% for pneumonia were found when children 6 months of age had an RR higher than 59 breaths/min, when those aged 6 to 11 months had an RR higher than 52 breaths/min, and when those 1 to 2 years old had an RR higher than 42 breaths/min.[47] Even in the face of physiologic compensation for fever, interpretation of the RR alone can help predict the presence of pulmonary disease.

Respiratory Pattern and Amplitude

Hyperventilation and hypoventilation can result from an extensive variety of disorders and may be related to pulmonary

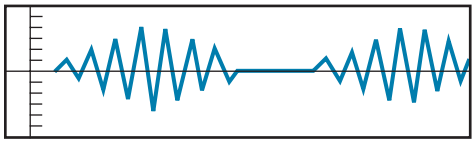
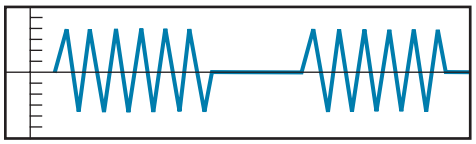
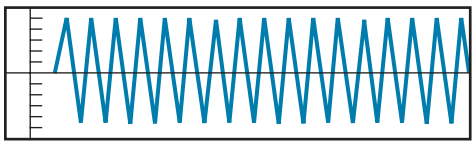
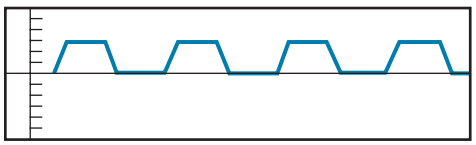
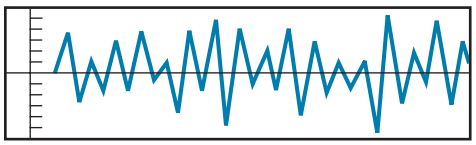
Cheyne-Stokes Respiration	 V_T with a progressive shallow-deep-shallow cycle (30 s–2 min)	CVA, trauma, masses involving a diffuse area of the forebrain, carbon monoxide poisoning, metabolic encephalopathy, altitude sickness, opiate effect
Biot's (Cluster) Respiration	 Periods of rapid respirations of nearly equal depth or V_T followed by regular periods of apnea	CVA or trauma involving the medulla or lower pons, mass effect on the medulla secondary to uncal or tentorial herniation, opiate effect
Kussmaul Respiration/ Hyperpnea	 Hyperventilation characterized by a consistent and deep respiratory pattern; the RR of patients with hyperpnea may be normal or rapid with increased V_T	Severe metabolic acidosis; Kussmaul respiration in diabetic ketoacidosis starts with a rapid and shallow pattern and later becomes rapid and deep
Apneustic Respiration	 Prolonged inspiratory phase followed by prolonged expiratory phases believed to be apneic phases	CVA, trauma, or mass effect involving the pons
Ataxic Respiration	 Erratic breathing pattern with irregular pauses and increasing episodes of apnea (progresses to agonal respiration)	CVA or trauma involving the medulla (indicates a very poor prognosis)

Figure 1.1 Abnormal respiratory patterns. *CVA*, Cerebrovascular accident; *RR*, respiratory rate; *V_T*, tidal volume. (Modified from D'Urbano J: Breathing Patterns, Breath Sounds, 2011. Available at http://www.BreathSounds.org.)

or extrapulmonary pathology. Abnormal respiratory patterns can be characteristic of metabolic or central nervous system pathologic conditions (Fig. 1.1) and may aid in the differential diagnosis. Kussmaul respirations describe the hyperventilation pattern seen in diabetic patients with ketoacidosis. Decreased RR is commonly seen with opiate toxicity. Hyperpnea, or a normal RR but clinically significant hyperventilation secondary to increased tidal volume, may be seen with salicylate poisoning.[48]

Recognition of subtle tachypnea can be difficult in the emergency setting, although it can be the solitary indicator of disease. Another instance of pathology that can confuse routine measurement of the RR is diaphragmatic breathing or retractions. The variability in counting respiratory effort versus effective respirations is not generally appreciated in a single recorded value.

Observe the respiratory patterns carefully in children. In infants, it is essential to distinguish normal *periodic breathing* from *apnea*. By definition, periodic breathing consists of three or more respiratory pauses longer than 3 seconds in duration with less than 20 seconds between pauses. There is no associated bradycardia or cyanosis. This contrasts with apnea and is a particular problem in preterm infants. Apnea is defined as a respiratory pause longer than 20 seconds. It may be associated with bradycardia and hypoxia.[41] Periodic breathing and apnea are believed to be disorders on a continuum, both stemming from abnormal physiologic control of respiration. Periodic breathing is considered a benign disorder. Infants with symptomatic apneic episodes that result in apparent life-threatening events are thought to be at increased risk for sudden infant death syndrome.[49]

PULSE

Examine the pulse to establish the cardiac rate and regularity of the rhythm. Though rarely diagnostic, peripheral pulses may yield clues about cardiac disease, such as aortic insufficiency, and information about the integrity of the peripheral vascular supply. Doppler ultrasound has utility in locating a pulse, assessing fetal heart tones beyond the first trimester of pregnancy, evaluating peripheral lower extremity vascular insufficiency with an ankle-brachial index, and assessing blood pressure in infants or in patients with low-flow states.

Physiology

Blood flowing into the aorta with each cardiac cycle initiates a pressure wave. Blood flows through the vasculature at approximately 0.5 m/sec, but pressure waves in the aorta move at 3 to 5 m/sec. Therefore palpated peripheral pulses represent pressure waves, not blood flow.

Indications and Contraindications

Assessment of blood flow by palpation of the pulse can be used to gauge the presence of cardiac contractility and not just the electrical rhythm. Caution should be taken to not overgeneralize the presence or strength of a pulse when predicting blood pressure. The necessity for repeated pulse evaluations is dictated by the clinical complaint and the status of the patient. Continuous monitoring is not routine but may be helpful when the clinical situation predicts significant variability in heart rate, as in the setting of sepsis.[50,51] An association between absence of a radial pulse or absence of both radial and femoral pulses and hypotension has been demonstrated in hypovolemic trauma patients. The variability in individual response prohibits the use of this parameter as an absolute gauge of blood pressure.[52]

No contraindications exist to assessment of the pulse rate. Keep in mind a few cautionary notes about examination of the carotid pulse. Avoid concurrent bilateral carotid artery palpation because this maneuver could theoretically endanger cerebral blood flow. Massage of the carotid sinus, found at the bifurcation of the external and internal carotid arteries at the level of the mandibular angle, may result in reflex slowing of the heart rate. To avoid inadvertent carotid sinus massage, palpate the carotid pulse at or below the level of the thyroid cartilage. In adults with atherosclerotic disease, there is a rare risk of precipitating a cerebrovascular event by vigorous palpation of the carotid artery. Minimize this risk by prior auscultation of the carotid artery. If a bruit is present, gently palpate the carotid pulse while avoiding vigorous palpation, or use a Doppler ultrasound probe to assess carotid flow instead.

