

REVIEW PAPER

The Physical Examination in Heart Failure—Part I

As we have moved into the era of accelerating advances in laboratory testing and technology, the underpinning procedure of all medicine, the physical examination, is at risk of extinction. The death knell may well be the retirement of the last generation of physicians proficient in the bedside examination; for these physicians during their early to mid careers, diagnoses emanated from the patient's history and a well-performed physical examination, perhaps supplemented by the high-tech modalities of the day—a chest x-ray and electrocardiogram (ECG).

In this regard, the medical profession, with its educational system, is its own culprit—another example of “We have met the enemy and he is us!” The course in history and physical examination typically offered during the second year of medical school is generally assigned few or no course credits by most medical schools. It is often regarded as an orphan course, joining medical ethics, statistics, and others trying to wedge themselves in among the fact-laden, higher-credit courses. Despite this meager status, most students are still attracted to the training sessions because the course offers them their first contact with real patients and tangible disease states and thus, it offers a sense of coming closer to being a real doctor. Somehow most students also sense that the physical examination is probably important in their future roles as physicians. At this point, their idealism is often stymied by the realities of our medical education and training systems.

Unfortunately, the instructors for this important course may be those who are the least trained, experienced,

The major components and merits of the physical examination of patients with heart failure are presented. A staging of the physical examination practiced by most heart failure specialists is proposed based on the information needed at the time of the encounter. The challenges to our subspecialty regarding this diagnostic tool are the prevention of its extinction during an era of seemingly limitless comprehensive laboratory testing, the promotion of its widespread use, and, over the years, the subjection of its components to the rigors of blinded controlled studies. Part I addresses the staging of the physical examination in heart failure, general observation of the patient, vital signs, and assessment of arterial pulse and neck veins. (CHF. 2007;13:41–47) ©2007 Le Jacq

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or capable of teaching the course. The course director frequently has to scramble to scrape together volunteer instructors, and those who are volunteered per quota from each clinical department, cognizant of the fact that the system does not value this teaching activity. At the end of the course, a thank-you note covers the time and instructorship of the volunteers—instead of any financial reward or academic recognition. Thus the instructors, often including residents from the departments of radiology, pathology, and dermatology, go through the motion of delivering what they possibly absorbed from the course text or syllabus the night before the teaching session. The students' questions may not be addressed with answers of understanding (much less, any depth) or, tragically, by a response suggesting that “If you're not sure, you'll get a CT scan anyway” (without

mentioning the \$2000 to \$4000 price tag, which would provide a more comprehensive response).

Students can fill some of the void during the clinical clerkships of the last 2 years of medical school, when they are exposed to the bedside skills of good clinicians. Instead of posing questions like “What did the echocardiogram show?” the good clinician will ask the student, “Do you really need an echo here?” or, if already performed, “Did the echo confirm your physical findings in this patient?” Following the advice of the clinician, a significant disparity between a physical finding and laboratory result will be taken back to the bedside by the student for clarification and instruction—an educational model for augmenting physical examination skills throughout one's career. Formal courses or training in more advanced levels of the physical examination are rarely available for senior medical

Table I. The Essential or Basic Components of the Heart Failure Examination

General appearance
Vital signs (blood pressure and heart rate) in recumbent and upright positions
Respiratory rate and pattern and body temperature as needed or observed
Jugular venous pressure and hepatojugular reflux
Auscultation of chest, percussion as needed
Precordial palpation and auscultation
Liver span (cephalocaudal dimension at midclavicular line) and tenderness
Examination for peripheral edema and perfusion
As needed or observed
Skin appearance and temperature (to touch) of hands and feet
Pulse character, contour, and duration (carotid and/or femoral sites)

students, residents, or subspecialty fellows. Many cardiology fellows are more likely to correctly select the proper stent type and size for an obstructive coronary lesion than correctly recognize a systolic murmur as tricuspid regurgitation at the bedside.

The reasons to attain and maintain the skill of performing a good diagnostic physical examination are several, presented at the end of Part II of this series. Meanwhile, we will begin with a brief definition.

The Physical Examination in Heart Failure

For the purpose of this paper, the physical examination is defined as all bedside examination methods that are noninvasive and nonelectronic. This definition brings the stethoscope, sphygmomanometer, percussion hammer, and similar portable mechanical tools into the realm of the physical examination.

