

# Tricuspid Valve Disease in Older Adults: Diagnosis and Management

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## Introduction

Tricuspid valve disease is rarely an isolated condition. Most cases are associated with other valvular or myocardial disease, pulmonary hypertension or systemic disorders. The tricuspid valve is located in the outflow tract of the right ventricle, and is the largest heart valve with an area of approximately 11cm<sup>2</sup>. The valvular apparatus includes the fibrous annulus, the leaflets (anterior, septal and posterior), the tendinae chordae and the papillary muscles. Given that the tricuspid valve's main function is to regulate inflow to the right ventricle, conditions affecting the tricuspid valve generally have an impact on the right atrium and the venous circulation. Similarly, disorders affecting the left or right ventricle or the pulmonary arterial system can impair tricuspid valve function.

This review focuses on the most common causes of tricuspid stenosis (TS) and regurgitation (TR) in older adults. In these patients, functional tricuspid regurgitation is by far the most frequent tricuspid disorder. In the evaluation of tricuspid valve disorders, a thorough physical examination is essential to provide information for a correct diagnosis. An overview of the most useful ancillary tests and treatment options is also presented.

## Tricuspid Stenosis

TS is characterized by progressive thickening with calcification and fusion of the leaflets, and frequently co-exists with TR and mitral-aortic valvular disease.<sup>1</sup> Rheumatic fever is the most frequent cause of TS worldwide, and other causes are uncommon. Atrial tumours or thrombi can cause tricuspid pseudostenosis

due to obliteration of the valve during ventricular filling in diastole. Placement of endocavitary catheters, such as pacemakers and defibrillators, can induce fibrosis of the leaflets and cause significant stenosis. Infectious endocarditis is exceptional in patients who are not intravenous drug users, shared needles being one of the means by which infection can enter the bloodstream. Carcinoid tumours metastatic to the liver may lead to TS and TR.

## Diagnosis

TS should be suspected in a patient with chronic right-sided heart failure, jugular vein distension, hepatomegaly, ascites and lower-extremity edema. A strong S1 heart sound and a diastolic murmur that increases with inspiration are clinical clues for the diagnosis TS.<sup>1</sup> A wide P wave on the electrocardiogram reflects atrial enlargement. Doppler-echocardiography determines the degree of stenosis by morphological and pressure-gradient criteria.<sup>2</sup>

## Management

Most cases of TS respond to medical treatment, most commonly with diuretics. If medical treatment is not sufficient to control symptoms, valve repair or replacement is indicated when the valve area is less than 2cm<sup>2</sup> or diastolic pressure gradient is greater than 5 mmHg.<sup>2,3</sup> Valvulotomy, in which the valve is opened surgically, can be effective for the rare cases of isolated TS. However, due to associated TR or significant mitral valve disease, more common procedures of choice are surgical annuloplasty or valve replacement with a bioprosthesis. Accurate anticoagulation

is required postoperatively due to the high risk of thromboembolism, even with use of bioprostheses.<sup>4</sup>

## Tricuspid Regurgitation

In most cases, TR is functional, caused by right ventricular dysfunction secondary to cardiac or pulmonary disease. For appropriate management, one must distinguish between TR with a normal tricuspid valve and TR with an abnormal tricuspid valve.

### Tricuspid Regurgitation with Anatomically Abnormal Valve

*Ebstein's anomaly* is the only congenital abnormality of the tricuspid valve with which the patient can be expected to have a long life expectancy. Ebstein's anomaly is characterized by a displaced attachment of the tricuspid valve into the ventricular cavity. With this low implantation, the chordae are shorter and the amplitude of movement of the tricuspid leaflets is limited, resulting in TR. The portion of the right ventricle that lies between the atrioventricular ring and the valve is "atrialized", with a thinned and dilated myocardium. Depending on the severity of tricuspid incompetence and the amount of atrialized right ventricle, the natural history of Ebstein's anomaly ranges from neonatal death to normal life expectancy.<sup>2,5</sup>

Although complaints of fatigue and dyspnea on exertion are most common, patients with Ebstein's anomaly may also be asymptomatic. A third of patients experience atrial tachycardia—often associated with Wolff-Parkinson-White accessory pathways—and may present with syncope due to a rapid ventricular response. A systolic murmur, grade 2 or 3, can be heard on the left side of the sternum. Doppler echocardiography demonstrates displacement of the tricuspid valve and the presence of TR. Symptomatic patients will benefit from repair of the tricuspid valve with anatomic recon-

struction or valve replacement, usually with a homograft.<sup>5</sup>

Isolated *tricuspid valve prolapse* occurs rarely. The condition is due to myxomatous degeneration of the leaflets and the chordae, and can be associated with annulus dilatation. More often, tricuspid prolapse is found in association with mitral valve prolapse. Tricuspid prolapse has a benign course and almost never requires specific treatment, except in the case of severe regurgitation or infectious endocarditis.<sup>1</sup>

