Jugular Venous Pulse: Window into the Right Heart

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Abstract: Although physicians began associating conspicuous neck veins with heart disease almost three centuries ago, the jugular venous pulse remains an often ignored component of the physical examination. Many physicians have not invested in the necessary understanding of the technique, and there is a misconception that its examination is difficult and of limited clinical value. When performed properly, evaluation of the jugular venous pulse can be extremely useful in distinguishing the cause of dyspnea and edema.

The normal jugular venous pulse is reviewed, and pulse wave abnormalities are described, including ways in which they can provide clues to the diagnosis of certain disease states, ranging from pericardial disease to conduction disturbances. The jugular venous pulse provides a window into the right heart and an occasional glimpse of left heart hemodynamics. By peering through this window, clinicians can gain valuable information in the diagnostic evaluation of the cardiovascular patient.

Key Words: jugular venous pulse, right atrial pressure, neck veins

Evaluation of the jugular venous pulse offers a window into the right heart, providing critical information regarding its hemodynamics. Unfortunately, there is a common impression that bedside analysis of the jugular venous pulse form is merely an academic exercise and that in actual practice, invasive measurement is required. Consequently, its analysis is an often overlooked component of the routine physical examination. We review the normal jugular venous pulse and describe how abnormalities of the pulse wave can provide clues to the diagnosis of certain disease states, ranging from pericardial disease to pulmonary hypertension to conduction disturbances.

Assessing the Jugular Venous Pulse

During assessment of the jugular venous pulse, the patient should be supine with their neck slightly turned to the left allowing for optimal visualization of the right neck. In contrast to the left internal jugular vein, the right internal jugular vein provides a more accurate estimation of right atrial pressure since it is in a direct line with the superior vena cava and right atrium. Furthermore, partial compression of the left innominate vein by the aorta impairs transmission of right pressure to the left internal jugular vein. It is classically taught that the head of the bed should be elevated to 45°.[1] The actual angle of the bed, however, is irrelevant as long as the top of the venous column is clearly seen.[2] The internal jugular vein itself lies too deep to the sternocleidomastoid muscle to be seen. However, its transmitted pulsations through the sur-

Key Points

- In patients with atrial fibrillation and severe tricuspid regurgitation, the jugular venous pulse can be easily mistaken for carotid pulsation.
- Cannon *a* waves result when there is loss of atrioventricular synchrony, a feature of several different types of arrhythmias.
- The presence of the *y* descent on physical examination can serve as a useful clue for excluding pericardial tamponade in a patient presenting with hypotension and distended neck veins.
- In contrast to traditional teaching, when the thorax is elevated to 30° or more, 10 cm should be added to the height of the venous column from the sternal angle to best obtain an estimate of the jugular venous pressure.
- In the absence of isolated right ventricular failure, a positive hepatojugular reflux suggests a pulmonary artery wedge pressure of 15 mm Hg or greater.

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rounding soft tissues to the surface of the skin are seen. After identifying the highest point of pulsation, the vertical distance between this pulsation and the sternal angle is measured.

If the venous pulsation is not observed initially, it may be either too high or too low. The position of the neck relative to the right atrium should be elevated or lowered accordingly. In patients with extremely high central venous pressure, the waveform may be best observed with the patient sitting upright at 90°. In such patients, rhythmic movement of the earlobes may be observed secondary to transmission of pulsations to the confluence of venous drainage in this area.

In many patients, the central venous pressure can be estimated by examination of the external jugular vein whose pulsations are more readily seen than the internal jugular vein during its course across the sternocleidomastoid muscle.[3,4] This method may be limited in the obese patient in whom visualization of the vein is often obscured by increased soft tissue mass and in critically ill patients.[5] In others, kinking and obstruction of the external jugular vein can occur at the base of the neck. In this instance, external jugular vein height reflects obstruction of drainage, not transmission of right atrial pressure.

The jugular venous pulse should not be mistaken for the carotid arterial pulsation. Excluding cases of absent atrial contraction (ie, atrial fibrillation), the venous pulse is distinguished by its double undulation (the *a* and *v* waves). The dominant movement of the venous pulse is inward, namely the *x* descent. Conversely, a single, sharp outward movement marks the carotid pulse.[6] Different maneuvers can be performed to further assist in differentiating between the two waveforms[7] (see Table 1). This is especially important in patients with atrial fibrillation and tricuspid regurgitation, where the absent double undulation and the prominent *v* wave of the jugular venous pulsation allow it to be easily mistaken for the carotid pulse.