As is true for most medical disciplines and laboratory studies, the physical examination for heart failure can be viewed as a staged procedure: a diagnostic modality with varying depths or levels of interrogation or investigation. (This is not to be confused or matched with levels of billing for reimbursement.) There is no physician or nurse who has enough time to perform a complete, in-depth examination during each patient encounter. For this reason, the examination for heart failure is best staged for practical levels of application—given however, that 100 heart failure specialists will have 100 different variations on the staging outlined here.

Level I. The Quick Look or *Augenblick*

Heart failure is one of the medical conditions that can, when appropriate, render itself for clinical assessment at a glance. A brief look at the patient at bedside or even from the doorway following therapy for acute heart failure/pulmonary edema will give you a rapid impression of the effectiveness of your treatment: whether the patient is improving or whether you need to stay or return shortly thereafter for reevaluation and possible modification of the treatment plan. The same quick glance may also be appropriate in the physician assessment of the outpatient who stops by the office for a more detailed visit with the nurse practitioner or simply for renewal of prescriptions. As is true for all aspects of the physical examination, the amount of information garnered from the *augenblick* is directly proportional to the clinical experience of the inquiring physician or nurse. (From a pragmatic perspective, the usual *augenblick*, although quite informative, does not take billing beyond level I.)

Occasionally, one adds palpation of the pulse, a quick look at the neck veins, or brief listen to the chest as part of the *augenblick* assessment.

Level II. The Basic or Essential Heart Failure Examination

This section focuses on the fundamental examination applied to the day-to-day, follow-up evaluation, and management of the heart failure inpatient or outpatient and thus is not

intended to be a complete or comprehensive dissertation on the physical examination. A level II examination is practical for good follow-up patient care. The first half of level III, the comprehensive examination, is presented in the following section.

The components of the basic or essential heart failure examination, listed in Table I, consist of general appearance; vital signs with blood pressure and heart rate in the supine or semirecumbent and upright positions; estimation of jugular venous pressure and testing for hepatojugular reflux; auscultation of the chest; precordial palpation and auscultation; assessment of liver span, tenderness, and pulsation; evaluation of lower extremity edema; and assessment of skin temperature and perfusion. The techniques used for each of these maneuvers are presented below with the discussion of the level III examination.

Most of the follow-up management decisions regarding further diagnostic testing, need for hospitalization (admission, continuation, or discharge), and adjustment of medications can be readily formulated based on the recent clinical history and this basic level II heart failure examination.

Level III. The Comprehensive Heart Failure Examination

This level of evaluation is generally employed during the physician's first encounter as a consultant (outpatient or inpatient) with the heart failure patient or as the admitting physician on hospitalization. The hemodynamic correlates of some of the major physical findings in heart failure are presented in Table II.

A. General Appearance. The *augenblick* is now widened to include a more general observation of the patient. This initial part of the examination reveals a patient's comfort level, state of well-being, and whether he/she is in any form of distress (for heart failure, this tends to be respiratory in origin and appearance). Any degree of

Table II. The Hemodynamic Correlates of Some of the Major Physical Findings in Heart Failure

Jugular vein (also see Figure 2)
Jugular venous pressure (in the absence of atrioventricular valvular stenosis): approximates right ventricular diastolic pressure and can provide a clue to the state of left ventricular diastolic pressure
Hepatojugular (abdominojugular) reflux of ≥ 3 cm when jugular venous pressure is < 10 cm: marginal or mild volume overload
Jugular v wave amplitude exceeds a wave amplitude: tricuspid regurgitation
Prompt, prominent y descents: restricted right ventricular filling
Arterial pulse and pressure (also see Figure 1)
Decreased amplitude and duration (carotid location): reduced stroke volume
Pulsus alternans and paradoxus (in the absence of cardiac tamponade): left ventricular dilatation and markedly depressed ejection fraction, stroke volume, and cardiac output
Dicrotic pulse: low stroke volume and elevated systemic vascular resistance
Decreased pulse pressure, pulse amplitude and duration: reduced stroke volume
Pulse pressure/systolic pressure $< 25\%$: reduced stroke volume and a depressed cardiac output of < 2.2 L/min/m ²
Precordium (also see Figure 1 in Part II)
Left parasternal lift (right ventricular heave): right ventricular pressure and/or volume overload
Hypokinetic (weakened) ventricular impulse: decreased ventricular contractility, often a reduced ejection fraction
Wide, sustained left ventricular impulse ($> 50\%$ of systole): increased left ventricular mass
Laterally displaced left ventricular impulse: ventricular chamber enlargement
Reduced intensity of S ₁ with unchanged PR interval: elevation of left ventricular end diastolic pressure
Increased intensity of P ₂ : pulmonary arterial hypertension (see Figure 2 Part II)
S ₄ gallop: increased ventricular stiffness in diastole (reduced ventricular compliance)
S ₃ gallop in adults (without severe atrioventricular valve regurgitation or pregnancy): increased ventricular diastolic pressure and filling
Systolic murmur along lower left sternal border, which increases on inspiration: tricuspid regurgitation
Extremities
Dependent edema: often, volume overload
Cool, moist hands and feet: low cardiac output, increased sympathetic tone, and elevated systemic vascular resistance

