Chapter 3
Assessment and Management of Tricuspid Valve Regurgitation

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Physical Exam in Tricuspid Regurgitation

Clinical evaluation of tricuspid regurgitation (TR) begins with cardiac auscultation. Classically, a pansystolic murmur at the left lower sternal border that increases with inspiration (Carvallo sign) is apparent with TR. The Carvallo sign, first described in 1946, is due to augmented venous return that accompanies inspiration and consequently intensifies regurgitant tricuspid flow. Maneuvers that increase venous return, such as hepatic compression, leg raise, and exercise, will also intensify the murmur. This has been demonstrated via phonocardiography tracings in the right atrium (RA) (Fig. 3.1) [1]. The presence of the Carvallo sign is a specific, not sensitive exam finding, as 20% of patients with moderate to severe TR were negative for this clinical finding in a sampled population [2]. In contrast, maneuvers that decrease venous return (Valsalva, standing) will reduce the intensity of the murmur.

With the progression of TR valvulopathy, the right ventricle (RV) can dilate. The murmur may then extend to the right lower sternal border, subxiphoid region, and toward the apex. In the setting of a dilated, failing RV, an S3 gallop can often be appreciated in the subxiphoid area.

Lancisi's sign, or jugular venous distension (JVD), with exaggerated "cv" wave, is visualized on neck exam. It is a consequence of the TR jet occurring during right ventricular systole with simultaneous venous return to the right atrium. The cv wave represents an exaggerated and broadened v wave usurping the normal c wave (closure of the tricuspid valve) and x descent (atrial relaxation) rendering the jugular venous pulsation monophasic (Fig. 3.2). Given this appearance and the fact that it is occasionally palpable, the cv wave may be mistaken for a carotid pulsation. However, the comparably latent upstroke, prominent y descent, and respirophasic changes that characterize jugular venous pressures can be recognized with a trained

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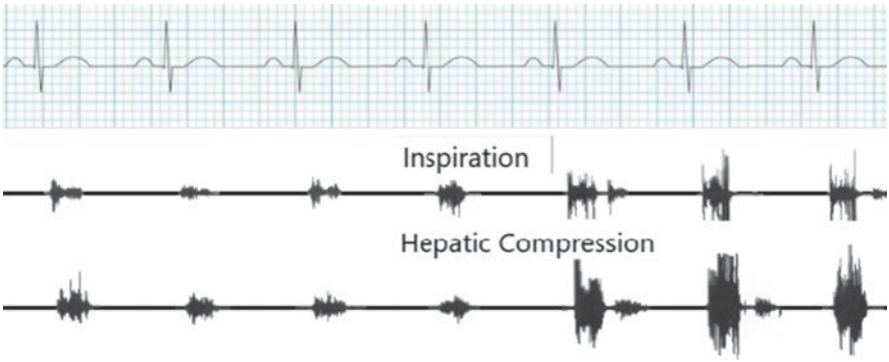

Fig. 3.1 Representation of phonocardiography tracing in the right atrium of a patient with TR demonstrating intensifying murmur with inspiration and applied hepatic pressure

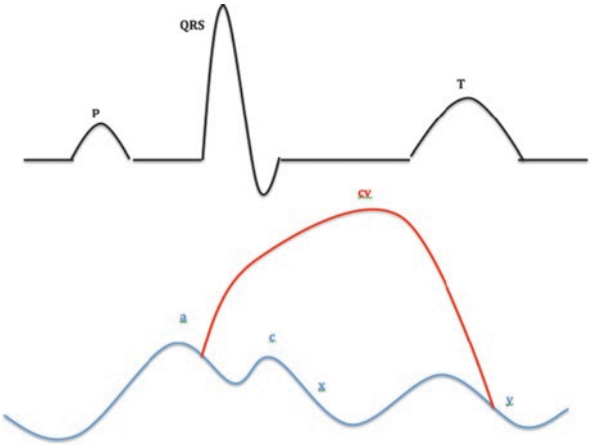

Fig. 3.2 Normal central venous pressure waveform (blue line) and change in waveform seen in severe tricuspid regurgitation (red line) with a fusion of c and v waves and absence of the x descent

Table 3.1 Commonly described physical exam findings in advanced tricuspid regurgitation

Maneuver	Description
Carvallo sign	Increase in TR murmur with deep inspiratory hold [3]
Lancisi's sign	JVD with exaggerated cv wave during systole [4]
Kussmaul's sign	Increase in jugular venous pulse with inspiration [5]
Augmented Carvallo	Increase in TR murmur with manual liver compression [6]

eye. Kussmaul's sign is another abnormality of the jugular venous waveform due to TR. It is the increase in the jugular venous pulse with maneuvers increasing venous flow (inspiration, hepatic compression, exercise, and leg raise) (Table 3.1).