As with TS, the most common cause of TR is *rheumatic fever*. TR is usually associated with mitral and aortic valve rheumatic disease,<sup>6</sup> and is far more common than TS in patients with rheumatic fever with coexisting mitral stenosis—40% versus 5%, respectively.<sup>2</sup> Fibrosis and calcification of the leaflets and chordae are characteristic of rheumatic tricuspid valve disease. Heart valve disease of rheumatic origin usually manifests by the fourth decade of life, but diagnosis at an advanced age is not uncommon. Treatment depends primarily on presence and degree of mitral or aortic valve disease.

*Endocarditis* as primary infection of the tricuspid valve represents only 1% of the cases of infectious endocarditis. Elderly patients with endocavitary devices, such as pacemakers or implantable defibrillators, are at higher risk of contracting tricuspid endocarditis.<sup>7</sup> The infection is manifested by fever and septic pulmonary embolism, and the most common causative organism is *Staphylococcus aureus*. Transthoracic echocardiography is the best imaging technique to visualize vegetations on the leaflets and chordae of the tricuspid valve. Management includes intravenous antibiotics for at least two weeks and, if applicable, removal of the endocavitary device. If the infection persists following these initial therapeutic measures, removal of the valve may be necessary, with optional valve replacement with a bioprosthesis.<sup>7,8</sup>

*Traumatic TR* may be caused by catheters, such as pacemakers or

implantable defibrillator electrodes, or by repeated endomyocardial biopsies in transplanted patients.<sup>9</sup> Accurate information on the status of the different valve components, which can be provided by echocardiography, is necessary for proper valve repair for severe TR.

*Connective tissue diseases* and systemic inflammatory disorders can be associated with TR. Marfan syndrome is one example of a dilatation of the annulus that is responsible for TR. Systemic lupus erythematosus and rheumatoid arthritis can also involve the tricuspid valve.<sup>10</sup>

Tricuspid valve involvement may occur in the course of *carcinoid syndrome*, the constellation of symptoms typically exhibited by patients with metastases from carcinoid tumours. Substances secreted by these tumours (e.g., serotonin) can affect the right atrium and right ventricle, the tricuspid and pulmonary valves and the pulmonary artery. Smooth muscle cell proliferation and collagen deposition characterize valvular lesions. Medical treatment is ineffective, and options of valve repair or replacement depend upon the prognosis of the carcinoid tumour. In general, however, presence of valve disease implies a poor prognosis.<sup>11</sup>

#### Tricuspid Regurgitation with Anatomically Normal Valve

In the absence of any specific disease of the tricuspid valve, TR is the result of the dilatation of the annulus, secondary to a right ventricle enlargement. TR also can be a consequence of pulmonary hypertension, which increases the right ventricle afterload. In these cases, TR can improve after normalization of the pulmonary hypertension.

*Right ventricle myocardial infarction* frequently presents with hypotension or cardiogenic shock due to a decreased right ventricular output. In this situation, TR is secondary to dilatation of the damaged ventricle, as well as ischemia of the chordae and papillary muscles.<sup>12</sup> Treatment during the acute phase includes percutaneous revascularization of the culprit artery—usually the right

coronary—when appropriate, use of inotropics (e.g., dobutamine) and re-establishment of an adequate ventricular rate with isoproterenol or a temporary pacemaker.

*Pulmonary hypertension* can also lead to TR. Precapillary hypertension is due to primary pulmonary hypertension or other pulmonary diseases that induce increased pulmonary vascular resistance with normal pressure in the pulmonary veins (e.g., cor pulmonale and terminal stages of chronic pulmonary disease). Postcapillary hypertension is due to left-sided heart failure (e.g., mitral valve disease, left ventricular failure).<sup>13</sup> Pulmonary embolism, whether acute or chronic, is a frequent cause of pulmonary hypertension. Extensive embolism can induce sudden pressure overload of the right ventricle, with dilatation and acute TR. In this case, immediate repermeabilization of the pulmonary vascular tree is required to preserve right ventricular output.<sup>14</sup>

#### Diagnosis

Jugular distension, ascites, edema and a pulsatile hepatomegaly are common signs of TR.<sup>1,3</sup> A pansystolic murmur that increases with inspiration can be heard on the left sternal border (until right ventricular failure develops and the ventricle is unable to increase filling and stroke volume during inspiration).<sup>1</sup> Atrial fibrillation also is common.