pallor, or simply a “doesn’t look good” appearance, can be due to a variety of conditions, but in heart failure this is often the complexion of low cardiac output and amplified neurohormonal activation, particularly when accompanied by a Cheyne-Stokes respiratory pattern (cyclic periods of hyperpnea alternating with apnea or hypopnea). The chronic anemia of heart failure can accentuate the pallor.

The state of well-being, mental status, and most forms of stress can generally be gleaned from observing the patient’s eyes, face, and gestures during the encounter, particularly during history-taking. Irrespective of what the patient may say—and patients will often relate a “feeling good” response or one they hope will please the physician—experienced clinicians know to take their visual impression beyond the patient’s remarks (“I’ve been feeling OK. . .”) to get to latent problems and concerns. The eyes simply don’t lie. For experienced clinicians, this all

comes under the general heading of “reading the patient.”

The general appearance and demeanor of the spouse, particularly a wife or daughter, can be invaluable in the assessment of a patient’s condition. Greeting and observing the wife, even as an *augenblick* across the room, speaks volumes relative to a patient’s physical and mental status, response to your treatment plan, and clinical course. Irrespective of a patient’s responses or remarks to your questions, the provider should never end a visit without inquiring about a spouse’s look of concern. Her eyes can reveal a great deal. Although there are exceptions, husbands and sons tend to be more difficult to read unless the patient is in more advanced stages of heart failure and is quite ill.

Loss of temporal, deltoid, thenar, and interosseus muscle mass (which can occur in the setting of concomitant obesity); hair loss; or rapid or premature graying indicate that heart

failure over time has taken its toll metabolically and systemically.

B. Height, Weight, and Body Mass Index. The day-to-day change in body weight is one of the most useful parameters for following and managing fluid volume status in heart failure. Body weight is a physical parameter that a patient can understand and readily employ in his/her self-care.

Obesity, as it evokes the metabolic syndrome, is a risk factor for the development of cardiac disease and heart failure. Its prognostic impact on the patient once heart failure has developed, however, remains somewhat controversial, with evidence supporting obesity as a predictor of a more favorable outcome. Obesity certainly does not improve dyspnea on exertion and is often the inertia or excuse used by the patient to keep from initiating an effective exercise and conditioning program. Obesity remains a risk factor for a less favorable outcome after cardiac

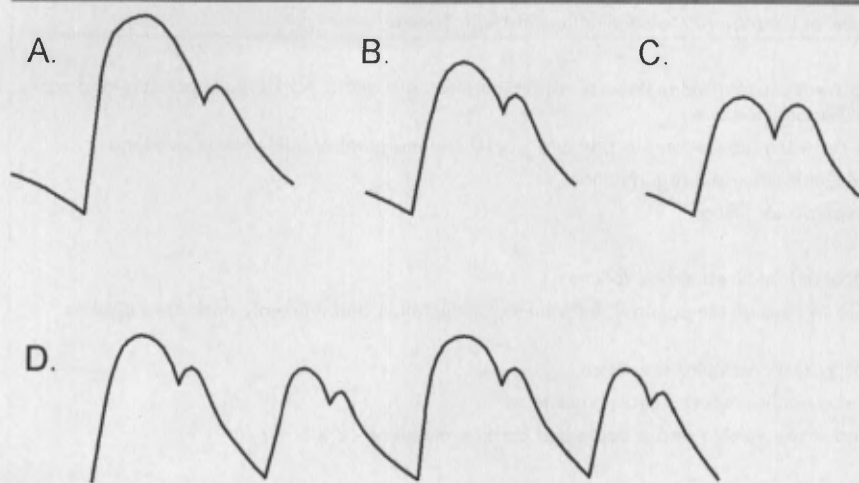


Figure 1. Sketches of some of the more common carotid pulse contours encountered in advanced stages of heart failure. (A) A normal pulse contour is provided for comparison. (B) Reduced pulse amplitude and duration in heart failure generally implicates a reduced stroke volume. (C) The reduced amplitude and duration of this pulse is followed by the relatively large reflected wave, rendering the dicrotic pulse of the markedly reduced stroke volume and cardiac output and elevated systemic vascular resistance of severe heart failure. (D) A sketch (top) and an actual pulse recording with electrocardiogram of pulsus alternans (bottom). Pulsus alternans, a weaker pulse alternating with a stronger pulse, can be palpated over the femoral or carotid arteries in patients with severe, advanced, low-output cardiac failure.

