Jugular Venous Pulse: Window into the Right Heart

Patrick J. Devine, MD, Lance E. Sullenberger, MD, Daniel A. Bellin, MD, and J. Edwin Atwood, MD

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.
contraction (i.e., atrial fibrillation), the venous pulse is distin-
guished from the carotid arterial pulsation. Excluding cases of absent atrial
obstruction of drainage, not transmission of right atrial pressure.
the neck. In this instance, external jugular vein height reflects
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.

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. 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. 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 (i.e., atrial fibrillation), the venous pulse is distin-
guished 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. Different maneuvers can be
performed to further assist in differentiating between the two
waveforms (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 contrac-
tion and relaxation of the right atrium and ventricle. Valuable
information about right-sided cardiac physiology can be
gleaned from careful inspection of its pulsations. Concomi-
tant auscultation and palpation of the carotid pulse can assist
the examiner in correctly identifying the components of the
jugular venous pulse.

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. 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 tem-
porarily related to the fourth heart sound (S4).

The x descent, considered to be the most obvious motion
of the venous pulse on examination, 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 exami-
nation, 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 up-
ward bulging of the tricuspid valve into the right atrium. 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 tricus-
pid 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. 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 de-
scent. 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. The y descent oc-
curs 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.

![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.](image)

<table>
<thead>
<tr>
<th>Maneuver</th>
<th>Effect on jugular venous pulse</th>
<th>Effect on carotid pulse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increasing head of bed elevation</td>
<td>Decreased</td>
<td>None</td>
</tr>
<tr>
<td>Decreasing head of bed elevation</td>
<td>Increased</td>
<td>None</td>
</tr>
<tr>
<td>Inspiration</td>
<td>Decreased</td>
<td>None (except in pulsus paradoxus)</td>
</tr>
<tr>
<td>Compression at base of neck</td>
<td>Obliteration of pulsations</td>
<td>None</td>
</tr>
<tr>
<td>Abdominal compression (HJR)</td>
<td>Increased</td>
<td>None</td>
</tr>
</tbody>
</table>

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

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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 ventriculally 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

<table>
<thead>
<tr>
<th>Resistance at level of tricuspid valve</th>
<th>Resistance above level of tricuspid valve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased a waves</td>
<td></td>
</tr>
<tr>
<td>Obstruction of tricuspid valve</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>Tricuspid stenosis (Carcinoid, rheumatic heart disease, congenital)</td>
<td>Peripheral pulmonary arterial branch stenosis</td>
</tr>
<tr>
<td>Right atrial myxoma</td>
<td>Pulmonic stenosis</td>
</tr>
<tr>
<td>Lupus endocarditis</td>
<td>Right ventricular hypertrophy</td>
</tr>
<tr>
<td>Right atrial thrombus</td>
<td>Right ventricular infarction</td>
</tr>
<tr>
<td>Tricuspid valve atresia</td>
<td>Right ventricular ischemia</td>
</tr>
<tr>
<td>Simultaneous atrial and ventricular systole</td>
<td></td>
</tr>
<tr>
<td>Type 1 second degree AV block</td>
<td></td>
</tr>
<tr>
<td>Ventricular pacing (DDI, VVI, VOO)</td>
<td></td>
</tr>
<tr>
<td>Isorhythmic dissociation</td>
<td></td>
</tr>
<tr>
<td>AV nodal reentry tachycardia</td>
<td></td>
</tr>
<tr>
<td>AV reentry tachycardia</td>
<td></td>
</tr>
<tr>
<td>Third degree AV bock</td>
<td></td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td></td>
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<tr>
<td>Premature ventricular beats</td>
<td></td>
</tr>
<tr>
<td>Premature supraventricular beats (atrial or junctional)</td>
<td></td>
</tr>
<tr>
<td>Severe first degree AV block</td>
<td></td>
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<tr>
<td>Junctional tachycardia</td>
<td></td>
</tr>
<tr>
<td>Atrial systole absent</td>
<td>Atrial systole attenuated</td>
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<tr>
<td>Atrial fibrillation</td>
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<tr>
<td>Atrial flutter</td>
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<tr>
<td>Sinus arrest</td>
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<tr>
<td>Blunted x descent</td>
<td></td>
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<tr>
<td>Steep x descent</td>
<td></td>
</tr>
<tr>
<td>Increased v waves</td>
<td></td>
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<tr>
<td>Tricuspid regurgitation</td>
<td></td>
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<tr>
<td>Atrial septal defect</td>
<td></td>
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<tr>
<td>Cardiac tamponade</td>
<td></td>
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<tr>
<td>Constrictive pericarditis</td>
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<tr>
<td>Tricuspid regurgitation</td>
<td></td>
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<tr>
<td>Restrictive cardiomyopathy</td>
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<tr>
<td>Steep y descent</td>
<td></td>
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<tr>
<td>Tricuspid regurgitation</td>
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<tr>
<td>Constrictive pericarditis</td>
<td></td>
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<tr>
<td>Blunted y descent</td>
<td></td>
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<tr>
<td>Conditions associated with tricuspid stenosis</td>
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<tr>
<td>Right atrial myxoma</td>
<td></td>
</tr>
<tr>
<td>Severe right ventricular hypertrophy</td>
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<tr>
<td>Sinus tachycardia</td>
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<tr>
<td>Cardiac tamponade</td>
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</table>
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 hypotrophy. 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 sign18 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 193022 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 H2O 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 H2O.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 H2O 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 pressure24 (see Fig. 2).
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 caused 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 asymptomatic 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

<table>
<thead>
<tr>
<th>Normal right atrial pressure</th>
<th>Elevated right atrial pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava syndrome</td>
<td>Acute renal failure with volume overload</td>
</tr>
<tr>
<td>AV fistula</td>
<td>Cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>Right ventricular failure</td>
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<td></td>
<td>Right ventricular infarction</td>
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<td></td>
<td>Pulmonary hypertension</td>
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<tr>
<td></td>
<td>Severe tricuspid regurgitation</td>
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<tr>
<td></td>
<td>Severe tricuspid stenosis</td>
</tr>
</tbody>
</table>

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.

References

34. Cook D, Simel D. Does this patient have abnormal central venous pressure? JAMA 1996;275:630–634.

Please see Jack P. Chen’s editorial on page 968 of this issue.