Procedure

Depending on the clinical scenario, pulses are palpable at numerous sites, although for convenience the radial pulse at the wrist is routinely used. Use the tips of the first and second fingers to palpate the pulse. The two advantages of this technique are that (1) the fingertips are quite sensitive, thereby enabling the pulse to be located easily and counted, and (2) the examiner's own pulse may be erroneously counted if the thumb is used. Pulses are easily palpated at the carotid, brachial, femoral, posterior tibial, and dorsalis pedis arteries. Palpate the pulse at the brachial artery to appreciate its contour and amplitude. Locate the pulse at the medial aspect of the elbow and note that it is more easily palpated when the elbow is held slightly flexed.[53] Determine the pulse rate by counting for 1 minute, particularly if any abnormality is present. Common convention in the acute care setting is to count a regular pulse for 15 seconds and multiply the resulting number by 4 to determine the beats per minute.

In neonates, use direct heart auscultation and umbilical palpation as the methods of choice to determine the heart rate. Instantaneous changes in newborn heart rates are best indicated to the resuscitation team by the clinician tapping out each heartbeat.[54] In unstable children, palpate the central arteries, particularly the femoral and brachial pulses, instead of the more peripheral arteries. In a comparison of four methods of determining the heart rate in infants, listening at the apex of the heart was found to be more accurate than palpation of the brachial, carotid, or femoral pulses.[55] Of the sites for palpation of the heart rate, the femoral artery has proved most valuable, especially in hypotensive infants.[56]

Interpretation

Pulse Rate

Consider the individual's physiology when interpreting the pulse. In infants and children, interpret the pulse rate with reference to age (see Table 1.1). Pulse varies with respiration: it increases with inspiration and slows with expiration. This is known as sinus dysrhythmia and is physiologic.

Although *bradycardia* is most commonly defined as a heart rate lower than 60 beats/min in adults, a well-conditioned athlete may have a normal resting heart rate of 30 to 40 beats/min.[57,58] As discussed earlier, a redefinition of bradycardia to less than 45 beats/min and tachycardia to greater than 95 beats/min has been proposed based on a normal healthy population.[22,59] Such definitions include 95% of the population and do not address any given individual's normal baseline rate.

Consider whether a patient's abnormal pulse rate is a primary or secondary condition. Examine the entire set of vital signs when attempting to discern the cause of the abnormal rate. For example, hyperthermia causes sinus tachycardia. Drug fever, typhoid fever, and central neurogenic fever are considerations when no corresponding tachycardia is found in a patient with elevated body temperature. Hypothermia, with its reduced metabolic demands, may be associated with bradycardia. Some disease states are defined by their effect on heart rate, such as thyrotoxicosis with tachycardia or myxedema coma with bradycardia.

Consider the medications that the patient may be taking or the presence of a mechanical pacemaker. Digitalis compounds, β-blockers, and antidysrhythmics may alter the normal heart rate and the ability of this vital sign to respond to a new physiologic stress. These cardioactive medications may cause the abnormality in the patient's heart rate. Nonprescription drugs can be equally significant in their effect on heart rate. Sympathomimetic drugs such as cocaine and methamphetamine increase heart rate, as do anticholinergic drugs.

Heart Rhythm

In addition to determining the pulse rate, obtain information about the regularity of the pulse by palpation. An irregular pulse suggests atrial fibrillation or flutter with variable block, and accurate assessment of the pulse should be carried out by

auscultation of the apical cardiac sounds. The apical pulse is frequently greater than the peripheral pulse because of inadequate filling time and stroke volume, with resultant nontransmitted beats. A greater pulse deficit generally reflects more severe disease.[60]

Pulse Amplitude and Contour

Accurate examination and description of pulse amplitude and contour can provide additional clinical information and aid in decision making. Superimposition of one pathophysiologic state on another may modify the pulse. For example, sepsis may result in variable pulse amplitudes, depending on the stage in the development of the disease. Early in sepsis, cardiac output increases and vascular resistance decreases, causing bounding pulses. In advanced sepsis or septic shock, falling cardiac output and increased vascular resistance are seen, and pulses are diminished.[61] Definable age-related changes in pulse amplitude and contour can be identified. Such changes are due to an increase in arterial stiffness, resulting in increased pulse wave velocity and progressively earlier wave reflection. This leads to increased pulse amplitude in the elderly at all commonly measured sites (carotid, femoral, and radial).[61] In addition to these age-related changes, pulse wave analysis may be useful in determining arterial stiffness and the likelihood of atherosclerotic disease in a vascular laboratory setting.[62] If present globally, weak pulses can be a significant finding in hypotensive patients, or an indication of limb ischemia if isolated to one extremity. Bounding pulses can be seen with a widened pulse pressure and are discussed later in the section on blood pressure. Routine measurement of pulse amplitude is not reproducible by simple palpation and requires instrumentation not available in EDs.

Pulses During Cardiopulmonary Resuscitation

Palpated femoral pulses during chest compression may represent either forward arterial blood flow or "to-and-fro" movement of blood from the right side of the heart to the venous system. A carotid pulse is preferred when assessing the adequacy of chest compressions during cardiopulmonary resuscitation (see Chapter 17).