transplantation, causing most programs to prefer a body mass index of no more than 30 kg/m² for listing the heart failure patient for the procedure.

C. Vital Signs. Pulse. The pulse rate provides information on the level of cardiac compensation and the adequacy of β -blocker therapy. The pulse

regularity provides information about the patient's underlying rhythm.

For the patient in atrial fibrillation, subtracting the peripheral pulse rate from the central or cardiac heart rate (determined from precordial auscultation) provides the pulse deficit; a large pulse deficit generally represents a rapid cardiac rate with a reduced

stroke volume for many of the ventricular contractions and usually reflects a compromised hemodynamic status. During atrial fibrillation, the central heart rate (precordial auscultation) at rest and during exercise (eg, walking about the room or hallway) serves as a useful index of the degree of atrioventricular nodal blockade. Inadequate control of ventricular rate during atrial fibrillation can by itself evoke heart failure, cause short- and long-term deterioration in the course of a patient's heart failure, and account for much of a patient's dyspnea on exertion.

The character and contour of the pulse are important (Figure 1). Carotid pulse amplitude and duration are largely indicative of left ventricular (LV) stroke volume. Loss of vascular compliance (stiffening of aorta and arteries) as in aging or vascular disease can accentuate systolic blood pressure and pulse amplitude, and thus amplify and mask the reduced stroke volume. The reduction in pulse duration, however, representing a shortened LV ejection time, may still be indicative of a reduced stroke volume in this setting. (As is true for all aspects of the physical examination, appreciation of pulse duration generally develops from experience, but can be greatly facilitated and reinforced by concomitant non-invasive measurement of LV ejection time from the carotid pulse.) A dicrotic pulse contour can be palpable in severe low-output heart failure (Figure 1).

In the absence of carotid occlusive disease, a minuscule or absent carotid pulse by palpation during sinus rhythm or rate-controlled atrial fibrillation is indicative of a marked reduction of stroke volume, but this finding can also represent severe aortic valvular stenosis (occasionally in the absence of a systolic outflow murmur). This lesion can be the cause of a patient's underlying cardiomyopathic ventricle and heart failure or certainly be a major condition complicating his/her heart failure.

The carotid and femoral arteries are generally the preferred sites to palpate pulsus paradoxus and pulsus

alternans (Figure 1). Both paradoxus and alternans in heart failure are a consequence of marked ventricular enlargement and systolic dysfunction with a reduced stroke volume and cardiac output, and these pulses often occur together in the same patient. Pulsus paradoxus is commonly encountered in patients with large, dilated, cardiomyopathic ventricles. In adult human heart failure, a readily palpable pulsus alternans is almost always indicative of LV enlargement with a markedly reduced ejection fraction and stroke volume. Nonsustained ventricular tachycardia or even a premature ventricular beat can bring out pulsus alternans over the ensuing 4 to 10 sinus beats in the patient who is approaching the advanced stages of cardiomyopathic systolic heart failure.

Pulse character and contour can provide diagnostic clues for underlying causes of or conditions contributing to a patient's heart failure; examples include hyperthyroidism and hypothyroidism and aortic valvular stenosis and insufficiency. Marked diminution or absence of peripheral pulses can be indicative of a markedly depressed stroke volume and/or the presence of severe occlusive vascular disease as a comorbid condition. Signs of chronic limb hypoperfusion and ischemia support the latter consideration and tend to shift one's impression of the underlying cardiac diagnosis for heart failure from nonischemic considerations to the conditions caused by atherosclerotic coronary arteries, such as postinfarction remodeling, hibernating myocardium, and related conditions.

Blood Pressure. As heart failure moves through its course of gradual deterioration, systemic systolic pressure falls while diastolic pressure remains unchanged or increases somewhat. Thus, pulse pressure (systolic minus diastolic pressure) decreases during this course. A pulse pressure/systolic pressure ratio of less than 0.25 implicates a cardiac index of less than 2.2 L/min/m² and a less favorable clinical outcome.^{1,2} Loss of vascular

compliance increases pulse pressure, limiting the application of this ratio in the elderly and in patients with vascular disease and related conditions.