Normal Jugular Venous Pulsations

The jugular venous pulse is primarily a volume wave, reflecting the ebb and flow of venous blood during contraction and relaxation of the right atrium and ventricle. Valuable information about right-sided cardiac physiology can be gleaned from careful inspection of its pulsations. Concomitant auscultation and palpation of the carotid pulse can assist the examiner in correctly identifying the components of the jugular venous pulse.

Table 1. Maneuvers used to distinguish the jugular venous pulse from the carotid pulse

Maneuver	Effect on jugular venous pulse	Effect on carotid pulse
Increasing head of bed elevation	Decreased	None
Decreasing head of bed elevation	Increased	None
Inspiration	Decreased	None (except in pulsus paradoxus)
Compression at base of neck	Obliteration of pulsations	None
Abdominal compression (HJR)	Increased	None

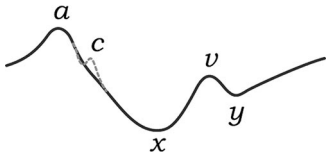

Fig. 1 A normal right atrial waveform. The *a* wave marks atrial contraction while the *x* descent represents atrial relaxation. The *v* wave marks atrial filling, and the *y* descent indicates atrial emptying.

The normal jugular venous pulse wave consists of three positive waves, *a, c,* and *v, and* two negative waves, *x* and *y* (see Fig. 1). The *a* wave is caused by right atrial systole, during which pressure is transmitted back to the jugular veins by contraction of the right atrium.[8] Since the *a* wave occurs in late diastole, it peaks just before the first heart sound and the carotid pulse upstroke. In certain individuals, it is temporally related to the fourth heart sound (S4).

The *x* descent, considered to be the most obvious motion of the venous pulse on examination,[9] occurs during systole and ends just before the second heart sound. The early portion of the *x* descent results from right atrial relaxation during atrial diastole. Although not appreciated on general examination, the *c* wave is noted on hemodynamic monitoring and interrupts the *x* descent. The *c* wave is initiated by the onset of right ventricular isovolumic systole with subsequent upward bulging of the tricuspid valve into the right atrium.[9] The *c* wave is temporally related to the first heart sound. The later portion of the *x* descent reflects the continued fall in right atrial pressure during early ventricular systole, as the tricuspid valve ring is pulled caudally by the contracting right ventricle.

The *v* wave terminates the *x* descent and results from the inflow of caval blood into the right atrium during late right ventricular systole when the tricuspid valve is still closed.[10] As the right atrium fills, its pressure begins to rise again. The *v* wave roughly coincides with the carotid pulse and peaks just after the second heart sound.

The descending limb of the *v* wave is termed the *y* descent. An early diastolic event, it is caused by the opening of the tricuspid valve and the rapid passive inflow of blood from the right atrium into the right ventricle.[11] The *y* descent occurs after the second heart sound. When present, the third heart sound corresponds to the nadir of the *y* descent. The *y* descent can be distinguished from the *x* descent due to the synchronicity of the latter with the carotid pulse; the *y* descent occurs out of phase with the carotid pulse.

Abnormal Jugular Venous Pulsations

The Abnormal *a* Wave

The amplitude of the *a* waves is heightened whenever the right atrium contracts against increased resistance. Resistance may occur *at* or *above* the level of the tricuspid valve (see Table 2). Cannon *a* waves describe exaggerated *a* waves that result when there is loss of atrioventricular synchrony. In this event, atrial and ventricular systole occur simultaneously, and the right atrium contracts against a closed tricuspid valve. Although most commonly seen intermittently with premature ventricular beats, cannon *a* waves are also observed in sustained arrhythmias with atrioventricular dissociation such as ventricular tachycardia, AV nodal reentry tachycardia and in certain modes of ventricularly paced patients. In fact, the presence of irregularly occurring cannon *a* waves can be a valuable clue in distinguishing ventricular tachycardia from supraventricular tachycardia with aberrancy in the patient with a wide complex tachyarrhythmia.[12] Wenckebach described an unusual cause of intermittent cannon *a* waves in patients with Type 1 second degree AV block in which the nonconducted P-wave occurred during the preceding QT interval.