ARTERIAL BLOOD PRESSURE

Systolic blood pressure changes with each heartbeat. Changes in arterial blood pressure over time may indicate success of treatment or worsening of the patient's overall condition. An abrupt reduction in a patient's arterial blood pressure usually indicates the need for immediate intervention or reconsideration of therapy. The current section discusses indirect blood pressure monitoring; intraarterial techniques are considered elsewhere. Discussion of the specific use of the Doppler device for measurement of pulse and blood pressure and for measurement of orthostatic blood pressure and changes in pulse follow this section. Despite an association between the absence of hypotension and the presence of a radial pulse or between hypotension and the absence of both radial and femoral pulses in the setting of trauma, the variability in individual responses prohibits the use of this parameter as an absolute gauge of blood pressure.[53]

Physiology

Arterial blood pressure indicates the overall state of hemodynamic interaction between cardiac output and peripheral vascular resistance. Arterial blood pressure is the lateral pressure or force exerted by blood on the vessel wall. It indirectly measures perfusion, and blood flow equals the change in pressure divided by resistance. Because peripheral vascular resistance varies, a normal blood pressure does not confirm adequate perfusion.[63] Mean arterial blood pressure (MAP) can be estimated by adding one third of the pulse pressure (i.e., the difference between systolic and diastolic blood pressure) to diastolic pressure or by using the following measure[64,65]:

$$\text{MAP} = \frac{\text{Diastolic pressure} \times 2 + \text{Systolic pressure}}{3}$$

Many modern bedside telemetry monitors automatically incorporate MAP measurements into the blood pressure systolic and diastolic pressure readings.

Indications and Contraindications

Patients with minor ambulatory complaints unrelated to the cardiovascular system may not necessarily need their blood pressure measured in the ED, and those with hemodynamic instability need frequent monitoring of blood pressure. In children, there is a significant amount of variability regarding standard situations that require measurement of blood pressure. In general, the younger the patient, the less likely blood pressure will be measured.[66,67] In newborns, infants, and even toddlers, capillary refill is sometimes substituted for standard blood pressure measurement, although viewing these tests as equivalent can lead to significant errors.

In low-flow states, Doppler measurement of blood pressure may be obtained rapidly. Repeated measurements will provide an evaluation of the adequacy of resuscitation in patients whose blood pressure cannot be auscultated by standard techniques and in those in whom intraarterial blood pressure measurements are either contraindicated or technically unobtainable.[68,69] Placing a catheter for direct intraarterial measurement of blood pressure may be performed safely in the ED, but is not standard of care and has a higher risk for complications. In particular, direct measurement of arterial pressure during pulseless electrical rhythms may help discriminate between a severe shock state and otherwise nonresuscitatable status.[70,71] Alternative noninvasive devices for continuous blood pressure measurement (CBPM) have been introduced clinically, with varying success. One common method of CBPM uses finger cuffs equipped with infrared (IR) photoplethysmography and sophisticated technology for quantification of finger blood pressure levels. Finapres (Ohmeda, Madison, WI) was the first commercial product using this technique, and several newer products are on the market today. A number of commercial systems use an alternative method of arterial applanation tonometry to measure CBPM. Further study is needed for the validation of devices using these techniques.[72,73]

Relative contraindications to specific extremity blood pressure measurement include an arteriovenous fistula, ipsilateral mastectomy, axillary lymphadenopathy, lymphedema, and circumferential burns over the intended site of cuff application.

Equipment

Two types of blood pressure monitoring equipment are currently available and used in EDs: cuff type and noninvasive waveform analysis.

Cuff Type

The equipment required for indirect blood pressure measurement includes a sphygmomanometer (cuff with an inflatable bladder, inflating bulb, controlled exhaust for deflation, and manometer) and a stethoscope, Doppler device (for auscultation), or oscillometric device.[74–76] A common practice in the prehospital and interhospital transport setting is to forego auscultatory blood pressure measurements with a stethoscope and instead obtain systolic values only by palpation of the first Korotkoff sound. This practice, though sometimes the only feasible method of obtaining any value in a noisy environment, poses a significant potential for error.

According to the American Heart Association guidelines, the sphygmomanometer cuff should be an appropriate size for the patient to ensure an accurate reading. The width of the bladder should be at least 40% of the distance of the limb's midpoint (i.e., from the acromion process to the lateral epicondyle). This published figure of the ideal width, when studied in a validation review, may be higher, up to approximately 50%.[77] The length of the bladder should be 80% of the midarm circumference or twice the recommended width.[68] Discrepancies in matching upper arm size with cuff size have been demonstrated to produce significant errors in critically ill populations when compared with invasive intraarterial blood pressure measurements.[78] The availability of appropriately sized cuffs appears to be a pervasive problem, especially as approximately 80% of patients do not fit the standard 12-cm large cuffs.[79] In one study, 90% of aneroid devices had only one size of cuff available.[80] A second study phase from this group showed no marked improvement in agreement of oscillatory and invasive measurements, despite using the correct cuff size.[81]

The manometers in common use are either aneroid, digital, or mercury gravity column, though the mercury type is much less common in modern use. All three types of manometers are convenient for bedside use, although the mercury gravity column must be placed vertically to ensure accurate measurements. An aneroid manometer uses a metal bellows that elongates with the application of pressure. This elongation is mechanically amplified and transmits the motion to the indicator needle.

Manometers require annual servicing. Mercury columns may require the addition of mercury to bring the edge of the meniscus to the zero mark. The air vent or filter at the top of the mercury column should also be checked for clogging. An aneroid manometer should be calibrated against a mercury column at least yearly. If the aneroid indicator is not at zero at rest, the device should not be used.[82] Digital manometers may not be validated for all patient groups and could give inaccurate readings.

Automatic sphygmomanometers may improve physiologic monitoring with their alarm and self-cycling capabilities. They offer indirect arterial blood pressure measurement with little pain and without the risks associated with invasive arterial lines.[83] Accuracy of measurements does not suffer during rapid cycling, and the potential for vascular injury from nearly continuous arterial compression dictates that most automated blood pressure units will revert back to less frequent (i.e., every 15 to 20 minutes) cycling as a safety precaution. Oscillometric blood pressure monitors detect motion of the blood pressure cuff transmitted from the underlying artery. A sudden increase in the amplitude of arterial oscillations occurs with systolic pressure and MAP, and an abrupt decrease occurs with diastolic pressure.[84]

There appears to be less variability with the oscillometric blood pressure method than with the auscultatory method in children. These results are not generalizable to the neonatal population, and errors are commonly encountered even when exhaustive measures are taken to control the environment.