In acute heart failure, blood pressure is often used as a guide to choosing interventions such as vasodilators, positive inotropic agents, vasopressors, or mechanical-assist devices. In chronic heart failure, blood pressure is a major parameter used to determine the adequacy of therapy, with a therapeutic target of systolic pressure less than 120 mm Hg. Blood pressure, along with symptoms, also indicates the possibility of excessive dosing. To more precisely assess the level and adequacy of vasodilator therapy or neurohormonal inhibition, particularly in the setting of certain comorbid conditions (eg, diabetic neuropathy, aging), blood pressure and heart rate are recorded in the supine (or semirecumbent) and upright positions after at least 60 seconds of stabilization in each position.

Respiration. For adult humans, a respiratory rate of 18 or more breaths per minute is regarded as tachypnea. An incremental rise in respiratory rate occurs in concert with increasing respiratory insufficiency and distress. As the patient's course advances into severe stages of chronic low-output heart failure, the regular respiratory pattern gradually changes to a breathing periodicity of hyperpnea (lasting 5–15 seconds) alternating with apnea or hypopnea (lasting 5–20 seconds); this finding is referred to as Cheyne-Stokes respiration or respiratory pattern. The bedside recognition of this pattern is obviously more difficult when hypopnea, rather than apnea, alternate with hyperpnea. The importance of recognizing Cheyne-Stokes breathing periodicity relates to an appreciation of the patient's heart failure status as persistent decompensation, markedly depressed cardiac output, and poor clinical outcome.

The Cheyne-Stokes respiratory pattern is accentuated during sleep, particularly when a sleep disorder, a

common feature of heart failure, is superimposed on the disturbed respiratory pattern. Patients frequently awaken with an alarming sense of choking or inability to breathe; at times, these events can be difficult to distinguish from paroxysmal nocturnal dyspnea. Spouses are often terrified in this setting because "Sometimes he just stops breathing!"

Temperature. As heart failure moves toward its advanced stages, limb and cutaneous vasoconstriction reduces the body's surface temperature, as recorded by surface (eg, forehead or ear) thermometers. Recordings taken via sublingual, rectal and voiding-urine thermometers offer more precise determinations of core body temperature in this setting.

Hypothermia in patients hospitalized with heart failure is an unfavorable prognosticator. On the other hand, a forehead or ear temperature recording of 99°F or more should raise concern of a comorbidity (eg, infection or phlebitis) exacerbating the patient's heart failure.

D. Neck Veins. The light beam of a penlight directed along the neck region overlying the internal jugular vein or a well lit room greatly enhance the evaluation of the blood column in this vessel. It is often necessary to examine both sides of the neck to detect and properly assess jugular venous pulsation, pressure, and other characteristics. The methods used to measure jugular venous pressure by physical examination are numerous. Ten cardiologists will likely determine the level of jugular venous distention and pressure 10 different ways.¹ Nevertheless, the unifying theme and the essential measurement is simply the vertical distance between the mid right atrium to the top of the observable column of blood in the internal jugular vein. The position of the mid right atrium is estimated to be near the intersection of the 4th or 5th intercostal space and the midaxillary line; the vertical line to the top of

