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Spontaneous Heart Rate Variability and Arrhythmias in Congestive Heart Failure

Thesis

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for the Master Degree in Internal Medicine

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INTRODUCTION AND AIM OF WORK

Neurohumoral modulation of the cardiovascular function is an important component of the haemodynamic alterations in patients with congestive heart failure (CHF) (Saul et al., 1987).

It has now been observed that there is a normal 24-hour variation in heart rate (HR) that reflects the spontaneous changes in autonomic activity of the heart (Clarke et al., 1976). CHF is also associated with mechanical, endocrine and biochemical changes that may predispose to the development of cardiac arrhythmias (Lab et al., 1982). These arrhythmias in patients with ischaemic cardiomyopathy appear to independently increase the risk of sudden cardiac death (Rrigger et al., 1986).

The aim of the present study is to measure and analyze the heart rate variability as a non-invasive mean of investigating the autonomic control to the heart in patients with ischaemic heart failure. Furthermore, the present study examined prevalence of each type of cardiac arrhythmias during 24-hours in those patients.

Review of literature

Congestive Heart Failure

Definition:

In pathophysiological terms, heart failure can be defined as an inability of the heart to deliver blood oxygen at a rate commensurate with the requirements of the metabolizing tissues despite normal or increased ventricular filling pressures. Clinically, heart failure is the familiar syndrome of dyspnoea, fatigue and oedema arising as a consequence of this inadequate delivery of oxygen (McKee et al., 1971).

Pathophysiology of heart failure:

1- Central haemodynamics:

Central haemodynamics of heart failure can be described in terms of the three major determinants of stroke volume, i.e. contractility, preload and afterload.

a) Contractility:

Until recently, loss of contractility has been equated with loss of functioning muscle through infarction or cardiomyopathic process. It is now apparent however, that surviving muscle may show abnormal contractile responsiveness for example, the normal inotropic response to catecholamines may be reduced (McMurray et al., 1990).

Abnormally high concentrations of circulating hormones, neurotransmitters and other toxins may injure or even destroy the remaining muscle (Braunwald et al., 1991).

Alterations of calcium homeostasis, sarcoplasmic reticulum function, myocardial metabolism, mitochondrial function and myosin isoforms have also been described in congestive heart failure (Schultheiss et al., 1990).

b) Preload:

Preload describes the relation of the end-diastolic fiber stretch to muscle function. It is increased in congestive heart failure due to extracellular fluid volume expansion, venoconstriction, a reduction in venous capacitance and in many cases a decrease in ventricular compliance. Because the left ventricular function curve is depressed in congestive heart failure, an increase in preload leads to little increase in cardiac output and when end-diastolic pressure reaches a critical level, pulmonary oedema will occur. Coronary perfusion pressure is also reduced by a high preload (Stevenson et al., 1990).

c) Afterload:

After load refers to the load applied after the onset of muscle contraction. Afterload equates to wall tension which is determined by 3 factors: intraventricular pressure, radius of endocardial tissue and wall thickness. Left ventricular systolic pressure is governed by aortic impedance which is mainly determined by systemic vascular resistance and much less by large artery compliance, blood viscosity and effective blood volume (Herrlin et al., 1991). Numerous factors operate to increase systemic vascular resistance and therefore afterload. Vascular stiffness is increased by vessel wall sodium content (Sinoway et al., 1987). The second factor is increased sympathetic neural traffic and circulating catecholamines that occur in heart failure (Ferguson et al., 1990).

2- Neuro-endocrine abnormalities:

Many neurohumoral systems are activated in congestive heart failure in an attempt to restore the reduced arterial pressure and organ perfusion typifying the syndrome (Remes et al., 1991). The sympathetic nervous system, renin-angiotensin-aldosterone system (RAAS), arginine-vasopressin system (AVP), neuropeptide Y and endothelin act to cause smooth muscle vasoconstriction. In high concentrations, angiotensin II and catecholamines may be directly cardiotoxic (Ferguson et al., 1990). Many other adverse hormonal abnormalities have been described in congestive heart failure (Edvinsson et al., 1990). They include increased levels of vasodilator natriuretic substances as prostaglandins, atrial natriuretic peptides (ANP), dopamine and calcitonin gene-related peptide (CGPP) aiming to counteract some of the vasoconstrictor antinatriuretic influences already described (Narthridge et al., 1984).

3- Cardiovascular reflexes:

Much of the neurohumoral activation found in congestive heart failure may arise from abnormal baroreflex function (Marin-Neto et al., 1991). The arterial and cardiopulmonary mechanoreceptors appear to be less able to inhibit the vasomotor centers in congestive heart failure (CHF). This leads to enhanced sympathetic nervous system outflow. Parasympathetic autonomic function is also abnormal in CHF and diminished vagal tone is thought to contribute to the loss of heart rate variability in CHF patients. Abnormal autonomic activity of this kind is considered to be a risk factor for ventricular arrhythmias (Rea et al., 1990).

4- Electrolyte abnormalities:

CHF is also characterized by plasma and cellular electrolyte abnormalities, partly as a result of neurohormonal stimulation and partly as a consequence of drug treatment (Cleland et al., 1985). Plasma and more importantly intracellular potassium and magnesium deficiency are common and relate to the risk of arrhythmias and prognosis in CHF. Their deficiency also increases the risk of digitalis toxicity (Gottlieb et al., 1990).

Muscle [H^3] ouabain binding sites and muscle calcium concentration have also been reported to be decreased in CHF and these changes may contribute to the muscle dysfunction found in CHF (Droup et al., 1988). Intracellular sodium may be increased in CHF and this has been suggested to cause vascular wall stiffness and contribute to impaired vasodilatation in CHF (Sinoway et al., 1987). Finally, plasma sodium may be low in CHF. This may indicate severe CHF with intense renin-angiotensin system and vasopressin activation with reduced renal water excretion (water excess) (Anand et al., 1989).

5- Renal changes in CHF:

Chronic heart failure is characterized by a reduction in renal blood flow (RBF) (Packer et al., 1986). This fall is due to a reduction in cardiac output and probably due to the effects of neurohumoral systems on the renal vasculature (afferent renal arterioles and renal capillary vessels).