In adult patients, numerous studies have focused on the reliability of auscultatory versus automated blood pressure measurements. Mercury column versus Dinamap readings showed increased disparity when systolic blood pressure was greater than 140 mm Hg, the range at which accuracy should be most rigorously sought to correctly identify hypertension. In general, automated blood pressure devices yield higher systolic and lower diastolic blood pressure.[85] The range of error in automated devices was, on average, 4.0 to 8.6 mm Hg.[86,87] Unfortunately, these studies represent populations without critical illness and do not reflect the accuracy of readings at the extremes of hypertension and hypotension, thus making generalization to an ED population difficult.[87,88] Like most durable medical equipment, sphygmomanometers are subject to deterioration with use. Automatic sphygmomanometer validation is a form of calibration and deserves quality control and traceability to ensure accurate results.[88]

Procedure

Obtain indirect blood pressure measurements at the patient's bedside by palpation, auscultation, Doppler, or oscillometric methods. The technique is straightforward and accurate when the equipment is well maintained, calibrated, and used by clinicians who follow accepted standards. The patient may be lying or sitting, as long as the site of measurement is at the level of the right atrium and the arm is supported.[68,76] Unless the arm is kept perpendicular to the body with the elbow resting on a desk, measurements will be 9 to 14 mm Hg higher, regardless of body position.[77,89] Allowing the arm to be parallel to the body when supine and supporting the arm perpendicular to the body when measuring blood pressure may create a pseudo-drop in blood pressure. These changes are thought to be dependent on the mechanical properties of the arteries themselves and not associated with hydrostatic pressure alone.[90]

To palpate arterial blood pressure, inflate the cuff to 30 mm Hg above the level at which the palpable pulse disappears. Once properly inflated, palpate directly over the artery and deflate the cuff at a rate of 2 to 3 mm Hg/sec. Report the initial appearance of arterial pulsations as the palpable blood pressure. This practice, known as the Riva-Rocci palpatory technique, has shown mixed results in yielding accurate estimations of blood pressure. One study determined that the average underestimation of systolic blood pressure was 6 mm Hg.[91] Another operative study looking at the combination of palpated systolic blood pressure and observed visual return of continuous pulse oximetry reported an underestimation of 10 to 20 mm Hg.[87] The same technique is used with the Doppler device, and the palpated pulse is replaced with the Doppler auditory signal. Measurement of arterial pressure by palpation and Doppler yields only estimates of systolic blood pressure. The Doppler method is preferred when determining blood pressure in infants.[88]

When auscultating blood pressure at the brachial artery, apply the blood pressure cuff approximately 2.5 cm above the antecubital fossa with the center of the bladder over the artery.[76] Apply the bell of the stethoscope directly over the brachial artery with as little pressure as possible.[92] *Systolic arterial blood*

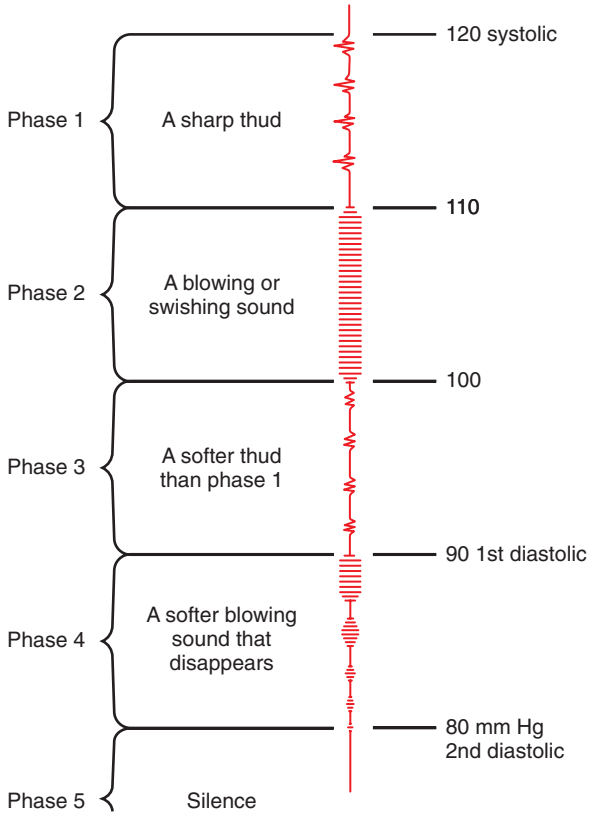


Figure 1.2 Korotkoff sounds. The first audible sound occurs in systole, and the sound disappears in diastole. (From Burnside JW, McGlynn TJ: *Physical diagnosis*, ed 17, Baltimore, 1986, Williams & Wilkins.)

pressure is defined as the first appearance of faint, clear, tapping sounds that gradually increase in intensity (Korotkoff phase I). *Diastolic blood pressure* is defined as the point at which the sounds disappear (Korotkoff phase V).[93] In children, phase IV defines diastolic blood pressure (Fig. 1.2).[7] Phase IV is marked by a distinct, abrupt muffling of sound when a soft, blowing quality is heard.

It is best to auscultate over the brachial artery because of accepted standardization of the measured values. Alternative sites include the radial, popliteal, posterior tibial, or dorsalis pedis arteries, although any fully compressible extremity artery may be used. Studies evaluating direct and indirect blood pressure measurements have demonstrated good correlation between these methods.[94,95]

Occasionally it may not be feasible to obtain proximal upper extremity blood pressure measurements because of patient access issues, particularly those encountered in the prehospital setting. Forearm measurements may be obtained more easily, though correlation to standard proximal upper extremity values has been controversial. An earlier study showed fair correlation to a brachial cuff measurement (within 20 mm Hg in 86% of systolic measurements and 94% of diastolic measurements).[96] A subsequent study demonstrated that forearm measurements cannot routinely replace upper arm measurements to determine blood pressure (the potential variance from an upper arm measurement is ± 19 mm Hg for systolic pressure).[97] Alternatively, noninvasive finger blood pressure measurements have shown promise when compared with standard upper extremity readings. The overall discrepancy in an ED study was 0.1 mm Hg with a standard deviation of ± 5.02 mm Hg when comparing finger blood pressure and invasive MAP via radial artery cannulation.[98]

Novel noninvasive continuous finger cuff technology offers the benefit of uninterrupted monitoring and has the advantage over invasive techniques of being safer and immediately available. Noninvasive finger cuff measurements have shown reasonable correlation, even in critically ill populations in the ED.[73] Wrist blood pressure has been shown to have good average accuracy in the surgical environment when compared with oscillometric devices. Patient comfort is reported to be greater with these devices. The typically stated contraindications to the acquisition of upper arm blood pressure (limitation after mastectomy, etc.) may not apply.[99]

The accuracy of the palpatory, Doppler, and oscillometric methods has also been investigated.[100–102] When phase I and V Korotkoff sounds are used, indirect methods typically underestimate systolic and diastolic pressure by several millimeters of mercury.[100–103] During shock, the palpatory and auscultatory methods underestimate simultaneous direct arterial pressure measurements.[104] The flush method, in which the return of color after deflation of the cuff is used to estimate blood pressure in infants, may underestimate systolic blood pressure by up to 40 mm Hg.[105] This method is unreliable and not recommended.