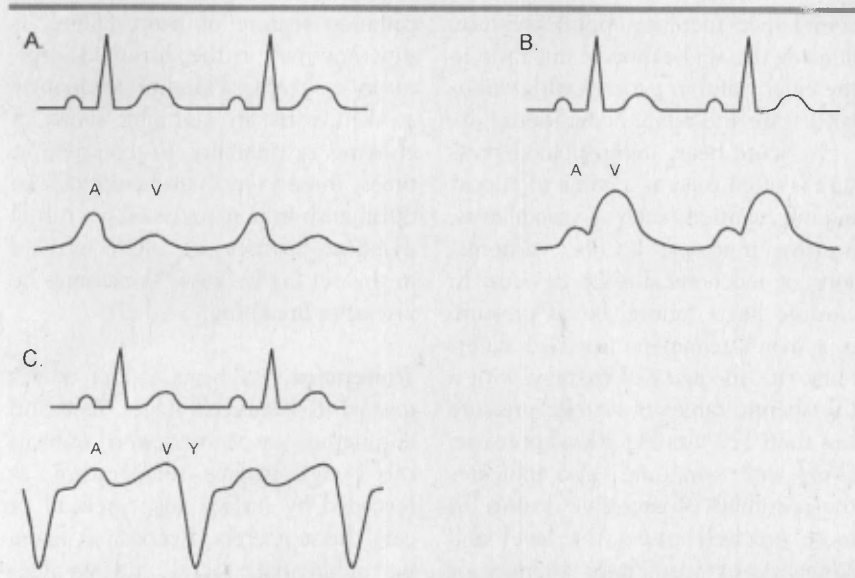


Figure 2. Sketches of some of the abnormal waveforms of the internal jugular vein in heart failure. Panel (A) shows the normal pattern when made visible during supine positioning or as recorded from an intravenous jugular catheter. The *a* wave, larger than the *v* wave, provides physical evidence that the patient is in sinus rhythm. (B) The waveform of tricuspid regurgitation. The *v* wave can be of varying sizes, but is invariably larger than the *a* wave. The *a* wave is not always apparent with a large *v* wave and can be absorbed by the upstroke of the *v* wave during tachycardia. (C) Prominent *y* descent is a common pattern of the jugular venous pulse in impaired right ventricular filling of restrictive cardiomyopathy, right ventricular hypertrophy, or constrictive pericardial disease.

the observable blood column in the internal jugular vein, irrespective of body angulation, is perpendicular to the floor. The external jugular vein, although more readily seen in most patients, can give a misleading impression and measurement of central venous pressure, particularly in the elderly with less elastic veins.

If a patient is followed by the same physician or nurse, the specific method employed is less important than the consistency of using the same method. In situations where the patient is likely to be examined by different providers over time, the most important aspect is to record the height of the column above the mid right atrium in centimeters (of blood as the actual content of the column, although centimeters of water is typically used as the pressure parameter; their relative fluid densities [specific gravities] are close enough to use either term). In other words, simply recording the observed vertical height of the column above the mid right atrium cuts through all methods and is generally the preferred

approach. If the examiner is not interested in taking the time needed to measure from the mid right atrium, recording the specific method (and then, the measurement using it) is helpful to the subsequent examiners in following the degree of venous distention and jugular venous pressure; for example, "The patient's jugular vein was noted 3 cm above the clavicle with the patient in a 45° semirecumbent position." Simply adding the vertical column height above the angle of Louis to the "presumed" distance from the angle of Louis to the mid right atrium (typically estimated as 5, 6, or 8 cm) still depends on noting body angulation and is fraught with considerable variability depending on body and chest size and habitus.

Some heart failure specialists simply examine the internal jugular veins while the patient is standing or sitting upright (90°) with legs dependent.¹ Venous distention above the clavicles, which are located about 12 to 16 cm above the right atrium in these positions for most patients, is then simply

viewed as elevated jugular venous pressure. For patients whose jugular venous activity is not noted above the clavicles, however, these body positions do not readily lend themselves to the addition of manual pressure to the abdominal region to determine the presence of hepatojugular reflux, indicative of latent or marginal volume overload in 30° or more semirecumbent positions.

A patient's jugular venous pressure can be so markedly elevated that venous pulsations along the neck are diminished or masked to the point of being difficult to detect, and thus render the false impression of nondistention and normal venous pressure. Examining the upper jugular region near the angle of the jaw and looking for prominent descents (generally, *y* descents) while the patient is inhaling (ie, inspiratory phase) or placing the patient in a standing or 90° sitting (with legs dependent) position will usually reveal jugular venous movement to uncover the elusive, marked elevation of jugular venous pressure.

The character and contour of jugular venous pulsation (*a* and *v* waves and descents); its movement with respiration (generally, a drop with inspiration); ascending distention of the jugular vein with firm abdominal pressure (to elicit venous filling via hepatojugular [abdominojugular] reflux of blood); loss of its waveforms and descents during venous occlusion (finger compression) at the base of the neck, which obliterates the transmission of waves and descents from the right atrium; palpation of the carotid pulse (typically deeper and slightly medial to the internal jugular vein); and other maneuvers allow rapid and unequivocal identification of the internal jugular vein and its clear distinction from the carotid pulse. Venous *v*-wave pulsation from tricuspid regurgitation can be strikingly prominent, and is frequently misinterpreted as a carotid pulse by less-experienced examiners. Respiratory shifts in the level of the jugular venous column (usually lower on inspiration) reflect comparable changes in intrathoracic pressure.