

**EFFECT OF VANADATE OLIGOMERS ON THE LUSITANIAN
TOADFISH HEART (*Halobatrachus didactylus*): FUNCTIONAL
NONINVASIVE ANALYSIS**

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Introduction

Vanadium effects on biological systems have become an important area of research due to the continuous increase of vanadium in the environment and to the pharmacological effects of some of its forms (Barceloux, 1999).

In biological systems vanadium exists mainly in the oxidation states +4 (vanadyl) and +5 (vanadate) (Barceloux, 1999). Previous studies have identified several physiopathological effects of vanadate on the cardiovascular system, such as negative inotropy and essential hypertension (Carmignani et al., 1998). Recently, it was suggested that different vanadate species, namely monomeric and decameric species, may contribute differently to oxidative stress and to histopathologic changes on cardiac tissue (Aureliano et al., 2002; Borges et al., 2002), which may be related to cardiac dysfunction.

In order to explore this hypothesis, systolic and diastolic functions of Lusitanian toadfish heart were analysed, using echocardiography, after acute *in vivo* exposure to “metavanadate” or “decavanadate” solutions.

Material and Methods

Specimens of the Lusitanian toadfish (n=10, body weight ranging 362-983 g) were caught at Ria Formosa lagoon (south coast of Portugal) and were divided in two groups: Meta and Deca, injected i.p. with 5 mM “metavanadate” or “decavanadate” solutions (1ml/Kg), respectively. Echocardiographic examinations were performed on anaesthetised animals at day 0 (control), 1 and 7 after metal intoxication, as described elsewhere (Coucelo et al., 1996).

Spectral records of systolic ventricular flow velocity were obtained by Doppler echocardiography and were used to determine: 1) maximum velocity (m/s), corresponding to peak of ejection flow; and 2) systolic time intervals: including pre ejection period, ventricular ejection time, acceleration time, deceleration time and total electromechanic systole. The Bernoulli equation was used to convert peak flow velocity into the pressure difference between the ventricle and the bulbus arteriosus. The Doppler velocity spectral records of ventricular filling flow, were used to determine maximum velocity (m/s), of both early (E wave) and late (A wave) ventricular filling and several diastolic time intervals: isovolumetric relaxation time, early filling period, diastasis, late filling period and total diastole.

All reported values are averages of at least five measurements in different cardiac cycles, and represent the % difference between values for control fish (day 0), and values for fish injected with “metavandate” or “decavandate”

Results and Discussion

“Metavandate” significantly reduced the systolic flow peak velocity ($p<0.05$), after both 1 and 7 days (Figure 1). However, decavanadate” only reduced cardiac function after 1 day. By day 7, peak systolic flow was not different from that in control fish. The pressure gradient between the ventricle and bulbus and stroke volume varied in accordance with ventricular systolic peak velocity.

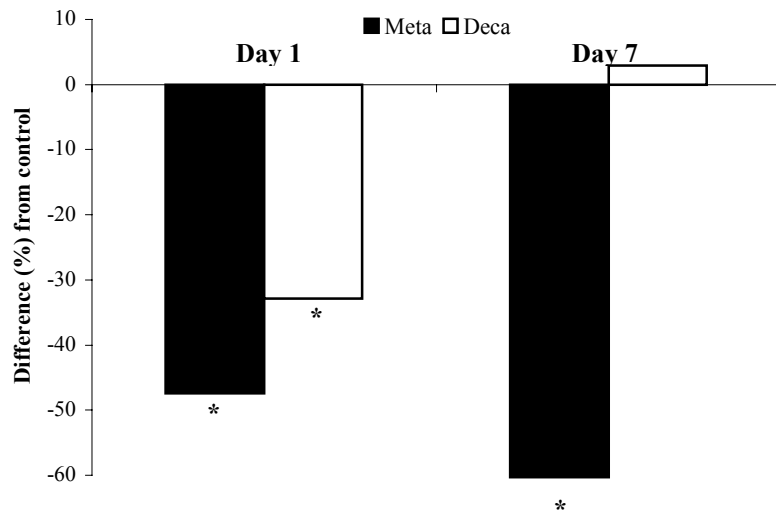


Figure 1. Effect of “metavanadate” or “decavanadate” solutions (5 mM) on systolic peak flow velocity at 1 and 7 days-post-injection; * $p < 0.05$.