Complications

Complications of indirect blood pressure measurements are minimal when the proper procedure is followed. Inadvertent prolonged application of an inflated blood pressure cuff may result in falsely elevated diastolic pressure and ischemia distal to the site of application.[78] Invasive blood pressure monitoring is associated with a number of potential problems (see Chapter 20).

Interpretation

Normal blood pressure increases with decreasing distance from the heart and aorta. Blood pressure tends to increase with age and is generally higher in males. Individual factors that influence blood pressure include body posture, emotional or painful stimuli, environmental influences, vasoactive foods or medications, and the state of muscular and cerebral activity. Exercise and sustained isometric muscular contraction increase blood pressure in proportion to the strength of the contraction. A normal diurnal pattern of blood pressure consists of an increase throughout the day with a significant, rapid decline during early, deep sleep.[106]

Normal lower limits of systolic blood pressure in infants and children can be estimated by adding 2 times the age (in years) to 70 mm Hg. The 50th percentile for a child's systolic arterial blood pressure from 1 to 10 years of age can be estimated by adding 2 times the age (in years) to 90 mm Hg. Children older than 2 years are considered hypotensive when systolic blood pressure is less than 80 mm Hg.[107] Children are able to maintain MAP until very late during shock.[108] The finding of a normal blood pressure in a child with signs of poor perfusion should not dissuade the clinician from appropriate treatment. Most adults are considered hypotensive if systolic blood pressure is lower than 90 mm Hg, but some individuals normally exhibit a systolic pressure in that range. In the elderly, the presence of normotension within defined or published limits may not be reassuring. When considering systolic blood pressure cutoffs

for trauma patients, 85 mm Hg for patients aged 18 to 35 years, 96 mm Hg for patients aged 36 to 64 years, and 117 mm Hg for those older than 65 years have been proposed as new standards for hypotension.[18] When accompanied by signs of shock, immediate treatment is indicated. In patients with shock, blood flow cannot be reliably inferred from heart rate and blood pressure values.[109,110]

Hypertension

Adults are hypertensive if either systolic or diastolic pressure consistently exceeds 140 or 90 mm Hg, respectively.[111,112] A meta-analysis showed strong correlation of blood pressure to vascular (and overall) mortality down to at least 115/75 mm Hg.[113] Some authors have suggested altering the blood pressure definitions to include an "optimal" blood pressure of 115/75 mm Hg.[13] Other authors have suggested incorporating blood pressure into a global cardiovascular risk assessment that includes other associated risk factors.[14,15] The applicability of population norms for hypertension in a stressful emergency situation is controversial. One should not make diagnostic or therapeutic decisions based solely on an abnormal initial measurement. Patients with hypertension require repeated measurements to assess whether therapy is required in the ED. Because sustained hypertension may be seen in more than one third of initially hypertensive ED patients, careful evaluation and follow-up are required.[114] Unfortunately, elevated blood pressure readings are still almost uniformly ignored or unrecognized in the emergency setting, particularly in children.[115] The phenomenon of *white coat hypertension* (WCH) is defined as a persistent elevation in blood pressure in the clinical setting only. The prevalence of WCH is between 20% and 94%, depending on the frequency of reassessment in the clinical setting.[116] It is unclear whether patients who have isolated hypertension in the clinical setting (WCH) are at increased risk for the development of hypertension and subsequent end-organ damage.[117]

Measurement Errors

Erroneous blood pressure measurements may result from several factors.[118] Falsely low blood pressure may be caused by using an overly wide cuff, by placing excessive pressure on the head of the stethoscope, or by rapid cuff deflation.[119,120] Falsely high blood pressure may be caused by the use of an overly narrow cuff, anxiety, pain, tobacco use, exertion, an unsupported arm, or slow inflation of the cuff.[121] There appears to be a statistically significant difference in the error rate associated with patients weighing more than 95 kg, whether from obesity or as a result of muscular upper arms from body building.[122,123]

Of note, 41% of adults observed at the University of Pittsburgh required non–standard-sized cuffs, and the use of small cuffs was associated with a mean error of 8.5 and 4.6 mm Hg in systolic and diastolic pressure, respectively.[120] Other studies have confirmed relatively high rates of inappropriately diagnosed hypertension in obese patients based on erroneous cuff size.[122] Other specific study populations in this area have been critically ill patients, in whom disparate cuff size can lead to significant inaccuracies based on arm circumference.[84]

Hypotensive patients have unreliable Korotkoff sounds, but Doppler measurements are well correlated with direct arterial systolic pressure measurements in these patients.[124] An auscultatory gap can be appreciated in hypertensive patients and may mislead the clinician. It is heard during the latter part of phase I and should not be confused with diastolic readings. Auscultation until the manometer reading approaches zero should prevent misinterpretation. In patients with aortic insufficiency or hyperthyroidism, in those who have just finished exercising, and in children younger than 5 years, measurement of diastolic blood pressure should occur at Korotkoff phase IV. Extremes of blood pressure, both hypotension and hypertension, have been found to be factors contributing to measurement errors in critically ill pediatric patients. Predictably, falsely high readings for noninvasive versus invasive measurements have been obtained in hypotensive patients and falsely low values in hypertensive states.[125]

Irregular heart rates may also interfere with accurate determination of blood pressure. Take a second or third reading, with 2 minutes of deflation between recordings, and obtain an average when premature contractions or atrial fibrillation are present.

Hemiplegic patients may exhibit different blood pressures in the affected and unaffected arms.[126] A flaccid extremity tends to yield lower systolic and diastolic pressure, whereas a spastic extremity tends to yield higher values than the extremity with normal motor tone. Although these differences are generally small, it is preferable to monitor blood pressure in the unaffected limb.

