Tricuspid prolapse diagnosed by echocardiographic studies and echocardiographic tricuspid prolapse also demonstrated angiographically have been reported. Correlation of tricuspid prolapse with phonocardiographic findings has not been made, and tricuspid prolapse in the absence of mitral prolapse has not been shown conclusively.

CASE REPORT

A 48-year-old Puerto Rican albino man who had essential hypertension controlled with diet and diuretic therapy and who had sarcoid with bilateral hilar adenopathy was noted to have an extra systolic sound. Echocardiographic studies were performed using a commercially available echocardiograph (Smith-Kline) with a 1.6-MHz 10-cm focused transducer that was 13 mm in diameter; data were recorded on a strip chart recorder (Smith-Kline Ekoline 21). The transducer was placed in the third left intercostal space, where the mitral valve was recorded with the transducer perpendicular to the wall of the chest. The transducer was then angled medially to the tricuspid valve, which could be demonstrated only in full expiration.

Figure 1 demonstrates prolapse of the anterior leaflet of the tricuspid valve, with the nonejection systolic click occurring at the time of maximal prolapse. Figure 2 shows no prolapse of either leaflet of the mitral valve in the presence of the nonejection systolic click. In inspiration the systolic click merged with the second heart sound.

DISCUSSION

Tricuspid prolapse has been demonstrated in 12 pa-

Echocardiographic Tricuspid Prolapse and Nonejection Systolic Click*

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Echocardiographic prolapse of the anterior leaflet of the tricuspid valve was shown to coincide with a nonejection systolic click with origin in the right side of the heart. In the presence of the systolic click, neither leaflet of the mitral valve prolapsed.

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Figure 1. Arrows point to nonejection systolic click on phonocardiogram (Phono) and systolic prolapse of anterior tricuspid leaflet on echocardiogram. TV, tricuspid valve.
patients, eight of whom had mitral prolapse. The auscultatory studies were reported as confirmed by phonocardiographic studies, but Figure 3 of the article by Chandraratna et al is the only phonocardiogram presented, and it does not demonstrate a nonejection systolic click. No other discussion of auscultatory findings was given. Although there may have been evidence for the lack of prolapse of the mitral valve in four patients, such evidence is not presented graphically.

Horgan et al reported the findings in a patient with atrial septal defect who had angiographic evidence for prolapse of the tricuspid valve. Figure 2 of that report has several echoes in systole which represent various portions of mitral leaflets. Several of these echoes strongly suggest prolapse of the mitral valve.

Maranhao et al reported simultaneous echocardiographic study of the tricuspid valve and phonocardiographic studies which did not demonstrate a systolic click or late systolic murmur. Tricuspid prolapse was shown angiographically. Augmentation of late systolic murmur or of systolic clicks (or both) by inspiration was noted as a noninvasive clue to tricuspid prolapse, but no further explanation was given.

Gooch et al reported on angiographic studies of mitral and tricuspid prolapse in which one patient of six with tricuspid prolapse by angiographic criteria had the transient appearance of a late systolic murmur in inspiration. That is not what one would expect in tricuspid prolapse, in which inspiration would increase right ventricular end-diastolic volume and result in a later appearance or disappearance of the nonejection click and late systolic murmur. The difficulty of proving prolapse of the tricuspid valve angiographically has been discussed by Horgan et al, Maranhao et al, and Gooch et al, as well as elsewhere.

Midsystolic clicks that were demonstrated in two heroin users who developed bacterial endocarditis were presented as arising from structures of the tricuspid valve; however, one of the patients had a nonejection click which did not change position in systole during inspiration, a feature not expected of a nonejection click arising from structures of the tricuspid valve. No echocardiographic, angiocardio-graphic, or surgical data were presented for either patient.

Cineangiographic studies have demonstrated billowing posterior mitral leaflets reaching the peak of prolapse at the time of a nonejection systolic click. The association of nonejection systolic clicks with systolic movement of the posterior leaflet in echocardiographic prolapse of the mitral valve has also been reported.

Prolapse of the mitral valve may be present and not be demonstrable echocardiographically; however, the presence of tricuspid valvular prolapse echocardiographically coinciding with a nonejection systolic click phonocardiographically with respiratory changes typical of a tricuspid nonejection click strongly supports the presence of tricuspid prolapse in the absence of mitral prolapse. These findings are analogous to findings previously reported in prolapse of the mitral valve.