

The Abdominojugular Reflux Sign

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PURPOSE: The abdominojugular reflux sign is useful in diagnosing right ventricular failure, but is often performed and interpreted incorrectly. Our objective was to review the history, epidemiology, and pathophysiology of the abdominojugular reflux sign.

METHODS: We conducted a MEDLINE search of the English language reports published between 1966 and 1999 and a manual search of bibliographies of relevant papers.

RESULTS: A positive abdominojugular reflux sign is defined by an increase in the jugular venous pressure of greater than 3 cm, sustained for greater than 15 seconds. In the absence of left heart failure, a positive abdominojugular reflux sign should prompt consideration of impaired right ventricular preload, a decrease in right ventricular compliance, a decrease in right ventricular systolic function or an elevation in right ventricular afterload. In patients presenting with dyspnea, the abdomino-

jugular reflux is useful in predicting congestive heart failure (LR+ 6.0 (95% CI; 0.8–51); LR– 0.78; (95% CI: 0.62 to 0.98)) and suggests pulmonary capillary wedge pressures of greater than 15 mm Hg (LR+ 6.7 (95% CI: 3.3 to 13.4); LR– 0.08 (95% CI: 0.01 to 0.52)).

CONCLUSION: The abdominojugular reflux is not specific to any one disorder, but rather is a reflection of a right ventricle that cannot accommodate augmented venous return. Constrictive pericarditis, right ventricular infarction, and restrictive cardiomyopathy are common causes of a positive finding. Left ventricular failure may also induce the sign, but only when the pulmonary capillary wedge pressure is greater than 15. The one diagnosis not seen with abdominojugular reflux is cardiac tamponade. *Am J Med.* 2000;109:59–61. ©2000 by Excerpta Medica, Inc.

In 1885, Pasteur (1) described an increase in jugular venous pressure in patients with tricuspid insufficiency after the application of pressure to the liver: “In several cases in which there was reason to suspect functional incompetence of the tricuspid valve, a physical sign has been present. This sign consists in a distension of the superficial veins of the neck, occurring when firm pressure is exerted over the liver in the direction of the spinal column.” Carvallo (2) extended this observation by noting that deep inspiration augmented murmurs of tricuspid insufficiency. He correctly postulated that the negative intrathoracic pressure during inspiration increased venous return to the right ventricle, thereby accentuating right-sided murmurs.

Kussmaul had described a paradoxical increase in the jugular venous pressure during inspiration in patients with constrictive pericarditis. Deep inspiration, Kussmaul reasoned, should have decreased jugular venous distension. “Inspiration generates a negative intrapleural pressure, which sucks the venous blood into the heart,” except in constrictive pericarditis when, “...the blood sucked into the chest cannot enter the heart, and the venous pressure rises” (2). In patients with “constrictive pericarditis and right ventricular failure, but not tamponade,” jugular venous pressure increased with inspiration,

suggesting that obstruction to flow into the right ventricle was responsible for the sign (3).

Rondot (4) subsequently realized that patients with constrictive pericarditis or right ventricular failure should exhibit the same sign with the application of abdominal pressure. If abdominal pressure increased venous return (as in Pasteur’s observation), and if constrictive pericarditis or right ventricular failure prevented accommodation of venous return (as in Kussmaul’s findings), then patients with a noncompliant right ventricle should exhibit a sustained increase in jugular distension when pressure was applied to the abdomen.

PERFORMANCE OF THE SIGN

A positive abdominojugular reflux sign is defined by an increase in the jugular venous pressure of greater than 3 cm that is sustained for longer than 15 seconds. The patient should be positioned so that the jugular venous pressure can be seen. There should be at least a 3-cm margin from the baseline jugular venous pressure to the angle of the jaw. Slow, steady abdominal pressure of 20 to 35 mm Hg should be applied to the middle of the abdomen. Sustained pressure for 15 seconds is sufficient to elicit the sign (5).