Numerous errors may occur in the accurate measurement of blood pressure. The only way to combat these errors is to first be cognizant of practices contributing to them. Unfortunately, few nurses can identify causes of potentially erroneous readings. In a study examining nurses' ability to obtain accurate readings, proper technique in determining systolic blood pressure could be identified 61% of the time; diastolic blood pressure, 71% of the time; and an auscultatory gap, 54% of the time. Nurses were able to correctly identify faulty equipment 58% of the time, assess cuff size 57% of the time, determine appropriate inflation pressure 29% of the time, note the appropriate deflation rate 62% of the time, and determine correct arm positioning 14% of the time.[127]

There is an increasing number of patients with heart failure, those receiving bridging measures to transplantation, or those treated with long-term circulatory augmentation devices in the form of left ventricular assist devices (LVADs); therefore it is useful to understand the difficulty in interpreting blood pressure measurements in these patients. All types of VADs fit into two categories: (1) pulsatile and (2) nonpulsatile. Pulse and blood pressure readings in patients with pulsatile VADs (Thoratec, HeartMate XVE, Novacor, C-Pulse) are comparable to values in the general non-VAD population. Nonpulsatile VADs (HeartAssist 5, Incor and Excor, Jarvik 2000, VentriAssist, MTIHeart LVAD, HVAD/MVAD, DuraHeart, DeBakey LVAD, HeartMateII/III) function by either centrifugal or axial blood flow, and this has a significant impact on the ability to detect pulses.[128] Typically, these patients appear to be well-perfused with adequate skin warmth and capillary refill even though pulses may be absent. Blood pressure readings can be obtained with these nonpulsatile flow devices, and diastolic blood pressure, pulse pressure values, and MAP vary significantly depending on the speed of the pump.[129]

Pulse Pressure

The difference between systolic and diastolic pressure is termed pulse pressure. For example, if the blood pressure is 120/80 mm Hg, the pulse pressure is 40 mm Hg. Increased pulse pressure (i.e., ≥60 mm Hg) is commonly observed with anemia, exercise, hyperthyroidism, arteriovenous fistula, aortic

regurgitation, increased intracranial pressure, and patent ductus arteriosus. A narrowed pulse pressure (≤20 mm Hg) may be a manifestation of hypovolemia, increased peripheral vascular resistance (as seen in early septic shock), or decreased stroke volume. A narrowed pulse pressure is classically noted in aortic stenosis and pericardial tamponade. Traditional vital sign measurements, such as systolic blood pressure and oxygen saturation, often fail to predict mortality or indicate the need for life-saving interventions or reductions in central blood volume until after the onset of cardiovascular collapse. There is evidence that an early indicator of reduced central blood volume in the presence of stable vital signs is the reduction in pulse pressure.[130]

Differential Brachial Artery Pressure

The presence of a systolic blood pressure difference of 10 to 20 mm Hg between the arms suggests a normal condition. If greater, it may indicate advanced focal atherosclerosis, coarctation of the aorta proximal to the left subclavian artery, type A aortic dissection, aortic arch syndromes, or other vascular processes preferentially affecting one extremity. The utility of upper extremity bilateral blood pressure measurements has recently come into question. One study found a 10-mm Hg systolic or diastolic difference in 53% of patients in the emergency setting and a 20-mm Hg or higher difference in 19% of patients.[131] Although these numbers have not generally been found to be of this magnitude in metaanalyses,[132] the unique setting of the study in the ED makes correlation particularly salient for the emergency physician. The reliability of peripheral pulse deficits in diagnosing or excluding type A aortic dissection is a frequently cited reason for evaluating blood pressure in both arms. In a metaanalysis, Teece and Hogg noted that the successful use of the absence of a clinical pulse deficit to exclude thoracic dissection in patients with chest pain was just 31%, and the authors concluded that peripheral pulse deficits are far too insensitive to warrant their use as a means of excluding thoracic aortic dissection in patients with chest pain.[133] Given that many in the general population have significant differences in blood pressure in each arm, the diagnostic value of this frequently cited indication for obtaining bilateral brachial blood pressure is unproven. Essentially, most patients with a type A aortic dissection will not have a measurable blood pressure discrepancy between the arms, and most of those who do have such a finding will not have dissection.

Brachial pressure differences did not appear to be linked to age, gender, race, MAP, cardiovascular risk, or final discharge diagnosis. Smaller interarm differences have been reported in the ED setting (18% in hypertensive patients and 15% in normotensive patients when a cutoff greater than 10 mm Hg was used).[16,17] Though not tested, a method proposed to minimize these differences in the ED is to take simultaneous blood pressure readings from both the left and right extremities with two calibrated automated blood pressure units.[134]

Pulse-Pressure Variation

Fluid resuscitation is an integral piece of the management of patients with circulatory failure, though administration of the proper amount and rate of parenteral fluids can be challenging. Fluid responsiveness, or the ability of the left ventricle to increase stroke volume in response to fluid administration, is an emerging concept that helps to address this challenge.[135] This concept is based on the physiology of the Frank–Starling curve and the knowledge that pulse pressure (systolic pressure minus diastolic pressure) is directly proportional to stroke volume. The variation in pulse pressure seen with the respiratory cycle, or *pulse-pressure variation*, reflects the magnitude of respiratory change on stroke volume. This is best demonstrated by the influence of mechanical ventilation on right ventricular preload. Studies have shown that a pulse-pressure variation of greater than 13% is highly predictive of fluid responsiveness in mechanically ventilated patients.[136]

Though many methods exist to assess fluid responsiveness, the standard method is done with passive leg raising (PLR). This "self-volume challenge" increases preload through translocation of venous blood from the lower extremities to the thorax. A patient who exhibits a rise of more than 10% in their aortic blood flow (measured with esophageal Doppler) or cardiac index (measured with thermodilution) is considered a "fluid responder," which is indicative of the need for further fluid administration.[135] This concept is becoming increasingly useful in optimizing the fluid management of critically ill patients.

Pulsus Paradoxus

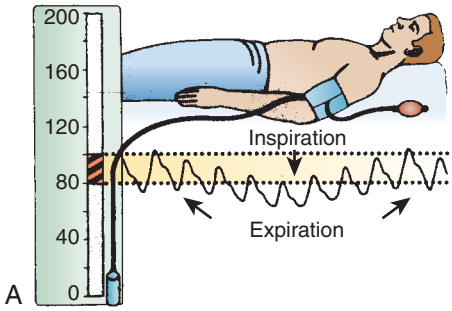
Normal respiration briefly decreases systolic blood pressure by approximately 10 mm Hg during inspiration. Pulsus paradoxus occurs when there is a greater than 12-mm Hg decrease in systolic blood pressure during inspiration. Pulsus paradoxus may occur in patients with chronic obstructive pulmonary disease, pneumothorax, severe asthma, or pericardial tamponade.[104]

To measure a paradoxical pulse, have the patient lie comfortably in the supine position at a 30- to 45-degree angle and breathe normally in an unlabored fashion (which are unusual conditions in a patient suspected of having cardiac tamponade, severe asthma, chronic obstructive pulmonary disease, or pneumothorax).[137] Inflate the blood pressure cuff well above systolic pressure and slowly deflate it until the systolic sounds that are synchronous with expiration are first heard (Fig. 1.3). Initially, the arterial pulse will be heard only during expiration and will disappear during inspiration. Deflate the cuff further until arterial sounds are heard throughout the respiratory cycle. Palpation at the radial or femoral arteries may yield complete disappearance during inspiration. When present, this technique is a quick bedside confirmation of the possibility of severe tamponade.