Table 2. Abnormalities in the jugular venous pulse and corresponding causes

	Resistance at level of tricuspid valve	Resistance above level of tricuspid valve
	Obstruction of tricuspid valve	
Increased *a* waves	Tricuspid stenosis (Carcinoid, rheumatic heart disease, congenital)	Pulmonary hypertension
	Right atrial myxoma	Peripheral pulmonary arterial branch stenosis
	Lupus endocarditis	Pulmonic stenosis
	Right atrial thrombus	Right ventricular hypertrophy
	Tricuspid valve atresia	Right ventricular infarction
	Simultaneous atrial and ventricular systole	Right ventricular ischemia
	Type 1 second degree AV block	
	Ventricular pacing (DDI, VVI, VOO)	
	Isorhythmic dissociation	
	AV nodal reentry tachycardia	
	AV reentry tachycardia	
	Third degree AV bock	
	Ventricular tachycardia	
	Premature ventricular beats	
	Premature supraventricular beats (atrial or junctional)	
	Severe first degree AV block	
	Junctional tachycardia	
	Atrial systole absent	Atrial systole attenuated
Absent *a* waves	Atrial fibrillation	
	Atrial flutter	Ebstein's anomaly
	Sinus arrest	
	Blunted *x* descent	Tricuspid regurgitation
		Atrial fibrillation
	Steep x descent	Cardiac tamponade
		Constrictive pericarditis
	Increased *v* waves	Tricuspid regurgitation
		Atrial septal defect
		Arteriovenous fistula
	Steep y descent	Tricuspid regurgitation
		Constrictive pericarditis
		Restrictive cardiomyopathy
	Blunted *y* descent	Conditions associated with tricuspid stenosis
		Right atrial myxoma
		Severe right ventricular hypertrophy
		Sinus tachycardia
		Cardiac tamponade

This phenomenon can also occur in a *regular* fashion in patients with severe first degree AV block.[13]

Causes of increased *a* waves distal to the tricuspid valve include pulmonic stenosis,[14] right ventricular hypertrophy, and right ventricular myocardial infarction or ischemia. The *a* wave is enhanced secondary to atrial contraction into a stiff, noncompliant right ventricle. The presence of a right-sided S4 can be an accompanying finding. The *a* wave will not be observed during periods of atrial asystole such as atrial fibrillation, atrial flutter, or sinus arrest. Ebstein anomaly is a rare cause of absent or attenuated *a* waves in which massive progressive dilation does not allow the right atrium to generate an effective mechanical systole.

The Abnormal *x* Descent

Vigorous right ventricular contraction leads to systolic collapse of the jugular venous pulse and exaggeration of the *x* descent. This is seen in cardiac tamponade and RV overload states such as atrial septal defects. The *x* descent is attenuated or even absent in tricuspid regurgitation.[15] The transmitted ventricular pressure to the right atrium due to the regurgitant jet prevents normal atrial relaxation. In atrial fibrillation, the *x* descent is not obvious because the right atrium has no discrete period for relaxation.

The Abnormal *v* Wave

Tricuspid regurgitation classically manifests prominent *v* waves. In addition to antegrade filling of the right atrium from the cavae during ventricular systole, there is retrograde filling of the right atrium due to an incompetent tricuspid valve, causing tall *v* waves.[11] These waves have also been described as Lancisi sign or *cv* waves due to the obliteration of the *x* descent in tricuspid regurgitation as previously described.[16] In severe tricuspid regurgitation, the dominant outward motion of the giant *v* wave makes it difficult to separate the jugular venous pulse from the carotid pulse.

Prominent *v* waves are also seen in atrial septal defects. A proposed mechanism is that the addition of left-to-right interatrial shunting to normal caval venous return during ventricular systole further increases right atrial pressure.[17]

In our observations, a previously undescribed cause of prominent *v* waves is noted occasionally in hemodialysis patients. The presence of an arteriovenous fistula in these patients and the subsequent accelerated shunting of blood into the venous system can simulate prominent *v* waves. It should be noted that the amplification of the jugular venous pulse in such patients is a peripheral phenomenon and not reflective of right atrial hemodynamics.

