K+, there is an increase conductance of K+ such that the ratio of $[K^+]^1$ is reduced and loss of negativity $[K^+]^0$
(depolarization) results.

Thus, perhaps again in an overly simplistic explanation, the effect of K+ depends on the type of cell, what property, e.g., automaticity or conduction is under consideration. Perhaps, more importantly, explanations for some of the observed electrophysiological effects of K+, defy accurate description and must await further study.

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Tricuspid Valve in Ebstein's Anomaly

To the Editor:

In the article by Lundstrom (Circulation 47: 597, 1973), the author studied 19 patients with Ebstein's anomaly by echocardiography. The tricuspid valve (TV) was found to be in an abnormally anterior position during the entire period of diastole. The speed of movement of the valve in diastole (E to F slope) could be measured in 10 patients and was found to average 22 mm/sec, a considerably reduced measurement.

This is contrary to the data obtained from a study of six patients with Ebstein's anomaly from our laboratory. The diastolic closure rate was normal in four patients and reduced in two patients. As evidenced by other reports, the diastolic closure rate of the TV appeared normal or minimally reduced. An abnormal anterior position of the TV during the entire period of diastole was observed in one patient. In the same group two patients exhibited a peculiar anterior motion of the TV during the early part of diastole, a finding that occurred in the mitral valve echogram in a patient with Ebstein's anomaly reported on by Tajik and his colleagues.

One of the most specific echocardiographic features of Ebstein's anomaly is the delayed or late closure of the TV. The exact mechanism of the late closure of the TV has not been elucidated but it can occur in patients with Type B Wolff-Parkinson-White syndrome where the right ventricle is depolarized early. This observation has cast doubt on the theory that the late systolic closure is due to right bundle branch block, the electrocardiographic pattern that is frequently present in Ebstein's anomaly. It is the author's belief that a mechanical factor directly related to the malformed TV with its large anterior leaflet is mainly responsible for the delayed closure. However this is contradicted in his observation that the amplitude of movement of the echo of the TV was not of special value in establishing the diagnosis of Ebstein's anomaly. Furthermore in 12 of his 19 patients, the amplitude of the TV was greater than that of the mitral valve (MV). In our present series of six patients, five patients exhibited a greater amplitude of movement of the TV (mean 42 mm) than the MV (mean 23 mm). One patient exhibited equal magnitude of TV and MV movement. Thus, if an almost two-fold increase in amplitude of movement of the TV as compared to the MV is recorded, Ebstein's anomaly should be seriously considered.

However the absence of this finding does not exclude Ebstein's anomaly. Excessive anterior motion of the TV can also occur in the hypoplastic left heart syndrome.

The degree of motion of the anteriorleaflet of the TV will depend upon anatomic variations. If the anterior leaflet is a large sail-like structure that retains part of its attachment to the true annulus, then the characteristic echocardiographic feature of excessive amplitude of movement of the TV will be recorded. However, if the leaflet is bound down to the atrialized portion of the right ventricle or to the annulus, normal or even diminished amplitude of movement of the TV will occur. The anterior leaflet of the TV may be functionally competent, incompetent or stenosed and thus a variable diastolic closure slope may be recorded by echocardiography.

It appears that no single pattern of movement of the TV is distinct for Ebstein's anomaly except delayed or late systolic closure and increased closing velocity of the TV (unpublished data).

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References


The author replies:

To the Editor:

Dr. Kotler's observations on the echocardiographic findings in Ebstein's anomaly of the tricuspid valve are somewhat different from those I have reported. To me this difference underlines the great variability of the malformation of the tricuspid valve (TV) in this anomaly. Additional reports about the echocardiographic findings in Ebstein's anomaly are therefore

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valuable. From a previous report by Dr. Kotler it seems as if his material consists mainly of adult patients while my material includes several infants and children. It is therefore likely that our materials differ in degree of malformation of the tricuspid valve. Dr. Kotler's letter has to be commented upon further on some points.