It is not necessary to apply pressure to the liver, as the principle of hydraulics is fulfilled by midline pressure to the abdomen. Midabdominal pressure may be preferable, as pressure on the liver may be sufficiently painful to elicit a Valsalva response. The augmented positive intrathoracic pressure from the Valsalva response will negate the venous inflow to the right heart induced by the abdominal pressure. For this reason, it is important that patients

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Manuscript submitted October 20, 1999, and accepted in revised form March 31, 2000.

Table. Physical Examination Signs Elicited by Increasing Venous Return

Sign	Maneuver	Response	Diagnosis
Pasteur's sign	Abdominal pressure	Increased jugular venous pressure	Tricuspid insufficiency (1)
Carvallo's sign	Deep inspiration	Increased murmur	Tricuspid insufficiency versus mitral insufficiency (17)
Kussmaul's sign	Deep inspiration	Sustained increase in jugular venous pressure	Constrictive pericarditis (18), right ventricular infarction (19), restrictive cardiomyopathy, pulmonary hypertension (6)
Rondot's sign (abdominojugular reflux)	Abdominal pressure	Sustained increase in jugular venous pressure	Constrictive pericarditis, right ventricular infarction, restrictive cardiomyopathy, pulmonary hypertension, right heart failure (12)

not experience discomfort or hold their breath during the examination. Until the physician can reliably reproduce the same pressure with each examination, a blood pressure cuff can be laid across the abdomen as a manometer to ensure consistency.

PHYSIOLOGIC PRINCIPLES UNDERLYING THE SIGN

Abdominal pressure increases venous return to the right heart, with a subsequent rise in the central venous pressure. In patients with normal cardiac function, this effect is transient, as the right heart compensates with an augmented right ventricular output (Starling's effect). Sustained central venous distension is indicative of failure of the right side of the heart to accommodate increased venous return.

To accommodate increased venous return, right ventricular output must increase. An elevation in right ventricular afterload due to pulmonary hypertension (6) or severe left ventricular failure (5,7,8) decreases right ventricular output. A decrease in systolic function due to myocardial infarction (9) or cardiomyopathy similarly reduces right ventricular stroke volume. Impaired right ventricular preload, as seen with tricuspid stenosis, prevents the increase in right ventricular output that is normally seen with increased venous return.

In the absence of signs of left- or right-side heart failure, a positive abdominojugular reflux sign suggests reduced right ventricular compliance. Constrictive pericarditis (10) and restrictive cardiomyopathy (11) reduce right ventricular compliance, thereby impairing the right ventricle's ability to accommodate the increased blood flow induced by abdominal compression.

The mechanism of sustained central venous pressure is analogous to a hydraulic brake system in an automobile. Pressure exerted on the abdomen compresses the veins in the mesenteric bed, increasing venous return to the right heart. A positive sign occurs when the venous circuit is engorged with blood (7) and there is an obstruction of

flow through the right ventricle (5). A patient with only one of these two conditions (ie, tricuspid stenosis with a low central venous volume) will not have the sign. This may account for the absence of this sign in patients who have been treated with diuretic agents.

Other mechanisms have been proposed for the abdominojugular reflux sign. Burch and Ray (7) proposed that it is due to increased sympathetic tone in patients with heart failure. However, the sign is not associated with either systemic or pulmonary vascular resistance, and this theory fails to explain the sign's presence in constrictive pericarditis, right ventricular infarction, and restrictive cardiomyopathy. Because the sign is due to hemodynamic shifts and not to a reflex arc, it is best called abdominojugular reflux rather than abdominojugular reflex.

Hultgren and Hamosh (12) have suggested that external hepatic pressure may cause a local tamponade of the right ventricle owing to upward movement of the diaphragm. When studied, however, the mean diaphragmatic excursion during abdominal compression was only 4 mm, hardly sufficient to induce tamponade (5). This theory also does not explain why midabdominal pressure, as opposed to right upper quadrant pressure, induces the same sign, or why paralysis of the diaphragm does not cause abdominojugular reflux.

