*Original Article*

# SINGLE-DOSE COLD MODIFIED BRETSCHNEIDER SOLUTION FOR MYOCARDIAL PROTECTION IN THE SURGICAL TREATMENT OF PATIENTS WITH AORTIC VALVE STENOSIS AND LEFT VENTRICULAR HYPERTROPHY

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## Summary

Aortic valve stenosis (AS) is predominantly caused by degenerative calcification in patients over 70 years. It obstructs the left ventricular outflow tract. The result is concentric left ventricular hypertrophy (LVH) wall stress and ischemia-induced myocardial fibrosis. [1] According to the European Association of Cardiothoracic Surgery, all high-grade AS patients are indicated for Aortic valve intervention [2]. One of the most recommendable methods for myocardial protection during surgical aortic valve replacement is antegrade delivery of cold crystalloid cardioplegia. Our retrospective study included 173 operated patients with AS and LVH. We observed the MB fraction of the enzyme creatine phosphokinase (CPK-MB) as a postoperative marker for myocardial hypoprotection in different patients. Our results showed that a single dose of modified Bretschnaider solution provided safe and long enough myocardial protection during conventional and minimally invasive aortic valve replacement in patients with AS and LVH [3].

**Keywords:** antegrade cardioplegia, Bretschneider solution, myocardial protection

## Introduction

Surgical aortic valve replacement under cardiac arrest is the approved treatment in patients with AS [2].

Application of HTK (Histidine, Tryptophan, Ketoglutarate) solution was reported by Bretschneider in the 1970s [4]. It is classified as intracellular, crystalloid cardioplegia due to its low sodium and calcium content. Sodium depletion of the extracellular space causes a hyperpolarization of the myocyte plasma membrane, inducing cardiac arrest in diastole. The mechanism of action differs from conventional 'extracellular' cardioplegic solutions with high potassium content and leads to cardiac arrest by membrane depolarization [5] (Table 1).

The high histidine content buffers the acidosis caused by the accumulation of anaerobic metabolites during the long ischaemic period, ketoglutarate improves adenosine triphosphate production during reperfusion, tryptophan stabilizes the cell membrane, and mannitol decreases cellular edema and acts as a

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