Transthoracic echocardiography is useful in determining whether TR is anatomic or functional, and to assess the area of regurgitation, the degree of pulmonary hypertension and the right ventricle size and function. Analysis of blood flow pattern within the vena cava and hepatic veins provides additional information on the severity of TR. Transesophageal echocardiography generally is not a useful diagnostic tool in tricuspid valve disorders, as the valve is in a plane far from the esophagus.

#### Management

Medical treatment with diuretics and sodium restriction is beneficial. If regurgitation is severe, annuloplasty is indi-

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cated in cases of functional TR or—depending on the degree of regurgitation and assessment of reversibility of the TR—surgical valve repair or replacement can be used. When TR is associated with left-sided heart disease, treatment depends on the primary condition. For patients with severe mitral valve disease, pulmonary hypertension and TR degrees less than 2/4, surgery on the tricuspid valve is not required if

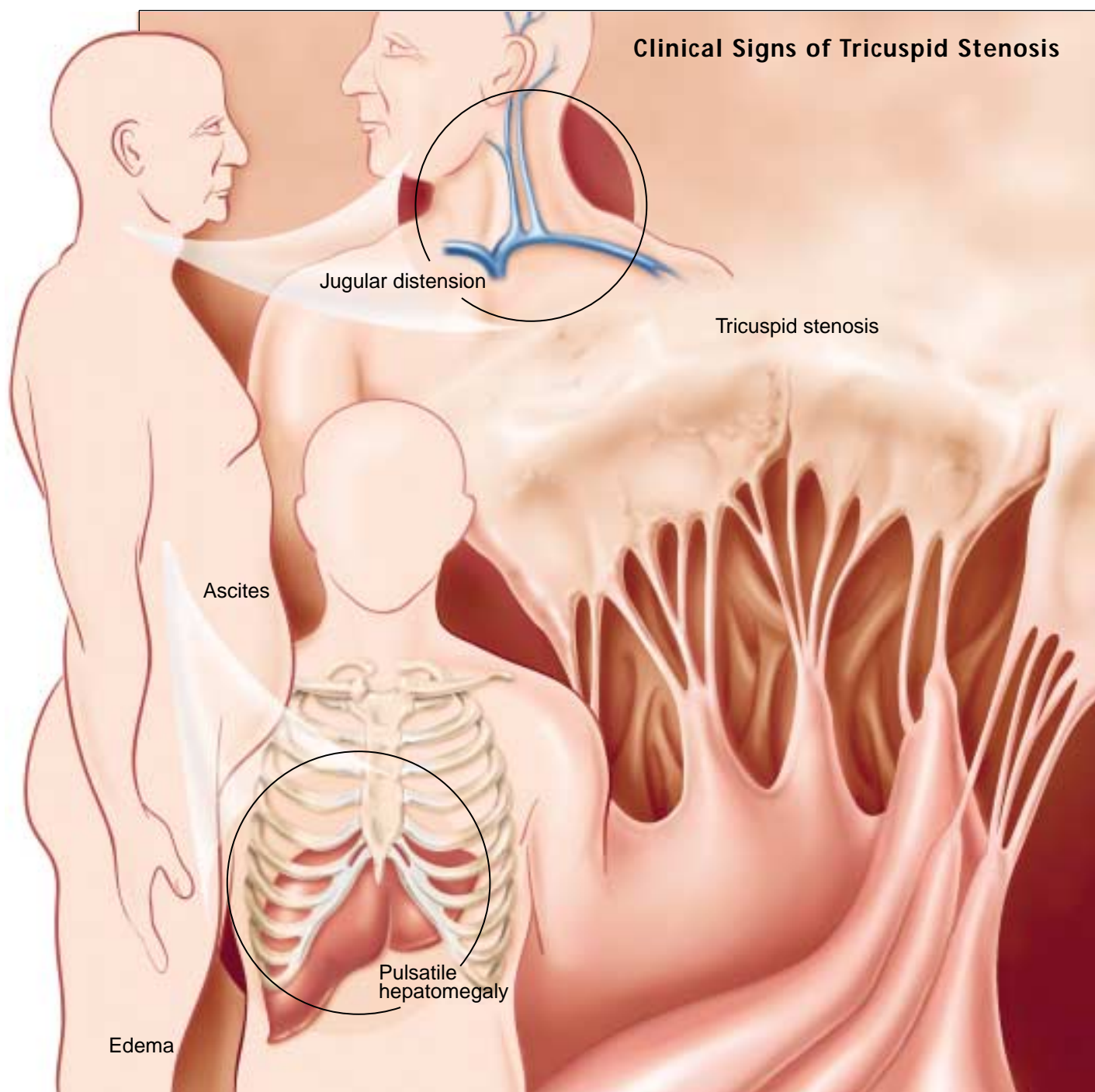
there is no significant impairment of the right ventricular function. In these cases, TR is likely to reverse after mitral valve replacement. With higher degrees of regurgitation (3/4 to 4/4) and right ventricular dilatation or dysfunction, tricuspid annuloplasty or replacement is indicated, depending on the extent and severity of lesions.<sup>8,15</sup>