Both Rondot and Kussmaul agreed that tamponade does not induce an elevated jugular venous pressure with their maneuvers. Although blood return to the right heart is increased in both maneuvers, in tamponade the right heart maintains its capacity to accommodate this extra volume.

CLINICAL EPIDEMIOLOGY OF THE SIGN

There are no prospective studies assessing the sensitivity and specificity of abdominojugular reflux in predicting tricuspid stenosis, constrictive pericarditis, right ventricular infarction, or primary pulmonary hypertension. Kussmaul's sign (sustained jugular venous pressure ele-

vation with inspiration) is seen in 33% of patients with pure constrictive pericarditis (13,14) and, 33% to 100% of patients with right ventricular infarction (9). To the extent that Kussmaul's sign is due to a similar physiology, abdominojugular reflux may have similar predictive ability.

The value of this sign in congestive heart failure has been a subject of great controversy (5). In patients with dyspnea, the abdominojugular reflux sign had a sensitivity of 24% and a specificity of 96% for detecting congestive heart failure (likelihood ratio if the sign is present [LR+] of 6.0, 95% confidence interval [CI] 0.8 to 51; likelihood ratio if the sign is absent [LR-] of 0.78, 95% CI 0.62 to 0.98) (15). Other investigators have suggested that abdominojugular reflux is specific only for right-sided heart failure or constrictive pericarditis (12). The heterogeneous nature of patients included in these studies may explain this disparity. Ewy (16) noted only a weak association with left ventricular ejection fraction but a strong association with an elevated pulmonary capillary wedge pressure. Forty-three of 44 patients with a negative sign had a pulmonary capillary wedge pressure <15 mm Hg, whereas 14 of 21 patients with a positive sign had a pulmonary capillary wedge pressure >15 mm Hg (LR+ 6.7, 95% CI 3.3 to 13.4; LR- 0.08, 95% CI 0.01 to 0.52). Butman et al (8) studied 52 patients referred for heart transplant. The presence of either an elevated jugular venous pressure or an abdominojugular reflux sign had a sensitivity of 81% and a specificity of 80% in predicting a pulmonary capillary wedge pressure of greater than 18 mm Hg (LR+ 4.1, 95% CI 1.7 to 9.9; LR- 0.23, 95% CI 0.11 to 0.50).

These studies suggest that in patients with heart failure, the sign is related to the degree of decompensation of the left ventricle as manifested by an elevated pulmonary capillary wedge pressure. Rondot's 1898 observations concur, noting that the probability of observing the sign correlates with the severity of the heart failure. "The hepatojugular reflux is usually seen in state of low output cardiac failure of cardiac or aortic origin when decompensation occurs. Its disappearance usually coincides with the disappearance of the symptoms of cardiac failure" (3). In Butman's study (8), the abdominojugular reflux sign was positive in 11 patients who had no jugular venous distension. The mean (\pm SD) pulmonary capillary wedge pressure of these patients was 22 mm Hg (\pm 5), suggesting that the abdominojugular reflux sign may be seen in patients with left-side heart failure even when right-side heart failure is not clinically apparent.

CONCLUSION

The abdominojugular reflux sign should not be considered specific to any one disorder but rather to indicate a right ventricle that cannot accommodate an increase in venous return (Table). Constrictive pericarditis, right

ventricular infarction, and restrictive cardiomyopathy are common causes of a positive finding; correctly performed, the abdominojugular reflux sign is a useful tool in diagnosing each. Left ventricular failure may also induce the sign, but only when the pulmonary capillary wedge pressure is greater than 15 mm Hg. All conditions require both inadequate right ventricular accommodation and a venous system that is full. Cardiac tamponade does not cause abdominojugular reflux.

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