The Abnormal *y* Descent

A slow *y* descent suggests obstruction to right atrial outflow, ie, tricuspid valve stenosis or increased resistance to right ventricular filling, as seen in severe right ventricular hypertrophy. The *y* descent can be attenuated or even absent in tachycardia due to a shortened diastole. In cardiac tamponade, early diastolic collapse of the right ventricle due to increasing intrapericardial pressure compromises right ventricular filling and can subsequently markedly attenuate the *y* descent. The presence of the *y* descent on physical examination can serve as a useful clue for excluding pericardial tamponade in a patient presenting with hypotension and distended neck veins.

A steep *y* descent is seen in diseases associated with elevated right atrial pressure and subsequent rapid right atrial ejection, such as tricuspid regurgitation, constrictive pericarditis, and restrictive cardiomyopathy. In tricuspid regurgitation, the exaggerated *y* descent results from relief of high right atrial pressure as the regurgitant enhanced right atrial volume is delivered back to the right ventricle in diastole.[11] In constrictive pericarditis, the prominent diastolic collapse is known as Friedreich sign[18] and can be accompanied by a pericardial knock on auscultation. The *y* descent typically becomes more pronounced with inspiration.

Jugular Venous Pressure

Estimation of jugular venous pressure is critical for both assessment of volume status and for detection of disease states marked by elevated right atrial pressure, such as cardiac tamponade, constrictive pericarditis, and restrictive cardiomyopathy.[19–21] Lewis in 1930[22] was the first to use the jugular vein as a manometer for the right atrium in which the height of the internal jugular venous pulsation allows for estimation of central venous pressure. The central venous pressure is classically approximated by adding 5 cm to the height of the jugular venous pulsation above the sternal angle with the patient sitting at an angle of 30 to 60°. This method assumes that the right atrium, which by convention has a venous pressure of zero, is approximately 5 cm below the sternal angle of Louis,[22] found at the junction of the manubrium and sternum at the level of the secondrib. This amount in centimeter of H_2O can be converted to mm Hg by multiplying by 0.736. The normal value for a mean right atrial pressure is 1 to 9 cm H_2O.[19] This method was challenged by Seth et al,[23] who studied the distance between the right atrium and sternal angle in 160 patients. They concluded that in the supine position, the average distance is 5.4 cm. However, at different angles of positioning, the average distance varied between 8 and 10 cm.[23] Therefore, adding 5 cm H_20 to the vertical distance measured on physical examination may be an accurate estimation of jugular venous pressure for patients in the supine position; however, when the head is elevated, this method may underestimate the true jugular venous pressure. It has been suggested that when the thorax is elevated to 30° or more, 10 cm should be added to the height of the venous column from the sternal angle to best obtain an estimate of the venous pressure[24] (see Fig. 2).

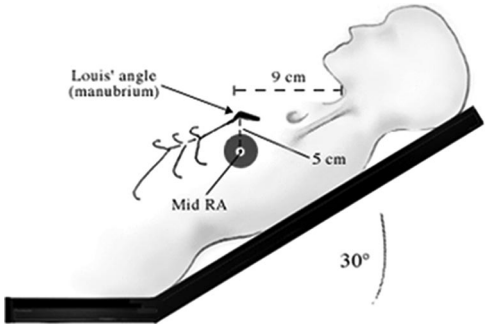

Fig. 2 Illustration of technique for measurement of central venous pressure. For angles up to 30°, 5 cm should be added to the distance measured between the angle of Louis and the highest venous pulsation. For angles greater than 30°, 10 cm should be added to this distance.

Elevated Jugular Venous Pressure

There are several causes of elevated jugular venous pressure. It usually, but not always, suggests that there is an underlying condition associated with increased right atrial pressure (see Table 3). The venous pressure is useful in discerning the etiology for edema and dyspnea. For example, in edematous states causes by decreased oncotic pressure, such as nephrotic syndrome, liver disease, and protein-losing enteropathy, intravascular volume is low and therefore neck veins should be flat on examination, ie, low jugular venous pressure. In edematous patients with renal failure and congestive heart failure, elevated jugular venous pressure is due to elevated right atrial pressure and is strongly suggestive of an elevated pulmonary artery wedge pressure.[25] In those patients with asymptomatic left ventricular dysfunction, the presence of an elevated jugular venous pressure is prognostically associated with a higher risk of progression to symptomatic heart failure.[26] Moreover, it is linked to a higher risk of hospitalization and death from heart failure in patients with symptomatic left ventricular dysfunction.[27]

In superior vena cava obstruction, the apparent elevation in jugular venous pressure is not a true reflection of right atrial pressure. External compression and collapse of the superior vena cava by a mass or complete intraluminal obstruction by thrombus prevents retrograde transmission of the right atrium waveform, leading to distended, yet nonpulsatile, neck veins.

