Tricuspid regurgitation in left ventricular assist device patients

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A useful clinical report should convey a single important message. In this journal, Krishnan et al. [1] show that concomitant correction of tricuspid valve regurgitation does not increase operative mortality for patients receiving an implantable left ventricular assist device (LVAD). This confirms the previous report of Pal et al. [2] who defined the impact of concurrent cardiac procedures on mortality in HeartMate II LVAD patients in a bridge to transplant clinical trial. These authors recorded a 5.8% operative mortality rate for patients requiring uncomplicated LVAD implantation with no apparent increase in risk for concurrent mitral or tricuspid repair. By contrast, patients who underwent an aortic valve procedure manifest a 30-day mortality rate of 25%.

There is understandable reticence to complicate or prolong LVAD surgery in the high-risk heart failure patient. This begs the question ‘Is tricuspid annuloplasty really beneficial?’. In patients with left ventricular dysfunction or mitral valve disease, elevated left atrial pressure is transmitted backwards through the lungs as pulmonary hypertension [3]. This produces pressure overload and dilatation of the right ventricle with annular enlargement and secondary tricuspid regurgitation. The regurgitant fraction impairs forward flow through the lungs, thereby reducing LVAD filling and cardiac output overall. Elevated right atrial pressure and systolic reversal of flow in the vena cava are responsible for end-organ venous congestion and the signs and symptoms of tricuspid regurgitation.

When the tricuspid annulus dilates, it is the mural part that increases in length. The straight (septal) side retains its dimension [4]. The right ventricular papillary muscle attachments arise directly from the upper septum and from variable attachments to the trabecular septum or free wall, close to the septum. As such, they are little affected by remodelling and right ventricular dilatation of the free wall. Thus, stretching of the mural annulus is the dominant mechanism in functional tricuspid regurgitation.

In a classic angiographic study, Simon et al. [5] showed impaired contraction of the stretched tricuspid annulus to be the predominant mechanism of tricuspid regurgitation. Fukada et al. [6] demonstrated an asymmetric reduction in annular contraction, which does not resolve when pulmonary artery pressure falls. The annulus becomes dilated, flattened and circular. Uncorrected moderate or severe tricuspid regurgitation perpetuates right ventricular dysfunction and is associated with progressive heart failure and premature death [5].

Functional tricuspid regurgitation can be dynamic and responsive to medical therapy. It is possible to identify gross insufficiency, which weeks later has become mild in response to diuretics. It is important to adopt liberal indications for repair and regard previous episodes of moderate or severe regurgitation (or a substantially dilated annulus) as an indication for surgery, particularly for destination therapy patients. When the right ventricle fails, increased diastolic pressure can cause septal shift and compression of the left ventricle, which may impair LVAD filling. When the LVAD reduces left ventricular volume, tricuspid regurgitation may be exacerbated acutely because of leftward shift of the intraventricular septum and increased venous return in response to LVAD flow [7].


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EDITORIAL COMMENT

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Functional tricuspid regurgitation can be dynamic and responsive to medical therapy. It is possible to identify gross insufficiency, which weeks later has become mild in response to diuretics. It is important to adopt liberal indications for repair and regard previous episodes of moderate or severe regurgitation (or a substantially dilated annulus) as an indication for surgery, particularly for destination therapy patients. When the right ventricle fails, increased diastolic pressure can cause septal shift and compression of the left ventricle, which may impair LVAD filling. When the LVAD reduces left ventricular volume, tricuspid regurgitation may be exacerbated acutely because of leftward shift of the intraventricular septum and increased venous return in response to LVAD flow [7].

Even when an LVAD reduces pulmonary artery pressure, tricuspid insufficiency tends to be progressive in the long term, eventually reducing transpulmonary flow and pump output. Our own experience shows that this is problematic for destination therapy patients who survive for several years and whose pump output falls when tricuspid regurgitation worsens. Krishan et al. [1] adopted an aggressive policy towards tricuspid repair, employing a rigid ring designed anatomically for the tricuspid valve. Virtually three-quarters of 51 consecutive LVAD patients underwent ring annuloplasty, which on average added 20 min to the duration of surgery. Navia et al. [8] recently reviewed the efficacy of different types of tricuspid repair and demonstrated that rigid prosthetic ring annuloplasty and the Kay technique of bi-cuspidisation by obliteration of the mural leaflet provided similar satisfactory long-term outcome. The Kay technique is simple, rapid and reliable [9]. In the Navia series, 75% of patients with bicuspidisation commissuroplasty remained free of significant tricuspid regurgitation, as did 69% of patients with ring annuloplasty. Other authors have reported high recurrence of tricuspid regurgitation following the De Vega repair, particularly in patients with severe annular dilatation or pulmonary hypertension [10].

In conclusion, persistent tricuspid regurgitation contributes to morbidity and mortality following LVAD implantation. Tricuspid ring annuloplasty or the Kay technique can provide an effective solution without increasing operative mortality. The aggressive approach to tricuspid repair as advocated by Krishnan et al. is justified particularly when prolonged support is anticipated.

REFERENCES