Before Dr. Kotler's letter arrived I had been informed that a normal speed of movement in diastole of the TV can be found in Ebstein's anomaly. This was found in an adult patient with a mild form of Ebstein's anomaly at this hospital and also shown in a recently published report. It is thus quite clear that the speed of movement in diastole of the TV in Ebstein's anomaly can be normal or reduced. A reduced diastolic closure slope could indicate a stenosed TV. This interpretation seems not likely however since there were no signs of tricuspid stenosis at catheterization and angiocardiography in our patients. To me it seems more likely that the reason for the reduced speed of movement of the TV in diastole is a reduced tricuspid valve flow. A corresponding reduced speed of movement of the mitral valve (MV) in diastole has been described in patients with a reduced mitral valve flow without mitral stenosis.

In all reports published on echocardiography in Ebstein's anomaly a delayed closure of the TV compared with the MV has been found. This, therefore, seems to be the most consistent specific echocardiographic finding in Ebstein's anomaly. The late TV closure even in a patient with Ebstein's anomaly and a type B ventricular preexcitation was also found in one of our patients. The reason for the delayed closure is still a matter of debate. It is possible that mechanical factors may be responsible for the delayed closure. It is, however, not necessary that these factors are related to the amplitude of movement of the anterior tricuspid leaflet. It is also possible that the delayed closure can be due to an altered pattern of ventricular contraction as suggested by Tajik et al. and in accordance with the findings of Fontana and Woolley.

The amplitude of movement of the anterior tricuspid leaflet is often increased but can be normal or even reduced. A reduced amplitude of movement was found recently in a newborn with Ebstein's anomaly (unpublished data). This infant died and autopsy revealed a severely malformed tricuspid valve where even the anterior leaflet was underdeveloped. In infants and children with various forms of congenital heart malformation it is not unusual to have a TV with a greater amplitude of movement than that of the MV. Because of this it was concluded that the amplitude of movement of the TV was not of special value in establishing the diagnosis of Ebstein's anomaly. I can, however, agree with Dr. Kotler that Ebstein's anomaly should be considered if an excessive amplitude of movement of the TV is found.

In his last sentence Dr. Kotler states that an increased closing velocity is distinct for Ebstein's anomaly. I have also observed this increased closing velocity but only in the patients with a great amplitude of movement of the TV. I think that this increased closing velocity is dependent upon a great amplitude of movement of the TV and related to the fact that the anterior tricuspid leaflet is in an unusually anterior position at the end of diastole. In some patients with Ebstein's anomaly the closing velocity has been found to be normal (unpublished data).

As a conclusion I would like to summarize: In Ebstein's anomaly of the tricuspid valve abnormal echocardiographic findings from the tricuspid valve region have been found in all cases reported. The constant and most specific finding has been a delayed tricuspid valve closure compared with the mitral valve closure. The amplitude of movement of the anterior tricuspid valve is often increased, but may be normal or even reduced. In cases with an increased amplitude of movement an increased closing velocity has been found. The speed of movement in diastole of the anterior tricuspid leaflet is often reduced but may be normal.

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Effects of Exercise on LVET

To the Editor:

In (Circulation 48: 74, 1973) Spodick and Quarry-Pigott study the effects of upright exercise on a bicycle ergometer on the left ventricular ejection time (LVET). With moderate upright ergometer exercise they concluded that there was no change in the LVET despite an increase in heart rate of approximately 37% above control. Despite the fact that there are some differences between walking exercise on a treadmill and sitting exercise on a bicycle ergometer, I feel that the readers could be misled into believing that upright exercise does not produce a change in the left ventricular ejection time. This is not true.

Our own studies have involved direct measurement of intraarterial pressure with a small catheter threaded up to the axillary artery in healthy subjects during walking exercise on a treadmill. In some of these subjects cardiac outputs were also measured simultaneously. Our results indicate that when subjects go from the supine to the standing position there is a fall in LVET of the order of magnitude of 50-100 milliseconds and that this is associated with a marked fall in stroke volume and a slight rise in heart rate. Immediately with

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Tricuspid Valve in Ebstein's Anomaly: The author replies:
NILS-RUNE LUNDSTRÖM

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