Table 3. Causes of elevated jugular venous pressure

Normal right atrial pressure	Elevated right atrial pressure
Superior vena cava syndrome	Acute renal failure with volume overload
AV fistula	Cardiomyopathy
	Right ventricular failure
	Right ventricular infarction
	Pulmonary hypertension
	Cardiac tamponade
	Constrictive pericarditis
	Restrictive cardiomyopathy
	Severe tricuspid regurgitation
	Severe tricuspid stenosis

Hepatojugular Reflux

Patients suspected of having right heart failure may have a normal resting jugular venous pressure. The hepatojugular or abdominojugular reflux test is useful for ascertaining right ventricular reserve in these conditions. First described by William Pasteur in 1885,[28] this test is marked by application of firm, sustained pressure over the upper right abdominal quadrant for 10 to 15 seconds while the patient maintains normal quiet breathing. This maneuver increases intra-abdominal pressure and the pressure gradient for venous flow from the abdomen to the thorax, resulting in augmented venous return to the right heart. The failing right heart is unable to handle this increased preload, and the jugular venous pressure remains elevated during sustained abdominal pressure.[29] Care should be taken not to elicit an induced Valsalva response since the associated increase in intrathoracic pressure will negate the intended increase in venous return from the hepatojugular reflux.

In normal individuals, the jugular venous pressure should increase only transiently by approximately 1 cm, but will normalize during the remainder of the maneuver.[30,31] In patients with right ventricular failure, however, sustained elevation of jugular venous pressure greater than 3 cm is observed during continued compression.[30] In addition to right heart failure, the hepatojugular reflux may be positive in constrictive pericarditis, restrictive cardiomyopathy, and tricuspid regurgitation. In the absence of right ventricular infarction or isolated right ventricular failure, a positive hepatojugular reflux reflects left heart hemodynamics and suggests a pulmonary artery wedge pressure of 15 mm Hg or greater.[31]

Kussmaul Sign

Inspiration generates a negative intrapleural pressure, creating a vacuum-like effect for augmenting flow through the right heart into the lungs, which results in a decline in right atrial and jugular venous pressure. Lack of decrease or an increase in jugular venous pressure during inspiration is known as Kussmaul sign.[32] Although the mechanism is poorly understood, some contend that the inspiratory increase in venous pressure is the direct consequence of an inspiratory increase in abdominal pressure transmitted through an already venous system to a right heart that is unable to accommodate greater diastolic distention.[33] Classically described in constrictive pericarditis,[34] the most common cause of Kussmaul sign is severe right-sided heart failure regardless of etiology.[34] In patients with ST elevation of the inferior wall, the presence of Kussmaul sign is strongly suggestive of a coexisting right ventricular myocardial infarction, exceeding 90% specificity and sensitivity.[35] It is also commonly seen in severe tricuspid

regurgitation. Contrary to historical teachings, Kussmaul sign is rarely ever seen in pericardial tamponade.[36]

Summary

Although physicians began associating conspicuous neck veins with heart disease almost three centuries ago, the jugular venous pulse remains an often ignored component of the cardiovascular physical examination. Many physicians have not invested in the necessary understanding of the technique, and there is a misconception that examination of the jugular venous pulse is difficult, time consuming, and of limited clinical value.

When performed properly, evaluation of the jugular venous pulse can be extremely useful in distinguishing the cause of dyspnea and edema. Attention to the components of the waveform can yield subtle clues to underlying cardiac diagnoses such as cardiac tamponade, constrictive pericarditis, and ventricular tachycardia. Furthermore, performance of the hepatojugular reflux and Kussmaul tests offer supplemental information concerning compromised right ventricular function and elevated left atrial pressure.

The jugular venous pulse provides a window into the right heart and an occasional glimpse of left heart hemodynamics. By peering through this window, clinicians can gain valuable information in the diagnostic evaluation of the cardiovascular patient.

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