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Mini Review

Tonic Vagal Effect on Cardiac Inotropy

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Abstract

Recent studies have shown that arterial blood flow to each individual tissue is regulated, such that the rate of oxygen delivery (DO_2) is precisely controlled at a tissue specific individual ratio to each tissue's rate of oxygen consumption (VO_2). As a result, the total of these individually controlled blood flows constitutes venous return to the heart [1,2]. The heart then 'puts out what it receives' [3]. Hence, the resulting cardiac output (CO) has been controlled at the tissues.

This, new insight, is supported by the experimental work of Donald and Shepherd on greyhounds [4]. The animals ran a 4-minute race twice firstly, when intact and secondly after full recovery from complete cardiac neural ablation. The cardiac output increase during the 4-minute race was the same for the second race, despite near complete loss of the normal, neurally mediated increase in heart rate seen in the intact dogs. The conclusion concerning the inotropic state of the heart, is that its adjustment is appropriate to the load, an intrinsic property of the heart. The inotropic state is therefore independent of the neural supply. The inotropic response to increased arterial pressure in the 'working heart–brainstem preparation' of Nalivaiko et al. [5] was, similarly, attributed to an intrinsic property of the heart.

The normal response to the inflow of blood to the heart, therefore involves an appropriate inotropic response, independent of neural connection. Any augmentation of the vagal input, depressing cardiac inotropy is abnormal, either engendered under experimental conditions, or a result of cerebral dysfunction. This, it is suggested, is the reason vagal effects are found experimentally. For example, 'in the human and pig heart the left vagus nerve (stimulation) can profoundly decrease the inotropic state of the left ventricular myocardium' [6].

Although there is normally no vagal influence on cardiac contractility, Machhada et. al [7]. have shown tonic activity in vagal preganglionic neurones in the dorsal motor nucleus of the vague nerve furthermore, they found that their inhibition increased cardiac contractility. Hence, this pathway, seems normally to sustain constant tone, rather than operating as an inotropic control. There is supportive 'evidence that there is a tonic parasympathetic restraint of the inotropic function of the heart in human' [8]. However, situations where subjects show evidence of negative cardiac inotropy are abnormal, since they interfere with the normal cardiac inotropic values appropriate to the cardiac output.

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