The maximum velocities of ventricular filling were also changed in the presence of vanadium: E and A waves velocities diminished significantly after 1 day in both groups, and after 7 days in just the Meta group (Figure 2). These results indicate a reduced ventricle filling, which may be related to decreased stroke volume. On other hand, the reduction of both E and A waves indicates a deficient ventricular relaxation and atrial contraction.

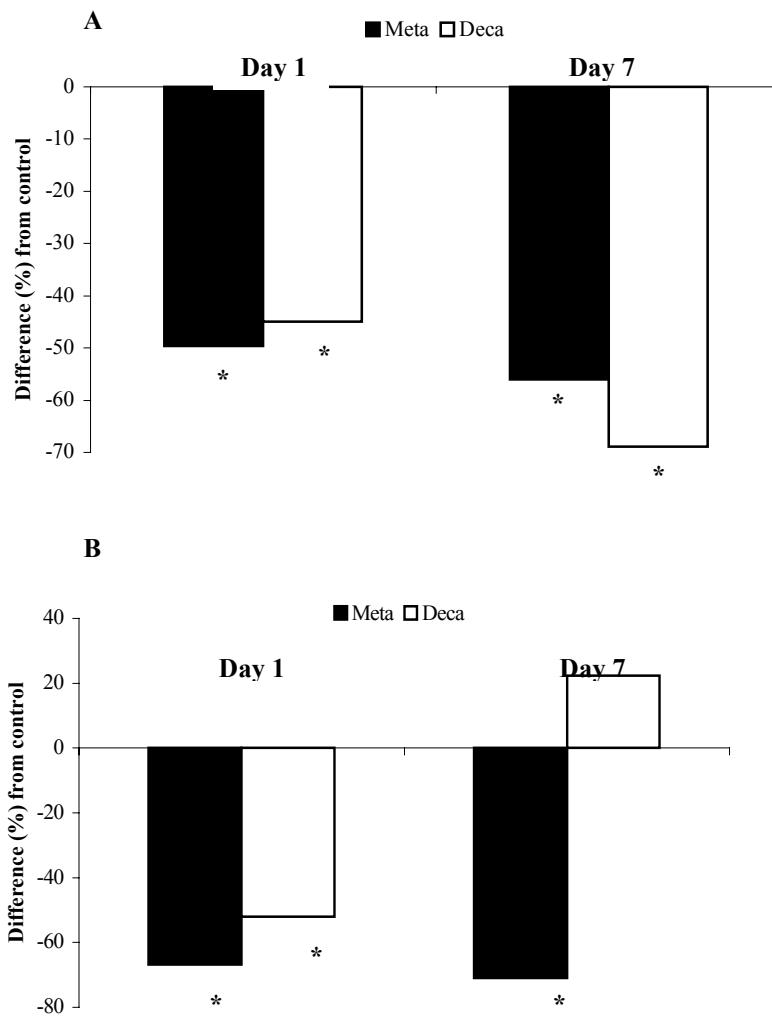


Figure 2. Effect of “metavandate” and “decavandate” (5 mM) exposure on diastolic flow peak velocity during the E (A) and A (B) waves; * $p < 0.05$.

Although the vanadium species used affected flow velocities, there was no evidence that systolic or diastolic time intervals or heart rate were affected by vanadium exposure.

In conclusion, vanadium significantly affects toadfish cardiac performance. The results obtained suggest that different vanadate species present in vanadium (V) solutions, may contribute to vanadium toxicity by decreasing ventricular filling flow and inducing diastolic ventricle dysfunction. Both ventricle relaxation and atrial contraction are depressed in the presence of vanadate compounds.

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