Assessment of volume status is integral to characterizing the consequences of TR and guides clinical management. Patients may present with hepatomegaly, ascites, and peripheral edema that reflect both the severity and chronicity of TR

Table 3.2 Numerical rating of lower extremity edema and associated exam findings

Grades of pitting edema	
0+	No pitting edema
1+	Mild; small depression that disappears immediately
2+	Moderate; depression disappears <10 seconds
3+	Moderate-severe; depression disappears 10–20 seconds
4+	Severe; depression that lasts >20 seconds

(Table 3.2). Liver congestion may progress to the point the liver is tender and pulsatile upon palpation. In rare cases, this pulsatility can be visualized in lower extremity varicose veins [7].

Despite a number of characteristic physical findings, these are generally insensitive for TR. Indeed, clinical findings are often subtle or absent in mild or moderate TR, and these cases are most often diagnosed incidentally via echocardiography [8]. Early investigation to delineate the prevalence of diagnostic features relied on invasive measurements such as intracardiac phonography and right ventriculography due to the lack of noninvasive imaging. In these studies, the triad of the Carvallo sign, prominent v waves, and pulsatile liver was specific but not sensitive. All patients with this triad were found to have severe TR; however, only 30% of patients with severe TR displayed all three physical exam findings. Sensitivity improved when the Carvallo sign was used alone or in conjunction with prominent v waves or pulsatile liver [1]. Addition of manual hepatic pressure increases the murmur of TR, identifying the Carvallo sign in an additional 17.6% of patients who lacked inspiratory murmur augmentation at baseline [6].

Medical Management of Tricuspid Regurgitation

Conventional medical therapy for patients with TR is reflected in a recent review of patients with severe TR at a tertiary care center. A total of 87 patients were identified over the preceding 2 years, with the majority receiving medical therapy ($n = 65$) rather than valve repair or replacement ($n = 22$). Of the entire cohort, nearly all were on diuretics (98%), while a minority were treated with aldosterone antagonists (35%). It is important to note that frequently implicated causes of secondary TR, namely atrial fibrillation and mitral regurgitation, were prevalent in this cohort [9].

Diuretics Diuretics decrease renal sodium reabsorption and encourage the movement of fluid into the urine, thereby reducing total body water. They are broadly classified into one of three categories depending on their site of action within the nephron (loop, thiazide, or potassium-sparing diuretics). While there is little randomized data on diuretics in heart failure, they relieve congestion and improve symptoms and exercise capacity [10].

There are no trials that address the effect of diuretic therapy on TR severity or subsequent outcomes. However, in patients with TR and evidence of congestion (hepatomegaly, ascites, lower extremity edema), diuretics are reasonable in an effort to achieve symptomatic relief. For maintenance therapy, an agent with greater oral bioavailability (torsemide, bumetanide) may be preferable to furosemide, although the efficacy of this strategy is not established. Other measures, such as salt and fluid restriction, daily weights, and tailored diuretic dosing, are also reasonable in these cases.

Potassium-sparing diuretics such as aldosterone antagonists are occasionally employed in patients with significant TR. These are often used in tandem with loop diuretics when hypokalemia is encountered. The hyperaldosteronism that accompanies liver cirrhosis or chronic right ventricular volume and pressure overload make this class of diuretic reasonable to prescribe when TR is accompanied by these conditions [9, 11]. Although being logical based on pathophysiology, inhibition of the renin–angiotensin–aldosterone system has not been established to preserve right ventricular or tricuspid valve function.

Pulmonary Hypertension Moderate or greater TR is observed in 20–50% of patients with pulmonary hypertension (PH), and portends a worse overall prognosis [11–14]. When TR is encountered in the setting of pulmonary hypertension, therapies that address the underlying cause of PH (Groups 2–5) or pulmonary pressures directly (Group 1 or recalcitrant PH) have variable reported effects on TR severity [13]. It is important to keep in mind when reviewing these studies, however, that a reduction in pulmonary pressures, not grade of TR, was the primary goal. Accordingly, the analyses regarding the effects of these agents on TR are often underpowered, or not reported.

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