An alternative method for determination of pulsus paradoxus is by visually observing the loss of the pulse oximetry waveform and then its reappearance.[138] The plethysmographic method has been validated in intensive care unit settings.

If the difference between inspiratory and expiratory pressure is greater than 12 mm Hg, the paradoxical pulse is abnormally wide.[137,138] Most patients with proven tamponade have a difference of 20 to 30 mm Hg or greater during the respiratory cycle.[137–139] This may not be true of patients with very narrow pulse pressures (typical of advanced tamponade), who have a "deceptively small" paradoxical pulse of 5 to 15 mm Hg.[139–141]

Pulsus paradoxus has been correlated with the level of impairment of cardiac output by tamponade. In an uninjured patient with pericardial effusion, a pulsus paradoxus greater than 25 mm Hg (in the absence of relative hypotension) is both sensitive and specific for moderate or severe versus mild tamponade.[137,142] An echocardiographic study found that an abnormal pulsus paradoxus had a sensitivity of 79%, a specificity of 40%, a positive predictive value of 81%, and a negative predictive value of 40% for right ventricular diastolic



A

PROCEDURE FOR THE MEASUREMENT OF PULSUS PARADOXUS

The patient should be reclining at a 30° to 45° angle and instructed to breathe normally.

1. Inflate a standard blood pressure cuff until Korotkoff sounds over the brachial artery disappear.
2. Lower pressure in the cuff a few millimeters of mercury per second until the first Korotkoff sounds appear during expiration.
3. Maintain pressure at this level and observe the disappearance of sounds during inspiration. Record this cuff pressure.
4. Very slowly lower cuff pressure until Korotkoff sounds are heard throughout the respiratory cycle. Record this cuff pressure.
5. The difference between pressures recorded in the two previous steps is then recorded as the measurement (in millimeters of mercury [mm Hg]) of pulsus paradoxus. A pulsus paradoxus >12 mm Hg is abnormal but nonspecific (see text).

B

Figure 1.3 **A,** Measurement of pulsus paradoxus. Note that systolic pressure varies during the respiratory cycle. Inspiration normally decreases systolic blood pressure slightly (termed paradoxical pulse), but if the difference between inspiratory and expiratory systolic pressure is greater than 12 mm Hg, the paradoxical pulse is abnormally wide. **B,** Technique for measurement of pulsus paradoxus. (A, From Stein L, Shubin H, Weil M: Recognition and management of pericardial tamponade. *JAMA* 1973;225:504. Copyright 1973, American Medical Association. Reproduced with permission.)

collapse.[142–144] The absence of a paradoxical pulse does not rule out tamponade.

In the pediatric population, pulsus paradoxus has been studied to determine the severity of obstructive and restrictive pulmonary disease,[143] most commonly asthma. A value of 15 mm Hg or greater correlates well with the clinical score, peak expiratory value, flow rate, oxygen saturation, and subsequent need for admission.[144]

Despite the disease entities that a widened pulsus paradoxus may suggest, it is a difficult test to perform adequately with only a sphygmomanometer. Because it is a useful clinical tool, new aids should be developed and used to reliably predict this important vital sign.[145]

Shock Index

The ratio of pulse rate to systolic blood pressure has been suggested as a measure of clinical shock. The shock index (SI) has a normal range of 0.5 to 0.7. Although calculating the SI is not standard of care in the ED, a number of clinical scenarios have been studied in which the SI can be used as a predictor of severe illness or injury. An SI above 0.85 to 0.90 suggests acute illness in medical patients and a marked increase in the potential for gross hemodynamic instability in trauma patients.[146–149] The SI has been studied for use in a variety of clinical scenarios from severe pneumonia to first-trimester risk for ectopic pregnancy to sepsis. It has been found to be a valid gauge of the severity of illness.[150–152] Some studies have found that the initial pulse rate alone had nearly the same predictive power as the SI for the severity of illness. Although the SI appears to correlate with the left ventricular stroke work index, it has little correlation with systemic oxygen transport in patients with hemorrhagic and septic shock.[153]

DOPPLER ULTRASOUND FOR EVALUATION OF PULSE AND BLOOD PRESSURE

Principles of Doppler Ultrasound

Doppler ultrasound is based on the Doppler phenomenon. The frequency of sound waves varies depending on the speed of the sound transmitter in relation to the sound receiver. Doppler devices transmit a sound wave that is reflected by flowing erythrocytes, and the shift in frequency is detected. Frequency shift can be detected only for blood flow greater than 6 cm/sec.

Indications and Contraindications

Doppler ultrasound is commonly used in the ED for the measurement of blood pressure in low-flow states, evaluation of lower extremity peripheral perfusion, and assessment of fetal heart sounds after the first trimester of pregnancy. Doppler's sensitivity allows detection of systolic blood pressure down to 30 mm Hg in the evaluation of a patient in shock. In a patient with peripheral vascular disease in whom there is concern about the adequacy of peripheral perfusion, the ankle-brachial index provides a rapid, reproducible, and standardized assessment.[152] Fetal heart sounds provide a baseline assessment of any pregnant patient with 12 weeks' gestation or longer in the setting of abdominal trauma or fetal distress as a result of a complication of pregnancy. The use of Doppler ultrasound for the evaluation of deep venous thrombosis is a valuable tool, and specific training and experience are required to attain proficiency. Discussion of this topic is beyond the scope of this chapter.

Equipment

A nondirectional Doppler device has a probe that houses two piezoelectric crystals. One crystal transmits the signal and the other receives it. Reflected signals are converted to an electrical signal and fed to an output that transforms them to an audible sound.

Probes with a frequency of 2 to 5 MHz are best for detecting fetal heart sounds. Frequencies of 5 to 10 MHz are appropriate for limb arteries and veins. The probes should be monitored periodically for electrical damage and integrity of the crystals. The sphygmomanometers used in conjunction with the Doppler device should be calibrated periodically, as described in the section on evaluation of blood pressure.