Elderly patients may have a higher operative risk due to the associated

comorbidities. The most prominent risks are a decreased left ventricular ejection fraction and presence of coronary artery disease. Nevertheless, patients in good risk categories should benefit from surgical options such as repair or replacement of valvular lesions.<sup>16</sup>

## Conclusions

Tricuspid valve disease in older adults is usually associated with pulmonary or



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left-sided heart disease. Proper management of the coexisting diseases may improve the symptoms of tricuspid dysfunction. Valve abnormalities, whether due to rheumatic fever, tumours, collagen diseases or other causes, need to be addressed in the context of the primary disease. Medical treatment can be effective in cases of mild or moderate tricuspid valve dysfunction, but if dysfunction is severe, valve repair by annuloplasty or replacement may be required. Increased operative risk attributed to age alone should not preclude the surgical correction of severe valve disease. ♦

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## References

1. Braunwald E. Heart disease: A textbook of cardiovascular medicine. 5th edition. Philadelphia: WB Saunders, 1997.
2. Waller BF, Howard J, Fess S. Pathology of tricuspid valve stenosis and pure tricuspid regurgitation. Part II. *Clin Cardiol* 1995;18:167-74.
3. Kratz J. Evaluation and management of tricuspid valve disease. *Cardiol Clin* 1991;9:397-407.
4. Guerra F, Bortolotti U, Thiene G, et al. Long-term performance of the Hancock porcine bioprosthesis in the tricuspid position. A review of forty-five patients with fourteen-year follow-up. *J Thorac Cardiovasc Surg* 1990;99:838-45.
5. Mair DD. Ebstein's anomaly: Natural history and management. *J Am Coll Cardiol* 1992;19:1047-8.
6. Daniels SJ, Mintz GS, Kotler MN. Rheumatic tricuspid valve disease. Two-dimensional echocardiographic, hemodynamic, and angiographic correlations. *Am J Cardiol* 1983;51:492-6.
7. Nandakumar R, Raju G. Isolated tricuspid valve endocarditis in nonaddicted patients: a diagnostic challenge. *Am J Med Sci* 1997;314:207-12.
8. Barbour DJ, Roberts WC. Valve excision only versus valve excision plus replacement for active infective endocarditis involving the tricuspid valve. *Am J Cardiol* 1986;57:475-8.
9. Williams MJ, Lee MY, DiSalvo TG, et al. Biopsy-induced flail tricuspid leaflet and tricuspid regurgitation following orthotopic cardiac transplantation. *Am J Cardiol* 1996;77:1339-44.
10. Laufer J, Frand M, Milo S. Valve replacement for severe tricuspid regurgitation caused by Libman-Sacks endocarditis. *Br Heart J* 1982;48:294.
11. Robiolio PA, Rigolin VH, Wilson JS, et al. Carcinoid heart disease: correlation of high serotonin levels with valvular abnormalities detected by cardiac catheterization and echocardiography. *Circulation* 1995;92:790-5.
12. Dhainaut JF, Ghannad E, Villemant D, et al. Role of tricuspid regurgitation and left ventricular damage in the treatment of right ventricular infarction-induced low cardiac output syndrome. *Am J Cardiol* 1990;66:289-95.
13. Kessler KM, Willens HJ, Mallon SM. Diastolic left ventricular dysfunction leading to severe reversible pulmonary hypertension. *Am Heart J* 1993;126:234-5.
14. Lualdi JC, Goldhaber SZ. Right ventricular dysfunction after acute pulmonary embolism. Pathophysiologic factors, detection, and therapeutic implications. *Am Heart J* 1995;130:1276-82.
15. Breyer RH, McClenathan JH, Michaelis LL, et al. Tricuspid regurgitation. A comparison of nonoperative management, tricuspid annuloplasty, and tricuspid valve replacement. *J Thorac Cardiovasc Surg* 1976;72:867-74.
16. Femes SE, Goldman BS, Ivanov J, et al. Valvular surgery in the elderly. *Circulation* 1989;80:177-90.