Procedure

Place the Doppler probe against the skin with an acoustic gel used as an interface. The gel ensures optimal transmission and

reception of the ultrasound signal and protects the crystals. In an emergency, water-soluble lubricant (e.g., Surgilube or K-Y Jelly) may be substituted for commercial acoustic gel. Angle the probe at 45 degrees along the length of the vessel to optimize frequency shifts and signal amplitude.

To evaluate peripheral perfusion, place a sphygmomanometer cuff proximal to where the arterial pulse is being evaluated and inflate it. Place the probe over the arterial pulse and slowly deflate the cuff. The pressure at which flow is first heard is the systolic pressure under the cuff, and not the pressure at the level of the Doppler probe.

In the evaluation of peripheral vascular disease, one may determine the ankle-brachial index. It is standard for this procedure to be performed in a formal vascular laboratory, and an approximation of pressures can be determined in the ED (Fig. 1.4 and Video 1.1). Usually, only the ankle-brachial index is considered for ED purposes. Examine both brachial arteries at the medial aspect of the antecubital fossa. Angle the probe until the most satisfactory signal is obtained. Inflate the cuff and slowly deflate it until the systolic pulse is heard.

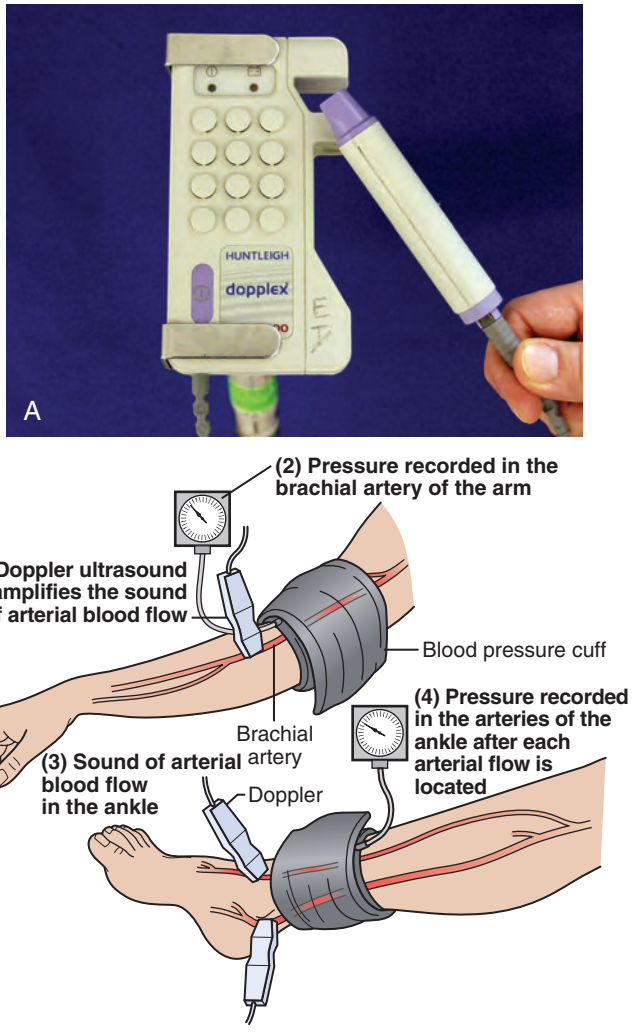


Figure 1.4 A, Handheld Doppler device with a speaker. Devices with an attached stethoscope are also used. **B,** Peripheral vascular testing is performed in a vascular laboratory, but an approximation of the integrity of the peripheral arterial circulation can be gleaned in the emergency department by using Doppler to determine systolic blood pressure in the foot and arm and calculate the ankle-brachial index.

Repeat the procedure for the posterior tibial and dorsalis pedis arteries of both lower extremities. This procedure may be done with oscillometric devices and lacks sensitivity in identifying disease.[153]

In evaluating fetal heart tones, because of variable positioning of the fetus, an examination of several locations and angles over the uterus must be performed in search for the optimal signal. It is best to begin in the mid-suprapubic area and then explore the uterus via angulation of the probe. Once tones are located, move the probe along the abdomen to reach a position closer to the origin of the sound. Distinguish fetal heart tones from placental flow by differentiating the quality of the fetal heart tones, which will not match the maternal pulse. The placental flow and maternal pulse should be identical.

Interpretation

As noted earlier, in low-flow states Doppler ultrasound can detect blood pressure as low as 30 mm Hg. Calculate the ankle-brachial index of each limb by dividing the higher systolic pressure of the posterior tibial or the dorsalis pedis artery of the limb by the higher of the systolic pressures in the brachial arteries. In normal individuals, the index should be greater than 1.0 (Fig. 1.5). Patients with mild to moderate claudication have values between 0.4 and 0.9. Values lower than 0.4 indicate severe impairment and are consistent with critical limb ischemia.[154] When the lower extremity has been amputated or injured, brachial-brachial indices can be used (i.e., comparison of systolic blood pressure in the injured or diseased upper extremity with the other extremity). Patients with ankle-brachial index values of 0.9 or lower have increased cardiovascular morbidity and mortality.[155] One study of 323 penetrating extremity wounds found that an ankle-brachial index (or brachial-brachial index) lower than 0.9 was 72.5% sensitive and 100% specific for vascular injuries.[156] Segmental lower extremity pressure measurements may help identify the level of the obstruction (Table 1.4).[157] Obese patients, diabetic patients, or those with calcified vessels that are not compressible may have abnormally high systolic pressure (e.g., 250 to 300 mm Hg) and indices that do not accurately reflect flow. Normal fetal heart tones should be between 120 and 140 beats/min. Fetal heart tones may be heard as early as the 12th week of gestation.

VITAL SIGN DETERMINATION OF VOLUME STATUS

Many techniques have been advocated to assess volume status. Unfortunately, most procedures lack a database against which to judge their reliability. Recommended methods include evaluation of skin color; skin turgor; skin temperature; supine, serial, and orthostatic vital signs; neck vein status; transcutaneous oximetry; and hemodynamic monitoring (e.g., monitoring of central venous pressure). Serial vital sign measurements have been used for assessing blood loss, but they do not reliably detect small degrees of blood loss.[154,158,159] Up to 15% of the total blood volume can be lost with minimal hemodynamic changes or any alteration in supine vital signs.[158,160] A decrease in pulse pressure occurs with acute blood loss,[161] but the patient's baseline blood pressure values are often unknown. Clinical examination of neck veins adds useful information and is less precise than measurement of central venous pressure. Most